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Cognitive, Mood, and Cardiovascular Reactivity and Recovery in Response to Sadness in Remitted Major Depressive Disorder

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COGNITIVE, MOOD, AND CARDIOVASCULAR REACTIVITY AND RECOVERY IN RESPONSE TO SADNESS IN REMITTED MAJOR DEPRESSIVE DISORDER

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A DISSERTATION

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By Olivia E. Bogucki

Dissertation Advisor: Dr. Emily A. P. Haigh

An Abstract of the Dissertation Presented
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Major depressive disorder is marked by high rates of relapse and recurrence. Research has suggested that formerly depressed individuals exhibit dysphoric mood or dysfunctional beliefs that are similar to currently depressed individuals while in a dysphoric, but not euthymic, mood and these changes prospectively predict relapse and recurrence over time. While there is still disagreement as to whether dysfunctional thinking or dysphoric mood characterizes remitted depression, these changes appear to be mood state dependent, or undetectable until activated by sadness. These findings have led to the hypothesis that cardiovascular functioning may also be mood state dependent in remitted depression; however, this has not yet been adequately assessed. The few studies (Bylsma et al., 2015; Rottenberg et al., 2005b; Yaroslavsky et al., 2013, 2014) that have investigated cardiovascular reactivity in response to sadness in formerly depressed individuals have methodological issues. No studies have examined a wide range of cardiovascular measures to assess cardiovascular reactivity to and recovery from a sad mood in an exclusively formerly depressed sample.

The proposed study aimed to characterize cognitive, mood, and cardiovascular reactivity to and recovery from a sad mood in individuals with a history of depression compared to healthy,

never depressed individuals. Participants (N = 132) included formerly depressed and healthy control individuals. Following screening, participants completed self-report measures of depressive and anxiety symptoms and a structured clinical interview. Eligible participants were randomly assigned to an experimental paradigm condition. During the experimental paradigm, participants were connected to psychophysiological equipment, participated in a sad or neutral music and autobiographical recall mood induction, and completed self-report measures of dysfunctional thoughts and dysphoric mood pre- and post-mood induction. Results suggested that mood, rather than cognitive, reactivity in response to a transient sad mood is present in formerly depressed individuals. Additionally, results suggested that reduced heart period recovery, rather than reactivity, following the induction of a transient sad mood is present in formerly depressed individuals. Results indicated that formerly depressed individuals exhibit increased sadness and impaired heart period recovery in response to a transient sad mood, which may be potentially malleable risk factors for depressive relapse and recurrence.

DEDICATION

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LIST OF ABBREVIATIONS

ACT	Acceptance and commitment therapy
ANOVA	Analysis of variance
ANCOVA	Analysis of covariance
ANS	Autonomic nervous system
APA_A	American Psychiatric Association
APA_B	American Psychological Association
AUDADIS-IV	Alcohol Use Disorder and Associated Disabilities Interview Schedule – DSM-IV Version
AV	Atrioventricular
BAI	Beck Anxiety Inventory
BDI-II	Beck Depression Inventory – Second Edition
$_{BL}$	Baseline
BMI	Body mass index
BP	Blood pressure
$_{C}$	Cubic
CAB	Cardiac autonomic balance
CAR	Cardiac autonomic regulation
CBT	Cognitive-behavioral therapy
CD	Currently depressed
CD/RD	Mixed sample of current and remitted major depressive disorder
CDI-2	Children's Depression Inventory – Second Edition
CIDI	Composite International Diagnostic Interview
CITI	Collaborative Institutional Training Initiative
CO	Cardiac output
CNS	Central nervous system
CT	Cognitive therapy
CVD	Cardiovascular disease
DAS	Dysfunctional Attitudes Scale
DAS-SF I	Dysfunctional Attitudes Scale – Short Form I
DAS-SF II	Dysfunctional Attitudes Scale – Short Form II
DAS-SF I & II	Dysfunctional Attitudes Scale – Short Form I & II
DBT	Dialectical behavior therapy
DIS	Diagnostic Interview Schedule
DPAX	DePression and AnXiety
$_{DS}$	Difference score
DSM	Diagnostic and Statistical Manual
DSM-5	Diagnostic and Statistical Manual – Fifth Edition
DSM-IV	Diagnostic and Statistical Manual – Fourth Edition
DSM-IV-TR	Diagnostic and Statistical Manual – Fourth Edition – Text Revision
FD	Formerly depressed
ECG	Electrocardiogram
GHS	General Health Screen
GSC	Galvanic Skin Conductance

GSR	Galvanic Skin Response
H	Hypothesis
HC	Healthy control
HDRS	Hamilton Depression Rating Scale
HF-HRV	High frequency HRV
HP	Heart period
HPA	Hypothalamic-Pituitary-Adrenal
HR	Heart rate
HRV	Heart rate variability
IBI	Interbeat interval
ICG	Impedance cardiography
IMRC	Innovative Media, Research, and Commercialization Center
IRB	Institutional Review Board for the Protection of Human Subjects
ISCA-D	Interview Schedule for Children and Adolescents: Diagnostic Version
$_{L}$	Linear
LF-HRV	Low frequency HRV
MBCT	Mindfulness-based cognitive therapy
MI/$_{MI}$	Mood induction
MBCT	Mindfulness-based cognitive therapy
MDD	Major depressive disorder
MMDL	Maine Mood Disorders Lab
NEMESIS	Netherlands Mental Health Survey and Incidence Study
NIMH	National Institute of Mental Health
PANAS-X	Positive and Negative Affect Schedule – Expanded Form
PANAS-X N	Positive and Negative Affect Schedule – Expanded Form, Negative Affect General Dimension Scale
PANAS-X P	Positive and Negative Affect Schedule – Expanded Form, Positive Affect General Dimension Scale
PANAS-X F	Positive and Negative Affect Schedule – Expanded Form, Fear Basic Negative Emotional Scale
PANAS-X H	Positive and Negative Affect Schedule – Expanded Form, Hostility Basic Negative Emotional Scale
PANAS-X G	Positive and Negative Affect Schedule – Expanded Form, Guilt Basic Negative Emotional Scale
PANAS-X S	Positive and Negative Affect Schedule – Expanded Form, Sadness Basic Negative Emotional Scale
PEP	Pre-ejection period
PHQ-9	Patient Health Questionnaire – 9
PNS	Parasympathetic nervous system
$_{PRE}$	Pre-mood induction
PRIME-MD	Primary Care Evaluation of Mental Disorders
$_{POST}$	Post-mood induction
PTT	Pulse transmission time
$_{Q}$	Quadratic
$_{RA}$	Reactivity

$_{RC}$	Recovery
RD	Remitted depressed
RDoC	Research domain criteria
REBT	Rational emotive behavior therapy
$_{RM}$	Repeated measures
RMSSD	Root Mean Square Successive Difference
RSA	Respiratory sinus arrhythmia
SA	Sinoatrial
SADSL	Schedule for Affective Disorders and Schizophrenia – Lifetime Version
SCID-I	Structured Clinical Interview for DSM-IV
SCID-IV-RV	Structured Clinical Interview for DSM-IV-TR – Research Version
SDNN	Standard Deviation of the Normal-to-Normal
SNS	Sympathetic nervous system
STAI-I	State-Trait Anxiety Inventory – I
STAI-II	State-Trait Anxiety Inventory – II
STAI-I & II	State-Trait Anxiety Inventory – I & II
SV	Stroke volume
TAU	Treatment as usual
TPR	Total peripheral resistance
TP-HRV	Total power HRV
TSST	Trier Social Stress Task
ULF-HRV	Ultra-low frequency HRV
VAS	Visual Analogue Scale
VC	Vasoconstriction
VLF-HRV	Very low frequency HRV
Z_{RES}	Residualized change scores
$_{2}$	Average obtained during a two-minute interval
$_{5}$	Average obtained during a five-minute interval

CHAPTER 1

INTRODUCTION

Major Depressive Disorder (MDD), characterized by prolonged depressed mood and/or lack of interest or pleasure, is a serious and debilitating mental illness. Prevalence and incidence rates obtained from large scale epidemiological studies have indicated that MDD is a commonly occurring disorder. Various psychosocial factors that have been associated with MDD include gender, age, race, ethnicity, national origin, and socioeconomic status. The course of MDD is marked by high rates of relapse and recurrence. Consequently, it is often considered a chronic disorder that typically recurs over time. While psychological and medical comorbidities commonly co-occur with MDD, the disorder itself is independently associated with elevated levels of functional impairment, disability, and death. The direct and indirect problems associated with MDD pose a significant health and economic burden for the individuals suffering with the disorder and society.

Diagnostic Criteria

According to the Diagnostic and Statistical Manual – Fifth Edition (DSM-5; American Psychological Association (APA_A), 2013), MDD is characterized by persistent depressed mood and/or diminished interest or pleasure in previously enjoyed activities. These symptoms can be based on the subjective experience reported by the individual or objective presentation observed by others. In addition to these cardinal symptoms, an individual must endorse four or more of the following disturbances in appetite, weight, sleep, psychomotor activity, energy level, self-conceptualization, cognitive ability, and suicidality. There is significant heterogeneity in symptom presentation, such as increases or decreases in appetite, weight, sleep, and psychomotor activity. Individuals with MDD often report persistent reductions in energy level despite

adequate rest, negative self-concept (e.g., worthlessness or inappropriate guilt), and impaired cognitive abilities (e.g., difficulty thinking or making decisions). In addition, a spectrum of suicidal thoughts and behaviors can occur, including recurrent thoughts of death or dying and suicidal ideations, plans, or attempts. A clinical diagnosis of depression requires that symptoms are present most of the day, nearly every day for two weeks or longer and result in marked impairment at school, work, or home or in social situations. In addition, these symptoms must be attributable to depression and cannot be better explained by a medical condition, substance use, or other psychiatric condition.

Recovery from a major depressive episode is classified as partial or full remission (APA$_A$, 2013). MDD in partial remission is defined as the absence of depressive symptoms for less than two months or the presence of depressive symptoms that cause marked impairment but do not meet the diagnostic threshold. MDD in full remission is defined as either the absence of any depressive symptoms for at least two months or the presence of one or two depressive symptoms that are mild in severity.

The diagnostic criteria for MDD allows for significant heterogeneity of symptoms. Researchers have criticized the architects of the DSM-5 for its reliance on a symptom-based, categorical approach, limited use of biological correlates, and inclusion of contradictory symptomology within a single diagnosis (e.g., increase or decrease in appetite; Casey et al., 2013). While the DSM-5 provides researchers and clinicians with the nomenclature necessary for communication, the field is currently exploring alternative classification systems such as the Research Domain Criteria (RDoC; Insel et al., 2010).

Occurrence

MDD is one of the most commonly occurring mental disorders (National Institute of Mental Health (NIMH), 2017). The occurrence of MDD within the general population has been assessed by large epidemiological studies using two different measurement methods: prevalence and incidence.

Prevalence

Prevalence is defined as the proportion of the population who have a condition during a specific period of time. The majority of studies report point prevalence rates obtained within a 12-month period or within an individual's lifetime. Several large-scale epidemiological studies using the DSM – Fourth Edition (DSM-IV; APA$_A$, 2000) diagnostic criteria have obtained a range of prevalence rates of MDD depending on the sample and methodology used.

Eaton, Kalaydjian, Scharfstein, Mezuk, and Ding (2007) reported the lowest 12-month prevalence rate of 2.70% among two cohorts using the Diagnostic Interview Schedule (DIS). Hasin, Goodwin, Stinson, and Grant (2005) found a 12-month prevalence rate of 5.30% and a lifetime prevalence rate of 13.20% using the Alcohol Use Disorder and Associated Disabilities Interview Schedule – DSM-IV Version (AUDADIS-IV). The majority of studies have used the Composite International Diagnostic Interview (CIDI) to diagnose depression, which have obtained higher prevalence rates of MDD. Kessler and colleagues (2003) attained a 12-month prevalence rate of 6.60% and a lifetime prevalence rate of 16.20%. Similarly, Kessler and colleagues (2005) found a lifetime prevalence rate of 16.60%. González, Tarraf, Whitfield, and Vega (2010) obtained a 12-month prevalence rate of 8.10% and a lifetime prevalence rate of 18.60%. Finally, Kessler and colleagues (2010) reported the highest 12-month prevalence rate of 8.30% and lifetime prevalence rate of 19.20%. Together, these epidemiological studies suggest

that the prevalence rate for MDD ranges between 2.70% and 8.30% during a 12-month period and 13.20% to 19.20% across an individual's lifespan.

Incidence

Incidence is defined as the number of new cases of a condition within a certain time period. The majority of studies report person-time incidence rates, which is defined as the number of new cases that occur within the amount of time that the sample of participants were at risk for developing the disease of interest. While prevalence rates are more commonly reported in the literature, they are influenced by the chronicity of a disorder (Palsson, Östling, & Skoog, 2001). MDD is characterized as a chronic disorder in a subset of individuals (Monroe, Anderson, & Harkness, in press; Richards, 2011); therefore, it is important to also assess the incidence rate for MDD. Several large-scale epidemiological studies using the DSM-IV diagnostic criteria have obtained a range of incidence rates of MDD depending on the measurement formula and methodology used.

Eaton and colleagues (2007) reported the lowest incidence rate of 1.90 per 1,000 person years using the DIS. Murphy, Laird, Monson, Sobol, and Leighton (2000) found similar incidence rates for two cohorts using the DePression and AnXiety (DPAX) interview. Incidence rates ranged from 4.50 per 1,000 person years for the cohort recruited from 1950 to 1970 to 3.70 per 1,000 person years for the cohort recruited from 1970 to 1992. Grant and colleagues (2009) obtained the highest incidence rate of 1.51 per 100 person years, which is equal to 15.10 per 1,000 person years, using the AUDADIS-IV. Together, these epidemiological studies suggest that the incident rate for MDD ranges between 1.90 and 15.10 per 1,000 person years.

Gender

One of the strongest predictors for the occurrence of MDD is gender. Epidemiological studies conducted in the United States have consistently shown that women report increased prevalence and incidence rates of MDD. Kessler and colleagues (2003) reported that the prevalence rate of MDD was elevated in women during a 12-month period (OR = 1.40, CI = 1.10, 1.80, $p < .05$) and over the course of their lifetime (OR = 1.70, CI = 1.50, 2.00, $p < .05$) using the CIDI. Hasin and colleagues (2005) replicated these results using the AUDADIS-IV, showing that the prevalence rate of MDD was two times higher in women over the course of their lifetime (OR = 2.00, CI = 1.80, 2.40). Eaton and colleagues (2007) obtained even more staggering results with the DIS, finding that the prevalence rate of MDD was over three times higher in women during a 12-month period (OR = 3.80, CI = 2.60, 5.60). In addition, the authors reported that the incidence rate of MDD was over two times higher in women during a 12-month period (OR = 2.60, CI = 1.50, 4.10). Similarly, Grant and colleagues (2009) found that women reported an elevated incidence rate of MDD (OR = 1.00) compared to men (OR = .50, CI = .37, .76) on the AUDADIS-IV.

These results have been replicated by epidemiological studies conducted in countries around the world, suggesting that increased rates of depression in women occurs cross-culturally. Kuehner (2003) completed a systematic review of epidemiological studies that assessed the prevalence of depression worldwide using various structured clinical interviews. Results indicated that the prevalence rates for depressive disorders were significantly higher in women compared to men, with a sex ratio of 1.70:1.00 for current prevalence and 2.10:1.00 for lifetime prevalence. These results held true for studies conducted outside of the United States, which obtained a sex ratio of 2.00:1.00 for both current and lifetime prevalence. Additional large-scale

studies have been conducted to specifically assess gender differences in depression. Seedat and colleagues (2009) obtained lifetime rates of MDD for individuals residing in 15 countries using the CIDI. Results revealed that the lifetime prevalence rate for MDD was significantly higher in women compared to men (OR = 1.90, CI = 1.80, 2.00). Together, these studies suggest that the prevalence and incidence of MDD is generally two times higher in women compared to men (Nolen-Hoeksema, 2001).

Several factors have been proposed to explain the elevated rates of MDD observed in women. First, it is possible that differences in depression are an artifact of diagnostic or methodological problems. Diagnostic issues include differences in the endorsement or experience of depressive symptoms among men and women. Research has shown that women are more likely to endorse more depressive symptoms than men, even though the two sexes experience similar levels of impairment (Piccinelli & Wilkinson, 2000). In addition, it has been suggested that there may be symptomatic differences between the sexes; women may be more likely to experience stereotypical depressive symptoms like sadness and men may be more likely to experience atypical depressive symptoms like irritability. Consequently, the diagnostic criteria for MDD has been criticized as overemphasizing the depressive symptoms that are typically experienced by females (Kuehner, 2003). Methodological issues include failure to account for differences in the course of the disorder or treatment seeking behaviors among men and women. Some research has suggested that females may be more likely to experience a singular depressive episode, which would account for difference in point prevalence rates but not lifetime prevalence rates (Piccinelli & Wilkinson, 2000). In addition, research has documented the differences in treatment seeking behaviors across the sexes, which may explain why women are more likely to be identified as having depression. Service utilization studies have shown that women are more

likely than men to seek medical and psychological treatment (Piccinelli & Wilkinson, 2000) while studies conducted in community and primary care settings have estimated that the prevalence of MDD is roughly equal across the sexes (Kuehner, 2003). The literature has identified some diagnostic or methodological issues that may account for the gender difference in depression.

Second, biological factors specific to women have been associated with increased vulnerability to developing depression. While differences in the heritability of depression have not be identified, genetic differences may make women more vulnerable to developing internalizing disorders (Kuehner, 2003). Sex hormones, such as estrogen, may directly or indirectly impact mood. Research has shown that changes in sex hormones are associated with changes in mood during puberty, postpartum, and premenstrual periods (Grigoriadis & Robinson, 2007; Kuehner, 2003). In addition, sex hormones influence the activity of regulatory mechanisms in the brain including neurotransmitters and components of the endocrine system such as the hypothalamic-pituitary-adrenal (HPA) and hypothalamic-pituitary-thyroid axes (Grigoriadis & Robinson, 2007; Kuehner, 2003).

Third, it has been proposed that psychosocial factors specific to women increase vulnerability to depression. Women possess less social status and power in society compared to men, which increases the likelihood for experiencing stressful life events such as sexual abuse and assault, trauma, and victimization (Grigoriadis & Robinson, 2007; Nolen-Hoeksema, 2001). In addition, society has subscribed certain social roles and expectations to women, which can lead to significant emotional distress due to personal, educational, professional, and financial limitations that may result in reduced freedom and autonomy (Kuehner, 2003; Piccinelli & Wilkinson, 2000).

Fourth, it has been proposed that psychological factors commonly observed in women may increase their vulnerability to depression. Differences in coping style appear to exist among men and women. Females tend to cope with stress using maladaptive strategies such as rumination, or internally dwelling on problems, while males tend to use more adaptive and active strategies such as distraction. Research has shown that rumination negatively impacts mood and problem solving strategies and plays a significant role in the development of depression (Nolen-Hoeksema, 2001). Additionally, some sex differences in self-perception have been identified. Women tend to report lower self-esteem, self-confidence, and perceived control compared to men, which may contribute to feelings of hopelessness and helplessness that are associated with depression (Grigoriadis & Robinson, 2007; Kuehner, 2003; Piccinelli & Wilkinson, 2000).

Finally, the presence of previous mental health problems in women during adolescence have been associated with increased vulnerability to depression across the lifespan. Research has shown that women are at an increased risk for developing anxiety and depression earlier in life compared to men (Piccinelli & Wilkinson, 2000). Early exposure to anxiety or depression is thought to make women more vulnerable to experiencing multiple depressive episodes over the course of their lifetime. This theory is based on the findings that the course of depression is typically chronic and anxiety increases risk for the occurrence of depressive episode in the future (Kuehner, 2003).

Age

As reviewed by Haigh, Bogucki, Sigmon, and Blazer (2018a), it has long been assumed that older age is related to an increased occurrence of MDD. This commonly held belief is likely due to the challenges typically associated with aging (e.g., changes in interpersonal relationships and reduced independence, financial stability, and physical and cognitive capacity), which can

result in significant emotional distress. However, the literature has consistently indicated that older adults are significantly less likely to experience MDD compared to their younger counterparts.

Large scale epidemiological and community-based studies have shown that younger adults report higher prevalence and incidence rates of MDD compared to older adults. Kessler and colleagues (2003) compared the prevalence of MDD across age groups using the CIDI. Participants 60 years or older reported significantly lower point (OR = 1.00, $\chi^2 = 42.3$, $p < .05$) and lifetime (OR = 1.00, $\chi^2 = 53.5$, $p < .05$) prevalence rates of MDD compared to younger participants. 12-month prevalence rates were significantly higher for participants age 18 to 29 (OR = 3.00, CI = 2.00, 4.40, $p < .05$) and 30 to 44 (OR = 1.80, CI = 1.10, 2.90, $p < .05$) while lifetime prevalence rates were significantly higher for participants age 18 to 29 (OR = 1.70, CI = 1.40, 2.20, $p < .05$), 30 to 44 (OR = 2.20, CI = 1.80, 2.80, $p < .05$), and 45 to 59 (OR = 2.00, CI = 1.60, 2.60, $p < .05$).

Kessler and colleagues (2005) replicated this result using the same structured interview, finding a statistically significant difference for the lifetime prevalence rate of MDD in participants age 18 to 29 (15.40%), 30 to 44 (19.80%), 45 to 59 (18.80%), and 60 or older (10.60%; $\chi^2 = 49.90$, $p < .05$). Eaton and colleagues (2007) compared incidence rates of MDD among middle age and older adults using the DIS. Younger participants age 30 to 44 (3.20%) and 45 to 64 (1.90%) reported elevated incidence rates of MDD compared to participants 65 years or older (0.00%). Grant and colleagues (2009) examined differences in the incidence of MDD across a wider age range with the AUDADIS-IV. Compared to participants 55 years or older (OR = 1.00), incidence rates for MDD were significantly higher in participants age 20 to 29 (OR = 2.00, CI = 1.19, 3.41, $p < .01$) and 30 to 54 (OR = 1.70, CI = 1.22, 2.47, $p < .01$).

Kessler and colleagues (2010) specifically assessed age-related differences in MDD using the CIDI. Results indicated that there was a statistically significant difference in the prevalence of MDD assessed across age groups. For the 30-day prevalence rate, participants in the 18 to 34 and 35 to 49-year age groups (3.70%) reported the highest rates while participants in the over 65 years age group reported the lowest rates (1.00%; $\chi^2 = 46.9$, $p < .05$). For the 12-month prevalence rate, participants in the 18 to 34-year age group (10.40%) reported the higher rates while participants in the over 65 years age group reported the lowest rates (2.60%, $\chi^2 = 103.50$, $p < .05$). For the lifetime prevalence rate, participants in the 35 to 49-year age group (22.70%) reported the higher rates while participants in the over 65 years age group reported the lowest rates (9.80%, $\chi^2 = 70.40$, $p < .05$). Together, these studies suggest that younger adults are at the highest risk for experiencing MDD. While the rates of MDD reported by middle age adults are typically lower than young adults, they are still elevated compared to elderly population.

Multiple factors have been proposed to explain why the rate of MDD differs across the lifespan. First, it is possible that recruitment and methodological issues account for the low rates of depression reported by older adults. Recruitment issues include premature death, failure to account for the elderly population that resides in assisted living or nursing home facilities, and diminished interest in participating in research. Methodological issues include recall bias and failure to endorse the presence of mental illness due to stigma and social desirability. However, there is limited evidence supporting these recruitment and methodological issues, which suggests that MDD does occur at different rates across the lifespan (as reviewed by Kessler et al., 2010). Second, psychological factors specific to older adults may be protective against depression later in life. Research has indicated that older adults report an increase in positive affect and well-being and a decrease in negative affect, which is theorized to be due to enhanced emotion

regulation abilities or a normalization of adverse events that occur during this phase of life (as reviewed by Haigh et al., 2018a). Therefore, it is possible that older adults are better able to cope with stressful life events, which are a causal risk factor for depression (Kendler, Karkowski, & Prescott, 1999), than their younger counterparts.

Race, Ethnicity, and National Origin

Race, ethnicity, and national origin impact the occurrence of MDD. Large scale epidemiological and community-based studies have identified some differences in the prevalence rates among racial and ethnic groups. Kessler and colleagues (2003) found that African American individuals reported the lowest lifetime prevalence rate of MDD (OR = .60, CI = .50, .80, $p < .05$) on the CIDI compared to individuals who identified as Hispanic, Caucasian, or other (OR = 1.00-1.20, CI = .80, 1.50). No significant differences in 12-month prevalence rates were identified between groups. Hasin and colleagues (2005) found that compared to Caucasian participants, Native American participants reported significantly higher lifetime prevalence rate of MDD (OR = 1.50, CI = 1.10, 2.10) while African American, Asian or Pacific Islander, and Hispanic participants reported a significantly lower lifetime prevalence rate of MDD (OR = .60-.70, CI = .40, .90) on the AUDADIS-IV. Williams and colleagues (2007) found that Caucasian participants reporting significantly higher lifetime prevalence rates of MDD (17.90%, $p < .001$) on the CIDI compared to African American (10.40%) and Caribbean Black (12.90%) participants. Similar to Kessler and colleagues (2003), no significant differences in 12-month prevalence rates reported on the CIDI were identified between groups.

Other studies have used the CIDI to examine the impact of other race-related variables, such as membership to a particular ethnic group and country of national origin. Alegría and colleagues (2007) examined differences in prevalence rates for among Latino individuals living

in the United States. Findings suggested that there were some group differences; participants of Mexican descent were significant less likely to experience any depressive disorder during their lifetime (OR = .57-.69, CI = .34, .99, $p < .05$) compared to Puerto Rican participants. However, this relationship was not found for 12-month prevalence rates. Alegría and colleagues (2008) investigated differences in prevalence rates among Latino and non-Latino individuals living in the United States. Findings indicated that non-Latino Caucasian participants had significantly higher lifetime prevalence rates for MDD (22.10%, $p < .001$) compared to all Latino participants (15.20%). Lifetime prevalence rates did not significantly differ among Latino subgroups ($p = .65$). Immigration status also had an impact on both groups, with participants born in the United States reporting significantly higher lifetime prevalence rates for both Latino ($p < .001$) and non-Latino ($p < .008$) groups. González and colleagues (2010) reported differences in prevalence rates for MDD based on ethnic group and immigration status. Results indicated that ethnic groups reported differences in both 12-month ($\chi^2 = 33.70$, $p < .001$) and lifetime ($\chi^2 = 4.60$, $p < .001$) prevalence rates for MDD, with Puerto Rican participants reporting the highest rates and Filipino, Vietnamese, and Chinese participants reporting the lowest rates. In addition, participants who were born in the United States reported higher 12-month ($\chi^2 = 28.20$, $p < .001$) and lifetime ($\chi^2 = 87.30$, $p < .001$) prevalence rates of MDD compared to foreign born participants.

Together, these studies suggest that Caucasian individuals are significantly more likely to experience MDD over the course of their lifetime than individuals from diverse racial or ethnic backgrounds, with the exception of Native American individuals. In addition, it appears that individuals born in the United States are at higher risk for developing MDD than foreign born individuals who immigrate from their country of origin.

Several factors have been proposed to explain the elevated rates of MDD observed in Caucasian individuals. First, it is possible that the low rates of depression in racial and ethnic minorities is attributable to cultural bias. The diagnostic system (i.e., DSM) used to assess the presence of mental disorders has been criticized for failing to adequately represent minority groups, which is likely to impact the assessment and diagnosis of mental illness (as reviewed by Kress, Eriksen, Rayle, & Ford, 2005). In addition, the clinicians who implement the diagnostic system may be culturally biased. Multicultural competence is a requirement of ethical practice; clinicians must assess a client's cultural identify to obtain an accurate formulation of a client's psychological, emotional, and behavioral functioning (as reviewed by Kress et al., 2005). However, it is possible that clinicians who have inadequate cultural training may exhibit a bias when assessing and diagnosing mental illness in culturally diverse clients. Second, the low rates of depression in racial and ethnic minorities may be due to cultural differences in the experience of depression, which has an impact on symptom reporting, treatment seeking behavior, and the therapeutic relationship (Kleinman, 2004). Finally, some race-related variables (e.g., ethnicity, national origin, acculturation status, etc.) are often overlooked by large epidemiology or community-based studies (Alegría et al., 2008; Williams et al., 2007). Additional research on the influence of these cultural factors on depression is warranted.

Socioeconomic Status

Socioeconomic status is defined as a combination of educational achievement and income level. Research has identified an association between socioeconomic status and depression. Cross-sectional studies have generally shown that individuals of low socioeconomic status report elevated levels of depressive symptoms compared to individuals of middle or high socioeconomic status (as reviewed by Gallo & Matthews, 2003). Longitudinal studies have

generally shown that low socioeconomic status prospectively predicts an increased risk for the development of MDD over time (as reviewed by Everson, Maty, Lynch, & Kaplan, 2002; Gallo & Matthews, 2003; Muntaner, Eaton, Miech, & O'Campo, 2004). Of note, some longitudinal studies that have used a specific epidemiological sample (i.e., Epidemiologic Catchment Area Study) or dichotomous variables to classify socioeconomic status have failed to find this association, suggesting that these null findings could be due to methodological differences across studies (see Gallo & Matthews, 2003 for review of discrepant results). Together, these results suggest that compared to individuals of higher socioeconomic status, individuals of lower socioeconomic status are generally at higher risk for experiencing depressive symptoms and a clinically-significant depressive episode over the course of their lifetime.

Building on this body of work, Lorant and colleagues (2003) conducted a meta-analysis of 51 articles to quantify the relationship between socioeconomic status and depression. Depression was assessed via self-report or structured clinical interviews, such that the final sample was comprised of individuals with clinical and non-clinical depression. Results indicated that the individuals of lower socioeconomic status reported higher prevalence (OR = 1.81, CI = 1.57, 2.10, $p < .001$) and incidence (OR = 1.24, CI = 1.04, 1.48, $p < .004$) rates of MDD compared to individuals of higher socioeconomic status. In addition, individuals of lower socioeconomic status were significantly more likely to experience persistent MDD (OR = 2.06, CI = 1.39, 3.05, $p < .001$). A dose dependent relationship between socioeconomic factors and depression appeared to exist. Increases in education and income were associated with decreases in the likelihood of depression, which suggests that there is a linear relationship between these variables. Of note, some of the studies utilized self-report measures to assess depressive symptoms rather than diagnostic measures, which limits the generalizability of results to clinical

samples. Overall, this body of research indicates that individuals of lower socioeconomic status are at higher risk for experiencing depressive symptoms and depression compared to individuals of higher socioeconomic status.

Multiple factors have been proposed to explain the elevated rates of MDD observed in individuals of lower socioeconomic status. First, it has been suggested that individuals of lower socioeconomic status are exposed to more stressful life events or have less resources to combat stressful life events. More specifically, these individuals may exhibit more maladaptive coping strategies and diminished perception of personal abilities, mastery, and control in response to stressful life events (Lorant et al., 2003). Second, it has been proposed that individuals of lower socioeconomic status experience significantly more strain due to larger societal factors. For example, these individuals are treated more negatively due to societal views and values associated with economic standing and public policy (Lorant et al., 2003). Therefore, it is possible that individual or societal factors or an interaction between these factors contribute to the relationship between socioeconomic status and depression.

Comorbidity

Psychological Comorbidity

MDD is marked by elevated rates of psychological comorbidities. Multiple large-scale epidemiological studies have calculated rates of disorders comorbid with MDD. Kessler and colleagues (2003) found that MDD was associated with elevated rates of comorbid psychological conditions on the CIDI. The majority of participants (64.00%) who endorsed experiencing MDD over the past 12 months also endorsed the presence of a comorbid psychological disorder, which included an anxiety (57.50%), impulse control (16.60%), or substance use (8.50%) disorder. An even higher percentage of participants (72.10%) who endorsed experiencing MDD over the

course of their lifetime also endorsed the presence of a comorbid psychological disorder, which included an anxiety (59.20%), impulse control (30.00%), or substance use (24.00%) disorder. The onset of MDD was generally proceeded by another psychological disorder, with MDD occurring first in a relatively small portion of participants (12.60% for 12-month prevalence, 12.30% for lifetime prevalence). Kessler and colleagues (2010) replicated these results using the same diagnostic measure. Anxiety (64.20%) and other mood (37.20%) disorders were the most common comorbid conditions while impulse control (14.70%) and substance use (10.70%) disorders were less likely, but still frequently reported within the sample. Results indicated that the majority of participants (75.80%) endorsed the presence of at least one comorbid psychological condition, with participants reporting one (25.60%), two (17.60%), or three or more (32.60%) comorbidities.

Hasin and colleagues (2005) examined a more expansive list of comorbid psychological conditions using the AUDADIS-IV. Participants who endorsed MDD during the past 12 months reported elevated rates of personality disorders (37.90%), anxiety disorders (36.10%), and nicotine dependence (26.00%). Of note, these participants reported relatively low rates of alcohol (14.10%) and drug (4.60%) use disorders. A different pattern of comorbidity emerged for participants who endorsed MDD at some point during their lifetime, including elevated rates of anxiety disorders (41.40%), alcohol use disorders (40.30%), personality disorders (30.80%), nicotine dependence (30.00%), and drug use disorders (17.20%). The authors concluded that individuals who endorse MDD over a 12-month period or their lifetime are highly likely to also endorse the presence of a comorbid condition during the same time span.

The literature has clearly shown that MDD is associated with elevated rates of psychological comorbidity. Across studies, anxiety disorders are the most prevalent class of

comorbid conditions. Personality, mood, impulse control, and substance use disorders also commonly co-occur with MDD.

Medical Comorbidity

MDD has been associated with an elevated risk for chronic medical conditions, such as asthma, arthritis, cardiovascular disorder (CVD), cancer, and diabetes (as reviewed by Chapman, Perry, & Strine, 2005). Epidemiological studies have provided additional evidence for the association between depression and medical conditions. Kessler and colleagues (2010) found that individuals who endorsed MDD on the CIDI over a 12-month period were highly likely to also endorse the presence of a comorbid medical condition during the same time span. Various types of musculoskeletal (48.20%), respiratory (43.50%), pain (41.30%), and cardiovascular (24.20%) disorders were commonly reported by individuals who have recently suffered from depression. Results indicated that the majority of participants (79.80%) endorsed the presence of at least one medical comorbidity, with participants reporting one (22.30%), two (21.10%), or three or more (36.40%) comorbidities.

These results have been replicated by large scale studies conducted around the world. Moussavi and colleagues (2007) assessed the association between MDD and medical conditions in a culturally diverse sample. Participants from 60 countries were assessed for the presence of chronic medical conditions based on reports of MDD using the CIDI. Prevalence rates for chronic medical conditions were relatively low, with rates ranging as high as 4.50% for angina and as low as 2.00% for diabetes. There was a strong association between depression and chronic medical conditions; participants with diabetes (9.30%), arthritis (10.70%), angina (15.00%), and asthma (15.00%) also reported experiencing comorbid depression. The rates of depression were even higher in participants who reported more than one chronic medical illness (23.00%), which

was significantly different from healthy participants free from such conditions (3.20%, $p < .001$). In addition, participants with chronic medical conditions and comorbid depression reported significantly lower health scores than non-depressant participants with one or more chronic medical conditions ($p < .0001$). Together, these results suggest that depression commonly co-occurs with comorbid medical conditions and has an additive negative impact on physical health.

The literature has indicated that depression is also associated with elevated rates of mortality. Depressed individuals are significantly more likely to die by suicide than the general population. Research has consistently shown that suicidal thoughts and behaviors are significantly more common in depressed individuals. Depressed individuals are about 20 times more likely to attempt and commit suicide compared to individuals who have never been depressed (as reviewed by Lépine & Briley, 2011). In addition, the presence of certain comorbid medical conditions in depressed individuals has been associated with an increased risk of death. More specifically, research has shown that individuals with CVD and comorbid depression or depressive symptoms are significantly more likely to die from cardiovascular-related causes than their non-depressed counterparts (as reviewed by Lépine & Briley, 2011). These results indicate that depression has a significant and negative impact on an individual's life trajectory.

As outlined by Katon (2003), there are several potential reasons for the relationship between depression and medical conditions. First, it is possible that depression is a risk factor for certain medical conditions. Depression is associated with negative health behaviors (e.g., inactivity and obesity) that may contribute to the development of medical conditions. Longitudinal studies that have found that depression prospectively predicts the risk for some medical conditions, such as diabetes and CVD, have provided some support for this hypothesis. Second, depression may result from the experience of developing a potentially life-threatening

medical condition. This experience can result in functional impairment, reduced quality of life, and feelings of helplessness, hopelessness, and stress, potentially leading to the development of depression. Third, depression may be the result of a medication prescribed to treat the medical condition. Depression is a known side effect for certain medications. However, depression has also been reported as a nonspecific side effect that is not due to the pharmacological mechanism of action (Barsky, Saintfort, Rogers, & Borus, 2002). Finally, it is possible that the medical condition causes physiological changes that leads to the development of depression. The potential mechanisms of change may be directly or indirectly related to the medical condition; direct effects include changes to the brain structure or function while indirect effects include changes in physiological systems (e.g., inflammation and cytokines) that has down-stream effects on the brain.

Individual and Societal Impact

Individual Impact

MDD is associated with significant cost to the individual and society. Individuals with MDD struggle with maladaptive thoughts, feelings, and behaviors that negatively impact multiple aspects of their lives. In addition, these individuals experience elevated functional impairment and reduced quality of life that make it more difficult to function within the confounds of society. Functional impairment is defined as diminished ability to perform everyday tasks. Backenstrass and colleagues (2006) assessed the impact of depression on different areas of functioning. Participants who reported minimal depressive symptoms (n = 56) or depressive symptoms consistent with major (n = 28) or minor (n = 38) depression on the Patient Health Questionnaire – 9 (PHQ-9) were compared to healthy control participants (n = 491). Results indicated that participants who reported symptoms consistent with MDD endorsed

experiencing significantly more days of minor impairment and missing daily activities or work due to illness compared to participants with subclinical depressive symptoms or healthy control participants ($p < .001$), suggesting that functional impairment is significantly worse in individuals with clinically significant symptoms. These results indicate that depression has a significant and negative impact on an individual's ability to function within everyday activities across multiple settings.

Quality of life is defined as diminished well-being due to disability in different aspects of functioning, including physical and emotional health, professional and economic standing, interpersonal relationships, and life satisfaction. Rapaport, Clary, Fayyad, and Endicott (2005) assessed differences in quality of life using a large sample of participants with MDD ($n = 242$) recruited from 11 multisite trials. Quality of life was assessed using the Quality of Life Enjoyment and Satisfaction Questionnaire. Results indicated that participants with MDD endorsed lower quality of life ratings for all categories compared to healthy control participants from the community ($n = 67$). The majority of participants with MDD (63.00%) reported clinically severe impairments in quality of life relative to healthy control participants (1.70%). Impairments were even more pronounced in chronic or double depression (i.e., MDD and dysthymia), with 85.00% of participants reporting clinically severe impairments in quality of life. These results indicate that depression has a significant and negative impact on an individual's ability to engage in positive and pleasurable activities.

Societal Impact

Overall, the literature suggests that depression has a negative impact on an individual's functional ability and quality of life. These impairments result in a significant burden that makes it difficult for depressed individuals to engage in typical aspects of daily life. Consequently,

depression is associated with increased disability rates and decreased workplace productivity (Lépine & Briley, 2011). Over the past several decades, the burden of depression has steadily risen. As outlined by Whitaker (2015), the disability rate rose from one in every 468 Americans in 1955 to one in every 184 Americans in 1987. This trend has continued to grow exponentially, with the disability rate reaching one in every 70 Americans in 2013. This finding does not appear to be confined to the United States, with other Western countries reporting similar increases in disability rates. According to Mathers, Fat, and Boerma (2008), MDD is currently the leading cause of disability and second leading cause of disease burden around the world. The negative impact of MDD is projected to significantly increase relative to other disorders over the next decade. Predictions have indicated that MDD will become the leading cause of disease burden by the year 2030. These projections have significant implications for an individual's ability to function within the confounds of society.

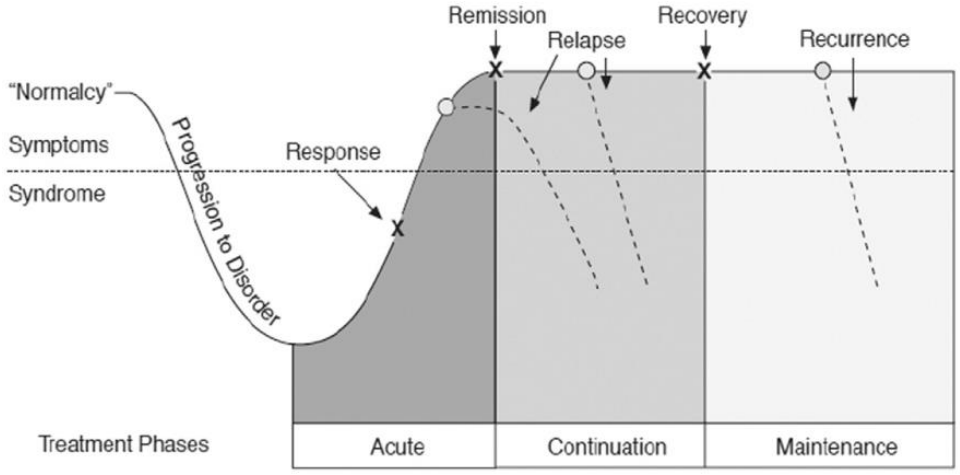
The complications directly and indirectly associated with MDD are extremely costly to society. The economic burden associated with MDD has risen in recent years. According to Greenberg, Fournier, Sisitsky, Pike, and Kessler (2015), the estimated cost of MDD has increased from $173.50 billion in 2005 to $210.50 billion in 2010. The rise in estimated cost was attributable to increases direct (i.e., medical and pharmaceutical treatment expenses) and indirect (i.e., suicide, work, and comorbidity related expenses) costs associated with depression or comorbid psychological and medical conditions. The literature has clearly shown that MDD represents a significant burden on individuals suffering from the disorder and society at-large.

Course

The course of MDD is characterized by different stages that occur within different phases of the disorder (Figure 1). As outlined by Bockting, Hollon, Jarrett, Kuyken, and Dobson (2015),

definitions for the stages of depression that occur within the phases of treatment have been proposed. The goal of the acute phase of treatment is to generate a treatment response, which is defined as a reduction in the severity of depressive symptoms (e.g., typically a 50.00% reduction compared to baseline). Over time, depressive symptoms begin to remit. The transition from the acute phase to the continuation phase of depression is marked by remission, which is defined as the absence of depressive symptoms for a period of time (e.g., typically two months) when an individual is considered to be generally well. Remission can be categorized as partial, unstable, or stable. Partial and unstable remission are characterized by the consistent (i.e., partial) or inconsistent (i.e., unstable) presence of some residual depressive symptom. During the continuation phase, depressive symptoms may increase to clinically significant levels. This phenomenon is characterized as relapse, which is defined as the reemergence of a depressive episode while an episode of depression is in remission. Alternatively, the transition from the continuation phase to the maintenance phase of depression is marked by recovery, which is defined as defined as the end of a depressive episode after a period of time (e.g., typically six to 12 months) when an individual is considered to no longer be depressed. During the maintenance phase, depressive symptoms may increase to clinically significant levels. This phenomenon is characterized as recurrence, which is defined as the experience of a new depressive episode after recovery from an episode. While these phases of depression are typically conceptualized within the stages of treatment, they can occur naturalistically without the influence of treatment.

Figure 1. *Diagram of the Stages of Depression from Bockting et al. (2015)*



The literature has consistently shown that MDD is marked by high rates of relapse and recurrence. Hardeveld, Spijker, De Graaf, Nolen, and Beekman (2010) conducted a systematic review of nine naturalistic studies that assessed the recurrence in MDD using a structured diagnostic interview. Prevalence rates for recurrence ranged from 21.00% to 40.00% within the first year, 42.00% to 75.00% within a five-year span, 67.00% within a 10-year span, and 35.00% to 85.00% within a 15-year span, indicating that recurrence is a commonly occurring phenomenon. These results have been replicated by multiple studies conducted over the past decade.

Poutanen and colleagues (2007) assessed the trajectory of depression in a sample of Finnish outpatients. Participants recruited from primary (n = 62) and psychiatric (n = 84) care settings were diagnosed with mild or severe depression using the Present State Examination interview. Depression status was re-assessed at a seven-year follow-up using the CIDI – Short Form. Results indicated that participants with mild and severe depression recruited from both settings showed elevated rates of depression during the follow-up period. Depression was present in 42.40% of participants with severe MDD and 48.30% of participants with mild MDD recruited from primary care settings. Similarly, depression was present in 61.50% of participants

with severe MDD and 68.50% of participants with mild MDD recruited from psychiatric care settings. While this study provides important information about the chronicity of depression across different healthcare settings, it does not decipher between relapse and recurrence rates.

Hardeveld, Spijker, De Graaf, Nolen, and Beekman (2013) assessed the recurrence of MDD in the Netherlands Mental Health Survey and Incidence Study (NEMESIS), a large community-based study of Dutch adults. Participants with a history of depression (n = 687) retrospectively reported the timing of their last depressive episode on the CIDI. Participants were longitudinally followed and depressive status was assessed after one and three years. The use of retrospective and longitudinal assessments enabled the calculation of cumulative recurrence rates, which spanned 20 years time. Results indicated that 19.70% of participants experienced a recurrent episode of depression during the follow-up period. Cumulative recurrence rates ranged from 2.50% at one year, 4.50% at two years, 13.20% at five years, 23.20% at 10 years, and 42.00% at 20 years, suggesting that the probability of recurrence of MDD increased as more time passed.

ten Doesschate, Bockting, Koeter, and Schene (2010) extended these findings by assessing the relapse and recurrence of MDD following treatment. Participants with a history of depression (n = 172) participated in a clinical trial of cognitive therapy (CT) or treatment as usual (TAU) for depression. Participants were assessed for depression at baseline and three, 12, 24, 36, and 66 months posttreatment using the Structured Clinical Interview for DSM-IV (SCID-I). Results indicated that across treatment conditions, the majority of participants (79.00%) experienced a relapse or recurrence of MDD over the 5.50-year follow-up period. While the authors did not specifically distinguish between relapse and recurrence, the results suggest that both phenomena commonly occur in individuals with a history of depression.

Johansson, Lundh, and Bjärehed (2013) replicated these results in a sample of Swedish outpatients who had successfully completed treatment. Participants with a history of depression (n = 51) who were in remission from MDD after exposure to psychotherapy or pharmacotherapy were recruited. Depression status was assessed at baseline and 12-month follow-up using the SCID-I. Results indicated that the majority of participants (61.00%) experienced a re-emergence of depression during the 12-month follow-up period, with no significant differences across groups based on the type of treatment modality. Recurrence (77.00%) was more common among these participants than relapse (23.00%), indicating that typically participants recovered from MDD posttreatment before experiencing a new depressive episode.

Together, these studies indicate that individuals with MDD experience high rates of relapse and recurrence. There is significant variability for the estimated occurrence of relapse and recurrence, with rates ranging from 2.50% to 85.00% over a period of one to 20 years. The majority of research has combined the terms relapse and recurrence, making it difficult to identify specific prevalence rate ranges for each term.

CHAPTER 2

SAD MOOD REACTIVITY AND VULNERABILITY TO DEPRESSION

Research clearly suggests that MDD is marked by high rates of relapse and recurrence and is typically viewed as a chronic mental illness (Richards, 2011). Most recent research estimates that half of individuals who experience a major depressive episode will experience a recurrence during their lifetime, with a subset of those individuals experiencing a relapsing-remitting trajectory marked by multiple episodes of depression (Monroe et al., in press).

Vulnerability Factors

Vulnerability to depressive relapse and recurrence likely reflects a complex interaction of biological, psychological, and environmental factors. Burcusa and Iacono (2007) completed a review of the literature to identify vulnerability factors for relapse and recurrence, which can be classified in different categories. Demographic vulnerability factors include female gender, lower socioeconomic status, and single relationship status. Clinical vulnerability factors include a higher number of previous depressive episodes, higher severity of first depressive episode, and the presence of comorbid pathology, especially other mood disorders. Familial vulnerability factors include a family history of psychopathology, especially depression or other mood disorders. Psychological vulnerability factors include negative cognitions and high levels of neuroticism. Psychosocial vulnerability factors include poor social support and exposure to stressful life events during childhood and adulthood. Recent studies on the prevalence of relapse and recurrence of depression have identified many of the same vulnerability factors, providing additional evidence for their predictive validity (Harveveld et al., 2010, 2013; Johansson et al., 2015; ten Doesschate et al., 2010).

Unfortunately, the majority of vulnerability factors that have been identified are not amenable to modification (e.g. female gender, multiple depressive episodes, stressful life events, etc.), which makes it difficult for medical and mental healthcare providers to prevent or intervene during the depressogenic cycle. Additional research is needed to identify and target malleable vulnerability factors that are related to increased risk for future episodes of depression. Two potentially malleable vulnerability factors that have been identified in the literature include cognitive and mood reactivity in response to a sad mood. The following section will review the theoretical models, experimental methodology, and empirical evidence related to cognitive and mood reactivity.

Cognitive Reactivity

Theoretical Models

Cognitive theories of depression propose that dysfunctional patterns of thinking represent a cognitive vulnerability that contributes to the etiology, maintenance, and reoccurrence of depression (Beck, 1967; Lau, Segal, & Williams, 2004; Scher, Ingram, & Segal, 2005). Indeed, a large body of empirical evidence has shown that currently, but not formerly, depressed individuals endorse elevated rates of dysfunctional thoughts (as reviewed by Teasdale, 1999). Interestingly, formerly depressed individuals remain at increased risk for depressive relapse or recurrence, despite no longer exhibiting cognitive vulnerability to depression while in a euthymic mood state. Several theoretical models have been proposed to account for differences in cognitive patterns observed between currently and formerly depressed individuals. In particular, the differential activation hypothesis and mood state dependent hypothesis have extended the cognitive model of depression originally proposed by Beck (1967) and suggest that maladaptive cognitions are dependent on an individual's current mood state.

The differential activation hypothesis. The differential activation hypothesis by Teasdale (1988) suggests that depression results in a myriad of cognitive changes that continue to persist after recovery from depression. Maladaptive cognitive patterns, including dysfunctional thinking and biased information processing, are activated by dysphoric mood. Over time, these cognitive patterns become associated with depressed mood and are hypothesized to maintain depressive symptoms in individuals with current MDD. According to this hypothesis, dysphoric mood can reactivate biased information processing and related cognitive patterns (e.g., dysfunctional beliefs) among individuals who have recovered from depression. In other words, whether dysfunctional thinking is activated among a formerly depressed individual depends on their current mood state. Formerly depressed individuals who are currently euthymic have low levels of dysfunctional thinking that resembles never depressed individuals. In contrast, formerly depressed individuals in a dysphoric mood state experience an increase in dysfunctional thinking similar to currently depressed individuals. The resurgence of maladaptive cognitive patterns during a dysphoric mood for formerly depressed individuals is hypothesized to increase the likelihood that an otherwise transient negative mood will develop into a depressive episode. Overall, this hypothesis proposes a cyclical relationship in which depressed mood leads to the activation of biased informational processing and dysfunctional thinking patterns that serve to maintain or initiate a depressed mood or depressive episode.

The differential activation hypothesis by Teasdale (1988) argues that maladaptive cognitive patterns are activated by dysphoric mood and perpetuate depressed mood. According to this theory, the maladaptive cognitive patterns associated with depression include a wide range of cognitive processes (e.g., biased attention, memory, and dysfunctional thinking patterns; as reviewed by Lau et al., 2004) that occur in individuals with both current or remitted MDD. A

similar theory, the mood state dependent hypothesis by Miranda and Persons (1988), focuses on a narrower range of maladaptive cognitive processes (i.e., dysfunctional beliefs only).

The mood state dependent hypothesis. The mood state dependent hypothesis by Miranda and Persons (1988) attempts to account for the differential patterns of dysfunctional thinking observed in currently and formerly depressed individuals. Previous research has shown that currently, but not formerly, depressed individuals endorse elevated levels of dysfunctional thinking. It was hypothesized that dysfunctional thinking is mood state dependent in formerly depressed individuals. Specifically, cognitive vulnerability in an individual with a history of depression is not explicitly present during a euthymic mood state but is evident when an individual is in a dysphoric mood state. According to this hypothesis, formerly depressed individuals exhibit cognitive reactivity, or a significant increase in dysfunctional thinking after exposure to a dysphoric mood. It is theorized that individuals with remitted MDD maintain latent cognitive vulnerabilities that are activated by a sad mood. Once activated, these patterns of dysfunctional thoughts are thought to increase risk for a subsequent depressive episode in individuals who have a history of depression.

The depression literature has examined the tenants of the differential activation and mood state dependent hypotheses. In the following sections, the experimental methodology used to investigate these theoretical models as well as the empirical evidence for these theoretical models is reviewed.

Mood Induction Procedures

Mood induction procedures induce a specific, transient mood state within an experimental setting, which enables researchers to investigate the cognitive, affective, and physiological factors that put an individual at risk for experiencing prolonged, maladaptive mood

states that are characteristic of psychopathology. Martin (1990) systematically reviewed the range of mood induction procedures that have been used in experimental psychology. While 14 different types of mood induction procedures have been empirically validated in the literature, this review will focus on music plus autobiographical recall procedure, which is most commonly used approach within the depression literature. During a music plus autobiographical recall mood induction, individuals listen to a piece of emotionally-valenced music and are instructed to recall an emotionally-valenced autobiographical memory to induce a particular mood. Research on negative affect would typically ask participants to think about a specific time in their life when they experienced sadness (i.e., autobiographical recall mood induction) while listening to a sad piece of non-lyrical music played at a slower rate (i.e., music mood induction).

Martin (1990) acknowledged that while there is not a universally accepted procedure for inducing transient mood states, the depression literature has identified the music plus autobiographical recall mood induction as an effective method for inducing a sad mood state. The sad music plus autobiographical recall mood induction has been compared to multiple self-report, behavioral, and performance-based measures. Overall, the sad music plus autobiographical recall mood induction has been shown to instate a transient despondent mood that is equivalent to an intermediate level of clinical depression in more than 75.00% of participants. In addition, the sad music plus autobiographical recall mood induction has been shown to induce a mood state that most closely resembles the cognitive, somatic, and emotional aspects of depression without the presence of residual anxiety. Of note, the transient mood state only persists for a few minutes, which indicates that these mood induction procedures are not only effective, but ethical. Overall, the sad music plus autobiographical recall mood induction

procedure has been empirically validated as a reliable and valid method for eliciting a sad mood within an experimental setting.

Empirical Evidence

Theoretical models have suggested that increases in maladaptive cognitive patterns in response to sadness contribute to the recurrence and maintenance of depression. Studies have assessed the presence of cognitive reactivity and its potential role as a predictor of relapse and recurrence. Cognitive reactivity is defined as the change in underlying negative cognitions in response to a sad mood induction. Cognitive reactivity is typically measured using the Dysfunctional Attitudes Scale (DAS), which is used to compare the change in dysfunctional beliefs assessed pre- and post-mood induction.

The differential activation and mood state dependent hypotheses have been investigated by a large body of literature. Studies have employed a variety of empirically-validated sad mood induction procedures to examine cognitive reactivity in participants with remitted MDD as these experimental procedures have been shown to transiently create the cognitive, somatic, and emotional experiences of depression in euthymic individuals. The majority of studies have used a combination of music and autobiographical recall to create a mild, transient sad mood (Fresco, Heimberg, Abramowtiz, & Bertram, 2006; Gemar, Segal, Sagrati, & Kennedy, 2001; Jarrett et al., 2012; Kuyken et al., 2010; Lau, Haigh, Christensen, Segal, & Taube-Schiff, 2012; Pfeiffer, Brockmeyer, Zimmermann, & Backenstrass, 2015; Segal, Gemar, & Williams, 1999; Segal et al., 2006; Van der Does, 2002, 2005). Though, some studies have used other empirically-supported techniques such as a combination of sad-valence music and self-statements (e.g., "I'm discouraged and unhappy about myself"; Dykman, 1997), a sad-valence film (i.e., clip of a son dealing with his father's death from *The Champ*; Brosse, Craighead, & Craighead, 1999;

Miranda, Gross, Persons, & Hahn, 1998), or a naturally occurring negative mood state (Miranda, Persons, & Byers, 1990; Roberts & Kassel, 1996).

In general, cross-sectional studies have shown that participants with remitted MDD report a significantly greater increase in dysfunctional attitudes following a negative mood state or after exposure to a sad mood induction compared to healthy control participants (Gemar et al., 2001; Lau et al., 2012; Miranda et al., 1990, 1998; Roberts & Kassel, 1996; Van der Does, 2002). However, some studies failed to find this association, with formerly depressed and never depressed participants reporting similar levels of dysfunctional attitudes following a negative mood state or after exposure to a sad mood induction (Brosse et al., 1999; Dykman, 1999; Fresco et al., 2006; Pfeiffer et al., 2015; Van der Does, 2005). Together, these results provide mixed evidence for the hypothesis that individuals with remitted MDD exhibit significantly more dysfunctional attitudes while in a dysphoric mood than their never depressed counterparts.

Longitudinal studies have investigated cognitive reactivity as a risk factor for relapse and recurrence of MDD. Segal and colleagues (1999) examined differences in cognitive reactivity between participants with remitted MDD who were successfully treated with cognitive-behavioral therapy (CBT; $n = 25$) or antidepressant medications ($n = 29$). At baseline, participants completed a sad mood music and autobiographical recall mood induction. One to five years later, participants were assessed for recurrence using the SCID-I. Results indicated that at baseline, formerly depressed participants treated with antidepressant medications reported a significantly greater increase in dysfunctional attitudes post-mood induction compared to those treated with CBT ($R^2 = .09$, $p < .05$). In addition, formerly depressed participants who reported an increase in cognitive reactivity post-mood induction were significantly more likely to experience a relapse during follow-up ($\chi^2 = 4.64$, $p < 001$). Results suggested that cognitive

reactivity may be a risk factor for subsequent relapse and proposed that CBT may be an efficacious treatment method for decreasing an individual's dysfunctional attitudes.

In a follow up study, Segal and colleagues (2006) sought to replicate the finding that the type of treatment for depression has an impact on subsequent cognitive reactivity. Participants with current MDD were recruited and randomly assigned to a treatment condition, including CBT (n = 88) or antidepressant medication (n = 56). After successful achieving remission from MDD, participants completed a sad mood music and autobiographical recall induction. 18-months later, participants were assessed for recurrence using the Longitudinal Interval Follow-Up Evaluation interview and Hamilton Depression Rating Scale (HDRS). Results indicated that formerly depressed participants who reported a significantly greater increase in dysfunctional attitudes following the mood induction were at greater risk for experiencing a relapse during the follow-up period ($\chi_1^2 = 7.12$, $p < .05$). Contrary to the Segal and colleagues (1999) findings, there was no significant difference in cognitive reactivity between treatment groups ($\chi_1^2 = .256$, $p > .05$), casting doubt on the hypothesis that CBT leads to a change in underlying dysfunctional beliefs. However, this study does provide additional support for the finding that the presence of cognitive reactivity predicts the recurrence of another depressive episode in participants with remitted MDD.

Kuyken and colleagues (2010) aimed to extend previous findings to a different type of psychotherapy: mindfulness-based cognitive therapy (MBCT). Participants in partial or full remission from recurrent MDD (i.e., three or more lifetime episodes of depression) were recruited and randomly assigned to a treatment condition, including MBCT plus discontinuation of antidepressant medication (n = 43) or maintenance of antidepressant medication (n = 37). After successful achieving remission from MDD, participants completed a sad mood music and

autobiographical recall induction. Fifteen months later, participants were assessed for recurrence using the SCID-I and depressive symptoms using the HDRS. Results indicated that formerly depressed participants who received MBCT reported a significantly greater increase in dysfunctional attitudes following the mood induction compared to formerly depressed participants who received antidepressant medication ($d = .47$, $p < .05$). While formerly depressed participants treated with antidepressant medications who reported increases in dysfunctional attitudes following the mood induction were at greater risk for elevated depressive symptoms and relapse during the follow-up period, this relationship was not found for participants who received MBCT ($\chi_1^2 = .01$, $p = .91$). This study provides additional support for the finding that the presence of cognitive reactivity predicts relapse in participants with remitted MDD and proposed that MCBT may be protect against future episodes of depression.

Jarrett and colleagues (2012) investigated the impact of cognitive reactivity on relapse and recurrence in formerly depressed participants who were at high risk for experiencing another depressive episode. Participants with recurrent MDD and elevated depressive symptomology ($n = 523$) who previously responded to CT were recruited and randomly assigned to an 8-month continuation treatment condition, including CT, antidepressant medication, or placebo. Before beginning continuation treatment, participants completed a sad mood music and autobiographical recall mood induction. Participants were assessed for relapse using the SCID-I and HRSD eight, 20, and 32-months after the start of continuation treatment. Contrary to previous research, there was no significant increase in dysfunctional attitudes following the mood induction ($p = .76$). While these results are not in line with the differential activation or mood state dependent hypotheses, are in line with previous research that has shown that participants who have received CBT exhibit less cognitive reactivity than participants who were treated with pharmacotherapy

(Segal et al., 1999). Additional analyses identified a relationship between unprimed dysfunctional attitudes and relapse of depression over time; formerly depressed participants in all conditions who endorsed higher dysfunctional attitudes pre-mood induction were at a greater risk for relapse at 20 ($\chi^2 = 3.93$, $p < .05$) and 32 ($\chi^2 = 4.49$, $p < .05$) months, regardless of posttreatment depressive symptom severity. Overall, this study suggests that the presence of dysfunctional attitudes during a euthymic, rather than dysphoric, mood state has negative implications for sustained remission in MDD.

The literature has found some support for the differential activation and mood state dependent hypotheses. Cross-sectional studies have generally suggested that individuals who have recovered from depression exhibit cognitive reactivity in response to a negative mood state or sad mood induction compared to individuals without a history of depression. In addition, longitudinal studies have shown that formerly depressed individuals who exhibit cognitive reactivity while euthymic or dysphoric have higher rates of relapse and recurrence over time. However, there are inconsistencies in this literature base. As a result, researchers have examined other forms of reactivity that may explain elevated rates of relapse and recurrence. In the following section, research examining mood reactivity in response to sadness will be reviewed.

Mood Reactivity

Theoretical Models

Some etiological theories of depression have focused on the experience of negative emotions, proposing that depressed individuals exhibit abnormal patterns of mood reactivity, or a significant change in mood state after exposure to a dysphoric mood. The cardinal symptoms of depression include sad, low mood and loss of interest or pleasure in activities that were previously enjoyable (APA$_A$, 2013). In this sense, depression is a disorder marked by low levels

of positive mood as well as high levels of negative mood (Rottenberg, Gross, & Gotlib, 2005b). Indeed, research has indicated that individuals with current MDD report fewer positive emotions and exhibit fewer positive responses to pleasurable stimuli. While depressed individuals have conventionally been thought to express more negative emotions, the empirical evidence is mixed. Some research has shown that individuals with current MDD exhibit greater responsivity to negative stimuli while other studies have found the opposite (Rottenberg, Gross, & Gotlib, 2005b). Theoretical models that have been proposed to explain these empirical findings include the positive attenuation, the negative potentiation, and the emotion context insensitivity hypotheses.

The positive attenuation hypothesis. The positive attenuation hypothesis proposes that currently depressed individuals exhibit a blunted emotional response to positively-valenced emotional stimuli. This hypothesis is primarily based on clinical observations of depression; the disorder is associated with symptoms related to reduced emotional (e.g., loss of interest), behavioral (e.g., psychomotor retardation), and physiological (e.g., reduced appetite, weight, and energy level) engagement that is adaptive and life sustaining (Rottenberg et al., 2005b). The literature has generally provided empirical support for the positive attenuation hypothesis. A meta-analysis by Bylsma, Morris, and Rottenberg (2007) found that positive emotional reactivity was lower for self-report ($p < .0001$, $d = -.70$) and behavioral ($p < .001$, $d = -.45$) measures in depressed participants compared to healthy control participants. Fewer studies have examined positive emotional reactivity using physiological methodology, resulting in similar results across depressed and never depressed participants ($p = .29$, $d = -.15$).

The negative potentiation hypothesis. The negative potentiation hypothesis proposes that currently depressed individuals exhibit an exaggerated emotional response to negatively-

valenced emotional stimuli. This hypothesis is also based on clinical observations of depression; the disorder is associated with symptoms related to negative mood states (e.g., depressed mood) and is often characterized by negative-valence behavioral reactions (e.g., crying and withdrawal; Rottenberg et al., 2005b). It has been theorized that negative mood states result in a cascade of cognitive changes that perpetuate depressogenic responsivity. As previously reviewed, the cognitive model of depression states that negative mood activates maladaptive cognitive patterns that lead to biased informational processing, dysfunctional thinking, and depressogenic behaviors (Beck, 1967). The cyclical relationship between depressogenic thoughts, feelings, and behaviors is hypothesized to initiate and maintain depression. The findings for the negative potentiation hypothesis have been inconsistent. For example, studies have found that currently depressed individual exhibit increased or decreased physiological reactivity to negatively-valenced stimuli (as reviewed by Rottenberg et al., 2005b). In addition, meta-analysis by Bylsma and colleagues (2007) found that negative emotional reactivity was lower for self-report ($p < .0001$, $d = -.36$) and physiological ($p < .05$, $d = -.22$) measures in depressed participants compared to healthy control participants. Results were less clear for negative emotional reactivity assessed by behavioral measures, resulting in non-significant differences between depressed and never depressed participants ($p = .54$, $d = -.05$). In general, the literature refutes the negative potentiation hypothesis and suggest that depressed individuals tend to exhibit blunted, rather than exaggerated, reactivity to negatively-valenced emotional stimuli.

The emotion context insensitivity hypothesis. The emotional context insensitivity hypothesis by Rottenberg and Gotlib (2004) builds upon the empirical findings related to the positive attenuation and negative potentiation hypotheses in proposing that depressed individuals exhibit blunted emotional reactivity in response to both positively and negatively-valenced

stimuli. The theory provides an evolutionary explanation to the emotional, behavioral, and physiological reactions observed in depressed individuals. It is theorized that depressed individuals disengage from positive and negative stimuli in their environment to protect themselves from potential danger. As a result, depressed individuals exhibit blunted emotional, behavioral, and physiological reactions in response to both positive and negative stimuli that are normative and idiographic in nature. This pattern of responsivity is not appropriate in relation to the environmental demands and results in a less adaptive response that is theorized to perpetuate depressive symptoms or lead to recurrence of depression.

While it was initially hypothesized that this pattern of responsivity would be observed in individuals with a history of depression, early empirical evidence (i.e., Rottenberg et al., 2005b) suggested that emotional, behavioral, and physiological reactivity is mood state dependent in formerly depressed individuals. Therefore, individuals who have a history of depression will exhibit blunted responsivity while in a dysphoric mood, but not during a euthymic mood. This pattern of responsivity is hypothesized to serve as a risk factor for experiencing a subsequent depressive episode. The depression literature assessing reactivity to sad mood induction procedures has primarily examined the emotion context insensitivity hypothesis. In the following section, empirical evidence supporting and refuting this theoretical model will be reviewed.

Mood Induction Procedures

Studies have typically obtained multiple measures of mood to assess how emotions changes in response to mood induction procedures. Within the cognitive reactivity literature, changes in mood have been employed as a manipulation check to ensure that the sad mood induction was in fact inducing a transient, dysphoric mood in participants. The aforementioned studies all found that formerly depressed and healthy controls participants endorsed an increase

in self-reported sad mood on the Visual Analogue Scale (VAS). Of the studies that examined group differences among formerly depressed and never depressed participants, none found significant variations in the degree of sadness endorsed by the two groups after exposure to the sad mood induction procedures (Brosse et al., 1999; Dykman, 1997; Fresco et al., 2006; Gemar et al., 2001; Lau et al., 2012; Miranda & Persons, 1988; Miranda et al., 1998; Segal et al., 2006; Solomon et al., 1998; Van der Does, 2002, 2005). These findings suggest that formerly depressed and healthy control participants show similar levels of mood reactivity in response to a sad mood induction.

Empirical Evidence

Given that some studies have failed to show cognitive reactivity to sadness in individuals with remitted depression, researchers examined other potential predictors of relapse and recurrence. Recent findings suggest that there may be a difference in mood reactivity among participants with remitted MDD. Mood reactivity is defined as the change in mood state in response to a sad mood induction. Mood reactivity is typically measured using the VAS, which compares an individual's mood state (i.e., happy, sad, depressed, etc.) pre- and post-mood induction

Before formally proposing the emotion context insensitivity hypothesis, Rottenberg, Kasch, Gross, and Gotlib (2002) investigated mood reactivity in currently depressed individuals. Depression was assessed using the SCID-I. Participants included individuals with current MDD (n = 72) and healthy control participants without a history of Axis I disorders (n = 33). Experimental procedures included a neutral film, two negative (i.e., sad and fear) films presented in a counterbalanced order and separated by an arithmetic task, and an amusing film. Self-report measures about emotional experiences were collected at baseline and after each emotional film.

Results indicated that participants with current MDD reported more sadness in response to the neutral ($R^2 = 25.11$, $p < .001$) and amusing ($R^2 = 8.25$, $p < .01$) films, but not the sad ($R^2 = 1.19$, $p > .10$) or fear ($R^2 = 2.05$, $p > .10$) films, compared to healthy control participants. In addition, participants with current MDD reported less amusement ($R^2 = 4.91$, $p < .05$) in response to the amusing film, but group differences were not statistically significant ($p > .05$). This study provided empirical evidence for the successive theory that currently depressed individuals exhibit inappropriate and insensitive mood reactivity to emotionally-valenced stimuli.

Rottenberg, Gross, and Gotlib (2005b) sought to extend these results to formerly depressed individuals. Depression was assessed using the SCID-I. Participants were individuals with current MDD ($n = 19$), remitted MDD ($n = 22$), and healthy control participants without a history of Axis I disorders ($n = 26$). Experimental tasks included normative and idiographic sad, happy, and neutral valenced films and imagery tasks (i.e., participants were instructed to create a visual picture in their mind of the previously watched film) presented in a counterbalanced order. Each film and imagery task were preceded by a one-minute resting baseline and followed by a one-minute filler task to reduce carry over effects. Results indicated that participants with current MDD reported similar levels of sadness ($p > .10$) and less happiness ($p < .001$) across all stimuli compared to the two other groups, suggesting that these participants did not respond to the emotional valence of the stimuli appropriately. Participants with current MDD who reported higher levels of sadness and lower levels of happiness in response to idiographic stimuli were more likely to have been depressed for a longer period of time. Participants with remitted MDD reported emotional (i.e., happy, amused, sad, and anxious) responses that were similar to healthy control participants ($p < .001$), indicating that both groups reported appropriate emotional reactions to emotionally-valenced stimuli. This led the authors to hypothesize that the emotion

context insensitivity hypothesis is mood state dependent, much like the previously reviewed cognitive vulnerabilities.

The literature has used sad mood induction procedures to investigate the applicability of the emotion context insensitivity hypothesis to formerly depressed individuals. Lethbridge and Allen (2008) examined if cognitive or mood reactivity predicted recurrence in participants with remitted MDD (n = 52). Depression was assessed with the SCID-I. At baseline, participants completed a sad mood music and autobiographical recall mood induction. Cognitive reactivity was assessed using the DAS while mood reactivity was assessed using the VAS. One year later, participants were assessed for recurrence using the SCID-I and reported stressful life events using the Stressful Life Events Questionnaire. In line with the cognitive reactivity literature, results showed that participants with remitted MDD reported significant decreases in happiness (t = 3.66-7.90, $p < .01$) and increases in sadness (t = -6.34--4.60, $p < .01$) on the VAS as well as significant increases in dysfunctional thinking (t = -43.83, $p < .01$) on the DAS post-mood induction. Mood reactivity on the happy scale of the VAS (i.e., decrease in happiness in response to the sad mood induction) and self-reported life stress was predictive of relapse. Formerly depressed participants who reported less decrease in happiness on the VAS or more life stress was significantly more likely to relapse at one-year follow-up. Mood reactivity on the depressed scale of the VAS (i.e., increase in depression in response to the sad mood induction) and cognitive reactivity on the DAS were both not predictive of relapse.

While these results suggest that blunted mood reactivity, rather than cognitive reactivity, in response to sadness predicts the recurrence of a new depressive episode in formerly depressed individuals, these findings are limited by methodological issues. First, the literature consistently uses a sad, rather than depressed, scale on the VAS. It is possible that participants with remitted

MDD reported less increase of negative mood on the depressed scale of the VAS than the sad scale of the VAS. These participants have experienced what a depressive episode is like, so they may be less likely to endorse a mood state of "depressed" when encountered with a transient, sad mood. Second, studies (i.e., van Rijsbergen et al., 2013) have hypothesized that it may be necessary to assess cognitive reactivity at multiple time points to examine its impact of MDD relapse and recurrence. This study conducted the sad mood music and autobiographical recall mood induction at a single time point, rather than at baseline and follow-up. Therefore, results from this study may have differed based on the methodological procedures that were employed.

van Rijsbergen and colleagues (2013) examined whether changes in cognitive or mood reactivity predicted relapse in participants with remitted MDD after treatment. Participants with remitted MDD were recruited and randomly assigned to a relapse prevention condition, including preventive CBT and TAU (n = 84) or TAU alone (n = 88). Participants completed the sad music and autobiographical recall mood induction at baseline and posttreatment. Cognitive reactivity was assessed using the DAS while mood reactivity was assessed using the VAS. Participants were assessed for relapse using the SCID-I at three, 12, 24, 36, and 66 months follow-up. Results showed that participants in all conditions who endorsed higher DAS scores before the mood induction were at a greater risk for relapse ($\chi^2 = 12.29$, $p < .001$), indicating that unprimed dysfunctional attitudes predicted relapse 5.50 years later. However, pre ($\chi^2 = 1.14$, $p = .29$) and posttreatment ($\chi^2 = 2.10$, $p = .15$) cognitive reactivity was not predictive of relapse. While this finding is contradictory to some of the literature (i.e., Kuyken et al., 2010; Segal et al., 1999, 2006), it is in line with the results obtained by Jarrett and colleagues (2012).

van Rijsbergen and colleagues (2013) also found that participants in all conditions who endorsed exhibited mood reactivity were at greater risk for relapse, indicating that mood

reactivity predicted relapse 5.50 years later. Of note, this relationship was only found for mood reactivity assessed posttreatment ($\chi^2 = 8.29$, $p = .004$), but not pretreatment ($\chi^2 = .06$, $p = .81$). Given that previous research has suggested that CBT reduces dysfunctional beliefs (Kuyken et al., 2012; Segal et al., 1999), exploratory analyses were conducted to see if there was a change in cognitive and mood reactivity before and after participation in the CBT relapse prevention condition. Results revealed that an increase in cognitive ($\chi^2 = 6.77$, $p = .01$) and mood ($\chi^2 = 6.85$, $p = .01$) reactivity posttreatment was predictive of relapse over the 5.50-year follow-up period. This perplexing finding lead the authors to suggest that cognitive reactivity may need to be assessed at multiple time points over the course of treatment in order to detect its true effect of relapse. Overall, these results suggest that mood reactivity predicts relapse over time but does not discount the role of cognitive reactivity in relapse prediction.

A growing body of research has supported the emotion context insensitivity hypothesis, indicating that currently depressed individuals exhibit blunted emotional, behavioral, and physiological reactivity in response to both negatively and positively-valenced stimuli (Bylsma et al., 2007; Rottenberg & Hindash, 2015). However, the applicability of the emotion context insensitivity hypothesis to formerly depressed individuals is less clear. A very limited literature base has suggested that blunted (i.e., Lethbridge & Allen, 2008) or exaggerated (i.e., van Rijsbergen et al., 2013) mood reactivity is predictive of another depressive episode. While these findings are important, they have not completely discounted the role that cognitive reactivity may play. In addition, cross-sectional research has not found differences in mood reactivity among formerly depressed and never depressed participants (Brosse et al., 1999; Dykman, 1997; Fresco et al., 2006; Gemar et al., 2001; Lau et al., 2012; Miranda & Persons, 1988; Miranda et al., 1998; Solomon et al., 1998; Van der Does, 2002, 2005), which suggests that mood reactivity

may not be measured properly by single session studies. Additional research is needed to examine whether formerly depressed individuals exhibit cognitive or mood reactivity in response to sadness and whether either form of reactivity contributes to relapse and recurrence of MDD.

Multiple mechanisms have been proposed to account for vulnerability to depressive relapse. While some risk factors are trait dependent (e.g., age of onset, severity of the first episode, number of symptoms, etc.; Burcusa & Iacono, 2007), theoretical models and empirical evidence has suggested that some risk factors may be state dependent. Cross-sectional research has shown that individuals with remitted MDD endorsed more dysfunctional thoughts (Gemar et al., 2001; Lau et al., 2012; Miranda et al., 1990, 1998; Roberts & Kassel, 1996) in response to a sad mood induction. Longitudinal research has suggested that individuals with remitted MDD who report elevated cognitive (Kuyken et al., 2010; Segal et al., 1999, 2006) or blunted (i.e., Lethbridge & Allen, 2008) or exaggerated (i.e., van Rijsbergen et al., 2013) mood reactivity in response to sadness are more likely to experience another depressive episode over time. While there is currently disagreement in the literature about whether cognitive or mood reactivity are markers of vulnerability for relapse in remitted MDD, there is clear support for the notion that such vulnerabilities are mood state dependent in remitted depression.

One way to advance our understanding of the relationship between reactivity to negative affect and vulnerability to depression is to move beyond self-report and examine cardiovascular reactivity in response to sadness. The next section will provide an overview of the physiological systems and psychophysiological markers related to cardiovascular functioning. Several theoretical models, and empirical evidence related to cardiovascular functioning, and negative affect including depression will be reviewed.

CHAPTER 3

CARDIOVASCULAR SYSTEM

The human body is composed of numerous systems that regulate bodily functions, maintain homeostasis, and enable an individual to respond to environmental stimuli. Multiple systems and organs play a role in the regulation of the cardiovascular system. The components that are most relevant to the cardiovascular system includes the nervous system and the heart.

The Nervous System

As outlined by Porges (1992), the nervous system is the executory structure responsible for communicating information from the brain and spinal cord via the central nervous system to the rest of the body via the peripheral nervous system. The peripheral nervous system is further branched into the somatic nervous system and the autonomic nervous system (ANS). The ANS is made up of two distinct systems: the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). The SNS is responsible for preparing and mobilizing the body to react to external stimuli presented within the environment, commonly referred to as the fight or flight response. The PNS is responsible for demobilizing the body and returning it to baseline functioning, also known as the relaxation and restoration response. The SNS and the PNS enable the coordination of bodily reactions in response to internal and external stimuli through contradictory, but complementary functions. The PNS is mainly responsible for maintaining homeostasis, or dynamic regulation of the internal organs to preserve or restore equilibrium, while the SNS is mainly responsible for reacting to stress, or an interruption in homeostasis.

While both branches of the ANS play an important role in cardiovascular functioning, psychological research primarily focuses on the activity of the PNS. Focus on the PNS is due to the fact that many of the cardiovascular measures are primarily under parasympathetic control

(i.e., heart rate (HR); Grossman & Taylor, 2007) or are thought to reflect parasympathetic control (i.e., respiratory sinus arrhythmia (RSA); Allen, Chambers, & Towers, 2007).

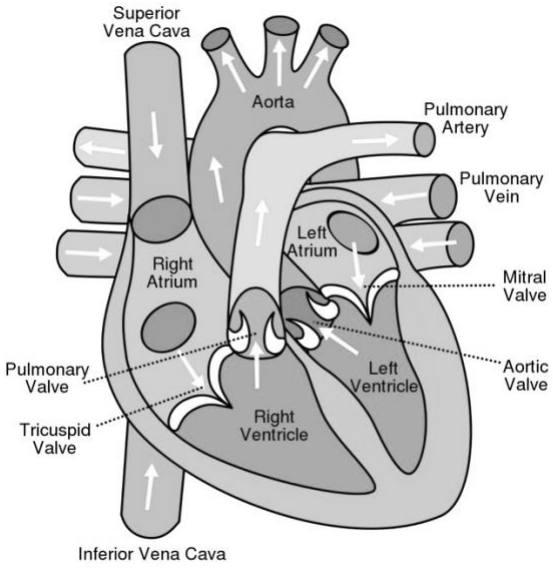
The Heart

The heart is a muscular organ located within the chest cavity that pumps blood throughout the body. The ANS is fundamental to cardiovascular functioning. The heart is connected to the ANS via the vagus nerve, one of the 12 cranial nerves that branch out from the brain to the body. The vagus nerve innervates the sinoatrial (SA) node and is responsible for determining the rate at which the heart beats; the SNS accelerates the heart while the PNS decelerates the heart (Porges, 1992).

The major structural components of the heart (Figure 2) include the chambers, valves, and nodes. As detailed by Katz (2010), the heart consists of four chambers, the left atrium, right atrium, left ventricle, and right ventricle, and two classes of valves, the atrioventricular (AV) valves and semilunar valves. The AV valves, including the tricuspid valve on the right side of the heart and the bicuspid valve on the left side of the heart, separate the atria from the ventricles. The semilunar valves, including the pulmonary valve on the right side of the heart and the aortic valve on the left side of the heart, separate the ventricles from the pulmonary artery or aorta. The heart contains two clusters of cells that control electrical impulses in the heart, the SA and the AV nodes. The SA node, located in the right atrium of the heart, is innervated by the vagus nerve and generates the electrical impulses that cause the contraction of the atrial muscles. The AV node, located in the center of the heart between the atria and ventricles, receives the electrical impulses from the SA node then regulates and transports the electrical impulses to the ventricles, which causes the contraction of the ventricular muscles. The heart contains two separate systems

of pumps on the left and right side that work in concert with one another. Similar processes occur within each side of the heart during the cardiac cycle.

Figure 2. *Diagram of the Heart (n.d.)*



The Cardiovascular System

The cardiovascular system is a complex structure that consists of the heart and vasculature that extends throughout the entire body. The cardiovascular system regulates the circulation of blood within the body, which follows a sequence of steps outlined by Berntson, Quigley, and Lozano (2007). Deoxygenated blood travels through veins from the organs and extremities to the heart. Deoxygenated blood enters the right atrium via the superior and inferior vena cava, passing through the tricuspid valve into the right ventricle. Deoxygenated blood then passes through the pulmonary valve into the pulmonary artery, which is connected to the lungs. Blood is circulated through capillaries in the lungs, enabling the absorption of oxygen and release of carbon dioxide. Oxygenated blood enters the left atrium via the lungs and pulmonary vein, passing through the mitral valve into the left ventricle. Oxygenated blood then passes through the aortic valve into the aorta. Oxygenated blood travels through arteries to the organs

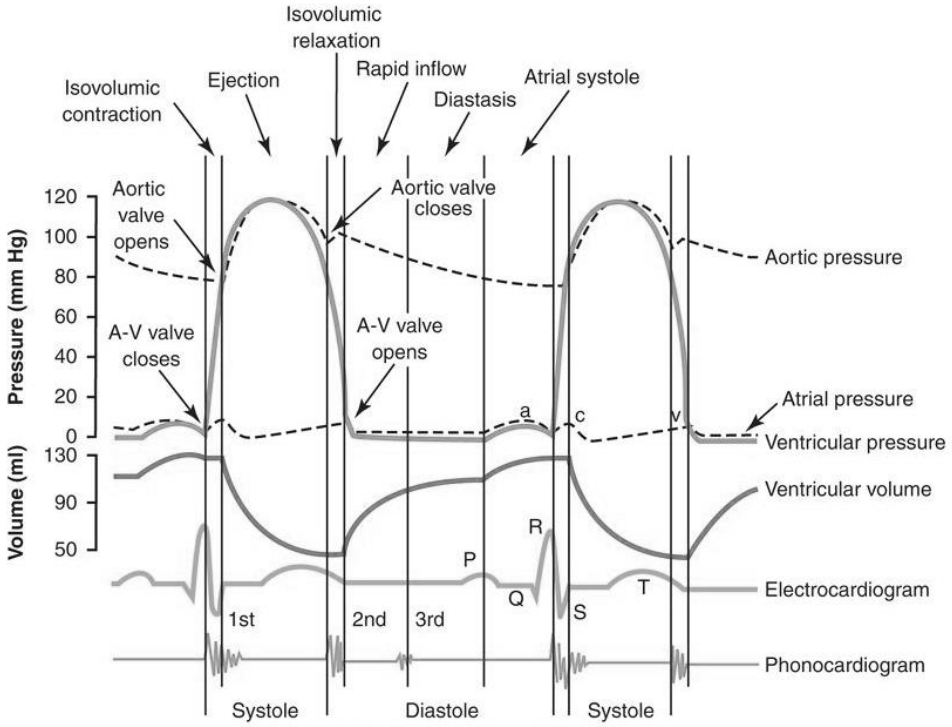
and extremities. Oxygenated blood is transported throughout the entire body via the vasculature and circulated through the capillaries in the trunk and extremities, enabling the release of oxygen and absorption of carbon dioxide. This sequence of steps occurs continuously, enabling the circulation of blood throughout the body.

The Cardiac Cycle

The cardiac cycle (Figure 3) represents the mechanical and electrical activity of the cardiovascular system that occurs during a single heartbeat. The cardiac cycle, as outlined by Berntson and colleagues (2007), includes two distinct phases: systole and diastole. Systole represents the contraction of the heart while diastole represents the relaxation of the heart. During the diastole phase, the heart is relaxed and the AV valves are open. The atria and ventricles fill with blood, resulting in an increase in the volume of blood in the ventricles. Depolarization of the SA node occurs in the right atrium and passes through the atrial muscle, which is represented on an electrocardiogram (ECG) as the P wave. Depolarization of the SA node causes the atrial muscles to contract. Pressure in the atria and ventricles increases, which causes the remainder of blood to flow into the ventricles. Depolarization of the AV node occurs in the center of the heart near the tricuspid valve and causes the ventricle muscles to contract, which leads to the closure of the AV valves. Together, this is represented on an ECG as the QRS complex. This marks the end of the diastole phase and the beginning of the systole phase. During the systole phase, pressure in the ventricles increases. The increase in ventricular pressure compared to pulmonary and aortic pressure leads to the opening of the semilunar valves. Blood is ejected through the pulmonary artery and aorta and the semilunar valves close. Pressure in the ventricles decreases, resulting in the repolarization of the ventricles, which is represented on an ECG as the T wave. This marks the end of the systole phase and the beginning of the next

cardiac cycle. This sequence of steps occurs continually in the heart so that blood can be pumped throughout the body.

Figure 3. *Diagram of the Cardiac Cycle from Berntson et al. (2007)*



Cardiovascular Markers

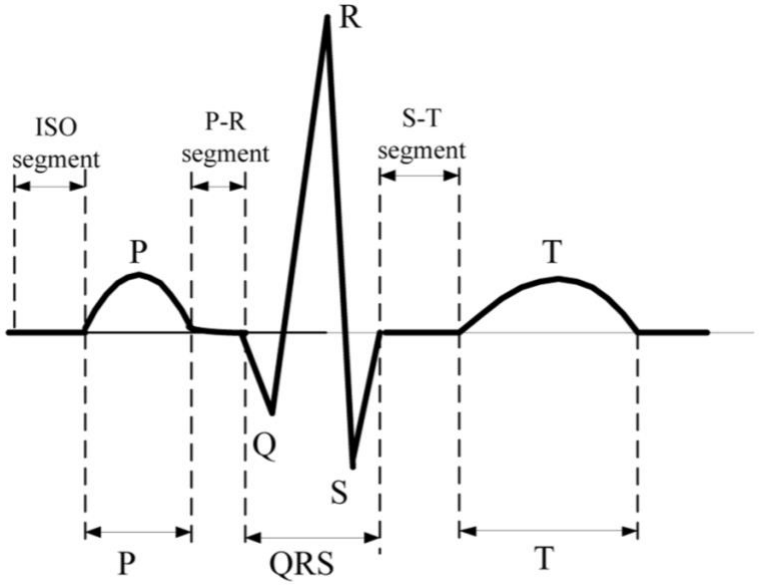
There are multiple cardiovascular markers that can be used to measure cardiovascular functioning. The cardiovascular measures of interest relevant for this overview include HR, heart period (HP), heart rate variability (HRV), RSA, cardiac output (CO), and pre-ejection period (PEP), which are obtained through ECG or a combination of ECG and impedance cardiography (ICG) as described below.

ECG

ECG (Figure 4) is a noninvasive technique for measuring the electrical activity of the heart. ECG is obtained through the application of noninvasive electrode sensors placed on the chest only or chest, legs, and arms. ECG measures the rate at which the heart beats as well as

certain electrical events that occur during the cardiac cycle (e.g., atrial and ventricular depolarization or ventricular repolarization). The ECG waveform is a visual representation of the electrical activity occurring in the heart, including the P wave, QRS complex, and T wave. The P wave represents atrial depolarization. The QRS complex represents ventricular depolarization. The T wave represents ventricular repolarization. In addition, the ECG waveform provides information on the speed of HR as well as the speed, magnitude, and direction of electrical events (Katz, 2010).

Figure 4. *ECG Waveform from Liang, Zhang, Tan, & Li (2014)*



HP and HR. HP is defined as the amount of time between heart beats measured in millisecond. For this investigation, HP will be used instead of HR, which is defined as the number of beats produced by the heart per minute. While HP and HR are reciprocal measurements of cardiovascular functioning, they are not linearly related and can generate discrepant results when there are significant differences across participants or changes within participants. Accordingly, research has indicated that HP should be used when changes in

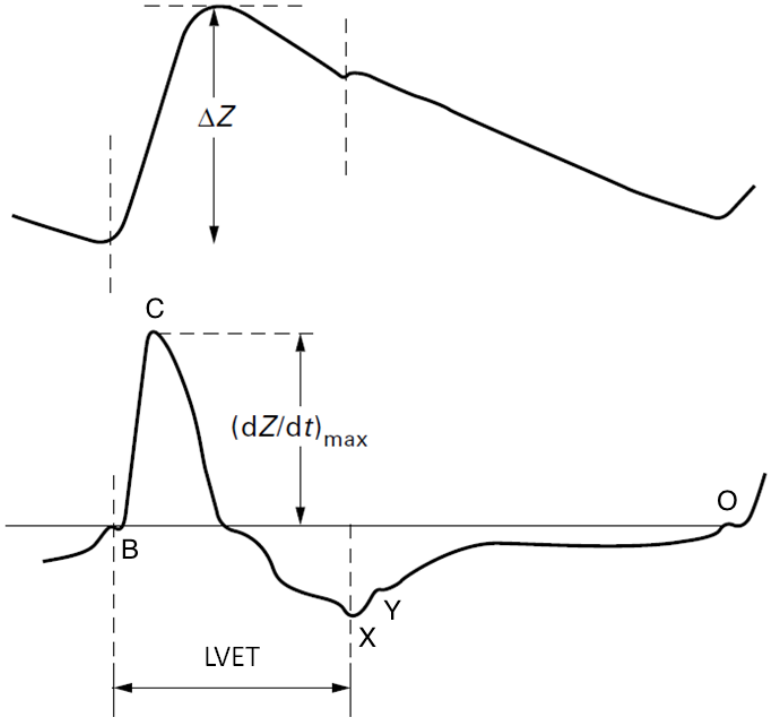
cardiovascular functioning are thought to be attributed to autonomic effects or differ significantly due to experimental tasks or group membership (Berntson et al., 2007).

ECG is used to assess HP, which is calculated by determining the interbeat interval (IBI) between successive R spikes on the ECG in milliseconds. Research has suggested that higher HP during experimental procedures that require attention indicates that an individual is attending to the stimuli that are presented in the environment. HP has been used in the literature as an index of arousal, task involvement, and mental load and effort (Jorna, 1992).

ICG

Similar to ECG, ICG (Figure 5) is a noninvasive technique for measuring the electrical activity of the heart. ICG can be obtained through the application of noninvasive electrode sensors placed on the chest and back. ICG measures changes in blood flow and vascular contraction throughout the chest cavity via resistance to electrical signal. ICG must be used in concert with ECG, as the ECG waveforms are used as a reference for the timing of the cardiac cycle (Berntson et al., 2007). The ICG waveform is a visual representation of electrical and mechanical events that occur in the heart, including the B, C, X, Y, and O points. The B point represents the opening of the aortic valve while the X point represents the closing of the aortic valve. The C point marks the peak of blood flow through the aorta. The Y point signifies the closing of the pulmonary valve. The O point indicates the closing of the mitral valve. In addition, the ICG waveform provides information on the speed of mechanical events that occur in the heart, volume of blood pumped through the heart, and amount of resistance exerted on the blood vessels (Berntson et al., 2007).

Figure 5. *ICG Waveform from Critchley (2013)*



HRV. HRV is defined as the beat-to-beat variability in HR and can be assessed using a combination of ECG and ICG. HRV can be determined via the time domain method, which calculates the IBI between successive R spikes on the ECG based on time. Standard Deviation of the Normal-to-Normal (SDNN) examines the IBI across a specific period (e.g., 24 hours), which provides a more comprehensive representation of variability with HRV (Carney et al., 2000). Root Mean Square Successive Difference (RMSSD) examines the IBI during a short time span, which provides a better representation of short-term changes in HRV (Carney et al., 2000). HRV can also be assessed via the frequency domain method, which calculates the IBI between successive R spikes on the ECG within certain frequencies. High frequency HRV (HF-HRV) examines the IBI within the high frequency band (.15-.40 Hertz), which takes respiration into account and approximates the amount of control the PNS exerts over the heart (Berntson et al., 1997, 2007). Changes in respiration impact HF-HRV as HR naturally accelerates during

inhalation and decelerates during exhalation. Consequently, HF-HRV represents vagal modulation, rather than vagal tone (Shaffer & Ginsberg, 2017). Low frequency HRV (LF-HRV) examines the IBI within the low frequency band (.05-.15 Hertz), which is thought to be impacted by both sympathetic and parasympathetic rhythms (Berntson et al., 1997, 2007).

Several less commonly used measures of HRV measures rely on the frequency domain method. For example, the LF/HF ratio is thought to approximate the degree of sympovagal balance (Vaccarino et al., 2008), but a significant body of evidence contradicts this hypothesis (Billman, 2013). Total power HRV (TP-HRV) assesses the IBI within the entire frequency band (< .40 Hertz), very low frequency HRV (VLF-HRV) assesses the IBI within a lower frequency band than LF-HRV (.0033-.039 Hertz), and ultra-low frequency HRV (ULF-HRV) assesses the IBI within the lowest frequency band (< .003 Hertz; Berntson et al., 1997; Vaccarino et al., 2008). While these measures are thought to be representative of both sympathetic and parasympathetic rhythms, they are not well characterized due to their limited use (Berntson et al., 1997, 2007).

RSA. RSA is defined as the beat-to-beat variability in HR during the respiration cycle. A combination of ECG and ICG can be used to assess RSA, which is calculated using the frequency domain method. The frequency domain method converts HP from time-domain to frequency-domain then calculates the IBI between successive R spikes on the ECG within the high frequency band (.15-.40 Hertz; Allen et al., 2007). Research has suggested that higher RSA is generally desirable as it indicates that an individual can flexibly respond to environmental stimuli (Berntson et al., 2007).

RSA sampled within the high frequency band is theorized to reflect the influence of the PNS on HR via the vagus nerve, which affects HR acceleration and deceleration during

respiration (Berntson et al., 1997, 2007). Greater parasympathetic input is thought to result in more acceleration of cardiac activity during respiration and more deceleration of cardiac activity after expiration, resulting in variable intervals between the heartbeats. Consequently, RSA has been used in the literature as an index of cardiac vagal control, which approximates the amount of control the PNS exerts over the heart (Grossman & Taylor, 2007). RSA is equivalent to the cardiac measure of high-frequency heart rate variability (HF-HRV) and the two terms are often used interchangeably in the literature (Allen et al., 2007).

CO. CO is defined as the volume of blood pumped by the heart per minute. CO is not only influenced by the rate at which the heart beats, but changes in the contractility, preload, and afterload of the cardiovascular muscles (Vincent, 2008). A combination of ECG and ICG are used to assess CO, which is calculated by multiplying HR and stroke volume (SV; CO = HR X SV), or volume of blood pumped through each ventricle per minute. Changes in CO is primarily controlled by HR as SV remains consistent across time. CO has been used in the literature to represent the efficiency of the heart. Research has suggested that higher CO indicates that the heart is functioning in an efficient manner (Berntson et al, 2007).

PEP. PEP is defined as the amount of contractile force produced by the heart. CO is influenced by changes in the contractility of the cardiovascular muscles (van Lien, Schutte, Meijer, & de Geus, 2013). PEP is calculated by determining the amount of time between the Q wave on an ECG, which represents the beginning of ventricular depolarization, and B on an ICG, which represents the opening of the aortic valve and beginning of ejection.

PEP is hypothesized to reflect the influence of the SNS on the heart (Berntson et al., 2007; van Lien et al., 2013). Research has suggested that higher PEP during experimental procedures that involve stress-based tasks indicates greater sympathetic control over the heart

and is associated with negative physiological responses (e.g., increase in cortisol and Hypothalamic-Pituitary-Adrenal (HPA) axis activation; as reviewed by Uchino, Smith, Holt-Lunstad, Campo, & Reblin, 2007). Consequently, PEP has been used in the literature as an index of sympathetic cardiac control, which approximates the amount of control the SNS exerts over the heart (Berntson et al., 2007).

In summary, the ANS is made up of two distinct systems that serve contradictory, but complementary, purposes: the SNS and the PNS. The SNS, or fight or flight response, is responsible for preparing and mobilizing the body to react to external stimuli presented within the environment. The PNS, or relaxation and restoration response, is responsible for demobilizing the body and returning it to baseline functioning. The heart is connected to the ANS via the vagus nerve, which connect the brain and the body. Consequently, the ANS is fundamental to and influential on cardiovascular functioning.

There are multiple cardiovascular markers that can be used to quantify cardiovascular functioning. The cardiovascular measures that were assessed in this investigation include HP, RSA, CO, and RSA, which are obtained through ECG or a combination of ECG and ICG. Each cardiovascular marker has been used as an index of physiological functioning. Higher HP during experimental tasks that involve attention is believed to be adaptive, as it is thought to represent arousal, task involvement, and mental load and effort. Higher RSA is hypothesized to be adaptive and has been used index of cardiac vagal control, which approximates the amount of control the PNS exerts over the heart. Higher CO is thought to be adaptive, as it has been used to represent the efficiency of the heart. Finally, lower PEP during experimental tasks that induce stress is hypothesized to be adaptive and has been used index of cardiac vagal control, which approximates the amount of control the SNS exerts over the heart.

Theoretical Models

Several theoretical models have been proposed to account for individual differences in cardiovascular functioning. Generally, these theories characterize patterns of cardiovascular functioning as adaptive or maladaptive. Relevant theoretical models include the polyvagal theory, biopsychosocial model of challenge and threat, and hawk-dove model.

The polyvagal theory. The polyvagal theory by Porges (1995) focuses on the impact of the ANS on physiological, psychological, and behavioral processes. The ANS contains two opposing systems: the sympathetic-adrenal system and the vagus system. The sympathetic-adrenal system mobilizes the body through the activation of SNS activity. The vagus system is further branched into two subsystems: the ventral vagal complex and the dorsal vagal complex. The ventral vagal complex contains myelinated vagal pathways that demobilizes the body through the inhibition of SNS activity, which is referred to as the vagal brake. The vagal brake activates the vagus nerve to reduce HR and blood pressure (BP), which produces a calming, restorative response that is adaptive (Porges, 2007). The dorsal vagal complex contains unmyelinated vagal pathways that immobilize the body through the inhibition of SA node, which is referred to as the dorsal vagal surge. The dorsal vagal surge also activates the vagus nerve to reduce HR and BP but is significantly more suppressing as it results in a shutdown physical and behavioral responsivity (Porges, 2001).

The polyvagal theory provides an evolutionary explanation to explicate the dynamic relationship between physiological, psychological, and behavioral processes. Each physiological state is characterized by a pattern of physical, psychological, and behavioral reactivity. More specifically, the sympathetic-adrenal system results in active avoidance, which include behavioral responses such as of fighting, escaping, or freezing. In addition, the sympathetic-

adrenal system is characterized by vagal withdrawal, or reduced RSA during an attention-demanding task compared to RSA at rest (Porges, 2007). The ventral vagal complex results in social engagement, which includes facial expressions, vocalizations, eye contact, head orientation, and other communicative behaviors. In addition, the ventral vagal complex is characterized by vagal augmentation, or increased RSA during an attention-demanding task compared to RSA at rest (Porges, 2007). The dorsal vagal complex results in behavioral immobilization, which includes passive avoidance, dissociation, and collapse. Of note, a valid cardiovascular index does not yet exist for the dorsal vagal complex as the system has less impact on the rate at which the heart pumps (Chapleau & Sabharwal, 2015). The polyvagal theory proposes that the underlying neurobiological structures are responsible for determining physiological, psychological, and behavioral response.

The polyvagal theory has been applied to the study of depression. Depression is a disorder characterized by maladaptive patterns of social, emotional, and behavioral responding. More specifically, research has shown that participants with current MDD exhibit deficits in social engagement (e.g., withdrawal from and impairment in social relationships) as well as emotional (e.g., flat affect) and behavioral (e.g., reduced startle response) inflexibility (Rottenberg, 2007b). Research has also identified some cardiovascular differences in depressed individuals that are in line with the polyvagal theory. Participants with current (Rottenberg, Wilhelm, Gross, & Gotlib, 2003) MDD show blunted RSA reactivity when crying in response to a sad film. In addition, depressed participants who exhibit this maladaptive pattern of cardiovascular reactivity report lower rates of recovery over time (Rottenberg, Salomon, Gross, & Gotlib, 2005a; Panaite et al., 2016). Empirical investigations of the polyvagal theory have found that infants and children who exhibit blunted RSA in response to socially-engaging, stress-

inducing, or attention-demanding tasks show maladaptive patterns of social, emotional, and behavioral responding (e.g., DiPietro, Porges, & Uhly, 1992; Huffman et al., 1998; Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996; Stifter & Corey, 2001; Stifter & Fox, 1990). Together, these studies suggest that the polyvagal theory can inform our understanding of cardiovascular reactivity in response to sadness in depressed populations.

The biopsychosocial model of challenge and threat. The biopsychosocial model of challenge and threat by Blascovich and Tomaka (1996) proposes a physiological basis for psychological states experienced in response to stressors. The model is typically examined within a goal-relevant situation, which can include motivated performance situations that require attention and cognition or passive situations that are attention demanding, but not cognitively draining. The goal-relevant situation leads to physiological and emotional responses, which are influenced by a combination of biological, physiological, cognitive, and interpersonal factors. The perception of goal-relevant situations is impacted by two components of cognitive appraisal: primary and secondary appraisal. Primary appraisal is defined as the amount of demands required by situation while secondary appraisal is defined as the amount of personal resources one has within a situation. Primary and secondary appraisals influence how an individual perceives a goal-relevant situation. When an individual perceives that he or she has the personal resources necessary to surmount the situational demands, the situation is viewed as a challenge. Conversely, when an individual perceives that he or she does not have the personal resources to meet the situational demands, the situation is viewed as a threat.

As outlined by Mendes, Major, McCoy, and Blascovich (2008), challenge and threat responses are theorized to result in differential patterns of performance and emotional and cardiovascular reactivity. Challenge results in an increase in performance on goal-relevant tasks,

which is associated with approach toward the task. Emotional reactions associated with challenge include positively-valenced emotions such as confidence and pride as well as externalized negatively-valenced emotions such as anger. The pattern of cardiovascular reactivity associated with challenge includes increased HR, CO, and vasoconstriction (VC) and decreased total peripheral resistance (TPR). Contrariwise, threat is associated with a decrease in performance on goal-relevant tasks, which is associated with avoidance of, vigilance towards, or feelings of defeat related to the task. Emotional reactions associated with threat include internalized negatively-valenced emotions such as shame and anxiety. The pattern of cardiovascular reactivity associated with threat includes increased HR, VC, and TPR and no change in CO. The biopsychosocial model of challenge and threat proposes that the perception of a situation influences physiological, psychological, and behavioral responsivity.

The biopsychosocial model of challenge and threat has been examined in relation to depression. Research has shown that participants with current MDD exhibit lower HR, HRV, RSA, and CO (Bylsma, Salomon, Taylor-Cliff, Morris, & Rottenberg, 2014; Salomon, Clift, Karlsdóttir, & Rottenberg, 2009; Salomon, Bylsma, White, Panaite, & Rottenberg, 2013) when exposed to stressors. While these findings are not in line with the patterns of cardiovascular reactivity hypothesized by the biopsychosocial model of challenge and threat, they do suggest an atypical pattern of cardiovascular reactivity in response to stress among currently depressed individuals.

The biopsychosocial model of challenge and threat has been explored in depressed populations within the context of stress, but not sadness. It is possible that individuals with current and remitted MDD show similar cardiovascular responses in stressful and sad contexts as

individuals may perceive that they have low personal resources in both types of high-demands situations.

The hawk-dove model. The hawk-dove model by Smith (1982) provides an evolutionary account of behavioral and physiological differences in response to stress. The model focuses on the concepts of allostasis, or the process of attaining homeostasis through the regulation of internal processes in response to external stressors, and allostatic load, or the impact of allostatic regulation on the body. The model proposes two architypes that represent an individual's typical response to allostasis: "hawks" and "doves" (Korte, Koolhaas, Wingfield, & McEwen, 2005). Individuals who are categorized as hawks exhibit aggressive, intrepid behavior. Their typical behavioral response is to either react or run away, which results in limited behavioral flexibility. Conversely, individuals who are categorized as doves exhibit non-aggressive, vigilant behavior. Their typical behavioral response is to freeze or hide, which results in high levels of behavioral flexibility. The behavioral patterns across groups are hypothesized to reflect differences in underlying physiology (Korte et al., 2005). More specifically, hawks show elevated sympathetic reactivity and reduced parasympathetic reactivity while doves show elevated parasympathetic reactivity and reduced sympathetic reactivity. These differences in autonomic reactivity are thought to result in varied cardiovascular responsivity, with hawks exhibiting increased sympathetic activity as indexed by lower HRV and doves exhibited increased parasympathetic activity as indexed by higher HRV (Korte et al., 2005).

The hawk-dove model has been extended to the study of depression. As reviewed by Korte and colleagues (2005), allostatic load appears to have distinct effects on individuals categorized as "hawks" or "doves." Hawks have been found to have higher rates of coronary heart disease and atherosclerosis due to elevated testosterone and BP and dominance of

sympathetic activity. Dominance of the sympathetic activity also impacts the immune system; reduced activity of the HPA axis leads to a hyper-immune state characterized by excessive inflammation and autoimmune responsivity. This physiological pathway is thought to contribute to the higher rates of atypical depression in hawks, which is characterized by increased appetite, weight gain, sleep, and social withdrawal. Doves more commonly show bradyarrhythmia, or an abnormal heart rhythm, due to dominance of parasympathetic activity. In addition, doves have been found to have higher rates of hypertension and atherosclerosis because of a cascade of physiological events; increased levels of cortisol lead to an increase in fat deposits, which results in elevated sympathetic activity. Neural differences in doves are thought to increase activity of the HPA axis and sympathetic system, contributing to the higher rates of melancholic depression, which is characterized by decreased appetite, weight, and sleep and increased feelings of helplessness, worthlessness, anxiety, and arousal. The hawk-dove model proposes that differences in physiological reactions may account for the presence of certain subtypes of depression observed in research and clinical settings.

Multiple theoretical models have been proposed to provide a link between behavioral, psychological, and physiological responding. These theoretical models have been extended to the study of depression, providing a theoretical explanation for the maladaptive patterns of cardiovascular responding that have been observed in depressed populations. Together, these theoretical and empirical works suggest that cardiovascular functioning is related to depression and can advance our understanding risk of relapse and recurrence. The following section will review research on cardiovascular functioning and reactivity in current and remitted major depressive disorder.

Cardiovascular Functioning in Depression

Current Major Depressive Disorder

The association between MDD and CVD is likely bidirectional, with depression contributing to the development of CVD and CVD related to increased risk of MDD (Lippi, Montagnana, Favaloro, & Franchini, 2009). A large body of work indicates that depressive symptoms and disorders are associated with an increased risk for CVD (see Haigh, Bogucki, Dearborn, Robbins, & Elias, 2018b for a review). Systematic reviews and meta-analysis have concluded that depressive symptoms or a clinical diagnosis of depression predict the development of coronary heart disease (Gan et al., 2014; Nicholson, Kuper, & Hemingway, 2006; Ruglies, 2002; Wulsin & Singal, 2003; Van der Kooy et al., 2007), myocardial infarction, stroke, and other forms of CVD (Van der Kooy et al., 2007). While evidence from these meta-analyses and systematic reviews are striking, there are significant methodological flaws (e.g., failure to exclude for CVD at baseline, publication bias, impartial adjustments, possibility of reverse causality; Nicholson et al., 2006) that temper the interpretations that can be made about the impact that depression has on the development of CVD.

Research suggests that cardiovascular events and CVD are associated with increased depressive symptomology and diagnosis. Cross-sectional studies have shown that a large proportion of individuals with CVD report elevated rates of depressive symptoms (11.00-50.00%) or meet the diagnostic criteria for MDD (26.00%; Brown, Barton, & Lambert, 2009). Longitudinal studies have shown that individuals with CVD who have comorbid depression report poorer adherence to medical interventions, reduced quality of life, and increased occurrence of subsequent cardiovascular events and mortality (Brown et al., 2009).

While the literature has established that a relationship exists between depression and CVD, the nature of this relationship remains unclear. Cardiovascular functioning has been explored as a potential mechanism of action due to its association with physical and psychological functioning (Rottenberg, 2007b). With respect to depression, various naturalistic and experimental paradigms have been used to assess how different psychological states impact cardiovascular functioning. The following section will review the literature base that examines cardiovascular functioning at rest and in response to stress and sadness in individuals who are currently depressed.

Cardiovascular functioning at rest. Cardiovascular functioning at rest is an important indicator of cardiovascular health. Cardiovascular functioning has been assessed through balance of the ANS (Thayer & Lane, 2009). Autonomic balance is evident when equipoise exists between the SNS and the PNS while autonomic imbalance is present when the SNS is overactive and the PSN is underactive. Previous research has found that autonomic imbalance, as indexed by higher HR, lower HRV and RSA, and slower HR recovery, is associated with increased risk of functional impairment, morbidity, and mortality (Phillips, Ginty, & Hughes, 2013; Thayer & Lane, 2009).

A large body of literature has investigated cardiovascular functioning in adults with current MDD at rest, which has employed a variety of activities (e.g., lay down, sit quietly, sleep, engage in daily activities, or complete a breathing task). Rottenberg (2007b) conducted a meta-analysis of 13 articles that compared RSA at rest in clinically depressed and healthy control participants. Results found lower resting RSA in currently depressed participants with ($p < .001$, $d = .28$) and without ($p < .001$, $d = .33$) a history of CVD compared to healthy control participants. While it is important to assess cardiovascular functioning in depressed individuals

with and without cardiovascular problems, the presence of CVD may be a confound and therefore should be controlled or excluded for when examining the relationship between depression and cardiovascular health (Kemp et al., 2010).

Building on the work of Rottenberg (2007b), Kemp and colleagues (2010) conducted a similar meta-analysis of 18 articles that compared multiple measures of HRV at rest in clinically depressed and healthy control participants. Importantly, none of the participants included in these analyses had a history of CVD. Results indicated that participants with current MDD showed lower resting time frequency HRV (p = .01, d = -.29), long-term HRV (p = .03, d = -.46), and HF-HRV (p = .03, d = -.21) and higher LF/HF ratio (p = .01, d = 066) compared to healthy control participants. In addition, current depressive symptom severity was negatively associated with HRV (p < .001, d = -.13), suggesting that more severe depressive symptoms were associated with lower resting HRV. Together, these meta-analyses provide evidence for the small, but significant association between depression and lower resting RSA and HRV measures. This area of inquiry is important as cardiovascular abnormalities have been previously shown to contribute to the relationship between depression and CVD (as reviewed by Rottenberg, 2007b).

Recent studies published after the aforementioned meta-analyses have found similar results in currently depressed participants without a history of CVD. Kikuchi and colleagues (2009) showed that participants with current MDD showed lower LF-HRV, but not HF-HRV, while laying down after a 20-minute resting period compared to participants with panic disorder (t = 2.54, p = .02) and healthy control participants (t = 2.47, p = .02). It was hypothesized that cardiovascular differences reflect lower baroreflex sensitivity, implicated in the regulation of BP. Kemp, Quintana, Felmingham, Matthews, and Jelinek (2012) found that compared to healthy control participants, participants with current MDD exhibited significantly lower HRV on all

measures (R^2 = 2.99, η^2_p = .19, p = .001). Specifically, participants with current MDD showed lower LF/HF ratio (R^2 = 7.71, p = .01, d = .42) and HF-HRV (R^2 = 7.71, p = .01, d = -.46) while seated compared to healthy control participants. In addition, subgroup differences were found for the current MDD group based on the presence of comorbid anxiety; participants with comorbid generalized anxiety disorder had significantly higher LF/HF (p = .03, d = .94) and lower HF-HRV (p = .01, d = .85) compared to participants without any comorbidities.

Chang and colleagues (2012) found that participants with current MDD showed significantly lower HRV variance, LF-HRV, HF-HRV (p's < .001), and LF/HF ratio (p = .061) while laying down after a 20-minute resting period compared to healthy control participants. In addition, subgroup differences existed in the current MDD group based on the presence of suicidal ideation. Participants with MDD and suicidal ideations had significantly lower HRV variance (p = .04) and HF-HRV (p = .01) compared to MDD participants without suicidal ideation. While all studies identified some differences in HRV among currently depressed participants, the specific cardiovascular abnormalities differed. This could be due to differences in samples (i.e., subsamples with comorbidities) or recording procedures (i.e., laying versus sitting, use of a pre-recording resting period). Overall, these studies provide additional support for the association between depression and poorer resting cardiovascular functioning and suggest that psychiatric comorbidity and suicidality may negatively impact cardiovascular functioning further.

In summary, depression appears to be associated with poorer cardiovascular functioning at rest compared to healthy control participants. More specifically, individuals with current MDD generally show lower resting RSA and HRV. This line of research is important as it is possible that these cardiovascular abnormalities contribute to the relationship between depression and

CVD. To more fully understand the relationship between depression and cardiovascular functioning, research has explored cardiovascular reactivity to emotionally-inducing stimuli (i.e., stress and sadness). The following section will review research on cardiovascular reactivity to stress among individuals who are currently depressed.

Cardiovascular reactivity in response to stress. Research suggests that the link between depression and CVD might be attributed to excessive cardiovascular reactivity in response to stress. Kibler and Ma (2004) conducted a meta-analysis to examine the strength of the relationship between depressive symptoms and cardiovascular reactivity to experimental stressors (e.g., mental arithmetic, Stroop, startle, cold pressor, mirror tracing, anger recall, verbal challenge, and caregiving story). Eleven empirical studies that primarily focused on the impact of stress on HR and BP were statistically examined. Results indicated that there was a moderate relationship between depressive symptoms and HR reactivity to stress (d = .37) and a weaker relationship between depressive symptoms and systolic (d = .13) and diastolic (d = .17) BP reactivity to stress. In addition, the effect size for HR and diastolic BP were significantly larger in samples that included participants with CVD compared to samples with participants free from cardiovascular problems (p's < .05). Of note, most studies utilized self-report measures to assess depressive symptoms rather than diagnostic measures, which limits the generalizability of results to clinical samples. While this study does not inform the directionality of the relationship between depression and CVD, the results suggest that depressive symptoms are related to cardiovascular reactivity in response to stress.

Additional research has been conducted on cardiovascular reactivity in response to experimental stress inductions in current MDD. Studies have employed a variety of empirically-validated stress inductions that are physically (e.g., handgrip and mirror tracing tasks; Nugent et

al., 2011; Rottenberg et al., 2007a; Salomon et al., 2009), cognitively (e.g., mental arithmetic and N-back tasks; Ehrenthal et al., 2010; Liang, Lee, Chen, & Chang, 2015; Nugent et al., 2011), emotionally (e.g., anger recall task; Ehrenthal et al., 2010), or socially (e.g., speech task; Panaite et al., 2016; Rottenberg et al., 2007a; Salomon et al., 2009) stressful in nature. Importantly all studies excluded for the presence of CVD. In general, studies showed that participants with current MDD exhibited lower cardiovascular reactivity as measured by HR, HRV, RSA, and CO in response to most stress induction tasks compared to healthy control participants (i.e., Ehrenthal et al., 2010; Liang et al., 2015; Nugent et al. 2011; Rottenberg et al., 2007a; Salomon et al., 2009; with the exception of Panaite et al., 2016). In addition, one study showed that participants with current MDD exhibited less HR recovery following the speech and mirror tracing tasks relative to healthy control participants (*p's* < .05; Salomon et al., 2009). Together, these results suggest that individuals with current MDD exhibit a less adaptive pattern of cardiovascular reactivity when faced with stress.

There is a growing body of evidence to suggest that depression is associated with maladaptive cardiovascular reactivity in response to stress. More specifically, individuals with current MDD generally show lower HR, HRV, RSA, and CO when exposed to stressful experimental tasks. Given that MDD is characterized by depressed mood, negative affect, and apathy, research has explored cardiovascular reactivity in response to sadness. In the following section, research examining cardiovascular reactivity in response to sadness in individuals who are currently depressed will be reviewed.

Cardiovascular reactivity in response to sadness. Research has investigated if the link between depression and CVD may be attributed to maladaptive cardiovascular reactivity in response to a sad mood (Table 1). This line of inquiry investigates if experimentally-induced

sadness has a significant impact of the cardiovascular functioning of individuals who are already experiencing a low, depressogenic mood.

Some studies have found differences in cardiovascular reactivity in response to sad-valenced stimuli among currently depressed participants. Rottenberg and colleagues (2003) examined cardiovascular reactivity in response to a sad film. Contrary to the studies on cardiovascular functioning at rest, no differences in RSA existed between women with current MDD and healthy control women at baseline. While RSA significantly increased for healthy control women who cried in response to the sad film ($R^2 = 12.65$, $p < .005$, $\varepsilon = .739$), there was no change in RSA among women with current MDD who cried ($R^2 = 2.64$, $p > .05$, $\varepsilon = .967$).

Jin, Steding, and Webb (2015) also found that there were no differences in HR and RSA between currently depressed and healthy control participants during baseline. While watching sad and amusing films, HR decreased significantly more in healthy control participants compared to participants with current MDD ($p < .05$, $\eta^2_p = .83$). This pattern of responding was not observed for RSA, suggesting that depressed individuals showed blunted cardiovascular reactivity for some, but not all, cardiovascular markers. Together, these studies suggest that cardiovascular functioning does not differ between depressed and non-depressed individuals at rest; however, differences emerge when exposed to sad stimuli.

Rottenberg and colleagues (2005a) showed that RSA reactivity in response to the sad mood induction predicted recovery from MDD. Specifically, currently depressed participants who exhibited vagal withdrawal (i.e., decrease in RSA from baseline) to the sad film had significantly higher rates of remission at 6-months follow-up ($p < .05$). Of note, this relationship was not found for the fear and amusing films. This study provides preliminary support for the notion that participants with current MDD show differential responsivity to sad stimuli, but not

other negatively-valenced stimuli (i.e., fear). Panaite and colleagues (2016) replicated the work of Rottenberg and colleagues (2005a). Currently depressed participants who exhibited reduced vagal withdrawal in response to a sad film had significantly higher depressive symptoms at 30-weeks follow-up ($b = 5.18$, $p = .002$). This relationship was not found for fear or amusing films ($p's > .05$). Together, these studies suggest that individuals with current MDD who exhibit blunted reactivity in response to sad-valenced stimuli are more likely to report elevated depressive symptoms and less likely to experience remission of depression over time.

In contrast, other studies have failed to find differences in cardiovascular reactivity in response to a sad mood induction among currently depressed participants. Rottenberg and colleagues (2005b) compared participants with current and remitted MDD to those without a history of Axis I disorders. The three groups did not show differences in HR in response to happy, neutral, and sad films that were previously experimentally validated or idiographic in nature ($p's > .10$). Of note, participants with current and remitted MDD exhibited a non-statistically significant increase in HR during all the experimental tasks. Tsai, Pole, Levenson, and Muñoz (2003) compared Latino women with current MDD to those without a history of Axis I disorders. Results indicated that current MDD and healthy control participants who watched sad and amusing films did not differ in terms of cardiac IBI ($p > .05$).

While methodological procedures were generally consistent across studies, Tsai and colleagues (2003) did use a different diagnostic assessment and sad mood induction procedure. The samples recruited and cardiovascular measures assessed also differed across studies, with some studies examining a restricted range of participants (e.g., Tsai et al. (2003) only included Latino participants) and cardiovascular markers (e.g., Rottenberg et al. (2005b) only examined HR). In addition, four studies did not assess or exclude for the presence of CVD (i.e., Rottenberg

et al., 2003, 2005a, 2005b; Tsai et al., 2003), which could potentially confound and cause discrepancies in the cardiovascular results. These methodological problems could have contributed to the differences observed across studies.

There is some evidence to suggest that depression is associated with maladaptive cardiovascular reactivity in response to sadness. Specifically, individuals with current MDD generally show blunted HR and RSA reactivity when exposed to sad mood induction procedures. However, results have not been consistent across the literature, with some studies failing to replicate these patterns of cardiovascular reactivity and showing an opposite trend of cardiovascular reactivity (i.e., Rottenberg et al., 2005b; Tsai et al., 2003).

Remitted Major Depressive Disorder

As previously detailed, MDD is characterized by high rates of relapse and recurrence. The chronic nature of the disorder suggests that a large proportion of individuals with a history of depression will experience another depressive episode over the course of their lifetime. Therefore, research is needed to identify risk factors for relapse and recurrence in euthymic individuals with a history of depression. Two prominent classes of risk factors include cognitive and mood vulnerabilities. The literature has suggested that cognitive and mood vulnerabilities that are present in currently depressed individuals remain latent in formerly depressed individuals until they are activated by dysphoric mood.

This body of evidence proposes that the differential activation and mood state dependent hypotheses may extend to other areas, such as cardiovascular functioning. It is plausible that the maladaptive patterns of cardiovascular reactivity observed in currently depressed individuals may also be mood state dependent. It would follow that individuals with a history of depression would show cardiovascular abnormalities in response to emotion-provoking stimuli such as

stress and sadness, but not while they were euthymic. To test this hypothesis, research has examined cardiovascular functioning at rest as well as cardiovascular reactivity in response to stress and sadness in adults and adolescents with remitted MDD; however, cardiovascular functioning among individuals with a history of depression has not been investigated to the same extent as current depression. The available research that examines cardiovascular functioning in adults and adolescents with remitted MDD is reviewed below.

Cardiovascular functioning at rest. Some studies have investigated cardiovascular functioning in adults with remitted MDD at rest or during the completion of daily life activities. Chang and colleagues (2013) examined HRV at rest among formerly depressed participants with or without a history of suicide ideation. Participants were free from CVD and other medical conditions (e.g., diabetes, hypertension, etc.) that could impact the recording of physiological responses. Depression was diagnosed using the Modified Schedule for Affective Disorders and Schizophrenia – Lifetime Version (SADSL) and the HDRS. Participants with remitted MDD were classified as having (n = 237) or not having (n = 233) a history of suicidal ideation during a past depressive episode. In addition to exploring cardiovascular differences based on a history of suicidality, participants with remitted MDD were compared to healthy controls (n = 462) without a history of MDD or suicidal ideation. HRV was assessed for five minutes while participants relaxed following a 20-minute baseline period. Results showed that participants with remitted MDD who endorsed a history of suicidal ideations have significantly lower HRV variance (p = .001), HF-HRV (p = .01), and LF-HRV (p = .004) than participants with remitted MDD without a history of suicidality and healthy control participants. No differences were identified between remitted MDD participants without a history of suicidality and healthy control participants (*p's* >

.05). Overall, these findings suggest that some differences in cardiovascular functioning exist at rest in participants with remitted MDD, but only among those with a history of suicidal ideation.

Vaccarino and colleagues (2008) evaluated HRV among a sample of twins with current or remitted MDD. Participants were free from CVD when initially evaluated in 1990; however, only coronary heart disease, myocardial infarction, and angina pectoris was assessed at the time of the current investigation. The DIS was originally used to diagnose current and past MDD. A total of 288 twins contributed psychophysiological data, which included participants with current (n = 7) and remitted (n = 61) MDD that were collapsed into a single group. History of MDD was re-confirmed by the SCID-I. HRV was recorded over the course of 24-hours and timing of daily life activities were matched across participants. While TP-HRV, ULF-HRV, VLF-HRV, and LF-HRV was significantly lower (p's < .05) in twins with current or remitted MDD, this relationship did not hold when additional variables (e.g., lifestyle factors, comorbid medical, and psychiatric conditions) were entered into the model. Several methodological flaws (e.g., poor screening for CVD and combined sample of current and remitted MDD participants), temper the finding that history of depression is not associated with lower HRV while at rest and during activities of daily living.

As reviewed, there does not appear to be significant differences in cardiovascular functioning at rest among individuals with remitted MDD. These findings are in line with the differential activation and mood state dependent hypotheses, which suggests that vulnerability to depression (e.g. maladaptive patterns of cardiovascular functioning) is only observable when formerly depressed individuals are faced with an emotional challenge. Therefore, it is possible that maladaptive patterns of cardiovascular reactivity observed in currently depressed participants will be present in formerly depressed participants exposed to stress. In the following

section, research examining cardiovascular functioning in response to stress in remitted MDD will be reviewed.

Cardiovascular reactivity in response to stress. A portion of the literature has investigated cardiovascular functioning in adults with remitted MDD in response to experimental stress inductions. Ahrens and colleagues (2008) examined HRV in adults with remitted MDD during stressful and cognitively challenging tasks. All participants were free from major ventricular or supraventricular arrhythmias and current medical conditions were endorsed as stable; however, the presence of CVD was not comprehensively assessed, which could potentially confound the cardiovascular results. Participants were women with remitted MDD (n = 22) and healthy women without a history of affective disorders (n = 20). Experimental tasks included completion of several stressful (i.e., speech and mental arithmetic) and cognitively demanding (i.e., computer concentration) tasks. HR and HRV was collected continuously during the experimental procedures. Three average HR measurements were computed during baseline, completion of tasks, and recovery. Five average HRV measurements were computed during baseline while supine and standing and during the speech, mental arithmetic, and computer concentration tasks. Results did not reveal any significant differences in HR and HRV between participants with remitted MDD and healthy control participants across the experimental paradigm (p's > .05). The authors theorized that the results might be due to the nature of the stressful tasks, stating that more demanding tasks may elicit differences in cardiovascular functioning across groups.

Salomon and colleagues (2013) used different methodological procedures to examine cardiovascular reactivity in response to stress in adults with remitted MDD. Participants were included if they did not endorse CVD and other medical conditions (e.g., head injury, substance

abuse, etc.) or medications (e.g., antipsychotics, beta blockers, etc.) that could impact the recording of physiological responses. Depression was assessed using the SCID-I. The sample was comprised of participants with current MDD (n = 50) or remitted MDD (n = 25) and healthy control participants without a history of Axis I disorders (n = 45). Participants watched a neutral video during the baseline period, completed an active (i.e., speech preparation and delivery under observation) and passive (i.e., forehead cold pressor task) stressful task in counterbalanced order, and watched a video during the recovery period. HR, PEP, and CO were collected continuously during the experimental procedures. Six average measurements were computed for each cardiovascular measure during baseline, the speech preparation, delivery, and recovery, and the cold pressor task and recovery.

Results did not reveal any significant differences in cardiovascular reactivity among currently or formerly depressed or healthy control groups during baseline (p's > .05) and the forehead cold pressor task (p's > .29). Salomon and colleagues (2013) speculated that the null findings might be because participants with current MDD only show reduced reactivity when confronted with an active task (e.g., speech) that they perceive as insurmountable, which is in line with the biopsychosocial model of challenge and threat. Differential patterns of cardiovascular reactivity were observed during components of the speech task. During the speech preparation, participants with current MDD showed significantly lower HR (R^2 = 3.74, p < .01, η^2_p = .10) and PEP (R^2 = 4.32, p < .05, η^2_p = .08) reactivity compared to participants with remitted MDD and healthy control participants. During the speech delivery, participants with current MDD showed significantly lower HR (R^2 = 8.14, p < .001, η^2_p = .14), CO (R^2 = 4.68, p < .05, η^2_p = .08), and PEP (R^2 = 9.33, p < .001, η^2_p = .16) reactivity compared to participants with remitted MDD and healthy control participants. Of note, participants with remitted MDD did not

significantly differ from healthy control participants on any cardiovascular measure during the speech preparation delivery, and recovery (*p's* > .05). Overall, these findings provide preliminary support for the notion that individuals with current MDD, but not remitted MDD, show a blunted cardiovascular response to active stressful tasks.

In a follow up study, Bylsma and colleagues (2014) sought to replicate the finding that adults with current but not remitted MDD exhibit blunted cardiovascular reactivity to active, stress inducing tasks. All participants were free from CVD and other medical conditions or medications that could impact the recording of physiological responses. The sample was comprised of participants with current MDD (*n* = 51) or remitted MDD (*n* = 25) and healthy controls without a history of Axis I disorders (*n* = 45). Experimental tasks included a baseline video, paced breathing baseline, an active (i.e., speech preparation and delivery under observation) and passive (i.e., forehead cold pressor task) stressful task in counterbalanced order, and recovery video. RSA was collected continuously during the experimental procedures. Eight average measurements were computed for RSA during baseline, the paced breathing task, the speech instructions, preparation, delivery, and recovery, and the cold pressor task and recovery.

Results revealed that cardiovascular measures did not significantly differ between groups during baseline and the forehead cold pressor task (*p's* > .05). However, Bylsma and colleagues (2014) did observe differential patterns of cardiovascular reactivity during the speech task. During the speech preparation, delivery, and recovery, participants with current MDD showed significantly lower RSA ($p < .05$) compared to participants with remitted MDD and healthy control participants. However, this relationship was no longer significant when adjustments were made for covariates ($p > .42$; i.e., sleep quality). In line with the previous study by Salomon and

colleagues (2013), findings suggested that individuals with current MDD, but not remitted MDD, exhibit a blunted cardiovascular response to active stressful tasks.

Wilson and colleagues (2016) examined cardiovascular reactivity in response to stress among adults with a history of MDD with or without a prior suicide attempt. All participants were free from CVD and other autonomic disorders (e.g., diabetes, Parkinson's disease, etc.) that could impact the recording of physiological responses. Participants were assessed for depression using the SCID-I and history of suicide using the Columbia University Suicide History Form and the Lethality Scale. Participants included women with remitted MDD with (n = 13) or without (n = 22) a previous suicide attempt. Experimental procedures included a resting baseline and the Trier Social Stress Task (TSST), a widely-used laboratory-based paradigm used to induce moderate levels of social stress. HF-HRV was collected continuously during the experimental procedures and averaged within each experimental phase. Results showed that HF-HRV did not significantly differ between groups during baseline (p = .09). However, participants with a history of suicide attempts showed significantly lower HF-HRV during the TSST (t = 5.4, p = .03) compared to participants without a history of suicide attempts. Findings echo previous studies on the impact of stress on cardiovascular functioning, suggesting that individuals with remitted MDD who have attempted suicide exhibit blunted cardiovascular reactivity in response to stressful tasks that are active.

While Wilson and colleagues (2016) found that adults with a history of depression show lower HF-HRV in response to stress, this study focused on a subset of the remitted depressed individuals (i.e., those with a history of suicide behavior) that is not representative or the entire population. Instead, the majority of the literature has shown that adults with remitted MDD do not exhibit a maladaptive pattern of cardiovascular reactivity when exposed to stressful stimuli

(i.e., Ahrens et al., 2008; Salomon et al., 2013; Bylsma et al., 2014). It is possible that the differential activation and mood state dependent hypotheses only apply to emotions that are relevant to depression and that the maladaptive patterns of cardiovascular reactivity observed in currently depressed participants will be present in formerly depressed participants who become sad. In the following section, research examining cardiovascular reactivity in response to sadness in a mixed sample of currently and formerly depressed participants will be reviewed.

Cardiovascular reactivity in response to sadness. Cardiovascular reactivity in response to a sad mood induction is separately reviewed below for research using a mixed adult and adolescent sample of current and remitted MDD participants as well as an adult sample of remitted MDD participants.

Cardiovascular reactivity in response to sadness in a mixed adult sample of current and remitted depression. A set of studies have examined cardiovascular reactivity in response to a sad mood induction in a mixed sample of adults with current and remitted MDD (Table 1). Yaroslavsky, Rottenberg, and Kovacs (2013) examined if RSA measured at rest and in response to a sad mood could predict current depressive symptoms and history of depression. Participants were adults with a history of MDD during adolescence (n = 113; 37.00% currently experiencing a depressive episode) and healthy control participants without a history of Axis I disorders (n = 93). Depressive symptoms were assessed using the BDI and Follow-Up Depression Scale, a clinician-rated scale for depressive symptom severity. Experimental tasks included a resting baseline and film-based mood inductions for joy, fear, sadness, anger, and disgust that were followed by resting periods. RSA was collected continuously during the experimental procedures.

Yaroslavsky and colleagues (2013) found that the combination of resting RSA and RSA reactivity, but not resting RSA and RSA reactivity alone, predicted current depressive symptoms and previous depression status. Participants who showed high resting RSA and RSA withdrawal (i.e., decrease in cardiac vagal control) in response to the sad mood induction were more likely to report lower depressive symptoms and less likely to have a history of depression (*p's* < .05). In contrast, the interaction between resting RSA and RSA augmentation (i.e., increase in cardiac vagal control) in response to the sad mood induction was not significant ($p = .97$).

Study 1 by Yaroslavsky, Rottenberg, and Kovacs (2014) investigated whether RSA measured at rest and in response to a sad mood could predict depressive history in a sample of women with juvenile-onset depression. Participants were adult women with a history of MDD during adolescence ($n = 27$; 48.00% currently experiencing a depressive episode) and healthy women without a history of Axis I disorders ($n = 43$). Experimental tasks included a resting baseline and film mood inductions for joy and sadness. RSA was collected continuously during the experimental procedures. The focus of this study was solely on RSA during rest and RSA in response to the sad mood induction; average RSA resting measurements were computed via resting baseline while average RSA reactivity was computed during the sad mood induction.

In Study 1, Yaroslavsky and colleagues (2014) found that participants who showed an abnormal pattern of RSA responding (i.e., high resting RSA and RSA augmentation or low resting RSA and RSA withdrawal) were more likely to endorse a history ($p < .05$) or current episode ($p < .001$) of depression. In addition, participants who showed a normal pattern of RSA responding (i.e., high resting RSA and RSA withdrawal or low resting RSA and RSA augmentation) were less likely to endorse a history or current episode of depression. These findings replicate their previous findings (Yaroslavsky et al., 2013), and suggest that

cardiovascular reactivity during a sad mood can be used to characterize the presence of current or past depression.

The aforementioned studies (Yaroslavsky et al., 2013, 2014 Study 1) showed that the interaction between RSA at rest and RSA reactivity was a significant predictor of depressive history and symptoms in participants with current and remitted MDD. However, there are significant methodological problems that limit the generalizability and validity of these findings. First, a portion of the participants endorsed currently experiencing a depressive episode during data collection, which resulted in a mixed sample consisting of both current and remitted MDD. It is possible that the findings are only applicable to the currently depressed participants as research has been shown that current MDD impacts cardiovascular functioning during a sad mood induction (e.g., Jin et al., 2015; Rottenberg et al., 2003; Rottenberg et al., 2005a; Panaite et al., 2016). Second, participants were not assessed for the presence of CVD. Previous research has indicated that CVD may be a confound and therefore should be controlled or excluded for when examining the relationship between depression and cardiovascular health (Kemp et al., 2010). Finally, the studies utilized the same sample of participants who experienced a depressive episode during adolescence. Of note, differences have been found between adolescent and adult depression (Kaufman, Martin, King, & Charney, 2001). Consequently, the results may not generalize to adult-onset depression. While the interaction between RSA at rest and RSA reactivity is an intriguing line of inquiry, additional methodologically sound research is needed to investigate if this pattern of cardiovascular reactivity truly characterizes remitted depression in adults.

Cardiovascular reactivity in response to sadness in a mixed adolescent sample of current and remitted depression. A set of studies examined cardiovascular reactivity in response

to a sad mood induction in a mixed sample of adolescents with current and remitted MDD (Table 1). Study 2 by Yaroslavsky and colleagues (2014) investigated if RSA measured at rest and in response to a sad mood could predict depressive history and symptoms in adolescents. Participants and another informant completed the Interview Schedule for Children and Adolescents: Diagnostic Version (ISCA-D), a semi-structured interview used to diagnose depression and the Children's Depression Inventory – Second Edition (CDI-2), a self-report form to assess depressive symptoms. Participants included 147 Hungarian proband-sibling pairs in which one sibling had a history of MDD during childhood (n = 132) and the other sibling also had a history of MDD during childhood (n = 36) or no history of MDD (n = 111). Of note, the authors did not provide detailed information about the sample and refer to previous studies for more information. These studies focus on currently depressed adolescents (e.g., Baji et al., 2009; Kiss et al., 2007; Tamás et al., 2007), suggesting that the sample of interest is not made up of purely formerly depressed participants. Experimental tasks included a paced breathing task and film-based sad mood induction. RSA was collected continuously during the experimental procedures. Average RSA resting measurements were computed during the paced breathing task while average RSA reactivity was computed by taking the difference between RSA during the paced breathing task and the sad mood induction.

In Study 2, Yaroslavsky and colleagues (2014) showed that an abnormal pattern of RSA at rest and in response to the sad mood induction (i.e., high resting RSA and RSA augmentation or low resting RSA and RSA withdrawal) were present in proband-sibling pairs where both children experienced depression (OR = 6.46, CI = 1.15, 36.47, $p < .05$), but not in proband-sibling pairs where only the proband experienced depression. These results, which replicate the finding from the first study reported in the manuscript (Yaroslavsky et al., 2014 Study 1), found

an abnormal pattern of RSA responding in depressed adolescents that corresponded with the abnormal pattern of RSA responding previously observed in adults.

Bylsma and colleagues (2015) examined cardiovascular responsivity to sadness and stress in adolescents with remitted MDD. Participants and another informant completed the ISCA-D to diagnose depression and the CDI-2 to assess depressive symptoms. Participants included adolescents with a history of MDD during childhood (n = 216; 14.80% currently experiencing a depressive episode) and healthy control participants without a history of Axis I disorders (n = 161). Experimental tasks included a neutral and sad film mood induction, unsolvable puzzle, handgrip task, and forehead cold pressor task followed by resting periods. RSA, PEP, cardiac autonomic balance (CAB), and cardiac autonomic regulation (CAR) was collected continuously during the experimental procedures. CAB was calculated by subtracting RSA and PEP and is an index of the balance between SNS and PNS activation while CAR was calculated by adding RSA and PEP and is an index of activation of both the SNS and the PNS. Average RSA, PEP, CAB, and CAR resting measurements were computed during the resting periods while average RSA, PEP, CAB, and CAR reactivity was computed for each experimental task.

Bylsma and colleagues (2015) did not find differences in cardiovascular responding between groups at baseline (p*'s* > .10); however, some group differences did emerge during the experimental paradigm. Participants with a history of MDD exhibited a greater increase in CAB during the unsolvable puzzle (p = .17) and handgrip tasks (p = .14), which is indicative of greater SNS and PNS activation. Conversely, healthy control participants exhibited a greater decrease in PEP (p = .001) and increase in CAR during the handgrip task (p = .03), which is indicative of greater SNS responding and less SNS and PNS activation. Interestingly, no differences were found between groups during the sad film mood induction (p*'s* > .05). While the authors attribute

this finding to the potency of the experimental task, the sad film clip (i.e., *The Champ*) has been empirically validated and widely used in the literature. Although these findings suggest that CAB increases in remitted depressed participants in response to stress but not sadness, it is possible that these results were due to the use of a mixed samples made up of participants with current and remitted MDD.

The aforementioned studies (Bylsma et al., 2015 Yaroslavsky et al., 2014 Study 2) showed that adolescents with current and remitted MDD exhibit an abnormal pattern of cardiovascular reactivity (i.e., interaction between RSA at rest and RSA reactivity, CAB, and CAR) in response to a sad mood compared to healthy control participants. However, these results must be evaluated in light of their significant methodological flaws. First, the studies used a mixed sample of current and remitted MDD participants, which could limit the generalizability of these findings to currently depressed rather than formerly depressed participants. Second, the medical screening procedures employed by these studies were insufficient. While participants were assessed for major medical disorders, they were not specifically evaluated for CVD. Previous research has suggested that the presence of CVD may be a confound and therefore should be controlled or excluded for when examining the relationship between depression and cardiovascular health (Kemp et al., 2010). Additional methodologically sound research is needed to investigate if an abnormal pattern of cardiovascular reactivity in response to sadness is consistently observed in adolescents with remitted depression. It should also be noted that differences have been found between adolescent and adult depression (Kaufman et al., 2001). Consequently, results from these studies may not generalize to adult samples.

Cardiovascular reactivity in response to sadness in an adult sample of remitted depression. Only one study has investigated cardiovascular functioning in adults with remitted

MDD in response to an experimentally-induced sad mood (Table 1). Rottenberg and colleagues (2005b) examined HR reactivity in response to a sad mood in adults with remitted MDD. Participants were free from medical conditions (e.g., head injury, substance abuse, etc.) that could impact the recording of physiological responses; however, CVD was not assessed, which could potentially confound the cardiovascular results. Depression was assessed using the SCID-I and included individuals with current MDD (n = 19) or remitted MDD (n = 22) and healthy controls without a history of Axis I disorders (n = 26). During the experimental paradigm, participants were instructed to watch sad, happy, and neutral valenced films presented in a counterbalanced order then imagine the scene in their mind. Each film and imagery task were preceded by a one-minute resting baseline and followed by a one-minute filler task to reduce carry over effects. HR was collected continuously during the experimental procedures and average measurements were computed. Results revealed that HR did not significantly differ across group, emotional valence, or stimulus type ($p > .10$). These findings provide preliminary support that cardiovascular reactivity does not differ based on depression status in the face of various emotional experiences.

The current literature examining cardiovascular reactivity in response to sadness for formerly depressed individuals is extremely limited. Rottenberg and colleagues (2005b) did not identify a significant difference in HR reactivity in response to sadness between participants with remitted MDD to healthy control participants. It is possible that a single cardiovascular marker is insufficient to characterize the pattern of cardiovascular reactivity in response to a sad mood induction. As explained by the theoretical models, a cascade of complex physiological processes is implicated in cardiovascular functioning. As such, it may be necessary to examine multiple measures of cardiovascular reactivity that are thought to index various aspects of the ANS in

order to truly characterize how individuals with remitted MDD react to a sad mood induction. Additional research is needed to empirically test this hypothesis.

Table 1. *Previous Research on Cardiovascular Reactivity to Sadness in Current and Remitted Depression by Sample*

CD in Adult Samples						
Study	Sample	Diagnostic Interview	Experimental Tasks	Cardiovascular Measures	CVD Assessed	Findings
Panaite et al. (2016)	CD = 49 RD = 24 HC = 45	SCID-I	Sad, fear, and happy films	RSA	Yes	. ↓ RSA withdrawal during sad film predicted ↑ MDD symptoms at follow-up in CD . No other predictive results for other films
Jin et al. (2015)	CD = 25 HC = 25	SCID-I	Sad and amusing films	HR, RSA	Yes	. ↓ HR decrease during sad and amusing films in CD than HC . No other group differences during baseline or other films
Rottenberg et al. (2003)	CD = 25♀ HC = 31♀	SCID-I	Sad and neutral films	RSA	No	. ↑ RSA after sad film in HC who cried during sad film . No ΔRSA after sad film in CD who cried during sad film
Rottenberg et al. (2005a)	CD = 55	SCID-I	Sad, fear, and amusing films	RSA	No	. RSA withdrawal to sad film predicted recovery from MDD at follow-up . No other predictive results for other films
Tsai et al. (2003)	CD = 12♀ HC = 10♀	PRIME-MD	Sad, amusing, and neutral films	Cardiac IBI	No	. No group differences in cardiac IBI during baseline or films
Combined CD/RD in Adult Samples						
Study	Sample	Diagnostic Interview	Experimental Tasks	Cardiovascular Measures	CVD Assessed	Findings
Yaroslavsky et al. (2013)	CD/RD = 113 HC = 93	SCID-I	Sad, anger, disgust, fear, and joy films	RSA	No	. Typical resting RSA X ΔRSA during sad film predicted HC status . No other results for sad or other films

Table 1 Continued

Yaroslavsky et al. (2014) Study 1	CD/RD = 27♀ HC = 43♀	SCID-I	Sad and joy films	RSA	No	. Atypical resting RSA X ΔRSA during sad film predicted CD/RD status . Typical resting RSA X ΔRSA during sad film not predictive of CD/RD status . No other predictive results for other film
Combined CD/RD in Adolescent Samples						
Study	Sample	Diagnostic Interview	Experimental Tasks	Cardiovascular Measures	CVD Assessed	Findings
Yaroslavsky et al. (2014) Study 2	Proband: CD/RD = 132 Siblings: CD/RD = 36 HC = 111	ISCA-D	Sad film	RSA	No	. Atypical resting RSA X ΔRSA during sad film observed in CD/RD proband/sibling pairs . No other group differences for sad film
Bylsma et al. (2015)	Proband: CD/RD = 216 HC = 161	ISCA-D	Sad and neutral film	RSA, PEP, CAB, CAR	No	. No group differences in RSA, PEP, CAB, or CAR during baseline or films
RD in Adult Samples						
Study	Sample	Diagnostic Measure	Experimental Tasks	Cardiovascular Measures	CVD Assessed	Findings
Rottenberg et al. (2005b)	CD = 19 RD = 22 HC = 26	SCID-I	Sad, happy, and neutral films and imagery tasks	HR	No	. No group differences in HR during baseline, films, or imagery tasks

Note. CD = current major depressive disorder; CD/RD = mixed sample of current and remitted major depressive disorder; RD = remitted major depressive disorder; HC = healthy control; SCID-I = Structured Clinical Interview for the DSM-IV; PRIME-MD = Primary Care Evaluation of Mental Disorders; ISCA-D = Interview Schedule for Children and Adolescents: Diagnostic Version; HR = heart rate; HRV = heart rate variability; RSA = respiratory sinus arrhythmia; IBI = interbeat interval; CAB = cardiac autonomic balance; CAR = cardiac autonomic regulation; ↓ = decrease; ↑ = increase; Δ = change; ♀ = women only.

CHAPTER 4

OVERVIEW AND STATEMENT PURPOSE

MDD is a serious, debilitating, and commonly occurring mental illness characterized by pronounced negative mood and/or lack of interest or pleasure (APA$_A$, 2013; Kessler et al., 2003). MDD is associated with negative psychological, medical, and economic outcomes. Research has found that individuals with MDD report high rates of morbidity, mortality, and functional impairment, which results in increased disability and decreased workplace productivity (Lépine & Briley, 2011). Consequently, MDD is currently the leading cause of disability and second leading cause of disease burden around the world (Mathers et al., 2008). The direct and indirect expenses associated with MDD are estimated to cost the United States $210.50 billion yearly (Greenberg et al., 2015). Clearly, MDD is associated with significant burden at the individual and societal level.

While research has typically focused on the acute aspect of depression, MDD is typically understood as a chronic illness due to high rates of relapse and recurrence (Richards, 2011). Results of a seminal study conducted by the NIMH found that within a 15-year period, up to 50.00% of individuals with MDD will experience a relapse, or reemergence of a depressive episode before MDD has remitted, and up to 85.00% will experience a recurrence, or experience of a new depressive episode after MDD has remitted (Mueller et al., 1999). This finding has been replicated throughout the literature over the past decade. Research has shown that the rate of relapse or recurrence in individuals with a history of MDD ranges from 2.50% to 77.00% over periods of time ranging from one to 20 years (Hardeveld et al., 2010, 2013; Johansson et al., 2015; Nöbbelin, Bogren, Mattisson, & Brådvik, 2018; Poutanen et al., 2007; ten Doesschate et al., 2010).

Vulnerability to depressive relapse and recurrence is likely multiply determined and due to a complex interaction of biological, psychological, and environmental factors (Burcusa & Iacono, 2007). Researchers have identified various clinical, demographic, familial, psychological, and psychosocial factors that may make an individual more susceptible to experiencing another depressive episode (Burcusa & Iacono, 2007; Harveveld et al., 2010, 2013; Johansson et al., 2015; ten Doesschate et al., 2010). The majority of these vulnerability factors are stable, unchangeable traits. More research is needed to identify malleable vulnerability factors that can be specifically targeted during treatment or following treatment for relapse prevention to reduce the occurrence of future episodes of depression.

Cognitive theories of depression have theorized that dysfunctional thinking patterns contribute to the occurrence, maintenance, and reoccurrence of depression. Research has indicated that currently depressed individuals endorse higher rates of dysfunctional thoughts; however, individuals who have recovered from depression report patterns of thoughts that are similar to never depressed individuals (as reviewed by Teasdale, 1999). These findings suggest that cognitive vulnerability to depression (i.e. dysfunctional thoughts) are no longer present after recovery from depression, despite the fact that individuals with a history of depression remain at high risk for relapse or recurrence. Research aimed to account for these discrepant findings has focused on individual differences in how one responds to a transient, sad mood. Specifically, research has sought to understand how individuals with a history of MDD respond to the experience of sadness in between depressive episodes.

The differential activation hypothesis by Teasdale (1988) and mood state dependent hypothesis by Miranda and Persons (1988) similarly hypothesized that cognitive vulnerabilities to depression remain latent in formerly depressed individuals until activated by a dysphoric

mood. In accordance with these theories, researchers have hypothesized that individuals who have recovered from depression exhibit maladaptive cognitions and affective states during a dysphoric mood, but not a euthymic mood. Empirical evidence has generally supported these theories, and found that compared to healthy control participants, those with remitted MDD report significant increases in dysfunctional beliefs (i.e., cognitive reactivity; Kuyken et al., 2010; Segal et al., 1999, 2006) or dysphoric mood (i.e., mood reactivity; Lethbridge & Allen, 2008; van Rijsbergen et al., 2013) in response to an experimentally-induced sad mood and prospectively predict relapse of depression over time. While there is still disagreement in the literature whether dysfunctional thinking patterns or dysphoric mood states characterize remitted MDD, cognitive and mood reactivity in response to sadness have been proposed as two potential pathways of vulnerability to relapse and recurrence for formerly depressed individuals.

Examination of cardiovascular functioning in response to negative affect may advance our understanding of the relationship between reactivity to sad mood and vulnerability to depressive relapse and recurrence. A large body of research has highlighted the important role of cardiovascular functioning in MDD. Depression and depressive symptoms are associated with an increased risk of CVD (Gan et al., 2014; Nicholson et al., 2006; Ruglies, 2002; Wulsin & Singal, 2003; Van der Kooy et al., 2007) and cardiovascular events. Similarly, CVD is associated with increased rates of depression and depressive symptoms (Brown et al., 2009). This relationship has led researchers to examine cardiovascular differences that can explain the susceptibility to CVD in this population. Maladaptive patterns of cardiovascular functioning have been identified in currently depressed individuals. Compared to non-depressed counterparts, individuals with current MDD show lower RSA and HRV while at rest (Chang et al., 2012; Kemp et al., 2010, 2012; Kikuchi et al., 2009; Rottenberg, 2007b), lower HR, HRV, RSA, and CO in response to

stress (Ehrenthal et al., 2010; Liang et al., 2015; Nugent et al. 2011; Rottenberg et al., 2007a; Salomon et al., 2009), and blunted HR and RSA in response to sadness (Jin et al., 2015; Panaite et al., 2016; Rottenberg et al., 2003, 2005a). Research on cardiovascular reactivity in response to sadness is mixed, with some studies failing to replicate these patterns of cardiovascular reactivity and showing an opposite trend of cardiovascular reactivity (i.e., Rottenberg et al., 2005b; Tsai et al., 2003).

In general, the cardiovascular abnormalities that have been identified in currently depressed individuals do not appear to be present in formerly depressed individuals. Formerly depressed and healthy control participants do not differ in cardiovascular functioning at rest (HRV; Chang et al., 2013; Vaccarino et al., 2008) or cardiovascular reactivity in response to stress (HR, HRV, RSA, CO, and PEP; Ahrens et al., 2008; Bylsma et al., 2014; Salomon et al., 2013). Thus far, it appears that the cardiovascular functioning of remitted depressed participants generally resembles that of healthy control participants. These findings led researchers to hypothesize that cardiovascular abnormalities may be mood-state dependent much like the cognitive and mood vulnerabilities that have been identified in remitted depression.

Cardiovascular reactivity is operationalized as the change in an individual's cardiovascular functioning in response to a sad mood induction. The literature on cardiovascular reactivity in response to sadness among formerly depressed individuals is limited to one study that recruited a sample of adults with remitted depression. Rottenberg and colleagues (2005b) failed to find cardiovascular differences among individuals with a history of depression; there were no significant differences in HR reactivity in response to a sad mood induction when comparing remitted depressed and healthy control participants. While other researchers have attempted to study this topic, interpretation of findings have been limited by serious

methodological flaws. Bylsma and colleagues (2015) did not identify any significant differences in RSA and PEP reactivity in response to a sad mood induction when comparing remitted depressed and healthy control adolescents. Conversely, Yaroslavsky and colleagues (2013, 2014 Study 2) found that a combined group of currently and formerly depressed adults and adolescents exhibited an abnormal pattern of RSA responding (i.e., high resting RSA and RSA augmentation or low resting RSA and RSA withdrawal during sad mood induction) compared to healthy control participants.

While some of these abovementioned studies point to differences in cardiovascular reactivity among formerly depressed individuals, there are significant concerns about the quality and generalizability of these studies due to multiple methodological issues. First, most of the literature has relied upon a mixed sample of participants who were currently or formerly depressed (Bylsma et al., 2015; Yaroslavsky et al., 2013, 2014 Studies 1 and 2; with the exception of Rottenberg et al., 2005b). The cardiovascular differences that were identified in these studies may be attributable to the inclusion of currently depressed participants or the presence of subclinical depressive symptom rather than remitted depression. As a result, only one study (i.e., Rottenberg et al., 2005b) has truly investigated cardiovascular reactivity in response to sadness in remitted depression.

Second, none of the abovementioned studies explicitly assessed for the presence of CVD (Bylsma et al., 2015; Rottenberg et al., 2005b; Yaroslavsky et al., 2013, 2014 Studies 1 and 2). Previous research has indicated that the presence of CVD may be a confound and therefore should be controlled for or be an exclusion criterion when examining the relationship between depression and cardiovascular health (Kemp et al., 2010). Therefore, it is possible that these results are attributable to underlying cardiovascular illness rather than remitted depression.

Additionally, it is possible that failure to find significant differences between groups is due to differences in cardiovascular status rather than psychological variables.

Third, most of the literature has examined a very limited range of cardiovascular measures (i.e., HR or RSA only; Rottenberg et al., 2005b; Yaroslavsky et al., 2013, 2014 Studies 1 and 2; with the exception of Bylsma et al., 2015). The use of a singular cardiovascular measure may be insufficient to characterize the complex pattern of cardiovascular reactivity, as different measures are thought to index different components of the regulatory systems (e.g., SNS versus PNS) that influence cardiovascular functioning. It may be necessary to examine multiple measures of cardiovascular reactivity that are thought to index various aspects of the ANS to truly characterize how individuals with remitted MDD react to a sad mood induction. Indeed, several theoretical models (i.e., the biopsychosocial model of challenge and threat by Blascovich and Tomaka (1996) and hawk-dove model by Smith (1982)), propose a complex pattern of cardiovascular or physiological reactivity in response to stress. Consequently, analyses must be conducted for those cardiovascular markers (e.g., CO and PEP) that have not been examined among individuals with remitted depression.

Fourth, none of the relevant studies investigated cardiovascular recovery (Bylsma et al., 2015; Rottenberg et al., 2005b; Yaroslavsky et al., 2013, 2014 Studies 1 and 2). Cardiovascular recovery is operationalized as the amount of time that it takes for an individual's cardiovascular functioning to return to baseline levels following a sad mood induction. Cardiovascular recovery provides an estimate of how long the physiological changes attributable to an emotionally-valenced stimulus persist after the stimulus has been removed. It has been suggested that the study of cardiovascular recovery has significant clinical utility. More specifically, cardiovascular recovery can identify factors that contribute to the development of psychopathology and

physiological dysfunction and disease (Linden, Earle, Gerin, & Christenfeld, 1997; Haynes, Gannon, Orimoto, O'Brien, & Brandt, 1991). Cardiovascular recovery following the induction of a transient mood state may be especially important as research has shown that individuals take a longer amount of time to habituate to emotional distress compared to stress (Linden et al., 1997). Given the hypothesis that the relationship between depression and CVD is due to long-standing cardiovascular abnormalities, it is necessary to investigate the role of cardiovascular recovery as a potential dormant mechanism of vulnerability to MDD that is activated by a dysphoric mood.

Fifth, a large portion of the literature has failed to compare cardiovascular reactivity in response to both sad and neutral mood inductions (Yaroslavsky et al., 2013, 2014 Studies 1 and 2; with the exceptions of Bylsma et al., 2015 and Rottenberg et al., 2005b). The differential activation hypothesis by Teasdale (1988) and mood state dependent hypothesis by Miranda and Persons (1988) theorize that formerly depressed individuals only exhibit vulnerabilities to depression when in a dysphoric mood. Based on these hypotheses, it is expected that formerly depressed participants would react in a maladaptive manner to the sad mood induction but not the neutral mood induction. Therefore, the comparison of reactivity to sad and neutral mood inductions is necessary to empirically test these hypotheses.

Finally, a large portion of this literature has been conducted in formerly depressed adolescents (Bylsma et al., 2015; Yaroslavsky et al., 2014 Study 2; with the exceptions of Rottenberg et al., 2005b and Yaroslavsky et al., 2013, 2014 Study 1). Differences in adolescent and adult depression have been identified. For example, depressed children and adolescents do not show elevated basal cortisol levels, abnormal cortisol and prolactin secretion, and reduced immunity cells like depressed adults (Kaufman et al., 2001), suggesting that physiological

correlates of depression may differ in the two age groups. As a result, these cardiovascular findings may not generalize to adult-onset depression.

The proposed study aimed to examine the cognitive, mood, and cardiovascular correlates in response to dysphoric mood among adults with a history of depression. The primary goal of this study was to characterize cardiovascular reactivity to and recovery from a sad mood in individuals with a history of MDD. To help clarify prior inconsistent results, the secondary goal of this study was to examine cognitive and mood reactivity in individuals with a history of MDD. This study will advance our understanding of potentially malleable vulnerability factors associated with a history of depression.

This study overcame several methodological weaknesses associated with prior work. First, while most of the literature has focused on a single cardiovascular measure, the current study examined multiple cardiovascular measures to better characterize the pattern of cardiovascular functioning in remitted depression. Second, the current study compared cardiovascular reactivity to and recovery from sad and neutral mood induction. Prior work has primarily focused on cardiovascular reactivity rather than recovery; however, it is possible that cardiovascular recovery may increase vulnerability to depression as well as contribute to cardiovascular abnormalities. Third, the current study recruited a sample of participants who have fully recovered from depression and are free from CVD and other related medical illnesses. Accordingly, results would not be attributable to current depression, residual depressive symptoms, or medical comorbidities.

Research Hypotheses

This study examined cognitive, mood, and cardiovascular reactivity and cardiovascular recovery in response to an experimentally-induced sad mood in remitted depression. Based on a

review of the applicable theoretical models and existing literature, the following hypotheses were proposed:

H_1 Formerly depressed participants exposed to the sad mood induction would report significantly higher levels of cognitive reactivity on the DAS post-mood induction than formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

H_2 Formerly depressed and healthy control participants exposed to the sad mood induction would report significantly higher levels of mood reactivity on the VAS post-mood induction than formerly depressed and healthy control participants exposed to the neutral mood inductions.

H_3 Formerly depressed participants exposed to the sad mood induction would exhibit a maladaptive pattern of cardiovascular reactivity (i.e., decreased HP, RSA, and CO and increased PEP) during the mood induction compared to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

H_4 Formerly depressed participants exposed to the sad mood induction would exhibit reduced cardiovascular recovery (i.e., decreased HP, RSA, and CO and increased PEP compared to baseline) during the recovery film compared to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

CHAPTER 5

METHODS AND PROCEDURES

The following methods and procedures were employed. The University of Maine Institutional Review Board (IRB) for the Protection of Human Subjects approved this study (reference number: 2015-09-04, Investigating the Role of Attention and Elaboration in Relapse to Depression), which is a large, ongoing study conducted by the Maine Mood Disorders Lab (MMDL).

Participant Recruitment

Participants included 132 individuals between the ages of 18 to 60 years who were currently undergraduate students enrolled at the University of Maine or individuals residing in the surrounding community. Participants completed online and in-person screening procedures to determine eligibility for the study. Participants that met inclusion and exclusion criteria were invited to participate in the experimental paradigm. Power analysis using G*Power 3.1.9.2 (Faul, Erdfelder, Lang, & Buchner, 2007), a statistical power calculation software, indicated that a sample of 128 participants would result in an 80.00% chance of detecting a medium effect between two groups (i.e., formerly depressed and healthy control participants) exposed to two versions of the experimental paradigm (i.e., sad or neutral mood induction).

Undergraduate Participant Pool Recruitment

Participants included undergraduate students recruited from the University of Maine Department of Psychology undergraduate participant pool. Recruitment was conducted through announcements posted on the Sona Systems (2017), a participant management software. Individuals recruited through the undergraduate participant pool initially completed electronic

screening self-report measures (Appendix C) in Qualtrics (2017), an electronic data capturing system, to determine eligibility for participating in session 1.

Participants recruited through the undergraduate pool were compensated for their participation with research participation credits. Participants were awarded up to two research participation credits for completing session 1 and one research participation credit for completing session 2. Participants who did not complete all study procedures received a prorated rate of research participation credits that reflected the amount of time that they spent in the laboratory (Appendix F). If participants recruited through the undergraduate pool already earned sufficient research participation credit, they were offered monetary compensation for their participation. Participants were paid $30 for completing session 1 and $15 for completing session 2. Participants who did not complete all study procedures received a prorated rate of payment that reflected the amount of time that they spent in the laboratory.

Community Recruitment

Participants also included individuals recruited from the community surrounding the University of Maine. Participants were recruited as part of a larger, ongoing study conducted by the MMDL. Recruitment was conducted through electronic flyers (Appendix A) posted on online announcement boards (i.e., University of Maine Announcements listserv, which was accessible to faculty, staff, and students at the university), online classified advertisement and social media websites (i.e., Craigslist and Facebook), and printed flyers placed in public areas within the surrounding community (i.e., local business and restaurants). Individuals recruited from the surrounding community completed electronic screening self-report measures (Appendix C) in Qualtrics (2017) to determine eligibility for participating in session 1.

Individuals recruited from the surrounding community were paid for their participation. Participants were paid $30 for completing session 1 and $15 for completing session 2. Participants who did not complete all study procedures received a prorated rate of payment that reflected the amount of time that they spent in the laboratory (Appendix G).

Experimenters

The primary author, Olivia E. Bogucki, served as the primary experimenter for this study. Study staff included clinical psychology graduate students and undergraduate research assistants who have completed the Collaborative Institutional Training Initiative (CITI) online training required by the IRB at the University of Maine. Clinical psychology graduate students scored self-report measures during screening, completed diagnostic clinical interviews during session 1, and determined eligibility during screening and session 1. Undergraduate research assistants aided in participant recruitment (e.g., posted advertisements and flyers and contacted participants), obtained informed consent and administered self-report measures during session 1 and 2, attached physiological sensors and monitored physiological recordings during session 2, and conducted self-report and psychophysiological data cleaning. Undergraduate research assistants were trained and supervised by clinical psychology graduate students. Clinical psychology graduate students were supervised by the MMDL Director and Principal Investigator, Emily A. P. Haigh, Ph.D.

Screening

Screening (Table 2) to determine eligibility for study session 1 participation was completed remotely. Advertisements (Appendix A) and the Sona Systems (2017) directed participants to complete an online survey hosted through Qualtrics (2017). Participants were presented with an informed consent document (Appendix B). The informed consent document

clearly stated that the purpose of this survey was to determine eligibility for the study. In addition, the informed consent document highlighted that participation in the study was voluntary and information obtained during the study would remain confidential. More specifically, participants were informed that data would be stored via a secure server, identification numbers would be assigned to de-identify their responses, and the subject key matching participant names and identification numbers would be encrypted and saved on an alternate computer. Despite these precautions, participants were made aware of potential risks associated with the study (e.g., loss of privacy and potential for emotional discomfort) as well as potential benefits (e.g., assistance in helping to better understand the study variables).

After electronically providing informed consent, participants were asked to provide their contact and demographic information and complete self-report measures (Appendix C). Self-report measures were presented in a standardized order and assessed current and past depressive (i.e., Beck Depression Inventory – Second Edition (BDI-II) and Patient Health Questionnaire – 9 (PHQ-9)) and current anxiety (i.e., Beck Anxiety Inventory (BAI)) symptom severity as well as language and visual abilities, learning disabilities, and current and past health conditions (i.e., General Health Screening (GHS)). Self-report measures were used to determine eligibility for participation in session 1.

Following the completion of the self-report measures, participants received a referral list (Appendix D). This referral list was presented as an information source, not something that must be followed. Finally, participants were alerted that they would be contacted via email if they were eligible to participate in the study.

Self-Report Measures

The following self-report measures were collected at screening.

BDI-II. The BDI-II (Beck, Steer, & Brown, 1996a; Appendix C) was used to evaluate the severity of current depressive symptoms. The BDI-II is a 21-item self-report measure that assesses cognitive, affective, somatic, and vegetative symptoms of depression. Respondents rate the severity of depressive symptoms experienced over the past two weeks on a scale from 0 to 3, with 0 indicating the symptom is not present and 3 indicating that the symptom is present and severe. Total scores range from 0 to 63, with higher scores indicating greater levels of depressive symptom severity.

Research on the reliability of the BDI-II indicates excellent internal consistency (α = .91-.94; Arnau, Meagher, Norris, & Bramson, 2001; Beck, Steer, Ball, & Ranieri, 1996b; Dozois, Dobson, & Ahnberg, 1998; Osman et al., 1997a; Steer, Ball, Ranieri, & Beck, 1997) and adequate test-retest reliability (Beck et al., 1996a). Research on the validity of the BDI-II indicates adequate construct validity; convergent validity was evidenced by strong to moderate correlations with other measures of depression and perceived mental health (Beck et al., 1996a; Arnau et al., 2001; Dozois et al., 1998; Steer et al., 1997) while discriminant validity was evidenced by low correlations with measures of social desirability (Osman et al., 1997a).

PHQ-9. The PHQ-9 (Kroenke, Spitzer, & Williams, 2001; Appendix C) was used to evaluate the severity of past depressive symptoms. The PHQ-9 is a 9-item self-report measure that assesses cognitive, affective, and vegetative symptoms of depression. Respondents rate the severity of the worst depressive symptoms experienced over the course of their lifetime during any two-week period on a scale from 0 to 3, with 0 indicating the symptom is not or rarely present and 3 indicating that the symptom is present nearly every day. Total scores range from 0 to 27, with higher scores indicating greater levels of depressive symptom severity.

Research on the reliability of the PHQ-9 indicates good internal consistency (α = .86-.89) and adequate test-retest reliability (Kroenke et al., 2001). Research on the validity of PHQ-9 indicates adequate convergent validity with other measures of depression and psychological distress (Martin, Rief, Klaiberg, & Braehler, 2006).

GHS. The GHS (Appendix C) was created by the MMDL to identify potential confounding variables that other researchers have excluded for when examining psychophysiological reactivity and recovery in remitted MDD (e.g., Bylsma et al., 2014; Chang et al., 2013; Salomon et al., 2013; Wilson et al., 2016). The GHS is a 9-item self-report measure that assesses language and visual abilities and learning disabilities that could significantly impair an individual's ability to understand the experimental paradigm as well as a range of current and past health conditions that could impact the recording of physiological responses. Respondents indicate the presence or absence of such conditions by selecting *Yes* or *No* to each question. In addition, respondents are provided a free response textbox to report more detailed information.

BAI. The BAI (Beck & Steer, 1990; Appendix C) was used to evaluate the severity of current anxiety symptoms. The BAI is a 21-item self-report measure that assesses cognitive, affective, and somatic symptoms of anxiety. Respondents rate the severity of anxiety symptoms experienced over the past two weeks on a scale from 0 to 3, with 0 indicating the symptom is not present and 3 indicating that the symptom is present and severe. Total scores range from 0 to 63, with higher scores indicating greater levels of anxiety symptom severity.

Research on the reliability of the BAI indicates excellent internal consistency (α = .90-.92; Beck, Epstein, Brown, & Steer, 1988; Fydrich, Dowdall, & Chambless, 1992; Osman, Barrios, Aukes, Osman, & Markway, 1993; Osman, Kopper, Barrios, Osman, & Wade, 1997b; Steer & Ranieri, 1993) and adequate test-retest reliability (Beck et al., 1988; Fydrich et al.,

1992). Research on the validity of the BAI indicates adequate construct validity; convergent validity was evidenced by moderate correlations with other measures of anxiety while discriminant validity was evidenced by low to moderate correlations with measures of depression (Beck et al., 1988; Fydrich et al., 1992; Osman et al., 1997b; Steer & Ranieri, 1993).

Eligibility Criteria

The screening phase included general eligibility criteria that was created for all participants and specific eligibility criteria that was created for formerly depressed and healthy control participants. Participants who met general and specific eligibility criteria during screening were sent an email (Appendix E) that included information on how to schedule session 1 using the Sona Systems (2017).

All participants. Participants were required to have been between 18 and 60 years of age. The GHS was used to assess for a multitude of different physical and psychological conditions. To ensure that participants were able to follow instructions associated with the experimental paradigm, participants were deemed ineligible for the study if they did not speak and read English fluently, were color blind, or had been diagnosed with a learning disability that interferes with their ability to read or process visual information. To diminish the likelihood of physical conditions known to impact the recording of physiological responses, participants were deemed ineligible for the study if they had experienced head trauma resulting in a loss of consciousness for over one hour, stroke, hemorrhage, brain tumors, medication-dependent diabetes, cardiovascular disease, heart disease, hypertension, or medical conditions specific to the central nervous system (i.e., epilepsy, transient ischemic attack, multiple sclerosis, amyotrophic lateral sclerosis, Alzheimer's disease, Parkinson's disease, and Huntington's

disease). Finally, participants were deemed ineligible for the study if they had undergone brain or neural surgery or brain radiation treatment.

In addition to the GHS, eligibility for healthy control participants was assessed using the BDI-II, PHQ-9, and BAI. The BDI-II was used to assess current depressive symptoms during the past two weeks while the PHQ-9 was used to assess previous depressive symptoms across the lifespan. The BAI was used to assess current anxiety symptoms during the past two weeks.

Formerly depressed participants. Formerly depressed participants were included in session 1 if they obtained a score of less than 9 on the BDI-II, which is indicative of minimal current depressive symptoms, and a score equal to or greater than 10 on the PHQ-9, which is indicative of moderate to severe past depressive symptoms. Formerly depressed participants were included in session 1 regardless of BAI scores as the presence of comorbid anxiety disorders was permissible.

Healthy control participants. Healthy control participants were included in session 1 if they obtained a score equal to or less than 8 on the BDI-II and PHQ-9, which is indicative of minimal current and past depressive symptoms. Healthy control participants were ineligible for session 1 if they obtained a score greater than 6 on the BAI. This exclusion criterion was determined based on research examining the optimal cut score for various anxiety disorder diagnoses (Leyfer, Ruberg, & Woodruff-Borden, 2006) and was intended to reduce the likelihood that potential healthy control participants would ultimately be excluded after session 1 due to the presence of any major DSM-IV diagnosis.

Session 1

Session 1 (Table 2) took place in the MMDL space located in the Innovative Media, Research, and Commercialization Center (IMRC) on the University of Maine campus.

Participants were greeted by an undergraduate research assistant trained in the standardized study procedures. The undergraduate research assistant introduced the study with the following statement: *"The purpose of the research is to learn about the emotional and physiological responses related to sad mood."* The undergraduate research assistant reviewed the informed consent document (Appendix H) with the participant, highlighting that participation in the study was voluntary and information obtained during the study would remain confidential. More specifically, participants were informed that data would be stored via a secure server, identification numbers would be assigned to de-identify their responses, and the subject key matching participant names and identification numbers would be encrypted and saved on an alternate computer. Despite these precautions, participants were made aware of potential risks associated with the study (e.g., loss of privacy and potential for emotional discomfort) as well as potential benefits (e.g., assistance in helping to better understand the study variables). After the undergraduate research assistant checked for comprehension and answered any questions, informed consent was obtained from the participant.

Participants were asked to provide demographic information and complete self-report measures (Appendix I) in Qualtrics (2017) on an electronic tablet. Self-report measures were presented in a randomized order and assessed current depressive (i.e., BDI-II) and anxiety (i.e., State-Trait Anxiety Inventory – I & II (STAI-I & II)) symptoms. Self-report measures were used as potential covariates. Following the completion of the self-report measures, clinical psychology graduate students obtained the Treatment History self-report measure to identify past and current therapeutic and psychopharmacological interventions. Current and past CBT and antidepressant medication use were used as potential covariates. Clinical psychology graduate students then conducted Structured Clinical Interview for DSM-IV-TR – Research Version (SCID-IV-RV) that

had been adapted in accordance with the DSM-5 with participants to further determine eligibility for participation in session 2. The clinical psychology graduate students recorded diagnoses that the participant endorsed on paper, which were later transferred to a de-identified electronic spreadsheet.

If a participant endorsed current suicidal ideation or intent, the clinical psychology graduate student completed a suicide risk assessment (Appendix J) and consulted with a licensed clinical psychologist affiliated with the University of Maine. If hospitalization was deemed necessary, the clinical psychology graduate student encouraged the participant to voluntarily go to the emergency department for an evaluation. The clinical psychology graduate student accompanied the individual to the hospital by following the participant in their own vehicle. If the participant declined to self-admit themselves to the emergency department and there was imminent risk to the participants' safety, the clinical psychology graduate student called law enforcement to escort the participant to the emergency department.

Following the completion of the SCID-IV-RV, participants received a referral list to the community counseling services as a potential resource (Appendix D). This referral list, which was presented as an information source, and not something that must be followed, was presented by a clinical psychology graduate student with the following statement: *"This referral list is provided for your information. If/when you would like counseling for distressing issues, these are some of the available options in this area. The list includes a variety of resources, some of which are low cost while others vary based on an hourly rate."*

The clinical psychology graduate student obtained height, weight, and waist and hip circumference measurements. Height and weight measurements were used to calculate body mass index (BMI). After all study procedures were completed, participants were thanked for

their participation during session 1. Participants were compensated for their participation of approximately two hours; participants recruited through the undergraduate participant pool were awarded up to two research participation credits depending on the amount of time spent in the laboratory while participants recruited from the surrounding community were awarded $30 payment.

Finally, clinical psychology graduate students determined eligibility for session 2 based on the eligibility criteria. Eligible participants were invited to participate in session 2 with the following statement: *"Based on this interview it appears that you qualify to complete an additional portion of this study that takes approximately one to two hours. This session will be worth one to two credits. If you are interested, we'd request you avoid wearing a dress, overalls, or a turtleneck shirt due to the physiological recordings we will be taking."* Clinical psychology graduate students enrolled interested participants in session 2 using the Sona Systems (2017). Ineligible participants were alerted that they are not eligible for the remainder of the study with the following statement: *"We are recruiting individuals who answer interview questions in a very specific way, and according to your responses you do not qualify for session 2 at this time. Thank you for your participation and we will be updating your Sona account with credits from this session within the following month."*

Self-Report Measures

The following self-report measures were collected at session 1.

BDI-II. The BDI-II (Beck, Steer, & Brown, 1996a; Appendix C) was used to evaluate the severity of current depressive symptoms at baseline.

STAI-I & II. The STAI-I & II (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983; Appendix I) was used to evaluate the severity of current anxiety symptoms. The STAI-I & II

contains two 20-item self-report measures that assesses cognitive, affective, and somatic symptoms of both state and trait anxiety. Respondents rate the severity of anxiety symptoms experienced at the moment for the state version and in general for the trait version from 1 to 4, with 1 indicating the symptom is almost never present and 4 indicating that the symptom is almost always present. Total scores for each version range from 20 to 80, with higher scores indicating greater levels of state or trait anxiety symptom severity.

Research on the reliability of the STAI-I & II indicates good to excellent internal consistency (α = .86-.95; Balsamo et al., 2016; Spielberger et al., 1993) and adequate test-retest reliability (Spielberger et al., 1993). Research on the validity of the STAI-I & II indicates adequate construct validity; convergent validity was evidenced by increased scores on the state form during stressful situations while discriminant validity was evidenced by decreased scores on the state form during relaxing situations (Spielberger, Gorsuch, & Luschene, 1970; Spielberger, 1983, 1989). In addition, adequate concurrent validity with other measures of similar affective states for the trait form has been found (Spielberger et al., 1970; Spielberger, 1989).

Interview Measures

The following interview measures were collected at session 1.

Treatment history. The Treatment History (Appendix C) self-report measure was created by the MMDL to identify current and past therapeutic and psychopharmacological interventions that other researchers have excluded for when examining cognitive, mood, and psychophysiological reactivity and recovery in remitted MDD (e.g., Lethbridge & Allen, 2008; Yaroslavsky et al., 2014 Studies 1 and 2). The Treatment History self-report measure is an 8-item clinician-administered self-report measure that assesses current and past therapy and

medication use for emotional or behavioral problems. Respondents are asked to indicate the presence or absence of such conditions by answering *Yes* or *No* to each question. In addition, respondents are asked report more detailed information about the types of therapy received (e.g., CBT) and medication prescribed (e.g., antidepressants).

SCID-IV-RV. The SCID-IV-RV (First, Gibbon, Spitzer, & Williams, 1995) was administered by clinical psychology graduate students trained in administration and scoring by the MMDL Director, Emily A. P. Haigh, Ph.D. The Director was available for supervision and consultation when necessary. The SCID-IV-RV is a semi-structured clinical interview that assesses current and past major DSM-IV clinical diagnoses based on the diagnostic criteria outlined in the Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition – Text Revision (DSM-IV-TR). The SCID-IV-RV had been adapted by the MMDL to be in accordance with the DSM-5 diagnostic criteria. Clinical interviews were audio-recorded to conduct fidelity checks and estimate inter-rater reliability.

Eligibility Criteria

Session 1 includes specific eligibility criteria for formerly depressed and healthy control participants. Individuals who met specific eligibility criteria during session 1 were scheduled for session 2.

Formerly depressed participants. Formerly depressed participants were included in the remainder of the study if they met diagnostic criteria for a past episode of MDD according to DSM-5 criteria (APA$_A$, 2013). Participants were excluded from the remainder of the study if they met diagnostic criteria for current MDD within the past month, current substance abuse within the past 6 months, current or past substance dependence, bipolar disorder, psychotic disorder, acute suicidal ideation, or mood episodes secondary to general medical conditions.

Healthy control participants. Healthy control participants were included in the remainder of the study if they were free from any current or past psychological disorder. Healthy control participants were excluded from the remainder of the study if they met diagnostic criteria for any current or past major DSM-5 diagnosis including mood, psychotic, substance use, anxiety, or eating disorders.

Session 2

Session 2 (Table 2) took place in the MMDL space located in Corbett Hall on the University of Maine campus. Participants were greeted by an undergraduate research assistant trained in the standardized study procedures. The undergraduate research assistant provided an overview of the study procedures with the following statement: *"Thank you for returning for session 2 of this study. We are interested in investigating the physiological effects of different mood states, so today we will measure your physiological responses to a video and some audio clips. There will also be some additional questionnaires for you to complete."* As part of the informed consent procedure, the undergraduate research assistant reminded the participant that their participation was entirely voluntary and that they could discontinue at any time without penalty (Appendix K). After the undergraduate research assistant checked for comprehension and answered any questions, informed consent was obtained from the participant.

Once in the physiological laboratory, participants answered questions about skin sensitivity and allergies to electrode gel, medical tape, or Band-Aids. Participants were asked to remove their jewelry and place it with other personal belongings (e.g., cell phones). Next, participants were asked to wash their hands with glycerin-rich soap and prompted to use the restroom, if necessary. Noninvasive electrode sensors were placed by a female undergraduate research assistant referred to as the experimenter. Before placement of the electrode sensors,

participants were briefed on electrode sensor placement and verbally alerted. While attaching electrode sensors, the experimenter engaged the participants in conversation to help them feel comfortable. Areas where electrode sensors were placed were cleaned with an abrasive alcohol swab.

During electrode sensor placement, another undergraduate research assistant referred to as the monitor examined the associated waveforms in Biolab 3.1, a physiological acquisition software created by MindWare Technologies Ltd. (2009), to ensure that electrode sensors were accurately placed. After the electrodes were correctly placed, the experimenter asked the participant to sit in a comfortable chair in front of a computer screen with uncrossed legs for the remainder of the study. The monitor selected the correct paradigm based on a predetermined randomization table. The participant completed the experimental paradigm including; baseline video, self-report measures, sad or neutral mood induction, self-report measures, recovery video, and self-report measures while physiological responding was continuously recorded.

After completion of the experimental paradigm, the experimenter assisted the participant in the removal of electrode sensors. The experimenter reviewed a debriefing form (Appendix M) with the participant and answered any questions about the study. After all study procedures were completed, participants were thanked for their participation during session 2. Participants were compensated for their participation of approximately one hour; participants recruited through the undergraduate participant pool were awarded one research participation credit while participants recruited from the surrounding community were awarded $15 payment.

Experimental Paradigm

The experimental paradigm was presented using Experimenter's Prime (E-Prime; Experimenter's Prime, 2015), an experimental research software suite. E-Prime (2015) enables

the creation and presentation of experimental paradigms as well as the collection and investigation of experimental data.

Baseline video. Participants completed a baseline period intended to allow physiological responses to normalize following electrode placement. Participants were prompted by instructions on the computer screen to put on over-ear headphones, sit still, and quietly watch a 10-minute neutrally valenced travel video about Alaska's Denali National Park (Kolbeinsson, 2016). The video consisted of plants, animals, weather, and geographical scenes set to instrumental music with minimal dialogue.

Self-report measures. Following the baseline period, participants were prompted by instructions on the computer screen to complete the first set of self-report measures (Appendix L) in Qualtrics (2017) on an electronic tablet. Self-report measures were presented in a randomized order and assessed depression-related thoughts (i.e., Dysfunctional Attitudes Scale – Short Form I (DAS-SF I)) and feelings (i.e., VAS and Positive and Negative Affect Schedule – Expanded Form (PANAS-X)).

Mood induction. Following the first set of self-report measures, participants were prompted by instructions on the computer screen to complete the sad or neutral mood induction. Prior to the sad mood induction, participants completed a 40-word sad (e.g., doomed, crying, hurt, etc.) or neutral (e.g., note, dial, zoom, etc.) emotional Stroop task that was part of a larger, ongoing study conducted by the MMDL. Words appeared individually on the computer screen, printed in red, green, yellow, or blue and participants were instructed to select the matching color key on the keyboard (i.e., f for red, g for green, h for yellow, and j for blue). Before each word is presented, a fixation cross (i.e., +) appeared on the screen for 700 milliseconds to help

participants focus their attention. Participants were collapsed into two groups based on mood induction condition (i.e., sad or neutral).

Sad mood induction. The sad mood induction methodology used a combination of music and autobiographical recall to create a mild, transient sad mood. This method has been empirically validated by previous research (e.g., Martin, 1990; Segal et al., 1999, 2006). Participants listened to a digitally re-mastered, half-speed, non-lyrical 7:38-minute piece of classical music entitled "Russia under the Mongolian Yoke" by Prokofiev. Simultaneously, participants were prompted to recall a time in their lives when they felt sad with the following statement: *"During this task, you will listen to a 7-minute piece of classical music on the computer. Please listen to the music and think about a specific time or situation when you felt depressed and/or low. If you find that your mind wanders, please go back to thinking about the specific time or situation when you felt depressed and/or low."* This text remained on the computer screen for the entire neutral mood induction.

Neutral mood induction. The neutral mood induction methodology used a combination of music and autobiographical recall to serve as a control condition. This mood induction method has been empirically validated by previous research, which showed that it does not result in a significant change in mood (e.g., Green, Sedikides, Saltzberg, Wood, & Forzano, 2003; Wood, Saltzberg, & Goldsamt, 1990). Participants listened to a digitally re-mastered, half-speed, non-lyrical 7:38-minute selection of classical music (i.e., Waltzes No. 11 in G flat, Op. 70, No. 1 and No. 12 in F minor, and Op. 70, No. 2 by Chopin). Simultaneously, participants were prompted to recall an uneventful day in their life that was neither especially happy nor sad with the following statement: *"During this task, you will listen to a 7-minute piece of classical music on the computer. Please listen to the music and think about a specific but unemotional day in detail.*

For example, this could be a typical day at school or work when everything followed your typical routine. If you find that your mind wanders, please go back to thinking about the specific but unemotional day." This text remained on the computer screen for the entire neutral mood induction.

Self-report measures. Following the mood induction, participants were prompted by instructions on the computer screen to complete a second set of self-report measures (Appendix L) in Qualtrics (2017) on an electronic tablet. Self-report measures were presented in a randomized order and assessed depression-related thoughts (i.e., Dysfunctional Attitudes Scale – Short Form II (DAS-SF II)) and feelings (i.e., VAS, PANAS-X).

Recovery video. Following the second set of self-report measures, participants completed a recovery period intended to evaluate the amount of time that it took for physiological responses to return to baseline levels. Participants were instructed on the computer screen to sit quietly while watching a different 10-minute neutral travel video about Alaska's Last Frontier. The video consisted of plants, animals, weather, and geographical scenes set to instrumental music with minimal dialogue. This recovery procedure was selected to increase similarity to the baseline procedure. In addition, this recovery procedure, which was passive yet attentionally demanding, was selected in lieu of a silent recovery to reduce the potential impact of cognitive processes (e.g., rumination) on recovery and minimize feelings of sadness before participants leave the laboratory (Linden et al., 1997).

Self-Report Measures

The following self-report measures were collected at session 2.

DAS-SF I & II. The DAS-SF I & II (Beevers, Strong, Meyer, & Pilkonis, 2007; Appendix L), an abbreviated version of the original Dysfunctional Attitudes Scale – Forms A

and B (DAS; Weissman & Beck, 1978), was used to evaluate changes in dysfunctional beliefs about oneself before and after the mood induction. The DAS-SF I & II are each 9-item self-report measures that assess an individual's beliefs about his or her self. Respondents rate their beliefs experienced most of the time on a scale of 1 to 4, with 1 indicating that they totally agree with the statement and 4 indicating that they totally disagree with the statement. Total scores range from 9 to 36, with lower scores indicating greater levels of the dysfunctional beliefs. Two versions of the DAS-SF have been created to reduce test-retest effects. Qualtrics (2017) does not allow the randomization of self-report measures across a multi-block experimental paradigm, so the DAS-SF I & II were presented in a fixed order; the DAS-SF I was always be presented before the mood induction while the DAS-SF II was always be presented after the mood induction.

Research on the reliability of the DAS-SF I & II indicates good internal consistency (*a* = .83-.94) and adequate test-retest reliability (Beevers et al., 2007). Research on the validity of DAS-SF I & II indicates adequate convergent validity as evidenced by moderate correlations with other measures of dysfunctional attitudes and adequate predictive validity as evidenced by significant prediction of posttreatment depressive symptom severity scores by pretreatment DAS I & II scores (Beevers et al., 2007). In addition, Beevers and colleagues (2007) found that there were no significant differences in residualized change scores for the DAS-A, DAS I, and DAS II (*p's* = .79-.93, *d's* = .00-.01) and the residualized change scores for the DAS-A, DAS I, and DAS II were very strongly correlated (*r's* = .84-.91), suggesting that the long and short forms of the DAS perform similarly.

VAS. The VAS (Appendix L) was used to evaluate subjective changes in mood before and after the mood induction. Respondents rate their current level of sadness on a scale of 0 to

100, with 0 indicating lower levels of sadness and 100 indicating higher levels of sadness. Total scores range from 0 to 100, with higher scores indicating greater levels of sadness. Participants were presented with a 100-millimeter line on an electronic tablet anchored by "not at all" at 0 and "extremely" at 100.

The VAS has been empirically validated by previous research. Studies have shown that the VAS is sensitive to change in emotion and stress states when standardized measures cannot be obtained due to time or experimental constraints (Cella & Perry, 1986). Change in sadness served as a manipulation check for sad and neutral mood inductions to ensure that they produced their intended moods. Research on the reliability of the VAS indicates adequate test-retest reliability (Cella & Perry, 1986; Folstein & Luria, 1973). Research on the validity of VAS indicates adequate concurrent validity with other measures of similar affective states (Cella & Perry, 1986; Folstein & Luria, 1973; Little & McPhail, 1973; Davies, Burrows, & Poynton, 1975).

PANAS-X. The PANAS-X (Watson & Clark, 1994; Appendix L), an expanded version of the original Positive and Negative Affect Schedule (PANAS; Watson et al., 1988), was used to evaluate changes in mood before and after the mood induction. The PANAS-X is a 60-item self-report measure that assess affect, including two general dimension scales (i.e., negative (PANAS-X N) and positive affect (PANAS-X P)), four basic negative emotional scales (i.e., fear (PANAS-X F), guilt (PANAS-X G), hostility (PANAS-X H), and sadness (PANAS-X S)), three basic positive emotional scales (i.e., joviality, self-assurance, and attentiveness), and four other affective states (i.e., shyness, fatigue, serenity, and surprise). Respondents rate the extent to which they are experiencing 60 affective adjectives on a Likert scale from 1 to 5, with 1 indicating that they have not or very slightly experienced the affective state and 3 indicating that

they have experienced the experienced the affective state extremely. Total scores for the general dimension scales are calculated by summing the 10 affective adjectives that comprise each scale and range from 10 to 50, with higher scores indicating greater levels of the respective affect. Total scores for specific affective state scales are calculated by summing the five (i.e., sadness) or six (i.e., fear, guilt, and hostility) affective adjectives that comprise each scale and range from 5 to 25 (i.e., sadness) or 6 to 30 (i.e., fear, guilt, and hostility), with higher scores indicating greater levels of the respective affect. Given this investigation's focus on negative emotionality, the negative and positive affect general dimension scales and basic negative emotional scales (i.e., fear, guilt, hostility, and sadness) were used.

Research on the reliability of the PANAS-X indicates excellent to good internal consistency (a = .83-.90) for the general dimension scales and excellent to acceptable internal consistency (a = .76-.93) for the specific affective state scales as well as adequate test-retest reliability (Watson & Clark, 1994). Research on the validity of PANAS-X indicates adequate construct validity; convergent validity was evidenced by strong correlations with other measures of similar affective states while discriminant validity was evidenced by moderate correlations with other measures of dissimilar affective states (Watson & Clark, 1994; Watson & Clark, 1997).

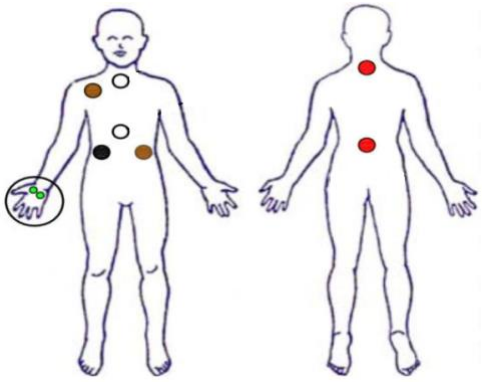
Cardiovascular Measures

Physiological responding was recorded throughout the entire experimental paradigm. The physiological recordings of interest were cardiovascular measures derived from ECG and ICG. In addition, as part of a larger ongoing study, two electrode sensors filled with isotonic electrode paste were placed on the heal of the participants' non-dominant hand to collect Galvanic Skin Response (GSR).

ECG. Five Galvanic Skin Conductance (GSC) electrode sensors were filled with electrode gel and placed on the participants' chest to measure the electrical activity of the heart and collect data that was used to calculate HP and RSA. MindWare Technologies Ltd. (2009) hardware and Biolab 3.1 analysis software set to collect ECG data falling within -5 and 5 volts with a sampling rate of 1,000 hertz were utilized in conjunction with the five GSC electrode sensors located on the participants' right collarbone, bottom left rib, bottom right rib, jugular notch, and sternum (Figure 6).

ICG. Two GSC electrode sensors were filled with electrode gel and placed on the participants' chest and back to measure the electrical activity of the heart and collect data that was used to calculate RSA, CO, and PEP. MindWare Technologies Ltd. (2009) hardware and Biolab 3.1 analysis software set to collect ICG data falling at a sampling rate of 1,000 Hertz and calibrated at .10 volts per one-ohm change for Z0 and 1.00 volts per ohms per second for dZ/dt were utilized in conjunction with the two GSC electrode sensors located on the participants' mid-back and upper-back parallel within 1.50 inches of the jugular notch and sternum sensors (Figure 6).

Figure 6. *Sensor Placement from MindWare Technologies Ltd. (2009)*



Note. Session 2 electrode placement for physiological data collection. Brown, white, and black circles represent GSC electrodes for ECG. Red circles represent GSC electrodes for ICG. Green circles represent GSR electrodes for GSR.

Table 2. *Study Procedure Chart*

Time	Task Category	Task Description
Screening	Self-report measures	Contact and demographic information GHS: language and visual abilities, current and past health conditions BDI-II: current depressive symptoms PHQ-9: past depressive symptoms BAI: current anxiety symptoms
Session 1	Self-report measures	Demographic information: age, sex, race, ethnicity, marital status, and education level BDI-II: current depressive symptoms STAI-I & II: current anxiety symptoms
	Physical measure	BMI
	Interview	Treatment History: current and past CBT and antidepressant use
		SCID-IV-RV: psychiatric diagnoses
Session 2	Baseline	ECG and ICG recording while viewing 10-minute clip of Alaska Denali Park Video
	Self-report measures	DAS-SF I: dysfunctional beliefs at baseline (i.e., baseline cognitions) VAS: dysphoric mood at baseline (i.e., baseline mood) PANAS-X: dysphoric mood at baseline (i.e., baseline mood)
	Mood induction	ECG and ICG recording while listening to 7:38-minute piece of sad/neutral music and recalling sad/neutral autobiographical memory
	Self-report measures	DAS-SF II: dysfunctional beliefs post-mood induction (i.e., cognitive reactivity to mood induction) VAS: dysphoric mood post-mood induction (i.e., mood reactivity to mood induction) PANAS-X: dysphoric mood post-mood induction (i.e., mood reactivity to mood induction)
	Recovery	ECG and ICG recording while viewing 10-minute clip of Alaska Wilderness Video
	Debriefing	Provide and review debriefing form

Note. GHS = General Health Screen; BDI-II = Beck Depression Inventory – Second Edition; PHQ-9 = Patient Health Questionnaire – 9; BAI = Beck Anxiety Inventory; STAI-I & II = State-Trait Anxiety Inventory – I & II; BMI = body mass index; SCID-IV-RV = Structured Clinical Interview for DSM-IV-TR – Research Version; ECG = electrocardiogram; ICG = impedance cardiography; DAS I = Dysfunctional Attitudes Scale – Short Form I; DAS II = Dysfunctional Attitudes Scale – Short Form II; VAS = Visual Analogue Scale; PANAS-X = Positive and Negative Affect Scale – Expanded Form.

CHAPTER 6

ANALYSES AND HYPOTHESIZED RESULTS

All analyses were conducted using IBM SPSS Statistics Version 25.0 (IBM Corporation, 2017).

Preliminary Analyses

Data Cleaning and Calculation

The following procedures were used to clean demographic, self-report, cognitive, mood, and cardiovascular data and calculate change scores for cognitive, mood, and cardiovascular data before analyses were conducted. All data were manually inspected for potential univariate outliers, defined as z-scores exceeding ± 3.00 (Daszykowski, Kaczmarek, Heyden, & Walczak, 2007). Winsorizing, a data transformation procedure that retains outliers by adjusting extreme values to the next most non-outlier extreme value, was utilized if necessary. Winsorization is an alternative to deleting outliers that reduces the skew of the distribution while preserving the general pattern of variability (Field, 2009). Outlier data were winsorized to address extreme values at the group by condition level. Outliers for cognitive, mood, and cardiovascular data was addressed differently depending on the type of analyses conducted. For analyses conducted using difference scores, outliers for pre- and post-mood induction data were not winsorized as participants' data points were dependent upon one another and would impact the validity of the difference scores. For analyses conducted using residualized change scores or repeated measures, outliers for pre- and post-mood induction data were winsorized as participants' data points were aggregated and therefore, would not impact the validity of the residualized change scores and pre- and post-mood induction data points.

Of note, this study drew from two distinct literature bases (i.e., cognitive and mood literature and cardiovascular literature) that use different techniques to calculate change scores. Over the years, there has been a fierce debate in the literature about the use of change scores (see Cronbach & Furby, 1970 for the argument against the use of change scores and Zimmerman & Williams, 1982a, 1982b for rebuttals). Today, the literature has generally come to the consensus that the difference score and residualized change score methods for calculating change scores are reliable and valid methods for assessing reactivity (Castro-Schilo & Grimm, 2018; Dimitrov & Rumrill, 2003; Linden et al., 1997; Llabre et al., 1991). To represent the conventions of each literature base, two different data calculation techniques for change scores were used: difference scores, which are typically used in the cardiovascular literature, and residualized change scores, which are typically used in the cognitive and mood literature.

Demographic information. Demographic information collected during session 2 (i.e., age, sex, race, ethnicity, marital status, education level, current and past CBT and antidepressant use, and BMI) were manually inspected for potential univariate outliers.

Self-report measures. Self-report measures collected during session 2 (i.e., BDI-II, STAI-I & II) were manually inspected for potential univariate outliers.

Cognitive measures. Cognitive measures collected during session 2 (i.e., DAS-SF I & II) were manually inspected for potential univariate outliers.

Difference scores for cognitive reactivity were calculated for the DAS by subtracting post-mood induction scores and pre-mood induction scores (i.e., $DAS_{POST} - DAS_{PRE}$). These values were used in the subsequent analyses to represent cognitive (i.e., DAS_{DS}) reactivity to the mood induction procedures. Previous research has shown difference scores are reliable measures of self-reported reactivity with the exception of cases in which pretest and post test scores have

equal variance and equal reliability (Dimitrov & Rumrill, 2003), which was not hypothesized for this study.

Simple linear regression for cognitive reactivity were used to create residualized change scores for the pre- and post-mood induction DAS. These values were used in the subsequent analyses to represent cognitive (i.e., Z_{RES}DAS) reactivity to the mood induction procedures. This technique has been used in studies investigating similar hypotheses and employing similar methodological procedures in an effort to make variability of pre-mood induction DAS scores independent from variability of post-mood induction DAS scores (Segal et al., 2006; Van Rijsbergen et al., 2013). Previous research has shown that while residualized change scores are reliable measures of self-reported reactivity and results in less error than difference scores when pre-test score variance is greater than posttest score variance (Dimitrov & Rumrill, 2003), which could have been the pattern of responding observed during this study. Of note, the use of residualized change scores can lead to an overly conservative test for self-reported reactivity (Dimitrov & Rumrill, 2003).

Mood measures. Mood measures collected during session 2 (i.e., VAS, and PANAS-X N, P, F, G, H, and S) were manually inspected for potential univariate outliers.

Difference scores for mood reactivity were calculated for the VAS and PANAS-X N, P, F, G, H, and S by subtracting post-mood induction scores and pre-mood induction scores (i.e., VAS_{POST} –VAS_{PRE}, PANAS-X N_{POST} –PANAS-X N_{PRE}, PANAS-X P_{POST} –PANAS-X P_{PRE}, PANAS-X F_{POST} –PANAS-X F_{PRE}, PANAS-X G_{POST} –PANAS-X G_{PRE}, PANAS-X H_{POST} – PANAS-X H_{PRE}, and PANAS-X S_{POST} –PANAS-X S_{PRE}). These values were used in the subsequent analyses to represent mood (i.e., VAS_{DS}) reactivity to the mood induction procedures. In addition, these values were used in the subsequent analyses as a manipulation

check for the mood induction procedures to ensure that a sad mood was induced (i.e., VAS_{DS}) and to assess if emotions other than sadness were induced (i.e., PANAS-X N_{DS}, PANAS-X P_{DS}, PANAS-X F_{DS}, PANAS-X G_{DS}, PANAS-X H_{DS}, and PANAS-X S_{DS}). Previous research has shown difference scores are reliable measures of self-reported reactivity with the exception of cases in which pretest and post test scores have equal variance and equal reliability (Dimitrov & Rumrill, 2003), which was not hypothesized for this study.

Simple linear regression for cognitive and mood reactivity were used to create residualized change scores for the pre- and post-mood induction VAS and PANAS-X N, P, F, G, H, and S. These values were used in the subsequent analyses to represent mood (i.e., Z_{RES}VAS) reactivity to the mood induction procedures. In addition, these values were used in the subsequent analyses as a manipulation check for the mood induction procedures to ensure that a sad mood was induced (i.e., Z_{RES}VAS) and to assess if emotions other than sadness were induced (i.e., Z_{RES}PANAS-X N, Z_{RES}PANAS-X P, Z_{RES}PANAS-X F, Z_{RES}PANAS-X G, Z_{RES}PANAS-X H, and Z_{RES}PANAS-X S). This technique has been used in studies investigating similar hypotheses and employing similar methodological procedures in an effort to make variability of pre-mood induction VAS scores independent from variability of post-mood induction VAS scores (Segal et al., 2006; Van Rijsbergen et al., 2013). Previous research has shown that while residualized change scores are reliable measures of self-reported reactivity and results in less error than difference scores when pre-test score variance is greater than posttest score variance (Dimitrov & Rumrill, 2003), which could have been the pattern of responding observed during this study. Of note, the use of residualized change scores can lead to an overly conservative test for self-reported reactivity (Dimitrov & Rumrill, 2003).

Cardiovascular measures. Multiple cardiovascular measures were used in this investigation including HP, RSA, CO, and PEP. Cardiovascular measures were calculated from ECG and ICG data. ECG and ICG data were collected with Mindware hardware and Biolab 3.1 (MindWare Technologies Ltd., 2009) acquisition software. ECG data fell within -5 and 5 volts with a sampling rate of 1,000 Hertz. The following calculation methods were used to compute ECG measures: entire for calculation method and Z0 for respiration signal to use. ICG data was sampled at a rate of 1,000 Hertz. Z0 was calibrated at .10 volts per one-ohm change while dZ/dt was calibrated at 1.00 volts per ohms per second. The following calculation methods were used to compute ICG measures: minimum value K to R interval for the ECG Q point (K = 35), the Framingham method for LVET windowing (LVET minimum = 300, LVET maximum = 600), the percentage of dZ/dt time + C (percent dZ/dt peak = 55.00%, C = 4; Lozano et al., 2008) for the ICG B point, the kubieck formula for SV, and measured for dZ/dt source. ECG and ICG data were ensemble averaged using 60 second epochs.

Specialized Biolab software modules (MindWare Technologies Ltd., 2009) were utilized to clean and calculate HP, RSA, CO, and PEP data. All data was visually screened and manually cleaned for artifacts before calculations are computed. HP and RSA were derived using Mindware's HRV Analysis 3.1.4 module. HP was calculated using the time series method to determine the IBI between successive R spikes on the ECG in milliseconds. RSA was calculated using the frequency domain method to determine the IBI between successive R spikes on the ECG within the high frequency band derived from a Fast Fourier Transform, which fell within .15 and .40 Hertz. In addition, the Z0 measure obtained via ICG was used to account for the impact of respiration rate on RSA. CO and PEP was derived using Mindware's Impedance Analysis 3.1.4 module. CO was calculated by multiplying HR by SV. PEP was calculated by

determining the time in milliseconds between the B point of dZ/dt from ICG and the Q point from ECG. Cardiovascular functioning at baseline, cardiovascular reactivity in response to the mood induction, and cardiovascular recovery were calculated by taking an average of the last two and five minutes of HP, RSA, CO, and PEP data for baseline (i.e., $HP_{BL2/BL5}$, $RSA_{BL2/BL5}$, $CO_{BL2/BL5}$, and $PEP_{BL2/BL5}$) and recovery (i.e., $HP_{RC2/RC5}$, $RSA_{RC2/RC5}$, $CO_{RC2/RC5}$, and $PEP_{RC2/RC5}$) and the first two and five minutes of HP, RSA, CO, and PEP data for the mood induction (i.e., $HP_{MI2/MI5}$, $RSA_{MI2/MI5}$, $CO_{MI2/MI5}$, and $PEP_{MI2/MI5}$). Cardiovascular data collected during session 2 were manually inspected for potential univariate outliers.

Difference scores for cardiovascular reactivity were calculated for each cardiovascular measure by subtracting the average obtained during the first two and five minutes of the mood induction from the average obtained during the last two and five minutes of baseline (i.e., $HP_{MI2/MI5} - HP_{BL2/BL5}$, $RSA_{MI2/MI5} - RSA_{BL2/BL5}$, $CO_{MI2/MI5} - CO_{BL2/BL5}$, and $PEP_{MI2/MI5} - PEP_{BL2/BL5}$). These values were used in the subsequent analyses to represent cardiovascular reactivity (i.e., $HP_{DS2RA/DS5RA}$, $RSA_{DS2RA/DS5RA}$, $CO_{DS2RA/DS5RA}$, and $PEP_{DS2RA/DS5RA}$) to the mood induction procedures. In addition, difference scores for cardiovascular recovery were calculated for each cardiovascular measure by subtracting the average obtained during the last two and five minutes of recovery from the average obtained during the last two and five minutes of baseline (i.e., $HP_{RC2/RC5} - HP_{BL2/BL5}$, $RSA_{RC2/RC5} - RSA_{BL2/BL5}$, $CO_{RC2/RC5} - CO_{BL2/BL5}$, and $PEP_{RC2/RC5} - PEP_{BL2/BL5}$). These values were used in the subsequent analyses to represent cardiovascular recovery (i.e., $HP_{DS2RA/DS5RC}$, $RSA_{DS2RA/DS5RC}$, $CO_{DS2RA/DS5RC}$, and $PEP_{DS2RA/DS5RC}$) from the mood induction procedures. This technique has been used in studies investigating similar hypotheses and employing similar methodological procedures to examine cardiovascular reactivity and recovery (Salomone et al., 2013; Yaroslavsky et al., 2013). Previous research has

shown that differences scores are reliable measures of psychophysiological reactivity across multiple experimental sessions with the exception of cases in which pretest and post test scores have equal variance and equal reliability (as reviewed by Llabre, Spitzer, Saab, Ironson, & Schneiderman, 1991), which was not hypothesized for this study.

Simple linear regression for cardiovascular reactivity was used to create residualized change scores. These values were used in the subsequent analyses to represent cardiovascular reactivity (i.e., $Z_{RES2/RES5}HP_{RA}$, $Z_{RES2/RES5}RSA_{RA}$, $Z_{RES2/RES5}CO_{RA}$, and $Z_{RES2/RES5}PEP_{RA}$) to the mood induction procedures. In addition, simple linear regression for cardiovascular recovery was used to create residualized change scores. These values were used in the subsequent analyses to represent cardiovascular recovery (i.e., $Z_{RES2/RES5}HP_{RC}$, $Z_{RES2/RES5}RSA_{RC}$, $Z_{RES2/RES5}CO_{RC}$, and $Z_{RES2/RES5}PEP_{RC}$) from the mood induction procedures. Previous research has shown that that residualized change scores are reliable measures of psychophysiological reactivity across multiple experimental sessions and results in less error than difference scores when pre-test score variance is greater than posttest score variance (as reviewed by Llabre et al., 1991), which could have been the pattern of responding observed during this study.

Data Analysis

The following procedures were used to analyze demographic, cognitive, mood, and cardiovascular data. All data were assessed for homogeneity of variance using Levene's test. Violation of homogeneity of variance ($p < .05$) indicates that the assumption underlying analyses is not met. If homogeneity of variance was violated for independent samples *t* tests, results for equal variances not assumed were reported. If homogeneity of variance was violated for one-way, factorial, or repeated measures analysis of variance (ANOVA), additional analyses were conducted to assess whether violation of homogeneity of variance were driven by the inclusion

of covariates. If homogeneity of variance was significant with covariates and not significant without covariates, calculating the residual of the residualized change scores to assess normality and transforming the residualized change scores to re-assess homogeneity of variance would not have the intended effect. Instead, the planned analyses were conducted with the α level decreased from .05 to .01.

There are multiple possibilities to remedy the violation this assumption which includes using a non-parametric test, transforming the data to reduce skewness, or decreasing the α level to reduce the likelihood of type II error (S. W. Ell, personal communication, February 9, 2016). Multiple steps were explored in an attempt to remedy the violation of this assumption. First, residuals of differences scores were calculated, and Kolmogorov-Smirnov's and Shapiro-Wilk's tests of normality were conducted (Field, 2009). If Levene's test for homogeneity of variance continued to be significant, this indicated that the assumption underlying ANOVA was still not met. Second, residualized change scores were transformed using the reciprocal transformation ($1/X_{iR}$). Of note, residualized change scores were reversed before conducting this transformation to preserve order ($X_{iR} = X_{HIGHEST} - X_i$). This transformation method was selected as the cognitive, mood, and cardiovascular data contained negative values, which cannot be transformed using log and square root transformations (Field, 2009). If Levene's test for homogeneity of variance continued to be significant, this indicated that the assumption underlying ANOVA was still not met. Given that there are not non-parametric tests for evaluating interactions (Grace-Martin, 2019a), the remaining option was to move forward with the planned analyses and decreased the α level from .05 to .01.

Of note, this study drew from two distinct literature bases (i.e., cognitive and mood literature and cardiovascular literature) that use different techniques to analyze data. As

previously noted by researchers (e.g., Castro-Schilo & Grimm, 2018), there is not one correct data analysis technique that can be used to test a study's hypotheses when experimental procedures (e.g., random assignment, use of experimental and control conditions) intended to distribute variability and error across conditions are employed. The calls for a consensus on data analysis techniques for reactivity and recovery have been left unanswered (Linden et al., 1997). To represent the conventions of each literature base, two different data analysis techniques were used: repeated measures ANOVAs, which are typically used in the cardiovascular literature, and factorial ANOVAs, which are typically used in the cognitive and mood literature.

Demographic information. Descriptive statistics were used to summarize the sample. Measures of central tendency and variability were used for continuous variables (i.e., age and BMI) while frequency statistics were used for categorical variables (i.e., sex, race, ethnicity, marital status, education level, and current and past CBT and antidepressant use). A series of independent samples *t*-tests were used to assess significant group differences of continuous variables (i.e., age and BMI) collected during session 1 (Table 3). In addition, a series of chi-square tests were used to assess significant group differences of categorical variables (i.e., sex, race, ethnicity, education level, marital status, and current and past CBT and antidepressant use) collected during session 1 (Table 3). These variables were considered as potential covariates in subsequent analyses.

Self-report measures. A series of independent samples *t*-tests were used to assess significant group differences of current depressive (i.e., BDI-II) and anxiety (i.e., STAI-I & II) symptoms collected during session 1 (Table 3). These variables were considered as potential covariates in subsequent analyses.

Cognitive measures. A series of 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) factorial ANOVAs and a 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) repeated measures ANOVA were conducted to examine changes in dysfunctional beliefs on the DAS pre- and post-mood induction. Analyses were conducted using difference scores (i.e., DAS_{DS}), residualized change scores (i.e., $Z_{RES}DAS$), and pre- and post-mood induction measures (i.e., DAS I and DAS II). These analyses were used to assess significant differences in cognitive reactivity based on depressive history and mood induction procedure. Planned comparisons were conducted using contrast analyses. It was expected that formerly depressed participants exposed to the sad mood induction would report significantly more dysfunctional beliefs compared to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

Mood measures. A series of one-way (condition: sad, neutral) ANOVAs and a one-way (condition: sad, neutral) repeated measures ANOVA were conducted to examine changes in sadness on the VAS pre- and post-mood induction. Analyses were conducted using difference scores (i.e., VAS_{DS}), residualized change scores (i.e., $Z_{RES}VAS$), and pre- and post-mood induction measures (i.e., VAS_{PRE} and VAS_{POST}). These analyses were used as a manipulation check for the mood induction procedures to ensure that a sad mood was induced. Significant differences between conditions was only expected post-mood induction. It was expected that formerly depressed and healthy control participants exposed to the sad mood induction would report significantly more dysphoric mood (> 10.00% change in mood state; Martin, 1990) after the mood induction compared to formerly depressed and healthy control participants exposed to the neutral mood induction.

In addition, a series of one-way (condition: sad, neutral) ANOVAs and a one-way (condition: sad, neutral) repeated measures ANOVA were conducted to assess changes in multiple emotions on the negative and positive affect general dimension and fear, guilt, hostility, and sadness basic negative emotional scales of the PANAS-X pre- and post-mood induction. Analyses were conducted using difference scores (i.e., PANAS-X N_{DS}, PANAS-X P_{DS}, PANAS-X F_{DS}, PANAS-X G_{DS}, PANAS-X H_{DS}, and PANAS-X S_{DS}), residualized change scores (i.e., Z_{RES}PANAS-X N, Z_{RES}PANAS-X P, Z_{RES}PANAS-X F, Z_{RES}PANAS-X G, Z_{RES}PANAS-X H, and Z_{RES}PANAS-X S), and pre- and post-mood induction measures (i.e., PANAS-X $N_{PRE/POST}$, PANAS-X $P_{PRE/POST}$, PANAS-X $F_{PRE/POST}$, PANAS-X $G_{PRE/POST}$, PANAS-X $H_{PRE/POST}$, and PANAS-X $S_{PRE/POST}$). These analyses were used as a manipulation check for the mood induction procedures to assess if emotions other than sadness were induced. Significant differences between conditions were only expected post-mood induction. It was expected that formerly depressed and healthy control participants exposed to the sad mood induction would report a significant increase in negative affect and a significant decrease in positive affect after the mood induction compared to formerly depressed and healthy control participants exposed to the neutral mood induction.

A series of 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) factorial ANOVAs and a 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) repeated measures ANOVA were conducted to examine changes in dysphoric mood on the VAS pre- and post-mood induction. Analyses were conducted using difference scores (i.e., VAS_{DS}), residualized change scores (i.e., Z_{RES}VAS), and pre- and post-mood induction measures (i.e., VAS_{PRE} and VAS_{POST}). These analyses were used to assess significant differences in mood reactivity based on depressive history and mood induction procedure. Planned comparisons were

conducted using contrast analyses. It was expected that formerly depressed and healthy control participants exposed to the sad mood induction would report significantly more dysphoric mood compared to formerly depressed and healthy control participants exposed to the neutral mood induction.

Cardiovascular measures. A series of one-way ANOVAs were used to assess significant group differences in cardiovascular functioning at baseline. Analyses were conducted using an average of the last two and five minutes of HP, RSA, CO, and PEP during baseline (e.g., $HP_{BL2/BL5}$, $RSA_{BL2/BL5}$, $CO_{BL2/BL5}$, and $PEP_{BL2/BL5}$). It was expected that formerly depressed and healthy control participants would exhibit similar cardiovascular functioning for HP, RSA, PEP, or CO during the baseline film.

A series of 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) factorial ANOVAs and a 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) repeated measures ANOVAs were used to assess significant group differences in cardiovascular reactivity during the mood induction. Analyses were conducted using difference scores (i.e., $HP_{DS2RA/DS5RA}$, $RSA_{DS2RA/DS5RA}$, $CO_{DS2RA/DS5RA}$, and $PEP_{DS2RA/DS5RA}$), residualized change scores (i.e., $Z_{RES2/RES5}HP_{RA}$, $Z_{RES2/RES5}RSA_{RA}$, $Z_{RES2/RES5}CO_{RA}$, and $Z_{RES2/RES5}PEP_{RA}$), and baseline and mood induction measures (i.e., $HP_{BL2/BL5/MI2/MI5}$, $RSA_{BL2/BL5/MI2/MI5}$, $CO_{BL2/BL5/MI2/MI5}$, and $PEP_{BL2/BL5/MI2/MI5}$). Planned comparisons were conducted using contrast analyses. It was expected that formerly depressed participants exposed to the sad mood induction would exhibit decreased HP, RSA, and CO and increased PEP during the mood induction compared to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

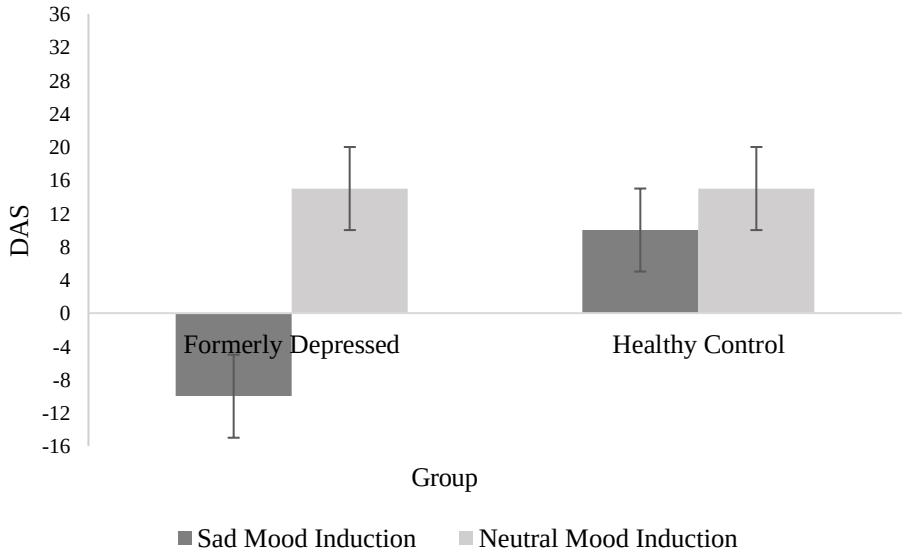
A series of 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) factorial ANOVAs and a 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) repeated measures ANOVAs were used to assess significant group differences in cardiovascular recovery after the mood induction. Analyses were conducted using difference scores (i.e., $HP_{DS2RC/DS5RC}$, $RSA_{DS2RC/DS5RC}$, $CO_{DS2RC/DS5RC}$, and $PEP_{DS2RC/DS5RC}$), residualized change scores (i.e., $Z_{RES2/RES5}HP_{RC}$, $Z_{RES2/RES5}RSA_{RC}$, $Z_{RES2/RES5}CO_{RC}$, and $Z_{RES2/RES5}PEP_{RC}$), and baseline and recovery measures (i.e., $HP_{BL2/BL5/RC2/RC5}$, $RSA_{BL2/BL5/RC2/RC5}$, $CO_{BL2/BL5/RC2/RC5}$, and $PEP_{BL2/BL5/RC2/RC5}$). Planned comparisons were conducted using contrast analyses. It was expected that formerly depressed participants exposed to the sad mood induction would exhibit reduced cardiovascular recovery during the recovery film compared to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood induction.

Hypothesis 1

Formerly depressed participants exposed to the sad mood induction would report significantly higher levels of cognitive reactivity on the DAS post-mood induction than formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions. Cognitive reactivity was operationalized as difference scores (i.e., DAS_{DS}) and residualized change scores (i.e., $Z_{RES}DAS$) for the DAS administered pre- and post-mood induction as well as pre- and post-mood induction measures (i.e., DAS-SF I and DAS II). A series of 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) factorial ANOVAs using difference scores and residualized change scores and a 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) repeated

measures ANOVA were used to assess the relationship between depression history, mood manipulation, and cognitive reactivity.

Figure 7. *Cognitive Reactivity Post-Mood Induction*



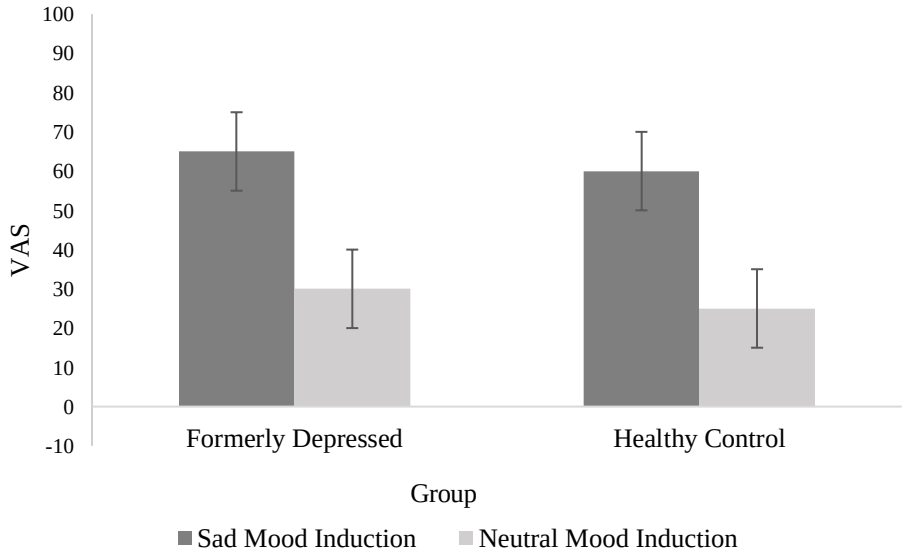
Note. Lower DAS scores indicate cognitive reactivity post-mood induction.

Hypothesis 2

Formerly depressed and healthy control participants exposed to the sad mood induction would report significantly higher levels of mood reactivity on the VAS post-mood induction than formerly depressed and healthy control participants exposed to the neutral mood induction. No significant differences in mood reactivity on the VAS post-mood induction were expected between formerly depressed and healthy control participants exposed to the sad mood induction. Mood reactivity was operationalized as difference scores (i.e., VAS_{DS}) and residualized change scores (i.e., $Z_{RES}VAS$) for the VAS administered pre- and post-mood induction as well as pre- and post-mood induction measures (i.e., VAS_{PRE} and VAS_{POST}). A series of 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) factorial ANOVAs using difference scores and standardized change scores and a 2 (group: formerly depressed, healthy control) X 2

(condition: sad, neutral) repeated measures ANOVA were used to assess the relationship between depression history, mood manipulation, and mood reactivity.

Figure 8. *Mood Reactivity Post-Mood Induction*



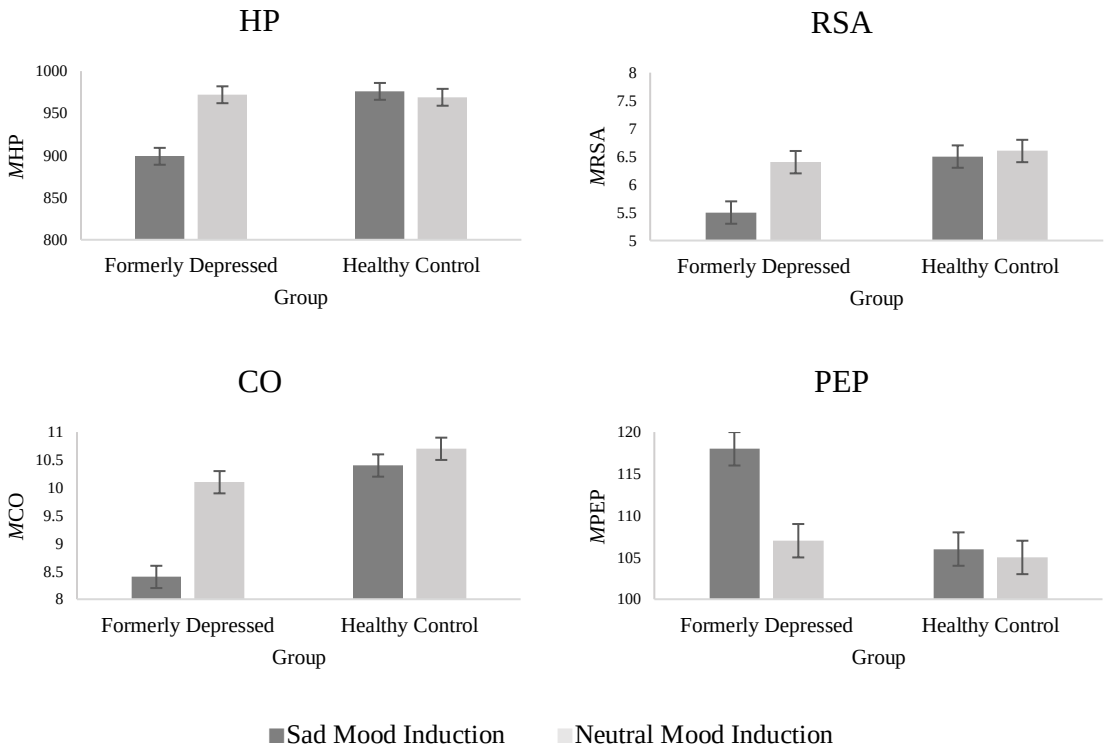
Note. Higher VAS scores indicate mood reactivity post-mood induction.

Hypothesis 3

Formerly depressed participants exposed to the sad mood induction would exhibit a maladaptive pattern of cardiovascular reactivity (i.e., decreased HP, RSA, and CO and increased PEP) during the mood induction compared to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions. An average of the first five minutes of the mood induction was computed for HP, RSA, PEP, and CO (i.e., $HP_{MI2/MI5}$, $RSA_{MI2/MI5}$, $CO_{MI2/MI5}$, and $PEP_{MI2/MI5}$) and serve as a mood induction measure. An average of the last five minutes of the mood induction was computed for HP, RSA, PEP, and CO (i.e., $HP_{BL2/BL5}$, $RSA_{BL2/BL5}$, $CO_{BL2/BL5}$, and $PEP_{BL2/BL5}$) and serve as a baseline measure. Cardiovascular reactivity was operationalized as the difference score for the average of each cardiovascular measure (i.e., $HP_{DS2RA/DS5RA}$, $RSA_{DS2RA/DS5RA}$, $CO_{DS2RA/DS5RA}$,

and $PEP_{DDS2RA/DS5RA}$) and residualized change scores for the average of each cardiovascular measure (i.e., $Z_{RES2/RES5}HP_{RA}$, $Z_{RES2/RES5}RSA_{RA}$, $Z_{RES2/RES5}CO_{RA}$, and $Z_{RES2/RES5}PEP_{RA}$) assessed during baseline and the mood induction (i.e., $HP_{BL2/BL5/MI2/MI5}$, $RSA_{BL2/BL5/MI2/MI5}$, $CO_{BL2/BL5/MI2/MI5}$, and $PEP_{BL2/BL5/MI2/MI5}$). A series of 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) factorial ANOVAs using difference scores and residualized change scores and a 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) repeated measures ANOVA were used to assess the relationship between history of depression, mood manipulation, and cardiovascular reactivity.

Figure 9. *Cardiovascular Reactivity during Mood Induction*

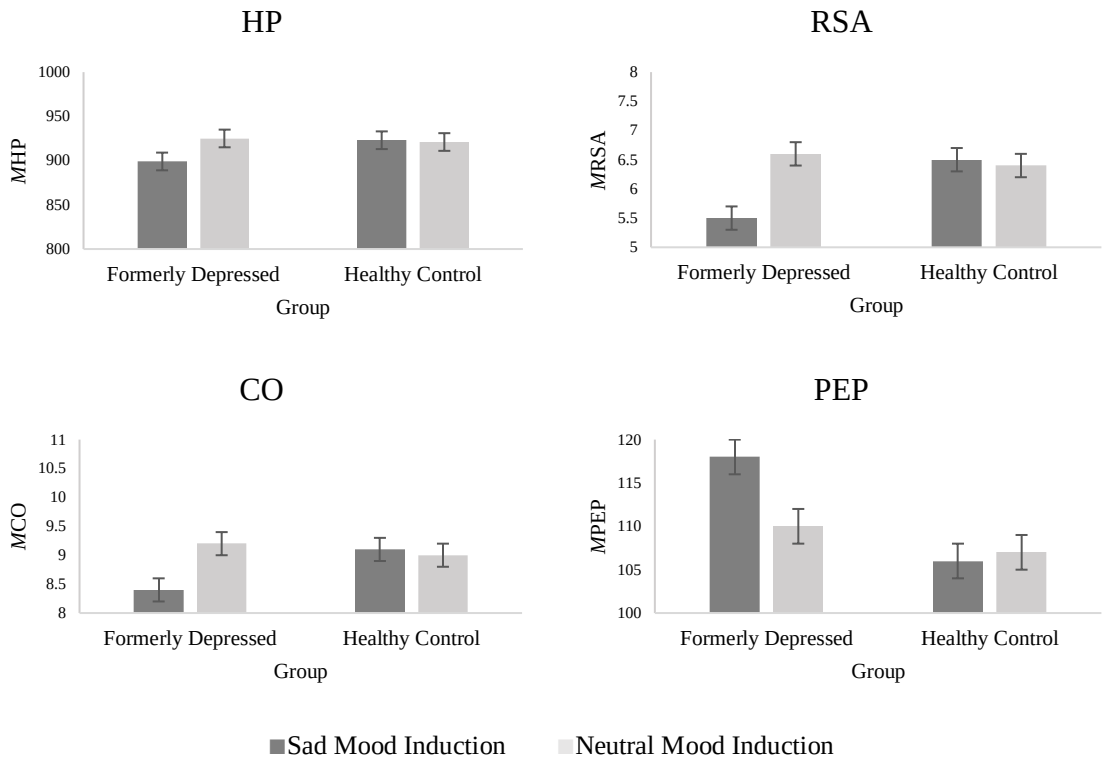


Note. Higher HP, RSA, and CO and lower PEP is considered to represent more adaptive cardiovascular reactivity in response to the mood induction procedures.

Hypothesis 4

Formerly depressed participants exposed to the sad mood induction would exhibit reduced cardiovascular recovery (i.e., decreased HP, RSA, and CO and increased PEP compared to baseline) during the recovery film compared to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions. An average of the last five minutes of recovery was computed for HP, RSA, PEP, and CO (e.g., $HP_{RC2/RC5}$, $RSA_{RC2/RC5}$, $CO_{RC2/RC5}$, and $PEP_{RC2/RC5}$) and serve as a recovery measure. An average of the last five minutes of the mood induction was computed for HP, RSA, PEP, and CO (i.e., $HP_{BL2/BL5}$, $RSA_{BL2/BL5}$, $CO_{BL2/BL5}$, and $PEP_{BL2/BL5}$) and serve as a baseline measure. Cardiovascular recovery was operationalized as the difference score for the average of each cardiovascular measure (i.e., $HP_{DS2RC/DS5RC}$, $RSA_{DS2RC/DS5RC}$, $CO_{DS2RC/DS5RC}$, and $PEP_{DS2RC/DS5RC}$) and residualized change scores for the average of each cardiovascular measure (i.e., $Z_{RES2/RES5}HP_{RC}$, $Z_{RES2/RES5}RSA_{RC}$, $Z_{RES2/RES5}CO_{RC}$, and $Z_{RES2/RES5}PEP_{RC}$) during the baseline and the recovery period (i.e., $HP_{BL2/BL5/RC2/RC5}$, $RSA_{BL2/BL5/RC2/RC5}$, $CO_{BL2/BL5/RC2/RC5}$, and $PEP_{BL2/BL5/RC2/RC5}$). A series of 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) factorial ANOVAs using difference scores and residualized change scores and a 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) repeated measures ANOVA were used to assess the relationship between history of depression, mood manipulation, and cardiovascular recovery.

Figure 10. *Cardiovascular Recovery during Recovery Film*



Note. A return to baseline cardiovascular functioning is considered to represent more adaptive cardiovascular recovery during the recovery period.

Table 3. *Study Procedure and Hypotheses Chart*

Time	Task Category	Task Description	Purpose
Screening	Self-report measures	Contact and demographic information	Exclusion criteria
		GHS: language and visual abilities, current and past health conditions	
		BDI-II: current depressive symptoms	
		PHQ-9: past depressive symptoms	
		BAI: current anxiety symptoms	
Session 1	Self-report measures	Demographic information: age, sex, race, ethnicity, marital status, and education level	Potential covariates
		BDI-II: current depressive symptoms	
		STAI-I & II: current anxiety symptoms	
	Physical measure	BMI: calculated via height and weight	
	Interview	Treatment History: current and past CBT and antidepressant use	
		SCID-IV-RV: psychiatric diagnoses	Exclusion criteria
Session 2	Baseline	ECG and ICG recording while viewing 10-minute clip of Alaska Denali Park Video	Baseline cardiovascular functioning
	Self-report measures	DAS-SF I: dysfunctional beliefs at baseline	Baseline cognitions
		VAS: dysphoric mood at baseline	Baseline mood, manipulation check
		PANAS-X: dysphoric mood at baseline	
	Mood induction	ECG and ICG recording while listening to 7:38 minute piece of sad or neutral music and recalling sad or neutral autobiographical memory	Cardiovascular reactivity, H_3
	Self-report measures	DAS-SF II: dysfunctional beliefs post MI (i.e., cognitive reactivity to MI)	Post MI cognitions, H_1
		VAS: dysphoric mood post MI (i.e., mood reactivity to MI)	Post MI mood, manipulation check, H_2
		PANAS-X: dysphoric mood post MI	Post MI mood, manipulation check
	Recovery	ECG and ICG recording while viewing 10-minute clip of Alaska Wilderness Video	Cardiovascular recovery, H_4
	Debriefing	Provide and review debriefing form	None

Note. GHS = General Health Screen; BDI-II = Beck Depression Inventory – Second Edition; PHQ-9 = Patient Health Questionnaire – 9; BAI = Beck Anxiety Inventory; STAI-I & II = State-Trait Anxiety Inventory – I & II; CBT = cognitive-behavioral therapy; BMI = body mass index; SCID-IV-RV = Structured Clinical Interview for DSM-IV-TR – Research Version; ECG = electrocardiogram; ICG = impedance cardiography; H = hypothesis; DAS I = Dysfunctional Attitudes Scale – Short Form I; DAS II = Dysfunctional Attitudes Scale – Short Form II; VAS = Visual Analogue Scale; PANAS-X = Positive and Negative Affect Scale – Expanded Form.

CHAPTER 7

RESULTS

The purpose of this study was to characterize cognitive, mood, and cardiovascular reactivity to and recovery from a sad mood induction in individuals with a history of depression compared to healthy, never depressed individuals. A transient sad or neutral mood was experimentally induced using an empirically validated music and autobiographical recall mood induction. Self-report measures were used to test the study hypotheses regarding cognitive and mood reactivity to the transient mood (i.e., Hypotheses 1 and 2). Cardiovascular measures were used to test the study hypotheses regarding cardiovascular reactivity to and cardiovascular recovery from the transient mood (i.e., Hypotheses 3 and 4).

Session 1

Demographic Information

Participants were recruited from the University of Maine psychology undergraduate participant pool and the surrounding community. The distribution of participants by recruitment source and group is presented in Table 4. While the majority of the final sample was drawn from the undergraduate participant pool (n = 87, 65.90%), over a third of the sample was recruited from the surrounding community (n = 45, 34.10%).

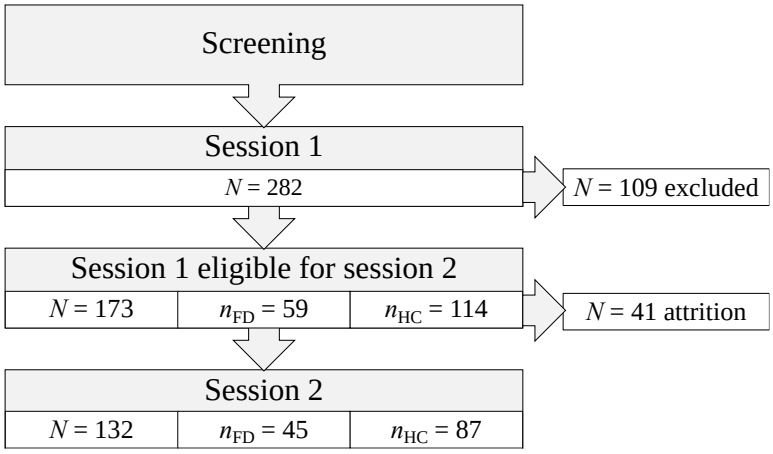
Table 4. *Recruitment Source by Group*

			Group			
	Total (N = 132)		FD (n = 45)		HC (n = 87)	
Recruitment source	*n*	%	*n*	%	*n*	%
Participant pool	87	65.90	21	46.70	66	75.90
Community	45	34.10	24	53.30	21	24.10

Note. FD = formerly depressed; HC = healthy control.

Participants who met the eligibility criteria during screening were invited to participate in session 1. 282 participants completed session 1. At session 1, 109 participants (38.65%) were excluded, leaving 173 participants (61.35%) who were eligible to participate in session 2. Of these participants, 59 participants met the inclusion criteria for the formerly depressed group and 114 participants met the inclusion criteria for the healthy control group. Between sessions 1 and 2, 41 participants (23.70%) were lost to follow-up. 132 participants (76.30%) completed session 2, with 45 participants in the formerly depressed group and 87 participants in the healthy control group. A flow chart of participant recruitment visually depicts this information (Figure 11).

Figure 11. *Flow Chart of Participant Recruitment*



Note. FD = formerly depressed; HC = healthy control.

Following session 1, participants in the two groups were randomly assigned to the two experimental conditions. The distribution of participants in the two experimental conditions by group is presented in Table 5. Formerly depressed and healthy control participants were assigned to the sad (n = 65) and neutral (n = 67) mood inductions roughly evenly, with slight differences in the size of groups due to between-session attrition from sessions 1 and 2.

Table 5. *Condition by Group*

			Group			
	Total (*N* = 132)		FD (*n* = 45)		HC (*n* = 87)	
Condition	*n*	%	*n*	%	*n*	%
SMI	65	49.20	21	46.70	44	50.60
NMI	67	50.80	24	53.30	43	49.40

Note. FD = formerly depressed; HC = healthy control; SMI = sad mood induction; NMI = neutral mood induction.

Descriptive statistics. Participants demographic information that was considered as potential covariates. Descriptive statistics for demographic information for the entire sample are presented in Table 6 while means, standard deviations, *p* values, and effect sizes for demographic information by group are presented in Tables 6 and 7. Participants in this study (*N* = 132) were predominantly younger (*M* = 20.79, *SD* = 5.65), female (*n* = 81, 61.40%), Caucasian (*n* = 115, 87.10%), never married (*n* = 121, 91.67%) college students (*n* = 114, 86.40%). These results are consistent with the demographic make-up of the University of Maine and surrounding community as Orono, Maine is a university town. Of note, the racial and ethnic make-up of the study is consistent with the location as the state of Maine currently has the highest proportion of Caucasian residents in the country at 94.70% (United States Census Bureau, 2017). A subset of the sample included older (range = 18-60), college educated (*n* = 16, 12.90%) adults. Participants' BMI was on average, in the overweight range (*M* = 25.16, *SD* = 5.65), which is consistent with recent estimates that 35.90% of Maine's population falls within the overweight range (Centers for Disease Control and Prevention, 2017). A low proportion of participants reported current or previous mental health treatment (psychotherapy = 2.00-8.00%, psychotropic medication = 7.00-12.00%).

Independent samples t-tests. Means, standard deviations, *p* values, and effect sizes for demographic information by group are presented in Table 7. Independent samples *t*-tests were

used to examine differences between groups for continuous variables (i.e., age and BMI). Levene's test for homogeneity of variance was significant for age ($F(129) = 26.96$, $p < .001$) but not BMI ($F(128) = .97$, $p = .33$), indicating that this assumption underlying *t*-test was met for BMI but not age. Consequently, results for equal variances not assumed are reported for age. Formerly depressed participants were significantly older ($t(49.01) = 2.27$, $p = .03$, CI = .34, 5.62, $d = .47$) than healthy control participants. There were no significant difference between groups for BMI ($t(128) = -.18$, $p = .86$, CI = -2.28, 1.90, $d = .03$).

Chi-square tests. Means, standard deviations, *p* values, and effect sizes for demographic information by group are presented in Table 7. Chi-square tests were used to examine differences between the two groups for categorical variables (i.e., sex, race, ethnicity, education level, marital status, and current and past CBT and antidepressant use). Unsurprisingly, formerly depressed participants reported significantly more past CBT use ($\chi^2(1) = 6.72$, $p = .04$, $w = .23$) and current ($\chi^2(1) = 16.54$, $p < .001$, $w = .35$) and past ($\chi^2(1) = 21.78$, $p < .001$, $w = .41$) antidepressant use than healthy control participants. There were no significant difference between groups for sex ($\chi^2(1) = 3.33$, $p = .07$, $w = .16$), race ($\chi^2(3) = 2.44$, $p = .49$, $w = .14$), Hispanic/Latino ($\chi^2(1) = 3.18$, $p = .08$, $w = .16$) or Franco-American ($\chi^2(1) = .06$, $p = .80$, $w = .02$) ethnicity, marital status ($\chi^2(2) = 5.18$, $p = .08$, $w = .20$), education level ($\chi^2(5) = 5.67$, $p = .34$, $w = .21$), and current CBT use ($\chi^2(1) = 4.82$, $p = .09$, $w = .19$). Treatment history data was not collected for 16 formerly depressed and 40 healthy control participants. Due to the large amount of missing data, current and past CBT and antidepressant use were removed from further analyses.

Table 6. *Descriptive Statistics for Demographic Information*

Measure	*M*	*SD*	Sample range
Age, years	20.79	5.65	18-60
BMI, kg/m^2	25.16	5.65	16.14-47.76

Note. BMI = body mass index.

Table 7. *Means, Standard Deviations, P Values, and Effect Sizes for Demographic Information by Group*

			Group					
	Total (*N* = 132)		FD (*n* = 45)		HC (*n* = 87)			
Variable	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	*d*
Age, years	20.79	5.65	22.77	8.43	19.79	3.11	.03	.47
BMI, kg/m^2	25.16	5.65	25.04	5.73	25.22	5.64	.86	.03
Variable	*n*	%	*n*	%	*n*	%	*p*	*w*
Sex							.07	.16
Male	50	37.90	12	26.70	38	43.70		
Female	81	61.40	32	71.10	49	56.30		
Race							.49	.14
Caucasian	115	87.10	41	91.10	74	85.10		
African American	4	3.00	0	N/A	4	4.60		
Asian American	4	3.00	1	2.20	3	3.40		
Multiple races	7	5.30	2	4.40	5	5.70		
Ethnicity								
Hispanic/Latino	6	4.50	0	N/A	6	6.90	.08	.16
Franco-American	10	7.60	3	6.70	7	8.00	.80	.02
Marital status							.08	.20
Married	8	6.10	4	8.90	4	4.60		
Never married	121	91.70	38	84.40	83	95.40		
Divorced	2	1.50	2	4.40	0	N/A		
Education level							.34	.21
High school	57	43.20	15	33.30	42	48.30		
1 year of college	30	22.70	9	20.00	21	24.10		
2+ years of college	27	20.50	12	26.70	15	17.20		
Associates degree	1	.80	0	N/A	1	1.10		
Bachelor's degree	9	6.80	5	11.10	4	4.60		
Doctoral degree	7	5.30	3	6.70	4	4.60		
Treatment history								
Past CBT	8	6.10	6	13.30	2	2.30	.04	.23
Current CBT	2	1.50	2	4.40	0	N/A	.09	.19
Past RX	12	9.10	11	24.40	1	1.10	< .001	.41
Current RX	7	5.30	7	15.60	0	N/A	< .001	.35

Note. FD = formerly depressed; HC = healthy control; BMI = body mass index; CBT = cognitive behavioral therapy; RX = antidepressant medication; One FD participant elected not to provide any demographic data and one HC participant elected not to provide race data. Treatment history data was not collected for 16 FD and 40 HC participants. Consequently, percentages for some category do not add up to 100.00%.

Self-Report Measures

During session 1, participants completed two self-report measures that were considered as potential covariates. The BDI-II (Beck et al., 1996a; Appendix C) was used to evaluate the severity of current depressive symptoms. Respondents rated the severity of depressive symptoms experienced over the past two weeks on a scale from 0 to 3, with 0 indicating the symptom is not present and 3 indicating that the symptom is present and severe. Total scores ranged from 0 to 63, with higher scores indicating greater levels of depressive symptom severity. The STAI-I & II (Spielberger et al., 1983; Appendix I) was used to assesses cognitive, affective, and somatic symptoms of both state and trait anxiety. Respondents rated the severity of anxiety symptoms experienced at the moment for the state version and in general for the trait version from 1 to 4, with 1 indicating the symptom is almost never present and 4 indicating that the symptom is almost always present. Total scores for each version ranged from 20 to 80, with higher scores indicating greater levels of state or trait anxiety symptom severity. Outlier data for the STAI-II (n = 1) was winsorized to address extreme values. No outlier data was present for the BDI-II or STAI-I.

Descriptive statistics. Descriptive statistics for session 1 self-report measures are presented in Table 8. The BDI-II demonstrated good internal consistency (α = .89) and the STAI-I demonstrated acceptable internal consistency (α = .71) in this study sample (Tavakol & Dennick, 2011). Cronbach's alpha indicated that the STAI-II was not a reliable measure of state anxiety symptoms (α = .40). Consequently, the measure was removed from further analyses (Tavakol & Dennick, 2011).

Table 8. *Descriptive Statistics for Session 1 Self-Report Measures*

Measure	*M*	*SD*	Sample range	Sample α
BDI-II	5.50	6.28	0-35	.89
STAI-I	45.35	5.67	32-64	.71
STAI-II	44.90	4.08	36-55	.40

Note. BDI-II = Beck Depression Inventory – Second Edition; STAI-I = State-Trait Anxiety Inventory – I; STAI-II = State-Trait Anxiety Inventory – II.

Independent samples t-tests. Means, standard deviations, *p* values, and effect sizes for session 1 self-report measures by group are presented in Table 9. Independent samples *t*-tests were used to examine differences between groups for current depressive (i.e., BDI-II) and state anxiety (i.e., STAI-I) symptoms. Levene's test for homogeneity of variance was significant for the BDI-II ($F(125) = 24.92$, $p < .001$) and STAI-I ($F(121) = 5.05$, $p = .03$), indicating that this assumption underlying *t*-test was not met. Consequently, results for equal variances not assumed are reported for the BDI-II and STAI-I. Formerly depressed participants reported significantly higher levels of current depressive symptoms on the BDI-II ($t(49.77) = 5.08$, $p < .001$, CI = 3.98, 9.19, $d = 1.06$) than healthy control participants. There were no significant difference between groups for current state anxiety symptoms on the STAI-I ($t(60.73) = -1.01$, $p = .32$, CI = -3.63, 1.19, $d = .20$).

Table 9. *Means, Standard Deviations, P Values, and Effect Sizes for Session 1 Self-Report Measures by Group*

			Group					
	Total (N = 127)		FD (n = 42)		HC (n = 85)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	*d*
BDI-II	5.50	6.28	9.91	7.99	3.32	3.68	< .001	1.06
	Total (N = 123)		FD (n = 40)		HC (n = 83)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	*d*
STAI-I	45.35	5.67	44.53	6.77	45.74	5.06	.32	.20

Note. FD = formerly depressed; HC = healthy control; BDI-II = Beck Depression Inventory – Second Edition; STAI-I = State-Trait Anxiety Inventory – I.

Session 2

Cognitive Measures

During session 2, participants completed two versions of one self-report measure that was used to assess cognitive reactivity to the mood induction (i.e., Hypothesis 1). The DAS-SF I & II (Beevers et al., 2007; Appendix L) was used to evaluate changes in dysfunctional beliefs about oneself before and after the mood induction. Respondents rated their beliefs experienced most of the time on a scale of 1 to 4, with 1 indicating that they totally agreed with the statement and 4 indicating that they totally disagreed with the statement. Total scores ranged from 9 to 36, with lower scores indicating greater levels of the dysfunctional beliefs. Cognitive reactivity was operationalized as difference scores (i.e., DAS_{DS}) and residualized change scores (i.e., $Z_{RES}DAS$) for the DAS administered pre- and post-mood induction as well as pre- and post-mood induction measures (i.e., DAS I and DAS II). No outlier data was present for the DAS-SF I, DAS-SF II, and DAS_{DS}.

Descriptive statistics. Descriptive statistics for session 2 cognitive measures are presented in Table 10. The DAS-SF I & II demonstrated acceptable internal consistency (α = .79-.80) in this study sample (Tavakol & Dennick, 2011). The difference (i.e., $_{DS}$) and residualized change (i.e., Z_{RES}/Z_{RES}) scores are calculated measures. Consequently, Cronbach's alpha could not be calculated.

Table 10. *Descriptive Statistics for Session 2 Cognitive Measures*

Measure	*M*	*SD*	Sample range	Sample α
DAS-SF I	27.67	3.87	19-36	.80
DAS-SF II	27.03	3.96	16-36	.79
DAS_{DS}	-.64	2.87	-8-8	
$Z_{RES}DAS$	.00	1.00	-2.51-2.95	

Note. DAS I = Dysfunctional Attitudes Scale – Short Form I; DAS II = Dysfunctional Attitudes Scale – Short Form II; $_{DS}$ = difference score; Z_{RES} = residualized change scores.

Hypothesis 1

A series of 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) factorial ANOVAs using difference scores and residualized change scores and a 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) repeated measures ANOVA were used to test the hypothesis that formerly depressed participants exposed to the sad mood induction would report significantly more dysfunctional beliefs on the DAS post-mood induction than formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions. Means, standard deviations, *p* values, and effect sizes for session 2 cognitive measures by group are presented in Table 11 while means, standard deviations, *p* values, and effect sizes for session 2 cognitive measures by group and condition are presented in Table 12. Planned comparisons were conducted using contrast analyses. Means, standard deviations, and *p* values for session 2 cognitive measures for planned comparisons are presented in Table 13.

DAS. Analyses conducted using the DAS are reviewed below.

2 X 2 factorial ANOVA – difference score. None of the covariates that were under consideration were significantly associated with cognitive reactivity (all *p*'s > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,122) = .43$, $p = .73$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,122) = 1.27$, $p = .26$, $\eta^2 = .01$) or condition ($F(1,122) = .23$, $p = .63$, $\eta^2 = .002$) nor the group by condition interaction ($F(1,122) = 1.81$, $p = .18$, $\eta^2 = .02$) were significant, indicating that there was no significant difference in cognitive reactivity on the DAS post-mood induction between groups or conditions or the group by condition interaction when using difference scores. Contrast analyses revealed that there was no significant difference in

cognitive reactivity on the DAS post-mood induction ($t(122) = -1.51$, $p_L = .23$, $p_Q = .18$, $p_C = .94$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 factorial ANOVA – residualized change score. None of the covariates that were under consideration were significantly associated with cognitive reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,122) = .68$, $p = .57$), indicating that this assumption underlying ANOVA was met. Formerly depressed participants reported significantly higher levels of dysfunctional beliefs on the DAS post-mood induction ($F(1,122) = 5.11$, $p = .03$, $\eta^2 = .04$) than healthy control participants. Neither the main effect of condition ($F(1,122) = .14$, $p = .71$, $\eta^2 = .001$) nor the group by condition interaction ($F(1,122) = 1.02$, $p = .31$, $\eta^2 = .008$) were significant, indicating that there was no significant difference in cognitive reactivity on the DAS post-mood induction between mood induction conditions or the group by condition interaction when using residualized change scores. Using contrast analyses, formerly depressed participants exposed to the sad mood induction reported significantly higher levels of dysfunctional beliefs on the DAS post-mood induction ($t(122) = -1.86$, $p_L = .03$, $p_Q = .31$, $p_C = .49$) compared to healthy control participants exposed to the sad mood induction and formerly depressed and healthy control participants exposed to the neutral mood induction.

2 X 2 repeated measures ANOVA. Current depressive ($p < .001$) and state anxiety ($p = .03$) symptoms were significantly associated with cognitive reactivity and were included in the final model. The remaining covariates that were under consideration were not significantly associated with cognitive reactivity (all *p's* > .05) and were dropped from the final model.

Levene's test for homogeneity of variance was not significant (DAS-SF I: $F(3,111) = .14$, $p = .93$; DAS-SF II: $F(3,111) = .04$, $p = .99$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,109) = .67$, $p = .42$, $\eta^2 = .006$) or condition ($F(1,109) = .03$, $p = .87$, $\eta^2 < .001$) nor the group by condition interaction ($F(1,109) = .79$, $p = .38$, $\eta^2 = .007$) were significant, indicating that there was no significant difference in cognitive reactivity on the DAS post-mood induction between groups or conditions or the group by condition interaction when using repeated measures. Contrast analyses revealed that there was no significant difference in cognitive reactivity on the DAS post-mood induction ($F(1,111) = .01$, $p = .92$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

Post-hoc power analyses. Post-hoc power analyses were conducted using G*Power 3.1.9.2 (Faul et al., 2007) to determine the power achieved by the DAS analyses. Results indicated that the current model had a power of .36 for the difference score, .17 for the residualized change score, and .13 for the repeated measures. Results suggested that given the current study's sample size, α level, and observed effect size, there was a 13 to 36% chance of detecting an effect depending on which analytic technique was used.

Sensitivity analyses. Sensitivity analyses were conducted using G*Power 3.1.9.2 (Faul et al., 2007) to determine the minimum effect size needed to obtain significant results for the DAS analyses. Results indicated that the required effect size was $f = .25$ for the factorial ANOVAs and $f = .28$ for the repeated measures ANOVA. Results suggested that given the current study's sample size and α level, at least a medium effect size was required to obtain significant results if a power of .80 was achieved.

Table 11. *Means, Standard Deviations, P Values, and Effect Sizes for Session 2 Cognitive Measures by Group*

			Group					
	Total (N = 126)		FD (n = 43)		HC (n = 83)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
DAS-SF I	27.67	3.87	26.23	3.87	28.40	3.68		
DAS-SF II	27.03	3.96	25.27	3.59	27.95	3.84		
DAS_{DS}	-.64	2.87	-1.02	2.94	-.45	2.83	.26	.01
$Z_{RES}DAS$	.00	1.00	-.27	.97	.14	.99	.03	.04
DAS_{RM}							.42	.006

Note. FD = formerly depressed; HC = healthy control; DAS I = Dysfunctional Attitudes Scale – Short Form I; DAS II = Dysfunctional Attitudes Scale – Short Form II; $_{DS}$ = difference score; Z_{RES} = residualized change scores; $_{RM}$ = repeated measures.

Table 12. *Means, Standard Deviations, P Values, and Effect Sizes for Session 2 Cognitive Measures by Group and Condition*

			Group and Condition									
	Total (N = 126)		FD/SMI (n = 20)		FD/NMI (n = 23)		HC/SMI (n = 42)		HC/NMI (n = 41)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
DAS-SF I	27.67	3.87	26.85	4.02	25.70	3.75	28.00	3.50	28.79	3.85		
DAS-SF II	27.03	3.96	25.30	3.50	25.25	3.75	27.95	3.81	27.95	3.92		
DAS_{DS}	-.64	2.87	-1.55	2.54	-.57	3.23	-.21	2.78	-.68	2.89	.18	.02
$Z_{RES}DAS$	.00	1.00	-.41	.82	-.15	1.10	.20	.98	.08	1.00	.31	.008
DAS_{RM}											.38	.007

Note. The difference between pre- and post-mood induction measures may not precisely equal the difference score as outlier data was addressed differently depending on the type of analyses conducted; FD = formerly depressed; HC = healthy control; SMI = sad mood induction; NMI = neutral mood induction; DAS I = Dysfunctional Attitudes Scale – Short Form I; DAS II = Dysfunctional Attitudes Scale – Short Form II; $_{DS}$ = difference score; Z_{RES} = residualized change scores; $_{RM}$ = repeated measures.

Table 13. *Means, Standard Deviations, and P Values for Session 2 Cognitive Measures for Planned Comparisons*

			Group and Condition						
	Total (N = 126)		FD/SMI (n = 20)		FD/NMI, HC/SMI, HC/NMI (n = 106)				
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	p_L	p_Q	p_C
DAS-SF I	27.67	3.87	26.85	4.02	27.83	3.84			
DAS-SF II	27.03	3.96	25.30	3.50	27.35	3.97			
DAS_{DS}	-.64	2.87	-1.55	2.54	-.47	2.90	.23	.18	.94
$Z_{RES}DAS$	.00	1.00	-.41	.82	.08	1.01	.03	.31	.49
DAS_{RM}							.92		

Note. FD = formerly depressed; HC = healthy control; SMI = sad mood induction; NMI = neutral mood induction; $_L$ = linear; $_Q$ = quadratic; $_C$ = cubic; DAS I = Dysfunctional Attitudes Scale – Short Form I; DAS II = Dysfunctional Attitudes Scale – Short Form II; $_{DS}$ = difference score; Z_{RES} = residualized change scores; $_{RM}$ = repeated measures.

Mood Measures

During session 2, participants completed one self-report measure twice that was used to assess mood reactivity to the mood induction (i.e., Hypothesis 2) and served as a manipulation check for the mood induction procedures to assess if feelings of sadness were induced. The VAS (Appendix L) was used to evaluate subjective changes in mood before and after the mood induction. Respondents rated their current level of sadness on a scale of 0 to 100, with 0 indicating lower levels of sadness and 100 indicating higher levels of sadness. Total scores ranged from 0 to 100, with higher scores indicating greater levels of sadness. Mood reactivity was operationalized as difference scores (i.e., VAS_{DS}) and residualized change scores (i.e., $Z_{RES}VAS$) for the VAS administered pre- and post-mood induction as well as pre- and post-mood induction measures (i.e., VAS_{PRE} and VAS_{POST}).

In addition, participants completed one self-report measure twice that was used as a manipulation check for the mood induction procedures to assess if emotions other than sadness were induced. The PANAS-X (Watson & Clark, 1994; Appendix L) was used to evaluate changes in mood before and after the mood induction. Respondents rated the extent to which they were experiencing 60 affective adjectives on a Likert scale from 1 to 5, with 1 indicating that were not or very slightly experiencing the affective state and 3 indicating that they were experiencing the affective state extremely. Total scores for the general dimension scale (i.e., negative and positive affect) were calculated by summing the 10 affective adjectives that comprise each scale and ranged from 0 to 10, with higher scores indicating greater levels of the respective affect. Total scores for specific affective state scales (i.e., fear, guilt, hostility, and sadness) were calculated by summing the three to eight affective adjectives that comprise each

scale and ranged from 0 to 4, 5, 6, or 8, with higher scores indicating greater levels of the respective affect.

Outlier data for the VAS_{DS} (n = 7), PANAS-X N_{DS} (n = 1), PANAS-X P_{DS} (n = 4), PANAS-X F_{DS} (n = 1), PANAS-X G_{DS} (n = 24), PANAS-X H_{DS} (n = 6), and PANAS-X S_{DS} (n = 3) were winsorized to address extreme values. Outlier data for the VAS_{PRE} (n = 3), PANAS-X N_{PRE} (n = 4), PANAS-X N_{POST} (n = 2), PANAS-X F_{PRE} (n = 4), PANAS-X F_{POST} (n = 2), PANAS-X G_{PRE} (n = 6), PANAS-X G_{POST} (n = 2), PANAS-X H_{PRE} (n = 2), PANAS-X H_{POST} (n = 2), PANAS-X S_{PRE} (n = 1) were winsorized to address extreme values. In addition, outlier data for the Z_{RES}VAS (n = 5), Z_{RES}PANAS-X N (n = 2), Z_{RES}PANAS-X P (n = 4), Z_{RES}PANAS-X F (n = 1), Z_{RES}PANAS-X G (n = 27), Z_{RES}PANAS-X H (n = 1), and Z_{RES}PANAS-X P (n = 4) were winsorized to address extreme values. No outlier data was present for the VAS_{POST}, PANAS-X P_{PRE}, PANAS-X P_{POST}, or PANAS-X S_{POST}.

Descriptive statistics. Descriptive statistics for session 2 mood measures are presented in Table 14. The pre- (i.e., $_{PRE}$) and post- (i.e., $_{POST}$) mood induction scores are single item measures. Consequently, Cronbach's alpha could not be calculated. The difference (i.e., $_{DS}$) and residualized change (i.e., Z_{RES}) scores are calculated measures. Consequently, Cronbach's alpha could not be calculated. All of the general dimension and basic negative emotion scales of the PANAS-X demonstrated good (α = .84-.90) or excellent (α = .91-.93) internal consistency in this study sample.

Table 14. *Descriptive Statistics for Session 2 Mood Measures*

Measure	*M*	*SD*	Sample range	Sample α
VAS_{PRE}	8.67	13.29	0-63	
VAS_{POST}	24.99	26.22	0-93	
VAS_{DS}	15.61	24.33	-17-93	
Z_{RES}VAS	-.14	.89	-1.30-3.05	
PANAS-X N_{PRE}	14.80	5.62	10-41	.90
PANAS-X N_{POST}	15.35	5.58	10-42	.89
PANAS-X N_{DS}	.47	2.33	-6-9	
Z_{RES}PANAS-X N	-.05	.75	-1.95-4.33	
PANAS-X P_{PRE}	29.82	6.42	12-43	.85
PANAS-X P_{POST}	27.66	7.55	10-46	.90
PANAS-X P_{DS}	-2.14	3.36	-14-7	
Z_{RES}PANAS-X P	.14	.91	-4.18-4.76	
PANAS-X F_{PRE}	8.96	3.81	6-26	.89
PANAS-X F_{POST}	8.94	3.79	6-22	.90
PANAS-X F_{DS}	-.06	1.58	-5-6	
Z_{RES}PANAS-X F	.02	.85	-2.81-3.83	
PANAS-X G_{PRE}	7.91	3.32	6-23	.91
PANAS-X G_{POST}	8.27	3.62	6-25	.93
PANAS-X G_{DS}	.24	1.36	-3-8	
Z_{RES}PANAS-X G	-.15	.36	-1.98-4.70	
PANAS-X H_{PRE}	8.46	3.19	6-27	.86
PANAS-X H_{POST}	8.66	3.27	6-30	.84
PANAS-X H_{DS}	.14	1.79	-4-8	
Z_{RES}PANAS-X H	-.12	.73	-1.59-6.98	
PANAS-X S_{PRE}	7.80	3.39	5-25	.89
PANAS-X S_{POST}	8.06	3.51	5-25	.90
PANAS-X S_{DS}	.20	1.83	-4-9	
Z_{RES}PANAS-X S	-10	.80	-1.76-4.87	

Note. The difference between pre- and post-mood induction measures may not precisely equal the difference score as outlier data was addressed differently depending on the type of analyses conducted; VAS = Visual Analogue Scale; $_{PRE}$ = pre-mood induction; $_{POST}$ = post-mood induction; $_{DS}$ = difference score; Z_{RES} = residualized change scores; PANAS-X = Positive and Negative Affect Scale – Expanded Form; N = negative affect general dimension scale; P = positive affect general dimension scale; F = fear basic negative emotional scale; H = hostility basic negative emotional scale; G = guilt basic negative emotional scale; S = sadness basic negative emotional scale.

Manipulation Check

A series of one-way (condition: sad, neutral) ANOVAs using difference scores and residualized change scores and a one-way (condition: sad, neutral) repeated measures ANOVA were used as a manipulation check for the mood induction procedures to ensure that a sad mood was induced. The manipulation was conducted using both the VAS and the PANAS-X negative and positive affect general dimension and fear, guilt, hostility, and sadness basic negative emotional scales.

Descriptive statistics. Descriptive statistics for session 2 measures used for the manipulation check are included in Table 14. Means, standard deviations, *p* values, and effect sizes for the VAS and PANAS-X negative and positive affect general dimension and fear, guilt, hostility, and sadness basic negative emotional scales by condition are presented in Table 15.

VAS. Analyses conducted using the VAS are reviewed below.

One-way ANOVA – difference score. Sex ($p = .02$) was significantly associated with the VAS and was included in the final model. The remaining covariates that were under consideration were not significantly associated with the VAS (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was significant ($F(1,120) = 47.46$, $p < .001$), indicating that this assumption underlying ANOVA was not met. Of note, this did not appear to be driven by the inclusion of the covariate. Without the covariate, Levene's test for homogeneity of variance was significant ($F(1,121) = 55.62$, $p < .001$).

In line with the planned analyses, multiple steps were explored in an attempt to remedy the violation of this assumption. First, residuals of differences scores were calculated and Kolmogorov-Smirnov's and Shapiro-Wilk's tests of normality were conducted. Residuals of difference scores were normally distributed for the formerly depressed participants exposed to

the sad mood induction condition (Kolmogorov-Smirnov: $D(17) = .15$, $p = .20$; Shapiro-Wilk: $D(17) = .95$, $p = .38$). However, residuals of difference scores were not normally distributed for the remaining groups and conditions (all *p's* < .05). Second, difference scores were transformed using the reciprocal transformation. Levene's test for homogeneity of variance continued to be significant ($F(1,121) = 23.84$, $p < .001$), indicating that this assumption underlying ANOVA was still not met. Consequently, the one-way ANOVA was conducted with the α level decreased from .05 to .01. Participants exposed to the sad mood induction reported significantly higher levels of sadness on the VAS post-mood induction ($F(1,119) = 44.68$, $p < .001$, $\eta^2 = .27$) than participants exposed to the neutral mood induction when using difference scores.

One-way ANOVA – residualized change score. Sex ($p = .02$) was significantly associated with the VAS and was included in the final model. The remaining covariates that were under consideration were not significantly associated with the VAS (all *p*'s > .05) and were dropped from the final model. Levene's test for homogeneity of variance was significant ($F(1,120) = 50.61$, $p < .001$), indicating that this assumption underlying ANOVA was not met. Of note, this did not appear to be driven by the inclusion of the covariate. Without the covariate, Levene's test for homogeneity of variance was significant ($F(1,121) = 49.74$, $p < .001$).

In line with the planned analyses, multiple steps were explored in an attempt to remedy the violation of this assumption. First, residuals of residualized change scores were calculated and Kolmogorov-Smirnov's and Shapiro-Wilk's tests of normality were conducted. Residuals of residualized change scores were normally distributed for the formerly depressed participants exposed to the sad mood induction (Kolmogorov-Smirnov: $D(17) = .15$, $p = .20$; Shapiro-Wilk: $D(17) = .95$, $p = .38$). However, residuals of residualized change scores were not normally distributed for the remaining groups and conditions (all *p's* < .05). Second, residualized change

scores were transformed using the reciprocal transformation. Levene's test for homogeneity of variance continued to be significant ($F(1,121) = 24.68$, $p < .001$), indicating that this assumption underlying ANOVA was still not met. Consequently, the one-way ANOVA was conducted with the α level decreased from .05 to .01. Participants exposed to the sad mood induction reported significantly higher levels of sadness on the VAS post-mood induction ($F(1,119) = 48.36$, $p < .001$, $\eta^2 = .29$) than participants exposed to the neutral mood induction when using residualized change scores.

One-way repeated measures ANOVA. Sex ($p = .04$) and current depressive symptoms ($p = .008$) were significantly associated with the VAS and were included in the final model. The remaining covariates that were under consideration were not significantly associated with the VAS (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant for the pre-mood induction measure (VAS_{PRE}: $F(1,116) = 2.92$, $p = .09$) but was significant for the post-mood induction measure (VAS_{POST}: $F(1,116) = 47.84$, $p < .001$), indicating that this assumption underlying ANOVA was not met. Of note, this did not appear to be driven by the inclusion of the covariates. Without the covariates, Levene's test for homogeneity of variance was not significant for the pre-mood induction measure (VAS_{PRE}: $F(1,121) = 1.54$, $p = .22$) but was significant for the post-mood induction measure (VAS_{POST}: $F(1,121) = 38.95$, $p < .001$).

In line with the planned analyses, multiple steps were explored in an attempt to remedy the violation of this assumption. Residuals could not be calculated as there was only one measure for each calculation. The pre- and post-mood induction measures were transformed using the reciprocal transformation. Levene's test for homogeneity of variance was not significant for the pre-mood induction measure (VAS_{PRE}: $F(1,121) = .005$, $p = .95$) but continued to be significant

for the post-mood induction measure (VAS_{POST}: $F(1,121) = 4.34$, $p = .04$), indicating that this assumption underlying ANOVA was still not met. Consequently, the one-way ANOVA was conducted with the α level decreased from .05 to .01. Participants exposed to the sad mood induction reported significantly higher levels of sadness on the VAS post-mood induction ($F(1,114) = 25.80$, $p < .001$, $\eta^2 = .19$) than participants exposed to the neutral mood induction when using repeated measures.

PANAS-X N. Analyses conducted using the PANAS-X negative affect general dimension scale are reviewed below.

One-way ANOVA – difference score. None of the covariates that were under consideration were significantly associated with negative affect on the PANAS-X (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(1,118) = 3.07$, $p = .08$), indicating that this assumption underlying ANOVA was met. The main effect of condition ($F(1,118) = 2.37$, $p = .13$, $\eta^2 = .02$) was not significant, indicating that there was no significant difference in reporting of negative affect on the PANAS-X between mood induction conditions when using difference scores.

One-way ANOVA – residualized change score. Current depressive symptoms ($p = .002$) were significantly associated with negative affect on the PANAS-X and were included in the final model. The remaining covariates that were under consideration were not significantly associated with negative affect on the PANAS-X (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was significant ($F(1,113) = 8.04$, $p = .005$), indicating that this assumption underlying ANOVA was not met. Of note, this did not appear to be driven by the inclusion of the covariate. Without the covariate, Levene's test for homogeneity of variance was significant ($F(1,118) = 6.44$, $p = .01$).

In line with the planned analyses, multiple steps were explored in an attempt to remedy the violation of this assumption. First, residuals of differences scores were calculated and Kolmogorov-Smirnov's and Shapiro-Wilk's tests of normality were conducted. Residuals of difference scores were normally distributed for the formerly depressed participants exposed to the sad (Kolmogorov-Smirnov: $D(23) = .15$, $p = .20$; Shapiro-Wilk: $D(23) = .93$, $p = .11$) and neutral (Kolmogorov-Smirnov: $D(17) = .20$, $p = .07$; Shapiro-Wilk: $D(17) = .91$, $p = .09$) mood inductions. However, residuals of difference scores were not normally distributed for the remaining groups and conditions (p's $< .05$). Second, difference scores were transformed using the reciprocal transformation. Levene's test for homogeneity of variance continued to be significant ($F(1,118) = 10.35$, $p = .002$), indicating that this assumption underlying ANOVA was still not met. Consequently, the one-way ANOVA was conducted with the α level decreased from .05 to .01. With the adjusted p value, the main effect of condition ($F(1,112) = 5.35$, $p = .02$, $\eta^2 = .05$) was not significant, indicating that there was no significant difference in reporting of negative affect on the PANAS-X between mood induction conditions when using residualized change scores.

One-way repeated measures ANOVA. Current depressive symptoms ($p < .001$) were significantly associated with negative affect on the PANAS-X and were included in the final model. The remaining covariates that were under consideration were not significantly associated with negative affect on the PANAS-X (all p's $> .05$) and were dropped from the final model. Levene's test for homogeneity of variance was not significant (PANAS-X N_{PRE}: $F(1,113) = .52$, $p = .47$; PANAS-X N_{POST}: $F(1,113) = .06$, $p = .80$), indicating that this assumption underlying ANOVA was met. The main effect of condition ($F(1,112) = .55$, $p = .46$, $\eta^2 = .005$) was not

significant, indicating that there was no significant difference in reporting of negative affect on the PANAS-X between mood induction conditions when using repeated measures.

PANAS-X P. Analyses conducted using the PANAS-X positive affect general dimension scale are reviewed below.

One-way ANOVA – difference score. BMI (p = .04) and current depressive symptoms (p = .004) were significantly associated with positive affect on the PANAS-X and were included in the final model. The remaining covariates that were under consideration were not significantly associated with positive affect on the PANAS-X (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(1,116) = 3.26$, p = .07), indicating that this assumption underlying ANOVA was met. The main effect of condition ($F(1,114) = 3.59$, p = .06, η^2 = .03) was not significant, indicating that there was no significant difference in reporting of positive affect on the PANAS-X between mood induction conditions when using difference scores.

One-way ANOVA – residualized change score. BMI (p = .04) and current depressive symptoms (p = .0014) were significantly associated with positive affect on the PANAS-X and were included in the final model. The remaining covariates that were under consideration were not significantly associated with positive affect on the PANAS-X (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was significant ($F(1,116) = 3.89$, p = .05), indicating that this assumption underlying ANOVA was not met. Of note, this appeared to be driven by the inclusion of the covariates. Without the covariates, Levene's test for homogeneity of variance was not significant ($F(1,121) = 2.65$, p = .11). Consequently, calculating the residual of the residualized change scores to assess normality and transforming the residualized change scores to re-assess homogeneity of variance would not have

the intended effect. Instead, the one-way ANOVA was conducted with the α level decreased from .05 to .01. The main effect of condition ($F(1,114) = 3.77$, $p = .06$, $\eta^2 = .03$) was not significant, indicating that there was no significant difference in reporting of positive affect on the PANAS-X between mood induction conditions when using residualized change scores.

One-way repeated measures ANOVA. Current state anxiety symptoms ($p < .001$) were significantly associated with positive affect on the PANAS-X and were included in the final model. The remaining covariates that were under consideration were not significantly associated with positive affect on the PANAS-X (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant (PANAS-X P_{PRE}: $F(1,113) = .44$, $p = .51$; PANAS-X P_{POST}: $F(1,113) = 1.85$, $p = .18$), indicating that this assumption underlying ANOVA was met. The main effect of condition ($F(1,112) = .78$, $p = .38$, $\eta^2 = .007$) was not significant, indicating that there was no significant difference in reporting of positive affect on the PANAS-X between mood induction conditions when using repeated measures.

PANAS-X F. Analyses conducted using the PANAS-X fear negative basic emotional scale are reviewed below.

One-way ANOVA – difference score. None of the covariates that were under consideration were significantly associated with fear on the PANAS-X (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(1,121) = .10$, $p = .76$), indicating that this assumption underlying ANOVA was met. The main effect of condition ($F(1,121) = .003$, $p = .96$, $\eta^2 < .001$) was not significant, indicating that there was no significant difference in reporting of fear on the PANAS-X between mood induction conditions when using difference scores.

One-way ANOVA – residualized change score. None of the covariates that were under consideration were significantly associated with fear on the PANAS-X (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(1,121) = 1.08$, $p = .30$), indicating that this assumption underlying ANOVA was met. The main effect of condition ($F(1,121) = .02$, $p = .89$, $\eta^2 < .001$) was not significant, indicating that there was no significant difference in reporting of fear on the PANAS-X between mood induction conditions when using residualized change scores.

One-way repeated measures ANOVA. Current depressive symptoms ($p < .001$) were significantly associated with fear on the PANAS-X and were included in the final model. The remaining covariates that were under consideration were not significantly associated with fear on the PANAS-X (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant (PANAS-X F_{PRE}: $F(1,116) = 1.70$, $p = .20$; PANAS-X F_{POST}: $F(1,116) = .12$, $p = .73$), indicating that this assumption underlying ANOVA was met. The main effect of condition ($F(1,115) = .08$, $p = .78$, $\eta^2 = .001$) was not significant, indicating that there was no significant difference in reporting of fear on the PANAS-X between mood induction conditions when using repeated measures.

PANAS-X G. Analyses conducted using the PANAS-X guilt negative basic emotional scale are reviewed below.

One-way ANOVA – difference score. Current depressive symptoms ($p < .001$) were significantly associated with guilt on the PANAS-X and were included in the final model. The remaining covariates that were under consideration were not significantly associated with guilt on the PANAS-X (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(1,116) = .66$, $p = .42$), indicating that this

assumption underlying ANOVA was met. Participants exposed to the sad mood induction reported significantly higher levels of guilt on the PANAS-X post-mood induction ($F(1,115) = 9.72$, $p = .002$, $\eta^2 = .08$) than participants exposed to the neutral mood induction when using difference scores.

One-way ANOVA – residualized change score. Current depressive symptoms ($p < .001$) were significantly associated with guilt on the PANAS-X and were included in the final model. The remaining covariates that were under consideration were not significantly associated with guilt on the PANAS-X (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(1,116) = 1.23$, $p = .27$), indicating that this assumption underlying ANOVA was met. Participants exposed to the sad mood induction reported significantly higher levels of guilt on the PANAS-X post-mood induction ($F(1,115) = 8.69$, $p = .004$, $\eta^2 = .07$) than participants exposed to the neutral mood induction when using residualized change scores.

One-way repeated measures ANOVA. Current depressive symptoms ($p < .001$) were significantly associated with guilt on the PANAS-X and were included in the final model. The remaining covariates that were under consideration were not significantly associated with guilt on the PANAS-X (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant (PANAS-X G_{PRE}: $F(1,116) = 1.32$, $p = .25$; PANAS-X G_{POST}: $F(1,116) = .02$, $p = .88$), indicating that this assumption underlying ANOVA was met. The main effect of condition ($F(1,115) = .02$, $p = .90$, $\eta^2 < .001$) was not significant, indicating that there was no significant difference in reporting of guilt on the PANAS-X between mood induction conditions when using repeated measures.

PANAS-X H. Analyses conducted using the PANAS-X hostility negative basic emotional scale are reviewed below.

One-way ANOVA – difference score. Current state anxiety symptoms (p = .01) were significantly associated with hostility on the PANAS-X and were included in the final model. The remaining covariates that were under consideration were not significantly associated with hostility on the PANAS-X (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was significant (F(1,114) = 5.92, p = .02), indicating that this assumption underlying ANOVA was not met. Of note, this did not appear to be driven by the inclusion of the covariate. Without the covariate, Levene's test for homogeneity of variance was significant (F(1,123) = 5.48, p = .02).

In line with the planned analyses, multiple steps were explored in an attempt to remedy the violation of this assumption. First, residuals of differences scores were calculated and Kolmogorov-Smirnov's and Shapiro-Wilk's tests of normality were conducted. Residuals of difference scores were normally distributed for one measure of normality for the formerly depressed participants exposed to the neutral mood induction (Shapiro-Wilk: D(23) = .93, p = .12). However, residuals of difference scores were not normally distributed for the remaining groups and conditions (p's < .05). Second, difference scores were transformed using the reciprocal transformation. Levene's test for homogeneity of variance continued to be significant (F(1,122) = 5.31, p = .02), indicating that this assumption underlying ANOVA was still not met. Consequently, the one-way ANOVA was conducted with the α level decreased from .05 to .01. The main effect of condition (F(1,113) = 2.99, p = .09, η^2 = .03) was not significant, indicating that there was no significant difference in reporting of hostility on the PANAS-X between mood induction conditions when using difference scores.

One-way ANOVA – residualized change score. Current depression (p = .004) and state anxiety (p = .003) symptoms were significantly associated with hostility on the PANAS-X and were included in the final model. The remaining covariates that were under consideration were not significantly associated with hostility on the PANAS-X (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was significant (F(1,111) = 3.85, p = .05), indicating that this assumption underlying ANOVA was not met. Of note, this did not appear to be driven by the inclusion of the covariates. Without the covariates, Levene's test for homogeneity of variance was significant (F(1,123) = 3.80, p = .05).

In line with the planned analyses, multiple steps were explored in an attempt to remedy the violation of this assumption. First, residuals of differences scores were calculated and Kolmogorov-Smirnov's and Shapiro-Wilk's tests of normality were conducted. Residuals of residualized change scores were normally distributed for one measure of normality for the formerly depressed participants exposed to the neutral mood induction (Shapiro-Wilk: D(23) = .93, p = .12). However, residuals of residualized change scores were not normally distributed for the remaining groups and conditions (p's < .05). Second, residualized change scores were transformed using the reciprocal transformation. Levene's test for homogeneity of variance continued to be significant (F(1,123) = 4.07, p = .05), indicating that this assumption underlying ANOVA was still not met. Consequently, the one-way ANOVA was conducted with the α level decreased from .05 to .01. The main effect of condition (F(1,109) = 2.32, p = .13, η^2 = .02) was not significant, indicating that there was no significant difference in reporting of hostility on the PANAS-X between mood induction conditions when using residualized change scores.

One-way repeated measures ANOVA. BMI (p =.05) and current depression symptoms (p < .001) were significantly associated with hostility on the PANAS-X and were included in the

final model. The remaining covariates that were under consideration were not significantly associated with hostility on the PANAS-X (all *p*'s > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant (PANAS-X H_{PRE}: $F(1,118) = 3.14$, $p = .08$; PANAS-X H_{POST}: $F(1,118) = 3.38$, $p = .07$), indicating that this assumption underlying ANOVA was met. The main effect of condition ($F(1,116) = 1.22$, $p = .27$, $\eta^2 = .01$) was not significant, indicating that there was no significant difference in reporting of hostility on the PANAS-X between mood induction conditions when using repeated measures.

PANAS-X S. Analyses conducted using the PANAS-X sadness negative basic emotional scale are reviewed below.

One-way ANOVA – difference score. Current depressive symptoms ($p = .03$) were significantly associated with sadness on the PANAS-X and were included in the final model. The remaining covariates that were under consideration were not significantly associated with sadness on the PANAS-X (all *p*'s > .05) and were dropped from the final model. Levene's test for homogeneity of variance was significant ($F(1,118) = 16.56$, $p < .001$), indicating that this assumption underlying ANOVA was not met. Of note, this did not appear to be driven by the inclusion of the covariate. Without the covariate, Levene's test for homogeneity of variance was significant ($F(1,123) = 11.42$, $p = .001$).

In line with the planned analyses, multiple steps were explored in an attempt to remedy the violation of this assumption. First, residuals of differences scores were calculated and Kolmogorov-Smirnov's and Shapiro-Wilk's tests of normality were conducted. Residuals of difference scores were normally distributed for the formerly depressed participants exposed to the neutral mood induction (Kolmogorov-Smirnov: $D(23) = .14$, $p = .20$; Shapiro-Wilk: $D(23) = .95$, $p = .30$) and for one measure of normality for the formerly depressed participants exposed to

the sad mood induction (Kolmogorov-Smirnov: $D(19) = .20$, $p = .06$). However, residuals of difference scores were not normally distributed for the remaining groups and conditions (all *p's* < .05). Second, difference scores were transformed using the reciprocal transformation. Levene's test for homogeneity of variance continued to be significant ($F(1,123) = 14.33$, $p < .001$), indicating that this assumption underlying ANOVA was still not met. Consequently, the one-way ANOVA was conducted with the α level decreased from .05 to .01. Participants exposed to the sad mood induction reported significantly higher levels of sadness on the PANAS-X post-mood induction ($F(1,117) = 11.67$, $p = .001$, $\eta^2 = .09$) than participants exposed to the neutral mood induction when using difference scores.

One-way ANOVA – residualized change score. Current depressive symptoms ($p < .001$) were significantly associated with sadness on the PANAS-X and were included in the final model. The remaining covariates that were under consideration were not significantly associated with sadness on the PANAS-X (all *p*'s > .05) and were dropped from the final model. Levene's test for homogeneity of variance was significant ($F(1,118) = 20.67$, $p < .001$), indicating that this assumption underlying ANOVA was not met. Of note, this did not appear to be driven by the inclusion of the covariate. Without the covariate, Levene's test for homogeneity of variance was significant ($F(1,123) = 17.35$, $p < .001$).

In line with the planned analyses, multiple steps were explored in an attempt to remedy the violation of this assumption. First, residuals of differences scores were calculated and Kolmogorov-Smirnov's and Shapiro-Wilk's tests of normality were conducted. Residuals of difference scores were normally distributed for the formerly depressed participants exposed to the neutral mood induction (Kolmogorov-Smirnov: $D(23) = .14$, $p = .20$; Shapiro-Wilk: $D(23) = .95$, $p = .30$) and for one measure of normality for the formerly depressed participants exposed to

the sad mood induction (Kolmogorov-Smirnov: $D(19) = .20$, $p = .06$). However, residuals of difference scores were not normally distributed for the remaining groups and conditions (all *p's* < .05). Second, residualized change scores were transformed using the reciprocal transformation. Levene's test for homogeneity of variance continued to be significant ($F(1,123) = 11.89$, $p = .001$), indicating that this assumption underlying ANOVA was still not met. Consequently, the one-way ANOVA was conducted with the α level decreased from .05 to .01. Participants exposed to the sad mood induction reported significantly higher levels of sadness on the PANAS-X post-mood induction ($F(1,117) = 12.76$, $p = .001$, $\eta^2 = .10$) than participants exposed to the neutral mood induction when using residualized change scores.

One-way repeated measures ANOVA. Current depressive symptoms ($p < .001$) were significantly associated with sadness on the PANAS-X and were included in the final model. The remaining covariates that were under consideration were not significantly associated with sadness on the PANAS-X (all *p*'s > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant (PANAS-X S_{PRE}: $F(1,118) = .12$, $p = .74$; PANAS-X S_{POST}: $F(1,118) = .82$, $p = .37$), indicating that this assumption underlying ANOVA was met. The main effect of condition ($F(1,117) = .13$, $p = .72$, $\eta^2 = .001$) was not significant, indicating that there was no significant difference in reporting of sadness on the PANAS-X between mood induction conditions when using repeated measures.

Table 15. *Means, Standard Deviations, P Values, and Effect Sizes for Measures Used for the Manipulation Check by Condition*

			Condition					
	Total (N = 123)		SMI (n = 60)		NMI (n = 63)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
VAS_{PRE}	8.67	13.29	9.05	14.90	8.30	11.64		
VAS_{POST}	24.99	26.22	38.18	28.23	12.22	16.00		
VAS_{DS}	15.61	24.33	28.33	27.25	3.49	12.32	< .001	.27
Z_{RES}VAS	-.14	.89	.52	1.09	-.52	.52	< .001	.29
VAS_{RM}							< .001	.19
	Total (N = 120)		SMI (n = 61)		NMI (n = 59)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
PANAS-X N_{PRE}	14.80	5.62	14.73	6.18	14.87	5.03		
PANAS-X N_{POST}	15.35	5.58	15.65	6.00	15.05	5.16		
PANAS-X N_{DS}	.47	2.33	.79	2.63	.14	1.93	.13	.02
Z_{RES}PANAS-X N	-.05	.75	.14	1.13	-.19	.71	.02[a]	.05
PANAS-X N_{RM}							.46	.005
	Total (N = 123)		SMI (n = 62)		NMI (n = 61)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
PANAS-X P_{PRE}	29.82	6.42	29.52	6.46	30.11	6.42		
PANAS-X P_{POST}	27.66	7.55	26.84	7.93	28.48	7.12		
PANAS-X P_{DS}	-2.14	3.36	-2.71	3.76	-1.56	2.81	.06	.03
Z_{RES}PANAS-X P	.14	.91	-.13	.95	.16	.68	.12	.02
PANAS-X P_{RM}							.38	.007
	Total (N = 123)		SMI (n = 61)		NMI (n = 62)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
PANAS-X F_{PRE}	8.96	3.81	8.94	4.30	8.98	3.30		
PANAS-X F_{POST}	8.94	3.79	8.89	3.92	9.00	3.68		
PANAS-X F_{DS}	-.06	1.58	-.05	1.61	-.07	1.56	.96	< .001
Z_{RES}PANAS-X F	.02	.85	-.02	.91	.003	1.02	.89	< .001
PANAS-X F_{RM}							.78	.001
	Total (N = 123)		SMI (n = 63)		NMI (n = 60)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
PANAS-X G_{PRE}	7.91	3.32	7.73	3.25	8.08	3.40		
PANAS-X G_{POST}	8.27	3.62	8.37	3.97	8.18	3.24		
PANAS-X G_{DS}	.24	1.36	.55	1.65	-.09	.87	.002	.08
Z_{RES}PANAS-X G	-.15	.36	.14	1.05	-.24	.51	.004	.07
PANAS-X G_{RM}							.90	< .001

Table 15 Continued

	Total (N = 125)		SMI (n = 63)		NMI (n = 62)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
PANAS-X H_{PRE}	8.46	3.19	8.49	3.59	8.42	2.77		
PANAS-X H_{POST}	8.66	3.27	8.97	3.82	8.34	2.57		
PANAS-X H_{DS}	.14	1.79	.43	2.15	-.15	1.29	.09	.03
Z_{RES}PANAS-X H	-.12	.73	.14	1.18	-.15	.73	.13	.02
PANAS-X H_{RM}							.27	.01
	Total (N = 125)		SMI (n = 63)		NMI (n = 62)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
PANAS-X S_{PRE}	7.80	3.39	7.52	3.65	8.08	3.12		
PANAS-X S_{POST}	8.06	3.51	8.32	3.96	7.79	3.00		
PANAS-X S_{DS}	.20	1.83	.71	2.18	-.32	1.18	.001	.09
Z_{RES}PANAS-X S	-10	.80	.24	1.16	-.28	.56	.001	.10
PANAS-X S_{RM}							.72	.001

Note. SMI = sad mood induction; NMI = neutral mood induction; VAS = Visual Analogue Scale; $_{DS}$ = difference score; Z_{RES} = residualized change scores; PANAS-X = Positive and Negative Affect Scale – Expanded Form; N = negative affect general dimension scale; [a] = not significant due to adjusted *p* value; P = positive affect general dimension scale; F = fear basic negative emotional scale; H = hostility basic negative emotional scale; G = guilt basic negative emotional scale; S = sadness basic negative emotional scale; $_{RM}$ = repeated measures.

Hypothesis 2

A series of 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) factorial ANOVAs using difference scores and residualized change scores and a 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) repeated measures ANOVA were used to test the hypothesis that formerly depressed and healthy control participants exposed to the sad mood induction would report significantly more dysphoric mood on the VAS post-mood induction than formerly depressed and healthy control participants exposed to the neutral mood inductions. Means, standard deviations, *p* values, and effect sizes for session 2 mood measures by group are presented in Table 16 while means, standard deviations, *p* values, and effect sizes for session 2 mood measures by group and condition are presented in Table 17. Planned comparisons were conducted using contrast analyses. Means, standard deviations, and *p* values for session 2 mood measures for planned comparisons are presented in Table 18.

VAS. Analyses conducted using the VAS are reviewed below.

2 X 2 factorial ANOVA – difference score. None of the covariates that were under consideration were significantly associated with mood reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was significant ($F(3,119) = 17.15$, $p < .001$), indicating that this assumption underlying ANOVA was not met.

In line with the planned analyses, multiple steps were explored in an attempt to remedy the violation of this assumption. First, residuals of differences scores were calculated and Kolmogorov-Smirnov's and Shapiro-Wilk's tests of normality were conducted. Residuals of difference scores were normally distributed for the formerly depressed participants exposed to the sad mood induction (Kolmogorov-Smirnov: $D(17) = .15$, $p = .20$; Shapiro-Wilk: $D(17) = .95$, $p = .38$). However, residuals of difference scores were not normally distributed for the remaining groups and conditions (all *p's* < .05). Second, difference scores were transformed using the reciprocal transformation. Levene's test for homogeneity of variance continued to be significant ($F(3,119) = 14.60$, $p < .001$), indicating that this assumption was still not met. Consequently, the one-way ANOVA was conducted with the α level decreased from .05 to .01.

Formerly depressed participants reported significantly higher levels of sadness on the VAS post-mood induction ($F(1,119) = 7.49$, $p = .007$, $\eta^2 = .06$) than healthy control participants. In addition, participants exposed to the sad mood induction reported significantly higher levels of sadness on the VAS post-mood induction ($F(1,119) = 51.17$, $p < .001$, $\eta^2 = .30$) than participants exposed to the neutral mood induction. The group by condition interaction was not significant ($F(1,119) = 3.20$, $p = .08$, $\eta^2 = .03$) when using difference scores. It is possible that this is due to lack of power rather than lack of effect. Using contrast analyses that did not assume equal variances, formerly depressed and healthy control participants exposed to the sad mood

induction reported significantly higher levels of sadness on the VAS post-mood induction ($t(37.12) = 6.23$, $p_L < .001$, $p_Q = .08$, $p_C < .001$) compared to formerly depressed and healthy control participants exposed to the neutral mood induction. In addition, formerly depressed participants exposed to the sad mood induction reported significantly higher levels of sadness on the VAS post-mood induction ($t(18.16) = 4.14$, $p_L < .001$, $p_Q = .08$, $p_C < .001$) compared to healthy control participants exposed to the sad mood induction and formerly depressed and healthy control participants exposed to the neutral mood induction.

2 X 2 factorial ANOVA – residualized change score. None of the covariates that were under consideration were significantly associated with mood reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was significant ($F(3,119) = 15.94$, $p < .001$), indicating that this assumption underlying ANOVA was not met.

In line with the planned analyses, multiple steps were explored in an attempt to remedy the violation of this assumption. First, residuals of residualized change scores were calculated and Kolmogorov-Smirnov's and Shapiro-Wilk's tests of normality were conducted. Residuals of residualized change scores were normally distributed for the formerly depressed participants exposed to the sad mood induction (Kolmogorov-Smirnov: $D(17) = .15$, $p = .20$; Shapiro-Wilk: $D(17) = .95$, $p = .38$). However, residuals of residualized change scores were not normally distributed for the remaining groups and conditions (all *p's* < .05). Second, residualized change scores were transformed using the reciprocal transformation. Levene's test for homogeneity of variance continued to be significant ($F(3,119) = 14.98$, $p < .001$), indicating that this assumption was still not met. Consequently, the factorial ANOVA was conducted with the α level decreased from .05 to .01.

Formerly depressed participants reported significantly higher levels of sadness on the VAS post-mood induction ($F(1,119) = 10.16$, $p = .002$, $\eta^2 = .08$) than healthy control participants. In addition, participants exposed to the sad mood induction reported significantly higher levels of sadness on the VAS post-mood induction ($F(1,119) = 53.94$, $p < .001$, $\eta^2 = .31$) than participants exposed to the neutral mood induction. The group by condition interaction was not significant ($F(1,119) = 2.18$, $p = .14$, $\eta^2 = .02$) when using residualized change scores. It is possible that this is due to lack of power rather than lack of effect. Using contrast analyses that did not assume equal variances, formerly depressed and healthy control participants exposed to the sad mood induction reported significantly higher levels of sadness on the VAS post-mood induction ($t(38.07) = 6.54$, $p_L < .001$, $p_Q = .14$, $p_C < .001$) compared to formerly depressed and healthy control participants exposed to the neutral mood induction. In addition, formerly depressed participants exposed to the sad mood induction reported significantly higher levels of sadness on the VAS post-mood induction ($t(18.22) = 4.37$, $p_L < .001$, $p_Q = .14$, $p_C < .001$) compared to healthy control participants exposed to the sad mood induction and formerly depressed and healthy control participants exposed to the neutral mood induction.

2 X 2 repeated measures ANOVA. None of the covariates that were under consideration were significantly associated with mood reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant for the pre-mood induction measure (VAS_{PRE}: $F(3,119) = 2.48$, $p = .06$) but was significant for the post-mood induction measure (VAS_{POST}: $F(3,119) = 12.07$, $p < .001$), indicating that this assumption underlying ANOVA was not met.

In line with the planned analyses, multiple steps were explored in an attempt to remedy the violation of this assumption. Residuals could not be calculated as there was only one measure

for each calculation. The pre- and post-mood induction measures were transformed using the reciprocal transformation. Levene's test for homogeneity of variance continued to be significant (VAS_{PRE}: $F(3,119) = 2.97$, $p = .04$; VAS_{POST}: $F(3,119) = 9.71$, $p < .001$), indicating that this assumption underlying ANOVA was still not met. Consequently, the factorial ANOVA was conducted with the α level decreased from .05 to .01.

Formerly depressed participants reported significantly higher levels of sadness on the VAS post-mood induction ($F(1,119) = 11.95$, $p = .001$, $\eta^2 = .09$) than healthy control participants. In addition, participants exposed to the sad mood induction reported significantly higher levels of sadness on the VAS post-mood induction ($F(1,119) = 23.60$, $p < .001$, $\eta^2 = .17$) than participants exposed to the neutral mood induction. The group by condition interaction was not significant ($F(1,119) = .02$, $p = .90$, $\eta^2 < .001$) when using repeated measures. Using contrast analyses that did not assume equal variances, formerly depressed and healthy control participants exposed to the sad mood induction reported significantly higher levels of sadness on the VAS post-mood induction ($F(1,121) = 22.79$, $p < .001$) compared to formerly depressed and healthy control participants exposed to the neutral mood induction. In addition, formerly depressed participants exposed to the sad mood induction reported significantly higher levels of sadness on the VAS post-mood induction ($F(1,121) = 14.58$, $p < .001$) compared to healthy control participants exposed to the sad mood induction and formerly depressed and healthy control participants exposed to the neutral mood induction.

Post-hoc power analyses. Post-hoc power analyses were conducted using G*Power 3.1.9.2 (Faul et al., 2007) to determine the power achieved by the VAS analyses. Results indicated that the current model had a power of .26 for the difference score, .16 for the residualized change score, and .01 for the repeated measures. Results suggested that given the

current study's sample size, α level, and observed effect size, there was a 1 to 26% chance of detecting an effect depending on which analytic technique was used.

Sensitivity analyses. Sensitivity analyses were conducted using G*Power 3.1.9.2 (Faul et al., 2007) to determine the minimum effect size needed to obtain significant results for the VAS analyses. Results indicated that the required effect size was $f = .31$ for the factorial ANOVAs and $f = .30$ for the repeated measures ANOVA. Results suggested that given the current study's sample size and α level, at least a medium effect size was required to obtain significant results if a power of .80 was achieved.

Table 16. *Means, Standard Deviations, P Values, and Effect Sizes for Session 2 Mood Measures by Group*

			Group					
	Total (N = 123)		FD (n = 40)		HC (n = 83)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
VAS_{PRE}	8.67	13.29	11.78	15.02	7.17	12.19		
VAS_{POST}	24.99	26.22	33.90	27.81	20.59	24.39		
VAS_{DS}	15.61	24.33	20.85	29.47	13.08	21.15	.007	.06
$Z_{RES}VAS$	-.14	.89	.25	1.15	-.14	.89	.002	.08
VAS_{RM}							.001	.09

Note. The difference between pre- and post-mood induction measures may not precisely equal the difference score as outlier data was addressed differently depending on the type of analyses conducted; FD = formerly depressed; HC = healthy control; VAS = Visual Analogue Scale; $_{PRE}$ = pre-mood induction; $_{POST}$ = post-mood induction; $_{DS}$ = difference score; Z_{RES} = residualized change scores; $_{RM}$ = repeated measures.

Table 17. *Means, Standard Deviations, P Values, and Effect Sizes for Session 2 Mood Measures by Group and Condition*

			Group and Condition									
	Total (N = 123)		FD/SMI (n = 17)		FD/NMI (n = 23)		HC/SMI (n = 43)		HC/NMI (n = 40)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
VAS_{PRE}	8.67	13.29	9.22	15.11	13.78	14.97	8.98	14.99	5.22	7.94		
VAS_{POST}	24.99	26.22	51.83	27.38	19.87	18.93	32.47	26.87	7.83	12.28		
VAS_{DS}	15.61	24.33	41.12	29.60	5.87	18.71	23.28	24.85	2.13	6.19	.08	.03
$Z_{RES}VAS$	-.14	.89	1.05	1.16	-.35	.71	.31	1.00	-.62	.34	.14	.02
VAS_{RM}											.90	< .001

Note. The difference between pre- and post-mood induction measures may not precisely equal the difference score as outlier data was addressed differently depending on the type of analyses conducted; FD = formerly depressed; HC = healthy control; SMI = sad mood induction; NMI = neutral mood induction; VAS = Visual Analogue Scale; $_{PRE}$ = pre-mood induction; $_{POST}$ = post-mood induction; $_{DS}$ = difference score; Z_{RES} = residualized change scores; $_{RM}$ = repeated measures.

Table 18. *Means, Standard Deviations, and P Values for Session 2 Mood Measures for Planned Comparisons*

			Group and Condition						
	Total (N = 123)		FD+HC/SMI (n = 60)		FD+HC/NMI (n = 63)				
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	p_L	p_Q	p_C
VAS_{PRE}	8.67	13.29	9.05	14.90	8.30	11.64			
VAS_{POST}	24.99	26.22	38.18	28.23	12.22	16.00			
VAS_{DS}	15.61	24.33	28.33	27.25	3.49	12.32	< .001	.08	< .001
$Z_{RES}VAS$	-.14	.89	.52	1.09	-.52	.52	< .001	.14	< .001
VAS_{RM}							< .001		
	Total (N = 123)		FD/SMI (n = 17)		FD/NMI, HC/SMI, HC/NMI (n = 106)				
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	p_L	p_Q	p_C
VAS_{PRE}	8.67	13.29	9.22	15.11	8.57	13.04			
VAS_{POST}	24.99	26.22	51.83	27.38	20.43	23.23			
VAS_{DS}	15.61	24.33	41.12	29.60	11.52	20.78	< .001	.08	< .001
$Z_{RES}VAS$	-.14	.89	1.05	1.16	-.18	.85	< .001	.14	< .001
VAS_{RM}							< .001		

Note. The difference between pre- and post-mood induction measures may not precisely equal the difference score as outlier data was addressed differently depending on the type of analyses conducted; FD = formerly depressed; HC = healthy control; SMI = sad mood induction; NMI = neutral mood induction; $_L$ = linear; $_Q$ = quadratic; $_C$ = cubic; VAS = Visual Analogue Scale; $_{PRE}$ = pre-mood induction; $_{POST}$ = post-mood induction; $_{DS}$ = difference score; Z_{RES} = residualized change scores; $_{RM}$ = repeated measures.

Cardiovascular Measures

During session 2, participants' physiological responding was recorded to assess cardiovascular reactivity to the mood induction (i.e., Hypothesis 3) and recovery from the mood induction (i.e., Hypothesis 3). The physiological recordings of interest are cardiovascular measures derived from ECG and ICG. ECG was used to collect data that was used to calculate HP and RSA. MindWare Technologies Ltd. (2009) hardware and Biolab 3.1 analysis software set to collect ECG data falling within -5 and 5 volts with a sampling rate of 1,000 hertz were utilized in conjunction with the five GSC electrode sensors located on the participants' right collarbone, bottom left rib, bottom right rib, jugular notch, and sternum. ICG was used to collect data that was used to calculate RSA, CO, and PEP. Of note, CO was dropped from analyses. Typical values for resting CO range from 4 to 12 liters per minute (J. Schmidt, personal communication, April 16, 209). Average CO values ranged from 1.62 to 365.73 during baseline, 1.49 to 390.58 during the mood induction, and 1.69 to 175.80 during recovery, indicating that this cardiovascular measure was not accurate. It was determined that this issue was due to inaccurate SV values. SV is not used to calculate HP, RSA, or PEP and therefore, these cardiovascular measures were not affected. MindWare Technologies Ltd. (2009) hardware and Biolab 3.1 analysis software set to collect ICG data falling at a sampling rate of 1,000 Hertz and calibrated at .10 volts per one-ohm change for Z0 and 1.00 volts per ohms per second for dZ/dt were utilized in conjunction with the two GSC electrode sensors located on the participants' mid-back and upper-back parallel within 1.50 inches of the jugular notch and sternum sensors.

Cardiovascular functioning at baseline was calculated for each cardiovascular measure using the average obtained during the last two and five minutes of baseline (i.e., $HP_{BL2/BL5}$,

$RSA_{BL2/BL5}$, $CO_{BL2/BL5}$, and $PEP_{BL2/BL5}$). No outlier data was present for HP_{BL2}, HP_{BL5}, RSA_{BL2}, RSA_{BL5}, PEP_{BL2}, and PEP_{BL5}.

Cardiovascular functioning for the mood induction was calculated for each cardiovascular measure using the average obtained during the first two and five minutes of the mood induction (i.e., $HP_{MI2/5}$, $RSA_{MI2/5}$, $CO_{MI2/5}$, and $PEP_{MI2/5}$). No outlier data was present for HP_{MI2}, HP_{MI5}, RSA_{MI2}, RSA_{MI5}, PEP_{MI2}, and PEP_{MI5}.

Cardiovascular functioning for recovery was calculated for each cardiovascular measure using the average obtained during the last two and five minutes of recovery (i.e., $HP_{RC2/5}$, $RSA_{RC2/5}$, $CO_{RC2/5}$, and $PEP_{RC2/5}$). Outlier data for the PEP_{RC2} (n = 1) were winsorized to address extreme values. No outlier data was present for HP_{RC2}, HP_{RC5}, RSA_{RC2}, RSA_{RC5}, and PEP_{RC5}.

Cardiovascular reactivity was operationalized as the difference score for the average of each cardiovascular measure (i.e., $HP_{DS2RA/DS5RA}$, $RSA_{DS2RA/DS5RA}$, $CO_{DS2RA/DS5RA}$, and $PEP_{DS2RA/DS5RA}$) and residualized change scores for the average of each cardiovascular measure (i.e., $Z_{RES2/RES5}HP_{RA}$, $Z_{RES2/RES5}RSA_{RA}$, $Z_{RES2/RES5}CO_{RA}$, and $Z_{RES2/RES5}PEP_{RA}$) assessed during baseline and the mood induction (i.e., $HP_{BL2/BL5/MI2/MI5}$, $RSA_{BL2/BL5/MI2/MI5}$, $CO_{BL2/BL5/MI2/MI5}$, and $PEP_{BL2/BL5/MI2/MI5}$). Outlier data for the RSA_{DS2RA} (n = 1), RSA_{DS5RA} (n = 1), PEP_{DS2RA} (n = 2), and PEP_{DS5RA} (n = 2) were winsorized to address extreme values. No outlier data was present for HP_{DS2RA} and HP_{DS5RA}. In addition, outlier data for the $Z_{RES2}RSA_{RA}$ (n = 1), $Z_{RES5}RSA_{RA}$ (n = 1), $Z_{RES2}PEP_{RA}$ (n = 2), and $Z_{RES5}PEP_{RA}$ (n = 2) were winsorized to address extreme values.

Cardiovascular recovery was operationalized as the difference score for the average of each cardiovascular measure (i.e., $HP_{DS2RC/DS5RC}$, $RSA_{DS2RC/DS5RC}$, $CO_{DS2RC/DS5RC}$, and $PEP_{DS2RC/DS5RC}$) and residualized change scores for the average of each cardiovascular measure (i.e., $Z_{RES2/RES5}HP_{RC}$, $Z_{RES2/RES5}RSA_{RC}$, $Z_{RES2/RES5}CO_{RC}$, and $Z_{RES2/RES5}PEP_{RC}$) during the mood

induction and the recovery period (i.e., $HP_{BL2/BL5/RC2/RC5}$, $RSA_{BL2/BL5/RC2/RC5}$, $CO_{BL2/BL5/RC2/RC5}$, and $PEP_{BL2/BL5/RC2/RC5}$). Outlier data for the RSA_{DS5RC} (n = 3), PEP_{DS2RC} (n = 4), and PEP_{DS5RC} (n = 2) were winsorized to address extreme values. No outlier data was present for HP_{DS2RA}, and HP_{DS5RA}, and RSA_{DS2RC}. In addition, outlier data for the $Z_{RES2}HP_{RC}$ (n = 1), $Z_{RES5}HP_{RC}$ (n = 1), and $Z_{RES2}PEP_{RC}$ (n = 4) were winsorized to address extreme values

Descriptive statistics. Descriptive statistics for session 2 cardiovascular measures are presented in Table 19. The baseline (i.e., $_{BL2/BL5}$), mood induction (i.e., $_{MI2/MI5}$), and recovery (i.e., $_{RC2/RC5}$) scores are single item measures. Consequently, Cronbach's alpha could not be calculated. The difference (i.e., $_{DS}$) and residualized change (i.e., Z_{RES2}/Z_{RES5}) scores are calculated measures. Consequently, Cronbach's alpha could not be calculated.

Table 19. *Descriptive Statistics for Session 2 Cardiovascular Measures*

Measure	*M*	*SD*	Sample range
HP_{BL2}	781.45	104.55	487.51-1,060.85
HP_{BL5}	785.51	105.88	538.41-1,045.88
HP_{MI2}	799.21	111.48	591.72-1,045.57
HP_{MI5}	788.89	108.15	600.29-1,049.48
HP_{RC2}	783.98	102.44	627.45-1,109.76
HP_{RC5}	787.42	103.62	633.77-1,109.32
HP_{DS2RA}	17.76	39.29	-79.84-159.61
$Z_{RES2}HP_{RA}$	.00	1.00	-2.48-3.64
HP_{DS5RA}	3.38	33.73	-91.22-126.04
$Z_{RES5}HP_{RA}$	.00	1.00	-2.67-3.48
HP_{DS2RC}	4.23	49.12	-97.39-236.03
$Z_{RES2}HP_{RC}$	.07	.78	-1.40-1.70
HP_{DS5RC}	3.70	37.99	-104.38-189.20
$Z_{RES5}HP_{RC}$	.03	.85	-2.69-1.29
RSA_{BL2}	5.84	1.04	3.75-9.19
RSA_{BL5}	5.83	1.01	3.65-9.37
RSA_{MI2}	6.03	1.15	1.81-9.49
RSA_{MI5}	5.89	1.13	1.36-9.44
RSA_{RC2}	5.82	1.00	3.45-8.13
RSA_{RC5}	5.79	.93	3.67-8.45
RSA_{DS2RA}	.24	.61	-1.22-1.97
$Z_{RES2}RSA_{RA}$	.02	.73	-1.52-1.89
RSA_{DS5RA}	.11	.45	-.72-1.78
$Z_{RES5}RSA_{RA}$	.03	.58	-1.02-1.38
RSA_{DS2RC}	-.02	.70	-1.82-1.99
$Z_{RES2}RSA_{RC}$	.00	1.00	-2.99-2.65
RSA_{DS5RC}	-.05	.43	-1.37-.96
$Z_{RES5}RSA_{RC}$	.00	1.00	-2.11-3.12
PEP_{BL2}	120.61	11.90	90-146.50
PEP_{BL5}	120.37	11.68	90-145.80
PEP_{MI2}	119.77	12.74	77.00-147.00
PEP_{MI5}	119.91	13.04	75.00-146.00
PEP_{RC2}	120.15	14.33	58.50-145.50
PEP_{RC5}	120.91	12.24	87.60-146.00
PEP_{DS2RA}	-.48	4.19	-10.50-9.00
$Z_{RES2}PEP_{RA}$	-.01	.65	-1.33-1.06
PEP_{DS5RA}	-.31	4.13	-18.80-10.40
$Z_{RES5}PEP_{RA}$	.04	.64	-1.26-1.47
PEP_{DS2RC}	.68	5.06	-17.00-15.50
$Z_{RES2}PEP_{RC}$	.09	.53	-1.80-1.51
PEP_{DS5RC}	.79	4.57	-16.00-11.00
$Z_{RES5}PEP_{RC}$	.00	1.00	-3.50-1.85

Note. The difference between baseline and mood induction or mood induction and recovery measures may not precisely equal the difference score as outlier data was addressed differently depending on the type of analyses conducted; HP = heart period at baseline; $_{BL}$ = baseline; $_{MI}$ = mood induction; $_{RC}$ = recovery; $_{2}$ = average obtained during a two-minute interval; $_{5}$ = average obtained during a five-minute interval; $_{DS}$ = difference score; $_{RA}$ = reactivity; Z_{RES} = residualized change scores; RSA = respiratory sinus arrhythmia; PEP = pre-ejection period.

Baseline Cardiovascular Functioning

A series of one-way ANOVAs were used to assess significant group differences in cardiovascular functioning at baseline. It was expected that formerly depressed and healthy control participants would exhibit similar cardiovascular functioning for HP, RSA, PEP, or CO during baseline.

HP – two-minutes. Analyses conducted for baseline cardiovascular functioning using two-minute averages of HP are reviewed below.

One-way ANOVA. Age (p = .01) and BMI (p = .04) were significantly associated with baseline two-minute HP functioning and were included in the final model. The remaining covariates that were under consideration were not significantly associated with two-minute HP functioning (all p*'s* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(1,90) = .04$, $p = .84$), indicating that this assumption underlying ANOVA was met. The main effect of group ($F(1,88) = .80$, $p = .37$, $\eta^2 = .009$) was not significant, indicating that there was no significant difference in baseline two-minute HP functioning between groups when using univariate analysis.

HP – five-minutes. Analyses conducted for baseline cardiovascular functioning using five-minute averages of HP are reviewed below.

One-way ANOVA. Age (p = .008) and BMI (p = .04) were significantly associated with baseline five-minute HP functioning and were included in the final model. The remaining covariates that were under consideration were not significantly associated with five-minute HP

functioning (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(1,90) = .03$, $p = .86$), indicating that this assumption underlying ANOVA was met. The main effect of group ($F(1,88) = .80$, $p = .37$, $\eta^2 = .009$) was not significant, indicating that there was no significant difference in baseline five-minute HP functioning between groups when using univariate analysis.

RSA – two-minutes. Analyses conducted for baseline cardiovascular functioning using two-minute averages of RSA are reviewed below.

One-way ANOVA. None of the covariates that were under consideration were significantly associated with baseline two-minute RSA functioning (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(1,93) = .05$, $p = .82$), indicating that this assumption underlying ANOVA was met. The main effect of group ($F(1,93) = .47$, $p = .50$, $\eta^2 = .005$) was not significant, indicating that there was no significant difference in baseline two-minute RSA functioning between groups when using univariate analysis.

RSA – five-minutes. Analyses conducted for baseline cardiovascular functioning using five-minute averages of RSA are reviewed below.

One-way ANOVA. None of the covariates that were under consideration were significantly associated with baseline five-minute RSA functioning (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(1,93) = .26$, $p = .61$), indicating that this assumption underlying ANOVA was met. The main effect of group ($F(1,93) = .80$, $p = .38$, $\eta^2 = .008$) was not significant, indicating that there was no significant difference in baseline five-minute RSA functioning between groups when using univariate analysis.

PEP – two-minutes. Analyses conducted for baseline cardiovascular functioning using two-minute averages of PEP are reviewed below.

One-way ANOVA. Current state anxiety symptoms (p = .03) were significantly associated with baseline two-minute PEP functioning and were included in the final model. The remaining covariates that were under consideration were not significantly associated with two-minute PEP functioning (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(1,84) = .19$, $p = .66$), indicating that this assumption underlying ANOVA was met. The main effect of group ($F(1,83) = 2.04$, $p = .16$, $\eta^2 = .02$) was not significant, indicating that there was no significant difference in baseline two-minute PEP functioning between groups when using univariate analysis.

PEP – five-minutes. Analyses conducted for baseline cardiovascular functioning using five-minute averages of PEP are reviewed below.

One-way ANOVA. Current state anxiety symptoms (p = .01) were significantly associated with baseline five-minute PEP functioning and were included in the final model. The remaining covariates that were under consideration were not significantly associated with five-minute PEP functioning (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(1,84) = .06$, $p = .80$), indicating that this assumption underlying ANOVA was met. The main effect of group ($F(1,83) = 1.60$, $p = .21$, $\eta^2 = .02$) was not significant, indicating that there was no significant difference in baseline five-minute PEP functioning between groups when using univariate analysis.

Hypothesis 3

A series of 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) factorial ANOVAs using difference scores and residualized change scores and a series of 2

(group: formerly depressed, healthy control) X 2 (condition: sad, neutral) repeated measures ANOVAs were used to test the hypothesis that formerly depressed individuals exposed to the sad mood induction would exhibit a maladaptive pattern of cardiovascular reactivity (i.e., decreased HP, RSA, and CO and increased PEP) during the mood induction compared to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions. Means, standard deviations, *p* values, and effect sizes for session 2 cardiovascular measures by group are presented in Table 20 while means, standard deviations, *p* values, and effect sizes for session 2 cardiovascular measures by group and condition are presented in Table 21. Planned comparisons were conducted using contrast analyses. Means, standard deviations, and *p* values for session 2 mood measures for planned comparisons are presented in Table 22.

HP – two-minutes. Analyses conducted for cardiovascular reactivity using two-minute averages of HP are reviewed below.

2 X 2 factorial ANOVA – difference score. None of the covariates that were under consideration were significantly associated with two-minute HP reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,92) = .94$, $p = .43$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,92) = 2.52$, $p = .12$, $\eta^2 = .03$) or condition ($F(1,92) = 1.15$, $p = .29$, $\eta^2 = .01$) nor the group by condition interaction ($F(1,92) = .24$, $p = .62$, $\eta^2 = .003$) were significant, indicating that there was no significant difference in two-minute HP reactivity between groups or conditions or the group by condition interaction when using difference scores. Contrast analyses revealed that there was no significant difference in two-minute HP reactivity ($t(92) = 1.62$, $p_L = .06$, $p_Q = .62$, $p_C = .80$) when comparing formerly depressed participants

exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 factorial ANOVA – residualized change score. None of the covariates that were under consideration were significantly associated with two-minute HP reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,92) = .95$, $p = .42$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,92) = 2.51$, $p = .12$, $\eta^2 = .03$) or condition ($F(1,92) = 1.14$, $p = .29$, $\eta^2 = .01$) nor the group by condition interaction ($F(1,92) = .25$, $p = .62$, $\eta^2 = .003$) were significant, indicating that there was no significant difference in two-minute HP reactivity between mood induction conditions or the group by condition interaction when using residualized change scores. Contrast analyses revealed that there was no significant difference in two-minute HP reactivity ($t(92) = 1.62$, $p_L = .07$, $p_Q = .62$, $p_C = .80$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 repeated measures ANOVA. Age ($p = .01$) and BMI ($p = .02$) were significantly associated with two-minute HP reactivity and were included in the final model. The remaining covariates that were under consideration were not significantly associated with two-minute HP reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant (HP_{BL2}: $F(3,88) = 1.54$, $p = .21$; HP_{MI2}: $F(3,88) = 2.00$, $p = .12$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,86) = .07$, $p = .79$, $\eta^2 = .001$) or condition ($F(1,86) = 1.78$, $p = .19$, $\eta^2 = .02$) nor the group by condition interaction ($F(1,86) = .61$, $p = .44$, $\eta^2 = .007$) were significant, indicating that there

was no significant difference in two-minute HP reactivity between groups or conditions or the group by condition interaction when using repeated measures. Contrast analyses revealed that there was no significant difference in two-minute HP reactivity ($F(1,88) = .54$, $p = .46$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

HP – five-minutes. Analyses conducted for cardiovascular reactivity using five-minute averages of HP are reviewed below.

2 X 2 factorial ANOVA – difference score. Franco-American ethnicity ($p = .002$) was significantly associated with five-minute HP reactivity and was included in the final model. The remaining covariates that were under consideration were not significantly associated with five-minute HP reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,91) = 1.60$, $p = .20$), indicating that this assumption underlying ANOVA was met. Formerly depressed participants exhibited significantly higher levels of five-minute HP reactivity during the mood induction ($F(1,90) = 4.13$, $p = .05$, $\eta^2 = .04$) than healthy control participants. Neither the main effect of condition ($F(1,90) = 2.10$, $p = .15$, $\eta^2 = .02$) nor the group by condition interaction ($F(1,90) = 1.22$, $p = .27$, $\eta^2 = .01$) were significant, indicating that there was no significant difference in five-minute HP reactivity between mood induction conditions or the group by condition interaction when using difference scores. Using contrast analyses, formerly depressed participants exposed to the sad mood induction reported exhibited higher levels of five-minute HP reactivity during the mood induction ($F(3,90) = 2.18$, $p = .10$) compared to healthy control participants exposed to the sad

mood induction and formerly depressed and healthy control participants exposed to the neutral mood induction.

2 X 2 factorial ANOVA – residualized change score. Franco-American ethnicity (p = .002) was significantly associated with five-minute HP reactivity and was included in the final model. The remaining covariates that were under consideration were not significantly associated with five-minute HP reactivity (all p*'s* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,91) = 1.41$, p = .25), indicating that this assumption underlying ANOVA was met. Formerly depressed participants exhibited significantly higher levels of five-minute HP reactivity during the mood induction ($F(1,90)$ = 4.49, p = .04, η^2 = .05) than healthy control participants. Neither the main effect of condition ($F(1,90) = 2.20$, p = .14, η^2 = .02) nor the group by condition interaction ($F(1,90) = 1.21$, p = .27, η^2 = .01) were significant, indicating that there was no significant difference in five-minute HP reactivity between mood induction conditions or the group by condition interaction when using residualized change scores. Using contrast analyses, formerly depressed participants exposed to the sad mood induction reported exhibited higher levels of five-minute HP reactivity during the mood induction ($F(3,90) = 2.32$, p = .08) compared to healthy control participants exposed to the sad mood induction and formerly depressed and healthy control participants exposed to the neutral mood induction.

2 X 2 repeated measures ANOVA. Age (p = .01) and BMI (p = .03) were significantly associated with five-minute HP reactivity and were included in the final model. The remaining covariates that were under consideration were not significantly associated with five-minute HP reactivity (all p*'s* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant (HP_{BL5}: $F(3,88) = 2.12$, p = .10; HP_{MI5}: $F(3,88) = 2.19$, p = .10),

indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,86) = .001$, $p = .97$, $\eta^2 < .001$) or condition ($F(1,86) = 2.17$, $p = .14$, $\eta^2 = .03$) nor the group by condition interaction ($F(1,86) = 1.38$, $p = .24$, $\eta^2 = .02$) were significant, indicating that there was no significant difference in five-minute HP reactivity between groups or conditions or the group by condition interaction when using repeated measures. Contrast analyses revealed that there was no significant difference in five-minute HP reactivity ($F(1,88) = 1.29$, $p = .26$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

Post-hoc power analyses. Post-hoc power analyses were conducted using G*Power 3.1.9.2 (Faul et al., 2007) to determine the power achieved by the HP reactivity analyses. Of note, analyses were only conducted using five-minute HP reactivity as this is a more stable measure than of HP reactivity. Results indicated that the current model had a power of .16 for the difference and residualized change scores and .19 for the repeated measures. Results suggested that given the current study's sample size, α level, and observed effect size, there was a 16 to 19% chance of detecting an effect depending on which analytic technique was used.

Sensitivity analyses. Sensitivity analyses were conducted using G*Power 3.1.9.2 (Faul et al., 2007) to determine the minimum effect size needed to obtain significant results for the HP reactivity analyses. Of note, analyses were only conducted using five-minute HP reactivity as this is a more stable measure than two-minute HP reactivity. Results indicated that the required effect size was $f = .29$ for the factorial ANOVAs and $f = .34$ for the repeated measures ANOVA. Results suggested that given the current study's sample size and α level, at least a medium effect size was required to obtain significant results if a power of .80 was achieved.

RSA – two-minutes. Analyses conducted for cardiovascular reactivity using two-minute averages of RSA are reviewed below.

2 X 2 factorial ANOVA – difference score. Education level (p = .01) was significantly associated with two-minute RSA reactivity and was included in the final model. The remaining covariates that were under consideration were not significantly associated with two-minute RSA reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,90) = .50$, $p = .69$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,89) = 2.49$, $p = .12$, $\eta^2 = .03$) or condition ($F(1,89) = .04$, $p = .85$, $\eta^2 < .001$) nor the group by condition interaction ($F(1,89) = .36$, $p = .55$, $\eta^2 = .004$) were significant, indicating that there was no significant difference in two-minute RSA reactivity between groups or conditions or the group by condition interaction when using difference scores. Contrast analyses revealed that there was no significant difference in two-minute RSA reactivity ($F(3,89) = 1.00$, $p = .40$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 factorial ANOVA – residualized change score. Sex (p = .04) and education level (p = .01) were significantly associated with two-minute RSA reactivity and were included in the final model. The remaining covariates that were under consideration were not significantly associated with two-minute RSA reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,90) = .13$, $p = .94$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,88) = .46$, $p = .50$, $\eta^2 = .005$) or condition ($F(1,88) = .24$, $p = .62$, $\eta^2 = .003$) nor the group

by condition interaction ($F(1,88) = .52$, $p = .47$, $\eta^2 = .006$) were significant, indicating that there was no significant difference in two-minute RSA reactivity between groups or conditions or the group by condition interaction when using residualized change scores. Contrast analyses revealed that there was no significant difference in two-minute RSA reactivity ($F(3,88) = .53$, $p = .66$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 repeated measures ANOVA. None of the covariates that were under consideration were significantly associated with two-minute RSA reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant (RSA_{BL2}: $F(3,91) = .87$, $p = .46$; RSA_{MI2}: $F(3,91) = .42$, $p = .74$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,91) = .35$, $p = .56$, $\eta^2 = .004$) or condition ($F(1,91) = .27$, $p = .61$, $\eta^2 = .003$) nor the group by condition interaction ($F(1,91) = 2.48$, $p = .12$, $\eta^2 = .03$) were significant, indicating that there was no significant difference in two-minute RSA reactivity between groups or conditions or the group by condition interaction when using repeated measures. Contrast analyses revealed that there was no significant difference in two-minute RSA reactivity ($F(1,93) = .73$, $p = .40$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

RSA – five-minutes. Analyses conducted for cardiovascular reactivity using five-minute averages of RSA are reviewed below.

2 X 2 factorial ANOVA – difference score. None of the covariates that were under consideration were significantly associated with five-minute RSA reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,91) = 1.38$, $p = .25$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,91) = 1.43$, $p = .23$, $\eta^2 = .02$) or condition ($F(1,91) = .83$, $p = .36$, $\eta^2 = .009$) nor the group by condition interaction ($F(1,91) = .14$, $p = .71$, $\eta^2 = .002$) were significant, indicating that there was no significant difference in five-minute RSA reactivity between groups or conditions or the group by condition interaction when using difference scores. Contrast analyses revealed that there was no significant difference in five-minute RSA reactivity ($t(91) = .89$, $p_L = .15$, $p_Q = .71$, $p_C = .78$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 factorial ANOVA – residualized change score. None of the covariates that were under consideration were significantly associated with five-minute RSA reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,91) = 1.88$, $p = .14$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,91) = .87$, $p = .35$, $\eta^2 = .009$) or condition ($F(1,91) = 1.19$, $p = .28$, $\eta^2 = .01$) nor the group by condition interaction ($F(1,91) = .68$, $p = .41$, $\eta^2 = .007$) were significant, indicating that there was no significant difference in five-minute RSA reactivity between mood induction conditions or the group by condition interaction when using residualized change scores. Contrast analyses revealed that there was no significant difference in five-minute RSA reactivity ($t(91) = .62$ $p_L = .19$, $p_Q = .41$, $p_C = .58$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants

exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 repeated measures ANOVA. None of the covariates that were under consideration were significantly associated with five-minute RSA reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant (RSA_{BL5}: $F(3,91) = 1.41$, $p = .24$, RSA_{MI5}: $F(3,91) = .19$, $p = .90$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,91) = .14$, $p = .71$, $\eta^2 = .002$) or condition ($F(1,91) = .67$, $p = .41$, $\eta^2 = .007$) nor the group by condition interaction ($F(1,91) = 2.12$, $p = .15$, $\eta^2 = .02$) were significant, indicating that there was no significant difference in five-minute RSA reactivity between groups or conditions or the group by condition interaction when using repeated measures. Contrast analyses revealed that there was no significant difference in five-minute RSA reactivity ($F(1,93) = .28$, $p = .60$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

Post-hoc power analyses. Post-hoc power analyses were conducted using G*Power 3.1.9.2 (Faul et al., 2007) to determine the power achieved by the RSA reactivity analyses. Of note, analyses were only conducted using five-minute RSA reactivity as this is a more stable measure than two-minute RSA reactivity. Results indicated that the current model had a power of .12 for the difference score, .13 for the residualized change score, and .20 for the repeated measures. Results suggested that given the current study's sample size, α level, and observed effect size, there was a 12 to 20% chance of detecting an effect depending on which analytic technique was used.

Sensitivity analyses. Sensitivity analyses were conducted using G*Power 3.1.9.2 (Faul et al., 2007) to determine the minimum effect size needed to obtain significant results for the RSA reactivity analyses. Of note, analyses were only conducted using five-minute RSA reactivity as this is a more stable measure than two-minute RSA reactivity. Results indicated that the required effect size was $f = .29$ for the factorial ANOVAs and $f = .32$ for the repeated measures ANOVA. Results suggested that given the current study's sample size and α level, at least a medium effect size was required to obtain significant results if a power of .80 was achieved.

PEP – two-minutes. Analyses conducted for cardiovascular reactivity using two-minute averages of PEP are reviewed below.

2 X 2 factorial ANOVA – difference score. Education level ($p = .01$) was significantly associated with two-minute PEP reactivity and was included in the final model. The remaining covariates that were under consideration were not significantly associated with cardiovascular reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,90) = 1.02$, $p = .39$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,89) = .005$, $p = .94$, $\eta^2 < .001$) or condition ($F(1,89) = .85$, $p = .36$, $\eta^2 = .009$) nor the group by condition interaction ($F(1,89) = .003$, $p = .96$, $\eta^2 < .001$) were significant, indicating that there was no significant difference in two-minute PEP reactivity between groups or conditions or the group by condition interaction when using difference scores. Contrast analyses revealed that there was no significant difference in two-minute PEP reactivity ($F(3,89) = .35$, $p = .79$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 factorial ANOVA – residualized change score. Education level (p = .04) was significantly associated with two-minute PEP reactivity and was included in the final model. The remaining covariates that were under consideration were not significantly associated with two-minute PEP reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,90) = .91$, $p = .44$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,89) = .004$, $p = .95$, $\eta^2 < .001$) or condition ($F(1,89) = .78$, $p = .38$, $\eta^2 = .009$) nor the group by condition interaction ($F(1,89) = .02$, $p = .89$, $\eta^2 < .001$) were significant, indicating that there was no significant difference in two-minute PEP reactivity between mood induction conditions or the group by condition interaction when using residualized change scores. Contrast analyses revealed that there was no significant difference in two-minute PEP reactivity ($F(3,89) = .35$, $p = .79$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 repeated measures ANOVA. Age (p = .05), race (p = .05), and current state anxiety symptoms (p = .02) were significantly associated with two-minute PEP reactivity and were included in the final model. The remaining covariates that were under consideration were not significantly associated with two-minute PEP reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant (PEP_{BL2}: $F(3,80) = .71$, $p = .55$; PEP_{MI2}: $F(3,80) = .63$, $p = .60$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,77) = .21$, $p = .65$, $\eta^2 = .003$) or condition ($F(1,77) = .81$, $p = .37$, $\eta^2 = .01$) nor the group by condition interaction ($F(1,77) = .29$, $p = .59$, $\eta^2 = .004$) were significant, indicating that there was no significant difference in two-

minute PEP reactivity between groups or conditions or the group by condition interaction when using repeated measures. Contrast analyses revealed that there was no significant difference in two-minute PEP reactivity ($F(1,79) = .17$, $p = .68$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

PEP – five-minutes. Analyses conducted for cardiovascular reactivity using five-minute averages of PEP are reviewed below.

2 X 2 factorial ANOVA – difference score. Race ($p = .004$) and education level ($p = .004$) were significantly associated with five-minute PEP reactivity and were included in the final model. The remaining covariates that were under consideration were not significantly associated with five-minute PEP reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,89) = .29$, $p = .83$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,87) = .03$, $p = .86$, $\eta^2 < .001$) or condition ($F(1,87) = .64$, $p = .43$, $\eta^2 = .007$) nor the group by condition interaction ($F(1,87) = .69$, $p = .41$, $\eta^2 = .008$) were significant, indicating that there was no significant difference in five-minute PEP reactivity between groups or conditions or the group by condition interaction when using difference scores. Contrast analyses revealed that there was no significant difference in two-minute PEP reactivity ($F(3,87) = .33$, $p = .80$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 factorial ANOVA – residualized change score. Education level (p = .008) was significantly associated with five-minute PEP reactivity and was included in the final model. The remaining covariates that were under consideration were not significantly associated with five-minute PEP reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,90) = .41$, $p = .75$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,89) = .05$, $p = .82$, $\eta^2 = .001$) or condition ($F(1,89) = .79$, $p = .38$, $\eta^2 = .009$) nor the group by condition interaction ($F(1,89) = .30$, $p = .59$, $\eta^2 = .003$) were significant, indicating that there was no significant difference in five-minute PEP reactivity between mood induction conditions or the group by condition interaction when using residualized change scores. Contrast analyses revealed that there was no significant difference in five-minute PEP reactivity ($F(3,89) = .30$, $p = .82$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 repeated measures ANOVA. Age (p = .04), race (p = .03), and current state anxiety symptoms (p = .02) were significantly associated with five-minute PEP reactivity and were included in the final model. The remaining covariates that were under consideration were not significantly associated with five-minute PEP reactivity (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant (PEP_{BL5}: $F(3,80) = .68$, $p = .57$; PEP_{MI5}: $F(3,80) = .49$, $p = .69$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,77) = .18$, $p = .67$, $\eta^2 = .002$) or condition ($F(1,77) = .54$, $p = .46$, $\eta^2 = .007$) nor the group by condition interaction ($F(1,77) = .35$, $p = .56$, $\eta^2 = .004$) were significant, indicating that there was no significant difference in

five-minute PEP reactivity between groups or conditions or the group by condition interaction when using repeated measures. Contrast analyses revealed that there was no significant difference in two-minute PEP reactivity ($F(1,80) = .05$, $p = .83$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

Post-hoc power analyses. Post-hoc power analyses were conducted using G*Power 3.1.9.2 (Faul et al., 2007) to determine the power achieved by the PEP reactivity analyses. Of note, analyses were only conducted using five-minute PEP reactivity as this is a more stable measure than two-minute PEP reactivity. Results indicated that the current model had a power of .14 for the difference score, .08 for the residualized change score, and .07 for the repeated measures. Results suggested that given the current study's sample size, α level, and observed effect size, there was a 7 to 14% chance of detecting an effect depending on which analytic technique was used.

Sensitivity analyses. Sensitivity analyses were conducted using G*Power 3.1.9.2 (Faul et al., 2007) to determine the minimum effect size needed to obtain significant results for the PEP reactivity analyses. Of note, analyses were only conducted using five-minute PEP reactivity as this is a more stable measure than two-minute PEP reactivity. Results indicated that the required effect size was $f = .29$ for the factorial ANOVAs and $f = .34$ for the repeated measures ANOVA. Results suggested that given the current study's sample size and α level, at least a medium effect size was required to obtain significant results if a power of .80 was achieved.

Hypothesis 4

A series of 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) factorial ANOVAs using difference scores and residualized change scores and a series of 2 (group: formerly depressed, healthy control) X 2 (condition: sad, neutral) repeated measures ANOVAs were used to test the hypothesis that formerly depressed individuals exposed to the sad mood induction would exhibit reduced cardiovascular recovery (i.e., decreased HP, RSA, and CO and increased PEP compared to baseline) during the recovery film compared to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions. Means, standard deviations, *p* values, and effect sizes for session 2 cardiovascular measures by group are presented in Table 20 while means, standard deviations, *p* values, and effect sizes for session 2 cardiovascular measures by group and condition are presented in Table 21. Planned comparisons were conducted using contrast analyses. Means, standard deviations, and *p* values for session 2 mood measures for planned comparisons are presented in Table 22.

HP – two-minutes. Analyses conducted for cardiovascular recovery using two-minute averages of HP are reviewed below.

2 X 2 factorial ANOVA – difference score. None of the covariates that were under consideration were significantly associated with two-minute HP recovery (all *p*'s > .05) and were dropped from the final model. Levene's test for homogeneity of variance was significant ($F(3,91) = 4.23$, $p = .008$), indicating that this assumption underlying ANOVA was not met.

In line with the planned analyses, multiple steps were explored in an attempt to remedy the violation of this assumption. First, residuals of difference scores were calculated and Kolmogorov-Smirnov's and Shapiro-Wilk's tests of normality were conducted. Residuals of

difference scores were normally distributed for the healthy control group (Kolmogorov-Smirnov: $D(65) = .09$, $p = .20$; Shapiro-Wilk: $D(65) = .96$, $p = .06$), formerly depressed participants exposed to the neutral mood induction (Kolmogorov-Smirnov: $D(15) = .13$, $p = .20$; Shapiro-Wilk: $D(15) = .95$, $p = .51$), and healthy control participants exposed to the sad mood induction (Kolmogorov-Smirnov: $D(34) = .08$, $p = .20$; Shapiro-Wilk: $D(34) = .97$, $p = .52$) and for one measure of normality for the formerly depressed group (Kolmogorov-Smirnov: $D(30) = .13$, $p = .18$), sad mood induction condition (Kolmogorov-Smirnov: $D(49) = .11$, $p = .17$), and formerly depressed participants exposed to the sad mood induction (Kolmogorov-Smirnov: $D(15) = .16$, $p = .20$). However, residuals of difference scores were not normally distributed for the remaining groups and conditions (all *p's* < .05). Second, difference scores were transformed using the reciprocal transformation. Levene's test for homogeneity of variance continued to be significant ($F(3,91) = 18.69$, $p < .001$), indicating that this assumption underlying ANOVA was still not met. Consequently, the factorial ANOVA was conducted with the α level decreased from .05 to .01.

With the adjusted *p* value, neither the main effects of group ($F(1,91) = 4.96$, $p = .03$, $\eta^2 = .05$) or condition ($F(1,91) = 4.09$, $p = .05$, $\eta^2 = .04$) nor the group by condition interaction ($F(1,91) = 5.55$, $p = .02$, $\eta^2 = .06$) were significant, indicating that there was no significant difference in two-minute HP recovery between groups or conditions or the group by condition interaction when using difference scores. For the main effects and interaction, it is possible that this is due to lack of power rather than lack of effect. Using contrast analyses that did not assume equal variances, formerly depressed participants exposed to the sad mood induction reported exhibited higher levels of five-minute HP reactivity during the mood induction ($t(15.26) = 1.31$, $p_L = .11$, $p_Q = .04$, $p_C = .53$) compared to healthy control participants exposed to the sad mood

induction and formerly depressed and healthy control participants exposed to the neutral mood induction.

2 X 2 factorial ANOVA – residualized change score. None of the covariates that were under consideration were significantly associated with two-minute HP recovery (all *p*'s > .05) and were dropped from the final model. Levene's test for homogeneity of variance was significant ($F(3,91) = 7.08$, $p < .001$), indicating that this assumption underlying ANOVA was not met.

In line with the planned analyses, multiple steps were explored in an attempt to remedy the violation of this assumption. First, residuals of residualized change scores were calculated and Kolmogorov-Smirnov's and Shapiro-Wilk's tests of normality were conducted. Residuals of residualized change scores were normally distributed for the healthy control group (Kolmogorov-Smirnov: $D(65) = .07$, $p = .20$; Shapiro-Wilk: $D(65) = .99$, $p = .92$), neutral mood induction condition (Kolmogorov-Smirnov: $D(46) = .12$, $p = .10$; Shapiro-Wilk: $D(46) = .97$, $p = .17$), formerly depressed participants exposed to the neutral mood induction (Kolmogorov-Smirnov: $D(15) = .13$, $p = .20$; Shapiro-Wilk: $D(15) = .95$, $p = .51$), and healthy control participants exposed to the sad mood induction (Kolmogorov-Smirnov: $D(34) = .08$, $p = .20$; Shapiro-Wilk: $D(34) = .97$, $p = .52$) and for one measure of normality for the formerly depressed group (Kolmogorov-Smirnov: $D(30) = .13$, $p = .18$), sad mood induction condition (Kolmogorov-Smirnov: $D(49) = .11$, $p = .17$), formerly depressed participants exposed to the sad mood induction (Kolmogorov-Smirnov: $D(15) = .16$, $p = .20$), and healthy control participants exposed to the neutral mood induction (Shapiro-Wilk: $D(31) = .94$, $p = .06$). However, residuals of difference scores were not normally distributed for the remaining groups and conditions (all *p's* < .05). Second, residualized change scores were transformed using the reciprocal

transformation. Levene's test for homogeneity of variance continued to be significant ($F(1,91) = 19.58$, $p < .001$), indicating that this assumption underlying ANOVA was still not met. Consequently, the factorial ANOVA was conducted with the α level decreased from .05 to .01.

With the adjusted p value, neither the main effects of group ($F(1,91) = 4.12$, $p = .05$, $\eta^2 = .04$) or condition ($F(1,91) = .16$, $p = .69$, $\eta^2 = .002$) nor the group by condition interaction ($F(1,91) = 4.58$, $p = .04$, $\eta^2 = .05$) were significant, indicating that there was no significant difference in two-minute HP recovery between groups or conditions or the group by condition interaction when using residualized change scores. For the main effects and interaction, it is possible that this is due to lack of power rather than lack of effect. Using contrast analyses that did not assume equal variances, formerly depressed participants exposed to the sad mood induction reported exhibited higher levels of five-minute HP reactivity during the mood induction ($t(15.24) = 1.44$, $p_L = .05$, $p_Q = .04$, $p_C = .58$) compared to healthy control participants exposed to the sad mood induction and formerly depressed and healthy control participants exposed to the neutral mood induction.

2 X 2 repeated measures ANOVA. BMI ($p = .05$) was significantly associated with two-minute HP recovery and was included in the final model. The remaining covariates that were under consideration were not significantly associated with two-minute HP recovery (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant for the mood induction measure (HP_{BL2}: $F(3,88) = .90$, $p = .45$) but was significant for the recovery measure (HP_{RC2}: $F(3,88) = 2.89$, $p = .04$), indicating that this assumption underlying ANOVA was not met. Of note, this appeared to be driven by the inclusion of the covariate. Without the covariate, Levene's test for homogeneity of variance was not significant (HP_{BL2}: $F(3,91) = 1.02$, $p = .39$; HP_{RC2}: $F(3,91) = 2.58$, $p = .06$). Consequently, calculating the

residual of the mood induction and recovery measures to assess normality or transforming the mood induction and recovery measures to re-assess homogeneity of variance would not have the intended effect. Instead, the factorial ANOVA was conducted with the α level decreased from .05 to .01. Neither the main effects of group ($F(1,87) = .05$, $p = .82$, $\eta^2 = .001$) or condition ($F(1,87) = 1.20$, $p = .28$, $\eta^2 = .01$) nor the group by condition interaction ($F(1,87) = .87$, $p = .35$, $\eta^2 = .01$) were significant, indicating that there was no significant difference in two-minute HP recovery between groups or conditions or the group by condition interaction when using repeated measures. Contrast analyses that did not assume equal variances revealed that there was no significant difference in two-minute HP recovery ($F(1,90) = .86$, $p = .36$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

HP – five-minutes. Analyses conducted for cardiovascular recovery using five-minute averages of HP are reviewed below.

2 X 2 factorial ANOVA – difference score. None of the covariates that were under consideration were significantly associated with five-minute HP recovery (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was significant ($F(3,91) = 3.08$, $p = .03$), indicating that this assumption underlying ANOVA was not met.

In line with the planned analyses, multiple steps were explored in an attempt to remedy the violation of this assumption. First, residuals of difference scores were calculated and Kolmogorov-Smirnov's and Shapiro-Wilk's tests of normality were conducted. Residuals of difference scores were normally distributed for the healthy control group (Kolmogorov-Smirnov: $D(65) = .06$, $p = .20$; Shapiro-Wilk: $D(65) = .97$, $p = .17$), neutral mood induction condition

(Kolmogorov-Smirnov: $D(46) = .10$, $p = .20$; Shapiro-Wilk: $D(46) = .95$, $p = .06$), formerly depressed participants exposed to the sad (Kolmogorov-Smirnov: $D(15) = .15$, $p = .20$; Shapiro-Wilk: $D(15) = .90$, $p = .09$) and neutral (Kolmogorov-Smirnov: $D(15) = .11$, $p = .20$; Shapiro-Wilk: $D(15) = .97$, $p = .91$) mood inductions, and healthy control participants exposed to the sad mood induction (Kolmogorov-Smirnov: $D(34) = .11$, $p = .20$; Shapiro-Wilk: $D(34) = .96$, $p = .22$) and for one measure of normality for the formerly depressed group (Kolmogorov-Smirnov: $D(30) = .12$, $p = .20$), sad mood induction condition (Kolmogorov-Smirnov: $D(49) = .11$, $p = .13$), and healthy control participants exposed to the neutral mood induction (Kolmogorov-Smirnov: $D(31) = .12$, $p = .20$). However, residuals of difference scores were not normally distributed for the remaining groups and conditions (all *p's* < .05). Second, difference scores were transformed using the reciprocal transformation. Levene's test for homogeneity of variance continued to be significant ($F(3,91) = 7.19$, $p < .001$), indicating that this assumption underlying ANOVA was still not met. Consequently, the factorial ANOVA was conducted with the α level decreased from .05 to .01.

Neither the main effects of group ($F(1,91) = 3.55$, $p = .06$, $\eta^2 = .04$) or condition ($F(1,91) = .65$, $p = .42$, $\eta^2 = .007$) nor the group by condition interaction ($F(1,91) = 3.66$, $p = .06$, $\eta^2 = .04$) were significant, indicating that there was no significant difference in five-minute HP recovery between groups or conditions or the group by condition interaction when using difference scores. For the main effects and interaction, it is possible that this is due to lack of power rather than lack of effect. Using contrast analyses that did not assume equal variances, formerly depressed participants exposed to the sad mood induction reported exhibited higher levels of five-minute HP reactivity during the mood induction ($t(15.87) = 1.61$, $p_L = .05$, $p_Q = .06$, $p_C = .90$) compared

to healthy control participants exposed to the sad mood induction and formerly depressed and healthy control participants exposed to the neutral mood induction.

2 X 2 factorial ANOVA – residualized change score. None of the covariates that were under consideration were significantly associated with five-minute HP recovery (all *p*'s > .05) and were dropped from the final model. Levene's test for homogeneity of variance was significant ($F(3,91) = 6.92$, $p < .001$), indicating that this assumption underlying ANOVA was not met.

In line with the planned analyses, multiple steps were explored in an attempt to remedy the violation of this assumption. First, residuals of difference scores were calculated and Kolmogorov-Smirnov's and Shapiro-Wilk's tests of normality were conducted. Residuals of difference scores were normally distributed for the healthy control group (Kolmogorov-Smirnov: $D(65) = .07$, $p = .20$; Shapiro-Wilk: $D(65) = .97$, $p = .15$), neutral mood induction condition (Kolmogorov-Smirnov: $D(46) = .09$, $p = .20$; Shapiro-Wilk: $D(46) = .98$, $p = .75$), formerly depressed participants exposed to the sad (Kolmogorov-Smirnov: $D(15) = .15$, $p = .20$; Shapiro-Wilk: $D(15) = .90$, $p = .09$) and neutral (Kolmogorov-Smirnov: $D(15) = .11$, $p = .20$; Shapiro-Wilk: $D(15) = .97$, $p = .91$) mood inductions, and healthy control participants exposed to the sad (Kolmogorov-Smirnov: $D(34) = .11$, $p = .20$; Shapiro-Wilk: $D(34) = .96$, $p = .22$) and neutral (Kolmogorov-Smirnov: $D(31) = .10$, $p = .20$; Shapiro-Wilk: $D(31) = .98$, $p = .75$) mood induction and for one measure of normality for the formerly depressed group (Kolmogorov-Smirnov: $D(30) = .12$, $p = .20$) and sad mood induction condition (Kolmogorov-Smirnov: $D(49) = .11$, $p = .13$). However, residuals of difference scores were not normally distributed for the remaining groups and conditions (all *p's* < .05). Second, difference scores were transformed using the reciprocal transformation. Levene's test for homogeneity of variance continued to be

significant ($F(3,91) = 13.14$, $p < .001$), indicating that this assumption underlying ANOVA was still not met. Consequently, the factorial ANOVA was conducted with the α level decreased from .05 to .01.

With the adjusted p value, neither the main effects of group ($F(1,91) = 4.91$, $p = .03$, $\eta^2 = .05$) or condition ($F(1,91) = 1.24$, $p = .27$, $\eta^2 = .01$) nor the group by condition interaction ($F(1,91) = 3.86$, $p = .05$, $\eta^2 = .04$) were significant, indicating that there was no significant difference in five-minute HP recovery between groups or conditions or the group by condition interaction when using residualized change scores. For the main effects and interaction, it is possible that this is due to lack of power rather than lack of effect. Using contrast analyses that did not assume equal variances, formerly depressed participants exposed to the sad mood induction reported exhibited higher levels of five-minute HP reactivity during the mood induction ($t(15.88) = 1.79$, $p_L = .02$, $p_Q = .05$, $p_C = 1.00$) compared to healthy control participants exposed to the sad mood induction and formerly depressed and healthy control participants exposed to the neutral mood induction.

2 X 2 repeated measures ANOVA. BMI ($p = .05$) was significantly associated with five-minute HP recovery and was included in the final model. The remaining covariates that were under consideration were not significantly associated with five-minute HP recovery (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant (HP_{BL5}: $F(3,88) = 1.22$, $p = .31$; HP_{RC5}: $F(3,88) = 2.23$, $p = .09$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,87) = .09$, $p = .77$, $\eta^2 = .001$) or condition ($F(1,87) = 1.71$, $p = .19$, $\eta^2 = .02$) nor the group by condition interaction ($F(1,87) = 1.27$, $p = .26$, $\eta^2 = .01$) were significant, indicating that there was no significant difference in five-minute HP recovery between groups or conditions or the group by

condition interaction when using repeated measures. Contrast analyses revealed that there was no significant difference in five-minute HP recovery ($F(1,90) = 1.36$, $p = .25$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

Post-hoc power analyses. Post-hoc power analyses were conducted using G*Power 3.1.9.2 (Faul et al., 2007) to determine the power achieved by the HP recovery analyses. Of note, analyses were only conducted using five-minute HP recovery as this is a more stable measure than two-minute HP recovery. Results indicated that the current model had a power of .27 for the difference and residualized change scores and .11 for the repeated measures. Results suggested that given the current study's sample size, α level, and observed effect size, there was a 11 to 27% chance of detecting an effect depending on which analytic technique was used.

Sensitivity analyses. Sensitivity analyses were conducted using G*Power 3.1.9.2 (Faul et al., 2007) to determine the minimum effect size needed to obtain significant results for the HP recovery analyses. Of note, analyses were only conducted using five-minute HP recovery as this is a more stable measure than two-minute HP recovery. Results indicated that the required effect size was $f = .36$ for the factorial ANOVAs and $f = .34$ for the repeated measures ANOVA. Results suggested that given the current study's sample size and α level, at least a medium effect size was required to obtain significant results if a power of .80 was achieved.

RSA – two-minutes. Analyses conducted for cardiovascular recovery using two-minute averages of RSA are reviewed below.

2 X 2 factorial ANOVA – difference score. None of the covariates that were under consideration were significantly associated with two-minute RSA recovery (all p's > .05) and

were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,90) = 1.39$, $p = .25$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,90) = .02$, $p = .88$, $\eta^2 < .001$) or condition ($F(1,90) = .85$, $p = .36$, $\eta^2 = .009$) nor the group by condition interaction ($F(1,90) = 2.17$, $p = .14$, $\eta^2 = .02$) were significant, indicating that there was no significant difference in two-minute RSA recovery between groups or conditions or the group by condition interaction when using difference scores. Contrast analyses revealed that there was no significant difference in two-minute RSA recovery ($t(90) = 1.32$, $p_L = .59$, $p_Q = .14$, $p_C = .45$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 factorial ANOVA – residualized change score. None of the covariates that were under consideration were significantly associated with two-minute RSA recovery (all p's $> .05$) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,90) = 1.93$, $p = .13$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,90) = .06$, $p = .81$, $\eta^2 = .001$) or condition ($F(1,90) = 1.08$, $p = .30$, $\eta^2 = .01$) nor the group by condition interaction ($F(1,90) = 1.05$, $p = .31$, $\eta^2 = .01$) were significant, indicating that there was no significant difference in two-minute RSA recovery between groups or conditions or the group by condition interaction when using residualized change scores. Contrast analyses revealed that there was no significant difference in two-minute RSA recovery ($t(90) = .94$, $p_L = .81$, $p_Q = .31$, $p_C = .30$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 repeated measures ANOVA. None of the covariates that were under consideration were significantly associated with two-minute RSA recovery (all *p*'s > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant (RSA_{BL2}: $F(3,90) = 1.09$, $p = .36$; RSA_{RC2}: $F(3,90) = 2.54$, $p = .06$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,90) = .99$, $p = .32$, $\eta^2 = .01$) or condition ($F(1,90) = .20$, $p = .66$, $\eta^2 = .002$) nor the group by condition interaction ($F(1,90) = .90$, $p = .35$, $\eta^2 = .01$) were significant, indicating that there was no significant difference in two-minute RSA recovery between groups or conditions or the group by condition interaction when using repeated measures. Contrast analyses revealed that there was no significant difference in two-minute RSA recovery ($F(1,92) = .46$, $p = .50$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

RSA – five-minutes. Analyses conducted for cardiovascular recovery using five-minute averages of RSA are reviewed below.

2 X 2 factorial ANOVA – difference score. None of the covariates that were under consideration were significantly associated with five-minute RSA recovery (all *p*'s > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,90) = 2.37$, $p = .08$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,90) = .06$, $p = .81$, $\eta^2 = .001$) or condition ($F(1,90) = .24$, $p = .63$, $\eta^2 = .003$) nor the group by condition interaction ($F(1,90) = 1.86$, $p = .18$, $\eta^2 = .02$) were significant, indicating that there was no significant difference in five-minute RSA recovery between groups or conditions or the group by condition interaction when using difference scores.

Contrast analyses revealed that there was no significant difference in two-minute RSA recovery ($t(90) = 1.08$, $p_L = .67$, $p_Q = .18$, $p_C = .74$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 factorial ANOVA – residualized change score. None of the covariates that were under consideration were significantly associated with five-minute RSA recovery (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,90) = 1.67$, $p = .18$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,90) = .001$, $p = .98$, $\eta^2 < .001$) or condition ($F(1,90) = .51$, $p = .48$, $\eta^2 = .006$) nor the group by condition interaction ($F(1,90) = .88$, $p = .35$, $\eta^2 = .01$) were significant, indicating that there was no significant difference in five-minute RSA recovery between mood induction conditions or the group by condition interaction when using residualized change scores. Contrast analyses revealed that there was no significant difference in two-minute RSA recovery ($t(90) = .88$, $p_L = .73$, $p_Q = .35$, $p_C = .53$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 repeated measures ANOVA. None of the covariates that were under consideration were significantly associated with five-minute RSA recovery (all p's > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant (RSA_{BL5}: $F(3,90) = 1.44$, $p = .24$; RSA_{RC5}: $F(3,90) = .73$, $p = .54$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,90) = .45$, $p = .50$, $\eta^2 = .005$) or condition ($F(1,90) = .20$, $p = .66$, $\eta^2 = .002$) nor the group by condition interaction

($F(1,90) = 1.05$, $p = .31$, $\eta^2 = .01$) were significant, indicating that there was no significant difference in five-minute RSA recovery between groups or conditions or the group by condition interaction when using repeated measures. Contrast analyses revealed that there was no significant difference in two-minute RSA recovery ($F(1,92) = .49$, $p = .49$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

Post-hoc power analyses. Post-hoc power analyses were conducted using G*Power 3.1.9.2 (Faul et al., 2007) to determine the power achieved by the RSA recovery analyses. Of note, analyses were only conducted using five-minute RSA recovery as this is a more stable measure than two-minute RSA recovery. Results indicated that the current model had a power of .28 for the difference score, .16 for the residualized change score, and .11 for the repeated measures. Results suggested that given the current study's sample size, α level, and observed effect size, there was a 11 to 28% chance of detecting an effect depending on which analytic technique was used.

Sensitivity analyses. Sensitivity analyses were conducted using G*Power 3.1.9.2 (Faul et al., 2007) to determine the minimum effect size needed to obtain significant results for the RSA recovery analyses. Of note, analyses were only conducted using five-minute RSA recovery as this is a more stable measure than two-minute RSA recovery. Results indicated that the required effect size was $f = .29$ for the factorial ANOVAs and $f = .34$ for the repeated measures ANOVA. Results suggested that given the current study's sample size and α level, at least a medium effect size was required to obtain significant results if a power of .80 was achieved.

PEP – Two-Minute. Analyses conducted for cardiovascular recovery using two-minute averages of PEP are reviewed below.

2 X 2 factorial ANOVA – difference score. None of the covariates that were under consideration were significantly associated with two-minute PEP recovery (all *p*'s > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,90) = 1.83$, $p = .15$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,90) = .006$, $p = .94$, $\eta^2 < .001$) or condition ($F(1,90) = .15$, $p = .70$, $\eta^2 = .002$) nor the group by condition interaction ($F(1,90) = .11$, $p = .75$, $\eta^2 = .001$) were significant, indicating that there was no significant difference in two-minute PEP recovery between groups or conditions or the group by condition interaction when using difference scores. Contrast analyses revealed that there was no significant difference in two-minute PEP recovery ($t(90) = -.01$, $p_L = .92$, $p_Q = .75$, $p_C = .71$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 factorial ANOVA – residualized change score. None of the covariates that were under consideration were significantly associated with two-minute PEP recovery (all *p*'s > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,90) = 2.46$, $p = .07$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,90) = .03$, $p = .86$, $\eta^2 < .001$) or condition ($F(1,90) = .17$, $p = .68$, $\eta^2 = .002$) nor the group by condition interaction ($F(1,90) = .04$, $p = .85$, $\eta^2 < .001$) were significant, indicating that there was no significant difference in two-minute PEP recovery between groups or conditions or the group by condition interaction when using residualized change scores. Contrast analyses revealed that there was no significant difference in two-minute

PEP recovery ($t(90) = .03$, $p_L = .98$, $p_Q = .85$, $p_C = .65$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 repeated measures ANOVA. Current state anxiety symptoms ($p = .01$) were significantly associated with two-minute PEP recovery and were included in the final model. The remaining covariates that were under consideration were not significantly associated with two-minute PEP recovery (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant (PEP_{BL2}: $F(3,81) = .86$, $p = .47$; PEP_{RC2}: $F(3,81) = 1.57$, $p = .20$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,80) = .08$, $p = .78$, $\eta^2 = .001$) or condition ($F(1,80) = .43$, $p = .52$, $\eta^2 = .005$) nor the group by condition interaction ($F(1,80) = .33$, $p = .57$, $\eta^2 = .004$) were significant, indicating that there was no significant difference in two-minute PEP recovery between groups or conditions or the group by condition interaction when using repeated measures. Contrast analyses revealed that there was no significant difference in two-minute PEP recovery ($F(1,82) = .03$, $p = .86$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

PEP – Five-Minute. Analyses conducted for cardiovascular recovery using five-minute averages of PEP are reviewed below.

2 X 2 factorial ANOVA – difference score. Hispanic/Latino ethnicity ($p = .03$) was significantly associated with five-minute PEP recovery and was included in the final model. The remaining covariates that were under consideration were not significantly associated with five-

minute PEP recovery (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,89) = 1.46$, $p = .23$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,88) = .65$, $p = .42$, $\eta^2 = .007$) or condition ($F(1,88) = .08$, $p = .78$, $\eta^2 = .001$) nor the group by condition interaction ($F(1,88) = .24$, $p = .62$, $\eta^2 = .003$) were significant, indicating that there was no significant difference in five-minute PEP recovery between groups or conditions or the group by condition interaction when using difference scores. Contrast analyses revealed that there was no significant difference in two-minute PEP recovery ($F(3,88) = .29$, $p = .83$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 factorial ANOVA – residualized change score. None of the covariates that were under consideration were significantly associated with five-minute PEP recovery (all *p*'s > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant ($F(3,90) = 2.39$, $p = .07$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,90) = .79$, $p = .38$, $\eta^2 = .009$) or condition ($F(1,90) = .01$, $p = .94$, $\eta^2 < .001$) nor the group by condition interaction ($F(1,90) = .22$, $p = .64$, $\eta^2 = .002$) were significant, indicating that there was no significant difference in five-minute PEP recovery between groups or conditions or the group by condition interaction when using residualized change scores. Contrast analyses revealed that there was no significant difference in two-minute PEP recovery ($t(90) = -.26$, $p_L = .41$, $p_Q = .64$, $p_C = .74$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the

neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

2 X 2 repeated measures ANOVA. Current state anxiety symptoms (p = .02) were significantly associated with five-minute PEP recovery and were included in the final model. The remaining covariates that were under consideration were not significantly associated with five-minute PEP recovery (all *p's* > .05) and were dropped from the final model. Levene's test for homogeneity of variance was not significant (PEP_{BL5}: $F(3,81) = .97$, $p = .41$; PEP_{RC5}: $F(3,81) = .57$, $p = .64$), indicating that this assumption underlying ANOVA was met. Neither the main effects of group ($F(1,80) = .01$, $p = .91$, $\eta^2 < .001$) or condition ($F(1,80) = .76$, $p = .39$, $\eta^2 = .009$) nor the group by condition interaction ($F(1,80) = .14$, $p = .71$, $\eta^2 = .002$) were significant, indicating that there was no significant difference in five-minute PEP recovery between groups or conditions or the group by condition interaction when using repeated measures. Contrast analyses revealed that there was no significant difference in two-minute PEP recovery ($F(1,82) = .07$, $p = .79$) when comparing formerly depressed participants exposed to the sad mood induction to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

Post-hoc power analyses. Post-hoc power analyses were conducted using G*Power 3.1.9.2 (Faul et al., 2007) to determine the power achieved by the PEP recovery analyses. Of note, analyses were only conducted using five-minute PEP recovery as this is a more stable measure than two-minute PEP recovery. Results indicated that the current model had a power of .08 for the difference score, .07 for the residualized change score, and .06 for the repeated measures. Results suggested that given the current study's sample size, α level, and observed

effect size, there was a 6 to 8% chance of detecting an effect depending on which analytic technique was used.

Sensitivity analyses. Sensitivity analyses were conducted using G*Power 3.1.9.2 (Faul et al., 2007) to determine the minimum effect size needed to obtain significant results for the PEP recovery analyses. Of note, analyses were only conducted using five-minute PEP recovery as this is a more stable measure than two-minute PEP recovery. Results indicated that the required effect size was $f = .29$ for the factorial ANOVAs and $f = .34$ for the repeated measures ANOVA. Results suggested that given the current study's sample size and α level, at least a medium effect size was required to obtain significant results if a power of .80 was achieved.

Table 20. *Means, Standard Deviations, P Values, and Effect Sizes for Session 2 Cardiovascular Measures by Group*

			Group					
	Total (N = 96)		FD (n = 31)		HC (n = 65)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
HP_{BL2}	781.45	104.55	788.61	115.45	778.03	99.71	.37	.009
HP_{BL5}	785.51	105.88	795.20	118.17	780.89	100.14	.37	.009
HP_{MI2}	799.21	111.48	815.35	120.99	791.51	106.77		
HP_{MI5}	788.89	108.15	809.20	119.87	779.20	101.64		
	Total (N = 95)		FD (n = 30)		HC (n = 65)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
HP_{RC2}	783.98	102.44	800.54	119.94	776.33	93.30		
HP_{RC5}	787.42	103.62	804.24	121.00	779.65	94.56		
	Total (N = 96)		FD (n = 31)		HC (n = 65)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
HP_{DS2RA}	17.76	39.29	26.74	42.78	13.48	37.10	.12	.03
$Z_{RES2}HP_{RA}$	.00	1.00	.24	1.09	-.10	.94	.12	.03
HP_{DS5RA}	3.38	33.73	14.00	39.80	-1.69	29.43	.05	.04
$Z_{RES5}HP_{RA}$	.00	1.00	.34	1.18	-.14	.88	.04	.05
HP_{RARM2}							.79	.001
HP_{RARM5}							.97	< .001
	Total (N = 95)		FD (n = 30)		HC (n = 65)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
HP_{DS2RC}	4.23	49.12	17.09	62.74	-1.70	40.59	.03[b]	.05
$Z_{RES2}HP_{RC}$	.07	.78	.29	1.32	-.14	.75	.05[b]	.04
HP_{DS5RC}	3.70	37.99	14.41	48.55	-1.24	31.19	.06	.04
$Z_{RES5}HP_{RC}$	.03	.85	.31	1.32	-.16	.74	.03[b]	.05
HP_{RCRM2}							.82	.001
HP_{RCRM5}							.77	.001
	Total (N = 95)		FD (n = 31)		HC (n = 64)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
RSA_{BL2}	5.84	1.04	5.68	1.13	5.91	.99	.50	.005
RSA_{BL5}	5.83	1.01	5.71	1.11	5.89	.96	.38	.008
RSA_{MI2}	6.03	1.15	6.02	1.09	6.03	1.18		
RSA_{MI5}	5.89	1.13	5.90	1.07	5.88	1.16		
	Total (N = 94)		FD (n = 30)		HC (n = 64)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
RSA_{RC2}	5.82	1.00	5.69	1.11	5.89	.95		
RSA_{RC5}	5.79	.93	5.71	1.00	5.83	.90		

Table 20 Continued

	Total (N = 95)		FD (n = 31)		HC (n = 64)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
RSA_{DS2RA}	.24	.61	.34	.65	.20	.59	.12	.03
$Z_{RES2}RSA_{RA}$	.02	.73	.13	.74	.02	.73	.50	.005
RSA_{DS5RA}	.11	.45	.19	.52	.07	.41	.23	.02
$Z_{RES5}RSA_{RA}$	.03	.58	.15	.66	.03	.58	.35	.009
RSA_{RARM2}							.56	.004
RSA_{RARM5}							.70	.002
	Total (N = 94)		FD (n = 30)		HC (n = 64)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
RSA_{DS2RC}	-.02	.70	-.004	.79	-.03	.66	.88	< .001
$Z_{RES2}RSA_{RC}$	.00	1.00	-.03	1.12	.03	.94	.81	.001
RSA_{DS5RC}	-.05	.43	-.04	.41	-.06	.44	.81	.001
$Z_{RES5}RSA_{RC}$	.00	1.00	.005	.91	-.002	1.04	.98	< .001
RSA_{RCRM2}							.32	.01
RSA_{RCRM5}							.50	.005
	Total (N = 95)		FD (n = 31)		HC (n = 64)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
PEP_{BL2}	120.61	11.90	121.05	11.10	120.40	12.34	.16	.02
PEP_{BL5}	120.37	11.68	120.70	11.13	120.21	12.02	.21	.02
PEP_{MI2}	119.77	12.74	120.73	11.01	119.31	13.56		
PEP_{MI5}	119.91	13.04	121.15	11.41	119.31	13.81		
	Total (N = 94)		FD (n = 30)		HC (n = 64)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
PEP_{RC2}	120.15	14.33	119.43	15.34	120.48	13.95		
PEP_{RC5}	120.91	12.24	120.51	10.72	121.09	12.96		
	Total (N = 95)		FD (n = 31)		HC (n = 64)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
PEP_{DS2RA}	-.48	4.19	-.32	4.24	-.56	4.20	.94	< .001
$Z_{RES2}PEP_{RA}$	-.01	.65	.10	.76	.05	.76	.95	< .001
PEP_{DS5RA}	-.31	4.13	.13	3.61	-.52	4.37	.86	< .001
$Z_{RES5}PEP_{RA}$	.04	.64	.11	.67	-.02	.81	.82	.001
PEP_{RARM2}							.65	.003
PEP_{RARM5}							.67	.002
	Total (N = 94)		FD (n = 30)		HC (n = 64)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
PEP_{DS2RC}	.68	5.06	.60	3.41	.71	5.70	.94	< .001
$Z_{RES2}PEP_{RC}$	.09	.53	.07	.39	.09	.59	.86	< .001
PEP_{DS5RC}	.79	4.57	.12	3.94	1.10	4.84	.42	.007
$Z_{RES5}PEP_{RC}$	.00	1.00	-.11	.73	.05	.92	.38	.009
PEP_{RCRM2}							.78	.001
PEP_{RCRM5}							.91	< .001

Note. The difference between baseline and mood induction or mood induction and recovery measures may not precisely equal the difference score as outlier data was addressed differently depending on the type of analyses conducted; FD = formerly depressed; HC = healthy control; HP = heart period at baseline; $_{BL}$ = baseline; $_{MI}$ = mood induction; $_{RC}$ = recovery; $_{2}$ = average obtained during a two-minute interval; $_{5}$ = average obtained during a five-minute interval; $_{DS}$ = difference score; $_{RA}$ = reactivity; Z_{RES} = residualized change scores; RSA = respiratory sinus arrhythmia; PEP = pre-ejection period; $_{RM}$ = repeated measures; ^{b} = only linear results are presented as analyses were run with covariates.

Table 21. *Means, Standard Deviations, P Values, and Effect Sizes for Session 2 Cardiovascular Measures by Group and Condition*

			Group and Condition									
	Total (N = 96)		FD/SMI (n = 15)		FD/NMI (n = 16)		HC/SMI (n = 34)		HC/NMI (n = 31)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*		
HP_{BL2}	781.45	104.55	787.33	131.66	789.82	102.36	784.73	87.35	770.69	112.74		
HP_{BL5}	785.51	105.88	798.91	134.77	791.72	104.65	785.17	87.23	776.20	113.92		
HP_{MI2}	799.21	111.48	820.97	129.38	810.08	116.58	800.55	98.33	781.59	116.15		
HP_{MI5}	788.89	108.15	823.91	130.36	795.40	111.60	784.78	91.44	773.08	112.99		
	Total (N = 95)		FD/SMI (n = 15)		FD/NMI (n = 15)		HC/SMI (n = 34)		HC/NMI (n = 31)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	SD	*M*	*SD*		
HP_{RC2}	783.98	102.44	816.44	135.64	784.64	104.21	773.14	83.03	779.83	104.71		
HP_{RC5}	787.42	103.62	824.47	133.33	784.01	108.06	779.60	85.45	779.71	105.08		
	Total (N = 96)		FD/SMI (n = 15)		FD/NMI (n = 16)		HC/SMI (n = 34)		HC/NMI (n = 31)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
HP_{DS2RA}	17.76	39.29	33.64	52.14	20.27	32.09	15.83	38.57	10.90	35.87	.62	.003
$Z_{RES2}HP_{RA}$	.00	1.00	.42	1.33	.08	.81	-.04	.98	-.16	.91	.62	.003
HP_{DS5RA}	3.38	33.73	25.00	44.83	3.69	32.53	-.39	30.55	-3.12	28.57	.27	.01
$Z_{RES5}HP_{RA}$	.00	1.00	.67	1.31	.03	.98	-.10	.91	-.19	.84	.27	.01
HP_{RARM2}											.44	.007
HP_{RARM5}											.24	.02
	Total (N = 95)		FD/SMI (n = 15)		FD/NMI (n = 15)		HC/SMI (n = 34)		HC/NMI (n = 31)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	SD	*M*	*SD*	*p*	η^2
HP_{DS2RC}	4.23	49.12	29.11	81.49	5.07	34.67	-11.58	39.91	9.14	39.12	.02[b]	.06
$Z_{RES2}HP_{RC}$	.07	.78	.56	1.68	.02	.79	-.32	.80	.05	.64	.04[b]	.05
HP_{DS5RC}	3.70	37.99	25.56	58.82	3.26	34.00	-5.57	33.63	3.51	28.04	.06	.04
$Z_{RES5}HP_{RC}$	.03	.85	.63	1.56	-.01	.98	-.24	.89	-.07	.55	.05[b]	.04
HP_{RCRM2}											.35	.01
HP_{RCRM5}											.26	.01

Table 21 Continued

	Total (N = 95)		FD/SMI (n = 15)		FD/NMI (n = 16)		HC/SMI (n = 32)		HC/NMI (n = 32)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*		
RSA_{BL2}	5.83	1.04	5.54	1.07	5.81	1.21	6.08	1.02	5.74	.94		
RSA_{BL5}	5.83	1.01	5.63	1.06	5.79	1.19	6.06	.97	5.71	.93		
RSA_{MI2}	6.03	1.15	5.92	1.02	6.11	1.19	6.32	1.04	5.75	1.25		
RSA_{MI5}	5.89	1.13	5.84	1.00	5.96	1.16	6.20	1.04	5.56	1.21		
	Total (N = 94)		FD/SMI (n = 15)		FD/NMI (n = 15)		HC/SMI (n = 32)		HC/NMI (n = 32)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*		
RSA_{RC2}	5.82	1.00	5.73	.86	5.65	1.34	6.01	.99	5.76	.91		
RSA_{RC5}	5.79	.93	5.70	.92	5.72	1.10	5.97	.94	5.70	.86		
	Total (N = 95)		FD/SMI (n = 15)		FD/NMI (n = 16)		HC/SMI (n = 32)		HC/NMI (n = 32)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
RSA_{DS2RA}	.24	.61	.37	.53	.30	.76	.24	.61	.16	.57	.55	.004
$Z_{RES2}RSA_{RA}$	.02	.73	.14	.61	.13	.86	.13	.71	-.09	.74	.47	.006
RSA_{DS5RA}	.11	.45	.22	.37	.17	.64	.14	.40	.01	.42	.71	.002
$Z_{RES5}RSA_{RA}$	.03	.58	.17	.47	.14	.81	.16	.57	-.10	.57	.41	.007
RSA_{RARM2}											.12	.03
RSA_{RARM5}											.15	.02
	Total (N = 94)		FD/SMI (n = 15)		FD/NMI (n = 15)		HC/SMI (n = 32)		HC/NMI (n = 32)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
RSA_{DS2RC}	-.02	.70	.18	.74	-.19	.82	-.07	.78	.02	.53	.14	.02
$Z_{RES2}RSA_{RC}$	.00	1.00	.20	.94	-.26	1.26	.03	1.10	.03	.78	.31	.01
RSA_{DS5RC}	-.05	.43	.05	.35	-.12	.46	-.10	.53	-.02	.34	.18	.02
$Z_{RES5}RSA_{RC}$	.00	1.00	.19	.79	-.18	1.00	-.03	1.19	.02	.88	.35	.01
RSA_{RCRM2}											.35	.01
RSA_{RCRM5}											.31	.01
	Total (N = 95)		FD/SMI (n = 15)		FD/NMI (n = 16)		HC/SMI (n = 32)		HC/NMI (n = 32)			
Measure	*M*		*SD*		*M*		*SD*		*M*			
PEP_{BL2}	120.61		11.90		118.97		11.36		123.00			
PEP_{BL5}	120.37		11.68		118.99		11.06		122.30			
PEP_{MI2}	119.77		12.74		118.73		10.52		122.59			
PEP_{MI5}	*119.91*		*13.04*		*119.29*		*10.98*		*122.89*			

Table 21 Continued

	Total (N = 94)		FD/SMI (n = 15)		FD/NMI (n = 15)		HC/SMI (n = 32)		HC/NMI (n = 32)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*		
PEP_{RC2}	120.15	14.33	120.47	10.68	118.40	19.26	120.66	12.65	120.28	15.51		
PEP_{RC5}	120.91	12.24	120.12	10.35	120.91	11.42	120.27	12.78	122.03	13.33		
	Total (N = 95)		FD/SMI (n = 15)		FD/NMI (n = 16)		HC/SMI (n = 32)		HC/NMI (n = 32)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
PEP_{DS2RA}	-.48	4.19	-.23	4.69	-.41	3.92	-.04	4.21	-1.13	4.19	.96	< .001
$Z_{RES2}PEP_{RA}$	-.01	.65	.10	.83	.09	.71	.15	.74	-.06	.77	.89	< .001
PEP_{DS5RA}	-.31	4.13	.31	3.73	-.04	3.62	-.31	4.95	-.77	3.67	.41	.008
$Z_{RES5}PEP_{RA}$	.04	.64	.15	.70	.07	.67	.03	.91	-.07	.70	.59	.003
PEP_{RARM2}											.59	.004
PEP_{RARM5}											.56	.004
	Total (N = 94)		FD/SMI (n = 15)		FD/NMI (n = 15)		HC/SMI (n = 32)		HC/NMI (n = 32)			
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*p*	η^2
PEP_{DS2RC}	.68	5.06	.63	3.64	.57	3.29	1.09	4.54	.28	6.84	.75	.001
$Z_{RES2}PEP_{RC}$	.09	.53	.08	.37	.06	.42	.13	.43	.06	.74	.85	< .001
PEP_{DS5RC}	.79	4.57	.35	2.95	-.11	4.84	.82	3.69	1.41	5.93	.62	.003
$Z_{RES5}PEP_{RC}$	.00	1.00	-.08	.54	-.15	.89	.003	.65	.11	1.16	.64	.002
PEP_{RARM2}											.57	.004
PEP_{RARM5}											.71	.002

Note. The difference between baseline and mood induction or mood induction and recovery measures may not precisely equal the difference score as outlier data was addressed differently depending on the type of analyses conducted; FD = formerly depressed; HC = healthy control; SMI = sad mood induction; NMI = neutral mood induction; HP = heart period at baseline; $_{BL}$ = baseline; $_{MI}$ = mood induction; $_{RC}$ = recovery; $_{2}$ = average obtained during a two-minute interval; $_{5}$ = average obtained during a five-minute interval; $_{DS}$ = difference score; $_{RA}$ = reactivity; Z_{RES} = residualized change scores; RSA = respiratory sinus arrhythmia; PEP = pre-ejection period; $_{RM}$ = repeated measures; [b] = only linear results are presented as analyses were run with covariates.

Table 22. *Means, Standard Deviations, and P Values for Session 2 Cardiovascular Measures for Planned Comparisons*

			Group and Condition						
	Total (N = 96)		FD/SMI (n = 15)		FD/NMI, HC/SMI, HC/NMI (n = 81)				
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*			
HP_{BL2}	781.45	104.55	787.33	131.66	780.36	99.70			
HP_{BL5}	785.51	105.88	798.91	134.77	783.03	100.47			
HP_{MI2}	799.21	111.48	820.97	129.38	795.18	108.28			
HP_{MI5}	788.89	108.15	823.91	130.36	782.40	103.16			
	Total (N = 95)		FD/SMI (n = 15)		FD/NMI, HC/SMI, HC/NMI (n = 80)				
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*			
HP_{RC2}	783.98	102.44	816.44	135.64	777.89	94.80			
HP_{RC5}	787.42	103.62	824.47	133.33	780.47	96.52			
	Total (N = 96)		FD/SMI (n = 15)		FD/NMI, HC/SMI, HC/NMI (n = 81)				
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	p_L	p_Q	p_C
HP_{DS2RA}	17.76	39.29	33.64	52.14	14.82	36.08	.06	.62	.80
$Z_{RES2}HP_{RA}$	.00	1.00	.42	1.33	-.06	.92	.07	.62	.80
HP_{DS5RA}	3.38	33.73	25.00	44.83	-.63	29.93	.10[b]		
$Z_{RES5}HP_{RA}$	.00	1.00	.67	1.31	-.11	.89	.08[b]		
HP_{RARM2}							.46		
HP_{RARM5}							.26		

Table 22 Continued

	Total (N = 95)		FD/SMI (n = 15)		FD/NMI, HC/SMI, HC/NMI (n = 80)				
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	p_L	p_Q	p_C
HP_{DS2RC}	4.23	49.12	29.11	81.49	-.43	39.43	.11	.04	.53
$Z_{RES2}HP_{RC}$	.07	.78	.56	1.68	-.11	.75	.05	.04	.58
HP_{DS5RC}	3.70	37.99	25.56	58.82	-.40	31.56	.05	.06	.90
$Z_{RES5}HP_{RC}$	.03	.85	.63	1.56	-.13	.79	.02	.05	1.00
HP_{RCRM2}							.36		
HP_{RCRM5}							.25		

	Total (N = 95)		FD/SMI (n = 15)		FD/NMI, HC/SMI, HC/NMI (n = 80)	
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*
RSA_{BL2}	5.84	1.04	5.54	1.07	5.89	1.03
RSA_{BL5}	5.83	1.01	5.63	1.06	5.87	1.00
RSA_{MI2}	6.03	1.15	5.92	1.02	6.05	1.17
RSA_{MI5}	5.89	1.13	5.84	1.00	5.90	1.16

	Total (N = 94)		FD/SMI (n = 15)		FD/NMI, HC/SMI, HC/NMI (n = 79)	
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*
RSA_{RC2}	5.82	1.00	5.73	.86	5.84	1.03
RSA_{RC5}	5.79	.93	5.69	.92	5.81	.94

	Total (N = 95)		FD/SMI (n = 15)		FD/NMI, HC/SMI, HC/NMI (n = 80)				
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	p_L	p_Q	p_C
RSA_{DS2RA}	.24	.61	.37	.53	.22	.62	.40[b]		
$Z_{RES2}RSA_{RA}$	.02	.73	.14	.61	.04	.75	.66[b]		
RSA_{DS5RA}	.11	.45	.22	.37	.09	.46	.15	.71	.78
$Z_{RES5}RSA_{RA}$	.03	.58	.17	.47	.05	.63	.19	.41	.58
RSA_{RARM2}							.40		
RSA_{RARM5}							.60		

Table 22 Continued

	Total (N = 94)		FD/SMI (n = 15)		FD/NMI, HC/SMI, HC/NMI (n = 79)				
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	p_L	p_Q	p_C
RSA_{DS2RC}	-.02	.70	.18	.74	-.06	.69	.59	.14	.45
$Z_{RES2}RSA_{RC}$	.00	1.00	.20	.94	-.03	1.01	.81	.31	.30
RSA_{DS5RC}	-.05	.43	.05	.35	-.07	.44	.67	.18	.74
$Z_{RES5}RSA_{RC}$	.00	1.00	.19	.79	-.04	1.03	.73	.35	.53
RSA_{RCRM2}							.50		
RSA_{RCRM5}							.49		
	Total (N = 95)		FD/SMI (n = 15)		FD/NMI, HC/SMI, HC/NMI (n = 80)				
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*			
PEP_{BL2}	120.61	11.90	118.97	11.37	120.92	12.04			
PEP_{BL5}	120.37	11.68	118.99	11.06	120.63	11.84			
PEP_{MI2}	119.77	12.74	118.73	10.52	119.96	13.17			
PEP_{MI5}	119.91	13.04	119.29	10.98	120.03	13.45			
	Total (N = 94)		FD/SMI (n = 15)		FD/NMI, HC/SMI, HC/NMI (n = 79)				
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*			
PEP_{RC2}	120.15	14.33	120.47	10.68	120.09	14.98			
PEP_{RC5}	120.91	12.24	120.12	10.35	121.06	12.62			
	Total (N = 95)		FD/SMI (n = 15)		FD/NMI, HC/SMI, HC/NMI (n = 80)				
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	p_L	p_Q	p_C
PEP_{DS2RA}	-.48	4.19	-.23	4.69	-.53	4.12	.79[b]		
$Z_{RES2}PEP_{RA}$	-.01	.65	.10	.83	.06	.74	.79[b]		
PEP_{DS5RA}	-.31	4.13	.31	3.73	-.43	4.21	.80[b]		
$Z_{RES5}PEP_{RA}$	.04	.64	.15	.70	.001	.78	.82[b]		
PEP_{RARM2}							.68		
PEP_{RARM5}							.83		

Table 22 Continued

	Total (N = 94)		FD/SMI (n = 15)		FD/NMI, HC/SMI, HC/NMI (n = 79)				
Measure	*M*	*SD*	*M*	*SD*	*M*	*SD*	p_L	p_Q	p_C
PEP_{DS2RC}	.68	5.06	.63	3.64	.68	5.31	.92	.75	.71
$Z_{RES2}PEP_{RC}$	.09	.53	.08	.37	.09	.56	.98	.85	.65
PEP_{DS5RC}	.79	4.57	.35	2.95	.87	4.83	.83[b]		
$Z_{RES5}PEP_{RC}$	.00	1.00	-.08	.54	.01	.91	.41	.64	.74
PEP_{RCRM2}							.86		
PEP_{RCRM5}							.79		

Note. The difference between baseline and mood induction or mood induction and recovery measures may not precisely equal the difference score as outlier data was addressed differently depending on the type of analyses conducted; FD = formerly depressed; HC = healthy control; SMI = sad mood induction; NMI = neutral mood induction; $_L$ = linear; $_Q$ = quadratic; $_C$ = cubic; HP = heart period at baseline; $_{BL}$ = baseline; $_{MI}$ = mood induction; $_{RC}$ = recovery; $_2$ = average obtained during a two-minute interval; $_5$ = average obtained during a five-minute interval; $_{DS}$ = difference score; $_{RA}$ = reactivity; Z_{RES} = residualized change scores; RSA = respiratory sinus arrhythmia; PEP = pre-ejection period; $_{RM}$ = repeated measures; [b] = only linear results are presented as analyses were run with covariates.

Bivariate Correlations. Bivariate correlations were used to examine correlations for cognitive, mood, and cardiovascular measures. Of note, only relevant variables were analyzed. Only five-minute averages for cardiovascular measures are presented as they are more a reliable measure of cardiovascular functioning than two-minute averages (S. K. McCoy, personal communication, June 14, 2019). A correlation matrix for cognitive, mood, and cardiovascular pre- and post-mood induction scores for the entire sample is presented in Table 23, correlation matrices for cognitive, mood, and cardiovascular pre- and post-mood induction scores by group are presented in Table 24, and correlation matrices for cognitive, mood, and cardiovascular pre- and post-mood induction scores by group and condition are presented in Table 25. Correlation matrices for cognitive, mood, and cardiovascular change scores for the entire sample are presented in Table 26, correlation matrices for cognitive, mood, and cardiovascular change scores by group are presented in Table 27, and correlation matrices for cognitive, mood, and cardiovascular change scores by group and condition are presented in Table 28.

Cognitive and mood measures. Bivariate correlations were used to investigate the relationship between cognitive and mood measures pre- and post-mood induction. Correlations between pre- and post-mood induction scores for the entire sample were examined. Pre- and post-mood induction measures were positively correlated for the DAS-SF I & II, VAS_{PRE} and VAS_{POST}, PANAS-X N_{PRE} and PANAS-X N_{POST}, PANAS-X P_{PRE} and PANAS-X P_{POST}, PANAS-X G_{PRE} and PANAS-X G_{POST}, and PANAS-X S_{PRE} and PANAS-X S_{POST} (r = .36-.91, $p \leq .001$ for all). These results suggest that pre- and post-mood induction scores for cognitive and mood measures all correlate with one another as expected.

Correlations between pre- and post-mood induction, difference, and residualized change scores for the entire sample were examined by measure. The DAS I was negatively correlated

with the VAS_{PRE}, PANAS-X N_{PRE}, PANAS-X N_{POST}, PANAS-X G_{PRE}, PANAS-X G_{POST}, PANAS-X S_{PRE}, and PANAS-X S_{POST} (r = -.31--.47, $p \leq .001$ for all). The DAS II was positively correlated with the PANAS-X P_{POST} (r = .27, $p \leq .01$) and negatively correlated with the VAS_{PRE}, VAS_{POST}, PANAS-X N_{PRE}, PANAS-X N_{POST}, PANAS-X G_{PRE}, PANAS-X G_{POST}, PANAS-X S_{PRE}, and PANAS-X S_{POST} (r = -.24--.46, $p \leq .01$-.001). Some of these correlations were also obtained when using difference and residualized change scores. The DAS_{DS} and Z_{RES}DAS was positively correlated with the PANAS-X P_{DS} and Z_{RES}PANAS-X P (r = .26-.29, $p \leq .01$-.001) and negatively correlated with the VAS_{DS} and Z_{RES}VAS (r = -.20--.23, $p \leq .05$ for all). Of note, the valences of these correlations are in the expected direction as lower scores on the DAS are indicative of increased dysfunctional beliefs. Therefore, these results suggest that increased dysfunctional thoughts pre- and post-mood induction were associated with increased dysphoric mood, negative affect, guilt, and sadness pre- and post-mood induction while increased dysfunctional thoughts post-mood induction were also associated with decreased positive affect post-mood induction. In addition, these results indicate that increased dysfunctional thoughts during the experimental paradigm was associated with increased dysphoric mood and decreased positive affect post-mood induction.

The VAS_{PRE} was positively correlated with the PANAS-X N_{PRE}, PANAS-X N_{POST}, PANAS-X G_{PRE}, PANAS-X G_{POST}, PANAS-X S_{PRE}, and PANAS-X S_{POST} (r = .33-.41, $p \leq .001$ for all). The VAS_{POST} was positively correlated with the PANAS-X N_{POST} and PANAS-X S_{POST} (r = .22-.39, $p \leq .05$-.001) and negatively correlated with the PANAS-X P_{POST} (r = -.27, $p \leq .01$). Correlations were also obtained between the VAS and PANAS-X subscales when using difference and residualized change scores. The VAS_{DS} and Z_{RES}VAS was positively correlated with the PANAS-X N_{DS}, G_{DS}, and S_{DS} and Z_{RES}PANAS-X N, G, and S (r = .24-.52, $p \leq .01$-.001)

and negatively correlated with the PANAS-X P_{DS} and Z_{RES}PANAS-X P (r = -.36--.38, $p \leq .001$ for all). These results suggest that increased dysphoric mood pre-mood induction was associated with increased negative affect, guilt, and sadness pre- and post-mood induction while increased dysphoric mood post-mood induction was associated with increased negative affect and sadness and decreased positive affect post-mood induction. In addition, these results indicate that increased dysphoric mood during the experimental paradigm was associated with increased negative affect, guilt, and sadness and decreased positive affect post-mood induction.

The PANAS-X N_{PRE} was positively correlated with the PANAS-X G_{PRE}, PANAS-X G_{POST}, PANAS-X S_{PRE}, and PANAS-X S_{POST} (r =.67-.87, $p \leq .001$ for all). The PANAS-X N_{POST} was positively correlated with the PANAS-X G_{PRE}, PANAS-X G_{POST}, PANAS-X S_{PRE}, and PANAS-X S_{POST} (r =.73-.84, $p \leq .001$ for all). These correlations were also obtained when using difference and residualized change scores. The PANAS-X N_{DS} and Z_{RES}PANAS-X N was positively correlated with the PANAS-X G_{DS}, Z_{RES}PANAS-X G, PANAS-X S_{DS}, and Z_{RES}PANAS-X S (r = .46-.64, $p \leq .001$ for all). These results suggest that increased negative affect pre- and post-mood induction was associated with increased guilt and sadness pre- and post-mood induction. In addition, these results indicate that increased negative affect during the experimental paradigm was associated with increased guilt and sadness post-mood induction.

While the PANAS-X P_{PRE} and PANAS-X P_{POST} were not associated with other pre- and post-mood induction PANAS-X measures, significant correlations were present when using difference and residualized change scores. The PANAS-X P_{DS} was negatively correlated with the PANAS-X S_{DS} (r = -.38, $p \leq .001$) while the Z_{RES}PANAS-X P was negatively correlated with the Z_{RES}PANAS-X G (r = -.40, $p \leq .001$). Generally, these results indicate that increased positive

affect during the experimental paradigm was associated with decreased guilt and sadness post-mood induction.

The PANAS-X G_{PRE} and PANAS-X G_{POST} was positively correlated with the PANAS-X S_{PRE} and PANAS-X S_{POST} (r =.60-.70, $p \leq .001$ for all). These correlations were also obtained when using difference and residualized change scores. The PANAS-X G_{DS} and Z_{RES}PANAS-X G was positively correlated with the PANAS-X S_{DS} and Z_{RES}PANAS-X S (r = .48-.52, $p \leq .001$ for all). These results suggest that increased guilt pre- and post-mood induction was associated with increased sadness pre- and post-mood induction. In addition, these results indicate that increased guilt during the experimental paradigm was associated with increased sadness post-mood induction.

Correlations between cognitive and mood pre- and post-mood induction, difference, and residualized change scores were evaluated between groups. For formerly depressed participants, most of the pre- and post-mood induction measures were positively correlated, including the DAS-SF I & II, PANAS-X N_{PRE} and PANAS-X N_{POST}, PANAS-X P_{PRE} and PANAS-X P_{POST}, PANAS-X G_{PRE} and PANAS-X G_{POST}, and PANAS-X S_{PRE} and PANAS-X S_{POST} (r = .69-.90, $p \leq .001$ for all). However, the VAS_{PRE} and VAS_{POST} (r = .14, $p > .05$) were not significantly correlated, suggesting that there was a significant difference in dysphoric mood pre- and post-mood induction for formerly depressed participants. The DAS_{DS} and Z_{RES}DAS were not correlated with any of the mood or affect measures. The VAS_{DS} and Z_{RES}VAS were positively correlated with the PANAS-X N_{DS}, G_{DS}, and S_{DS} and Z_{RES}PANAS-X N, G, and S (r = .40-.56, $p \leq .05$-.001) and negatively correlated with the PANAS-X P_{DS} and Z_{RES}PANAS-X P (r = -.52, $p \leq .001$ for all). The PANAS-X N_{DS} and Z_{RES}PANAS-X N were positively correlated with the PANAS-X G_{DS}, Z_{RES}PANAS-X G, PANAS-X S_{DS}, and Z_{RES}PANAS-X S (r = .56-.74, $p \leq .001$

for all). The PANAS-X P_{DS} and Z_{RES}PANAS-X P were negatively correlated with the PANAS-X S_{DS} and Z_{RES}PANAS-X G (r = -.49--.56, $p \leq .001$ for all). Finally, the PANAS-X G_{DS} and Z_{RES}PANAS-X G were positively correlated with the PANAS-X S_{DS} and Z_{RES}PANAS-X S (r = .60-.65, $p \leq .001$ for all). Overall, these correlations show a similar pattern to those previously reported for the entire sample.

For healthy control participants, all pre- and post-mood induction measures were positively correlated, including the DAS-SF I & II, VAS_{PRE} and VAS_{POST}, PANAS-X N_{PRE} and PANAS-X N_{POST}, PANAS-X P_{PRE} and PANAS-X P_{POST}, PANAS-X G_{PRE} and PANAS-X G_{POST}, and PANAS-X S_{PRE} and PANAS-X S_{POST} (r = .46-.91, $p \leq .001$ for all). Importantly, these results suggest that there was no difference in dysphoric mood pre- and post-mood induction for healthy control participants. The DAS_{DS} and Z_{RES}DAS was positively correlated with the PANAS-X P_{DS} and Z_{RES}PANAS-X P (r = .25-.28, $p \leq .05$ for all). The VAS_{DS} and Z_{RES}VAS were positively correlated with the PANAS-X S_{DS} and Z_{RES}PANAS-X G (r = .41-.50, $p \leq .001$ for all) and negatively correlated with the PANAS-X P_{DS} and Z_{RES}PANAS-X P (r = -.29-.32, $p \leq .01$ for all). The PANAS-X N_{DS} and Z_{RES}PANAS-X N were positively correlated with the PANAS-X G_{DS}, Z_{RES}PANAS-X G, PANAS-X S_{DS}, and Z_{RES}PANAS-X S (r = .36-.48, $p \leq .001$ for all). The PANAS-X P_{DS} and Z_{RES}PANAS-X P were negatively correlated with the PANAS-X S_{DS} and Z_{RES}PANAS-X G (r = -.34--.37, $p \leq .01$-.001). Finally, the PANAS-X G_{DS} and Z_{RES}PANAS-X G were positively correlated with the PANAS-X S_{DS} and Z_{RES}PANAS-X S (r = .29-.30, $p \leq .01$ for all). Overall, these correlations show a similar pattern to those previously reported for the entire sample.

Correlations between cognitive and mood pre- and post-mood induction, difference, and residualized change scores were evaluated between groups and conditions. For formerly

depressed participants exposed to the sad mood induction, most of the pre- and post-mood induction measures were positively correlated, including the DAS-SF I & II, PANAS-X N_{PRE} and PANAS-X N_{POST}, PANAS-X P_{PRE} and PANAS-X P_{POST}, PANAS-X G_{PRE} and PANAS-X G_{POST}, and PANAS-X S_{PRE} and PANAS-X S_{POST} (r = .77-.87, $p \leq .001$ for all). However, the VAS_{PRE} and VAS_{POST} (r = .16, $p > .05$) were not significantly correlated, suggesting that there was a significant difference in dysphoric mood pre- and post-mood induction for formerly depressed participants exposed to the sad mood induction. The DAS_{DS} was negatively correlated with the VAS_{DS} (r = -.58, $p \leq .05$) while the Z_{RES}DAS was not correlated with any of the mood or affect measures. The VAS_{DS} and Z_{RES}VAS were positively correlated with the PANAS-X N_{DS}, PANAS-X S_{DS}, Z_{RES}PANAS-X N, and Z_{RES}PANAS-X G (r = .49-.57, $p \leq .05$ for all). The PANAS-X N_{DS} and Z_{RES}PANAS-X N were positively correlated with the PANAS-X G_{DS}, Z_{RES}PANAS-X G, PANAS-X S_{DS}, and Z_{RES}PANAS-X S (r = .52-.78, $p \leq .05$-.001). The PANAS-X P_{DS} and Z_{RES}PANAS-X P were negatively correlated with the PANAS-X S_{DS} and Z_{RES}PANAS-X G (r = -.57--.65, $p \leq .01$-.001). Finally, the PANAS-X G_{DS} and Z_{RES}PANAS-X G were positively correlated with the PANAS-X S_{DS} and Z_{RES}PANAS-X S (r = .59-.65, $p \leq .01$ for all). Overall, these correlations show a similar pattern to those previously reported for the entire sample.

For formerly depressed participants exposed to the neutral mood induction, most of the pre- and post-mood induction measures were positively correlated, including the DAS-SF I & II, PANAS-X N_{PRE} and PANAS-X N_{POST}, PANAS-X P_{PRE} and PANAS-X P_{POST}, PANAS-X G_{PRE} and PANAS-X G_{POST}, and PANAS-X S_{PRE} and PANAS-X S_{POST} (r = .63-.95, $p \leq .001$ for all). However, the VAS_{PRE} and VAS_{POST} (r = .37, $p > .05$) were not significantly correlated, suggesting that there was a significant difference in dysphoric mood pre- and post-mood

induction for formerly depressed participants exposed to the neutral mood induction. Of note, this correlation was stronger than the correlation for formerly depressed participants exposed to the sad mood induction (r = .16, p > .05). The DAS_{DS}, Z_{RES}DAS, VAS_{DS}, Z_{RES}VAS, PANAS-X P_{DS}, Z_{RES}PANAS-X P, PANAS-X G_{DS}, and Z_{RES}PANAS-X G were not correlated with any of the mood or affect measures. The PANAS-X N_{DS} and Z_{RES}PANAS-X N were positively correlated with the PANAS-X G_{DS}, Z_{RES}PANAS-X G, PANAS-X S_{DS}, and Z_{RES}PANAS-X S (r = .62-.72, $p \leq$ .01-.001). Overall, these correlations show a similar pattern to those previously reported for the entire sample with the exception of some of the mood and affective measures.

For healthy control participants exposed to the sad mood induction, all pre- and post-mood induction measures were positively correlated, including the DAS-SF I & II, VAS_{PRE} and VAS_{POST}, PANAS-X N_{PRE} and PANAS-X N_{POST}, PANAS-X P_{PRE} and PANAS-X P_{POST}, PANAS-X G_{PRE} and PANAS-X G_{POST}, and PANAS-X S_{PRE} and PANAS-X S_{POST} (r = .41-.92, $p \leq$ .01-.001). Importantly, these results suggest that there was no difference in dysphoric mood pre- and post-mood induction for healthy control participants exposed to the sad mood induction. The DAS_{DS} and Z_{RES}DAS were not correlated with any of the mood or affect measures. The VAS_{DS} and Z_{RES}VAS were positively correlated with the PANAS-X S_{DS} and Z_{RES}PANAS-X G (r = .40-.51, $p \leq$ .01-.001). The PANAS-X N_{DS} and Z_{RES}PANAS-X N were positively correlated with the PANAS-X G_{DS}, Z_{RES}PANAS-X G, PANAS-X S_{DS}, and Z_{RES}PANAS-X S (r = .42-.53, $p \leq$ .01-.001). The PANAS-X P_{DS} and Z_{RES}PANAS-X P were negatively correlated with the PANAS-X S_{DS} and Z_{RES}PANAS-X G (r = -.40--.41, $p \leq$ .01 for all). Finally, the PANAS-X G_{DS} and Z_{RES}PANAS-X G were positively correlated with the PANAS-X S_{DS} and Z_{RES}PANAS-X S (r = .34, $p \leq$ .01 for all). Overall, these correlations show a similar pattern to those previously reported for the entire sample.

For healthy control participants exposed to the neutral mood induction, all pre- and post-mood induction measures were positively correlated, including the DAS-SF I & II, VAS_{PRE} and VAS_{POST}, PANAS-X N_{PRE} and PANAS-X N_{POST}, PANAS-X P_{PRE} and PANAS-X P_{POST}, PANAS-X G_{PRE} and PANAS-X G_{POST}, and PANAS-X S_{PRE} and PANAS-X S_{POST} (r = .64-.94, $p \leq .001$ for all). Importantly, these results suggest that there was no difference in dysphoric mood pre- and post-mood induction for healthy control participants exposed to the neutral mood induction. The Z_{RES}DAS was positively correlated with Z_{RES}PANAS-X P (r = .36, $p \leq .05$). The Z_{RES}PANAS-X N was positively correlated with the, Z_{RES}PANAS-X G and Z_{RES}PANAS-X S (r = .39-.40, $p \leq .05$ for all). The DAS_{DS}, VAS_{DS}, Z_{RES}VAS, PANAS-X N_{DS}, PANAS-X P_{DS}, Z_{RES}PANAS-X P, PANAS-X G_{DS}, and Z_{RES}PANAS-X G were not correlated with any of the mood or affect measures. Overall, these correlations show a similar pattern to those previously reported for the entire sample with the exception of some of the mood and affective measures.

Cardiovascular measures. Bivariate correlations were used to investigate the relationship between cardiovascular measures pre- and post-mood induction. Correlations between pre- and post-mood induction, difference, and residualized change scores for the entire sample were examined. Five-minute HP during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .94-.96, $p \leq .001$ for all). Five-minute HP reactivity and recovery were positively correlated when using difference and residualized change scores (r = .59, $p \leq .001$ for all). Five-minute RSA during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .75-.90, $p \leq .001$ for all). Five-minute RSA reactivity and recovery were positively correlated when using difference and residualized change scores (r = .46-.47, $p \leq .001$ for all). Five-minute PEP during baseline, mood induction, and recovery were positively correlated when using pre- and post-

mood induction (r = .89-.91, $p \leq .001$ for all). Finally, five-minute PEP reactivity and recovery were positively correlated when using difference and residualized change scores (r = .37-.45, $p \leq .001$ for all). These results suggest that pre- and post-mood induction scores for cardiovascular measures all correlate with one another as expected.

Correlations between cardiovascular pre- and post-mood induction, difference, and residualized change scores were evaluated between groups. For formerly depressed participants, five-minute HP during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .95-.97, $p \leq .001$ for all). Five-minute HP reactivity and recovery were positively correlated when using difference and residualized change scores (r = .54-.55, $p \leq .05$ for all). Five-minute RSA during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .89-.93, $p \leq .001$ for all). Five-minute RSA reactivity and recovery were positively correlated when using difference and residualized change scores (r = .48-.53, $p \leq .01$ for all). Five-minute PEP during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .92-.96, $p \leq .001$ for all). Five-minute PEP reactivity and recovery were positively correlated when using difference and residualized change scores (r = .59-.60, $p \leq .05$ for all). Overall, these correlations show a similar pattern to those previously reported for the entire sample.

For healthy control participants, five-minute HP during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .94-.97, $p \leq .001$ for all). Five-minute HP reactivity and recovery were positively correlated when using difference and residualized change scores (r = .45-.48, $p \leq .05$ for all). Five-minute RSA during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .69-.88, $p \leq .001$ for all). Five-minute RSA reactivity and recovery were

positively correlated when using difference and residualized change scores (r = .46, $p \leq .001$ for all). Five-minute PEP during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .96-.97, $p \leq .001$ for all). Five-minute PEP reactivity and recovery were positively correlated when using difference and residualized change scores (r = .39-.46, $p \leq .05$ for all). Overall, these correlations show a similar pattern to those previously reported for the entire sample.

Correlations between cardiovascular pre- and post-mood induction, difference, and residualized change scores were evaluated between groups and conditions. For formerly depressed participants exposed to the sad mood induction, five-minute HP during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .94-.98, $p \leq .001$ for all). Five-minute HP reactivity and recovery were not correlated when using difference and residualized change scores. Five-minute RSA during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .94-.96, $p \leq .001$ for all). Five-minute RSA reactivity and recovery were positively correlated when using difference and residualized change scores (r = .65-.67, $p \leq .01$ for all). Five-minute PEP during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .93-.95, $p \leq .001$ for all). Five-minute PEP reactivity and recovery were not correlated when using difference and residualized change scores. Overall, these correlations show a similar pattern to those previously reported for the entire sample with the exception of some cardiovascular measures.

For formerly depressed participants exposed to the neutral mood induction, five-minute HP during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .93-.97, $p \leq .001$ for all). Five-minute HP reactivity and recovery

were positively correlated when using difference and residualized change scores (r = .88-.89, $p \leq$ .05 for all). Five-minute RSA during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .85-.93, $p \leq .001$ for all). Five-minute RSA reactivity and recovery were not correlated when using difference and residualized change scores. Five-minute PEP during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .91-.98, $p \leq .001$ for all). Five-minute PEP reactivity and recovery were not correlated when using difference and residualized change scores. Overall, these correlations show a similar pattern to those previously reported for the entire sample with the exception of some cardiovascular measures.

For healthy control participants exposed to the sad mood induction, five-minute HP during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .92-.97, $p \leq .001$ for all). Five-minute HP reactivity and recovery were positively correlated when using residualized change, but not difference, scores (r = .59, $p \leq .01$). Five-minute RSA during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .85-.92, $p \leq .001$ for all). Five-minute RSA reactivity and recovery were positively correlated when using difference and residualized change scores (r = .49-.50, $p \leq .01$ for all). Five-minute PEP during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .96-.98, $p \leq$ .001 for all). Five-minute PEP reactivity and recovery were positively correlated when using difference, but not residualized change, scores (r = .63, $p \leq .05$). Overall, these correlations show a similar pattern to those previously reported for the entire sample with the exception of some cardiovascular measures.

For healthy control participants exposed to the neutral mood induction, five-minute HP during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .95-.97, $p \leq .001$ for all). Five-minute HP reactivity and recovery were not correlated when using difference and residualized change scores. Five-minute RSA during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .47-.90, $p \leq .01$-.001). Five-minute RSA reactivity and recovery were positively correlated when using difference and residualized change scores (r = .47-.48, $p \leq .01$ for all). Five-minute PEP during baseline, mood induction, and recovery were positively correlated when using pre- and post-mood induction (r = .92-.97, $p \leq .001$ for all). Five-minute PEP reactivity and recovery were not correlated when using difference and residualized change scores. Overall, these correlations show a similar pattern to those previously reported for the entire sample with the exception of some cardiovascular measures.

Cognitive, mood, and cardiovascular measures. Bivariate correlations were used to investigate the relationship between cognitive, mood, and cardiovascular measures pre- and post-mood induction. Correlations between pre- and post-mood induction, difference, and residualized change scores for the entire sample were examined. The majority of the self-report measures (with the exception of the PANAS-X P_{PRE}, PANAS-X P_{POST}, and Z_{RES}VAS) were not significantly correlated with HP, RSA, or PEP.

Correlations between pre- and post-mood induction, difference, and residualized change scores were evaluated between groups. For formerly depressed participants, the majority of the self-report measures (with the exception of the VAS_{POST} and PANAS-X N_{DS}) were not significantly correlated with HP, RSA, or PEP. For healthy control participants, the majority of the self-report measures (with the exception of the PANAS-X P_{PRE}, PANAS-X P_{POST}, VAS_{DS},

PANAS-X G_{DS}, PANAS-X S_{DS}, Z_{RES}DAS, Z_{RES}VAS, Z_{RES}PANAS-X G, and Z_{RES}PANAS-X S) were not significantly correlated with HP, RSA, or PEP. Overall, these correlations show a similar pattern to those previously reported for the entire sample with the exception of some measures. However, one notable association was observed. The post-mood induction VAS was negatively correlated with five-minute HP during baseline, mood induction, and recovery (r = -.56--.57, $p \leq .05$ for all), suggesting that formerly depressed participants who experience an increase in sadness post-mood induction exhibit decreased HP during baseline, mood induction, and recovery. These correlations run counter to the trends found for Hypotheses 2, 3, and 4. Consequently, additional analyses were conducted for the group and condition interaction, which revealed that the relationship between the VAS_{POST} and HP during baseline, mood induction, and recovery was more robust in formerly depressed participants exposed to the neutral mood induction (r = -.51--.62, $p > .19$ for all) than those exposed to the sad mood induction (r = -.31--.46, $p > .19$ for all).

Correlations between pre- and post-mood induction, difference, and residualized change scores were evaluated between groups and conditions. For formerly depressed participants exposed to the sad mood induction, the majority of the self-report measures (with the exception of the Z_{RES}DAS and $Z_{RES}RSA_{RC}$) were not significantly correlated with HP, RSA, or PEP. For formerly depressed participants exposed to the neutral mood induction, the majority of the self-report measures (with the exception of the PANAS-X N_{POST} and PEP_{BL5}, PANAS-X G_{PRE}, PANAS-X G_{POST}, and PEP_{RC5} and VAS_{DS}, Z_{RES}VAS, RSA_{DS5RA}, and $Z_{RES}RSA_{RA}$) were not significantly correlated with HP, RSA, or PEP. For healthy control participants exposed to the sad mood induction, the majority of the self-report measures (with the exception of the Z_{RES}DAS, Z_{RES}VAS, and $Z_{RES}PEP_{RA}$, VAS_{DS} and PEP_{DS5RC}, PANAS-X P_{DS}, PANAS-X S_{DS}, and

HP_{DS5RA}, PANAS-X S_{DS} and HP_{DS5RC}, Z_{RES}PANAS-X P and $Z_{RES}HP_{RA}$, and Z_{RES}PANAS-X G and $Z_{RES}HP_{RC}$) were not significantly correlated with HP, RSA, or PEP. For healthy control participants exposed to the neutral mood induction, the majority of the self-report measures (with the exception of the PANAS-X N_{POST}, PANAS-X P_{PRE}, and PEP_{BL5}, PANAS-X P_{PRE} and PEP_{RC5}, VAS_{DS} and HP_{DS5RA}, PANAS-X S_{DS} and PEP_{DS5RC}, Z_{RES}DAS and $Z_{RES}HP_{RC}$, Z_{RES}PANAS-X N, Z_{RES}PANAS-X G, and $Z_{RES}PEP_{RC}$) were not significantly correlated with HP, RSA, or PEP. Overall, these correlations show a similar pattern to those previously reported for the entire sample with the exception of some measures.

Table 23. *Correlation Matrix for Cognitive, Mood, and Cardiovascular Pre- and Post-Mood Induction Scores for the Entire Sample*

Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.
1. DAS I																				
2. DAS II	.73[e]																			
3. VAS_{PRE}	-.31[e]	-.24[d]																		
4. VAS_{POST}	-.17	-.26[d]	.36[e]																	
5. PANAS-X N_{PRE}	-.47[e]	-.43[e]	.37[e]	.08																
6. PANAS-X N_{POST}	-.45[e]	-.40[e]	.41[e]	.22[c]	.91[e]															
7. PANAS-X P_{PRE}	.21[c]	.12	-.06	-.07	.08	.06														
8. PANAS-X P_{POST}	.22[c]	.27[d]	-.13	-.27[d]	.08	.02	.85[e]													
9. PANAS-X G_{PRE}	-.47[e]	-.44[e]	.34[e]	.08	.87[e]	.79[e]	-.07	-.07												
10. PANAS-X G_{POST}	-.43[e]	-.46[e]	.33[e]	.17	.80[e]	.84[e]	-.04	-.11	.90[e]											
11. PANAS-X S_{PRE}	-.44[e]	-.42[e]	.35[e]	.14	.78[e]	.73[e]	-.08	-.07	.70[e]	.67[e]										
12 PANAS-X S_{POST}	-.45[e]	-.43[e]	.37[e]	.39[e]	.67[e]	.75[e]	-.05	-.17	.60[e]	.70[e]	.84[e]									
13. HP_{BL5}	-.03	-.02	.11	-.18	.06	.00	.02	.09	.04	-.05	-.12	-.03								
14. HP_{MI5}	-.04	.004	.12	-.21	.13	.03	.10	.16	.03	-.09	-.07	-.01	.95[e]							
15. HP_{RC5}	-.12	-.13	.11	-.21	.10	.04	.09	.14	.01	-.04	-.06	.02	.94[e]	.96[e]						
16. RSA_{BL5}	.02	-.01	.07	.07	.09	.01	.09	.08	.10	.04	.08	.06	-.02	.04	-.03					
17. RSA_{MI5}	-.01	.003	.14	.17	.02	-.01	.15	.11	.05	-.003	-.01	-.01	.13	.14	.08	.75[e]				
18. RSA_{RC5}	-.03	-.03	.11	.08	.13	.06	.16	.14	.14	.09	.10	.06	.02	.07	.02	.90[e]	.80[e]			
19. PEP_{BL5}	.06	.06	-.16	-.29	-.13	-.17	.25	.34[c]	-.13	-.07	-.27	-.27	.23[c]	.22[c]	.20[c]	-.19	-.13	-.07		
20. PEP_{MI5}	-.04	-.03	-.09	-.21	-.04	-.10	.27	.33[c]	-.07	-.04	-.16	-.16	.23[c]	.23[c]	.21[c]	-.20	-.14	-.07	.91[e]	
21. PEP_{RC5}	.07	.03	-.15	-.26	-.03	-.09	.38[c]	.44[d]	-.11	-.06	-.18	-.15	.23[c]	.25[c]	.25[c]	-.06	-.06	.03	.89[e]	.91[e]

Note. DAS I = Dysfunctional Attitudes Scale – Short Form I; DAS II = Dysfunctional Attitudes Scale – Short Form II; VAS = Visual Analogue Scale; $_{PRE}$ = pre mood induction; $_{POST}$ = post mood induction; PANAS-X = Positive and Negative Affect Scale – Expanded Form; N = negative affect general dimension scale; P = positive affect general dimension scale; G = guilt basic negative emotional scale; S = sadness basic negative emotional scale; HP = heart period at baseline; $_{BL}$ = baseline; $_{5}$ = average obtained during a five-minute interval; $_{MI}$ = mood induction; $_{RC}$ = recovery; RSA = respiratory sinus arrhythmia; PEP = pre-ejection period; [c] = $p \leq .05$; [d] = $p \leq .01$; [e] = $p \leq .001$.

Table 24. *Correlation Matrices for Cognitive, Mood, and Cardiovascular Pre- and Post-Mood Induction Scores by Group*

FD

Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.
1. DAS I																				
2. DAS II	.69[e]																			
3. VAS_{PRE}	-.03	.13																		
4. VAS_{POST}	.21	.04	.14																	
5. PANAS-X N_{PRE}	-.55[e]	-.50[e]	.22	-.12																
6. PANAS-X N_{POST}	-.49[e]	-.46[d]	.16	.11	.90[e]															
7. PANAS-X P_{PRE}	.03	-.02	-.10	.07	.15	.15														
8. PANAS-X P_{POST}	-.05	-.01	-.04	-.23	.28	.17	.83[e]													
9. PANAS-X G_{PRE}	-.53[e]	-.54[e]	.19	-.13	.87[e]	.78[e]	-.02	.13												
10. PANAS-X G_{POST}	-.49[e]	-.58[e]	.15	.02	.80[e]	.88[e]	.03	.07	.86[e]											
11. PANAS-X S_{PRE}	-.48[e]	-.35[c]	.29	-.05	.76[e]	.72[e]	-.13	.02	.70[e]	.69[e]										
12 PANAS-X S_{POST}	-.44[d]	-.31[c]	.15	.19	.66[e]	.76[e]	.10	.03	.53[e]	.70[e]	.84[e]									
13. HP_{BL5}	.28	.28	-.06	-.56[c]	.06	-.08	.06	.10	.07	-.06	-.35	-.38								
14. HP_{MI5}	.18	.21	.03	-.57[c]	.19	.01	.09	.09	.12	-.05	-.23	-.26	.97[e]							
15. HP_{RC5}	.14	.07	-.001	-.57[c]	.12	.02	.08	.04	.08	-.001	-.23	-.20	.95[e]	.96[e]						
16. RSA_{BL5}	-.12	-.21	.13	-.02	.23	.11	.08	.16	.19	.13	.27	.16	-.45	-.35	-.40					
17. RSA_{MI5}	-.08	-.14	.11	.08	.16	.07	.10	.20	.12	.04	.16	.06	-.36	-.29	-.30	.89[e]				
18. RSA_{RC5}	-.06	-.11	.08	-.02	.28	.18	.11	.24	.23	.17	.31	.18	-.33	-.28	-.34	.93[e]	.91[e]			
19. PEP_{BL5}	-.04	.03	-.26	-.24	-.24	-.09	.01	.09	-.22	-.05	-.47	-.36	.12	.02	-.02	-.67[d]	-.65[d]	-.57[c]		
20. PEP_{MI5}	-.07	.06	-.21	-.21	-.14	-.04	.04	.11	-.12	-.03	-.33	-.25	.12	.03	-.08	-.64[c]	-.66[d]	-.56[c]	.96[e]	
21. PEP_{RC5}	.04	-.05	-.22	-.15	-.12	.04	.20	.17	-.13	.03	-.26	-.13	.06	.004	.08	-.57[c]	-.57[c]	-.52	.92[e]	.94[e]

HC

Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.
1. DAS I																				
2. DAS II	.72[e]																			
3. VAS_{PRE}	-.44[e]	-.39[e]																		
4. VAS_{POST}	-.28[c]	-.31[d]	.46[e]																	
5. PANAS-X N_{PRE}	-.33[d]	-.29[c]	.48[e]	.14																
6. PANAS-X N_{POST}	-.34[d]	-.25[c]	.58[e]	.20	.91[e]															
7. PANAS-X P_{PRE}	.20	.06	.02	-.06	.19	.16														
8. PANAS-X P_{POST}	.21	.24[c]	-.10	-.22[c]	.19	.14	.85[e]													
9. PANAS-X G_{PRE}	-.33[d]	-.27[c]	.46[e]	.14	.82[e]	.76[e]	.04	-.01												
10. PANAS-X G_{POST}	-.29[d]	-.27[c]	.46[e]	.15	.72[e]	.75[e]	.08	-.01	.91[e]											
11. PANAS-X S_{PRE}	-.33[d]	-.40[e]	.35[e]	.19	.74[e]	.67[e]	.08	.04	.62[e]	.56[e]										
12 PANAS-X S_{POST}	-.38[e]	-.42[e]	.52[e]	.48[e]	.62[e]	.68[e]	-.01	-.14	.61[e]	.62[e]	.80[e]									
13. HP_{BL5}	-.28	-.21	.30	.09	.02	.08	-.01	.10	.01	-.02	.12	.31								
14. HP_{MI5}	-.24	-.16	.28	.11	.11	.13	.04	.15	.01	-.03	.17	.30	.97[e]							
15. HP_{RC5}	-.34	-.29	.28	.07	.12	.14	.04	.15	-.01	.01	.18	.28	.94[e]	.95[e]						
16. RSA_{BL5}	.07	.05	.05	.14	-.001	-.02	.07	.01	.06	.03	-.03	.03	.24	.24	.16					
17. RSA_{MI5}	.03	.06	.15	.21	-.09	-.07	.18	.09	-.001	-.05	-.14	-.06	.37	.33	.24	.69[e]				
18. RSA_{RC5}	-.03	-.01	.14	.15	.03	.01	.17	.09	.10	.08	-.02	.01	.22	.23	.20	.88[e]	.76[e]			

Table 24 Continued

19.	PEP_{BL5}	.12	.05	-.02	-.30	-.002	-.19	.36	.45[c]	.40	-.02	-.06	-.17	.27	.36	.29	.02	.07	.16		
20.	PEP_{MI5}	-.03	-.12	.08	-.17	.08	-.10	.40[c]	.46[c]	.09	.04	.08	-.02	.35	.43[c]	.38	.03	.08	.19	.97[e]	
21.	PEP_{RC5}	.05	-.004	-.01	-.24	.11	-.08	.42[c]	.51[d]	.09	.06	.02	-.04	.35	.44[c]	.38	.12	.10	.24	.96[e]	.96[e]

Note. FD = formerly depressed; HC = healthy control; DAS I = Dysfunctional Attitudes Scale – Short Form I; DAS II = Dysfunctional Attitudes Scale – Short Form II; VAS = Visual Analogue Scale; PRE = pre mood induction; POST = post mood induction; PANAS-X = Positive and Negative Affect Scale – Expanded Form; N = negative affect general dimension scale; P = positive affect general dimension scale; G = guilt basic negative emotional scale; S = sadness basic negative emotional scale; HP = heart period at baseline; BL = baseline; 5 = average obtained during a five-minute interval; MI = mood induction; RC = recovery; RSA = respiratory sinus arrhythmia; PEP = pre-ejection period; [c] = $p \leq .05$; [d] = $p \leq .01$; [e] = $p \leq .001$.

Table 25. *Correlation Matrices for Cognitive, Mood, and Cardiovascular Pre- and Post-Mood Induction Scores by Group and Condition*

FD/SMI

Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.
1. DAS I																				
2. DAS II	.78[e]																			
3. VAS_{PRE}	-.17	.01																		
4. VAS_{POST}	.42	.28	.16																	
5. PANAS-X N_{PRE}	-.85[e]	-.63[d]	.20	-.46																
6. PANAS-X N_{POST}	-.76[e]	-.60[d]	.09	-.23	.87[e]															
7. PANAS-X P_{PRE}	.17	.06	-.05	.06	.10	.04														
8. PANAS-X P_{POST}	.07	-.03	-.07	-.31	.35	.12	.77[e]													
9. PANAS-X G_{PRE}	-.79[e]	-.68[e]	.03	-.49[c]	.90[e]	.77[e]	-.10	.13												
10. PANAS-X G_{POST}	-.78[e]	-.71[e]	-.01	-.35	.80[e]	.89[e]	-.08	.01	.87[e]											
11. PANAS-X S_{PRE}	-.77[e]	-.54[c]	.09	-.40	.88[e]	.82[e]	-.21	.01	.82[e]	.79[e]										
12 PANAS-X S_{POST}	-.67[d]	-.51[c]	-.03	-.12	.72[e]	.84[e]	.04	-.07	.60[d]	.79[e]	.81[e]									
13. HP_{BL5}	.29	.15	-.47	-.36	.002	-.18	.50	.42	-.04	-.14	-.31	-.19								
14. HP_{MI5}	.22	.06	-.42	-.31	.12	-.12	.56	.45	-.01	-.12	-.20	-.03	.98[e]							
15. HP_{RC5}	.16	-.003	-.35	-.46	.002	-.11	.45	.36	-.07	-.06	-.29	-.07	.95[e]	.94[e]						
16 RSA_{BL5}	-.18	-.15	.50	.19	.32	.27	.23	.26	.13	.17	.34	.33	-.76[c]	-.67[c]	-.74[c]					
17. RSA_{MI5}	-.09	.02	.49	.13	.26	.21	.32	.39	-.02	.02	.25	.24	-.70[c]	-.64	-.67[c]	.94[e]				
18. RSA_{RC5}	-.17	-.02	.34	.07	.41	.37	.27	.36	.15	.20	.42	.39	-.74[c]	-.67[c]	-.74[c]	.94[e]	.96[e]			
19. PEP_{BL5}	-.03	-.14	-.08	-.14	.06	.33	.03	.12	.12	.39	-.25	-.17	.08	-.01	.12	-.51	-.52	-.46		
20. PEP_{MI5}	-.09	-.16	-.21	-.06	.22	.37	.06	.11	.22	.38	-.11	-.06	.09	.04	.07	-.49	-.55	-.46	.95[e]	
21. PEP_{RC5}	.06	-.07	-.18	-.18	.16	.26	.22	.25	.07	.25	-.20	-.10	.25	.20	.27	-.51	-.53	-.48	.93[e]	.95[e]

FD/NMI

Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.
1. DAS I																				
2. DAS II	.63[e]																			
3. VAS_{PRE}	.15	.14																		
4. VAS_{POST}	-.17	-.07	.37																	
5. PANAS-X N_{PRE}	-.24	-.41[c]	.29	.21																
6. PANAS-X N_{POST}	-.26	-.35	.30	.31	.95[e]															
7. PANAS-X P_{PRE}	-.17	-.10	-.10	-.05	.21	.25														
8. PANAS-X P_{POST}	-.15	-.01	-.03	-.11	.24	.26	.93[e]													
9. PANAS-X G_{PRE}	-.27	-.44[c]	.32	.27	.88[e]	.84[e]	.08	.12												
10. PANAS-X G_{POST}	-.21	-.46[c]	.38	.32	.81[e]	.87[e]	.14	.18	.91[e]											
11. PANAS-X S_{PRE}	-.14	-.17	.52[d]	.36	.58[d]	.58[d]	-.05	.04	.57[d]	.53[d]										
12 PANAS-X S_{POST}	-.25	-.13	.47[c]	.43[c]	.55[d]	.63[e]	.14	.20	.49[c]	.52[d]	.94[e]									
13. HP_{BL5}	.36	.20	-.49	-.62	-.15	-.23	-.62	-.41	-.01	.05	-.67	-.79								
14. HP_{MI5}	.25	.01	-.52	-.58	-.07	-.16	-.58	-.47	.002	.04	-.64	-.74	.97[e]							
15. HP_{RC5}	.12	-.16	-.44	-.51	-.08	-.16	-.55	-.55	-.01	.03	-.62	-.69	.93[e]	.97[e]						
16. RSA_{BL5}	-.08	-.31	.000	-.20	.16	-.02	-.05	.06	.28	.15	.22	.01	-.21	-.15	-.13					
17. RSA_{MI5}	-.10	-.31	.000	.10	.08	-.04	-.09	.02	.28	.11	.08	-.12	-.08	-.02	.03	.85[e]				

Table 25 Continued

18.	RSA_{RC5}	.04	-.20	.02	-.12	.16	-.01	-.04	.12	.32	.19	.20	-.06	.08	.11	.08	.93[e]	.87[e]			
19.	PEP_{BL5}	-.07	.43	-.48	-.73	.72	-.81[c]	-.05	.03	-.80	-.79	-.79	-.69	.33	.23	.23	-.89[c]	-.83[c]	-.73		
20.	PEP_{MI5}	-.01	.53	-.39	-.65	-.70	-.77	-.01	.12	-.74	-.73	-.70	-.61	.24	.09	-.09	-.85[c]	-.89[c]	-.70	.98[e]	
21.	PEP_{RC5}	-.06	.26	-.54	-.54	-.80	-.83	-.03	-.39	-.98[d]	-.98[e]	-.53	-.30	-.31	-.26	-.18	-.91[c]	-.95[c]	-.95[c]	.91[e]	.96[e]

HC/SMI

Measure		1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.
1.	DAS I																				
2.	DAS II	.71[e]																			
3.	VAS_{PRE}	-.55[e]	-.46[d]																		
4.	VAS_{POST}	-.35[c]	-.38[c]	.41[d]																	
5.	PANAS-X N_{PRE}	-.30[c]	-.26	.54[e]	.13																
6.	PANAS-X N_{POST}	-.30[c]	-.25	.69[e]	.24	.92[e]															
7.	PANAS-X P_{PRE}	.04	.01	.06	.01	.36[c]	.33[c]														
8.	PANAS-X P_{POST}	.02	.17	-.05	-.13	.33[c]	.31[c]	.82[e]													
9.	PANAS-X G_{PRE}	-.34[c]	-.35[c]	.56[e]	.19	.73[e]	.67[e]	.18	.05												
10.	PANAS-X G_{POST}	-.31[c]	-.34[c]	.56[e]	.18	.63[e]	.68[e]	.23	.08	.89[e]											
11.	PANAS-X S_{PRE}	-.31[c]	-.45[d]	.42[d]	.33[c]	.74[e]	.71[e]	.25	.17	.51[e]	.43[d]										
12	PANAS-X S_{POST}	-.38[c]	-.47[e]	.59[e]	.65[e]	.48[e]	.58[e]	.08	-.11	.45[d]	.47[e]	.74[e]									
13.	HP_{BL5}	-.24	-.16	.26	.16	.11	.08	-.05	.07	.10	.01	.32	.45								
14.	HP_{MI5}	-.22	-.11	.23	.09	.19	.14	-.02	.16	.08	-.02	.34	.38	.97[e]							
15.	HP_{RC5}	-.36	-.25	.22	.09	.22	.15	.01	.16	.04	.02	.36	.36	.92[e]	.95[e]						
16.	RSA_{BL5}	.13	.03	-.08	.10	-.05	-.11	.14	.002	.01	-.05	.01	.02	.45	.36	.25					
17.	RSA_{MI5}	.04	-.01	-.01	.16	-.03	-.09	.15	.03	.12	-.02	.05	.04	.47	.37	.27	.92[e]				
18.	RSA_{RC5}	-.07	-.05	.07	.14	.01	-.03	.24	.11	.19	.08	.03	-.01	.34	.28	.25	.85[e]	.89[e]			
19.	PEP_{BL5}	-.08	.09	.27	-.13	.24	.12	.14	.38	.31	.20	.09	-.04	.55[c]	.60[c]	.61[c]	.38	.46	.58[c]		
20.	PEP_{MI5}	-.30	-.13	.38	.07	.37	.24	.29	.45	.40	.29	.30	.16	.58[c]	.62[c]	.65[c]	.30	.40	.54	.96[e]	
21.	PEP_{RC5}	-.13	.02	.26	-.03	.31	.16	.21	.43	.33	.18	.20	.06	.55[c]	.61[c]	.61[c]	.37	.44	.55	.98[e]	.98[e]

HC/NMI

Measure		1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.
1.	DAS I																				
2.	DAS II	.73[e]																			
3.	VAS_{PRE}	-.30	-.33[c]																		
4.	VAS_{POST}	-.22	-.42[d]	.64[e]																	
5.	PANAS-X N_{PRE}	-.37[c]	-.32	.45[d]	.33[c]																
6.	PANAS-X N_{POST}	-.38[c]	-.25	.43[d]	.20	.90[e]															
7.	PANAS-X P_{PRE}	.35[c]	.13	-.01	.03	-.02	-.05														
8.	PANAS-X P_{POST}	.42[d]	.35[c]	-.15	-.21	-.02	-.08	.88[e]													
9.	PANAS-X G_{PRE}	-.33[c]	-.20	.41[d]	.23	.91[e]	.86[e]	-.10	-.09												
10.	PANAS-X G_{POST}	-.28	-.20	.34[c]	.22	.84[e]	.84[e]	-.10	-.15	.94[e]											
11.	PANAS-X S_{PRE}	-.37[c]	-.36[c]	.38[c]	.34[c]	.76[e]	.68[e]	-.15	-.19	.73[e]	.70[e]										
12	PANAS-X S_{POST}	-.38[c]	-.37[c]	.39[c]	.28	.82[e]	.80[e]	-.13	-.19	.81[e]	.83[e]	.92[e]									
13.	HP_{BL5}	-.33	-.32	.53	.16	-.20	.11	.03	.11	-.16	-.07	.29	.06								
14.	HP_{MI5}	-.30	-.35	.43	.19	-.18	.09	.14	.18	-.18	-.07	-.29	.08	.97[e]							

Table 25 Continued

15.	HP_{RC5}	-.33	-.42	.48	.16	-.11	.13	.08	.15	-.13	-.01	-.17	.16	.96[e]	.95[e]						
16.	RSA_{BL5}	.02	.09	.17	-.11	.07	.05	.04	.10	.01	.08	-.05	-.01	.11	.15	.14					
17.	RSA_{MI5}	.04	.16	.27	-.15	-.16	-.09	.28	.27	-.11	-.12	-.27	-.22	.52	.47	.37	.47[d]				
18.	RSA_{RC5}	.02	.04	.20	-.07	.06	.04	.12	.14	-.004	.05	-.06	-.01	.19	.23	.21	.90[e]	.63[e]			
19.	PEP_{BL5}	.50	.22	-.35	-.21	-.41	-.56[c]	.59[c]	.45	-.35	-.32	-.25	-.28	-.17	-.02	-.17	-.11	.15	-.06		
20.	PEP_{MI5}	.44	.12	-.29	-.17	-.45	-.53	.53	.39	-.40	-.33	-.22	-.22	-.02	.14	-.02	-.01	.19	.03	.97[e]	
21.	PEP_{RC5}	.42	.18	-.34	-.24	-.27	-.33	.66[d]	.54	-.27	-.08	-.25	-.07	.02	.18	.03	.12	.20	.15	.92[e]	.92[e]

Note. FD = formerly depressed; HC = healthy control; SMI = sad mood induction; NMI = neutral mood induction; DAS I = Dysfunctional Attitudes Scale – Short Form I; DAS II = Dysfunctional Attitudes Scale – Short Form II; VAS = Visual Analogue Scale; PRE = pre mood induction; POST = post mood induction; PANAS-X = Positive and Negative Affect Scale – Expanded Form; N = negative affect general dimension scale; P = positive affect general dimension scale; G = guilt basic negative emotional scale; S = sadness basic negative emotional scale; HP = heart period at baseline; BL = baseline; 5 = average obtained during a five-minute interval; MI = mood induction; RC = recovery; RSA = respiratory sinus arrhythmia; PEP = pre-ejection period; [c] = $p \leq .05$; [d] = $p \leq .01$; [e] = $p \leq .001$.

Table 26. *Correlation Matrices for Cognitive, Mood, and Cardiovascular Change Scores for Entire Sample*

Measure		1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1.	DAS_{DS}											
2.	VAS_{DS}	-.20[c]										
3.	PANAS-X N_{DS}	.04	.24[d]									
4.	PANAS-X P_{DS}	.26[d]	-.36[e]	-.05								
5.	PANAS-X G_{DS}	-.15	.28[d]	.58[e]	-.17							
6.	PANAS-X S_{DS}	-.02	.48[e]	.46[e]	-.38[e]	.48[e]						
7.	HP_{DS5RA}	.18	-.11	-.24	.08	-.24	-.18					
8.	HP_{DS5RC}	.06	-.18	.01	.05	.15	-.08	.59[e]				
9.	RSA_{DS5RA}	.05	.15	.12	-.08	.05	.03	-.23	-.02			
10.	RSA_{DS5RC}	.10	-.10	.05	.04	.10	-.08	-.15	.19	.47[e]		
11.	PEP_{DS5RA}	.01	.17	-.21	-.14	-.28	-.02	.13	.08	-.12	.04	
12.	PEP_{DS5RC}	.07	-.06	-.31	.04	-.27	.15	.15	.06	-.28	-.16	.37[e]

Measure		1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1.	Z_{RES}DAS											
2.	Z_{RES}VAS	-.23[c]										
3.	Z_{RES}PANAS-X N	-.01	.29[d]									
4.	Z_{RES}PANAS-X P	.29[e]	-.38[e]	-.06								
5.	Z_{RES}PANAS-X G	-.07	.52[e]	.53[e]	-.40[e]							
6.	Z_{RES}PANAS-X S	-.17	.28[d]	.64[e]	-.15	.52[e]						
7.	$Z_{RES5}HP_{RA}$	.18	-.12	-.22	.11	-.13	-.27					
8.	$Z_{RES5}HP_{RC}$	-.03	-.28	.05	.11	-.002	.12	.59[e]				
9.	$Z_{RES5}RSA_{RA}$	.04	.20[c]	.05	-.08	.004	.01	-.09	-.16			
10.	$Z_{RES5}RSA_{RC}$	.07	-.04	.01	.04	-.09	.08	-.06	.04	.46[e]		
11.	$Z_{RES5}PEP_{RA}$	-.12	.22	-.14	-.16	.08	-.28	.12	.08	-.12	.16	
12.	$Z_{RES5}PEP_{RC}$	.05	-.08	-.24	.09	.18	-.24	.21[c]	.17	-.15	-.04	.45[e]

Note. DAS = Dysfunctional Attitudes Scale; $_{DS}$ = difference score; VAS = Visual Analogue Scale; PANAS-X = Positive and Negative Affect Scale – Expanded Form; N = negative affect general dimension scale; P = positive affect general dimension scale; G = guilt basic negative emotional scale; S = sadness basic negative emotional scale; HP = heart period at baseline; $_5$ = average obtained during a five-minute interval; RSA = respiratory sinus arrhythmia; PEP = pre-ejection period; Z_{RES} = standardized residualized change scores; [c] = $p \leq .05$; [d] = $p \leq .01$; [e] = $p \leq .001$.

Table 27. *Correlation Matrices for Cognitive, Mood, and Cardiovascular Change Scores by Group*

					FD						
Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. DAS_{DS}											
2. VAS_{DS}	-.31										
3. PANAS-X N_{DS}	-.09	.47[d]									
4. PANAS-X P_{DS}	.24	-.52[e]	-.18								
5. PANAS-X G_{DS}	-.20	.40[c]	.71[e]	-.26							
6. PANAS-X S_{DS}	-.06	.56[e]	.56[e]	-.56[e]	.60[e]						
7. HP_{DS5RA}	.22	-.18	-.34	-.36	-.31	-.01					
8. HP_{DS5RC}	.40	-.36	.09	-.15	.16	.29	.54[c]				
9. RSA_{DS5RA}	.15	.22	.16	.06	.03	.11	-.38	.18			
10. RSA_{DS5RC}	.12	-.05	.07	.18	.14	.01	-.72[d]	-.41	.53[d]		
11. PEP_{DS5RA}	.35	-.06	-.39	-.02	-.42	-.13	.02	-.41	-.41	-.06	
12. PEP_{DS5RC}	.02	-.25	-.54[c]	-.23	-.30	-.01	.23	-.003	-.06	-.17	.60[c]
Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. $Z_{RES}DAS$											
2. $Z_{RES}VAS$	-.25										
3. Z_{RES}PANAS-X N	-.07	.48[d]									
4. Z_{RES}PANAS-X P	.22	-.52[e]	-.17								
5. Z_{RES}PANAS-X G	-.02	.53[e]	.61[e]	-.49[e]							
6. Z_{RES}PANAS-X S	-.22	.40[c]	.74[e]	-.23	.65[e]						
7. $Z_{RES5}HP_{RA}$	.07	-.19	-.31	-.35	.10	-.33					
8. $Z_{RES5}HP_{RC}$	.11	-.43	.05	-.16	.33	.10	.55[c]				
9. $Z_{RES5}RSA_{RA}$	.14	.25	.05	.14	-.03	-.003	-.13	.12			
10. $Z_{RES5}RSA_{RC}$	.19	-.04	-.03	.32	-.09	.12	-.50	-.50	.48[d]		
11. $Z_{RES5}PEP_{RA}$	.35	-.04	-.39	-.02	-.07	-.43	.05	-.43	-.44	-.15	
12. $Z_{RES5}PEP_{RC}$	.07	-.22	-.47	-.21	.07	-.24	.21	.06	-.11	-.19	.59[c]
					HC						
Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. DAS_{DS}											
2. VAS_{DS}	-.11										
3. PANAS-X N_{DS}	.13	.03									
4. PANAS-X P_{DS}	.25[c]	-.29[d]	-.02								
5. PANAS-X G_{DS}	-.12	-.01	.44[e]	-.14							
6. PANAS-X S_{DS}	.02	.41[e]	.36[e]	-.34[d]	.30[d]						
7. HP_{DS5RA}	.07	.09	-.19	.10	-.01	-.38					
8. HP_{DS5RC}	-.15	-.03	-.10	.09	.45[c]	-.44[c]	.48[c]				
9. RSA_{DS5RA}	.03	.08	.10	-.11	.003	-.04	-.08	-.10			
10. RSA_{DS5RC}	.10	-.13	.04	.01	.08	-.14	.08	.36	.46[e]		
11. PEP_{DS5RA}	-.26	.42[c]	.11	-.22	.04	.13	-.15	.10	.05	.10	
12. PEP_{DS5RC}	.03	.27	.09	.10	-.03	.39[c]	.06	.08	-.43[c]	-.18	.46[c]
Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. $Z_{RES}DAS$											
2. $Z_{RES}VAS$	-.16										
3. Z_{RES}PANAS-X N	.06	.10									
4. Z_{RES}PANAS-X P	.28[c]	-.32[d]	.004								
5. Z_{RES}PANAS-X G	-.07	.50[e]	.43[e]	-.37[e]							
6. Z_{RES}PANAS-X S	-.08	.01	.48[e]	-.09	.29[d]						
7. $Z_{RES5}HP_{RA}$	.12	.09	-.08	.14	.30	-.06					
8. $Z_{RES5}HP_{RC}$	-.14	-.16	.07	.21	-.35	.51[d]	.45[c]				
9. $Z_{RES5}RSA_{RA}$	.03	.16	.06	-.13	.02	-.05	-.07	-.29			
10. $Z_{RES5}RSA_{RC}$	.04	-.04	.04	-.03	-.10	.08	.06	.20	.46[e]		
11. $Z_{RES5}PEP_{RA}$	-.43[c]	.48[c]	.20	-.25	.26	.04	-.15	.16	.05	.09	
12. $Z_{RES5}PEP_{RC}$	-.07	.20	.11	.17	.39[c]	-.14	.16	.14	-.20	.01	.39[c]

Note. FD = formerly depressed; HC = healthy control; DAS = Dysfunctional Attitudes Scale; $_{DS}$ = difference score; VAS = Visual Analogue Scale; PANAS-X = Positive and Negative Affect Scale – Expanded Form; N = negative affect general dimension scale; P = positive affect general dimension scale; G = guilt basic negative emotional scale; S = sadness basic negative emotional scale; HP = heart period at baseline; $_5$ = average obtained during a five-minute interval; RSA = respiratory sinus arrhythmia; PEP = pre-ejection period; Z_{RES} = standardized residualized change scores; [c] = $p \leq .05$; [d] = $p \leq .01$; [e] = $p \leq .001$.

Table 28. *Correlation Matrices for Cognitive, Mood, and Cardiovascular Change Scores by Group and Condition*

					FD/SMI						
Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. DAS_{DS}											
2. VAS_{DS}	-.58[c]										
3. PANAS-X N_{DS}	-.20	.57[c]									
4. PANAS-X P_{DS}	.20	-.46	-.15								
5. PANAS-X G_{DS}	-.03	.22	.73[e]	-.15							
6. PANAS-X S_{DS}	-.21	.57[c]	.52[c]	-.65[d]	.59[d]						
7. HP_{DS5RA}	-.04	.39	-.33	-.47	.01	.24					
8. HP_{DS5RC}	-.09	-.41	.30	-.10	.61	.45	.16				
9. RSA_{DS5RA}	.25	-.21	.11	.24	-.10	.06	-.56	.32			
10. RSA_{DS5RC}	.38	-.38	.09	.34	-.01	-.11	-.87[d]	-.21	.67[d]		
11. PEP_{DS5RA}	.29	.28	-.48	-.28	-.44	-.09	.28	-.50	-.42	-.05	
12. PEP_{DS5RC}	-.25	-.10	-.62	-.40	-.19	.13	.13	-.10	.08	.16	.58
Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. Z_{RES}DAS											
2. Z_{RES}VAS	-.43										
3. Z_{RES}PANAS-X N	-.10	.52[c]									
4. Z_{RES}PANAS-X P	.13	-.49	-.11								
5. Z_{RES}PANAS-X G	-.10	.49[c]	.56[c]	-.57[c]							
6. Z_{RES}PANAS-X S	-.11	.19	.78[e]	-.11	.65[d]						
7. $Z_{RES5}HP_{RA}$	-.35	.37	-.34	-.53	.40	-.01					
8. $Z_{RES5}HP_{RC}$	-.40	-.45	.24	-.17	.50	.57	.10				
9. $Z_{RES5}RSA_{RA}$	.44	-.08	.01	.32	-.002	-.07	-.24	.26			
10. $Z_{RES5}RSA_{RC}$	.58[c]	-.29	.06	.46	-.004	.04	-.54	-.44	.65[d]		
11. $Z_{RES5}PEP_{RA}$	.21	.25	-.46	-.30	-.02	-.46	.32	-.49	-.53	-.21	
12. $Z_{RES5}PEP_{RC}$	-.17	-.13	-.56	-.35	.15	-.14	.16	.04	-.09	-.05	.56
					FD/NMI						
Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. DAS_{DS}											
2. VAS_{DS}	.14										
3. PANAS-X N_{DS}	.18	.32									
4. PANAS-X P_{D5}	.19	-.30	-.11								
5. PANAS-X G_{DS}	-.29	.10	.72[e]	-.07							
6. PANAS-X S_{DS}	.39	.29	.62[e]	-.26	.39						
7. HP_{DS5RA}	-.13	.79	-.20	-.78	-.54	.07					
8. HP_{DS5RC}	.85	.05	-.06	-.38	-.55	.60	.89[c]				
9. RSA_{DS5RA}	.11	.57[c]	.24	-.01	.21	.16	-.17	.16			
10. RSA_{DS5RC}	-.02	-.12	-.05	.21	.19	.003	-.63	-.46	.47		
11. PEP_{DS5RA}	.38	-.49	.32	.51	.15	-.09	-.70	-.46	-.35	.03	
12. PEP_{DS5RC}	.35	.16	-.06	.19	-.45	-.44	-.03	-.06	-.38	-.63	.77
Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. Z_{RES}DAS											
2. Z_{RES}VAS	.07										
3. Z_{RES}PANAS-X N	.06	.35									
4. Z_{RES}PANAS-X P	.22	-.28	-.09								
5. Z_{RES}PANAS-X G	.36	.29	.69[e]	-.17							
6. Z_{RES}PANAS-X S	-.26	.14	.62[d]	-.07	.34						
7. $Z_{RES5}HP_{RA}$	-.47	.69	-.13	-.77	.29	-.51					
8. $Z_{RES5}HP_{RC}$	.37	.15	-.25	-.36	.71	-.56	.88[c]				
9. $Z_{RES5}RSA_{RA}$	-.01	.58[c]	.09	.07	-.10	.12	.08	.10			
10. $Z_{RES5}RSA_{RC}$	-.02	-.15	-.28	.37	-.44	.05	-.41	-.34	.41		
11. $Z_{RES5}PEP_{RA}$	.55	-.19	.36	.52	-.06	.15	-.74	-.64	-.30	-.05	
12. $Z_{RES5}PEP_{RC}$	.39	.77	.25	.13	.05	-.39	-.13	-.22	-.12	-.31	.79

Table 28 Continued

HC/SMI

Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. DAS_{DS}											
2. VAS_{DS}	-.19										
3. PANAS-X N_{DS}	-.01	.03									
4. PANAS-X P_{DS}	.26	-.26	-.01								
5. PANAS-X G_{DS}	-.10	-.09	.53[e]	-.09							
6. PANAS-X S_{DS}	-.01	.40[d]	.42[d]	-.40[d]	.34[c]						
7. HP_{DS5RA}	.26	-.22	-.25	.55[c]	-.16	-.65[c]					
8. HP_{DS5RC}	-.03	-.15	-.23	.18	.44	-.68[d]	.53				
9. RSA_{DS5RA}	.14	.08	.01	.07	-.004	-.05	-.33	-.22			
10. RSA_{DS5RC}	.30	-.13	.12	.12	.18	-.16	.15	.46	.50[d]		
11. PEP_{DS5RA}	-.48	.53	-.05	-.27	.10	.05	-.14	.08	.23	.24	
12. PEP_{DS5RC}	-.31	.56[c]	-.49	.13	-.50	-.12	.27	.04	-.08	-.09	.63[c]

Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. Z_{RES}DAS											
2. Z_{RES}VAS	-.22										
3. Z_{RES}PANAS-X N	-.06	.10									
4. Z_{RES}PANAS-X P	.27	-.28	.02								
5. Z_{RES}PANAS-X G	-.12	.51[e]	.42[d]	-.41[d]							
6. Z_{RES}PANAS-X S	-.08	-.04	.52[e]	-.07	.34[c]						
7. $Z_{RES5}HP_{RA}$	.21	-.21	-.13	.55[c]	-.54	-.16					
8. $Z_{RES5}HP_{RC}$	.04	-.29	-.05	.32	-.65[c]	.52	.59[d]				
9. $Z_{RES5}RSA_{RA}$	.04	.17	-.05	-.01	-.02	-.07	-.31	-.50			
10. $Z_{RES5}RSA_{RC}$	.16	-.02	.09	.06	-.14	.16	.08	.28	.49[d]		
11. $Z_{RES5}PEP_{RA}$	-.64[c]	.60[c]	.08	-.29	.25	.10	-.16	.10	-.05	.11	
12. $Z_{RES5}PEP_{RC}$	-.28	.47	-.40	.27	-.02	-.53	.38	.04	.10	.06	.51

HC/NMI

Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. DAS_{DS}											
2. VAS_{DS}	-.20										
3. PANAS-X N_{DS}	.27	-.30									
4. PANAS-X P_{DS}	.29	-.30	.01								
5. PANAS-X G_{DS}	-.16	.13	.32	-.23							
6. PANAS-X S_{DS}	.03	-.16	.25	-.12	.15						
7. HP_{DS5RA}	-.32	.58[c]	-.51	-.25	.16	-.18					
8. HP_{DS5RC}	-.50	.18	.06	.04	.49	-.08	.34				
9. RSA_{DS5RA}	-.07	-.34	.17	-.27	-.04	-.14	-.20	-.15			
10. RSA_{DS5RC}	-.23	.07	-.04	-.28	-.13	-.02	-.06	.09	.48[d]		
11. PEP_{DS5RA}	.02	.06	.29	-.06	-.13	.21	-.29	.09	-.22	-.21	
12. PEP_{DS5RC}	.22	-.08	.52	.17	.36	.72[d]	-.13	.12	-.66[c]	-.32	.39

Measure	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. Z_{RES}DAS											
2. Z_{RES}VAS	-.27										
3. Z_{RES}PANAS-X N	.20	-.31									
4. Z_{RES}PANAS-X P	.36[c]	-.27	.04								
5. Z_{RES}PANAS-X G	-.04	-.002	.39[c]	-.17							
6. Z_{RES}PANAS-X S	-.10	.20	.40[c]	-.16	.13						
7. $Z_{RES5}HP_{RA}$	-.28	.38	-.41	-.21	-.12	.18					
8. $Z_{RES5}HP_{RC}$	-.59[c]	.07	.27	.11	.16	.49	.18				
9. $Z_{RES5}RSA_{RA}$	.03	-.30	.10	-.21	-.12	-.09	-.26	-.22			
10. $Z_{RES5}RSA_{RC}$	-.14	-.06	-.05	-.21	.01	-.13	-.12	.03	.47[d]		
11. $Z_{RES5}PEP_{RA}$	-.13	.15	.29	-.07	.21	-.14	-.28	.27	-.08	-.06	
12. $Z_{RES5}PEP_{RC}$	.10	-.08	.61[c]	.17	.74[d]	.46	-.01	.26	-.48	-.07	.35

Note. FD = formerly depressed; HC = healthy control; SMI = sad mood induction; NMI = neutral mood induction; DAS = Dysfunctional Attitudes Scale; $_{DS}$ = difference score; VAS = Visual Analogue Scale; PANAS-X = Positive and Negative Affect Scale – Expanded Form; N = negative affect general dimension scale; P = positive affect general dimension scale; G = guilt basic negative emotional scale; S = sadness basic negative emotional scale; HP = heart period at baseline; $_{5}$ = average obtained during a five-minute interval; RSA = respiratory sinus arrhythmia; PEP = pre-ejection period; Z_{RES} = standardized residualized change scores; [c] = $p \leq .05$; [d] = $p \leq .01$; [e] = $p \leq .001$.

CHAPTER 8

DISCUSSION

Vulnerability to depressive relapse and recurrence is difficult to delineate as it likely reflects a complex interaction of biological, psychological, and environmental factors. The majority of research on vulnerability to depressive relapse and recurrence has identified stable, unchangeable traits that are not amenable to modification (Burcusa & Iacono, 2007). Consequently, it is currently challenging for medical and mental healthcare providers to prevent or intervene during the depressogenic cycle. More research is needed to identify malleable vulnerability factors that can be specifically targeted during treatment or following treatment for relapse prevention to reduce the occurrence of future episodes of depression.

Four such factors that have been proposed as potential pathways of vulnerability to relapse and recurrence for formerly depressed individuals include cognitive, mood, and cardiovascular reactivity to and recovery from sadness. There is still disagreement in the cognitive and mood reactivity literature whether dysfunctional thinking patterns or dysphoric mood states characterize remitted MDD. While the literature has examined cardiovascular functioning in formerly depressed individuals, there are significant concerns about the quality and generalizability of studies focused on cardiovascular reactivity in response to sadness in remitted MDD due to multiple methodological issues. There have been no studies conducted to date that have focused on cardiovascular recovery from sadness in remitted MDD. The present study aimed to characterize cognitive, mood, and cardiovascular reactivity to and recovery from a sad mood induction in individuals with a history of depression compared to healthy, never depressed individuals.

The first aim of this study was to examine cognitive reactivity in response to an experimentally-induced sad mood in remitted MDD. Theories of depression have proposed that dysfunctional patterns of thinking represent a cognitive vulnerability that contributes to the etiology, maintenance, and reoccurrence of depression (Beck, 1967; Lau, Segal, & Williams, 2004; Scher, Ingram, & Segal, 2005). While these dysfunctional patterns of thinking have been observed in currently, but not formerly, depressed individuals (as reviewed by Teasdale, 1999), formerly depressed individuals continue to be at increased risk for depressive relapse and recurrence. Theoretical models, including the differential activation hypothesis by Teasdale (1988) and mood state dependent hypothesis by Miranda and Persons (1988), have proposed that cognitive vulnerabilities remain latent in formerly depressed individuals, are activated by dysphoric mood, and perpetuate depressed mood. Cognitive reactivity has been proposed as a predictor of relapse and recurrence.

A large body of literature has investigated cognitive reactivity in remitted MDD. Cross-sectional studies have generally shown that formerly depressed individuals exhibit cognitive reactivity in response to sadness compared to individuals without a history of depression (Gemar et al., 2001; Lau et al., 2012; Miranda et al., 1990, 1998; Roberts & Kassel, 1996; Van der Does, 2002). In addition, longitudinal studies have shown that formerly depressed individuals who exhibit cognitive reactivity while euthymic or dysphoric have higher rates of relapse and recurrence over time (Jarrett et al., 2012; Kuyken et al., 2010; Segal et al., 1999, 2006). However, there are inconsistencies in this literature base. More specifically, some studies have failed to find cross-sectional differences in cognitive reactivity in response to sadness between formerly depressed and never depressed participants (Brosse et al., 1999; Dykman, 1999; Fresco et al., 2006; Pfeiffer et al., 2015; Van der Does, 2005). In addition, one study has failed to find

that cognitive reactivity was predictive of relapse longitudinally (Lethbridge & Allen, 2008). The current study sought to contribute to this investigation and clarify prior inconsistent results. It was hypothesized that formerly depressed participants exposed to the sad mood induction would report significantly higher levels of dysfunctional beliefs on the DAS-SF II post-mood induction than formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

The second aim of this study was to examine mood reactivity in response to an experimentally-induced sad mood in remitted MDD. Theories of depression have proposed that differential affective responding to emotionally-valenced stimuli represent an affective vulnerability that contributes to the etiology and reoccurrence of depression (Rottenberg & Gotlib, 2004). While depression is typically conceptualized as a condition marked by sad, low mood, there do appear to be differences in the expression of both positive and negative emotions among currently depressed individuals (Rottenberg et al., 2005b). Theoretical models, including the positive attenuation hypothesis, the negative potentiation hypothesis, and the emotional context insensitivity hypothesis by Rottenberg and Gotlib (2004), have proposed that depression is marked by differential affective responding to emotionally-valenced stimuli that perpetuates depressive symptoms or leads to recurrence of depression. Much like cognitive vulnerabilities, it has been proposed that differential affective responding to emotionally-valenced stimuli may remain latent in formerly depressed individuals, be activated by dysphoric mood, and perpetuate depressed mood (Rottenberg et al., 2005b). Mood reactivity has been proposed as a predictor of relapse and recurrence.

A smaller body of literature has investigated mood reactivity in remitted MDD. Cross-sectional studies that principally focused on cognitive reactivity have failed to find differences in

mood reactivity among formerly depressed and never depressed participants (Brosse et al., 1999; Dykman, 1997; Fresco et al., 2006; Gemar et al., 2001; Lau et al., 2012; Miranda & Persons, 1988; Miranda et al., 1998; Solomon et al., 1998; Van der Does, 2002, 2005). Longitudinal studies have generally shown that formerly depressed individuals who exhibit blunted (i.e., decrease happiness in response to a sad mood induction; Lethbridge & Allen, 2008) or exaggerated (i.e., increased sadness in response to a sad mood induction; van Rijsbergen et al., 2013) mood reactivity are more likely to experience another depressive episode. However, this is a limited literature base that requires additional inquiry. More specifically, some studies have failed to find cross-sectional differences in mood reactivity in response to sadness between formerly depressed and never depressed participants (Brosse et al., 1999; Dykman, 1997; Fresco et al., 2006; Gemar et al., 2001; Lau et al., 2012; Miranda & Persons, 1988; Miranda et al., 1998; Solomon et al., 1998; Van der Does, 2002, 2005). In addition, only two longitudinal studies evaluating whether mood reactivity is predictive of relapse and recurrence have been conducted.

While there is currently disagreement in the literature about whether cognitive or mood reactivity are markers of vulnerability for relapse in remitted MDD, there is clear support for the notion that such vulnerabilities are mood state dependent in remitted depression. The current study sought to contribute to this investigation and clarify prior inconsistent results. It was hypothesized that formerly depressed and healthy control participants exposed to a sad mood induction would report significantly higher levels of dysphoric mood on the VAS post-mood induction than formerly depressed and healthy control participants exposed to the neutral mood inductions, with no significant differences in formerly depressed and healthy control participants exposed to the sad mood induction.

Psychological research has attempted to identify biological and physiological correlates of psychological conditions, rather than relying solely on subjective self-report measures. Individual differences in cardiovascular functioning have been observed in a variety of different psychological conditions, including current MDD (Chang et al., 2012; Ehrenthal et al., 2010; Jin et al., 2015; Kemp et al., 2010, 2012; Kikuchi et al., 2009; Liang et al., 2015; Nugent et al. 2011; Panaite et al., 2016; Rottenberg, 2007b; Rottenberg et al., 2003, 2005a, 2007a; Salomon et al., 2009; with the exception of Rottenberg et al., 2005b; Tsai et al., 2003). Theoretical models, including the polyvagal theory by Porges (1995), biopsychosocial model of challenge and threat by Blascovich and Tomaka (1996), and hawk-dove model by Smith (1982), have proposed that there is an association between behavioral, psychological, and physiological responding. These theories have been explored in currently depressed individuals but have not yet been adequately assessed in formerly depressed individuals. While it appears that the cardiovascular functioning of formerly depressed participants generally resembles that of healthy control participants at rest or in response to stress (Ahrens et al., 2008; Bylsma et al., 2014; Chang et al., 2013; Salomon et al., 2013; Vaccarino et al., 2008), it is plausible that the differences in cardiovascular functioning observed in currently depressed individuals may be mood state dependent in formerly depressed individuals.

The third aim of this study was to explore cardiovascular reactivity in response to an experimentally-induced sad mood in remitted MDD. A small body of literature has investigated cardiovascular reactivity in remitted MDD. While some cross-sectional studies have shown that formerly depressed individuals exhibited an abnormal pattern of cardiovascular reactivity in response to sadness compared to individuals without a history of depression (Yaroslavsky et al., 2013, 2014 Study 2), others have failed to find a significant difference among formerly

depressed and never depressed participants (Bylsma et al., 2015; Rottenberg et al., 2005b). As previously reviewed, there are significant concerns about the quality and generalizability of these due to multiple methodological issues (e.g., mixed sample of current and remitted depression, lack of assessment of CVD, limited range of cardiovascular measures assessed, lack of control group, limited investigation of adult-onset depression). The current study sought to advance this area of inquiry and address these methodological issues. It was hypothesized that formerly depressed individuals exposed to the sad mood induction would exhibit a maladaptive pattern of cardiovascular reactivity (i.e., decreased HP and RSA and increased PEP) during the mood induction compared to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

The fourth aim of this study was to explore cardiovascular recovery in response to an experimentally-induced sad mood in remitted MDD. Cardiovascular recovery provides an estimate of how long the physiological changes attributable to an emotionally-valenced stimulus persist after the stimulus has been removed. Research has suggested that cardiovascular recovery can result in the identification factors that contribute to the development of psychopathology and physiological abnormalities (Linden, Earle, Gerin, & Christenfeld, 1997; Haynes, Gannon, Orimoto, O'Brien, & Brandt, 1991). Unfortunately, none of the research on cardiovascular reactivity in remitted MDD has assessed cardiovascular recovery (Bylsma et al., 2015; Rottenberg et al., 2005b; Yaroslavsky et al., 2013, 2014 Study 2). The current study sought to establish this area of inquiry in remitted MDD. It was hypothesized that formerly depressed individuals exposed to the sad mood induction would exhibit reduced cardiovascular recovery (i.e., decreased HP, RSA, and CO and increased PEP compared to baseline) during the recovery

film compared to formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions.

Self-Report Measures

Formerly depressed participants reported significantly higher levels of current depressive symptoms on the BDI-II than healthy control participants. Formerly depressed participants reported a mean score of 9.91 out of 63 on the BDI-II, which suggests the presence of mild depressive symptoms. Healthy control participants reported a mean score of 3.32 out of 63 on the BDI-II, which suggests the presence of minimal depressive symptoms. Despite the presence of some residual depressive symptoms, none of the formerly depressed participants met diagnostic criteria for a major depressive episode within the last month while none of the healthy control participants met diagnostic criteria for a major depressive episode within their lifetime.

Formerly depressed and healthy control participants reported similar levels of state anxiety symptoms on the STAI-I. Formerly depressed participants reported a mean score of 44.53 out of 80 on the STAI-I while healthy control participants obtained a mean score of 45.74 out of 80 on the STAI-I. The scores reported by this sample are slightly above the proposed cut scores of 39 to 40 for clinically significant symptoms of state anxiety on the STAI-I (Addolorato et al., 1999; Knight, Waal-Manning, & Spears, 1983). While no formal predictions were set, the lack of significant difference between groups was surprising given the fact that state anxiety symptoms have been shown to highly correlate with measures of depression (Julian, 2011). Consequently, it would be reasonable to foresee that formerly depressed participants would have reported greater state anxiety symptoms on the STAI-I than healthy control participants given the fact that they reported significantly higher levels of depressive symptoms reported on the BDI-II.

Cognitive and Mood Measures

As expected, measures of cognition and mood assessed at different time points during the experimental paradigm were significantly correlated. More specifically, the DAS-SF I & II, VAS, and PANAS-X N, P, G, and S were significantly and positively correlated respectively when using pre- and post-mood induction measures. These findings suggested that while change in dysfunctional thoughts, dysphoric mood, and affective states were observed across the experimental paradigm for some measures, measures of cognition and mood continued to correlate throughout the experimental paradigm.

In addition, measures of cognition and mood were significantly correlated when using difference and residualized change scores. The DAS was positively correlated with the PANAS-X P and negatively correlated with the VAS. Of note, the valences of these correlations are in the expected direction as lower scores on the DAS are indicative of increased dysfunctional beliefs. The VAS was positively correlated with the PANAS-X N, G, and S and negatively correlated with the PANAS-X P. The PANAS-X N was positively correlated with the PANAS-X G and S while the PANAS-X P was negatively correlated with some measures of the PANAS-X G and S. Finally, the PANAS-X G was positively correlated with the PANAS-X S. These findings indicated an association between dysfunctional thoughts, dysphoric mood, and affective states across the experimental paradigm.

Correlations observed in the formerly depressed and healthy control groups were similar to those observed in the entire sample with one important exception; the pre- and post-mood induction VAS measures were not significantly correlated in the formerly depressed group, suggesting that there was a significant difference in dysphoric mood pre- and post-mood induction for formerly depressed participants. While this was true for formerly depressed

participants exposed to both the sad and neutral mood induction, the correlation for pre- and post-mood induction VAS measures was stronger for formerly depressed participants exposed to the neutral mood induction (r = .37, p > .05) compared to formerly depressed participants exposed to the sad mood induction (r = .16, p > .05). These findings indicated that all formerly depressed participants report a change in dysphoric mood pre- and post-mood induction, with a greater change reported by formerly depressed participants exposed to the sad mood induction.

Cognitive Reactivity

Contrary to expectations, there were no significant differences in cognitive reactivity on the DAS post-mood induction in formerly depressed participants exposed to the sad mood induction when using difference scores, residualized change scores, and repeated measures (see Figures 12 to 14). Post-hoc analyses revealed that the current study had a relatively low chance (13-36%) of detecting an effect due to insufficient power while sensitivity analyses indicated that at least a medium effect size was necessary to detect an effect if sufficient power had been obtained.

Figure 12. *Cognitive Reactivity – Difference Score Post-Mood Induction*

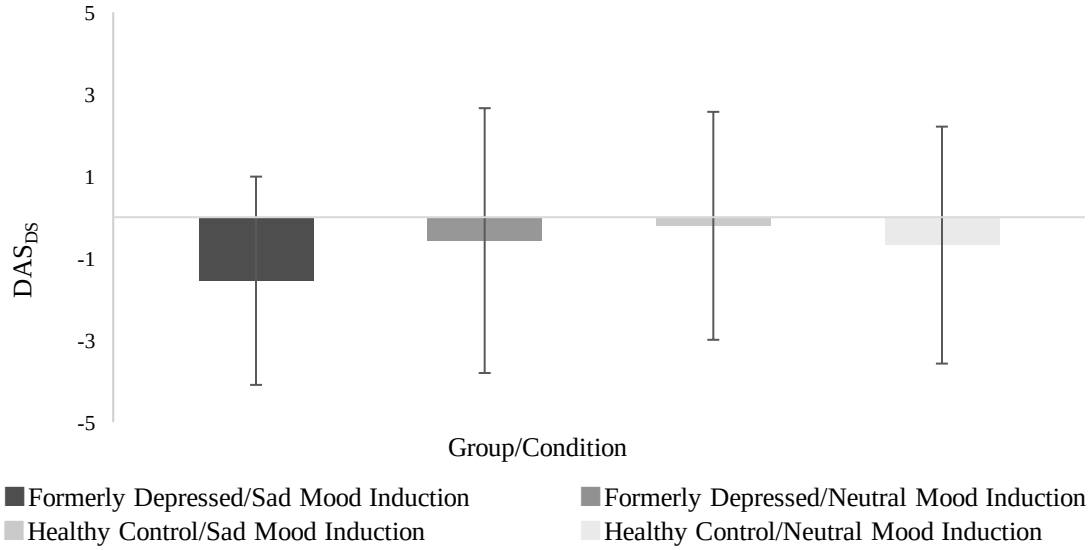


Figure 13. *Cognitive Reactivity – Residualized Change Score Post-Mood Induction*

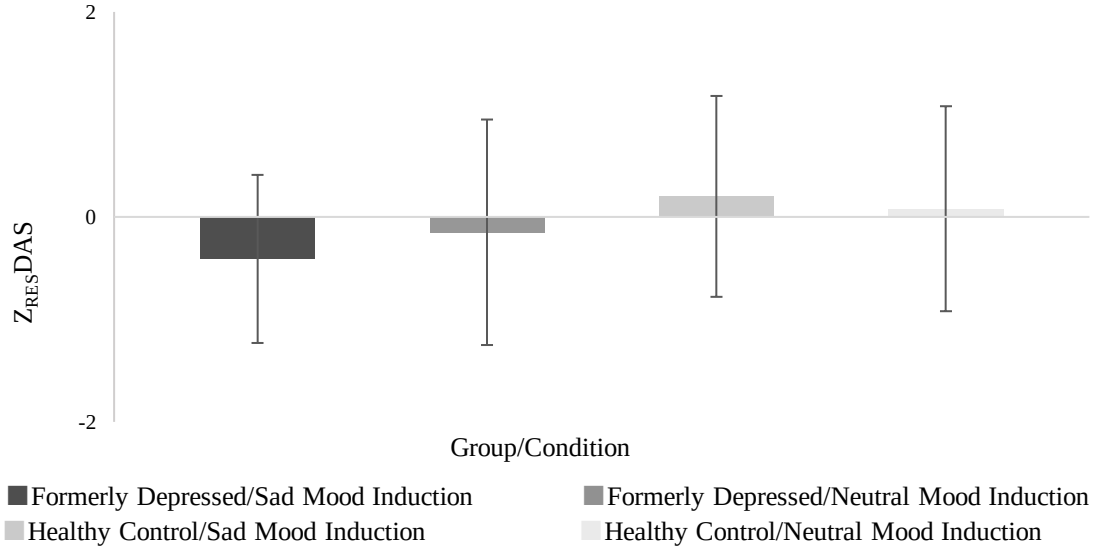
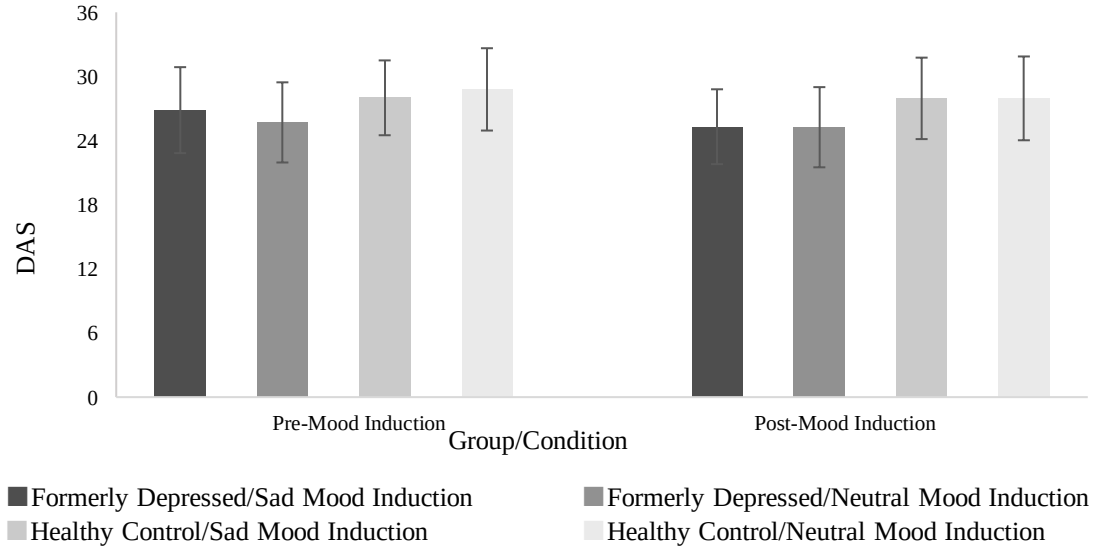


Figure 14. *Cognitive Reactivity – Repeated Measures Pre- and Post-Mood Induction*



However, there was one significant difference in cognitive reactivity that was identified using multiple comparisons. Formerly depressed participants reported significantly higher levels of dysfunctional beliefs on the DAS post-mood induction than healthy control participants when using residualized change scores. Of note, this measure can be counterintuitive to interpret as lower scores on the DAS are indicative of greater levels of the dysfunctional beliefs. This finding

is in line with the majority of the cognitive reactivity literature, which has found that formerly depressed participants report higher levels of dysfunctional beliefs in response to a transient sad mood compared to healthy controls (Gemar et al., 2001; Lau et al., 2012; Miranda et al., 1990, 1998; Roberts & Kassel, 1996; Van der Does, 2002). Notably, much of this research has elected to use residualized change scores rather than difference scores. In the current study, significant differences in cognitive reactivity may have been found between groups when using residualized change scores, but not difference scores, due to increased power with the former analysis. Analyses conducted with residualized change scores have more power due to smaller standard error and are therefore more likely to detect an effect (Castro-Schilo et al., 2018). In addition, research has shown that results can differ when analyses are conducted using difference scores and residualized change scores due to Lord's paradox, which postulates that this difference occurs when the pattern or lack of pattern of change differs between groups and when baseline differences on the predictor are stable, change equally, or change unequally (as reviewed by Castro-Schilo et al., 2018).

Contrast analyses indicated that formerly depressed participants exposed to the sad mood induction reported significantly higher levels of dysfunctional beliefs on the DAS post-mood induction compared to healthy control participants exposed to the sad mood induction and formerly depressed and healthy control participants exposed to the neutral mood induction when using residualized change scores. As previously mentioned, analyses conducted with residualized change scores have more power due to smaller standard error (Castro-Schilo et al., 2018) and the planned contrasts have the most power of all analyses as they only assess a circumscribed set of comparisons (Field, 2009). Therefore, it is questionable if the significant planned contrast results with residualized change scores but not difference scores and repeated measures is simply a

reflection of inflated type I error. The lack of consistent finding was unexpected as a significant proportion of the available research has found that formerly depressed individuals exhibit cognitive reactivity in response to sadness compared to healthy controls (Gemar et al., 2001; Lau et al., 2012; Miranda et al., 1990, 1998; Roberts & Kassel, 1996; Van der Does, 2002). However, there has been disagreement in the literature, with a subset of studies failing to find differences in cognitive reactivity in response to sadness based on depressive history (Brosse et al., 1999; Dykman, 1999; Fresco et al., 2006; Pfeiffer et al., 2015; Van der Does, 2005).

One potential reason for the current study's finding may be the measure that was employed. The DAS-SF I & II, an abbreviated version of the original DAS, was used to measure cognitive reactivity. The DAS-SF I & II was chosen over the DAS because it is significantly shorter (9-items versus 40-items) and the two measures have been shown to have similar psychometric properties. As previously noted, Beevers and colleagues (2007) found that there were no significant differences in residualized change scores for the DAS-A, DAS I, and DAS II (p = .79-.93, d = .00-.01) and the residualized change scores for the DAS-A, DAS I, and DAS II were very strongly correlated (r = .84-.91), suggesting that the long and short forms of the DAS perform similarly. The current study had the unique challenge of obtaining multiple self-report measures while participants were connected to psychophysiological equipment. The DAS-SF I & II was selected to minimize the amount of attentional demands, time, and movement needed to answer self-report measures as all of these factors can adversely impact psychophysiological recording. While none of the studies investigating cognitive reactivity have used the DAS-SF I & II, this is not because the measure is viewed unfavorably by depression researchers but rather, due to the fact that only four of these studies were conducted after the creation of the DAS in 2007. It is possible that different results may have been obtained using the DAS in the current

study as there may have been more variability with the 40-item measure. Additional research is needed to explore whether or not the DAS-SF I & II performs similarly to the DAS when assessing cognitive reactivity in a sample of formerly depressed participants.

Manipulation Check

As expected, participants exposed to the sad mood induction reported significantly higher levels of sadness on the VAS post-mood induction than participants exposed to the neutral mood induction when using difference scores, residualized change scores, and repeated measures. This change in pre- and post-mood induction measures (28.33%, increase of 28.33 points of 100-point scale) far exceeded the requirement of greater than 10.00% change in mood state that has been commonly used in the literature to indicate that a mood induction procedure has induced its intended mood state (Martin, 1990). In addition, participants exposed to the neutral mood induction did not exhibit a significant change in sadness (3.39%, increase of 3.39 points of 100-point scale) when assessing pre- and post-mood induction measures. Overall, these results suggest that the mood induction procedure successfully induced a transient sad mood in participants exposed to the sad mood induction and did not induce a transient sad mood in participants exposed to the neutral mood induction.

As expected, participants exposed to the sad mood induction reported significantly higher levels of sadness on the PANAS-X post-mood induction than participants exposed to the neutral mood induction when using difference scores and residualized change scores. While there were no significant differences between conditions when using repeated measures, it is possible that this is due to lower power in the between-subjects portion of this analysis (Guo, Logan, Glueck, & Muller, 2013). This change in pre- and post-mood induction measures aligns with the aforementioned changes in sadness on the VAS and corroborates the claim that the mood

induction procedures successfully induced a transient sad mood in participants exposed to the sad mood induction and did not induce a transient sad mood in participants exposed to the neutral mood induction.

As expected, there were no significant differences in reporting of positive affect, fear, and hostility on the PANAS-X between conditions when using difference scores, residualized change scores, and using repeated measures. While not significant due to the adjusted *p* value used due to violations of homogeneity of variance, participants exposed to the sad mood induction reported significantly higher levels of negative affect on the PANAS-X post-mood induction than participants exposed to the neutral mood induction when using residualized change scores but not difference scores or repeated measures. The PANAS-X negative affect general dimension scale contains the following affective states: afraid, scared, nervous, jittery, irritable, hostile, guilty, ashamed, upset, and distressed. It is possible that this finding was due to the fact that participants exposed to the sad mood induction reported elevations on the PANAS-X negative affect general dimension scale and PANAS-X guilt basic negative emotions scale, which both include some of the same affective states (i.e., guilty, ashamed). Overall, these results generally suggest that the mood induction procedure did not induce unintended general or specific affective status in participants exposed to both the sad and neutral mood inductions.

Contrary to expectations, participants exposed to the sad mood induction reported significantly higher levels of guilt on the PANAS-X post-mood induction than participants exposed to the neutral mood induction when using difference scores and residualized change scores. While there were no significant differences between conditions when using repeated measures, it is possible that this is due to lower power in the between-subjects portion of this analysis (Guo, Logan, Glueck, & Muller, 2013). Examination of the data indicated that this

pattern of responding was observed in formerly depressed participants exposed to the sad mood induction rather than healthy control participants exposed to the sad mood induction ($F(1,119) = 14.92$, $p < .001$, $\eta^2 = .11$). While it was not anticipated that emotions other than sadness would be reported, the presence of guilt makes intuitive sense given its association with the construct of depression. According to schema theory, which significantly influenced cognitive conceptualizations of depression, the depressogenic schema is associated with "themes of personal deficiency, worthlessness, self-blame, guilt, deprivation, and rejection" (Martin, 1990, p. 687). Excessive or inappropriate guilt is commonly experienced during a major depressive episode, insomuch as guilt is a symptom in the diagnostic criteria for MDD (APA$_A$, 2013) and included as a question in multiple clinician rating scales (e.g., Hamilton Rating Scale for Depression (HAM-D)) and self-report measures (e.g., BDI-II, PHQ-9, Center for Epidemiologic Studies of Depression Scale (CES-D), and Geriatric Depression Scale (GDS)) of depression.

Research has consistently found that the constructs of depression and guilt are associated with each other (as reviewed by Orth, Berking, & Burkhardt, 2006). A study by Ghatavi, Nicolson, MacDonald, Osher, and Levitt (2002) investigated whether guilt is state and/or trait dependent in depression. Participants included individuals with current MDD ($n = 34$), remitted MDD ($n = 22$), chronic cardiac illness ($n = 20$), and healthy control participants without a history of Axis I disorders ($n = 59$). Of note, individuals with chronic cardiac illness were recruited as a comparison group free from psychiatric conditions with "similar global functioning" (Ghatavi et al., p. 308). Results indicated that participants with current MDD reported significantly higher levels of state guilt than all other participants while participants with remitted MDD reported significantly higher levels of state guilt than cardiac and healthy controls. In addition, participants with current and remitted MDD reported similar levels of trait guilt, which were

significantly higher than cardiac and healthy controls. This study suggested that formerly depressed individuals experience elevated levels of state guilt compared to individuals without a history of depression as well as levels of trait guilt that are comparable to currently depressed individuals. In line with the differential activation and mood state dependent hypotheses, it is possible that elevations in state guilt observed in currently depressed individuals may remain latent in formerly depressed individuals until activated by a dysphoric mood. Additional research is needed to investigate whether or not guilt is mood state dependent in remitted MDD.

Mood Reactivity

As expected, participants exposed to the sad mood induction reported significantly higher levels of dysphoric mood on the VAS post-mood induction than participants exposed to the neutral mood induction when using difference scores, residualized change scores, and repeated measures. This finding was in line with the literature that used a combination of music and autobiographical recall and observed mood reactivity in all participants who were subjected to the sad mood induction condition (Fresco et al., 2006; Gemar et al., 2001; Jarrett et al., 2012; Kuyken et al., 2010; Lau et al., 2012; Pfeiffer et al., 2015; Segal et al., 1999, 2006; Van der Does, 2002, 2005). Additionally, this finding makes intuitive sense; engaging in an emotionally-valenced auditory and cognitive task induced the intended affective response.

Contrary to expectations, formerly depressed participants reported significantly higher levels of dysphoric mood on the VAS post-mood induction than healthy control participants when using difference scores, residualized change scores, and repeated measures. While the interaction between group and condition was not significant (see Figures 15-17), it is possible that this is due to lack of power rather than lack of effect. Examination of the means and standard deviations for the VAS pre- and post-mood induction measures, difference scores, and

residualized change scores do suggest that there are significant discrepancies in the levels of dysphoric mood reported by the different groups and conditions, with formerly depressed participants exposed to the sad mood induction reporting elevated post-mood induction scores on the VAS compared to all other groups. Examination of the *p* values (p = .08-.14) for difference scores and residualized change scores indicate that these analyses were approaching significance and may have been significant if sufficient power had been obtained. In addition, examination of the effect sizes (η^2 = .02-.03) for difference scores and residualized change scores indicate that they were in the small to medium range. While this was not true when using repeated measures, it is possible that this is due to lower power in the between-subjects portion of this analysis (Guo, Logan, Glueck, & Muller, 2013). Post-hoc analyses revealed that the current study had a relatively low chance (1-26%) of detecting an effect due to insufficient power while sensitivity analyses indicated that at least a medium effect size was necessary to detect an effect if sufficient power had been obtained.

Figure 15. *Mood Reactivity – Difference Score Post-Mood Induction*

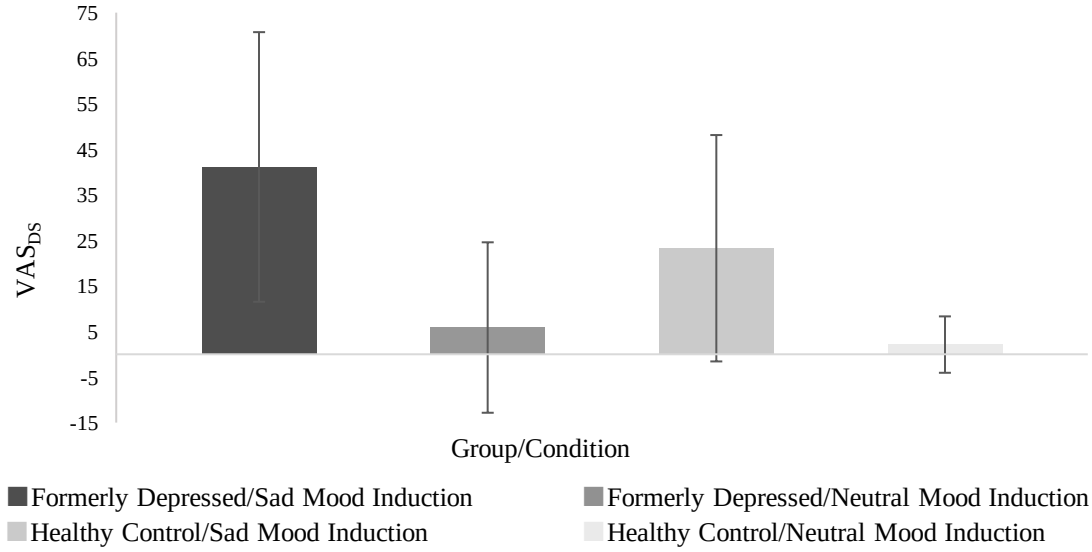


Figure 16. *Mood Reactivity – Residualized Change Score Post-Mood Induction*

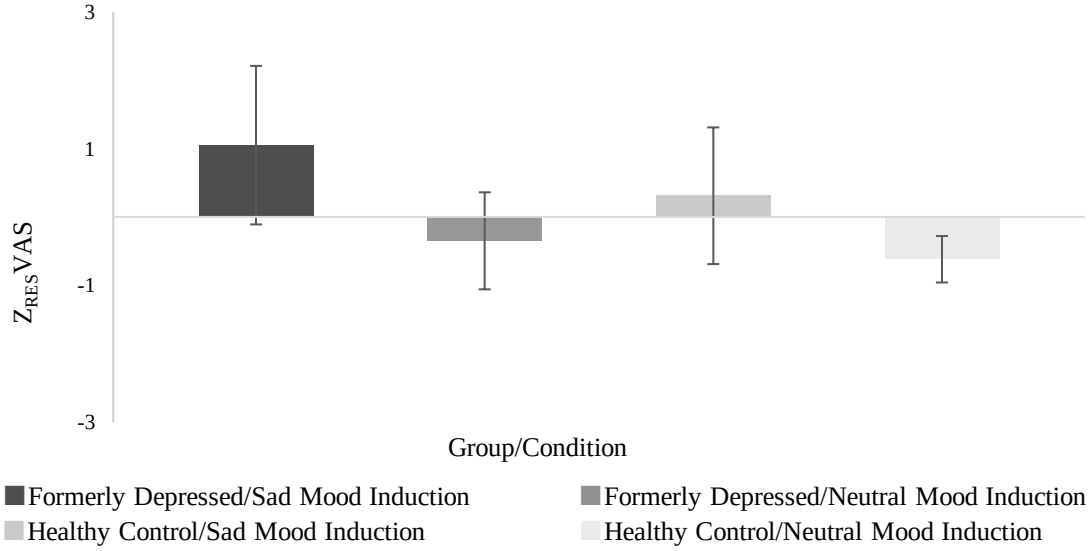
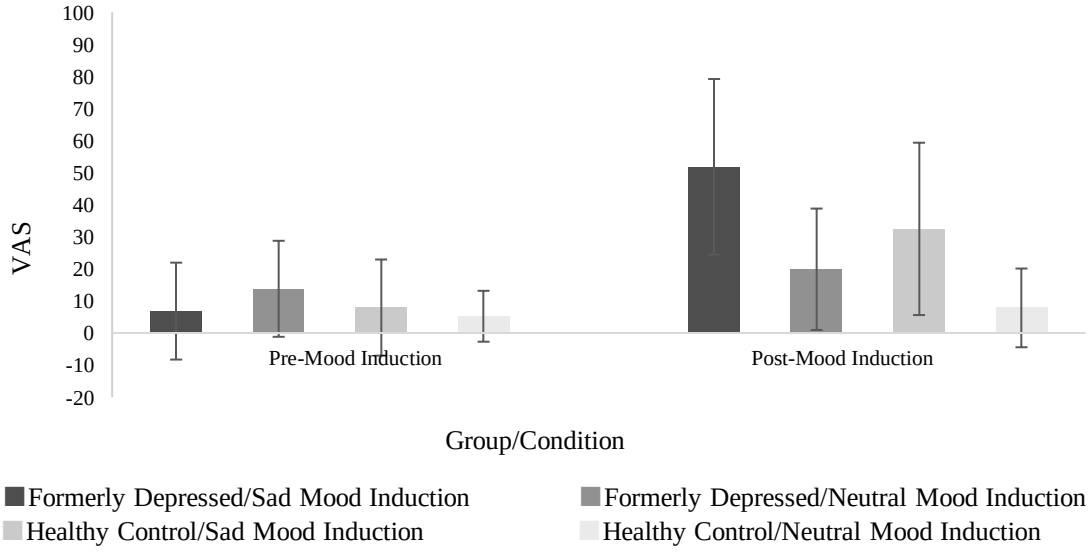


Figure 17. *Mood Reactivity – Repeated Measures Pre- and Post-Mood Induction*



Contrast analyses indicated that formerly depressed participants exposed to the sad mood induction reported significantly higher levels of dysphoric mood on the VAS post-mood induction compared to healthy control participants exposed to the sad mood induction and formerly depressed and healthy control participants exposed to the neutral mood induction when using difference scores, residualized change scores, and repeated measures. Of all analyses, the

planned contrasts have the most power as they only assess a circumscribed set of comparisons (Field, 2009). Unfortunately, these findings are challenging to clearly interpret as they are obfuscated by differing results obtained when disparate analytic techniques are employed and marginally insignificant results that are likely attributable to insufficient power.

Taken together, results generally appear to suggest that formerly depressed participants exposed to the sad mood induction exhibited elevated mood reactivity in response to sadness compared to healthy control participants exposed to the sad mood induction and formerly depressed and healthy control participants exposed to the neutral mood induction. This finding runs contrary to all of the cross-sectional studies that have failed to find any group differences in mood reactivity between formerly depressed and never depressed participants (Brosse et al., 1999; Dykman, 1997; Fresco et al., 2006; Gemar et al., 2001; Lau et al., 2012; Miranda & Persons, 1988; Miranda et al., 1998; Segal et al., 2006; Solomon et al., 1998; Van der Does, 2002, 2005).

There are a few potential reasons for the discrepant findings when comparing the current study to the literature base. First, a subset of the studies employed a different measure to evaluate mood reactivity (Multiple Affect Adjective Check List (MAACL); Dykman, 1997; Fresco et al., 2006; Miranda & Persons, 1988; Solomon et al., 1998). Second, the studies that employed the VAS or a similar Likert-scale mood rating measure (e.g., "not at all" for 0 to "extremely" for 10 rating of sadness without a visual representation of this rating system) have used a restricted range of potential scores (Gemar et al., 2001; Lau et al., 2012; Miranda et al., 1998; Segal et al., 2006; Van der Does, 2002, 2005). Only one study (Brosse et al., 1999) used the same version of the VAS employed in the current study, which obtained a rating of participants' current mood state on a 100-millimeter line anchored by "not at all" at 0 and "extremely" at 100. It is possible

that this version of the VAS captured a greater level of variability in participants' mood state and therefore, was more likely to identify differences than the other mood rating measures.

Cardiovascular Functioning

As expected, measures of cardiovascular functioning assessed at different time points during the experimental paradigm were significantly correlated. More specifically, five-minute HP, RSA, and PEP during baseline, mood induction, and recovery were significantly and positively correlated when using pre- and post-mood induction measures while five-minute HP, RSA, and PEP reactivity and recovery were significantly and positively correlated when using difference and residualized change scores. These findings suggested that while change in cardiovascular functioning was observed across the experimental paradigm for some measures, measures of cardiovascular functioning continued to correlate throughout the experimental paradigm.

Baseline Cardiovascular Functioning

As expected, there were no significant differences in baseline cardiovascular functioning for HP, RSA, or PEP assessed with both two- and five-minute averages between groups when using univariate analysis. The lack of significant differences in baseline cardiovascular functioning between groups is consistent with previous studies that have failed to find significant differences in cardiovascular functioning at rest among individuals with remitted MDD (Bylsma et al., 2014, 2015; Chang et al., 2013; Rottenberg et al., 2005b; Salomon et al., 2013; Vaccarino et al., 2008). In addition, this finding is in line with the differential activation and mood state dependent hypotheses, which suggests that vulnerabilities to depression remain latent in formerly depressed individuals and are only observable during a dysphoric mood state.

Cardiovascular Reactivity

Contrary to expectations, the hypothesized pattern of cardiovascular reactivity (i.e., decreased HP and RSA and increased PEP) was not observed in formerly depressed participants exposed to the sad mood induction when using difference scores, residualized change scores, and repeated measures. The hypothesized pattern of cardiovascular reactivity was based on a combination of theoretical models and previous empirical findings examining differences in cardiovascular reactivity in the response to sadness in formerly depressed individuals. In the current study sample, a different pattern of cardiovascular reactivity (i.e., increased HP and RSA and blunted PEP) in response to the sad mood induction emerged among formerly depressed participants, albeit without significant differences for the group by condition interaction. Examination of the effect size values indicated that effect sizes ranged from non-existent (η^2 = < .001) to in the small to medium range (η^2 = .03). Post-hoc analyses revealed that the current study had a relatively low chance (7-20%) of detecting an effect due to insufficient power while sensitivity analyses indicated that at least a medium effect size was necessary to detect an effect if sufficient power had been obtained. Clearly, the current study was under powered to detect such an effect across the different cardiovascular measures.

However, some significant differences in cardiovascular reactivity were identified using multiple comparisons. These differences were mainly found in HP, which is defined as the amount of time between heart beats measured in millisecond. HP was used in lieu of HR, which is defined as the number of beats produced by the heart per minute. While HP and HR are reciprocal measurements of cardiovascular functioning, they are not linearly related and can generate discrepant results when there are significant differences across participants or changes within participants. HP was selected because the current study hypothesized that changes in

cardiovascular functioning would be attributable to autonomic effects and cardiovascular differences were ascribed to the experimental task (condition: sad, neutral) and group membership (group: formerly depressed, healthy control; Berntson et al., 2007).

Results revealed that formerly depressed participants exhibited significantly higher levels of five-minute HP reactivity during the mood induction than healthy control participants when using difference scores and residualized change scores (see Figures 21-23). Of note, differences between groups were not observed when using two-minute HP reactivity (see Figures 18-20). While this was not true when using repeated measures for both two- and five-minute HP reactivity, it is possible that this is due to lower power in the between-subjects portion of this analysis (Guo, Logan, Glueck, & Muller, 2013).

Examination of the means and standard deviations for the two- and five-minute HP baseline and mood induction measures suggest that this is due to differences in resulting averages when using the two approaches to calculate reactivity. Both groups showed similar cardiovascular functioning at baseline. In addition, both groups showed a cardiovascular reaction in response to the mood induction. For the two-minute HP averages, formerly depressed participants exhibited an increase in HP of 26.74 milliseconds compared to baseline cardiovascular functioning during the mood induction while healthy control participants exhibited an increase in HP of 13.48 milliseconds during the mood induction compared to baseline cardiovascular functioning. For the five-minute HP averages, formerly depressed participants exhibited an increase in HP of 14.00 milliseconds during the mood induction compared to baseline cardiovascular functioning while healthy control participants exhibited a decrease in HP of 1.69 milliseconds during the mood induction compared to baseline cardiovascular functioning. Together, these findings suggest that the magnitude of cardiovascular

reactivity differed when using two-minute HP averages and the extent to which cardiovascular reactivity attenuated over time differed when using five-minute HP averages.

Figure 18. *Two-Minute Heart Period – Difference Score during Mood Induction*

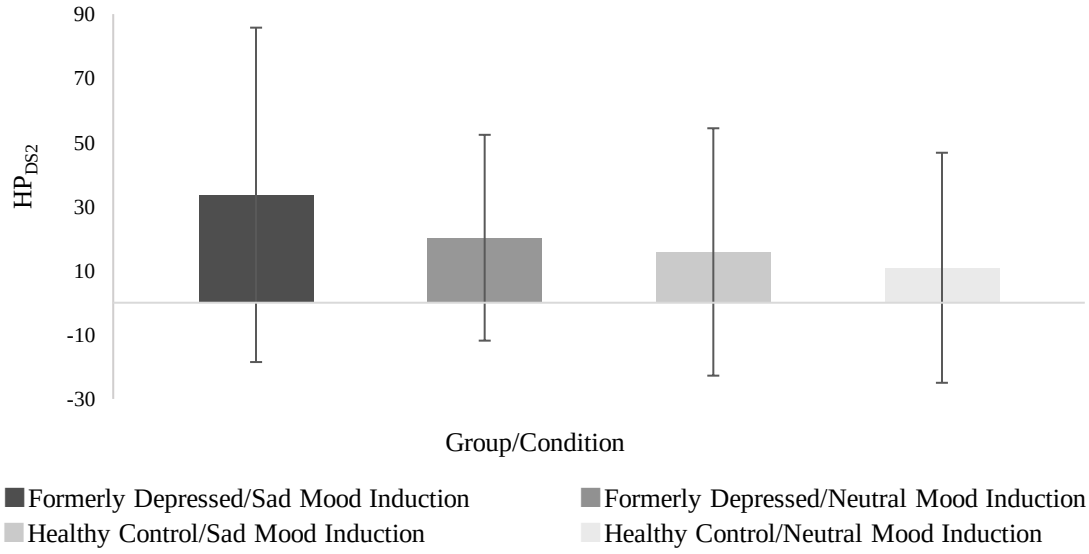


Figure 19. *Two-Minute Heart Period – Residualized Change Score during Mood Induction*

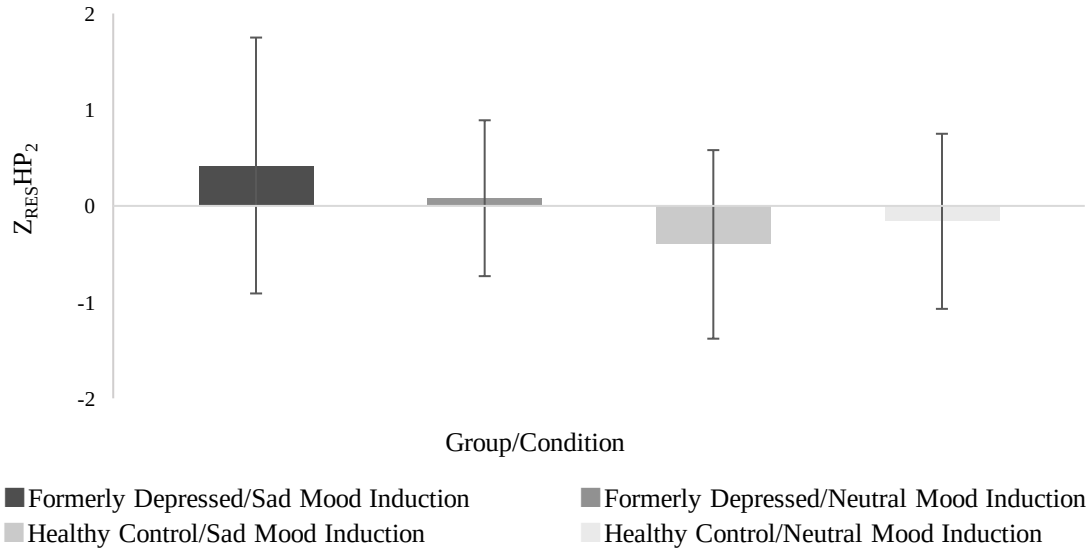


Figure 20. *Two-Minute Heart Period – Repeated Measures Pre- and during Mood Induction*

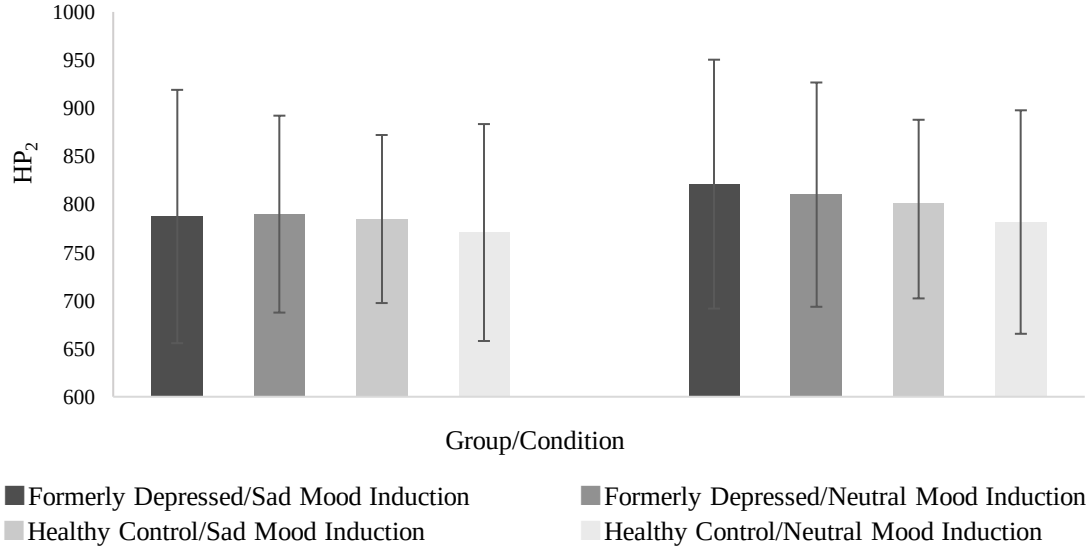


Figure 21. *Five-Minute Heart Period- Difference Score during Mood Induction*

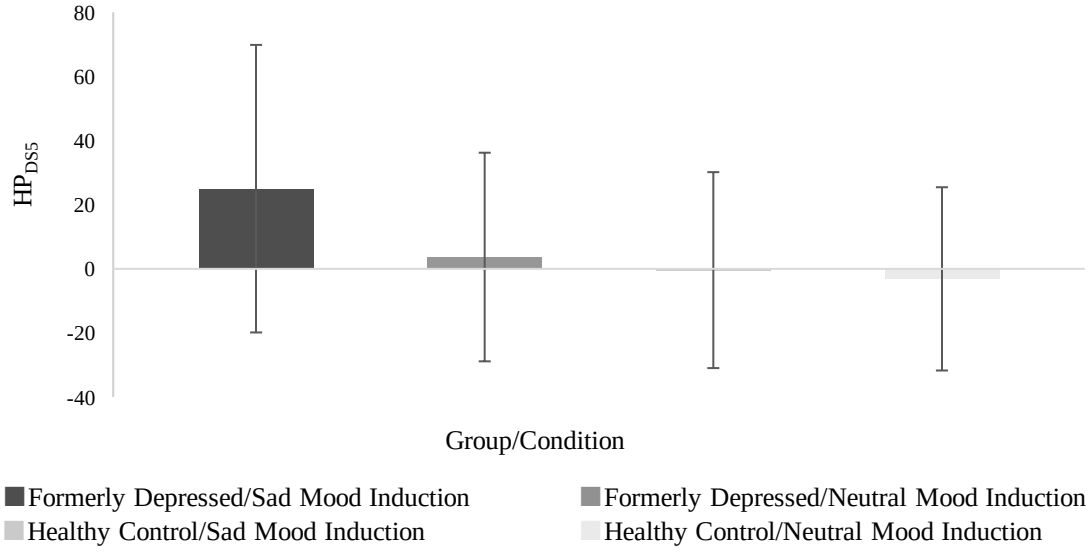


Figure 22. *Five-Minute Heart Period – Residualized Change Score during Mood Induction*

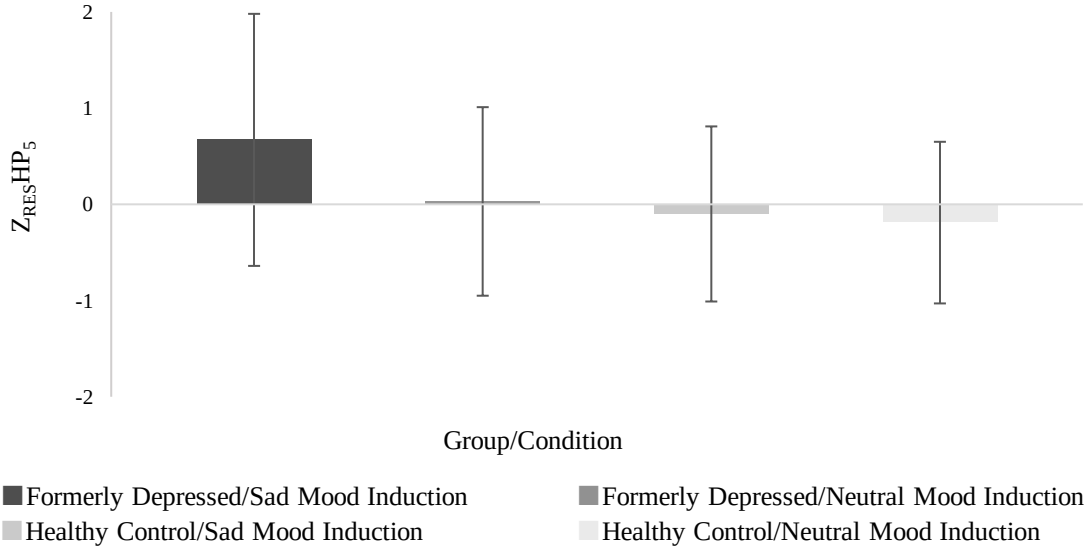
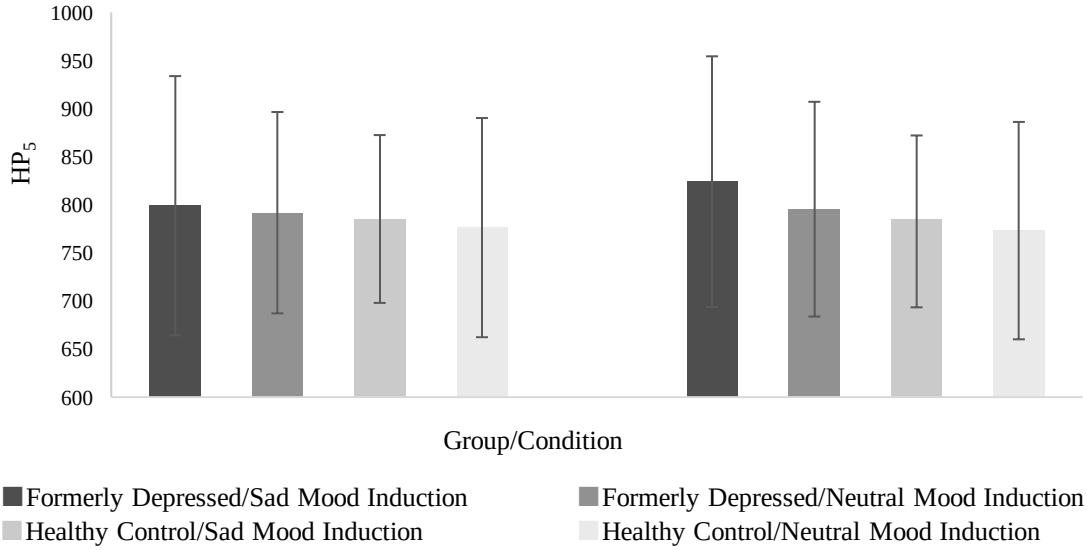


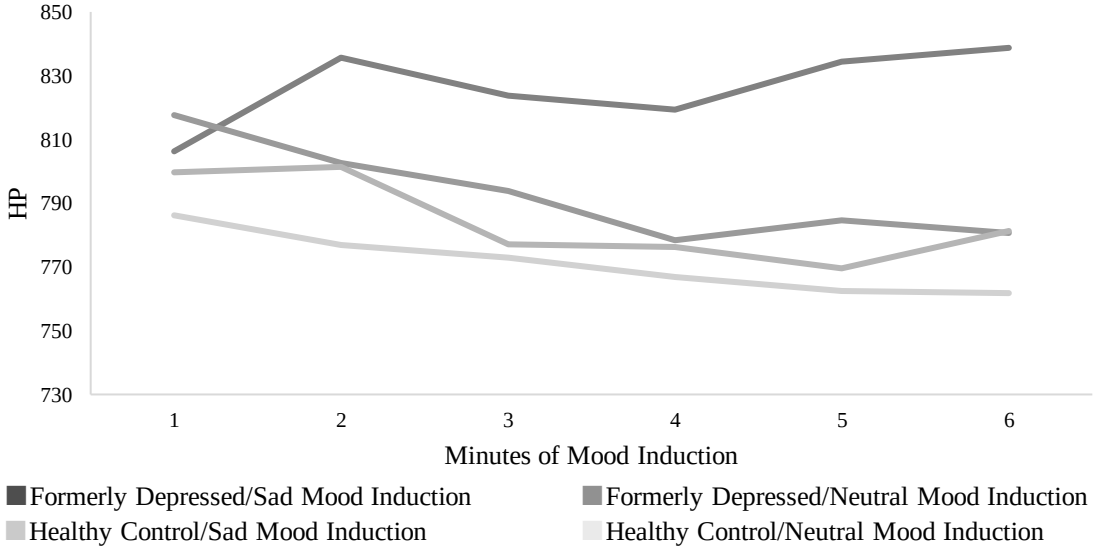
Figure 23. *Five-Minute Heart Period – Repeated Measures Pre- and during Mood Induction*



Multiple comparisons and contrast analyses were not significant for HP, RSA, or PEP. While not statistically significant, examination of the *p* values (*p* = .06-.08) for the contrast analyses for two-minute HP difference scores and residualized change scores and five-minute HP residualized change scores indicate that these analyses were approaching significance and may have been significant if sufficient power had been obtained. While this was not true when using

repeated measures for both two- and five-minute HP reactivity contrast analyses, it is possible that this is due to lower power in the between-subjects portion of this analysis (Guo, Logan, Glueck, & Muller, 2013). Both of these findings are meaningful. The difference in magnitude of cardiovascular reactivity for two-minute HP suggests that when observed over a short period of time, formerly depressed individuals exposed to a sad mood induction may exhibit more pronounced HP reaction compared to healthy control individuals exposed to a sad mood induction as well as formerly depressed and healthy control individuals exposed to a neutral mood induction (see Figure 24). The extent to which cardiovascular reactivity attenuated over time significantly differed for five-minute HP, which suggests that when observed over a more extended period of time, formerly depressed individuals exposed to a sad mood induction may exhibit an elevated HP in response to an emotionally-valenced stimulus compared to healthy control individuals exposed to a sad mood induction as well as formerly depressed and healthy control individuals exposed to a neutral mood induction (see Figure 24). The latter finding points to importance of the remitted MDD literature moving beyond its persistent focus on cardiovascular reactivity to additionally investigate differences in cardiovascular recovery.

Figure 24. *Minute by Minute Heart Period during Mood Induction*



Cardiovascular Recovery

Contrary to expectations, the expected maladaptive patterns of cardiovascular recovery (i.e., decreased HP and RSA and increased PEP compared to baseline) was not observed during the recovery film in formerly depressed participants exposed to the sad mood induction when using difference scores, residualized change scores, and repeated measures. The hypothesized pattern of cardiovascular recovery was also based on a combination of theoretical models and previous empirical findings examining differences in cardiovascular recovery in response to stress among formerly depressed individuals given the lack of investigation of cardiovascular recovery in the response to sadness in formerly depressed individuals. In the current study sample, a different pattern of cardiovascular recovery (i.e., increased HP and RSA and blunted PEP) during the recovery film emerged among formerly depressed participants, albeit without significant differences the group by condition interaction for RSA and PEP. Examination of the effect size values indicated that effect sizes ranged from non-existent (η^2 = < .001) to small to medium (η^2 =.05). Post-hoc analyses revealed that the current study had a relatively low chance

(6-28%) of detecting an effect due to insufficient power while sensitivity analyses indicated that at least a medium effect size was necessary to detect an effect if sufficient power had been obtained. The current study was under powered to detect such an effect in some of the cardiovascular measures.

However, some significant differences in cardiovascular recovery were identified using multiple comparisons. While not significant due to the adjusted *p* value used due to violations of homogeneity of variance, formerly depressed participants exhibited significantly higher levels of two-minute HP during the recovery film when using difference and residualized change scores as well as five-minute HP during the recovery film when using residualized change scores than healthy control participants. The difference in magnitude of cardiovascular recovery was not large enough to reach statistical significance for five-minute HP recovery using difference scores. Examination of the means and standard deviations for the baseline and recovery measures and difference score do suggest that there are discrepancies in HP recovery exhibited by the different groups. Examination of the *p* values (p = .02-.05) indicate that this analysis was approaching significance and may have been significant if sufficient power had been obtained. In addition, examination of the effect sizes (η^2 = .04-.06) for the two-minute HP difference scores and residualized change scores and five-minute HP residualized change scores indicate that they were in the small to medium and medium range.

While not significant due to the adjusted *p* value used due to violations of homogeneity of variance, formerly depressed participants exposed to the sad mood induction exhibited significantly higher levels of two-minute HP during the recovery film when using difference and residualized change scores than formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions (see

Figures 25-27). In addition, formerly depressed participants exposed to the sad mood induction exhibited significantly higher levels of five-minute HP during the recovery film when using residualized change scores than formerly depressed participants exposed to the neutral mood induction and healthy control participants exposed to the sad and neutral mood inductions (see Figures 28-30). While five-minute HP recovery using difference scores did not meet statistical significance, examination of the *p* values ($p = .06$) indicated that these analyses were approaching significance and may have been significant if sufficient power had been obtained. In addition, examination of the effect size ($\eta^2 = .04$) for the five-minute HP difference score indicate that they were in the small to medium range. While there were no significant differences between groups and conditions when using repeated measures, it is possible that this is due to lower power in the between-subjects portion of this analysis (Guo, Logan, Glueck, & Muller, 2013).

Figure 25. *Two-Minute Heart Period during Recovery Film*

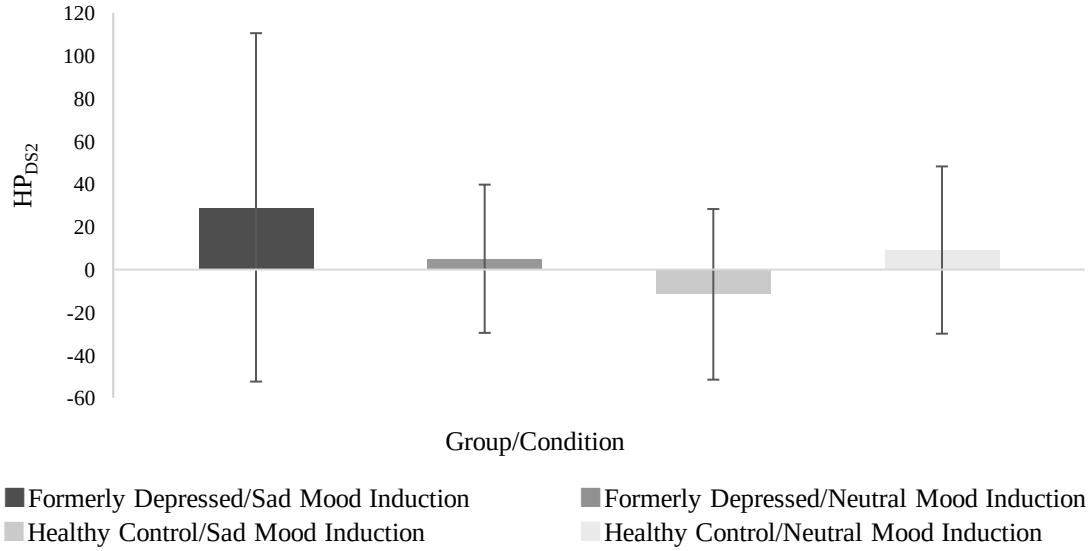


Figure 26. *Two-Minute Heart Period – Residualized Change Score during Recovery Film*

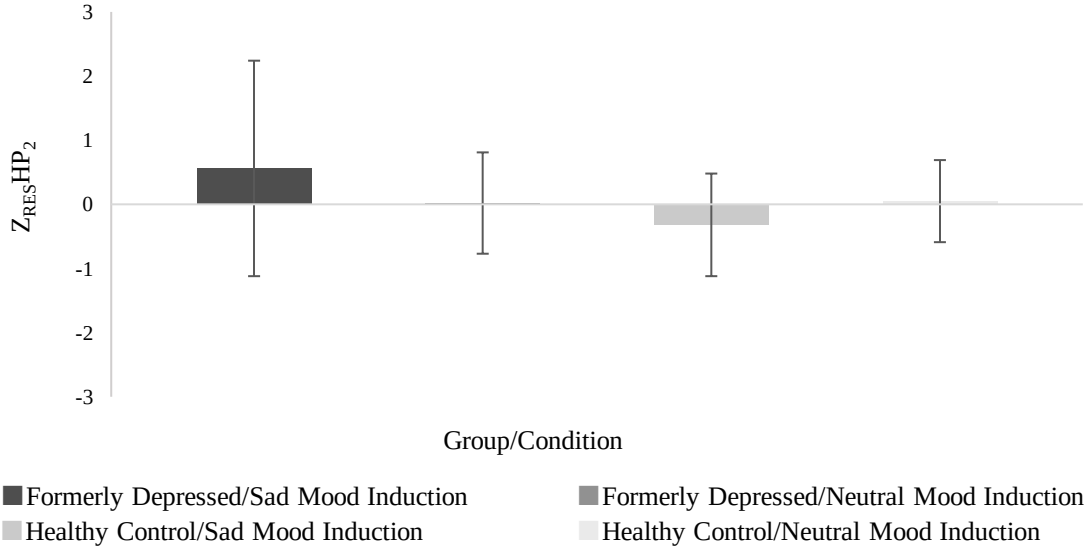


Figure 27. *Two-Minute Heart Period – Repeated Measures Pre- and during Recovery Film*

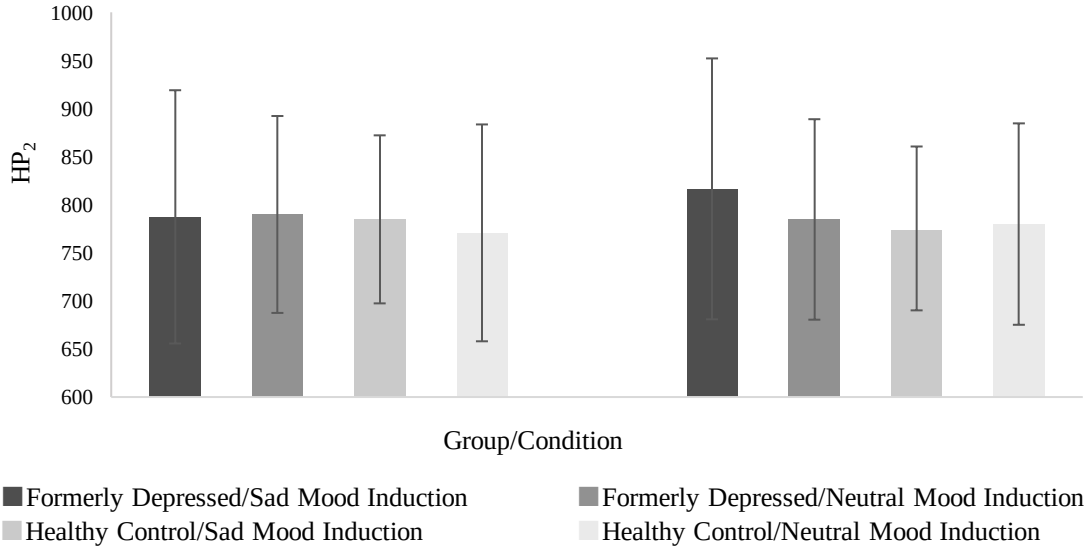


Figure 28. *Five-Minute Heart Period during Recovery Film*

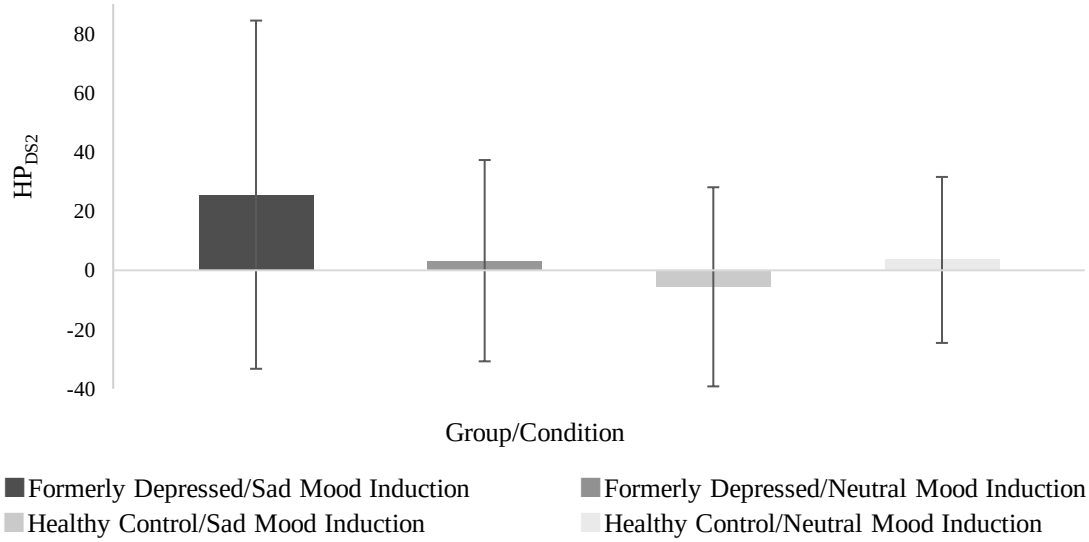


Figure 29. *Five-Minute Heart Period – Residualized Change Score during Recovery Film*

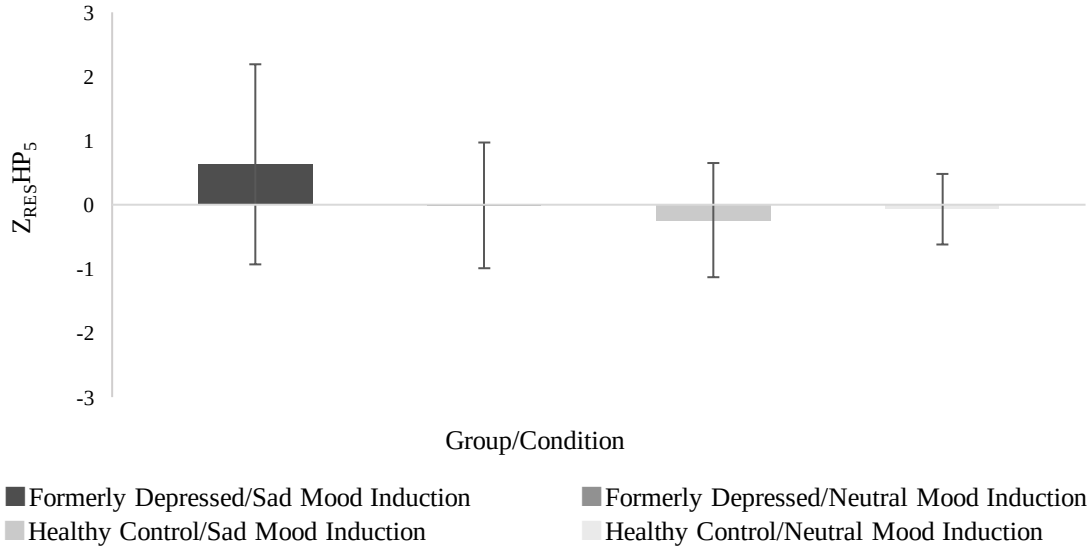
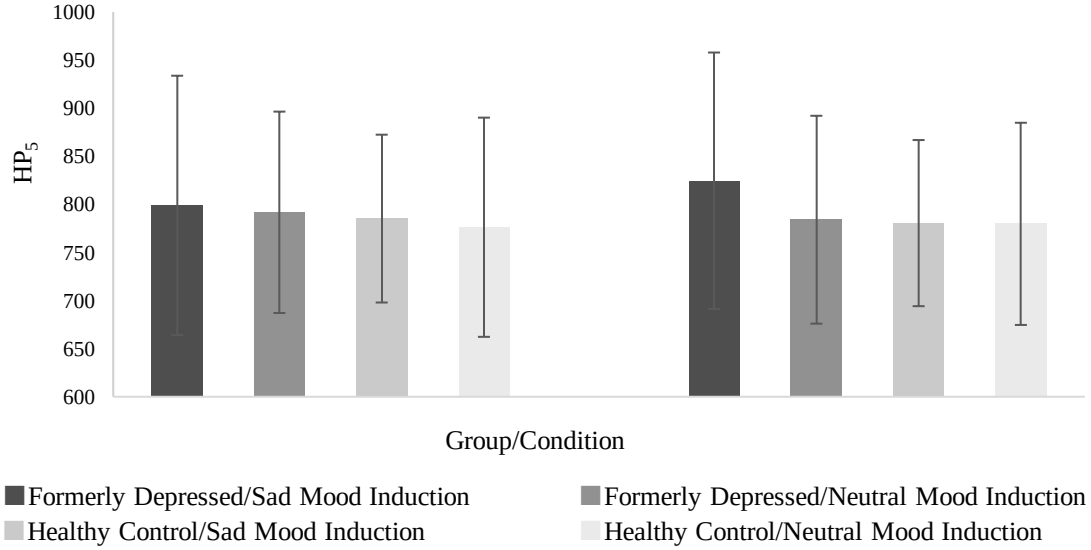


Figure 30. *Five-Minute Heart Period – Repeated Measures Pre- and during Recovery Film*



Contrast analyses were not significant for RSA and PEP but were for some HP measures. Contrast analyses indicated that formerly depressed participants exposed to the sad mood induction exhibited significantly higher levels of two-minute HP when using residualized change scores and five-minute HP when using difference scores and residualized change scores during the recovery film compared to healthy control participants exposed to the sad mood induction and formerly depressed and healthy control participants exposed to the neutral mood induction. While this was not true when using repeated measures for both two- and five-minute HP recovery, it is possible that this is due to lower power in the between-subjects portion of this analysis (Guo, Logan, Glueck, & Muller, 2013).

Examination of the means and standard deviations for the two- and five-minute HP baseline and recovery measures suggest that the two approaches to calculating recovery resulted in slightly different findings. Both groups showed similar cardiovascular functioning at baseline. However, there was a stark difference in cardiovascular recovery obtained during the recovery film. For the two-minute HP averages, formerly depressed participants exposed to the sad mood

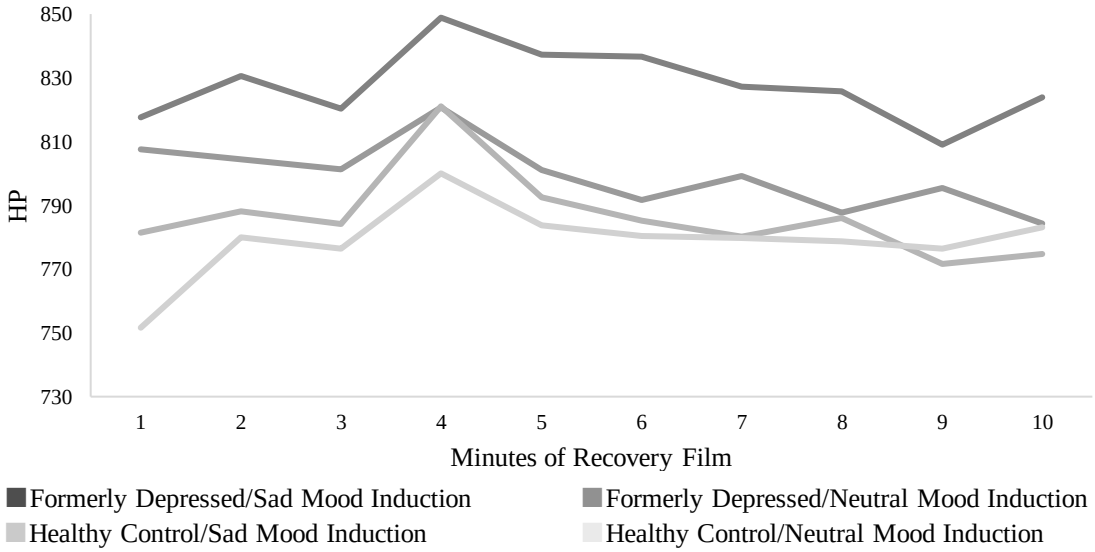
induction exhibited an increase in HP of 29.11 milliseconds during the recovery film compared to baseline cardiovascular functioning. While formerly depressed and healthy control participants exposed to the neutral mood induction did not return to baseline cardiovascular functioning levels, their HP during the recovery film was significantly lower than formerly depressed participants exposed to the sad mood induction. Interestingly, healthy control participants exposed to the sad mood induction actually exhibited HP levels that were lower than their baseline cardiovascular functioning levels during the recovery film.

For the five-minute HP averages, formerly depressed participants exposed to the sad mood induction exhibited an increase in HP of 25.00 milliseconds during the recovery film compared to baseline cardiovascular functioning. Formerly depressed and healthy control participants exposed to the neutral mood induction showed a decrease of HP compared to their two-minute HP averages that was closer to their baseline cardiovascular functioning levels. Healthy control participants exposed to the sad mood induction continued to exhibit HP levels that were lower than their baseline cardiovascular functioning levels during the recovery film, albeit to a lesser degree. Together, this suggests that the magnitude of cardiovascular recovery differed when comparing two- and five-minute HP averages and the extent to which cardiovascular recovery attenuated over time differed across groups and conditions when using two- and five-minute HP averages.

Both of these findings are meaningful. The difference in attenuation of cardiovascular recovery for two-minute HP suggests that when observed over a short period of time, formerly depressed individuals exposed to a sad mood induction may exhibit less reduction in HP during recovery compared to healthy control individuals exposed to a sad mood induction as well as formerly depressed and healthy control individuals exposed to a neutral mood induction (see

Figure 31). The difference in attenuation of cardiovascular recovery for five-minute HP suggests that when observed over an extended period of time, formerly depressed individuals exposed to a sad mood induction may continue to exhibit elevated HP during recovery compared to healthy control individuals exposed to a sad mood induction as well as formerly depressed and healthy control individuals exposed to a neutral mood induction (see Figure 31). Together, these findings suggest that cardiovascular recovery following a transient sad mood is impaired among formerly depressed individuals when examined using both two- and five-minute HP averages.

Figure 31. *Minute by Minute Heart Period during Recovery Film*



Implications

Several important implications can be drawn from the current study. The finding that generally, formerly depressed individual exposed to the sad mood induction experienced elevated levels of mood, rather than cognitive, reactivity provides a meaningful data point in the inconsistent literature base. Empirical evidence has found that compared to healthy control participants, those with remitted MDD report significant increases in dysfunctional beliefs (i.e., cognitive reactivity; Kuyken et al., 2010; Segal et al., 1999, 2006) or dysphoric mood (i.e., mood

reactivity; van Rijsbergen et al., 2013) in response to an experimentally-induced sad mood and prospectively predict relapse of depression over time. There has been disagreement in the literature whether dysfunctional thinking patterns or dysphoric mood states characterize remitted MDD, with recent research advancing the idea that mood reactivity may be an important construct of interest. While the current study sought to add clarity to the literature, results raise questions about what may be driving the different findings between studies. Additional research is needed to advance our understanding of these potentially malleable vulnerability factors associated with a history of depression, whether it be identification of mediators, moderators, or predictors of cognitive and mood reactivity.

In terms of treatment, the finding that formerly depressed individual exposed to the sad mood induction experienced elevated levels of mood reactivity may have important implications for psychotherapy. The evidence-based treatments for depression that are currently recognized by Division 12 of the American Psychological Association (APA$_B$, 2016a) include the following 13 treatment modalities: acceptance and commitment therapy (ACT), behavioral activation, cognitive behavioral analysis system of psychotherapy, CBT, CT, emotion focused therapy, interpersonal psychotherapy, problem-solving therapy, rational emotive behavioral therapy (REBT), reminiscence/life review therapy, self-management/self-control therapy, self-system therapy, and short-term psychodynamic therapy. Of note, the majority of the second and third wave therapies listed above are theorized to impact emotions indirectly. More specifically, the cognitive model that second wave psychotherapies (i.e., CBT, CT, REBT) are based on theorize that an individual's perception of an event results in automatic thoughts that spurs a cascade of behavioral, emotion, and physiological responses (Beck, 1964). Consequently, the cognitive model postulates that restructuring negative automatic thoughts, as well as intermediate and core

beliefs further along during the course of therapy, changes behavioral and emotional reactions (Beck, 2011). In other words, traditional cognitive-behavioral approaches to psychotherapy result in alterations of emotional responses through indirect techniques.

Third wave psychotherapies (e.g., ACT, dialectical behavior therapy (DBT), mindfulness-based cognitive therapy (MBCT)) are more closely connected by the techniques utilized rather than theoretical underpinning. Third wave therapies typically employ mindfulness strategies to increase awareness and acceptance of internal experiences including thoughts, feelings, and physiological sensations (Brown, Gaudiano, & Miller, 2013). In addition, some of these psychotherapies employ emotion-focused techniques. For example, DBT, an empirically-supported treatment for borderline personality disorder (BPD; Division 12 of the APA$_B$, 2016b) that traditionally consists of individual psychotherapy, group-based skills training, phone consultation, and team consultation (Linehan, 1993), focuses on building emotion regulation, distress tolerance, mindfulness, and interpersonal effectiveness skills. The emotion regulation module includes skills such as understanding and identifying emotions, changing undesirable emotions, reducing vulnerability to emotions, and managing intense emotions. The distress tolerance module includes skills such as crisis survival and radical acceptance of emotional reactions (Linehan, 2015).

While individuals with MDD and BPD exhibit different deficits in emotion regulation, it is possible that the emotion regulation and distress tolerance skills that are integral to DBT may be beneficial to formerly depressed individuals. Theoretical and empirical evidence suggests that vulnerability associated with depression remain latent in individuals who have recovered from a depressive episode until activated by dysphoric mood. This dysphoric mood is hypothesized to be exacerbated over time, to the point of resulting in a depressive relapse or recurrence. It is

possible that providing individuals with remitted MDD emotion regulation and distress tolerance skills at the final phase of treatment initially targeting current MDD or during booster sessions for relapse prevention may increase their ability to recognize when a dysphoric mood occurs and select the appropriate strategies (e.g., mindfulness, problem solving, opposite action, cognitive restructuring; Linehan, 2005) to manage these emotions effectively and prevent the onset of a prolonged depressed mood. Research is needed to empirically test whether or not teaching these skills to at-risk remitted MDD populations could result in reductions in depressive relapse and recurrence over time.

While there were no significant differences in cardiovascular reactivity when examining the group by condition interaction, formerly depressed individuals generally exhibited a trend of elevated levels of HP and reduced attenuation of HP during the sad mood induction when using planned comparisons. As previously noted, HP has been used in the literature as an index of arousal, task involvement, and mental load and effort (Jorna, 1992). This finding suggests that formerly depressed individuals may experience elevated arousal during a sad mood and be more engaged in and devote more cognitive resources to the mood induction task. While not examined in the current investigation, one potential mechanism of action that may be explored to explain increased task involvement and mental load and effort in formerly depressed individuals during a sad mood induction is rumination given its prevalence in this population (Olatunji, Naragon-Gainey, & Wolitzky-Taylor, 2013). Importantly, this finding points to potential differences in cardiovascular recovery in this population. The current study makes an important contribution to the literature as it was the first to investigate cardiovascular recovery following a sad mood induction in formerly depressed participants.

The finding that generally, formerly depressed individual exposed to the sad mood induction exhibited elevated levels of HP during the recovery film greatly extends the current literature. A review of the remitted MDD literature indicates that only two studies have assessed cardiovascular reactivity in formerly depressed individuals, albeit in response to stress rather than sadness. Both of these studies suggested that formerly depressed individuals exhibited cardiovascular recovery following a speech stress test that resembled healthy control, rather than currently depressed, individuals (Bylsma et al., 2014; Salomon et al., 2009). Thus, this was the first study to investigate multiple measures of cardiovascular recovery from a transient sad mood in formerly depressed individuals. Using planned comparisons, the current study found that individuals with a history of depression take significantly longer to return to baseline levels of HP following a sad mood induction when using both two- and five-minute averages compared to healthy, never depressed individuals. This finding is striking as the study's sample consisted of younger (M = 20.79, SD = 5.65), healthy individuals free from a variety of physical and mental health conditions that could have confounded the results. It is possible that formerly depressed individuals who are older, have experienced more depressive episodes over the course of their lifetime, and have comorbid physical and mental health conditions would exhibit a more pronounced cardiovascular response to a transient sad mood or experience more negative repercussions due to impaired HP recovery.

Results suggest that formerly depressed individuals exposed to a sad mood induction experience elevated arousal during recovery and have more difficulty disengaging from a task. While not examined in the current investigation, one potential mechanism of action to explain the lasting impact of a sad mood induction on formerly depressed individuals is continued

rumination during the recovery period. Future research could investigate this possibility by asking participants to complete a state measure of rumination following the recovery period.

The lasting impact of a transient sad mood on formerly depressed individuals may explain the susceptibility to CVD in this population. Higher HP during experimental tasks that involve attention is believed to be adaptive, as it is thought to represent arousal, task involvement, and mental load and effort. However, elevated HP during recovery may not be adaptive, especially if this persists for extended periods of time or occurs with regular frequency. The closest clinical variable that could serve as a proxy for elevated HP during recovery is resting HR, or the number of beats produced by the heart per minute while completely at rest. Research has indicated that elevated resting HR is associated with an increased risk of negative health outcomes, including cardiovascular events, CVD, and mortality, in individuals with and without pre-existing cardiovascular problems (Ho et al., 2014). A recent meta-analysis by Zhang, Shen, and Qi (2015) investigated the relationship between resting heart rate and all-cause and cardiovascular mortality. 46 prospective cohort studies met the inclusion and exclusion criteria, resulting in data from over 1,000,000 participants drawn from the general, rather than medically compromised, population. Results indicated that with every incremental increase of 10 beats per minute resting HR, there was an increase in the relative risk of all-cause (RR = 1.09, CI = 1.07-1.12) and cardiovascular mortality (RR = 1.08, CI = 1.06-1.12) when cardiovascular risk factors were controlled for. Subgroup analyses were conducted to examine group differences in mortality risk. Using 45 beats per minute resting HR as a reference group, the risk of all-cause mortality increased linearly with resting HR while the risk of cardiovascular mortality significantly increased at 90 beats per minute resting HR. This meta-analysis, along with a large body of literature, has indicated that resting HR is an independent predictor of mortality in the

general population (Zhang et al., 2015, p. E60). Therefore, elevated HP in response to a transient sad mood and impaired HP recovery is likely maladaptive.

It is possible that the impaired HP recovery observed in formerly depressed individuals exposed to the sad mood induction in the current study may increase vulnerability to depression and contribute to the development of psychopathology and physiological abnormalities (Linden et al., 1997; Haynes, Gannon, Orimoto, O'Brien, & Brandt, 1991). It is important to note that two characterizations of cardiovascular functioning were not captured by the experimental paradigm: the typical length of impaired HP recovery and occurrence of HP reactivity and recovery. First, impaired HP recovery may extend longer than was captured by the current study, which observed cardiovascular recovery over a 10-minute period. Given that HP continued to be elevated in formerly depressed individuals exposed to the sad mood induction at the 10-minute mark (see Figure 31), it is not currently known how long the elevation in HP would typically persist. Second, HP reactivity and impaired HP recovery in response to a transient sad mood likely occurs more often than was modeled in the experimental paradigm. Moreover, fluctuations in mood are common during daily life; a dysphoric mood may arise when an individual is reminded of a sad memory, reflects on a past failure, experiences a social slight, or engages in an interpersonal conflict. Therefore, elevations in HP and the resulting impaired HP recovery likely occurs at multiple times during the day and may last for extended amounts of time. There is a distinct possibility that formerly depressed individuals may regularly exhibit a pattern of repeated HP reactivity and impaired HP recovery in response to a transient sad mood, which impacts their cardiovascular health and functioning in a clinically significant manner. Further study of the duration of impaired HP recovery and incident of HP reactivity and recovery is needed to experimentally and clinically validate this theory.

In addition, it is plausible that impaired HP recovery following a transient sad mood in formerly depressed individuals are implicated in the well-established relationship between depression and CVD (Haigh & Bogucki, 2017; Haigh et al., 2018b). If formerly depressed individuals exhibit a pattern of repeated HP reactivity and impaired HP recovery in response to a transient sad mood, cardiovascular abnormalities may arise. While the current study did not directly explore this connection, it is an important first step for investigating this hypothesis. More research is needed to replicate these findings, extend these findings using longitudinal methods and if longitudinal results identify an association between cardiovascular recovery and physiological abnormalities, investigate biological mechanisms to determine the pathophysiological process at play and the efficacy of interventions (e.g., respiratory feedback, biofeedback, emotion regulation strategies) designed to facilitate cardiovascular recovery (Sharpley, 2002).

Finally, the variable results obtained using different timing for (i.e., two- versus five-minutes) and approaches (i.e., difference versus residualized change score) to calculate cardiovascular reactivity and recovery illustrate the challenge of conducting psychophysiological research. Unfortunately, there is little consistency across the literature as to how many minutes should be averaged to calculate reactivity and recovery or which time segments of reactivity or recovery should be selected. More research is needed to establish more formal guidelines as to the appropriate amount of time and timing of segments that researchers should select depending on their research question, experimental task, and population of interest, among other factors (Linden et al., 1997). This knowledge may be helpful for moving psychophysiological research forward as it would likely reduce some of the ambiguity associated with psychophysiological

analyses and increase standardization across studies, increasing the ease of cross-study comparisons.

Strengths

The current study has several strengths, which meaningfully extend the findings of previous research. First, the current study addressed the multiple methodological issues present in previous research. Prior literature has relied upon a mixed sample of participants who were currently or formerly depressed (Bylsma et al., 2015; Yaroslavsky et al., 2013, 2014 Studies 1 and 2; with the exception of Rottenberg et al., 2005b) and examined a very limited range of cardiovascular measures (i.e., HR or RSA only; Rottenberg et al., 2005b; Yaroslavsky et al., 2013, 2014 Studies 1 and 2; with the exception of Bylsma et al., 2015). A large portion of the literature has failed to compare cardiovascular reactivity in response to both sad and neutral mood inductions (Yaroslavsky et al., 2013, 2014 Studies 1 and 2; with the exceptions of Bylsma et al., 2015 and Rottenberg et al., 2005b) and was conducted in formerly depressed adolescents (Bylsma et al., 2015; Yaroslavsky et al., 2014 Study 2; with the exceptions of Rottenberg et al., 2005b and Yaroslavsky et al., 2013, 2014 Study 1) rather than adults. Finally, none of the literature explicitly assessed for the presence of CVD or investigated cardiovascular recovery (Bylsma et al., 2015; Rottenberg et al., 2005b; Yaroslavsky et al., 2013, 2014 Studies 1 and 2).

Accordingly, this is the first study to examine a broad range of cardiovascular measures to assess cardiovascular reactivity to and recovery from sadness in an exclusively remitted MDD adult sample free from CVD. The current study employed a quasi-experimental design, which compared two groups (i.e., formerly depressed, healthy control) randomly assigned to two experimental conditions (i.e., sad and neutral mood induction). Together, the quasi-experimental

design, stringent inclusion and exclusion criteria, and control of potential confounding variables allowed for competing explanations to be ruled out.

Second, the current study determined eligibility and group assignment using a structured clinical interview, the SCID-IV-RV, that had been adapted in accordance with the DSM-5. The SCID was developed in an effort to increase diagnostic reliability for DSM diagnoses through the use of standardized questions that aligned with diagnostic criteria and consistent language to enhance interrater agreement (Bergman & Fors, 2005). To be accurately determined, clinical diagnoses must be evaluated using a structured or semi-structured clinical interview rather than self-report measures due to the biases associated with these instruments (Paulhus & Vazire, 2007). The use of the SCID-IV-RV in the current study allowed for diagnostic accuracy across the different experimenters evaluating whether or not participants met the inclusion and exclusion criteria.

Third, the current study employed multiple reliable and standardized methods that are in line with the recommendations of RDoC. Under the negative valence system, there are multiple constructs including acute threat ("fear"), potential harm ("anxiety"), sustained threat, frustrative non-reward, and loss. MDD aligns most closely with the construct of loss in the negative valence system (National Institute of Mental Health, 2011). RDoC recommends the use of multiple levels of analysis to assess a construct in an effort to obtain a more comprehensive understanding of the pathological mechanisms underlying current diagnostic categories and eventually, create a dimensional diagnostic system (Cuthbert, 2014). The RDoC negative valence systems workgroup has identified multiple behavioral assessment methods for studying the construct of loss including rumination, withdrawal, worry, crying, sadness, loss-relevant recall bias, attentional bias to negative valenced information, guilt, morbid thoughts, psychomotor

retardation, anhedonia, increased self-focus, deficits in executive functioning, loss of drive, decreased libido, shame, amotivation, memory impairments, and intrusive thoughts (National Institute of Mental Health, 2011). In this study, cognitive reactivity aligns most closely with intrusive thoughts while mood reactivity aligns most closely with sadness. In addition, the RDoC negative valence system has identified multiple physiological assessment methods for studying the construct of loss including ANS, HPA, and neuroimmune dysregulation and prolonged psychophysiological reactivity (National Institute of Mental Health, 2011). In this study, cardiovascular reactivity aligns most closely with prolonged psychophysiological reactivity, and could also be subsumed under cardiovascular recovery. While the current study did assign group membership according to DSM diagnostic criteria, it did adopt a more dimensional approach in line with the recommendations of RDoC.

Limitations and Future Directions

The current study has several limitations, which can be addressed by future research. First, the study sample was recruited from the University of Maine psychology undergraduate participant pool and the surrounding community. The study sample was predominantly younger, female, Caucasian, never married college students despite significant efforts to recruit a more diverse sample. Consequently, the study sample is not representative of the U.S. population. While the reported rate of depression is higher women (Eaton et al., 2007; Grant et al., 2009; Hasin et al., 2005; Kessler et al., 2003; Kuehner, 2003, Seedat et al., 2009), younger to middle-aged adults (Eaton et al., 2007; Grant et al., 2009; Haigh et al., 2018a; Kessler et al., 2003, 2005, 2010), and Caucasian individuals (Alegría et al., 2008; Williams et al., 2007), more research is needed on risk for recurrence of depression across the population. Future research should attempt to recruit a more diverse and representative sample.

Second, the sample size recruited for the current study was relatively small. Significant efforts were made to recruit a sample size that was large enough to detect an effect should it be present. Power analysis using G*Power 3.1.9.2 (Faul et al., 2007) indicated that a sample of 128 participants would result in an 80.00% chance of detecting a medium effect between two groups (i.e., formerly depressed and healthy control participants) exposed to two versions of the experimental paradigm (i.e., sad or neutral mood induction). While the recruitment target was met (N = 132), even slightly exceeded, distribution of participants was uneven between groups (n_{FD} = 45, n_{HC} = 87) and conditions (n_{SMI} = 65, n_{NMI} = 67). The main issue with unequal sample sizes when conducting ANOVA analyses is that it can impact homogeneity of variance. ANOVA is robust statistical test that can handle "moderate departures" from homogeneity of variance (Grace-Martin, 2019b). In the current study, multiple steps were explored in an attempt to remedy the violation of homogeneity of variance, including transforming the data to reduce skewness, or decreasing the α level to reduce the likelihood of type II error (S. W. Ell, personal communication, February 9, 2016).

There were a variety of reasons for uneven distribution between groups and conditions in the current study. In regard to groups, it was challenging to recruit formerly depressed participants that met the inclusion and exclusion criteria. This is likely multifactorial and may be due to stigma associated with mental illness, elevated rates of comorbid medical and mental health conditions in depressed populations, and the chronic, recurrent nature of depression that may have resulted in eligible participants identified at screening who were subsequently excluded when assessed using the SCID-IV-RV at a later timepoint. Contrariwise, it was significantly easier to recruit healthy control participants. In regard to conditions, participants were assigned to the conditions roughly evenly, with slight differences in the size of groups due

to between-session attrition from sessions 1 and 2. Finally, the power analysis conducted a priori may not have been precise as it was it was challenging to determine the anticipated effect size. First, the current study drew from two distinct literature bases (i.e., cognitive and mood literature and cardiovascular literature) that had different conventions and effect sizes of findings. Second, the cardiovascular literature for remitted depression was limited and often neglected to report effect sizes for findings. Future research that aims to replicate these findings should ensure that an adequate sample size is recruited to detect small to medium effects. Both researchers and editors should ensure that effect sizes are reported to allow for accurate power analyses when replicating results. In addition, future research that obtains an adequate sample size could consider dividing formerly depressed individuals into subgroups of variables that may mediate or moderate the relationship between depression and cognitive, mood, and cardiovascular reactivity and recovery in an effort to better predict recurrence of depression and advance precision medicine (Monroe et al., in press).

Third, the inclusion and exclusion criteria used in the current study were stringent. The inclusion and criteria omitted potential confounding variables that could have impacted psychophysiological measures. All participants were excluded from the study if they did not speak and read English fluently, were color blind, had been diagnosed with a learning disability that interferes with their ability to read or process visual information, had experienced certain physical conditions known to impact the recording of physiological responses, or underwent brain or neural surgery or brain radiation treatment. In addition, the inclusion and criteria omitted participants who met diagnostic criteria for certain psychiatric disorders. Formerly depressed participants were excluded if they met diagnostic criteria for current MDD within the past month, current substance abuse within the past 6 months, current or past substance dependence,

bipolar disorder, psychotic disorder, acute suicidal ideation, or mood episodes secondary to general medical conditions while healthy control participants were excluded if they met diagnostic criteria for any current or past psychological disorder. Consequently, the entire sample was exceptionally healthy, both physically and mentally. The rigor of the inclusion and exclusion criteria may have had the unintended consequence of selecting for a sample that may not be representative of depressed individuals seeking treatment in the community, which should be considered to avoid inappropriately extrapolating results to community-samples. Future research may consider using more lenient criteria, when possible, to obtain a more realistic sample of treatment seeking patients. More specifically, formerly depressed participants with diverse psychiatric presentations and healthy control participants with a history of non-depressive disorders may be included or considered as a separate comparison group.

Fourth, CO, which was originally proposed as a cardiovascular measure, was dropped from analyses due to concerns its accuracy. Typical values for resting CO range from 4 to 12 liters per minute (J. Schmidt, personal communication, April 16, 209). Average CO values ranged from 1.62 to 365.73 during baseline, 1.49 to 390.58 during the mood induction, and 1.69 to 175.80 during recovery, indicating that this cardiovascular measure was not accurate. It was determined that this issue was due to inaccurate SV values. SV is not used to calculate HP, RSA, or PEP and therefore, these cardiovascular measures were not affected. Future research should include CO as a cardiovascular measure to evaluate the efficiency of the heart in response to a transient sad mood (Berntson et al, 2007). In addition, future research should expand beyond cardiovascular measures to also include neuroendocrine and immunological measures in an effort to understand their relationship and the pathopsychophysiological cascades that contribute to dysfunction and disease (Linden et al., 1997).

Fifth, treatment history (i.e., current and past CBT and antidepressant use), which was originally proposed as a potential covariate, was not adequately captured in the study sample. Treatment history data was not collected for 16 formerly depressed and 40 healthy control participants. Due to the large amount of missing data, treatment history was removed from further analyses. CBT use was originally proposed as a potential covariate because previous research has suggested that engagement in CBT is associated with lower levels of cognitive reactivity (Jarrett et al., 2012; Kuyken et al., 2010; Segal et al., 1999). None of the cardiovascular studies conducted in remitted MDD participants reported participants' engagement in CBT interventions or psychotherapy more broadly (Ahrens et al., 2008; Bylsma et al., 2014, 2015; Chang et al., 2012; Rottenberg et al., 2005b; Salomon et al., 2013; Wilson et al., 2016; Yaroslavsky et al., 2013, 2014a, 2014b). Antidepressant use was originally proposed as a potential covariate because previous research has found an association between antidepressant use and certain measures of cardiovascular functioning (e.g., RSA; Licht et al., 2008). While the majority of the cardiovascular studies conducted in remitted MDD participants did not exclude for the use of antidepressant medications (Ahrens et al., 2008; Bylsma et al., 2014, 2015; Rottenberg et al., 2005; Salomon et al., 2013; Wilson et al., 2016; Yaroslavsky et al., 2013, 2014a, 2014b, with the exception of Chang et al., 2012), only a subset of these studies considered antidepressant medication as a potential covariate (i.e., Rottenberg et al., 2005b; Salomon et al., 2013; Vaccarino et al., 2008; Yaroslavsky et al., 2013, 2014a, 2014b). Of those studies, there were no significant differences based on medication status. Future research may consider including both CBT and antidepressant use as potential covariates to further expand upon the research base.

Sixth, potential mediators and moderators of cardiovascular reactivity and recovery were not adequately captured in the current sample. As previously mentioned, one potential mechanism of action that may be explored to explain elevated cardiovascular reactivity and impaired cardiovascular recovery is rumination. This could be investigated by asking participants to record the thoughts that they were having during the mood induction and recovery period. Of note, it would be interesting to compare differences in cardiovascular recovery and rumination among formerly depressed individuals using different recovery tasks: a film that consists of potentially distracting visual and auditory stimuli, a piece of music that consists of potentially distracting auditory stimuli, a meditative exercise that consists of potentially distracting spoken words, and a silent recovery period without any distractions. Another potential mechanism of action that may be explored to explain impaired cardiovascular recovery is impaired mood recovery. This could be investigated by asking participants to complete the VAS after the recovery period. Future research may consider including these and other potential mediators and moderators to further expand upon the research base.

Conclusions

Increasing our understanding of vulnerability to depressive relapse and recurrence of depression is necessary to reduce the burden of this often-debilitating disorder, with an exigency to identify potentially malleable factors that can be targeted during treatment or following treatment for relapse prevention. Four potentially malleable factors that may be implicated in depressive relapse and recurrence include cognitive, mood, and cardiovascular reactivity to and recovery from sadness. Results from the current study suggest that mood, rather than cognitive, reactivity in response to a transient sad mood is observed in formerly depressed individuals. In addition, results from the current study suggest that reduced HP recovery, rather than other

measures of cardiovascular recovery or cardiovascular reactivity, following the induction of a transient sad mood is observed in formerly depressed individuals. Additional research is needed to replicate previous results indicating that mood reactivity is predictive of depressive relapse and recurrence as well as assess if the differences in cardiovascular responding observed in formerly depressed individuals persist over time and contribute to the development of MDD.

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APPENDICES

Appendix A. *Recruitment Flyers*

THE UNIVERSITY OF MAINE

HAVE YOU NEVER BEEN DEPRESSED?

Help the Maine Mood Disorders lab at UMaine in Orono learn more about how changes in mood impact risk for depression by participating in a **paid research study**.

Take our survey if you:

- ✓ Have **no history** of depression, anxiety, or any other emotional disorder
- ✓ Do not suffer from alcohol abuse or dependence
- ✓ Are between 18 and 60 years of age

If you qualify:

- Session 1 (Interview for about 1.5 hours in our lab; $20 compensation)
- Session 2 (Physiological recording while you complete a computer task in the lab for about 1 hour; $15 compensation)
- Session 3 (Online questionnaires that will take about 30 minutes; entered in drawing with 1 in 10 chance to win a $25 VISA Card)

TAKE OUR ONLINE SURVEY TO SEE IF YOU ARE ELIGIBLE!

Scan this QR code
or Visit: tinyurl.com/k3s2mrp
or Text: 207-518-8089
or Email: mainemooddisorderslab@gmail.com

PAST history of depression?

Volunteers needed for a paid research study

Help the Maine Mood Disorders lab at UMaine in Orono learn more about how changes in mood impact risk for depression by participating in a **paid research study.**

If you qualify, participation may include:

- Session 1 (Interview at UMaine for about 1.5 hours; **$20** compensation)
- Session 2 (Physiological recording while you complete a computer task at UMaine for about 1 hour; **$15** compensation)
- Session 3 (Online questionnaires that will take about 30 minutes; entry into drawing with **1 in 10** chance to win a **$25** VISA Card)

Who is Eligible? Take our online survey *if* you:

- Have a history of depression but are NOT currently depressed
- Do not suffer from alcohol abuse or dependence
- Are between 18 and 60 years of age

Have questions or interested in participating? Scan this QR code, visit tinyurl.com/k3s2mrp, call/text 207-518-8089, or email our lab at mainemooddisorderslab@gmail.com.

Maine Mood Disorders Lab
329 Corbett Hall
University of Maine
Orono, Maine 04469-5782
207-518-8089

1865 THE UNIVERSITY OF MAINE

Appendix B. *Screening Consent Forms*

Welcome to the University of Maine Psychology Department Pre-screening Questionnaires!

You have chosen to participate in research studies to meet your research experience requirement. One option to partially complete this requirement is to complete the prescreening questionnaires. The purpose of this screening is to find individuals who score in certain ranges on questionnaires for several different research projects. Dr. Fayeza Ahmed, Dr. Emily Haigh, Dr. Jordan LaBouff, Dr. Rebecca Macaulay, Dr. Shannon McCoy, Dr. Doug Nangle, and Dr. Rebecca Schwartz-Mette, professors in the Psychology Department, are conducting this screening. Based on your responses, you may be contacted to participate in one or more studies or you may not be contacted at all.

What Will You Be Asked to Do?

If you decide to participate, you will be asked to complete a series of questions about yourself, your attitudes, and your beliefs. This prescreening survey will take no longer than 60 minutes.

Risks

You may be asked questions like the following: Have you ever had a panic attack; have you ever been depressed; how do you feel about your political party; do you experience any of the following premenstrual symptoms (e.g., difficulty concentrating, depressed mood, breast tenderness); do you think fat people tend to be fat pretty much through their own fault; would you be upset if you learned that your son was gay; if I was hanging out with a homosexual person, I would worry that other people would think I was a homosexual too; do you consider yourself a Christian; have you lost interest in sex completely; are you feeling down, depressed, or hopeless; do you think that you may be dirty or contaminated; do you think Male homosexuality is a perversion; etc. You may become uncomfortable answering some of the questions. If you have any concerns, please contact Melissa Jankowski (Graduate Student Coordinator of the Psychology Subject Pool) on First Class. If any of the questions or content raises concerns that you wish to discuss and debrief with a professional, you should contact the University of Maine's Counseling Services at Cutler Health Center (207-581-1392).

Compensation:

You will receive 1 hour of research credit for participating in this study. You must reach the finishing page of the survey to receive credit.

Confidentiality

We need your name if you wish to be contacted for participation in one of the research projects. This information will not be shared with anyone other than the individuals' research teams named above, and identifying information will be kept separate in a different file (i.e., you will be identified by an arbitrary number). The data file without identifying information will be kept on password protected computers in locked laboratories indefinitely. The keyed file linking your

name with your arbitrary identifier will be stored separately on a password protected drive in a locked laboratory or office, using software that provides additional security. The prescreening data and key linking names with ID numbers will be destroyed at the end of the semester.

Voluntary

Participation is voluntary. While skipping an occasional answer is acceptable, in order for the data to be useful, most questions must be answered. You must reach the finishing page of the survey to receive credit. If you decide at any point that you would rather not continue with the prescreener, you can do the article reviews OR participate in those studies that do not involve the prescreener (and there are plenty of these studies).

Contact Information

If you have questions about this screening, please contact Dr. Jordan LaBouff (207-581-2826), 352 Little Hall, or e-mail: sona.admin@umit.maine.edu). If you have questions about your rights as a research participant, please contact Gayle Jones, Assistant to the University of Maine's Protection of Human Subjects Review Board, at 207-581-1498 (or e-mail gayle.jones@umit.maine.edu).

By clicking "yes" below, I consent to participate in this study.

☐ Yes – I consent to participate and I am over 18 years old

☐ No – I do not consent

☐ I am under 18 but I would like to participate

Attention and Elaboration Study: Prescreen
The University of Maine at Orono
Prescreening Informed Consent Document (Community Participants)

You are invited to participate in a research project being conducted by Dr. Emily Haigh, in the Department of Psychology at the University of Maine. The purpose of the research is to learn about the emotional and physiological responses related to sad mood. You must be at least 18 years of age to participate.

What Will You Be Asked to Do?

Complete a set of online questionnaires to determine if you are eligible for the study.

As part of the online survey you will answer questions about how you're feeling (e.g. "After I overeat, occasionally I feel guilt or self-hate," "I feel guilty all the time," or "I am disgusted with myself.") and different types of thoughts that people sometimes have (e.g., "I worry about making mistakes" or "I do not need the approval of other people in order to be happy"). This portion of the study will take about 15-20-minutes total.

If you are eligible for the study, you will receive an email inviting you to sign up to complete Session 1 in the lab. During Session 1, participants will complete questionnaires and an interview, where they will be asked about their mood (e.g., "In the past month, have you been feeling depressed or down?") and different symptoms (e.g., "In the past month, have you had trouble sleeping?"). After the interview, a graduate student will measure your height and weight. Session 1 will take about 1.50 hours.

Based on information gathered during Session 1, some participants will be asked to take part in Session 2. If you are eligible and decide to participate in the second part, you will be scheduled for another session that will take place on a different day. For Session 2, you will be asked to participate in physiological recording (sensors to detect electrical impulses will be attached to your chest and back) while you complete the following: self-report questionnaires, a computerized attention task, and listen to either a sad or neutral piece of music designed to induce a short-lasting sad mood or no change in mood.

Participants that complete the second portion of the study will be invited to complete a third and final portion of the study. For this part of the study, you will receive an email with a link to some questions about your mood and whether you have experienced any recent stressful events.

Risks

It is possible that you may feel uncomfortable when answering questions about yourself. At any point during the study, you have the right to skip questions you do not wish to answer, or stop the session and choose not to participate in the remainder of the study. You will not need to provide a reason for stopping the session. You will receive a list of referrals for counseling services at the end of this questionnaire that can be downloaded. If you indicate that you wish to harm yourself, Dr. Emily Haigh will contact you by email.

Benefits

While this study will have no direct benefit to you, this research will help us learn more about how experiencing brief sad mood relates to depression.

Compensation

While there is no direct compensation for filling out this prescreen, by answering these questions you may qualify for Session 1 and Session 2 in our laboratory. These sessions include monetary compensation for time and travel expenses.

Confidentiality

We need your name if you wish to be contacted for participation in this research project. This information is not shared with anyone outside of the lab. Identifying information will be kept separate in a different file. A code number will be used to protect your identity. The data file without identifying information will be kept on a password protected computer in a locked laboratory indefinitely. The keyed file linking your name and code number will be stored separately on a password protected computer in the investigator's locked office and will only be accessible by Dr. Emily Haigh, Maine Mood Disorders Lab graduate students, and research assistants who have been trained to deal with sensitive material. Your name or other identifying information will not be reported in any publications. The key linking your name to the data will be destroyed two years after data analysis is complete, which we anticipate will be in December, 2018. The key and the data files will be stored on separate computers. All data will be kept indefinitely by the investigators. You may decide that you do not want your data used in this research. If you would like your data removed from the study and permanently deleted, please email your request to the Principal Investigator, Dr. Emily Haigh, at emily.a.haigh@maine.edu.

Voluntary

Participation is voluntary. If you choose to take part in this study, you may stop at any time. You may also skip any questions you do not wish to answer.

Contact Information

If you have any questions about this study, please contact Emily Haigh at Emily.a.haigh@maine.edu. If you have any questions about your rights as a research participant, please contact Gayle Jones, Assistant to the University of Maine's Protection of Human Subjects Review Board, at 581-1498 or via e-mail gayle.jones@umit.maine.edu.

Future Studies

Would you be interested in being contacted for future studies conducted in the lab for monetary compensation?

☐ Yes ☐ No

By clicking "Yes" below, you indicate that you have read and understand the above information and agree to participate.

If you are no longer interested, please click "No" to exit to questionnaire.

☐ Yes ☐ No

Appendix C. *Screening Self-Report Measures*

Contact Information

Thank you for your interest in our study! Please provide your contact information below so we can contact you if you are eligible.

Please provide your full name: _____

Please provide your email address: _____

Please provide your phone number (including area code): _____

Demographic Information

To start with, we would like to get some background information from you.

1. What is your age? _____

2. What is your date of birth (MM/DD/YYYY)? _____

3. What is your gender?
   ○ Male
   ○ Female

4. What is your marital situation (please check one)?
   ○ Married
   ○ Separated
   ○ Never married/single
   ○ Common law marriage
   ○ Divorced
   ○ Widowed

5. Do you consider yourself to be Hispanic or Latino (i.e., a person of Mexican, Puerto Rican, Cuban, South or Central American, or other Spanish culture of origin, regardless of race)?
   ○ Yes
   ○ No

6. Do you consider yourself to be Franco-American?
   ○ Yes
   ○ No

7. What is your race?
   ○ Native American or Alaska Native (i.e. a person having origins in any of the original peoples of North, Central, or South America)
   ○ Asian (i.e. a person having origins in any of the original peoples of the Far East, Southeast Asia, or the Indian subcontinent including, for example, Cambodia, China, India, Japan, Korea, Malaysia, Pakistan, the Philippine Islands, Thailand, and Vietnam)
   ○ Black or African American (i.e. a person having origins in any of the black racial groups of Africa)
   ○ Native Hawaiian or Other Pacific Islander (i.e. a person having origins in any of the .. original peoples of Hawaii, Guam, Samoa, or other Pacific Islands)
   ○ White (i.e. a person having origins in any of the peoples of Europe, the Middle East, or North Africa)
   ○ Multiple races
   ○ None of the above

8. What is the highest grade in school you have completed (please check one)?
   - ○ Less than High School
   - ○ High School
   - ○ 1 year of college or technical school
   - ○ 2 or more years of college but did not graduate
   - ○ 4 years of college with degree
   - ○ Postgraduate, M.D., Ph.D.
   - ○ A.A. or other degree that is not a B.A. or B.S.

General Health Screen (GHS)

Please answer yes or no to the following questions:

1. Do you speak and read English fluently?
   ○ Yes
   ○ No

2. Are you color-blind?
   ○ Yes
   ○ No

3. Have you ever been diagnosed with any learning disabilities that interfere with your ability to read or process visual information?
   ○ Yes
   ○ No

4. Have you lost consciousness for more than one hour ever?
   ○ Yes
   ○ No

5. Have you ever been diagnosed with any neurological disorder, such as Alzheimer's Disease, Parkinson's Disease, Huntington's Disease?
   ○ Yes
   ○ No

6. Have you ever had a stroke, hemorrhage, or brain tumor?
   ○ Yes
   ○ No

7. Have you ever had brain/neural surgery or brain radiation treatment (e.g. for brain tumor)?
   ○ Yes
   ○ No

8. Do you have multiple seizures or Epilepsy?
   ○ Yes
   ○ No

9. Have you ever been diagnosed with cardiovascular disease? Heart disease? Hypertension? Medication-dependent diabetes?
   ○ Yes
   ○ No

Comments: _____

Beck Depression Inventory – Second Edition (BDI-II)

This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the ONE STATEMENT in each group that best describes the way you have been feeling during the PAST TWO WEEKS, INCLUDING TODAY. Bubble in the number beside the statement you have picked. If several statements in the group seem to apply equally well, bubble in the highest number for that group. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness
   ⓪ I do not feel sad.
   ① I feel sad much of the time.
   ② I am sad all the time.
   ③ I am so sad or unhappy that I can't stand it.

2. Pessimism
   ⓪ I am not discouraged about my future.
   ① I feel more discouraged about my future than I used to be.
   ② I do not expect things to work out for me.
   ③ I feel my future is hopeless and will only get worse.

3. Past Failure
   ⓪ I do not feel like a failure.
   ① I have failed more than I should have.
   ② As I look back, I see a lot of failures.
   ③ I feel I am a total failure as a person.

4. Loss of Pleasure
   ⓪ I get as much pleasure as I ever did from the things I enjoy.
   ① I don't enjoy things as much as I used to.
   ② I get very little pleasure from the things I used to enjoy.
   ③ I can’t get any pleasure from the things I used to enjoy.

5. Guilty Feelings
   ⓪ I don't feel particularly guilty.
   ① I feel guilty over many things I have done or should have done.
   ② I feel quite guilty most of the time.
   ③ I feel guilty all of the time.

6. Punishment Feelings
   ⓪ I don't feel I am being punished.
   ① I feel I may be punished.
   ② I expect to be punished.
   ③ I feel I am being punished.

7. Self-Dislike
⓪ I feel the same about myself as ever.
① I have lost confidence in myself.
② I am disappointed with myself.
③ I dislike myself.

8. Self-Criticalness
⓪ I don't criticize or blame myself more than usual.
① I am more critical of myself than I used to be.
② I criticize myself for all of my faults.
③ I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes
⓪ I don't have any thoughts of killing myself.
① I have thoughts of killing myself, but I would not carry them out.
② I would like to kill myself.
③ I would kill myself if I had the chance.

10. Crying
⓪ I don't cry any more than I used to.
① I cry more than I used to.
② I cry over every little thing.
③ I feel like crying, but I can't.

11. Agitation
⓪ I am no more restless or wound up than usual.
① I feel more restless or wound up than usual.
② I am so restless or agitated that it's hard to stay still.
③ I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest
⓪ I have not lost interest in other people or activities.
① I am less interested in other people or things than before.
② I have lost most of my interest in other people or things.
③ It's hard to get interested in anything.

13. Indecisiveness
⓪ I make decisions about as well as ever.
① I find it more difficult to make decisions than usual.
② I have much greater difficulty in making decisions than I used to.
③ I have trouble making any decisions.

14. Worthlessness
⓪ I do not feel I am worthless.
① I don’t consider myself as worthwhile and useful as I used to.
② I feel more worthless as compared to other people.
③ I feel utterly worthless.

15. Loss of Energy
⓪ I have as much energy as ever.
① I have less energy than I used to have.
② I don’t have enough energy to do very much.
③ I don’t have enough energy to do anything.

16. Changes in Sleeping Pattern
⓪ I have not experienced any change in my sleeping pattern.
① I sleep somewhat more than usual.
① I sleep somewhat less than usual.
② I sleep a lot more than usual.
② I sleep a lot less than usual.
③ I sleep most of the day.
③ I wake up 1-2 hours early and can't get back to sleep.

17. Irritability
⓪ I am no more irritable than usual.
① I am more irritable than usual.
② I am much more irritable than usual.
③ I am irritable all the time.

18. Changes in Appetite
⓪ I have not experienced any change in my sleeping appetite.
① My appetite is somewhat less than usual.
① My appetite is somewhat more than usual.
② My appetite is much less than usual.
② My appetite is much greater than usual.
③ I have no appetite at all.
③ I crave food all the time.

19. Concentration Difficulty
⓪ I can concentrate as well as ever.
① I can't concentrate as well as usual.
② It's hard to keep my mind on anything for very long.
③ I find I can't concentrate on anything.

20. Tiredness or Fatigue
⓪ I am no more tired or fatigued than usual.
① I get tired or fatigued more easily than usual.
② I am too tired or fatigued to do a lot of the things I used to do.
③ I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex
⓪ I have not noticed any recent change in my interest in sex.
① I am less interested in sex than I used to be.
② I am much less interested in sex now.
③ I have lost interest in sex completely.

Patient Health Questionnaire – 9 (PHQ-9)

For the *two weeks* in your life when you felt the *most* blue, sad, or depressed, how often were you bothered by any of the following problems?

		Rarely/ Not at all	Several days	More than half the days	Nearly every day
1.	Little pleasure or interest in doing things	0	1	2	3
2.	Feeling down, depressed or hopeless	0	1	2	3
3.	Trouble falling or staying asleep or sleeping too much	0	1	2	3
4.	Feeling tired or having little energy	0	1	2	3
5.	Poor appetite or overeating	0	1	2	3
6.	Feeling bad about yourself – or that you are a failure or you have let yourself or your family down	0	1	2	3
7.	Trouble concentrating on things, such as reading the newspaper or watching television	0	1	2	3
8.	Moving or speaking slowly so that other people could have noticed. Or the opposite – being so fidgety or restless that you have been moving around a lot more than usual	0	1	2	3

		Not at all	Somewhat	Very	Extremely
9.	How difficult did these problems make it for you to do your work, take care of things at home, or get along with other people?	0	1	2	3

Beck Anxiety Inventory (BAI)

Below is a list of common symptoms of anxiety. Please carefully read each item in the list. Indicate how much you have been bothered by that symptom during the past month, including today, by circling the number in the corresponding space in the column next to each symptom.

		Not at all	Mildly, but it didn't bother me much	Moderately – it wasn't pleasant at times	Severely – it bothered me a lot
1.	Numbness or tingling	0	1	2	3
2.	Feeling hot	0	1	2	3
3.	Wobbliness in legs	0	1	2	3
4.	Unable to relax	0	1	2	3
5.	Fear of worst happening	0	1	2	3
6.	Dizzy or lightheaded	0	1	2	3
7.	Heart pounding/racing	0	1	2	3
8.	Unsteady	0	1	2	3
9.	Terrified or afraid	0	1	2	3
10.	Nervous	0	1	2	3
11.	Feeling of choking	0	1	2	3
12.	Hands trembling	0	1	2	3
13.	Shaky/unsteady	0	1	2	3
14.	Fear of losing control	0	1	2	3
15.	Difficulty breathing	0	1	2	3
16.	Fear of dying	0	1	2	3
17.	Scared	0	1	2	3
18.	Indigestion	0	1	2	3
19.	Faint/lightheaded	0	1	2	3
20.	Face flushed	0	1	2	3
21.	Hot/cold sweats	0	1	2	3

Appendix D. *Counseling Resources*

If you feel upset after having completed the study or find that some questions or aspects of the study triggered distress, talking with a qualified clinician may help. The following represents a list of resources that you may contact. These resources are options and in no way do they reflect an endorsement by the University of Maine.

Counseling Services		
ON-CAMPUS RESOURCES Available for UMaine Faculty, Staff, and Students		
Counseling Center **Cutler Health Building** **(Gannet Hall side)** **(FREE to UMaine students)**	**207-581-1392** http://www.umaine.edu/counseling/	Weekdays 8:00 am-4:30 pm After business hours, call UMaine Police,
Psychological Services Center **330 Corbett Hall** (Sliding fee scale; costs are your responsibility)	**207-581-2034** http://umaine.edu/clinicalpsychology/psychological-services-center/	Weekdays 8:00 am-4:30 pm
COMMUNITY RESOURCES Available to Anyone		
Community Health & Counseling Services **42 Cedar Street** **Bangor, ME 04401** (Any costs are your responsibility)	**207-947-0366** http://www.chcs-me.org/	Weekdays 8:00 am-5:00 pm
Maine Warm Line (Any costs are your responsibility)	**1-888-771-9276** http://www.thecommunityconnector.org/directory/profile/maine-warm-line	7 days/week 5:00 pm-8:00 am
Maine Suicide and Crisis Hotline (Any costs are your responsibility)	**1-888-568-1112** http://www.maine.gov/suicide/youth/index.htm	7 days/week 24 hours
Psychological Services Center **330 Corbett Hall** (sliding fee scale)	**207-581-2034** http://umaine.edu/clinicalpsychology/psychological-services-center/	Weekdays 8:00 am-4:30 pm
Contact Your Primary Care Provider (Any costs are your responsibility)		
NATIONAL RESOURCES		
Mental Health Services Locator http://store.samhsa.gov/mhlocator		
National Suicide Prevention Lifeline, Toll-Free, 24-hour Hotline, **1-800-273-TALK (1-800-273-8255)**		

Appendix E. *Recruitment Email*

Hello,

Thank you very much for your interest in the research we are conducting at the Maine Mood Disorders Lab. We appreciate you taking the time to contact us and complete the online survey.

Your responses to the survey indicate you qualify for the next step of our study, an in-person interview in our lab. This will take approximately two hours. This session will involve the completion of several online questionnaires about how you are feeling and different types of thoughts people sometimes have. You will then participate in an interview that will ask you about your mood and different symptoms related to disorders like depression and anxiety (e.g., In the past month, have you had trouble sleeping?). More details will be provided once you are scheduled.

Using your name and email address, we created you a Sona Systems account. Sona is an online resource we use for scheduling.

Please visit Sona here to schedule a time that works for you to come to the lab for approximately 1.50 to 2 hours, using the username and password below:

Username: FIRSTNAME.LASTNAME
Password: blackbear

You are only eligible to participate in "Attention and Elaboration Session 1." Please click on this study and enter the following password: **blackbear**.

Thank you again for your interest in the Maine Mood Disorders Lab. We look forward to seeing you soon.

Best,

The Maine Mood Disorders Lab
MMDL Website
Visit us on Facebook

Appendix F. *Research Participation Credit Schedule*

Credit will be awarded based on the amount of time that it takes for a participant to complete the session. Allow participants who are distressed to end participation in the study (discuss this with a graduate student) without loss of payment based on hours spent in the laboratory to the nearest half hour, as indicated below.

Session 1:

Up to ½ hour	.50 research participation credit
½ hour to 1 hour	1 research participation credit
1 hour to 1 ½ hours	1.50 research participation credits
1 ½ hours to 2 hours (or session completion)	2 research participation credits

Session 2:

Up to ½ hour	.50 research participation credit
½ hour to 1 hour (or session completion)	1 research participation credit

Appendix G. *Payment Schedule*

Regardless of time spent in lab, pay full amount if participant completes the session. Allow participants who are distressed to end participation in the study (discuss this with a graduate student) without loss of payment based on hours spent in the laboratory to the nearest half hour, as indicated below.

Session 1:

Up to ½ hour	$5.00
½ hour to 1 hour	$10.00
1 hour to 1 ½ hours	$15.00
1 ½ hours to 2 hours (or session completion)	$20.00

Session 2:

Up to ½ hour	$8.00
½ hour to 1 hour (or session completion)	$15.00

Appendix H. *Session 1 Consent Forms*

Attention and Elaboration Study: Session 1
The University of Maine at Orono
Informed Consent Document (PSY 100, 212)

You are invited to participate in a research project being conducted by Dr. Emily Haigh, in the Department of Psychology at the University of Maine. The purpose of the research is to learn about the emotional and physiological responses related to sad mood. You must be at least 18 years of age to participate.

What Will You Be Asked to Do?

If you decide to participate, you will complete an online survey and an interview in the lab. As part of the online survey you will answer questions about how you're feeling (e.g. *"After I overeat, occasionally I feel guilt or self-hate.")* and different types of thoughts that people sometimes have (e.g., *"I worry about making mistakes" or "I do not need the approval of other people in order to be happy*"). This portion of the study will take about 30-minutes total.

Next, you will participate in an interview. During the interview, you will be asked about your mood (e.g., "*In the past month, have you been feeling depressed or down*?") and different symptoms that are related to disorders like depression and anxiety (e.g., "*In the past month, have you had trouble sleeping?*") The interview will take about 1.50 hours. With your consent, we will audio-record the interview. The audio-record will be used to confirm that the interview was conducted properly by the researcher. Even if you agree to be audio-recorded, you may ask us to stop or destroy the audio file at any time during or after the study is completed. After the interview, a graduate student will measure your height and weight.

Based on information gathered during the interview and questionnaires, some participants will be asked to take part in a second part of the study. If you are eligible and decide to participate in the second part, you will be scheduled for another session that will take place on a different day.

During the second part of the study, you will be given a description of the study and asked to give consent for the procedures involved. Briefly, you will be asked to participate in physiological recording (sensors to detect electrical impulses will be attached to your chest and back) while you complete the following: self-report questionnaires, a computerized attention task and listen to either a sad or neutral piece of music designed to induce a short-lasting sad mood or no change in mood.

Participants that complete the second portion of the study will be invited to complete a third and final portion of the study. For this part of the study, you will receive an email with a link to some questions about your mood and whether you have experienced any recent stressful events.

Risks

It is possible that you may feel uncomfortable when answering questions about yourself. At any point during the study, you have the right to skip questions you do not wish to answer, or stop the session and choose not to participate in the remainder of the study. You will not need to provide a reason for stopping the session. You will receive a list of referrals for counseling services at the end of your session today.

Benefits

While this study will have no direct benefit to you, this research will help us learn more about how experiencing brief sad mood relates to depression.

Compensation

You will receive 1 research credit for each hour of participation. Since the interview is expected to take 1.50 hours and the survey is expected to take 30-minutes, it is likely that you will earn 2 credits today.

Confidentiality

Your name will not appear on any of the documents. A code number will be used to protect your identity. This code is stored on a file with software designed to provide added security. Data will be kept in the investigator's locked office and will only be accessible by Dr. Emily Haigh, Maine Mood Disorders Lab graduate students, and research assistants who have been trained to deal with sensitive material. Your name or other identifying information will not be reported in any publications. The key linking your name to the data will be destroyed two years after data analysis is complete, which we anticipate will be in 2018. All data, including audio recordings, will be kept indefinitely by the investigators. The key and the data files will be stored on separate computers.

Voluntary

Participation is voluntary. If you choose to take part in this study, you may stop at any time. You may also skip any questions you do not wish to answer. You will earn 1 credit for each hour of participation with the possibility of earning 2 credits today.

Contact Information

If you have any questions about this study, please contact Emily Haigh at Emily.a.haigh@maine.edu. If you have any questions about your rights as a research participant, please contact Gayle Jones, Assistant to the University of Maine's Protection of Human Subjects Review Board, at 581-1498 or via e-mail gayle.jones@umit.maine.edu.

Audiotaping

I agree to audio recording the interview.

☐ Yes ☐ No

Future Studies

Would you be interested in being contacted for future studies conducted in the lab for monetary compensation?

☐ Yes ☐ No

Your signature below indicates that you have read and understand the above information and agree to participate. You will receive a copy of this form.

_____ _____

Signature Date

Attention and Elaboration Study: Session 1
The University of Maine at Orono
Informed Consent Document (Community Participants)

You are invited to participate in a research project being conducted by Dr. Emily Haigh, in the Department of Psychology at the University of Maine. The purpose of the research is to learn about the emotional and physiological responses related to sad mood. You must be at least 18 years of age to participate.

What Will You Be Asked to Do?

If you decide to participate, you will complete an online survey and an interview in the lab. As part of the online survey you will answer questions about how you're feeling (e.g. "After I overeat, occasionally I feel guilt or self-hate.") and different types of thoughts that people sometimes have (e.g., *"I worry about making mistakes" or "I do not need the approval of other people in order to be happy").* This portion of the study will take about 30-minutes total.

Next, you will participate in an interview. During the interview, you will be asked about your mood (e.g., "*In the past month, have you been feeling depressed or down?*") and different symptoms that are related to disorders like depression and anxiety (e.g., "*In the past month, have you had trouble sleeping?*"). The interview will take about 1.50 to 2 hours. With your consent, we will audio-record the interview. The audio-record will be used to confirm that the interview was conducted properly by the researcher. Even if you agree to be audio-recorded, you may ask us to stop or destroy the audio file at any time during or after the study is completed. After the interview, a graduate student will measure your height and weight.

Based on information gathered during the interview and questionnaires, some participants will be asked to take part in a second part of the study. If you are eligible and decide to participate in the second part, you will be scheduled for another session that will take place on a different day.

During the second part of the study, you will be given a description of the study and asked to give consent for the procedures involved. Briefly, you will be asked to participate in physiological recording (sensors to detect electrical impulses will be attached to your chest and back) while you complete the following: self-report questionnaires, a computerized attention task and listen to either a sad or neutral piece of music designed to induce a short-lasting sad mood or no change in mood.

Participants that complete the second portion of the study will be invited to complete a third and final portion of the study. For this part of the study, you will receive an email with a link to some questions about your mood and whether you have experienced any recent stressful events.

Risks

It is possible that you may feel uncomfortable when answering questions about yourself. At any point during the study, you have the right to skip questions you do not wish to answer, or stop the session and choose not to participate in the remainder of the study. You will not need to

provide a reason for stopping the session. You will receive a list of referrals for counseling services at the end of your session today.

Benefits

While this study will have no direct benefit to you, this research will help us learn more about experiencing how brief sad mood relates to depression.

Compensation

You will receive $20 for participating in this research session to compensate you for your time and travel expenses. If you do not complete the session you will receive compensation pro-rated to the nearest half hour.

Confidentiality

Your name will not appear on any of the documents. A code number will be used to protect your identity. This code is stored on a file with software designed to provide added security. Data will be kept in the investigator's locked office and will only be accessible by Dr. Emily Haigh, Maine Mood Disorders Lab graduate students and research assistants who have been trained to deal with sensitive material. Your name or other identifying information will not be reported in any publications. The key linking your name to the data will be destroyed in about two years after data analysis is complete, which we anticipate will be in 2018. All data, including audio recordings, will be kept indefinitely by the investigators. The key and the data files will be stored on separate computers.

Voluntary

Participation is voluntary. If you choose to take part in this study, you may stop at any time. You may also skip any questions you do not wish to answer.

Contact Information

If you have any questions about this study, please contact Emily Haigh at Emily.a.haigh@maine.edu. If you have any questions about your rights as a research participant, please contact Gayle Jones, Assistant to the University of Maine's Protection of Human Subjects Review Board, at 581-1498 or via e-mail at gayle.jones@umit.maine.edu.

Audiotaping

I agree to audio recording the interview.

☐ Yes ☐ No

Future Studies

Would you be interested in being contacted for future studies conducted in the lab for monetary compensation?

☐ Yes ☐ No

Your signature below indicates that you have read and understand the above information and agree to participate. You will receive a copy of this form.

_____ _____

Signature Date

Appendix I. *Session 1 Self-Report Measures*

Demographic Information

To start with, we would like to get some background information from you.

1. What is your age? _____

2. What is your gender? _____

3. What is your date of birth (MM/DD/YYYY)? _____

4. What is your marital situation (please check one)?
   ○ Married
   ○ Separated
   ○ Never married/single
   ○ Common law marriage
   ○ Divorced
   ○ Widowed

5. Do you consider yourself to be Hispanic or Latino (i.e., a person of Mexican, Puerto Rican, Cuban, South or Central American, or other Spanish culture of origin, regardless of race)?
   ○ Yes
   ○ No

6. Do you consider yourself to be Franco-American?
   ○ Yes
   ○ No

7. What is your race?
   ○ Native American or Alaska Native (i.e. a person having origins in any of the original peoples of North, Central, or South America)
   ○ Asian (i.e. a person having origins in any of the original peoples of the Far East, Southeast Asia, or the Indian subcontinent including, for example, Cambodia, China, India, Japan, Korea, Malaysia, Pakistan, the Philippine Islands, Thailand, and Vietnam)
   ○ Black or African American (i.e. a person having origins in any of the black racial groups of Africa)
   ○ Native Hawaiian or Other Pacific Islander (i.e. a person having origins in any of the .. original peoples of Hawaii, Guam, Samoa, or other Pacific Islands)
   ○ White (i.e. a person having origins in any of the peoples of Europe, the Middle East, or North Africa)
   ○ Multiple races
   ○ None of the above

8. What is the highest grade in school you have completed (please check one)?
   - ○ Less than High School
   - ○ High School
   - ○ 1 year of college or technical school
   - ○ 2 or more years of college but did not graduate
   - ○ 4 years of college with degree
   - ○ Postgraduate, M.D., Ph.D.
   - ○ A.A. or other degree that is not a B.A. or B.S.

Beck Depression Inventory – Second Edition (BDI-II)

This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the ONE STATEMENT in each group that best describes the way you have been feeling during the PAST TWO WEEKS, INCLUDING TODAY. Bubble in the number beside the statement you have picked. If several statements in the group seem to apply equally well, bubble in the highest number for that group. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness
   ⓪ I do not feel sad.
   ① I feel sad much of the time.
   ② I am sad all the time.
   ③ I am so sad or unhappy that I can't stand it.

2. Pessimism
   ⓪ I am not discouraged about my future.
   ① I feel more discouraged about my future than I used to be.
   ② I do not expect things to work out for me.
   ③ I feel my future is hopeless and will only get worse.

3. Past Failure
   ⓪ I do not feel like a failure.
   ① I have failed more than I should have.
   ② As I look back, I see a lot of failures.
   ③ I feel I am a total failure as a person.

4. Loss of Pleasure
   ⓪ I get as much pleasure as I ever did from the things I enjoy.
   ① I don't enjoy things as much as I used to.
   ② I get very little pleasure from the things I used to enjoy.
   ③ I can’t get any pleasure from the things I used to enjoy.

5. Guilty Feelings
   ⓪ I don't feel particularly guilty.
   ① I feel guilty over many things I have done or should have done.
   ② I feel quite guilty most of the time.
   ③ I feel guilty all of the time.

6. Punishment Feelings
   ⓪ I don't feel I am being punished.
   ① I feel I may be punished.
   ② I expect to be punished.
   ③ I feel I am being punished.

7. Self-Dislike
⓪ I feel the same about myself as ever.
① I have lost confidence in myself.
② I am disappointed with myself.
③ I dislike myself.

8. Self-Criticalness
⓪ I don't criticize or blame myself more than usual.
① I am more critical of myself than I used to be.
② I criticize myself for all of my faults.
③ I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes
⓪ I don't have any thoughts of killing myself.
① I have thoughts of killing myself, but I would not carry them out.
② I would like to kill myself.
③ I would kill myself if I had the chance.

10. Crying
⓪ I don't cry any more than I used to.
① I cry more than I used to.
② I cry over every little thing.
③ I feel like crying, but I can't.

11. Agitation
⓪ I am no more restless or wound up than usual.
① I feel more restless or wound up than usual.
② I am so restless or agitated that it's hard to stay still.
③ I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest
⓪ I have not lost interest in other people or activities.
① I am less interested in other people or things than before.
② I have lost most of my interest in other people or things.
③ It's hard to get interested in anything.

13. Indecisiveness
⓪ I make decisions about as well as ever.
① I find it more difficult to make decisions than usual.
② I have much greater difficulty in making decisions than I used to.
③ I have trouble making any decisions.

14. Worthlessness
⓪ I do not feel I am worthless.
① I don’t consider myself as worthwhile and useful as I used to.
② I feel more worthless as compared to other people.
③ I feel utterly worthless.

15. Loss of Energy
⓪ I have as much energy as ever.
① I have less energy than I used to have.
② I don’t have enough energy to do very much.
③ I don’t have enough energy to do anything.

16. Changes in Sleeping Pattern
⓪ I have not experienced any change in my sleeping pattern.
① I sleep somewhat more than usual.
① I sleep somewhat less than usual.
② I sleep a lot more than usual.
② I sleep a lot less than usual.
③ I sleep most of the day.
③ I wake up 1-2 hours early and can't get back to sleep.

17. Irritability
⓪ I am no more irritable than usual.
① I am more irritable than usual.
② I am much more irritable than usual.
③ I am irritable all the time.

18. Changes in Appetite
⓪ I have not experienced any change in my sleeping appetite.
① My appetite is somewhat less than usual.
① My appetite is somewhat more than usual.
② My appetite is much less than usual.
② My appetite is much greater than usual.
③ I have no appetite at all.
③ I crave food all the time.

19. Concentration Difficulty
⓪ I can concentrate as well as ever.
① I can't concentrate as well as usual.
② It's hard to keep my mind on anything for very long.
③ I find I can't concentrate on anything.

20. Tiredness or Fatigue
⓪ I am no more tired or fatigued than usual.
① I get tired or fatigued more easily than usual.
② I am too tired or fatigued to do a lot of the things I used to do.
③ I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex
⓪ I have not noticed any recent change in my interest in sex.
① I am less interested in sex than I used to be.
② I am much less interested in sex now.
③ I have lost interest in sex completely.

State-Trait Anxiety Inventory – I (STAI-I)

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you feel *right* now, that is, *at this moment.* There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

		Not at all	Somewhat	Moderately so	Very Much So
1.	I feel calm	1	2	3	4
2.	I feel secure	1	2	3	4
3.	I am tense	1	2	3	4
4.	I feel strained	1	2	3	4
5.	I feel at ease	1	2	3	4
6.	I feel upset	1	2	3	4
7.	I am presently worrying over possible misfortune	1	2	3	4
8.	I feel satisfied	1	2	3	4
9.	I feel frightened	1	2	3	4
10.	I feel comfortable	1	2	3	4
11.	I feel self-confident	1	2	3	4
12.	I feel nervous	1	2	3	4
13.	I am jittery	1	2	3	4
14.	I feel indecisive	1	2	3	4
15.	I am relaxed	1	2	3	4
16.	I feel content	1	2	3	4
17.	I am worried	1	2	3	4
18.	I feel confused	1	2	3	4
19.	I feel steady	1	2	3	4
20.	I feel pleasant	1	2	3	4

State-Trait Anxiety Inventory – II (STAI-II)

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the appropriate number to the right of the statement to indicate how you *generally* feel.

		Not at all	Somewhat	Moderately so	Very Much So
21.	I feel pleasant	1	2	3	4
22.	I feel nervous and restless	1	2	3	4
23.	I feel satisfied with myself	1	2	3	4
24.	I wish I could be as happy as others seem to be	1	2	3	4
25.	I feel like a failure	1	2	3	4
26.	I feel rested	1	2	3	4
27.	I am "calm, cool, and collected"	1	2	3	4
28.	l feel that d difficulties are piling up so that I cannot overcome them	1	2	3	4
29.	l worry too much over something that really doesn't matter	1	2	3	4
30.	l am happy	l	2	3	4
31.	I have disturbing thoughts	1	2	3	4
32.	I lack self-confidence	1	2	3	4
33.	I feel secure	1	2	3	4
34.	l make decisions easily	1	2	3	4
35.	l feel inadequate	1	2	3	4
36.	I am content	1	2	3	4
37.	Some unimportant thought runs through my mind and bothers me	1	2	3	4
38.	l take disappointments so keenly that l can 't put them out of my mind	1	2	3	4
39.	I am a steady person	1	2	3	4
40.	I get in a state of tension or turmoil as I think over my recent concerns and interests	1	2	3	4

Treatment History

Current Treatment

Are you currently seeing a therapist for emotional or behavioral problems? ① ③

What type of therapy do you participate in (e.g., counseling, CBT, MBT, family, couples, group, or interpersonal therapy)?

Notes:

Are you currently being prescribed medication for emotional or behavioral problems? ① ③

What type of medication are you prescribed (i.e., name of and purpose of medication and duration taken)?

Notes:

Past Treatment

Have you ever see a therapist for emotional or behavioral problems? ① ③

What type of therapy did you participate in (e.g., counseling, CBT, MBT, family, couples, group, or interpersonal therapy)?

Notes:

Have you ever been prescribed medication for emotional or behavioral problems? ① ③

What type of medication were you prescribed (i.e., name of and purpose of medication and duration taken)?

Notes:

Appendix J. *Session 1 Suicide Risk Assessment*

Questions to ask if you think someone may be at risk for suicide:

Suicidal Ideation:
Are you currently suicidal?

Intent:
Do you think you would ever harm yourself or attempt suicide?

OR

Have you considered ways of killing yourself?

Plan/Preparations:
Do you have a suicide plan or have you made preparations for committing suicide?

Means:
Do you have means to kill yourself?

Suicide Attempt:
Have you ever attempted suicide?

IF YES, THEN
When was your last suicide attempt?

When Students will need to speak with a clinician:

If **intent** + **suicide plan** / **intent** + **means / suicide plan** + **means / suicide attempt within last 2 weeks** + **suicide ideation** = Student NEEDS to **speak with a clinician.**

*Use your judgment. If there is any question about whether a student (who has endorsed one or more of the above items) should speak with a clinician, consult with the clinician. For example, if a student endorses active suicidal ideation, but does not endorse intent or plan, you may still want to touch base with a clinician.

*If Dr. Haigh is unavailable, contact Dr. O'Grady or Dr. Schwartz-Mette. (Contact information on next page).

*If they are unavailable, walk student to the counseling center.

Checking in with students who endorse some of the questions, but DON'T NEED to speak with a clinician (Can use script below but don't have to say this verbatim):

"I noticed that you endorsed (say what they endorsed). There are some very effective ways to help with some of the concerns we spoke about during the interview today. I have a list of referrals you may consider. I would strongly recommend these services to help with the way you have been feeling."

Regardless of whether the student is at risk for suicide or not, offer them the list of mental health referrals.

Contact Information:

Emily Haigh:
207-581-2025 (office); 215-317-0133 (cell)

April O'Grady:
207-945-3935 (home); 207-478-9742 (cell)

Rebecca Schwartz-Mette:
207-581-2048 (office); 573-239-2202 (cell)

Counseling Services at UMaine:
207-581-1392
5721 Cutler Health Center, Room 125
Orono, Maine 04469

Campus Police:
207-581-4040

Appendix K. *Session 2 Consent Forms*

Attention and Elaboration Study: Session 2
The University of Maine at Orono
Informed Consent Document (PSY 100, 212)

You are invited to participate in a research project being conducted by Dr. Emily Haigh in the Department of Psychology at the University of Maine. The purpose of the research is to learn about the emotional and physiological responses related to sad mood. You must be at least 18 years of age to participate.

What Will You Be Asked to Do?

A trained female research assistant will place sensors on your body in order to record electrical activity of the heart, skin, and facial muscle groups. Once the sensors are placed on your body, you will be asked to sit comfortably in front of a computer in a small room. You will then be asked to complete the following tasks: watch a short video about Alaska's Denali Mountain, answer some questions about how you're feeling (e.g. check a box to indicate whether you are *interested, upset, nervous*), complete a short computer task and listen to either a sad or neutral piece of music designed to induce a short-lasting sad mood or no change in mood. This portion of the study will take approximately 1-hour total.

Risks

It is possible that you may feel uncomfortable when answering questions about yourself. At any point during the study, you have the right to skip questions you do not wish to answer, or stop the session and choose not to participate in the remainder of the study. You will not need to provide a reason for stopping the session. You will receive a list of referrals for counseling services at the end of your session today.

Benefits

This study will have no direct benefit to you, though it will help to better understand how individuals process emotional information and how this relates to risk for depression.

Compensation

Students will earn 1 credit for their participation, unless they no longer require research points for course credit (e.g., have already earned 5 research credits as required by PSY 100). In this case, students will receive $15 for their participation. Monetary compensation is only available to students who have met course research credit requirements.

Confidentiality

The code number you have been assigned during session 1 will again be used to protect your identity. This code is stored on a file with software designed to provide additional security. All

data will be kept in the investigator's locked office and will only be accessible by Dr. Emily Haigh and Maine Mood Disorders Lab graduate students and research assistants who have completed training in order to deal with sensitive material. Your name or other identifying information will not be reported in any publications. As previously described, the key linking your name to the data will be destroyed in approximately two years after data analysis is complete, which we anticipate will be in December, 2018. All data will be kept indefinitely by the investigators. The key and the data files will be stored on separate computers. You may decide that you do not want you data used in this research. If you would like you data removed from the study and permanently deleted, please email your request to the Principal Investigator, Dr. Emily Haigh, at emily.a.haigh@maine.edu.

Voluntary

Participation is voluntary**.** If you choose to take part in this study, you may stop at any time. You may also skip any questions you do not wish to answer. If you are participating for monetary compensation, you will receive $15 for participating in this research session to compensate you for your time and travel expenses. If you do not complete the session, you will receive compensation pro-rated to the nearest half hour.

Contact Information

If you have any questions about this study, please contact Emily Haigh at Emily.a.haigh@maine.edu. If you have any questions about your rights as a research participant, please contact Gayle Jones, Assistant to the University of Maine's Protection of Human Subjects Review Board, at 581-1498 (or e-mail gayle.jones@umit.maine.edu).

Your signature below indicates that you have read and understand the above information and agree to participate. You will receive a copy of this form.

_____ _____

Signature Date

Attention and Elaboration Study: Session 2
The University of Maine at Orono
Informed Consent Document (Community Participants)

You are invited to participate in a research project being conducted by Dr. Emily Haigh in the Department of Psychology at the University of Maine. The purpose of the research is to learn about the emotional and physiological responses related to sad mood. You must be at least 18 years of age to participate.

What Will You Be Asked to Do?

A trained female research assistant will place sensors on your body in order to record electrical activity of the heart, skin, and facial muscle groups. Once the sensors are placed on your body, you will be asked to sit comfortably in front of a computer in a small room. You will then be asked to complete the following tasks: watch a short video about Alaska's Denali Mountain, answer some questions about how you're feeling (e.g. check a box to indicate whether you are *interested, upset, nervous*), complete a short computer task and listen to either a sad or neutral piece of music designed to induce a short-lasting sad mood or no change in mood. This portion of the study will take approximately 1-hour total.

Risks

It is possible that you may feel uncomfortable when answering questions about yourself. At any point during the study, you have the right to skip questions you do not wish to answer, or stop the session and choose not to participate in the remainder of the study. You will not need to provide a reason for stopping the session. You will receive a list of referrals for counseling services at the end of your session today.

Benefits

This study will have no direct benefit to you, though it will help to better understand how individuals process emotional information and how this relates to risk for depression.

Compensation

You will receive $15 for your participation.

Confidentiality

The code number you have been assigned during session 1 will again be used to protect your identity. This code is stored on a file with software designed to provide additional security. All data will be kept in the investigator's locked office and will only be accessible by Dr. Emily Haigh and Maine Mood Disorders Lab graduate students and research assistants who have completed training in order to deal with sensitive material. Your name or other identifying information will not be reported in any publications. As previously described, the key linking your name to the data will be destroyed in approximately two years after data analysis is

complete, which we anticipate will be in 2018. All data will be kept indefinitely by the investigators. The key and the data files will be stored on separate computers.

Voluntary

Participation is voluntary. If you choose to take part in this study, you may stop at any time. You may also skip any questions you do not wish to answer. You will receive $15 for participating in this research session to compensate you for your time and travel expenses. If you do not complete the session you will receive compensation pro-rated to the nearest half hour.

Contact Information

If you have any questions about this study, please contact Emily Haigh at Emily.a.haigh@maine.edu. If you have any questions about your rights as a research participant, please contact Gayle Jones, Assistant to the University of Maine's Protection of Human Subjects Review Board, at 581-1498 (or e-mail gayle.jones@umit.maine.edu).

Your signature below indicates that you have read and understand the above information and agree to participate. You will receive a copy of this form.

_____ _____

Signature Date

Appendix L. *Session 2 Self-Report Measures*

Dysfunctional Attitudes Scale – Short Form (DAS-SF I)

The sentences below describe people's attitudes. Circle the number which best describes how much each sentence describes your attitude. Your answer should describe the way you think most of the time.

		Totally Agree	Agree	Disagree	Totally Disagree
1.	If I don't set the highest standards for myself, I am likely to end up a second-rate person.	1	2	3	4
2.	My value as a person depends greatly on what others think of me.	1	2	3	4
3.	People will probably think less of me if I make a mistake.	1	2	3	4
4.	I am nothing if a person I love doesn't love me.	1	2	3	4
5.	If other people know what you are really like, they will think less of you.	1	2	3	4
6.	If I fail at my work, then I am a failure as a person.	1	2	3	4
7.	My happiness depends more on other people than it does me.	1	2	3	4
8.	I cannot be happy unless other people admire me.	1	2	3	4
9.	It is best to give up your own interests in order to pleasure other people.	1	2	3	4

Dysfunctional Attitudes Scale – Short Form (DAS-SF II)

The sentences below describe people's attitudes. Circle the number which best describes how much each sentence describes your attitude. Your answer should describe the way you think most of the time.

		Totally Agree	Agree	Disagree	Totally Disagree
1.	If I am to be a worthwhile person, I must be truly outstanding in at least one major respect.	1	2	3	4
2.	If you don't have other people to lean on, you are bound to be sad.	1	2	3	4
3.	I do not need the approval of other people in order to be happy.	1	2	3	4
4.	If you cannot do something well, there is little point in doing it at all.	1	2	3	4
5.	If I do not do well all the time, people will not respect me.	1	2	3	4
6.	If others dislike you, you cannot be happy.	1	2	3	4
7.	People who have good ideas are more worthy than those who do not.	1	2	3	4
8.	If I do not do as well as other people, it means I am an inferior human being.	1	2	3	4
9.	If I fail partly, it is as bad as being a complete failure.	1	2	3	4

Visual Analogue Scale (VAS)

We are interested in knowing about your current mood. Please mark an ‘X’ on the line below to indicate how you feel right now. Use the labels above the line to help you in your judgment.

Sadness

Stressed

Happy

PANAS-X

This scale consists of a number of words and phrases that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you feel this way. Use the following scale to record your answers:

1 Very Slightly or not at all	2 A Little	3 Moderately	4 Quite of Bit	5 Extremely
_____ cheerful	_____ sad	_____ active	_____ angry at self	
_____ disgusted	_____ calm	_____ guilty	_____ enthusiastic	
_____ attentive	_____ afraid	_____ joyful	_____ downhearted	
_____ bashful	_____ tired	_____ nervous	_____ sheepish	
_____ sluggish	_____ amazed	_____ lonely	_____ distressed	
_____ daring	_____ shaky	_____ sleepy	_____ blameworthy	
_____ surprised	_____ happy	_____ excited	_____ determined	
_____ strong	_____ timid	_____ hostile	_____ frightened	
_____ scornful	_____ alone	_____ proud	_____ astonished	
_____ relaxed	_____ alert	_____ jittery	_____ interested	
_____ irritable	_____ upset	_____ lively	_____ loathing	
_____ delighted	_____ angry	_____ ashamed	_____ confident	
_____ inspired	_____ bold	_____ at ease	_____ energetic	
_____ fearless	_____ blue	_____ scared	_____ concentrating	
_____ disgusted	_____ shy	_____ drowsy	_____ dissatisfied with self with self	

Appendix M. *Debriefing Form*

Debriefing Form for Participation in a Research Study
University of Maine

Thank you for your participation in our study. Your participation is greatly appreciated.

Purpose of the Study:

The purpose of this study is to examine how the way you think and the way your body physiologically responds (e.g. heart rate) to emotional stimuli relates to depression. This study is important because it may help us understand how short periods of sad mood lead some individuals to develop lasting depressed mood.

In this study you completed an interview and several questionnaires about how you think and feel. You also completed an attention task (e.g. computer task) and using sensors to detect electrical impulses we measured physiological arousal (e.g. heart rate) as you listened to music designed to either make you feel sad or no change in your mood.
We expect to find that participants with a history of depression who completed an attention task with negative words and listened to the sad music will report more sad mood and have a stronger physiological response than individuals without a history of depression. Previous research has shown that individuals with depression have difficulty turning their attention away from negative stimuli and have negative repetitive thoughts in response to sad mood; however, little research has examined how these factors relate to physiological functioning.

Do you have any questions about the study? When you were doing the study what did you think the study was about? Was there any part of the study that was difficult? How is your mood now?

We realize that some of the questions asked may have provoked an emotional reaction. As researchers, we do not provide mental health services and we will not be following up with you after the study. However, we want to provide every participant in this study with a comprehensive and accurate list of clinical resources that are available, should you decide you need assistance at any time. Please see information pertaining to local resources at the end of this form.

Confidentiality:

You may decide that you do not want your data used in this research. If you would like your data removed from the study and permanently deleted please email your request to Principal Investigator, Dr. Emily Haigh @ Emily.a.haigh@maine.edu.
Whether you agree or do not agree to have your data used for this study, you will still receive compensation for your participation.

Final Report:

If you would like to learn about the results of the study, let the researcher know and we will email you a summary of the results at the end of the study.

Further Reading(s):

If you would like to learn more about cognitive vulnerability to depression please see the following references:

Farb, N. A. S., Irving, J. A., Anderson, A. K., & Segal, Z. V. (2015). A two-factor model of relapse/recurrence vulnerability in unipolar depression. *Journal of Abnormal Psychology*, *124*(1), 38–53. http://doi.org/10.1037/abn0000031

Key, B. L., Campbell, T. S., Bacon, S. L., & Gerin, W. (2008). The influence of trait and state rumination on cardiovascular recovery from a negative emotional stressor. *Journal of Behavioral Medicine*, *31*(3), 237–248. http://doi.org/10.1007/s10865-008-9152-9

Lethbridge, R., & Allen, N. B. (2008). Mood induced cognitive and emotional reactivity, life stress, and the prediction of depressive relapse. *Behaviour Research and Therapy*, *46*(10), 1142–1150. http://doi.org/10.1016/j.brat.2008.06.011

Useful Contact Information:

If you have any questions or concerns regarding this study, its purpose or procedures, or if you have a research-related problem, please feel free to contact the Principal Investigator, Dr. Emily Haigh at 207-581-2053. If you have other concerns about this study or would like to speak with someone not directly involved in the research study, you may contact the Chair of the Department of Psychology (Dr. Michael Robbins, Michael_Robbins@umit.maine.edu)

If you have any questions concerning your rights as a research subject, you may contact Gayle Jones at the University of Maine Institutional Review Board for the Protection of Human Subjects at (207) 581-1498 or gayle.jones@umit.maine.edu.

(Counseling Resource List Attached – see Appendix D)

BIOGRAPHY OF THE AUTHOR

Olivia Emily Theodosia Anfrosina Depole Bogucki was born in Bristol, Connecticut on December 7, 1989 to Gerard Bogucki, D.M.D. and Jayne Depole-Bogucki, M.A. She was raised in Bristol, Connecticut and graduated from Bristol Central High School in May 2008. She attended the University of Connecticut in Storrs, Connecticut and received her Bachelor of Arts Honors degree in Psychology and Bachelor of Arts degree in Human Development and Family Studies in May 2012. As an undergraduate, she worked in the laboratories of Kimberli R. H. Treadwell, Ph.D. and Dean G. Cruess, Ph.D. at the University of Connecticut and Douglas S. Mennin, Ph.D. at Yale University in New Haven, Connecticut and Hunter College in New York, New York. She co-authored one manuscript and three research presentations at local and national conferences. Before completing her graduate education, she worked at Massachusetts General Hospital in Boston, Massachusetts in the Pediatric Psychopharmacology and Adult ADHD Program. She co-authored two manuscripts and four research presentations at local, national, and international conferences.

Olivia entered the clinical psychology doctoral program at the University of Maine in Orono, Maine in August 2014 under the mentorship of Emily A. P. Haigh, Ph.D. As a graduate student, she conducted research within the Maine Mood Lab, focusing on the cognitive, affective, and physiological processes the contribute to the etiology and maintenance of major depressive disorder. She co-authored one manuscript, one brief, one op-ed, and encyclopedia chapter and four research presentations at local and national conferences. In addition, she served as a student investigator for the Maine-Syracuse Longitudinal Study under the direction of Merrill F. Elias, Ph.D., MPH focusing broadly on depressive symptoms, cardiovascular health and disease, and cognitive and physical functioning. She co-authored two manuscripts, one book

chapter, and seven research presentations at local and national conferences. She was a student member of the Association for Behavioral and Cognitive Therapy (ABCT), Anxiety and Depression Association of America (ADAA), Society for Health Psychology, and Maine Psychological Association and a member of the Scholars Strategy Network. She held multiple administrative and service positions including Associate Director of the University of Maine Psychological Services Center, assistant to the Director of Clinical Training of the University of Maine, student representative of her graduate program, student ambassador for the ABCT, graduate fellow for the Scholars Strategy Network, and chair of multiple committees at the University of Maine.

Olivia received her Master of Arts in Psychology in August 2016 and moved onto doctoral candidacy. She was awarded multiple university-wide and national awards during her graduate education including the University of Maine Correll Fellowship, University of Maine President Susan J. Hunter Teaching Fellowship, Scholars Strategy Network Graduate Fellowship, Maine Academic Prominence Initiative Dissertation Grant, University of Maine Graduate School Government Travel Grant, University of Maine Department of Psychology Travel Grant, Beck Institute for Cognitive Behavior Therapy Graduate Student Scholarship, and National Register of Health Service Psychologists National Psychologist Trainee Register Credentialing Scholarship. In addition, she was invited and granted funding to attend the 45th Annual American Heart Association Ten-Day Seminar on the Epidemiology and Prevention of Cardiovascular Disease and Stroke in Tahoe City, California.

Olivia is currently completing her predoctoral internship at the VA San Diego Healthcare System/University of California, San Diego Psychology Internship Training Program where she focuses on behavioral medicine, primary care mental health integration, spinal cord

injury/disease, and inpatient psychiatry consultation liaison. She served as the co-chief of her internship program. After receiving her degree, she will complete a two-year postdoctoral fellowship in Clinical Health Psychology at the Mayo Clinic in Rochester, Minnesota with a major emphasis on integrated behavioral health. She plans to conduct clinical research on the bidirectional relationship between depression and cardiovascular disease as well as primary, secondary, and tertiary prevention for these conditions in primary and specialty care settings. She is a candidate for the Doctor of Philosophy degree in Psychology from the University of Maine in August 2019.