THE ROLE OF COGNITIVE EMOTION REGULATION STRATEGIES, EMOTIONAL EXPRESSIVITY, AND AMBIVALENCE OVER EMOTIONAL EXPRESSION ON COGNITIVE STRESS APPRAISALS AND CARDIOVASCULAR REACTIVITY DURING AN ACUTE INTERPERSONAL STRESS RECALL TASK

by

Chantel Monique Ulfig

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Associate Professor David K. Chatkoff, Chair
Associate Professor Michelle T. Leonard

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List of Abbreviations

ACTH	Adrenocorticotrophic Hormone
AEQ	Ambivalence Over Emotional Expressiveness Questionnaire
CERQ	Cognitive Emotion Regulation Questionnaire
CRH	Corticotrophin-Releasing Hormone
CVD	Cardiovascular Disease
CVR	Cardiovascular Reactivity
DBP	Diastolic Blood Pressure
EES	Emotional Expressivity Scale
HF	High Frequency
LF	Low Frequency
ms^2	Milliseconds-Squared
nu	Normalized Units
PANAS	Positive and Negative Affect Schedule
RMSSD	Root Mean Square of the Successive Differences
SA Node	Sinoatrial Node
SAM	Stress Appraisal Measure
SBP	Systolic Blood Pressure
SDNN	Standard Deviation of the NN Interval

Abstract

Introduction: Cardiovascular disease (CVD) is the leading cause of death in the United States. Primary risk factors only account for 50% of the new cases of CVD; therefore, other biopsychosocial factors must be involved. While stress and emotions, such as anger and anxiety, have been linked to CVD and changes in cardiovascular reactivity (CVR), the purpose of this study was to examine the associations between maladaptive cognitive emotion regulation strategies, emotional expressivity, and ambivalence over emotional expression with cognitive appraisals of threat/stressfulness and CVR to an acute interpersonal stress recall task. It was hypothesized that: 1) A positive association would be found between maladaptive cognitive emotion regulation strategies and appraisal of threat and stressfulness; 2) A positive association would be found between maladaptive cognitive emotion regulation strategies and SBP, DBP, pulse, LF reactivity, and LF/HF reactivity, and a negative association with RMSSD and HF reactivity; 3) A negative association would be found between emotional expressivity and appraisal of threat and stressfulness; 4) A negative association would be found between emotional expressivity and SBP, DBP, pulse, LF reactivity, and LF/HF reactivity, and a positive association with RMSSD and HF reactivity; 5) A positive association would be found between ambivalence over emotion expression and appraisal of threat and stressfulness; 6) A positive association would be found between ambivalence over emotion expression and SBP, DBP, pulse, LF reactivity, and LF/HF reactivity, and a negative association with RMSSD and HF reactivity; 7) It was unclear how expression, ambivalence, and cognitive appraisals are related, thus a mediational analysis was planned.

Methods: The sample consisted of 82 European Americans ($n = 48$), Arab Americans ($n = 33$), and other ($n = 1$) undergraduate females (Age; M = 20.11, SD = 3.64). Baseline cardiovascular measures were collected including SBP, DBP, pulse, and HRV measures including RMSSD, LF (ms^2), LF (nu), HF (ms^2), HF (nu), and LF/HF. Participants were engaged in a stress recall task while all cardiovascular data were obtained. At the completion of the task each participant completed measures of cognitive appraisal of threat/stressfulness (Stress Appraisal Measure), cognitive emotion regulation strategies (Cognitive Emotion Regulation Questionnaire: CERQ), emotional expressivity (Emotional Expressivity Scale: EES), and ambivalence over emotional expression (Ambivalence over Emotional Expressiveness Questionnaire: AEQ).

Results: No associations were found between the CERQ, EES, or AEQ and blood pressure and pulse reactivity. CERQ-Self-Blame was marginally associated with threat appraisal ($r = .21$), stressfulness appraisal ($r = .18$), LF (nu) ($\Delta R^2 = .03$) and HF (nu) reactivity ($\Delta R^2 = .03$) (all $p < .1$) in the expected direction. CERQ-Rumination had a marginal association with threat appraisal ($r = .21$, $p < .1$), and a statistically significant association with stressfulness appraisal ($r = .31$), LF (nu) reactivity ($\Delta R^2 = .08$), HF (nu) reactivity ($\Delta R^2 = .08$), and LF/HF reactivity ($\Delta R^2 = .08$) (all $p < .01$) in the expected direction. CERQ-Catastrophizing had a statistically significant association with threat appraisal ($r = .37$, $p < .01$), and a marginal association with stressfulness appraisal ($r = .20$, $p < .1$) in the expected direction. The aggregate maladaptive CERQ subscales displayed a statistically significant association with threat ($r = .32$, $p < .01$) and stressfulness ($r = .28$, $p < .05$) appraisals, and a marginal association with LF (nu) reactivity ($\Delta R^2 = .03$), HF (nu) reactivity ($\Delta R^2 = .03$), and lnLF/HF reactivity ($\Delta R^2 = .03$) (all $p < .1$) in the expected direction. Contrary to what was expected, no statistically significant associations were found between the EES and spectral components of HRV. Ambivalence over emotional expression displayed a statistically significant association with appraisal of threat ($r = .31$) and stressfulness ($r = .31$) (all $p < .01$) and a marginal

association with LF (nu) reactivity ($\Delta R^2 = .03$), HF (nu) reactivity ($\Delta R^2 = .03$), and lnLF/HF reactivity ($\Delta R^2 = .03$) (all $p < .1$). Based on the lack of associations for EES, mediational analyses for EES, AEQ, and cognitive appraisals were not conducted. Discussion: In general, maladaptive cognitive emotion regulation strategies and ambivalence over emotional expression predicted cognitive appraisals of threat and stressfulness and normalized units of spectral components. Thus, these results may suggest that these emotional concepts are important within the context of CVD. Interestingly, emotional expressivity, contrary to expectations, did not predict either cognitive stress appraisals or CVR, and may not play a significant role in CVD. Also, unexpectedly, no associations were found with blood pressure. [Keywords: Cognitive appraisal, cardiovascular reactivity, heart rate variability reactivity, cognitive emotion regulation, emotional expressivity, ambivalence over emotional expression].

Chapter I

Introduction

Cardiovascular disease (CVD) is the leading cause of death in the United States affecting approximately 40% of the adult population (American Heart Association, 2001). CVD is defined as disorders affecting the heart and circulatory systems, including coronary heart disease and cerebrovascular disease (Foreyt & Carlos Poston II, 1996). From a global standpoint, CVD resulted in 17.5 million deaths in 2005, which accounted for an astonishing 30% of all deaths worldwide (Chida & Steptoe, 2010), and 38% of all deaths in North America (Hansson, 2005). CVD is the number one leading cause of death for European men under the age of 65, and the second most common cause of death for European women (Hansson, 2005). Only half of the cases of CVD can be predicted from typical primary risk factors, such as family history, smoking, obesity, hypertension and diabetes. Since these primary risk factors only account for 50% of the new cases of CVD, there must be other biopsychosocial factors involved (Trieber et al., 2003).

The psychosomatic hypothesis of stress and health states that psychological stress weakens the body's optimal functioning ability and leads to disease and mortality (Kiecolt-Glaser et al., 2002; McEwen, 1998). It has been debated in the literature whether psychosocial factors can *cause* coronary artery disease as an etiological mechanism or represent a process that exacerbates the underlying disease process. However, the literature provides evidence that there is an *association* between psychological stress and cardiovascular reactivity and disease (Schwartz et al., 2003). Psychological stress plays a

significant role on CVD by potentially causing excessive activation of the sympathetic nervous system and related cardiac events (Rozanski, Blumenthal & Kaplan, 1999). There is no plausible way to completely remove stress from an individual's life, and since psychological stress has been shown to be such an active aggravator of cardiac problems, it is an important topic of study. From a behavioral medicine perspective, determining the associations between stress responses, biopsychosocial factors, and CVD may be highly valuable in increasing awareness and understanding of the disease process in its entirety, as well as better predicting pre-clinical diagnoses and courses of action to remedy or postpone the progression of disease (Hilmert, Ode, Zielke, & Robinson, 2010). The biopsychosocial model provides a structure for considering the multiple mechanisms involved in the etiology and progression of CVD. Although a significant number of studies have examined the role of hostility, anger, stress, depression, and anxiety on the etiology and progression of CVD, less attention has been devoted to the way that individuals manage their emotional states, emotional expression, and whether or not they are ambivalent over their emotional expression, and the relationship to physiological reactions, in particular to cardiovascular reactivity (CVR). Determining whether these emotion concepts are related to greater CVR can provide potential insight into the etiology of CVD and have important clinical implications regarding maladaptive cognitive strategies and stress management. Previous studies have attempted to determine a link between personality "traits" and CVR, as well as "state" emotional experiences and CVR, yet this has resulted in somewhat inconsistent findings (Andreassi, 1997; Habra, Linden, Anderson, & Weinberg, 2003; Lawler, Schmied, Armstead, & Lacy, 1990). This study examines the associations between maladaptive cognitive emotional regulation

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strategies, emotional expressivity, and ambivalence over emotional expression, with individual's cognitive appraisal of threat and stressfulness, and their CVR in response to an acute interpersonal stress recall task.

Physiology of Cardiovascular Disease

Pathologically, coronary artery disease or coronary atherosclerosis is the underlying cause of most myocardial infarctions. Atherosclerosis is an inflammatory syndrome where cholesterol, immune components, and waste products accumulate in the peripheral blood vessels resulting in a progressive narrowing of the blood vessels. This results in a restriction of blood flow interfering with the ability to supply oxygenated blood to the heart (Foreyt & Carlos Poston II, 1996; Gianaros & Sheu, 2009). The atherosclerosis process in its entirety involves multiple events; including endothelial damage, generation of adhesion molecules, platelet adherence, collection of monocytes and lymphocytes on the arterial wall, proinflammatory cytokines, and accumulation of lipids which may form stable or unstable plaques (Black & Garbutt, 2002). Atherosclerosis can ultimately result in arrhythmias (irregular cardiac rhythms), angina (chest pain), hypertension, heart failure and myocardial infarctions (heart attacks) (Gianaros & Sheu, 2009). Environmental, emotional, and behavioral factors have been associated with the progression of plaque development and with the likelihood that plaques in the atherosclerotic arteries will rupture and cause blood clots (Gianaros & Sheu, 2009), resulting in a myocardial infarction. Psychological stress, associated with an increase in blood pressure and sympathetic nervous system arousal, in particular, has been implicated in the genesis and progression of atherosclerosis (Black & Garbutt, 2002; Matthews et al., 1986; Steptoe & Kivimaki, 2012).

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Psychosocial Risk Factors for CVD

While emotions and psychological stress in particular have been associated with the genesis and progression of CVD and atherosclerosis, there is not one agreed upon definition of psychological stress. As a construct, stress has been conceptualized using many models and the associations between the various models of stress and CVD are mixed. The following provides a background of the three main models of stress, by Cannon, Selye, and Lazarus, often covered in the cardiovascular literature.

Models of stress.

Cannon: fight-or-flight and homeostasis. Even though Walter Cannon did not use the term "stress," he was still considered influential to the concept and understanding of stress from a physiological standpoint. When studying animal models, Cannon determined that environmental threats activated the sympathetic nervous system, which caused an increase in catecholamines, helping to prepare the animal for danger in order to be able to fight or to flee the situation (Cannon, 1932 as cited in Holmes et al., 2006). Cannon coined the term "fight-or-flight" when describing these animal's responses to outside threats. He also developed the concept of "homeostasis" as representing the idea that human bodies have many mechanisms simultaneously functioning to maintain internal constancy and resist change. Cannon stated that homeostasis was important for survival. The body's responses of the sympathoadrenal system to outside influences represented "a response to threats to homeostasis." He viewed the sympathetic nervous system (particularly when the body is releasing adrenaline and catecholamines by the adrenal cortex in response to activation of sympathetic nerves) as the body's physical

adaptation and response to dangerous situations (Cannon, 1929 as cited in Filaretova, 2012).

Selye: general adaptation syndrome. Hans Selye was another major contributor to the concept of stress. Selye proposed the General Adaptation Syndrome, as three stages of coping that an individual goes through when they encounter a stressor. The three stages include an alarm stage, where an individual incurs a fight-or-flight response and acute symptoms surrounding an anxiety response occurs; adaptation, where the individual tries to resist the stressor, and may display some disappearance of acute symptoms; and exhaustion, with loss of resistance accompanied by physical and mental depletion (Selye, 1936 as cited in Filaretova, 2012). Selye defined stress as "a nonspecific response of the body to any demand presented to it" and also introducing the concept of a "stressor" as the agent that elicits the bodily response of stress (Selye, 1976 as cited in Filaretova, 2012, p. 195).

Prolonged exhaustion can result in physical diseases of the body (Goldstein & Kopin, 2007). Feelings of exhaustion and loss of energy are two main premonitory symptoms typically associated with myocardial infarction and sudden cardiac death (Kop, 1999). In studies assessing premonitory symptoms and myocardial infarctions, it was revealed that signs of exhaustion differentiated those patients who have had a myocardial infarction and those who have not (Appels, Falger, & Schouten, 1993; Falger, 1992). An additional study conducted on coronary angioplasty patients indicated; one and a half years after their cardiac surgery, patients who reported states of exhaustion prior to the angioplasty procedure had more than a two-fold increased risk of new cardiac events occurring (Kop, Appels, Mendes de Leon, de Swart, & Bar, 1993).

Lazarus: cognitive appraisal. Of particular importance to the current study is the concept of stress proposed by Lazarus; defined as the process by which individuals perceive and respond to stressors that are appraised as harmful, threatening, or challenging. According to Lazarus and Folkman's (1984), transactional model of stress and coping, the way an individual interprets internal or external stimuli affects the impact of these events. Appraisal of events refers to the individual's unique and specific interpretation of the situation to evaluate and characterize the significance of the events experienced. Appraisals may occur quite rapidly and may occur outside of an individual's awareness. Once a situation is interpreted as important, a biologically based emotion process will begin, based on the appraisal of the situation. Changes in psychological, behavioral, and physiological domains occur, that prepare the individual to respond in an adaptive manner based on the perceived situation. The cognitive-relational theory and transactional model of stress are based on the view that the individual and the environment have a bidirectional relationship, which is mutually reciprocal. The integration of both the individual and environment is imperative when appraising an event. Appraisal of an event depends on a specific environment that is evaluated by a specific person, with their own unique psychological characteristics (Folkman, Lazarus, Gruen, & DeLongis, 1986).

Primary appraisals involve the individual's assessment of the significance of the event to their own personal well-being (if there is potential harm or benefit to their health or well-being) (Folkman, Lazarus, Gruen, & DeLongis, 1986). The events encountered can be primarily appraised as irrelevant, benign-positive, or stressful. Based on cognitive-relational theory, three forms of stress appraisals can be distinguished; harm/loss, threat,

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and challenge. Harm/loss appraisals occur after the event has already occurred, while threat and challenge appraisals are relevant to anticipatory events (Lazarus, 1966; Lazarus & Folkman, 1984). Secondary appraisal then refers to the individual's evaluation of what can be done (coping) in response to the situation to overcome, prevent harm, or to improve the situation (Folkman, Lazarus, Gruen, & DeLongis, 1986). The transactional model suggests that the physiological stress response (e.g. sympathetic activation, parasympathetic withdrawal) of an individual is partially determined by the personal relevance of an event and the type of cognitive appraisal (e.g. threat, challenge) made in response to that event (primary appraisal), along with the available coping resources and strategies (secondary appraisal).

Based on both the transactional model of stress and the cognitive-relational theory, Peacock & Wong (1990) developed the Stress Appraisal Measure (SAM), which assesses both primary and secondary appraisal to anticipatory stress. Three dimensions of primary appraisal of anticipatory stress are deciphered between that include: threat, challenge, and centrality. Threat appraisal involves the "potential for harm/loss in the future," challenge appraisal involves "the anticipation of gain or growth from the experience," and centrality refers to "the perceived importance of an event for one's well-being" (Peacock & Wong, 1990, p.228). Three secondary appraisal dimensions include the individual's perception that the situation is controllable-by-self, controllable by others, and uncontrollable-by-anyone. The SAM, which is used in the current study, also includes a scale of overall perceived stressfulness (Peacock & Wong, 1990).

Stress, cognitive appraisals, and physiological responses. Since, Lazarus's model of stress is of particular importance to this study, the cardiovascular responses

typically associated with different cognitive appraisals of stress will be reviewed in greater detail.

The challenge and threat theory (Blascovich & Tomaka, 1996) suggests that not all autonomic reactivity is automatically a sign of distress. When an event is appraised as threatening or challenging, both circumstances can cause an increase in heart rate, indicating that a response is activated (Mendes, Reis, Seery, & Blascovich, 2003). This theory suggests that only CVR responses associated with the cognitive appraisal of *threat* would be indicative of pathological reactivity responses. The appraisal of *challenge* is likely to be beneficial for the individual (Blascovich & Tomaka, 1996). Therefore, based on the challenge and threat theory, individuals who evaluate the environment or task as *threatening*; including danger, uncertainty, required effort, and exceeding their coping resources of knowledge, abilities, and support; have increased activation of the sympathetic-adrenal-medullary system (which causes an increase in left ventricular contractility), as well as, activation of the pituitary-adrenal-cortical axis (which inhibits vasodilation and often produces vasoconstriction.) Vasoconstriction along with inhibition of vasodilation of arteries increases total peripheral resistance. In contrast, the appraisal of *challenge* is associated with a slightly different, more beneficial cardiovascular response. *Challenge* is also associated with activation of the sympathetic-adrenal-medullary system, increased left ventricular contractility, and increased cardiac output, (similar to a threat cardiovascular response), but a challenge appraisal cardiovascular response is also associated with increases in epinephrine, vasodilation, and a decrease in total peripheral resistance (Blascovich & Tomaka, 1996). When appraising a situation as *challenging*, the cardiovascular response is typically associated with quicker

cardiovascular recovery, while appraisal of *threat* is associated with slower cardiovascular recovery (Dienstbier, 1989), indicating extended activation of the sympathetic nervous system. Thus, the challenge and threat theory suggests that cognitive appraisals of threat are related to physiological reactivity (due to vasoconstriction and inhibition of vasodilation), indicating a potential important link between threat appraisal, CVR, and CVD. The cognitive appraisal of challenge on the other hand that has been associated with perceptions of having the ability to effectively cope, producing vasodilation and therefore, display a beneficial physiological response (Mendes, Reis, Seery, & Blascovich, 2003).

Ethnic differences have also been found in the literature between cognitive appraisal and CVR. In an investigation conducted by Chatkoff & Leonard (2009), examining differences between European Americans and Arab Americans on cognitive appraisal and CVR, it was indicated that in response to a mental arithmetic stress task, Arab Americans reported greater threat and stressfulness appraisals than European Americans. When utilizing a stress recall task, Arab Americans only reported greater threat appraisal than their European American counterparts. Arab Americans displayed less CVR and lower systolic and diastolic blood pressure, during a stress recall task when compared to European Americans, as well as, slower heart rate recovery (Chatkoff & Leonard, 2009). This study displays support for potential ethnic differences in associations between cognitive appraisals and CVR.

Whether considering Cannon's model, Selye's general adaptation syndrome, or Lazarus's threat appraisal concept of stress, all models agree that there are physiological consequence of stress. The body, initially, reacts to perceived stressors through several

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brain regions. The brain-body pathway shows how psychological stress can produce physiological changes and increased risks for diseases. There are several levels-of-response that are activated from stress related activity including; the corticolimbic systems, midbrain and brainstem relay pathways and neuromodulatory systems, and peripheral target organs. The corticolimbic systems (cingulate, insula, and amygdala) are involved with the cognitive appraisal of stressors and regulate stress induced blood pressure changes. When a situation is appraised as threatening, the corticolimbic systems then signal the midbrain and brainstem relay pathways, as well as the neuromodulary systems to produce adaptive coping behaviors (Gianaros & Sheu, 2009). These adaptive coping behaviors driven through changes in peripheral sympathetic and parasympathetic systems are then thought to affect the appropriate target organ (Gianaros & Sheu, 2009).

The above activation of the sympathetic nervous system results in several other effects including activating the sympatho-adreno-medullary system and the hypothalamic-pituitary-adrenocortical system. The sympatho-adreno-medullary system releases catecholamines and the hypothalamic-pituitary-adrenocortical system releases glucocorticoids. The "fight-or-flight" response involves the hypothalamus secreting corticotrophin-releasing hormone (CRH). CRH causes the pituitary to release adrenocorticotrophic hormone (ACTH). ACTH along with activation from the reticular formation results in sympathetic activation. The sympathetic nervous system innervates the adrenal medulla resulting in the release of epinephrine and norepinephrine into the bloodstream. The ACTH, acting as a hormone, also causes the adrenal cortex to release corticosteroids to return the body to its original homeostatic levels (Bonfiglio et al., 2011). The stress hormones, epinephrine and norepinephrine, cause the "fight-or-flight

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response" of the sympathetic nervous system. The physiological effects that occur, in response to the flood of stress hormones, are increased breathing, increased heart rate, and increased blood pressure (Juster, McEwen & Lupien, 2010). Prolonged stress results in high levels of the stress hormones, that if elevated long enough can damage the brain and body.

The stress response evolved from the inflammatory response, which is the most primitive of the body's protective mechanisms (Black & Garbutt, 2002). The physiological reactions that occur in response to the body's stress response help the body fight off infections. That is, the corticosteroids and catecholamines, that are some of the major stress hormones, also initiate the production of cytokines which are involved in the inflammatory response. Thus, the inflammatory response, which is related to atherosclerosis, as mentioned above, is actually contained within the stress response, and it is suggested that recurrent stress causes endothelial damage and platelet adhesions in the walls of arteries (Black & Garbutt, 2002). Endothelial injury in the arteries can be produced because of large increases in blood pressure as well as higher levels of cortisol resulting from the body reacting to a stressor (Bairey, Krantnz, & Rozanski, 1993).

When the sympathetic nervous system is working in overdrive there are excessive amounts of stress hormones, inflammation, elevated cortisol levels, and other indirect behavioral changes that may reduce baroreflex performance (Lucini, Fede, Parati & Pagani, 2005). Baroreflex is a negative feedback mechanism that maintains blood pressure near its homeostatic set point. Baroreflex controls blood pressure by adjusting the sympathetic and parasympathetic nervous systems control over heart rate and cardiac output. Stretch information from cardiopulmonary baroreceptors and chemoreceptors

signal changes in blood pressure and regulates this negative feedback loop (Gianaros & Sheu, 2009). Baroreflex performance is a critically important protective factor in regards to cardiac autonomic reflex mechanisms. When this protective factor is decreased, this leads to higher risk of hypertension (Lucini, Fede, Parati & Pagani, 2005).

Timing of stress. Both acute, short lived stress, and chronic, long lasting stress, has been associated with the risk of developing CVD. Some of the types of chronic stress related to CVD and cardiovascular related mortality are marital stress, caregiving stress, and low socioeconomic status stress (Schwartz et al., 2003). Acute stress on the other hand is of particular importance to this current study. In the laboratory, acute psychological stress is typically induced and studied through the use of several tasks, such as; verbal mental arithmetic tasks (subtracting by seven from a large three digit number), public speaking tasks, and stress recall tests (recalling a stressful event, trying to re-live it and describe in as much detail as possible) (Brown et al., 2007; Chatkoff, Maier, & Klein, 2010; Waldstein et al, 1999).

Acute stress. Acute stress is short lived and may cause short term increases in blood pressure, increases in heart rate, reduce the necessary threshold for cardiac arrhythmia to occur, impair endothelial functioning, and possibly induce sudden cardiac death (Lucini, Fede, Parai & Pagani, 2005). Many forms of acute stress have previously been studied. One type of acute stress studied in the literature is bereavement. Bereavement was found to be associated with an increased risk of future cardiac events (Kaprio, Koskenvuo & Rita, 1987). Cardiac related mortality rates were the highest immediately after a person entered the bereavement phase. There was a two-fold higher risk for men and a three-fold higher risk for women in this study (Kaprio, Koskenvuo &

Rita, 1987). Another example of an acute life stress is an earthquake. A study on the Los Angeles earthquake in 1994 showed a dramatic increase in sudden cardiac deaths the day the earthquake occurred. During an average day, 4.6 sudden cardiac deaths occurred; however, on the exact day of the earthquake the cardiac death rate rose to 24 deaths (Leor, Poole & Kloner, 1996). Even though events such as earthquakes do not have a daily or monthly occurrence, one incident can have profound effects on an individual's health. In a study analyzing heart rate following a CO_2 inhalation test on students undergoing the natural stressor of an academic examination, those in the examination group displayed significantly higher heart rate reactivity, higher perceived stress, higher stress scores, lower salivary cortisol levels, and slower systolic blood pressure recovery than those in the non-exam group (Loft et al., 2007). Thus, even less extreme, transient, natural occurrences of acute stress in individuals' daily lives can alter cardiovascular responses.

Recall of a stressful event and CVR. Physiological reactions to stress are not only experienced at the exact moment the stressor is encountered, but there are also emotions, behaviors, and physiological reactions that occur before the stressor, and remain activated after the stressor ends. Psychological and physiological responses may occur and recur for long periods of time, such as if the individual ruminates over the event or re-appraises the event. Following a stressful encounter, individuals may ruminate and reflect on the causes and consequences related to the distressing event, perpetuating the cardiovascular response (Schwartz et al., 2003). Additionally, and in line with the transactional model, imagined or recalled stressors can elicit a response. For example, when participants were asked merely to recall a stressful event and imagine it as vividly as possible, they showed

increased blood pressure (Glynn, Christenfeld, & Gerin, 2002). The recall of a stressful event can evoke the same physiological reactions as actually experiencing the event. Sustained cardiovascular activation, greater reactivity to stress and a slower recovery from a stressful event is associated with an increased risk for future cardiovascular diseases (Chida & Steptoe, 2010). The current study, will assess laboratory induced acute stress, in the form of an acute interpersonal stress recall task, and the resulting cognitive appraisals of threat/stressfulness and CVR response, as well as the association with various emotion concepts in participants.

      Emotions. Emotions are another psychosocial factor related to stress and CVD. "What is an emotion?" was first discussed by William James in 1884. The difficulty in the literature today is that there are multiple definitions of emotions, similar to how there are multiple definitions of stress. Current models postulate that the definition of emotion incorporates several important aspects. It is proposed that emotions are 1) responses to both external stimuli, as well as, internally developed mental representations, 2) involve changes in the experiential system, behavioral system, and physiological system, 3) are separate and distinct from "moods", (since emotions are thought to have behavioral response tendencies towards a specific identifiable trigger or objects, whereas moods may give rise to action tendencies, but often affect cognitions rather than actions), 4) can include both learned and unlearned responses to a stimuli, and 5) appraisal processes of the situation can be involved to help determine the individuals interpretation of the significance of the stimuli along with their goals (Gross, 1998; Ochsner & Gross, 2005). Some important distinguishing features separating emotions from other closely related terms are that e*motions* are relatively short lived, verses *emotion episodes,* which are

longer lived and extended in both time and space, (Gross, 1998) and *emotions* are fluctuating, while *moods* are more pervasive and sustained in nature (APA, 1994).

Emotions are thought to serve several different functions, such as facilitating individuals decision making processes, preparing the individual for a physiological motor response, and providing information regarding the connection between the individual and the environment. Emotions also serve social functions, such as, providing information on others behavioral intentions and having an effect on determining individual's social behaviors (Gross, 1998).

Biological Risk Factors Linking Psychological Stress & Emotions to CVD

Cardiovascular reactivity (CVR). Many studies have examined CVR to stressful stimuli as a risk factor for CVD. CVR is defined as a change from a resting state in hemodynamic activity as a result of a psychological or physical challenge, and refers to the magnitude of the cardiovascular response (Gianaros & Sheu, 2009; Andreassi, 1997). Many aspects of CVR have been assessed, including blood pressure, heart rate, cardiac output, and endothelial function. Heart rate and blood pressure are the typically measures of CVR in the literature (Sherwood & Turner, 1992). Throughout this paper, CVR refers to both blood pressure reactivity as well as heart rate variability reactivity.

Growing evidence suggests that *excessive* activation of the sympathetic nervous system in response to chronic psychosocial stress is related to CVD. Sympathetic nervous system hyper-reactivity is also known as CVR. The "reactivity hypothesis" proposes that CVR may play an important role in the development of CVD (Treiber et al., 2003). The normal sympathetic nervous system response increases both heart rate and blood pressure, however, this can become exaggerated with stress and prolonged when the

individual encounters repeated stressors over time. In several studies, the accumulation of atherosclerosis in the coronary artery occurred at a faster rate in those who had excessive and exaggerated heart rate and blood pressure responses to a stress induced challenge (Rozanski, Blumenthal & Kaplan, 1999).

Interestingly, CVR has been associated with brain regions involved in psychological stress, cognitive appraisals, and emotions. More specifically neuroimaging testing has indicated that cardiac changes in blood pressure, heart rate, and heart rate variability (HRV) is directly correlated with neural activity in the cingulate cortex, insula, and amygdala. The role of the cingulate cortex is to support cognitive and emotional functions, as well as regulating stress induced blood pressure reactivity. Specifically, the perigenual anterior cingulate cortex is important in appraising challenging demands, developing aversive behavioral states, and regulating autonomic response to stressors. The posterior cingulate cortex is important in the appraisal of events and monitoring the environment for stressful and threatening stimuli. The insula is activated in response to aversive events and stimuli. The role of the insula is to send efferent and afferent neuronal connections to areas of the brain that control and innervate the heart and vasculature. Brain imaging evidence suggests that the insula is involved with cardiovascular regulation, by way of the sympathetic, parasympathetic, and baroreflex pathways. The amygdala of the brain is involved with processing emotions and stressors. The amygdala regulates cardiovascular reactivity by controlling the baroreflex. The baroreflex then controls changes in blood pressure by regulating and adjusting sympathetic and parasympathetic control over heart rate, cardiac output, and vascular resistance. If there is increased activity of the corticolimbic system there is typically

exaggerated cardiovascular reactivity (Gianaros & Sheu, 2009). Next, literature on various types of reactivity (blood pressure reactivity and HRV reactivity) will be discussed.

Blood pressure reactivity. Blood pressure reactivity is one of the most consistent and significant predictors of atherosclerosis and coronary heart disease. A change in blood pressure is a typical cardiovascular reaction to a stressor. This change in blood pressure occurs when sympathetic and parasympathetic nervous system activity is adapted to direct the blood away from the viscera and towards the muscles to prepare an individual to escape from the potential stressor (Gianaros & Sheu, 2009). The degree of CVR is predictive of later development of CVD (Light, Dolan, Davis, & Sherwood).

A study examining individuals with early blood pressure reactivity and later development of high blood pressure, suggests that blood pressure reactivity in children is predictive of blood pressure as adults. In 1937, the blood pressure reactivity of 300 children between the ages of 7 and 17 were measured, and then assessed again 45 years later in 1982. In 1982, 71% of those who had hyper-reactive blood pressure as children, later had high blood pressure as adults, as compared to the 19% of those who were normal blood pressure reactors as children and later developed high blood pressure, suggesting the stability of CVR (work of Hines, cited by Sallis, Dimsdale and Caine, 1988). Matthews, Woodall, & Allen's (1993) study examined blood pressure responses in adults and their children. The study indicated that larger systolic and diastolic blood pressure responses in adults to a mental and physical laboratory challenge was associated with increased resting *diastolic blood pressure* 6.5 years later. When assessing the children of these adults, the greater the systolic and diastolic changes in blood pressure in

response to a laboratory challenge, the higher the resting *systolic blood pressure* was 6.5 years later at follow-up. This study proposed that individuals who have exaggerated stress induced blood pressure reactivity at a younger age are at higher risk for elevated blood pressure in the future (Matthews, Woodall, & Allen, 1993). Another study, examined the blood pressure responses to three different psychological challenges (cold pressor, star tracing, and video game task), and future incidences of hypertension in black and white men and women enrolled in the coronary artery risk development in young adults (CARDIA) study. Results from the CARDIA study indicated that compared to whites, blacks had greater increases in diastolic blood pressure when conducting the cold pressor task and video game task, and smaller increases in systolic blood pressure during the star tracing task. Furthermore, compared with women, men had greater increases in diastolic blood pressure during the cold pressor task and star tracing task, as well as, greater increases in systolic blood pressure during the video game task and star tracing task. Overall, the study showed that the greater the systolic and diastolic blood pressure changes were during all of the psychological challenges, the earlier the occurrence of hypertension (Matthews et al., 2004). The CARDIA study also assessed whether blood pressure changes during psychological stress (utilizing a star tracing task and a video game task) predicted coronary calcification thirteen years later in young healthy adults. This study indicated that blood pressure reactivity to the video game psychological stress task predicted the presence of coronary calcification thirteen years later in adults who are absent of hypertension and diabetes. The association between blood pressure reactivity was dependent on the type of psychological stressor utilized, such that the star tracing

task did not predict calcification. It was unclear why the development of coronary calcification is differed by type of stressor (Matthews, Zhu, Tucker, & Whooley, 2006).

Heart rate variability reactivity. Heart rate variability is generally defined as the overall variability in the timing between heartbeats. This variability is heavily influenced by sympathetic and parasympathetic innervation of the sinoatrial node of the heart (SA node) (Chandra, Yeates, & Wong, 2003). This node controls activation of the heartbeat. Other factors such as barosensors also affect HRV via influence on the peripheral nervous system. There are two broad classifications of measures of HRV including time dependent measures and frequency domain spectral analysis measures. Time dependent measures include the standard deviation of the time intervals between heartbeats (SDNN) and the root mean square of the successive differences (RMSSD); a measure of variability between successive heartbeats as measured between R-R intervals of adjacent QRS peaks in an electrocardiogram recording. The QRS complex represents the electric events of a heart beat that are typically seen on an electrocardiogram recording. Spectral analysis on the other hand, allows for measures of the relative contribution of the sympathetic and parasympathetic systems on overall variability (Task Force, 1996).

When conducting a spectral analysis to determine the relative contribution of the different branches of the autonomic nervous system, Fast-Fourier (including all data) or autoregressive math techniques (attempting to eliminate noise) can be conducted on R-R interval data to yield spectral power bands (Berntson et al., 1997; Chandra, Yeates, & Wong, 2003). Typically, power within the range of 0.15 - 0.40Hz is considered high frequency (HF) and represents a mix of sympathetic and parasympathetic activity, while frequencies in the range of 0.04 - 0.15Hz are considered low frequency (LF), and

represent sympathetic contributions (Task Force, 1996). Some controversy exists regarding whether LF rhythms represent fluctuations of mainly the sympathetic nervous system communicating with the SA node, or whether LF rhythms reflect fluctuations in both the sympathetic and parasympathetic branches (Berntson, et al., 1997; Goedhart et al., 2008). A majority of research suggests that LF represents HRV from both of the branches, yet increased sympathetic activation. The ratio of LF/HF displays a measure of the balance between the sympathetic and parasympathetic nervous systems (Liao et al., 1995). Thus, spectral analysis yields quantitative data on sympathetic and parasympathetic contributions to HRV.

Based on complexity theory, system variability is vital for individuals' stability, adaptability, and health (Thayer & Sternberg, 2006). For the body to achieve optimal functioning, it is necessary to have flexible regulation of energy expenditure. A static imbalance may increase the chances of the individual becoming vulnerable to pathology. Rigid regularity is associated with poor health outcomes such as disease, mortality, and morbidity (Thayer & Sternberg, 2006). Normal heart rate rhythm is controlled by membrane processes of the cardiac SA node, which is controlled by both the sympathetic and parasympathetic nervous system (Berntson, et al., 1997). An individual's heart rate normally varies beat-by-beat based on parasympathetic innervation of the heart, which is transmitted from the brain after communicating with the vagus nerve. When autonomic imbalance occurs, where one of the autonomic branches dominates over the other branch, there is a lack of flexibility within the system. In response to stressful situations, the sympathetic nervous system typically dominates and is hyperactive, while the parasympathetic nervous system is hypoactive, potentially leading to pathological

conditions. Therefore, when conveying the same concept to HRV, low HRV is associated with a greater risk of disease and death (Thayer & Sternberg 2006).

   Heart rate variability and CVD. It has been suggested that decreases in parasympathetic modulation may expose the heart to unrestricted stimulation by the sympathetic nervous system, increasing vulnerability to develop atherosclerosis, coronary artery disease and arrhythmias (Gorman & Sloan, 2000). Overall, low HRV is associated with sudden cardiac death (Gorman & Sloan, 2000). An indirect correlation is seen between HRV and blood pressure variability. Increased HRV is associated with decreased blood pressure variability (Gorman & Sloan, 2000).

   The Atherosclerosis Risk in Communities (ARIC) study is a longitudinal study that collected data on cardiovascular and pulmonary diseases by the National Heart, Lung, and Blood Institute, obtaining data on 15,800 individuals to determine if there were age, race, or sex differences in autonomic cardiac function. Based on the ARIC study, those who were older tended to have lower levels of LF HRV compared to younger age groups. LF HRV was also lower in blacks than in white, and was lower for women than in men. In regards to HF HRV, population levels of HF HRV were lower in older groups, levels of HF HRV were higher in blacks than in whites, and levels of HF HRV were similar for men and women. The HF/LF ratio was similar in both older age groups and younger age groups, was higher in blacks than in whites, and was higher in women than in men. This study is consistent with previous studies displaying that LF and HF is inversely associated with age, women had lower LF and higher HF/LF ratios than men, and blacks had lower LF, higher HF, and higher HF/LF ratios when compared to whites (Liao et al., 1995).

Emotion Related Concepts

While there is a substantial body of literature on the association between psychological stress and CVD, as well as the association between emotions, such as anger and anxiety to CVD, relatively few studies have examined the role of emotion management strategies on cardiovascular functioning and CVR to acute stressors. To this author's knowledge there are no published studies on the association between the emotional concepts of cognitive emotion regulation, emotional expressivity, and ambivalence over emotional expression and spectral components of HRV and HRV reactivity, which are important factors in cardiovascular health.

Emotional regulation. The capacity for individuals to control their emotions is thought to be essential for human adaptation (Oschner & Gross, 2005). Emotion regulation has been referred in the literature as the processes by which an individual influences which emotions they have, when they have the emotions, and how the emotions are experienced and expressed (Gross, 1998). This definition includes five important aspects: 1) Individuals may maintain, increase, or decrease both positive and negative emotions, 2) there may or may not be overlap in neural emotion circuits, and there can be differences in emotional regulation processes across different emotions, 3) this definition focuses on the individual's regulation of self, not attempting to influence others' emotions, 4) there is a continuum between conscious and unconscious emotion regulation, (from conscious, effortful, and controlled regulation, to unconscious, effortless, and automatic regulation), and 5) there are no assumptions that emotion regulation is intrinsically good or bad (Gross, 1998).

The term emotional regulation has been defined in various ways. Emotional regulation can be described in terms of *behavioral* emotional regulation actions, as well as *cognitive* emotional regulation strategies. Emotional regulation can also be used to refer to two different conceptualization of emotion regulation; 1) the regulation of emotions themselves or 2) the regulation of something else by using emotions (Gross & Munoz, 1995). The conceptualization of cognitive emotion regulation will be further reviewed.

Cognitive emotion regulation. *Cognitive emotion regulation* as described by Garnefski, Kraaij, & Spinhoven (2001), which is the focus of the current study, refers to the conscious, cognitive way of managing and controlling the intake of emotionally arousing information. Cognitive emotion regulation is thought to fall under the broader category of emotion regulation; "all the extrinsic and intrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions" (Gross, 1999; Thompson 1994, p.27 as cited in Garnefski & Kraaij, 2007). There can be vast differences between cognitions that individuals use to regulate their emotions. Cognitive emotion regulation strategies refer to individual's thoughts and cognitions following the experience of a threatening or stressful event, and the cognitive methods that are used to change, control, or maintain an emotional state (Garnefski & Kraaij, 2007).

Garnefski & Kraaij (2007) distinguished on a theoretical and empirical basis between nine different cognitive emotion regulation strategies that occur in response to a stressful or threatening event: 1) self-blame, 2) acceptance, 3) rumination, 4) positive refocusing, 5) refocusing on planning, 6) positive reappraisal, 7) putting into perspective, 8) catastrophizing, and 9) blaming others, and developed the Cognitive Emotion

Regulation Questionnaire (CERQ). *Self-blame* is defined as "thoughts of putting the blame for what you have experienced on yourself." *Acceptance* is defined as "thoughts of accepting what you have experienced and resigning yourself to what has happened." *Rumination* refers to "focusing on your thoughts and thinking about the feelings and thoughts associated with the negative event." *Positive refocusing* refers to "thinking about joyful and pleasant issues instead of thinking about the actual event." *Refocusing on planning* is defined as "thinking about what steps to take and how to handle the negative event." *Positive reappraisal* refers to "thoughts of creating a positive meaning of the event in terms of personal growth." *Putting into perspective* is defined as "thoughts of brushing aside the seriousness of the event/emphasizing the relativity when comparing it to other events." *Catastrophizing* refers to "thoughts of explicitly emphasizing the terror of what you have experienced." Finally, *blaming others* refers to "thoughts of putting the blame for what you have experienced on the environment or another person" (Garnefski & Kraaij, 2007). The CERQ is the most comprehensive measure that has made a separate distinction between cognitive and behavioral emotion regulation strategies, and solely measures cognitive regulation strategies.

An important distinction must also be made between cognitive emotion regulation and cognitive coping, which are two narrowly related concepts. Cognitive emotion regulation theory considers cognitive emotion regulation as purely cognitive and separate from behavioral strategies. Coping involves problem-focused coping, as well as emotion-focused dimensions, which includes both cognitive and behavioral strategies (Garnefski et al., 2001; Garnefski, van den Kommer et al., 2002; Garnefski & Kraaij, 2007). Other measures of coping often involve a mixture of both cognitive and behavioral components,

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such as the COPE measure; (Carver, 1997) and the Emotion Regulation Questionnaire (Gross & John, 2003).

Cognitive emotion regulation and psychological/physiological effects. In the literature, the conceptualization of the terms self-blame, rumination, and catastrophizing, may vary slightly, therefore, only prior research related directly to self-blame, rumination, and catastrophizing (theoretically maladaptive cognitive emotion regulation strategies) as measured and defined by the CERQ will be presented. Within the psychological disorders, anxiety and mood disorders are partially related to ineffective regulation of persistent and negative emotions (Campbell-Sills, Barlow, Brown, & Hofmann, 2006). Within the cognitive emotion regulation strategies, researchers have been unable to state that specific cognitive emotion regulation strategies are always healthy and beneficial, while others are always unhealthy and harmful; however, theoretically more adaptive strategies have been related to better outcomes, and theoretically more maladaptive strategies have been related to poorer psychological and physiological outcomes (Garnefski et al., 2001). Nevertheless, in general, the more cognitive emotion regulation strategies an individual uses, regardless if they are theoretically described as adaptive or maladaptive, the more depression and anxiety symptoms are reported (Garnefski & Spinhoven, 2001; Garnefski, Teerds, Kraaij, Legerstee, & Kommer, 2004). Results from Garnefski & Spinhoven (2001) further indicate that after controlling for the influence of the other variables, the maladaptive strategies of self-blame, rumination, and catastrophizing were positively related to depression and anxiety and an increase in symptoms reported. This may indicate the potential importance of further studying these maladaptive cognitive emotion regulation

strategies since, as mentioned above, depression and anxiety has been related to increased CVR and CVD.

In a study conducted by Martin & Dahlen (2005), multiple hierarchical regressions were conducted to analyze which of the cognitive emotion regulation strategies would best predict depression, anxiety, stress, and anger. Results indicated that depression was predicted by self-blame, rumination, catastrophizing, acceptance, and low positive reappraisal. Anxiety was predicted by the same cognitive emotion regulation strategies, excluding acceptance. Stress was predicted by self-blame, rumination, and low positive reappraisal. Anger was predicted by rumination, catastrophizing, and low positive reappraisal. These results coincide with coping theory that predicts that engagement in maladaptive cognitive regulation styles increases stress/distress experienced (Lazarus, 1993; Lazarus & Folkman, 1984, Martin & Dahlen, 2005). Another study displayed similar results indicating higher levels of rumination and catastrophizing are related to increased stress and reporting of excessive worry in both males and females, while self-blame was related to worry for females only (Zlomke & Hahn, 2010). A majority of the studies in the literature have focused mainly on general distress and mood and anxiety disorders, and have not directly researched blood pressure reactivity and spectral components of HRV.

Emotional expressivity. Similar to other terms in the emotion literature, there have been several different conceptualizations of emotional expression, which will be reviewed. In 2000, Gross, John & Richards defined emotional expression in terms of nonverbal actions: "behavioral changes that usually accompany emotion, including the face, voice, gestures, posture, and body movement (p. 712)." Later, in 2001, Kennedy-

Moore & Watson defined emotional expression as "observable verbal and nonverbal behaviors that communicate or symbolize emotional experience (p. 187)." Throughout the literature, some researchers refer to only behavioral expressivity as emotional expressivity, assessing the tendency to express emotions in a behavioral manner, while others in the literature focus on the verbal expression of emotions, defining it as "the process of translating the emotional message into words, whether in the written or spoken channel (Berry & Pennebaker, 1998, p.70)."

There have been inconsistent findings in the literature regarding how emotional expressivity should be defined and evaluated. Some studies have found emotional expression to best be predicted by a one-factor model, while other studies, have found several important factors involved in the concept of emotional expressivity. In a study by Barr, Kahn, & Schneider (2008), exploratory factor analysis suggested that seven emotion-expression factors including affect intensity, ambivalence about expression, disclosure of negative emotion, disclosure of emotion, disclosure of lack of affect, expression of positive emotion, and secret keeping, are explained by two main second-order factors: Emotional constraint and emotional expression. The Emotional Expressivity Scale (EES) on the other hand, developed by Kring, Smith, and Neale (1994), defined emotional expressivity as a one-factor model, measuring emotional expressivity in general. Emotional expressivity as a stable characteristic was conceptualized as the extent to which individuals' outwardly display their emotions, including facial, verbal, or other behaviors that are brought about from individual's emotions (Dobbs, Sloan, Karpinski, 2007; Kring, Smith, & Neale, 1994). The definition of emotional expression identified in the current study will be referring to this

conceptualization, as emotional expressivity in general, regardless of the valence of the expression (positive or negative) and regardless of the mode of expression (facial, verbal, or gestural). A significant gender difference has been shown with regards to emotional expressivity as measured by the EES, with females scoring significantly higher than males (Kring, Smith, & Neale, 1994).

There are several different methods that have been used to assess an individual's emotional expressivity; observational coding systems where researchers code the participant's emotional expression, psychophysiology methods of facial electromyography activity to assess emotional expression (e.g., Deschamps, Schutte, Kenemans, Matthys, & Schutter, 2012), and participant self-report (e.g.; the emotional expressivity scale and the Berkeley expressivity scale) (Gross & John, 1995; Kring, Smith, & Neale, 1994). Several benefits of self-report measures are that emotional expression can be assessed both inside and outside the laboratory, and can assess different modalities of emotional expression such as facial expressions, body gestures, vocalization, and individual's perceptions of emotional expression. However, drawbacks of self-report measures of emotional expression include the necessity of the individual having an accurate perception and reflection on their emotional expressivity, the ability to recall, and reporting biases (Dobbs, Sloan, & Karpinski, 2007).

Emotional expressivity and psychological/physiological effects. The literature has typically focused on emotional expressivity in regards to the psychological and physiological benefits of disclosure. Vast amounts of research suggest that talking or writing about emotions is beneficial, since it can foster added insight and increase supportive interactions with others (Kennedy-Moore & Watson, 2001). Emotional

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expression has been related to higher life satisfaction (Kamal, Rehman, Ahmad, & Nawaz, 2013; Stanton, Kirk, Cameron, Danoff-Burg, 2000). Physical health benefits suggest that emotional expression is helpful from a biological standpoint, such that blood pressure is reduced, muscle tension is lessened, and immune functioning is increased (Esterling et al., 1994, Pennebaker et al., 1988; Petrie, Booth, & Pennebaker, 1998).

Expression of distressing information has been shown to have both positive and negative consequences in relation to distress. In a review of how emotional expression may alleviate distress, Kennedy-Moore & Watson (2001) indicated that there are three main possible mechanisms through which expression can alleviate distress. The first is through experiential and cognitive learning. For example, individuals who have experienced traumatic events may be overwhelmed or afraid of expressing their emotions. Psychotherapy utilizing expression has been shown to help clients learn that their painful feelings are not unbearable, to change their maladaptive thoughts and beliefs about their emotional responses, and to perceive their distress as tolerable and manageable rather than debilitating. The second way expression may alleviate distress is by eliciting added insight. When individuals put their emotions into words, it has been shown to help individuals understand, recognize, and interpret their feelings, by providing better clarity. Expression may also lead to added insight by changing the individual's appraisal of the event. Thirdly, expression may alleviate distress by enhancing social support and social relationships with others.

Conflicting research suggests that emotional expression is also related to aversive effects, such as increasing intrusive thoughts, increasing distress, sustaining the grief process, and immunosuppression (Mendes, Reis, Seery, & Blascovich, 2003). Self-

disclosing potentially stigmatizing events may bring about some adverse consequences. Feelings of shame, embarrassment, rejection, or betrayal can occur if the recipients of the disclosure of emotion respond negatively (Kennedy-Moore & Watson, 2001). Some of these conflicting results in research may depend on the definition of emotional expressivity used, the content of disclosure, and context of the environment where emotional expression has taken place.

Stanton, Kirk, Cameron, & Danoff-Burg (2000) conducted a study where participants viewed a stress-inducing film, and were randomly assigned to either talk about facts from the film or to talk about their emotional reactions. This same procedure was completed for a second time, 48 hours later, and during this phase participants were free to discuss facts or emotions. Results indicated that individuals who reported both disruptive thoughts and expressed their emotions had lower autonomic nervous system arousal after discussing their emotions during the second phase. A dose-response relationship was found where, the more the participants talked about their emotions the lower the physiological arousal. Participants in the fact condition displayed an inverse relationship. This study suggests that attending to emotions allows for habituation to a stressful situation to occur and lower autonomic arousal, which can be suggestive of a lesser stress response and lesser CVR.

In a study examining physiological responses during emotional expression, and challenge and threat cardiovascular patterns, it was found that emotional expression to an empathetic same-sex stranger brought about a *challenge* cardiovascular pattern of reactivity (beneficial reactivity). Results assessing opposite-sex dyads indicated that emotional expression elicited patterns of cardiovascular *threat* (pathological reactivity).

These findings suggest that gender and context of emotional expression may influence CVR effects (Mendes, Reis, Seery, & Blascovich, 2003). Some hypothesized mechanisms for this difference in physiological responses include gender differences, perceived support, and comfort and familiarity (Mendes, Reis, Seery, & Blascovich, 2003).

  Ambivalence over emotional expression. Pennebaker (1985) suggested that lack of emotional expression is not only what is responsible for psychological and physical distress; but that it is the combination of lack of expression along with a desire to express that is related to distress. The general concept of "ambivalence" refers to "rapidly changing or simultaneous intense and opposing emotional feelings towards an object" (Raulin, 1984 as cited in King, 1998, p. 753). Ambivalence over emotional expression is defined as conflict over one's style of emotional expression. Individuals may be expressive or inexpressive; however, they may still be in conflict over their desire and style of expression (King & Emmons, 1990). There are three domains of ambivalence over emotional expression as shown in Figure 1: Domain 1) individuals who are inexpressive and are inhibiting their desire to express their emotions, Domain 2) individuals who are expressive, when they do not desire to express their emotions, and Domain 3) individuals who are expressive and later regret their emotional expression. Ambivalence incorporates both inhibition as well as rumination (King & Emmons, 1990). Pennebaker (1985) coined the term "active inhibitor" to refer to the process of willfully preventing oneself from their desired action. The "active inhibition" is then related to individuals experiencing obsessive thoughts regarding their inhibited action.

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Ambivalence over emotional expression can be geared both towards negative as well as positive emotions (King, 1998). Individuals may avoid expressing their emotions following the experience of repeated negative consequences. These individuals may be using avoidance in order to protect themselves from negative personal consequences such as rejection, criticism, or humiliation (Kennedy-Moore & Watson, 2001). This expressive avoidance may be continually negatively reinforced by the absence of negative consequences when the individual does not disclose emotional information. The ambivalence construct should distinguish between healthy and unhealthy expressive styles, such as for individuals who are relaxed and quiet verse those who are repressed and tense inhibitors (King & Emmons, 1990).

A gender difference has also been found in relation to ambivalence over emotional expression. Women have been found to be both more expressive and have increased ambivalence over their emotional expression than men. It has also been shown that women were specifically more expressive and ambivalent of positive emotions (King & Emmons, 1990).

Ambivalence over emotional expression and psychological/physiological effects. Ambivalence over emotional expression has previously been viewed as an important mediator between individual's emotional styles and both their psychological and physical health, and has been related to psychological distress (King & Emmons, 1990). Prior research has indicated that individuals who inhibit their desire to express themselves regarding traumatic life events are at a greater risk for the development of social and physiological problems (King & Emmons, 1990). In King and Emmons's study (1990) ambivalence over emotional expression was negatively correlated with measures of

psychological well-being and positively correlated with measures of psychological ill-being. Ambivalence over emotional expression was negatively correlated with life satisfaction and self-esteem, and was positively correlated with daily negative affect, obsessive compulsive tendencies, depression, paranoid ideation, and phobic anxiety. Partial correlation coefficients were calculated between the AEQ and measures of well-being while controlling for emotional expression (as measured by the Emotional Expressiveness Questionnaire), and none of the partial correlations decreased, thus indicating ambivalence is crucial in the association (King & Emmons, 1990). In a study conducted by Barr, Kahn, & Schneider (2008) on emotional expression and comfort with expression and psychological distress, greater ambivalence over emotional expression and greater secret keeping was associated with greater general distress and positively related to anxious arousal.

Ambivalence over emotional expression was also shown to be positively correlated to negative affect and confusion in reading emotions in others. In a study conducted by King (1998), individuals with low levels of ambivalence who were highly expressive reported themselves as the least confused over reading others emotions, those who were inexpressive and non-ambivalent reported some confusion over reading facial expressions, while individuals who were highly ambivalence over emotional expression indicated confliction in their own emotions, as well as, regarding other's emotions. It was suggested that the individual's conflicting feelings may lead to mistrust in others' expressions and that individuals who are ambivalent over their own emotional expression tend to be in conflict with understanding others' emotions (King, 1998). The ability to perceive emotions is important in emotional competence and emotional development.

Ambivalence over emotional expression has been negatively related to marital satisfaction and dissatisfaction with social support (King, 1998), and positively related to depression (Brockmeyer et al., 2013). Emmons & Colby (1995) found that individuals who are highly ambivalent over their emotional expression tend to be less likely to benefit from social support and less likely to receive social support from others. Individuals high in ambivalence may also be more likely to utilize maladaptive coping strategies to cope with stressful life events such as escaping into fantasy, distancing, self-blame, and other blame, which are associated with poorer psychological adjustment (Tucker, Winkelman, Katz, & Bermas, 1999). Studies have also indicated a positive association between ambivalence over emotional expression and levels of depression. In a study conducted by Brockmeyer et al. (2013) assessing a clinical sample of patients with major depressive disorder verse a control group, those diagnosed with major depressive disorder reported greater ambivalence over emotional expression, and displayed positive correlations between levels of ambivalence and levels of depression.

The above review reflects that most studies on emotional ambivalence and health have focused on psychological factors as opposed to physiological mechanisms. However, it should be noted that many of the psychological factors examined, such as depression and anxiety, have been linked to CVD etiology or progression, via altered CVR (Mussleman, 1998).

The Present Study

As described, a substantial body of literature has demonstrated an association between stress, cognitive appraisals associated with stress, and CVR potentially contributing to significant CVD events. Additionally, emotions such as anger and anxiety

have also been tied to CVD via changes in CVR (Guerrero & Palmero, 2010; Kop, 1999; Rozanski, Blumenthal, & Kaplan, 1999; Smith & Glazer, 2004). There has also been evidence to support the associations between maladaptive cognitive emotion regulation strategies, emotional expressivity, and ambivalence over emotion expression to anxiety, depression, and general distress (and these psychological factors have in turn been linked to greater CVR). However, only a relatively few number of studies have examined the role of some cognitive emotion regulation strategies on cardiovascular functioning and CVR to acute stressors, and no published studies to this authors knowledge have examined the possible associations between emotional expressivity and ambivalence over emotion expression to cognitive appraisals of threat/stressfulness and CVR. A few studies have shown that cognitive emotion regulation strategies, such as rumination and cognitive reappraisal, have been found to predict CVR including measures of preejection period, cardiac output and time domain measures of HRV (Denson et al., 2011; Mauss et al., 2007; Ray et al., 2008). Despite these studies, several cognitive emotion regulation strategies have not been examined within the context of acute stress induced CVR. In addition, spectral components of HRV and HRV reactivity, important factors in cardiovascular health (Thayer, Yamamoto, & Brosschot, 2010), have not been examined as outcome variables in emotional expression and emotion management studies.

Given that the maladaptive cognitive emotion regulation strategies have been related to anxiety, depression, stress, and anger (Garnefski & Spinhoven, 2001; Martin & Dahlen, 2004) and that coping theory predicts engagement in maladaptive regulation styles increases distress, (Lazarus & Folkman, 1984; Martin & Dahlen, 2005) along with the finding that self-blame, rumination, and catastrophizing have been related to

increased stress and worrying (Zlomke & Hahn, 2010), it would be reasonable to hypothesize that maladaptive cognitive emotion regulation strategies may be associated with cognitive appraisals of threat/stressfulness and to increased CVR. Also, since emotional expressivity has vast amounts of research on the benefits of emotional expression/disclosure in reducing blood pressure, and reducing muscle tension (Esterling et al., 1994; Pennebaker et al., 1988; Petrie, Booth, & Pennebaker, 1998 as cited in Mendes, Reis, Seery, & Blascovich, 20003), it would be reasonable to hypothesize that emotionally expressive individuals should show lesser cardiovascular reactivity when involved in an acute stress task. If the individual gains clarity and insight from their emotional expression this may alleviate distress, and provide habituation to the stressful situation and lower automatic arousal (Stanton, Kirk, Cameron & Danoff-Burg, 2000), which can be suggestive of reduced cognitive appraisals of threat/stressfulness and lesser CVR. Greater ambivalence over emotion expression has been associated with general distress and anxious arousal, less life satisfaction, and depression (Barr, Kahn, & Schneider, 2008; King & Emmons, 1990). Given that ambivalence over emotional expression has been related to confusion in reading emotions in others (King, 1998) and highly ambivalent individuals are less likely to benefit from social support and receive social support from others (Emmons & Colby, 1995), these individuals may have poorer psychological adjustments to stressors. Someone who is ambivalent over their emotional expression may find it threatening to be in a situation where they are required to be more emotionally expressive, therefore, it would be expected that in the face of an acute stress recall task requiring emotional expressivity these individuals may appraise the task as threatening/stressful, thus increasing arousal. For example, those individuals who tend to

fall within the domain of being inexpressive despite their desire to express their emotions may cognitively appraise the interpersonal interaction as threatening with the potential for ridicule, shame, embarrassment, betrayal, or vulnerability (Kennedy-Moore & Watson, 2001; Mongrain & Zuroff, 1994). Also, the inhibition of emotional expression may prevent the individual from gaining positive interactions with others, and may prevent them from adapting to challenging situations and from appraising an event as challenging rather than threatening or stressful. Thus, it would be reasonable to hypothesize that a positive association between ambivalence, cognitive threat and stressfulness appraisal, and CVR would be expected. Finally, given that it has been suggested that emotional expressivity and ambivalence over emotional expression may interact, a potential mediating effect between the constructs should be examined.

The purpose of the present study is to examine the associations between maladaptive cognitive emotion regulation strategies, emotional expressivity, and ambivalence over emotional expression to cognitive threat and stressfulness appraisals. In addition, the possible association between maladaptive cognitive emotion regulation strategies, emotional expressivity, and ambivalence over emotional expression to CVR, specifically blood pressure and pulse reactivity and HRV reactivity, will be assessed.

Hypothesis of the Present Study.

Hypothesis 1: *A positive association will be found between the cognitive emotion regulation strategies of self-blame, rumination, catastrophizing, and appraisal of threat and stressfulness to an acute stress recall task, and a positive association will be found between the aggregate cognitive emotion regulation strategies (sum of self-blame, rumination, and catastrophizing) and appraisal of threat and stressfulness to an acute*

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stress recall task. An association is not expected with the appraisal of challenge to an acute stress recall task.

Hypothesis 2: *A positive association will be found between the individual cognitive emotion regulation strategies (self-blame, rumination, and catastrophizing), as well as the aggregate cognitive emotion regulation strategies (sum of self-blame, rumination, and catastrophizing) and SBP, DBP, pulse, LF cardiovascular reactivity, and LF/HF reactivity, and a negative association with RMSSD and HF cardiovascular reactivity.*

Hypothesis 3: *A negative association will be found between emotional expression and appraisal of threat and stressfulness to an acute stress recall task. An association is not expected with the appraisal of challenge to an acute stress recall task.*

Hypothesis 4: *A negative association will be found between emotional expression and SBP, DBP, pulse, LF cardiovascular reactivity, and LF/HF reactivity, and a positive association with RMSSD and HF cardiovascular reactivity.*

Hypothesis 5: *A positive association will be found between ambivalence over emotional expression and appraisal of threat and stressfulness to an acute stress recall task. An association is not expected with the appraisal of challenge to an acute stress recall task.*

Hypothesis 6: *A positive association will be found between ambivalence over emotional expression and SBP, DBP, pulse, LF cardiovascular reactivity, and LF/HF reactivity, and a negative association with RMSSD and HF cardiovascular reactivity.*

Hypothesis 7: *It is unclear how the variables of emotional expression, ambivalence over emotional expression, and threat appraisal are related to each other.*

In order to test competing models we will run two mediating analysis. The first, with ambivalence over emotional expression as the mediating variable between emotional expression and threat appraisal. The second, with emotional expression as the mediating variable between ambivalence over emotional expression and threat appraisal.

Chapter II

Methods

Participants

Data for the current study included a final sample of 82 undergraduate students attending the University of Michigan-Dearborn, enrolled in introductory psychology courses in the behavioral sciences department. There were initially 103 participants who gave consent for the study and completed a demographics questionnaire (Appendix A). From those 103 participants, 18 were excluded based on the exclusion criteria (see below), two were excluded because of equipment failure, and one additional participant was excluded based on outlier analysis with LF (ms^2) at 5.2 standard deviations and LF/HF at 7.5 standard deviations. Participants were recruited and screened via University of Michigan-Dearborn Introductory Psychology Pool (SONA), an online undergraduate participation system, where students were informed of available research studies on campus, and were given the opportunity to receive course credit for their participation. This study is part of a larger study assessing differences in European Americans and Arab Americans females on cardiovascular reactivity and recovery following a stress recall task. Due to the needs of the larger study, the current study was only open to females, age 18 and older, identifying as European American or Arab American. The current study consisted of females who identified as European Americans ($n = 48$), Arab American ($n = 33$), and other ($n = 1$). Participants had an average age of $M = 20.110$ years ($SD = 3.641$, range = 18 - 42).

Participants were excluded from the entire study if they had a family history of heart attack or stroke prior to the age of 50, had any type of implanted medical device, or had medical or psychiatric conditions that would affect cardiovascular functioning (including: high blood pressure, heart attack, chest pain, irregular heartbeat, stroke, cardiovascular problems, asthma, diabetes, kidney disease, and psychiatric disorders). Participants were also excluded from the entire study if they took medications that could affect cardiovascular functioning (e.g. stimulants, steroids, anti-inflammatory medications, blood pressure medications, anti-depressants, anti-anxiety, mood stabilizers, or other psychiatric medications), over the counter medications for cold, flu, pain, or allergy, and if they were pregnant or breastfeeding. Participants who arrived to the study and did not meet the inclusion and exclusion criteria were immediately excluded from the remainder of the study and were given partial credit for their participation. Participants were also excluded from the study if they have consumed caffeine or alcohol in the last twelve hours, or any tobacco products in the last three hours.

Research Design

A correlational study was conducted examining associations between the variables of interest. An a priori power analysis was conducted to determine the necessary sample size which indicated a need for 80 participants. The sample size of 80 was determined based on previous studies analyzing blood pressure reactivity, not based on HRV data. It was realized that this may be an insufficient sample size to find significant effects for spectral components of HRV, given the large standard deviations associated with spectral components, (described in the physiological measures and HRV software section of the methods section), however, the sample size of 80 was chosen

because, an ideal sample size of 150, would be unfeasible due to the time constraints for completing a master's level thesis.

Measures

Demographic and screening questionnaire. The demographics and screening questionnaire was completed by all the participants to determine if the individual qualified to participate in the study based on the exclusion and inclusion criteria. The demographics and screening questionnaire is a 22-item questionnaire to assess participant's age, ethnicity, religion, exclusion criteria related to the current study, as well as behavioral questions related to smoking, caffeine, and exercise.

Positive and negative affect schedule (PANAS 1 & 2). (Appendix B) The Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988) is a 5-point scale that contains 31-words that describe different positive and negative feelings and emotions. Positive and negative affect are highly distinctive dimensions, and the PANAS is suggested to provide independent measures of positive and negative affect (Crawford & Henry, 2004). Positive affect refers to pleasurable engagement with the environment, where the participant feels energetic, enthusiastic, active, and alert. High positive affect entails an individual with high energy, pleasurable engagement, and complete concentration. Negative affect reflects unpleasurable engagement, general subjective distress, and multiple aversive mood states underlying general subjective distress, such as lethargy, sadness, anger, disgust, guilt, fear, and nervousness. Participants rate their self-reported mood with a 5-point Likert scale (1 = very slightly or not at all, 2 = a little, 3 = moderately, 4 = quite a bit, and 5 = extremely). Participants were asked to complete the PANAS on two separate occasions. Participants completed PANAS 1 following baseline

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measurement, prior to the stress recall task. They were instructed to "Indicate how you predominately feel *right now*." Participants then completed the PANAS 2 immediately following the stress recall task. Participants were then instructed to "Indicate how you predominately felt *during the recall task*." These measures were used to assess if the stress recall task did elicited a change in affect in the participants, as a manipulation check. In the current study, a 20-item scoring of the PANAS was utilized. High internal consistency, Cronbach's alpha's, for state measures of affect using the PANAS have been reported at .84 or greater. Cronbach's alphas have ranged from .86 to .90 for positive affect, and from .84 to .87 for negative affect (Watson, Clark & Tellegen, 1988). In the current study, Cronbach's alpha for PANAS 1 assessing positive affect was .84, and assessing negative affect was .72. Cronbach's alpha for PANAS 2 assessing positive affect was .84, and assessing negative affect was .80.

<u>Stress appraisal measure (SAM).</u> (Appendix C) Cognitive appraisal of threat to an acute stress recall task was measured using the threat and challenge subscales of the stress appraisal measure (SAM), as well as a scale to index overall stressfulness (Peacock & Wong, 1990). The questionnaire consisted of 12-items, instructing the participants to answer regarding "your thoughts about the *recall task*, and how you view this situation *right now*." Each subscale consists of four items using a 5-point Likert scale (1 = not at all, 2 = slightly, 3 = moderately, 4 = considerably, and 5 = extremely). The threat subscale is designed to measure the perceived harm or loss from a situation, whereas the challenge subscale primarily taps the perceived growth or gain from a situation. Some sample questions include "to what extent can I become a stronger person because of this problem?", "Does this situation make me feel anxious?", and "Does this situation tax or

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exceed my coping resources?" Validation studies indicate that these subscales tap relatively distinct dimensions of appraisal related to the overall stress experience. The reliabilities of the scales were assessed, displaying Cronbach's alpha's ranging from .74 to .81 (Peacock & Wong, 1990). In the current study, SAM-Threat displayed a Cronbach's alpha of .60, which is acceptable. The SAM-Challenge subscale displayed a Cronbach's alpha of .55, which should be interpreted with caution because it represents poor internal consistency. SAM-Stressfulness displayed a Cronbach's alpha of .60, which is acceptable. Two participants were missing one item on the SAM, and mean substitutions were used for these participants.

Cognitive emotion regulation questionnaire (CERQ). (Appendix D) Cognitive emotion regulation strategies used in response to the experience of a stressful life event were measured using The Cognitive Emotion Regulation Questionnaire (Garnefski et al., 2001; Garnefski, Kraaij, & Spinhoven, 2002). This is a 36-item questionnaire that consists of nine subscales, each subscale containing four items. The nine subscales that are assessed in the complete 36-item questionnaire are Self-blame, Acceptance, Rumination, Positive Refocusing, Refocus on Planning, Positive Reappraisal, Putting into Perspective, Catastrophizing, and Blaming Others. In the current study, based on potential cardiovascular implications, only the three maladaptive subscales; Rumination, Catastrophizing, and Self-blame totaling 12-items were assessed. The instrument utilizes a 5-point Likert scale (1 = almost never, 5 = almost always). A sample item from the Rumination subscale is "I am preoccupied with what I think and feel about what I have experienced." A sample item from the Catastrophizing subscale is "I often think that what I have experienced is the worst that can happen to a person." A sample item from the

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Self-Blame subscale is "I feel that I am the one who is responsible for what has happened." Cognitive emotion regulation strategies have been found to have strong and consistent relationships with emotional problems, depression, and anxiety. Psychometric studies indicate a high internal consistency, Cronbach's alpha ranging from .75 to .86 at first measurement and .75 to .87 at follow-up. Specifically for the subscales of rumination with adults a Cronbach's alpha of .83 was reported, the catastrophizing subscale reported a Cronbach's alpha of .79, and the self-blame subscale reported a Cronbach's alpha of .75. After a one year follow-up, test-retest reliabilities of the subscales were good, with Pearson correlations between first and second measurements of .55 for the self-blame subscale, .60 for the rumination subscale, and .61 for the catastrophizing subscale (Garnefski & Kraaij, 2007). The current study displayed high internal consistencies. Current analysis revealed that the CERQ- Self-Blame subscale indicated a Cronbach's alpha of .87. The CERQ-Rumination subscale indicated a Cronbach's alpha of .74. The CERQ-Catastrophizing subscale indicated a Cronbach's alpha of .85. An aggregate of the maladaptive cognitive emotion regulation strategies (the sum of self-blame, rumination, and catastrophizing) was also assessed in the current study, which has not previously been validated. This scale obtained a good internal consistency with a Cronbach's alpha of .88.

Emotional expressivity scale (EES). (Appendix E) Self-reported measures of emotional expressivity were measured using the EES (Kring, Smith, & Neale, 1994). This is a 17-item self-report measure intended to measure individual differences in emotional expressivity and outward display of emotion, in general. This scale assesses a one factor model of emotional expressivity. This measure is not intended to measure

specific content of the emotional expression. Participants rate their responses on a 6-point Likert scale (1 = never true, 6 = always true). Some sample items from the EES include "Even when I'm experiencing strong feelings, I don't express them outwardly," "I can't hide the way I am feeling," and "I am able to cry in front of other people." Psychometric studies indicate a high internal consistency, with Cronbach's alpha of .93 (Dobbs, Sloan, & Karpinski, 2007), as well as a high test retest reliability, convergent validity, and discriminant validity (Kring, Smith, & Neale, 1994). In the current study, similar reliability scores were obtained. The Cronbach's alpha for items on the EES was .92, displaying high internal consistency.

  Ambivalence over emotional expressiveness questionnaire (AEQ). (Appendix F) The Ambivalence over Emotional Expressiveness Questionnaire utilizes a "personal striving" framework, and measures ambivalent emotional strivings. Ambivalence over emotional expression can occur in various forms such as, wanting to express but not being able to, expressing but not wanting to, and expressing emotions and later regretting the expression. The AEQ is a 28-item self-report trait measure assessing the conflict within the individual, between the desire to express information and their actual behavior of expression. Participants rate their response on a 5-point Likert scale (1 = never feel this way, 2 = occasionally feel this way, 3 = sometimes feel this way, 4 = often feel this way, and 5 = frequently feel this way). Participants were informed to answer each item based on its overall meaning; if a statement consisted of two thoughts participants were told to only give the item a high rating if both thoughts applied to them. For example, some of the items included in the AEQ were "When I am really proud of something I accomplished I want to tell someone, but I fear I will be thought of as conceited," "Often

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I'd like to show others how I feel, but something seems to be holding me back," and

"When someone bothers me, I try to appear indifferent even though I'd like to tell them

how I feel." Psychometric studies have indicated a high internal consistency with a

Cronbach's alpha of .89 and a test-retest correlation of .78 (King & Emmons, 1990). The

current study also displayed high internal consistency, with a Cronbach's alpha of .91.

One participant was missing one item on the AEQ, and a mean substitution was

calculated for this item.

Level of task engagement. Participant's level of task engagement was measured

using a subjective rating from both the researcher and the research assistant. Task

engagement was rated on a 3-point scale, with 1 = low task engagement, 2 = medium task

engagement, and 3 = high task engagement. The participant's level of task engagement

was then determined by taking the average of the researcher and research assistant's

subjective scoring. In the current study, the participants displayed a task engagement of

$M = 1.793$ ($SD = 0.657$). Of the 82 participants, participants receiving a task engagement

rating of 1 ($n = 24$), 1.5 ($n = 13$), 2 ($n = 27$), 2.5 ($n = 9$) and 3 ($n = 9$) was obtained.

Counting emotion words. The number of emotion words a participant used

during the stress recall task was measured using a counter as an additional objective

measure of emotional expressivity and participant task engagement. The research

assistant counted the total number of emotion words the participant used during the stress

recall task, including only negative emotion words and physiological reactions used to

describe an emotional state. The participant must have verbally stated the emotion word.

It was not included in the emotional count if the participant agreed with an emotion word

the researcher reflected back to them, without directly verbalizing the emotion. Research

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assistants were provided with exact directions and examples of which words should be counted as emotion words (Appendix G). In the current study, the number of emotion words used by a participant during the stress recall task indicated $M = 8.34$ ($SD = 3.907$, range $0 - 18$). This method of counting emotion words was not previously validated, and should be interpreted with caution.

Physiological measures and heart rate variability software. Blood pressure was collected using a Critikon Dinamap Vital Signs Monitor 1846sxp automated blood pressure machine. Both systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured. Pulse was assessed using an electrocardiogram, an ECG 100C module attached to the Biopac MP150 system. Biopac systems EL503 EKG/ECHO, Stress Gel Vinyl 1-3/8" Electrodes were attached to the participant to collect heart rate data. AcqKnowledge version 4.1 was used to visually inspect segments of data. The AcqKnowledge raw electrocardiogram was then exported from AcgKnowledge into Kubios to edit and analyze the R-R data. Within the Kubios HRV software, artifact correction for R-R interval series was set to low. The 5-minute segments of R-R intervals during baseline and recall task were again visually inspected to ensure that all of the correct peaks were indicated in the software. Time-domain and frequency-domain variables of HRV were calculated using Kubios software. The time-domain variable of interest that was calculated by Kubios was RMSSD. In the frequency-domain, Fast Fourier Transformation technique was used to calculate the spectral powers in milliseconds-squared (ms^2) and normalized units (nu). The variables of LF (ms^2), LF (nu), HF (ms^2), HF (nu), and LF/HF were generated. Both milliseconds-squared and normalized units were computed, because milliseconds-squared is a raw measurement of

power, where there can be much greater variability between individuals, and large standard deviations. Spectral components calculated in normalized units represents "the relative value of each power component in proportion to the total power minus the very low frequency component" (Task Force, 1996, p. 358.). A measurement in normalized units emphasizes how the sympathetic and parasympathetic nervous system changes relative to one another, and displays the controlled and balanced behaviors of these two branches of the autonomic nervous system. Spectral components measured in normalized units have a much smaller standard deviation than spectral components measured in milliseconds-squared. Therefore, both milliseconds-squared and normalized units provide slightly different, but significant information regarding HRV. Default Kubios HRV Software defined LF (ms^2) as having a frequency range of 0.04 - 0.15Hz and HF (ms^2) as having a frequency range of 0.15 - 0.4Hz, which is in agreement with the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (1996).

Procedure

A correlational design was conducted to analyze the potential associations between the variables of interest. Participants were recruited through the online undergraduate participation system (SONA). The SONA advertisement was only made visible to females who were over the age of 18, and who identified as a European American or Arab American. During the recruitment process potential participants were informed that they will be excluded from the study if they have consumed alcohol or caffeine within the last twelve hours, or tobacco in the last three hours prior to the study. Participants who attended the study and stated they did consume alcohol, caffeine, or

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tobacco within the indicated time frames were given partial credit on SONA towards their behavioral science course, and were excluded from further data collection. The participants were asked to wear a tank top or similar top under their clothing, so that the researcher could place electrodes on the participants' chest to collect an electrocardiogram, and place a blood pressure cuff around the participants' upper right arm. All of the female participants were paired with female researchers, and two female researchers were present at all times.

Each participant arrived individually for the study. Upon arrival to the laboratory, participants were provided with an informed consent form (Appendix H) and asked if they had any questions regarding the study. Once informed consent was obtained, participants completed a demographics questionnaire. Participants were excluded from any further data collection if they failed to meet inclusion and exclusion criteria. If the participant was excluded at this point in the study, they received SONA course credit for a half hour, were debriefed, and their data was not included in data analysis. Participant's height and weight were also measured. Height was measured to the half-inch, and weight was measured to the nearest whole pound and recorded on the demographics form.

Upon completion of the demographics screening process, participants who continued with the study were prepared for physiological data collection. Two passive electrodes were attached to collect the electrocardiogram (one on the right upper chest and one on the left lower chest). See the attached figure (Appendix I) for the approximate locations. The ground lead was placed on the lower back due to equipment requirements for the needs of the larger study. A blood pressure cuff was placed on the upper right arm. In order to place the electrodes, the participant was taken into a private room in the

laboratory and notified that access to the upper body is required to attach the physiological equipment. They were asked to remove their outer shirt, leaving on their tank top (or similar garment). They were given the option to wear a medical gown if they chose. The researcher left the room asking the participants to come out when they are ready. The researcher applied vinyl gloves and cleaned the participants skin with alcohol wipes to provide a clean placement surface for the electrodes. The ECG electrodes and blood pressure cuff were then attached. Participants were notified of the importance of remaining as still as possible during data collection. Participants were shown the computer monitor and were told to shrug their shoulders in order to demonstrate just how sensitive the equipment is and the importance of sitting as still as possible. The monitor was then turned out of sight from the participant for the remainder of the study, so they could not witness their cardiovascular activity on the screen. If participants did move, the research assistant reminded the participants not to move, and continued with data collection. Once physiological instrumentation was completed, the participants were asked to sit quietly without moving for ten minutes. The last five minutes of this electrocardiogram recording was used to provide a baseline of cardiovascular functioning (four blood pressure readings, three minutes apart). At the conclusion of baseline, participants were asked to complete the Positive and Negative Affect Schedule (PANAS) 1 Questionnaire. Participants then engaged in the stress recall task.

An interpersonal stress recall task was utilized as the emotionally arousing stressor. Participants were instructed to "recall a time in your past when you were experiencing a great deal of stress or were having significant difficulty dealing with the demands placed on you by the action of others. Choose a very stressful time in your life,

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but one that you are willing to talk to the researcher about. It is important that you try to recall the event and your feelings about it in as much detail as possible, as if it were really happening right now." This task was selected since this is a typical acute stressor that has been shown to reliably activate the cardiovascular system (Glynn, Christenfeld, & Gerin, 2002). It is intended to increase physiological arousal in response to discussing a personally stress provoking event. In addition, the construct of ambivalence over emotional expression requires a person-to-person encounter. The participants were given two minutes to prepare what stressful event they were going to discuss. The participants were then asked, "Now tell me about the event and how you felt during this time. Don't just talk about it, but describe it to me as if it were really happening right now." The stress recall task itself ran for five minutes. The three primary researchers were graduate students in the health psychology program at the University of Michigan-Dearborn, and all received training on eliciting a stress response from the participants based on Lazarus's model of stress (focusing on danger, threat, loss, helplessness, a lack of coping, and lack of control), and were assessed on their skills and signed off by the primary investigator of the study determining their competence. During the stress recall task, the researcher used clinical therapeutic skills that elicited emotional content from the participant, by reflecting emotion words, and probing for additional information regarding the individual's thoughts and feelings about the experience. The researcher used several prompts, for example; "Did you feel a sense of threat during this time?" "Did you feel that you have much control over what is happening?" "Describe your feelings about the situation." "Describe what was happening in the exact moment."

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"Describe what was going on physically in your body in that exact moment." Three blood pressure readings, two minutes apart were collected during the recall task.

At the conclusion of the task the participants were asked to sit quietly for the next ten minutes (five blood pressure readings, two minutes apart). This is the cardiovascular recovery period used for the needs of the larger study. At the conclusion of the ten minute recovery period, the participants were told that physiological data collection was complete and were asked to complete the Stress Appraisal Measure and the PANAS 2. Instrumentation was then disconnected, the electrodes were removed, and the participant was escorted back into the private room to get dressed.

Participants then completed the CERQ, EES, and the AEQ. All of the instruments were checked for completion, and if a participant did not fill out an item, there were asked if they skipped the item on purpose or if they are willing to provide an answer to the item. At the conclusion of the inventories, the participant were debriefed about the study and thanked for their participation. Participants were compensated for their time by receiving course credit towards their introductory psychology course. Participants were also provided with a debriefing form (Appendix J) that included information regarding phone numbers of counseling and support services available at the University of Michigan – Dearborn, if the participants felt they needed to receive additional services following their participation in the research study. The study was also completed during the university counseling center's business hours, to be able to escort the participant to the counseling center if need be, to ensure the participant's safety.

Statistical Analysis Related to the Hypothesis

All analyses were performed using SPSS Statistics, Version 20.

Hypothesis 1: To test this hypothesis twelve correlations were conducted examining the association between CERQ subscales of self-blame, rumination, catastrophizing, and an aggregate maladaptive cognitive emotion regulation scale (calculated by obtaining a sum of the three previously names subscales) and cognitive appraisals of threat, stressfulness, and challenge, as measured by the SAM.

Hypothesis 2: To test this hypothesis nine hierarchical linear regressions were conducted for each subscale, equaling 36 hierarchical linear regressions, with SBP, DBP, pulse, RMSSD, LF (ms^2), LF (nu), HF (ms^2), HF (nu), and LF/HF from the stress recall task entered into the regression as dependent variables. Baseline calculations from all of the variables were entered into the hierarchical linear regression under block 1 of the independent variable, and the CERQ subscales of interest were entered under block 2 of the independent variable.

Hypothesis 3: To test this hypothesis three correlations were conducted examining the association between emotional expressivity as measured by the EES and cognitive appraisals of threat, stressfulness, and challenge as measured by the SAM.

Hypothesis 4: To test this hypothesis nine hierarchical linear regressions were conducted with SBP, DBP, pulse, RMSSD, LF (ms^2), LF (nu), HF (ms^2), HF (nu), and LF/HF from the stress recall task entered into the regression as dependent variables. Baseline calculations from all of the variables were entered into the hierarchical linear regression under block 1 of the independent variable, and the EES total score was entered under block 2 of the independent variable.

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Hypothesis 5: To test this hypothesis three correlations were conducted examining the association between ambivalence over emotional expression as measured by the AEQ and cognitive appraisals of threat, stressfulness, and challenge as measured by the SAM.

Hypothesis 6: To test this hypothesis nine hierarchical linear regressions were conducted with SBP, DBP, pulse, RMSSD, LF (ms^2), LF (nu), HF (ms^2), HF (nu), and LF/HF from the stress recall task entered into the regression as dependent variables. Baseline calculations from all of the variables were entered into the hierarchical linear regression under block 1 of the independent variable, and the AEQ total score was entered under block 2 of the independent variable.

Hypothesis 7: Initially, two mediating analysis were planned on being completed between emotional expressivity and threat appraisal with ambivalence over emotional expression as the mediating variable, and between ambivalence over emotional expression and threat appraisal with emotional expression as the mediating variable. However, since no associations were found for emotional expressivity, this analysis was not conducted.

Chapter III

Results

Descriptives

Descriptive statistics for Age, BMI, SAM-Threat, SAM-Challenge, SAM-Stressfulness, CERQ-Self-Blame, CERQ-Rumination, CERQ-Catastrophizing, CERQ-Aggregate Maladaptive Cognitive Emotion Regulation Strategies, Emotional Expressivity, Ambivalence over Emotional Expression, Baseline Systolic Blood Pressure, Baseline Diastolic Blood Pressure, Baseline Pulse, and Baseline HRV variables are displayed in Table 1 below. Skewness and kurtosis were assessed in the sample. The following variables displayed excessive skewness and were transformed using a natural logarithm; RMSSD, LF (ms^2), HF (ms^2), LF/HF ratio. All regressions were calculated using both transformed and non-transformed variables. Differences in the statistical significance for transformed verse non-transformed variables were only found for the hierarchical linear regressions of CERQ-Self-Blame and HF (ms^2), the aggregate cognitive emotion regulation strategies and LF/HF, and the AEQ and LF/HF. All other results are reported with non-transformed variables for ease of interpretation.

Manipulation Check

Before evaluating the primary hypothesis, a manipulation check was conducted to ensure that the stress recall task elicited both the anticipated psychological and physiological responses. Baseline and recall task data for blood pressure, time and

frequency domain spectral components of HRV, and PANAS positive and negative

affect, including results from paired samples t-test's and Cohen's d of effect sizes are

shown below in Table 2. As can be seen, as anticipated, a significant change occurred

across all variables except RMSSD and HF (ms^2) spectral components. Thus, the task

was effective in eliciting the expected stress recall response.

In addition, because this sample was composed exclusively of European

Americans and Arab Americans, independent t-tests were conducted to compare baseline

means of the two groups, which is shown below in Table 3. There were no significant

differences found between European Americans and Arab Americans on age, BMI,

systolic blood pressure, and diastolic blood pressure. There were statistically significant

differences between European Americans and Arab Americans with regards to pulse rate,

with Arab Americans displaying a lower baseline pulse rate than European Americans, as

well as baseline HRV measures of LF (ms^2) (t = 2.16, p < 0.05) and LF/HF (t = -2.21, p <

0.05), with Arab Americans demonstrating greater LF power (ms^2) and lesser LF/HF

ratio. It should be noted that the hierarchical linear regressions controlled for differences

in baseline HRV measures.

Hypothesis 1 Results: *A positive association will be found between the cognitive emotion regulation strategies of self-blame, rumination, catastrophizing, and appraisal of threat and stressfulness to an acute stress recall task, and a positive association will be found between the aggregate cognitive emotion regulation strategies (sum of self-blame, rumination, and catastrophizing) and appraisal of threat and stressfulness to an acute stress recall task. An association is not expected with the appraisal of challenge to an acute stress recall task.*

The correlations between cognitive emotion regulation strategies and stress

appraisal measures are shown below in Table 4. As expected, the variables were

associated in the expected direction, in a statistically significant or marginally significant manner, such that greater self-blame, rumination, catastrophizing, and the aggregate scores were associated with greater threat and stressfulness appraisal. As expected, there was not a significant association between the cognitive emotion regulation strategies and challenge appraisal.

There were marginally significant associations between CERQ-Self-Blame and threat and stressfulness appraisals. There was a marginally significant association between CERQ-Rumination and threat appraisal, and a statistically significant association between CERQ-Rumination and stressfulness appraisal. There was a statistically significant association between CERQ-Catastrophizing and threat appraisal, and a marginally significant association between CERQ-Catastrophizing and stressfulness appraisal. There were statistically significant associations between the aggregate maladaptive cognitive emotion regulation strategies and both threat and stressfulness appraisals.

Hypothesis 2 Results: *A positive association will be found between the individual cognitive emotion regulation strategies (self-blame, rumination, and catastrophizing), as well as the aggregate cognitive emotion regulation strategies (sum of self-blame, rumination, and catastrophizing) and SBP, DBP, pulse, LF cardiovascular reactivity, and LF/HF reactivity, and a negative association with RMSSD and HF cardiovascular reactivity.*

Hierarchical linear regressions between the cognitive emotion regulation strategies, blood pressure, and cardiovascular reactivity's are shown below (CERQ-Self-Blame in Table 5, CERQ-Rumination in Table 6, CERQ-Catastrophizing in Table 7, and CERQ-Aggregate in Table 8). No associations were found between the cognitive emotion regulation strategies and blood pressure and pulse reactivity. There were several

statistically significant and marginally significant associations found between the cognitive emotion regulation strategies and HRV reactivity in the expected direction.

CERQ-Self-Blame displayed marginally significant associations with LF (nu) reactivity and HF (nu) reactivity. CERQ-Rumination displayed statistically significant associations with LF (nu) reactivity, HF (nu) reactivity, and LF/HF reactivity. CERQ-Catastrophizing did not display any significant associations with HRV reactivity. The aggregate cognitive emotion regulation strategies displayed marginally significant associations with LF (nu) reactivity, HF (nu) reactivity, and the natural logarithm of LF/HF reactivity.

Hypothesis 3 Results: *A negative association will be found between emotional expression and appraisal of threat and stressfulness to an acute stress recall task. An association is not expected with the appraisal of challenge to an acute stress recall task.*

The correlations between emotional expressivity and stress appraisal measures are shown below in Table 9. No statistically significant associations were found between the EES and stress appraisal measures.

Hypothesis 4 Results: *A negative association will be found between emotional expression and SBP, DBP, pulse, LF cardiovascular reactivity, and LF/HF reactivity, and a positive association with RMSSD and HF cardiovascular reactivity.*

Hierarchical linear regressions between the EES and cardiovascular reactivity's are shown below in Table 10. No statistically significant associations were found between emotional expressivity as measured by the EES, blood pressure and pulse reactivity, and HRV reactivity.

Hypothesis 5 Results: *A positive association will be found between ambivalence over emotional expression and appraisal of threat and stressfulness to an acute stress recall task. An association is not expected with the appraisal of challenge to an acute stress recall task.*

The correlations between ambivalence over emotional expression and the stress appraisal measures are shown below in Table 11. As expected, statistically significant associations were found between ambivalence over emotional expression and both threat and stressfulness appraisals, such that greater ambivalence over emotional expression predicts greater threat and stressfulness appraisal. As expected, there was not a significant association between ambivalence over emotional expression and challenge appraisal.

Hypothesis 6 Results: *A positive association will be found between ambivalence over emotional expression and SBP, DBP, pulse, LF cardiovascular reactivity, and LF/HF reactivity, and a negative association with RMSSD and HF cardiovascular reactivity.*

Hierarchical linear regressions between ambivalence over emotional expression, blood pressure and pulse reactivity, and HRV reactivity's are shown below in Table 12. No associations were found between ambivalence over emotional expression and blood pressure and pulse reactivity. There were three marginally significant associations found between ambivalence over emotional expression and LF (nu) reactivity, HF (nu) reactivity, and the natural logarithm of the LF/HF reactivity in the expected direction.

AEQ displayed a marginally significant association with LF (nu) reactivity, such that greater ambivalence over emotional expression predicts greater LF (nu) reactivity. AEQ displayed a marginally significant association with HF (nu) reactivity, such that greater ambivalence over emotional expression predicts greater HF (nu) withdrawal. AEQ displayed a marginally significant association with the natural logarithm of LF/HF reactivity, such that the greater the ambivalence over emotional expression the greater the natural logarithm of the LF/HF reactivity.

Hypothesis 7 Results: *It is unclear how the variables of emotional expression, ambivalence over emotional expression, and threat appraisal are related to each other. In order to test competing models we will run two mediating analysis. The first, with ambivalence over emotional expression as the mediating variable between emotional expression and threat appraisal. The second, with emotional expression as the mediating variable between ambivalence over emotional expression and threat appraisal.*

Given that no associations were found between emotional expressivity (as measured by the EES), cognitive appraisals, and CVR; neither of the mediational analysis, analyzing emotional expressivity and ambivalence over emotional expression as potential mediating variables were able to be conducted.

Chapter IV

Discussion

The purpose of this study was to examine how maladaptive cognitive emotion regulation strategies; self-blame, rumination, catastrophizing, and the aggregate of the maladaptive coping strategies (sum of self-blame, rumination, and catastrophizing), emotional expressivity, and ambivalence over emotional expression are associated with cognitive appraisals of threat and stressfulness, blood pressure reactivity, and HRV reactivity in response to an acute interpersonal stress recall task.

An initial manipulation check was conducted that showed that the stress recall task used in this study did in fact elicit the anticipated psychological and physiological responses. The results of physiological data analyzed by paired-sample t-tests indicated a significant change from baseline measurements to recall task measurements of blood pressure, pulse, LF spectral components, HF spectral components, and the ratio between low frequency and high frequency spectral components. A manipulation check was also conducted with a psychological measurement (PANAS) analyzing an anticipated change in positive and negative affect from baseline measurements to recall task measurements, in such that positive affect decreased and negative affect increased following the recall task. These expected changes imply that indeed the recall task did elicit a stress response as anticipated.

Given the vast amount of results presented; particularly that some marginal and significant associations were found between the psychological variables and normalized

spectral components and ratios of HRV (the relative contribution of sympathetic and parasympathetic activation) and unexpectedly, no significant associations were found between any of the emotion concepts and blood pressure and pulse reactivity; this will be discussed first. After the discussion of this somewhat confusing finding, regarding the lack of associations with blood pressure and pulse reactivity, this will be followed by a discussion of each individual hypothesis, and additional specific factors potentially contributing to the results and implications of the results.

Lack of Significant Associations with Blood Pressure and Pulse Reactivity

The finding of sympathetic arousal and parasympathetic suppression in the absence of blood pressure changes is unexpected. In fact, change scores of normalized spectral components are not correlated to change scores of blood pressure. This finding was unexpected and is somewhat confusing in light of the fact that the scores for the CERQ subscales and AEQ were associated with threat and stressfulness appraisals. Thus, a post-hoc analysis was conducted to assess the potential association between threat and stress appraisals and blood pressure and pulse reactivity. Unexpectedly, neither threat nor stressfulness appraisals were associated with blood pressure or pulse reactivity (all $p's >$ .05). This finding may be best explained by the profile of appraisal scores. A post-hoc analysis was completed indicating that the appraisal of challenge was greater than the appraisal of threat ($p < .05$). This indicates that the female participants fundamentally perceived the stress recall task as challenging. As mentioned above, a challenging appraisal is generally associated with greater activation of the sympatho-adreno-medullary system and lesser activation of the hypothalamic-pituitary-adrenocortical

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system. This resulting lack of vasoconstriction generally leads to lesser blood pressure reactivity in the presence of sympathetic activation.

Gender and gender-matched dyads. Female researchers and same-sex, gender-matched researcher/participant dyads could explain why the stress recall task was perceived as challenging rather that threatening or stressful. The gender-matched dyads, which only consisted of female researchers and female participants, may have influenced this finding. As stated in the introduction, prior research has found that women are generally more expressive than men, and emotional expression to a same-sex stranger is experienced as more positive. This same-sex interaction is associated with lessened CVR, more typical of challenging physiological response, which has been implicated as being protective and resilient to illnesses (Blascovich & Tomaka, 1996; Mendes, Reis, Seery, & Blascovich, 2003). Same-sex partners have been shown to be able to relate more, be more familiar with each other, and be more comfortable sharing their emotions with each other because of this level of familiarity, when compared to opposite-sex partners (Rime et al., 1991). The perceived similarity between the dyads facilitates acceptance and willingness to express. In a previous study, emotional expression to opposite-sex dyads was found to be threatening and elicit a cardiovascular response typical for threat appraisal (Mendes, Reis, Seery, & Blascovich, 2003). Perhaps, the female participants in this study, felt more comfortable, supported, and accepted by the female researchers, and thus displayed lesser blood pressure and pulse reactivity.

Research also suggests that females provide more emotional support than males, and social support has been linked to dampened CVR and lower mortality rates from CVD (Thorsteinsson & James, 1999). Another study indicated that social support has

been associated with lower systolic blood pressure in women (Linden, Chambers, Maurice, & Lenz, 1993). Both males and females who receive social support from a female have lesser CVR when compared to those who did not have support from another female (Glynn, Christenfeld, & Gerin, 1999). Based on the traditional gender role, social support is typically considered feminine, since females tend to be more expressive and emotion focused (Burleson & Gilstrap, 2002; Kunkel & Burleson, 1999). A possibility is that since all female researchers were used, and the female researchers were trained to elicit emotional expression from the participant by the use of clinical micro-skills, this may have enhanced the participant's perception of support, attenuating participant's blood pressure and pulse reactivity. Thus, given the interpersonal nature of the task, the gender of the participants, and gender-matching dyads, lesser blood pressure reactivity may have been found due to the appraisal of this task as challenging. Lesser reactivity could explain the lack of associations between appraisals and reactivity. That is, given the gender of the participants and the use of gender matching dyads, participants may have experienced a greater sense of emotional support (as suggested by the challenge appraisals) leading to overall lesser reactivity (though still statistically significant). Additionally, it may be that while the CERQ subscales, aggregate scale, and ambivalence over emotion expression scale measure typical or trait patterns of emotional strategies, the supportive nature of the laboratory task may have led to these women not engaging in typical maladaptive patterns. That is, the supportive nature of the task may have provided an environment eliciting lesser effects of self-blame, rumination, catastrophizing, and ambivalent cognitions.

Mixed literature. It should also be noted that these unexpected finding may reflect the fact that there is a mixed literature on the associations between psychological stress and CVR. Psychological stress has not always been found to predict cardiovascular responses. A review of the effects of stressors and acute stressor reactivity was conducted by Gump and Matthews (1999); approximately half of the studies demonstrated increased physiological reactivity, while the other half demonstrated reduced acute stress reactivity. For example, the review indicated that three out of four studies of occupational stressors found reduced reactivity, and in studies examining family stressors there was a mixture of positive, negative, and no association with CVR. Having a substantial number of studies that demonstrates a reduced reactivity, may suggest that factors such as habituation, participant-task dynamics, or acute-chronic stress experiences may influence the association between psychological stress and CVR. There has also been a mixed literature on the association between cognitive appraisal and CVR. For example, Kline et al. (1999) failed to find a significant association between threat appraisal and hemodynamic response during a mental arithmetic task.

Anticipation/preparation period. In addition, it has been suggested that physiological responses during the preparation and anticipatory period may be greater than during the stress task itself, indicating that anticipation may be more threatening that the stressor (Birnbaum, 1964 as cited in Feldman, Cohen, Hamrick, & Lepore, 2004). The stress and coping model predicts that threat appraisal is anticipatory, potentially expecting a cardiovascular response prior to presence of the stressor. This was supported by Feldman, Cohne, Hamrick, & Lepore (2004) who examined appraisal and cardiovascular responses to a public speaking task. Their results indicated that the

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anticipatory preparation for the speaking task lead to greater threat appraisal and CVR when compared to participant preparation to just read aloud (low anticipatory stress). Also, during the actual speaking task, there was not a significant increase in cardiovascular response above and beyond what was elicited during preparation. It may be that differences related to anticipatory verse recall task appraisals, in combination with the timing of the SAM administration, may have accounted for the generally lower reliabilities for the SAM in the current study. Participants may have based their appraisals on the preparatory period verse the recall task period. This in turn may be related to the lack of association between appraisals and CVR.

Potential non-linear associations. Additionally, given that blood pressure and pulse values were averages from three distinct readings across the stress recall task, while spectral components represent an average across the entire stress recall task time, it may be that the timing associated with these variables resulted in lower associations. That is, habituation to the stress recall task could result in non-linear changes to nervous system activation/suppression that does not match the three discrete blood pressure and recordings. Additionally, the timing of sympathetic arousal may be randomly distributed to the reflections and probing of the researcher during the recall task and may not line up with the discrete blood pressure readings. Such non-linear processes have been discussed as a possible explanation for mixed results in the stress/CVR literature, and support the idea that nonlinear models may better represent the influence of stress on CVR (Chatkoff, Maier, & Klein, 2010; Dienstbier, 1989). The physiological toughness model suggests a nonlinear association of stress, in such that, those with moderate levels of stress show a physiological toughening when compared to those with low and severe stress that show

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increased reactivity (Dienstbier, 1989). In Chatkoff, Maier, & Klein's study assessing chronic stress and CVR and recovery, there were no linear associations between stress and reactivity, yet associations between stress and diastolic blood pressure were found when using quadratic modeling, such that those with moderate levels of stress displayed less CVR than those with low and high levels of stress. More sophisticated data collection methods (beat to beat blood pressure collection) combined with more sophisticated HRV data analysis methods such as joint time-frequency domain analysis might shed greater light on this issue.

Participant task engagement. Similarly, in terms of the limited findings reported above and related to the recall task itself, participant task engagement may be an important factor. Maier, Waldstein, & Synowski (2003) found that task engagement is crucial. A greater level of task engagement was correlated with increased diastolic blood pressure reactivity during a mental arithmetic task. They also found that challenge appraisals were related to task engagement. A post-hoc analysis of subjective task engagement was examined in the current study and found an average task engagement level between low and medium ($M = 1.79$) for participants. This lower subjective rating of task engagement suggests that participants may not have chosen to fully engage in the stress recall task. Additionally, bivariate correlations demonstrated that the average task engagement was statistically positively associated with systolic and diastolic change scores and marginally associated with pulse ($r = .46, p < .01; r = .39, p < .01; r = .19, p < .10$ respectively).

The participants may also have discussed a topic that was not emotionally stressful for them to a high extent, given that they may have been uncomfortable to

discuss a particularly stressful time in their life with the researcher and may have chosen a safer topic for discussion. For example, in the current study, participants discussed topics that ranged from an exam they had to take in class, to discussing a major family illness, or dealing with an alcoholic parent.

Overall, gender, gender-matching dyads, mixed literature, anticipatory/preparatory periods, potential non-linear associations, and participant task engagement may explain the lack of significant associations with blood pressure and pulse reactivity across all of the emotion concepts of interest, while still having some significant and marginally significant results regarding HRV reactivity. Next, each individual hypothesis will be further discussed.

Hypothesis 1 Discussion

It was anticipated that maladaptive cognitive emotion regulation strategies (measured by the CERQ) would be positively associated with cognitive appraisals of threat and stressfulness (measured by the SAM.) As anticipated, marginally significant positive associations were found for CERQ-Self-Blame and both threat and stressfulness appraisals. In addition, a statistically significant positive association was found between CERQ-Rumination and stressfulness, while the association with threat was marginally significant. There was a statistically significant positive association between CERQ-Catastrophizing and threat appraisal, and a marginally significant positive association between CERQ-Catastrophizing and stressfulness appraisal. Also as anticipated, there were statistically significant associations between the aggregate maladaptive cognitive emotion regulation strategies and both threat and stressfulness appraisals in the expected direction. Thus, the hypothesis was partially supported, since not all of the variables

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displayed statistically significant associations, however, all the variables were either significant or marginally significant in the expected direction. Also, as anticipated with all of the maladaptive cognitive emotion regulation strategies, there were no associations with the appraisal of challenge (which is theoretically a beneficial, motivating, and adaptive form of appraisal.)

These results indicate that the greater the use of self-blame, rumination, and catastrophizing as a cognitive method to regulate individual's emotional experiences, the greater the overall cognitive appraisals of threat and stressfulness within the context of an interpersonal stressor. These results are important, because this suggests clinical cognitive targets for stress reduction. Cognitive emotion regulation strategies should be given consideration when working with clients who have difficulties with stress management. Further, the results indicating that the aggregate maladaptive cognitive emotion regulation strategies; (sum of self-blame, rumination, and catastrophizing), has statistically significant positive associations with threat ($r = .323$, $p < .01$) and stressfulness appraisals ($r = .278$, $p <. 05$) is a novel finding. The other individual cognitive emotion regulation strategies only displayed a mixture of both significant and marginally significant associations with threat and stressfulness appraisals. This suggests that individuals may adopt a more global negative emotional regulation style that could inform research and clinical work. Additionally, the consistency of the above findings in terms of both the internal reliability and consistency between aggregate and subscale associations with cognitive appraisal provides further support for the use of an aggregate measure, along with assessing individual strategies.

Hypothesis 2 Discussion

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Blood pressure and pulse reactivity. While it was anticipated that cognitive emotion regulation strategies as measured by the CERQ would be associated with blood pressure and pulse reactivity, this was universally not supported. No significant associations between any of the CERQ subscales or aggregate scale and any of the blood pressure or pulse reactivity measures were found as discussed above.

Heart rate variability reactivity. It was also anticipated that the cognitive emotion regulation strategies as measured by the CERQ would be associated with HRV reactivity; however, this was only partially supported.

Self-blame. The CERQ-Self-Blame subscale displayed a marginally significant positive association with LF (nu) reactivity, and a marginally significant negative association with HF (nu) reactivity as predicted. As expected individuals who utilize greater emotion regulation strategies involving cognitive self-blame may display greater relative sympathetic reactivity and greater relative parasympathetic withdrawal potentially indicating a physiological stress response.

Rumination. The CERQ-Rumination subscale displayed statistically significant positive associations with LF (nu) reactivity and LF/HF reactivity, and a statistically significant negative association with HF (nu) reactivity as expected. Individuals who utilize greater emotion regulation involving rumination show greater relative sympathetic reactivity and greater relative parasympathetic withdrawal, along with a higher LF/HF reactivity. Thus, these individuals display a significant stress response to the interpersonal task.

Catastrophizing. There were no significant associations with the CERQ-Catastrophizing subscale and HRV reactivity. The lack of associations between

catastrophizing and CVR (both blood pressure and HRV reactivity) during a recall task may be related to the findings reported in the literature suggesting that catastrophizing has previously been related to increased depressive and anxiety symptoms (Garnefski, Kraaij, & Spinhoven, 2001; Garnefski, Teerds, Kraaij, Legerstee, & Kommer, 2004), and not specifically physiological distress. In a study conducted on how cognitive emotion regulation strategies predict depression, anxiety, stress, and anger; depression and anxiety were predicted by all three of the maladaptive strategies, yet stress was only predicted by self-blame and rumination (Martin & Dahlen, 2005). Therefore, catastrophizing may not predict stress as well as the other two maladaptive cognitive emotional regulation strategies of interest, and may not be as detrimental of a cognitive strategy to utilize in relation to CVR. In addition, to understand this perplexing finding, it may be helpful to draw from cross-disciplinary research for an explanation of why the hypotheses were only marginally significant. For example, within the context of chronic pain, Sullivan (2012) suggests through the communal coping model that pain catastrophizing is an attempt by the pain patient to elicit emotional or instrumental support from others. In the present studying, utilizing an interpersonal stress recall task, those who endorse catastrophizing may have found the supportive nature of the researcher during this task, to be meeting their needs for affiliation and emotional support. Thus, while the participant may still appraise the recall task as threatening, they also may have found the researchers use of emotional reflections to be meeting their general relational needs, resulting in less reactivity. Thus, it may be that catastrophizing as a cognitive emotion regulation strategy within the context of an emotionally supportive environment may

result in lesser physiological reactivity, while self-blame and rumination as cognitive emotion regulation strategies may be less buffered by empathetic interactions.

Aggregate. The aggregate of the maladaptive cognitive emotion regulation strategies (the sum of self-blame, rumination, and catastrophizing) displayed marginally significant positive associations with LF (nu) reactivity and the natural logarithm of LF/HF reactivity, and a marginally significant negative association with HF (nu) reactivity as anticipated. This indicates greater relative sympathetic reactivity and greater relative parasympathetic withdrawal in response to a stress recall task. Thus, these individuals display a marginal physiological stress response.

It should also be noted that the rumination subscale of the CERQ demonstrated the strongest association with HRV reactivity. This was the only CERQ strategy that displayed statistically significant associations to some of the HRV spectral components. This suggests that rumination may overall be more detrimental than the other two maladaptive coping strategies.

Hypothesis 3 Discussion

While it was anticipated that an association would be found between emotional expressivity and threat and stress appraisal, in such that, lesser emotional expressivity would be associated with greater appraisal of threat and stressfulness, this was not displayed in the current study. There were no statistically significant or marginally significant associations between emotional expressivity (as measured by the EES), and cognitive appraisal of threat or stressfulness.

Based on the lack of association between emotional expressivity and threat appraisal, a post-hoc test was conducted to analyze if there was a correlation between

emotional expressivity and the participants subjective rating of task engagement. Post-hoc analyses revealed that there were no associations between emotional expressivity (measured by the EES) and participant's level of task engagement when discussing a stressful event during the recall task. This indicates that the participant's typical level of emotional expressivity was not related to their level of engagement during the recall task. This may indicate a lack in terms of external validity, given that the laboratory results may not generalize to the participant's typical emotional expressivity outside of the laboratory.

Furthermore, literature suggests that those who express their emotions, compared with those who just express facts regarding the situation, show a lower autonomic arousal response. Those who express more facts than emotions display greater autonomic arousal, indicating that when participants attended to their emotions, habituation to the stressful situation occurred (Stanton, Kirk, Cameron, Danoff-Burg, 2000). While this is related to physiological reactivity and is important for hypothesis 4 discussed below, it also has implication for cognitive appraisals based on Lazarus's model. To explore the potentially applicability of this explanation, a post-hoc analysis was conducted completing a correlational analysis between the number of emotion words expressed by the participant (measured by the emotion counter) and their appraisal of threat, stressfulness, and challenge (measured by SAM). There were no significant associations between the number of emotion words expressed and threat and stressfulness appraisals ($p > .05$). There was a statistically significant positive association between the number of emotion words expressed and the appraisal of challenge ($r = 0.25$, $p < .05$). This suggests that greater emotional expressivity would not necessarily be associated with cognitive

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appraisal of threat and stressfulness. Thus, in this study, it may be that when the participants were told to discuss a stressful life event, while the researcher focused on the concepts of danger, loss, threat, helplessness, lack of coping, and lack of control, and used clinical skills to eliciting emotions, perhaps a lower cardiovascular response was seen from the participants associated with an appraisal of challenge. Furthermore, the use of therapeutic micro-skills to elicit emotions may have limited the utility of the EES as a trait measure. In fact, a post-hoc analysis indicated that the number of emotion words (measured by the emotion counter) was not associated with EES scores ($p > .05$). This also has implications for hypothesis 4.

Hypothesis 4 Discussion

While it was anticipated that an association would be found between emotional expressivity and CVR, this hypothesis was not supported. There were no statistically significant or marginally significant associations between emotional expressivity (as measured by the EES), and blood pressure and pulse reactivity variables, or HRV reactivity variables.

The lack of significant associations between the EES and blood pressure and pulse reactivity and HRV reactivity measures is most likely related to the results from hypothesis 3. Given that the hypothesized association between EES scores and CVR was based on the premise that EES would be associated with cognitive appraisals of threat and stressfulness, a lack of findings for hypothesis 3 partially explains the null findings for CVR. This may be further compounded by the lack of an association in the sample between threat and stressfulness and CVR as discussed above.

Another potential explanation for the lack of significant results relating emotional expressivity to physiological reactivity may be because emotional expressivity was measured as a one-factor model, and perhaps a two-factor model of emotional expression verse emotional suppression may have been a better fit when analyzing associations to CVR. The literature suggests that emotional expression and emotional suppression does not simply lie on a continuum of the amount of emotional disclosure. Emotional suppression is defined as "the conscious inhibition of one's own emotional expressive behavior while emotionally aroused" (Gross & Levenson, 1993, p.970). Emotional suppression has been related to higher negative affect, lower positive affect, poorer social adjustment, and a decrease in general well-being (Gross & John, 2003). Higher emotional suppression is thought to be related to both anxiety and mood disorders. It has been shown in previous studies that participants who suppressed their emotion indicated an increase in cardiac interbeat interval, increases in sympathetic arousal, increases in systolic and diastolic blood pressure, and decreases in somatic activity when compared to the non-suppression group (Gross & Levenson, 1993). Suppression was associated with increases in sympathetic activation of the cardiovascular system; however, it was not related to parasympathetic activation of the cardiovascular system. The hydraulic model has been used to explain the negative correlation found between behavioral expression and physiological responses. The hydraulic model proposes that when expression of emotion is inhibited, the expressive signs are discharged through other channels (Gross & Levenson, 1993). Analyzing emotional suppression referring specifically to inhibition of emotional expression may fit better with CVR and HRV research.

There has also been mixed literature regarding emotional expression. There have been many studies that indicate the benefits of emotional disclosure from a biological standpoint, such that blood pressure is reduced, muscle tension is lessened, and immune functioning is increased (Esterling et al., 1994, Pennebaker et al., 1988; Petrie, Booth, & Pennebaker, 1998). There has also been conflicting results displayed in a study where an increase in emotional expressivity predicted poorer adjustment across time. The authors suggested these results may have been obtained because those who are high on expression and emotional processing may have high neuroticism, rumination, and/or excessive support seeking (Stanton, Kirk, Cameron, & Danoff-Burg, 2000), and well as, intrusive thoughts (Major & Gramzow, 1999) and increased distress (Bonanno, Keltner, Holen, & Horowitz, 1995). These are concepts which are typically related with a poorer physiological response. Therefore, a mixture of results may explain the lack of significant associations for emotional expressivity in the current study.

Hypothesis 5 Discussion

It was anticipated that ambivalence over emotional expression (as measured by the AEQ) would be positively associated with cognitive appraisal measures of threat and stressfulness (as measured by the SAM). This hypothesis was fully supported. As anticipated, there were statistically significant positive associations between ambivalence over emotional expression and both threat and stressfulness appraisals, in such that greater ambivalence over emotional expression indicated greater appraisal of threat and stressfulness. Also, as anticipated, there was no association between ambivalence over emotion expression and appraisal of challenge, which is theoretically a beneficial, motivating, and adaptive form of appraisal. This is an important finding because the

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concept of ambivalence over emotional expression is growing in the literature. Several studies mention ambivalence as a factor of importance involved in emotional expression, suggesting that researching emotional expression alone does not encompass all of the significant aspects of expressivity (King & Emmons, 1990; King, 1998; Brockmeyer et al., 2013).

Hypothesis 6 Discussion

While it was anticipated that ambivalence over emotional expression as measured by the AEQ would be associated with CVR, this was almost universally not supported. No significant associations between the AEQ and any cardiovascular reactivity measures were found. Only several marginally significant associations were found with HRV spectral components.

Blood pressure and pulse reactivity. No significant or marginal associations were found for ambivalence over emotional expression and blood pressure or pulse reactivity. This lack of findings for blood pressure and pulse reactivity may be related to the lesser ambivalence (state response vs trait measure) during the recall task itself due to the supportive nature of the task, as well as, relatively greater appraisals of challenge and resulting cardiovascular implications as described above. It may also be that the degree of control over the material the participant discussed limited their stress response to the task. Perceived control has been shown to influence individual's physiological responses of challenge and threat states. In a meta-analysis review of acute psychological stressors and cortisol response, Dickerson & Kemeny (2004) found that even if an individual perceives an uncontrollable situation as controllable, a lesser physiological response occurs compared to individuals who perceive the situation as uncontrollable. It may be that if the

participant perceived the stress recall task as controllable, they may have shown lesser CVR than if the individual perceived the task as uncontrollable. The participants did indeed have control over what stressful event they discussed during the stress recall task. The influence of perceived control and the biological and subjective stress response was previously studied, and results indicated that individuals who perceived themselves as having control over a stressor and who had a more internal locus of control showed a reduced stress response as measured by cortisol (Bollini, Walker, Hamann, Kestler, 2004). This may be partially supported by the fact that while ambivalence was associated to threat and stressfulness, appraisals of threat and stressfulness were not associated with physiological reactivity.

Heart rate variability reactivity. Only three marginal associations were found between AEQ and HRV reactivity components. AEQ and LF (nu) reactivity was marginally significant, such that greater ambivalence over emotional expression predicts greater LF (nu) spectral power. Greater LF spectral power indicates greater relative sympathetic reactivity. The AEQ also displayed a marginally significant negative association with HF (nu) reactivity, such that greater ambivalence over emotional expression predicts greater relative parasympathetic withdrawal. There was a marginally significant association between AEQ and the natural logarithm of LF/HF reactivity, such that greater ambivalence over emotional expression predicts the greater the natural logarithm of LF/HF reactivity.

Ambivalence may be an imperative factor to consider when analyzing the construct of emotional expressivity. Even when there was a lack of CVR in regards to emotional expression as measured by the EES, there were some marginally significant

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results when the individual experienced ambivalence over their emotional expressivity. The combination of the lack of emotional expression along with ambivalence over emotional expression is what Pennebaker (1985) suggests is related to the development of health problems. The current study's results may indicate that emotional expressivity as a single factor may not be involved enough, and other areas related to cognitive dissonance are important to focus on in a clinical setting. Similar results were found in another study assessing ambivalence and secret keeping indicating that greater ambivalence over emotional expression as well as secret keeping (wanting to keep personal information private and being apprehensive about disclosing the information) was associated with greater distress and anxious arousal (Barr, Kahn, & Schneider, 2008). The reluctance and cognitive dissonance involved with these concepts may be a potential area of focus within cognitive behavioral therapy in a clinical setting.

Since ambivalence over emotional expression has previously been positively associated with depression, negative affect, psychological ill-being, obsessive/compulsive tendencies, paranoid ideation, and phobic anxiety (Brockmeyer et al., 2013; King, 1998) and negatively associated with life satisfaction and self-esteem (King & Emmons, 1990), and now marginally significant results showed a positive association with increased sympathetic nervous system activation and a decrease in parasympathetic nervous system activation, this can be an important area of focus related to both psychological and physical distress.

The lack of statistically significant associations may be partially related to the AEQ's failure to differentiate the relative contribution of the multiple sub-constructs. For example, those individuals in Domain 1 (expressive despite their lack of desire to

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express) may respond differently, psychologically and physiologically, relative to those in Domain 2 (inexpressive despite their desire to express). Additionally, the ruminative quality associated with Domain 3 (expression and regret), may result in yet a different response pattern, particularly in terms of physiological recovery from a stressor. Thus, the marginal findings with sympathetic and parasympathetic reactivity as measured by spectral components should be considered within the context of the multiple domains of ambivalence over emotional expression.

Overall, these results indicate that the maladaptive cognitive emotion regulation strategies and ambivalence over emotional expression generally predicts cognitive appraisals of threat and stressfulness, as well as, at least to some extent (marginally and significantly) predicts relative sympathetic and parasympathetic activity, as was expected.

Strengths and Limitations of the Current Study

Limitations. There were several limitations of the current study. The first limitation is that data were collected only from undergraduate students attending the University of Michigan-Dearborn. Another limitation of the study is that only European American and Arab American females were included in the study, due to the needs of the larger study that this study was involved in. Therefore, there was not a great diversity of ethnicities or gender, which limits the generalizability of these results to the general population. Gender and ethnic differences in appraisal and emotional strategies may be important considerations.

Secondly, the above findings for the cognitive emotion regulation strategies (self-blame, rumination, catastrophizing, and aggregate) and ambivalence over emotional

expression, and HRV reactivity suggest that these emotion concepts are associated with a notable physiological stress response as measured by HRV reactivity to an interpersonal stress recall task, as expected. However, only normalized units and ratios were significant or marginally significant for ambivalence over emotional expression and any of the maladaptive cognitive emotion regulation strategies. HRV reactivity as measured in milliseconds squared may not have been associated with any of the emotion concepts to a statistically significant level for several reasons. First of all, given the time limitation imposed by the requirements of a master's program, a limited sample size of 82 participants may have been too small to run analysis for some spectral components. A larger sample would have facilitated analysis of spectral components. The non-normalized spectral components have great variation between individuals. For example, LF (ms^2) at recall task ranged between 62 and 10,418, with a $SD = 1610.67$, and HF (ms^2) ranged between 21 and 14,487, with a $SD = 1922.42$. This very large variation in HRV spectral components could explain the lack of expected results based on the sample size of 82. The same spectral components calculated using normalized units, have a much smaller range, and smaller standard deviation, for example, LF (nu) ranged between 28.5 and 94.8, $SD = 14.59$, and HF (nu) ranging from 5.2 to 71.4, $SD = 14.57$. This may explain why the significant and marginally significant results obtained were with normalized units and ratios, rather than the raw spectral power reported by milliseconds squared. Finally, it should be noted that only one type of stress recall task, which was highly interpersonal in nature, was used.

Strengths. A particular strength of this study is that it is the first study to examine the potential associations between cognitive emotional regulation strategies, emotional

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expressivity, and ambivalence over emotional expression within the context of acute stress induced CVR, examining spectral components of HRV and HRV reactivity. Given some of the findings relating these emotion concepts to threat and stressfulness appraisals, and the limited associations to HRV, further studies are warranted to examine emotional processes and cardiovascular health risk.

Another strength is that there is a significant body of literature examining these emotion based variables within the constructs of depression, anxiety, and overall well-being, yet very few studies have conceptualized these emotion based variables within the context of psychological stress and the stress response. This study adds to this limited literature.

A methodological strength of the study is that the stress recall task was prompted by three researchers in a Masters level graduate psychology program. The same three researchers ran all of the participants, reducing the amount of variability in researcher presentation. The researchers had specific training in eliciting a stress response based on Lazarus theory of stress, prompting for danger, loss, threat, helplessness, lack of coping skills, and a lack of control.

The strict exclusion criterion utilized was necessary in order to prevent changes in the participant's sympathetic and parasympathetic nervous system based on substances, caffeine, stimulants, cardiovascular disease, or medication. Having strict exclusion criteria help ensure that the associations found between CVR and the emotion concepts are not confounded by other outside influences.

Future Research

Future research on these emotion related topics are necessary to determine if there are gender or cultural differences. It would be beneficial for future studies to conduct analyses on a non-college population to help display greater generalizability to other populations. Also, based on the lack of results in relation to emotional expressivity as measured as a one-factor model, by the EES, future research may want to focus on analyzing emotion expressivity as a two-factor model, analyzing emotional expression and emotional suppression. Previous literature displays stronger results in relation to emotional suppression rather than emotional expression when analyzing associations to physiological responses and poorer outcomes.

Additionally, the use of an interpersonal stress recall task delivered in a supportive environment may have influenced the results. Future research may want to use a stress recall task, as well as some other task, such as an evaluated speech task or verbal mental arithmetic task, in order to induce stress without directly eliciting emotional expression, and without interacting in a supportive interpersonal manner. In a previous study conducted by Chatkoff & Leonard (2009), it was found that Arab Americans displayed a greater appraisal of threat and stress during a mental arithmetic task compared to a stress recall task. Since the emotion concepts of cognitive emotion regulation (as measured by the CERQ), emotional expression (as measured by the EES), and ambivalence over emotion expression (as measured by the AEQ) are all considered from a "trait" perspective, it may be that the type of stress task, in a laboratory setting, with gender matched researcher/participant dyads limited the hypothesized associations. Perhaps, using a less interpersonal task may help elicit the participant's typical traits. This may be particularly salient with the cognitive emotion regulation strategy of

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catastrophizing, given the potential link to the communal coping model as described above. A review of mixed literature between psychological stress and CVR shows that different stressors elicit a different stress response, under different circumstances, for different individuals (Gump & Matthews, 1999). Moreover, in terms of ambivalence over emotional expression, each of the three domains within the construct should be examined separately to determine if differences in the associations to cognitive appraisal and HRV reactivity exist.

Finally, since in the current study, a subjective rating (as determined by the researcher and research assistant) of participant level of task engagement displayed $M =$ 1.79, which is between low and medium task engagement, a way to increase participants level of task engagement would be beneficial in future studies. For example, a post-hoc analysis was completed, and when participants were excluded from data analysis if they obtained a task engagement score of 1 (indicating low level of task engagement), an additional significant result was found. CERQ-Catastrophizing (which did not initially yield significant results), displayed a statistically significant association with HF (ms^2) spectral power in the expected direction. Thus, increasing participant task engagement and power may facilitate additional significant results in future studies.

Table 1: Descriptive Statistics of the Sample Data (Non-Transformed)

Variable	N	M	SD	SEM
Age	82	20.110	3.641	.402
BMI	82	23.876	5.463	.603
SAM-Threat	82	10.711	3.170	.350
SAM-Challenge	82	11.963	3.049	.337
SAM-Stressfulness	82	14.549	2.727	.301
CERQ-Self-Blame	82	10.256	3.644	.402
CERQ-Rumination	82	11.646	3.426	.378
CERQ-Catastrophizing	82	8.402	3.665	.405
CERQ-Aggregate	82	30.305	8.748	.966
Emotional Expressivity	82	59.671	13.882	1.533
Ambivalence over EE.	82	77.614	19.126	2.112
Baseline SBP	82	112.067	10.003	1.105
Baseline DBP	82	68.329	5.971	.659
Baseline Pulse	82	78.537	10.920	1.206
Baseline RMSSD	82	40.113	29.497	3.257
Baseline LF (ms^2)	82	968.240	934.372	103.184
Baseline LF (nu)	82	56.368	16.972	1.874
Baseline HF (ms^2)	82	1044.550	2033.320	224.543
Baseline HF (nu)	82	43.568	17.063	1.884
Baseline LF/HF	82	1.787	1.403	.155

Note. BMI = Body Mass Index, SAM = Stress Appraisal Measure, CERQ = Cognitive Emotion Regulation Questionnaire, Aggregate = Sum of self-blame, rumination, & catastrophizing subscales, EE = emotional expression, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, RMSSD = Root Mean Square of Successive Difference, LF = Low frequency spectral power, HF = High frequency spectral power, ms^2 = milliseconds squared, nu = normalized units, LF/HF = Ratio of Low frequency to High Frequency spectral powers.

Table 2: Manipulation Check of Psychological and Physiological Responses (Non-Transformed)

	Baseline			Recall Task			Cohen's d
	n	M	SD	n	M	SD	
SBP	82	112.067	10.003	82	129.670	14.057	1.44**
DBP	82	68.329	5.971	82	81.936	8.336	1.87**
Pulse	82	78.537	10.919	82	88.886	13.220	0.85**
RMSSD	82	40.113	29.497	82	39.400	27.693	-.02
LF (ms^2)	82	968.240	934.372	82	1781.72	1610.665	.62**
LF (nu)	82	56.368	16.972	82	67.871	14.592	.73**
HF (ms^2)	82	1044.550	2033.320	82	1114.760	1922.417	.04
HF (nu)	82	43.568	17.063	82	32.111	14.570	-.72**
LF/HF	82	1.787	1.403	82	3.139	3.115	.56**
PANAS-P	82	24.556	6.953	82	21.778	7.134	-.39**
PANAS-N	82	13.790	3.771	82	23.025	6.995	1.64**

Note: SBP = Systolic blood pressure, DBP = Diastolic blood pressure, RMSSD = Root Mean Square of Successive Difference, LF = Low frequency spectral power, HF = High frequency spectral power, ms^2 = milliseconds squared, nu = normalized units, LF/HF = Ratio of Low frequency to High Frequency spectral powers, PANAS-P = Positive subscale of the Positive and Negative Affect Schedule (20-item scoring), PANAS-N = Negative subscale of the Positive and Negative Affect Schedule (20-item scoring).
**= Paired samples t-test p < 0.01

Table 3: Independent T-Tests Comparing Means Between Ethnicity

Variable	European Americans		Arab Americans		Cohen's
	$n = 48$		$n = 33$		d
	M	SD	M	SD	
Age	20.48	4.486	19.58	1.871	-.26
BMI	24.075	5.992	23.814	4.584	-.05
Baseline SBP	113.573	10.997	109.985	8.211	-.37
Baseline DBP	68.760	5.812	67.606	6.286	-.19
Baseline Pulse	81.229	10.228	74.606	11.017	-.62**
Baseline LF (ms^2)	766.210	514.465	1276.480	1286.442	.52*
Baseline LF (nu)	58.944	17.149	52.482	16.461	-.38
Baseline HF (ms^2)	637.350	612.215	1658.91	3043.049	.47
Baseline HF (nu)	41.065	17.419	47.348	16.326	.37
Baseline LF/HF	2.048	1.606	1.414	0.973	-.48*

Note: BMI = Body Mass Index, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, LF = low frequency spectral power, HF = high frequency spectral power, LF/HF = Ratio of Low frequency to High Frequency spectral power, ms^2 = milliseconds squared, nu = normalized units,
* = independent samples t-test p < 0.05
**= Independent samples t-test p < 0.01

Table 4: Correlations between the CERQ and SAM

	CERQ-SB	CERQ-RUM	CERQ-CAT	CERQ-AGG	SAM-Threat	SAM-Challenge	SAM-Stress
CERQ-SB	--	.631**	.368**	.818**	.206 $^+$	-.086	.183 $^+$
CERQ-RUM		--	.498**	.863**	.209 $^+$	.122	.305**
CERQ-CAT			--	.767**	.371**	.104	.195 $^+$
CERQ-AGG				--	.323**	.056	.278*
SAM-Threat					--	-.154	.570**
SAM-Challenge						--	.112
SAM-Stress							--

Note: CERQ = Cognitive emotion regulation questionnaire, SB = CERQ Self-Blame subscale, RUM = CERQ Rumination subscale, CAT = CERQ Catastrophizing subscale, AGG = Aggregate cognitive emotion regulation strategies (sum of self-blame, rumination, and catastrophizing), SAM = Stress Appraisal Measure, Stress = Stressfulness subscale
*** = p < 0.01*
** = p < 0.05*
$^+$ = p < 0.10

Table 5: Hierarchical Linear Regressions of CERQ-Self-Blame and Cardiovascular Reactivity Controlling for Baseline Cardiovascular

Psychological Variable	Physiological Variable HR Step	R	R^2	ΔR^2	B	t	B
CERQ SB							
	Systolic BP						
	Step 1: BL SBP	.687	.472	--	--	--	--
	Step 2: CERQ SB	.687	.472	.000	-.043	-.135	-.011
	Diastolic BP						
	Step 1: BL DBP	.660	.436	--	--	--	--
	Step 2: CERQ SB	.664	.441	.005	.160	.816	.070
	Pulse						
	Step 1: BL Pulse	.668	.447	--	--	--	--
	Step 2: CERQ SB	.672	.451	.005	-.248	-.820	-.068
	RMSSD						
	Step 1: BL RMSSD	.868	.754	--	--	--	--
	Step 2: CERQ SB	.869	.755	.001	-.221	-.517	-.029
	LF (ms^2)						
	Step 1: BL LF (ms^2)	.742	.550	--	--	--	--
	Step 2: CERQ SB	.742	.550	.000	2.501	.074	.006
	LF (nu)						
	Step 1: BL LF (nu)	.480	.230	--	--	--	--
	Step 2: CERQ SB	.513	.263	.032[+]	.721	1.863	.180
	lnHF (ms^2)						

Step 1: BL lnHF (ms^2)	.791	.626	--	--	--	--
Step 2: CERQ SB	.793	.630	.003	-.018	-.832	-.057

HF (nu)

Step 1: BL HF (nu)	.473	.224	--	--	--	--
Step 2: CERQ SB	.505	.255	.031$^+$	-.709	-1.825	-.177

LF/HF

Step 1: BL LF/HF	.292	.085	--	--	--	--
Step 2: CERQ SB	.300	.090	.005	.058	.629	.068

Note: BL = Baseline, CERQ = Cognitive Emotion Regulation Questionnaire, SB = CERQ Self-Blame subscale, BP = Blood pressure, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, RMSSD = Root Mean Square of Successive Difference, LF = Low frequency spectral power, HF = High frequency spectral power, (ms^2) = milliseconds squared, (nu) = normalized units, LF/HF = Ratio of Low frequency to High Frequency spectral powers, lnHF = Natural logarithm of high frequency spectral power.
$^+ = p < 0.10$

Table 6: Hierarchical Linear Regressions of CERQ-Rumination and Cardiovascular Reactivity Controlling for Baseline Cardiovascular

Psychological Variable	Physiological Variable HR Step	R	R^2	ΔR^2	B	t	B
CERQ- Rum							
	Systolic BP						
	Step 1: BL SBP	.687	.472	--	--	--	--
	Step 2: CERQ Rum	.687	.473	.001	.130	.378	.032
	Diastolic BP						
	Step 1: BL DBP	.660	.436	--	--	--	--
	Step 2: CERQ Rum	.670	.449	.013	.284	1.381	.117
	Pulse						
	Step 1: BL Pulse	.668	.447	--	--	--	--
	Step 2: CERQ Rum	.669	.447	.000	.065	.201	.017
	RMSSD						
	Step 1: BL RMSSD	.868	.754	--	--	--	--
	Step 2: CERQ Rum	.869	.755	.001	-.286	-.631	-.035
	LF (ms^2)						
	Step 1: BL LF (ms^2)	.742	.550	--	--	--	--
	Step 2: CERQ Rum	.747	.558	.007	40.927	1.142	.087
	LF (nu)						
	Step 1: BL LF (nu)	.480	.221	--	--	--	--
	Step 2: CERQ Rum	.555	.290	.077**	1.183	2.968	.278
	HF (ms^2)						

Step 1: BL HF (ms^2)	.876	.767	--	--	--	--
Step 2: CERQ Rum	.878	.772	.005	-40.536	-1.338	-.072

HF (nu)

Step 1: BL HF (nu)	.473	.224	--	--	--	--
Step 2: CERQ Rum	.547	.300	.076**	-1.170	-2.922	-.275

LF/HF

Step 1: BL LF/HF	.292	.085	--	--	--	--
Step 2: CERQ Rum	.400	.160	.075**	.248	2.648	.273

Note: BL = Baseline, CERQ = Cognitive Emotion Regulation Questionnaire, Rum = CERQ Rumination subscale, BP = Blood pressure, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, RMSSD = Root Mean Square of Successive Difference, LF = Low frequency spectral power, HF = High frequency spectral power, (ms^2) = milliseconds squared, (nu) = normalized units, LF/HF = Ratio of Low frequency to High Frequency spectral powers
** = p < 0.01

Table 7: Hierarchical Linear Regressions of CERQ-Catastrophizing and Cardiovascular Reactivity Controlling for Baseline Cardiovascular

Psychological Variable	Physiological Variable HR Step	R	R^2	ΔR^2	B	t	B
CERQ- Cat							
	Systolic BP						
	Step 1: BL SBP	.687	.472	--	--	--	--
	Step 2: CERQ Cat	.690	.477	.005	.273	.869	.071
	Diastolic BP						
	Step 1: BL DBP	.660	.436	--	--	--	--
	Step 2: CERQ Cat	.663	.439	.003	.121	.627	.053
	Pulse						
	Step 1: BL Pulse	.668	.447	--	--	--	--
	Step 2: CERQ Cat	.668	.447	.000	-.018	-.061	-.005
	RMSSD						
	Step 1: BL RMSSD	.868	.754	--	--	--	--
	Step 2: CERQ Cat	.868	.754	.000	.097	.230	.013
	LF (ms^2)						
	Step 1: BL LF (ms^2)	.742	.550	--	--	--	--
	Step 2: CERQ Cat	.744	.554	.004	27.180	.822	.062
	LF (nu)						
	Step 1: BL LF (nu)	.480	.230	--	--	--	--
	Step 2: CERQ Cat	.481	.232	.001	-.139	-.354	-.035
	HF (ms^2)						

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Step 1: BL HF (ms²)	.876	.767	--	--	--	--
Step 2: CERQ Cat	.876	.767	.000	6.796	.238	.013

HF (nu)

Step 1: BL HF (nu)	.473	.224	--	--	--	--
Step 2: CERQ Cat	.475	.226	.002	.171	.435	.043

LF/HF

Step 1: BL LF/HF	.292	.085	--	--	--	--
Step 2: CERQ Cat	.293	.086	.001	.025	.272	.029

Note: BL = Baseline, CERQ = Cognitive Emotion Regulation Questionnaire, Cat = CERQ Catastrophizing subscale, BP = Blood pressure, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, RMSSD = Root Mean Square of Successive Difference, LF = Low frequency spectral power, HF = High frequency spectral power, (ms²) = milliseconds squared, (nu) = normalized units, LF/HF = Ratio of Low frequency to High Frequency spectral powers

Table 8: Hierarchical Linear Regressions of CERQ-Aggregate (Sum of Self-Blame, Rumination, & Catastrophizing) and Cardiovascular Reactivity Controlling for Baseline Cardiovascular

Psychological Variable	Physiological Variable HR Step	R	R^2	ΔR^2	B	T	B
CERQ- Agg							
	Systolic BP						
	Step 1: BL SBP	.687	.472	--	--	--	--
	Step 2: CERQ Agg	.688	.473	.001	.061	.458	.038
	Diastolic BP						
	Step 1: BL DBP	.660	.436	--	--	--	--
	Step 2: CERQ Agg	.667	.445	.009	.093	1.148	.098
	Pulse						
	Step 1: BL Pulse	.668	.447	--	--	--	--
	Step 2: CERQ Agg	.669	.447	.001	-.036	-.287	-.024
	RMSSD						
	Step 1: BL RMSSD	.868	.754	--	--	--	--
	Step 2: CERQ Agg	.868	.754	.000	-.064	-.363	-.020
	LF (ms^2)						
	Step 1: BL LF (ms^2)	.742	.550	--	--	--	--
	Step 2: CERQ Agg	.744	.554	.004	11.514	.822	.063
	LF (nu)						
	Step 1: BL LF (nu)	.480	.230	--	--	--	--
	Step 2: CERQ Agg	.509	.259	.029[+]	.282	1.747	.169
	HF (ms^2)						

Step 1: BL HF (ms^2)	.876	.767	--	--	--	--
Step 2: CERQ Agg	.878	.771	.004	-14.222	-1.196	-.065

HF (nu)

Step 1: BL HF (nu)	.473	.224	--	--	--	--
Step 2: CERQ Agg	.501	.251	.027[+]	-.273	-1.680	-.164

lnLF/HF

Step 1: BL lnLF/HF	.433	.188	--	--	--	--
Step 2: CERQ Agg	.465	.216	.028[+]	.010	1.693	.169

Note: BL = Baseline, CERQ = Cognitive Emotion Regulation Questionnaire, Agg = Aggregate CERQ (Sum of self-blame, rumination, & catastrophizing subscales), BP = Blood pressure, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, RMSSD = Root Mean Square of Successive Difference, LF = Low frequency spectral power, HF = High frequency spectral power, (ms^2) = milliseconds squared, (nu) = normalized units, LF/HF = Ratio of Low frequency to High Frequency spectral powers, lnLF/HF = Natural logarithm of ratio of Low frequency to High Frequency spectral powers
[+] = *p < 0.10*

Table 9: Correlations between Emotional Expressivity and SAM

	EES	SAM-Threat	SAM-Challenge	SAM-Stressfulness
EES	--	-.135	.034	-.040
SAM-Threat		--	-.154	.570**
SAM-Challenge			--	.112
SAM-Stressfulness				--

Note: EES = Emotional expressivity Scale, SAM = Stress Appraisal Measure
**= Correlation is significant at p < 0.01

Table 10: Hierarchical Linear Regressions of Emotional Expressivity and Cardiovascular Reactivity Controlling for Baseline Cardiovascular

Psychological Variable	Physiological Variable HR Step	R	R^2	ΔR^2	B	t	B
EES							
	Systolic BP						
	Step 1: BL SBP	.687	.472	--	--	--	--
	Step 2: EES	.689	.474	.002	.051	.602	.050
	Diastolic BP						
	Step 1: BL DBP	.660	.436	--	--	--	--
	Step 2: EES	.662	.424	.002	-.028	-.552	-.047
	Pulse						
	Step 1: BL Pulse	.668	.447	--	--	--	--
	Step 2: EES	.668	.447	.000	-.002	-.027	-.002
	RMSSD						
	Step 1: BL RMSSD	.868	.754	--	--	--	--
	Step 2: EES	.870	.757	.003	.107	.962	.054
	LF (ms^2)						
	Step 1: BL LF (ms^2)	.742	.550	--	--	--	--
	Step 2: EES	.743	.552	.001	4.472	.511	.039
	LF (nu)						
	Step 1: BL LF (nu)	.480	.230	--	--	--	--
	Step 2: EES	.480	.231	.000	.011	.102	.010
	HF (ms^2)						

Step 1: BL HF (ms^2)	.876	.767	--	--	--	--
Step 2: EES	.877	.770	.003	7.794	1.039	.056

HF (nu)

Step 1: BL HF (nu)	.473	.224	--	--	--	--
Step 2 EES	.474	.224	.000	-.014	-.137	-.014

LF/HF

Step 1: BL LF/HF	.292	.085	--	--	--	--
Step 2: EES	.294	.087	.001	.009	.353	.038

Note: BL = Baseline, EES = Emotional Expressivity Scale, BP = Blood pressure, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, RMSSD = Root Mean Square of Successive Difference, LF = Low frequency spectral power, HF = High frequency spectral power, (ms^2) = milliseconds squared, (nu) = normalized units, LF/HF = Ratio of Low frequency to High Frequency spectral powers.

Table 11: Correlations between Ambivalence over Emotional Expression and SAM

	AEQ	SAM- Threat	SAM-Challenge	SAM-Stressfulness
AEQ	--	.313**	.038	.305**
SAM- Threat		--	-.154	.570**
SAM- Challenge			--	.112
SAM- Stressfulness				--

Note: AEQ = Ambivalence over emotional expressiveness questionnaire, SAM = Stress Appraisal Measure
*** = p < 0.01*

Table 12: Hierarchical Linear Regressions of Ambivalence over Emotional Expression and Cardiovascular Reactivity Controlling for Baseline Cardiovascular

Psychological Variable	Physiological Variable HR Step	R	R^2	ΔR^2	B	T	B
AEQ							
	Systolic BP						
	Step 1: BL SBP	.687	.472	--	--	--	--
	Step 2: AEQ	.694	.481	.009	-.073	-1.188	-.099
	Diastolic BP						
	Step 1: BL DBP	.660	.436	--	--	--	--
	Step 2: AEQ	.662	.438	.002	.021	.561	.047
	Pulse						
	Step 1: BL Pulse	.668	.447	--	--	--	--
	Step 2: AEQ	.670	.449	.002	-.032	-.550	-.046
	RMSSD						
	Step 1: BL RMSSD	.868	.754	--	--	--	--
	Step 2: AEQ	.868	.754	.000	-.021	-.259	-.015
	LF (ms^2)						
	Step 1: BL LF (ms^2)	.742	.550	--	--	--	--
	Step 2: AEQ	.745	.555	.005	5.822	.915	.069
	LF (nu)						
	Step 1: BL LF (nu)	.480	.230	--	--	--	--
	Step 2: AEQ	.512	.262	.031[+]	.135	1.830	.177
	HF (ms^2)						

Step 1: BL HF (ms^2)	.876	.767	--	--	--	--
Step 2: AEQ	.876	.768	.001	-3.494	-.631	-.035

HF (nu)

Step 1: BL HF (nu)	.473	.224	--	--	--	--
Step 2: AEQ	.505	.255	.031[+]	-.135	-1.820	-.177

lnLF/HF

Step 1: BL ln*LF/HF*	.433	.188	--	--	--	--
Step 2: AEQ	.467	.218	.030[+]	.005	1.751	.174

Note: BL = Baseline, AEQ = Ambivalence over emotional expressiveness questionnaire, BP = Blood pressure, SBP = Systolic blood pressure, DBP = Diastolic blood pressure, RMSSD = Root Mean Square of Successive Difference, LF = Low frequency spectral power, HF = High frequency spectral power, (ms^2) = milliseconds squared, (nu) = normalized units, lnLF/HF = Natural logarithm of the ratio of Low frequency to High Frequency spectral powers.
[+] = *p < 0.10*

FIGURES

Figure 1: Domains of Ambivalence over Emotional Expression
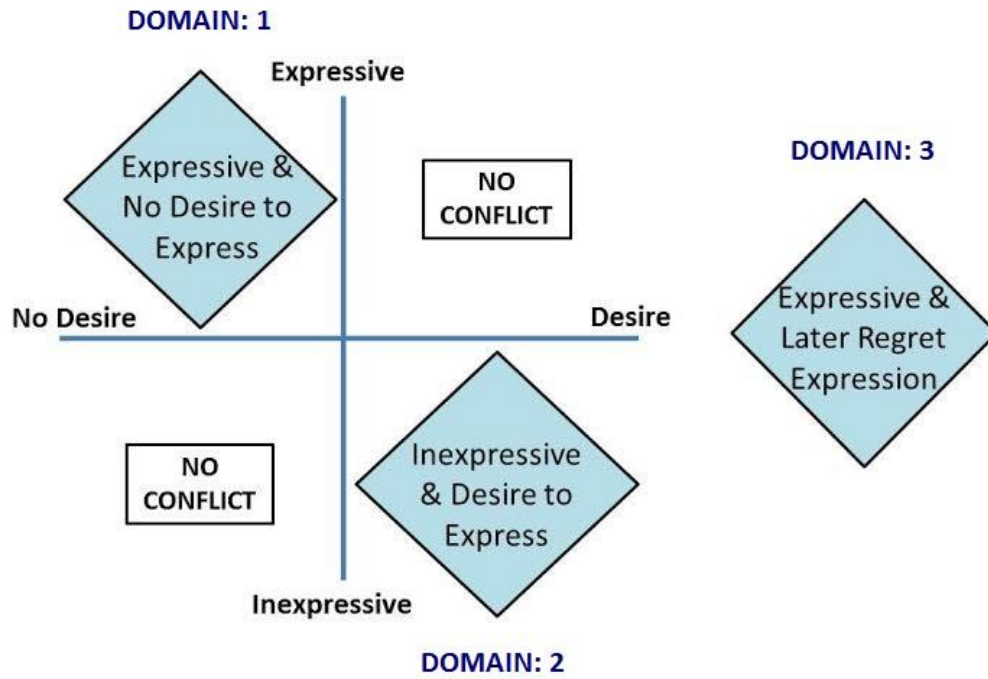
APPENDIX A: Demographic and Screening Questionnaire

IDENTIFICATION CODE # _____

Preliminary Questions

*A. Have you had any food or drink with caffeine or alcohol in the last 12 hours?
     YES____   NO____

*B. Have you had any tobacco products in the last 3 hours?
     YES____   NO____

If you answered YES to either of the above questions, please STOP and return the questionnaire to the researcher

_____

1. Age: _____

2. Height: _____

3. Weight: _____

4. Ethnicity:
    ____ Arab American
    ____ European American (White, not of Hispanic Origin)
    ____ Other: _____

5. Religion:
    ____ Atheist
    ____ Agnostic
    ____ Buddhism
    ____ Christianity
    ____ Hinduism
    ____ Islam
    ____ Judaism

_____ Other: _____

6. How many years have you lived in the United States? _____

*7. Are you currently taking any of the following types of medications?

      Stimulants (for example, Ritalin, Concerta)
      Steroids (for example, prednisone, asthma medications)
      Anti-inflammatory medications (for example, NSAIDs, Motrin/ibuprofen)
      Blood pressure medications
      Anti-depressants
      Anti-anxiety medications
      Mood stabilizers
      Other psychiatric medications

      YES___ NO___

*8. Are you currently using any over-the-counter medications, or have you in the past 2 weeks, for cold, flu, allergy, or pain?
      YES___ NO___

*9. Have you ever been told that you have high blood pressure?
      YES___ NO___

*10. Do you have a medical device (for example, a pacemaker or any implanted device)?
      YES___ NO___

*11. Do you have, or have had, any of the following medical disorders?

      Heart Attack
      Chest Pain
      Irregular Heart Beat
      Stroke
      Cardiovascular Problems
      Asthma
      Diabetes
      Kidney Disease
      Current Diagnosis of a Psychiatric Disorder such as depression or anxiety

      YES___ NO___

*12. Have you ever been diagnosed with migraine headaches?
      YES___ NO___
      If yes: Frequency and intensity _____

*13. Are you pregnant or breast feeding?
  YES___ NO___

*14. Do you have a family history of heart attack or stroke prior to age 50?
  YES___ NO___

15. Do you have any other chronic illnesses?
  YES___ NO___

16. Do you smoke?
  YES___ NO___

17. If you smoke cigarettes, on average, how many cigarettes do you smoke per day?
  Amount: _____

18. If you smoke something other than cigarettes (for example, cigars, flavored tobacco), how much do you smoke per day?
  Amount: _____

19. How many caffeine drinks do you drink in a day?
  Amount: _____

20. Do you exercise regularly (increase heart rate for at least 20 minutes, 3x /week)?
  YES____ NO____

21. How would you rate your current overall health? (Circle the best answer)

  Poor   Fair   Good   Very Good   Excellent

To be filled out by researcher:

Electrode Distance:	Count:

Task Engagement – Researcher	Task Engagement – Research Assistant

APPENDIX B: PANAS #1

This scale contains a number of words that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to the word. Indicate how you predominately feel **right now**. Base your answers on the following scale.

1	2	3	4	5
Very slightly or not at all	a little	moderately	quite a bit	extremely

_____ interested   _____ attentive

_____ distressed   _____ jittery

_____ excited   _____ active

_____ upset   _____ afraid

_____ strong   _____ in control

_____ guilty   _____ involved

_____ scared   _____ motivated

_____ hostile   _____ anxious

_____ enthusiastic   _____ angry

_____ proud   _____ sad

_____ irritable   _____ happy

_____ alert   _____ helpless

_____ ashamed   _____ frustrated

_____ inspired   _____ able to communicate

_____ nervous   _____ tense

_____ determined

APPENDIX C: SAM

This questionnaire is concerned with your **thoughts about the <u>Recall Task</u>** that you are currently completing. There are no right or wrong answers. Please respond according to how you view this situation right NOW. Please answer ALL questions. Answer each question by CIRCLING the appropriate number corresponding to the following scale.

		Not at all	Slightly	Moderately	Considerably	Extremely
1.	Does this situation create tension in me?	1	2	3	4	5
2.	Does this situation make me feel anxious?	1	2	3	4	5
3.	Is this going to have a positive impact on me?	1	2	3	4	5
4.	How eager am I to tackle this problem?	1	2	3	4	5
5.	To what extent can I become a stronger person because of this problem?	1	2	3	4	5
6.	Will the outcome of this situation be negative?	1	2	3	4	5
7.	Does this situation tax or exceed my coping resources?	1	2	3	4	5
8.	To what extent am I excited thinking about the outcome of this situation?	1	2	3	4	5
9.	How threatening is this situation?	1	2	3	4	5
10.	To what extent do I perceive this situation as stressful?	1	2	3	4	5
11.	To what extent does this event require coping efforts on my part?	1	2	3	4	5
12.	Is this going to have a negative impact on me?	1	2	3	4	5
13.	To what extent did you feel understood by the researcher?	1	2	3	4	5
14.	To what extent did you feel engaged in the discussion?	1	2	3	4	5
15.	To what extent was the topic that you discussed a meaningful event for you?	1	2	3	4	5

APPENDIX D: CERQ

How do you cope with events?

Everyone gets confronted with negative or unpleasant events now and then, and everyone responds to them in his or her own way. By the following questions you are asked to indicate **what you generally think**, when you experience negative or unpleasant events.

	(almost) never	some-times	regularly	often	(almost) always
1. 1 feel that I am the one to blame for it	1	2	3	4	5
2. I often think about how I feel about what I have experienced	1	2	3	4	5
3. I often think that what I have experienced is much worse than what others have experienced	1	2	3	4	5
4. I feel that I am the one who is responsible for what has happened	1	2	3	4	5
5. I am preoccupied with what I think and feel about what I have experienced	1	2	3	4	5
6. I keep thinking about how terrible it is what I have experienced	1	2	3	4	5
7. I think about the mistakes I have made in this matter	1	2	3	4	5
8. I want to understand why I feel the way I do about what I have experienced	1	2	3	4	5
9. I often think that what I have experienced is the worst that can happen to a person	1	2	3	4	5
10. I think that basically the cause must lie within myself	1	2	3	4	5
11. I dwell upon the feelings the situation has evoked in me	1	2	3	4	5
12. I continually think how horrible the situation has been	1	2	3	4	5

APPENDIX E: EES

DIRECTIONS: The following statements deal with you and your emotions. Please circle the number from the following scale that best describes YOU in each of the statements.

1. I don't express my emotions to other people.

Never True	Rarely True	Occasionally True	Usually True	Almost Always True	Always True
1	2	3	4	5	6

2. Even when I'm experiencing strong feelings, I don't express them outwardly.

Never True	Rarely True	Occasionally True	Usually True	Almost Always True	Always True
1	2	3	4	5	6

3. Other people believe me to be very emotional.

Never True	Rarely True	Occasionally True	Usually True	Almost Always True	Always True
1	2	3	4	5	6

4. People can "read" my emotions.

Never True	Rarely True	Occasionally True	Usually True	Almost Always True	Always True
1	2	3	4	5	6

5. I keep my feelings to myself.

Never True	Rarely True	Occasionally True	Usually True	Almost Always True	Always True
1	2	3	4	5	6

6. Other people aren't easily able to observe what I'm feeling.

Never True	Rarely True	Occasionally True	Usually True	Almost Always True	Always True
1	2	3	4	5	6

7. I display my emotions to other people.

Never True	Rarely True	Occasionally True	Usually True	Almost Always True	Always True
1	2	3	4	5	6

8. People think of me as an unemotional person.

Never True	Rarely True	Occasionally True	Usually True	Almost Always True	Always True
1	2	3	4	5	6

9. I don't like to let other people see how I am feeling.

Never True	Rarely True	Occasionally True	Usually True	Almost Always True	Always True
1	2	3	4	5	6

10. I can't hide the way I am feeling.

Never True	Rarely True	Occasionally True	Usually True	Almost Always True	Always True
1	2	3	4	5	6

11. I am not very emotionally expressive.

Never True	Rarely True	Occasionally True	Usually True	Almost Always True	Always True
1	2	3	4	5	6

12. I am often considered indifferent by others.

Never True	Rarely True	Occasionally True	Usually True	Almost Always True	Always True
1	2	3	4	5	6

13. I am able to cry in front of other people.

Never True	Rarely True	Occasionally True	Usually True	Almost Always True	Always True
1	2	3	4	5	6

14. Even if I am feeling very emotional, I don't let others see my feelings.

Never True	Rarely True	Occasionally True	Usually True	Almost Always True	Always True
1	2	3	4	5	6

15. I think of myself as emotionally expressive.

Never True	Rarely True	Occasionally True	Usually True	Almost Always True	Always True
1	2	3	4	5	6

16. The way I feel is different from how others think I feel.

Never True	Rarely True	Occasionally True	Usually True	Almost Always True	Always True
1	2	3	4	5	6

17. I hold my feelings in.

Never True	Rarely True	Occasionally True	Usually True	Almost Always True	Always True
1	2	3	4	5	6

APPENDIX F: AEQ

Please answer each item with the view to its overall meaning. Thus if a statement consisted of two thoughts, subjects were encouraged to give the item a high rating only if both thoughts applied to them.
Please circle the best answer to each question based on the following:

1 = **Never** Feel This Way
2 = **Occasionally** Feel This Way
3 = **Sometimes** Feel This Way
4 = **Often** Feel This Way
5 = **Frequently** Feel This Way

1. I want to express my emotions honestly but I am afraid that it may cause me embarrassment or hurt.	1	2	3	4	5
2. I try to control my jealousy concerning my boyfriend/girlfriend even though I want to let them know I'm hurting	1	2	3	4	5
3. I make an effort to control my temper at all times even though I'd like to act on these feelings at times.	1	2	3	4	5
4. I try to avoid sulking even when I feel like it.	1	2	3	4	5
5. When I am really proud of something I accomplish I want to tell someone, but I fear I will be thought of as conceited.	1	2	3	4	5
6. I would like to express my affection more physically but I am afraid others will get the wrong impression.	1	2	3	4	5
7. I try not to worry others even though sometimes they should know the truth.	1	2	3	4	5
8. Often I'd like to show others how I feel, but something seems to be holding be back.	1	2	3	4	5
9. I strive to keep a smile on my face in order to convince others I am happier than I really am.	1	2	3	4	5
10. I try to keep my deepest fears and feelings hidden, but at times I'd like to open up to others.	1	2	3	4	5

11. I'd like to talk about my problems with others, but at times I just can't.	1	2	3	4	5
12. When someone bothers me, I try to appear indifferent even though I'd like to tell them how I feel.	1	2	3	4	5
13. I try to refrain from getting angry at my parents even though I want to at times.	1	2	3	4	5
14. I try to show people I love them, although at times I am afraid that it may make me appear weak or too sensitive.	1	2	3	4	5
15. I try to apologize when I have done something wrong but I worry that I will be perceived as incompetent.	1	2	3	4	5
16. I think about acting when I am angry but I try not to.	1	2	3	4	5
17. Often I find that I am not able to tell others how much they really mean to me.	1	2	3	4	5
18. I want to tell someone when I love them, but it is difficult to find the right words.	1	2	3	4	5
19. I would like to express my disappointment when things don't go as well as planned, but I don't want to appear vulnerable.	1	2	3	4	5
20. I can recall a time when I wish that I had told someone how much I really cared about them.	1	2	3	4	5
21. I try to hide my negative feelings around others, even though I am not being fair to those close to me.	1	2	3	4	5
22. I would like to be more spontaneous in my emotional reactions but I just can't seem to do it.	1	2	3	4	5
23. I try to suppress my anger, but I would like other people to know how I feel.	1	2	3	4	5
24. It is hard to find the right words to indicate to others what I am really feeling.	1	2	3	4	5
25. I worry that if I express negative emotions such as fear and anger, other people will not approve of me.	1	2	3	4	5
26. I feel guilty after I have expressed anger to someone.	1	2	3	4	5
27. I often cannot bring myself to express what I am really feeling.	1	2	3	4	5

28. After I express anger at someone, it bothers me for a long time.	1	2	3	4	5

APPENDIX G: Emotion Counting Instructions for Research Assistant

Instructions: Count the total number of emotion words the participant uses during the stress recall task, including both emotions and physiological responses used to describe an emotional state.

- **Count each emotion word the participant uses during the stress recall task.**
    - For example: I felt *angry*, I was *scared*, It was a *miserable* situation to be in, I was *frustrated*, It was a *chaotic* situation, There was nothing I could do I was *helpless,* I was *desperate*, It was such an *intimidating* feeling, I felt *rejected*
- **Only count negative emotion words.**
    - Empty, Worried, Distressed, Awful, Inferior, Useless, Powerless, Worthless, Overwhelmed, Nervous, Shocked, Annoyed, Excluded, Abandoned, Humiliated
    - NOT- Enthusiastic, Excited, Devoted, Thrilled, Cherished, Like, Glad, Appreciative
- **Only count emotion words the participant verbally says.**
    - It should NOT count as an emotion word if the participant agrees with the researcher, without using the emotion word themselves. The participant must repeat the emotion word back in these instances.
        - For example: The researcher reflects back "You felt helpless in that situation" and the participant says "Yes" or "Exactly" – DOES NOT count.
        - For example: The researcher reflects back "You felt helpless in that situation" and the participant says "Yes, I did feel *helpless*" – COUNT
- **Include each time the participant describes a physiological response related to an emotional state.**
    - For example: My heart was racing, My palms were sweating, I turned bright red, I felt extremely tired, My muscles were tense, My jaw was clenched, I had a panic attack, I was shaking, My heart was pounding out of my chest
- **Record <u>the number of emotion words</u> (on the participant demographic form.)**

- **Record <u>how engaged you perceived the participant to be</u> while discussing their stressful life event (on the participant demographics form.)**
  - Both researchers should **individually** rate the participant on a **1-2-3** rating scale.
  - (1= low engagement, 2= medium engagement, 3= highly engaged)

APPENDIX H: Informed Consent

EXPERIMENTAL SUBJECT POOL PARTICIPATION

CONSENT FORM

The psychology faculty considers participation in experimental research by subjects to be an educational experience for the students as well as a most important service to the research of the University. As a part of my participation in an Introductory Psychology course at the University of Michigan – Dearborn, I hereby agree to serve as a research subject for this experiment. I have read and understood the "Subject Pool Participation" description information that I viewed when I registered on the SONA System website as a research participant. I have been informed that I may choose **not** to serve as a research subject and may instead participate in another research-related activity at no expense to my academic record or standing. I also understand that I may withdraw at any time from today's study without penalty or loss of research participation credit.

The procedure in today's study involves completing this consent form; a demographic and screening questionnaire, which includes medical and behavioral questions that can affect the study. In addition, I understand that I will be asked to complete a several questionnaires that assess emotional and cognitive functioning. I will also have my blood pressure and cardiac function assessed. Blood pressure will be monitored using a non-invasive blood pressure arm cuff, similar to what I would experience at a physician's office. A non-invasive electrocardiogram will be obtained by placing two passive electrodes, one on the right upper chest and one on the upper left abdomen. No voltage is applied to these electrodes. Cardiac output will be measured by placing two electrodes on the back of the neck and two additional electrodes on the lower back. An electric current of 400 micro-amps is applied between these electrodes. This is an extremely small current that you cannot feel. I further understand that I will have to give the female researcher access to place these electrodes and I have worn an appropriate tank top or related garment. I also understand that a small belt will be placed around my chest to measure my breathing. Finally, I understand that I will be asked to discuss with a researcher a time in my life that was very stressful.

I understand that the risks associated with my participation include: the possibility that some participants may feel angry or sad after recalling a stressful time in their life. Physical risks include the possibility of slight discomfort when the electrodes are removed (similar to removing tape or a band-aid), and mild discomfort from the blood pressure cuff when it inflates. *I understand that I will receive compensation for my research participation in the form of 1.5 credits toward my four-credit research requirement in my Introductory Psychology class if I complete the entire study or 0.5 credits if I am able to only complete the demographics questionnaire.* I understand that

no identifying information will be obtained from me, and that all of my responses today will be anonymous and confidential.

It has been explained to me that I will not receive any direct benefits from my participation as a research subject in today's study).

I have entered into this agreement with the understanding that: a) this research project has been approved by the UM-D Institutional Review Board (IRB Dearborn); b) my participation will require no more than 1.5 hours; c) the purpose and procedure as well as the benefits and risks of the study have been explained to me; and d) the results, when available, will be reported to me upon my request.

I understand that if I have questions about the study I may contact David Chatkoff, Ph.D. (Chatkoff@umd.umich.edu).

I understand that if I have questions regarding my rights as a research participant, or wish to obtain information, ask questions, or discuss concerns with someone other than the researcher(s), I may contact Debra Schneider in the IRB Administration Office, Office of Research and Sponsored Programs, 1055 Administration Building, University of Michigan-Dearborn, Evergreen Rd., Dearborn, MI 48128-2406, 313-593-5468 or email irb-dearborn@umd.umich.edu.

I hereby authorize the University of Michigan – Dearborn to utilize information gathered from my participation in this experiment for research and teaching purposes. It is understood that my name and identity will not go beyond the original experimenter's records and will be confidential unless I specifically authorize it to be used in any other.

Signature_____

Name: _____
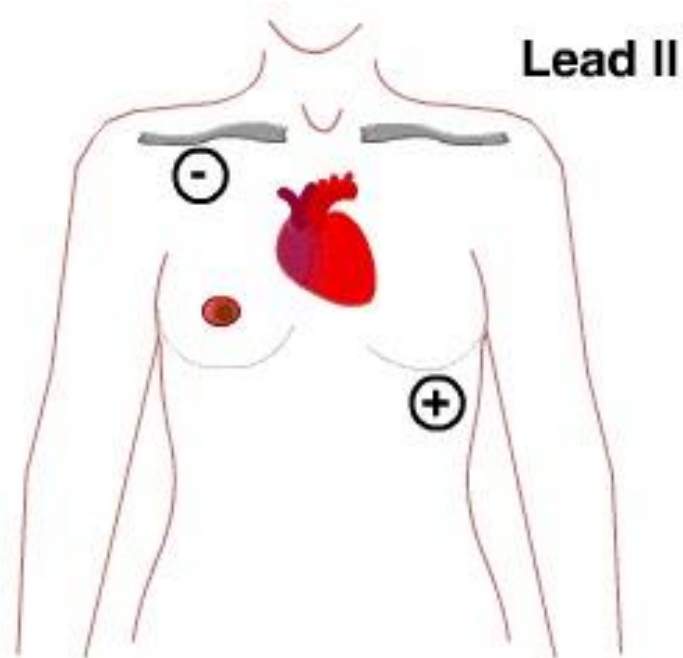
Address: _____

Enrolled in: Psychology 170_____ 171_____

Psychology Instructor_____

To be filled by
experimenter:
Experiment:

APPENDIX I: Electrocardiogram Electrode Placement

Lead II

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Health Interactive © 1999

APPENDIX J: Debriefing Form

University of Michigan – Dearborn

POST PARTICIPATION INFORMATION

Thank you for your participation in this research project. This sheet is provided as a reminder that should your participation in this project lead to a desire to seek additional services, you may contact any of the agencies listed below.

UM-D Counseling and Support Services (UM-D students only)   313-593-5430

Henry Ford Medical Center- Fairlane for Students, Faculty
and Staff (UM-D students only)   313-982-8495

Please feel free to contact either of these agencies, and once again thank you for your participation.

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