

ABSTRACT

Title of Dissertation: THE EFFECTS OF MOOD AND STRESS ON
CARDIOVASCULAR RESPONSES

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The “reactivity hypothesis” posits that elevations in cardiovascular reactivity may increase the risk of developing adverse cardiovascular events over time (Meininger, Liehr, Chan, Smith, & Mueller, 2004; Treiber, Kamarck, Schneiderman, Sheffield, Kapuku, & Taylor, 2003). Negative emotions and stress may exacerbate cardiovascular reactivity, as represented by increases in blood pressure and heart rate responses (Frasure-Smith & Lesperance, 2005; Lesperance, Frasure-Smith, Talajic, & Bourassa, 2002). However, prior studies have reported mixed results (Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000). Therefore the primary goal of the present study was to examine the effects of stimuli intended to elicit either sadness (negative mood induction) or mental stress (stressor tasks) on blood pressure and heart rate responses, in particular, when placed alongside each other in one experimental

paradigm. One hundred and six participants were randomized to one of four conditions: Mood Induction (Sad or Neutral) Only or Mood Induction (Sad or Neutral) plus Stressor Tasks. Continuous measures of heart rate and blood pressure were collected, in addition to periodic self-report measurements of sadness and perceived stress. It was first hypothesized that participants in the Mood Induction plus Stressor Task conditions would exhibit greater blood pressure and heart rate responses than the participants in the Mood Induction Only conditions. It was also hypothesized that the elicitation of a negative emotion would not predict significant increases in blood pressure and heart rate. Third, it was hypothesized that the elicitation of a negative emotion would not affect the relationship between the stressor tasks and blood pressure and heart rate. Finally, it was hypothesized that males would exhibit greater blood pressure responses than female participants. Data were analyzed using reactivity scores in a series of statistical analyses. Results supported several of the hypotheses, confirming that certain stressor tasks significantly increase blood pressure and heart rate responses. Findings also provided evidence that a negative mood induction is associated with significant increases in blood pressure and heart rate responses, even though these responses may not be unique to this specific negative emotion. Implications for future work, including clinical applications, are discussed.

THE EFFECTS OF MOOD AND STRESS ON
CARDIOVASCULAR REACTIVITY

By

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Dedication

I would like to acknowledge the astounding guidance and motivation from my friends and colleagues. I would like to dedicate this dissertation to my family. A special feeling of deep gratitude to my parents, Luz and Omar Sadak, my brother and sister-in-law, Karim and Katie Sadak, and my husband, Matthew Urcinas. I am blessed to have your continual support, encouragement, and love. Particularly to my wonderful husband who is the joy of my life. I would also like to express my appreciation to those around me who consistently helped me keep perspective on what is important in life.

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Chapter 1: Introduction

Cardiovascular disease (CVD) is one of the most prevalent health problems in the nation (Joynt, Whellan, & O'Connor, 2003). Over 79 million American adults have one or more types of cardiovascular disease (CVD) and cardiovascular diseases remain the single largest killer of American males and females (American Heart Association, 2007; American Heart Association, 2008). According to the American Heart Association (2008), there are several factors associated with the development of cardiovascular diseases that one can modify, treat, or control by changing your lifestyle or taking medicine. These risk factors include cigarette smoking, abnormal blood lipid levels, hypertension, diabetes, abdominal obesity, and a lack of physical activity. The American Heart Association (AHA) also identifies other factors that may contribute to the development of cardiovascular disease, but that have not been as strongly confirmed as risk factors (American Heart Association, 2008). These contributing factors include variables such as nutrition, alcohol consumption, and psychosocial variables.

The American Heart Association has proposed that one contributing psychosocial variable may be stress; even though it does not clearly cause cardiovascular diseases, it can set off a chain of events that contribute to the development of fatal and nonfatal cardiovascular events (American Heart Association, 2011). The National Heart, Lung, and Blood Institute (2011) also posits that although variation exists among patients regarding the sources, measurement, and intensity of mental stress, those that report high stress levels in studies show significantly increased risk of cardiovascular events. Although stress is not currently considered to be a primary risk factor, the traditional risk factors do not account for all of the variance in

cardiovascular disease, and there has been a growing body of research examining the contributions of psychosocial and psychological factors on adverse cardiovascular events (Joukamaa, Heliövaara, Knekt, Aromaa, Raitasalo, & Lehtinen, 2001; Joynt, Whellan, & O'Connor, 2003; Frasure-Smith & Lesperance, 2005; Maes, Ruckoanich, Chang, Mahanonda, & Berk, 2011; Patterson, Zakowski, Hall, Cohen, Wollman, & Baum, 1995; Smith, Uchino, Berg, Florsheim, Pearce, Hawkins, Henry, Beveridge, Skinner, & Hopkins, 2008; Tindle, Davis, & Kuller, 2010). Several studies, especially within the behavioral medicine domain, have lent support to the positive relationship between psychosocial variables such as stress and psychological variables such as negative emotions and nonfatal and fatal cardiovascular events (Ariyo, Haan, Tangen, Rutledge, Cushman, Dobs, & Furberg, 2000; Barefoot & Schroll, 1996; Frasure-Smith, & Lesperance, 2005; Lesperance, Frasure-Smith, Talajic, & Bourassa, 2002; Ibishi, Musliu, Kamberi, Qirko, Brojak, Lezha, Haxhiu, & Masala, 2009; Van der Kooy, van Hout, Marwijk, Marten, Stehouwer, & Beekman, 2007). Studies have also found that men endorsing certain elements of anger, such as anger expression, are nearly twice as likely to have heart disease as men who did not (American Heart Association, 2011; Shivpuri, Gallo, Mills, Matthews, Elder, Talavera, 2011). Findings have also suggested that patients scoring high on hostility scales, showed higher rates of cardiovascular related events such as ischemia and a longer duration of ischemia during daily activities (Guerrero & Palmero, 2010; Shimbo, Chaplin, Kuruvilla, Wasson, Abraham, & Burg, 2009). High levels of anxiety and depressive symptoms, including sadness, have also been indicated as predictors of several cardiovascular events, especially hypertension and coronary heart disease (Kubzansky, Kawachi, Weiss, & Sparrow, 1998; Kubzansky,

Cole, Kawachi, Vokonas, & Sparrow, 2006; Rutledge, Linke, Krantz, Johnson, Bittner, Eastwood, Eteiba, Pepine, Vaccarino, Francis, Vido, & Merz, 2009; Suls & Bunde, 2005). Individuals experiencing negative emotions may be at a greater risk to develop negative cardiovascular events even after adjusting for age, education, smoking, BMI, blood pressure, cholesterol, and alcohol consumption (Haukkala, Kontinen, Uutela, Kawachi, & Laatikainen, 2009; Nabi, Kivimaki, De Vogil, Marmot, & Singh-Manoux, 2009; Schwartzman & Glaus, 2000; Taylor & Barr, 2010). Therefore stress and negative emotions may be significant predictors of adverse cardiovascular events (Herrero, Gadea, Rodriguez-Alarcon, Espert, & Salvador, 2010; Mausbach, Rabinowitz, Patterson, Grant, & Schulz, 2007; Salomon, Clift, Karlsdottir, & Rottenberg, 2009; Smith, Uchino, Berg, Florsheim, Pearce, Hawkins, Henry, Beveridge, Skinner, Hopkins, & Yoon, 2008; von Kanel, Dimsdale, Adler, Patterson, Mills, & Grant, 2004).

Cardiovascular Reactivity

The variables that may shape the positive association between stress or negative emotions and the development of adverse cardiovascular events remain somewhat unclear. However, several factors have been hypothesized as mechanisms through which stress and certain negative emotions may lead to or exacerbate cardiovascular events.

One mechanism, most relevant to the hypotheses, may be cardiovascular reactivity (Gendolla & Krusken, 2002; Knox, Barnes, Kiefe, Lewis, Iribarren, Matthews, Wong, & Whooley, 2006; Neumann & Waldstein, 2001). Cardiovascular reactivity (CVR) is generally defined as cardiovascular changes or elevations to psychological or physical stimuli. The cardiovascular changes are commonly evaluated by calculating the arithmetic difference between the average of each blood pressure and heart rate measure

at baseline and the average during the presentation of stimuli. The blood pressure and heart rate measures often include systolic blood pressure (SBP reactivity, mmHg), diastolic blood pressure (DBP reactivity, mmHg), and heart rate (HR reactivity, bpm) (Clark, 2003; Gerin, Milner, Chawla, Pickering, & Phil, 1995; Herd, 1991; Knox, Hausdorff, & Markovitz, 2002; Murphy, Stoney, Alpert, & Walker, 1995; Sloan, Shapiro, Bagiella, Myers, & Gorman, 1999). Along with cardiovascular reactivity there is the reactivity hypothesis which maintains that over time, these frequent and acute elevations in blood pressure (BP) and heart rate (HR) may lead to a multitude of changes or deleterious effects in the cardiovascular system (Allen, Bocek, & Burch, 2011; Knox, Hausdorff, & Markovitz, 2002; Cacioppo, 1998; Cacioppo, Berntson, Malarkey, Kiecolt-Glaser, Sheridan, Poehlmann, Bureson, Ernst, Hawkley, & Glaser, 1998; Katholi, McCreath, Whooley, Williams, Zhu, & Markovitz, 2004; Manuck, 1994; Matthews, Zhu, Tucker, & Whooley, 2006; Treiber, Kamarck, Schneiderman, Sheffield, Kapuku, & Taylor, 2003). For instance, over time, pressure on blood flow that is often high, potentially as a reaction to negative emotions or stressors, may create weak areas or tears in the blood vessel walls which may then lead to plaque build-up or blood clots which may then lead to hypertension and other negative cardiovascular events (Cacioppo, Berntson, Malarkey, Kiecolt-Glaser, Sheridan, Poehlmann, Bureson, Ernst, Hawkley, & Glaser, 1998; Cacioppo, 1998). Therefore stress and negative emotions may increase the risk to develop an adverse cardiovascular event through a cascade of psychological and physiological variations in variables, including cardiovascular reactivity. (Brinkmann & Gendolla, 2007; Joynt, Whellan, & O'Connor, 2003; Levin & Linden, 2008; Schwartzman & Glaus, 2000). Cardiovascular reactivity (CVR) may be one underlying

mechanism explaining the relationship between specific psychological factors and negative cardiovascular events (Glynn, L'Italien, Sesso, Jackson, & Byring, 2002; Treiber, Kamarck, Schneiderman, Sheffield, Kapuku, & Taylor, 2003; Uchino, Holt-Lunstad, Bloor, & Campo, 2005). If cardiovascular reactivity is indeed one underlying mechanism in the relationship between negative emotions and adverse cardiovascular events, then there should be a positive association between negative emotions and cardiovascular reactivity. Researchers have examined the relationship between negative emotions and cardiovascular reactivity in the laboratory by using negative mood inductions, which include the presentation of a stimulus intended to elicit a negative emotion, while assessing blood pressure and heart rate (Brinkmann & Gendolla, 2007; Joynt, Whellan, & O'Connor, 2003; Levin & Linden, 2008; Kreibig, Wilhelm, Roth, & Gross, 2007; Schwartzman & Glaus, 2000).

Negative Emotions and Cardiovascular Reactivity

Several of these experimental paradigms have resulted in evidence that suggests that the elicitation of negative emotions in the laboratory, significantly elevates blood pressure and heart rate when compared to baseline measurements (Hamer, Tanaka, Okamura, Tsuda, & Steptoe, 2007; Kreibig, Wilhelm, Roth, & Gross, 2007; Lepore, Revenson, Weinberger, Weston, Frisina, Robertson, Portillo, Jobes, and Cross, 2006; Prkachin, Williams-Avery, Zwaal, & Mills, 1999; Thornton & Hallas, 1999). For example, Neumann & Waldstein (2001) found that a negative mood induction, comprised of a verbal recall task intended to elicit sadness or anger, was effective in increasing the intended emotions and resulted in significant increases in blood pressure from baseline. In addition, participants who were presented with the task that evoked sadness exhibited

higher increases in blood pressure than participants who were presented with the task that evoked anger. Another study presented participants with mood inductions consisting of different emotion-inducing film clips (Waldstein, Kop, Schmidt, Haufler, Krantz, & Fox, 2000). The healthy adult participants completed this portion of the experimental session while having their blood pressure and heart rate responses continually measured. The results showed that participants within the negative mood inductions exhibited increases in the intended emotion (anger) and significant increases in cardiovascular reactivity. Despite the significant increase in blood pressure and heart rate responses from baseline, the magnitude of these elevations was extremely low when compared to other studies examining the elicitation of anger. In contrast to these studies, there is another set of studies that has provided evidence suggesting that negative mood inductions or the elicitation of negative emotions are not associated with significant increases in blood pressure and heart rate despite self-reported increases in negative emotions following the mood induction. (Allen, Bocek, & Burch, 2011; Carroll, Phillips, Hunt, & Der, 2007; Hilmert & Kvasnicka, 2010; Gendolla & Krusken, 2001) Many of these studies include an emotion-inducing stimulus or task (read a passage, listen to music, watch a film clip, or visually recall an experience) and have resulted in participants exhibiting no significant change in blood pressure or heart rate responses from baseline or a negative association between the mood induction and cardiovascular responses (Codispoti, Surcinelli, & Baldaro, 2008; Deichert, Flack, & Craig, 2005; Etzel, Johnsen, Dickerson, Tranel, & Adolphs, 2006; Nater, Abbruzzese, Krebs, & Ehlert, 2006).

Methodological Considerations

Although the above area of research has greatly assisted in the exploration of the effects of eliciting negative emotions on vascular responses, the existing literature also has some inconsistencies. For instance, some studies have suggested that the induction of negative emotions significantly increases blood pressure and heart rate while other studies have indicated no significant association between eliciting negative emotions and blood pressure and heart rate responses. This inconsistency in data may stem from a myriad of variables including the composition of the mood inductions commonly used in the research (Gerrards-Hesse, Spies, & Hesse, 1994). For example, many of the studies that suggest a positive association contain mood inductions that consist of the presentation of a negative emotion-inducing stimulus (film clip, piece of music, written passage) or a negative emotion-specific stressor task (recall or speech task modified to elicit a specific emotion). Whereas many of the studies suggesting that there is no significant association between negative emotions and vascular responses consist of mood inductions comprised solely of the presentation of a negative emotion-inducing stimulus (film clip, listening to a piece of music, or reading a written passage).

In addition to the inconsistency in the composition of mood inductions, there is a distinction in the mean increases in blood pressure and heart rate responses within the studies suggesting a positive relationship between negative emotions and vascular responses. The studies that used mood inductions that included a negative emotion-specific stressor task (recall or speech task modified to elicit a specific emotion) or both a negative emotion-specific stressor task and negative emotion-inducing stimulus (film clip, piece of music, written passage) reported approximately three to five times greater

increases in blood pressure and heart rate responses than the studies with mood inductions comprised of solely a negative emotion-inducing stimulus. In addition, the studies that included a negative emotion-specific stressor task as part of their mood induction and found a positive association between the elicitation of negative emotions and vascular responses, seemed to fail in differentiating the effects of the negative valence of the stimuli versus the stimuli itself on vascular measures. The elevations in blood pressure and heart rate may have had less to do with the negative valence and more to do with the potential stress or arousal induced by the event itself. The task itself may exert additional direct physiological influences. Therefore the mechanism of change is less clear and it is difficult to determine whether the valence of the induction or the task itself provides greater influence, if any, on blood pressure and heart rate responses. This is a critical point given the literature suggesting the many ways in which stress may affect cardiovascular health, in particular, through increases in blood pressure and heart rate when an individual is engaged in a standardized non-specific stressor task (Joynt, Whellan, & O'Connor, 2003).

Stress and Cardiovascular Reactivity

A long-standing consensus among mainstream researchers holds that stress affects physiological responses, and most relevant to the hypotheses, that individuals exhibit significant increases in blood pressure and heart rate responses when completing certain stressor tasks (Kelsey, Ornduff, & Alpert, 2007; Linden, Gerin, & Davidson, 2003; Nealey-Moore, Smith, Uchino, Hawkins, & Olson-Cerny, 2007; Treiber, Kamarck, Schneiderman, Sheffield, Kapuku, & Taylor, 2003). As previously cited, the reactivity hypothesis posits that each exaggerated cardiovascular response (increases in blood

pressure and heart rate) may have a pathogenic effect (Cacioppo, Berntson, Malarkey, Kiecolt-Glaser, Sheridan, Poehlmann, Burleson, Ernst, Hawkley, & Glaser, 1998). Many studies have demonstrated that individuals who exhibited greater increases in systolic and diastolic blood pressure during certain stressor tasks were more likely to report negative cardiovascular events, such as hypertension, anywhere from 18 months to thirteen years later than individuals who had exhibited lower blood pressure responses (Matthews, Katholi, McCreath, Whooley, Williams, Zhu, & Markovitz, 2004; Mausbach, Rabinowitz, Patterson, Grant, & Schulz, 2007; Ming, Adler, Kessler, Fogg, Matthews, Herd, & Rose, 2004). These associations remained after controlling for variables such as baseline blood pressure readings, the development of untreated hypertensive blood pressure prior to follow-up assessments, race, gender, education, body mass index, age, and resting blood pressure.

This lends support to the idea that stress may play a role in the directionality or strength of the relationship between eliciting negative emotions and cardiovascular responses (Moseley & Linden, 2006). This also suggests the importance of examining the possibility of stressor task effects on blood pressure and heart rate when included as part of a mood induction. Therefore it may also be informative to include another measure to assess factors, such as perceived stress, that may be introduced during a mood induction task and may overcome other variable effects on cardiovascular responses. Stressors in the laboratory contribute to elevations in blood pressure and heart rate and while focusing on the physiological effects has been informative, laboratory research has had the tendency to exclude assessments of perceived stress to correspond with the physiological measures. Defining stress in terms of physiological reactivity and through the extent to

which an individual perceives the event as stressful allows one to create a more complete assessment of the effects of stress on cardiovascular reactivity. Therefore, the inclusion of the measurement of stress-related changes (blood pressure and heart rate responses) along with the inclusion of the measurement of the experience of stress (perceived state of stress) may provide improved understanding of the corresponding blood pressure responses and an overall more accurate interpretation of data that can provide additional credence to sample data.

Gender and Cardiovascular Reactivity

Stress responses are complex and incorporate multiple components including gender. However, the effects of gender differences on stress-induced blood pressure and heart rate responses are mixed and a clear pattern of differences has yet to be determined (Dedovic, Engert, Pruessner, & Wadiwalla, 2009; Delahanty, Dougall, Hayward, Forlenza, Hawk, & Baum, 2000; Fox, Garcia, Kemp, Milivojec, Kreek, & Sinha, 2006; Kolk & van Well, 2007; Lawler, Wilcox, & Anderson, 1995; Light, Turner, Hinderliter, & Sherwood, 1993; Rausch & Auerbach, 2008; Whited & Larkin, 2009). Many studies have shown that men tend to exhibit greater increases in blood pressure responses than do women when exposed to a stressor task in the laboratory (Allen & Matthews, 1997; Davis & Matthews, 1996; Lash, Gillespie, Eisler, & Southard, 1991; Lawler, Wilcox, & Anderson, 1995; Yeh, Huang, Chou, & Wan, 2009). Lepore, Ragan, & Jones (2000) had participants watch a film clip about the Holocaust and then generate a speech on the footage. There were significant blood pressure and heart rate differences between male and female participants, with males exhibiting greater reactivity than females on all measures. Allen, Stoney, Owens, & Matthews (1993) had participants complete three

different psychological tasks including a mirror tracing, stroop, and mental arithmetic task. The male participants exhibited significantly greater blood pressure than female participants in response to the subset of tasks. However, this is not always the case and at times females have shown to be equally physiologically reactive to males with blood pressure responses and demonstrate greater heart rate reactivity than men (Matthews & Stoney, 1988; Nater, Abbruzzese, Krebs, & Ehlert, 2006; Newton, 2009; Stoney, Matthews, McDonald, Johnson, 1988; Yeh, Huang, Chou, & Wan, 2009). Therefore the potential for gender to moderate cardiovascular reactivity was also a point of interest and was examined in exploratory analysis given previously identified gender differences in cardiovascular activation in response to stress reactivity (Pollard & Schwartz, 2003; Traustadottir, Bosch, & Matt, 2003).

The Present Study

Prior studies within the mood induction literature have provided important findings regarding the relationship between negative emotions and cardiovascular reactivity. However, there is still a need to further examine several basic but key components in this area of research. For instance, different experimental methods have been used to induce emotions, in particular, studies have used a single manipulation comprised of the presentation of an emotion-inducing stimulus or a manipulation comprised of an emotion-specific stressor task. In addition, the studies that suggested a positive association seem to allocate little attention to the possible effects of the negative valence of the manipulation versus the stimulus itself on blood pressure and heart rate. These studies also seem to neglect the possible additive effects of the stressor tasks on blood pressure and heart rate and on the perception of stress.

These limitations make it difficult to implicate one construct as the primary underlying force increasing blood pressure and heart rate and may overlook the magnitude of the effects of stress on cardiovascular reactivity. Cacioppo & Tassinary (1990) summarize the merits of using physiological data to understand psychological phenomena. These authors contend that in particular, physiological data can be used as indices of psychological states, such as stress. A physiological perspective can also afford psychology a deeper understanding of the physiological mechanisms involved in affective states. Because negative emotions and stress are potentially relevant to important health issues such as hypertension, and to mental health issues such as depression and anxiety, it is deserving of attention. Therefore, although this research has been accumulating over many years, many unanswered questions remain, and more systematic inquiry is needed to provide crucial progress in understanding these complex constructs. The examination of blood pressure and heart rate responses following a negative mood induction (film clip) with or without non-specific stressor tasks (mental arithmetic and speech challenge) is intended to extend improve assessment and methodology. There were two neutral mood induction conditions as well. In the neutral conditions, participants either watched a standardized neutral film clip or watched the same standardized neutral film clip followed by the non-specific stressor tasks. Neutral conditions were included with the intention of further controlling the experimental paradigm and supporting the idea that a negative mood induction or the elicitation of a single negative emotion, by itself, may not be responsible for the significant elevations in blood pressure and heart rate responses. Such findings were intended to highlight the relative contributions of negative mood states and stress to cardiovascular reactivity, and

thus potential long term risk for adverse cardiovascular events. In brief, the main objective in conducting the current research was to show the magnitude of the effect of stressor tasks on cardiovascular reactivity when combined with the presentation of an emotion-inducing stimulus (film clip) within a mood induction. The hypotheses of this study were as follows:

Primary Hypothesis: Hypothesis 1. Participants in the Mood Induction plus Stressor Task Conditions would exhibit greater blood pressure and heart responses than the participants in the Mood Induction Only Conditions.

- This would be evidenced by Time Period x Condition interaction wherein there would be a greater magnitude increase in blood pressure and heart rate responses for the stressor conditions than for the mood induction only conditions.

Hypothesis 2. The elicitation of a negative emotion would not predict significant increases in blood pressure and heart rate.

Hypothesis 3. The elicitation of a negative emotion would not affect the relationship between the stressor tasks and blood pressure and heart rate.

Hypothesis 4. Males would exhibit greater blood pressure responses than female participants.

Chapter 2: Methods

Design Overview

A 2x2 (Condition X Time Period) repeated-measures design was employed. The *Results* section of this paper provides more information regarding how this design was used within the statistical analysis to enable the appropriate testing of each hypothesis.

Apparatus & Measures

Please refer to Table 1 for a chart outlining the instruments used to measure each variable.

Table 1

Overview of Study Variables and Instruments

<u>Variable</u>	<u>Measurements</u>
Cardiovascular Reactivity	Systolic (mmHg)/ Diastolic Blood Pressure (mmHg) Heart Rate (bpm)
Depressive Symptoms	Beck Depression Inventory – Second Edition
General Anxiety	Penn State Worry Questionnaire
Demographics	Demographics Form
Social Desirability	Social Desirability Scale
Negative Emotion	Mood and Stress Inventory
Perceived Stress	Mood and Stress Inventory

Demographics. A demographics and health status questionnaire, developed by the experimenter, was administered to participants. It included questions regarding age, race, parental education level, hypertension, and heart disease. This instrument asked about

cardiovascular conditions that may affect psychophysiological measurements. Because the sample for this study is young in age, it was anticipated that the base rates of these person characteristics would be low. This was confirmed in the data analysis.

Mood and Stress Inventory. The University of Wales Institute of Science and Technology (UWIST) Mood Adjective Checklist (Matthews, Jones, & Chamberlain, 1990; UMACL) is a self-report 24 item measure that was used to assess participants' mood and stress state prior to baseline measurement and at the end of the experimental session. In particular, the present study analyzed responses from the hedonic tone and tense arousal items on the scale. The hedonic items consist of positive (happy, joyful, contented, and cheerful) and negative (sad, frustrated, depressed, dissatisfied) components to measure negative impact. In addition, the tense arousal items in the scale were used to measure current state of stress. This measure was chosen because it provides several superior aspects, compared to other state measures, that allow for discrimination between different dimensions of mood states (Matthews, Jones, & Chamberlain, 1990). In addition, this scale has the ability to assess the, "specificity or generality of stress effects" (Matthews, Jones, & Chamberlain, 1990, p.31). Participants responded by circling the number that best represents the degree to which they feel each adjective on a four-point scale. On this scale the number one represents "definitely not," the number two represents "slightly not", the number three represents "slightly" and the number four represents "definitely". The coefficient alpha for the hedonic tone positive is .77, the hedonic negative is .76, and the tense arousal is .79. In total the scale reliability ranges from .85 - .90 (Matthews, Jones, & Chamberlain, 1990).

Depressive Symptoms. The Beck Depression Inventory (BDI-II) is a 21-item self-report measure created to assess depressive symptoms (Beck & Steer, 1993; Beck, Steer, & Brown, 1996). Each item is rated on a scale ranging from 0 (normal) to 3 (most severe) and the total score can range between 0 to 63. Scores ranging from 0 to 13 are considered not depressed, scores from 14-19 mildly depressed, 20 to 28 moderately depressed, and 29 to 36 severely depressed (Beck, Steer, & Brown, 1996). Research has shown that a cutoff of 15 or greater is a valid score for identifying probable index cases of clinically significant or diagnosable major depression (Hunt, Auriemma, & Cashaw, 2003).

Depressive symptoms may increase emotional reactivity and influence the effectiveness of the manipulation making the sample more heterogeneous (Kriebig, Wilhelm, Roth, & Gross, 2007). Therefore, there was an examination of the role of depressive symptoms using the BDI-II. The BDI-II was used because it has been found to be a reliable and valid instrument to measure depression in a variety of normal and psychiatric populations (Beck, Steer, & Garbin, 1988). In addition, researchers have repeatedly found that this measure displays high internal consistency among American college students (Alansari, 2005; Beer, Steer, & Brown, 1996). The BDI-II has also been found to have good convergent and discriminant validity, in that the measure correlates strongly with depression items of the Structured Clinical Interview for Depression (Rowland, Lam, & Leahy, 2005). This measure has yielded test-re-test reliability of .96 and an internal consistency of .92 (Sprinkle, Lurie, Insko, Atkinson, Jones, Logan, & Bissada, 2002).

General Anxiety. The Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990) is a 16-item inventory commonly used trait measure of anxiety intended to assess a person's overall tendency to experience worry. It is designed

to capture the frequency, intensity and uncontrollability of worry in general, without referring to the content of specific topics. It has demonstrated great utility in screening individuals likely to meet criteria for GAD (Fresco, Mennin, Heimberg, & Turk, 2003). All items are rated on a five-point Likert-type scale ranging from 1 (not at all typical of me) to 5 (very typical of me). While 11 items are positively scored, the remaining five items (items 1, 3, 8, 10 and 11) require reverse scoring. In this way, a single total score is obtained by summing all items, and higher scores represent higher levels of pathological worry. Adequate internal consistency for the PSWQ, ranging between 0.86 and 0.95, has been consistently reported in both clinical and non-clinical samples (Fresco, Heimberg, Mennin, & Turk, 2002; Meyer et al., 1990). In addition, the test-retest reliability of the PSWQ over different time intervals ranges between 0.74 and 0.93 across three independent college samples (Meyer et al., 1990). The instrument has been proved to be useful in both adult and elderly samples, in terms of discriminating individuals with GAD from those with other anxiety disorders, as well as from the control groups (Behar, Alcaine, Zuellig, & Borkovec, 2003; Brown, 2003; Fresco et al., 2003). The convergent validity of the PSWQ is supported by significant correlations with other anxiety constructs (Belzer, D’Zurilla, & Maydeu-Olivares, 2002; Burns, Keortge, Fornea, & Sternberger, 1996; Davey, 1993; Dugas, Freeston, & Ladouceur, 1997; Stober & Joorman, 2001; van Rijsoort, Emmelkamp, & Vervaeke, 1999). Symptoms of anxiety such as worry may increase emotional and stress reactivity and influence the effectiveness of the manipulation ultimately skewing data.

Social Desirability Bias. The Social Desirability Scale-17 (Stober, 1999; SDS-17) is a self-report measure of social desirability bias that is defined as a readiness to give

biased, distorted self descriptions that portray oneself in a manner that can make a favorable impression on other (Blake, Valdiserri, Neuendorf, & Nemeth, 2006). The SDS-17 was constructed in a similar style as the Marlowe-Crowne Social Desirability Scale but with more up-to-date items. The SDS-17 is composed of 16 true-false items and one's score is increased by a true response on nine items and by a false response on seven items (Blake, Valdiserri, Neuendorf, & Nemeth, 2006). Therefore high scores indicate a strong tendency to respond in a socially desirable fashion. This scale has a Cronbach's alpha of .72, a test-retest correlation of .82 across a four-week period, and a correlation of .74 with the Marlow-Crowne Scale demonstrating substantial convergent validity (Scherrer & Dobson, 2009; Stober, 2001). Researchers have recommended that this measure be included in experimental paradigms using mood induction measures to further support that the effectiveness of the manipulation was not due to demand characteristics (Coan & Allen; 2007).

Film Clips. A ten-minute clip was used from the film "The Champ," in which the main character experiences a violent demise in the presence of a loved one for the Negative Mood Induction conditions. A ten-minute clip was used from the film "Alaska Wild Denali" for the Neutral Mood Induction conditions. This film clip consists of a silhouette of a mountain, a midnight sky, and other figures from nature while a narrator talks about the Alaskan midnight sky. Researchers have used these clips in previous mood-induction studies and they have been reported to be effective (Coan & Allen, 2007; Rottenberg, Ray, & Gross, 2007). The films were presented on a 13-inch flat panel television approximately five feet from the participant. This mood induction procedure was chosen because it has been deemed, "the most rigorous method of varying mood

states applied either in the laboratory or in the field” (Codispoti, Surcinelli, & Baldaro; Gerrards-Hesse, Spies, & Hesse, 1994). The use of this film as a mood induction procedure allows for a stimulus that can be very specific in the emotion induced (Coan & Allen, 2007). In addition, films seem to elicit emotions more automatically than other than other mood induction procedures therefore there may be less of a need to deceive participants about the purpose of the experimental paradigm in order to avoid implicit demand characteristics with participants (Kreibig, Wilhelm, Roth, & Gross, 2007).

Arithmetic Task. In the Mental Arithmetic Task, a tape instructed participants to begin with the number 956 and serially subtract eight. The participants were guided by the tape to say each answer out loud every three seconds for three minutes. This type of stress task has been used extensively in cardiovascular reactivity research and has been successful in eliciting reliable acute blood pressure responses (Clark, 2003; Glynn, Christenfeld, & Gerin, 2002; Edens, Larkin, & Abel, 1992; Hernandez, Larkin, Whited, 2009; Matthews, Woodall, & Allen, 1993).

Speech Task. In the Speech Task, participants were given three minutes to mentally organize a speech and three minutes to give a speech out loud about a neutral topic. The participants were told through a pre-recorded tape that the nature of the speech should not fall within a necessarily positive or negative category but should pertain to a neutral subject matter (Chen, Gilligan, Coups, & Contrada, 2005). Previous studies have successfully used speech tasks as a stressor to elicit reliable acute blood pressure and heart rate responses (Clark, 2006; Fontana, Diegnan, Villeneuve, & Lepore, 1999; Christenfeld, Gerin, Linden, Sanders, Mathur, Deich, & Pickering, 1997; Lepore, 1995).

Cardiovascular Measures and Apparatus. An apparatus measured cardiovascular measurements automatically, every sixty seconds, and examined during the baseline period, the mood induction, and the mood induction and arithmetic periods combined. Consistent with previous reports, reactivity scores were calculated for diastolic blood pressure (DBP reactivity, mmHg), systolic blood pressure (SBP reactivity, mmHg), and heart rate (HR reactivity, bmp) (Clark, 2003; Gerin, Milner, Chawla, Pickering, & Phil, 1995; Herd, 1991; Murphy, Stoney, Alpert, & Walker, 1995; Treiber, Kamarck, Schneiderman, Sheffield, Kapuku, & Taylor, 2003). Reactivity Scores were determined by subtracting the average measurement of the diastolic blood pressure and systolic blood pressure readings taken during the tasks from the average baseline measurement. These measurements were carried out using the IBS: SD-700A Automated Blood Pressure Pulse Rate Monitor (Thought Technology, Canada). Responses to acute psychological stressors in the laboratory, “may index the way that individuals respond to ordinary psychological demands” (Chida & Hamer, 2008). Researchers have shown that stress-induced BP readings in the laboratory are reliable and that measurements taken during one experimental session may be related to real-life events and over time remain fairly consistent within-person and across time (Kamarck, Jennings, Pogue-Geile, & Manuck, 1994; Light, Dolan, Davis, Sherwood, 1992; Spencer, 2002).

Procedure

Description of Laboratory Space

Each participant entered a room, was immediately escorted to the back of the room, and asked to sit in a cushioned chair. There were two large desks in front of the chair. On top of the desk farthest from the participant, there was a computer and a blood

pressure monitor facing the opposite direction of the participant. The laboratory space had blank white walls and was lit with two standard overhead lights.

Participant Selection

This study was approved by the University of Maryland, College Park (UMCP) Institutional Review Board. In accordance with a meta-analytic study, the range of effect sizes found have been 0.14, 0.38, 0.61 for small, moderate, and large effects (Kibler & Ma, 2004). Therefore based on previous literature, a power analysis was conducted ($1-\beta = .80$; $\alpha = .05$, $f = .30$) and revealed that a minimum of 90 total participants would be required for this study. One hundred and six participants, 18-25 years of age, were recruited from an advertisement in the SONA system which is used by the Psychology Department to allow students from the psychology undergraduate experimental pool at the UMCP to sign up for studies in return for credit. The utilization of a healthy young sample was an initial prior step to studying a more pertinent, yet biologically vulnerable population (Waldstein, Kop, Schmidt, Haufler, Krantz, & Fox, 2000). In addition, it is common that experimental work within academic institutions recruit participants who are undergraduates so that experimental credits may be given for participation. Lastly, this age sample tends to exhibit a low base rate of cardiovascular problems (American Heart Association, 2007). This allows for the highest protection possible of human subjects regarding cardiovascular measurements. A description of the experimental study was also provided to participants and asked that they refrain from smoking or drinking caffeine 12 hours prior to the study. In addition, the investigators contacted and reminded each participant who signed up of this stipulation the day before the experimental session. Exclusion criteria from participation included reports of cardiovascular problems and the

use of cardiovascular medication to avoid any exacerbations in disease symptoms due to the acute stress in the laboratory (Forman, Stampfer, & Curhan, 2009). Exclusion criteria from analysis included a self-report of smoking any substance or drinking caffeine 12 hours prior to the study. The investigator did not exclude these participants from participation but observed their data for descriptive purposes due to the capability of these drugs to increase blood pressure measurements when exposed to mental stressors (MacDougall, Musante, Howard, Hanes, & Dembroski, 1986; Robinson & Cinciripini, 2006). No participants met this criteria. All participants signed a written consent form and participants received one credit for approximately one hour of time. Detailed sample information may be found in the *Results* section of this paper. Please refer to Table 2 for a summary of inclusion and exclusion criteria for participants.

Table 2

Participation inclusion and exclusion criteria

Inclusion	Justification for Inclusion
18-25 year old college students	Initial prior step to studying a more pertinent, yet biologically vulnerable population It is common that experimental work within academic institutions recruit participants who are undergraduates so that experimental credits may be given for participation Exhibit a lowest base rate of cardiovascular problems which allows for the highest protection possible of human subjects regarding cardiovascular measurements
Exclusion	Justification for Exclusion
Self-report of a cardiovascular disease or Self-report use of medicine intended for cardiovascular problems	Acute stress in experimental session may be immediately harmful

Laboratory Session

After a student agreed to participate, s(he) was randomly assigned to one of four conditions: Neutral Mood Induction only, Negative Mood Induction only, Neutral Mood Induction plus Stressor Tasks, and Negative Mood Induction plus Stressor Task. Table 3 consists of a summary of the laboratory protocol. Table 4 provides an overview of the four experimental conditions.

Table 3

Summary of Laboratory Protocol

<u>TIME</u>	<u>Mood Induction plus Stressor</u>	<u>TIME</u>	<u>Mood Induction Only</u>
Pre-Session	Randomly Assigned Participant		Randomly Assigned Participant
t -22	Informed Consent Demographics Questionnaire Affect/Stress Inventory Beck Depression Inventory Penn State Worry Questionnaire	t-22	Informed Consent Demographics Questionnaire Affect/Stress Inventory Beck Depression Inventory Penn State Worry Questionnaire
t - 12	Equipment put into place Experimenter left room Baseline BP/HR measures	t-12	Equipment put into place Experimenter left room Baseline BP/HR measures
0	Experimenter entered room Experimenter played film Experimenter left room	0	Experimenter entered room Experimenter played film Experimenter left room
t +12	Experimenter entered room Affect/Stress Inventory Tape with task instructions turned on Experimenter left room	t+12	Experimenter entered room Affect/Stress Inventory Experimenter left room
t + 17	Arithmetic Task Speech Task	t +17	Experimenter entered room Social Desirability Scale
t +26	Experimenter entered room Affect/Stress Inventory	t+22	Social Desirability Scale Participant Debriefed
t + 33	Social Desirability Scale Participant Debriefed		

Table 4

Overview of Four Conditions

		Mood Induction	
		M ₁ negative	M ₂ neutral
Stressor	S ₁ arithmetic task speech task	M₁S₁	M₂S₁
	S ₂ no stressor task	M₁S₂	M₂S₂

Experimental session with Mood Induction and Mental Stressor Tasks. Upon arrival, the investigator randomly assigned participants to one of the four conditions: Neutral Mood Induction only, Negative Mood Induction only, Neutral Mood Induction plus Stressor Tasks, and Negative Mood Induction plus Stressor Task. Informed consent was obtained and then participants completed a demographics questionnaire, an affect and state of stress questionnaire (UWIST-UMACL), a questionnaire measuring worry (PSWQ), and a depressive symptom questionnaire (BDI-II). After these measures were completed, the participants' responses on a self-report measure of cardiovascular disease and use of medicine intended for cardiovascular problems were reviewed. If the participant provided a positive response to either or both questions, the experiment was stopped, and the participant was debriefed and excused from the experiment. There were no participants that responded positively to these two questions. Therefore after the initial measures, the experimenter put on plastic gloves, placed a blood pressure cuff on the distal humerus of the nondominant arm and a sensor on the index finger of the opposite

arm, and left the room. Baseline cardiovascular measurements of systolic blood pressure, diastolic blood pressure, and heart rate were taken for ten minutes. After the apparatus took baseline blood pressure measurements, the experimenter returned to the laboratory, reminded the participant that he/she would be watching a film clip, and then turned on the sad or neutral film. After the ten-minute film clip, the experimenter entered the room, administered the affect and state of stress questionnaire (UWIST-UMACL), and then played a tape that instructed participants on the arithmetic and speech task. These tasks were counterbalanced. After the tasks were completed, participants filled out the affect and state of stress questionnaire (UWIST-UMACL) and a social desirability scale (SDS-17). The experimental session ended with a debriefing session that included a form that contains information regarding the nature of the experimental session and contact information of the principal and student investigator.

Experimental session with Negative Mood Induction or Neutral Film only. This procedure was identical to the previously explained condition. The only difference was that there was no completion of an arithmetic and speech task in these conditions.

Chapter 3: Results

Sample Demographics

Analysis was completed with the data collected from 106 participants. In the sample, the female participants made up 52.8 % of the sample and male participants made up 47.2% of the sample. The mean age of the participants was 19 ($SD = 1.42$) years old. The majority of participants (91.5%) described their race as White and Non-Hispanic. All participants reported they did not have a past or current cardiovascular disease and they had never had psychological treatment. All participants also reported they had never taken and currently do not take any medication used to treat cardiovascular symptoms or psychological symptoms or disorders. The majority of participants (93.7%) reported they did not smoke cigarettes or chew tobacco in the thirty days prior to the experimental session. All participants reported they did not use any kind of tobacco product within 24 hours of the experimental session. A large percentage of participants (75.5%) reported they consume some kind of caffeinated beverage, and all participants reported they did not consume caffeinated beverages within 24 hours of the experimental session. The highest percentage of participants reported “mother and father academic qualifications” that fell within the category of Bachelor’s degree. The mean baseline score on the PSWQ was 24.67 ($SD = 3.79$), indicating non-clinically significant levels of worry in individuals. The mean baseline score on the BDI-II was .65 ($SD = 1.70$), indicating extremely low to non-existent depressive symptoms. The mean baseline score on the Social Desirability Scale was 6.00 ($SD = 2.99$), suggesting that the average participant answered the self-report questionnaires in an unbiased manner regarding social desirability. Participants demonstrated scores representing low baseline Negative

Emotions ($M = 31.21$; $SD = 3.71$) and Perceived Stress ($M = 12.14$; $SD = 4.12$). All participants were undergraduate students at UMCP at the time of their participation.

Differences on Baseline Measures

To measure differences among conditions at baseline, one-way analyses of variance (ANOVAs) were conducted with each of the following variables: smoking of cigarettes or chewing tobacco, number of cigarettes or chewing tobacco in the last 30 days, caffeinated beverage consumption, negative emotion, perceived stress, depressive symptoms, worry, and social desirability. No significant differences between conditions were found for Use of Tobacco Products [$F(3,102) = 1.37, p = .257$], Tobacco products in the last thirty days [$F(2,6) = .22, p = .806$], Caffeinated Beverage Consumption [$F(3,102) = .429, p = .733$], Negative Emotion [$F(3,102) = 1.18, p = .321$], Perceived Stress [$F(3,102) = 2.19, p = .09$], Worry [$F(3,102) = .591, p = .892$] or Social Desirability [$F(3,102) = .406, p = .749$].

Differences in BDI-II scores were examined across conditions and it was revealed that there were significant differences among conditions ($F(3,102) = 5.00, p = .003$). Due to a violation of homogeneity of variance across groups (Levene Statistic = 21.60; variances were unequal), A Dunnett T3 test was conducted, which assumes unequal variances. Using this stringent test, there were no significant differences among groups (all P 's $>.05$). Moreover, further examination of the BDI scores indicated that there were two outliers (two BDI scores that were greater than 2.5 standard deviations above the mean) that contributed to the unequal variances. These two data points were removed from any further analyses and there were no significant differences among conditions in BDI scores. One way ANOVAs were also conducted for all cardiovascular measures to

test for baseline differences between conditions. There were no significant differences among conditions on systolic blood pressure, $F(3,102) = 2.55, p = .06$, diastolic blood pressure, $F(3,102) = .97, p = .41$, and heart rate, $F(3,102) = 2.4, p = .07$.

Data Reduction

Data were reduced through a series of steps. Each portion of the protocol was separated into a time period to correspond to laboratory events. Physiological samples, each consisting of systolic blood pressure, diastolic blood pressure, and heart rate, were then measured at each Time Period. Cardiovascular reactivity calculations were then performed using the difference between blood pressure and heart rate responses between the baseline time period and one of the post time periods depending on the hypothesis examined. The data reduction is detailed in Table 5.

Table 5

Summary of Data Reduction

Period	Events	Calculation performed
Baseline	Baseline	Average of samples
Time Two Post All Conditions	During Movie Mood Induction	Average of samples
Time Three Post Stressor Conditions Only	During Stressor Tasks	Average of samples

Manipulation Check

In order to test the effectiveness of the negative mood induction manipulation, a paired sample *t*-test was conducted and it was found that participants in the negative mood induction groups reported a significantly higher degree (expressed through lower scores on the ACL) of negative emotions ($M = 2.16, SD = .19$) than those in the neutral mood groups ($M = 5.45, SD = .27$), $t(74) = 6.99, p < .001$. These results indicate that the manipulation was effective.

Hypothesis Testing

Please refer to Table 6 for details on grouping of Conditions for Hypothesis Testing.

Table 6

Grouping of Conditions for Hypothesis Testing

Conditions	Hypothesis One	Hypothesis Two and Three
Stressor	Negative Mood Induction plus Stressor Tasks Neutral Mood Induction plus Stressor Tasks	Negative Mood Induction plus Stressor Tasks versus Neutral Mood Induction plus Stressor Tasks
No Stressor Tasks	Negative Mood Induction Only Neutral Mood Induction Only	
Negative Mood Inductions		Negative Mood Induction Only Negative Mood Induction plus Stressor Tasks versus
Neutral Mood Inductions		Neutral Mood Induction Only Neutral Mood Induction plus Stressor Tasks

Hypothesis One – Participants in the Mood Induction plus Stressor Tasks conditions will exhibit greater blood pressure and heart rate responses than the participants in the Mood Induction Only conditions.

In order to examine the primary hypothesis, a Mixed Model General Linear Model (GLM) was used with Condition (Mood Induction plus Stressor Tasks vs. Mood Induction only) as a categorical between-subjects factor and Time Period (Baseline vs. Posts) as a categorical within-subjects factor. Therefore the four conditions were collapsed into two groups with one group including the two Mood Induction plus Stressor conditions (Stressor Task Conditions) and one group including the Mood Induction only conditions (No Stressor Task Conditions). There was a Condition X Time Period interaction on systolic blood pressure $F(1,104) = 233.013, p < .001$ (Figure 1). There was a Condition X Time Period interaction on diastolic blood pressure $F(1,104) = 140.714, p < .001$ (Figure 2). There was a Condition X Time Period interaction on heart rate $F(1,104) = 270.718, p < .001$ (Figure 3). In order to further investigate the nature of this interaction, two repeated measures t-tests were conducted for each Condition (Mood Induction plus Stressor Task and Mood Induction Only) on SBP, DBP, and HR. These analyses indicated that although the cardiovascular responses significantly increased from Baseline to Post for the Mood Induction plus Stressor conditions (Negative and Neutral Mood Inductions plus Stressor Tasks) and Mood Induction Only conditions (Negative and Neutral Mood Inductions only), the magnitude of the increase was greater for the Mood Induction plus Stressor conditions. Therefore the magnitude of the effect was greater for the Mood Induction plus Stressor conditions from Baseline to Post (Time Three) than the Mood Induction only conditions from Baseline to Post (Time Two).

Please refer Table 7 for data demonstrating the magnitude of the effect (means and standard deviations).

Figure 1

Interaction of Condition and Time Period on Systolic Blood Pressure

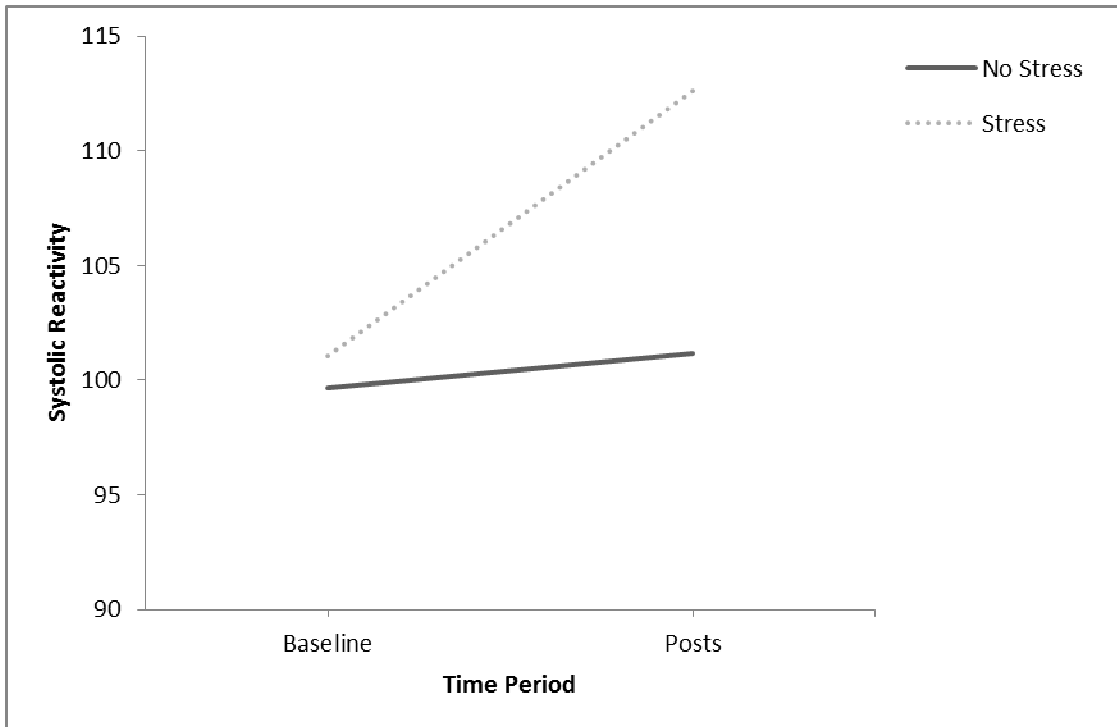


Figure 2

Interaction of Condition and Time Period on Diastolic Blood Pressure

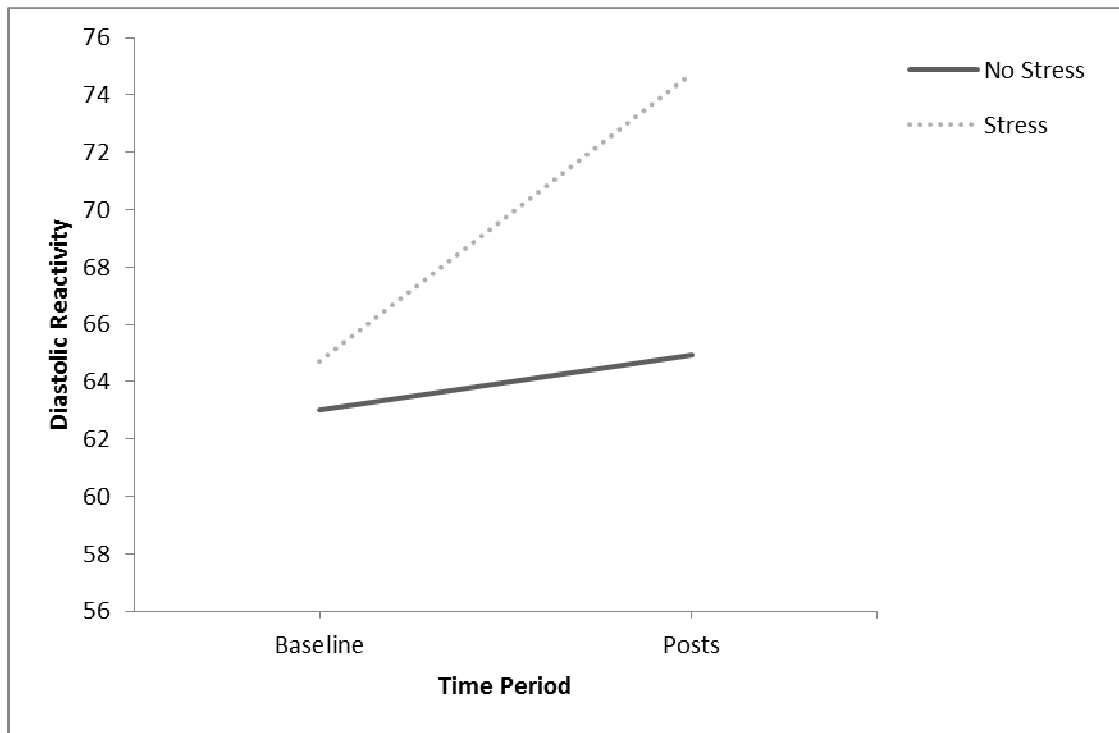


Figure 3

Interaction of Condition and Time Period on Heart Rate

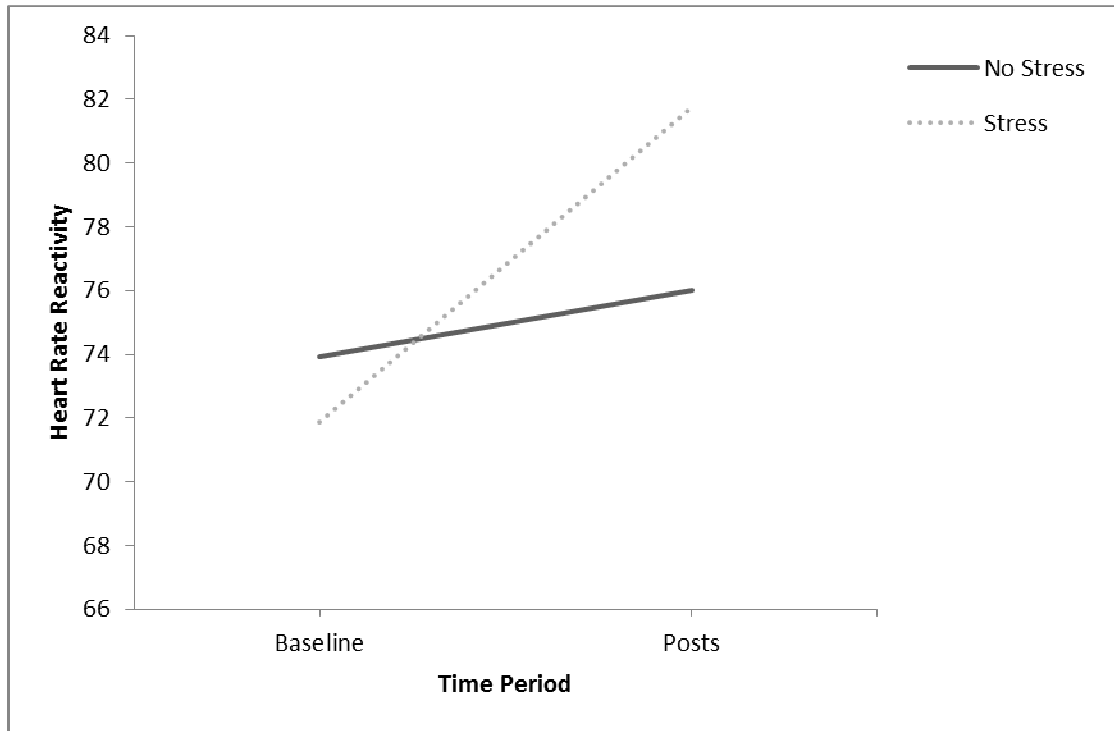


Table 7

Summary of Mean Differences in Hypothesis One

Source	NS Conditions at Baseline	NS Conditions at Post	S Conditions at Baseline	S Conditions at Post	<i>t</i>	<i>df</i>	<i>p</i>
SBP***	99.7 (4.8)	101.2 (4.7)	101 (3.7)	112 (5.5)	21.4	61	<i>p</i> <.001
DBP***	63.0 (5.8)	64.9 (5.8)	64.7 (7.5)	74.8 (7.7)	21.5	61	<i>p</i> <.001
HR***	73.9 (8.6)	76.0 (8.7)	71.9 (6.3)	81.8 (6.6)	29.6	61	<i>p</i> <.001

Note. Standard deviations appear in parentheses next to means. SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; HR = Heart Rate; NS = No Stressor; S = Stressor. ***=*p*<.001.

Hypothesis Two - The elicitation of a negative emotion will not predict significant increases in blood pressure and heart rate.

To test the second hypothesis, a Mixed Model GLM was conducted with Condition (Neutral Mood Induction only vs. Negative Mood Induction only) as a between- subjects variable and Time Period (Baseline versus Time Two Post) as a within subjects factor. Therefore the four conditions were collapsed and the Negative Mood Induction conditions (Negative Mood Induction only and Negative Mood Induction plus Stressor Task) were in one group and the Neutral Mood Induction conditions (Neutral Mood Induction only and Neutral Mood Induction plus Stressor Task) in another group. There was no Condition X Time Period interaction on systolic blood pressure $F(3,102) = .605, p = .614$, diastolic blood pressure ($F(3,102) = 1.174, p = .323$), or heart rate ($F(3,105) = .486, p = .693$). There were no significant differences evidenced in systolic blood pressure, diastolic blood pressure, and heart rate responses from Baseline to Time Two Post (from the beginning to the end of the Mood Inductions) despite being presented a negative or neutral mood induction. There were no significant differences in mean increases of blood pressure and heart rate responses based on the valence of the mood induction.

Hypothesis Three - The elicitation of a negative emotion will not affect the relationship between the stressor tasks and blood pressure and heart rate responses.

To further examine negative emotions, a Mixed Model GLM was conducted with Condition (Neutral Mood Induction plus Stressor Tasks vs. Negative Mood Induction plus Stressor Tasks) as a between subject variable and Time Period (Baseline vs. Time Three Post) as a within subjects factor. There was no Condition X Time Period

interaction on systolic blood pressure $F(1, 60) = .256, p = .614$, diastolic blood pressure $F(1,60) = .263, p = .610$, or heart rate $F(1,60) = .02, p = .880$. There were no significant physiological differences. Therefore, the two Mood Induction plus Stressor Task conditions evidenced similar increases in systolic blood pressure, diastolic blood pressure, and heart rate from Baseline to Time Three (from the beginning to the end of the Stressor Tasks) whether they received a negative or neutral mood induction. Again, there were no significant differences in increases of vascular responses based on the valence of the mood induction.

Hypothesis Four - Males will exhibit greater blood pressure responses than female participants within stressor conditions.

To explore gender differences, a Mixed Model GLM was conducted with Gender as a between subject variable and Time Period (Baseline vs. Time Three Post) as a within subjects factor. There was no Gender X Condition interaction on all cardiovascular measures between all time points (all p 's $> .05$) except diastolic blood pressure. There was a significant Gender X Time Period (Baseline vs. Time Three) interaction for diastolic pressure, $F(1, 60) = 4.98, p = .02$ (Figure 4). Males exhibited significantly greater increases than females on diastolic blood pressure only within the Mood Induction plus Stressor conditions during the stressor tasks. The nature of the interaction was one of magnitude therefore both males and females exhibited significant increases in diastolic blood pressure responses from baseline to throughout the Stressor Tasks but males demonstrated even greater increases than females. Please refer to Table 8 for data demonstrating the magnitude of the effect (means and standard deviation).

Figure 4

Interaction of Gender and Time Period on Diastolic Blood Pressure

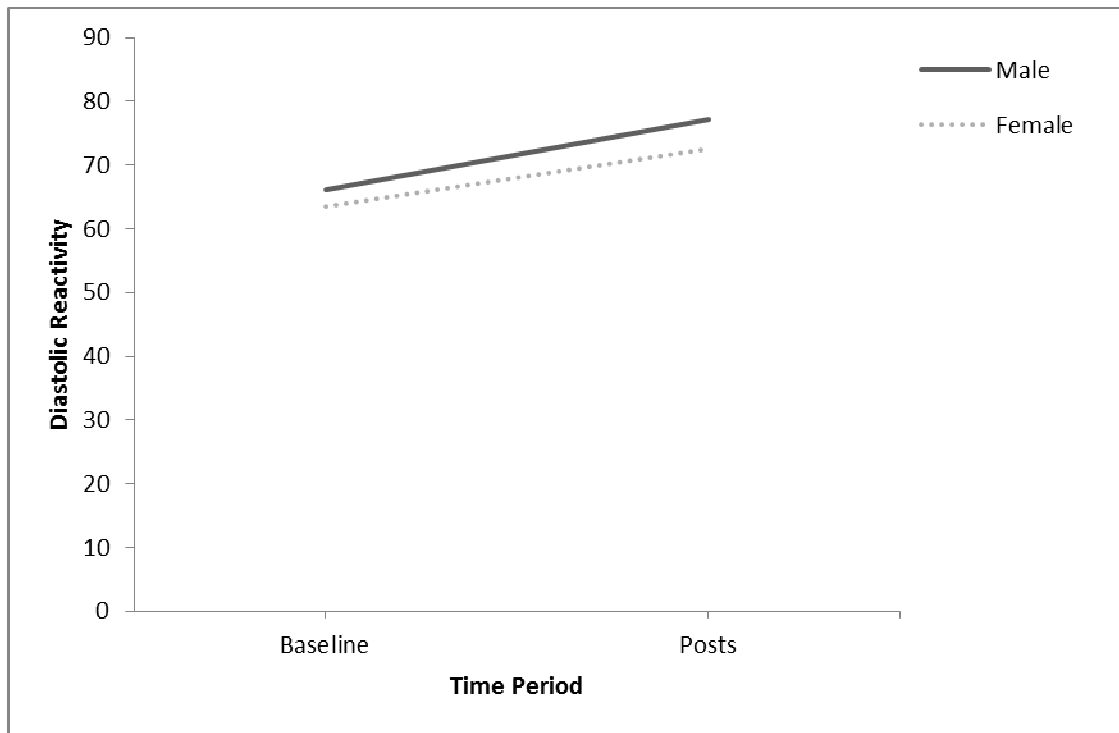


Table 8

Summary of Mean Differences Between Males and Females in Stressor Conditions

Source	Males at Baseline	Males at Post	Females at Baseline	Females at Post	<i>t</i>	<i>df</i>	<i>p</i>
SBP	101.3 (3.9)	113.7 (5.6)	100.9 (3.6)	111.6 (5.2)	--	31	<i>p</i> =.091
DBP***	66.0 (7.1)	77.1 (7.5)	63.5 (7.7)	72.5 (7.3)	20.7	31	<i>p</i> <.001
HR	71.9 (6.8)	81.7 (7.3)	71.8 (5.9)	81.8 (6.1)	--	31	<i>p</i> =.74

Note. Standard deviations appear in parentheses next to means. SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; HR = Heart Rate. No *t* - tests were completed for SBP and HR because there was no interaction present. ***=*p*<.001.

Chapter 4: Discussion

Cardiovascular reactivity assessment is one approach to measuring the dynamic nature of the cardiovascular system in order to establish heightened cardiovascular reactivity as an important individual difference and elucidate the role of psychosocial factors in the pathogenesis of cardiovascular disease. The “reactivity hypothesis” posits that cardiovascular reactivity or exaggerated cardiovascular responses, such as elevations in systolic and diastolic blood pressure, to stimuli, may increase the risk of developing various forms of cardiovascular disease over time (Meininger, Liehr, Chan, Smith, & Mueller, 2004; Treiber, Kamarck, Schneiderman, Sheffield, Kapuku, & Taylor, 2003). This risk may be hypothetically increased by the elicitation of negative emotions and stress. However, studies that have utilized negative mood inductions to test the effects of negative emotions on vascular responses have reported mixed results (Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000).

The inconsistency in results may, in part, be due to the differences in composition of the mood inductions, the possible distinction between the effects of the emotion-specific portion of the manipulation and the event itself on blood pressure and heart rate responses, and the possible effects of the stressor task on the perception of stress. It is difficult to determine whether the negative valence of the mood induction, the event itself, or a combination of the negative stimulus and stress tasks constitute the primary influence on blood pressure and heart rate responses. The mechanism of change is not clear within the studies suggesting a positive association between the elicitation of negative emotions and certain cardiovascular responses. Therefore the major aim of the present study was to examine the effects of discrete stimuli intended to elicit either

sadness (negative mood induction) or mental stress (stressor tasks) on blood pressure and heart rate responses, in particular, when placed alongside each other in one experimental paradigm. In addition, the current study intended to provide data on perceived stress and explore gender effects on vascular responses.

It was hypothesized that participants in the Mood Induction plus Stressor Task conditions would exhibit greater blood pressure and heart rate responses than the participants in the Mood Induction Only conditions. Data supported this hypothesis. Participants within the Mood Induction plus Stressor Task conditions exhibited more than double the increase in blood pressure and heart rate responses from baseline, when compared with participants in the Mood Induction Only conditions. Even though blood pressure and heart rate responses significantly increased from baseline measurements in all conditions, participants within the conditions including non-specific stressor tasks exhibited significantly greater change in their blood pressure and heart rate responses. These results further support the research suggesting that acute stressor events may predict significant increases in cardiovascular responses. In addition, the differences of magnitude in cardiovascular reactivity between the Mood Induction plus Stressor Task and Mood Induction Only conditions is a pattern not only of statistical significance but potential clinical importance when taking into account that, “each 20-mm HG increase is systolic and diastolic blood pressure doubles the risk for cardiovascular diseases” (Ostir, Berges, Markides, & Ottenbacher, 2006). Many individuals face a multitude of stressors on a daily basis. If these acute stressors are accompanied by heightened blood pressure or heart rate responses, especially at the magnitude observed in the present study, the long-term deleterious nature of cardiovascular reactivity becomes somewhat more

understandable. Every exaggerated blood pressure response to a stressor may convert into an acute surge of blood that, repeatedly increases the pressure against the walls of the arteries and gradually damages the walls of the arteries, potentially impairing different components of the cardiovascular system such as blood flow (Purebl, Birkás, Csoboth, Szumska, & Kopp, 2006). This pathway to impairment is just one way in which excessive reactivity may be positively associated with negative cardiovascular events over time. Present results thus provide further support for the considerable effects stress may have on blood pressure and heart rate, supporting the hypothesis that repetitive stress may contribute to long-term negative effects on the cardiovascular system. This also provides support for the need of research, beyond the scope of this dissertation, focusing on the particular patterns of blood flow surrounding stress and testing the hypothesis that these patterns may have long-term deleterious effects.

The difference in blood pressure and heart rate elevations between the Mood Induction plus Stressor Tasks and Mood Induction Only conditions may have been due to several environmental and psychological factors. In regards to the external variables, the Mood Induction plus Stressor Task conditions included two additional stimuli when compared with the Mood Induction Only Conditions. The two additional stimuli, the mental arithmetic and speech task, are considered active tasks and have been characterized as stimulating greater effects on blood pressure and heart rate than a passive task such as watching a film clip (Dishman, Jackson, & Nakamura; 2002; Gianaros, Matthews, Jennings, Manuck, and Hariri, 2008; Schwerdtfeger & Rosenkaimer, 2011). Active tasks may involve more cognitive participation, performance demands, and perceived stress, whereas a passive stimulus may only require recognition

without any performance demands. This is consistent with the participants within the Mood Induction plus Stressor conditions who self-reported significant increases in stress from baseline, whereas participants within the Mood Induction Only Conditions did not.

Coping strategies may also have had an effect on psychological variables such as coping strategies. For instance passive coping may, in part, explain why participants in the Mood Induction plus Stressor conditions exhibited greater increases in their vascular responses. In the traditional transactional perspective, two types of coping have been identified (Lazarus & Folkman, 1984). Active coping is thought to alleviate stress by allowing the individual to exert control over the stressor, thereby attenuating its effect. Active coping involves planning, problem-solving, and seeking instrumental support. By contrast, in passive coping, attention is internally directed toward the individual's emotional, cognitive, and physiological experiences of a particular stressor. It may entail a variety of coping responses, such as the processing and expression of emotion, denial, avoidance, and seeking emotional support. The long-standing consensus among mainstream researchers holds that active coping is more effective at alleviating stress (Amirkhan, 1990; Dixon, Heppner, Burnett, Anderson, & Wood, 1993; Folkman & Lazarus, 1988; Nezu, 1987), whereas passive coping is associated with maladaptive outcomes, such as self-reported stress and depressive symptoms (Barnes & Lightsey, 2005; Caughy, O'Campo, & Muntaner, 2004; DeGenova; Patton, Jurich, & MacDermind, 1994; Ravindran, Matheson, Griffiths, Merali, & Anisman, 2002; Wegner & Zanakos, 1994) In addition, some theorists have speculated that passive coping strategies are associated with heightened diastolic reactivity and higher blood pressure longitudinally because they prolong sympathetic activation in the short-term (Armstead, Lawler,

Gorden, Cross, & Gibbons, 1989; Clark & Anderson, 2001; Shwerdtfeger, Schmukle, & Egloff, 2005; Silvestrini & Gendolla, 2009). Therefore the greater elevations in blood pressure and heart rate responses within the Mood Induction plus Stressor conditions may be due, in part, to the implementation of passive coping or how the participants decided to direct his/her attentional resources. However, coping is a dynamic process and a “one-size-fits-all” approach to coping with stress may not account for the changes.

It was also hypothesized that the negative mood induction (elicitation of sadness) would not predict significant increases in cardiovascular responses. The findings showed no significant difference in blood pressure and heart rate responses between the Negative Mood and the Neutral Mood Induction Only conditions from Baseline to Time Two Post (throughout the film clips). A related hypothesis stated that the negative mood induction (elicitation of sadness) would not moderate the effects of stress on blood pressure and heart rate responses. Study results showed no significant difference in blood pressure and heart rate responses between the Negative Mood Induction plus Stressor Task conditions and the Neutral Mood Induction plus Stressor Task conditions. Despite participants within the negative mood induction groups reporting significant increases in sadness from baseline and participants in the neutral mood induction groups reporting no significant increases in sadness, the valence of the mood induction did not predict a unique pattern of physiological responses at any time point. Therefore significant increases in blood pressure and heart rate responses, within the negative mood induction groups, may have been due primarily to the arousal from having to mobilize attention to a demanding stimulus (a film clip placed in front of participants). Affective responses may not uniquely correspond to physiological responses. Therefore, one may attribute these

findings to the nature of the stimulus and not to the valence of the stimulus used within the negative mood induction.

The data provided results suggesting that the stimulus used in the Negative Mood Induction conditions produced emotional change in a negative direction and significantly increases physiological responses. However, it may have perhaps lacked the degree of potency to exceed the level of arousal produced by the neutral mood induction in this specific sample. The stimulus may have needed an even greater potency for this particular sample to match or surpass the magnitude of reactivity produced by the neutral film clip. The emotional stimulus seems to not have been preferentially processed in comparison with neutral mood induction. This may explain why previous studies, using a variety of mood induction stimuli, suggested there is not a positive relationship between negative mood inductions and cardiovascular responses. As mentioned earlier, participants did not significantly differ at baseline on negative emotions.

The analyses did not entirely support the hypothesis concerning gender. Both males and females in all conditions exhibited significant increases in blood pressure and heart rate responses from baseline. However, males exhibited significantly greater diastolic blood pressure reactivity than females within the Mood Induction plus Stressor conditions during the stressor tasks. There were no other significant differences between males and females. It is somewhat unclear why the magnitude of increase in diastolic activation was significantly greater for males than females in the Mood Induction plus Stressor conditions. As considered earlier, an inhibited or passive coping approach may increase the likelihood of greater diastolic blood pressure reactivity and participants within the Mood Induction plus Stressor Task conditions may have responded to the

stressors with passive coping strategies (Armstead, Lawler, Gorden, Cross, & Gibbons, 1989; Clark & Anderson, 2001; Gramer & Saria, 2007; Shwerdtfeger, Schmukle, & Egloff, 2005; Silvestrini & Gendolla, 2009). Also, greater systolic blood pressure reactivity has been characterized as an index of effort (Burgo & Gendolla, 2009). Taken together, this may indicate that the males perceived these stressors differently than females and therefore used passive coping strategies to a greater extent than females and put in less active effort when faced with the stressor tasks. In addition, many studies have speculated that gender differences in cardiovascular responses to laboratory stressors may be due to sex role socialization. This may result in men being more competitive than women, and therefore that men might show greater cardiovascular reactivity to competitive laboratory mental stressors than women (Lash, Gillespie, Eisler, & Southard, 1991). Given the potential gender differences in perceived stress and cardiovascular responses to challenges, it is recommended that future studies assess what is driving the different acute stress responses between men and women. Emotional and stress activation may require more careful examination among men and women separately.

Limitations and Future Directions

There were several limitations that warrant identification and may indicate future directions for research. First, as noted above, the manipulations that were used generate some concerns. In particular, the mood induction, composed of a film clip, was intended to elicit a specific negative emotion. The type of mood induction used has been identified as a standardized procedure to significantly induce sadness and it did significantly elevate

self-reported sadness and cardiovascular responses in participants in the present study. However manipulation measures that were used may have been insufficient in assessing the complexity of sadness and stress (Kreibig, Wilhelm, Roth, & Gross, 2007). When viewed from a dimensional approach, emotions may exist as affective states positioned on a continuous dimension where they may be intercorrelations or overlap among affective states (Gerrards-Hesse, Spies, & Hesse, 1994; Shapiro, Jamner, Goldstein, & Delfino, 2001). Therefore a mood induction intended to elicit a single emotion may have the potential to induce several emotions. These emotions may integrate and affect each other and ultimately lead to different or unexpected cardiovascular responses. Sadness and stress encompass a broad system of sentiments and reactions and a single self-report measure used to index each variable may not sufficiently tap into the potential and assorted components elicited by each stimulus. Therefore future studies that include measures that take into account a broad spectrum of affective states may assist in a more comprehensive understanding of the participants experience during and following a mood induction and stressor tasks in the laboratory.

Another limitation regarding the composition of the mood induction is that the present study used only one method to elicit emotions (film clip). The use of other mood induction methods (relived emotion, imagery, slides) on the same sample may have helped in drawing a conclusion as to whether the current findings represent a distinctive emotion-behavior relationship. A direct comparison of these methods in a comprehensive design may also be a reasonable goal in order to attempt a replication of the present findings. It may also be beneficial to replicate this type of study but with the elicitation

of a broader range of negative emotions in order to learn the full range of possible effects of sadness and stress on cardiovascular reactivity.

Another limitation may be that the laboratory design used here was short-term and used only small intervals of time (e.g., minutes), this limits the generalizability of findings to everyday life. For example, the time taken to complete each stressor task was less than ten minutes, which is arguably much less time than is ordinarily spent in real life situations. Moreover, in “real world” conditions, an individual’s response is not always halted in order to move onto the next task – individuals may respond in their preferred ways for several minutes to many hours without interruption. However, in the laboratory, their attention is rather quickly diverted to the next task. These obvious differences in the response process may cause some of the current findings to poorly translate to real-life settings. Future research should seek to increase ecological validity. For instance, one interesting paradigm would be to study ambulatory blood pressure and heart rate alongside daily ratings of sadness or stressful events. Such a research design would allow for a snapshot of how blood pressure and heart rate responses may respond in concrete, demanding, and emotionally salient situations that laboratories cannot always replicate. In addition, the laboratory protocol created at the outset of the study provided an outline of the length of each measurement period, however, these guidelines could not account for individual differences in speed. Therefore, this possible limitation might have caused differences in the ways that participants responded to the protocol. For example, it is tenable that a participant who went through the speaking portion of the speech task very quickly may not have engaged in the task as well as one who completed the task deliberately and thoughtfully. However, it is unlikely that these differences are tied

systematically to any particular condition to the point that the above results are invalidated. Also implementing a longitudinal design in the future may assist in learning the reliability of these cardiovascular responses in the laboratory and their predictive validity for ambulatory cardiovascular measures or future cardiovascular events.

Furthermore, even though participants were asked about past and current mental health treatment, full psychiatric screenings were not performed with participants. This may have resulted in the inclusion of participants who met criteria for psychiatric disorders beyond the scope of the BDI-II and PSWQ and may have affected how the mood inductions were cognitively appraised and then manifested through physiological responses. Given the proper resources and time, future studies may aim to have participants present to the laboratory for a longer period of time to complete a full psychiatric screening and to have participants present for a second time to examine the reliability of the mood induction and stressor tasks to induce specific emotions. This recommendation is given with caution due to other variables that may be introduced with repeated exposure to stimuli such as practice effects and habituation.

Another limitation is the homogeneity of the sample examined in the current study. All participants were college undergraduates from the University of Maryland, College Park Introductory Psychology Participant Pool. In addition, participants scored extremely low on the BDI-II in comparison to the average BDI-II scores in college samples (Carmody, 2005). This sample may respond to the mood induction and stressor tasks in a unique way and may need stimuli that elicit more intense emotional states in order to differentiate their relations to physiological measures. In addition, it is not known whether the current findings would generalize to other populations in which

cultural or age differences may exist or to populations in which individuals are leading less healthy lifestyles. These issues are all reasons to replicate this study with a more representative sample. Future research may also aim to examine a population with greater risk for adverse cardiovascular events such as older individuals or individuals with family history of cardiac instability. However, it is notable that significant results were found even in this highly select sample, suggesting that the current hypotheses might be supported in a “real-life” sample. Unfortunately, it is impossible to ascertain whether the current results extend to other demographic groups based on the present data.

Conclusion

The overall findings of the current study emphasize several issues that, if supported by additional research, may suggest the development and implementation of psychosocial interventions. The current study provides evidence that certain stressor tasks significantly increase blood pressure and heart rate responses in a statistically significant and potentially clinically important manner. It also provides evidence that the induction of sadness is associated with significant increases in blood pressure and heart rate responses, even though these responses may not be unique to this specific negative emotion. It has been hypothesized that exaggerated blood pressure and heart rate responses to negative emotions and stress may position an individual at higher risk to develop negative cardiovascular events (Cacioppo, 1998; The National Heart Lung and Blood Institute, 2011). Although sadness and stress are complex constructs, the association between stress/negative emotions and cardiovascular responses in the analysis suggests that the adaptive management of recurrent stress and negative emotions may position healthy

individuals at lower risk for negative cardiovascular events, even if the baseline risk may already be relatively low. Therefore it is important to continue to put forth effort and learn the pathways through which psychosocial and psychological factors may work to influence health and develop related cognitive-behavioral interventions practical for primary care and mental health settings.

Appendix A: Literature Review

Cardiovascular Disease

Cardiovascular disease (CVD) is one of the most prevalent health problems in the nation (Joynt, Whellan, & O'Connor, 2003). Over 79 million American adults have one or more types of cardiovascular disease (CVD) and cardiovascular diseases remain the single largest killer of American males and females (American Heart Association, 2007; American Heart Association, 2008). According to the American Heart Association (2007), there are six major risk factors you can modify, treat, or control by changing your lifestyle or taking medicine. These risk factors include cigarette smoking, abnormal blood lipid levels, hypertension, diabetes, abdominal obesity, and a lack of physical activity. The American Heart Association (AHA) also identifies other factors that may contribute to the development of cardiovascular disease, but that have not been as strongly confirmed as risk factors (American Heart Association, 2008). These contributing factors include variables such as nutrition, alcohol consumption, and psychosocial variables. The American Heart Association has proposed that one contributing psychosocial variable may be stress; even though it does not clearly cause cardiovascular diseases, it can set off a chain of events that contribute to the development of fatal and nonfatal cardiovascular events (American Heart Association, 2011). The National Heart, Lung, and Blood Institute (2011) also posits that although variation exists among patients regarding the sources, measurement, and intensity of mental stress, those that report high stress levels in studies show significantly increased risk of cardiovascular events. Although stress is not currently considered to be a primary risk factor, the traditional risk factors do not account for all of the variance in cardiovascular disease, and there has been a growing

body of research examining the contributions of psychosocial and psychological variables on adverse cardiovascular events (Joukamaa, Heliövaara, Knekt, Aromaa, Raitasalo, & Lehtinen, 2001; Joynt, Whellan, & O'Connor, 2003; Frasure-Smith & Lesperance, 2005; Maes, Ruckoanich, Chang, Mahanonda, & Berk, 2011; Patterson, Zakowski, Hall, Cohen, Wollman, & Baum, 1995; Smith, Uchino, Berg, Florsheim, Pearce, Hawkins, Henry, Beveridge, Skinner, & Hopkins, 2008; Tindle, Davis, & Kuller, 2010). Several studies, especially within the behavioral medicine domain, have lent support to the positive relationship between stress and other psychological variables, including negative emotions, and nonfatal and fatal cardiovascular events (Ariyo, Haan, Tangen, Rutledge, Cushman, Dobs, & Furberg, 2000; Barefoot & Schroll, 1996; Frasure-Smith, & Lesperance, 2005; Lesperance, Frasure-Smith, Talajic, & Bourassa, 2002; Ibishi, Musliu, Kamberi, Qirko, Brojak, Lezha, Haxhiu, & Masala, 2009; Van der Kooy, van Hout, Marwijk, Marten, Stehouwer, & Beekman, 2007). Studies have also found that men endorsing certain elements of anger, such as anger expression, are nearly twice as likely to have heart disease as men who did not (American Heart Association, 2011; Shivpuri, Gallo, Mills, Matthews, Elder, Talavera, 2011). Findings have also suggested that patients scoring high on hostility scales, showed higher rates of cardiovascular related events such as ischemia, greater damage by infarction, and a longer duration of ischemia during daily activities (Guerrero & Palmero, 2010; Shimbo, Chaplin, Kuruvilla, Wasson, Abraham, & Burg, 2009). High levels of anxiety and depressive symptoms, including sadness, have also been indicated as predictors of several cardiovascular events, especially hypertension and coronary heart disease (Kubzansky, Kawachi, Weiss, & Sparrow, 1998; Kubzansky, Cole, Kawachi, Vokonas, & Sparrow, 2006; Rutledge,

Linke, Krantz, Johnson, Bittner, Eastwood, Eteiba, Pepine, Vaccarino, Francis, Vido, & Merz, 2009; Suls & Bunde, 2005). Individuals experiencing negative emotions may be at a greater risk to develop negative cardiovascular events even after adjusting for age, education, smoking, BMI, blood pressure, cholesterol, and alcohol consumption (Haukkala, Konttinen, Uutela, Kawachi, & Laatikainen, 2009; Nabi, Kivimaki, De Vogil, Marmot, & Singh-Manoux, 2009; Schwartzman & Glaus, 2000; Taylor & Barr, 2010). Therefore stress and negative emotions may be significant predictors of negative cardiovascular events (Herrero, Gadea, Rodriguez-Alarcon, Espert, & Salvador, 2010; Mausbach, Rabinowitz, Patterson, Grant, & Schulz, 2007; Salomon, Clift, Karlsdottir, & Rottenberg, 2009; Smith, Uchino, Berg, Florsheim, Pearce, Hawkins, Henry, Beveridge, Skinner, Hopkins, & Yoon, 2008; von Kanel, Dimsdale, Adler, Patterson, Mills, & Grant, 2004).

Negative Emotions and Cardiovascular Disease (CVD)

Despite the lack of emphasis on psychological risk factors by major cardiology organizations and because traditional risk factors do not account for all of the variance for cardiovascular disease, there has been a growing body of research examining the relationship between mental health factors and adverse cardiovascular events (Joukamaa, Heliovaara, Knekt, Aromaa, Raitasalo, & Lehtinen, 2001; Joynt, Whellan, & O'Connor, 2003; Frasure-Smith & Lesperance, 2005; Maes, Ruckoanich, Chang, Mahanonda, & Berk, 2011; Patterson, Zakowski, Hall, Cohen, Wollman, & Baum, 1995; Smith, Uchino, Berg, Florsheim, Pearce, Hawkins, Henry, Beveridge, Skinner, & Hopkins, 2008; Tindle, Davis, & Kuller, 2010). Several studies have lent support to the positive relationship between particular psychological factors, such as negative emotions, and nonfatal and

fatal cardiovascular events (Ariyo, Haan, Tangen, Rutledge, Cushman, Dobs, & Furberg, 2000; Barefoot & Schroll, 1996; Frasure-Smith, & Lesperance, 2005; Lesperance, Frasure-Smith, Talajic, & Bourassa, 2002; Ibishi, Musliu, Kamberi, Qirko, Brojak, Lezha, Haxhiu, & Masala, 2009; Van der Kooy, van Hout, Marwijk, Marten, Stehouwer, & Beekman, 2007). Correlational studies have contributed to the examination of the association between depressive symptoms and cardiovascular pathologies. One study investigated the contributions of depression to the development of a CVD through the examination of healthy adult caregivers (Mausbach, Rabinowitz, Patterson, Grant, & Schulz, 2007). The researchers in this study followed a large group of caregivers, who fell within their late forties to early seventies, over an 18-month period. At baseline, participants completed multiple measures including a scale assessing depressive-like symptoms and an assessment to rule out any current medical or psychiatric diagnoses. Measures examining physical health, medication, depressive-like symptoms, and psychosocial variables were administered at the 6, 12, and 18th month mark following the baseline assessment. The findings indicate that greater depressive symptoms may be predictive of an earlier onset of a diagnosis of a cardiovascular disease such as heart disease, congestive heart failure, angina, and myocardial infarctions. Although the risk was relatively low during the 18 month follow-up, these results suggest that depressive symptoms may, “shorten the time it takes caregivers to develop a cardiovascular disease” (Mausbach, Rabinowitz, Patterson, Grant, & Schulz, 2007, p. 542). The researchers noted that one limitation of their study was the limited follow-up time span of 18 months. The researchers recommended that future studies include a more extensive follow-up period to account for the more typical period in which it takes a

cardiovascular problem to manifest. Other researchers interested in the onset and progression of CVD examined the relationship between negative emotions and future cardiovascular events in a group of healthy adults (Pollard & Schwartz, 2003). Participants completed four assessment periods also over an 18-month time span. Each assessment included the administration of an adjective checklist measuring the degree of various emotions including affective states such as anxious, jittery, dissatisfied, sad, relaxed, and cheerful. Participants also had their blood pressure taken twice during each of the four assessment periods. The researchers found that participants who endorsed a greater degree and number of negative emotions throughout the experiment, also exhibited greater elevations in blood pressure measurements over the four assessment periods when compared to participants who endorsed greater positive affective states. This portion of the sample did not only exhibit greater blood pressure responses but measurements within the hypertensive range. This relationship between negative emotions and blood pressure patterns suggests that negative emotions may play a role in the development of a negative cardiovascular event such as hypertension. Another set of researchers examined the association between negative emotions and CVD through a 15-year-study (Haukkala, Kontinen, Uutela, Kawachi, & Laatikainen, 2009). After an initial medical examination, participants completed a psychosocial questionnaire including questions on psychiatric and medical history, health behaviors, and an assessment of current depressive symptoms. The researchers followed up on the participants by checking the Causes of Death Register and the Hospital Discharge Register over a 15-year period. Participants who scored within the highest quartile of the depressive symptoms scale at baseline showed an increase risk for CVD nonfatal

negative events when compared to the participants who scored lower on depressive symptoms. These results suggest that depressive symptoms may predict new cardiovascular events even after adjustment for age, education, smoking, BMI, blood pressure, cholesterol, and alcohol consumption. Researchers have also examined negative affect as a predictor of coronary heart disease (Nabi, Kivimaki, De Vogil, Marmot, & Singh-Manoux, 2009). In a report using data from the Whitehall II study, these researchers evaluated participants at baseline on their endorsement of a multitude of negative emotions representative of negative affect. Researchers also assessed participant self-reports of cardiovascular outcomes twelve years later. The results showed that individuals that were categorized within the highest third percentile of negative affect had greater incidents of coronary heart disease over a twelve-year period than the participants who scored in the lower negative affect quartiles. In addition, this link remained significant after controlling for other common risk factors of coronary heart disease such as age, smoking status, and body mass index. These results lend support to the positive association between negative emotions and the development of certain cardiovascular diseases. Another study considered the effects of negative emotions on cardiovascular disease by examining the impact of negative affect through self-report and spousal report (Smith, Uchino, Berg, Florsheim, Pearce, Hawkins, Henry, Beveridge, Skinner, Hopkins, & Yoon, 2008). More specifically, researchers examined the associations of negative emotions such as anxiety, depressive symptoms, and anger with the development of coronary artery calcification (a component of coronary artery disease). The researchers recruited a large number of healthy middle-aged couples for the experimental session so that the participants' significant other would act as a collateral reporter. All participants

completed a baseline measure to assess each participant's endorsement of negative emotions. Researchers used this information to categorize participants into either a negative or positive emotion condition. Calcification was measured through the withdrawal of blood and two coronary artery scans on a multidetector-imaging scanner. The results showed that the participants with higher scores on scales measuring anxiety symptoms, depressive symptoms, and anger, were also found to have greater coronary artery calcification over time. Therefore there was a positive relationship between a set of negative emotions and a specific negative cardiovascular event. However, these associations were only significant when taking into account the collateral reports, but not self-reports. These affective states seem to provide some degree of risk for the development of a component of coronary artery disease. The researchers noted that the discrepancy in reporting between participant and partner were not surprising, in part, due to the power of social desirability. There are a number of correlational and longitudinal studies that have added support to the significant relationship between negative emotions and increased risk for the development of cardiovascular events.

Cardiovascular Reactivity

The variables that may shape the relationship between negative emotions and the development of cardiovascular events remain somewhat unclear. However, several factors have been hypothesized as mechanisms through which certain negative emotions may lead to or exacerbate cardiovascular events. One mechanism most relevant to the hypotheses may be cardiovascular reactivity (Gendolla & Krusken, 2002; Knox, Barnes, Kiefe, Lewis, Iribarren, Matthews, Wong, & Whooley, 2006; Neumann & Waldstein, 2001). Physiological reactivity refers to changes in endocrinological, neurobiological,

and other physiological parameters that occur following the introduction of an environmental stimulus (Kibler & Ma, 2004; Sharpley, 2002). It has been hypothesized that the autonomic nervous system, particularly as it shifts from parasympathetic to sympathetic dominance in response to a stimulus, is key in physiological adaptations to environmental stimuli (Dawson, Schell, & Catania, 1977; Porges, 2003). A subset of physiological reactivity, cardiovascular reactivity (CVR), is routinely used as a measure of autonomic activation resulting from environmental stimuli. Typically, paradigms using an analogue of mental or physical stimuli are used to measure an individual's ability to adapt successfully. Two parameters are chiefly used in this regard: measures of systemic resistance (e.g., blood pressure) and measures of cardiac output (e.g., heart rate). Cardiovascular reactivity is commonly evaluated by calculating the arithmetic difference between the average of each parameter above at baseline and the average during the presentation of stimuli. Cacioppo & Tassinari (1990) summarize the merits of using physiological data to understand psychological phenomena. These authors contend that in particular, physiological data can be used as indices of psychological states, such as stress. A physiological perspective can also afford psychology a deeper understanding of the physiological mechanisms involved in affective states. Because negative emotions and stress are potentially relevant to important health issues such as hypertension, and to mental health issues such as depression and anxiety, it is deserving of attention. The reactivity hypothesis maintains that over time the frequent and acute elevations in blood pressure (BP) and heart rate (HR) that are associated with negative emotions, stress, or other stimuli may lead to a multitude of changes or deleterious effects in the cardiovascular system (Allen, Bocek, & Burch, 2011; Knox, Hausdorff, & Markovitz,

2002; Cacioppo, 1998; Cacioppo, Berntson, Malarkey, Kiecolt-Glaser, Sheridan, Poehlmann, Burleson, Ernst, Hawkley, & Glaser, 1998; Katholi, McCreath, Whooley, Williams, Zhu, & Markovitz, 2004; Manuck, 1994; Matthews, Zhu, Tucker, & Whooley, 2006; Treiber, Kamarck, Schneiderman, Sheffield, Kapuku, & Taylor, 2003). For instance, sudden and repeated increases in the blood flow in the arteries, as a reaction to negative emotions or stressors, over time can create blood clots which may result in hypertension and other negative cardiovascular events (Cacioppo, Berntson, Malarkey, Kiecolt-Glaser, Sheridan, Poehlmann, Burleson, Ernst, Hawkley, & Glaser, 1998; Cacioppo, 1998). The mechanism through which negative emotions may increase risk for cardiovascular events may be physiological (Brinkmann & Gendolla, 2007; Joynt, Whellan, & O'Connor, 2003; Levin & Linden, 2008; Schwartzman & Glaus, 2000). Cardiovascular reactivity (CVR) may be one underlying mechanism explaining the relationship between specific psychological factors and negative cardiovascular events (Glynn, L'Italien, Sesso, Jackson, & Byring, 2002; Treiber, Kamarck, Schneiderman, Sheffield, Kapuku, & Taylor, 2003; Uchino, Holt-Lunstad, Bloor, & Campo, 2005). Therefore negative emotions may increase the risk to develop a cardiovascular event through a mechanism involving a cascade of psychological and physiological variations in variables including cardiovascular reactivity. (Brinkmann & Gendolla, 2007; Joynt, Whellan, & O'Connor, 2003; Levin & Linden, 2008; Schwartzman & Glaus, 2000).

Negative Emotions and Cardiovascular Reactivity

The role of CVR, in the relationship between negative emotions and cardiovascular events, has been examined in the laboratory using a wide array of experimental paradigms including the presentation of an emotion-inducing stimulus,

labeled a mood induction, to participants in order to elicit negative emotions while assessing blood pressure and heart rate (Brinkmann & Gendolla, 2007; Joynt, Whellan, & O'Connor, 2003; Levin & Linden, 2008; Kreibig, Wilhelm, Roth, & Gross, 2007; Schwartzman & Glaus, 2000). Several of these experimental paradigms have resulted in evidence suggesting that eliciting negative emotions in a laboratory setting significantly elevates blood pressure and heart rate when compared to baseline measurements (Hamer, Tanaka, Okamura, Tsuda, & Steptoe, 2007; Kreibig, Wilhelm, Roth, & Gross, 2007; Lepore, Revenson, Weinberger, Weston, Frisina, Robertson, Portillo, Jobes, and Cross, 2006; Prkachin, Williams-Avery, Zwaal, & Mills, 1999; Thornton & Hallas, 1999). For example, researchers have used emotional recall tasks to examine relations between eliciting a negative emotion and patterns of cardiovascular responses (Prkachin, Williams-Avery, Zwaal, & Mills, 1999). Researchers asked participants to recall and write down a particularly emotional incident that fell into one of five categories: happiness, anger, fear, sadness, and disgust. The emotional recall task served as both a mood induction and a stressor task. In addition, the researchers simplified these emotional states by classifying each as either a positive or a negative emotional state. Blood pressure and skin temperature counts were taken of each participant at baseline and during the task. At the end of the session, researchers gave participants a questionnaire to measure the effectiveness of the mood induction and the results showed significant manipulation effects of each emotion. The results also showed that blood pressure measurements were elevated for all emotional states when compared to baseline but were significantly higher during the negative conditions than for the positive conditions, in particular, within the sadness condition. Of note, participants in the positive

and negative mood conditions did not significantly differ in cardiovascular responses at baseline. These researchers demonstrated the strong possibility that, “negative emotional states like anger, fear, and sadness are accompanied by a sufficient beta-adrenergic drive to raise blood pressure to levels that exceed those attained during happiness” (Prkachin, Williams-Avery, Zwaal, & Mills, 1999, p. 264). This study lends support to the hypotheses that incorporate increased patterns of cardiovascular reactivity in the association between negative emotions and cardiovascular disease. Another study also examined the relationship between the elicitation of negative emotions and cardiovascular reactivity by using an emotional recall task (Deichert, Flack, & Craig, 2005). Healthy participants completed a feelings rating form to indicate the degree to which each felt anger, sadness, happiness, disgust, and contempt. Following this form and baseline measurements of blood pressure and heart rate, participants were asked to perform a recall task which included imagining an angry, happy and sad experience and then writing about it as a performance task. At the end of the experimental session, participants completed the same feelings rating form given prior to the tasks. The results showed that blood pressure and heart rate measurements were significantly higher when collapsing the data across the entire recall and writing tasks when compared to baseline. In addition, the results showed that participants within the anger mood induction exhibited the greatest increase in blood pressure responses when compared to the happy and sad induction conditions. Another set of researchers investigated the link between specific emotions and cardiovascular responses through a mood induction asking healthy participants to recall a happy or sad emotional experience while multiple cardiovascular measures were taken in the lab (Gendolla & Krusken, 2001). The researchers found that

the participants exhibited varying blood pressure and heart rate responses dependent on the valence of the stimulus. The participants in the sad condition did not exhibit significant increases in their blood pressure and heart rate responses, however, the participants in the happy condition did exhibit significant increases in both parameters. Neumann & Waldstein (2001) found that a negative mood induction, comprised of an emotion eliciting verbal recall stressor tasks, intended to elicit sadness or anger, was effective in increasing the intended emotions and resulted in significant increases in blood pressure. In addition, participants who completed the task that evoked sadness exhibited higher increases in blood pressure than participants who completed the task that evoked anger. Researchers have examined changes in affective states and corresponding cardiovascular reactivity using emotion-inducing speech tasks (Hamer, Tanaka, Okamura, Tsuda, & Steptoe, 2007). This study examined the effects of negative emotions on cardiovascular reactivity by using speech tasks intended to induce depressed and angry moods and act as a mental challenge. A healthy group of participants were administered scales at baseline and after the emotion specific speech tasks assessing depressive symptoms and anger. Researchers also measured blood pressure and heart rate measurements at baseline, during the tasks, and following each task. Participants completed one speech task intended to induce a depressed emotional state and then one speech task intended to induce an angry emotional state. The order of completion of each speech task was counterbalanced for participants and there was a thirty-minute recovery period between each emotion inducing speech task. The findings showed that the depressive speech task induced significantly high self-reported rates of depression and the anger inducing speech induced significantly high self-reported anger. In regards to

depressive symptoms, the results showed that participants who endorsed greater depressive symptoms prior to the emotion-specific stressor tasks exhibited higher blood pressure and heart rate responses from baseline than the participants who scored lower on depressive symptoms throughout the entire experimental procedure. Researchers have also examined changes in affective states and corresponding cardiovascular reactivity using emotion-inducing films. Another study presented participants with mood inductions consisting of film clips intended to elicit either happiness or anger (Waldstein, Kop, Schmidt, Haufler, Krantz, & Fox, 2000). The healthy adult participants completed this portion of the experimental session while having their blood pressure and heart rate responses continually measured. The results showed that participants in both emotion-specific conditions exhibited increases in the intended emotion and similar cardiovascular reactivity. Despite the significant increase in blood pressure and heart rate responses from baseline, the magnitude of these elevations was extremely low fluctuating from .8 to 4 points. These films did not seem to have the ability to induce a degree of arousal of clinical significance.

Another set of researchers examined the association between negative emotions and CVR in healthy participants by using mood inductions including film clips intended to induce fear or sadness (Kreibig, Wilhelm, Roth, & Gross, 2007). Participants were randomly placed in either condition and completed a self-report adjective likert scale prior to and following the showing of the film indicating the strength of their emotion while having their blood pressure measured. Through the use of this emotion intensity scale the results showed that the elicitation of each emotion was effective in inducing greater sadness or fear from baseline responses in participants within each condition. In addition,

participants in all conditions exhibited significant increases in blood pressure and heart rate responses and the participants in the sadness and fear conditions exhibited similar heightened cardiovascular reactivity. Of note, there were no significant differences in baseline blood pressure and heart rate measurements between conditions.

Several studies also provide null findings suggesting that negative mood inductions or the elicitation of negative emotions are not associated with significant increases in blood pressure and heart rate despite self-reported increases in negative emotions following the mood induction. (Allen, Bocek, & Burch, 2011; Carroll, Phillips, Hunt, & Der, 2007; Hilmert & Kvasnicka, 2010; Gendolla & Krusken, 2001) Many of these studies include an emotion-inducing stimulus (read a passage, listen to music, watch a film clip, or visually recall an experience) and have resulted in participants exhibiting no significant change in blood pressure or heart rate responses from baseline (Codispoti, Surcinelli, & Baldaro, 2008; Deichert, Flack, & Craig, 2005; Etzel, Johnsen, Dickerson, Tranel, & Adolphs, 2006; Nater, Abbruzzese, Krebs, & Ehlert, 2006).

One set of researchers examined the relationship between negative emotions and cardiovascular responses with the use of a mood induction comprised of several pieces of music (Etzel, Johnsen, Dickerson, Tranel, & Adolphs, 2006). The music was used to induce various emotions including sadness and fear while measuring blood pressure and heart rate responses. Despite participants' self-reported increases in their feelings of sadness or fear, the mood induction was not positively associated with heart rate reactivity. In addition, heart rate responses between the two negative mood inductions did not significantly differ. These findings seem to contradict the way in which physiological mechanisms may correlate the elicitation of negative emotions with

cardiovascular events. Carroll, Phillips, Hunt, & Der (2007) examined the association between depressive emotions and cardiovascular reactions with the use of a stressor task. Participants completed a scale assessing depressive symptoms and had their blood pressure and heart rate measured at baseline and during an auditory serial arithmetic test. The findings showed that depressive scores were not positively associated with blood pressure and heart rate reactions therefore participants scoring higher on the depressive symptoms scale did not exhibit significant increases in blood pressure or heart rate. This suggests that excessive reactivity may not be a pattern distinctive of negative emotions. The examination of mood effects on cardiovascular responses continued with another study that included a mood induction comprised of a film clip (Silvestrini & Gendolla, 2007). A group of participants were emotionally induced either into a negative, neutral or positive state with the use of a film clip. Blood pressure and heart rate responses were taken throughout the experimental session. Participants also completed a self-report measure of mood prior to the mood induction and at the end of the experiment. Again, despite effective mood manipulations, the analyses showed there was no blood pressure or heart rate reactivity during the mood inductions in all conditions where, “reactivity in all cells was close to zero” (Silviestrini & Gendolla, 2007, p. 657). These findings contradict emotion-specific cardiovascular responses and any exaggerated cardiovascular responses to increases in negative emotions since the elicitation of a negative emotion did not significantly elevate blood pressure or heart rate responses. Another set of researchers also evaluated the relationship between negative emotions and cardiovascular reactivity with the use of a music based mood induction (Allen, Bocek, & Burch, 2011). Participants were presented with two pieces

of music previously shown to elicit sadness and gloominess and reduce feelings of happiness. Participants completed an adjective checklist to test the effectiveness of the mood induction and had blood pressure and heart rate measurements taken throughout the entire experiment. The results showed no significant association between the negative music mood induction and cardiovascular reactivity. This finding seems to contradict the potential negative outcomes that have been hypothesized to result from the elicitation of negative emotions and may ultimately lead to diminished cardiovascular functioning.

Limitations in Literature

Although the above area of research has greatly assisted in the exploration of the effects of mood inductions on vascular responses, the existing literature also has some inconsistencies. For example, some studies have suggested that the induction of negative emotions significantly increases blood pressure and heart rate while other studies have indicated no significant association between eliciting negative emotions and blood pressure and heart rate responses. This inconsistency in data may stem from a myriad of variables including the composition of the mood inductions (Gerrards-Hesse, Spies, & Hesse, 1994). Many of the studies that suggest a positive association contain mood inductions consisting of the presentation of a negative emotion-inducing stimulus (film clip, piece of music, written passage) or a negative emotion-specific stressor task (recall or speech task modified to elicit a specific emotion). Whereas many of the studies suggesting that there is no association between negative emotions and vascular responses mainly consist of mood inductions comprised solely of the presentation of a negative emotion-inducing stimulus (film clip, listening to a piece of music, or reading a written

passage). In addition to the inconsistency in the composition of mood inductions, there is a distinction in the mean increases in blood pressure and heart rate responses, within the studies showing support for a positive relationship between negative emotions and vascular responses. When further examining the results within the studies suggesting a positive relationship, the experiments that included mood inductions comprised of a negative emotion-specific stressor task (recall or speech task modified to elicit a specific emotion) or a negative emotion-specific stressor task and negative emotion-inducing stimulus (film clip, piece of music, written passage) reported approximately three to five times greater increases in blood pressure and heart rate responses than the studies with mood inductions comprised of solely a negative emotion-inducing stimulus. The same studies that included a negative emotion-specific stressor task as their mood induction and suggested a positive association between the elicitation of negative emotions and vascular responses, also seemed to fail in differentiating the effects of the negative valence of the stimuli versus the stimuli itself on vascular measures. The elevations in blood pressure and heart may have had less to do with the negative emotion and more to do with the potential stress induced by a stressor task such as recall task. The task itself may exert additional direct physiological influences. The elevations in blood pressure and heart rate in one set of studies may have had more to do with the arousal induced by the event itself irrespective of its negative valence. Cacioppo & Tassinary (1990) summarize the merits of using physiological data to understand psychological phenomena. These authors contend that in particular, physiological data can be used as indices of psychological states, such as stress. A physiological perspective can also afford psychology a deeper understanding of the physiological mechanisms involved in

affective states. Because negative emotions and stress are potentially relevant to important health issues such as hypertension, and to mental health issues such as depression and anxiety, it is deserving of attention. Finally, although this research has been accumulating over the past twenty years, many unanswered questions remain, and more systematic inquiry is needed to provide crucial progress in understanding these complex constructs. Therefore the mechanism of change is less clear and it is difficult to determine whether the valence of the induction or the task itself provides greater influence, if any, on blood pressure and heart rate responses. This is a critical point given the literature suggesting the many ways in which stress may affect cardiovascular health, in particular, through increases in blood pressure and heart when an individual is engaged in a standardized non-specific stressor task (Joynt, Whellan, & O'Connor, 2003).

A long-standing consensus among mainstream researchers holds that stress affects physiological responses, and most relevant to the present examination, that individuals exhibit significant increases in blood pressure and heart rate responses when completing certain standardized stressor tasks (Kelsey, Ornduff, & Alpert, 2007; Linden, Gerin, & Davidson, 2003; Nealey-Moore, Smith, Uchino, Hawkins, & Olson-Cerny, 2007; Treiber, Kamarck, Schneiderman, Sheffield, Kapuku, & Taylor, 2003). As previously cited, the reactivity hypothesis posits that each of these exaggerated responses (increases in blood pressure and heart rate) to a stressor event may have a pathogenic effect (Cacioppo, Berntson, Malarkey, Kiecolt-Glaser, Sheridan, Poehlmann, Bursleson, Ernst, Hawkey, & Glaser, 1998; Mausbach, Rabinowitz, Patterson, Grant, & Schulz, 2007; Ming, Adler, Kessler, Fogg, Matthews, Herd, & Rose, 2004). Studies examining the association between stress, cardiovascular reactivity, and future cardiovascular events have

demonstrated that individuals who exhibited higher levels of blood pressure and heart rate responses to a stressor were also 2.4 times more likely to experience or die of coronary heart disease-related causes when compared to participants who had lower reactivity results over multiple decades (Keys, Taylor, Blackburn, Brozek, Anderson, & Simonson, 1971). Furthermore, after controlling for factors such as age, obesity, cigarette smoking, alcohol consumption, and parental history, studies have demonstrated that cardiovascular reactivity during certain stressor tasks is predictive of cardiovascular events, such as hypertension and coronary calcification several years later (Knox, Hausdorff, & Markovitz, 2002; Matthews, Katholi, McCreath, Whooley, Willaims, Zhu, & Markovitz, 2004; Matthews, Zhu, Tucker, & Whooley, 2006). Matthews, Woodall, and Allen (1993) measured the cardiovascular reactivity of male and female adults to two psychological stressors (serial subtraction and mirror-image tracing). The results demonstrated that at follow up (6.5 years after initial measurements), blood pressure and heart rate numbers were higher for the men and women who had higher blood pressure counts during the stressor tasks. Once again, this association remained after controlling for age, obesity, and resting blood pressure at the start of the initial session. Researchers have also examined the role of cardiovascular reactivity in the prediction of future blood pressure levels by observing the effects of work stress on blood pressure in a sample of 218 white males (Ming, Adler, Kessler, Fogg, Matthews, Herd, & Rose, 2004). Researchers assessed blood pressure reactivity on 15 different occasions each day for five days over a four-year period while participants were performing their day-to-day work tasks. There was a follow up period two decades after the initial measurements where researchers consulted past participants to assess if they were in the range for

hypertension. The findings demonstrated that individuals, who exhibited the highest increases in systolic blood pressure between the years of 1974 and 1978, were also 3.5 times more likely to report incidents of hypertension twenty years later than individuals who had exhibited lower systolic responses. Cardiovascular reactivity to work stress may be a long-term predictor of future blood pressure levels. A closer examination of the CARDIA study, an ongoing prospective study of the natural history of cardiovascular risk development in young adulthood, tested cardiovascular reactivity to psychological stress as a predictor of hypertension in men and women, ages 18-30 yrs at entry of the experiment (Matthews, Katholi, McCreath, Whooley, Williams, Zhu, & Markovitz, 2004). Researchers measured the participant's blood pressure counts at baseline and during the administration of three different psychological tasks. The researchers found that thirteen years after the initial blood pressure assessments and after adjusting for race, gender, education, body mass index, age, and resting blood pressure, the larger the increases in blood pressure during each of the three tasks, the earlier participants reported an incident of hypertension. An incident of hypertension operationalized as taking hypertensive medication prescribed by a doctor or generating one blood pressure measurement at or above 140mg/90mg. Cardiovascular reactivity may also play a role in the prediction of the onset of other cardiovascular problems, such as coronary calcification. The CARDIA study was examined again to test whether blood pressure changes during psychological stress in a laboratory setting predicted future cardiovascular problems, specifically coronary calcification (CaC) in young healthy adults (Matthews, Zhu, Tucker, & Whooley, 2006). The researchers found that the participants who had the greatest increases in blood pressure and heart rate during one of

the stressor tasks (video game) at the three different assessment intervals, were more likely to have been diagnosed with coronary calcification thirteen years later. This association persisted after adjustment for interim hypertension. These studies lend support to the idea of stress playing a role in the directionality of the relationship between negative emotions and blood pressure and heart rate responses. This also suggests the importance of examining the possibility of discrete stressor task effects on blood pressure and heart rate when included as part of a mood induction. Stressor tasks may induce the same degree or greater arousal than the elicitation of a specific negative emotion (Moseley & Linden, 2006). Therefore it may also be informative to include another measure to assess factors, such as perceived stress, that may be introduced during a mood induction task and overcome other variable effects on cardiovascular responses. Stressors in the laboratory contribute to elevations in blood pressure and heart rate and while focusing on the physiological effects has been informative, laboratory research, has had the tendency to exclude assessments of perceived stress to correspond with the physiological measures. Defining stress in terms of physiological reactivity and through the extent to which an individual perceives the event as stressful allows one to create a more complete assessment of the effects of stress on cardiovascular reactivity. Therefore, the inclusion of the measurement of stress-related changes (blood pressure and heart rate responses) along with the inclusion of the measurement of the experience of stress (perceived state of stress) may provide improved understanding of the corresponding blood pressure responses and an overall more accurate interpretation of data that can provide additional credence to sample data.

Stress responses are complex and incorporate multiple components including gender. However, the effects of gender differences on stress induced blood pressure and heart rate responses are mixed and a clear pattern of differences has yet to be determined (Dedovic, Engert, Pruessner, & Wadiwalla, 2009; Fox, Garcia, Kemp, Milivojec, Kreek, & Sinha, 2006; Kolk & van Well, 2007; Rausch & Auerbach, 2008; Whited & Larkin, 2009). Many studies have shown that men tend to exhibit greater increases in blood pressure responses than do women when exposed to a stressor task in the laboratory (Allen & Matthews, 1997; Davis, & Matthews, 1987; Lash, Gillespie, Eisler, & Southard, 1991; Lawler, Wilcox, & Anderson, 1995; Light, Turner, Hinderliter, & Sherwood, 1993; Yeh, Huang, Chou, & Wan, 2009). Lepore, Ragan, and Jones (2000) had participants watch a film clip about the Holocaust and then generate a speech on the footage. There were significant blood pressure and heart rate differences between male and female participants, with males exhibiting greater reactivity than females. Allen, Stoney, Owens, & Matthews (1993) had participant's complete three different psychological tasks including a mirror tracing, stroop, and mental arithmetic task. The male participants exhibited significantly greater blood pressure than female participants in response to the subset of tasks. However, this is not always the case and at times females have shown to be equally physiologically reactive to males with blood pressure responses and demonstrate greater heart rate reactivity than men (Matthews & Stoney, 1988; Nater, Abbruzzese, Krebs, & Ehlert, 2006; Newton, 2009; Stoney, Matthews, McDonald, Johnson, 1988; Yeh, Huang, Chou, & Wan, 2009). One study examined the pattern of sex differences in cardiovascular responses to stress by providing social tasks to participants (Whited & Larkin, 2009). Researchers randomly assigned participants to either a conflict

or a comfort interaction with a confederate. The social tasks asked that the participants engage the confederate in a conversation to either persuade him/her to do something (conflict) or show understanding (comfort). Blood pressure measurements were continuously taken from baseline to the end of the session. The results showed blood pressure and heart rate reactivity in all conditions but no sex differences in reactivity. Therefore the potential for gender to moderate cardiovascular reactivity was also a point of interest in the present study and examined in exploratory analysis given previously identified gender differences in cardiovascular activation in response to stress reactivity (Pollard & Schwartz, 2003; Traustadottir, Bosch, & Matt, 2003).

Present Study

Prior studies within the mood induction literature have provided important findings regarding the relationship between negative emotions and cardiovascular reactivity. However, there is still a need to further examine several basic but key components in this area of research. For instance, different experimental methods have been used to induce emotions, in particular, studies have used a single manipulation comprised of the presentation of an emotion-inducing stimulus or a manipulation comprised of an emotion-specific stressor task or both an emotion-inducing stimulus and an emotion-specific stressor task. In addition, the studies that suggested a positive association seem to allocate little attention to the possible distinction between the effects of the emotion-specific portion of the manipulation and the task itself, especially the stressor tasks, on blood pressure and heart rate. These studies seem to neglect the possible effects of the stressor tasks on blood pressure and heart rate and on the perception of stress. These limitations make it difficult to implicate one construct as the primary

underlying force increasing blood pressure and heart rate and may overlook the magnitude of the effects of stress on cardiovascular reactivity. There were two neutral mood induction conditions as well. In the neutral conditions, participants either watched a standardized neutral film clip or watched the same standardized neutral film clip followed by the non-specific stressor tasks. The neutral conditions were included with the intention of further controlling the experimental paradigm and supporting the idea that the elicitation of a single negative emotion, through the stimulus of a sad film clip, by itself, is not responsible for the significant elevations in blood pressure and heart rate responses. Therefore, the negative valence of the mood induction would not predict cardiovascular reactivity. In addition, there was an examination of the magnitude of the effect of stressor tasks on cardiovascular reactivity when combined with the presentation of an emotion-inducing stimulus (film clip) within a mood induction. Such findings were intended to highlight the relative contributions of negative mood states and stress to cardiovascular reactivity, and thus potential long term risk for negative cardiovascular events. In brief, the main objective in conducting the current research was to examine the contributions of a negative emotion (sadness) and neutral stressor tasks on blood pressure and heart responses within one experimental session.

Such findings may help in highlighting the relative contributions of negative mood states and stress to cardiovascular reactivity, and thus risk for cardiovascular disease.

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