# The Relationship between Depression and Cardiovascular Reactions to Acute Psychological Stress

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**Abstract**

The co-morbidity of depression and cardiovascular disease has been well documented yet the etiological root and mechanism of the relationship has remained unsubstantiated. The present study sought to characterize the cardiovascular response to acute psychological stress in a young healthy population using an orthostatic challenge and paced auditory serial addition task. High frequency heart rate variability (HRV) and blood pressure was monitored during the stress tasks and reactivity and recovery measures were derived. Self reported depressive symptomatology, collected using the Beck Depression Inventory (BDI) was indirectly related to systolic blood pressure and heart rate reactivity. BDI was also related to anticipatory threat appraisals such that higher BDI scores conferred greater threat appraisal. HRV measures were unrelated to BDI scores and neither threat appraisal nor HRV mediated the relationships between BDI score and cardiovascular reactivity. These results accord with the notion that depression is associated with a blunted stress response. Further research should aim to further characterize the mechanism linking depression with blunted reactivity so that this biomarker of cardiovascular dysfunction may be utilized in a clinical setting.

**Abbreviations:**
PASAT- Paced Auditory Serial Addition Task
BDI- Beck Depression Inventory
BMI- Body Mass Index
HF HRV- High Frequency Heart Rate Variability
SBP- Systolic Blood Pressure
DBP- Diastolic Blood Pressure
MAP- Mean Arterial Pressure

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The Relationship between Depression and Cardiovascular Reactions to Acute Psychological Stress

## Introduction

### Mechanistic Links between Depression and Cardiovascular Disease

The relationship between depression and cardiovascular disease has received extensive attention across a wide spectrum of research disciplines. Reviews of clinical research have suggested that elevated depressive symptomatology is predictive of cardiovascular disease (Rugulies, 2002; Koponen et al., 2010; Wulsin & Singal, 2003) while other studies have documented an increase in mortality among cardiovascular disease patients who develop depression (Barth et al., 2004; Bush et al., 2001; Lesperance et al., 2002) suggesting a bi-directional relationship between depression and cardiovascular disease.

Studies aimed at elucidating the mechanism responsible for the co-morbidity of depression and cardiovascular disease have yielded a number of theories, often reflective of the research field positing the theory. A decrease in heart rate variability, observed as a signal of nervous system imbalance, has been observed in depressed patients (Carney et al., 2001; Rechlin et al., 1994) leading autonomic nervous system researchers to suggest autonomic imbalance in the form of high sympathetic activity or low parasympathetic activity as the common etiological root of the depression and cardiovascular disease. In a similar manner, psychoneuroendocrinologists have posited that a hyperactive hypothalamic-pituitary-adrenal axis (HPA-axis) is responsible for linking depression and cardiovascular disease. This conjecture is supported by Burke and colleagues (2005) who found depression conferred a differential cortisol response to social stress characterized by sustained elevated levels of cortisol following conclusion of acute stress tasks. Finally, cardiovascular reactivity to acute psychological stress has garnered much support as a intervening factor between depression and cardiovascular disease since research has shown that cardiovascular reactivity is predictive of depression five years later

(Phillips et al., 2011a). More than this, individuals with depression exhibited blunted blood pressure reactivity to acute psychological stressors when compared to non-depressed controls (York et al., 2007; Phillips, 2010; Carroll et al., 2007). Further, a small study conducted by Brindle and colleagues (2011) observed blunted blood pressure recovery in young, cardiovascular disease free individuals scoring high on the Beck Depression Inventory.

Although distinct from each of other, each mechanistic proposition can be envisioned as a minor contributor to the whole phenotype exhibited by an individual with depression and cardiovascular disease. It may be that an underlying vagal dysfunction associated with depression leads to decreased heart rate variability and blunted blood pressure reactivity while high levels of cortisol following psychological stressors inhibit the natural blood pressure recovery mechanisms leading to hypertension. However, assessing these relationships is beyond the scope of the current investigation. The current examination aimed to evaluate cardiovascular reactions of blood pressure and heart rate variability as a function of depressive symptomatology in an effort to further understand the relationship between depression and cardiovascular function in generally healthy young adults.

**Cardiovascular Reactivity**

The cardiovascular reactivity hypothesis posits that exaggerated blood pressure and heart rate responses to acute psychological stress confer adverse cardiovascular outcomes (Obrist, 1981). Since its advent, this paradigm has equipped researchers with a simple method for assessing and predicting cardiovascular function and, as a result, has been heavily utilized. Research subscribing to this paradigm has demonstrated that exaggerated blood pressure responses to acute psychological stress predict increased carotid intima-media thickening (Jennings et al., 2004) and hypertension (Borghi et al., 1986). However, recent research has begun to show that this is only part of the story and that cardiovascular recovery from acute psychological stress and blunted cardiovascular reactivity need to be included in the reactivity hypothesis framework (Phillips, 2011b). Blunted cardiovascular reactivity has been linked

with obesity, poor self-reported health status, and poor immune function (Phillips, 2010; Phillips et al., 2009). Similarly, impaired blood pressure recovery from acute psychological stress is predictive of carotid intima-media thickening (Steptoe et al., 2006a), myocardial infarction (Laukkanen et al., 2004), and longitudinal changes in blood pressure (Stewart and France, 2001; Steptoe and Marmot, 2005).

Lovallo (2003) posits that cardiovascular reactions to acute psychological stress are the result of an interaction between three levels of regulation. At the *cognitive-emotional level* frontal lobe structures, in conjunction with the amygdala, appraise situations and assess the degree of threat present in the stressor. The *hypothalamic level* includes structures responsible for maintaining and moderating cardiovascular function. These structures react to the outputs of the higher executive brain regions and modulate the cardiovascular and respiratory systems accordingly. Finally, in the *peripheral tissues*, outputs from the hypothalamic level are executed, resulting in a response. Lovallo (2005) maintains that variability at any of these three levels interacts to form an individual cardiovascular response. For example, differential appraisal at the cognitive-emotional level, variable activation of brainstem regions, and local tissue regulation are all sources of variability that contribute to a complete reaction.

The present study aimed to utilize the framework provided by Lovallo (2003) to explore the mechanisms responsible for the differential cardiovascular reactions observed in participants exhibiting depressive symptomatology. Two of the levels, cognitive-emotional and hypothalamic, were assessed in the current examination.

**Stress Appraisal, Depression, and Cardiovascular Reactivity**

Induced acute psychological stress has been utilized by researchers to probe the intricacies of the human stress response. To date, many stress paradigms exist that require participants to perform a range of stressful behaviors. Examples include public speaking in the Trier Social Stress Test (Kirschbaum et al., 1993), mental arithmetic in the Paced Auditory Serial Addition Task (Carroll et. al., 2007), and

color determination in the Stroop Color-Word Interference Task (Gianoros et al., 2005). According to the stress and coping model proposed by Lazarus and Folkman (1984), appraisal of a situation is critical for eliciting an appropriate physiological response. Further, the appraisal process occurs in two phases: primary and secondary. At the primary level, stressful events can be appraised as harm/loss, threat, or challenge; each conferring a different reaction to the stressor. Harm/loss and threat appraisals signify a stressor that has already or has the potential to cause harm or loss to a person respectively. Challenge appraisals, on the other hand, are associated with stressors that afford opportunities for potential growth or advancement of the individual. Secondary appraisal concerns ability of one to cope with a stressor and it is the interaction between primary and secondary appraisals that shape the physiological response to a stressor.

Research relating stress appraisal to cardiovascular responses to stress has shown that differential appraisals give rise to a spectrum of reaction phenotypes. A study conducted by Tomaka and colleagues (1993) showed that appraisals of threat or challenge were accompanied by distinct cardiovascular reactions. Challenge appraisals were accompanied by an increase in blood pressure and decrease in vascular reactivity while threat appraisals conferred an opposite reaction. As a follow up, the same research group (Tomaka et al., 1997) showed that differential modulation of pre-ejection period, total peripheral resistance, and cardiac output can be achieved by manipulating instruction sets to elicit challenge or threat appraisals in participants providing more evidence for the intimate relationship between stress appraisal and cardiovascular reactivity.

Still others suggest anticipatory appraisal as a more powerful predictor of subsequent stress evoked physiological responses and that the interaction between cognitive appraisal and physiological reactions is best understood from this perspective (Feldman et al., 2004). To date, no study has examined the direct relationship between cardiovascular reactions to acute psychological stress and anticipatory appraisal. However, research has suggested that anticipatory appraisal influences

neuroendocrine responses to stress (Wirtz et al., 2007). Studies utilizing the Primary Appraisal Secondary Appraisal (PASA) scale showed that high stress index scores, representative of high levels of anticipated stress on the part of the participant, were associated with higher secretion of TNF-α (Wirtz et al., 2007) and increased blood coagulation (Wirtz et al., 2006), in response to psychological stress. Further, primary appraisal seems to explain much of the variance observed in cortisol responses to the Trier Social Stress Task (Gaab et al., 2005).

A paucity of literature exists examining stress appraisal and its concomitant physiological response in depressed individuals. A single study conducted by Meyer and colleagues (1989) found that children with depression appraised stressful events with more negative emotion when compared to non depressed controls. Further, negative emotion has been observed to coincide with threat appraisals (Feldman et al., 2004) which have been characterized by decreases in cardiovascular reactivity to psychological stress.

**Orthostatic Challenge, Depression, and Cardiovascular Reactivity**

Orthostatic challenge is a simple test requiring participants to stand while the homeostatic response to hypotension is recorded. Given that this action entails little physical or psychological strain, it has been taken by researchers to represent a purely physiological response (Orr et al., 1998) and has been utilized to assess individual differences is homeostatic regulation.

Studies examining the relationship between depression and orthostatic response have shown that depression is associated with significantly higher heart rate increases (Carney et al., 1999) and norepinephrine secretions (Musselman et al., 1998). Similarly, Stress and colleagues (2003) observed an impaired blood pressure response to orthostatic challenge in depressed children.

**Heart Rate Variability (HRV) and Depression**

Heart rate variability is the beat-to-beat fluctuations in heart period. These fluctuations are autonomic in origin and therefore heart rate variability provides a reliable index of autonomic nervous

system function. Work by Mezzacapa and colleagues (2001) showed that under laboratory conditions vagal tone, indexed by high frequency (HF) oscillations in heart rate (0.15-0.4 Hz), significantly fluctuated in response to physical and psychological stressors. Specifically, an acute vagal withdrawal was observed in response to a mental arithmetic psychological stressor while a compensatory increase in vagal tone occurred upon completion of the task during recovery.

In relation to depression, a meta-analysis conducted by Rottenberg (2007) found that depression was associated with a significant decrease in heart rate variability in physically healthy and cardiovascularly compromised individuals. Studies examining 24-hour heart rate variability observed significant decreases in healthy depressed individuals (Rechlin et al., 1994), coronary heart disease patients (Stein et al., 2000), and depressed patients recovering from myocardial infarction (Carney et al., 2001). This finding was extended by research in the Netherlands Study of Depression and Anxiety (Licht et al., 2008) that observed a decrease in HF variation across a period of 1.5 hours in depressed individuals. However, tricyclics and serotonin selective reuptake inhibitors were found to heavily influence heart rate variability in this sample.

In an effort to more fully understand the relationship between depression and heart rate variability studies have explored the change in HF variation following depression treatment. Carney and colleagues (2000) observed an increase in daytime heart rate variability in depressed individuals after 16 sessions of cognitive behavioral therapy. This finding also held true for non-pharmacological therapies such as acupuncture (Chambers & Allen, 2002). Further HF reactivity to a sad film clip was shown to predict recovery from depression independent of medication use (Rottenberg et al., 2005). The observation that depression and heart rate variability respond favorably to treatment suggests heart rate variability and depression have a common etiological root or are causally related.

Measures of heart rate variability have also been applied to acute stress paradigms. Generally, depression has been characterized by significantly greater vagal withdrawal, indexed by changes in HF

variation, during acute stress (Sheffield et al., 1998; Hughes & Stoney, 2000). Contrary to these findings are studies by Shinba (2008) and Gentzler (2009) who found that depression was associated with significantly less vagal withdrawal in adults and children respectively. Still others (Rottenberg et al., 2007a; Rottenberg et al., 2003) have oberserved vagal activation in response to acute psychological stress and crying. It has been suggested that the reason for such inconsistencies are rooted in variability in depression metric, stress paradigm, and heart rate indices. The Paced Auditory Serial Addition Task (PASAT) was utilized in the current investigation because it has been shown to reliably perturb the cardiovascular system (Carroll et al., 2005, 2007; Ring et al., 2002).

Few studies have examined vagal rebound in depressed individuals. Two studies conducted by Rottenberg (2005, 2007a) utilizing two different stress paradigms found depressed individuals characteristically failed to demonstrate a compensatory vagal activation following conclusion of stressors. It has been suggested that this may manifest in dysfunction of cardiovascular homeostatic mechanism. Given the dearth of research examining vagal recovery this study aimed to characterize vagal recovery in individuals scoring high and low on a common self-report measure of depression.

Overall, the purpose of the present study was to characterize the relationship between cardiovascular reactions to stress and depression and elucidate the mechanisms behind such a relationship. First, it was hypothesized that increases in depression scores, as measured by the Beck Depression Inventory (BDI) would confer reductions in cardiovascular reactivity and recovery from physical and social stress. Mediating this relationship it was hypothesized that increases in BDI scores would be associated with decreases in vagal withdrawal in response to a stressor, decreases in vagal rebound following conclusion of a stressor, and increases in anticipatory threat appraisals of induced stressors.

## Methods

### Participants

Participants (N=120) were recruited from undergraduate classes. At the discretion of course instructors, participants were awarded extra credit for their participation. During the first phase of the study questionnaire data was collected from all recruited participants (n=120). In an effort to ensure an even distribution of Beck Depression Inventory (see below) scores, a select number of participants (n=53) were recruited back for the stress testing phase of the study (discussed below). This experiment was approved by the Institutional Ethics Committee.

### Questionnaires

Upon arrival at the testing room participants completed an informed consent, demographics questionnaire (Appendix, Pg. 45) and Beck Depression Inventory (BDI). The demographics questionnaire contained information regarding daily caffeine intake, level of activity, and history of cardiovascular disease and depression.

#### Beck Depression Inventory (BDI).

The BDI (Beck et al., 1961) is a 21-item self report of depressive symptoms. The scale is designed to asses levels of symptoms commonly associated with depression i.e. guilt, self-hate, and loss of libido. Participants rank their symptoms on a 4 point scale ranging from 0-3. Scores can range from 0-63 with scores at or above 10 reflecting clinical level symptoms (Deardorff & Funabiki, 1985). In addition, this scale has shown to be reliable with an α-Cronbach value of 0.86 (Beck et al., 1961).

### Paced Auditory Serial Addition Task (PASAT)

The PASAT is a socially evaluative acute psychological stress task shown to reliably elicit a significant cardiovascular response (Phillips, 2010). Participants were instructed to sit quietly during the baseline and recovery phases of the task. During the stress phase participants were presented with a string of numbers. Participants were required to add consecutive numbers while retaining the most

recent number in memory so that it could be added to the next number. As the task progressed the numbers were presented to the participants at a faster pace. Before commencement of the PASAT participants were told that they would be videotaped and that their responses will be scored for accuracy. Each phase lasted 8 minutes and 4 measures of systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and pulse were obtained in each phase with the aid of a Dinamap Critikon Vital Signs Monitor SX1846 (Dinamap, Tampa, Florida). From the pulse measurement heart rate was determined. Heart rate variability (HRV: discussed below) was also assessed during the task utilizing an Embla S4500 Polysomnograph (Embla, Broomfield, Colorado).

**Stress Appraisal Measures**

Prior to the beginning of the PASAT and after receiving instructions pertaining to completion of the PASAT participants completed an anticipatory stress appraisal questionnaire. This questionnaire is an adaptation of the commonly used Stress Appraisal Measure created by Peacock and Wong (1990) and has been utilized in the past (Feldman et al., 2004). The questions are designed to evaluate the levels of threat and control felt by participants before a psychological stressor is applied. Participants respond on a standard Lickert scale ranging from 1-5 anchored by strongly disagree and strongly agree respectively.

Following completion of the PASAT participants responded to a reflective stress appraisal questionnaire. The structure and questions are the same as the anticipatory scale with the exception that the questions are worded in the past tense. This scale measures levels of harm and control felt by the participant upon completion of a psychological stressor. Both the anticipatory and reflective scales have been shown to be reliable with $\alpha$ Cronbach values of 0.81 and 0.80 respectively.

A post-PASAT inventory (Appendix, Pg. 46) was completed in conjunction with the reflective stress appraisal measure. This was a self-designed inventory aimed as assessing the level of anxiety induced by the PASAT and the level of engagement of the participant.

**Orthostatic Challenge**

*O*rthostatic challenge was assessed by having participants rise from a sitting to a standing position with arms still at their sides for 5 minutes. A 5 minute baseline period preceded the challenge and a 5 minute recovery period followed standing in which participants sat motionless in a chair. During each period three measures of SBP, DBP, MAP, and pulse were taken. HRV was measured continually in all three periods.

**Heart Rate Variability**

Participants were outfitted with two trans-thoracic Ag/AgCl wet EKG electrodes (Embla, Broomfield, Co). RemLogic software (Embla) was utilized to sample heart rate continuously at 500Hz (Hughes & Stoney, 2000) during both experimental conditions. High frequency (HF; 0.4Hz-0.15Hz) power was quantified after removal of artifact with the help of a fast Fourier transformation.

**Experimental Design**

Phase one of the experiment consisted of attaining informed consent as well as BDI and CES-D questionnaire data from participants. Participants were then re-contacted for physiological testing.

Upon arrival to the lab for phase two participants were seated in a sound attenuated room for instrumentation. Following instrumentation participants completed the orthostatic challenge and PASAT as well as anticipatory and reflective stress appraisal questionnaires.

**Data Reduction**

**Cardiovascular Parameters.**

Baseline, stress, and recovery phase measurements were averaged to form 3 and 4 phase means for the orthostatic challenge and PASAT, respectively. Reactivity was defined as the average difference between baseline and stress phase means. As a result of the discordance in the literature that exists in defining cardiovascular recovery, several recovery measures were computed. Recovery was defined as the average difference between baseline and recovery phase means (Stewart et al., 2001)

and percent recovery from stress phase means (Cole et al., 1999). In addition, recovery was dichotomized such that failure to recover was defined as a failure to return to baseline phase means at the conclusion of the recovery phase (Steptoe et al., 2006a). Finally, recovery was analyzed utilizing time point recovery measures in which the difference between time specific recovery measures and baseline phase means represented time specific recovery (Phillips et al., 2009). Orthostatic data was also reduced to time point change scores. This meant subtracting challenge phase scores from the baseline average. This allowed for a more time specific analysis of orthostatic response.

**Heart Rate Variability.**

Baseline, stress, and recovery phase measurements were averaged to create 3 and 4 phase means for the orthostatic challenge and PASAT respectively. In addition, time point changes for reactivity and recovery were computed for a more precise analysis of changes in HRV. Time point reactivity values were calculated by subtracting individual stress phase measures from baseline phase mean. Time point recovery was computed by subtracting individual recovery phase measures from stress phase means so that vagal rebound could be assessed.

## Statistics

**Control Analysis.**

Gender effects on BDI scores and cardiovascular reactivity were assessed with an ANOVA. If significant gender effects were observed gender would be controlled for throughout the entire statistical analysis. The relationship between body mass index (BMI) and cardiovascular reactions were also examined using linear regression. In the event of a significant relationship, BMI would be controlled for in the remaining analyses. BMI was calculated according to the definition provided by the CDC (2011): (weight (lbs)/ height (in.)$^2$) x 703.

**Blood Pressure Data.**

The same statistical analyses were carried out for the orthostatic challenge and PASAT. A whole group one way ANOVA comparing baseline and stress phases was used to confirm cardiovascular perturbation. Simple linear regression was utilized to explore the relationship between cardiovascular reactivity and recovery change scores and BDI. To analyze recovery two additional methods were utilized. An ANOVA was utilized to assess the difference in BDI scores between those who failed to recover to baseline and those who fully recovered. Finally, a repeated measures MANCOVA with baseline values as covariates was utilized to assess differences in blood pressure during the recovery phase between three BDI score groups (low (n=21), M=4.14(2.78), middle (n=19), M=14.63(2.79), high (n=6), M=28.17(4.71), all significantly different, $p<.05$).

**Heart Rate Variability Data.**

The same statistical analyses were carried out for the orthostatic challenge and PASAT. All HRV data was normalized by natural log transformation. A whole group one way ANOVA comparing baseline and stress phases was used to confirm HRV perturbation. Simple linear regression was utilized to the relationship between BDI and time specific reactivity and recovery change scores.

**Moderator and Mediator Analysis.**

In an effort to further elucidate the relationship between depression and cardiovascular responses to orthostatic challenge and acute psychological stress, heart rate variability was analyzed as a moderating and mediating variable. In other words, did heart rate variability represent an independent contributor to cardiovascular responses alongside BDI (moderator) or did it represent a mechanistic step in the relationship between BDI and cardiovascular reactions (mediator). To test HRV as a moderating factor CVR was regressed across BDI, HRV, and BDI X HRV. A significant effect of BDI x HRV was taken to implicate HRV as a moderator of cardiovascular reactions (Baron & Kenny, 1983).

Mediation was assessed using the methods outlined by Baron and Kenny (1983). Briefly, a series of regressions were carried assessing the effect of 1) BDI on cardiovascular reactions (c), 2) BDI on HRV (a), and 3) HRV on cardiovascular reactions while controlling for BDI (b) (See Figure 1). HRV was defined as a mediator if the effect of BDI on cardiovascular reactions was significant and decreased upon inclusion of HRV. If HRV was found to be a mediator, an *a priori* decision was made to conduct a bootstrapped (1000 times) Sobel (1982) test as outlined by Preacher and colleagues (2004) to further characterize mediation effects using a more statistically rigorous test. This directly evaluates the standard errors of the mediating effects of HRV on cardiovascular reactions ($ab = c\text{-}c'$) against the null hypothesis ($c\text{-}c' = 0$) thus reducing Type II error (Preacher & Hayes, 2004). The same analyses were carried out using primary stress appraisal measures to examine the role of stress appraisal as a moderator or mediator of the cardiovascular response to stress. Only Sobel tests results are presented in this discussion.

## Results

### Participant Demographics

After implementing exclusion criteria (daily smoking (n=6), diagnosed cardiovascular disease (n=6), and failure to complete the BDI (n=1)) a final sample of 107 participants was attained (Table 1). After implementing exclusion criteria for phase two (daily smoking (n=1), cardiovascular disease (n=5), and SBP hypertension (>140mmHg) at baseline (n=1) 46 participants were included in statistical analyses.

### Control Analyses

Gender did not significantly influence BDI scores (males: 10.07(7.27), females: 12.28(9.26), $F(1,44)=0.625$, p=ns) or cardiovascular reactivity changes scores (all $p>.05$). BMI did not significantly affect cardiovascular reactivity change scores (all $p>.05$). Gender and BMI were not controlled for in the rest of the statistical analyses.

**Cardiovascular Reactivity**

Repeated measures ANOVAs confirmed a significant perturbation of the cardiovascular system by the orthostatic challenge and PASAT (Table 2). Regression analyses showed no significant trends between BDI score and cardiovascular reactivity change scores during the orthostatic challenge. Significant relationships were noted between BDI score and heart rate, $t(44) = -2.94$, $p = .005$, $R^2 = .164$ and SBP, $t(44) = -2.07$, $p = .045$, $R^2 = .089$ reactivity change scores during the PASAT such that higher BDI scores were associated with blunted reactivity (Figure 2).

**Cardiovascular Recovery**

Cardiovascular recovery was defined by four methods found in the literature. When defined as the difference between recovery and baseline phase means no significant relationships were found (Table 3). Linear regression revealed a significant relationship between BDI score and percent recovery of heart rate, $t(44) = 6.67$, $p = .028$, $R^2 = .105$ and SBP (Table 4), $t(44) = 9.19$, $p = .022$, $R^2 = .114$. ANOVA comparing BDI scores between those with delayed and adequate recovery revealed no significant difference between groups (Figure 3). A repeated measures MANCOVA revealed significant differences between BDI groups for SBP, $F(2,42) = 5.26$, $p = .009$ (Figure 4). *Post-hoc* analyses revealed a significant difference of SBP between low and middle groups at stress phase, $F(2,43) = 4.35$, $P = .019$, recovery phase time 3, $F(2,43) = 8.10$, p=,001, and recovery phase time 4, $F(2,43) = 3.37$, $p = .044$. In addition a significant difference was found between middle and high groups at recovery time 3, $F(2,43) = 8.10$, p=,001.

**Vagal Activity during Stress Tasks**

Table 5 depicts the relationships between vagal reactivity and BDI scores during the orthostatic challenge and PASAT. No significant relationships were observed in the orthostatic challenge. A significant relationship was found at time point 8 in the PASAT such that increases in BDI were associated with decreases in vagal withdrawal, $t(42) = 2.18$, $p = .035$, $R^2 = .102$. Significant relationships were observed between BDI score and vagal rebound during the PASAT at recovery times 2-5 (Table 6).

Increases in BDI scores were associated with decreases in vagal rebound following conclusion of the stressor.

**Depression and Threat Appraisal**

Regression analyses revealed a significant relationship between BDI score and anticipatory threat appraisal, $t(43)=4.87$, $p=.000$, $R^2= .356$ (Figure 5A). Anticipatory threat/control ratios were also significantly related to BDI scores, $t(43)=4.28$, $p=.000$, $R^2=.298$ (Figure 5B). Anticipatory threat appraisal (Figure 6a), $t(43)=-2.26$, $p=.029$, $R^2=.106$, and threat/control ratios (Figure6B), $t(43)=-2.19$, $p=.034$, $R^2=.101$, were also significantly related to related to SBP blood pressure reactivity such that increased appraisals of threat were associated with blunted reactivity.

**Mediation Analyses**

Bootstrapped Sobel tests failed to reveal any significant mediation effects of HRV reactivity or stress appraisal on heart rate (Figure 7) or SBP reactivity (Figure 8). Similarly, no mediation effects of the same variables were observed for percent cardiovascular recovery of heart rate and SBP (data not shown).

## Discussion

**Depression and Cardiovascular Reactions**

The principle goal of the current study was to elucidate the mechanisms responsible for the relationship observed by many (Carroll et al., 2007; Phillips, 2010; York et al., 2007; Phillips et al., 2011; de Rooij et al., 2010; Schwerdtfeger et al., 2011; Salomon et al., 2009), but not all (Betensky et al., 2010), that increases in depressive symptomatology are associated with blunted cardiovascular reactions to laboratory stressors. The current study observed several trends. First, increases in BDI scores were associated with decreases in both reactivity and recovery. Second, BDI scores were associated with vagal rebound such that BDI increases conferred less rebound. No relationship was found between BDI score and vagal withdrawal. Finally, BDI scores trended directly with threat appraisal measures but neither

appraisal nor vagal activity was found to mediate the relationship between BDI and blunted reactivity and recovery.

Given that depression has been implicated as an independent risk factor for cardiovascular disease (Musselman et al., 1998) and has been linked with increased risk of coronary heart disease (Rugulies et al., 2002), ischemic heart disease (Barth et al., 2004), platelet dysfunction (Bruce et al., 2005) and total mortality (Barefoot et al., 1996) it is not entirely unexpected that depression is related to blunted cardiovascular reactivity. Research examining blunted reactivity has shown that it is related to obesity, poor self-reported health status, and poor immune function (Phillips, 2010; Phillips et al., 2009) leading researchers to suggest that blunted reactivity may contribute to or mediate the co-morbidity of depression and cardiovascular disease. The decreases in heart rate and SBP reactivity associated with higher BDI scores observed in this study adds support to this literature and is noteworthy given depression was assessed using self-report measures and the sample was young, generally healthy, and free of known cardiovascular disease.

Whereas cardiovascular reactivity has benefitted from a widely accepted operational definition, cardiovascular recovery has suffered from inconsistent operationalization and measurement diversity. As a result of this, cardiovascular recovery has remained largely unexplored in comparison to reactivity during the same time period (Linden et al., 1997; Haynes et al., 1991). A meta-analysis by Schuler and colleagues (1997) found that blunted recovery was related to risk factors for hypertension. Although a review of the issues inherent in recovery research is beyond the scope of this study, four of the most commonly utilized definitions of recovery are addressed in the current study in relation to depression.

Studies defining cardiovascular recovery as the difference between recovery and baseline phase means have found that individuals exhibiting blunted recovery of SBP are at increased risk of acute myocardial infarction (Laukkanen et al., 2004), while others have found that decreases in recovery are associated with poor mental health (Steptoe et al., 2006b) and are predictive of longitudinal increases in

resting blood pressure (Steptoe et al., 2005; Stewart et al., 2001). When defined this way significant relationships were not observed in the current study between depression and recovery from the PASAT. This observation falls in line with the findings of Salomon and colleagues (2009) who reported no difference in blood pressure recovery between depressed and non-depressed individuals. However, in relation to heart rate recovery the current study is at odds with the Salmon et al study (2009). We do not report a blunting of heart rate recovery with increases in depressive symptomatology whereas they reported impaired heart rate recovery among depressed individuals. It has been suggested by the Goswami and colleagues (2010) that since heart rate is predominantly mediated by the autonomic nervous system heart rate recovery from mental arithmetic stressors mostly occurs within the first two minutes following stressor conclusion. It may be that since the first pulse measurement was recorded two minutes into the recovery phase in the current study, we failed to capture any relationship that may have existed within the first two minutes.

Although studies exploring cardiovascular recovery are sparse in the psychosomatic literature, the exercise literature has benefitted from an intense research interest in cardiovascular recovery. Studies from this literature are commonly focused on parasympathetic reactivation following cessation of exercise and as a result commonly define recovery with relation to stress phase means (Cole et al., 1999). Blunted post-stress recovery has been linked with increased risk of ischemic stroke (Kurl et al., 2001), hypertension (Singh et al., 1999), and mortality (Cole et al., 1999) while other studies have found that those with angina pectoris (Hashimoto et al., 1993) and coronary artery disease (McHam et al., 1999) also exhibit blunted recovery. Work by Gordon and colleagues (2011) found that participants with major depressive disorder exhibited blunted recovery 1 minute after exercise cessation. This finding was corroborated by the work of Hughes and colleagues (2006, 2008) who found that depression status predicted blunted heart rate recovery. The current study observed, utilizing this technique, a significant relationship between BDI score and heart rate and SBP recovery such that increases in BDI conferred

decreases in recovery. In this context, these findings begin to suggest that blunted recovery may be a means by which depression is cardiotoxic. It can be imagined that as a depressed individuals confront daily stressors, failure to recover would increase what McEwen (2003) describes as allostatic load in the form of high arterial pressure which is detrimental on a chronic basis.

Finally, cardiovascular recovery has also been dichotomized as to whether baseline values are reached after a predetermined time period (Steptoe et al., 2006a). A study by Steptoe and colleagues (2006a) found that individuals failing to return to baseline exhibited greater intima-media thickness. Results of the current study failed to show any difference in BDI score between those individuals who returned to baseline values and those who failed to fully recover. A study by Phillips and colleagues (2009) assessed recovery with a repeated measures MANCOVA. It was found that blunted diastolic blood pressure recovery was characteristic of individuals who exhibited adequate reactions to influenza vaccines. When analyzed this way, it was found that individuals in the high BDI group exhibited significantly higher SBP during the last four minutes of the recovery phase. In fact, during the last six minutes of the recovery period, participants in the high BDI group exhibited an increase in SBP. It may be that the eight minute recovery period became a stressor itself resulting in an increase in blood pressure (Linden et al., 1997).

**Depression, Anticipatory Stress Appraisal, and Cardiovascular Reactions**

Given that stress appraisal represents a complex interplay between cognitive, emotional, and physiological processes (Blascovich & Mendes, 2000), the current study aimed to evaluate the relationship between self-reported depressive symptoms, stress appraisal, and cardiovascular reactions to acute psychological stress. In line with current literature (Folkman & Lazarus, 1986; Pakenham, 1999; Ferguson et al., 2000) this study found that BDI scores trended positively with anticipatory threat appraisal and threat/control appraisal ratio scores. It is probable that the direct relationship between

BDI and threat/control appraisal score was driven by the direct relationship between BDI and threat appraisal for no association was found between BDI and anticipatory control appraisal.

Tomaka and colleagues (1997) have suggested that stress appraisals result in a threat or challenge response in the sense that each is characterized by a distinct cardiovascular response. Although the literature is fairly inconsistent at this time some have suggested that threat appraisals trigger responses from the sympatho-adrenomedullary system and hypothalamic-pituitary-adrenal axis and thus should be characterized by exaggerated reactivity (Blascovich & Mendes, 2000; Compas, 2006). Consistent with this idea are the findings of Maier and colleagues (2003) who observed a direct relationship between threat appraisal scores and diastolic blood pressure reactivity. Others (Tomaka et al., 1993; Wright & Kirby, 2003) maintain the notion that threat appraisals elicit a reactivity that is more vascular in nature and that heart rate reactivity does not vary as a function of appraisal (Blascovich et al., 2000). The current study reports a negative relationship between SBP reactivity and threat and threat/control appraisals. Although this finding seems to be at odds with both sides of the literature, two factors support this finding. First, work by Folkman and colleagues (1986) observed a relationship between depressive symptoms and the usage of escape/avoidance coping style. It may be that those who scored high on the BDI and also appraised the PASAT as more threatening utilized this coping style and in the process became psychologically or emotionally *disconnected* from the task resulting in blunted cardiovascular response. Second, given that one of the contributing factors of SBP is the chronotropic and inotropic properties of the heart (Klabunde, 2011) and threat appraisals are characterized by a lack in modulation of cardiac function, it may be that a lack in cardiac modulation influenced SBP enough as to curtail the SBP reactivity of individuals scoring high on the BDI.

Despite the significant relationships between BDI, stress appraisal, and SBP reactivity, mediation analyses revealed no significant mediation effect of stress appraisal. This falls in line with the findings of

Kelsey and colleagues (2004) who also failed to observed stress appraisal as a mediator of the relationship between social evaluation and cardiovascular reactions.

With respect to the measurement of stress appraisal in the current study one limitation warrants discussion. The method utilized herein as well as by Feldman (2004) and Tomaka (1993) has received criticism for departing from the original construct of stress appraisal set forth by Lazarus and Folkman (1986). Opponents (Maier et al., 2003) argue that in the current method threat and challenge appraisals are the result of primary and secondary appraisals and are maintained as two ends of the same spectrum while in the original conception threat and challenge represented two possible results of primary appraisal only. Although subtle, this deviation implies that threat and challenge are mutually exclusive which may not be the case in real life situations.

**Depression, Heart Rate Variability, and Cardiovascular Reactions**

Utilization of measures of heart rate variability within the task reactivity paradigm has provided researchers with a window into the autonomic changes associated with cognitive and emotional challenge (Sandercock, et al., 2005). In terms of autonomic reactivity, a surge of studies have assessed vagal reactivity among depressed individuals. Studies by Rottenberg (2005), Hughes (2000), Sheffield (1998), and Tulen (1996) have shown that depression is associated with increased vagal withdrawal in response to emotion, cognitive, and orthostatic stressors respectively. Although this trend has been observed in a college sample (Hughes et al., 2000) and elderly population (Sheffield et al., 1998), other studies have shown that depression may be associated with blunted vagal reactivity (Gentzler, 2009; Shinba, 2008). The current study observed no association between BDI scores and vagal withdrawal during orthostatic challenge or PASAT. It may be that since the current sample was comprised of generally healthy, young individuals the orthostatic challenge was not sensitive enough to detect individual differences in cardiovascular regulation. Similarly, it may be that even though the PASAT was shown to significantly perturb the cardiovascular, it may not have represented as much of a challenge to

the sample in focus as it would to the general population given the participants in this study were college undergraduates and may use arithmetic skills more often than a nonstudent sample. Because of this it may not have perturbed the autonomic nervous system enough to detect individual differences.

A similar focus has been afforded to vagal rebound following stressor conclusion. In the current study BDI was not significantly related to vagal rebound in the orthostatic challenge. It is presumed that the reasoning provided above may play a significant role in the null finding. However, regression analyses revealed a relationship between BDI and vagal rebound such that higher BDI scores were associated with diminished vagal rebound during the first five minutes of the recovery period following conclusion of the PASAT. This finding accords with the reports of Rottenberg (2003, 2007b) who found that depressed individuals failed to exhibit vagal rebound following conclusion of crying episodes and speech tasks. A failure to reactivate the vagal innervations of the heart following a stressful episode may implicate a compromised regulatory system in the etiology of depression-cardiovascular disease co-morbidity (Rottenberg, 2003).

Mediation analyses revealed that neither vagal reactivity nor vagal rebound mediated the relationship between BDI and cardiovascular reactivity. As suggested by Hjortskov (2004), heart rate variability and blood pressure may change independently in response to stressors as a function of their influences from the central nervous system and adjacent tissues respectively. It may be that the two measurement endpoints in the current study in fact represent cardiovascular endpoints rather than intermediates in a single cardiovascular response.

**Limitations**

This study suffers from several limitations. First, respiration parameters were not controlled for when measuring high frequency heart rate variability. Although the literature exploring heart rate variability suffers from inconsistency in this domain and many authors fail to control for respiration parameters (Sheffield et al., 1998; Ring et al., 2002, Stein et al., 2000; Chambers et al., 2002;

Mezzacappa et al., 2001) some maintain that respiration parameters such as tidal volume and respiration rate must be controlled for, especially when using experimental paradigms that necessitate speech, since the frequency of respiration falls within the high frequency range (Grossman et al., 1991; Cyranowski et al., 2011). With that said the speech demand for the PASAT was very low so the influence of respiration may be minimal. Regardless, the results regarding heart rate variability must be interpreted with caution.

Second, no participant inquiry was made regarding medication use. It is plausible that the participants reporting high BDI score were utilizing anti-depressant medication which has been shown to suppress vagal activity (Rottenberg, 2007b). This could explain the relationship between BDI and vagal rebound following conclusion of the PASAT.

Third, the sample consisted of remarkably homogenous participants. All were young, free of cardiovascular disease, and generally healthy. Although this makes the findings in the current study notable given the homogeneity, the homogeneity may have washed out any relationship that may have existed between cardiovascular and psychological variables increasing the risk of Type II error. Fourth, due to practical constraints a modified form of the PASAT had to be adopted in which the experimenter was not present in the same room as the participants. It is possible that, even though the PASAT was shown to produce a significant perturbation of the cardiovascular system, the lack of direct social evaluation could have lessened the stressfulness of the task resulting in a dampened cardiovascular response. Finally, given that correlational nature of the statistics no speculation can be made as to directionality of the relationships observed.

**Future Research**

Given the limitations of this study future research should seek to establish these relationships as Salomon (2009) did in a young population with clinically diagnosed depression. Further, given the ubiquitous influence of autonomic nervous system in stress reactions further effort should directed

towards characterizing the autonomic milieu, in a controlled manner (respiration), of those with depression and how those characteristics predispose young individuals to cardiovascular disease in the future. Finally, given the dearth of research regarding cardiovascular recovery, the literature would benefit from extended recovery periods and a more rigorous examination of the psychological determinants of recovery that may play a role in the blunted recovery associated with depression.

**Conclusion**

The current study reports an indirect relationship between BDI score and heart rate and SBP reactivity to acute psychological stress in a young adult population free of cardiovascular disease. Although a direct relationship between BDI and threat appraisal was observed neither appraisal nor high frequency heart rate variability were found to mediate the heart rate and blood pressure response to the stressor. At the present, a majority of research has regarded cardiovascular reactivity as a mechanistic intermediate between depression and cardiovascular disease (Lavollo & Gerin, 2003; Lovallo, 2005). It follows that depression is related to blunted (Phillips, 2010; Carroll, et al., 2007) or exaggerated (Kibler & Ma, 2004) reactivity to acute stressors of which these abnormal reactivities confer risk of cardiovascular disease. It may be that in addition to being a mechanistic intermediate between depression and cardiovascular disease abnormal reactivity may act as a biomarker of a more pervasive regulatory dysfunction that gives rise to both psychological and somatic disease states. Given that cardiovascular reactivity is relatively easy to assess and that disadvantageous cardiovascular reactions have been well documented in young populations suffering from high levels of depressive symptomatology (Phillips, 2010; Carroll et al., 2007; Salomon et al., 2009; Brindle et al., 2011) it follows that cardiovascular reactivity might be utilized clinically to identify young individuals at risk so that early interventional treatments against depression and cardiovascular disease can be implemented.

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**Appendix**

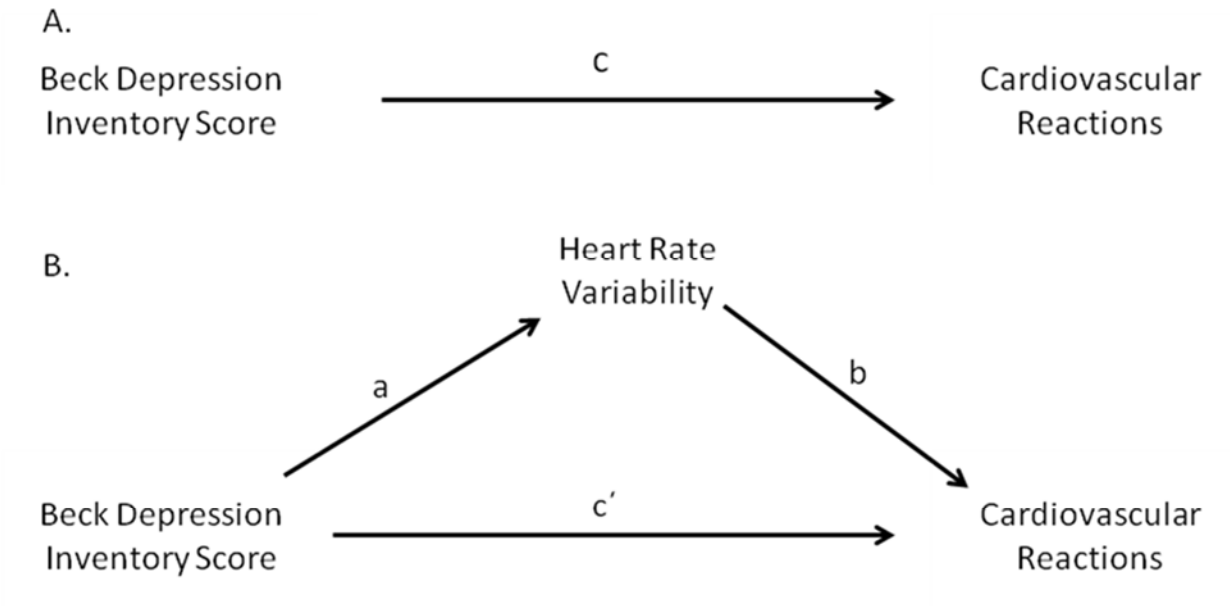


Figure 1. Theoretical meditation models of heart rate variability. **A.** The relationship between Beck Depression Inventory (BDI) and cardiovascular reactions to acute psychological stress. **B.** Theoretical mediation of the BDI-cardiovascular reactions relationship by HRV.

Table 1

*Demographics of Phase Samples*

| | n | Sex[1] | BMI | BDI |
|---|---|---|---|---|
| Phase One | 107 | 70.1% | 24.63 (4.31) | 10.07 (7.59) |
| Phase Two | 46 | 69.6% | 24.79 (4.56) | 11.61 (8.69) |

[1] Percent Female

Note: Values are given in Mean (SD)

Table 2

Cardiovascular Measures During Baseline, Stress, and Recovery Phases (n=44)

| | Paced Auditory Serial Addition Task | | | Orthostatic Challenge | | |
|---|---|---|---|---|---|---|
| Cardiovascular Measure | Baseline | Stress | Recovery | Baseline | Stress | Recovery |
| Mean Arterial Pressure | 78.00(7.63) | 88.30(6.69)* | 79.16(6.84) | 79.78(7.74) | 87.31(7.95)* | 79.28(8.03) |
| Heart rate | 74.61(9.76) | 85.15(11.06)* | 76.86(8.27) | 73.79(8.98) | 86.00(8.69)* | 74.38(8.60) |
| Systolic Blood Pressure | 109.93(12.70) | 121.40(14.26)* | 110.35(12.54) | 112.35(13.43) | 116.29(14.55)* | 111.24(13.28) |
| Diastolic Blood Pressure | 61.08(6.67) | 68.67(6.83)* | 61.59(6.45) | 61.77(6.43) | 68.68(6.40)* | 62.06(6.40) |

*p<.05 from baseline phase

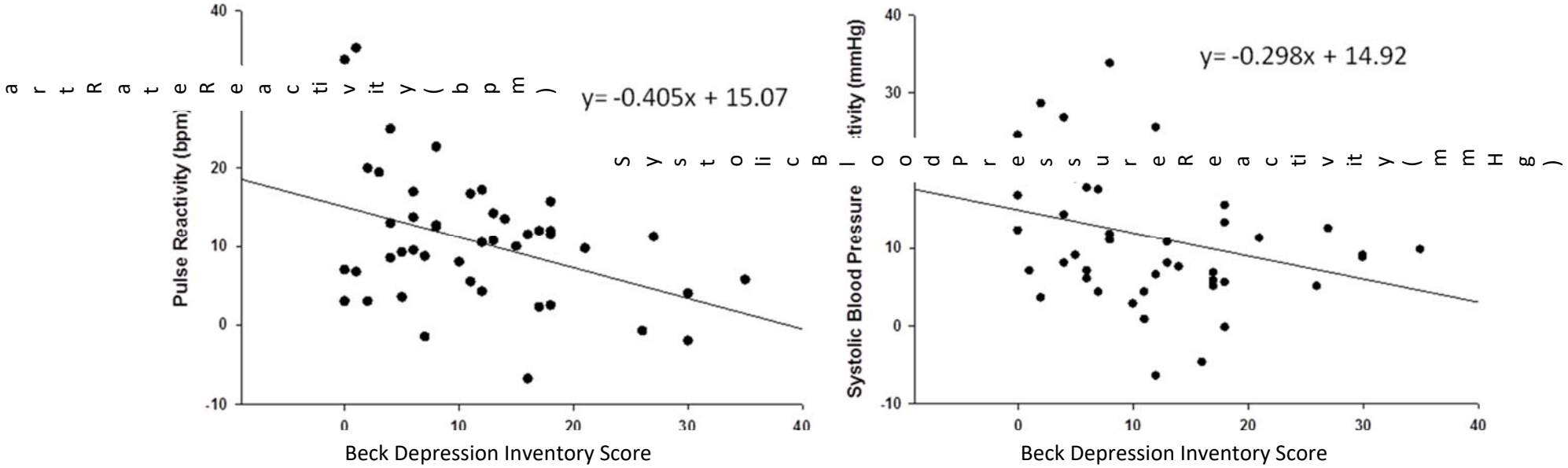


Figure 2. Relationship between Beck Depression Inventory score and **A.** Heart rate reactivity and **B.** Systolic blood pressure reactivity during the Paced Auditory Serial Addition Task.

Table 3

*The Relationship between Beck Depression Inventory Scores and Cardiovascular Recovery from the PASAT (n=46)*

| Cardiovascular Recovery Measure | B | β | $R^2$ |
|---|---|---|---|
| Heart rate | -0.088 | -0.151 | 0.023 |
| Systolic Blood Pressure | -0.046 | -0.084 | 0.007 |
| Diastolic Blood Pressure | -0.032 | -0.078 | 0.006 |
| Mean Arterial Pressure | -0.082 | -0.177 | 0.031 |

Table 4

*The Relationship between Beck Depression Inventory Scores and Percent Cardiovascular Recovery from the PASAT (n=46)*

| Cardiovascular Recovery Measure | B | β | $R^2$ |
|---|---|---|---|
| Heart rate | -0.352 | -0.323* | 0.105 |
| Systolic Blood Pressure | -0.201 | -0.337* | 0.114 |
| Diastolic Blood Pressure | -0.121 | -0.186 | 0.035 |
| Mean Arterial Pressure | -0.120 | -0.204 | 0.042 |

*$p<.05$

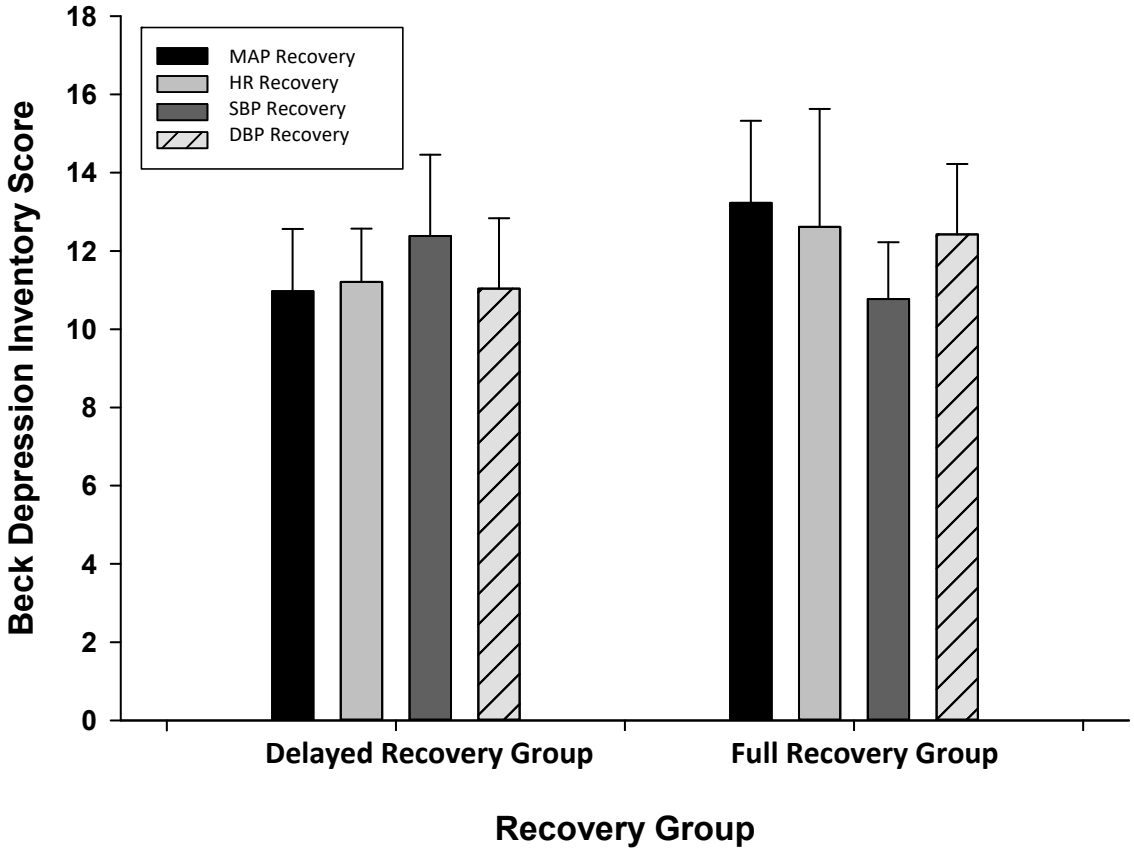


Figure 3. Group comparison of Beck Depression Inventory Score between participants who successfully recovered to baseline level following conclusion of the Paced Auditory Serial Addition Task and those who failed to fully recover to baseline. No significant differences were observed.

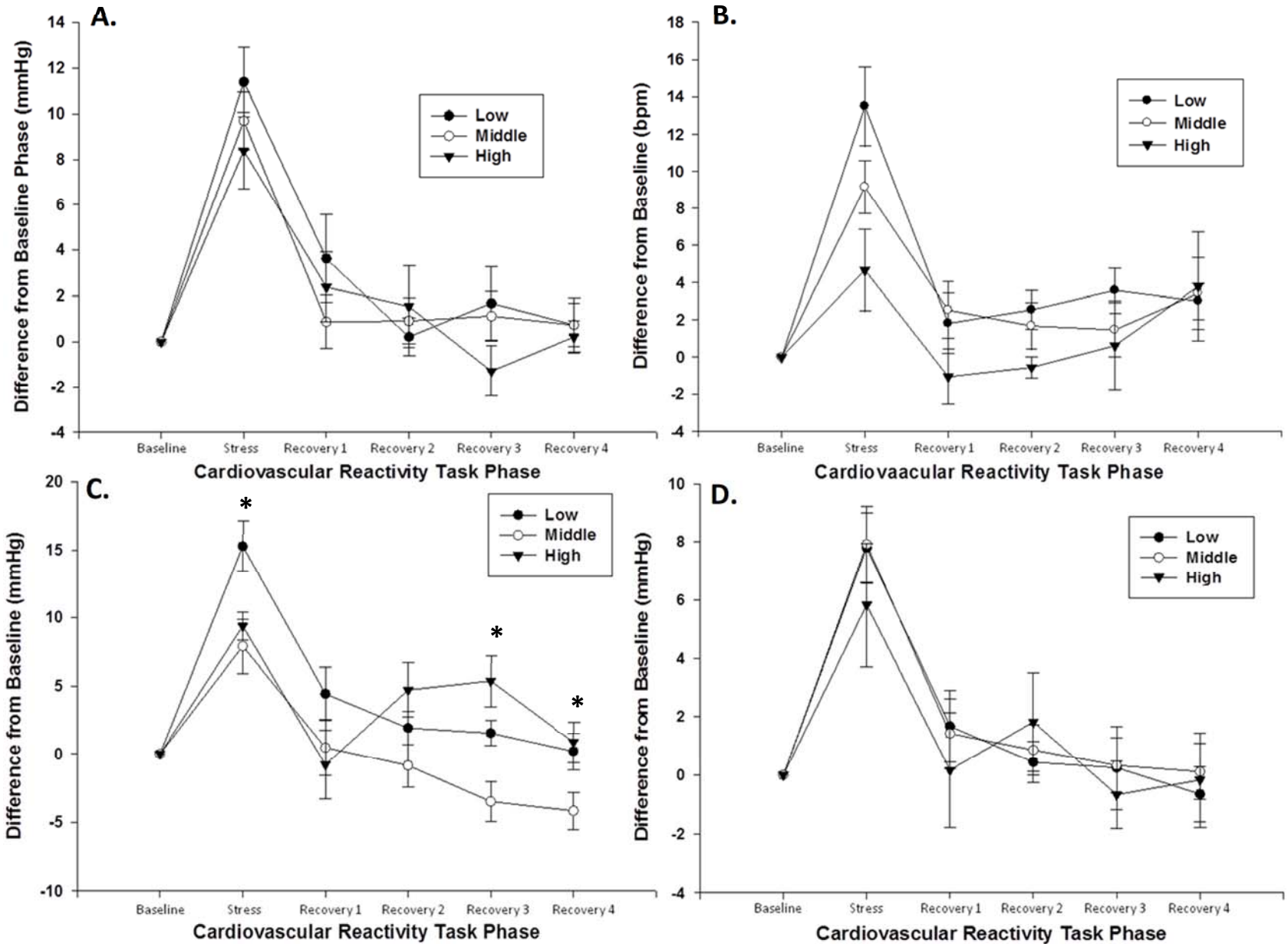


Figure 4. MANCOVA Results for Time Point Analysis of Cardiovascular Recovery. **A)** Mean Arterial Pressure, **B)** Heart rate, **C)** Systolic Blood Pressure, **D)** Diastolic Blood Pressure. Asterisks in **C.** denote significant difference ($p<.05$) between groups. Note: Y-axis varies between graphs

Table 5

*The Relationship between Beck Depression Inventory Scores and High Frequency Heart Rate Variability Reactivity (n=44)*

| | Paced Auditory Serial Addition Task | | | Orthostatic Challenge | | |
|---|---|---|---|---|---|---|
| Cardiovascular Measure | B | β | $R^2$ | B | β | $R^2$ |
| Overall Reactivity | 0.010 | 0.169 | 0.029 | 0.001 | 0.020 | 0.000 |
| Reactivity T1 | 0.003 | 0.038 | 0.001 | 0.000 | 0.004 | 0.000 |
| Reactivity T2 | 0.015 | 0.189 | 0.036 | -0.003 | -0.050 | 0.002 |
| Reactivity T3 | -0.005 | -0.069 | 0.005 | 0.009 | 0.146 | 0.021 |
| Reactivity T4 | 0.004 | 0.052 | 0.003 | 0.001 | 0.016 | 0.000 |
| Reactivity T5 | 0.004 | 0.061 | 0.004 | -0.003 | -0.046 | 0.002 |
| Reactivity T6 | 0.008 | 0.110 | 0.012 | | | |
| Reactivity T7 | 0.019 | 0.228 | 0.052 | | | |
| Reactivity T8 | 0.028 | 0.319* | 0.102 | | | |

Notes: Time point reactivity measures are defined as the difference between time point stress phase measure and baseline phase mean
*p<.05

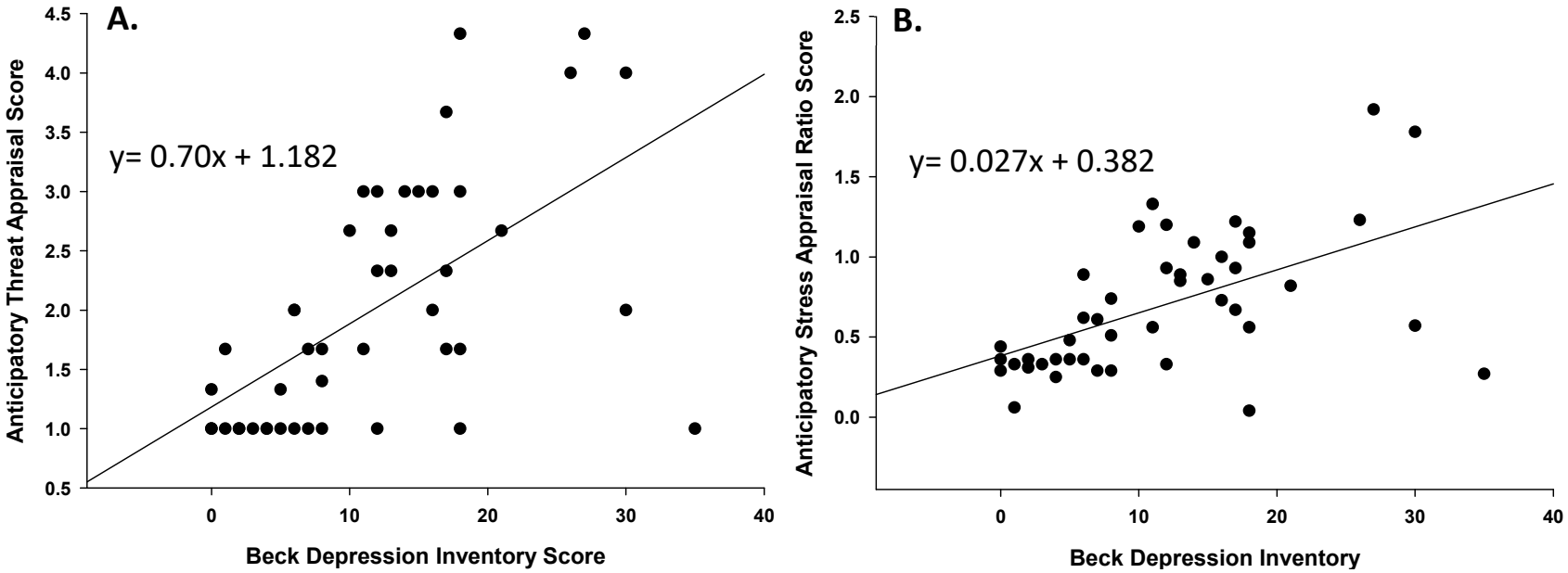


Figure 5. Relationships between Beck Depression Inventory Scores and **A.** Anticipatory Threat Appraisal Score and **B.** Anticipatory Threat/Control Ratio Score.

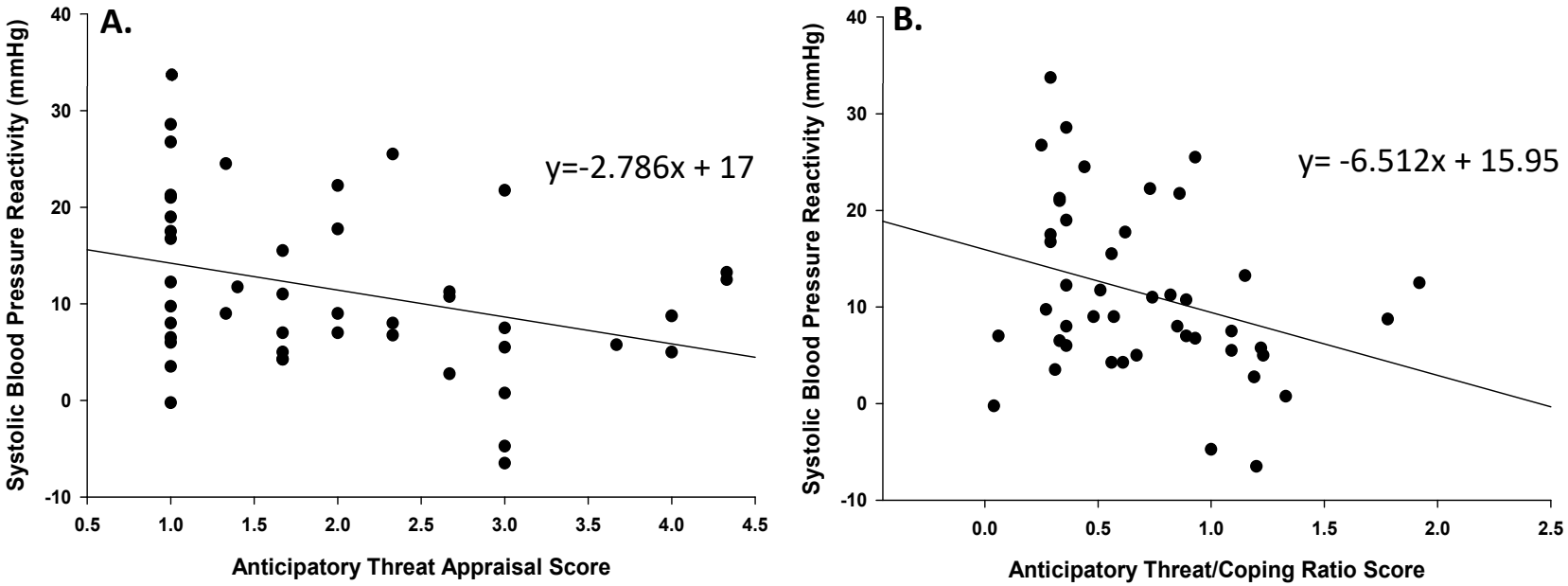


Figure 6. Relationships between systolic blood pressure reactivity and **A.** Anticipatory Threat Appraisal Score and **B.** Anticipatory Threat/Control Ratio Score.

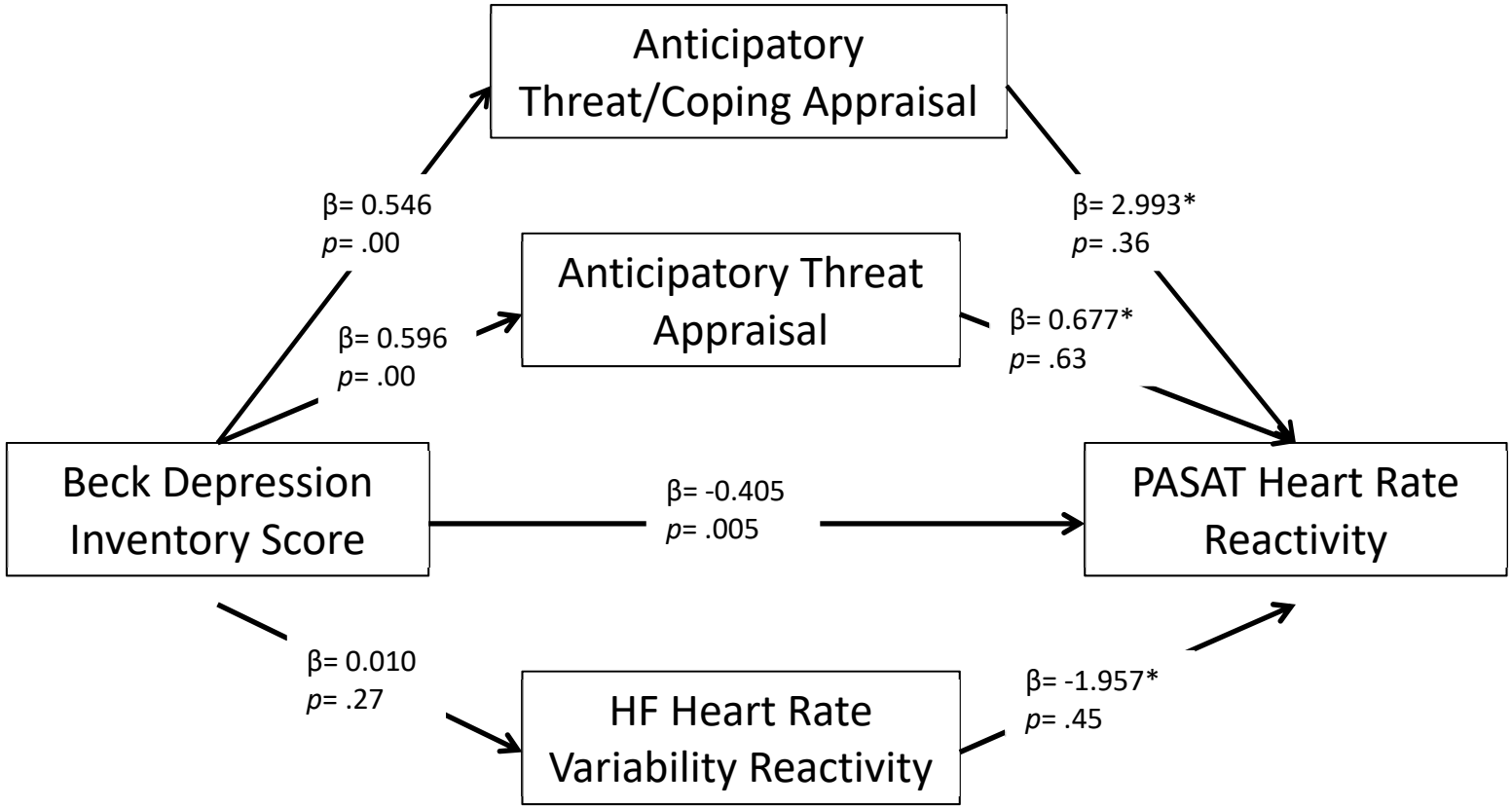


Figure 7. Mediation Analyses for Heart rate Reactivity during PASAT. No significant mediation was observed. Asterisks indicate regressions in which BDI was used as a control variable.

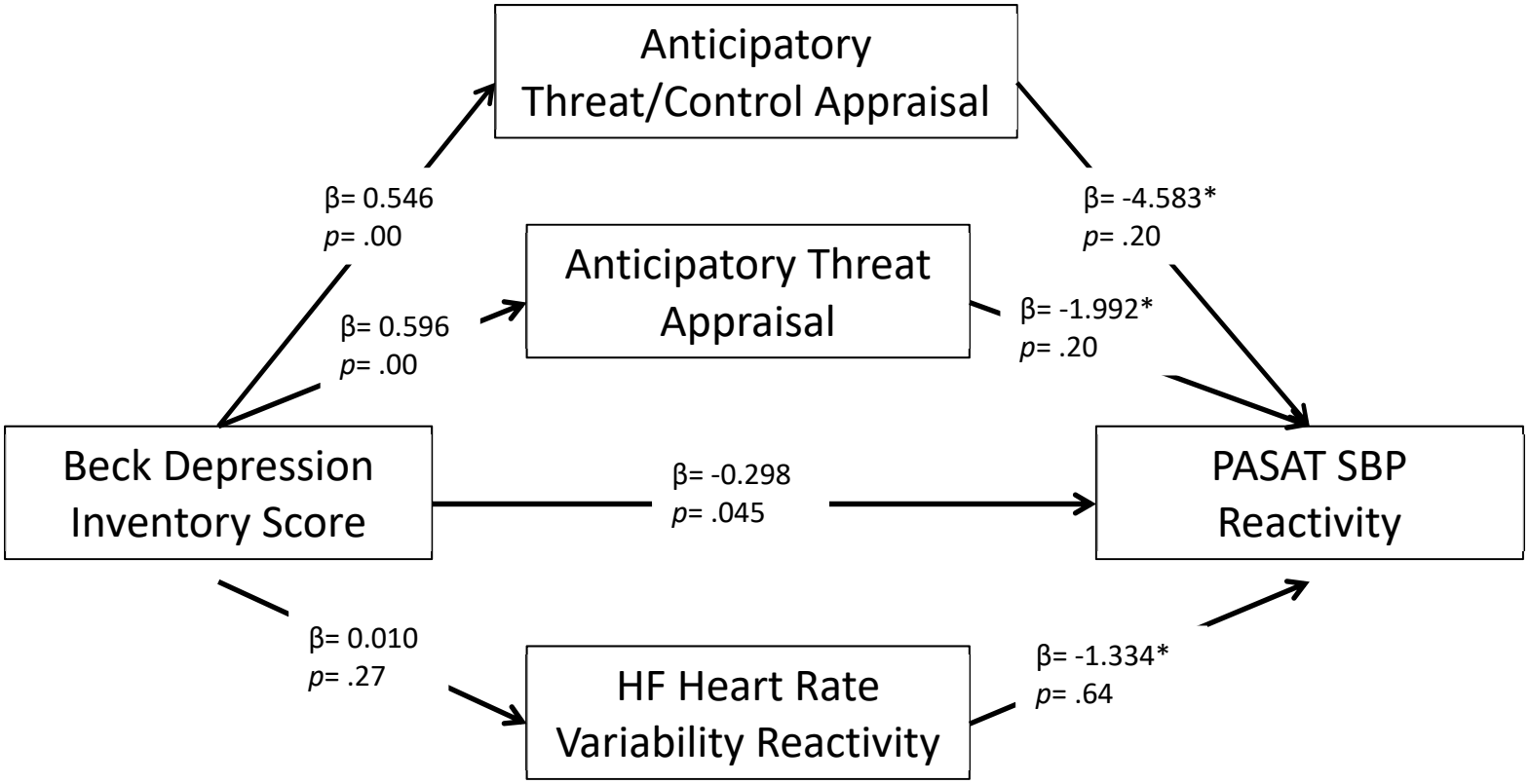


Figure 8. Mediation Analyses for Systolic Blood Pressure Reactivity during PASAT. No significant mediation was observed. Asterisks indicate regressions in which BDI was used as a control variable.

**RESEARCH PARTICIPANT CONSENT FORM**

# The Relationship between Mood and Cardiovascular Reactions to Acute Psychological Challenge

Ryan C.Brindle & Sarah M. Conklin, PhD.
Allegheny College Department of Psychology and Neuroscience

**Purpose of Research:** The purpose of this research is to characterize the relationship between depression and cardiovascular reactivity.

**Specific Procedures:** In participating in this project, I understand that I will be asked to complete questionnaires regarding health and psychological status. I understand that I may be asked to return for a **second testing session** where blood pressure and heart rate will be recorded while I complete a challenging mental arithmetic task and orthostatic challenge (standing).

**Risk to the Individual:** I understand that the risks of participating in this study are no more than I would experience in everyday life. I also acknowledge that due to the compression of the blood pressure cuff slight discomfort may be felt while blood pressure recordings are taken.

**Confidentiality:** I understand that at no time will my personal information or responses be made public, as all participants of this study will be given an encoded number to be referred to throughout the experiment. I understand that the data that I provide today is completely confidential and that the researcher is not interested in any one person's responses. Furthermore, I understand that the data will be stored securely in the Department of Psychology at Allegheny College and that this consent form will be equally secure but stored separately from my data. Further, I understand that any bonus points or compensation being awarded to me are not affected by the nature of my data provided.

**Voluntary Nature of Participation:** I understand that I do NOT have to participate in this study. Furthermore, I understand that if I do not agree to participate, I can withdrawal my participation at any time.

**Contact Information:** If I have any questions or concerns regarding this research project, I can contact Dr. Sarah Conklin at 814-332-2392 or via email at sarah.conklin@allegheny.edu or Ryan Brindle at 973-975-7048 or brindlr@allegheny.edu If I have any questions or concerns about the treatment of research participants (including myself), I can contact Dr. Jamie Lombardi, the chair of the Allegheny College Ethics Committee via email at jalombar@allegheny.edu.

**Study Results**: Results of this study are available upon request. If you would like a copy of your data obtained, please fill in your email address and/or Allegheny Box # below:

_____ _____

Email Box#

_____

Participant's Name (Please Print)

_____ _____

Participant's Signature Date

**RESEARCH PARTICIPANT DEBRIEFING FORM**

# The Relationship between Mood and Cardiovascular Reactions to Acute Psychological Challenge

Ryan C. Brindle & Sarah M. Conklin, PhD.
Allegheny College Department of Psychology & Neuroscience

Thank you for your participation in this study! Your assistance is greatly appreciated!

**Purpose of Research**: The purpose of this research is to investigate the relationship between mood and cardiovascular reactivity. It was hypothesized that depressed mood would react less to acute psychological challenge.

**Confidentiality**: The data you have provided will be kept in the strictest of confidence and will never be shared with the public in connection with your name. Your identity remains confidential and all data and forms obtained are securely stored in the Department of Psychology at Allegheny College. Furthermore, only group averaged data will reported in paper and posters summarizing the findings of this study.

**Concerns**: You are asked to not share the purpose of this experiment with other possible participants in an effort to help eliminate confounds.

**Contact Information**: Please feel free to contact the following if you have any questions or concerns.

**To Learn More:** If you have a more general interest in this area or research, you may also wish to consult the following references:

Musselman, D.L., Evans, D.W., & Nemeroff, C.B. (1998). The relationship of depression to cardiovascular disease. *Archives of General Psychiatry, 55*, 580-592.

Carney, R.M., Freedland, K.E., & Veith, R.C. (2005). Depression, the autonomic nervous system and coronary heart disease. *Psychosomatic Medicine, 67*(S1), S29-S33.

Carroll, D., Philips, A.C., Hunt, K., & Der, G. (2007). Symptoms of depression and cardiovascular reactions to acute psychological stress: Evidence from a population study. *Biological Psychology, 75*, 68-74.

- If you have any questions or concerns pertaining to this research project you may contact **Dr. Sarah Conklin** at **814-332-2392** or via email at sarah.conklin@allegheny.edu or Ryan Brindle at 973-975-7048 or brindlr@allegheny.edu

- If you have any questions of a psychological nature, you may contact the Allegheny College Counseling Center at **814-332-4368**. The Counseling Center is open **Monday-Friday 8am-5pm.**

- If you have any health-related concerns, please contact the Allegheny College Health Center at **814-332-4355** or your personal physician.

THANKS AGAIN FOR YOUR PARTICIPATION

**Demographic Sheet**

Participant #:_____

Date: _____

Height: _____ Feet _____ Inches

Weight _____ lbs.

Gender: M F

Average Number of Caffeinated Beverages (i.e. Coffee, soda, pop, energy drinks) per Day_____

Do you smoke Daily? Y N
Do you smoke on weekends? Y N

Do you exercise? Y N
How many times per week do you exercise (circle one): 1 2 3 4 5 6 7

How long on average do you exercise (circle one): 15minutes 30minutes 45 minutes 1hour 2 hours

What kind of exercise (all that apply): Aerobic Strength Other:_____

Have **you** ever been diagnosed with any cardiovascular condition? Y N

Is there a history of the following in your **family**?

| | | |
|---|---|---|
| Cardiovascular Disease (i.e. heart attack, atherosclerosis) | Y | N |
| Hypertension (High Blood Pressure) | Y | N |
| Depression | Y | N |

In relation to the question above, what family members have had **cardiovascular disease**?(Circle all that apply)

Mother Father Siblings Grandmother (maternal) Grandfather (maternal)

Grandmother (Paternal) Grandfather (Paternal) Other: _____

In relation to the question above, what family members have had **hypertension**? (Circle all that apply)

Mother Father Siblings Grandmother (maternal) Grandfather (maternal)

Grandmother (Paternal) Grandfather (Paternal) Other: _____

In relation to the question above, what family members have had **depression**? (Circle all that apply)

Mother Father Siblings Grandmother (maternal) Grandfather (maternal)

Grandmother (Paternal) Grandfather (Paternal) Other: _____

***Post PASAT Self report questionnaire***

*Please circle what applies.*

**How difficult did you find the task?**

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Not at all Difficult | 0 | 1 | 2 | 3 | 4 | 5 | 6 | Extremely difficult |

**How stressful did you find the task?**

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Not at all stressful | 0 | 1 | 2 | 3 | 4 | 5 | 6 | Extremely stressful |

**How arousing (exciting) did you find the task?**

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Not at all Arousing | 0 | 1 | 2 | 3 | 4 | 5 | 6 | Extremely arousing |

**How well do you think you performed?**

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Not very Well | 0 | 1 | 2 | 3 | 4 | 5 | 6 | Very well |

**How confusing did you find the task?**

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Not at all confusing | 0 | 1 | 2 | 3 | 4 | 5 | 6 | extremely confusing |

**How engaging did you find the task?**

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Not at all engaging | 0 | 1 | 2 | 3 | 4 | 5 | 6 | Extremely engaging |

**How embarrassing did you find the task?**

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Not at all embarrassing | 0 | 1 | 2 | 3 | 4 | 5 | 6 | Extremely embarrassing |

*version 1 27/3/07*