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DEPRESSION, ARTERIAL BARORECEPTOR SENSITIVITY, AND CARDIAC AUTONOMIC CONTROL AT REST AND DURING LABORATORY STRESSORS IN HEALTHY YOUNG MEN AND WOMEN

DISSERTATION

Presented in Partial Fulfillment of the Requirements for the Degree Doctor of Philosophy in the Graduate School of The Ohio State University

By
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The Ohio State University
2001

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Depression is a risk factor for mortality following myocardial infarction (MI), and one possible mechanism is autonomic nervous system functioning. Depression may be associated with reduced parasympathetic nervous system activity and/or increased sympathetic nervous system activity, increasing risk of ventricular arrhythmias and death. Although heart rate variability (HRV), a measure of the autonomic nervous system, has been related to depression in cardiac patients, investigations with healthy samples have yielded unclear results. Depressed mood was recently shown to be related to the magnitude of changes in the parasympathetically-mediated high frequency component of HRV (HF) during stressors among healthy young men and women. No relationship was observed between depression and HF at rest, although HF does not accurately reflect individual differences in levels of parasympathetic nervous system tone. The purpose of this investigation was to examine the relationship between depression and arterial baroreceptor sensitivity (BRS), a more adequate between-subjects measure of the parasympathetic nervous system. HF and pre-ejection period (PEP) were included as measures of the parasympathetic and sympathetic nervous systems, respectively. Lipid measures were also included to examine several more exploratory hypotheses regarding cholesterol and the autonomic nervous system. Participants (21 men and 29 women) completed psychosocial
measures and completed a laboratory stress protocol that included a challenging videotaped speech task and a forehead cold pressor task designed to evoke distinct patterns of autonomic nervous system activity. Measures of cardiovascular system responses, including blood pressure, heart rate, PEP, HF, BRS, and lipids were taken throughout the protocol. Depressed individuals did not differ from non-depressed individuals in their basal level of HF or BRS, but contrary to expectations had higher values of PEP. Depression was not related to changes in HF or PEP, but more depressed individuals had smaller decreases in BRS during the speech task. Cholesterol was unrelated to depression and most cardiovascular measures. Important limitations of the study include the relatively small sample size, restricted range of depression scores, and a restricted range of ages. If depression confers an increased risk of death after MI via altered autonomic functioning, treating depression and/or manipulating the autonomic nervous system may improve survival.
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INTRODUCTION

DEPRESSION, ARTERIAL BARORECEPTOR SENSITIVITY, AND CARDIAC AUTONOMIC CONTROL AT REST AND DURING LABORATORY STRESSORS IN HEALTHY YOUNG MEN AND WOMEN

Coronary heart disease (CHD) is the leading cause of death in the United States and the industrialized world. Given the profound impact of behavioral and psychological factors on its initiation, progression, treatment, and endpoints, CHD remains a major research focus in behavioral medicine. One area of increasing interest is the role of psychological risk factors for mortality following myocardial infarction (MI). Anxiety (Kawachi, Sparrow & Vokonas, 1994; Kubzansky, Kawachi, Weiss & Sparrow, 1998; Thomas, Friedmann, Wimbush & Schron, 1997), social support (Farmer et al., 1996), and depression have all been advanced as risk factors for mortality following MI. In particular, accumulating evidence implicates depression (Ahern et al., 1993; Frasure-Smith, 1991; Frasure-Smith, Lesperance, Juneau, Talajic & Bourassa, 1999; Frasure-Smith, Lesperance & Talajic, 1993; Frasure-Smith, Lesperance & Talajic, 1995a; Ladwig, Kieser, Konig, Breithardt & Borggreve, 1991; Lesperance, Frasure-Smith & Talajic, 1996).
Research in this area has progressed to the point that clinical treatment of depression following MI is increasingly being advocated to reduce the risk of mortality (Lesperance & Frasure-Smith, 1996), although the effectiveness of such treatment at reducing risk is not known (Carney, Freedland, Veith & Jaffe, 1999b). Toward this end, two randomized, controlled trials are in progress. One multi-center clinical trial (ENRICHD; Enhancing Recovery in Coronary Heart Disease) sponsored by the National Heart, Lung, and Blood Institute (NHLBI) is currently underway to evaluate the effects of a psychosocial intervention among post-MI patients who are depressed and/or have low social support (Blumenthal et al., 1997). Another trial, SADHART, is investigating the effect of a pharmacological treatment for depression (sertraline) on risk of mortality following MI (Carney et al., 1999b). At the time this document was written, the ENRICHD trial had completed participant recruitment, and follow-up data were being collected. Although the results of these trials, when available, will be instructive, the mechanisms by which depression confers an increased risk of mortality following MI will still not be fully understood. The purpose of the proposed study is to investigate one potential mechanism that may ultimately explain the relationship between depression and mortality following MI.

Depression and Mortality Following MI

A number of investigations have reported a higher risk of mortality, cardiac events, and arrhythmias in depressed patients following MI. In one early example, depression predicted cardiac deaths and arrhythmic events at 6 months after MI (Ladwig
Depression was measured using a self-report questionnaire designed for the study. This effect remained marginally significant after controlling for all the other CHD risk factors measured in the study.

In the Cardiac Arrhythmia Pilot Study (CAPS; Ahern et al., 1993), depression as measured by the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock & Erbaugh, 1961) was associated with a 1.38 relative risk of death or cardiac arrest, after controlling for measures of disease severity (e.g., previous MI, left ventricular ejection fraction, use of beta-blockers, use of digitalis). The patients in this sample were 502 individuals selected for having more than 10 premature ventricular contractions (PVC's) per hour or more than 5 episodes of non-sustained ventricular tachycardia recorded 6 to 60 days after their MI. The levels of depression reported in this study were generally sub-clinical, although those participants who died had scores indicative of mild depression.

Perhaps the most compelling evidence is found in the Montreal Heart Institute studies by Frasure-Smith and colleagues (Frasure-Smith et al., 1993; Frasure-Smith et al., 1995a; Lesperance et al., 1996). In these studies, major depression defined by either DSM-III-R criteria or a BDI score greater than 9 predicted post-MI mortality at 6 and 18 months. The effects were sustained after controlling for several measures of disease severity, including previous MI, Killip Class, and PVC's > 10 per hour. The sample consisted of 222 primarily male (74%) patients, and ages ranged from 24 to 88 years, with a mean of 60. In this sample, most depression related mortality occurred during the first 6 months, and depression seemed to interact with PVC's such that depressed people
with PVC's >10 had a much greater chance of dying than all other patients. Additionally, all of the patients with BDI scores > 10, PVC's > 10, and low left ventricular ejection-fractions (≤ 35%) died, prompting the authors to suggest that a potential physiological mechanism linking depression and post-MI mortality is altered autonomic functioning.

A follow-up study combined these data with data from the control group of a psychosocial intervention for post-MI patients (Frasure-Smith et al., 1997), in order to increase the number of women in the sample (Frasure-Smith et al., 1999). This allowed them to test whether gender interacts with depression to predict risk of mortality following MI. The total sample included 283 women and 613 men; depression was assessed using the BDI, and a 12-month follow-up was completed. Again, depression was independently related to cardiac mortality after controlling for other predictors such as age, disease severity, and smoking. In addition, women were found to be more depressed than men and were more likely to have BDI scores indicative of at least mild levels of depression (BDI ≥ 10), even after controlling for other demographic, disease severity, and psychological variables. Gender did not interact with depression to predict cardiac mortality, supporting the interpretation that gender differences in depression did not account for gender differences in cardiac mortality.

Related constructs

Constructs closely related to depression have also been studied, both separately and together with depression, as risk factors for mortality following MI. For example, it is widely accepted that clinical anxiety and depression frequently occur together, and that measures of anxiety and depression are highly correlated. One leading model, based
in part on psychometric analyses, proposes that anxiety and depression share a common factor of general affective distress, with specific factors differentiating anxiety (physiological hyperactivity) and depression (low positive affect; Clark & Watson, 1991). Unfortunately, studies evaluating depression, anxiety, social support, and other potential risk factors for mortality following MI typically are not grounded in well-developed theories explaining the interrelationships of these variables and their possible roles in conferring risk. Some studies combine these and other variables into more general categories, and some focus on the specific predictive power of one or more variable(s). Nevertheless, studies of the role of anxiety, psychological distress, and social support in post-MI mortality could inform a study of a potential mechanism linking depression and post-MI mortality.

Anxiety and psychological distress. Anxiety, depression, and a measure of general psychological distress predicted ventricular arrhythmias over a one year period after controlling for other predictors of arrhythmias (Follick et al., 1988). In this prospective study of 125 patients, general psychological distress was measured using the SCL-90 (Derogatis, Lipmman & Covi, 1973). Anxiety, depression, and the other subscales of the SCL-90 were not analyzed separately due to the high intercorrelations (i.e., .88 for depression and anxiety).

General psychological stress was also shown to predict mortality and subsequent MI in 229 men randomly assigned to the control condition of the Ischemic Heart Disease Life Stress Monitoring Program (Frasure-Smith, 1991). Psychological stress was measured during the patients’ hospital stays using the General Health Questionnaire
(Goldberger, 1972). Men characterized by high psychological stress were found to have over twice the risk of mortality than those characterized by low stress, after controlling for other predictors of mortality.

One recently reported investigation argued that a single personality trait accounts for the increased risk of mortality associated with anxiety, depression, and anger (Denollet & Brutsaert, 1998). In this prospective study of 87 patients with left ventricular ejection fraction (LVEF) < 50%, depression was measured using the pessimism and despair scales of the Millon Behavioral Health Inventory (Millon, Green & Meagher, 1982), anxiety was measured using the STAI (Van Der Ploeg, Defares & Spielberger, 1980), and anger was measured with the Trait Anger Scale (Van Der Ploeg et al. 1980). The investigators classified some patients as having a Type D personality, or the tendency to express negative emotions as defined by social inhibition and negative affectivity. Negative affectivity was operationalized as high anxiety, and social inhibition was measured by the social inhibition scale of the Heart Patients Psychological Questionnaire (Erdman, Duivenvoorden, Verhage, Kazemier & Hugenholtz, 1986). Over a follow-up averaging almost 8 years, the 27 patients classified as Type D (i.e., above the median on the two measures) were 4.7 times more likely to experience cardiac death or a non-fatal MI than those classified as not Type D, after controlling for LVEF. The measures of depression, anxiety, and anger did not add to the predictive power. These authors concluded that the Type D personality trait accounts for the relationship between various measures of negative affect and risk of mortality following MI.
An editorial comment questioned this conclusion on several grounds (Carney, 1998). The study used different measures from those reported in previous studies, which could account for different patterns of results. In addition, the risk contributed by each component of the Type D personality may not be the same (e.g., there is no epidemiological evidence that anger has an effect on post-MI prognosis). Furthermore, the physiological mechanisms relating components of the Type D personality to risk may be different.

Measures of depression, anxiety, anger, and social support were all included in one study by Frasure-Smith and colleagues (Frasure-Smith et al., 1993). In contrast to the Follick et al. study (Follick et al., 1988), high correlations among the variables were not reported. The highest correlation was .43, between the BDI and the state anxiety scale of the STAI, thus providing little evidence for a unitary construct. In addition to a BDI score greater than 10, state anxiety and a history of major depression were related to cardiac events (acute coronary syndromes such as MI) and probably arrhythmic events (survived cardiac arrest and arrhythmic deaths). However, all of these psychological risk factors except for social support were independently related to prognosis after controlling for measures of disease severity.

**Social support.** In addition to anxiety and general psychological stress/distress, low levels of social support have been related to a poorer prognosis following MI in some, but not all studies (Case, Moss, Case, McDermott & Eberly, 1992; Frasure-Smith et al., 1993; Ruberman, Weinblatt, Goldberg & Chaudhary, 1984). In the β-Blocker Heart Attack Trial, social isolation and stress predicted a more than four-fold increase in
mortality risk in 2320 men after controlling for other variables (Ruberman et al., 1984). In this study, social isolation and life stress were measured 2-3 months following MI using an interview-based questionnaire. The follow-up period was 36 months, and patients classified as both socially isolated and high in life stress were at the greatest risk. Similarly, social support was shown to predict mortality in the Multicenter Diltiazem Post-infarction Trial (Case et al., 1992). Patients in the placebo condition (n = 1234) were interviewed 3 to 15 days following their MI and were followed for an average of 2.1 years. Lower social support, defined as living alone, was an independent risk factor, after controlling for other physiologic and non-physiologic risk factors.

More recently, the relationship between social support and long-term mortality following acute MI was investigated in Hispanic and Caucasian patients (Farmer et al., 1996). Mexican Americans (n = 292) and non-Hispanic Caucasians (n = 304) were followed for an average of 43 months following their index MI. Greater social support, as assessed by a hospital interview and defined by marital status and living situation, predicted greater long-term survival in Mexican Americans, but not Caucasians.

Unfortunately, these studies tended not to measure other important variables such as depression and anxiety, and used relatively rudimentary measures of social support (e.g., living situation). As previously described, the only study using validated measures of depression, anxiety, and social support failed to find a relationship between social support and post-MI cardiac events (Frasure-Smith et al., 1993). Possible explanations for the negative results are the considerably smaller sample size and the failure of the larger studies to include validated measures of depression and anxiety. That is, Frasure-
Smith et al. suggested that social support may be a less powerful predictor than depression and anxiety, and that social support may have failed to reach statistical significance due to the smaller sample size of their study.

Together, these studies generally confirm that constructs related to depression are also risk factors for mortality following MI. The relationships between anxiety, depression, social support, and related constructs have not been clearly defined in this literature, and the unique contribution of each construct to an increased risk of mortality following MI is not conclusively established. In the present study, well-validated measures of anxiety and social support will be in order to assess the contribution of these constructs to any relationships observed between depression and potential mechanisms for the increased risk of post-MI mortality associated with depression.

*Mechanisms linking depression and mortality following MI*

A number of mechanisms have been identified as possibly responsible for the increased risk of mortality following MI conferred by depression (see Carney, Freedland, Rich & Jaffe, 1995a for review).

*Treatment adherence.* First, it is possible that depressed patients exhibit poorer adherence to treatment. To the extent that treatment adherence improves prognosis, this would help to explain the relationship between depression and mortality. In general, adherence rates to cardiovascular disease prevention strategies are poor, and treatment compliance has been shown to be a predictor of clinical outcome (Burke, Dunbar-Jacob & Hill, 1997). There is evidence that depression is associated with poorer adherence to both cardiac rehabilitation and beneficial lifestyle changes (Guiry, Conroy, Hickey &
For example, a recent report suggests that depression, as measured by the BDI, was associated with decreased treatment adherence measured 4 months post-MI (e.g., diet, exercise, smoking cessation; Ziegelstein, Bush & Fauerbach, 1998). However, adherence to treatment is related to coronary heart disease outcomes even when the adherence is to placebo medications (McDermott, Schmitt & Wallner, 1997), suggesting that nonadherence per se is associated with increased risk of mortality. On these grounds several groups (Carney et al., 1995a; Ziegelstein et al., 1998) have argued that depression that is responsible for both reduced adherence and poorer prognosis.

*Disease severity.* Perhaps the most intuitively appealing alternative explanation for a causal link between depression and mortality following MI is the possibility that depressed patients have more severe cardiac disease. Depression was associated with indicators of disease severity in at least two studies supporting depression as a risk factor for mortality following MI. Specifically, depression was associated with a history of treatment for hypertension, diabetes, previous MI, thrombolytic treatment, Killip class, and LVEF in one report (Frasure-Smith, Lesperance & Talajic, 1995a), and marginally related to LVEF in another (Denollet & Brutsaert, 1998). Most researchers in this area argue that differential disease severity does not explain the increased risk conferred by depression, because measures of disease severity are routinely employed as control variables (Carney et al., 1995a; Carney et al., 1999b; Frasure-Smith et al., 1993; Frasure-Smith et al., 1995a; Frasure-Smith et al., 1995b). Nevertheless, the ability of statistical methods to account for control variables is dependent on the reliability of those variables, and unreliability in the measures tends to systematically under-correct for
those variables (Cook, 1979). In addition, investigations of the relationship between
depression and the physiological mechanisms thought responsible for the increased risk
of mortality have yielded discrepant results (reviewed below). Therefore, tests of the
relationship between depression and each potential mechanism should be conducted with
healthy samples, as well as post-MI samples, to ensure that differential disease severity
is not a confounding variable.

*Cardiotoxic effects of antidepressants.* Another possible explanation for the
increased risk of mortality exhibited by depressed cardiac patients is the potentially
cardiotoxic side effects of anti-depressant medications. Tricyclic antidepressants
(TCA’s) appear to affect the cardiovascular system in different ways among cardiac
patients and healthy individuals. For example, TCA’s seem to have benign or even
beneficial effects on the cardiovascular system among healthy individuals. They do not
appear to impair left ventricular function, can reduce blood pressure, slow cardiac
conduction, and have an antiarrhythmic effect at therapeutic levels (Roose & Spatz,
1998). For post-MI patients with ischemic heart disease, TCA’s may be associated with
increased risk. Despite the fact that ventricular premature depolarizations are a risk factor
for mortality following MI, their suppression by treatment with antiarrhythmic drugs
does not reduce mortality (Furberg, 1983), and has even been associated with an
increased risk of death (Cardiac Arrhythmia Suppression Trial (CAST) Investigators,
1989; Cardiac Arrhythmia Suppression Trial II Investigators, 1992). Therefore, to the
extent that TCA’s have antiarrhythmic effects, they may not be safe for depressed
cardiac patients (Roose & Spatz, 1998).
However, possible cardiotoxic side effects of antidepressants are an unlikely explanation for the relationship between depression and mortality following MI (Carney et al., 1995a). Most patients who are depressed following MI are neither diagnosed nor treated (Freedland, Lustmann, Carney & Hong, 1992). In addition, the other major class of antidepressants, selective serotonin reuptake inhibitors (SSRI's) such as fluoxetine and sertraline, appear to have no adverse cardiovascular effects when taken by depressed cardiac patients (Roose & Spatz, 1998). As previously mentioned, a randomized clinical trial of sertraline is currently in progress with the expectation that it may reduce the risk of mortality following MI (Carney et al., 1999b).

Association with other risk factors. Depression is associated with known risk factors for heart disease such as smoking and hypertension. For example, individuals with a history of major depression are more likely to be smokers and are less likely to stop smoking (Glassman et al., 1990). However, depression predicts mortality independent of daily smoking (Frasure-Smith et al., 1995b). Depressed individuals are also more likely to have high blood pressure (Wells, Golding & Burnam, 1989). Again, a relationship between depression and mortality independent of history of treatment for hypertension has been demonstrated (Frasure-Smith et al., 1999). Therefore, it is unlikely that an association with other known risk factors fully accounts for the relationship between depression and mortality following MI.

Altered autonomic nervous system functioning. The mechanism currently receiving the most attention for explaining the relationship between depression and post-MI mortality is the possibility that depressed cardiac patients have altered autonomic
nervous system functioning. There are at least two physiological explanations linking altered autonomic nervous system functioning with an increased risk of cardiac death.

First, depressed cardiac patients may have increased sympathetic nervous system activity characterized by elevated plasma catecholamines. Elevated catecholamines may increase platelet aggregation, leading to an increased risk of myocardial ischemia and MI (Nemeroff, Musselman & Evans, 1998; Sheps, Sheffield & Carney, 1999). Interestingly, serotonin and catecholamines are relatively weak agonists of platelet aggregation, but amplify platelet aggregation in response to strong agonists (De Clerck, Xhonneux & de Courcelles, 1988). Combined with the prevailing view that depression is associated with dysregulated serotonin and norepinephrine (Siever & Davis, 1985), this hypothesis provides a link between central nervous system models of depression and a potential mechanism of increased risk for cardiac events. One study (Nemeroff et al., 1998) reported that depressed patients had enhanced baseline platelet activation and responsiveness, and speculated that exaggerated platelet reactivity may contribute to the increased risk of CHD and post-MI mortality observed in depressed patients; catecholamines were not measured in this study. However, Carney, Freedland, Veith, and Jaffe (Carney et al., 1999a) recently reported that depressed and nondepressed cardiac patients had similar plasma norepinephrine levels at rest, as well as similar changes in norepinephrine during an orthostatic challenge. While intriguing, this potential mechanism has not been adequately researched to allow any firm conclusions.

The second possible physiological mechanism linking depression and increased risk of mortality following MI is that depressed individuals may have increased cardiac
sympathetic and/or decreased cardiac parasympathetic (i.e., vagal) nervous system functioning. Both are known to increase the risk of ventricular fibrillation and sudden cardiac death (Podrid, Fuchs & Candinas, 1990; Schwartz, Snebold & Brow, 1976; Schwartz & Stone, 1980; Schwartz & Vanolli, 1981; Verrier & Lown, 1984). This possibility was suggested in at least one study (Frasure-Smith et al., 1995a) by a statistical interaction of depression with premature ventricular contractions. Specifically, those patients with BDI scores over 10 and more than 10 premature ventricular contractions per hour were most likely to die.

Cardiac parasympathetic nervous system functioning is particularly important in the development of cardiac arrhythmias. Parasympathetic tone helps to prevent arrhythmias by maintaining the electrical stability of the heart. In healthy individuals, enough parasympathetic tone is present to prevent fatal arrhythmias. When parasympathetic tone is decreased, as occurs as a consequence of MI, cardiac arrhythmias are more likely to occur (Verrier & Dickerson, 1994). Consistent with this reasoning, reduced HRV, an index partially determined by parasympathetic nervous system functioning, is a powerful predictor of increased mortality following MI (Bigger et al., 1992; Klieger, Miller, Bigger, Moss & The Multicenter Post-Infarction Research Group, 1987). Therefore, following MI, depression may contribute to a potentially dangerous reduction in parasympathetic tone, which can predispose patients to lethal arrhythmic events.
Depression and Heart Rate Variability

Investigations of heart rate variability (HRV) provide most of the evidence that altered autonomic nervous system functioning is the physiological mechanism by which depression increases post-MI patients’ risk of mortality. Some investigators also interpret differences in heart rate as evidence of changed autonomic functioning in depressed individuals (e.g., (Carney et al., 1999a). Heart rate and HRV reflect sympathetic, parasympathetic, and non-autonomic influences (e.g., differences in physical activity), and do not allow precise inferences regarding the autonomic origins of the group differences observed in these measures (Bernston et al., 1997; Berntson, Cacioppo & Quigley, 1991). Nevertheless, group differences in parasympathetic and/or sympathetic functioning would likely be reflected in corresponding group differences in HR and HRV.

Components of HRV have also been interpreted as measures of autonomic nervous system functioning (see for review Bernston et al., 1997; Task Force, 1996). The high frequency component of HF, or the variability occurring within the range of .12-.40 Hz, has been validated as an index of parasympathetic cardiac control (Cacioppo et al., 1994). This component of HRV is also commonly referred to as respiratory sinus arrhythmia, as it essentially reflects respiratory gating of parasympathetic influences on the heart (Berntson, Cacioppo & Quigley, 1993). No component of HRV has been adequately validated as an index of sympathetic activity (Bernston et al., 1997).
Investigations with Cardiac Patients. Investigations of HRV among depressed cardiac patients have reported an association between depression and reduced heart rate variability in coronary artery disease (CAD) patients (Carney et al., 1988; Carney et al., 1995b; Krittayaphong et al., 1997).

For example, one study (Carney et al., 1988) reported that the 9 depressed CAD patients enrolled in the study had lower HRV than did the 43 non-depressed patients, although the results did not reach statistical significance. Depression was assessed using contemporary psychiatric diagnostic criteria (American Psychological Association, 1994) HRV was assessed from 24-hr ambulatory EKG monitoring, and was defined as the standard deviation of R-R intervals over 5-minute recording intervals. Later, using the same measure of HRV, Carney et al. (Carney et al., 1995b) reported that 19 CAD patients diagnosed with major or minor depression had lower HRV than 19 age, sex, and smoking status-matched non-depressed patients. Another study comparing CAD patients divided into more and less depressed groups using a median split of scores on the depression scale of the Minnesota Multiphasic Personality Inventory (MMPI) reported that 22 patients higher depression scores had lower HRV than 20 with lower depression scores (Krittayaphong et al., 1997). In this investigation, HRV was also assessed as the standard deviation of R-R intervals over 5-minute recording intervals from 24-hr ambulatory EKG monitoring.

Although promising, one particular difficulty with investigations of cardiac patients is that reduced HRV is associated with CAD (Hayano et al., 1990; Martin et al., 1987) and is a consequence of MI (Bigger et al., 1992; Bigger et al., 1988; Klieger et al., 1987)
1987; Liao et al., 1996; Lombardi et al., 1987). Thus, depression and reduced HRV co-occur with CAD and following MI but may not be causally related. Tests of the relationship between depression and HRV involving non-cardiac samples are needed to control for the effects of MI on HRV.

Investigations with Healthy Participants. In contrast to the research with CAD patients, most studies using healthy samples have not found reduced HRV in depressed participants (Lehofer et al., 1997; Tulen et al., 1996; Yeragani et al., 1991). For example, individuals diagnosed with major depression using a structured clinical interview exhibited HF similar to that of sex- and age-matched controls. However, depressed individuals taking TCA’s did have lower HF in relation to depressed individuals not treated with TCA’s (Lehofer et al., 1997). The authors concluded that depression was not associated with altered parasympathetic functioning, but that the anticholinergic effects of TCA’s resulted in lower parasympathetic tone among medicated patients. Similarly, no differences in baseline HF were reported between women diagnosed with depression using the Hamilton Rating Scale (Hamilton, 1967) and age-matched controls (Tulen et al., 1996), although the small sample size may have resulted in insufficient power. Additionally, individuals diagnosed with major depression had HF similar to controls, whereas individuals diagnosed with panic disorder had lower HF than depressed and control participants when standing (Yeragani et al., 1991).

Not all of the studies with healthy samples have failed to find a relationship between depression and HRV. A recent study reported that depression was related to HF during a laboratory stress protocol. Of a sample of 60 healthy women, the 15 with the
highest BDI scores had lower HF throughout the protocol than the 15 with the lowest scores (Light, Kothandapani & Allen, 1998). In addition, the women with the highest BDI scores showed shorter pre-ejection periods (PEP) throughout the protocol, suggesting that they had increased sympathetic nervous system activity.

*Interpreting HRV.* In addition to the fact that differential disease severity may confound research with depressed and non-depressed cardiac patients, one reason for the discrepant findings between cardiac and non-cardiac samples may be the measurement of autonomic nervous system functioning. That is, studies reporting an association of depression and HRV in cardiac patients generally used measures derived from 24-hr EKG monitoring, such as the standard deviation of all normal R-R intervals within 5-minute epochs (Carney et al., 1995b; Krittayaphong et al., 1997). In contrast, the studies of HRV and depression in healthy samples generally employ shorter collection periods and specifically index the high frequency component (0.12-.40 Hz) of HF. The use of differing measures of HRV may reconcile the findings among cardiac patients and healthy participants. For example, one recent investigation of depression and HRV in cardiac samples that reported no baseline differences in HF (Sheffield et al., 1998). No investigations of depression and HRV in healthy samples have used measures derived from 24-hr ambulatory monitoring. Clearly such a study would help to clarify the literature.

These findings in cardiac and healthy samples are not consistent, and firm conclusions are not yet possible. The speculative reconciliation of the findings based on different measures of HRV does not rule out the possibility that depressed individuals
have reduced parasympathetic nervous system tone. One serious limitation of studies using HF to measure parasympathetic nervous system activity is the fact that this measure is much more interpretable as an index of changes in parasympathetic functioning than as a measure of parasympathetic tone (Bernston et al., 1997; Grossman & Kollai, 1993). Stated differently, within-subject analyses using HF accurately reflect changes in parasympathetic control of the heart, but between-subject analyses do not accurately measure differences in basal parasympathetic activity. Therefore, the finding that depressed and non-depressed participants do not differ at baseline with respect to HF does not necessarily rule out a between-subject difference in parasympathetic functioning.

*Depression and HF reactivity in a cardiac sample.* In addition to being more interpretable with respect to the autonomic origins, changes in (rather than baseline measures of) HF may be associated with depression. The one study that examined HF during a laboratory stress protocol in depressed cardiac patients reported that depressed individuals had larger HF changes than did non-depressed individuals (Sheffield et al., 1998). Depression was measured using the depression scale of the MMPI, and depression status was defined by median split. The results were interpreted to indicate greater reductions in parasympathetic cardiac control during psychological stress in more depressed patients.

*Depression and HRV reactivity in a healthy sample.* We recently examined the relationship between depression and HF in a non-cardiac patient sample at rest and during mental stress (Hughes & Stoney, 2000). Healthy college students completed a
laboratory stress protocol including a resting baseline, videotaped speaking task, and forehead cold pressor task. Parasympathetic cardiac control was measured as HF using power spectrum analysis. More depressed participants, as defined by a median split on BDI scores, had greater reductions in HF during the speaking task and smaller increases in HF during the forehead cold pressor task relative to less depressed participants. No differences between more and less depressed participants were found in baseline values of HF. These results indicate that depression is related to the magnitude of changes in parasympathetic cardiac control during mental stress, a finding similar to the effect observed in cardiac patients (Sheffield et al., 1998). Because these results were obtained with a non-cardiac sample, it is unlikely that the relationship between depression and parasympathetic cardiac control can be accounted for by disease severity. In addition, covarying anxiety did not eliminate the relationship between depression and changes in HF during stressors, nor was anxiety independently related to changes in HF during stressors. Unfortunately, a measure of social support was not included.

Studies using HF do not conclusively test the hypothesis that depression is related to parasympathetic tone. Still, the investigation of HF reactivity during stress may be warranted in its own right. Considerable data implicate psychological stress in cardiac arrhythmias and sudden death (see for review Kamarck & Jennings, 1991), and altered autonomic functioning plays a central role in vulnerability to stress-induced arrhythmia (Verrier & Lown, 1984). For example, one descriptive study found stressful experiences such as interpersonal conflicts to precede arrhythmic events in 25 of 117 cardiac patients (Reich, DeSilva, Lown & Murawski, 1981). In addition, greater
exercise-induced decreases in HF were observed in dogs susceptible to ventricular fibrillation than in non-susceptible dogs (Billman & Hoskins, 1989). To the extent that mental stress elicits dangerous decreases in parasympathetic control of the heart, findings that depression is related to the magnitude of stress related changes in HF may help to explain the relationship between depression and increased risk of mortality following MI.

Cardiac Arterial Baroreflex

In order to investigate potential depression-related differences in parasympathetic tone, measures that reflect between-subjects differences are needed. The cardiac arterial baroreceptor reflex is known to be responsible for much of parasympathetic tone (Kollai, Jokkel, Bonyhay, Tomcsanyi & Naszlady, 1994). Although, arterial baroreceptor reflexes for other end organs, such as those involved in regulating the vasculature, also include information about the sympathetic nervous system. In addition, measures of the cardiac baroreflex sensitivity (BRS) have been used in between-subjects investigations for many years.

Cardiac baroreflex and parasympathetic tone. The carotid arteries and aorta contain pressure-sensing nerves (i.e., baroreceptors) that respond to stretching of the artery wall caused by changes in arterial pressure. The firing of these baroreceptors is transmitted to the central nervous system and triggers reflex excitation of parasympathetic cardiac nerves that reduce heart rate to buffer changes in blood pressure (Chapleau, Hajduczok & Abboud, 1991). More specifically, input from the baroreceptors is relayed to the nucleus tractus solitarius, which exerts excitatory effects on
preganglionic vagal moto-neurons in the nucleus ambiguus (Spyer & Jordan, 1980). The arterial baroreceptors also fire in the absence of rapid transitions in arterial pressure, maintaining a tonic level of vagal activity (La Rovere, Mortara, Pinna & Bernardi, 1995).

The sensitivity of these reflexes, termed baroreceptor sensitivity (BRS), is a measure of the integrity of a primary contributor to PNS tone. The contribution of the cardiac arterial baroreflex to parasympathetic tone is substantial. For example, one investigation obtained a correlation of .81 between BRS and parasympathetic tone as revealed by pharmacological blockade (Kollai et al., 1994). In this study, conservative estimates of the baroreflex contribution to parasympathetic tone averaged 69%. Thus, the baroreflex is a primary determinant of parasympathetic tone. Other mechanisms, most likely central vagal drive (Berntson et al., 1993), were responsible for a smaller but substantial contribution to parasympathetic tone.

In addition, a recent comparison of the use of BRS and HF as between-subjects measures of parasympathetic tone concluded that BRS was a better predictor of parasympathetic tone (del Paso, Langewitz, Robles & Perez, 1996). Whereas HF and BRS were highly correlated, and both were abolished by parasympathetic blockade, only BRS predicted parasympathetic tone. At rest, BRS accounted for more than 97% of the variability in heart period under sympathetic blockade, although the small sample size ($N = 6$) in this study suggests that this estimate may not be very reliable.

In addition to measuring the integrity of a reflex that strongly contributes to parasympathetic nervous system tone, there is evidence that BRS includes information
not revealed by HRV measures. In one study, the correlations between BRS and components of HRV derived from 24-hour ambulatory monitoring ranged from 0.32 to 0.47 (Mancia et al., 1986). In another study, correlations between BRS and HRV did not exceed .63, prompting the authors to conclude that HRV and BRS are not entirely redundant (La Rovere et al., 1995). More importantly, each contributes independently to the prediction of risk of mortality following MI (La Rovere, Bigger, Marcus, Mortara & Schwartz, 1998).

*Measurement of BRS.* There are several methods for measuring BRS. The oldest involves the use of vasoactive drugs, most often phenylephrine, to induce changes in arterial pressures without directly affecting the heart or central nervous system (La Rovere et al., 1995; Sleight, 1992a). The resulting increases in heart period are then plotted against the preceding increases in systolic blood pressure, and the slope of the regression line is taken as an index of BRS (La Rovere et al., 1995). However, this method suffers from the limitation that blood pressure is manipulated directly, making it impossible to measure baroreflex regulation of blood pressure or peripheral resistance at the same time (Sleight, 1992b). This method also suffers from relatively poor standardized with respect to the dosage used and the time course of administration, which can alter the BRS estimate (Watkins, Grossman & Sherwood, 1996).

Another invasive method of measuring BRS involves the use of suction applied to the neck with a lead collar or other device. Negative or positive pressure is applied to the neck to activate or deactivate the carotid artery baroreceptors, which results in local blood pressure changes and concomitant heart rate changes used to estimate BRS. The
disadvantages of this method are potentially compromised patient comfort and the fact that the baroreceptors in the aorta are not stimulated, although this does not have a large effect (La Rovere et al., 1995). This method, as well as the phenylephrine method, provides measures of BRS during an experimental manipulation. Associated limitations include the fact that naturally occurring BRS is not measured, the fact that BRS can not be measured during different activities (Di Renzo, Parati, Mancia, Pedotti & Castiglioni, 1997), and the potentially confounding effects of anxiety, caused by the procedures, which is known to reduce BRS (Watkins et al., 1996).

Fortunately, non-invasive methods of measuring BRS have been developed. These involve the analysis of continuously recorded blood pressure and heart rate signals to reveal spontaneous BRS. Two data analytic strategies have been used most commonly. The “sequence technique” is based on time domain analyses, and the power spectrum analysis technique is based on frequency domain analyses (La Rovere et al., 1995). The sequence technique is based on automatic scanning of the SBP and RR interval beat-to-beat series, searching for spontaneous sequences of three or more consecutive heart beats characterized by progressive SBP increases, accompanied with a one-beat lag, by progressive RR lengthening or visa-versa (progressive SBP reduction followed by concomitant RR shortening). For each sequence, the regression line is computed between the SBP and RR values that form the sequence, and the slope of this line is taken as an index of BRS (Di Renzo et al., 1997). One common power spectrum analysis technique involves decomposing the SBP and RR waves into power spectra at different frequencies using Fast-Fourier transformations. Cross-spectral analysis is then
used to determine the magnitude of the RR interval changes associated with the SBP oscillations. BRS is measured using the cross spectrum of RR interval and SBP for frequencies from .010 to .129 Hz (Watkins et al., 1996). These two non-invasive methods are highly correlated and have been shown to provide similar information about BRS (Watkins et al., 1996). Correlations between estimates of BRS derived from the phenylephrine method and these non-invasive method have been reported as high as 0.94 (power spectrum analysis technique (Robbe et al., 1987)) and 0.96 (sequence technique; Parlow, Viale, Annat, Hughson & Quintin, 1995), although the samples were small (N = 8) and homogeneous.

Using the phenylephrine method and both non-invasive methods, one group of investigators reported significant correlations of 0.48 (power spectrum analysis technique) and 0.50 (sequencing technique; Watkins et al., 1996) with the phenylephrine method. Although the correlations were smaller than those previously reported, the sample was larger (N = 43) and included both hypertensive and normotensive participants. Importantly, similar information regarding reduced BRS associated with hypertension was obtained with invasive and non-invasive methods (e.g., about 30% lower BRS).

*BRS is related to mortality after MI.* Estimates of BRS provide a between-subjects measure related to parasympathetic tone. Like 24-hour measures of HRV, BRS is related to clinical outcomes following MI. Specifically, BRS predicts susceptibility to ventricular arrhythmia after MI in both animal (De Ferrari, Vanoli, Cerati & Schwartz,
1992) and human models (Farrel et al., 1991), as well as mortality after MI (Hohnloser et al., 1993; La Rovere, Spechia, Mortara & Schwartz, 1988; La Rovere et al., 1998).

Animal models use dogs prepared with a healed myocardial infarction. BRS is measured using the phenylephrine method, and dogs are classified as susceptible to ventricular arrhythmia or non-susceptible using a submaximal exercise test in which the circumflex coronary artery is occluded for 2 minutes to simulate myocardial ischemia. Dogs that experience fibrillation are considered susceptible, and this susceptibility is reproducible. A strong relation between BRS and susceptibility was reported (De Ferrari, Vanoli, Cerati & Schwartz, 1992), such that over 90% of the dogs with the lowest levels of BRS were susceptible, compared to only 12% of the dogs with the highest levels of BRS.

In a human model of susceptibility, programmed electrical stimulation of the ventricle was used to identify those patients susceptible and non-susceptible to sustained ventricular tachycardia (Farrel et al., 1991). A total of 68 patients were tested 7-10 days post-MI, HRV was measured from 24-hr EKG recordings, and BRS was assessed using the phenylephrine method. In susceptible patients, HRV and BRS were both reduced, and BRS was the strongest risk for susceptibility to ventricular arrhythmia. Strikingly, those patients with the lowest BRS exhibited over 35 times the risk as those patients with the highest BRS.

With respect to naturally-occurring arrhythmias following MI, lower BRS was observed in men exhibiting sustained ventricular tachycardia during 24-hour EKG monitoring relative to men without ventricular tachycardia (La Rovere et al., 1988).
Lower BRS was also observed in the 6 patients that died during the follow-up period. A similar finding was reported in 14 post-MI patients with at least one episode of ventricular fibrillation or tachycardia and 14 post-MI in whom no arrhythmia were observed (Hohnloser et al., 1993). Participants were carefully matched for age, sex, the location of the infarct, the extent of CAD, LVEF, blood pressure, and resting heart rate. Measures of HRV from 24-hr monitoring did not differ between the groups, but BRS assessed using the phenylephrine technique was lower in the group that experienced arrhythmia.

A large prospective study of 1284 patients with a recent MI has recently been reported, termed the Autonomic Tone and Reflexes After Myocardial Infarction Study (La Rovere et al., 1998). In this study, both HRV from 24-hr monitoring and BRS measured using the phenylephrine method predicted mortality during the 21-month follow-up period. The predictive power of each variable was not significantly different, but HRV and BRS predicted mortality better when used together. This was taken as evidence that HRV and BRS each contribute independent predictive power.

_BRS is related to depression in cardiac patients._ Given that BRS is a measure related to parasympathetic tone, and that BRS is related to clinical outcomes following MI, BRS may be a valuable measure to examine the hypothesis that altered parasympathetic nervous system functioning accounts for the increased risk of mortality conferred by depression. One study has investigated the relationship between depression and BRS in a cardiac sample (Watkins & Grossman, 1999). Among CAD patients, BDI assessed depression is associated with reduced BRS after controlling for age, a finding
consistent with the relationship between depression and HRV observed in cardiac samples. The 16 patients with the highest depression scores (BDI > 9) had about 30% lower BRS than the 14 least-depressed patients (BDI < 3). The mean BDI score for each group was not reported. In this study, BRS measured at rest using the power spectrum analysis technique. However, like HRV, BRS is reduced following MI (De Ferrari et al., 1992b), suggesting a need for similar studies in healthy samples to rule out the potential confound of disease severity.

**BRS is related to anxiety.** Because anxiety is a risk factor for arrhythmic events, at least two studies have investigated the relationship between anxiety and BRS. Using healthy men and women, trait anxiety was measured using the STAI, and BRS and HF were both measured non-invasively at rest using power spectrum analysis (Watkins, Grossman, Krishman & Sherwood, 1998). Anxiety was significantly negatively correlated with BRS ($r = -0.3$) and HRV (HF; $r = -0.26$). Patients in the quartile highest in trait anxiety ($N = 23$) had approximately 30% lower BRS than did those in the quartile lowest in trait anxiety ($N = 22$). More recently, BRS and HF were measured at rest using the same techniques at rest in healthy older adults diagnosed with major depression (Watkins, Grossman, Krishnan & Blumenthal, 1999). The severity of depression, as measured by BDI, was not significantly correlated with BRS ($r = -0.18$) or HRV ($r = -0.19$). State anxiety, as measured by the STAI, was significantly correlated with BRS ($r = -0.32$) but not with HRV ($r = -0.17$). In addition, the relationship of state anxiety to HRV and BRS was independent of BDI scores. Unfortunately, both studies suffer from the lack of a non-depressed control group.
This Study

There are no studies investigating the relationship between depression and BRS in healthy samples, and there are no studies investigating the relationship between depression and BRS at rest and during mental stress.

*Depression and BRS at rest and during mental stress*

The primary purpose of this study was to examine the relationship between depression and arterial baroreflex sensitivity (BRS) in healthy participants at rest and during mental stress. At rest, BRS can be interpreted as a measure of a reflex responsible for much of parasympathetic tone (del Paso et al., 1996; Kollai et al., 1994), although sympathetic influences are present to a lesser degree. During mental stress, typical simultaneous increases in heart rate and blood pressure strongly suggest that BRS is reduced during mental stress. This was confirmed by one investigation with human subjects reporting a reduction in BRS during a mental arithmetic task (Steptoe & Sawada, 1989). In addition, the evidence that depression is related to the magnitude of changes in parasympathetic cardiac control (Hughes & Stoney, 2000; Sheffield et al., 1998) suggests that stress-induced reductions in BRS may be related to depression. Therefore, two different stress tasks were included in the protocol. For comparability with Hughes and Stoney (Hughes & Stoney, 2000), a very similar laboratory task protocol was used in this study, including a speech task and a forehead cold pressor task. The speech task was designed to cause increased sympathetic nervous system activity and decreased parasympathetic nervous system activity, whereas the forehead cold pressor task was included primarily to increase parasympathetic nervous system activity.
Depression and cardiac autonomic control

Another purpose of this study was to examine the relationship between depression and autonomic cardiac control. Both HF and pre-ejection period (PEP), a non-invasive measure of sympathetic nervous system control of the heart (Cacioppo et al., 1994), were measured. Like HF, PEP is best used as a within-subjects measure. Including HF and PEP allowed for an attempt to replicate the previously reported relationship between depression and the magnitude of changes in parasympathetic cardiac control (Hughes & Stoney, 2000), and extended the investigation to test for a possible relationship between depression and the magnitude of changes in sympathetic cardiac control.

Relationships between BRS, HF, and cholesterol

Although not related to the primary hypotheses, the relationship between cholesterol, HF, and BRS was also examined. One previous report suggested that elevated total and low-density lipoprotein cholesterol (LDL-c) may be related to reduced HRV (Danev, Nikolova, Kerekovska & Svetoslavov, 1997), and another suggested that elevated LDL-c may be related to reduced BRS (Koskinen et al., 1995). Elevated cholesterol is a strong risk factor for CHD (Shekelle et al., 1981). Cholesterol is implicated in the process of atherosclerosis, and changes in cholesterol during mental stress may be a risk factor for CHD (Stoney & Hughes, 1999). Therefore, it is reasonable to hypothesize that people with elevated cholesterol or exaggerated cholesterol responses to stress may develop reduced BRS. Potential relationships between cholesterol and autonomic mechanisms that confer an increased risk of mortality following MI are
intriguing, and no studies investigating the relationship between depression and autonomic nervous system activity have included measures of cholesterol.

However, the most commonly observed relationship between depression and cholesterol is an inverse association in which individuals with very low cholesterol are more likely to be depressed (Morgan, Palinkas, Barrett-Connor & Wingard, 1993; Suarez, 1999). This suggests that elevated cholesterol is not responsible for the increased risk associated with depression, although studies relating depression and cholesterol in young healthy adults have not been conducted. Though exploratory, the importance of cholesterol and cholesterol responses to stress in conferring risk for CHD and mortality (Manninen et al., 1992) recommended the inclusion of cholesterol measures in this study.

Hypotheses

1. It is hypothesized that participants with higher depression scores will have lower baseline values of BRS than will participants with lower depression scores.

2. It is hypothesized that participants with higher depression scores will have greater reductions in BRS during the speech task and smaller increases during the forehead cold pressor task, relative to participants with lower depression scores.

3. It is hypothesized that participants with higher depression scores will have larger reductions in HF during the speech task and smaller increases in HF during the forehead cold pressor task, than will participants with lower depression scores.
4. It is hypothesized that participants with higher depression scores will have larger reductions in PEP during the stress tasks compared to participants with lower depression scores.

5. It is hypothesized that anxiety and social support will be correlated with depression, but will not account for the relationship between depression and BRS or the relationship between depression and autonomic cardiac control.

6. The relationship between depression and cholesterol will be examined, with no a priori hypothesis.

7. It is hypothesized that LDL-c cholesterol will exhibit an inverse relationship with HF and BRS.
METHOD

Participants

A total of 58 men ($N = 26$) and women ($N = 32$) were initially enrolled in this study. Six participants did not complete the protocol (see Table 1). The data from one additional individual was excluded because he reported being a smoker, and from one woman who was much more than 20% over her ideal body weight. All remaining participants were healthy, nonsmoking, normotensive (<90 mmHg resting diastolic blood pressure), within 20% of ideal body weight for their height, and not taking antidepressant medication or any medicine affecting cholesterol or blood pressure. The final sample size included 50 men ($N = 21$) and women ($N = 29$) for most analyses. The reasons for incomplete data are presented in Table 1.
<table>
<thead>
<tr>
<th>Number of participants</th>
<th>Reason³</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Indicated having smoked on a questionnaire</td>
</tr>
<tr>
<td>1</td>
<td>More than 20% over ideal body weight</td>
</tr>
<tr>
<td>5</td>
<td>Became faint and discontinued the protocol</td>
</tr>
<tr>
<td>1</td>
<td>Became nauseous and discontinued the protocol</td>
</tr>
</tbody>
</table>

Table 1: *Incomplete Data*

³Insufficient sample size precluded exploring whether or not participants excluded from the analyses differed systematically from those who were included.

Participants were recruited from the Research Experience Program in the Department of Psychology. An attempt was made to recruit participants that were "high" and "low" on depression scores, thus the mechanism for recruiting participants was to telephone potential participants identified through a group pre-screening using the Center for Epidemiologic Studies-Depression questionnaire (CESD; Radloff, 1977). Students with CESD scores greater than 20, and those with scores less than or equal to 10, were invited to participate. A cutoff score of 16 on the CESD corresponds to the 80th percentile and has been used to identify probable cases of clinical depression (Radloff, 1977). Participants reporting elevated blood pressure, cholesterol, possible heart disease, diabetes, other medical conditions, antidepressant use, and weight greater than 20% over ideal body weight on the prescreen were not contacted.
The CESD was used instead of the BDI for three reasons. First, unlike the BDI it does not include a question indicating potential for self-harm, and therefore represents less legal risk to investigators when used in contexts where rapid identification of at-risk students and prompt intervention are impractical. Secondly, another investigator at The Ohio State University routinely prescreens all sections of Psychology 100 using the BDI. Using the BDI as a prescreening instrument for this study would have resulted in a reduced allotment of students to prescreen and the identification of an insufficient number of prospective participants, or would have necessitated sharing the prescreen data. While this investigator was very generous in sharing prescreen data in a previous collaboration, they were not fully available until week 5 of the 10-week quarter. Thus, insufficient time may have remained in the quarter to recruit and schedule sufficient participants if prescreen data had been shared for this study. Finally, although the BDI was used in analyses for consistency with previous research, the CESD is also appropriate because it has been used in recent epidemiological research linking depression and risk of CHD (Ferketich, Schwartzbaum, Frid, & Moeschberger, 2000; Mendes de Leon et al., 1998).

In addition to recruiting participants from the Research Experience Program, attempts were made to recruit additional participants through the Psychological Services Center in the Department of Psychology, the Counseling and Consultation Service at The Ohio State University, and through advertisements directed at Ohio State University students. The attempt to recruit participants from the Counseling and Consultation Service used a brochure distributed by counselors to their clients. However, no
prospective participants responded from any of these recruiting sources. The prescreen, advertisement, brochure, and telephone prescreening are included in Appendix A.

Participants received partial course credit and $10 as compensation for completing the study. Depression status (High versus Low) was determined based upon responses to the BDI at the time of participation. The BDI was used as the primary measure of depression for consistency with previous research in our lab (Hughes & Stoney, 2000), as well as with the strongest research implicating depression as a risk factor for mortality following MI (Frasure-Smith et al., 1993; Frasure-Smith et al., 1995a; Frasure-Smith et al., 1995b). Participant demographic characteristics are presented in Table 2.
<table>
<thead>
<tr>
<th>Variable</th>
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<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td>Low</td>
</tr>
<tr>
<td>(N)</td>
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<td>12</td>
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<tr>
<td>Age in years ((M \pm SD))</td>
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<td>19.1 ± 1.2</td>
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<td>Ethnicity(^a)</td>
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<tr>
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<td>3</td>
</tr>
</tbody>
</table>

Table 2: *Demographic Data by Gender and BDI Median Split*

\(^a\)Insufficient expected values precluded the use of Chi square tests of independence.

\(^b\)Some participants failed to respond to this item.
Stressors

A challenging speech task and a forehead cold pressor task were employed as stress tasks in this study. Two different tasks were chosen to elicit different cardiovascular responses during stress. The speech task typically elicits a decrease in HF (Berntson et al., 1994; Hughes & Stoney, 2000), a decrease in BRS (Steptoe & Sawada, 1989), a decrease in PEP (Berntson et al., 1994), and increases in blood pressure (Hughes & Stoney, 2000; Stoney & Hughes, 1999). The speech task required participants to mentally prepare and then deliver a 3-minute videotaped speech about a hypothetical situation. The situation was one in which the participant has been falsely accused of shoplifting and has to defend him or herself to a police officer in a store’s security office. The speech was video-recorded on a closed-circuit system, and participants were instructed that their speech would be rated later for poise, articulation, and appearance. The forehead cold pressor task was intended to evoke the diving reflex, which is known to increase parasympathetic activity (Durel et al., 1993; Khurana, Watabiki, Hebel, Toro & Nelson, 1980), and should therefore result in an increase in HF (Hughes & Stoney, 2000) and BRS. The forehead cold pressor task consisted of placing a bag of ice water on the participant’s forehead for 3 minutes. The bag of ice water was a standard cold-compress type, such as is used to apply ice to a headache or sore muscle. The bag of ice water contained 200 ml water and 800 ml ice, and measured 4° C.
Psychosocial Measures

Participants completed demographic measures, health and medical history questionnaires, and several other psychosocial measures designed to assess emotional, psychological, and physical functioning. The psychosocial measures were the BDI inventory (BDI; Beck et al., 1961), the Interpersonal Support Evaluation List (ISEL; Cohen, Merelstein, Kamarck & Hoereman, 1985), the Cohen Perceived Stress Scale (PSS; Cohen, Kamarck & Merelstein, 1983), the Positive and Negative Affect Scale (PANAS; Watson, Clark & Tellegen, 1988), the Anger Expression scale (AES; Spielberger et al., 1985), and the State-Trait Anxiety Inventory (Spielberger, Gorsuch & Luschene, 1990). The PANAS provides measures of positive and negative affect (PANAS-P and PANAS-N, respectively). The AES is scored to provide three measures; anger-in, anger-out, and anger-control. All measures are included in Appendix B.

Non-invasive Measures

Height and weight were measured prior to beginning the laboratory protocol. Participants were weighed to the nearest \( \frac{1}{2} \) pound (0.23 kg) using a standard balance scale with the participants in street clothes with shoes removed. Heights were measured to the nearest \( \frac{1}{2} \)" (0.64 cm) using a ruled metal bar permanently affixed to the scale for that purpose. BMI was computed as weight divided by height squared (kg/m\(^2\)). Blood pressure, impedance cardiogram, and EKG data were collected continuously throughout each phase of the protocol. All non-invasive physiological data were collected and analyzed on a personal computer using ANS Suite (Department of Psychology, The Ohio
State University, Columbus OH, 43210), which is custom software programmed using Labview software (National Instruments, 6504 Bridge Point Pkwy, Austin, TX 78730).

Blood pressure was measured using a Cortronics model 7000 arterial tonometry monitor (Colin Medical Instruments Corp., 5850 Farinon Drive, University Technology Park, San Antonio, TX 78249), providing systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and pulse rate. The blood pressure waveform was digitized at 500 Hz, and peaks (SBP) and troughs (DBP) were located by calculating the first and second derivatives of the blood pressure waveform and eliminating peaks and troughs falling outside basic amplitude and timing parameters. Each minute of data was visually inspected for errors in signal detection, and missing and incorrectly marked peaks and troughs were corrected manually. MAP was calculated from SBP and DBP on a beat-by-beat basis, and average SBP, DBP, and MAP values were averaged over each minute to provide one blood pressure value for each minute.

Impedance cardiogram and EKG data were measured using a Minnesota model 304B Impedance Cardiograph (Surcom Inc., Minneapolis, MN). The impedance cardiograph was used to measure electrocardiogram (EKG), basal thoracic impedance ($Z_0$), and the first derivative of the impedance signal ($dZ/dt$). Four disposable Mylar band electrodes were placed in a standard tetrapolar configuration following established guidelines (Sherwood et al., 1990). Specifically, two were placed circumferentially around the base and upper part of the neck and two were placed around the thorax at the level of the xiphisternal junction and
the lower region of the rib cage. Signals were digitized at 500 Hz. The EKG signal was sampled continuously at 500 Hz from three disposable EKG spot electrodes placed in a tetrapolar configuration.

HR and HF were calculated from the EKG signal. First, R-wave detection was performed using ANS Suite, which locates peaks and troughs using low pass filtered versions of the first and second derivatives of the EKG signal. Each minute of EKG data was visually inspected for errors in signal detection, and missing and incorrectly marked beats were corrected manually. After R-wave detection, an interbeat interval (IBI) series was generated using a previously described algorithm (Bemtson, Quigley, Jang & Boysen, 1990). A heart period time series was created from the IBI series using a “weighted” beat algorithm (Bemston, Cacioppo & Quigley, 1995). This algorithm also detected sharp transitions in the heart period time series, which were removed by smoothing because they potentially distort the Fast Fourier Transform and do not have implications for HF. In order to remove very large ultra-low frequency trends (including the DC component) from the input signal, a linear (first order) polynomial was fit to, and subtracted from, the heart period time series (Litvack, Oberlander, Carney & Saul, 1995). The heart period time series was then band pass filtered using an interpolated finite impulse response filter (Neuvo, Cheng-Yu & Mitra, 1984). The mean heart rate was calculated at 60,000 divided by the mean of the heart period time series. The power spectrum of the heart period time series was calculated using a Fast Fourier Transform (FFT) and expressed as msec²/Hz. The HF value (msec²) was calculated as the natural log of the area under the power spectrum within the corner frequencies of the band pass filter (.12-.40).
To calculate pre-ejection period (PEP), R-peaks were detected and marked as described above using the EKG signal. The EKG and dZ/dt signal was inspected on a beat-by-beat basis. Incorrectly marked R peaks were again corrected manually and dZ/dt artifacts were eliminated by deleting the accompanying R peak. An ensemble averaged EKG and dZ/dt was created and the Q, R, B, and X points were located using ANS Suite. Each ensemble average was visually inspected and miss-marked points were manually corrected. PEP was defined as the time (ms) from the onset of the EKG Q-wave to the B-point of the dZ/dt wave.

To calculate BRS, a technique known as the sequence technique was used. The sequence technique is based on automatic scanning of the SBP and pulse interval (PI) beat-to-beat series. The sequence technique locates spontaneous sequences of three or more consecutive heart beats characterized by progressive SBP increases, accompanied with a one-beat lag, by progressive PI lengthening or visa-versa (progressive SBP reduction followed by concomitant PI shortening). These sequences are termed triplets. For each sequence, or triplet, the regression line was computed between the SBP and PI values that form the sequence, and the slope of this line was taken as an index of BRS (ms/mmHg) (Di Renzo et al., 1997). In order to eliminate very extreme scores (i.e., artifacts) from the BRS data, upper and lower limits were defined using 2 SD from the mean BRS value for all minutes analyzed for a particular subject. All BRS data were then analyzed again on a minute-by-minute basis, and all triplets falling outside the
upper and lower limit were deleted. This procedure resulted in deleting a total of 194 triplets, or 2.1% of the total number. Thus, almost 98% of the data were retained, despite the deletion of extreme values.

Because rate and depth of breathing affects values of HF (Grossman, Karemaker & Wieling, 1991), respiratory rate and amplitude were measured for possible use as covariates in analyses of HF. Respiration signals were derived from impedance cardiography signals using impedance pneumography (Ernst, 1999). The impedance signals were decimated to 250 Hz. The respiratory signal was then linear detrended and band pass filtered using the same interpolated finite impulse response filter described above. The respiratory power spectrum was calculated using a Fast Fourier Transform and scaled to units$^2$/Hz. The mean respiratory rate and amplitude were calculated from the respiratory power spectrum using waveform moment analysis (Cacioppo & Dorffman, 1987). Following data analysis, it was apparent that values of respiratory amplitude exhibited unrealistically extreme inter-subject variability. That is, a subset of participants had respiration amplitude values many times those of the rest of the sample. Therefore, respiration amplitude was not used in any analyses.

Lipid Measures

Blood samples for lipid analyses were collected during the last 2 minutes of the baseline, stress task, and recovery periods. Blood samples were centrifuged on site in a refrigerated (4 °C) centrifuge (IEC Centra GP8R). Serum was stored at -80 °C in an ultrafreezer (Revco Scientific Inc. model ULT 2186-7-A12, Asheville, N.C.) for later analysis. All lipid measures were analyzed in the laboratory of the Ohio State University
Medical Center. Total cholesterol was determined by enzymatic methods on a Beckman CX4 (Wybenga, Pileggi, Dirstine & DiGiorgio, 1970). Triglycerides, blanked for free glycerol, was determined using Beckman reagent (Buccolo & David, 1973). High density lipoprotein cholesterol (HDL-c) was determined following precipitation of lower density lipoproteins with heparin-MnCl₂ and dextran sulfate (Gidez, Miller, Burnstein, Slagle & Eder, 1982; Warnick & Albers, 1978). LDL-c was calculated using the Friedewald equation (Friedewald, Levy & Fredrickson, 1972).

Hematologic Measures

Hematocrit and hemoglobin were measured for each blood sample using a Coulter quantitative automated hematology analyzer (Miami, FL 33196), so that the extent of changes in plasma volume could be calculated. Change in plasma volume was calculated from hematocrit and hemoglobin values using the equation recommended by Dill and Costill (Dill & Costill, 1974). Calculating change in plasma volume allowed lipid values during the stressors to be adjusted where changes in lipid values are observed, in order to determine if this mechanism alone explains the changes (Stoney & West, 1997).

Procedure

The experimenter contacted each prospective participant and described the purpose and procedure of the study to him or her. Persons agreeing to participate were instructed not to eat or drink anything except water for 12 hours prior to their appointment, and to avoid exercise the morning before they participated.
All testing took place in The General Clinical Research Center at The Ohio State University Medical Center on weekdays between 7:30 a.m. and 1:30 p.m. Written informed consent was obtained prior to participation in the study protocol. Participants first completed psychosocial questionnaires and had their height and weight measured by the investigators. The four mylar bands for the impedance cardiography and the three electrodes for the EKG were then applied. Participants then sat in a comfortable chair and the arterial tonometry monitor cuff was applied to the wrist of their non-dominant arm in the manner specified by the manufacturer’s instructions. An indwelling (19-gauge) catheter was inserted into the antecubital fossa of the dominant arm for the collection of blood samples.

Following instrumentation, participants relaxed for 30 minutes in order to acclimate to the experimental environment, during which they listened to music on headphones. Immediately following the acclimation period, participants completed a 10-minute baseline followed by the laboratory stress tasks in a counterbalanced order. The stress tasks were separated by a 10-minute resting recovery period, and an additional 10-minute resting recovery period followed the final stress task. Following every resting period, participants completed the PANAS and an impressions questionnaire (included in Appendix B). Figure 1 depicts the timeline for the laboratory protocol.
Order 1: Speech Task First

<table>
<thead>
<tr>
<th>Acclimation</th>
<th>Baseline</th>
<th>Speech</th>
<th>Rest</th>
<th>Cold</th>
<th>Rest</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30'</td>
<td>10'</td>
<td>5'</td>
<td>10'</td>
<td>3'</td>
<td>10'</td>
</tr>
</tbody>
</table>

Order 2: Forehead Cold Pressor Task First

<table>
<thead>
<tr>
<th>Acclimation</th>
<th>Baseline</th>
<th>Cold</th>
<th>Rest</th>
<th>Speech</th>
<th>Rest</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>30'</td>
<td>10'</td>
<td>3'</td>
<td>10'</td>
<td>5'</td>
<td>10'</td>
</tr>
</tbody>
</table>

Figure 1: Timeline for Laboratory Protocol

Analytic strategy

To increase reliability, baseline, stress task, and recovery values for each physiological measure were calculated by averaging the last five minutes of each phase of the laboratory protocol. Reactivity (change) scores were calculated by subtracting the baseline value from each stress task value. All omnibus tests were conducted at the .05 level of significance, and all follow-up tests involving comparisons of mean values were corrected for family-wise error rates using Tukey’s Honestly Significant Difference.
(HSD) tests (Kirk, 1968). All omnibus tests involving more than 2 levels of a repeated factor were corrected for possible violations of the sphericity assumption using the Greenhouse-Geisser correction for degrees of freedom.

All dependent variables were examined for violations of the normality assumption, resulting in the application of log transformations to BRS, HDL-c, and triglyceride values for all subsequent analyses. Non-transformed (raw) values of these variables are reported in the tables and figures for ease of interpretation. Order effects were examined using 2 (order) x 2 (phase) mixed model analyses of variance (ANOVAs) for each dependent variable. A significant interaction of order and phase was observed for total cholesterol $F(4,146) = 3.51, p < 0.05$. Simple main effects of phase for each order revealed that total cholesterol increased during the speech task, but only when the speech task was presented first. The order term was included in all further analyses involving repeated total cholesterol measurements.

Groups were defined as high (High) or low (Low) in depressed mood based on median splits of BDI scores. BDI means, standard deviations, and ranges by gender and BDI median split are presented in Table 3. Visual inspection of the distributions of BDI scores seemed to indicate that a larger range and more variability was present among women’s BDI scores. Therefore, an ANOVA testing the statistical assumption of homogeneous variances was conducted, confirming that women’s BDI scores exhibited more variability, $F(28,20) = 2.45, p = .042$. However, men and women had similar BDI scores, $t(48) = 1.1, p = .29$. 

47
Variable | Men | Women
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>High</td>
<td>Low</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>BDI (M, SD)*</td>
<td>7.3 (2.9)</td>
<td>1.0 (1.0)</td>
<td>9.4 (5.4)</td>
<td>0.6 (0.8)</td>
</tr>
<tr>
<td>BDI Range</td>
<td>3-10</td>
<td>0-2</td>
<td>3-22</td>
<td>0-2</td>
</tr>
</tbody>
</table>

Table 3: *BDI Means and Ranges by Gender and BDI Median Split*

*Note:* BDI = Beck Depression Inventory. * Men’s and women’s average BDI scores were not significantly different, *t*(48) = 1.1, *p* = .29.

A series of 2 (Depressed Mood: High vs. Low) x 2 (Gender: Women vs. Men) ANOVAs were conducted to examine possible group differences in age and physical characteristics (i.e., height, weight, and body mass index). A series of independent t-tests were conducted to determine if men and women differed with respect to scores on the psychological questionnaires. A series of 2 (Depressed Mood: High vs. Low) x 2 (Gender: Women vs. men) ANOVAs were conducted to examine effects of depressed mood and gender on baseline values of each physiological variable, which included a test of the first hypothesis (i.e., that depression would be related to BRS at baseline). Next, a series of oneway ANOVAs (Time: Baseline, Speech task, Recovery, Forehead cold pressor task, Recovery) was conducted for every physiological variable to determine if the stress tasks elicited cardiovascular reactivity. As is typical in our
laboratory, analyses demonstrating lipid reactivity to the stress tasks were repeated using values adjusted for changes in plasma volume in order to determine if this physiological mechanism alone explains the changes (Stoney & West, 1997). Next, a series of 2 (Depressed Mood: High vs Low) x 2 (Gender: Women vs men) x 2 (Stress Task: Speech vs Forehead cold pressor) mixed-model ANOVAs were conducted to determine if depression was related to the magnitude of changes in BRS, HF, and PEP, thus testing hypotheses 2-4. To test the fifth hypothesis, a series of Pearson product-moment correlations were conducted to examine the relationships between measures of depression, anxiety, and social support. In addition, measures of anxiety and social support were used as covariates in those analyses showing a relationship between depression and BRS, HF, and PEP to determine whether or not anxiety and social support could also account for the relationship. To explore possible relationships between depression and cholesterol, depression was correlated with baseline values of total cholesterol, HDL-c, LDL-c, and triglycerides. Finally, measures of total cholesterol, HDL-c, and LDL-c were correlated with values of HF and BRS, as well as HF, PEP, and BRS changes during stress. These correlations included a test of the seventh hypothesis, that LDL-c would exhibit an inverse relationship with HF and BRS.
RESULTS

*Age and physical characteristics.* Results of a series of 2 (Depressed Mood) x 2 (Gender) ANOVAs for age and physical characteristics revealed expected significant gender effects for weight ($F(1,46) = 22.7 \ p = .0001$) and height ($F(1,46) = 37.9 \ p < .0001$). Inspection of means indicated that men were heavier ($M = 75.5, SD = 9.4$) and taller ($M = 175, SD = 7$) than women ($M = 64.0, SD = 7.6$ and $M = 162, SD = 8$, respectively). No significant effects were observed for age or BMI (all $F$'s < 1.4, all $p$'s > .25). Age and physical characteristics for each group are presented in Table 4.
Table 4: Physical Characteristics by Gender and BDI Median Split

*Significant gender effects, $p < .05$. 

Baseline values. Results of a series of 2 (Depressed Mood) x 2 (Gender )

ANOVA on baseline values of non-invasive cardiovascular measures, lipid measures, and hematological measures revealed significant gender effects for heart rate ($F(1,45) = 4.87, p = .03$), SBP ($F(1,45) = 8.66, p = .005$), HDL-c ($F(1, 43) = 8.42, p = .006$), hemoglobin ($F(1, 39) = 52.1, p < .0001$), and hematocrit ($F(1, 39) = 53.9, p < .0001$). Inspection of means indicated that men had lower heart rates ($M = 67.1, SD = 9.3$) than women ($M = 74.4, SD = 12.0$), that men had higher SBP ($M = 121.5, SD = 9.7$) than women ($M = 112.6, SD = 10.2$), that men had lower HDL-c ($M = 42.6, SD = 10.6$) than women ($M = 53.0, SD = 14.7$), that men had higher hemoglobin ($M = 14.8, SD = 0.8$) than women ($M = 12.8, SD = 0.9$), and that men had higher hematocrit ($M = 42.2, SD = 2.1$) than women ($M = 36.9, SD = 2.3$).
In addition to the gender effects, a significant effect of depressed mood was noted for PEP ($F(1,43) = 5.27, p = .027$). Inspection of means revealed that participants low in depressed mood had lower PEP ($M = 128.03, SD = 14.4$) than those high depressed mood ($M = 138.3, SD = 16.4$). No other significant effects of gender or depressed mood on baseline values were noted (all $F$'s $< 1.57$, all $p$'s $> .21$). Average baseline values of non-invasive physiological measures for each group are presented in Table 5. Baseline values of lipid and hematological measures for each group are presented in Table 6.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men</th>
<th></th>
<th>Women</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>High</td>
<td>Low</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Heart Rate (bpm)*</td>
<td>68.7 ± 9.4</td>
<td>65.9 ± 9.4</td>
<td>74.4 ± 13.8</td>
<td>74.5 ± 10.4</td>
</tr>
<tr>
<td>SBP (mmHg)*</td>
<td>118.9 ± 7.1</td>
<td>123.6 ± 11.3</td>
<td>111.4 ± 10.5</td>
<td>113.8 ± 10.2</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>58.7 ± 6.9</td>
<td>61.5 ± 8.7</td>
<td>64.0 ± 6.7</td>
<td>61.6 ± 7.4</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>78.8 ± 5.8</td>
<td>82.2 ± 9.2</td>
<td>79.8 ± 7.6</td>
<td>79.0 ± 7.5</td>
</tr>
<tr>
<td>HF (ln[ms²])</td>
<td>7.0 ± 1.1</td>
<td>6.4 ± 1.3</td>
<td>6.3 ± 1.3</td>
<td>6.4 ± 1.0</td>
</tr>
<tr>
<td>PEP (ms)**</td>
<td>140.44 ± 9.6</td>
<td>128.2 ± 12.4</td>
<td>136.9 ± 20.2</td>
<td>127.9 ± 16.5</td>
</tr>
<tr>
<td>BRS (ms/mmHg)</td>
<td>15.1 ± 8.3</td>
<td>13.6 ± 8.4</td>
<td>14.6 ± 10.3</td>
<td>16.8 ± 9.6</td>
</tr>
</tbody>
</table>

Table 5: Baseline Noninvasive Physiological Measures by Gender and BDI Median Split

*Significant gender effects, $p < .05$. **Significant depressed mood effect, $p < .05$. 

Note: Values represent $M ± SD$. SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; HF = high frequency heart rate variability; PEP = pre-ejection period; BRS = baroreceptor sensitivity.
<table>
<thead>
<tr>
<th>Variable</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Total cholesterol (mg/dl)</td>
<td>155.0 ± 20.7</td>
<td>149.2 ± 19.9</td>
</tr>
<tr>
<td>LDL-c (mg/dl)</td>
<td>96.3 ± 20.7</td>
<td>91.1 ± 21.3</td>
</tr>
<tr>
<td>HDL-c (mg/dl)*</td>
<td>41.4 ± 9.0</td>
<td>43.4 ± 11.9</td>
</tr>
<tr>
<td>Triglycerides (mg/dl)</td>
<td>86.5 ± 45.9</td>
<td>73.4 ± 21.5</td>
</tr>
<tr>
<td>HGB*</td>
<td>14.8 ± 0.8</td>
<td>14.8 ± 0.8</td>
</tr>
<tr>
<td>HCT*</td>
<td>42.2 ± 2.4</td>
<td>42.2 ± 2.1</td>
</tr>
</tbody>
</table>

Table 6: Baseline Lipid and Hematological Measures by Gender and BDI Median Split

Note: Values represent $M \pm SD$. LDL-c = low density lipoprotein cholesterol; HDL-c = high density lipoprotein cholesterol; HGB = hemoglobin; HCT = hematocrit.
*Significant gender effects, $p < .05$.

The 2 (Depressed Mood) x 2 (Gender) ANOVAs on baseline values of BRS tested the first hypothesis of the study that those high in depressed mood would have lower baseline values of BRS. The ANOVA results are presented in Table 7. Contrary to expectations, no main effect or interaction for depression was found.
Table 7 Analysis of Variance for Effect of Depression and Gender on Baseline BRS

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>$F$</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>BDI Median Split (Depression)</td>
<td>1</td>
<td>0.12</td>
<td>.73</td>
</tr>
<tr>
<td>Gender</td>
<td>1</td>
<td>0.08</td>
<td>.78</td>
</tr>
<tr>
<td>Depression x Gender</td>
<td>1</td>
<td>0.82</td>
<td>.37</td>
</tr>
<tr>
<td>Error</td>
<td>44</td>
<td>(0.38)</td>
<td></td>
</tr>
</tbody>
</table>

Note: Value in parentheses represents mean square errors.

Psychosocial values. A series of independent t-tests was used to examine possible gender differences in psychosocial variables. As shown in Table 8, men had lower PSS scores than women. No other gender differences were observed for psychosocial variables.
<table>
<thead>
<tr>
<th>Variable</th>
<th>Men</th>
<th>Women</th>
<th>t (df)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CESD</td>
<td>14.1 ± 12.2</td>
<td>17.1 ± 14.3</td>
<td>0.7 (48)</td>
</tr>
<tr>
<td>BDI</td>
<td>3.7 ± 3.8</td>
<td>5.2 ± 5.9</td>
<td>1.1 (48)</td>
</tr>
<tr>
<td>ISEL</td>
<td>34.4 ± 4.4</td>
<td>35.1 ± 4.3</td>
<td>0.6 (47)</td>
</tr>
<tr>
<td>Marlowe Crowne</td>
<td>15.4 ± 5.5</td>
<td>16.7 ± 4.9</td>
<td>1.0 (47)</td>
</tr>
<tr>
<td>PSS</td>
<td>23.1 ± 5.7</td>
<td>27.8 ± 9.0</td>
<td>2.2* (47)</td>
</tr>
<tr>
<td>PANAS-Negative</td>
<td>18.2 ± 8.1</td>
<td>17.5 ± 7.8</td>
<td>0.3 (48)</td>
</tr>
<tr>
<td>PANAS-Positive</td>
<td>11.6 ± 2.0</td>
<td>12.6 ± 3.8</td>
<td>1.0 (48)</td>
</tr>
<tr>
<td>State Anxiety</td>
<td>33.6 ± 7.7</td>
<td>36.2 ± 11.1</td>
<td>0.9 (46)</td>
</tr>
<tr>
<td>Trait Anxiety</td>
<td>34.0 ± 10.8</td>
<td>38.8 ± 12.1</td>
<td>1.4 (45)</td>
</tr>
<tr>
<td>Anger-In</td>
<td>16.5 ± 4.8</td>
<td>17.3 ± 4.8</td>
<td>0.5 (47)</td>
</tr>
<tr>
<td>Anger-Out</td>
<td>14.9 ± 4.3</td>
<td>15.4 ± 4.1</td>
<td>0.4 (47)</td>
</tr>
<tr>
<td>Anger-Control</td>
<td>25.5 ± 5.2</td>
<td>23.9 ± 4.3</td>
<td>1.2 (47)</td>
</tr>
</tbody>
</table>

Table 8: Psychosocial Data for Men and Women

Note: CESD values reflect scores from prescreen administration. PANAS values reflect scores from the PANAS administration after the resting baseline. *p < .05

Physiological responses during stress

Blood pressure and heart rate. In order to verify that the stress tasks elicited significant cardiovascular reactivity, a series of oneway repeated measures ANOVAs were performed on SBP, DBP, MAP, and HR values during each phase of the laboratory protocol. As expected, significant effects were found for all variables, all F's > 16.58, all p's < .0001, all $\epsilon > .58$. Table 9 presents values of SBP, DBP, MAP, and HR at each phase of the laboratory protocol. Post-hoc comparisons using Tukey's HSD revealed that SBP and HR increased from baseline values during the speech task, but did not
significantly change during the forehead cold pressor task. For DBP and MAP, *post-hoc* comparisons revealed significant increases from baseline during both the speech and cold-pressor tasks.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>Speech</th>
<th>Recovery</th>
<th>Cold Pressor</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP (mmHg)</td>
<td>116.2±10.9</td>
<td>129.6±17.2</td>
<td>119.8±15.0</td>
<td>120.7±15.0</td>
<td>115.8±13.6</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>61.7±7.8</td>
<td>73.5±10.9</td>
<td>64.1±10.6</td>
<td>66.0±12.3</td>
<td>63.8±10.7</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>79.9±7.6</td>
<td>91.2±11.9</td>
<td>82.7±11.0</td>
<td>84.3±12.5</td>
<td>81.1±11.0</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>71.2±11.5</td>
<td>84.6±11.8</td>
<td>72.4±11.1</td>
<td>70.2±10.9</td>
<td>74.1±10.7</td>
</tr>
</tbody>
</table>

Table 9: *Mean values of SBP, DBP, MAP, and HR at each phase of the laboratory protocol*

*Note:* Values represent M ± SD. Dissimilar superscripts indicate significantly different values, *p* < .05. SBP = systolic blood pressure: DBP = diastolic blood pressure: MAP = mean arterial pressure: HR = heart rate.

To examine possible relationships between depression, gender and cardiovascular reactivity, a series of 2 (Depressed Mood) x 2 (Gender) x 2 (Stress Task) mixed-model ANOVAs were conducted for SBP, DBP, MAP, and HR change scores. No significant main effects or interactions involving depressed mood or gender were observed, all *F*'s < 2.22, all *p*'s > .14. Significant main effects of stress task revealed that blood pressure and heart rate changes were larger during the speech task than during the forehead cold pressor task, all *F*'s > 20.5, all *p*'s < .0001.

*Autonomic measures.* Results of a series of oneway ANOVAs on HF, PEP, and BRS verified that the stress tasks elicited significant changes in cardiac autonomic
control, all $F$'s $> 10.87$, all $p$'s $< .0001$. Table 10 presents values of HF, PEP, and BRS at each phase of the laboratory protocol. Post-hoc testing revealed that HF decreased significantly during the speech task, but did not significantly change during the forehead cold pressor task. Similarly, BRS decreased during the speech task but did not significantly change during the forehead cold pressor task, whereas PEP decreased during the speech task and increased during the forehead cold pressor task.

Table 10: Mean values of HF, PEP, and BRS at each phase of the laboratory protocol

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>Speech</th>
<th>Recovery</th>
<th>Cold Pressor</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>HF (ln[ms$^2$])</td>
<td>6.51$^a \pm 1.18$</td>
<td>5.96$^b \pm 1.17$</td>
<td>6.22$^c \pm 1.35$</td>
<td>5.43$^d \pm 1.13$</td>
<td>6.16$^e \pm 1.13$</td>
</tr>
<tr>
<td>PEP (ms)</td>
<td>132.8$^a \pm 16.2$</td>
<td>121.5$^b \pm 16.9$</td>
<td>131.6$^c \pm 15.0$</td>
<td>135.6$^d \pm 16.2$</td>
<td>133.1$^e \pm 16.5$</td>
</tr>
<tr>
<td>BRS (ms/mmHg)</td>
<td>15.27$^a \pm 9.5$</td>
<td>11.24$^b \pm 7.6$</td>
<td>15.5$^c \pm 11.2$</td>
<td>18.0$^d \pm 11.6$</td>
<td>14.6$^e \pm 9.8$</td>
</tr>
</tbody>
</table>

Note: Values represent $M \pm SD$. Dissimilar superscripts indicate significantly different values, $p < .05$. HF = high frequency heart rate variability; PEP$^*$ = pre-ejection period; BRS = baroreceptor sensitivity.

To test the main hypotheses of the study, namely that depressed mood would be related to the magnitude of changes in cardiac autonomic control, a series of 2 (Depressed Mood) x 2 (Gender) x 2 (Stress Task) mixed-model ANOVAs were conducted on changes in HF, PEP, and BRS.

The ANOVA results for changes in HF are presented in Table 11. The ANOVA results for changes in PEP are presented in Table 12. The ANOVA results for changes in
BRS are presented in Table 13. Contrary to expectations, no effect of depressed mood was observed for changes in HF and PEP. For changes in BRS, depressed mood interacted with stress task, $F(1,39) = 5.68, p = .02$. The analysis of HF change scores was not changed when repeated with changes in respiration rate used as covariates. Simple main effects of depression at each stress task and inspection of means indicated that participants high in depressed mood exhibited smaller decreases in BRS during the speech task than participants low in depressed mood, $F(1,39) = 5.19, p = 0.03$. In contrast, depressed mood was unrelated to changes in BRS during the forehead cold pressor task, $F(1,39) =1.56, p = 0.22$. Figures 2-4 depict changes in HF, PEP, and BRS during the stress tasks by depressed mood.
<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>F</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Between Subjects</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI Median Split (Depression)</td>
<td>1</td>
<td>0.08</td>
<td>.92</td>
</tr>
<tr>
<td>Gender</td>
<td>1</td>
<td>0.17</td>
<td>.67</td>
</tr>
<tr>
<td>Depression x Gender</td>
<td>1</td>
<td>0.31</td>
<td>.58</td>
</tr>
<tr>
<td>Error</td>
<td>44</td>
<td>(0.80)</td>
<td></td>
</tr>
<tr>
<td><strong>Within Subjects</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress Task</td>
<td>1</td>
<td>18.23</td>
<td>.0001</td>
</tr>
<tr>
<td>Stress Task x Depression</td>
<td>1</td>
<td>0.8</td>
<td>.38</td>
</tr>
<tr>
<td>Stress Task x Gender</td>
<td>1</td>
<td>0.23</td>
<td>.63</td>
</tr>
<tr>
<td>Stress Task x Depression x Gender</td>
<td>1</td>
<td>2.85</td>
<td>.09</td>
</tr>
<tr>
<td>Error (Stress Task)</td>
<td>44</td>
<td>(0.31)</td>
<td></td>
</tr>
</tbody>
</table>

Table 11: *Analysis of Variance for Effect of Depression and Gender on HF Changes During Stress*

*Note:* Values in parentheses represent mean square errors.
Table 12: Analysis of Variance for Effect of Depression and Gender on PEP Changes During Stress

<table>
<thead>
<tr>
<th>Source</th>
<th>df</th>
<th>( F )</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Between Subjects</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI Median Split (Depression)</td>
<td>1</td>
<td>0.52</td>
<td>.48</td>
</tr>
<tr>
<td>Gender</td>
<td>1</td>
<td>0.82</td>
<td>.37</td>
</tr>
<tr>
<td>Depression x Gender</td>
<td>1</td>
<td>0.24</td>
<td>.84</td>
</tr>
<tr>
<td>Error</td>
<td>42</td>
<td>(54.24)</td>
<td></td>
</tr>
<tr>
<td><strong>Within Subjects</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress Task</td>
<td>1</td>
<td>130.43</td>
<td>.0001</td>
</tr>
<tr>
<td>Stress Task x Depression</td>
<td>1</td>
<td>0.88</td>
<td>.75</td>
</tr>
<tr>
<td>Stress Task x Gender</td>
<td>1</td>
<td>0.10</td>
<td>.35</td>
</tr>
<tr>
<td>Stress Task x Depression x Gender</td>
<td>1</td>
<td>0.52</td>
<td>.47</td>
</tr>
<tr>
<td>Error (Stress Task)</td>
<td>44</td>
<td>(35.32)</td>
<td></td>
</tr>
</tbody>
</table>

*Note: Values in parentheses represent mean square errors.*
Table 13: *Analysis of Variance for Effect of Depression and Gender on BRS Changes During Stress*

*Note:* Values in parentheses represent mean square errors.
Figure 2: Changes in HF by Depressed Mood

Figure 3: Changes in PEP by Depressed Mood
Exploratory analyses of Depression and HF, PEP, and BRS. Because the median split occurred at a BDI score of 3, the mean BDI scores in the high-depressed mood group were quite low. Exploratory correlation analyses were used in addition to the planned ANOVA analyses. Because more variability was observed among women's BDI scores, exploratory correlations using baseline values were conducted for men and women separately, as well as for the combined sample. Table 14 presents correlations of BDI with baseline values of blood pressure, heart rate, HF, PEP, and BRS. Results of these analyses mirrored the ANOVAs. Specifically, BDI score was positively correlated with PEP, and BDI score was not correlated with any other variable at baseline. All correlations were similar for men, women, and the combined sample. Table 15 presents correlations of BDI with cardiovascular reactivity during the speech and forehead cold pressor stress task.
pressor stress tasks. For HF, PEP, and BRS, the results of these analyses mirror the results of the ANOVAs. Specifically, a marginally significant positive correlation between BDI scores and BRS changes during the speech task was observed, indicating that individuals higher in depression scores had smaller decreases in BRS.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Men</th>
<th>Women</th>
<th>Combined Sample</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>BDI</td>
<td>p</td>
<td>BDI</td>
</tr>
<tr>
<td>SBP</td>
<td>-.20</td>
<td>.39</td>
<td>-.11</td>
</tr>
<tr>
<td>DBP</td>
<td>-.15</td>
<td>.52</td>
<td>.13</td>
</tr>
<tr>
<td>MAP</td>
<td>-.18</td>
<td>.44</td>
<td>.03</td>
</tr>
<tr>
<td>HR</td>
<td>-.01</td>
<td>.97</td>
<td>-.8</td>
</tr>
<tr>
<td>HF</td>
<td>.19</td>
<td>.40</td>
<td>.09</td>
</tr>
<tr>
<td>PEP</td>
<td>.53</td>
<td>.01</td>
<td>.47</td>
</tr>
<tr>
<td>BRS</td>
<td>.19</td>
<td>.43</td>
<td>.01</td>
</tr>
</tbody>
</table>

Table 14: Pearson Product Moment Correlation Coefficients of BDI with Baseline Values of SBP, DBP, MAP, HR, HF, PEP, and BRS for Men, Women, and the Combined Sample

Note: SBP = systolic blood pressure: DBP = diastolic blood pressure: MAP = mean arterial pressure: HF = high frequency heart rate variability: PEP = pre-ejection period: BRS = baroreceptor sensitivity. Significant correlations are bolded.
Table 15: *Pearson Product Moment Correlation Coefficients of BDI with Changes During Stress Tasks*

*Note:* SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; HF = high frequency heart rate variability; PEP = pre-ejection period; BRS = baroreceptor sensitivity. Significant correlations are bolded.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Speech Task</th>
<th></th>
<th>Forehead Cold Pressor Task</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
<td>p</td>
<td>r</td>
<td>p</td>
</tr>
<tr>
<td>Change in SBP</td>
<td>-.18</td>
<td>.21</td>
<td>.10</td>
<td>.50</td>
</tr>
<tr>
<td>Change in DBP</td>
<td>-.34</td>
<td>.02</td>
<td>.08</td>
<td>.55</td>
</tr>
<tr>
<td>Change in MAP</td>
<td>-.30</td>
<td>.04</td>
<td>.09</td>
<td>.52</td>
</tr>
<tr>
<td>Change in HR</td>
<td>.09</td>
<td>.55</td>
<td>.04</td>
<td>.78</td>
</tr>
<tr>
<td>Change in HF</td>
<td>.03</td>
<td>.86</td>
<td>.08</td>
<td>.56</td>
</tr>
<tr>
<td>Change in PEP</td>
<td>.11</td>
<td>.48</td>
<td>.11</td>
<td>.47</td>
</tr>
<tr>
<td>Change in BRS</td>
<td>.27</td>
<td>.07</td>
<td>-.26</td>
<td>.09</td>
</tr>
</tbody>
</table>

*Lipid measures.* In order to determine whether or not the stress tasks elicited significant lipid reactivity, a series of oneway ANOVAs were performed using total cholesterol, HDL-c, LDL-c, and triglycerides. Table 16 presents values of total cholesterol, HDL-c, LDL-c, and triglycerides at each phase of the laboratory protocol. As previously noted, the order effect was retained in the total cholesterol analysis. A significant interaction of order and phase was observed for total cholesterol $F(4,146) = 3.51, p = 0.01, \varepsilon = .86$. Simple main effects of phase for each order revealed that total cholesterol increased during the speech task, but only when the speech task was presented first. No other lipid measure changed during the laboratory protocol, all $F$'s < 1.62, all $p$'s > .17. When the total cholesterol analysis was repeated using values
corrected for plasma volume shift, the order by phase interaction was no longer significant, \( F = 2.22, p = .09 \). Thus, changes in total cholesterol during the speech task did not exceed what can be accounted for by plasma volume shift.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>Speech</th>
<th>Recovery</th>
<th>Cold Pressor</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC (mg/dl)</td>
<td>156.9 ± 29.4</td>
<td>159.0 ± 29.8</td>
<td>157.8 ± 29.4</td>
<td>156.9 ± 27.8</td>
<td>157.3 ± 29.2</td>
</tr>
<tr>
<td>HDL-c (mg/dl)</td>
<td>49.4 ± 14.6</td>
<td>49.8 ± 13.9</td>
<td>49.9 ± 14.3</td>
<td>49.8 ± 13.7</td>
<td>49.6 ± 14.6</td>
</tr>
<tr>
<td>LDL-c (mg/dl)</td>
<td>92.4 ± 25.3</td>
<td>93.9 ± 25.9</td>
<td>92.9 ± 24.8</td>
<td>92.2 ± 23.4</td>
<td>92.8 ± 24.7</td>
</tr>
<tr>
<td>Trigs (mg/dl)</td>
<td>75.9 ± 55.3</td>
<td>77.0 ± 54.2</td>
<td>75.0 ± 52.4</td>
<td>74.7 ± 51.9</td>
<td>74.7 ± 51.8</td>
</tr>
</tbody>
</table>

Table 16: Mean values of Total Cholesterol, HDL-c, LDL-c, and Triglycerides at Each Phase of the Laboratory Protocol

Note: TC = total cholesterol; LDL-c = low density lipoprotein cholesterol; HDL-c = high density lipoprotein cholesterol; Trigs = Triglycerides.

Relationship between depression, social support, and anxiety. To test the fifth hypothesis of the study, a series of correlation analyses were conducted to examine the relationships between measures of depression, anxiety, and social support. These correlations are reported in Table 17. As predicted, depression was negatively correlated with social support \( (r = -.72) \) and positively correlated with trait anxiety \( (r = .84) \).
Table 17: Intercorrelations of Psychosocial Measures

<table>
<thead>
<tr>
<th>Variable</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. CESD</td>
<td>.75</td>
<td>-.64</td>
<td>.62</td>
<td>.49</td>
<td>.75</td>
<td></td>
</tr>
<tr>
<td>2. BDI</td>
<td>-.72</td>
<td>.69</td>
<td>.54</td>
<td>.83</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. ISEL</td>
<td>-.58</td>
<td>-.54</td>
<td>-.77</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. TMAS</td>
<td></td>
<td>.49</td>
<td>.77</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. State Anxiety</td>
<td></td>
<td></td>
<td></td>
<td>.66</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Trait Anxiety</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: CESD scores are from a prescreening administration. All p’s < .00005

To determine if the relationship between depression and baseline values of PEP could be accounted for by anxiety and/or social support, measures of anxiety and social support were used as covariates in a series of 2 (Depression) x 2 (Gender) ANOVAs. When state anxiety, trait anxiety, or social support was used as a covariate, depression was no longer related to PEP at baseline, all F’s < 2.23, all p’s > .14. Because the range of BDI scores was small, depression, state and trait anxiety, and social support were subsequently used in a multiple linear regression predicting PEP values at baseline. The bivariate correlations were significant at p < .05 for each of the predictor variables: depression (r = .47), state anxiety (r = .32), trait anxiety (r = .35), and social support (r = -.48). The beta weights of the multiple linear regression for these variables predicting PEP values at baseline are reported in Table 18. Although the equation containing these four variables accounted for 28% of the variance in PEP values at baseline, F(4,39) = 3.85, p < .01, no variable exhibited a significant beta weight. Collectively, these analyses
suggest that the relationship between depression and PEP values at baseline was not unique to depression and can also be partially accounted for by social support and anxiety.

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Beta Weights</th>
<th>Beta</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Depression</td>
<td>.443</td>
<td></td>
<td>1.7</td>
<td>.09</td>
</tr>
<tr>
<td>2. State Anxiety</td>
<td>.14</td>
<td></td>
<td>0.8</td>
<td>.43</td>
</tr>
<tr>
<td>3. Trait Anxiety</td>
<td>-.40</td>
<td></td>
<td>1.3</td>
<td>.19</td>
</tr>
<tr>
<td>4. Social Support</td>
<td>-.36</td>
<td></td>
<td>1.6</td>
<td>.12</td>
</tr>
</tbody>
</table>

Table 18: Beta Weights Obtained in Multiple Linear Regression Analyses Predicting PEP Values at Baseline.

To determine if the relationship observed between depression, stress task, and changes in BRS could be accounted for by anxiety and/or social support, measures of anxiety and social support were used as covariates in a series of 2 (depression) x 2 (gender) x 2 (stress task) mixed model ANOVAs on changes in BRS. When state anxiety was used as a covariate, the significant interaction of depressed mood and stress task remained, $F(1,34) = 5.86, p = .02$. When trait anxiety or social support was used as a covariate, depression no longer interacted with stress task, $F$'s $< 2.23, p$'s $> .14$. These results suggest that the interaction of depression and stress task predicting changes in BRS cannot be accounted for by state anxiety, but can be partially accounted for by trait anxiety and social support.
Exploratory analyses of anxiety, social support and HF, PEP, and BRS. Because depression was unrelated to HF and BRS, exploratory correlation analyses were used in addition to the planned analyses to determine if anxiety or social support instead were related to HF, PEP, or BRS. Table 19 presents correlations of state anxiety, trait anxiety, and social support with baseline values of blood pressure, heart rate, HF, PEP, and BRS. Results of these analyses indicate that anxiety and social support were largely unrelated to baseline values, with the exception of the relationships with PEP already reported in the regression analysis above. However, state anxiety was negatively correlated with SBP, indicating that individuals with higher state anxiety scores had lower SBP. Tables 20 and 21 present correlations of these variables with cardiovascular reactivity during the speech task and cold-pressor task, respectively. These analyses largely mirror the results of the exploratory correlational analyses using depression, as state anxiety, trait anxiety, and social support were rarely correlated with cardiovascular reactivity. However, a few exceptions were observed. During the speech task, a non-significant trend was observed for trait anxiety to be related to DBP changes ($p = .08$). In addition, trends were observed for social support to be related to changes in DBP ($p = .06$) and MAP ($p = .09$), and a trend was observed for social support to be inversely related to BRS changes ($p = .09$). During the forehead cold pressor task, a trend was observed for state anxiety to be related to changes in MAP ($p = .07$), and state anxiety was related to changes in SBP ($p = .03$).
<table>
<thead>
<tr>
<th>Variable</th>
<th>State Anxiety</th>
<th></th>
<th></th>
<th>Trait Anxiety</th>
<th></th>
<th></th>
<th></th>
<th>ISEL</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
<td>p</td>
<td>r</td>
<td>p</td>
<td>r</td>
<td>p</td>
<td>r</td>
<td>p</td>
<td>r</td>
<td>p</td>
</tr>
<tr>
<td>SBP</td>
<td>-.29</td>
<td>.04</td>
<td>-.22</td>
<td>.14</td>
<td>.13</td>
<td>.37</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DBP</td>
<td>-.05</td>
<td>.74</td>
<td>.05</td>
<td>.74</td>
<td>.01</td>
<td>.99</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MAP</td>
<td>-.17</td>
<td>.25</td>
<td>-.07</td>
<td>.63</td>
<td>.07</td>
<td>.66</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR</td>
<td>-.04</td>
<td>.78</td>
<td>.12</td>
<td>.44</td>
<td>-.07</td>
<td>.66</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HF</td>
<td>.10</td>
<td>.49</td>
<td>.02</td>
<td>.85</td>
<td>-.06</td>
<td>.71</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PEP</td>
<td>.32</td>
<td>.03</td>
<td>.35</td>
<td>.02</td>
<td>-.48</td>
<td>.01</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BRS</td>
<td>.09</td>
<td>.56</td>
<td>.03</td>
<td>.85</td>
<td>.04</td>
<td>.81</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 19: Pearson Product Moment Correlation Coefficients of State Anxiety, Trait Anxiety, and Social Support with Baseline Values of SBP, DBP, MAP, HR, HF, PEP, and BRS

Note: SBP = systolic blood pressure: DBP = diastolic blood pressure: MAP = mean arterial pressure: HF = high frequency heart rate variability: PEP = pre-ejection period: BRS = baroreceptor sensitivity. Significant correlations are bolded.
<table>
<thead>
<tr>
<th>Variable</th>
<th>State Anxiety</th>
<th></th>
<th>Trait Anxiety</th>
<th></th>
<th>ISEL</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r</td>
<td>p</td>
<td>r</td>
<td>p</td>
<td>r</td>
<td>p</td>
</tr>
<tr>
<td>Change in SBP</td>
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<td>.49</td>
<td>-.11</td>
<td>.45</td>
<td>.16</td>
<td>.27</td>
</tr>
<tr>
<td>Change in DBP</td>
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<td>.22</td>
<td>-.26</td>
<td>.08</td>
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<td>.06</td>
</tr>
<tr>
<td>Change in MAP</td>
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<td>.64</td>
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<td>.09</td>
</tr>
<tr>
<td>Change in HR</td>
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<td>.26</td>
<td>.02</td>
<td>.88</td>
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<td>.28</td>
</tr>
<tr>
<td>Change in HF</td>
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<td>.95</td>
<td>.07</td>
<td>.64</td>
<td>.01</td>
<td>.99</td>
</tr>
<tr>
<td>Change in PEP</td>
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<td>.20</td>
<td>.19</td>
<td>.09</td>
<td>.53</td>
</tr>
<tr>
<td>Change in BRS</td>
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<td>.34</td>
<td>.21</td>
<td>.17</td>
<td>-.26</td>
<td>.09</td>
</tr>
</tbody>
</table>

Table 20: *Pearson Product Moment Correlation Coefficients of Anxiety and Social Support with Changes During the Speech Task*

*Note*: SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; HF = high frequency heart rate variability; PEP = pre-ejection period; BRS = baroreceptor sensitivity.

<table>
<thead>
<tr>
<th>Variable</th>
<th>State Anxiety</th>
<th></th>
<th>Trait Anxiety</th>
<th></th>
<th>ISEL</th>
<th></th>
</tr>
</thead>
<tbody>
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<td>p</td>
<td>r</td>
<td>p</td>
<td>r</td>
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<td>.03</td>
<td>.19</td>
<td>.21</td>
<td>-.17</td>
<td>.26</td>
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<tr>
<td>Change in DBP</td>
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<td>.14</td>
<td>.22</td>
<td>.15</td>
<td>-.23</td>
<td>.12</td>
</tr>
<tr>
<td>Change in MAP</td>
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<td>.07</td>
<td>.21</td>
<td>.16</td>
<td>-.21</td>
<td>.15</td>
</tr>
<tr>
<td>Change in HR</td>
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<td>-.03</td>
<td>.85</td>
<td>.13</td>
<td>.39</td>
</tr>
<tr>
<td>Change in HF</td>
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<td>.70</td>
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<td>.11</td>
</tr>
<tr>
<td>Change in PEP</td>
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<td>.18</td>
<td>.25</td>
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<tr>
<td>Change in BRS</td>
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<td>-.25</td>
<td>.10</td>
<td>.12</td>
<td>.42</td>
</tr>
</tbody>
</table>

Table 21: *Pearson Product Moment Correlation Coefficients of Anxiety and Social Support with Changes During the Forehead Cold Pressor Task*

*Note*: SBP = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial pressure; HF = high frequency heart rate variability; PEP = pre-ejection period; BRS = baroreceptor sensitivity. Significant correlations are bolded.

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Relationship between depression and lipid measures. To explore the sixth hypothesis, that depression would be related to cholesterol and lipid measures, depression was correlated with baseline values of total cholesterol, HDL-c, LDL-c, and triglycerides. These correlations are presented in Table 22. Depression was not significantly correlated with any lipid measures.

<table>
<thead>
<tr>
<th>Variable</th>
<th>r</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Total Cholesterol (mg/dl)</td>
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<td>.28</td>
</tr>
<tr>
<td>HDL-c (mg/dl)</td>
<td>.06</td>
<td>.69</td>
</tr>
<tr>
<td>LDL-c (mg/dl)</td>
<td>-.19</td>
<td>.20</td>
</tr>
<tr>
<td>Triglycerides (mg/dl)</td>
<td>-.10</td>
<td>.51</td>
</tr>
</tbody>
</table>

Table 22: Pearson Product Moment Correlation Coefficients of BDI Scores with Lipid Measures

Note: HDL-c = high density lipoprotein cholesterol; LDL-c = low density lipoprotein cholesterol.

Relationships between lipids and autonomic measures. To test the seventh and final hypothesis of the study, that LDL-c cholesterol would exhibit an inverse relationship with HF and BRS, measures of total cholesterol, HDL-c, and LDL-c were correlated with values of PEP, HF and BRS, as well as HF, PEP, and BRS changes during stress. These correlations are presented in Tables 23 and 24. Cholesterol measures were consistently uncorrelated with autonomic measures at baseline. Cholesterol measures were also largely uncorrelated with changes in autonomic measures at baseline.
However, non-significant trends were observed for total cholesterol and LDL-c to be negatively related to changes in BRS during the speech task, indicating that participants with higher cholesterol levels tended to have larger decreases in BRS during the speech task.

<table>
<thead>
<tr>
<th>Variable</th>
<th>PEP</th>
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<th>HF</th>
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<th>BRS</th>
<th></th>
</tr>
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<td>p</td>
<td>r</td>
<td>p</td>
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<td>p</td>
</tr>
<tr>
<td>Total Cholesterol (mg/dl)</td>
<td>.08</td>
<td>.61</td>
<td>.15</td>
<td>.30</td>
<td>.15</td>
<td>.31</td>
</tr>
<tr>
<td>HDL-c (mg/dl)</td>
<td>.06</td>
<td>.66</td>
<td>.11</td>
<td>.46</td>
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<tr>
<td>LDL-c (mg/dl)</td>
<td>.06</td>
<td>.67</td>
<td>.12</td>
<td>.43</td>
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<td>.57</td>
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</tbody>
</table>

Table 23: Pearson Product Moment Correlation Coefficients of Total Cholesterol, HDL-c, and LDL-c with Baseline Values of PEP, HF, and BRS

Note: HF = high frequency heart rate variability; PEP = pre-ejection period; BRS = baroreceptor sensitivity; HDL-c = high density lipoprotein cholesterol; LDL-c = low density lipoprotein cholesterol.
<table>
<thead>
<tr>
<th>Variable</th>
<th>PEP</th>
<th></th>
<th>HF</th>
<th></th>
<th>BRS</th>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Speech Task</td>
<td>-.19</td>
<td>.23</td>
<td>-.20</td>
<td>.19</td>
<td>-.28</td>
<td>.06</td>
</tr>
<tr>
<td>Cold pressor</td>
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<td>.62</td>
<td>-.05</td>
<td>.76</td>
<td>.05</td>
<td>.71</td>
</tr>
<tr>
<td>HDL-c</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Speech Task</td>
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<td>.95</td>
<td>-.17</td>
<td>.26</td>
<td>-.08</td>
<td>.63</td>
</tr>
<tr>
<td>Cold Pressor</td>
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<td>.12</td>
<td>-.14</td>
<td>.36</td>
<td>.03</td>
<td>.86</td>
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<tr>
<td>LDL-c</td>
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<td></td>
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<tr>
<td>Speech Task</td>
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<td>.19</td>
<td>-.15</td>
<td>.31</td>
<td>-.27</td>
<td>.08</td>
</tr>
<tr>
<td>Cold Pressor</td>
<td>-.17</td>
<td>.28</td>
<td>-.02</td>
<td>.91</td>
<td>.10</td>
<td>.52</td>
</tr>
</tbody>
</table>

Table 24: Pearson Product Moment Correlation Coefficients of Total Cholesterol, HDL-c, LDL-c, and Triglycerides with Changes of PEP, HF, and BRS During the Stress Tasks.

Note: HF = high frequency heart rate variability; PEP = pre-ejection period; BRS = baroreceptor sensitivity; HDL-c = high density lipoprotein cholesterol; LDL-c = low density lipoprotein cholesterol.
DISCUSSION

RESEARCH FINDINGS, LIMITATIONS, AND FUTURE DIRECTIONS

Research Findings

The primary purpose of this study was to examine the relationship between depression and BRS at rest and during stress. A secondary purpose of the study was to examine the relationship between depressed mood and autonomic cardiac control. The third major purpose of this study was to explore the relationship between depression, lipids, BRS, and HF.

Depressed Mood and BRS. Hypothesis 1. It was hypothesized that participants with higher depression scores would have lower baseline values of BRS than participants with lower depression scores. This hypothesis was not supported. Depression scores were unrelated to levels of BRS in the planned ANOVA analysis using a median split of BDI scores to define groups with higher and lower depression scores. Because of the restricted range of BDI scores and relatively lower statistical power of ANOVA designs, exploratory correlation analyses were also conducted. These additional analyses revealed that neither depression scores, nor scores on measures of related constructs such as anxiety and social support, were related to BRS at baseline.
These findings are in contrast to those reported by Watkins and Grossman (Watkins & Grossman, 1999), although several features of the current study may help to explain the discrepant findings. First, the Watkins and Grossman study was performed using a sample of patients with coronary artery disease, whereas this study used participants who self-reported an absence of cardiovascular disease. It is possible the relationship between depression and BRS is different in cardiac patients than in healthy controls. In addition, the participants in this study were much younger, and it is possible that a relationship between depression and BRS takes many years to develop. In addition, the levels of depression in this study may have been too low for a strong test of the hypothesis. Finally, participant ratings of difficulty, competence, effort, and anxiety during the resting baseline were not different for participants higher and lower in depressed mood (all $F$’s $< 2.79$, $p$’s $>.10$). Positive and negative affect measured after the resting baseline were also unrelated to depressed mood (all $F$’s $< 1.4$, $p$’s $>.24$). If a relationship between depression and BRS depends upon the psychological state of the participant at the time of measurement, the failure to demonstrate a relationship in this study may be due to similar psychological states in the more and less depressed participants.

Hypothesis 2. It was hypothesized that participants with higher depression scores would have greater reductions in BRS during the speech task and smaller increases during the forehead cold pressor task, relative to participants with lower depression scores. This hypothesis was not supported. An unexpected interaction of depressed mood and stress task was observed. Smaller decreases in BRS were exhibited by more
depressed participants during the speech task, relative to less depressed participants. This finding was counter to expectations. Exploratory correlation analyses were similar to the ANOVA results, with a marginally significant positive correlation between BDI scores and changes in BRS during the speech task.

Analyses of the impression questionnaires and PANAS measures completed at rest and after each task may help to explain the negative finding for depressed mood and changes in BRS during the forehead cold pressor task. Perceptions of difficulty, competence, effort, and anxiety measured after the resting baseline and each task were not different for participants higher and lower in depressed mood (all $F$’s < 2.79, $p$’s > .10). Positive and negative affect measured after the baseline and each task were also unrelated to depressed mood (all $F$’s < 1.4, $p$’s > .24). Again, if a relationship between depression and changes in BRS depends upon the psychological state of the participant at during the stressor, the failure to demonstrate a relationship in this study may be due to similar psychological states in the more and less depressed participants.

Unfortunately, these analyses of task perceptions and affect do little to explain the unexpected relationship between depressed mood and changes in BRS during the speech task. Because this finding is counterintuitive, and because this is the first study to investigate depression and changes in BRS during stress tasks, additional research is required before this result is interpreted to mean that depression is inversely related to changes in BRS during stress tasks designed to evoke a reduction in the parasympathetic nervous system.
Depressed mood and cardiac autonomic control. Hypothesis 3. It was hypothesized that participants with higher depression scores would have larger reductions in HF during the speech task and smaller increases in HF during the forehead cold pressor task, than would participants with lower depression scores. This hypothesis was not supported, as no relationship between depression scores and changes in HF was observed. Consistent with previous research in our laboratory (Hughes & Stoney, 2000), no relationship was observed between depressed mood and baseline values of HF. Thus, in this study depressed mood was unrelated to parasympathetic cardiac control. These results fail to replicate an important finding in our previous study (Hughes & Stoney, 2000). One explanation for the replication failure is that the levels of depression among the more depressed participants were several points lower in this study than in the previous study. Another possible reason for our failure to replicate is that depression was unrelated to task impressions and positive and negative affect in this study. If the more depressed participants in the Hughes & Stoney (2000) study experienced greater stress or negative affect during the tasks than the less depressed participants, this would help to explain the discrepant results. Unfortunately, task impressions questionnaires were not included in the earlier research, so comparisons are not possible.

Hypothesis 4. It was hypothesized that participants with higher depression scores would have larger reductions in PEP during the stress tasks compared to participants with lower depression scores. This hypothesis was not supported, as no relationship between depression scores and changes in PEP was observed. At rest, participants with higher depression scores unexpectedly had larger values of PEP, indicating apparent
lower levels of sympathetic input to the heart, although PEP is not a very good between-subjects measure. This finding is in contrast with a report finding shorter PEP among women with higher BDI scores than those with lower scores (Light et al., 1998).

Related psychosocial variables. Hypothesis 5. It was hypothesized that anxiety and social support would be correlated with depression, but would not account for the relationship between depression and BRS or the relationship between depression and autonomic cardiac control. The fifth hypothesis was partially supported. Specifically, social support and anxiety were related to depression in expected ways. However, most aspects of this hypothesis could not be tested. Depressed mood was largely unrelated to measures of the autonomic nervous system, so few further analyses with anxiety and social support were conducted. One analysis did indicate that trait anxiety and social support partially accounted for the relationship between depression and changes in BRS. Thus, the expectation that depression would be uniquely related to changes in BRS was not supported.

Relationships between lipids BRS, and HF. Hypothesis 6. The relationship between depression and lipids was examined, with no a priori hypotheses. These exploratory analyses revealed few significant relationships of depression scores with either levels of cholesterol or changes in cholesterol during stress. Again, relatively low levels of depression may have prevented positive findings. In addition, the fact that lipids generally did not increase during the stress tasks may help to account for the lack of a relationship between depression and changes in lipids.
Hypothesis 7. It was hypothesized that LDL-c cholesterol would exhibit an inverse relationship with HF and BRS. The seventh hypothesis was not supported. Instead, marginally significant negative correlations were observed for both total cholesterol and LDL-c with changes in BRS during the speech task. Thus, participants with higher atherogenic cholesterol exhibited a tendency to have larger reductions in BRS during the speech task. These findings do not replicate previous reports that elevated total and LDL cholesterol were related to reduced HRV (Danev et al., 1997) and that elevated LDL-c was associated with reduced BRS (Koskinen et al., 1995). One possible explanation is that the sample was too young for sufficient progression of disease processes that may underlie relationships between lipid and autonomic measures.

Other Findings. Scores on the Cohen Perceived Stress Scale (PSS; Cohen et al., 1983) were related to gender ($p < .05$), with women having higher scores. This indicates that women reported higher levels of global perceived stress during the previous week. However, measuring perceived stress added little to the results of the study. Specifically, PSS scores were highly correlated with scores on measures of depression, state anxiety, trait anxiety, and social support (all $r$'s > .58, all $p$'s < .0001) and exhibited relationships with non-invasive measures at baseline similar to those observed for depression. That is, perceived stress during the previous week was positively correlated with PEP ($r = .35, p = .02$), but was unrelated to all other non-invasive measures (all $r$'s < .21, all $p$'s > .16). In addition, no important interactions were observed with gender and no important relationships between PSS scores and changes in noninvasive measures were observed. That is, 2 (Perceived Stress) x 2 (Gender) ANOVAs on baseline values using median
splits of PSS scores to define groups higher and lower in perceived stress indicated that PSS was related to PEP, but was not related to any other measure and did not interact with gender (all $F$'s < .69, all $p$'s > .41). Furthermore, 2 (Perceived Stress) x 2 (Stress Task) x 2 (Gender) ANOVAs on change scores using median splits of PSS scores to define groups higher and lower in perceived stress indicated that PSS was not related to changes in these variables during the stress tasks and did not interact with gender (all $F$'s < 3.2, all $p$'s > .08).

Research Limitations

This study was subject to a number of limitations that may help to explain the findings. The sample size was relatively small and consisted of young individuals with a limited age range. The range of depression scores was quite small and most participant’s depression scores were relatively low. The method of measuring BRS was not ideal. Finally, alternative statistical procedures might have been used, and the experiment-wise Type I error rate was not controlled.

Selection and participation. Relatively few participants completed the study and significant data loss was experienced due to complications such as fainting. This limited the power of the statistical analyses, although this would not have been a serious problem if medium to large effect sizes were observed for the main hypotheses. That is, a power analysis had been conducted prior to conducting the study to estimate an appropriate sample size for this study. An effect size for the hypothesized between-group difference in BRS was calculated from recently reported data (Watkins & Grossman, 1999). Defined as the difference between the group means divided by a pooled standard
deviation (i.e., Cohen's d; Cohen, 1988), the calculated effect size was 0.8. Based on an
effect size of 0.8, a minimum sample size of 42 was needed to find a significant
difference at an alpha level of 0.05 with a power of 0.80. With the obtained sample size
of 50, an effect size of 0.715 would be statistically significant at $p = .05$ with a power of
0.80. However, the observed effect size for the hypothesized between-group difference
in BRS was .06. The number of participants necessary for significant results at $p = .05$
with a power of 0.80 is 6872. Therefore, it would be more practical to attempt to increase
the effect size, perhaps by recruiting more severely depressed participants, than to
increase the power by recruiting more participants.

Second, the age range of the participants was quite small. One effect this may
have had was to control for age related changes in autonomic measures (Craft &
Schwartz, 1995; De Meersman, 1993; Hartikainen et al., 1994). In this case, reduced
age-related variance may have made relationships between these measures and
depression more easily observed. However, if depression exerts an effect on BRS or the
autonomic nervous system, and if this effect takes many years to develop, using only
young participants may have limited our ability to find a relationship between these
measures and depression. It is known that structural changes in the large arteries are
responsible for part of the decreased BRS associated with hypertension, atherosclerosis,
and aging (Chapleau, Cunningham, Sullivan, & Abboud, 1995). If depression were
related to BRS primarily by accelerating hypertensive and atherosclerotic disease
processes, using only young subjects would have limited the opportunity to document a
relationship. However, previous research with a similarly aged sample (Hughes &
Stoney, 2000) suggests that a larger age range would not be necessary to observe a relationship between depression and changes in HF during stress. Unfortunately, the course of a possible relationship between depression and the autonomic nervous system has not been evaluated. Future studies should include a larger range of ages and ideally longitudinal designs.

**Range of BDI scores.** The range of BDI scores in this study was quite small (see Table 3), and the BDI scores in the “high” depressed mood group created by the median split were relatively low. The median split occurred at a BDI score of 3, and the highest score was only 10 for men and 22 for women. The mean BDI score in the more depressed group was only 8.6, below the cutoff of 10 for mild depression and below the cutoff of 9 used in the Watkins and Grossman study (Watkins & Grossman, 1999). This major limitation of the study, perhaps more than any other, may account for our failure to replicate previous research (Hughes & Stoney, 2000) and our failure to support the primary hypotheses.

There are several possible reasons that the depression scores were so low. First, prospective participants were prescreened with the CESD instead of the BDI due to practical limitations specific to the Ohio State University. Although all participants recruited in the “depressed” group had CESD scores above the cutoff score for probable clinical depression (16), a certain level of regression to the mean and/or amelioration of more transient depressed mood in the time that elapsed between prescreen and participation probably occurred. Although no statistical relationship was observed between BDI scores and participant mortality, some of the participants that were unable
to complete the study had higher BDI scores (i.e., 10, 14, and 17). Finally, no participants were successfully recruited from the two clinical samples identified at Ohio State, the Psychology Department Clinic and Counseling and Consultation Services. This may be due in part to the relatively low amount of compensation available for participants (i.e., $10) or the fact that the primary investigator was not actively involved in clinical work at either of these locations during the study. Future grant-funded research should recruit clinical samples and assess participants for clinical levels of depression prior to their participation.

Measurement of BRS. Another important limitation of this study is the method of measuring BRS. Among the non-invasive methods of measuring BRS, the sequencing technique used in this study is perhaps the simplest and was chosen largely for convenience. Unfortunately, using this method resulted in some extreme values that had to be eliminated by re-editing the data and deleting triplets more than 2 SD from the mean. Furthermore, although the sequencing technique of measuring BRS correlates with invasive methods (Watkins et al., 1996), the evidence that BRS is related to mortality following MI is based on research with invasive methods MI (Hohnloser et al., 1993; La Rovere et al., 1988; La Rovere et al., 1998). In addition, the report of reduced BRS among depressed cardiac patients used the power spectrum analysis technique (Watkins & Grossman, 1999). It is possible that more encouraging results would have been obtained with a different method of measuring BRS. In particular, a spectral
analysis technique that correlated highly with the phenylephrine method would have been preferable (Robbe et al., 1987), but software capable of computing BRS with this technique was not available for this study.

Statistical considerations. Another limitation of this study is the choice of statistical analyses. First, relatively liberal statistical analyses were used. Specifically, 2 (Depressed Mood: High vs Low) x 2 (Gender: Women vs men) by 2 (Stress Task: Speech vs Forehead cold pressor) mixed-model ANOVAs using change scores were used to test the hypotheses relating depressed mood and changes in non-invasive measures during the stress tasks. In these analyses, a main effect of depressed mood would indicate a relationship between depressed mood and physiological changes during stress. This could be considered liberal compared to the alternative of using 2 (Depressed Mood: High vs Low) x 2 (Gender: Women vs men) by 5 (Phase: Baseline, Stress Task 1, Recovery 1, Stress Task 2, Recovery 2) ANOVAs, in which case an interaction of Depressed Mood with Phase would indicate a relationship between depressed mood and changes during stress.

However, the choice of statistical test was guided by the fact that a priori hypotheses were specified, the use of similar designs in previous research (Hughes & Stoney, 2000), and the desire to allow every opportunity for the hypotheses to be supported. If the hypotheses had been supported with these analyses, they could have been qualified in the discussion and repeated with the more stringent design.

Second, the change score analyses were performed even though one-way ANOVAs showed that some measures did not change during both stress tasks. For
example, HF and BRS decreased during the speech task but exhibited non-significant increases during the forehead cold pressor. It could be argued that change score analyses that include measures which did not significantly change, such as HF and BRS during the forehead cold pressor task, are inappropriate. Again, it was decided to test every a priori hypothesis and to allow every opportunity for the hypotheses to be tested.

Third, the use of median split ANOVA designs is less powerful than regression techniques. However, ANOVA designs were chosen for compatibility with previous research. In addition, these designs allow for easily interpretable findings, especially where interactions are observed, whereas regression designs can be more cumbersome. Because the range of BDI scores was relatively small, exploratory correlation analyses were conducted to help reduce the impact of relatively lower power. The results of the correlation analyses essentially mirrored the ANOVA designs, suggesting that the use of median split ANOVA designs did not result in a failure to document important relationships.

*Experiment-wise Type I error rate.* All a priori hypotheses were tested at the .05 level of significance, and no effort was made to control for the experiment-wise Type I error rate. Therefore, 5% of the analyses conducted should be significant by chance alone. Because a large number of comparisons were made in this study, some of those that were significant may represent Type I errors. For this reason, it is important to replicate findings and to avoid over-interpreting the results. In this study, the relationship observed between depression and PEP at baseline is one example of a finding that should be replicated prior to firm conclusions.
Future Studies

Although the results of this study are somewhat inconclusive, the questions asked in this research have important implications for cardiovascular behavioral medicine. Documenting a relationship between depression and the arterial baroreflex contribution to autonomic control of the heart would provide evidence supporting the hypothesis that depression confers additional risk following MI through the physiological mechanism of altered autonomic functioning. Demonstrating a relationship between depression and BRS in healthy samples would also help to rule out the alternative hypothesis that the relationship between depression and HRV observed in cardiac patients is due to differential degrees of disease severity between the depressed and less depressed groups. A methodologically strong study that fails to find a relationship between depression and BRS, and depression and autonomic control, in healthy young individuals would suggest that the apparent relationship between depression and autonomic nervous system functioning in cardiac patients is a result of the disease process.

Other researchers are currently conducting research on depression and BRS with cardiac patients, and the literature on depression and other measures of autonomic functioning continues to grow. However, a study that includes both depressed and non-depressed cardiac patients and age-matched healthy controls has not been conducted. Incorporating an age-matched control group of persons found to be free of coronary artery disease would eliminate some of the limitations of this study (e.g., restricted age range) and would more adequately test the hypothesis that depression confers an increased risk of mortality through autonomic nervous system functioning.
Although the research at this point is mixed, if a relationship between depression and autonomic functioning is clearly established, the nature of this relationship could be more clearly characterized. Studies of depression and the autonomic nervous system typically assume that depression exerts a static deleterious effect. However, specific central nervous system pathways have not been described for this relationship, whereas they have been for anxiety and autonomic functioning (Berntson, Sarter & Cacioppo, 1998).

It is also possible that a relationship between depression and the autonomic nervous system is secondary to coping responses or other processes. The ANS is extremely sensitive to immediate psychological states, environmental demands, and coping efforts. Perhaps depressed and/or anxious patients experience more frequent or severe perturbations of the ANS, instead of having chronically altered ANS functioning. This view was recently stated succinctly by one group of researchers; "autonomic reactions often mirror the pattern of exaggerated affective/behavioral response, rather than reflecting a primary abnormality in autonomic regulation" (Berntson et al., 1998, p. 227). Individual difference constructs such as emotional responsivity, or the tendency to respond with exaggerated negative affect to daily events, is one way of relating depression and anxiety with ANS responses. Emotional responsivity has been related to ambulatory blood pressure (Carels, Blumenthal & Sherwood, 2000), myocardial ischemia (Carels et al., 1999), and levels of depression and anxiety (Carels et al., 1999). Future research incorporating constructs such as emotional responsivity and ambulatory
monitoring of autonomic functioning may reveal that a relationship between depression and autonomic functioning is due primarily to depression eliciting frequent experiences of negative affect or other similarly dynamic processes.

As suggested in the introduction, another important research topic is the role of related psychological constructs in the relationship between depression and the autonomic nervous system. Although there are clearly articulated theories relating anxiety and the autonomic nervous system (Association, 1994; Barlow, 2000; Berntson et al., 1998; Clark & Watson, 1991), there are few theories positing a relationship between depression and the autonomic nervous system. Future studies that avoid selection biases and use measures with equivalent reliabilities and validities are necessary to explicate the unique contributions of depression, anxiety, social support, and other related constructs to alterations in autonomic nervous system functioning.

Finally, if altered autonomic nervous system functioning is determined to be an important mechanism by which depression confers an increased risk of post-MI mortality, it will be important to evaluate the effects of interventions for depression or altered autonomic nervous system functioning. A group of investigators recently considered whether or not treating depression can reduce mortality following MI (Carney et al., 1999b). A number of issues remain unresolved, but these researchers suggested that treating depression might not improve autonomic functioning, and might therefore not reduce post-MI. Fortunately, when the results of the ENRICHD and SADHART trials are available, the effects of treating depression on post-MI mortality will be clearer.
REFERENCES


of non-specific symptoms. *International Journal of Psychiatry in Medicine, 22, 221-229.*


Appendix A:

Advertisement

Brochure

Telephone Prescreen

Letter to Participants
Emotions and Your Heart:  
Would you volunteer for a research study?

Consider participating as a volunteer in a study being conducted at The Ohio State University designed to examine the effect of emotions and mental challenges on blood pressure and heart rate responses in healthy men and women.

Description and Location:
The purpose of this study is to understand the effect of emotions and mental challenges on cholesterol, heart rate, blood pressure, and related measures. The study requires one visit on a weekday morning to the Clinical Research Center at the Ohio State University Medical Center or the Cardiovascular Behavioral Medicine Laboratory.

Eligibility:
Men and women between the ages of 18 and 30.
You must be a non-smoker.
You must not be taking antidepressant or anti-anxiety medications.
AHealthy: You must not have heart disease.
    You must not be taking medications that affect your heart.
    You must not have high blood pressure.
    You must not have high cholesterol.

Benefits:
You will be paid $10.00 if you participate in this study.
You can receive a statement of your cholesterol and blood pressure.
You can receive REP credit if you are a Psychology 100 student.

For further information, contact:
    Joel Hughes
    (614) 688-3346
    The Ohio State University
Cardiovascular
Behavioral
Medicine Laboratory.
The Ohio State
University
206 Townshend Hall
1885 Neil Avenue Mall
Columbus, OH 43210

Emotions
And
Your Heart

A Research Study
by
The Ohio State University
Cardiovascular Behavioral Medicine
Laboratory
(614) 688-3895
Funded
by
The National Institutes of Health
Catherine M. Stoney, Ph.D.
Joel W. Hughes, M.A.
Principal Investigators

Emotions and Your Heart
Researchers in the Cardiovascular Behavioral Medicine Laboratory are working to better understand how stress affects health. We are seeking volunteers to participate in a study of how different moods and emotions affect cholesterol, heart rate, blood pressure, and related measures.

Studies have demonstrated that brief mental stress, such as experienced when giving a speech, increases the “bad” cholesterol, as well as blood pressure and heart rate. We are seeking to understand the effects of different emotions (especially feelings of depression) on these physiological processes.

This study takes place at the Clinical Research Center at the Ohio State University Medical Center. It requires one visit of about 2.5 hours on a weekday morning.

This study is being conducted by Joel Hughes, M.A. and Catherine Stoney, Ph.D.

You can participate if you are:

♦ A man or woman between the ages of 18 and 30.
♦ A non-smoker.
♦ You are not taking medication for depression or anxiety.
♦ You are not pregnant.
♦ You are “healthy:”
  ↩ You must not have heart disease.
  ↩ You must not be taking medications that affect your heart
  ↩ You must not have high blood pressure.
  ↩ You must not have high cholesterol.

What are the incentives and benefits of participating?

♦ We can pay you a small compensation ($10) for completing the study.
♦ You will receive a light breakfast or lunch after the study.
♦ You can receive complete information on your cholesterol, HDL, LDL, triglycerides, heart rate, and blood pressure.

♦ Satisfaction in knowing that you are a part of a program that is contributing to the advancement of science and that our efforts may help others.

How can I participate in the Emotions and Your Heart Study?

If you are interested in participating or would like to receive additional information, please call Joel Hughes at 688-3895.

After a brief telephone conversation, we will schedule you at your earliest convenience.
Telephone Prescreening

Telephone Screening Interview: Emotions and your heart.

NAME:__________________________ DATE OF CONTACT:________
TIME:________________________

Hello this is and I am calling about a study entitled “Emotions and your heart” being run at the Ohio State University. Do you have a few minutes to talk about it now?

The purpose of study is to understand the relationship between emotions, mental challenges, and measures of the functioning of the cardiovascular system such as cholesterol, heart rate, and blood pressure.

We are investigating physiological changes that occur as people react to everyday types of challenges. For example, blood pressure increases during challenging laboratory tasks. We are attempting to determine if some of these changes during challenges are related to different emotions that people feel. Would you like to hear more about the study?

This study is being run at the Clinical Research Center at The Ohio State University Medical Center. If you are interested and eligible, here is what would happen. This study involves one visit of approximately 2.5 hours to the Medical Center on a weekday morning. You would need to come without eating or drinking anything except water for 12 hours, so that your cholesterol levels will not be affected by recent meals.

During the study, your bodily reactions will be measured while you perform two tasks. Specifically, we will ask you to prepare and then deliver an impromptu speech on videotape and place a cold-compress on your forehead for a few minutes. These tasks are not designed to embarrass you, but are intended to be challenging enough to cause momentary changes in blood pressure and heart rate variability. The cold-compress is similar to one you would use for a headache or sore muscle. It’s cold but does not harm you in any way.

We will measure your bodily reactions such as your cholesterol, heart rate, blood pressure, and breathing before, during, and after these tasks. The measurement equipment we use is as follows: first, we would place a blood pressure cuff around your arm. Next, we will tape sensors around your neck and chest to measure the electrical activity of the heart as well as your breathing. Then we will place a small needle in one of your arm veins for drawing blood. The blood samples will be used to measure cholesterol (a type of fat). We will take a total of five samples, which is about 5 ounces of blood. After the needle is in your arm, you will not feel it, although we will ask you to try to keep from moving your arm.

You will also be asked to complete some questionnaires regarding the emotions you feel, your mood, your health behaviors, and your health. You will be paid $10.00 for completing participation in this study. You can also receive a summary of your blood pressure and cholesterol responses when you were in the study, although it takes awhile for the cholesterol results to come back and we will mail those results to you. Do you have any questions? Are you interested in participating in this study? If so, I would like to ask you some questions to see if you are eligible to participate in this study. All of your answers to these questions will remain confidential, and will only be made available as necessary to the immediate members of the research team.
NAME: _________________________________ TELEPHONE: ____________________________
ADDRESS: ____________________________________________________________

1. What is your age? ______________________________________________________________ (18-30)
2. Do you smoke? ________________________________________________________________ (N)
3. What is your height? ____________________________________________________________ (within 20% of ideal weight)
4. What is your weight? ____________________________________________________________
5. What is your ethnic group? ______________________________________________________
   (This is not required to participate, but we want to be sure to include groups that are often underrepresented in health research).
6. Are you under a physicians care for a chronic health problem(s)? ________
   If yes, describe: ________________________________________________________________
7. Are you diabetic? ________
   If yes, ___________________________________________
8. Are you medically restricted in any activities? ________
   If yes, ___________________________________________
9. Have you ever been told you had heart problems, a heart murmur or congenital heart disease? ________
   If yes, ___________________________________________
10. Have you ever had hepatitis? ________
11. Do you have a history of lung, liver, kidney, respiratory, or blood pressure problems? ________
12. Do you regularly take non-prescription medications? ________
   If yes, ___________________________________________
13. Do you regularly take any prescription medications? ________
   If yes, ___________________________________________
14. Do you take medicine for High Blood Pressure or High Cholesterol? (N) ________
15. Do you take medicine for depression or anxiety? (N) ________
16. Are you allergic to anything? ________
   If yes, ___________________________________________
(FOR WOMEN: 17. Are you pregnant or do you believe you could be pregnant, meaning that you have used some form of birth control for at least one month? ________ (N)

IF NOT ELIGIBLE:
I am sorry, but you are not eligible to participate in this study. Would you like for me to put your name on a list of people who may be interested in other, similar studies that we are conducting?

IF ELIGIBLE:
Schedule DATE: ____________________
TIME: ____________________________

Instructions: The Clinical Research Center is in the Ohio State University Medical Center. I will meet you in the lobby by the elevator on the 10th floor. Take the elevator to the 10th floor and wait in the lobby. Thanks so much. See you soon.

   It is very important that you do not eat or drink anything except water for 12 hours before participating. This is after ________ p.m. the night before.
   It is important that you don’t engage in vigorous exercise during the morning before participating.
   It is also important that you wear clothes that allow us to attach the sensors to your neck and chest. For example, short sleeves are preferable (for women: it is better not to wear a dress).
Emotions and Your Heart Study

Thank you again for participating in my dissertation research. Below are your cholesterol and blood pressure values.

Your cholesterol values while resting:

<table>
<thead>
<tr>
<th></th>
<th>mg/dl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Cholesterol</td>
<td></td>
</tr>
<tr>
<td>Low Density Lipoprotein Cholesterol (LDL)</td>
<td></td>
</tr>
<tr>
<td>High Density Lipoprotein Cholesterol (HDL)</td>
<td></td>
</tr>
<tr>
<td>Triglycerides</td>
<td></td>
</tr>
</tbody>
</table>

Cholesterol is a fatty substance produced by the body that is an essential part of every cell. Cholesterol is also found in foods of animal origin; eggs, milk, meat, fish, and poultry. Saturated fat in the diet is also easily converted to cholesterol by the body.

Generally, the American Heart Association recommends that total cholesterol levels remain between 200-240 mg/dl or lower, that low density lipoprotein cholesterol (LDL) levels remain at 130 mg/dl or lower, and that high density lipoprotein cholesterol (HDL) levels remain at 35 mg/dl or higher. However, it is important to note that many factors can temporarily alter cholesterol levels. LDL is often called the “bad” cholesterol, and high levels of LDL are a risk factor for heart disease. HDL is often called the “good” cholesterol, and high levels help to protect against heart disease.

The American Heart Association recommends that triglyceride levels remain below 400 mg/dl. Normal triglycerides are below 200, and values between 200 and 400 are considered borderline-high. Triglycerides are strongly influenced by eating, so high values are common if you did not fast 12 hours before the blood sample was taken.

Your blood pressure and heart rate values while resting:

<table>
<thead>
<tr>
<th></th>
<th>mm/Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood Pressure</td>
<td></td>
</tr>
<tr>
<td>Heart rate</td>
<td></td>
</tr>
</tbody>
</table>

The higher number, or systolic blood pressure, represents the pressure when the heart is beating. The lower number, or diastolic blood pressure, represents the pressure when the heart is resting between beats. The systolic number is always stated first and the diastolic number is always second. According to the American Heart Association, blood pressure of less than 140/90 is considered normal for adults. A blood pressure that is consistently over 140/90 is considered elevated or high. However, many factors can temporarily alter blood pressure, so consistent readings across time are typically used to determine if a person’s blood pressure is too high. Blood pressure of 120/80 or less is considered optimal for risk of heart disease and stroke. Usually, the lower your blood pressure the better, although extremely low blood pressure would need to be evaluated by a physician if it produces symptoms such as lightheadedness or fainting.

If your cholesterol or blood pressure is too high, you should ask your physician about your cholesterol or blood pressure at your next visit. In addition, moderate physical exercise and a diet low in cholesterol and saturated fat helps to control cholesterol levels.

If you have any questions about the study, you are welcome to call me at 688-3895 or e-mail hughes.312@osu.edu.

Joel W. Hughes, M.A.
Appendix B

Psychosocial Questionnaires
Emotions and Your Heart: Personal and Family Information

Participant No.: _______ Date: _______________
Social security No.: ________________________
Age: ________________________
Date of Birth.: ________________________________________________________________
Please fill in:

In terms of ethnic group, I consider myself to be ____________________________

1. My ethnicity is
   (1) Indian
   (2) Asian, Asian American, or Oriental
   (3) Black or African-American
   (4) Hispanic of Latino
   (5) White, Caucasian, European, not Hispanic
   (6) American Indian
   (7) Mixed, my parents are from two different groups
   (8) Other (write in) ________________________________

2. My father’s ethnicity is (use the numbers 1-8 from question 1)

3. My mother’s ethnicity is (use the numbers 1-8 from question 1)

4. Was English your first language?

5. What is your total family income (include income from main wage-earners in the home)?
   ______$0-$14,999
   ______$15,000-$29,999
   ______$30,000-$49,999
   ______$50,000-$74,999
   ______$75,000-$99,999
   ______$100,000 and above

6. What class do you consider yourself to be a member of?
   ______ Lower class
   ______ Working class
   ______ Lower middle class
   ______ Upper middle class
   ______ Upper class
7. What is the highest level of education you have attained?
   _____ Less than high school graduate
   _____ High school graduate
   _____ Some college (attended school for some time or obtained associate's degree)
   _____ College graduate (received bachelor's degree)
   _____ More than college graduate (Some graduate level courses, master's, doctorate).

8. What is the highest level of education attained by your mother?
   _____ Less than high school graduate
   _____ High school graduate
   _____ Some college (attended school for some time or obtained associate's degree)
   _____ College graduate (received bachelor's degree)
   _____ More than college graduate (Some graduate level courses, master's, doctorate).

9. What is the highest level of education attained by your father?
   _____ Less than high school graduate
   _____ High school graduate
   _____ Some college (attended school for some time or obtained associate's degree)
   _____ College graduate (received bachelor's degree)
   _____ More than college graduate (Some graduate level courses, master's, doctorate).

Health Information

10. What prescription medication(s) do you take?

11. Are you on a diet? Yes  No
   If yes, what kind?
   _____ fasting (number of days at a time?____)
   _____ calorie counting (number of calories per day?____)
   _____ carbohydrates counting (number per day?____)
   _____ low fat (% of calories from fat?____)
   _____ other, please specify:____________________________
Participant Address Information

Please write your address below so that we can mail you information about your cholesterol and blood pressure. You will receive this information as soon as possible, although the laboratory tests may take several months to be completed.

Name: 

Address: 

Phone Number: 
Beck Depression Inventory

INSTRUCTIONS: This questionnaire consists of 20 groups of statements. After reading each group of statements carefully, circle the number (0, 1, 2, or 3) which best describes the way you have been feeling the PAST WEEK, INCLUDING TODAY! Circle the number beside the statement you picked. If several statements in the group seem to apply equally well, circle each one. Be sure to read all the statements in each group before making your choice.

1. 0 I do not feel sad.
   1 I feel sad.
   2 I am sad all the time and I can't snap out of it.
   3 I am so sad or unhappy that I can't stand it.

2. 0 I am not particularly discouraged about the future.
   1 I feel discouraged about the future.
   2 I feel I have nothing to look forward to.
   3 I feel that the future is hopeless and that things cannot improve.

3. 0 I do not feel like a failure.
   1 I feel I have failed more than the average person.
   2 As I look back on my life, all I can see is a lot of failures.
   3 I feel I am a complete failure as a person.

4. 0 I get as much satisfaction out of things as I used to.
   1 I don't enjoy things the way I used to.
   2 I don't get real satisfaction out of anything anymore.
   3 I am dissatisfied or bored with everything.

5. 0 I don't feel particularly guilty.
   1 I feel guilty a good part of the time.
   2 I feel quite guilty most of the time.
   3 I feel guilty all of the time.

6. 0 I don't feel I am being punished.
   1 I feel I may be punished.
   2 I expect to be punished.
   3 I feel I am being punished.

7. 0 I don't feel disappointed in myself.
   1 I am disappointed in myself.
   2 I am disgusted with myself.
   3 I hate myself.
|   | 0 | I don’t feel I am any worse than anybody else.  
|   | 1 | I am critical of myself for my weaknesses or mistakes.  
|   | 2 | I blame myself all the time for my faults.  
|   | 3 | I blame myself for everything bad that happens.  
|---|---|---|
|   | 9 | I don’t have any thoughts of killing myself.  
|   | 1 | I have thoughts of killing myself but I would not carry them out.  
|   | 2 | I have definite plans about killing myself.  
|   | 3 | I would kill myself if I had the chance.  
|---|---|---|
|   | 10 | I don’t cry anymore than usual.  
|   | 1 | I cry more now than I used to.  
|   | 2 | I cry all the time now.  
|   | 3 | I used to be able to cry, but now I can’t even though I want to.  
|---|---|---|
|   | 11 | I am no more irritated now than I ever am.  
|   | 1 | I get annoyed or irritated more easily than I used to.  
|   | 2 | I feel irritated all the time now.  
|   | 3 | I don’t get irritated at all by the things that used to irritate me.  
|---|---|---|
|   | 12 | I have not lost interest in other people.  
|   | 1 | I am less interested in other people than I used to be.  
|   | 2 | I have lost most of my interest in other people.  
|   | 3 | I have lost all of my interest in other people.  
|---|---|---|
|   | 13 | I make decisions about as well as I ever could.  
|   | 1 | I put off making decisions more than I used to.  
|   | 2 | I have greater difficulty in making decisions than before.  
|   | 3 | I can’t make decisions at all anymore.  
|---|---|---|
|   | 14 | I don’t feel I look any worse than I used to.  
|   | 1 | I am worried that I am looking old or unattractive.  
|   | 2 | I feel that there are permanent changes in my appearance that make me look unattractive.  
|   | 3 | I believe that I look ugly.  
|---|---|---|
|   | 15 | I can work about as well as before.  
|   | 1 | It takes an extra effort to get started at doing something.  
|   | 2 | I have to push myself very hard to do anything.  
|   | 3 | I can’t do any work at all.  

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16. 0 I can sleep as well as usual.
   1 I get tired more easily than I used to.
   2 I wake up 1-2 hours earlier than usual and I find it hard to get back to sleep.
   3 I wake up several hours earlier than I used to and cannot get back to sleep.

17. 0 I don’t get more tired than usual.
   1 I get tired more easily than I used to.
   2 I get tired from doing almost anything.
   3 I am too tired to do anything.

18. 0 My appetite is no worse than usual.
   1 My appetite is not as good as it used to be.
   2 My appetite is much worse now.
   3 I have no appetite at all anymore.

19. 0 I haven’t lost much weight, if any, lately.
   1 I have lost more than 5 pounds.
   2 I have lost more than 10 pounds.
   3 I have lost more than 15 pounds

I am purposely trying to lose weight by eating less. Yes___ No

20. 0 I am no more worried about my health than usual.
   1 I am worried about physical problems such as aches and pains; or upset stomach; or constipation.
   2 I am sad all the time and I can’t snap out of it.
   3 I am so sad or unhappy that I can’t stand it.

21. 0 I have not noticed any recent change in my interest in sex.
   1 I am less interested in sex than I used to be.
   2 I am much less interested in sex now.
   3 I have lost interest in sex completely.
Interpersonal Support Evaluation List

Participant no.: ________________________ Date: ____

This scale is made up of a list of statements, each of which may or may not be true about you. For each statement, circle probably true (T) if the statement is true about you, or probably false (F) if the statement is not true about you. You may find that many of the statements are neither clearly true nor clearly false. In these cases, try to decide quickly whether probably true (T) or probably false (F) is most descriptive of you. Although some questions will be difficult to answer, it is important that you pick one alternative or the other. Remember to circle only one of the alternatives for each statement. Please read each item quickly but carefully before responding.

T F 1. There is someone I could turn to for advice about changing my job or finding a new one.
T F 2. No one I know would throw a birthday party for me.
T F 3. If I need a quick emergency loan of $100, there is someone I could get it from.
T F 4. In general, people don't have much confidence in me.
T F 5. When I need suggestions for how to deal with a personal problem, I know there is someone I can turn to.
T F 6. When I feel lonely, there are several people I could call and talk to.
T F 7. If I had to mail an important letter at the post office by 5:00 and couldn't make it, there is someone who could do it for me.
T F 8. I think that my friends feel that I'm not very good at helping them solve problems.
T F 9. I feel that there is no one with whom I can share my most private worries and fears.
T F 10. If I wanted to go out of town for the day, I would have a hard time finding someone to go with me.
T F 11. If I were sick and needed someone to drive me to the doctor, I would have trouble finding someone.
T F 12. I am more satisfied with my life than most people are with theirs.
T F 13. There is really no one I can trust to give me good financial advice.
T F 14. If I wanted to have lunch with someone, I could easily find someone to join me.
T F 15. If I got stranded 10 miles out of town, there is someone I could call to come and get me.
T F 16. Most people I know think highly of me.
T F 17. There is at least one person I know whose advice I really trust.
T F 18. I feel that I'm on the fringe of my circle of friends.
T F 19. If I needed a ride to the airport very early in the morning, I would have a hard time finding anyone to take me.
T F 20. I am closer to my friends than most people.
T F 21. There is someone I can turn to for advice about handling hassles over household responsibilities.
T F 22. If I decided on a Friday afternoon that I would like to go to a movie that evening, I could find someone to go with me.
T F 23. If I needed someone to help in moving to a new home, I would have a hard time finding someone to help me.
T F 24. I have a hard time keeping pace with my friends
T F 25. There are very few people I trust to help solve my problems.
T F 26. I don't often forget to do things with others.
T F 27. If for some reason I were put in jail, there is someone I could call who would bail me out.
T F 28. I have someone who takes pride in my accomplishments.
T F 29. There is really no one who can give me objective feedback about how I'm handling my problems.
T F 30. There are several different people with whom I enjoy spending my time.
T F 31. If I were sick, there would be almost no one I could find to help me out with my daily chores.
T F 32. I am able to do things as well as most other people.
T F 33. There is someone whom I feel comfortable going to for advice about sexual problems.
T F 34. Most people I know don't enjoy the same things that I do.
T F 35. There is no one I could call on if I needed to borrow a car for a few hours.
T F 36. Most of my friends are more interesting than I am.
T F 37. If a family crisis arose, few of my friends would be able to give me good advice about handling it.
T F 38. I regularly meet or talk to members of my family or friends.
T F 39. If I had to go out of town for a few weeks, someone I know would look after my home (plants, pets, yard, etc.).
T F 40. Most of my friends are more successful at making changes in their lives than I am.
Emotions and Your Heart; COHEN Perceived Stress Scale
Participant No:____________________

Thoughts and Feelings During the Past Week
The questions in this scale ask you about your feelings and thoughts during the PAST WEEK. In each case, you will be asked to indicate HOW OFTEN you felt or thought a certain way. Although some of the questions are similar, there are differences between them and you should treat each one as a separate question. The best approach is to answer each question fairly quickly. That is, don't try to count up the number of times you felt a particular way, but rather indicate the alternative that seems like a reasonable estimate. Use the scale below for the following questions.

1 = never
2 = almost never
3 = sometimes
4 = fairly often
5 = very often

___ 1. In the LAST WEEK, how often have you been upset because of something that happened unexpectedly?
___ 2. In the LAST WEEK, how often have you feel that you were unable to control the important things in your life?
___ 3. In the LAST WEEK, how often have you felt "stressed"?
___ 4. In the LAST WEEK, how often have you feel confident about your ability to handle your personal problems?
___ 5. In the LAST WEEK, how often have you felt that things were going your way?
___ 6. In the LAST WEEK, how often have you found that you could not cope with all the things that you had to do?
___ 7. In the LAST WEEK, how often have you been able to control irritations in your life?
___ 8. In the LAST WEEK, how often have you felt that you were on top of things?
___ 9. In the LAST WEEK, how often have you been angered because of things that happened that week outside of your control.
___ 10. In the LAST WEEK, how often have you felt difficulties were piling up so high that you could not overcome them?
Emotions and Your Heart (Anger Expression Scale)

Participant No:_____________________

Instructions: For the following items, indicate the number which best describes how you generally act or feel when you are angry.

1 = almost never
2 = sometimes
3 = often
4 = almost always

1. I control my temper. ______________________________________________________
2. I express my anger. ______
3. I keep things in. ______
4. I control my behavior. ______
5. I pout or sulk. ______
6. I withdraw from people. ______
7. I control my angry feelings. ______
8. I make sarcastic remarks to others. ______
9. I keep my cool. ______
10. I do things like slam doors. _______________________________________________
11. I boil inside, but I don't show it. __________________________________________
12. I try to be tolerant and understanding. ____________________________________
13. I argue with others. ______
14. I tend to harbor grudges that I don't tell anyone about. ______
15. I strike out at whatever infuriates me. ______
16. I am secretly quite critical of others. ______
17. I am usually quite patient with others. ______
18. I am angrier than I am willing to admit. ______
19. I calm down faster than most people. ______
20. I say nasty things. ______
21. I am irritated a great deal more than people are aware. ______
22. I lose my temper. ______
23. If someone annoys me, I am apt to tell him or her how I feel. ______
24. I stop myself from losing my temper. ______
SELF-EVALUATION QUESTIONNAIRE
(Spielberger State-Trait Anxiety Inventory)
Developed by C.D. Spielberger, R.L. Gorsuch and R. Lushene

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement then choose the number that best describes how you feel right now, that is, at this moment. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

1 = NOT AT ALL
2 = SOMEWHAT
3 = MODERATELY SO
4 = VERY MUCH SO

1. I feel calm
2. I feel secure
3. I am tense
4. I am regretful
5. I feel at ease
6. I feel upset
7. I am presently worrying over possible misfortunes
8. I feel rested
9. I feel anxious
10. I feel comfortable
11. I feel self-confident
12. I feel nervous
13. I am jittery
14. I feel “high strung”
15. I am relaxed
16. I feel content
17. I am worried
18. I feel over-excited and “rattled”
19. I feel joyful
20. I feel pleasant
DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then choose the number that best describes how you generally feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

21. I feel pleasant
22. I tire quickly
23. I feel like crying
24. I wish I could be as happy as others seem to be
25. I am losing out on things because I can't make up my mind soon enough
26. I feel rested
27. I am "calm, cool, and collected"
28. I feel that difficulties are piling up so that I cannot overcome them
29. I worry too much over something that really doesn't matter
30. I am happy
31. I am inclined to take things hard
32. I lack self-confidence
33. I feel secure
34. I try to avoid facing a crisis or difficulty
35. I feel blue
36. I am content
37. Some unimportant thought runs through my mind and bothers me
38. I take disappointments so keenly that I can't put them out of my mind
39. I am a steady person
40. I get in a state of tension or turmoil as I think over my recent concerns and interests
IMPRESSIONS QUESTIONNAIRE

On each of the following scales, please indicate your impressions of the resting period you just completed.

1. Relaxing quietly was:

   1  2  3  4  5
   Not Very Difficult          Very Difficult

2. While relaxing quietly, I felt:

   1  2  3  4  5
   Not At All Competent        Very Competent

3. While relaxing quietly, I:

   1  2  3  4  5
   Tried Hard                 Didn’t Try Hard

4. In general, when I have to relax quietly, I feel:

   1  2  3  4  5
   Not At All Anxious         Very Anxious
COLD PRESSOR TASK IMPRESSIONS QUESTIONNAIRE

On each of the following scales, please indicate your impressions of the task you just completed.

1. This task was:

   1  2  3  4  5
   Not Very Difficult  Very Difficult

2. During this task, I felt:

   1  2  3  4  5
   Not At All  Very Competent
   Competent

3. During this task, I:

   1  2  3  4  5
   Tried Hard  Didn’t Try Hard

4. How much pain did you experience during this task:

   1  2  3  4  5
   None  Very much
SPEECH TASK IMPRESSIONS QUESTIONNAIRE

On each of the following scales, please indicate your impressions of the task you just completed.

1. This task was:

   1  2  3  4  5
   Not Very Difficult Very Difficult

2. During this task, I felt:

   1  2  3  4  5
   Not At All Very Competent
   Competent

3. During this task, I:

   1  2  3  4  5
   Tried Hard Didn’t Try Hard

4. In general, when I have to give a speech, I feel:

   1  2  3  4  5
   Not At All Very Anxious
Emotions and Your Heart (Positive and Negative Affect Scale)

Participant No.:___________________

This scale consists of a number of words that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you are feeling this way right now. Use the following scale to record your answers.

1  2  3  4  5
Very slightly  A little  Moderately  Quite a bit  Extremely

___  1. Interested
___  2. Distressed
___  3. Excited
___  4. Upset
___  5. Strong
___  6. Guilty
___  7. Scared
___  8. Hostile
___  9. Enthusiastic
___ 10. Proud
___ 11. Irritable
___ 12. Alert
___ 13. Ashamed
___ 14. Inspired
___ 15. Nervous
___ 16. Determined
___ 17. Attentive
___ 18. Jittery
___ 19. Active
___ 20. Afraid