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The Effects of Rumination, Hostility, and Distraction on Cardiovascular Reactivity and Recovery From Anger Recall in Healthy Women

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The Effects of Rumination, Hostility, and Distraction on Cardiovascular Reactivity and
Recovery From Anger Recall in Healthy Women

by

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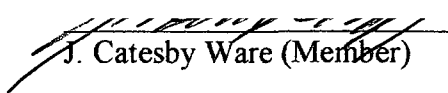
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ABSTRACT

THE EFFECTS OF RUMINATION, HOSTILITY, AND DISTRACTION ON CARDIOVASCULAR REACTIVITY AND RECOVERY FROM ANGER RECALL IN HEALTHY WOMEN

Meghan K. McLain
Old Dominion University, 2010
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Cardiovascular reactivity and recovery following an emotional stressor may play a crucial role in mediating the relation between psychosocial factors (e.g. hostility and anger) and cardiovascular disease. Hostility has been associated with trait rumination. Trait rumination, a tendency to focus attention on negative thoughts and emotions and be prone to feelings of revenge, is not adequately captured in current measures of hostility. The current study examined whether trait rumination, indexed by the Dissipation-Rumination Scale, has an independent effect of increasing cardiovascular reactivity and prolonging cardiovascular recovery from angry events above and beyond hostility as measured by the Cook-Medley Hostility Scale. The effect of distraction on cardiovascular recovery from anger recall was also examined. Diastolic and systolic blood pressure (DBP; SBP), heart rate (HR), high and low frequency heart rate variability (HF; LF), preejection period (PEP), stroke index (SI), cardiac index (CI) and total peripheral resistance index (TPR) were collected from 80 healthy women (ages 18-30) during a 15-min baseline, a 3-min anger recall, and a 10-min recovery. Half of the participants were randomly assigned to a distraction condition (i.e. reading a neutral article) during recovery. Hierarchical regressions, controlling for hostility scores,

revealed that trait rumination was predictive of increased SI ($p < .03$) during the anger-recall task. Trait rumination also predicted slower post-task recovery for HR ($p < .007$) and SI ($p < .001$). An interaction between trait rumination and distraction was found to predict SI ($p < .01$) and CI ($p < .04$), such that those with high trait rumination experienced a greater benefit from distraction than individuals low in trait rumination and high in trait rumination in the no distraction condition. Thus, trait rumination appears to increase cardiac reactivity and prolong recovery from anger, independent of hostility, which may partly explain interrelations among anger, stress responses, and cardiovascular disease risk. These findings also suggest that distraction may be a useful intervention to reduce the physiological impact of trait rumination.

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INTRODUCTION

The objectives of this study are two-fold. The first objective is to examine the effects of trait rumination, independent of hostility, on cardiovascular reactivity during an anger-recall task and post-task cardiovascular recovery. The second objective is to examine the potential use of distraction as an intervention to reduce the consequences of rumination on cardiovascular recovery.

Cardiovascular reactivity and post-stress recovery have been hypothesized to play a mediating role in the relationship between psychosocial factors (such as hostility, anger, and depression) and cardiovascular disease (Krantz & Manuck, 1984; Lovallo, 2005; Steptoe & Marmot, 2006). That is, the contribution of psychosocial factors to cardiovascular disease may be partially mediated by the recurrence of cardiovascular reactions to psychological stress. Evidence for this hypothesis is derived from studies that have found that increased cardiovascular reactivity in response to stress is predictive of hypertension and coronary heart disease (CHD) (Fredrikson & Matthews, 1990; Krantz & Manuck, 1984). Delayed cardiovascular recovery to emotional stressors may contribute to cardiovascular disease in a similar manner. For instance, it has been found that high levels of anger, hostility, and stress have been associated with decreased vagal rebound (Mezzacappa, Kelsey, Katkin, & Sloan, 2001), slower systolic blood pressure, and pre-ejection period (Neumann, Waldstein, Sollers, Thayer, & Sorkin, 2004) during recovery and poorer recovery of diastolic blood pressure and high-frequency heart rate variability (Key, Campbell, Bacon, & Gerin, 2008). Furthermore, delayed cardiovascular recovery after a psychological stressor has been shown to predict real life blood pressure (Trivedi, Sherwood, Strauman, & Blumenthal, 2008).

Hostility has been found to be associated with trait rumination (Neumann, Waldstein, Sollers, Thayer, & Sorkin, 2001). Trait ruminators are conceptualized as those individuals who focus their attention on negative thoughts and emotions and are prone to feelings of revenge (Caprara, 1986). These characteristics are also consistent with descriptions of hostile individuals who may be more prone to ruminate (Neumann et al., 2004). However, rumination is not adequately captured in current measures of hostility (Cook & Medley, 1954). Trait rumination may also have a differential effect on cardiovascular reactivity and recovery that may help further explain the relationship between hostility and cardiovascular disease.

Trait rumination may help to explain the association between cardiovascular recovery and disease because it may prolong the psychological and physiological responses to anger provocation (Neumann et al., 2004). Furthermore, distraction may inhibit the psychological and physiological responses to stress thereby decreasing cardiovascular reactivity and risk for disease (Neumann et al., 2004). The current study on extant data aims to establish whether there is a relation between trait rumination and risk for cardiovascular disease, independent of traditional measures of hostility. Results will further our understanding of the potentially harmful effects of cognitive coping styles on cardiovascular function, above and beyond current knowledge of the negative prospective association between hostility and cardiovascular disease. This investigation may also provide evidence for possible treatment techniques (i.e. distraction) to reduce the potentially harmful effects of rumination on cardiovascular function.

Epidemiology of Cardiovascular Disease

Since the year 1900, cardiovascular disease has contributed to more illness and death than any other disease or incident (Rosamond et al., 2007). Data from 2004 revealed that cardiovascular disease was the underlying cause of 36.3% of all deaths. More than 147,000 of these deaths were in individuals under the age of 65. An estimated 2,400 people die from cardiovascular disease each day. Approximately 79.4 million Americans have one or more types of cardiovascular disease. Nearly 72 million individuals suffer from high blood pressure. Over 15 million are afflicted with coronary heart disease and over 5 million have been affected by a stroke. In addition, the direct and indirect health care costs of cardiovascular disease in 2007 have been estimated to be 431.8 billion dollars (Rosamond et al., 2007). Therefore, it is vital to investigate the causes and course of cardiovascular disease to establish more effective methods of treatment and prevention.

The devastating consequences of cardiovascular disease have led to an abundance of research into its etiology. Biological risk factors that have been found to predict cardiovascular disease such as cigarette smoking, obesity, and high cholesterol levels only account for approximately 50% of the variance (Brand, Rosenman, Sholtz, & Friedman, 1976). Thus, a number of researchers have started to examine psychological and behavioral risk factors that may account for a substantial proportion of the unexplained variance. For example, depression has been found to be an independent risk factor in the development of a wide range of cardiovascular diseases (Van der Kooy et al., 2007). Likewise, hostility has been found to prospectively predict cardiovascular

disease (Hecker, Chesney, Black, & Frautshci, 1988; Shekelle, Gale, Ostfeld, & Paul, 1983; Koskenvuo et al., 1988).

Cardiovascular Reactivity

The association of psychosocial factors, such as hostility, to cardiovascular disease may in part be mediated by cardiovascular reactivity. Cardiovascular reactivity refers to “an individual’s propensity to experience cardiovascular reactions of greater or lesser magnitude, in relation to those of other persons, when encountering behavioral stimuli experienced as engaging, challenging, or aversive” (Manuck, 1994, p. 7). There are substantial differences in individuals’ cardiovascular reactivity to stress, which appear to be relatively stable over time (Kamarck, Jennings, Pogue-Geile, & Manuck, 1994). These individual differences may be important because there is strong evidence that cardiovascular overreactivity is a potential risk factor for the development of atherosclerosis, hypertension, and coronary heart disease (Gidron, Kupper, Kwajitall, Winter & Denollet, 2007; Manuck, 1994; Fredrikson & Matthews, 1990; Johnston, Tuomisto, & Patching, 2008).

Research indicates that negative emotions, such as hostility, may lead to parasympathetic and sympathetic nervous system dysregulation (Bleil, Gianaros, Jennings, Flory, & Manuck, 2008). These cardiovascular responses are often measured by examining increases in heart rate, systolic and diastolic blood pressure that are associated with psychological stress (Kamarck et al., 1989). Researchers also commonly measure autonomic dysregulation using heart rate variability (HRV). This is computed by conducting a spectral analysis of the variability in interbeat intervals of HR. The power components derived from the spectral analysis are characterized by the area under

the curve for a particular frequency range. The high-frequency power component (HFHRV) is an indication of parasympathetic control over HR in the range of normal adult respiration (0.15-0.40 Hz) (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). There is ample prospective evidence that attenuated parasympathetic control over cardiovascular activity, which is measured by decreased resting HFHRV, is predictive of cardiovascular morbidity and mortality (Bigger, Fleiss, Rolnitzky, Steinman, 1993; Huikuri, et al., 1999; Tsuji et al., 1996).

It has long been suspected that dysregulated physiological activity to stress is indicative of CHD risk. Physicians in the 1930's posited that an exaggerated blood pressure response to placing the hand or foot in ice water was predictive of increased hypertension risk (Hines & Brown, 1932). Cardiovascular reactivity may lead to the development of cardiovascular disease through a number of pathways. For example, hypertension is thought to result from increased cardiovascular reactivity via changes in blood flow which leads to increased vascular resistance. This, in turn, may cause exaggerated responses of vascular tissue growth factors to rises in blood pressure. The increase in vascular tissue growth factors along with increased pressure and wall tension may lead to permanent increases in peripheral resistance via thickening of the blood vessel wall (Lovallo & Gerin, 2003). Cardiovascular reactivity may contribute to the development of atherosclerosis through other pathways. Vascular wall and endothelial shear stresses interacting with platelet activity (Markovitz & Matthews, 1991), cortisol responses (Balsalobre et al., 2000), and lipid levels have all been implicated as underlying mechanisms (Hajjar & Nicholson, 1995).

Researchers often employ HRV to examine the relation between negative emotions and autonomic dysregulation. For example, several studies have demonstrated that patients with cardiovascular disease who have depressive and anxious disorders have suppressed HRV and HFHRV when compared to controls (Carney, et al., 2001; Lavoie, et al., 2004; Pitzalis et al., 2001). The association between anger and HRV is less clear and is often limited to samples of healthy individuals. Furthermore, research is mixed in regards to the direction of this association and is further complicated by different anger inducements (e.g. anger expression and inhibition) as well as demographic variables used in the studies (e.g. men and women) (Horsten, et al., 1999; Ramaekers, Ector, Demyttenaere, Rubens, & Van de Werf, 1998; Sloan et al., 1994; Virtanen, et al., 2003).

The current study extends the research to date by examining other psychological constructs related to anger (i.e. hostility and rumination) as well as capturing a more dynamic view of cardiovascular function via impedance cardiography. Impedance cardiography is used to examine the underlying functional components of cardiovascular responses such as stroke volume, cardiac contractility, and total peripheral resistance (Kamarck et al., 1989).

Cardiovascular Poststress Recovery

Until recently most of the research examining the effects of stressors on cardiovascular function has focused on cardiovascular reactivity. However, the correlation of cardiovascular reactivity with psychological measures and cardiovascular disease is often modest (e.g., Manuck, 1994). Furthermore, cardiovascular reactivity occurs at approximately the same level for both positive and negative emotions (Jacob et al., 1999) and thus one might assume that both positive and negative emotions would lead

to negative cardiovascular outcomes. However, research reveals that only negative emotions are consistently related to prolonged cardiovascular responses (Brosschot and Thayer, 2003).

As a consequence of the above findings a number of researchers have hypothesized that the association of psychosocial factors and cardiovascular disease may be further understood in terms of individual differences in cardiovascular recovery after exposure to a psychological stressor. Cardiovascular recovery is defined as “either the time required to return to pretask baseline levels after termination of a stressor or the degree of elevation above pretask baseline levels within a predetermined post-task interval” (Stewart & France, 2001, p. 106).

Prolonged cardiovascular recovery has been implicated as a potential risk factor in the development of cardiovascular disease (Mezzacappa et al., 2001; Brosschot & Thayer, 1998; Hocking-Schuler & O’Brien, 1997; Earle, Linden & Weinberg, 1999). Early support for this association was derived from research demonstrating that hypertensive individuals had slower cardiovascular recovery following a laboratory stressor (Falkner & Kushner, 1989; Fredrikson & Engel, 1985). Additionally, it has been found that healthy children of individuals with hypertension also have prolonged cardiovascular recovery following a psychological stressor (Anderson, Lane, Taguchi, & Williams, 1989; Anderson, Lane, Taguchi, Williams & Houseworth, 1989).

Evidence of an association between cardiovascular recovery and exercise also yields support for the use of cardiovascular recovery as an indication of cardiovascular health. It has been shown, for example, that individuals who are aerobically trained have accelerated cardiovascular recovery following mental stressors (Sinyor, Golden, Steiner,

& Seraganian, 1986; Sinyor, Schwartz, Peronnet, Brisson, & Seraganian, 1983). Such findings suggest that cardiovascular recovery may mediate the beneficial association between fitness and cardiovascular health (Schuler & O'Brien, 1997). Other evidence of the relation of cardiovascular recovery to CHD is found in studies of stressful life events. The association of stressful life events and risk for CHD suggests that the relation of cardiovascular recovery to stressful life events may be an indicator of CHD risk (Pardine & Napoli, 1983; Schuler & O'Brien, 1997). It has also been postulated that slowed cardiovascular recovery to daily stressors may induce physiological strain on the cardiovascular system, which may lead to the development of CHD (Hart & Jamieson, 1983).

It has been theorized that prolonged cardiovascular recovery may be indicative of chronic sympathetic activity, which may lead to a decrease of beta-adrenergic receptors in the cardiovascular system (Amerena & Julius, 1995; Hart & Jamieson, 1983; Pollack & Obrist, 1988). This down-regulation of beta-adrenergic receptors may cause permanent suppression of cardiac output as well as an increase in peripheral vascular resistance, causing chronic high blood pressure (Amerena & Julius, 1995). It has been further hypothesized that when the body's normal response to stress is prolonged the cardiovascular system will be overexposed to chemical mediators, such as cortisol, which can lead to long-term damage (McEwen, 2006). That is, chronic overexposure to stress hormones, elevated blood pressure, and heart rate may contribute to damage of the blood vessels and abnormal inflammatory responses which over time may enhance the probability of cardiovascular accidents such as strokes and heart attacks. In fact, prolonged cardiovascular recovery of blood pressure is predictive of long-term changes

in blood pressure and cardiovascular regulation (Stewart & France, 2001; Trivedi, et al., 2008).

Hostility and Cardiovascular Disease

Psychosocial factors, such as hostility, can have a negative influence in the development and prognosis of cardiovascular disease (Kubzansky, Davidson, & Rozanski, 2005; Shekelle, Gale, Ostfeld, & Paul, 1983; Koskenvuo et al., 1988). Tendencies toward anger and confrontational behavior have long been postulated to be risk factors for CHD (Osler, 1901). Research was originally inspired by Friedman and Rosenman's (1959) description of the Type A behavior pattern, which consisted of a combination of an excessive time orientation, competitiveness, a propensity toward hostile behavior, and aggressiveness. This personality construct has been prospectively associated with the development of cardiovascular disease (Woodall & Matthews, 1989). Furthermore, the Western Collaborative Group Study found that individuals who exhibited Type A personality characteristics were at more than twice the risk of developing cardiovascular disease than those who did not exhibit this personality pattern (Dembroski, MacDougall, Costa, & Grandits, 1989).

Although early research on the cardiovascular effects of Type A personality appeared conclusive, later studies did not obtain the same results (Dembroski et al., 1989; Shekelle et al., 1985). Thus, researchers started investigating specific components of the Type A construct. Findings from this subsequent research have indicated that hostility may be the part of the Type A behavior pattern that is related to the development of cardiovascular disease (e.g., Dembroski et al., 1989; Smith, 1992). In support of this conclusion data from early work on Type A behavior was reanalyzed to yield a

significant association between hostility and cardiovascular disease (Dembroski et al., 1989).

There have been several disagreements about the conceptual definition of hostility. These definitions often blur the psychological, emotional and behavioral aspects of hostility. For example, hostility has been defined as “a set of negative attitudes, beliefs, and appraisals concerning others” (Smith, 1992). According to Smith (1992), this definition implies that the hostile individual views others as untrustworthy and as sources of frustration, provocation, and maltreatment. Other researchers have defined dispositional hostility as “a cynical, suspicious, and resentful attitude toward others, often leading to negative social exchanges and more opportunities to experience anger” (Kubzansky et al., 2005, p. 12). The current study utilizes the Cook-Medley hostility scale, which is derived from the MMPI. According to the authors, this measure captures the psychological, behavioral, and emotional aspects of hostility (Cook & Medley, 1954).

Several studies have demonstrated an association between hostility and negative cardiovascular outcomes. More specifically, hostility has been associated with carotid atherosclerosis (Knox et al., 2000; Matthews, Owens, Kuller, Sutton-Tyrrell & Jansen-McWilliams, 1998) and coronary artery calcification (Iribarren et al., 2000). Furthermore, a predictive relationship has been found between hostility and coronary heart disease morbidity and mortality (Barefoot, Dahlstrom & Williams, 1983), increased coronary events (i.e. myocardial infarction) (Chaput et al., 2002), and decreased survival time of patients diagnosed with coronary artery disease (Boyle et al., 2004).

It has been hypothesized that the relation between hostility and cardiovascular disease may partially be mediated by cardiovascular and neuroendocrine overreactivity (Smith, 1992). That is, individuals with greater levels of dispositional hostility show exaggerated increases in blood pressure, heart rate, and stress hormones when faced with a stressor. Furthermore, hostile individuals may be prone to experiencing more frequent and severe episodes of anger and may be more vigilant due to mistrust. These characteristics may lead to increased psychophysiological reactivity. This repeated enhanced reactivity over time may then lead to the development of cardiovascular disease and may also impair immune function.

There have been inconsistent findings from research attempting to clarify the association between hostility and cardiovascular reactivity. Some studies using nonsocial stressors, such as the Stroop color-word task, mental arithmetic, and cold pressor have demonstrated a relation between hostility and cardiovascular reactivity (Smith, 1992). However, a number of other studies using nonsocial stressors have found that hostility is not associated with cardiovascular reactivity (Glass, Lake, Contrada, Kehoe & Erlanger, 1983; Kamarck, Manuck & Jennings, 1990).

Conversely, studies using stressors of an interpersonal nature that arouse anger and other related emotions tend to consistently demonstrate that hostile individuals have exaggerated cardiovascular reactivity. Specifically, a number of studies have revealed that hostile individuals produce greater reactivity during a stressful word-identification task only when it is combined with harassment (Suarez, Harlan, Peoples & Williams, 1993; Suarez & Williams, 1989). Mixed findings in this area may also be partially explained by the inadequate measurement of cognitive response styles, such as

rumination, that may prolong both emotional and cardiovascular recovery from a stressor (Gerin, Davidson, Christenfeld, Goyal, & Schwartz, 2006; Glynn, Christenfeld, Gerin, 2007).

Suarez & Williams (1989) reported that anger-evocation may lead to prolonged cardiovascular recovery in individuals with high levels of hostility in addition to eliciting increased cardiovascular reactivity during the stressor. Thus, cardiovascular reactivity and recovery phases have been postulated to be distinct processes which likely result from differing physiological mechanisms (Gerin et al., 2006). Cardiovascular recovery, in particular, may help explain the association between hostility and cardiovascular disease. Neumann et al. (2004) found that individuals with higher levels of dispositional hostility had slower recovery on several cardiovascular measures following an anger-recall task. Despite the association between cardiovascular reactivity, recovery and disease, the underlying physiological (e.g. hemodynamic patterns) and psychological (e.g. emotions, cognitions) processes of cardiovascular reactivity and recovery are not fully understood. Thus, further research is needed in this area, which should include the examination of techniques employed by individuals to regulate their emotions. Two relatively common responses to negative emotions, rumination and distraction, will be examined in the current study.

Rumination and Distraction

Despite the apparent relation between hostility and cardiovascular function, a number of studies have failed to find an association (e.g., Leon, Finn, Murray & Bailey, 1988; O'Malley, Jones, Feuerstein & Taylor, 2000). Thus, it may be important to examine mood regulation strategies in conjunction with negative mood. Mood regulation

strategies are used by individuals to maintain, eliminate, or change emotions (Thayer, Newman & McClain, 1994). Rumination and distraction are two opposing strategies that are commonly utilized by individuals to modulate their negative emotions.

Rumination refers to “thoughts and behaviors that focus the individual’s attention on the negative mood, the causes and consequences of this mood, and self-evaluations related to the mood” (Rusting & Nolen-Hoeksema, 1998, p. 790). Rumination is distinct from constructive problem-solving because there is a tendency to perseverate on the consequences and causes of one’s anger, other negative emotions, and the event without thoughts about how to change the distressing situation (Lyubomirsky & Nolen-Hoeksema, 1995). Evidence for this differentiation is derived from research demonstrating that individuals with higher levels of rumination engage significantly less in problem-solving (Lang, 1984; Larsen & Diener, 1992).

Rumination is thought to occur when there is a discrepancy between an individual’s goals and what is actually occurring, and an individual may continue to ruminate until the goal has been met or disregarded (Thomsen, 2006). Wañke and Schmid (1996) postulated that when there is a perceived lack of control about this goal discrepancy there is an even greater tendency to ruminate. Rumination has also been theorized to occur when a discrepancy exists between events and an individual’s schema and may work to decrease this discrepancy by adapting schema or fitting the event into preexisting schema (Clark, 1996; Tait & Silver, 1989).

Several negative consequences of rumination have been identified. Individuals with higher levels of rumination report more negative emotions in general (Seegerstrom, Tsao, Alden, & Craske, 2000). Ruminators have also been found to have less control

over their intrusive thoughts than those low in rumination (Watkins, 2004). Rumination has also been associated with more chronic symptoms of depression (Nolen-Hoeksema, Morrow, & Fredrickson, 1993) as well as more frequent episodes of depression (Nolen-Hoeksema, 2000). A literature review by Lyubomirsky and Tkach (2003) highlights several other consequences of high levels of rumination including decreased motivation, impaired inhibition, higher levels of stress, poor health behaviors, difficulties in social relationships, as well as poor concentration, cognition, and problem solving. Brain imaging studies have revealed that participants with higher levels of rumination have increased amygdala activation after being shown negative pictures and words (Ray et al., 2005; Siegle, Steinhauer, Thase, Stenger, & Carter, 2002). In addition, high ruminators exhibit greater cortisol release following a stressor (Roger & Jamieson, 1988).

The current study examines angry rumination, which is the same as rumination defined by Rusting and Nolen-Hoeksema (1998), but with a focus on anger. Research on angry rumination is relatively scarce compared to the wealth of research to date that has focused on depressive rumination. However, most preliminary findings suggest angry rumination has negative consequences similar to depressive rumination. It has been found that men diagnosed with depression tend to ruminate on anger and injustice (Cochran & Rabinowitz, 2000; Cochran & Rabinowitz, 2003). Depression has also been shown to be associated with rumination about the fear of getting angry and/or hurting others (Brody, Haag, Kirk, & Solomon, 1999). Sukhodolsky, Golub, and Cromwell (2001) investigated the consequences of several different aspects of angry rumination including vengeful thoughts, angry afterthoughts, and angry memories. The researchers found that all of these aspects of angry rumination were associated with a decrease in life

satisfaction. Furthermore, the engagement in angry rumination following an anger-recall task is predictive of prolonged recovery of blood pressure (Gerin et al., 2006). Angry rumination may also lead to the development and maintenance of anger-control problems (Novaco, 1979).

Despite the conceptual distinction between anger-focused and depression-focused rumination it has been found that they are associated with each other and that anger rumination also contributes to depression (Gilbert, Cheung, Irons, & McEwan, 2005). Of note, the Gilbert et al. (2005) study did not find an association between vengeful thoughts and depression, perhaps indicating that these types of thoughts empower the individual which protects against the helplessness associated with depression. Angry rumination may also be linked to depression through its association with shame (Gilbert et al., 2005). Shame has been shown to be associated with and predictive of depressive symptoms (Andrews, Qian, & Valentine, 2002; Gilbert, 2000; Gilbert et al., 2005). Further, shameful thoughts are associated with both angry and depressive rumination (Gilbert et al., 2005; Tangney, Wagner, Barlow, Marschall, & Gramzow, 1996).

Several mechanisms have been identified for how rumination may contribute to depression. Rumination has been shown to mediate risk factors of depression such as self-criticism, neediness, and negative cognitive styles (Spasojevec & Alloy, 2001). Rumination has also been shown to affect autobiographical and other memories in a way that promotes depression (Teasdale & Green, 2004; Watkins & Teasdale, 2004). Rumination may also be predictive of depression through its association with maladaptive coping skills, negative content, and attentional focus (Nolen-Hoeksema, Grayson, & Larson, 1999; Nolen-Hoeksema, Morrow, & Fredrickson, 1993). Symptoms

associated with depression may also increase rumination. For example, social isolation may provide more opportunity for ruminative thought cycles which may exasperate depression (Gilbert et al., 2005).

Distraction, in contrast to rumination, “involves focusing attention away from the mood and its causes onto pleasant or neutral stimuli that are engaging enough to prevent the mind from wandering back to the source of negative affect” (Rusting & Nolen-Hoeksema, 1998, p. 790). Distraction may reduce negative mood by delaying thoughts about the causes of that mood (Bahrke & Morgan, 1978). Furthermore, distraction may work by occupying an amount of working memory that may otherwise be used to engage in rumination (Eber & Tesser, 1992). Distraction may also function by increasing an individual’s engagement in pleasurable activities (Trask & Sigmon, 1999). Distraction is also thought to promote thoughts and behaviors centered on problem-solving (Lyubomirsky & Nolen-Hoeksema, 1993). In fact, those who repeatedly think about an emotional event have a longer and/or more intense emotional experience in comparison to those who are distracted (Morrow & Nolen-Hoeksema, 1990). Furthermore, engaging in a distracting activity has been shown to attenuate depressive episodes (Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksema, Morrow, & Fredrickson, 1993).

Individuals have varying degrees of propensity toward using rumination.

Rumination-dissipation can be understood in terms of two opposing sides on a continuum of a postulated personality dimension (Caprara, 1986). Those with a tendency toward decreased rumination and accelerated dissipation are able to dismiss negative affect and the wish to retaliate against transgressors. On the other end of the continuum are those who tend to have relatively more delayed dissipation and increased rumination. These

individuals may be more prone to retaining thoughts and feelings of anger. High-ruminators-low dissipaters have characteristics similar to descriptions of individuals with high levels of hostility (Kubzansky, 2005). It is possible, then, that individuals with high levels of hostility may have a greater propensity to ruminate than individuals with lower levels of hostility.

Research suggests that those high in rumination may experience greater levels of distress for longer periods following the onset of a negative emotion compared to people low in rumination (Lyubomirsky & Nolen-Hoeksema, 1995). Accordingly, these researchers posit that during ruminative states, negative cognitions and memories are more prominent which may exaggerate negative mood and inhibit adaptive problem solving skills. In fact, there is evidence that rumination leads to increased levels of anger, while distraction decreases anger (Rusting & Nolen-Hoeksema, 1998). This research suggests that the use of distraction as a mood regulating strategy leads to more adaptive problem solving and prevents the exaggeration of negative mood.

As an extension of these findings, it is hypothesized that rumination about a stressor may prolong cardiovascular recovery. In support of this hypothesis, a number of studies have found that rumination is associated with slower blood pressure and heart rate variability recovery following a psychological stressor (Key et al., 2008; Glynn, Christenfeld, & Gerin, 2002; Gerin et al., 2006). Thus, it can be hypothesized that rumination may extend the psychophysiological effects of a stressor by perpetuating negative cognitions about the stressor even after its termination. Trait rumination may also be associated with increased cardiovascular reactivity through its shared variance

with other psychosocial factors that have been shown to increase reactivity, such as hostility (Neumann et al., 2004).

Rumination is not adequately captured in current measures of hostility despite the fact that it may be an important dimension of hostility. Differential levels of trait rumination that were not controlled for in past research may help explain the mixed findings of the cardiovascular effects of hostility. That is, it may not be an individual's propensity toward hostility that has a negative impact on their cardiovascular health. Rather, an individual's tendency to remain in a negative state after an anger inducing event may prolong cardiovascular recovery which may have a more damaging consequence.

Trait rumination may have an independent effect on cardiovascular reactivity and recovery, above and beyond current measures of hostility and anger. Determining the cardiovascular effects of rumination may help further explain the relationship between hostility and cardiovascular disease. Therefore, it is important to evaluate rumination as part of the cognitive component of hostility and its effects on cardiovascular responses to stress should be examined. In order to do so, the current study aims to examine the effects of rumination on cardiovascular function independent of hostility.

Distraction, on the other hand, may work to decrease or prevent rumination and thereby potentially reduce the possible deleterious effects of negative emotions on cardiovascular function. In fact, distraction has been shown to accelerate cardiovascular recovery especially in individuals engaging in ruminative responses (Neumann et al., 2001; Glynn et al., 2002; Gerin et al., 2006). In addition, distraction may be an effective strategy to reduce anger (Rusting & Nolen-Hoeksema, 1998) and has been shown to be

one of the most effective techniques in reducing negative emotions (Thayer, Newman, & McClain, 1994).

Statement of the Problem

Dispositional hostility has been associated with the development and prognosis of cardiovascular disease. However, several studies have failed to find this association (e.g., Leon, Finn, Murray & Bailey, 1988; O'Malley, Jones, Feuerstein & Taylor, 2000). Rumination, a proposed cognitive component of hostility, may enhance cardiovascular reactivity and prolong recovery, above and beyond current and commonly used measures of hostility. This may further clarify the relation between hostility and cardiovascular disease. In addition, distraction may reduce the potentially harmful effects of rumination on cardiovascular function by preventing or decreasing the sustainment of negative emotions and accelerating cardiovascular recovery.

The current study aims to examine the relation between trait rumination, above and beyond a commonly used measure of hostility, and cardiovascular reactivity to recovery from a personally-relevant anger-recall task. This study also investigates the potentially beneficial effects of distraction in decreasing rumination and its associated maintenance of cardiovascular arousal following a stressor. In order to do so, a comparison will be made between a standard recovery period and a recovery period in which the participant engages in a distracting activity.

As compared to prior studies in this area, the data was collected in the present study in a more psychometrically sound and ecologically valid manner. Past studies have assumed that during the recovery period participants were engaged in rumination (e.g., Haynes, Gannon, Orimoto, O'Brien, & Brandt, 1991). This study, on the other hand,

assessed the frequency of rumination during the recovery period to validate that the strategy was actually being used. In addition, the present study achieved greater ecological validity by having participants express their emotions verbally rather than using imagery (Siegman, Dembroski, & Ringel, 1990) and by having them recall personally-relevant events rather than exposing them to generic films or scripts.

The present study gained a relatively more thorough depiction of hemodynamic changes during exposure to the stressor and poststress recovery than prior studies in this area. This was obtained through the use of impedance cardiography (Sherwood, Allen, Fahrenber, Kelsey, Lovallo, & van Doornen, 1990). This technique is a non-invasive measurement of continuous hemodynamic adjustments (e.g. cardiac output and total peripheral resistance) that cause blood pressure fluctuations.

The current study also uses a more sophisticated method to assess cardiovascular recovery than prior studies. Excursion measures or area under the curve establish a more reliable estimate of cardiovascular activity by utilizing all relevant data points, instead of any single point. This technique is also superior to those used in prior studies because measurements can be calculated independently of cardiovascular reactivity measures by covarying reactivity in the analyses.

Participants in the current study were limited to women for several reasons. Very little research in the area of hostility and cardiovascular function has been focused on women despite evidence of differential findings. For example, one study found that hostile men had a stronger cardiac deceleration to a stress task than women (Ruiz, Uchino, & Smith, 2006). Thus, it is important to establish a body of research that is specific to women because results from men may not always generalize to them.

Furthermore, women have been found to engage in ruminative processes more often than men (Nolen-Hoeksema, Larson, & Grayson, 1999) thus providing a more robust sample of the cognitive construct.

This study sets forth four specific hypotheses:

1. Trait rumination will predict greater cardiovascular reactivity during the anger-recall task and prolonged cardiovascular recovery after the task, independent of dispositional hostility.
2. Participants in the distraction condition will experience accelerated cardiovascular recovery as compared to those in the standard recovery period.
3. Trait rumination and distraction will interactively predict cardiovascular recovery measures. Specifically, individuals with higher levels of trait rumination will experience a greater benefit (i.e. accelerated cardiovascular recovery) from distraction than individuals low in trait rumination and those high in trait rumination in the no distraction condition.
4. Individuals with higher levels of trait rumination will have greater peripheral vascular resistance responses (i.e., total peripheral resistance) and decreased or more variable cardiac responses (i.e., cardiac output and stroke volume) compared to those with lower levels of trait rumination during both the anger-recall task and poststress recovery.

METHOD AND PROCEDURE

Participants

Participants consisted of eighty female university students (18-30 years) who were recruited from the University of Maryland, Baltimore County (UMBC) via advertisement flyers and introductory psychology classes at UMBC. The sample included individuals of Caucasian (54%), African-American (34%), and Asian-American (12%) ethnicities. Participants had a body mass index less than 30 kg/m² and according to self-report did not smoke. Participants had no medical history of cardiovascular disease, diabetes, or psychiatric disorders by self-report. They also denied using any prescribed medications (including oral contraceptives) or over-the-counter medication that may impact cardiovascular function within two months prior to their participation in the study. Participants were asked not to consume any caffeine for 12 hours before the study in order to avoid its effects on cardiovascular responses (Green, Kirby, & Suls, 1996). Participants were also asked not to use any alcohol for 24 hours before the study.

Questionnaires

Dissipation-Rumination Scale (DRS)

An individual's dispositional tendency to engage in rumination following an emotional stressor was measured by the Dissipation-Rumination Scale (Caprara, 1986) (see Appendix A). Only a relatively small number of studies to date have examined the relation between rumination and cardiovascular function (e.g. Key et al., 2008; Glynn, Christenfeld, & Gerin, 2002; Gerin et al., 2006). Among these studies, several different measures of rumination have been used. The current study chose the DRS because it focuses on angry rumination and has strong psychometric properties.

The DRS is a six-position Likert-type scale (i.e., 0=completely false for me, 1=fairly false for me, 2=false to a certain extent, 3=true to a certain extent, 4=fairly true for me, 5=completely true for me) consisting of 20 items. Low dissipaters-high ruminators obtain higher total scores than high dissipaters-low ruminators. The scale was administered to 366 Italian university students (136 females) and 291 university students from the United States (80 females) to determine the scales internal consistency, test-retest reliability, and construct validity (Caprara, 1986). This study concluded that the scale has relatively good internal consistency, with reported alpha coefficients ranging from 0.79 to 0.87 (Caprara, 1986). Its test-retest (24-hour interval) correlation was estimated to be 0.81 (Caprara, 1986). Evidence for the scales construct validity was established by determining that high ruminators, according to the scale, were more likely to retaliate (administer higher voltage shocks to a confederate) following an insult than low ruminators (Caprara, 1986). Similarly, another study found that low dissipaters-high ruminators, as indicated by scores on the DRS, were more likely to retaliate aggressively following provocation (Collins & Bell, 1997).

Cook and Medley Hostility Scale (Ho)

The MMPI-based Ho scale was used to measure dispositional hostility (Cook & Medley, 1954) (Copyrighted material not included in Appendix). Research has demonstrated that this scale measures various aspects of interpersonal hostility including aggressive behavior directed at others, beliefs about others' trustworthiness, and negative emotions related to interpersonal interactions (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989). The scale may also contain items that measure social avoidance and neuroticism.

This measure is commonly used to examine the effects of hostility on cardiovascular function (e.g. Barefoot, Dahlstrom, & Williams, 1983; Shekelle, Gale, Ostfeld, Paul, 1983). Such research was inspired by the original finding that scores on the Ho scale were associated with coronary artery disease as evidenced by angiography results (Williams et al., 1980). There are several studies that support the Ho scale's ability to predict negative health outcomes. Barefoot and colleagues (1983) found that the Ho scale was predictive of CHD events at a 30-year follow-up. A prospective study of 1877 middle-aged men found that Ho scores were predictive of CHD events after a 20-year follow up period (Shekelle, Gale, Ostfeld, & Paul, 1983). Furthermore, Ho scores have been found to predict all cause mortality at follow-ups (Shekelle et al., 1983; Barefoot et al., 1983). However, there are also several studies that have failed to find this association (e.g. Leon, Finn, & Bailey, 1987; McCranie, Watkins, Brandsma, & Sisson, 1986). Based on the established use of the Ho scale in studies examining the impact of hostility on cardiovascular function the current study also used this scale in order to make more direct comparisons with the results of past research.

The Ho scale consists of 50 true-false items. Higher levels of dispositional hostility are indicated by greater total scores on this scale. Two separate samples consisting of 85 and 135 male and female undergraduate students were recruited to establish the psychometric properties of the Ho scale (Smith & Frohm, 1985). Results of this study indicated that the Ho scale has good internal consistency with estimates of Chronbach's alphas averaging around 0.80 (Smith & Frohm, 1985). The Smith and Frohm (1985) study also found that the Ho scale was significantly correlated with self-reports of anger, supporting its construct validity, as well as other measures of hostility,

supporting its criterion validity (Smith & Frohm, 1985). One and four year test-retest correlations of more than $r = .80$ have been demonstrated in samples of medical students and middle-aged adults (Barefoot, Dahlstrom, & Williams, 1983; Shekelle, Gale, Ostfeld, & Paul, 1983).

The State-Trait Anger Expression Inventory (STAXI)

The STAXI is a measure of experience, expression, and control of anger (Spielberger, 1988) (Copyrighted material not included in Appendix). The current study administered the STAXI in order to assess the level of state anger that was present following the anger-recall task as well as during and after the recovery period. The STAXI was chosen for use in this study because of the strong reliability and validity of the scale.

Only the state anger subscale (S-Anger) was used in the final analyses. This subscale consists of 10 items on a four point frequency scale (i.e., 1= “not at all”, 2= “somewhat”, 3= “moderately so”, and 4= “very much so”). It purportedly measures the intensity of anger that is occurring “right now, at this moment” or at another appointed time. The normative data for this scale are based on 1,900 individuals comprised of normal adults (N=1,644; 977 females) and hospitalized psychiatric inpatients (N=276; 105 females). From this data, the Cronbach alpha of the S-Anger subscale was estimated to be about 0.84, indicating good internal consistency (Spielberger, 1988). The construct validity of this subscale has been demonstrated in studies that have shown individuals score higher on the S-Anger subscale when completing frustrating tasks as compared to neutral tasks (Spielberger, 1988).

Revised Impact of Events Scale (IES)

Two types of cognitive coping styles, rumination and avoidance, are assessed by the IES (Horowitz, Wilner, & Alvarez, 1979) (See Appendix B). Seven of the items (i.e., intrusion subset = 1, 4, 5, 6, 10, 11, and 14) from this scale were used to determine the degree of intrusive thoughts about the anger-recall task that participants experienced during the recovery period (Horowitz et al., 1979). Higher scores indicate more frequent intrusive thoughts. The avoidance subset from this scale was also administered but is not a primary concern in the present study. The IES is setup so that any event can be used in the items as *it*. “The anger recall task” was substituted for the word *it* in the present study. Participants were also asked to rate how often they experienced thoughts about the angry event during the recovery period on a 5-point scale (0=never, 4=very often).

Horowitz and colleagues (1979) administered the IES to 66 outpatient adults who had recently undergone a stressful life event. The researchers reported that the intrusion subset of the IES had a test-retest (1 week interval) reliability of 0.89 (Horowitz et al., 1979). In addition, the internal consistency of this subscale was reported to be good with a Chronbach’s alpha of 0.78 (Horowitz et al., 1979). Evidence for the scale’s construct validity was demonstrated in participants with stress response syndromes who attained lower IES scores after receiving 4 months of psychotherapy (Horowitz et al., 1979).

Cardiovascular Measures

A Critikon Dinamap Vital Signs Monitor was used to measure systolic (SBP) and diastolic blood pressure (DBP), and mean arterial pressure (MAP) [model 8100: Critikon (Johnson & Johnson), Tampa, FL]. Electrocardiogram (ECG) measurements of heart rate were obtained from two electrodes attached bilaterally to the chest. A Hewlett-Packard Contract Transducer (Model # 21050A) on the second intercostal space on the left sternal

border was used to obtain heart sounds. Grass biological amplifiers were used to filter and amplify the ECG and heart sound signals. Noninvasive estimates of preejection period, stroke volume, left ventricular ejection time, cardiac output, and total peripheral resistance were gathered using impedance derived signals (dZ/dt) [IFM Minnesota Impedance Cardiograph, model 304B].

Further descriptions of the measures from impedance cardiography will follow (Sherwood et al., 1990):

Stroke volume index (SI) - the volume of blood ejected by the left ventricle in one heart beat (or cardiac cycle) adjusted for body surface area [Range = 30-65 (ml/beat/m²)].

Cardiac index (CI) – the volume of blood pumped by the left ventricle in one minute adjusted for body surface area [Range = 2.6-4.2 (L/min/m²)].

Preejection period (PEP) – the time difference between the onset of left ventricular ejection and the onset of electrical systole. PEP indirectly measures beta-adrenergic activation of the heart [Range = 100-130 (msec)].

Total peripheral resistance (TPR) – the resistance to blood flow caused by vasoconstriction which increases viscosity between the blood and the blood vessel [Range = 700-1600 (dynes/cm⁵/s)].

Heart rate variability is a measure of beat-to-beat variations in heart rate. Time and frequency domain measures of HRV were estimated in the current study. Time domain analyses provide the root mean of successive differences in R-R intervals (r-MSSD) and heart rate. According to the Task Force guidelines (Task Force, 1996), spectral analyses were performed on the beat-to-beat intervals derived from the electrocardiogram (ECG) data collection to obtain both low-frequency (LF: 0.04-0.15

Hz) and high-frequency (HF: 0.15-0.40 Hz) components using an autoregressive algorithm. LF and HF power are thought to measure different autonomic nervous system influences. HF power is thought to be an indicator of respiratory-modulated parasympathetic (vagal) outflow, while LF power measures baroreceptor-mediated regulation of blood pressure (Berntson, 1997; Friedman & Thayer, 1998) which is regulated primarily by sympathetic control and varying amounts of parasympathetic influences. To further clarify sympathetic influences on the heart, a LF/HF ratio is computed, which is thought to be a measure of sympathovagal balance (Malliani, Lombardi, Pagani, & Cerutti, 1990).

Procedures

Participants completed a two-hour laboratory session. They were compensated with \$10.00 and/or two credits toward their final introductory psychology grade. Participants were informed that if they chose to discontinue at any time they would still receive their compensation.

Each session began with the participants reading and signing a consent form (approved by the UMBC Institutional Review Board). The height and weight of each participant was then measured and impedance bands, ECG electrodes, heart sound microphone, and a blood pressure cuff (on nondominant arm) were attached for cardiovascular monitoring. Two impedance bands were placed around the neck and two were placed around the chest one inch below the xiphoid process (i.e., tetrapolar band-electrode configuration) (Sherwood et al., 1990).

Throughout the session participants sat in a comfortable chair in a sound-attenuated, temperature controlled room. Participants underwent a fifteen-minute

baseline period, a three-minute anger recall task, and a ten-minute recovery period. Cardiovascular monitoring occurred during the last six minutes of the baseline period and throughout the task and recovery periods. Blood pressure was measured in ninety-second intervals during the baseline period and in sixty-second intervals during the task and recovery periods, according to established criteria (Debski et al., 1991). Cardiovascular signals – ECG, heart sounds, dZ/dt (first derivative of the change in thoracic impedance), and Z_0 (basal thoracic impedance) – were continuously collected using computerized analog to digital conversion at 1000 samples per second (Debski et al., 1991).

A personally-relevant recall task was used to elicit anger. Instructions for this task were modified from those adapted by Waldstein et al. (2000) (see Appendix C). Each participant was asked to remember and talk about an event that occurred within the last year that made them angry, frustrated, irritated, or upset. This task lasted for three minutes and was followed by a ten-minute recovery period. During recovery, participants were randomly assigned using a random number table to experience a distraction technique (i.e. reading an article about the possibility of life in outer space. See Weinberger, Schwartz, & Davidson, 1979) or a standard recovery period (i.e., no distraction). After the recovery period the state anger scale was administered. Participants gave a retrospective self-report of state anger to both the task and recovery periods after the end of the recovery period. At this time, the participant also completed the Cook and Medley Ho Scale and the Dissipation-Rumination Scale. Participants were also asked questions (written specifically for this study) about the amount of time they spent reading the article and thinking about the anger recall task during the recovery period (see Appendix D).

Upon completion of data collection the participant was unhooked from the equipment and given a debriefing about similar research findings and the goals and rationale of this study. The participants were then offered an opportunity to ask any questions and were compensated for their time. Finally, they were asked not to discuss the details of the experiment with others until the completion of the project to reduce demand characteristics.

Data Reduction

Blood pressure data (i.e., SBP and DBP) during the baseline period and the anger recall task were averaged. That is, the last three blood pressure readings were averaged for the baseline period. The three blood pressure readings obtained during the recall task were also averaged. Computer software designed at the University of Pittsburgh (Debski et al., 1991) was used to ensemble-average and score ECG and impedance waveforms in 30 second intervals for the baseline, task, and recovery periods. Stroke volume was calculated using a value of 135 ohm*cm for blood resistivity ($SV = LVET * dZ/dT * 12 / z_0^2 * \rho$) (Kubicek, Karnegis, Patterson, Witsoe, & Mattson, 1966). Cardiac output was calculated as $(HR * SV) / 1000$. Stroke volume index (SI) and cardiac index (CI) were calculated by dividing SV and CO by body surface area, in order to adjust for individual differences in body mass [$weight (kg)^{.425} \times height (cm)^{.725} \times .007184$]. Total peripheral resistance was computed with the equation $MAP / CO \times 80$. Systolic time intervals, PEP and LVET, were coded in the following millisecond intervals: the Q-wave of the digitized ECG to the B-point of the dZ/dt waveform; the B-point to X-wave of the dZ/dT waveform (i.e., coincident with the closure of the aortic valve – the second heart sound). Measures obtained from impedance cardiography data

were averaged for the baseline and task periods. Arithmetic change scores or delta scores (task value-baseline value) were computed as an index of task-induced SBP, DBP, PEP, SI, CI, and TPR response during the recall task (Llabre, Spitzer, Saab, Ironson, Schneiderman, 1991). The final analyses for the anger recall task used these physiological change scores.

A recovery slope is the slope and form of cardiovascular variables (i.e., the units of change in a variable per unit of time) and the functional form (e.g., linear, parabolic of that change) (Haynes et al., 1991). An excursion measure, which accounts for the recovery slope, was used to calculate the area under the curve minus the baseline mean during the recovery period for each cardiovascular measure from each participant. This measure is derived by the Trapezoidal Rule [Excursion = $0.5 * \text{fixed time interval} (\text{measure 1} + 2 * \text{measure 2} + 2 * \text{measure 3} + \dots + \text{last measure}) - (\text{baseline mean of measure} * \text{fixed time interval})$] (Neumann et al., 2001).

Data Analyses

Trait Rumination and Distraction were the primary predictor variables while the main dependent variables were SBP, DBP, HR, PEP, SI, CI, TPR, LF, HF, and LF-HF. Before investigating the hypotheses set forth by this study several preliminary analyses were conducted.

Pearson r and Point-Biserial correlations and analyses of variance (ANOVAs) were conducted to examine the relationship of resting heart rate and blood pressure and sample characteristics, including education, age, BMI, alcohol and caffeine consumption, family history of hypertension, and ethnicity, to psychological characteristics and to cardiovascular measures. If associations were found to be significant, those variables

were included as covariates in multiple regression analyses of cardiovascular reactivity and recovery. Independent samples t-tests were also conducted to establish if there were differences among the control and distraction groups on age, BMI, alcohol intake, caffeine intake, resting cardiovascular measures, trait rumination, dispositional hostility, or state anger and state rumination during the task.

Individuals high in trait anger and hostility often have higher levels of trait rumination which is likely to be used as a coping mechanism. To validate this relationship Pearson r correlations were conducted on estimates of trait rumination (DRS) and measures of trait anger (STAXI scales). For further descriptive purposes, Pearson r correlations were also conducted to examine the relations among state and trait rumination, hostility, and S-Anger. That is, scores from the DRS, the Cook and Medley Ho scale, the revised IES, and the S-Anger subscale from the STAXI were intercorrelated.

In order to check the manipulation of the distraction condition, Pearson r correlations of the time participants spent reading during the recovery period (item 1 of the Reading Manipulation Check) and cardiovascular recovery measures were conducted.

To determine whether trait rumination influenced cardiovascular reactivity during the anger recall task (Hypotheses 1 & 4), multiple regression analyses were conducted on SBP, DBP, HR, PEP, SI, CI, TPR, LF, HF, LF-HF arithmetic change scores with Trait Rumination as the predictor variable. Respective baseline cardiovascular means were controlled for in these analyses. Dispositional hostility was also entered as a covariate in these regression analyses. Multiple regression analyses were also performed with Trait Rumination, Distraction and their interaction as predictors to determine their effects on

cardiovascular recovery (including the excursions of SBP, DBP, HR, PEP, SI, CI, TPR, LF, HF, and LF-HF) (Hypotheses 1, 2, 3, 4). Respective cardiovascular reactivity means during the anger task and dispositional hostility were controlled for in these analyses. Any sample characteristics found to be significantly associated with cardiovascular measures or psychological characteristics in the preliminary analyses were also included as covariates.

RESULTS

Power Analysis

A power analysis was conducted using Trait Rumination, Distraction, and their interaction as predictor variables and SBP, DBP, HR, PEP, SI, CI, and TPR as the dependent variables. A power analysis was conducted for each primary predictor and dependent variable using effect sizes obtained from Neumann et al. (2001) (see Tables 1, 2, 3, and 4 for results). Results from the power analysis indicated that for power = .80, $\alpha = 0.05$, and a medium effect size ($R = 0.35$), a total number of 77 participants are required. Eighty participants were collected in order to create two equal manipulation groups.

Table 1
Power Calculations for Predicting Cardiovascular Responses to Trait Rumination During the Task Period (Effect Sizes from Neumann, 2001)

Cardiovascular Measure	Effect Size (d)	Total Number of Participants Needed (N)
SBP	0.04	197
DBP	0.06	131
HR	0.25	34
PEP	0.15	55
CI	0.06	133
SI	0.32	27
TPR	0.10	81

Table 2
Power Calculations for Predicting Cardiovascular Responses to Trait Rumination During the Recovery Period (Effect Sizes from Neumann, 2001)

Cardiovascular Measure	Effect Size (d)	Total Number of Participants Needed (N)
SBP	0.14	59
DBP	0.04	197
HR	0.37	24
PEP	0.04	197
CI	0.41	22
SI	0.08	101
TPR	0.05	159

Table 3
Power Calculations for Predicting Cardiovascular Responses to Distraction During the Recovery Period (Effect Sizes from Neumann, 2001)

Cardiovascular Measure	Effect Size (d)	Total Number of Participants Needed (N)
SBP	0.05	159
DBP	0.04	197
HR	0.24	35
PEP	0.24	35
CI	0.11	74
SI	0.18	46
TPR	0.04	197

Table 4
Power Calculations for Predicting Cardiovascular Responses to Trait Rumination X Distraction During the Recovery Period (Effect Sizes from Neumann, 2001)

Cardiovascular Measure	Effect Size (d)	Total Number of Participants Needed (N)
SBP	0.09	90
DBP	0.05	159
HR	0.18	46
PEP	0.20	42
CI	0.06	133
SI	0.28	31
TPR	0.23	37

Sample Demographics

Pearson r and Point-Biserial correlations as well as ANOVAs were conducted to analyze the relation of sample characteristics (i.e., age, BMI, education, parental history of hypertension, and self-reported average alcohol, caffeine consumption, and ethnicity) to trait rumination, hostility, and cardiovascular measures (see Tables 5, 6, and 7 for results).

Table 5
Means and Standard Deviations For Demographic, Psychosocial Measures, and Cardiovascular Measures by Distraction Condition

	All		Control Group		Distraction Group	
	<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>
<u>Demographics</u>						
Age (years)	19.0	1.5	19.0	1.6	19.0	1.5
BMI (kg/m ²)	23.1	3.3	22.5	3.4	23.6	3.0
Education (years)	12.7	1.3	12.7	1.4	12.7	1.0
Caffeine Intake (8oz. drinks/day)	1.1	0.9	1.2	0.9	1.2	0.9
Alcohol Intake (Drinks/week)	0.8	1.6	0.6	1.2	0.9	1.9
Family History of Hypertension						
Positive (at least one parent)	41%					
Negative (neither parent)	59%					
<u>Psychosocial Measures:</u>						
Hostility	20.8	7.6	21.2	7.9	20.5	7.6
State Anger (Baseline)	10.3	0.8	10.4	0.9	10.3	0.7
State Anger (Task)	18.7	6.1	18.6	5.7	18.0	6.5
State Anger (Recovery)	12.9	4.1	14.1*	4.5	11.8*	3.5
State Rumination (Task)	12.0	4.4	12.4	3.9	11.7	4.8
State Rumination (Recovery)	10.5	5.5	11.5 ⁺	5.6	9.5 ⁺	5.4
Trait Rumination	37.6	13.9	38.5	13.6	37.0	14.7
<u>Cardiovascular Measures (Baseline)</u>						
SBP (mmHg)	106	7.1	106	5.3	107	8.5
DBP (mmHg)	57	6.1	58	5.0	56	6.9
HR (bpm)	77	10.7	78	10.9	75	10.5
PEP (msec)	104.4	12.2	103.8	12.0	105.1	12.7
CI (L/min/m ²)	4.2	0.9	4.3	0.8	4.2	1.0
SI (ml/beat/m ²)	58	15.9	57	14.0	58	17.9
TPR (dynes/cm ⁵ /s)	918	231.5	917	198	919	264
LF (Hz)	648.2	539.2	570.1	418.2	735.1	649.7
HF (Hz)	1262.7	1419.7	1034.4	1211.3	1516.3	1617.8

* $p < 0.01$, ⁺ $p < 0.06$

Table 6
Pearson Product Moment Correlations of Demographic Variables with Trait Rumination, Dispositional Hostility, and Cardiovascular Change Scores and Excursion Measures

	Age (years)	Ethnicity	BMI (kg/m ²)	Education (years)	Caffeine Intake (8oz. drinks/day)	Alcohol (Drinks/wk)
<u>Trait Rumination</u>	-0.05	0.04	0.23*	-0.04	0.04	-0.03
<u>Dispositional Hostility</u>	-0.05	0.09	0.23*	-0.10	0.03	0.02
<u>Task:</u>						
SBP (mmHg)	0.10	-0.26*	-0.15	-0.08	0.03	-0.10
DBP (mmHg)	-0.07	-0.21	-0.94	-0.03	0.06	0.06
HR (bpm)	0.17	-0.17	-0.22*	0.08	0.02	0.02
PEP (msec)	-0.18	0.20	0.23*	0.05	0.11	0.15
CI (L/min/m ²)	0.31*	-0.14	-0.18	0.10	-0.05	0.08
SI (ml/beat/m ²)	0.06	0.17	0.13	0.01	0.02	0.01
TPR (dynes/cm ⁵ /s)	-0.16	0.03	0.15	-0.06	-0.03	0.03
LF (Hz)	-0.18	-0.04	0.22	-0.10	0.26	0.13
HF (Hz)	0.08	0.11	0.23	-0.03	0.19	-0.20
<u>Recovery:</u>						
SBP (mmHg)	-0.05	-0.13	-0.17	-0.20	0.04	-0.03
DBP (mmHg)	-0.06	-0.03	0.16	0.04	-0.03	0.10
HR (bpm)	0.11	-0.02	-0.02	0.07	0.08	0.08
PEP (msec)	0.17	0.05	0.15	0.02	-0.01	0.19
CI (L/min/m ²)	0.06	-0.10	-0.27*	0.14	0.13	-0.08
SI (ml/beat/m ²)	0.17	-0.11	-0.28*	0.16	-0.02	0.07
TPR (dynes/cm ⁵ /s)	0.01	0.15	0.21	-0.10	0.03	-0.08
LF (Hz)	-0.06	-0.18	-0.21	-0.12	-0.13	-0.03
HF (Hz)	0.06	0.08	0.09	-0.01	0.11	-0.24

* $p < 0.05$

Table 7
Pearson Product Moment Correlations of Baseline Cardiovascular Measure with Cardiovascular Reactivity Measures

	Baseline								
	SBP (mmHg)	DBP (mmHg)	HR (bpm)	PEP (msec)	CI (L/min/m ²)	SI (ml/beat/m ²)	TPR (dynes/cm ⁵ /s)	LF (Hz)	HF (Hz)
SBP (mmHg)	0.13								
DBP (mmHg)		0.18							
HR (bpm)			-0.29*						
PEP (msec)				-0.15					
CI (L/min/m ²)					-0.18				
SI (ml/beat/m ²)						-0.58*			
TPR (dynes/cm ⁵ /s)							0.37*		
LF (Hz)								0.45**	
HF (Hz)									0.35*

* $p < 0.05$, ** $p < 0.01$

Results of the Pearson r correlations revealed a significant positive correlation between CI during the task and age ($r = 0.31, p < .05$). Significant positive correlations were also found between BMI and hostility ($r = 0.23, p < .04$), trait rumination ($r = 0.23, p < .04$), and resting SBP ($r = 0.43, p < .001$). BMI was found to be negatively correlated to resting CI ($r = -0.30, p < .007$) as well as CI ($r = -0.24, p < .04$) and HR ($r = -0.23, p < .05$) reactivity during the anger recall task. Ethnicity was found to be negatively correlated to SBP reactivity during the task ($r = -0.26, p < .02$). There were also significant negative correlations between resting HR and HR during the task ($r = -0.23, p < .05$) and resting SI and SI reactivity ($r = -0.58, p < .001$). A positive correlation was also found between resting TPR and TPR reactivity ($r = 0.37, p < .001$).

Based on these correlations BMI and the baseline cardiovascular measures were included as covariates in the multiple regression analyses for cardiovascular reactivity

and recovery. Age and education were associated with the cardiovascular measures and the predictors, but not to a significant degree. To err on the side of caution, age and education were also included as covariates in the regression analyses. No significant associations were found between family history of hypertension and cardiovascular measures.

An ANOVA was conducted with ethnicity on cardiovascular reactivity and recovery. Ethnicity was only significantly correlated to SBP during task and thus it was included in the regression analyses predicting SBP during task. Due to the absence of a main effect for ethnicity on all the other cardiovascular measures as well as being limited by the small subgroup sample size, ethnicity was not included as a covariate in the final regression analyses in general.

To examine whether there were any significant differences between the control and distraction groups on age, alcohol intake, BMI, caffeine intake, resting cardiovascular measures, dispositional hostility, state anger, trait rumination, and state rumination an independent samples t-test was conducted (see Table 5 for descriptives). Results indicated that there were no significant differences between the two groups on these measures.

Manipulation Checks

In order to determine the associations between hostility, state and trait rumination, state anger, and the amount of time spent reading (Question 2 in Appendix G) and thinking (Questions 1 in Appendix G) during the recovery period, Pearson r correlations were conducted. That is, intercorrelations were conducted among scores from the Dissipation-Rumination Scale, the revised IES, the Cook and Medley Ho scale,

the S-Anger scale from the STAXI for the task and recovery intervals, and the Manipulation Check items (Question 1 and 2 from Appendix G) (see Tables 8-10 for results).

Table 8
Pearson Product Moment Correlations of Dispositional Hostility, Trait Rumination, State Anger, and State Rumination and Cardiovascular Change Scores During the Task

	Trait Rumination	Dispositional Hostility	State Anger		State Rumination	
			Task	Recovery	Task	Recovery
Trait Rumination	-----	0.54*	-----			
<u>State Anger</u>						
Task	0.57*	0.20 ⁺	-----			
Recovery	0.27*	0.12	0.41*	-----		
<u>State Rumination</u>						
Task	0.36*	0.13	0.48*	0.37*	-----	
Recovery	0.31*	0.12	0.44*	0.58*	0.59	-----
<u>Task Change Scores:</u>						
SBP (mmHg)	-0.10	-0.12	0.09	-----	0.10	-----
DBP (mmHg)	-0.06	-0.03	0.10	-----	0.11	-----
HR (bpm)	-0.32*	-0.27*	-0.06	-----	-0.06	-----
PEP (msec)	-0.03	0.15	0.04	-----	0.07	-----
CI (L/min/m ²)	0.01	-0.06	-0.06	-----	-0.09	-----
SI (ml/beat/m ²)	0.31*	0.15 ⁺	0.03	-----	-0.06	-----
TPR (dynes/cm ⁵ /s)	-0.07	-0.004	0.12	-----	0.10	-----
LF (Hz)	0.21	0.09	0.15	-----	0.10	-----
HF (Hz)	0.17	0.13	0.04	-----	-0.05	-----

* $p < 0.05$, ⁺ $p < 0.10$

Table 9
Pearson Product Moment Correlations of Trait Rumination, Dispositional Hostility, State Anger, and State Rumination and Cardiovascular Excursion Measures During Recovery

	Trait Rumination	Dispositional Hostility	State Anger		State Rumination	
			Task	Recovery	Task	Recovery
<u>Control Condition</u>						
SBP (mmHg)	0.03	0.23	0.17	0.24	-----	0.31 ⁺
DBP (mmHg)	-0.08	-0.10	0.06	-0.16	-----	0.21
HR (bpm)	-0.43*	-0.18	-0.22	-0.13	-----	-0.20
PEP (msec)	0.17	-0.06	-0.08	-0.10	-----	-0.08
CI (L/min/m ²)	0.38*	-0.17	-0.12	-0.03	-----	0.26 ⁺
SI (ml/beat/m ²)	0.08	-0.04	-0.14	-0.05	-----	-0.22
TPR (dynes/cm ⁵ /s)	0.19	0.05	-0.39*	0.32 ⁺	-----	0.44*
<u>Distraction Condition</u>						
SBP (mmHg)	-0.03	0.13	0.16	0.20	-----	0.26 ⁺
DBP (mmHg)	0.06	0.18	0.17	-0.26	-----	0.003
HR (bpm)	-0.21	0.07	-0.13	0.08	-----	0.13
PEP (msec)	0.01	0.11	-0.03	-0.20	-----	0.01
CI (L/min/m ²)	0.34*	-0.19	-0.23	0.02	-----	0.03
SI (ml/beat/m ²)	-0.23	-0.13	-0.07	0.04	-----	-0.02
TPR (dynes/cm ⁵ /s)	0.27	0.25	-0.25	-0.01	-----	0.10
<u>Both Conditions</u>						
SBP (mmHg)	0.001	0.18*	0.16	0.24*	-----	0.20*
DBP (mmHg)	0.005	0.06	0.12	-0.18	-----	0.12
HR (bpm)	-0.29*	-0.03	-0.15	-0.10	-----	-0.08
PEP (msec)	0.02	0.004	-0.04	-0.21	-----	-0.08
CI (L/min/m ²)	0.37*	-0.16 ⁺	-0.18	0.03	-----	-0.08
SI (ml/beat/m ²)	-0.09	-0.06	-0.11	0.05	-----	-0.07
TPR (dynes/cm ⁵ /s)	0.10	0.13	-0.31*	0.13	-----	0.24*

* $p < 0.05$, ⁺ $p < 0.10$

Table 10
Pearson Product Moment Correlations among State Anger, State Rumination, and Cardiovascular Measures During Recovery with the Amount of Time Spent Reading During Recovery, the Amount of Time Spent Thinking About the Angry Event During Recovery, and the Distraction Condition

	Reading	Thinking	Distraction Condition
Thinking	-0.19	-----	-----
Trait Rumination	0.05	0.04	-----
Dispositional Hostility	0.21	0.05	-----
<u>State Anger</u>			
Task	-0.24	0.43*	-----
Recovery	-0.40*	0.54*	-----
<u>State Rumination</u>			
Task	-0.28	0.23	-----
Recovery	-0.41*	0.55*	-----
<u>Recovery Excursions</u>			
SBP (mmHg)	-0.43*	0.23	-0.11
DBP (mmHg)	0.04	0.11	-0.05
HR (bpm)	-0.09	-0.27 ⁺	0.26*
PEP (msec)	0.36*	-0.13	0.23*
CI (L/min/m ²)	-0.29 ⁺	0.05	-0.16 ⁺
SI (ml/beat/m ²)	-0.31 ⁺	0.06	-0.17 ⁺
TPR (dynes/cm ⁵ /s)	-0.61	0.02	0.03
LF (Hz)	0.23	0.20	-0.14
HF (Hz)	0.39	0.11	0.07

* $p < 0.05$, ⁺ $p < 0.10$

Results revealed that trait rumination was positively and significantly related to state anger both during the task ($r = 0.57, p < .001$) and during recovery ($r = 0.27, p < .01$). Trait rumination was also positively correlated with state rumination both during the task ($r = 0.36, p < .001$) and during recovery ($r = 0.31, p < .004$). In addition, trait rumination was positively correlated to dispositional hostility ($r = 0.54, p < .001$). State anger during the task was related to state anger during recovery ($r = 0.41, p < .001$), with time spent thinking about the angry event during recovery ($r = 0.43, p < .002$), and with state rumination during recovery ($r = 0.44, p < .001$). State anger during recovery was positively associated with state rumination during the task ($r = 0.37, p < .001$) and during

recovery ($r = 0.58, p < .001$). State anger during recovery was also negatively related to time spent reading the article during recovery ($r = -0.49, p < .004$) and positively related to time spent thinking about the angry event during recovery ($r = 0.54, P < .001$). State rumination during the task was associated with state rumination during recovery ($r = 0.59, p < .001$). Finally, state rumination during recovery was negatively associated with amount of time spent reading the article ($r = -0.53, p < .001$) and positively correlated with amount of time spent thinking about the angry event during the recovery period ($r = 0.55, p < .001$).

To examine whether reading was a successful distracter to reduce anger and state rumination (see Table 5 for descriptives), independent samples t-tests were performed with the Distraction condition as the independent variable and state anger and state rumination during recovery as the dependent variables. A marginally significant difference was found between the groups on state rumination during recovery, $t(78) = 1.93, p < .057$, such that individuals in the control condition reported higher levels of state rumination about the angry event than individuals in the distraction condition. Further, a significant difference was found between the groups on state anger during recovery, $t(78) = 2.61, p < .01$, such that those participants in the control group had greater levels of anger than those participants in the distraction group.

In order to examine the associations among state anger and state rumination with cardiovascular reactivity and recovery Pearson r correlations were conducted (see Tables 8-10 for results). State anger was found to be significantly related to SBP ($r = 0.24, p < .04$) during recovery. State rumination was correlated with SBP ($r = 0.30, p < .007$) and TPR ($r = 0.24, p < .05$) during recovery.

In order to determine the effectiveness of the distraction condition, Pearson r correlations were conducted on the time spent reading during recovery (Question 1 in Appendix G) and the cardiovascular recovery excursions. The amount of time spent reading during recovery was associated with longer PEP ($r = 0.43, p < .01$), lower SBP ($r = -0.53, p < .001$), lower SI ($r = -0.44, p < .01$), and lower CI ($r = -0.42, p < .02$) (see Table 10).

Repeated measures ANOVAs (Time: Baseline to Task) were computed on all cardiovascular measures to determine if the anger recall task produced significant cardiovascular reactions. It was found that all cardiovascular measures except PEP were significantly changed from baseline to task (see Table 11).

Table 11
Repeated Measures ANOVAs of Time (Baseline to Task) on Cardiovascular Measures

		F	Df	<i>p</i>	partial η^2
Cardiovascular Measures					
SBP	(mmHg)	302.22	(1, 79)	0.0001*	.793
DBP	(mmHg)	333.45	(1, 79)	0.0001*	.808
HR	(bpm)	214.17	(1, 77)	0.0001*	.736
PEP	(msec)	0.66	(1, 77)	0.42	.009
CI	(L/min/m ²)	4.74	(1, 77)	0.032*	.058
SI	(ml/beat/m ²)	134.87	(1, 77)	0.0001*	.637
TPR	(dynes/cm ⁵ /s)	115.01	(1, 77)	0.0001*	.602
LF	(Hz)	14.11	(1, 37)	0.001*	.276
HF	(Hz)	5.46	(1, 37)	0.025*	.128

* $p < 0.05$

Multiple Regression Analyses

Prediction of Cardiovascular Reactivity by Trait Rumination, Controlling for

Dispositional Hostility

Zero-order correlations were conducted to determine the relations of trait rumination and dispositional hostility to cardiovascular reactivity measures (see Table 9

for results). Trait rumination was found to be significantly related to lower HR ($r = -0.32, p < .002$) and greater SI ($r = 0.31, p < .003$) responses.

Multiple regression analyses were performed to examine the effects of trait rumination on cardiovascular reactivity during the anger recall task, controlling for dispositional hostility. Specifically, SBP, DBP, HR, PEP, SI, CI, TPR, LF, HF and LF-HF arithmetic change scores were used as the dependent variables and trait rumination was inserted as the independent variable. Dispositional hostility, BMI, age, education, and respective baseline cardiovascular measures were entered as covariates. It was found that trait rumination significantly predicted increased reactivity of SI ($\beta = 0.27, p < .03, pr^2 = .261$), above and beyond dispositional hostility (see Table 12 and Figure 1).

Table 12

Multiple Regression Analyses for Trait Rumination Predicting Cardiovascular Reactivity Responses During the Anger Recall Task, Controlling for Dispositional Hostility

	Criterion	β	p	pr^2
SBP	(mmHg)	-0.025	0.852	.0090
DBP	(mmHg)	-0.023	0.870	.0004
HR	(bpm)	-0.187	0.150	.0289
PEP	(msec)	-0.187	0.153	.0292
CI	(L/min/m ²)	0.081	0.547	.0052
SI	(ml/beat/m ²)	0.276	0.027*	.0681
TPR	(dynes/cm ⁵ /s)	-0.145	0.280	.0169
LF	(Hz)	0.230	0.269	.0392
HF	(Hz)	0.078	0.626	.0003
LF-HF	(Hz)	0.076	0.734	.0046

* $p < 0.05$

Prediction of Cardiovascular Recovery by Trait Rumination and Distraction, Controlling for Dispositional Hostility

Pearson r and Point-Biserial correlations were conducted to determine the associations of trait rumination, distraction condition, and dispositional hostility on cardiovascular recovery measures. Trait rumination was positively related to CI ($r =$

0.37, $p < .001$) and negatively related to HR ($r = -0.29$, $p < .006$) during recovery.

Distraction was positively related to HR ($r = 0.26$, $p < .01$), and PEP ($r = 0.23$, $p < .02$) excursions, and negatively associated with SI ($r = -0.17$, $p < .07$) recovery. Finally, dispositional hostility was found to be marginally associated with SBP ($r = 0.18$, $p < .053$) and CI ($r = -0.16$, $p < .092$) recovery (see Tables 9 and 10 for results).

Multiple regression analyses were computed to examine the effects of Trait Rumination, Distraction, and their interaction on cardiovascular recovery from the anger recall task (i.e., excursions of SBP, DBP, HR, PEP, SI, CI, TPR, LF, HF, and LF-HF), controlling for dispositional hostility. Age, BMI, education, respective cardiovascular reactivity means, and dispositional hostility were entered as covariates in these analyses (see Tables 13-15 for results and Tables 16-19 for descriptives). Results revealed that trait rumination independently predicted slower recovery of SI ($\beta = 0.55$, $p < .001$, $pr^2 = .135$) (see Figure 1) and HR ($\beta = -0.52$, $p < .007$, $pr^2 = .106$) (see Figure 2), controlling for dispositional hostility. Distraction was found to independently predict faster post-task recovery of SBP ($\beta = -0.25$, $p < .03$, $pr^2 = .069$) (see Figure 3), DBP ($\beta = -0.23$, $p < .05$, $pr^2 = .053$) (see Figure 4), HR ($\beta = 0.23$, $p < .05$, $pr^2 = .059$) (see Figure 5), PEP ($\beta = 0.24$, $p < .05$, $pr^2 = .055$) (see Figure 6), SI ($\beta = -0.25$, $p < .02$, $pr^2 = .071$) (see Figure 7), and LF ($\beta = -0.29$, $p < .04$, $pr^2 = .133$) (see Figure 8), controlling for dispositional hostility. The interaction of trait rumination and distraction independently predicted accelerated cardiovascular recovery of SI ($\beta = -0.37$, $p < .01$, $pr^2 = .084$) (see Figure 9) and CI ($\beta = -0.31$, $p < .04$, $pr^2 = .057$) (see Figure 10), controlling for dispositional hostility. More specifically, it was found that individuals with high trait rumination experienced a greater

acceleration of SI and CI recovery from the distraction condition than individuals with low trait rumination.

Table 13

Multiple Regression Analyses for Trait Rumination Predicting Cardiovascular Recovery Responses (2, 5, and 10 minutes) Following the Anger Recall Task, Controlling for Dispositional Hostility

Criterion	β	p	pr^2
<u>2 minutes</u>			
SBP (mmHg)	0.014	0.914	.0002
DBP (mmHg)	-0.076	0.582	.0042
HR (bpm)	-0.182	0.150	.0286
PEP (msec)	-0.159	0.257	.0177
CI (L/min/m ²)	-0.132	0.276	.0161
SI (ml/beat/m ²)	0.258	0.037*	.0586
TPR (dynes/cm ⁵ /s)	0.052	0.677	.0025
LF (Hz)	-	-	-
HF (Hz)	-	-	-
LF-HF (Hz)	-	-	-
<u>5 minutes</u>			
SBP (mmHg)	-0.013	0.918	.0001
DBP (mmHg)	-0.206	0.134	.0303
HR (bpm)	-0.228	0.073 ⁺	.0441
PEP (msec)	-0.057	0.677	.0025
CI (L/min/m ²)	-0.379	0.005*	.1050
SI (ml/beat/m ²)	0.199	0.144	.0289
TPR (dynes/cm ⁵ /s)	0.158	0.225	.0207
LF (Hz)	-	-	-
HF (Hz)	-	-	-
LF-HF (Hz)	-	-	-
<u>10 minutes</u>			
SBP (mmHg)	-0.109	0.373	.0110
DBP (mmHg)	-0.040	0.774	.0012
HR (bpm)	-0.344	0.014*	.0858
PEP (msec)	0.019	0.889	.0003
CI (L/min/m ²)	-0.380	0.005*	.1063
SI (ml/beat/m ²)	-0.060	0.686	.0023
TPR (dynes/cm ⁵ /s)	0.155	0.264	.0188
LF (Hz)	0.286	0.121	.0756
HF (Hz)	0.112	0.400	.0228
LF-HF (Hz)	0.053	0.575	.0102

* $p < 0.05$, ⁺ $p < 0.10$

Table 14
Multiple Regression Analyses for Distraction Condition Predicting Cardiovascular Recovery Responses (2, 5, and 10 minutes) Following the Anger Recall Task, Controlling for Dispositional Hostility

Criterion	β	p	pr^2
<u>2 minutes</u>			
SBP (mmHg)	-0.244	0.026*	.0671
DBP (mmHg)	-0.203	0.078 ⁺	.0424
HR (bpm)	0.091	0.389	.0104
PEP (msec)	-0.044	0.708	.0019
CI (L/min/m ²)	-0.073	0.469	.0072
SI (ml/beat/m ²)	-0.142	0.168	.0259
TPR (dynes/cm ⁵ /s)	-0.057	0.585	.0042
LF (Hz)	-	-	-
HF (Hz)	-	-	-
LF-HF (Hz)	-	-	-
<u>5 minutes</u>			
SBP (mmHg)	-0.138	0.187	.0237
DBP (mmHg)	-0.222	0.058 ⁺	.0484
HR (bpm)	0.162	0.127	.0320
PEP (msec)	0.216	0.059 ⁺	.0492
CI (L/min/m ²)	-0.078	0.494	.0064
SI (ml/beat/m ²)	-0.260	0.020*	.0718
TPR (dynes/cm ⁵ /s)	0.028	0.796	.0010
LF (Hz)	-	-	-
HF (Hz)	-	-	-
LF-HF (Hz)	-	-	-
<u>10 minutes</u>			
SBP (mmHg)	-0.094	0.365	.0114
DBP (mmHg)	-0.104	0.378	.0108
HR (bpm)	0.245	0.039*	.0610
PEP (msec)	0.229	0.051 ⁺	.0534
CI (L/min/m ²)	-0.128	0.265	.0174
SI (ml/beat/m ²)	-0.186	0.113	.0353
TPR (dynes/cm ⁵ /s)	0.082	0.489	.0072
LF (Hz)	-0.231	0.101	.0847
HF (Hz)	-0.090	0.378	.0253
LF-HF (Hz)	-0.017	0.822	.0017

* $p < 0.05$, ⁺ $p < 0.10$

Table 15
Multiple Regression Analyses for Trait Rumination X Distraction Predicting Cardiovascular Recovery Responses (2, 5, and 10 minutes) Following the Anger Recall Task, Controlling for Dispositional Hostility

Criterion	β	p	pr^2
<u>2 minutes</u>			
SBP (mmHg)	-0.119	0.462	.0077
DBP (mmHg)	-0.220	0.189	.0246
HR (bpm)	0.222	0.159	.0282
PEP (msec)	0.242	0.180	.0256
CI (L/min/m ²)	-0.309	0.042*	.0571
SI (ml/beat/m ²)	-0.372	0.013*	.0841
TPR (dynes/cm ⁵ /s)	0.293	0.064 ⁺	.0488
LF (Hz)	-	-	-
HF (Hz)	-	-	-
LF-HF (Hz)	-	-	-
<u>5 minutes</u>			
SBP (mmHg)	0.056	0.719	.0018
DBP (mmHg)	-0.009	0.956	.00005
HR (bpm)	0.244	0.117	.0346
PEP (msec)	-0.077	0.666	.0027
CI (L/min/m ²)	-0.122	0.460	.0077
SI (ml/beat/m ²)	-0.251	0.130	.0320
TPR (dynes/cm ⁵ /s)	0.178	0.285	.0166
LF (Hz)	-	-	-
HF (Hz)	-	-	-
LF-HF (Hz)	-	-	-
<u>10 minutes</u>			
SBP (mmHg)	0.051	0.745	.0015
DBP (mmHg)	0.088	0.614	.0036
HR (bpm)	0.255	0.137	.0331
PEP (msec)	-0.731	0.695	.0023
CI (L/min/m ²)	-0.034	0.836	.0006
SI (ml/beat/m ²)	-0.250	0.142	.0313
TPR (dynes/cm ⁵ /s)	0.089	0.607	.0041
LF (Hz)	-0.019	0.917	.0004
HF (Hz)	0.043	0.760	.0032
LF-HF (Hz)	-0.022	0.829	.0016

* $p < 0.05$, ⁺ $p < 0.10$

Table 16
Marginal Means and Standard Errors for Baseline Cardiovascular Measures by Rumination Groups

Cardiac Measures		Rumination			
		Low		High	
		<u>M</u>	<u>SE</u>	<u>M</u>	<u>SE</u>
SBP	(mmHg)	106.20	1.15	106.02	1.05
DBP	(mmHg)	56.27	1.06	57.24	0.97
HR	(bpm)	78.84	2.27	77.22	2.44
PEP	(secs)	105.48	2.99	104.78	3.01
CI	(L/min/m ²)	3.99	0.21	4.50	0.20
SI	(ml/beat/m ²)	52.56	3.17	59.69	3.27
TPR	(dynes/cm ⁵ /s)	955.67	44.97	807.91	47.59
LF	(Hz)	603.20	128.06	716.15	134.79
HF	(Hz)	1091.97	345.04	1494.07	363.17
LF-HF	(Hz)	1.05	0.27	1.05	0.30

Table 17
Marginal Means and Standard Errors for Cardiovascular Reactivity by Rumination Groups

Cardiac Measures		Rumination			
		Low		High	
		<u>M</u>	<u>SE</u>	<u>M</u>	<u>SE</u>
SBP	(mmHg)	120.20	1.64	121.77	1.50
DBP	(mmHg)	71.58	1.46	72.78	1.34
HR	(bpm)	93.43	2.57	90.04	2.75
PEP	(secs)	104.45	2.92	103.07	2.94
CI	(L/min/m ²)	3.90	0.23	4.27	0.23
SI	(ml/beat/m ²)	41.56	2.97	48.58	3.07
TPR	(dynes/cm ⁵ /s)	304.74	46.64	232.98	49.36
LF	(Hz)	1320.99	340.10	1498.56	357.97
HF	(Hz)	648.25	251.67	795.54	264.90
LF-HF	(Hz)	4.55	1.33	5.17	1.45

Table 18
Marginal Means and Standard Errors for Cardiovascular Recovery by Rumination Group

Rumination Group		Recovery		
		2-min(SE)	5-min(SE)	10-min(SE)
Low	SBP(mmHg)	108.76(1.34)	107.61(1.23)	106.60(1.14)
High	SBP(mmHg)	109.14(1.23)	107.91(1.13)	107.65(1.04)
Low	DBP(mmHg)	55.64(1.59)	55.19(1.33)	52.23(1.42)
High	DBP(mmHg)	59.33(1.88)	59.47(1.88)	58.87(1.67)
Low	HR(bpm)	82.02(2.45)	82.27(2.39)	81.64(2.13)
High	HR(bpm)	79.00(2.63)	78.39(2.56)	79.80(2.28)
Low	PEP(secs)	102.65(3.39)	105.14(3.19)	105.54(3.21)
High	PEP(secs)	103.47(3.42)	104.29(3.22)	106.01(3.23)
Low	CI(L/min/m ²)	4.08(0.21)	4.06(0.23)	4.15(0.22)
High	CI(L/min/m ²)	4.40(0.21)	4.43(0.23)	4.35(0.22)
Low	SI(ml/beat/m ²)	49.57(3.09)	49.84(3.30)	50.39(2.96)
High	SI(ml/beat/m ²)	57.18(3.18)	57.46(3.47)	56.62(3.05)
Low	TPR(dynes/cm ⁵ /s)	1019.78(63.47)	965.91(63.00)	950.361(48.20)
High	TPR(dynes/cm ⁵ /s)	885.42(67.17)	865.39(66.67)	825.84(51.01)
Low	LF(Hz)	-	-	501.00(193.86)
High	LF(Hz)	-	-	1106.01(204.04)
Low	HF(Hz)	-	-	808.88(268.59)
High	HF(Hz)	-	-	1274.77(282.70)
Low	LF-HF(Hz)	-	-	1.26(0.47)
High	LF-HF (Hz)	-	-	1.87(0.51)

Table 19
*Marginal Means and Standard Errors for Baseline and Task Cardiovascular Measures
 and Cardiovascular Recovery by Distraction Group*

Distraction Group		Baseline(SE)	Task (SE)
Control	SBP(mmHg)	106.54(1.06)	124.12(1.50)
Distraction	SBP(mmHg)	106.00(1.06)	118.95(1.50)
Control	DBP(mmHg)	57.94(0.96)	74.76(1.34)
Distraction	DBP(mmHg)	55.77(0.96)	70.00(1.34)
Control	HR(bpm)	79.35(2.26)	93.00(2.59)
Distraction	HR(bpm)	76.67(2.01)	90.43(2.30)
Control	PEP(secs)	104.82(2.91)	102.68(2.87)
Distraction	PEP(secs)	105.62(2.62)	105.20(2.59)
Control	CI(L/min/m ²)	4.29(0.20)	4.11(0.22)
Distraction	CI(L/min/m ²)	4.21(0.19)	4.04(0.21)
Control	SI(ml/beat/m ²)	56.05(3.08)	45.82(2.91)
Distraction	SI(ml/beat/m ²)	56.25(2.85)	44.42(2.69)
Control	TPR(dynes/cm ⁵ /s)	912.63(47.38)	1221.88(83.80)
Distraction	TPR(dynes/cm ⁵ /s)	883.95(43.80)	1147.37(77.44)
Control	LF(Hz)	561.38(121.28)	1351.22(317.46)
Distraction	LF(Hz)	744.72(127.95)	1457.17(334.92)
Control	HF(Hz)	1008.33(325.95)	495.42(237.23)
Distraction	HF(Hz)	1545.33(343.88)	955.74(250.27)
Control	LF-HF(Hz)	1.38(0.25)	6.10(1.25)
Distraction	LF-HF (Hz)	0.74(0.26)	3.60(1.36)

Group		2-min(SE)	5-min(SE)	10-min(SE)
Control	SBP(mmHg)	110.11(1.22)	108.49(1.12)	107.61(1.04)
Distraction	SBP(mmHg)	108.05(1.22)	106.93(1.12)	106.89(1.04)
Control	DBP(mmHg)	58.27(1.18)	55.92(1.20)	54.45(1.29)
Distraction	DBP(mmHg)	54.24(1.18)	53.65(1.14)	51.29(1.29)
Control	HR(bpm)	80.81(2.44)	81.44(2.40)	81.33(2.12)
Distraction	HR(bpm)	80.29(2.17)	79.34(2.13)	80.16(1.88)
Control	PEP(secs)	102.11(3.30)	104.06(3.10)	104.97(3.12)
Distraction	PEP(secs)	104.29(2.98)	105.54(2.80)	106.82(2.82)
Control	CI(L/min/m ²)	4.36 (0.20)	4.31(0.21)	4.42(0.21)
Distraction	CI(L/min/m ²)	4.12((0.19)	4.19(0.20)	4.10(0.20)
Control	SI(ml/beat/m ²)	55.52(3.01)	54.09(3.26)	55.39(2.90)
Distraction	SI(ml/beat/m ²)	51.25(2.78)	53.14(3.01)	51.63(2.65)
Control	TPR(dynes/cm ⁵ /s)	932.89(65.61)	980.48(63.77)	890.58(51.69)
Distraction	TPR(dynes/cm ⁵ /s)	1013.98(60.63)	891.91(58.93)	925.20(47.77)
Control	LF(Hz)	-	-	882.37(196.96)
Distraction	LF(Hz)	-	-	678.66(207.80)
Control	HF(Hz)	-	-	955.37(255.66)
Distraction	HF(Hz)	-	-	1108.77(269.72)
Control	LF-HF(Hz)	-	-	2.13(0.44)
Distraction	LF-HF (Hz)	-	-	1.03(0.47)

Figure 1
Marginal Means and Standard Errors of SI changes from Baseline to Task through the Recovery Period by Low and High Trait Rumination Groups

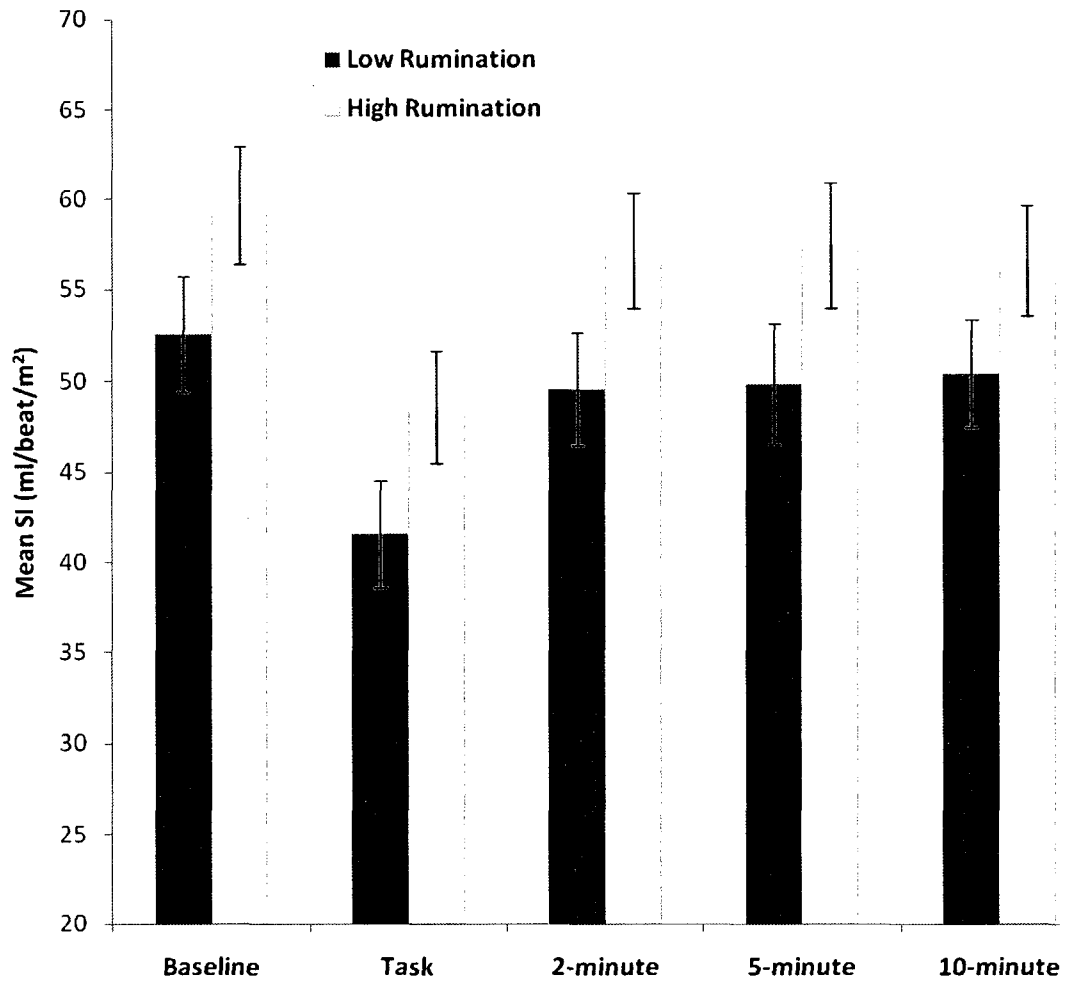


Figure 2
Marginal Means and Standard Errors of HR changes from Baseline to Task through the Recovery Period by Low and High Trait Rumination Groups

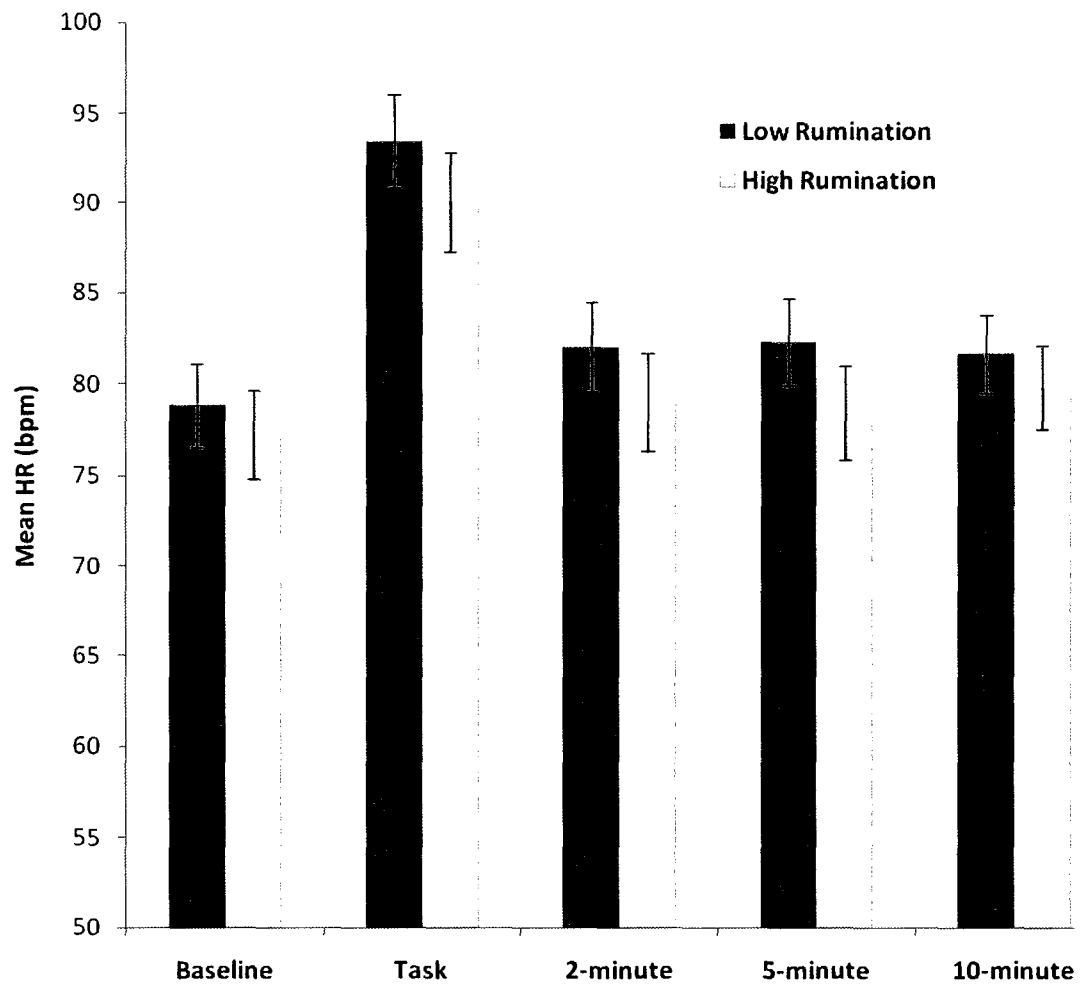


Figure 3
Marginal Means and Standard Errors of SBP changes from Baseline to Task through the Recovery Period by Distraction Group

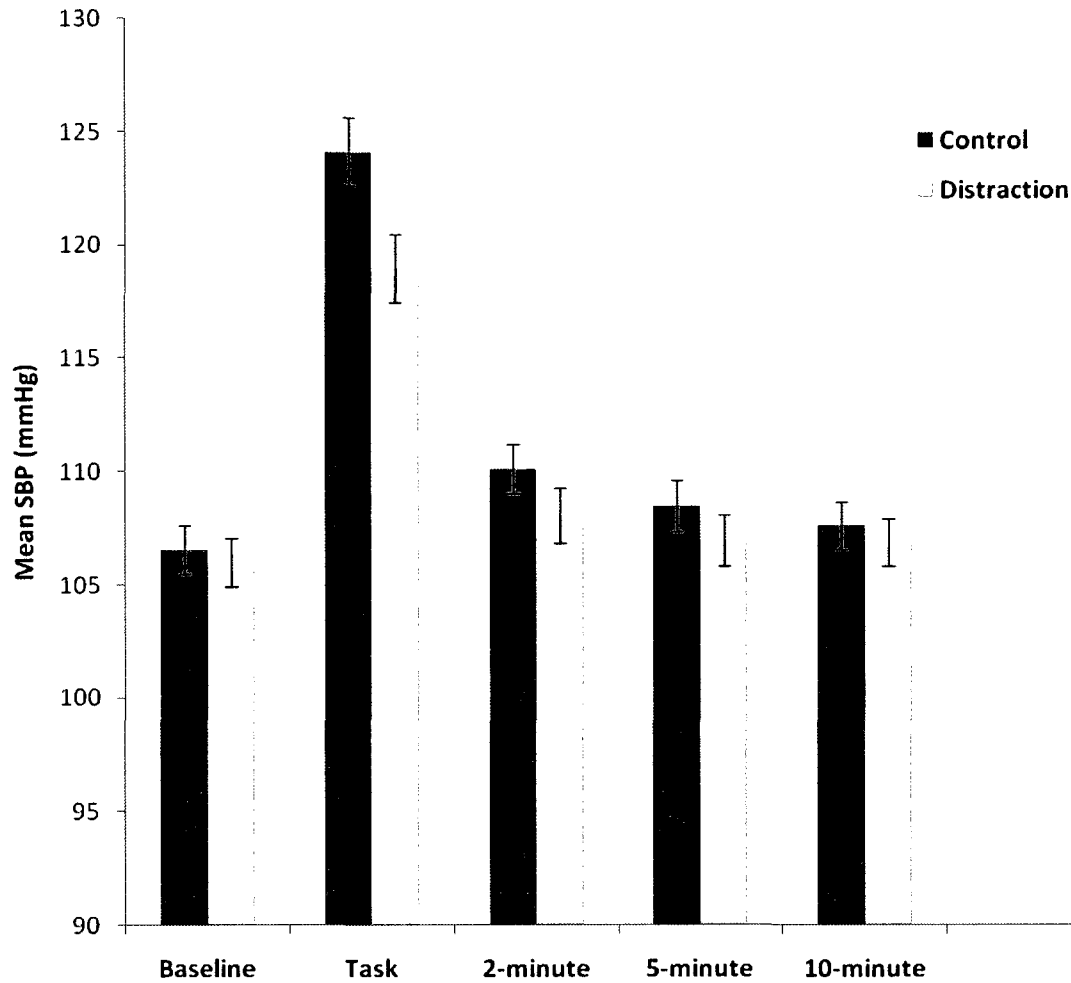


Figure 4
Marginal Means and Standard Errors of DBP changes from Baseline to Task through the Recovery Period by Distraction Group

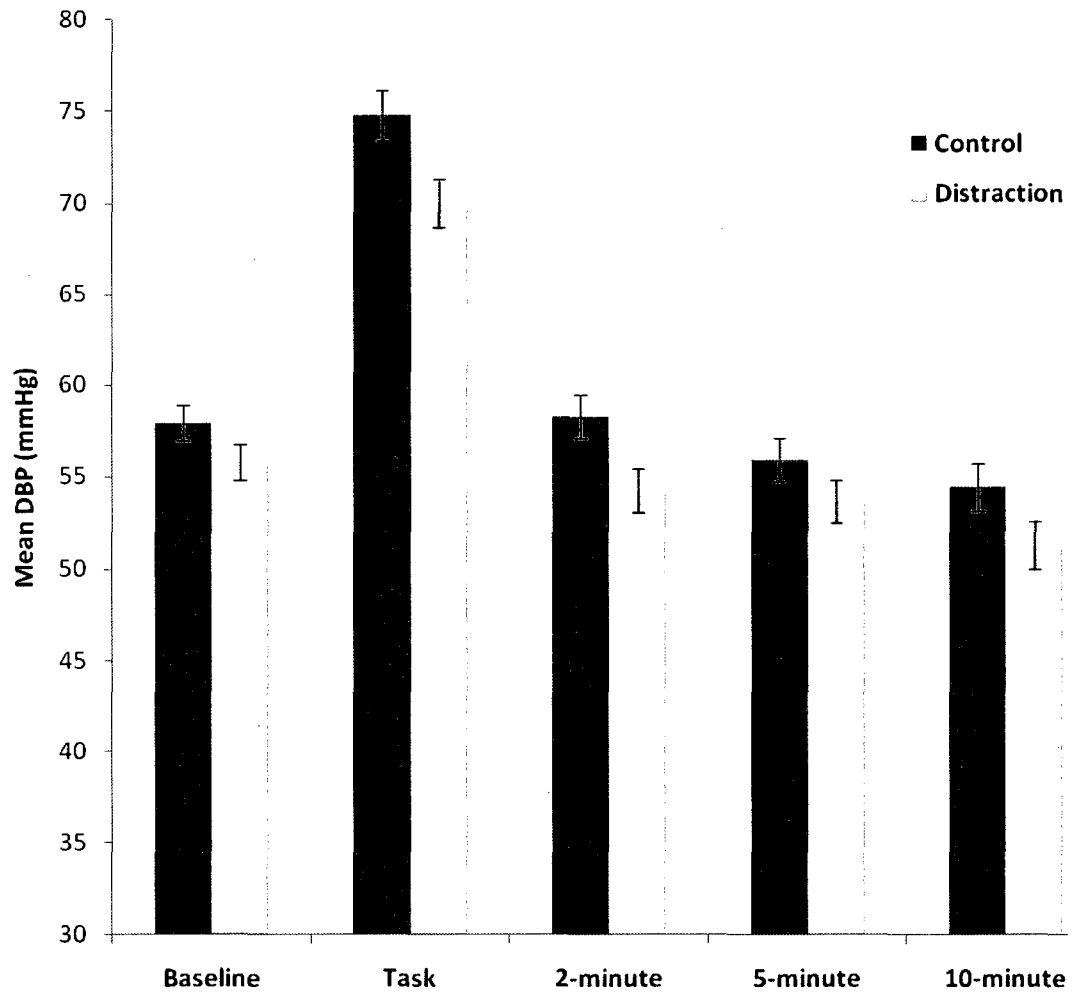


Figure 5
Marginal Means and Standard Errors of HR changes from Baseline to Task through the Recovery Period by Distraction Group

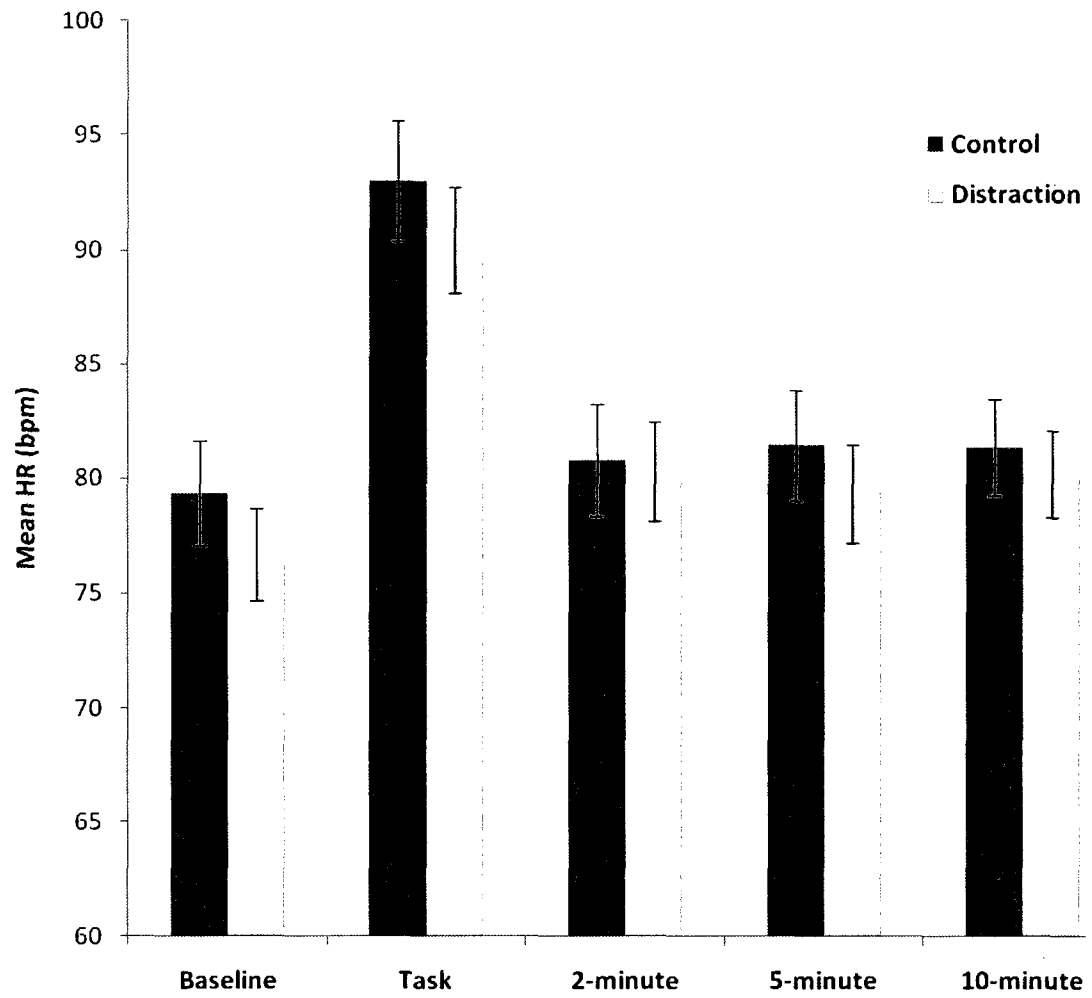


Figure 6
Marginal Means and Standard Errors of PEP changes from Baseline to Task through the Recovery Period by Distraction Group

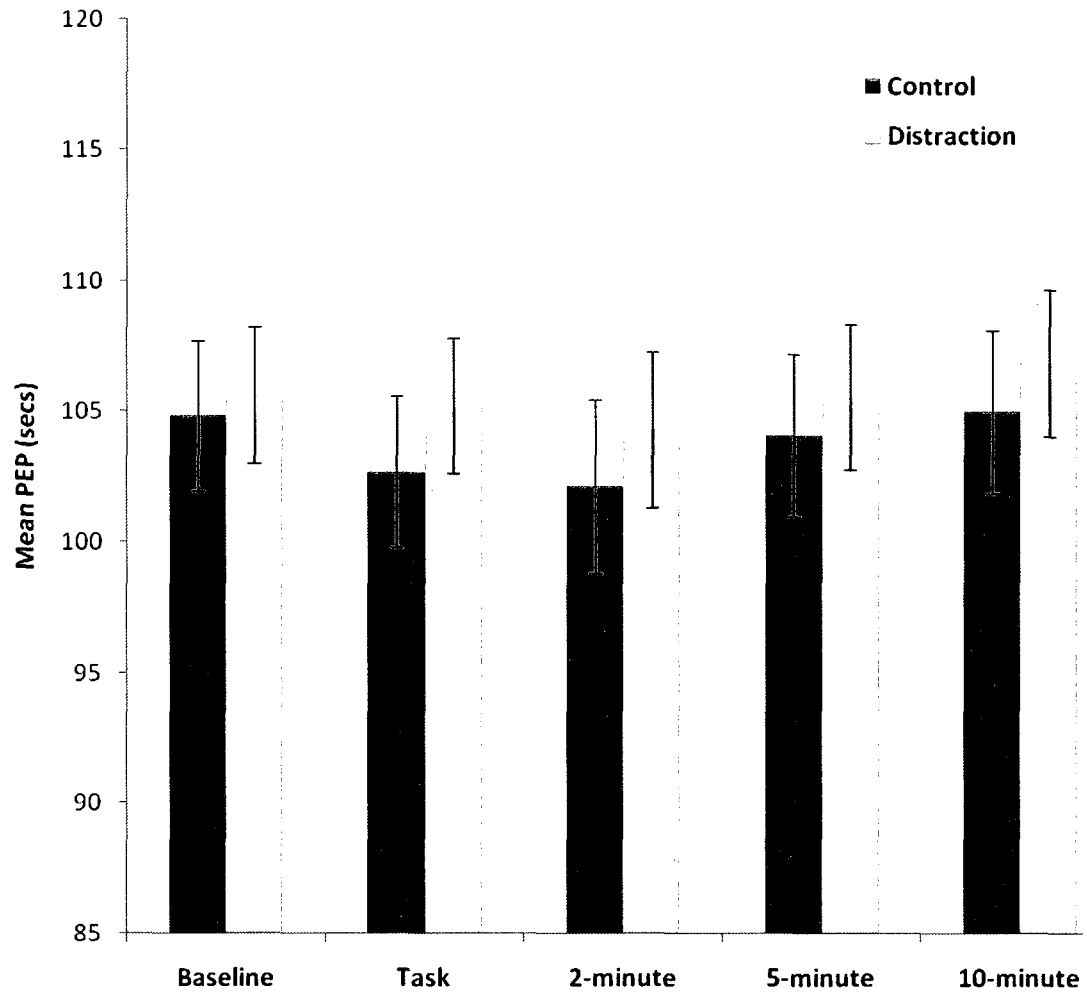


Figure 7
Marginal Means and Standard Errors of SI changes from Baseline to Task through the Recovery Period by Distraction Group

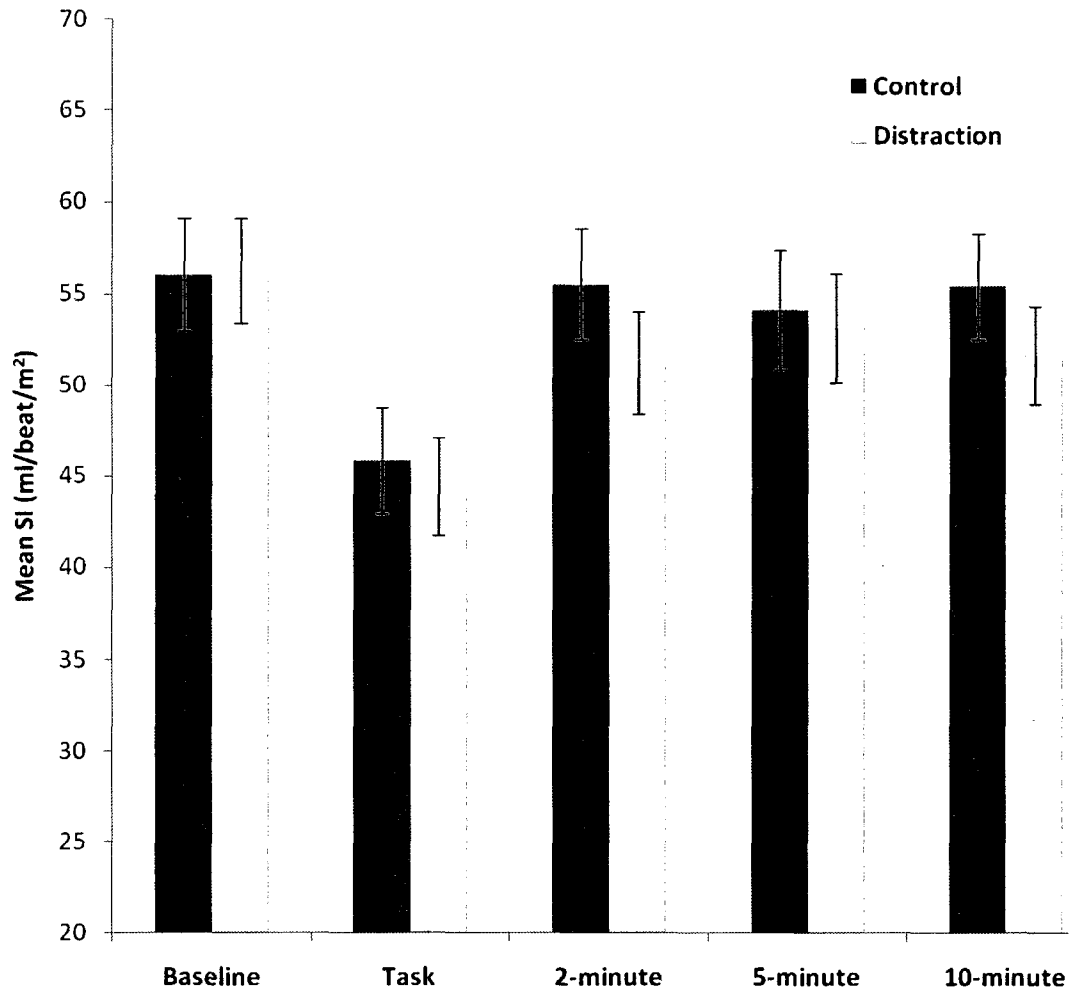


Figure 8
Marginal Means and Standard Errors of LF changes from Baseline to Task through the Recovery Period by Distraction Group

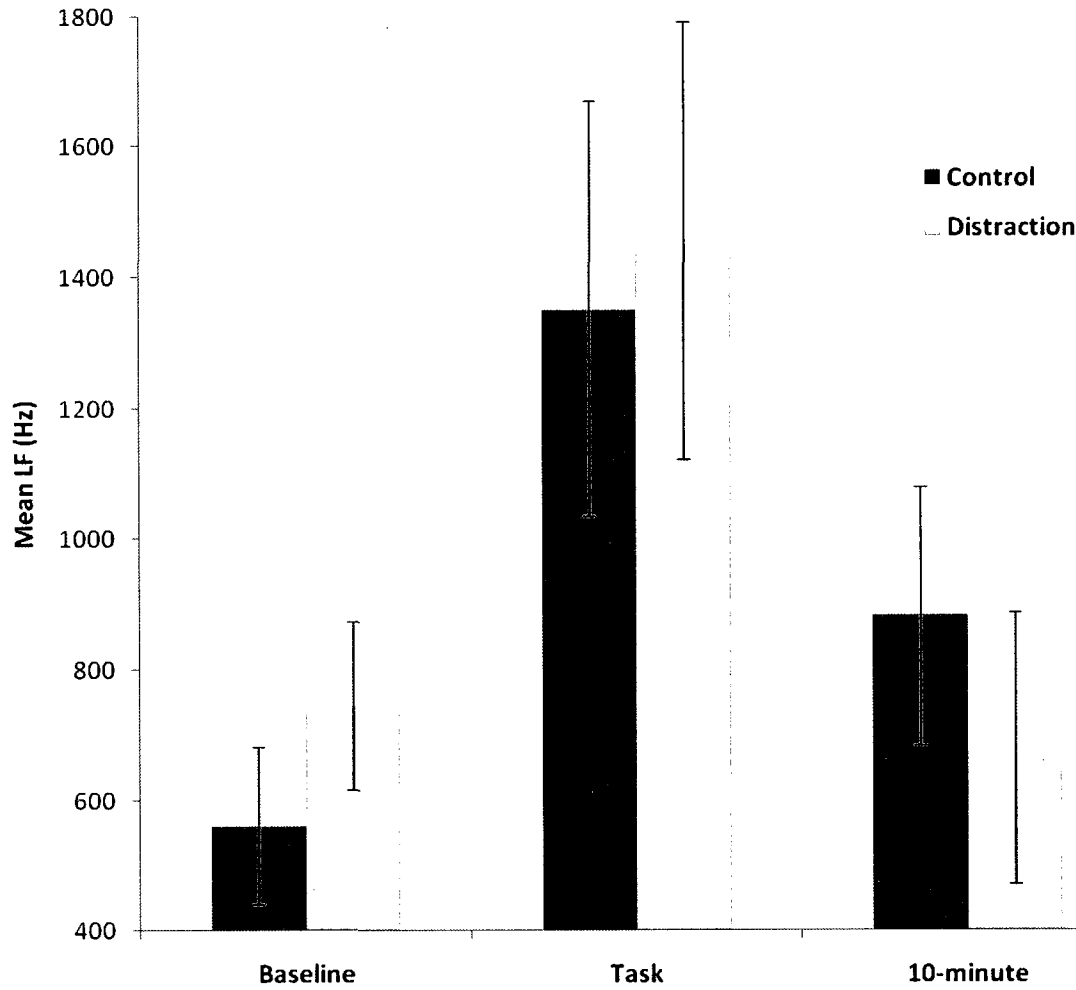


Figure 9
Estimated Marginal Means of SI Recovery Responses for the Trait Rumination and Distraction Interaction

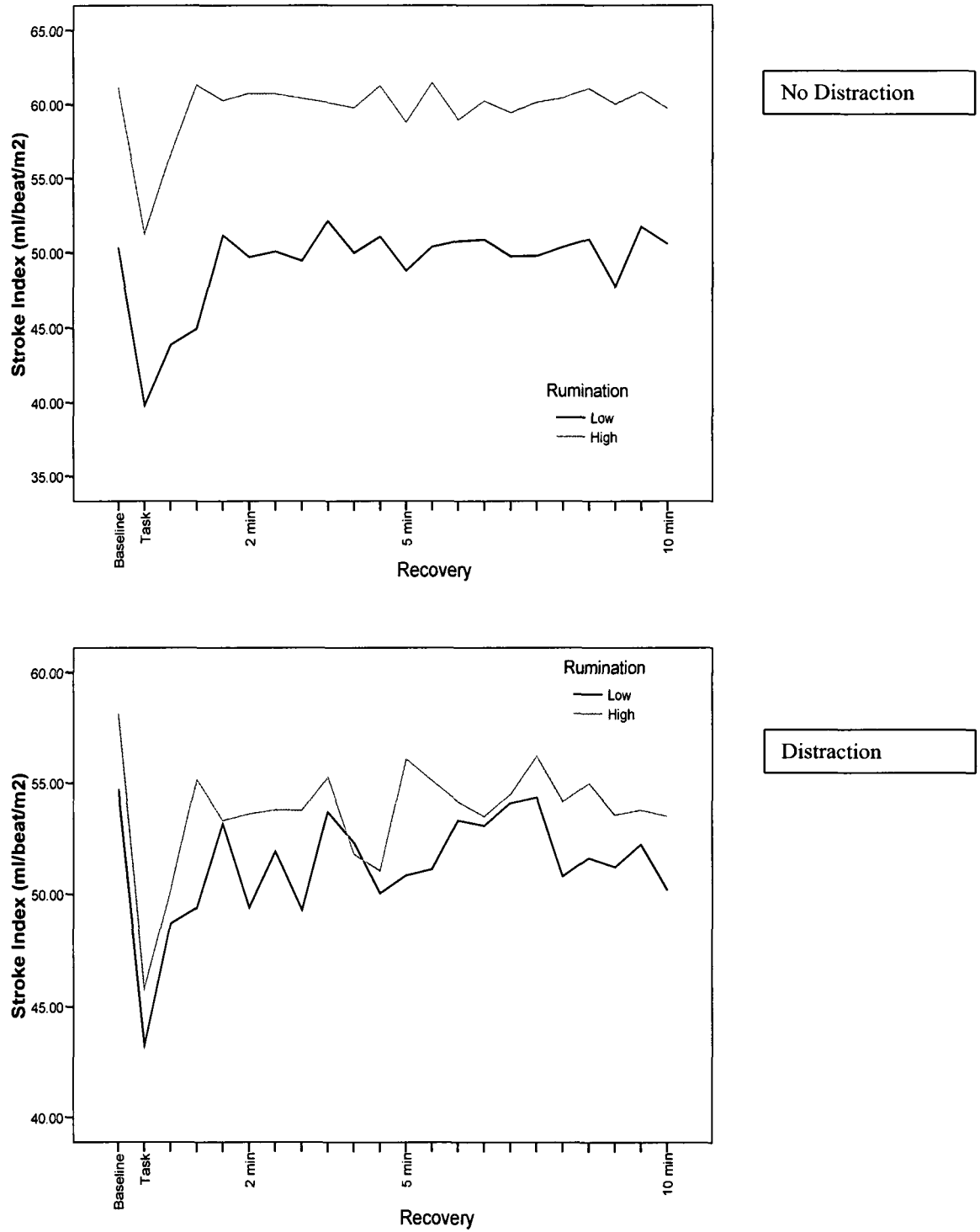
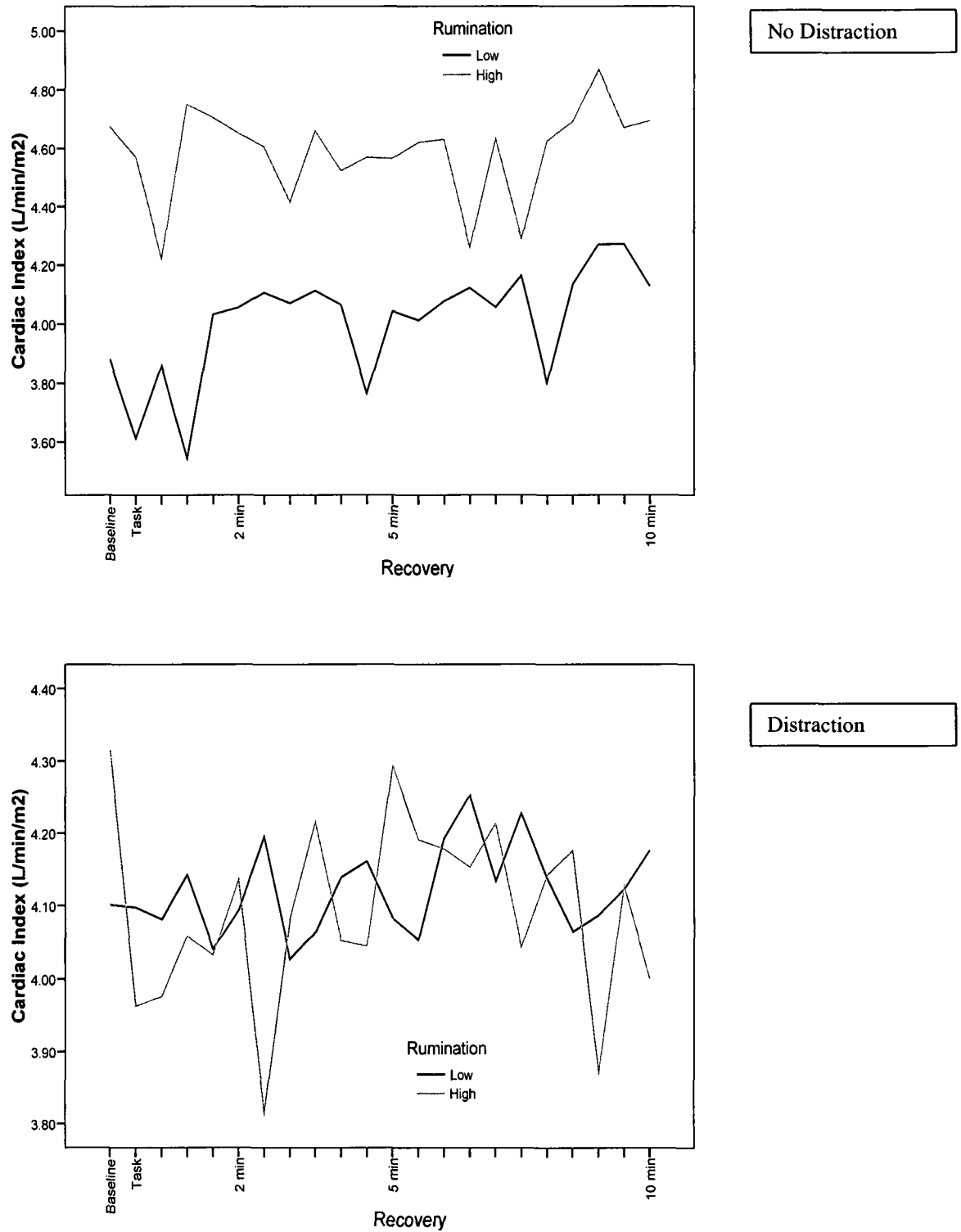


Figure 10
Estimated Marginal Means of CI Recovery Responses for the Trait Rumination and Distraction Interaction



DISCUSSION

The present study hypothesized that trait rumination would predict increased cardiovascular reactivity and prolonged cardiovascular recovery, independent of dispositional hostility, following an anger-recall task. It was also hypothesized that distraction would decrease or prevent rumination thus decreasing its deleterious effects on cardiovascular function. Finally, it was hypothesized that distraction would lead to more accelerated cardiovascular recovery in individuals with a greater tendency to engage in rumination as compared to those low in rumination.

Trait Rumination and Cardiovascular Reactivity

It was proposed that trait rumination would predict increased cardiovascular reactivity, independent of dispositional hostility, following the anger-recall task. The results indicated that trait rumination independently predicted increased reactivity of SI. These findings are consistent with previous research demonstrating that rumination about an emotionally arousing task was related to an increased cardiovascular response (Glynn, Christenfeld, & Gerin, 2002).

In the Glynn et al. (2002) study, rumination was associated with an increased blood pressure response. However, the current study found an increased cardiovascular response only in regards to SI. This may be due to the differences in emotional tasks, with the Glynn et al.'s (2002) study utilizing a cognitively distressing task and the current study using a personally relevant anger-recall task. The elicitation of different emotions may have different effects on cardiovascular responses (Feldman, Cohen, Lepore, Matthews, Kamarck, & Marsland, 1999; Gerin et al., 1999).

The different results may also highlight the importance of examining cardiovascular recovery in conjunction with cardiovascular reactivity, as there tends to be inconsistencies in the literature regarding negative emotions and cardiovascular reactivity (Glass, Lake, Contrada, Kehoe & Erlanger, 1983; Kamarck, Manuck & Jennings, 1990; Smith, 1992). Though methodological differences may often play a role in differential results, some of the discrepancy may be due to a limited or an inconsistent relation between negative affect and cardiovascular reactivity.

Trait Rumination and Cardiovascular Recovery

Results of the present study indicate that trait rumination is predictive of prolonged HR and SI responses, above and beyond dispositional hostility, following an anger-recall task. This delayed recovery of cardiovascular function may reflect extended beta-adrenergic activation and/or parasympathetic withdrawal. The present study also found that during the recovery period trait rumination was associated with increased levels of state rumination and anger.

Gerin and colleagues (2006) proposed a model that describes how rumination may lead to prolonged cardiovascular recovery. According to these researchers, the cognitive component of rumination instigates negative affect (i.e. anger, anxiety, and/or sadness) and that these emotions lead to increased autonomic activity (e.g. increased heart rate). Gerin et al. (2006) further postulate that these processes are reciprocal, such that elevations in autonomic arousal may promote negative emotions and in turn negative affect may lead to the maintenance of increased cardiovascular responses. Similarly, negative emotions may increase ruminative cognitions which may, in turn, promote negative emotions.

The finding that trait rumination is related to prolonged cardiovascular recovery is relatively consistent with prior research. One study found that older adults who ruminated about an aversive event had delayed blood pressure recovery (Melamed, 1996). Further, Glynn, Christenfeld, and Gerin (2002) found that young adults who spent more time thinking about a stressful math task that was coupled with harassment were more prone to prolonged recovery of blood pressure. In contrast to these findings, the current study failed to find a significant relation between blood pressure and trait rumination. This may be due to variations in the measurement of cardiovascular responses. For example, Glynn et al. (2002) measured blood pressure continuously throughout the recovery period. Alternatively, the current study measured blood pressure in 60-second intervals, which may have overlooked subtle changes in blood pressure recovery. Variations in results may also be partially explained by the different tasks that were used in these studies. Glynn and colleagues (2002) used a cognitively distressing task, which is systematically different than explicit and personalized anger expression which was used in the present study. Several researchers have posited that these differences in emotional reactions may have differential effects cardiovascular responses (Feldman, Cohen, Lepore, Matthews, Kamarck, & Marsland, 1999; Gerin et al., 1999).

Gerin, Davidson, Christenfeld, Goyal, and Schwartz (2006) also found that trait rumination was associated with prolonged cardiovascular recovery of blood pressure using the same anger-recall task that was used in the current study. These differential results may be due to several other methodological differences between the current study and Gerin et al.'s (2006) study. Gerin and colleagues (2006) examined both males and females while the current study only examined women. Further, the current study

measured rumination retrospectively after the end of the recovery period. Gerin et al. (2006), on the other hand, used a “thought sampling” technique, in which ruminative thoughts were measured throughout the recovery period. Finally, the current study’s anger-recall task interval and recovery period were briefer in duration than the Gerin et al. (2006) study, which may have potentially been too short of a timeframe to capture differences in blood pressure recovery. Future research should take these factors into account when attempting to replicate these findings.

Distraction and Cardiovascular Recovery

As expected, distraction independently predicted accelerated cardiovascular recovery of several cardiovascular dimensions, following the anger-recall task. Specifically, distraction predicted faster post-task recovery of SBP, DBP, HR, PEP, SI, and LF. These accelerated recovery responses may be due to decreased beta-adrenergic activation and/or increased vagal tone. The distraction manipulation utilized appears to have been successful in decreasing levels of state rumination and state anger. That is, those individuals in the distraction group indicated decreased levels of state rumination and state anger during the post-task recovery period compared to those individuals in the control condition.

The finding that distraction predicted accelerated cardiovascular recovery is consistent with previous research (Neumann et al., 2004; Gerin et al., 2006). Gerin et al. (2006) found that participants in a distraction condition reported decreased levels of anger and had faster recovery of blood pressure and heart rate following an anger-recall task than those participants in a control condition. Findings of the present study were generally consistent with Gerin et al.’s (2006) study despite the fact that the distraction

manipulations used were substantially different. In contrast to the current study, which provided instruction to the participants to read a neutral article, Gerin and colleagues (2006) placed multiple distracters in the room (e.g. toys and magazines) that participants could engage themselves in if they decided to do so. This may be a good model for future research which may find an even greater effect of distraction when the manipulation is personally-relevant.

Several other researchers have demonstrated an accelerated cardiovascular recovery response to distraction. Relaxation/meditation has been shown to hasten recovery of blood pressure following the cold pressor task and exercise (Patel, 1975). In addition, other researchers have found that photographs were effective in accelerating recovery of blood pressure after an anger-recall task (Schwartz, Gerin, Davidson, & Christenfeld, 2000). The current study expanded previous research by demonstrating similar hemodynamic patterns across a broader array of cardiovascular measures.

The Interaction of Trait Rumination and Distraction

Consistent with this study's hypothesis, the interaction of trait rumination and distraction was predictive of accelerated cardiovascular recovery. Specifically, those with high trait rumination experienced greater benefit from the distraction manipulation than low ruminators with regard to accelerated cardiovascular recovery of SI and CI. This finding is consistent with previous research which has examined the effects of the interaction of trait rumination and distraction on heart rate and blood pressure. Gerin et al. (2006) reported a significant interaction, such that high trait ruminators had the slowest cardiovascular recovery following an anger-recall task when they were not

exposed to the distracter. Again, the current study adds to this existing literature by examining multiple cardiovascular outcome measures.

Strengths, Limitations, and Future Directions

A discussion of strengths and limitations of the current study in relation to other research in this area may help guide future research. One advantage of the current study was the use of a personally relevant anger-recall task which increased the ecological validity of this study. The verbal expression of emotion is thought to improve the validity of an emotional task (Seigman et al., 1990). Furthermore, having participants recall personally-relevant situations is likely to be more effective in eliciting anger than exposing them to films or scripts that are generically used for all participants. Conversely, there is limited research on the anger-recall task employed in this study and thus the validity of using this technique in this type of research is not fully understood. Furthermore, social conflict and harassment have been shown to provoke greater levels of anger than the task used in this study, which may provide richer results (Davis et al., 2000). One may argue, however, that normal daily stressors rarely involve harassment, and thus it is important to investigate more common levels of anger-provocation and their effects on cardiovascular function. Furthermore, it should be noted that the anger task used in the current study resulted in significant cardiovascular responses for every cardiovascular measure except PEP.

A strength and limitation of the study is the inclusion of only young, healthy women as participants. Research examining the cardiovascular effects of psychological factors to date has grossly underrepresented women. Furthermore, since women have been found to ruminate more than men (Thayer, Newman, & McClain, 1994), there is a

need to understand its effect on women's cardiovascular function. In contrast, having a limited sample in terms of demographic variables limits the generalizability of the results of this study to other populations. For example, it has been shown that anger-focused rumination decreases with age (Phillips, Henry, Hosie, & Milne, 2006). Future replication of the current study should include men and women across different age ranges and health statuses to expand the generalizability and to examine whether these variables have a mediating or moderating effect on the relationships found in the current study.

The use of impedance cardiography to obtain a dynamic picture of cardiovascular reactivity and recovery strengthens the current study. The present study used archival data from research that was the first to collect impedance cardiography data to examine the relations among hostility, trait rumination, and cardiovascular responses. However, the sample size may have not been large enough to capture potentially significant effects for each of the cardiovascular measures used (see Tables 1, 2, 3, and 4).

The assessment of state rumination and anger throughout the task and recovery phases was an advantage of the present study. Previous research has not measured these constructs during the recovery period, and instead assumed that they persisted throughout this time (Haynes et al., 1991; Linden et al., 1997). Obtaining this data is important in order to verify the validity of the manipulation as well as provide support for the construct validity of the DRS scale used to measure trait rumination. However, the retrospective measurement of state anger and state rumination following the recovery period may be a weakness of the study. Future research may increase the reliability and

validity of these measures by obtaining them during the recovery period or directly after the task.

The state rumination measure used in the current study may have been somewhat problematic because it does not differentiate between maladaptive and adaptive rumination. Adaptive rumination is characterized by problem solving cognitions which may actually accelerate cardiovascular recovery. This is in contrast to maladaptive rumination which is characterized by more worrisome thoughts without moving the person toward a resolution. This failure to differentiate between maladaptive and adaptive rumination coping styles may have confounded the results of the present study and future research should use more accurate measures. Despite this, it is important to note the presence of a positive relation between state rumination and anger in the current study. This suggests that subjects in this study were using a greater level of maladaptive rumination coping.

The use of the Ho scale may also be a limitation of the current study. Cook and Medley (1954) used the Ho scale to predict scores on the Minnesota Teacher Attitude Inventory (MTAI), which has been shown to predict the level of rapport between teachers and pupils. The researchers found that the Ho scale was more predictive of MTAI scores in males than females. It has also been shown that high scores on the Ho scale are more related to cynicism than overt hostility in women (Han, Weed, Calhoun, & Butcher, 1995). Given that the current study consisted of an all female sample this may be somewhat problematic. However, other research has demonstrated that the scale measures several different facets of interpersonal hostility across genders (Barefoot et al.,

1989). Further, the psychometric strengths of the scale as well as its history of use in cardiovascular research may validate its use in this study.

Most of the previous research examining cardiovascular recovery has used change scores and repeated measures ANOVA to measure recovery (e.g. Glynn, Gerin, & Christenfeld, 1998; Schwartz et al., 2000). This method omits important information about the recovery process, whereas the excursion measures (i.e., area under the recovery curve minus prestress levels) used in the current study captures the full range of data and the speed and rate of recovery. On the other hand, the current study did not use continuous monitoring of blood pressure, which may enhance measurement sensitivity in future research.

The distraction condition used in the current study may also have some limitations. First, the laboratory in which the present study was conducted offered some opportunities for visual distraction (e.g. the presence of a picture, computer, table, and other laboratory supplies) (Schwartz et al., 2000). Some participants may have taken advantage of this natural opportunity to distract themselves which would decrease differences between the control and distraction groups. On the other hand, having opportunities for distraction provides a more ecologically valid comparison of the distraction and control groups. Second, the distracter used (i.e. reading a neutral article) may be relatively less potent than other potential distracters. A more engaging distracter, such as one that produces positive affect or one that is tailored to personal interests, may be more effective in distracting participants from ruminative thoughts.

In addition, it has been postulated that distraction is similar to repression and may represent a maladaptive coping style (Linehan, 1993). Distraction may also require a

substantial amount of effort in order to keep negative emotions from conscious awareness, which might actually result in an increase of negative thoughts (Wegner, 1994). Thus, researchers have proposed that distraction may only provide a short-term relief from ruminative thought processes. Alternatively, mindfulness training and meditation may provide a more enduring benefit. Evidence for this is found in a study demonstrating that individuals in a meditation condition achieved greater decreases in dysphoric mood than those in a distraction condition (Broderick, 2005). Future research should consider other forms of coping styles and/or mood regulation strategies in order to identify more clinically effective methods both in terms of psychological and physiological benefits.

Summary and Clinical Implications

The current study found that trait rumination was predictive of increased cardiovascular reactivity and prolonged cardiovascular recovery following an anger-recall task, independent of dispositional hostility. Because prolonged cardiovascular recovery has been associated with cardiovascular disease (e.g., Mezzacappa et al., 2001; Brosschot & Thayer, 1998; Hocking-Schuler & O'Brien, 1997; Earle, Linden & Weinberg, 1999) the results of this study indicate that trait rumination, independent of hostility, may place individuals at a greater risk to develop cardiovascular disease.

Results of the present study revealed that distraction decreased state anger and state rumination and accelerated the cardiovascular recovery of several dimensions. It was also found that high trait ruminators experienced an even greater benefit from the distraction manipulation in terms of hastened cardiovascular recovery. These results suggest that distraction may be used as a clinical intervention to reduce rumination for the

psychological as well as the physiological benefits. Furthermore, results suggest that clinicians may be wise to screen their clients for the tendency to ruminate so that a distraction intervention can be used in session and/or taught for enduring benefits.

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APPENDIX A

The Dissipation-Rumination Scale (DRS)

The Dissipation-Rumination Scale (DRS)

Using the following Scale, indicate the response which reflects your *first* reaction to each statement by placing a "x" in the box under the response scale before each item. Please do not leave out any item and be spontaneous and accurate as much as possible within the limits of choices offered below.

	Completely True for Me	Fairly True for Me	True to a Certain Extent	Fairly False for Me	Completely False for Me	
1	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	I never help those who do me wrong.
2	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	I will always remember the injustices I have suffered.
3	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	The more time that passes, the more satisfaction I get from revenge.
4	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	It is easy for me to establish good relationships with people.
5	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	It takes many years for me to get rid of a grudge.
6	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	When somebody offends me, sooner or later I retaliate.
7	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	I do not forgive easily once I am offended.
8	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	I often bite my fingernails.
9	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	I won't accept excuses for certain offenses.
10	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	I hold a grudge, for a very long time, towards people who have offended me.
11	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	I remain aloof towards people who annoy me, in spite of any excuses.
12	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	I can remember very well the last time I was insulted.
13	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	I am not upset by criticism.
14	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	I enjoy people who like jokes.
15	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	I still remember the offenses I have suffered, even after many years.
16	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	If somebody harms me, I am not at peace until I can retaliate.
17	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	When I am outraged, the more I think about it, the angrier I feel.
18	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	I like people who are free.
19	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	I am often sulky.
20	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	Sometimes I can't sleep because of a wrong done to me.

APPENDIX B

Revised Impact of Events Scale (IES)

Revised IES

ID#: _____

Date: _____

Please circle a number for each item, indicating how frequently these comments were true for you during the recovery period. If they did not occur during that time, please circle the "not at all" column.

	Not at all	Rarely	Sometimes	Often
1. I thought about it when I didn't mean to.....0		1	2	3
2. I avoided letting myself get upset when I thought About it or was reminded of it..... 0		1	2	3
3. I tried to remove it from memory..... 0		1	2	3
4. I had trouble relaxing or closing my eyes because pictures or thoughts about it came into my mind... 0		1	2	3
5. I had waves of strong feelings about it..... 0		1	2	3
6. I had flashbacks about it..... 0		1	2	3
7. I stayed away from reminders of it..... 0		1	2	3
8. I felt as if it hadn't happened or it wasn't real..... 0		1	2	3
9. Pictures about it popped into my mind..... 0		1	2	3
10. Other things kept making me think about it..... 0		1	2	3
11. I was aware that I still had a lot of feelings about it, but I didn't deal with them..... 0		1	2	3
12. I tried not to think about it..... 0		1	2	3
13. Any reminder brought back feelings about it..... 0		1	2	3
14. My feelings about it were kind of numb.....0		1	2	3

APPENDIX C

Instructions for the Anger Recall Task

For the angry recall task, the participant was given the following instructions:

The purpose of this part of the study is to check your physiology when you feel angry, irritated, or upset. The way we have found it best for most people to do this is to identify a recent incident, an incident that has occurred in the past year, in which you got really angry, frustrated, or irritated. In fact, one that when you think about it, still makes you angry. It may be, for example, an unpleasant encounter with a co-worker, or an argument you had with someone in your family or a close friend. Choose any recent situation where you were really irritated or upset with another person. Don't be embarrassed about how you felt or what happened, because the more realistic your feelings are, the more we will learn about you physiology. Sometimes, to relive a situation, it is good to take a moment to remember where you were, who you were with, what was said and done, and to close your eyes and almost see the situation.

The participant was then asked the following questions to ensure that the incident is appropriate for the study and that it has a clear beginning and end.

Can you think of a situation like that? Who did you get angry with?

When did this incident occur? Where did this incident occur?

Tell me in one sentence what led up to the incident.

Tell me in one sentence about the events that made up the incident.

For the next three minutes, I would like you to recreate the incident as best you can. Starting with the (beginning of the event as reported by the participant) and going to the (end of the event as reported by the participant). Tell me what you said and did, how the other person responded, what you were thinking and feeling, and what happened after that. (If the participant does not seem to be following these instructions, then ask how he/she felt at several points during the event). After the next thirty seconds, I will ask you to speak out loud and say whatever you want to say about this incident. Prepare now, and speak when I tell you to begin.

APPENDIX D

Reading Manipulation Check

Directions: Please respond to the following questions concerning the article you read by circling the appropriate answer.

1. **During the prior rest period**, how much of the time did you spend thinking about the event you described earlier?

4	3	2	1
all of the time	most of the time	some of the time	none of the time

2. **During the prior rest period**, how much of the time did you spend reading the article?

4	3	2	1
all of the time	most of the time	some of the time	none of the time

3. Was the article about
- a. the movie E.T.
 - b. the planet Jupiter
 - c. scientists looking for life forms in our universe

4. From the information provided in the article, have astronomers found a planet that is capable of supporting life?

Yes No

5. Did you have trouble concentrating on the article?

Yes No

if yes, Why? _____

Vita

Meghan McLain (Rautiola), daughter of Michael McLain and Jennifer Swihart, was born in Jackson, Michigan. By the time she graduated from The Da Vinci Institute high school in 2000, she had concurrently completed an associate's degree. After several years of studying abroad she obtained a B.S. in Neuroscience and a B.A. in Psychology at the University of Otago, Dunedin, New Zealand. Norfolk State University awarded her a M.A. in Clinical and Community Psychology in 2009. Meghan expects to receive her PsyD from the Virginia Consortium Program in Clinical Psychology in 2011.