

UNIVERSITY OF CALGARY

The Influence of Rumination on Cardiovascular Recovery from Stress

by

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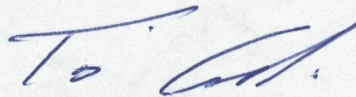
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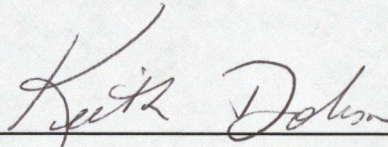
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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies for acceptance, a thesis entitled "The Influence of Rumination on Cardiovascular Recovery from Stress" submitted by Brenda L. Key in partial fulfillment of the requirements for the degree of Master of Science.



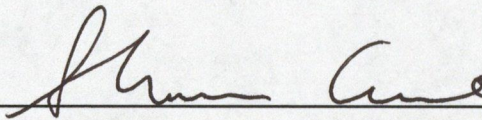
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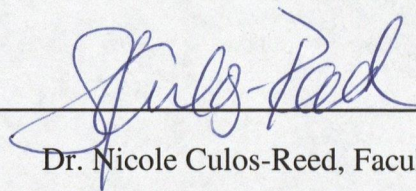
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## **Abstract**

To assess the influence of trait and state rumination on cardiovascular recovery following a stressor, cardiovascular data was collected from 64 undergraduate women during a 10-minute baseline period, 5-minute public speaking task and a 15-minute recovery period followed by 24-hour ambulatory blood pressure monitoring. Trait rumination was assessed using the Stress Reactive Rumination Scale and state rumination was assessed using thought reports. Results indicated that low trait ruminators who were ruminating 10 minutes after the termination of the stressor had inferior diastolic blood pressure and high-frequency heart rate variability recovery compared to low trait ruminators who were not ruminating. State rumination was not associated with cardiovascular recovery in high trait ruminators. Ambulatory blood pressure monitoring revealed that high trait ruminators had less nighttime diastolic blood pressure dipping. Rumination may play a role in the association between stress and hypertension by prolonging cardiovascular recovery and extending physiological arousal into nighttime sleep.

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## **List of Abbreviations**

|               |                                       |
|---------------|---------------------------------------|
| <b>ABPM</b>   | ambulatory blood pressure monitoring  |
| <b>ANCOVA</b> | analysis of covariance                |
| <b>ANOVA</b>  | analysis of variance                  |
| <b>AUC</b>    | area under the curve                  |
| <b>BP</b>     | blood pressure                        |
| <b>CO</b>     | cardiac output                        |
| <b>DBP</b>    | diastolic blood pressure              |
| <b>ECG</b>    | Electrocardiogram                     |
| <b>HF-HRV</b> | high frequency heart rate variability |
| <b>HR</b>     | heart rate                            |
| <b>SBP</b>    | systolic blood pressure               |
| <b>SRRS</b>   | stress reactive rumination scale      |
| <b>SV</b>     | stroke volume                         |
| <b>TPR</b>    | total peripheral resistance           |
| <b>VAS</b>    | visual analogue scale                 |

## **The Influence of Rumination on Cardiovascular Recovery from Stress**

Psychological and physiological responses to stress have been implicated in the development of hypertension (Obrist, 1981; Manuck, Kasprowicz, & Muldoon, 1990; Light, 1981). Rumination may play a role in this association by prolonging psychological and physiological arousal that accompanies stress. The purpose of the present study is to evaluate the association between rumination and cardiovascular recovery from a negative emotional stressor.

*Cardiovascular reactivity* is generally defined as the change that occurs in the heart and the blood vessels in response to a stressor (Obrist, Light, James, & Strogatz, 1987). Physiological changes that typically occur in stressful situations, such as increases in heart rate (HR) and blood pressure (BP), are part of a natural and adaptive response mechanism. In some cases this physiological response may be exaggerated relative to the demands of the stressor. According to the cardiovascular reactivity hypothesis when individuals respond to stressful stimuli in an exaggerated manner they set in motion a cascade of pathophysiologic events that may lead to tissue damage, system dysregulation and finally disease (Obrist et al., 1987; Lovallo & Gerin, 2003). Consistent with the reactivity hypothesis, exaggerated cardiovascular reactivity has been implicated in the development of hypertension (Manuck et al., 1990; Carroll, Ring, Hunt, Ford, & Macintyre, 2003; Treiber et al., 2003), however the strength of the prospective associations is typically modest (Carroll et al., 2001). For example, results from a 10-year prospective study that evaluated BP reactivity and future BP status indicated SBP reactivity accounted for 4.5% of the variance in future blood pressure status (Carroll et al., 2001).

A potential flaw in the cardiovascular reactivity hypothesis is that it focuses on the acute responses that occur during the presentation of laboratory stress tasks while ignoring the subsequent recovery period. *Cardiovascular recovery* is generally defined as the time period required for

physiological functioning to return to baseline levels after encountering a stressor (Linden, Earle, Gerin, & Christenfeld, 1997). If acute elevations in physiological activation are damaging to the cardiovascular system then variables which serve to prolong the stress related elevation (i.e. variables influencing cardiovascular recovery) may be damaging as well. Investigators have hypothesized that the total duration of BP elevation may be of greater importance in the development of hypertension relative to peak reactions to a stressor (Linden et al., 1997; Schwartz et al., 2003). For example, Borghi and colleagues (1986) found DBP recovery from a cognitive challenge was a more useful predictor of future hypertension than cardiovascular reactivity in borderline hypertensive subjects at 5 year follow-up. Poor cardiovascular recovery may be a marker of chronic sympathetic activation which is a possible pathway to the development of hypertension (Julius, 1993; Amerena & Julius, 1995; Brosschot & Thayer, 1998).

### Rumination & Cardiovascular Responses

Rumination is one personality factor that has recently been hypothesized to influence cardiovascular recovery (Brosschot & Thayer, 2003; Glynn, Christenfeld, & Gerin, 2002; Gerin, Davidson, Christenfeld, Goyal, & Schwartz, 2006). Rumination has been defined in many different ways (Siegle, Moore, & Thase, 2004; Nolen-Hoeksema, 1998), however for the purposes of this study *rumination* will be defined as repetitive thoughts focusing on problematic situations or events as well as the emotions and symptoms these evoke and the possible consequences (Thomsen, Mehlsen, Christensen, & Zachariae, 2003). Several researchers have concluded that the tendency to ruminate is a relatively stable trait and that differences in the tendency to ruminate exist (Just & Alloy, 1997; Knowles, Tai, Christensen, & Bentall, 2005; Nolen-Hoeksema & Morrow, 1993). The *rumination arousal model* described by Gerin and colleagues (Gerin et al., 2006) has been proposed to explain the potential mechanisms through which rumination may influence cardiovascular recovery. This model hypothesizes that engaging in rumination following a stressor sets in motion an

intertwined set of processes: the cognition leads to negative emotions (i.e. anger, sadness or anxiety) and the emotion produces elevated autonomic activation (e.g. causing BP elevation). The processes involved in this model may be synergistic: increased autonomic arousal may prolong negative emotions (and vice-versa) and the prolonged negative emotions may promote ruminative thoughts (and vice-versa).

Findings regarding reactivity and recovery from emotional stressors lend support to the hypothesis that rumination plays a role in prolonging physiological reactions. Positive and negative emotions result in approximately equal physiological arousal or reactivity (Jacob et al., 1999). Based on the reactivity hypothesis, it would be expected that both positive and negative emotions would be equally related to sustained elevations in BP, however only negative emotions are associated with hypertension (Brosschot & Thayer, 2003). A possible explanation for this finding is that recovery tends to be longer following negative emotional stressors (Brosschot & Thayer, 2003). By delaying cardiovascular recovery following negative emotions rumination may help to explain the association between negative emotions and harmful cardiovascular outcomes such as hypertension (Brosschot & Thayer, 2003; Glynn et al., 2002).

#### *Rumination and Cardiovascular Recovery*

Findings of recent studies support the hypothesis that rumination may be associated with prolonged BP recovery. Investigators have examined the effects of rumination versus distraction and emotional versus unemotional stressors on cardiovascular recovery. Distraction is thought to prevent rumination (Nolen-Hoeksema & Morrow, 1993) and therefore may serve as a useful tool to compare responses between participants who are allowed to ruminate versus those who are distracted and thus thought to be prevented from ruminating. In a study examining cardiovascular recovery following acute stressors in the laboratory, recovery was relatively faster for tasks that did not arouse emotion

compared to emotional tasks (Glynn et al., 2002). The magnitude of cardiovascular reactivity was unrelated to the emotionality. Furthermore, participants who were exposed to a distraction, and presumably could not ruminate during the recovery period, showed superior BP recovery compared to those without the distraction. A similar study evaluated the influence of a distraction versus no distraction condition on cardiovascular recovery following an anger recall task. Trait rumination was assessed using a self-report measure and a thought sampling technique was used during the recovery period to assess state rumination. High trait ruminators in the no distraction condition had the poorest BP recovery (Gerin et al., 2006). Thus, results suggest that rumination is associated with prolonged BP recovery while distraction is associated with hastened BP recovery.

Ambulatory monitors have been used in order to evaluate the impact of rumination on cardiovascular responses to emotional stressors in a more naturalistic setting. Brosschot and Thayer (2003) used ambulatory HR monitors to evaluate physiological arousal associated with positive versus negative emotional events and reported that HR recovery was prolonged after negative emotions relative to positive. Further, an investigation utilizing ambulatory BP monitoring (ABPM) revealed that high trait rumination was associated with higher mean ambulatory BP values even when controlling for established hypertension risk factors, such as parental history of hypertension and gender (Oseitutu et al., 2001).

In summary, rumination may be a mechanism through which negative emotional stressors lead to delayed recovery and preliminary findings suggest that trait rumination is associated with elevated ambulatory BP. The central goal of the current study is to evaluate the influence of rumination on cardiovascular recovery from negative emotional stress. Based on the previous literature examining the link between rumination, negative emotions and cardiovascular recovery the following hypotheses were proposed:

1. High trait ruminators would have inferior cardiovascular recovery relative to low trait ruminators.
2. High trait ruminators who are ruminating (state rumination) would have inferior cardiovascular recovery relative to high trait ruminators who were not ruminating and low trait ruminators regardless of whether or not they were ruminating.
3. High trait ruminators would have higher mean 24-hour ambulatory BP relative to low trait ruminators.

## Method

### *Participants*

A screening sample of 287 undergraduate students was used to identify 64 female participants (39 low and 25 high trait ruminators) between the ages of 18-45 who met the inclusion criteria of scoring either above 530 (high trait ruminator) or below 380 (low trait ruminator) on the Stress Reaction Rumination Scale (SRRS; Robinson & Alloy, 2003). The cut points for the SRRS were determined based on a tercile split of pilot data collected from a sample of 104 undergraduate students. Exclusion criteria included overt cardiovascular disease, including arrhythmia or history of cerebrovascular disease, current use of mood-altering medication (which may influence self-reports of rumination), and current use of anti-hypertensive medications, or other medication known to substantially affect BP.

### *Procedures*

#### *Screening Session*

Participants were asked to complete a questionnaire package that included a demographic questionnaire, general health questionnaire and the Stress Reactive Rumination Scale (SRRS).

### *Laboratory Stress Assessment.*

Following instrumentation with cardiovascular recording equipment, participants sat quietly for 10 minutes during which baseline measures were recorded. Following the baseline period participants engaged in the public speaking task described below.

*Public speaking task:* Participants were asked to speak about a recent stressful negative life event that they found difficult to stop thinking about. They were given 2 minutes to prepare their 5-minute speech. This task induces anxiety and also brings to mind an event that the participant has ruminated about in the past increasing the likelihood that the participant will ruminate during the subsequent recovery period. Additionally, this task has the capability to induce anxious, sad or angry rumination depending on which event the participant chooses to speak about which will allow this study to evaluate the generalizability of previous findings examining angry rumination and cardiovascular recovery.

After the conclusion of the public speaking task participants were asked to complete a scale that characterized the nature of the negative emotional stressor. In order to determine whether the participant was ruminating during the recovery period a thought report technique was utilized 5 minutes and 10 minutes after the termination of the public speaking task (assessment of state rumination). Following the recovery period participants were asked to elaborate on their thought reports.

### *Ambulatory Assessment*

At the conclusion of the laboratory assessment participants were instrumented for 24-hour ambulatory BP monitoring (ABPM).

## Measures

### *Psychosocial Measures*

*Stress Reactive Rumination Scale (SRRS).* The SRRS (Robinson & Alloy, 2003) is a 25-item scale that was adapted from the Response Styles Questionnaire-Ruminative Response Scale (RSQ-RRS; Nolen-Hoeksema & Morrow, 1991). The SRRS was developed by constructing items that were believed to assess the cognitive tendency to focus on the negative attributions and inferences that comprise the negative inferential style in response to major life stressors. The reliability (retest reliability  $r = .71, p < .001$ ) and internal consistency ( $\alpha = .89$ ) of this scale are considered adequate. The current study similarly found the SRRS to have adequate internal consistency,  $\alpha = .91$ . The SRRS was chosen as the measure to select high and low trait ruminators as this scale was designed to measure rumination in a college age sample and therefore contains instructions that may be particularly relevant to this population (see Appendix A). Additionally, the scale was designed to measure rumination in a manner that is not confounded with depressive symptoms which is a limitation of many other self-report rumination scales (Robinson & Alloy, 2003).

*Visual Analogue Scales (VAS).* Visual analogue scales were employed to measure the perceived magnitude and importance of the negative emotional event that the participant chose to speak about during the public speaking/rumination task in the laboratory (see Appendix B). Six VAS were utilized to measure how upsetting the event was at the time, how upsetting it was to talk about the event today, whether the event has been resolved and what emotions this event evoked (sadness, anxiety or anger). The six VAS were administered immediately following the public speaking task. Similar measures have been used for pain ratings and have been shown to be sensitive indices (e.g., Jensen, Karoly, & Brewer, 1986).

*State Rumination.* In order to assess state rumination a thought report technique was utilized during which the participant wrote down one or two words about what they were thinking about 5

minutes and 10 minutes after the termination of the public speaking task. Following the laboratory recovery period participants were asked to elaborate on what they wrote. Thought reports were coded by the experimenter as either ruminating about the speech or not ruminating about the speech. If it was unclear how to code the thought report from the participant's description they were directly asked whether they were ruminating about their speech.

*Sleep Quality.* Due to concerns about the effects of sleep disruption associated with ABPM on nighttime BP values (Ben-Dov, Ben-Arieh, Mekler, & Bursztyn, 2005) a four-item measure was used to assess sleep quality on a 5-point scale: "how difficult was it to fall asleep?", "how difficult was it to stay asleep?", "how would you rate your quality of sleep?", "how did your sleep compare to most nights?". Accurate sleep and wake times increase the validity of nighttime BP dipping measures (Gosse, Ansoborlo, Lemetayer, & Clementy, 1996); therefore, time of sleep onset and awakening were also collected.

#### *Laboratory Cardiovascular Measures*

Measurements of SBP and DBP (in mmHg) were obtained at 1 minute intervals using an automatic BP monitor (Accutor Plus, Data Scope Corp., Mont Vale, NJ, USA) and a BP cuff on the upper part of the non-dominant arm. Continuous measures of heart rate (HR; in beats per minute), stroke volume (SV; in ml/beat), total peripheral resistance (TPR; in  $\text{dyne} \cdot \text{sec}^{-\text{cm}^{-5}}$ ), and cardiac output (CO; in L/min) were recorded non-invasively using a HIC-2000 Bio-electric Impedance Cardiograph, Cardiac Output Program (C.O.P.) developed by Bio-Impedance Technology (Chapel Hill, NC, and IBM PC). A tetrapolar band-electrode configuration was used. Two recording electrode bands were positioned circumferentially around the base of the neck and two bands were positioned around the thorax. Three adhesive silver-silver chloride spot electrodes were used to independently measure the electrocardiogram (ECG) signal. Two spot electrodes were placed bilaterally on the upper rib cage,

with a ground spot electrode placed on the right hipbone. Mean values of HR, SV, TPR and CO were obtained from the recordings of the C.O.P. system for each minute.

*Heart Rate Variability.* Heart rate variability (HRV) offers a non-invasive and quantitative method of investigating autonomic effects on the heart. HRV refers to the complex beat-to-beat variation in HR produced by the interplay of sympathetic and parasympathetic neural activity (Stein, Bosner, Kleiger, & Conger, 1994; Levy, 1990). Spectral analyses techniques can distinguish among sources of HRV, as these rhythms occur at different frequencies. Typically, relative power in high frequency (HF) areas ( $>.015$  Hz) has been used to infer parasympathetic nervous activity.

The ECG signal was sampled (at 1000 Hz), digitized and stored for offline processing using Nevrokard software (Bio-Impedance Technology Inc., Chapel Hill, NC, USA). Before calculating HRV, R-wave markers in the ECG signal were evaluated for artifacts by visual inspection. Suspected artifacts were corrected manually. After artifact correction HF-HRV was determined in accordance with current guidelines (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). Spectral power values were determined (in normalized units) using fast Fourier transformations for 5 minute segments.

### *Ambulatory Measures*

Participants were instrumented with an oscillometric ambulatory BP monitor (Oscar 2 Ambulatory BP Monitor, Suntech Medical Instruments, Inc., Raleigh, NC, USA) that has been validated according to the International Protocol for the validation of blood pressure measuring devices (Jones, Bilous, Winship, Finn, & Goodwin, 2004). BP was measured at 20 minute intervals during the day and 30 minute intervals during the night. Participants recorded their posture, activity level and what they are doing in an ambulatory diary whenever ambulatory BP measurements were taken (see Appendix C).

## Data Analysis

All data analyses were performed using SPSS for Windows version 14.0 (SPSS Inc., Chicago, IL, USA). To verify that the trait rumination groups were comparable on demographic variables a series of one-way (high vs. low trait ruminators) between-subjects ANOVAs were performed on the demographic variables.

### *Association between State and Trait Rumination*

A series of Pearson chi-square tests (high and low trait rumination x ruminating and not ruminating) were used to assess the association between trait rumination and state rumination 5 and 10 minutes after the termination of the stressor.

### *Baseline Cardiovascular Measures and Cardiovascular Reactivity*

ECG and impedance waveforms were ensemble averaged and scored in 60 second intervals for the entire laboratory session. Outcome measures derived from these readings (HR, SV, TPR, CO, HF-HRV) and BP measurements taken at one-minute intervals were averaged to obtain a mean value for the final 2 minutes of the baseline period (allowing for adaptation to instrumentation; Manuck, Kasprovicz, Monroe, Larkin, & Kaplan, 1989) and the entire five minutes of the public speaking task. To obtain reactivity values, change scores for each of the cardiovascular measures were calculated using the difference between the mean baseline and public speaking task values.

In order to assess the association between trait rumination and baseline cardiovascular measures a series of one-way (high vs. low trait rumination) between-subjects ANOVAs were performed on baseline cardiovascular values. Similar analyses were performed to assess the association between trait rumination and cardiovascular reactivity values.

### *Cardiovascular Recovery*

To summarize the recovery data, an area-under-the-curve (AUC) technique was used to calculate a recovery value for the BP and impedance cardiography values (HR, SV, TPR, CO). AUC

is considered to be a superior technique to summarize recovery data because it assesses both the speed and amount of recovery (Linden et al., 1997; Christenfeld, Glynn, & Gerin, 2000). AUC was calculated in reference to baseline values for the entire 15 minute recovery period, thus the recovery values emphasize changes over time rather than the distance from zero (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003).

Consistent with published guidelines, recordings of HRV were analyzed in 5 minute segments and processed using the frequency domain method. This method produces values which summarize 5 minute segments and not minute-by-minute measurements which are needed for calculating AUC. Therefore, HRV recovery was calculated based on change scores from baseline values (e.g. summary of HRV for minutes 1-5 of the recovery period – HRV baseline).

To determine the association between trait rumination and cardiovascular recovery a series of one-way (high vs. low trait rumination) between-subjects ANCOVAs were conducted on the recovery variables. Reactivity was used as a covariate in each of the analyses (e.g. HF-HRV reactivity was used as a covariate in the analysis of HF-HRV recovery).

In order to test the interaction effect of state and trait rumination on cardiovascular recovery a series of 2 (high vs. low trait ruminator) x 2 (ruminating vs. not ruminating) between-subjects ANCOVAs were conducted using reactivity as a covariate on the recovery variables. Significant interactions were followed up with one-way (ruminating vs. not ruminating) between-subjects ANCOVAs (co-varying for reactivity) in high and low trait ruminator groups. Bonferroni corrections were used to adjust for the increased chance of Type 1 error.

### *Emotions and Cardiovascular Reactivity and Recovery*

The emotions related to the negative event that the participant chose to speak about during the public speaking task were assessed using three VASs (the degree of anger, sadness and anxiety). Pearson correlations were used to assess possible associations between VAS scale ratings of anger,

sadness and anxiety following the public speaking task and cardiovascular reactivity variables.

Partial correlations were used to assess the association between ratings of anger, sadness and anxiety and cardiovascular recovery variables controlling for reactivity.

### *Ambulatory Blood Pressure Monitoring*

All ambulatory BP readings were reviewed, and artifactual readings were deleted following criteria described elsewhere (Hinderliter, Light, & Willis, 1991). Less than 10% of BP values were determined to be artifactual. Mean awake, asleep and 24-hour SBP and DBP values were calculated based on all valid ABPM readings. Participant reported time of sleep onset and awakening were used to determine the asleep period. BP dipping was defined as the difference between mean awake and mean asleep BP. To assess the association between trait rumination and ABPM a series of one-way (high vs. low trait rumination) ANOVAs were performed on the dependent variables mean awake SBP, mean awake DBP, mean asleep SBP, mean asleep DBP, mean 24-hour SBP and mean 24-hour DBP. A series of one-way (high vs. low trait rumination) ANCOVAs (co-varying for daytime BP and sleep quality) were performed on SBP and DBP dipping.

## Results

### *Participant Characteristics*

Participants had a mean age of 22.4 ( $\pm$  5.9) years and a mean body mass index of 22.5 ( $\pm$  2.8). Most of the participants were White (65.6%). A series of one-way ANOVAs indicated no significant differences between high and low trait rumination groups on any of the demographic characteristics.

### *Association between State and Trait Rumination*

Results of the Pearson chi-square test indicated that there was a significant association between trait rumination and state rumination 5 minutes after the termination of the stressor  $\chi^2(1, N = 64) = 7.35, p = .007$ . High trait ruminators were more likely than low trait ruminators to report that they were ruminating 5 minutes following the end of the stressor. A similar analysis revealed no

association between state and trait rumination 10 minutes after the termination of the stressor ( $p > .05$ ).

### *Baseline Cardiovascular Values*

A one-way ANOVA indicated an association between trait rumination and baseline SBP,  $F(1,62) = 4.24, p = .044$ . High trait ruminators had lower SBP at baseline. Therefore, baseline SBP was used as a covariate in all further analyses involving SBP. Findings indicated no association between trait rumination and any other baseline cardiovascular values (Table 1).

### *Cardiovascular Reactivity*

Results of the one-way ANOVA revealed that low trait ruminators had greater SV reactivity,  $F(1,62) = 5.41, p = .023$ . Similar analyses revealed no association between trait rumination and other reactivity values (Table 1).

### *Cardiovascular Recovery*

*Effects of Trait Rumination on Cardiovascular Recovery.* The one-way ANCOVA indicated that high trait ruminators had significantly greater DBP AUC,  $F(1,61) = 4.60, p = .036$ , reflecting inferior recovery (across the 15 minute recovery period) compared to low trait ruminators (Table 2). Similar ANCOVAs revealed no significant associations between trait rumination and any of the other recovery variables (Table 2). The next set of analyses more fully addresses the association between trait rumination and cardiovascular recovery.

*Effects of Trait Rumination x State Rumination Interaction on Recovery.* A 2 x 2 ANCOVA revealed a significant interaction between rumination 10 minutes after the termination of the stressor and trait rumination on DBP AUC  $F(1,59) = 5.96, p = .018$  (Figure 1). Post hoc analyses revealed that rumination 10 minutes after the termination of the stressor was significantly associated with greater DBP AUC in low trait ruminators,  $F(1,36) = 7.30, p = .010$ , indicating inferior recovery relative to low trait ruminators who were not ruminating. Similar post-hoc analyses revealed that state

rumination 10 minutes after the termination of the stressor was not significantly associated with DBP AUC in high trait ruminators. Results indicated no significant main effects of state or trait rumination on DBP AUC.

A similar pattern of results was seen for HF-HRV recovery. Results revealed that there was a significant interaction between rumination 10 minutes after the termination of the stressor and trait rumination on HF-HRV recovery,  $F(1,59) = 7.10, p = .010$  (Figure 2). Post hoc analysis revealed that state rumination 10 minutes after the termination of the stressor was associated with inferior HF-HRV recovery,  $F(1,36) = 7.83, p = .008$ , in low trait ruminators. In high trait ruminators, there were no significant associations between state rumination 10 minutes after the termination of the stressor and HF-HRV recovery. Additionally, results indicated a significant main effect of trait rumination on HF-HRV recovery,  $F(1,59) = 8.54, p = .005$ . There was no significant main effect of state rumination on HF-HRV recovery ( $p > .05$ ).

Results of 2 x 2 ANCOVAs indicated no significant interaction between rumination 10 minutes after the termination of the stressor and trait rumination on any of the other recovery variables. The results of similar analyses revealed no significant interactions between rumination 5 minutes after the termination of the stressor and trait rumination on any of the recovery indices.

#### *Emotions and Cardiovascular Reactivity and Recovery*

Correlational analyses revealed no significant associations between ratings of sadness, anger or anxiety and cardiovascular reactivity values. Partial correlations (controlling for reactivity) similarly indicated no significant associations between emotions and cardiovascular recovery.

#### *Ambulatory Blood Pressure Monitoring*

Results of the one-way ANOVAs indicated no significant associations between trait rumination and mean awake, asleep or 24-hour ambulatory BP values (Table 3). Analysis of nighttime DBP dipping using a one-way ANCOVA indicated that high trait ruminators had less DBP

dipping than low trait ruminators  $F(1,60) = 6.43, p = .014$ . Similar analyses of SBP dipping revealed no significant association between trait rumination and SBP dipping (Table 3).

### Discussion

The present study examined the influence of trait and state rumination on cardiovascular recovery from a negative emotional stressor. The main finding was that low trait ruminators who were ruminating 10 minutes following the termination of the stressor had inferior DBP and HF-HRV recovery compared to low trait ruminators who were not ruminating. State rumination was not associated with cardiovascular recovery in high trait ruminators. This finding was contrary to our hypothesis that high trait ruminators who were ruminating would have the poorest cardiovascular recovery. Previous research examining the effects of distraction and trait rumination on BP recovery following an anger recall stress task found that high trait ruminators in a no distraction condition (i.e. were allowed to ruminate) had the poorest DBP and SBP recovery (Gerin et al., 2006). Differences in methodology may have contributed to this discrepancy in findings. First, Gerin et al. (2006) used a measure of trait rumination that was designed specifically to measure angry rumination whereas the trait rumination scale utilized in the current study measures the tendency to ruminate about any type of negative event. Second, Gerin and colleagues' (2006) no-distraction condition is not equivalent to state rumination in the present study. In the former study a thought report technique revealed that only 31% of reported thoughts in the no-distraction condition were related to the anger recall stress task (i.e. 31% of participants in the no-distraction group indicated state rumination). In the current study a thought report technique was also used, however only participants who indicated their thoughts were related to the stress task were included in the state rumination group (i.e. 100% of participants in the state rumination group indicated state rumination). Third, the two studies used different emotional stressors. In the former study an anger recall task was used, whereas in the current study participants recalled any negative emotional event. The anger recall task resulted in

greater SBP and DBP reactivity than the stress task used in the current study (e.g. mean SBP reactivity = 20mmHg in the current study, mean SBP reactivity = 28mmHg in the former study). This discrepancy in reactivity suggests that the anger recall task used in the former study may be more stressful and the influence of state and trait rumination on cardiovascular recovery may therefore be more evident in the former investigation.

Similar to the findings for DBP, a significant interaction effect of state and trait rumination on HF-HRV recovery was observed. Low trait ruminators who were ruminating 10 minutes following the termination of the stressor had reduced HF-HRV during recovery compared to low trait ruminators who were not ruminating. HF-HRV is used to infer parasympathetic nervous activity. Reduced HF-HRV indicates parasympathetic withdrawal (reflecting continued arousal) and is associated with cardiovascular disease (Huikuri et al., 1999; Tsuji et al., 1996). To our knowledge, this is the first study to examine the influence of state and trait rumination on HRV. Previous studies examining the effects of worry (a construct similar to rumination) on HRV indicated that worry duration was associated with decreased HRV, indicating continued arousal (Brosschot & Thayer, 2006). Additionally, an investigation evaluating the influence of hostility and distraction on cardiovascular recovery following an anger recall task revealed an association between distraction and higher HF-HRV during recovery, suggesting that those who were prevented from ruminating had superior HF-HRV recovery (Neumann, Waldstein, Sellers, Thayer, & Sorkin, 2004). Thus, our results support previous findings indicating that rumination may extend HRV recovery suggesting prolonged parasympathetic withdrawal and therefore increased cardiovascular disease risk.

In contrast to our hypothesis, high trait rumination was not associated with higher mean 24-hour ambulatory BP. This finding is inconsistent with that of Oseitutu et al. (2001) who found that high trait ruminators had higher mean 24-hour ambulatory BP. This discrepancy in findings may be

due to the use of a young female sample in the current study which resulted in a relatively low and restricted range of mean 24-hour BP values (i.e. 24-hour SBP range = 106-142 in the current study, SBP range = 101-174 in the former study). Future studies should utilize a sample of men and women of diverse age and ethnicity in order to obtain a greater range of mean ambulatory BP values and also to increase the generalizability of the findings. An examination of the circadian variation in BP revealed that high trait ruminators had less nighttime DBP dipping relative to low trait ruminators. Previous research indicates that even when blood pressure is within the normal range, each 5% increment decrease in BP dipping has been associated with a 20% to 30% rise in the risk of cardiovascular death (Ohkubo et al., 2002). Thus, dipping status may be of importance in the early identification of individuals at increased risk for hypertension (Cuspidi et al., 2001; Devereux & Pickering, 1991; Liu et al., 2003; Fagard, Staessen, & Thijs, 1995; Verdecchia et al., 2002). Consequently, high trait ruminators may be at increased risk of hypertension by virtue of their inferior DBP dipping profile.

Systolic blood pressure recovery and SBP dipping were not associated with state or trait rumination in any of the performed analyses. Previous research using an anger recall task similarly found that trait rumination was significantly associated with DBP recovery but not SBP recovery in the no-distraction condition (Gerin et al., 2006). Although results were not significant for SBP the findings involving DBP are meaningful as DBP is considered to be reflective of the flexibility of artery walls and to have increased prognostic significance in young people (below the age of 45) (Malhorta & Townsend, 2000).

Recent investigations of the association between rumination and cardiovascular recovery have focused on anger related stress (Gerin et al., 2006; Neumann et al., 2004). A strength of the current study was that we utilized a negative emotional stressor that was not restricted to evoke only anger.

The emotion evoked by the stressor (anger, sadness, and/or anxiety) was not associated with cardiovascular recovery. Therefore, cardiovascular recovery from varying emotional stressors may be influenced by rumination.

In addition to the limitations already noted, a key limitation of the current study was that state rumination was narrowly defined as ruminating about the laboratory stress task (rumination about other events during the recovery period was not assessed). High trait ruminators could have been ruminating about events other than the stress task and this may explain why high trait ruminators' cardiovascular recovery did not appear to be influenced by state rumination as defined in the current study. Future studies should assess whether state rumination following stress influences cardiovascular recovery regardless of whether this rumination relates to the most recent stressor.

The current study suggests that rumination may detrimentally influence cardiovascular functioning. Interventions targeting rumination may affect cardiovascular recovery and the associated risk of future hypertension in high trait ruminators. One simple intervention targeting rumination that has recently shown promising results of a physiological nature (i.e., decreased somatic complaints) is worry postponement (Brosschot & Van der Doef, 2006). The impact of an intervention designed to decrease rumination may be especially relevant in individuals who have multiple risk factors for CVD including a ruminative response style.

In summary, the results of the current study provide support for the hypothesis that state rumination is associated with delayed cardiovascular recovery in young women who do not normally ruminate. Furthermore, findings indicate that high trait rumination is associated with decreased DBP dipping. Taken together, results suggest that rumination may play a role in the association between stress and hypertension by prolonging cardiovascular activation following stress and extending physiological arousal into nighttime sleep.

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Table 1

*Association between Trait Rumination and Cardiovascular Baseline Values*

| Cardiovascular Measure (units)  | Trait Rumination Mean (SD) |                 |                 | ANOVA Results |          |
|---------------------------------|----------------------------|-----------------|-----------------|---------------|----------|
|                                 | High Ruminators            | Low Ruminators  | Total           | <i>F</i> (df) | <i>p</i> |
| Baseline                        |                            |                 |                 |               |          |
| SBP (mmHg)                      | 107.42 (7.24)              | 111.56 (8.22)   | 109.95 (8.06)   | 4.24 (1,62)   | .044*    |
| DBP (mmHg)                      | 66.22 (5.73)               | 69.14 (8.50)    | 68.00 (7.62)    | 2.28 (1,62)   | .136     |
| HR (beats/minute)               | 76.84 (9.11)               | 77.12 (9.15)    | 77.00 (9.07)    | 0.01 (1,62)   | .907     |
| SV (ml/beat)                    | 138.98 (43.31)             | 154.83 (42.10)  | 148.64 (42.95)  | 2.12 (1,62)   | .151     |
| TPR (dyne-sec <sup>cm-5</sup> ) | 653.18 (198.72)            | 598.63 (148.41) | 619.94 (170.44) | 1.58 (1,62)   | .214     |
| CO (L/min.)                     | 10.49 (2.74)               | 11.73 (2.52)    | 11.24 (2.66)    | 3.47 (1,62)   | .067     |
| HF-HRV (n.u.)                   | 47.17 (19.99)              | 54.17 (18.77)   | 51.44 (19.41)   | 2.02 (1,62)   | .161     |

*Note.* Reactivity values presented as change scores from baseline, SBP = systolic blood pressure, DBP = diastolic blood pressure, HR = heart rate, SV = stroke volume, TPR = total peripheral resistance, CO = cardiac output, HF-HRV = high frequency heart rate variability, n.u. = normalized units

\**p* < .05

Table 2

*Association between Trait Rumination and Cardiovascular Reactivity Values*

| Cardiovascular Measure (units)  | Trait Rumination Mean (SD) |                |                | ANOVA Results |          |
|---------------------------------|----------------------------|----------------|----------------|---------------|----------|
|                                 | High Ruminators            | Low Ruminators | Total          | <i>F</i> (df) | <i>p</i> |
| Reactivity                      |                            |                |                |               |          |
| SBP (mmHg)                      | 18.88 (7.29)               | 21.39 (7.84)   | 20.41 (7.67)   | 1.65 (1,62)   | .204     |
| DBP (mmHg)                      | 13.86 (5.71)               | 14.12 (9.21)   | 14.02 (7.98)   | 0.02 (1,62)   | .899     |
| HR (beats/minute)               | 18.54 (11.90)              | 20.73 (12.30)  | 19.88 (12.10)  | 0.49 (1,62)   | .485     |
| SV (ml/beat)                    | -8.43 (16.56)              | -19.99 (20.98) | -15.47 (20.02) | 5.41 (1,62)   | .023*    |
| TPR (dyne-sec <sup>cm-5</sup> ) | 20.46 (98.79)              | 47.85 (97.65)  | 37.15 (98.24)  | 1.18 (1,62)   | .280     |
| CO (L/minute)                   | 1.90 (2.51)                | 1.28 (1.79)    | 1.52 (2.10)    | 1.33 (1,62)   | .253     |
| HF-HRV (n.u.)                   | -20.56 (19.48)             | -26.88 (17.41) | -24.41 (18.36) | 1.83 (1,62)   | .181     |

*Note.* Reactivity values presented as change scores from baseline, SBP = systolic blood pressure, DBP = diastolic blood pressure, HR = heart rate, SV = stroke volume, TPR = total peripheral resistance, CO = cardiac output, HF-HRV = high frequency heart rate variability, n.u. = normalized units

\*  $p < .05$

Table 3

*Association between Trait Rumination and Cardiovascular Recovery*

|                      | Trait Rumination<br>Mean (SD) |                   |                  | ANOVA<br>Results |          |
|----------------------|-------------------------------|-------------------|------------------|------------------|----------|
|                      | High<br>Ruminators            | Low<br>Ruminators | Total            | <i>F</i> (df)    | <i>p</i> |
| Recovery             |                               |                   |                  |                  |          |
| SBP AUC <sup>a</sup> | 43.33 (45.15)                 | 60.65 (70.03)     | 53.89 (61.71)    | 0.92 (1,60)      | .342     |
| DBP AUC              | 9.72 (65.80)                  | -22.65 (87.47)    | -10.00 (80.73)   | 4.60 (1,61)      | .036*    |
| HR AUC               | 0.46 (45.98)                  | 10.74 (55.41)     | 6.72 (51.80)     | 0.25 (1,61)      | .615     |
| SV AUC               | 145.42 (156.57)               | 78.02 (214.71)    | 104.35 (195.56)  | 1.16 (1,61)      | .286     |
| TPR AUC              | -237.48 (486.60)              | -219.68 (958.44)  | -226.63 (802.72) | 0.23 (1,61)      | .632     |
| CO AUC               | 10.92 (16.57)                 | 8.02 (16.65)      | 9.16 (16.55)     | 0.11 (1,61)      | .744     |
| HF-HRV recovery      | -4.77 (14.91)                 | -11.99 (14.61)    | -9.17 (15.03)    | 1.92 (1,61)      | .171     |

*Note.* Reactivity was used as a covariate in all analyses, AUC = area under the curve, SBP = systolic blood pressure, DBP = diastolic blood pressure,

HR = heart rate, SV = stroke volume, TPR = total peripheral resistance, CO = cardiac output, HF-HRV = high frequency heart rate variability

<sup>a</sup>Due to group differences at baseline, baseline SBP was used as a covariate in analysis of SBP AUC

\*  $p < .05$

Table 4

*Association between Trait Rumination and Ambulatory Blood Pressure*

|                          | Trait Rumination<br>Mean (SD) |                   |               | ANOVA<br>Results |          |
|--------------------------|-------------------------------|-------------------|---------------|------------------|----------|
|                          | High<br>Ruminators            | Low<br>Ruminators | Total         | <i>F</i> (df)    | <i>p</i> |
| Awake SBP                | 124.00 (7.80)                 | 127.15 (9.60)     | 125.92 (9.01) | 1.90 (1,62)      | .174     |
| Awake DBP                | 74.88 (5.33)                  | 75.69 (7.49)      | 75.38 (6.70)  | 0.22 (1,62)      | .640     |
| Asleep SBP               | 112.00 (10.69)                | 111.27 (7.95)     | 111.56 (9.05) | 0.10 (1,62)      | .756     |
| Asleep DBP               | 63.56 (9.86)                  | 59.86 (6.37)      | 61.31 (8.05)  | 3.34 (1,62)      | .072     |
| 24-Hour SBP              | 120.32 (8.16)                 | 123.08 (8.67)     | 122.00 (8.52) | 1.61 (1,62)      | .209     |
| 24-Hour DBP              | 71.72 (6.24)                  | 71.61 (6.76)      | 71.66 (6.51)  | 0.00 (1,62)      | .951     |
| SBP dipping <sup>a</sup> | 13.04 (5.84)                  | 15.97 (7.84)      | 14.83 (7.22)  | 1.20 (1,60)      | .279     |
| DBP dipping <sup>a</sup> | 11.78 (6.38)                  | 15.89 (5.90)      | 14.29 (6.38)  | 6.43 (1,60)      | .014*    |

*Note.* Dipping was calculated as a change score (awake - asleep), SBP = systolic blood pressure, DBP = diastolic blood pressure

<sup>a</sup>Mean awake blood pressure and sleep quality used as covariates

\*  $p < .05$

Figure 1

*Association between trait rumination by state rumination interaction and DBP AUC*

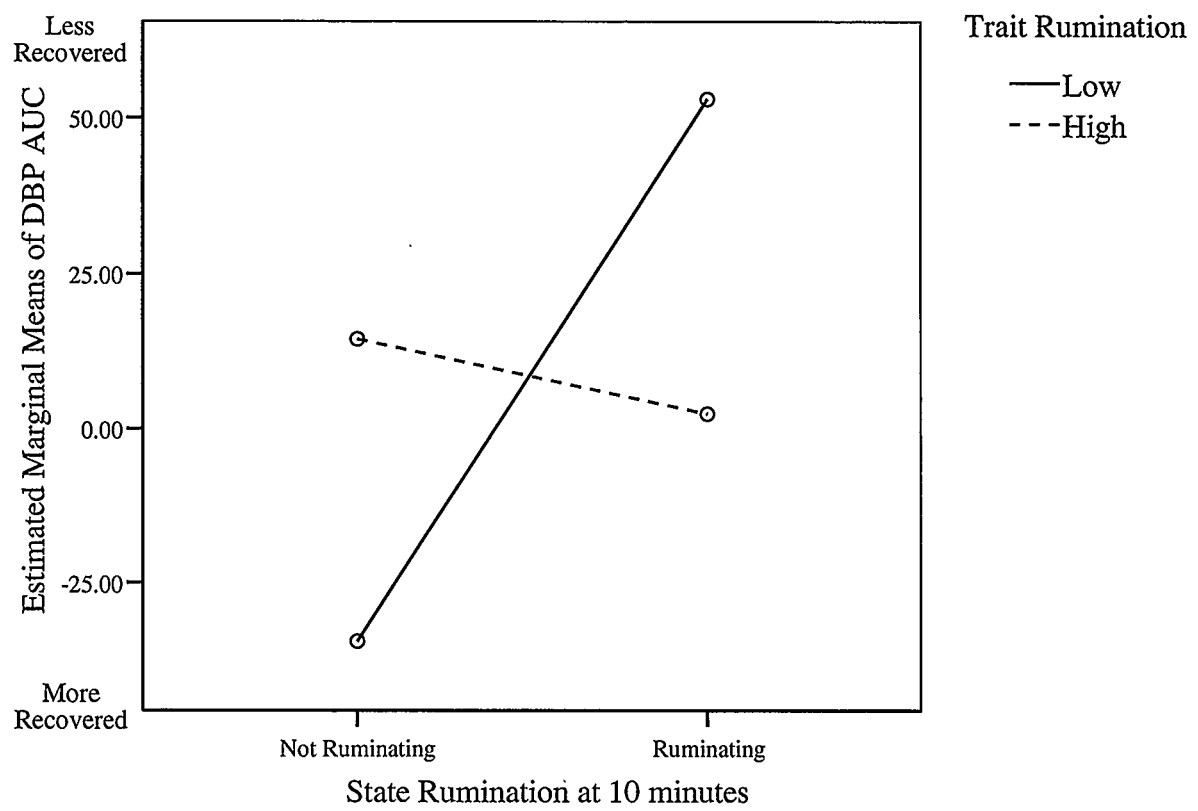
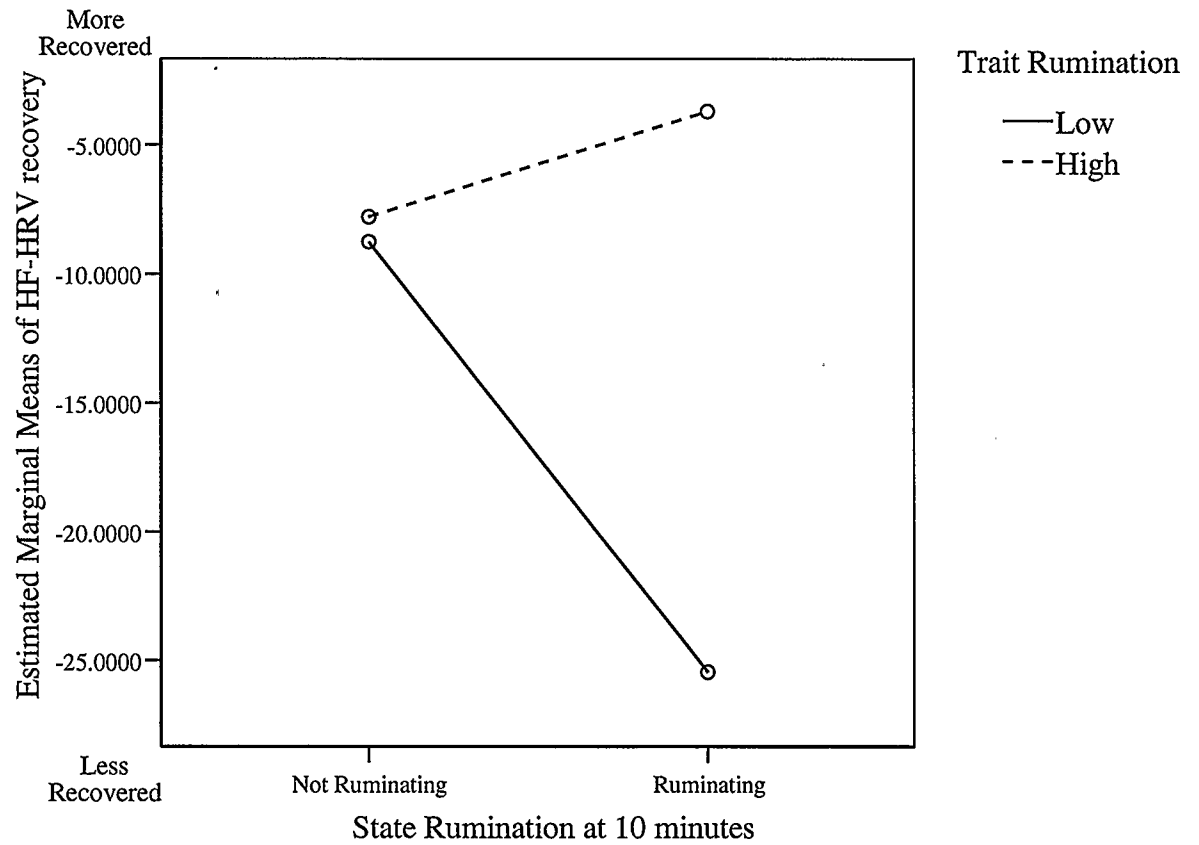


Figure 2

*Association between trait rumination by state rumination interaction and HF-HRV recovery*



## Appendix A

### Stress Reactive Rumination Scale



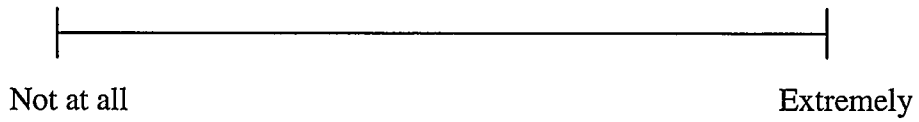


## Appendix B

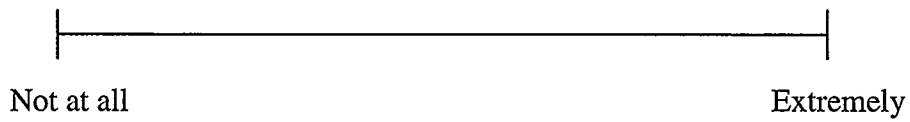
### Visual Analogue Scales

Ratings of Negative Emotional Stressor Spoken about During Public Speaking Task

At the time of this event how upsetting would you say this event was for you?



How upsetting did you find it talking about this event today?

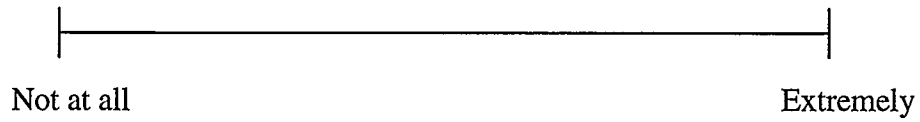


Would you say that this event and the issues or emotions surrounding it have been resolved?

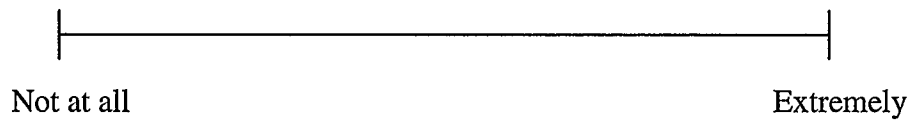


What emotions would you say this event caused you to experience, either during or after the event?

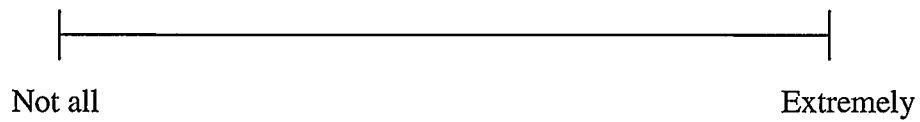
Did it cause you to feel sad?



Did it cause you to feel angry?



Did it cause you to feel anxious?



Appendix C  
Ambulatory Diary

**Time:** \_\_\_\_\_

**Location:** (Circle one)

Home      School Work      Other: \_\_\_\_\_

**Posture:** (Circle one)

Sitting      Standing      Laying Down

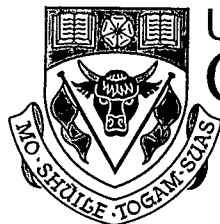
**Current Activity:** (Circle ALL that apply)

|                  |                   |                 |
|------------------|-------------------|-----------------|
| Walking          | Talking/Listening | Eating/Drinking |
| Household Chores | School Work       | Driving         |
| Exercise         | Other: _____      |                 |

**Smoking:** Since your last entry have you had any tobacco? (Circle one)

Yes                      No

Appendix D  
Ethics Approval



## CERTIFICATION OF INSTITUTIONAL ETHICS REVIEW

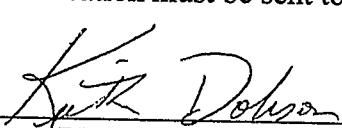
This is to certify that the Conjoint Faculties Research Ethics Board at the University of Calgary has examined the following research proposal and found the proposed research involving human subjects to be in accordance with University of Calgary Guidelines and the Tri-Council Policy Statement on *"Ethical Conduct in Research Using Human Subjects"*. This form and accompanying letter constitute the Certification of Institutional Ethics Review.

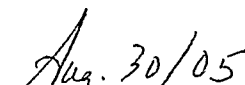
File no: **4515**  
 Applicant(s): **Brenda L. Key**  
 Department: **Psychology**  
 Project Title: **The Influence of Rumination on Cardiovascular Recovery From Stress**  
 Sponsor (if applicable):

### Restrictions:

**This Certification is subject to the following conditions:**

1. Approval is granted only for the project and purposes described in the application.
2. Any modifications to the authorized protocol must be submitted to the Chair, Conjoint Faculties Research Ethics Board for approval.
3. A progress report must be submitted 12 months from the date of this Certification, and should provide the expected completion date for the project.
4. Written notification must be sent to the Board when the project is complete or terminated.

  
 Keith Dobson, Ph.D,  
 Acting Chair  
 Conjoint Faculties Research Ethics Board

  
 Date: **Aug. 30/05**

**Distribution:** (1) Applicant, (2) Supervisor (if applicable), (3) Chair, Department/Faculty Research Ethics Committee, (4) Sponsor, (5) Conjoint Faculties Research Ethics Board (6) Research Services.