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Perseverative Cognition, Cognitive Load, and Distraction in Recovery from Stress

by

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A thesis submitted in partial fulfillment
of the requirements for the degree of
Master of Arts
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Abstract

Perseverative cognition is the repetitive cognitive representation of a stressor, which includes the concepts of worry and rumination. These thoughts delay post-stress cardiovascular recovery, which may lead to an increased risk for cardiovascular disease. This may be due to the negative emotional content of perseverative cognition or because it involves cognitive effort. The aim of this study was to identify the unique influences of negative emotional content and cognitive effort during recovery. Participants ($N = 120$) were given a demanding task purportedly as a measure of intelligence and then given false negative feedback. Immediately following, participants engaged in one of four recovery instruction conditions: think about task performance, perform a cognitive load task, watch a distracting video, or remain quietly seated. EKG, impedance cardiography, and blood pressure were recorded throughout. Perseverative cognition and cognitive load both resulted in significantly less heart rate recovery compared to the distracting video. Higher test motivation and anxiety were related to more blunted reactivity and delayed recovery of respiratory sinus arrhythmia. Reduced recovery during perseverative cognition and cognitive effort indicate that the cognitive load produced by perseveration is the pernicious component that explains its link to increased risk for cardiovascular disease. Further, the relationship between motivation/anxiety and blunted reactivity and recovery suggest effort may be important in the link between perseverative cognition and cardiovascular disease.

Background

The American Heart Association (2009) estimates that nearly 80 million American adults (one in three) have some form of cardiovascular disease (CVD). Measures of CVD include high blood pressure, coronary heart disease (encompassing myocardial infarction and angina pectoris), heart failure, and stroke. An estimated 35.3% of all deaths in 2005 were a result of CVD, accounting for more deaths than any other cause. The American Heart Association (2009) has identified many risk factors such as high cholesterol, high blood pressure, smoking, and diabetes. While these traditional risk factors are important in the development of CVD, research demonstrates that extreme acute and chronic stress can also be risk factors for CVD (Krantz & McCeney, 2002). A recently developed theory, the perseverative cognition hypothesis, indicates that a stressor may not necessarily be extreme or chronic to act as a risk factor for CVD (Brosschot, Pieper, & Thayer, 2005). Perseverative cognition may extend a mild acute stressor and increase the chance of CVD without the stressor being explicitly present. The mechanism by which perseverative cognition increases the risk for CVD is not completely understood and needs to be evaluated in greater detail.

Perseverative Cognition

Perseverative cognition is defined as the repetitive cognitive representation of a psychological stressor, and therefore includes the concepts of worry, rumination, and anticipatory stress (Brosschot, Gerin, & Thayer, 2006). Rumination is an activity that involves repetitively and passively focusing on symptoms of distress and the causes and

consequences of these symptoms (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). People who ruminate fixate on problems that they experience instead of actively trying to solve those problems. Worry is different from rumination in that the person attempts to actively engage in problem-solving an uncertain situation, but the thoughts and images that come from the problem-solving are more fixated on the negative outcomes (Borkovec, Robinson, Pruzinsky, & DePree, 1983). Finally, anticipatory stress is different from both rumination and worry because the cognition is focused on a very specific known stressor that is coming. While all of these are different, they all share the common characteristic of creating a recurring thought, which turns an acute stressor into something that is more long term (Brosschot et al., 2006). This perseverative cognition changes a short stress response to a prolonged stress response, which leads to pathogenic states that can increase the risk for organic diseases, like CVD.

Rumination is specifically linked to depression; during periods of depression, individuals who engage in rumination fixate on the negative causes of distress, which can increasingly impair problem solving, damage motivation and initiative, and reduce social support, making it more likely for the individual to develop a depressive disorder (Nolen-Hoeksema et al., 2008). In individuals with no signs of heart disease at baseline, depressive symptoms prospectively increase the risk for the development of CVD (Wulsin & Singal, 2003). Also, the focus on possible negative outcomes in worry relates strongly to fear, and has been identified as one of the defining factors in anxiety disorders (American Psychiatric Association, 1994). Similar to depression, general anxiety disorder is also associated with an elevated risk for developing CVD (Barger & Sydeman, 2005). Perseverative cognition in the form of worry and rumination may lead to

depression and anxiety disorders, contributing to the risk of CVD. While depression and anxiety disorders are both related to higher levels of CVD, this relationship may be clouded by their high comorbidity (Goodwin, Davidson, & Keyes, 2009). When Goodwin et al. (2009) controlled for the relationship of anxiety disorders, the significant relationship between depression and CVD no longer remained.

Perseverative cognition can also occur independently from depression and anxiety disorders. In a series of experiments, Meyer, Miller, Metzger, and Borkovec (1990) developed the Penn State Worry Questionnaire (PSWQ), a highly reliable measure of trait worry that was not correlated with indices of anxiety and depression in individuals with generalized anxiety disorder. Trait worry was found to predict the frequency and duration of worrying in a sample of high school students, even after controlling for anxiety (Brosschot & van der Doef, 2006). Worry also predicted the number of health complaints reported by these students. Kubzansky et al. (1997) also found that in healthy elderly men, the duration of time spent thinking about social and financial issues was related to future levels of CVD. Perseverative cognition through worry can increase the frequency and duration of worrying, which may lead to an increased risk of CVD.

Allostatic load model. One mechanism through which perseverative cognition results in CVD is allostatic load. The allostatic load model (McEwen & Seeman, 1999) explains how the normal functioning of the stress system can be pushed to abnormal limits and lead to CVD. Allostasis is defined as the process that maintains homeostasis. However, if any system responsible for allostasis is taxed too much, the system is pushed into a state of allostatic load, identified as the breakdown of the system due to excessive wear and tear.

McEwen and Seeman (1999) have identified three types of allostatic load, which can build on themselves and result in a disease state. Type 1 allostatic load is related to the magnitude and frequency of stress (McEwen, 1998). Repeated or exaggerated responses can itself lead to a disease state or push the system into Type 2 or 3 allostatic load. Type 2 allostatic load is a failure to shut down the response and return to baseline while Type 3 allostatic load is a failure to respond to the stressful challenge. The development of disease is not consistently seen at only one stage, but could result from any of these types of allostatic load.

Existing literature has linked perseverative cognition to all of these allostatic load pathways. Asking individuals to worry can result in physiological activation, and if worry is frequent, this would be an example of the repeated-hits pathway (Thayer, Friedman, & Borkovec, 1996). Women high in trait worry have been shown to respond with greater reactivity to a non-cued noise blast than women low in trait worry, an example of the exaggerated response pathway (Delgado et al., 2009). Individuals high in trait rumination demonstrated delayed recovery from an anger recall task, an example of the failure-to-shut-down pathway (Gerin, Davidson, Christenfeld, Goyal, & Schwartz, 2006). Finally, individuals with anxiety disorders showed blunted reactivity compared to control subjects when asked to worry using mental imagery, an example of the failure-to-respond pathway (Lyonfields, Borkovec, & Thayer, 1995).

Perseverative cognition may relate to all three types of allostatic load, but an important distinction must be made in the allostatic load induction mechanisms. Type 1 allostatic load occurs during the initial stressor and as such, is captured by examining cardiovascular reactivity. Similarly, Type 3 allostatic load is a dysfunction of

cardiovascular reactivity; the system demonstrates a blunted response to the stressor. However, Type 2 allostatic load is different in that the dysfunction occurs after the stressor itself has terminated. In these situations, the disease state is related to delayed cardiovascular recovery. Historically, cardiovascular stress research has focused on reactivity and ignored recovery, with two-thirds of the articles published reporting reactivity without recovery (Linden, Earle, Gerin, & Christenfeld, 1997). Perseverative cognition mirrors this trend and much of the literature focuses on the reactivity mechanisms, but delayed cardiovascular recovery may be just as important in the prolonged stress response seen in perseverative cognition (Brosschot, Pieper, & Thayer, 2005).

Perseverative cognition manipulations. Perseverative cognition is commonly induced in the laboratory by prompting either worry induction or anger recall (Thayer et al., 1996; Gerin et al., 2006). The prompts involve asking participants to worry as they normally do, or to think back to a time when they were made upset. Both of these manipulations result in cardiovascular reactivity and can explain how perseverative cognition may lead to CVD through the repeated-hits pathway seen in Type 1 allostatic load. Worry induction and anger recall can both lead to increases in cardiovascular activity, but at the same time, moral dilemma tasks, asking participants to think about whether hypothetical actions are right or wrong, also produce reactivity that is similar to that of a worry induction task (Verkuil, Brosschot, Borkovec, & Thayer, 2009).

Additionally, when comparing these two groups' self-reported intensity of thought for the worry induction and moral dilemma tasks were not different, suggesting that cognitive

activity in general may be responsible for the increase in cardiovascular activity and not perseverative cognition, *per se*.

Recovery from a stressor may also be affected by perseverative cognition as both moral dilemma questions (Glynn, Christenfeld, & Gerin, 2002) and reading an article on life in space (Neumann, Waldstein, Sollers, Thayer, & Sorkin, 2004) resulted in faster recovery from a mental arithmetic stressor with harassment compared to a group given no instructions. Reading the article about space was found to reduce the number of thoughts the participants had during the recovery period, suggesting that both reading the article and thinking about moral dilemma questions circumvented ruminative thoughts through distraction. The groups who did nothing during the recovery period would then presumably be engaging in perseverative cognition. This may be the case considering that individuals who were harassed during a mental arithmetic task did not recover to the same level as those who did not receive harassment (Suarez, Harlan, Peoples, & Williams, 1993). Anger is usually elicited by harassment during a mental arithmetic (Glynn et al., 2002). Hostility may be important to recovery from the task because hostility is defined as reacting to common events with anger and resentment, and expressing those feelings with antagonism and disagreeableness (Felsten, 1996). High trait hostility is predictive of both slower and less recovery from mental arithmetic with harassment (Neumann et al., 2004; Suarez et al., 1993), potentially due to hostile individuals who are not allowed to outwardly express anger engaging in perseverative cognition, leading to the overall reduction in recovery (Suchday, Carter, Ewart, Larkin, & Desiderato, 2004).

Perseverative cognition may increase the risk for CVD through prolonged and repeated activation of the cardiovascular system, but the same level of activity happens in response to an equally challenging moral dilemma task (Verkuil et al., 2009). Also, perseverative cognition that leads to delayed recovery may be the result of greater thinking in general (Neumann et al., 2004). It is difficult to separate cognitive load from perseverative cognition in both of these studies. When looking only at cognitive load, increasing mental load by requiring more letters to be remembered during a visual search task resulted in an increased cardiovascular reactivity, which was not related to performance on the task (Althaus, Mulder, Mulder, van Roon, & Minderaa, 1998). Perseverative cognition may increase the risk for CVD simply by increasing cognitive load, and nothing that is specific to the cognitive focus on the previous stressor.

Evidence suggests that perseverative cognition is unique from cognitive load because anger recall and logical problems produce similar levels of cardiovascular reactivity, but following these tasks, there is greater activity after anger recall than cognitive load (Ottaviani, Shapiro, Davydov, & Goldstein, 2008). Perseverative cognition is the repeated mental representation of past psychological stressors (Brosschot et al., 2006), and the stressful event may be held in mental activity after the termination of the task. This may point to the specific mechanism by which perseverative cognition increases the risk for developing CVD. It is important to separate the effects of perseverative cognition from simple cognitive load tasks, as well as identifying differences between the two actions after active engagement.

Cardiovascular Responses during Perseverative Cognition

Heart rate. High resting heart rate has been identified as a risk factor for all-cause mortality (Palatini & Julius, 1997). Perseverative cognition may lead to higher heart rate through allostatic load. For example, women high in trait worry responded to a non-cued noise blast with a greater increase in heart rate (Delgado et al., 2009), an example of Type 1 allostatic load. In a different sample of women, higher trait worry was related to higher resting heart rate, which continued through a battery of laboratory stressors (Knepp & Friedman, 2008). Further, higher resting heart rate is observed among individuals with generalized anxiety disorder (Thayer et al., 1996). However, in individuals with anxiety disorders, heart rate is blunted in response to worry induction (Lyonfields et al., 1995), an example of Type 3 allostatic load. Greater reactivity or sustained activation in individuals with high trait worry may lead to the blunted reactivity seen in anxiety disorders.

These systematic changes in heart rate may be explained through the influence of the parasympathetic nervous system. Heart rate is under the control of both the sympathetic and parasympathetic branches of the autonomic nervous system. At rest, the parasympathetic nervous system has a tonic inhibitory effect on heart rate, via the vagus nerve. This tonic inhibitory effect has been termed the “vagal brake” (Porges, 2007). In response to a stressor, heart rate can be increased through activation of the sympathetic nervous system or inhibition of parasympathetic nervous system. The reduced parasympathetic activity leading to increased heart rate is likened to a car brake; during a stressor, reduced parasympathetic influence, or a release of the “brake” that slows heart rate during rest, causes increased heart rate. The influence of the parasympathetic

nervous system is most evident in variability in the timing between heart beats coincident with respiration. Upon inhalation, parasympathetic influence is blocked and heart rate accelerates, while exhalation releases the block and heart rate decelerates. This phenomenon is known as respiratory sinus arrhythmia (RSA) because of this relationship to respiration, and is considered a strong measure of parasympathetic activity (Bernston et al., 1997).

Lower resting RSA is considered a risk factor for CVD. For example, lower RSA is a predictor of future hypertension in initially normotensive males (Singh et al., 1998). Similarly, RSA levels have been found to be a predictor for future cardiac events (Tsuji et al., 1996). The allostatic load pattern seen with heart rate can also be observed with respect to RSA. Brosschot, Van Dijk, and Thayer (2007) related daily levels of worry to lower levels of RSA during waking and sleep. Low RSA levels during sleep can also be induced experimentally through anticipatory threat of a speech to be delivered upon awakening (Hall et al., 2004). This extended reduction in RSA from worry may again lead to the Type 2 and 3 allostatic load patterns of lower resting RSA (Thayer et al., 1996) and reduced reactivity (Lyonfields et al., 1995) seen in individuals with anxiety disorder.

Blood pressure. Exaggerated blood pressure responses have long been identified as a risk factor for future CVD (Krantz & Manuck, 1984). Less recovery from a mental arithmetic stressor in individuals with borderline hypertension has also been related to future development of hypertension (Borghi, Costa, Boschi, Mussi, & Ambrosioni, 1986). Almost all evidence linking blood pressure elevations to perseverative cognition involves the anger recall task or harassment. Increases in blood pressure are observed in

response to an anger recall task (Glynn, Christenfeld, & Gerin, 2007). Perseveration is then related to higher blood pressure being sustained following an anger recall event (Suchday, Carter, Ewart, Larkin, & Desiderato, 2004). Harassment also causes slower blood pressure recovery when compared to the same stressor without harassment (Glynn et al., 2002). Further, both anger and harassment have been linked to hostility (Suarez et al., 1993; Suchday et al., 2004). However, unlike the work on heart rate & RSA, there is far less evidence that perseverative cognition influences blood pressure without anger recall or harassment. Contrada, Wright, and Glass (1984) demonstrated that anticipation of a future mental arithmetic task raises blood pressure. Combining this evidence with the idea that angry rumination delays recovery (Neumann et al., 2004) suggests that perseverative cognition during recovery may delay the return of blood pressure to baseline. Thus, it may be important to separate hostility from the effects of perseverative cognition on reactivity and recovery.

Worry and Sleep

As the discussion above indicates, it may be important to consider sleep quality in the relationship between perseverative cognition and CVD. Few studies have directly examined this connection, but one study showed worry and intrusive thoughts reduce sleep quality (Hall et al., 1998). More research has looked at the relationship between daily levels of stress and cardiovascular measurements during sleep. For example, Ituarte, Kamarck, Thompson, & Bacanu (1999) found higher heart rate during sleep related to the number of stressful events over a 6-month period. Vrijkotte, van Doornen, and de Geus (2000) have also found lower RSA levels during sleep in individuals with a job coping strategy that is characterized by an inability to stop thinking about events from

the workplace. In fact, the lower level of RSA during sleep was related to future risk of hypertension. Being unable to separate from work contains the same cognitive fixation on negative and uncontrollable situations that is relevant in perseverative cognition. Cardiovascular measures obtained during sleep may be related to worry (Hall et al., 1998), but the mediating effects of worry may also be seen influencing sleep quality.

Current Study

The past research points to the ability of perseverative cognition to increase heart rate and blood pressure, while reducing RSA, which may all act as potential risk factors in the development of CVD. However, cognitive load tasks are also able to produce changes in the cardiovascular system that are similar to those produced by perseverative cognition (Verkuil et al., 2009; Ottaviani et al., 2008). The current study sought to compare perseverative cognition, cognitive load, and distraction immediate following a stressor and the differences in recovery following those tasks.

All participants engaged in the same stressful task, a battery of math, verbal and visuospatial questions. To foster perseverative cognition, participants were told the task tested intelligence and were given feedback that they had performed poorly. The task was completed without verbalization from the participant because tasks which rely on verbal responses have been shown to increase blood pressure without being related to the stressful task (Lynch, Long, Thomas, Malinow, & Katcher, 1981). Verbal responses may also increase RSA reactivity independent from stress as there is more reactivity in a verbal arithmetic task than a keyboard driven one (Sloan, Korten, & Myers, 1991). RSA changes during speaking may be partially related to the influence respiration has on RSA that is independent of the effect of vagal input (Task Force, 1996). Speaking can create artifacts in respiration because vocalization require irregular expiration of air, making accurate measurement of respiratory rate and volume difficult (Wintjes, 1992). No

verbalization occurred during the stressor or following it, to control for respiratory effects and to measure respiration more reliably as a covariate in the analysis of RSA.

A drawback of mental tasks without verbalization is less reactivity compared to the same task with speaking (Tomaka, Blascovich, & Swart, 1994). To maximize reactivity, the task included factors shown to enhance reactivity; motivated performance, social-evaluative threat, and uncontrollability (Dickerson & Kemeny, 2004).

Uncontrollability is a major factor that leads to greater reactivity, including cortisol (Dickerson & Kemeny, 2004), blood pressure, and norepinephrine responses (Peters et al., 1998), as well as greater suppression of immunological function (Brosschot et al., 1998). Uncontrollability was added by giving the battery of tasks in a randomized order, with no control over the timing available for each question. Previous literature has used harassment to enhance motivated performance and social-evaluative threat. However, in order to avoid eliciting anger in the present study, providing a performance comparison to peers was used to increase evaluative threat. Reactivity to mental arithmetic with harassment has been related to hostility (Suarez et al., 1993), so given the presentation of the task as a test of cognitive abilities and the feedback comparisons to peers, levels of test motivation and anxiety were measured.

The recovery phase included manipulations of perseverative cognition, distraction and task-related cognition. Distraction may circumvent perseverative cognition and lead to faster recovery (Glynn et al., 2002), but less thinking may also create the same effect (Neumann et al., 2004). Giving participants a similarly paced perseverative cognition task and moral dilemma task may separate these effects. A distraction group without directed cognition was included to identify how cognition specifically affects recovery.

Finally, a control group was given no distraction and allowed to think following the task; recovery in this group may be most related to state worry.

The literature suggests perseverative cognition may be related to CVD through reduced parasympathetic control on heart rate. The facial cold pressor test is a measure of the trigeminal-brainstem-vagal pathway, and can be used as a purely physiological measure of vagal reactivity (Khurana et al., 1980). When an ice pack is placed on the forehead, a response similar to the dive reflex reduces heart rate via vagal mechanisms. Khurana et al. (1980) found less reduction in heart rate in individuals with disease states like diabetes and neurological disorders like stroke or multiple sclerosis. This purely physiological task may display how worry and perseverative cognition may lead to damage to the parasympathetic nervous system through allostatic load.

Hypotheses

The following hypotheses were tested in the present study:

- 1) Increases in blood pressure and heart rate as well as decreases in RSA will be observed in a task that involves uncontrollability, motivated performance, and social-evaluation. These increases will be independent from the effects of hostility.
- 2) Perseverative cognition following the task will result in delayed recovery.
- 3) Cognitive load and distraction following the task will both facilitate recovery.
- 4) Recovery in a control group with no instructions will be related to worry.
- 5) Cardiovascular activity during the second recovery period will also be related to worry.
- 6) Response to a facial cold pressor will be reduced in individuals with high worry.

Method

Participants

A total of 120 participants were recruited from the undergraduate pool at the University of South Florida and were compensated with course credit. The data from three participants could not be used in the analyses because of problems with the equipment, and four participants were removed due to reports of suspicion. The final sample was a total of 113, which included 21 male participants and 92 female participants. The age of the participants ranged from 18-51 ($M = 20.92$, $SD = 5.08$).

Participants were free of any diagnosis of cardiovascular disease, hypertension, and arrhythmias. Also, participants who were taking any medication that could affect the cardiovascular system, had diabetes, or were pregnant were excluded. All participants were asked to avoid alcohol, caffeine, nicotine, nonprescription drugs, and physical exercise for the 2 hours prior to coming to the lab.

Measures

Psychosocial measures.

Anxiety. The Beck Anxiety Inventory (BAI) was used to measure self-reported levels of anxiety (Beck, Epstein, Brown, & Steer, 1988). The 21-item instrument has participants rate anxiety symptoms such as “Numbness or tingling” over the past two weeks using a 4-point Likert scale (*Not at all* to *Severely – I could barely stand it*). The BAI demonstrated a very high internal reliability ($\alpha = 0.91$).

Depression. The Beck Depression Inventory – II (BDI-II) is a 21-item questionnaire that is commonly used to measure severity of depression (Beck, Steer, & Brown, 1996). The BDI-II contains groups of statements about depression symptoms like sadness, loss of pleasure, and self-dislike. Individuals have to pick one out of the four statements that best describes feeling over the past two weeks. The BDI-II also had high internal reliability ($\alpha = 0.89$).

Worry. The Penn State Worry Questionnaire (PSWQ) was administered to measure trait levels of worry (Meyer, Miller, Metzger, & Borkovec, 1990). This questionnaire measures the excessiveness, duration and uncontrollability of worry. An example statement in this questionnaire is, “Once I start worrying, I cannot stop,” with agreement scored on a 5-point Likert scale (*Not at all typical of me* to *Very typical of me*). The 16-item PSWQ displayed a very high internal reliability ($\alpha = 0.94$).

Sleep quality. The Pittsburgh Sleep Quality Index (PSQI) was administered to measure sleep quality (Buysee, Reynolds, Monk, Berman, & Kupfer, 1989). This scale measures seven different aspects of sleep quality over the past month, such as latency to sleep onset, duration, and disturbances, with the reliability between those seven components scores documented at 0.83. This index includes questions such as, “During the past month, how many hours of *actual sleep* did you get at night? (This may be different than the number of hours you spend in bed).” Internal reliability between those seven component scores was not high when obtained for this study ($\alpha = 0.60$).

Hostility. An adaptation of the Cook-Medley Hostility scale (Cook & Medley, 1954) was used to measure hostility. The 27-item scale has been correlated highly to the Cook-Medley Hostility scale (Woodall & Matthews, 1989). This scale includes

statements such as, “When someone does me a wrong I feel I should pay him back if I can, just for the principle of the thing.” There was good internal reliability obtained from the shorter 27-item version of the scale ($\alpha = 0.82$).

Motive to avoid failure. The 6-item Motive to Avoid Failure (MaF: Hagtvet & Benson, 1997) instrument was used to measure motivation in uncertain situations, and is highly correlated to other measures of test anxiety. The MaF obtained has a high internal reliability ($\alpha = 0.84$) and includes statements such as, “I am afraid of failing when I am given a task which I am uncertain that I can solve.” Individuals scored typicality of feeling the statements on a 4-point Likert scale (*Almost never to Almost always*).

Manipulation check. Following recovery, participants were asked how much thinking about the previous task occurred during the recovery period. Participants were then asked to rate how difficult the tasks were perceived.

Cardiovascular reactivity tasks.

Cold pressor task. A reusable ice pack was placed on the participants’ forehead for three minutes. The temperature of the ice pack was kept between 0°C and 3°C. If the task became unbearable, the participant was allowed to remove the ice pack. No participant removed the ice pack over the three minute period. This task was used to reduce heart rate and increase RSA through the activity of the trigeminal-brainstem-vagal pathway (Khurana et al., 1980).

Stress task. The stress task consisted of a computerized battery of questions. The task was designed to maximize reactivity, by fostering motivated performance, uncontrollability, and social-evaluation. Motivated performance was elicited by stating time as well as accuracy on the task would be recorded and scored. Uncontrollability was

maximized by switching between three types of problems: mental arithmetic, mental rotation, and verbal antonyms. Previous literature has demonstrated that mental arithmetic using only keyboard input increases heart rate (Sloan et al., 1991), mental rotation tasks elevates blood pressure (Smith & O’Keeffe, 1988), and verbal problems result in cardiovascular reactivity (Ottaviani, Shapiro, Goldstein, James, & Weiss, 2006). Switching between problem types was used to elevate uncontrollability and produce more reactivity. The duration of each question was set at up to eight seconds based on previous literature (Smith & O’Keeffe, 1988). Finally, social-evaluation was used by telling participants the scores would be compared to other students that had taken the test (Smith, Nealey, Kircher, & Limon, 1997). The mental rotation and verbal questions were presented as multiple choice questions to allow responses to all questions, but uncertainty over the correct answer. Following the task, all participants were given performance feedback on each of the three categories as well as overall performance on all of the categories combined. The combined score on all three categories was presented as slightly below average in order to be negative feedback that the participant could persevere on following the task.

Recovery tasks. Participants were randomly assigned to one of four separate recovery groups during the first four-minutes following the task. The cognitive task group was given a series of moral dilemmas presented on the screen for a fixed amount of time. These dilemmas were not personally relevant and were open ended to encourage thinking, with an example as “Bill is applying for a job, but his experience is not that good. Should Bill lie on his application to get the job? How big of a lie is acceptable?” Participants were asked to think about the statements, but not respond to rule out the

possibility of being judged and evaluated by others. Moral dilemmas presented in this way can be used to create an equivalent amount of cognition and physiological arousal as a worry group (Verkuil et al., 2009). The perseverative cognition group was presented with equally worded questions that addressed performance on the stressful task, such as, “Are you surprised by your score on the previous task? How do you usually compare to other students in your classes? Is your performance often better or worse than others?” Similar to the cognition group, these questions were presented at the same pacing, and responses were not elicited. Participants are instructed only to think about these open ended statements. The distraction group was presented with a four-minute clip documenting how hammers are made. This clip was chosen because it did not present human faces and other emotional content and could be displayed without sound while still being understandable, and should require less cognition from either worry or moral dilemmas. Finally, the control group was asked to sit quietly for the entire four-minutes. Following the first four-minutes, all of the groups were instructed to sit quietly for the next four-minutes, during which time no tasks and no distractions were presented.

Physiological Recording Apparatus

A Biopac MP150 system was used to measure electrocardiogram (ECG), impedance cardiography and respiration signals. An ECG100 amplifier was recorded using Cleartrace LT disposable Ag/AgCl electrodes (Conmed Andover Medical, Haverhill, MA), placed in a modified Lead II configuration on the participant’s chest. ECG was sampled at 1000 Hz. Respiration was measured with two TSD201 respiratory effort transducers placed around the abdomen and the chest and amplified using two

RSP100C respiration amplifiers sampling at 1000 Hz (Biopac Systems, Inc., Goleta, CA). A fixed volume bag was used to calibrate respiratory depth.

Impedance cardiographic signals were measured using a Biopac NICO100C (Biopac Systems, Inc., Goleta CA). A small current measuring 4mA, 100kHz signal was transmitted through disposable aluminum/mylar band electrodes around the neck and chest according to published guidelines (Sherwood et al., 1990). Transthoracic impedance waveforms (Z_0 , dZ/dt) were measured using a tetrapolar lead configuration. This signal was sampled at 1000Hz per channel by a PC. ECG, respiration, and impedance cardiography was acquired using AcqKnowledge 3.7.2 software (Biopac Systems, Inc.). Systolic (SBP) and diastolic (DBP) blood pressure were measured using an Accutorr Plus non-invasive blood pressure monitor (Datascope Corp., Mahwah, NJ) guidelines (Shapiro et al., 1996).

Procedure

Upon arrival, the participant was asked to read the informed consent form. Following the consent process, participants completed a questionnaire assessing exercise, food, caffeine, and nicotine consumption, as well as medication use. Height and weight were measured, and questionnaires were then given. After the participant finished the questionnaires, the experimenter attached two bands of Mylar tape to the participant's neck and two bands around the torso according to published guidelines for impedance cardiography (Sherwood, et al. 1990). The experimenter then used alcohol to clean the skin beneath the right collarbone and beneath the left ribcage before placement of the two Ag-AgCl electrodes in a modified lead II configuration.

Participants were then be led into a small room and seated in a comfortable chair, where the experimenter attached leads to the Mylar bands for impedance cardiography. A blood pressure cuff was then attached to the participant's left arm and several measurements were taken to ensure the equipment was working properly. Respiration transducers were also placed around the chest and abdomen to measure respiration. The experimenter then left the room and instructed the participant to watch a neutral movie about Alaska for the ten-minute resting baseline period. This task was chosen because prior research indicates that a minimally demanding task (i.e., vanilla baseline) produces a more stable estimate of physiological function than a baseline with no task (Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992).

Following the baseline participants received a state emotion questionnaire followed by instructions for the stress task. The participant was informed the experiment was examining how physiological responses were related to speed and accuracy when test taking. Each participant was then given the three minutes of the stress task followed by a one-minute display of performance. The eight-minute recovery period then followed. Participants were randomly assigned to one of the four groups before arrival, and at this point the specific instructions for the recovery group were presented. The manipulation check of the recovery task was administered followed by a manipulation check of the reactivity task. After the recovery period and before the cold pressor test, there was another five-minute resting period during which participants continued to watch the Alaska film. This ensured that all participants recovered to baseline levels prior to the cold pressor task. The participant was asked to leave the cold bag on the forehead for three minutes. After the cold pressor test, there was another ten-minute

recovery in which all groups were asked to remain seated quietly. Participants were given a manipulation check for the cold task and the final state emotion questionnaire. Finally, the participant was checked for suspicion, and then given a full debriefing.

Data Quantification and Reduction

RSA was calculated using MindWare HRV 2.51 Software module (MindWare Technologies, Ltd., Gahanna, OH). R-wave markers in the ECG signal were evaluated for artifacts by visual inspection and by the MAD/MED artifact detection algorithm (Berntson, Quigley, Jang, & Boysen, 1990) implemented in the MindWare software. Suspected artifacts were corrected manually (<1% of all R-waves in past work needed correction). This approach accords with current guidelines for frequency domain methods to determine heart rate variability (Berntson et al., 1997; Task Force, 1996). To arrive at minute-by-minute estimates of heart rate and RSA during baselines and tasks, a 60-second time series of inter-beat intervals (IBIs: the time in milliseconds between sequential ECG R spikes) was created from an interpolation algorithm that has a 250-ms sample time. This 60-second IBI time series was linearly-detrended, mean-centered, and tapered using a Hamming window. Spectral-power values were determined (in ms^2/Hz) with fast Fourier transformations, and the power values in the 0.15–0.50 Hz spectral bandwidth was integrated (ms^2). These spectral-power values were then natural-log transformed prior to statistical analyses because of distributional violations. The natural-logged spectral-power value in the 0.15–0.50 Hz bandwidth was the indicator of RSA for each minute. Averages across minutes for each experimental epoch were calculated. Primary measures of RSA reactivity and recovery were the arithmetic difference in these scores between task/recovery values and baseline values.

Mindware was used to calculate respiration rate from spectral analysis of thoracic impedance. The value of respiration obtained by analysis of thoracic impedance is highly related to that obtained by traditional strain-gauge measurement (Ernst et al., 1999). Change scores for respiration were made from baseline and also used in reactivity and recovery analyses. Respiration rate can change levels of RSA if changed from slow to rapid breathing (Berntson et al., 1997). To control for respiration, minute by minute respiration levels were residualized out of the corresponding minutes of RSA. This residualized RSA was used as a dependent variable and treated the same way as all other physiological variables.

To calculate reactivity of blood pressure, SBP and DBP readings were analyzed separately and averaged for each segment of the laboratory procedure. Thus, each participant had a calculated mean SBP and DBP value for the baseline, stress reactivity task, and recovery task. Reactivity was calculated as difference scores between each of the task segments and the baseline segment. Thus, each participant had a mean SBP and DBP change score (i.e. reactivity score) for each task.

In addition to change scores from baseline, participants' BP and HR recovery was also analyzed in alternate ways. Participants were considered fully recovered if their values returned to baseline levels during the recovery period (i.e., a difference score of zero). If a participant did not reach full recovery by the end of the recovery period, the full time was used as his or her score. In addition to time to recover, area under the curve (AUC) was calculated. This analysis compares the ratio of amount of physiological recovery compared to activation in a set time period (Kario et al., 2002). AUC may

reveal specific timing relationships of perseverative cognition that cannot be measured by looking solely at return to baseline and final recovery.

Statistical Analysis

The data were analyzed using SPSS (SPSS Inc., Chicago IL). ANOVAs were run on the four different recovery groups with the dependent variable being recovery of HR, RSA, and BP. If necessary, ANCOVAs were used with age, gender, BMI, baseline measurements, reactivity, MaF, and PSWQ as covariates.

Results

Demographic and Baseline Measurements

To ensure equivalence across the four experimental groups, ANOVAs were conducted with demographic and dispositional characteristics as the dependent measures. The groups did not differ in age or BMI as seen in Table 1. Chi-square analysis also revealed no group differences in gender composition $\chi^2(3, N = 113) = 0.93, p = .81$, caffeine use $\chi^2(3, N = 113) = 1.96, p = .58$, or nicotine use $\chi^2(3, N = 113) = 2.13, p = .55$. For trait psychosocial measures, the groups differed significantly in depression with a post hoc test revealing the control group exhibited higher scores on the BDI-II than the cognition group. The groups also differed significantly in worry with the control group scoring higher on the PSWQ than the cognition group. Finally, a significant difference was also observed in sleep quality with the cognition group exhibiting lower PSQI scores than the distraction and perseveration groups. There were no significant differences found on anxiety, hostility, or motive to avoid failure. All of these means and statistical analyses are presented in Table 1.

Table 1
Baseline Demographic and Psychosocial Means by Group

Measure	Control <i>M (SD)</i>	Cognition <i>M (SD)</i>	Perseveration <i>M (SD)</i>	Distraction <i>M (SD)</i>	<i>F</i>	<i>p</i>
Age	20.39 (3.64)	19.96 (2.78)	22.85 (7.85)	20.48 (4.29)	1.90	.13
BMI	25.23 (6.01)	24.56 (5.05)	25.44 (5.37)	24.27 (5.04)	0.30	.83
Gender						
Female	24	23	23	22		
Male	4	5	5	7		
Caffeine Users	15	13	18	17		
Nicotine Users	1	2	2	4		
BAI	9.65 (7.53)	7.04 (10.31)	7.89 (4.86)	6.64 (7.00)	0.81	.49
BDI	9.86 (7.48)*	4.86 (4.21)*	6.68 (4.15)	7.37 (8.67)	2.87	.04
CMHo	62.71 (10.85)	61.50 (5.92)	58.43 (8.74)	63.14 (9.86)	1.56	.20
MaF	13.07 (4.17)	12.07 (2.80)	11.61 (2.97)	13.34 (4.36)	1.43	.24
PSQI	6.26 (3.06)	4.39 (2.20)*†	6.82 (2.21)*	6.76 (3.54)†	4.59	<.01
PSWQ	50.54 (12.82)*	39.50 (15.12)*	46.39 (13.31)	45.66 (12.59)	3.19	.03

Note. *, † Significantly different from group with same symbol. BMI = Body Mass Index, BAI = Beck Anxiety Inventory; BDI = Beck Depression Inventory; CMHo = Cook-Medley Hostility Scale; MaF = Motive to Avoid Failure; PSQI = Pittsburgh Sleep Quality Index; PSWQ = Penn State Worry Questionnaire

Equivalency across experimental groups for the physiological measurements of heart rate (HR), respiratory sinus arrhythmia (RSA), residualized RSA, respiratory rate (RR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) were tested using ANOVAs. Baseline physiological means and ANOVA results are presented in Table 2 with no significant differences between the four groups for HR, RSA, residualized RSA, RR, SPB, or DBP. Age, gender, and BMI have known influences on cardiovascular measures, so they were added as covariates in the baseline analysis of physiological measures. With these covariates added, there were no significant differences for any of the physiological measurements. Due to group differences, BDI and PSWQ scores were added as covariates in a parallel analysis of baseline differences. In a third analysis, PSQI was also added as a covariate, but all three variations of covariates revealed virtually identical results, so only the findings with the covariates of age, gender, BMI, BDI, and PSWQ are reported and used in following analyses due to the low reliability ($\alpha = .60$) of the PSQI scale. Again, there were no significant differences between groups for HR, $F(3, 103) = 0.15, p = .93$, RSA, $F(3, 103) = 0.22, p = .89$, RR, $F(3, 103) = 0.93, p = .43$, residualized RSA, $F(3, 103) = 0.23, p = .88$, SBP, $F(3, 104) = 0.58, p = .63$, or DBP, $F(3, 103) = 0.04, p = .99$.

Table 2

Baseline Physiological Means by Group

Measure	Control <i>M (SD)</i>	Cognition <i>M (SD)</i>	Perseveration <i>M (SD)</i>	Distraction <i>M (SD)</i>	<i>F</i>	<i>p</i>
Heart Rate	73.08 (10.89)	71.49 (10.47)	71.32 (9.42)	72.90 (11.09)	0.22	.89
RSA	6.48 (.92)	6.60 (1.05)	6.36 (1.25)	6.68 (1.10)	0.46	.71
Respiration Rate	16.89 (2.67)	16.11 (2.40)	15.92 (2.08)	15.98 (2.98)	0.87	.46
Residualized RSA	.04 (.23)	.01 (.27)	.06 (.24)	.02 (.35)	0.17	.92
SBP	109.96 (11.22)	108.00 (8.32)	108.71 (9.43)	110.28 (10.34)	0.33	.80
DBP	64.49 (5.82)	64.73 (5.93)	65.67 (8.38)	65.06 (8.12)	0.14	.93

Note. RSA = Respiratory Sinus Arrhythmia; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure

Reactivity Measurements

To test the first part of hypothesis 1, that increases in blood pressure and heart rate as well as decreases in RSA will be observed during the stressful task and feedback, repeated measures ANOVAs were used to determine if physiological activity changed from baseline to task, and then in the minute following the task when negative feedback was presented. The Greenhouse-Geisser correction was used to adjust all within-subjects degrees of freedom. A significant effect of the task emerged for all physiological measures; means and the results of the repeated measures ANOVAs are presented in Table 3. Pairwise comparisons using Bonferroni correction demonstrated that HR, RR, SBP, and DBP all increased during the task. RSA and residualized RSA decreased during task. These findings support hypothesis 1. While the feedback was being presented, HR, RR, SBP, and DBP decreased from task levels. HR and SBP were significantly reduced during feedback presentation, but they were still significantly elevated from baseline measurements. During feedback presentation, RSA and residualized RSA remained suppressed from baseline and did not differ from task values.

Table 3

Physiological Reactivity from Baseline to Feedback

Measure	Baseline	Task	Feedback	<i>F</i>	<i>p</i>
	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>		
Heart Rate	72.21 (10.38)	76.86 (12.17)*	73.78 (10.87)*†	47.35	<.01
RSA	6.53 (1.08)	6.14 (1.17)*	6.26 (1.24)*	19.16	<.01
Respiration Rate	16.22 (2.55)	18.15 (2.90)*	16.37 (3.45)†	21.22	<.01
Residualized RSA	0.03 (0.28)	-0.12 (0.50)*	-0.17 (0.59)*	6.20	<.01
Systolic Blood Pressure	109.36 (9.81)	116.03 (11.64)*	111.84 (11.76)*†	70.58	<.01
Diastolic Blood Pressure	64.78 (7.15)	70.69 (8.86)*	65.52 (8.71)†	57.51	<.01

Note. * Significantly different from baseline, † Feedback significantly different from task. RSA = Respiratory Sinus Arrhythmia

To test the second part of hypothesis 1, that task increases will be independent from the effects of hostility, reactivity change scores were analyzed for group differences using ANCOVAs, controlling for age, gender, BMI, and hostility, but also depression and worry due to the baseline differences between groups. There were no group differences in task reactivity for HR, RSA, RR, residualized RSA, SBP, or DBP. Means of the groups and ANCOVA results are presented in Table 4. There were also no group differences during feedback presentation for any of the measures seen in Table 4. Given that the stressful task involved a performance based test, ANCOVAs were also used with motive to avoid failure added. There was no significant influence when MaF was added to the ANCOVAs.

Table 4

Physiological Change Scores from Baseline during Task and Feedback

Measure	Task				<i>F</i>	<i>p</i>
	Control <i>M (SD)</i>	Cognition <i>M (SD)</i>	Perseveration <i>M (SD)</i>	Distraction <i>M (SD)</i>		
Heart Rate	4.60 (4.89)	3.44 (4.33)	6.23 (6.91)	4.28 (8.29)	1.11	.35
RSA	-0.42 (0.72)	-0.39 (0.63)	-0.48 (0.62)	-0.27 (0.69)	0.43	.73
Respiration Rate	1.76 (3.35)	1.94 (2.82)	1.88 (3.47)	2.11 (3.41)	0.16	.92
Residualized RSA	-0.11 (0.66)	-0.20 (0.55)	-0.22 (0.55)	-0.08 (0.62)	0.38	.77
SBP	5.93 (4.88)	6.18 (5.84)	8.18 (6.88)	6.17 (7.52)	0.67	.57
DBP	5.85 (8.15)	5.29 (4.78)	6.71 (5.40)	4.75 (8.07)	0.47	.70
Measure	Feedback				<i>F</i>	<i>p</i>
	Control <i>M (SD)</i>	Cognition <i>M (SD)</i>	Perseveration <i>M (SD)</i>	Distraction <i>M (SD)</i>		
Heart Rate	1.55 (4.24)	1.51 (4.29)	2.50 (4.25)	0.75 (5.78)	0.61	.61
RSA	-0.31 (0.59)	-0.37 (0.92)	-0.35 (0.92)	-0.09 (0.51)	0.99	.40
Respiration Rate	-0.32 (3.86)	0.97 (2.57)	-0.48 (3.40)	0.43 (4.54)	1.22	.31
Residualized RSA	-0.29 (0.55)	-0.28 (0.82)	-0.26 (0.68)	-0.01 (0.61)	1.46	.23
SBP	0.92 (5.40)	2.37 (4.59)	3.43 (6.88)	3.18 (8.22)	0.76	.52
DBP	1.11 (5.23)	1.68 (4.03)	0.48 (5.74)	-1.05 (7.01)	1.72	.17

Note. All values are change scores from baseline. Numbers are for demonstrative purposes. All analyses were run using adjusted means. RSA = Respiratory Sinus Arrhythmia; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure

Multiple regression analyses were then performed on physiological change scores to determine if hostility was a significant predictor of reactivity, controlling for age, gender, and BMI. Hostility did not predict any changes during the task, but hostility significantly predicted a greater RR ($b = 0.08$, $t(105) = 2.53$, $p = .01$) during the feedback presentation. As previously stated, given that the stressful task was based on performance, motive to avoid failure could have also been a significant predictor after controlling for age, gender, and BMI. Motive to avoid failure was found to be a significant predictor of task reactivity with higher scores predicting less RSA ($b = 0.04$, $t(105) = 2.07$, $p = .04$) and residualized RSA ($b = 0.03$, $t(105) = 2.18$, $p = .03$) suppression. Motive to avoid failure also predicted a smaller increase in SBP ($b = -0.46$, $t(106) = -2.59$, $p = .01$). During the presentation of feedback, motive to avoid failure also significantly predicted greater RR ($b = .19$, $t(104) = 2.22$, $p = .03$).

Task Performance

To ensure that all groups performed equally, an ANOVA was conducted on the number of questions answered correctly. There were no significant differences in performance between the groups, $F(3, 109) = 1.57$, $p = .20$. Multiple regression analysis showed that BAI, BDI, CMHo, MaF, or PSWQ did not predict performance on the task.

Recovery

To test hypotheses 2 & 3, ANCOVA was performed to compare recovery values for the four different recovery groups during the first manipulated four-minute recovery time period with baseline levels, reactivity, age, gender, BMI, BDI, and PSWQ as covariates. Means and statistical analyses are presented in Table 5. There was a significant group difference in HR during the manipulated recovery period. Bonferroni

corrected pairwise comparisons revealed that the distraction group exhibited greater recovery than both the cognition and perseveration groups. No significant differences by condition emerged for RSA, RR, residualized RSA, SBP, or DBP. MaF was used as a potential covariate in reactivity, and was also added to the ANCOVA for recovery, but again, no differences in the analyses were observed. Area under the curve (AUC) and time to recover were also calculated for HR, SBP, and DBP. RSA and RR were not analyzed using these techniques because RSA and respiration tend to return to resting levels within the first minute or two after the end of the task. Group means and statistical analyses are presented in Table 6. ANCOVA with the same covariates as before revealed a significant group difference of the AUC analysis for heart rate during the manipulated recovery. There was a larger HR AUC for distraction over the cognition and perseveration groups, revealing the same pattern of increased recovery due to distraction. No significant differences in AUC were found for SBP or DBP. MaF added as a covariate caused no differences in AUC analyses.

Table 5

Physiological Change Scores from Baseline during Manipulated and Common Recovery

Measure	Manipulated				<i>F</i>	<i>p</i>
	Control <i>M (SD)</i>	Cognition <i>M (SD)</i>	Perseveration <i>M (SD)</i>	Distraction <i>M (SD)</i>		
Heart Rate	0.10 (2.83)	0.62 (1.98)*	1.56 (3.11)*	-1.74 (3.35)	7.30	<.01
RSA	0.36 (0.60)	0.33 (0.60)	0.28 (0.58)	0.05 (0.57)	1.38	.25
Respiration Rate	-0.43 (1.96)	-0.28 (1.60)	0.40 (1.90)	0.60 (2.03)	1.89	.14
Residualized RSA	0.002 (0.40)	-0.06 (0.39)	-0.11 (0.40)	-0.08 (0.43)	0.61	.61
SBP	0.07 (4.16)	1.11 (4.34)	1.59 (5.29)	-1.17 (4.90)	1.30	.28
DBP	-0.88 (4.97)	-0.69 (4.93)	0.74 (4.11)	-2.45 (6.10)	1.40	.25
Measure	Common				<i>F</i>	<i>p</i>
	Control <i>M (SD)</i>	Cognition <i>M (SD)</i>	Perseveration <i>M (SD)</i>	Distraction <i>M (SD)</i>		
Heart Rate	1.05 (2.84)*	0.67 (3.02)	0.26 (2.53)	0.16 (2.98)	3.32	.02
RSA	-0.18 (0.53)	-0.13 (0.28)	-0.12 (0.37)	-0.15 (0.41)	0.72	.54
Respiration Rate	-0.52 (2.86)	0.05 (1.55)	0.17 (1.59)	-0.23 (2.15)	1.10	.35
Residualized RSA	-0.18 (0.45)	-0.14 (0.28)	-0.10 (0.39)	-0.13 (0.35)	1.00	.40
SBP	0.07 (4.99)	0.73 (3.84)	0.71 (4.22)	-0.12 (4.68)	0.07	.98
DBP	1.37 (4.00)	0.33 (4.10)	-0.06 (4.43)	0.30 (4.75)	2.09	.11

Note. *Significantly different from distraction group All values are change scores from baseline. Numbers are for demonstrative purposes. All analyses were run using adjusted means. RSA = Respiratory Sinus Arrhythmia; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure

Table 6
Alternative Recovery Analyses of Heart Rate and Blood Pressure

Area Under the Curve						
Manipulated						
Measure	Control <i>M (SD)</i>	Cognition <i>M (SD)</i>	Perseveration <i>M (SD)</i>	Distraction <i>M (SD)</i>	<i>F</i>	<i>p</i>
Heart Rate	75.50 (20.94)	63.06 (28.05)*	64.70 (21.87)*	88.23 (11.56)	5.68	<.01
SBP	65.87 (27.65)	59.30 (27.45)	64.29 (23.92)	73.49 (24.90)	0.83	.48
DBP	72.63 (24.35)	72.66 (28.71)	68.40 (27.44)	79.58 (26.16)	1.08	.36
Common						
Measure	Control <i>M (SD)</i>	Cognition <i>M (SD)</i>	Perseveration <i>M (SD)</i>	Distraction <i>M (SD)</i>	<i>F</i>	<i>p</i>
Heart Rate	72.14 (32.13)	76.50 (27.70)	81.33 (23.28)	79.48 (26.55)	1.53	.21
SBP	76.24 (30.03)	76.14 (25.19)	81.98 (18.80)	85.28 (25.55)	0.38	.77
DBP	75.06 (23.58)	76.31 (28.99)	82.84 (25.21)	72.83 (29.78)	1.91	.13
Time to Recover						
Measure	Control <i>M (SD)</i>	Cognition <i>M (SD)</i>	Perseveration <i>M (SD)</i>	Distraction <i>M (SD)</i>	<i>F</i>	<i>p</i>
Heart Rate	3.48 (3.08)	3.22 (2.50)	4.07 (2.91)	2.42 (2.47)	0.95	.42
SBP	4.15 (2.60)	4.80 (2.65)	4.14 (2.43)	3.45 (2.20)	1.44	.24
DBP	3.85 (2.65)	3.64 (2.50)	4.30 (2.64)	3.00 (1.98)	1.62	.19

Note. * Significantly different from distraction group. All values are change scores from baseline. Numbers are for demonstrative purposes. All analyses were run using adjusted means. SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure

To test hypothesis 4, multiple regression analysis was conducted on the control group to determine if worry was a significant predictor of recovery values after controlling for baseline measures, reactivity, age, gender and BMI. Worry was not a significant predictor for any physiological variable. Motive to Avoid Failure also could have been a potential predictor given the type of stressor, but was not significant. While not a specific hypothesis, due to the potential importance of test anxiety in the evaluative task, multiple regression analyses were also conducted across all groups with both worry and motive to avoid failure added as predictors, after controlling for baseline measures, reactivity, age, gender, and BMI. These analyses revealed that higher motive to avoid failure predicted less residualized RSA recovery ($b = -.02, t(103) = -2.01, p = .05$).

To test hypothesis 5, that worry would be related to recovery during the second recovery phase, multiple regression analyses were run on all physiological change scores across all groups with worry and baseline, reactivity, age, gender, and BMI as predictors. Worry was not a significant predictor of recovery during this time period. These analyses were also conducted adding MaF, but did not significantly predict the change scores. Although there was no hypothesis that condition would be related to the second recovery, it is possible the effects seen during the first recovery may have persisted into the second. Group means and ANCOVA results are presented in Table 5. Using the baseline measurements, reactivity, previous recovery, age, gender, BMI, BDI, and PSWQ as covariates, there was a significant difference in HR with bonferonni corrected comparisons revealing a difference between distraction and the control group. We found no significant differences for RSA, RR, residualized RSA, SBP, or DBP. ANCOVAs were also conducted on the area under the curve for HR, SBP, or DBP and revealed no

differences between groups. Another method of recovery quantification, time to recover in minutes, was calculated for HR, SBP, and DBP. Again, no significant differences were found for time to recover for HR, SBP, or DBP. The alternate recovery analyses can be found in Table 6.

Manipulation Check

Self-reported measures of thinking about the stressor during the first and second recovery periods were compared across groups. No significant differences emerged between groups in how much individuals reported they thought about the tasks during the manipulated recovery period, $F(3, 109) = 0.76, p = .52$, or the second recovery period, $F(3, 109) = 0.49, p = .67$. Using multiple regression analyses to determine how BAI, BDI, CMHo, MaF, and PSWQ predicted the amount of thought revealed no differences due to these traits.

Cold-Pressor Task

Using a paired samples *t*-test to compare the second baseline to the cold task, we found that in response to the cold task HR and RR significantly decreased. Significant increases were found for all of the other variables of RSA, residualized RSA, SBP, and DBP. Means for the entire sample are presented in Table 7. In order to test hypothesis 6, that cold-pressor responses would be related to worry, multiple regression analysis was used to examine if worry was a significant predictors of change, PSWQ was not a significant predictors of the change in physiological measures after controlling for baseline and demographic values.

Table 7

Physiological Reactivity to Cold

Measure	Baseline	Cold	<i>F</i>	<i>p</i>
	<i>M (SD)</i>	<i>M (SD)</i>		
Heart Rate	71.68 (10.22)	69.95 (9.64)*	4.14	<.01
RSA	6.44 (1.08)	6.73 (1.10)*	-4.78	<.01
Respiration Rate	16.19 (2.72)	15.71 (2.94)*	2.02	.05
Residualized RSA	-0.02 (0.27)	0.21 (0.44)*	-4.33	<.01
Systolic Blood Pressure	108.92 (9.74)	113.29 (10.80)*	-9.12	<.01
Diastolic Blood Pressure	65.50 (7.08)	70.27 (8.63)*	-9.89	<.01

Note. * Significantly different from baseline. RSA = Respiratory Sinus Arrhythmia

Discussion

Evaluation of Specific Aims

Previous literature has demonstrated that perseverative cognition may result in an increased risk of developing cardiovascular disease. This increased risk may be related to the allostatic load model of stress, with perseverative cognition affecting reactivity and recovery from a stressor. The primary hypothesis of this study was that by manipulating recovery conditions, perseverative cognition would delay recovery, while distraction and cognitive load would enhance recovery. This hypothesis was only partially supported.

Immediately following the stressor, individuals were shown either questions asking about performance on the task, moral dilemma questions, a distracting movie, or nothing. The distracting movie led to a significant reduction in heart rate compared to perseveration and cognitive load, which supports previous findings in which distracting tasks were related to faster recovery (Glynn et al., 2002; Neumann et al., 2004). Of importance to note, both studies compared a cognitive distraction to a group receiving no distraction; a finding potentially related to the fewer number of thoughts reported by the distraction group (Neumann et al., 2004). Unfortunately, the hypothesis that cognitive load would enhance recovery was not supported in the present study; the cognitive load task resulted in smaller recovery to baseline than the distraction group and no difference from the perseverative cognition group. However, this is in line with prior findings, in which individuals actively engaging in cognitive load do not differ in cardiovascular

activity from those engaging in perseverative cognition (Verkuil et al., 2009; Ottaviani et al., 2008).

The group differences found during the first recovery period may be related to amount of cognitive activity, with the distracting movie allowing for a reduction in thought, while the moral dilemmas and the questions about performance increased the total number of thoughts. Cognitive activity may also provide an explanation for why the control group did not differ from any of the other groups. Giving participants nothing to do during recovery could allow individuals to worry about the previous tasks, think about unrelated ideas, or relax as instructed. Unfortunately, self-reported thinking about the stressor was not different between any of the groups. Without a difference in the amount of thought, it is difficult to conclude that cognitive activity caused the differences between the distraction group, and perseverative cognition and cognitive load groups. After the manipulated recovery, all groups engaged in a second common recovery period. One group difference that was not predicted was greater heart rate in the control group compared to the distraction group. Differences in cognitive activity may also explain this effect. While the other groups were engaged in a task, the control group sat quietly for the first four minutes of recovery. During the second recovery period, those individuals may have become bored and agitated, increasing cognitive activity.

Cognitive activity may be important for heart rate during the first and second recovery periods, but another explanation for the results is the higher baseline levels of depression and worry in the control group. Both of these variables were statistically controlled in the analyses, but individuals high on both of these traits and given no instructions could be especially prone to engage in perseverative cognition. During the

first recovery period, the control group could have thought about the previous stressor in a similar manner to the perseverative cognition group. The self-generated perseverative cognition could then be maintained during the second recovery period, leading to the elevated heart rate of the control group. This explanation demonstrates how perseverative cognition can maintain a representation of a stressor and delay cardiovascular recovery following the termination of the original stimulus.

The specific hypothesis made about the second recovery period, that recovery would be related to dispositional worry was not supported by the data. One possible explanation is that that self-reported thinking about the stressor was reduced from the first to the second recovery period. Thus, the stressor was not sufficient to induce a sustained level of perseverative cognition after the first four minute recovery period. Ottaviani et al. (2008) found differences following perseverative cognition and cognitive load tasks after anger recall, but anger may be more potent at maintaining a greater amount of thought than the manipulation of the present study.

Previous Research and Implications

Previous literature indicated that dispositional traits of worry and anxiety could affect the response pattern to a stressor in a manner indicative of allostatic load. In the present study, only motive to avoid failure was predictive of how individuals responded to the task. The motive to avoid failure scale is related to factors of test anxiety and achievement motivation (Hagvet & Benson, 1997). The significant negative relationship between motive to avoid failure and cardiovascular reactivity demonstrates a pattern opposite to the positive relationship between hostility and reactivity during anger recall (Suarez et al., 1993) and greater reactivity to tasks in those with high worry (Delgado et

al., 2009). However, the relationship is in line with the blunted reactivity of individuals with anxiety disorders to a worry induction task (Lyonfields et al., 1995). Additionally, the findings of the present study fit in well with the literature on effort and cardiovascular reactivity. For example, when a difficult task makes avoidance of an aversive stimulus highly likely, systolic blood pressure reactivity is significantly greater compared to reactivity during a similarly difficult task with a low probability of avoiding the stimulus (Wright, Williams, & Dill, 1992). In other words, when the perceived chance of success on a task is low, effort may be withheld, leading to lower cardiovascular reactivity. Due to the relationship with test anxiety, a high motive to avoid failure may result in less effort on the difficult task, leading to smaller reactivity. Effort may explain the relationship between motive to avoid failure and response pattern, but is not supported by the results of the present study. Motive to avoid failure did not predict performance on the stressful task.

The pattern of reduced task-related RSA withdrawal and recovery for individuals with a high motive to avoid failure is similar to the reduced RSA reactivity seen in individuals with anxiety disorders (Lyonfields et al., 1995) and the pattern of reduced fluctuation of RSA and cardiovascular activity seen in individuals with major depressive disorder (Rottenberg et al., 2007; Salomon et al., 2009). This observed pattern of reduced reactivity and delayed recovery may be an important mechanism by which perseverative cognition, through worry and rumination, could lead to the increased risk for cardiovascular disease seen in individuals with depression and anxiety disorders (Wulsin & Singal, 2003; Barger & Sydemann, 2005). Reduced reactivity may be related to withholding effort due to a focus on possible negative outcomes, and then the repeated

mental focus on the event leading to delayed recovery. This may eventually lead to the greater resting levels of cardiovascular activity seen in individuals with anxiety disorders (Thayer et al., 1996). Unfortunately, there were no differences in resting baseline activity for individuals high in worry, anxiety, or depression. Resting differences may not have been seen in the present study because the deleterious effects of allostatic load due to perseverative cognition have not yet affected the relatively young sample.

Dispositional worry, anxiety, and depression were not related to reactivity to the cold pressor task as hypothesized. This may also be due to the sample being a predominately young, health, undergraduate population. Previously observed differences in cold pressor reactivity were in individuals with disease states and neurological disorders (Khurana et al., 1980). The damaging effects of worry, anxiety, and depression may need extended periods of allostatic load to cause significant differences in reactivity to the cold pressor task.

Limitations

The young and healthy sample was one limitation to this study, specifically for examining physiological factors such as resting differences and reaction to the cold pressor task. While the young and healthy sample hindered finding any differences in resting physiology or reaction to the cold pressor task, it is beneficial to the main hypothesis. While previous research has found reactivity differences due to worry (Delgado et al., 2009) and anxiety disorders (Lyonfields et al., 1995), the dispositional traits of worry, anxiety, and depression were not significantly related to reactivity in the present study. The induced difference between recovery of distraction from cognitive

load and perseverative cognition highlights the importance of cognitive load on recovery in a manner independent from reactivity.

A major limitation to the study was reliance on retrospective self-report measures of thought during the recovery conditions. No behavioral measures were collected during the recovery periods to prevent evaluative stress from being added to the recovery conditions. As noted earlier, evaluation can increase cardiovascular responses and would create a confound with the manipulation. It may be possible to give participants disposable scrap paper or some other method of a running tally of thoughts that adds the behavioral measure without additional stress. Similarly, there was no method of separating thoughts related to the stressor and those not related to the stressor. The questions focused on how much the individual engaged in perseverative cognition, meaning thought specifically about the previous stressor. Due to the lack of group differences in self-reported perseveration following the stressor, a question measuring general cognition may have provided more telling information. Finally, a measure of effort during the stressor could be important. While performance on the stressor should reflect effort, this relationship could be reduced if the task is not sufficiently difficult.

There are also strengths to the present study. Directly comparing control, cognition, non-cognitive distraction, and perseveration provided a method of evaluating the importance of cognition in perseverative cognition. The finding that distraction led to enhanced heart rate recovery compared to perseveration and cognition supports previous research that demonstrated anger recall, worry induction, and cognitive load tasks produce comparable levels of physiological activation (Verkuil et al., 2009; Ottaviani et

al., 2008). Perseverative cognition may lead to allostatic load simply because it involves sustained thought; whether these thoughts are related to the stressor may not be critical.

Summary and Conclusions

Cognitive load leads to physiological activation, but the negative affect laden focus of perseverative cognition may uniquely maintain the increased cognition. The definitions of worry (Borkovec et al., 1993) and rumination (Nolen-Hoeksema et al., 2008) characterize the cognitive representations of the stressor as uncontrollable and intrusive. The reduced RSA recovery seen in individuals high in test anxiety supports the idea that a negative perception of the stressor is important for the cognitive representation to remain active. In a similar manner, previous literature has demonstrated delayed recovery in those high in anger in response to harassment (Neumann et al., 2004; Suarez et al., 1993). This influence on recovery may be related to the idea that desire for control has a multiplicative risk to perseverative cognition (Brosschot et al., 2006). Individuals unable to react with anger to harassment maintain greater cardiovascular activity during recovery (Suchday et al., 2004) suggesting that individuals high in anger may desire to respond, but without being given the opportunity instead engage in perseverative cognition. In a similar manner, individuals high in test anxiety may have wished to avoid engaging in the challenging task, resulting in more perseverative cognition leading to less recovery. In both situations, the inability to control a situation adequately may contribute to replaying the stressful event.

The findings of this study suggest that cognitive processes may play an important factor in delaying recovery from stress. The role that perseverative cognition may play is in maintaining the cognitive activity. It is important for future research to examine

potentially important components of perseverative cognition like control and effort.

Dispositional traits may be related to perseverative cognition by affecting perceived control and subsequent effort in a manner that is important to the blunted reactivity and delayed recovery seen in depression (Rottenberg et al., 2007; Salomon et al., 2009).

Understanding these different components will be important in determining how perseverative cognition specifically contributes to cardiovascular disease.

References

- Althaus, M., Mulder, L. J. M., Mulder, G., van Roon, A. M., & Minderaa, R. B. (1998). Influence of respiratory activity on the cardiac response pattern to mental effort. *Psychophysiology*, *35*, 420-430.
- American Heart Association. (2009). Heart disease and stroke statistics 2009 update: A report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. *Circulation*, *119*, e21-e181.
- American Psychiatric Association (1994). *Diagnostic and Statistical Manual of Mental disorders*, 4th ed. Washington, DC: American Psychiatric Press.
- Barger, S. D., & Sydemann, S. J. (2005). Does generalized anxiety disorder predict coronary heart disease risk factors independently of major depressive disorder? *Journal of Affective Disorders*, *88*, 87-91.
- Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology*, *56*(6), 893-897.
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the BDI-II*. San Antonio, TX: Psychological Corporation.
- Berntson, G. G., Bigger, T. J., Eckberg, D. L., Grossman, P., Kaufmann, P. G., Malik, M. et al. (1997). Heart rate variability: Origins, methods, and interpretive caveats. *Psychophysiology*, *34*, 623-648.

- Berntson, G. G., Quigley, K. S., Jang, J., & Boysen, S. T. (1990). An approach to artifact identification: Application to heart period data. *Psychophysiology*, *27*, 586-598.
- Borghetti, C., Costa, F. V., Boschi, S., Mussi, A., & Ambrosioni, E. (1986). Predictors of stable hypertension in young borderline subjects: A five-year follow-up study. *Journal of Cardiovascular Pharmacology*, *8*(S5), S138-S141.
- Borkovec, T. D., Robinson, E., Pruzinsky, T., & DePree, J. A. (1983). Preliminary exploration of worry: Some characteristics and processes. *Behavioral Research Theory*, *21*(1), 9-16.
- Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research*, *60*, 113-124.
- Brosschot, J. F., Godaert, G. L. R., Benschop, R. J., Olf, M., Ballieux, R. E., & Heijnen, C. J. (1998). Experimental stress and immunological reactivity: A closer look at perceived uncontrollability. *Psychosomatic Medicine*, *60*, 359-361.
- Brosschot, J. F., Pieper, S., & Thayer, J. F. (2005). Expanding stress theory: Prolonged activation and perseverative cognition. *Psychoneuroendocrinology*, *30*, 1043-1049.
- Brosschot, J. F., & van der Doef, M. (2006). Daily worrying and somatic health complaints: Testing the effectiveness of a simple worry reduction intervention. *Psychology & Health*, *21*(1), 19-31.
- Brosschot, J. F., van Dijk, E., & Thayer, J. F. (2007). Daily worry is related to low heart rate variability during waking and the subsequent nocturnal sleep period. *International Journal of Psychophysiology*, *63*, 39-47.

- Buysee, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index (PSQI): A new instrument for psychiatric research and practice. *Psychiatry Research*, 28, 193-213.
- Contrada, R. J., Wright, R. A., & Glass, D. C. (1984). Task difficulty, Type A behavior pattern, and cardiovascular response. *Psychophysiology*, 21(6), 638-646.
- Cook, W. W., & Medley, D. M. (1954). Proposed hostility and pharisaic virtue scales for the MMPI. *Journal of Applied Psychology*, 38, 414-418.
- Delgado, L. C., Guerra, P., Perakakis, P., Mata, J. L., Perez, M. N., & Vila, J. (2009). Psychophysiological correlates of chronic worry: Cued versus non-cued fear reaction. *International Journal of Psychophysiology*, 74, 280-287.
- Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, 130(3), 355-391.
- Ernst, J. M., Litvack, D. A., Lozano, D. L., Cacioppo, J. T., & Berntson, G. G. (1999). Impedance pneumography: Noise as signal in impedance cardiography. *Psychophysiology*, 36, 333-338.
- Felsten, G. (1996). Five-factor analysis of Buss-Durkee Hostility Inventory neurotic hostility and expressive hostility factors: Implications for health psychology. *Journal of Personality Assessment*, 67(1), 179-194.
- Gerin, W., Davidson, K. W., Christenfeld, N. J., Goyal, T., & Schwartz, J. E. (2006). The role of angry rumination and distraction in blood pressure recovery from emotional arousal. *Psychosomatic Medicine*, 68, 64-72.

- Glynn, L. M., Christenfeld, N., & Gerin, W. (2002). The role of rumination in recovery from reactivity: Cardiovascular consequences of emotional states. *Psychosomatic Medicine, 64*, 714-726.
- Glynn, L. M., Christenfeld, N., & Gerin, W. (2007). Recreating cardiovascular responses with rumination: The effects of a delay between harassment and its recall. *International Journal of Psychophysiology, 66*, 135-140.
- Goodwin, R. D., Davidson, K. W., & Keyes, K. (2009). Mental disorders and cardiovascular disease among adults in the United States. *Journal of Psychiatric Research, 43*, 239-246.
- Gramer, M., & Saria, K. (2007). Effects of social anxiety and evaluative threat on cardiovascular responses to active performance situations. *Biological Psychology, 74*, 67-74.
- Greene, J.D., Sommerville, R.B., Nystrom, L.E., Darley, J.M., & Cohen, J.D. (2001). An fMRI investigation of emotional engagement in moral judgment. *Science, 293*, 2105-2108.
- Hagtvet, K. A., & Benson, J. (1997). The motive to avoid failure and test anxiety responses: Empirical support for integration of two research traditions. *Anxiety, Stress, and Coping, 10*, 35-57.
- Hall, M., Baum, A., Buysse, D. J., Prigerson, H. G., Kupfer, D., J., & Reynolds, C. F. (1998). Sleep as a mediator of the stress-immune relationship. *Psychosomatic Medicine, 60*(1), 48-51.

- Hall, M., Vasko, R., Buysse, D., Ombao, H., Chen, Q., Cashmere, J. D. et al. (2004). Acute stress affects heart rate variability during sleep. *Psychosomatic Medicine*, 66, 56-62.
- Hodapp, V., Heiligtag, U., & Stö rmer, S. W. (1990). Cardiovascular reactivity, anxiety and anger during perceived controllability. *Biological Psychology*, 30, 161-170.
- Ituarte, P. H., Kamarck, T. W., Thompson, H. S., & Bacanu, S. (1999). Psychosocial mediators of racial differences in nighttime blood pressure dipping among normotensive adults. *Health Psychology*, 18, 393-402.
- Jennings, J. R., Kamarck, T., Stewart, C., Eddy, M., & Johnson, O. (1992). Alternate cardiovascular baseline assessment techniques: Vanilla or resting baseline. *Psychophysiology*, 29, 742-750.
- Kario, K., Schwartz, J. E., Gerin, W., Robayo, N., Maceo, E., & Pickering, T. G. (2002). Psychological and physical stress-induced cardiovascular reactivity and diurnal blood pressure variation in women with different work shifts. *Hypertension Research*, 25(4), 543-551.
- Khurana, R. K., Watabiki, S., Hebel, J. R., Toro, R., & Nelson, E. (1980). Cold face test in the assessment of trigeminal-brainstem-vagal function in humans. *Annals of Neurology*, 7, 144-149.
- Knepp, M. M., & Friedman, B. H. (2008). Cardiovascular activity during laboratory tasks in women with high and low worry. *Biological Psychology*, 79, 287-293.
- Krantz, D. S., & Manuck, S. B. (1984). Acute psychophysiologic reactivity and risk of cardiovascular disease: A review and methodologic critique. *Psychological Bulletin*, 96(3), 435-464.

- Krantz, D. S., & McCeney, M. K. (2002). Effects of psychological and social factors on organic disease: A critical assessment of research on coronary heart disease. *Annual Review of Psychology, 53*, 341-369.
- Kubzansky, L. D., Kawachi, I., Spiro, A., Weiss, S. T., Vokonas, P. S., & Sparrow, D. (1997). Is worrying bad for your heart? A prospective study of worry and coronary heart disease in the Normative Aging Study. *Circulation, 95*, 818-824.
- Laumann, K., Gärling, T., Stormark, K. M. (2003). Selective attention and heart rate responses to natural and urban environments. *Journal of Environmental Psychology, 23*, 125-134.
- Linden, W., Earle, T. L., Gerin, W., & Christenfeld, N. (1997). Physiological stress reactivity and recovery: Conceptual siblings separated at birth. *Journal of Psychosomatic Research, 42*(2), 117-135.
- Lynch, J. J., Long, J. M., Thomas, S. A., Malinow, K. L., & Katcher, A. H. (1981). The effects of talking on the blood pressure of hypertensive and normotensive individuals. *Psychosomatic Medicine, 43*(1), 25-33.
- Lyonfields, J. D., Borkovec, T. D., & Thayer, J. F. (1995). Vagal tone in Generalized Anxiety Disorder and the effects of aversive imagery and worrisome thinking. *Behavior Therapy, 26*, 457-466.
- McEwen, B. S. (1998). Stress, adaptation, and disease: Allostasis and Allostatic Load. *Annals New York Academy of Sciences, 33-44*.
- McEwen, B. S., & Seeman, T. (1999). Protective and damaging effects of mediators of stress: Elaborating and testing the concepts of allostasis and allostatic load. *Annals New York Academy of Sciences, 1999, 30-47*.

- Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy*, 28, 487-495.
- Neumann, S. A., Waldstein, S. R., Sollers, J. J., III, Thayer, J. F., & Sorkin, J. D. (2004). Hostility and distraction have differential influences on cardiovascular recovery from anger recall in women. *Health Psychology*, 23(6), 631-640.
- Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology*, 100, 569-582.
- Nolen-Hoeksema, S., Wisco, B. E., & Lyubomirsky, S. (2008). Rethinking Rumination. *Perspectives on Psychological Science*, 3(5), 400-424.
- Ottaviani, C., Shapiro, D., Goldstein, I. B., James, J. E. & Weiss, R. (2006). Hemodynamic profile, compensation deficit, and ambulatory blood pressure. *Psychophysiology*, 43, 46-56.
- Palatini, P., & Julius, S. (1997). Heart rate and the cardiovascular risk. *Journal of Hypertension*, 15, 3-17.
- Peters, M. L., Godaert, G. L. R., Ballieux, R. E., van Vliet, M., Willemsen, J. J., Sweep, F. C. G. J. et al. (1998). Cardiovascular and endocrine responses to experimental stress: Effects of mental effort and controllability. *Psychoneuroendocrinology*, 23(1), 1-17.
- Porges, S. W. (2007). The polyvagal perspective. *Biological Psychology*, 74(2), 116-143.
- Rottenberg, J., Clift, A., Bolden, S., & Salomon, K. (2007). RSA fluctuation in major depressive disorder. *Psychophysiology*, 44, 450-458.

- Shapiro, D., Jamner, L. D., Lane, J. D., Light, K. C., Myrtek, M., Sawada, Y. et al. (1996). Blood pressure publication guidelines. *Psychophysiology*, *33*, 1-12.
- Singh, J. P., Larson, M. G., Tsuji, H., Evans, J. C., O'Donnell, C. J., & Levy, D. (1998). Reduced heart rate variability and new-onset hypertension - Insights into pathogenesis of hypertension: The Framingham Heart Study. *Hypertension*, *32*(2), 293-297.
- Sloan, R. P., Korten, J. B., & Myers, M. M. (1991). Components of heart rate reactivity during mental arithmetic with and without speaking. *Physiology & Behavior*, *50*, 1039-1045.
- Smith, T. W., Nealey, J. B., Kircher, J. C., & Limon, J. P. (1997). Social determinants of cardiovascular reactivity: Effects of incentive to exert influence and evaluative threat. *Psychophysiology*, *34*, 65-73.
- Smith, T. W., & O'Keeffe, J. L. (1988). Cross-situational consistency of cardiovascular reactivity. *Biological Psychology*, *27*, 237-243.
- Suarez, E. C., Harlan, E., Peoples, M. C., & Williams, R. B., Jr. (1993). Cardiovascular and emotional responses in women: The role of hostility and harassment. *Health Psychology*, *12*(6), 459-468.
- Suchday, S., Carter, M. M., Ewart, C. K., Larkin, K. T., & Desiderato, O. (2004). Anger cognitions and cardiovascular recovery following provocation. *Journal of Behavioral Medicine*, *27*(4), 319-341.
- Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. (1996). Heart rate variability: Standards of

- measurement, physiological interpretation, and clinical use. *European Heart Journal*, 17, 354-381.
- Thayer, J. F., Friedman, B. H., & Borkovec, T. D. (1996). Autonomic characteristics of Generalized Anxiety Disorder and worry. *Biological Psychiatry*, 39, 255-266.
- Tomaka, J., Blascovich, J., & Swart, L. (1994). Effects of vocalization on cardiovascular and electrodermal responses during mental arithmetic. *International Journal of Psychophysiology*, 18, 23-33.
- Tsuji, H., Larson, M. G., Venditti, F. J., Manders, E. S., Evans, J. C., Feldman, C. L. et al. (1996). Impact of reduced heart rate variability on risk for cardiac events: The Framingham Heart Study. *Circulation*, 94(11), 2850-2855.
- Verkuil, B., Brosschot, J. F., Borkovec, T. D., & Thayer, J. F. (2009). Acute autonomic effects of experimental worry and cognitive problem solving: Why worry about worry? *International Journal of Clinical and Health Psychology*, 9(3).
- Vrijkotte, T. G. M., van Doornen, L. J. P., de Geus, E. J. C. (2000). Effects of work stress on ambulatory blood pressure, heart rate, and heart rate variability. *Hypertension*, 35(4), 880-886.
- Wientjes, C. J. E. (1992). Respiration in psychophysiology: Methods and applications. *Biological Psychology*, 34, 179-203.
- Woodall, K. L., & Matthews, K. A. (1989). Familial environment associated with Type A behaviors and psychophysiological responses to stress in children. *Health Psychology*, 8(4), 403-426.

Wright, R. A., Williams, B. J., & Dill, J. C. (1992). Interactive effects of difficulty and instrumentality of avoidant behavior on cardiovascular reactivity.

Psychophysiology, 29(6), 677-686.

Wulsin, L. R., & Singal, B. M. (2003). Do depressive symptoms increase the risk for the onset of coronary disease? A systematic quantitative review. *Psychosomatic*

Medicine, 65(2), 201-210.