STRESS AND SLEEP: PREDICTORS OF FAILURE TO RECOVER

by

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Abstract

Delayed recovery after cardiovascular response to a stressor is currently being recognized as a marker and likely contributing factor in the development of cardiovascular disease. Interestingly, the psychological variables that predict delayed recovery appear similar to those associated with poor sleep quality. As such, poor sleep may be another index of delayed recovery. This study attempted to expound the relationship between psychological predictors of recovery and sleep and determine whether these outcomes do indeed share common predictors. Sleep quality is defined as total sleep time determined by actigraph measurements. One hundred and thirty six participants were subjected to a mental stress task coupled with harassment after which speed of recovery was assessed. In these same individuals, sleep quality data for the night following the lab stress procedure were collected. Results were not in support of our overall hypothesis. Slower rates of recovery were associated with caffeine consumption prior to experiment time, as well as anger rumination, however, with identifiable gender effects, while total sleep time was predicted by hostility in the overall sample and by higher rates of worry in male participants.
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Introduction

Psychological stress and the resulting physiological activation are now recognized as a risk factor in a myriad of diseases. In particular, the role of psychological stress in cardiovascular disease development has received a great deal of attention (for review, see Schwartz et al., 2003). Cardiovascular diseases remain the leading cause of death in Canada, accounting for approximately 32% of all deaths (Statistics Canada, 2007). Less than 50% of the variance of new cases of cardiovascular disease can be explained by classic risk factors such as obesity, smoking and family history (Roig, Castaner, Simmons, Patel, Ford, & Cooper, 1987). In light of this, researchers have turned their attention to the role of stress in the development of cardiovascular disease. Prospective studies have indicated that chronic cardiovascular activation—physiological responses to stress including elevated blood pressure (BP) and heart rate (HR)—plays a role in the development of risk factors for cardiovascular disease including hypertension (Treiber, Musante, Kapuku, Davis, Litaker, & Davis, 2001). Further, it is now being recognized that prolonged activation is pivotal in creating a pathogenic state (Brosschot, Pieper & Thayer, 2005). This study explores a critical component of prolonged activation, namely slowed or delayed recovery after a stressor, as evinced by recovery after a laboratory stress task, and by sleep analysis—with the view that poor sleep is an indicator of delayed recovery. In addition, we seek to describe the psychological variables associated with slow recovery.

Reactivity vs. Prolonged Activation

Studies that describe psychological stress as a risk factor for disease have been based primarily on the reactivity hypothesis, where frequent and exaggerated stress responses of short duration are thought to play the primary pathogenic role in disease development (Brosschot et al., 2005). Reactivity refers to the magnitude of elevation in an individual’s
physiological response (for example, increased BP, HR and elevated levels of the stress hormone cortisol) in response to an aversive, challenging, or engaging stressor (Linden, Gerin, & Davidson, 2003; Schwartz et al., 2003). Over time, chronic stress induced elevations in HR, and especially BP, are thought to create lasting detrimental changes in cardiovascular functioning, including elevation of the tonic BP level, leading to development of cardiovascular disease (Schwartz et al., 2003). Reactivity is a relatively stable individual trait characteristic, and reactivity testing in the laboratory can achieve acceptable levels of test-retest reliability (surpassing the .80 mark) when values are aggregated across lab tasks (Kamarck, & Lovallo, 2003). Individuals high in BP reactivity during a cold pressor stress task were more likely to be hypertensive at 45-year follow-up (Wood, Sheps, Elveback & Schirger, 1984) and at 20- and 36-year follow-up (Menkes et al., 1989). However, this research has been criticized for lacking generalizability in that elevated BP and HR levels in laboratory stress tasks fail to map onto responses to real world challenges, and hence, fail to account for interaction effects of environment exposure and individual difference factors. As well, reactivity research ignores the duration of physiological activation, which is seen as the primary pathogenic pathway to cardiovascular disease development (Linden, Earle, Gerin, & Christenfeld, 1997; Schwartz et al., 2003).

Underrepresented in the accumulated literature on stress and cardiovascular response is the concept of recovery, whereby the cardiovascular system normalizes after activation. Recovery research was largely ignored due to the lack of a theoretical model describing prolonged activation (Linden et al., 1997; Brosschot et al., 2005). Ursin and Murison (1983) introduced the concept of sustained activity in the 1980s, but it was not until the 1990s that the importance of prolonged activation in disease development began to be recognized (Brosscho: et al., 2005). One component of prolonged activation, delayed recovery, accounts
for the duration of the response and is therefore in keeping with current stress models (e.g., allostatic load models, see Linden et al., 1997; McEwen, 1998) that suggest overall physiological activation over time is the primary pathogenic pathway to disease development (Brosschot et al., 2005). The allostatic load model maintains that the stress response is generally adaptive, as it alerts the body to recognize and cope with challenges within the environment; however, this same adaptive response can become extremely pathogenic if the stress response is frequent and prolonged (McEwen, 1998). In order to respond to challenges from environmental stimuli an individual must first interpret a stimulus as a threat, which in turn causes physiological activation of stress response systems, such as the autonomic nervous system (ANS) and hypothalamic-pituitary-adrenal axis (HPA). Physiological activation of these systems releases catecholamine from the nerves and adrenal medulla, stimulating the release of corticotrophin from the pituitary, which acts to mediate the release of cortisol from the adrenal cortex (McEwen, 1998). Physiological activation also mobilizes energy stores, increases cardiovascular/pulmonary tone to facilitate oxygen delivery to tissue, and reduces digestion, inflammatory responses, and immune system functioning (Van Reeth, Weibel, Spiegel, Leproult, Dugovic, & Maccari, 2000). Following activation, negative feedback systems return the body to baseline states. If negative feedback mechanisms are successful, the stress response is adaptive in alerting an individual to a challenge or threat in the environment, thus priming the body to physically deal with the stressor. Once the stressor is terminated, so too is the stress response. However, stress response systems that are over- or under-active create pathogenic wear and tear on the body. McEwen (1998) describes three types of physiological activation that make up allostatic load: frequent stress, failed shut down (chronic activity and failure to shut off the stress response, [i.e., delayed
recovery]) and inadequate response (failure to respond to a challenging stressor, causing other systems to become active).

A comprehensive meta-analysis by Shuler and O’Brien (1997) has shown that delayed BP recovery after a stress response is associated with hypertension. As well, longitudinal studies show that borderline hypertension is predicted by delayed recovery when assessed after a laboratory stress task (Stewart & France, 2000), and familial hypertension was predicted by slow recovery following a submaximal exercise task when assessed 10-years later (Tanji, Champlin, Wong, Lew, Brown, & Amsterdam, 1989). Further, delayed HR recovery at initial assessment predicted overall mortality 5- (Nishime, Cole, Blackstone, Pashkow, & Lauer, 2000) and 6-years later in cardiac patients (Cole, Blackstone, Pashkow, Snader, & Lauer, 1999) when recovery was assessed after a symptom-limited exercise (i.e., treadmill exercise). Increased attention to recovery has uncovered pathways to disease development above and beyond the effects of cardiovascular reactivity, however, there is still much to be explored in this domain.

**Psychological Variables and Recovery**

While there have been gains in explicating the relationship between recovery and cardiovascular disease, laboratory studies assessing recovery after activation of the cardiovascular stress response have been critiqued as lacking ecological validity due to the use of short term, primarily physical stressors, which may not map onto real world stressors and subsequent cardiovascular functioning (Schwartz et al., 2003). However, interpersonal tasks, particularly those evoking anger and hostility, may overcome this weakness (Linden, Rutledge, & Con, 1998). Such tasks tend to be more representative of chronic life stressors implicated in cardiovascular disease development (i.e., job strain, marital stress) than the other commonly used cognitive and physical tasks. Interpersonal tasks have been shown to
yield elevated reactivity patterns as well as delayed recovery patterns in the cardiovascular and endocrine stress response systems (Linden et al., 1998), likely due to their propensity to elicit rumination. Glynn, Christenfeld and Gerin, (2002) postulated that only emotion-based tasks, namely interpersonal tasks that elicit negative affect such as anger (e.g., harassment during an arithmetic task) were associated with delayed BP recovery (independent of initial reactivity) and likely attributable to perseverative rumination or mental rehearsal.

These interpersonal, emotion-based tasks have implicated trait anger/hostility (Anderson, Linden & Habra, 2005), anger response styles (Lai & Linden, 1992), and trait-rumination (Glynn et al., 2002) as important determinants of cardiovascular recovery. Anderson et al. (2005) found that during an arithmetic task coupled with harassment, individuals low in hostility exhibited greater reactivity but faster recovery (which the authors suggest is an adaptive response), while those high in trait hostility showed less reactivity but slower recovery. This study serves to reaffirm the conceptualization of reactivity and recovery as independent areas of analysis, as it did not support the hypothesis that individuals with greater reactivity would also show slower recovery rates (i.e., both markers of a maladaptive stress response).

Anger expression style has also been associated with differential recovery, with identifiable gender effects. Faber & Burns (1995) found that frequent anger expression in men and infrequent anger expression in women was linked to slow recovery rates. Conversely, Lai and Linden (1992) found faster recovery rates in men given the opportunity to express anger and in women with anger-in tendencies. Given these discrepant findings Brosschot and Thayer (1998) posit that social reality forces more anger inhibition than anger expression and is associated with slower recovery rates, and is therefore the style associated
with cardiovascular disease development. The association between anger expression style and recovery, however, needs further explication.

Rumination has also been identified in the maintenance of a stress response. Individuals prone to rumination may not only have immediate slower recovery times making them more susceptible to pathogenesis, but rumination about the stressful experience could lead to physiological reactivation increasing the chronicity of the stress response (Glynn et al., 2002). Gerin, Davidson, Christenfeld, Goyal and Schwartz (2006) found that individuals high in anger rumination showed the most elevated BP levels when distraction was not available.

While psychological variables can hinder recovery, certain individual factors can serve to accelerate recovery. Faster recovery is evident in individuals who are physically fit (Blumenthal et al., 1988; Cox, Evans, & Jamieson, 1979) and gender-specific analyses have indicated that women recover faster than men (Jackson, Treiber, Turner, Davis, & Strong, 1999).

While these findings identify the association between various psychological and demographic variables and recovery, further research is needed to describe this association using ecologically valid empirical studies, namely, by assessing recovery in the natural environment, as opposed to utilizing laboratory stress tasks that fail to capture the interaction between individual and environmental variables.

Sleep and Stress Recovery

Recognizing the limits of ecological validity of acute lab stressors, researchers have begun to look for other ways to study non-recovery phenomena. The psychological variables that appear to determine delayed recovery from acute stressors in the lab overlap with those predicting poor sleep quality, including increases in worry and rumination (Hall, Buysse,
Dew, Prigerson, Kupfer, & Reynolds, 1997). Therefore, it makes sense to direct attention to quality of sleep as another index of non-recovery.

Sleep is posited to be the main restorative period that has evolved, not only to help protect animals from harm in periods of darkness, but to allow the body time to heal and consolidate information at the level of the central nervous system (Zeman & Reading, 2005). It is also a time of reduced cardiovascular load as slow-wave or non-REM sleep (NREM) is associated with reduced BP and HR and increased parasympathetic activity. REM sleep is associated with increases in BP and HR and sympathetic activity (Parish & Shepard, 1990). In humans REM sleep accounts for a very small amount of total sleep time, with more REM sleep experienced after sleep deprivation (termed rebound sleep; Beersma, Dijk, Blok & Everhardus, 1990). Alterations in sleep patterns or duration may therefore increase cardiovascular load, which, if chronic, could lead to detrimental cardiovascular alterations, paving the way for disease development. Indeed, sleep duration has been reliably linked to morbidity and mortality in a series of epidemiological studies (Dew et al., 2003; Schwartz, Cornoni-Huntley, Cole, Hays, Blazer, & Schocken, 1998) and insomnia has been linked with cardiovascular disease (see Bonnet & Arand, 2007; Schwartz, Anderson, Cole, Cornoni-Huntley, Hays, & Blazer, 1999).

Stress has been found to significantly impact sleep quality and nocturnal cardiovascular functioning in humans. Indirect evidence for the detrimental impact of stress on sleep has come from studies showing sleep disturbance following a major life stressor such as death or divorce (for review see Van Reeth et al., 2000), while direct evidence linking stress and sleep comes from studies showing changes in sympathetic activation involving HPA axis hormones. The HPA axis involved in the stress response is also implicated in the sleep cycle. Cortisol and corticotrophin levels are at a minimum within the
first period of the sleep cycle while there is a corresponding increase in growth hormone secretion. HPA hormones increase with lighter sleep becoming maximal shortly after sleep wakening (Van Reeth et al., 2000). High levels of corticotrophin and cortisol, found with an increase in daily stress, have been associated with greater sleep latency, disturbed sleep, and enhanced vigilance (Steiger, 2002).

Acute stress has also been associated with nocturnal decreases in parasympathetic activation and low heart rate variability (HRV), along with increased HR, indicating stress-induced alterations in cardiovascular functioning (Hall et al., 2004). Low HRV and high HR are risk factors for cardiovascular disease (Brosschot et al., 2007). Brosschot and colleagues (2007) also found an association between daily stressors and low nocturnal HRV and increased HR. Importantly the authors found that the link between daily stressors and nocturnal cardiovascular activity was mediated by worry duration, while trait anxiety was only predictive of daytime HRV and HR, indicating that worry may play a central role in the relationship between stress, recovery and cardiovascular disease.

The literature on nocturnal non-dipping of BP has also implicated psychological constructs in the alterations in cardiovascular functioning at night, namely, anger and hostility (Thomas, Nelesen, & Dimsdale, 2004), which are also associated with delayed recovery from laboratory stress. Individuals referred to as “non-dippers” do not show typical patterns of BP reduction (i.e., 10% reduction at night versus daytime levels) during sleep periods. Non-dipping is associated with increased cardiovascular complications and mortality (O'Brien et al., 1988; Ohkubo et al., 2002).

Other psychological variables that have been shown to predict recovery from interpersonal lab stressors have also been shown to predict sleep quality. For example, rumination was found to be predictive of poorer sleep quality, over and above depression,
anxiety and angry mood (Thomsen, Mehl sen, Christensen, & Zachariae, 2003). Further, experimentally induced pre-sleep rumination predicted poorer sleep quality when compared to a distracter condition (Guastella & Moulds, 2007). Trait anger has also been found to be predictive of sleep quality in middle aged men and women (Shin, Kim, Yi, Lee, Lee & Shin, 2005) and hostility was predictive of reduced hours of sleep in juveniles and young offenders (Ireland & Culpin, 2006).

While there is some evidence for psychological variables predicting sleep quality this literature is still very limited. Most available studies use only subjective sleep data (e.g., Pittsburg Sleep Quality Index) rather than objective data that may more accurately capture changes in sleep architecture. Further, most studies examine very few psychological predictors in the same study, thereby limiting the knowledge of what variables predict sleep over and above other related variables. Therefore, I deem it necessary to elucidate the relationships between these variables both with respect to recovery during a lab stress task and to sleep quality. Given that these variables are similar to those predicting recovery from lab stressors, coupled with evidence of definitive cardiovascular alterations during poor sleep, it is promising to conceptualize poor sleep as a failure to recover and it is in this vein that this study is conducted.

Objectives

Given that cardiovascular disease development is multi-factorial in nature, it is important to identify risk factors early on. Identification of such factors can aid in the design and implementation of interventions and health promotion campaigns to increase public knowledge regarding preventative measures. As such, it is necessary to uncover the possible pathogenic connection between stress and sleep, with the view that poor sleep is an index of delayed recovery from stress. Additional research is needed to explicate the connection
between the psychological markers of delayed recovery from acute stressors and sleep quality. Our primary objectives of the study are to replicate earlier findings that rumination following stressor termination slows physiological recovery from acute stress, to learn about the association between rumination, hostility/anger, worry, stress and poor sleep, and to test whether the same individuals who show slow recovery from acute stress also have poorer quality of sleep, thereby spanning three related domains of research that have traditionally been studied independently (see Figure 1). Specifically, the following research objectives were assessed:

Do psychological variables, in particular trait anger, hostility and rumination, predict sleep over and above other related variables?

Do these same variables predict delayed recovery from a laboratory stress task?

Does speed of recovery from a laboratory stress task predict sleep quality?

This research is timely, as only one study has viewed sleep as an index of non-recovery to date. Furthermore, there is a paucity of research using psychological variables to predict sleep quality. As such, I hope to add to the literature on factors predicting sleep and on delayed recovery as part of a pathophysiological model.

Methods

Participants

The 136 participants (98 female, 38 male; age range, 18 – 43 years; mean age 21.99, \(SD = 4.89\); 31% Caucasian, 56% Asian, 13% other) were recruited from the University of British Columbia via the UBC Psychology Subject Pool and advertisements posted around campus and in the community. Subjects received course credit or an honorarium in exchange for their participation. Inclusion criteria for this study included being over the age of 18 years and possessing a working knowledge of the English language. Exclusion criteria
included the use of antihypertensive medication, a current blood pressure level exceeding
160/100 mmHg, long-term use of prescription or non-prescription sleep medication and the
frequent use of anxiolytics. Twenty-eight additional individuals were excluded from the final
analysis for the following reasons: fifteen did not complete the lab session, eight did not meet
inclusion/exclusion criteria, and five were excluded due to experimental error.

Measures

Demographic Information

Participants were required to complete a demographic questionnaire indicating their
race, age, gender, chronic illness profile, and any sleep disorders they may have, including
insomnia or sleep apnea as diagnosed by a physician.

Participants also completed questionnaires, chosen for their psychometric properties,
to obtain information on psychological variables that may predict sleep quality. These
questionnaires were uploaded onto a hand-held PC device to increase the reliability of data
acquisition and scoring, as all data were downloaded directly into a software package for
auto-scoring, avoiding scoring and data entry errors.

Worry

Participants completed the Penn State Worry Questionnaire (PSWQ; Meyer, Miller,
Metzger, & Borkovec, 1990) to provide an index of worry. The 16-item self-report
questionnaire is the most often employed measure of worry, used to assess generality,
excessiveness, and uncontrollability dimensions of pathological worry (Fresco, Douglas,
Heimberg, & Turk, 2003). Responses are made on a 5-point scale, ranging from “not at all
typical of me” to “very typical of me”, with higher total scores indicating increased levels of
pathological worry. Example items include, “When I am under pressure, I worry a lot”, and
"I am always worrying about something"; five of the 16 items are reverse scored in order to reduce possible acquiescence (i.e., "I never worry about anything") (Meyer et al., 1990). The PSWQ has good test-retest reliability over intervals as long as 8-10 weeks (r=.92), and high internal consistency (α=.94). Further, validity has been established by showing that the PSWQ can detect diagnosable levels of generalized anxiety disorder (GAD; characterized by excessive worry) in college students and distinguish between GAD and posttraumatic stress disorder in clinical samples (Meyer et al., 1990).

Anger Response Styles

The Behavioral Anger Response Questionnaire (BARQ; Linden, Hogan, Rutledge, Chawla, Lenz, & Leung, 2003) is a 37-item questionnaire consisting of six factor-analytically derived subscales, namely: Assertion (6 items), Direct Anger-Out (7 items), Social Support-Seeking (6 items), Rumination (6 items), Avoidance (6 items), and Diffusion (6 items). The BARQ was developed to capture a broad range of coping mechanisms involved in the anger experience, rather than just anger-in/anger-out models captured by traditional measures such as the State-Trait Anger Expression Inventory (STAXI; Spielberger, 1988). Within both hypertensive and normal samples, the BARQ has been shown to predict blood pressure (Hogan & Linden, 2004), thus indicating its usefulness in assessing psychological factors in cardio-pathology. Example items from each of these six subscales include. “I give the person I am angry with a piece of my mind (that is, I tell him/her very directly how I feel)” (Direct-Anger Out); “I express my feelings by playing music, writing a poem, or painting” (Diffusion); “In a calm voice, I tell the angering person how I honestly feel” (Assertion); “I leave the situation. Some time later I call a friend or family member to share my feelings” (Social Support-Seeking); “I just wait to feel better” (Avoidance); and, “In my mind, I replay the frustrating event several times over” (Rumination). Participants are to indicate how often
they behave in such a manner on a 5-point scale, ranging from 1 (rarely) to 5 (very frequently). Thus, higher scores indicate more behavior endorsement on each of the subscales. The BARQ has shown good psychometric properties with a mean internal consistency $\alpha$ of .75 across the six subscales, ranging from .65 (Diffusion) to .85 (Direct-Anger Out), and 1-month test-retest reliability for the subscales ranging from .51 (Assertion) to .85 (Direct-anger out). The 6-factor structure of the scale was supported using factor analysis, and comparisons with subscales of the NEO Five-Factor Model of Personality (FFM; Costa & McCrae, 1985) provided evidence for construct validity (Linden et al., 2003). Further, the BARQ displayed incremental validity above and beyond the STAXI-2 in predicting emotional and cognitive responses to anger provocation (Martin & Dahlen, 2007).

For the purpose of our study only three of the BARQ subscales were utilized in analyses, namely, diffusion, rumination and direct anger out, as they have either shown to predict cardiovascular parameters in previous research (Linden, et al., 2008, Hogan & Linden, 2003) or tap into constructs shown to predict physiological reactivity or recovery. For example, direct anger out corresponds to the Spielberger Anger Expression Scale (SAES; Spielberger, Johnson, Russell, & Crane, 1985) anger-out construct ($r = .72$), and anger rumination most closely corresponds most with an anger-in tendency ($r = .49$; Linden et al., 2003).

Hostility/Aggression

Participants also completed the Buss-Perry Aggression Questionnaire (AQ; Buss & Perry, 1992). The AQ is a 29 item scale with four subscales, two of which assess different outward manifestations of aggression, namely the physical and verbal aggression subscales (items 1-15) and two measuring affective and cognitive domains of aggression—the anger and hostility scales (items 15-29), respectively. Summing of the subscales gives a global
score of aggression. Items include statements such as, “I have threatened people I know” (Physical Aggression), “I sometimes feel like a powder keg ready to explode” (Anger), “I often find myself disagreeing with people” (Verbal Aggression), and “Other people always seem to get the breaks” (Hostility). Participants are to rate themselves on a 1-7 scale from "extremely uncharacteristic of me" to "extremely characteristic of me". The AQ has shown good internal consistency for all subscales (α=.72 to .89) as well as for the global aggression score (α=.89). Test-retest reliability over a 9-week period ranged from r=.72 to .80. Further, confirmatory factor analysis supported the factor structure of the scale (Buss & Perry, 1992). The factor structure was also supported using a Canadian sample (Harris, 1995).

The anger and hostility scales were utilized in subsequent analyses as they are theoretically related to the dependent variables.

Sleep Measures

Subjective and objective measures of sleep quality were also obtained. The Pittsburg Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) assesses participants’ retrospective recall of sleep patterns during the last month (i.e., month prior to study commencement). The PSQI is a 19-item self-report measure that assesses overall sleep quality and dysfunction over a 1-month period. Participants report, for example, on the time of day they wake up and sleep, how long it takes to fall asleep (i.e., sleep latency), the number of hours of sleep per night, and the number of times in the past month they had trouble sleeping for reasons such as ‘being too hot’, or ‘having trouble breathing’. The 19 items compile to create seven composite scores, weighted on a 0-3 scale. These composite scores include sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medications, and daytime dysfunction. Summing of the composite scores provides a global score, which ranges from 0-21, with higher scores
indicating poorer sleep quality. The PSQI has demonstrated good internal consistency ($\alpha=.83$). Further, the PSQI showed good test-retest reliability of $r=.85$ for the global score and a range from .65 (medication use) to .84 (sleep latency) for each of the seven composite scores (Buysse et al., 1989). Internal consistency was $\alpha=.80$ for the global score (range, $\alpha=.70-.78$). PSQI global scores were highly correlated with single or multi-item measures of sleep quality, indicating convergent validity, but did not correlate with unrelated constructs, indicating adequate discriminant validity (Buysse et al., 1989).

Actigraph monitors, worn on the wrist, provided an objective index of sleep quality by measuring sleep/wake times indicated by motor activity. The monitor includes an accelerometer that provides data on sleep architecture, i.e., sleep length and depth, motor activity during sleep, as well as pinpointing sleep onset latency, and wake times, (Mini-Mitter, Respironics, Bend, Oregon; Canadian supplier, Biolynx/Montreal), by translating motor activity into numeric representation. Movement is sampled frequently and is then aggregated to provide a 1-minute recording, which is then stored in the devices’ internal memory until the data are downloaded for use (Sadeh, Hauri, Kripke, & Lavie, 1995). Actigraph measurements when compared with “gold standard” Polysomnography (PSG) show minute-by-minute sleep/wake scoring agreement rates of 75-95% for normal subjects. Further, they show correlations of .83 to .97 for measures of sleep/wake times, sleep efficiency and total sleep time (for review see, Sadeh et al., 1995). The “Gold standard” PSG is unfortunately an expensive and cumbersome measurement process that must be employed in a laboratory, and involves the attachment of electrodes to an individual while sleeping in order to monitor brain activity (EEG), eye movement (EOG), muscle activity (EMG), and breathing and respiratory rates (Kushida, Chang, Gakery, Guilleminault, Carrillo, & Dement, 2001). In contrast, the actigraph is inexpensive, non-cumbersome, non-invasive and
can be employed in the home, and is therefore more ecologically valid. Nevertheless, studies have found that actigraphy can overestimate sleep duration due to lengthy periods of inactivity (as when an individual may lay motionless in bed for a period of time before actually falling asleep), or underestimate sleep duration for those who sleep restlessly. Consequently, it is recommended that actigraphy data be combined with subjective sleep data to enhance accuracy of acquired data (Kushida et al., 2001). Using this coupled data collection procedure, Kushida and colleagues (2001) found no significant difference between PSG and combined actigraphy and subjective recordings of total sleep time and sleep efficiency. In order to reduce redundancy in measures only total sleep time, namely the amount of time between time to bed and wake time that is scored as sleep according to the actigraph software, was utilized as sleep efficiency and other variables are calculated using total sleep time.

In order to obtain a subjective measure of sleep quality, participants were required to complete a sleep diary for the night of actigraph assessment. The sleep diary asks for a detailed report of that night’s activities and sleep patterns, including: the time they went to bed, the estimated time they fell asleep, the estimated time it took to fall asleep and the number of caffeinated and alcoholic beverages they consumed that day and so forth (see Appendix A). Time to bed and wake times from the subjective sleep diary were used to set the sleep interval required for calculation of sleep variables (i.e., total sleep time) using the actigraph software.

Blood Pressure

BP Monitors were be attached to a participant’s nondominant arm for the duration of the lab portion of the study to allow for the analysis of reactivity during the arithmetic stress task and time for relative recovery to baseline following the stress task. The VSM-100
BpTRU automatic blood pressure device has been found to be a reliable non-invasive measurement within pediatric and adult populations (ages 3-83 years) (Mattu, Heran, & Wright, 2004). Specifically, when compared to standard auscultatory mercury sphygmomanometer measurements, 89.2% of the BpTRU measurements were within 5 mmHg, with 96.4% and 99.3% of these measures being within 10-and 15 mmHg, respectively (Mattu et al., 2004). Furthermore, in a sample of hypertensive patients, the BpTRU clinic blood pressure monitor was found to correlate significantly better with daytime ambulatory BP ($r = 0.57$) than clinic averages ($r = 0.15$) (Beckett & Godwin, 2005).

**Procedure**

Participants enrolled in the study either via the Psychology Subject Pool website or directly with the Co-Investigator (A.T.E.) via e-mail or telephone contact. The participants were screened for eligibility and then came to the lab to sign consent forms and initiate the study procedure. Hand-held PCs containing self-report questionnaires were provided to participants and instructions were given as to their use. Upon completion of the questionnaires they then had the BpTRU monitor attached to their non-dominant arm and baseline BP and HR was assessed for a 10-minute period.

After completion of the self-report measures and baseline BP and HR readings, subjects participated in a lab stress procedure (counting down from 9000 by increments of 7 while receiving mild harassment, e.g., “You’re always subtracting way too slow. You have to do it much faster”) (see Appendix B). This procedure has been used repeatedly in the laboratory and it has reliably triggered large responses and relatively slow recovery (Anderson et al., 2005). Immediately following the lab stress procedure BP and HR were continually monitored every 2-minutes for the next 20-minutes in order to assess degree and time of recovery relative to baseline. Following recovery participants were required to
complete a written manipulation check to ascertain whether they “saw through” the experimental manipulation (i.e., the harassment).

Following the lab portion of the study, participants were debriefed and provided with detailed instructions about use of the actigraphs and the required diary completion, and were advised to return to the lab the following morning. During the next 16 hours, motor movement and sleep quality was assessed via joint use of the self-report diary and actigraphy. Also assessed during this period (via diaries) were: daily activities (i.e., exercise, physically strenuous events), perceived stress, food and beverage intake (including alcohol), use of prescription and non-prescription drugs, perceived quality of sleep, and degree of acute rumination/worry. Upon returning the actigraph the next day, five BP readings were taken (and averaged) to compare BP change over 24 hours, and subjects were fully debriefed both verbally and in writing.

**Statistical Analyses**

**Hypothesis Testing**

Hypothesis testing was performed using multiple linear regressions with SPSS software. Primary analyses focused on predicting SBP and DBP reactivity and recovery, and objective and subjective sleep quality from psychological factors.

**SBP and DBP Reactivity and Recovery Calculation**

No primary research question is directed at predicting reactivity. However, reactivity affects the speed of recovery. Therefore, the effect of reactivity on recovery needs to be controlled for. In order to provide clarity for this control variable I describe the analysis and core findings of reactivity, however, results of this analysis are discussed in Appendix C.

Baseline BP values were calculated by averaging the last two baseline readings of a
10-minute pre-task assessment period. These two values are the most representative of resting BP and averaging them provides additional reliability. Raw change scores for reactivity were calculated by subtracting the baseline average from the average of all four task readings. Similarly, raw change scores for recovery were calculated by subtracting the baseline average from the average of the first six recovery readings given that BP did not decrease further following the sixth reading.

Sleep Quality Calculation

A nightly sleep index (i.e., total sleep time) was calculated using the compiled actigraph data and software. Time to bed and wake times from the subjective sleep diary were used to set the desired sleep interval.

Regression Analyses

To avoid regression model overfitting covariates predicting our dependent variables were selected by analyzing zero-order correlations for each dependent variable (for the analyses results see below). Covariates and independent variables (IV) were centered if continuous or dummy coded if categorical and subsequently entered into individual hierarchical regression equations predicting physiological dependant variables (DV) in the following way:

- SBP and DBP reactivity: Step 1: covariates, Step 2: psychological IVs, Step 3: gender x psychological IV interactions.
- SBP and DBP recovery: Step 1: covariates, Step 2: psychological IVs, Step 3: gender x psychological IV or psychological IV x reactivity

Interactions between gender x psychological IV and psychological IV x reactivity predicting reactivity and recovery are largely exploratory and not intended to constitute our
primary analyses. Each interaction term was entered individually into a separate regression analysis. Only significant interactions are reported in the results section.

Total sleep time was analyzed in a similar manner, but with psychological variables and physiological variables as predictors. Also included in the model was a fourth step taking into account significant interaction terms from the recovery analyses. Steps 3 and 4 of this model are, again, largely exploratory and are intended to assess whether reactivity or recovery values predict sleep quality and whether the same variables that predict recovery also predict sleep. Variables predicting total sleep time were entered as follows:

Total sleep time: Step 1: covariates, Step 2: psychological IVs, Step 3: reactivity and recovery values, Step 4: significant interactions predicting recovery, Step 5: gender x psychological IV or psychological IV x reactivity/recovery

Step 5 interactions are entered individually into separate regression equations and are considered exploratory with only significant interactions reported.

To assess whether the same psychological variables predict objective and subjective sleep parameters we also performed hierarchical regression with PSQI global score as the DV. IVs were entered as:

PSQI: Step 1: covariates, Step 2: psychological IVs.

Results

Manipulation Check

To assess whether our harassment manipulation succeeded in increasing cardiovascular activation we conducted two paired samples t-tests to determine whether means at baseline, task and recovery differed significantly from one another (see Figure 2). All three means differed significantly for both SBP and DBP. SBP baseline and task had a
mean difference of -15.6 mmHg ($t(135) = -22.32, p < .001$) indicating an increase during task. SBP task and recovery means differed by 11.1 mmHg ($t(135) = 17.60, p < .001$) indicating a reduction in activation during recovery, and baseline and recovery means differed by -4.5 mmHg ($t(135) = -9.90, p < .001$) indicating that, although SBP significantly decreased after task, the mean value did not fully return to baseline. Similarly, DBP baseline and task means differed by -15.3 mmHg ($t(135) = -27.07, p < .001$), task and recovery means differed by 11.2 mmHg ($t(135) = 19.46, p < .001$), and baseline and recovery means differed by -4.1 mmHg ($t(135) = -11.39, p < .001$).

To further analyze the success of our harassment manipulation during the lab portion of the experiment, participants were provided with a manipulation check following the recovery period to assess whether they were aware of the intentional harassment. Forty-four individuals indicated by way of written response that they were aware of the intentional provocation. Responses coded as ‘aware’ included statements such as, “The purpose of the experiment was to try to make me angry”. Inter-rater reliability of the dichotomized coding system for three different coders was 99%. Any disagreement was resolved through discussion of all three coders. A MANOVA, conducted to assess whether reactivity or recovery values differed between those who were aware of the intentional harassment or unaware, revealed no significant group differences (all $p$ values $> .41$; see Table 1 for means and $SD$s of physiological values for aware vs. unaware participants) therefore all subjects were included in the analyses regardless of their reported awareness.

**Covariate Selection**

Descriptives of all covariates and predictor and criterion variables are outlined in Table 2 and correlations among study variables are listed in Tables 3, 4, and 5.
Given the number of variables that could potentially influence our dependent variables we deemed it necessary to analyze the zero-order correlations between criterion variables and biological and behavioral variables before inclusion in our final hierarchical regression analyses as a means to select critical control variables. While this type of post-hoc analysis is not always desirable (Babyak, 2004), we thought it essential in order to maintain power for our main IVs of interest, namely, our psychological and physiological predictors.

SBP and DBP Reactivity and Recovery

Habitual exercise, calculated as self-reported minutes/week, marginally correlated with SBP reactivity \( (r = -.14, p < .10) \). Age \( (r = -.17, p < .05) \), gender \( (r = -.29, p = .001) \) and habitual exercise \( (r = -.18, p < .05) \), were significantly correlated with DBP reactivity. Consumption of caffeine (defined as consumption up to 6 hours prior to experiment time given the 4-6 hour half-life of caffeine, see Benowitz, 1990) before the experiment \( (r = .15, p < .10) \), and time of day the experiment was conducted \( (r = -.15, p < .10) \) were marginally correlated with SBP recovery. Gender was significantly correlated \( (r = -.21, p < .05) \) with DBP recovery, while age was marginally correlated \( (r = -.14, p < .10) \). Therefore, age, gender, habitual exercise, caffeine consumption and experiment time were all included as covariates in subsequent physiological analyses.

Sleep Variables

Our sleep DV was total sleep time, calculated by the actigraph software as sleep duration in minutes minus intervals coded as wake during sleep via a software-derived

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1 Excluded covariates were: daily caffeine intake coded ‘yes’ or ‘no’ and total caffeine intake by number of times consumed (58% of participants consumed caffeine); alcohol coded ‘yes’ or ‘no’ and by total number of beverages consumed (11% of participants); daily exercise coded ‘yes’ or ‘no’ and daily exercise duration (39% of participants); and experience of a stressful event (33% of participants). All \( p \) values > .10.
algorithm. Thus, while males had a mean sleep duration of 389.6 and females a mean of 422.6 minutes, their respective total sleep times were 354.8 and 388.6 minutes. Gender \((r = .20, p < .05)\) and napping \((r = -.28, p = .001)\) were significantly correlated with total sleep time, while habitual exercise was marginally correlated with total sleep time \((r = -.14, p = .10)\). Therefore, these variables were retained as covariates for subsequent sleep analyses.

For the PSQI only gender marginally correlated with the measure \((r = -.16, p < .10)\).

We also explored whether our covariates showed gender differences. There were no gender differences in those who drank caffeine before the experiment (23.7% of males and 28.6% of females; Fisher Exact \(p > .50\)) or in the time of day each gender participated in the experiment (69.4% of males and 74% of females participated in the experiment in the afternoon; Fisher Exact \(p > .50\)). Further, no gender differences were found for individuals who napped versus those who did not (28.9% of males napped and 32.3% of females napped; Fisher Exact \(p > .50\)). An independent sample t-test indicated that there were also no mean gender differences in age \((M = 21.7, SD = 5.3\) for males, \(M = 22.1, SD = 4.8\) for females, \(t(134) = -.45, p > .50\)) or amount of exercise per week \((M = 184.8, SD = 132.2\) for males, \(M = 147.0, SD = 166.8\) for females, \(t(134) = 1.25, p > .20\)).

**Regression Analyses**

**Reactivity and Recovery**

Individual hierarchical linear regression analyses were conducted to determine fit for each of our physiological and sleep parameters.

For individual variables predicting SBP reactivity only diffusion in response to anger was found to be significant \((\beta = -.21, t(131) = -2.11, p < .05)\) with higher levels of diffusion associated with reduced SBP reactivity (for full results see Table 6). High levels of reported habitual exercise marginally predicted reduced SBP reactivity \((\beta = -.17, t(131) = -1.89, p < .05)\).
For DBP reactivity, gender ($\beta = -0.31, t(131) = -3.72, p < .001$) and habitual exercise ($\beta = -0.20, t(131) = -2.34, p < .05$) were significant predictors, with males showing increased reactivity and more habitual exercise associated with less reactivity. There were no significant psychological predictors of DBP reactivity ($-0.14 < \beta < 0.10$, all $p$ values $> 0.18$).

After controlling for SBP reactivity, caffeine consumption prior to the experiment marginally predicted decreased SBP recovery ($\beta = 0.14, t(131) = 1.80, p < .10$; note that higher recovery scores correspond to slower recovery), as did rumination ($\beta = 0.19, t(131) = 1.93, p = .06$). There was also a significant gender x rumination interaction ($\beta = 0.31, t(131) = 2.02, p < .05$; see Figure 3 for depiction of the relationship in the original metric). Given that gender was dummy coded (0 male, 1 female), the variable was reverse coded and the regression analysis rerun to allow for testing of the significance of the simple slopes of the comparison group (Aiken & West, 1991). This analysis indicated that the simple slope for rumination on SBP recovery for females was significant ($\beta = 0.29, t(131) = 2.66, p < .01$), however, the slope for males was not ($\beta = -0.08, t(131) = -0.47, p > .10$), indicating that rumination predicted decreased SBP recovery for females only.

After controlling for DBP reactivity, caffeine consumption before the experiment marginally predicted slower DBP recovery ($\beta = 0.15, t(131) = 1.77, p < .10$). No psychological variables independently predicted DBP recovery, however, there was a significant gender x rumination interaction ($\beta = 0.36, t(131) = 2.02, p < .05$; see Figure 4 for depiction of the relationship in the original metric). Tests of simple slopes indicated that the slope for rumination on DBP recovery for females was not significant ($\beta = 0.10, t(131) = 1.0, p > .10$), while the slope for males was marginally significant ($\beta = -0.31, t(131) = -1.76, p < .10$). This result signifies a trend for rumination to predict greater DBP recovery in males only.
Total Sleep Time

Females showed significantly more total sleep time as compared to males ($\beta = .19$, $t(132) = 2.32, p < .05$), and individuals who napped showed significantly less total sleep time ($\beta = -.28, t(132) = -3.40, p = .001$). Hostility also significantly predicted total sleep time ($\beta = -.20, t(132) = -2.05, p < .05$) with more hostility associated with less total sleep time. None of the physiological scores (reactivity or recovery) approached significance for predicting total sleep time ($-.19 < \beta < .16$, all $p$ values $>.19$), nor did the gender x rumination interaction that predicted SBP and DBP recovery ($\beta = -.004, t(132) = -.26, p > .50$). However, when other interactions were explored in a 5th step in the hierarchical analysis there was a significant gender x PSWQ interaction ($\beta = .50, t(132) = 2.74, p < .01$, see Figure 5). Tests of simple slopes for PSWQ scores on total sleep time indicated that the slope for females was not significant ($\beta = .17, t(132) = 1.41, p > .10$), while the slope for males was significant ($\beta = -.46, t(132) = -2.26, p < .05$). Therefore, higher rates of worry predicted less total sleep time only for males. See Table 8 for description of the hierarchical analysis for total sleep time.

Pittsburgh Quality Sleep Index

Higher scores on the PSWQ significantly predicted higher scores on the PSQI ($\beta = .30, t(135) = 2.97, p < .01$), while gender approached significance ($\beta = -.16, t(135) = -1.83, p < .10$) with males reporting higher scores (see Table 9).

Despite some small differences in mean scores on other psychological measures, there were no other significant gender x psychological measure interactions (see Table 10). Nor were there any significant psychological measure x reactivity/recovery interactions for either recovery or sleep variables.
Discussion

The primary aim of the current study was to explicate the relationship between psychological constructs of anger, hostility, rumination and worry with respect to recovery and sleep quality. As hypothesized, results indicate that hostility, worry and rumination predict recovery, as well as sleep parameters, however, they do so differentially. Our second and third hypotheses, namely, that the same psychological variables that predict recovery in the lab would also predict sleep quality, and that recovery rates in the lab predict sleep quality, were not supported.

Manipulation Check

Results indicate that the desired reactivity/recovery pattern in response to our harassment manipulation was achieved. The mental arithmetic task combined with harassment was associated with large physiological responses that differed from baseline and recovery values. However, rather surprisingly, manipulation checks provided following the lab stress procedure indicated there were no differences between individuals coded as ‘aware’ of the intentional anger provocation and ‘unaware’ on any index of physiological change. The null result was not due to a lack of power. For example, we had 97.6% power to detect a significant 2-tailed difference of only 2 points in SBP or DBP mmHg change, with a \( SD = 6 \) and alpha of \( p = .05 \). To account for this finding we suggest that it is possible that participants asked themselves questions about the underlying hypothesis only once the task had progressed to the recovery period or at least well into the task period. Alternatively, they may not have thought much about the intention to provoke anger because the task itself was quite engaging. If this was the case, then speculation as to the true purpose of the task (which psychology undergraduates are prone to do) may not have affected the task (although this supposition does not account for the lack of difference in recovery values that were also

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found). Therefore, insight into the true purpose of the experiment might only have occurred with written a priori prompting to consider it. Another possibility is that the mental arithmetic task itself may have been enough to elicit large physiological responses independent of the harassment, however, the magnitude of task responses (average SBP change of 15.6 mmHg, and DBP change of 15.3 mmHg) correspond more with responses for harassed conditions as opposed to non-harassed conditions in studies examining BP change dependent on task characteristics (Earle, Linden & Weinberg, 1999; Suarez, & Williams, 1990), and analysis of Figure 2 indicates increased physiological responses upon implementation of the harassment (T2 to T4 compared to T1).

**Stress Recovery**

Given that manipulation checks revealed satisfactory evidence for success of the experimental manipulation other findings can be interpreted.

Results from the lab portion of the study indicate that the construct of rumination in response to anger may be important in predicting cardiovascular recovery from stress. This provides evidence for the hypothesis that rumination may serve to delay the time to recover from stress due to perseverative cognitions that may prolong or reactivate a negative emotional response leading to more deleterious health consequences (for review see Brosschot, Gerin & Thayer, 2006). Our results show that rumination trended toward predicting SBP recovery. As well, differential gender effects were found for the relationships between rumination and both SBP and DBP recovery. Rumination was not correlated with gender, indicating that women and men obtained similar scores on the measure of angry rumination. However, women high in anger rumination showed less recovery than those low in rumination, while men high in rumination showed more recovery than those low in rumination. Similar levels of recovery where found at low levels of
rumination for both men and women. That rumination served to slow recovery in women is in partial agreement with previous literature showing decreased recovery times following experimentally induced rumination, however, the gender effects were opposite to those found here with men showing less recovery than women (Glynn, Christenfeld & Gerin, 2002). It is not clear whether this discrepancy in research findings is perhaps due to the directed rumination component of the aforementioned study, as compared to the spontaneously occurring rumination tendencies potentially occurring in the current study. Nor is it clear how high rumination scores in men facilitate recovery. A caveat to all gender specific analyses in the study is that we did have a large discrepancy in the male to female ratio of participants and multiple steps in the hierarchical regression analyses. Therefore, we may not have had adequate power to fully assess gender specific rumination with respect to BP. Thus, this preliminary finding is in need of further exploration.

While rumination in response to anger as measured by the BARQ predicted cardiovascular parameters in our lab stress procedure, direct anger out did not. Research on anger coping styles have found inconsistent results with regard to which style is associated with more pronounced cardiovascular responses to stress. Most studies have dichotomized anger-out vs. anger-in styles in accordance with Spielberger and colleagues’ (1985) conceptualization of anger expression, although, these studies have often shown opposing findings (e.g., Faber & Burns, 1995; Lai & Linden, 1992). If one follows the anger-in vs. anger-out classification then our study lends support for the anger-in hypothesis rather than anger-out, as Linden and colleagues (2003) found BARQ rumination to correlate most with anger-in ($r = .49$) and direct anger out to correlate highly with anger-out ($r = .72$). The current trend, however, is to examine a broader scope of anger expression styles and our findings add to this body of work by showing significant findings for a rumination expression
style associated with cardiovascular responses to laboratory stress (for discussion of the impact of the anger expression style of diffusion on reactivity see Appendix C).

Direct anger-out, was most correlated with trait anger ($r = .47$), rather than other anger expression styles, and trait anger and hostility also did not predict cardiovascular responses in the lab stress procedure, indicating that the variables that failed to predict our cardiovascular parameters may be somewhat interrelated. That trait anger and hostility were not found to impact reactivity or recovery is in accordance with recent bodies of work indicating that anger coping style is the primary determinant of cardiovascular responses to stress (Hogan & Linden, 2004; Brosschot & Thayer, 1998). Our findings are also consistent with studies that assess both trait hostility and anger coping within the same analysis, suggesting that it is anger coping style that predicts cardiovascular functioning over and above individual traits (Linden et al., 2008). This may indicate that how one copes with anger or provocation may be more important than more enduring dispositions.

We also did not find evidence that trait worry predicted cardiovascular activation patterns during the lab stress procedure, suggesting that the related trait of rumination may be more influential in recovery from acute stress.

This study also captures the potential detrimental role of caffeine on recovery from stress. Few studies were found that reported the impact of caffeine on recovery, instead focusing on reactivity, and we suggest this may be an important area of investigation with implications for cardiovascular health. Caffeine consumption did not influence stress reactivity, which was somewhat unexpected given past literature showing that caffeine can have an additive effect on blood pressure in laboratory stress procedures (Lane, Adcock, Williams, & Kuhn, 1990; Greenburg, & Shapiro, 1987). However, most studies have assessed caffeine consumption immediately prior to stress provocation, while subjects in this
study were coded as having consumed caffeine up to 6 hours prior to experiment time.
Greenburg and Shapiro (1987) found that increased BP reactivity was apparent for 45
minutes after consumption, suggesting that our coding strategy may have been too liberal.

**Total Sleep Time**

Sleep quality was significantly predicted by psychological variables. While trait
hostility did not predict recovery in the lab stress procedure, hostility did predict total sleep
time as indicated by actigraph measurements. This coincides with previous research
indicating this relationship in self-reported sleep disturbances (Brisette & Cohen, 2002;
Koskenvuo et al., 1988), and research finding higher nightly ambulatory blood pressure in
individuals high in hostility (Jamner, Shapiro, Goldstein, & Hug, 1991).

There was also a significant gender x worry interaction predicting total sleep time.
Overall women had more total sleep time than men, and men who were high in worry
showed significantly less total sleep time than those low in worry as assessed by the PSWQ.
This relationship was not found for women. This finding is interesting in light of the fact that
rumination, a related construct, facilitated recovery in men and hindered recovery in women
in the lab stress procedure. Women often show higher rumination and worry tendencies than
men (Robichaud, Dugas, & Conway, 2003), however this study indicates this may not
translate into the domain of sleep. The finding also conflicts with a study conducted by
Brosschot and colleagues (2007) who did not find an association between trait worry
(measured with the PSWQ) on nocturnal heart rate and heart rate variability, however these
researchers did not report a test of a gender x worry interaction. Again, gender specific
interactions such as this finding must be interpreted with caution due to the discrepant male
to female ratio in this study.
Self-Reported Sleep

Worry also significantly predicted self-reported sleep, while hostility did not. Nor were there gender differences with respect to self-reported sleep. This finding highlights the need for objective assessment of sleep as utilized in this study to fully capture the relationship between psychological variables and sleep quality.

Sleep and Recovery

The current study was undertaken in part to provide evidence that the same psychological variables that predict recovery in the lab also predict poor sleep, and whether recovery from the lab stress procedure predicts sleep quality, thereby indicating a general non-recovery tendency. While previous research has indicated this relationship it has not been assessed using the same population sample. Contrary to our hypothesis we did not find evidence that the same psychological variables that predicted recovery in the lab stress procedure also predicted total sleep time, nor did we find evidence that recovery from the lab stress procedure predicted total sleep time. Therefore, support for the view of sleep as an additional marker of delayed recovery within the same individual was not found.

Future Directions

Although we did not find evidence for the hypothesis that delayed recovery and sleep are markers of the same non-recovery tendency, our results are not entirely discouraging. Rumination was found to influence recovery in the current study, and previous research has found it to be associated with disturbed sleep (Thomsen et al., 2003). Further, worry and rumination are thought to be highly related but distinct constructs with perseverative thought a key component of each (Brosschot et al., 2006; Fresco, Frankel, Mennin, & Heimberg, 2002). In our study worry and rumination were moderately correlated \( r = .48 \), indicating
that if an individual is high in one domain they are likely high on the other. Further, hostility and rumination were significantly correlated ($r = .33$). This indicates that there was some interrelatedness among predictors of both of our posited indices of recovery. Therefore, it may be that there is an overall disposition that better accounts for both reduced cardiovascular recovery and poor sleep. Neuroticism, for example, may hold this potential. Linden and colleagues (2003) found that anger rumination moderately correlated with neuroticism ($r = .48$). Furthermore, the PSWQ ($r = .74$; Molina & Borkovec, 1994), and the AQ hostility measure were found to highly correlate with neuroticism ($r = .61$; Sharpe & Desai, 2001). Neuroticism has also been linked to poor self-reported sleep quality (Dorsey & Bootzin, 1997; Gray & Watson, 2002; Williams & Moroz, 2009) and poor stress reactivity and recovery (for review see Chida & Hamer, 2008). Future studies should address whether neuroticism, or other more global dispositions are associated with delayed recovery and objectively measured sleep quality.

Another potential confound of the current study was that we only assessed one night of sleep. Obtaining a more aggregated index of sleep may more accurately capture an individual's habitual sleep pattern and therefore represent a stronger test of the expected relationship between delayed recovery and sleep quality.

Further, future studies should address the relationship between recovery and sleep quality in a more ecologically valid manner. Assessment of stress recovery by way of ambulatory BP monitoring combined with nightly actigraphy assessment, for example, may show the desired result. The current study did not find a significant association between the experience of a daily stressor and sleep quality, however, we did not assess frequency or severity of daily stressors. The next planned project intends to examine the same main hypotheses as examined in this study, improving on the current study design by obtaining
seven nights worth of objective sleep data and diary data assessing daily stressors, combined with a period of ambulatory BP monitoring to assess daily and nightly cardiovascular parameters.

**Strengths and Limitations**

Strengths of this study include the analysis of objectively measured sleep quality, as compared to the sole use of self-report in the majority of the existing literature, and use of a harassment protocol that has been shown to produce large physiological responses in a variety of studies. The study also attempted to assess multiple potentially influential variables in the same analyses to determine which variables predicted our DVs over and above other related variables, however, this came with the consequence of a loss of power due to our limited sample size and multi-step regression analyses. Therefore, we may not have had the power to detect potentially meaningful interactions among study variables. Other limitations include an uneven gender and ethnic ratio in our sample population, thereby limiting our interpretation of subgroup results. As well, as indicated above, we only obtained one night of sleep assessment, which may not have accurately captured an individual’s habitual sleep pattern. The study sample also consisted largely of 1st and 2nd year undergraduate students, and students have been found to have highly variable sleep patterns, and increased sleep difficulties as compared to non-student populations (Buboltz, Brown, & Soper, 2001; Lack, 1986). Therefore, our study findings may not replicate to other non-student populations. This also has implications for assessing only one night of sleep.

Limitations also include a potential failure to identify critical control variables. For example, we did not assess smoking, which is known to have cardiovascular implications (Perkins, Epstein, Jennings, & Stiller, 1986); this may not have been overly problematic due to the low frequency of smoking in undergraduate populations (11.3%; Adlaf, Gliksman,
Demers, & Newton-Taylor, 2003). Additionally, we did not assess depression, which has been found to impact both cardiovascular responsivity and sleep quality (for review see Kibler & Ma, 2004; Hall et al., 2000).

Further changes in emotional state or cognitive processing during the laboratory stress protocol were not assessed, and therefore, data interpretation pertains only to perceived change in cardiovascular parameters with no information as to the mechanisms that may underlie these responses.

**Contributions to the Research Field**

Despite the identified limitations, this study provides substantial contributions to research in the domain of cardiovascular activation and sleep. Many of the contributions have been described above but a few findings deserve additional attention. For instance, we found that the anger provocation protocol used here produced large physiological responses, regardless of reported awareness of the intentional harassment. This type of protocol requires deception or at least concealment of the hypothesis in order to mimic real-life provocations and researchers routinely conduct post-experimental validity checks to assess the success of deception in order to rule out competing hypotheses. We posit that concealment of the hypothesis in anger provocation experiments is usually effective and may not be a threat to a study's internal validity (Linden, Talbot Ellis, & Millman, 2009).

Support was found for the hypothesis that anger coping styles are primary determinants of cardiovascular responses in the laboratory, and this coincides with Brosschot and Thayer's (1998) proposition that social reality forces more anger inhibition (here rumination) than anger expression and is consequently the style most detrimental to cardiovascular health. This line of research is unique in assessing both anger coping styles
and trait anger/hostility in the same individuals and therefore provides new insight into the pathological style associated with disease development.

Analyses also provide preliminary findings for a new line of inquiry into the effect of caffeine on cardiovascular recovery rates. While a substantial portion of the existing literature reports the impact of caffeine on reactivity, few, if any, studies discuss the impact on recovery.

Further, the study was unique in analyzing the impact of psychological variables on sleep quality. Few studies have assessed this relationship using objective sleep parameters, focusing instead on subjective sleep reports. Evident in this study was the impact of hostility on sleep, a relationship that has not received much attention in the existing literature (a literature search for hostility predicting objective sleep parameters was unsuccessful in revealing any such studies). Given that hostility has been linked to cardiovascular disease, and that hostility did not influence cardiovascular responses over and above anger expressions style, the impact of hostility on sleep may provide additional insight into a potential pathophysiological link to cardiovascular disease development.

Lastly, this research provides insight into possible areas of intervention targeting prevention of cardiovascular disease and known risk factors such as hypertension. For instance, reducing caffeine intake may reduce stress related cardiovascular activation. Further, anger coping styles may be more modifiable than more enduring trait anger and hostility and as such, psychological interventions promoting healthier anger coping styles may also serve to decrease the impact of stress on cardiovascular disease. Rumination before sleep has also been found to be modifiable with instructions to ruminate on a stressful event shown to decrease self-reported sleep quality in those high in trait rumination compared to a distracter condition (Guastella & Moulds, 2007), and the same may hold true.
for worry. Techniques such as mindfulness-based interventions may prove beneficial in this domain (Carlson & Garland, 2005).

In sum, this research was successful in elucidating the relationships between psychological variables and cardiovascular activation and sleep quality. Identification of such relationships is essential in determining the pathophysiological pathway to cardiovascular disease development. Future studies are needed to build on preliminary findings evinced in this line of inquiry.
Table 1
Reactivity and Recovery Means and Standard Deviations for Aware vs. Unaware Participants

<table>
<thead>
<tr>
<th>Variable</th>
<th>Aware of Intention</th>
<th>Unaware of Intention</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$M$</td>
<td>$SD$</td>
</tr>
<tr>
<td>SBP Reactivity</td>
<td>15.14</td>
<td>9.22</td>
</tr>
<tr>
<td>DBP Reactivity</td>
<td>13.68</td>
<td>6.28</td>
</tr>
<tr>
<td>SBP Recovery</td>
<td>4.71</td>
<td>5.41</td>
</tr>
<tr>
<td>DBP Recovery</td>
<td>5.08</td>
<td>4.02</td>
</tr>
</tbody>
</table>

Note: Values indicate raw change scores
Table 2
Means and Standard Deviations for Covariates, Psychological, Physiological and Sleep Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>$M$</th>
<th>$SD$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Covariates</strong></td>
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</tr>
<tr>
<td>Age</td>
<td>21.99</td>
<td>4.89</td>
</tr>
<tr>
<td>% Female participants</td>
<td>72.1</td>
<td></td>
</tr>
<tr>
<td>% Caffeine intake prior to experiment</td>
<td>27.2</td>
<td></td>
</tr>
<tr>
<td>% Participated in experiment in the afternoon</td>
<td>70.6</td>
<td></td>
</tr>
<tr>
<td>% Nappers</td>
<td>30.9</td>
<td></td>
</tr>
<tr>
<td>Reported exercise/week</td>
<td>157.53</td>
<td>158.35</td>
</tr>
<tr>
<td><strong>Physiological variables</strong></td>
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<td></td>
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<tr>
<td>SBP Baseline</td>
<td>101.06</td>
<td>8.45</td>
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Note: Sample sized for individual statistics may vary; maximum $N = 136$
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*p < .05, **p < .10
Table 4
Zero-order correlations between Total Sleep Time and Predictors

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*p < .05, **p < .10
Table 5
Zero-order Correlations between the Pittsburgh Quality Sleep Index and Predictors

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*p < .05, **p < .10
Table 6
Results of Hierarchical Regression Analyses for SBP and DBP Reactivity

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Step 2

|                         |         |     |         |              |         |     |         |              |
| BARQ Direct Anger Out   | -.04    | -.33 | .02     | .17          |         |     |
| BARQ Diffusion         | -.21*   | -2.11 | -.04    | -.47         |         |     |
| BARQ Rumination        | .12     | 1.11 | -.14    | -1.37        |         |     |
| AQ Anger               | .09     | .85  | .10     | 1.02         |         |     |
| AQ Hostility           | .07     | .69  | .09     | .86          |         |     |
| PSWQ                   | -.14    | -1.22 | .03     | .24          |         |     |

$^{*} p<.05, \ **p<.01, \ ***p<.001$

Note: Regression analysis is based on pairwise deletions.
Table 7
Results of Hierarchical Regression Analyses for SBP and DBP Recovery

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*p<.05, **p<.01, ***p<.001
Note: SBP and DBP reactivity were controlled for in each respective analysis. Regression analysis is based on pairwise deletions.
Table 8
Results of Hierarchical Regression Analyses for Total Sleep Time

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* p < .05, ** p < .01, ***p < .001

Note: Regression analysis is based on pairwise deletions.
Table 9
Results of Hierarchical Regression Analyses for the Pittsburg Quality Sleep Index

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<th>Predictors</th>
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*p<.05, p<.10, ***p<.001
Note: Regression analysis is based on pairwise deletions.
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<td>AQ Hostility</td>
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<tr>
<td>Global PSQI Score</td>
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Figure 1
Experimental Domains

Blood Pressure Recovery

Psychological Variables

Sleep Parameters
Figure 2

Total Systolic and Diastolic Activation Change from Baseline over Experiment Time
Figure 3
Mean Change of Systolic Blood Pressure as a Function of Rumination and Gender
Figure 4
Mean Change of Diastolic Blood Pressure as a Function of Rumination and Gender
Figure 5
Total Sleep Time as a Function of Worry and Gender
References


Lane, J. D., Adcock, A., Williams, R. B., & Kuhn, C. M. (1990). Caffeine Effects on Cardiovascular and Neuroendocrine Responses to Acute Psychosocial Stress and
Their Relationship to Level of Habitual Caffeine Consumption. Psychosomatic Medicine, 52, 320-336.


Appendices

Appendix A

Sleep Diary

Instructions: Please fill out this sleep diary the morning following your laboratory visit (to coincide with the day and night during which you wore the activity and sleep monitor).

Subject Number:

Time you got up (date & time)

Date: ____________________________  Time: ____________________________

Did this represent a “typical” day for you? Yes □  No □

Sleep Details

Estimated time went to bed:

Estimated time fell asleep:

Estimated time to actually fall asleep: (sleep latency)

Did you nap at any time throughout the day? Yes □  No □

If yes, please indicate,

Number of naps:

Time of day when naps taken: (i.e., 1pm-2pm in the afternoon)

Total length of naps (minutes):

Lifestyle Factors

Did you consume any caffeinated beverages during the previous day? (i.e., coffee, tea (non-herbal), hot-chocolate, Coca-cola, etc.) Yes □  No □
Please list the type of beverage(s), amount and the time consumed:

Beverage Type 1: ____________________________
Time: ________________________________

Beverage Type 2: ____________________________
Time: ________________________________

Beverage Type 3: ____________________________
Time: ________________________________

Did you consume any alcoholic beverages during the previous day?
(i.e., wine, spirits, beer, etc.)
Yes ☐
No ☐

Please list the type of beverage(s), amount (i.e., can, glass, etc.) and the time consumed:

Beverage Type 1: ____________________________
Time: ________________________________

Beverage Type 2: ____________________________
Time: ________________________________

Beverage Type 3: ____________________________
Time: ________________________________

Did you consume any prescription or non-prescription drugs the previous evening?
(i.e., ativan, marijuana, etc.)
Yes ☐
No ☐

Please list the type of drugs and the time consumed:

Drug Type 1: ____________________________
Time: ________________________________

Drug Type 2: ____________________________
Time: ________________________________

Drug Type 3: ____________________________
Time: ________________________________

Did you do any exercise during the previous day?
Yes ☐
No ☐

If yes, please indicate:

Type of activity:
(i.e., jogging, yoga, aerobics, weight lifting, etc.)
Duration (minutes):

Time of day:  
(i.e., 1pm-2pm in the afternoon)

Did you experience any changes in appetite during the day?  
Yes  □  No  □

If yes, please indicate,  
Increased Appetite  □
Decreased Appetite  □

Emotional Well-Being

Did you experience any stressful events during the previous day?  
Yes  □  No  □

(i.e., argument with friend or family member, exam, date, etc.)

If yes, please indicate,  
What was the event?
What time did this occur?

How did you feel yesterday?  
(please select only one option)  
Not very good  □  Fairly Alert  □
Somewhat tired  □  Wide awake  □

Indicate your irritability level yesterday:  
(please select only one option)  
None  □  Moderate  □
Some  □  Fairly High  □
High  □

The three statements on the left in the table below represent difficulties staying awake. For each day of the week, record how frequently during the day you experience this level of sleepiness.

0 =Not at all  1=Occasionally  2=Some of the time  3=Most of the time  4=All the time
<table>
<thead>
<tr>
<th>Mon</th>
<th>Tues</th>
<th>Wed</th>
<th>Thurs</th>
<th>Fri</th>
<th>Sat</th>
<th>Sun</th>
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<tbody>
<tr>
<td>I fought off/ignored a need to sleep</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>I dozed off/fell asleep without meaning to</td>
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<td></td>
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<tr>
<td>I needed caffeine or another stimulant drug to stay awake</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Please provide any comments below regarding concerns you may have regarding your sleep patterns:
Appendix B

Harassment Script

Script 1: "Look [participant name], you're always subtracting way too slow. You've got to do it much faster. Continue where you stopped."

Script 2: "[participant name], you're still too slow and also inaccurate. This can't be your best. Now try it again from where you left off."

Script 3: "You're obviously not good enough at doing this, now try harder. Keep going!"
Appendix C

Reactivity Discussion

Results from the lab portion of this study indicate that habitual diffusion in response to anger describes those individuals with a lesser acute physiological response to a stressor. Importantly, this finding corresponds with previous research implicating this construct in BP non-dipping at night. Linden and colleagues (2008) found that diffusion was the only psychological predictor of BP dipping with individuals higher in diffusion showing greater SBP dipping indicating a healthier nightly cardiovascular activation pattern. Diffusion in response to anger can be conceptualized as first recognizing the anger and then diluting the emotive response by directing attention to other tasks (Linden et al., 2008). Therefore, participants higher in diffusion may have been able to recognize their anger response to provocation and consequently address their response faster throughout the stress task, perhaps by redirecting the anger emotion to the task itself, thereby decreasing their physiological response. Note that this study was not able to assess whether or not individuals higher in anger diffusion processed anger in a distinct manner, or indeed if any of the subjects responded to the provocation with the anger emotion, only that diffusion differentially predicted reactivity. Therefore, interpretations into the potential cognitive mechanisms underlying these differences are merely speculative.

While gender was correlated with diffusion, with women obtaining a slightly higher mean score, there was not a significant gender x diffusion interaction predicting SBP reactivity.

DBP reactivity was not predicted by any of our psychological variables or by any gender x psychological variable interactions. Contrary to SBP reactivity, however, males showed greater DBP reactivity even after inclusion of psychological measures indicating that
gender is an independent predictor of reactivity. This finding coincides with previous literature using harassment-based anger provocation protocols (Earle, Linden, & Weinberg, 1999).

While SBP and DBP are related indices of cardiovascular activation with increases in one corresponding with increases in the other, they are treated here as separate variables of interest consistent with existing literature (for review see Linden et al., 1997). SBP and DBP reactivity were highly correlated ($r = .60$), yet, results indicate that each was influenced differentially by predictor variables, lending support to the conceptualization of SBP and DBP as non-overlapping constructs of interest. Further, important to note here is that trait elevations in negative affect, namely anger, hostility and worry, were less important in predicting reactivity outcomes than was diffusion.

Our findings also indicate the impact of behavioral variables on reactivity. For instance, the results highlight the buffering effect of physical exercise on stress reactivity as individuals who reported more exercise minutes per week also showed a corresponding decrease in reactivity. Physical fitness has been shown to modulate cardiovascular reactivity and researchers speculate this may be due to a number of factors, including increased sensitivity to β-adrenergic stimulation, resulting in less sympathetic nervous system activity, changes in hemodynamics, or in the release of insulin (Crews & Landers, 1999). That physical fitness did not influence cardiovascular recovery is in keeping with the hypothesis that fitness serves to modulate sympathetic nervous system activity rather than parasympathetic nervous system activity (Shulhan, Scher, & Furedy, 1986). However, other studies have found evidence that physical fitness facilitated recovery time suggesting there may also be overall hemodynamic alterations resulting in BP changes (Blumenthal et al., 1988; Cox, Evans, & Jamieson, 1979).
Appendix D

The University of British Columbia
Office of Research Services
Behavioural Research Ethics Board
Suite 102, 6190 Agronomy Road,
Vancouver, B.C. V6T 1Z3

CERTIFICATE OF APPROVAL - FULL BOARD

<table>
<thead>
<tr>
<th>PRINCIPAL INVESTIGATOR:</th>
<th>INSTITUTION / DEPARTMENT:</th>
<th>UBC BREB NUMBER:</th>
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<tbody>
<tr>
<td>Wolfgang Linden</td>
<td>UBC/Arts/Psychology, Department of</td>
<td>H07-02656</td>
</tr>
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| INSTITUTION(S) WHERE RESEARCH WILL BE CARRIED OUT: |
|-----------------|-----------------|
| Institution     | Site            |
| UBC             | Vancouver (excludes UBC Hospital) |

Other locations where the research will be conducted: N/A

<table>
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<th>CO-INVESTIGATOR(S):</th>
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<tr>
<td>Alena Talbot Ellis</td>
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<tr>
<td>Sleep and stress: Predictors of failure to recover</td>
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The application for ethical review and the document(s) listed above have been reviewed and the procedures were found to be acceptable on ethical grounds for research involving human subjects.

Approval is issued on behalf of the Behavioural Research Ethics Board and signed electronically by one of the following