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Sleep Duration and Cardiovascular Responses to Stress in Undergraduate Men

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Abstract

Short sleep has been related to incident cardiovascular disease, but physiological mechanisms accounting for this relationship are largely unknown. This study examines sleep duration and cardiovascular stress responses in 79 healthy, young men. Sleep duration was assessed by wrist actigraphy for seven nights. Participants then completed a series of laboratory stress tasks while heart rate and blood pressure were monitored. Shorter total sleep time was related to a greater reduction in high-frequency heart rate variability during stress tasks, and to prolonged elevations in heart rate and diastolic pressure following tasks. Associations were independent of age, race, body mass index, caffeine intake, and smoking status. In sum, healthy young men with shorter actigraphy-assessed sleep exhibit less cardiac vagal activity, and poorer heart rate and diastolic blood pressure recovery, upon encountering stressful stimuli, than those with longer sleep.

Keywords

sleep; actigraphy; reactivity; recovery; heart rate; blood pressure; vagal; stress; heart rate variability

Sleep Duration and Cardiovascular Responses to Stress in Undergraduate Men Self-report and objective measures of short sleep duration have been associated with incident coronary heart disease, myocardial infarction, and death from cardiovascular causes (Ayas et al., 2003; Chen et al., 2008; Ikehara et al., 2009; King, Knutson, Rathouz, Sidney, Liu, & Lauderdale, 2008). Disruptions in autonomic function and elevations in blood pressure are among the proposed mechanisms accounting for the relationship between short sleep and cardiovascular morbidity. For instance, short sleep has been associated with increased heart rate, blood pressure, and sympathovagal balance in cross-sectional studies (Barnett & Cooper, 2008; Knutson et al., 2009; Rodríguez-Colón et al., 2011), and data from some, but

not all, experimental sleep deprivation studies demonstrate that sleep loss alters autonomic activity and increases blood pressure (Meier-Ewart et al., 2004; Zhong et al., 2005).

Sleep duration and stress are often inversely related. Short sleep may be more likely to occur in the midst of heightened psychological stress (Kashani, Eliasson, & Vernalis, 2012; Mosca & Aggarwal, 2012; Roberts, Roberts, & Xing, 2011), and events on days preceded by a shorter night of sleep are described as more distressing (Hamilton et al., 2008; Kumari, Badrick, Ferrie, Perski, Marmot, & Chandola, 2009). In addition to subjective reports of stress, there is also evidence that sleep loss may influence neural modulation of the autonomic stress response. For instance, Yoo et al. (2007) demonstrated that acute sleep deprivation leads to increased functional connectivity between the amygdala and autonomic-activating centers of the brainstem in reaction to negative stimuli. Few studies, however, have examined whether such findings translate to sleep duration being correlated with elevated cardiovascular responses to acute, stressful stimuli. Exaggerated and prolonged blood pressure responses to psychological stress have been shown to predict cardiovascular morbidity and mortality, independent of baseline blood pressure (Chida & Steptoe, 2010). There is also evidence that stressor-evoked decreases in heart rate variability (an index of cardiac vagal control; Gianaros et al., 2005; Matthews, Solomon, Brady, & Allen, 2003; Steptoe & Marmot, 2005) and delayed heart rate recovery following acute psychological stress (Chida & Steptoe, 2010) are related to subclinical heart disease and increases in blood pressure over time. Thus, examination of how sleep and psychological stress interact to influence cardiovascular activity may be relevant in understanding the link between short sleep duration and the development or progression of cardiovascular disease.

Two studies have investigated the effects of experimental sleep deprivation on cardiovascular stress responses in humans. One reported that a night of sleep deprivation increased blood pressure reactivity during a speech task, but not during a Stroop color-word interference task (Franzen et al., 2011), while the other reported no effect of sleep deprivation on heart rate or blood pressure responses during a battery of psychological and physical stressors (Kato, Sigurdsson, Narkiewicz, Pesek, & Somers, 2000). Apart from sleep deprivation studies, Stepanski et al. (1994) showed that adults with insomnia slept for a shorter duration and had greater heart rate during a psychomotor task than those without insomnia, and Palesh et al. (2008) showed that decreased actigraphy-measured sleep continuity over three nights was associated with lower respiratory sinus arrhythmia during the Trier Social Stress task in women with metastatic breast cancer. Thus, while the few investigations of sleep and cardiovascular stress responses offer some evidence that the two may be linked, they either have focused on experimental sleep deprivation or clinical populations. It is unclear how these findings might generalize to healthy individuals who experience short sleep in more naturalistic settings.

The current study examines whether normative variation in sleep duration is associated with cardiovascular responses to psychological stress in a sample of healthy, undergraduate men. Wrist actigraphy assessments of sleep duration over one week are examined in relation to blood pressure, heart rate, and high-frequency heart rate variability (HF-HRV) responses to acute psychological stressors in the laboratory. We hypothesize that shorter actigraphy-assessed sleep duration will be associated with exaggerated reactivity and prolonged recovery of cardiovascular parameters in response to stress.

Method

Participants

Eighty undergraduate men between the ages of 18-30 were recruited from the University of Pittsburgh to participate in a study of “Stress and Sleep.” Only men were included, in order

to eliminate sex-specific effects on cardiovascular responses to stress and sleep. Specifically, cardiovascular stress response patterns differ by sex (Stoney, Davis, & Matthews, 1987), and the associations between stress reactivity and future cardiovascular risk status are more pronounced in men than in women (Chida & Steptoe, 2010). Men also have shorter, less efficient sleep than women (Goel, Kim, & Lao, 2005; Lauderdale et al., 2006). Participants were recruited through an undergraduate research pool, in which students receive course credit for participating in research studies, or through advertisements on campus. Exclusion criteria included engagement in overnight or shift work, a diagnosed sleep disorder, regular medication use for sleep (defined as taking sleep medication three nights/week), binge drinking (five drinks at a time in the past month), reporting a body mass index (BMI) reflective of obesity ($BMI > 30$), anti-hypertensive or cardiac medication use, and likelihood of a depressive disorder as reflected by endorsement of one of two critical depression diagnostic criteria (“decreased interest or pleasure” or “feeling down, depressed, or hopeless” for more than half of the time over the past two weeks; Spitzer, Kroenke, & Williams, 1999) or antidepressant use. Eligible participants were also screened on self-reports of habitual sleep duration over the past month. The study recruited roughly equal groups of self-reported short sleepers (47%; defined as individuals who reported a habitual nocturnal sleep duration of 6 hours) and average length sleepers (53%; defined as individuals who reported a habitual nocturnal sleep duration of 7-8 hours), in order to obtain a range of variation in sleep duration. Participants completed informed consent and received either monetary reimbursement or undergraduate research credit for study completion.

Measures

Actigraphy—An Actiwatch-16 (Philips Respironics, Inc.) was worn on the non-dominant wrist continuously for seven days and nights to measure sleep as inferred from movement and acceleration patterns. The actiwatch is widely used in research studies and has been validated against polysomnography (Tyron, 2004). Data were stored in one-minute epochs. Major rest intervals were determined by the “tried to go to sleep” and “woke up for the day” times recorded in participant sleep diaries (see below). Validated software algorithms (version 5.0) were then used to estimate sleep parameters within the designated rest intervals, using the medium detection threshold. The main sleep variable hypothesized to be related to cardiovascular responses to stress was *total sleep time*, defined as the actual time scored as sleep within a given rest interval, excluding periods of wakefulness after sleep onset. Total sleep time was averaged across the seven nights preceding laboratory stress testing. Actigraphy-assessed daytime naps were assessed using a software algorithm that automatically detects and creates minor rest intervals based on when the participant is immobile. A rest interval duration minimum of 15 minutes was used to auto-detect naps (i.e., the scoring program searched for daytime sleep only within minor rest intervals greater than or equal to 15 minutes). *Daily nap minutes* were averaged across the seven days of the study.

Sleep diary—During the actigraphy assessment period, participants completed morning diaries in which they recorded sleep variables from the prior night (time tried to fall asleep, latency to sleep, wake after sleep onset, time of final morning wake-up, sleep quality). Participants chose between completing paper diaries versus using a secure internet website. Seventy-six participants chose to complete the internet-based diaries. The internet-based diary provided time-stamped entries; entries were flagged if they were completed more than 3 hours before or after the reported wake time, in order to assess participant compliance. Less than 6% of all entries were flagged.

Psychophysiological stress protocol—Participants were instructed to abstain from caffeinated beverages, tobacco products, and exercise for three hours before the

psychological stress protocol, and they avoided alcohol for 12 hours/the night before the protocol. The start time of the protocol ranged from 8:30 am – 3:00 pm, dependent upon participant availability; 80% of participants started the session before 1:30 pm. After arriving at the laboratory, participants were seated in a comfortable chair and instrumented for physiological recording. This included electrocardiogram (ECG) recordings using a modified lead II configuration to measure heart rate and HF-HRV, a standard blood pressure cuff with an automated monitor (GE Dinamap V100/Milwaukee WI), and a respiratory belt (Respiration Monitor Belt, Vernier, Beaverton, OR, USA). The blood pressure cuff was placed over the brachial artery on the nondominant arm, and the automated monitor was placed in the control room so that readings could not be observed by the participant. A respiratory belt measuring pressure changes corresponding to thoracic expansion and contraction was placed on the chest between the fifth and eighth ribs, as described by Egizio, Eddy, Robinson, and Jennings (2011). The band was wrapped tightly enough to allow only the experimenter's index and middle fingers to fit underneath, and the pressure of the belt was adjusted to approximately 100 kPa using an air bladder.

After physiological recording equipment was arranged, participants sat quietly and watched a nature video for a 10-minute rest period in order to minimize movement and boredom, and as recommended by Piferi, Kline, Younger, and Lawler (2000). Baseline blood pressure readings were taken at Minutes 4, 6, and 8 of the baseline period, while heart rate and respiration were monitored continuously. After the baseline period, participants completed two blocks of tasks. The first block included two cognitive computer tasks: the Stroop color-word interference task (four minutes) and the multi-source interference task (four minutes) (Bush, Shin, Holmes, Rosen, & Vogt, 2003). Both of these tasks were performance-titrated at a 60% accuracy level in order to minimize individual differences in task performance, which may be related to sleep (Durmer & Dinges, 2005). In the Stroop task, participants identified the color of a target word that briefly appeared in the center of a monitor by selecting one of four identifier words displayed at the bottom of the monitor. Response selection was made using a standard computer keyboard; the keys corresponded with the placement of the identifier words displayed on the monitor. The target word always appeared in a color incongruent with the target word, and all of the identifier words appeared in incongruent colors as well (i.e., the target word “blue” may have appeared in red font). In the multi-source interference task, three numbers were briefly displayed in the center of the monitor, and participants were asked to identify the target number that was different than the other two numbers. Response selection was again made using a computer keyboard. The target number always appeared in a position that was incompatible with its spatial position on the keyboard. As an example, if the target number “2” appeared in the *middle* spot among three numbers, participants would need to suppress the tendency to select the *middle* button on the computer keyboard, and instead select the key that displayed the number “2” on it. In the second block of tasks, participants were instructed to prepare and deliver a speech in which they defended themselves from a theoretical traffic violation in front of a video camera. This block included four minutes of speech preparation followed by four minutes of speech delivery. Participants were told that the speech would be recorded and later evaluated for clarity, persuasiveness, and style, and they were instructed to continue talking for the entire four-minute period. The two cognitive tasks always occurred first, in a counter-balanced order, followed by the speech task. Each of the cognitive tasks was followed by a five-minute recovery period in which participants rested quietly, and the speech task was followed by a final seven-minute recovery period. During each of the three recovery periods, participants appraised the previous task on three dimensions (*valence*, *arousal*, and *control*) using a Likert-type scale. Ratings for each dimension were averaged across the computer and speech tasks. Three blood pressure readings were taken at two-minute intervals during both task and recovery periods, and heart rate was monitored continuously throughout the protocol.

Physiological data processing—The ECG signal was digitized (12 bit), sampled at 500 Hz, and stored for off-line processing. QRS waves were identified using “PhysioScripts,” a collection of publically available scripts used for processing physiological data (Christie & Gianaros, 2012), implemented in the R computer environment (Ihaka & Gentleman, 1996; R Development Core Team, 2010). Processing steps included QRS detection, using an algorithm that detects the amplitude of the digitally filtered ECG waveform as well as its first derivative, followed by interbeat interval (time in milliseconds between successive R-spikes) extraction and artifact detection. Epochs with greater than or equal to 20% artifact were excluded (1% of all epochs were excluded). Estimates of HF-HRV were then derived using the band-limited variance method, with high-frequency band cut-offs of 0.15 to 0.40 Hz (Allen, Chambers, & Towers, 2007). HF-HRV values were natural log transformed before use in analysis. Respiration was processed using a custom algorithm. Briefly, the respiratory waveform was bandpass filtered (.05 - .5 Hz, 10 sec Hamming window) and local maxima (inspirations) and minima (expirations) were identified within a specified time window based on the shortest expected respiratory period (2 sec or .5 Hz). Unbalanced inspirations and expirations (e.g., two inspirations with no intervening expiration) were then corrected by removing the member of the paired values with the lesser absolute magnitude (i.e., the smaller inspiration or the larger expiration). The respiratory variable used as in analyses of HF-HRV was mean respiratory rate, defined as the inverse of the mean of all periods between inspiratory peaks (alternative analyses run with peak respiratory frequency produced identical results).

Cardiovascular stress responses—Baseline levels were calculated for each cardiovascular parameter. For heart rate and HF-HRV, values were averaged across the final seven minutes of the baseline period; for systolic and diastolic blood pressure, readings taken at Minutes 4, 6, and 8 of the baseline period were averaged. Reactivity estimates were calculated for heart rate and HF-HRV by averaging values measured continuously throughout the four minutes of each task; for blood pressure, readings taken at Minutes 1 and 3 of each task were averaged. Recovery levels were calculated for heart rate and HF-HRV by averaging values measured continuously throughout the five minutes after each task (seven minutes after the final speech task); for blood pressure, readings taken at Minutes 1, 3, and 5 following each task were averaged.

Because reactivity and recovery values were correlated across tasks, principle components analysis was applied to evaluate whether each cardiovascular measure seemed to be assessing a common underlying construct across tasks or whether the measures were task specific. Factor analysis was performed separately for HR, HF-HRV, and blood pressure reactivity and recovery. The Kaiser-Meyer-Olkin measure of sampling adequacy was above the recommended value of .6, and Bartlett’s test of sphericity was significant ($p < .05$) in all cases. Results of the factor analysis revealed one factor each for HR, HF-HRV, and blood pressure reactivity and recovery, with loadings of .7 or higher, eigenvalues ranging from 1.9 to 3.5, and explained variance above 59% (59% - 97%) for each factor. As these data suggest strong communality across tasks, we chose to aggregate stress responses across tasks as described below. Aggregating across stress tasks is also suggested in order to increase reliability and generalizability of estimates (Kamarck, Jennings, & Manuck, 1993; Kamarck & Lovallo, 2003).

In order to create cardiovascular stress response variables for use in analysis, we first calculated one standardized residual for each cardiovascular parameter (heart rate, HF-HRV, systolic blood pressure, diastolic blood pressure) using linear regression to predict the task average from the baseline average. These residuals thus represent the change in each cardiovascular parameter from baseline to task, controlling for baseline values. Task HF-HRV was additionally regressed on task respiration rate to remove variance due to

breathing. Standardized residuals were then averaged across the Stroop, MSIT, and speech preparation and delivery tasks to produce four composite indices of reactivity: one each for heart rate, HF-HRV, systolic, and diastolic blood pressure. Similarly, mean recovery scores were calculated for each cardiovascular parameter using linear regression to predict the recovery average from the baseline average and the task average. Recovery HF-HRV was additionally regressed on recovery respiration rate to remove variance due to breathing. Standardized residuals were then averaged across the Stroop, MSIT, and speech preparation and delivery tasks to produce four composite indices of recovery: one each for heart rate, HF-HRV, systolic, and diastolic blood pressure. Thus, outcomes included one reactivity index and one recovery index for each of the four cardiovascular parameters.

Demographics, body mass index, and health habits—Participants self-reported their age and race/ethnicity. BMI was based on height and weight measurements taken in the laboratory and was calculated by dividing body weight in kilograms by height in meters squared. Participants reported daily number of caffeine servings and cigarette use, among other variables, in an evening diary during the study period. Average daily caffeine intake and cigarette use were averaged across the seven days of the study.

Procedure

The University of Pittsburgh Institutional Review Board approved the study. General study procedure consisted of two in-person sessions, separated by a week of actigraphy monitoring. During the first session, participants completed a battery of psychosocial, health, and sociodemographic questionnaires. BMI and resting blood pressure were measured, and actigraphy and diary use were explained in detail. Participants then wore a wrist actigraph and completed sleep diaries for 7 nights. The actigraph was worn for an additional night(s) if the participant's schedule did not permit returning to the laboratory in exactly one week. After wearing the actigraph for at least 7 nights, participants returned to the laboratory to complete the psychophysiological stress protocol. Participants confirmed that they had not drunk alcohol within the previous 12 hours and that they had not used tobacco products, used caffeine, or exercised within the previous three hours before testing was started.

Statistical Analysis

Associations between actigraphy-assessed total sleep time and cardiovascular stress responses were examined in a series of linear regression models. Each reactivity and recovery index was tested as an outcome in a separate model. The first step of the model included total sleep time and was additionally adjusted for age, race, BMI, and daily caffeine and nicotine use. As task appraisals may be one pathway through which sleep is associated with cardiovascular stress responses, ratings of task valence, control, and arousal were included as covariates in the second step of the model as potential mediators. In the third step, analyses were adjusted for actigraphy-assessed naps to determine whether associations between total sleep time and stress responses were independent of daytime sleep. Naps were also tested as moderators of sleep duration in a set of analyses examining the cross-product of nighttime sleep duration and daytime napping.

Results

Sample Characteristics

Of the eighty male undergraduates that completed the protocol, one was excluded from analyses due to a reported history of heart murmur and an inability to detect R waves on his ECG. Thus, the final sample included 79 participants. Sample characteristics are shown in Table 1. The average age of participants was just over 19 years. Eighty percent self-

identified their race/ethnicity as “White or Caucasian,” 11.4% identified as “Asian” or “Pacific Islander,” and 8.9% identified as “Black or African American.” Mean actigraphy-assessed total sleep time was just over 6 hours per night. Mean sleep quality as reported in the morning sleep diary was 3.4 on a 1 to 5 Likert-type scale with “1” indicating “very bad” sleep quality and “5” indicating “very good” sleep quality. Based on resting blood pressure at the initial session, one participant met criteria for hypertension (≥ 140 mmHg systolic pressure, ≥ 80 mmHg diastolic pressure). Results of all statistical analyses were unchanged when this participant’s data were removed. We therefore include these data in analyses.

Cardiovascular Stress Responses

Mean baseline, task, and recovery values for heart rate, HF-HRV, and blood pressure are shown in Table 2. All cardiovascular parameters changed significantly from baseline to stress tasks ($ps < .01$). Heart rate, systolic blood pressure, and diastolic blood pressure remained elevated during task recovery periods relative to baseline ($ps < .01$). HF-HRV recovery values were not significantly different than baseline values ($t(78) = .59, p = .56$). None of the reactivity or recovery indices correlated with laboratory session start time ($ps > .10$).

Total Sleep Time and Cardiovascular Stress Responses

Results from linear regression models examining associations among actigraphy-assessed total sleep time and cardiovascular reactivity and recovery are shown in Table 3.

Heart rate—Total sleep time was not associated with heart rate reactivity. Shorter time spent asleep was associated with poorer heart rate recovery, accounting for about 5% of the variance (Figure 1). Follow-up analyses showed that time spent asleep was associated with heart rate during the first two minutes of the recovery period ($\beta = -.26, p = .03$) but was not associated with heart rate during the final two minutes of recovery ($\beta = -.20, p = .11$). Associations between total sleep time and heart rate recovery were similar for the cognitive ($\beta = -.23, p = .07$) and the speech ($\beta = -.25, p = .05$) tasks. Adjustment for ratings of task valence, arousal, or control did not alter the association between time spent asleep and heart rate recovery.

High-frequency heart rate variability—Shorter actigraphy total sleep time was related to a greater reduction in HF-HRV during stress tasks; sleep time accounted for approximately 7% of the variance in HF-HRV reactivity (Figure 2). In follow-up analyses, shorter time asleep was associated with a greater reduction in HF-HRV during cognitive tasks ($\beta = .33, p = .005$) but was not related to change in HF-HRV during the speech preparation and delivery tasks ($\beta = .18, p = .15$). Time spent asleep was not related to HF-HRV during recovery periods. These associations remained similar after models were adjusted for ratings of task valence, arousal, or control.

Blood pressure—Actigraphy-assessed total sleep time was not associated with systolic or diastolic blood pressure reactivity, nor was it associated with systolic blood pressure recovery. Shorter time spent asleep was associated with poorer diastolic blood pressure recovery, accounting for approximately 6% of the variance (Figure 3). Shorter time spent asleep was associated with higher diastolic blood pressure after cognitive tasks ($\beta = -.29, p = .01$) but not after the speech task ($\beta = -.16, p = .20$). These associations remained similar after models were adjusted for ratings of task valence, arousal, or control.

Actigraphy total sleep time on the night prior to psychophysiological protocol—Total sleep time as assessed by actigraphy on the night before the laboratory session was not associated with any of the cardiovascular stress reactivity (HR: $\beta = .08, p = .64$; HF-

HRV: $\beta = -.02, p = .90$; systolic blood pressure: $\beta = -.15, p = .40$; diastolic blood pressure: $\beta = -.09, p = .64$) or recovery parameters (HR: $\beta = .02, p = .92$; HF-HRV: $\beta = .03, p = .86$; systolic blood pressure: $\beta = -.01, p = .97$; diastolic blood pressure: $\beta = .18, p = .28$).

Naps—Seventy-eight percent of participants took at least one nap throughout the study period. Actigraphy total sleep time and actigraphy-detected naps were correlated at $r = -.4, p < .001$. Adjusting for actigraphy-assessed daily nap minutes did not change the association of total sleep time with HF-HRV reactivity ($\beta = .29, p = .03$) or the association of total sleep time with diastolic blood pressure recovery ($\beta = -.26, p = .05$). Adjustment for nap minutes reduced the association between total sleep time and heart rate recovery to non-significance ($\beta = -.22, p = .10$). Longer daily nap minutes were associated with higher heart rate recovery before adjusting for nocturnal total sleep time ($\beta = .23, p = .05$); however, this association also was attenuated when both daytime naps and nocturnal sleep time were included in the same model ($\beta = .16, p = .20$). The cross-product of nighttime sleep and daytime naps was not associated with any of the cardiovascular reactivity or recovery outcomes ($ps > .20$).

Discussion

This study examined the hypothesis that shorter actigraphy-assessed total sleep time over one week would be related to increased and prolonged heart rate, HF-HRV, and blood pressure responses to stressful laboratory tasks in healthy, undergraduate men. Shorter actigraphy-assessed total sleep time was associated with greater reductions in HF-HRV during stress tasks, and with elevated diastolic blood pressure following stress tasks. Shorter actigraphy-assessed sleep also was related to elevated heart rate following stress tasks; however, this association was no longer significant after adjusting for daytime naps. Actigraphy-assessed total sleep was not related to heart rate reactivity or blood pressure reactivity, nor was it related to HF-HRV recovery after tasks. Thus, hypothesized relationships among actigraphy-assessed sleep and cardiovascular stress responses were only partially supported by the data.

One pathway through which sleep duration may be related to cardiovascular stress responses is through perceptions of challenge or arousal related to stressful stimuli. Statistical adjustment for task appraisal ratings, however, did not affect observed associations. Previous work shows that correlations between cognitive task appraisals and cardiovascular reactivity range from weak to moderate (Feldman, Cohen, Lepore, Matthews, Kamarck, & Marsland, 1999; Gianaros, Sheu, Remo, Christie, Critchley, & Wang, 2009; Maier, Waldstein, & Synowski, 2003), suggesting that other factors influence the stress response. For instance, resting regional cerebral blood flow in corticolimbic areas has been associated with cardiovascular reactivity, independent of reports of stress (Gianaros et al., 2009). Thus, the possibility that shorter actigraphy-assessed sleep is associated with heightened cardiovascular stress responses via altered neural processes, independent of perceptions of stress, cannot be ruled out based on the current data.

Actigraphy assessment of total sleep time was not related to systolic blood pressure responses to stress, but shorter sleep duration was associated with higher diastolic blood pressure during stress recovery. These findings are somewhat in contrast to results recently reported by Franzen and colleagues (2011); in that study, one night of experimental sleep deprivation resulted in increased systolic pressure reactivity to a laboratory speech task in healthy young adults. Different results may be due to the fact that Franzen et al.'s study demonstrated an effect of acute, extreme sleep loss, while the present study examined naturally occurring variation in sleep duration that may possibly reflect a more chronic pattern of behavior. How the severity or chronicity of sleep loss might differentially affect

blood pressure reactivity versus recovery, however, is not known. The literature linking delayed blood pressure recovery to cardiovascular outcomes is stronger and more consistent for systolic, rather than diastolic, blood pressure recovery (Chida & Steptoe, 2010). Therefore, the degree to which prolonged blood pressure recovery from stress contributes to the development of progression of cardiovascular disease among those with short sleep remains unclear.

Shorter actigraphy total sleep time was associated with greater reductions in HF-HRV during psychological stress. This finding is generally consistent with Palesh et al.'s 2008 study, in which decreased sleep continuity assessed by actigraphy was related to lower overall respiratory sinus arrhythmia during the Trier Social Stress Test in women with breast cancer, as well as with animal literature showing that experimental sleep deprivation leads to reduced vagal antagonism during stress responses (Sgoifo, Buwalda, Roos, Costoli, Merati, & Meerlo, 2005). One study in 6-to-12-year-old children, however, reported contradictory results, such that actigraphy-assessed shorter sleep was associated with *less* vagal withdrawal during a reaction time task (El-Sheikh & Buckhalt, 2005).

Diminished HF-HRV at rest or during clinic visits is associated with progression of coronary artery calcification and an increased risk for cardiac events (Tsuji et al., 1996; Rodrigues, Ehrlich, Hunter, Kinney, Rewers, Snell-Bergeon, 2010); however, the long-term cardiovascular implications of vagal withdrawal during stress are less clear. Gianaros et al. (2005) reported that a greater reduction in HF-HRV during preparation for a speech task was related to more extensive coronary artery and aortic calcification in a cross-sectional study, and Matthews et al. (2003) reported that lower mean successive differences in interbeat intervals during laboratory stress tasks predicted increases in diastolic blood pressure over 3 years in children and adolescents. Steptoe and Marmot (2005) also demonstrated that lower HRV (assessed by the root mean square of successive difference in R-R intervals) during stress tasks predicted increases in diastolic blood pressure over 3 years in middle-aged men and women free from cardiovascular disease and hypertension. In contrast, Heponiemi et al. (2007) observed a link between greater decreases in respiratory sinus arrhythmia during stress tasks and lower carotid intima-media thickness two years later. Thus, data from the current and previous studies are generally consistent with stressor-evoked reductions in vagal activity being a pathway connecting sleep disruption and cardiovascular risk, but more data are needed to support a link between vagal withdrawal during stress and cardiovascular outcomes.

Prolonged heart rate recovery following mental stress is a predictor of subsequent cardiovascular risk, with outcomes consisting of increased intima-media thickness (Heponiemi et al., 2007) and blood pressure (Stewart, Janicki, & Kamarck, 2006) across 2-3 year follow-ups. In the present study, shorter actigraphy total sleep time was related to higher heart rate during task recovery, suggesting that either the process by which heart rate is restored after a challenge is impaired among individuals with short sleep, or that these individuals have higher levels of anticipatory arousal before a challenging task. Follow-up analyses supported the former hypothesis, as the association between sleep time and heart rate was significant during the two minutes following task completion but not during the two minutes before an upcoming task. However, these data should be interpreted with caution, given that they are based on only a two-minute interval. It is unclear why there was a relationship between sleep and HF-HRV reactivity -but not heart rate reactivity, and between sleep and heart rate recovery - but not HF-HRV recovery. Heart rate and HF-HRV changed throughout the stress protocol in an inverse fashion; however, the correlations ranged from $r = -.3$ to $-.5$, suggesting that the two constructs are not purely bipolar and likely have differing autonomic origins. While HF-HRV is believed to primarily reflect vagal efferent activity to the heart, increases in heart rate may be a function of increased

sympathetic activity, decreased parasympathetic activity, or combinations of activation and/or inhibition of both branches (Bernston, Cacioppo, & Quigley, 1993). Using an autonomic blockade design, Imai et al. (1994) showed that at two minutes post-exercise, there are both parasympathetic and sympathetic contributions to heart rate. In the current study, the fact that total sleep time was associated with heart rate recovery at two minutes post-stress, but not with HF-HRV recovery, may possibly suggest that short sleep duration is more closely linked to a dysregulation of sympathetic, rather than parasympathetic, recovery. A more precise measure of sympathetic cardiac influence would be needed to directly support this hypothesis.

Naps may possibly act as a buffer or moderator of sleep duration, negating or lessening the associations between shorter nighttime sleep and increased cardiovascular activity. Analyses examining the interaction of nocturnal sleep and napping, however, were not related to any of the reactivity or recovery outcomes in the current study. Instead, the relationship between actigraphy-assessed total sleep time and heart rate recovery was no longer significant after adjusting for daily actigraphy-detected nap minutes. As nocturnal sleep and daytime naps were inversely correlated, and as neither variable was associated with heart rate recovery when forced into the same model, it is likely that the two variables accounted for overlapping variance in heart rate recovery. Longer naps may disrupt nighttime sleep, and shorter nighttime sleep may lead to increased daytime napping (Owens et al., 2010). Thus, the cooccurrence of shorter nocturnal sleep and increased daytime naps may reflect an underlying dysregulation of the sleep cycle. The literature on napping and cardiovascular risk has been mixed, with some epidemiological studies reporting a protective effect (Kalandidi et al., 1992; Naska, Oikonomou, Trichopoulou, Psaltopoulou, & Trichopoulos, 2007) and others reporting elevated risk associated with naps (Stone et al., 2009; Tanabe et al., 2010). Using an experimental design, Brindle and Conklin (2011) demonstrated that a longer daytime nap was associated with accelerated blood pressure recovery following mental stress, relative to a shorter nap in university students. Disentangling the potential negative versus positive effects of napping on the cardiovascular system, especially in the context of inadequate sleep, warrants further investigation.

As only undergraduate men participated in this study, results may not generalize to women, or individuals of different ages and socioeconomic backgrounds. There are gender differences in sleep, such that women report poorer sleep quality than men but have longer, more efficient sleep as assessed by actigraphy and polysomnography (Goel, Kim, & Lao, 2005; Lauderdale et al., 2006). Moreover, there are mixed data on whether the links between sleep characteristics and cardiovascular outcomes differ by gender, with some studies reporting stronger effects in men (Mallon, Broman, & Hetta, 2002; Rod et al., 2011) and others reporting stronger effects in women (Cappuccio et al., 2007; Meisinger, Heier, Lowel, Schneider, & Doring, 2007). Thus, it will be important for future studies to examine whether similar relationships between short sleep and stressor-evoked cardiovascular responses exist in women. Our sample size also was somewhat small relative to the number of predictors examined, which may further limit the reliability or generalizability of results.

The data are consistent with a model in which chronic sleep deprivation affects the cardiovascular system, and there is some evidence showing that experimentally shortening sleep influences stressor-evoked cardiovascular responses (Brindle & Conklin, 2011; Franzen et al., 2011). It is just as plausible, however, that heightened cardiovascular reactivity results in shorter sleep duration, or that shorter sleep, reduced vagal activity, and prolonged stress recovery are all indicators of underlying autonomic dysregulation or increased arousal. The study was limited in its ability to fully delineate the autonomic processes underlying heart rate and blood pressure responses to stress. A continuous measure of blood pressure would allow for examination of beat-to-beat changes and

variability in the pressor response, and assessment of cardiac output and total peripheral resistance would delineate myocardial versus vascular activation. Future work that combines experimental sleep manipulation – either sleep extension or partial sleep restriction - with more comprehensive measures of cardiovascular reactivity and recovery will help clarify whether sleep is causally related to cardiovascular stress responses and elucidate relevant underlying autonomic processes.

There were both strengths and drawbacks to studying college students. The fact that all study participants were young and free from cardiovascular disease, and nearly all were free from hypertension, decreased the potential of underlying disease influencing cardiovascular stress responses. College students tend to have relatively flexible, self-determined schedules, characterized by frequent daytime naps and irregular sleep patterns (Kloss, Nash, Horsey, & Taylor, 2010). On the one hand, this may have allowed for more naturally occurring variation in sleep between individuals and an opportunity to study the effects of naps; on the other, it is possible that participants were able to sleep in later and minimize sleep debt. Examining a sample that is more likely to be mildly sleep deprived due to environmental demands (i.e., working adults) might reveal stronger associations between sleep duration and cardiovascular stress responses. Similarly, it is unknown what proportion of participants sleeping for shorter durations in this study were “natural” short sleepers versus physiologically sleep deprived. There is evidence that individuals vary in their biological sleep need (Tucker, Dinges, & Van Dongen, 2007; Van Dongen, Baynard, Maislin, & Dinges, 2004), and results may have different implications for an individual who needs 8 hours of sleep but is getting 6 hours, than for an individual with a biological sleep need of 6 hours. Future work should also consider chronobiological factors when studying sleep and cardiovascular stress responses. For instance, individuals’ circadian preference, or morningness versus eveningness, may affect both sleep duration as well as cardiovascular responses to stress, with some recent evidence suggesting evening types may be more reactive (Roeser et al., 2012). Finally, it should be noted that the speech task used in this study required participants to speak for four minutes, which may have influenced HF-HRV, in particular. We attempted to account for this effect by adjusting for respiration rate in statistical analyses.

In sum, prior data has demonstrated that sleep deprivation has an activating effect on the body’s autonomic stress system, as characterized by elevations in heart rate and blood pressure, and decreases in parasympathetic cardiac influence (Meier-Ewart et al., 2004; Spiegel, Leproult, & Van Cauter, 1999; Zhong et al., 2005). Findings from the current study extend prior work to demonstrate that shorter actigraphy assessments of naturally occurring sleep duration across one week are related to greater reductions in HF-HRV during psychological stress, as well as poorer heart rate and diastolic blood pressure during stress recovery, in healthy, male college students. Sleep duration was not associated with heart rate or blood pressure reactivity, or with HF-HRV recovery following stress. Continuing to combine findings from experimental studies that manipulate sleep duration with observational studies of normative sleep patterns may help uncover the mechanisms underlying the relationship between short sleep duration and cardiovascular disease.

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Figure 1.

Recovery heart rate, in units of unadjusted beats per minute change from baseline, averaged across task, and displayed by tertile of actigraphy-assessed total sleep time. Standard errors are represented by the error bars.

Figure 2.

High-frequency heart rate variability, in unadjusted ln units change from baseline, averaged across task, and displayed by tertile of actigraphy-assessed total sleep time. The error bars represent standard errors.

Figure 3.

Recovery diastolic blood pressure, in units of unadjusted mmHg change from baseline, averaged across task, and displayed by tertile of actigraphy-assessed total sleep time. The error bars represent standard errors.

Table 1

Participant Characteristics

<u>Variable</u>	<u>Mean</u>	<u>Range</u>
Age	19.34 (2.02)	18.0 – 29.0
Body Mass Index	24.0 (3.22)	18.19 – 31.83
Resting Systolic Blood Pressure (Visit 1)	120.42 (8.25)	104.33 – 141.33
Resting Diastolic Blood Pressure (Visit 1)	67.21 (6.85)	55.33 – 84.67
Daily Caffeine Servings	1.24 (1.22)	0 – 5.57
Daily Cigarettes Smoked	.49 (1.99)	0 – 11.57
Actigraphy-Assessed Total Sleep Time (Hrs)	5.78 (.82)	3.61 – 7.68
Actigraphy-Assessed Daily Nap Minutes	26.54 (26.78)	0 – 154.33

Mean (Standard Deviation) Heart Rate, High-Frequency Heart Rate Variability, and Blood Pressure Values throughout Laboratory Psychological Stress Protocol

Table 2

	Baseline	Stress	Stress Recovery
Heart Rate (beats per minute)	69.49 (10.09)	76.98 (10.15)	72.48 (8.99)
High-Frequency Heart Rate Variability (ln units)	6.33 (.91)	6.04 (.82)	6.28 (.84)
Systolic Blood Pressure (mm/Hg)	113.67 (8.11)	125.76 (10.65)	116.66 (8.33)
Diastolic Blood Pressure (mm/Hg)	62.77 (5.05)	72.34 (6.56)	64.44 (4.88)

Table 3

Standardized Coefficients from Linear Regression Models Examining Associations between Actigraphy-Measured Total Sleep Time and Residualized Cardiac and Blood Pressure Stress Responses

	Heart Rate		HF-HRV		Systolic Blood Pressure		Diastolic Blood Pressure	
	Reactivity β	Recovery β	Reactivity β	Recovery β	Reactivity β	Recovery β	Reactivity β	Recovery β
Actigraphy Total Sleep Time	-.09	-.26*	.29*	.12	.10	.08	-.01	-.28*

* *Note.* $p < .05$. Analyses are adjusted for age, race, BMI, daily caffeine use, daily nicotine use; HF-HRV analyses are also adjusted for respiration rate.