CARDIOVASCULAR RESPONSES TO DAILY STRESSORS AS A FUNCTION OF HABITUAL SLEEP CHARACTERISTICS

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Submitted to the Graduate Faculty of

the Kenneth P. Dietrich School of Arts and Sciences in partial fulfillment

of the requirements for the degree of

Master of Science in Clinical and Health Psychology

University of Pittsburgh

2014

UNIVERSITY OF PITTSBURGH

THE KENNETH P. DIETRICH SCHOOL OF ARTS AND SCIENCES

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Experimental and epidemiological data suggest a link between sleep parameters and cardiovascular disease risk. Cardiovascular stress responses constitute one putative mechanism connecting the two. The present study sought to examine the independent and interactive effects of objective indices and self-reports of sleep on cardiovascular responses to daily stress. The study utilizes two days of ambulatory blood pressure data paired with daily diary assessments from 224 middle aged adults. Participants wore a wrist actigraph for nine nights from which sleep duration and efficiency measures were derived. They also completed a subjective sleep quality instrument. Diary ratings of momentary task demand and social conflict served as naturalistic stressors. Hierarchical linear models examined the moderating role of usual sleep characteristics on blood pressure (BP) and heart rate (HR) levels at times of high versus low stress, and at times following initiating stressor. Lower sleep efficiency was associated with greater average HR and heightened systolic and diastolic BP responses to social conflict. Poorer global sleep quality predicted greater BP and HR responses to demand. Worse sleep quality also predicted prolonged elevations in HR responses to demand. Associations were independent of age, race, body mass index, sex, apnea-hypopnea index, and factors affecting BP readings. In sum, middle-aged Black and White adults with lower actigraphy-assessed sleep efficiency exhibit greater BP responses to social conflict. Those with poorer sleep quality show exaggerated BP and HR responses as well as prolonged HR elevations to demand than those with better sleep quality.

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1.0 INTRODUCTION

The foremost purpose of the current study is to gain a clearer understanding of the effect of sleep on cardiovascular responses to daily assessments of psychosocial stress. First, the relationship between average ambulatory blood pressure (BP) and heart rate (HR) levels and sleep duration, efficiency, and global subjective sleep quality will be characterized, and a number of demographic and clinical variables will be examined as covariates. It is hypothesized that markers of inadequate (i.e. shorter, less efficient, poorer quality) sleep will predict greater average ambulatory cardiovascular (CV) responses. Despite the fact that both inadequate sleep and daily psychosocial stressors have been linked to sympathovagal imbalance, no studies have considered both of these factors in stressor-evoked blood pressure and heart rate responses in an ecological momentary assessment framework. Therefore, we next examine the interaction between daily psychosocial stressors (demand and conflict) and usual sleep characteristics (duration, efficiency, and global subjective sleep quality) for its association with ambulatory blood pressure and heart rate measurements taken at the time of stress ratings. The analysis then assesses the unique contributions of these average sleep parameters to cardiovascular responses at the time *following* stressor onset, to approximate cardiovascular recovery in the context of a field study.

The present study has several important implications, including an improved understanding of how duration, efficiency and subjective sleep quality are associated with

psychosocial stress and autonomic nervous system activity in everyday life. Importantly, self-identification of stressors allows for exploration of individual differences in stress perception between participants and varying magnitudes of stress appraisal within participants.

Taken together, two seemingly independent lines of research indicate that (a) shorter, more fragmented, poorer quality sleep contributes to and results in psychological distress and that (b) sleep curtailment augments CV responses to laboratory stressors. Yet, a gap exists in our understanding of the posited negative synergistic effects relating inadequate sleep to stressful daily experiences. Positive associations between metrics of less adequate sleep and stressor-evoked cardiovascular responses will offer preliminary support that the deleterious sleep-stress relationships suggested by experimental deprivation studies extend to ambulatory populations. Findings may also shed light on determinants of the phenotype characterized by exaggerated stress reactivity. Lastly, detected associations may further elucidate the mechanisms linking poor sleep to cardiovascular disease (CVD).

1.1 SLEEP DURATION

1.1.1 Self-reported duration, incident CVD and mortality.

Epidemiologic studies link self-reported short and long sleep duration with myocardial infarction, stroke and CHD (Ikehara, et al., 2009; Magee, Kritharides, Attia, McElduff, & Banks, 2012; Qureshi, et al., 1997). A meta-analysis of fifteen prospective studies indicated that self-reported short sleepers (defined here as ≤5-6 hours a night) carried greater risk of developing or dying of CHD and stroke than those sleeping ≥ 7-8 hours a night (Cappuccio, Cooper, D'Elia,

Strazzullo, & Miller, 2011). Notably, these studies lacked information on sleep continuity and sleep disordered breathing, the latter of which is a strong independent risk factor for CVD (Shahar et al., 2001). Furthermore, 40% of included participants in the reviewed studies were geriatric (>65 years old), limiting generalization to the middle-aged population. However, at least two other meta-analyses support an association between short sleep and increased risk for death (Cappuccio, et al., 2010; Gallicchio & Kalesan, 2009).

1.1.2 Self-reported duration and CVD risk factors

Subjective sleep duration is also associated with major CVD risk factors. Gangswich and colleagues' analysis of self-reported NHANES sleep logs linked short sleep and type II diabetes mellitus, obesity and hypertension (Gangwisch et al., 2006, 2007; Gangwisch, Malaspina, Boden-Albala, & Heymsfield, 2005). Their findings align with data from non-US samples (Magee et al., 2012). Graded relationships may exist with specific risk factors; one study showed increased hypertension risk with decreased sleep duration (Gottlieb, Redline, Nieto, Baldwin, Newman, Resnick, et al., 2006). Short and to a lesser extent long sleep predict poor cardiometabolic outcomes such as obesity, elevated BMI (Knutson & Van Cauter, 2008; Marshall, Glozier, & Grunstein, 2008; S. R. Patel & Hu, 2008) and the metabolic syndrome (Hall, Muldoon, Jennings, Buysse, Flory & Manuck, 2008). In the latter study, prevalence of the metabolic syndrome persisted only for self-reported short sleepers after adjusting for antihypertensive medication use.

In sum, results linking self-reported sleep duration to CVD risk factors, clinical events, and mortality suggest a curvilinear relationship that is inconsistently supported after accounting for potential confounders. Factors that may contribute to divergent findings include unmeasured

sleep disordered breathing or other chronic medical illness, psychosocial variables such as low socioeconomic status (exposure to ambient noise/light) or depressive symptomatology (hyper- or insomnia), and lack of more robust self-report or objective assessments that capture other important sleep features.

1.1.3 Objective sleep duration and CVD risk

Few studies have employed actigraphy or PSG to objectively measure sleep duration as it relates to cardiovascular outcomes. Among middle-aged adults, the Coronary Artery Risk Development in Young Adults (CARDIA) study of 495 black and white participants suggested that chronically reduced sleep duration and efficiency predicted higher systolic (SBP) and diastolic (DBP) blood pressure. Short sleepers carried 37% greater risk of developing hypertension in this sample (Knutson, Van Cauter, Rathouz, Yan, Hully, Liu, et al., 2009). Short sleep also predicted incident coronary artery calcification in 61 participants (King et al., 2008). Another study of adolescents found that those with actigraphy weeknight SE $\leq$85% were 3.5 times more likely to have pre-hypertension. Among healthy adults, short sleep (actigraphy) predicted greater carotid intimal media thickness (Nakazaki et al., 2012). Lastly, studies indicate that PSG measures of sleep efficiency relate to other CVD risk factors including prothrombotic and inflammatory markers (von Kanel et al., 2006; von Känel, Loredo, Ancoli-Israel, Mills, Natarajan, & Dimsdale, 2007).

1.2 SLEEP CONTINUITY

Studies assessing cardiovascular outcomes and sleep continuity, or the ease with which one initiates and maintains sleep, are far fewer than those for duration. Reports of objective measurement of sleep efficiency in relation to cardiovascular disease are fewer yet. Using data from Heart SCORE, Troxel and colleagues found that self-reported difficulty falling asleep, perceived unrefreshing sleep and loud snoring predicted metabolic syndrome development. After adjusting for AHI, only loud snoring predicted metabolic syndrome (Troxel et al., 2010). Self-reported difficulty with sleep onset and maintenance are also related to hypertension (Phillips & Mannino, 2007) and myocardial infarction (Meisinger, Heier, Lowel, Schneider, & Doring, 2007). Self-reported difficulty in sleep initiation rather than total duration has been linked with three times the relative risk of CVD mortality, independent of depressive symptoms and other major CVD risk factors (Mallon, Broman, & Hetta, 2002). Measures of sleep continuity appear to provide unique prognostic information. Adjusting for sleep disordered breathing may improve our understanding of sleep and cardiovascular risk relationships.

1.3 CARDIOVASCULAR STRESS RESPONSES: A MECHANISM LINKING SLEEP CHARACTERISTICS AND CVD

One mechanism by which short and fragmented sleep may give rise to CVD is through exaggerated and prolonged cardiovascular responses to stress. Menkes and colleagues (1989) operationally define cardiovascular reactivity as changes in SBP, DBP and HR from one's measured resting value in response to a psychological stressor. The cardiovascular reactivity hypothesis posits that repeated and exaggerated sympathetic nervous system responses lead to

cardiovascular disease over time (Krantz & Manuck, 1984) through increases in inflammatory markers, thrombotic plaque development and changes in vasculature elasticity, among other mechanisms. Since this time, theories suggest that prolonged recovery, defined as slow return to resting BP and HR levels after stressor exposure, also predicts pathophysiological arterial changes implicated in CVD (Linden, Rutledge & Con, 1998; Schwartz et al., 2003).

1.3.1 Acute CV stress responses and CVD

A number of prospective studies suggest that persons who show exaggerated cardiovascular reactivity to physical and mental stress are at heighted risk for cardiovascular disease. Chida and Steptoe's meta-analysis of cardiovascular responses to psychological laboratory stressors indicates that both reactivity to and recovery from psychological stressors predict future cardiovascular risk (2010). Far more studies examined the role of reactivity as compared to recovery (36 versus 5, correspondingly). Greater cardiovascular reactivity was significantly associated with future cardiovascular risk (Chida & Steptoe, 2010). Of the cardiovascular reactivity parameters measured, (including pre-ejection period, cardiac output, heart rate variability, myocardial ischemic response, SBP, DBP, and HR) only SBP and DBP predicted outcomes, namely incident hypertension and elevated SBP and DBP. BP did not reliably predict increases in IMT.

1.3.2 Persistent CV responses and CVD

Prolonged cardiovascular recovery, defined in experimental studies as above-baseline CV activation during the post-task recovery period, also engenders greater cardiovascular risk.

Recovery parameters most consistently associated with risk were prolonged elevations in HR and SBP, where risk was defined as greater carotid IMT and elevated SBP and DBP at follow-up (Chida & Steptoe, 2010). As compared to reactivity, prolonged recovery prospectively predicted each tested CV outcome, illustrating its potential prognostic value.

In total, studies indicate that greater cardiovascular reactivity and prolonged cardiovascular recovery constitute putative risk factors for cardiovascular disease. Cardiovascular response parameters that most consistently predict CVD risk are SBP and DBP. Evidence implicates HR only as measured in the context of cardiovascular recovery. Data supporting a link between HR and CV risk remains tenuous as compared to BP.

1.4 WHY MIGHT INADEQUATE SLEEP INFLUENCE CARDIOVASCULAR REACTIVITY AND RECOVERY?

1.4.1 Normal sleep

Normative changes in BP and HR occurring during consolidated sleep confer hemodynamic stability (Meerlo, Sgoifo, & Suchecki, 2008; Trinder, Waloszek, Woods, & Jordan, 2012; Wolk, Gami, Garcia-Touchard, & Somers, 2005). Increases in BP and HR occurring during REM resemble wakefulness, while activity becomes more slowed during NREM. As approximately 80% of nightly sleep is spent in NREM, sleep is a time of cardiovascular quiescence or restfulness coined the “cardiovascular holiday” (Trinder, 2007).

1.4.2 Effect of experimental sleep curtailment on average CV levels

Mixed findings related to sympathetic activation result from experimental sleep restriction and deprivation. For instance, short sleep leads to increases in BP and HR (Barnett & Cooper, 2008; Meier-Ewert et al., 2004), and greater sympathovagal balance as evidenced by reduced HF-HRV (Spiegel, Leproult, & Van Cauter, 1999; Zhong et al., 2005) in young adults. Of the identified studies measuring changes in average cardiovascular activity in response to total sleep deprivation (TSD), two found increased resting DBP following one night of total sleep deprivation (TSD) (Kato et al., 2000; Ogawa et al., 2003), and one found no difference following TSD (Pagani et al., 2009). However, small sample sizes (all n's≤18) comprised of predominantly of young (mean age = 26) males (two studies enrolled only men) preclude generalization of these results.

We are aware of two studies documenting ABP changes alongside sleep restriction. Lusardi and colleagues examined within-person differences in 24-hr ambulatory BP following a 5-hr restriction vs. 8-hr normal sleep. Elevated ambulatory SBP and HR occurred the day following restriction (Lusardi et al., 1996). Similarly, working-class Chinese men showed elevated next-day BP, HR, and urinary catecholamine excretion after sleeping four as compared to eight hours (Tochikubo, Ikeda, Miyajima, Ishii, 1996). Both studies suggest greater sympathetic activation following sleep loss but do not document daily stress levels.

1.4.3 Effects of experimental sleep deprivation on CV stress responses

To date, three studies have examined individual differences in laboratory stress responses after 24-hour total sleep deprivation compared to a night of "normal" sleep (Franzen, et al., 2011;

Kato, et al., 2000; Yang, Durocher, Larson, DellaValla, & Carter, 2012). Kato and colleagues had 8 middle-aged subjects (2 female) presented for observationally-confirmed TSD. After each sleep condition, participants underwent four separate two-minute stressors: one mental (serial subtraction) and three physical (isometric hand grip, cold pressor, maximal forearm ischemia). Investigators found no differences in CV stress reactivity or recovery between sleep conditions. Yet, elevated resting morning SBP (~ 4 mm Hg, $p<.012$) resulted after deprivation.

Franzen and colleagues (2011) used a similar study design to examine the effects of one night of TSD on CV parameters in 20 young adults (mean age = 23 years; 55% female). TSD increased resting SBP, but acute lab stressors did not induce greater CV reactivity. No differences to the 10-minute Stroop task occurred, but participants were more SBP reactive to a speech task (5 min prep; 5 min delivery) under deprivation and tended to be more DBP reactive. No differences in post-task recovery were observed. This work suggests that individuals may be more sensitive to stressors with a social-evaluative component following sleep loss.

One explanation for null reactivity findings may rest in the tasks used; BP reactivity effect sizes for this version of the Stroop are modest at best (Gianaros et al., 2009). Given individual differences in BP reactivity, larger sample sizes may be needed to detect interactive effects of stress and sleep deprivation. Other sleep researchers suggest that TSD itself may impose stress that operates above-and-beyond the effects of conventional laboratory stressors, reducing the perception of task-related stress (see Meerlo et al., 2010). This may be especially true among participants with healthy usual sleep patterns. Furthermore, the effects of more normative sleep loss (as seen in habitual short or less efficient sleepers) remain unknown.

Another study of healthy young adults (mean age 22; n=28) (Yang, et al., 2012) measured the effect of TSD on cold pressor test (CPT) and mental arithmetic using actigraphy to

ensure compliance. This may have mitigated “first night effects” associated with acclimating to the sleep lab environment. TSD elicited greater HR reactivity to and prolonged recovery from the CPT relative to the rested condition. Notably, reactivity differences to the math task were detected three minutes into the serial subtraction task, suggesting that the length of exposure to mental stress may be important.

Divergent results among these three studies can be attributed to a number of factors. First, individual differences in stress response may “wash out” effects in samples sizes so small (e.g. 8 participants). Second, findings across studies cannot be easily compared due to differences in gender breakdown, stressor type (physical vs. mental) and deprivation environment (in-home vs. in-lab). Third, none of these studies controlled for order effects, namely the order in which subjects encountered TSD. In short test-retest frames, learning effects are sizable. Being exposed to stressors while well-rested may confer benefits that allow participants to better adapt to the second exposure of stress protocols.

1.4.4 Effect of typical sleep on stress responses

To date, only one study has examined the relationship between usual sleep characteristics and stress reactivity (Mezick et al., in press, 2013) using a matched-sample of 37 short and 42 "normal" sleeping undergraduate males. Sleep duration and efficiency were measured using actigraphy. Subjects underwent the Stroop, multisource interference and speech tasks while continuous HRV alongside stressor-evoked HR and BP were assessed. Greater HF-HRV withdrawal during stress tasks and higher HR and DBP during post-stress recovery were associated with shorter sleep duration. Greater HF-HRV withdrawal during stress and higher post-stress HR were related to lower sleep efficiency. Interestingly, elevations in post-stress

recovery disappeared after adjustment for daytime napping. The homogenous sample of young, college-educated white males limits inference to the general population.

1.5 LIMITATIONS OF THE EXISTING LITERATURE

Our knowledge of cardiovascular stress responses is based on experimental studies with small sample sizes. As reviewed above, these pose significant concerns to external validity. Field studies circumvent these limitations and have yet to examine sleep.

The above studies raise several concerns. First, laboratory stressors often do not duplicate the quality, duration or types of life stressors seen outside the laboratory. As such, they may fail to generate cardiovascular responses that reflect a participant's typical response profile. Second, the type of sleep deprivation invoked in experimental lab studies, whether partial or total, may not accurately depict the sleep inadequacy experienced by individuals on a nightly basis, accrued over time (i.e. chronic partial sleep restriction). Third, in-lab TSD is a stressor in and of itself (Meerlo, Sgoifo & Suchecki, 2008) whose independent effects on BP are not easily disentangled from stressor-evoked BP responses. Fourth, a body of literature suggests that ABPM is a more reliable predictor of CV risk than clinic or laboratory measures; by extension, repeated stressor-evoked BP measurements are more reliable than a single assessment of laboratory stress reactivity. Fifth, the negative effects of momentary stress are likely not restricted to a single incident. No studies have examined this in the context of sleep, as noted in the above review. However, outside the sleep literature, at least one ABPM study has found evidence for "carryover effects", noting elevated SBP and HR at the reading following psychosocial stress (Kamarck et al., 1998). In sum, little is known about characteristic sleep

patterns and their influence on stress responses. The concept that sleep may be associated with increased cardiovascular responses to daily stress is outlined in the embedded figure:

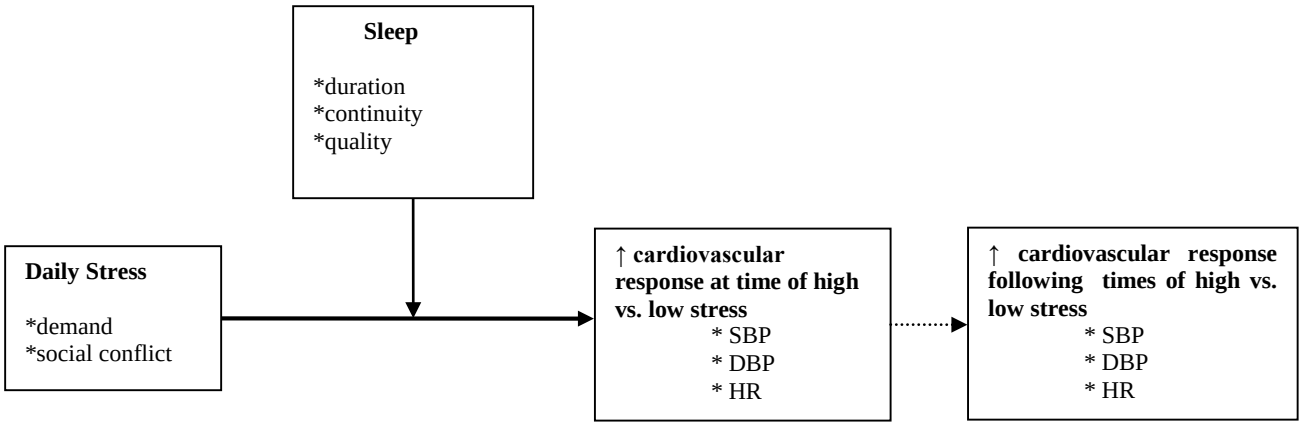

Figure 1. Proposed model of sleep's moderating role on CV responses to daily stressors

To address limitations of the existing epidemiologic and experimental literature noted above, we made use of field data to examine the relationship between stressor-evoked CV responses in the context of usual sleep patterns among 224 middle-aged African American and Caucasian adults recruited through Sleep SCORE. The study measured sleep through nine nights of actigraphy and two nights of in-home polysomnography (PSG). In addition, participants engaged in 48 hours of ambulatory blood pressure monitoring during which they rated measures of momentary stress (demand and social conflict) at each daytime blood pressure reading. The study collected objective as well as subjective sleep metrics, both of which predict CV risk. The objective measures used in this analysis include actigraphy-assessed sleep duration and efficiency; apnea hypopnea index (AHI) obtained from PSG serves as a covariate. The Pittsburgh Sleep Quality Index, a subjective rating of sleep, will also be examined.

The aims of this project were to examine the following hypotheses:

1. Those with less adequate sleep (defined as shorter duration, lower efficiency, or lower PSQI global sleep quality) show elevated cardiovascular parameters (SBP, DBP or HR) throughout the monitoring period.
2. At times of high relative to low stress (defined as conflict or demand), individuals with less adequate sleep show greater average cardiovascular response (increased SBP, DBP and HR) compared to those with more adequate sleep.
3. Following a more stressful event, individuals with less adequate sleep have greater average SBP, DBP and HR at time of next assessment than those with more adequate sleep.

2.0 METHOD

2.1 PARTICIPANTS

The Sleep SCORE sample is a cross-sectional cohort recruited from Heart SCORE, a larger community-based study examining cardiovascular risk factors and racial disparities in the Pittsburgh metropolitan area. The details of recruitment and enrollment have been reported elsewhere in detail (Aiyer et al., 2007). Briefly, at Sleep SCORE baseline eligible participants were aged 45–74 years. Exclusion criteria included pregnancy, treatment for severe sleep concerns (defined by nightly use of medications for a sleep disturbance and/or treatment of sleep apnea via continuous positive airway pressure treatment), insulin or oral diabetes medication use, and shift work. While including those with hypertension, the study protocol excluded individuals with past myocardial infarction, stroke, or interventional cardiology procedures.

Within this Sleep SCORE cohort, the mean age was 59.7 ± 5.2 years; half were female, and roughly half were Black, married, and had earned college degree or above. The Institutional Review Board at the University of Pittsburgh approved the study protocol and all study participants provided their written informed consent. Data collection for the parent study included demographics, medical history, anthropometrics, lipids/lipoproteins, physical activity, and psychological status. Enrollment in the parent study began on June 16, 2003 and concluded on October 11, 2006. Among the 224 originally in the daily diary study, nine were excluded

from analyses because of missing data for taking the BP cuff off for > 3 hours (n = 6), not completing daily diary assessments (>50% missing data) (n = 1), non-viable actigraphy data (n = 1), or multiple sclerosis symptoms (n = 1). Therefore, analyses involving sleep duration were based on final sample sizes of 215 individuals.

Please see **Table 1** for relevant sample characteristics. Please see **Figure 2** for a flow diagram of sample inclusion and exclusion in analyses.

2.2 RESEARCH PROTOCOL

Sleep SCORE enrollees were approached during Heart SCORE assessment visits. After receiving study details and providing informed consent approved by the University of Pittsburgh's Institutional Review Board, they were scheduled for a sleep study within three months. The study protocol lasted 10 days and involved actigraphy, polysomnography, and self-reported measures of sleep quantity and quality. During nights 1-9, subjects wore watch-like wrist actigraphy devices, which track rest and activity patterns through physical movement. Polysomnography data were obtained through in-home sleep studies conducted over nights 1 and 2 of the study. At the beginning of the sleep protocol, participants also completed the Pittsburgh Sleep Quality Index (PSQI), which is a 19-item self-rated questionnaire that evaluates subjective sleep quality over the previous month (Buysse et al., 2008). During days 3 and 4, ambulatory BP and HR were measured over a 48 hour period in 30 minute intervals during waking hours and 60 minute intervals during nighttime hours, described in greater detail below. Concomitant with daytime blood pressure readings, an electronic diary system was used to assess psychosocial stress (i.e. perceived demand and social conflict) as well as factors known to influence blood pressure.

2.3 PERSON-LEVEL COVARIATES

Anthropometric measures taking height and weight were used to calculate a Body Mass Index (BMI) for each participant. A clinic stadiometer measured height in centimeters with participants' shoes removed. Weight was taken using a clinic standard digital scale, using the same scale for each participant. Subjects were asked to self-report race. For analytic purposes, Asians (of whom there were 4) were combined with Whites. Participants reported diagnoses of primary hypertension and/or cardiac medication use at baseline assessment. Antihypertensives influence cardiovascular responses and may predict sleep quality and fatigue (Safar et al., 1987; Ko et al., 2002). Use of any reported cardiovascular medication was coded dichotomously as medicated vs. non-medicated. In-home polysomnography (PSG) was conducted on nights 1 and 2 of the study in order to obtain information on sleep stages (not used in the present analyses) and sleep-disordered breathing, termed Apnea Hypopnea Index (AHI). The monitor used was the Compumedics Siesta (Charlotte, North Carolina). On the first night of PSG, participants were monitored for sleep-disordered breathing using nasal pressure, inductance plethysmography, and fingertip oximetry. American Academy of Sleep Medicine Task Force standards were used to classify apneas and hypopneas from which the Apnea Hypopnea Index was derived.

2.4 SLEEP VARIABLES

Two additional methods were used to collect sleep data: actigraphy, and self-report. Actigraphs are watch-like activity monitors that are worn on the wrist to track rest and activity patterns via physical movement. They were worn throughout study duration (Nights 1 – 9) to provide a more representative sample of typical sleep/wake patterns than two nights of PSG alone. Diary reports completed on Nights and Mornings 1-10 assessed subjective sleep duration and were used in conjunction with actigraphy to track sleep and wake times throughout the study (Nights and Mornings 1-10). Participants self-reported global sleep quality on Day 2.

2.4.1 Actigraphy

Participants wore an Actiwatch-64 (Respironics, Inc., Bend, Oregon) on their non-dominant wrist, continuously for nine nights. Data were stored in 1-minute epochs and sleep parameters were estimated using validated MiniMitter software (Respironics, Inc.) algorithms. Each morning, participants recorded the amount they slept the previous evening (Matthews et al., 2008). Readings that fell within the participant's self-reported wake times were confirmed via a separate self-report diary and were used to code actigraphy data. The two variables considered in analyses are **total sleep time (TST**; actual sleep time excluding periods of wakefulness during the night), and **sleep efficiency (SE**; percentage of time in bed spent sleeping over total amount of time in bed). For each variable, values were averaged across the nine nights to obtain mean values used in analyses. Sleep efficiency was log-transformed [1 – natural log(value)], due to its

skewed distribution. All data reduction protocols followed established algorithms accompanying device software.

2.4.2 Pittsburgh Sleep Quality Index

The Pittsburgh Sleep Quality Index, administered on Night 2, is a 19-item self-report questionnaire assessing sleep over the past month that yields a global score ranging between 0 and 21. Higher scores indicate poorer sleep quality. The instrument has a sensitivity of 89.6% and specificity of 86.5% (kappa = 0.75, $p < 0.001$) distinguishing clinically referred patients from healthy controls (Buysse et al., 1989). The PSQI has an alpha coefficient of **.83** signifying high internal consistency, and a test-retest reliability of **.85** in distinguishing "good" from "poor" sleepers (Buysse et al., 1989). It has a test-retest reliability of .87 among those with primary insomnia (Backhaus et al., 2002). The clinical threshold for poor quality sleep is a global score greater than 5. The PSQI global score was used in analyses.

2.5 AMBULATORY ASSESSMENTS

A daily diary administered via an electronic Palm Pilot device asked participants to answer 16 questions at the time of each BP assessment. Items queried environmental factors known to influence ambulatory measurement of BP and HR, as well as psychosocial stressors.

2.5.1 Covariates affecting blood pressure and heart rate

At each BP assessment, participants rated *posture* (lying down, sitting or standing); *talking* at the time of BP assessment (yes/no); *substance intake* (yes/no to any of the following: food, nicotine, caffeine, or alcohol) within 30 minutes of BP reading; and level of *physical activity* (none, light, moderate, heavy; coded continuously as 0[none] to 4 [heavy]) within 10 minutes of BP reading. Blood pressure assessment-level covariates predicting one or more of the cardiovascular outcomes at a $p \leq .10$ were included in final models.

Two items comprised momentary *demand*, asking whether the activity that the participant engaged in at the time of blood pressure assessment required (i) working hard; and/or (ii) working fast. Items were coded 1-6 where 6 indicated the highest level of perceived demand. These two items were summed to create a single score for perceived demand, ranging from 2 (answering the lowest "1" in response to each question), to 12 (answering the highest "6" in response to each question). On average, participants rated demand 41 times (range 12 – 57) throughout the timeframe of measurement. 215 participants were included in these analyses. No one in this final analytic sample endorsed less than 5 demanding events.

Two items asked participants when they last interacted with someone (at time of blood pressure, 1-10 minutes before blood pressure reading, greater than 10 minutes before BP reading, or no one since last BP reading) and with whom they interacted (spouse/partner, other family or relative(s), other friend(s), coworker(s), or other(s)). Two additional items required participants to rate the quality of their most recent social interaction, on the same forced yes/no 1- to 6-point Likert scale. They queried (i) was [the last interaction] pleasant (ii) did someone treat you badly. The item on positive interaction was reverse-coded and summed with the negative item. This provided a score of perceived *conflict* from 2 to 12 (where 12 was the most negative). The total

number of interactions was computed by counting the number of times the participants indicated they were interacting with another person at the time of cuff inflation. Participants must have endorsed a minimum of five readings to be included in analyses. Nine participants did not have at least 5 interactions. The remaining 206 participants endorsed an average of 28 social interactions (range = 5 – 53) over the assessment period.

2.5.2 Ambulatory Blood Pressure and Heart Rate Monitoring

Ambulatory BP and HR were assessed using SpaceLabs model 90217 (Issaquah, WA) worn for ≥2 workdays and nights. They were programmed to take BP readings every 30 minutes during the day (from 8:00 AM to 9:00 PM) and hourly during the night (from 9:00 PM to 8:00 AM). Participants were told to wear the monitor at all times except when bathing. The self-report assessments completed each morning were used to identify waking BP data as participants started and ended their BP assessment at various times of day depending on work and home commitments. To be included, individuals had to have a minimum of 16 daytime readings across the measurement period.

Consistent with ABPM analysis methods put forth by Marler and colleagues (1988), BP readings were considered artifactual and were deleted if systolic blood pressure (SBP) was <70 mm Hg or >250 mm Hg and diastolic blood pressure (DBP) was less <45 mm Hg or >150 mm Hg. Furthermore, if heart rate (HR) readings were <40 or > 200 beats/min, they were considered artifactual. To ensure that analyses were performed on the same data, if one reading was deleted for a given time entry, the remaining HR or BP readings from that time point were also deleted. An average of 62.34 readings per person remained (SD = 7.61; range 20 - 68) that were accompanied by diary data (whole sample; irrespective of accompanying demand or conflict

endorsements). Following these data cleaning procedures, all cardiovascular outcomes were normally distributed.

2.5.3 Compliance with daily diary measures

Among the 215 included in the analytic sample, on average, 88.4% of data were accompanied by any diary data (meaning that participants answered at least one diary item 88.4% of the time that the cuff inflated and recorded viable BP readings). Of these, 81.6% (mean 41; range: 12-57) were accompanied by demand ratings.

93.4% endorsed having at least one social interaction. Of those individuals who endorsed being in an interaction, we saw 86.7% compliance, where they went on to rate questions on conflictual interactions. The average number of "conflict" ratings was 28 (range 5-53).

3.0 DATA ANALYTIC PLAN

3.1 PRELIMINARY ANALYSES

ANOVA with random effects models were used to determine whether cardiovascular response to demand as well as conflict varied across participants. For models where there was significant variability in cardiovascular response between participants, an additional model was constructed with random slopes and intercepts at the first level modeled as outcomes at the next level up. At level 1 (moment level), the between-moment variability in cardiovascular response for each individual was modeled as a function of demand or conflict and covariates influencing blood pressure assessment. An alpha level of 0.05 was used for all analyses.

Main analyses examined the variability in assumed sleep duration, efficiency and self-reported sleep quality. The intraclass correlation coefficient (ICC), a statistic for quantifying the relative magnitude of within and between-person variance components in a multi-level model, was calculated for sleep parameters by dividing between-person variance by total variance.

3.2 HYPOTHESES

3.2.1 Hypothesis 1: Main Effects

Individuals with less adequate (shorter, less efficient, more fragmented or lower PSQI global quality) sleep would show greater average cardiovascular parameters compared to individuals with more adequate sleep. This hypothesis was tested using Hierarchical Linear Modeling to allow for adjustment of within-subjects effects (modeled at Level 1) and between-subject effects (modeled at Level 2). More specifically, level 1 modeled CV outcomes, while adjusting for covariates known to affect ABPM including posture, talking, substance intake and physical activity level (referred to henceforward as moment-level predictors). Momentary stressors were not included in these analyses. A separate model was constructed for each CV outcome: SBP, DBP and HR. Between-subject covariates of race, Apnea Hypopnea Index (AHI), body mass index (BMI), gender and age served as level-2 covariates in the regression models. Sleep characteristics (TST, SE and global PSQI) were entered one at a time at the intercept level alongside the covariates mentioned above. Covariates were centered around the grand mean such that the intercept reflected the expected value of the cardiovascular outcome for a person whose score on the level 2 predictor variable equaled the sample mean. This manner of centering is consistent with methods in EMA and ABPM (see Blackwell, Mendes de Leon & Miller, 2011). Models were tested to determine whether a random or fixed slope best fit the data. A random intercept was postulated, such that levels of cardiovascular response could differ randomly among subjects. Model fit was assessed with full maximum likelihood estimation with the Akaike Information Criterion (Hox, 2002).

3.2.2 Hypothesis 2: Interactive effects of sleep and stress on momentary CV responses

At the time of high relative to low conflict (or demand), individuals with less adequate (shorter, less efficient, or lower quality) sleep would show greater average cardiovascular response (greater SBP, DBP, HR), as compared to individuals with more adequate sleep.

As described above, HLM was used to account for within-subject relationships of stress predicting CV response, while adjusting for moment-level BP assessment covariates when analyzing between-subject effects at level 2 (adjusting for person-level covariates and each sleep characteristic taken separately and in interaction with the stress measures). The difference between H2 models and H1 models rests in the introduction of the psychosocial stress variable. To test significant interaction, momentary stress predictors (e.g. conflict) were entered at level 1 and interacted with each sleep characteristic at level 2, to see if sleep characteristics alongside person-level covariates affected the slope of stress on CV response. This value is noted as β_3. The interaction term of interest is the sleep characteristic multiplied by stress variable slope (e.g. conflict*SE). The strength of this model is that it controls for person-level covariates affecting the intercept of CV response to stress (e.g. BMI), while adjusting for assessment-level characteristics (e.g. posture). It also assesses the interaction of sleep and stress (e.g. conflict*SE), adjusting for person-level characteristics (e.g. BMI). To summarize, momentary psychosocial stress predictors and moment-level ABPM covariates were nested within-subject while each sleep measure was examined as a between-subject moderator. Models were tested to determine whether a random or fixed slope would best fit the data. A random intercept was postulated, such that overall levels of cardiovascular response could differ randomly. Therefore,

momentary psychosocial stress predictors and relevant moment-level covariates were nested within-subject and each sleep measure was examined as a between-subject moderator.

3.2.3 Hypothesis 3: Interactive effects of sleep and stress on persistent CV responses

Following times of high relative to low conflict (or demand), individuals with less adequate sleep (shorter, less efficient sleep, or lower quality sleep) would show greater average HR, SBP and DBP.

In order to test for lagged effects, the same set of models described for the within-moment (acute CV response) analyses were repeated with time lagged one period out. BP and HR at the next time-frame of assessment, indicated as time 1 (e.g. SBP t_{1ij}), served as the dependent variable. To adjust for any novel stressors occurring in this new time-frame, stress levels at time 1 (e.g. demand $\mathbf{t1}_{ij}$) were statistically controlled for by adding time 1 stress as a predictor to the Level 1 model. Note that moderating sleep effects are tested using time 0 stress (e.g. demand $\mathbf{t0}_{ij}$). This allows us to test the effect of the initiating or initial stressor on later CV responses. Sample model specifications for lagged and acute responses are shown in **Appendix A**. Statistical interpretation of output is provided in **Appendix B**.

4.0 RESULTS

4.1 SAMPLE CHARACTERISTICS

Non-transformed demographic and sleep data appear in **Table 1**, based on the final 215 included in analyses.

Table 2 contains information on ABPM variables including SBP, DBP, HR, and relevant moment-level predictors. On average, the sample had elevated SBP of 130 mm Hg. This falls within the "upper normal" range. About a third of the sample met criteria for hypertension, a third exhibited high-normal blood pressure, and the remaining third was in normal ranges of blood pressure.

Stress endorsements decreased linearly throughout the day. Demand decreased at a rate of .03 units at each reading throughout the day ($p<.001$). Similarly, conflict decreased at a rate of .005 units throughout the day ($p<.001$). To adjust for these effects, a time-of-day variable was added to level-1 analyses.

4.2 RELATIONSHIPS BETWEEN SLEEP CHARACTERISTICS AND STRESS VARIABLES

Unconditional multi-level models were completed to test the relationship between each sleep characteristic and each stressor. Specifically, the outcome was the stressor, and the predictor was the sleep characteristic (averaged over the recording period for TST and SE) adjusted by age, sex, race, BMI and AHI. Models showed that average sleep duration was not related to *average levels* of demand (p = .38), or conflict endorsement (p = .48). Sleep efficiency also showed no relation to demand or conflict (all ps > .7). Lastly, global sleep quality was not related to ratings of demand (p = .73) or conflict (p = .60). Time-lagged models of conflict (or demand) the day before predicting sleep characteristics the following night, or sleep on night 1 (for example) predicting next-day endorsement of stress may show divergent findings; they will be examined in follow-up analyses with PSG variables.

Analyses on within-person relationship between stress predictors indicate that demand and conflict show a low to moderate within-person Spearman correlation (r = .31), suggesting that they are related, but not at a statistical threshold that would warrant combining them.

Unconditional models were explored to ensure a relationship existed between the sleep variable and CV outcome, prior to adding covariates to the model. Significant models (models for which we found significant evidence of a relationship) are presented in Appendix A.

4.3 HYPOTHESIS 1

Individuals with less adequate (shorter, less efficient, or lower quality) sleep would show greater average cardiovascular levels as compared to individuals with more adequate sleep.

We found that lower sleep efficiency predicted greater HR, but not BP (see **Table 4A**). Specifically, lower sleep efficiency predicted greater average HR over two days of ABPM. This persisted after adjustment for age, race, BMI, AHI and cardiovascular medication use (Beta = 4.07, $p<.05$; see line c in **Table 4A**). On the whole, Blacks had elevated DBP and HR as compared to Whites throughout the days of ABPM (Beta = 2.03, $p<.05$). Women had higher HR values than men on average, at times of ABP assessments (Beta = 1.57, $p<.05$). When examining effects by stressor type, we found that both Blacks and males had higher HR values at times of demand as compared to Whites and females (Beta = 3.14; Beta = 1.17, respectively; ps$<.05$). No significant race or sex effects were apparent for CV responses to conflict.

4.4 HYPOTHESIS 2

At the time of high relative to low conflict (or demand), individuals with less adequate (shorter, less efficient, or lower quality) sleep would show greater SBP, DBP and HR, as compared to individuals with more adequate sleep.

4.4.1 Sleep efficiency

Significant interactive effects were detected for BP responses to conflict as a function of sleep efficiency (See **Table 4C**). In other words, less efficient sleep predicted greater SBP responses to conflict (Beta = .93, $p<.01$; see line g). **Table 6** provides significant covariates and model output. Lower SE also predicted greater DBP responses at times of greater conflict. (Beta = .73; $p = .01$; **Table 7** displays model output). **Figure 3** illustrates the simple slope interaction plots for sleep efficiency and conflict predicting BP. Significant effects were observed when controlling for age, sex, race, BMI, and AHI. Results remained unchanged after adjusting for cardiovascular medication status.

A conservative approach to HLM interaction modeling is to control for person-level covariates not only at the intercept level, but also at the interaction level. The observed relationships increased in significance after controlling for person-level covariates at the interaction-level ($ps < .01$), meaning that the relationship of conflict and sleep efficiency on BP remained significant after controlling for the effects of age, race, BMI, gender, and AHI. Results for testing of simple slopes indicated that each slope was significantly different from 0, and regions of significance indicated that these slopes were significantly different for any level of conflict above 2.43 ($ps<.05$ for each slope). Therefore, for any conflict level above the baseline (of no conflict, a response of "1" to each question), simple slopes were significantly different from one another. Plots were computed in R using Preacher, Curran & Bauer's code (2013). Conversely, demand and sleep efficiency together did not predict any CV responses (all $ps>.1$; as noted in **Table 4B**)

4.4.2 Sleep duration

No significant relationships between stressor-evoked CV response and sleep duration were detected. Findings are displayed in **Tables 4B & 4C**.

4.4.3 Subjective sleep quality (PSQI)

Significant interactive effects were detected for sleep quality and CV responses to demand only (**Table 4B**). As perceived sleep quality worsened, CV responses to demand increased. More specifically, SBP (Beta =.06; $p<.05$; full model in Table 8), DBP (Beta =.04; $p<.05$; full model in Table 9) and HR (Beta = .05; $p<.05$; full model in Table 10) responses to demand increased. Interaction plots for responses to demand can be seen in **Figures 4, 5** and **6**. Notably, those participants who were one standard deviation above mean PSQI scores (i.e. PSQI score ≥ 8) surpassed the clinical threshold for disturbed sleep, which is 5 (Buysse et al., 1989). Cardiovascular responses to conflict, as a function of sleep quality, were non-significant (*p*s$\geq$.2; results noted in **Table 4C**).

4.5 HYPOTHESIS 3

Following times of high versus low conflict (or demand), those with less adequate sleep (shorter, less efficient, lower quality sleep) will display greater HR, SBP and DBP.

One significant result did appear, aligning with hypotheses. A higher PSQI score (worse sleep quality) predicted higher HR, thirty minutes following a demanding event. This

effect remained after adjusting for the effects of newly introduced demand. However, all other lagged-analyses showed no effects of sleep on persistent CV responses to daily stressors (*p*s>.2). Please see **Tables 5A and 5B** for a summary of these results, including corresponding Beta and *p* values for each analysis.

5.0 DISCUSSION

This study examined whether sleep attributes predicted cardiovascular responses to self-rated daily stressors throughout two days of ambulatory monitoring in middle-aged Black and White adults. First, it queried that worse sleep (defined as lower duration, efficiency, and subjective sleep quality, among continuous measures) predicted elevated cardiovascular parameters throughout the monitoring period. Indeed, those with worse sleep efficiency had greater average HR levels. Second, it examined the extent to which usual sleep characteristics (duration, efficiency and quality) predicted CV responses at times that coincided with two types of daily stressors: social conflict and task demand. Those with poorer subjective sleep quality displayed greater SBP, DBP and HR at times of higher task demand. Also, those with worse sleep efficiency showed exaggerated SBP and DBP responses to social conflict. Lastly, it modeled persistent CV responses to stress through creating an ambulatory data analogue to the cardiovascular "recovery" period documented in experimental stress paradigms. These analyses revealed that poorer quality sleep predicted elevated HR at the time following initiating demand. The present investigation's main contribution lies in its statistical methodology, which extends analytic approaches from experimental sleep and stress reactivity studies to field data, through modeling real-time responses to daily challenges in the context of habitual sleep characteristics. We hope that these approaches may be used in other ABPM data to augment the external validity

of experimental findings, and to understand how CV responses differ between people as a function of their usual sleep attributes, and within-person as a function of varying levels of stress.

5.1 THE EFFECT OF SLEEP CHARACTERISTICS ON AVERAGE ABPM PARAMETERS

The first aim examined whether SBP, DBP or HR levels varied as a function of sleep efficiency, duration or quality. Results indicated that worse sleep efficiency predicted greater average HR levels throughout the time of ABPM. The associated effect size was sizeable; the largest among all significant outcomes, suggesting that for every one unit decrease in sleep efficiency (which was log-transformed, limiting exact unit interpretation), there was a ~4 beat/min increase in average HR levels among participants in this sample. To the extent to which insomnia represents lowered sleep efficiency, the idea that elevated HR relates to poor sleep continuity is not new. Elevated day and nighttime HR has been documented among insomniacs as compared to matched controls (Bonnet & Arand, 1996). Similarly, adults with insomnia who exhibited markers of poor sleep continuity (more wake time after sleep onset, as compared to controls) showed greater increases in HR during a general reaction time task (Stepanski, 1994).

Counter to expectations, no associations between sleep parameters and average blood pressure levels were detected. One plausible explanation may reside in method of assessing sympathetic activation. Findings from experimental fragmentation studies suggest that differences in cardiac autonomic control only appear when using more advanced signal

processing methodology, namely respiratory sinus arrhythmia (RSA) monitoring. When healthy young adults were awoken at 15- to 30-minute intervals, no difference in mean HR or BP levels were detected, but normative increases in RSA accompanying consolidated sleep during the control condition were absent during the fragmentation protocol (Chiacham, Trinder, Carrington & Khoo, 2008). One possible direction may reside in RSA monitoring alongside objective sleep measurement in (a) non-clinical samples, and (b) the absence of experimental arousal protocols (which limit generalizability).

Lack of findings linking sleep duration to blood pressure levels may also be due to the timeframe of analysis used (which considered all BP measurements from wake time to sleep onset). Notably, roughly 40% of the study sample consisted of actigraphy-confirmed "short sleepers" who slept less than 6 hours per night. Experimental analogues of "short sleep," where participants lost half a night's sleep, document elevations in morning BP readings, more so for systolic than diastolic BP levels (Lusardi et al., 1996, 1999). This aligns with findings from total sleep deprivation studies, which report heightened early-morning BP levels (Kato et al., 2000). Given that the current sample had an average of 54 BP readings per person (gross number of BP readings; not those linked to stressor endorsement), or ~18 readings per day, the diurnal shift in BP may have washed out elevations in morning BP levels. To test this theory, we isolated readings to morning-only BP levels (0-3 hours after waking). Main effects of sleep duration appeared, wherein shorter sleep time predicted elevated morning BP levels (Beta = 1.07, $p<.05$).

5.2 THE EFFECT OF SLEEP CHARACTERISTICS ON ACUTE CV RESPONSES TO DAILY STRESSORS

The second aim evaluated whether sleep characteristics predicted CV responses to demand as well as to social conflict. We found that lower sleep efficiency predicted elevated systolic and diastolic BP responses to social conflict only. These results are notable for two reasons. First, they suggest that metrics of sleep continuity may hold predictive utility in connection with stressor-evoked blood pressure responses. Second, they corroborate the general experimental stress literature, indicating that social-evaluative stressors evoke greater BP responses as compared to cognitive stressors (Al'Absi et al., 1997; Kelsey et al., 2000; Franzen et al., 2011). Follow-up analyses confirmed these findings. In unconditional models where both stressors were entered into the model to assess comparative predictive value, we found that conflict explained more variance in BP responses than demand.

Ambulatory monitoring studies from the occupational health literature also uphold the notion that social stress carries unique effects even when adjusting for putative protective factors. For instance, in working couples, higher social-evaluative threat has been associated with greater SBP and DBP after accounting for work-related stressors and buffers such as perceived competence and control (Smith, Birmingham & Uchino, 2012). Similarly, in another study of hypertensive and matched-normotensive middle-aged adults, those with hypertension showed exaggerated BP reactivity to daily work stressors, including social conflict (Baba, Ozawa, Nakamoto, Ueshima & Omae, 1990).

When isolating those who met clinical thresholds for sleep efficiency deficits (<80% SE), we saw a similar pattern to that observed in hypertensive patients. The magnitude of SBP responses to conflict nearly doubled for “inefficient sleepers” as compared to those with sleep

efficiency ≥ 80% ($Beta_{interaction,\ dichotomized\ efficiency}$ = 1.81, p<.01, versus initial analyses where SE was treated as a continuous measure: $Beta_{interaction,\ continuous\ efficiency}$ = .93, p<.01). These finding suggest that those with insomnia (for which this sleep efficiency threshold is a diagnostic criteria) may be especially prone to exaggerated BP responses. Considering the reactivity hypothesis, such heightened stress responses patterns may engender negative cardiovascular risk profiles over time. Future work may want to assess stressor-evoked BP response patterns as a function of chronicity and recency of insomnia symptoms.

Contrary to hypotheses, results indicated that sleep duration did not impact CV responses to stress. This was unexpected, given the experimental work indicating that sleep deprivation engenders exaggerated BP reactivity to socially-mediated stressors especially. Recall that Franzen and colleagues (2011) suggested that total sleep deprivation heightened subjective perception of stress during the paradigm's baseline period, with trends persisting into the Stroop and recovery periods. Evidence from self-report also suggests that adults rate environmental stimuli as more distressing on days preceded by a shorter night's sleep (Hamilton et al., 2008; Kumari et al., 2009). For experimental findings to have been supported in the Sleep SCORE cohort, one would expect habitual short sleepers to endorse greater levels of demand or conflict. However, our preliminary analyses found that duration did *not* predict stress level endorsement (ps>.60). Another explanation may be that habitual short sleep leads to dampened cognitive appraisal of stressful events, or a "desensitization effect," such that more potent stressors are needed to elicit a BP response. In support of this theory, a body of work espouses trait-like differential vulnerability to at least one dimension of stress: negative affect. For instance, after restricting sleep to 6 hours/night, some individuals endorse less negative affect than they do when well-rested (Van Dongen et al., 2004). In addition, over a two-week period, sleep

curtailment to 4 or even 6 hours/night significantly diminishes cognitive functioning; such curtailment may also blunt perceptions of cognitive task difficulty (Van Dongen et al., 2003). Findings from medical residents under chronic sleep restriction conditions demonstrate this notion: while their cognitive performance declines as a function of cumulative sleep debt, residents fail to detect such debilities believing themselves to be just as competent as when well-rested (Robbins & Gottlieb, 1990; Veasey et al., 2002; Zohar et al., 2005). Reduced attentional arousal and impaired central processing are two of the primary cognitive deficits that arise from sleep restriction (Ratcliffe & Van Dongen, 2009). Jointly, they impact the appraisal of environmental stimuli, event valence and relative importance. Taken together, evidence suggests that chronic partial sleep restriction maps onto individual differences in cognitive appraisal processes that are directly implicated in and central to the evaluation of environmental stressors.

Lastly, lower perceived global sleep quality predicted greater SBP, DBP and HR responses, but only in relation to perceived task demand. Recall that demand queried how hard and fast one was working. Both demand ratings and the PSQI are subject to person-level self-report biases. However, given that PSQI assessments were unrelated to endorsements of demand, it seems unlikely that findings simply reflect a participant's pattern of subjective reporting. It is possible that decreased sleep quality affects CV responses to cognitive or task-related stimuli (as characterized by demand), and may affect downstream cognitive processes such as one's perceived ability to complete the task. To augment our understanding of the relationship between PSQI and demand, future studies should inquire whether task demand is viewed as goal-enhancing or goal-disruptive (Zohar et al., 2005). Likewise, other cognitive processes, such as self-affirmation, may buffer the peripheral physiological impact of poor sleep quality, efficiency, or duration. It is important to note that sleep quality assesses a number of

factors not captured by 9 nights of actigraphy, including typical environmental disturbances. It also queries over a longer duration of time (30 days vs. 9 nights). These findings suggest the utility of subjective sleep measures in predicting CV responses to daily task demands, apart from objective sleep measures.

5.3 THE EFFECT OF SLEEP CHARACTERISTICS ON PERSISTENT CV RESPONSES TO DAILY STRESSORS

Poorer subjective sleep quality predicted prolonged HR elevations to demand. This analogue of experimental recovery only partially mimics the pattern of results found in experimental studies. In fact, the picture relating sleep quality to heart rate levels remains less clear, in the context of prior experimental findings. The literature reviewed earlier noted that only one out of four of the studies tended to find increased HR after cognitive stressor exposure (Franzen et al., 2011). As other work posits that correlations between cognitive task appraisals and HR recovery are moderate at best (Ginaros et al., 2009, Franzen et al., 2011 Maier, Waldstein & Synoswki, 2003), factors apart from subjective cognitive distress ratings may influence stressor-evoked recovery. Alternatively, it could be that neural correlates of stressor-evoked cardiovascular recovery differ in the context of poor sleep quality. Metrics aligning with poor sleep quality have been linked with increased amygdala reactivity to negative emotional stimuli (Yoo et al., 2007; Walker et al., 2005) and greater functional connectivity between the amygdala and brainstem regions responsible for autonomic activation. The latter may represent a compensatory neural response to inadequate sleep. Yet, stressor-evoked autonomic responses have been shown to correlate with greater resting corticolimbic activity, independent of

subjective perceptions of stress (Gianaros, et al., 2009). Further work on the functional neural correlates of stressor-evoked autonomic responses in the context of objective and subjective sleep metrics are needed to elucidate these relationships. It is possible that metrics of inadequate sleep contribute to prolonged cardiovascular recovery through corticolimbic influences that operate quite apart from perceptions or appraisals of stress.

With regard to the overall lack of BP recovery findings, we posit that at least two factors, in addition to the neural processes mentioned above, may be at play. First, with regard to conflict, the small number of successive assessments limited analysis of persistent exposure to conflict. This applied less so to endorsements of demand. Second, we may be inadequately able to model the effects of persistent stress without adjusting for all covariates affecting ABPM at the moment of initiating stressor, and the moment of newly introduced lagged stressor. Events that occur less frequently do not have sufficient power to be modeled with moment-level covariates. In general, using such a large number of covariates limits effect size interpretation, contributes to model instability, and can leave models over-determined. It is plausible that such linear modeling methods do not account for small levels of non-linear growth in the context of successive stressor exposure.

5.4 CONSIDERATIONS FOR USING ACTIGRAPHY: SLEEP ONSET LATENCY

While the field of sleep medicine widely employs actigraphy to assess sleep characteristics, research highlights drawbacks in its assessment of SE. The major contention is that actigraphy does not adequately account for sleep onset latency, or the period of time it takes one to fall asleep. The watch-like devices tracking movement to approximate sleep and wake are incapable

of detecting actual time of sleep onset. Building on initial validation studies of actigraphy, sleep onset latency measurements were improved to a .94 correlation (between PSG and actigraphy) when the definition of sleep onset changed to more stringent criteria (Tyron, 2003). Similar criteria were employed in the current study's coding of sleep onset: marked as the beginning of the first 10-minute period of continuous sleep with no more than one minute of movement-related "wake" within the period. To help mitigate sleep onset estimations, common practice is to corroborate actigraphy measures with self-reported sleep diary measures.

Assessment of inadequate sleep hygiene depends on contextual information not garnered from PSG or actigraphy. In a commentary on the fidelity of actigraphy, Tyron suggests collecting multimodal subjective data (2003) to approximate what he terms "inadequate sleep hygiene", an extrinsic sleep disorder. Our analytic approach makes use of such data, comparing actigraphy against PSQI findings. Findings suggest that unique information may be provided from this subjective data, as each metric was related to different CV outcomes and stressors. This aligns with a body of work indicating weak correlations between PSQI and objective measures of sleep (Buysse et al., 1991; Doi et al., 2000; Backhaus et al., 2002).

The aforementioned concerns notwithstanding, precise estimates of actigraphy-assessed SE are less of a concern in our analyses for several reasons. First, we have the added benefit of PSG, known as the gold standard in sleep measurement, from two nights of in-home recording. Our actigraphy findings are consistent with results from PSG-derived measures of sleep efficiency in this sample. Second, correlations between PSG and actigraphy were high in this sample. For total sleep time, correlations were .42 when looking at all nights of actigraphy (9 nights) and .63 when looking at the 2 nights of actigraphy that overlapped with PSG (*p*'s for both correlations <.001). For sleep efficiency, a similar relationship between actigraphy and

PSG held, with .34 and .52 for 9 and 2 nights respectively (*p*'s <.001). Lastly, our analytic approach yields relative relationships rather than specific incremental values of sleep's effect on cardiovascular stress responses.

6.0 SUMMARY AND CONCLUSION

As mentioned, the non-consecutive nature of naturally occurring stressors in this sample made testing for recovery difficult, especially in the context of conflictual interactions. An alternative may have been to examine CV responses at time 1, and as opposed to adjusting for new stressor levels, simply adjust for BP at the time *before* the initiating stressor, to create an artificial "baseline". Analyzing previous night's sleep as it contributes to next-day stress appraisals and CV responses in a feed-forward manner, alongside investigating daytime stress appraisals and CV responses as they predict the next night's sleep, may help us to disentangle the nature of the sleep-stress relationship. More discrete time-series analyses present the next step in evaluating these questions. Furthermore, assessing decision latitude alongside demand ratings may allow us to better characterize perceptions of control. Lastly, more robust predictors of daily stress, such as metrics of perceived judgment from others in the context of social interactions, may help characterize the evaluative nature of stressful interactions.

The current study's strengths lie in its design and data analytic approach. Clearly, through its repeated measures, ABPM allows for more reliable within-person assessments of BP and HR that augment between-person comparisons. Furthermore, ABPM may provide unique prognostic information on CVD risk. The present analytic approach attempted to model cardiovascular "reactivity" and "recovery" outside the laboratory. Findings shed light on possible determinants of exaggerated CV responses. Sleep characteristics predicted CV

parameters in relation to specific stressors: while efficiency moderated the effect of conflict on BP responses, quality predicted BP and HR levels in response to demand. These findings suggest that both subjective and objective sleep measurements may predict distinct cardiovascular response outcomes to specific stressors.

The ambulatory nature of the study provides external validity for experimental work in both the sleep and cardiovascular reactivity domains, through measuring habitual sleep patterns alongside characteristic BP and HR responses in settings relevant to participants' daily lives. Notably, the cross-contextual nature of ABPM allows for characterization of work and home response patterns. Future work in the realm of sleep-related, stressor-evoked reactivity may want to compare response patterns across these environmental domains to assess hypotheses related to differential exposure (e.g. increased reactivity at work as a function of time spent at work) and susceptibility (e.g. individual differences in physiological response profiles and stress endorsements relative to home versus work environments). Reviews of the literature examining the effects of specific daily stressors show that CV responses obtained outside the laboratory are often larger than those recorded in experimental studies (Zanstra & Johnston, 2011). However, ambulatory studies cataloguing patterns of stressor-evoked reactivity in relation to prospective incident CVD are few in number. Field data that capture objectively-measured habitual sleep characteristics will undoubtedly provide better metrics of the sleep-stress relationship, and shed further light on how the reactivity hypothesis, posited to cause CVD, relates to sleep inadequacy.

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APPENDIX A

SAMPLE MODEL SPECIFICATIONS FOR ACUTE AND PERSISTENT CARDIOVASCULAR RESPONSES

Example of **acute stress model specification** for relationship between perceived demand and SBP as a function of sleep efficiency (SE):

Level-1 Model

$$SBP\ t_0\ ij = \beta 0j + \beta 1j*(talking_{ij}) + \beta 2j*(substance\ intake_{ij}) + \boldsymbol{\beta 3j}*(perceived\ demand\ t_{0}ij) + \beta 4j*(posture_{ij}) + \beta 5j*(physical\ activity_{ij}) + r_{ij}$$

Level-2 Model

$$\beta 0j = \gamma 00 + \gamma 01*(age_j) + \gamma 02*(BMI_j) + \gamma 03*(SE_j) + \gamma 04*(race_j) + \gamma 05*(gender_j) + \gamma 06*(cardiovascular\ medication\ use_j) + \gamma 07*(AHI_j) + u0j$$

$$\beta 1j = \gamma 10$$

$$\beta 2j = \gamma 20$$

$$\beta 3j = \gamma 30 + \boldsymbol{\gamma 31}*(SE_j) + \gamma 32*(age_j) + \gamma 33*(BMI_j) + \gamma 34*(race_j) + \gamma 35*(gender_j) + \gamma 36*(cardiovascular\ medication\ use_j) + \gamma 37*(AHI_j) + u3j$$

$$\beta 4j = \gamma 40$$

$$\beta 5j = \gamma 50$$

[Continued Appendix A]

Example of **persistent stress model specification** for relationship between perceived demand and SBP as a function of SE at t_{0+1} (one 30-minute epoch post-stressor; denoted as t1 in models).

Level-1 Model

$$SBP\ t_1\ ij = \beta 0j + \beta 1j*(talking_{ij}) + \beta 2j*(substance\ intake_{ij}) + \boldsymbol{\beta 3j}*(perceived\ demand\ t_0 ij) + \beta 4j*(posture_{ij}) + \beta 5j*(physical\ activity_{ij}) + \beta 6j*(perceived\ demand\ t_1 ij) + rij$$

Level-2 Model

$$\beta 0j = \gamma 00 + \gamma 01*(age_j) + \gamma 02*(BMI_j) + \gamma 03*(SE_j) + \gamma 04*(ethnicity_j) + \gamma 05*(gender_j) + \gamma 06*(cardiovascular\ medication\ use_j) + \gamma 07*(AHI_j) + u0j$$

$$\beta 1j = \gamma 10$$

$$\beta 2j = \gamma 20$$

$$\beta 3j = \gamma 30 + \boldsymbol{\gamma 31}*(SE_j) + \gamma 32*(age_j) + \gamma 33*(BMI_j) + \gamma 34*(ethnicity_j) + \gamma 35*(gender_j) + \gamma 36*(cardiovascular\ medication\ use_j) + \gamma 37*(AHI_j) + u3j$$

$$\beta 4j = \gamma 40$$

$$\beta 5j = \gamma 50$$

$$\beta 6j = \gamma 60$$

APPENDIX B

TABLES AND FIGURES

Table 1. Sample characteristics

Variable	*n (%)*	*M (SD)*	*Range*
Female	106 (49.3)		
Race			
White	117		
Black	93		
Asian	4		
	214		
Current smoker	19 (8.9)		
Cardiac/Hypertensive Medication Use	50 (27.2)		
Age		59.9 (7.2)	45.6 – 77.6
Body Mass Index		29.5 (5.1)	17.9 – 45.3
Sleep Efficiency (%time asleep/time in bed)		80.5 (8.0)	45.3 – 93.2
Sleep Duration (TST; in hrs)		6.7 (.9)	4.3 – 9.4
Pittsburgh Sleep Quality Index		6.4 (3.3)	0 – 18
AHI		13.2 (15.1)	0 – 77.2
% < 5	61 (28.3)		
% 5 – 15	94 (43.7)		
% > 15	60 (27.9)		

[a] This category includes those who answered "yes" to cardiac/hypertensive medication use.
[b] Please note that these metrics refer final analytic sample n = 215.

Table 2. Variability in cardiovascular outcomes and stress predictors

Variable	Mean (SD)	Range
Heart Rate		
Mean	76.4 (14.6)	32 – 185
Within-Person Variability	10.3	
Systolic Blood Pressure		
Mean	129.7 (16.5)	77 – 210
Within-Person Variability	11.5	
Diastolic Blood Pressure		
Mean	78.9 (12.8)	33 – 140
Within-Person Variability	8.4	
Demand		
Mean	4.74 (2.71)	2 – 12
Within-Person Variability	1.2 (.6)	.2 – 1.9
Conflict		
Mean	3.18 (1.49)	2 – 12
Within-Person Variability	.7 (.2)	.1 – 1.5

Table 3. Bivariate correlations between sleep duration, efficiency, quality and stressor

Variable	1	2	3	4	5	6	7	8	9
1. Mean Duration	—								
2. Mean Efficiency	-.55**	—							
3. Pittsburgh Sleep Quality Index	-.19**	.13**	—						
4. Average Demand	.01	-.03	.01	—					
5. Average Conflict	-.01	.01	.05**	.10*	—				
6. BMI	-.15**	.10**	.07**	-.06**	-.01	—			
7. AHI	-.11**	.08**	-.06**	-.01	-.11	.11**	—		
8. Gender	-.08**	-.07**	.20**	-.03**	-30**	.13**	-.26**	—	
9. Cardiac Medication Use	-.11	.01	.01	.04*	.17*	.20**	.21**	.20**	—

* Correlation is significant at $p \leq .05$ level.
** Correlation is significant at $p \leq .01$ level.
NOTE: Sleep Efficiency is transformed such that higher numbers indicate lower sleep efficiency. It is reverse-coded, explaining the negative correlations seen.

Table 4. Regression coefficients from fully adjusted momentary response models

Main Effect Models

	Average Transformed Sleep Efficiency (higher = worse SE)		**Average Actual Sleep Time (higher = longer TST)**		**PSQI (higher = worse quality)**	
	Coefficient (SE)	*p*-value	Coefficient (SE)	*p*-value	Coefficient (SE)	*p*-value
SBP						
Main effect (a)	.88(2.43)	.72	-.51(.98)	.60	.12(.26)	.63
DBP						
Main effect (b)	1.86(1.74)	.29	-.60(.71)	.40	.01(.05)	.95
HR						
Main effect (c)	**4.07(2.04)**	**.04**	-.22(.80)	.79	.34(.22)	.12

Models adjusted for age, race/ethnicity, BMI, gender, cardiovascular medication use, and AHI at the person-level. Time-varying covariates included talking, substance intake, posture and physical activity adjusted for at the time of each ABP measurement.
p-values in red refer to significant difference from sample average.
Note that main effects models are modeled without demand, conflict or an interaction term. This is the main effect of each sleep parameter on the CV outcome, adjusted for the ABP and person-level covariates listed above.

Table 5. Regression coefficients from fully adjusted interaction models for demand

	Average Transformed Sleep Efficiency (higher = worse SE)		Average Actual Sleep Time (higher = longer TST)		PSQI (higher = worse quality)	
	Coefficient (SE)	*p*	Coefficient (SE)	*p*	Coefficient (SE)	*p*
SBP						
Interaction with demand (d)	-.18(.25)	.29	.02 (.11)	.89	.06(.03)	.04
DBP						
Interaction with demand (e)	-.20(.17)	.23	.01(.07)	.90	.04(.02)	.03
HR						
Interaction with demand (f)	-.40(.25)	.11	-.02(.11)	.82	.05(.02)	.04
mean # of readings (range) % compliance (answering stressor items)	41 (12-57) (81.6% compliance)					

Average # Demand Events: Only one value is given for all models using demand as the moderator, because the only thing changing between models was the sleep variable. All actigraphy and PSQI data were complete for each subject in dataset (no missing sleep variable values).

Compliance: % compliance is based on answering demand questions if ANY other diary measure was answered. % compliance overall (# demand items answered/total # blood pressure readings = 62.3%)

Models adjusted for age, race/ethnicity, BMI, gender, cardiovascular medication use, and AHI at the person-level. Time-varying covariates included talking, substance intake, posture and physical activity adjusted for at the time of each ABP measurement.

p-values in red refer to significant difference from sample average.

Table 6. Regression coefficients from fully adjusted interaction models for conflict

	Average Transformed Sleep Efficiency (higher = worse SE)		**Average Actual Sleep Time (higher = longer TST)**		**PSQI (higher = worse quality)**	
	Coefficient (SE)	*p*-value	Coefficient (SE)	*p*-value	Coefficient (SE)	*p*-value
SBP						
Interaction with conflict (g)	**.93(.33)**	**<.01**	-.06(.15)	.72	.02(.03)	.53
DBP						
Interaction with conflict (h)	**.83(.28)**	**.01**	-.19(.13)	.13	.04(.03)	.20
HR						
Interaction with conflict (i)	-.31(.32)	.33	.06(.14)	.66	.02(.04)	.59
mean # of readings (range) % compliance (answering stressor items)	28 (0 – 53) (86.7% compliance)					

Average # Conflict Events: Only one value is given for all models using conflict as the moderator, because the only variable changing in each model is the sleep variable. All actigraphy and PSQI data were complete for each subject in the dataset (no missing sleep variable values).

Compliance Notes: Compliance is compared to whether participant had a social interaction before the blood pressure reading (#responses to conflict questions/#endorsement of interaction). Also note, this gives total # of social interactions (not total # of negative social interactions).

Models adjusted for age, race/ethnicity, BMI, gender, cardiovascular medication use, and AHI at the person-level. Time-varying covariates included talking, substance intake, posture and physical activity adjusted for at the time of each ABP measurement.
p-values in red refer to significant difference from sample average.

Table 7. Regression coefficients from fully adjusted lagged models for demand

	Average Transformed Sleep Efficiency (higher = worse SE)		**Average Actual Sleep Time (higher = longer TST)**		**PSQI (higher = worse quality)**	
	Coefficient (SE)	*p*-value	Coefficient (SE)	*p*-value	Coefficient (SE)	*p*-value
SBP	-.17(.10)	.49	.001(.11)	.99	.03(.03)	.38
DBP	-.17(.17)	.30	.02(.06)	.78	-.004(.02)	.81
HR	-.22(.21)	.30	.06(.12)	.62	**.04 (.02)**	**.03**
mean # of contiguous demand readings (range)					35 (9 – 53)	

Table 8. Regression coefficients from fully adjusted lagged models for conflict

	Average Transformed Sleep Efficiency (higher = worse SE)	**Average Actual Sleep Time (higher = longer TST)**			**PSQI (higher = worse quality)**	
	Coefficient (SE)	*p*-value	Coefficient (SE)	*p*-value	Coefficient (SE)	*p*-value
SBP	.32(.50)	.32	-.12(.19)	.52	.001(.05)	.98
DBP	.17(.25)	.50	-.12(.13)	.38	.02(.03)	.56
HR	-.14(.32)	.65	-.04(.17)	.81	-.01(.04)	.73

mean # of contiguous conflict readings (range)	19 (1 – 52)

Table 9. Random intercepts and slopes as outcomes in regression model of SBP response to demand as a function of PSQI

Fixed Effect	Coefficient	(SE)	*t*-ratio	*p*-value
Model for mean SBP response, β_0				
Intercept, γ_{00}	130.04	1.72	75.62	<0.001
Age, γ_{01}	0.39	0.12	3.18	0.002
Race, γ_{02}	-1.22	1.86	-0.65	0.51
BMI, γ_{03}	0.08	0.17	0.46	0.64
AHI, γ_{04}	0.37	0.88	0.42	0.678
Sex, γ_{05}	1.10	1.78	0.62	0.54
CV Medication Use, γ_{06}	-0.01	1.01	-0.01	0.98
Demand (group mean), γ_{07}	-0.68	0.55	-1.24	0.22
Posture (group mean), γ_{08}	3.10	4.57	0.67	0.49
Substance use (group mean), γ_{09}	-0.74	6.00	-0.12	0.90
Physical activity (group mean), γ_{010}	3.73	3.56	1.05	0.30
Talking (group mean), , γ_{011}	20.89	4.96	4.22	<0.001
PSQI, γ_{012}	0.12	0.26	0.45	0.652
For posture slope, β_1				
Intercept, γ_{10}	-2.52	0.24	-10.34	<0.001
For demand slope, β_2				
Intercept, γ_{20}	-0.24	0.19	-1.28	0.19
Age, γ_{21}	0.01	0.013	1.13	0.26
Race, γ_{22}	0.59	0.19	3.15	0.002
BMI, γ_{23}	0.04	0.02	2.11	0.03
AHI, γ_{24}	-0.02	0.10	-0.18	0.86
Sex, γ_{25}	0.01	0.19	0.04	0.97
PSQI, γ_{26}	0.06	0.03	2.05	0.04
For talking slope, β_3				
Intercept, γ_{30}	0.73	0.268171	2.704	0.007
For physical activity slope, β_4				
Intercept, γ_{40}	1.76	0.25	7.13	<0.001
For substance intake slope, β_5				
Intercept, γ_{50}	-1.53	0.34	-4.45	<0.001
Random Effect	*Variance Component*	*d.f.*	*χ2*	*p*
Var. in individual means (τ_{00})	132.60	200	10410.44	<0.001
Var. in conflict slope (τ_{11})	0.79	214	526.02	<0.001
Var. within individuals (σ^2)	12.65			

Table 10. Random intercepts and slopes as outcomes in regression model of DBP response to demand as a function of PSQI

Fixed Effect	Coefficient	(SE)	*t*-ratio	*p*-value
Model for DBP Response, β_0				
Intercept, γ_{00}	81.38	1.23	66.41	<0.001
Age, γ_{01}	-0.07	0.09	-0.81	0.42
Race, γ_{02}	-2.60	1.32	-1.97	0.05
BMI, γ_{03}	-0.24	0.12	-1.91	0.06
AHI, γ_{04}	0.29	0.63	0.46	0.65
Sex, γ_{05}	-1.79	1.26	-1.42	0.16
CV Medication Use, γ_{06}	-0.11	0.72	-0.15	0.88
Demand (group mean), γ_{07}	-0.55	0.39	-1.41	0.16
Posture (group mean), γ_{08}	-2.21	3.27	-0.67	0.50
Substance use (group mean), γ_{09}	3.35	4.29	0.78	0.44
Physical activity (group mean), γ_{010}	2.99	2.55	1.18	0.24
Talking (group mean), , γ_{011}	15.68	3.55	4.42	<0.001
PSQI, γ_{012}	-0.04	0.19	-0.19	0.85
For posture slope, β_1				
Intercept, γ_{10}	-3.26	0.18	-18.33	<0.001
For demand slope, β_2				
Intercept, γ_{20}	-0.18	0.12	-1.49	0.14
Age, γ_{21}	-0.01	0.01	-0.71	0.48
Race, γ_{22}	0.40	0.12	3.29	0.00
BMI, γ_{23}	0.02	0.01	1.96	0.05
AHI, γ_{24}	0.03	0.06	0.41	0.68
Sex, γ_{25}	0.03	0.12	0.29	0.78
CV Medication Use, γ_{26}	-.13	0.17	0.96	0.53
PSQI, γ_{27}	0.04	0.02	2.28	0.02
For talking slope, β_3				
Intercept, γ_{30}	1.56	0.20	7.98	<0.001
For physical activity slope, β_4				
Intercept, γ_{40}	0.34	0.18	1.91	0.06
For substance intake slope, β_5				
Intercept, γ_{50}	-1.21	0.25	-4.83	<0.001
Random Effect	*Variance Component*	*d.f.*	χ^2	*p-value*
Var. in individual means (τ_{00})	67.25	204	9761.01	<0.001
Var. in conflict slope (τ_{11})	0.53	214	411.0	<0.001
Var. within individuals (σ^2)	7.65			

Table 11. Random intercepts and slopes as outcomes in regression model of HR response to demand as a function of PSQI

Fixed Effects	Coefficient	(SE)	*t*-ratio	*p*-value
Model for HR Response, β_0				
Intercept, γ_{00}	77.06	1.56	49.26	<0.001
Age, γ_{01}	-0.20	0.10	-1.94	0.05
Race, γ_{02}	-3.81	1.64	-2.33	0.02
BMI, γ_{03}	-0.01	0.16	-0.04	0.97
AHI, γ_{04}	-0.23	0.71	-0.32	0.75
Sex, γ_{05}	3.54	1.46	2.43	0.02
CV Medication Use, γ_{06}	0.48	0.81	0.59	0.56
Demand (group mean), γ_{07}	-0.15	0.47	-0.32	0.75
Posture (group mean), γ_{08}	0.29	3.40	0.09	0.93
Substance use (group mean), γ_{09}	5.38	5.62	0.96	0.34
Physical activity (group mean), γ_{010}	6.96	3.10	2.25	0.03
Talking (group mean), γ_{011}	7.01	3.90	1.80	0.07
PSQI, γ_{012}	0.15	0.23	0.67	0.50
For posture slope, β_1				
Intercept, γ_{10}	-6.27	0.30	-20.65	<0.001
For demand slope, β_2				
Intercept, γ_{20}	0.29	0.19	1.58	0.12
Age, γ_{21}	-0.02	0.02	-1.42	0.16
Race, γ_{22}	0.60	0.19	3.22	0.00
BMI, γ_{23}	-0.01	0.02	-0.62	0.54
AHI, γ_{24}	0.07	0.12	0.57	0.57
Sex, γ_{25}	-0.23	0.19	-1.22	0.22
CV Medication Use, γ_{26}	-.15	0.14	1.1	0.58
PSQI, γ_{27}	0.05	0.03	2.02	0.04
For talking slope, β_3				
Intercept, γ_{30}	0.20	0.26	0.77	0.44
For physical activity slope, β_4				
Intercept, γ_{40}	3.53	0.33	10.62	<0.001
For substance intake slope, β_5				
Intercept, γ_{50}	-0.56	0.31	-1.83	0.07

Random Effect	*Variance Component*	*d.f.*	χ^2	*p-value*
Var. in individual means (τ_{00})	87.67	204	8778.85	<0.001
Var. in demand slope (τ_{11})	.76	214	558.58	<0.001
Var. within individuals (σ^2)	8.67			

Table 12. Random intercepts and slopes as outcomes in regression model of SBP response to conflict as a function of sleep efficiency

Fixed Effects	Coefficient	(SE)	*t*-ratio	*p*-value
For Mean SBP Response, β_0				
Intercept, γ_{00}	129.77	(2.02)	68.26	<.001
Age, γ_{01}	.34	(.12)	3.44	<.001
Race, γ_{02}	-1.28	(2.08)	-.96	.34
BMI, γ_{03}	.05	(.17)	.41	.68
Sex, γ_{04}	1.47	(1.68)	.56	.48
AHI, γ_{05}	.35	(.97)	.36	.71
CV Medication Use, γ_{06}	1.65	(1.84)	.90	.37
Sleep Efficiency, γ_{07}	.58	(2.69)	.07	.88
Posture (group mean) γ_{08}	.58	(4.39)	.41	.63
Substance intake (group mean) γ_{09}	-.87	(6.44)	.11	.85
Conflict, (group mean) γ_{010}	1.95	(.82)	-.15	.88
Physical activity, (group mean) γ_{011}	3.48	(3.84)	.86	.37
Talking, (group mean) γ_{012}	18.98	(5.13)	3.51	<.001
For time-of-day slope, β_1				
Intercept, γ_{10}	.14	(.03)	3.66	<.001
For posture slope, β_2				
Intercept, γ_{20}	-2.07	(.42)	-4.82	<.001
For conflict slope, β_3				
Intercept, γ_{20}	-.28	(.41)	-.69	.49
Age, γ_{21}	.01	(.02)	.60	.54
Race, γ_{22}	.15	(.28)	.54	.59
BMI, γ_{23}	.01	(.03)	.42	.67
AHI, γ_{24}	.08	(.13)	.61	.54
Sex, γ_{25}	.48	(.31)	1.55	.12
CV Medication Use, γ_{27}	-.01	(.27)	-.05	.96
Sleep Efficiency, γ_{26}	.93	(.33)	2.78	.006
For talking slope, β_4				
Intercept, γ_{40}	.57	(.34)	1.68	.107
For physical activity slope, β_5				
Intercept, γ_{50}	2.30	(.32)	6.56	<.001
For substance intake slope, β_6				
Intercept, γ_{60}	-1.72	67(.48)	-3.44	<.001

Random Effects	*Variance component*	*df*	χ^2	*p*-value
Var. in individual means (τ_{00})	138.45	200	1227.	<.001
Var. in conflict slope (τ_{11})	.79	205	378.4	<.001
Var. within individuals (σ^2)	9.87			

Note: Higher Beta values indicate worse sleep efficiency due to log transformation

Table 13. Random intercepts and slopes as outcomes in regression model of DBP response to conflict as a function of sleep efficiency

Fixed Effects	Coefficient	(SE)	*t*-ratio	*p*-value
For Mean DBP Response, β_0				
Intercept, γ_{00}	81.33	(1.50)	54.16	<0.001
Age, γ_{01}	-.08	(.09)	-0.81	.41
Race, γ_{02}	-2.94	(1.35)	-2.18	.03
BMI, γ_{03}	-.22	(.13)	-1.69	.09
Sex, γ_{04}	-1.91	(1.29)	-1.48	<0.001
AHI, γ_{05}	.14	(.67)	.21	.83
CV Medication Use, γ_{06}	1.50	(1.30)	1.15	.25
Sleep Efficiency, γ_{07}	.56	(1.77)	.07	.75
Posture (group mean) γ_{08}	-2.80	(3.39)	.41	.41
Substance intake (group mean) γ_{09}	6.44	(4.45)	.11	.85
Conflict, (group mean) γ_{010}	.18	(.57)	-.15	.88
Physical activity, (group mean) γ_{011}	2.05	(2.49)	.86	.37
Talking, (group mean) γ_{012}	14.25	(3.80)	3.51	<.001
For time-of-day slope, β_1				
Intercept, γ_{10}	.14	(.03)	3.66	<.001
For posture slope, β_2				
Intercept, γ_{20}	-2.07	(.42)	-4.82	<.001
For conflict slope, β_3				
Intercept, γ_{20}	-0.226232	.41	(-.82)	.42
Age, γ_{21}	0.011367	.02	(.73)	.47
Race, γ_{22}	0.248127	.28	(1.12)	.27
BMI, γ_{23}	-0.025511	.03	(-1.12)	.26
AHI, γ_{24}	0.189769	.13	(1.58)	.12
Sex, γ_{25}	0.398938	.31	(1.71)	.08
CV Medication Use, γ_{27}	-.13	.27	(-.62)	.54
Sleep Efficiency, γ_{26}	.83	.28	(2.43)	.01
For talking slope, β_4				
Intercept, γ_{40}	1.42	(.22)	6.49	<.001
For physical activity slope, β_5				
Intercept, γ_{50}	.71	(.32)	3.65	<.001
For substance intake slope, β_6				
Intercept, γ_{60}	-1.33	(.29)	-4.51	<.001

Random Effects	*Variance component*	*df*	χ^2	*p*-value
Var. in individual means (τ_{00})	73.24	200	1214.	<.001
Var. in conflict slope (τ_{11})	.39	205	381.2	<.001
Var. within individuals (σ^2)	8.19			

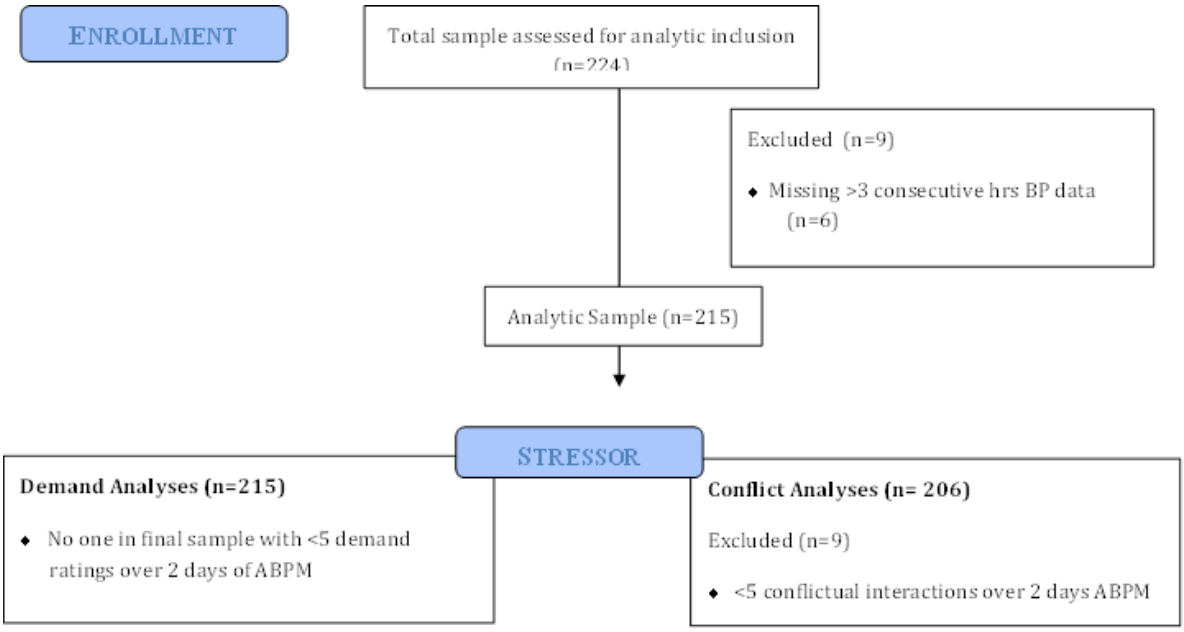
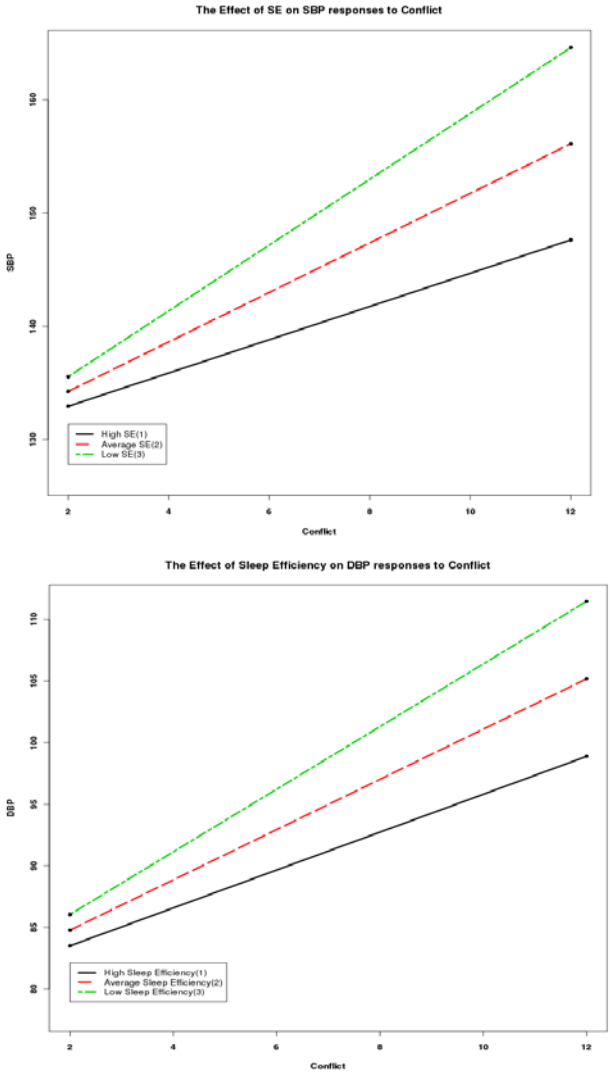
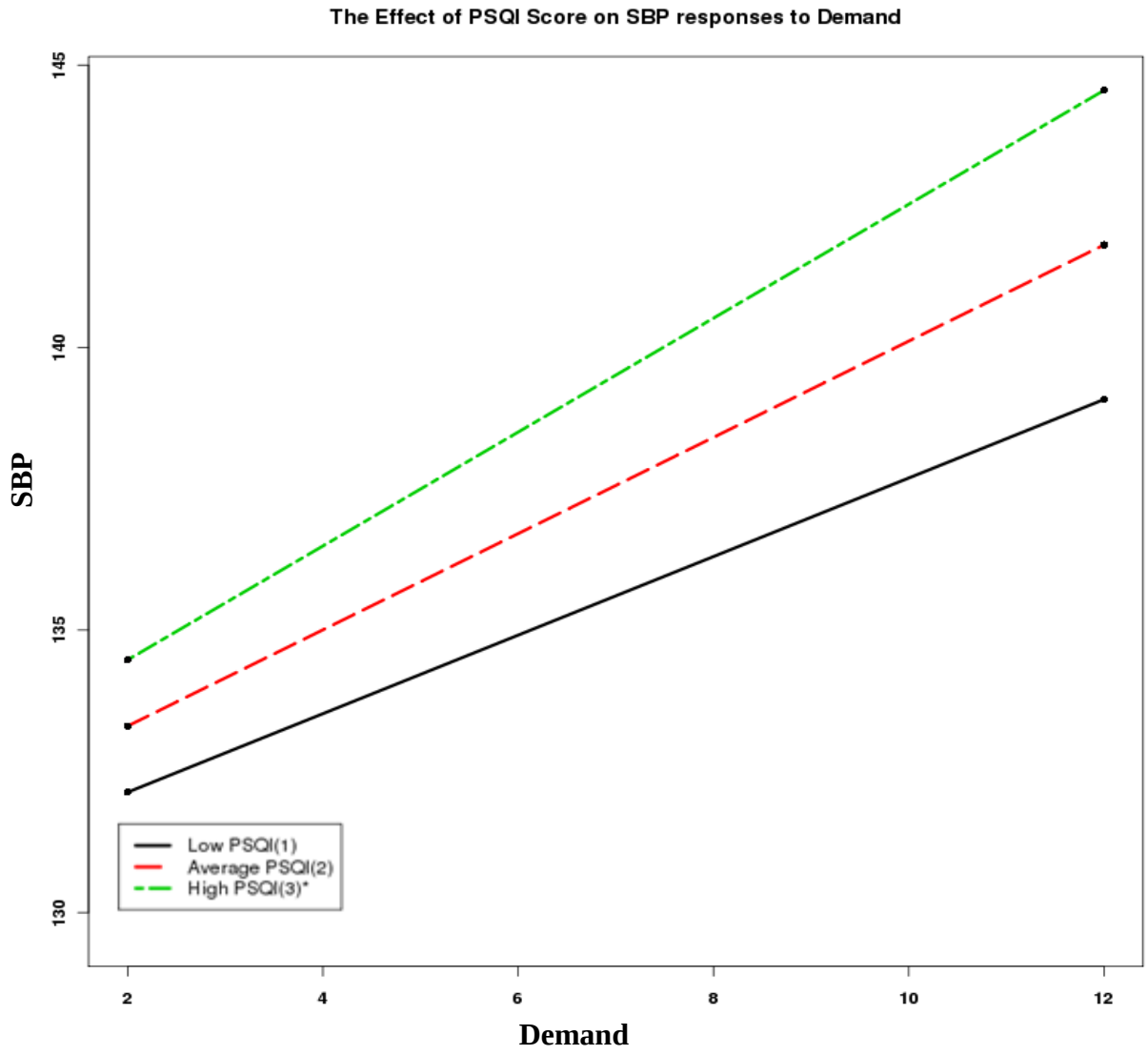


Figure 2. Data analysis flow diagram



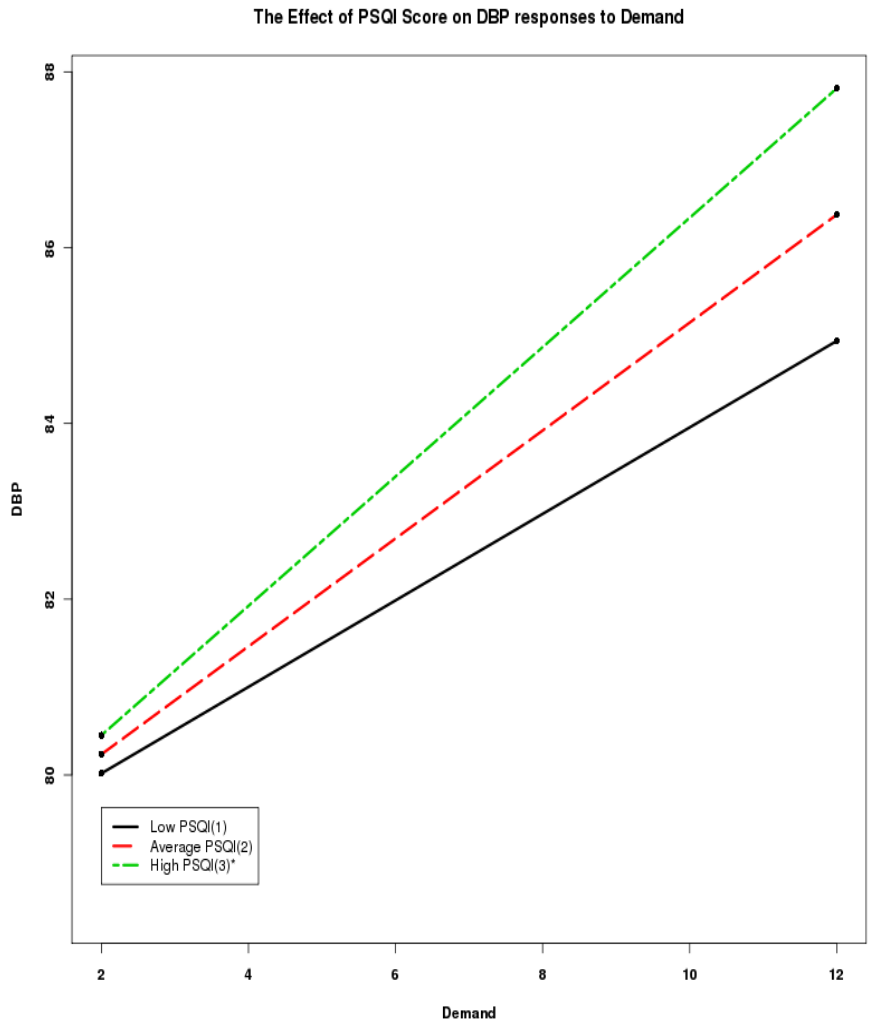
Note: The sleep efficiency by conflict interaction term was associated with SBP (top panel) at β = .93, p <.01 and with DBP (bottom panel) at β = .73, p =.01.

Figure 3. Simple slopes (interaction plot) of BP responses to conflict as a function of low, average and high sleep efficiency



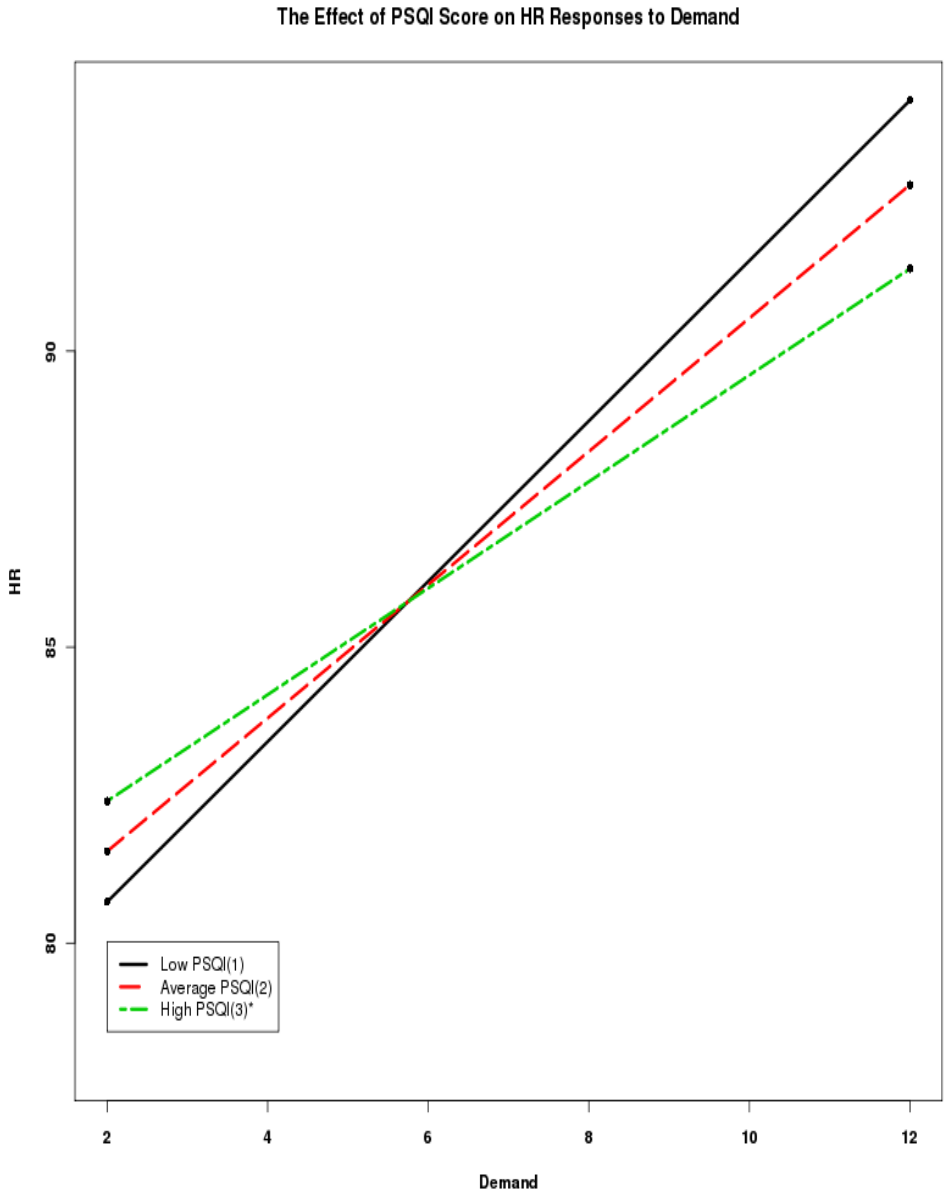
Note: Higher scores indicate worse subjective sleep quality. The sleep quality by demand interaction term was associated with SBPat $\beta = .06$, $p < .05$.

Figure 4. Simple slopes (interaction plot) of SBP responses to demand as a function of low, average, and high PSQI score



Note: Higher scores indicate worse subjective sleep quality. The sleep quality by demand interaction term was associated with SBPat $\beta = .06$, $p < .05$.

Figure 5. Simple Slopes (Interaction Plot) of DBP Responses to Demand as a Function of Low, Average and High PSQI Score



Note: Higher scores indicate worse subjective sleep quality. The sleep quality by demand interaction term was associated with HR at $\beta = .4.1$, $p < .05$. Significant differences in responses exist at demand levels above 9.

Figure 6. Simple Slopes (Interaction Plot) of HR Responses to Demand as a Function of Low, Average and High PSQI Score