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Nocturnal Blood Pressure Non-Dipping, Posttraumatic Stress Disorder, and Sleep Quality in Women

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Abstract

Background—Women with posttraumatic stress disorder (PTSD) have poor sleep quality and increased risk of cardiovascular disease (CVD). Non-dipping of nocturnal blood pressure may be an explanatory factor for the relationship between sleep and CVD found in previous research.

Purpose—The current study was designed to determine if non-dipping nocturnal blood pressure was associated with trauma exposure, PTSD diagnosis, PTSD symptoms, and sleep quality in a sample of women.

Methods—Participants completed 24 hours of ABPM and self-report questionnaires. Non-dipping was defined as less than 10% reduction in blood pressure during sleep.

Results—The frequency of non-dippers did not differ by diagnostic status ($d=.15$). However, non-dippers endorsed more traumatic event categories ($d=.53$), more PTSD hyperarousal symptoms ($d=.53$), poorer overall sleep quality ($d=.59$), more frequent use of sleep medication ($d=.62$), greater sleep-related daytime dysfunction ($d=.58$), and longer sleep onset latencies ($d=.55$) than dippers.

Conclusions—Increased attention to nocturnal blood pressure variation may be needed to improve blood pressure control in trauma-exposed women.

Keywords

circadian blood pressure; PTSD; sleep quality; cardiovascular disease; psychological trauma

Introduction

Heart disease is the leading cause of death in US women¹, and hypertension is a major risk factor for the development of heart disease. Recent research in the area of hypertension control has focused increasingly on the clinical relevance of nocturnal blood pressure in

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predicting target organ damage^{2, 3}. In their 1988 letter to Lancet, O'Brien and colleagues first coined the term "non-dipper" to describe those whose mean nocturnal blood pressure does not vary significantly from mean daytime blood pressure⁴. In a recent review of prospective cohort studies, Hansen, et al.⁵ demonstrated that non-dipping blood pressure, defined as an average nighttime blood pressure reduction that is less than 10% of average daytime blood pressure, is a significant predictor of cardiovascular events in hypertensive patients and a significant predictor of mortality in both hypertensive patients and population-based samples.

The process by which nocturnal blood pressure variation becomes blunted remains unclear, but a number of factors have been identified as potential mechanisms underlying deficits in nighttime blood pressure reduction. Blood pressure dipping seems to be impacted by sleep quality⁶ and is related to the pattern of sleep stages across the night⁷. Stress seems to be a particularly salient factor in impacting nocturnal blood pressure as well. In sleep apnea patients, treatment resulted in normalization of sleep and a parallel reduction in both nocturnal blood pressure and stress hormone levels⁸ (e.g., cortisol, epinephrine and norepinephrine). In hypertensive adults, both poor sleep quality and high perceived stress were found to be independent predictors of non-dipping blood pressure⁹.

Stress hormones have a circadian pattern which varies with sleep. For example, the stress hormone epinephrine tends to decrease upon sleep onset and increase upon awakening, and blood pressure variance closely parallels epinephrine variation across the sleep cycle¹⁰. Cortisol is another stress hormone which varies across the sleep cycle, and is also related to nocturnal blood pressure variation. In a sample of healthy adults, reduced variation in diurnal cortisol was associated with decreased nocturnal blood pressure dipping, and this effect was found to be independent of important covariates¹¹. Other research has shown that both epinephrine and norepinephrine levels and perceived stress decreased in response to pharmacotherapy for poor sleep quality in non-dipping hypertensives⁹. Moreover, this longitudinal study found that a significantly greater proportion of non-dipping poor sleepers were converted to dippers in the pharmacotherapy group (50%) relative to the placebo control group (8.7%)¹². This finding suggests that sleep quality and stress hormone levels are closely related to nocturnal blood pressure regulation in those with non-dipping blood pressure. Collectively, the findings on sleep, stress and nocturnal blood pressure suggest that there is a close connection between the sleep process itself, the circadian regulation of physiological arousal, and nocturnal blood pressure variability.

Although several studies have found relationships between blood pressure and psychological symptoms, such as anxiety and depression¹³⁻¹⁶, fewer studies have examined psychological symptoms and circadian blood pressure variation. Scuteri and colleagues¹⁷ found a lesser drop in nocturnal blood pressure among those with depression, and Linden and colleagues¹⁸ found that non-dippers consumed more alcohol and were more likely to have an anger coping style that involves difficulty "letting go" of anger. Mezick and colleagues¹⁹ found that non-dipping blood pressure is associated with high hostility and low life purpose, and Fallo and colleagues²⁰ found a significant relationship between stressful life circumstances and nocturnal blood pressure dipping in normotensive outpatients, but not in hypertensive patients. Other research has examined non-dipping of blood pressure as it relates to psychosocial factors, and has demonstrated associations between non-dipping blood pressure, and income and education in African Americans²¹, and acculturation level in Mexican American women²². Spruill and colleagues²³ found that, relative to whites, non-dipping blood pressure among blacks is partially explained by marital status (being unmarried) and lower education, whereas Stepnowsky and colleagues²⁴ found that socioeconomic status was an independent predictor of non-dipping blood pressure, after adjusting for racial differences.

Non-dipping blood pressure and Posttraumatic Stress Disorder (PTSD) have several overlapping associations. As outlined above, non-dipping blood pressure is associated with increased stress and greater sleep difficulties, in addition to both hypertension and cardiovascular disease. A PTSD diagnosis involves hyperarousal, re-experiencing of stress and sleep difficulties²⁵, and having a diagnosis of PTSD places one at greater risk of hypertension²⁶ and death due to early onset of coronary heart disease²⁷. In a sample of women, we found evidence of these shared associations across several previous studies, including one study focused on baroreceptor sensitivity²⁸. Baroreceptors are sensors located in the wall of blood vessels which detect blood pressure changes and relay this information to the central nervous system. The sensitivity of these receptors (i.e., baroreceptor sensitivity), is widely considered to be an index of the body's ability to regulate blood pressure²⁹. We found that women having a PTSD diagnosis had lower resting baroreceptor sensitivity (lesser ability to regulate blood pressure), higher heart rate³⁰ and poorer sleep quality³¹ than women without PTSD.

To our knowledge, only one study has focused on nocturnal blood pressure non-dipping and PTSD. In a sample of young African American adults (60% female), Mellman and colleagues³² found that nocturnal blood pressure non-dipping was associated with lifetime PTSD, subthreshold lifetime PTSD, and PTSD severity. Whereas all participants in the Mellman et al sample were exposed to trauma, only one participant in this sample met full criteria for current PTSD. The Mellman et al sample was also unique in that the sample was composed entirely of African Americans who are at greater risk of hypertension³³ and possibly non-dipping nocturnal blood pressure². Research has also demonstrated gender differences in blood pressure regulation³⁴.

The current study was designed to further the line of research pursued by Mellman and colleagues by comparing systolic blood pressure dippers to non-dippers on PTSD diagnostic status, exposure to trauma, trauma symptoms (severity and frequency), and sleep quality in a racially heterogeneous sample of women. Based upon the literature reviewed above, we hypothesized that there would be a higher proportion of women meeting criteria for a diagnosis of current PTSD among non-dippers than dippers (Hypothesis 1), and that non-dippers would endorse greater exposure to trauma, more PTSD symptoms, and lower sleep quality than dippers (Hypothesis 2).

Method

Participants

Data were obtained from a prior study examining hostility, health and PTSD in women. Women were recruited for the parent study through advertisements on bulletin boards at Veterans Administration (VA) and community hospitals between 2001 and 2005, which read "Women who have a history of trauma (such as physical or sexual assault or natural disaster), may be eligible to participate in a research study examining the effects of women's beliefs and attitudes on health." Institutional review board approval was obtained from the university and VA medical centers where the study was conducted. Study procedures were fully explained and all participants provided informed consent.

The parent study was designed to compare women across 3 conditions: women with PTSD (either with or without depression); women with depression only (no PTSD); and a comparison group of women without PTSD or depression. The women having depression only (no PTSD) were not included in the current analyses. Women recruited for the PTSD condition must have met criteria for current PTSD. Women recruited for the non-PTSD comparison condition were excluded if they met criteria for current or lifetime PTSD or current or lifetime major depressive disorder. Women meeting criteria for current alcohol or

substance dependence/abuse or psychotic disorders (including schizophrenia and bipolar with active manic symptoms) were excluded from both conditions of the parent study.

As depicted in Figure 1 above, 198 women were screened between 2001 and 2005. Participants screened for the comparison group (i.e., neither PTSD nor MDD) were excluded if they met criteria for lifetime PTSD ($n = 27$) or MDD ($n = 5$), resulting in 32 exclusions. An additional 18 women either declined enrollment for other reasons or were lost to follow-up prior to enrollment. Thus, a total of 148 women were ultimately enrolled in the study and were classified into one of four groups based upon clinical interviews: current PTSD only ($n = 32$), current PTSD and current MDD ($n = 40$), current MDD only ($n = 24$), and the comparison group, which had neither PTSD nor MDD ($n = 52$). Women having current MDD only were not included in these analyses, resulting in 24 exclusions. The remaining 124 participants completed ambulatory blood-pressure monitoring and met criteria for inclusion in either the PTSD group or the comparison group. Since there are no empirically-based criteria for the optimal number of readings to be used to evaluate circadian blood pressure variation, we conducted the current analyses on women having at least 3 sleeping blood pressure readings and at least 10 waking blood pressure readings ($n = 61$). These criteria were chosen because they allowed us to maximize power while largely achieving consistency with the suggestion of O'Brien et al that at least 2/3 of ambulatory blood pressure readings are available for analysis³⁵. This guideline was achieved for the majority (79%) of participants in our sample. Excluded participants ($n = 63$), having less than 3 nighttime and 10 daytime blood pressure readings available for analyses, had a higher body mass index (BMI) ($M = 32.13$, $SD = 8.88$) than included participants ($M = 27.99$, $SD = 8.41$) $F(1, 122) = 7.09$, $p < .01$, but did not otherwise differ in terms of age, race, marital status, cardiovascular disease medication use, sedative hypnotic medical use, PTSD severity, or self-reported sleep quality.

Measures

Eligibility Measures

Clinical interviews were conducted by psychology graduate students and college-educated diagnostic raters ($n = 8$) who received extensive training (including didactic education and supervised experiential learning). Training involved observing and rating the administration of clinical interviews by trained interviewers; administering the interviews while being observed by a trained interviewer; attending regular meetings with the supervising psychologist and other staff to assure consistency across raters; and rating of 5 interview tapes for comparison against all other trained raters in the lab. All clinical evaluations were supervised by a licensed clinical psychologist. Inter-rater reliability among the raters was excellent (average kappa = .94).

Demographic and Deployment Characteristics—Study participants reported demographic information on a paper and pencil questionnaire.

PTSD Diagnosis—The Clinician Administered PTSD Scale (CAPS)³⁶ was used to determine PTSD diagnosis for purposes of study eligibility. The CAPS is a structured clinical interview evaluating PTSD symptoms over the past month, including the frequency and intensity of each symptom. The CAPS is considered to be the gold standard for the assessment of PTSD (National Center for PTSD, Boston, USA). It has demonstrated psychometric properties across a range of clinical populations and settings³⁷. In a series of studies conducted by Weathers and colleagues^{36, 38}, the CAPs demonstrated good internal consistency (Cronbach's alpha = 0.94) and test-retest reliabilities ranging from $r = .90$ to $r = .98$. Convergent validity has been demonstrated with the SCID³⁹ and with clinician-rated diagnoses⁴⁰. In the current study, the CAPs interview was discontinued upon meeting

minimal criteria for PTSD diagnostic category, or if the potential participant failed to meet a required criterion. For example, if the potential participant failed to endorse a criterion B symptom, the interview was discontinued as they could not then meet criteria for PTSD. Conversely, if the potential participant met an adequate number of criterion B symptoms for a diagnosis, the remaining item B criteria were not assessed, and the interviewer skipped to criterion C symptoms. Thus, the CAPS interview was conducted only for purposes of determining if PTSD diagnosis was present rather than determining the frequency and severity of each criterion.

Axis I Disorders—The Structured Clinical Interview for *DSM-IV* (SCID)⁴¹ was used to assess Axis I diagnoses other than PTSD. Current diagnosis was determined using a 1-month time frame for all disorders, except substance abuse for which we employed a 3-month time frame to define current substance abuse/dependence. The SCID is the most widely used clinical interview for the assessment of Axis I disorders in research settings, and has moderate to excellent inter-rater agreement for Axis I Disorders⁴².

Outcome Measures

Study participants completed the following self-report measures during their initial visit to the study laboratory.

Sleep Quality

Pittsburgh Sleep Quality Index (PSQI)⁴³: The PSQI, used to assess general sleep quality and disturbances over a 1-month interval, employs a self-rating scale. The PSQI includes 4 open-ended questions and 19 self-report ratings on a 0-3 scale. The PSQI is a widely used instrument in clinical sleep research and has established validity.

The PSQI yields both global and subscale scores of sleep quality, ranging from 0 to 21. In the validation study, the PSQI displayed high internal consistency (Cronbach's $\alpha=0.83$) and high test-retest reliability (Pearson correlation= 0.85), and a cut-off score of 5 was found to be both sensitive (89.6%) and specific (86.5%) for determining sleep quality relative to clinical and laboratory-based assessment measures⁴³. The PSQI has been evaluated in a range of populations, including older adults^{44, 45} depressed persons⁴⁶, primary insomnia⁴⁷, and medical populations⁴⁸. The one-year stability of PSQI scores was established in a sample of middle-aged adults⁴⁹.

PTSD Symptoms

Davidson Trauma Scale (DTS)⁵⁰: The DTS was used to assess PTSD symptom frequency and severity. It is a brief self-report measure assessing the 17 diagnostic symptoms of PTSD as outlined in the DSM-IV. Respondents rate each PTSD symptom on a 5-point scale in terms of both frequency (0="not at all" to "every day") and severity (0="not at all distressing" to 4="extremely distressing"). PTSD symptom cluster scores can be calculated from the DTS as well (i.e., intrusions, avoidance/numbing, and hyperarousal). In the DTS development sample, the measure demonstrated good test-retest reliability ($r=0.86$) and internal consistency (Cronbach's $\alpha=0.97$ for frequency; $\alpha=0.98$ for severity; and $\alpha=0.99$ for frequency + severity;). Convergent validity of the DTS was established against the SCID, with a total DTS score of 40 having a consistency of 83% with SCID diagnosis. Longitudinal analyses revealed the DTS to be predictive of symptom severity across time and sensitive to treatment effects.

Trauma Exposure

Traumatic Life Events Questionnaire (TLEQ)⁵¹: Exposure to trauma was assessed using the TLEQ, which is a 22-item scale assessing the occurrence, frequency, and distress associated with specific traumatic events. Respondents are asked the number of times that an event occurred, and whether Criterion A symptoms were present for each of 22 events. The primary objective of this measure is to capture exposure to a range of traumatic experiences. The authors report that the frequency of traumatic events endorsed in across several studies is considerably higher in the TLEQ relative to other inventories of trauma exposure. The authors of the measure established test-retest reliabilities across four populations, as follows: individuals receiving treatment for alcohol and drug abuse (83%); military Veterans (84%); battered women (86%); and college students (92%).

Ambulatory Blood Pressure Monitoring—Ambulatory blood pressure monitoring (ABPM) was conducted using either the Accutracker II (Suntech Medical Instruments, Raleigh, North Carolina) or the Spacelabs 90207 ambulatory recorder. These recorders have demonstrated reliability and validity^{52, 53}. The Spacelabs 90207 recorder was used for women with greater arm circumference (14% of the parent sample) for whom the Accutracker II did not provide valid readings. Participants were told to engage in typical daily activities while being monitored, to retain the non-dominant arm (with monitoring cuff) at their side whenever the recorder operated, and to wear the monitor for 24 hours. The monitor was programmed to collect measurements every 30 ± 5 minutes during waking hours, and every 90 minutes ± 5 minutes during sleep. Sleep and wake times were determined in advance on the basis of self-reported sleep/wake schedules. Ambulatory heart rate and systolic and diastolic blood pressure readings were obtained on each measurement occasion. Monitors were programmed to reassess blood pressure readings that were out of range. Each measurement occasion was rated as asleep or awake on the basis of self-reported sleep and wake times during the 24-hour monitoring period.

Covariates

The following variables which have established relationships with blood pressure were evaluated as potential covariates: age; body mass index; race; self-reported hypertension diagnosis; cardiac-affecting medication usage; sedative hypnotic medication usage; and acute stress during ABPM readings during the day preceding nighttime ABPM. Participants were designated as positive for “cardiac-affecting medications” if they endorsed use of medications in any of the following categories: beta-blockers; alpha adrenergics; anticholinergics; or other antihypertensives. Participants were classified as positive for “sedative hypnotic medication use if they endorsed use of any of the following medications: chloral hydrate; eszopiclone; zolpidem; or halcyon. Acute stress at the time of ABPM recordings was reported by participants. Each time the blood pressure recorder operated during waking hours, participants recorded their stress level on a scale from 1 to 5 (low stress to high stress) on a diary provided by research staff. The average of the acute stress readings on the day preceding the night of ABPM was used in the analyses described below.

Depression

Since a significant portion of the PTSD group met criteria for co-morbid depression, we also compared dippers to non-dippers on depression symptoms and diagnosis of major depressive disorder (MDD). Depression symptoms were assessed using the Beck Depression Inventory-II (BDI-II)⁵⁴ which is a 21-item inventory assessing depression. The BDI-II has been validated in both clinical and non-clinical populations. Scores on the BDI-II range from 0 to 63, with categorical ratings as follows: 0-13 (minimal depression); 14-19 (mild depression); 20-28 (moderate depression); and 29-63 (severe depression). The internal consistency

(Cronbach's $\alpha=0.91$) and test-retest reliability ($r=0.93$) of the BDI-II is well-established⁵⁵.

Diagnosis of MDD was established using the SCID (described above) during the eligibility screening procedure.

Procedure

On the first laboratory visit, participants were screened for eligibility with the CAPS and the SCID (and urine samples were collected for analysis as an objective measure of self-reported substance use). If found to be eligible, they also completed the TLEQ, DTS, and PSQI. On the second visit, which ranged from 2 days to 2 weeks after the initial visit, eligible participants arrived at the laboratory between 8:00 AM and 9:00 AM, during which time they were instructed in the use of their ambulatory blood pressure/heart rate monitor. As noted above, participants recorded their current stress level on a scale from 1 to 5 (low stress to high stress) on each time the blood pressure recorder operated during waking hours. Participants returned this diary, along with all study equipment, at a third and final laboratory visit. All study participants were compensated \$250 (\$50 for screening interview and \$200 at study completion).

Analyses

As consistent with previous sleep research³², we defined a nighttime blood pressure reading as one that occurred during the self-reported sleep period based upon sleep logs. All other readings were defined as a waking blood pressure reading. Dippers were defined as those having a day versus night difference in blood pressure greater than or equal to 10% of daytime blood pressure⁵. For Hypothesis 1, PTSD diagnostic status was compared to dipping status using standard Chi-square procedures for categorical measures. For Hypothesis 2, the *F* distribution was used to compare dippers to non-dippers on the following continuous variables for all women in the sample: trauma exposure, PTSD symptoms, and sleep quality. PTSD diagnostic status was not considered for Hypothesis 2 since our sample size was not sufficient to incorporate numerous interaction terms. Cohen's *d* effect sizes (ES) were calculated for each outcome by dividing the mean difference between groups on each continuous variable by the pooled standard deviation. Cohen⁵⁶ proposed the following guidelines for interpreting the *d* statistic: small ES=0.20; moderate ES=0.50, and large ES=0.80. The Phi coefficient was used to assess the strength of the relationship between binary variables. All analyses were conducted using SPSS Version 18. Missing data was only an issue for sleep quality analyses. Five study participants (4 in the PTSD group/1 in the control group) were missing data for individual items on the PSQI. Listwise deletion was used to address missing PSQI data, resulting in an *N* of 56 for analyses of the relationship between dipping status and sleep quality (Hypothesis 2).

Results

More than half of participants were minorities (51%), 38% were married, 64% were working, and 15% were military Veterans. Participants had an average of 16 years of education. Mean systolic blood pressure (SBP) and diastolic blood pressure (DBP) at night was 107 (SD=10.0) and 63 (SD=8.4), respectively. Mean SBP and DBP during the day was 120.3 (SD=11.4) and 74.6 (SD=8.8), respectively.

Table 1 summarizes comparisons between dippers (*N*=36) and non-dippers (*N*=25) on demographic and health characteristics. Dippers did not differ from non-dippers on age, BMI, depression symptoms, average acute stress, race, self-reported hypertension diagnosis, cardiac-affecting medication use, sedative-hypnotic medication use, psychiatric medication

use, use of Serotonin-Selective Reuptake Inhibitors (SSRIs), anti-anxiety medication use, or current Major Depressive Disorder diagnosis. Drugs included in the psychiatric medication category included the following: anti-depressants, anxiolytics, lithium, neuroleptics, sedative-hypnotics, stimulants and anti-convulsants. There was a small to moderate effect size for BMI differences between dippers and non-dippers with the latter having higher BMI.

Table 2 summarizes comparisons between dippers and non-Dippers on PTSD diagnosis, trauma exposure and PTSD symptoms. Almost all (60/61) participants endorsed exposure to at least one lifetime traumatic experience, and 54 of 61 (89%) endorsed at least one trauma meeting criterion A for PTSD diagnosis. Dipping status did not differ significantly by PTSD diagnostic status ($X^2=1.41$, $p=0.23$). However, non-dippers endorsed a higher number of categories of traumatic events meeting criterion A for a diagnosis of PTSD. There was a trend ($F=4.02$, $p=.05$) for greater frequency of PTSD symptoms in non-dippers relative to dippers. No differences were found between dippers and non-dippers on overall PTSD severity ($p=.13$). Non-dippers endorsed significantly more Criterion D hyperarousal symptoms ($F=4.39$, $p=.04$) than dippers. We also found a trend ($F=3.87$, $p=.05$) for more numbing/avoidance symptoms of PTSD in non-dippers than dippers.

Table 3 summarizes comparisons between dippers and non-dippers on sleep quality variables. Relative to dippers, non-dippers were found to have poorer overall sleep quality ($F=5.33$, $p=.03$), more frequent use of sleep medications ($F=6.26$, $p=.02$), greater sleep-related daytime dysfunction ($F=5.39$, $p=.02$), and greater sleep onset latencies ($F=4.02$, $p=.05$).

Since Major Depressive Disorder was highly co-morbid in the PTSD group, we conducted additional analyses to determine if women having the combination of depression and PTSD differed from women having PTSD only on dipping status. We found that women having the combination of PTSD and MDD did not differ from women having only PTSD on SBP dipping status ($\chi^2=.009$, $p=.923$).

Comment

In the current study, we hypothesized that there would be a higher proportion of women meeting criteria for a diagnosis of current PTSD among non-dippers than dippers (Hypothesis 1), and that non-dippers would endorse greater exposure to trauma, more PTSD symptoms, and lower sleep quality than dippers (Hypothesis 2). Our hypotheses were partially supported. The proportion of women with PTSD did not differ by nocturnal blood pressure dipping status. We did find, however, that non-dipping nocturnal blood pressure was associated with greater trauma exposure, more hyperarousal symptoms of PTSD, poorer overall sleep quality, more frequent sleep medication usage, greater sleep-related daytime dysfunction, and longer sleep onset latencies in a sample of women with and without current PTSD. Our findings are consistent with those of Mellman, Brown, Jenifer, Hipolito, & Randall (2009) wherein non-dipping was correlated with a diagnosis of lifetime PTSD and subthreshold lifetime PTSD symptoms. However, our findings differ in that our sample was comprised entirely of women, and all participants in the PTSD group met diagnostic criteria for current PTSD. The composition of our sample in terms of ethnicity differed from Mellman et al. (2009) as well. Our sample included only 38% African Americans. Although our study was not powered to include race as a covariate across analyses, we did not find differences in blood pressure dipping status by race. Our findings are also consistent with the literature on circadian blood pressure variation in non-PTSD samples, wherein non-dipping blood pressure was found to be associated with poorer sleep quality⁵⁷⁻⁵⁹. Interestingly, acute stress on the day preceding ABPM was strongly *unrelated* to dipping

status, suggesting that it may be the chronicity of stress which is most relevant to nocturnal blood pressure variation.

To our knowledge, our study is the first to examine nocturnal blood pressure non-dipping in a sample comprised of a significant portion of individuals meeting criteria for a diagnosis of current PTSD. Our finding, that the hyperarousal symptoms of PTSD have the strongest relationship to non-dipping blood pressure, is consistent with the literature reviewed above establishing a strong link between the circadian regulation of physiological arousal, nocturnal blood pressure variability and sleep. This finding is also consistent with those of Buckley et al⁶⁰ in their review of studies examining basal cardiovascular activity in adults with PTSD. Buckley and colleagues found evidence of elevated heart rate and blood pressure among those with PTSD across numerous studies. These studies used a range of assessment approaches, with only 8 using the preferred ambulatory monitoring approach. In a subsequent study, Buckley and colleagues⁶¹ employed 24-hour ambulatory monitoring and found that male Veterans with PTSD had higher resting heart rates and increased blood pressure reactivity relative to those without PTSD. The association of hyperarousal and non-dipping is also consistent with research in those having chronic insomnia. Lafranchi and colleagues⁶² found that, compared to good sleepers, those with chronic insomnia have lesser day-to-night systolic blood pressure decline and higher nighttime systolic blood pressure; evidence of increased hyperactivity of the central nervous system. In light of the association between symptoms of hyperarousal and non-dipping blood pressure, it is not surprising to have found a relationship between difficulties with sleep onset and non-dipping status since sleep onset difficulties are often related to cognitive and physiological arousal⁶³. Less clear is the significant finding of more frequent use of sleep medications in non-dippers. It may be that more frequent use of sleep medication is simply a proxy for more significant sleep difficulties. Conversely, it may be that the sleep medications themselves reduce blood pressure variability in some way. Future research is needed to further explore this finding.

As discussed above, we reported on a sample of women from the parent study in a previous paper. Women having a diagnosis of PTSD had lesser ability to regulate blood pressure, higher heart rates, and poorer sleep quality than women without PTSD. As such, our failure to find a clear relationship between PTSD diagnosis and non-dipping blood pressure in this same sample is somewhat unexpected. Exploring the relative contribution of various PTSD symptoms across both dipping status and BRS in those with PTSD is an interesting avenue for future research.

Our study is also the first to examine nocturnal blood pressure non-dipping and psychological trauma in women. Since previous research suggests the possibility of sex differences in the sleep architecture of those with PTSD⁶⁴, the use of a gender-homogenous sample eliminates statistical error introduced by the moderating effects of gender, if present. Future research should also examine the role of gender as moderator of associations between circadian blood pressure, sleep, and psychological trauma, and how these combined variables may be related to target organ damage. Indeed, some evidence suggests that the adverse impact of non-dipping blood pressure on target organs may be greater in women than in men⁶⁵. Future studies should utilize objective measures of sleep, and explore the possibility that PTSD symptoms and sleep quality may interact to predict nocturnal blood pressure dipping status. The small sample size of our study precluded a number of other interaction analyses that could have been helpful as well. Future studies should be powered to evaluate both PTSD diagnostic status and dipping status concurrently.

The findings of our research should be considered in light of several limitations. First, although there are no empirically-based criteria for the optimal number of readings to be used to evaluate circadian blood pressure variation, some have suggested that at least 2/3 of

ambulatory blood pressure readings are needed³⁵. Since only 79% of our sample met this criterion, we conducted sensitivity analyses to compare findings for our larger sample to those for the sample meeting the standard referenced above and did not find substantive differences in effect size across the 2 samples. However, it is still possible that our findings reflect Type I error; that by chance, we observed the hypothesized relationships in our subsample but they were not present in the original sample. The consistency of our findings with those of Mellman et al³² seems to argue against the likelihood of this possibility.

Second, the data used to conduct these analyses were drawn from a larger sample. As discussed above, the subsample meeting criteria for a minimum number of readings had a lower BMI than participants from the parent study whose data were excluded. Given the infancy of this area of research, the relevance of this finding is unclear. But, the established relationship between blood pressure and BMI could lead one to infer that the exclusion of these individuals might have impacted our findings in some way, including the possibility that differences in dipping status would have been more pronounced had these higher BMI participants been included in analyses.

Third, although we did not find differences between dippers and non-dippers on important covariates, a larger sample size would have facilitated concurrent evaluation of outcomes and covariates. Although research involving ambulatory blood pressure monitoring can involve high participant burden and cost, larger samples will be needed in future research to fully evaluate the role of covariates.

Finally, our findings should be considered in light of the limitations that are inherent to the use of a convenience sample, and the potential for self-selection bias of participants who are compensated for their involvement in research. In addition, causation cannot be inferred from these cross-sectional analyses.

In spite of the preliminary nature of our findings, we observed a moderate relationship between non-dipping blood pressure, trauma exposure, PTSD symptoms and sleep quality in a sample of women. O'Brien⁶⁶ argues that non-dippers should be targeted for more aggressive blood pressure control treatment. Since our findings suggest a possible relationship between non-dipping blood pressure and trauma in women, additional research is warranted to determine if this population requires increased attention to improve blood pressure control.

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References

1. Centers for Disease Control and Prevention. [Accessed October 5, 2012] Web-based Injury Statistics Query and Reporting System (WISQARS). Analysis of National Vital Statistics System 2007 data. at http://www.cdc.gov/injury/wisqars/leading_causes_death.html
2. Routledge F, McFetridge-Durdle JA, Dean CR. Night-time blood pressure patterns and target organ damage: A review. *Can J Cardiol.* 2007; 23:132–138. [PubMed: 17311119]

3. Verdecchia P, Angeli F, Gentile G, Mazzotta G, Gattobigio R, Reboldi G. Clinical Significance of the Blood Pressure Changes from Day to Night. *Curr Hypertens Rev.* 2011; 7:13–19.
4. O'Brien E, Sheridan J, O'Malley K. Dippers and non-dippers. *Lancet.* 1988; 2:397. [PubMed: 2899801]
5. Hansen T, Li Y, Boggia J, Thijs L, Richart T, Staessen JA. Predictive Role of the Nighttime Blood Pressure. *Hypertension.* 2011; 57:3–10. [PubMed: 21079049]
6. Sherwood A, Routledge FS, Wohlgemuth WK, Hinderliter AL, Kuhn CM, Blumenthal JA. Blood Pressure Dipping: Ethnicity, Sleep Quality, and Sympathetic Nervous System Activity. *Am J Hypertens.* 2011; 24:982–988. [PubMed: 21633397]
7. Sayk F, Teckentrup C, Becker C, et al. Effects of selective slow-wave sleep deprivation on nocturnal blood pressure dipping and daytime blood pressure regulation. *Am J Phys-Reg I.* 2010; 298:R191–R197.
8. Marrone O, Salvaggio A, Bue A, et al. Blood pressure changes after automatic and fixed CPAP in obstructive sleep apnea: relationship with nocturnal sympathetic activity. *Clin Exp Hypertens.* 2011; 33:373–380. [PubMed: 21529314]
9. Huang Y, Mai W, Hu Y, et al. Poor sleep quality, stress status, and sympathetic nervous system activation in nondipping hypertension. *Blood Press Monit.* 2011; 16:117–123. [PubMed: 21562453]
10. Dodt C, Breckling U, Derad I, Fehm HL, Born J. Plasma Epinephrine and Norepinephrine Concentrations of Healthy Humans Associated With Nighttime Sleep and Morning Arousal. *Hypertension.* 1997; 30:71–76. [PubMed: 9231823]
11. Holt-Lunstad J, Steffen PR. Diurnal Cortisol Variation is Associated With Nocturnal Blood Pressure Dipping. *Psychosom Med.* 2007; 69:339–343. [PubMed: 17470665]
12. Huang Y, Mai W, Cai X, et al. The effect of zolpidem on sleep quality, stress status, and nondipping hypertension. *Sleep.* 2012; 13:263–68.
13. Hildrum B, Romild U, Holmen J. Anxiety and depression lowers blood pressure: 22-year follow-up of the population based HUNT study, Norway. *BMC Public Health.* 2011; 11:601. [PubMed: 21797992]
14. Patten S, Williams JV, Lavorato DH, Campbell NR, Eliasziw M, Campbell TS. Major depression as a risk factor for high blood pressure: epidemiologic evidence from a national longitudinal study. *Psychosom Med.* 2009; 71:273–279. [PubMed: 19196807]
15. Hildrum B, Mykletun A, Holmen J, Dahl AA. Effect of anxiety and depression on blood pressure: 11-year longitudinal population study. *Brit J Psychiat.* 2008; 193:108–113.
16. Shinagawa M, Otsuka K, Murakami S, et al. Seven-day (24-h) ambulatory blood pressure monitoring, self-reported depression and quality of life scores. *Blood Press Monit.* 2002; 7:69–76. [PubMed: 12040249]
17. Scuteri A, Spalletta G, Cangelosi M, et al. Decreased nocturnal systolic blood pressure fall in older subjects with depression. *Aging-Clin Exp Res.* 2009; 21:292–297. [PubMed: 19959917]
18. Linden W, Klassen K, Phillips M. Can psychological factors account for a lack of nocturnal blood pressure dipping? *Ann Behav Med.* 2008; 36:253–258. [PubMed: 19067099]
19. Mezick E, Matthews KA, Hall M, et al. Low life purpose and high hostility are related to an attenuated decline in nocturnal blood pressure. *Health Psych.* 2010; 29:196–204.
20. Fallo F, Barzon L, Rabbia F, et al. Circadian blood pressure patterns and life stress. *Psychother Psychosom.* 2002; 71:350–356. [PubMed: 12411770]
21. Hickson D, Diez Roux AV, Wyatt SB, et al. Socioeconomic position is positively associated with blood pressure dipping among African-American adults: the Jackson Heart Study. *Amer J Hypertens.* 2011; 24:1015–1021. [PubMed: 21654853]
22. Fortmann A, Gallo LC, Roesch SC, et al. Socioeconomic Status, Nocturnal Blood Pressure Dipping, and Psychosocial Factors: A Cross-Sectional Investigation in Mexican-American Women. *Ann Behav Med.* 2012
23. Spruill T, Gerin W, Ogedegbe G, Burg M, Schwartz JE, Pickering TG. Socioeconomic and psychosocial factors mediate race differences in nocturnal blood pressure dipping. *Amer J of Hypertens.* 2009; 22:637–642. [PubMed: 19325537]
24. Stepnowsky C, Nelesen RA, DeJardin D, Dimsdale JE. Socioeconomic status is associated with nocturnal blood pressure dipping. *Psychosom Med.* 2004; 66:651–655. [PubMed: 15385687]

25. Neylan TC, Marmar CR, Metzler TJ, et al. Sleep Disturbances in the Vietnam Generation: Findings From a Nationally Representative Sample of Male Vietnam Veterans. *Amer J Psychiat*. 1998; 155:929–933. [PubMed: 9659859]
26. Granado NS, Smith TC, Swanson GM, et al. Newly Reported Hypertension After Military Combat Deployment in a Large Population-Based Study. *Hypertension*. 2009; 54:966–973. [PubMed: 19752293]
27. Boscarino JA. A Prospective Study of PTSD and Early-Age Heart Disease Mortality Among Vietnam Veterans: Implications for Surveillance and Prevention. *Psychosom Med*. 2008; 70:668–676.
28. Ulmer CS, Calhoun PS, Edinger JD, Wagner R, Beckham JC. Sleep Disturbance and Baroreceptor Sensitivity in Women with Posttraumatic Stress Disorder. *J Trauma Stress*. 2009; 22:643–647. [PubMed: 19960521]
29. La Rovere M, Pinna GD, Raczak G. Baroreflex sensitivity: Measurement and clinical implications. *Ann Noninvas Electro*. 2008; 13:191–207.
30. Beckham JC, Flood AM, Dennis MF, Calhoun PS. Ambulatory Cardiovascular Activity and Hostility Ratings in Women with Chronic Posttraumatic Stress Disorder. *Biol Psychiat*. 2009; 65:268–272. [PubMed: 18692171]
31. Calhoun PS, Wiley M, Dennis MF, Means MK, Edinger JD, Beckham JC. Objective Evidence of Sleep Disturbance in Women With Posttraumatic Stress Disorder. *J Trauma Stress*. 2007; 20:1009–1018. [PubMed: 18157880]
32. Mellman T, Brown DD, Jenifer ES, Hipolito MMS, Randall OS. Posttraumatic Stress Disorder and Nocturnal Blood Pressure Dipping in Young Adult African Americans. *Psychosom Med*. 2009; 71:627–630. [PubMed: 19483123]
33. Centers for Disease Control and Prevention. Perceived Insufficient Rest or Sleep Among Adults --- United States, 2008. *Morbidity and Mortality Weekly Report*. 2009; 58:1175–1179. [PubMed: 19875979]
34. Reckelhoff J. Gender Differences in the Regulation of Blood Pressure. *Hypertension*. 2001; 37:1199–1208. [PubMed: 11358929]
35. O'Brien E, Asmar R, Beilin L, et al. Practice guidelines of the European Society of Hypertension for clinic, ambulatory and self blood pressure measurement. *J Hypertens*. 2005; 23:697–701. [PubMed: 15775768]
36. Blake DD, Weathers FW, Nagy LM, Kaloupek DG, Gusman FD, Charney DS, Keane TM. The development of a Clinician-Administered PTSD Scale. *J Trauma Stress*. 1995; 8:75–90. [PubMed: 7712061]
37. Weathers FW, Keane TM, Davidson JR. Clinician-administered PTSD scale: a review of the first ten years of research. *Depress Anxiety*. 2001; 13:132–56. [PubMed: 11387733]
38. Mueser K, Rosenberg SD, Fox L, Salyers MP, Ford JD, Carty P. Psychometric evaluation of trauma and posttraumatic stress disorder assessments in persons with severe mental illness. *Psychol Assessment*. 2001; 13:110–117.
39. Foa E, Tolin DF. Comparison of the PTSD Symptom Scale–Interview Version and the Clinician-Administered PTSD Scale. *J Trauma Stress*. 2000; 13:181–191. [PubMed: 10838669]
40. Hovens JE, van der Ploeg HM, Klaarenbeek MT, Bramsen I, Schreuder JH, Rivero VV. The assessment of posttraumatic stress disorder with the Clinician Administered PTSD Scale: Dutch results. *J Clin Psychol*. 1994; 50:325–336. [PubMed: 8071438]
41. First, MB.; Spitzer, RL.; Gibbon, M.; Williams, JBW. Structured Clinical Interview for the DSM-IV Axis I Disorders. New York: Biometrics Research, New York State Psychiatric Institute; 1996.
42. Lobbetael J, Leurgans M, Arntz A. Inter-rater reliability of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID I) and Axis II Disorders (SCID II). *Clin Psychol Psychot*. 2011; 18:75–79.
43. Buysse DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: A new instrument for psychiatric practice and research. *Psychiat Res*. 1989; 28:193–213.
44. Spira A, Beaudreau SA, Stone KL, et al. A Reliability and validity of the Pittsburgh Sleep Quality Index and the Epworth Sleepiness Scale in older men. *J Gerontol A-Biol*. 2012; 67:433–439.

45. Beaudreau S, Spira AP, Stewart A, et al. Validation of the Pittsburgh Sleep Quality Index and the Epworth Sleepiness Scale in older black and white women. *Sleep Med.* 2012; 13:36–42. [PubMed: 22033120]
46. Hayashino Y, Yamazaki S, Takegami M, Nakayama T, Sokejima S, Fukuhara S. Association between number of comorbid conditions, depression, and sleep quality using the Pittsburgh Sleep Quality Index: results from a population-based survey. *Sleep Med.* 2010; 11:366–371. [PubMed: 20219425]
47. Backhaus J, Junghanns K, Broocks A, Riemann D, Hohagen F. Test-retest reliability and validity of the Pittsburgh Sleep Quality Index in primary insomnia. *J Psychosom Res.* 2002; 53:737–740. [PubMed: 12217446]
48. Carpenter J, Andrykowski MA. Psychometric evaluation of the Pittsburgh Sleep Quality Index. *J Psychosom Res.* 1998; 45:5–13. [PubMed: 9720850]
49. Knutson K, Rathouz PJ, Yan LL, Liu K, Lauderdale DS. Stability of the Pittsburgh Sleep Quality Index and the Epworth Sleepiness Questionnaires over 1 year in early middle-aged adults: the CARDIA study. *Sleep.* 2006; 29:1503–1506. [PubMed: 17162998]
50. Davidson JRT, Book SW, Colket JT, Tupler LA. Assessment of a new self-rating scale for post-traumatic stress disorder. *Psychol Med.* 1997; 27:153–160. [PubMed: 9122295]
51. Kubany ES, Haynes SN, Leisen MB, et al. Development and preliminary validation of a brief broad-spectrum measure of trauma exposure: the Traumatic Life Events Questionnaire. *Psychol Assess.* 2000; 12:210–24. [PubMed: 10887767]
52. Amore J, Geake WB. Evaluation of the Critikon 8100 and Spacelabs 90207 non-invasive blood pressure monitors using a test stimulator. *J Hum Hypertens.* 1997; 11:163–169. [PubMed: 9175568]
53. White W, Lund-Johansen P, McCabe EJ, Omvik P. Clinical evaluation of the Accutacker II ambulatory blood pressure monitor: Assessment of performance in two countries and comparison with sphygmomanometry and intra-arterial blood pressure at rest and during exercise. *J Hypertens.* 1989; 7:967–975. [PubMed: 2628497]
54. Beck, AT.; Steer, RA.; Brown, GK. Beck Depression Inventory Manual-2nd Edition. San Antonio, Tx: Psychological Corporation; 1996.
55. Dozois, DJA.; Covin, R. The Beck Depression Inventory-II (BDI-II), Beck Hopelessness Scale (BHS), and Beck Scale for Suicidal Ideation (BSS). In: Hersen, M., editor. *Comprehensive Handbook of Psychological Assessment*. John Wiley & Sons; Hoboken, NJ: 2004.
56. Cohen, J. *Statistical power analysis for the behavioral sciences*. 2nd. Hillsdale, NJ: Lawrence Earlbaum Associates; 1988.
57. Pedulla M, Silvestri R, Lasco A, et al. Sleep structure in essential hypertensive patients: differences between dippers and non-dippers. *Blood Press.* 1995; 4:232–237. [PubMed: 7496562]
58. Sherwood A, Routledge FS, Wohlgemuth WK, Hinderliter AL, Kuhn CM, Blumenthal JA. Blood pressure dipping: ethnicity, sleep quality, and sympathetic nervous system activity. *Amer J Hypertens.* 2011; 24:982–988. [PubMed: 21633397]
59. Loreda J, Nelesen R, Ancoli-Israel S, Dimsdale JE. Sleep quality and blood pressure dipping in normal adults. *Sleep.* 2004; 27:1097–1103. [PubMed: 15532203]
60. Buckley TC, Kaloupek DG. A meta-analytic examination of basal cardiovascular activity in posttraumatic stress disorder. *Psychosom Med.* 2001; 63:585–594. [PubMed: 11485112]
61. Buckley TC, Mozley SL, Bedard MA, Cecile Dewulf A, Greif J. Preventive Health Behaviors, Health-Risk Behaviors, Physical Morbidity, and Health-Related Role Functioning Impairment in Veterans with Post-Traumatic Stress Disorder. *Mil Med.* 2004; 169:536–40. [PubMed: 15291186]
62. Lanfranchi P, Pennestri MH, Fradette L, Dumont M, Morin CM, Montplaisir J. Nighttime blood pressure in normotensive subjects with chronic insomnia: implications for cardiovascular risk. *Sleep.* 2009; 32:760–766. [PubMed: 19544752]
63. Wuyts J, De Valck E, Vandekerckhove M, et al. The influence of pre-sleep cognitive arousal on sleep onset processes. *Int J Psychophysiol.* 2012; 83:8–15. [PubMed: 21963535]
64. Kobayashi I, Boarts JM, Delahanty DL. Polysomnographically measured sleep abnormalities in PTSD: A meta-analytic review. *Psychophysiology.* 2007; 44:660–669. [PubMed: 17521374]

65. Verdecchia P, Schillaci G, Boldrini F, Guerrieri M, Porcellati C. Sex, cardiac hypertrophy and diurnal blood pressure variations in essential hypertension. *J Hypertens.* 1992; 10:683–692. [PubMed: 1321197]
66. O'Brien E. Dipping Comes of Age-The Importance of Nocturnal Blood Pressure. *Hypertension.* 2009; 53:446–447. [PubMed: 19171787]

Figure 1.

Table 1
Comparing SBP Non-Dippers to Dippers on Demographic and Health Characteristics

Measure	Combined N=61	Non-Dippers N=25	Dippers N=36	Statistic	Significance	Effect Size
	Mean (SD)	Mean (SD)	Mean (SD)	F	p	d
Age	38.28 (12.08)	40.16 (11.98)	36.97 (12.15)	1.03	0.32	0.26
Body Mass Index	27.99 (8.41)	30.08 (10.87)	26.53 (5.91)	2.70	0.11	0.42
Beck Depression Inventory Score	12.88 (10.49)	15.33 (10.93)	11.19 (9.98)	2.35	0.13	0.39
Acute Stress Score during ABPM	1.67 (.57)	1.68 (.62)	1.66 (.55)	0.00	0.99	0.04
	within group	within group	within group	χ^2	P	ϕ
Race				0.72	0.70	0.11
African American %	38	44	33			
Caucasian %	49	44	53			
Other %	13	12	14			
Hypertension Diagnosis % (N)	13 (8)	12 (3)	14 (5)	0.05	0.83	0.03
Cardiac-Affecting Medication % (N)	19.7 (12)	20 (5)	19.4 (7)	<0.01	0.96	<0.01
Sedative Hypnotic Medication % (N)	1.6 (1)	4 (1)	0 (0)	1.46	0.23	0.46
Psychiatric Medication % (N)	26.2 (16)	56.3 (9)	43.8 (7)	2.09	0.15	0.50
SSRI % (N)	14.8 (9)	24.0 (6)	8.3 (3)	2.88	0.09	0.35
Anti-Anxiety Medication % (N)	13.1 (8)	16.0 (4)	11.1 (4)	0.31	0.58	0.32
Major Depression Diagnosis % (N)	37.7 (23)	44 (11)	33.3 (12)	0.72	0.40	0.11

Cohen's d-small=0.20, moderate=0.50, large=0.80, SBP=Systolic Blood Pressure, SSRI=Selective Serotonin Reuptake Inhibitor.

Table 2
Comparing SBP Non-Dippers to Dippers on PTSD Diagnosis, Trauma Exposure and PTSD Symptoms

Measure	Combined Sample N=61	Non-Dippers N=25	Dippers N=36	Statistic	Effect Size
	within group % (N)	within group % (N)	within group % (N)	χ^2	ϕ
PTSD Diagnosis	59 (36)	68 (17)	53 (19)	1.41	0.23
	Mean (SD)	Mean (SD)	Mean (SD)	F	d
Criterion A traumatic events	5.77 (4.71)	7.24 (4.99)	4.75 (4.20)	4.35*	.04
DTS-Total Score	39.87 (34.98)	49.32 (33.74)	33.31 (34.76)	3.21	.08
DTS-Frequency of Sx	19.51 (17.21)	24.68 (17.21)	15.92 (16.49)	4.02 [^]	.05
DTS-Severity of Sx	20.36 (18.17)	24.64 (17.00)	17.39 (18.59)	2.41	.13
DTS-Criterion B Intrusion Sx	10.30 (9.87)	11.40 (8.70)	9.53 (10.66)	0.53	.47
DTS-Criterion C Numbing/Avoidance Sx	16.69 (15.96)	21.40 (16.87)	13.42 (14.65)	3.87 [^]	.05
DTS-Criterion D Hyperarousal Sx	12.89 (11.61)	16.52 (10.86)	10.36 (11.58)	4.39*	.04

[^] p=.05

* p<.05

Cohen's d-small=0.20, moderate=0.50, large=0.80, SBP=Systolic Blood Pressure, Sx=Symptoms

Table 3

Comparing SBP Non-Dippers to Dippers on Sleep Quality

Measure	Combined Sample <i>N</i> =56	Non-Dippers <i>N</i> =25	Dippers <i>N</i> =31	Statistic	Significance	Effect Size
	Mean (SD)	Mean (SD)	Mean (SD)	<i>F</i>	<i>p</i>	<i>d</i>
PSQI Subjective Sleep Quality	1.27 (0.90)	1.40 (0.87)	1.16 (0.93)	0.96	0.33	0.27
PSQI Sleep Latency	1.30 (1.11)	1.64 (1.15)	1.03 (1.02)	4.40*	0.04	0.55
PSQI Sleep Duration	1.16 (0.93)	1.32 (0.95)	1.03 (0.91)	1.33	0.25	0.31
PSQI Habitual Sleep Efficiency	0.83 (1.08)	1.04 (1.21)	0.68 (0.94)	1.59	0.21	0.33
PSQI Sleep Disturbances	1.50 (0.74)	1.64 (0.70)	1.39 (0.76)	1.64	0.21	0.34
PSQI Use of Sleep Medications	0.66 (1.12)	1.04 (1.31)	0.36 (0.84)	5.65*	0.02	0.61
PSQI Daytime Dysfunction	1.05 (0.88)	1.36 (0.95)	0.81 (0.75)	5.93*	0.02	0.63
PSQI Total Score	7.79 (5.05)	9.44 (5.34)	6.45 (4.45)	5.22*	0.03	0.59

* *p*<.05Cohen's *d*-small=0.20, moderate=0.50, large=0.80, SBP=Systolic Blood Pressure