Research Paper

Objective short sleep duration and 24-hour blood pressure

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ABSTRACT

Background: Short sleep duration is a contributor to cardiovascular disease (CVD) events and mortality. Short sleep duration is associated with an increased risk of high clinic blood pressure (BP). BP measured outside the clinic using 24-h ambulatory blood pressure monitoring (ABPM) is a better predictor of an individual's CVD risk. We examined the association between objectively-assessed sleep duration and 24-h ambulatory blood pressure (ABP).

Methods: A total of 893 working adults underwent sleep and ABPM. Participants were fitted with an ABPM device, and measures were taken at 28–30 min intervals. Objective sleep duration, and times of wakefulness and sleep during the 24-h ABPM period were derived from wrist-worn actigraphy. Linear regression, adjusted for age, sex, race/ethnicity, body mass index, smoking status, and diabetes were conducted on the relationship between sleep duration and the ABP measures.

Results: Mean age of participants (final n = 729, 59.5% female, 11.9% Hispanic) was 45.2 ± 10.4 y. Mean actigraphy-derived sleep duration was 6.8 ± 1.2 h. Sleep duration <6 h was associated with a 1.73 mmHg higher 24-h systolic BP (p = 0.031) and 2.17 mmHg higher 24-h diastolic BP (p < 0.001). Shorter sleep duration was not associated with mean awake or asleep systolic BP (p = 0.89 and p = 0.92) or mean awake or asleep diastolic BP (p = 0.30 and p = 0.74).

Conclusions: To our knowledge, this is the largest study conducted which assessed sleep duration objectively while measuring 24-h ABP. Shorter sleep duration is associated with higher 24-h BP and potentially cardiovascular risk.

1. Introduction

Ambulatory blood pressure (ABP) refers to blood pressure (BP) measured outside of clinic in the naturalistic environment using an ABP monitor (ABPM), typically over a 24 h period [1]. ABPM can identify several BP phenotypes not captured by clinic BP (CBP) alone, including the 24-h BP profile [1]. High 24-h BP is associated with cardiovascular disease (CVD) events and mortality and has a stronger association with CVD than CBP [1]. Recent guidelines recommend assessment of 24-h BP [2]. Since high 24-h BP may precede the development of high CBP [2], understanding behavioral risk factors for high 24-h BP may confer opportunities for risk modification before the onset of clinic hypertension.

Short sleep duration, which affects ~30% of US adults [3], is a known contributor to CVD events and mortality [3,4]. Self-reported short sleep is linked to increased prevalence and incidence of high CBP [3], but few studies examined the association between sleep duration and ABP [5]. Most prior studies are limited to pediatric/adolescent populations [6,7] or included individuals with prehypertension/stage 1 hypertension or obstructive sleep apnea (OSA) [8], which is itself associated with high 24-h BP [9]. Importantly, most studies assessed sleep duration via self-report [6,8] which is subject to recall bias compared to objective methods like wrist actigraphy [10]. Only one study [11] examined objective sleep duration and ABP among adults, but did not report the association of 24-h BP with sleep. We therefore examined the association between objective sleep duration and 24-h BP in an adult multi-ethnic community sample.

2. Methods

Data come from the Masked Hypertension (MHT) Study, described in detail elsewhere [12]. This was a multi-site study of the prevalence and predictors of masked hypertension (i.e., ambulatory hypertension despite non-high CBP) [1], that enrolled 1011 adults from the New York Metropolitan area. Inclusion criteria were: age ≥21 years, working ≥17.5 h/week, and proficiency in spoken and written English. Exclusion criteria were: screening systolic BP (SBP) > 160 mmHg or diastolic BP (DBP) > 105 mmHg, use of antihypertensive or cardiovascular medications (other than statins), history of overt CVD (myocardial infarction, stroke, percutaneous transluminal coronary angioplasty), presence of chronic renal, liver, thyroid, or adrenal disease, cancer not in remission for ≥6 months, pregnancy, active substance abuse, or serious mental health illness.

Participants attended several laboratory visits over a 4-week period. Three auscultatory BP readings were taken using a mercury sphygmomanometer at each visit. CBP was calculated as the mean of the three readings at Visit 1. Two weeks later, 893 participants were fitted with an ABPM (Model 90,207; Spacelabs, Snoqualmie, WA) and a wrist-worn actigraphy device (ActiWatch; Phillips Respironics, Bend, OR) to measure sleep. Devices were returned 24 h later.

ABP measurements were taken at 28-min intervals over a 24-h period during a workday. Participants completed an ABPM log, including bedtime, waketime, naps, and if they removed equipment (e.g., to exercise or shower). Sleep duration, and times of wakefulness and sleep during the 24-h ABPM period, were defined by actigraphy, supplemented by diary reports. Night shift work was defined as a work shift that was self-reported to start at, or after, 11:00pm and ended at, or before, 7:00am [13]. A minimum of 10 valid ABP readings was required to compute mean awake ABP and a minimum of 5 valid sleep readings was required to compute mean sleep ABP in accordance with the International Database of Ambulatory Blood Pressure in Relation to Cardiovascular Outcome [IDACO] criteria [14]. Of 893 participants, those who had <10 valid awake readings or <5 sleep readings (n = 121), or were missing data (n = 33 and n = 10 missing body mass index [BMI] and Pittsburgh Sleep Quality Index [PSQI], respectively) were excluded. A total of 729 participants with complete data were included in these primary analyses. As a sensitivity analysis, we replicated our analyses using the 2013 European Society of Hypertension (ESH) [15,16] criteria to define a complete 24-h ABPM recording. These criteria require ≥70% of targeted readings with a minimum of 20 awake and 7 sleep readings. Use of these more stringent criteria excluded an additional 38 participants, reducing the sample size from 729 to 691 for this sensitivity analysis. Procedures were approved by the Institutional Review Boards of Columbia University Medical Center and Stony Brook University and all participants provided written informed consent. Descriptive statistics are expressed as mean ± standard deviation or percent. Linear regression, adjusted for age, sex, race/ethnicity, BMI, smoking status, and diabetes were conducted to predict ABP measures from sleep duration. Supplemental models corrected for CBP and self-reported sleep quality (PSQI rating). Sensitivity analyses were repeated using ESH [15,16] criteria to predict ABP from sleep duration. Analyses were performed with SAS version 9.4 (SAS Institute Inc, Cary, NC).

3. Results

Participants (n = 729, 59.5% female,11.9% Hispanic, 1.2% night shift work) were 45.2 ± 10.4 years old (Table 1). Mean sleep duration was 6.8 ± 1.2 h, with 22% of participants sleeping <6 h. Poor sleep quality (PSQI score >5) was reported in 43.4% of participants. Mean awake SBP/DBP was 123.0 ± 10.3/77.4 ± 7.4 mmHg, mean sleep SBP/DBP was 106.2 ± 10.5/61.9 ± 7.6 mmHg, and mean 24-h BP was 118.2 ± 9.8/73.0 ± 7.1 mmHg.

In adjusted analyses, sleep duration was not associated with mean awake or sleep SBP (p = 0.89 and p = 0.92) or mean awake or sleep DBP

Table 1
Participant characteristics.

	N = 729
Age, years	45.2 ± 10.4
Female sex, %	59.5
Race/Ethnicity	
Black, %	6.3
Hispanic, %	11.9
BMI, kg/m^2	27.6 ± 5.4
Total cholesterol, mg/dL	193.33 ± 36.73
High-density lipoprotein cholesterol, mg/dL	58.22 ± 16.20
Low-density lipoprotein cholesterol, mg/dL	114.50 ± 33.45
Diabetes, %	3.7
Framingham Risk Score	2.06 ± 2.89
Mean sleep duration, hours	6.8 ± 1.2
Short sleep (< 6 h), %	22.0
Mean PSQI score	5.7 ± 3.3
Poor sleep quality (PSQI >5), %	43.4
Night shift work, %	1.2
Mean clinic BP	
Systolic, mmHg	114.2 ± 12.9
Diastolic, mmHg	74.6 ± 9.3
Mean awake ambulatory BP	
Systolic, mmHg	123.0 ± 10.3
Diastolic, mmHg	77.4 ± 7.4
Mean sleep ambulatory BP	
Systolic, mmHg	106.2 ± 10.5
Diastolic, mmHg	61.9 ± 7.6
Mean 24-h ambulatory BP	
Systolic, mmHg	118.2 ± 9.8
Diastolic, mmHg	73.0 ± 7.1

Data are expressed as percentage or mean ± SD. BMI: Body mass index; BP: Blood pressure; PSQI: Pittsburgh Sleep Quality Index.

(p = 0.30 and p = 0.74, Table 2). Shorter sleep duration predicted higher mean 24-h SBP, B = −0.61 mmHg/h, p = 0.031 and higher mean 24-h DBP, B = −0.80 mmHg/h, p < 0.001. Short (<6 h) sleep vs. ≥6 h was associated with a 1.73 mmHg higher 24-h SBP (p = 0.031) and 2.17 mmHg higher 24-h DBP (p < 0.001). The association remained statistically significant after adjusting for CBP (24-h SBP, B = −0.60 mmHg/h, p = 0.006; 24-h DBP, B = −0.96 mmHg/h, p < 0.001) or, separately, PSQI score (24-h SBP, B = −0.61 mmHg/h, p = 0.031; 24-h DBP, B = −0.80 mmHg/h, p < 0.001).

Supplemental Table 1 depicts patient characteristics (n = 691) when analyses were repeated using the ESH [15,16] criteria to define a complete ABPM period. As in the primary analyses, sleep duration was not associated with mean awake or sleep SBP (p = 0.67 and p = 0.78) or mean awake or sleep DBP (p = 0.54 and p = 0.74, Supplemental Table 2). Shorter sleep duration predicted higher mean 24-h DBP, B = −0.70 mmHg/h, p = 0.002. Although shorter sleep duration no longer predicted mean 24-h SBP, B = −0.53 mmHg/h, p = 0.072, the coefficient was similar in magnitude to our primary analyses (−0.61 mmHg/h, p = 0.031, Table 2). Short (<6 h) sleep vs. ≥6 h was associated with a 1.34 mmHg higher 24-h SBP (p = 0.106) and 1.86 mmHg higher 24-h DBP (p = 0.003). The association between sleep duration and 24-h DBP remained statistically significant after adjusting for

Table 2
Estimates (95% confidence interval) of mean sleep, awake, and 24-h ambulatory blood pressure associated with sleep duration (hours).

	Beta-coefficient (95% CI)	P-value
Mean awake systolic blood pressure	0.04 (−0.54, 0.63)	0.89
Mean awake diastolic blood pressure	−0.24 (−0.68, 0.21)	0.30
Mean sleep systolic blood pressure	−0.03 (−0.64, 0.57)	0.92
Mean sleep diastolic blood pressure	−0.08 (−0.54, 0.38)	0.74
Mean 24-h systolic blood pressure	−0.61 (−1.16, −0.06)	0.031
Mean 24-h diastolic blood pressure	−0.80 (-1.22, -0.38)	<0.001

Adjusted for age, sex, race/ethnicity, body mass index, smoking status, and diabetes status.

for CBP (24-h DBP, B = −0.92 mmHg/h, p < 0.001) and, separately, PSQI score (24-h DBP, B = −0.70 mmHg/h, p = 0.002).

4. Discussion

Results demonstrate that actigraphy-derived sleep duration is associated with 24-h mean ABP among working adults not on antihypertensive medications. Short sleep (<6 vs. ≥6 h) was associated with ~2 mmHg higher systolic and diastolic 24-h BP. Shorter sleep duration was not associated with awake or sleep ABP.

Both 24-h ABP [17] and short sleep [3,5] are associated with increased CVD events and mortality. Yet, studies examining the association between sleep and 24-h BP among healthy adults are scarce. In a crossover study of 36 adults with newly diagnosed clinic hypertension, 24-h BP was higher during 1 week of sleep restriction (4 h/night) vs. 1 week of full sleep (7 h/night) [18]. In participants with prehypertension (n = 66) and OSA (n = 153), there was an association between self-reported shorter sleep duration and 24-h ABP [8]. However, self-reported and objectively-measured sleep duration are weakly correlated [10]. Further, as prehypertension/stage 1 hypertension and OSA are associated with high 24-h BP levels [9], the association between self-reported sleep duration and 24-h BP reported in these studies may have been confounded. Our study extends previous work by using objectively assessed measures of sleep duration and examining this association in a large multi-ethnic community sample of adults not on antihypertensive medications.

Shorter sleep duration was not associated with awake or sleep ABP in this study including in sensitivity analyses when using ESH [15,16] criteria to define a complete ABPM period. Different criteria have been proposed for defining a complete 24-h ABPM [14,16,19–22]. The ESH criteria are more stringent than the IDACO criteria as they require a higher number of readings to define a complete ABPM recording. Consequently, fewer individuals will meet ESH criteria and therefore the overall sample size will be smaller which may affect generalizability of results [19]. While the association between sleep duration and 24-h SBP was no longer statistically significant in our study utilizing ESH criteria, the magnitude of the effect was similar to our primary analyses. Further, shorter sleep duration still predicted higher 24-h DBP when using the ESH criteria to define a complete ABPM recording.

The observed association between shorter sleep and 24-h BP may be explained by the fact that awake BP is higher than sleep BP in most individuals [1] and awake BP contributes proportionately more to the 24-h mean BP in those with short (er) sleep. Thus, spending more time awake (as occurs with shorter sleep duration) likely contributes to higher overall 24-h BP [23].

To our knowledge, this is the largest study which simultaneously assessed objective sleep duration and 24-h ABP. However, these data are cross-sectional, and actigraphy was conducted for 24 h so may not reflect habitual sleep. Further, our sample was relatively healthy and is approximately representative of US adults with no history of CVD who do not take antihypertensive medication. Despite our sample including young participants (≥21 years old) with an average age of 45.2 years old, surprisingly, 43.4% of participants reported poor sleep quality. Prior studies have demonstrated that older adults, rather than younger adults, report poor sleep quality [24,25]. Poor sleep quality has been associated with high ambulatory BP [11]. Although there was a high prevalence of self-reported poor sleep quality in our sample, the association between sleep duration and 24-h BP remained statistically significant even after adjustment for self-reported poor sleep quality. Lastly, our sample also included a small number of night shift workers. Since night shift work requires that shift workers remain awake at night and sleep during daylight hours, night shift workers have an altered sleep-wake cycle and circadian pattern of sleep [26]. Night shift workers also have a disrupted circadian pattern of BP [26] and night shift work has been associated with high awake BP and 24-h BP [27,28]. In our study,

less than 2% were night shift workers and therefore it remains unlikely that the association between sleep duration and 24-h BP was driven by this small subset of participants.

In conclusion, these findings indicate that shorter sleep duration is associated with higher 24-h ABP and potentially cardiovascular risk. Short sleep duration may therefore be a modifiable behavioral target for reducing 24-h ABP.

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Author statements

Marwah Abdalla MD: Conceptualization, Methodology, Writing-Original draft preparation, Writing- Reviewing and Editing.

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Conflict of interest

There are no conflicts of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijchy.2020.100062.

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