HHS Public Access

Author manuscript

J Sleep Res. Author manuscript; available in PMC 2021 December 01.

Published in final edited form as:

J Sleep Res. 2020 December ; 29(6): e12988. doi:10.1111/jsr.12988.

Associations of objectively measured sleep characteristics and incident hypertension among police officers: the role of obesity

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Summary

This study investigated the associations of baseline sleep onset latency, wake after sleep onset, longest wake episode, number of awakenings, sleep efficiency, and sleep duration with incident hypertension during a seven-year follow-up (n=161, 68% men) and the joint effect of insufficient sleep and obesity on incident hypertension. Sleep parameters were derived from 15-day actigraphy data. Relative risks and 95% confidence intervals were estimated using robust Poisson regression model. Each 10-minute increase in sleep onset latency was associated with an 89% higher risk of hypertension (95% confidence interval=1.12–3.20). Each 10-minute increase in longest wake episode was associated with a 23% higher risk of hypertension (95% confidence interval=1.01–1.50) and each 10% decrease in sleep efficiency was associated with a 50% higher risk of hypertension (95% confidence interval 1.02–2.22). These associations were independent of demographic and lifestyle characteristics, depressive symptoms, shift work, sleep duration, and body mass index. Having <6 hours of sleep and a body mass index ≥30 kg/m^2 increased the risk of hypertension (relative risk=2.81; 95% confidence interval=1.26–6.25) than having ≥6 hours of sleep and a body mass index <30 after controlling for confounders. Relative excess risk due to interaction was 3.49 (95% confidence interval=−1.69–8.68) and ratio of relative risk was 3.21 (95% confidence interval=0.72–14.26). These results suggest that poor sleep quality is a risk factor for hypertension. Longitudinal studies with larger sample sizes are warranted to examine the joint effect between insufficient sleep and obesity on development of hypertension.

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Author contributorship:

CCM conceived the research question, drafted the manuscript and revised the manuscript extensively for its important intellectual contents; JKG performed data analyses and revised the paper critically; RB made contributions to the data analysis and critically edited the intellectual contents of the manuscript; LEC, JMV, DF, and MEA made contributions to acquisition of data and revised the manuscript extensively for its important intellectual contents. All the authors approved the final version of the paper.

Conflict of interest: none declared

Keywords

sleep initiation; sleep maintenance; additive interaction; multiplicative interaction

Introduction

Sleep is essential to maintaining good physical and mental health. The literature on sleep physiology shows that nonrapid and rapid eye movement alternate cyclically during sleep and that each stage is associated with distinct brain activities and physiological changes such as blood pressure and heart rate fluctuation (Institute of Medicine Committee on Sleep & Research, 2006). Previous studies have suggested that sleep disorders (such as difficulty initiating and maintaining sleep) are associated with hypertension (Seravalle, Mancia, & Grassi, 2018; Thomas & Calhoun, 2017) and sleep deprivation is a risk factor for hypertension (Itani, Jike, Watanabe, & Kaneita, 2017; Makarem et al., 2019; Tobaldini et al., 2017).

Although there have been quite a few epidemiological studies that have reported associations of sleep duration and quality with hypertension (Lo, Woo, Wong, & Tam, 2018), to our knowledge, only a few prospective studies could be identified (Clark et al., 2016; Fernandez-Mendoza et al., 2012; Knutson et al., 2009; Lin, Liu, Lin, Chung, & Chien, 2017; Wang et al., 2017). Among the few prospective studies, the accuracy of self-reported sleep might be affected by physical and psychological factors (Matthews et al., 2018). In addition, the available evidence may be not applicable to police officers, a unique occupational group with a high level of stress and a high percentage of shift work, both of which have been associated with poor sleep quality (Fekedulegn et al., 2016; Ma et al., 2019).

The epidemic of obesity has become one of the major public health issues in the United States and has proportionately increased with that of short sleep duration over the past decade. A few epidemiological studies have shown the association of obesity with insufficient sleep duration (i.e., < 7 hours) to be prospectively bidirectional (Koolhaas et al., 2019). On one hand, shorter sleep duration is associated with higher body mass index (BMI) and on the other hand, a higher BMI is associated with a shorter sleep duration after six years. It is well known that obesity has a synergistic effect with obstructive sleep apnea (OSA) on hypertension and cardiovascular disease (Carter & Watenpaugh, 2008) but the joint effect of insufficient sleep and obesity in hypertension is less well known.

The objectives of the present study were to investigate (1) if sleep quality at baseline was associated with subsequent incident hypertension and (2) if there was a joint effect between insufficient sleep and obesity at baseline on the risk of hypertension after an average of seven-year follow-up among police officers. We hypothesized that 1) poorer objectively measured sleep quality, indicated by longer sleep onset latency, longer awake time after sleep onset or longer duration of the longest wake episode, more awakenings, lower sleep efficiency, and shorter sleep duration at baseline, would be associated with a higher risk of hypertension; and 2) insufficient sleep (< 6 hours) and obesity (BMI ≥ 30 kg/m^2) at baseline would have a joint effect on the development of hypertension.

Methods

Study Design and Participants

The police officers who participated in the Buffalo Cardio-metabolic Occupational Police Stress (BCOPS) Study at baseline (2004–2009) and the first follow-up examination (2010–2014) comprised the study sample for the present analyses. The objectives of the BCOPS Study have been described elsewhere (Violanti et al., 2006). Briefly, it is an ongoing collaboration between the National Institute for Occupational Safety and Health (NIOSH) and colleagues at the University at Buffalo to study the effects of policing and occupational stress on adverse metabolic and subclinical cardiovascular outcomes. All participants provided informed consent at each visit. Institutional Review Boards of the State University of New York at Buffalo and NIOSH approved both the baseline and the follow-up studies.

At baseline, 464 police officers were examined. Among these officers, 276 returned for the follow-up exam. For the present analysis, officers who had hypertension or had been taking any antihypertension medication at baseline (n = 58) and those who had missing information on sleep measures (n = 57) at baseline were excluded, resulting in a final study sample of 161 (68% men).

Assessment of Hypertension

At each visit, officers completed a six-hour comprehensive examination that included interviewer-instructed questionnaires and physiologic assessments. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured in the supine position at baseline and the seated position at follow-up for each participant after they had rested for five minutes. Three measurements of SBP and DBP were taken at a two-minute interval at each visit which were averaged to obtain the final values (Gu et al., 2018). Information on antihypertensive medications was collected through an at-home questionnaire that asked participants to list all the prescribed and over-the-counter medications taken in the past 30 days prior to the examination. Hypertension (at baseline and follow-up) was defined as SBP of 140 mmHg or higher, DBP of 90 mmHg or higher, or taking antihypertensive medications (James et al., 2014).

Objective Sleep Measures

Each participant was instructed to wear a sleep watch (actigraph) on his/her non-dominant wrist for an average of 15 days (range: 3–24 days). The actigraphy data were collected using the Micro Motionlogger Sleep Watch™ (Ambulatory Monitoring Inc., NY) and an Octagonal Motionlogger Computer Interface with ACT #25.111PS and the first version of analysis software Action4 (cat.# 21.123, Ambulatory Monitoring, Inc., NY). The Cole Kripke algorithm was used for sleep scoring (Cole, Kripke, Gruen, Mullaney, & Gillin, 1992).

Each of the sleep parameters included sleep onset latency, wake after sleep onset, number of awakenings, and sleep efficiency was defined using the definition reported by the US Sleep Foundation (Ohayon et al., 2017). Briefly, sleep onset latency is the time that it takes a participant to fall asleep in bed. Participants were instructed to press an event marker button

of the actigraph when lying down in bed and when the lights were turned off. Technically, we calculated sleep onset latency as the number of minutes from the time the subject lay down in bed to the time the subject was first scored as asleep by the algorithm. Wake after sleep onset is the total amount of awake time between the first persistent sleep onset and the final awakening excluding sleep onset latency. Number of awakenings refers to the number of awake episodes where the officer was awake for longer than five minutes. Sleep efficiency is the percentage of total sleep time during the time spent in bed (i.e., the interval from the time a participant went to bed to the time the participant arose). Sleep duration refers to the number of hours slept during time in bed and did not include napping during work.

In addition to the aforementioned sleep parameters, the longest wake episode was also derived as an additional measure of sleep maintenance since the wake after sleep onset might be affected by family responsibilities such as caring for children or sick family members, etc. The longest wake episode might be a stronger indicator of poor sleep quality. Each sleep parameter was computed by averaging the daily value over the period when the sleep watch was worn.

Confounders

Potential confounders were identified from the literature and by following the principles of confounding selection (Maldonado & Greenland, 1993; VanderWeele & Shpitser, 2011). The selected confounders were age, sex, race/ethnicity, alcohol consumption, physical activity (PA), cigarette smoking, depressive symptoms, shift work, BMI, OSA symptoms, and daily caloric intake.

Information on the confounders was collected at the baseline interview. Race/ethnicity was reported as white, African American, and Hispanic. Alcohol consumption was collected using the Hutchinson Diet History questionnaire and the daily alcohol intake for each participant was provided by the Fred Hutchinson Cancer Research Center. Information on PA was collected through the 7-Day Recall questionnaire (Sallis et al., 1985) and a PA index score was computed for each participant that included PA during occupational, household, and sports activities. Cigarette smoking was reported as current, former, and never. Depressive symptoms total score was derived from the Center for Epidemiological Studies Depression Scale (Radloff, 1977). The details of shift work derivation can be found in a previous BCOPS publication (Fekedulegn et al., 2016). Briefly, each participant's shift was derived from payroll history (i.e., from 1996 to the date of baseline examination) and was classified as day, afternoon, or midnight according to the highest percentage of time worked among the three shifts. Weight and height were measured, and BMI was computed for each participant using the standard formula (i.e., weight in kilograms divided by height in meters squared). OSA symptoms were obtained using the Survey Screen for Prediction of Apnea questionnaire (Maislin et al., 1995) asking the participants two questions 1) "Do you know or has someone told you that you snort or gasp while sleeping?"; and 2) "Do you know or has someone told you that breathing stops or you choke or struggle for breath while sleeping?". Each question was rated as 0: never; 1: rarely; 2: 1–2 times a week; 3: 3–4 times a week; and 4: 5–7 times a week. A symptom frequency index was computed by averaging the non-missing values for the responses to the two questions. Information on daily food

consumption was collected using the Diet History Questionnaire and the daily caloric intake was determined by the Fred Hutchinson Cancer Research Center.

Statistical methods

Descriptive statistics of the study population at baseline were compared by the hypertension status at follow-up. Unadjusted and adjusted relative risks (RR) along with corresponding 95% confidence intervals (CI) were estimated using the robust Poisson regression model available in PROC GENMOD. Sleep duration, BMI, and OSA seemed to be key factors contributing to the risk of hypertension and the three interwind closely (Drager et al., 2019). Therefore, the three factors were added to the regression model along with the other selected potential confounders sequentially: first was sleep duration; the second was BMI; and the last was OSA symptoms index. Specifically, four adjusted models were performed for each sleep quality parameter. The first model controlled for age only. The second model controlled for age, sex, race/ethnicity, daily alcohol consumption, PA, cigarette smoking status, depressive symptoms, shift work, and sleep duration. In the third model, BMI was controlled along with the confounders in the second model and in the fourth model, OSA symptoms index was also controlled.

All the sleep parameters were examined as continuous variables and as binary variables using the cut-points for poor sleep quality recommended by the US Sleep Foundation or previous studies. Specifically, unadjusted and multi-variable adjusted RRs for a 10-minute increase in sleep onset latency, 10-minute increase in wake after sleep onset, 10-minute increase in longest wake episode, each additional nightly awakening, 10% decrease in sleep efficiency, and one-hour decrease in sleep duration were computed. Similarly, unadjusted and multivariable adjusted RRs for each poor sleep indicator were computed as well. The specific cut-points for the poor sleep indicators were defined as: ≥ 45 minutes of sleep onset latency, ≥ 4 times of awakenings, ≥ 41 minutes of wake after sleep onset, and low sleep efficiency was defined as ≤ 64% for age of 25 years or younger, and ≤ 74% for age > 25 years (Ohayon et al., 2017). For the longest wake episode, we used ≥ 31 minutes as an indicator of poor sleep quality which is consistent with the recommended quantitative criterion for wake after sleep onset in insomnia research (Lichstein, Durrence, Taylor, Bush, & Riedel, 2003). The cut-point of insufficient sleep duration was six hours to be consistent with a previous prospective study (Fernandez-Mendoza et al., 2012) and a BCOPS Study (Violanti et al., 2009).

When examining the effect of interaction between insufficient sleep and obesity on the risk of hypertension, all confounders were included in the model, that is, the potential confounders in the associations between sleep duration and hypertension and between obesity and hypertension (VanderWeele & Knol, 2014). Therefore, daily caloric intake was added to the model in addition to controlling for age, sex, race, daily alcohol consumption, PA, cigarette smoking status, depressive symptoms, and shift work. Unadjusted and multivariable adjusted RRs and ratios of RRs as well as 95% CIs were computed for the following four strata:

- RR_{11} = relative risk of hypertension when sleep < 6 hours and BMI ≥ 30 kg/m^2
- RR_{10} = relative risk of hypertension when sleep ≥ 6 hours and BMI ≥ 30 kg/m^2

- R_{01} = relative risk of hypertension when sleep < 6 hours and BMI < 30 kg/m^2
- R_{00} = referent (sleep ≥ 6 hours and BMI < 30 kg/m^2)

The effect of interaction was evaluated on both additive and multiplicative scales. The measure of multiplicative interaction was assessed using the ratio of the RRs and was computed using the formula RR_{11}/ (RR_{10} x RR_{01}) (VanderWeele & Knol, 2014). The ratio of RRs > 1 indicates greater effect of interaction between insufficient sleep and obesity than the product of the effect of sleep insufficiency and obesity considered alone. The additive interaction was quantified as the relative excess risk due to interaction (RERI) and computed using the formula RERI= RR_{11} - RR_{10} - RR_{01} + 1 (Rothman, Greenland, & Lash, 2008). RERI > 0 indicates that the effect of interaction between insufficient sleep and obesity is greater than the additivity of insufficient sleep and obesity presented alone. In addition, selection bias was examined by comparing the characteristics of participants who were included in the present study and those who were excluded from the analyses. The unadjusted and multivariable adjusted RRs and the ratios of RRs along with corresponding 95% CIs were estimated using the robust Poisson regression model which is available in PROC GENMOD. The unadjusted and multivariable adjusted RERI and the corresponding 95% CI were estimated using the delta method (Hosmer & Lemeshow, 1992) which is available in PROC NLMIXED. Type I error rate was set to 5% for all the significance tests. All statistical analyses were performed using SAS 9.4 software (SAS Institute, Inc., Cary, NC).

Results

The baseline characteristics of the study sample by hypertension status at follow-up are reported in Table 1. Among the 161 police officers, the mean BMI was 28.1 kg/m^2. The officers with hypertension had a significantly larger BMI (30.9 kg/m^2 vs. 27.5 kg/m^2; p = <0.001), a higher level of daily caloric intake (1918.0 vs. 1805.6; p = 0.007), a lower PA score (13.7 vs. 21.6; p = 0.005), a higher frequency of OSA symptoms (1.3 vs. 0.7; p = 0.015) than those who were normotensive. Men were more likely than women to develop hypertension during the follow-up (89.3% vs. 10.7%; p = 0.007).

Table 2 presents the bivariate associations of continuous and dichotomized sleep parameters as well as BMI with risk of hypertension. All the six sleep parameters were significantly associated with an increased risk of hypertension. A 10-minute increase in three of the five sleep quality parameters was each associated with a significantly higher risk of hypertension (RR = 2.44, 96% CI: 1.73–3.44 for sleep onset latency; RR = 1.15, 95% CI: 1.06–1.24 for wake after sleep onset; and RR = 1.36, 95% CI: 1.21–1.53 for longest wake episode). For each additional nightly awakening, there was a 19% higher risk of hypertension (95% CI: 1.05–1.35). A 10% percent decrease in sleep efficiency was associated with 52% increased risk of hypertension (95% CI: 1.28–1.80). Every hour decrease in sleep was associated with 35% increased risk of hypertension (95% CI: 1.11–1.63). Among the participants who had the longest wake episode ≥ 31 minutes, the risk of developing hypertension was three times that of those who had < 31 minutes of the longest sleep episode (RR = 2.99, 95% CI: 1.56–5.75). Similarly, the participants who had low sleep efficiency, the risk of hypertension tripled that of those who had high sleep efficiency (RR = 2.99, 95% CI: 1.56–5.75). The

participants who had < 6 hours of sleep had an increased risk of hypertension (RR = 2.29; 95% CI: 1.17–4.45) than those who had > 6 hours of sleep and those who had a BMI ≥ 30 kg/m^2 had an increased risk of hypertension (RR = 3.33; 95% CI: 1.72–6.49) as well.

Table 3 shows the adjusted associations of the sleep parameters with risk of hypertension. The continuous version of sleep onset latency, wake after sleep onset, longest wake episode, and number of awakenings were all significantly and positively, but sleep efficiency and sleep duration were inversely associated with the risk of hypertension after adjustment for age (model 1). In addition, ≥ 31 minutes of longest wake episode and low sleep efficiency were both associated with two times higher risk of hypertension than those who had < 31 minutes of sleep onset latency or high sleep efficiency (95% CI: 1.55–5.75). In model 2, after controlling for the multiple potential confounders including age, sex, race/ethnicity, alcohol consumption, PA, smoking, depressive symptoms, shift work, and sleep duration, the continuous version of sleep onset latency, longest wake episode, and sleep efficiency remained significantly associated with the risk of hypertension (RR = 1.77, 95% CI: 1.02–3.05 for a 10-minute increase in sleep onset latency; RR = 1.26, 1.02–1.56 for a 10-minute increase in the longest wake episode; and RR = 1.54, 1.04–2.29 for 10% decrease in sleep efficiency). The significant associations remained when BMI entered the model (model 3) but disappeared when OSA symptoms index was also controlled (model 4). We did not observe any significant association of poor sleep indicators with the risk of hypertension from the multivariable adjusted models.

Table 4 shows the unadjusted and adjusted joint effects of insufficient sleep duration and obesity on the risk of hypertension on the additive and multiplicative scales controlling for multiple confounders. The unadjusted risk of hypertension among the officers who had both < 6 hours of sleep and BMI ≥ 30 kg/m^2 was more than fourfold that among those who had ≥ 6 hours of sleep and BMI < 30 kg/m^2 (RR = 4.72; 95% CI: 2.26–9.86; $P < 0.001$), and the risk was attenuated to 2.81 (95% CI: 1.26–6.25) after controlling for the potential confounders. The unadjusted interaction on additive scale was positive (RERI = 3.06, 95% CI: 0.12–6.00; $p = 0.042$). After controlling for the confounders, the statistical test for the additive interaction was not statically significant (RERI = 3.49, 95% CI: −1.69–8.68; $p = 0.158$). On the multiplicative scale, the ratio of RRs for interaction was 2.91 (95% CI: 0.60–4.06; $p = 0.184$) from unadjusted model and was 3.21 (95% CI: 0.72–14.26; $p = 0.126$) from the fully adjusted model.

Discussion

The results from the present study show that sleep onset latency and longest wake episode were positively associated with incident hypertension while sleep efficiency was inversely associated with incident hypertension. These associations remained significant after controlling for age, sex, race/ethnicity, alcohol consumption, PA, cigarette smoking status, depressive symptoms, shift work, sleep duration, and BMI. The effect of interaction between insufficient sleep duration and obesity on the risk of hypertension was not statistically significant on the additive and multiplicative scales after controlling for the potential confounders including age, sex, race/ethnicity, alcohol consumption, PA, cigarette smoking status, depressive symptoms score, shift work, and daily caloric intake.

To our knowledge, the present study was the first to examine the associations of objectively measured sleep characteristics with the risk of hypertension among police officers. The observed independent associations provided temporal link between poor sleep quality and hypertension which support the findings from a recent meta-analysis from 22 studies (21 cross-sectional studies and one prospective study) reporting that self-reported poor sleep quality was associated with higher odds of hypertension (Lo et al., 2018).

Results from the present study were partly consistent with the findings of previous studies that examined the associations of sleep characteristics (measured by actigraphy) with hypertension. One prospective study that examined the associations of sleep duration and sleep efficiency with incident hypertension reported that both shorter sleep duration and lower sleep efficiency were significantly and inversely associated with the higher odds of incident hypertension after adjustment of age, sex and race but the associations attenuated in the fully adjusted models (Knutson et al., 2009). Another longitudinal study examined the associations of several sleep characteristics including sleep duration, sleep efficiency, sleep onset latency, and wake after sleep onset with the risk of hypertension among older men (mean age: 75.1 years) but did not find any associations of sleep measures with the risk of hypertension (Fung et al., 2013). However, in the present study, the associations of sleep onset latency and sleep efficiency with the risk of hypertension remained significant after controlling for the multiple confounders. The discrepancies between the present study and the previous findings might be explained by 1) residual confounding in the present study or over-adjustment in the previous study when the study participants were in the same age category (mean age of 40.7 years in the present study and 40.1 years in (Knutson et al., 2009); and 2) the effects of poor sleep on hypertension might vary by age groups for the participants in (Fung et al., 2013) were more than 25 years older than the present cohort. Future longitudinal studies among participants with large age variations are warranted so that these associations can be examined across age groups within the same studies.

Due to the high cost of polysomnography, only a few population studies have examined associations of hypertension with sleep characteristics that were measured by polysomnography. A case-control study examined the associations of sleep onset latency, number of awakenings, sleep efficiency, and sleep duration with hypertension status (Friedman, Bradley, Ruttanaumpawan, & Logan, 2010). The researchers reported that patients with controlled hypertension or resistant hypertension had significantly lower sleep efficiency and shorter sleep duration than that among persons who were normotensive after adjusting for age, sex, BMI, apnea hypopnea index and diabetes. However, there were no significant differences in sleep onset latency and number of awakening with incident hypertension. In a previous longitudinal study among a general population with a mean age of 47 years, < 6 hours of sleep alone was significantly associated with increased odds of incident hypertension but the association attenuated after adjustment for age, sex, race/ethnicity, caffeine, cigarettes smoking, alcohol consumption, and depression which is consistent with the findings in this present study. More epidemiological studies that examine the effects of sleep characteristics (determined using polysomnography) on hypertension are warranted, especially among police officers who may have different sleep characteristics due to the high level of work stress and shift work.

The observed sleep-hypertension associations in the present study were supported by studies on human physiology that have suggested several potential mechanisms mediating difficulty initiating or maintaining sleep and hypertension risk. The common outcomes of disordered sleep are abnormal neural circulatory control, attenuated baroreflex sensitivity, increased sympathetic neural cardiovascular reactivity to stress (Li et al., 2015), enhanced hypothalamus-pituitary-adrenal axis activity, and cortical activation during sleep (Mansukhani, Covassin, & Somers, 2019) which all have detrimental effects on blood pressure regulation.

To our knowledge, the interaction between sleep duration and BMI on the development of hypertension has not been examined in previous studies. Although the interaction between sleep duration and BMI was not statistically significant, we hesitate to conclude that there was no interaction between the two exposures due to the small sample size. The joint effect between short sleep duration and obesity on hypertension needs to be examined in future longitudinal studies with larger sample sizes for this unique working population.

A few limitations should be considered when interpreting the findings from the present study. First, due to the relatively small sample size, the statistical power to detect a significant association might have been limited. When self-reported sleep apnea symptoms index was adjusted, the sample size reduced from 161 to 105 mainly due to the fact that over a quarter of the sample had missing values on OSA symptoms index. This limited our ability to clearly interpret the reduction in the risk of hypertension when the variable was controlled in the multivariable regression model. Second, the present results might be affected by selection bias due to the large percentage of participants (41.7%) who were excluded because of missing information. However, the participants in the present analyses showed a lower mean BMI and a longer mean sleep duration than those who were excluded (Supplemental Table S). These differences could have caused an underestimation of risk. Third, compared to the sleep characteristics measured by polysomnography, actigraphy tends to overestimate total sleep time and sleep efficiency and to underestimate sleep onset latency and wake after sleep onset in adults regardless of chronic physical health conditions. A recent meta-analysis of 96 studies in adults with and without chronic conditions (Conley et al., 2019) concluded that actigraphy significantly underestimated sleep onset latency compared to PSG in both healthy adults (by 8.1 minutes) and those with chronic conditions (by 7.7 minutes). In addition, we did not collect sleep diary data and the participants did not press the event marker button on the actigraph to identify the lights-off and lights-on times which would have allowed us to generate a more accurate estimate of sleep onset latency and efficiency. Although the participants were instructed to press the event marker button of the actigraph when lying down in bed, lights off, lights on, and when physically getting out of bed, nearly all the participants used the event maker only when lying down. However, the most likely effect of these measurement errors would be to draw the observed associations toward null. Despite these limitations, the prospective study design, objectively measured sleep parameters from an average of 15 days, and blood pressure measured at baseline and follow-up were major strengths compared with the previous prospective studies that used self-reported sleep parameters (Cheng, Pillai, Mengel, Roth, & Drake, 2015; Lin et al., 2017).

In summary, the present study provided evidence that difficulty initiating sleep, difficulty maintaining sleep, and lower sleep efficiency at baseline were associated with a higher risk of hypertension after a seven-year follow-up among this group of police officers. The associations were independent of potential confounders including age, sex, race/ethnicity, alcohol consumption, PA, cigarette smoking, depressive symptoms score, shift work, sleep duration, and BMI. The interaction between insufficient sleep and obesity on the additive and multiplicative scales were not statistically significant after controlling for potential confounders.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements

This work was supported by the National Institute for Occupational Safety and Health (contract number 200-2003-01580 for baseline study and grant number 1R01OH009640-01A1 for the follow-up study).

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Table 1.

Characteristics by hypertension status among police officers, BCOPS prospective study 2004–2014, N = 161.

	Total		Hypertension	Normotensive	
Characteristics	**N**	**Mean (SD) or %**	**Mean (SD) or %**	**Mean (SD) or %**	***p*-value**
Age, years	161	40.7 (7.7)	40.7 (8.2)	40.7 (7.6)	0.975
Alcohol, grams/day	158	10.2 (14.3)	15.0 (15.1)	9.1 (14.0)	0.048
Body mass index, kg/m^2	161	28.1 (4.1)	30.9 (4.2)	27.5 (3.8)	<0.001
Calorie intake, Cal/day	158	1825.6 (756.0)	1918.0 (646.5)	1805.6 (778.4)	0.007
Depressive symptoms score	160	7.8 (6.5)	8.3 (6.7)	7.7 (6.4)	0.687
Physical activity index	161	20.6 (18.8)	13.7 (11.9)	21.6 (1.7)	0.005
Sleep apnea symptoms index	118	0.8 (1.0)	1.3 (0.7)	0.7 (0.9)	0.015
Body mass index, kg/m^2					
<30	115	71.4	42.9	77.4	<0.001
≥30	46	28.6	57.1	22.6	
Race/ethnicity					
Caucasian & Hispanics	136	85.5	85.7	85.5	0.976
African American	23	14.5	14.3	14.5	
Sex					
Men	109	67.7	89.3	63.2	0.007
Women	52	32.3	10.7	36.8	
Shift work					
Day	65	42.8	35.7	44.4	0.594
Afternoon	53	34.9	42.9	33.1	
Midnight	34	22.4	21.4	22.6	
Smoking status					
Current	27	16.9	17.9	16.7	0.243
Former	43	26.9	14.3	29.6	
Never	90	56.3	67.9	53.8	

Abbreviation: BCOPS, Buffalo Cardio-metabolic Occupational Police Stress; SD, standard deviation; Note: *p*-values were from Student's T-test or Chi-square test.

Table 2.

Unadjusted associations of sleep parameters and BMI with risk of hypertension, BCOPS prospective study, 2004–2014, N=161.

Sleep parameters	Hypertensive (n = 28)	Normotensive (n = 133)		
	Mean (SD)	Mean (SD)	Risk Comparison	RR (95% CI)
Sleep onset latency, minutes	6.3 (6.1)	3.4 (2.8)	10-minute increase	**2.44 (1.73-3.44)**
Wake after sleep onset, minutes	66.4 (38.8)	46.1 (28.8)	10-minute increase	**1.15 (1.06-1.24)**
Longest wake episode, minutes	27.0 (18.5)	17.2 (10.0)	10-minute increase	**1.36 (1.21-1.53)**
Awakenings, counts	4.5 (2.5)	3.4 (2.0)	1-awakening increase	**1.19 (1.05-1.35)**
Sleep efficiency, %	79.0 (14.8)	87.0 (8.7)	10% decrease	**1.52 (1.28-1.80)**
Sleep duration, hours	5.8 (1.5)	6.5 (1.3)	1-hour decrease	**1.35 (1.11-1.63)**
	No.	**No.**	**%**	
Sleep onset latency, minutes				NA
≥45	0	0	0.00	
<45	28	133	17.39	
Wake after sleep onset, minutes				
≥41	19	67	22.09	1.84 (0.89–3.82)
<41	9	66	12.00	Referent
Longest wake episode, minutes				
≥31	9	13	40.91	**2.99 (1.56–5.75)**
<31	19	120	13.67	Referent
Awakenings, counts				
≥4	15	45	25.00	1.94 (0.993–3.80)
<4	13	88	12.87	Referent
Sleep efficiency, %[†]				
Low	9	13	40.91	**2.99 (1.56–5.75)**
High	19	120	13.67	Referent
Sleep duration, hours				
< 6	15	39	27.78	**2.29 (1.17–4.45)**
≥ 6	13	94	12.15	Referent
Body mass index, kg/m^2				

J Sleep Res. Author manuscript; available in PMC 2021 December 01.

Sleep parameters	Hypertensive (n = 28)	Normotensive (n = 133)		
	Mean (SD)	Mean (SD)	Risk Comparison	RR (95% CI)
≥30	16	30	34.8	**3.33 (1.71–6.49)**
<30	12	103	10.4	Referent

Abbreviations:

BCOPS, Buffalo Cardio-metabolic Occupational Police Stress; BMI, body mass index; CI, confidence interval; NA, not applicable; RR, relative risk.

†: For age of ≤25, ≤64% is low; for age >25 years, ≤74% is high.

P < 0.05 for the bolded values

Table 3.

Adjusted-associations of continuous sleep parameters with risk of hypertension, BCOPS prospective study, 2004–2014, N = 161

Sleep parameters	Model1 RR (95% CI)	Model 2 RR (95% CI)	Model 3 RR (95% CI)	Model 4 RR (95% CI)
	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)
Sleep onset latency, 10 minutes	**2.47 (1.75–3.48)**	**1.77 (1.02–3.05)**	**1.89 (1.12–3.20)**	1.93 (0.89–4.18)
Wake after sleep onset, 10 minutes	**1.15 (1.06–1.24)**	1.09 (0.97–1.21)	1.06 (0.94–1.18)	1.03 (0.90–1.17)
Longest wake episode, 10 minutes	**1.36 (1.20–1.53)**	**1.26 (1.02–1.56)**	**1.23 (1.01–1.50)**	1.21 (0.93–1.58)
Number of awakenings, count	**1.19 (1.05–1.36)**	1.09 (0.94–1.28)	1.06 (0.90–1.24)	1.01 (0.84–1.23)
Sleep efficiency, 10% decrease	**1.52 (1.28–1.80)**	**1.54 (1.04–2.29)**	**1.50 (1.02–2.22)**	1.47 (0.89–2.43)
Sleep duration, one-hour decrease	**1.35 (1.11–1.64)**	1.24 (0.94–1.64)	1.15 (0.85–1.54)	1.09 (0.70–1.69)
Sleep onset latency[†], minutes				
≥45	NA	NA	NA	NA
<45				
Wake after sleep onset, minutes				
≥41	1.86 (0.89–3.89)	1.32 (0.57–3.03)	1.27 (0.55–2.95)	1.19 (0.49–2.92)
<41	Referent	Referent	Referent	Referent
Longest wake episode, minutes				
≥31	**2.99 (1.56–5.75)**	2.03 (0.82–5.04)	1.60 (0.62–4.09)	1.46 (0.44–4.90)
<31	Referent	Referent	Referent	Referent
Awakenings, counts				
≥4	1.94 (0.99–3.80)	1.53 (0.69–3.37)	1.43 (0.69–2.97)	1.24 (0.44–3.44)
<4	Referent	Referent	Referent	Referent
Sleep efficiency, %[‡]				
Low	**2.99 (1.55–5.75)**	1.93 (0.73–5.14)	1.46 (0.51–4.20)	1.38 (0.40–4.82)
High	Referent	Referent	Referent	Referent

Abbreviations:

BCOPS, Buffalo Cardio-metabolic Occupational Police Stress; BMI, body mass index; CI, confidence interval; NA, not applicable; RR, relative risk.

Model 1: Adjusted for age.

Model 2: Adjustments including age, sex, race, alcohol, physical activity, smoking status, depressive symptoms, shift work, and sleep duration.

Model 3: Adjustments including the variables in Model 2 and BMI.

Model 4: Adjustments including the variables in Model 3 and sleep apnea symptoms index.

†: None of the participants had a latency ≥45 minutes in the present study sample.

‡: For age of ≤25, ≤64% is low; for age >25 years, ≤74% is low.

P < 0.05 for the bolded values

Table 4.

Interaction between sleep duration and obesity on risk of hypertension, BCOPS prospective study, 2004–2014, N = 161.

	Sleep duration (hours)				
	Sleep ≥ 6 (n = 107)		**Sleep < 6 (n = 54)**		
Unadjusted model	**N (Hypertension/Normotension)**	**RR (95% CI); *p-value***	**N (Hypertension/Normotension)**	**RR (95% CI); *p-value***	**RRs (95% CI) and *p*-value for insufficient sleep within strata of BMI**
BMI < 30 kg/m^2	9/76	1.00	3/27	0.94 (0.27–3.26); 0.928	0.94 (0.27–3.26); 0.928
BMI ≥ 30 kg/m^2	4/18	1.72 (0.58–5.06); 0.327	12/12	**4.72 (2.26–9.86); <0.001**	**2.75 (1.04–7.27); 0.042**
RRs (95% CI) and *p*-value for obesity across strata of sleep duration		1.72 (0.58–5.06); 0.327		**5.00 (1.59–15.72); 0.006**	
Measure of interaction on additive scale: RERI **= 3.06 (95% CI: 0.12–6.00); *P* = 0.042.**					
Measure of interaction on multiplicative: Ratio of RRs = 2.91 (95% CI: 0.60–14.06); *P* = 0.184.					
Adjusted model		**RR (95% CI); *p-value***		**RR (95% CI); *p-value***	**RRs† (95% CI) and *p*-value for insufficient sleep within strata of BMI**
BMI < 30 kg/m^2		1.00		0.76 (0.21–2.76); 0.680	0.76 (0.21–2.76); 0.680
BMI ≥ 30 kg/m^2		1.15 (0.42–3.18); 0.790		**2.81 (1.26–6.25); 0.011**	2.45 (0.98–6.12); 0.056
RRs† (95% CI) *p*-value for obesity for obesity across strata		1.15 (0.42–3.18); 0.790		**3.69 (1.22–11.12); 0.021**	

Abbreviations: BCOPS, Buffalo Cardio-metabolic Occupational Police Stress; CI, confidence interval; RR, relative risk; RERI, relative excess risk due to interaction.

Measure of interaction on additive scale: RERI (95% CI) = 3.49 (−1.69–8.68); $p = 0.158$.

Measure of interaction on multiplicative: Ratio of RRs (95% CI) = 3.21 (0.72–14.26); $p = 0.126$.

†: RRs were adjusted for age, sex, race, alcohol consumption, physical activity, smoking status, depressive symptoms, shift work, and daily caloric intake