Received: 27 August 2019 | Revised: 14 January 2020 | Accepted: 15 January 2020

DOI: 10.1111/jsr.12988

Journal of Sleep Research | ESRS

REGULAR RESEARCH PAPER

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

Claudia C. Ma[1] | Ja Kook Gu[1] | Ruchi Bhandari[2] | Luenda E. Charles[1] | John M. Violanti[3] | Desta Fekedulegn[1] | Michael E. Andrew[1]

[1]Bioanalytics Branch, Health Effects Laboratory Division, National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, Morgantown, WV, USA

[2]Department of Epidemiology, School of Public Health, West Virginia University, Morgantown, WV, USA

[3]Department of Epidemiology and Environmental Health, School of Public Health and Health Professions, University at Buffalo, State University of New York, Buffalo, NY, USA

Correspondence
Claudia C. Ma, National Institute for Occupational Safety and Health, MS L-4050 1095 Willowdale Road, Morgantown, WV, USA.
Email: iia4@cdc.gov

Funding information
National Institute for Occupational Safety and Health, Grant/Award Number: 1R01OH009640-01A1

Abstract

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 7-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 a robust Poisson regression model. Each 10-min increase in sleep onset latency was associated with an 89% higher risk of hypertension (95% confidence interval [CI] = 1.12–3.20). Each 10-min increase in longest wake episode was associated with a 23% higher risk of hypertension (95% CI = 1.01–1.50) and each 10% decrease in sleep efficiency was associated with a 50% higher risk of hypertension (95% CI = 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 hr of sleep and a body mass index ≥30 kg/m^2 increased the risk of hypertension (relative risk = 2.81; 95% CI = 1.26–6.25) compared with having ≥6 hr of sleep and a body mass index <30 after controlling for confounders. Relative excess risk due to interaction was 3.49 (95% CI = −1.69–8.68) and ratio of relative risk was 3.21 (95% CI = 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 of insufficient sleep and obesity on development of hypertension.

KEYWORDS

additive interaction, multiplicative interaction, sleep initiation, sleep maintenance

1 | INTRODUCTION

Sleep is essential to maintaining good physical and mental health. The literature on sleep physiology shows that non-rapid 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 (Colten & Altevogt, 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 hr) 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 6 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 on hypertension is less well known.

The objectives of the present study were to investigate (a) if sleep quality at baseline was associated with subsequent incident hypertension and (b) if there was a joint effect of insufficient sleep and obesity at baseline on the risk of hypertension after an average 7-year follow-up among police officers. We hypothesized that (a) poorer 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 (b) insufficient sleep (<6 hr) and obesity (BMI ≥30 kg/m^2) at baseline would have a joint effect on the development of hypertension.

2 | METHODS

2.1 | 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 examination. 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).

2.2 | Assessment of hypertension

At each visit, officers completed a 6-hr 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 5 min. Three measurements of SBP and DBP were taken at 2-min intervals 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 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).

2.3 | 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.) and an Octagonal Motionlogger Computer Interface with ACT #25.111PS and the first version of analysis software Action4 (cat.# 21.123, Ambulatory Monitoring, Inc.). 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 and 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 on 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 5 min. 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 because 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.

2.4 | 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. The total score for depressive symptoms 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 metres squared). OSA symptoms were obtained using the Survey Screen for Prediction of Apnea questionnaire (Maislin et al., 1995), with two questions: (a) "Do you know or has someone told you that you snort or gasp while sleeping?" and (b) "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 (one to two times a week), 3 (three to four times a week) and 4 (five to seven 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.

2.5 | Statistical methods

Descriptive statistics for the study population at baseline were compared by the hypertension status at follow-up. Unadjusted and adjusted relative risks (RRs), along with corresponding 95% confidence intervals (CIs), 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 were closely intertwined (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; second was BMI; and last was the 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, the 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 multivariable adjusted RRs for a 10-min increase in sleep onset latency, 10-min increase in wake after sleep onset, 10-min increase in longest wake episode, each additional nightly awakening, 10% decrease in sleep efficiency and 1-hr 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 min of sleep onset latency, ≥4 times of awakening and ≥41 min of wake after sleep onset, and low sleep efficiency was defined as ≤64% for age 25 years or younger and ≤74% for age >25 years (Ohayon et al., 2017). For the longest wake episode, we used ≥31 min 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 6 hr, 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 hr and BMI ≥30 kg/m^2
- RR_{10} = relative risk of hypertension when sleep ≥6 hr and BMI ≥30 kg/m^2
- R_{01} = relative risk of hypertension when sleep <6 hr and BMI <30 kg/m^2
- R_{00} = referent (sleep ≥6 hr 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 $RR_{11}/(RR_{10} \times RR_{01})$ (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. The 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.).

3 | 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 = <.001), a higher level of daily caloric intake (1918.0 vs. 1805.6; p = .007), a lower PA score (13.7 vs. 21.6; p = .005) and a higher frequency of OSA symptoms (1.3 vs. 0.7; p = .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 = .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-min increase in three of the five sleep quality parameters was 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; 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 a 52% increased risk of hypertension (95% CI = 1.28–1.80). Every hour's 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 min, the risk of developing hypertension was three times that of those who had <31 min for the longest sleep episode (RR = 2.99, 95% CI = 1.56–5.75). Similarly, for the participants who had low sleep efficiency, the risk of hypertension was triple that of those who had high sleep efficiency (RR = 2.99, 95% CI = 1.56–5.75). The participants who had <6 hr of sleep had an increased risk of hypertension (RR = 2.29; 95% CI = 1.17–4.45) compared with those who had >6 hr 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 min for the longest wake episode and low sleep efficiency were both associated with a two times higher risk of hypertension compared with those who had <31 min of sleep onset latency or high sleep efficiency (95% CI = 1.56–5.75). 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-min increase in sleep onset latency; RR = 1.26, 95% CI = 1.02–1.56 for a 10-min increase in the longest wake episode; and RR = 1.54, 95% CI = 1.04–2.29 for a 10% decrease in sleep efficiency). The significant associations remained when BMI entered the model (Model 3), but disappeared when the 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 hr of sleep and BMI ≥30 kg/m^2 was more than fourfold that among those who had ≥6 hr of sleep and BMI <30 kg/m^2 (RR = 4.72; 95% CI = 2.26–9.86; p < .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 the additive scale was positive (RERI = 3.06, 95% CI = 0.12–6.00; p = .042).

TABLE 1 Characteristics by hypertension status among police officers, BCOPS prospective study 2004–2014, *N* = 161

Characteristics	Total		Hypertension	Normotensive	
	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)	.975
Alcohol, g/day	158	10.2 (14.3)	15.0 (15.1)	9.1 (14.0)	.048
Body mass index, kg/m^2	161	28.1 (4.1)	30.9 (4.2)	27.5 (3.8)	<.001
Calorie intake, Cal/day	158	1,825.6 (756.0)	1,918.0 (646.5)	1,805.6 (778.4)	.007
Depressive symptoms score	160	7.8 (6.5)	8.3 (6.7)	7.7 (6.4)	.687
Physical activity index	161	20.6 (18.8)	13.7 (11.9)	21.6 (1.7)	.005
Sleep apnea symptoms index	118	0.8 (1.0)	1.3 (0.7)	0.7 (0.9)	.015
Body mass index, kg/m^2					
<30	115	71.4	42.9	77.4	<.001
≥30	46	28.6	57.1	22.6	
Race/ethnicity					
Caucasian & Hispanics	136	85.5	85.7	85.5	.976
African American	23	14.5	14.3	14.5	
Sex					
Men	109	67.7	89.3	63.2	.007
Women	52	32.3	10.7	36.8	
Shift work					
Day	65	42.8	35.7	44.4	.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	.243
Former	43	26.9	14.3	29.6	
Never	90	56.3	67.9	53.8	

Note: p-values were from Student's *T* test or Chi-square test.

Abbreviations: BCOPS, Buffalo Cardio-metabolic Occupational Police Stress; *SD*, standard deviation.

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 = .158). On the multiplicative scale, the ratio of RRs for interaction was 2.91 (95% CI = 0.60–4.06; p = .184) from the unadjusted model and was 3.21 (95% CI = 0.72–14.26; p = .126) from the fully adjusted model.

4 | DISCUSSION

The results from the present study show that sleep onset latency and longest wake episode were positively associated with incident hypertension, whereas 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 a temporal link between poor

TABLE 2 Unadjusted associations of sleep parameters and BMI with risk of hypertension, BCOPS prospective study, 2004–2014, *N* = 161

	Hypertensive (*n* = 28)	Normotensive (*n* = 133)			
Sleep parameters	**Mean (*SD*)**	**Mean (*SD*)**	**Risk comparison**	**RR (95% CI)**	***p*-value**
Sleep onset latency, min	6.3 (6.1)	3.4 (2.8)	10-min increase	**2.44 (1.73–3.44)**	**<.001**
Wake after sleep onset, min	66.4 (38.8)	46.1 (28.8)	10-min increase	**1.15 (1.06–1.24)**	**<.001**
Longest wake episode, min	27.0 (18.5)	17.2 (10.0)	10-min increase	**1.36 (1.21–1.53)**	**<001**
Awakenings, counts	4.5 (2.5)	3.4 (2.0)	1-awakening increase	**1.19 (1.05–1.35)**	**.007**
Sleep efficiency, %	79.0 (14.8)	87.0 (8.7)	10% decrease	**1.52 (1.28–1.80)**	**<.001**
Sleep duration, hr	5.8 (1.5)	6.5 (1.3)	1-hr decrease	**1.35 (1.11–1.63)**	**.002**
	No.	No.	%		
Sleep onset latency, min					
≥45	0	0	0.00	N/A	N/A
<45	28	133	17.39		
Wake after sleep onset, min					
≥41	19	67	22.09	1.84 (0.89–3.82)	NS
<41	9	66	12.00	Referent	
Longest wake episode, min					
≥31	9	13	40.91	**2.99 (1.56–5.75)**	**.001**
<31	19	120	13.67	Referent	
Awakenings, counts					
≥4	15	45	25.00	1.94 (0.993–3.80)	.052
<4	13	88	12.87	Referent	
Sleep efficiency, %[a]					
Low	9	13	40.91	**2.99 (1.56–5.75)**	**.001**
High	19	120	13.67	Referent	
Sleep duration, hr					
<6	15	39	27.78	**2.29 (1.17–4.45)**	**.015**
≥6	13	94	12.15	Referent	
Body mass index, kg/m^2					
≥30	16	30	34.8	**3.33 (1.71–6.49)**	**<.001**
<30	12	103	10.4	Referent	

Abbreviations: BCOPS, Buffalo Cardio-metabolic Occupational Police Stress; BMI, body mass index; CI, confidence interval; N/A, not applicable; NS, not significant; RR, relative risk.

p < .05 for the bold values.

[a]For age of ≤25, ≤64% is low; for age >25 years, ≤74% is high.

sleep quality and hypertension, which supports the findings from a recent meta-analysis of 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 (a) residual confounding in the present study or over-adjustment in the previous study when the

TABLE 3 Adjusted-association of continuous sleep parameters with risk of hypertension, BCOPS prospective study, 2004–2014, *N* = 161

	Model 1	Model 2	Model 3	Model 4
Sleep parameters	**RR (95% CI); *p*-value**	**RR (95% CI); *p*-value**	**RR (95% CI); *p*-value**	**RR (95% CI)**
Sleep onset latency, 10 min	**2.47 (1.75–3.48); <.001**	**1.77 (1.02–3.05); .042**	**1.89 (1.12–3.20); .018**	1.93 (0.89–4.18)
Wake after sleep onset, 10 min	**1.15 (1.06–1.24); <.001**	1.09 (0.97–1.21)	1.06 (0.94–1.18)	1.03 (0.90–1.17)
Longest wake episode, 10 min	**1.36 (1.20–1.53); <.001**	**1.26 (1.02–1.56); .031**	**1.23 (1.01–1.50); .043**	1.21 (0.93–1.58)
Number of awakenings, count	**1.19 (1.05–1.36); .007**	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); <.001**	**1.54 (1.04–2.29); .030**	**1.50 (1.02–2.22); .040**	1.47 (0.89–2.43)
Sleep duration, 1-hr decrease	**1.35 (1.11–1.64); .003**	1.24 (0.94–1.64)	1.15 (0.85–1.54)	1.09 (0.70–1.69)
Sleep onset latency[a], min				
≥45	NA	NA	NA	NA
<45				
Wake after sleep onset, min				
≥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, min				
≥31	**2.99 (1.56–5.75); .001**	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, %[b]				
Low	**2.99 (1.56–5.75); .001**	1.93 (0.73–5.14)	1.46 (0.51–4.20)	1.38 (0.40–4.82)
High	Referent	Referent	Referent	Referent

Note: *p*-values for the non-bold RR were all >.05. 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.

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

$p < .05$ for the bold values.

[a]None of the participants had a latency ≥45 min in the present study sample.

[b]For age of ≤25, ≤64% is low; for age >25 years, ≤74% is low.

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 (b) the effects of poor sleep on hypertension varying by age groups, because 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 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 awakenings with incident hypertension. In a previous longitudinal study among a general population with a mean age of 47 years, <6 hr of sleep alone was significantly associated with increased odds of incident hypertension but the association attenuated after adjustment for age, sex, race/ethnicity, caffeine, cigarette 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

TABLE 4 Interaction between sleep duration and obesity on risk of hypertension, BCOPS prospective study, 2004–2014, *N* = 161

	Sleep duration (hr)				
	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); .928	0.94 (0.27–3.26); .928
BMI ≥ 30 kg/m^2	4/18	1.72 (0.58–5.06); .327	12/12	**4.72 (2.26–9.86); <.001**	**2.75 (1.04–7.27); .042**
RRs (95% CI) and *p*-value for obesity across strata of sleep duration		1.72 (0.58–5.06); .327		**5.00 (1.59–15.72); .006**	
Measure of interaction on additive scale: RERI = **3.06 (95% CI: 0.12–6.00); *p* = .042**. Measure of interaction on multiplicative: Ratio of RRs = 2.91 (95% CI: 0.60–14.06); *p* = .184.					
Adjusted model	**RR (95% CI); *p*-value**		**RR (95% CI); *p*-value**	**RRs[a] (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); .680	0.76 (0.21–2.76); .680	
BMI ≥ 30 kg/m^2	1.15 (0.42–3.18); .790		**2.81 (1.26–6.25); .011**	2.45 (0.98–6.12); .056	
RRs[a] (95% CI) *p*-value for obesity for obesity across strata of sleep duration	1.15 (0.42–3.18); .790		**3.69 (1.22–11.12); .021**		
Measure of interaction on additive scale: RERI (95% CI) = 3.49 (–1.69 to 8.68); *p* = .158. Measure of interaction on multiplicative: Ratio of RRs (95% CI) = 3.21 (0.72–14.26); *p* = .126.					

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

p < .05 for the bold values.

[a]RRs were adjusted for age, sex, race, alcohol consumption, physical activity, smoking status, depressive symptoms, shift work, and daily caloric intake.

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 effect of 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 of 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 the 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 the 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 (Table S1). 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 min) and those with chronic conditions (by 7.7 min). 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 on the actigraph when lying down in bed, when lights were turned off, when lights were turned 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 7-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 effect of an interaction between insufficient sleep and obesity on the additive and multiplicative scales was not statistically significant after controlling for potential confounders.

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).

CONFLICT OF INTEREST

No conflicts of interest declared.

AUTHOR CONTRIBUTIONS

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.

DISCLAIMER

The findings and conclusions in this report are those of the author(s) and do not necessarily represent the official position of the National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention.

ORCID

Claudia C. Ma https://orcid.org/0000-0001-5639-5978
Ja Kook Gu https://orcid.org/0000-0002-0245-5899
Desta Fekedulegn https://orcid.org/0000-0002-1820-4373

REFERENCES

Carter, R. 3rd, & Watenpaugh, D. E. (2008). Obesity and obstructive sleep apnea: Or is it OSA and obesity? *Pathophysiology*, *15*(2), 71–77. https://doi.org/10.1016/j.pathophys.2008.04.009

Cheng, P., Pillai, V., Mengel, H., Roth, T., & Drake, C. L. (2015). Sleep maintenance difficulties in insomnia are associated with increased incidence of hypertension. *Sleep Health*, *1*(1), 50–54. https://doi.org/10.1016/j.sleh.2014.11.004

Clark, A. J., Salo, P., Lange, T., Jennum, P., Virtanen, M., Pentti, J., … Vahtera, J. (2016). Onset of impaired sleep and cardiovascular disease risk factors: A longitudinal study. *Sleep*, *39*(9), 1709–1718. https://doi.org/10.5665/sleep.6098

Cole, R. J., Kripke, D. F., Gruen, W., Mullaney, D. J., & Gillin, J. C. (1992). Automatic sleep/wake identification from wrist activity. *Sleep*, *15*(5), 461–469. https://doi.org/10.1093/sleep/15.5.461

Colten, H. R., & Altevogt, B. M. (Eds.) (2006). *Sleep disorders and sleep deprivation: An unmet public health problem*. Washington, D.C.: National Academies Press.

Conley, S., Knies, A., Batten, J., Ash, G., Miner, B., Hwang, Y., … Redeker, N. S. (2019). Agreement between actigraphic and polysomnographic measures of sleep in adults with and without chronic conditions: A systematic review and meta-analysis. *Sleep Medicine Reviews*, *46*, 151–160. https://doi.org/10.1016/j.smrv.2019.05.001

Drager, L. F., Santos, R. B., Silva, W. A., Parise, B. K., Giatti, S., Aielo, A. N., … Bensenor, I. M. (2019). OSA, short sleep duration, and their interactions with sleepiness and cardiometabolic risk factors in adults: The ELSA-Brasil Study. *Chest*, *155*(6), 1190–1198. https://doi.org/10.1016/j.chest.2018.12.003

Fekedulegn, D., Burchfiel, C. M., Charles, L. E., Hartley, T. A., Andrew, M. E., & Violanti, J. M. (2016). Shift work and sleep quality among urban police officers: The BCOPS Study. *Journal of Occupational and Environmental Medicine*, *58*(3), e66–e71. https://doi.org/10.1097/jom.0000000000000620

Fernandez-Mendoza, J., Vgontzas, A. N., Liao, D., Shaffer, M. L., Vela-Bueno, A., Basta, M., & Bixler, E. O. (2012). Insomnia with objective short sleep duration and incident hypertension: The Penn State Cohort. *Hypertension*, *60*(4), 929–935. https://doi.org/10.1161/hypertensionaha.112.193268

Friedman, O., Bradley, T. D., Ruttanaumpawan, P., & Logan, A. G. (2010). Independent association of drug-resistant hypertension to reduced sleep duration and efficiency. *American Journal of Hypertension*, *23*(2), 174–179. https://doi.org/10.1038/ajh.2009.220

Fung, M. M., Peters, K., Ancoli-Israel, S., Redline, S., Stone, K. L., & Barrett-Connor, E. (2013). Total sleep time and other sleep characteristics measured by actigraphy do not predict incident hypertension in a cohort of community-dwelling older men. *Journal of Clinical Sleep Medicine*, *9*(6), 585–591. https://doi.org/10.5664/jcsm.2756

Gu, J. K., Charles, L. E., Klein, R., Grady, L. M., Ma, C. C., Allison, P., … Andrew, M. E. (2018). Association between blood pressure and retinal vessel diameters among police officers in the US Northeast. *Journal of Occupational and Environmental Medicine*, *60*(3), 234–240. https://doi.org/10.1097/jom.0000000000001246

Hosmer, D. W., & Lemeshow, S. (1992). Confidence interval estimation of interaction. *Epidemiology*, *3*(5), 452–456. https://doi.org/10.1097/00001648-199209000-00012

Itani, O., Jike, M., Watanabe, N., & Kaneita, Y. (2017). Short sleep duration and health outcomes: A systematic review, meta-analysis, and meta-regression. *Sleep Medicine*, *32*, 246–256. https://doi.org/10.1016/j.sleep.2016.08.006

James, P. A., Oparil, S., Carter, B. L., Cushman, W. C., Dennison-Himmelfarb, C., Handler, J., … Ortiz, E. (2014). 2014 evidence-based guideline for the management of high blood pressure in adults: Report from the panel members appointed to the Eighth Joint National Committee (JNC 8). *JAMA*, *311*(5), 507–520. https://doi.org/10.1001/jama.2013.284427

Knutson, K. L., Van Cauter, E., Rathouz, P. J., Yan, L. L., Hulley, S. B., Liu, K., & Lauderdale, D. S. (2009). Association between sleep and blood pressure in midlife: The CARDIA sleep study. *Archives of Internal Medicine*, *169*(11), 1055–1061. https://doi.org/10.1001/archinternmed.2009.119

Koolhaas, C. M., Kocevska, D., Te Lindert, B. H. W., Erler, N. S., Franco, O. H., Luik, A. I., & Tiemeier, H. (2019). Objectively measured sleep and body mass index: A prospective bidirectional study in

middle-aged and older adults. *Sleep Medicine*, *57*, 43–50. https://doi.org/10.1016/j.sleep.2019.01.034

Li, Y., Vgontzas, A. N., Fernandez-Mendoza, J., Bixler, E. O., Sun, Y., Zhou, J., … Tang, X. (2015). Insomnia with physiological hyperarousal is associated with hypertension. *Hypertension*, *65*(3), 644–650. https://doi.org/10.1161/hypertensionaha.114.04604

Lichstein, K. L., Durrence, H. H., Taylor, D. J., Bush, A. J., & Riedel, B. W. (2003). Quantitative criteria for insomnia. *Behavior Research and Therapy*, *41*(4), 427–445. https://doi.org/10.1016/S0005-7967(02)00023-2

Lin, C. L., Liu, T. C., Lin, F. H., Chung, C. H., & Chien, W. C. (2017). Association between sleep disorders and hypertension in Taiwan: A nationwide population-based retrospective cohort study. *Journal of Human Hypertension*, *31*(3), 220–224. https://doi.org/10.1038/jhh.2016.55

Lo, K., Woo, B., Wong, M., & Tam, W. (2018). Subjective sleep quality, blood pressure, and hypertension: A meta-analysis. *Journal of Clinical Hypertension (Greenwich)*, *20*(3), 592–605. https://doi.org/10.1111/jch.13220

Ma, C. C., Hartley, T. A., Sarkisian, K., Fekedulegn, D., Mnatsakanova, A., Owens, S., … Andrew, M. E. (2019). Influence of work characteristics on the association between police stress and sleep quality. *Safety and Health at Work*, *10*(1), 30–38. https://doi.org/10.1016/j.shaw.2018.07.004

Maislin, G., Pack, A. I., Kribbs, N. B., Smith, P. L., Schwartz, A. R., Kline, L. R., … Dinges, D. F. (1995). A survey screen for prediction of apnea. *Sleep*, *18*(3), 158–166. https://doi.org/10.1093/sleep/18.3.158

Makarem, N., Shechter, A., Carnethon, M. R., Mullington, J. M., Hall, M. H., & Abdalla, M. (2019). Sleep duration and blood pressure: Recent advances and future directions. *Current Hypertension Reports*, *21*(5), 33. https://doi.org/10.1007/s11906-019-0938-7

Maldonado, G., & Greenland, S. (1993). Simulation study of confounder-selection strategies. *American Journal of Epidemiology*, *138*(11), 923–936. https://doi.org/10.1093/oxfordjournals.aje.a116813

Mansukhani, M. P., Covassin, N., & Somers, V. K. (2019). Apneic sleep, insufficient sleep, and hypertension. *Hypertension*, *73*(4), 744–756. https://doi.org/10.1161/hypertensionaha.118.11780

Matthews, K. A., Patel, S. R., Pantesco, E. J., Buysse, D. J., Kamarck, T. W., Lee, L., & Hall, M. H. (2018). Similarities and differences in estimates of sleep duration by polysomnography, actigraphy, diary, and self-reported habitual sleep in a community sample. *Sleep Health*, *4*(1), 96–103. https://doi.org/10.1016/j.sleh.2017.10.011

Ohayon, M., Wickwire, E. M., Hirshkowitz, M., Albert, S. M., Avidan, A., Daly, F. J., … Vitiello, M. V. (2017). National Sleep Foundation's sleep quality recommendations: First report. *Sleep Health*, *3*(1), 6–19. https://doi.org/10.1016/j.sleh.2016.11.006

Radloff, S. L. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, *1*(3), 385–401. https://doi.org/10.1177/014662167700100306

Rothman, K. J., Greenland, S., & Lash, T. L. (2008). *Modern epidemiology*. Philadelphia, PA: Lippincott Williams & Wilkins.

Sallis, J. F., Haskell, W. L., Wood, P. D., Fortmann, S. P., Rogers, T., Blair, S. N., & Paffenbarger, R. S. Jr (1985). Physical activity assessment methodology in the Five-City Project. *American Journal of Epidemiology*, *121*(1), 91–106.

Seravalle, G., Mancia, G., & Grassi, G. (2018). Sympathetic nervous system, sleep, and hypertension. *Current Hypertension Reports*, *20*(9), 74. https://doi.org/10.1007/s11906-018-0874-y

Thomas, S. J., & Calhoun, D. (2017). Sleep, insomnia, and hypertension: Current findings and future directions. *Journal of the American Society of Hypertension*, *11*(2), 122–129. https://doi.org/10.1016/j.jash.2016.11.008

Tobaldini, E., Costantino, G., Solbiati, M., Cogliati, C., Kara, T., Nobili, L., & Montano, N. (2017). Sleep, sleep deprivation, autonomic nervous system and cardiovascular diseases. *Neuroscience and Biobehavioral Reviews*, *74*(Pt B), 321–329. https://doi.org/10.1016/j.neubiorev.2016.07.004

VanderWeele, T. J., & Knol, M. J. (2014). A tutorial on interaction. *Epidemiologic Methods*, *3*(1), 33–72. https://doi.org/10.1515/em-2013-0005

VanderWeele, T. J., & Shpitser, I. (2011). A new criterion for confounder selection. *Biometrics*, *67*(4), 1406–1413. https://doi.org/10.1111/j.1541-0420.2011.01619.x

Violanti, J. M., & Aron, F. (1994). Ranking police stressors. *Psychological Reports*, *75*(2), 824–826. https://doi.org/10.2466/pr0.1994.75.2.824

Violanti, J. M., Burchfiel, C. M., Hartley, T. A., Mnatsakanova, A., Fekedulegn, D., Andrew, M. E., … Vila, B. J. (2009). Atypical work hours and metabolic syndrome among police officers. *Archives of Environmental & Occupational Health*, *64*(3), 194–201. https://doi.org/10.1080/19338240903241259

Violanti, J. M., Burchfiel, C. M., Miller, D. B., Andrew, M. E., Dorn, J., Wactawski-Wende, J., … Trevisan, M. (2006). The Buffalo Cardio-Metabolic Occupational Police Stress (BCOPS) pilot study: Methods and participant characteristics. *Annals of Epidemiology*, *16*(2), 148–156. https://doi.org/10.1016/j.annepidem.2005.07.054

Wang, D., Zhou, Y., Guo, Y., Zhang, R., Li, W., He, M., … Chen, W. (2017). The effect of sleep duration and sleep quality on hypertension in middle-aged and older Chinese: The Dongfeng-Tongji Cohort Study. *Sleep Medicine*, *40*, 78–83. https://doi.org/10.1016/j.sleep.2017.09.024

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

How to cite this article: Ma CC, Gu JK, Bhandari R, et al. Associations of objectively measured sleep characteristics and incident hypertension among police officers: The role of obesity. *J Sleep Res*. 2020;29:e12988. https://doi.org/10.1111/jsr.12988