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Associations of Sleep Measures with Retinal Microvascular Diameters among Police Officers

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Abstract

Purpose—We examined cross-sectional associations of sleep measures with central retinal artery equivalent (CRAE) and central retinal vein equivalent (CRVE) and investigated whether sex and race/ethnicity modified these associations.

Methods—Participants (N = 202; 78% white; 71% men) were enrolled in the Buffalo Cardio-metabolic Occupational Police Stress study (2011–2014). Wrist actigraphy was used to assess sleep measures including sleep duration, sleep efficiency, sleep onset latency, wake after sleep onset, number of awakenings, and longest wake episode. CRAE and CRVE were obtained from fundus photography of retina and were averaged from both eyes per officer.

Results—We observed significant associations between sleep efficiency and CRVE (β = −2.81 μm; *P*= .046), and between longest wake episode and CRVE (β = 3.24 μm; *P*= .021). Adjustments for demographics, lifestyles, and physical and psychological factors attenuated these associations. Sex modified the independent associations of sleep onset latency and longest wake episode with CRVE. One standard deviation (SD) increase in sleep onset latency was associated with 4.81 μm larger CRVE (*P*= .028) (*P*-interaction = 0.098), and one SD increase in longest wake episode was associated with 4.62 μm larger CRVE (*P*= .032) (*P*-interaction = 0.115) among men. Race/ethnicity also modified the association between longest wake episode and CRVE (*P*-interaction = 0.088). A significant association was observed only among white officers (β = 4.96 μm; *P*= .025).

Conclusions—We found that poor sleep quality, measured by longest wake episode, was positively and independently associated with retinal venular diameter among white and male

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officers. Longitudinal studies are warranted to assess whether poor sleep quality is a risk factor for retinal diameter changes.

Keywords

Actigraphy; fundus photography; police officer; retinal microvasculature; sleep quality

Introduction

Sleep deprivation and poor sleep quality are common among law enforcement officers. The prevalence of short sleep duration (i. e., < 7 hours) was 69.7% among police officers,[1] which is almost double that among the total U.S. workers (38%).[2] The prevalence of poor sleep quality was 51% among police officers[3] compared to 19.2% among the total U.S. workforce.[2] Previous epidemiological studies have reported the detrimental effects of poor sleep on cardiovascular health.[4] Clinically diagnosed insomnia is associated with a 68% increased risk of acute myocardial infarction and an 85% increased risk of stroke.[5]

The easily accessible retinal vasculature provides a window to detect the microvasculature changes of heart.[6] The narrowing of retinal arterioles, represented by narrower central retinal artery equivalent (CRAE), and the dilation of retinal veins, indicated by wider central retinal vein equivalent (CRVE) predict increased cardiovascular risk.[7,8] Therefore, identifying risk factors for CRAE and CRVE may be critical to cardiovascular disease (CVD) prevention.

The national survey data in the United States have shown that the prevalence of CVD differs by racial/ethnic background and sex.[9] African-Americans have higher age-adjusted prevalence of hypertension (32.1% vs. 23.5%) and stroke (4.3% vs. 2.7%) than Hispanics and Non-Hispanic whites; similarly, the prevalence in men is higher than women (26.0% vs. 23.1% for hypertension and 3.3% vs. 2.5% for stroke).

Previous studies have reported that African Americans have shorter objectively measured sleep duration than Caucasians,[10] and that sleep quality varies by sex.[11] It has also been reported that retinal arteriolar and venular calibres vary significantly across major racial/ethnic groups including Whites, Blacks, Hispanics, and Chinese.[12]

Most previous reports examining the associations of sleep disorders with the retinal microvasculature were limited to sleep apnoea.[13,18] To our knowledge, there was only one study that has examined the associations of poor sleep quality and insufficient sleep duration (< 7 hours) with microvascular function.[19] The researchers found that < 7 hours of sleep was associated with lower microvascular function among women but not among men, and that poor sleep quality was not associated with microvascular function regardless of sex. After reviewing the literature, we found that the epidemiological evidence supporting the association of poor sleep with CRAE or CRVE is lacking. Nevertheless, poor sleep quality and insufficient sleep duration are associated with inflammation that is responsible for the structural changes and pathological features of retinal microvascular system.[6] These findings provided biological plausibility for a sleep-CRAE or sleep-CRVE association.

Recently, the Sleep Foundation has recommended several sleep characteristics including sleep onset latency, wake after sleep onset (WASO), number of awakenings, sleep efficiency, and sleep duration measured by actigraphy to be used as indicators of sleep quality.[20,21] To our knowledge, whether these indicators are associated with retinal microvasculature have not yet been examined. Therefore, the objectives of the present study were to 1) examine whether the recommended sleep measures were associated with CRAE and CRVE; and 2) assess whether these associations were modified by sex and race/ethnicity in a sample of police officers.

Materials and methods

Study participants

Participants were police officers examined during the first follow-up of the Buffalo Cardio-Metabolic Occupational Police Stress (BCOPS) longitudinal study. The objectives and design of the BCOPS study have been described previously.[22] Briefly, the BCOPS study aimed to examine associations of psychosocial measures, lifestyles, and physiological biomarkers of stress with subclinical metabolic and vascular disease markers among police officers. The first BCOPS follow-up study was conducted among 281 police officers and all examinations were performed between 2011 and 2014 at the State University of New York at Buffalo in Buffalo, New York, USA. Retinal examination was performed during the first follow-up period.[23,24] Among the 281 participants, 46 officers were excluded due to missing information on retinal examination (19 officers were recruited after the completion of the retinal examination and 27 declined the retinal examination). Further exclusions included 29 officers who had missing information on any of the sleep parameters, one officer who had missing information on retinal arteriolar measurement, and three officers who had influential sleep duration (<3 hours), resulting in a final sample of 202 officers for analyses. The tenets of the Declaration of Helsinki were followed, and the study was approved by the State University of New York at Buffalo Institutional Review Board and the National Institute for Occupational Safety and Health Human Subjects Review Board. All participants from the follow-up study signed the consent form.

Objectively measured sleep parameters

Objectively measured sleep duration and sleep quality were derived from actigraphy data. Each participant was instructed to wear a sleep watch on his/her wrist of the non-dominant hand for 15 days and completed a sleep diary for the same period, which was used for the actigraphy data editing. The actigraphy data were collected using the Micro Motion Logger Sleep Watch™ (Ambulatory Monitoring Inc., NY) and were analysed using the Action-W software with Cole Kripke algorithm for sleep scoring. Numerous parameters that describe sleep quantity and quality were then derived. In the present analyses, we decided to use total sleep time, sleep efficiency, sleep onset latency, WASO, and number of awakenings as independent variables for they have been shown to be associated with health outcomes and wellness among general population.[20,21] The definition of each sleep parameter was consistent with previous studies.[20,21] In short, total sleep time was the number of hours slept during time in bed and did not include napping during work. Sleep efficiency was the percentage of time spent asleep during the time spent in bed, and sleep onset latency was the

time it took a participant to fall asleep after getting to bed. To mark time in bed, each participant was instructed to press the event marker button of the actigraph when first lying down in bed each night and when getting out of bed at the end of a sleep period. In addition, participants were also instructed to record times the actigraph was off wrist (e.g., when taking shower). The participants also kept a sleep diary for each day which consisted of time in bed (in bed time and get up time) as well as start time of work and work hours. Data from sleep diary were used to cross-check time in bed information obtained from the actigraph. Sleep onset latency was then derived as the number of minutes from the time the subject first lay down in bed to the time the subject was first scored as asleep by the sleep-scoring algorithm.

WASO was the total duration of wakefulness after the first persistent sleep, i.e., 20 continuous minutes of sleep with no more than one-minute of wakefulness. The number of awakenings referred to the total number of awake episodes with each lasting at least 5 minutes, excluding the final wakening. In addition to the above sleep parameters, longest wake episode (i.e., the duration of the longest wake episode during time in bed) was used because we hypothesized that the longest wake episode would be a stronger indicator of poor sleep quality than WASO. The estimate for each sleep measure was the average across the 15 days the sleep watch was worn.

Retinal microvasculature

Retinal microvascular measurements were obtained from fundus photography. Retinal imaging was performed by research associates who were certified through trainings provided by the Ocular Epidemiology Reading Centre (OERC) at the University of Wisconsin-Madison. The CR-2 Digital Non-Mydriatic Retinal Camera (Canon Inc., Lake Success, NY, USA) equipped with a Canon EOS 60D (18 megapixels) camera back and the Canon Retinal Image Control Software was used for imaging. The imaging procedure followed a standardized written protocol. Before imaging, each participant was seated in a windowless room with the lights turned off to allow the pupils to dilate naturally at least 4 mm in diameter. After a 5-minute waiting period, if an eye did not dilate to the desired size, the small pupil feature was used in imaging the eye. Two 45° colour retinal images were taken per eye, with the right eye always first for each participant. One image was centered on the optic nerve (field 1) and the second on the macula (field 2). After images were taken for each participant, the photographer transmitted the quality images to the OERC reading centre. The OERC staff monitored the image quality and provided feedbacks for improvements. An image that had fewer than four acceptable measurements of either vessel type was considered ungradable.

All the fundus images were graded using the Interactive Vessel Analyzer (IVAN) (V1.0). The vessels coursing through the grid between 0.5 to 1.0-disc diameters (diameter of 1800 μm) from the disc margin were manually identified as arterioles or venules. The IVAN takes the six largest arterioles and the six largest venules from the disc nerve centred image to calculate the CRAE and CRVE using the Parr-Hubbard-Knudtson's[25] revised formula: $CRAE = 0.88 * \left(w_1^2 + w_2^2\right)^{1/2}$ and $CRVE = 0.95 * \left(w_1^2 + w_2^2\right)^{1/2}$, in which w_1 is the diameter of a larger arteriole or venule, and w_2 is the diameter of a smaller arteriole or venule. The

iteration procedure of pairing up the six arterioles or the six venules has been described in detail by Knudtson et al.[25] The image scale factor for the present study is 4500 μm. If one of the six largest vessels was ungradable, either CRAE or CRVE was not computed.

Potential confounders and effect modifiers

The potential confounders were selected through the following steps. First, a list of potential confounders was selected from the literature. Then we removed any variables that were known to be in the causal pathway between poor sleep quality and CRAE or CRVE from the list. The final list of potential confounders included age, sex, race/ethnicity, body mass index (BMI), physical activity, alcohol consumption, smoking status, hypertension, sleep apnoea, police stress, and shift work. Sex and race/ethnicity were also selected as potential effect modifiers in the associations of sleep parameters with CRAE and CRVE based on these three questions: 1) is there a difference in the prevalence of the outcome between groups? 2) is there a difference in the prevalence of the exposure between groups? and 3) does the relationship between the exposure and outcome differ between groups?[26]

Information on the identified confounders and effect modifiers were objectively measured during the interview, obtained from self-reported and interviewer-administered questionnaires, or obtained from payroll records. Weight, height, and blood pressure were measured following the standardized protocol described previously.[27] BMI was computed using the standard formula. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured in seated position for each participant who had rested for 5 minutes.[24] Information on antihypertension medications was collected through a questionnaire that asked all the prescribed and over-the-counter medications taken in the past 30 days as of the examination. Each participant's hypertension status was derived and was categorized as controlled hypertension (i.e., having SBP < 140 mmHg and DBP < 90 mmHg and taking antihypertensive medications), uncontrolled hypertension (with SBP ≥ 140 mmHg or DBP ≥ 90 mmHg regardless of medication use), or normotensive (i.e., having SBP < 140 mmHg and DBP < 90 mmHg and free from antihypertensive medications) as described in the previous BCOPS study.[24] The measurement of fasting serum glucose,[27] glycated hemoglobin (HbA1 c), white blood cell count,[28] and C-reactive protein (CRP)[29] have been described previously.

Physical activity referred to the hours spent on sports, work, and household-related activities in the previous seven days and were collected using the 7-Day Recall questionnaire,[30] replacing weekdays by days worked and weekends by days off duty to integrate the participants' shift work characteristic. Total hours of physical activity were calculated for each participant by summing the time spent on moderate, hard, and very hard activity during the seven days. Alcohol intake per week was derived from the information collected on usual food choice and usual food use through a food frequency questionnaire. One drink was defined as a 12-ounce can or bottle of beer, one medium glass of wine, or one shot of liquor. Smoking status was self-reported as current, former, or never.

Information on sleep apnoea was obtained using the Survey Screen for Prediction of Apnoea questionnaire[31] asking the participants three questions 1) "Do you know or has someone told you that you snort or gasp while sleeping?"; 2) "Do you know or has someone told that you

snore loudly while sleeping?"; and 3) "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: never, rarely, 1–2 times a week, 3–4 times a week, and 5–7 times a week. Symptoms frequency index was computed by averaging the no missing values for the responses to the three questions. In the present analysis, we categorized the frequency index into three levels as 1) no symptom, if the index score was equal to zero; 2) moderate, if the score was between zero and two; and 3) severe, if the index was equal to or greater than two.

Police stress was assessed using the 60-item (stressor) Spielberger Police Stress Survey.[32] The means of how to derive stress index has been described previously.[33] Briefly, each participant reported the frequency of each stressor occurring in the past year and provided a rating score for the intensity of the stressor on the scale from 0–100. Then, a total stress index was calculated for each participant by multiplying the frequencies of each stressor in past year by the rating score and then summing these products over all 60 items. A higher score indicated a higher stress level.

The details of shift work derivation can be found in a previous BCOPS publication.[34] Briefly, the electronic work history records provided detail information on work schedule, start, and end time on the daily bases. A 10-hour permanent non-rotating shift work system was implemented by the police department since 1994. Day (if started between 4:00 AM to 11:59 AM), afternoon (if started between 12:00 PM to 7:59 PM) or midnight shift (if started between 8:00 PM to 3:59 AM) was derived for each participant from the work history data.

Diabetes status was defined as yes if meeting any of the following conditions 1) fasting plasma glucose ≥ 7.0 mmol/L or non-fasting glucose ≥11.0 mmol/L or HbA1 c ≥ 7.5%; and 2) taking antidiabetic medications.[35]

Statistical analysis

Descriptive statistics for the identified potential confounders, effect modifiers, and sleep parameters were compared by racial/ethnic background and sex. Age-adjusted associations of each potential confounder and effect modifier with CRAE and CRVE were examined using partial Pearson's correlation coefficient. Unadjusted and multivariable-adjusted associations of each standardized sleep parameter (i.e., sleep duration, sleep efficiency, sleep onset latency, WASO, number of awakenings, and longest wake episode) with CRAE and CRVE were examined using simple and multiple linear regression model controlling for age, sex, race/ethnicity, BMI, physical activity, alcohol consumption, smoking status, hypertension status, apnoea symptoms frequency index, log-transformed police stress, and shift work.

Effect modifications of sex and race/ethnicity on the associations of each sleep variable with CRAE and CRVE were tested using stratified analysis by constructing simple and multiple regression models which included interaction terms. In the multiple regression model, adjustments for the effect modification of sex were age, race/ethnicity, BMI, physical activity, alcohol consumption, smoking status, hypertension status, apnoea symptoms frequency index, log-transformed police stress, and shift work. While the adjustments for the effect modification of race/ethnicity were the above potential cofounders, except that race/

ethnicity was replaced by sex. The type I error rate was set at 5% for the associations of sleep parameters with CRAE and CRVE, and 20% for the interaction tests to account for reduced power of testing interaction terms. All analyses were performed using the SAS software, Version 9.4 (SAS Institute, Cary NC).

Results

Table 1 presents the characteristics of the study participants by race/ethnicity and sex. The mean age was 48.6 (8.3) years. Black officers had significantly shorter sleep duration than white officers (6.3 hours vs. 7.0 hours, $P < .001$) and men had shorter sleep duration than women (6.8 hours vs. 7.1 hours, $P = .046$). Men also reported higher prevalence of severe sleep apnoea symptoms frequency index score than women (31.5% vs. 8.6%, $P < .001$).

Table 2 shows the age-adjusted associations of the characteristics with CRAE and CRVE. Older age was significantly associated with a narrower mean CRAE but was not associated with CRVE. A higher BMI was significantly associated with a narrower mean CRAE (r = −0.1075; $P < .001$) but not with CRVE. White officers had narrower CRAE and CRVE than black officers (153.1μm vs.157.7 μm, $P = .044$ in CRAE; 219.3 μm vs. 231.1 μm, $P < .001$ in CRVE). Hypertension status was significantly associated with CRAE; officers having uncontrolled hypertension had narrower CRAE (146.2μm) than the controlled (152.2 μm) and the normotensive group (155.6μm) ($P = .011$).

Table 3 presents the unadjusted and multivariable-adjusted associations of each sleep parameter with CRAE and CRVE among the total study sample. None of the sleep variables was associated with CRAE. Lower sleep efficiency and longer duration of the longest wake episode were associated with wider CRVE (β = −2.81 μm per one SD decrease in sleep efficiency, $P = .046$; and β = 3.24 μm per one SD increase in the longest wake episode, $P = .021$). The significant associations attenuated (β = −2.22 μm, $P = .171$ for sleep efficiency and β = 2.21 μm, $P = .146$ for longest wake episode) after adjustments for the potential confounders including age, sex, race/ethnicity, BMI, physical activity, alcohol consumption, smoking status, hypertensive status, apnoea frequency symptoms index, log-transformed police stress, and shift work. The associations of the other sleep measures with CRVE were not statistically significant.

Table 4 shows the unadjusted- and multivariable-adjusted associations of each sleep parameter with CRVE stratified by sex and race/ethnicity. The association between sleep onset latency and CRVE was significantly modified by sex (P-interaction = 0.098). One SD increase in sleep onset latency was associated with 4.81 μm increase in CRVE ($P = .028$) among male officers after controlling for the confounders, but no association was observed among female officers. In addition, sex modified the association between the longest wake episode and CRVE (P-interaction = 0.115). Specifically, one SD increase in the longest wake episode was associated with 4.62 μm increase in CRVE ($P = .032$) among men but the association was not seen among women.

The association between the longest wake episode and CRVE was also significantly and independently modified by racial/ethnic background (P-interaction = 0.088). One SD

increase in the longest wake episode was associated with 4.96 μm increase in CRVE (P = .025) after controlling for the potential confounders, but the association was not significant among African Americans.

Table S reports the effect modification of sex and race/ethnicity on the associations of sleep parameters with CRAE. A statistically significant association between sleep hours and CRAE was observed among African Americans (β = −4–35 μm, P = .045) but was not evident among white officers (P-interaction = 0.059). We did not observe any statistically significant associations between the other sleep parameters with CRAE in the stratified analyses.

Discussion

In the present study, we examined the cross-sectional associations of objectively measured sleep characteristics including sleep duration, sleep efficiency, sleep onset latency, WASO, number of awakenings, and longest wake episode with retinal arteriolar diameter and retinal venule diameter. Sleep efficiency and longest wake episode were positively and significantly associated with CRVE, but the associations attenuated after adjustments for age, sex, race/ethnicity, BMI, physical activity, alcohol consumption, smoking status, hypertensive status, apnoea symptoms index, police stress, and shift work. Both sex and racial/ethnic background significantly modified the positive association between longest wake episode and CRVE such that the positive and independent associations were observed only among male or white officers but not among female or black officers. Sex also modified the association between sleep onset latency and CRVE in the way that the independent association was significant only among men but not among women.

To our knowledge, the present study is the first to examine the associations of objectively measured sleep measures (other than obstructive sleep apnoea) with retinal vasculature diameters and the effect modification of sex and race/ethnicity on the associations. Longitudinal analyses are warranted to determine the potential causal link between sleep quality and retinal vascular change. It might be worthwhile to mention that the associations of sleep efficiency, sleep onset latency, WASO, number of awakenings, and the longest wake episode with CRVE were all in the hypothesized direction although the associations did not reach to the statistically significant level. More epidemiological studies are warranted to explore the impacts of sleep disorders on retinal microvascular system.

Effect modification by sex

In the present study, sex modified the associations of sleep onset latency and longest wake episode with CRVE; and the associations were significant only among men. These results were consistent with that from the Multi-Ethnic Study of Atherosclerosis[15] reporting a significant association between obstructive sleep apnoea and wider CRVE among men but not among women. The significantly higher level of glucose and higher prevalence of diabetes might explain the gender differences in the associations. Another possible factor contributing to the gender difference may be the protective effect of oestrogen among female officers[36] who are mostly under the age of menopause or on oestrogen-related medications.

Finally, the small sample size of female officers might reduce the statistical power of testing a potential association.

Effect modification by race/ethnicity

The present result showed that the positive association between longest wake episode and CRVE was significant among whites but was not evident among African Americans. We need to be cautious when interpreting these results. First, the small sample of African Americans might reduce the power of detecting a potential association. Second, the significant effect modification of race/ethnicity might be biased by residual confounding. Therefore, future epidemiological studies with a larger sample size of African Americans and longitudinal studies are needed.

The unique finding in the present study is that compared to the WASO, the longest wake episode had a stronger association with CRVE. The mean time of WASO includes all wake episodes, some of which may not be caused by disordered sleep but rather are due to family needs such as waking up to take care of dependents. However, the longest wake episode may reflect the true inability to return to sleep.

We were not surprised by the result that none of the sleep parameters were associated with CRAE in the present study, for previous studies have found that CVD risk factors have different impact on retinal arterioles and venules. For example, hypertension is associated with narrower CRAE and inflammatory markers are associated with wider CRVE.[37]

The mechanisms underlying the relationship between poorer sleep quality and wider CRVE are not yet well explored. The most common explanation is that normal sleep is manifested by a cyclic alternation between non-rapid eye movement (NREM) and rapid eye movement (REM) sleep lasting 7–9 hours. REM is associated with activated sympathetic nerve activity[38] and NREM sleep is characterized by parasympathetic predominance.[39] During sleep, various physiological changes vary across stages of NREM and REM sleep.[39] Chronic disruptions of the sleep stages, such as insomnia, is shown to be a state of hyperarousal due to abnormal modulation of the autonomic nervous system.[40] The increased sympathetic nervous system activity has major effects on systemic conditions such as impaired glucose metabolism[41,42] and increased level of systemic inflammation[5] which may contribute to damages of endothelium function.

Biological studies have demonstrated that the retina and brain have the highest density of pericytes in the body.[43] Increased blood glucose concentrations could contribute to pericyte loss.[44] When blood vessels lose pericytes, they will become dilated.[43] In the exploratory analysis, we also observed that the levels of white blood cell count and CRP were positively associated with the longest wake episode and were significantly higher among men and white officers, which might explain the modification role of sex and race/ethnicity. However, the challenge of this explanation is that poor sleep was not associated with CRAE although pericytes are abundant both in retinal arterioles and venules. A study has shown the vasodilation in the porcine retina ex vivo after exposure to glucose concentration,[45] but it is not clear in human retina in vivo.

Limitations and strengths

Several limitations need to be considered when interpreting the findings from the present study. First, the cross-sectional study design precluded us from making any causal inferences. Second, our study participants were relatively young. Retinal abnormalities (CRAE narrowing and CRVE widening) are more common in older people.[46,47] Therefore, our findings may not be generalizable to older populations. Second, the small sample size of female officers or African Americans might reduce the statistical power to detect potential associations among the two groups. Third, unmeasured confounding could contribute to the observed associations or could have even masked the true association. Despite the limitations, the present study tested the relationship of several sleep parameters from actigraphy data with retinal vascular diameters which extended previous research that was limited to sleep apnoea and retinal microvasculature. Another strength was the increased reliability and reduced measurement error using an average of 15-day of actigraphy monitoring that captured the sleep variations over a 15-day of shift work cycle.

Conclusions

In conclusion, the present cross-sectional study found that sleep quality measured by actigraphy, indicated by sleep onset latency, was positively associated with CRVE among male officers, and the longest wake episode was positively associated with retinal CRVE among male or white police officers. These associations were independent of the selected potential confounders. None of the sleep parameters were associated with CRAE. Future longitudinal studies are warranted to examine the potential causal link between poor sleep quality and retinal microvasculature change.

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Table 1.

Descriptive statistics for sample demographic characteristics, lifestyles, and sleep characteristics of the study participants by race/ethnicity and by sex, BCOPS Study (N = 202).

		Total (N = 202)	White (n = 162)	Black (n = 40)		Men (n = 144)	Women (n = 58)	
Characteristics	**No.**	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**P-value**	**Mean (SD)**	**Mean (SD)**	**P-value**
Age (years)	202	48.6 (8.3)	48.8 (8.8)	47.9 (6.0)	0.452	48.6 (8.8)	48.5 (7.0)	0.912
Physical activity (hours/wk)	201	7.5 (8.5)	8.2 (9.1)	4.7 (4.4)	<0.001	8.0 (9.7)	6.3 (4.3)	0.081
Alcohol intake (No. of drink/wk)	201	5.6 (10.3)	6.0 (10.2)	3.9 (10.6)	0.258	6.7 (11.8)	2.8 (3.3)	<0.001
Body mass index (kg/m^2)	202	29.3 (4.6)	29.2 (4.7)	29.9 (3.9)	0.374	30.4 (4.4)	26.7 (3.8)	<0.001
Police stress index	189	9.3 (1.2)	9.5 (1.0)	8.6 (1.5)	<0.001	9.3 (1.2)	9.4 (1.1)	0.514
Glucose (mmol/L)	202	5.4 (1.2)	7.2 (5.3)	4.6 (2.1)	<0.001	5.5 (1.3)	5.1 (0.6)	<0.001
White blood cell (10^9/L)	202	5.8 (1.7)	5.9 (1.7)	5.3 (1.4)	0.051	5.9 (1.8)	5.5 (1.4)	0.038
C-reactive protein (mg/L)	202	2.5 (2.5)	2.5 (2.6)	2.5 (2.1)	0.978	2.4 (2.4)	2.7 (2.7)	0.352
Sleep duration, hours	202	6.8 (1.0)	7.0 (1.0)	6.3 (1.0)	<0.001	6.8 (1.0)	7.1 (1.1)	0.046
Sleep efficiency, %	202	90.8 (6.1)	91.1 (6.3)	89.8 (5.6)	0.264	90.6 (6.4)	91.2 (5.4)	0.536
Sleep onset latency, minutes	202	4.0 (4.3)	3.9 (3.6)	4.4 (6.6)	0.660	3.9 (3.7)	4.4 (5.7)	0.524
WASO, minutes	202	31.5 (25.0)	31.3 (25.5)	32.6 (23.0)	0.765	32.7 (27.4)	28.7 (24.1)	0.221
Number of awakenings, count	202	2.5 (1.6)	2.5 (1.6)	2.6 (1.4)	0.917	2.5 (1.7)	2.6 (1.4)	0.842
Longest wake episode, minutes	202	14.1 (11.2)	13.5 (6.8)	16.9 (17.8)	0.242	14.0 (9.8)	14.4 (14.3)	0.854
		%	%	%		%	%	
Sex	202							
Men		71.3	75.3	55.0	0.011	N/A	N/A	
Women		28.7	24.7	45.0		N/A	N/A	
Apnea symptoms frequency index	201							
No symptom (index = 0)		16.9	16.2	20.0	0.268	11.2	31.0	<0.001
Moderate (0< index<2)		58.2	56.5	65.0		57.3	60.3	
Severe (index≥2)		24.9	27.3	15.0		31.5	8.6	
Sleep medication use	202							
Yes		19.8	21.6	12.5	0.196	20.1	19.0	0.850
No		80.2	78.4	87.5		79.9	81.0	
Race/ethnicity	202							

Characteristics	No.	Total (N = 202) Mean (SD)	White (n = 162) Mean (SD)	Black (n = 40) Mean (SD)	P-value	Men (n = 144) Mean (SD)	Women (n = 58) Mean (SD)	P-value
White		78.2	N/A	N/A		81.9	69.0	0.021
Black		19.8	N/A	N/A		15.3	31.0	
Hispanic		2.0	N/A	N/A		2.8	0.0	
Cigarette smoking status	201							
Current		10.0	11.7	2.6	0.017	10.4	8.8	0.051
Former		32.8	35.8	20.5		27.8	45.6	
Never		57.2	52.5	76.9		61.8	45.6	
Hypertension status	202							
Uncontrolled		8.4	7.4	12.5	0.397	9.0	6.9	0.009
Controlled		23.8	25.3	17.5		29.2	10.3	
No		67.8	67.3	70.0		61.8	82.8	
Shift work	201							
Day		49.3	41.6	80.0	<0.001	38.5	75.9	<0.001
Afternoon		32.8	39.1	7.5		39.2	17.2	
Night		17.9	19.3	12.5		22.2	6.9	
Diabetes	202			0.680				0.083
Yes		6.8	5.0			8.3	1.7	
No		93.2	95.0			91.7	98.3	

Abbreviations:

BCOPS = Buffalo Cardio-metabolic Occupational Police Stress.

SD = standard deviation.

N/A = not applicable.

WASO = wake after sleep onset.

Police stress index was log transformed; *P*-values were obtained from the Student's t-test, the Chi-square test, or Fisher's exact test; and number of awakenings refer to wake episodes lasting at least 5 minutes.

Table 2.

Age-adjusted associations of sample characteristics with CRAE and CRVE, BCOPS Study (N = 202).

	CRAE		CRVE	
Variable	**r**	***P*-value**	**r**	***P*-value**
Age	−0.1571	0.026	−0.0357	0.614
Physical activity (hours/week)	0.0062	0.931	−0.0583	0.413
Alcohol intake (No. of drinks/week)	−0.0616	0.388	−0.0137	0.847
Body mass index (kg/m^2)	−0.1075	<0.001	0.0598	0.401
Police stress index	−0.0629	0.394	−0.1056	0.152
Glucose (mmol/L)	0.0201	0.775	0.1602	0.022
White blood cell (10^9/L)	0.0834	0.242	0.1948	0.006
C-reactive protein (mg/L)	0.0770	0.280	0.1904	0.007
	Mean (SE) (μm)	**P-value**	**Mean (SE) (μm)**	***P*-value**
Sex				
Women	154.4 (1.7)	0.785	221.6 (2.6)	0.977
Men	153.8 (1.1)		221.7 (1.7)	
Race/ethnicity				
White	153.1 (1.0)	0.044	219.3 (1.6)	<0.001
Black	157.7 (2.0)		231.1 (3.1)	
Cigarette smoking Status				
Current	156.0 (2.9)	0.161	224.0 (4.5)	0.733
Former	151.4 (1.6)		220.3 (2.5)	
Never	155.1 (1.2)		222.1 (1.9)	
Sleep medication use				
Yes	153.6 (2.1)	0.807	222.0 (1.6)	0.673
No	154.1 (1.0)		220.5 (3.2)	
Apnea symptoms frequency index				
No symptom (index = 0)	153.2 (2.2)	0.365	220.1 (3.4)	0.685
Moderate (0< index<2)	155.1 (1.2)		222.8 (1.9)	
Severe (index≥2)	152.1 (1.9)		220.5 (2.9)	
Hypertension status				
Uncontrolled	146.2 (1.1)	0.011	219.3 (1.7)	0.769
Controlled	152.2 (1.9)		220.1 (2.9)	
No hypertension	155.6 (3.1)		222.4 (4.9)	
Shift work				
Day	154.5 (1.4)	0.131	221.7 (2.1)	0.145
Afternoon	151.7 (1.6)		218.8 (2.5)	
Night	157.0 (2.2)		227.0 (3.4)	
Diabetes				
Yes	152.0 (3.7)	0.617	227.0 (5.6)	0.324
No	154.1 (1.0)		221.3 (1.5)	

Abbreviations:

BCOPS = Buffalo Cardio-metabolic Occupational Police Stress.

CRAE = central retinal artery equivalent.

CRVE = central retinal vein equivalent.

r = Pearson's correlation coefficient.

Table 3.

Linear associations of sleep parameters with CRAE and CRVE among police officers, BCOPS Study, N = 202.

	CRAE				CRVE			
Sleep variable	**Unadjusted β(SE)**	***p*-value**	**Adjusted† β(SE)**	***p*-value**	**Unadjusted β(SE)**	***p*-value**	**Adjusted† β(SE)**	***P*-value**
Sleep duration, hours	−1.30 (0.93)	0.163	−0.74 (1.02)	0.473	−2.22 (1.40)	0.115	0.09 (1.58)	0.954
Sleep efficiency (%)	−0.51 (0.93)	0.582	−1.66 (1.05)	0.117	−2.81 (1.40)	0.046	−2.22 (1.62)	0.171
Sleep onset latency, minutes	0.81 (0.93)	0.385	1.23 (0.97)	0.203	2.18 (1.40)	0.122	2.18 (1.48)	0.143
WASO, minutes	0.07 (0.93)	0.942	1.27 (1.04)	0.226	2.43 (1.40)	0.084	2.19 (1.60)	0.173
Number of awakenings	−0.77 (0.93)	0.410	0.42 (1.05)	0.690	1.05 (1.41)	0.455	0.88 (1.62)	0.589
Longest wake episode, minutes	0.75 (0.93)	0.420	1.52 (0.99)	0.126	3.24 (1.39)	0.021	2.21 (1.51)	0.146

Abbreviations:

BCOPS = Buffalo Cardio-metabolic Occupational Police Stress.

CRAE = central retinal artery equivalent.

CRVE = central retinal venule equivalent.

SE = standard error.

WASO = wake after sleep onset.

The regression coefficient (β) of CRAE or CRVE corresponding to one standard deviation increase in each sleep measure; and number of awakenings refer to wake episodes lasting at least 5 minutes.

†: Adjustments included age, sex, race/ethnicity, body mass index, physical activity, alcohol consumption, smoking status, hypertension status, apnea symptoms index, log-transformed police stress index, and shift work.

Table 4.

Linear associations of sleep parameters with CRVE stratified by sex and race/ethnicity among police officers, BCOPS study, N = 202.

	Men				Women				
Sleep variable	**Unadjusted β(SE)**	**P-value**	**Adjusted β(SE)†**	**P-value**	**Unadjusted β(SE)**	**P-value**	**Adjusted β(SE)†**	**P-value**	***P*-interaction†**
Sleep duration, hours	−1.09 (1.72)	0.526	0.84 (1.88)	0.655	−4.71 (2.50)	0.061	−1.50 (2.68)	0.575	0.462
Sleep efficiency (%)	−2.76 (1.59)	0.084	−2.24 (1.83)	0.223	−3.02 (3.02)	0.318	−2.16 (3.12)	0.489	0.983
Sleep onset latency, minutes	3.37 (1.98)	0.090	4.81 (2.16)	0.028	0.97 (2.01)	0.630	−0.09 (2.01)	0.964	0.098
WASO, minutes	2.33 (1.52)	0.126	1.91 (1.72)	0.269	3.07 (3.76)	0.415	3.76 (3.85)	0.331	0.655
Number of awakenings	1.25 (1.59)	0.434	1.03 (1.83)	0.573	0.33 (3.07)	0.913	0.37 (3.18)	0.909	0.852
Longest wake episode, minutes	5.19 (1.89)	0.007	4.62 (2.14)	0.032	0.96 (2.05)	0.642	−0.07 (2.07)	0.972	0.115
	White Americans				**African Americans**				***P*-interaction‡**
	Unadjusted β(SE)	***P*-value**	**Adjusted β(SE) ‡**	***P*-value**	**Unadjusted β (SE)**	***P*-value**	**Adjusted β(SE) ‡**	***P*-value**	
Sleep duration, hours	−1.00 (1.61)	0.534	0.18 (1.76)	0.919	−0.87 (3.18)	0.785	−0.24 (3.35)	0.943	0.910
Sleep efficiency (%)	−2.67 (1.50)	0.076	−2.35 (1.73)	0.175	−1.35 (3.40)	0.691	−1.49 (3.67)	0.686	0.824
Sleep onset latency, Minutes	3.62 (1.84)	0.051	4.01 (2.07)	0.054	−0.03 (2.03)	0.989	0.27 (2.11)	0.899	0.206
WASO, minutes	2.54 (1.50)	0.091	2.37 (1.69)	0.164	1.26 (3.37)	0.710	1.10 (3.72)	0.769	0.745
Number of awakenings	1.53 (1.50)	0.308	1.27 (1.71)	0.460	−1.65 (3.43)	0.631	−1.60 (3.83)	0.677	0.477
Longest wake episode, minutes	5.27 (1.92)	0.007	4.96 (2.20)	0.025	0.08 (1.93)	0.965	−0.08 (2.01)	0.969	0.088

Abbreviations:

BCOPS = Buffalo Cardio-metabolic Occupational Police Stress.

CRVE = central retinal venule equivalent.

SE = standard error.

WASO = wake after sleep onset.

The regression coefficient (β) of CRVE (μm) corresponding to one standard deviation increase in each sleep measure; and number of awakenings refer to wake episodes lasting at least 5 minutes.

†: Adjustments included age, race/ethnicity, body mass index, physical activity, alcohol consumption, smoking status, hypertension status, sleep apnoea symptoms index, log-transformed police stress index, and shift work.

‡: Adjustments included age, sex, body mass index, physical activity, alcohol consumption, smoking status, hypertension status, apnoea symptoms index, log-transformed police stress index, and shift work.