Sleep Duration and Biomarkers of Metabolic Function Among Police Officers

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Objective: To investigate associations between sleep duration and biomarkers of metabolic function among police officers. **Methods:** Metabolic markers were measured using standardized methods and sleep duration was assessed from questionnaire data. Mean levels of leptin and adiponectin were assessed across five levels of sleep duration using ANCOVA and linear regression models. **Results:** Police officers ($n = 443$) who reported an average of less than 5 hours and at least 8 hours of sleep had higher levels of leptin compared to those who reported an average of 5 to 7 hours of sleep. These associations were stronger and statistically significant among women, officers with BMI of 24.9 kg/m^2 or less and smaller abdominal height (< 20 cm), and officers who primarily worked on the day shift. **Conclusion:** Short and long sleep duration were associated with higher leptin levels and may have implications for obesity-related conditions.

Learning Objectives

- Outline previously reported associations of leptin and adiponectin with sleep, adiposity, and health risks.
- Review the new findings on the relationship between sleep and leptin levels in police officers, including subgroups in which these associations appear strongest.
- Discuss the clinical and research implications of the findings, including the possible association between sleep duration and cardiovascular risk.

Adequate sleep duration is important for optimal endocrine, neurologic, and metabolic function.[1–4] Short (and sometimes long) sleep duration have been shown to be associated with obesity, increased lipids and blood pressure, and cardiovascular disease (CVD).[5–8] Van Cauter and colleagues[9] reviewed several adverse health effects of short sleep duration that include impaired carbohydrate metabolism and dysregulation of the hormones associated with appetite (leptin, ghrelin, insulin). Other studies have shown that short sleep duration is associated with reduced levels of leptin[10–12] and adiponectin,[13] although these findings have been inconsistent.[14,15] Results from a few studies have shown a curvilinear relationship between sleep duration and obesity or obesity risk factors.[11,16–18]

Adiponectin and leptin are protein hormones that are primarily secreted by adipocytes. Adiponectin improves glucose utilization, stimulates fatty acid oxidation, and acts on the central nervous system to influence the control of body weight.[19] As an anti-inflammatory protein, adiponectin also protects endothelial cells and vascular function, and regulates metabolism and insulin sensitivity.[20,21] Among Japanese men, abdominal (visceral) adiposity decreased with increasing tertiles of adiponectin.[22] Low levels of adiponectin (< 4.0 μg/mL) have been found to be significantly associated with obesity, metabolic syndrome, and coronary artery disease[23–25] and higher levels have been shown to be associated with a lower risk of type 2 diabetes.[26] Leptin plays a key role in regulating energy intake and energy expenditure in a variety of organ systems including the reproductive, renal, and cardiovascular systems.[27] However, increased levels of leptin have been consistently reported to be directly associated with systemic inflammation, immune-mediated disorders, cerebrovascular disease, and CVD.[23,28–30]

Neylan and colleagues[31] showed that police officers often experience poor sleep quality. Police officers also frequently work long hours or multiple jobs and experience occupationally related fatigue.[32,33] Officers' sleep duration may be negatively affected by several occupational stressors such as shiftwork, long work hours, negative interpersonal interactions with supervisors and coworkers, and traumatic events.[33,34] Because police officers have been reported to have higher prevalence of CVD compared to the general population,[35,36] it is important to identify factors that may be associated with clinical or subclinical CVD in this occupational cohort. The main objective of our study was to investigate the cross-sectional associations of sleep duration with serum levels of adiponectin and leptin among police officers. Secondary objectives were to assess whether these associations are modified by sex, body mass index (BMI), abdominal height, and shiftwork status.

METHODS

Study Participants

During 2004 to 2009, all police officers employed at the Buffalo, New York Police Department (estimated to be approximately 710 in 2004) were invited to participate in a comprehensive examination. Some of them had previously participated in the 1999 to 2000 and 2001 to 2002 examinations. Data were collected at The Center for Health Research, School of Public Health and Health Professions, University at Buffalo, State University of New York.[37] The State University of New York at Buffalo internal review board approved the study and informed consent was obtained from all participants. Twenty-one officers (from among 464 officers) who did not have complete information on sleep duration or the metabolic markers were excluded from the analysis resulting in a sample size of 443 officers, 111 women, and 332 men.

Markers of Metabolic Function

Venous blood was drawn at the clinic during the morning hours from officers who had fasted for the previous 12 hours. Serum was removed from the blood after centrifugation and aliquots were frozen at -80°C. At a later date, the aliquots were shipped to the University of Vermont where laboratory analysis was performed. Adiponectin

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(Acrp30) was measured using an ELISA technique (Quantikine Human Adiponectin/Acrp20 Immunoassay, R&D Systems, Minneapolis, MN). Leptin was measured using the LINCOplex kit, panel B that is based on the Luminex technology (Linco Research, Inc, St Charles, MN). The general normal ranges of these markers are as follows: adiponectin: 1.0 to 20.0 μg/mL [30 to 600 mIU/mL]; leptin: (males) 1.1 to 12.0 ng/mL [33 to 360 μIU/mL] and (females) 3.8 to 77.0 ng/mL [114 to 2301 μIU/mL].

Sleep Duration

Sleep duration was assessed from questionnaire data. Officers were asked how many hours, on average, they slept each 24-hour period during the previous five weekdays (ie, Sunday through Thursday) and during the previous weekend (ie, Friday and Saturday). The hours of sleep reported for the five weekdays were multiplied by five and the hours reported for the weekend days were multiplied by two. The hours were then summed and divided by seven to give the total hours of sleep per 24-hour period during the previous 7 days.

Covariates

Officers were given self- and interviewer-administered questionnaires to provide information on demographic characteristics, lifestyle behaviors, and medical history. For educational status, they checked one of eight choices from "less than 12 years of school" to "graduate degree." These categories were collapsed into three levels to allow adequate numbers in each category. Officers were asked how often they consumed alcoholic beverages with one drink defined as a 12-oz. can or bottle of beer, one medium glass of wine, or one shot of liquor. The total number of drinks per month (of each type) was summed and then divided by four to give the approximate total number of drinks consumed per week. Officers reported their smoking status as current, former, or never.

Officers were weighed and height was measured. Body mass index (BMI) was calculated as weight (in kilograms) divided by height (in meters) squared. Abdominal height was measured with the participant in a supine position. The participant was asked to gently inhale, exhale, and then relax at rest. A caliper was used to measure the midsection, one inch above the iliac crests. The caliper slightly touched, but did not compress, the abdomen. Three measurements of abdominal height (to the nearest 0.1 cm) were taken and the average value was used as the participant's abdominal height.

Physical activity during the previous 7 days was obtained with the Seven-Day Physical Activity Recall questionnaire used in the Stanford Five-City Project.[38] Participants reported the duration (hours per week, hours per weekend) and intensity (moderate, hard, very hard) of three types of physical activity (occupational, household, and sports). A total physical activity score was then computed by summing the intensities of the three types of physical activity performed during the weekday and weekend.

Electronic work history data, from 1994 to 2006, were available on a daily basis for 428 participants. The time participants started their shift was used to classify each record into one of the following three shifts: day shift, if the start time of the record is between 4 AM and 11 AM; afternoon shift, if the start time is between 12 noon and 7 PM; midnight shift, if the start time is between 8 PM and 3 AM. Total hours worked as well as hours worked at the day, afternoon, and midnight shift were computed for each participant by summing all records. Taking into account the length of time a participant was working (from first date of work history to date of current examination), the computed hours were standardized to a weekly basis (hours worked per week) and percentage of total hours worked on each shift was calculated. A variable that indicates the dominant shift worked was created by assigning the participant to the shift with the highest percentage of hours worked.

Statistical Analysis

In univariate analysis, descriptive statistics of all variables were obtained stratified by sex and the *P*-values for the difference between men and women were obtained using the Student's *t* test for continuous variables and the chi-square test of independence for categorical variables. The association between selected covariates and sleep duration (also adiponectin and leptin) was assessed using analysis of variance (ANOVA) and Pearson's correlation. Analysis of variance and analysis of covariance (ANCOVA) were utilized to obtain the mean values of adiponectin and leptin across five categories of sleep duration. *P*-values for linear trend were obtained from linear regression models and for quadratic associations, from GLM models. Effect modification was assessed for sex, BMI (< 25 vs ≥ 25 kg/m^2), abdominal height (≤ 20 vs > 20 cm), and shiftwork status. On the basis of previous research or on the association of several variables with the independent and dependent variables in the present study, age, sex, and shiftwork status were included in the ANCOVA multivariate models. Alcohol intake, education, and physical activity had no influence on the unadjusted association and were therefore omitted from the final models. Leptin was log-transformed for analyses because of its nonnormal distribution and then results were back-transformed for reporting. Leptin results were converted from pg/mL to ng/mL and adiponectin results were converted from ng/mL to μg/mL to improve readability. SAS version 9.2 was used to analyze the data.[39]

RESULTS

The mean age of all officers ($n = 443$) was 42.2 years (SD = 8.3) (Table 1). Currently employed officers ($n = 400$) had a mean age ($\pm$ SD) of 41.5 $\pm$ 6.7 years, retired officers ($n = 33$) 59.3 $\pm$ 6.7 years, and new recruits ($n = 15$) 28.1 $\pm$ 4.8 years (data not shown). Compared to men, women had significantly higher mean levels of adiponectin (17.7 vs 10.1 μg/mL; $P < 0.001$) and leptin (12.3 vs 7.6 ng/mL; $P < 0.001$). Approximately 30% of all officers reported sleeping $\geq$ 7 hours per 24-hour period; 37.2% reported sleeping less than 6 hours. Sleep duration was significantly different between women and men, with, for example, 39.6% of women compared to 27.1% of men getting at least 7 hours of sleep per 24-hour period; $P = 0.008$. There was also a significant difference in shiftwork status among women and men (eg, 69.2% of women worked the day shift compared to 34.8% of men; $P < 0.001$).

Results for the association between several characteristics and the two metabolic markers, stratified by sex, are presented in Table 2. Body mass index was negatively associated with adiponectin (women: $r = -0.416$; $P < 0.05$) and positively associated with leptin (women: $r = 0.675$ and men: $r = 0.631$, both $P < 0.05$). Abdominal height was also inversely correlated with adiponectin and positively correlated with leptin. A positive correlation was observed between alcohol intake and adiponectin that was statistically significant among women only ($r = 0.221$). Smoking status, educational level, and shiftwork were not significantly associated with either biomarker. Additional analyses showed that shiftwork status was significantly associated with sleep duration among male officers (data not shown). Men who worked the day shift reported an average of 6.5 hours of sleep, whereas those who worked the afternoon and night shifts reported an average of 6.2 and 5.9 hours of sleep per 24-hour period, respectively, $P = 0.003$. No other covariate was significantly associated with sleep duration.

In Table 3, the associations between sleep duration and metabolic markers stratified by sex are presented. Neither adiponectin nor leptin showed significant linear associations with sleep duration. However, a borderline significant U-shaped curve was observed in the association between leptin and sleep duration among women only. After adjustment for age and shiftwork status, the mean level of leptin was lowest among female officers who

TABLE 1. Demographic and Other Characteristics of Participants by Sex, 2004–2009

	All ($n = 443$)	Women ($n = 111$)	Men ($n = 332$)	*P*
Age (years)	42.2 ± 8.3	41.1 ± 6.4	42.6 ± 8.9	0.043
Alcohol (drinks/wk)	5.5 ± 9.5	3.4 ± 4.5	6.3 ± 10.5	<0.001
Adiponectin (μg/mL)*	11.7 (11.1, 12.3)	17.7 (16.2, 19.3)	10.1 (9.6, 10.7)	<0.001
Leptin (ng/mL)*	8.5 (7.7, 9.4)	12.3 (10.3, 14.5)	7.6 (6.7, 8.5)	<0.001
Physical activity	21.0 ± 17.5	21.4 ± 17.4	20.8 ± 17.5	0.738
Education				
≤ HS/GED	49 (11.1)	5 (4.5)	44 (13.4)	0.031
< 4 yrs college	242 (55.0)	68 (61.3)	174 (52.9)	
≥ 4 yrs college	149 (33.9)	38 (34.2)	111 (33.7)	
Smoking status				
Current	257 (58.7)	48 (44.4)	209 (63.3)	<0.001
Former	110 (25.1)	32 (29.6)	78 (23.6)	
Never	71 (16.2)	28 (25.9)	43 (13.0)	
Sleep				
0–4.9	40 (9.0)	13 (11.7)	27 (8.1)	0.008
5.0–5.9	125 (28.2)	31 (27.9)	94 (28.3)	
6.0–6.9	144 (32.5)	23 (20.7)	121 (36.5)	
7.0–7.9	87 (19.6)	32 (28.8)	55 (16.6)	
≥ 8.0	47 (10.6)	12 (10.8)	35 (10.5)	
BMI (kg/m^2)				
Normal (≤ 24.9)	83 (18.7)	56 (50.5)	27 (8.1)	<0.001
Overweight (≥ 25)	360 (81.3)	55 (49.6)	305 (91.9)	
Abdominal height (cm)				
≤ 20	185 (41.8)	89 (80.2)	96 (28.9)	<0.001
> 20	258 (58.2)	22 (19.8)	236 (71.1)	
Shift work				
Day	183 (43.6)	74 (69.2)	109 (34.8)	<0.001
Afternoon	139 (33.1)	18 (16.8)	121 (38.7)	
Night	98 (23.3)	15 (14.0)	83 (26.5)	

Results are means ± SD or number (percent).

P values are for differences between women and men from Student's *t* tests and χ^2 tests of independence.

*Results reported as means (95% confidence intervals); analyses were completed using log-transformed data and results presented were back-transformed.

reported an average of 6 to 6.9 hours of sleep (leptin = 8.9 ng/mL; 95% confidence interval [CI] = 5.7 to 13.7), and highest among those who reported an average of less than 5 and at least 8 hours of sleep per 24-hour period (18.7 ng/mL; 95% CI = 11.2 to 31.3 and 13.2 ng/mL; 95% CI = 7.6 to 22.8, respectively); *P*-value for quadratic model = 0.054. The association among men also had a curvilinear U-shape although it was not statistically significant.

The associations between sleep duration and metabolic markers, stratified by BMI, are shown in Table 4. After adjustment for age, sex, and shiftwork status, leptin was significantly associated with sleep duration among the officers with BMI of less than 25.0 kg/m^2. This association was U-shaped. Officers reporting an average of 5 to 6.9 hours of sleep had the lowest mean levels of leptin (1.9 ng/mL) whereas officers reporting the shortest and longest sleep duration had at least twice as much (6.5 ng/mL; 95% CI = 2.7 to 15.5 and 4.4 ng/mL; 95% CI = 1.9 to 10.5, respectively); *P*-value for quadratic model = 0.015. Leptin levels among officers with BMI of at least 25.0 kg/m^2 were approximately three times higher than in officers with BMI less than 25.0 kg/m^2. Among officers in the higher BMI group, the sleep duration/leptin association was also U-shaped and was statistically significant until adjustment for age; *P*-value for quadratic model = 0.042. This association in the fully adjusted model was mildly attenuated; *P*-value for quadratic model = 0.069.

The main associations were also stratified by abdominal height and the results were similar to that observed with BMI (data not shown). The U-shaped association between sleep duration and leptin was observed among officers with abdominal height of 20 cm or less, and this association was statistically significant ($P = 0.048$). Among officers with larger abdominal height, the U-shaped association, though still apparent, was not statistically significant.

The associations between sleep duration and metabolic markers were stratified by shiftwork status (data not shown). As observed in previous results, no associations were observed between sleep duration and adiponectin. A significant U-shaped association was observed between sleep duration and leptin, and surprisingly, only among officers who worked on the day shift; $P = 0.025$. Overall, officers who worked on the day shift had higher mean levels of leptin compared to those who worked on the other two shifts. None of the stratifying variables (i.e., sex, BMI, abdominal height, and shiftwork status) significantly modified the association of sleep duration with adiponectin or leptin.

DISCUSSION

The results of our study showed that sleep duration was not significantly associated in a linear manner with either adiponectin or leptin. However, we observed U-shaped associations, that is, higher mean levels of leptin at both ends of sleep duration that was

TABLE 2. Demographic and Other Characteristics by Metabolic Markers, Stratified by Sex, 2004–2009

	Adiponectin		Leptin	
	Women ($n = 111$)	**Men ($n = 332$)**	**Women ($n = 111$)**	**Men ($n = 332$)**
Age (years)	0.093	0.207*	0.155	0.057
BMI (kg/m^2)	− 0.416*	− 0.108	0.675*	0.631*
Abdominal height (cm)	− 0.389*	− 0.072	0.599*	0.611*
Alcohol (drinks/week)	0.221*	0.033	− 0.114	0.034
Physical activity	0.197*	0.017	− 0.069	− 0.112*
Smoking status				
Current	16.9 (14.4, 19.8)	9.9 (9.3, 10.6)	12.6 (9.5, 16.8)	7.6 (6.5, 8.9)
Former	19.2 (16.4, 22.5)	10.3 (9.0, 11.7)	12.9 (9.6, 17.2)	8.2 (6.5, 10.4)
Never	17.6 (15.0, 20.6)	10.9 (9.6, 12.4)	10.0 (7.2, 14.0)	6.8 (5.2, 9.0)
*P***	0.506	0.528	0.488	0.682
Education				
≤ HS/GED	14.8 (6.2, 35.3)	10.4 (8.6, 12.6)	15.6 (8.3, 29.2)	8.6 (6.2, 12.0)
< 4 yrs college	17.8 (15.9, 19.9)	9.8 (9.1, 10.6)	11.4 (9.1, 14.3)	8.4 (7.2, 9.8)
≥ 4 yrs college	17.9 (15.3, 20.9)	10.5 (9.5, 11.5)	13.5 (10.1, 18.1)	6.3 (5.0, 7.9)
*P****	0.406	0.952	0.739	0.110
Shift work				
Day	18.0 (16.0, 20.2)	10.5 (9.5, 11.6)	13.1 (10.6, 16.1)	8.2 (6.7, 10.0)
Afternoon	18.9 (15.2, 23.5)	10.3 (9.4, 11.3)	9.9 (6.9, 14.4)	7.3 (5.8, 9.0)
Night	16.2 (13.5, 19.3)	9.4 (8.4, 10.4)	12.6 (6.8, 23.6)	7.6 (6.0, 9.7)
*P***	0.622	0.283	0.519	0.716

Results are Pearson's correlation coefficients or means (95% confidence interval).
*P <0.05.
**P are for any difference between the means.
***P are for trends from linear contrasts

TABLE 3. Mean Levels of Metabolic Markers by Categories of Sleep Duration Stratified by Sex, 2004–2009

	Hours of sleep per 24-hr period						
	< 5.0	**5.0–5.9**	**6.0–6.9**	**7.0–7.9**	**≥ 8.0**	***P****	***P*†**
Women	***n* = 13**	***n* = 31**	***n* = 23**	***n* = 32**	***n* = 12**		
Adiponectin (μg/mL)							
Model 1	16.3 (12.5, 21.2)	17.5 (14.8, 20.8)	17.4 (14.2, 21.2)	18.0 (15.2, 21.3)	19.6 (14.9, 25.9)	0.965	0.871
Model 2	16.3 (12.4, 21.5)	17.3 (14.4, 20.8)	19.3 (15.3, 24.3)	18.2 (15.0, 22.0)	19.6 (14.6, 26.2)	0.889	0.775
Leptin (ng/mL)							
Model 1	19.6 (12.0, 31.9)	13.7 (10.0, 18.8)	9.1 (6.3, 13.1)	11.1 (8.1, 15.1)	12.8 (7.7, 21.3)	0.222	0.055
Model 2	18.7 (11.2, 31.3)	13.2 (9.3, 18.8)	8.9 (5.7, 13.7)	10.7 (7.4, 15.4)	13.2 (7.6, 22.8)	0.316	0.054
Men	***n* = 27**	***n* = 94**	***n* = 121**	***n* = 55**	***n* = 35**		
Adiponectin (μg/mL)							
Model 1	10.1 (8.3, 12.3)	9.9 (8.9, 11.0)	10.0 (9.2, 11.0)	10.4 (9.1, 11.9)	10.9 (9.2, 12.9)	0.949	0.589
Model 2	10.2 (8.4, 12.4)	9.9 (8.9, 11.0)	10.0 (9.2, 11.0)	10.0 (8.7, 11.6)	10.7 (9.0, 12.8)	0.773	0.536
Leptin (ng/mL)							
Model 1	10.3 (6.8, 15.6)	6.9 (5.5, 8.6)	6.9 (5.6, 8.4)	8.9 (6.7, 11.9)	8.3 (5.8, 11.9)	0.698	0.140
Model 2	10.6 (6.9, 16.2)	7.1 (5.6, 9.0)	6.8 (5.6, 8.4)	9.0 (6.5, 12.5)	8.7 (5.9, 12.8)	0.641	0.107

Results are means (95% confidence intervals).
**P*-values are from linear regression models.
†*P*-values are for quadratic models (U-shaped) from orthogonal polynomial contrasts coefficients.
Model 1: Unadjusted.
Model 2: Adjusted for age and shiftwork status.
P-values for interaction by sex adiponectin, $P = 0.706$; leptin, $P = 0.274$.

TABLE 4. Mean Levels of Metabolic Markers by Categories of Sleep Duration, Stratified by BMI, 2004–2009

	Hours of sleep per 24-hr period						
	< 5.0	5.0–5.9	6.0–6.9	7.0–7.9	≥ 8.0	*P**	*P*†
Normal (< 25.0 kg/m²)	***n* = 8**	***n* = 23**	***n* = 22**	***n* = 20**	***n* = 10**		
Adiponectin (μg/mL)							
Model 1	15.2 (11.0, 21.0)	18.3 (15.2, 22.2)	15.3 (12.6, 18.6)	17.6 (14.4, 21.6)	19.3 (14.5, 25.8)	0.474	0.790
Model 2	14.3 (11.1, 18.5)	16.2 (13.7, 19.0)	14.8 (12.4, 17.7)	14.4 (11.9, 17.3)	16.2 (12.6, 20.9)	0.857	0.891
Leptin (ng/mL)							
Model 1	8.3 (3.0, 22.6)	3.2 (1.7, 5.7)	2.5 (1.4, 4.6)	7.1 (3.8, 13.5)	5.9 (2.4, 14.5)	0.380	0.072
Model 2	6.5 (2.7, 15.5)	1.9 (1.1, 3.4)	1.9 (1.0, 3.5)	3.5 (1.9, 6.7)	4.4 (1.9, 10.5)	0.482	0.015
Overweight (≥ 25.0 kg/m²)	***n* = 32**	***n* = 102**	***n* = 122**	***n* = 67**	***n* = 37**		
Adiponectin (μg/mL)							
Model 1	11.2 (9.3, 13.4)	10.2 (9.2, 11.3)	10.4 (9.4, 11.4)	11.5 (10.1, 13.1)	11.1 (9.3, 13.2)	0.869	0.507
Model 2	12.5 (10.4, 15.1)	11.9 (10.6, 13.4)	12.6 (11.2, 14.2)	12.8 (11.1, 14.7)	13.3 (11.1, 16.0)	0.839	0.630
Leptin (ng/mL)							
Model 1	14.2 (10.4, 19.3)	10.1 (8.5, 12.0)	8.7 (7.4, 10.2)	10.5 (8.5, 13.0)	10.3 (7.7, 13.7)	0.376	0.042
Model 2	17.4 (12.9, 23.4)	13.9 (11.4, 16.8)	11.9 (9.9, 14.5)	12.6 (10.0, 15.9)	13.8 (10.3, 18.6)	0.293	0.069

Results are means (95% confidence intervals).
**P*-values are from GLM regression models.
†*P*-values are for quadratic models (U-shaped) from orthogonal polynomial contrasts coefficients.
Model 1: Adjusted for age.
Model 2: Adjusted for age, sex, and shiftwork status.
P-values for interaction by BMI: adiponectin, $P = 0.686$; leptin, $P = 0.610$.

statistically significant among women, officers with lower BMI and smaller abdominal height, and officers who worked the day shift. The U-shaped association was also observed among the other groups (ie, men, those with larger BMI) although those results were not statistically significant. In addition, the mean levels of leptin were generally slightly higher among persons in the short sleep category than among those in the long sleep category.

Our findings are inconsistent with the results from some previous studies.[10–12] Among participants in the Quebec Family Study, levels of circulating leptin were reduced, compared to predicted values, among those with self-reported short sleep duration.[10] In a randomized, crossover clinical study, 12 healthy young men were studied and results showed that leptin levels were 18% lower when the men spent 4 hours in bed compared to when they spent 10 hours in bed.[12] In contrast, other studies reported increased leptin levels with short sleep duration. In a study conducted among children, short sleep (defined as <10 hours/day in children younger than 10 years, otherwise < 9 hours/day) was associated with higher leptin levels in girls and lower adiponectin levels in boys.[13] An experimental study conducted among 15 healthy young women showed that sleep restriction, as measured by actiwatch, affected morning leptin levels.[14] The participants were permitted to get adequate sleep in the laboratory for 2 days before being restricted to 3 hours of sleep during the second night, remaining in complete darkness during that time. Morning leptin values increased significantly on the postsleep-restriction morning relative to baseline, whereas evening leptin concentrations did not differ between days. Pejovic and colleagues[15] also reported that leptin levels were significantly increased after one night of total sleep loss. Moreover, the authors observed that the associations between sleep duration and leptin levels were influenced in an opposite manner depending on the presence or absence of a stressful environment.

Although we could not identify studies showing a U-shaped relationship between sleep duration and the two metabolic biomarkers, a few studies reported the curvilinear relationship between sleep duration and obesity or obesity risk factors.[11,16–18] Hall and colleagues[16] reported that both short and long sleep duration was associated with metabolic syndrome in midlife adults. In a community-based cross-sectional study, actigraphic (but not self-reported) sleep duration had a significant U-shaped relationship with BMI.[17] The authors found that both short sleepers (< 5 and 5 to 6 hours) and long sleepers (≥ 8 hours) were more likely to be obese, compared to participants who slept 7 to 8 hours. However, after adjustment for sleep fragmentation, the association between short sleep and obesity was no longer significant. Exclusion of participants with probable sleep apnea only marginally changed these associations.

Our results showing an association between short and long sleep duration with higher levels of leptin should be interpreted with caution. As Knutson and Turek[40] correctly pointed out, it is not clear that sleeping 9 hours or more produces health risks, especially for healthy persons, and no biological mechanisms have been identified showing that long sleep duration causes disease or death. Furthermore, the prevalence of short sleep duration in the American population has been shown to be much higher (20% to 30%) than long sleep duration (4% to 5%).[1,41] Therefore, sleep deprivation is of greater public health importance, and research to address short sleep duration and its health consequences may provide greater public health benefits.

The scientific evidence on the adverse health consequences of increased leptin levels is well documented. Leptin plays a key role in modulating immune responses and studies suggest that it may have dual roles.[42] Leptin deficiency may increase susceptibility to the effects of infectious diseases, but increased levels have been shown to be involved in the development and progression of autoimmunity.[42] From the review article by Anubhuti and Arora,[43] leptin is also closely intertwined with glucose and lipid metabolism. Leptin suppresses insulin secretion from human islets; however, insulin increases leptin production. Obese individuals have severe

insulin resistance with high circulating levels of both insulin and leptin. In obesity, hyperinsulinemia may precede insulin resistance and cause increased leptin production from larger adipose cell mass. Leptin resistance results from the failure elevated leptin levels to restore normal energy and metabolic homeostasis. Hyperleptinemia induces hypertension, accelerates thrombogenesis, and is associated with decreased arterial distensibility, atherosclerosis, and certain types of cancers.[43]

There are a few limitations to this study. First, the cross-sectional design allows us to determine association but not the temporal relationship or causality. Second, information on sleep duration was obtained from subjective self-reported data and not from actigraph data. Therefore, some inaccuracies may exist, but they are not expected to differentially bias our data. Another limitation is that data were not available for sleep duration on the night prior to blood collection for leptin and adiponectin. However, this omission is not expected to seriously bias our results since sleep data were available for the previous 7 days. Some strengths of this study include collection of medical data at a single clinic site using standardized protocols and equipment. Blood was collected from all officers under similar standardized conditions, during the early morning after they had fasted for 12 hours, thus minimizing any variability that may be due to the circadian patterns of leptin and adiponectin. Measurement of the metabolic markers was performed by experienced staff in an accredited research laboratory. Sleep duration was divided into five categories to compare the levels of biomarkers among very short and very long sleepers. This is an important study on an occupational cohort known to be exposed to considerable occupational stress and who may sometimes experience sleep deprivation. Our results may be generalizable to individuals with sleep problems/restrictions because of any reason and those working under similar work organizational characteristics. Another strength of the study is the adequate sample size of women. Very few published studies have been identified that have conducted any type of research on women police officers. Our overall sample size was also adequate to allow for stratified analyses by several variables.

CONCLUSION

In this occupational cohort, results show that police officers, who reported an average of less than 5 hours and at least 8 hours of sleep had higher levels of leptin compared to those who reported an average of 5 to 7 hours of sleep. These associations were stronger and statistically significant among women, officers with BMI of 24.9 kg/m^2 or less and smaller abdominal height (< 20 cm), and officers who primarily worked on the day shift. Our results therefore suggest that very short and very long sleep durations may be associated with cardiovascular risk factors and CVD-related conditions. The results should not be interpreted to mean that long sleepers may be at higher risk for these conditions. We did not find a significant association between sleep duration and adiponectin levels. Factors that would enhance future investigations of this research question would be the use of actigraphy (over several nights) to objectively assess sleep duration, inclusion of sleep quality data to determine its association with these biomarkers, and utilization of a longitudinal study design.

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