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Sleep Duration and Incidence of Type 2 Diabetes: The Multiethnic Cohort

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Abstract

Objectives—As an emerging risk factor for the rising incidence of type 2 diabetes, we examined sleep duration in relation to type 2 diabetes and several biomarkers.

Design—Prospective cohort recruited 1993–1996.

Setting—The Multiethnic Cohort in Hawaii and California.

Participants—A cohort of 151,691 white, African American, Japanese American, Native Hawaiian, and Latino participants; 9,695 cohort members had biomarker measurements.

Measurements—Sleep duration was self-reported at cohort entry. Diabetes status was obtained from three questionnaires and confirmed by three administrative data sources. Biomarkers were measured by standard assays 9.6±2.1 years after cohort entry. We estimated diabetes risk as a time varying outcome using Cox regression adjusted for body mass index assessed at three time points and other known confounders and computed adjusted means of biomarkers by sleep hours.

Results—During 7.9±3.5 years of follow-up, 8,487 new diabetes cases were diagnosed. Long sleep duration (≥9 hours), as compared to 7–8 hours, was significantly associated with higher incidence (HR, 1.12; 95% CI 1.04, 1.21), but the 4% elevated incidence for short sleep duration (<6 hours) did not reach significance (95% CI 0.99, 1.09). After stratification, the associations appeared stronger in Japanese American than other ethnic groups and in participants without comorbidity. Hours of sleep were positively associated with CRP and triglycerides and inversely related to HDL-cholesterol and adiponectin but not with leptin levels and HOMA-IR.

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Conclusion—In this multiethnic population, the 12% higher diabetes risk for long sleep hours may be mediated through inflammation, a poor lipid profile, and lower adiponectin levels.

Keywords

Prospective cohort; incidence; ethnicity; biomarkers; inflammation; blood lipids

INTRODUCTION

The rising health burden due to type 2 diabetes around the world has stimulated a search for new etiologic factors (1). Besides excess body weight, physical activity, and dietary composition, the role of the pineal hormone melatonin in transmitting circadian timing information to the pancreatic islets has emerged because it affects the blood-glucose-regulating function of the islet cells and inhibits insulin secretion during the night (2). In the last 50 years, the average self-reported sleep duration in the United States has decreased by 1.5–2 hours in parallel with an increasing prevalence of obesity and type 2 diabetes (3). Strong epidemiologic evidence supports an association of sleep duration with diabetes development. In a pooled study (4) and in a later meta-analysis (5), low (6) and high (8) hours of sleep predicted a higher risk in a U-shaped pattern with the lowest risk at 7–8 hours. The respective risk estimates per 1-hour shorter or longer sleep were 1.09 (95% confidence interval [CI] 1.04–1.15) and 1.14 (95% CI 1.03–1.26). In the Multiethnic Cohort (MEC), short and long sleep duration was associated with 15–25% higher all-cause and cardiovascular mortality (6). In women only, diabetes-specific mortality was also elevated with short and long sleep duration. Despite this extensive evidence on the role of sleep in diabetes etiology (7), a number of questions remain open. First, less is known about this relation in non-white ethnic groups although ethnic differences are likely given the high prevalence of obesity in some ethnic groups and stronger associations of obesity with type 2 diabetes incidence in Asians and Native Hawaiians than in whites (8–10). Second, confounding by obesity status is likely and detailed information on body mass index (BMI) is needed. Third, biological mechanisms for a potential association are not well understood. C-reactive protein (CRP), HDL-cholesterol, triglycerides, leptin, and adiponectin are some of the possible mediators for sleep duration on diabetes risk (11, 12). The current analysis determined the association of self-reported sleep duration assessed at cohort entry with diabetes incidence in five ethnic groups using self-reported diagnoses confirmed by administrative data und controlling for repeated BMI measures. In addition, possible biologic mechanisms for an association were examined using biomarkers of inflammation, adiposity, and metabolism among a subset of cohort members.

PARTICIPANTS AND METHODS

Study Population

The MEC was established in 1993–1996 to study the association of lifestyle and genetics with cancer and other chronic diseases among primarily whites, Native Hawaiians and Japanese Americans in Hawaii and African Americans and Latinos in California (13). The study has been approved by the Institutional Review Boards of the University of Hawaii and the University of Southern California. A total of 215,831 participants aged 45–75 years at

recruitment entered the cohort with a self-reported ethnic distribution of 26% Japanese American, 23% white, 22% Latino, 16% African American, 7% Native Hawaiian, and 6% Other. Depending on sex and ethnicity, the response rate varied between 19% and 51%. The MEC includes all education levels although cohort members were somewhat better educated than the general population (13).

Questionnaire and Follow-up Data

At cohort entry in 1993–1996 (QX1), detailed exposure information was collected by means of a 26-page, self-administered questionnaire (Spanish version for Latinos in California in addition to English) (13) that included an extensive validated food frequency questionnaire (FFQ) specifically developed to include foods commonly consumed by the five ethnic groups (14), height, body weight, physical activity, smoking, medications, medical conditions, family history, reproductive history, and demographics. Based on a large recipe database (15), intakes of foods, nutrients and total energy were computed. Physical activity was assessed by items asking about hours of sedentary, moderate, and vigorous activities; this information was used to compute daily Metabolic Equivalents of Tasks (METs). The question “on the average, during the last year, how many hours in a day did you sleep including naps” offered 6 categories (5, 6, 7, 8, 9, and 10 hours). As partial validation, the association of habitual sleep duration with objective measures of energy balance was demonstrated in a small subset of white and African American cohort members (16). Updated information on BMI and diabetes status was available from QX2 (1999–2002) returned by 84% of the cohort and QX3 (2003–2009) completed by approximately 50% of cohort members. Each of the questionnaires included the question “Has your doctor ever told you that you had diabetes?” At cohort entry, 25,858 out of 215,831 (12%) participants self-reported diabetes; all of these were excluded as prevalent cases. The percentage increased in QX2 and QX3 to 14% and 18%. Given the age structure of the MEC, all cases were considered type 2 diabetes. Self-reports were consistent across subsequent questionnaires for 37,728 (93%) individuals. Regular linkages with death record files in Hawaii and California were performed and provided information on all deaths until 2009 when the last QX3s were returned.

For this analysis, only participants with self-reported diabetes confirmed by administrative data were considered incident cases. Three sources of administrative data were available: Medicare claims (17), a linkage with health insurance plans in Hawaii (8), and hospital discharge diagnosis data in California (18). For 1999–2012, a Medicare linkage provided information on 114,309 cohort members who were fee-for-service beneficiaries (17). The information for 69,061 (32% of cohort) beneficiaries enrolled in managed care plans does not offer sufficient detail to establish a diagnosis. For fee-for-service plans, the Chronic Condition Warehouse supplies information on diabetes using at least one inpatient, skilled nursing facility, or home health claim or two hospital outpatient or carrier claims during a 2 year period (19). In Hawaii only, records for MEC members alive in 2007 were linked to the diabetes care registries of the two major insurers in Hawaii covering at least 90% of the population (8). The State of California provides diagnosis data for each patient who is treated as an inpatient in a licensed general acute care hospital in California (20) using the same algorithm as Medicare. Of 114,309 Medicare beneficiaries with fee-for-service plans,

44,718 (39%) were classified as cases. The percentages were lower in the California hospital discharge data (24%) and the Hawaii health plan linkage, which classified 15,110 out 88,004 (17%) participants as cases. When all three data sources were considered, 83% of self-reports were confirmed by at least one administrative data source. The major reasons for lack of confirmation were not being part of Medicare due to young age or managed care plans, not being a member of a linked health plan in Hawaii, and death.

Biomarker Assays

In 2001–2006, a MEC biospecimen subcohort of 68,740 cohort members (49.7% of eligible) with morning blood samples from predominantly fasting individuals was established. Part of this cohort ($n=12,578$) was characterized for a number of biomarkers by selecting controls from genetic case-control studies (breast, prostate, colorectal, and lung cancer). Only individuals who had fasted 8 hours and had not been diagnosed with diabetes before the blood draw were included in the analysis ($N=9,700$). Adiponectin and leptin from plasma were measured by enzyme linked immunosorbent assay (ELISA) kits (R&D Systems, Inc., Minneapolis, MN; Cat No. DRP300 and DLP00). Insulin was assessed in serum using an ELISA kit (EMD Millipore, Billerica, MA; Cat No. EZHI-14K). All ELISA protocols were followed in accordance with the manufacturer's instructions. A Cobas Mira Plus chemistry autoanalyzer (Roche Diagnostics (Indianapolis, IN) was used to measure serum glucose (kit from Randox, Kearneysville, WV), CRP, HDL-cholesterol, and triglycerides (Pointe Scientific, Inc. Canton, MI) per manufacturer's instructions. HOMA-IR was calculated as $(\text{fasted insulin [mU/L]} \times \text{fasted glucose [mg/dL]})/405$.

Statistical analysis

Of the total cohort, the following exclusions were made (some overlap) resulting in 151,691 observations: 13,994 other ethnicity, 28,153 prevalent diabetes at cohort entry, 9,152 invalid diet, 7,662 missing information on sleep variables, 3,132 missing BMI, 22,045 missing physical activity, and 4 no follow-up time. Participants with missing smoking status were coded accordingly and included as a separate category. After applying a diabetes definition requiring a positive self-report and a claims diagnosis to maximize specificity, 8,487 incident diabetes cases were identified.

Cox regression stratified by age (<55, 55–64, 65 years) was applied to assess the association between sleep hours and diabetes incidence by estimating hazard ratios (HR) and 95% CI for 3 categories with 7–8 hours as the reference and for 6 categories with 7 hours as reference. Cases were censored at the earliest of time of first recorded diabetes diagnosis (year of QX or claim), whereas the follow-up time for non-cases ended on the date when QX2 or QX3 was answered, at the end of the year before QX2 (1998) or QX3 (2002) was mailed for non-respondents, or on the date of death. Based on previous analyses in the MEC, the following covariates were included (21, 22): age at cohort entry, sex, ethnicity, education, BMI (time-varying from QX1-3), smoking status, physical activity (METs, log transformed), alcohol intake, total energy (log transformed), intake of red meat and dietary fiber (per 1,000 Kcal, log transformed), coffee and soda consumption (servings/day). Effect modification by sex, ethnicity, age (<60 vs. 60 years), BMI (<25 vs. 25 kg/m²), and presence of self-reported cardiovascular disease (heart attack or stroke) at cohort entry was

evaluated by assessing the Wald statistic for cross-product interaction terms and by stratified analyses.

For the biomarker analysis, general linear models were applied to compute geometric means for each of the six sleep duration categories while adjusting for the same covariates as in the Cox models. Trend tests were performed using continuous log-transformed biomarkers and HOMA-IR values to correct for the deviation of the distributions from normality and an ordinal variable (1–6) to denote sleep duration.

RESULTS

The current analysis was conducted among 69,097 men and 82,594 women with a nearly equal number of diabetes cases by sex (4,204 men, 4,283 women). The majority (57.8%) of cohort members reported 7–8 hours of sleep per day (Table 1). For 6 categories of sleep duration, the respective proportions were 8.9% (< 5 hours), 24.6% (6 hours), 32.6% (7 hours), 25.2% (8 hours), 6.7% (9 hours), and 2.0% (≥ 10 hours). The age-adjusted incidence rates of diabetes across the 3 categories were 7.60 (< 6 hours), 6.68 (7–8 hours), and 8.48 (≥ 9 hours) per 1,000 person-years. The distribution of sleeping hours did not differ much by sex; however, Japanese Americans were more likely to report short duration (< 6 hours) and Latinos and whites more likely to report long duration (≥ 9 hours) than the other groups. Participants who reported more or less than 7–8 sleep hours were older and more likely to be obese and current smokers, and reported higher energy intake, less education, and less physical activity. BMI differed significantly ($p<0.0001$) across sleep duration categories with respective values for the three categories of 26.5, 26.0, and 26.7 kg/m².

Overall, long but not short sleep duration was associated with incident type 2 diabetes among men and women (Table 2); the risk estimate among those sleeping ≥ 9 vs. 7–8 hours was 1.12 (95% CI 1.04, 1.21), but short sleep duration (< 6 hours) did not reach significance (HR, 1.04; 95% CI 0.99, 1.09). In a model without BMI, the respective HRs for short and long sleep duration were 1.10 (95% CI 1.05, 1.15) and 1.14 (95% CI 1.05, 1.23) indicating that a small proportion of risk is mediated by overweight/obesity. Separate risk estimates for individual categories of < 5, 6, 7, 9, and ≥ 10 hours as compared to 7 hours among all participants, were 1.07 (95% CI 0.99–1.16), 1.02 (95% CI 0.97–1.08), 1.00 (95% CI 0.94–1.06), 1.12 (95% CI 1.03–1.23), and 1.11 (95% CI 0.96–1.29), respectively.

The interactions of sleeping duration with sex, age, and BMI were not significant and the risk estimates were similar to the overall results without reaching statistical significance. Despite a non-statistically significant interaction term ($p_{\text{interaction}}=0.14$), the risk estimates differed across ethnic groups. Only for Japanese Americans, diabetes incidence was elevated for short (HR 1.09; 95% CI 1.01, 1.18) and long duration (HR 1.22; 95% CI 1.05, 1.43) of sleep. No statistically significant association of long sleep duration with diabetes incidence was seen for the other ethnic groups although the relative risk estimates were greater than 1 for African Americans, Native Hawaiians, and Latinos. In Native Hawaiians only, short duration (< 6 hours) vs. 7–8 hours of sleep was associated with a lower incidence (HR 0.85; 95% CI 0.74, 0.98). In models stratified according to self-reported heart attack and stroke at

cohort entry, only among participants without a comorbidity did long sleep duration predict a higher diabetes incidence (HR 1.16; 95% CI 1.07, 1.25).

Of the current study population, 9,700 (6.4%) participants without diabetes before blood draw had information for at least one biomarker assessed in fasting samples 9.7 ± 2.1 years after cohort entry. The percentages were similar for men (6.6%) and women (6.3%). By design, the ethnic distribution differed greatly: the proportion of whites (1.4%) and Japanese Americans (5.1%) included was smaller, whereas relatively more African Americans (8.6%), Latinos (9.4%), and Native Hawaiians (17.9%) were part of the subcohort. The biomarker subcohort was relatively similar in age, BMI, education, and lifestyle factors to the analytic cohort, but had fewer current smokers (11.9%) and participants with diabetes (N=424). The 407 of cases diagnosed before the blood draw were excluded from the biomarker analysis. The small number of incident diabetes cases after blood draw (N=17) did not allow a formal mediation analysis. Due to missing measurements, the number of observations for individual biomarkers varied slightly (Table 3).

Serum CRP was significantly higher by 0.32 mg/L (10%) with more sleep hours (Table 3). Leptin levels showed little association with sleep duration, but adiponectin concentrations were 13% lower with more sleep ($p_{\text{trend}}=0.0003$). HDL-cholesterol ($p_{\text{trend}}<0.0001$) was lower and triglycerides were higher ($p_{\text{trend}}=0.04$) in the highest vs. lowest sleep duration categories. The relation of fasting HOMA-IR with sleep hours was curvilinear with the highest value at 8 hours ($p_{\text{trend}}=0.07$).

DISCUSSION

Within this multiethnic population, longer sleep duration predicted a significant 12% higher risk of type 2 diabetes in men and women while the 4% higher risk for short sleep duration (< 6 hours) as compared to those reporting 7–8 hours did not reach statistical significance. The association appeared stronger among MEC members without cardiovascular comorbidity, Japanese Americans reporting short and long sleep hours, and Native Hawaiians with short sleep duration. The biomarker findings support the idea that inflammation, a poor lipid profile, and low adiponectin levels but not insulin resistance mediate the association of long sleep hours with type 2 diabetes.

The current finding of shorter sleep duration among non-white ethnic groups agrees with national survey data (23) that reported shorter self-reported sleep hours for individuals with minority status and low socioeconomic status. The U-shaped pattern with the lowest risk at 7–8 hours and the higher diabetes incidence associated with long sleep hours is also consistent with previous reports (4, 5). One meta-analysis (4) estimated a 50% higher incidence for long sleep duration (> 9 hours) and another one (5) a 14% higher for each additional hour of sleep. However, the small risk estimate of 4% for short sleep duration (< 6 hours) in the MEC disagrees with previous estimates of a 28% higher risk for short sleep duration (< 6 hours) and 9% per hour (4, 5). This may be due to the age structure of the MEC participants; a large proportion of the participants are retired and may not experience lack of sleep due to long working hours.

Among the limited reports on ethnic differences, the Multi-Ethnic Study of Atherosclerosis (MESA) described that short but not long sleep duration as assessed by an objective method was associated with a higher rate of abnormal fasting glucose (24), but this relation disappeared after adjustment for sleep apnea and showed no evidence interaction by ethnicity. In a cross-sectional analysis of the National Health and Nutrition Examination Survey (25), 7 hours of sleep were associated with the lowest cardiometabolic risk score in whites, whereas African Americans had an optimal score at 8 hours of sleep. At this time, we cannot offer a definite explanation for the strong U-shaped association in Japanese Americans; under-reporting of sleep, the possibility of good sleep quality and health status despite short hours, and residual confounding may be responsible.

As to biologic mechanisms for an association between sleep and type 2 diabetes, insulin resistance, lower leptin levels, and systemic inflammation have been hypothesized (12). Our findings agree with reports on higher CRP with longer sleep duration in a Cleveland report (26), a survey of Taiwanese adults (27), the Nurses' Health Study (NHS) (11), and an investigation among Japanese with long and short sleep duration (28). Our findings related to HDL-cholesterol and triglycerides are similar to the Japanese study reporting a quadratic trend (28), but the NHS showed no association for either biomarker (11). Our null results on leptin agree with the NHS (11), but the inverse association of adiponectin with sleep duration are contrary to the null findings in the NHS and the Taiwanese study (11, 27). Sleep duration had a quadratic association with HOMA-IR in the Taiwanese study with the lowest level for 6.5–8.5 hours (27), a result conflicting with the current study that detected the highest insulin resistance at 8 hours. This may be due to selection bias (only 6.4% of cohort members in the full analysis had biomarkers), incomplete case ascertainment, or an indication for mechanisms other than insulin resistance, although a U-shaped relation between sleep quantity and A1C levels in the United States National Health and Nutrition Examination Survey supported glucose control as a biologic mechanism (29).

In this large cohort with multiple data sources, self-reported diagnoses were highly consistent over 10 years of follow-up and more than 80% of self-reported diagnoses were confirmed by administrative data sources. In general, self-reports of diabetes status were found to be fairly reliable in many previous studies (30–33) despite some exceptions (34). Due to changes in diabetes definition over time, a higher number of cases are to be expected among MEC participants during later observation years (35). Other strengths of the study include a large sample size, the ethnic diversity, a wide range of diabetes incidence rates across ethnic groups (36), the availability of important covariates associated with type 2 diabetes incidence, repeated assessments of BMI, and biomarker measurements for a substantial subset of the cohort.

Limitations of the current study include potential errors in self-reports of sleep duration, and the lack of information on sleep quality, apnea, and night shift work, which are predictors for type 2 diabetes in previous reports. For example, the risk was 5–60% higher among rotating night shift workers in two US cohorts (37), Japanese workers with poor sleep quality (38), and Australian cohort members even after adjusting for baseline health (39). The importance of sleep apnea was demonstrated in the MESA report mentioned above (24). As the biomarker subcohort did not equally represent all five ethnic groups in the MEC, the current

results may have been affected by selection bias. Given the small number of incident diabetes cases with pre-diagnostic biomarkers, the mediating effect of biomarkers on the association between sleep hours and diabetes incidence could not be tested. The one-time assessment of health status and biomarkers during the study period is another weakness as levels may vary over time due to changes in lifestyle and health status. Furthermore, due to the collinearity between location (Hawaii vs. Los Angeles) and ethnic group, it was not possible to separate the influence of these two variables on diabetes incidence.

CONCLUSIONS

The current analysis from the MEC detected a higher diabetes incidence among participants with long but not short sleep hours in the overall population after adjusting for BMI as a time-varying variable and a large number of known risk factors. As possible biologic mechanisms, higher inflammation, a worse lipid profile, and lower adiponectin levels detected in participants with the longest sleep hours may be responsible for the higher diabetes risk among long sleepers, but due to the selection of biomarker subcohort, the role of biomarkers as mediators could not be confirmed. The adverse influence of long sleep duration despite adjustment for changes in BMI over time and the inclusion of a large number of known nutritional, lifestyle, and anthropometric risk factors strengthens the evidence for this finding although it cannot be ruled out that long sleep duration is the sign of underlying chronic disease, both diabetes and other conditions, a finding that may also be reflected in the biomarker levels. This study contributes novel findings related to ethnic differences to the growing literature related to the effects of sleep duration on physiological functions and disease risk, but the stronger association between sleep hours and diabetes incidence among Japanese Americans needs to be explored in future studies after including information on sleep quality.

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Table 1

Characteristics of the Study Population, Multiethnic Cohort, 1993–2009

Characteristic	Category units	6 hours	7–8 hours	9 hours
Participants	Number	50,922	87,698	13,071
Total follow-up time	Person years	396,244	697,809	96,049
Type 2 diabetes cases	Number	3,013	4,658	816
Age-adjusted incidence	per 1,000 pyrs	7.60	6.68	8.48
Age at cohort entry ^a	Years	59.4 (8.8)	59.6 (8.9)	61.1 (9.0)
Age at diabetes diagnosis ^a	Years	69.2 (9.0)	69.9 (8.8)	70.3 (8.7)
Mean follow-up time ^a	Years	7.8 (3.5)	8.0 (3.4)	7.3 (3.5)
Body mass index ^a	kg/m ²	26.5 (5.1)	26.0 (4.7)	26.7 (5.2)
Energy intake ^a	kcal/day	2,137 (1,044)	2,117 (982)	2,306 (1,122)
Red meat ^a	g/1,000 kcal/day	26.6 (16.3)	25.7 (15.9)	26.9 (16.5)
Dietary fiber ^a	g/1,000 kcal/day	11.5 (4.4)	11.8 (4.3)	11.5 (4.4)
Physical activity ^a	METs ^b	1.69 (0.30)	1.61 (0.29)	1.51 (0.29)
Sex (%)	Male	32.5	58.5	9.0
	Female	34.4	57.3	8.3
Ethnicity	White	23.6	66.3	10.1
	African American	41.5	48.3	10.2
	Native Hawaiian	41.3	50.3	8.4
	Japanese American	37.8	56.9	5.3
	Latino	32.2	57.4	10.4
Education (%)	<12 years	34.8	55.0	10.2
	13–15 years	34.1	57.6	8.3
	16 years	31.3	62.0	6.7
Smoking status (%)	Never	34.4	58.1	7.5
	Past	32.1	58.7	9.2
	Current	37.4	53.1	9.5
Alcohol intake, drinks (%)	<1 mo	35.9	55.7	8.4
	1/mo–<1/day	32.4	59.6	8.0
	1/day	28.9	60.9	10.2
Coffee intake (%)	0–<1/day	35.6	55.6	8.8
	1/day	31.6	59.3	9.1
	2/day	32.5	59.5	8.0
	3/day	34.7	57.2	8.1
Soda intake, per week (%)	None	31.9	59.9	8.2
	1/day	34.3	57.5	8.2

Characteristic	Category units	6 hours	7–8 hours	9 hours
	2/day	35.0	55.4	9.6
Comorbidity at cohort entry (heart attack/stroke) (%)	No	33.4	58.4	8.3
	Yes	36.0	51.8	12.2

^aValues represent means and standard deviations

^bMetabolic equivalent of tasks

Table 2

Self-reported Sleep Duration and Incidence of Type 2 Diabetes Multiethnic Cohort, 1993–2009^a

Variable	Category	Cases/Population	6 Hours			7–8 Hours			9 Hours			<i>p</i> _{Interaction}
			HR	95% CI	HR	HR	95% CI	HR	HR	95% CI	HR	
All		8,487/151,691	1.04	0.99	1.09	1.00	1.00	1.00	1.12	1.04	1.21	
Sex	Men	4,204/69,097	1.03	0.96	1.10	1.00	1.00	1.00	1.13	1.01	1.26	0.75
	Women	4,283/82,594	1.05	0.98	1.12	1.00	1.00	1.00	1.12	1.01	1.25	
Age	<60 years	4,436/77,240	1.06	1.01	1.14	1.00	1.00	1.00	1.14	1.03	1.28	0.84
	60 years	4,051/74,451	0.99	0.93	1.07	1.00	1.00	1.00	1.09	0.99	1.22	
Ethnicity	White	1,679/41,681	1.08	0.96	1.20	1.00	1.00	1.00	1.02	0.87	1.19	0.14
	African Am.	1,242/23,069	0.98	0.87	1.11	1.00	1.00	1.00	1.17	0.97	1.40	
	Nat. Hawaiian	908/10,813	0.85	0.74	0.98	1.00	1.00	1.00	1.12	0.89	1.40	
	Japanese Am.	2,779/45,628	1.09	1.01	1.18	1.00	1.00	1.00	1.22	1.05	1.43	
	Latino	1,879/30,500	1.05	0.95	1.16	1.00	1.00	1.00	1.10	0.94	1.27	
BMI (kg/m ²)	<25	1,593/67,190	1.08	0.97	1.20	1.00	1.00	1.00	1.16	0.97	1.39	0.07
	25	6,894/84,501	1.03	0.97	1.08	1.00	1.00	1.00	1.11	1.03	1.21	
Comorbidity	No	7,514/139,197	1.04	0.99	1.10	1.00	1.00	1.00	1.16	1.07	1.25	<0.01
	Yes	970/12,381	0.92	0.80	1.06	1.00	1.00	1.00	0.85	0.68	1.06	

^a Hazard ratios (HR) and 95% CIs were obtained by Cox regression stratified by age at cohort entry (<55, 55–64, 65 years), adjusted for sex, BMI (time-varying), ethnicity, smoking status, education, physical activity, alcohol intake, total energy intake, coffee intake, soda intake, red meat consumption, and dietary fiber intake. For comorbidity, 113 participants have missing information.

Table 3

Geometric Means of Biomarkers by Sleep Duration, Multiethnic Cohort, 1993–2009

Biomarker	Hours of sleep	N	Mean ^a	P _{trend} ^b
C-reactive protein, mg/L	5	883	3.15	0.01
	6	2653	3.34	
	7	3285	3.33	
	8	2193	3.44	
	9	536	3.44	
	10	142	3.47	
Leptin, ng/mL	5	884	17.2	0.28
	6	2652	17.6	
	7	3286	17.1	
	8	2195	17.2	
	9	535	16.7	
	10	143	18.4	
Adiponectin, µg/mL	5	879	10.1	0.0003
	6	2643	9.9	
	7	3270	9.7	
	8	2190	9.4	
	9	535	9.5	
	10	143	8.8	
HDL-Cholesterol, mg/dL	5	879	49.9	<0.0001
	6	2644	49.7	
	7	3277	49.1	
	8	2189	48.4	
	9	535	48.0	
	10	142	46.9	
Triglycerides, mg/dL	5	880	113	0.04
	6	2651	111	
	7	3282	114	
	8	2192	115	
	9	536	117	
	10	142	118	
HOMA-IR	5	875	1.71	0.07
	6	2627	1.71	
	7	3267	1.72	
	8	2176	1.92	
	9	531	1.59	
	10	142	1.77	

^a Geometric means adjusted for age at Qx1, sex, ethnicity, BMI at Qx1, smoking status, education, physical activity, intake of total energy, alcohol, coffee, soda, red meat, and dietary fiber and excluding participants with <8 hours of fasting and a diagnosis of diabetes before blood collection

^b p-value obtained from general linear models using log-transformed values of biomarkers

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