Published in final edited form as:

Sleep Health. 2018 October ; 4(5): 448–455. doi:10.1016/j.sleh.2018.07.001.

Sleep duration and excess heart age among US adults

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Abstract

Objectives: Insufficient sleep negatively impacts the cardiovascular system. No study has examined the association between sleep duration and heart age (person's predicted vascular age based on cardiovascular disease [CVD] risk profile). This study examines association between sleep duration and excess heart age (EHA; difference between heart age and chronological age) among US adults.

Design and participants: Cross-sectional 2007-2014 National Health and Nutrition Examination Survey data for respondents aged 30-74 years without CVD or stroke (n = 12,775).

Measurements: Self-reported sleep duration was classified into 5 categories (≤5, 6, 7, 8, and ≥9 hours). We used sex-specific Framingham heart age algorithm to calculate heart age and multivariable linear regression to examine association between sleep duration and EHA.

Results: A total of 13.4% (95% confidence interval 12.5-14.3), 24.2% (23.1-25.2), 31.0% (29.8-32.3), 25.9% (25.0-26.9), and 5.5% (5.0-6.1) reported sleeping ≤5, 6, 7, 8, and ≥9 hours, respectively. We observed a nonlinear relationship between sleep duration and EHA using 7 hours as reference: EHA (adjusted for sociodemographics, body mass index, physical activity, Healthy Eating Index-2010, sleep disorder, and depression status) was 5.1 (4.8-5.8), 4.5 (3.9-5.1), 3.7 (3.3-4.0), 4.5 (4.1-5.0), and 4.1 (3.3-4.9) years for sleep durations of ≤5, 6, 7, 8 and ≥9 hours, respectively (P = .015 for quadratic trend). EHA was significantly higher among participants with lower education, lower income, and obesity.

Conclusion: Mean adjusted EHA was lowest among adults who reported sleeping 7 hours per night and increased as adults reported sleeping fewer or more hours. Discussing sleep duration in the context of EHA may be helpful for patients and clinicians.

Keywords

Sleep duration; Heart age; Cardiovascular disease; Risk factors

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Conflict of interest disclosures: None were reported.

Introduction

Sleep insufficiency is a public health problem. Approximately 34.8% of adults in the United States (US) report insufficient sleep (<7 hours per 24-hour period).[1] Insufficient sleep has been estimated to contribute up to $411 billion in economic losses each year to the US due to mortality and lost productivity.[2] Whereas insufficient sleep has fiscal consequences, its cumulative exposure has detrimental impacts on multiple body systems, notably the cardiovascular system. Studies have shown associations between sleep duration and several cardiovascular disease (CVD) risk factors, including high blood pressure, smoking, high blood cholesterol, diabetes, and being overweight or obese.[3,4] This indicates the potential role of sleep duration as a significant CVD risk factor.

Cardiovascular disease was responsible for more than 800,000 deaths in 2015 (https://www.cdc.gov/nchs/data/nvsr/nvsr66/nvsr66_06.pdf) and costs the US approximately $320 billion each year.[5] Multivariable prediction models have been developed to help with CVD prevention and management.[6–9] Most of these models use an individual's risk factor profile to estimate absolute risk of having a cardiovascular event or stroke within a certain period of time (eg, in the next 10 years). However, predicted absolute risk is a concept that can be difficult for patients to understand, which poses the possibility of providing false assurance for people with high life-time, but low short-term CVD risk. Therefore, its effectiveness in promoting lifestyle changes or adherence to recommended therapeutic interventions may be limited.[9–11]

Although studies have shown an association between sleep duration and CVD risk factors, researchers have yet to combine this information in a form that simplifies the process of communicating CVD risk with the general public. In 2008, the Framingham Heart Study introduced the concept of *heart age*, defined as the predicted age of a person's vascular system based on their cardiovascular risk profile.[12] Heart age and its comparison with chronological age (defined as excess heart age [EHA]), represent an alternative, simplified way of communicating risk for developing CVD. The objective of this study is to examine the association between sleep duration and EHA using data from nationally representative samples of US adults in 2007-2014. We hope to further examine the relationship between sleep and cardiovascular health as well as to improve and simplify communication of CVD risk with the use of EHA rather than predicted absolute CVD risk score.

Participants and methods

Study population

The National Health and Nutrition Examination Survey (NHANES) is a cross-sectional assessment of the US population's health. The survey is conducted by the Centers for Disease Control and Prevention's National Center for Health Statistics, with information gathered through interviews, medical examinations, and laboratory tests. The survey provides demographic information and laboratory data for a sample representative of civilian US residents who are not institutionalized.[13] In this study, we pooled NHANES data sets from 2007-2014 to assess the association between sleep duration and EHA. Adults aged

30 to 74 years were included consistent with the age range used in the Framingham Heart Study's heart age calculation.[12]

Exposure variable

Self-reported sleep duration was assessed based on response to the question, "How much sleep do you usually get at night on week-days or workdays (hours)?" Responses were classified into 5 categories, ≤5, 6, 7, 8, and ≥9 hours of sleep, consistent with 2 previous studies, both of which examined sleep duration's impact on cardiovascular health outcomes.[14,15]

Outcome variable

To calculate heart age, the sex-specific laboratory-based Framingham Risk Score (FRS) was used to estimate 10-year risk for developing CVD for each participant.[12] Parameters from the FRS models were used to calculate predicted heart age and EHA (calculated as the difference between predicted heart age and chronological age).[12] The laboratory-based FRS models included 8 variables: age, sex, systolic blood pressure (SBP), hypertension treatment status (yes/no), smoking status (current/no), diabetes (yes/no), high-density lipoprotein cholesterol (HDL-C), and total cholesterol (continuous). SBP was calculated using the average of the last 2 measurements of blood pressure for participants who had 3 readings, the last measurement for participants who had 2 readings, and the only measurement for participants who had 1 blood pressure reading. Hypertension treatment status was determined from affirmative responses to the following questions: "Were you told on 2 or more different visits that you had hypertension, also called high blood pressure?", "Because of your (high blood pressure/hypertension), have you ever been told to take prescribed medicine?", "Are you now taking prescribed medicine?" Smoking status was determined from responses to 2 questions: (1) "Have you smoked at least 100 cigarettes in your entire life?" (yes/no) and (2) "Do you now smoke cigarettes?" (every day/some days/not at all). Current smokers were classified as those participants who answered "yes" to question 1 and either "every day" or "some days" to question 2. All other answer combinations represented nonsmokers. Participants were considered to have diabetes if they met at least 1 of 4 conditions: (1) if they responded affirmatively to the question "has a doctor told you that you have diabetes?", (2) if they had a level of fasting plasma glucose ≥126 mg/dL, (3) if they had a concentration of glycated hemoglobin ≥6.5%, or (4) if they responded affirmatively to the question "are you now taking diabetic pills to lower your blood sugar?" Those people who did not meet any of those 4 criteria were classified as not having diabetes. Total cholesterol concentrations and HDL-C were measured with an enzymatic method on Roche Modular P chemistry analyzers from 2007 to 2012, and on Roche Modular P and Roche Cobas 6000 chemistry analyzers during the 2013-2014 cycle.[16]

Covariates

Other demographic data included race/ethnicity (non-Hispanic White, non-Hispanic Black, Mexican American, other), educational level (<high school graduate, high school graduate, and >high school graduate), and poverty income ratio (PIR)–the ratio of household income to the poverty threshold after accounting for inflation and family size (<1.3, 1.3-3.49, ≥3.5, missing).[17] Participants with missing PIR (n = 725) were recoded as a separate category.

Body mass index (BMI, kg/m^2) values were calculated using measured height and weight values. Leisure-time physical activity (PA) was categorized into 3 groups: (1) *meeting the recommended amount of PA*, defined as ≥150 minutes of moderate or ≥ 75 minutes of vigorous intensity/wk; (2) *not meeting the recommended amount of PA*, defined as 1-149 minutes of moderate or 1-74 minutes of vigorous intensity/wk; and (3) *inactive*, defined as 0 min/wk.[18] The 2010 Healthy Eating Index (HEI-2010) is a measure of diet quality derived from the first-day 24-hour dietary recall. The HEI-2010 scores range from 0 to 100, with a higher score indicating a healthier diet.[19] Sleep disorder status was determined by the question, "Have you ever been told by a doctor or other health professional that you have a sleep disorder?" Depression symptoms were assessed using the Patient Health Questionnaire (PHQ-9), a validated 9-item screening instrument that asks about the frequency of symptoms of depression over the past 2 weeks.[20] Those who responded "not at all," "several days," "more than half the day," and "nearly every day" received a score of 0-3, respectively. PHQ-9 scores range from 0 to 27, with higher scores indicating more severe depression. The PHQ-9 scores ≥10 had a sensitivity of 88% and a specificity of 88% for major depression.[20] We classified participants as having depression (PHQ-9 score ≥10) and nondepression (PHQ-9 score <10). Participants who did not complete the PHQ-9 (n = 1118) were included as a separate depression category.

Statistical analysis

Among 16,478 NHANES 2007-2014 participants aged 30-74 years, 3703 (22.5%) were excluded, including 99 pregnant women; 1302 participants with self-reported coronary heart disease, myocardial infarction, or stroke at baseline; and 2302 participants with missing data (sleep duration, n = 19; CVD risk factors, n = 1473; covariates, n = 810). This left 12,775 participants for analysis. Descriptive statistics for participant characteristics by sleep duration category are reported in Table 1.

We estimated the weighted prevalence and means (95% confidence intervals [CI]) of selected covariates by categories of self-reported sleep duration and tested for significance across sleep duration categories based on t tests for continuous variables and Wald F tests for categorical variables. We performed multivariable linear regression analysis to examine the association between EHA and sleep duration. We estimated age, age squared, sex, race/ethnicity, educational attainment, and PIR adjusted mean EHA (95% CI) (model 1), and additional adjusted means, which included the variables from model 1 as well as BMI, physical activity, HEI-2010, sleep disorder, and depression status (model 2). We tested for interactions between EHA and selected covariates by including cross-product terms in the multivariable regression models based on Wald F tests and presented *P* values for interaction. We conducted stratified analyses to explore whether the associations between EHA and sleep duration varied by age group (30-44, 45-59, and 60-74 years), race/ethnicity, educational attainment (<high school, high school or GED, and >high school), PIR (<1.3, 1.3-3.49, and ≥3.5), or BMI (normal, overweight, and obese). Additionally, we analyzed the association between sleep duration and the risk of having an EHA ≥10 years. To do so, we dichotomized EHA into ≥10 years versus <10 years (1/0) and used multivariable logistic regression to estimate the adjusted prevalence ratio (95% CI) with 7 hours as reference. Many studies suggested the nonlinear relationship between sleep duration and

risk for CVD events and deaths.[21] We used sleep duration as a continuous variable and included the quadratic term of sleep duration in the multivariable linear or logistic regression models to test for linear or quadratic associations. Data were analyzed using Statistical Analysis System (SAS version 9.3) and Statistical Software for Analyzing Correlated Data (SUDAAN version 11) using procedures that accounted for the survey's complex sampling design. All tests were 2-sided, and $P < .05$ was considered significant.

Results

Among the study population, 13.4% (95% CI 12.5-14.3) reported a sleep duration of ≤5 hours, 24.2% (23.1-25.2) reported 6 hours, 31.0% (29.8-32.3) reported 7 hours, 25.9% (25.0-26.9) reported 8 hours, and 5.5% (5.0-6.1) reported ≥9 hours (Table 1). There were statistically significant increases in the mean age and proportion of women, non-Hispanic Whites, adults living in households with higher incomes (PIR ≥3.5), and nonsmokers across increasing sleep duration categories. There were significant decreases in mean BMI and SBP, in addition to significant increases in HDL cholesterol levels, across increasing sleep duration categories (Table 1).

We observed a nonlinear association between sleep duration and EHA among all participants ($P = .015$ for quadratic trend), which remained consistent for women ($P = .035$) but not for men ($P = .257$) (Table 2 model 2). Adjusted mean EHA was significantly higher for adults sleeping ≤5 hours (5.1 years, $P < .001$), 6 hours (4.5 years, $P < .05$), or 8 hours (4.5, $P < .05$) compared with adults sleeping 7 hours (3.7 years) (Table 2). Among men, the adjusted mean EHA was significantly higher among those sleeping ≤5 hours (5.6 years, $P < .05$) or 8 hours (5.4 years, $P < .05$) compared with 7 hours (4.2 years). Among women, the adjusted mean EHA was significantly higher among those sleeping ≤5 hours (4.7 years, $P < .05$) compared with 7 hours (3.2 years). There were some differences in sex-stratified results with respect to patterns by age, race/ethnicity, and socioeconomic status (Table 3). For example, short sleep duration was associated with higher EHA among both non-Hispanic White men and women, remained consistent among non-Hispanic Black women and Mexican American men, although not statistically significant, but not among non-Hispanic men and Mexican American women.

We saw a nonlinear relationship between sleep duration and the risk of having an EHA ≥10 years among all participants ($P = .028$ for quadratic trend) and among women ($P = .014$) but not among men ($P = .445$) (Table 4). With 7 hours as reference, the prevalence ratio for the risk of having an EHA ≥10 years increased approximately 15% for those who slept 6 hours and 25% for those who slept ≤5 hours. These associations were similar among men and women (Table 4). There were no significant association between those who slept 8 or ≥9 hours compared to those who slept 7 hours for risk of EHA ≥10 years.

Discussion

Using data from nationally representative samples, our results suggested a nonlinear relationship between sleep duration and EHA or risk of having an EHA ≥10 years among all participants and among women but not among men. Participants who slept ≤5, 6, or

8 hours had adjusted EHAs 1.4, 0.8, and 0.8 years older, respectively, than those who slept 7 hours. Results from the stratified analyses suggested that the association between sleep duration and EHA varied by sex, age group (30-44, 45-59, and 60-74 years), and socioeconomic status. Overall, adjusted EHA was significantly higher among participants with lower education levels, lower PIRs, and obesity regardless of sleep duration.

Many studies have suggested that short or long sleep duration is associated with metabolic syndrome[22–24] and individual CVD risk factors, such as increased blood pressure or hypertension,[4,24–33] dyslipidemia,[34–36] obesity,[34,37] and diabetes.[38,39] Our finding of a nonlinear relationship between sleep duration and EHA or risk of having an EHA ≥10 years was consistent with other studies that used the FRS[40] or the atherosclerotic cardiovascular disease Pooled Cohort Risk Equations to predict 10-year cardiovascular risk,[4] and findings from many cohort studies of sleep duration and risk of CVD events or CVD mortality.[21,41,42] However, as far as we are aware, our study is the first to present sleep's impact on cardiovascular risk in a more understandable and translatable way by using EHA to express CVD risk. Evidence from population-based epidemiologic studies and sleep intervention studies suggest the importance of adequate sleep in reducing the risk of CVD and mortality. Sleep duration coupled with EHA might provide a new and simplified way for clinicians and public health workers to communicate sleep's impact on CVD risk and may help their patients better understand the CVD risks and benefits associated with sleep even among those who exercise regularly and maintain healthy diets.

Although the nonlinear association remained largely consistent by age, sex, race/ethnicity, and other covariates, there were a few noticeable differences. For example, there appeared to be no association between sleep duration and EHA among non-Hispanic Black men and Mexican American women. The reasons for the observed nonassociation are unclear but may in part be due to the fact that the FRS was developed among mainly non-Hispanic White participants or the limited sample size, especially among women, for the stratified analysis. Adjusted EHA was higher among participants with lower education level, lower PIR, or obesity. These findings were consistent with the findings of other studies that showed the higher prevalence of CVD risk factors associated with lower SES[43,44] and among people with obesity.[5,45,46]

Several biological mechanisms have been identified to explain the harmful effects of insufficient sleep on the cardiovascular system. One possible way short sleep duration may increase CVD risk is by modifying inflammatory and cholesterol pathways–as sleep decreases, the expression of genes encoding cholesterol transporters decreases, and the expression in pathways involved in inflammatory responses increases.[47] Studies suggest that short sleep increases the activity of pathways for β-cell activation, interleukin-8 production, and NF-kB signaling.[48] This activity, if maintained, would lead to chronic, low-level inflammation, which contributes to CVD risk.[48] Short sleep also causes cells to downregulate the satiety hormone leptin and upregulate the appetite-stimulating hormone ghrelin.[49] This increases hunger and food intake, resulting in overeating, which could ultimately lead to obesity.[48,50] Furthermore, experimentally induced sleep loss in healthy people has been found to decrease insulin sensitivity without adequate compensation in β-cell function.[49] This results in reduced glucose tolerance and increased diabetes risk.[48]

In contrast with short sleep, there are no studies published to date that we are aware of on possible mechanisms linking long sleep duration to cardiovascular risk. Several studies considered the possibility that a combination of variables, including unemployment, impaired mental health, and low SES, could account for the often observed association between long sleep and adverse health risks.[51–53] Another important reason to concentrate on short sleep duration is that many more people were short sleepers than long sleepers.[51] In our study, only 5.5% of participants reported sleeping ≥9 hours as compared to 37.6% reporting ≤6 hours. It is worth noting that our results suggested that participants who slept 8 hours had significantly higher EHAs compared to those who slept 7 hours. Some studies considered 8 hours an adequate amount of sleep, as it was associated with lower CVD events and mortality[31,54,55]; however, other studies showed that 8 hours of sleep was associated with increased risk for CVD events and mortality.[4,21,56] The higher EHA may be mainly driven by the high prevalence of diabetes and current smoking among participants reporting 8 hours of sleep compared to those reporting 7 hours of sleep (Table 1). However, there were no significant differences in the risk of having an EHA ≥10 years between those who reported 8 or 9 hours of sleep compared to those who slept 7 hours. The reasons for this inconsistency were unclear and could be due to the inadequate adjustment of confounders and limited sample size for participants who slept ≥9 hours. Further study is needed to clarify these issues.

The main strengths of our study include the use of nationally representative samples to study the association between sleep duration and EHA and control for a comprehensive number of covariates in addition to the CVD risk factors used in the FRS. The findings in this study are subject to at least 4 limitations. First, the Framingham Heart Study consisted of predominantly non-Hispanic Whites, and the heart age algorithm may not apply to other racial/ethnic groups. Although the Pooled Cohort Risk Equations included non-Hispanic White and non-Hispanic Black adults in CVD risk prediction, the parameters for calculating heart age were not available.[57] Second, NHANES asked for participants' sleep duration on workdays or weekdays, with no data about sleep duration on weekends or free days. The self-reported sleep duration in NHANES might reflect the "typical" rather than the mean sleep duration. Studies have suggested that social jetlag, the absolute difference between mid-sleep on workdays or weekdays and mid-sleep on weekends or free days, is associated with increased risk for obesity, diabetes, and risk of CVD.[58,59] Third, limited sample size might contribute to the observed differences between sleep duration and EHA in the stratified analyses. Finally, we adjusted for diagnosed sleep disorders in our analysis; however, a majority of sleep disorders may be undiagnosed among US adults.[60]

Conclusions

One in 3 US adults aged 30 to 74 years reported sleeping less than the recommended 7 hours. The mean adjusted EHA appeared to be lowest among adults who reported sleeping 7 hours per night and increased as adults reported sleeping fewer or more hours. Sleep duration coupled with EHA may prove helpful for clinicians to communicate with their patients and for patients to better understand the cardiovascular risks and benefits associated with sleep.

Acknowledgments

We thank Robert Merritt, Division for Heart Disease and Stroke Prevention, Centers for Disease Control and Prevention, for his helpful comments and support of the project. We thank Dr Jeff Durmer, Fusion Health, for his insight and suggestions.

Disclaimer:

The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

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Table 1

Characteristics of participants aged 30-74 years by self-reported sleep duration, NHANES 2007-2014

Characteristics	Total n = 12,775	Self-reported sleep duration (h)					*P* value[a]
		≤5 h n = 2048	6 h n = 3216	7 h n = 3523	8 h n = 3255	≥9 h n = 733	
Demographics							
Total, % (95% CI)	100	13.4 (12.5-14.3)	24.2 (23.1-25.2)	31.0 (29.8-32.3)	25.9 (25.0-26.9)	5.5 (5.0-6.1)	
Age							
Mean (95% CI)	49.2 (48.9-49.6)	48.1 (47.4-48.8)	48.7 (48.3-49.2)	49.3 (48.8-49.8)	49.7 (49.0-50.4)	51.3 (50.2-52.4)	**<.001**
Age group, % (95% CI)							
30-44	38.5 (37.0-40.0)	39.7 (36.7-42.6)	39.2 (37.1-41.3)	37.3 (35.0-39.7)	39.4 (36.8-42.2)	34.5 (30.5-38.7)	**<.001**
45-59	39.6 (38.3-41.0)	44.0 (41.0-47.1)	41.3 (38.8-43.9)	40.6 (38.4-42.8)	40.6 (33.6-38.5)	32.7 (28.3-37.5)	
60-74	21.9 (20.9-23.0)	16.3 (14.3-18.6)	19.5 (17.9-21.2)	22.1 (20.2-24.1)	24.6 (22.4-26.9)	32.7 (28.4-37.3)	
Sex, % (95% CI)							
Men	48.1 (47.1-49.1)	47.9 (45.3-50.4)	52.5 (49.9-55.1)	50.1 (48.2-52.0)	43.5 (41.0-46.0)	39.2 (34.8-43.8)	**<.001**
Women	51.9 (50.9-53.0)	52.1 (49.6-54.7)	47.5 (44.9-50.1)	49.9 (48.0-51.9)	56.5 (54.0-59.0)	60.8 (56.2-65.3)	
Race/ethnicity, % (95% CI)							
Non-Hispanic White	69.8 (66.2-73.2)	58.3 (53.0-63.3)	66.3 (62.2-70.1)	75.6 (72.2-78.7)	71.2 (67.5-74.7)	72.3 (68.9-79.1)	**<.001**
Non-Hispanic Black	10.4 (8.8-12.2)	20.2 (16.8-24.0)	12.4 (10.2-15.0)	6.5 (5.3-7.8)	8.4 (7.1-9.8)	9.3 (7.1-12.1)	**<.001**
Mexican American	8.2 (6.6-10.3)	7.3 (5.9-9.1)	8.7 (6.7-11.1)	7.4 (5.7-9.4)	9.3 (7.5-11.6)	8.2 (5.8-11.5)	.286
Other	11.6 (10.2-13.2)	14.3 (11.9-17.0)	12.7 (11.1-14.5)	10.6 (9.2-12.2)	11.1 (9.3-13.2)	8.2 (6.0-11.1)	**<.001**
Education, % (95% CI)							
<High school	16.2 (14.6-17.8)	21.6 (19.4-24.0)	15.2 (13.4-17.3)	13.2 (11.3-15.4)	17.0 (15.2-19.0)	19.5 (16.1-23.4)	.572
High school	21.7 (20.3-23.1)	26.5 (24.0-29.0)	23.6 (21.7-25.6)	17.9 (15.9-20.0)	22.0 (20.3-23.9)	21.2 (17.1-26.0)	**.016**
>High school	62.2 (59.8-64.5)	52.0 (48.5-55.4)	61.2 (58.5-63.8)	68.9 (65.7-72.0)	61.0 (58.0-63.8)	59.3 (53.7-64.8)	**.026**
PIR, % (95% CI)[b]							
<1.3	16.7 (15.0-18.5)	23.7 (21.1-26.5)	16.5 (14.7-18.6)	11.9 (10.5-13.4)	17.6 (15.6-19.9)	23.2 (19.3-27.8)	.989
1.3-3.49	30.9 (29.1-32.7)	35.9 (32.6-39.4)	31.3 (29.0-33.6)	28.6 (25.8-31.5)	31.1 (28.6-33.7)	29.0 (25.0-33.4)	**.011**
≥3.5	45.5 (42.9-48.2)	32.2 (28.5-36.2)	44.9 (41.5-48.3)	53.5 (50.1-56.8)	44.3 (41.5-47.2)	41.7 (36.2-47.4)	**.005**
CVD risk factors							
SBP (mm Hg)							
Mean (95% CI)	121.6 (121.1-122.1)	122.6 (121.7-123.5)	121.8 (120.9-122.7)	121.1 (120.4-121.8)	121.8 (120.9-122.7)	120.4 (118.8-122.0)	**.009**

Characteristics	Total n = 12,775	Self-reported sleep duration (h)					
		≤5 h n = 2048	6 h n = 3216	7 h n = 3523	8 h n = 3255	≥9 h n = 733	*P* value[a]
Total cholesterol (mg/dL)							
Mean (95% CI)	200.8 (199.8-201.8)	202.0 (199.6-204.4)	199.1 (197.4-200.8)	200.3 (198.9-201.8)	202.1 (200.1-204.2)	202.1 (197.7-206.5)	.543
HDL-C (mg/dL)							
Mean (95% CI)	53.2 (52.7-53.6)	51.5 (50.5-52.4)	52.2 (51.2-53.2)	53.6 (52.9-54.3)	54.1 (53.2-54.9)	54.4 (52.9-55.9)	<**.001**
Hypertension, % (95% CI)							
No	68.3 (67.0-69.5)	65.9 (63.5-68.3)	66.6 (64.5-68.5)	70.8 (68.7-72.7)	68.3 (65.7-70.7)	67.4 (63.3-71.2)	.284
Yes	31.7 (30.5-33.1)	34.1 (31.7-36.5)	33.4 (31.5-35.5)	29.2 (27.3-31.3)	31.7 (29.3-34.3)	32.6 (28.8-36.7)	
Participants with hypertension under treatment, % (95% CI)	72.1 (69.8-74.3) (n = 4539)	67.7 (63.0-72.0) (n = 814)	72.5 (68.4-76.2) (n = 1181)	73.4 (69.7-76.7) (n = 1130)	72.3 (68.2-76.1) (n = 1143)	75.0 (67.1-81.4) (n = 271)	.113
Diabetes, % (95% CI)							
No	88.0 (87.3-88.8)	85.0 (83.1-86.9)	86.8 (85.2-88.3)	90.4 (89.0-91.7)	88.1 (86.6-89.5)	87.0 (83.4-90.0)	.169
Yes	12.0 (11.3-12.7)	15.0 (13.2-17.0)	13.2 (11.8-14.8)	9.6 (8.3-11.0)	11.9 (10.5-13.4)	13.0 (10.0-16.6)	
Smoke, % (95% CI)							
Nonsmoker	80.3 (79.0-81.5)	69.9 (66.9-72.8)	77.9 (76.0-79.8)	85.2 (83.6-86.7)	82.7 (80.7-84.6)	75.9 (71.2-80.2)	**.002**
Current	19.7 (18.5-21.0)	30.1 (27.3-33.1)	22.1 (20.2-24.0)	14.8 (13.3-16.4)	17.3 (15.4-19.4)	24.1 (19.9-28.8)	
Covariates							
BMI (kg/m^2)							
Mean (95% CI)	29.2 (29.0-29.4)	30.4 (30.0-30.8)	29.6 (29.2-29.9)	28.9 (28.6-29.1)	28.8 (28.4-29.1)	28.8 (28.4-29.3)	<**.001**
Physical activity, % (95% CI)							
Meet recommendation	29.6 (28.1-31.4)	22.9 (20.4-25.6)	27.7 (25.5-30.1)	32.8 (30.2-35.5)	31.7 (29.5-33.9)	28.0 (24.1-32.1)	**.001**
Not meet recommendation	17.0 (15.9-18.2)	14.8 (13.0-16.7)	16.6 (14.5-18.9)	18.2 (16.7-19.8)	17.4 (15.9-19.1)	16.0 (12.8-19.7)	.445
Inactive	53.3 (51.3-55.3)	62.3 (59.5-65.1)	55.7 (52.7-58.6)	49.0 (46.4-51.7)	50.9 (48.3-53.4)	56.1 (51.5-60.6)	**.002**
HEI-2010							
Mean (95% CI)	51.2 (50.5-51.8)	47.1 (46.3-48.0)	50.1 (49.2-51.0)	52.7 (51.9-53.6)	52.6 (51.7-53.4)	50.7 (49.0-52.4)	<**.001**

[a] *P* value for testing significance across categories of self-reported sleep duration based on *t* test for continuous variable and Wald F test for categorical variables.

[b] There were 725 participants with missing PIR.

Table 2

Mean EHA (95% CI) by self-reported sleep duration and sex, NHANES 2007-2014

	Self-reported sleep duration (h)				
	≤5 h n = 2048	**6 h n = 3216**	**7 h n = 3523**	**8 h n = 3255**	**≥9 h n = 733**
Total[a]					
Model 1[b]	5.9 (5.3-6.6)**	4.8 (4.1-5.4)*	3.4 (3.0-3.9)	4.2 (3.7-4.7)*	4.0 (3.1-4.8)
Model 2[c]	5.1 (4.8-5.8)**	4.5 (3.9-5.1)*	3.7 (3.3-4.0)	4.5 (4.1-5.0)*	4.1 (3.3-4.9)
Men	n = 999	n = 1626	n = 1731	n = 1479	n = 310
Model 1[b]	6.3 (5.3-7.3)**	5.2 (4.4-6.0)*	4.0 (3.4-4.6)	5.3 (4.0-6.0)	4.5 (3.0-6.0)
Model 2[c]	5.6 (4.7-6.5)*	5.1 (4.4-5.9)	4.2 (3.6-4.8)	5.4 (4.8-6.1)*	4.6 (3.2-6.1)
Women	n = 1049	n = 1590	n = 1792	n = 1776	n = 423
Model 1[b]	5.6 (4.6-6.6)**	4.3 (3.6-5.0)*	2.9 (2.3-3.6)	3.3 (2.6-3.9)	3.7 (2.7-4.7)
Model 2[c]	4.7 (3.6-5.8)*	3.9 (3.2-4.6)	3.2 (2.5-3.7)	3.8 (3.2-4.3)	3.7 (2.7-4.7)

*P value < .05.

**P value < .001 in EHA as compared to 7 hours of sleep.

[a]P values for the interaction between sex and sleep duration were .143 for model 1 and .573 for model 2 based on Wald F test; P values for quadratic trend between sleep duration and EHA were .015, .257 and .035 for total, men, and women, respectively, based on Wald F test; all tests were 2-sided.

[b]Model 1 adjusted for age, age squared, sex, race/ethnicity, educational attainment, and PIR.

[c]Model 2 additionally adjusted for BMI, physical activity, HEI-2010, sleep disorder, and depression status.

Table 3

Adjusted mean EHA (95% CI) by self-reported sleep duration, sex, and selected covariates, NHANES 2007-2014

	Men[a,b]					Women[a,b]				
	Self-reported sleep duration (h)					Self-reported sleep duration (h)				
Characteristics	**≤5 h**	**6 h**	**7 h**	**8 h**	**≥9 h**	**≤5 h**	**6 h**	**7 h**	**8 h**	**≥9 h**
Age										
30-44	4.6 (3.0-6.2)	4.2 (3.0-5.3)	3.3 (2.2-4.3)	4.0 (2.7-5.3)	3.9 (2.4-5.5)	2.3 (0.8-3.8)	2.6 (1.4-3.9)	2.0 (0.9-3.2)	2.2 (1.2-3.2)	3.2 (1.6-4.7)[c]
45-59	6.0 (4.7-7.4)	6.1 (5.0-7.2)	4.8 (3.7-5.8)	6.7 (5.6-7.8)[*]	5.5 (2.1-9.0)[c]	5.3 (3.5-7.1)	4.5 (3.2-5.7)	4.2 (3.1-5.3)	4.5 (3.2-5.7)	3.4 (1.3-5.5)[c]
60-74	7.0 (4.1-4.3)	5.0 (2.8-7.1)	5.0 (2.8-7.2)	5.9 (3.5-8.3)	4.4 (1.0-7.8)[c]	8.2 (5.3-3.2)[**]	4.9 (2.6-7.3)	3.2 (1.7-4.7)	5.0 (2.9-7.1)	4.7 (1.9-7.4)
Race/ethnicity										
Non-Hispanic White	5.7 (4.5-6.9)[*]	5.0 (4.0-5.9)	3.8 (3.1-4.6)	5.5 (4.6-6.3)[*]	3.7 (1.9-5.5)	4.9 (3.4-6.5)[*]	3.8 (2.8-4.7)	2.7 (1.9-3.4)	3.5 (2.7-4.3)	3.7 (2.5-4.9)
Non-Hispanic Black	5.0 (3.8-6.2)	5.7 (4.6-6.9)	5.8 (4.3-7.2)	6.0 (4.6-7.4)	6.1 (2.9-9.3)	6.0 (4.6-7.5)	5.9 (4.6-7.2)	5.4 (4.1-6.7)	6.1 (4.4-7.7)	3.8 (1.1-6.5)
Mexican American	5.0 (2.5-4.6)	4.4 (3.2-5.6)	3.9 (2.9-4.9)	5.0 (3.3-6.6)	4.5 (2.2-6.7)	2.5 (0.1-3.4)	2.3 (1.0-3.6)	2.6 (1.0-4.1)	2.3 (0.9-3.7)[c]	2.8 (0.0-5.6)[c]
Education										
<High school	7.9 (6.2-9.5)[*]	5.8 (4.5-7.1)	5.1 (3.7-6.6)	5.9 (4.7-7.1)	6.3 (3.9-8.7)	6.0 (3.6-8.3)	6.9 (5.4-8.4)	5.9 (44-7.4)	5.8 (44-7.2)	5.7 (3.3-8.0)
High school	6.8 (4.8-8.9)	7.1 (5.4-8.8)	4.8 (3.2-6.4)	6.9 (5.5-8.3)	5.0 (2.6-7.4)	5.1 (3.6-6.7)	4.4 (3.1-5.7)	4.0 (2.5-5.5)	3.7 (1.9-5.6)	5.0 (2.0-8.0)
>High school	4.3 (3.0-6.6)	4.1 (3.3-5.0)	3.6 (3.0-4.2)	4.8 (3.9-5.7)[*]	4.0 (1.5-6.4)[c]	4.5 (3.1-7.4)[*]	3.0 (2.1-3.9)	2.3 (1.3-3.2)	3.3 (2.5-4.0)	2.8 (1.5-4.1)
PIR										
<1.3	8.7 (7.1-10.4)[*]	7.0 (5.6-8.4)	6.3 (5.1-7.5)	8.4 (6.9-9.9)[*]	7.2 (4.5-10.0)	7.1 (5.9-8.4)	6.5 (5.2-7.8)	6.0 (4.4-7.5)	5.6 (4.2-6.9)	8.2 (6.3-10.1)
1.3-3.49	5.9 (4.3-7.6)[*]	5.4 (4.1-6.8)	4.0 (3.0-4.9)	6.0 (4.9-7.1)[*]	3.9 (0.8-7.0)[c]	4.4 (2.8-5.9)	5.0 (3.9-6.1)	4.0 (2.8-5.1)	5.0 (3.7-6.4)	3.4 (1.2-5.6)[c]
≥3.5	4.0 (2.5-7.5)	4.3 (3.2-5.4)	3.4 (2.6-4.3)	3.9 (2.9-4.9)	4.7 (2.3-7.1)	4.8 (2.4-7.5)[*]	1.9 (0.7-3.1)[c]	1.5 (0.4-2.6)[c]	2.3 (1.3-3.3)	2.2 (0.5-3.9)[c]
BMI										
Obese (≥30)	7.9 (6.2-1.5)	7.7 (6.5-9.0)	7.7 (6.3-9.0)	9.0 (7.7-10.3)	7.3 (4.8-9.9)	7.7 (6.2-0.5)	8.0 (6.9-9.0)	6.6 (5.4-7.9)	7.0 (6.0-8.1)	5.4 (3.7-7.2)
Overweight (25-<30)	4.9 (3.4-6.4)	4.1 (3.1-5.1)	3.1 (2.3-3.9)	4.4 (3.4-5.3)[*]	4.8 (2.0-7.6)[c]	3.9 (2.2-5.6)	2.2 (0.8-3.6)[c]	2.4 (1.4-3.4)	3.9 (2.9-4.9)[*]	5.3 (2.9-7.6)[*]
Normal (<25)	3.3 (2.0-4.7)[*]	2.7 (1.3-4.1)[*]	0.5 (−0.5-1.5)[c]	1.5 (0.4-2.5)[c]	0.3 (−1.7-2.2)[c]	2.2 (0.0-4.5)[*c]	0.4 (−0.9-1.7)[c]	−0.4 (−1.3-0.5)[c]	−0.4 (−1.5-0.7)[c]	0.1 (−2.0-2.3)[c]

[*] *P* value < .05.

[**] *P* value < .001 in EHA as compared to 7 hours of sleep or pairwise comparisons.

[a] Adjusted for age, sex, race/ethnicity, educational attainment, PIR, BMI, physical activity, HEI-2010, sleep disorder, and depression status.

[b] *P* values for the interaction between subgroups and sleep duration were .744 for age group, .027 for race/ethnicity, .335 for education, .422 for PIR, and .161 for BMI among men. The corresponding *P* values for interactions were .117, .652, .631, .435, and .020 for women based on Wald F test; *P* values for quadratic trend between sleep duration and EHA were .053 for non-Hispanic White women, .025 for women aged 60-74 years, .008 for men with <high school, .056 for women in PIR <1.3, and .001 for overweight women, respectively, based Wald F test; all tests were 2-sided.

[c] The relative standard error of the point estimate is greater than 30%, and the point estimate might not be stable.

Table 4

Adjusted prevalence ratio (95% CI) of EHA ≥10 years by self-reported sleep duration and sex, NHANES 2007-2014

	Self-reported sleep duration (h)				
	≤5 h n = 2048	**6 h n = 3216**	**7 h n = 3523**	**8 h n = 3255**	**≥9 h n = 733**
Total[a]					
Model 1[b]	1.38 (1.22-1.57)	1.21 (1.06-1.39)	1.00	1.09 (0.98-1.22)	1.08 (0.95-1.22)
Model 2[c]	1.25 (1.11-1.40)	1.15 (1.01-1.31)	1.00	1.10 (0.99-1.22)	1.06 (0.94-1.19)
Men	n = 999	n = 1626	n = 1731	n = 1479	n = 310
Model 1[b]	1.40 (1.16-1.68)	1.20 (0.99-1.46)	1.00	1.11 (0.92-1.34)	0.95 (0.70-1.29)
Model 2[c]	1.27 (1.06-1.52)	1.16 (0.96-1.40)	1.00	1.10 (0.93-1.30)	0.93 (0.68-1.26)
Women	n = 1049	n = 1590	n = 1792	n = 1776	n = 423
Model 1[b]	1.37 (1.16-1.61)	1.21 (1.05-1.40)	1.00	1.08 (0.95-1.24)	1.19 (1.00-1.41)
Model 2[c]	1.22 (1.05-1.43)	1.13 (0.98-1.30)	1.00	1.10 (0.96-1.24)	1.16 (0.99-1.37)

[a] *P* values for the interaction between sex and sleep duration were .677 for model 1 and .703 for model 2 based on Wald F test; *P* values for quadratic trend between sleep duration and EHA were .028, .445, and .014 for total, men, and women, respectively, based on Wald F test, all tests were 2-sided.

[b] Model 1 adjusted for age, sex, race/ethnicity, educational attainment, and PIR.

[c] Model 2 additionally adjusted for physical activity, sleep disorder, depression status, HEI-2010, and BMI.