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Habitual Sleep Duration Associated with Self-Reported and Objectively-Determined Cardiometabolic Risk Factors

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Abstract

Background—Self-reported short and/or long sleep duration have been associated with adverse cardiometabolic health outcomes in laboratory and epidemiologic studies, but interpretation of such data has been limited by methodological issues.

Methods—Adult respondents of the 2007–2008 US National Health and Nutrition Examination Survey (NHANES) were examined in a cross-sectional analysis (N=5,649). Self-reported sleep duration was categorized as very short (<5hrs), short (5–6hrs), normal (7–8hrs) or long (≥9hrs). Obesity, diabetes, hypertension, and hyperlipidemia were assessed by self-reported history and objectively. Statistical analyses included univariate comparisons across sleep duration categories for all variables. Binary logistic regression analyses, cardiometabolic factor as outcome and with sleep duration category as predictor, were assessed with and without covariates. Observed relationships were further assessed for dependence on race/ethnicity.

Results—In adjusted analyses, very short sleep was associated with self-reported hypertension (OR=2.02, 95%CI[1.45, 2.81], p<0.0001), self-reported hyperlipidemia (OR=1.96, 95%CI[1.43, 2.69], p<0.0001), objective hyperlipidemia (OR=1.41, 95%CI[1.04, 1.91], p=0.03), self-reported diabetes (OR=1.76, 95%CI[1.13, 2.74], p=0.01), and objective obesity (OR=1.53, 95%CI[1.13, 2.06], p=0.005). Regarding short sleep (5–6hrs), in adjusted analyses, elevated risk was seen for self-reported hypertension (OR=1.22, 95%CI[1.02, 1.45], p=0.03) self-reported obesity (OR=1.21, 95%CI[1.03, 1.43], p=0.02) and objective obesity (OR=1.17, 95%CI[1.00, 1.38], p<0.05). Regarding long sleep (≥9hrs), no elevated risk was found for any outcomes. Interactions with Race/Ethnicity were significant for all outcomes; race/ethnicity differences in patterns of risk varied by outcome studied. In particular, the relationship between very short sleep and obesity was strongest among Blacks/African-Americans and the relationship between short sleep and

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hypertension is strongest among non-Hispanic Whites, Blacks/African-Americans, and non-Mexican Hispanics/Latinos.

Conclusions—Short sleep duration is associated with self-reported and objectively-determined adverse cardiometabolic outcomes, even after adjustment for many covariates. Also, these patterns of risk depend on race/ethnicity.

Keywords

Sleep Duration; Epidemiology; Obesity; Cardiovascular Disease; Hypertension; Cholesterol; Diabetes

INTRODUCTION

Both self-reported short and long sleep duration have been associated with elevated risk of obesity in both children and adults[1-7], as well as cardiovascular disease[8-10], diabetes[4, 11, 12], hypertension[2, 13, 14], and other negative health outcomes[2, 15-17], including all-cause mortality[16, 18-21]. These findings have been summarized in reviews utilizing both quantitative[8, 19, 20, 22, 23] and narrative[3, 16, 17, 24-28] approaches.

Small-scale experimental studies have shown that short-term sleep deprivation may lead to metabolic dysregulation[29-34], elevated inflammation[35, 36], and elevated blood pressure[37]. Whether these findings translate to associations with habitual sleep in general population samples remains unclear. Epidemiologic studies, while typically more generalizable, carry methodological limitations such as inconsistent definitions of ‘short’ and ‘long’ sleep duration categories, inconsistent assessments (e.g., differently-worded survey items)[17], variable measurement techniques (e.g., diaries, actigraphy, polysomnography)[38], and occasionally non-representative samples (i.e., large scale samples that are restricted to defined regions[39], older[40, 41] or younger[6, 13, 42] adults, or a single gender[40, 43-45] or race/ethnicity[46]). Further, and, perhaps most importantly, assessments of disease and/or disease risk are often confined to subjective questions (e.g., “Have you ever been diagnosed with...”), rather than objective determinations based on medication use and physiologic measurements (e.g., blood pressure, serum cholesterol, fasting glucose levels). It is plausible that the use of such measures, in combination with measures of self-rated sleep duration, may allow for a more precise determination of whether laboratory findings generalize to the population at large. In the present study, associations between sleep duration and cardiometabolic risk factors were evaluated using data from the 2007-2008 National Health and Nutrition Examination Survey (NHANES). Specifically, short and long sleep duration were assessed for their association with self-reported and objective measures of hypertension, hyperlipidemia, diabetes, and/or obesity while utilizing (1) a census-weighted, nationally-representative sample, (2) previously established categories of self-reported sleep duration[2, 17, 47, 48], (3) simultaneous assessment of risk domains, and (4) statistical controls for differential effects of race/ethnicity. It was hypothesized that short and long sleep duration, relative to normal sleep duration, would be associated with increased cardiometabolic risk, even after accounting for self-reported/objective differences in risk assessment and confounding factors. The primary goal of these analyses is to replicate and extend previous findings in a gold-standard, representative sample. Specifically, we attempt to clarify whether the relationships differ depending on how outcomes are determined and we attempt to demonstrate that these relationships are further complicated by race/ethnicity differences, which have only rarely been examined[49, 50].

METHODS

Data Source

Participants included respondents to the 2007-2008 National Health and Nutrition Examination Survey (NHANES), a national survey conducted by the Centers for Disease Control and Prevention to assess the health and nutritional characteristics of children and adults[51-53]. The NHANES methodology, surveys, manuals, and procedures are available on the NHANES website (<http://www.cdc.gov/nchs/nhanes>). Participants responded to questionnaires assessing demographic, socioeconomic, health, and other domains during face-to-face, in-home interviews. To supplement self-report data, physical examination data were gathered in mobile medical facilities. Additional laboratory tests including blood samples were also collected on-site. African Americans, Hispanics, and adults over 60 were over-sampled to increase the power to detect differences in these groups. The NHANES is designed to ensure generalizability to the entire population across all ages. Given the complexity of the survey design, coupled with variable probabilities of selection, the data used in the following analyses were also weighted to control for representativeness, by following the procedures outlined in the current NHANES Analytic and Reporting Guidelines[52]. Presently, data on adults ages 18-80+ years (M=49.3, SD=18.6) with complete data on our variables of interest were analyzed. All respondents provided informed consent. Consent forms are available online (http://www.cdc.gov/nchs/nhanes/nhanes2007-2008/current_nhanes_07_08.htm).

Measures Sleep Duration—Sleep duration was assessed with the survey item, “How much sleep do you usually get at night on weekdays or workdays?” Responses were coded in whole numbers. Based on previous studies[2, 47, 54], responses were categorized as “very short” (<5 hours), short (5-6 hours), normal (7-8 hours) or long (9 hours). These categories were based on existing literature examining cardiovascular and metabolic consequences of habitual sleep duration and experimental sleep restriction.

Hypertension—Self-reported hypertension was assessed with the survey item, “Have you ever been told by a doctor or other health professional that you had hypertension, also called high blood pressure?” Objective hypertension was assessed as any one of the following: (1) endorsement of the question, “Because of high blood pressure/hypertension, have you ever been told to take prescribed medicine?” (2) a report of a current antihypertensive medication during a medical history evaluation, or (3) measured blood pressure during the medical examination of >140/90 mmHg. Procedures for blood pressure collection, including equipment lists, equipment maintenance standards, test administration procedures, and interpretation guidelines, are in the publicly-available NHANES Physician Examination Procedures Manual[55]. Briefly, measurements are obtained from rested, seated participants, with three measurements at least 30 seconds apart, and a fourth reading if any of the first three were questionable. The values reported are the mean of the available non-questionable recordings.

Hyperlipidemia—Self-reported hyperlipidemia was assessed with the survey item, “Have you ever been told by a doctor or other health professional that your blood cholesterol level was high?” Objective hyperlipidemia was assessed as any of the following: (1) endorsement of the question, “To lower your blood cholesterol, have you ever been told by a doctor or other health professional to take prescribed medicine?” (2) a report of a current statin or other lipid lowering medication during a medical history evaluation, or (3) measured serum cholesterol during the medical examination of >240 mg/dL.

Diabetes—Self-reported diabetes was assessed with the survey item, “Other than during pregnancy, have you ever been told by a doctor or other health professional that you have diabetes or sugar diabetes?” Objective diabetes was assessed as any of the following: (1) endorsement of the question, “Are you now taking insulin?” or “Are you now taking diabetic pills to lower your blood sugar?” (2) a report of a current hypoglycemic or other diabetic medication during a medical history evaluation, or (3) measured fasting glucose of >125 mg/dL.

Obesity—Body mass index (BMI) was computed using both self-reported height and weight, and objectively-measured height and weight recorded during a physical exam. A BMI ≥ 30 Kg/m² is considered to be obese.

Data Management and Statistical Analyses Covariates—The present study adjusted for potential confounding factors by including the following covariates in the analyses: age, sex, race/ethnicity (Non-Hispanic White, Black/African-American, Mexican-American, Other Hispanic/Latino, or Asian/Other), and acculturation (English only, English & Spanish, Spanish only, or other), as all of these factors are known to affect sleep behaviors as well as health outcomes[56-59]; Socioeconomic factors suspected to affect sleep and health outcomes[1, 56], which included education level (less than high school, high school, some college, college graduate), access to health insurance (none, public, or private), home ownership (yes or no), and food security (normal borderline, low, or very low; measured using standard USDA criteria[60]). Other health risk factors included current smoking (yes or no) and regular caffeine use (yes or no). Other covariates were considered, including income, alcohol, and others. However, non-response to these items (e.g., >800 missing responses to alcohol items) suggested that their inclusion as a covariate would significantly impinge on inferences. Other potential covariates were also not included. Since a nearly unlimited number of factors could potentially be associated with any of the cardiometabolic risk factors assessed in the current study, it is unlikely that any model could fully account for these. Further, adding many covariates introduces the problem of compounded measurement error, especially for self-reported measures. In addition, since many of those variables that may affect cardiometabolic risk factors could at least partially be a consequence of changes in sleep, including these as covariates and then examining the unique variance explained by sleep leads to biased statistical estimates. Therefore, the present study adopts an approach of inclusion for the sake of generalizability rather than exclusion based on potential confounding conditions. This also allows for maximal comparison across studies, though interpretations of these data need to be made in the context that population surveys may include individuals with other conditions.

Summary Variables—Cardiometabolic outcomes, including self-reported and objective hypertension, hyperlipidemia, diabetes and obesity, were operationalized as dichotomous variables. Sleep duration was operationalized as a categorical variable, with normal sleep (7-8 hours/night) as a reference. All summary variables were inspected with regards to distribution and central tendency.

Analytic Strategy—Univariate comparisons across sleep duration categories for all variables were evaluated using Chi-Square tests. For the primary analyses, binary logistic regression analyses, with cardiometabolic outcome and sleep duration category as predictor, were assessed with and without covariates. To assess whether the observed relationships depended on race/ethnicity (based on prior sleep and cardiovascular literature), terms were evaluated for sleep duration by race/ethnicity interactions, in a model with all other covariates. If an interaction term was significant, analyses stratified by race/ethnicity were explored. Note: the number of covariates entered into the analyses was limited to mitigate

reporting bias and to avoid the creation of models that were over-controlled. Two-tailed P-values of <0.05 were used as the threshold for the determination of significance. All statistical calculations were performed using STATA/SE version 12 (STATA Corp, College Station TX).

RESULTS

Characteristics of the Sample

The analyses included data from $N=5,649$ respondents, whose characteristics are summarized in **Table 1**. Of $N=6,220$ participants who responded to the sleep duration item, 571 were excluded because they were missing some, or all, of the following: at least one of the sociodemographic or socioeconomic covariates ($N=94$), smoking ($N=154$), hypertension ($N=6$), diabetes ($N=6$), and objective height and weight ($N=311$). Chi-square tests show group differences according to demographic, socioeconomic and health variables, supporting their inclusion as covariates. In addition, overall group differences were seen for self-reported and objective hypertension, hyperlipidemia, diabetes, and obesity.

Regression Analyses—Results of binary logistic regression analyses, using normal sleep (7-8 hours) as the reference category can be seen in **Table 2**. Unadjusted analyses revealed that very short sleep (<5 hrs) was associated with elevated risk of all outcomes, relative to normal sleep. After adjustment for covariates, significant relationships remained for self-reported hypertension, self-reported and objective hyperlipidemia, self-reported diabetes, and objective obesity. All other variables were attenuated to trends ($p<0.10$). Regarding short sleep (5-6hrs), in unadjusted analyses, short sleep (5-6hrs) was associated with elevated risk was seen for self-reported and objective hypertension and self-reported and objective obesity. In adjusted analyses, all relationships except objective hypertension persisted. Regarding long sleep (>9 hrs), elevated risk was found only for objective diabetes, in unadjusted analyses only.

To ensure that the observed relationships were not confounded by obesity, adjusted analyses were recomputed, including BMI as a covariate. All relationships remained significant except for elevated hyperlipidemia in very short sleep ($p=0.052$).

Race/Ethnicity Interactions

In adjusted models, significant interactions between sleep duration category and race/ethnicity were found for self-reported hypertension ($p<0.0001$), objective hypertension ($p<0.0001$), self-reported hyperlipidemia ($p=0.007$), objective hyperlipidemia ($p=0.005$), self-reported diabetes ($p=0.001$), objective diabetes ($p=0.0003$), self-reported obesity ($p<0.0001$), and objective obesity ($p=0.0001$). Thus, adjusted analyses were stratified for these outcomes. With the caveat that restricted samples reduce statistical power, especially in the less populous groups, individual findings were explored using stratified analyses. Results of these analyses are found in **Supplementary Figure 1** and **Supplementary Table 1**. Overall, for non-Hispanic white respondents, elevated risk was found for self-reported hypertension, self-reported hyperlipidemia, objective hyperlipidemia, and self-reported diabetes among very short sleepers, no outcomes among short sleepers, and objective hyperlipidemia among long sleepers. For black/African-American respondents, elevated risk of self-reported hypertension, self-reported obesity, and objective obesity were found for very short sleep. For Mexican-Americans, elevated risk of self-reported hypertension and self-reported obesity were found for short sleep and decreased self-reported hyperlipidemia was seen in long sleep. For other Hispanic/Latino respondents, elevated risk of self-reported hypertension was found for very short sleep and objective diabetes for short sleep. Among

Asian/other respondents, elevated risk for self-reported and objective hyperlipidemia were found for very short sleep.

DISCUSSION

The present study evaluated the associations between sleep duration and self-reported/objective cardiometabolic risk factors. The hypotheses that both short and long sleep would be associated with self-reported and objective hypertension, hyperlipidemia, diabetes, and obesity, were partially confirmed. Very short sleep (<5hrs) was most strongly associated with the widest range of adverse outcomes, followed by short sleep (5-6hrs). This is consistent with previous studies, which have generally found habitual sleep in this range to be associated with the largest risk. Altman and colleagues(1) found that sleep duration <5 hours was associated with elevated risk of self-reported obesity, hypertension, hypercholesterolemia, heart attack and stroke, whereas average nightly sleep duration of 5-6 hours was associated with a greatly attenuated risk that was frequently non-significant, relative to 7 hours. Other studies have identified a similar pattern, suggesting the shortest sleepers are at the greatest risk while those in the more normative short sleep range (~6 hours) are generally not at increased risk[2, 54]. Although it is unclear why results across studies reflecting 6-hour sleepers are inconsistent in this regard, it may reflect a measurement issue (e.g., variability in sleep duration assessments) or characteristics of those in this group (e.g., more heterogeneity regarding resilience to sleep loss).

Long sleep was not found to be associated with risk factors, especially after adjustment. Although several studies have identified risks associated with long sleep duration[61], the present study did not detect any such risks. Previous literature on long sleep is highly variable, perhaps since self-reported long sleep is frequently limited to the unemployed, ill, or retired [44, 62], and may reflect long time in bed rather than long time asleep.

Sleep Duration and Hypertension

The present findings regarding hypertension are consistent with previous literature demonstrating elevated risk in short sleepers, especially when measured by self-report. For example, several previous studies have shown that, when hypertension is measured by self-report, prevalence is higher in the context of short sleep duration[1, 2, 63-67] and incidence may also be higher[14]. However, the present findings also indicate associations between short sleep and objectively determined hypertension were largely accounted for by covariates. This finding is in contrast to several previous studies that have found that short sleep is associated with objectively-measured hypertension[68] and a recent meta-analysis demonstrating this effect across studies with short sleep (RR=1.21 [95%CI 1.05, 1.40]) [68]. This inconsistency may be due to several of the following factors: (1) The definition of objective hypertension includes current or past treatment with antihypertensive medication, which may introduce imprecision in the determination; (2) Antihypertensive medications may themselves alter sleep, though this would probably be biased towards short sleep[69]; (3) Hypertension is widely prevalent, such that a population-based sample of hypertensive subjects may represent a very heterogeneous group, thus limiting precision of estimates. It is possible that these and other factors may potentially explain why laboratory studies have found inconsistent relationships between measured blood pressure and sleep duration[68].

Sleep Duration and Hyperlipidemia

Few studies have assessed associations between short sleep duration and hyperlipidemia and/or hypercholesterolemia. One previous study found that short sleep in adolescents was associated with high cholesterol[70], and a recent study found self-reported hypercholesterolemia in the general population to be associated with short sleep duration

and self-reported insufficient sleep[2]. Alternatively, other research has found that long sleep duration was associated with high cholesterol among the elderly[71]. The present study did find a relationship between short sleep duration and hyperlipidemia, however, only in the very short sleepers. This is consistent with a previous finding from this cohort that elevated c-reactive protein (also a risk factor for atherosclerosis) is elevated in the shortest sleep duration category, but not among less extreme short sleepers[54].

Sleep Duration and Diabetes

The literature regarding the relationship between habitual sleep duration and diabetes is highly variable[3, 72, 73]. The present study found that only very short sleep associated with elevated diabetes risk when measured using the standard survey question. When an objective determination was used, elevations were not found. This suggests that the inconsistencies in the literature are likely partially due to weak associations in the presence of confounders and measurement variability.

Sleep Duration and Obesity

Findings for obesity reflected the results of previously-published studies.[74] Short sleep duration is associated with obesity. Interestingly, most self-reported obesity associations were significant only before including confounders into the analysis, whereas findings regarding objectively-determined obesity were more consistent. This may be reflected in improved reliability of measurement.

Self-Reported Versus Objective Determinations

Across domains, associations were generally stronger for self-reported determinations of risk factors, compared to objective determinations. In some cases, only the association with self report was significant (and this pattern was reversed in obesity). This is in contrast to a recent meta-analysis, which found that associations between short sleep and incident hypertension were stronger if hypertension was determined objectively[68]. This analysis was cross-sectional, which may explain the difference. Perhaps self-reported sleep and self-reported health variables correlate due to other confounders, such as social desirability biases, state experiences of stress, etc. For example, a previous study found that self-reported sleep quality correlated with standard measures, but tended to correlate better with depression ratings[75]. Also, the time window of self reports (complete history) and objective determinations (which may reflect more recent or state-dependent factors) may play a role, and recall biases may differentially affect these determinations. Finally, perhaps measurement error within the nonstandard sleep duration item is partially responsible.

Sleep Duration Categories

Another pattern that emerges from this study and other recent studies[e.g., 2, 47, 48, 76-78] is that the phenomenon of short sleep duration as measured using population surveys represents at least two subgroups – “very short sleep” of <5 hours, which is more strongly associated with adverse outcomes but represents only a small fraction of the population and “short sleep” of 5-6 hours, which accounts for the majority of short sleepers but is generally less strongly associated with adverse outcomes. This may be due to reporting characteristics, since the “short sleep” group may be more heterogeneous and may be more likely to include more individuals who are relatively unimpaired due to being a “natural short sleeper” who requires less sleep[17, 79] or may be more resilient to sleep loss[80-83], in addition to those who are obtaining insufficient sleep. This underscores the need for further research identifying and classifying subgroups of short sleepers who may be at increased (or decreased) risk of adverse outcomes.

Interactions by Race/Ethnicity

Few studies have addressed whether the relationship between sleep duration and cardiometabolic disease depends on race. One such study found sleep duration mediated racial differences in hypertension[49], while other research indicates the relationship of sleep duration to baseline C-reactive protein levels depends on race/ethnicity, with different patterns reflected throughout different groups.[54] The results of the present study extend the later findings; for all outcomes except objective diabetes, the relationship between sleep duration and cardiometabolic disease differed by race/ethnicity. Frequently, these differences were subtle, but they may identify unique patterns of risk that would otherwise be overlooked. For example, blacks/African-Americans are more likely to be short sleepers in population-based studies that used subjective self-report[77, 84], as well as regional studies that utilized actigraphy[85], and the present study shows the relationship between short sleep duration and obesity is stronger in this group.

Other interesting patterns emerged in these analyses. For example, the relationship between very short sleep and self-reported hypertension was seen for all groups except Mexican-Americans and Asians/Others, with the strongest effect seen among Other Hispanics/Latinos. Interestingly, the relationship between objective hypertension and very short sleep was only seen among other Hispanics/Latinos. Not only does this suggest a unique relationship among non-Mexican Hispanics/Latinos, but it demonstrates the heterogeneity among Hispanics/Latinos, with no effect seen in the largest subgroup, (Mexican-Americans). There is currently very little known about sleep and health in the US Hispanic/Latino population[59] but other NHANES data suggests that non-Mexican Hispanics/Latinos are at increased risk for a number of sleep complaints, which are not more common among Mexican-Americans[77, 78].

Regarding hyperlipidemia, the association with very short sleep was most evident among non-Hispanic whites and Asians/Others. Other minority groups did not show an association (except that Mexican-American long sleepers had less self-reported, but not objective, hyperlipidemia). One previous study showed an association between very short sleep duration and hyperlipidemia in a population sample[2], though this study also showed that the association is largely accounted for by perceived insufficiency of sleep, rather than sleep duration per se. So, this relationship may be partially explained by different levels of perceived insufficient sleep. In the current NHANES sample, non-restorative sleep (which may be an overlapping construct with perceived sleep insufficiency, is highest among non-Hispanic whites and Asians/others[78].

Regarding diabetes, associations with very short sleep were seen most clearly among non-Hispanic Whites, and not other groups. This may explain relatively weak effects in other large population studies[1] and stronger effects in samples with more whites[86], though this conflicts with other reports showing a stronger relationship in black Americans[11].

It is also important to note that race/ethnicity can be recognized as a risk factor for cardiometabolic disease[87]. For example, increased obesity in racial minorities is well-described[88], as is increased prevalence of hypertension in African-Americans[89-92] and increased levels in Hispanics/Latinos of diabetes[93]. Immigrant status may also be an important factor in the relationship among sleep, race/ethnicity and cardiometabolic health. Although this analysis is outside of the scope of the present study, an analysis of this dataset shows that immigrants generally reported healthier sleep than US-born respondents[77, 78]. In addition, though this analysis focused on race/ethnicity, other sociodemographic factors may be relevant as well. For example, prior reports have found gender differences in the relationship between sleep and cardiometabolic disease risk factors, generally with women demonstrating a stronger relationship[48, 94, 95]. In the present study, women were less

likely to be short or long sleepers, though they may be more susceptible to the associated adverse health outcomes.

Potential Causal Pathways Linking Sleep Duration to Cardiometabolic Disease

Although a full accounting of potential mechanisms linking short sleep duration and cardiometabolic disease is beyond the scope of this paper, it is important to note several possibilities. Sleep loss is associated with a pro-inflammatory state [96], with noted elevations in a number of pro-inflammatory biomarkers such as interleukin-6 [97-103] and tumor necrosis factor [36, 97, 98, 100, 104-106]. Increased inflammation may be implicated in a number of cardiovascular disease processes, including development of atherosclerotic plaques and end stage thrombotic complications [107]. Inflammation may play a causal role in insulin sensitivity and obesity as well [108]. In addition, sleep loss may produce alterations in hypothalamic-pituitary-adrenal axis activity [109-112], which plays an important role linking stress and cardiovascular disease [113]. Other potential pathways may involve insulin dysregulation [33, 34, 114-117], decreased leptin (a hormone secreted by adipocytes that contributes to satiety) and increased ghrelin (a hormone secreted by the stomach to stimulate hunger) [118, 119]. Several behavioral factors may also play a role in the relationship between sleep duration and cardiometabolic disease. For example, sleep duration may be related to unhealthy dietary patterns [47, 120], decreased physical activity [121-123], and other health risk factors.

Study Limitations

The present study is limited to a cross-sectional analysis on prevalent subclinical disease and cannot make inferences of causality. Since NHANES limits self-reports of sleep duration to integer values, which likely reflect usual habits in recent times, some misclassification bias may have occurred, which would have mitigated the strength of the associations that were found in the present analysis. A better approach would have been to prospectively assess sleep duration using a daily diary [124, 125] and an optimal approach would have also included concurrent actigraphy [38, 49, 126, 127]. Despite this limitation, sleep duration has been shown to correlate reasonably well with objectively measured sleep ($r=0.47$) [38].

Another limitation of the study oddly enough derives from one of its primary strengths: sample size. While the present study had sufficient sample size at the extremes of sleep duration to detect the observed effects, individuals at these extremes represent a small portion of the population. This suggests that although these relationships are likely to exist within the population, they are most relevant for only a small subset of the general population. Our distribution of sleep duration values is consistent with that reported by other cohorts. For example, the Nurses' Health Study [128] reported 5% short sleepers and 5% long sleepers and the MrOS Study reported 8% long sleepers [44].

Finally, while many studies have shown BMI to be a useful measure of central obesity and cardiovascular risk, BMI does not account for muscle mass, gender- or age-related differences in body fat distribution, or differences in relationships between adiposity and cardiometabolic disease among ethnorracial subgroups. Alternatives to BMI include waist-to-hip ratio, neck circumference and the index of body adiposity ($[\text{Hip}/\text{Height}]^{1.5}$) [129, 130], which may better approximate central adiposity and associated cardiometabolic disease. These data were not collected for NHANES, and nor were data regarding menopausal status, menstrual cycle phase or use of hormone replacement therapy. Therefore, residual confounding may exist. However, this would likely attenuate the association between sleep duration and cardiometabolic disease towards the null, indicating the present findings likely represent an underestimation of the true association. Future studies should also include the addition of a social desirability scale to assess for trends in

biased reporting, and to further address the issues relating to a discrepancy between self-reported and objective assessments.

Conclusions

Short sleep duration (and very short sleep duration in particular) is associated with adverse cardiometabolic outcomes. This relationship persists with outcomes measured either self-reported or objectively, and persists even after adjustment for a wide array of covariates. Long sleep duration showed less robust association with these outcomes. Further, these relationships depended on race/ethnicity. Future studies should use more precise measures of sleep duration, consider the role of sociodemographic factors, and include a longitudinal design to assess outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Characteristics of the Sample, Stratified by Sleep Duration Category

Variable	Category	Sleep Duration Categories					χ^2 p-value
		Overall Sample	Very Short Sleep (<5 Hours)	Short Sleep (5-6 Hours)	Normal Sleep (7-8 Hours)	Long Sleep (9 Hours)	
N		100%	6.16%	33.78%	52.68%	7.38%	
Age	18-24	11.04%	10.28%	6.93%	10.89%	21.45%	<.0001
	25-44	36.98%	37.84%	38.18%	37.28%	27.35%	
	45-64	36.04%	35.88%	41.77%	38.10%	22.50%	
	65-79	12.02%	12.30%	9.93%	10.56%	18.63%	
	80+	3.93%	3.72%	3.20%	3.17%	10.07%	
Sex	Female	48.37%	48.56%	44.59%	50.66%	38.30%	0.0029
Race/Ethnicity	Non-Hispanic White	69.75%	72.69%	57.70%	66.08%	73.00%	<.0001
	Black/African-American	11.17%	8.16%	22.27%	14.64%	10.22%	
	Mexican American	8.22%	9.26%	4.40%	7.07%	8.31%	
	Other Hispanic/Latino	4.95%	4.699%	6.53%	5.07%	5.20%	
	Asian/Other	5.92%	5.198%	9.11%	7.14%	3.28%	
Education	Less Than High School	7.12%	7.29%	10.41%	5.52%	11.01%	<.0001
	Some High School	13.38%	11.69%	20.26%	14.41%	16.86%	
	High School Graduate	25.93%	24.45%	29.24%	27.79%	26.37%	
	Some College	28.57%	27.46%	30.46%	30.92%	24.67%	
	College Graduate	25.01%	29.10%	9.63%	21.37%	21.10%	
Insurance	Uninsured	19.63%	18.71%	22.20%	20.63%	20.24%	<.0001
	Public Insurance	15.56%	13.65%	30.18%	14.87%	23.50%	
	Private Insurance	64.81%	67.64%	47.62%	64.50%	56.26%	
Smoking	Yes	23.63%	20.83%	39.82%	25.21%	26.31%	<.0001
Acculturation	English Only	84.36%	83.50%	87.18%	85.40%	84.09%	0.2364
	English & Spanish	6.17%	6.55%	5.69%	5.51%	6.60%	
	Spanish Only	4.93%	5.42%	2.61%	4.31%	5.83%	
	Other	4.55%	4.54%	4.52%	4.77%	3.48%	
Food Security	Full	80.98%	83.59%	64.30%	79.58%	79.32%	<.0001
	Marginal	7.43%	6.92%	14.36%	7.20%	7.29%	
	Low	7.92%	6.81%	12.46%	8.73%	9.50%	
	Very Low	3.68%	2.69%	8.88%	4.49%	3.88%	
Caffeine Use	Yes	89.33%	90.24%	88.49%	88.54%	86.41%	0.1363
Own Home	No	29.71%	27.82%	37.80%	30.84%	33.48%	0.0042

Variable	Category	Sleep Duration Categories					χ^2 p-value
		Overall Sample	Very Short Sleep (<5 Hours)	Short Sleep (5-6 Hours)	Normal Sleep (7-8 Hours)	Long Sleep (≥ 9 Hours)	
Hypertension	Subjective	29.72%	27.25%	45.09%	30.93%	32.17%	<.0001
	Objective	37.85%	35.85%	45.57%	39.40%	40.68%	0.0109
Hyperlipidemia	Subjective	29.66%	28.88%	40.22%	28.94%	31.41%	0.0079
	Objective	33.20%	32.29%	41.47%	32.43%	38.11%	0.0187
Diabetes	Subjective	9.66%	8.70%	16.87%	9.77%	11.48%	0.0005
	Objective	27.51%	25.63%	32.48%	28.98%	31.94%	0.0148
Obesity	Subjective	30.09%	28.14%	37.91%	33.32%	24.14%	0.0001
	Objective	33.37%	31.52%	44.21%	35.88%	27.61%	0.0001

Table 2

Unadjusted and Adjusted Odds Ratios (OR) and 95% Confidence Intervals (95% CI) of Associations between Sleep Duration and Cardiometabolic Disease Outcomes

	Very Short Sleep	OR (95% CI)	p	Short Sleep	OR (95% CI)	p	Long Sleep	OR (95% CI)	p
Unadjusted									
Hypertension (Self-Reported)	2.19	(1.65, 2.92)	<.0001	1.20	(1.02, 1.40)	0.0257	1.27	(0.97, 1.66)	0.0865
Hypertension (Objective)	1.50	(1.13, 1.99)	0.0049	1.16	(1.00, 1.35)	0.0452	1.23	(0.95, 1.59)	0.1176
Hyperlipidemia (Self-Reported)	1.66	(1.23, 2.22)	0.0008	1.00	(0.85, 1.18)	0.9700	1.13	(0.85, 1.49)	0.3986
Hyperlipidemia (Objective)	1.49	(1.11, 1.98)	0.0074	1.01	(0.86, 1.17)	0.9372	1.29	(0.99, 1.69)	0.0631
Diabetes (Self-Reported)	2.13	(1.44, 3.14)	0.0001	1.14	(0.91, 1.43)	0.2680	1.36	(0.95, 1.94)	0.0891
Diabetes (Objective)	1.78	(1.17, 2.70)	0.0072	1.16	(0.92, 1.46)	0.2052	1.46	(1.01, 2.11)	0.0431
Obesity (Self-Reported)	1.56	(1.17, 2.08)	0.0025	1.28	(1.09, 1.49)	0.0022	0.81	(0.61, 1.09)	0.1615
Obesity (Objective)	1.72	(1.29, 2.29)	0.0002	1.22	(1.04, 1.42)	0.0123	0.83	(0.63, 1.09)	0.1862
Adjusted*									
Hypertension (Self-Reported)	2.02	(1.45, 2.81)	<.00001	1.22	(1.02, 1.45)	0.0326	1.17	(0.85, 1.61)	0.3270
Hypertension (Objective)	1.34	(0.96, 1.87)	0.0854	1.19	(1.00, 1.43)	0.0536	1.11	(0.80, 1.53)	0.5387
Hyperlipidemia (Self-Reported)	1.96	(1.43, 2.69)	<.00001	1.06	(0.89, 1.27)	0.5210	1.26	(0.92, 1.72)	0.1539
Hyperlipidemia (Objective)	1.41	(1.04, 1.91)	0.0276	0.99	(0.84, 1.17)	0.9037	1.29	(0.95, 1.74)	0.1003
Diabetes (Self-Reported)	1.76	(1.13, 2.74)	0.0124	1.07	(0.84, 1.37)	0.5652	1.19	(0.80, 1.76)	0.3968
Diabetes (Objective)	1.50	(0.94, 2.39)	0.0907	1.10	(0.86, 1.41)	0.4345	1.31	(0.87, 1.97)	0.2019
Obesity (Self-Reported)	1.29	(0.95, 1.75)	0.0988	1.21	(1.03, 1.43)	0.0226	0.86	(0.63, 1.16)	0.3203
Obesity (Objective)	1.53	(1.13, 2.06)	0.0053	1.17	(1.00, 1.38)	0.0496	0.87	(0.65, 1.16)	0.3514

* Adjusted analyses include age, sex, race/ethnicity, acculturation, education, insurance, home ownership, food security, smoking, and caffeine