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Individual Differences in Habitual Short Sleep Duration and Dysfunction: Subjective Health Versus Objective Cardiovascular Disease Risk

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Objective: The adverse health effects of short sleep duration (i.e., six or fewer hours per night) are well established, including an increased risk of cardiovascular disease (CVD) and related mortality. However, there is heterogeneity in perceived sleep need among habitual short sleepers (HSS), with a sizable minority reporting no sleep-related daytime dysfunction. It has not been determined whether health risk associated with short sleep duration is consistent across individuals with and without reported dysfunction. The current study examined self-rated health (SRH), previously demonstrated to predict CVD risk, and objective CVD risk among HSS with and without reported dysfunction in the National Health and Nutrition Examination Surveys (NHANES). *Method*: Participants were adults age 40–79 in the 2005–2006 and 2007–2008 NHANES cycles. Assessments included the single item SRH (*poor* to *excellent*), self-reported average sleep duration, and self-reported daytime sleep-related dysfunction. Ten-year atherosclerotic CVD and high lifetime CVD risk (≥39%) were calculated using previously validated algorithms. *Results*: HSS with no reported dysfunction rated their overall health significantly better than those with reported dysfunction; however, the "no dysfunction" HSS group evidenced modestly, though significantly, higher 10-year CVD risk compared with their dysfunction-reporting counterparts. High lifetime CVD risk, including younger adults age 20–39, was slightly higher for persons not reporting dysfunction, with the exception of short sleepers at the highest level of dysfunction who had the highest prevalence of high lifetime risk. *Conclusions*: Findings suggest that the absence of perceived sleep-related dysfunction does not confer lower CVD risk, despite higher SRH.

Keywords: habitual short sleep, daytime dysfunction, cardiovascular disease risk, self-rated health, NHANES

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The health risks of short sleep duration are well established. Epidemiological research has demonstrated that getting less than the recommended 7–9 hr of sleep each night (Watson et al., 2015) is associated with a long and growing list of adverse health outcomes, most notably cardiovascular disease (CVD) risk and CVD-related mortality (e.g., Covassin & Singh, 2016). Approximately 30% of adults report routinely getting six or fewer hours of sleep in a 24-hr period (Luckhaupt et al., 2010) consistent with the descriptor "habitual short sleeper" (HSS; Grandner et al., 2010). Consequently, habitual short sleep is a major public health concern. For most of these individuals, short sleep has the predicted association with perceived daytime dysfunction (i.e., fatigue, difficulty staying alert). Yet, there is a subset of short sleeping adults —an estimated 10% (Curtis et al., 2016)—who report *no* dysfunction. There is initial evidence that short sleepers without reported dysfunction may not be accurate in their subjective perceptions. For example, these "no dysfunction" short sleepers evidence objective cognitive impulsivity at the same level as other short sleepers (Curtis et al., 2018). These same short sleepers also show brain activity patterns consistent with sleep onset in resting functional magnetic resonance imaging (fMRI) assessment, suggesting that they have difficulty maintaining alertness under low environmental stimulation (Curtis et al., 2016).

Critical questions include whether short sleepers who do not report daytime dysfunction face the same objective health risks as other short sleepers and whether they differ in their self-rated

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Steven D. Barger served as lead for data curation and formal analysis, contributed equally to writing – original draft and served in a supporting role for conceptualization. Brian J. Curtis contributed equally to writing – review and editing and served in a supporting role for funding acquisition.

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J_ID: ART NO: 10.1037/hea0001085 Date: 27-May-21 Page: 2 Total Pages: 11 4/Color Figure(s) ARTTYPE="ResearchArticle"

health (SRH). The purpose of the current study was to examine both subjective (SRH) and objective (10-year and lifetime CVD risk) health of short sleepers with and without reported dysfunction in a large, nationally representative sample (National Health and Nutrition Surveys [NHANES]). Additionally, other sleep parameters, as well as demographic characteristics were examined to provide further phenotyping information on HSS with no reported daytime dysfunction.

Habitual Short Sleep Duration and CVD Risk

The association between short sleep duration and CVD risk is well-established. Multiple reviews and meta-analyses find increased risk of CVD (e.g., Covassin & Singh, 2016; Yin et al., 2017); though see Kwok et al., 2018, for contradictory evidence). Short sleep duration is also associated with risk of incident cardiovascular events (Daghlas et al., 2019; Wang et al., 2020) and prevalent hypertension (Grandner et al., 2018).

Relevant to the current study, prior research has established associations between habitual short sleep and CVD in NHANES. Ford (2014) found that deviations from 7 hr/night (i.e., both fewer and more hours/night) were associated with greater 10-year predicted CVD risk. Grandner and colleagues (2014) reported evidence that very short habitual sleep (less than 5 hr) was associated with self-reported hypertension, self-reported and objective hyperlipidemia, self-reported diabetes, and objective obesity; 5–6 hr/night was associated with self-reported hypertension and objective obesity. Reported sleep duration of 5 hr or fewer has also been associated with risk of hypertension over an 8-year follow-up in NHANES (Gangwisch et al., 2006), as well as decreased odds of ideal cardiovascular health using American Heart Association metrics (Cash et al., 2020). In addition, short sleep duration ($\leq$7 hr/night) has been associated with greater prevalence of stroke and hyperlipidemia in comparison with recommended sleep duration (7–9 hr/night; Krittanawong et al., 2020). In summary, the majority of prior studies find elevated CVD risk among persons with short sleep duration, both in terms of individual CVD risk factors and in terms of composite 10-year estimates for hard CVD events. However, no prior studies have evaluated reported sleep dysfunction in the context of CVD risk nor have they evaluated longer CVD risk horizons, that is, lifetime CVD risk. Evaluation of lifetime CVD risk is particularly important given that 10-year risk estimates underestimate lifetime CVD risk, particularly among women and younger men (Marma et al., 2010).

Habitual Short Sleep Duration and SRH

SRH, in the form of a general health rating from "poor" to "excellent," is a key subjective health variable, predicting all-cause mortality (e.g., Benyamini & Idler, 1999), as well as cardiovascular mortality specifically (Barger et al., 2016; Mavaddat et al., 2014). SRH has robust associations with short sleep duration. Prior research has demonstrated that reports of short sleep duration are associated with poorer SRH, including groups such as workers over age 55 (Coombe et al., 2019) and young adults (Štefan et al., 2017). Pertinent to the current study, sleep duration has been associated with SRH in NHANES. Individuals rating their general health as poor report an average of 45 min less sleep per night than those rating their health as excellent (Cepeda et al., 2016). It has not been determined whether this association is moderated by perceived dysfunction among short sleepers. Although it would be expected that individuals who routinely get less than the recommended amount of sleep would report lower SRH, this may not apply to individuals who experience less dysfunction and, by extension, less need for the sleep.

The Current Study

The current study utilized NHANES to examine SRH, 10-year CVD risk, and lifetime CVD risk among habitual short sleepers (HSS) with and without reported sleep-related daytime dysfunction. Although 10-year CVD risk scores predict hard clinical endpoints and capture synergistic risk due to comorbidity, age, and sex are heavily weighted in the risk algorithm (Marma et al., 2010) and modest age imbalances across sleep groups could generate appreciable 10-year risk differences not attributable to sleep per se We address this by controlling for age and sex in 10-year risk analyses and by examining lifetime CVD risk. Lifetime CVD risk estimates are insensitive to age and sex (Lloyd-Jones et al., 2006) and provide consistent risk estimates across racial groups and age cohorts (Berry et al., 2012). Over 50% of the adult U.S. population has low 10-year CVD risk but high lifetime CVD risk (Marma et al., 2010) and lifetime risk estimates provide a robust and as yet unexamined complement to 10-year risk scores. We hypothesized that HSS with no reported dysfunction would have higher SRH compared with their dysfunction-reporting counterparts, but would evidence comparable CVD risk.

Method

Overview

We first estimate the population prevalence of sleep duration, sleep dysfunction, and combinations of sleep duration and dysfunction among respondents aged 16 years and over. We then compare our three outcome measures, SRH, 10-year atherosclerotic CVD (ASCVD) risk and lifetime CVD risk, across sleep types. Because 10-year ASCVD risk calculations are valid for adults 40- to 79-years-old free of diagnosed CVD (Goff et al., 2014), comparisons of SRH and ASCVD risk are restricted to this subgroup (N = 4,654). SRH data are based upon a somewhat smaller sample size of 4,475. Lifetime CVD risk can be validly estimated among persons aged 20–79 so in addition to the 10-year ASCVD subgroup, we provide complementary lifetime CVD risk analysis including those 20–39 years of age (N = 8,289).

Lifetime risk is classified in five graded categories using established risk factors (smoking, blood pressure, cholesterol, or diabetes). Marma et al. (2010) create low (optimal, not-optimal) and high (elevated, one major, or two or more major risk factors) lifetime risk categories based upon these strata. Low and high correspond to absolute lifetime risks of $\leq$39% versus $\geq$39% and are based upon a natural CVD risk separation observed between those categories in the Framingham cohorts (Lloyd-Jones et al., 2006) and in a set of pooled cohorts comprising over a quarter million people (Berry et al., 2012).

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J_ID: ART NO: 10.1037/hea0001085 Date: 27-May-21 Page: 3 Total Pages: 11 4/Color Figure(s) ARTTYPE="ResearchArticle"

Data Source

We analyzed the 2005–2006 and 2007–2008 cycles of the NHANES to capture assessments of both sleep duration and dysfunction (see below). NHANES includes an in-person interview and a physical examination. All participants provide informed consent (or assent if minors). The data collection was approved by the National Center for Health Statistics (NCHS) Ethical Review Board (https://www.cdc.gov/nchs/nhanes/irba98.htm).

All analyses incorporated the complex survey design. Four-year weights were calculated to represent the civilian, noninstitutionalized U.S. population. Prevalence analyses incorporated interview weights whereas SRH and CVD risk analyses incorporated Mobile Examination Center weights (Johnson et al., 2013). We used Stata MP 16.1 for analysis (Stata Corp, College Station, TX).

Sleep Assessments

Sleep duration was assessed by asking "How much sleep do you usually get at night on weekdays or workdays?" Hours of sleep in whole numbers were recorded. A small number of values (, 1% of the sample) were top coded at 12 hr of sleep. Sleep duration was categorized as short sleeper (six or fewer hours hours/night) and medium-length (or "conventional") sleeper (7–9 hr). Individuals reporting - 9 hr of sleep per night are included in the prevalence estimates but are excluded from the remaining analyses.

Sleep-related daytime dysfunction was assessed with three items: "In the past month how often did you feel unrested during the day, no matter how many hours of sleep you have had?" "In the past month how often did you feel excessively or overly sleepy during the day?" and "In the past month how often did you not get enough sleep?" Responses ranged from *never* to *almost always*. Dysfunction was coded as any response other than *never*, consistent with prior reports using daytime dysfunction items from the Pittsburgh Sleep Quality Index (Curtis et al., 2016, 2018).

To establish convergent validity for the sleep dysfunction group classification, we examined items from the Functional Outcomes of Sleep Questionnaire (Weaver et al., 1997). The eight items in NHANES are in the following form: "Difficulty__ when tired?" (e.g., *concentrating*) on a 4-point ranging from *no difficulty* to *extreme difficulty*. To further contextualize HSS groups, we examined self-reported sleep latency (minutes to fall asleep), nighttime awakenings, early morning awakening, and frequency of sleep medication use (all ratings on 5-point Likert scale, *never* to *almost always*).

Self-Rated Health

SRH was assessed in the Mobile Examination Center using a computer assisted personal interviewing system (Centers for Disease Control and Prevention, 2008, 2009b). Participants were asked, "Would you say your health in general is *excellent*, *very good*, *good*, *fair* or *poor*?" and we scored these responses from highest (*excellent* = 5) to lowest (*poor* = 1).

Cardiovascular Risk Assessment

Age, sex, race/ethnicity, smoking status, and antihypertensive medication use were participant reported. Lipids (high density and total cholesterol) and hemoglobin A1c (HbA1c) were assessed by blood draw. Physician blood pressure (BP) examiners took three consecutive BP readings after participants sat quietly for 5 min. Average systolic and diastolic BP were calculated from these readings (Centers for Disease Control and Prevention, 2007, 2009a). Diabetes mellitus was defined as HbA1c $6.5% or reported physician diagnosis of diabetes, current use of insulin or oral hypoglycemic medications (Expert Committee on the Diagnosis and Classification of Diabetes Mellitus, 2000; Rethy et al., 2020)

We calculated 10-year ASCVD using an age, sex, and race-specific algorithm. In addition to age, sex, and race, this algorithm incorporates systolic BP, total and HDL cholesterol, diabetes, smoking, and antihypertensive medication use. Further details on scoring are in Goff et al. (2014) and on coding are in Barger et al. (2017). This score provides the percentage risk for any of the following: fatal coronary heart disease, fatal stroke, nonfatal stroke, or nonfatal myocardial infarction. As per scoring guidelines, we restricted our calculations to participants 40- to 79-years-old who were free of diagnosed ASCVD at baseline. We also truncated risk probabilities at 1% and 30% (Goff et al., 2014).

Lifetime risk was scored as in Berry et al. (2012). *Optimal risk* is defined by unmedicated systolic and diastolic values less than 120 and 80 mmHg, respectively; unmedicated total cholesterol , 180 mg/dL; being a nonsmoker and free of diabetes. *Not-optimal risk* is the same as optimal except for higher BP or total cholesterol, that is, systolic values of 120–139 mmHg or diastolic values 80–89 mmHg; or total cholesterol 180–199 mg/dL. *Elevated risk* is defined by yet higher BP (140–159 mmHg or 90–99 mmHg) or cholesterol (200–239 mg/dL). The remaining two risk categories are defined by the presence of major CVD risk factors, either *one major* or *two or more major* risk factors. Major risk factors are defined by high BP (- = 160 mmHg systolic or - =100 mmHg diastolic; or antihypertensive medication use), high total cholesterol (- = 240 mg/dL; or taking lipid lowering medications), being a current smoker or having diabetes.[1] Thus, a smoker with BP of 140/91 mmHg and total cholesterol of 201 mg/dL without diabetes or medications would be classified as having one major risk factor. Because lifetime risk scores are applicable for persons aged 20–79 years we provide complementary lifetime CVD risk analysis including persons 20–39 years of age, who were combined with the 40- to 79-year-old group for these analyses (N = 8,289). Existing CVD exclusions were determined by any affirmative response to questions about previously diagnosed stroke, heart attack, coronary heart disease, or congestive heart failure.

[1] We note it seems counterintuitive that persons with "elevated risk" based on untreated blood pressure (BP; 140–159 mm/Hg systolic/90–99 mm/Hg diastolic) would be considered lower risk compared with those with treated blood pressure in the optimal range (, 120 mm/Hg systolic/ , 80 mm/Hg diastolic). However, the extent of this apparent paradox is dependent upon several factors including (1) the number of persons defined as elevated risk solely because of BP; (2) the number of persons with treated blood pressure values lower than the subgroup in #1; and (3) the untreated BP of persons currently using antihypertensive medication, which is unknown. If all treated blood pressures are below the elevated risk category then it would seem paradoxical to assign them to a higher risk category, particularly if all of them were in the "optimal" rather than "not optimal" categories. However, if all or most treated BP values are in the elevated risk range or higher, then there would be no paradox as their untreated BP is presumably much higher. Given than rV75% of persons with hypertension are not controlling their blood pressure (https://www.cdc.gov/bloodpressure/facts.htm) we suspect the number of persons with disparate lifetime risk categories based solely on BP patterns as described above are rare and would not materially alter lifetime risk estimates.

J_ID: ART NO: 10.1037/hea0001085 Date: 27-May-21 Page: 4 Total Pages: 11 4/Color Figure(s) ARTTYPE="ResearchArticle"

Covariates

We also conducted sensitivity analyses controlling for education, as well as self-reports of cancer, sleeping pill use, alcohol use, and major depression. We coded cancer as present if the respondent reported any cancer history, excluding nonmelanoma skin cancer. Sleeping pill use was any reported use over the last month. Alcohol use was scored as current drinkers who binge drink ($5 drinks in a day in the previous year) versus abstainers, ex-drinkers and current drinkers who did not binge (Vaeth et al., 2014). Persons missing alcohol data were assigned to the nonbinge category. Sex-specific definitions of binge drinking (i.e., $4 drinks for women) were not adopted until the 2011–2012 NHANES cycle. Persons were classified as depressed if they scored $10 on the Patient Health Questionnaire-9 (Kroenke et al., 2001). Missing PHQ data was modeled as a covariate.

Predictions and Analysis

Self-Rated Health and 10-Year ASCVD Risk

We expected that HSS reporting daytime dysfunction would have poorer SRH than those not reporting dysfunction. In contrast, we expected comparable 10-year risk among short sleepers who did and did not report dysfunction. Finally, we expected an association of sleep duration with 10-year risk wherein short sleepers would have higher 10-year risk compared with conventional sleepers, irrespective of reported dysfunction.

For the first two predictions, we regressed each outcome on an indicator variable reflecting whether or not short sleepers reported dysfunction. To evaluate higher-order patterns that could modify interpretation of the planned contrasts we conducted follow-up analyses using a model including terms for sleep duration (0–6 vs. 7–9 hr), reported dysfunction (none vs. any), and the interaction of duration and dysfunction. We used the sleep duration coefficient in this larger model to evaluate the association of sleep duration with 10-year ASCVD risk.

We used a survey based least squares regression as the large sample size ensures unbiased estimates (Lumley et al., 2002). In addition to these planned pairwise contrasts, we also evaluated in an exploratory fashion pairwise differences for sleep duration and sleep dysfunction.

Lifetime CVD Risk

We regressed the binary lifetime risk variable on sleep duration (0–6 vs. 7–9 hr), reported dysfunction (none vs. any), and the interaction of duration and dysfunction. For this analysis we used a survey-based generalized linear model (GLM) with a Poisson distribution and a log link with robust standard errors (Zou, 2004). This model produces incidence rate ratios (IRR), which represent the incidence of the exposed group divided by the unexposed group, rather than a ratio of odds, which do not provide consistent estimates of the rate ratio (Thompson et al., 1998; Zou, 2004). Sensitivity analyses using all five lifetime risk categories are provided in the online supplemental materials.

Supplemental Analysis of Levels of Sleep-Related Dysfunction

Although the main focus of the study was on HSS-NRD compared with all other short sleepers reporting some degree of dysfunction, we also examine a graded dysfunction variable. The graded dysfunction variable was based on the mean of the four dysfunction questions. Dysfunction means could range from 0–4 but high mean values for dysfunction (above 3) had small cell sizes. We collapsed mean dysfunction into a four-level variable comprised of *no dysfunction* (0), *some dysfunction* (1), *moderate dysfunction* (2), and *high dysfunction* (3). Indicator variables were used with the *no dysfunction* group as the referent.

Results

Sleep Duration and Sleep Dysfunction Prevalence

The majority of the population (61%) reported sleeping 7–9 hr per night but a large minority (36%) reported sleeping six or fewer hours. This proportion of short sleepers represents 82,414,000 (95% confidence interval, CI [78,070,000, 86,860,000]) U.S. residents aged 16 and over. Parameter estimates of sleep duration by sleep dysfunction are in Table 1. Characteristics of the ASCVD analytic sample are in Table 2. The percent of the population reporting habitual short sleep duration with no reported dysfunction (HSS-NRD) was 11%—consistent with a prior report of 10% in a different nationally representative, but younger sample (the Human Connectome Project; Curtis et al., 2016). These short sleepers were more likely to be male and were slightly older compared with those reporting dysfunction, suggesting the need to consider age and sex in analyses (see Table 2).

T1 T2

Other Sleep-Related Characteristics of HSS Groups

HSS with no reported dysfunction provided ratings indicating significantly less difficulty with daytime functional outcomes compared with their dysfunction-reporting counterparts and were statistically equivalent to conventional sleepers with no reported

Table 1
Sleep Duration by Sleep Dysfunction, 2005–2008 NHANES

	Full sample(n = 12,606)		Dysfunction(n = 9,921)		No dysfunction(n = 2,685)	
Sleep duration	Percent	95% CI	Percent	95% CI	Percent	95% CI
6 hr or less	36	[34, 38]	89	[88, 90]	11	[10, 12]
7–9 hr	61	[59, 63]	81	[79, 82]	19	[18, 21]
9 or more hours	3	[2, 3]	72	[67, 76]	28	[24, 33]

Note. NHANES = National Health and Nutrition Examination Survey; CI = confidence interval. All values are weighted to represent the civilian noninstitutionalized U.S. population aged 16 years and older. Dysfunction was defined by any experience of feeling unrested, feeling overly sleepy, or not getting enough sleep.

J_ID: ART NO: 10.1037/hea0001085 Date: 27-May-21 Page: 5 Total Pages: 11 4/Color Figure(s) ARTTYPE="ResearchArticle"

Table 2
Baseline Characteristics of 2005–2008 NHANES Participants Aged 40–79 by Sleep Duration and Reported Sleep Dysfunction

Characteristic	Total		Conventional sleeper (7–9 hr per night)				Short sleeper (6 or fewer hours per night)			
			No dysfunction		Dysfunction		No dysfunction		Dysfunction	
Participants, *N*	4,654		758		2,025		276		1,595	
Age years, mean, *SE*	54.3	0.3	56.9 [a]	0.7	54.1 [b]	0.4	56.5 [a]	0.8	53.1 [c]	0.4
Female sex, % *N*	52.7	2,394	46.7 [a]	333	56.0 [b]	1,130	40.8 [ac]	102	51.5 [ad]	829
Race/ethnicity, % *N*										
Mexican American	6.0	831	10.7	200	4.6	320	9.1	53	6.2	258
Other Hispanic	3.5	377	5.7	81	2.7	140	6.5	28	3.6	128
White (non-Hispanic)	76.1	2,326	69.8	305	83.4	1,234	53.1	76	70.1	711
Black (non-Hispanic)	9.5	961	10.3	156	5.5	275	24.2	106	13.4	424
Other race	4.9	159	3.5	16	3.8	56	7.2	13	6.7	74
Education level, % *N*										
, High school	16.5	1,321	23.8	307	13.2	463	26.5	109	17.5	442
High school diploma	25.5	1,114	25.7	168	24.0	489	25.5	60	27.8	397
Some college	28.9	1,207	22.0	147	29.2	546	27.2	67	31.3	447
College graduate or higher	29.0	1,011	28.6 [a]	136	33.6 [b]	526	20.8 [ac]	40	23.3 [ad]	309
Self-rated health, % *N*										
Poor	2.2	139	0.9	12	2.2	55	0.5	3	3.0	69
Fair	12.6	874	11.0	133	10.6	324	11.7	52	16.4	365
Good	37.2	1,765	36.2	301	35.8	753	34.0	95	40.0	616
Very good	33.7	1,282	32.7	191	36.8	636	30.1	73	29.9	382
Excellent	10.7	415	15.0	89	12.0	200	16.2	35	6.3	91
Missing	3.6	179	4.1	32	2.7	57	7.5	18	4.4	72
Cardiovascular risk factors										
Mean systolic blood pressure (mm/Hg), *SE*	125.0	0.4	127.5	0.9	124.0	0.6	127.5	1.3	125.3	0.4
Currently using antihypertensive medication, % *N*	31.4	1,645	35.6	273	29.0	677	27.5	97	34.1	598
Hypertensive, % *N*	41.1	2,160	47.6	387	38.4	875	41.7	140	42.8	758
Missing	0.1	3	0.0	0	0.1	2	0.0	0	0.0	1
Mean total cholesterol, mg/dL, *SE*	206.1	0.9	207.3	2.0	206.4	1.1	209.3	3.4	204.7	1.6
Mean high density cholesterol, mg/dL, *SE*	54.1	0.4	54.1	0.9	54.8	0.5	54.0	1.4	53.1	0.6
Diabetes, % *N*	12.1	811	15.1	174	9.6	291	12.3	54	14.7	292
Current smoker, % *N*	20.6	970	19.5	145	17.7	372	27.9	67	24.5	386
Ten-year ASCVD risk, absolute % (*SD*)	7.7	6.6	10.2	8.9	7.1	5.9	10.3	8.7	7.3	6.3
High lifetime CVD risk, % N	84	3,985	88	672	82	1,708	88	243	84	1,362

Note. CVD = cardiovascular disease; ASCVD = Atherosclerotic cardiovascular disease. Hypertensive is defined as systolic blood pressure $140 mm/Hg or diastolic blood pressure $90 mm/Hg or taking antihypertensive medication. Diabetes mellitus was defined as hemoglobin A1c (HbA1c) $6.5% or reported physician diagnosis of diabetes, current use of insulin, or oral hypoglycemic medications. One value of education was missing. We evaluated differences for sex, age, and education across sleep/dysfunction groups. Different superscripts for those variables denote statistically significant pairwise differences between sleep/dysfunction groups. Percentages are weighted to represent the civilian, noninstitutionalized population.

dysfunction. With respect to other sleep assessments, HSS-NRD reported shorter sleep latency (though greater than conventional sleepers without dysfunction), less frequent nighttime or early morning awakenings, and were less likely to report taking sleep medication compared with their dysfunction reporting counterparts (see online Supplemental Materials Table 1 and Figures 1–5).

SRH by Sleep Type and Dysfunction

Consistent with predictions, short sleepers reporting dysfunction had poorer SRH than short sleepers not reporting dysfunction and this persisted after adjustment for age and sex, as well in the fully adjusted model (covarying education, depression, alcohol use, cancer, and sleep medication use; see Table 3). The age and sex-adjusted average for those reporting dysfunction was 3.2 (95% CI [3.1, 3.3]) versus 3.5 (95% CI [3.4, 3.7]) for those not reporting dysfunction. The follow up analysis showed only a significant interaction (see Table 3) wherein short sleepers reporting dysfunction had the lowest SRH and the remaining three groups were statistically equivalent, contrast $F(2, 31) = .41$, $p = .67$. Thus, only the combination of short sleep duration and dysfunction was associated with poorer SRH.

Graded sleep-related dysfunction showed a similar and linear pattern—the average for short sleepers not reporting dysfunction was 3.5 (95% CI [3.4, 3.7]) whereas the other three dysfunction levels for HSS were lower (3.4, 3.3, and 3.0, respectively). The follow-up analysis with both sleep types and graded dysfunction showed SRH was higher for those with *some dysfunction* and lower for those with *high dysfunction*. There was also an interaction where, at the *some dysfunction* level, SRH was higher for conventional versus short sleepers (see online Supplemental Materials Figure 6).

Cardiovascular Risk by Habitual Short Sleep Duration and Dysfunction

Although we predicted that the HSS groups would show equivalent 10-year CVD risk, 10-year risk was significantly higher among HSS-NRD (absolute risk 10.3%; 95% CI [9.1–11.5%]) versus short sleepers reporting dysfunction (absolute risk 7.3%; 95% CI [6.7–7.7%]), $b = -3.0$ (95% CI [−4.4, −1.5]), $t(31) = -4.36$, p , .001. This difference between short sleeper groups was no longer significantly with age and sex in the model (adjusted means

J_ID: ART NO: 10.1037/hea0001085 Date: 27-May-21 Page: 6 Total Pages: 11 4/Color Figure(s) ARTTYPE="ResearchArticle"

Table 3

Unadjusted and Adjusted Regression Models for Self-Rated Health, 10-Year Atherosclerotic Cardiovascular Disease Risk (ASCVD), and High (≥39%) Lifetime CVD Risk by Sleep Duration and Sleep Dysfunction, 2,005–2008 NHANES

	Unadjusted			Age and sex adjusted			Fully adjusted[b]		
	B	CI	*p*	*B*	CI	*p*	*B*	CI	*p*
Self-rated health by binary and graded dysfunction in short sleepers									
Dysfunction (binary 1 = yes)	20.33	[−0.5, −0.15]	.001	20.33	[−0.51, −0.15]	.001	20.27	[−0.44, −0.09]	.005
Dysfunction (graded)	20.17	[−0.23, −0.11]	.001	20.17	[−0.23, −0.11]	, .001	20.11	[−0.16, −0.05]	, .001
N	1,781			1,781			1,781		
Self-rated health by sleep duration and binary dysfunction									
Short sleeper (1 = yes)	0.02	[−0.18, 0.21]	.858	0.01	[−0.18, 0.21]	.891	0.06	[−0.13, 0.24]	.531
Dysfunction (binary 1 = yes)	−0.05	[−0.16, 0.06]	.354	−0.05	[−0.16, 0.06]	.405	−0.07	[−0.17, 0.03]	.190
Interaction	20.28	[−0.47, −0.08]	.008	20.28	[−0.48, −0.08]	.008	20.19	[−0.38, −0.01]	.041
N	4,475			4,475			4,473		
Ten-year ASCVD risk (%) by binary and graded dysfunction in short sleepers									
Dysfunction (binary 1 = yes)	22.99	[−4.39, −1.59]	, .001	−0.52	[−1.23, 0.19]	.144	22.51	[−4.02, −1.01]	.002
Dysfunction (graded)	21.03	[−1.51, −0.55]	, .001	0.06	[−0.21, 0.33]	.660	20.95	[−1.45, −0.44]	.001
N	1,871			1,871			1,871		
Ten-year ASCVD risk (%) by sleep duration and binary dysfunction									
Short sleeper (1 = yes)	0.13	[−1.64, 1.90]	.884	0.09	[−0.77, 0.95]	.833	−0.01	[−1.71, 1.70]	.995
Dysfunction (binary 1 = yes)	23.10	[−4.13, −2.08]	, .001	21.07	[−1.57, −0.57]	, .001	22.67	[−3.75, −1.58]	, .001
Interaction	0.11	[−1.70, 1.92]	.902	0.56	[−0.33, 1.44]	.207	0.08	[−1.69, 1.84]	.931
N	4,654			4,654			4,652		
Ten-year ASCVD risk (%) by sleep duration and graded dysfunction									
Short sleep	0.10	[−1.68, 1.88]	.910	0.09	[−0.77, 0.95]	.828	−0.06	[−0.86, 0.74]	.885
Dysfunction = 1	21.89	[−3.18, −0.60]	.006	21.25	[−1.83, −0.66]	, .001	20.97	[−1.64, −0.30]	.006
Dysfunction = 2	23.50	[−4.57, −2.44]	, .001	21.10	[−1.68, −0.52]	.001	20.84	[−1.47, −0.21]	.011
Dysfunction = 3	24.37	[−5.44, −3.29]	, .001	−0.59	[−1.25, 0.06]	.075	−0.47	[−1.24, 0.30]	.221
Sleep 3 D1	0.11	[−2.22, 2.44]	.924	0.42	[−0.74, 1.59]	.466	0.30	[−0.79, 1.38]	.584
Sleep 3 D2	0.56	[−1.39, 2.51]	.561	0.39	[−0.52, 1.30]	.388	0.36	[−0.59, 1.30]	.449
Sleep 3 D3	0.97	[−0.96, 2.90]	.312	0.40	[−0.73, 1.54]	.474	0.36	[−0.73, 1.44]	.506
N	4,654			4,654			4,652		
High ($39%) lifetime CVD risk[a] by binary and graded dysfunction in short sleepers ages 40–79									
Dysfunction (1 = yes)	0.96	[0.90, 1.01]	0.121	0.98	[0.93, 1.04]	0.501	0.94	[0.89, 1.00]	0.041
Dysfunction (graded)	1.00	[0.97, 1.02]	0.722	1.01	[0.98, 1.03]	0.557	0.98	[0.96, 1.01]	0.180
N	1,871			1,871			1,871		
High ($ 39%) lifetime CVD risk[a] by sleep duration and binary dysfunction ages 40–79									
Short sleeper (1 = yes)	1.01	[0.95, 1.07]	0.800	1.01	[0.95, 1.08]	0.746	1.01	[0.94, 1.07]	0.809
Dysfunction (1 = yes)	0.94	[0.89, 0.99]	0.016	0.96	[0.91, 1.01]	0.103	0.94	[0.89, 0.99]	0.016
Interaction	1.02	[0.95, 1.10]	0.546	1.03	[0.96, 1.10]	0.433	1.01	[0.94, 1.08]	0.829
N	4,654			4,654			4,652		
High ($ 39%) lifetime CVD risk[a] by sleep duration and binary dysfunction ages 20–79									
Short sleeper (1 = yes)	1.01	[0.94, 1.07]	0.869	1.02	[0.95, 1.08]	0.617	1.01	[0.94, 1.08]	0.870
Dysfunction (1 = yes)	0.92	[0.88, 0.97]	0.002	0.98	[0.94, 1.02]	0.228	0.93	[0.88, 0.98]	0.009
Interaction	1.07	[0.99, 1.16]	0.096	1.06	[0.99, 1.14]	0.114	1.05	[0.96, 1.13]	0.277
N	8,289			8,289			8,285		
High ($ 39%) lifetime CVD risk[a] by sleep duration and graded dysfunction Ages 20–79									
Sleep duration	1.01	[0.94, 1.07]	0.869	1.02	[0.95, 1.08]	0.617	1.01	[0.94, 1.08]	0.868
Dysfunction = 1	0.95	[0.90, 1.00]	0.061	0.96	[0.92, 1.01]	0.089	0.96	[0.91, 1.02]	0.180
Dysfunction = 2	0.94	[0.89, 0.99]	0.024	0.99	[0.94, 1.05]	0.770	0.94	[0.88, 1.00]	0.068
Dysfunction = 3	0.87	[0.81, 0.93]	, .001	0.97	[0.90, 1.03]	0.327	0.86	[0.80, 0.92]	, .001
Sleep 3 D1	1.04	[0.95, 1.15]	0.387	1.04	[0.95, 1.14]	0.403	1.03	[0.93, 1.13]	0.602

(*table continues*)

J_ID: ART NO: 10.1037/hea0001085 Date: 27-May-21 Page: 7 Total Pages: 11 4/Color Figure(s) ARTTYPE="ResearchArticle"

Table 3 (*continued*)

	Unadjusted			Age and sex adjusted			Fully adjusted[b]		
	B	CI	*p*	*B*	CI	*p*	*B*	CI	*p*
Sleep 3 D2	1.02	[0.93, 1.12]	0.638	0.99	[0.91, 1.08]	0.881	1.01	[0.92, 1.11]	0.832
Sleep 3 D3	1.16	[1.05, 1.29]	0.005	1.12	[1.02, 1.23]	0.024	1.14	[1.03, 1.26]	0.014
N	8,289			8,289			8,285		

Note. NHANES = National Health and Nutrition Examination Survey.
[a] Incidence rate ratios (exponentiated coefficients). Significant regression coefficients appear in bold. [b] Covariates = age, sex, education, depression, alcohol use, cancer diagnosis, sleep medication use. Self-rated health categories are *poor* (1), *fair* (2), *good* (3), *very good* (4), and *excellent* (5). ASCVD risk is the 10-year percentage risk for a new fatal or nonfatal heart attack or stroke or coronary heart disease death. High lifetime CVD risk is risk $39% (Marma et al., 2010). Conventional sleep duration is 7–9 hr per night. Short sleep duration is six or fewer hours per night. Dysfunction was defined by any experience of feeling unrested, feeling overly sleepy, or not getting enough sleep. Estimates are weighted to represent the civilian noninstitutionalized U.S. population aged 40–79 years.

of 8.1% (95% CI [7.5–8.7%]) and 7.6% (95% CI [7.2–8.0%]) but was significant when adjusting for all covariates (adjusted means of 9.9%; 95% CI [8.6–11.2%]) and 7.4% (95% CI [7.0–7.8%]). Follow-up analysis with the first-order and interaction terms revealed only a significant dysfunction coefficient which was robust to covariates (see Table 3).

Thus, the lower 10-year ASCVD risk among short sleepers reporting dysfunction was explained by the association of reported sleep dysfunction with ASCVD risk rather than an interaction of sleep dysfunction with duration. Participants reporting no sleep-related dysfunction had 1–3% higher 10-year risk than those reporting dysfunction depending upon the model (see Table 3).

Follow-up analysis examining graded dysfunction also revealed an association with risk (see Table 3). For the fully adjusted model the *no dysfunction* group (absolute risk 8.2%; 95% CI [7.7–8.7%]) had higher risk compared with *some dysfunction* (absolute risk 7.4%; 95% CI [6.8–7.9%]) and *moderate dysfunction* (absolute risk 7.5%; 95% CI [7.0–8.0%]) but not *high dysfunction* (absolute risk 7.9%; 95% CI [7.3–8.5%]). The latter three groups were statistically equivalent ($F(2, 30) = 1.37$, $p = .270$).

Lifetime CVD Risk

The percent of respondents with high lifetime CVD risk by sleep duration and reported dysfunction is shown in Table 2. The analysis of sleep duration, sleep dysfunction and their interaction in the 40- to 79-year-old ASCVD-eligible sample revealed an association of dysfunction with high lifetime risk in unadjusted and fully adjusted models. Participants 20–79 years old (i.e., adding ages 20–39 to the sample) also evidenced an association with dysfunction for unadjusted and fully adjusted models. When examining graded dysfunction in this larger group we consistently observed an interaction between sleep duration and dysfunction. In the fully adjusted model the percentage of short sleepers with high lifetime risk (76%; 95% CI [72–80%]) was higher than the percentage of conventional sleepers with high lifetime risk (67%; 95% CI [63–70%]) but only in the *high dysfunction* category (IRR = 1.14; 95% CI [1.03–1.26], $p = .014$; see also online Supplemental Materials Figure 7). Sensitivity analyses using the five graded categories of lifetime CVD risk are in online Supplemental Materials Figure 8.

Discussion

Do individuals who report being unimpaired by sleeping less than the recommended amount (i.e., 7–9 hr/night) have lower objective disease risk compared with HSS in general? The current study examined SRH and objective CVD risk among HSS who do and do not report sleep-related dysfunction (i.e., sleepiness, feeling unrested). HSS with no reported dysfunction evidenced higher SRH compared with their dysfunction-reporting counterparts, but modestly, though significantly, greater 10-year ASCVD risk. Persons with no reported dysfunction had slightly higher prevalence of high lifetime CVD risk across sleep duration groups, which is consistent with the 10-year risk pattern and generalizes that risk to younger age groups (i.e., 20- to 39-years-old). Notably, however, when examining graded dysfunction short sleepers reporting the highest dysfunction had higher lifetime CVD risk. This is the first report that examined lifetime CVD risk by sleep duration and sleep-related dysfunction.

The current findings add to a growing literature examining whether individuals who report no impairment from habitual short sleep duration are functioning as well as they report themselves to be. Prior research indicates that all short sleepers, regardless of reported dysfunction, evidence greater reward-related impulsivity (delay discounting; Curtis et al., 2018). Additionally, short sleepers without reported dysfunction show the same resting state functional connectivity patterns indicative of loss of wakefulness as their dysfunction-reporting counterparts. That is, despite instructions to remain awake with eyes open during a resting-state fMRI protocol, HSS appeared to have difficulty maintaining alertness (Curtis et al., 2016). The current study extends this pattern of findings—a discrepancy between self-assessments and objective indicators—to physical health. Short sleepers without reported dysfunction resembled recommended-length sleepers with respect to SRH, but evidenced modestly higher 10-year and comparable lifetime CVD risk to other HSS (with the exception of those at the highest level of sleep-related dysfunction). Thus, current findings suggest that perceptions of less need for sleep do not translate to less risk for established health consequences of short sleep duration, at least with respect to CVD.

Results of the current study support the supposition that the distinction between HSS who do and do not report daytime dysfunction is consequential. Preliminary evidence suggests that short

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J_ID: ART NO: 10.1037/hea0001085 Date: 27-May-21 Page: 8 Total Pages: 11 4/Color Figure(s) ARTTYPE="ResearchArticle"

sleepers without perceived dysfunction may be characterized by high reward drive, impulsivity, extraversion, and hypomania, whereas those reporting dysfunction are characterized by behavioral inhibition, neuroticism, and may meet criteria for insomnia with short sleep duration (see Williams et al., 2019). Findings of the current study support these hypothesized distinctions: HSS reporting dysfunction endorsed significantly greater symptoms of insomnia compared with HSS-NRD (i.e., sleep latency, nighttime and early morning awakenings, use of sleep medication).

Given these associations, a vigilance regulation model, initially formulated to characterize attention-deficit-hyperactivity disorder (ADHD0 and mania (Hegerl & Hensch, 2014) may be relevant to understanding these HSS. This model posits that individuals characterized by high reward drive and activity level may routinely seek out stimulating activities as a behavioral strategy to override underlying daytime fatigue and sleepiness. In turn, these behavioral motivation tendencies driving habitual short sleep, along with a lack of perceived negative consequences (i.e., no subjective daytime dysfunction) result in further cognitive functioning deficits. In other words, a feed-forward, reciprocal pattern may lead to increasing inaccuracy in judging personal functioning. Indeed, individuals with objectively poorer cognitive or intellectual functioning tend to (erroneously) assess their abilities to be greater than they are (aka the Dunning-Kruger effect; Kruger & Dunning, 1999).

Inaccuracy in self-judgments of alertness can have serious negative consequences. For example, individuals who do not accurately perceive sleepiness are at risk for microsleeps while driving. Indeed, drowsy driving leads to thousands of motor vehicle accidents and related deaths each year (see Higgins et al., 2017). Further, inaccurate perceptions of health may preclude taking appropriate self-regulatory steps to manage health. The association with CVD risk is particularly important in this regard—delay in seeking medical attention in the context of myocardial infarction or stroke can result in more severe impairment or death. Inaccurate perception of health might also include failing to make health behavior changes (e.g., smoking cessation, exercise). Although the focus of the current study was on habitual short sleep, an unexpected finding was the importance of perceived sleep-related dysfunction in general. Future research should further investigate these associations, perhaps targeting poor interoceptive awareness as a potential mechanism.

Despite the implications for safety and functioning, a lack of perceived dysfunction in relation to habitual short sleep may have some advantages. Perceived daytime difficulties in relation to sleep (i.e., attributing poor functioning to sleep loss) can lead to further daytime dysfunction, such as withdrawing from activities, which may place people at risk for depression. Thus, the lack of perceived dysfunction may allow individuals to stay positively engaged in life activities. It is also the case that monitoring for "sleep related threat" including daytime symptoms of fatigue attributed to inadequate sleep is a risk factor for insomnia (Semler & Harvey, 2007).

Found associations with CVD risk suggest that interventions to extend sleep may be warranted and there is promising preliminary evidence for such approaches (e.g., Haack et al., 2013); however, there may be impediments to treatment engagement with some short sleepers. In particular, the lack of foreseeable negative consequences of short sleep means that emerging intervention strategies for "bedtime procrastination" (Kroese et al., 2016) that target raising awareness of such negative outcomes (e.g., Massar & Chee, 2019) may not be effective. Preliminary research phenotyping short sleepers not reporting dysfunction (Curtis et al., 2011) suggests that such individuals rarely seek treatment for sleep disturbance and when they do it is often at the behest of concerned family members. Structured intervention to remove access to stimulating nighttime activities may be the primary option, though without the usual negative consequences of short sleep, there may be challenges with adherence.

Conclusions, Limitations, and Future Directions

It is not uncommon to hear some individuals claim that they do not need the recommended 7–9 hr of sleep/night, citing a lack of perceived fatigue or daytime dysfunction. The current study examined whether such individuals have lower objective health risk compared with HSS who report daytime difficulties. Findings indicated that individuals who report no dysfunction and, by extension, less perceived need for sleep, evidence modestly, though significantly, higher 10-year and comparable high ($\$$39%) lifetime CVD risk compared with short sleepers with the more typical reports of daytime fatigue. This study also indicates that sleep-related (lifetime) CVD risk is evident in much younger adults than previously recognized—an important finding given that lifetime risk indicators are all modifiable. Examination of levels of dysfunction suggest that short sleepers at the highest levels of dysfunction may be a particularly vulnerable group with respect to lifetime CVD risk—a finding with important clinical implications.

The preponderance of current evidence for an association between short sleep duration and health has derived from either experimental manipulation of sleep (i.e., acute effects of sleep loss) or epidemiological studies. What these approaches do not consider are individual differences in perceived dysfunction among HSS. Examination of individual differences in habitual short sleep bridges the gap between traditional survey and experimental sleep studies (Grandner et al., 2010). Strengths of the current study include the examination of both self-reported and objective health risk indicators in a large, nationally representative sample. Findings of the current study are limited to self-reported sleep duration; future research should confirm habitual short sleeper status with daily sleep diaries and actigraphy. A more granular evaluation of sleep duration was not feasible given that the dysfunction questions were only assessed in two NHANES cycles. It should also be noted that 10-year CVD risk calculations exclude individuals who already have CVD; patterns of health risk may differ when individuals with disease are considered. Although CVD is one of the most consequential health outcomes in relation to short sleep duration, future research should examine individual differences in habitual short sleep in relation to other objective health indicators. Overall, results of the current study add to a growing literature examining reported daytime dysfunction and sleep need among HSS. Findings indicate that these variations are of import both with respect to accuracy of self-assessment, as well as objective disease risk.

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