Racial and Socioeconomic Disparities in Sleep and Chronic Disease: Results of a Longitudinal Investigation

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Objectives: Sleep problems appear to differentially affect racial minorities and people of lower socioeconomic status (SES). These population subgroups also have higher rates of many debilitating diseases such as obesity, type 2 diabetes mellitus (T2DM), hypertension, coronary heart disease, stroke, and mortality. Considering the presence of social disparities in sleep and chronic disease, this research aims to assess the role of sleep disparities in the incidence of obesity, T2DM, hypertension, and/or cardiovascular disease (CVD).

Design: The Boston Area Community Health (BACH) Survey is a population-based random-sample cohort of 5502 participants aged 30–79. Sleep restriction (≤5 hours/night) and restless sleep were assessed at baseline. Health status was ascertained at baseline and approximately 5 years later among 1610 men and 2535 women who completed follow-up.

Setting: Participants completed an in-person, home visit, interview at baseline (2002–2005) and follow-up (2006–2010).

Participants: Boston, Massachusetts residents (2301 men, 3201 women) aged 30–79 years from three racial groups (1767 Black, 1876 Hispanic, 1859 White) participated in the BACH Survey.

Results: There were significant differences in the prevalence of sleep-related problems at baseline by both race and SES as well as significant disparities in the incidence of T2DM, high blood pressure and cardiovascular disease at follow-up. Restless sleep was associated with an increased risk of obesity, T2DM, and CVD. However, we found that sleep does not mediate social disparities in health outcomes.

Conclusions: Results from the BACH Survey confirm large social disparities in health outcomes as well as large social disparities in short sleep duration and restless sleep. However, sleep did not appear to mediate the relationship between race, SES, and health disparities. (*Ethn Dis.* 2013;23[4]:499–507)

Key Words: Disparities, Sleep Quality, Chronic Disease

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Introduction

In the United States, 25–30% of the adult population suffers from a chronic sleep disorder or sleep deprivation that are proven contributors to disability, morbidity and mortality.[1] It is estimated that hundreds of billions of dollars are spent each year on direct medical costs for sleep restriction and impairment.[1,2] Scientists are now beginning to recognize the downstream health consequences of sleep-related problems, including increased risk for obesity,[3,4] type 2 diabetes mellitus (T2DM),[5–8] hypertension,[9,10] coronary heart disease,[11] stroke,[12,13] and mortality.[14] Sleeping fewer than five hours a night more than doubles the risk of prediabetes,[15] angina, coronary heart disease, heart attack or stroke.[16] Restless sleep is associated with a 50% increased risk of myocardial infarction (MI).[17] Recent research indicates that sleep restriction results in physiological changes that may have profound implications for these common chronic diseases.[18] There are several mechanisms by which sleep disturbances and/or deprivation may contribute to weight gain and incident obesity. Short sleep increases cortisol and insulin secretion thereby promoting fat storage. Increases in ghrelin and reductions in leptin which stimulate appetite and inhibit satiety regulating signals to the brain, respectively, can lead to increased intake of high fat and high carbohydrate foods.[4] In addition, insufficient or inadequate sleep may lead to decreased energy expenditure, further increasing the risk for weight gain and incident obesity.[19,20] Increased insulin production coupled with impaired glucose metabolism, greatly increase the risk for type 2 diabetes, as well.[21] Short sleep increases blood pressure and sympathetic hyperactivity which provide two potential mechanisms for the link between sleep and cardiovascular events.[1] Sleep restriction and poor sleep quality are now being seen as major risk factors for obesity and obesity-related disease, right along with the two of the most commonly identified risk factors: lack of exercise and overeating.[4,22]

Sleep problems appear to differentially affect racial minorities[23–25] and those of lower socioeconomic status (SES).[25–28] Research suggests that the racial disparities in sleep are partially explained by SES and other related factors (eg, occupation and financial strain).[25,27,29–32] Most studies examining social determinants of sleep have documented worse sleep among minority groups,[24,25,33–37] however there is still some disagreement among studies.[27,38] For example, Patel et al found that African-Americans were 65% more likely than Whites to report poor sleep quality and Hispanics were 59% more likely.[28] However, a study conducted by the same authors found no differences in trouble falling/staying asleep among African Americans vs Whites and found that Hispanics were actually less likely to report these sleep complaints. These conflicting findings underscore the need for additional research estimating the prevalence of sleep-related complaints

among racially diverse populations. Low income, education, and overall SES were frequently associated with reduced opportunities to obtain sufficient sleep and with adverse environmental conditions that compromise sleep quality.[27,39]

Racial minorities and people of lower SES also have higher rates of many debilitating diseases such as obesity, T2DM, hypertension, coronary heart disease, stroke, and mortality. Compared with non-Hispanic White adults, the risk of obesity is 51% and 21% higher among Black and Hispanic adults, respectively.[3] Prevalence of diagnosed T2DM is 77% higher among Black, and 66% higher among Hispanic adults, compared with White adults.[40,41] High blood pressure is twice as common among Blacks as Whites.[42] Blacks are more likely to die of heart disease than Whites, and die younger.[42] Research has indicated that SES plays a role in the causal pathway in many of these chronic diseases.[43,44] The public health implications of these social disparities are profound and have elevated calls for achieving health equity and eliminating disparities to national priority status.[45,46]

The impact of sleep on health outcomes is an area of active research as is our understanding of social inequalities in health. It has been proposed that sleep loss and poor sleep quality may increase the ''allostatic'' load among racial minorities and people of lower SES thereby facilitating the development of chronic conditions such as obesity, diabetes, hypertension and CVD.[39] Therefore, the objective of this research is to examine the role of sleep in the relationship between race, socioeconomic factors and adverse health outcomes. To test the hypothesis that sleep may explain part of the racial or SES gradient in health, we analyzed data from a population-based, racially diverse longitudinal cohort study, the Boston Area Community Health (BACH) Survey.[47,48]

The objective of this research is to examine the role of sleep in the relationship between race, socioeconomic factors and adverse health outcomes.

METHODS

The BACH Survey recruited a random sample of 5502 Boston, Massachusetts residents (2301 men/3201 women) aged 30–79 years from three racial groups (1767 Black/1876 Hispanic/1859 White). Participants completed an in-person interview at baseline (2002–2005) and approximately 5 years later (2006–2010). Further details on methods have been previously published.[47,48] All participants provided written informed consent. The study was approved by the New England Research Institutes' Institutional Review Board. Completed follow-up interviews were obtained for 4145 individuals, resulting in an overall conditional response rate of 80.5%. The mean (SD) time to follow-up was 4.8 (.6) years.

Measures

Race was self-reported by survey participants according to two separate survey questions: ''Do you consider yourself to be Spanish, Hispanic, or Latino (Latina)?'' and ''What do you consider yourself to be? Select one or more of the following'' with response categories of American Indian or Alaska Native, Asian, Black or African American, Native Hawaiian or other Pacific Islander, White or Caucasian, and Other (Specify). These are the standard questions used in the United States as recommended by the Office of Management and Budget.[49] The racial categories used in this research are: 1) non-Hispanic Black, referred to as Black (individuals who self-identified as Black or African American and includes individuals who indicated multiple racial response categories); 2) Hispanic of any race, referred to as Hispanic; and 3) non-Hispanic White, referred to as White (individuals who self-identified as White or Caucasian). Socioeconomic status was determined as a combination of standardized levels of education and income in the Northeast[50] and categorized such that 1/4 of the sample was lower, 1/2 middle, and 1/4 upper SES.

Sleep restriction and restless sleep at baseline were assessed by self-report. Sleep restriction was defined as typically experiencing $\leq$5 hours of sleep per night, the greatest risk category for angina, coronary heart disease, heart attack and stroke,[16] and was assessed among men as part of a hypogonadism screener.[51] Restless sleep was defined as experiencing restless sleep much of the time during the past week.

Obesity was defined as a body mass index (BMI) $\geq$ 30kg/m^2. Height and weight were measured by trained field staff. High blood pressure was ascertained by a combination of self-report of a high blood pressure diagnosis, measured SBP $\geq$ 140mm Hg or DBP $\geq$ 80mm Hg, or anti-hypertensive medication. Type 2 diabetes and cardiovascular disease (myocardial infarction, angina, congestive heart failure, coronary artery bypass, or angioplasty) were ascertained by self-report. Incident cases of disease were defined as new cases at follow-up, among those who were free from the disease of interest at baseline.

Baseline variables were considered as potential confounding variables, including age (categorical, decades), sex (M/F), marital status, alcohol use (low/medium/high), physical activity (low, medium, high), smoking (current/former/never), and anti-depressant use. Race (Black/Hispanic/White) was considered in the SES analyses and SES (lower/middle/upper) was considered as a mediator in the race analyses. Additionally, the following medication coalitions were considered as potential confounders:

Table 1. Descriptive characteristics of the cohort by baseline sleep parameters[a]

	Sleep Restriction[b], *n*=1610	Poor Sleep Quality, *n*=4145
% Reporting Sleep Complaint (95% CI)	**18.2 (14.9–22.0)**	**37.8 (35.1–40.7)**
Race		
Black	31.7 (26.0–37.9)	42.9 (38.8–47.0)
Hispanic	20.0 (14.1–27.5)	43.3 (37.5–49.4)
White	12.5 (8.3–18.3)	34.5 (30.7–38.4)
SES		
low	27.8 (21.9–34.5)	49.1 (44.5–53.7)
medium	19.8 (14.8–26.0)	38.1 (34.1–42.3)
high	8.3 (4.5–14.8)	26.7 (22.1–31.9)
Age		
<40y	20.2 (13.8–28.5)	34.9 (30.0–40.1)
40–49y	20.5 (14.8–27.6)	42.6 (37.3–48.0)
50–59y	14.9 (10.6–20.6)	41.4 (37.1–45.8)
60–69y	13.9 (9.3–20.3)	36.5 (31.5–41.9)
70+	12.9 (6.9–22.9)	33.3 (25.9–41.5)
Sex		
Male	NA	33.5 (29.5–37.8)
Female		41.6 (38.2–45.1)
Married	17.0 (12.2–23.2)	32.7 (28.5–37.1)
Alcoholic drinks		
0	16.8 (12.7–21.8)	42.1 (37.8–46.6)
<1 drink/day	19.1 (13.4–26.3)	35.2 (31.0–39.6)
1-<3 drinks/day	14.9 (9.6–22.4)	35.9 (30.4–41.7)
3+ drinks/day	27.4 (17.7–39.9)	40.3 (30.7–50.6)
Body mass index		
Normal	13.1 (8.9–18.7)	31.8 (26.7–37.4)
Overweight	16.6 (12.0–22.4)	36.2 (31.9–40.8)
Obese	23.8 (17.4–31.7)	44.3 (40.2–48.4)
Physical activity		
Low	19.6 (12.4–29.6)	48.5 (43.8–53.3)
Middle	14.4 (10.9–19.0)	35.5 (31.7–39.5)
High	23.4 (17.0–31.3)	30.7 (25.6–36.3)
Smoking status		
Non-smoker	16.0 (10.7–23.3)	35.7 (31.7–39.8)
Former	11.9 (8.4–16.6)	34.3 (29.8–39.1)
Current	27.7 (20.7–35.9)	46.2 (40.9–51.7)
Self reported health		
Good/excellent	16.4 (12.8–20.7)	34.9 (32.0–38.0)
Fair/poor	30.8 (24.0–38.5)	55.9 (50.6–61.1)
Comorbidities		
Diabetes		
Yes	27.3 (18.5–38.3)	58.6 (50.1–66.7)
No	17.4 (13.9–21.7)	36.3 (33.4–39.3)
HBP		
Yes	25.5 (19.8–32.3)	45.1 (40.7–49.6)
No	15.6 (11.9–20.2)	35.3 (32.1–38.6)
CVD		
Yes	26.6 (18.5–36.7)	47.8 (39.5–56.1)
No	17.5 (14.1–21.6)	37.1 (34.3–40.0)

Table 1. Continued

	Sleep Restriction[b], *n*=1610	Poor Sleep Quality, *n*=4145
% Reporting Sleep Complaint (95% CI)	**18.2 (14.9–22.0)**	**37.8 (35.1–40.7)**
Medication usage		
Secondary sedation coalition		
Yes	25.0 (16.2–36.3)	51.8 (46.6–56.9)
No	16.3 (13.0–20.3)	33.3 (30.1–36.5)
Primary stimulation coalition		
Yes	9.5 (2.7–28.8)	44.6 (33.5–56.3)
No	18.4 (15.1–22.4)	37.6 (34.8–40.5)
Secondary stimulation		
Yes	20.5 (12.3–32.2)	43.4 (38.0–49.0)
No	17.4 (14.1–21.4)	35.7 (32.6–39.0)
Antidepressant		
Yes	22.0 (9.6–42.9)	54.4 (46.9–61.7)
No	17.6 (14.5–21.3)	34.9 (32.0–37.9)

[a] Weighted row % and 95% confidence interval for categorical variables; weighted mean and 95% confidence interval for continuous variables.
[b] Men only.

secondary sedation (medications with sedating effects without primary indication for sleep disorders), primary stimulation (medications with stimulant effects with primary indication for sleep disorders), and secondary stimulation (medications with stimulant effects without primary indication for sleep disorders).

Statistics

Logistic regression was used to model the relation of race and SES with incident outcomes (obesity, T2DM, hypertension, and cardiovascular disease), controlling for confounding variables. Mediation was tested using Baron and Kenny's four steps by estimating the following: 1) the association between the determinants (race or SES) and the outcome(s) (ie, obesity, T2DM, HBP, and CVD), 2) the association between the determinants and the mediator (sleep), 3) the association between the mediator and the outcome(s), and 4) the effect that the mediator has on the determinant – outcome relationship.[52] The last two steps were accomplished by introducing sleep parameters into a logistic regression model with the exposure variables (race and SES). The odds ratios (ORs) and 95% confidence intervals (CI) were reported. Results were considered statistically significant if null hypotheses could be rejected at the .05 level (two-sided).

In order to reduce the bias due to data that are not missing completely at random[53–55] and minimize reductions in precision, multiple imputation was implemented using the Multivariate Imputation by Chained Equations (MICE)[56] algorithm in R.[57] Fifteen multiple imputation datasets were created. Imputations were conducted separately for each racial by sex combination to preserve interaction effects, and the complex survey sample design was taken into account. The proportion of missing data was <1% on all variables except for SES, which was missing for 5.5% primarily because of missing data on household income. Observations were weighted inversely to their probability of selection and weights were post-stratified to the Boston census population in 2000. Analyses were conducted in SUDAAN 9.0.1 (Research Triangle Institute, Research Triangle Park, NC).

RESULTS

Table 1 provides characteristics of the BACH survey population by baseline sleep parameters. Among men, 18.2% (*n*=331) reported short sleep duration. The baseline prevalence of restless sleep was 37.8% (men and women). Sleep restriction was more prevalent among racial minorities, lower SES groups, younger age groups, heavy drinkers (≥3 drinks per day), obese participants, current smokers, and among participants who reported fair or poor health at baseline. Trends with restless sleep were similar. The cumulative incidence of obesity, T2DM, HBP and CVD were 13% (334 incident cases), 3% (194 incident cases), 13% (494 incident cases), and 3% (193 incident cases), respectively.

There were significant differences in the prevalence of sleep-related problems at baseline by both race and SES (Figure 1). Black and Hispanic men were more likely to report sleeping ≤ 5 hours per night than White men (Black: 32%, Hispanic: 20%, White: 12%, *P*=.0001). Lower and middle class men were also more likely to report short sleep duration compared to men of higher SES (lower: 28%, middle: 20%, upper: 8%, *P*<.0001). The racial disparities in short sleep persisted when SES was accounted for.

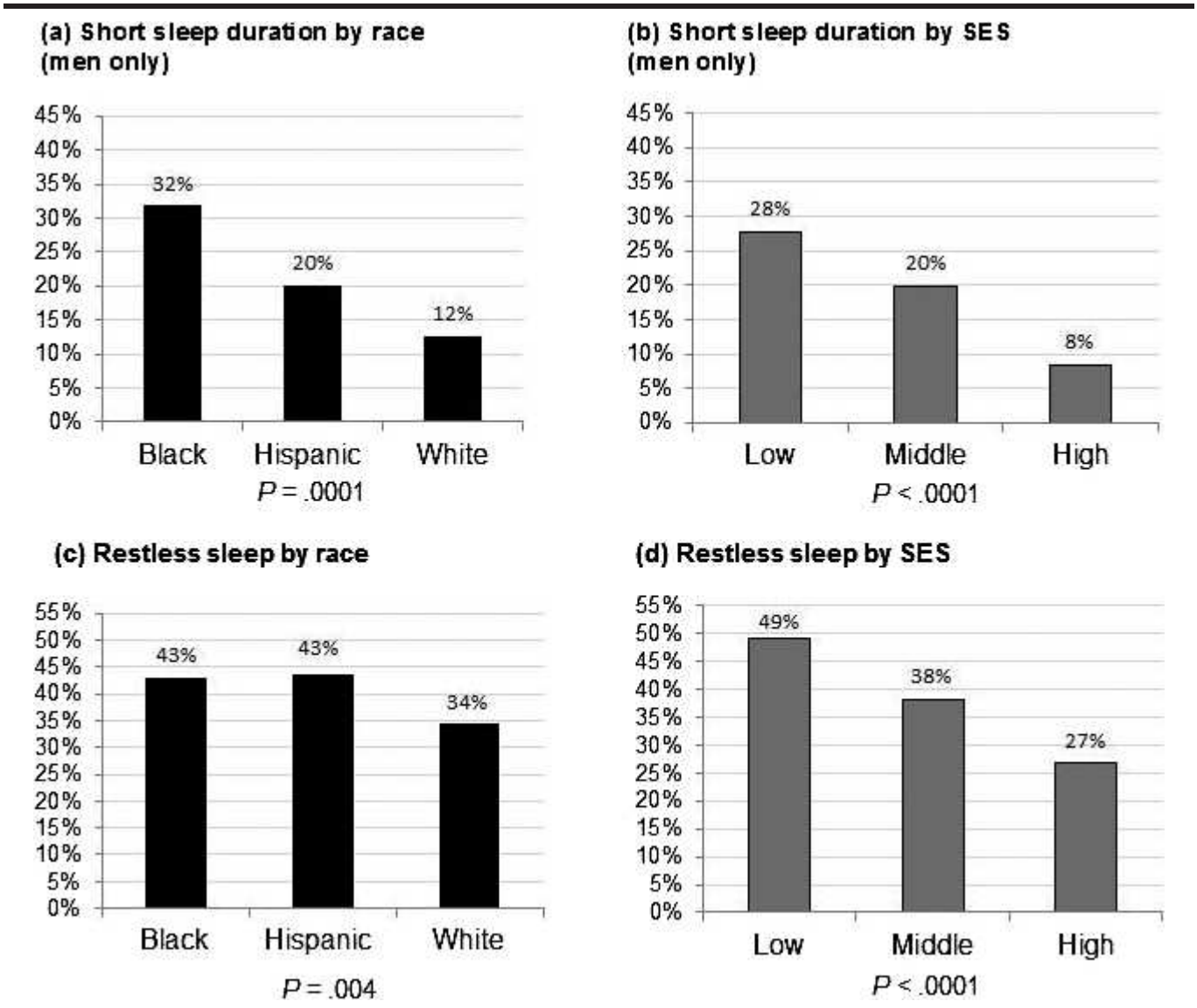

Fig 1. Social disparities in sleep in the BACH Survey

Likewise, the SES disparities persisted when race was included. There were significant disparities in restless sleep as well with Blacks and Hispanics having a higher prevalence of restless sleep (Black: 43%, Hispanic: 43%, White: 34%, P=.004). Lower and middle class adults were also more likely to report restless sleep at baseline (lower: 49%, middle: 38%, upper: 27%, P<.0001). The racial disparities in restless sleep were greatly attenuated within SES strata indicating SES was the greater driver of these disparities.

Figure 2 shows the main effect of sleep on the incidence of the outcomes of interest. Experiencing restless sleep at baseline was associated with a 66% increase in the incidence of obesity (OR=1.66, 95% CI: 1.10–2.49, P=.02) and a 50% increase in the incidence of T2DM and CVD (OR=1.50, P=.08 and OR=1.53, P=.06, respectively), although these latter results were not statistically significant.

The incidence of T2DM was higher among racial minorities and people of lower SES (Table 2, Models 1 and 3), controlling for age and sex. Black and Hispanic participants were 2 times more likely to develop T2DM than Whites. Men and women of lower SES were 9 times more likely to develop T2DM, and middle class adults 3.6 times more likely, when compared to their upper class counterparts. Similarly, the incidence of HBP was higher among Blacks, Hispanics, and lower SES participants. The incidence of CVD was 2.8 times higher among lower SES versus upper SES adults. There were no significant differences in the incidence of obesity by race or SES.

Table 2 (Models 2 and 4) show mediation analyses of restless sleep on racial and SES disparities in obesity,

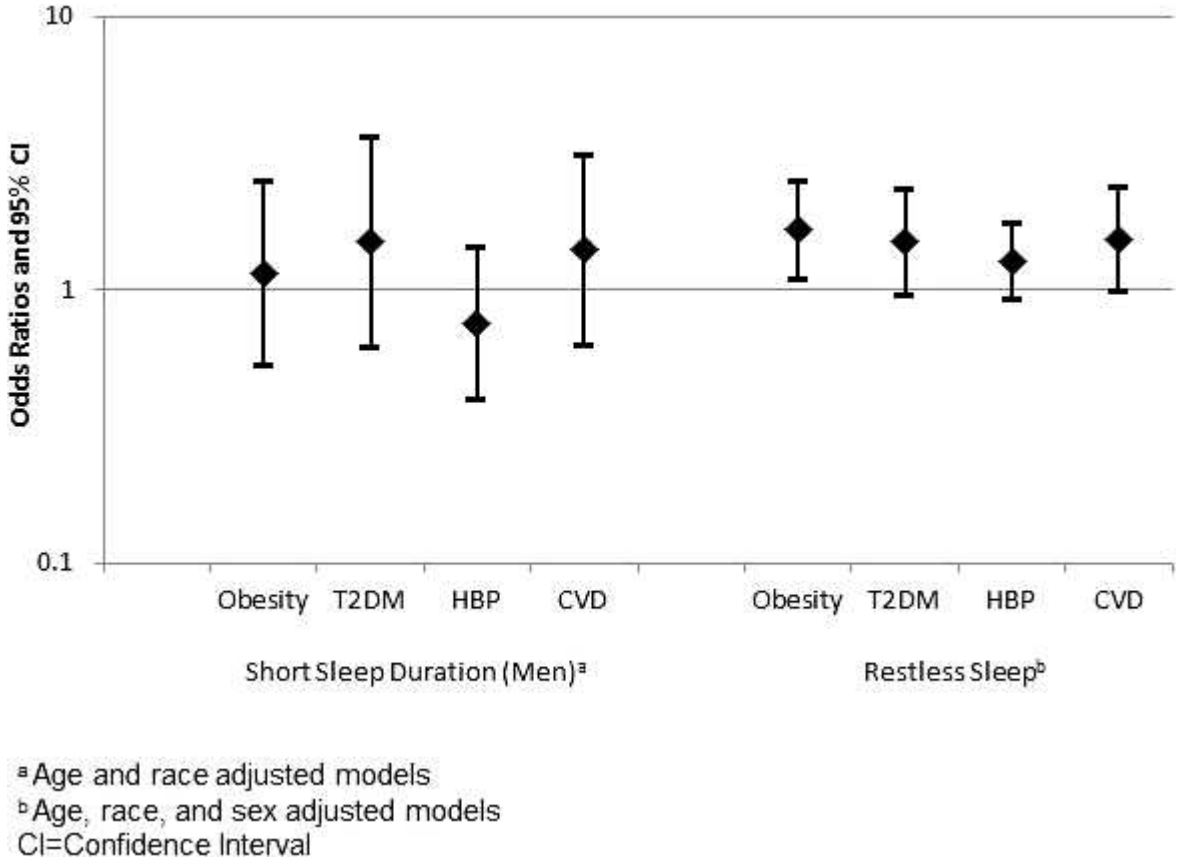

Fig 2. What is the main effect of sleep on incidence of disease in the BACH Survey?

T2DM, HBP, and CVD. These models demonstrate that although there were significant racial disparities in the incidence of T2DM and HBP, and significant SES disparities in the incidence of T2DM, HBP, and CVD—the introduction of restless sleep to the model did influence the measure of effect (OR). The same analyses were conducted to test for a mediation effect of short sleep duration among men, with no evidence of mediation (results not shown).

DISCUSSION

Data from this population-based longitudinal study provide further evidence that sleep loss and restless sleep are common problems and that sleep has important implications for overall health. Restless sleep was associated with an increased risk of obesity and marginally associated with an increased risk of T2DM and CVD. These results underscore the public health consequences of sleep related problems, which are among the most common and readily treatable health problems.

The prevalence of sleep-related problems,[35] the incidence of chronic disease[58,59] and the magnitude of social disparities[35,41,60,61] in the BACH Survey were similar to other population health studies. The BACH Survey demonstrated large racial and SES disparities in both short sleep duration and restless. The incidence of T2DM, HBP and CVD were significantly higher among racial minorities and/or lower SES individuals.

The primary question that we posed here was whether upstream disparities in sleep were reflected in disparities in adverse health outcomes. To our knowledge, this is among the first studies to examine whether sleep disparities in racial or SES groups are involved in disparities in health outcomes. While social disparities in sleep and in the incidence of obesity, T2DM, HBP, and CVD were highly significant and even mirrored one another, we found that sleep does not have a significant role in mediating racial or SES differences in health outcomes. To add context, when we examined the contribution of BMI, a prominent risk factor for T2DM, to SES disparities in T2DM, BMI reduced the OR for SES approximately 10%, whereas adding restless sleep to the same model only reduced the measure of effect by 2%.

The absence of objective sleep measurements and the lack of sleep duration data among women are important limitations to this study. Although in-clinic or ambulatory polysomnography

Table 2. Logistic regression models of social disparities on incident disease outcomes

Model	Covariates	Odds Ratios (95% CI)			
		Obesity	Diabetes	HBP	CVD
1. Base[a]	Race				
	Black vs White	1.18 (.72–1.94)	1.38 (.77–2.48)	2.03 (1.35–3.04)[c]	.82 (.46–1.45)
	Hispanic vs White	1.26 (.75–2.10)	1.03 (.55–1.93)	1.28 (.80–2.05)	.84 (.48–1.45)
	SES				
	Lower vs Upper	1.16 (.66–2.04)	8.19 (2.62–25.58)[c]	2.38 (1.44–3.91)[c]	3.07 (1.45–6.52)[c]
	Middle vs Upper	1.07 (.65–1.76)	3.35 (1.08–10.37)[c]	1.32 (.82–2.12)	1.75 (.83–3.70)
2. Base + Restless Sleep	Race				
	Black vs White	1.16 (.72–1.89)	1.39 (.78–2.49)	2.03 (1.35–3.05)[c]	.82 (.46–1.45)
	Hispanic vs White	1.25 (.74–2.10)	1.04 (.56–1.93)	1.29 (.80–2.07)	.84 (.48–1.47)
	SES				
	Lower vs Upper	1.04 (.59–1.83)	7.80 (2.54–23.93)[c]	2.30 (1.39–3.83)[c]	2.86 (1.35–6.05)[c]
	Middle vs Upper	1.02 (.62–1.68)	3.28 (1.06–10.14)[c]	1.31 (.81–2.10)	1.68 (.79–3.55)
	Restless sleep	1.65 (1.09–2.49)[c]	1.30 (.84–2.01)	1.19 (.85–1.66)	.72 (.47–1.11)
3. Fully Adjusted[b]	Race				
	Black vs White	1.02 (.61–1.73)	1.16 (.62–2.16)	2.19 (1.42–3.38)[c]	.71 (.39–1.29)
	Hispanic vs White	1.20 (.68–2.12)	1.02 (.52–1.96)	1.40 (.83–2.36)	.74 (.38–1.43)
	SES				
	Lower vs Upper	.76 (.42–1.39)	3.87 (1.25–12.03)[c]	1.93 (1.14–3.27)[c]	1.77 (.77–4.07)
	Middle vs Upper	.86 (.52–1.42)	2.32 (.72–7.46)	1.19 (.73–1.93)	1.41 (.65–3.07)
4. Fully Adjusted[b]+ Restless Sleep	Race				
	Black vs White	1.01 (.60–1.69)	1.16 (.62–2.16)	2.19 (1.42–3.38)[c]	.71 (.39–1.29)
	Hispanic vs White	1.20 (.67–2.14)	1.02 (.53–1.97)	1.40 (.83–2.36)	.74 (.38–1.44)
	SES				
	Lower vs Upper	.72 (.39–1.31)	3.84 (1.24–11.94)[c]	1.93 (1.13–3.29)[c]	1.76 (.76–4.05)
	Middle vs Upper	.84 (.51–1.38)	2.31 (.71–7.46)	1.19 (.73–1.94)	1.41 (.65–3.05)
	Restless sleep	.69 (.45–1.05)	1.05 (.67–1.64)	1.01 (.70–1.46)	.97 (.60–1.56)

[a] Base model is adjusted for age and sex.

[b] The fully adjusted model includes all model parameters in the base model as well as all parameters inducing a 10% change in the estimate of race or SES (marital status, physical activity, # of alcoholic beverages per day, smoking status, self-reported health status, obesity, diabetes, high blood pressure, CVD, use of stimulants, and use of anti-depressants).

[c] $P<.05$.

provides a more objective measure of sleep loss and sleep quality than a subjective measure of inadequate or restless sleep, it was not feasible in the context of this particular observational survey. Furthermore, perceived inadequate or restless sleep are similar to sleep complaint measures provided in a primary care setting to indicate a sleep disturbances and other symptoms of sleep disorders.[62,63] The restless sleep measure utilized in this study has shown to be consistently and positively associated with sleep symptoms, sleep burden, and high risk obstructive sleep apnea, as measured by the Berlin Sleep Questionnaire.[64,65] Indeed, in a cross-sectional analysis of the follow-up data we also found a positive association between the restless sleep measure and risk for sleep apnea as measured by the Berlin Sleep Questionnaire (OR= 1.63, 95% CI: 1.27–2.11), a result which was consistent across age, sex, race, SES, and BMI. While some research has indicated that self-reported data on sleep duration typically overestimates the true duration of sleep it has been demonstrated that the reporting bias is non-differential across sex and racial groups.[34,66] In the context of our findings, it is thus possible that the overall prevalence of short sleep duration and restless sleep are underreported and a true mediation effect between sleep disparities and social disparities in disease could be missed. We suggest that future research is needed in order to fully resolve this research question. Studies with either objectively measured, or with expanded measures of sleep assessed over multiple time points in a longitudinal setting, may find greater evidence of mediation than we found in this study. Strengths of the study include its prospective design and the diverse, community-based, random sample participant population.

Restless sleep was associated with an increased risk of obesity and marginally associated with an increased risk of T2DM and CVD.

In conclusion, results from the BACH Survey confirm large social disparities in health outcomes as well as large social disparities in short sleep

duration and restless sleep. Although sleep-related problems appeared to be independently associated with adverse health outcomes, sleep did not appear to mediate the relationship between race, SES, and health disparities.

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REFERENCES

1. Colten H, Altevogt B, eds. *Institute of Medicine Report: Sleep Disorders and Sleep Deprivation, An Unmet Public Health Problem*. Washington, D.C.: The National Academies press; 2006.
2. Stamatakis KA, Kaplan GA, Roberts RE. Short sleep duration across income, education, and race/ethnic groups: population prevalence and growing disparities during 34 years of follow-up. *Ann Epidemiol*. 2007;17(12):948–955.
3. Hairston KG, Bryer-Ash M, Norris JM, Bowden DW, Wagenknecht LE. Sleep duration and five-year abdominal fat accumulation in a minority cohort: the IRAS family study. *Sleep*. 2009;33(3):289–295.
4. Patel SR, Hu FB. Short sleep duration and weight gain: a systematic review. *Obesity (Silver Spring)*. 2008;16(3):643–653.
5. Ayas N, White D, Al-Delaimy W, et al. A prospective study of self-reported sleep duration and incident diabetes in women. *Diabetes Care*. 2003;26(2):380–384.
6. Cappuccio FP, D'Elia L, Strazzullo P, Miller MA. Quantity and quality of sleep and incidence of type 2 diabetes: a systematic review and meta-analysis. *Diabetes Care*. 2010;33(2):414–420.
7. Gangwisch JE, Heymsfield SB, Boden-Albala B, et al. Sleep duration as a risk factor for diabetes incidence in a large U.S. sample. *Sleep*. 2007;30(12):1667–1673.
8. Yaggi HK, Araujo AB, McKinlay JB. Sleep duration as a risk factor for the development of type 2 diabetes. *Diabetes Care*. 2006;29(3):657–661.
9. Gangwisch J, Heymsfield S, Boden-Albala B, et al. Short sleep duration as a risk factor for hypertension: analyses of the first national health and nutrition examination survey. *Hypertension*. 2006;47(5):833–839.
10. Meier-Ewert HK, Ridker PM, Rifai N, et al. Effect of sleep loss on C-reactive protein, an inflammatory marker of cardiovascular risk. *J Am Coll Cardiol*. 2004;43(4):678–683.
11. Ayas NT, White DP, Manson JE, et al. A prospective study of sleep duration and coronary heart disease in women. *Arch Intern Med*. 2003;163(2):205–209.
12. Yaggi HK, Concato J, Kernan WN, Lichtman JH, Brass LM, Mohsenin V. Obstructive sleep apnea as a risk factor for stroke and death. *N Engl J Med*. 2005;353(19):2034–2041.
13. Qureshi AI, Giles WH, Croft JB, Bliwise DL. Habitual sleep patterns and risk for stroke and coronary heart disease: a 10-year follow-up from NHANES I. *Neurology*. 1997;48(4):904–911.
14. Gallicchio L, Kalesan B. Sleep duration and mortality: a systematic review and meta-analysis. *J Sleep Res*. 2009;18(2):148–158.
15. Engeda J, Mezuk B, Ratliff S, Ning Y. Association between duration and quality of sleep and the risk of pre-diabetes: evidence from NHANES. *Diabet Med*. 2013;30(6):676–680.
16. Sabanayagam C, Shankar A. Sleep duration and cardiovascular disease: results from the National Health Interview Survey. *Sleep*. 2010;33(8):1037–1042.
17. Schwartz SW, Cornoni-Huntley J, Cole SR, Hays JC, Blazer DG, Schocken DD. Are sleep complaints an independent risk factor for myocardial infarction? *Ann Epidemiol*. 1998;8(6):384–392.
18. Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. *Lancet*. 1999;354(9188):1435–1439.
19. Knutson KL. Impact of sleep and sleep loss on glucose homeostasis and appetite regulation. *Sleep Med Clin*. 2007;2(2):187–197.
20. Taheri S, Lin L, Austin D, Young T, Mignot E. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. *PLoS Med*. 2004;1(3):e62.
21. Spiegel K, Knutson K, Leproult R, Tasali E, Van Cauter E. Sleep loss: a novel risk factor for insulin resistance and Type 2 diabetes. *J Appl Physiol*. 2005;99(5):2008–2019.
22. Patel SR. Reduced sleep as an obesity risk factor. *Obes Rev*. 2009;10 Suppl 2:61–68.
23. Hale L, Do DP. Racial differences in self-reports of sleep duration in a population-based study. *Sleep*. 2007;30(9):1096–1103.
24. Song Y, Ancoli-Israel S, Lewis CE, Redline S, Harrison SL, Stone KL. The association of race/ethnicity with objectively measured sleep characteristics in older men. *Behav Sleep Med*. 2011;10(1):54–69.
25. Mezick EJ, Matthews KA, Hall M, et al. Influence of race and socioeconomic status on sleep: Pittsburgh SleepSCORE project. *Psychosom Med*. 2008;70(4):410–416.
26. Gellis LA, Lichstein KL, Scarinci IC, et al. Socioeconomic status and insomnia. *J Abnorm Psychol*. 2005;114(1):111–118.
27. Grandner MA, Patel NP, Gehrman PR, et al. Who gets the best sleep? Ethnic and socioeconomic factors related to sleep complaints. *Sleep Med*. 2010;11(5):470–478.
28. Patel NP, Grandner MA, Xie D, Branas CC, Gooneratne N. ''Sleep disparity'' in the population: poor sleep quality is strongly associated with poverty and ethnicity. *BMC Public Health*. 2010;10:475.
29. Hale L, Hill TD, Burdette AM. Does sleep quality mediate the association between neighborhood disorder and self-rated physical health? *Prev Med*. 2010;51(3–4):275–278.
30. Fiorentino L, Marler M, Stepnowsky C, Johnson S, Ancoli-Israel S. Sleep in older African Americans and Caucasians at risk for sleep-disordered breathing. *Behav Sleep Med*. 2006;4(3):164–178.
31. Hall M, Buysse DJ, Nofzinger EA. Financial strain is a significant correlate of sleep continuity disturbances in late-life. *Biol Psychol*. 2008;77(2):217–222.
32. Hall MH, Matthews KA, Kravitz HM, et al. Race and financial strain are independent correlates of sleep in midlife women: the SWAN sleep study. *Sleep*. 2009;32(1):73–82.
33. Redline S, Kirchner HL, Quan SF, Gottlieb DJ, Kapur V, Newman A. The effects of age, sex, ethnicity, and sleep-disordered breathing on sleep architecture. *Arch Intern Med*. 2004;164(4):406–418.
34. Lauderdale DS, Knutson KL, Yan LL, et al. Objectively measured sleep characteristics among early-middle-aged adults: the CARDIA study. *Am J Epidemiol*. 2006;164(1):5–16.
35. National Sleep Foundation. Sleep in America Poll: Summary Findings 2010. 2010.
36. Stamatakis K, Kaplan G, Robers R. Short sleep duration across income, education and race/ethic groups: population prevalence and growing disparities over 34 years of follow-up. *Ann Epidemiol*. 2007;17(12):948–955.
37. Baldwin CM, Ervin A, Mays MZ, et al. Sleep Disturbances, Quality of Life, and Ethnicity: The Sleep Heart Health Study. *J Clin Sleep Med*. 2010;6(2):176–183.
38. Blazer DG, Hays JC, Foley DJ. Sleep complaints in older adults: a racial comparison. *J Gerontol*. 1995;50(5):M280–284.
39. Van Cauter E, Spiegel K. Sleep as a mediator of the relationship between socioeconomic status and health: a hypothesis. *Ann N Y Acad Sci*. 1999;896:254–261.
40. Centers for Disease Control and Prevention. *National Diabetes Fact Sheet: National Estimates and General Information on Diabetes and Prediabetes in the United States, 2011*. Atlanta, Georgia; 2011.

41. Centers for Disease Control and Prevention. *Differences in Prevalence of Obesity Among Black, White, and Hispanic Adults — United States, 2006–2008.* Washington, DC; 2009.
42. Centers for Disease Control and Prevention. CDC Health Disparities and Inequalities Report – United States, 2011. *MMWR Morb Mortal Wkly Rep*. January 14, 2011 2011;Supplement/Vol. 60.
43. Link CL, McKinlay JB. Disparities in the prevalence of diabetes: is it race/ethnicity or socioeconomic status? Results from the Boston Area Community Health (BACH) survey. *Ethn Dis*. 2009;19(3):288–292.
44. Brunner EJ, Marmot MG, Nanchahal K, et al. Social inequality in coronary risk: central obesity and the metabolic syndrome. Evidence from the Whitehall II study. *Diabetologia*. 1997;40(11):1341–1349.
45. U.S. Department of Health and Human Services. Healthy People 2020: Leading Health Indicators. www.healthypeople.gov/2020/default.aspx. Accessed October 7, 2012.
46. Institute of Medicine. *Unequal Treament: Confronting Racial and Ethnic Disparities in Health Care.* Washington, DC; 2003.
47. McKinlay JB, Link CL. Measuring the urologic iceberg: design and implementation of the Boston Area Community Health (BACH) Survey. *European Urology*. 2007; 52(2):389–396.
48. Piccolo RS, Araujo AB, Pearce N, McKinlay JB. Cohort Profile: The Boston Area Community Health (BACH) survey. *Int J Epidemiol*. 2012;doi: 10.1093/ije/dys198.
49. Office of Management and Budget. Recommendations from the Interagency Committee for the Review of the Racial and Ethnic Standards to the Office of Management and Budget concerning changes to the standards for the classification of federal data on race and ethnicity, Revisions to the standards for the classification of federal data on race and ethnicity. In: Register F ed. 1997a, 1997b; 36873–36946.
50. Green LW. Manual for scoring soceioeconomic status for research on health behavior. *Public Health Rep*. 1970;85(9):815–827.
51. Smith KW, Feldman HA, McKinlay JB. Construction and field validation of a self-administered screener for testosterone deficiency (hypogonadism) in ageing men. *Clin Endocrinol (Oxf)*. 2000;53(6):703–711.
52. Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. *J Pers Soc Psychol*. 1986;51(6):1173–1182.
53. Rubin DB. *Multiple Imputation for Nonresponse in Surveys*. New York: J. Wiley & Sons; 1987.
54. Little R, DB R. *Statistical Analysis with Missing Data.* 2nd ed. New York: Wiley; 2002.
55. Schafer J. *Analysis of Incomplete Multivariate Data*. New York: Chapman and Hall; 1997.
56. van Buuren S, Groothuis-Oudshoorn K. Mice: Multivariate imputation by chained equations in R. *J Stat Softw*. 2011;45(3):1–67.
57. *R: A Language and Environment for Statistical Computing* [computer program]. Vienna, Austria: R Foundation for Statistical Computing; 2012.
58. Kenny SJ, Aubert RE, Geiss LS. Prevalence and incidence of non-insulin-dependent diabetes. In: Harris MICC, Stern MP, et al. *Diabetes in America*. Bethesda, Md: National Institute of Health, National Institute of Diabetes, and Digestive and Kidney Diseases, 1995;47–67.
59. Cooper R, Cutler J, Desvigne-Nickens P, et al. Trends and disparities in coronary heart disease, stroke, and other cardiovascular diseases in the United States: findings of the national conference on cardiovascular disease prevention. *Circulation*. 2000;102(25): 3137–3147.
60. Beckles GL, Zhu J, Moonesinghe R. Diabetes - United States, 2004 and 2008. *MMWR Surveill Summ*. Jan 14 2011;60 Suppl:90–93.
61. Mensah GA, Mokdad AH, Ford ES, Greenlund KJ, Croft JB. State of disparities in cardiovascular health in the United States. *Circulation*. 2005;111(10):1233–1241.
62. Grover M, Mookadam M, Armas D, et al. Identifying patients at risk for obstructive sleep apnea in a primary care practice. *J Am Board Fam Med*. 2011;24(2):152–160.
63. Stevenson JE. Diagnosis of sleep apnea. *WMJ*. 2003;102(1):25–27, 46.
64. Netzer NC, Stoohs RA, Netzer CM, Clark K, Strohl KP. Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. *Ann Int Med*. 1999;131(7):485–491.
65. Fulop T, Hickson DA, Wyatt SB, et al. Sleep-disordered breathing symptoms among African-Americans in the Jackson Heart Study. *Sleep Med*. 2012;13(8):1039–1049.
66. Voderholzer U, Al-Shajlawi A, Weske G, Feige B, Riemann D. Are there gender differences in objective and subjective sleep measures? A study of insomniacs and healthy controls. *Depress Anxiety*. 2003;17(3):162–172.

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