

Published in final edited form as:

Clin Pract (Lond). 2013 September ; 10(5): . doi:10.2217/cpr.13.47.

Linking sleep duration and obesity among black and white US adults

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SUMMARY

Aims—The effect of race/ethnicity on the risk of obesity associated with short or long sleep durations is largely unknown. This study assessed whether the sleep–obesity link differentially affects black and whites.

Methods—Analysis was based on data obtained from 29,818 adult American respondents from the 2005 National Health Interview Survey, a cross-sectional household interview survey.

Results—Multivariate-adjusted odds ratios for obesity associated with short sleep (< 6 h) among blacks and whites were 1.98 (95% CI: 1.69–2.30) and 1.20 (95% CI: 1.10–1.31), respectively, and with long sleep (> 9 h) for blacks and whites were 1.48 (95% CI: 1.14–1.93) and 0.77 (95% CI: 0.67–0.89), respectively (all $p < 0.001$).

Conclusion—Race/ethnicity may have significantly influenced the likelihood of reporting obesity associated with short and long sleep durations. Relative to white respondents, an excess of 78% of black respondents showed increased obesity odds associated with short sleep. Black long sleepers also showed increased odds for obesity, but white long sleepers may be at a reduced obesity risk.

Available data do not incontrovertibly demonstrate the optimal sleep amount necessary for wellness and survival. Evidence indicates that a single night of 4-h sleep restriction evoked significant increases in cellular and genomic inflammatory markers among healthy adults [1,2]. Evidence also suggests that individuals experiencing less than the population modal sleep duration [3,4] might be at greater risk of becoming obese [5–7]. The literature in the last decade provides ample epidemiologic evidence in support of the apparent link between individuals' weight and habitual sleep duration. Most studies have shown a U-shaped relationship between these two measures, such that short and long sleepers are at increased risk of becoming obese [6–9]. Furthermore, longitudinal studies document that sleep duration is also a predictor of increased body weight [9,10].

Several investigations have examined the relative contribution of many sociodemographic and medical comorbidities to the short sleep–obesity link. Studies have shown significant associations of short sleep with obesity even after adequate statistical control for age, sex, education, physical activity, depression and alcohol consumption [8,10,11]. This is also the case when hypertension, diabetes and arthritis were adjusted for [8,11,12]. We should also note that those associations remained significant after multivariate adjustment for sleep apnea and sleep disturbances [13]. Although previous research ascertained effects of those confounders on relationships between short sleep and obesity, effects of race/ethnicity on such associations are largely unknown. It is not yet fully elucidated whether race/ethnicity could be a proxy for factors such as socioeconomic position or culture, which could potentially affect health measures.

This is important given epidemiologic evidence demonstrating that short sleep and black race/ethnicity are associated and considering the greater prevalence of obesity among blacks, relative to whites [14,15]. Our previous analysis revealed that, independent of several demographic and medical factors, blacks were more likely to report both shorter and longer sleep time than white respondents, suggesting greater variation in their habitual sleep time [15]. Extreme sleep durations, as noted among blacks, are within the sleep time intervals commonly associated with early mortality and ill health [5–7]. Such data have raised concern among public health advocates because of the association of short and/or long sleep with diseases that disproportionately afflict blacks. In the present study, we investigated the associations of short and long sleep duration with obesity while ascertaining the independent role of race/ethnicity on such associations.

Methods

Participants

Complete data for this analysis was provided by American adults ($n = 29,818$; age range: 18–85 years) participating in the 2005 National Health Interview Survey (NHIS). Of the sub-sample, 85% were whites and 15% were blacks; 44% were men and 56% were women.

Procedures

The NHIS is a cross-sectional household interview survey conducted annually by the National Center for Health Statistics of the Centers for Disease Control and Prevention. The NHIS uses a multistage area probability design that permits representative sampling of US households. Probability samples of the civilian adult population of all 50 states and the District of Columbia were obtained. Respondents were grouped according to place of residence in the USA, referred to as geographic residence: North East, Midwest, South and West. It is estimated that the final sample was characterized by a response rate of 69%. No significant differences in the demographic characteristics between responders and nonresponders were found. Details on sample design can be found in Design and Estimation for the NHIS, 1995–2005 [16].

Participants provided sociodemographic data and information about physician-diagnosed chronic conditions during face-to-face interviews conducted by trained interviewers from the US Census Bureau. The chronic conditions included hypertension, heart disease, cancer, diabetes and arthritis. Participants also estimated habitual sleep duration (using full hour units i.e., 5 h, 6 h, 7 h and so on) by responding to the following question: “On average, how many hours of sleep do you get in a 24-h period?” Sleep duration was coded as either short sleep (< 6 h/night) or long sleep (≥ 9 h/night), referenced to 7–8 h/night sleepers. No information on specific sleep disorders was elicited during the interview.

Participants also reported depressed moods (i.e., feeling of sadness, hopelessness, worthlessness and poor effort) experienced in the past 30 days. The depression severity score utilized in our analysis represents a composite score, summing answers from the above-mentioned four items; scores ranged from 0 to 20 with low scores indicating greater depression.

Activity limitation was measured by asking respondents whether they were able to walk a quarter of a mile without assistance; respondents were classified as 'limited' if they answered "very difficult" or "unable" to perform this activity or 'not limited' if they were able to do it.

Surveys were conducted using computer-assisted personal interviewing, which utilizes a computer program for data collection that guides the interviewer through the questionnaire. The interviewer enters survey responses directly into the computer. The program determines through a computer algorithm whether data entered by the user match against all possible responses to specific questions; the program also checks for consistency against other data collected during the interview, and saves the responses into a survey data file [101].

Statistical analysis

The present analyses focused on addressing differences between black and white Americans. Thus, we excluded the other races from our analyses. Furthermore, there were too few cases in other groups to support robust statistical analyses.

As the NHIS data set amalgamates data from different samples using a multistage area probability sampling design, all analyses performed in this study utilized weighted statistics based on the final weights provided with the NHIS data set. These weights represent a product of weights for corresponding units computed in each of the sampling stages.

Frequency and measures of central tendency were used to describe the sample. Skewed distributions (i.e., BMI and sleep duration) were log-transformed before performing preliminary statistical analyses. In preliminary analyses, Pearson and Spearman correlations were used to explore relationships between variables of interest. Analysis of variance was used for group mean comparisons, and χ^2 test was employed to assess differences in categorical variables. For descriptive purposes, the sleep measure classified participants into three groups: those reporting short sleep (< 6 h) or long sleep (> 9 h) referenced to those who reported sleeping 7–8 h. These cut-off points were chosen based on previous research showing obesity risk associated with short and long sleep durations [4,17,102]. The dependent variable was also a binary measure, categorizing participants into two groups: obese versus non-obese; obesity was defined as BMI ≥ 30 kg/m² [18]. Based on the K-6 scaling system, a score ≥ 13 indicated emotional distress [17].

To test the hypothesis that short sleep (< 6 h) and long sleep (> 9 h) were associated with obesity (BMI ≥ 30 kg/m²), we utilized multivariate logistic regression modeling. BMI was self-reported. In the first model, interactions of short sleep with black and white race/ethnicity were assessed, while adjusting effects of several covariates; black and white race/ethnicity was dummy-coded. In the second model, interactions of long sleep with black and white race/ethnicity were ascertained, while adjusting effects of covariates. Covariates entered in the models were: age, sex, income, geographic residence, smoking and drinking history, depression [19], activity limitations and a history of hypertension, diabetes, arthritis or heart disease. Before constructing the model, univariate logistic regressions were performed to assess associations between hypothesized predictors and the dependent variable. All analyses were performed using SPSS 18.0 (IBM Corporation, NY, USA), using appropriate weights to adjust for the use of complex design [20].

Results

As seen in **Table 1**, blacks were slightly younger than their white counterparts. Fewer blacks indicated in the interview that they completed at least high school, and fewer blacks reported household income of at least US\$35,000 compared with their white counterparts. White Americans were more likely to report a diagnosis of arthritis and heart disease, while black Americans were characterized by a greater likelihood of reporting hypertension and diabetes. The prevalence of obesity (BMI ≥ 30 kg/m²) was greater among blacks than among whites (52 vs 38%; odds ratio [OR]: 1.80; 95% CI: 1.66–1.95; $p < 0.0001$).

In the multivariate-adjusted regression models, we ascertained whether there were interacting effects between black or white race/ethnicity and short sleep (Model A) and long sleep (Model B) on obesity. As shown in **Table 2**, multivariate-adjusted ORs for obesity associated with short sleep among blacks and whites were 1.96 (95% CI: 1.68–2.29; $p < 0.0001$) and 1.23 (95% CI: 1.12–1.44; $p < 0.0001$), respectively. ORs for obesity associated with long sleep for blacks and whites were 1.45 (95% CI: 1.11–1.88; $p < 0.0001$) and 0.78 (95% CI: 0.67–0.89; $p < 0.0001$), respectively. Thus, while increased risk was noted for black long sleepers, the reverse seems to have been true for white long sleepers [21]. Associations of each of the sociodemographic and medical factors adjusted in the models are also shown in **Table 2**.

Discussion

Interest in studying short sleep as a potential modifiable metabolic risk marker is anchored by epidemiologic evidence indicating a gradual decline in population sleep time [3,14,22–25]. In the same time span, there has been a commensurate increase in the number of Americans becoming obese [103]. In tandem with previous research, an analysis of the NHIS data has demonstrated a U-shaped association between sleep duration and obesity, with greater obesity risk associated with sleeping too little or too much [6–9,26]. Our analysis also demonstrated that individuals' race/ethnicity may have a significant effect on the short and long sleep obesity link reported in the sleep literature.

The primary finding of the study was that an excess of 78% of blacks in the NHIS data had greater odds of being a short sleeper and obese, relative to their white counterparts. The observation of race/ethnic effect was independent of individuals' sex, age, income, geographic residence, activity limitations and depression or medical comorbidities such as hypertension, diabetes, heart disease or arthritis. These results demonstrate that race/ethnicity is a factor that should be considered in epidemiologic analysis of the link between obesity and short sleep. Multivariate-adjusted ORs for black and white short sleepers exhibiting obesity risk were 1.98 and 1.20, respectively. Hence, both black and white short sleepers have relatively increased odds of being obese. While it cannot be said that short sleep causes obesity, since our analysis was based on cross-sectional data, it is apparent that individuals of the black race/ethnicity sleeping 6 h or less might be at greater risk of becoming obese [27].

Despite the fact that we controlled for ten covariates in our multivariate logistic models, interactions between short sleep and black and white race/ethnicity on obesity remained significant. Nonetheless, it is as yet unclear what factors mediated excess odds of obesity noted for black short sleepers. Both medical and lifestyle factors might play a role. Conceivably, short sleep among some of the black participants might reflect undiagnosed sleep apnea. Unfortunately, the present database did not include data permitting such determination. Nonetheless, we are guided by available findings from a study comparing 225 black and 622 white volunteers, aged 2–86 years, showing that 31% of blacks versus

10% of whites had sleep apnea [28]. Emerging evidence suggests that sleep apnea might be influenced by genes within pathways involved with obesity, inflammatory processes and craniofacial features, with differing risk profile for blacks and whites [29–31].

A previous study examining factors affecting sleep duration over a period of 34 years has shown that socioeconomic position is a robust determinant of short sleep duration, even after adjusting for health-related characteristics linked to short sleep duration. Results of a cross-sectional analysis of data obtained from 32,749 Americans in 1990, ages 18 years or older, indicated that living in an inner city, which is common among blacks, was associated with increased risk of short sleeping [32]. Our analyses adjusted effects of economic status and geographic residence on short sleep. Notwithstanding, other indices of socioeconomic position such as work schedule might have contributed to the curtailment of sleep time among blacks. Evidence from a study assessing the impact of shift work and race/ethnicity on the diurnal rhythm of blood pressure shows that black shift workers experienced shorter sleep relative to shift workers from other ethnic groups [33].

When viewed in the context of health disparities research, sleep duration may be a key factor in understanding diseases that disproportionately burden blacks. Whether sleep time in this population is curtailed by lifestyle choices or restricted by sleep fragmentation, as is the case in sleep apnea, blacks may be at increased risk of developing diabetes and cardiovascular disease due to excess obesity and short sleep. Data from the Nurse's Health Study suggests that curtailed sleep duration resulting from sleep fragmentation induced by sleep apnea may lead to the development or exacerbation of diabetes [34,35]. Evidence shows that short sleep is a precursor for inflammatory processes, which are important risk markers for diabetes and cardiovascular disease. Although the mechanism linking chronic sleep restriction and obesity is not fully delineated, targeting short sleep duration might be a novel and effective method of preventing and treating obesity among at-risk individuals.

The secondary finding of our analysis is that race/ethnicity also interacted with long sleep in predicting the odds of being obese. Compared with average sleepers (7–8 h), black long sleepers were 48% more likely to be obese, whereas white long sleepers were 23% less likely to be obese. Adjusting for the presence of depressed moods and economic status – two factors commonly linked to sleeping long hours – did not affect those associations. The finding of increased odds for obesity associated with sleeping longer than 8 h among blacks is consistent with previous data [9,36]. A recent study showed that individuals sleeping 9–10 h nightly were 21% more likely to become obese over a 6-year period than were those reportedly sleeping 7–8 h [37]. Of note, the sleep–obesity associations were not substantially affected by adjustment for food intake, exercise or medical confounders. We surmise that blacks sleeping 9 h or longer are also at increased risk of being obese, although to a lesser extent relative to those sleeping 6 h or less.

It is not clear whether white long sleepers were characterized by reduced odds of being obese. Conceivably, the mechanisms underlying potential effects of long sleep on obesity differ between black and white ethnicities. It may be that blacks reporting long hours of sleep spend less time engaged in social activities that would provide adequate, healthy life choices; hence their propensity to become obese. Another likely explanation may be that medical comorbidity associated with obesity might predispose susceptible blacks to spending more time in bed. Oversleeping among white respondents might reflect greater prevalence of other factors promoting long sleep such as depression. Evidence shows a high prevalence of depression among individuals of the white race/ethnicity [38,39]. While this apparent discrepancy awaits verification by other analyses, our findings might explain why sleep durations greater than 7 h are not consistently associated with either an increased or decreased obesity risk in the extant literature [11]. Plausibly, associations between long

sleep and obesity vary based upon sociodemographic groupings, suggesting that stratification of epidemiologic sleep data by race/ethnicity might be necessary in clarifying interpretation of associations between long sleep and obesity.

Conclusion

Future studies should investigate the mediators of excess odds of obesity associated with short sleep among blacks. They should also examine whether short sleep has a direct effect on likelihood of becoming obese through dysregulation of neuroendocrine functioning (e.g., leptin and ghrelin) or whether short sleepers might engage in activities that lead to obesity (e.g., greater opportunity for food consumption or unhealthy eating habits). Our analyses adjusted effects of ten covariates, which have shown significant associations in previous research. An important limitation of the current findings is that both sleep duration and medical data were based on subjective reports obtained by a trained interviewer. Future studies should examine the accuracy of self-reported data (e.g., sleep time and/or weight and height) in this population. Evidence from appraisal research suggests that recall bias may influence the accuracy of self-reported data among blacks [40,41]. It is of interest to examine whether short or long sleep durations obtained from actigraphy would also be associated with increased risk of obesity derived from waist circumference or skinfold thickness measurement. Our analysis did not adjust for other important factors such as sleep apnea, insomnia, excessive daytime sleepiness or fatigue, shift work and dietary habits, which have adverse effects on sleep duration; such data were not available in the NHIS data. Our study explored the relationship between race/ethnicity and obesity, and did not establish any cause and effect relationships among race/ethnicity, sleep durations and obesity or address the possibility of reverse causality because we used a cross-sectional design. Although estimates were based on analyses adjusting for stratification and clustering effects using weights provided by NHIS, further examination of data was not made using SUDAAN (RTI International, NC, USA), which is sometimes recommended for standard error ascertainment in parametric analysis. The explanatory models presented in this article relied on nonparametric analyses.

Acknowledgments

This work was supported by funds from NIH (RO1HL78566, R01MD004113 and R25HL105444). The authors have no other relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript apart from those disclosed.

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Practice points

- Both short and long sleep durations were associated with obesity among black and white respondents.
- Individuals habitually sleeping 6 h or less might be at greater risk for obesity, although obesity risk seems greater for blacks.
- Race/ethnicity may have significantly influenced the likelihood of reporting obesity associated with short and long sleep durations.
- Obesity risk reduction interventions should incorporate evidence-based sleep improvement therapies.

Future perspective

Notwithstanding the study limitations, results suggest that individuals habitually sleeping 6 h or less might be at greater risk for obesity, although obesity risk seems greater for blacks. Obesity-risk reduction interventions should incorporate evidence-based sleep improvement therapies. They might also consider the importance of physical activity, which is known to affect both sleep amount and weight.

Table 1

Comparison of sociodemographic and health data of black and white Americans participating in the 2005 National Health Interview Survey.

Variable	Black [†]	White [†]	F/ χ^2
Age \pm SD (years)	45.56 \pm 6.91	48.02 \pm 18.02	72 [*]
Completed High School (%)	76	81	53 [*]
Household income >US\$35,000 (%)	16	24	140 [*]
Hypertension (%)	36	27	133 [*]
Diabetes (%)	12	8	62 [*]
Heart disease (%)	6	8	29 [*]
Arthritis (%)	22	24	11 [*]

* p < 0.0001.

[†] Sample included 4473 blacks (15%) and 25,345 whites (85%).

Table 2

Associations of sociodemographic, medical and sleep factors with obesity.

Variables	Model A: short sleep		Model B: long sleep	
	OR	95% CI	OR	95% CI
Age	0.98*	0.97–0.99	0.98*	0.98–0.99
Sex	0.64*	0.59–0.70	0.63*	0.57–0.71
Income	1.18*	1.07–1.31	1.04	0.92–1.17
Geographic residence	0.99	0.95–1.03	1.01	0.95–1.06
Smoking history	1.10*	1.06–1.14	1.06*	1.01–1.12
Drinking history	0.97*	0.95–0.98	0.96*	0.94–0.98
Hypertension	3.08*	2.77–3.43	3.03*	2.65–3.47
Heart disease	0.89	0.756–1.05	0.87	0.72–1.07
Arthritis	1.43*	1.27–1.60	1.65*	1.42–1.90
Diabetes	3.46*	2.89–4.14	2.85*	2.31–2.52
Depression	0.98	0.97–0.99	0.98	0.96–1.00
Activity limitations	0.54*	0.48–0.62	0.57*	0.48–0.67
Sleep duration + black	1.96*	1.68–2.29	1.45*	1.11–1.88
Sleep duration + white	1.23*	1.12–1.44	0.78*	0.67–0.89

The sleep duration measure included individuals who reported short sleep (< 6 h) or long sleep (> 9 h) versus those sleeping 7–8 h habitually.

OR: Odds ratio.

* $p < 0.0001$.