Short Sleep Duration Is Associated with Higher Energy Intake and Expenditure among African-American and Non-Hispanic White Adults[1,2]

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Abstract

Habitual short sleep duration appears to increase the risk of obesity. The objective of this paper is to investigate the association of habitual sleep duration with objective measures of energy balance. One hundred twelve African-American and 111 non-Hispanic whites aged 21–69 y participated in a cross-sectional study of dietary assessment and biomarkers. Participants reported the mean number of hours per day spent sleeping over the past year. Short sleep duration was defined as ≤6 h/d of sleep. Energy intake (kilocalories) was objectively assessed using the 2-point doubly labeled water technique to determine total energy expenditure, which is approximately equal to energy intake. Physical activity energy expenditure (kilocalories) was estimated as total energy expenditure minus each participant's calculated basal metabolic rate and the thermogenic effect of food. Compared with participants who slept ≤6 h, individuals who slept 8 h were significantly less likely to be obese (OR: 0.33; 95% CI: 0.14, 0.79). However, this association was not linear across 6–9 h of sleep (P-trend = 0.16). There was an inverse association between sleep and energy intake (P-trend = 0.07): compared with ≤6 h/d, adults who reported ≥9 h sleep consumed 178 fewer kcal/d. There was also an inverse association between sleep and physical activity (P-trend = 0.05): compared with ≤6 h/d of sleep, adults who reported 9 h of usual sleep expended 113 fewer kcal/d in physical activity. These data indicate that, compared with longer sleep duration, adults who report habitual short sleep duration have somewhat higher physical activity energy expenditure but considerably higher energy intake. Habitual short sleep duration appears to be 1 of the facets of modern life leading to a mismatch between energy intake and physical activity.   J. Nutr. 144: 461–466, 2014.

Introduction

Considerable evidence supports an association between short sleep duration and increased risk of overall mortality and chronic diseases, such as type 2 diabetes mellitus and cardio-vascular disease (1–11). Although the mechanisms underlying these relations are not fully understood, short sleep duration has been associated with metabolic changes that may increase risk of chronic disease, such as insulin resistance and chronic inflammation (4,5,9,12–15). The data are especially strong regarding the association of short sleep duration with weight gain and obesity (5,9,16–21). To result in weight gain, short sleep duration must cause increased energy intake and/or reduced energy expenditure. However, conflicting results were found in small experimental studies (n = 10–20 participants) that investigated the effects of short-term sleep deprivation on dietary intake or physical activity (22–27). However, the effects of artificially induced, and often extreme, short-term sleep deprivation on adults in a laboratory setting may not provide information relevant to the effect of more modest and habitual sleep deprivation on energy homeostasis in free-living populations. The Nurses' Health Study (n = 68,183) found that self-reported energy intake decreased by ~25 kcal for each hour of sleep reduction (5). However, this finding is likely confounded because energy intake is underreported and obese adults are known to underreport to a greater degree than normal-weight adults (28–31). The National Health Survey found slightly lower self-reported physical activity among short sleepers (32), whereas other epidemiologic studies have found no relation (5) or an increase in activity (33). However self-reported physical activity has considerable error relative to objective measures (34).

The objective of this study was to investigate the association of habitual sleep duration with the risk of obesity and objective measures of energy intake and physical activity. Participants were

healthy adults who participated in the University of California, Los Angeles (UCLA) Energetics Study of dietary assessment methods and biomarkers (n = 223). Half of the participants were African American. Energy intake and physical activity energy expenditure were estimated using the doubly labeled water method. To our knowledge, this is the first study to test whether habitual sleep duration in humans influences objectively measured energy intake and physical activity under free-living conditions.

Participants and Methods

Participants and study protocol. The UCLA Energetics Study was conducted between July 2006 and June 2009. Participants were recruited using public postings and online advertisements (35). Eligible participants were African-American and non-Hispanic white adults between the ages of 21 and 69 y who resided within 80 km of UCLA. Eligibility requirements included having a stable weight for the previous 6 mo, willingness to maintain current dietary and physical activity habits for the duration of the study, ability to read and speak English, and access to a telephone. Participants were required to be nonsmokers because cigarette smoking may reduce the accuracy of doubly labeled water measurements (36). Potential participants were excluded if they reported any of the following: gastrointestinal surgery, intestinal disease, pancreatic disease, diabetes, hemophilia, alcoholism, mental disorder, hypothyroidism, bipolar or seizure disorders, congestive heart failure, renal failure or other conditions affecting fluid balance, treatment with supplemental oxygen, and use of antiretroviral, anti-neoplastic, anti-ulcer/anti-reflux, or central nervous system drugs.

Study participants visited the UCLA General Clinical Research Center twice over a 2-wk period. At the first visit, they gave written consent, had their height and weight measured, completed questionnaires on sociodemographic and psychosocial factors, and were administered a dose of doubly labeled water. At visit 2, participants were weighed and completed an exit questionnaire and the doubly labeled water protocol (see below). Participants were compensated $150 at the end of the clinical visits and $50 if they completed the study protocol. The UCLA Institutional Review Board approved the study protocol, and all participants provided written informed consent.

Sleep assessment. Sleep was assessed using a question from the Multiethnic Cohort Study (37) that asked about the average number of hours per day spent sleeping for the past year (≤ 5, 6, 7, 8, 9, or ≥ 10). There were only 12 responses for ≤ 5 h sleep per day, which was combined with 6 h. There only 4 responses for ≥ 10 h/d, which was combined with 9 h. Short sleep duration was defined as ≤ 6 h/d (2).

Assessment of energy intake. We assessed energy intake (kilocalories per day) using doubly labeled water to determine total energy expenditure (TEE). In weight-stable groups such as ours, TEE is approximately equal to energy intake and provides an unbiased estimate of current energy intake. TEE was assessed using doubly labeled water (38), which measures CO_2 production over a 2-wk period and thus is a form of indirect calorimetry (39,40). Briefly, at the first visit, participants ingested doubly labeled water in a single dose composed of 1.8 g of 10% $H_2^{18}O$ and 0.12 g of 99.9 atom percent ^2H_2O per kilogram of estimated total body water. After dosing, urine specimens were obtained at 2, 3, and 4 h. Approximately 14 d later, 2 consecutive urine specimens were collected 1 h apart. Urine samples were stored at $-20°C$ until analyzed by isotope ratio MS.

Dilution spaces for ^2H and ^{18}O were determined using the calculations described by Coward et al. (40). Total body water was calculated as the mean of the dilution spaces of ^2H and ^{18}O after correction for isotopic exchange (1.041 for ^2H and 1.007 for ^{18}O). The CO_2 production rate was determined by measuring the difference between the elimination rates of labeled oxygen and hydrogen using the 2-point doubly labeled water method outlined by Schoeller (41). The CO_2 production rate was converted into energy expenditure using a food quotient of 0.86 to predict the respiratory quotient (42,43). The doubly labeled water method has been validated against indirect calorimetry

and found to be accurate to 1–2% (36). Reliability studies of doubly labeled water energy expenditure measurement have reported CVs from 5% over a 2-wk interval (31,44) to 8% over a 6-mo interval (31).

Physical activity energy expenditure. Physical activity energy expenditure (kilocalories per day) was estimated using a 2-step procedure. First, the Harris-Benedict equation was used to estimate each participant's basal metabolic rate based on height, weight, age, and gender (45). The thermic effect of meals was assumed to be 10% of TEE (46). Therefore, physical activity energy expenditure was calculated as follows: (0.9) TEE minus the participant's calculated basal metabolic rate.

Statistical analyses. Univariate analyses were used to examine the association of participant characteristics with usual hours of sleep. The χ^2 test was used to test for the association of sleep duration with gender, race, education, marital status, percentage obese, and employment status. ANOVA was used to examine the association of sleep duration with age and basal metabolic rate. The Kruskal-Wallis test was used to test the association of short sleep duration with BMI (kilograms per meters squared) and energy intake.

Logistic regression was used to model the association of self-reported sleep duration with overweight/obesity (defined as BMI ≥ 25). Linear regression was used to model the association of sleep duration with energy intake and physical activity energy expenditure (both kilocalories per day). Age, gender, education, and race were selected for inclusion in all models based on a priori hypotheses and the univariate results. In the linear regression models, weight was also included as a confounder of the relation of sleep with energy intake and physical activity expenditure. The significance level for point estimates was set at 5%; significance level for linear test of trends was set at 10%. All analyses were completed using the R language for statistical computing, version 2.11.0 (47).

Results

Among 333 consented individuals, 268 were enrolled, 262 completed the study, and 223 had complete data and were included in this analysis. **Table 1** gives participant characteristics stratified by hours of self-reported sleep duration per day over the past year. The mean ($\pm$SD) age of the sample was 38 $\pm$ 13 y, 65% were female, 50% were African American, and the remainder were non-Hispanic white. This was a well-educated sample, with 60% of participants having at least a college education. Thirty-five percent of participants reported ≤ 6 h of sleep per day, 36% reported 7 h, 20% reported 8 h, and 9% reported ≥ 9 h. Participants reporting ≤ 6 h of sleep per day tended to be older, African American, and less educated (all $P < 0.05$). Overall, 29% of participants were overweight and 24% were obese. The median objectively assessed energy intake was 2450 kcal/d, with an interquartile range of 2090–2910 kcal/d. This was a weight-stable sample that experienced no significant change in weight during the 2-wk doubly labeled water measurement interval (data not shown). **Figure 1** graphically shows the unadjusted associations of habitual sleep duration with BMI, energy intake, and physical activity energy expenditure: sleep was inversely associated with BMI ($P < 0.001$) and energy intake ($P = 0.03$), with no statistically significant association with physical activity ($P = 0.25$).

Table 2 gives the odds of being overweight or obese (BMI $\geq$ 25) by usual hours of sleep per day after adjustment for age, gender, race, and education. The association of sleep with BMI was not linear (P-trend = 0.16). However, compared with participants who slept ≤ 6 h, individuals who slept 8 h were significantly less likely to be obese (OR: 0.33; 95% CI: 0.14, 0.79).

As shown in **Table 3**, there was an inverse association between sleep and energy intake after adjustment for age, gender, weight,

TABLE 1 Participant characteristics by usual sleep duration over the past year in a doubly labeled water study[1]

	Overall (n = 223)	≤6 h/d (n = 79)	7 h/d (n = 81)	8 h/d (n = 44)	≥9 h/d (n = 19)	P
Age, y	38 ± 12	43 ± 13	36 ± 12	33 ± 11	37 ± 13	<0.001[2]
Females, % (n)	65 (145)	62 (49)	65 (53)	64 (28)	79 (15)	0.58[3]
African American, % (n)	50 (112)	70 (55)	38 (31)	41 (18)	42 (8)	<0.001[3]
Education, % (n)						0.001[3]
Some college or less	40 (90)	46 (36)	32 (26)	41 (18)	53 (10)	
College graduate	44 (99)	47 (37)	46 (37)	52 (23)	11 (2)	
Postgraduate	15 (34)	8 (6)	22 (18)	7 (3)	37 (7)	
Marital status, % (n)						0.52[3]
Married/living with partner	24 (50)	23 (17)	25 (19)	26 (10)	21 (4)	
Divorced/widowed/separated	17 (36)	24 (18)	13 (10)	10 (4)	21 (4)	
Never married	59 (123)	53 (39)	62 (48)	64 (25)	58 (11)	
Employment status, % (n)						0.81[3]
Employed	71 (150)	70 (52)	73 (57)	74 (29)	63 (12)	
Unemployed/unknown	29 (60)	30 (22)	27 (21)	26 (10)	37 (7)	
BMI	25.2 (22.7–29.8)	28.0 (24.4–30.9)	25.0 (21.8–29.6)	23.7 (21.7–25.5)	24.6 (21.8–26.5)	<0.001[4]
BMI > 30 kg/m,[3] % (n)	24 (54)	34 (27)	23 (19)	14 (6)	10 (19)	0.03[3]
Energy intake,[5] kcal/d	2450 (2090–2910)	2540 (2170–2940)	2490 (2100–2980)	2380 (2080–2860)	2050 (1910–2420)	0.22[4]
Basal metabolic rate,[6] kcal/d	1470 ± 249[4]	1520 ± 238[4]	1470 ± 248[4]	1460 ± 266[4]	1310 ± 192[4]	0.013[2]

[1] Values are means ± SDs, medians (IQRs), or percentages (n).
[2] P values calculated using ANOVA.
[3] Values calculated using χ^2 test.
[4] Values calculated using Kruskal-Wallis test.
[5] Estimated using doubly labeled water to assess total energy expenditure.
[6] Calculated using the Harris-Benedict equation.

race, and education (P-trend = 0.07). Specifically, compared with ≤6 h of sleep, participants who reported longer sleep consumed fewer total kilocalories per day: −50 for 7 h, −41 for 8 h, and −187 for ≥9 h. Also shown in Table 3, there was a significant inverse association between sleep and physical activity (P-trend = 0.05) Compared with ≤6 h of sleep, participants who reported longer sleep also expended less energy in physical activity (in kcal/d): −39 for 7 h, −46 for 8 h, and −114 for ≥9 h.

Discussion

Consistent with the literature (5,9,16,18), we found that self-reported short sleep duration over the past year was associated with higher risk of overweight/obesity. There appeared to be a U-shaped relation between sleep and BMI, which is consistent with several reports in the literature (21,48,49). However, the sample sizes were modest with higher amounts of sleep, with only 19 participants reporting that they usually slept ≥9 h/d.

These results also indicate that individuals who reported short sleep duration had higher amounts of energy intake, as observed in many experimental studies (23,24,26,50,51). Mechanisms by which insufficient sleep may increase energy intake include the following: 1) more opportunities for eating during the day and at night; 2) emotional stress and psychologic distress; 3) increased susceptibility to food reward; 4) greater energy needed to sustain extended wakefulness; and 5) increases in appetite driven by hormones, such as leptin and ghrelin (4,10,52–56).

We also found that individuals who reported short sleep duration had higher amounts of physical activity, consistent with some (23,25,57) but not all (51) studies. It is notable that, for most people, ≥90% of energy expenditure from physical activity is accounted for by activities of daily living, such as sitting, standing, walking, and other occupational, volitional, and spontaneous activities (58–60). Therefore, increased physical activity energy expenditure associated with short sleep may occur because participants are awake more hours per day.
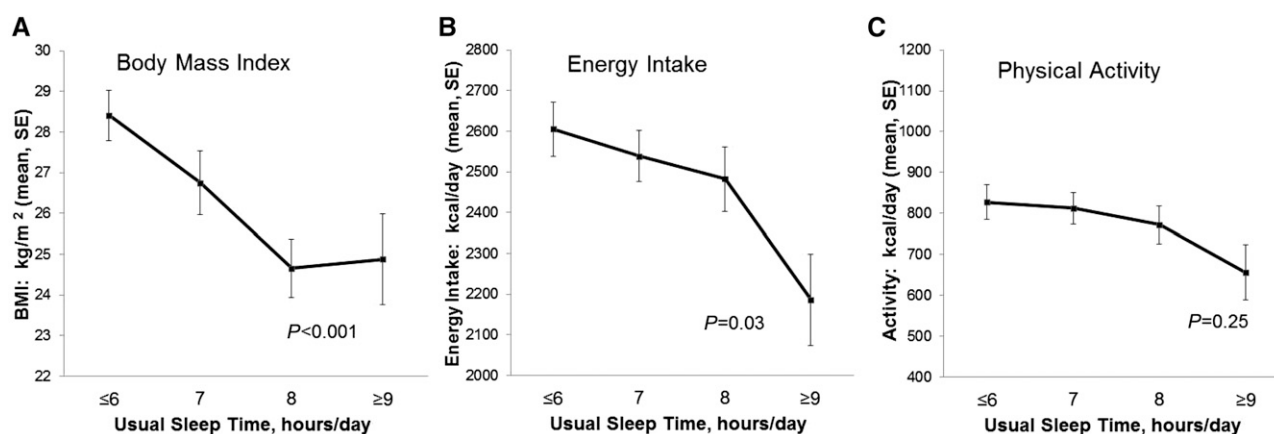

FIGURE 1 The association of usual sleep duration with BMI (A), energy intake (B), and physical activity (C) among African-American and non-Hispanic white adults. Values are means ± SEs; n = 79 for ≤6 h, n = 81 for 7 h, n = 44 for 8 h, and n = 19 for ≥9 h (n = 223).

TABLE 2 Association of usual sleep duration over the past year with overweight/obesity (BMI ≥ 25) among adult participants in a doubly labeled water study ($n = 223$)[1]

Logistic regression model	n	OR (95% CI)	
		Unadjusted models	Multivariable adjusted[2]
Usual sleep, h/d			
≤6	79	1	1
7	81	0.42 (0.22, 0.81)	0.73 (0.35, 1.52)
8	44	0.19 (0.09, 0.43)	0.33 (0.14, 0.79)
≥9	19	0.30 (0.11, 0.94)	0.50 (0.16, 1.51)
P-trend[3]		0.17	0.16

[1] BMI calculated as weight in kilograms divided by height in meters squared.

[2] Adjusted for age, gender, race, and education.

[3] P-trend calculated with linear regression models fitting ORs on sleep categories (≤6, 7, 8, or ≥9) or mean age per each age category.

Several biologic mechanisms were proposed for how sleep may influence the risk of obesity. In particular, sleep modulates the 24-h pattern of secretion of 2 key hormones involved in appetite and energy regulation: leptin and ghrelin (61). Specifically, partial sleep deprivation appears to result in decreased circulating leptin and increased ghrelin, both of which lead to increased appetite (62). There is also considerable evidence that short sleep duration leads to abnormalities in glucose metabolism, leading to insulin resistance and increased fat storage (62,63). Finally, there is some suggestion that inadequate sleep can promote a proinflammatory state via increased concentrations of circulating leukocytes and cytokines, which have been associated with an increased risk of type 2 diabetes (63,64).

The Energetics Study was cross-sectional and, therefore, we cannot definitively establish whether the exposure (i.e., sleep) preceded or resulted from the outcome (i.e., obesity or energy balance). It is possible that obesity increased the risk of short sleep duration via obesity-associated conditions, such as obstructive sleep apnea, gastroesophageal reflux, asthma, and heart failure (17). However, this was a young and healthy sample, and these conditions were rare.

The major strength of this analysis was the objective assessment of energy intake in a large sample of free-living adults, which is unique in the literature on sleep and obesity. A limitation of this study was that we did not have an objective assessment of sleep. Average hours of sleep per day over the past year was based on 1 question, providing only a rudimentary assessment of habitual sleep duration (65), which suggests that the associations observed in this study were attenuated. We were also unable to identify the reasons for short sleep, which can include voluntary sleep curtailment, insomnia, and sleep apnea or other sleep disorders. Another limitation concerns our use of an equation to estimate participants' basal metabolic rate instead of indirect calorimetry. Nonetheless, the objective estimate of physical activity used in this analysis captured both volitional exercise and non-exercise activity thermogenesis and therefore was more comprehensive than self-reports of recreational physical activity (66). Although this was a relatively large sample for a doubly labeled water study, some of the sleep strata had small sample sizes, and therefore this analysis was likely somewhat underpowered for a definitive test of these associations. Finally, biomarker data, such as glycemic control, are not available in this study.

Approximately one-third of this sample reported short sleep duration, which is similar to data from the 2004–2007 National Health Interview Survey ($n = 29,818$) in which 28% of U.S. adults reported sleeping ≤6 h/night (32). African Americans may be at particular risk of the adverse effects of inadequate sleep (67). Similar to our findings, the National Health Interview Survey found that, compared with whites, African Americans were more likely to report short sleep ($P < 0.0001$) (68).

Interventions to improve sleep duration and quality represent a novel and promising approach to weight management because sleep habits are amenable to improvement (69). Sleep interventions may also be a useful adjunct to weight loss interventions. In a laboratory study of adults ($n = 11$) subjected to 14 d of caloric restriction with 8.5 or 5.5 of sleep, participants with 8.5 h of sleep experienced 1.4 kg of fat loss compared with 0.6 kg with 5.5 h sleep (70). Randomized trials are needed to test the effect of sleep interventions on weight, energy intake, and physical activity, particularly in minority populations.

To summarize, these data indicate that, compared with longer sleep duration, adults who report habitual short sleep duration have a somewhat higher physical activity energy expenditure but considerably higher energy intake. Short sleep duration appears to be 1 of the facets of modern life leading to a mismatch between energy intake and expenditure that leads to a risk of obesity. Over the past several decades, chronic sleep

TABLE 3 Association of usual sleep duration over the past year with energy intake (kilocalories per day) and physical activity energy expenditure (kilocalories per day) among adult participants in a doubly labeled water study ($n = 223$)[1]

	n	Model 1 energy intake[2] (kcal)		Model 2 physical activity[3] (kcal)	
		Unadjusted β (SE)	Multivariable adjusted β (SE)	Unadjusted β (SE)	Multivariable adjusted β (SE)
Intercept	223	2604.9 (63.2)	2358.9 (70.5)	827.3 (38.9)	694.4 (58.2)
Usual sleep, h/d					
≤6	79	0	0	0	0
7	81	−65.6 (88.8)	−50.4 (60.9)	−14.6 (54.6)	−39.3 (50.3)
8	44	−122.6 (105.6)	−40.7 (72.3)	−55.3 (65.0)	−45.6 (59.7)
≥9	19	−418.4 (143.5)	−187.3 (96.0)*	−171.0 (88.3)	−113.5 (79.3)
P-trend[4]		0.03	0.07	0.25	0.05

[1] Linear regression models. Multivariable models adjusted for age, gender, weight, race, and education. *$P < 0.05$ for the multivariable adjusted β variable.

[2] Estimated using doubly labeled water to assess total energy expenditure.

[3] Estimated as total energy expenditure minus basal metabolic rate (calculated using Harris-Benedict equation) and the thermogenic effect of food.

[4] P-trend values were calculated with linear regression models fitting point estimates on sleep categories (≤6, 7, 8, or ≥9).

deprivation has grown to epidemic proportions concurrent with the rise in obesity (17). In the 2011 U.S. National Sleep Foundation poll, 63% of Americans said their sleep needs were not being met during the week (71). In addition to the putative effects of short sleep duration on increased risk of mortality, chronic diseases, and obesity, inadequate sleep is associated with work absenteeism and diminished job performance, car accidents, degraded cognition, decreased quality of life, and increased healthcare use (72–75). The results of this study reinforce the importance of diagnosing sleep disorders and promoting adequate sleep, especially among overweight and obese adults (1).

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