



Original Article

Associations of self-reported and actigraphy-assessed sleep characteristics with body mass index and waist circumference in adults: moderation by gender



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ABSTRACT

Objectives: Self-reported sleep duration has been linked to body mass index (BMI) and waist circumference in previous work; however, data regarding if these associations are stronger in men or women have been mixed, and few studies have objectively measured sleep. We investigated self-reported and actigraphy-assessed sleep characteristics in relation to BMI and waist circumference and examined the extent to which these associations differ by gender.

Design: Archived cross-sectional data collected from 2004 to 2006 from the National Survey of Midlife Development in the United States (MIDUS) Biomarkers Study were used. Participants included 1248 adults (43% men) who reported their habitual sleep duration, and a subset of participants ($N = 441$; 40% men) who underwent seven nights of wrist actigraphy.

Results: Self-reported total sleep time (TST), actigraphy-assessed TST, and actigraphy-assessed sleep efficiency (SE) were inversely associated with BMI in the full sample of both men and women. Gender moderated associations between actigraphy assessments of sleep and anthropometric variables; however, TST and SE were related to BMI and waist circumference in women only. Associations between sleep and waist circumference were independent of BMI.

Conclusions: Sleep duration and sleep continuity are associated with body weight and distribution of body fat, but these associations were stronger or were only present in women.

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1. Introduction

A cross-sectional association between reported sleep duration and body mass index (BMI) has been well-established over the past decade. A 2008 meta-analysis of 17 studies demonstrated that adults reporting five or fewer hours of sleep per night were at higher odds of being obese, and that every additional hour of sleep was associated with a .35 kg/m² decrease in BMI [1]. A smaller literature suggested that shorter sleep also may be related to higher waist circumference, which correlates with cardiovascular risk factors more closely than BMI, and that it is an independent predictor of diabetes mellitus (DM), cardiovascular morbidity, and mortality [2–11]. Approximately half of the studies that have investigated sleep and waist circumference suggest that the two are related and are independent of BMI [2,7,9].

There are gender differences in sleep, such that men have shorter less efficient sleep yet report fewer sleep problems than women [12]. However, it is unclear if sleep duration is differentially related to anthropometric measures by gender. Several of the studies that have examined gender-specific links in adult samples report that associations of sleep with BMI and waist circumference are stronger or exist only in women [13–15], while others report that the links are stronger or only present in men [6,16–18]. Still others have failed to observe differences by gender [19–21].

The bulk of the literature examining sleep in relation to BMI is based on self-reported sleep, typically assessed by questionnaires that inquire about habitual sleep duration. The correlation between self-reported sleep and objectively measured sleep duration is moderate [22,23], and inclusion of wrist actigraphy in addition to subjective data may help identify the specific sleep characteristics that are most closely related to obesity. For example, several studies have shown that aspects of sleep continuity that can be assessed with actigraphy such as sleep efficiency (SE) also may correlate with BMI [20,24–27]. We are not aware of any studies that have examined actigraphy-assessed SE in relation to body fat distribution. Actigraphy assessment may be especially informative when investigating gender differences in the links

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between sleep and body weight. The degree of agreement between self-reported and actigraphy-assessed sleep duration has been shown to vary by demographic and physical health factors, including gender and obesity [28]. For instance, in the Coronary Artery Risk Development in Young Adults study, individuals with a lower BMI were more likely to overestimate their sleep duration relative to actigraphy than individuals with a higher BMI [22]. Thus, it is possible that individuals who weigh less are characterized as longer sleepers independent of actual sleep time, which could influence the covariation of BMI and sleep duration based on self-report.

In our study, we investigated associations between sleep and anthropometric variables using both subjective and objective measures of sleep. Specifically, we examined self-reported sleep duration, actigraphy-assessed sleep duration, and actigraphy-assessed SE for their associations with measured BMI and waist circumference in a large sample of adults. We hypothesized that shorter less efficient sleep would be associated with higher BMI and with higher waist circumference. We then explored the extent to which these associations differed between men and women.

2. Methods

2.1. Participants and design

Participants were from the Midlife in the United States (MIDUS) II study, a longitudinal follow-up of the original MIDUS I study ($N = 7108$). The MIDUS I study began in 1994 and was designed to investigate the role of psychosocial and behavioral factors in physical and mental health differences in adults. From 2004–2006, the Biomarker Project of MIDUS II was conducted to obtain comprehensive biologic assessments in a subsample of MIDUS I respondents. Participants were considered eligible for the Biomarker Project if they took part in the original MIDUS I study and were able to travel to the clinical research site. Biomarker data collection was performed at three General Clinical Research Centers (University of California Los Angeles, University of Wisconsin, and Georgetown University). All analyses in our study used archived data from the MIDUS II Biomarker Project subsample, which included 1255 participants ages 34–84 years who provided informed consent (as approved by The University of Wisconsin Madison Health Sciences Institutional Review Board). Fifty-seven percent of participants were women and 93% reported their primary race group as white. Participants at all three sites provided self-reported sleep duration data. Seven participants had missing or incomplete self-reported sleep duration data and were excluded from analyses, yielding a total sample size of 1248. Objective sleep assessments, obtained with an Actiwatch wrist activity monitor, were collected at one research site only (University of Wisconsin). Thus analyses examining actigraphy assessments of sleep were based on 441 participants.

2.2. Measures

2.2.1. Anthropometrics

Height, weight, and waist circumference were measured using a standardized procedure. BMI was calculated by dividing body weight in kilograms by height in meters squared. Waist circumference was measured around the narrowest point between the ribs and iliac crest and recorded to the nearest millimeter.

2.2.2. Sleep

Self-reported sleep duration was assessed using item 4 on the Pittsburgh Sleep Quality Index (PSQI), a self-report instrument developed to assess sleep quality over the past month [29]. The PSQI item 4 asks: “During the past month, how many hours of

actual sleep did you get at night (This may be different than the number of hours you spend in bed)?” Participants completed the PSQI during or just before the clinic visit. In addition to completing the PSQI, participants at University of Madison, WI, underwent an actigraphy protocol, which included wearing a Mini Mitter Actiwatch®-64 wrist monitor continuously for 7 consecutive days following the clinic visit, along with completing a daily sleep diary over the same period. The Actiwatch was configured to collect activity data in 30-s epochs. Data were downloaded and stored in a database for processing with Actiware software. Sleep diary data were used to identify the time that participants first tried to fall asleep and the time that they finally woke. Actigraphy-assessed total sleep time (TST) was defined as the total amount of minutes scored as sleep in a given diary-defined interval. Actigraphy-assessed SE was defined as the percentage of the interval that was spent sleeping. Actigraphy-assessed TST and SE were averaged across the seven nights of the study for use in analysis.

2.2.3. Gender and covariates

Gender was self-reported. Current smoking status and engagement in regular physical activity (“Do you engage in regular exercise, or activity, of any type for 20 min or more at least 3 times per week?”) were determined by self-report and were dichotomously coded. Archived data for sleep apnea were not available; therefore, self-reports of sleep disturbance over the past month due to coughing or snoring as assessed by PSQI item 11e was used as a proxy for sleep-disordered breathing. Those who reported that coughing/snoring disrupted their sleep one or more times per week were compared to those that reported a disturbance less than once per week or never. Depressive symptoms were continuously measured with the Center for Epidemiological Studies Depression Scale [30]. Self-reported medical conditions were dichotomously coded and included current or previous heart disease, transient ischemic attack (TIA) or stroke, and DM. Use of sleep medication was assessed using the PSQI item #7, which asks about prescription or over-the-counter sleep medication use over the past month. Those who reported using sleep medication less than once a week or not at all were compared to those who reported using sleep medication once a week or more. Data on self-reported menopausal status were only available for a subset of women ($N = 508$, 71%). Women were considered postmenopausal if they reported that they had not had a menstrual period in the past 3 months due to menopause or hysterectomy.

2.3. Statistical analysis

Linear regression models were used to examine the associations between sleep and anthropometric variables. Covariates were selected a priori and all models were adjusted for age, gender, smoking status, physical activity, sleep disturbance due to coughing/snoring, sleep medication use, disease status (heart disease, TIA or stroke and DM), and depressive symptoms. Models examining waist circumference as an outcome were additionally adjusted for BMI. Data on menopausal status were available for a subset of participants. Because inclusion of menopausal status as a covariate would have resulted in a loss of over 200 women, we did not include this variable as a standard covariate. Rather, we reported results of exploratory analyses testing the associations of sleep with BMI and waist circumference after adjusting for menopausal status in addition to the covariates listed above and results from models testing for interactions between sleep and menopausal status. We also reported results from models that explored interactions between sleep and age.

Self-reported TST, actigraphy-assessed total sleep time, and actigraphy-assessed SE were initially examined in three separate models. In the first step of analyses, we tested for main

associations of sleep characteristics with BMI in the full sample (the term *full sample* was used to indicate that both men and women were included in analyses). A gender \times sleep interaction term was entered in the second step of the model, using the relevant sleep variable. If an interaction was observed at $P < .10$, the sample was stratified by gender and the association between the sleep variable and BMI was separately examined in men and women using linear regression. The second set of analyses used an identical process to examine the associations among sleep parameters, gender, and waist circumference. We also conducted a set of analyses with both actigraphy-assessed TST and actigraphy-assessed SE in the same model to determine which was more strongly associated with outcomes. Finally, as both short and long sleep duration have been related to BMI in previous reports, we investigated if curvilinear relationships between sleep duration and BMI or waist circumference existed in the full, mixed gender sample and in men and women separately. No curvilinear relationships were observed; thus we reported results for linear tests only.

3. Results

3.1. Sample characteristics

Sample characteristics are displayed in Table 1. Age of participants ranged from 34 to 84 years. Men had shorter actigraphy-assessed TST and decreased actigraphy-assessed SE than women; men and women did not differ in self-reports of TST.

3.2. Sleep measures

In the full sample of both men and women, self-reported and actigraphy-assessed TST were correlated at $r = .34$, $P < 0.001$; self-reports were about 40 min longer than actigraphy assessments, on average. Correlations were similar in men and women ($r = .36$ and $.34$, respectively). Actigraphy-assessed TST and SE were correlated at $r = .61$, $P < 0.001$.

Analysis of variance and t tests were used to determine if discrepancies between measures of TST differed by gender or BMI. Discrepancies between self-reported and actigraphy-measured TST differed by gender; whereas men overestimated their sleep time relative to actigraphy by 54 min, women overestimated by

32 min on average ($t [434] = -2.63$; $P = .009$). Discrepancies between self-reported and actigraphy-measured TST did not differ by BMI group (normal weight vs overweight vs obese) in the full sample of men and women ($F [2, 433] = .62$; $P = .80$), or within men and women separately (men: $F (2, 169) = .09$; $P = .91$ and women: $F [2, 261] = .85$; $P = .43$). When examining Pearson product moment correlations, BMI did not correlate with self-report/actigraphy discrepancies ($r = .01$, $P = .80$). Waist circumference was marginally correlated with self-report/actigraphy discrepancies ($r = .09$, $P = .06$). When the sample was stratified by gender, waist circumference was not correlated with discrepancies in either men ($r = .03$, $P = .70$) or women ($r = .07$, $P = .22$).

3.3. Associations of covariates with BMI and waist circumference

In models that included covariates only, lack of regular physical activity ($\beta = .17$, $P < .001$), non-smoking ($\beta = -.10$, $P = .03$), frequent nighttime coughing/snoring ($\beta = .17$, $P < 0.001$), and DM ($\beta = -.16$, $P < .001$) were associated with higher BMI. In models adjusted for all covariates and for BMI, male gender ($\beta = .29$, $P < 0.001$), frequent nighttime coughing/snoring ($\beta = .07$, $P = 0.01$), TIA/stroke ($\beta = -.06$, $P = .04$), and higher BMI ($\beta = .79$, $P < .001$) were associated with higher waist circumference.

3.4. Sleep characteristics, BMI, and waist circumference

Associations between sleep parameters and anthropometric outcomes in the full sample of both men and women, and among men and women separately are displayed in Table 2 for BMI and in Table 3 for waist circumference.

3.4.1. Self-reported TST

In the sample including both men and women, shorter self-reported sleep was associated with higher BMI after adjusting for all covariates. The interaction between gender and self-reported TST was not related to BMI ($\beta = .04$, $P = .17$). Self-reported sleep time was not associated with waist circumference in the sample of both men and women. There was a significant interaction between gender and self-reported TST in relation to waist circumference ($\beta = .03$, $P = .05$). However, after stratifying the sample

Table 1

Sample characteristics of participants with self-reported sleep duration data ($N = 1248$) and participants with actigraphy data ($N = 441$).

	Self-reported sleep duration			Actigraphy		
	Full sample ($N = 1248$) Mean (SD)	Men ($N = 538$) Mean (SD)	Women ($N = 710$) Mean (SD)	Full sample ($N = 441$) Mean (SD)	Men ($N = 175$) Mean (SD)	Women ($N = 266$) Mean (SD)
Age (y)	54.5 (11.7)	55.2 (11.9)	54.1 (11.5)	54.1 (11.6)	55.4 (11.7)	53.2 (11.5)
Body mass index (kg/m ²)	29.8 (6.6)	29.7 (5.4)	29.9 (7.4)	30.6 (7.3)	30.0 (5.7)	31.0 (8.2)
Waist circumference (cm)	97.6 (17.0)	103.9 (15.8)**	92.9 (16.4)**	98.7 (17.5)	104.0 (15.8)**	95.2 (17.7)**
PSQI total sleep time (h)	6.9 (1.2)	6.9 (1.2)	6.9 (1.3)	6.8 (1.3)	6.8 (1.3)	6.9 (1.4)
Actigraphy total sleep time (h)				6.2 (1.1)	5.9 (1.2)**	6.3 (1.1)**
Actigraphy sleep efficiency (%)				79.2 (10.6)	76.6 (11.9)**	80.9 (9.2)**
CES-D depressive symptoms	8.7 (8.2)	8.2 (7.8)	9.0 (8.4)	9.1 (8.3)	8.9 (8.4)	9.2 (8.3)
	<u>No (%)</u>	<u>No (%)</u>	<u>No (%)</u>	<u>No (%)</u>	<u>No (%)</u>	<u>No (%)</u>
Regular physical activity	954 (76.4)	418 (77.7)	536 (75.5)	316 (71.7)	129 (73.7)	187 (70.3)
Current smoker	186 (14.9)	89 (16.5)	97 (13.7)	69 (15.6)	33 (18.9)	36 (13.5)
Regular sleep medication use (≥ 1 /week)	227 (18.2)	81 (15.1)*	146 (20.6)*	74 (16.8)	23 (13.1)	51 (19.2)
Heart disease	142 (11.4)	84 (15.6)‡	58 (8.2)‡	45 (10.2)	29 (16.6)‡	19 (7.1)‡
Transient ischemic attack/stroke	53 (4.2)	24 (4.5)	29 (4.1)	22 (5.0)	7 (4.0)	15 (5.6)
Diabetes mellitus	155 (12.4)	70 (13.0)	85 (12.0)	72 (16.3)	30 (17.1)	42 (15.8)
Sleep trouble due to cough/snore (≥ 1 /week)	266 (21.3)	121 (22.5)	145 (20.5)	92 (21.3)	42 (24.0)	50 (18.8)

Abbreviations: SD, standard deviation; y, years; h, hour; CES-D, Center for Epidemiological Studies–Depression Scale.

‡ $P \leq .05$, represent difference between men and women in t tests or χ^2 tests.

* $P \leq .01$, represent difference between men and women in t tests or χ^2 tests.

** $P \leq .001$, represent difference between men and women in t tests or χ^2 tests.

Table 2

Standardized regression coefficients from linear regression models of sleep and body mass index in the full sample and by gender.

	Full sample β	Men β	Women β
PSQI total sleep time	-.06*	-.03	-.07
Actigraphy total sleep time	-.16***	-.06	-.20***
Actigraphy sleep efficiency	-.12*	-.04	-.17**

Abbreviation: PSQI, Pittsburgh Sleep Quality Index.

*Standardized regression coefficient indicates the change in one standard deviation of body mass index given a one standard deviation increase in the independent variable.

^bCovariates include age, regular physical activity, smoking status, sleep disruption due to coughing/snoring, disease status, sleep medication use, and depressive symptoms.

^cEach independent variable was entered in a separate linear regression model.

* $P \leq .05$.

** $P \leq .01$.

*** $P \leq .001$.

Table 3

Regression coefficients from linear regression models of sleep and waist circumference in the full sample and by gender.

	Full sample β	Men β	Women β
PSQI total sleep time	.02	.04	-.001
Actigraphy total sleep time	-.03	.03	-.07*
Actigraphy sleep efficiency	-.05	.05	-.13***

Abbreviation: PSQI, Pittsburgh Sleep Quality Index.

*Standardized regression coefficient indicates the change in one standard deviation of waist circumference given a one standard deviation increase in the independent variable.

^bCovariates include age, physical activity, smoking status, sleep disruption due to coughing/snoring, disease status, sleep medication use, and depressive symptoms.

^cEach independent variable was entered in a separate linear regression model.

* $P \leq .05$.

*** $P \leq .001$.

by gender, self-reported sleep time was not related to waist circumference in men or women.

3.4.2. Actigraphy-assessed TST

Shorter actigraphy-assessed sleep time was associated with higher BMI in the full sample of men and women. Gender and actigraphy-assessed sleep time interacted in predicting BMI ($\beta = .10$, $P = .03$). In analyses stratified by gender, shorter actigraphy-assessed sleep was related to higher BMI in women, though there was no relationship in men (Fig. 1). In the full sample, shorter actigraphy-assessed sleep was not related to higher waist circumference. However, there was a marginally significant interaction between gender and actigraphy-assessed TST in predicting waist circumference ($\beta = .05$, $P = .06$). After stratifying by gender, shorter actigraphy-assessed sleep was associated with greater waist circumference in women, but there was no association in men.

3.4.3. Actigraphy-assessed SE

Decreased SE was associated with higher BMI in the mixed gender sample, and gender interacted with actigraphy-assessed SE in predicting BMI ($\beta = .11$, $P = .03$). Stratifying the sample by gender showed that poorer SE was related to higher BMI in women only. In a subsequent model that included actigraphy-assessed TST in addition to SE, the beta for SE was attenuated ($\beta = -.09$, $P = .26$), while TST remained inversely associated with BMI ($\beta = -.17$, $P = .02$) in women. In men, neither sleep parameter was associated with BMI (SE: $\beta = -.07$, $P = .51$; TST: $\beta = .02$, $P = .89$).

In the mixed gender sample, SE was not associated with waist circumference. However, the interaction between gender and actigraphy-assessed SE was related to waist circumference

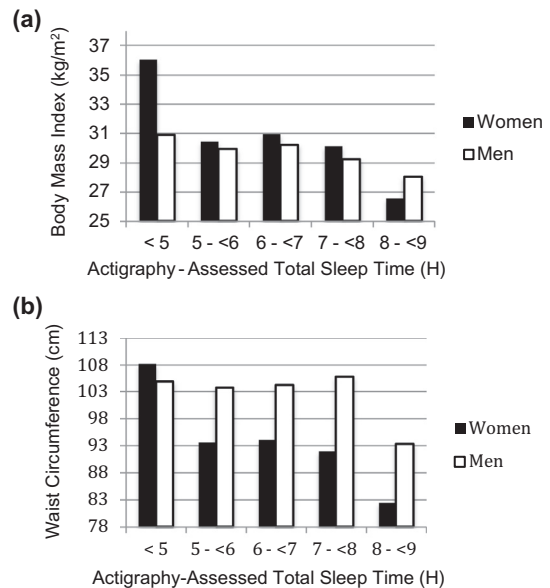


Fig. 1. (a) Unadjusted means of body mass index by categories of actigraphy-assessed total sleep time (TST) averaged across 7 nights in women and men. (b) Unadjusted means of waist circumference by categories of actigraphy-assessed TST averaged across 7 nights in women and men.

($\beta = .09$, $P < 0.001$). After stratifying by gender, lower SE was associated with higher waist circumference in women, with no association in men (Fig. 2). A subsequent model included both actigraphy-assessed TST and SE. In women, SE ($\beta = -.12$, $P = .004$) but not total sleep time ($\beta = -.02$, $P = .56$), remained related to waist circumference. In men, neither sleep parameter was associated with waist circumference (SE: $\beta = .05$, $P = .45$; TST: $\beta = .01$, $P = .94$).

3.5. Exploratory analysis of menopausal status and age

After adjusting for covariates, menopausal status was not associated with BMI ($\beta = .08$, $P = .17$) or waist circumference ($\beta = -.01$, $P = .94$). Adjustment for menopausal status did not alter the associations between sleep and BMI or between sleep and waist circumference reported above. Menopausal status did not interact with self-reported TST, actigraphy TST, or actigraphy SE in predicting BMI or waist circumference (all P s $> .20$). Age did not interact with self-reported TST, actigraphy TST, or actigraphy SE in predicting BMI or waist circumference ($P = .25$ for all). When participants aged > 65 years ($n = 232$, 18.6%) were excluded, results remained similar to those reported above.

4. Discussion

Reports of habitual sleep duration have been linked to BMI in multiple studies [1], and there is mounting evidence that actigraphy assessments of sleep duration also covary with BMI [20,25]. Prior data regarding whether or not these relationships differ by gender are mixed [6,13–18]. Among all participants in our study, increased BMI was associated with shorter and less efficient sleep; however, further examination by gender showed that these associations were only present in women. Links between sleep and waist circumference also were moderated by gender, with actigraphy-assessed sleep duration and SE relating to waist circumference in women only. Thus although our results generally replicate associations between sleep and anthropometric characteristics found in

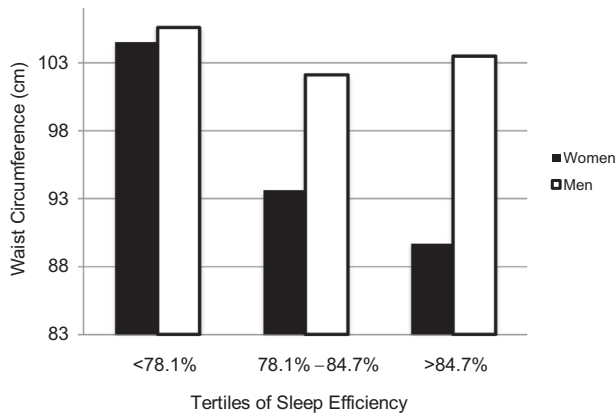


Fig. 2. Unadjusted means of waist circumference by tertiles of actigraphy-assessed sleep efficiency averaged across 7 nights in men and women.

previous samples combining both genders, follow-up analysis revealed that these associations were primarily limited to women.

Several physiologic and behavioral mechanisms linking shorter sleep to higher BMI have been suggested. In cross-sectional studies short sleep was associated with increased appetite and altered eating patterns [31–34], and experimental sleep restriction had an impact on appetite-regulating hormones, including leptin and ghrelin [35,36]. A handful of studies have suggested that the relationships among sleep loss and several of these mechanisms may be stronger in women than in men [37–39]. For instance, a study in which participants were restricted to 4 h in bed for 5 nights demonstrated increases in morning plasma leptin levels, especially among women [37], and Charles et al. reported a U-shaped relationship between hours of self-reported sleep and plasma leptin among women only in a cross-sectional study of 443 male and female police officers [38]. A recent experiment demonstrated that five nights of partial sleep deprivation (five hours of sleep per night) resulted in increased caloric intake, especially at night, during ad libitum food availability in both men and women, compared to sufficient sleep (nine hours per night) [39]. Interestingly, men gained weight regardless of the sleep condition, while women gained weight only when their sleep was restricted to five hours. They maintained weight during sufficient sleep, leading the authors to suggest that sleep loss may lead to reduced dietary restraint among women [39]. Short and poor quality sleep have been more strongly related to other cardiovascular risk factors including hypertension and peripheral inflammatory markers in women relative to men [40–43]. Because women often report needing more sleep and feeling less rested than men [44,45], it is possible that the physiologic consequences of sleep loss may be more pronounced in women.

Among women in our study, actigraphy-assessed shorter sleep time and decreased SE were related to higher waist circumference, independent of BMI. Disrupted or short sleep may affect the distribution of body fat through activating effects on the hypothalamic–pituitary–adrenal axis, which releases cortisol and contributes to the development of abdominal fat deposition [46–49]. Short or inefficient sleep also may be markers for reduced slow-wave sleep, which was linked to increased waist circumference in a 2010 study of 400 women in Sweden [9]. The fact that actigraphy assessments of sleep were related to waist circumference independent of overall body weight may have important implications for cardiometabolic disease, as increased abdominal adiposity is more closely linked to a heightened cardiac risk profile than is BMI [11]. Indeed, a growing literature shows that the incidence of cardiovascular disease and DM may be elevated among individuals with short or inefficient sleep [50].

Habitual TST as reported on the PSQI was moderately correlated with actigraphy TST. However, participants overestimated their

habitual sleep duration relative to actigraphy by about 40 min, and discrepancies between subjective and objective sleep duration measures were larger among men than women. Overall, these data suggest that self-reported and actigraphy-assessed sleep duration are related but not synonymous constructs, and that the degree of agreement between the two measures may vary depending on tertiary factors. Discrepancies between reported and actigraphy-measured sleep did not differ by BMI, and therefore it is unlikely that weight-related differences in perceptions of sleep influenced results. Actigraphy assessments of sleep were more strongly associated with body weight and waist circumference than self-report. It is possible that aspects of sleep duration captured by actigraphy (i.e., movement throughout the night) are more closely associated with the physiologic mechanisms implicated in regulation of weight than perceptions of duration. It also may be that use of an objective measure across 7 nights reduces some of the error variance associated with a retrospective report of habitual sleep.

Finally, decreased SE was related to higher BMI and waist circumference in women. A small number of studies have reported similar relationships between sleep continuity and BMI [20,24–27]. Actigraphy TST and SE were highly correlated in our study, making their independent contributions difficult to disentangle. However, when both variables were included in statistical models, TST was more strongly linked to BMI, whereas actigraphy SE was more strongly linked to waist circumference. A 2010 study demonstrated that experimental sleep fragmentation across two nights caused alterations in insulin sensitivity, glucose effectiveness, and morning cortisol levels, despite the preservation of TST [51]. Similar metabolic changes have been implicated in overall weight gain and in changes in deep abdominal adipose tissue, among women [52]. In addition, Corbalán-Tutau and Garaulet [53] recently reported that number of awakenings throughout the night, as measured by self-report, co-varied with visceral fat in women with normal weight. Because deficits in sleep duration and efficiency may have different origins, as well as different physiological correlates and consequences, it is important for future studies to examine both variables for their independent links with health.

Our study has a number of important limitations. Prior evidence of sleep as a predictor of weight gain or increases in waist circumference has been inconsistent [54]. For instance, Lauderdale et al. [25] reported that shorter actigraphy sleep time was related to higher BMI in a cross-sectional study, but did not predict weight gain over a 5-year period. Our study could not determine causality, and it is possible that higher BMI caused shorter sleep in women. Because data were drawn from a large epidemiological sample, a number of confounding factors may have influenced the observed associations. One possibility is common genetic variation; a recent study showed that a polymorphism in the circadian locomotor output cycles kaput gene, *CLOCK*, may associate with shorter sleep duration and resistance to weight loss and higher plasma ghrelin concentrations [55]. It also is possible that the observed associations between sleep and anthropometric characteristics partially reflect the influence of sleep-disordered breathing. The prevalence of obstructive sleep apnea is higher among those with higher body weight and central adiposity, both of which contribute to structural defects that compromise airway expansion [56]. We were unable to assess sleep apnea in our study, and instead adjusted for a measure of frequent sleep disruption due to snoring or coughing, which may over-estimate true cases of apnea [57]. Other potential confounding factors include unmeasured medical morbidity and night or shift work, which has been linked to overweight and obesity [58]. We did not adjust for dietary habits, which may mediate the links between sleep disruption and obesity.

There were fewer men than women in the MIDUS cohort, and it is possible that we were unable to observe a relationship between sleep and anthropometric characteristics in men due to a lack of

power. However, all gender-stratified analyses were performed after an interactive effect was detected in the full mixed gender sample, and post hoc power analysis suggests that the sample of men was adequate to detect a small- to medium-sized effects ($f^2 = .08$).

Associations between sleep and BMI or waist circumference were not moderated by menopausal status; however, these analyses were performed on a subset of women due to limited data. As hormonal changes associated with the menopausal transition may contribute to sleep disruption [59] and increases in weight and deposition of abdominal fat [60], further work investigating if the links between sleep and weight are moderated by menopausal status is warranted. Other limitations of our study include use of a self-report measure of physical activity, which likely captured only gross differences in activity level. Use of an activity log or an accelerometer would have provided more valid data. Finally, our sample was predominantly White. Racial/ethnic minorities are disproportionately affected by overweight and obesity [61], and black individuals in particular have shorter and less efficient sleep than white individuals [62]. It will be important to determine if our results generalize to other racial and ethnic groups.

In sum, shorter self-reported sleep was associated with higher BMI in a large sample of adult men and women. Gender moderated associations between actigraphy-assessed sleep and anthropometric characteristics, such that shorter and less efficient sleep were related to higher BMI and waist circumference in women only. Future research on physiologic and behavioral mechanisms linking sleep and body weight may elucidate gender differences in vulnerability to sleep loss and reveal if certain pathways are more relevant in men vs women.

Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: <http://dx.doi.org/10.1016/j.sleep.2013.08.784>.

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References

- [1] Cappuccio FP, Taggart FM, Kandala NB, et al. Meta-analysis of short sleep duration and obesity in children and adults. *Sleep* 2008;31(5):619–26.
- [2] Chaput JP, Despres JP, Bouchard C, et al. Short sleep duration preferentially increases abdominal adiposity in adults: preliminary evidence. *Clin Obes* 2011;1(4–6):141–6.
- [3] de Koning L, Merchant AT, Pogue J, et al. Waist circumference and waist-to-hip ratio as predictors of cardiovascular events: meta-regression analysis of prospective studies. *Eur Heart J* 2007;28:850–6.
- [4] Jacobs EJ, Newton CC, Wang Y, et al. Waist circumference and all-cause mortality in a large US cohort. *Arch Intern Med* 2010;170:1293–301.
- [5] Klein S, Allison DB, Heymsfield SB, et al. Waist Circumference and Cardiometabolic Risk: a Consensus Statement from Shaping America's Health: Association for Weight Management and Obesity Prevention; NAASO, the Obesity Society; the American Society for Nutrition; and the American Diabetes Association. *Obesity (Silver Spring)* 2007;15:1061–7.
- [6] Ko GT, Chan JC, Chan AW, et al. Association between sleeping hours, working hours and obesity in Hong Kong Chinese: the 'better health for better Hong Kong' health promotion campaign. *Int J Obes (Lond)* 2007;31:254–60.
- [7] Najafian J, Mohammadifard N, Siadat ZD, et al. Association between sleep duration and body mass index and waist circumference. *Iran J Med Sci* 2010;35:140–4.
- [8] Stranges S, Cappuccio FP, Kandala NB, et al. Cross-sectional versus prospective associations of sleep duration with changes in relative weight and body fat distribution: the Whitehall II Study. *Am J Epidemiol* 2008;167:321–9.
- [9] Theorell-Haglow J, Berne C, Janson C, et al. Associations between short sleep duration and central obesity in women. *Sleep* 2010;33:593–8.
- [10] Wang Y, Rimm EB, Stampfer MJ, et al. Comparison of abdominal adiposity and overall obesity in predicting risk of type 2 diabetes among men. *Am J Clin Nutr* 2005;81:555–63.
- [11] Zhu S, Wang Z, Heshka S, et al. Waist circumference and obesity-associated risk factors among whites in the third National Health and Nutrition Examination Survey: clinical action thresholds. *Am J Clin Nutr* 2002;76:743–9.
- [12] Manber R, Gress J, Baker F. Sex differences in sleep and sleep disorders: a focus on women's sleep. *Int J Sleep Disord* 2006;1:7–15.
- [13] Cournot M, Ruidavets JB, Marquie JC, et al. Environmental factors associated with body mass index in a population of Southern France. *Eur J Cardiovasc Prev Rehabil* 2004;11:291–7.
- [14] St-Onge MP, Perumean-Chaney S, Desmond R, et al. Gender differences in the association between sleep duration and body composition: the Cardia Study. *Int J Endocrinol* 2010;2010:726071.
- [15] Lopez-Garcia E, Faubel R, Leon-Munoz L, et al. Sleep duration, general and abdominal obesity, and weight change among the older adult population of Spain. *Am J Clin Nutr* 2008;87:310–6.
- [16] Meyer KA, Wall MM, Larson NI, et al. Sleep duration and BMI in a sample of young adults. *Obesity (Silver Spring)* 2012;20:1279–87.
- [17] Watanabe M, Kikuchi H, Tanaka K, et al. Association of short sleep duration with weight gain and obesity at 1-year follow-up: a large-scale prospective study. *Sleep* 2010;33:161–7.
- [18] Yi S, Nakagawa T, Yamamoto S, et al. Short sleep duration in association with CT-scanned abdominal fat areas: the Hitachi Health Study. *Int J Obes (Lond)* 2012;37:129–34.
- [19] Chaput JP, Despres JP, Bouchard C, et al. Short sleep duration is associated with reduced leptin levels and increased adiposity: results from the Quebec family study. *Obesity (Silver Spring)* 2007;15:253–61.
- [20] van den Berg JF, Knvistingh Neven A, Tulen JH, et al. Actigraphic sleep duration and fragmentation are related to obesity in the elderly: the Rotterdam Study. *Int J Obes (Lond)* 2008;32:1083–90.
- [21] Vorona RD, Winn MP, Babineau TW, et al. Overweight and obese patients in a primary care population report less sleep than patients with a normal body mass index. *Arch Intern Med* 2005;165:25–30.
- [22] Lauderdale DS, Knutson KL, Yan LL, et al. Self-reported and measured sleep duration: how similar are they? *Epidemiology* 2008;19:838–45.
- [23] Lockley SW, Skene DJ, Arendt J. Comparison between subjective and actigraphic measurement of sleep and sleep rhythms. *J Sleep Res* 1999;8:175–83.
- [24] Javaheri S, Storfer-Isser A, Rosen CL, et al. Sleep quality and elevated blood pressure in adolescents. *Circulation* 2008;118:1034–40.
- [25] Lauderdale DS, Knutson KL, Rathouz PJ, et al. Cross-sectional and longitudinal associations between objectively measured sleep duration and body mass index: the CARDIA Sleep Study. *Am J Epidemiol* 2009;170:805–13.
- [26] Tworoger SS, Davis S, Vitiello MV, et al. Factors associated with objective (actigraphic) and subjective sleep quality in young adult women. *J Psychosom Res* 2005;59:11–9.
- [27] Vgontzas AN, Bixler EO, Tan TL, et al. Obesity without sleep apnea is associated with daytime sleepiness. *Arch Intern Med* 1998;158:1333–7.
- [28] Vitiello MV, Larsen LH, Moe KE. Age-related sleep change: gender and estrogen effects on the subjective-objective sleep quality relationships of healthy, noncomplaining older men and women. *J Psychosom Res* 2004;56:503–10.
- [29] Buysse DJ, Reynolds 3rd CF, Monk TH, et al. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res* 1989;28:193–213.
- [30] Radloff LS. The CES-D scale: a self-report depression scale for research in the general population. *Appl Psychol Meas* 1977;1:385–401.
- [31] Imaki M, Hatanaka Y, Ogawa Y, et al. An epidemiological study on relationship between the hours of sleep and life style factors in Japanese factory workers. *J Physiol Anthropol Appl Human Sci* 2002;21:115–20.
- [32] Kim S, DeRoo LA, Sandler DP. Eating patterns and nutritional characteristics associated with sleep duration. *Public Health Nutr* 2011;14:889–95.
- [33] Nishiura C, Noguchi J, Hashimoto H. Dietary patterns only partially explain the effect of short sleep duration on the incidence of obesity. *Sleep* 2010;33:753–7.
- [34] Weiss A, Xu F, Storfer-Isser A, et al. The association of sleep duration with adolescents' fat and carbohydrate consumption. *Sleep* 2010;33:1201–9.
- [35] Schmid SM, Hallschmid M, Jauch-Chara K, et al. A single night of sleep deprivation increases ghrelin levels and feelings of hunger in normal-weight healthy men. *J Sleep Res* 2008;17:331–4.
- [36] Spiegel K, Leproult R, Tasali E, et al. Sleep curtailment results in decreased leptin levels, elevated ghrelin levels and increased hunger and appetite. *Annals Int Med* 2004;141:846–50.
- [37] Simpson NS, Banks S, Dinges DF. Sleep restriction is associated with increased morning plasma leptin concentrations, especially in women. *Biol Res Nurs* 2010;12:47–53.
- [38] Charles LE, Gu JK, Andrew ME, et al. Sleep duration and biomarkers of metabolic function among police officers. *J Occup Environ Med* 2011;53:8317.
- [39] Markwald RR, Melanson EL, Smith MR, et al. Impact of insufficient sleep on total daily energy expenditure, food intake, and weight gain. *PNAS* 2013;110:5695–700.
- [40] Cappuccio FP, Stranges S, Kandala NB, et al. Gender-specific associations of short sleep duration with prevalent and incident hypertension: the Whitehall II Study. *Hypertension* 2007;50:693–700.

- [41] Miller MA, Kandala NB, Kivimaki M, et al. Gender differences in the cross-sectional relationships between sleep duration and markers of inflammation: Whitehall II Study. *Sleep* 2009;32:857–64.
- [42] Stranges S, Dorn JM, Cappuccio FP, et al. A population-based study of reduced sleep duration and hypertension: the strongest association may be in premenopausal women. *J Hypertens* 2010;28:896–902.
- [43] Suarez EC. Self-reported symptoms of sleep disturbance and inflammation, coagulation, insulin resistance and psychosocial distress: evidence for gender disparity. *Brain Behav Immun* 2008;22:960–8.
- [44] Baldwin CM, Kapur VK, Holberg CJ, et al. Associations between gender and measures of daytime somnolence in the Sleep Heart Health Study. *Sleep* 2004;27:305–11.
- [45] National Sleep Foundation. *Sleep in America poll: summary of findings*. Washington, DC: National Sleep Foundation; 2012.
- [46] Bjorntorp P. The regulation of adipose tissue distribution in humans. *Int J Obes Relat Metab Disord* 1996;20:291–302.
- [47] Pedersen SB, Jonler M, Richelsen B. Characterization of regional and gender differences in glucocorticoid receptors and lipoprotein lipase activity in human adipose tissue. *J Clin Endocrinol Metab* 1994;78:1354–9.
- [48] Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. *Lancet* 1999;354:1435–9.
- [49] Spiegel K, Leproult R, L'hermite-Baleriaux M, et al. Leptin levels are dependent on sleep duration: relationships with sympathovagal balance, carbohydrate regulation, cortisol, and thyrotropin. *J Clin Endocrinol Metab* 2004;89:5762–71.
- [50] Mezick EJ, Hall M, Matthews KA. Are sleep and depression independent or overlapping risk factors for cardiometabolic disease? *Sleep Med Rev* 2011;15:51–63.
- [51] Stamatakis KA, Punjabi NM. Effects of sleep fragmentation on glucose metabolism in normal subjects. *Chest* 2010;137:95–101.
- [52] Gower BA, Hunter GR, Chandler-Laney PC, et al. Glucose metabolism and diet predict changes in adiposity and fat distribution in weight-reduced women. *Obesity* 2010;18:1532–7.
- [53] Corbalan-Tutau MD, Madrid JA, Garaulet M. Timing and duration of sleep and meals in obese and normal weight women. Association with increase blood pressure. *Appetite* 2012;59:9–16.
- [54] Magee L, Hale L. Longitudinal associations between sleep duration and subsequent weight gain: a systematic review. *Sleep Med Rev* 2012;16:231–41.
- [55] Garaulet M, Sanchez-Moreno C, Smith CE, et al. Ghrelin, sleep reduction and evening preference: relationships to CLOCK 3111 T/C SNP and weight loss. *PLoS One* 2011;6:e17435.
- [56] Schwartz AR, Patil SP, Laffan AM, et al. Obesity and obstructive sleep apnea: pathogenic mechanisms and therapeutic approaches. *Proc Am Thorac Soc* 2008;5:185–92.
- [57] Young T, Palta M, Dempsey J, Peppard PE, et al. Burden of sleep apnea: rationale, design, and major findings of the Wisconsin Sleep Cohort study. *WMJ* 2009;108:2469.
- [58] Antunes LC, Levandovski R, Dantas G, et al. Obesity and shift work: chronobiological aspects. *Nutr Res Rev* 2010;23:155–68.
- [59] Trenell MI, Marshall NS, Rogers NL. Sleep and metabolic control: waking to a problem? *Clin Exp Pharmacol Physiol* 2007;34:19.
- [60] Lee CG, Carr MC, Murdoch SJ, et al. Adipokines, inflammation, and visceral adiposity across the menopausal transition: a prospective study. *J Clin Endocrinol Metab* 2009;94:110410.
- [61] Wang Y, Beydoun MA. The obesity epidemic in the United States—gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and metaregression analysis. *Epidemiol Rev* 2007;29:6–28.
- [62] Ruiter ME, Decoster J, Jacobs L, et al. Normal sleep in African-Americans and Caucasian-Americans: a meta-analysis. *Sleep Med* 2011;12:209–14.