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ORIGINAL ARTICLE

The association between sleep duration and obesity in older adults

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Background: Reduced sleep has been reported to predict obesity in children and young adults. However, studies based on self-report have been unable to identify an association in older populations. In this study, the cross-sectional associations between sleep duration measured objectively and measures of weight and body composition were assessed in two cohorts of older adults.
Methods: Wrist actigraphy was performed for a mean (s.d.) of 5.2 (0.9) nights in 3055 men (age: 67–96 years) participating in the Osteoporotic Fractures in Men Study (MrOS) and 4.1 (0.8) nights in 3052 women (age: 70–99 years) participating in the Study of Osteoporotic Fractures (SOF). A subgroup of 2862 men and 455 women also underwent polysomnography to measure sleep apnea severity.
Results: Compared to those sleeping an average of 7–8 h per night, and after adjusting for multiple risk factors and medical conditions, a sleep duration of less than 5 h was associated with a body mass index (BMI) that was on average 2.5 kg/m^2 (95% confidence interval (CI): 2.0–2.9) greater in men and 1.8 kg/m^2 (95% CI: 1.1–2.4) greater in women. The odds of obesity (BMI $\geqslant$30 kg/m^2) was 3.7-fold greater (95% CI: 2.7–5.0) in men and 2.3-fold greater in women (95% CI: 1.6–3.1) who slept less than 5 h. Short sleep was also associated with central body fat distribution and increased percent body fat. These associations persisted after adjusting for sleep apnea, insomnia and daytime sleepiness.
Conclusions: In older men and women, actigraphy-ascertained reduced sleep durations are strongly associated with greater adiposity.
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Keywords: sleep duration; sleep deprivation; central obesity; geriatrics; insomnia; sleepiness

Introduction

Obesity has become a global epidemic. The prevalence of adult obesity in the United States has more than doubled from 15% in the late 1970s to 31% by 2000.[1] Parallel to the increase in weight has been a reduction in sleep times. Only 35% of American adults reported obtaining 8 h of sleep in 1998 and that number had further fallen to 26% by 2005.[2]

Multiple studies of children and young adults have observed an association between reduced sleep time and increased weight, suggesting that those who report sleeping less are more likely to be obese.[3–6] However, critical reviews of this literature have pointed out substantial weaknesses in this evidence including the overwhelming reliance on self-report for sleep duration, inadequate length of monitoring when objective measurements of sleep were performed and inadequate control for important potential confounders

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such as sleep apnea and depression that can adversely impact both sleep duration and weight.[7–9] In addition, several studies have suggested that the association between short sleep and obesity does not exist or weakens at older ages.[10,11] However, the reliance on self-report to assess sleep duration may lead to substantial misclassification, particularly in older populations where sleep disorders such as insomnia and sleep apnea are common leading to misperception of time spent asleep. A recent study using objective measurement of sleep duration over multiple nights in the elderly did find a strong association between reduced sleep and obesity, while no association was found in this same cohort when reduced sleep was defined based on self-report.[12]

The prevalence of obesity in those over 60 is similar to the overall adult prevalence.[13] Older populations also suffer from a high prevalence of diabetes, hypertension and heart disease, conditions which are causally linked to, or exacerbated by, obesity. Furthermore, sleep difficulties increase in prevalence with age making reduced sleep durations potentially much more common in this group. Using cross-sectional data derived from two multi-center observational studies in older populations, we performed one of the first assessments of the relationship between objectively measured sleep duration and obesity in an older population with detailed evaluation of whether any identified association is independent of sleep apnea, daytime sleepiness and insomnia.

Materials and methods

Study population

Men were participants in the prospective Osteoporotic Fractures in Men Study (MrOS). During the baseline examination from 2000 to 2002, 5995 community-dwelling men 65 years or older were enrolled at six clinical centers in the United States: Birmingham, Alabama; Minneapolis, Minnesota; Palo Alto, California; Pittsburgh, Pennsylvania; Portland, Oregon; and San Diego, California. The MrOS Sleep Study, an ancillary study of the parent MrOS cohort, was conducted between December 2003 and March 2005 and recruited 3135 of these participants (exceeding the goal of 3000 participants) for a comprehensive sleep assessment of whom 3110 underwent actigraphy. Of the 2860 participants who did not participate in the sleep study; 1997 were unwilling, 332 were not screened because recruitment goals were met, 344 died before the sleep study visit, 150 were ineligible because of exclusion criteria and 37 withdrew from the study before the sleep visit.

Women were participants in the prospective Study of Osteoporotic Fractures (SOF). During the baseline examination from 1986 to 1988, 9704 community-dwelling white women 65 years or older were enrolled from population-based listings in four areas of the United States: Baltimore, Maryland; Minneapolis, Minnesota; Pittsburgh, Pennsylvania; and Portland, Oregon. An additional 662 African-American women were enrolled between 1997 and 1998. Assessment of sleep occurred during the eighth examination in this cohort, which took place from January 2002 to April 2004. A total of 4727 women (84% of survivors) participated, of whom 3676 had a clinical visit and 3219 underwent actigraphy.

For both studies, individuals were not eligible to participate if they reported bilateral hip replacement or required the assistance of another person in ambulation at the baseline examination. Details of the cohorts have been published.[14–16] The protocol for MrOS and SOF were approved by the institutional review boards at all of the participating institutions. All participants provided written informed consent.

Sleep duration

Average nightly sleep duration was obtained using wrist actigraphy (Sleepwatch-O, Ambulatory Monitoring Inc., Ardsley, NY, USA) in both cohorts. The actigraph, which measures acceleration using a piezoelectric biomorph-ceramic cantilevered beam, was worn on the wrist of the non-dominant hand. Patients were asked to wear the actigraph for a minimum of five nights in MrOS and three nights in SOF. Average use (s.d.) was 5.2 (0.9) nights in MrOS and 4.1 (0.8) nights in SOF. Data were collected continuously and stored in 1-min epochs. The digital integration mode of analysis, which sums the absolute level of acceleration on a second-by-second basis over the epoch, was used to quantify the amount of movement in each minute from which sleep–wake status could be inferred. This technique for estimating sleep duration has been validated against polysomnography in these cohorts.[17,18] Action W-2 software (Ambulatory Monitoring, Inc.) was used to analyze the raw data,[19] and the University of California San Diego scoring algorithm was used to determine sleep–wake status.[20]

While wearing the actigraph, participants completed sleep diaries, which included time into and time out of bed and times the actigraph was removed. This information was used in editing the actigraphy data files to set intervals for when the patiant was in bed, and to delete time when the actigraph was removed from analyses. Actigraphy is most accurate in distinguishing wake from sleep at night; thus, nocturnal sleep duration (amount of sleep obtained between time to bed and time out of bed) was used as the primary measure of sleep duration (averaged across all nights of measurement). Inter-scorer reliability for this measure has been previously found to be high in our group (intra-class coefficient = 0.95) and this measure has been shown to have good concordance with total sleep time from polysomnography.[12,21] Nocturnal sleep duration was categorized into <5, 5 to <7, 7 to <8 and $\geqslant 8$ h. Secondary analyses were conducted using the total amount of sleep across a 24-h period from 0900 to 0900 hours, again averaging over all days of measurement. Because few patients had a 24-h sleep time below 5 h when napping was included, the categories used for these analyses were <6, 6 to <7, 7 to <8 and $\geqslant 8$ h.

Because of potential differences between weeknight and weekend sleep patterns, secondary analyses were performed limiting sleep data to that collected on weeknights (Sunday to Thursday nights).

Obesity and body fat measures
During the home or clinic visits, body weight was measured with a standard balance beam or digital scale, height with a wall-mounted Harpenden stadiometer (Holtain, England), and these measurements were used to calculate body mass index (BMI). Obesity was defined as a BMI $\geqslant$30 kg/m^2. Circumferences at the hip and waist were also measured in a standardized manner for all men in MrOS and for the subset of women in SOF who underwent polysomnography (see below). In the MrOS cohort, percent body fat and percent lean mass were obtained for the entire body as well as specific to the trunk using dual energy X-ray absoptiometry (Hologic QDR-4500 W, Bedford, MA, USA).

Covariates
All participants completed questionnaires, which included items about demographics, medical history, physical activity, smoking and alcohol use. Caffeine consumption was estimated based on self-report of the average daily number of cups of caffeinated coffee and tea or cans of caffeinated soda consumed. Participants were asked to bring in all current medications used within the preceding 30 days. All prescription medications were entered into an electronic database and each medication was matched to its ingredient(s) based on the Iowa Drug Information Service Drug Vocabulary (College of Pharmacy, University of Iowa, Iowa City, IA, USA).[22] The Geriatric Depression Scale (GDS) was used to assess depressive symptoms, and the standard cutoff of six or more symptoms was used to define depression.[23] In MrOS, the level of activity was assessed using the Physical Activity Scale for the elderly,[24] whereas in SOF, physical activity was assessed by asking women if they walked for exercise.

Self-reported information about sleepiness was assessed using the Epworth Sleepiness Scale (ESS).[25] An ESS $>$10 was used to define excessive daytime sleepiness.[26] The Pittsburgh Sleep Quality Index (PSQI) was used to assess sleep quality.[27] Insomnia was defined as either taking more than 30 min to fall asleep or waking up in the middle of the night three or more times a week based on responses to the PSQI.

In-home sleep studies using unattended polysomnography (SOF: Siesta unit; MrOS: Safiro unit; Compumedics, Abbotsford, Australia) were performed in 2956 MrOS participants and a subset of 461 women in SOF recruited from two clinical centers (Minneapolis and Pittsburgh). The recording montage was identical and included C3/A2 and C4/A1 electroencephalography, bilateral electrooculography, submental electromyography, thoracic and abdominal respiratory effort, airflow (nasal-oral thermocouple and nasal pressure), finger pulse oximetry, electrocardiography, body position and bilateral leg movements. Sleep staging was carried out using standard criteria.[28] Apneas were defined as a complete or almost complete cessation of airflow for more than 10 s. Hypopneas were defined as a $>$30% reduction in amplitude of either respiratory effort or airflow for more than 10 s associated with a $\geqslant$3% oxygen desaturation. The apnea hypopnea index (AHI) was computed as the average number of apneas and hypopneas per hour of recorded sleep. An AHI $\geqslant$15 was used to define moderate-to-severe sleep apnea.[29]

Statistical analyses
Each cohort was analyzed separately but in parallel manner. Baseline characteristics were summarized by category of sleep duration at night and differences were compared using analysis of variance for normally distributed continuous data, Kruskal–Wallis tests for continuous skewed data and χ^2 tests for categorical data.

Multivariate regression models were used to assess the independent relationship between sleep duration measures (average sleep duration at night and average sleep duration over 24 h) with each of the weight-related outcomes using linear regression for continuous outcomes and logistic regression for the dichotomous outcome of obesity. Covariates were selected based on being associated with both sleep duration and obesity in prior studies. All models included study site, age, race (Caucasian vs non-Caucasian), level of education ($<$12, 12–16, $>$16 years), history of diabetes, stroke, heart disease (angina, myocardial infarction, heart failure, coronary revascularization procedure or pacemaker placement), use of antidepressants, use of benzodiazepines, smoking status (never, past, current), alcohol consumption (continuous), caffeine consumption (continuous), current depression (by GDS) and level of physical activity (continuous in MrOS and dichotomous in SOF) as covariates. Additional analyses adjusted for AHI to control for sleep apnea severity. Evidence for effect modification was assessed by stratifying analyses based on sleepiness or insomnia symptoms as well as by assessing the significance of an interaction term with sleep duration. All analyses were performed using SAS statistical software (version 9.1, SAS Institute Inc., Cary, NC, USA).

Results

A total of 3055 men (mean age 76.4 years) and 3052 women (mean age 83.6 years) had actigraphic data of sufficient quality to be included in these analyses (98 and 95% of the sample studied respectively). On average, nocturnal sleep duration (mean ± s.d.) was 384 ± 74 min for the men in MrOS and 405 ± 78 min for the women in SOF. In these elderly cohorts, mean sleep duration was similar on weeknights vs weekends (381 vs 390 min in MrOS and 404 vs 407 min in SOF). Baseline data on each cohort by nocturnal sleep duration are shown for men in Table 1 and for women in Table 2. Among both men and women, short sleepers (<5 h) were more likely to have co-morbidities. Antidepressant use was more common among both short and long sleepers. Among men, short sleep was associated with lower levels of education and greater rates of cigarette smoking. In both genders, short sleep duration was associated with increased prevalence of sleep apnea and symptoms of sleepiness. In contrast, no clear association was found between sleep duration and insomnia symptoms.

In both men and women, mean BMI was greatest in those sleeping the least (Figure 1). In multivariable analyses, mean BMI was 2.48 kg/m^2 (95% CI: (2.02, 2.93)) greater in men and 1.75 kg/m^2 (1.09, 2.42) greater in women among those sleeping less than 5 h compared to those with 7–8 h of sleep ($P<0.0001$ for both men and women). Short sleepers were also found to have greater waist circumference (Figure 2). Relative to 7–8 h sleepers, men sleeping less than 5 h had a 6.7 cm (5.4, 8.0) greater waist circumference, while in women this difference was 5.4 cm (1.0, 9.9) ($P<0.0001$ for men and $P<0.02$ for women). Hip circumference was 4.7 cm (3.7, 5.7) and 5.0 cm (1.0, 8.9) greater in short sleepers in men and women, respectively ($P<0.0001$ for men and $P<0.02$ for women).

Results from men in MrOS, for whom dual energy X-ray absoptiometry measures of total and body regional fat were obtained, suggested the increases in these measures were due to increases in fat mass (Figure 3). Percent body fat was 1.7% (1.1, 2.4) greater and percent body fat in the trunk was 1.9% (1.1, 2.7) greater in those sleeping less than 5 h compared to

Table 1 Demographic characteristics among men in the MrOS cohort by sleep duration

	Sleep duration (h)				P-*value*
	<5 (n=376)	*5 to <7 (n=1713)*	*7 to <8 (n=742)*	*⩾8 (n=224)*	
Age (years)	76.5 ± 5.7	76.1 ± 5.4	76.5 ± 5.7	77.8 ± 5.9	0.0003
Caucasian race	326 (86.7)	1538 (89.8)	681 (91.8)	204 (91.1)	0.06
Education					0.005
High school or less	104 (27.7)	367 (21.4)	129 (17.4)	51 (22.8)	
Some college or college graduate	152 (40.4)	687 (40.1)	320 (43.1)	93 (41.5)	
Graduate school	120 (31.9)	659 (38.5)	293 (39.5)	80 (35.7)	
History of					
Diabetes mellitus	68 (18.1)	220 (12.9)	93 (12.5)	29 (13.0)	0.04
Heart disease	147 (39.2)	558 (32.7)	239 (32.2)	70 (31.3)	0.07
Stroke	17 (4.5)	55 (3.2)	30 (4.0)	12 (5.4)	0.29
Alcohol use					0.11
Non-user	146 (39.1)	592 (34.7)	229 (31.0)	87 (39.4)	
<1 drink per week	37 (9.9)	222 (13.0)	89 (12.0)	23 (10.4)	
1–2 drinks per week	43 (11.5)	218 (12.8)	94 (12.7)	22 (10.0)	
3–5 drinks per week	54 (14.5)	266 (15.6)	120 (16.2)	31 (14.0)	
6–13 drinks per week	70 (18.8)	325 (19.1)	163 (22.0)	38 (17.2)	
14 or more drinks per week	23 (6.2)	83 (4.9)	45 (6.1)	20 (9.1)	
Caffeine intake, mg day^{-1}	261 ± 266	237 ± 249	229 ± 241	201 ± 196	0.15
Cigarette smoking					0.001
Never smoker	124 (33.0)	689 (40.2)	295 (39.8)	97 (43.3)	
Past smoker	234 (62.2)	994 (58.0)	434 (58.5)	125 (55.8)	
Current smoker	18 (4.8)	30 (1.8)	13 (1.8)	2 (0.9)	
PASE Score	140 ± 75	150 ± 72	140 ± 69	140 ± 71	0.002
Depression	31 (8.2)	100 (5.9)	55 (7.4)	18 (8.0)	0.20
Current use of benzodiazepines	19 (5.1)	67 (3.9)	37 (5.0)	15 (6.7)	0.21
Current use of antidepressants	35 (9.3)	122 (7.1)	53 (7.1)	31 (13.8)	0.003
Apnea hypopnea index	23.8 ± 18.6	16.7 ± 14.4	15.1 ± 14.1	15.5 ± 14.2	<0.0001
Moderate to severe sleep apnea	196 (57.0)	701 (43.8)	263 (37.4)	84 (39.6)	<0.0001
Epworth Sleepiness Scale (range 0–24)	7.5 ± 4.2	6.3 ± 3.6	5.6 ± 3.4	4.9 ± 3.6	<0.0001
Excessive daytime sleepiness	83 (22.1)	218 (12.7)	71 (9.6)	21 (9.4)	<0.0001
Insomnia	239 (63.6)	1051 (61.4)	468 (63.1)	147 (65.6)	0.55

Summary data expressed as mean ± s.d. for continuous variables and *N* (%) for categorical variables. Moderate-to-severe sleep apnea defined as an apnea hypopnea index ⩾15.[29] Excessive daytime sleepiness defined as Epworth Sleepiness Scale >10.[26] Insomnia defined as reporting either sleep onset or sleep maintenance difficulties occurring three or more times a week on the Pittsburgh Sleep Quality Index. MrOS, Osteoporotic Fractures in Men Study; PASE, Physical Activity Score for the Elderly.

Table 2 Demographic characteristics among women in the SOF cohort by sleep duration

	Sleep duration (h)				P-*value*
	<5 (n=*265*)	*5 to <7* (n=*1449*)	*7 to <8* (n=*908*)	*⩾8* (n=*430*)	
Age (years)	83.5±4.3	83.3±3.6	83.6±3.7	84.5±4.1	<0.0001
Caucasian race	219 (82.6)	1275 (88.0)	837 (92.2)	396 (92.1)	<0.0001
Education					0.14
High school or less	164 (61.9)	857 (59.1)	554 (61.1)	250 (58.3)	
Some college or college graduate	66 (24.9)	432 (29.8)	259 (28.6)	144 (33.6)	
Graduate school	35 (13.2)	160 (11.0)	94 (10.4)	35 (8.2)	
History of					
Diabetes mellitus	44 (16.6)	170 (11.8)	80 (8.8)	45 (10.5)	0.004
Heart disease	109 (41.1)	473 (32.7)	272 (30.0)	153 (35.6)	0.005
Stroke	48 (18.1)	173 (12.0)	112 (12.4)	69 (16.1)	0.01
Alcohol use					0.14
Non-user	174 (65.7)	848 (58.6)	517 (57.1)	260 (60.5)	
Once in last 30 days	25 (9.4)	147 (10.2)	94 (10.4)	42 (9.8)	
2–3 times in last 30 days	30 (11.3)	197 (13.6)	117 (12.9)	53 (12.3)	
1–2 days per week	9 (3.4)	90 (6.2)	68 (7.5)	26 (6.1)	
3–4 day per week	3 (1.1)	62 (4.3)	39 (4.3)	10 (2.3)	
5–6 days per week	7 (2.6)	37 (2.6)	22 (2.4)	8 (1.9)	
Every day	17 (6.4)	66 (4.6)	49 (5.4)	31 (7.2)	
Caffeine intake (mg day^{-1})	198±230	196±213	198±213	173±187	0.45
Cigarette smoking					0.10
Never smoker	150 (56.8)	947 (65.4)	569 (62.7)	273 (63.5)	
Past smoker	101 (38.3)	464 (32.0)	315 (34.7)	146 (34.0)	
Current smoker	13 (4.9)	38 (2.6)	23 (2.5)	11 (2.6)	
Walks for exercise	63 (24.1)	555 (38.9)	358 (39.9)	145 (34.0)	<0.0001
Depression	32 (12.1)	161 (11.1)	100 (11.0)	68 (15.9)	0.05
Current use of benzodiazepines	20 (7.6)	100 (6.9)	63 (7.0)	39 (9.1)	0.47
Current use of antidepressants	52 (19.6)	165 (11.4)	105 (11.6)	95 (22.1)	<0.0001
Apnea hypopnea index	24.0±18.4	16.1±15.5	14.2±14.2	13.4±11.6	0.002
Moderate-to–severe sleep apnea	23 (63.9)	87 (39.6)	49 (33.8)	15 (27.8)	0.003
Epworth sleepiness scale (range 0–24)	7.0±4.5	6.0±3.8	5.0±3.5	4.9±3.7	<0.0001
Excessive daytime sleepiness	53 (20.0)	183 (12.7)	68 (7.5)	32 (7.4)	<0.0001
Insomnia	135 (50.9)	720 (49.8)	475 (52.4)	243 (56.5)	0.09

Summary data expressed as mean±s.d. for continuous variables and *N* (%) for categorical variables. Moderate-to-severe sleep apnea defined as an apnea hypopnea index ⩾15.[29] Excessive daytime sleepiness defined as Epworth Sleepiness Scale >10.[26] Insomnia defined as reporting either sleep onset or sleep maintenance difficulties occurring three or more times a week on the Pittsburgh Sleep Quality Index. SOF, Study of Osteoporotic Fractures.

those with 7–8 h of sleep ($P<0.0001$ for both comparisons). In fact, the relationship between percent body fat and sleep duration exhibited a U-shaped association such that compared to those with 7–8 h of sleep, men sleeping 8 h or more had 0.9% (0.1, 1.7) greater overall percent body fat ($P=0.03$) and 1.2% (0.3, 2.1) greater percent body fat in the trunk ($P=0.01$).

Short sleep durations were also associated with increased likelihood of obesity (Table 3). Relative to those sleeping 7–8 h, men sleeping less than 5 h were 3.7 (2.7, 5.0) times more likely to be obese and women sleeping less than 5 h were 2.3 (1.6, 3.1) times more likely to be obese. The odds ratios (ORs) for obesity associated with sleeping 5–7 h were 1.5 (1.2, 1.9) and 1.3 (1.0, 1.6) for men and women, respectively.

To assess whether the association between sleep duration and obesity was explained by sleep apnea, secondary analyses were performed adjusting for AHI (Table 3). Among men, the odds of obesity increased 1.4-fold (1.3, 1.5) with every 10 unit increase in AHI. Adjusting for AHI attenuated the association between short sleep and obesity by 18%; however, a significant association persisted. Mean BMI was 2.03 kg/m^2 (1.57, 2.48) greater and the OR for obesity was 3.0 (2.2, 4.2) in those sleeping less than 5 h compared to 7–8 h sleepers. Additional analyses were performed restricting to those individuals with an AHI <15. In this subgroup, short sleep remained associated with greater adiposity. Among men, mean BMI was 1.99 kg/m^2 (1.36, 2.63) greater among short sleepers and the OR for obesity in short sleepers was 2.7 (1.6, 4.5). Although the sample size was much smaller in SOF

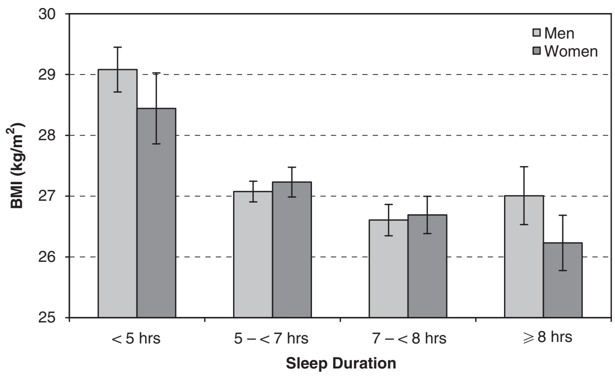

Figure 1 Association between body mass index and sleep duration among older men and women. The adjusted mean body mass index with 95% confidence interval is displayed by nocturnal sleep duration for male participants in MrOS and female participants in SOF. Analyses are adjusted for study site, age, race, level of education, smoking status, alcohol and caffeine consumption, use of benzodiazepines and antidepressants, depression, physical activity, history of diabetes, heart disease and stroke. MrOS: Osteoporotic Fractures in Men Study; SOF: Study of Osteoporotic Fractures.

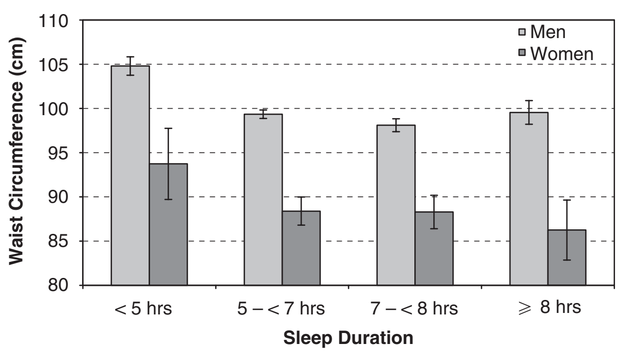

Figure 2 Association between waist circumference and sleep duration among older men and women. The adjusted mean waist circumference with 95% confidence interval is displayed by nocturnal sleep duration for male participants in MrOS and female participants in SOF. Analyses are adjusted for study site, age, race, level of education, smoking status, alcohol and caffeine consumption, use of benzodiazepines and antidepressants, depression, physical activity, history of diabetes, heart disease and stroke. MrOS: Osteoporotic Fractures in Men Study; SOF: Study of Osteoporotic Fractures.

due to the limited number of sleep studies performed, the patterns were very similar in women (that is, the association between short sleep and obesity was attenuated by 14% after adjusting for AHI).

Among men, neither daytime sleepiness nor insomnia symptoms were associated with obesity after controlling for sleep duration. Stratified analyses found similar associations between short sleep and obesity in men with and without excessive daytime sleepiness and with and without insomnia (Table 4). Among women, sleepiness was also not associated with obesity but insomnia was an independent predictor with an OR of 1.3 (1.1, 1.6). In stratified analyses, the association between short sleep and obesity appeared

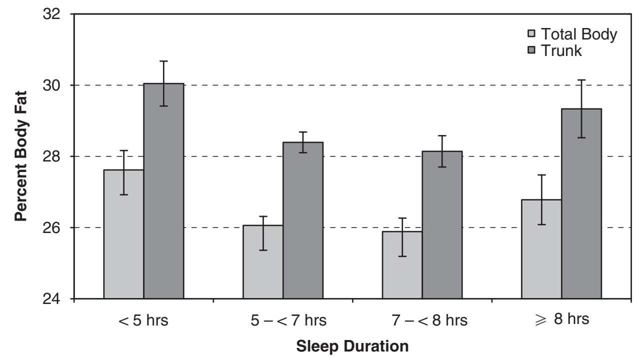

Figure 3 Association between percent body fat and sleep duration among older men. The adjusted mean percent fat mass with 95% confidence interval for whole body and trunk by nocturnal sleep duration in male participants of the Osteoporotic Fractures in Men Study. Analyses are adjusted for study site, age, race, level of education, smoking status, alcohol and caffeine consumption, use of benzodiazepines and antidepressants, depression, physical activity, history of diabetes, heart disease and stroke.

stronger among women with daytime sleepiness and women with insomnia. However, formal statistical testing of an interaction between daytime sleepiness and sleep duration or insomnia and sleep duration was not significant.

Using total sleep time over the 24-h period as the sleep duration measure instead of nocturnal sleep time had no substantial effect on the relationships found. In multivariate models, mean BMI was 1.54 kg/m^2 greater (1.10, 1.97) among men and 1.23 kg/m^2 greater (0.56, 1.90) among women with a 24-h sleep duration less than 6 h compared to 7–8 h ($P<0.0001$ for men and $P<0.001$ for women). The ORs for obesity in those sleeping less than 6 h relative to those sleeping 7–8 h were 2.4 (1.8, 3.2) and 1.7 (1.2, 2.4) in men and women, respectively. The association between nocturnal sleep duration and obesity measures was similar when restricted to data obtained on weeknights (data not shown).

Discussion

In both the MrOS and SOF cohorts, we found that older men and women with reduced amounts of sleep as measured by actigraphy had an elevated BMI. Sleeping 5 or fewer hours per night was associated with a 3.7-fold greater odds of obesity among men and 2.3-fold increase among women compared to those sleeping 7–8 h per night. Sleep disturbances are extremely common in older populations. Epidemiologic data indicate that 12–54% of older individuals report symptoms of sleep difficulties on a consistent basis.[30,31] As a result, any causal relationship between disrupted sleep and obesity would have substantial importance from a public health standpoint.

Not only was short sleep duration associated with increased BMI, it was associated with an increased fat mass,

Table 3 Association between sleep duration and obesity among older men and women

	Sleep duration (h)			
	<5	5 to <7	7 to <8	⩾8
Men (MrOS)				
Overall ($n=3030$)	3.70 (2.72–5.04)	1.51 (1.18–1.93)	1.00	1.20 (0.77–1.86)
Including adjustment for AHI ($n=2841$)	3.04 (2.19–4.23)	1.44 (1.11–1.86)	1.00	1.25 (0.79–1.96)
Restricted to AHI <15 ($n=1608$)	2.70 (1.64–4.45)	1.37 (0.95–1.97)	1.00	1.59 (0.87–2.89)
Women (SOF)				
Overall ($n=2939$)	2.26 (1.64–3.13)	1.29 (1.05–1.60)	1.00	0.84 (0.61–1.15)
Including adjustment for AHI ($n=454$)	1.94 (0.85–4.42)	1.29 (0.79–2.12)	1.00	0.78 (0.35–1.77)
Restricted to AHI <15 ($n=280$)	2.26 (0.59–8.69)	1.21 (0.64–2.29)	1.00	0.95 (0.36–2.51)

The odds ratio (with 95% confidence interval) for obesity by sleep duration category relative to those sleeping 7–8 h is displayed. All models adjust for study site, age, race, level of education, smoking status, alcohol and caffeine consumption, use of benzodiazepines and antidepressants, depression, physical activity, history of diabetes, heart disease and stroke. AHI, apnea hypopnea index; MrOS, Osteoporotic Fractures in Men Study; SOF, Study of Osteoporotic Fractures.

Table 4 Association between sleep duration and obesity among older men and women stratified by self-reported sleep symptoms

	Sleep duration (h)			
	<5	5 to <7	7 to <8	⩾8
Men (MrOS)				
No excessive daytime sleepiness ($n=2640$)	3.64 (2.59–5.13)	1.57 (1.20–2.04)	1.00	1.31 (0.83–2.07)
Excessive daytime sleepiness ($n=390$)	3.04 (1.39–6.67)	0.98 (0.48–1.99)	1.00	0.27 (0.05–1.55)
No insomnia ($n=1142$)	3.40 (2.06–5.63)	1.11 (0.75–1.64)	1.00	0.84 (0.39–1.79)
Insomnia ($n=1888$)	3.93 (2.63–5.86)	1.83 (1.32–2.54)	1.00	1.48 (0.86–2.56)
Women (SOF)				
No excessive daytime sleepiness ($n=2620$)	1.80 (1.25–2.59)	1.25 (1.00–1.57)	1.00	0.84 (0.60–1.16)
Excessive daytime sleepiness ($n=319$)	8.14 (3.22–20.5)	1.88 (0.90–3.95)	1.00	0.90 (0.25–3.26)
No insomnia ($n=1415$)	1.51 (0.92–2.46)	1.03 (0.75–1.42)	1.00	0.78 (0.48–1.28)
Insomnia ($n=1524$)	3.21 (2.06–5.00)	1.54 (1.16–2.06)	1.00	0.88 (0.58–1.33)

The odds ratio (with 95% confidence interval) for obesity by sleep duration category relative to those sleeping 7–8 h is displayed. All models adjust for age, race, level of education, smoking status, alcohol and caffeine consumption, use of benzodiazepines and antidepressants, depression, physical activity, history of diabetes, heart disease and stroke. Excessive daytime sleepiness defined as Epworth Sleepiness scale >10.[26] Insomnia defined as reporting either sleep onset or sleep maintenance difficulties occurring three or more times a week on the Pittsburgh Sleep Quality Index. MrOS, Osteoporotic Fractures in Men Study; SOF, Study of Osteoporotic Fractures.

supporting a role for chronic sleep restriction in obesity pathogenesis. Furthermore, reduced sleep was associated with increased waist circumference and truncal fat, suggesting an association with central abdominal fat, the fat depot most strongly associated with diabetes and cardiovascular morbidity.[32–34] Although our results suggest a stronger association between short sleep and obesity in men, direct comparison is not possible as the women (SOF cohort) were nearly a decade older than the men (MrOS cohort).

Sleeping more than 8 h was also associated with increased truncal and total body fat in the MrOS cohort. In addition, a similar (but nonsignificant trend) towards a U-shaped association between sleep duration and obesity risk, BMI, waist and hip circumferences was found in MrOS. In contrast, an inverse linear association appears to exist between sleep duration and adiposity measures in the SOF cohort. This difference in patterns may be due to gender differences in susceptibility to altered sleep habits as has been suggested in some recent studies,[35,36] but again, this may also reflect differences in age or other factors between the two cohorts.

Our findings suggest a strong association between reduced sleep times and obesity in older populations. This study is one of the first analyzing the relationship between sleep duration and obesity to use actigraphy in order to objectively measure sleep duration and to do so in older adults. In the vast majority of previous studies on the association between sleep and adiposity, sleep duration has been assessed on the basis of self-report. The validity of self-report is unclear. It has been argued that questions about usual sleep time may measure overall health rather than sleep duration itself.[37] Evidence for systematic bias in self-reported sleep questions exists as responses overestimate the amount of sleep compared to measurements made by actigraphy or polysomnography.[38,39]

Our findings stand in contrast to previous reports on the relationship between sleep and obesity in older populations. Data from the National Health and Nutrition Examination

Survey (NHANES I) suggested that although obesity was associated with reduced self-reported sleep durations in younger adults, no relationship existed in those over the age of 50 years.[10] A European population-based telephone surveys of adults over the age of 55 years found that obesity was not an independent predictor of self-reported sleep duration.[11] In contrast, our results are very similar to those from a recent study of 983 older Dutch adults that also used actigraphy to quantify sleep duration.[12] Averaging across men and women, sleeping less than 5 h was associated with a 1.6 kg/m^2 greater BMI and a 2.8 greater odds of obesity in that cohort.

Our study is also the first in the field to formally assess sleep apnea severity and to assess the relationship between sleep duration and obesity independent of this factor, which may lead to a causal relationship in the opposite direction (obesity predisposes to sleep apnea, which causes reduced sleep durations). Sleep apnea is highly prevalent in older adults,[40] and associated with both reduced sleep times and obesity.[41,42] Previous research has ignored this explanatory pathway or attempted to address it by the use of data on self-reported snoring or previous physician diagnosis of sleep apnea.[43,44] However, these measures are insensitive and do not capture severity of sleep apnea. In these analyses, particularly in men, we were able to show a modest attenuation of the association between reduced sleep and obesity after adjusting for sleep apnea severity suggesting a portion of the association can be explained by this pathway. However, we demonstrated a significant association between short sleep and obesity persisted after controlling for AHI or after restricting analyses to those without significant sleep apnea.

Sleep duration may vary in association with many underlying risk factors and medical and psychiatric conditions. Standardized collection of a broad variety of data allowed us to adjust for factors such as depression, physical activity, medication use and chronic illnesses. We extended previous findings by systematically assessing the consistency of associations in groups with and without insomnia and with and without sleepiness. In men, only minor differences in the magnitude of association between short sleep and obesity were observed regardless of reported sleepiness or insomnia symptoms. In contrast, larger effects were suggested among women with sleep symptoms. The significance of these differences, however, was not supported by finding a statistically significant interaction term, and overall, our findings suggest that the effect of sleep deprivation on weight regulation is likely to be independent of the etiology of the reduced sleep (for example, insomnia vs voluntary curtailment of sleep).

It has been reported in an elderly cohort where sleep was based on self-report that while a short nighttime sleep duration is associated with obesity, the obese are more likely to take daytime naps so that overall sleep duration for a 24-h period is not associated with obesity.[45] In contrast, in this study using actigraphy to assess sleep duration, reduced nighttime sleep duration and reduced 24-h sleep duration were both associated with elevated BMI.

Our study has many strengths, including large cohorts of men and women, objective measures of sleep duration using actigraphy, comprehensive assessments of BMI and body composition, and collection of important covariates such as severity of sleep apnea. Several limitations should also be recognized. We cannot exclude the potential for selection bias as the frailest subjects in the parent cohorts were probably less likely to participate in the sleep assessments. In addition, because this work is cross-sectional, a causal direction cannot be definitively established. However, experimental work has confirmed that sleep deprivation can have metabolic effects that may be relevant to weight homeostasis. Two nights of sleep restriction to 4 h per night has been found to lead to reductions in circulating leptin, elevations in ghrelin and increases in self-report of hunger and appetite.[46]

Sleep disruption and reduced sleep times are highly prevalent in older populations. In fact, it is commonly suggested that reduced sleep is a normal part of aging. Our data would suggest that reduced sleep is associated with obesity in older cohorts to a similar extent as that observed in children and younger adults. Given the greater prevalence of obesity-related complications, including diabetes, cardiovascular disease and cancer, the importance of ensuring adequate sleep may actually be more important from a morbidity standpoint in this age group. Our work underscores the need for further research including longitudinal studies with repeated measures of both sleep and weight over time as well as interventional studies where sleep duration is manipulated to better define whether poor sleep predisposes to adverse metabolic consequences in older populations.

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