Sleep duration, general and abdominal obesity, and weight change among the older adult population of Spain1–4

Esther López-García, Raquel Faubel, Luz León-Muñoz, María C Zuluaga, José R Banegas, and Fernando Rodríguez-Artalejo

ABSTRACT

Background: Short sleep duration is associated with obesity and weight gain among children and young adults. However, there are few studies on the elderly, with conflicting results.

Objective: We examined the association of habitual sleep duration with obesity and weight change among the population aged ≥60 y in Spain.

Design: This prospective study was conducted from 2001 to 2003 on 3576 persons whose habitual sleep duration was self-reported in 2001. The outcomes were obesity [body mass index (BMI; in kg/m²) ≥30], severe obesity (BMI ≥35), and abdominal obesity (waist circumference >102 cm in men and >88 cm in women) in 2001 and weight gain ≥5 kg in the period 2001–2003.

Results: Compared with subjects who slept 7 h, subjects who slept ≤5 h had a greater frequency of obesity [odds ratio (OR): 1.33; 95% CI: 1.00, 1.77] and severe obesity (OR: 2.08; 95% CI: 1.31, 3.32). In addition, sleeping 8 h was associated with obesity (OR: 1.39; 95% CI: 1.11, 1.75) and severe obesity (OR: 1.82; 95% CI: 1.21, 2.73). Similarly, subjects sleeping 9 h were more likely to have severe obesity (OR: 1.57; 95% CI: 1.00, 2.47). Among women, weight gain ≥5 kg was more frequent among subjects sleeping ≤5 h (OR: 3.41; 95% CI: 1.34, 8.69), 8 h (OR: 3.03; 95% CI: 1.29, 7.12), and 9 h (OR: 3.77; 95% CI: 1.55, 9.17). No association was observed between sleep duration and abdominal obesity.

Conclusions: Among older adults, sleeping ≤5 h and sleeping 8 or 9 h was associated with obesity and with short-term weight gain in women. Am J Clin Nutr 2008;87:310–6.

KEY WORDS Sleep duration, obesity, weight change, elderly

INTRODUCTION

Spain and other developed countries have witnessed an obesity epidemic during the past decades (1, 2). This epidemic has important public health consequences because obesity is associated with numerous diseases and disability (3), greater use of health services (4), and shorter life expectancy (5). Because obesity treatments are of limited efficacy, the identification of new determinants of excess weight that could lead to preventive strategies is of the greatest interest. Among these new determinants, habitual sleep curtailment has recently received much attention.

In Western societies, obesity has raised in parallel with a decrease in hours of sleep (6), because of biological factors, such as sleep and other medical disorders, and because of behavioral and social factors, such as child care, shift-work, and round-the-clock entertainment. Experimental evidence in animals also shows that circadian rhythm is associated with obesity and metabolic dysfunction (7), as well as clinical evidence in humans that sleep debt has a harmful effect on carbohydrate metabolism and endocrine function (8). Epidemiologic studies on children and adolescents have shown a consistent and strong association between shorter sleep duration and greater frequency of both current and future obesity (9). In adults, the results are less consistent, although a number of cross-sectional studies have reported that obesity is more frequent among those who sleep less (10–15). Furthermore, the 3 longitudinal studies published to date have shown that sleeping <7 h is, in the long term, associated with obesity in young adults (11, 16) and with weight gain and incident obesity in middle-aged women (17).

To our knowledge, however, only 2 studies have examined this relation in older adults, and these studies have yielded conflicting results. Gangwisch et al (11) analyzed longitudinal data in persons aged 50–67 y and 68–86 y in the United States without observing significant differences in body weight by sleep duration. In contrast, in a cross-sectional study on persons aged ≥60 y in Paris, Ohayon and Vecchierini (18) observed a higher frequency of excess weight among participants sleeping ≤4.5 h than participants who slept 6–8 h.

Finally, we know of no previous study, whether in the elderly or any other age group, that has investigated the relation between sleep duration and abdominal obesity. This relation is important because sleep curtailment is associated with metabolic disorders, such as insulin resistance (8), and because abdominal obesity has greater metabolic effects than does general obesity (19).

Accordingly, this study examined the cross-sectional association between habitual sleep duration and general and abdominal obesity among the older adult population of Spain. In addition, it

1 From the Department of Preventive Medicine and Public Health, School of Medicine, Universidad Autónoma de Madrid, Madrid, Spain (EL-G, RF, LL-M, MCZ, JRB, and FR-A) and CIBER on Epidemiology and Public Health (CIBERESP), Spain (EL-G, RF, LL-M, MCZ, JRB, and FR-A).

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4 Reprints not available. Address correspondence to F Rodríguez-Artalejo, Departamento de Medicina Preventiva y Salud Pública, Facultad de Medicina, Universidad Autónoma de Madrid, C/ Arzobispo Morcillo 2, 28029 Madrid, Spain. E-mail: fernando.artalejo@uam.es.

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examined the longitudinal relation between sleep duration and short-term weight change.

SUBJECTS AND METHODS

Study design and participants

The study methods were reported elsewhere (4, 20). Briefly, this was a prospective, population-based cohort study. The cohort was established in 2001 and followed for 2 y. In 2001, information was obtained on 4008 persons (1739 men and 2269 women) representative of the noninstitutionalized, Spanish population aged ≥60 y. Data were collected through home-based personal interview and physical examination by trained and certified personnel.

In 2003, an attempt was made to contact the participants again, being successful with 3235 participants (1411 men and 1824 women). The participants contacted did not differ significantly from participants lost to follow-up in any sociodemographic or lifestyle characteristic, except for the number of chronic diseases diagnosed and reported in 2001, which was 1.4 among participants followed up and 1.2 among participants lost to follow-up. In 2003, data were collected through telephone interviews conducted by trained staff members. In Spain evidence shows that information on lifestyle and use of health services obtained from phone interviews is valid and reliable in comparison with that obtained in household face-to-face interviews (21, 22).

All study participants or cohabiting next-of-kin gave informed consent to participate. The study was approved by the Clinical Research Ethics Committee of the "La Paz" University Teaching Hospital in Madrid, Spain.

Study variables

Principal variables

The 3 dependent variables were body mass index (BMI; in kg/m²) based on measured weight and height in 2001, measured waist circumference (WC) in 2001, and weight change in the period 2001–2003 determined as the difference in weight reported in 2001 and 2003.

Body weight was measured to the nearest 0.1 kg with the use of a calibrated precision scale (Seca Model 812; Vogel & Halke, Hamburg, Germany) with participants lightly dressed and in stocking feet. Height was measured to the nearest 0.1 cm with the use of a portable wall-mounted stadiometer (KaWe, Asperg, Germany) with participants in stocking feet and standing straight against a wall without skirting board. Measurement of weight and height, with the use of standardized procedure (23), was validated by the researchers repeating the measurements in a random sample of 100 participants. Intraclass correlation coefficients between the measurements taken by trained staff members and measurements taken by the researchers were 0.97 for weight and 0.92 for height. BMI was calculated. Obesity was defined as BMI ≥ 30 and severe obesity as BMI ≥ 35.

WC was measured with participants lightly clothed, with the use of a flexible, inelastic belt-type measuring tape. WC was deemed to be located at the midpoint between the lowest ribs and the iliac crest. WC was measured with the use of standardized procedures (23) and was also validated in a random sample of 100 participants. The intraclass correlation coefficient was 0.89. Abdominal obesity was defined as WC > 102 cm in men and > 88 cm in women.

To determine change in body weight, both in 2001 and in 2003, participants were asked the following question, “Can you tell me approximately how much you weigh, when undressed and barefoot?” In our study, there was a good correlation between reported and measured weight in 2001 (intraclass correlation coefficient: 0.95), thus making it likely that reported weight was also valid in 2003. The principal independent variable was sleep duration in 2001, obtained from the following question: “How many hours do you usually sleep per day (including sleep at night and during the day)?”

Potential confounders

In 2001, information was obtained on variables that previous studies had associated with sleep duration, obesity, or both. Specifically, a questionnaire was used to collect data on sex, age (in y), and physical activity (inactive, occasional activity, and regular activity). Evidence suggests that this classification of physical activity predicts weight change in study participants (24).

Information was also gathered on smoking (never-smoker, ex-smoker, and smoker) and consumption of alcoholic beverages (abstainer, exdrinker, moderate consumption, and excessive consumption). The threshold separating excess from moderate consumption was an intake of alcohol ≥20 g/d in women and > 30 g/d in men. Data were also collected on coffee consumption (no consumption, <1, 1–2, and >2 cups/d), educational level (no formal education, primary, secondary, and university), and social network, ascertained from the number of social links (married status, cohabitation, frequent contact with friends, and frequent contact with family) (25). In addition, we asked about perceived health, classifying participants as having good health (excellent, very good, or good) or poor health (fair, poor, or very poor). Further data were collected on depression with the need for treatment and on the number of the following chronic diseases diagnosed by the physician and reported by the participant: chronic obstructive pulmonary disease, ischemic heart disease, stroke, arthritis, cataracts without treatment, diabetes mellitus, Parkinson disease, cancer at any site, and arterial hypertension. Earlier studies have shown good agreement between self-reported diseases and clinical history among the elderly (26, 27).

Finally, participants were asked about waking up during sleep and the use of anxiolytic medication.

In 2003, information was obtained by telephone on the above-mentioned variables and on whether the participants had tried to change their weight intentionally during the period 2001–2003. The possible responses were yes and no.

Statistical analysis

Cross-sectional analysis

This analysis examined the relation between sleep duration and frequency of general and abdominal obesity in 2001. Of the 4008 participants at baseline, the following participants were excluded: 25 for having an extreme BMI (<18.5 or >45), 323 for lacking WC data or because the values were extreme (<60 or >160 cm), and 84 for not reporting sleep duration. Accordingly, the analyses were conducted with 3576 participants (1577 men and 1999 women).

Differences in baseline characteristics across sleep duration categories were examined with the use of variance analysis and the chi-square test. Associations of sleep duration with general and abdominal obesity were summarized with the use of odds
ratios (ORs) and their 95% CIs, obtained from logistic regression. Sleep duration expressed in hours was modeled with the use of dummies, because in other studies the relation between hours of sleep and obesity was not monotonic (11–14, 17, 18). The category of 7 h of sleep was used as reference, for comparison with earlier studies on older adults (11, 18). We tested the overall association between sleep duration and obesity with a likelihood ratio test that compared a model with dummy terms for sleep categories with a model without such terms. Because mild degrees of obesity may not increase mortality among the elderly (28, 29), analyses were done separately for obesity and severe obesity. Analyses were adjusted for confounders in 2001, modeled with dummies. A secondary analysis was performed removing physical activity from the models, because less physical activity, resulting from greater daytime sleepiness among those sleeping less, has been proposed as a mechanism of the study relation (9).

Longitudinal analysis

To examine whether sleep duration in 2001 predicted weight change in the period 2001–2003, we used information on the 3235 participants who could be followed. Of those participants, we excluded 245 because of death and 428 for missing data (279 on weight in 2001 or 2003, 46 on sleep duration, and 103 on confounding variables). Finally, we further excluded 22 participants for having an extreme BMI (<18.5 or >45) and 204 whose weight had changed by >10 kg in 2 y, because this change is implausible or might be due to severe disease. Hence, the final analyses were conducted with 2335 participants (1064 men and 1271 women). In comparison with the 3576 participants in the cross-sectional study, the 2335 included in the longitudinal analyses were slightly younger (70.7 ± 7.2 y compared with 71.6 ± 7.7 y), had a lower baseline BMI (28.2 ± 4.2 compared with 28.9 ± 4.4), a higher frequency of men (45.6% compared with 44.1%), a lower frequency of persons with no formal education (48.5% compared with 51.6%), and a lower fraction of sedentary participants (40.0% compared with 43.0%). Moreover, participants in the longitudinal analysis slept 7.9 ± 1.9 h/d compared with 8.0 ± 1.9 h/d among participants in the cross-sectional study. None of these differences was statistically significant.

Analyses were performed with the use of polytomous logistic regression (30). We estimated one OR for the loss of ≥5 kg and another for the gain of ≥5 kg. In both cases, comparison was made with participants whose weight had changed <5 kg. We chose 5 kg as the cutoff because it approximately corresponded with 7% of the body weight in our sample and because it is a reasonable target for weight loss through lifestyle changes (31). The independent variables were sleep duration and confounders in 2001, modeled as in the cross-sectional analysis. Analyses were also adjusted for baseline BMI, which has been shown to predict weight change in the study participants, and for intentionality of weight change, because voluntary changes (eg, for medical prescription) may overlap with those associated with sleep duration (24).

Because the study relation might be influenced by changes in potential confounders during the period 2001–2003, in a secondary analysis the models were additionally adjusted for the following variables in 2003: physical activity, smoking, alcohol consumption, perceived health, and social network. Models were also adjusted for the number of diseases diagnosed in the period 2001–2003, because involuntary weight loss may be a manifestation of underlying disease (32).

Analyses were performed on the total participants and separately by sex, because in some cases results varied with sex. Interactions of sex with sleep duration were tested with the use of a likelihood ratio test that compared a model with 5 interaction terms (products of sex-by-sleep category) with a model without such terms. All statistical tests were 2-sided, and statistical significance was set at P < 0.05. Analyses were done with the use of the SAS program, version 9.1 for WINDOWS (SAS Institute, Cary, NC) (33).

RESULTS

Cross-sectional analysis

The mean (±SD) age of participants was 71.6 ± 7.7 y (71.0 ± 8.0 y for men and 72.1 ± 7.6 y for women). Overall, 9.8% of participants slept ≤5 h, 11.4% slept 6 h, 14.9% slept 7 h, 26.2% slept 8 h, 16.5% slept 9 h, and 21.1% slept ≥10 h. Prevalence of obesity was 35.9% (31.0% in men and 40.7% in women) and that of severe obesity was 8.8% (4.8% in men and 12.8% in women). For abdominal obesity, prevalence was 63.5% (49.1% in men and 78.0% in women).

In Table 1, we show the characteristics of the study participants stratified by sleep duration. In comparison with participants in the extreme categories of sleep duration (≤5 h and ≥10 h), participants who slept 7 or 8 h were slightly younger (P < 0.05, Tukey’s test), engaged in occasional or regular physical activity, consumed alcohol more frequently, and had a higher educational level. Furthermore, they reported a greater number of social links, a higher frequency of good perceived health, and a lower number of chronic diseases (P < 0.05, Tukey’s test). Among participants with shorter sleep duration, there was a higher frequency of women, diagnosed depression, and use of anxiolytic medication. Finally, the greater the number of hours of sleep, the more likely the participants were to awaken during the night. The variables associated with sleep duration were similar in men and women (data not shown).

In Table 2, we present the OR of general and abdominal obesity at baseline, according to sleep duration. Sex- and age-adjusted analyses showed that, compared with participants who slept 7 h, participants sleeping ≤5 h had a higher frequency of obesity (OR: 1.45; 95% CI: 1.09, 1.92) and severe obesity (OR: 2.36; 95% CI: 1.50, 3.74). Sleeping 8 h was also associated with obesity (OR: 1.41; 95% CI: 1.12, 1.76) and severe obesity (OR: 1.86; 95% CI: 1.24, 2.78). Similarly, participants who slept 9 h had a greater frequency of severe obesity (OR: 1.68; 95% CI: 1.08, 2.61).

These results did not change substantially after adjustment for potential confounders (Table 2). Similarly, no change was observed when physical activity was withdrawn from the models (data not shown).

No statistically significant differences in the frequency of obesity were found between participants who slept 6 or ≥10 h and participants who slept 7 h. Neither was any association observed between sleep duration and frequency of abdominal obesity. No evidence suggests that results varied with sex, because P values for the interaction of sex with sleep duration were >0.05 for all models in Table 2.
Longitudinal analysis

During the 2-y follow-up, 15.2% of participants lost ≥5 kg (14.9% in men and 15.5% in women), whereas 9.9% increased their baseline weight ≥5 kg (11.3% in men and 8.4% in women). The OR of weight change in the period 2001–2003 according to sleep duration in 2001 is shown in Table 3. Results varied with sex (P for the interaction of sex with sleep duration was 0.02, both in the model with partial adjustment and in the model with full adjustment). In analyses adjusted for age and baseline BMI, women who slept ≤5 h gained ≥5 kg more frequently than did women who slept 7 h (OR: 3.61; 95% CI: 1.45, 9.01). Also in women, gaining ≥5 kg was also more frequent among participants sleeping 8 h (OR: 3.02; 95% CI: 1.30, 7.02) and 9 h (OR: 4.23; 95% CI: 1.78, 10.04). However, no overall association between sleep duration and weight gain was observed in men or in the total of study participants.

The results did not change substantially when the analyses were adjusted for confounders in 2001 and intentional weight change (Table 3). Similarly, no material change was observed after additional adjustment for physical activity, smoking, alcohol consumption, perceived health, and social network in 2003 and for the number of diseases diagnosed in the period 2001–2003 (data not shown). Finally, sleep duration measured in 2001 was not associated with weight loss of ≥5 kg in the following 2 y (Table 3).

DISCUSSION

Our results showing that short sleep duration is associated with obesity are in line with those of Ohayon and Vecchierini (18) who, in a cross-sectional study among older adults, observed that subjects who slept ≤4.5 h had excess weight (BMI > 27)
more frequently than did subjects who slept 6–8 h (OR: 3.6; 95% CI: 1.0, 13.1). In our study, the association was stronger when obesity was defined as BMI ≥ 35 than when it was defined as BMI ≥ 30. This finding is consistent with a study on middle-aged women, in which the relative risk of weight gain associated with short sleep duration rose with the cutoff used to define weight gain (17). Finally, although it was suggested that the strength of the association between short sleep duration and obesity declines with age (9, 11, 16), in our study the association between sleeping ≤5 h and severe obesity was of a magnitude similar to that of a cross-sectional study on young adults (11).

There is considerable variability across studies about the form of the dose-response relation between sleep duration and obesity, particularly in the long sleep range. Among men and women aged 30–60 y, Taheri et al (12) observed a U-shaped relation, with a minimal BMI in a sleep duration of 7–8 h. Similarly, Kripke et al (13), in their large-sized study on persons aged 30–102 y, observed a U-shaped relation in women, whereby both short and long sleep durations were associated with greater BMI. In contrast, in men they described a monotonic inverse relation between BMI and sleep duration in the range of 3 to ≥10 h. Vioque et al (10) observed that frequency of obesity was 24% lower for each additional hour of sleep in the range of ≤6 to ≥9 h among persons aged ≥15 y. However, among subjects aged 31–49 y who were followed during 8–10 y, Gangwisch et al (11) observed that BMI decreased as sleep duration increased from 2–4 to 6 h, but thereafter BMI remained approximately stable when sleep duration increased to ≥10 h. Similarly, Patel et al (17) observed that, in women aged 39–65 y who were followed during 16 y, sleep was associated with incident obesity and greater weight gain when its duration was <7 h, but this association was not in evidence for durations >7 h. Ohayon and Vecchierini (18) likewise failed to observe any association between long sleep and obesity. Finally, in our study, sleeping ≤5 h and sleeping 8 or 9 h were both associated with a higher frequency of obesity than was sleeping 7 h. However, sleeping ≥10 h was not associated with obesity, making it difficult to establish whether the dose-response relation is U-shaped. These inconsistencies in results between studies might be due to differences in the age and frequency of obesity among participants, the type of study design (cross-sectional compared with longitudinal), the duration of follow-up, and the degree of adjustment for confounders. Results may also seem to conflict because not all studies report results by age.

In our study, sleeping ≤5 h and sleeping 8 or 9 h were both associated with a weight gain of ≥5 kg in 2 y in women. Because these results proceed from a prospective follow-up, and because they are in line with results of the cross-sectional analysis, they reinforce the hypothesis that sleep duration is associated with obesity in the elderly. However, we do not have an explanation for this association being observed only in women. Other researchers have observed sex differences in the study association, but participant’s age ranged from 3 through 102 y and data were not disaggregated by age. In addition, present knowledge of the mechanisms for the association between sleep duration and obesity is insufficient to explain sex differences. Thus, our results on this particular issue should be confirmed in future studies.

Finally, although sleep duration was associated with general obesity, it showed no such association with abdominal obesity. This discrepancy may be linked to the different pathogenic mechanisms of each type of obesity. Among the elderly, the frequency of general obesity decreases with age, but the frequency of abdominal obesity remains stable or even increases (34). Moreover, the frequency of abdominal obesity was high in our cohort, so that the effect of sleep duration on abdominal obesity would have had to be marked to be discernible. Indeed, in a previous study, the association between sleep duration and weight change was less pronounced among subjects with excess weight (17).

### Table 2

Odds ratios (95% CIs) of obesity, severe obesity, and abdominal obesity in 2001, according to habitual sleep duration in 2001

<table>
<thead>
<tr>
<th>Sleep duration (hours per 24-h period)</th>
<th>All (n = 350)</th>
<th>6 (n = 409)</th>
<th>7 (n = 532)</th>
<th>8 (n = 938)</th>
<th>9 (n = 591)</th>
<th>≥10 (n = 756)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model adjusted for age and sex</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td>1.45 (1.09, 1.92)</td>
<td>1.18 (0.90, 1.55)</td>
<td>1.41 (1.12, 1.76)</td>
<td>1.14 (0.89, 1.47)</td>
<td>1.08 (0.85, 1.37)</td>
<td>0.01</td>
</tr>
<tr>
<td>Severe obesity</td>
<td>2.36 (1.50, 3.74)</td>
<td>1.31 (0.80, 2.14)</td>
<td>1.86 (1.24, 2.78)</td>
<td>1.68 (1.08, 2.61)</td>
<td>1.28 (0.81, 2.00)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Abdominal obesity</td>
<td>1.22 (0.89, 1.65)</td>
<td>1.01 (0.76, 1.35)</td>
<td>1.04 (0.82, 1.32)</td>
<td>1.09 (0.84, 1.41)</td>
<td>1.11 (0.87, 1.42)</td>
<td>0.82</td>
</tr>
<tr>
<td>Model with full adjustment</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td>1.33 (1.00, 1.77)</td>
<td>1.14 (0.86, 1.50)</td>
<td>1.39 (1.11, 1.75)</td>
<td>1.07 (0.82, 1.38)</td>
<td>0.96 (0.75, 1.23)</td>
<td>0.005</td>
</tr>
<tr>
<td>Severe obesity</td>
<td>2.08 (1.31, 3.32)</td>
<td>1.29 (0.78, 2.12)</td>
<td>1.82 (1.21, 2.73)</td>
<td>1.57 (1.00, 2.47)</td>
<td>1.13 (0.71, 1.80)</td>
<td>0.004</td>
</tr>
<tr>
<td>Abdominal obesity</td>
<td>1.14 (0.84, 1.56)</td>
<td>1.00 (0.75, 1.34)</td>
<td>1.04 (0.82, 1.32)</td>
<td>1.06 (0.82, 1.39)</td>
<td>1.06 (0.82, 1.36)</td>
<td>0.97</td>
</tr>
</tbody>
</table>

1 Obtained from a likelihood ratio test that compared a model with dummy terms for sleep duration categories with a model without such terms. There was no significant interaction of sex with sleep duration.

2 BMI (in kg/m^2) ≥ 30.

3 BMI ≥ 35.

4 Waist circumference >102 cm in men or >88 cm in women.

5 Logistic regression model adjusted for sex, age (60–69, 70–79, ≥80 y), physical activity (inactive, occasional, regular), smoking (nonsmoker, exsmoker, smoker), alcohol consumption (abstainer, exdrinker, moderate consumption, excessive consumption), coffee consumption (no consumption, <1, 1–2, >2 cups/d), educational level (no formal education, primary, secondary, university), number of social links, perceived health (good or poor), number of chronic diseases (0, 1, ≥2), depression (yes or no), arousal from sleep at night (yes or no), and intake of anxiolytic medication (yes or no).
TABLE 3
Odds ratios (95% CIs) of ≥5-kg weight gain and loss in the period 2001–2003, according to habitual sleep duration in 2001

<table>
<thead>
<tr>
<th></th>
<th>Sleep duration (hours per 24-h period)</th>
<th>Overall P for differences across categories (^1)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>≤5</td>
<td>6</td>
</tr>
<tr>
<td><strong>Model adjusted for age, sex, and basal BMI</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>244</td>
<td>274</td>
</tr>
<tr>
<td>Men</td>
<td>87</td>
<td>115</td>
</tr>
<tr>
<td>Women</td>
<td>158</td>
<td>159</td>
</tr>
<tr>
<td>Gain ≥5 kg</td>
<td>1.65 (0.90, 3.06)</td>
<td>1.46 (0.80, 2.67)</td>
</tr>
<tr>
<td>Loss ≥5 kg</td>
<td>1.07 (0.67, 1.71)</td>
<td>0.90 (0.56, 1.44)</td>
</tr>
<tr>
<td><strong>Model with full adjustment (^2)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>244</td>
<td>274</td>
</tr>
<tr>
<td>Men</td>
<td>87</td>
<td>115</td>
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<td>Women</td>
<td>158</td>
<td>159</td>
</tr>
<tr>
<td>Gain ≥5 kg</td>
<td>1.57 (0.84, 2.93)</td>
<td>1.47 (0.80, 2.70)</td>
</tr>
<tr>
<td>Loss ≥5 kg</td>
<td>1.07 (0.66, 1.73)</td>
<td>0.89 (0.55, 1.43)</td>
</tr>
</tbody>
</table>

\(^1\) Obtained from a likelihood ratio test that compared a model with dummy terms for sleep duration categories with a model without such terms. \(P = 0.02\) for the interaction of sex with sleep duration for weight gain in both models.

\(^2\) Polytomous logistic regression model adjusted for sex (except in stratified models), age (60–69, 70–79, ≥80 y), baseline BMI (<25.0, 25.0–29.9, ≥30.0), physical activity (inactive, occasional, regular), smoking (nonsmoker, exsmoker, smoker), alcohol consumption (abstainer, exdrinker, moderate consumption, excessive consumption), coffee consumption (no consumption, 1, 1–2, >2 cups/d), educational level (no formal education, primary, secondary, university), number of social links, perceived health (good or poor), number of chronic diseases (0, 1, ≥2), depression (yes or no), arousal from sleep at night (yes or no), intake of anxiolytic medication (yes or no), and intentional weight change (yes or no).

Mechanisms of the association between sleep duration and obesity

The possible mechanisms are not known in detail, but evidence suggests that sleep affects some of the mediators of appetite control (35). In particular, short sleep duration is associated with lower concentrations of leptin, an anorexigenic hormone released by adipocytes, and with higher concentrations of ghrelin, an orexigenic hormone produced mainly in the stomach (12, 36). As a result, short sleep duration is associated with greater hunger and appetite. It was also postulated that short sleep duration would produce daytime sleepiness, which would, in turn, lead to less physical activity. Accordingly, in our study, participants who slept ≤5 h engaged in physical activity less frequently than participants who slept 7 h. Yet, as in other studies (16, 17), adjustment for physical activity did not change our results materially. Finally, it was also suggested that sleep may modulate the basal metabolic rate (37) and non–exercise-activity thermogenesis (38). However, the mechanisms whereby long sleep duration could lead to obesity are even less well understood.

Methodologic aspects

For a correct interpretation of the study results, some methodologic comments are needed. First, this study is unique because it analyzes the relation between sleep duration and obesity in a representative sample of the older adult population of an entire country. Moreover, unlike previous studies on older adults (18), our study had a prospective follow-up, which improves causal inferences with respect to cross-sectional studies.

Second, we controlled for a number of confounders, but no information was gathered on sleep apnea, which can be due to obesity and, in turn, influence sleep duration. Persons with sleep apnea tend to lengthen the duration of sleep to compensate for the loss of rest (39). Nevertheless, we observed no association with obesity in sleep durations ≥10 h. Furthermore, sleep apnea is associated with worse subjective health and frequent arousals from sleep (39), and the analyses were adjusted for both variables.

Third, the information on sleep was self-reported. However, the correlates of sleep duration in our study coincide with those of previous studies (17, 18), which supports the validity of the sleep measurement. Moreover, although median sleep duration in our study (8 h) was higher than in older adults in France (6–8 h) (18) or in the United States (7 h) (40), it was similar to that reported in another representative sample of the Spanish elderly (41).

Finally, our cohort was limited to whites living in a Mediterranean country with a high prevalence of obesity. Thus, future studies should establish the extent to which our results are applicable to other older populations that differ in ethnicity and culture.
Conclusion

Among older adults, short as well as 8- and 9-h habitual sleep duration is associated with obesity and with short-term weight gain in women. However, no association was observed between sleep duration, abdominal obesity, and weight loss among older Spanish adults. Because this is one of the first studies on the older adult population, our results should be confirmed in future investigations. Moreover, the mechanisms of the relation between sleep duration and obesity in the elderly require further research.

The author’s responsibilities were as follows—EL-G, RF, LL-M, MCZ, JRB, and FR-A: provided study concept and design and performed critical revision of the manuscript for important intellectual content; JRB and FR-A: acquired data; EL-G, RF, and FR-A: analyzed and interpreted data; EL-G and FR-A: drafted the manuscript, provided statistical expertise, obtained funding, and provided administrative, technical, or material support; FR-A: provided study supervision. None of the authors had a personal or financial conflict of interest.

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