pii: jc-18-00435 https://dx.doi.org/10.5664/jcsm.7668

JCSM
Journal of Clinical Sleep Medicine

SCIENTIFIC INVESTIGATIONS

Association Between Self-Reported Sleep Duration and Body Composition in Middle-Aged and Older Adults

Xiao Tan, PhD[1]; Olga E. Titova, PhD[1]; Eva Lindberg, MD, PhD[2]; Sölve Elmståhl, MD, PhD[3]; Lars Lind, MD, PhD[4]; Helgi B. Schiöth, PhD[1]; Christian Benedict, PhD[1]

[1]Department of Neuroscience, Uppsala University, Sleep Research Laboratory, Uppsala, Sweden; [2]Department of Medical Sciences, Respiratory, Allergy and Sleep Research, Uppsala University, Uppsala, Sweden; [3]Department of Health Sciences, Division of Geriatric Medicine, Lund University, Sweden CRC, Skåne University Hospital, Malmö, Sweden; [4]Department of Medical Sciences, Cardiovascular Epidemiology, Uppsala University, Uppsala, Sweden.

Study Objectives: The current study sought to examine whether self-reported sleep duration is linked to an adverse body composition in 19,709 adults aged 45 to 75 years.
Methods: All variables used in the current study were derived from the Swedish EpiHealth cohort study. Habitual sleep duration was measured by questionnaires. Body composition was assessed by bioimpedance. The main outcome variables were fat mass and fat-free mass (in kg). Analysis of covariance adjusting for age, sex, fat mass in the case of fat-free mass (and vice versa), leisure time physical activity, smoking, and alcohol consumption was used to investigate the association between sleep duration and body composition.
Results: Short sleep (defined as ≤ 5 hours sleep per day) and long sleep (defined as 8 or more hours of sleep per day) were associated with lower fat-free mass and higher fat mass, compared with 6 to 7 hours of sleep duration ($P < .05$).
Conclusions: These observations could suggest that both habitual short and long sleep may contribute to two common clinical phenotypes in middle-aged and older humans, ie, body adiposity and sarcopenia. However, the observational nature of our study does not allow for causal interpretation.
Keywords: body fat, elderly, fat-free mass, middle-aged, sleep
Citation: Tan X, Titova OE, Lindberg E, Elmståhl S, Lind L, Schiöth HB, Benedict C. Association between self-reported sleep duration and body composition in middle-aged and older adults. *J Clin Sleep Med.* 2019;15(3):431–435.

BRIEF SUMMARY
Current Knowledge/Study Rationale: Healthy body composition is a key determinant of overall fitness and health, especially for middle-aged to older adults. However, the link between sleep duration and body composition has rarely been studied in older populations.
Study Impact: Short and long sleep were associated with lower fat-free mass and higher fat mass. Short and long sleep may therefore contribute to two common clinical phenotypes in middle-aged and older humans, ie, body adiposity and sarcopenia.

INTRODUCTION

Body composition refers to different compartments of the body, notably fat mass (mainly consisting of subcutaneous and visceral fat tissue) and lean mass (including muscles, bones, internal organs, ligaments, and tendons). By using bioelectrical impedance analysis technology, body composition can be assessed in larger populations.[1] The bioimpedance instruments measure the impedance of the body to a small electric current, and thus estimate body composition in a two-component model, namely fat-free mass (lean mass excluded by the essential fat in the bone marrow and internal organs) and fat mass.[1,2] Epidemiological studies suggest that an adverse body composition, ie., excessive loss of fat-free mass and/or gain in fat mass, is linked to higher mortality and morbidity, especially in older adults.[3–7] Therefore, identifying factors that may contribute to the development of an adverse body composition in older adults is a public health priority.

Emerging evidence suggests that habitual short and long sleep durations are associated with higher body adiposity in humans. By using wrist actigraphy across several nights in a cohort of older adults (n = 6,107, aged 67 to 96 years), it has, for instance, been shown that a sleep duration of less than 5 hours per night is linked to 3.7-fold higher odds of obesity in men, and 2.3-fold higher odds in women, compared with self-reported sleep duration of 7 to 8 hours.[3] Pooled evidence from adult populations (n = 197,906) further indicates that compared to self-reported sleep duration of 7 to 8 hours per day, a habitual sleep duration of ≤ 6 hours per day is associated with a 45% higher risk of future obesity.[8] In addition to short sleep duration, a relationship between long sleep duration (typically defined as sleep longer than 9 hours per day) and obesity has been described.[9,10]

It should be noted that sleep duration outside the recommended range is also linked with lower fat-free mass, including skeletal muscle. By utilizing bioimpedance, a U-shaped association between sleep duration and the likelihood of sarcopenia has been observed in 488 community-dwelling adults (264 women) aged 65 years and older.[11] Compared to adults reporting 6 to 8 hours of sleep per day, those reporting less than 6 hours of sleep per day had a nearly threefold increased likelihood of sarcopenia, whereas adults reporting 8 or more hours of sleep had an almost twofold increased risk of sarcopenia.[11]

Because most studies so far have focused on the association of sleep duration with either fat mass or fat-free mass, the current study sought to investigate whether self-reported sleep duration is linked to an adverse body composition (assessed by bioimpedance) in a cohort of middle-aged and older adults. A recent experimental study in young men found that acute sleep loss decreased levels of structural proteins in skeletal muscle and increased levels of proteins linked to adipogenesis in adipose tissue.[12] Hence, we hypothesized that short sleep duration would be linked to higher fat mass and lower fat-free mass.

METHODS

Study Population

Data from the Swedish EpiHealth study (www.epihealth.se) were used for the current analysis.[13] EpiHealth is a two-center cohort study aimed at investigating the interactions between genotypes and lifestyle factors regarding development of common degenerative disorders. Invitation letters were sent to a random sample of adults aged 45 to 75 years, who were residents in the counties of Uppsala and Skåne according to the Swedish Population Registry. Participants were asked to fill out an internet-based questionnaire and visit one of the two study centers for physical investigations (located in Uppsala and Malmö, Sweden). Data were collected between 2011 and 2015. The initial sample size was 20,534 adults (56.6% women), from which 50 were excluded due to missing information about sleep duration, 484 due to missing bioimpedance data, 268 because of fat mass or fat-free mass values greater than three standard deviations from the mean, and 23 because there were no data on smoking. Therefore, data from 19,709 participants were available for the analysis. The study was approved by the Ethics Committee at Uppsala University. Participants gave written informed consent to participate.

Assessment of Sleep Duration

Habitual sleep duration (assessed by the question "How many hours per day do you usually sleep?") was measured by a internet-based questionnaire. The response options were: 4 hours or less, 5 hours, 6 hours, 7 hours, 8 hours, 9 hours, 10 hours or more. Due to small sample sizes in the extreme sleep duration categories, the sleep duration categories 4 hours or less and 5 hours were merged into 5 hours or less. In addition, 9 hours and 10 hours or more were combined into 9 hours or more.

Assessment of Body Composition

Fat-free mass and fat mass were estimated by bioelectrical impedance analysis at the study center. The bioelectrical impedance analysis system (Tanita BC-418, Tanita Corp., Tokyo, Japan) incorporates eight contact electrodes (two on each hand and foot). Fat-free mass and fat mass were calculated using the measured impedance and body weight, as well as sex and height.

Other Variables

Body height and weight were determined at the study centers. Height was measured without shoes to the nearest 0.5 cm using a stadiometer. Weight was measured to the nearest 0.1 kg using the bioelectrical impedance analysis system. Body mass index was calculated as weight (kg) per squared height (m^2). Age in years of the participant at the time of investigation was calculated based on the birth time in the Swedish Population Registry. Smoking status (trichotomized into current, past, and never smoker), frequency of alcohol consumption (four levels: never/don't know, one alcoholic drink per week, two to three alcoholic drinks per week, or more than three alcoholic drinks per week), and leisure time physical activity level (five levels, with higher scores representing higher leisure time physical activity levels) were collected by the internet-based questionnaire.

Statistical Analysis Including Description of Covariates

All statistical analyses were performed using SPSS version 24.0 (IBM Corp., Armonk, New York, United States). Descriptive data are presented as means ± standard deviation, unless otherwise specified. All statistical analyses were adjusted for age at the time of investigation, sex, smoking status, frequency of alcohol consumption, and leisure time physical activity level. When analyzing the association between sleep duration (fixed factor) and fat mass, the analysis of covariance was additionally adjusted for the fat-free mass. Conversely, fat mass was entered as covariate in the analysis of covariance when investigation possible differences in fat-free mass between the sleep duration groups. Histogram plots of nonstandardized residuals verified normal distribution of residuals. The aim of the current study was to investigate main effects of sleep duration. Hence, no interactions between sleep duration and potential confounders were modeled. Overall, $P \leq .05$ was considered statistically significant.

RESULTS

Population characteristics, split by sleep duration groups, are described in **Table 1**. As revealed by analysis of covariance utilizing sleep duration as fixed factor, while adjusting for fat mass, sex, exact age, leisure time physical activity level, smoking status, and alcohol consumption, there was a main effect of sleep duration on fat-free mass ($P < .001$). *Post hoc* comparisons (all *P* values are fully adjusted and Bonferroni corrected) revealed that those who reported either to sleep ≤ 5 hours, 8 hours, or ≥ 9 hours per day all exhibited a lower fat-free mass than those who slept 6 to 7 hours per day (except for the contrast 6 hours versus ≤ 5 hours; **Figure 1**). No differences in fat-free mass was found between the 6-hour and 7-hour groups ($P > .99$; **Figure 1**).

With respect to fat mass, a main effect of sleep duration was observed ($P < .001$; adjusted for fat-free mass, sex, exact age, leisure-time physical activity level, smoking status, and alcohol consumption). *Post hoc* comparisons (all *P* values are fully adjusted and Bonferroni corrected) showed that those

Table 1—Main characteristics of the population.

Variable	Sleep Duration (hours)					*P*
	≤ 5	6	7	8	≥ 9	
n (%)	1,622 (8.2)	4,793 (24.3)	8,206 (41.6)	4,343 (22.0)	745 (3.8)	–
Women/men (n)	1,009/613	2,612/2,181	4,593/3,613	2,513/1830	437/308	< .001 *
Age (years), mean ± SD	61.4 ± 8.2	60.1 ± 8.4	59.9 ± 8.5	62.2 ± 8.5	64.1 ± 8.0	< .001 †
Body mass index (kg/m^2), mean ± SD	26.9 ± 4.1	26.2 ± 3.7	25.8 ± 3.6	26.0 ± 3.8	26.7 ± 4.0	.008 ‡
Fat mass (kg), mean ± SD	25.2 ± 8.5	23.4 ± 7.8	23.0 ± 7.7	23.9 ± 7.9	25.6 ± 8.3	.007 ‡
Fat-free mass (kg), mean ± SD	52.1 ± 11.0	53.7 ± 11.3	53.3 ± 11.0	52.4 ± 10.6	52.1 ± 10.6	.002 ‡
Smoking history (n)						< .001 *
Yes	166	369	558	345	59	
Never	689	2,055	3,368	1,848	353	
Former	767	2,369	4,280	2,140	333	
Frequency of alcohol intake (n)						< .001 *
Never or don't know	57	99	145	73	20	
1 time/wk	730	1,889	2,919	1,574	282	
2–3 times/wk	706	2,445	4,451	2,233	321	
> 3 times/wk	129	360	691	463	122	
Leisure time physical activity (n)						< .001 *
1	95	205	230	150	42	
2	386	1,098	1,758	832	163	
3	690	1,916	3,341	1,864	319	
4	348	1,274	2,382	1,229	178	
5	103	300	495	268	43	

Leisure time physical activity is measured on a scale of 1 (primarily sedentary) to 5 (exhaustive activity for at least 60 min/d). * = chi-square test, † = Kruskal-Wallis test, ‡ = analysis of covariance (adjusting for sex). *P* values are unadjusted. SD = standard deviation.

reporting either to sleep ≥ 8 hours or ≤ 5 hours per day had a higher fat mass than those who slept 6 to 7 hours per day. No differences in fat mass was found between the 6-hour and 7-hour groups (P = .688; **Figure 1**).

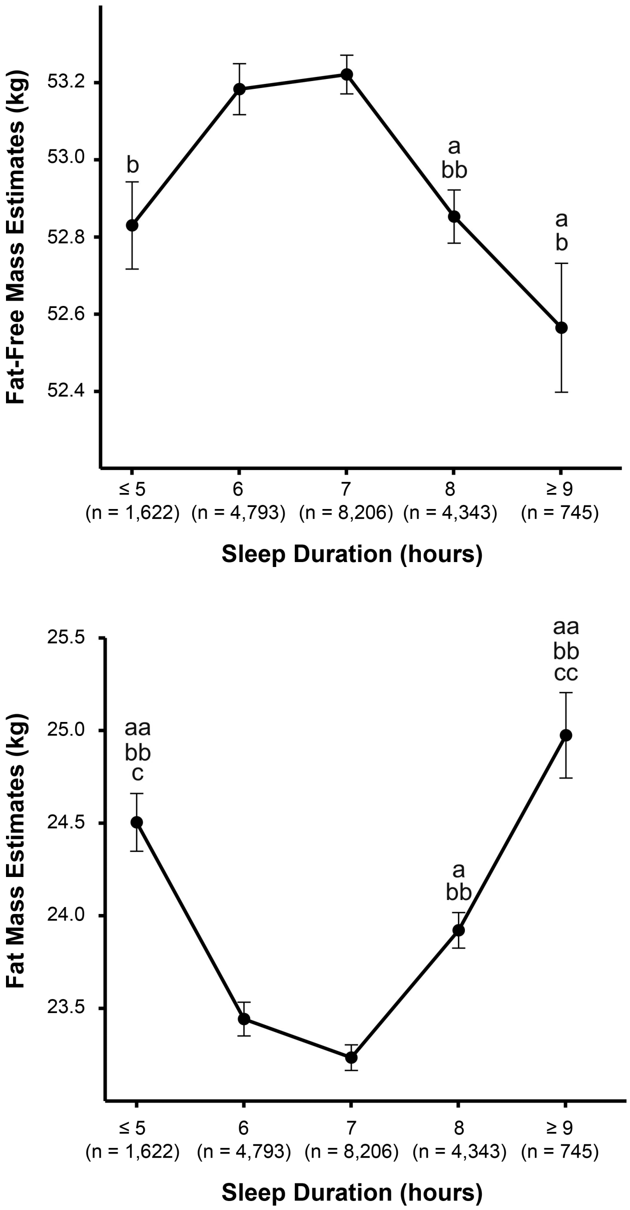
DISCUSSION

In this large cross-sectional cohort study of Swedish middle-aged and older men and women, we found that self-reported habitual sleep duration was associated with participants' body composition, while adjusting for participants', sex, exact age, leisure time physical activity level, smoking status, and alcohol consumption. Specifically, habitual short (≤ 5 hours per day) and long (8 hours or more) sleep were both associated with lower fat-free mass and higher fat mass, compared with 6 to 7 hours of sleep. These observations could suggest that both short and long sleep may contribute to two common clinical phenotypes in humans, ie, body adiposity and sarcopenia. Supporting the latter hypothesis, a recent study in which young male adults were exposed to acute sleep loss versus normal sleep provides possible mechanistic insight into how a lack of sleep may cause adverse alterations in body composition. Specifically, critical differences at a number of molecular levels in how fat tissue and skeletal muscle respond to acute sleep loss were found. Whereas acute sleep loss induced a molecular catabolic signature in skeletal muscle, mirrored by changes in blood metabolites, an adiposity-promoting DNA methylation signature in adipose tissue was found.[12] The findings of this acute sleep deprivation study do not explain the observed association between self-reported long sleep duration and low fat-free mass and high fat mass, respectively. On the one hand, long sleep duration is proposed to impair whole-body energy metabolism through multiple possible compounding mechanisms, including poor sleep quality, sedentary lifestyle, unhealthy dietary choices, and desynchrony between circadian and behavioral states.[9] On the other hand, long sleep duration may also appear as a consequence of metabolic diseases such as type 2 diabetes.[7] Hence, it cannot be ruled out that habitual long sleep duration—as a trait—could also partially arise due to adverse alterations to body composition.

Few prior studies have evaluated the association between sleep duration and body composition. A study involving more than 16,000 middle-aged men and women from South Korea found that reports of sleeping longer than 9 hours per day were associated with sarcopenia, compared with 7 hours of sleep. In the same study, no association between short sleep (defined as < 7 hours per day) and sarcopenia was found.[14] One explanation for why we found a link between short sleep duration and lower fat-free mass whereas the other did not could be related to between-study differences in age of participants (mean age, current versus Korean study: 60.7 years versus 44.1 years), ethnicity of participants, or methods to estimate body composition.

When interpreting our results, several limitations apply. First, our study cannot prove cause and effect. Another limitation is that the sleep duration variable was based on

Figure 1—Fat-free and fat mass in the EpiHealth study, stratified by self-reported habitual sleep duration.



Data are presented as mean ± standard error of the mean. All estimates derive from analysis of covariance. Estimates are adjusted for age, sex, fat mass (in case of fat-free mass shown in the upper panel), fat-free mass (in case of fat mass shown in the bottom panel), leisure time physical activity, smoking, and alcohol consumption. All *P* values are fully adjusted and Bonferroni corrected. A value of $P < .05$ is considered significant, and significant values are indicated with the letters "a," "b," or "c" in the following manner: versus 6-hour sleep duration a = $P < .05$, aa = $P < .001$; versus 7-hour sleep duration b = $P < .05$; bb = $P < .001$; and versus 8-hour sleep duration c = $P < .05$, cc = $P < .001$.

self-reports. Self-reported sleep duration was not validated by polysomnography or actigraphy. Although self-reported sleep duration is widely used in epidemiological sleep research, there is evidence that self-reported sleep duration does only weakly correlate with objective measures of sleep.[15] A significant discrepancy between sleep quantity evaluated by actigraphy and self-reports (up to 1 hour) has, for instance, been found in a sample of 45 community-dwelling elderly people.[16] Importantly, the magnitude of this discrepancy was negatively associated with the subjective degree of positive mood at the time of subjective assessment of sleep quantity,[14] which has not been measured in our study. Thus, observational and interventional studies utilizing objectively measured sleep duration (eg, by wrist actigraphy and polysomnography) are needed to fully disentangle the connection between sleep duration and body composition in middle-aged and older humans.

REFERENCES

1. Sergi G, De Rui M, Stubbs B, Veronese N, Manzato E. Measurement of lean body mass using bioelectrical impedance analysis: a consideration of the pros and cons. *Aging Clin Exp Res*. 2017;29(4):591–597.
2. Wells JC, Fewtrell MS. Measuring body composition. *Arch Dis Child*. 2006;91(7):612–617.
3. Flegal KM, Kit BK, Orpana H, Graubard BI. Association of all-cause mortality with overweight and obesity using standard body mass index categories: a systematic review and meta-analysis. *JAMA*. 2013;309(1):71–82.
4. Bales CW, Ritchie CS. Sarcopenia, weight loss, and nutritional frailty in the elderly. *Annu Rev Nutr*. 2002;22:309–323.
5. Kelley GA, Kelley KS. Is sarcopenia associated with an increased risk of all-cause mortality and functional disability? *Exp Gerontol*. 2017;96:100–103.
6. Kim TN, Park MS, Yang SJ, et al. Prevalence and determinant factors of sarcopenia in patients with type 2 diabetes: the Korean Sarcopenic Obesity Study (KSOS). *Diabetes Care*. 2010;33(7):1497–1499.
7. Aubertin-Leheudre M, Lord C, Goulet ED, Khalil A, Dionne IJ. Effect of sarcopenia on cardiovascular disease risk factors in obese postmenopausal women. *Obesity (Silver Spring)*. 2006;14(12):2277–2283.
8. Wu Y, Zhai L, Zhang D. Sleep duration and obesity among adults: a meta-analysis of prospective studies. *Sleep Med*. 2014;15(12):1456–1462.
9. Tan X, Chapman CD, Cedernaes J, Benedict C. Association between long sleep duration and increased risk of obesity and type 2 diabetes: a review of possible mechanisms. *Sleep Med Rev*. 2018;40:127–134.
10. Theorell-Haglöw J, Berglund L, Berne C, Lindberg E. Both habitual short sleepers and long sleepers are at greater risk of obesity: a population-based 10-year follow-up in women. *Sleep Med*. 2014;15(10):1204–1211.
11. Chien MY, Wang LY, Chen HC. The relationship of sleep duration with obesity and sarcopenia in community-dwelling older adults. *Gerontology*. 2015;61(5):399–406.
12. Cedernaes J, Schönke M, Orzechowski P, et al. Acute sleep loss results in genome-wide DNA methylation state and metabolic fuel utilization in humans. *Sci Adv*. 2018;4(8):eaar8590.
13. Lind L, Elmståhl S, Bergman E, et al. EpiHealth: a large population-based cohort study for investigation of gene-lifestyle interactions in the pathogenesis of common diseases. *Eur J Epidemiol*. 2013;28(2):189–197.
14. Kwon YJ, Jang SY, Park EC, Cho AR, Shim JY, Linton JA. Long sleep duration is associated with sarcopenia in Korean adults based on data from the 2008-2011 KNHANES. *J Clin Sleep Med*. 2017;13(9):1097–1104.
15. Girschik J, Fritschi L, Heyworth J, Waters F. Validation of self-reported sleep against actigraphy. *J Epidemiol*. 2012;22(5):462–468.
16. Baillet M, Cosin C, Schweitzer P, et al. Mood influences the concordance of subjective and objective measures of sleep. *Front Aging Neurosci*. 2016;8:181.

ACKNOWLEDGMENTS

The authors thank all the participants of the EpiHealth study and the nurses working at the EpiHealth test centers. Author contributions: XT and CB: study design, OET, EL, SE, and LL: data acquisition, XT and CB: data analysis and interpretation, XT and CB: manuscript writing. XT, OET, EL, SE, LL, HBS, and CB: critical review and revision. XT and CB are the guarantors of this work and, as such, had full access to

all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication July 20, 2018
Submitted in final revised form October 30, 2018
Accepted for publication November 6, 2018
Address correspondence to: Xiao Tan, PhD, Department of Neuroscience, Uppsala University, Husargatan 3, Box 593, 751 24 Uppsala, Sweden; Tel: +46-18-471-4326; Email: xiao.tan@neuro.uu.se

DISCLOSURE STATEMENT

Work for this study was performed at Uppsala University, Sweden. All authors have seen and approved this version of the manuscript. The EpiHealth study was funded by the Swedish Research Council. Work of CB was supported by the Novo Nordisk Foundation (NNF14OC0009349), the Swedish Research Council (2015-03100), the Swedish Folksam Foundation, and the The Family Ernfors Foundation. Work of XT was supported by the Åke Wiberg Foundation (M17-0088, M18-0169) and the Thuring Foundation (2017-00313, 2018-00365). The funders did not have any role in design of the study, interpretation of the discussed literature, or in the writing process. The authors report no conflicts of interest.