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Shorter sleep duration is associated with greater visceral fat mass in US adults: Findings from NHANES, 2011–2014

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ABSTRACT

Habitual declines in sleep duration and increased rates of obesity are public health concerns worldwide. Accumulating evidence suggests a prominent link between reduced sleep duration and weight gain. Our cross-sectional study investigated the relationship between sleep duration and body fat distribution in US adults. We extracted data for 5151 participants (2575 men and 2576 women) aged 18–59 years from the US National Health and Nutrition Examination Survey 2011–2012 and 2013–2014. Weekday or workday night-time sleep duration was estimated using an in-home interview questionnaire. Dual-energy x-ray absorptiometry scans were used to determine regional body fat mass (arms, legs, trunk [android and gynoid], and abdominal [subcutaneous and visceral]). Multiple linear regression and restricted cubic spline analyses were performed after adjusting for several demographic, anthropometric, and nutritional covariates. There was a significant negative association between sleep duration and visceral fat mass overall (β: −12.139, $P < 0.001$) and by sex (men: β: −10.096, $P < 0.001$; women: β: −11.545, $P = 0.038$), after adjusting for age, ethnicity, body mass index, total body fat mass, daily energy and alcohol intake, sleep quality and sleep disorder status. Sleep duration and visceral fat appeared to plateau at ≥ 8 h of daily sleep. Sleep duration is negatively associated with visceral fat mass accumulation during adulthood with possibly no benefits beyond 8 h of sleep per day. Mechanistic and prospective studies are required to confirm the effect of sleep duration on visceral adiposity and determine its causes.

1. Introduction

Obesity is a global health challenge, affecting approximately 600 million adults worldwide. In the United States alone, more than two-thirds of adults are considered obese (body mass index [BMI] ≥ 30 kg/m^2), which contributes to substantial metabolic and financial burden [1]. Increased adiposity throughout the lifespan is linked with several metabolic abnormalities such as insulin resistance, type 2 diabetes, non-alcoholic fatty liver, cardiovascular disease, and cancer [2]. The rapid increase in the prevalence of obesity may be associated with intrinsic (i.e. genetics) and extrinsic factors, including physical inactivity and over-nutrition [3].

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Accumulation of adipose tissue is sex-specific and interindividual differences in body fat distribution are linked to sex hormone profiles, genetics, and epigenetic mechanisms [4].

Emerging evidence suggests that sleep deprivation may be an important regulator in metabolic health. In the United States, sleep disorders affect approximately 70 million people, a critical issue that may be manifested due to physiological, psychological, and environmental factors [5]. Sleep is regulatory for metabolic function, including immune and hormonal status [6,7]. Specifically, sleep can modulate appetite and thus plays an important role in reducing obesity [8]. Additionally, research has also demonstrated a pronounced effect of sleep regulation on body fat distribution, which may further exacerbate metabolic health [9]. Indeed, sleep disorders and insufficient sleep can alter neuroendocrine system activity, a major mediator of whole-body metabolism [10]. Interestingly, a bidirectional relationship between sleep and body weight changes has been proposed given that weight loss strategies may promote better sleep quality [11] and decrease wakefulness after sleep onset [12]. However, considering the impact sleep disruption may impose on metabolic health, the majority of research has focused on elucidating its influence on body weight regulation.

At present, a plethora of studies have explored the relationship of sleep duration with regional body fat mass, without accounting for whole-body adiposity and the concurrent effect of other regions of adipose tissue. The purpose of this observational study was to comprehensively investigate the potential association between sleep duration with regional body fat mass in US adults.

2. Methods

2.1. Study design and participants

Data from study participants aged 18–59 years were collected from two consecutive survey cycles in NHANES: 2011–2012 and 2013–2014. The cut-off age was selected based on data availability for sleep duration, sleep quality, sleep disorder status, and body fat distribution.

2.2. Sleep duration and regional body fat mass assessment

Quantity of sleep, in terms of duration during the night on weekdays or workdays, was assessed through an in-home interview questionnaire, using the Computer-Assisted Personal Interviewing (CAPI) system. Responses ranged from 1 to 12, with 1 indicating 1 h of sleep to 12 indicating ≥12 h of sleep. Dual x-ray absorptiometry (DXA) scans were administered to assess regional body fat mass using Hologic Discovery model A densitometers. Regional body fat mass areas included the limbs, trunk (android and gynoid), and abdominal (subcutaneous and visceral). Fat mass was quantified in grams (g). Participants with no information on any of the above measures, were excluded from the study.

2.3. Covariates

Age (years), ethnicity (race), BMI (kg/m^2), total fat mass (g), daily energy (kcal), and alcohol intake (g) were classified as covariates alongside sleep quality and sleep disorder status. These variables were considered to be potential confounders in the relationship between body fat mass and sleep duration. With the exception of total fat mass, all covariates were considered as categorical data.

Participants were categorized by age into the following groups: 18–29, 30–39, 40–49, 50–59 years. Ethnic groups consisted of Mexican American, other Hispanic, non-Hispanic White, non-Hispanic Black, non-Hispanic Asian and other (multi) race. Energy and alcohol intake were computed through the average of the 24-h dietary recalls and categorized as low, moderate, or high. Low energy intake in men was considered <2000 kcal/d, moderate as 2000–3000 kcal/d, and high >3000 kcal/d. Low energy intake in women was considered <1600 kcal/d, moderate as 1600–2400 kcal/d, and high as > 2400 kcal/d. Low alcohol intake in men was considered <15 g/d, moderate as 15–30 g/d, and high as > 30 g/d. Low alcohol intake in women was considered <10 g/d, moderate as 10–20 g/d and high as > 20 g/d. A BMI of <18 kg/m^2 was classified as low, 18–24.9 kg/m^2 as moderate, and ≥25 kg/m^2 as high. Sleep quality was categorized as Yes/No responses based on self-reported difficulties in sleeping as reported to a general practitioner or other health professional. Sleep disorder status was classified as a Yes/No response based on diagnosis by a doctor or other health professional.

2.4. Statistical analyses

Multiple linear regression analyses were used to assess the association between daily sleep duration and body fat mass by adipose tissue region upon adjustment of all covariates. Sleep duration was treated both as a continuous (Model 1) and as a categorical (Model 2) variable. For the categorical variable, individuals were categorized into four groups by the amount of reported daily sleep: ≤6, 7–8, 9–10 or ≥11 h. Restricted cubic splines were employed to model the non-linear and dose-response relationship between sleep duration and regional body fat mass with three knots after adjustment. Results from the multiple regression analyses were described by sleep duration as a continuous variable. Additional confirmation of the effect and of the dose-response relationship in terms of significance was established using sleep duration as a categorical variable and by assessing the subgroup effect of sex. Collinearity in terms of linear intercorrelation between the explanatory and covariate variables in the multiple regression model was assessed using the variance inflation factor. Statistical significance was established as $p < 0.05$. Statistical analysis was ensued using IBM SPSS Statistics v28.

3. Results

3.1. Characteristics of study population

Data for sleep duration and regional body fat mass were available for 5151 participants. Baseline characteristics of sociodemographic, anthropometric, and nutritional relevance amongst all participants are outlined in Table 1 and by sex in Tables S1 and S2. When sleep duration was considered as a continuous variable, the study cohort consisted of 5125 participants and excluded 26 individuals with a reported scores of 12 or more hours of sleep duration (Table S3). The total cohort had a mean age of 37.4 years and was composed equally based on sex (≈50%). Participants were primarily non-Hispanic white (38%) and Black (22%). Participants had an energy intake that was primarily within the recommended range of consumption (44%), with low alcohol intake (81%), and high BMI (73%). The majority of participants reported no trouble sleeping (78%) and were mainly free of any sleep disorders (85%).

3.2. Sleep duration and regional body fat mass

Sleep duration was negatively associated with visceral fat mass when expressed as a continuous (Model 1: $\beta = -12.139$, $P < 0.001$) and categorical (Model 2: $\beta = -26.661$, $P = 0.002$) variable after adjustment for all covariates (Table 2). A difference of 1 h in daily sleep duration corresponded to an increase of 12.1 g in visceral fat mass (Model 1). No associations with limb (arms: $P = 0.992$; legs:

Table 1
Socio-demographic, anthropometric and nutritional characteristics of all participants (n = 5151). Values are expressed as count (percentage) unless otherwise specified.

Characteristics	
Age	
18-29	1658 (32)
30-39	1153 (22)
40-49	1198 (23)
50-59	1142 (22)
Sex	
Male	2575 (50)
Female	2576 (50)
Ethnicity	
Mexican American	698 (14)
Other Hispanic	489 (10)
Non-Hispanic White	1963 (38)
Non-Hispanic Black	1121 (22)
Non-Hispanic Asian	676 (13)
Other Race - Including Multi-Racial	204 (4)
Body mass index	
Low	72 (1)
Normal	1676 (33)
High	3403 (66)
Energy intake	
Low	1809 (35)
Moderate	2262 (44)
High	1080 (21)
Alcohol intake	
Low	4153 (81)
Moderate	412 (8)
High	586 (11)
Arm fat (g)	
Minimum	319.4
Average	1667.1 (12)
Maximum	7248.0
Leg fat (g)	
Minimum	574.5
Average	4890.5
Maximum	20863.1
Trunk fat (g)	
Minimum	2283.1
Average	12889.8
Maximum	47541.6
Abdominal fat (g)	
Minimum	241.4
Average	2070.4
Maximum	5893.1
Visceral fat (g)	
Minimum	26.8
Average	469.9
Maximum	1918.7
Subcutaneous fat (g)	
Minimum	111.0
Average	1600.5
Maximum	5264.6
Android fat (g)	
Minimum	296.6
Average	2352.1
Maximum	10626.5
Gynoid fat (g)	
Minimum	659.7
Average	4591.0
Maximum	16843.6
Total fat (g)	
Minimum	4902.0
Average	27173.9
Maximum	102288.7
Sleep duration (hr)	
Minimum	2.0
Average	6.8
Maximum	12.0
0–6 h	2064 (40)
7–8 h	2751 (53)
9–10 h	304 (6)
11–12+ hours	32 (1)
Sleep quality	
Trouble sleeping	1157 (23)
No trouble sleeping	3994 (78)
Sleep disorder status	
Yes	792 (15)
No	4359 (85)

P = 0.074), trunk (P = 0.051) (android [P = 0.157] and gynoid [P = 0.600]), and abdominal (P = 0.166) [subcutaneous (P = 0.471)] fat mass were found following adjustment. Subgroup analysis based on sex after adjustment of covariates revealed a significant negative association between sleep duration and visceral fat mass in men (β: −10.096, $P < 0.01$) and women (β: −11.545, P = 0.038), even after adjustment for covariates (Table 3, Table S4). Dose-response curves indicated a linear relationship between sleep duration and visceral fat mass, following adjustment for covariates (Fig. 1). A plateau in visceral fat mass changes was observed over 8 h of sleep duration per day in the restricted cubic spline of Model 2. No signs of linear multi-intercorrelation between the effect of sleep duration against other covariates on visceral fat mass were observed (Table S5).

4. Discussion

In this large US cohort aged 18–59 years, there was a significant negative association between sleep duration and visceral fat mass in men and women with a plateau effect at ≥ 8 h of sleep per day, after adjustment for sociodemographic, anthropometric, and nutritional covariates. Our study adds further evidence to the notion that chronic sleep restriction may be a potential contributor for visceral fat mass adiposity.

These findings may be clinically significant as visceral adiposity is associated with metabolic perturbations such as insulin resistance and type 2 diabetes [13], increasing the risk of endothelial and cardiometabolic dysfunctions [14,15]. These detrimental changes may be linked to circadian misalignments in the suprachiasmatic nuclei of the brain's anterior hypothalamus, a major regulator of sleep/wake cycles in mammals [16] and abnormalities in brain tissue volume [17]. Under conditions of sleep deprivation, experimental work has revealed that several brain regions are involved in dysfunctional cognitive, motivation, and reward processing [18–20]. In particular, appetite evaluation centers within the frontal and insular cortex that are implicated in food choice desirability assessment were blunted, with amygdala reactivity amplified at a subcortical level. These altered brain mechanisms are significant contributors of neuroendocrine and appetite hormone dysregulation [10,21]. In this way, it may be speculated that a concomitant decrease in leptin along with elevated ghrelin levels and hyperactivity of the orexin system, could explain higher energy intake and subsequent weight gain upon sleep restriction [22,23]. Other hormones affected by sleep restriction include testosterone, which decreased by 10–15% in a cohort of young healthy men who underwent 5 days of inadequately short total sleep time (5 h), a condition estimated to affect at least 15% of the US working population. Additionally, insufficient sleep may also undermine dietary interventions to counteract increased adiposity levels as a result of increased insulin resistance, reduced glucose tolerance and compensatory hyperinsulinemia. In particular, short-term sleep duration (~5.5 h/d) is accompanied by lower body fat losses compared to individuals with adequate sleep (~8.5 h/d) [24]. These changes were followed by a greater fat-free mass reduction along with increased subjective feelings of hunger, highlighting the potential ramifications that sleep loss may impose on body composition and sleep loss interventions. Taken together, strategies to

Table 2
Multiple linear regression analysis of the association between sleep duration and regional body fat mass after covariate adjustment of age, ethnicity, BMI, total fat mass, daily energy, and alcohol intake, sleep quality and sleep disorder status. Sleep duration was treated both as a continuous (Model 1) and as a categorical (Model 2) variable.

Regional Body Fat Mass	Model 1			Model 2[a]		
	β	P	R^2	β	P	R^2
Arms	0.025	0.992	0.917	1.785	0.756	0.917
Legs	−14.674	0.074	0.884	−23.537	0.194	0.884
Trunk	30.019	0.051	0.951	46.951	0.166	0.951
Abdominal	4.221	0.166	0.924	9.531	0.155	0.924
Visceral	−12.139[b]	<0.001**	0.624	−26.661[b]	0.002	0.624
Subcutaneous	−1.777	0.471	0.926	−2.997	0.581	0.926
Android	5.888	0.157	0.919	14.172	0.122	0.919
Gynoid	−3.138	0.600	0.918	−4.452	0.736	0.918

**$P = 0.000828$.
[a] Participants were categorized in to four groups by the amount of reported daily sleep: ≤6, 7–8, 9–10 or ≥11 h.
[b] Unstandardized simple linear regression coefficient of sleep duration against predicted visceral fat mass following multiple linear regression analysis.

counteract the changes of visceral adipose tissue in parallel with lean mass losses derived by chronic sleep deprivation may, in part, assist combat metabolic perturbations pertinent to disproportionate body fat accumulation. Future studies should also evaluate changes in sleep duration during fat loss strategies, considering its impact on body composition.

This is the first study to comprehensively explore the association between sleep duration and regional body fat mass. Multiple studies have proposed an optimal sleep duration based on the notion that a higher risk of obesity and increased adiposity coincides with a daily sleep duration of <8 h [25–27]. However, it is unclear what factors may contribute to differential effects of sleep duration on visceral fat mass accumulation considering the absence of a beneficial effect over 8 h of sleep. Prospective studies aimed at verifying the effect of sleep duration at different chronotypes on visceral fat mass are imperative. These studies will allow for specific recommendations on sleep duration in adults for decreasing the potential risk of higher visceral adiposity and obesity.

Our analysis revealed that sleep duration was not associated with arm, leg, trunk (android and gynoid) and abdominal (subcutaneous) fat mass. These findings are inconsistent with previous studies that reported a link between a short sleep duration and greater trunk, android and gynoid [28,29], abdominal, subcutaneous [30], and total fat mass [31]. However, these previous studies investigated the relationship between sleep quantity and regional body fat depots concurrently and from the same cohort. In this context, studies have proposed distinct associations among different body fat areas with brain processes that may be responsible for the short sleep-induced metabolic impact. For example, visceral adiposity has been correlated with cerebellar changes in both function and structure, which are brain regions involved in cognitive, motor, and emotional mechanisms [32]. In addition, cross-sectional associations have revealed an inverse association between total brain volume and visceral fat compared to abdominal adiposity [33], whereas brain atrophy has been positively associated with leptin insufficiency [34]. These changes suggest a fat depot specific effect of sleep loss, and support the recommendation of a comprehensive regional fat assessment amongst studies that investigate changes in body fat during periods of sleep deprivation.

Table 3
Multiple linear regression analysis of the association between sleep duration and regional body fat mass after covariate adjustment of age, ethnicity, BMI, total fat mass, daily energy, and alcohol intake, sleep quality and sleep disorder status. Sleep duration was treated both as a continuous (Model 1) and as a categorical (Model 2) variable.

Visceral Fat Mass	Model 1			Model 2[a]		
	β[b]	P	R^2	β[b]	P	R^2
Men	−10.096	<0.001**	0.640	−23.379	0.003	0.639
Women	−11.545	0.038	0.617	−24.177	0.033	0.617

**$P = 0.000548$.
[a] Participants were categorized in to four groups by the amount of reported daily sleep: ≤6, 7–8, 9–10 or ≥11 h.
[b] Unstandardized simple linear regression coefficient of sleep duration against predicted visceral fat mass following multiple linear regression analysis.

4.1. Strengths and limitations

The present study is the first to investigate the relationship between sleep duration and regional body fat mass, using a large nationally representative database based on the US population. Multiple covariates were adjusted to accurately estimate the linear relationship between these two variables. However, our study has several limitations. Cross-sectional surveys cannot provide a causal relationship between dependent and independent variables. Additionally, assessment of sleep quantity, quality and disorder status were made based on a single in-home interview questionnaire (CAPI) system, without reporting on other aspects of sleep such as sleep latency, habitual sleep efficiency, use of sleeping medication, and daytime dysfunction which are included in more sophisticated sleep questionnaires such as that of Pittsburgh Sleep Quality Index [35]. Equally, information on physical disorders that have proposed association with obesity, such as obstructive sleep apnoea and sleep-related movement disorders, were not controlled for due to lack of data availability. Further, body fat mass was estimated via DXA, which is a less reliable assessment tool compared with computed tomography and magnetic resonance imaging. Moreover, no data were available regarding employment patterns, given that different working conditions (i.e. shift working) are linked with circadian misalignment and reduced sleep duration [36]. In this context, it has also been suggested that sleep duration may be influenced by day of week (i.e. weekdays vs weekend days) [37], the inclusion of naps [38], and seasonal changes in sleep pattern [39]; data of this nature is not available in NHANES. It also bears mentioning that whilst alcohol was included as a covariate, caffeine intake was not accounted for in our investigation. Finally, body fat distribution data was collected in adult individuals aged 18–60 years, and thus conclusions in terms of associations between sleep duration and body fat distribution in younger or older populations cannot be extrapolated.

5. Conclusions

Sleep duration is negatively associated with visceral fat mass in US adults aged between 18 and 59 years of age. No associations between sleep duration and arm, leg, trunk (android and gynoid)

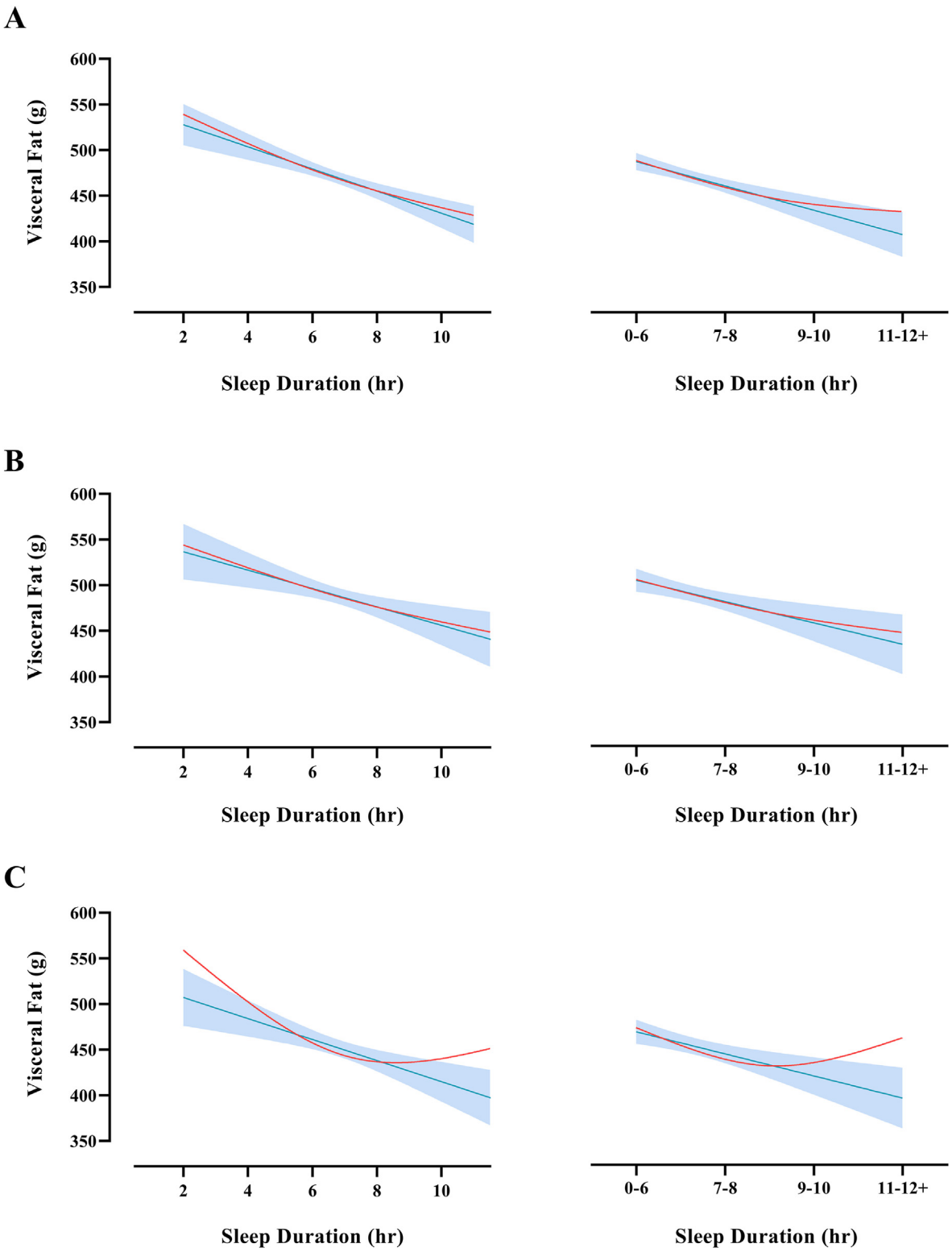

Fig. 1. Linear and spline models of sleep duration and visceral fat mass in all participants.
(A) and by gender (males: B, females: C) after covariate adjustment of age, ethnicity, BMI, total fat mass, daily energy, and alcohol intake, sleep quality and sleep disorder status. Sleep duration was treated both as a continuous (Model 1) and as a categorical (Model 2) variable. Participants in Model 2 were categorized in to four groups by the amount of reported daily sleep: $\leq$ 6, 7-8, 9-10 or $\geq$ 11 hours..

and abdominal (subcutaneous) fat mass were found. Effects of sleep duration on visceral fat mass accumulation may be explained by dysregulated brain activity at regions linked with neurohormonal imbalance. Prospective and mechanistic studies could assist in verifying with greater accuracy the effect of sleep duration on visceral adiposity and determine its causes. Future clinical and experimental studies may elucidate strategies to counteract the potentially negative impact on body fat composition in response to chronic and cumulative sleep loss.

Statement of significance

In Western societies, obesity has raised in parallel with a habitual decline in hours of sleep. Emerging evidence suggests a prominent link between reduced sleep duration and weight gain. To address this, we investigated the association between sleep duration and body fat distribution in US adults. Our cross-sectional study of 5151 US participants aged 18–59 years revealed that sleep duration was negatively associated with visceral fat mass, reaching a plateau effect at $\geq$ 8 h of sleep per day. Prospective and mechanistic investigations may help to more precisely confirm and pinpoint the origins of the relationship between sleep duration and visceral obesity.

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CRediT authorship contribution statement

Panagiotis Giannos: Conceptualization, Data curation, Formal analysis, Writing – original draft, Project administration. **Konstantinos Prokopidis:** Conceptualization, Data curation, Formal analysis, Writing – original draft, Project administration. **Darren G. Candow:** Writing – review & editing. **Scott C. Forbes:** Writing – review & editing. **Kamil Celoch:** Writing – review & editing. **Masoud Isanejad:** Writing – review & editing. **Vanja Pekovic-Vaughan:** Writing – review & editing. **Oliver C. Witard:** Writing – review & editing. **Brendan M. Gabriel:** Writing – review & editing. **David Scott:** Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.sleep.2023.03.013.

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