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Habitual sleep variability, mediated by nutrition intake, is associated with abdominal obesity in adolescents

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Abstract

Objective—To investigate habitual sleep duration (HSD) and habitual sleep variability (HSV) in relation to abdominal obesity and nutrient intakes as mediating factors in adolescents.

Methods—We analyzed data from 305 adolescents who participated in the Penn State Child Cohort follow-up examination. An actigraphy device was used for 7 consecutive nights to calculate HSD and HSV. Abdominal obesity was assessed by dual-energy x-ray absorptiometry. The Youth/Adolescent Food Frequency Questionnaire was used to obtain daily total caloric, protein, fat, and carbohydrates intakes. Linear regression models were used to associate HSD and HSV with abdominal obesity and to qualitatively identify mediating factors. The mediating effect was quantitatively estimated by mediation models.

Results—After adjusting for major covariates and HSD, higher HSV was significantly associated with abdominal obesity measures. For example, with 1-hour increase in HSV, android/gynoid fat ratio and visceral fat area increased by 0.02 cm^2 (standard error = 0.01, $p = 0.03$) and 6.86 cm^2 (standard error = 2.82, $p = 0.02$), respectively. HSD was not associated with abdominal obesity in HSV-adjusted models. Total caloric, fat, and carbohydrate intakes were significant mediating factors. For instance, 20% of the association between HSV and visceral fat can be attributed to carbohydrate intake.

Conclusions—Higher HSV, not HSD, is significantly associated with abdominal obesity, which can be partially explained by increased caloric intake, especially from carbohydrate, in adolescents. This study suggests that more attention should be paid to establish and maintain regular sleep patterns in adolescents.

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Keywords

Sleep variability; Abdominal obesity; Nutrition Intake

1. Introduction

The worldwide epidemic of overweight and obesity among children is of great concern, as childhood overweight and obesity track into adulthood [1]. It is has been reported that obese children are approximately seven times more likely to become obese adults compared to normal-weight children [2]. Previous literature has also demonstrated associations between childhood obesity and increased risk of metabolic alterations and disease, such as insulin resistance, dyslipidemia, and metabolic syndrome [3,4]. More importantly, excessive accumulation of adipose tissue in the abdominal region is an independent and more cardiometabolically relevant risk factor than general obesity, as measured by body mass index (BMI). For example, visceral fat accumulation is associated not only with quantitative changes in serum lipid but also with qualitative changes in lipoproteins, such as small dense low-density lipoproteins (LDL), and conveys greater insulin resistance than other adipose tissue [5].

In parallel with the marked increase in the prevalence of obesity, sleep deprivation and sleep disturbances have become a frequent complaint. In an analysis carried out by the U.S. Centers for Disease Control and Prevention (CDC) in 2009, 35.3% of U.S. adults had less than 7 hours of sleep per night, compared with approximately 29% in the 2004–2006 National Health Interview Survey [6,7]. Previous studies also indicated that self-reported short sleep duration and insomnia were prevalent in adolescents and young adults [8,9]. Several studies reported an association between subjectively measured short sleep duration and obesity in both children and adults [10–13]. Since subjectively measured short sleep duration has a weak correlation with objectively measured sleep duration [14], it can be argued that subjectively measured sleep duration may serves as a surrogate of stress, anxiety, and depression [15]. Within this theoretical framework, previous studies reported associations between anxiety, depression, emotional stress, and obesity [16–18]. Therefore, the observed association between subjectively-measured short sleep duration and obesity may be confounded by participants' psychological conditions [15,19]. On the other hand, the relationship between objectively measured sleep duration and obesity has been inconsistent [19–23].

Because of the improvement and availability of actigraphy for multiple nights of sleep measurements, objectively measured habitual sleep pattern, represented by habitual sleep duration and intraindividual variability of sleep duration, has been used in the sleep field [24,25]. Although the habitual sleep duration is an indicator of the average length of sleep, intraindividual variability focuses on the individual's sleep–wake rhythm. The study of the variability of sleep duration could provide new insight into sleep research. However, little is understood regarding the association between sleep duration variability and obesity. Recently, Kjeldsen et al. reported that both habitual sleep duration and sleep duration variability were associated with dietary risk factors for obesity in Danish school children

[26]. Specifically, in a cross-sectional analysis of 676 adolscents, these investigators found that short sleep duraton and high sleep variability were related to increased consumption of energy-dense food and sugar-sweetned beverages. Therefore, it is plausible that habitual sleep pattern is associated with excessive food and energy intake and consequently is related to obesity.

Therefore, this study was designed to investigate the associations between objectively measured habitual sleep duration (HSD), habitual sleep variability (HSV), and abdominal obesity in a population-based sample of adolescents. Our secondary objective was to examine the potential mediating role of energy intake in the habitual sleep and abdominal obesity relationship.

2. Methods

2.1. Population

We used available data from 421 adolescents who completed the follow-up examination of the Penn State Children Cohort (PSCC) study. Recruitment methods and examination procedures for the PSCC baseline study have been published elsewhere [27,28]. A total of 700 children aged 6–12 years participated in the baseline examination, conducted in 2002–2006. Among the 700 subjects, 421 returned and completed the follow-up examination during 2010–2013, yielding a response rate of 60%. Loss to follow-up was mainly due to subjects moving out of the central Pennsylvania area. However, no major difference in the baseline demographic characteristics was observed between subjects who participated in the follow-up study examination and those who did not. The participants were examined in the Clinical Research Center in Pennsylvania State University College of Medicine. After undergoing a whole-body dual-energy x-ray absorptiometry (DXA) scan, a detailed physical examination and questionnaire-based data collection protocol were performed. Actigraphy (GT3X+; ActiGraph, Pensacola, FL, USA) was used to measure physical activity level and sleep duration. The participant stayed overnight in a sleep laboratory to complete a standardized polysomnography (PSG) recording. After collecting morning blood, saliva, and urine samples, the participants were released with the actigraphy device and a set of questionnaires about their habitual behaviors. The study protocol was approved by Penn State University College of Medicine Institutional Review Board. Written informed consent was obtained from participants if they were at least 18 years of age or from their parents or legal guardians if they were younger than 18 years.

2.2. Sleep variables

The actigraphy device worn on the wrist of nondominant hand during bedtime was used to assess sleep duration for 8 consecutive nights over the study period, in combination with the sleep diary that recorded “bed time” and “out of bed time” on a nightly basis. The actigraphy data were exported to a designated computer for analysis. After removing artifacts, the actual sleep durations were obtained by using ActLife 6 software (ActiGraph LLC, Pensacola, FL, USA). Sleep data for the first night was excluded from the calculation, as it was measured under a 9-hour sleep protocol in a laboratory environment. HSD and HSV were computed to assess participants’ habitual sleep patterns. The average of sleep durations

across 7 nights was used to represent HSD. The intrasubject standard deviation (SD) of the 7-night sleep duration was used to represent HSV. Participants with less than 5 nights, i.e., less than 70% of 7 nights, of sleep data, were excluded from the analysis.

2.3. Abdominal obesity variables

Whole-body DXA scan was used to measure the adipose tissue distribution in abdominal region. DXA scan was performed by using Hologic Discovery W scanner (Hologic Inc., Walthma, MA, USA). This method uses two beams of low-energy x-ray that collected by the detectors after attenuation by the body tissue through which they have passed. Soft tissue is resolved by using mass attenuation coefficients derived from tissue equivalent standards for fat-free and fat tissue. Subjects were required to remove all metal, plastic, and rubber materials to avoid any impact on x-ray beams. According to our standardized operation protocol, daily quality control and calibration were performed on the DXA machine to ensure the validity of the data. Android region, gynoid region, visceral adipose tissue (VAT), and subcutaneous adipose tissue (SAT) were selected as regions of interest (ROI) to assess abdominal obesity. Detailed ROI-defining methods were as described elsewhere [29,30]. To minimize the misclassification of ROI, all ROI identified by Hologic APEX 4.0 software (Hologic Inc., Bedford, MA, USA) were visually verified by a single experienced investigator. The total fat area (TAT area) in the abdominal region was calculated as the sum of VAT and SAT areas. The android/gynoid fat mass ratio (AGR), android/whole body fat mass proportion (AWP), gynoid/whole body fat mass proportion (GWP), VAT, SAT, and TAT areas were used in this report.

2.4. Nutrition intake variables

To investigate the potential mediating role of energy intake in the relationship between habitual sleep pattern and abdominal obesity, a Youth/Adolescent Questionnaire [31,32] was used to assess participants' daily energy intake. Briefly, the participants were asked to report the frequency of consumption of 152 food items over 1 year before the study. Frequencies for each of the 152 food items were analyzed and converted into a series of nutrient indices representing daily nutrition intake. For this report, we included daily total energy, total fat, protein, and carbohydrate intakes to represent the participants' dietary habits. Subjects with a daily total energy intake less than 500 kcal or more than 5000 kcal were excluded from the analysis because of implausible responses to the questionnaire.

2.5. Other covariates

Subjects' demographic information, including age, race, and sex, was collected via a self-administered questionnaire. Subjects' height and weight were measured to calculate BMI percentile as a marker of general obesity. Age- and gender-adjusted BMI percentile was calculated based on the formula and data from the 2000 CDC growth charts.

2.6. Statistical analysis

Among 421 participants in the follow-up examination, 116 individuals were excluded from the analysis due to insufficient nights of sleep data (n = 94), missing data on DXA variables (n = 25), and/or implausible daily total caloric intake (n = 7). Thus, the effective sample size

for this report is 305. Summary statistics of the demographics were calculated as mean (SD) and proportions. Linear regression models were used to assess the impact of habitual sleep pattern on abdominal obesity. Initially, sleep variables were included in the models individually; that is, only one sleep variable entered the model as the independent variable to investigate the relationship between habitual sleep pattern and abdominal obesity. Both unadjusted and major demographic covariates, including age, sex, and race, were fitted in an adjusted model. To focus on the association between habitual sleep pattern and abdominal obesity, BMI percentile was purposely controlled in the adjusted models. We then included both HSD and HSV in the same model to control for each other as our final model, together with the aforementioned covariables.

To explore the potential mediating role of energy intake in the association between habitual sleep pattern and abdominal obesity, total caloric and macronutrients intake were further adjusted in the models. The differences due to the additional adjustment of the potential mediating factors were used to elucidate the mediating role of these energy intake–related factors qualitatively. All of the above analyses were performed using the SAS statistical package version 9.3 (SAS Institute, Cary, NC, USA).

Finally, an R package, “Mediation” [33], was used to quantitatively estimate the mediating effects and direct effects of various energy intake–related factors in the association between HSV and VAT. The R-based (version 3.1.0) Mediation package estimated the confidence intervals using bootstrap with 1000 resamples, with a two-sided p value of ≤0.05 indicating statistical significance.

3. Results

3.1. Demographic characteristics of the study population

The demographic characteristics of the population are shown in Table 1. The mean (SD) age of the participants was 16.7 (2.3) years, with 53% male and 79% white. The average BMI percentile of the sample was 66. The mean (SD) of HSD and HSV were 7.0 (0.8) and 1.2 (0.6) hours, respectively. On average, the participants slept 0.4 hour longer during weekends than weekdays. Such a difference in sleep duration between weekdays and weekends may serve as a major contributor of HSV. However, the HSV cannot be fully explained by the difference in sleep duration between weekdays and weekends.

3.2. Association between habitual sleep pattern and abdominal obesity

The associations between 1-hour increases in HSD and HSV and DXA measures are presented in Table 2. The regression coefficient (β), standard error (SE), and p value from both the unadjusted model and various covariates adjusted models (Model 1–Model 2d) shows the impacts of HSD and HSV on DXA measurements separately. In the unadjusted models, increased HSV is consistently associated with increased abdominal adipose tissue distribution, except GWP. In contrast, HSD was associated with decreased AGR and VAT area but increased GWP. After controlling for the major demographic variables and BMI percentile (Model 1), the significance of the associations between HSV and DXA variables remained the same, whereas the magnitude of the β values were substantially reduced. For

example, with a 1-hour increase in HSV, the associated increase in VAT area deceased from 10.2 cm^2 (SE = 3.7) to 6.6 cm^2 (SE = 2.8, $p = 0.02$). However, the same adjustment of major covariables completely diminished any significant association between HSD and abdominal obesity. These findings are confirmed by purposely including HSD and HSV in the same model (Model 2). Although no statistically significant association between HSD and abdominal obesity was found in the adjusted models, there is a consistent trend that HSD is negatively related to abdominal fat. Additional adjustment for Tanner stage did not alter the results.

3.3. Mediating effect of nutrition intake

Because HSV, but not HSD, was significantly associated with nutrition intake [34] (e.g., a 1-hour increase in HSV is associated with approximately a 200-kcal increase in total caloric intake), we further investigated the potential mediating role of energy intake in the association between HSV and abdominal obesity. The relationships between HSV and DXA measurements, after additional adjustment of nutrition intake, are shown in Models 2a–2d of Table 2, respectively, for the adjustments of total caloric, total fat, carbohydrate, and protein intakes. Comparing the regression coefficients in Model 2, the regression coefficients in Models 2a–2c were substantially decreased when controlling for total calorie, total fat, and carbohydrate, but no major change when adjusting for protein intake (Model 2d). For example, the impact of 1-hour increase in HSV on AGR decreased by 16% (0.019 vs 0.016) after adjusting for total caloric or total fat intake. The β value between HSV and VAT area decreased by 20% (5.5 vs 6.9), after controlling for carbohydrate intake. This suggests that a notable proportion of association between increased HSV and abdominal obesity can be explained by energy intake, especially from fat and carbohydrate.

Because the largest attenuation in the regression coefficient after adjusting for the energy intake factors was observed in the association between HSV and VAT area, we further performed the mediation analysis to quantify the “direct,” mediation, and total effects of 1-hour increase in HSV on VAT area. The results from these quantitative mediation analyses are presented in Table 3 as the β values, 95% CIs, and *p* values. Briefly, there are significant mediation effects from total caloric and carbohydrate intakes, whereas there remained a significant “direct” effect between HSV and VAT area. In other words, HSV was associated with VAT area, partially through the mediating of energy intake, whereas a significant amount of the association was through other factors, quantified as the “direct” effect from these models. For example, a 1-hour increase in HSV was associated with a 6.7-cm^2 higher VAT area, which was equivalent to 12% of the total VAT area (55.9 cm^2), of which approximately 20% (1.3 cm^2) can be attributed to the mediating effect of carbohydrate intake. However, 80% of the change was due to other factors (comprising a “direct” effect).

4. Discussion

We observed that increased HRV, not HSD, was associated with abdominal adiposity in a sample of healthy adolescents. Such an association could be partially attributed to excessive food consumption. These associations persisted after adjusting for major covariates,

including general obesity as measured by BMI percentile. It indicated that greater variability in sleep duration is an independent risk factor for abdominal obesity during adolescence.

Adipose tissue has been identified as an active endocrine and paracrine organ that releases large number of cytokines and bioactive mediators, such as leptin, adiponectin, interleukin-6, and tumor necrosis factor–α, which influence not only body weight but also insulin resistance, diabetes, lipid levels, coagulation, fibrinolysis, inflammation, and atherosclerosis [2–4]. More importantly, it has been generally accepted that the accumulation of abdominal fat is an independent and more metabolically relevant risk factor for cardiometabolic abnormalities than is general obesity [4]. Our findings suggested that sleep–wake rhythm plays a critical role in sleep hygiene, which may eventually contribute to risk of cardiometabolic disorders.

4.1. Sleep duration and abdominal obesity

Although habitual sleep duration showed a negative association with obesity in the unadjusted models, the relationship was eliminated after adjusting for major demographic covariables and BMI percentile. It is consistent with majority of the previous studies that reported a lack of association between sleep duration and obesity [19–21]. For example, Vgontzas et al. reported that sleep duration, measured by actigraphy, was not related to incident obesity [21]. Although some investigators reported a significant association [22,23], there were some major limitations. For instance, Theorell-Haglow et al. [23] mearesured sleep duration based on 1 night of PSG in the laboratory environment, which may not represent the participants' habitual sleep duration. It is worth noting that the lack of association should not be generalized to adolescents whose habitual sleep duration is outside the range of our data, for which the mean sleep duration was 7 hours per night, with an SD of less than 1 hour. It should also be pointed out that there is a consistent trend indicating a negative association between habitual sleep duration and obesity measures, although none of the regression coefficients reached statistical significance. New studies with more heterogeneous habitual sleep duration may be warranted.

4.2. Sleep variability and abdominal obesity

Some authors have reported significant associations between objectively-measured, night-to-night sleep variability and stress, depression, and insomnia [24,25]. However, the association of sleep variability with obesity, especially abdominal obesity, is rarely investigated. The key finding from our data is the consistent positive association between HSV and abdominal obesity measures, even after controlling for major covariates and HSD. Specifically, with a 1-hour increase in HSV, the fat content increased by more than 10% of the total fat in the corresponding region (e.g., 6.7 cm^2/55.9 cm^2 for VAT area). Furthermore, the findings from the models when both HSD and HSV were entered into the same model (e.g., the finding that only HSV, but not HSD, was significantly related to abdominal obesity) are suggestive of a more important role of HSV in the development of abdominal obesity than the of HSD. We controlled for BMI percentile in all of our models; thus our findings are independent of general obesity and represent the first study to report such an association, to our knowledge. Excessive android/gynoid fat mass and their respective ratio to whole-body fat mass has been repeatedly used in previous literature as a marker of

abdominal obesity and reported as a risk factor for cardiovascular morbidities [35]. Abdominal fat tissue, especially visceral fat, is more lipolytic and metabolically relevant than other fat tissues, and therefore a more prominent risk factor for insulin resistance and other cardiometabolic dysfunction [5]. Our findings on the relationship between sleep variability and these DXA measures suggested a critical role of establishing and maintaining a regular sleep–wake rhythm in preventing and reducing the risk of abdominal obesity and, consequently, cardiometabolic disorders.

4.3. Mediating role of nutrition intake

The mechanism linking sleep pattern and abdominal obesity is unclear. Galli et al. reported that short sleep and sleep apnea were related to higher energy intake, especially from fat, in a sample of obese subjects with chronic sleep deprivation [36]. In a more recently published study, Kjeldsen et al. reported that high sleep duration variability, measured by actigraphy, was associated with dietary risk of obesity [26]. Difference in sleep duration between weekday and weekend, which is a contributor to sleep variability, may also relate to changes in eating behavior [37]. Therefore, it can be hypothesized that obesity-promoting diet may play a mediating role in the relationship between HSV and abdominal obesity. In this study, we attempted to investigate the mediating roles of energy intake in the HSV and abdominal obesity relationship using two methods. First, the energy-mediating pathway is supported by the substantial attenuation in the regression coefficients of HSV in relation to abdominal obesity measures, after entering the potential mediating factors such as total caloric and carbohydrate intakes. Second, we confirmed the above qualitative findings using the recently available R package Mediation so that we could quantify the actual contribution from the mediators and the contribution from the “direct” effect. In general, although energy intake was identified as an important mediating factor in our data, the HSV and abdominal obesity relationship cannot be fully explained by energy intake alone. Thus, there are other etiological pathways that we can only postulate. For instance, fatigue, a known correlate of variability in sleep duration in adolescents [38], may be associated with less physical activity, and thus, be associated with obesity. It is also plausible that other sleep-related psychological comorbidities, including emotional stress, depression, and anxiety, may be mediating factors because of their known association with sleep variability [24]. Previous longitudinal data also implied a relationship between depression in adolescence and subsequent obesity [18,39,40]. For example, Goodman and Whitaker reported that depressed adolescents had a twofold higher risk of developing obesity in a 1-year cohort study [39].

4.4. Strengths and limitations

Several strengths of this study are worth noting. First, we used objectively measured sleep duration over 7 consecutive nights, including weekend nights, to capture habitual sleep patterns. Most previous studies used subjectively reported sleep duration [10–12], which are highly susceptible to biases or may serve as a surrogate of sleep disturbances and emotional stress [14]. Previous studies using objective measures of sleep duration used shorter-term PSG or actigraphy, except for three studies [24–26]. However, these studies did not focus on sleep variability or abdominal obesity. Second, we used DXA to assess abdominal obesity. Unlike anthropometric measurements such as waist circumference, DXA provided highly accurate and precise direct measurements of abdominal fat distribution, especially inside the

abdominal wall. Although computed tomography (CT) and magnetic resonance imaging (MRI) are considered as the gold standard to assess abdominal obesity, the radiation, time, and cost associated with these techniques has limited their availability and use in research settings. As DXA showed a strong correlation ($r > 0.9$) with these gold standard technologies, it is considered as a practical and promising tool to assess fat distribution in larger samples [41]. Third, we purposely included the HSD and HSV in the same regression model to control for each other, and adjusted for BMI percentile in all models to focus on the impact of habitual sleep pattern on abdominal obesity, which has a larger impact on cardiometabolic risk. As a result, we were able to elucidate the potential impact of habitual sleep duration and sleep duration variability on abdominal obesity beyond general obesity. Finally, we used the mediation models to quantify the mediating role of energy intake. This method enabled us to differentiate and quantify the mediation effects of energy intake and of other sources ("direct" effect).

Our study also has some potential limitations. First, it is possible, but very remotely so, that a participant may accidentally forget to wear the actigraphy device. However, the actigraphy data were reviewed by an experienced investigator to identify and remove artifacts, and to confirm compliance based on participants' daily logs. To minimize the impact of noncompliance, we further excluded those subjects with less than 5 nights of good-quality actigraphy data. Second, only 60% of the population-based random sample of the PSCC cohort participated in the follow-up examination, which may introduce selection bias. However, no significant difference in demographic characteristics was found between subjects who participated in the follow-up study and those lost to follow-up. Third, the average habitual sleep duration in our sample of adolescents was 7 hours, with an SD of less than 1 hour. Such a homogeneous sample may yield limited statistical power to detect the association between sleep duration and obesity. Moreover, although the latest technology and computing algorithm enabled DXA to distinguish visceral fat from subcutaneous fat, it is less precise than CT. DXA has also been reported to overestimate fat tissue and to underestimate muscle tissue, especially for morbidly low-weight individuals [41]. However, as our participants were healthy adolescents, it is less prone to this misclassification. Although less likely, our findings may be confounded by other, uncontrolled covariates. Finally, as this report is based on a cross-sectional analysis of the data, causal inference cannot be drawn from this study.

5. Conclusions

In conclusion, results from this study suggest that in population-based healthy adolescents, higher objectively measured habitual sleep variability, not habitual sleep duration, is significantly associated with abdominal obesity, independent of general obesity. More importantly, this association can be partially explained by higher energy intake, especially of carbohydrate. Therefore, establishing and maintaining a regular sleep pattern may reduce the risk of cardiometabolic disorders such as insulin resistance, dyslipidemia, and metabolic syndrome. However, to a large extent, the association cannot be explained by energy intake; thus we suggest that more studies are needed to identify other mediating factors for such an association.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Habitual sleep duration is not associated with abdominal obesity.
- High sleep variability is significantly related to abdominal obesity.
- Excessive energy intake is a mediating factor in the association.
- Establishing a regular sleep pattern may reduce the risk of abdominal obesity.

Table 1

Demographic characteristics of the study population.

Characteristic	Overall (N = 305)	Male (n = 160)	Female (n = 145)	*p*
Age (years)	16.7 (2.3)	16.5 (2.3)	16.9 (2.2)	0.07
White (%)	78.7	81.2	75.9	0.27
BMI percentile	66.0 (28.1)	64.1 (29.9)	68.1 (25.8)	0.22
HSD (h)	7.0 (0.8)	6.9 (0.8)	7.2 (0.9)	<0.01
HSV (h)	1.2 (0.6)	1.2 (0.6)	1.2 (0.7)	0.26
Weekday sleep duration (h)	6.9 (0.9)	6.7 (0.8)	7.1 (1.0)	<0.01
Weekend sleep duration (h)	7.3 (1.4)	7.2 (1.3)	7.4 (1.4)	0.29
AGR	0.36 (0.11)	0.38 (0.11)	0.33 (0.10)	<0.01
AWP (%)	6.3 (1.5)	6.3 (1.6)	6.3 (1.4)	0.80
GWP (%)	18.0 (2.4)	16.8 (1.8)	19.4 (2.3)	<0.01
VAT area (cm^2)	55.9 (39.4)	62.2 (35.1)	57.2 (43.7)	0.28
SAT area (cm^2)	222.9 (159.0)	161.1 (138.8)	291.6 (152.0)	<0.01
TAT area (cm^2)	282.8 (192.5)	223.3 (171.2)	348.9 (193.8)	<0.01
Total caloric intake (kcal)	1782.4 (704.8)	1863.2 (784.0)	1693.3 (595.5)	0.03
Total fat intake (g)	64.3 (27.4)	68.0 (30.1)	60.2 (23.5)	0.01
Carbohydrate intake (g)	235.0 (96.4)	244.3 (106.8)	224.7 (82.5)	0.07
Protein intake (g)	70.7 (29.0)	73.3 (32.0)	67.7 (25.0)	0.09

Results are expressed as mean (SD) and percentage for continuous and categorical variables, respectively.

Table 2

Regression coefficients (SE) and *p* values in association between habitual sleep pattern and DXA measurements.

	Unadjusted β (SE)	Model [a] β (SE)	Model 2[b] β (SE)	Model 2a[c] β (SE)	Model 2b[d] β (SE)	Model 2c[e] β (SE)	Model 2d[f] β (SE)
AGR							
HSV	0.023 (0.010) *	0.018 (0.008) *	0.019 (0.008) *	0.016 (0.008) *	0.016 (0.008) *	0.015 (0.008)	0.018 (0.008) *
HSD	−0.017 (0.008) *	−0.003 (0.006)	−0.003 (0.006)	−0.004 (0.006)	−0.004 (0.006)	−0.004 (0.006)	−0.003 (0.008)
AWP							
HSV	0.34 (0.14) *	0.21 (0.11) *	0.21 (0.11) *	0.19 (0.11)	0.20 (0.11) *	0.19 (0. 11)	0.21 (0.11) *
HSD	−0.15 (0.10)	−0.02 (0.08)	−0.01 (0.08)	−0.03 (0.08)	−0.02 (0.08)	−0.03 (0.08)	−0.02 (0.08)
GWP							
HSV	−0.08 (0.23)	−0.31 (0.20)	−0.31 (0.20)	−0.25 (0.20)	−0.25 (0.20)	−0.25 (0.20)	−0.29 (0.20)
HSD	0.51 (0.16) **	0.18 (0.15)	0.18 (0.15)	0.20 (0.15)	0.20 (0.15)	0.20 (0.15)	0.19 (0.15)
VAT Area							
HSV	10.17 (3.66) **	6.59 (2.82) *	6.86 (2.82) *	6.03 (2.86) *	6.42 (2.85) *	5.53 (2.88)	6.69 (2.84) *
HSD	−5.38 (2.71) *	−0.42 (2.12)	−0.46 (2.10)	−0.85 (2.11)	−0.66 (2.11)	−1.02 (2.10)	−0.59 (2.11)
SAT Area							
HSV	45.40 (14.73) **	25.60 (9.57) **	25.64 (9.58) **	23.91 (9.74) *	24.08 (9.69) *	22.22 (9.77) *	25.68 (9.65) *
HSD	−5.07 (10.99)	−3.91 (7.21)	−4.06 (7.14)	−4.88 (7.18)	−4.32 (7.17)	−5.51 (7.17)	−4.04 (7.19)
TAT Area							
HSV	55.57 (17.83) **	32.46 (12.07) **	32.50 (12.09) **	29.93 (12.28) *	30.50 (12.22) *	27.75 (12.31) *	32.37 (12.16) **
HSD	−10.45 (13.29)	−4.33 (9.10)	−4.52 (9.00)	−5.73 (9.05)	−4.98 (9.05)	−6.53 (9.03)	−4.63 (9.06)

AGR, android/gynoid fat mass ratio; AWP, android/whole body fat mass proportion; GWP, gynoid/whole body fat mass proportion; HSD, habitual sleep duration; HSV, habitual sleep variability; VAT, visceral adipose tissue; SAT, subcutaneous adipose tissue; TAT, total adipose tissue.

[a] Model 1: Adjusted for age, race, sex, and body mass index percentile. HSV and HSD were entered into separate models.

[b] Model 2: Adjusted for age, race, sex, and body mass index percentile. HSV and HSD were included the same model to adjust for each other.

[c] Model 2a: Model 2 + additional adjustment of total caloric intake.

[d] Model 2b: Model 2 + additional adjustment of total fat intake.

[e] Model 2c: Model 2 + additional adjustment of carbohydrate intake.

[f] Model 2d: Model 2 + additional adjustment of protein intake.

* $p < 0.05$;

** $p < 0.01$.

Table 3

Regression coefficients, 95% confidence intervals, and *p* values in association between 1-hour increase in habitual sleep variability and visceral adipose tissue area from mediation models.

Mediation factor	Effect type	β	95% CI	*p*
Total caloric intake	Direct	6.17	0.41, 12.26	0.02
	Mediation	0.84	0.08, 1.86	0.04
	Total	7.01	1.47, 13.13	0.02
Total fat intake	Direct	6.38	0.98, 11.94	0.04
	Mediation	0.36	−0.18, 1.51	0.28
	Total	6.74	1.06, 12.13	0.04
Carbohydrate intake	Direct	5.45	0.24, 10.99	0.05
	Mediation	1.29	0.23, 3.03	<0.01
	Total	6.74	1.32, 12.42	0.02
Protein intake	Direct	6.44	0.30, 13.18	0.04
	Mediation	0.18	−0.30, 0.94	0.48
	Total	6.62	0.25, 13.45	0.04

All models adjusted for age, race, sex, body mass index percentile, and habitual sleep duration.