Sleep Medicine 16 (2015) 1489–1494

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Original Article

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

Fan He [a], Edward O. Bixler [b], Jiangang Liao [a], Arthur Berg [a], Yuka Imamura Kawasawa [c], Julio Fernandez-Mendoza [b], Alexandros N. Vgontzas [b], Duanping Liao [a,*]

[a] Department of Public Health Sciences, The Pennsylvania State University College of Medicine, Hershey, PA, USA
[b] Sleep Research and Treatment Center, Department of Psychiatry, The Pennsylvania State University College of Medicine, Hershey, PA, USA
[c] Institute for Personalized Medicine, Departments of Biochemistry and Molecular Biology and Pharmacology, The Pennsylvania State University College of Medicine, Hershey, PA, USA

ARTICLE INFO

Article history:
Received 4 April 2015
Received in revised form 20 July 2015
Accepted 21 July 2015
Available online 14 September 2015

Keywords:
Sleep variability
Abdominal obesity
Nutrition intake

ABSTRACT

Objective: To investigate habitual sleep duration (HSD) and habitual sleep variability (HSV) in relation to abdominal obesity and nutrient intake 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 seven 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.

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 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 important, 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 tissues [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 seven 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 serve 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].

* Corresponding author. Department of Public Health Sciences, The Pennsylvania State University College of Medicine, 90 Hope Drive, Suite 2200 A210, Hershey, PA 17033, USA. Tel.: +1 (717) 531 4149; fax: +1 (717) 531 5779.
E-mail address: dliao@psu.edu (D. Liao).

http://dx.doi.org/10.1016/j.sleep.2015.07.028
1389-9457/

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 adolescents, these investigators found that short sleep duration and high sleep variability were related to increased consumption of energy-dense food and sugar-sweetened 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 the nondominant hand during bedtime was used to assess sleep duration for eight 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 were excluded from the calculation, as they were measured under a 9-hour sleep protocol in a laboratory environment. HSD and HSV were computed to assess the participants' habitual sleep patterns. The average of sleep durations across seven nights was used to represent HSD. The intrasubject standard deviation (SD) of the seven-night sleep duration was used to represent HSV. Participants with less than five nights, ie, less than 70% of seven 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 the abdominal region. DXA scan was performed by using Hologic Discovery W scanner (Hologic Inc., Waltham, MA, USA). This method uses two beams of low-energy x-ray that were 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 tissues. 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 the 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

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