Original Article

Short sleep duration is a novel independent risk factor for overweight and obesity

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ABSTRACT

الأهداف: التحقق من ارتباط قلة النوم (SD) وتأثيرها على كتلة الجسم (BMI) لدى الشباب.

المنهجية: أجريت هذه الدراسة خلال الفترة من يناير وسبتمبر 2022م وكان عدد المشاركين 1724 طالب من طالب العلوم الصحية الذين تتراوح أعمارهم بين 16-22 سنة من جامعة الإمام عبد الرحمن بن فيصل، أجري قياس الوزن والطول ومن ثم حساب مؤشر كتلة الجسم باستخدام المعادلة (كتلة الجسم = الوزن بالكيلوجرام/الطول بالمترالمربع). أجرينا تقسيم الطالب المشاركين إلى 3 مجموعات بناءا على مدة النوم SD الخاصة بهم: مدة النوم القصيرة≤ 7 ساعات/24 ساعة، مدة النوم الطبيعية =7-9 ساعات/24ساعة، مدة النوم الزائدة ≥9 ساعات 24/ساعة.

النتائج: متوسط عمر المشاركين (الانحراف المعياري) بين 19±3.2 سنة، مؤشر كتلة الجسم للأشخاص الذين ينامون فترة قصيرة (24.2 كجم/متر مربع) وهو أعلى بكثير مقارنة بالأشخاص الذين ينامون في المعدل الطبيعي (23.03 كجم/متر مربع (أو ينامون لفترات طويلة) 23.2 كجم/متر مربع p=0.041. بالإضافة إلى ذلك كانت قلة النوم (SD) مرتبطة بشكل كبير بزيادة الوزن (OW) والسمنة (OB) (p=0.01)، وأيضاً وجد تحليل الانحدار اللوجستي متعدد التغيرات أن قلة النوم SD هي عامل خطر مستقل لزيادة الوزن والسمنة حيث احتفظت بأهميتها (p=0.049) حتى بعد ضبط العوامل الأخرى المؤثرة على كتلة الجسم مثل قلة النشاط البدني وزيادة تناول الطعام، تبين أن من زيادة الوزن والسمنة.

الخلاصة: قلة النوم SD هي عامل خطر مستقل لزيادة مؤشر كتلة الجسم، مما قد يؤدي إلى زيادة بمقدار 1.47 مره أعلى في معدل زيادة الوزن والسمنة.

Objectives: To investigate the association between sleep duration (SD) and body mass index (BMI) in young adults.

Methods: This cross-sectional study was carried out between January and September 2022, and the participants were 1724 health sciences students aged 16-22 years, from Imam Abdulrahman Bin Faisal University, Dammam, Saudi Arabia. Measurement of height and weight was carried out and BMI was calculated by the formula=weight in kg/height in m^2. Based on SD, subjects were divided into 3 groups: short SD: <7 hours/24 hours; normal SD: 7-9 hours/24 hours; and increased SD: >9 hours/24 hours.

Results: The participants' mean age was 19±3.2 years. Short sleepers had significantly higher BMI (24.2 kg/m^2, p<0.001) relative to normal (23.03 kg/m^2) and long sleepers (23.2 kg/m^2, p=0.041). In addition, short sleep duration (SSD) was associated with overweight (OW) and obesity (OB) (p=0.01). Moreover, multivariate logistic regression analysis found SSD to be an independent risk factor for OW and OB, as it retained its significance (p=0.049) even after adjusting for the major confounding factors of BMI (namely, decreased physical activity and increased food intake). Moreover, it emerged that SSD could lead to 1.47 times higher odds ratio of OW and OB.

Conclusion: Participants with a SSD had significantly higher BMI than normal and long sleepers. Short sleep duration is an independent risk factor for increased BMI, which potentially leads to 1.47 times increased odds ratio of having OW and OB.

Keywords: short sleep duration, overweight, obesity, body mass index

Saudi Med J 2023; Vol. 44 (11): 1160-1166
doi: 10.15537/smj.2023.44.11.20230111

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Received 14th February 2023. Accepted 20th September 2023.

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Disclosure. Author has no conflict of interests, and the work was not supported or funded by any drug company.

Overweight (OW) and obesity (OB) are defined as excessive or abnormal fat accumulation that represents a risk to health. Over the last 40 years, OW and OB has raised 4-fold, rising from 4-18% globally.[1] Overweight and OB is also high amongst the Saudi population. According to the 2019 Saudi Arabia World Health Survey, the general frequency for OW is 38% and for OB is 20% in Saudi Arabia.[2] Overweight and OB are well-known risk factors for cardiovascular, metabolic, musculoskeletal diseases, and various other adverse health outcomes.[3]

Excessive food intake and reduced physical activity (PA) are the major documented behavioral risk factors for OW and OB.[4] But recently, sleep duration (SD) has also been identified as a health behavior that can affect an individual's body mass index (BMI).[5-8] Because, parallel to an increase in BMI (from 4-18%), a significant reduction in SD has also been documented over the past 50 years. Various studies have reported a global average decrease in SD from 1.5-2 hours/24 hours, and the proportion of young adults sleeping less than 7 hours/24 hours has almost doubled (from 15.6-37.1%).[9,10]

Normal sleep is important for regulating the health and functions of the human body. But evidence has grown in the past decade that in addition to its effects on the optimal performance of numerous physiological mechanisms, adequate sleep may be one of the important factors in maintaining normal body weight.[5-9,11]

Many studies support the view that SD affects BMI, although the nature of this relationship remains uncertain. The majority of the authors associate short sleep duration (SSD) to an increased obesity risk, in contrary to the others who document that SSD can lead to a decreased body weight.[7-9,12] In addition, some authors have reported an inverse linear association between SD and BMI, indicating that increasing sleep duration lowers BMI linearly.[6,13-15] Meanwhile others have documented a U-shaped association implying that both short and long SD can lead to increased BMI.[16,17] However, other studies have failed to find any association between SD and BMI.[18-20]

As the existing studies are inconclusive, in terms of demonstrating the clear relationship between SD and BMI, so this study was designed to close the gap. Moreover, while analyzing an association between SD and BMI, most of the existing studies have neglected the effects of major confounders on BMI (including sedentary behavior and increased food intake).[11-16]

Therefore the objectives of this study were: I) to determine the association between SD and BMI; II) to investigate the association between SD and BMI after adjusting for the major confounders of BMI (namely, sedentary behavior and increased food intake); and III) to analyze whether decreased SD is an independent risk factor for increased BMI.

Methods. This cross-sectional study was carried out between January and December 2022 on 1724 students of Imam Abdulrahman Bin Faisal University in Dammam, Saudi Arabia, aged 16-22 years. Ethical approval was obtained by the Deanship of Scientific Research of Imam Abdulrahman Bin Faisal University in Dammam, Saudi Arabia (IRB no.: 2022-01-011).

Calculation of the sample size was carried out by epidemiologic statistics for public health tools software. Estimated sample size was 787 participants. Sample size was calculated by taking 9% of p (risk of OW and OB in short sleep duration), 95% of confidence level (1-α), and 2% of margin of error.[7]

Data collection was carried out by convenience sampling technique. Multiple 5 minutes briefing sessions were carried out in various classrooms to describe the study rationale. A total of 1893/4000 subjects showed willingness to be a part of this study, out of them 1724 were included in this study, whereas 169 students were excluded because of: I) a diagnosed sleep disorder (including sleep apnea, insomnia, narcolepsy, and excessive sleepiness); II) any chronic illness (including diabetes mellitus, hypothyroidism, depression, and anxiety disorder); and III) using sleeping pills, anti-anxiety pills, or any other medication for more than 6 months. The response rate was 43.1%.

The selected subjects signed the informed consent, filled out the online questionnaire, and their anthropometric measurements were carried out under the direct supervision of the author, in Physiology laboratory. The study was in accordance with the principles of Helsinki Declaration.

After measuring the weight and height, BMI was measured by the formula=weight in kg/height in m^2. On the basis of BMI calculation, the subjects were categorized into 3 main groups: underweight (UW): BMI of ≤18.5; normal weight (NW): BMI of ≤24.9; and OW and OB: BMI of >25 in m^2.[21]

The online questionnaire inquired on SD, eating habits, and PA in the last one year. Sleep duration was calculated from the short form of the perceived quality index questionnaire by asking the following questions regarding the sleep habits of the subjects: I) what is your usual time to go to bed?; II) how long does it take you to fall asleep (mention in minutes)?; and III) what is your usual rise up time in the morning?[22]

The calculation of time in the bed (TIB) was carried out by subtracting "time to go to bed" from "rising up time in morning".

Sleep onset latency (SOL) was calculated by number of minutes obtained to fall asleep after going to bed. Sleep duration was calculated as: TIB-SOL.[23]

Based on the latest guidelines of the sleep foundation,[3] students were categorized into 3 main groups based on the number of sleeping hours: SSD: <7 hours/24 hours; normal SD: 7-9 hours/24 hours; and increased SD: >9 hours/24 hours.[24]

Physical activity was identified by a 7-item international physical activity questionnaire (IPAQ). Students were categorized into 3 groups of low, moderate, and high PA, based on their IPAQ score. Students with low PA were grouped as inactive, whereas the active group included the students with medium and high activiy.[25]

Regarding the food intake, students were grouped into the following 3 categories: less food intake: one meal/day; normal food intake: 2 or 3 meals/day; and high food intake: >3 meals/day.

Frequency of junk food intake was evaluated as follows: "do you eat the following foods frequently (deep fried food, packed and processed food, sweet fast food, energy drinks, carbonated drink, sweet beverages and sugary juices)?" If the subjects were taking junk food ≥5 days/week, they were categorized as "frequent junk eaters".[26]

Statistical analysis. The data was analyzed by the Statistical Package for the Social Sciences, version 20 (IBM Corp., Armonk, NY, USA). Demographic data was analyzed by using descriptive statistics.

Comparison of mean BMI between various sleep groups was carried out by one-way analysis of variance (ANOVA). Whereas, pairwise comparison of BMI between various sleep categories was carried out by the least significant difference (LSD) test.

The comparison of the distribution of "high food in takers", "frequent junk eaters", UW, NW, and OW and OB subjects into various sleep categories was carried out by simple Chi-square test.

Multivariate logistic regression analysis was carried out to find the odds ratios (ORs) and independent association of OW and OB with various lifestyle behaviors. A *p*-value of <0.05 was considered significant.

Results. The mean age of the students was 19±3.2 years. The average BMI of the study population was 23.6±5.7 kg/m^2. Table 1 shows the basic demographic characteristics of the study participants.

A significant difference of BMI between various sleep categories was indicated by one way ANOVA (p<0.001). So, a pairwise comparison between various sleep categories was carried out by LSD test which highlighted a significant difference between the BMI of short versus normal sleepers (p<0.001). Thereby, indicating that short sleepers have significantly higher BMI than normal and long sleepers (Table 2).

Although long sleepers also had a higher mean BMI (23.2 kg/m^2) than normal sleepers, but this difference was not significant (p=0.770; Table 2). Therefore, we conclude that a U-shaped association does not exist between BMI and SD.

Table 3 shows the comparison of the distribution of UW, NW, and OW and OB subjects into various sleep categories. Data indicated that the percentage of OW and OB subjects falling into the category of SSD group (34%) was significantly higher (p=0.010) than the percentage of OW and OB subjects in the normal (26.90%) and long sleeper (29.30%) groups. Thereby, indicating that SSD may be one of the factors accountable for OW and OB in young adults.

We also targeted to find an association between food

Table 1 - Demographic data of 1724 study participants.

Demographic categories	n (%)
Gender	
Females	956 (55.4)
Males	768 (44.5)
Age (years)	
16 - >19	982 (56.9)
19-22	742 (43.0)
College	
Foundation year	212 (12.2)
College of medicine	754 (43.7)
College of nursing	456 (26.4)
College of clinical pharmacy	213 (12.3)
College of dentistry	89 (5.16)
Body mass index	
Underweight	256 (14.8)
Normal weight	968 (56.1)
Overweight + obese	500 (29.0)
Sleep duration	
Short sleepers	393 (22.8)
Normal sleepers	959 (55.6)
Long sleepers	372 (21.6)
Physical activity	
Inactive	539 (31.0)
Active	1185 (68.7)
Number of meals (day)	
Consuming ≥3 meals/day	553 (32.0)
Consuming ≤3 meals/day	1171 (67.9)
Intake of junk food	
Consuming junk food ≥5 days/week	883 (51.2)
Consuming junk food <5 days/week	841 (48.7)

Values are presented as numbers and precentages (%).

Table 2 - Comparing mean body mass index between various groups (n=1724) of sleep duration (one-way analysis of variance).

Sleep duration	n (%)	Mean BMI	*P*-values
Shorts sleepers (group1)	393 (22.7)	24.2±6.5	
Normal sleepers (group2)	959 (55.6)	23.03±5.5	0.001*
Long sleepers (group3)	372 (21.6)	23.2±5.7	
Pairwise comparison of BMI between various sleep groups by LSD test			
Short sleepers versus normal sleepers (G1 vs G2)			<0.001*
Short sleepers versus long sleepers (G1 vs G3)			0.041
Long sleepers versus normal sleepers (G3 vs G1)			0.770

Values are presented as numbers and precentages (%). *Statistically significant. BMI: body mass index, LSD: least significant difference

Table 3 - The association of various risk factors with sleep duration (Chi-square test).

Factors	Short sleepers (n=393)	Normal sleepers (n=959)	Long sleepers (n=372)	*P*-values
UW	46 (11.7)	152 (15.8)	58 (15.6)	0.0527
NW	213 (54.2)	549 (57.2)	206 (55.4)	0.312
OW+OB	134 (34.1)	258 (26.9)	108 (29.0)	0.010*
Frequent intake of junk food	225 (57.4)	455 (48.6)	203 (54.4)	<0.003*
High food intake	161 (41.0)	265 (27.6)	127 (34.1)	<0.001*
Decreased physical activity	154 (39.1)	253 (26.3)	132 (35.4)	<0.001*

Values are presented as numbers and precentages (%). *Statistically significant. UW: underweight, NW: normal weight, OW: overweight, OB: obesity

intake and SD, and it was analyzed that most of the "frequent junk eaters" (57.40%) were short sleepers. Moreover, a significantly higher percentage of the subjects (40.97%) with a "higher food intake" were also found to be short sleepers. In addition, a signification positive association was also found between decreased PA and SSD (Table 3).

Univariate analysis was carried out to find the association of increased BMI with various risk factors, and increased BMI was found to be positively associated with SSD, "high food intake", and "decreased PA" (Table 4). We also aimed to analyze whether or not, SSD is an independent risk factor for increased BMI. Therefore, multivariate logistic regression analysis was applied which revealed that controlling for food intake and PA did not mitigate the relationship between increased BMI and SSD. Therefore, it can be revealed that SSD is an independent risk factor for OW and OB, as it retained its significance (p=0.049), even after adjusting for the major confounding factors of BMI (namely, decreased PA and high food intake). Moreover, the study results showed that SSD can lead to 1.47 times increased OR of having OW and OB (Table 4).

Discussion. This study highlighted that short sleepers have significantly higher BMI than normal and long sleepers, thereby, indicating SSD may be one of the causes of OW and OB in the young adults. Some other studies have also narrated identical reports.[7-9] A study by Lytle et al[7] recruiting 723 subjects, indicated that a chronic pattern of SSD of ≤6/24 hours was associated with a higher BMI. Peltzer et al[8] also highlighted a strong relationship between SSD and OB in a population of 1821 young adults. A recent systemic review and meta-analysis by Zhou Q et al[27] concluded that the risk of OB significantly increased with SSD. It was observed that compared to 7/24 hours of SD, the risk of OW and OB raised to 9% for every one hour decrease in the SD.[27]

Various mechanisms can be referenced to explain the link between SSD and weight gain. Calvin et al[28] proposed that SSD provides extended hours for food consumption, thereby, increasing caloric intake leading to weight gain. Our study findings also agree with this fact, as our short sleeper group reported consuming a higher number of meals/days relative to the normal sleepers. Furthermore, some studies using the objective measures of energy intake indicated that the subjects sleeping ≤6/24 hours consumed 178 kcal/day more than those sleeping ≥9/24 hours.[29] McNeil et al[30] imposed sleep deprivation in a group of subjects, observing an increase in the caloric intake of 200-500 kcal/day.

In addition, SSD is also reported to be associated with a greater tendency to select/consume saturated fats and high-calorie foods resulting in a higher total energy

Table 4 - The association of overweight and obesity with various lifestyle behaviors.

Lifestyle behaviors	Univariate analysis (unadjusted ORs)		Multivariate analysis (adjusted ORs)	
	Overweight/obese	*P*-values	Overweight/obese	*P*-values
Physical activity				
Inactive (low physical activity based on IPAQ scores)	1.17 (1.06-2.1)	0.037*	1.6 (1.01-2.5)	0.046*
High food intake				
Consuming >3 meals/day	1.6 (1.3-2)	<0.001*	1.5 (1.014-2.1)	0.042*
Frequent intake of junk food				
Consuming junk food ≥5 days/week	1.001 (0.8-1.3)	0.9	1.1 (0.6-1.3)	0.57
Short sleep duration				
<7 hours/24 hours	1.4 (1.07-1.7)	0.011*	1.47 (1.01-2.2)	0.049*

Values are presented as odd ratios (OR) and 95% confidence interval (CI). *Statistically significant.
IPAQ: the International Physical Activity Questionnaire

intake. St-Onge et al[31] observed that sleep timings and SSD appear to affect food choices. A review by Dashti et al[32] summarized that SSD is linked to poor eating habits, including: irregular eating behaviors, low intake of fruits, vegetables, dietary fiber, and whole grains, but increased consumption of high-energy and low-quality foods (namely, fast foods, energy-dense highly palatable snacks, carbonated drinks, and sugars). In agreement with these researches, our study results also demonstrated that, high intake of junk food in short sleepers can lead to an overall increased energy intake and higher risk of increased BMI.

The causal pathway of overeating in short sleepers can be linked to disturbed neurohormonal and metabolic profiles. Ghrelin and leptin are the 2 main appetite-regulating hormones. Leptin is an anorexigenic hormone responsible for appetite suppression, whereas, Ghrelin is an orexigenic hormone causing appetite stimulation.[33,34] Various studies have demonstrated that SSD causes a significant disruption in the normal leptin and ghrelin levels. A review by Lin J et al[35] summarized that SSD is associated with increased ghrelin and decreased leptin levels, resulting in increased food intake leading to OW and OB.

Moreover, SSD may lead to daytime fatigue and therefore reduced PA, which can contribute to weight gain. These observations are in line with our study results where the short sleepers subjects were found to be physically inactive as compared to the normal sleepers. A review article by Kline et al[36] concludes that, in general, the subjects with poor sleep and SSD are less active than similar subjects without sleep complaints.

In addition to the above-mentioned facts, a chief finding from our study was that SSD retained its significance as an independent risk factor for OW and OB even after adjusting for food intake and PA. This independent association can be due to the fact that SSD can interfere with the normal circadian rhythm and functions of the hypothalamic-pituitary-adrenal axis; thereby, directly influencing various metabolismand endocrine secretion patterns.[37] This can lead to disrupted adiponectin, cortisol, insulin, interleukin-6, and growth hormone levels, all of which are linked to an increased risk for OW and OB. Moreover, the human body may consider the lack of sleep as a biological stressor causing energy conservation as a compensatory response.[38] Spaeth et al[39] observed that short sleepers had lower morning resting metabolic rate, which may indicate that SSD generates metabolic changes focused on energy conservation causing weight gain.

Although SSD disrupts the levels of various metabolic and stress hormones including ghrelin, leptin, adiponectin, cortisol, insulin, and growth hormone; but it is still debatable that whether these hormone directly affect metabolism or influence the food selection. Therefore, further future research is recommended in this regard.

Contrary to our findings, certain authors reported that SSD can reduce body weight, while others have documented no association between SD and BMI.[12,18-20] These inconsistencies can arise due to differences in study designs, sample size, population, and interventions.

Study strengths & limitations. The strengths of our study includes: significant study findings; a large sample size; measurement of anthropometric variables in the laboratory; adequate data on the confounding variables; use of standardized questionnaires; and recruiting health science students as subjects, thereby enhancing the precision response rate. There were several potential limitations of our study. First, the information regarding sleep duration was subjective and self-reported. Although there is a high correlation

between subjective and objective estimates of sleep duration,[40] still we recommend future replication of our research by using objective sleep measures including polysomnography and actigraphy. Secondly, it was a cross-sectional study so the analysis of the the causal effect of SSD on BMI was not possible. Therefore, further retrospective, prospective, and follow-up studies are advised in this regard. Moreover, this study has not analyzed the metabolic or stress hormones, and future research is needed in this regard. Finally, this study was carried out on the Saudi population, therefore our results might not be generalized to other races and ethnic groups.

We observed a strong association between SSD and increased BMI, therefore we indicate that SSD may be a major contributing factor behind the increased prevalence of OW and OB in the young Saudi population. Therefore, it is recommended to enhance awareness regarding the importance of sleep in young adults, because improved SD may improve the balance of appetite-regulating hormones, which can help to decrease the burden of OW and OB in young adults.

In conclusion, our study results have shown a positive association between SSD and increased BMI. Thereby, indicating that SSD may be one of the causes for OW and OB in our young adults. Short sleep duration was also found to be an independent risk factor for OW and OB as it retained its significance even after adjusting for the major confounding factors of BMI (namely, decreased PA and increased food intake). Moreover, it was analyzed that SSD can lead to 1.47 times increased OR of having OW and OB.

Acknowledgment. *The author gratefully acknowledge Dr. Lubna AlAsoom, Maha AlHamdan, Laila H. Al-Yousef, and Noha A. Alfayadh for laboratory and technical assistance. The author also would like to thank Academic proof reader Ltd. (www.academicproofreader.com) for the English language editing.*

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