Short sleep time increases lipid intake in obese adolescents

Menor tempo de sono aumenta a ingestão de lipídios em adolescentes obesos

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ABSTRACT

Objective: The aims of the present study were to verify whether sleep pattern could influence the food intake profile as well as to examine the impact of body composition on sleep pattern in obese adolescents. Methods: This is a cross-sectional study comprised of 55 post-puberty adolescents (15 to 19 years of age) with body mass indices greater than the 95th percentile. The anthropometric variables analysed were waist circumference, body weight, height, body mass index and body composition. Nutritional data were obtained throughout a 3-day dietary record, and sleep parameters were recorded using a 7-day sleep diary. Statistical analyses were performed using multiple linear regressions with significance set at \( p < 0.05 \), and the effect size \((r)\) was calculated for both models. Results: The models models of multiple linear regression analyses adjusted by gender revealed that body fat mass (kg) was an independent predictor of greater obesity severity, indicating the importance of sleep for body weight homeostasis. Furthermore, the data suggest that the total fat mass might be associated with higher sleep latency, contributing with a reduction in sleep time on lipid intake. It was observed that a reduction in sleep time might contribute to the development and maintenance of obesity through an increase in fat intake. Furthermore, the data suggest that the total fat mass might be associated with higher sleep latency, contributing with a reduction in sleep duration, confirmed by an expressive effect size. Conclusion: The results indicate that an association between reduced sleep duration and irregular eating habits can promote a vicious cycle difficult obesity control in adolescents.

Keywords: adolescent, body fat distribution, lipids, obesity, sleep, sleep disorders.

RESUMO

Objetivos: Os objetivos do presente estudo foram verificar se o padrão de sono poderia influenciar no perfil de ingestão alimentar, assim como examinar o impacto da composição corporal no padrão de sono de adolescentes obesos. Métodos: Estudo transversal composto por 55 adolescentes pós-púberes (com idade entre 15 e 19 anos), com Índice de massa Corporal (IMC) maior que o percentil 95. As variáveis antropométricas analisadas foram a circunferência da cintura, peso corporal, altura, IMC e composição corporal. Os dados nutricionais foram obtidos através do registro alimentar de 3 dias; e o padrão de sono foi obtido usando um diário do sono de 7 dias. Análises estatísticas foram feitas usando regressões lineares múltiplas, adotando \( p < 0.05 \) como significância. O tamanho do efeito foi calculado para ambos os modelos. Resultados: Ambos os modelos de regressão ajustados por gênero mostraram que a gordura corporal (kg) foi um fator independente para a latência do sono; e o tempo total de sono apresentou-se como fator independente para ingestão de lipídios. Foi observado que a redução no tempo total de sono pode contribuir para o desenvolvimento e manutenção da obesidade, através de um possível aumento da ingestão lipídica. Além disso, os dados sugerem que a gordura corporal total pode estar associada com uma maior latência pro sono, contribuindo com a redução do tempo total de sono, confirmado pelo expressivo tamanho do efeito. Conclusão: Os resultados indicam uma associação entre menor tempo de sono e hábitos alimentares irregulares, levando a um ciclo vicioso, dificultando o controle da obesidade em adolescentes.

Descritores: adolescente, distribuição da gordura corporal, lipídios, obesidade, sono, transtornos do sono.

INTRODUCTION

Sleep curtailment is a very common occurrence in the current today, especially among adolescents who are involved in many activities such as school-related duties and recreational activities, such as sports, social networking, and language course. It is well documented that adolescents require more sleep than adults and elderly people, and a minimum of 9 hours of sleep has been recommended. Recently, studies have shown that sleep loss is associated with many health issues such as obesity and its co-morbidities. For instance, Gupta et al. showed that each additional hour of sleep decreased the odds of obesity by 80%. In agreement with these findings, another study highlighted that obese individuals with less sleep per night presented greater obesity severity, indicating the importance of sleep for body weight homeostasis.

Concomitantly, obesity is a multifactorial disease that affects millions of people worldwide, and one of the main
causes of obesity is the imbalance between energy intake and expenditure. The incidence of obesity during adolescence increases the chances of obesity in adulthood, which can reduce the individual’s quality and duration of life and cause sleep disorders.

Epidemiological data have shown a significant association between weight gain and short sleep time. Furthermore, an association between reduced sleep duration and irregular eating habits, snacking between meals, excessive food seasoning and low consumption of vegetables has been demonstrated. In a recent study of South Korean adolescents, sleep time was inversely associated with body mass index (BMI) levels, and reduced sleep was strongly correlated with a greater risk of being overweight and becoming obese. In fact, some studies have suggested that altered pattern of the sleep-wake cycle and feeding behaviour were associated with changes in body weight, although the mechanisms by which short sleep contributes to increased adiposity remain unclear. Moreover, emerging evidence suggests that sleep-deprived humans have preferences for energy-dense foods, although only a single study has tested this hypothesis on teenagers and found a positive association between less hours of sleep and increased carbohydrate intake.

Because sleep impairment can lead to nutritional imbalance and obese adolescents already have detrimental effects on the energy balance, it is important to investigate whether the total sleep time results in undesirable changes in the food intake profile that underlies the connection between insufficient sleep and obesity.

A single study showed that adult obese individuals have longer sleep latency when compared with non-obese however, it was not evaluated the possible association with adiposity measures. In addition, it remains unknown the relationship of sleep latency and body composition in obese adolescents. Thus, the purpose of this current study was to verify whether sleep time could exert a significant influence on the food intake profile as well as determine the impact that body composition has on latency of sleep among obese adolescents.

MATERIALS AND METHODS

Subjects
A total of 55 obese adolescents (15 to 19 years of age) presenting simple obesity based on a body mass index (BMI) > 95th percentile (Centers for Disease Control and Prevention) entered the Interdisciplinary Obesity Program of the Universidade Federal de São Paulo (UNIFESP) between January 2009 and 2010. The inclusion criteria for the post-pubescent stage were based on the Tanner scale stage 5 for both boys and girls. Non-inclusion criteria were as follows: other metabolic or endocrine diseases such as hypothyroidism and Cushing Syndrome; chronic alcohol consumption; previous use of drugs, such as anabolic-androgenic steroids, psychotropics, anorectics, or hypoglycaemic, which may affect appetite regulation; and pregnancy.

The study was conducted in accordance with the principles of the declaration of Helsinki and was approved by the Ethical Committee of the Universidade Federal de São Paulo (# 0135/04) and registered in ClinicalTrials.gov (NCT01358773). Informed consent was obtained from all subjects and/or their parents.

Anthropometric variables and body composition
Subjects were weighed on a scale to the nearest 0.1 kg while wearing light clothing but not shoes. Height was measured to the nearest 0.5 cm with a wall-mounted stadiometer (Sanny, model ES 2030), and waist circumference was measured with a non-stretchable tape measure and recorded to the nearest 0.1 cm. The BMI was calculated as body weight divided by height squared. Body composition was measured by plethysmography in a BOD POD body composition system (version 1.69; Life Measurement Instruments, Concord, CA, USA).

Food variables
Energy intake was calculated based on a 3-day dietary record. Because most obese people under-report their food consumption, each adolescent was asked to record their diet with help from their parents. Trained nutritionist instructed the subjects to record in as detailed a manner as possible every item that they either drank or ate, the time they ingested it, the amount consumed, and how the food was prepared. The degree of under-reporting may still be substantial; however, this is a validated method for the assessment of dietary consumption. Portions were measured in terms of familiar volumes and sizes. The dietician taught the parents and adolescents how to record food consumption. The same dietician transferred these dietary data to a computer, and the nutrient composition was analysed by a PC program developed at the Universidade Federal de São Paulo (Nutwin software, for Windows, version 1.5) that used data from western and local food tables. Parents were also encouraged by a dietician to call if they needed extra information. The distributions of the nutrients were analysed using the values recommended by Dietary References Intakes (DRI).

Sleep parameters
Sleep can be evaluated through many ways, and whereas the polysomnography is the gold standard instrument, it requires a high investment. Also, considering that polysomnographic measurements are cumbersome and expensive, it was necessary to select a suitable method for the sleep assessment. The sleep diary is a validated subjective methodology in which the patient takes notes of specific sleep variables during a week that allows the calculation of important information such as total sleep time, sleep latency, sleep efficiency, total time in bed, awakenings, and the subject’s own perception of sleep. In spite of its limitation, the sleep diary tends to be correlated with objective measure of sleep. Therefore, a 7-day sleep diary was used to record the following variables: nocturnal sleep time, night awakenings, sleep efficiency, total time in bed, sleep latency and how the volunteer perceived their sleep. Sleep efficiency was determined by the percentage of time spent asleep over time from sleep onset (sleep latency) to last awakening, thus an increased latency time can impair total sleep time.
Statistical analysis
The Gaussian distribution of variables was verified with a Shapiro-Wilk's W test, and variables with normal distribution were expressed as mean ± standard deviation (SD) while non-parametric variables were expressed as median (minimum and maximum) in a descriptive table. The z-score of the non-parametric variables was also obtained. A correlation study was performed, but just as an exploratory method. Comparisons between girls and boys were made using the independent t-test. Two models of multiple regression analyses were performed. In the first model the sleep latency was set as dependent model while gender and fat mass (kg) were the independent variables to verify how they could affect it. In the second model the total sleep time was chosen as a dependent variable and gender as well lipid intake (%) were the independent variables, to verify how total sleep time could influence food patterns. The magnitude of effect size was calculated in both models to evaluate the reliability of the analyses (N2 / N1 + df), considering as an expressive result values above 0.3. The results with p values < 0.05 were considered statistically significant. Statistical analyses were performed using SPSS (version 18 for Windows).

RESULTS
The sample population totalled 55 volunteers of both genders (35 girls and 20 boys) with a mean age of 17 years. The descriptions of the analysed variables are shown in Table 1. All anthropometric variables portray pathogenic obesity, including a high percentile of fat mass, waist circumference higher than recommended by the World Health Organization (WHO) and reduction in the proportion of lean mass\(^{(10)}\). Based on the average BMI (37.26 kg/m\(^2\)), most of the teenagers presented class II obesity. Significant differences were found in the body compositions based on gender, whereas boys presented greater lean body mass (kg) and less percentage of fat mass. Boys also presented higher values of waist circumference.

Table 1. Anthropometric characteristics of obese adolescents.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Entire group (n = 55)</th>
<th>Girls (n = 35)</th>
<th>Boys (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass (Kg)</td>
<td>107.25 ± 17.02</td>
<td>104.40 ± 13.98</td>
<td>112.22 ± 20.80</td>
</tr>
<tr>
<td>BMI (Kg/m2)</td>
<td>37.26 ± 4.4</td>
<td>37.49 ± 4.39</td>
<td>36.81 ± 4.5</td>
</tr>
<tr>
<td>Fat mass (%)</td>
<td>46.25 ± 5.0</td>
<td>47.44 ± 4.71</td>
<td>44.17 ± 5.11</td>
</tr>
<tr>
<td>Lean mass (%)</td>
<td>53.4 ± 8.2</td>
<td>53.21 ± 7.97</td>
<td>53.92 ± 9.0</td>
</tr>
<tr>
<td>Fat mass (Kg)</td>
<td>50.4 ± 8.7</td>
<td>49.73 ± 7.3</td>
<td>51.85 ± 11.0</td>
</tr>
<tr>
<td>Lean mass (Kg)</td>
<td>57.48 ± 9.0</td>
<td>54.66 ± 7.30</td>
<td>62.28 ± 10.65</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>101.8 ± 10.5</td>
<td>99.4 ± 9.41</td>
<td>107.12 ± 10.56</td>
</tr>
</tbody>
</table>

BMI: body mass index; * t test significant difference.

Regarding the food parameters, the mean distribution of the macronutrients was within the Dietary Reference Intake (DRI) recommendations: carbohydrates (52.8%), protein (18.9%) and lipids (28.7%). Despite the mean of lipid intake being within the recommendations, when we fractionated the lipid profile by the percentage of Energy Intake (EI), the mean values of saturated fat (9.73%), monounsaturated fat (7.3%) or polyunsaturated fat (3.5%) did not reach the recommended values\(^{(27)}\). When it was analysed the food intake according to gender, it was observed that boys showed a significantly higher energy intake.

The data obtained from the sleep diary showed that most of the adolescents (63.3%) slept less than 8 hours/night, and 87.27% of them reported sleep latency lower than 30 minutes. The sleep efficiency was an important variance among the teenagers; however, the differences in the sleep variables between boys and girls were not statistical significant. Sleep variables and dietary patterns are disclosed in Table 2.

Table 2. Sleep variables and dietary patterns of obese adolescents.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Entire group (n = 55)</th>
<th>Girls (n = 35)</th>
<th>Boys (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total energy intake</td>
<td>1824.61 ± 590.3</td>
<td>1693 ± 466.88</td>
<td>2048.25 ± 719.64</td>
</tr>
<tr>
<td>Lipid intake (%)</td>
<td>28.7 ± 5.3</td>
<td>28.62 ± 5.55</td>
<td>28.97 ± 4.97</td>
</tr>
<tr>
<td>Saturated fat (%)</td>
<td>9.7 ± 2.7</td>
<td>9.77 ± 2.95</td>
<td>9.66 ± 2.55</td>
</tr>
<tr>
<td>Monounsaturated fat (%)</td>
<td>7.3 ± 2.6</td>
<td>7.28 ± 2.95</td>
<td>7.33 ± 2.92</td>
</tr>
<tr>
<td>Polysaturated fat (%)</td>
<td>3.5 ± 1.4</td>
<td>3.5 ± 1.26</td>
<td>3.65 ± 1.7</td>
</tr>
<tr>
<td>Carbohydrates intake (%)</td>
<td>52.80 ± 7.4</td>
<td>54.17 ± 6.66</td>
<td>50.47 ± 8.3</td>
</tr>
<tr>
<td>Protein Intake (%)</td>
<td>18.9 ± 4.8</td>
<td>17.78 ± 3.51</td>
<td>20.99 ± 6.08</td>
</tr>
<tr>
<td>Total time in bed (h)</td>
<td>8 ± 1.3</td>
<td>7.95 ± 1.26</td>
<td>8.10 ± 1.4</td>
</tr>
<tr>
<td>Total sleep time (h)</td>
<td>7.5 ± 1.2</td>
<td>7.44 ± 1.20</td>
<td>7.6 ± 1.41</td>
</tr>
<tr>
<td>Sleep latency (minutes)*</td>
<td>12.14 (3.4 - 46.4)</td>
<td>12.57 (3.4 - 37.1)</td>
<td>11.42 (4.85 - 46.42)</td>
</tr>
<tr>
<td>Sleep efficiency (%)*</td>
<td>94.2 (41.0 - 97.7)</td>
<td>93.82 (41 - 97.7)</td>
<td>94.57 (86.65 - 97.34)</td>
</tr>
<tr>
<td>Well-being on awakening (%)</td>
<td>72.04 ± 16.23</td>
<td>69.41 ± 15.57</td>
<td>76.8 ± 16.72</td>
</tr>
<tr>
<td>Sleep satisfaction (%)</td>
<td>75.28 ± 16.23</td>
<td>73.5 ± 17.48</td>
<td>78.57 ± 17.25</td>
</tr>
</tbody>
</table>

* non-parametric data presented as median and minimum and maximum values; † t test significant difference.

Two multiple linear regression analyses were performed and in the first model adjusted by gender with sleep latency as a dependent variable revealed that body fat mass (kg) was an independent predictor (\(β = 0.48; p = 0.003\)) (r = 0.95) (Table 3). In the second model, having the lipid intake (%) as a dependent variable, demonstrated that total sleep time was a predictor factor (\(β = -1.32; p = 0.02\)) (r = 0.91) (Table 4).

A negative association was discovered between total sleep time and lipid intake (%), indicating that a reduced sleep time increases lipid consumption. Additionally, a higher fat mass was associated with longer time required for the volunteers to fall asleep. The main findings of this study are illustrated and hypothesized in Figure 1.
agreement with a previous study, which also showed influence of less hours of sleep on food behaviour, in which teenager girls with short sleep duration presented higher carbohydrate consumption\(^{(21)}\). However, the authors did not find any association between sleep patterns and body fat. Alteration in both lipid and carbohydrate intake can change the lipid pathway and induce a positive energy balance leading to obesity and its co-morbidities\(^{(34,35)}\).

Increased lipid consumption and less hours of sleep has been linked in the adult population, especially among shift workers\(^{(34,36)}\). In fact, a study performed with Greek women showed an association between sleep duration and saturated fat\(^{(19)}\). High levels of fat intake are related to the development of non-alcoholic fatty liver disease, atherosclerosis and dyslipidemia, in addition to the increase of body weight through a positive energetic balance\(^{(38-41)}\). Collectively, these results reinforce the importance of early nutritional strategies to prevent and control metabolic diseases, especially in adolescents.

Additionally, short sleep time may affect orexigenic and anorexigenic hormones, thus altering satiety and appetite. Previously, Spiegel et al.\(^{(19)}\) showed that sleep deprivation reduced by 18% and increased by 28% the concentrations of leptin and ghrelin, respectively. In the same study, it was demonstrated that sleep debt was associated with an increase in hunger ratings by 24% on the 10-cm visual analogue scale and appetite ratings by 23%. Thus, all these mentioned factors might explain the alterations on food behaviour among those who sleep less.

In the present study, a vicious cycle can be hypothesized (Figure 1), since less hours of sleep could influence fat consumption, which could lead to a weight gain and apparently reducing sleep time through higher sleep latency. This hypothesis could be reinforced by an expressive effect size obtained in both multiple linear regression models (Tables 3 and 4). In agreement, recent evidence suggests that adipose tissue has an important role in endocrine system regulation, energy homeostasis, satiety signalling, and the biological clock, suggesting the link between sleep and obesity as causes and consequences\(^{(32)}\).

A possible explanation for increased sleep latency in those with higher fat mass could be the effects of excess weight, especially on the respiratory system. Considering that severe obesity is associated with an anatomically narrowed pharynx, which would constitute a resistive load that might contribute to sleep disruption\(^{(47)}\). Although, we did not find an association between abdominal obesity and sleep changes, some studies with adults have shown that this kind of obesity was related to a reduction of sleep time and sleep efficiency\(^{(7,46)}\). In addition, a single report showed that obese adults without obstructive sleep apnea had higher sleep latency and presented more sleep fragmentation and less REM sleep than did control subjects\(^{(39)}\).

Our results contribute to better understanding the perpetuation of obesity through feeding behaviours induced by fewer hours of sleep and how it can promote modifications in this highly complex mechanism of energy balance in obese adolescents. We can conclude that sufficient hours of sleep may be essential for maintaining metabolism, even though the mechanisms involved in this process are not yet well defined.
Influence of sleep on obesity

Figure 1 hypothesized how obesity and total sleep time can corroborate each other, worsening the pathology.

Our data suggest a tendency of reduced sleep time among obese adolescents (Table 2), which is consistent with previous studies[8,46]. Many hypotheses have been proposed to address the question of sleep loss in adolescents, being the syndrome of the sleep phase delay (a tendency to stay up later at night and to sleep later in the morning) and the morning school period some of the mainly causes[7,47]. Moreover, a recent study showed that a school time delay of 30 minutes was able to increase 45 minutes of sleep, and the number of students that slept at least 8 hours increased from 16.4% to 54.7%[48]. Therefore, schools should consider a later time of study for adolescents. In the current study, 80% of the students started school activities in the morning (data not shown).

Although sleep has fundamental functions in energy conservation, immunity, metabolism regulation, neural maintenance, memory consolidation and behaviour, the total sleep time has decreased in past years[49]. Sleep debt has been associated with many complications, with obesity and eating disorders being the main factors, which reflects on the worldwide obesity epidemic[50,51]. However, some obese patients can have adequate consumption, like those in the present study, and present an altered energy balance due to the neuroendocrine dysfunction caused by obesity[22].

The small sample size, a lack of control group and subjective assessments represent the limitations of this study. However, our data were empowered by an expressive effect size, suggesting that the vicious cycle between short sleep time, an increased lipid intake, fat mass deposition and sleep latency may occur in obese teenagers. Further investigations with objective analyses and larger sample sizes should be performed for a better understanding of the mechanisms and to extend the results to the general teenage population.

CONCLUSION

The results indicate that less hours of sleep might contribute to the maintenance of obesity in adolescents through an increase in fat intake. Furthermore, we show that the total fat mass is associated with higher sleep latency, contributing to a reduction in sleep time. These data suggest a vicious cycle between eating disorders, obesity and reduced sleep, which can lead to a diminished quality of life in teenagers.

COMPETING INTERESTS

The authors declare that they have no competing interests.

REFERENCES


