

Original article

Relationship of evening meal with sleep quality in obese individuals with obstructive sleep apnea



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ARTICLE INFO

Article history:

Received 18 September 2018

Accepted 30 September 2018

Keywords:

Sleep apnea

Obesity

Food intake

Energy expenditure

Meal distribution

SUMMARY

Purpose: To determine the relationship between habitual food intake, resting energy expenditure and sleep pattern in obstructive sleep apnea (OSA) patients.

Methods: Forty-five OSA obese males were included in the study. All participants were submitted to nocturnal polysomnography, body composition measurements by plethysmography, resting energy expenditure (REE) analysis by indirect calorimetry and they filled in a 3-day food record.

Results: No differences in body composition, REE and food intake were found between the moderate and severe OSA groups. A trend towards higher energy intake in the severe OSA group was observed, compared to the moderate group ($p = 0.08$). Significant associations between apnea–hypopnea index (AHI) with body weight, body mass index (BMI) and resting energy expenditure (REE) were found. Higher food intake in the evening period was positively correlated with sleep stage NREM1, arousal index, and AHI and negatively correlated with sleep stage NREM3 and sleep efficiency. A multivariate linear regression showed energy intake at breakfast to be a significant negative predictor of AHI; protein intake (g/kg) showed a positive association, while energy intake at breakfast and at dinner were negative predictors of sleep efficiency; and energy intake at dinner was a negative predictor of stage NREM1 sleep.

Conclusions: We conclude that higher amounts of food intake during the evening period may diminish sleep quality in moderate and severe sleep apnea patients. In addition, despite observing no differences between OSA severity groups, a moderate correlation between REE and sleep quality and OSA exists.

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1. Introduction

Obstructive sleep apnea (OSA) and obesity are tightly related [1]. Peripheral and visceral fat accumulation predisposes and increases OSA's severity through anatomical changes in the upper airway and increased metabolic and humoral factors released by fat, such as leptin and inflammatory cytokines [2]. Treating obesity in OSA

patients improves disease severity and comorbidities [3]. A potential association between energy balance and sleep-disordered breathing is also discussed in the literature. OSA may favor a positive energy balance, contributing to the development of obesity [4]. The fragmentation of sleep that is caused by OSA may lead to excessive sleepiness and lower energy expenditure (EE) during the day, which is associated with a higher energy intake, contributing to weight gain and obesity development.

In recent years, energy intake and the distribution and regularity of meals have been discussed [5,6]. Hermengildo et al. [7] showed that the practice of ingesting a higher percentage of daily energy at lunch and a lower percentage at dinner is

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associated with lower risk of weight gain. In addition to the relationship between meal distribution and body weight, the intake of energy over the course of a day has been related to sleep quality. The influence of sleep on energy intake and vice versa has been extensively investigated [8–11]. In healthy subjects, energy intake, especially in the evening, has been correlated with worse sleep quality parameters, such as sleep latency and efficiency [10]. All of the above associations might be more relevant in people living with OSA. The consequences of OSA vary from excessive diurnal somnolence [12] to increased risk of metabolic and cardiovascular disease [13].

Lifestyle modification and weight loss are strongly recommended for the treatment of OSA [14]; however, obese individuals face major difficulties in following such recommended changes in lifestyle. Possibly, minor recommendations for changing daily life habits – such as changes in meal times and composition – might be easier to achieve than more complex feeding plans. We hypothesize, therefore, that individuals with OSA who ingest poor quality evening meals present diminished sleep quality, when compared to those ingesting better quality meals. Aside from the increasing interest in diet composition and sleep quality in OSA patients, few studies have discussed the importance of food intake in sleep patterns in these patients. This study proposes to investigate the association between habitual food intake and resting energy expenditure with sleep parameters in OSA patients.

2. Subjects

The participants of this study responded to a media advertisement for a longitudinal study related to sleep apnea and weight loss. The sample of the present study represents the basement evaluation of the longitudinal study. The potential volunteers were evaluated according to inclusion and exclusion criteria. The inclusion criteria were; aged between 30 and 55 years, males, obese (Body Mass Index $> 30 \text{ kg/m}^2 < 45 \text{ kg/m}^2$) diagnosed as OSA (moderate or severe). The exclusion criteria were; the presence of metabolic diseases such as diabetes mellitus, dyslipidemia or thyroid diseases, shift workers, sleep disturbances other than OSA, smoking, physical exercise practitioners or weight loss treatments. The participants were not treated for sleep apnea or obesity. Written, informed consent was obtained before participation in the study, which was approved by the Human Research Ethics Committee at the Federal University of Sao Paulo and was registered with clinicaltrials.gov (NCT01985035).

3. Materials and methods

3.1. Clinical polysomnography (PSG)

To diagnose OSA and determine its severity, we performed full in-laboratory PSG at the sleep laboratory of the Sleep Institute, Federal University of São Paulo. Sleep state stages, respiratory events and arousals were scored according to standard criteria [15] by a single experienced sleep technician. For hypopnea classification, the following criteria were used; reductions of $\geq 30\%$ from the pre-event baseline signal using nasal pressure, duration $\geq 10 \text{ s}$ and higher than 4% desaturation. Apnea was scored when a drop of $\geq 90\%$ from the pre-event baseline signal was determined, using nasal pressure, with a duration of longer than 10 s [15]. The apnea–hypopnea index (AHI) was then calculated from these measurements to provide information about OSA severity; moderate severity corresponds to $\text{AHI} > 15 < 30 \text{ events/h}$; high severity corresponds to $\text{AHI} > 30 \text{ events/h}$.

3.2. Food intake

Food intake was accessed using a self-administered food record for 3 non-consecutive days. For a better understanding of food habits, the food diary was filled in for two week days and one weekend day. All participants were instructed to provide details about all foods and beverages consumed over the evaluated day as well as times of intake. Portion sizes were estimated by common household utensils. The Virtual Nutri Plus[®] software was used for calculating caloric and macronutrient intakes.

3.3. Resting energy expenditure measurements

REE was measured using the device Quark CPET[®] (COSMED). The tests were conducted in the morning, during the fasting state, and in a controlled temperature room (between 20 and 30 °C). The participant was asked to lay down and rest without sleeping, for 30 min before the test beginning. After the rest period, 30 min of recording were obtained.

3.4. Anthropometric and body composition measurements

Body weight was measured using a Filizola scale to the nearest 0.1 kg and height was measured to the nearest 0.5 cm using a stadiometer (Sanny, ES 2030). Body mass index (BMI) was calculated by dividing each individual's body weight by his height squared. Waist and hip circumferences were taken using an in-elastic measuring tape. Body composition was estimated by plethysmography using the BOD POD body composition system (version 1.69, Life Measurements Instruments, Concord, CA) [16].

3.5. Statistical analysis

After the normality investigation of the data, all the variables were presented as means \pm standard deviation. We proceeded to a comparison of the selected variables, between participants classified as moderate and severe OSA (Student's *t*-test for independent samples were used to compare them). Pearson correlations were performed to verify associations among anthropometric, body composition, resting energy expenditure and food intake with sleep parameters. Additionally, different models of linear multivariate regression analysis were used to investigate associations between sleep parameters and meal distribution over the day. The regression models were performed using a backward stepwise method, considering only variables with 95% significance ($p < 0.05$) in the final models. SPSS (IBM; version 20.0) and Prism (GraphPad; version 6.0) software were used for statistical analyses.

4. Results

After recruitment using local media, a total of 163 men of between 30 and 55 years old with previous complaints of OSA responded to the advertisement. After exclusion criteria were applied and polysomnography examinations were performed for OSA diagnosis, only 45 patients were included in the study, with a mean age of 40.8 ± 6.9 years. For a better understanding of the effects of OSA on the variables studied, results are presented for all participants and groups of OSA severity (moderate: $\text{AHI} > 15 < 30 \text{ events/h}$; and severe: $\text{AHI} > 30 \text{ events/h}$). No statistical differences were observed between OSA severity groups for the weight, BMI, and others anthropometric and body composition measurements (Table 1). When analysis were made for all participants, a positive correlation between AHI and body weight ($r = 0.33$; $p = 0.024$), BMI ($r = 0.45$; $p = 0.024$), and body fat ($r = 0.305$; $p = 0.041$).

Table 1
Age and anthropometric characteristics of OSA patients.

Variables	All participants, N = 45	Moderate, N = 18	Severe, N = 27	p Value
Age (years)	40.8 ± 6.9	41.4 ± 6.9	40.4 ± 6.8	0.630
Body mass (kg)	108.5 ± 13.7	107.9 ± 10.5	108.8 ± 15.53	0.845
Height (m)	1.76 ± 0.06	1.78 ± 0.05	1.75 ± 0.07	0.178
BMI (kg/m ²)	35.0 ± 3.8	34.3 ± 3.3	35.5 ± 3.9	0.271
Waist circumference (cm)	113.3 ± 9.2	116.9 ± 9.8	113.1 ± 9.6	0.968
Hip circumference (cm)	112.6 ± 13.1	109.7 ± 18.0	114.6 ± 7.9	0.235
Fat (kg)	42.9 ± 10.3	42.5 ± 10.3	43.1 ± 10.4	0.852
Fat (%)	39.0 ± 5.7	39.1 ± 6.6	39.0 ± 5.1	0.990
Fat-free mass (kg)	65.4 ± 7.1	65.4 ± 6.0	65.4 ± 7.7	0.977
Fat-free mass (%)	60.8 ± 5.8	61.0 ± 6.5	60.7 ± 5.4	0.862

Values are means ± SD. BMI: body-mass index; Student's *t* test was performed between moderate and severe groups.

As expected, the severe OSA group presented a more superficial sleep, as demonstrated by the higher percentage of stage N1 sleep and a lower percentage of stage N3 sleep. AHI, respiratory disturbance index (RDI), arousal index (AI), minimum SaO₂ and time spent in SaO₂ < 90% were worse in the severe OSA group than in the moderate OSA group (Table 2).

No significant differences were found for EE and for food intake measurements between the OSA severity groups (Table 3). There was a trend towards increased energy intake in the severe group, while macronutrient intake distribution was similar between the groups. Interestingly, moderate associations were found between REE (kcal/day) and AHI ($r = 0.33$; $p = 0.025$); sleep efficiency ($r = -0.29$; $p = 0.004$), percent of stage NREM1 sleep ($r = 0.41$; $p = 0.005$) and arousal index ($r = 0.36$; $p = 0.014$).

On average, our sample ingested 3.9 ± 0.7 meals per day, and no significant differences were observed between the OSA severity groups (3.9 ± 0.6 and 3.9 ± 0.7 for moderate and severe groups, respectively; $p = 0.864$; Fig. 1). The moderate and severe groups demonstrated similar percentages of caloric intake at all meals (breakfast: 20.7 ± 7.0% and 16.2 ± 6.5% for the moderate and severe groups, respectively; $p = 0.061$; lunch: 37.4 ± 7.4% and 37.4 ± 8.1% for the moderate and severe groups, respectively; $p = 0.977$; evening meal: 28.8 ± 7.0% and 34.2 ± 10.8% for the moderate and severe groups, respectively; $p = 0.110$). In addition, the macronutrient composition of the evening meal did not correlate with any of the sleep parameters.

Pearson's correlations revealed associations between evening dinner size (percentage of total caloric intake) and sleep parameters (Fig. 2). The caloric intake at dinner was positively associated with stage NREM1 sleep ($r = 0.55$; $p < 0.001$) and AHI ($r = 0.35$; $p = 0.03$), and negatively associated with stage NREM3 sleep ($r = -0.53$; $p < 0.001$) and sleep efficiency ($r = -0.35$; $p = 0.03$). No

significant correlations were found between other meals and sleep patterns. Different models of a multivariate linear regression were calculated (Table 4) using different sleep variables as dependent variables. Dietary variables, such as total energy and nutrient intake and percentage of caloric intake in different meals, were tested as independent variables. The first model used AHI as a dependent variable, and only the energy intake at breakfast (percentage of TCI) was a significant negative predictor. The second model used sleep efficiency as a dependent variable, and protein intake (g/kg) showed a positive association with sleep efficiency, while energy intake at breakfast and at dinner were negative predictors. In the third model, stage NREM1 sleep was the dependent variable, and only the energy intake at dinner (percentage of TCI) was a significant negative predictor.

5. Discussion

The principal finding of the current study was that food intake in the evening was associated with a diminished sleep quality in sleep apnea subjects. To our knowledge, this is the first study to show this association based on habitual food intake in OSA patients. These results indicate that a higher food intake in the evening may contribute to OSA severity, and recommendations regarding the amount and time of food intake close to the sleeping period should be included in the current OSA treatment protocol.

The relationship between body mass and sleep apnea is well known, and obesity is one of the most important risk factors for OSA. Our study is in agreement with others regarding the positive association between body weight and BMI with AHI [17,18]. Weight loss is highly encouraged as an adjunct therapy for OSA [19] and, considering the current obesogenic environment, as well as the difficulties in losing weight or maintaining a healthy body weight,

Table 2
Polysomnography data of sleep apnea patients studied.

Variables	All participants, N = 45	Moderate, N = 18	Severe, N = 27	p Value
Latency (min)	10.7 ± 11.6	10.9 ± 9.8	10.5 ± 12.7	0.916
TST (min)	365.6 ± 51.2	369.9 ± 47.6	362.7 ± 53.3	0.653
Sleep efficiency (%)	86.7 ± 9.9	89.1 ± 8.0	85.1 ± 10.8	0.195
NREM_Stage 1 (%)	17.7 ± 11.6	11.5 ± 6.2	21.8 ± 12.5	0.003
NREM_Stage 2 (%)	47.8 ± 9.3	48.1 ± 10.3	47.6 ± 8.6	0.866
NREM_Stage 3 (%)	15.2 ± 8.9	19.7 ± 7.3	12.2 ± 8.6	0.005
REM (%)	19.3 ± 7.2	20.7 ± 7.8	18.4 ± 6.7	0.297
WASO (min)	44.6 ± 40.1	34.9 ± 34.3	51.1 ± 42.4	0.195
RDI (n/h)	50.3 ± 23.4	28.1 ± 4.9	65.1 ± 18.7	0.000
AHI (n/h)	47.3 ± 25.2	22.8 ± 3.1	63.6 ± 19.6	0.000
Arousal index (n/h)	36.0 ± 19.2	21.6 ± 7.7	45.6 ± 18.6	0.000
SaO ₂ minimum (%)	75.5 ± 8.7	81.3 ± 4.3	71.6 ± 8.8	0.000
Time spent SaO ₂ < 90% (% of TST)	15.4 ± 17.3	5.1 ± 6.5	22.6 ± 18.8	0.001

Values are means ± SD. TST: total sleep time; NREM: non-rapid eye movement sleep; REM: rapid eye movement sleep; AHI: apnea/hypopnea index; SaO₂: arterial blood oxygen saturation. Student's *t* test was performed between moderate and severe groups.

Table 3
Resting metabolic rate, energy and macronutrient intake in moderate and severe OSA male subjects.

Variables	All participants, N = 45	Moderate, N = 18	Severe, N = 27	p Value
RMR (kcal/day)	2372.2 ± 332.3	2321 ± 327.3	2406.0 ± 331.5	0.414
RMR/kg (kcal/kg)	22.0 ± 2.8	21.6 ± 2.8	22.3 ± 2.7	0.393
RMR/LBM (kcal/kg)	36.5 ± 5.3	35.8 ± 5.8	37.0 ± 4.8	0.463
RQ	0.869 ± 0.09	0.875 ± 0.09	0.566 ± 0.1	0.754
kcal (day)	2921.5 ± 639.6	2709.0 ± 504.2	3083.5 ± 683.0	0.082
Carbohydrates (g/day)	339.9 ± 90.1	325.0 ± 85.1	351.3 ± 92.1	0.394
Protein (g/day)	122.5 ± 31.1	118.0 ± 29.8	125.9 ± 31.6	0.459
Lipids (g/day)	113.2 ± 30.2	105.3 ± 21.9	119.1 ± 34.1	0.179
Carbohydrates (g/kg/day)	2.65 ± 1.48	3.06 ± 0.87	3.35 ± 0.91	0.352
Protein (g/kg/day)	1.17 ± 0.34	1.11 ± 0.29	1.21 ± 0.36	0.371
Lipids (g/kg/day)	0.88 ± 0.50	0.99 ± 0.21	1.14 ± 0.37	0.352
Carbohydrates (%)	58.1 ± 6.0	58.3 ± 6.0	58.0 ± 6.0	0.917
Protein (%)	21.3 ± 3.7	21.1 ± 3.3	21.4 ± 3.9	0.856
Lipids (%)	19.6 ± 3.5	19.4 ± 4.1	19.7 ± 3.1	0.790

Values are medians means ± SD. RMR: resting metabolic rate; RQ: respiratory quotient; LBM: lean body mass. Student's *t* test was performed between moderate and severe groups.

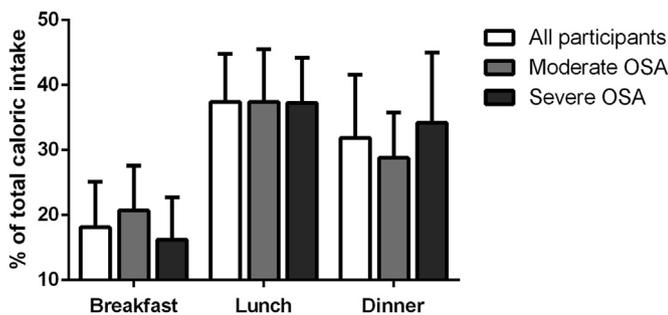


Fig. 1. Meal distribution in moderate and severe OSA patients.

Table 4
Multivariate linear regression analysis.

Model	Dependent variable	Predictors		
		Variable	B (SE)	p
Model 1	AHI, $R^2 = 0.10$	Breakfast (%TCI)	-0.36 (0.16)	0.03
Model 2	Sleep efficiency, $R^2 = 0.28$	PTN/kg/day	0.96 (0.35)	0.01
		Breakfast (%TCI)	-0.54 (0.18)	0.007
		Dinner (%TCI)	-0.51 (0.19)	0.013
Model 3	N1 (% TST), $R^2 = 0.32$	Dinner (%TCI)	-0.54 (0.21)	0.016

AHI: apnea–hypopnea index; NREM1: sleep stage non REM 1; PTN: protein; TCI: total caloric intake.

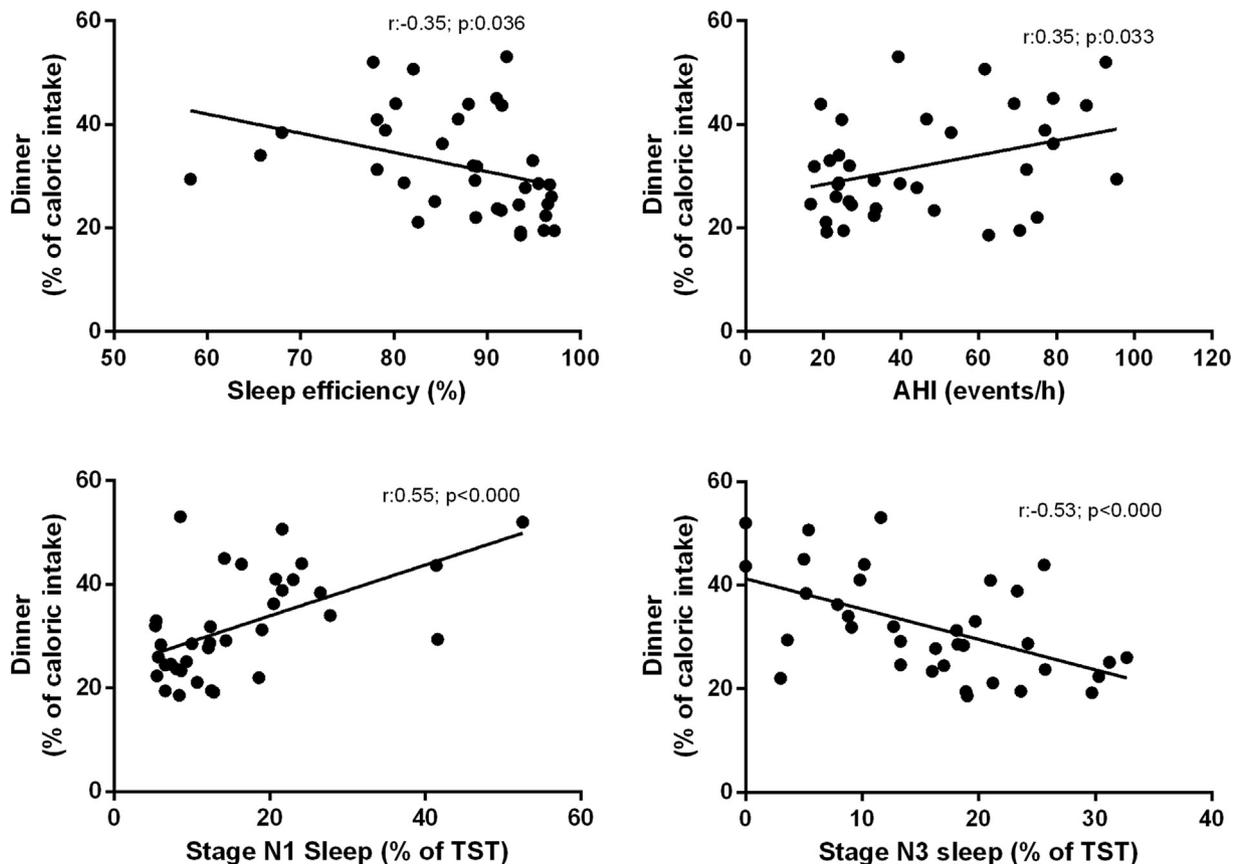


Fig. 2. Pearson correlations between sleep patterns and food intake during the evening period in OSA patients.

special attention and incessant encouragement to achieve improvements in diet quality should be given by all professionals engaged in OSA treatment.

Sleep quantity and quality have been related to body composition changes; in animals, sleep restriction is related to a large negative energy balance and fat-free mass loss [20,21]. In humans, sleep restriction in association with an energy-restricted diet also resulted in higher fat-free mass loss [22], and associations between body composition and AHI in OSA individuals have also been reported [23,24]. In a large sample, Kosacka et al. [23] showed that severe OSA patients have reduced muscle mass and total body water compared with control subjects. In contrast, our study did not detect differences in body composition between OSA severity groups. The positive correlation between AHI and fat mass corroborate the association between weight gain and OSA severity. The use of body composition, especially body fat measurement, might be important in OSA patients [24].

Although controversial, there is some evidence to suggest that EE is altered in OSA patients. Stenlöf et al. [25] observed higher REE in OSA than in snoring subjects. Major et al. [26] found a negative correlation between the time spent in SaO_2 of lower than 90% and REE in twenty-four adult OSA patients. Furthermore, O'Driscoll et al. [27] described a significant association between AHI and total (24 h) EE. Our study did not find significant differences in REE between the OSA severity groups, but in agreement with the aforementioned studies, we found associations, though weak, between total REE and AHI and total arousals. Methodological differences may be responsible for controversial results in the literature; however, it is possible that high OSA severity and increased arousals over the night contribute to higher REE. It is important to emphasize that REE is only one component of total EE, and other components such as activities related to EE and the thermic effect of food might also be altered in OSA patients.

Associations between food intake and sleep patterns have been previously described in healthy individuals. In a study of 52 healthy women and men, Crispim et al. [10] found that a higher food intake close to sleeping periods is associated with worse sleep quality. The percentage of caloric intake at night was positively associated with sleep latency and negatively associated with sleep efficiency in women. In contrast to our results, Crispim et al. [10] and others [28] found relationships between macronutrient intake and sleep quality. To our knowledge, only one study has evaluated the effect of food intake on sleep parameters in OSA patients. In a crossover study, Trakada et al. [29] tested the effects of a 360 kcal meal and of a hypercaloric (1800 kcal) fat-rich meal 2 h before sleep in OSA patients. The authors found an increase in total sleep time, AHI and the total number of obstructive and central apneas after the fat-rich meal. These results suggest the importance of food intake control before sleep in sleep apnea subjects. It is important to highlight the fact that the fat-rich meal in this study was also hypercaloric—with more than twice the calories of a normal meal—and that 70% of the calories came from fat, which is an unusual meal composition in daily life. Aside from the importance of the experimental design of the study, this kind of meal does not represent the habitual food intake of the general adult population. Our study found associations between food intake in the evening and sleep patterns with usual meals of approximately 1000 kcal (32% of total caloric intake in all participants) and a macronutrient distribution that was similar to that of the all-day food intake. Our multivariate linear regression analysis showed associations between AHI, sleep efficiency and stage NREM1 sleep with calorie intake during breakfast and dinner. Additionally, protein intake was a predictor of sleep efficiency, in agreement with the findings of other studies that have suggested that increased protein intake is related to better sleep parameters in non-OSA subjects [30,31].

Circadian patterns influence food intake and digestion, absorption and metabolic functions [32]. Sleep is known to decrease digestive function [32], and the discomfort of increased gastric volume during the night might contribute to less deep sleep [10]. This might be particularly important for OSA patients. In addition to the acute effects of a big meal before sleep on sleep quality, another important issue is whether eating more in the evenings is associated with weight gain in OSA patients. Some studies report that people who eat greater amounts of food in the evenings usually have more unhealthy overall dietary habits. In an obese and sleep-restricted population, Lucassen et al. [33] found that the evening chronotype was associated with unhealthy eating patterns, such as eating late, reduced meal numbers during the day and eating large portion sizes. Moreover, the evening chronotype individuals were more likely to have sleep apnea. Gastroesophageal reflux disease (GERD) has also been linked to sleep quality and, especially, OSA. GERDs are associated with obesity, and studies suggest that OSA patients have a higher incidence of GERD [34]. Elevated amounts of food in the evenings contribute to worsening GERD symptoms, which, in turn, are related to reduced sleep quality.

The relationship between sleep quality and diet might be of concern, especially for sleep apnea patients. Reduced quality of sleep is likely to increase the caloric intake from fat and carbohydrates, reduce the intake of healthy foods, such as vegetables and fruits, and may induce more irregular meal patterns and the more unhealthy snacks [35]. On the other hand, unhealthy eating habits contribute to weight gain and worsening OSA. Nutritional counselling to recommend the increased intake of fruits, vegetables, fiber-rich foods and less high-energy density foods is important to help improve sleep quality in OSA patients.

Our study presents some limitations. Due to the observational nature of this study, we are not able to precisely detect causality between energy intake and nutrient intake in the evening and sleep quality. However, considering the study by Trakada et al. [29] and other studies with healthy individuals, the findings of our study support the idea that OSA patients should consider their eating habits before sleeping. Our sample was possibly not big enough to detect significant differences in body composition and REE between different severities of OSA. Furthermore, the food intake of the participants was self-reported, a common limitation across studies using food data and, finally, the records were not made on the same day as that on which the polysomnography study was conducted, but a three-day food diary is a reliable instrument to detect food habits. The use of food intake recall on the day of the polysomnography exam would be a better measurement of the energy and nutrient intake effect on sleep patterns, but would not be a good method for detecting habitual food intake. Our results reflect the habitual food intake of these patients.

In conclusion, the current study demonstrates that higher food intake in the evening might be deleterious for sleep quality and OSA severity in moderate and severe sleep apnea patients, we can also conclude that OSA severity is associated with increased resting EE. More studies to examine meal quality and quantity are recommended for OSA patients.

Statement of authorship

We declare that all authors participated enough to be considered an author of the present manuscript. Also, all authors have seen and approved the final version of this manuscript.

Conflict of interest statement

We declare that CMM, MPDR, MVLS, HKMA, SMT, SMLR, ST, MTM: Have no conflicts to disclose and do not have a financial

relationship with a commercial entity that has an interest in the subject of this manuscript.

Funding

CNPq (Conselho Nacional de Desenvolvimento Científico e Tecnológico), CAPES (Coordenação de Aperfeiçoamento Pessoal de Nível Superior), and FAPESP (Fundação de Amparo à pesquisa do Estado de São Paulo – Fund number: 2012/09700-2) provided financial support in the form of research scholarships and project funding. The sponsor had no role in the design or conduct of this research.

Acknowledgments

The authors thank the Associação Fundo de Incentivo à Pesquisa (AFIP), Centro de Estudos em Psicobiologia e Exercício (CEPE), CNPq (Conselho Nacional de Desenvolvimento Científico e Tecnológico), CAPES (Coordenação de Aperfeiçoamento Pessoal de Nível Superior), FAPESP (Fundação de Amparo à pesquisa do Estado de São Paulo – Fund number: 2012/09700-2), Lemann Foundation, and Mrs. Nicola A. Zorzetto for the English review.

References

- Romero-Corral A, Caples SM, Lopez-Jimenez F, Somers VK. Interactions between obesity and obstructive sleep apnea. *Chest* 2010;137:711–9. <https://doi.org/10.1378/chest.09-0360>.
- Schwartz AR, Patil SP, Squier S, Schneider H, Kirkness JP, Smith PL. Obesity and upper airway control during sleep. *J Appl Physiol* 2010;108:430–5. <https://doi.org/10.1152/jappphysiol.00919.2009>.
- Patel SR, Mehra R. The weighty issue of obesity management in sleep apnea. *Chest* 2015;148:1127–9. <https://doi.org/10.1378/chest.15-1010>.
- Fekete K, Boutou AK, Pitsiou G, Chavouzis N, Pataka A, Athanasiou I, et al. Resting energy expenditure in OSAS: the impact of a single CPAP application. *Sleep Breath* 2016;20:121–8. <https://doi.org/10.1007/s11325-015-1194-y>.
- Bertéus Forslund H, Lindroos AK, Sjöström L, Lissner L. Meal patterns and obesity in Swedish women – a simple instrument describing usual meal types, frequency and temporal distribution. *Eur J Clin Nutr* 2002;56:740–7. <https://doi.org/10.1038/sj.ejcn.1601387>.
- Alhussain MH, MacDonald IA, Taylor MA. Irregular meal-pattern effects on energy expenditure, metabolism, and appetite regulation: a randomized controlled trial in healthy normal-weight women. *Am J Clin Nutr* 2016;104:21–32. <https://doi.org/10.3945/ajcn.115.125401>.
- Hermengildo Y, López-García E, García-Esquinas E, Pérez-Tasigchana RF, Rodríguez-Artalejo F, Guallar-Castillón P. Distribution of energy intake throughout the day and weight gain: a population-based cohort study in Spain. *Br J Nutr* 2016;115:1–8. <https://doi.org/10.1017/S0007114516000891>.
- McNeil J, Doucet É, Brunet J-F, Hintze LJ, Chaumont I, Langlois É, et al. The effects of sleep restriction and altered sleep timing on energy intake and energy expenditure. *Physiol Behav* 2016;164:157–63. <https://doi.org/10.1016/j.physbeh.2016.05.051>.
- Broussard JL, Kilkus JM, Delebecque F, Abraham V, Day A, Whitmore HR, et al. Elevated ghrelin predicts food intake during experimental sleep restriction. *Obesity (Silver Spring)* 2016;24:132–8. <https://doi.org/10.1002/oby.21321>.
- Crispim CA, Zimberg IZ, dos Reis BG, Diniz RM, Tufik S, de Mello MT. Relationship between food intake and sleep pattern in healthy individuals. *J Clin Sleep Med* 2011;7:659–64. <https://doi.org/10.5664/jcsm.1476>.
- Nedelcheva AV, Kessler L, Imperial J, Penev PD. Exposure to recurrent sleep restriction in the setting of high caloric intake and physical inactivity results in increased insulin resistance and reduced glucose tolerance. *J Clin Endocrinol Metab* 2009;94:3242–50. <https://doi.org/10.1210/jc.2009-0483>.
- Rodenstein D. Sleep apnea: traffic and occupational accidents – individual risks, socioeconomic and legal implications. *Respiration* 2009;78:241–8. <https://doi.org/10.1159/00022811>.
- Loke YK, Brown JW, Kwok CS, Niruban A, Myint PK. Association of obstructive sleep apnea with risk of serious cardiovascular events: a systematic review and meta-analysis. *Circ Cardiovasc Qual Outcomes* 2012;5:720–8. <https://doi.org/10.1161/CIRCOUTCOMES.111.964783>.
- Thomasouli M-A, Brady EM, Davies MJ, Hall AP, Khunti K, Morris DH, et al. The impact of diet and lifestyle management strategies for obstructive sleep apnoea in adults: a systematic review and meta-analysis of randomised controlled trials. *Sleep Breath* 2013. <https://doi.org/10.1007/s11325-013-0806-7>.
- Berry RB, Budhiraja R, Gottlieb DJ, Gozal D, Iber C, Kapur VK, et al. Rules for scoring respiratory events in sleep: update of the 2007 AASM manual for the scoring of sleep and associated events. *J Clin Sleep Med* 2012;8:597–619. <https://doi.org/10.5664/jcsm.2172>.
- Fields DA, Goran MI, McCrory MA. Body-composition assessment via air-displacement plethysmography in adults and children: a review. *Am J Clin Nutr* 2002 Mar;75(3):453–67.
- Peppard PE, Young T, Palta M, Dempsey J, Skatrud J. Longitudinal study of moderate weight change and sleep-disordered breathing. *J Am Med Assoc* 2000;284:3015–21. <https://doi.org/10.1001/jama.284.23.3015>.
- Preto JJ, Gyulay SG, Hensley MJ. Trends in anthropometry and severity of sleep-disordered breathing over two decades of diagnostic sleep studies in an Australian adult sleep laboratory. *Med J Aust* 2010;193:213–6.
- Qaseem A, Holty JE, Owens DK, Dallas P, Starkey M, Shekelle P. Management of obstructive sleep apnea in adults: a clinical practice guideline from the American College of Physicians. *Ann Intern Med* 2013;159:471–83. <https://doi.org/10.7326/0003-4819-159-7-201310010-00704>.
- Hipolide DC, Suchecki D, Pinto AP, Chiconelli Faria E, Tufik S, Luz J. Paradoxical sleep deprivation and sleep recovery: effects on the hypothalamic-pituitary-adrenal axis activity, energy balance and body composition of rats. *J Neuroendocrinol* 2006;18:231–8. <https://doi.org/10.1111/j.1365-2826.2006.01412.x>.
- Mônico-Neto M, Giampá SQ de C, Lee KS, de Melo CM, Souza H de S, Dáttilo M, et al. Negative energy balance induced by paradoxical sleep deprivation causes multicompartimental changes in adipose tissue and skeletal muscle. *Int J Endocrinol* 2015;2015:1–6. <https://doi.org/10.1155/2015/908159>.
- Nedelcheva AV, Kilkus JM, Imperial J, Schoeller DA, Penev PD. Insufficient sleep undermines dietary efforts to reduce adiposity. *Ann Intern Med* 2010;153:435–41.
- Kosacka M, Korzeniewska A, Jankowska R. The evaluation of body composition, adiponectin, C-reactive protein and cholesterol levels in patients with obstructive sleep apnea syndrome. *Adv Clin Exp Med* 2013;22:817–24.
- Bezerra PC, do Prado M, Gaio E, Franco OL, Tavares P. The use of dual-energy X-ray absorptiometry in the evaluation of obesity in women with obstructive sleep apnea-hypopnea syndrome. *Eur Arch Otorhinolaryngol* 2013;270:1539–45. <https://doi.org/10.1007/s00405-012-2291-1>.
- Stenlöf K, Grunstein R, Hedner J, Sjöström L. Energy expenditure in obstructive sleep apnea: effects of treatment with continuous positive airway pressure. *Am J Physiol* 1996;271:E1036–43.
- Major GC. Does the energy expenditure status in obstructive sleep apnea favour a positive energy balance? *Clin Investig Med* 2007;30:262–8.
- O'Driscoll DM, Turton AR, Copland JM, Strauss BJ, Hamilton GS. Energy expenditure in obstructive sleep apnea: validation of a multiple physiological sensor for determination of sleep and wake. *Sleep Breath* 2013;17:139–46. <https://doi.org/10.1007/s11325-012-0662-x>.
- Afaghi A, O'Connor H, Chow CM. High-glycemic-index carbohydrate meals shorten sleep onset. *Am J Clin Nutr* 2007;85:426–30.
- Trakada G, Steiropoulos P, Zarogoulidis P, Nena E, Papanas N, Maltezos E, et al. A fatty meal aggravates apnea and increases sleep in patients with obstructive sleep apnea. *Sleep Breath* 2014;18:53–8. <https://doi.org/10.1007/s11325-013-0847-y>.
- Lindseth G, Lindseth P, Thompson M. Nutritional effects on sleep. *West J Nurs Res* 2013;35:497–513. <https://doi.org/10.1177/0193945911416379>.
- Grandner MA, Jackson N, Gerstner JR, Knutson KL. Dietary nutrients associated with short and long sleep duration. Data from a nationally representative sample. *Appetite* 2013;64:71–80. <https://doi.org/10.1016/j.appet.2013.01.004>.
- Tahara Y, Shibata S. Chronobiology and nutrition. *Neuroscience* 2013;253:78–88. <https://doi.org/10.1016/j.neuroscience.2013.08.049>.
- Lucassen EA, Zhao X, Rother KI, Mattingly MS, Courville AB, de Jonge L, et al. Evening chronotype is associated with changes in eating behavior, more sleep apnea, and increased stress hormones in short sleeping obese individuals. *PLoS One* 2013;8. <https://doi.org/10.1371/journal.pone.0056519>.
- Mulrennan SA, Knuiman MW, Divitini ML, Cullen DJ, Hunter M, Hui J, et al. Gastro-oesophageal reflux and respiratory symptoms in Busselton adults: the effects of bodyweight and sleep apnoea. *Intern Med J* 2012;42:772–9. <https://doi.org/10.1111/j.1445-5994.2011.02535.x>.
- Peuhkuri K, Sihvola N, Korpela R. Diet promotes sleep duration and quality. *Nutr Res* 2012;32:309–19. <https://doi.org/10.1016/j.nutres.2012.03.009>.