Dietary Inflammatory Index of Obese Individuals with Obstructive Sleep Apnea: A Descriptive Study

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Abstract

The objective of the present study was to describe the dietary inflammatory profile and its correlations with sleep parameters of obese individuals with obstructive sleep apnea (OSA). Forty individuals underwent nocturnal polysomnography, anthropometric measurements, body composition by plethysmography, assessment of food consumption by three-day food records, and blood collection for the lipid, glycemic and hormonal profile. Food consumption data were evaluated semiquantitatively, quantitalively assessment, and calculation of the dietary inflammatory index (DII) was perfomed. The results demonstrated a predominantly proinflammatory dietary profile. The participants showed a low intake of fruit and vegetables. Additionally, a low consumption of fiber, magnesium, vitamin D, and vitamin E was observed, although there was an adequate distribution of macronutrients. In conclusion, although the inflammatory profile did not correlate with OSA, the study showed a directly proportional relationship between adequate dietary patterns and better sleep quality.

Keywords
- obstructive sleep apnea
- dietary inflammatory index
- dietary patterns
- sleep

Introduction

Obstructive sleep apnea (OSA) is a respiratory disorder characterized by repeated upper airway obstructions during sleep, culminating in recurrent pauses or decreased respiratory flow.[1] Obesity and OSA have been shown to be strongly associated and the metabolic pathways by which inflammation is triggered are similar for both. These conditions are related to excess body adipose tissue, which has an important proinflammatory role, responsible for the production of proinflammatory cytokines.[2]

Currently, many studies have investigated the role of diet in modulating systemic inflammation. These investigations have pointed to some dietary patterns associated with the production of anti-inflammatory biomarkers such as the intake of fruits, vegetables, whole grains, and fish, and low

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content of sugar and fat. Otherwise, patterns based on the intake of red meat, alcohol, and processed and refined foods have been associated with increased proinflammatory status.[3]

The dietary inflammatory index (DII), developed by Cavicchia et al.[4] and Shivappa et al.,[5] has been used to evaluate the inflammatory potential of the diet. It addresses the effect of certain foods, nutrients, and food components on the main markers of systemic inflammation, such as C-reactive protein (CRP), interleukin 1-β (IL-1β), interleukin 4 (IL-4), interleukin 6 (IL-6), interleukin 10 (IL-10) and tumor necrosis factor alpha (TNF-α).[5] Several studies have associated the DII with chronic noncommunicable diseases, such as cardiometabolic diseases, obesity, diabetes, and certain types of cancer, as well as an increased risk of mortality.[6,7] However, in Brazil, only one study[8] has evaluated the association between the DII and OSA.

Thus, the aim of the present study is to assess the DII and its association with OSA and sleep parameters in a sample of obese men diagnosed with OSA. As a secondary aim, biochemical, body composition and dietary quality aspects were correlated with the DII and sleep parameters.

Materials and Methods

The present study is secondary to a broader investigation;[9] the original protocol was approved by the Ethics Committee on Research in Human Beings of Universidade Federal de São Paulo and registered at clinicaltrials.gov (NCT01985035). The participants provided consent by signing the Informed Consent Form.

Sample Selection

The participants were recruited through media outreach with pamphlets and advertisements in local newspapers and magazines. The recruitment and data collection occurred between 2014 and 2015. The potential participants were regarding the inclusion criteria, namely, male sex, age between 30 and 55 years, and body mass index (BMI) between 30 kg/m^2 and 45 kg/m^2. The exclusion criteria were absence of metabolic diseases such as diabetes mellitus, dyslipidemia or thyroid disease, sleep disorders other than OSA, performance of shift work, physical activity, and being submitted to weight loss treatments at the time of the research.

Diagnosis and Classification of OSA

For the evaluation of sleep parameters and the diagnosis of OSA, polysomnography was performed using the Embla digital system (Embla Systems Inc., Broomfield, CO, United States). The variables obtained were total sleep time, sleep efficiency, sleep latency, wake after sleep onset, rapid eye movement (REM) sleep latency, microarousals, non-REM (NREM) sleep stages, REM sleep, the apnea-hypopnea index (AHI), the respiratory disorder index, thoracic and abdominal respiratory effort by uncalibrated inductance plethysmography, and oxyhemoglobin saturation (SpO2). The analysis of the variables and of the severity of OSA were performed based on the criteria of the American Academy of Sleep Medicine.[10]

Biochemical Evaluation

Blood samples were collected from all participants. The biochemical parameters analyzed were glycemia, total cholesterol and fractions from the enzymatic colorimetric method, cortisol, insulin, and insulin-like growth Factor I (IGF-I) by the chemiluminescence method. For the hormones, the enzyme-linked immunosorbent assay (ELISA) method was used to obtain data on the following: leptin (EZHL-80SK; EDM Millipore, Burlington, MA, United States), acylated ghrelin (EZGRA-88K; EDM Millipore), adiponectin (EZHADP-61K; EDM Millipore), and irisin (EK-067–29; Phoenix Pharmaceuticals, Inc., Burlingame, CA, United States).

Anthropometry and Body Composition

The anthropometric measurements included weight, height, BMI, and waist circumference. Body composition was obtained through the air displacement plethysmography method and analyzed using the BOD POD body composition system (Life Measurement, Inc., Concord, CA, United States).

Quantitative Assessment of Food Intake

Food records of three nonconsecutive days (including one day of the weekend) were used to assess the food intake. The participants were instructed to detail the food and beverages they had consumed, as well as their quantities, for all meals. Energy, macronutrient and micronutrient intake were analyzed using the Nutrition Data System for Research (NDSR; Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN, United States) software, version 2013. Data from flavonoid compounds were obtained through specific food composition databases.[11,12] Finally, each food component was inserted separately in the Multiple Source Method (MSM; Department of Epidemiology, German Institute of Human Nutrition Potsdam-Rehbrücke, Nuthetal, Germany) software to correct the intra-individual variability of the food intake. The adequacy of nutrient ingestion was investigated according to the estimated average requirement (EAR) for the micronutrients, and according to the Acceptable Macronutrient Distribution Range (AMDR) for the macronutrients.[13]

Calculation of the Dietary Inflammatory Index

For the calculation of the inflammatory dietary profile, we included the largest number of food components among the 45 suggested by the authors.[4,5] As such, the components included were: total energy (Kcal); fats, carbohydrates and total proteins (g); alcohol (g); cholesterol (mg); total saturated fatty acids (g); total monounsaturated fatty acids – MUFAs (g); total polyunsaturated fatty acids – PUFAs (g); total fiber (g); total vitamin A in terms of retinol equivalents (mcg); equivalents of beta-carotene (mcg); vitamin D (calciferol; mcg); vitamin E (total α tocopherol; mg); vitamin C (ascorbic acid; mg); vitamin B1 (thiamine; mg); vitamin B2–riboflavin (mg); vitamin B3 (niacin; mg); vitamin B6 (mg); total folate (mcg); vitamin B12 (cobalamin; mcg); magnesium (mg); iron (mg); zinc (mg); selenium (mcg); caffeine (mg); total trans fatty acids (g); omega 3 (g); omega 6 (g); isoflavones (mg); onion (g); garlic (g); green/black tea

(g); pepper (g); anthocyanins (mg); flavan-3-ols (mg); flavanones (mg); flavonols (mg); and flavones (mg). In the present study, there was no consumption of eugenol, turmeric, thyme/oregano, rosemary, ginger, and saffron.

The calculation of the DII was performed in a Microsoft Excel (Microsoft Corp. Redmond, WA, United States) spreadsheet using the values of average overall daily intake, standard deviation (SD), and overall inflammatory effect score. For each component, the following steps were performed: first, the Z score was calculated by subtracting the average intake of the individual by the mean global reference intake of each food component, followed by the division of this value by the SD of the same component. The second step was to convert the Z score into a percentile. In the third step, the percentile was transformed into a centralized percentile, multiplied by two and subtracted from one to achieve a symmetrical score. In the fourth step, the value obtained was then multiplied by the inflammatory effect score of the food component, providing the inflammatory effect score of each food component evaluated at the end. Finally, to obtain the general inflammatory effect for each individual, the inflammatory effects of all the food components were added. With the inclusion of all 45 food components, the final score could range from -8.87, which indicates a more anti-inflammatory diet, and +7.98, which indicates a more proinflammatory diet, according to data from the authors of the index.[4,5] The minimum values reflected a higher anti-inflammatory effect, and the maximum values indicated a proinflammatory effect.

Semiquantitative and Qualitative Description of the Diet

A semiquantitative evaluation of the consumption of vegetables, legumes and fruits was also performed. To quantify the portions consumed, the table of equivalences available in the previous version of the Food Guide for the Brazilian population was used.[14] The food intake was also classified according to the NOVA classification.[15] The categories are defined as follows: unprocessed or minimally processed foods, processed foods, and ultra-processed foods.[15]

Data Analysis

First, the variables were analyzed for normal distribution using the Shapiro–Wilk test. The results for the continuous variables are presented as mean($\pm$ SD) values. Pearson correlations and simple regression analysis were performed to evaluate the relationships regarding the DII and the other variables. The Student *t*-test was used to compare variables between moderate and severe OSA groups. The level of significance adopted was of 95% ($p < 0.05$). All analyses were performed using the IBM SPSS Statistics for Windows (IBM Corp., Armonk, NY, United States) software, version 20.0.

Results

The present study was conducted with 40 obese male participants diagnosed with moderate (AHI ranging from 15 to 30 events/h) or severe (AHI >30 events/h) OSA. The mean age was of 40.77 $\pm$ 7.17 years, and the mean BMI, of 34.7 $\pm$ 3.6 kg/m^2 (range: 29.6 kg/m^2 to 43.0 kg/m^2), indicating a sample of obese individuals. ►**Table 1** describes the anthropometric, body composition and biochemical measurements of the sample. Regarding the biochemical variables, on average, increased levels of total cholesterol (192.9 $\pm$ 34.26 mg/dL), insulin (17.7 $\pm$ 7.7 µUI/mL), and leptin (12.58 $\pm$ 7.48 ng/mL) were observed. Reduced levels of high-density lipoprotein (HDL) cholesterol (39.6 $\pm$ 6.55 mg/dL) were also observed.

The sleep parameters are presented in ►**Table 2**. Most participants presented severe OSA, reduced total sleep time (368.5 $\pm$ 50.01 m), and increased NREM stage-1 sleep.

The DII presented a mean value of 1.109 $\pm$ 1.369 (minimum: -2.791; maximum: 3.66); this positive mean value indicated a proinflammatory dietary profile. For the DII calculation, 39 food components were included, and 24 of them contributed to the proinflammatory profile (►**Table 3**). The principal contributors to an anti-inflammatory effect were omega-3 fatty acids (-0.422 $\pm$ 0.015), zinc (-0.269 $\pm$ 0.102), flavonols (-0.268 $\pm$ 0.146), and PUFAs (-0.217 $\pm$ 0.147). We also compared the DIIs of the moderate ($n = 19$; DII = 1.24 $\pm$ 1.35) and severe ($n = 21$; DII = 1.12 $\pm$ 1.28) OSA groups, but

Table 1 Anthropometric, body composition and biochemical measurements of obese men with OSA ($N = 40$).

Variable	Mean $\pm$ SD	Variable	Mean $\pm$ SD
Body mass index (kg\m^2)	34.7 $\pm$ 3.6	LDL cholesterol (mg/dL)	117.2 $\pm$ 29.2
Waist circumference (cm)	112 $\pm$ 8.6	VLDL cholesterol (mg/dL)	13.09 $\pm$ 14.9
Fat mass (kg)	41.8 $\pm$ 10.2	Triglycerides (mg/dL)	180.5 $\pm$ 74.5
Fat Mass (%)	38.9 $\pm$ 6.05	Insulin (µIU/mL)	17.7 $\pm$ 7.7
Fat-Free Mass (kg)	64 $\pm$ 5.4	Cortisol (ug/dL)	11.96 $\pm$ 6.17
Fat-Free Mass (%)	60.95 $\pm$ 6.12	IGF-1 (ng/dL)	149.1 $\pm$ 39.32
Glucose (mg/dL)	106.7 $\pm$ 10.51	Leptin (ng/mL)	12.58 $\pm$ 7.48
Total cholesterol (mg/dL)	192.9 $\pm$ 34.26	Adiponectin (ng/mL)	11 $\pm$ 7.9
HDL cholesterol (mg/dL)	39.6 $\pm$ 6.55	Irisin (ng/mL)	3.9 $\pm$ 1.0

Abbreviations: HDL, high-density lipoprotein; IGF-1, insulin-like growth factor 1; LDL, low-density lipoprotein; VLDL, very low-density lipoprotein; OSA, obstructive sleep apnea; SD, standard deviation.

Table 2 Sleep parameters in obese individuals with OSA ($N = 40$).

Variable	Mean ± SD	Variable	Mean ± SD
Sleep latency (min)	9.37 ± 8.42	Respiratory disturbance index (n/h)	44.8 ± 23.9
REM latency (min)	106.07 ± 53.05	Apnea-hypopnea index (n/h)	42 ± 25.4
TST (min)	368.5 ± 50.01	Apneas/hour	24.25 ± 27
Sleep efficiency (%)	87.3 ± 9.05	Hypopneas/hour	17.6 ± 9.7
NREM 1 (%)	15.12 ± 10.6	O_2 Saturation (%)	94.2 ± 1.52
NREM 2 (%)	47.5 ± 9.18	Mean O_2 Saturation (%)	91 ± 9.8
NREM 3 (%)	16.72 ± 8.72	Maximum O_2 Saturation (%)	77.4 ± 8.8
REM sleep (%)	20.6 ± 6.72	Arousal index REM (n/h)	44.4 ± 24.1
WASO (min)	42.9 ± 37.2	Arousal index NREM (n/h)	41 ± 26.3
Arousals	192.32 ± 121.0	Time in O_2 saturation < 90% (%TST)	12.6 ± 16.6
Arousal index (n/h)	31.7 ± 19.4		

Abbreviations: NREM, non-rapid eye movement; REM, rapid eye movement; TST, total sleep time; OSA, obstructive sleep apnea; SD, standard deviation; WASO, wake after sleep onset.

no statistically significant differences were observed ($p = 0.791$). In the moderate OSA group, the DII ranged from -2.79 to 3.49, and, in the severe group, from -2.01 to 3.81.

The adequacy of nutrient ingestion was also investigated. According to the EARs for sex and age, a mean intake below the EAR was observed for magnesium (164.95 ± 38.4 mg), vitamin D (1.12 ± 0.07 mcg), and vitamin E (7.1 ± 2.34 mg). Regarding THE macronutrients, all of the participants presented adequate intake according to the AMDRs. The mean fiber intake was below the recommended value (20.4 ± 10.9 g).

The qualitative and semiquantitative analyses of the diet showed that the unprocessed or minimally processed food group presented one of the highest frequencies of intake during the observed days (8.23 ± 2.9 times/day). Perhaps there was a reduced frequency of some components in this category, such as vegetables and fruits, which did not reach the frequency of once a day.

There were no significant correlations between the DII and sleep parameters in the present sample (►**Fig. 1**), neither were there correlations regarding the DII and biochemical, body composition and dietary parameters. Regarding the qualitative dietary assessments, a positive correlation was found between the frequency of intake of unprocessed food and the percentage of NREM stage-3 sleep ($r = 0.336$; $p = 0.034$) and a negative correlation with the number of mixed apneas throughout the night ($r = -0.339$; $p = 0.033$). Other than that, the frequency of consumption of ultra-processed foods was positively correlated with total arousals during the night ($r = 0.330$; $p = 0.038$). Simple linear regression models confirmed the positive association between the consumption of unprocessed food and sleep quality.

Discussion

The present study sought to assess the DII in a sample of obese men with OSA and the correlations regarding the index and sleep and health parameters. We found a trend towards a proinflammatory dietary profile, which was associated with a reduced diet quality (reduced intake of fruits, vegetables, fiber, and micronutrients). These results show a poor dietary quality, which is in accordance to what is expected in obese individuals, such as those who composed the sample of the present study. We could not find any correlations involving the DII and sleep parameters.

The DII has been considered an important predictor of the inflammatory potential of diet, and it has been used in several studies since its development.[4,5] Recent research and investigators have woken up to the impact of dietary patterns on sleep parameters and the risk of developing sleep apnea, beyond the effect of weight and body composition on sleep quality and the inflammatory potential of the diet received attention.

Godos et al.[16] showed, in a large sample of Italian adults, that higher DIIs were associated with reduced chance to have better sleep quality according to Pittsburg Sleep Quality Index (PSQI; model adjusted for basal characteristics of age, sex, marital status, educational level, smoke, alcohol consumption, and physical activity). Associations regarding objective (wearable sleep monitoring) and subjective sleep measurements and the energy-density inflammatory index (E-DII) in the non-OSA population have already been demonstrated: a more proinflammatory diet is associated with increased wake-after-sleep-onset time and general sleep quality measured by the PSQI.[17] Recently, two studies found that a proinflammatory dietary pattern is associated with a higher risk of developing OSA in non-OSA subjects.[18,19]

Evidence regarding OSA and dietary inflammatory profiles is scarce. Similar to our results, Lopes et al.,[8] studying 296 OSA patients, found no association involving the DII and sleep parameters measured by polysomnography. The authors[8] found that DIIs ranging from -4.37 to 3.41 and higher DIIs (fourth quintile) were associated with diurnal somnolence. Our results showed a similar DII range, from -2.79 to 3.66, indicating a more proinflammatory profile. It is important to mention that the scores found by Lopes et al.[8] were only based on 27 items of the DII, in comparison to the

Table 3 Ranking of dietary inflammatory effects from the most to the least inflammatory component ($N = 40$).

DII component	Mean ± SD	Minimum	Maximum	95%CI	
Beta-carotene	0.354 ± 0.204	-0.260	0.552	0.289	0.420
Isoflavones	0.342 ± 0.055	0.075	0.359	0.324	0.359
Magnesium	0.332 ± 0.077	-0.069	0.415	0.307	0.356
Tea	0.312 ± 0.246	-0.536	0.391	0.233	0.391
Flavan-3-ols	0.260 ± 0.054	0.030	0.304	0.243	0.277
Flavones	0.218 ± 0.508	-0.616	0.616	0.032	0.404
Vitamin E	0.214 ± 0.246	-0.418	0.418	0.136	0.293
Total fat	0.197 ± 0.127	-0.13	0.298	0.156	0.238
Garlic	0.173 ± 0.159	-0.198	0.330	-0.198	0.330
Saturated Fat	0.135 ± 0.204	0.242	0.373	0.07	0.201
Energy	0.131 ± 0.065	-0.098	0.18	0.11	0.152
Vitamin D	0.130 ± 0.269	-0.446	0.436	0.043	0.216
Vitamin A	0.072 ± 0.140	-0.401	0.250	0.027	0.117
Vitamin C	0.067 ± 0.306	-0.423	0.411	-0.030	-0.165
Cholesterol	0.056 ± 0.059	-0.085	0.110	0.037	0.075
Anthocyanidin	0.055 ± 0.048	-0.104	0.079	0.039	0.070
Caffeine	0.043 ± 0.037	-0.022	0.082	0.031	0.055
Pepper	0.033 ± 0.060	-0.128	0.094	0.014	0.053
VitaminB12	0.031 ± 0.03	-0.022	0.082	0.021	0.041
Carbohydrates	0.027 ± 0.067	-0.088	0.097	0.005	0.048
Trans fat	0.019 ± 0.044	-0.094	0.115	0.005	0.033
Proteins	0.019 ± 0.002	0.005	0.021	0.018	0.02
Iron	0.018 ± 0.013	-0.020	0.031	0.014	0.022
Fiber	0.016 ± 0.360	-0.663	0.061	-0.132	0.099
MUFA	-0.004 ± 0.004	-0.009	0.006	-0.005	-0.002
Riboflavin	-0.016 ± 0.028	-0.061	0.064	-0.025	-0.007
Thiamin	-0.029 ± 0.038	-0.089	0.058	-0.041	-0.017
Niacin	-0.038 ± 0.055	-0.156	0.089	-0.055	-0.020
Alcohol	-0.054 ± 0.360	-0.270	0.270	-0.136	0.027
Flavanones	-0.087 ± 0.187	-0.250	0.249	-0.147	-0.028
Omega 6	-0.087 ± 0.050	-0.154	0.017	-0,103	-0,071
Onion	-0.089 ± 0.099	-0.298	-0.244	-0.121	-0.057
Folate	-0.158 ± 0.042	-0.190	-0.023	-0.172	-0.145
VitaminB6	-0.187 ± 0.106	-0.349	0.045	-0.221	-0.153
Selenium	-0.189 ± 0.005	-0.191	-0.165	-0.191	-0.187
PUFAs	-0.217 ± 0.147	-0.336	0.222	-0.264	-0.170
Zinc	-0.269 ± 0.102	-0.313	0.229	-0.302	-0.237
Flavonols	-0.268 ± 0.146	-0.467	0.210	-0.315	-0.221
Omega 3	-0.422 ± 0.015	-0.435	-0.373	-0.427	-0.417
DII	**1.109 ± 1.369**	**-2.791**	**3.66**	**0.671**	**1.547**

Abbreviations: 95%CI, 95% confidence Interval; DII, dietary inflammatory index; MUFAs, monounsaturated fatty acids; PUFAs, polyunsaturated fatty acids; SD, standard deviation.

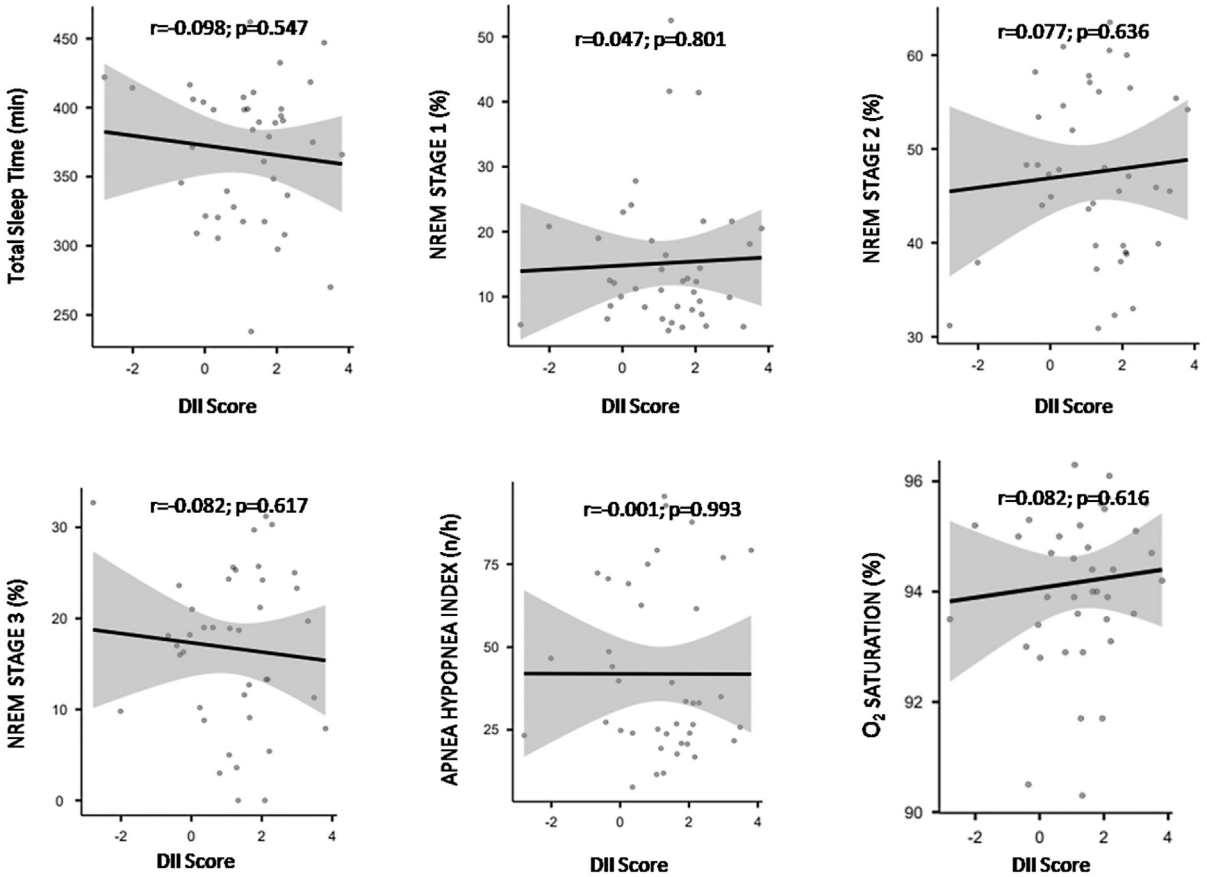

Fig. 1 Pearson correlation regarding the DII and sleep parameters. Abbreviations: DII, dietary inflammatory index; NREM, non-rapid eye movement sleep.

39 items used in the present study, possibly due to the use of a food frequency questionnaire, which limits the quantitative analysis of the diet. The reduction in items evaluated in the final DII might reduce the accuracy of the determination of the inflammatory potential of the diet.

We hypothesized that the DII would be more positive due to the low quality of the diet observed in obese people. This fact can be explained by the possible protective effect of a dietary pattern based on the consumption of unprocessed foods, regardless of the low consumption of fruits and vegetables. Thus, considering the findings of the present study, an association between unhealthy eating behaviors and poor sleep quality is visible. Poor diet quality might be an additional factor that increases the the inflammatory profile of OSA, contributing to systemic chronic inflammation.

There is evidence that diet quality is associated with worse sleep patterns. In a longitudinal study, Reid et al.[20] demonstrated that individuals with moderate and severe OSA present lower diet quality, mainly due to lower consumption of whole grains and increased consumption of red/processed meat. The results of th present study showed a positive association between the consumption of unprocessed or minimally processed foods and NREM stage-3 sleep. However, when separately analyzing the consumption of fruits and vegetables, which are components of the unprocessed food group, we found low consumption of these items. This shows that the unprocessed foods consumed were not fruits and vegetables. These foods groups are rich in important nutrients to achieve better health, such as fiber and micronutrients, which may explain the failure of the sample to reach the recommendations for these nutrients. The present study has limitations; due to the cross-sectional design, we were not able to establish causal relationships; the study is a secondary to a broader investigation,[9] so the sample calculation was not performed; the study included a small sample, composed only of male subjects, which prevents its generalizability; however, sleep was directly measured through polysomnography in all participants. Additionally, the present study used a quantitatively dietary evaluation method (three-day food record) corrected by the intraindividual variability, which makes the DII calculated reliable in terms of the food routine of the participants. Moreover, the food intake method used enabled the inclusion of 39 of the 45 components of the original DII, which makes the index strong to evaluate the inflammatory potential of the diet.

We conclude that our sample of obese individuals with moderate to severe OSA presented a proinflammatory dietary profile and poor dietary quality, which might contribute to their systemic inflammatory status. Although there was no correlation between the DII and OSA, a relationship between unprocessed food consumption and better

sleep quality was observed. The adoption of healthy eating habits might improve sleep quality in individuals with OSA and contribute to the prevention of associated diseases.

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Conflict of Interests
The authors have no conflict of interests to declare.

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