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Association between inflammatory potential of the diet and sleep parameters in sleep apnea patients

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ABSTRACT

Objectives: The aim of this study was to analyze the association between the inflammatory potential of diet and sleep parameters in individuals with obstructive sleep apnea (OSA) and to evaluate the sensitivity and specificity of the dietary inflammatory index (DII) at predicting sleep pattern.
Methods: Patients diagnosed with mild to severe OSA were included in the study (N = 296). Sleep pattern was analyzed by polysomnography and subjective sleep parameters. DII scores were calculated from a validated food frequency questionnaire. Receiver operating characteristic curve analysis and generalized linear models were conducted.
Results: DII scores were efficient at predicting apnea severity ($P < 0.05$) and daytime sleepiness ($P = 0.02$) in age stratification and predicting rapid eye movement latency in obese individuals ($P = 0.03$). No significant associations were found between DII scores and the majority of sleep parameters. The DII was only associated with daytime sleepiness; patients with a more proinflammatory diet (quintile 4) showed more subjective sleepiness than the group with a more anti-inflammatory diet (quintile 1; $P < 0.05$).
Conclusion: Findings from this study indicated that the DII could be sensitive and specific for predicting apnea severity in individuals commonly associated with OSA. Although the DII was not associated with most of the sleep parameters, the few associations found demonstrated the need for more studies that evaluate whether DII is associated with the risk for OSA symptoms.

Introduction

Obstructive sleep apnea (OSA) is a worldwide public health problem [1–3]. OSA is characterized by recurrent episodes of apneas associated with cyclic changes in oxyhemoglobin saturation, alterations in heart rate, and blood pressure during sleep [3]. The full pathogenesis of OSA is not clear; some studies suggest that in addition to mechanical factors in the airway, there may be an inflammatory etiology, once systemic inflammatory response mediators are upregulated in individuals with OSA [4,5].

Previous studies have consistently found evidence that obesity, which is a state of low-grade chronic inflammation, is a well-established leading risk factor for OSA [6,7]. In this sense, the pattern of the individual's diet could enhance the risk for OSA or worsen its

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severity by increasing the chance of developing obesity [8]. Some studies have shown that dietary patterns, mainly those with higher inflammatory potential, could have a profound influence on obesity [9,10] and chronic inflammation [11–13].

The dietary inflammatory index (DII) was developed to estimate the inflammatory potential of an individual's diets [14]. The overall score is dependent on the whole diet, not just certain nutrients or foods [13]. A high DII score indicates a proinflammatory diet, whereas a low DII score indicates that the diet is more anti-inflammatory [14]. The DII has been validated and used in diverse populations to assess the effect of dietary inflammation on the risk for developing and surviving various chronic diseases [10–15]. However, to the best of our knowledge, there is no information about the effect of a proinflammatory diet on sleep patterns and apnea severity in individuals with OSA.

Considering the body of evidence on the influence of dietary patterns on inflammation and obesity [9–13], the association of obesity and sleep apnea [6,7], and higher levels of inflammation parameters in patients with OSA [4,5], we hypothesized that a diet with a higher inflammatory potential could be associated with worse sleep patterns and apnea severity in these individuals. The main objective of this study was to analyze the association between the inflammatory potential of diet and sleep parameters in individuals with OSA and to evaluate the sensitivity and specificity of the DII in predicting sleep patterns and apnea severity.

Materials and methods

Study population

Patients with sleep complaints who were suspected to have sleep apnea were referred by their physicians to a polysomnography (PSG) examination in a private sleep clinic where this cross-sectional study was conducted. Data collection was performed from August 2016 to March 2017. During the study period, volunteers were eligible to participate if they were 18 to 60 y of age, reported no previous apnea treatments such as surgery or continuous positive airway pressure (CPAP) therapy, had no previous diagnosis of sleep disorders, and did not take medications that could affect sleep. Of the 409 patients who agreed to participate in this research, 296 were included in the study. Of the 113 excluded, 95 had an apnea-hypopnea index (AHI) <5, 13 had energy consumption >4000 kcal, 4 had an incomplete PSG, and one outlier was severely obese with a mild sleep apnea.

Initially, a questionnaire with personal information (age, alcohol consumption, smoking habit, and medication use), sociodemographic characteristics, and previous diseases was administered. Physical measurements were collected, and participants also answered questions about subjective sleep, fatigue, and dietary habits, which are described in detail later.

This study was approved by the Ethics Committee of the Federal University of Uberlandia and was conducted according to international ethical standards. An informed written consent was obtained from all volunteers before starting the study.

Physical measurements

Physical measurements were taken using standardized methods [16,17] and included neck circumference at the thyroid cartilage level, waist circumference at the level of the umbilicus, body weight (kg), height (m), and systolic and diastolic blood pressures (mm Hg). Participants were classified according to body mass index (BMI) [16].

Subjective sleep and fatigue evaluations

Subjective sleep evaluations were obtained by questionnaires that evaluated daytime sleepiness, sleep quality, and fatigue. Daytime sleepiness was measured using the Epworth Sleepiness Scale [18], sleep quality was assessed using the Pittsburgh Sleep Quality Index [19], and fatigue was assessed using the Chalder Scale [20].

Polysomnography

Objective approaches of sleep were evaluated using a full-night PSG, which was performed using a digital system (Alice Sleepware version 2.8.78, Respironics Inc., Murrysville, PA, USA) at the sleep clinic during the individual's habitual sleep time. Trained technicians visually scored all PSGs and the exam was performed according to specific criteria that define the sleep stages and apneas and hypopneas classification [21].

Individuals were classified with OSA diagnosis if they had an AHI ≥15 or ≤5 to ≤14.9 and presented at least one of the following complaints: breathing interruptions during sleep, loud snoring, daytime sleepiness, and fatigue [21].

Dietary inflammatory index

A validated semiquantitative food frequency questionnaire (FFQ) [22] was administered at recruitment to assess habitual dietary intake of the participants over the previous year. The Nutrition Data System for Research software (version 2014, University of Minnesota, Minneapolis, MN, USA) was used to quantify the energy and nutrients consumed as obtained by the FFQ. Following this, these data were adjusted for energy intake using the residual method [23].

FFQ dietary data were used to calculate DII scores for all study participants. The development and construct validation of the DII, as well as its calculation, have been described previously [15]. Dietary data for each study participant were first linked to a regionally representative global database that provided a robust estimate of means and SDs for each of the food parameters considered (i.e., foods, nutrients, and other food components) [15]. A *Z*-score was created for each food component for each participant by subtracting the standard global mean from the amount reported, and then this value was divided by the SD. To minimize the effect of right skewing, this value was converted to a proportion, which was then multiplied by 2 and one was subtracted to center the value around zero. This centered proportion score was then multiplied by the respective inflammatory effect score of the food parameters (derived from a literature review and scoring of 1943 "qualified" articles) to obtain the individual's food parameter-specific DII score. For the current FFQ, data were available for a total of 27 food parameters: energy (kcal); carbohydrates; protein; total fat; fiber; cholesterol; saturated, monounsaturated, polyunsaturated, and trans fatty; ω-6 and ω-3 fatty acids; thiamin; riboflavin; niacin; selenium; iron; magnesium; zinc; vitamins A, C, D, E, B_6, and B_{12}; alcohol; and caffeine. The food parameter–specific DII scores were then summed to create the overall DII score for each individual in the study. The greater the DII score, the more proinflammatory the diet, whereas more negative values represented more anti-inflammatory diets.

Physical activity level

The short form of the International Physical Activity Questionnaire was used to evaluate physical activity level. This questionnaire was translated into Portuguese and validated by Matsudo et al. [24].

Statistical analysis

Data are presented as the mean and interquartile range (IQR). DII values were converted to quintiles (quintile 1: −4.37 to −2.37; quintile 2: −2.36 to −1.68; quintile 3: −1.67 to −0.87; quintile 4: −0.86 to −0.23; quintile 5: −0.22 to 3.41). Data were assessed for outliers using visual analysis via scatter plot and IQR from means. Generalized linear models (GLMs) were used to assess median differences between quintiles 1 and 5. The qualitative variables were analyzed using a χ^2 test. The receiver operating characteristic (ROC) curve analysis was used to detect the efficacy of the DII at predicting sleep disorders and apnea severity by objective and subjective sleep parameters. The cutoff values used for each objective and subjective parameters to detect sleep pattern were in accordance with the literature [18–20,25]. The ROC curve analysis was stratified in different models based on the most common associated factors in sleep pattern and OSA: non-stratification, stratification for BMI, age, and sex.

To determine the effects of the DII on PSG and subjective sleep and fatigue parameters, GLMs were used. Individual tests were done for each PSG and subjective parameter (dependent variables) and quintiles of DII (independent variables) using γ, linear, or tweedie distributions for continuous variables. The best model was chosen based on the Akaike Information Criterion (AIC). To establish possible confounders associated with each PSG and subjective parameters, independent multivariate logistic regression models were performed using backward stepwise elimination ($P \leq 0.15$). In addition, collinearity diagnostics tests were done between these variables of adjustment. Individual tests also were conducted for each nutrient (dependent variables) for different quintiles of the DII (independent variables), using age, BMI, and sex as confounders. All variables used to adjust models were in table notes. Multiple comparisons were performed using sequential Sidak post hoc test. All statistical analyses were performed using SPSS version 20 (IBM, Armonk, NY, USA). For statistical significance, α error was set at 5%.

Results

Table 1 shows that patients with OSA and a more anti-inflammatory diet (quintile 1) were older ($P = 0.001$) and presented a lower neck circumference ($P = 0.02$) than the group with a more proinflammatory diet (Table 1). The majority of the participants in quintile 1 (more anti-inflammatory diet) were

Table 1
OSA participants' characteristics according to DII quintiles

	Quintile 1 (n = 59)	Quintile 2 (n = 59)	Quintile 3 (n = 60)	Quintile 4 (n = 59)	Quintile 5 (n = 59)	*P*-value*
Age (y)	45.6 (43.1–48.1)	43.1 (40.6–45.7)	40.3 (37.8–42.8)	38.5 (36–41)	38.3 (35.8–40.9)	**0.001**
Sex						
Men	45.8	61	68.3	66.1	79.7	**0.023**
Marital status						
With partner	70.5	72.2	73.2	72.9	70	0.57
Physical measurements						
BMI (kg/m^2)	30.7 (29.5–32.1)	31 (29.7–32.3)	30.9 (29.6–32.2)	31.1 (29.8–32.4)	31.5 (30.2–32.8)	0.99
Waist circumference (cm)	101.8 (98.8–104.9)	102.5 (99.4–105.6)	102.7 (99.7–105.8)	104.5 (101.4–107.7)	105.6 (102.5–108.8)	0.62
Neck circumference (cm)	38.4 (37.4–39.3)	38.8 (37.9–39.8)	39.7 (38.7–40.7)	39.2 (38.2–40.2)	40.6 (39.6–41.6)	**0.02**
SBP (mm Hg)	120.8 (117.4–124.3)	126 (122.5–129.7)	124.5 (121–128.1)	123.2 (119.7–126.7)	125.4 (121.9–129.1)	0.48
DBP (mm Hg)	76.3 (73–79.6)	76.4 (73.1–79.6)	79.7 (76.4–82.9)	77.5 (74.2–80.7)	76.5 (73.2–79.8)	0.99

BMI, body mass index; DBP, diastolic blood pressure; SBP, systolic blood pressure.
Age and physical measurements are presented by mean (interquartile range), multiple comparisons generalized linear models; other variables are presented by percent, χ^2 test. **Bold** values represent $P < 0.05$.
*For continuous variables, *P*-value represents the difference between quintiles 1 and 5; for categorical variables, *P*-value represents the association between DII quintile and dependent variables.

women and, in quintile 5 (more proinflammatory diet), men ($P = 0.02$; Table 1).

Table 2 shows that patients with sleep apnea and a more proinflammatory diet (quintile 5) had higher intakes of total fat, saturated fat, and carbohydrates than the group with a more anti-inflammatory diet (quintile 1; $P < 0.001$ for all parameters). On the other hand, the consumption of ω-3 fatty acids and fiber were significantly lower in quintile 5 than in quintile 1 ($P < 0.001$ for all parameters).

The DII was not a sensitive and specific tool for predicting sleep pattern and it did not show efficacy in predicting apnea severity (AHI) when confounder factors were not controlled (Table 3). However, when the sample population was stratified by age and BMI, the DII was efficient at predicting REM latency in patients with obesity ($P = 0.03$), AHI for adults 51 to 60 y of age ($P < 0.05$), and daytime sleepiness for those adults 18 to 30 y of age ($P = 0.02$; Table 3). Multiple comparisons showed no differences between patients with OSA in DII quintiles 1 and 5 (Table 4).

Table 2
Adjusted mean of energy and nutrient intake by DII quintiles

	Quintile 1	Quintile 2	Quintile 3	Quintile 4	Quintile 5	*P*-value 1 vs 5
Energy	2313.4 (2147.8–2491.9)	2273.4 (2112.3–2446.7)	2059.4 (1910.9–2219.4)	2131.7 (1977.8–2297.6)	2192.6 (2031.5–2366.5)	0.87
Fat	80.1 (76.8–83.3)	82.2 (79–85.3)	80.8 (77.6–83.9)	81.4 (78.2–84.6)	90.1 (86.7–93.5)	<**0.001**
Carbohydrate	303.5 (291.9–315)	298.3 (287–309.6)	306.6 (295.3–317.9)	303.4 (292–314.8)	272.3 (260.3–284.4)	**0.003**
Protein	106.1 (101.2–111.1)	106.8 (101.8–111.7)	101.3 (96.6–106)	100 (95.3–104.7)	106.7 (101.5–111.9)	0.99
Alcohol	6.8 (5.1–9)	5.4 (4.1–7.1)	4.8 (3.6–6.4)	5.6 (4.2–7.4)	6.8 (5.2–8.9)	0.99
Cholesterol	346 (315.1–383.7)	314.9 (287.3–348.4)	308.1 (281.3–340.6)	299.1 (272.8–330.9)	329 (299.3–365.1)	0.95
Saturated	22.9 (21.6–24.4)	24.1 (22.8–25.6)	24.4 (23–25.8)	24.9 (23.6–26.4)	29.2 (27.8–30.8)	<**0.001**
Monounsaturated	27.5 (26.2–28.9)	27.7 (26.3–29)	27.2 (25.9–28.6)	27.2 (25.9–28.6)	29.6 (28.2–31.1)	0.26
Polyunsaturated	16.3 (15.6–17.1)	16.3 (15.6–17.1)	15.7 (14.9–16.4)	15.5 (14.8–16.3)	15.9 (15–16.7)	0.97
Trans	3 (2.7–3.3)	3.3 (2.9–3.7)	3.1 (2.8–3.5)	3.4 (3–3.8)	3.6 (3.2–4)	0.19
ω-3 fatty acids	2.4 (2.3–2.5)	2.4 (2.3–2.5)	2.2 (2.2–2.3)	2.2 (2.1–2.3)	2.1 (2–2.2)	<**0.001**
Fiber	29.5 (28–31.2)	27.1 (25.7–28.6)	25.3 (24–26.7)	22.8 (21.6–24.1)	17.8 (16.9–18.8)	<**0.001**
Iron	12.7 (12.1–13.3)	13.6(13–14.3)	12.6 (12–13.2)	12.2 (11.6–12.7)	12.3 (11.8–12.9)	0.82
Zinc	15 (14.1–15.9)	15.3 (14.4–16.2)	15.2 (14.3–16.1)	14.6 (13.6–15.5)	15.1 (14.1–16)	0.99
Selenium	134.2 (128.1–141)	141.1 (134.7–148.2)	132.2 (126.2–138.8)	131.7 (125.7–138.3)	138.1 (131.3–145.3)	0.94

BMI, body mass index; DII, dietary inflammatory index.
Model adjusted for age, BMI, and sex. **Bold** values represent $P < 0.05$.

Table 3
ROC curve for the ability of DII to predict sleep pattern and apnea severity in stratified groups*

PSG variables	TST (min)	Sleep efficiency (%)	Sleep latency (min)	REM latency (min)	WASO (min)	N1 (%TST)	N2 (%TST)	N3 (%TST)	R sleep (%TST)	AHI	Poor sleep quality	Daytime sleepiness	Fatigue
	AUC	AUC	AUC	AUC	AUC	AUC	AUC	AUC	AUC	AUC	AUC	AUC	AUC
Non-stratification													
All	0.5 (0.4–0.6)	0.6 (0.4–0.7)	0.6 (0.4–0.8)	0.5 (0.5–0.6)	0.5 (0.5–0.6)	0.5 (0.5–0.6)	0.5 (0.4–0.6)	0.5 (0.5–0.6)	0.6 (0.5–0.7)	0.5 (0.5–0.6)	0.5 (0.5–0.6)	0.5 (0.4–0.6)	0.5 (0.5–0.6)
BMI													
Eutrophic	0.5 (0.2–0.7)	0.1 (0.0–0.2)	0.9 (0.7–1)	0.6 (0.3–0.8)	0.5 (0.3–0.7)	0.4 (0.2–0.6)	0.5 (0.3–0.8)	0.6 (0.3–0.8)	0.7 (0.5–1)	0.5 (0.3–0.8)	0.4 (0.2–0.7)	0.4 (0.2–0.6)	0.4 (0.2–0.6)
Overweight	0.5 (0.4–0.7)	0.6 (0.4–0.7)	0.6 (0.5–0.7)	0.4 (0.3–0.5)	0.5 (0.4–0.6)	0.5 (0.4–0.6)	0.4 (0.3–0.6)	0.5 (0.4–0.7)	0.5 (0.3–0.6)	0.5 (0.4–0.6)	0.6 (0.5–0.7)	0.6 (0.4–0.7)	0.6 (0.5–0.7)
Obesity	0.5 (0.4–0.6)	0.6 (0.4–0.8)	0.5 (0.2–0.7)	**0.6 (0.5–0.7)**	0.5 (0.5–0.6)	0.6 (0.5–0.7)	0.5 (0.4–0.6)	0.6 (0.5–0.6)	0.6 (0.5–0.7)	0.5 (0.4–0.6)	0.5 (0.4–0.6)	0.5 (0.4–0.6)	0.5 (0.4–0.6)
Age													
18–30 y	0.6 (0.4–0.8)	0.6 (0.4–0.9)	0.5 (0.3–0.8)	0.5 (0.4–0.7)	0.5 (0.3–0.7)	0.6 (0.4–0.8)	0.5 (0.3–0.7)	0.4 (0.2–0.6)	0.4 (0.2–0.6)	0.5 (0.4–0.7)	0.6 (0.5–0.8)	**0.7 (0.6–0.9)**	0.6 (0.4–0.8)
31–50 y	0.5 (0.4–0.6)	0.6 (0.4–0.7)	0.6 (0.3–0.9)	0.6 (0.5–0.7)	0.5 (0.5–0.6)	0.5 0.4–0.6)	0.5 (0.4–0.6)	0.5 (0.4–0.6)	0.6 (0.5–0.7)	0.5 (0.5–0.6)	0.5 (0.4–0.6)	0.5 (0.5–0.6)	0.5 (0.4–0.6)
51–60 y	0.5 (0.3–0.7)	0.4 (0.1–0.7)	0.9 (0.7–1.0)	0.5 (0.4–0.7)	0.5 (0.3–0.6)	0.6 (0.4–0.7)	0.5 (0.4–0.7)	0.6 (0.5–0.7)	0.7 (0.5–0.8)	**0.4 (0.2–0.5)**	0.5 (0.3–0.7)	0.4 (0.2–0.5)	0.4 (0.3–0.6)
Sex													
Women	0.5 (0.4–0.7)	0.6 (0.3–0.8)	0.7 (0.4–0.9)	0.5 (0.4–0.6)	0.5 (0.4–0.6)	0.5 (0.4–0.6)	0.4 (0.3–0.6)	0.6 (0.4–0.7)	0.6 (0.4–0.7)	0.6 (0.5–0.7)	0.5 (0.4–0.6)	0.5 (0.4–0.6)	0.4 (0.3–0.6)
Men	0.5 (0.4–0.6)	0.6 (0.4–0.7)	0.5 (0.3–0.8)	0.6 (0.5–0.7)	0.5 (0.5–0.6)	0.6 (0.5–0.7)	0.5 (0.4–0.6)	0.6 (0.5–0.7)	0.5 (0.4–0.7)	0.5 (0.4–0.6)	0.6 (0.5–0.6)	0.5 (0.4–0.6)	0.5 (0.4–0.6)

AHI, apnea–hypopnea index; AUC, area under the curve; DII, dietary inflammatory index; ROC, receive operating characteristic; R sleep, rapid eye movement sleep stage; TST, total sleep time; WASO, wakefulness after sleep onset.
Bold values represent $P < 0.05$.
*Data are presented as AUC values (95% VI). Cutoff values for objective sleep parameters include TST: group 1< 420 min and group 2 ≥ 420 min; sleep efficiency: group 1 <85% and group 2 ≥85%; sleep latency: group 1 ≤30 min and group 2 >30 min; R sleep latency: group 1 ≤120 min and group 2 >120 min; WASO: group 1 ≤30 min and group 2 >30 min; N1: group 1 ≤5% and group 2 >5%; N2: group 1 ≤55% and group 2 >55%; N3: group 1 <20% and group 2 ≥20%; R sleep: group 1 <20% and group 2 ≥20%; AHI: group 1 >5 and <15 and group 2 ≥15.

Table 4
Effect of DII on objective sleep parameters (PSG)*

Dependent Variables	Quintile 1	Quintile 2	Quintile 3	Quintile 4	Quintile 5	*P*-value 1 vs 5
TST (min)	436.4 (421.8–451)	435.2 (420–450.4)	440 (424.5–455.4)	444.8 (429.4–460.3)	432.9 (417.5–448.4)	0.99
Sleep efficiency (%)	90.3 (88.3–92.3)	92.4 (90.2–94.6)	90.6 (88.6–92.6)	90.9 (88.8–93)	90.1 (88.1–92.1)	0.99
Sleep latency (min)	3.8 (2.4–6)	2.8 (1.7–4.5)	3.5 (2.2–5.4)	2.2 (1.4–3.6)	2.9 (1.8–4.6)	0.91
REM latency (min)	198.5 (170.1–231.5)	189.9 (162.3–222.2)	214.4 (184.6–249)	212.7 (183.3–246.8)	221.2 (190.1–257.4)	0.89
WASO (min)	41.54 (34.7–49.7)	31.1 (25.8–37.5)	39.2 (32.6–47)	38.9 (32.2–47)	38.8 (32–47)	0.99
N1 (%TST)	12.7 (9.9–16.3)	10.3 (8–13.4)	12.8 (9.9–16.7)	10.8 (8.4–13.8)	11.5 (8.8–15)	0.96
N2 (%TST)	48.1 (45.3–51)	47 (44.3–49.9)	49.3 (46.5–52.3)	47.7 (44.9–50.6)	46.2 (43.4–49.2)	0.97
N3 (%TST)	24 (20.8–27.3)	26.9 (23.6–30.2)	23.3 (20–26.7)	25.6 (22.3–28.8)	28.5 (25.3–31.8)	0.28
R sleep (%TST)	14 (12.2–16)	14.2 (12.4–16.3)	13 (11.3–14.9)	13.8 (12–15.9)	12.5 (10.7–14.6)	0.92
AHI	25.6 (21.4–30.6)	23.9 (19.9–28.7)	26 (21.7–31.1)	22.6 (18.7–27.2)	21.9 (18.3–23.3)	0.92

AHI, apnea–hypopnea index; BMI, body mass index; DBP, diastolic blood pressure; DII, dietary inflammatory index; R sleep, rapid eye movement sleep stage; SBP, systolic blood pressure; TST, total sleep time; WASO, wakefulness after sleep onset.
Bold values represent $P < 0.05$.
*Data are presented as adjusted mean (95% CI). Model adjusted for TST: DBP, marital status, smoking habit, waist and neck circumferences, and carbohydrate intake; sleep efficiency: physical activity, smoking habit, protein intake, and SBP; sleep latency: sex, BMI, marital status, neck circumference, and AHI; R sleep latency: age, waist circumference, household income, education, SBP, and fat intake; WASO: SBP, work status, protein intake, and waist circumference; N1: sex, age, marital status, household income, work status, DBP, and protein intake; N2: AHI, sex, protein and carbohydrate intake; N3: age, sex, AHI, and smoking habits; R sleep: sex, napping, AHI, SBP, alcohol consumption, and work status; AHI: age, education, alcohol consumption, and neck circumference.

Table 5 shows that the DII was only associated with daytime sleepiness; patients with more proinflammatory diets (quintile 4) showed significantly more sleepiness than the group with an anti-inflammatory diet (quintile 1 referent; Exp [B] = 0.47, 95% confidence interval, 0.22–0.99; $P < 0.05$).

Table 5
Effects of DII quintiles on subjective sleep parameters

Dependent variables	Exp(B)	Wald χ^2	95% CI	*P*-value
Poor sleep quality				
Quintile 1	1 (Referent)	1 (Referent)	1 (Referent)	1 (Referent)
Quintile 2	1.3	0.4	0.6–2.5	0.54
Quintile 3	1	0	0.5–1.9	0.88
Quintile 4	1	0	0.5–2	0.99
Quintile 5	0.6	2	0.3–1.2	0.16
Daytime sleepiness				
Quintile 1	1 (Referent)	1 (Referent)	1 (Referent)	1 (Referent)
Quintile 2	0.6	2.5	0.3–1.2	0.11
Quintile 3	0.9	0.1	0.4–1.9	0.78
Quintile 4	0.5	3.9	0.2–1	**<0.05**
Quintile 5	1.4	0.9	0.7–3.1	0.36
Fatigue				
Quintile 1	1 (Referent)	1 (Referent)	1 (Referent)	1 (Referent)
Quintile 2	0.6	1.9	0.2–1.3	0.17
Quintile 3	0.5	2.6	0.2–1.2	0.11
Quintile 4	1.5	0.8	0.6–3.7	0.36
Quintile 5	1	0	0.4–2.3	0.97

BMI, body mass index; DII, Dietary Inflammatory Index.
Bold values represent $P < 0.05$.
Model adjusted for poor sleep quality: BMI, waist and neck circumferences, physical activity; Ddaytime sleepiness: diastolic blood pressure, alcohol consumption, energy intake, and household income; fatigue: BMI, sex, physical activity, shiftwork, and neck circumference.

Discussion

To the best of our knowledge, this is the first time that the associations between DII and sleep and apnea severity by PSG and subjective sleep parameters have been tested. The DII showed sensitivity and specificity at predicting a poor sleep pattern and apnea severity when the sample was stratified by BMI and age. Even with a limited clinical significance, we observed an association between daytime sleepiness and the inflammatory potential of diet.

We hypothesized that food pattern, mainly its inflammatory potential, could be a predictor of OSA severity and a poor sleep pattern in our sample, once diet was correlated with obesity, which is a state of low-grade chronic inflammation, and could partly mediate the association between dietary inflammatory potential and OSA. Although the DII could predict AHI in older participants (Table 3), a group with higher incidence of OSA [26], we could not find an association between the inflammatory potential of diet and apnea severity (Table 4). This might have occurred because of the inclusion of adjustment variables in the GLMs other than the stratified variables used to produce the ROC curve. The low sensitivity and specify of the DII at predicting the remaining sleep parameters in the other stratified models is worth noting. A possible explanation for these results is that food pattern may affect OSA indirectly (i.e., through its effect on excess weight, which in turn affects OSA). This could explain why we only observed significant differences in neck circumference, which is a risk factor for OSA [26].

The inflammatory potential of diet was associated with daytime sleepiness (Table 5), particularly in young adults (Table 3). Cao et al. [8] found that a fatty meal was positively associated with daytime sleepiness in patients with apnea. In some ways, the present results corroborated Cao et al.'s data in that total and saturated fat could have been responsible for the association between daytime sleepiness and the inflammatory potential of the diet, as found in the present study (Table 5). Individuals with a more proinflammatory diet presented higher consumption of these nutrients than did

individuals with a more anti-inflammatory diet (Table 2). Other studies have shown that a fatty meal was positively associated with AHI [27], saturated fat and carbohydrates were positively associated with faster sleep onset [28,29], higher amounts of protein could increase difficulty in maintaining sleep and decrease difficulty in initiating sleep [30], and a decrease in the ingestion of fiber could increase the number of arousals [28]. In the present study, only daytime sleepiness showed significant differences according to DII quintiles. Unlike the present study, these other studies of single nutrients and foods [27–30] did not account for complex interactions inherent in whole diets [30–33] because the dietary interactions may confound these associations [34]. Foods or nutrients are usually not eaten in isolation, so their effects will tend to amplify or attenuate their individual effects [15]. Furthermore, the high correlation among nutrients and across foods may interfere in the risk estimation owing to multicollinearity and the possible loss of statistical power [15]. In this way, the evaluation of the effects of a whole diet or dietary patterns, which were assessed in this study by the DII, interfere in the risk for the prevalence or worsening of diseases could be more effective than the evaluation of a single nutrient because foods and nutrients act together [15].

The major strength of the present study is that we evaluated sleep parameters by PSG and used a validated and reproducible FFQ, which allowed for a comprehensive assessment of major nutrient sources in the diet. The study had certain limitations. Although we adjusted for several potential confounding variables, we cannot completely rule out that this study was confounded by unmeasured variables such as inflammatory biomarkers; on the other hand, we adjusted our analysis for the main factors related to sleep and apnea, which are BMI, neck circumference, sex, and age [26]. The sample size of this study was relatively small in comparison with other population cohorts, but nonetheless it had sufficient power to detect significant associations. However, this could be insufficient to provide a robust estimate of the association between the DII and OSA severity and objective sleep parameters. Another limitation of the study is its cross-sectional design, which did not allow determination of cause and effect.

Findings from this study demonstrated that DII scores could be sensitive and specific enough to reflect on a few alterations in sleep pattern in some stratified models. The use of a relatively inexpensive but strictly developed self-reporting measure, such as a validated FFQ, is the only feasible option [35] to evaluate the effects of dietary patterns on some diseases, mainly ones that have expensive diagnosis tests such as PSG to detect OSA. Moreover, although we did not find associations between the inflammatory potential of diet and most sleep parameters, diets with more inflammatory potential could increase the risk for the symptoms of OSA such as daytime sleepiness. Further studies are required to confirm the results presented in this study and to provide causality in the relationship between a proinflammatory diet and sleep parameters and the possible mechanisms involved in this association.

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