

# Plant-based and vegetarian diets are associated with reduced obstructive sleep apnoea risk

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# Shareable abstract (@ERSpublications)

While a healthy plant-based dietary index (PDI) is linked to a reduced risk of obstructive sleep apnoea, an unhealthy PDI increases the risk. These associations vary between males and females. Further longitudinal studies are warranted. https://bit.ly/48LoeVk

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# Abstract

*Introduction* Obstructive sleep apnoea (OSA) and obesity commonly coexist. Weight loss and exercise are recommended management options for OSA. However, most of the current evidence on diet and OSA is focused on calorie restriction rather than diet quality. The aim of the present study was to determine the association of plant-based dietary indices (PDI) with OSA risk.

*Methods* Cross-sectional data from 14210 participants of the National Health and Nutrition Examination Survey who provided dietary information using the 24-hour recall method were used. PDI – including healthy (hPDI), unhealthy (uPDI) and pro-vegetarian diet index (PVDI) – were determined. OSA risk was determined using the STOP-BANG questionnaire. Logistic regression was used to determine the relationship between dietary indices and OSA risk.

*Results* Higher adherence to PDI (odds ratio (OR)<sub>Q5</sub> versus Q1</sub>=0.81; 95% confidence interval (CI): 0.66–1.00), hPDI (OR=0.83; 95% CI: 0.69–1.01) and PVDI (OR=0.84; 95% CI: 0.68–1.05) was inversely associated with OSA risk, whereas higher consumption of an unhealthy plant-based diet (OR=1.22; 95% CI: 1.00–1.49) was positively associated with OSA. Sex differences in estimates were observed for PDI in males (OR=0.71; 95% CI: 0.56–0.90) versus females (OR=0.93; 95% CI: 0.68–1.28), hPDI in males (OR=0.90; 95% CI: 0.68–1.18) versus females (OR=0.77; 95% CI: 0.54–1.09) and uPDI in males (OR=1.13; 95% CI: 0.89–1.44) versus females (OR=1.42; 95% CI: 1.03–1.97) but not for PVDI.

*Conclusions* Higher adherence to a healthy plant-based diet is associated with reduced OSA risk, while an unhealthy plant-based diet has a positive association. The magnitude of these associations differs by sex. Further longitudinal studies are warranted.

# Introduction

Almost one billion people worldwide are estimated to have obstructive sleep apnoea (OSA) [1], a condition that triples the risk of cardiometabolic diseases [2], such as cardiovascular disease (CVD) [3], diabetes [4] and mortality [5]. The primary predictor of OSA is obesity, especially central obesity [6]. Effective treatment to reduce OSA severity includes lifestyle modifications, such as weight loss through exercise and diet [7, 8].

Most dietary intervention studies focused on OSA have emphasised weight reduction *via* caloric restriction and specific dietary elements [8–10], rather than holistic dietary patterns. In addition, a considerable portion of research in this domain is derived from secondary evaluations of randomised clinical trials. For instance, the Positive Pressure Long-term Efficacy Study (APPLES study) identified associations between certain dietary constituents, such as protein and total fat, and OSA [8, 11]. Another longitudinal investigation involving OSA patients showed links between diet inflammatory properties and sleep metrics [12]. Although these studies have focused on the influence of specific dietary elements, dietary patterns based on plant-based diets may have a potential effect on OSA risk *via* different mechanisms, including reduced inflammation and adiposity [13–15]. Thus, considering overall adherence to a plant-based diet and investigating its association with OSA risk is crucial to design appropriate control and prevention strategies.

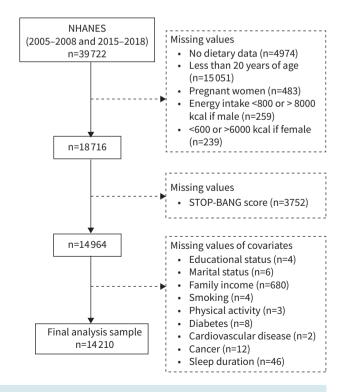
Despite considerable variation among studies, healthy plant-based diets, defined by a low frequency of animal food consumption, have been shown to reduce the risk of developing CVD [16], obesity [14, 17] and type 2 diabetes [18]. High-quality nutrients in plant-based diets is one of the factors for reduced risk of these metabolic conditions [19] through weight loss [17], enhanced glycaemic control [17], better blood pressure regulation [20], improved lipid profiles, diminished low-grade inflammation [21] and a healthier gut microbiota [22]. However, certain plant-based diets characterised by refined grains, high sugar and salt content are associated with adverse outcomes and as such, may be considered "unhealthy" [23].

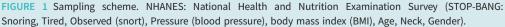
In a randomised clinical trial of 89 overweight or obese men with moderate to severe OSA, an 8-week interdisciplinary weight loss and lifestyle intervention (including healthy dietary intervention) reduced OSA severity [24]. Similarly, a clinical trial found a clinically meaningful decrease of ~4 points on the Epworth Sleepiness Scale after switching from a standard Western diet to a whole-food plant-based diet for 21 days in 14 OSA patients [25]. However, no population-based studies have examined the association of plant-based and vegetarian diets with OSA risk. In this study, we determined a plant-based dietary index (PDI), healthy PDI (hPDI), unhealthy PDI (uPDI) and a pro-vegetarian diet index (PVDI) [18, 26, 27] to investigate their associations with OSA risk.

# **Methods**

# Study design and population

Data from the National Health and Nutrition Survey (NHANES) were used. NHANES is an ongoing study that gathers nationally representative data in the United States of America (USA). The data can be accessed through the National Center for Health Statistics website, where users can download the datasets [28]. Data were obtained through interviews, medical assessments and laboratory investigations. For this study, we utilised data from four cycles of the NHANES study (2005–2008 and 2015–2018), which included a total of 39 722 participants. Data from 14 210 participants were used for analysis (figure 1). The National





Center for Health Statistics Ethics Review Board granted ethical clearance for the NHANES study [29]. Additional ethical approval for this specific study was obtained from the Flinders University Human Research Ethics Committee (6547).

# Assessment of dietary intake

Dietary data were collected in each cycle of NHANES using a 24-hour recall method. A more detailed description of the methods can be found elsewhere [30]. Briefly, the US Department of Agriculture's (USDA) Automated Multiple-Pass Method was used to collect dietary data over 2 days (first face-to-face and second *via* telephone) for the 2005–2007 and 2015–2018 cycles. For this study, we only used dietary data collected during the face-to-face interview on the first day. The micro- and macronutrient contents of the food were determined using the USDA Food and Nutrient Database for Dietary Studies (FNDDS) [30]. Additionally, the participants' data were linked to the USDA's Food Patterns Equivalents Database, which disaggregates foods and beverages into 37 USDA Food Patterns components [31].

## PDI and PVDI

We calculated four plant-based diet indices (PDI, hPDI, uPDI and PVDI). A detailed description of the calculation of each diet index has been provided in previous studies [18, 26, 27]. Briefly, 18 food groups for the PDI, hPDI and uPDI and 12 food groups for PVDI (supplementary table S1) were used. These food groups were categorised as healthy plant foods (whole grains, fruits, vegetables, nuts, legumes, tea and coffee), less healthy plant foods (refined grains, potatoes, sugar-sweetened beverages, sweets and desserts, salty foods) and animal foods (animal fat, dairy, eggs, fish or seafood, meat, miscellaneous animal foods) for the PDI, hPDI and uPDI. Plant foods (grains, fruits, vegetables, nuts, legumes, potatoes) and animal foods (animal fat, dairy, eggs, fish or seafood, meat) were used for PVDI.

The 18 food groups were divided into deciles of consumption, and each decile was assigned a score between 1 and 10. For PDI, participants received a score of 10 for each plant food group for which they were above the highest decile of consumption, a score of 9 for which they were above the second highest decile but below the highest decile, and so on, with a score of 1 for consumption below the lowest decile (positive scores). On the other hand, participants received a score of 1 for each animal food group for which they were above the highest decile of consumption and so on (reverse scores). For hPDI, positive scores were given to healthy plant food groups, and reverse scores to less healthy plant food groups and animal food groups. For PVDI, we used the same approach as hPDI. For uPDI, positive scores were given to less healthy plant food groups, and reverse scores to healthy plant food groups. The 18 food group scores for an individual were summed to obtain the indices, with a possible score range of 18 (lowest possible score) to 180 (highest possible score). The indices were analysed as quintiles, with energy intake adjusted in our models.

# Sleep apnoea risk

The STOP-BANG tool (Snoring, Tired, Observed (Snort), Pressure (blood pressure), Body mass index (BMI), Age, Neck, Gender) was used to assess the risk of sleep apnoea [32]. Supplementary table S2 provides details on the assessment methods, the scoring criteria and frequency. More detailed explanation of how questions from NHANES were used, scores were constructed, and review of sensitivity and specificity of the STOP-BANG tool can be found in our previous work [15, 33]. As there were no data on neck circumference, waist circumference was used as a substitute. Neck and waist circumference are highly and significantly correlated (r=0.64) [34]. The STOP-BANG score ranges from 0 to 8, with scores of 2 or lower considered low risk, scores of 3 to 4 considered intermediate risk, and scores of 5 or higher considered high risk. In addition, a score of 2 or higher AND meeting one of the following criteria – male sex, BMI >35, or waist circumference >102 cm for males or >88 cm for females – was considered high risk [32]. High *versus* low/intermediate OSA risk category was used in the main analysis. Sensitivity analysis based on OSA50 and STOP was described in the supplementary method.

# **Covariates**

Various sociodemographic characteristics (age, sex, race, marital status, income), behavioural factors (smoking, physical activity, sleep duration and alcohol consumption) and chronic conditions (CVD, cancer and diabetes) were taken into account in our analyses. Supplementary table S3 provides details on the assessment methods and units used for each variable.

# Statistical analysis

Our analyses accounted for the complex survey design using NHANES-assigned weights, population sampling units and strata. To summarise categorical and continuous variables, we used frequency, mean±sD, and median (interquartile range). We used generalised linear regression with binomial family and

link function to determine the association of dietary indices with OSA. Four models were developed: model 1 adjusted for sociodemographic characteristics (sex, age, race, marital status, education and income) and energy intake; model 2 additionally adjusted for smoking, physical activity and alcohol consumption; model 3 additionally adjusted for CVD, cancer and diabetes; and model 4 additionally adjusted for sleep duration. As some of the covariates could mediate rather than confound the association of diet and OSA, the model-based approach provided more insight into how these variables affected the estimates with and without their inclusion. We also used restricted cubic splines (3 knot) to determine the association of PDI, hPDI, uPDI and PVDI (as continuous variables) with OSA. We determined the significance for nonlinearity using the likelihood ratio test. All analyses were performed using STATA 16.0 and R (R Foundation for Statistical Computing; Vienna, Austria). We conducted several sensitivity analyses to further explore the association between diet and OSA risk (supplementary method).

# Results

# Participant characteristics

Half (49.4%) of participants were male. Insufficient physical activity was reported by 32.8% of the participants, while 20.5% were current smokers. The prevalence of snoring was 28.5%. Based on the STOP-BANG score, 50.5% of participants had intermediate to severe risk of sleep apnoea, while 25.1% were classified as high risk (table 1). The mean±sD of the dietary constructs are provided in table 1. The distribution of dietary indices is presented in supplementary figure S1.

# Association of PDI and PVDI with sleep apnoea risk

Participants in the fifth (highest) quintile of PDI had 19% lower odds (OR=0.81; 95% CI: 0.66–1.00) of sleep apnoea risk compared to those in the first quintile (p-value for trend=0.008). Participants in the fifth quintile of hPDI had 17% lower odds (OR=0.83; 95% CI: 0.69–1.01) of sleep apnoea risk, while those in the fifth quintile of uPDI had 22% higher odds (OR=1.22; 95% CI: 1.00–1.49) of sleep apnoea risk, compared to those in the first quintile. We also found that higher adherence to the pro-vegetarian diet (higher PVDI) was associated with a lower risk of OSA (table 2).

Nonlinearity test on the association between the dietary constructs and OSA risk was not significant (figure 2).

# Association of PDI and PVDI with sleep apnoea risk by sex

Differences in estimates were observed for PDI (OR=0.71; 95% CI: 0.56–0.90 in males *versus* OR=0.93; 95% CI: 0.68–1.28 in females), hPDI (OR=0.90; 95% CI: 0.68–1.18 in males *versus* OR=0.77; 95% CI: 0.54–1.09 in females) and uPDI (OR=1.13; 95% CI: 0.89–1.44 in males *versus* OR=1.42; 95% CI: 1.03–1.97 in females) but not for PVDI (table 3).

Non-linear tests on the associations between the dietary constructs and OSA risk with respect to sex were not significant (figure 3).

# Sensitivity analyses

The results reveal significant inverse associations of PDI, hPDI and PVDI with both STOP and STOP-BANG scores (as count), while positive associations were found with uPDI (supplementary tables S4 and S5). The use of intermediate and high-risk STOP-BANG scores to indicate OSA risk did not alter the findings (supplementary table S6). However, when self-reported doctor-diagnosed OSA was used (supplementary table S7), the associations of different dietary indices with OSA (n=6998) had wider confidence intervals. With the exception of reported snort, the other components of STOP-BANG (snore, tiredness, hypertension, BMI and waist circumference) were found to have inverse associations with PDI, hPDI and PVDI. Conversely, uPDI showed positive associations with STOP-BANG components (supplementary table S8). The results of the associations between dietary indices and sleep apnoea remained consistent when the OSA50 tool was used to determine apnoea risk (supplementary table S9).

# Discussion

Our findings demonstrate the role of plant-based dietary indices in influencing OSA risk. Specifically, participants with the highest adherence to a general and healthy plant-based diet (PDI and hPDI, respectively) have markedly reduced odds of OSA risk compared to their counterparts with the lowest adherence. Conversely, high adherence to uPDI is associated with increased odds of OSA risk. The association with PVDI further supports the potential protective role of certain vegetarian-leaning dietary patterns. A sex difference is observed regarding hPDI and uPDI; females exhibit decreased odds of OSA risk associated with higher adherence to hPDI, whereas they show increased odds of OSA risk with uPDI, a trend not observed in males. Overall PDI is primarily associated with reduced odds of OSA risk in males, not females. This suggests potential sex-specific mechanisms or susceptibilities that warrant further exploration.

Characteristics	Proportion (%
Male	49.4
Education	13.1
Less than high school	13.5
School diploma (including GED)	24.7
More than high school	61.8
Income	
Under US\$200 000	15.3
US\$20,000–34 999	18.5
U\$\$35,000–54 999	17.8
U\$\$55,000–74 999	12.5
US\$75 000 and above	35.9
Marital status	
Married/living with partner	66.8
Widowed	4.5
Divorced	9.5
Separated	2.3
Never married	16.9
Race	
Mexican American	8.1
Other Hispanic	4.9
Non-Hispanic White	69.2
Non-Hispanic Black	10.3
Physical activity level	10.0
Insufficient	32.8
Adequate	11.3
More than adequate	56.0
Smoking	50.0
Never	54.4
Ex-smoker	25.1
Smoker	20.5
Diabetes	12.6
Cancer	9.7
CVD	7.7
High blood pressure	32.6
Obese	16.1
Snored	28.5
Snort	5.6
Tired	30.5
High waist circumference	56.0
Self-reported doctor-diagnosed sleep apnoea (n=6998)	4.9
STOP score	ч.5
0	37.0
1	36.9
2	19.2
3	5.6
4	5.6
4 STOP-BANG score	1.2
	7.3
0 1	21.4
2 3	21.8 20.6
4	16.2
5	8.3
6	3.3
7	0.8
8	0.2
Sleep apnoea risk (STOP-BANG)	
Low	50.5
Intermediate	24.4
High	25.1

Continued

TABLE 1 Continued	
Characteristics	Proportion (%)
Sleep apnoea risk (OSA50) %	
Low risk	57.8
High risk	42.2
PDI <sup>#</sup>	94.5±14.5
uPDI <sup>#</sup>	108.9±17.3
hPDI <sup>#</sup>	105.7±16.2
PVDI <sup>#</sup>	63.7±11.4

CVD: cardiovascular disease; GED: general educational development; STOP-BANG: Snoring, Tired, Observed (snort), Pressure (blood pressure), Body mass index (BMI), Age, Neck, Gender. PDI: plant-based dietary index; hPDI: uPDI: unhealthy PDI; healthy PDI; PVDI: pro-vegetarian dietary index. <sup>#</sup>: dietary indices and corresponding mean±sp values.

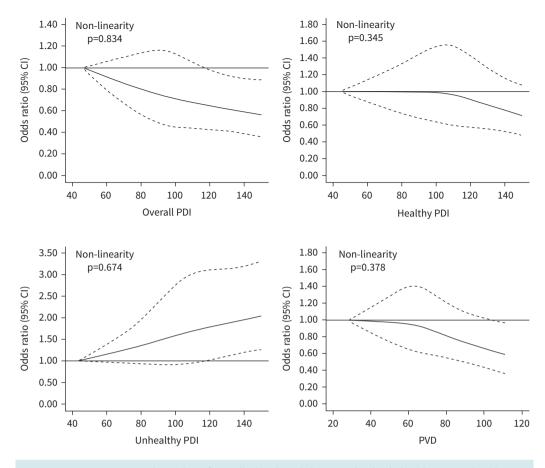
# Limitation of the study

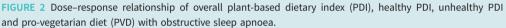
There are limitations that should be considered when interpreting our results. The cross-sectional nature of the study prevents us from inferring causality between dietary indices and OSA risk. Moreover, the reliance on 24-hour recall data for dietary information may not be indicative of typical consumption patterns and might introduce recall biases. Although the STOP-BANG criteria employed to assess OSA is not the standard diagnostic tool, it has been well validated and widely accepted as a risk tool, and has high sensitivity [32, 35]. We also used OSA-50 as a sensitivity analysis to check the robustness of findings. Additionally, we used the individual components of STOP-BANG to determine associations with PBD.

Models		p-value for trend				
	Q1	Q2	Q3	Q4	Q5	
Overall PDI						
Model 1	1.00	1.02 (0.84–1.24)	0.90 (0.73-1.13)	0.80 (0.64–0.99)	0.77 (0.63–0.93)	< 0.001
Model 2	1.00	1.02 (0.84–1.24)	0.92 (0.74–1.15)	0.82 (0.66–1.02)	0.80 (0.65–0.98)	0.003
Model 3	1.00	1.01 (0.83-1.23)	0.91 (0.73–1.14)	0.82 (0.66-1.02)	0.81 (0.65–0.99)	0.006
Model 4	1.00	1.01 (0.83-1.22)	0.92 (0.73–1.15)	0.83 (0.66–1.03)	0.81 (0.66-1.00)	0.008
Healthy PDI						
Model 1	1.00	1.01 (0.84-1.20)	0.85 (0.72-1.02)	0.93 (0.77–1.13)	0.77 (0.63–0.94)	0.007
Model 2	1.00	1.00 (0.84–1.20)	0.86 (0.72–1.02)	0.95 (0.78–1.15)	0.80 (0.65–0.97)	0.018
Model 3	1.00	1.02 (0.85–1.22)	0.87 (0.72–1.04)	0.95 (0.77-1.16)	0.81 (0.67-0.99)	0.023
Model 4	1.00	1.03 (0.86–1.23)	0.87 (0.73–1.05)	0.96 (0.79–1.18)	0.83 (0.69–1.01)	0.042
Unhealthy P	DI					
Model 1	1.00	1.17 (0.97–1.41)	1.28 (1.05–1.55)	1.42 (1.13–1.77)	1.36 (1.11–1.66)	0.001
Model 2	1.00	1.14 (0.94–1.38)	1.21 (1.00–1.48)	1.34 (1.06–1.69)	1.27 (1.03–1.55)	0.010
Model 3	1.00	1.13 (0.93–1.37)	1.24 (1.01–1.52)	1.33 (1.05–1.69)	1.25 (1.02–1.53)	0.010
Model 4	1.00	1.11 (0.92–1.34)	1.21 (0.99–1.48)	1.32 (1.04–1.67)	1.22 (1.00-1.49)	0.016
Pro-vegetaria	an diet					
Model 1	1.00	0.91 (0.73–1.13)	0.89 (0.74–1.07)	0.82 (0.67–1.00)	0.76 (0.61–0.94)	0.005
Model 2	1.00	0.93 (0.75–1.16)	0.92 (0.76–1.11)	0.87 (0.71–1.06)	0.82 (0.66–1.01)	0.043
Model 3	1.00	0.94 (0.76-1.18)	0.92 (0.76–1.13)	0.88 (0.71–1.08)	0.83 (0.66–1.03)	0.069
Model 4	1.00	0.95 (0.76-1.18)	0.93 (0.76-1.13)	0.89 (0.72-1.11)	0.84 (0.68-1.05)	0.099

Model 1 was adjusted for sex, age, race, marital status, education, income and energy intake. Model 2 was additionally adjusted for smoking, physical activity and alcohol consumption. Model 3 was additionally adjusted for cardiovascular disease, cancer and diabetes. Model 4 was additionally adjusted for sleep duration. Q5 indicates higher adherence (higher consumption) to specific dietary construct and *vice versa* for Q1. PDI: plant-based dietary index. <sup>#</sup>: sleep apnoea was based on STOP-BANG (Snoring, Tired, Observed (snort), Pressure (blood pressure), Body mass index (BMI), Age, Neck, Gender) tool. Only high-risk OSA cases were included (overall prevalence 27.5%).

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## Comparison with other studies

Although there are a handful of studies that have evaluated associations between overall diet quality and OSA [12, 15, 36–40], there are no previous studies that have examined the association between plant-based diet and OSA risk. However, studies have shown that plant-based diets are associated with lower risk of obesity [14, 25, 41], a major cause of OSA. Other dietary patterns based on overall dietary behaviour have also been associated with OSA risk. For instance, a Mexican survey showed that people with high adherence to "industrialised dietary pattern" (high in sugar-sweetened beverages, fast foods, and alcohol, coffee or tea) were more likely to experience daytime symptoms and OSA compared with the traditional pattern (high in legumes and tortilla) [40]. A pre-post pilot clinical trial found a reduced daytime sleepiness after switching from a standard Western diet to a whole-food plant-based diet only for 3 weeks in patients with OSA [25]. In three prospective US cohorts [37], while higher diet quality (higher Alternate Healthy Eating Index scores) was associated with lower risk of OSA, higher dietary inflammatory potential was associated with increased risk of OSA.

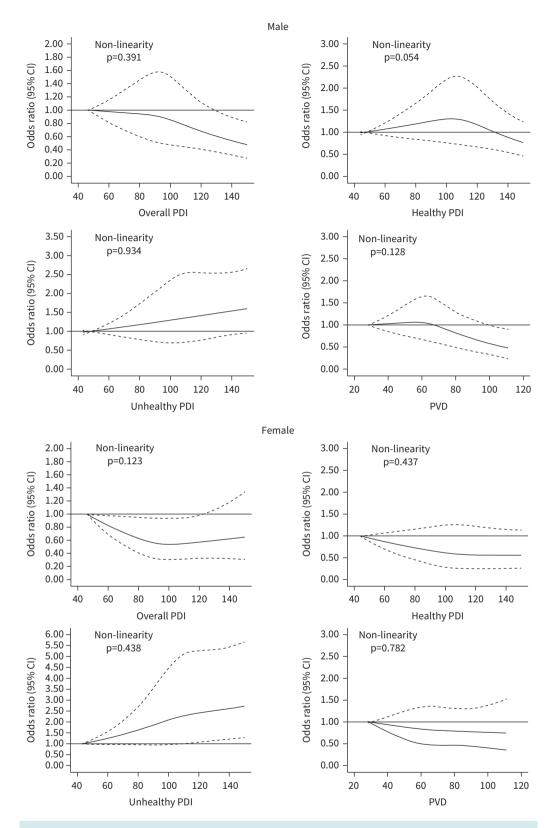
Previous studies also showed the distribution of macronutrients intake associated with some of the individual components of the STOP-BANG questionnaire. For instance, in an iso-caloric substitution analysis, substitution of fat and carbohydrate with protein was inversely associated with excessive daytime sleepiness (EDS), and substitution of protein with fat and carbohydrate was positively associated with EDS [36]. BOVE *et al.* [38] also found that a diet high in fat had twice the severity of OSA in overweight patients. A cross-sectional analysis of the APPLES study showed that participants with severe sleep disordered breathing, indicated by the respiratory disturbance index, consumed a diet that was higher in cholesterol, protein, total fat and total saturated fatty acids [11].

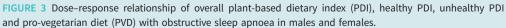
Our findings reveal sex-based differences in the association between PDI and OSA risk. Consistent with the current study, in our previous findings, we observed a sex difference in OSA risk based on a healthy lifestyle (diet, exercise, smoking, alcohol consumption and BMI), which demonstrated a stronger

Models Q1			Odds ratio (9	5% CI)		p-value for trend
	Q1	Q2	Q3	Q4	Q5	
Male						
Overall PD	I					
Model 1	1.00	0.90 (0.69-1.17)	0.95 (0.72-1.27)	0.84 (0.64-1.11)	0.66 (0.53-0.83)	0.0010
Model 2	1.00	0.91 (0.70–1.19)	0.98 (0.73–1.31)	0.87 (0.66–1.15)	0.70 (0.55–0.89)	0.0080
Model 3	1.00	0.91 (0.70-1.19)	0.95 (0.71-1.28)	0.87 (0.66-1.15)	0.70 (0.55–0.89)	0.0080
Model 4	1.00	0.91 (0.70–1.19)	0.95 (0.71–1.28)	0.87 (0.65–1.15)	0.71 (0.56–0.90)	0.0100
Healthy PI	DI					
Model 1	1.00	1.08 (0.82-1.44)	0.99 (0.81-1.21)	0.89 (0.68-1.16)	0.87 (0.66-1.14)	0.096
Model 2	1.00	1.08 (0.82-1.44)	0.99 (0.81-1.22)	0.88 (0.67-1.15)	0.87 (0.67-1.14)	0.090
Model 3	1.00	1.10 (0.82-1.46)	1.01 (0.82-1.24)	0.88 (0.68-1.15)	0.87 (0.66-1.15)	0.096
Model 4	1.00	1.10 (0.83–1.46)	1.02 (0.83-1.25)	0.89 (0.69-1.16)	0.90 (0.68-1.18)	0.144
Unhealthy	PDI					
Model 1	1.00	1.23 (0.97–1.56)	1.24 (0.94–1.64)	1.22 (0.92-1.60)	1.23 (0.96–1.57)	0.127
Model 2	1.00	1.20 (0.94-1.52)	1.19 (0.91–1.57)	1.16 (0.86-1.55)	1.17 (0.91-1.49)	0.284
Model 3	1.00	1.18 (0.93-1.51)	1.23 (0.93–1.64)	1.17 (0.86–1.58)	1.17 (0.92–1.49)	0.247
Model 4	1.00	1.15 (0.91–1.46)	1.21 (0.91-1.60)	1.15 (0.85–1.56)	1.13 (0.89–1.44)	0.317
Pro-vegeta	rian di	. ,	· · · · ·	· · · · ·	· · · ·	
Model 1	1.00	0.81 (0.62-1.07)	0.93 (0.73–1.18)	0.85 (0.65-1.11)	0.68 (0.52–0.89)	0.017
Model 2	1.00	0.83 (0.62–1.11)	0.96 (0.75–1.23)	0.89 (0.68–1.17)	0.73 (0.55–0.96)	0.075
Model 3	1.00	0.83 (0.62-1.11)	0.95 (0.75–1.22)	0.89 (0.68–1.18)	0.72 (0.54–0.96)	0.077
Model 4	1.00	0.84 (0.63–1.13)	0.96 (0.74–1.22)	0.92 (0.70-1.22)	0.74 (0.56–0.99)	0.125
Female			· · · · · ·			
Overall PD	1					
Model 1	1.00	1.07 (0.82-1.40)	0.81 (0.62-1.06)	0.69 (0.51-0.94)	0.85 (0.62-1.16)	0.031
Model 2	1.00	1.08 (0.83-1.39)	0.82 (0.64–1.07)	0.72 (0.54–0.97)	0.88 (0.65-1.19)	0.057
Model 3	1.00	1.08 (0.83-1.41)	0.86 (0.67–1.12)	0.76 (0.57-1.01)	0.93 (0.68–1.28)	0.163
Model 4	1.00	1.08 (0.83-1.41)	0.87 (0.67-1.13)	0.76 (0.57-1.02)	0.93 (0.68–1.28)	0.169
Healthy PI						
Model 1	1.00	0.86 (0.64–1.14)	0.89 (0.66–1.20)	0.85 (0.62-1.16)	0.67 (0.48–0.95)	0.022
Model 2	1.00	0.86 (0.65–1.15)	0.91 (0.67–1.23)	0.88 (0.64–1.21)	0.73 (0.52–1.03)	0.077
Model 3	1.00	0.86 (0.64–1.15)	0.91 (0.66–1.25)	0.88 (0.64–1.21)	0.76 (0.53–1.08)	0.117
Model 4	1.00	0.86 (0.64–1.15)	0.91 (0.66–1.25)	0.89 (0.65–1.22)	0.77 (0.54–1.09)	0.145
Unhealthy		(	(		(	
Model 1	1.00	1.29 (0.99–1.67)	1.53 (1.14–2.06)	1.70 (1.30-2.23)	1.63 (1.18–2.24)	< 0.001
Model 2	1.00	1.24 (0.95–1.62)	1.45 (1.07–1.95)	1.57 (1.19–2.08)	1.48 (1.07–2.06)	0.005
Model 3	1.00	1.26 (0.96–1.66)	1.42 (1.05–1.91)	1.53 (1.16–2.01)	1.44 (1.04–1.99)	0.009
Model 4	1.00	1.25 (0.96–1.65)	1.40 (1.05–1.88)	1.52 (1.15–2.00)	1.42 (1.03–1.97)	0.010
Pro-vegeta				,	,	
Model 1	1.00	0.79 (0.64–0.98)	0.73 (0.56–0.94)	0.74 (0.63–0.88)	0.69 (0.53–0.89)	0.008
Model 2	1.00	0.79 (0.64–0.98)	0.73 (0.57–0.95)	0.76 (0.65–0.91)	0.74 (0.57–0.97)	0.041
Model 3	1.00	0.80 (0.64–0.99)	0.74 (0.57–0.97)	0.78 (0.65–0.92)	0.73 (0.55–0.96)	0.038
Model 4	1.00	0.81 (0.65–1.01)	0.75 (0.57–0.99)	0.80 (0.67–0.95)	0.75 (0.56–1.00)	0.068

Model 1 was adjusted for age, race, marital status, education, income and energy intake. Model 2 was additionally adjusted for smoking, physical activity and alcohol consumption. Model 3 was additionally adjusted for cardiovascular disease, cancer and diabetes. Model 4 was additionally adjusted for sleep duration (hours). Q5 indicates higher adherence (higher consumption) to specific dietary construct and *vice versa* for Q1. <sup>#</sup>: sleep apnoea was based on STOP-BANG (Snoring, Tired, Observed (snort), Pressure (blood pressure), Body mass index (BMI), Age, Neck, Gender) tool. Only high-risk OSA cases were included (prevalence in males= 31.5%; females=23.4%).

association between a healthy lifestyle and OSA in females [15]. The observed sex-specific associations warrant further exploration. Potential underlying biological mechanisms or external behavioural factors might account for these differences. For instance, during menopause, hormonal changes, specifically the decline in oestrogen levels, may contribute to an increased risk of OSA *via* the sexual hormone receptors (*i.e.* estradiol and progesterone) in the carotid body [42]. The majority of unhealthy plant-based foods, such as refined grains, potatoes, sugar-sweetened beverages and sweets and desserts, have very high non-complex carbohydrates, thereby increasing the energy imbalance. Alternatively, avoiding uPDI and





intaking hPDI, especially phytoestrogen-rich legumes (especially soy) and legume-derived foods [43], may alleviate the risk of OSA in postmenopausal women.

# Potential mechanisms

The effect of a healthy plant-based diet on OSA could be attributed to reduced inflammation and a lower risk of obesity. The anti-inflammatory effect of hPDI on OSA could be partially related to various anti-inflammatory components such as high levels of antioxidant nutrients like vitamin C and E, high levels of B-vitamins and low levels of detrimental dietary factors (*e.g.* nitrates and nitrites) [13–15, 19]. A meta-analysis of 18 observational studies indicated that vegetarianism is associated with lower serum concentrations of high-sensitivity C-reactive protein [21]. In our previous studies, we also found that an anti-inflammatory diet, a healthy diet or the consumption of healthy plant-based foods were all linked to a decreased risk of developing obesity [14, 15].

Furthermore, plant-based diets that are low in fat and high in fibre have the potential to positively alter the microbial composition of the intestine, shifting the microbiome environment towards beneficial bacteria like *Prevotella* and *Bacteroides*, while reducing the presence of *Firmicutes* [44]. Imbalances in the gut microbiota in turn have also been associated with conditions such as obesity, CVD and depression [45, 46]. Available evidence suggests there is a bidirectional relationship between OSA and the composition of the microbiota [46]. In animal models, sleep fragmentation and intermittent hypoxaemia alter the gut microbiome composition and reduce intestinal epithelial barrier integrity, which promotes insulin resistance and systemic inflammation [47, 48]. This evidence may suggest that changes in the gut microbiome might predispose individuals to the development of OSA or sleep disruption. In summary, one of the potential mechanisms through which a healthy plant-based diet could benefit OSA patients could be modifications in the gut microbiome [44].

# Conclusions

The evidence presented in this study highlights the significant link of plant-based diets and OSA risk, with particular emphasis on the protective role of diets like the healthy plant-based diet. Such findings prompt consideration for re-evaluation of dietary recommendations to a shift towards emphasising healthy plant-based diets that are rich in anti-inflammatory components and antioxidant nutrients and low in harmful dietary factors. These diets not only influence OSA risk but also have the potential to modulate other pathophysiological mechanisms, possibly affecting various other health conditions, from obesity to CVD. Furthermore, the distinct sex-specific patterns observed call for more personalised dietary interventions. Additional research with longitudinal data is crucial to substantiate these new findings, particularly focusing on how plant-based diets affect sleep apnoea risk, especially in the context of maintained body weight.

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Conflict of interest: Outside the submitted work, D.J. Eckert has had research grants from Bayer, Takeda, Invicta Medical, Apnimed, Eli Lilly and Withings, and has served on scientific advisory boards or as a consultant for Apnimed, Invicta, Mosanna, Takeda and Bayer. The remaining authors have no potential conflicts of interest to disclose.

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### References

1 Benjafield AV, Ayas NT, Eastwood PR, *et al.* Estimation of the global prevalence and burden of obstructive sleep apnoea: a literature-based analysis. *Lancet Respir Med* 2019; 7: 687–698.

- 2 Yeghiazarians Y, Jneid H, Tietjens JR, *et al.* Obstructive sleep apnea and cardiovascular disease: a scientific statement from the American Heart Association. *Circulation* 2021; 144: e56–e67.
- 3 Marin JM, Carrizo SJ, Vicente E, *et al.* Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study. *Lancet* 2005; 365: 1046–1053.
- 4 Reutrakul S, Mokhlesi B. Obstructive sleep apnea and diabetes: a state of the art review. *Chest* 2017; 152: 1070–1086.
- 5 Xie C, Zhu R, Tian Y, *et al.* Association of obstructive sleep apnoea with the risk of vascular outcomes and all-cause mortality: a meta-analysis. *BMJ Open* 2017; 7: e013983.
- 6 Peppard PE, Young T, Palta M, *et al.* Longitudinal study of moderate weight change and sleep-disordered breathing. *JAMA* 2000; 284: 3015–3021.
- 7 Edwards BA, Bristow C, O'Driscoll DM, *et al.* Assessing the impact of diet, exercise and the combination of the two as a treatment for OSA: a systematic review and meta-analysis. *Respirology* 2019; 24: 740–751.
- 8 Kuna ST, Reboussin DM, Strotmeyer ES, *et al.* Effects of weight loss on obstructive sleep apnea severity. Ten-year results of the sleep AHEAD study. *Am J Respir Crit Care Med* 2021; 203: 221–229.
- 9 Schiavo L, Pierro R, Asteria C, et al. Low-calorie ketogenic diet with continuous positive airway pressure to alleviate severe obstructive sleep apnea syndrome in patients with obesity scheduled for bariatric/metabolic surgery: a pilot, prospective, randomized multicenter comparative study. Obes Surg 2022; 32: 634–642.
- 10 de Melo CM, Dos Santos Quaresma MVL, Del Re MP, et al. One-month of a low-energy diet, with no additional effect of high-protein, reduces Obstructive Sleep Apnea severity and improve metabolic parameters in obese males. *Clin Nutr ESPEN* 2021; 42: 82–89.
- 11 Vasquez MM, Goodwin JL, Drescher AA, *et al.* Associations of dietary intake and physical activity with sleep disordered breathing in the Apnea Positive Pressure Long-Term Efficacy Study (APPLES). *J Clin Sleep Med* 2008; 4: 411–418.
- 12 Farrell ET, Wirth MD, McLain AC, *et al.* Associations between the dietary inflammatory index and sleep metrics in the energy balance study (EBS). *Nutrients* 2023; 15: 419.
- 13 Turner-McGrievy GM, Wirth MD, Shivappa N, *et al.* Randomization to plant-based dietary approaches leads to larger short-term improvements in Dietary Inflammatory Index scores and macronutrient intake compared with diets that contain meat. *Nutr Res* 2015; 35: 97–106.
- 14 Wang YB, Shivappa N, Hébert JR, *et al.* Association between dietary inflammatory index, dietary patterns, plant-based dietary index and the risk of obesity. *Nutrients* 2021; 13: 1536.
- 15 Melaku YA, Reynolds AC, Appleton S, *et al.* High-quality and anti-inflammatory diets and a healthy lifestyle are associated with lower sleep apnea risk. *J Clin Sleep Med* 2022; 18: 1667–1679.
- 16 Salehin S, Rasmussen P, Mai S, *et al.* Plant based diet and its effect on cardiovascular disease. *Int J Environ Res Public Health* 2023; 20: 3337.
- 17 Greger M. A whole food plant-based diet is effective for weight loss: the evidence. *Am J Lifestyle Med* 2020; 14: 500–510.
- 18 Satija A, Bhupathiraju SN, Rimm EB, *et al.* Plant-based dietary patterns and incidence of type 2 diabetes in US men and women: results from three prospective cohort studies. *PLoS Med* 2016; 13: e1002039.
- 19 Li D. Chemistry behind vegetarianism. J Agric Food Chem 2011; 59: 777–784.
- 20 Yokoyama Y, Nishimura K, Barnard ND, *et al.* Vegetarian diets and blood pressure: a meta-analysis. *JAMA Intern Med* 2014; 174: 577–587.
- 21 Haghighatdoost F, Bellissimo N, de Zepetnek JO T, *et al.* Association of vegetarian diet with inflammatory biomarkers: a systematic review and meta-analysis of observational studies. *Public Health Nutr* 2017; 20: 2713–2721.
- 22 Tomova A, Bukovsky I, Rembert E, *et al.* The effects of vegetarian and vegan diets on gut microbiota. *Front Nutr* 2019; 6: 47.
- 23 Gan ZH, Cheong HC, Tu YK, et al. Association between plant-based dietary patterns and risk of cardiovascular disease: a systematic review and meta-analysis of prospective cohort studies. Nutrients 2021; 13: 20211105.
- 24 Carneiro-Barrera A, Amaro-Gahete FJ, Guillén-Riquelme A, *et al.* Effect of an interdisciplinary weight loss and lifestyle intervention on obstructive sleep apnea severity: the INTERAPNEA randomized clinical trial. *JAMA Netw Open* 2022; 5: e228212.
- 25 Patel K, Lawson M, Cheung J. Whole-food plant-based diet reduces daytime sleepiness in patients with OSA. Sleep Med 2023; 107: 327–329.
- 26 Kim H, Rebholz CM, Garcia-Larsen V, et al. Operational differences in plant-based diet indices affect the ability to detect associations with incident hypertension in middle-aged US adults. J Nutr 2020; 150: 842–850.
- 27 Martínez-González MA, Sánchez-Tainta A, Corella D, et al. A provegetarian food pattern and reduction in total mortality in the Prevención con Dieta Mediterránea (PREDIMED) study. Am J Clin Nutr 2014; 100: Suppl 1, 320s–328s.

- 28 Centers for Disease Control and Prevention. National Health and Nutrition Examination Survey response rates and population totals. Date last accessed: 2 January 2020. Date last updated: 2 February 2024. wwwn.cdc. gov/nchs/nhanes/ResponseRates.aspx
- 29 McQuillan GM, McLean JE, Chiappa M, *et al.* National Health and Nutrition Examination Survey Biospecimen Program: NHANES III (1988–1994) and NHANES 1999–2014. *Vital Health Stat* 2015; 2: 1–14.
- 30 Ahluwalia N, Dwyer J, Terry A, *et al.* Update on NHANES dietary data: focus on collection, release, analytical considerations, and uses to inform public policy. *Adv Nutr* 2016; 7: 121–134.
- 31 US Department of Health and Human Services. Dietary guidelines. Date last accessed: 30 March 2021. Date last updated: 9 February 2024. www.health.gov/dietaryguidelines/
- 32 Chung F, Abdullah HR, Liao P. STOP-bang questionnaire: a practical approach to screen for obstructive sleep apnoea. *Chest* 2016; 149: 631–638.
- 33 Sweetman A, Lechat B, Appleton S, *et al.* Association of co-morbid insomnia and sleep apnoea symptoms with all-cause mortality: analysis of the NHANES 2005–2008 data. *Sleep Epidemiol* 2021; 2: 100043.
- 34 Joshipura K, Muñoz-Torres F, Vergara J, *et al.* Neck circumference may be a better alternative to standard anthropometric measures. *J Diabetes Res* 2016; 2016: 6058916.
- 35 Chung F, Chau E, Yang Y, *et al.* Serum bicarbonate level improves specificity of STOP-bang screening for obstructive sleep apnea. *Chest* 2013; 143: 1284–1293.
- 36 Melaku YA, Reynolds AC, Gill TK, *et al.* Association between macronutrient intake and excessive daytime sleepiness: an iso-caloric substitution analysis from the north West Adelaide health study. *Nutrients* 2019; 11: 2374.
- 37 Liu Y, Tabung FK, Stampfer MJ, *et al.* Overall diet quality and proinflammatory diet in relation to risk of obstructive sleep apnea in 3 prospective US cohorts. *Am J Clin Nutr* 2022; 116: 1738–1747.
- 38 Bove C, Jain V, Younes N, *et al.* What you eat could affect your sleep: dietary findings in patients with newly diagnosed obstructive sleep apnea. *Am J Lifestyle Med* 2021; 15: 305–312.
- **39** Potts KS, Wallace ME, Gustat J, *et al.* Diet quality and sleep characteristics in midlife: the Bogalusa heart study. *Nutrients* 2023; 15: 2078.
- 40 Gaona-Pineda EB, Martinez-Tapia B, Rodríguez-Ramírez S, *et al.* Dietary patterns and sleep disorders in Mexican adults from a National Health and Nutrition Survey. *J Nutr Sci* 2021; 10: e34.
- 41 Reid M, Maras JE, Shea S, *et al.* Association between diet quality and sleep apnea in the Multi-Ethnic Study of Atherosclerosis. *Sleep* 2019; 42: zsy194.
- **42** Martins FO, Conde SV. Gender differences in the context of obstructive sleep apnea and metabolic diseases. *Front Physiol* 2021; 12: 792633.
- 43 Domínguez-López I, Yago-Aragón M, Salas-Huetos A, *et al.* Effects of dietary phytoestrogens on hormones throughout a human lifespan: a review. *Nutrients* 2020; 12: 2456.
- 44 Beam A, Clinger E, Hao L. Effect of diet and dietary components on the composition of the gut microbiota. *Nutrients* 2021; 13: 2795.
- 45 Alam MJ, Puppala V, Uppulapu SK, et al. Human microbiome and cardiovascular diseases. *Prog Mol Biol Transl Sci* 2022; 192: 231–279.
- 46 Cai Y, Juszczak HM, Cope EK, et al. The microbiome in obstructive sleep apnea. Sleep 2021; 44: zsab061.
- 47 Poroyko VA, Carreras A, Khalyfa A, *et al.* Chronic sleep disruption alters gut microbiota, induces systemic and adipose tissue inflammation and insulin resistance in mice. *Sci Rep* 2016; 6: 35405.
- 48 Moreno-Indias I, Torres M, Montserrat JM, *et al.* Intermittent hypoxia alters gut microbiota diversity in a mouse model of sleep apnoea. *Eur Respir J* 2015; 45: 1055–1065.