

SCIENTIFIC INVESTIGATIONS

High-quality and anti-inflammatory diets and a healthy lifestyle are associated with lower sleep apnea risk

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Study Objectives: Most studies on diet and sleep apnea focus on calorie restriction. Here we investigate potential associations between dietary quality (Healthy Eating Index [HEI], Dietary Inflammatory Index [DII]) and overall healthy lifestyle with sleep apnea risk.

Methods: National Health and Nutrition Examination Survey data (waves 2005–2008 and 2015–2018; n = 14,210) were used to determine HEI, DII, and their quintiles, with the fifth quintile indicating highest adherence to each dietary construct. A healthy lifestyle score was determined using diet, smoking, alcohol intake, and physical activity level. The STOP-BANG questionnaire was used to define sleep apnea risk. Generalized linear regression models with binomial family and logit link were used to investigate potential associations. The models were adjusted for socioeconomic status, lifestyle factors, and chronic conditions.

Results: The prevalence of high sleep apnea risk was 25.1%. Higher DII was positively associated with sleep apnea (odds ratio_{Quintile 5 vs Quintile 1} = 1.55; 95% confidence interval, 1.24–1.94; *P* for trend < .001), whereas higher HEI was associated with reduced sleep apnea risk (odds ratio_{Quintile 5 vs Quintile 1} = 0.72; 95% confidence interval, 0.59–0.88; *P* for trend = .007). Higher healthy lifestyle score was also associated with decreased odds of sleep apnea (*P* for trend < .001). There was a significant interaction between healthy lifestyle and sex with sleep apnea risk (*P* for interaction = .049) whereby females with higher healthy lifestyle scores had a lower risk of sleep apnea compared to males.

Conclusions: Higher-quality and anti-inflammatory diets and a healthier overall lifestyle are associated with lower sleep apnea risk. These findings underline the importance of strategies to improve overall diet quality and promote healthy behavior, not just calorie restriction, to reduce sleep apnea risk.

Keywords: diet, dietary quality, Dietary Inflammatory Index, dietary pattern, lifestyle, inflammation, adiposity, sleep apnea

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BRIEF SUMMARY

Current Knowledge/Study Rationale: Calorie restriction has been the focus of lifestyle change interventions in reducing sleep apnea risk and severity. This study is unique in demonstrating potential benefits of high-quality and anti-inflammatory diets and a healthy lifestyle in reducing sleep apnea risk in a large community-based sample.

Study Impact: We highlighted that, in designing behavioral interventions, not only targeting weight loss through calorie restriction and/or physical activity but also aiming to improve overall dietary quality and promote a healthy lifestyle are essential. Future research should confirm the findings of our study using randomized controlled trials and/or longitudinal studies.

INTRODUCTION

Obstructive sleep apnea (OSA) is a common breathing disorder characterized by intermittent obstruction of the upper airway during sleep.¹ Globally, it is estimated that 1 billion people have sleep apnea.² OSA is associated with elevated risk of cardiometabolic diseases such as cardiovascular disease (CVD),³ diabetes,⁴ and premature mortality.⁵ Obesity, and in particular central obesity, is the strongest predictor of sleep apnea.⁶ Weight loss through modification of lifestyle is an effective treatment for sleep apnea.⁷ Behavioral health interventions targeted to improve a healthy lifestyle, such as exercise,⁸ diet, or a combination of the 2,^{9,10} can also reduce OSA severity.

Most diet intervention studies for OSA have focused on weight loss through calorie restriction^{11,12} or targeting specific

dietary components^{13,14} rather than overall dietary quality. Indeed, most studies in this area are secondary analyses of randomized clinical trials. For instance, specific diet components such as protein and total fat were associated with sleep apnea in the Apnea Positive Pressure Long-term Efficacy Study (APPLES; n = 320).¹³ Conversely, a proinflammatory diet in 296 patients with OSA was not associated with sleep parameters, including the apnea-hypopnea index (AHI).¹⁵ A key limitation of these findings is that the population groups were clinical referral samples and thus do not represent people with OSA in the general community. Most participants in these prior studies were also male. Some studies were conducted in a specific ethnic group.¹⁶ Thus, whether the findings also apply to women or other ethnic groups is unknown, particularly given known sex and ethnicity differences in OSA.^{17,18} Although

these studies have advanced knowledge of the potential relationships of diet and dietary components with sleep apnea, additional research with larger samples is required to examine the role of overall diet quality and inflammatory properties of diet on sleep apnea risk at the population level.

In the current study, we aimed to investigate potential associations between dietary quality and overall healthy lifestyle with sleep apnea risk. We hypothesized that overall diet quality, inflammatory properties of diet, and overall lifestyle are associated with sleep apnea risk in the general population.

METHODS

Study design and population

In this study, we used data from 4 cycles of the National Health and Nutrition Examination Survey (NHANES; 2005–2008 and 2015–2018), where sleep apnea risk data were available.¹⁹ Data were collected using interview, medical assessment, and laboratory investigations. A total of 39,722 individuals participated in the 4 cycles. We used data from 14,210 participants after excluding those with missing data (Figure 1). NHANES was conducted in accordance with ethics approvals from the National Center for Health Statistics Ethics Review Board.²⁰

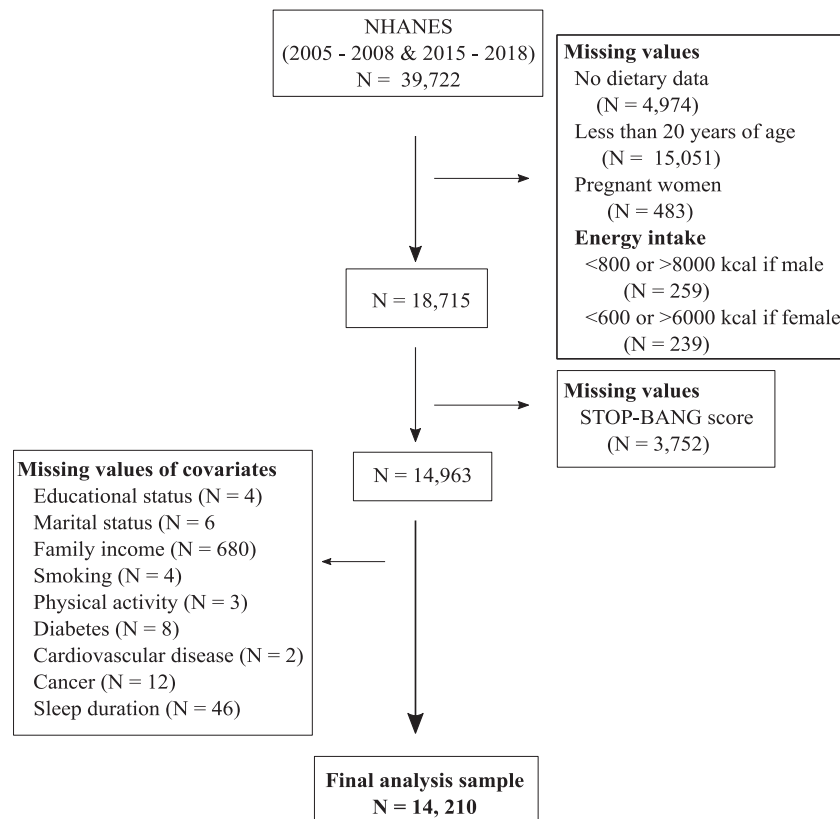
Assessment of dietary intake

In each cycle of NHANES, dietary data were collected using a 24-hour recall method.²¹ Detailed methods are provided elsewhere.²¹ In short, for the 2005–2008 and 2015–2018 cycles, the U.S. Department of Agriculture (USDA) Automated Multiple-Pass Method was used to collect dietary data over 2 days (first, face-to-face, and second, using the telephone). In this study, we used the first-day dietary data collected using face-to-face interview in the primary analysis. The USDA Food and Nutrient Database for Dietary Studies (FNDDS) was used to determine micro- and macronutrient contents of food.²¹ In addition, the USDA Food Patterns Equivalents Database was linked with participants' data. The Food Patterns Equivalents Database disaggregates foods and beverages into 37 USDA Food Patterns components.²²

Healthy Eating Index and Dietary Inflammatory Index

We constructed dietary indices that reflect overall diet quality and inflammatory properties of each participant's diet. Overall diet quality was assessed using the Healthy Eating Index (HEI; HEI-2015).²³ Scoring of the HEI is provided in Table S1 in the supplemental material. In brief, a score of either 0–5 or 0–10 (dependent on dietary components) was assigned to each HEI

Figure 1—Sampling scheme.



NHANES = National Health and Nutrition Examination Survey, STOP-BANG = Snoring, Tired, Observed apneas, Pressure (blood pressure), Body mass index, Age, Neck, Gender.

component. Higher scores indicate greater adherence to the recommended levels per day. Each of the components is scored on a density basis out of 1,000 calories, except for fatty acids, which are considered as a ratio of unsaturated to saturated fatty acids. A total of 13 foods, food groups, and nutrients were included, with overall possible HEI scores ranging from 0 (lowest adherence) to 100 (highest adherence). The diet components included in the calculation were as follows: total fruits (0–5), whole fruits (0–5), total vegetables (0–5), greens and beans (0–5), whole grains (0–10), dairy (0–10), total protein foods (0–5), seafood and animal proteins (0–5), fatty acids (0–10), refined grains (0–10), sodium (0–10), added sugars (0–10), and saturated fats (0–10).²³

Using previously described methodology,²⁴ we determined the energy-adjusted DII using alcohol and 26 nutrients: carbohydrates; fat; protein; vitamins A, B1, B2, B3, B6, B12, C, D, and E; saturated fatty acids, monounsaturated fatty acids, and polyunsaturated fatty acids; omega-3; omega-6; fiber; cholesterol; iron; magnesium; zinc; selenium; folic acid; beta carotene; and caffeine. Data were not available for 18 food parameters: *trans*-fat, anthocyanidins, eugenol, flavanol, flavones, flavonols, flavonones, isoflavones, garlic, ginger, onion, saffron, turmeric, pepper, thyme/oregano, rosemary, and green and black tea. Energy intake was not considered in computing DII as it was used for energy adjustment. In the calculation, means and standard deviations (SDs) of all nutrients were determined based on nutrient intakes of 11 populations around the world.²⁴ We subtracted respondents' intake from the standard mean and divided by the SD to calculate *z*-scores. Inflammatory scores of each nutrient were derived from the literature.²⁴ These were multiplied by *z*-scores, resulting in nutrient-specific inflammatory scores. Overall DII was then calculated by summation of these inflammatory scores.²⁴ To control for differences in total energy intake, we adjusted the DII per 1,000 calories consumed. Higher DII values indicate a more proinflammatory diet.

Assessment and definition of healthy lifestyle factors

The healthy lifestyle score is composed of 4 lifestyle factors²⁵: diet, physical activity, smoking, and alcohol intake (Table S2 in the supplemental material). Levels of physical activity were assessed and scored using the validated Global Physical Activity Questionnaire Analysis (GPAQ).²⁶ Specifically, the number of minutes that participants spent each week doing moderate to vigorous activities requiring at least 4 metabolic equivalent of task units (METs) per hour was estimated.²⁷

Smoking status was determined as follows: never, former (smoked > 100 cigarettes in lifetime but does not currently smoke), and current (smoked > 100 cigarettes in lifetime and smokes currently). Alcohol intake was determined using 24-hour recall dietary data. We defined low-risk lifestyle factors as moderate alcohol intake (5–15 g alcohol/day in women, 5–30 g alcohol/day in men),²⁸ not smoking, physical activity levels of ≥ 600 MET-minutes per week at moderate to vigorous intensity, and an HEI score in the upper 2 quintiles. One point was given for each of the 4 lifestyle behavior categories. Possible scores for healthy lifestyle ranged from 0 to 4, with higher scores representing a healthier lifestyle.²⁵ Given the small

number of participants with a score of 4 (*n* = 181), scores of 3 or 4 were combined to provide possible healthy lifestyle scores of 0, 1, 2, and 3–4.

Sleep apnea risk

Sleep apnea risk was assessed using the STOP-BANG (Snoring, Tired, Observed apnea, Pressure, Body mass index, Age, Neck, Gender) questionnaire.²⁹ Details on methods of assessment of each component, scoring criteria, and frequency are provided in Table S3 in the supplemental material. Observed apneas were determined based on a question: "In the past 12 months, how often did you snort, gasp, or stop breathing while you were asleep?" We used waist circumference as a substitute for neck circumference as there were no data on neck circumference. Neck and waist circumferences are highly correlated (*r* = .64).³⁰ Total STOP-BANG scores ranged from 0–8. Scores < 2 are considered low risk, 3–4 intermediate risk, and > 5 high risk. In addition, if the BANG score is greater than or equal to 2 and a participant is (1) male or (2) has a body mass index (BMI) > 35 kg/m² or (3) exceeded the sex-specific waist circumference cutoff (male, > 102 cm; female, > 88 cm), the participant was considered to be at high risk of sleep apnea.²⁹ STOP-BANG scores > 3 have a sensitivity of 84% and specificity of 56.4% in identifying sleep apnea (AHI > 5 events/h sleep).²⁹ In the main analysis, we used high sleep apnea risk (hereafter referred to as sleep apnea risk) as an outcome variable. In the sensitivity analysis, we combined the intermediate and high sleep apnea risk groups together and assessed the association with different dietary indices.

In further sensitivity analyses, we also used the OSA-50 tool³¹ as an alternative approach to determine sleep apnea risk. The OSA-50 has 4 components: waist circumference (waist circumference > 102 cm if male or > 88 cm if female; 3 points), snoring (3 points), stop breathing (2 points), and age (≥ 50 years, 2 points). The score ranges from 0 to 10 and those with scores ≥ 5 were considered to have high sleep apnea risk. With a cutoff score of ≥ 5, the OSA-50 has been shown to have a sensitivity of 100% and specificity of 29% to detect moderate-to-severe OSA.³¹

Covariates

We considered sociodemographic characteristics (age, sex, race, marital status, income), behavioral factors (smoking, physical activity, sleep duration, and alcohol consumption), and chronic conditions (CVD, cancer, and diabetes) in our analyses. Systemic inflammation was assessed based on C-reactive protein (CRP) (*n* = 13,623).

Statistical analysis

All analyses account for the complex survey design using NHANES-assigned weights, population sampling units, and strata. Proportion, mean (SD), and median (interquartile range) were used to summarize categorical and continuous variables, as appropriate. The association of dietary indices with high sleep apnea risk was determined using generalized linear regression with binomial family and log-link function. We developed 4 models to investigate the potential associations

between dietary indices and sleep apnea: model 1 was adjusted for sociodemographic characteristics (sex, age, race, marital status, education, and income); model 2 was additionally adjusted for smoking and physical activity; model 3 was additionally adjusted for CVD, cancer, and diabetes; and model 4 was additionally adjusted for sleep duration. For the association of HEI and sleep apnea risk, models 2, 3, and 4 were additionally adjusted for alcohol consumption. We determined *P* for trend using quintiles of HEI and DII as continuous variables. Given that some covariates such as diabetes and CVD could potentially mediate (rather than confound) the association of diet and sleep apnea risk, a model-based approach both with and without including covariates provides more insight into how these variables may affect estimates.

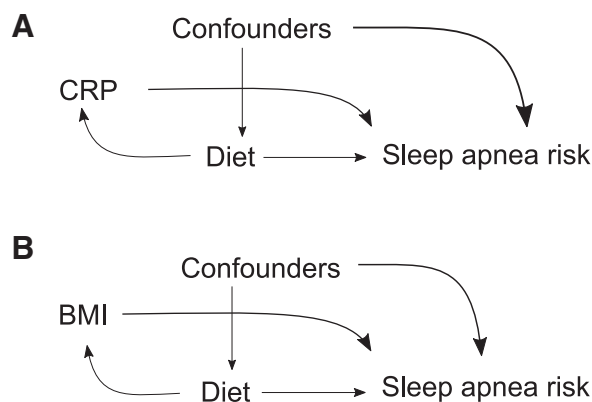
We developed 3 models to investigate the potential association between healthy lifestyle and sleep apnea. Model 1 was adjusted for sex, age, race, marital status, education, and income; model 2 was additionally adjusted for CVD, cancer, and diabetes; and model 3 was additionally adjusted for sleep duration. Multiplicative interaction terms were used to test for differences in association of HEI, DII, and healthy lifestyle with sleep apnea risk by sex and other relevant covariates (race, income, CVD, and diabetes status). Furthermore, we considered that systemic inflammation and adiposity could mediate the association of diet and sleep apnea risk (Figure 2). Therefore, we computed the proportion of association³² of HEI and DII (as continuous variables) with sleep apnea risk that was mediated by systematic inflammation (CRP; log-transformed, continuous; *n* = 13,632) and adiposity (BMI, continuous; *n* = 14,210) (model 3). The Stata *medeff* command (StataCorp, College Station, TX) was used for mediation analysis. We did not use complex survey design weighting as Stata does not support the use of the *svy* command with *medeff*.

Post-estimation commands such as “margins” and “marginplot” were used to produce figures of joint association between healthy lifestyle score and sex against the probability of high sleep apnea risk. All analyses were performed using Stata version 16.0 or R (R Foundation for Statistical Computing, Vienna, Austria).

Sensitivity analyses

We performed several sensitivity analyses. First, we used STOP-BANG and STOP scores as a count outcome variable. In this analysis, we used a generalized linear model with Poisson family and log link and calculated the incidence rate ratio. Second, we considered both intermediate- and high-risk groups in the STOP-BANG score as an outcome variable. Third, we determined the association of diet and sleep apnea using self-reported physician-diagnosed OSA as the outcome. Data on self-reported physician-diagnosed sleep apnea were available only in the 2005–2008 waves (*n* = 6,998). Fourth, we explored the association of diet with each of the STOP-BANG components (Snoring, Tiredness, Observed apnea, Hypertension, Body mass index, and waist circumference). Fifth, we used the OSA-50 to define sleep apnea risk. Sixth, we used an average of day 1 and day 2 dietary data for HEI and DII for those who completed the 2-day dietary interview (*n* = 12,284). Finally, we determined the ratio of reported energy intake

Figure 2—Direct acyclic graph depicting the potential mediation.



Direct acyclic graph depicting the potential mediation by (A) CRP and (B) BMI in the association between diet and sleep apnea risk. BMI = body mass index, CRP = C-reactive protein.

(day 1) and basal metabolic rate based on weight, height, age, and sex. Dietary intake was classified into 3 categories based on this ratio: underreported (minimum to 0.96), plausible (0.96 to 2.49), and overreported (2.49 to maximum).³³ We then used data from those who had plausible dietary intakes (*n* = 9,951; 70.0%).

RESULTS

Participant characteristics

Half (49.4%) of the participants were male. One-third (32.8%) had low levels of physical activity and one-fifth (20.5%) were current smokers. Diabetes was the most common chronic condition (12.6%), followed by CVD (9.7%) and cancer (7.7%). Snoring was reported by 28.5% of participants and 25.1% were at high risk of sleep apnea, with an intermediate risk or higher of 50.5% according to STOP-BANG. The prevalence of apnea risk based on OSA-50 was 42.2% (Table 1). The mean (SD) of the HEI was 41.9 (12.1) and the DII was 1.2 (1.8). The range for the HEI was 2 to 90 and was -5.39 to 5.38 for the DII. The Pearson correlation of the DII and HEI was -0.62 (Figure S1 in the supplemental material).

The HEI, DII, and sleep apnea risk

Table 2 shows the association of the HEI and DII with sleep apnea in all participants and by sex. Participants in the fifth quintile of HEI had 28% (odds ratio = 0.72; 95% confidence interval [CI], 0.59–0.88) lower odds of sleep apnea compared with those in the first quintile (*P* for trend = .007). Participants in the fifth quintile of DII had 55% (odds ratio: 1.55; 95% CI, 1.24–1.94) increased odds of sleep apnea compared with those in the first quintile (*P* for trend < .001). While a higher HEI was associated with a lower prevalence of sleep apnea in males and females, higher adherence to a proinflammatory diet (higher DII) was associated with increased prevalence of sleep apnea.

Table 1—Characteristics of study participants.

Characteristics	Overall, %	Healthy Eating Index			Dietary Inflammatory Index		
		Q1	Q3	Q5	Q1	Q3	Q5
Sex							
Male	49.4	52.9	49.3	44.5	38.3	54.3	52.7
Education							
Less than high school	13.5	18.5	13.9	8.6	9.8	13.6	17.7
School diploma (including GED)	24.6	30.8	23.3	16.3	17.1	24.6	31.3
More than high school	61.8	50.7	62.9	75.1	73.1	61.8	51
Income							
Under \$20,000	15.3	20.5	15	11.3	11.9	15.1	20.3
\$20,000–\$34,999	18.5	20.8	17.8	16	16.3	18.6	20.4
\$35,000–\$54,999	17.8	17.3	17.7	18.4	16.3	19.1	17.5
\$55,000–\$74,999	12.5	13.1	13.7	9.4	9.6	12.1	13.8
\$75,000 and above	35.9	28.3	35.9	44.9	45.8	35.2	28
Marital status							
Married/living with partner	66.8	61.3	65.7	71.6	71.3	64.6	61.4
Widowed	4.5	3	4.6	6.1	5	5.5	3.5
Divorced	9.5	10.7	10.2	9.1	8.6	10.4	10.9
Separated	2.3	2.9	2.4	1.3	1.3	2	3.1
Never married	17	22.2	17.1	11.9	13.7	17.6	21.1
Race/ethnicity							
Mexican American	8.1	8.4	8.3	6.5	6.7	8.9	6.9
Other Hispanic	4.9	4.8	4.8	4.8	5.3	5.1	4.1
Non-Hispanic White	69.2	68.8	67.6	71.1	71	69.1	68.4
Non-Hispanic Black	10.3	12.1	11.3	7.7	6.3	10.3	15.4
Other race, including multiracial	7.5	5.8	8	10	10.6	6.6	5.2
Physical activity level							
Low (< 600 MET-minutes/w)	32.8	35.3	35.9	24.4	26.9	33.4	35
Moderate (≥ 600 and < 1,200 MET-minutes/wk)	11.3	10.9	10.7	12.2	12.7	9.9	10.8
High (≥ 1,200 MET-minutes/wk)	56	53.8	53.3	63.4	60.4	56.8	54.1
Smoking							
Never	54.4	46	54.2	62.6	63.3	53.3	46
Ex-smoker	25.1	20.8	26.3	29	27.1	25.3	21.7
Smoker	20.5	33.2	19.5	8.4	9.6	21.4	32.2
Healthy lifestyle score							
0	9.6	17.8	13.7	0	2.8	9.9	17.6
1	32.1	48.2	43.1	9.2	15	34.3	44.7
2	36.9	31.8	35.8	38.1	39.9	36.1	32.5
3–4	21.4	2.2	7.4	52.8	42.3	19.7	5.2
Diabetes	12.6	11.5	12.9	14.5	13.8	12.3	10.9
Cancer	9.7	7.6	9.3	11.8	11.8	8.3	8.7
CVD	7.7	7.2	7.5	8.4	8.1	6.8	8.3
High blood pressure	32.6	30.8	32	36.8	34.2	32.3	31.4
Obese (> 35 kg/m ²)	16.1	20.5	16.5	13.3	11.3	15.2	20.3
Snored	28.5	31.8	28.9	22.1	21.5	29.9	32
Observed apnea	5.6	5.9	6.2	4.2	4.7	5.6	6.3
Tired	30.5	35.7	30	26.2	25.6	30.1	37.3
High waist circumference	56	58.2	55.6	53.7	51.5	55.1	60.1

(continued on following page)

Table 1—Characteristics of study participants. (Continued)

Characteristics	Overall, %	Healthy Eating Index			Dietary Inflammatory Index		
		Q1	Q3	Q5	Q1	Q3	Q5
STOP score							
0	37	33.1	36.4	41.1	43.3	36.4	31.7
1	36.9	38.6	38.4	35.2	33.9	37.3	38.9
2	19.2	20.3	18.3	17.8	17.1	19.7	21.5
3	5.6	6.7	5.6	4.8	4.6	5.5	6.4
4	1.2	1.2	1.4	1	1	1.2	1.4
STOP-BANG score							
0	7.3	5.7	7.4	8.6	11.3	6.5	5
1	21.4	21.5	20.1	22.2	23.6	21.8	19.9
2	21.8	22.5	22.2	22.4	20.9	21.1	22.3
3	20.6	20.9	22.2	19	18.4	21.7	22.4
4	16.2	15.5	16	14.5	15.2	16.6	16.3
5	8.3	9.2	7.7	9	7.1	8	9.1
6	3.3	3.4	3.3	3.4	3.1	2.7	3.9
7	0.8	1	0.8	0.8	0.5	1	0.8
8	0.2	0.2	0.3	0.1	0	0.5	0.3
Sleep apnea risk (STOP-BANG)							
Low	50.5	49.8	49.7	53.3	55.8	49.5	47.2
Intermediate	24.4	22.6	26.5	23.1	22.4	25.3	24.2
High	25.1	27.6	23.8	23.6	21.8	25.2	28.5
Sleep apnea risk (OSA-50)							
Low risk	57.8	61.2	57.6	56.2	58.6	57.3	58.6
High risk	42.2	38.8	42.4	43.8	41.4	42.7	41.4
Self-reported doctor-diagnosed sleep apnea (n = 6,998)	4.9	4.8	5.3	4.7	4.7	4.5	4.6

Sleep apnea was based on STOP-BANG (Snoring, Tired, Observed apnea, Pressure [blood pressure], Body mass index, Age, Neck, Gender). CVD = cardiovascular disease, GED = General Educational Development, MET = metabolic equivalent of task, Q = quintile.

There was no significant interaction between dietary indices and sex in predicting sleep apnea risk.

Figure 3 depicts the association of HEI (continuous) and DII (continuous) with sleep apnea risk and the mediating effects of CRP (continuous, log-transformed) and BMI (continuous). One-quarter of the association of HEI (27%; 95% CI, 19%–39%) and DII (24%; 95% CI, 20%–31%) with sleep apnea risk was mediated by systemic inflammation. BMI mediated almost half of the association (HEI, 47%; 95% CI, 35%–71%; DII, 51%; 95% CI, 42%–67%).

Healthy lifestyle and sleep apnea

Associations of healthy lifestyle with sleep apnea risk stratified by sex are depicted in **Table 3**. Healthy lifestyle was associated with reduced odds of sleep apnea in males, females, and both sexes. The association was stronger in females than in males. There was a significant interaction between healthy lifestyle and sex for sleep apnea risk (*P* for interaction = .048). A marginal probability of sleep apnea risk by healthy lifestyle and sex is depicted in **Figure 4**. Females with higher healthy lifestyle

scores had lower odds of sleep apnea compared with males. The association of healthy lifestyle score with sleep apnea was stronger than individual components (diet quality, physical activity, optimal alcohol intake, and not smoking). While smoking and alcohol intake had a stronger association with sleep apnea in males, physical activity and alcohol had a stronger link in females (**Table 3**).

Sensitivity analyses

Associations between the HEI and DII with STOP score and STOP-BANG score as count variables are shown in **Table S4** in the supplemental material. There were significant inverse associations between HEI with STOP and STOP-BANG scores and positive associations with DII. Using both intermediate and high-risk STOP-BANG scores to indicate OSA risk did not change the result (**Table S4**). There were no associations between HEI and DII with self-reported physician-diagnosed sleep apnea (n = 6,998) (**Table S4**). Except for reported observed apnea, almost all other components of STOP-BANG

Table 2—Association between diet and sleep apnea.

Models	Odds Ratio (95% CI)					P for Trend
	Q1	Q2	Q3	Q4	Q5	
Healthy Eating Index[#]						
All participants						
Model 1	1.00	0.77 (0.63–0.94)	0.71 (0.59–0.85)	0.75 (0.65–0.86)	0.64 (0.53–0.77)	< .001
Model 2	1.00	0.78 (0.64–0.95)	0.73 (0.60–0.87)	0.79 (0.68–0.92)	0.71 (0.58–0.86)	.004
Model 3	1.00	0.79 (0.64–0.97)	0.73 (0.61–0.87)	0.80 (0.69–0.93)	0.71 (0.58–0.86)	.004
Model 4	1.00	0.79 (0.65–0.98)	0.73 (0.61–0.87)	0.81 (0.70–0.94)	0.72 (0.59–0.88)	.007
Males (n = 7,257) ⁺						
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)	.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)	.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)	.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)	.068
Females (n = 6,953) ⁺						
Model 1	1.00	0.71 (0.54–0.93)	0.64 (0.48–0.86)	0.72 (0.53–0.97)	0.56 (0.41–0.77)	.004
Model 2	1.00	0.73 (0.56–0.97)	0.67 (0.50–0.89)	0.77 (0.57–1.06)	0.65 (0.47–0.89)	.038
Model 3	1.00	0.75 (0.56–1.00)	0.67 (0.51–0.89)	0.79 (0.58–1.09)	0.67 (0.49–0.90)	.049
Model 4	1.00	0.74 (0.56–1.00)	0.67 (0.51–0.88)	0.80 (0.58–1.10)	0.67 (0.50–0.91)	.056
Dietary Inflammatory Index⁺						
All participants						
Model 1	1.00	1.10 (0.92–1.33)	1.29 (1.05–1.58)	1.49 (1.26–1.78)	1.70 (1.36–2.11)	< .001
Model 2	1.00	1.06 (0.87–1.28)	1.22 (0.99–1.50)	1.38 (1.15–1.66)	1.56 (1.24–1.96)	< .001
Model 3	1.00	1.06 (0.87–1.29)	1.24 (1.01–1.53)	1.40 (1.16–1.67)	1.58 (1.26–1.97)	< .001
Model 4	1.00	1.04 (0.85–1.28)	1.24 (1.00–1.53)	1.38 (1.14–1.66)	1.55 (1.24–1.94)	< .001
Male (n = 7,257) ⁺						
Model 1	1.00	1.25 (0.95–1.63)	1.36 (1.09–1.70)	1.50 (1.15–1.97)	1.50 (1.09–2.06)	.005
Model 2	1.00	1.21 (0.91–1.59)	1.32 (1.05–1.66)	1.43 (1.08–1.88)	1.43 (1.03–1.98)	.015
Model 3	1.00	1.23 (0.93–1.63)	1.35 (1.07–1.71)	1.49 (1.13–1.98)	1.49 (1.06–2.08)	.007
Model 4	1.00	1.23 (0.92–1.63)	1.37 (1.08–1.74)	1.47 (1.11–1.95)	1.46 (1.05–2.03)	.009
Females (n = 6,953) ⁺						
Model 1	1.00	0.98 (0.77–1.26)	1.16 (0.88–1.53)	1.22 (0.96–1.56)	1.94 (1.45–2.60)	< .001
Model 2	1.00	0.94 (0.73–1.21)	1.10 (0.84–1.45)	1.10 (0.86–1.41)	1.71 (1.27–2.31)	.001
Model 3	1.00	0.94 (0.72–1.22)	1.11 (0.84–1.46)	1.05 (0.81–1.37)	1.65 (1.23–2.23)	.002
Model 4	1.00	0.93 (0.72–1.22)	1.10 (0.84–1.45)	1.05 (0.81–1.36)	1.64 (1.22–2.20)	.002

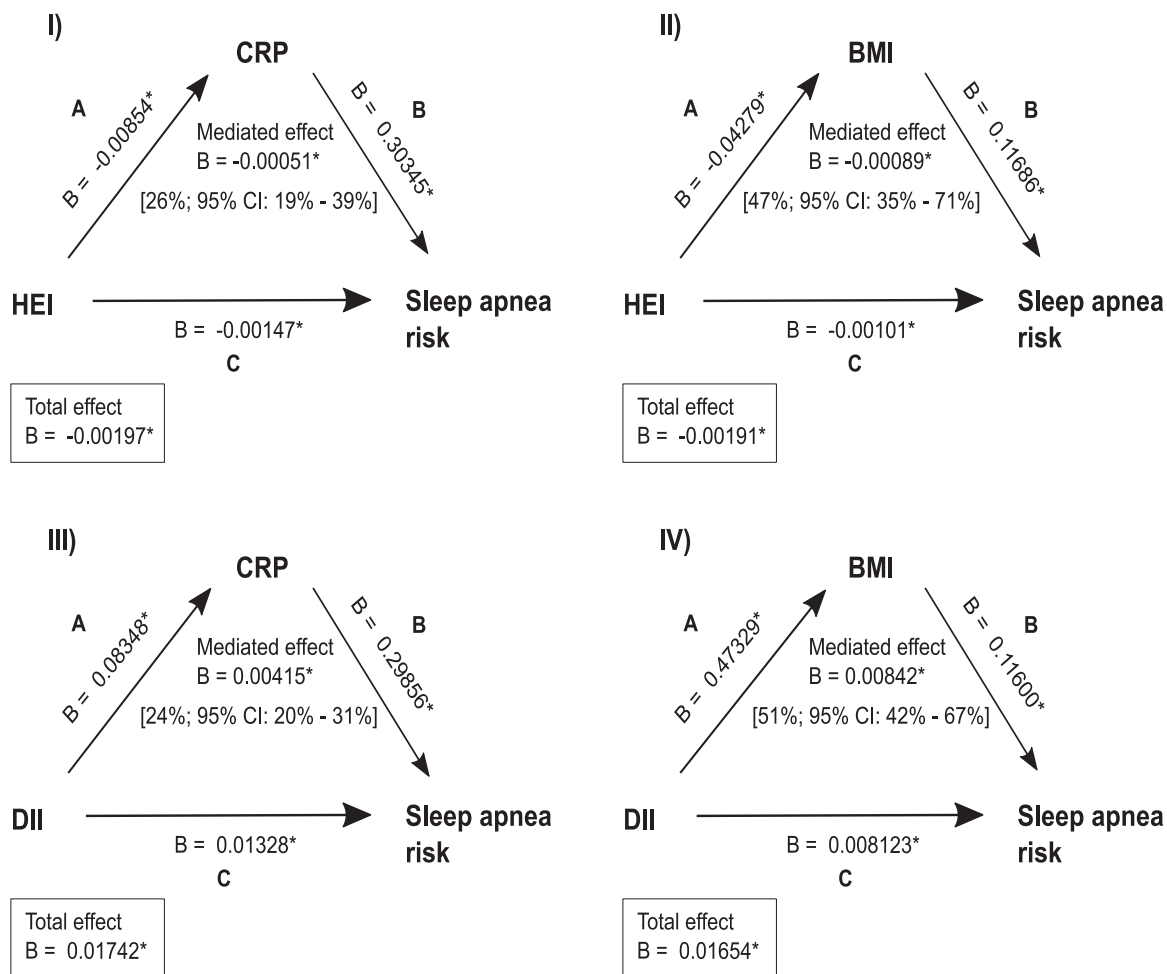
Sleep apnea was based on the STOP-BANG (Snoring, Tired, Observed apnea, Pressure [blood pressure], Body mass index, Age, Neck, Gender) tool. Only high-risk OSA cases were included. Model 1 was adjusted for sex, age, race, marital status, education, and income. Model 2 was additionally adjusted for smoking and physical activity. 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. [#]Models 2, 3, and 4 were additionally adjusted for alcohol consumption. ⁺Sex was not included in the models. CI = confidence interval, OSA = obstructive sleep apnea, Q = quintile.

(Snore, Tiredness, Hypertension, BMI, and waist circumference) were inversely associated with HEI and positively with DII (Table S5 in the supplemental material). The associations of HEI and DII and sleep apnea were consistent when the OSA-50 was used to define apnea risk (Table S6 in the supplemental material). Using the mean HEI and DII of 2-day dietary assessment data for those only with plausible energy intakes did not change the main findings (Table S7 and Table S8 in the supplemental material).

DISCUSSION

This large community-based study shows that, irrespective of caloric intake, higher overall dietary quality and an anti-inflammatory diet are inversely associated with sleep apnea. In addition, an overall healthy lifestyle (higher diet quality, higher physical activity level, not smoking, and optimal alcohol consumption) is associated with reduced sleep apnea risk. This association differs by sex, with a stronger association in

Figure 3—Association (β -coefficient) of HEI (I and II) and DII (III and IV) with sleep apnea and mediation effect of CRP (I and III) and BMI (II and IV).



HEI and DII were associated with both CRP (A) and sleep apnea risk (C, direct effect). CRP and BMI were also associated with sleep apnea (B). *Indicates significant association (all P values were $< .001$). BMI = body mass index, CI = confidence interval, CRP = C-reactive protein, DII = Dietary Inflammatory Index, HEI = Healthy Eating Index.

females. Several sensitivity analyses confirmed the robustness of these findings. This study underlines the potential benefit of improving overall diet quality and health behaviors beyond calorie restriction to support reductions in sleep apnea and the combined effects of OSA and poor diet and lifestyle habits on detrimental health consequences. Further, sex differences highlight that these more holistic lifestyle considerations are particularly important for women with sleep apnea risk, who have largely been overlooked in the sleep apnea literature to date.

Comparison with previous studies

Our findings show dose-dependent associations between the HEI and sleep apnea risk. Studies that investigate overall diet quality with sleep apnea risk in nonclinical samples are scarce. In the Men Androgens Inflammation Lifestyle Environment and Stress (MAILES) study of 784 men in Australia, no significant associations between sleep apnea determined with the AHI from polysomnography and a Western diet (characterized by

high intake of processed meat, snacks, red meat, and takeaway foods) and a “prudent” diet (high intake of vegetables, fruits, legumes, high-fiber bread, nuts, and fish) or a mixed dietary pattern were observed.³⁴ There may be several factors underlying these discrepant findings. Unlike our study, which used an a priori approach to examine overall diet quality (HEI), dietary patterns in the previous study³⁴ were determined using an a posteriori (data-driven) approach. Although a prudent dietary pattern might have a high correlation with the HEI, it might not necessarily reflect the overall dietary quality. In addition, unlike the current study, sleep apnea was objectively defined based on AHI.³⁴ Dietary associations appear to be smaller than several other lifestyle factors, so clearly one needs larger sample sizes.

In contrast to the current study, which used dietary patterns, previous studies^{14,35} used single nutrients and foods (which we instead report cumulatively incorporated in the HEI) to examine associations with sleep apnea. For instance, higher intake

Table 3—Association between healthy lifestyle score and sleep apnea.

All Participants					
Healthy lifestyle score [£]	0	1	2	3–4	P for Trend
Model 1	1.00	0.68 (0.54–0.85)	0.60 (0.50–0.72)	0.40 (0.31–0.51)	< .001
Model 2	1.00	0.68 (0.54–0.86)	0.62 (0.52–0.76)	0.42 (0.33–0.53)	< .001
Model 3	1.00	0.69 (0.55–0.87)	0.63 (0.52–0.77)	0.43 (0.34–0.54)	< .001
Components of healthy lifestyle [¥]	No	Yes	P Value		
Diet quality	1.00	0.91 (0.79–1.04)	.157		
Physical activity	1.00	0.73 (0.65–0.83)	< .001		
Optimal alcohol intake	1.00	0.76 (0.61–0.94)	.012		
Not smoking	1.00	0.73 (0.65–0.82)	.000		
Males [†]					
Healthy lifestyle score [£]	0	1	2	3–4	P for Trend
Model 1	1.00	0.79 (0.60–1.03)	0.66 (0.52–0.84)	0.49 (0.37–0.66)	< .001
Model 2	1.00	0.79 (0.60–1.05)	0.68 (0.53–0.86)	0.51 (0.38–0.69)	< .001
Model 3	1.00	0.80 (0.60–1.05)	0.69 (0.54–0.89)	0.53 (0.39–0.71)	< .001
Components of healthy lifestyle [¥]	No	Yes	P Value		
Diet quality	1.00	0.91 (0.77–1.07)	.227		
Physical activity	1.00	0.81 (0.68–0.96)	.018		
Optimal alcohol intake	1.00	0.80 (0.64–0.99)	.040		
Not smoking	1.00	0.75 (0.64–0.88)	.001		
Females [†]					
Healthy lifestyle score [£]	0	1	2	3–4	P for Trend
Model 1	1.00	0.57 (0.42–0.77)	0.54 (0.41–0.72)	0.31 (0.21–0.44)	< .001
Model 2	1.00	0.58 (0.43–0.78)	0.57 (0.43–0.77)	0.33 (0.23–0.46)	< .001
Model 3	1.00	0.58 (0.43–0.78)	0.58 (0.43–0.78)	0.33 (0.23–0.46)	< .001
Components of healthy lifestyle [¥]	No	Yes	P Value		
Diet quality	1.00	0.92 (0.75–1.12)	.400		
Physical activity	1.00	0.67 (0.56–0.79)	< .001		
Optimal alcohol intake	1.00	0.57 (0.38–0.85)	.007		
Not smoking	1.00	0.69 (0.58–0.82)	<.001		

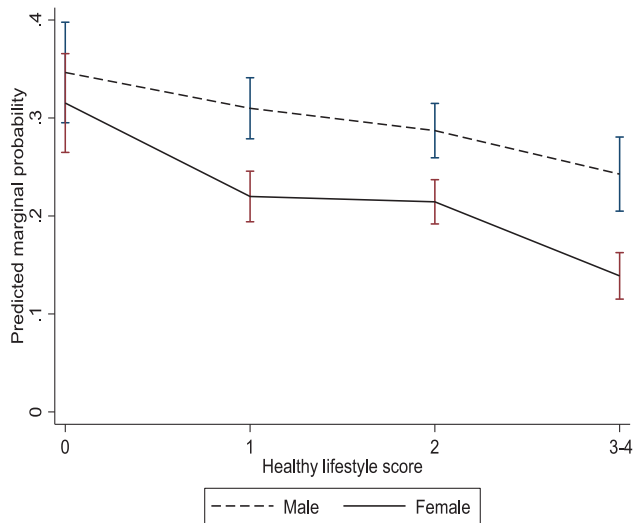
Values are presented as odds ratio (95% CI). Sleep apnea was based on STOP-BANG (Snoring, Tired, Observed apnea, Pressure [blood pressure], Body mass index, Age, Neck, Gender) score. Model 1 was adjusted for sex, age, race, marital status, education, and income. Model 2 was additionally adjusted for cardiovascular disease, cancer, and diabetes. Model 3 was additionally adjusted for sleep duration. [£]Healthy lifestyles include nonsmokers; exercise ≥ 600 MET-minutes/wk at moderate–vigorous intensity; diet in upper three-fifths of HEI; consuming moderate alcohol (moderate: 5–15 g alcohol/day in women, 5–30 g alcohol/day in men). For each of these lifestyles, score of 1 = healthy and 0 = unhealthy. The score ranges from 0 to 4; 0 indicates not a healthy lifestyle (most-unhealthy lifestyle) and 4 indicates an all-healthy lifestyle (most-healthy lifestyle). [¥]The estimates are based on model 3. Components of healthy lifestyle were mutually adjusted. [†]Sex was not included in the models. CI = confidence interval, HEI = Healthy Eating Index, MET = metabolic equivalent of task.

of fiber was associated with reduced OSA severity in a cross-sectional analysis of 75 patients.¹⁴ A positive association of high fat intake and increased AHI was also reported in the MAILES study.³⁵ Low fiber and high saturated fat and sugar intakes are also positively associated with arousals from sleep.³⁶ Broadly, these findings are in line with our results of inverse associations between an overall healthy diet with sleep apnea. However, foods and nutrients are clearly not consumed in isolation. Thus, approaches that reflect overall dietary patterns are likely to be more meaningful than studies of single foods or nutrients that may fail to adequately account for

synergetic effects of a mixed diet and uncontrolled dietary confounders on health outcomes.

Previous studies have also reported associations between diet and individual components of the STOP-BANG questionnaire. For instance, we have previously reported a positive association of saturated fat intake and daytime sleepiness in a community-based study,³⁷ and a similar finding was reported with total fat intake in another study.³⁵ In the present analysis, we extended these findings to sleep apnea risk and found an inverse association of overall diet quality and each individual STOP-BANG component, except for observed apnea during

Figure 4—Marginal probability of sleep apnea by sex and healthy lifestyle score.



Model was adjusted for age, race, marital status, education, income, cardiovascular disease, cancer, and diabetes and sleep duration. There was a strong interaction between unhealthy lifestyle and sex ($P = .0489$). Healthy lifestyles include nonsmokers; exercise ≥ 600 MET-minutes/week at moderate-vigorous intensity; diet in upper three-fifths of HEI; and consuming moderate alcohol (moderate: 5–15 g alcohol/day in women, 5–30 g alcohol/day in men). For each of these lifestyles, scores of 1 = healthy and 0 = unhealthy. The score ranges from 0 to 4; 0 indicates not a healthy lifestyle (most-unhealthy lifestyle) and 4 indicates an all-healthy lifestyle (most-healthy lifestyle). Sleep apnea was based on the STOP-BANG (Snoring, Tired, Observed apnea, Pressure [blood pressure], Body mass index, Age, Neck, Gender) tool. HEI = Healthy Eating Index, MET = metabolic equivalent of task.

sleep. A systematic review that included 10 randomized controlled trials reported that dietary interventions in patients with sleep apnea are effective in reducing sleep apnea severity.⁹ In our analysis, STOP-BANG and STOP scores treated as count variables are likely to be reasonable proxy measures of sleep apnea severity and demonstrated that improved diet quality (HEI) was associated with a lower score of sleep apnea. These results, in combination with previous findings, support the importance of healthier dietary changes to reduce sleep apnea risk and severity.

Unlike the current findings of a positive dose–response association between a proinflammatory diet and sleep apnea, an earlier study of 296 participants found that a proinflammatory diet was not associated with sleep apnea severity.¹⁵ These discrepant findings could reflect the much smaller sample size and the focus on a clinical sample. A study on systemic inflammation assessed using CRP during childhood and adolescence was prospectively associated with increased risk of OSA and found that the association of obesity with sleep apnea was mostly explained by inflammation.³⁸ In line with these previous findings, we demonstrated that one-quarter of the association between DII and sleep apnea risk was mediated by systemic inflammation also assessed using CRP. Given that the DII was positively associated with snoring independent of obesity in our

study, snoring-related inflammation may have a direct effect on sleep apnea risk. Indeed, this concept is supported by other lines of investigation. For example, sleep apnea severity improves in obese men with OSA when inflammation is treated.³⁹ Sleep apnea prevalence is also lower in patients with spondyloarthritis who receive treatment for inflammation.⁴⁰ Inflammation has also been proposed to be a contributing mechanism to OSA pathogenesis in people with multiple sclerosis.⁴¹

Our finding of positive associations of a proinflammatory diet with sleep apnea risk could also be partly explained by diet-induced inflammation in obesity, and particularly central obesity.⁴² This could increase the risk of sleep apnea directly¹⁰ or indirectly via increased systemic inflammation.³⁸ In our analysis, we found a positive association between DII and a high BMI (> 35 kg/m²), and that half of the association between DII and sleep apnea risk was mediated by BMI. Previous studies have demonstrated that use of continuous positive airway pressure without weight loss fails to improve markers of systemic inflammation.⁴³ Together, these findings support the important underlying effects of obesity-induced inflammation in the pathogenesis of sleep apnea.

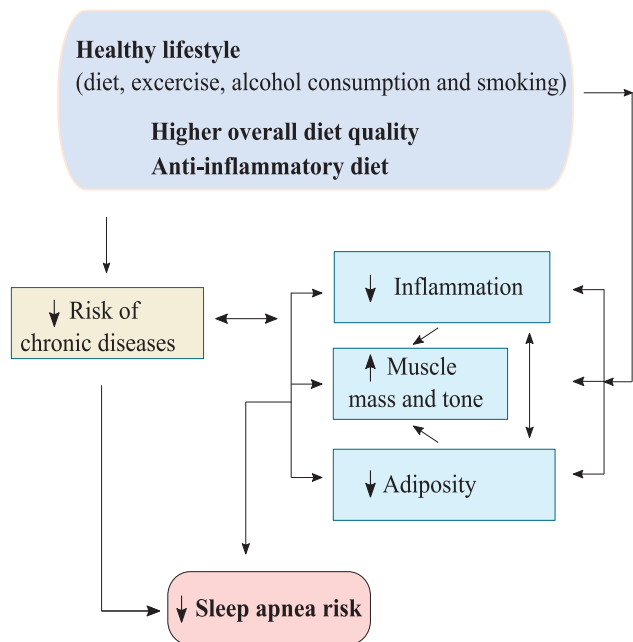
Consistent with the healthy lifestyle findings in the current study, a systematic review on the effectiveness of lifestyle interventions on OSA also showed that lifestyle changes effectively reduce sleep apnea severity.¹⁰ In the included studies, the interventions targeted reduced weight through diet, exercise, or a combination of the 2. The meta-analysis results demonstrated that diet alone, exercise alone, or the combination of the 2 were all effective in reducing BMI, AHI, and 4% oxygen desaturation index. A more recent systematic review further supports the importance of adopting a healthy lifestyle in reducing sleep apnea risk and severity.⁹

Previous studies focused mainly on weight loss through diet and exercise interventions and did not consider smoking and alcohol consumption,⁴⁴ despite their established importance for sleep apnea risk.^{45,46} Thus, lifestyle interventions that target risk, severity, and consequences of sleep apnea should clearly consider smoking and alcohol intake in addition to diet and exercise. Overall, the findings from the current study suggest that combinations of lifestyle interventions may be more important for sleep apnea management than individual interventions. Furthermore, and consistent with a previous study,⁴⁴ intervention impacts may also differ by sex and may require tailoring.

Potential mechanisms

Figure 5 depicts potential pathways through which a healthy lifestyle and a high-quality and anti-inflammatory diet could affect sleep apnea risk. The status of chronic conditions, systemic inflammation, adiposity, and muscle mass/tone and their interactions could play key roles in mediating the association of overall lifestyle and diet with sleep apnea risk. A higher-quality diet is associated with reduced risk of chronic diseases,⁴⁷ which are either directly linked to reduced risk of sleep apnea⁴⁸ or are indirectly linked through decreased systemic inflammation and adiposity and increased muscle mass/tone,⁴⁹ which eventually result in reduced sleep apnea.^{38,50–52} Similarly, a healthy lifestyle and high-quality and anti-inflammatory diets may directly reduce the risk of inflammation⁵³ and adiposity⁵⁴ and increase

Figure 5—Mechanism of action through which overall lifestyle and diet could affect sleep apnea.



muscle mass/tone,⁵¹ which may, again, reduce sleep apnea risk directly or indirectly through decreased risk of chronic diseases. Our study further supports that inflammation and adiposity are likely to play significant direct and indirect roles in relationships between diet and sleep apnea.

Strengths and limitations

A key strength of this study is the use of comprehensive diet and lifestyle risk factor data from a large community-based study that addresses clear gaps in sex-specific understanding of the relationship between diet and sleep apnea. We also applied multiple criteria to define sleep apnea and used extensive sensitivity analyses to confirm the consistency and robustness of our main findings. Despite these strengths, limitations of this study should be considered when interpreting the findings. We used dietary data from the first-day 24-hour recall in which day-to-day variability in food intake may not be accounted for and may lead to measurement bias. On the other hand, recall bias and omission of food groups are reduced with this method.⁵⁵ Our sensitivity analysis using the mean of the 2-day dietary data also showed consistent results. We did not consider nutrient intake from supplementation as these data were not available. In addition, although STOP-BANG and OSA-50 are well-validated tools with high sensitivity, discrimination of participants without moderate and severe sleep apnea (specificity) is more modest.²⁹ Therefore, future community-based research should investigate the associations of healthy lifestyle, HEI, and DII with sleep apnea presence and severity determined via overnight polysomnography. Furthermore, we used waist circumference instead of neck circumference as NHANES did not have this measurement. Last, as the findings of this study are

based on cross-sectional data and sleep apnea might affect food choice and intake,⁵⁶ it is not possible to establish causality.

CONCLUSIONS

In summary, healthy and anti-inflammatory diets, regardless of calorie intake, are significantly associated with reduced sleep apnea risk. In addition, overall healthy lifestyle factors are associated with reduced risk of sleep apnea. These findings, combined with established evidence,^{9,10} strongly support the importance of preventing and managing sleep apnea and its severity using dietary and overall lifestyle interventions separately and in combination. The promotion of a high-quality diet, anti-inflammatory foods and nutrients, and an overall healthy lifestyle is likely to help prevent sleep apnea and reduce adverse health consequences including premature mortality when used as both primary prevention and secondary intervention strategies. Clinicians and dietitians should recognize the role of diet and overall lifestyle modification in reducing sleep apnea risk and severity. Given that our associations were independent of calorie intake, holistic dietary modifications may also afford a more achievable and realistic approach for patients who do not engage well with calorie-restriction approaches.

Although previous studies have investigated the role of macronutrient intake in sleep apnea, further investigation using longitudinal studies and randomized controlled trials should be conducted and include rigorous analysis techniques necessary for dietary studies (eg, Melaku et al³⁷) to elucidate causal links. Specifically targeting macronutrient composition will be critical because dietary interventions that change overall dietary pattern and habits, and that target weight reduction through calorie restriction, often fail over the long term.⁵⁷ In addition, weight loss without improved systemic inflammation may not be effective in reducing risk and severity of sleep apnea.³⁸ In addition to diet quality, meal timing⁵⁸ could be an important factor in sleep apnea that needs further investigation.

First-line treatment of sleep apnea with continuous positive airway pressure is often associated with poor compliance and does not reduce the incidence of myocardial infarction, atrial fibrillation, heart failure, or all-cause mortality⁵⁹ or reduce secondary cardiovascular events.^{60,61} Thus, dietary interventions and adoption of an overall healthy lifestyle clearly remain important avenues needed to prevent adverse health outcomes of sleep apnea and to improve overall health and well-being of patients. This is particularly critical because these interventions more specifically target key underlying causes of sleep apnea and associated symptomatology, such as obesity and inflammation, rather than alleviating symptoms alone. The role of diet and an overall healthy lifestyle in reducing and mitigating risks, severity, and consequences of sleep apnea clearly warrants further investigation using randomized controlled trials and longitudinal studies.

ABBREVIATIONS

- AHI, apnea-hypopnea index
- BMI, body mass index

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CI, confidence interval
 CRP, C-reactive protein
 CVD, cardiovascular disease
 DII, Dietary Inflammatory Index
 HEI, Healthy Eating Index
 MET, metabolic equivalent of task
 NHANES, National Health and Nutrition Examination Survey
 OSA, obstructive sleep apnea
 STOP-BANG, Snoring, Tired, Observed apnea, Pressure, Body mass index, Age, Neck, Gender

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