BMC Nutrition

RESEARCH

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Dietary inflammatory potential exacerbates sleep disturbances in hyperlipidemia: mediation by BMI and neuro-metabolic pathways

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Abstract

Background Hyperlipidemia is closely associated with chronic low-grade inflammation and metabolic disorders. The Dietary Inflammatory Index (DII) reflects the pro-inflammatory potential of the diet, but its association with sleep disorders among individuals with hyperlipidemia remains unclear. This study aims to explore the association between DII and sleep disorders in adults with hyperlipidemia and analyze the mediating effect of body mass index (BMI).

Methods Data from 13,195 participants with hyperlipidemia from the National Health and Nutrition Examination Survey (NHANES) in the United States (2007–2018) were included. Sleep disorders were defined based on self-reported physician diagnosis. DII scores were calculated based on 22 dietary components. Weighted multivariable logistic regression, restricted cubic splines, curve fitting models, and threshold analysis were used to evaluate the association between DII and sleep disorders. Finally, the mediating effect of BMI was analyzed.

Results A total of 13,195 participants were included, of whom 1,598 reported sleep disorders. After adjusting for all covariates using weighted logistic regression, each 1-unit increase in DII was associated with a 5.7% higher prevalence of sleep disorders (odds ratio [OR] = 1.057, 95% confidence interval [CI]: 1.012–1.105). When DII was categorized into four groups, the OR for Q4 versus Q1 was 1.295 (95% CI: 1.039–1.614, $P = 0.0269$), with a significant trend (trend test $P < 0.05$). Restricted cubic spline and curve fitting models showed a linear relationship between DII and sleep disorder prevalence. Subgroup analysis indicated a stronger association between DII and sleep disorders among participants with hyperlipidemia who were under 60 years old. Mediation analysis further revealed that BMI mediated 26.07% of the association between DII and sleep disorders ($P < 0.05$).

Conclusion The results of this cross-sectional study show a positive association between pro-inflammatory diets and the risk of sleep disorders in patients with hyperlipidemia. A significant trend was observed across the quartiles of the Dietary Inflammatory Index (DII). The adjusted odds ratio (OR) for the highest quartile (Q4) compared with the lowest quartile (Q1) was 1.295 (95% confidence interval [CI]: 1.039–1.614, $P = 0.0269$), which was particularly evident in the

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young and middle-aged population (< 60 years old). Reducing the dietary inflammatory index may improve sleep health through weight loss (mediated by BMI) and direct anti-inflammatory effects. Future interventional studies are needed to clarify the causal impact of dietary inflammation regulation on sleep quality and the underlying molecular mechanisms, so as to provide a scientific basis for precision nutritional intervention.

Keywords Dietary inflammatory index, Sleep disturbances, Hyperlipidemia, Body mass index, Mediation effect

Introduction

As a key risk factor for metabolic disorders, the increasing prevalence of hyperlipidemia is closely related to a sedentary lifestyle and an obesogenic dietary patter [1, 2]. Chronic low-grade inflammation is a core mechanism connecting metabolic dysregulation and systemic complications. The DII, as a tool for quantifying the pro-inflammatory potential of diets, has research results suggesting a significant association with obesity [3], insulin resistance [4], and cardiovascular diseases [5, 6]. It is worth noting that patients with hyperlipidemia are more sensitive to dietary inflammation due to abnormal lipid metabolism and vascular endothelial dysfunction [7–9]. However, the interaction between their dietary inflammation levels and extra-metabolic health outcomes such as sleep remains unclear.

Sleep disorders (including poor sleep quality and insufficient sleep duration) are a growing public health problem, with more than 30% of the global population affected by sleep problems, including insomnia, sleep-disordered breathing, and poor sleep quality [10]. Inflammatory pathways play an important role in sleep disorders, and pro-inflammatory cytokines can interfere with the circadian rhythm and the function of the hypothalamic-pituitary-adrenal axis [11, 12]. The prevalence of sleep disorders is significantly higher in people with hyperlipidemia due to lipid-mediated neuroinflammation and abnormal vascular function. However, whether dietary inflammation affects sleep by regulating inflammatory pathways, and the bidirectional relationship among diet, metabolism, and sleep architecture still need to be elucidated.

Body mass index, as a modifiable anthropometric index, may be a key intermediate factor. Adipose tissue, as an endocrine organ, secretes inflammatory adipokines, and obesity-related mechanical factors (such as obstructive sleep apnea) further exacerbate sleep disorders [13, 14]. A pro-inflammatory diet affects sleep health by promoting adipogenesis and visceral fat accumulation. However, no study has systematically explored the mediating role of BMI in the relationship between DII and sleep disorders in people with hyperlipidemia. Clarifying this mechanism is of great clinical significance for the screening of management targets for comorbidities.

Based on the cross-sectional data from the NHANES in the United States, this study aims to: (1) analyze the association between DII and sleep disorders in adults with hyperlipidemia; and (2) quantify the mediating effect of BMI in the above association. By means of the well-established biomarker detection and dietary recall system of NHANES, this study uses statistical models to investigate the potential pathway through which dietary inflammation may influence sleep health via BMI, aiming to address a gap in understanding regarding nutritional intervention, metabolic diseases, and sleep improvement in high-risk populations.

Method

Research design and study subjects

This cross-sectional study used data from the NHANES 2007–2018 to explore the possible associations among the DII, BMI, and sleep disorders. NHANES provides samples representative of the entire U.S. population, covering extensive data related to health, nutrition, and demographics. The survey adopted a complex multistage probability cluster sampling method. All study participants signed informed consent forms, and this study was approved by the Research Ethics Review Board of the National Center for Health Statistics. Figure 1 shows the participant screening process: initially, 59,842 participants were included, and they were gradually excluded due to no diagnosis of hyperlipidemia, incomplete data on sleep disorders and DII scores, missing demographic characteristics, incomplete laboratory data, missing questionnaire information, etc. Finally, 13,195 participants were included.

Assessment of sleep disorders

In this study, sleep disorders were assessed using the NHANES question: "Have you ever been told by a doctor or other health professional that you have a sleep disorder?" Participants answering "yes" were classified as having a sleep disorder; those answering "no" were classified as not having a sleep disorder. Participants answering "refuse to answer", "don't know", or with missing data were excluded from the analysis. Trained interviewers administered this question in participants' homes using the Computer-Assisted Personal Interview (CAPI) system. The CAPI system incorporates built-in consistency checks to minimize data entry errors, and online help screens assisted interviewers in understanding questionnaire terminology.

It is important to acknowledge that this measure relies on self-reported, clinician-diagnosed sleep disorders

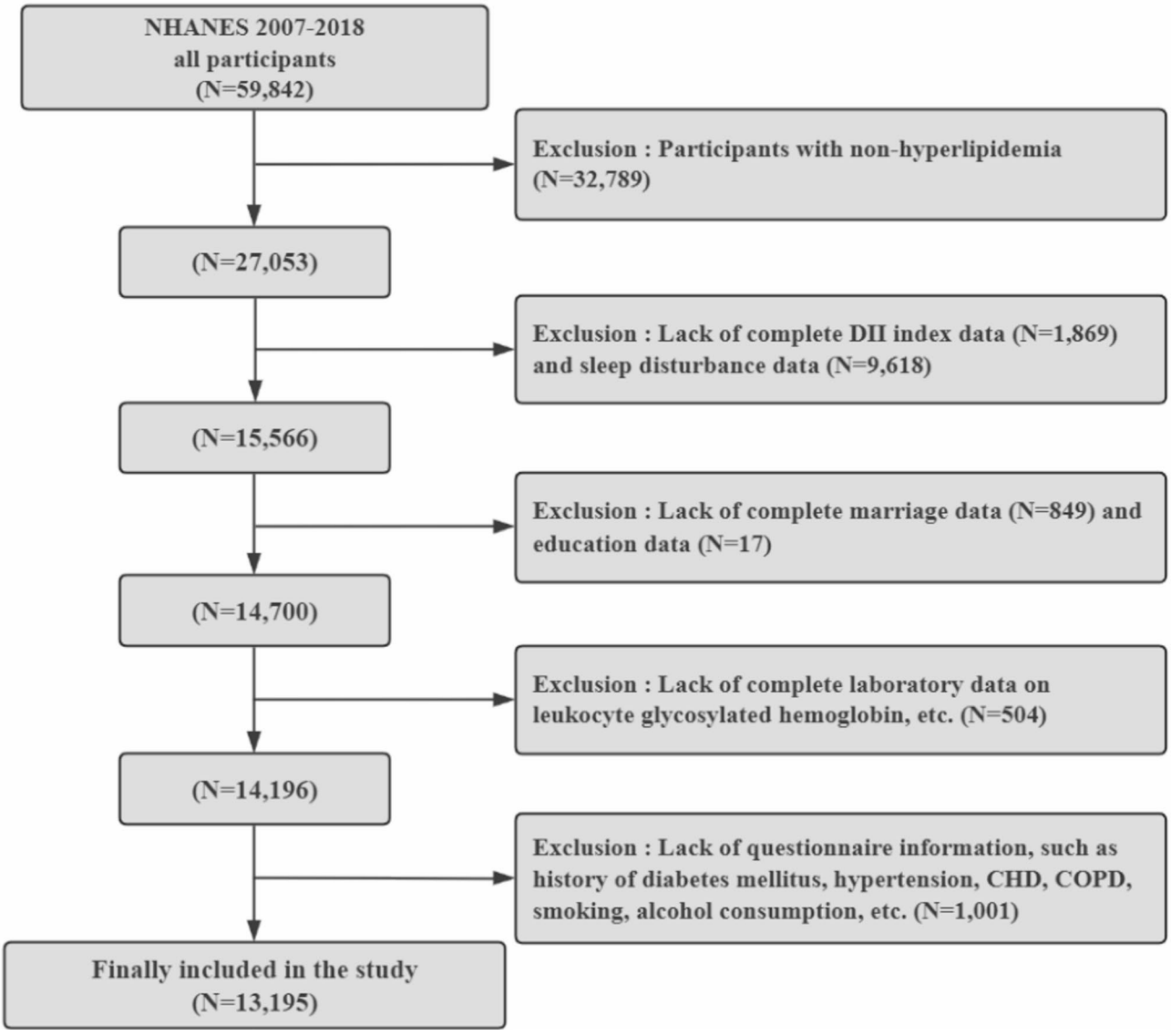

Fig. 1 Flow chart

captured by a single yes/no question. While this specific question has been used in multiple published studies [15, 16], this approach has inherent limitations: (1) Recall and Classification Bias: Reliance on participant recall of a clinician's diagnosis is susceptible to memory inaccuracies and potential misunderstanding of the diagnosis provided.(2) Broad Categorization: The term "sleep disorder" encompasses a wide range of conditions (e.g., insomnia, sleep apnea, restless legs syndrome). This single-item measure cannot differentiate between specific types of sleep disorders, which may have distinct etiologies and relationships with diet and metabolism.(3) Under-diagnosis: Individuals with undiagnosed sleep disorders are necessarily classified as "no" sleep disorder, potentially leading to misclassification.

DII assessment

The DII score is used to describe the level of dietary inflammation and was initially developed based on 45 dietary variables. For this analysis, we calculated the DII for each participant using dietary data from NHANES. We extracted 22 nutrients (alcohol, vitamin B_{12}, vitamin B_6, caffeine, carbohydrates, cholesterol, energy, total fat, dietary fiber, folic acid, iron, monounsaturated fatty acids (MUFA), polyunsaturated fatty acids (PUFA), saturated fatty acids (SFA), niacin, protein, vitamin A, vitamin C, vitamin D, vitamin E, zinc, and vitamin B_2) to calculate the DII. The calculation method of DII is to subtract the extracted nutrient levels from the global average intake and then divide by the standard deviation of the global average intake. This value is converted into a centered percentile score to minimize the impact of

"right-skewness". Multiply the centered percentile of each nutrient by its respective inflammation score, and finally sum up these new values to obtain the overall DII index for each participant. A higher DII score indicates a higher level of dietary inflammation; a lower score indicates a lower level of dietary inflammation [17, 18].

Covariates

Based on previous research and clinical considerations, this study included the following potential covariates affecting the association between DII and sleep disorders, categorized as:

(1) Demographic and socioeconomic factors: Age, sex (male/female), ethnicity (Non-Hispanic White, Non-Hispanic Black, Mexican American, Other), educational attainment (< high school, high school graduate, >high school), marital status (living with partner, living without partner), and poverty-income ratio (PIR).
(2) Lifestyle and behavioral factors: Smoking status (never, former, current), alcohol consumption (never, mild, moderate, heavy, former), and physical activity (assessed by walking/bicycling habits: yes/no).
(3) Clinical variables and comorbidities: BMI (kg/m^2), hypertension (yes/no), diabetes mellitus (including impaired fasting glucose [IFG] and impaired glucose tolerance [IGT]; yes/no), coronary heart disease (CHD; yes/no), history of stroke (yes/no), chronic obstructive pulmonary disease (COPD; yes/no), white blood cell count (WBC), and glycated hemoglobin (HbA1c).

Statistical analysis

Statistical analysis of the study was performed using R software (version 4.4.3). The study analyzed NHANES data from six cycles spanning 2007 to 2018. All analyses utilized sampling weights and incorporated primary sampling units and stratification factors to ensure the national representativeness of the results. Continuous variables were expressed as mean ± standard deviation, and *p*-values were obtained through t-tests. Percentages of categorical variables (presented as weighted N, %) and their associated *p*-values were calculated through weighted chi-square tests.A weighted multivariate logistic regression model was used to analyze the relationship between DII and sleep disorders, as well as the relationship between DII and BMI. Both DII and BMI were categorized into quartiles for analysis. Three models were constructed: (1) Model 1: An unadjusted model (without covariates).(2) Model 2: A model adjusted for age, sex, and ethnicity.(3) Model 3: A model adjusted for all covariates: age, sex, ethnicity, marital status, PIR, education, smoking, alcohol, physical activity, WBC, HbA1c, hypertension, diabetes, stroke, CHD, and COPD.Multicollinearity among covariates was evaluated using variance inflation factors (VIF). A VIF < 5 indicated an acceptable level, and no significant collinearity was found among the covariates in this study (Table S1).

Restricted cubic splines (RCS), curve fitting models, and threshold effect models were used to explore potential nonlinear relationships and threshold effects between DII (as a continuous variable) and sleep disorders. Subgroup analyses were conducted to further examine the association between DII and sleep disorders in different populations.Mediation analysis was performed to evaluate BMI's role as a mediator in the DII-sleep disorders pathway, estimating the indirect effect, direct effect, total effect, and the mediation proportion calculated as (indirect effect / total effect) × 100%. The "mediation" package in R was used. A two-sided *p*-value less than 0.05 was considered statistically significant.

Results

Baseline characteristics of the study population

Table 1 shows that a total of 13,195 participants were finally included in this study, divided into a non-sleep disorder group (N = 11,897) and a sleep disorder group (N = 1,298). Comparative analyses indicated that participants in the sleep disorder group were older (mean age: 52.04 ± 17.12 vs. 55.13 ± 14.62, P < 0.001), with higher glycated hemoglobin (5.84 ± 1.11 vs. 6.10 ± 1.23, P < 0.001), a higher proportion of males (51.69%), and higher BMI (29.52 ± 6.45 vs. 33.90 ± 8.11, P < 0.001). It is noteworthy that the mean DII was also higher in the sleep disorder group (1.57 ± 1.87 vs. 1.77 ± 1.86, P < 0.001). The prevalence of hypertension was significantly higher in the sleep disorder group (64.25% vs. 35.75%, P < 0.001). No significant differences were found in marital status or income.

Association between DII, BMI and sleep disorders

Table 2 presents the associations of DII and BMI quartiles with sleep disorders across three logistic regression models. DII and BMI were categorized into quartiles (Q1–Q4), with Q1 as the reference group. Results from the three logistic regression models show that higher quartiles of both DII and BMI consistently indicate increased odds of sleep disorders, and the highest quartile (Q4) remains statistically significant across all models.

(1) DII Associations: In the unadjusted model, individuals in the highest DII quartile (Q4) had 37% higher odds of sleep disorders than Q1 (OR = 1.371; 95% CI: 1.137–1.654; P = 0.0016). After adjusting for age, sex, and ethnicity (Model 2), this association strengthened to 48% higher odds (OR = 1.477; 95% CI: 1.201–1.817; P = 0.0005). In the fully adjusted model (Model 3), a 30% significant increase in

Table 1 Characteristics of the participant population

Demographic Factor	Without Sleep disorder (*N* = 16,480)	Sleep disorder (*N* = 1,598)	*P*-value
Age, mean(SD)	49.16 ± 17.78	53.54 ± 15.21	< 0.001
Poverty, mean(SD)	2.54 ± 1.57	2.46 ± 1.59	0.045
WBC, mean(SD)	7.14 ± 2.40	7.56 ± 2.83	< 0.001
HBA1C, mean(SD)	5.73 ± 1.04	6.00 ± 1.17	< 0.001
TG, mean(SD)	153.65 ± 122.66	169.85 ± 135.97	< 0.001
HDL, mean(SD)	52.57 ± 15.81	49.17 ± 15.16	< 0.001
BMI, mean(SD)	28.70 ± 6.52	33.16 ± 8.29	< 0.001
DII, mean(SD)	1.51 ± 1.89	1.69 ± 1.88	< 0.001
SEX n(%)			0.09
Female	8245 (50.03%)	764 (47.81%)	
Male	8235 (49.97%)	834 (52.19%)	
Ethical n(%)			< 0.001
Non-Hispanic White	7511 (45.58%)	869 (54.38%)	
Non-Hispanic Black	3277 (19.88%)	324 (20.28%)	
Mexican American	2555 (15.50%)	149 (9.32%)	
Other Race	3137 (19.04%)	256 (16.02%)	
MARITAL n(%)			0.682
Living with partner	9812 (59.54%)	943 (59.01%)	
Living without partner	6668 (40.46%)	655 (40.99%)	
Education			0.013
< High school	4157 (25.22%)	350 (21.90%)	
Completed high school	3764 (22.84%)	376 (23.53%)	
>High school	8559 (51.94%)	872 (54.57%)	
Smoke n(%)			< 0.001
Never	9135 (55.43%)	706 (44.18%)	
Former	3942 (23.92%)	507 (31.73%)	
Now	3403 (20.65%)	385 (24.09%)	
Alcohol n(%)			< 0.001
never	2297 (13.94%)	187 (11.70%)	
mild	5365 (32.55%)	503 (31.48%)	
moderate	2486 (15.08%)	224 (14.02%)	
heavy	3462 (21.01%)	266 (16.65%)	
former	2870 (17.42%)	418 (26.16%)	
Hypertension n(%)			< 0.001
No	9808 (59.51%)	626 (39.17%)	
Yes	6672 (40.49%)	972 (60.83%)	
Diabetes n(%)			< 0.001
no	12,211 (74.10%)	971 (60.76%)	
yes	2833 (17.19%)	493 (30.85%)	
IFG	672 (4.08%)	73 (4.57%)	
IGT	764 (4.64%)	61 (3.82%)	
Hyperlipidemia n(%)			< 0.001
No	4583 (27.81%)	300 (18.77%)	
Yes	11,897 (72.19%)	1298 (81.23%)	
CHD n(%)			< 0.001
No	15,887 (96.40%)	1461 (91.43%)	
Yes	593 (3.60%)	137 (8.57%)	
COPD n(%)			< 0.001
No	15,599 (94.65%)	1402 (87.73%)	
Yes	881 (5.35%)	196 (12.27%)	
Stroke n(%)			< 0.001
No	15,906 (96.52%)	1473 (92.18%)	

Table 1 (continued)

Demographic Factor	Without Sleep disorder (*N* = 16,480)	Sleep disorder (*N* = 1,598)	*P*-value
Yes	574 (3.48%)	125 (7.82%)	
Walk/bicycle n(%)			< 0.001
No	12,090 (73.36%)	1257 (78.66%)	
Yes	4390 (26.64%)	341 (21.34%)	

Table 2 Weighted logistic regression model of DII indexes and BMI values and sleep disorders

Exposure	Model1	Model2	Model3
	OR(95%CI) *P*-value	OR(95%CI) *P*-value	OR(95%CI) *P*-value
DII	1.070 (1.030, 1.112) 0.0010	1.087 (1.042, 1.134) 0.0003	1.057 (1.012, 1.105) 0.0176
DII quartile			
Q1	***Ref.***	***Ref.***	***Ref.***
Q2	0.959 (0.802, 1.147) 0.6487	0.985 (0.823, 1.180) 0.8708	0.920 (0.769, 1.101) 0.3700
Q3	1.225 (0.989, 1.516) 0.0680	1.290 (1.040, 1.599) 0.0239	1.166 (0.940, 1.445) 0.1700
Q4	1.371 (1.137, 1.654) 0.0016	1.477 (1.201, 1.817) 0.0005	1.295 (1.039, 1.614) 0.0269
P for Trend	1.128 (1.056, 1.204) 0.0006	1.156 (1.076, 1.242) 0.0002	1.107 (1.026, 1.196) 0.0128
BMI	1.079 (1.068, 1.091) < 0.0001	1.087 (1.075, 1.100) < 0.0001	1.080 (1.067, 1.093) < 0.0001
BMI quartile			
Q1	***Ref.***	***Ref.***	***Ref.***
Q2	1.113 (0.781, 1.586) 0.5550	1.067 (0.750, 1.519) 0.7183	1.105 (0.775, 1.575) 0.5857
Q3	1.733 (1.311, 2.290) 0.0003	1.675 (1.268, 2.213) 0.0006	1.645 (1.243, 2.176) 0.0013
Q4	3.978 (2.978, 5.312) < 0.0001	4.190 (3.124, 5.619) < 0.0001	3.781 (2.777, 5.148) < 0.0001
P for Trend	1.705 (1.559, 1.866) < 0.0001	1.754 (1.597, 1.928) < 0.0001	1.661 (1.507, 1.831) < 0.0001

Q: quartiles; OR: odds ratio; CI: confidence interval; Ref: reference.

Model1: Crude

Model2: Adjust: Age, Sex, Ethnicity

Model3: Adjust: Age, Sex, Ethnicity; marital; poverty; education; smoke; alcohol; hypertension; diabetes; stroke; walk_bicycle; COPD; CHD; WBC; HBA1C;

Table 3 Linear relationship between DII index and BMI value

Exposure	Model1 β (95%CI) *P*-value	Model2 β (95%CI) *P*-value	Model3 β (95%CI) *P*-value
DII	0.290 (0.229, 0.352) <0.00001	0.214 (0.152, 0.275) <0.00001	0.148 (0.088, 0.209) <0.00001

CI: confidence interval;

Model1: Crude

Model2: Adjust: Age, Sex, Ethnicity

Model3: Adjust: Age, Sex, Ethnicity; marital; poverty; education; smoke; alcohol; hypertension; diabetes; stroke; walk_bicycle; COPD; CHD; WBC; HBA1C

odds remained (OR = 1.295; 95% CI: 1.039–1.614; $P = 0.0269$).

(2) BMI Associations: In the unadjusted model, the highest BMI quartile (Q4) showed 3.978-fold higher odds of sleep disorders versus Q1 (OR = 3.978; 95% CI: 2.978–5.312; $P < 0.0001$). After full adjustment (Model 3), Q4 individuals still exhibited 3.781-fold higher odds (OR = 3.781; 95% CI: 2.777–5.148; $P < 0.0001$).

(3) As shown in Table 3, the relationship between DII and BMI: All models demonstrated a consistent positive correlation between DII and BMI ($P < 0.05$). In Model 3, each 1-unit increase in DII was associated with a 0.148-unit increase in BMI (β = 0.148; 95% confidence interval: 0.088–0.209; $P < 0.05$). A similar trend was also observed in the curve-fitting model (Fig. 2).

Nonlinear analysis of the relationship between DII and sleep disorders

Findings from the restricted cubic spline analysis (depicted in Fig. 3A) and the curve fitting model (illustrated in Fig. 3B) suggested a linear relationship between the DII and sleep disorders, as evidenced by a non-linearity *P*-value greater than 0.05. Threshold effect analysis, as shown in Table 4, determined that in Model II, the inflection point (K) was 4.215. When DII was less than K, the β value (95% CI) of Effect 1 was 1.034 (0.999, 1.071) with a *P* value of 0.0593. When DII exceeded K, the β value (95% CI) of Effect 2 was 2.057 (1.006, 4.203) and the *P* value

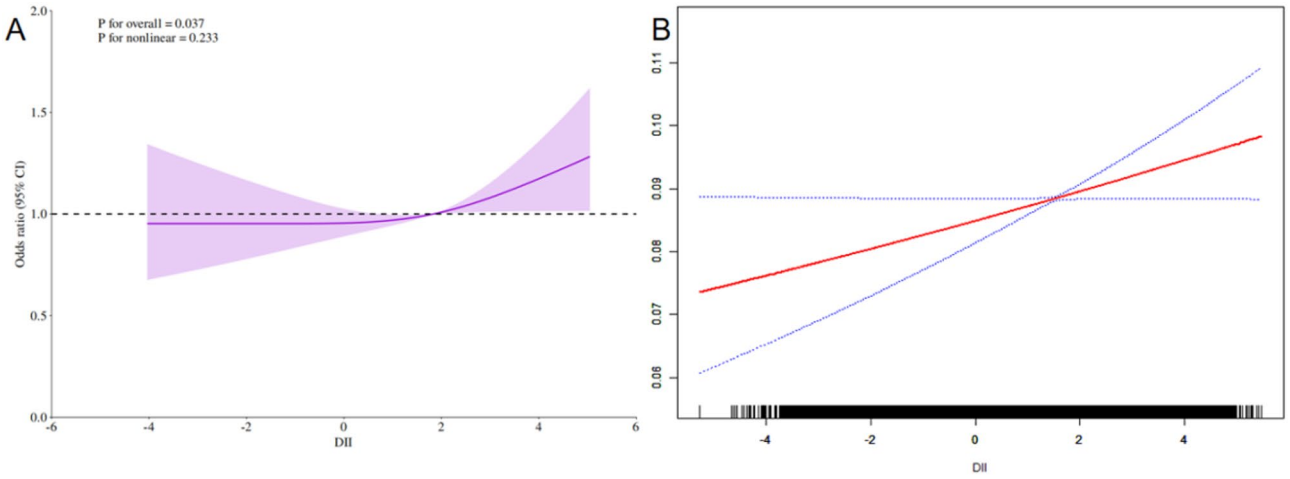

Fig. 2 Correlation between DII index and BMI index. *Adjusted Age, Sex, Ethnicity; marital; poverty; education; smoke; alcohol; hypertension; diabetes; stroke; walk_bicycle; COPD; CHD; WBC; HBA1C*

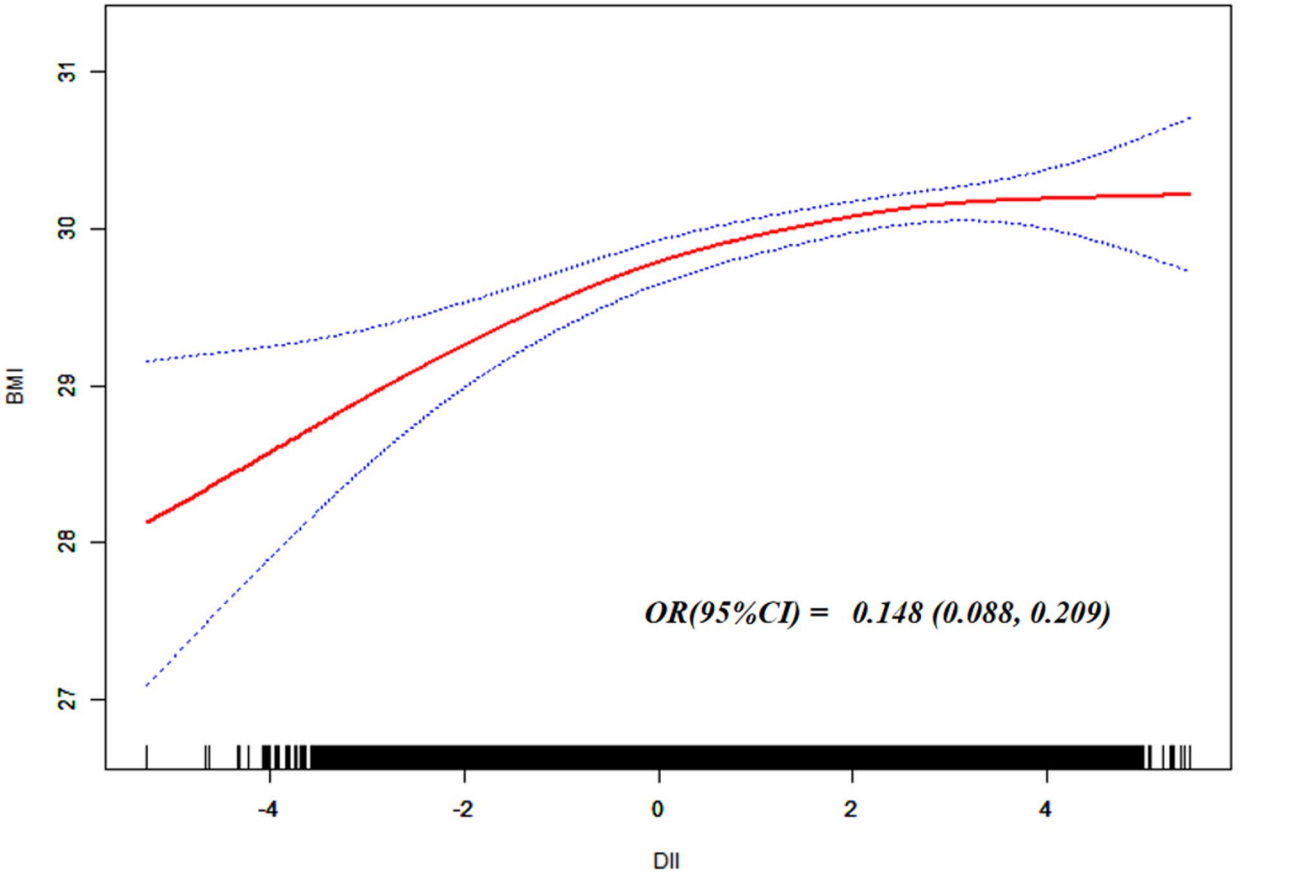

Fig. 3 RCS model and curve fitting model. **A**: Restriction cubic spline model of DII index and sleep disorders. **B**: Curve fitting model of DII index and sleep disorder. *All models adjust for Age, Sex, Ethnicity; marital; poverty; education; smoke; alcohol; hypertension; diabetes; stroke; walk_bicycle; COPD; CHD; WBC; HBA1C*

Table 4 Threshold effect analysis of DII indicators and outcome variables

Exposure:	SLEEP
Model II	
Breakpoint (K)	4.215
Effect 1 for the segment < K	1.034 (0.999, 1.071) 0.0593
Effect 2 for the segment > K	2.057 (1.006, 4.203) 0.0480
Log-likelihood ratio test	0.072

Model II: Adjust: Age, Sex, Ethnicity; marital; poverty; education; smoke; alcohol; hypertension; diabetes; stroke; walk_bicycle; COPD; CHD; WBC; HBA1C

Table 5 Subgroup analysis

Variable	*N*%	OR (95%CI)	*P*	*P* for interaction
Sex				
female	6726	1.079 (1.029, 1.133)	0.0018	0.601
male	6469	1.063 (1.018, 1.110)	0.0053	
Age				0.5641
<40	3454	1.099 (1.017, 1.187)	0.0176	
40–60	4661	1.079 (1.027, 1.134)	0.0027	
≥60	5080	1.038 (0.990, 1.089)	0.1204	
Ethical				**0.0198**
Non-Hispanic White	6316	1.062 (1.018, 1.107)	0.0053	
Non-Hispanic Black	2405	0.982 (0.915, 1.054)	0.6101	
Mexican American	2026	1.065 (0.962, 1.180)	0.2264	
Other Race	2448	1.142 (1.054, 1.238)	0.0012	
Marital				0.4685
Living with partner	8138	1.072 (1.029, 1.116)	0.0008	
Living without partner	5057	1.042 (0.991, 1.095)	0.1073	
Hypertension				0.1568
no	6863	1.091 (1.035, 1.149)	0.0011	
yes	6332	1.030 (0.990, 1.071)	0.149	
COPD				0.5775
no	12,306	1.054 (1.020, 1.090)	0.0019	
yes	889	1.064 (0.972, 1.165)	0.1785	
CHD				0.5074
no	12,510	1.063 (1.028, 1.098)	0.0003	
yes	685	1.030 (0.929, 1.141)	0.5786	
Stroke				0.1757
no	12,588	1.060 (1.026, 1.096)	0.0004	
yes	607	0.975 (0.870, 1.092)	0.6603	
WALK/BICYCLE				0.2301
no	10,025	1.046 (1.010, 1.084)	0.0116	
yes	3170	1.104 (1.030, 1.183)	0.0053	

was 0.0480. The P value of the log-likelihood ratio test was 0.072, suggesting that under this model, there was a linear association between the Dietary Inflammatory Index (DII) and sleep status.

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Subgroup analyses, presented in Table 5, were carried out based on factors such as sex, age, ethnicity, marital status, hypertension, COPD, CHD, stroke, and walking/bicycling habits. These analyses uniformly demonstrated a significant positive correlation between the DII index and sleep disorders across all subgroups. An interaction was observed in the ethnic subgroup, while the remaining subgroups showed stability, with no significant interactions (interaction *P*-values > 0.05). Results of the subgroup analyses further indicated that the relationship between the DII index and sleep disorders was more pronounced among individuals under 60 years of age and those with hyperlipidemia. This implies that additional dietary intervention and control strategies may be necessary.

Mediating effect

In the mediating model depicted in Fig. 4, the DII served as the independent variable, sleep disorders functioned as the dependent variable, and BMI acted as the mediating

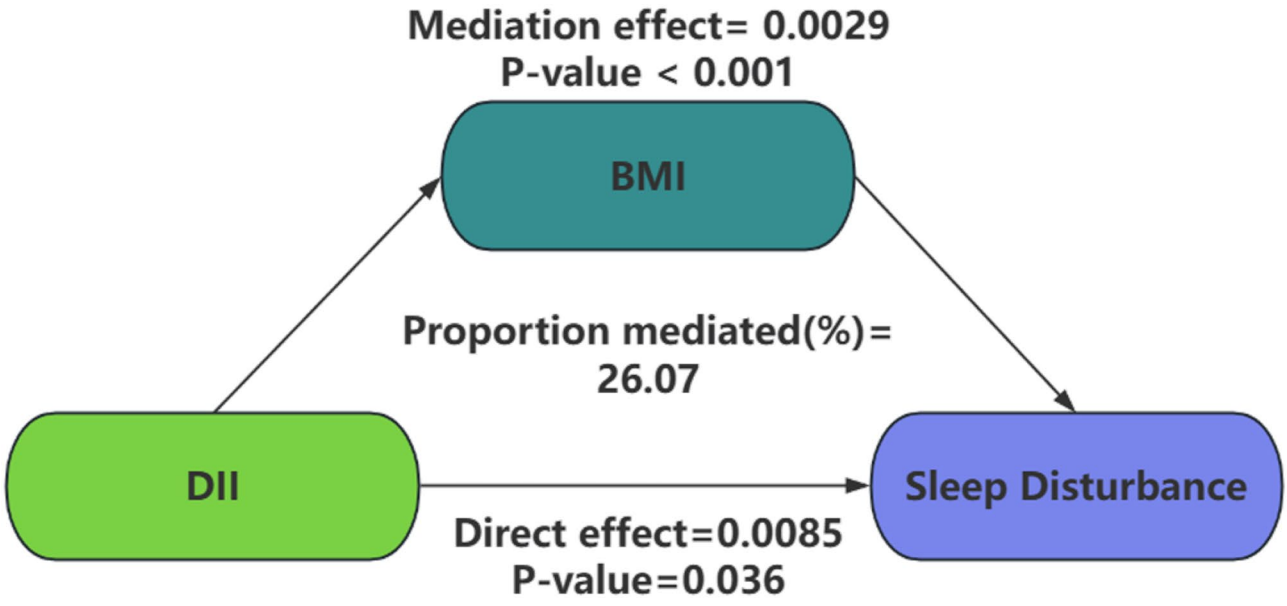

Fig. 4 Mediation analysis chart. *Adjusted Age, Sex, Ethnicity; marital; poverty; education; smoke; alcohol; hypertension; diabetes; stroke; walk_bicycle ; COPD; CHD; WBC; HBA1C*

Table 6 Mediation analysis

	Estimate	95% CI lower	95% CI upper	*P*-value
Total effect	0.011279	0.003152	0.019727	0.0060
Mediation effect	0.002941	0.001742	0.004115	< 0.0001
Direct effect	0.008517	0.000742	0.017103	0.0360
Propotion mediated	0.260710	0.126477	0.775937	0.0060

variable. As presented in Table 6 and illustrated in Fig. 4, following the adjustment for various covariates, a significant association was identified between BMI and sleep disorders. The overall effect value of the DII index was measured at 0.011279. After applying the adjustments for all covariates as per Model 3, the mediating role of BMI became evident. The indirect effect was determined to be 0.002941 ($P < 0.001$), while the direct effect was 0.008517 ($P < 0.036$). Calculations revealed that the proportion of the mediating effect accounted for 26.07% ($P = 0.006$).

Discussion

By analyzing data from 13,195 participants with hyperlipidemia in the NHANES, this study found a potential association between the DII and sleep disorders. Each 1-unit increase in DII was associated with a 5.7% increase in the prevalence of sleep disorders (OR = 1.057, 95% CI: 1.012, 1.105). Additionally, this association showed a potential linear trend in both DII quartile grouping and restricted cubic spline analysis. Mediation effect analysis indicated that BMI might play a partial mediating role in the association between DII and sleep disorders, with approximately 26.07% of the effect possibly being mediated by BMI ($P < 0.05$). Subgroup analysis further revealed that the association was more significant in individuals under 60 years old and those with comorbid hyperlipidemia. These findings provide key evidence for dietary intervention and sleep health management in hyperlipidemia patients, suggesting that reducing the pro-inflammatory potential of the diet may improve sleep outcomes through weight loss and direct anti-inflammatory effects.

Previous studies have shown that, for example, a study on obese and overweight Iranian women found that higher DII scores were significantly associated with poorer sleep quality (Pittsburgh Sleep Quality Index (PSQI) score > 5 was considered poor sleep quality). After adjusting for confounders, participants in the highest quartile of DII scores had higher global PSQI scores. According to the continuous DII scores, a significant positive correlation between DII and sleep quality was observed in all models [19]. Meanwhile, Setayesh L et al. also found that approximately 58% of participants in the higher DII quartiles had sleep disorders. The results showed a negative association between sleep quality and DII in both the crude model ($\beta = 0.17$, $p = 0.01$) and the fully adjusted model ($\beta = 0.24$, $p < 0.001$), indicating that women with higher DII may have poorer sleep quality [20]. A cross-sectional study by Han Y et al. found that DII scores were higher in hyperlipidemia patients than in

non-hyperlipidemia patients. Multivariate logistic regression analysis showed that the association between DII and hyperlipidemia remained statistically significant after adjusting for many confounders, whether DII was treated as a continuous or categorical variable [20]. Additionally, a study by Martínez-Cerón E et al. on patients with obstructive sleep apnea (OSA) found that the prevalence of dyslipidemia might increase with the severity of OSA, with prevalences of 31%, 33%, 42%, and 51% in non-OSA subjects, mild, moderate, and severe OSA patients, respectively [21]. Farrell ET et al. also found that anti-inflammatory dietary intervention had a significant effect on improving sleep efficiency [22]. This is consistent with our study conclusion: DII is potentially linearly and positively correlated with sleep disorders. After controlling for potential confounders in Model 3, the OR value for the highest quartile group compared to the lowest quartile group was 1.295 (95% CI: 1.039–1.614, $P = 0.0269$), suggesting that long-term pro-inflammatory diets may have adverse effects on sleep. Different from previous studies, our findings extend prior discoveries, indicating that hyperlipidemia patients may be more sensitive to diet-induced sleep impairment.

Subgroup analysis showed that the potential association between DII and sleep disorders might be more significant in individuals under 60 years old. This could be attributed to the higher obesity rate among middle-aged and young people, unhealthy lifestyles (such as high intake of ultra-processed foods), differences in activity habits (such as lack of exercise) [23], and the stronger sensitivity of this group to dietary inflammation, which makes the effect of DII more prominent, suggesting a potential vicious cycle [24]. Delpino, F. M. et al. found that high UPF intake was significantly associated with sleep disorders (such as insomnia and daytime drowsiness), especially in adolescents and young adults [25]. Meanwhile, high UPF intake may further impair sleep quality, and this cyclic relationship is particularly significant in young people. Changes in dietary patterns from adolescence to early adulthood (such as increased UPF intake) occur simultaneously with reduced sleep time [26, 27]. In contrast, chronic low-grade inflammation is common in the elderly, such as changes in immune system function [28], oxidative stress [29], etc., which are not mainly caused by dietary factors but rather by the combined action of other factors, such as chronic stress [30] and environmental pollution [31], which may weaken the strength of the association.

Meanwhile, this study first verified the mediating effect of BMI in the association between DII and sleep disorders among individuals with hyperlipidemia. This association was independent of confounding factors such as age, race, socioeconomic status, and comorbidities. The findings suggest that the potential link between DII and sleep disorders may involve metabolic pathways dependent on BMI (such as obesity-related inflammation and mechanical compression) and direct inflammatory mechanisms independent of BMI. This provides a theoretical basis for multidimensional interventions on the "diet-inflammation-sleep" axis. Multiple associative mechanisms are involved in this process. As a typical chronic low-grade inflammatory disease, hyperlipidemia may render patients more sensitive to dietary inflammation due to lipid metabolism disorders and vascular endothelial dysfunction. Previous studies have shown that: First, research by Nasab MG, Farrell ET, and others has found that a high-DII diet (rich in pro-inflammatory foods) activates inflammatory pathways such as NF-κB, leading to increased levels of pro-inflammatory cytokines like IL-6 and TNF-α [22, 32].

Meanwhile, pro-inflammatory cytokines (e.g., IL-6, TNF-α) may affect the secretion of melatonin and disrupt sleep patterns, with potential involvement of the hypothalamic-pituitary-adrenal (HPA) axis in this regulatory process [33, 34]. A study by Lin WL et al. found that high cholesterol leads to sleep fragmentation by activating the sympathetic nervous system (manifested as an increased LF/HF power ratio) [35]. Oxidative stress, as indicated by elevated late-stage lipid peroxidation markers such as 8-iso-prostaglandin (8-ISO), may contribute to poor sleep quality in patients with coronary artery disease, potentially exacerbating cardiovascular pathophysiology through mechanisms like endothelial dysfunction and localized inflammation [36]. Furthermore, pro-inflammatory diets (high DII) promote energy excess and adipose tissue accumulation, leading to increased BMI. Obesity not only directly disrupts sleep structure through mechanical factors (such as sleep apnea caused by airway obstruction) [37, 38]but also induces systemic inflammatory responses. For example, Yao L et al. found that pro-inflammatory factors released by adipose tissue can affect the hypothalamic sleep regulatory center through the blood-brain barrier [39]. In addition, obesity-related genes (such as FTO) and circadian clock genes (CLOCK, BMAL1) may play a key role in regulating sleep rhythms, which suggests that there may be a potential genetic link between obesity-related genes and circadian clock mechanisms in sleep regulation [40]. Furthermore, Brady EM and van Dijk observed that higher BMI is associated with insulin resistance, and both BMI and insulin resistance show relationships with sleep duration (with a U-shaped association for BMI and sleep), suggesting a potential indirect link between BMI, insulin resistance, and sleep regulation [41, 42].

The main strengths of this study include: (1) Using a large nationally representative sample from NHANES to ensure the generalizability of the results; (2) Systematically evaluating the association between DII and

sleep disorders and potential mechanisms through multiple statistical models; (3) Incorporating rich covariates (sociodemographic, lifestyle, comorbidities) to reduce confounding bias. However, this study still has certain limitations: (1) Although it shows an association between pro-inflammatory diets and sleep disorders, cross-sectional data cannot clarify whether dietary inflammation directly causes sleep disorders. Longitudinal cohort studies or randomized controlled trials (RCTs) are needed in the future to verify the causal relationship between the two. (2) Sleep disorders were assessed through self-reported physician diagnoses, which may involve recall bias and classification bias; "sleep disorders" cover multiple diseases, and this method cannot distinguish specific types, affecting the precision of the results; undiagnosed patients were classified as having no sleep disorders, potentially underestimating the prevalence and weakening the association. Future studies should use objective measurement tools such as polysomnography or targeted questionnaires to deeply explore the relationship between dietary inflammation, BMI, and sleep health. (3) The mediation analysis only explored the single variable of BMI without integrating other biomarkers (such as C-reactive protein, IL-6). Future research needs to construct multi-dimensional mediation models. Nevertheless, this study provides a new perspective for the comprehensive management of hyperlipidemia patients, suggesting that reducing DII (such as increasing intake of fruits, vegetables, and whole grains and reducing processed foods and saturated fats) may simultaneously improve weight and sleep health, thereby reducing the risk of cardiovascular and metabolic diseases.

Conclusion

The results of this cross-sectional study indicate a potential positive association between pro-inflammatory diets and the risk of sleep disorders in patients with hyperlipidemia. There was a significant trend across the quartiles of the Dietary Inflammatory Index (DII) (trend test $P<0.05$). Compared with the lowest quartile (Q1), the highest quartile (Q4) had an adjusted odds ratio (OR) of 1.295 (95% confidence interval [CI]: 1.039–1.614, $P=0.0269$), which was particularly evident in the young and middle-aged population (<60 years old). Reducing the dietary inflammatory index may improve sleep health through weight loss (mediated by BMI) and direct anti-inflammatory effects. In clinical management, DII can be incorporated into nutritional assessment indicators, and individualized dietary plans can be formulated in combination with BMI monitoring. Meanwhile, future interventional studies are needed to clarify the causal impact of dietary inflammation regulation on sleep quality and the underlying molecular mechanisms, providing a scientific basis for precision nutritional intervention.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s40795-025-01157-4.

Supplementary Material 1

Acknowledgements
During the process of this study, no assistance was received from any individual, organization or institution.

Author contributions
Yueyu Zhang conceived and designed the study, conducted the main experiments, analyzed the data, and wrote the initial manuscript. Yi Tang participated in experimental design and data collection. Xinyi Chen assisted with data processing and preliminary analysis. Yu Wang contributed to the interpretation of experimental results. Kangrui Zhang helped verify experimental data and prepare figures. Juncang Wu supervised the project, offered key methodological and analytical guidance, obtained funding, and reviewed and revised the manuscript.

Funding
This work was supported by grants from "Hefei Key Common Technology R&D"Projects (GJ2022SM07), "The Hospital - level Scientific Research Fund Project of Hefei Second People's Hospital" Project (2024ykc012),"Key Projects in Natural Sciences" Project (2024byzd401).

Data availability
Data supporting the findings of this study can be furnished by the corresponding author, subject to a justified request.

Declarations

Ethics approval and consent to participate
This research was carried out in compliance with the ethical principles outlined in the 1964 Declaration of Helsinki and its subsequent revisions. The data collection for NHANES had been approved by the Research Ethics Review Board (ERB) of the National Center for Health Statistics (NCHS). As an individual researcher leveraging the publicly accessible NHANES dataset, there was no requirement to submit an application to the institutional internal review board (IRB). Informed consent was obtained from all participants, and the study was approved by the Research Ethics Review Board of the National Center for Health Statistics.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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Received: 10 May 2025 / Accepted: 18 August 2025
Published online: 07 October 2025

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