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ORIGINAL RESEARCH

The Dietary Inflammatory Index (DII) and COPD: A Cross-Sectional Study from the NHANES

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Background: While diet may impact the risk of chronic obstructive pulmonary disease (COPD), the relationship between COPD and dietary inflammatory potential remains largely unexplored. This study aimed to evaluate the association between COPD status and the dietary inflammatory index (DII).

Methods: Utilizing NHANES data from 2013 to 2018, the study investigated the relationship between Dietary Inflammatory Index (DII) scores, derived from 24-hour dietary recall interviews, and COPD status. Weighted logistic regression and restricted cubic spline (RCS) analyses were employed to assess this association. Additionally, stratified and interaction analyses were conducted to evaluate the consistency of the relationship and identify potential modifiers.

Results: Individuals diagnosed with COPD demonstrated significantly elevated DII scores in comparison to those without COPD. An increment of one unit in the DII was correlated with an increased risk of developing COPD, as indicated by an odds ratio (OR) of 1.05 (95% CI:1.09, 1.21; P=0.007). Following comprehensive multivariate adjustments, the odds ratio for COPD, when comparing individuals in the highest quartile of DII scores to those in the lowest quartile, was 1.34 (95% CI:1.01, 1.77; P<0.001). A positive linear association was observed between DII and COPD, although the relationship was nonlinear (P=0.618). Moreover, the association between DII and COPD was consistent across various stratified analyses.

Conclusion: The study results imply that consuming a pro-inflammatory diet is connected to a greater chance of developing COPD among US residents. Dietary strategies aimed at reducing inflammation might help in preventing COPD and associated illnesses.

Keywords: diet, inflammation, dietary inflammatory index, chronic obstructive pulmonary disease, National Health and Nutrition Examination Survey, NHANES

Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a prevalent, preventable, and manageable condition marked by persistent and progressive airflow limitation due to an enhanced inflammatory response in the airways and lungs from exposure to harmful particles or gases.[1] As reported in the Global Burden of Disease Study 2019, COPD ranks as the third leading cause of death globally, following ischemic heart disease and stroke.[2] In the United States, it significantly contributes to morbidity and mortality, affecting an estimated 16 million adults, though underdiagnosis suggests higher actual numbers.[3] Globally, COPD prevalence is around 10.3%based on the Global Initiative for Chronic Obstructive Lung Disease criteria, equating to approximately 391.9 million cases among individuals aged 30 to 79 years.[4] The disease's economic impact is staggering, with annual direct costs exceeding $32 billion in the US alone, compounded by indirect costs from lost productivity and caregiver burdens.[5] COPD pathogenesis involves environmental factors, genetic predisposition, and inflammatory processes, characterized by chronic inflammation in the airways and lung parenchyma. This leads to structural changes such as airway remodeling, emphysema, and increased mucus production, driven by immune cells like neutrophils, macrophages, and T lymphocytes, which release pro-inflammatory cytokines and mediators contributing to tissue damage and airflow obstruction.

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Recent research underscores the critical impact of diet on COPD pathogenesis and progression.[6] The relationship between diet and malnutrition in COPD patients is a critical. Malnutrition is a prevalent yet frequently overlooked comorbidity in patients with COPD, with dietary inadequacies playing a central role in its pathogenesis. Studies indicate that approximately 17–55% of COPD patients exhibit malnutrition, characterized by insufficient energy and protein intake, weight loss, and reduced fat-free mass, which exacerbates disease severity and functional decline.[7] Systemic inflammation, driven by cytokines such as TNF-α, and elevated resting energy expenditure (REE) contribute to a catabolic state, further compromising nutritional status despite adequate food consumption.[8] For instance, malnourished COPD patients demonstrate significantly lower spirometric values (eg, FEV1 and FVC) and diminished exercise capacity, as evidenced by reduced 6-minute walk distances and peak oxygen uptake.[9] Notably, protein-calorie malnutrition correlates with respiratory muscle wasting, impaired diaphragm contractility, and increased dyspnea, as measured by the modified Medical Research Council (mMRC) scale.[10] In summary, dietary choices may result in inadequate nutrition for patients with COPD, and malnutrition can affect COPD-related inflammation, with certain patterns either worsening or alleviating the disease. A diet high in meat consumption, saturated fats, and refined carbohydrates is linked to a higher COPD risk, while a diet rich in fruits, vegetables, and whole grains, which contain antioxidants and anti-inflammatory agents, may offer protective benefits. Antioxidants like vitamins A, C, E, and carotenoids are crucial in reducing oxidative stress, a key factor in COPD, and are associated with better lung function and lower COPD risk.[11] Studies show that higher carotenoid intake correlates with reduced COPD incidence, highlighting the role of dietary antioxidants in mitigating lung oxidative damage.[12] Additionally, dietary fiber, which is fermented into anti-inflammatory short-chain fatty acids by gut microbiota, is linked to improved lung health and reduced COPD risk. This emphasizes the significance of a balanced diet in managing and potentially preventing COPD.[13]

The Dietary Inflammatory Index (DII) evaluates the inflammatory potential of a diet by assigning inflammatory weights to various food items and nutrients based on their association with inflammatory biomarkers.[14] This scoring system categorizes diets from anti-inflammatory to pro-inflammatory, offering insights into dietary influences on health outcomes, particularly chronic diseases like cardiovascular disease, diabetes, asthma, COPD, and cancer.[11,15–18] There is an association between higher DII scores and a heightened risk of early COPD along with reduced lung function.[19] The association between diet, inflammation, and COPD is recognized; yet, there remains a scarcity of data regarding the specific role of dietary inflammation, as measured by the DII, in influencing COPD in real-world contexts. The objective of this research is to investigate this relationship by utilizing data from the National Health and Nutrition Examination Survey (NHANES) to improve comprehension and guide dietary prevention strategies for COPD.

Materials and Methods

Data Sources

The NHANES evaluates the health and nutritional status of the US civilian noninstitutionalized population. It covers topics like chronic diseases, dietary intake, physical activity, and health behaviors. Participants undergo standardized physical exams in mobile centers, where trained technicians measure health metrics such as height, weight, blood pressure, and conduct laboratory tests. According to the Ethical Review Methods for Life Science and Medical Research Involving Human Participants, Article 32 exempts certain research from requiring ethical approval under specific conditions. Research utilizing legally obtained public data or data derived from non-intrusive observation of public behavior does not require ethical approval.[20] Our study complies with exemption conditions by using legally obtained public data and anonymized informational data, ensuring no interference with public behavior. The study's design, data gathering, and analysis adhered strictly to the STROBE guidelines, guaranteeing methodological rigor and ethical compliance. In addition, the study protocols were sanctioned by the Institutional Review Board of the National Center for Health Statistics (NCHS).[21] Since the study relies on publicly accessible data, no patient consent or hospital ethics approval was required, as the data is de-identified and meets exemption criteria for informed consent and ethical approval.

The DII scores data were solely obtained from NHANES cycles between 1999 and 2018, targeting adults aged 18 to 80. Conversely, data on COPD outcomes were solely accessible from the NHANES cycles carried out from 2013 to

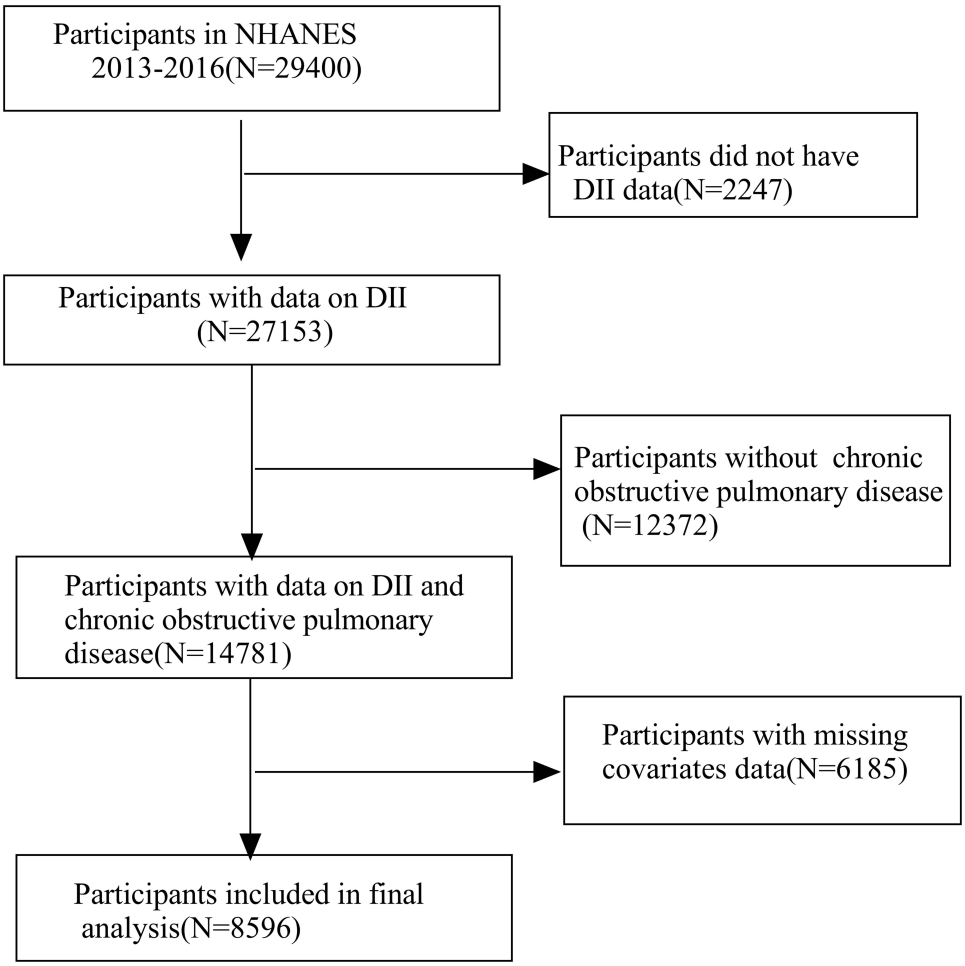

Figure 1 Flowchart of participants selection.
Abbreviations: NHANES, National Health and Nutrition Examination Survey.

2018. In our study, we used NHANES data that is publicly available, sourced from surveys carried out in 2013–2014, 2015–2016, and 2017–2018. A total of 29,400 people participated in these extensive surveys, offering crucial demographic details. We initially excluded 2247 individuals without DII data, which resulted in a group of 27,153 participants aged 18 to 79 years with available DII information. Following this, 12,372 participants lacking COPD data were excluded, along with those missing information on 6185 covariates. Once these exclusions were made, our final analysis sample was composed of 8596 subjects, including 4450 males and 4146 females. A detailed study flowchart illustrating this process is provided in Figure 1.

Measurement of DII

Between 1999 and 2018, participants in NHANES provided comprehensive dietary data through two meticulously designed rounds of validated 24-hour dietary recall surveys. These surveys established a robust framework for evaluating individuals' dietary intake over the specified period. To investigate the inflammatory potential associated with various dietary patterns among participants, researchers employed the DII. The methodology for constructing and validating the DII has been thoroughly addressed in previous studies, ensuring that the index is both reliable and comprehensive.[22] In this research, a total of 28 different dietary components were included in the DII calculation, offering a multifaceted view of dietary influences on inflammation. This extensive list encompasses essential macronutrients such as carbohydrates, proteins, total fats, and alcohol, as well as dietary fiber and cholesterol. Notably, the assessment also accounted for different types of fatty acids, including saturated, monounsaturated, and polyunsaturated fatty acids, alongside specific

fatty acid profiles like n-3 and n-6. Vitamins were also represented in the analysis, incorporating vital nutrients such as vitamins A, B1 (thiamine), B2, B6, B12, C, D, and E, and various minerals, including iron, magnesium, zinc, selenium, folate, and carotenoids, as well as caffeine and overall energy intake, thereby providing a comprehensive assessment of dietary contributions to inflammation.[23]

COPD Outcomes

Based on prior research, a self-reported diagnosis of COPD was established by a physician and includes conditions such as chronic bronchitis and emphysema, in addition to COPD itself.[24,25] Participants were classified as having COPD through a composite assessment of three self-reported items from a COPD questionnaire administered during individual interviews. The questions posed were: Has a physician ever diagnosed you with chronic bronchitis? Have you ever been diagnosed with emphysema by a physician? Has a healthcare provider ever diagnosed you with COPD?Respondents affirming any of these questions were classified as having COPD, whereas those who did not were categorized as non-COPD.

Covariates Assessment

Previous research[26,27] and clinical assessments have identified confounding factors including age, gender, ethnicity, marital status, education level, and poverty income ratio(PIR), along with alcohol consumption, smoking behaviors, levels of physical activity, cardiovascular conditions(CVD), hypertension, cancer, and diabetes mellitus (DM). Information was gathered through standardized questionnaires during family interviews. Ethnicity data was self-reported and categorized as non-Hispanic white, non-Hispanic black, Mexican American, and other racial groups. Marital status was classified as married, never married, living with a partner, or other statuses, which encompass widowed, divorced, or separated. Educational attainment was divided into three levels. Education levels were classified as under high school, high school, and above high school. Family income was divided into three categories according to the family poverty income ratio. Income levels were categorized as low (≤1.3), medium (>1.3 to 3.5), and high (>3.5). Individuals who did not smoke were defined as those who had never engaged in smoking or had smoked fewer than 100 cigarettes in their lifetime. Former smokers were classified as individuals who had consumed at least 100 cigarettes during their lifetime but had since ceased smoking. Conversely, current smokers were defined as those who had smoked a minimum of 100 cigarettes and continued to do so.[28] The classification of alcohol consumption included non-drinkers, light to moderate drinkers (up to 2 drinks per day for men and up to 1 drink per day for women), and heavy drinkers (more than 2 drinks per day for men and more than 1 drink per day for women).[29] Physical activity was classified into two categories. Individuals are categorized as insufficiently active if they engage in 1 to 5 moderate or 1 to 3 vigorous leisure activities weekly, while those exceeding these levels are considered active.[30] Cardiovascular disease (CVD) encompasses conditions such as heart failure, coronary artery disease, angina, heart attacks, and cerebrovascular accidents.[26] For more information on these factors, please visit the NHANES website.

Statistical Analyses

Sample weights were applied in all analyses due to NHANES's complex survey design. For continuous variables, the median and interquartile range (IQR) were used, and categorical variables were represented by unweighted frequencies and weighted percentages. Dietary Inflammatory Index(DII)scores were divided into four categories, with the highest group (Q4) indicating diets with the greatest pro-inflammatory potential, and the lowest quartile (Q1) indicating diets with anti-inflammatory properties. Differences among groups were assessed using the weighted chi-square test or Kruskal–Wallis *H*-test. The association between DII scores and COPD was examined using weighted logistic regression models, calculating odds ratios (ORs) and 95%confidence intervals (CIs) for each DII quartile, with the lowest quartile as the reference. A restricted cubic spline analysis illustrated the potential non-linear relationship between COPD and DII. The initial model was unadjusted, Model 1 adjusted for age, sex, race or ethnicity, marital status, education level, and poverty income ratio, while Model 2 further adjusted for BMI group, smoking status, drinking status, physical activity, hypertension, diabetes, cancer, and cardiovascular disease (CVD). Statistical interaction tests were conducted to verify

the reliability of observed changes. Analyses were performed using R software version 4.4.0, with two-sided p-values below 0.05 considered statistically significant.

Study Participants and Baseline Characteristics

The baseline characteristics of the individuals are shown in Table 1. A total of 8596 adults were enrolled in our study, comprising 4450 males (51.77%) and 4146 females (48.23%). Among these participants, 691 individuals (8.04%) suffered from COPD. The COPD group consisted of older individuals, mainly Non-Hispanic White, who were less often married, had lower incomes, and engaged less in physical activities compared to those without COPD. They

Table 1 The Baseline Characteristics of Participants Sourced From the NHANES Database

Variables	Overall (n=8596)	COPD (n=691)	Non-COPD (n=7905)	*P* value
Age (years)	46.00 (32.00, 61.00)	61.00 (43.00, 80.00)	45.00 (32.00, 60.00)	<0.001
Age group				<0.001
<60 years	7144 (83.11)	397 (77.69)	6747 (83.45)	
≧60 years	1452 (16.89)	114 (22.31)	1338 (16.55)	
Sex (%)				<0.001
Male	4450 (51.77)	304 (43.99)	4146 (52.45)	
Female	4146 (48.23)	387 (56.01)	3759 (47.55)	
BMI Group				0.552
Normal (≤25kg/m^2)	3179 (36.98)	182 (35.62)	2997 (37.07)	
Overweight (25–30kg/m^2)	2256 (26.24)	145 (28.38)	2111 (26.11)	
Obesity (>30kg/m^2)	3161 (36.77)	184 (36.01)	2977 (36.82)	
Race and ethnicity (%)				<0.001
Mexican American	1180 (13.73)	29 (4.20)	1151 (14.56)	
Non-Hispanic White	3628 (42.21)	459 (66.43)	3169 (40.09)	
Non-Hispanic Black	1781 (20.72)	100 (14.47)	1681 (21.27)	
Other race	2007 (23.35)	103 (14.91)	1904 (24.09)	
PIR Group				<0.001
≤1.3	2328 (27.08)	228 (33.00)	2100 (26.57)	
1.3–3.5	3279 (38.15)	292 (42.26)	2987 (37.79)	
>3.5	2989 (34.77)	171 (24.75)	2818 (35.65)	
Educational level (%)				0.163
Below high school	1309 (15.23)	117 (16.93)	1192 (15.08)	
High school	1939 (22.56)	167 (24.17)	1772 (22.42)	
Above high school	5348 (62.21)	407 (58.90)	4941 (62.50)	
Marital status (%)				<0.001

(*Continued*)

Table 1 (Continued).

Variables	Overall (n=8596)	COPD (n=691)	Non-COPD (n=7905)	*P* value
Married	4317 (50.22)	306 (44.28)	4011 (50.74)	
Widowed/Divorced/Separated	1702 (19.80)	251 (36.32)	1451 (18.36)	
Never married	1730 (20.13)	100 (14.47)	1630 (20.62)	
Living with a partner	847 (9.85)	34 (4.92)	813 (10.28)	
Smoking status (%)				<0.001
Never smoker	4549 (52.92)	256 (37.05)	4293 (54.31)	
Former smoker	2121 (24.67)	244 (35.31)	1877 (23.74)	
Current smoker	1926 (22.41)	191 (27.64)	1735 (21.95)	
Drinking status				<0.001
Never drinker	4332 (50.40)	395 (57.16)	3937 (49.80)	
Light/moderate drinker	2909 (33.84)	196 (28.36)	2713 (34.32)	
Heavier drinker	1355 (15.76)	100 (14.47)	1255 (15.88)	
Physical activity (%)				<0.001
Insufficiently activity	2820 (32.81)	310 (44.86)	2510 (31.75)	
Sufficient activity	5776 (67.19)	381 (55.14)	5395 (68.25)	
CVD (%)	300 (3.49)	77 (11.14)	223 (2.82)	<0.001
Cancer (%)	787 (9.16)	170 (24.60)	617 (7.81)	<0.001
Hypertension (%)	3419 (39.77)	408 (59.04)	3011 (38.09)	<0.001
Diabetes (%)	1433 (16.67)	167 (24.17)	1266 (16.02)	<0.001
DII score	0.99 (−0.59, 2.22)	1.51 (−0.17, 2.43)	0.94 (−0.62, 2.21)	<0.001
DII score categories[4]				<0.001
Q1 (−4.68, 0.23)	2149 (25.00)	135 (19.54)	2014 (25.48)	
Q2 (0.24, 1.84)	2149 (25.00)	144 (20.84)	2005 (25.36)	
Q3 (1.85, 3.05)	2149 (25.00)	207 (29.96)	1942 (24.57)	
Q4 (3.06, 5.30)	2149 (25.00)	205 (29.67)	1944 (24.59)	

Abbreviations: BMI, body mass index; PIR, Ratio of family income to poverty; CVD, cardiovascular disease; COPD, chronic obstructive pulmonary disease.

exhibited a higher likelihood of being current smokers and a greater prevalence of cardiovascular disease, hypertension, diabetes, and cancer. Participants with COPD generally exhibited higher DII scores compared to those without COPD. Supplementary Table S1 presents the characteristics of subjects categorized by DII score quartiles. An elevated DII score correlated with increased probabilities of being female, a current smoker, Non-Hispanic Black, never married, having reduced physical activity, and possessing lower income. Additionally, participants with elevated DII scores exhibited lower educational levels. Moreover, individuals in the highest inflammation quartile (T4) demonstrated an increased likelihood of having COPD.

Table 2 Association Between the Dietary Inflammatory Index and COPD in the Multiple Regression Model

DII	Model1		Model2		Model3	
	OR (95% CI)	P-value	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value
Continuous	1.15 (1.09, 1.21)	<0.001	1.10 (1.04, 1.15)	<0.001	1.05 (1.01, 1.11)	0.007
Categories						
Q1	ref		ref		ref	
Q2	1.19 (0.89, 1.59)	0.240	1.14 (0.85, 1.53)	0.385	1.10 (0.82, 1.48)	0.521
Q3	1.75 (1.34, 2.29)	<0.001	1.53 (1.16, 2.01)	0.003	1.22 (1.12, 1.62)	<0.001
Q4	1.82 (1.40, 2.38)	<0.001	1.82 (1.12, 1.95)	0.005	1.34 (1.01, 1.77)	<0.001
Trend test		<0.001		<0.001		0.003

Notes: Model 1 was analyzed without adjusting for any covariates. Model 2, factors such as age, sex, race or ethnicity, marital status, education level, and poverty income ratio were adjusted. Model 3, adjustments were made for BMI group, smoking status, alcohol use, physical activity, hypertension, diabetes, cancer, and CVD, in addition to the factors considered in Model 1.
Abbreviations: BMI, body mass index; OR, odds ratio; 95% CI, 95%confidence interval; ref, reference; COPD, chronic obstructive pulmonary disease; CVD, cardiovascular disease.

Association of DII with COPD

We conducted a multivariable logistic regression analysis to explore the relationship between DII and COPD (Table 2). Both the unadjusted and multivariate-adjusted logistic regression models demonstrated a positive association, indicating that an increase in DII is correlated with a higher likelihood of developing COPD. In the final model (Model 3), which accounted for all covariates, a one-unit increase in DII, treated as a continuous variable, was associated with a 10% increase in the risk of developing COPD (OR:1.05, 95% CI:1.01–1.11; *P*=0.007). When DII was categorized into quartiles, the odds ratios with 95%confidence intervals for COPD across the quartiles were 1.10 (0.82, 1.48), 1.34 (1.01, 1.77), and 1.34 (1.01, 1.77), respectively, after adjusting for all covariates in Model 3. Furthermore, the restricted cubic spline model revealed a linear relationship between DII and COPD (*P* for nonlinearity=0.618) (Figure 2).

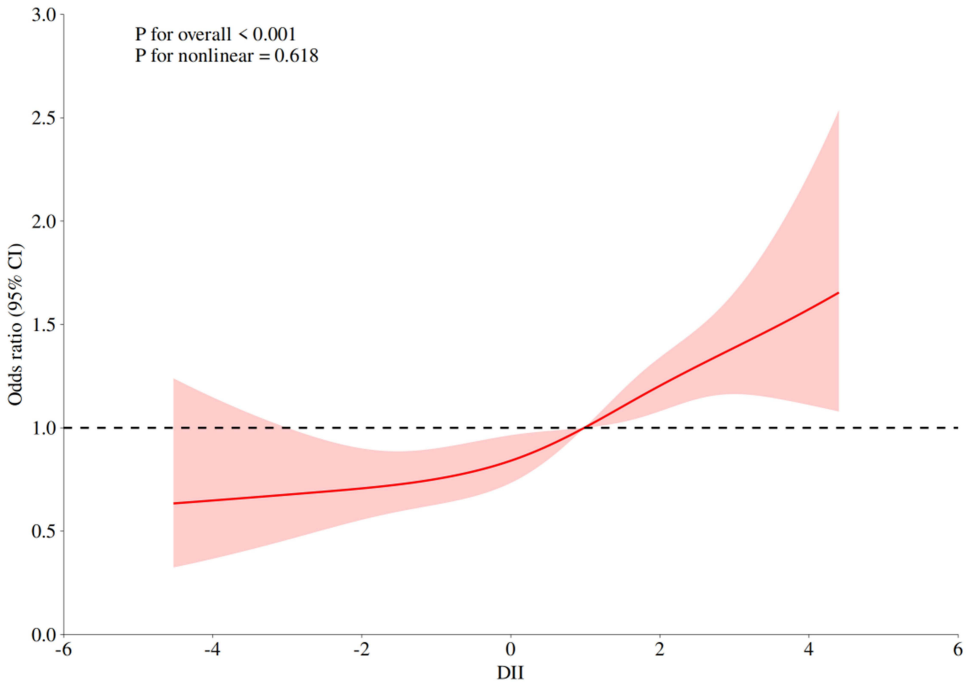

Figure 2 Restricted spline curve for the association between DII and COPD.
Abbreviations: DII, Dietary Inflammatory Index; COPD, Chronic Obstructive Pulmonary Disease.

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Subgroup Analysis

This study performed stratified and interaction analyses to assess the reliability of the association between the DII and COPD incidence across different subgroups. Table 3 illustrates that the subgroup analysis revealed differing associations between DII scores and COPD; however, a predominantly positive correlation was observed in most categories. The interaction test results revealed that cancer status significantly modified the relationship between DII scores and COPD (*P* for interaction<0.05).

Table 3 Stratified Analysis of the Associations Between DII and COPD

Subgroup	N (%)	OR (95% CI)	*P*	*P* for Interaction
All patients	8596 (100.00)	1.11 (1.07~1.16)	<0.001	
Sex				0.701
Male	4450 (51.77)	1.11 (1.04~1.18)	0.002	
Female	4146 (48.23)	1.09 (1.03~1.16)	0.004	
BMI Group		1.02 (0.92~1.12)	0.763	0.568
Normal (≤25kg/m^2)	3179 (36.98)	1.35 (1.22 ~ 1.50)	<0.001	
Overweight (25–30kg/m^2)	2256 (26.24)	1.37 (1.21 ~ 1.54)	<0.001	
Obesity (>30kg/m^2)	3161 (36.77)	1.46 (1.32 ~ 1.62)	<0.001	
Age group				0.551
<60 years	7144 (83.11)	1.38 (1.29 ~ 1.48)	<0.001	
≧60 years	1452 (16.89)	1.45 (1.26 ~ 1.66)	<0.001	
Race and ethnicity (%)				0.105
Mexican American	1180 (13.73)	1.07 (0.86~1.32)	0.534	
Non-Hispanic White	3628 (42.21)	1.10 (1.04~1.16)	<0.001	
Non-Hispanic Black	1781 (20.72)	1.03 (0.92~1.15)	0.563	
Other race	2007 (23.35)	1.25 (1.12~1.40)	<0.001	
Educational level (%)				0.459
Below high school	1309 (15.23)	1.18 (1.05~1.32)	0.004	
High school	1939 (22.56)	1.08 (0.98~1.18)	0.122	
Above high school	5348 (62.21)	1.11 (1.05~1.17)	<0.001	
Marital status (%)				0.122
Married	4317 (50.22)	1.13 (1.06~1.21)	<0.001	
Widowed/Divorced/Separated	1702 (19.80)	1.03 (0.96~1.11)	0.422	
Never married	1730 (20.13)	1.13 (1.01~1.26)	0.036	
Living with a partner	847 (9.85)	1.27 (1.03~1.57)	0.025	
Smoking status (%)				0.919
Never	4549 (52.92)	1.12 (1.04~1.20)	0.002	
Former	2121 (24.67)	1.12 (1.04~1.21)	0.002	

(*Continued*)

Table 3 (Continued).

Subgroup	N (%)	OR (95% CI)	*P*	*P* for Interaction
Current	1926 (22.41)	1.10 (1.01~1.19)	0.035	
Drinking status				0.793
Never drinker	4332 (50.40)	1.11 (1.05~1.18)	<0.001	
Light/moderate drinker	2909 (33.84)	1.11 (1.03~1.21)	0.010	
Heavier drinker	1355 (15.76)	1.16 (1.03~1.31)	0.012	
Physical activity (%)				0.608
Insufficiently activity	2820 (32.81)	1.11 (1.04~1.19)	0.002	
Sufficient activity	5776 (67.19)	1.09 (1.03~1.15)	0.003	
Hypertension				0.178
YES	3419 (39.77)	1.08 (1.02~1.14)	0.011	
NO	5177 (60.23)	1.14 (1.07~1.22)	<0.001	
CVD status				0.195
Yes	300 (3.49)	1.02 (0.89~1.17)	0.776	
NO	8296 (96.51)	1.13 (1.08~1.18)	<0.001	
Cancer				<0.001
Yes	787 (9.16)	0.97 (0.89~1.06)	0.468	
NO	7809 (90.84)	1.16 (1.11~1.22)	<0.001	
Diabetes				0.216
YES	1433 (16.67)	1.06 (0.97~1.16)	0.219	
NO	7163 (83.33)	1.13 (1.07~1.19)	<0.001	
PIR				0.130
≤1.3	2328 (27.08)	1.15 (1.06~1.25)	<0.001	
1.3–3.5	3279 (38.15)	1.13 (1.04~1.23)	0.004	
>3.5	2989 (34.77)	1.04 (0.95~1.14)	0.422	

Abbreviations: BMI, body mass index; OR, odds ratio; 95% CI, 95%confidence interval; ref, reference; COPD, chronic obstructive pulmonary disease; CVD, cardiovascular disease; PIR, ratio of family income to poverty.

Discussion

This is the first study to explore the relationship between the DII and COPD in a large group of adults from the general population. Our analysis identified a strong positive correlation between DII and COPD prevalence, persisting after covariate adjustments. After extensive multivariable adjustments, a statistically significant association was observed. Compared to individuals in the lowest DII quartile (Q1), those in the highest DII quartile (Q4) exhibited a significantly increased OR of 1.34 (95% CI 1.01, 1.77, $P<0.001$) for COPD. A linear positive relationship was identified between DII and COPD, highlighting the influence of pro-inflammatory diets on heightened COPD risk. Furthermore, our subgroup analyses consistently indicated a strong association between DII and COPD. These findings indicate that DII could be an important marker for COPD. An anti-inflammatory diet for patients with COPD includes fruits rich in antioxidants, such as berries and oranges, as well as natural antioxidant and anti-inflammatory substances.[31] The diet may also incorporate

vitamin or probiotic supplements, vegetables like spinach and kale, whole grains such as oats and brown rice, and fatty fish high in omega-3 fatty acids, including salmon and mackerel.[32] Additionally, nuts and seeds, such as almonds and flaxseeds, provide essential nutrients.[33] This dietary approach offers crucial evidence for developing dietary guidelines aimed at improving outcomes for COPD patients.

Limited epidemiological research has investigated the link between DII and COPD. A prior study involving 3962 participants aged 20 to 49 demonstrated that higher DII scores are significantly linked to reduced lung function, particularly affecting Forced Expiratory Volume in one second (FEV1) and Forced Vital Capacity (FVC). An increase in the DII score was associated with a reduction in FEV1 between 0.43 L and 0.58 L, indicating that dietary inflammation might play a role in the decline of lung function in early COPD cases.[34] Another study suggested that higher DII scores were positively correlated with lower lung function, with each unit increase in DII associated with a decrease in FEV1 by approximately 3.44 units. This also suggests that a diet high in inflammatory potential may lead to further airway obstruction and respiratory symptoms thereby worsening airflow restriction.[35] In a separate analysis involving 121 COPD patients. The study revealed that as the severity of COPD increased, the quality of diet, as measured by the DII, was inversely related to lung function parameters.[36] Specifically, patients with more pro-inflammatory dietary patterns exhibited poorer lung function outcomes, emphasizing the importance of dietary interventions in managing COPD.

Epidemiologic and clinical studies on the DII and lung diseases, particularly COPD, are limited. However, experimental research suggests that DII may impact lung health by influencing chronic inflammation, oxidative stress, and related factors. Chronic inflammation is crucial in COPD development, and dietary factors can modulate these inflammatory states. For instance, certain foods and dietary components can either exacerbate or alleviate inflammation, impacting the overall inflammatory burden on the body. The DII measures the inflammatory potential of a person's diet and is associated with inflammatory markers like C-reactive protein and cytokines, including IL-6 and TNF-α.[37] These markers are crucial in the inflammatory processes of COPD, with oxidative stress also playing a key role in its pathogenesis.[12] The disease is linked to an imbalance between reactive oxygen species production and the body's capacity to detoxify these intermediates or repair the damage. Diets high in antioxidants, such as those rich in fruits and vegetables, may help mitigate oxidative stress and its detrimental effects on lung function. Conversely, a pro-inflammatory diet, which is often low in these protective nutrients, may exacerbate oxidative stress and contribute to the progression of COPD.[38] In addition to inflammation and oxidative stress, vascular resistance is also a critical aspect of COPD pathophysiology. Chronic inflammation can lead to vascular remodeling and increased vascular resistance, which further complicates the respiratory function in COPD patients. The DII may influence these vascular changes through its effects on systemic inflammation and oxidative stress, thereby impacting pulmonary vascular resistance and overall lung health.[18] Overall, the association between DII and COPD emphasizes the potential of dietary changes to impact chronic inflammation, oxidative stress, and vascular resistance, key factors in the disease's pathogenesis.

This research possesses significant strengths. Firstly, it represents the first exploration of the link between DII an COPD in a representative population within the United States. Secondly, a thorough examination of measurement techniques and protocols was conducted utilizing the NHANES database. Thirdly, we accounted for potential confounders and formulated robust conclusions across various subgroups. Nonetheless, our investigation encountered certain limitations. Our study has limitations. To begin with, the retrospective cohort design presents challenges in fully mitigating confounding bias and establishing cause-and-effect relationships. Secondly, using participants'memories for dietary assessments can lead to recall bias, and any alterations in eating habits during interviews might have been overlooked. Additionally, due to limited data availability, the evaluation of DII was confined to 28 of the originally suggested 45 dietary parameters, potentially affecting the precision of assessing inflammatory potential. Furthermore, the absence of pulmonary function data led to their exclusion as confounding variables, while self-reported COPD may introduce potential misclassification bias into our analysis. Finally, this study did not evaluate specific nutritional elements, particularly the relationship between vitamin D, vitamin C, and COPD. Although a statistically significant correlation exists between the DII) and COPD, the limited sample size and unidentified confounding factors result in a minimal risk difference, which may limit its clinical significance. As a result, upcoming prospective studies should target larger sample sizes to improve the applicability and significance of the results while exploring the causal link between DII and COPD outcomes further. Residual or unidentified confounding effects may persist in observational

research. To delve deeper into the causal association between dietary inflammation and COPD outcomes, future studies need to incorporate thorough confounder adjustments, improved dietary assessments, and additional DII parameters.

Conclusion

Our study concludes that a pro-inflammatory diet is significantly linked to an increased risk of COPD in the United States. These findings could provide important insights for future studies focused on highlighting the significance of dietary health, especially for those adhering to pro-inflammatory diets. We advise individuals with these dietary patterns to enhance their consumption of anti-inflammatory foods to lower the risk of COPD.

Data Sharing Statement

This study's datasets are accessible in the NHANES database (https://www.cdc.gov/nchs/nhanes/index.htm).

Consent for Publication

All authors have read this manuscript and consent to publish it.

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Author Contributions

Zhi Jian Luo is the first author for this study. All authors significantly contributed to the work, including conception, study design, execution, data acquisition, analysis, and interpretation, as well as drafting, revising, or critically reviewing the article. They approved the final version for publication and agreed to be accountable for all aspects of the work involved in data acquisition, analysis, interpretation, or execution; participated in drafting, revising, or conducted a critical review of the article, approved the final version for publication, consented to the journal submission, and accepted responsibility for all aspects of the work.

Disclosure

The authors have no relevant financial or non-financial interests to disclose for this work.

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