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The association between Planetary Health Diet Index (PHDI) and chronic obstructive pulmonary disease (COPD): the mediating role of dietary inflammatory index (DII)

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Abstract

Background Given global changes in the environment and dietary habits, it is critical to understand the potential impact of dietary factors and dietary inflammation on respiratory diseases, including COPD. Studying these relationships can help develop more effective prevention strategies. PHDI is a dietary scoring system designed to balance human health and environmental sustainability by promoting increased consumption of plant-based foods and reduced intake of red meat, sugar, and highly processed foods. In contrast, DII quantifies the inflammatory potential of a diet. This study examines the association between PHDI and COPD and assesses whether DII mediates this relationship.

Methods We used subgroup analysis, smooth curve fitting, and multivariable logistic regression to investigate the connection between PHDI and the occurrence of COPD. Furthermore, a mediation analysis was carried out to investigate any possible correlation between DII and the link between PHDI and COPD.

Results 30,304 participants were included in this investigation, and 1,498 of them reported COPD events. For every 10-point increase in PHDI and each unit increase in DII was associated with a 9% reduction (OR = 0.91, 95% CI: 0.86, 0.97) and an 8% increase (OR = 1.08, 95% CI: 1.02, 1.13) in the prevalence of COPD, respectively, when all variables were adjusted for using multivariable logistic regression. Additionally, the results remain robust when PHDI and DII are converted to tertile. An investigation of smooth curve fitting showed a linear correlation between the risk of COPD and PHDI. The results of the mediation analysis showed that 17.95% of the relationship between PHDI and COPD was mediated by DII ($p = 0.034$).

Conclusions Higher PHDI levels are associated with a lower prevalence of COPD. Additionally, DII appears to mediate this relationship, suggesting that an anti-inflammatory diet may provide benefits.

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Keywords Planetary health diet index, Dietary inflammatory index, Chronic obstructive pulmonary disease, NHANES, Mediation analysis

Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a chronic inflammatory respiratory disease characterized by irreversible and progressive airflow limitation [1]. It is marked by airway inflammation and remodeling, which lead to airway narrowing and airflow obstruction [2]. Statistically, millions of people are diagnosed with COPD annually, with a prevalence of 9.7% in individuals aged 40 and older, significantly higher compared to 2.7% in those under 40 [3]. COPD not only impacts patients' quality of life but also leads to severe complications and mortality, ranking as the third leading cause of death globally [4] and one of the most burdensome diseases worldwide [3]. The primary risk factors for COPD include long-term smoking, air pollution, occupational dust exposure, diet, and genetics [5, 6]. Given its high prevalence and substantial burden on healthcare systems, there is an urgent need to identify and effectively manage modifiable risk factors to provide a basis for preventing and slowing the progression of this disease.

The Planetary Health Diet (PHD) is a healthy and sustainable dietary pattern proposed by the EAT-Lancet Commission on "Healthy Diets from Sustainable Food Systems" [7]. According to the EAT-Lancet report, adhering to the recommendations for a healthy and sustainable diet could prevent approximately 11 million deaths annually [7]. The Planetary Health Diet Index (PHDI) employs a progressive scoring system that takes into account all recommended values of the reference diet, serving as an effective and reliable nutritional metric for evaluating the PHD. A high PHDI score indicates a nutritionally rich and balanced diet, while a low score suggests otherwise. Diet has been reported to be linked with the development of COPD [8–11]. Diets rich in vegetables, fruits, and fish are associated with a reduced risk of developing COPD [12, 13].

Diet plays a crucial role in regulating inflammatory responses, and studies have found that diets with high inflammatory potential, as measured by the Dietary Inflammatory Index (DII), are closely associated with an increased risk of COPD [14, 15]. The DII is a tool used to assess the inflammatory potential of diets, quantifying the impact of different foods on the body's inflammatory response. A higher positive DII score indicates a more pro-inflammatory diet, while a lower negative DII score suggests an anti-inflammatory diet [16]. However, there is still a lack of in-depth research on whether DII serves as a potential mediator in the relationship between the Pro-Healthy Diet Index (PHDI) and COPD. Given that COPD is a disease highly associated with systemic chronic inflammation, exploring the mediating effect of DII in the PHDI-COPD relationship is important. This study utilizes the National Health and Nutrition Examination Survey (NHANES) database from the 2005–2018 cycle, which not only includes extensive dietary and health data but also allows for longitudinal data analysis over multiple years. This approach enables a deeper exploration of the mediation role of DII in the association between PHDI and COPD, providing more compelling evidence.

Methods

Study participants

This study included participants from the National Health and Nutrition Examination Survey (NHANES) from 2005 to 2018. NHANES is a cross-sectional survey conducted by the National Center for Health Statistics (NCHS) aimed at collecting health and nutrition information from the non-institutionalized U.S. population. Figure 1 illustrates the data selection process used in our study. Out of 70,190 participants, individuals under the age of 20 and pregnant women ($n = 40,149$) were excluded, along with those lacking PHDI ($n = 6,653$), DII ($n = 2,082$), and COPD data ($n = 2$). Ultimately, 30,304 participants with complete information were included in the study.

The study protocol was ethically approved by the NCHS Research Ethics Review Board and informed consent was obtained in writing from all adult participants, in accordance with the Declaration of Helsinki. Our secondary analysis adheres to the STROBE guidelines for cross-sectional studies [17], eliminating the need for additional Institutional Review Board approval. For more details regarding NHANES methodology and ethical considerations, please visit the CDC and NCHS websites at https://www.cdc.gov/nchs/nhanes/index.htm.

Planetary Health Diet Index

The Planetary Health Diet Index (PHDI) is a calorie-based index developed according to the dietary recommendations proposed by the EAT-Lancet Commission [7]. Its performance has been validated [18]. It includes the following fourteen food groups: dairy products, red and processed meats, poultry, eggs, fish, whole grains, whole fruits, non-starchy vegetables, nuts and seeds, legumes and unsaturated oils, starchy vegetables, added sugars, and fruit juices. While consumption of the final eight categories is discouraged, the first six categories are components of sufficiency and are encouraged for consumption. A theoretical PHDI range of 0 to 140 is

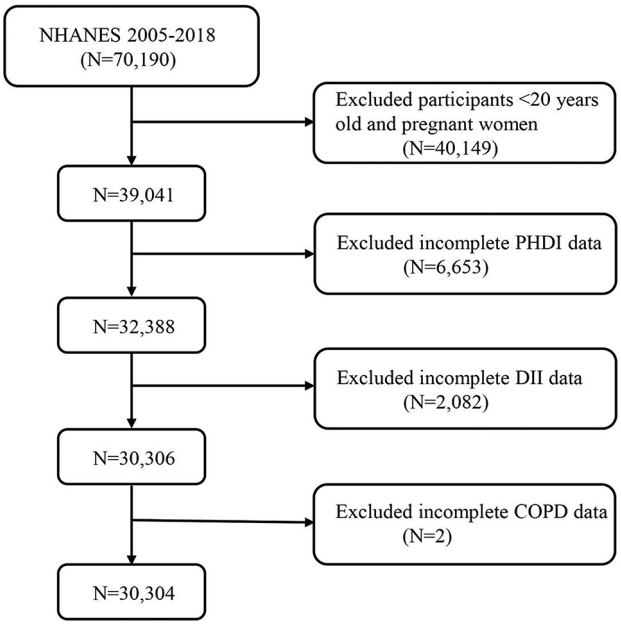

Fig. 1 A flow diagram of eligible participant selection in the National Health and Nutrition Examination Survey

obtained by assigning a score of 0 to 10 to each dietary group. Table S1 provides examples of foods and ingredients included in the PHDI components.

COPD assessment

In this study, COPD was confirmed based on the previously published Medical Conditions Questionnaire (MCQ) [19] and the ratio of Forced Expiratory Volume in 1 s (FEV1) to Forced Vital Capacity (FVC) after inhalation of a bronchodilator (FEV1/FVC < 0.70). Additionally, two questions from the MCQ, specifically MCQ160G and MCQ160K, were included: "Have you ever been told you have emphysema?" and "Have you ever been told you have chronic bronchitis?" If a participant answered "yes" to either of these questions, they were diagnosed with COPD [2, 20, 21].

Definition of DII

A crucial metric for assessing the propensity for inflammation of various food ingredients, including vitamins and minerals, is DII [15]. A diet that is pro-inflammatory has a DII score of ≥ 0; on the other hand, an anti-inflammatory diet has a score of < 0. Furthermore, harmful dietary habits are indicated by higher DII scores, whereas those that are healthier are indicated by lower DII scores. The supplemental materials contain the precise algorithm used to determine the DII score.

Covariables

Based on previous studies [22–24], we selected covariates to evaluate the potential impact of confounding factors as comprehensively as possible. These covariates include age, gender, race, marital status, education level, family poverty income ratio (PIR), obesity, hypertension, diabetes, and self-reported hyperlipidemia. For detailed information on these covariates, please refer to Table S2.

Statistical analysis

To ensure the data were representative of the national population, sampling weights were applied in all analyses. The weighting variable used in our study was the two-day dietary sample weight (WTDR2D), and we computed new weights for the 2005–2018 period as 1/7 × WTDR2D [25]. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequency (percentage). A weighted t-test was used for comparing continuous variables, and a weighted chi-square test was applied for categorical variables [26]. Weighted logistic regression was employed to explore the association between PHDI and COPD. Three logistic regression models were developed: Model 1: No adjustments for potential confounding factors. Model 2: Adjusted for covariates including age, gender, education level, marital status, PIR, and race. Model 3: Further adjustments on top of Model 2 for obesity, hypertension, diabetes, and hypercholesterolemia. Additionally, in Model 3, PHDI was treated as a continuous variable, and restricted cubic spline plots (RCS) were used to illustrate linear or non-linear associations between PHDI and COPD risk. Subsequently, a stratified subgroup analysis was performed based on Model 3, examining covariates. An interaction analysis followed to explore potential variations in the associations among subgroups.

This study conducted multiple sensitivity analyses to verify the robustness of the results. First, multiple imputation by chained equations (MICE) was performed to address missing data and repeat the primary analyses. We used multiple imputation based on five imputed datasets to estimate missing values for obesity, hypertension, diabetes, and high cholesterol [15]. Additionally, participants with asthma were excluded for further analysis to assess the potential impact of this condition on the findings. A mediation analysis was conducted based on the preconditions of a "statistically significant association between PHDI and DII" and a "statistically significant association between DII and COPD" to investigate whether the influence of PHDI on COPD occurrence was mediated by DII [15]. The R software's "mediation" package calculated the mediation effect [27]. Data processing was conducted using the R statistical software (version 4.3.1). A two-sided p-value of less than 0.05 was considered statistically significant.

Result

Baseline characteristics

This study analyzed 30,304 samples, representing approximately 138.5 million individuals, and found a COPD prevalence of 5%, closely linked to various demographic and socioeconomic factors. Higher COPD prevalence was observed among older adults, males, married individuals, and white populations. Additionally, higher education levels and non-poverty status were associated with increased COPD risk. The study also revealed that individuals with chronic hypertension and hyperlipidemia had significantly higher COPD prevalence. Notably, the COPD group exhibited higher DII levels and lower PHDI levels compared to the non-COPD group. For further details, see Table 1.

The association between PHDI and COPD

As shown in Table 2, we used three different models to assess the relationship between the Planetary Health Diet Index (PHDI) and chronic obstructive pulmonary disease (COPD). In Model 3, after adjusting for all variables, a 10-point increase in PHDI was associated with a 9% reduction in COPD prevalence [OR: 0.91 (95% CI: 0.86, 0.97)]. Additionally, in Model 3, compared to the first Tertile (T1) of PHDI, the third Tertile (T3) was associated with a 27% lower likelihood of COPD occurrence [OR: 0.73 (95% CI: 0.59, 0.90)]. Moreover, from T1 to T3, the odds ratios (ORs) steadily decreased as PHDI increased, with a significant trend (P for trend = 0.004). The results in Models 1 and 2 were similarly consistent.

As shown in Fig. 2, PHDI was significantly negatively correlated with the prevalence of COPD (overall $P < 0.001$; non-linear $P = 0.461$). Subgroup analysis indicated a consistent relationship between PHDI and COPD prevalence across all subgroups (Fig. 3).

DII and risk of COPD

Table 2 shows the relationship between COPD and DII. In model 3, after controlling for all variables, the odds of COPD rose by 38% in the third Tertile (T3) relative to the first Tertile (T1) of DII [OR = 1.38 (1.14, 1.68)]. The positive correlation between COPD and DII remained statistically significant (OR = 1.08, 95% CI: 1.02, 1.13) when DII was regarded as a continuous variable. The results of Models 1 and 2 are likewise consistent.

Association of PHDI and DII

After accounting for every covariate, Table 3 revealed a statistically significant correlation between PHDI and DII (β=-0.31, 95% CI: -0.33, -0.29, $P < 0.001$).

Mediation effect

The analysis presented above indicates that our study satisfies the requirements needed to do a mediation analysis. We saw the mediation effect of DII after adjusting for all variables (Fig. 4). The link between PHDI and COPD risk was mediated to 17.95% (mediation proportion = indirect effect / (indirect effect + direct effect) *100%, $P = 0.034$) by DII (indirect effect = -0.00128, $P = 0.032$; direct effect = -0.00585, $P = 0.028$). Consequently, it is possible to see DII as a mediating factor in the association between PHDI and the likelihood of COPD.

Sensitivity analysis

To ensure the stability of the results, this study performed multiple interpolations of missing data for obesity, hypertension, diabetes, and high cholesterol, which remained consistent with the primary results (Table S3). In addition, the results remained robust after excluding participants with asthma (Table S4).

Discussion

This study used nationally representative data from NHANES (2005–2018) to evaluate the relationship between PHDI and COPD. The findings suggest that a higher PHDI has a protective effect against COPD. Dietary patterns characterized by a high PHDI were associated with a lower prevalence of COPD. Mediation analysis indicated that DII partially mediated the relationship between the PHDI and COPD. This suggests that PHDI may influence the development of COPD by reducing the inflammatory potential of the diet.

Consistent with other studies [28], our subgroup analysis revealed that the negative correlation between PHDI and COPD varied across different racial groups. A stronger negative correlation was observed in racial groups other than Mexican Americans, non-Hispanic Whites, and non-Hispanic Blacks. These differences may reflect variations in dietary patterns among different races, and further identifying these dietary differences could be more beneficial in preventing COPD. Additionally, our study found a significant interaction effect of obesity on the relationship between PHDI and COPD, indicating that the protective effect of the PHD diet is more pronounced in non-obese individuals. Non-obese participants adhering to the PHD diet were associated with a lower prevalence of COPD, possibly due to obesity-related systemic inflammation diminishing the potential anti-inflammatory effects of PHDI. This finding suggests that an individual's weight status may play a crucial role in the effectiveness of dietary interventions, warranting further research.

Patients with COPD often experience reduced exercise tolerance, a higher risk of hospitalization, and a poor quality of life due to breathing difficulties [29]. It is also commonly believed that late-stage COPD is incurable, with treatment focusing only on controlling the symptoms [8]. In developed countries, exacerbations of COPD

Table 1 Baseline characteristics of all participants were stratified by COPD

Characteristic	Overall, *N* = 138,494,182 (100%)	Non-COPD, *N* = 131,783,335 (95%)	COPD, *N* = 6,710,847 (5%)	*P* Value
No. of participants in the sample	30,304	28,806	1,498	-
Age (%)				**< 0.001**
20–40	48,438,740 (35%)	48,093,892 (36%)	344,849 (5%)	
41–60	51,759,908 (37%)	49,069,719 (37%)	2,690,189 (40%)	
> 60	38,295,534 (28%)	34,619,725 (26%)	3,675,810 (55%)	
Gender (%)				0.124
Male	66,273,405 (48%)	62,853,418 (48%)	3,419,987 (51%)	
Female	72,220,777 (52%)	68,929,917 (52%)	3,290,860 (49%)	
Race (%)				**< 0.001**
Non-Hispanic White	96,242,789 (69%)	90,692,058 (69%)	5,550,730 (83%)	
Non-Hispanic Black	15,460,051 (11%)	14,979,384 (11%)	480,666 (7.2%)	
Other	16,442,857 (12%)	15,882,845 (12%)	560,012 (8%)	
Mexican American	10,348,486 (8%)	10,229,047 (8%)	119,439 (1.8%)	
Married/live with partner (%)				0.825
No	50,322,959 (36%)	47,860,679 (36%)	2,462,280 (37%)	
Yes	88,171,223 (64%)	83,922,656 (64%)	4,248,567 (63%)	
Education level (%)				**< 0.001**
Below high school	20,534,190 (15%)	19,129,081 (15%)	1,405,109 (21%)	
High School or above	117,959,992 (85%)	112,654,254 (85%)	5,305,738 (79%)	
PIR (%)				**< 0.001**
Not Poor	103,183,888 (80%)	98,502,572 (80%)	4,681,316 (75%)	
poor	26,284,200 (20%)	24,728,646 (20%)	1,555,554 (25%)	
Obesity (%)				0.317
No	85,395,628 (62%)	81,391,330 (62%)	4,004,299 (60%)	
Yes	51,904,120 (38%)	49,273,715 (38%)	2,630,405 (40%)	
Hypertension (%)				**< 0.001**
No	83,779,079 (60%)	80,970,018 (61%)	2,809,062 (42%)	
Yes	54,709,066 (40%)	50,807,280 (39%)	3,901,786 (58%)	
Diabetes (%)				**< 0.001**
No	118,465,984 (86%)	113,406,931 (86%)	5,059,053 (75%)	
Yes	20,025,504 (14%)	18,373,709 (14%)	1,651,795 (25%)	
High cholesterol (%)				**< 0.001**
No	76,027,325 (61%)	73,234,698 (62%)	2,792,627 (44%)	
Yes	47,682,539 (39%)	44,177,749 (38%)	3,504,790 (56%)	
PHDI (mean (SD))	61.50 (15.04)	61.53 (15.06)	60.90 (14.62)	0.337
PHDI, Tertile (%)				0.549
T1	46,167,092 (33%)	43,855,875 (33%)	2,311,217 (34%)	
T2	46,162,082 (33%)	43,871,643 (34%)	2,290,439 (34%)	
T3	46,165,009 (34%)	44,055,818 (33%)	2,109,192 (32%)	
DII (mean (SD))	1.00 (1.71)	0.99 (1.71)	1.24 (1.75)	**< 0.001**
DII, Tertile (%)				**< 0.001**
T1	46,150,966 (33%)	44,242,483 (34%)	1,908,483 (28%)	
T2	46,165,474 (34%)	44,070,143 (33%)	2,095,330 (32%)	
T3	46,177,743 (33%)	43,470,709 (33%)	2,707,034 (40%)	

Mean (SD) for continuous variables: the P value was calculated by the weighted Students T-test

Percentages (weighted N, %) for categorical variables: the P value was calculated by the weighted chi-square test

Abbreviation: PHDI, Planetary Health Diet Index; DII, dietary inflammatory index; COPD, Chronic obstructive pulmonary disease; PIR, Ratio of family income to poverty

represent one of the largest burdens on healthcare systems. In the European Union, the prevalence of COPD, already very high, is likely to continue increasing until at least 2030 [30]. To reduce COPD patients' suffering and alleviate the societal burden of COPD, it is crucial to shift the focus of interventions from alleviating symptoms, exacerbations, and complications of advanced disease to preventing the onset of COPD.

The prevalence and mortality of COPD are directly related to the prevalence of smoking [31–33]. However,

Table 2 Association between PHDI, DII, and COPD, NHANES 2005–2018

Characteristics	Model 1 [OR (95% CI)]	*p*-value	Model 2 [OR (95% CI)]	*p*-value	Model 3 [OR (95% CI)]	*p*-value
PHDI - COPD						
Continuous (per 10 scores)	0.89 (0.84, 0.94)	< 0.001	0.91 (0.85, 0.96)	< 0.001	0.91 (0.86, 0.97)	0.002
Tertile						
T1	1 (ref.)		1 (ref.)		1 (ref.)	
T2	0.83 (0.69, 1.00)	0.054	0.86 (0.72, 1.03)	0.093	0.85 (0.70, 1.03)	0.100
T3	0.69 (0.57, 0.85)	< 0.001	0.72 (0.59, 0.88)	0.002	0.73 (0.59, 0.90)	0.004
P for trend	< 0.001		0.002		0.004	
DII - COPD						
Continuous	1.12 (1.07, 1.17)	< 0.001	1.09 (1.04, 1.15)	< 0.001	1.08 (1.02, 1.13)	0.004
Tertile						
T1	1 (ref.)		1 (ref.)		1 (ref.)	
T2	1.17 (0.96, 1.41)	0.110	1.15 (0.95, 1.39)	0.200	1.09 (0.89, 1.34)	0.400
T3	1.61 (1.35, 1.92)	< 0.001	1.46 (1.20, 1.77)	< 0.001	1.38 (1.14, 1.68)	0.002
P for trend	< 0.001		< 0.001		0.002	

Model 1: no covariates were adjusted

Model 2: age, gender, education level, marital, PIR, and race were adjusted

Model 3: age, gender, education level, marital, PIR, race, obesity, hypertension, diabetes, and high cholesterol were adjusted

Abbreviation: PHDI, Planetary Health Diet Index; DII, dietary inflammatory index; COPD, Chronic obstructive pulmonary disease; PIR, Ratio of family income to poverty; OR, odds ratio; CI, confidence interval

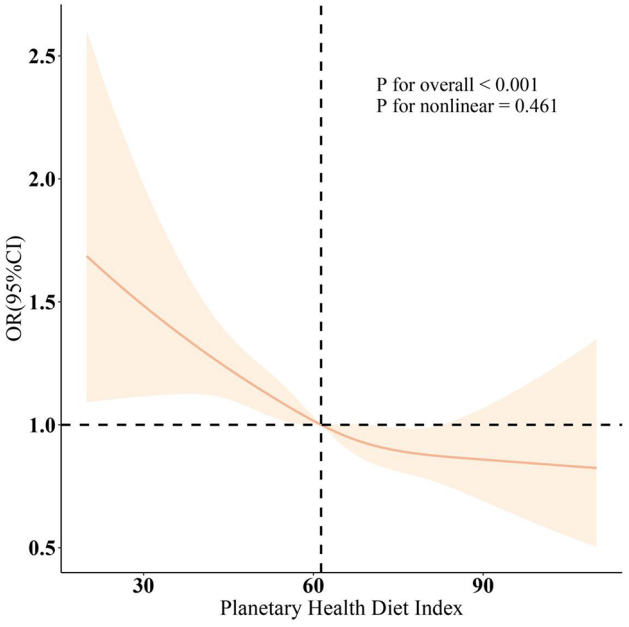

Fig. 2 Dose–response relationships between PHDI and COPD. OR (solid lines) and 95% confidence levels (shaded areas) were adjusted for age, gender, education level, marital, PIR, race, obesity, hypertension, diabetes, and high cholesterol

smoking is not the only risk factor for COPD [34]. Although the GOLD guidelines do not explicitly identify diet as a risk factor for COPD, studies have shown that malnutrition, inadequate caloric intake, insufficient protein consumption, and poor quality of life in outpatient settings are associated with increased COPD prevalence [10, 35]. Research exploring the relationship between dietary patterns and COPD indicates that a "Western" diet, characterized by low fiber content and rich in refined grains, red and processed meats, fried potatoes, eggs, and soft drinks, is positively associated with the prevalence of COPD. In contrast, a "prudent" diet, characterized by high fiber intake and rich in vegetables, fruits, whole grains, and fish, is negatively associated with COPD prevalence [36–39]. These findings suggest that these two dietary patterns may be linked to the risk of developing COPD.

Our study is the first to investigate the relationship between adherence to the EAT-Lancet recommendations for a sustainable diet and the prevalence of COPD. The Planetary Health Diet Index (PHDI) is a vital scoring tool within EAT-Lancet, encompassing components such as nuts, fruits, vegetables, whole grains, fish, and dairy. According to relevant studies, each gram increase in total fiber intake (up to 25 g per day) reduces COPD risk by 3% (95% CI 2%-5%) [40]. The findings of our study indicate that the PHDI among COPD patients is lower than that of participants without COPD (60.90 vs. 61.53, $p < 0.001$). The results from adjusted models also showed a significant negative association between PHDI and COPD prevalence among participants. This suggests that increasing the PHDI plays an important role in reducing the prevalence of COPD. It reminds us to consider the types of foods included in the PHDI in our daily lives to minimize the risk of developing COPD.

We consider that the mechanisms explaining the negative correlation between PHDI and COPD prevalence are multifaceted and complex. The primary mechanisms include: (1) Anti-inflammatory and Antioxidant Effects of Dietary Fiber: Dietary fiber influences the gut microbiota,

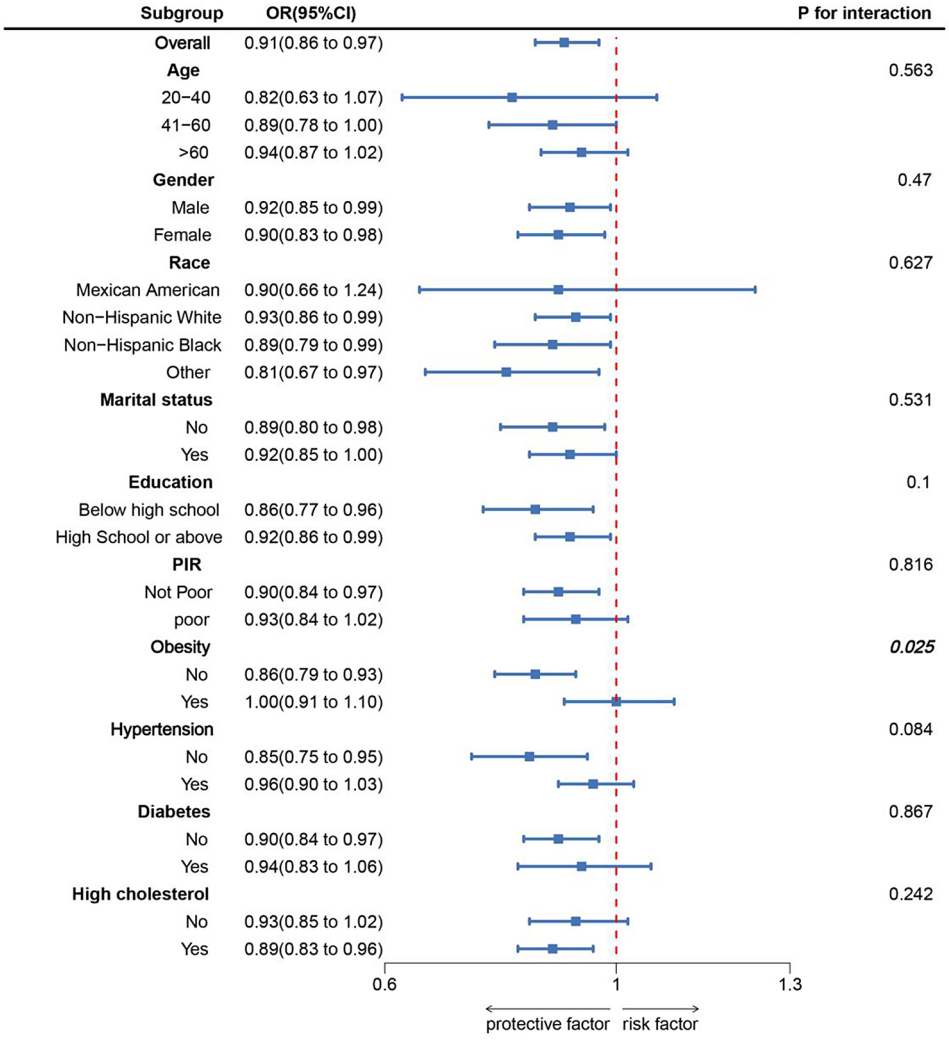

Fig. 3 Subgroup analysis between PHDI and COPD. ORs were calculated as per 10 scores increase in PHDI. Analyses were adjusted for age, gender, education level, marital, PIR, race, obesity, hypertension, diabetes, and high cholesterol

Table 3 Multivariate linear regression of PHDI and DII

	β	95%CI	*P*-value
PHDI - DII	-0.31	(-0.33, -0.29)	< 0.001

Adjusted for age, gender, education level, marital, PIR, race, obesity, hypertension, diabetes, and high cholesterol

which in turn affects the host immune system. Fermentation of dietary fiber by gut symbiotic bacteria produces a large amount of short-chain fatty acids (SCFAs, such as acetate, propionate, and butyrate) related to immune response [41, 42]. These SCFAs are not confined to the gut; they are absorbed into the bloodstream and reach every organ in the body. Studies suggest that circulating SCFAs can reduce the inflammatory response in lung tissue through activation of free fatty acid receptors and/or epigenetic regulation [43–45]. Other studies indicate that SCFAs can lower pro-inflammatory mediators (like IL-6, and CRP) [44, 46]. Dietary fiber might also reduce oxidative stress in the lungs through antioxidants present in vitamin-rich foods (e.g., spinach, and carrots) [47, 48]. (2) Malnutrition Due to Inappropriate Diet: Research shows that COPD is often associated with chronic muscle wasting, which worsens with age and poor dietary patterns, where the rate of protein degradation far exceeds the synthesis rate. Many COPD patients may also experience increased circulating leptin levels, which negatively affect dietary intake, in turn impacting the quality and function of respiratory muscles [49]. These studies and findings collectively highlight the significant role of PHDI in COPD, providing strong theoretical support for our research.

Inflammatory responses play a crucial role in the pathophysiology of COPD [50], and recent studies have shown that dietary intake can influence inflammation levels [51]. DII, as a measure of the inflammatory effects of diet, can quantify the association between diet and

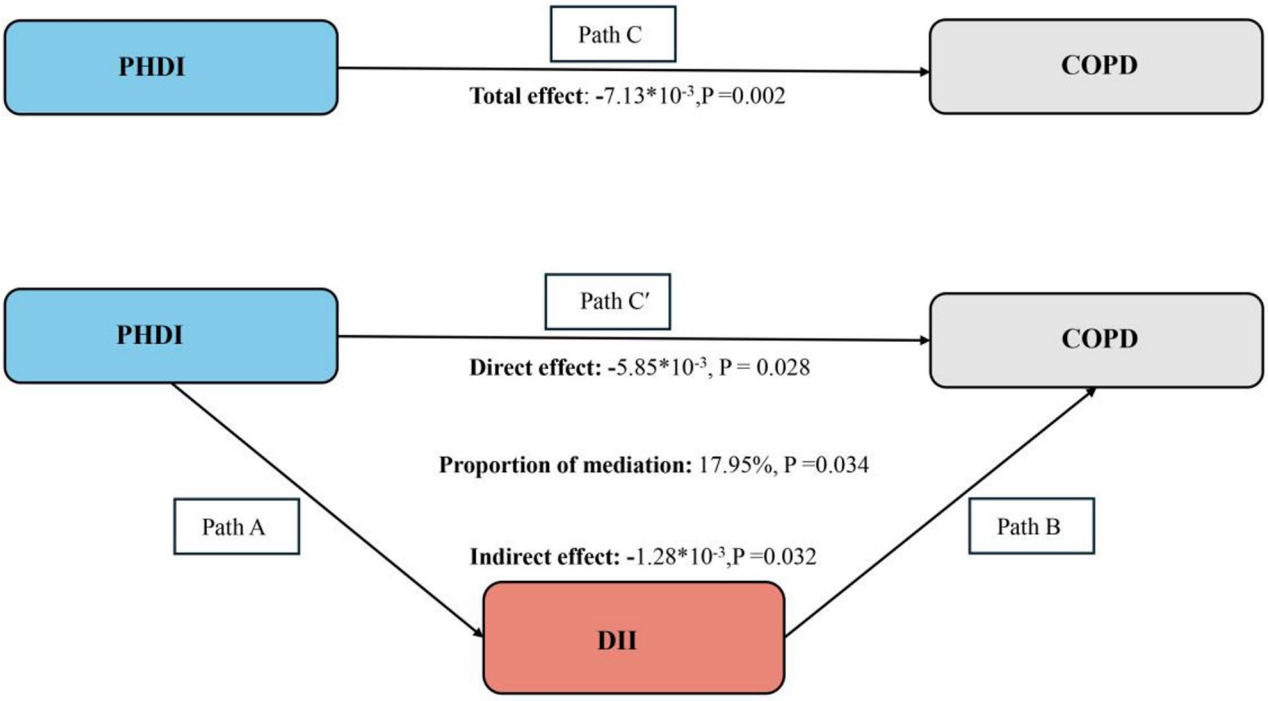

Fig. 4 Schematic diagram of the mediation effect analysis. Path C indicates the total effect; path C′ indicates the direct effect. The indirect effect is estimated as the multiplication of paths A and B (path A*B). The mediated proportion is calculated as indirect effect/ (indirect effect + direct effect) × 100%. Abbreviation: PHDI, Planetary Health Diet Index; DII, dietary inflammatory index; COPD, Chronic obstructive pulmonary disease. Analyses were adjusted for age, gender, education level, marital, PIR, race, obesity, hypertension, diabetes, and high cholesterol

COPD risk. PHDI promotes a diet rich in plant-based, minimally processed foods, which are high in antioxidants and anti-inflammatory components. These dietary elements can reduce systemic inflammation by lowering DII values, potentially offering protective effects against COPD. Reducing inflammation not only helps prevent the onset of COPD but may also slow its progression. Furthermore, the current international research focus leans towards the integrated prevention and control of chronic diseases in conjunction with environmental factors. The importance of PHDI has gained increased attention, emphasizing the significance of sustainable diets for public health [7]. Thus, studying the role of DII as a mediator in the relationship between PHDI and COPD aligns with global research trends in healthful and environmentally friendly dietary strategies. This approach not only elucidates the mechanisms by which dietary pattern improvements affect respiratory health but also offers innovative dietary intervention pathways for COPD prevention and management, bearing substantial academic and public health value. Given the high prevalence of COPD and its severe impact on quality of life, exploring the potential of dietary interventions is particularly important. By reducing the inflammatory potential of diets, it may be possible to alleviate symptoms in COPD patients and reduce the economic burden and consumption of healthcare resources. Therefore, in-depth research into the mediating role of DII between PHDI and COPD not only aids in uncovering the complex relationship between diet and respiratory diseases but also provides a scientific foundation for developing diet-based COPD prevention and management strategies.

This study demonstrated that DII mediated 17.95% of the association between PHDI and COPD, suggesting that dietary inflammatory potential plays a significant role in this relationship. Mechanistically, high-PHDI diets are typically rich in anti-inflammatory and antioxidant components, such as dietary fiber, vitamins C and E [52], and polyphenols, which may reduce systemic inflammation markers (e.g., C-reactive protein, IL-6, and TNF-α) and support lung health. DII reflects the inflammatory potential of a diet, and higher DII levels may exacerbate systemic inflammation and oxidative stress [53], both of which contribute to COPD pathophysiology. Inflammation may damage alveolar epithelial cells, promote airway remodeling, and degrade pulmonary elastic fibers, further worsening COPD outcomes. However, the partial mediation observed suggests that PHDI influences COPD risk through additional pathways beyond DII. For instance, high-PHDI diets may positively modulate the gut microbiome, increasing the production of short-chain fatty acids, which have been shown to support lung function. Furthermore, the antioxidant properties of high-PHDI diets may independently reduce oxidative

stress, mitigating lung tissue damage. Other unmeasured factors, such as environmental exposures, genetic predispositions, or socioeconomic status, may also play a role in the PHDI-COPD relationship. Future research should incorporate multiple potential mediators to better understand the comprehensive impact of PHDI on COPD risk.

The study possesses several strengths. Firstly, it is the first to utilize PHDI to predict the risk of COPD, which holds significant clinical implications and aligns with global sustainable development goals. Secondly, by employing data from the National Health and Nutrition Examination Survey (NHANES) spanning 2005 to 2018, the study uses a nationally representative weighted sample, enhancing the generalizability and applicability of the findings. Thirdly, the research incorporates mediation analysis to explore the role of DII in the relationship between PHDI and COPD, providing a more detailed understanding of the underlying mechanisms. Lastly, comprehensive consideration and control of potential confounding factors ensure the robustness and reliability of the study results.

This study, based on cross-sectional data from the NHANES database, provides valuable insights into the association between the Planetary Health Diet Index (PHDI) and chronic obstructive pulmonary disease (COPD). However, several limitations should be acknowledged. (1) The cross-sectional design of the study does not allow for the determination of causal relationships between PHDI and COPD. (2) dietary data were collected using 24-hour dietary recall methods, which are susceptible to recall bias and social desirability effects, potentially impacting the accuracy of PHDI scores. Additionally, the PHDI calculation is derived from global dietary recommendations, which, while universal in intent, may not fully capture region-specific dietary patterns. For instance, dietary staples and cultural food preferences prevalent in certain populations may not align with the recommendations used to compute the PHDI. Future research should consider integrating cultural dietary characteristics to improve the index's applicability across diverse populations. (3) There are numerous risk factors for COPD, and although we attempted to include as many confounding factors as possible, it is still possible that known or unknown risks were not captured. (4) The NHANES database represents primarily the U.S. population, limiting the generalizability of our findings to other regions. Differences in dietary habits, socioeconomic conditions, and healthcare systems may contribute to variability in the PHDI-COPD relationship. Thus, future studies using multi-regional and multi-ethnic datasets are necessary to validate these findings and explore region-specific differences.

Conclusion

The study indicates that DII plays a significant mediating role in the relationship between PHDI and COPD. This finding offers a novel perspective on the mechanisms linking dietary patterns and dietary inflammation markers with respiratory health issues. Future prospective cohort studies and fundamental experiments could be conducted to explore the underlying mechanisms further, thereby guiding dietary recommendations for COPD patients and promoting the sustainability of both planetary and human health. These insights provide new strategies for personal health management and offer scientific evidence for formulating public health policies.

Abbreviations

PHDI	Planetary Health Diet Index
DII	Dietary inflammatory index
COPD	Chronic obstructive pulmonary disease
NHANES	National Health and Nutrition Examination Survey

Supplementary Information

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Supplementary Material 1

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Author contributions

H.G. contributed to the original draft, Methodology, and Formal analysis. K.Z. contributed to the original draft, Methodology, Supervision, Project administration, and Formal analysis. S.C. contributed to Conceptualization, Methodology, Validation, Formal analysis, Resources, and Data curation. S.H. was involved in Writing– review & editing, Supervision, Project administration, and Investigation.

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Data availability

The corresponding author can provide the datasets used and/or analyzed in this study upon reasonable request.

Declarations

Ethics approval and consent to participate

The NCHS Ethics Review Board approved this study's human subjects components, which followed the Declaration of Helsinki. For every subject, written informed permission was acquired.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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References

1. Christenson SA, Smith BM, Bafadhel M, Putcha N. Chronic obstructive pulmonary disease. Lancet. 2022;399:2227–42.
2. Rabe KF, Hurd S, Anzueto A, Barnes PJ, Buist SA, Calverley P, et al. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. Am J Respir Crit Care Med. 2007;176:532–55.
3. Halbert RJ, Natoli JL, Gano A, Badamgarav E, Buist AS, Mannino DM. Global burden of COPD: systematic review and meta-analysis. Eur Respir J. 2006;28:523–32.
4. The top 10. causes of death [Internet]. [cited 2024 Jul 22]. Available from: https://www.who.int/news-room/fact-sheets/detail/the-top-10-causes-of-death
5. Yang IA, Jenkins CR, Salvi SS. Chronic obstructive pulmonary disease in never-smokers: risk factors, pathogenesis, and implications for prevention and treatment. Lancet Respir Med. 2022;10:497–511.
6. Mannino DM, Buist AS. Global burden of COPD: risk factors, prevalence, and future trends. Lancet. 2007;370:765–73.
7. Willett W, Rockström J, Loken B, Springmann M, Lang T, Vermeulen S, et al. Food in the Anthropocene: the EAT-Lancet Commission on healthy diets from sustainable food systems. Lancet. 2019;393:447–92.
8. Hsieh M-J, Yang T-M, Tsai Y-H. Nutritional supplementation in patients with chronic obstructive pulmonary disease. J Formos Med Assoc. 2016;115:595–601.
9. Beijers RJHCG, Steiner MC, Schols AMWJ. The role of diet and nutrition in the management of COPD. Eur Respiratory Review: Official J Eur Respiratory Soc. 2023;32:230003.
10. Nguyen HT, Collins PF, Pavey TG, Nguyen NV, Pham TD, Gallegos DL. Nutritional status, dietary intake, and health-related quality of life in outpatients with COPD. Int J Chron Obstruct Pulmon Dis. 2019;14:215–26.
11. Shaheen SO, Jameson KA, Syddall HE, Aihie Sayer A, Dennison EM, Cooper C, et al. The relationship of dietary patterns with adult lung function and COPD. Eur Respir J. 2010;36:277–84.
12. Baldrick FR, Elborn JS, Woodside JV, Treacy K, Bradley JM, Patterson CC, et al. Effect of fruit and vegetable intake on oxidative stress and inflammation in COPD: a randomised controlled trial. Eur Respir J. 2012;39:1377–84.
13. Kaluza J, Larsson SC, Orsini N, Linden A, Wolk A. Fruit and vegetable consumption and risk of COPD: a prospective cohort study of men. Thorax. 2017;72:500–9.
14. Wang S, Wang Y, Hu X, Lu L. Association between dietary inflammation index and asthma COPD overlap. Sci Rep. 2024;14:8077.
15. Wu R, Gong H. The association between non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio and chronic obstructive pulmonary disease: the mediating role of dietary inflammatory index. Front Nutr. 2024;11:1427586.
16. Mo T, Wei M, Fu J. Dietary inflammatory index and type 2 diabetes in US women: a cross-sectional analysis of the National Health and Nutrition Examination Survey, 2007–2018. Front Nutr. 2024;11:1455521.
17. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP, et al. The strengthening the reporting of Observational studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Lancet. 2007;370:1453–7.
18. Cacau LT, De Carli E, de Carvalho AM, Lotufo PA, Moreno LA, Bensenor IM, et al. Development and validation of an Index based on EAT-Lancet recommendations: the Planetary Health Diet Index. Nutrients. 2021;13:1698.
19. Shi Y, Zhang J, Huang Y. Prediction of cardiovascular risk in patients with chronic obstructive pulmonary disease: a study of the National Health and Nutrition Examination Survey database. BMC Cardiovasc Disord. 2021;21:417.
20. Güder G, Brenner S, Angermann CE, Ertl G, Held M, Sachs AP, et al. GOLD or lower limit of normal definition? A comparison with expert-based diagnosis of chronic obstructive pulmonary disease in a prospective cohort-study. Respir Res. 2012;13:13.
21. Vollmer A, Vollmer M, Lang G, Straub A, Shavlokhova V, Kübler A, et al. Associations between Periodontitis and COPD: an Artificial Intelligence-based analysis of NHANES III. J Clin Med. 2022;11:7210.
22. Chen C, Yang T, Wang C. The Dietary Inflammatory Index and early COPD: results from the National Health and Nutrition Examination Survey. Nutrients. 2022;14:2841.
23. Liu H, Tan X, Liu Z, Ma X, Zheng Y, Zhu B, et al. Association between Diet-Related Inflammation and COPD: findings from NHANES III. Front Nutr. 2021;8:732099.
24. Liang X, Chou OHI, Cheung BM. The Association between systemic arterial hypertension and chronic obstructive Pulmonary Disease. Results from the U.S. National Health and Nutrition Examination Survey 1999–2018: a cross-sectional study. Chronic Obstr Pulm Dis. 2023;10:190–8.
25. Huang S, He Q, Wang X, Choi S, Gong H. Associations of the planetary health diet index (PHDI) with asthma: the mediating role of body mass index. BMC Public Health. 2024;24:2305.
26. Feng G, Huang S, Zhao W, Gong H. Association between life's essential 8 and overactive bladder. Sci Rep. 2024;14:11842.
27. Wu Y, Tan Z, Zhen J, Liu C, Zhang J, Liao F, et al. Association between diet soft drink consumption and metabolic dysfunction-associated steatotic liver disease: findings from the NHANES. BMC Public Health. 2023;23:2286.
28. Saeed MA, Gribben KC, Alam M, Lyden ER, Hanson CK, LeVan TD. Association of Dietary Fiber on Asthma, respiratory symptoms, and inflammation in the Adult National Health and Nutrition Examination Survey Population. Ann Am Thorac Soc. 2020;17(9):1062–8.
29. O'Donnell DE, Milne KM, James MD, de Torres JP, Neder JA. Dyspnea in COPD: new mechanistic insights and management implications. Adv Ther. 2020;37:41–60.
30. Gibson GJ, Loddenkemper R, Lundbäck B, Sibille Y. Respiratory health and disease in Europe: the new European lung White Book. Eur Respir J. 2013;42(3):559–63.
31. Menezes AMB, Perez-Padilla R, Jardim JRB, Muiño A, Lopez MV, Valdivia G, et al. Chronic obstructive pulmonary disease in five latin American cities (the PLATINO study): a prevalence study. Lancet (London England). 2005;366:1875–81.
32. Martinez FJ, Han MK, Allinson JP, Barr RG, Boucher RC, Calverley PMA, et al. At the Root: defining and halting progression of early chronic obstructive Pulmonary Disease. Am J Respir Crit Care Med. 2018;197:1540–51.
33. Fukuchi Y, Nishimura M, Ichinose M, Adachi M, Nagai A, Kuriyama T, et al. COPD in Japan: the Nippon COPD Epidemiology study. Respirology (Carlton. Vic). 2004;9:458–65.
34. Celli BR, Halbert RJ, Nordyke RJ, Schau B. Airway obstruction in never smokers: results from the Third National Health and Nutrition Examination Survey. Am J Med. 2005;118:1364–72.
35. Chai X, Chen Y, Li Y, Chi J, Guo S. Lower geriatric nutritional risk index is associated with a higher risk of all-cause mortality in patients with chronic obstructive pulmonary disease: a cohort study from the National Health and Nutrition Examination Survey 2013–2018. BMJ open Respiratory Res. 2023;10:e001518.
36. Brigham EP, Steffen LM, London SJ, Boyce D, Diette GB, Hansel NN, et al. Diet Pattern and respiratory morbidity in the atherosclerosis risk in communities Study. Ann Am Thorac Soc. 2018;15:675–82.
37. Varraso R, Fung TT, Hu FB, Willett W, Camargo CA. Prospective study of dietary patterns and chronic obstructive pulmonary disease among US men. Thorax. 2007;62:786–91.
38. Varraso R, Fung TT, Barr RG, Hu FB, Willett W, Camargo CA. Prospective study of dietary patterns and chronic obstructive pulmonary disease among US women. Am J Clin Nutr. 2007;86:488–95.
39. Grooms KN, Ommerborn MJ, Pham DQ, Djoussé L, Clark CR. Dietary fiber intake and cardiometabolic risks among US adults, NHANES 1999–2010. Am J Med. 2013;126:1059–e10671.
40. Szmidt MK, Kaluza J, Harris HR, Linden A, Wolk A. Long-term dietary fiber intake and risk of chronic obstructive pulmonary disease: a prospective cohort study of women. Eur J Nutr. 2020;59:1869–79.
41. Cronin P, Joyce SA, O'Toole PW, O'Connor EM. Dietary Fibre modulates the gut microbiota. Nutrients. 2021;13:1655.
42. Greenberg NA, Gassull MA, Meier R. Short-chain fatty acids: ready for prime time? Nutr Clin Pract. 2006;21(6):639–40; author reply 640.
43. Wood LG. Diet, obesity, and Asthma. Annals Am Thorac Soc. 2017;14:S332–8.
44. Marsland BJ. Regulation of inflammatory responses by the commensal microbiota. Thorax. 2012;67:93–4.

45. Trompette A, Gollwitzer ES, Yadava K, Sichelstiel AK, Sprenger N, Ngom-Bru C, et al. Gut microbiota metabolism of dietary fiber influences allergic airway disease and hematopoiesis. Nat Med. 2014;20:159–66.
46. Maslowski KM, Vieira AT, Ng A, Kranich J, Sierro F, Yu D, et al. Regulation of inflammatory responses by gut microbiota and chemoattractant receptor GPR43. Nature. 2009;461:1282–6.
47. Saura-Calixto F. Dietary fiber as a carrier of dietary antioxidants: an essential physiological function. J Agric Food Chem. 2011;59:43–9.
48. Palafox-Carlos H, Ayala-Zavala JF, González-Aguilar GA. The role of dietary fiber in the bioaccessibility and bioavailability of fruit and vegetable antioxidants. J Food Sci. 2011;76:R6–15.
49. Wüst RCI, Degens H. Factors contributing to muscle wasting and dysfunction in COPD patients. Int J Chron Obstruct Pulmon Dis. 2007;2:289–300.
50. Wang Y, Xu J, Meng Y, Adcock IM, Yao X. Role of inflammatory cells in airway remodeling in COPD. COPD. 2018;13:3341–8.
51. Calle MC, Andersen CJ. Assessment of dietary patterns represents a potential, yet variable, measure of inflammatory status: a review and update. Dis Markers. 2019;2019:3102870.
52. Huang S, Hu H, Gong H. Association between the Planetary Health Diet Index and biological aging among the U.S. population. Front Public Health. 2024;12:1482959.
53. Fang B, Wang Z, Nan G. Dietary inflammatory potential and the risk of cognitive impairment: a meta-analysis of prospective cohort studies. J Nutr Health Aging. 2025;29:100428.

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