Original Research

Association Between Dietary Fiber Intake and Prevalence of Chronic Obstructive Pulmonary Disease in a Middle-Aged and Elderly Population: a Study Based on the National Health and Nutrition Examination Survey Database

Jun Jin,[1] Yuemei Bian,[2] Zhongyun Gu,[3] Maoen Lin[1]

[1]Department of Respiratory Medicine, Hangzhou Ninth People's Hospital, Hangzhou City, Zhejiang Province, China

[2]Clinical Nutrition Department, Hangzhou Ninth People's Hospital, Hangzhou City, Zhejiang Province, China

[3]General Surgery, Hangzhou Ninth People's Hospital, Hangzhou City, Zhejiang Province, China

Address correspondence to:
Maoen Lin
Department of Respiratory Medicine
Hangzhou Ninth People's Hospital
No. 98, Yilong Road, Yipong Street
Qiantang District, Hangzhou City
Zhejiang Province
311225, China
Phone: 8613868387799
Email: maoenlinlin@163.com

***Running Head*: Dietary Fiber Intake and Prevalence of COPD**

Keywords: chronic obstructive pulmonary disease; dietary fiber intake; logistic regression; NHANES; white blood cell counts

Abbreviations:

Funding Support: None

***Date of Acceptance*:** February 5, 2024 | ***Publication Online Date*:** February 14, 2024

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

Citation: Jin J, Bian Y, Gu Z, Lin M. Association between dietary fiber intake and prevalence of chronic obstructive pulmonary disease in a middle-aged and elderly population: a study based on the National Health and Nutrition Examination Survey database. *Chron Obstr Pulm Dis.* 2024; Published online February 14, 2024. https://doi.org/10.15326/jcopdf.2023.0457

This article has an online supplement.

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

Abstract

Objective: This study aimed to investigate dietary fiber (DF) intake with prevalence of chronic obstructive pulmonary disease (COPD) in the middle-aged and elderly population through analysis of the National Health and Nutrition Examination Survey (NHANES) data.

Methods: The study utilized data from three cycles of NHANES database (2007-2012). The exposure variable was DF intake, and the outcome variable was COPD prevalence. Weighted logistic regression was utilized to construct relationship models between the two variables. Confounding factors were adjusted, and subgroup analysis was to explore association of DF intake with COPD. Restricted cubic spline (RCS) analysis investigated non-linear relationship between DF intake and COPD. Finally, mediation analysis was performed to determine whether the influence of DF intake on COPD prevalence is mediated through alteration of white blood cell (WBC) counts.

Results: This study included a total of 7,301 eligible participants aged >40 years. The results of the study indicated that an increase in DF intake significantly reduced the prevalence of COPD (OR: 0.98, 95% CI: 0.96-0.99, $p<0.001$), and DF intake was correlated with lung function indicators (FEV1). Stratified analysis revealed that an increased DF intake significantly reduced the risk of COPD in male individuals, middle-aged individuals (aged 40-59 years), those with BMI≤30 kg/m^2, individuals with a history of smoking, and alcohol consumers ($p<0.05$). Through RCS analysis exploring the nonlinear association between DF intake and COPD prevalence, the critical threshold for the impact of DF intake on COPD prevalence was 15.10 gm. When DF intake was ≥15.10 g/d, it effectively reduced the prevalence of COPD. Mediation analysis results indicated that the WBC count partially mediated the association between DF intake and COPD, with a mediation proportion of 9.89% ($p=0.006$).

Conclusion: Increased DF intake was linked to decreased prevalence of COPD, particularly in men and middle-aged people. WBC counts may be an important pathway linking DF intake and COPD.

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

1. Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a common disease[1], characterized by inflammation and remodeling of the lower respiratory tract and lung parenchyma, as well as the activation of inflammatory and immune processes[2]. COPD is progressive and not only manifests in the lungs but is also associated with disease development in other parts of the body, including increased risks of cardiovascular diseases, osteoporosis, and depression, severely impacting people's physical health[3, 4]. In 2019, there were approximately 212.3 million COPD cases globally, leading to 3.3 million deaths[5], making it the third primary cause of death globally after coronary heart disease and stroke[6]. COPD is more prevalent in the middle-aged and elderly population, with a prevalence rate of up to 13.7% in individuals over the age of 40. COPD affects a large population, and unfortunately, even with optimal treatment, patients experience periodic exacerbations leading to a decrease in lung function and quality of life, elevated death risk, and higher treatment costs[7]. Currently, available drug treatments for COPD have shown limited effectiveness, emphasizing the importance of identifying effective preventive and therapeutic factors to lower prevalence of COPD.

Smoking is a key environmental risk factor for COPD. Nevertheless, despite sharing a similar smoking history, not everyone gets COPD. Therefore, genetic susceptibility, environmental pollution[8], and even dietary[9] factors are believed to affect prevalence of COPD. A high dietary fiber (DF) intake lowers lung inflammation and risk of developing COPD through its antioxidant and anti-inflammatory properties[10]. DF increases concentration of short-chain fatty acids (SCFAs) by altering composition of the gut microbiota[11]. These byproducts exist in the systemic circulation and can protect lung function and prevent COPD by regulating macrophages, neutrophils[12], and alleviating pulmonary inflammation[13]. Several prospective studies[14-16] have reported the association between DF intake and the prevalence of COPD. A large-scale prospective study conducted in the United States indicated that, after adjusting for age, gender, and other confounding factors in a multivariate model, the risk of newly diagnosed COPD decreases with an increase in total fiber intake[16]. Moreover, similar results are observed in gender-specific analyses.

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

A study involving a large cohort of men (45,058 participants) found a robust negative correlation between fiber intake and COPD among current smokers/ex-smokers[14]. Another cohort study focusing on women also found a negative correlation between high DF intake and the risk of COPD. The study explored the negative correlation between total DF intake and the risk of COPD in patients with different smoking statuses (current, former smokers, never smokers)[15]. However, the above studies only examined the relationship between DF and COPD prevalence and did not delve into the potential mediating effect of DF intake on COPD prevalence.

Therefore, based on previous studies, we utilized data from three cycles (2007-2012) of the National Health and Nutrition Examination Survey (NHANES) to investigate the relationship between DF intake and COPD prevalence. Furthermore, we conducted a detailed analysis of the relationships among DF intake, white blood cell (WBC) count, and COPD prevalence. Understanding the importance of dietary factors in respiratory health can provide insights for future public health interventions aimed at preventing COPD in the middle-aged and elderly population.

2. Methods

2.1 Data source and study population

NHANES database, conducted by the National Center for Health Statistics (NCHS) in the United States, was used as the data source (http://www.cdc.gov/nchs/nhanes.htm). NHANES is a stratified, multistage study that combines interviews, physical examinations, and laboratory tests to assess health and nutritional status of the U.S. population. The database is freely accessible and has been approved by the Institutional Review Board of the NCHS, with informed consent obtained from the participants.

This study selected information from 30,442 respondents across three consecutive cycles (2007-2012) in the database. The exposure variable in the research was DF intake, and the outcome variable was the occurrence of COPD. The study initially involved data collection from 11,763 respondents aged 40 and above. Exclusions were made for 3,805 individuals lacking COPD diagnostic or DF intake information, as well as 657 cases with missing covariates (gender, race,

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

BMI, smoking and drinking status, platelet count, neutrophil count, lymphocyte count, White Blood Cell (WBC) count, monocyte count, eosinophil count, and basophil count). Ultimately, 7,301 respondents with complete and qualifying information were included in the study. The detailed participant selection process is illustrated in Figure 1.

2.2 DF intake

Data collection for DF intake was based on 24-hour dietary recall questionnaire in NHANES database. Dietary recall of the participants was conducted twice: first during Mobile Examination Center (MEC) visit and then through a telephone follow-up 3 to 10 days later. The DF intake of the participants was evaluated based on the USDA's Food and Nutrient Database for Dietary Studies (FNDDS). The data from both recalls were averaged for participants with no missing data, and the DF intake was log-transformed to approximate a normal distribution[17].

2.3 COPD

In this study, COPD was confirmed through a medical conditions questionnaire and the forced expiratory volume in one second (FEV1)/forced vital capacity (FVC) ratio after bronchodilator inhalation. In the NHANES data collection questionnaire, the presence of COPD was determined if respondent answered "yes" to either of the two questions: "Have you ever been told that you have chronic bronchitis?" or "Have you ever been told that you have emphysema?" Additionally, a diagnosis of COPD was made if FEV1/FVC ratio after bronchodilator inhalation was less than 0.70[1, 18].

2.4 Covariates

Gender, age, race, BMI, smoking and alcohol consumption status, platelet count, neutrophil count, eosinophil count, lymphocyte count, WBC count, monocyte count, and basophil count were identified as potential confounding factors in this study. COPD is a major public health issue in individuals aged 40 and above; therefore, this study only included participants aged 40 and above[1,

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

[19]. The age of the participants was categorized as middle-aged (40-59 years) and older adults (≥60 years). Male and female were used to categorize gender. Race was categorized as Mexican American, other Hispanic, non-Hispanic white, non-Hispanic black, and other races. BMI was classified as ≤25 kg/m^2, 25-30 kg/m^2, or >30 kg/m^2. Smoking status was categorized as "now smoking," "former smoking," or "never smoked"[20]. Alcohol consumption was determined by the response to the question "Had at least 12 alcohol drinks/1 yr?" with options of "yes" or "no" (one drink refers to 12 ounces of beer, 5 ounces of wine, or 1.5 ounces of distilled spirits)[21]. Complete blood cell count (CBC) with WBC differential was tested by VCS method. The Beckman Coulter MAXM instrument at the MEC provided CBC information and blood cell distribution for the participants[22]. The collected CBC information included platelet count, WBC count, lymphocyte count, neutrophil count, basophil count, monocyte count, and eosinophil count.

2.5 Statistical analysis

In this study, the 'tableone' package was used to generate baseline tables. The sample size and proportions of categorical variables, as well as mean and standard deviation of continuous variables (unweighted n; n(%), mean, and SD adjusted for weights), were calculated and presented per the presence or absence of COPD. The 'survey' package was utilized to construct weighted logistic regression models for association of DF intake with COPD in the middle-aged and older adult population. Separate weighted logistic regression models were developed for continuous and categorical variables in the unadjusted model, and stratified analysis was performed for categorical variables. DF intake was stratified into quartiles, and a weighted logistic regression model adjusted for confounding factors was constructed using the 'survey' package to assess association of DF intake with COPD, incorporating stratified analysis and baseline table discrepancies for gender subgroup analysis. Restricted cubic splines (RCS) were employed in the unadjusted and adjusted weighted logistic regression models to dissect association of DF intake with COPD. The 'mediation' package was utilized to examine possible mediating effect of DF intake on COPD, with bootstrap testing conducted to estimate mediation proportions and corresponding 95% CIs

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

for each of the 1000 bootstrapped samples in the unadjusted model. The models in this study included: Crude (unadjusted); model I (adjusted for gender, age, and race); model II (adjusted for gender, age, race, BMI, smoking, and alcohol consumption status); and model III (adjusted for gender, age, race, BMI, smoking, alcohol consumption status, platelet count, WBC count). R (V4.2.1) was used for all statistical calculations, and a two-sided *p*-value <0.05 was regarded as statistically significant.

3. Results

3.1 Baseline characteristics of participants

The distribution of baseline characteristics among 7,301 participants aged 40 years and above is presented in Table 1. The proportion of males (47.8%) was lower than females (52.2%), with middle-aged (40-59 years) and elderly (≥60 years) individuals accounting for 67.1% and 32.9%, respectively. Prevalence of COPD among participants was 13.2%. Clinical characteristics of the participants stratified by presence of COPD revealed significant statistical differences (p<0.05) in gender, race, age, smoking status, neutrophil count, WBC count, and eosinophil count among groups. COPD patient population was predominantly elderly individuals (78.3%) and non-Hispanic White (82.0%). Baseline results showed that the mean DF intake of COPD patients (16.05 ± 8.06) was significantly lower than that of non-COPD patients (17.73 ± 8.86) (p<0.001).

3.2 Association of CBC/DF intake with COPD

A weighted logistic regression model was employed to assess association of DF intake with COPD. Results without adjusting for any confounding factors are presented in Table 2. According to the data, there was a significant positive correlation between WBC count (OR: 1.08, 95% CI: 1.03-1.12, p<0.001) and the prevalence of COPD. Additionally, an increase in DF intake was associated with a significant reduction in the risk of COPD (OR: 0.98, 95% CI: 0.96-0.99, p<0.001). Furthermore, we explored the relationship between DF intake and lung function (indicating the severity of COPD), as shown in the supplementary table. We found a positive correlation between

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

DF intake and FEV1 (OR: 1.02, 95% CI: 1.01-1.04, $p<0.001$), with no statistical significance in FEV1/FVC ($p>0.05$).

3.3 Stratified analysis

Stratified analysis of the weighted logistic regression model for DF intake and COPD was conducted based on gender, race, age, smoking status, BMI, and alcohol consumption, as shown in Table 3. Among males, middle-aged individuals (40-59 years), individuals with BMI≤30 kg/m^2, those with a history of smoking, and drinkers, an increased DF intake was significantly linked to reduced prevalence of COPD ($p<0.05$).

3.4 Subgroup analysis

As presented in Table 4, in unstratified population, the Crude model (OR: 0.98, 95% CI: 0.96-0.99, $p<0.001$), model I (OR: 0.97, 95% CI: 0.96-0.99, $p<0.001$), model II (OR: 0.98, 95% CI: 0.97-1.00, $p<0.05$), and model III (OR: 0.98, 95% CI: 0.97-1.00, $p<0.05$) all indicated a significant reduction in prevalence of COPD with increased DF intake. Furthermore, impact of DF intake levels on prevalence of COPD was investigated and compared to first quartile (Q1) of DF intake, any quartile in the four models significantly reduced the risk of COPD ($p<0.05$). Moreover, there was a trend in prevalence of COPD with changes in quartile intervals of DF intake ($p<0.05$). Furthermore, when considering the overall population, participants in the second to fourth quartiles (Q2-Q4) of DF intake exhibited an approximately 35% lower probability of developing COPD compared to those in the first quartile (Q1). Subgroup analysis based on gender (Table 5) showed that in males, the Crude model (OR: 0.97, 95% CI: 0.96-0.99, $p<0.001$), model I (OR: 0.97, 95% CI: 0.96-0.99, $p<0.001$), model II (OR: 0.98, 95% CI: 0.97-1.00, $p<0.05$), and model III (OR: 0.98, 95% CI: 0.97-1.00, $p<0.05$) all showed a significant decrease in prevalence of COPD with increased DF intake, while no significant relation was seen in the female population.

3.5 Nonlinear association of DF intake with prevalence of COPD

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

Figure 2 depicts the dose-response relationship between DF intake and the prevalence of COPD. Results from RCS curves indicated a non-linear association detected in both the Crude model (p for non-linearity =0.0326) and Model I (p for non-linearity =0.002). However, this non-linear association disappeared in Model II and Model III, which were further adjusted for confounding factors (p for non-linearity >0.05). Across all four models in the RCS curves, there was a consistent negative correlation between DF intake and the prevalence of COPD, suggesting a stable association between the two. Additionally, based on the data, the critical threshold for the impact of DF intake on COPD prevalence was 15.10 gm. When DF intake exceeded 15.10 gm, it could effectively lower the prevalence of COPD.

3.6 Mediation analysis

As depicted in Figure 3, unadjusted model indicated a significant effect of DF intake on COPD (p<0.001). The mediation effect generated through WBC count was also highly significant in relation to prevalence of COPD (p=0.002), indicating that WBC count partially mediated association of DF intake with COPD, with a mediation proportion of 9.89% (p=0.006). Therefore, we believed that the increase in DF intake may lower prevalence of COPD by regulating WBC count.

4. Discussion

In this study, we used data from the nationally representative NHANES (2007-2012) to assess the association between DF intake and the prevalence of COPD. The results suggested that DF intake was associated with a reduction in the prevalence of COPD, especially in middle-aged and elderly men, and may be related to the severity of COPD. Mediation analysis showed that increased DF intake may reduce the prevalence of COPD by regulating WBC count.

The pathophysiology of COPD involves various inflammatory cell types, including macrophages[23], neutrophils[24], and T cells[25], which coordinate and sustain the inflammatory response in the lungs in response to toxic gases. In addition to pulmonary reactions, chronic

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

systemic low-grade inflammation is also commonly observed in COPD patients[26, 27]. Studies have shown that acute-phase proteins such as CRP, fibrinogen, and various proinflammatory cytokines such as IL-1β[28], chemokines[29], and TNF-α[30] increase in COPD patients, especially as the disease progresses, and the complications caused by these factors are main causes of death in COPD patients[31].

DF is an important component of healthy diet, and increasing evidence suggests its significant role in various chronic diseases[32, 33]. In a previous cross-sectional study, participants with the highest intake of DF had a 63% lower risk of increased CRP concentrations than those with the lowest total fiber intake[34]. This finding was confirmed in another survey study[35]. Earlier studies have suggested a positive correlation between a "Western" dietary (high in refined grains, red and processed meats, fried potatoes, eggs, and soft drinks) with low fiber content and COPD risk[36], while a "prudent" dietary featured by high fiber intake (abundant in vegetables, fruits, whole grain foods, and fish) was negatively associated with COPD prevalence[37]. The results of this study indicated that participants in the Q2-Q4 DF intake quartiles had a probability of developing COPD approximately 35% lower than those in Q1. This finding is consistent with previous research, highlighting the significant role of increased DF intake in reducing the prevalence of COPD. Furthermore, our study revealed a correlation between DF intake and the severity of COPD, showing a positive association with FEV1. This aligns with existing literature, which links low DF intake to decreased lung function indicators and increased rates of airflow limitation in participants[38]. It is also consistent with the severity of airway damage in elderly male COPD patients[39]. On the contrary, higher DF intake is associated with improved lung function and reduced prevalence of COPD[40]. These data further underscore the potential role of a diet rich in fiber-rich foods in enhancing pulmonary health.

In this study, the difference in fiber intake between participants with and without COPD was approximately 1.7 g/day. Simultaneously, the results of the RCS curve analysis indicated that the critical threshold for the impact of DF intake on COPD prevalence was 15.10 g. When DF intake exceeded 15.10 g, it could effectively lower the prevalence of COPD, while lower intake may

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

potentially increase the risk of COPD occurrence. Additionally, based on findings from related studies, each 1g increase in total fiber intake (up to 25 g/day) is associated with a 3% reduction in COPD risk (95% CI 2-5%)[15]. The data in this study indicated that individuals with COPD (16.05 g/day) and those without COPD (17.73 g/day) had DF intake within a similar range. We reasonably speculated that there might be a critical threshold for DF intake within this range that contributed to the occurrence of COPD. This further suggests that increasing DF intake may have a significant impact on reducing the prevalence of COPD.

We speculated that the potential mechanism by which DF protects against COPD may be related to its anti-inflammatory effects in regulating systemic inflammation. DF can influence the levels of chronic inflammation by altering intestinal pH, reducing membrane permeability, and activating GPCRs. SCFAs from the fermentation of DF play a crucial role in the anti-inflammatory process[41]. DF is the main source of SCFAs in the intestines, with acetate, propionate, and butyrate being the highest in concentration in the human body[42]. Numerous studies have shown that SCFAs decrease production of pro-inflammatory mediators and increase release of anti-inflammatory mediators, thus mitigating inflammation. For example, butyrate can inhibit the production of pro-inflammatory mediators in lipopolysaccharide (LPS) and cytokines, including TNF-α, IL-6[43], and nitric oxide[44], while increasing release of anti-inflammatory cytokine IL-10[45]. An animal experimental study demonstrated that oral administration of butyrate effectively lowers concentrations of TNF-α, IL-1β, and nitric oxide in bronchoalveolar lavage fluid and decreases alveolar hemorrhage and neutrophil infiltration in mice with acute lung injury induced by tracheal instillation of LPS[46]. Other studies have indicated that butyrate and propionate inhibit TNF-α secretion and NF-κB activity, suppress expression of the anti-inflammatory cytokine IL-10 in LPS-activated monocytes and neutrophils through activation of GPCRs and suppression of HDAC[47-50]. Acetate can inhibit LPS-induced TNF-α and IL-6 secretion in human monocytes by activating free fatty acid receptors[51].

Furthermore, association of DF intake with COPD was controversial when considering gender differences. Previous studies presented a significant negative correlation between total DF

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

intake and COPD in female populations (RR=0.62, 95% CI: 0.46-0.85; p<0.01)[16]. In contrast, in our study, the relationship between the two variables was not significant in the female population (OR: 0.97, 95% CI: 0.94-1.00; p=0.069). However, a study conducted on a male population (HR=0.62; 95% CI: 0.50-0.78, p<0.0001) showed results congruous with our study trend (OR: 0.97, 95% CI: 0.96-0.99; p<0.001)[14]. In the results mentioned above, it is evident that association of DF intake with prevalence of COPD differs by gender. This difference may be attributed to factors such as tobacco use rates and variations in DF intake among different populations. Studies have found a significant protective impact of DF against COPD in populations with a history of smoking, but no such association was observed in never-smokers[14, 15], and the prevalence of tobacco use is higher in males than females[52]. Additionally, research conducted on the elderly population has found that inadequate DF intake is more common in males[53]. Therefore, ensuring an adequate DF intake is particularly effective in reducing prevalence of COPD in middle-aged males. However, the mechanisms underlying the gender differences are still unclear and require further detailed experiments for exploration.

COPD is a chronic disease that progresses to systemic inflammation. As inflammation continues, the concentration of pro-inflammatory cytokines in lung tissues and systemic serum gradually increases, along with enhanced oxidative stress. Activated WBCs and inflammatory markers such as TNF-α significantly elevate in COPD patients[54]. The primary cause of chronic inflammation in COPD is the recruitment of WBC subsets, neutrophils, and lymphocytes. Once activated, neutrophils release elastase, tissue proteases, matrix metalloproteinases, and myeloperoxidase (MPO), which actively participate in pathological mechanisms of emphysema and COPD, leading to the destruction of lung tissues in COPD patients[55]. Current research has focused on potential neutrophilic inflammation in COPD, and studies have demonstrated a significant relationship between the two[56]. Additionally, eosinophils have been proposed as a personalized clinical biomarker for reducing inhaled corticosteroid (ICS) treatment in COPD patients[8]. Eosinophils, composed of bilobed nuclei and large cytoplasmic granules, are inflammatory WBCs that can be recruited to the lungs under certain conditions to participate in

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

the inflammatory response in COPD[57]. They have the capacity to synthesize and release chemokines (such as CCL5, CCL11, CCL13), growth factors (TGF), cytokines (IL-2, IL-3, etc.)[58-60], and cytotoxic granule proteins (mainly basic proteins, eosinophil peroxidase, eosinophil cationic protein, eosinophil-derived neurotoxin)[61], which exert pro-inflammatory effects. Our results displayed a significant positive correlation between neutrophil count, WBC count, eosinophil count, and the prevalence of COPD. Consequently, WBC count plays an extremely pivotal role in COPD diagnosis and treatment, and increased WBC count is considered an independent criterion for antibiotic treatment in exacerbated COPD patients in most clinical guidelines[62].

In the stratified analysis of the relationship model between DF intake and the prevalence of COPD after adjusting for confounding factors in this study, an issue arises where the effect sizes appear similar, but the confidence intervals are wide. This could be attributed to small sample sizes in certain subgroups, potentially leading to insufficient statistical power and influencing the significance of some effects. Moreover, based on the data from the model, the impact of DF on COPD prevalence in different population strata shows relatively minor variations, indicating a need for exploration within the broader cohort of the overall population. Considering the generality of our study results in the entire population and the model constructed after adjusting for numerous confounding factors related to COPD, as well as the exploration through intermediate analysis of the associations among DF intake, WBC count, and COPD, this study provides valuable guidance for our future research endeavors.

However, the study has limitations. Firstly, being a cross-sectional study, it cannot establish a causal relationship between DF intake and COPD. Secondly, the calculation of DF intake relies on data obtained from dietary questionnaire surveys, which may be subject to memory bias. Additionally, the DF intake of COPD patients may be restricted due to the impact of the disease (e.g., patients with severe airway damage may find food preparation and consumption more challenging due to respiratory difficulties and fatigue). Moreover, during the follow-up period, COPD diagnosis was based on post-bronchodilator values and a fixed FEV1/FVC ratio, as well as

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

self-reporting. However, the study cannot entirely rule out the possibility of misdiagnosis leading to underdiagnosis or overdiagnosis of COPD in some subjects. Finally, COPD is influenced by numerous factors, and despite including as many relevant covariates as possible in the model, the study cannot eliminate the impact of unmeasured or residual covariates. Despite these limitations, our research demonstrates a negative correlation between increased DF intake and COPD prevalence.

5. Conclusion

Through an in-depth analysis of three consecutive cycles of nationally representative NHANES data from 2007 to 2012, we have explored the relationship between DF intake and the prevalence of COPD. The results indicated a significant reduction in the prevalence of COPD with an increase in DF intake. Stratified analysis revealed that the increased intake of DF significantly reduced the risk of COPD in populations with a history of smoking, alcohol consumption, middle-aged and elderly individuals, as well as those with a BMI ≤ 30 kg/m^2. RCS analysis explored the dose-response relationship between DF and the prevalence of COPD, suggesting an effective reduction in COPD prevalence when DF intake > 15.10 grams. Mediation analysis delved into the association between DF intake and COPD, suggesting that the increased intake of DF may reduce the risk of COPD by modulating WBC count. This study provided new insights into understanding the relationship between DF and COPD. Furthermore, through mediation analysis of WBC count, we revealed that DF may influence the development of COPD by regulating systemic inflammation levels. This offers a crucial foundation for future in-depth research and provides valuable insights for developing dietary strategies beneficial in preventing COPD.

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

Declaration

Author contribution

(I) Conception and design: Jun Jin

(II) Administrative support: Yuemei Bian

(III) Provision of study materials or patients: Zhongyun Gu

(IV) Collection and assembly of data: Jun Jin, Maoen Lin

(V) Data analysis and interpretation: Yuemei Bian

(VI) Manuscript writing: Jun Jin, Zhongyun Gu

(VII) Final approval of manuscript: All authors

Conflict of interest statement

The authors declare that they have no conflict of interest.

Ethics approval

Not applicable

Data availability statement

The data in this study are available from the corresponding author on reasonable request.

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

Reference

1. 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.
2. Uwagboe I, Adcock IM, Lo Bello F, Caramori G, Mumby S. New drugs under development for COPD. Minerva Med. 2022; 113:471-96.
3. Erhabor GE, Adeniyi B, Arawomo AO, Akinwalere O, Adetona G, Fagbohun FT, et al. Acute Exacerbation of COPD: Clinical Perspectives and Literature Review. West Afr J Med. 2021; Vol. 38:1129-42.
4. Agusti A, Soriano JB. COPD as a systemic disease. COPD. 2008; 5:133-8.
5. Safiri S, Carson-Chahhoud K, Noori M, Nejadghaderi SA, Sullman MJM, Ahmadian Heris J, et al. Burden of chronic obstructive pulmonary disease and its attributable risk factors in 204 countries and territories, 1990-2019: results from the Global Burden of Disease Study 2019. BMJ. 2022; 378:e069679.
6. Organization WH. The top 10 causes of death. 2020; Available from: https://www.who.int/news-room/fact-sheets/detail/the-top-10-causes-of-death.
7. Burt L, Corbridge S. COPD exacerbations. Am J Nurs. 2013; 113:34-43; quiz 4.
8. Agusti A, Celli BR, Criner GJ, Halpin D, Anzueto A, Barnes P, et al. Global Initiative for Chronic Obstructive Lung Disease 2023 Report: GOLD Executive Summary. Am J Respir Crit Care Med. 2023; 207:819-37.
9. Schols AM, Ferreira IM, Franssen FM, Gosker HR, Janssens W, Muscaritoli M, et al. Nutritional assessment and therapy in COPD: a European Respiratory Society statement. Eur Respir J. 2014; 44:1504-20.
10. 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.
11. 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.
12. Jang YO, Kim OH, Kim SJ, Lee SH, Yun S, Lim SE, et al. High-fiber diets attenuate emphysema development via modulation of gut microbiota and metabolism. Sci Rep. 2021; 11:7008.
13. Vaughan A, Frazer ZA, Hansbro PM, Yang IA. COPD and the gut-lung axis: the therapeutic potential of fibre. J Thorac Dis. 2019; 11:S2173-S80.
14. Kaluza J, Harris H, Wallin A, Linden A, Wolk A. Dietary Fiber Intake and Risk of Chronic Obstructive Pulmonary Disease: A Prospective Cohort Study of Men. Epidemiology. 2018; 29:254-60.
15. 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.

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

16. Varraso R, Willett WC, Camargo CA, Jr. Prospective study of dietary fiber and risk of chronic obstructive pulmonary disease among US women and men. Am J Epidemiol. 2010; 171:776-84.
17. Zhang HR, Yang Y, Tian W, Sun YJ. Dietary Fiber and All-Cause and Cardiovascular Mortality in Older Adults with Hypertension: A Cohort Study Of NHANES. J Nutr Health Aging. 2022; 26:407-14.
18. Vollmer A, Vollmer M, Lang G, Straub A, Shavlokhova V, Kubler A, et al. Associations between Periodontitis and COPD: An Artificial Intelligence-Based Analysis of NHANES III. J Clin Med. 2022; 11.
19. Andre S, Conde B, Fragoso E, Boleo-Tome JP, Areias V, Cardoso J, et al. COPD and Cardiovascular Disease. Pulmonology. 2019; 25:168-76.
20. Zhang Y, Liu W, Zhang W, Cheng R, Tan A, Shen S, et al. Association between blood lead levels and hyperlipidemiais: Results from the NHANES (1999-2018). Front Public Health. 2022; 10:981749.
21. Dong X, Li S, Sun J, Li Y, Zhang D. Association of Coffee, Decaffeinated Coffee and Caffeine Intake from Coffee with Cognitive Performance in Older Adults: National Health and Nutrition Examination Survey (NHANES) 2011-2014. Nutrients. 2020; 12.
22. Liu B, Wang J, Li YY, Li KP, Zhang Q. The association between systemic immune-inflammation index and rheumatoid arthritis: evidence from NHANES 1999-2018. Arthritis Res Ther. 2023; 25:34.
23. Huang H, Feng H, Zhuge D. M1 Macrophage Activated by Notch Signal Pathway Contributed to Ventilator-Induced Lung Injury in Chronic Obstructive Pulmonary Disease Model. J Surg Res. 2019; 244:358-67.
24. Hoenderdos K, Condliffe A. The neutrophil in chronic obstructive pulmonary disease. Am J Respir Cell Mol Biol. 2013; 48:531-9.
25. Dancer R, Sansom DM. Regulatory T cells and COPD. Thorax. 2013; 68:1176-8.
26. Walter RE, Wilk JB, Larson MG, Vasan RS, Keaney JF, Jr., Lipinska I, et al. Systemic inflammation and COPD: the Framingham Heart Study. Chest. 2008; 133:19-25.
27. Sin DD, Man SF. Why are patients with chronic obstructive pulmonary disease at increased risk of cardiovascular diseases? The potential role of systemic inflammation in chronic obstructive pulmonary disease. Circulation. 2003; 107:1514-9.
28. Obling N, Backer V, Hurst JR, Bodtger U. Nasal and systemic inflammation in Chronic Obstructive Pulmonary Disease (COPD). Respir Med. 2022; 195:106774.
29. Traves SL, Culpitt SV, Russell RE, Barnes PJ, Donnelly LE. Increased levels of the chemokines GROalpha and MCP-1 in sputum samples from patients with COPD. Thorax. 2002; 57:590-5.
30. Yao Y, Zhou J, Diao X, Wang S. Association between tumor necrosis factor-alpha and chronic obstructive pulmonary disease: a systematic review and meta-analysis. Ther Adv Respir Dis. 2019; 13:1753466619866096.
31. Cavailles A, Brinchault-Rabin G, Dixmier A, Goupil F, Gut-Gobert C, Marchand-Adam S, et al. Comorbidities of COPD. Eur Respir Rev. 2013; 22:454-75.
32. Soliman GA. Dietary Fiber, Atherosclerosis, and Cardiovascular Disease. Nutrients. 2019; 11.

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

33. Ranganathan N, Anteyi E. The Role of Dietary Fiber and Gut Microbiome Modulation in Progression of Chronic Kidney Disease. Toxins (Basel). 2022; 14.
34. Ma Y, Griffith JA, Chasan-Taber L, Olendzki BC, Jackson E, Stanek EJ, 3rd, et al. Association between dietary fiber and serum C-reactive protein. Am J Clin Nutr. 2006; 83:760-6.
35. Grooms KN, Ommerborn MJ, Pham DQ, Djousse L, Clark CR. Dietary fiber intake and cardiometabolic risks among US adults, NHANES 1999-2010. Am J Med. 2013; 126:1059-67 e1-4.
36. Bakolis I, Hooper R, Thompson RL, Shaheen SO. Dietary patterns and adult asthma: population-based case-control study. Allergy. 2010; 65:606-15.
37. 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.
38. Hanson C, Lyden E, Rennard S, Mannino DM, Rutten EP, Hopkins R, et al. The Relationship between Dietary Fiber Intake and Lung Function in the National Health and Nutrition Examination Surveys. Ann Am Thorac Soc. 2016; 13:643-50.
39. Kim T, Choi H, Kim J. Association Between Dietary Nutrient Intake and Chronic Obstructive Pulmonary Disease Severity: A Nationwide Population-Based Representative Sample. COPD. 2020; 17:49-58.
40. Kan H, Stevens J, Heiss G, Rose KM, London SJ. Dietary fiber, lung function, and chronic obstructive pulmonary disease in the atherosclerosis risk in communities study. Am J Epidemiol. 2008; 167:570-8.
41. Swann OG, Kilpatrick M, Breslin M, Oddy WH. Dietary fiber and its associations with depression and inflammation. Nutr Rev. 2020; 78:394-411.
42. Malesza IJ, Malesza M, Walkowiak J, Mussin N, Walkowiak D, Aringazina R, et al. High-Fat, Western-Style Diet, Systemic Inflammation, and Gut Microbiota: A Narrative Review. Cells. 2021; 10.
43. Zhou T, Xu H, Cheng X, He Y, Ren Q, Li D, et al. Sodium Butyrate Attenuates Diabetic Kidney Disease Partially via Histone Butyrylation Modification. Mediators Inflamm. 2022; 2022:7643322.
44. Vinolo MA, Rodrigues HG, Hatanaka E, Sato FT, Sampaio SC, Curi R. Suppressive effect of short-chain fatty acids on production of proinflammatory mediators by neutrophils. J Nutr Biochem. 2011; 22:849-55.
45. Saemann MD, Bohmig GA, Osterreicher CH, Burtscher H, Parolini O, Diakos C, et al. Anti-inflammatory effects of sodium butyrate on human monocytes: potent inhibition of IL-12 and up-regulation of IL-10 production. FASEB J. 2000; 14:2380-2.
46. Ni YF, Wang J, Yan XL, Tian F, Zhao JB, Wang YJ, et al. Histone deacetylase inhibitor, butyrate, attenuates lipopolysaccharide-induced acute lung injury in mice. Respir Res. 2010; 11:33.
47. Chang PV, Hao L, Offermanns S, Medzhitov R. The microbial metabolite butyrate regulates intestinal macrophage function via histone deacetylase inhibition. Proc Natl Acad Sci U S A. 2014; 111:2247-52.
48. Usami M, Kishimoto K, Ohata A, Miyoshi M, Aoyama M, Fueda Y, et al. Butyrate and

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

trichostatin A attenuate nuclear factor kappaB activation and tumor necrosis factor alpha secretion and increase prostaglandin E2 secretion in human peripheral blood mononuclear cells. Nutr Res. 2008; 28:321-8.
49. Kaisar MMM, Pelgrom LR, van der Ham AJ, Yazdanbakhsh M, Everts B. Butyrate Conditions Human Dendritic Cells to Prime Type 1 Regulatory T Cells via both Histone Deacetylase Inhibition and G Protein-Coupled Receptor 109A Signaling. Front Immunol. 2017; 8:1429.
50. Yang W, Liu H, Xu L, Yu T, Zhao X, Yao S, et al. GPR120 Inhibits Colitis Through Regulation of CD4(+) T Cell Interleukin 10 Production. Gastroenterology. 2022; 162:150-65.
51. Eslick S, Williams EJ, Berthon BS, Wright T, Karihaloo C, Gately M, et al. Weight Loss and Short-Chain Fatty Acids Reduce Systemic Inflammation in Monocytes and Adipose Tissue Macrophages from Obese Subjects. Nutrients. 2022; 14.
52. Eisenbaum E, DiNitto DM, Bishop-Fitzpatrick L. Gender differences in tobacco use among U.S. Special Olympics athletes. Disabil Health J. 2018; 11:466-70.
53. Silva GMD, Assumpcao D, Barros MBA, Barros Filho AA, Corona LP. [Low intake of dietary fibers among the elderly: 2014/2015 ISACAMP population-based study]. Cien Saude Colet. 2021; 26:3865-74.
54. van Iersel LEJ, Beijers R, Gosker HR, Schols A. Nutrition as a modifiable factor in the onset and progression of pulmonary function impairment in COPD: a systematic review. Nutr Rev. 2022; 80:1434-44.
55. Paliogiannis P, Fois AG, Sotgia S, Mangoni AA, Zinellu E, Pirina P, et al. Neutrophil to lymphocyte ratio and clinical outcomes in COPD: recent evidence and future perspectives. Eur Respir Rev. 2018; 27.
56. Coban Agca M, Aksoy E, Duman D, Ozmen I, Yildirim E, Gungor S, et al. Does eosinophilia and neutrophil to lymphocyte ratio affect hospital re-admission in cases of COPD exacerbation? Tuberk Toraks. 2017; 65:282-90.
57. David B, Bafadhel M, Koenderman L, De Soyza A. Eosinophilic inflammation in COPD: from an inflammatory marker to a treatable trait. Thorax. 2021; 76:188-95.
58. Moqbel R, Levi-Schaffer F, Kay AB. Cytokine generation by eosinophils. J Allergy Clin Immunol. 1994; 94:1183-8.
59. Davoine F, Lacy P. Eosinophil cytokines, chemokines, and growth factors: emerging roles in immunity. Front Immunol. 2014; 5:570.
60. Shimizu S, Takezawa-Yasuoka K, Ogawa T, Tojima I, Kouzaki H, Shimizu T. The epidermal growth factor receptor inhibitor AG1478 inhibits eosinophilic inflammation in upper airways. Clin Immunol. 2018; 188:1-6.
61. Guilpain P, Guillevin L, Mouthon L. [Eosinophil granule cationic proteins: eosinophil activation markers]. Rev Med Interne. 2006; 27:406-8.
62. Laue J, Reierth E, Melbye H. When should acute exacerbations of COPD be treated with systemic corticosteroids and antibiotics in primary care: a systematic review of current COPD guidelines. NPJ Prim Care Respir Med. 2015; 25:15002.

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

Table 1. Distribution of characteristics in COPD and non-COPD patients

Characteristics	Total	Non-COPD	COPD	*p*-value
Overall	7301	6410 (86.8)	891 (13.2)	
Gender, N (%)				**0.022**
Female	3666 (52.2)	3253 (52.9)	413 (60.0)	
Male	3635 (47.8)	3157 (47.1)	478 (40.0)	
Age, (years), N (%)				**<0.001**
40-59	4123 (67.1)	3721 (68.6)	402 (21.7)	
≥60	3178 (32.9)	2689 (31.4)	489 (78.3)	
Race, N (%)				**<0.001**
Mexican American	1084 (6.3)	1033 (7.0)	51 (1.9)	
Other Hispanic	802 (4.6)	740 (4.9)	62 (2.0)	
Non-Hispanic White	3377 (73.8)	2795 (72.3)	582 (82.0)	
Non-Hispanic Black	1594 (10.2)	1438 (10.6)	156 (13.9)	
Other races	444 (5.1)	404 (5.2)	40 (0.2)	
BMI, (kg/m^2), N (%)				0.399
≤25	1698 (25.4)	1461 (25.1)	237 (38.1)	
25-30	2626 (36.0)	2334 (36.4)	292 (32.3)	
>30	2977 (38.6)	2615 (38.5)	362 (29.6)	
Smoking, N (%)				**<0.001**
Never smoked	3692 (51.1)	3449 (54.3)	243 (30.6)	
Former smoking	2143 (29.9)	1822 (29.2)	321 (41.1)	
Now Smoking	1466 (19.0)	1139 (16.4)	327 (28.2)	
Alcohol consumption, N (%)				0.252
No	2020 (21.7)	1827 (22.0)	193 (28.3)	
Yes	5281 (78.3)	4583 (78.0)	698 (71.7)	
Platelets, (10^9 cells/L), mean (SD)	248.22 (66.95)	248.10 (67.23)	249.04 (65.13)	0.810
Neutrophils, (10^9 cells/L), mean (SD)	4.23 (1.68)	4.20 (1.69)	4.48 (1.59)	**<0.001**
Lymphocyte, (10^9 cells/L), mean (SD)	2.06 (0.74)	2.06 (0.75)	2.08 (0.70)	0.493
WBC count, (10^9 cells/L), mean (SD)	7.07 (2.14)	7.02 (2.15)	7.39 (2.00)	**<0.001**
Monocyte, (10^9 cells/L), mean (SD)	0.54 (0.20)	0.53 (0.20)	0.55 (0.18)	0.082
Eosinophils, (10^9 cells/L), mean (SD)	0.20 (0.17)	0.19 (0.17)	0.23 (0.18)	**<0.001**
Basophils, (10^9 cells/L), mean (SD)	0.04 (0.07)	0.04 (0.07)	0.05 (0.06)	0.131
Dietary fiber, (gm), mean(SD)	17.51 (8.77)	17.73 (8.86)	16.05 (8.06)	**<0.001**

Note: Categorical variables are presented as n (%), and continuous variables are presented as mean (sd); n is unweighted, while n (%), mean, and sd are weighted. The % calculation for the COPD column is the result after weight adjustment.

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

Table 2. Logistic regression models of CBC/dietary fiber and COPD

Outcomes	OR (95% CI)	*p*-value
Platelets	1.00 (1.00, 1.00)	0.8
WBC count	**1.08 (1.03, 1.12)**	**<0.001**
Lymphocyte	1.04 (0.93, 1.17)	0.5
Neutrophils	**1.10 (1.05, 1.16)**	**<0.001**
Monocyte	1.42 (0.95, 2.12)	0.078
Eosinophils	**2.50 (1.43, 4.39)**	**<0.001**
Basophils	3.20 (0.36, 28.3)	0.3
Dietary fiber	**0.98 (0.96, 0.99)**	**<0.001**

Note: No adjustment for any confounding factors.

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

Table 3. Relationship models between dietary fiber and COPD stratified by variables

Participants	OR	95% CI1	*p*-value
Gender, N (%)			
Female	0.97	0.94-1.00	0.069
Male	0.97	0.96-0.99	<0.001
Age, (years), N (%)			
40-59	0.97	0.95-0.99	0.002
≥60	0.99	0.97-1.00	0.110
Race, N (%)			
Mexican American	0.97	0.93-1.01	0.100
Other Hispanic	0.97	0.93-1.02	0.200
Non-Hispanic White	0.98	0.96-0.99	<0.001
Non-Hispanic Black	0.96	0.93-0.98	<0.001
Other races	0.99	0.92-1.06	0.700
BMI, (kg/m^2), N (%)			
≤25	0.97	0.95-1.00	0.022
25-30	0.97	0.95-1.00	0.013
>30	0.98	0.96-1.00	0.064
Smoking, N (%)			
Never smoked	0.99	0.96-1.01	0.200
Former smoking	0.98	0.96-1.00	0.016
Now Smoking	0.99	0.97-1.02	0.500
Alcohol consumption, N (%)			
No	0.98	0.94-1.01	0.200
Yes	0.98	0.96-0.99	<0.001

Note: No adjustment for any confounding factors.

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

Table 4. Relationship models between dietary fiber intake and COPD risk adjusted for different confounding factors

	OR (95% CI)			
Participants	**Crude**	**Model I**	**Model II**	**Model III**
All participants	0.98 (0.96-0.99) ***	0.97 (0.96-0.99) ***	0.98 (0.97-1.00) *	0.98 (0.97-1.00) *
Dietary fiber (gm)				
Q1 (≤10.65)	Ref.	Ref.	Ref.	Ref.
Q2 (10.65-15.30)	0.66 (0.52-0.83) ***	0.61 (0.49-0.77) ***	0.67 (0.53-0.86) ***	0.67 (0.53-0.86) ***
Q3 (15.30-21.70)	0.65 (0.52-0.80) ***	0.59 (0.48-0.73) ***	0.71 (0.57-0.89) **	0.71 (0.57-0.90) **
Q4 (>21.70)	0.60 (0.47-0.77) ***	0.56 (0.43-0.74) ***	0.72 (0.54-0.97) *	0.72 (0.54-0.95) *
***p* for trend**	<0.001	<0.001	<0.001	<0.001

Note: Crude: Unadjusted;

Model I: Adjusted for gender, age, and race;

Model II: Adjusted for gender, age, race, BMI, smoking, and alcohol consumption;

Model III: Adjusted for gender, age, race, BMI, smoking, alcohol consumption, platelet count, WBC count.

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

Table 5. Subgroup analysis of the relationship between dietary fiber intake and COPD by gender adjusted for different confounding factors.

	OR (95% CI)			
Participants	**Crude**	**Model I**	**Model II**	**Model III**
Gender				
Female	0.97 (0.94-1.00)	0.97 (0.94-1.00)	0.98 (0.95-1.02)	0.98 (0.95-1.02)
Male	0.97 (0.96-0.99) ***	0.97 (0.96-0.99) ***	0.98 (0.97-1.00) *	0.98 (0.97-1.00) *

Note: Crude: Unadjusted;

Model I: Adjusted for age and race;

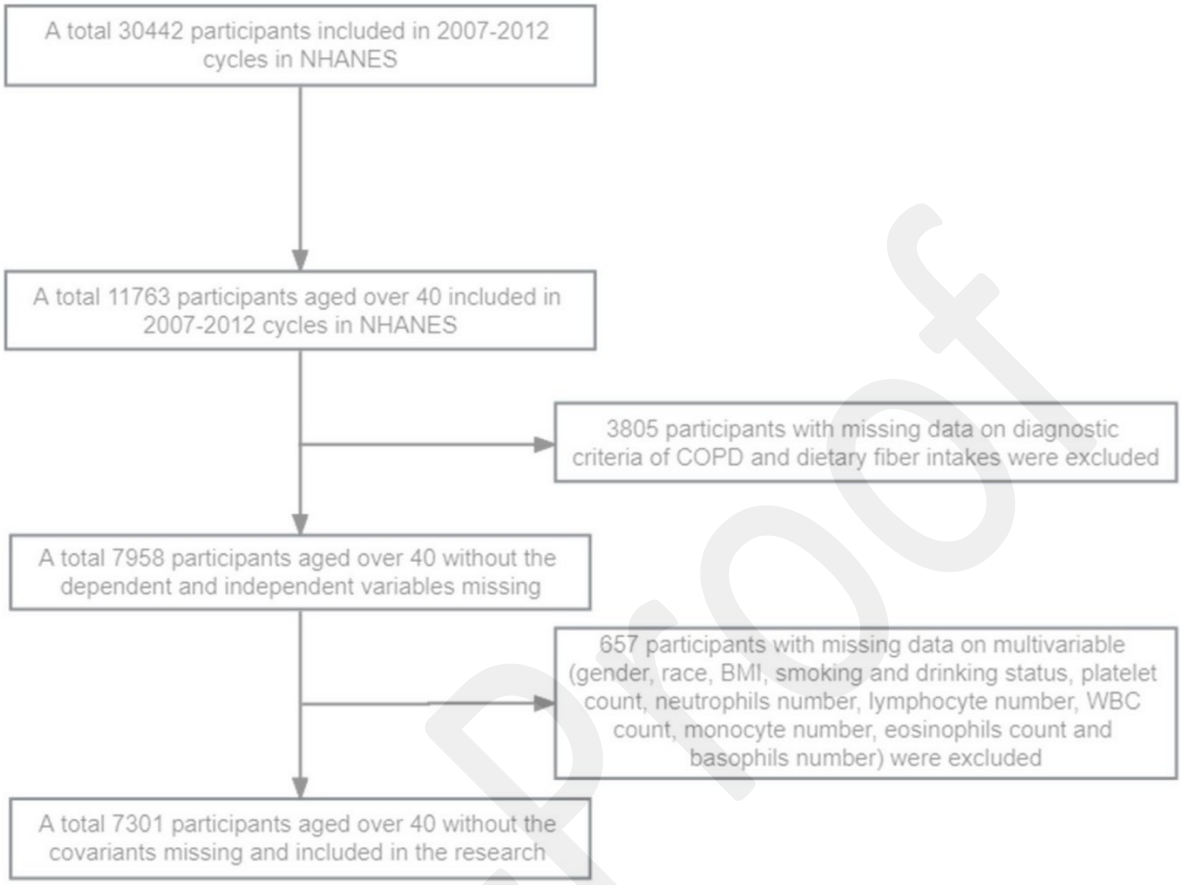
Model II: Adjusted for age, race, BMI, smoking, and alcohol consumption;

Model III: Adjusted for age, race, BMI, smoking, alcohol consumption, platelet count, WBC count.

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

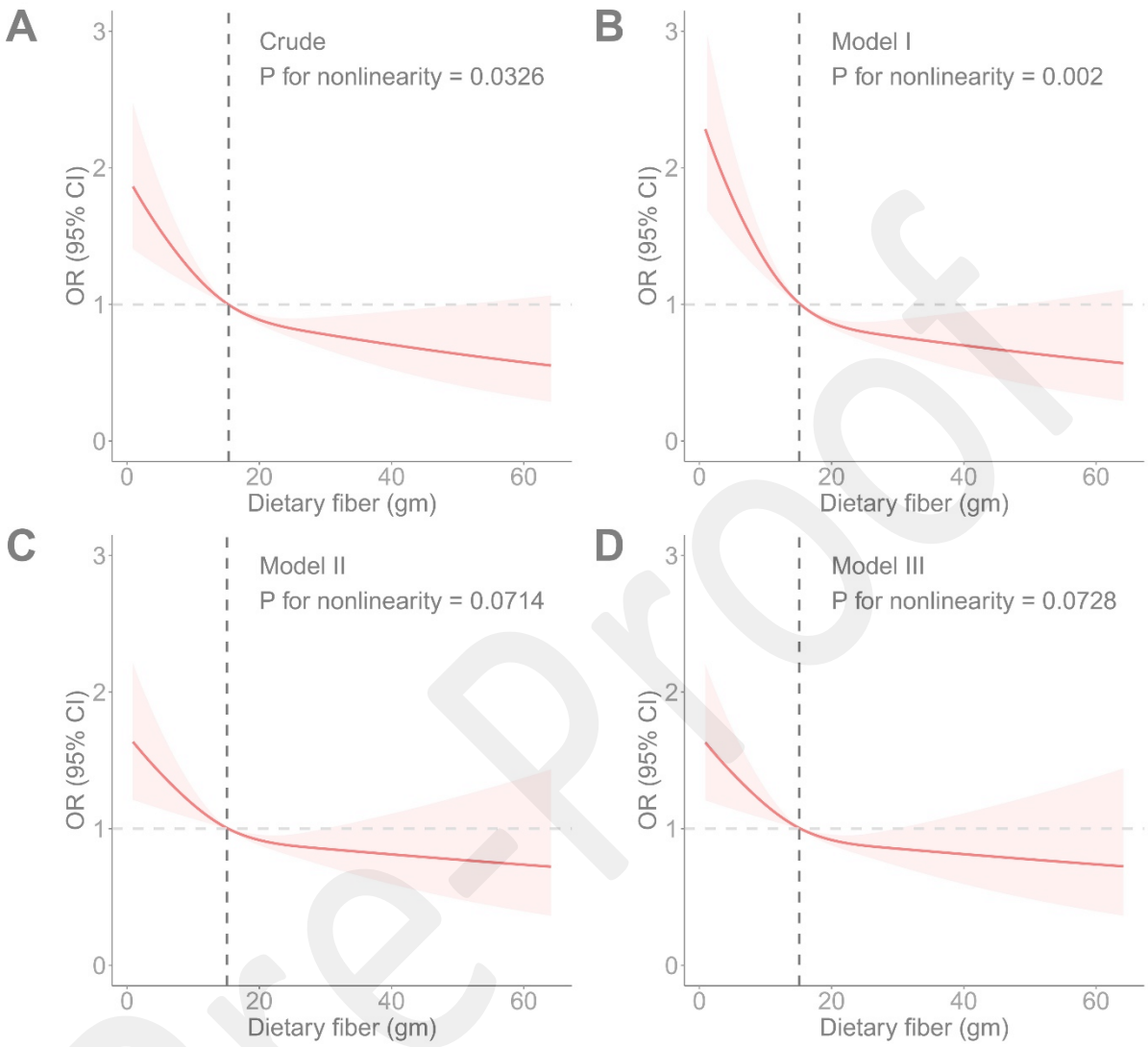
Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

Figure 1: Flowchart of sample selection in NHANES 2007-2012



Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

Figure 2: RCS plots depicting association between DF intake and prevalence of COPD



Crude: Unadjusted;

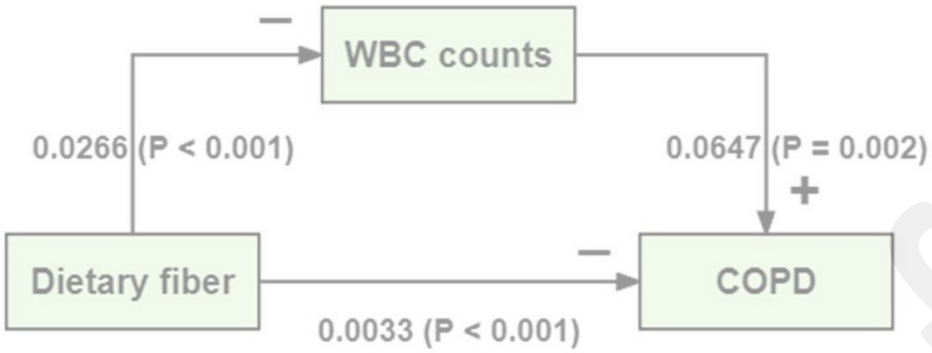
Model I: Adjusted for gender, age and race;

Model II: Adjusted for gender, age, race, BMI, smoking, and alcohol consumption;

Model III: Adjusted for gender, age, race, BMI, smoking, alcohol consumption, platelet count, WBC count.

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

Figure 3: Mediation analysis of WBC count in association between DF and COPD



Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457

Online Supplement

Supplementary table. Logistic regression model for dietary fiber intake and lung function

Outcomes	OR (95% CI)	P-value
FEV1*	1.02 (1.01, 1.04)	<0.001
FEV1/FVC	1.00 (1.00, 1.00)	0.50

Published online February 14, 2024 https://doi.org/10.15326/jcopdf.2023.0457