

Editorial

Diet and COPD: A Gut Feeling About Pathogenesis

Laura R.C. Dowling, BBioMedS (Hons)^{1,2} Hayley A. Scott, PhD^{1,2}

1. School of Biomedical Sciences and Pharmacy, University of Newcastle, Callaghan, Australia
2. Immune Health Research Program, Hunter Medical Research Institute, New Lambton Heights, Australia

Abbreviations:

%pred=percentage predicted; **APDQS**=A Priori Diet Quality Score; **CARDIA**=Coronary Artery Risk Development in Young Adults study; **COPD**=chronic obstructive pulmonary disease; **FEV₁**=forced expiratory volume in 1 second; **NHANES**=National Health and Nutrition Examination Survey

Funding Support:

not applicable

Citation:

Dowling LRC, Scott HA. Diet and COPD: a gut feeling about pathogenesis. *Chronic Obstr Pulm Dis.* 2024;11(2):133-135. doi: <https://doi.org/10.15326/jcopdf.2024.0508>

Publication Dates:

Date of Acceptance: not applicable

Published Online Date: March 26, 2024

Address correspondence to:

Hayley A. Scott, PhD
Immune Health Research Program
Hunter Medical Research Institute
Lot 1 Kookaburra Circuit
New Lambton Heights NSW Australia 2305
Email: Hayley.Scott@newcastle.edu.au

Keywords:

COPD; plant-based diet; gut microbiota; A Priori Diet Quality Score

Editorial

Chronic obstructive pulmonary disease (COPD) is one of the leading causes of morbidity and mortality worldwide. Consequently, there is an urgent need to determine modifiable risk factors that contribute to both its development and progression. Dietary intake is being increasingly recognized as a potential modifiable risk factor for COPD, as research indicates it has a key role in the development of a wide range of other chronic health conditions including cardiovascular disease, type 2 diabetes, and metabolic syndrome.¹⁻³

However, research investigating the impact of diet on lung disease is extremely limited. Fruits and vegetables are one dietary component that may reduce the risk of lung disease, as they are high in antioxidants such as anthocyanins, beta-carotene, and lycopene, which have been identified to play a role in reducing oxidative stress and protecting against oxidant-mediated inflammation.⁴ Fruits and vegetables are also a major source of dietary fiber, which is known to modulate the gut microbiome.⁵ Both the gut microbiome and bacterial metabolites produced in the gut are recognized for their involvement in immune signaling and have been shown to have a role in the progression of other airway diseases, including asthma.⁶

The study by [Jackson and colleagues](#) in this issue of the *Journal* was a prospective cohort study exploring the impact of a plant-centered diet on the lung health of young ever-smokers. The research utilized a subset (n=1351) of data from the Coronary Artery Risk Development in Young Adults (CARDIA) study, a longitudinal cohort study designed to evaluate the risk of developing cardiac disease. A strength of this study is that the authors used the A Priori Diet Quality Score (APDQS), which distinguishes between nutritionally rich and nutritionally poor plant-based foods, as it is well recognized that a plant-based diet is not necessarily nutritious. The findings indicate that consuming a nutritionally rich, plant-centered diet, high in fruits and vegetables, is associated with a lower risk of radiographic emphysema in young adults with a history of having ever smoked. The risk of developing emphysema was more than 5 times lower in the highest APDQS quintile compared with the lowest quintile (i.e., 4.5% versus 25.4%). For each one standard deviation increase in the APDQS, there was a 42% reduction in the odds of developing emphysema. Interestingly, there was no significant interaction between APDQS and pack-year smoking history ($p=0.20$), suggesting that the effects of diet quality and smoking history on the development of emphysema appear to be independent of each other. An additional strength is that this study utilized computed tomography records to objectively assess the role of diet on emphysema risk over a prolonged follow-up period of 25 years. This study supports the findings of a meta-analysis that focused specifically on fruits and vegetables, which found the relative risk of developing COPD was

reduced by 25% in the group with the highest versus lowest consumption of fruits and vegetables.⁷

Dietary fiber is a major constituent of fruits and vegetables, and is recognized for its ability to reinforce the intestinal epithelial barrier function, enhance the proliferation of gut microbiota and their metabolites (such as short chain fatty acids, bile acids and sphingolipids), and reduce the expression of pro-inflammatory cytokines.^{8,9} Therefore, dietary fiber may be responsible for at least some of the beneficial effects of a nutritionally rich, plant-centered diet. The study by [Jin and colleagues](#) in this issue of the *Journal* utilized cross-sectional data from the National Health And Nutrition Examination Survey (NHANES) to investigate the relationship between dietary fiber intake and COPD prevalence in a cohort of adults (n=7301) aged >40 years. The analysis revealed a higher dietary fiber intake was associated with a lower odds of current COPD, particularly in middle-aged men. Those in the lowest quartile of fiber intake had an approximately 35% higher probability of having COPD compared with those who had a higher fiber intake. Notably, the U.S. and other national dietary guidelines recommend a dietary fiber intake of 25-30g/day.¹⁰ The study by Jin and colleagues highlights that people with and without COPD exhibit a significantly lower fiber intake than national guidelines; people with COPD consumed only 16±8g/day, which was significantly lower than people without COPD (18±9g/day), although this group also fell well below recommendations. The authors found a nonlinear association between dietary fiber intake and COPD prevalence, with a critical threshold of approximately 15g/day. Beyond this threshold, dietary fiber effectively lowered the prevalence of COPD. This is interesting, as this cutpoint falls below the recommended dietary intake, suggesting that even a small increase in fiber intake at the

population level may help reduce the prevalence of COPD. Given so few people in this cohort consumed what would be considered a high fiber diet, it is difficult to determine whether this would further reduce COPD prevalence. This is where randomized controlled trials may be useful, as participants can be counseled to consume a diet that is high in fruits, vegetables, and fiber. Very few clinical trials have been conducted in this space. However, one randomized controlled trial found that an increased consumption of fruits and vegetables was associated with an increase in forced expiratory volume in 1 second (FEV₁) percentage predicted (%pred) in people with COPD over the 3-year study period, while those who followed their usual diet exhibited a decline in FEV₁ %pred.¹¹ A strength of the study conducted by Jin and colleagues is that it included a far larger cohort than could be easily recruited into a clinical trial, providing important epidemiological data suggesting that dietary fiber is associated with a lower prevalence of COPD.

In summary, the data presented in both papers published in this issue provide evidence that dietary intake is a modifiable risk factor for the development of COPD. These studies strengthen the current evidence base and can facilitate informed policy and practice in the prevention and treatment of COPD. Together, they support the promotion of a nutritionally rich, plant-centered diet that is high in fiber for the prevention of COPD. Further investigation of fruits, vegetables, and fiber for both the prevention and treatment of COPD is warranted, including examination of potential mechanisms by which these dietary components elicit their protective effects.

Declaration of Interest

The authors have no conflicts of interest to declare.

References

1. Threapleton DE, Greenwood DC, Evans CE, et al. Dietary fibre intake and risk of cardiovascular disease: systematic review and meta-analysis. *BMJ*. 2013;347:f6879. <https://doi.org/10.1136/bmj.f6879>

2. Chandalia M, Garg A, Lutjohann D, von Bergmann K, Grundy SM, Brinkley LJ. Beneficial effects of high dietary fiber intake in patients with type 2 diabetes mellitus. *N Engl J Med*. 2000;342(19):1392-1398. <https://doi.org/10.1056/NEJM200005113421903>

3. Chen JP, Chen GC, Wang XP, Qin L, Bai Y. Dietary fiber and metabolic syndrome: a meta-analysis and review of related mechanisms. *Nutrients*. 2017;10(1):24. <https://doi.org/10.3390/nu10010024>

4. Tan BL, Norhaizan ME, Liew WP, Sulaiman Rahman H. Antioxidant and oxidative stress: a mutual interplay in age-related diseases. *Front Pharmacol*. 2018;9:1162. <https://doi.org/10.3389/fphar.2018.01162>

5. Menni C, Jackson MA, Pallister T, Steves CJ, Spector TD, Valdes AM. Gut microbiome diversity and high-fibre intake are related to lower long-term weight gain. *Int J Obes (Lond)*. 2017;41(7):1099-1105. <https://doi.org/10.1038/ijo.2017.66>

6. Thorburn AN, McKenzie CI, Shen S, et al. Evidence that asthma is a developmental origin disease influenced by maternal diet and bacterial metabolites. *Nat Commun*. 2015;6(1):7320. <https://doi.org/10.1038/ncomms8320>

7. Zhai H, Wang Y, Jiang W. Fruit and vegetable intake and the risk of chronic obstructive pulmonary disease: a dose-response meta-analysis of observational studies. *Biomed Res Int*. 2020;2020:3783481. <https://doi.org/10.1155/2020/3783481>

8. Jang YO, Kim OH, Kim SJ, et al. High-fiber diets attenuate emphysema development via modulation of gut microbiota and metabolism. *Sci Rep*. 2021;11(1):7008. <https://doi.org/10.1038/s41598-021-86404-x>

9. Hou K, Wu Z-X, Chen X-Y, et al. Microbiota in health and diseases. *Signal Transduct Target Ther*. 2022;7(1):135. <https://doi.org/10.1038/s41392-022-00974-4>

10. Anderson JW, Baird P, Davis RH Jr, et al. Health benefits of dietary fiber. *Nutr Rev*. 2009;67(4):188-205. <https://doi.org/10.1111/j.1753-4887.2009.00189.x>

11. Keranis E, Makris D, Rodopoulou P, et al. Impact of dietary shift to higher-antioxidant foods in COPD: a randomised trial. *Eur Respir J*. 2010;36(4):774-780. <https://doi.org/10.1183/09031936.00113809>