5-2021

Impact of Dietary Fiber and Omega6/3 Intake on COPD Outcomes in U.S. Adults

Jacey Hain
University of Nebraska Medical Center

Impact of Dietary Fiber and Omega6/3 Intake on COPD Outcomes in U.S. Adults

Jacey Hain, Epidemiology

UNMC College of Public Health

Committee chair: Dr. Tricia LeVan, PhD

Committee members: Dr. Abraham Mengist, PhD and Dr. Corrine Hanson, PhD, RD, LMNT, FAND

Abstract

COPD is the 4th leading cause of death in the United States and is the leading cause of hospitalizations in adults.[1] Although there is no cure for COPD, symptoms can be managed to increase the quality of life of those affected by the disease. Dietary omega-3 and -6 as well as fiber intakes have been noted as mitigating the burden of disease through their anti-inflammatory properties. We set out to look at the synergistic effect of both omega-3,-6, -6:-3 ratio and fiber on COPD, lung function and respiratory morbidities. We used data from the National Heath and Nutritional Examination Survey (NHANES) to examine the relationship of COPD lung function outcomes (COPD yes/no, COPD GOLD classification, and FEV_1/FVC score) and symptoms with omega-3, -6, omega 6:3 ratio and fiber intakes. We found that dietary fiber intake was associated with COPD status (p=0.0381), cough (p=0.0109), and chronic bronchitis (p=0.0392). We found no significant associations between omega-3 intake and COPD and respiratory outcomes or between omega-6 intake and COPD and respiratory outcomes. We did observe an interaction of fiber quartiles and omega 6 (p=0.0269) and omega 6:3 (p=0.0142) on COPD status. Future studies should examine this interaction further.

Chapter 1- Introduction & Background

COPD

COPD affects 1 in 8 Americans age 45 and older and is the 4th leading cause of death in the US.[1] Currently there are 16 million people diagnosed with COPD, and likely others have the disease, but have not been diagnosed.[2] COPD is also a leading cause of hospitalization in US adults, particularly in older populations,[3] and it is estimated that the cost of COPD patient-related care in 2020 will be $49 billion.[1]

The Global Initiative for Chronic Obstructive Lung Disease describes COPD as, "a disease state characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal inflammatory response of the lungs to noxious particles or gases".[4] One of the main risk factors for COPD is smoking. Also included are air pollution, asthma, occupational exposures, airway hyperresponsiveness, and specific germline genetic variations.[2]

While there is no cure for COPD, disease symptoms can be managed to improve quality of life. An American Lung Association survey stated that half (51%) of all COPD patients say their condition limits their ability to work. The condition also limits COPD patients in normal physical exertion (70%), household chores (56%), social activities (53%), sleeping (50%), and family activities (46%).[5] Pharmacologic treatments do not improve damage to the lung, but can slow lung function decline.[2]

Recent studies have shown that factors that down-regulate inflammatory activity might be particularly useful in diseases characterized by continuous inflammation, like COPD.[6] Many chronic conditions, such as cancer, obesity, autoimmune diseases, rheumatoid arthritis, asthma and depression, are associated with increased production of inflammatory mediator including thromboxane A_2 (TXA_2), leukotriene B_4 (LTB_4), IL-1β, IL-6, tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP). It has been shown that all these inflammatory factors increase with omega-6 fatty acid intake and decrease with omega-3 fatty acid intake.[7]

Dietary Omega -3 and -6

Dietary intake of omega-3 and omega-6 fatty acids, also known as n-3 and n-6 polyunsaturated fatty acids (PUFA), have been shown to be associated with serum inflammatory

markers.[8] A diet rich in omega-6 fatty acids shifts the physiological state to one that is proinflammatory.[9] Many studies have provided evidence that incorporation of alternative fatty acids may modify inflammatory and immune reactions and that omega-3 fatty acids in particular are potent therapeutic agents for inflammatory diseases.[7]

Previous studies have shown an inverse association between the n-3 fatty acid alpha-linolenic acid (ALA) intake and several proinflammatory biomarkers, including CRP, IL-6 and TNF-α.[8] This was seen in both healthy populations and among subjects with cardiovascular disease. In contrast, intake of omega-6 fatty acids, specifically arachidonic acid (ARA), were associated with proinflammatory molecules IL-6 and CRP.[8] Shahar et al. reported a dose-dependent decrease in risk of chronic obstructive pulmonary disease with increasing intake of n−3 fatty acids, docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA).[10] They also stated that even though most of the anti-inflammatory effects of n-3 fatty acids were demonstrated using dietary supplements that provide doses much higher than the average intake, very small increases in intake may have health benefits, showing potential of a cumulative effect.[10] An additional study showed that inclusion of omega−3 fatty acids in the diet can suppress the production of both TNF-α and IL-1β, two proinflammatory cytokines.[11]

Pizzini and colleagues demonstrated the anti-inflammatory proprieties of omega-3 in sixty-four COPD patients receiving either an omega-3 (alpha-linolenic acid (ALA)) or an omega-6 (linoleic acid (LA)) fatty acid supplementation diet for two years.[12] In addition to various clinical improvements, patients consuming the omega-3 rich diet showed significant decrease in proinflammatory molecules, specifically, leukotriene B4 levels in serum and sputum, as well as TNFα and IL-8 levels in sputum.[12]

There is also growing evidence that the omega 3 fatty acid docosapentaenoic acid (DPA) may have both independent and shared health outcomes with EPA and DHA and may be important when considering total dietary omega 3 intake.[13]

On the other hand, there are some studies that highlight the conflicting evidence supporting the association between omega-3, omega-6 and COPD.[24] They noted the variation in methodology for the limited available studies and the need for further investigation, because some studies report no association, some report a negative association, and some report a positive association.

Fiber

Studies have also examined the role of fiber in diseases characterized by chronic inflammation. The beneficial association of dietary fiber with lung function and COPD has been attributed to the anti-inflammatory and/or antioxidant properties of fiber.[14] Fiber intake has been associated with reduced levels of CRP, a marker of systemic inflammation. Other mechanisms in which fiber may modulate inflammation include slowing the absorption of glucose, decreasing lipid oxidation, or influencing the production of anti-inflammatory cytokines by the gut flora.[14] Kan et al. found a statistically significant dose-response relationship between lung function (FEV_1, FVC, FEV_1/FVC ratio) and dietary fiber from all sources, in both basic and adjusted models.[14] Statistically significant associations between total fiber intake and FEV_1 were observed in all subgroups, with little evidence of interaction.[14]

In a prospective study, Varraso et al. examined two large cohorts of US women and men, where they found that participants with a higher total fiber intake had a lower risk of incident COPD, even after adjustment for potential confounders.[15] They also reported a significant and

independent association between total fiber intake and the risk of COPD, particularly in women.[15]

A systematic review and meta-analysis by Sevedrezazadeh et al. reported that longitudinal studies have supported the inverse association between fiber intake and COPD, which was related to anti-inflammatory properties of dietary fiber. Their analysis found that fiber intake was associated with lower levels of CRP and pro-inflammatory cytokines and higher levels of several anti-inflammatory cytokines.[16] They also stated that fiber intake reduced the risk of COPD, chronic bronchitis, and emphysema, and COPD symptoms according to cohort studies.[16]

A study examining 10-year trends in fiber intake reported that mean intakes of fiber from the 2001-2010 NHANES population were well below the current recommendations for all age groups.[17] Their analysis also showed no increase in fiber intake in adults ages 19 to 50 years, and a 0.8g/day increase for adults ages 51+.

Aims

To date, no studies have looked at the synergistic effect of both omega-3,-6, -6:-3 ratio and fiber on COPD, lung function and respiratory morbidities. We hope to shed light on the function of these dietary components in COPD and their possible therapeutic effect to reduce the burden and/or symptoms of COPD. In addition, this study seeks to determine whether the relationship between the omega-6/omega-3 ratio, COPD, and COPD respiratory symptoms is modified by fiber intake. We anticipate that a low dietary omega-6:3 ratio and increased fiber intake are associated with a lower prevalence of COPD compared to those with a high omega-6/omega-3 ratio and fiber intake.

Chapter 2- Methods

Study population

The study population was U.S. adults ≥40 years of age enrolled in the National Health and Nutritional Examination Study (NHANES) from 2007 to 2012. NHANES is a cross-sectional survey that targets the U.S. noninstitutionalized population of all ages and is designed to assess health and nutrition through interviews and physical examinations. Sampling involves a complex, multistage, clustered design and is conducted by the National Center for Health Statistics (NCHS). Participants aged 40 years and older were included in the analysis if they had complete data for all lung function and nutrient variables (Figure 1). Pregnant women, those with improbable values for dietary variables, those missing BMI, smoking status, and poverty data were excluded. Participants with asthma were excluded as well.

COPD and respiratory outcomes

COPD was treated as a dichotomous variable. A participant was considered to have COPD if they had a pre bronchodilator forced expiratory volume in one second (FEV_1)/forced vital capacity (FVC) ratio<0.70, do not have an asthma diagnosis, and have smoked 100 or more cigarettes in their lifetime. To examine the severity of COPD, we used % predicted FEV_1. To grade COPD airflow limitation severity, we used the Global Initiative for Chronic Obstructive Lung Disease (GOLD) classification based on FEV_1 scores. GOLD 1 represents mild COPD (FEV_1≥ 80% predicted), GOLD 2 represents moderate (50% ≤FEV_1<80% predicted), GOLD 3 is severe (30% ≤FEV_1< 50% predicted), and GOLD 4 is very severe (FEV_1<30% predicted).

To examine respiratory symptoms, examined wheeze, cough, phlegm and chronic bronchitis. Participants were asked if they had experienced wheezing or whistling in their chest in the past 12 months. For cough, participants were asked if they usually cough on most days for three consecutive months or more during the year. Finally, participants were asked if they bring up phlegm on most days for three consecutive months or more during the year. A participant was considered to have chronic bronchitis if they have both chronic cough and chronic phlegm. For these variables, responses were recorded as a yes or no. Respiratory symptom outcomes were used in addition to lung function outcomes to examine both the quality and quantity of lung function.

Dietary intake of omega-3/omega-6 and fiber

Using the NHANES 24-hour dietary interview (What We Eat in America), the types and amounts of foods and beverages consumed during the 24 hours prior to the interview were estimated, as well as energy, nutrient and other components of those foods. The questionnaire was used twice during NHANES data collection, the first in-person and the second 3 to 10 days later over the telephone. From that, total nutrient intakes were calculated. We used the average of the data for day one and two. For this study, to determine the total omega-3 intake we used values for docosahexaenoic acid (22:6, Docosahexaenoic acid (DHA)), eicosapentaenoic acid (20:5, Eicosapentaenoic acid (EPA)), octadecatetraenoic acid (18:4, Stearidonic acid, SDA), docosapentanoic acid (22:5, Docosapentaenoic acid (DPA)), and octadecatrienoic acid (18:3, Alpha-linolenic acid (ALA)). To determine the total omega-6 intake we used values for octadecadienoic acid (18:2, Linolenic acid (LA)) and eicosatetraenoic acid (20:4, Arachidonic acid (AA)). We chose to use these omega-3 and -6 isomers based on their categorization in

previous published research.[18,19] Fiber intakes were also be assed using nutrient intake data. We computed the ratio for omega 6:3. Fiber and omegas were examined using quartiles (Q1 low, Q2, Q3, Q4 high).

Additional Covariates

Age was measured in years. Sex was grouped by male and female. Height and weight were used to calculate BMI. BMI below 18.5 was considered underweight, 18.5-24.9 is normal or healthy weight, 25.0-29.9 is overweight, and 30.0 and above is considered obese. Race was split into four categories, non-Hispanic white, non-Hispanic black, Mexican American and other Hispanic and other race including multi-racial. Smoking status was assessed by asking participants about their smoking habits. Participants were asked how many cigarettes they smoked in their entire life and if they now smoke cigarettes. This was categorized as current smoker, former smoker, or never smoker. The poverty ratio is a ratio of family income to poverty levels commonly used to establish eligibility for U.S. food assistance programs and was used to adjust for socioeconomic status. Education level was assessed using respondent's highest level of education.

Statistical Analysis

All statistical analyses were done using SAS 9.4. Descriptive statistics and univariate analysis were done to show distribution and summarize the data for our variables including age, sex, BMI, race, poverty ratio, education, smoking status, wheeze, phlegm, cough, bronchitis, FEV_1/FVC, % predicted FEV_1, % predicted FVC, fiber, omega-3, omega-6, and omega-6:3.

They were also stratified by COPD status. Analyses included mean, median, and standard error of the mean. The complex survey design of NHANES was accounted for during our analysis.

Multivariable logistic regression was used to look at the association of lung function variables (COPD yes/no, COPD GOLD classification) as well as respiratory symptoms (wheeze, cough, chronic bronchitis and phlegm) and omega-3, -6, omega 6:3 ratio and fiber intake levels using PROC SURVEYLOGISTIC. Linear regression was used to examine the relationship between the lung function continuous variables FEV_1/FVC ratio, % predicted FEV_1, and % predicted FVC and all dietary variables.

Multivariable regression models were adjusted for the covariates BMI (underweight <18.5, normal 18.5-24.9, and overweight/obese 25+), poverty ratio category(≤1.9, 2-4.9, 5+), sex (male, female), age category (40-49 years, 50-59 years, 60-69 years, and 70-19 years), race category (Non-Hispanic white, non-Hispanic black, Mexican American and other Hispanic, and other race, including multiracial), mean energy intake (calories/day), and smoking status (current smoker, former smoker, never smoker). These covariates were chosen based on their biological relevancy and based on previous research.

We also explored any possible interactions between fiber and omega quartiles and each covariate in the models for each COPD and respiratory outcome.

Chapter 3- Results

After implementing eligibility criteria and accounting for missing data, 6938 participants were included in the analysis (Figure 1). The study population had a mean age of 55.7 years and consisted of 47.9% males and 52.1% females (Table 1). The majority of the study population was Non-Hispanic White (75.4%), with a mean poverty income ratio of 3.3±0.05. The average

BMI was 29.2kg/m^2. About half (51.9%) of the population reported being a high school grad/GED and having some college or an AA degree. Most of the population were nonsmokers (82%). Those with COPD tended to be male (60.6%), Non-Hispanic white (82.2%), and overweight/obese (66%). They were also older, less educated, in a lower SES group as indicted by the poverty income ratio, and more likely to be current or former smokers. Those with COPD also tended to have higher prevalence of respiratory symptoms wheeze (20.4%), chronic phlegm (17.9%), chronic cough (20.5%), and chronic bronchitis (13.7%) (Table 2). As expected, they also had a lower lung function measured by FEV_1/FVC and % predicted FEV_1.

The mean dietary fiber intake was 17.8 g/day. High dietary fiber intake was seen in those without COPD (Table 3). Mean omega-3 intake was 1.82 g/day and mean omega-6 intake was 16.42 g/day. The average omega 6:3 ratio was 9.69. Those with COPD had a slightly higher mean Omega-6 (17.25g/day (±0.54)) intake compared to those without COPD (16.30g/day (±0.18)) ($p<0.0001$).

Multivariable analysis revealed associations between fiber intake and COPD status ($p=0.038$), cough ($p=0.011$), and chronic bronchitis ($p=0.039$) (Table 4). We found increased odds of COPD with low fiber intake (OR=1.3; 95% CI 0.9-1.7; $p=0.010$), but no liner trend was observed ($p=0.08$). Lower odds of cough was seen with low fiber intake (OR=0.6; 95% CI 0.4-0.8; $p=0.001$) and a liner trend was observed ($p=0.003$). The same association was seen between fiber and bronchitis, lower odds of bronchitis with low fiber intakes (OR=0.6; 95% CI 0.4-1.0; $p=0.005$) and a liner trend was observed ($p=0.044$).

No significant associations were observed between omega-3 intake and COPD and respiratory outcomes or between omega-6 intake and COPD and respiratory outcomes. Overall, a higher omega 6:3 ratio was associated with cough ($p=0.015$). An association was also

observed between fiber intake and % predicted FEV_1 (p=0.0001) as well as fiber intake and % predicted FVC (p<0.0001) (Table 5).

Tests for interaction revealed significant interactions of fiber quartiles with omega 6 (high/low) (p=0.027) and omega 6:3 (high/low) (p=0.014) in predicting COPD status. There was also significant interaction between for fiber quartiles and omega 3 (high/low) in predicting bronchitis (p=0.037) and wheeze (p=0.013). We found that the effect of fiber on COPD outcome depends on the amount of omega-6 and omega-6:3. When omega-6 was low, the odds of having COPD were increased across all levels of fiber (Table 6). Meanwhile, the odds of COPD were lower when omega-6 intake was high across all levels of fiber. The same trend was seen with omega-6:3, when omega-6:3 was low, the odds of having COPD were increased and in the group with high omega-6:3, the odds of COPD were lower.

Chapter 4- Discussion

The goal of this study was to examine the relationship of fiber, omega-3 and omega-6 intake with COPD and respiratory outcomes in the U.S. adult (>40 years) NHANES population. The results support our hypothesis that lower fiber intake is associated with higher odds of COPD. We found the inverse to be true for chronic cough and chronic bronchitis, lower odds of these two respiratory symptoms with low fiber intake. Concurrent with these observations, lung function as measured by % predicted FEV1 and FVC was positively correlated with fiber intake. In contrast, intake of omega -3 or -6 and the omega-6:omega-3 ratio was not associated with COPD or any of the respiratory symptoms tested. We also found increased odds of COPD across all levels of fiber when omega-6 intake was low and when omega-6:3 ratio is low, which is the first observation of this interaction.

Data from our study support the protective role of a high-fiber diet against COPD. Consumption of a high-fiber diet was associated with lower odds of COPD. A linear pattern for wheeze, cough, phlegm, and chronic bronchitis was found, suggesting dietary fiber consumption also plays a protective role in respiratory outcomes. Fiber was also found to be positively associated with lung function as measured by % predicted FEV_1 and FVC. These findings are consistent with previous reports.

One study examining men and women with COPD in the US and adjusting for the same covariates found a negative association between total dietary fiber intake and risk of newly diagnosed COPD.[15] A study that followed Sweedish men for an average of 13 years found a negative association between total fiber intake and incident COPD in current and ex-smokers.[26] A 2019 systematic review and meta-analysis also reported a negative association between fiber consumption and risk of COPD.[16] Additionally, a study using the NHANES 2009-2010 population found that high fiber intake was associated with normal lung function as measured by % predicted FEV_1 and FVC, which is consistent with the results from our study.[20] They also found no association between dietary fiber intake and COPD severity based on the GOLD scale was found, which is also consistent with our results.[20]

In the current study, the protective effects of omega-3 on COPD and respiratory symptoms were not observed. Another study using the NHANES 2007-2012 population also found lack of association between omega-3 intake (DHA+EPA+ALA) and respiratory symptoms.[21] A review by Pinizzi and colleagues concluded the evidence showed weak benefits of omega-3 consumption on reducing the risk of COPD.[12] On the other hand, many previous studies reported a protective role of dietary omega-3 intake on COPD and respiratory morbidities. One study by Lemoine et al. reported a positive association between omega-3 intake

and COPD.[23] They used a different summation of total omega intake different from our study (EPA+DHA+ALA) that was reported from a food frequency questionnaire in a population of adults in Maryland.[23] Another study examining 250 clinically stable COPD patients using two years of dietary data to measure DHA, EPA, and ALA intake reported lower levels of serum inflammatory markers in individuals with high ALA intake.[8] Also, a study enrolling current or former smokers found that increased DHA and EPA consumption was associated with lower risk of COPD.[10] The anti-inflammatory mechanism of the omega-3s EPA, DHA , and ALA are well researched, but there is not as much information regarding the other omega-3s considered in our study (SDA and DPA), presenting a potential limitation.

Overall, our population with COPD had a higher mean omega-6 intake, but no significant associations were found with any of the outcomes. Previous studies note a potential adverse association between omega-6 intake and COPD and respiratory outcomes using the same total omega-6 intake measure we used (LA+AA). Batlle et al. found lower serum inflammatory markers in COPD patients with higher omega-3 intake and higher inflammatory markers in those with higher omega-6 intake, supporting the proposed mechanisms.[8] A systematic review by Scoditti et al. assessed diet quality and COPD and lung function outcomes.[25.] They concluded that an overall high-quality diet was more important than individual nutrients when considering lung function and COPD outcomes. A study using a total omega-6 intake of LA and AA in 250 clinical COPD patients noted that higher AA intake was associated with higher levels of serum inflammatory markers.[8] The study by Lemoine et al. found omega-6 intake (LA+AA) to be associated with lower lung function, measured by % predicted FEV_1.[23] The same study also described an adverse association between the omega-6/omega-3 ratio and respiratory symptoms

in those with COPD. In our study, total omega-6/omega-3 was associated with cough, but otherwise, our results do not indicate an association with the ratio and the other outcomes.

Few studies have examined the omega-6/omega-3 ratio. One review noted the importance of a lower omega-6/omega-3 ratio to reduce the risk of many chronic inflammatory diseases, although COPD was not considered.[28] Many of the studies examining these omega-3 and omega-6 relationships used smaller populations and different dietary assessments as well as different outcome measurements. Although we used the same measure of omega-6, we included two additional components (SDA and DPA) in our omega-3 summation. These differing aspects of the studies could be responsible for the difference in findings and determining the optimal calculation would be beneficial for future research.

Systematic reviews conducted in 2015 and 2019 both noted the conflicting evidence and methodological variation in previous studies examining the omega-3, omega-6 and COPD relationship.[24,25] Some studies report benefits of increased omega-3 intake on COPD and no effect with omega-6, while others report a negative association between omega-6 consumption and no effect of omega-3 on COPD.[25] The differences in findings illustrate the complexity of using diet as an exposure. We must consider how total dietary intakes are determined when interpreting the results as well as how the populations are selected and what other dietary variables are considered and accounted for.

Although some studies have examined the interactive effects of omega-3 and omega-6, interactions between omega-3, omega-6, and fiber intake have not yet been explored until now. In this study, we found increased odds of COPD across all levels of fiber when omega-6 intake was low and when omega-6:3 ratio is low, and lower odds with high omega-6 and omega-6:3 intake. We hypothesized that lower fiber and higher omega-6 intake would be associated with

higher odds of COPD, but these results suggest otherwise. The protective effect of fiber is seen with high omega-6 consumption, but the odds of COPD are increased with low omega-6 consumption across all levels of fiber. The same trends are found with omega-6:3. To our knowledge, this is the first study to show these interactions with COPD.

Our calculation for total omega-3 intake included ALA, which generally has a very poor conversion rate to other compounds that function to resolve inflammation, namely EPA and DHA. Because of this poor conversion, much of the ALA consumed is not biologically available for other functions besides being an energy source and may not provide the same health benefits DHA and EPA have been shown to provide.[29] Since ALA comprises most of the total omega-3 Americans consume, our analysis could be affected by the inclusion of ALA in the total omega-3, and should be considered when discussing the results.

When this interaction is considered, it opposes our hypothesis about the individual impact of omega-6 intake and highlights the complexity these dietary variables have on their own and as they work together in the body. In the body, omega-3 and omega-6 compete for enzymes during metabolism. This can impact gene expression and the type and level of inflammation that occurs, ultimately impacting outcomes in our study.[25] Since our research indicates that the effectiveness of fiber depends on the level of omega-6, omega-6 could possess some anti-inflammatory properties at high levels in the body. It could also have an additive effect when paired with increasing fiber levels. Research examining individual omega-3 and omega-6 intake have not accounted for other dietary variables, so additional data examining this omega-fiber interaction is very limited.

One considerable limitation of the study was the use of diet variables. To quantify intake, we used the NHANES 24-hour dietary interview (What We Eat in America). It consists

of two separate interviews and relies on accurate recall from the participant about what and when they ate. Although this method is valid, it is worth noting that self-reporting can be imprecise and recall error can occur. Also, these interviews give a good idea of short-term consumption, but they can lack accuracy about actual daily consumption habits.

In addition, the complexity of nutrient metabolism makes it difficult to determine exactly how the body is processing these nutrients without considering the differing metabolism of each participant. These nutrients have many different functions, and without measuring any biomarkers, it's difficult to determine how they are impacting the body. To overcome this, including a serum biomarker in this analysis would allow measurement of inflammatory changes and draw more definite conclusions about the impact of our dietary variables. Also, people who consume appropriate levels of nutrients highlighted in this study likely perform other healthy habits in their daily life. We tried to control for this by adjusting our models for important lifestyle factors such as age, race, BMI, smoking status, education level, and poverty level.

The use of the NHANES population is a strength of our study. This is a large, nationally representative sample, and our results are generalizable to the adult U.S. population. Also, using diet variables presents a look at a modifiable risk factor that people can make practical changes to in their life to improve health. By adding to the growing body of literature supporting the association between dietary fiber and omega-3 intake and COPD outcomes and expanding education programs about dietary influences, we can provide an alternative method of improving health.

Future research regarding this topic would benefit from a deeper dive into the dietary aspect of the study, including strict consideration of how total omega-3 and omega-6 intake is quantified. By looking into the detail of the mechanisms of action of omega-3, omega-6, and

fiber in COPD patients, it would allow us to understand in more detail how these nutrients are affecting the outcomes. Including a serum inflammation biomarker in the analysis would be a good addition to advance this investigation and further explain initial findings.

As a whole, this study provides evidence for the support of the anti-inflammatory effects of high fiber intake to lower odds of COPD and a number of respiratory morbidities in the U.S. adult population. Our study also provides promising evidence about the functions of omega-3 and omega-6 with COPD and lung function, as well as a new look at the interaction between fiber and omega-3 and omega-6 with COPD. To determine the effects of omega-3 and omega-6 intake on these outcomes and gain better insight on the interactions, further investigation is necessary.

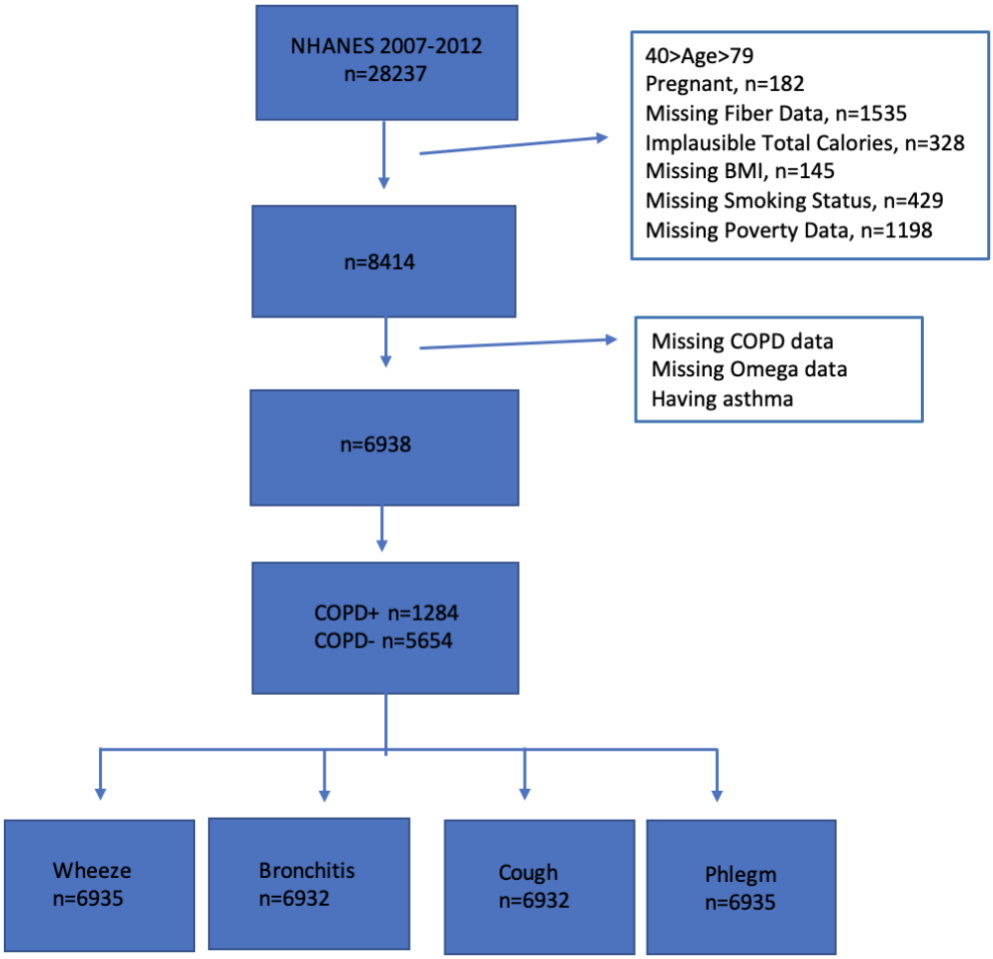

FIGURE 1. Strengthening the Reporting of Observational Studies in Epidemiology **(**STROBE) flow diagram.

Variable		Overall	COPD+	COPD-	p-value
Age, years		55.70 (±0.19)	60.16(±0.42)	54.71(±0.22)	<0.0001
Age, years	40-49	1975 (33.2)	200 (19.2)	1775 (36.2)	<0.0001
	50-59	1832 (32.1)	272 (28.2)	1560 (33.0)	
	60-69	1878 (22.0)	427 (31.1)	1451 (20.0)	
	70-79	1253 (12.7)	385 (21.6)	868 (10.8)	
Sex	Male	3412 (47.9)	834 (60.6)	2578 (45.1)	<0.0001
	Female	3526 (52.1)	450 (39.4)	3076 (54.9)	
BMI, kg/m^2		29.17 (±0.13)	27.86 (±0.18)	29.46 (±0.15)	<0.0001
BMI, kg/m^2	Underweight: <18.5	86 (1.2)	38(2.6)	48 (0.91)	<0.0001
	Normal: 18.5-24.9	1581 (24.9)	376 (31.4)	1205 (23.4)	
	Overweight: 25.0-29.9	2466 (36.2)	445 (35.9)	2021 (36.2)	
	Obesity I:30.0-34.9	1633 (22.1)	270 (19.5)	1363 (22.7)	
	Obesity II: 35.0-39.9	699 (9.4)	98 (6.7)	601 (10.0)	
	Extremely Obese: 40+	473 (6.2)	57 (3.9)	416 (6.7)	
Race	Non-Hispanic White	3270 (75.4)	780 (82.2)	2490 (73.9)	<0.0001
	Non-Hispanic Black	1538 (9.9)	258 (7.9)	1280 (10.4)	
	Mexican American/ Other Hispanic	1686 (9.6)	197 (5.2)	1489 (10.6)	
	Other race, including multiracial	444 (5.0)	49 (4.7)	395 (5.1)	
Poverty Ratio		3.30 (±0.05)	3.00 (±0.09)	3.37 (±0.06)	<0.0001
Education	< High School	1874 (16.8)	413 (22.7)	1461 (15.5)	<0.0001
	≥ High School and Some College	3447 (51.9)	690 (59.2)	2757 (50.2)	
	≥ College	1613 (31.3)	181 (18.0)	1432 (34.3)	
Smoking Status	Never	3523 (51.8)	0 (0)	3523 (51.8)	<0.0001
	Former	2091 (30.2)	735 (57.2)	1356 (24.2)	
	Current	1324 (18.0)	549 (42.8)	775 (12.5)	

TABLE 1. Study population demographic characteristics stratified by COPD status.
Results are displayed as mean ± standard error of the mean for continuous variables and as n (%) for categorical variables. PROC SURVEYMEANS and PROC SURVEYFREQ were used for this analysis.
Definitions: COPD= prebronchodilator FEV1/FVC ratio < 0.7, no asthma diagnosis, and smoked 100 or more cigarettes in their lifetime; BMI= body mass index; FEV1= forced expiratory volume in one second; FVC= forced vital capacity; poverty ratio= a ratio of family income to poverty levels commonly used to establish eligibility for U.S. food assistance programs.

Variable		Overall	COPD+	COPD-	p-value
Wheeze in past year	No	6175 (89.6)	1019 (79.6)	5156 (91.8)	<0.0001
	Yes	760 (10.4)	265 (20.4)	495 (8.2)	
Chronic Phlegm	No	6374 (92.5)	1065 (82.1)	5309 (94.8)	<0.0001
	Yes	561 (7.5)	219 (17.9)	342 (5.2)	
Chronic Cough	No	6273 (90.4)	1030 (79.5)	5243 (92.8)	<0.0001
	Yes	659 (9.6)	254 (20.5)	405 (7.2)	
Chronic Bronchitis	No	6573 (95.0)	1115 (86.3)	5458 (96.9)	<0.0001
	Yes	365 (5.0)	169 (13.7)	196 (3.1)	
FEV1/FVC		0.759 (±0.002)	0.631 (±0.003)	0.779 (±0.001)	<0.0001
% Predicted FEV1		95.55 (±0.37)	80.18 (±0.93)	98.02 (±0.29)	<0.0001
% Predicted FVC		98.13 (±0.28)	96.78 (±0.81)	98.35 (±0.27)	<0.0001

TABLE 2. Study population clinical characteristics stratified by COPD status.
Data are reported as mean ± standard error of the mean. PROC SURVEYMEANS was used for this analysis. *Definitions:* Current wheeze= "yes" to the question "In the past 12 months, have you had wheezing or whistling in your chest?"; Chronic Cough= a positive answer to the question "Do you usually cough on most days for three consecutive months or more during the year?"; Chronic Phlegm= "yes" to the question "Do you bring up phlegm on most days for 3 consecutive months or more during the year?"; Chronic Bronchitis= both chronic cough and phlegm.

Variable		Overall	COPD+	COPD-	p-value
Fiber (g/day)		17.83 (±0.22)	16.84 (±0.47)	17.97 (±0.24)	<0.0001
Fiber Quartiles	Q1, <11.6 g/d	1822 (24.5)	261 (32.0)	1561 (23.9)	0.0041
	Q2, 11.6-16.1g/d	1610 (25.4)	169 (22.9)	1441 (25.8)	
	Q3, 16.1-22.5g/d	1520 (24.6)	172 (20.1)	1348 (25.2)	
	Q4, >22.5g/d	1475 (25.1)	175 (24.9)	1300 (25.1)	
Omega 3 (g/day)		1.82 (±0.02)	1.90 (±0.06)	1.81 (±0.02)	<0.0001
Omega 3 Quartiles	Q1, <1.1 g/d	1824 (25.0)	200 (23.9)	1624 (25.1)	0.37
	Q2, 1.1-1.6g/d	1682 (25.0)	205 (23.3)	1477 (25.3)	
	Q3, 1.6-2.3g/d	1518 (25.0)	177 (24.2)	1341 (25.1)	
	Q4, >2.3g/d	1403 (25.0)	195 (28.6)	1403 (24.5)	
Omega 6 (g/day)		16.42 (±0.16)	17.25 (±0.54)	16.30 (±0.18)	<0.0001
Omega 6 Quartiles	Q1, <10.2g/d	1916 (25.0)	217 (26.0)	1699 (24.9)	0.23
	Q2, 10.2-14.7g/d	1644 (25.0)	196 (21.9)	1448 (25.5)	
	Q3, 14.7-20.7g/d	1511 (25.0)	183 (23.9)	1328 (25.2)	
	Q4, >20.7g/d	1356 (25.0)	181 (28.3)	1175 (24.5)	
Omega 6:3		9.69 (±0.07)	9.66 (±0.19)	9.70 (±0.07)	<0.0001
Omega 6:3 Quartiles	Q1, <7.6	1732 (25.0)	210 (26.3)	1522 (24.8)	0.81
	Q2, 7.6-9.1	1609 (25.0)	211 (26.0)	1398 (24.9)	
	Q3, 9.1-11.0	1574 (25.0)	180 (24.3)	1394 (25.1)	
	Q4, >11.0	1512 (25.0)	176 (23.5)	1512 (25.3)	

TABLE 3. Dietary intake of fiber and omega -3 and 6- fatty acids stratified by COPD status**.**
Data are reported as mean ± standard error of the mean. PROC SURVEYMEANS was used for this analysis.

Quartile of Dietary Fiber Intake							
Variable	Q1 <11.6 g/d	Q2 11.6-16.1 g/d	Q3 16.1-22.5 g/d	Q4 >22.5 g/d	overall p-value	Q1 vs Q4 p-value	P-trend
COPD +/-	1.3(0.9-1.7)	1.0(0.7-1.5)	0.8(0.6-1.1)	1	0.0381	0.0100	0.08
COPD GOLD	0.8(0.5-1.1)	0.8(0.5-1.3)	0.8(0.6-1.1)	1	0.33	0.34	0.72
Wheeze	0.8(0.5-1.1)	0.7(0.5-1.1)	0.8(0.6-1.1)	1	0.39	0.39	0.0323
cough	0.6(0.4-0.8)	0.8(0.5-1.1)	0.9(0.7-1.3)	1	0.0109	0.0012	0.0031
phlegm	0.7(0.4-1.0)	0.8(0.5-1.3)	0.9(0.6-1.4)	1	0.08	0.0117	0.06
bronchitis	0.6(0.4-1.0)	0.9(0.6-1.5)	1.1(0.7-1.8)	1	0.0392	0.0054	0.0437

Quartile of Dietary Omega-3 Intake							
Variable	Q1, <1.1 g/d	Q2, 1.1-1.6g/d	Q3, 1.6-2.3g/d	Q4, >2.3g/d	overall p-value	Q1 vs Q4 p-value	P Trend
COPD +/-	1.0(0.7-1.4)	0.8(0.6-1.1)	0.8(0.6-1.2)	1	0.33	0.44	0.57
COPD GOLD	0.8(0.6-1.2)	0.8(0.5-1.1)	0.9(0.6-1.3)	1	0.55	0.61	0.23
wheeze	0.9(0.6-1.3)	0.8(0.6-1.2)	1.0(0.7-1.4)	1	0.69	0.61	0.90
cough	1.1(0.8-1.5)	1.0(0.7-1.3)	1.2(0.9-1.5)	1	0.49	0.61	0.75
phlegm	0.9(0.6-1.4)	1.0(0.7-1.6)	1.0(0.7-1.4)	1	0.90	0.57	0.99
bronchitis	0.9(0.6-1.6)	1.0(0.7-1.6)	1.0(0.6-1.7)	1	0.97	0.69	0.97

Quartile of Dietary Omega-6 Intake							
Variable	Q1, <10.2g/d	Q2, 10.2-14.7g/d	Q3, 14.7-20.7g/d	Q4, >20.7g/d	overall p-value	Q1 vs Q4 p-value	P Trend
COPD +/-	1	0.8(0.6-1.0)	0.9(0.7-1.1)	0.9(0.6-1.3)	0.34	0.90	0.50
COPD GOLD	1	1.0(0.7-1.4)	1.0(0.7-1.4)	1.2(0.7-1.9)	0.76	0.39	0.20
wheeze	1	0.9(0.7-1.2)	1.0(0.7-1.4)	0.9(0.6-1.4)	0.90	0.70	0.88
cough	1	1.0(0.7-1.4)	1.2(0.8-1.7)	0.9(0.6-1.4)	0.50	0.33	0.53
phlegm	1	0.8(0.6-1.2)	1.2(0.8-2.0)	1.0(0.6-1.7)	0.15	0.86	0.88
bronchitis	1	1.0(0.6-1.5)	1.3(0.8-2.1)	1.0(0.6-2.0)	0.52	0.90	0.93

Quartile of Dietary Omega-6:3 Intake							
Variable	Q1, <7.6	Q2, 7.6-9.1	Q3, 9.1-11.0	Q4, >11.0	overall p-value	Q1 vs Q4 p-value	P Trend
COPD +/-	1	1.0(0.7-1.4)	0.9(0.6-1.2)	0.9(0.6-1.2)	0.67	0.41	0.96
COPD GOLD	1	0.9(0.6-1.2)	0.8(0.6-1.2)	0.9(0.6-1.4)	0.78	0.79	0.60
wheeze	1	1.1(0.8-1.5)	1.1(0.8-1.5)	0.9(0.7-1.3)	0.59	0.30	0.15
cough	1	0.7(0.5-1.0)	1.1(0.8-1.6)	0.8(0.6-1.1)	0.0146	0.42	0.39
phlegm	1	0.9(0.6-1.2)	1.0(0.7-1.6)	1.0(0.7-1.5)	0.74	0.61	0.69
bronchitis	1	0.7(0.4-1.2)	1.0(0.6-1.6)	1.0(0.7-1.5)	0.39	0.44	0.61

TABLE 4. Relationship of respiratory outcomes with dietary quartiles.
Data are reported as odds ratios with 95% confidence intervals. PROC SURVEYLOGISTIC was used for this analysis. It was adjusted for age category, BMI (underweight <18.5, normal 18.5-24.9, and overweight/obese 25+), poverty ratio category (≤1.9, 2-4.9, 5+), sex, race, mean energy intake, and smoking status. For fiber and omega 3, Q4 was used as the reference category and valued at 1. For omega 6 and omega 6:3, Q1 was used as the reference category and valued at 1.

FIBER		
Variable	ß-coefficient	p-value
FEV_1/FVC ratio	-0.0013	0.22
FEV_1 (% Predicted)	0.975	0.0001
FVC (% Predicted)	1.108	<0.0001

OMEGA 3		
Variable	ß-coefficient	p-value
FEV_1/FVC ratio	-0.00013	0.91
FEV_1 (% Predicted)	-0.067	0.84
FVC (% Predicted)	-0.089	0.79

OMEGA 6		
Variable	ß-coefficient	p-value
FEV_1/FVC ratio	-0.0000094	0.99
FEV_1 (% Predicted)	-0.1409	0.64
FVC (% Predicted)	-0.1136	0.63

OMEGA 6:3		
Variable	ß-coefficient	p-value
FEV_1/FVC ratio	0.000845	0.48
FEV_1 (% Predicted)	-0.0249	0.93
FVC (% Predicted)	-0.121	0.65

TABLE 5. Relationship of lung function and dietary omega -3 and -6 fatty acids.
Data are reported as regression coefficients with associated p-values. PROC SURVEYLOGISTIC was used for this analysis. It was adjusted for age (40-49 years, 50-59 years, 50-69 years, 70-79 years), BMI (<18.5, 18.5-24.9, ≥25), poverty ratio category (≤1.9, 2-4.9, ≥5), sex, race, mean energy intake, and smoking status.

		Quartile of Dietary Fiber Intake			
Variable	Interaction p-value	Q1, <10.2g/d	Q2 10.2-14.7g/d	Q3 14.7-20.7g/d	Q4 >20.7g/d
Omega-6	0.0269				
High		0.8 (0.4,1.3)	0.7 (0.5,1.2)	0.7 (0.5,1.0)	1
Low		2.2 (1.5,3.3)	1.8 (1.1,2.9)	1.3 (0.8,2.1)	1
Omega 6:3	0.0142				
High		0.8 (0.5,1.3)	0.6 (0.3,0.9)	0.5 (0.4,0.8)	1
Low		1.8 (1.2,2.7)	1.8 (1.1,2.9)	1.2 (0.8,1.7)	1
Omega 3	0.84				
High		1.2 (0.7,2.0)	1.0 (0.7,1.5)	0.8 (0.5,1.1)	1
Low		1.5 (0.9,2.3)	1.2 (0.7,2.0)	1.0 (0.6,1.7)	1

TABLE 6. Interaction of omega -3, -6, -6:3 and fiber for COPD status (yes/no).
Data are reported as odds ratios with 95% confidence intervals. PROC SURVEYLOGISTIC was used for this analysis and adjusted for age (40-49 years, 50-59 years, 50-69 years, 70-79 years), BMI, kg/m^2 (<18.5, 18.5-24.9, >25), poverty ratio category (≤1.9, 2-4.9, ≥ 5), sex, race, mean energy intake, and smoking status. For fiber, Q4 was used as the reference category and valued at 1.

References

1. COPD National Action Plan. (n.d.). Retrieved from https://www.nhlbi.nih.gov/health-topics/education-and-awareness/COPD-national-action-plan

2. COPD. (n.d.). Retrieved from https://www.nhlbi.nih.gov/health-topics/copd

3. Mannino, D. M. (2015, December 16). COPD: Epidemiology, Prevalence, Morbidity and Mortality, and Disease Heterogeneity. Retrieved from https://www.sciencedirect.com/science/article/pii/S0012369215353769

4. Global Initiative for Chronic Obstructive Lung Disease - Global Initiative for Chronic Obstructive Lung Disease. (2020, November 19). Retrieved from https://goldcopd.org/

5. Feldman, G. (2013). Improving the quality of life in patients with chronic obstructive pulmonary disease: Focus on indacaterol. *International Journal of Chronic Obstructive Pulmonary Disease,*89. doi:10.2147/copd.s31209

6. Schwartz, J. (2000). Role of polyunsaturated fatty acids in lung disease. *The American Journal of Clinical Nutrition,71*(1). doi:10.1093/ajcn/71.1.393s

7. Simopoulos, A. (2002). The importance of the ratio of omega-6/omega-3 essential fatty acids. *Biomedicine & Pharmacotherapy,56*(8), 365-379. doi:10.1016/s0753-3322(02)00253-6

8. Batlle, J. D., Sauleda, J., Balcells, E., Gómez, F. P., Méndez, M., Rodriguez, E., . . . Garcia-Aymerich, J. (2012). Association between Ω3 and Ω6 fatty acid intakes and serum inflammatory markers in COPD. *The Journal of Nutritional Biochemistry,23*(7), 817-821. doi:10.1016/j.jnutbio.2011.04.005

9. Simopoulos, A. P. (2016). An Increase in the Omega-6/Omega-3 Fatty Acid Ratio Increases the Risk for Obesity. *Nutrients*. doi:10.3390/nu8030128

10. Shahar, E., Folsom, A. R., Melnick, S. L., Tockman, M. S., Comstock, G. W., Gennaro, V., . . . Szklo, M. (1994). Dietary n-3 Polyunsaturated Fatty Acids and Smoking-Related Chronic Obstructive Pulmonary Disease. *New England Journal of Medicine,331*(4), 228-233. doi:10.1056/nejm199407283310403

11. James, M. J., Gibson, R. A., & Cleland, L. G. (2000). Dietary polyunsaturated fatty acids and inflammatory mediator production. *The American Journal of Clinical Nutrition,71*(1), 343-348. doi:https://doi.org/10.1093/ajcn/71.1.343s

12. Pizzini, A., Lunger, L., Sonnweber, T., Weiss, G., & Tancevski, I. (2018). The Role of Omega-3 Fatty Acids in the Setting of Coronary Artery Disease and COPD: A Review. *Nutrients,10*(12), 1864. doi:10.3390/nu10121864

13. Richter, C. K., Bisselou, K. S., Nordgren, T. M., Smith, L., Appiah, A. K., Hein, N., . . . Skulas-Ray, A. C. (2019). N-3 Docosapentaenoic Acid Intake and Relationship with Plasma Long-Chain n-3 Fatty Acid Concentrations in the United States: NHANES 2003–2014. *Lipids,54*(4), 221-230. doi:10.1002/lipd.12146

14. Kan, H., Stevens, J., Heiss, G., Rose, K. M., & London, S. J. (2007). Dietary Fiber, Lung Function, and Chronic Obstructive Pulmonary Disease in the Atherosclerosis Risk in Communities Study. *American Journal of Epidemiology,167*(5), 570-578. doi:10.1093/aje/kwm343

15. Varraso, R., Willett, W. C., & Camargo, C. A. (2010). Prospective Study of Dietary Fiber and Risk of Chronic Obstructive Pulmonary Disease Among US Women and Men. *American Journal of Epidemiology,171*(7), 776-784. doi:10.1093/aje/kwp455

16. Seyedrezazadeh, E., Pour Moghaddam, M., Ansarin, K., Asghari Jafarabadi, M., Sharifi, A., Sharma, S., & Kolahdooz, F. (2019). Dietary Factors and Risk of Chronic Obstructive Pulmonary Disease: A Systemic Review and Meta-Analysis. *Tanaffos,*294-309.

17. Mcgill, C., Iii, V., & Devareddy, L. (2015). Ten-Year Trends in Fiber and Whole Grain Intakes and Food Sources for the United States Population: National Health and Nutrition Examination Survey 2001–2010. *Nutrients,7*(2), 1119-1130. doi:10.3390/nu7021119

18. Chen, J., Sun, B., & Zhang, D. (2019). Association of Dietary n3 and n6 Fatty Acids Intake with Hypertension: NHANES 2007–2014. *Nutrients,11*(6), 1232. doi:10.3390/nu11061232

19. Dong, X., Li, S., Chen, J., Li, Y., Wu, Y., & Zhang, D. (2020). Association of dietary ω-3 and ω-6 fatty acids intake with cognitive performance in older adults: National Health and nutrition examination Survey (NHANES) 2011–2014. *Nutrition Journal,19*(1). doi:10.1186/s12937-020-00547-7

20. Hanson, C., Lyden, E., Rennard, S., Mannino, D. M., Rutten, E. P., Hopkins, R., & Young, R. (2016). The Relationship between Dietary Fiber Intake and Lung Function in the National Health and Nutrition Examination Surveys. *Annals of the American Thoracic Society,13*(5), 643-650. doi:10.1513/annalsats.201509-609oc

21. S, C. M., Brigham, E. P., Woo, H., Hanson, C. K., Mccormack, M. C., Koch, A., . . . Hansel, N. N. (2019). Omega-3 fatty acid intake and prevalent respiratory symptoms among U.S. adults with COPD. *BMC Pulmonary Medicine,19*(1). doi:10.1186/s12890-019-0852-4

22. Saeed, M. A., Gribben, K. C., Alam, M., Lyden, E. R., Hanson, C. K., & Levan, T. D. (2020). Association of Dietary Fiber on Asthma, Respiratory Symptoms, and Inflammation in the Adult National Health and Nutrition Examination Survey Population. *Annals of the American Thoracic Society,17*(9), 1062-1068. doi:10.1513/annalsats.201910-776oc

23. Lemoine, C., Brigham, E., Woo, H., Koch, A., Hanson, C., Romero, K., . . . Hansel, N. (2020). Relationship between Omega-3 and Omega-6 Fatty Acid Intake and Chronic Obstructive Pulmonary Disease Morbidity. *Annals of the American Thoracic Society,17*(3), 378-381. doi:10.1513/annalsats.201910-740rl

24. Fulton, A. S., Hill, A. M., Williams, M. T., Howe, P. R., & Coates, A. M. (2015). Paucity of evidence for a relationship between long-chain omega-3 fatty acid intake and chronic obstructive pulmonary disease: A systematic review. *Nutrition Reviews,73*(9), 612-623. doi:10.1093/nutrit/nuv017

25. Scoditti, E., Massaro, M., Garbarino, S., & Toraldo, D. M. (2019). Role of Diet in Chronic Obstructive Pulmonary Disease Prevention and Treatment. *Nutrients,11*(6), 1357. doi:10.3390/nu11061357

26. Kaluza, J., Harris, H., Wallin, A., Linden, A., & Wolk, A. (2018). Dietary Fiber Intake and Risk of Chronic Obstructive Pulmonary Disease. *Epidemiology,29*(2), 254-260. doi:10.1097/ede.0000000000000750

27. Wald, E. L., Borst, B. V., Gosker, H. R., & Schols, A. M. (2013). Dietary fibre and fatty acids in chronic obstructive pulmonary disease risk and progression: A systematic review. *Respirology,19*(2), 176-184. doi:10.1111/resp.12229

28. Simopoulos, A. (2003). Importance of the Ratio of Omega-6/Omega-3 Essential Fatty Acids: Evolutionary Aspects. *World Review of Nutrition and Dietetics Omega-6/Omega-3 Essential Fatty Acid Ratio: The Scientific Evidence*,1-22. doi:10.1159/000073788

29. Anderson, B. M., & Ma, D. W. (2009). Are all n-3 polyunsaturated fatty acids created equal? *Lipids in Health and Disease,8*(1), 33. doi:10.1186/1476-511x-8-33

IRB Approval

IRB approval is not required because the data was obtained from the public database NHANES.

Curriculum vitae

Jacey Hain

1811 North 98th St. ∞ Lincoln, NE 68505
Phone: 402.580.1101 ∞ Email: j.hain116@gmail.com

Education

Master of Public Health, Epidemiology **Expected May 2021**
University of Nebraska Medical Center College of Public Health, Omaha, NE

Bachelor of Science, Biochemistry and Molecular Biology **May 2018**
Nebraska Wesleyan University, Lincoln, NE

Professional Experience

Redzone Fitness & Training **September 2020-April 2021**
Group Fitness Instructor
- Create workouts for and lead group fitness classes

Dzewaltowski Lab, UNMC **December 2019-August 2020**

Student Research Assistant
- Collected observational and quantitative data during youth activity sessions
- Processed quantitative data using SAS
- Analyzed video data using specific coding criteria

East-Central District Health Department, Columbus, NE **May 2020-July 2020**
Student Intern (Applied Practice Experience, UNMC)
- Developed an After-Action Report for the COVID-19 response
- Cleaned and presented BRFSS data using SAS and Microsoft Excel
- Reviewed and updated department policies and procedures
- Completed FEMA IS-100 and IS-700 NIMS training

LabCorp **May 2018-February 2019**
Cytology Specialty Lab Assistant
- Performed general laboratory tasks including specimen processing and slide preparation
- Completed patient data and record entry
- Provided client customer service over the phone and through email

Congressional Sportsmen's Foundation, Washington, DC **January 2017-May 2017**
Brad Rowse Policy Intern
- Selected to study in Nebraska Wesleyan University's Capitol Hill Internship Program
- Conducted sportsmen and conservation-related issue research and created summary documents
- Assisted with Foundation events
- Performed general administrative tasks

Nebraska Center for Virology, Brown Lab **June 2016-May 2018**
Undergraduate Researcher (INBRE Scholar)

- IDeA Networks of Biomedical Research Excellence Scholar Program, funding: NIH P20-GM103427
- 40 hours/week during the summer and 10 hours/week during the academic year
- Conducted research focusing on the immune response to influenza using a variety of immunology assays and analyses (qRT-PCR, RNA extraction)
- Collected, analyzed, and interpreted data
- Participated in lab meetings
- Created and presented oral and poster overviews of research background and findings
- Formal research presentations: West Coast Biological Science Undergraduate Research Conference 2018, INBRE Research Conference 2017, 2018, Nebraska Academy of Sciences 2018

Volunteer and Leadership Experience

- Student Response Team, UNMC — August 2019-present
- Fundraising Co-Head for Relay for Life, NWU — Fall 2015-Spring 2016
- New Student Orientation Leader, NWU — Winter 2014-January 2017
- Academic Honors List (GPA > 3.75), NWU — Fall 2014-May 2018
- Outstanding Performance in First Year Chemistry, NWU — Fall 2014-Spring 2015
- Teammate's Mentor, Lincoln, NE — Winter 2015-May 2018

Computer Skills

- SAS
- Microsoft Office

Biography

I am currently a Master of Public Health Student at the University of Nebraska Medical Center College of Public Health in the department of epidemiology. I received my Bachelor of Science degree in biochemistry and molecular biology from Nebraska Wesleyan University. During the summer of 2020, I worked at the East-Central District Health department and assisted with the department's COVID-19 response. I also worked as a student research assistant in Dr. Dzewaltowski's lab in the health promotion department at UNMC. These experiences confirmed my future career interests of working in a health department or being involved with public health research.