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ORIGINAL RESEARCH

Prevalence and Prognostic Significance of Systemic Inflammation Index and Diet Quality in Patients with Chronic Obstructive Pulmonary Disease: Evidence from the Cohort Study of NHANES 2007–2018

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Background: Chronic obstructive pulmonary disease (COPD) is the third leading cause of death worldwide. Previous studies have explored the relationship between different dietary patterns, systemic inflammation index (SII)and the risk of COPD. However, the joint effects and interactions between SII and HEI-2015 in COPD have not been fully investigated. This study aimed to explore the relationships between COPD and SII and HEI-2015.

Methods: Data from the National Health and Nutrition Examination Surveys (NHANES) were utilized.Univariate and multivariate logistic regression analyzed the associations between SII and HEI–2015 with COPD. Restricted cubic spline (RCS) model analyzed the relationship between SII and HEI-2015 and COPD.Use the area enclosed under the ROC curve (AUC) to represent its predicted value. Interaction indices and subgroup analyses were performed. The Kaplan-Meier curve was used to evaluate the impact on mortality of COPD patients.

Results: This study included 10,898 participants.After adjusting,logistic regression analysis showed that higher SII (OR=1.03, 95% CI: 1.01–1.05) were associated with an increased risk of COPD, while higher HEI-2015 (OR=0.97, 95% CI: 0.96–0.99) reduced the risk.The RCS model observed a non-linear relationship between SII and HEI-2015 and COPD risk. Additionally, ROC showed a more significant advantage in predicting COPD prevalence (AUC=0.68). Interaction analysis indicated that SII and HEI-2015 might be independent influencing factors for COPD risk. Kaplan–Meier survival curves showed a lower all-cause mortality rate among in the group with high SII and low HEI-2015 ($p < 0.0001$).

Conclusion: The results of this study indicate that a higher SII level and a lower HEI-2015 are associated with COPD risk. COPD patients with higher SII levels combined with lower HEI - 2015 levels have a higher all-cause death risk.

Keywords: chronic obstructive pulmonary disease, systemic inflammation index, dietary quality, NHANES

Introduction

Chronic obstructive pulmonary disease (COPD) is an increasingly serious global health issue, imposing a huge burden on countries around the world. According to the World Health Organization (WHO), as of 2019, COPD has become the third-leading cause of death globally.[1] Approximately 4.7% of the global population is affected by COPD, In the United States alone, approximately 15.5 million adults have been diagnosed with COPD, with the total economic burden

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estimated at $101 billion in 2020.[2] In Asia, the figure is as high as 6.2%.[3] COPD is a chronic progressive disease characterized by partially reversible airflow limitation.[4] Smoking is widely recognized as the main risk factor,[5] but identifying and managing other potential risk factors is a crucial for reducing the health and economic burden of COPD patients.

In recent years, increasing evidence suggests that systemic immune-inflammation index (SII) and dietary quality (DQ) play important roles in the development of COPD.[6,7] SII is a comprehensive index that measures systemic immune-inflammatory responses in the body. Initially developed as a prognostic indicator for adverse outcomes in cancer patients, SII is calculated as: platelet count × neutrophil count / lymphocyte count.[8] Studies have shown that SII can objectively reflect the balance between inflammation and immunity in cancer patients.[9,10] Moreover, SII has been extended to various diseases, including coronary heart disease,[11] rheumatoid arthritis,[12] and liver fibrosis,[13] and is used to track treatment responses. Recent studies have indicated that SII levels are positively correlated with COPD prevalence.[14] Higher SII levels are associated with a higher prevalence of COPD, and COPD patients with higher SII levels have an increased risk of all-cause mortality.[14] In addition, studies have shown that NLR, PLR, and SII levels are elevated in frequent AECOPD patients, serving as cost-effective markers for exacerbation frequency and severity.[15] Therefore, elevated SII levels may be a key risk factor for poor prognosis in COPD patients.

Additionally, dietary quality is closely related to COPD. Numerous studies have emphasized the importance of specific dietary patterns. Increasing epidemiological evidence suggests that high-quality diets are associated with better lung function and a lower risk of COPD.[16] Diets rich in fruits, vegetables, whole grains, and fish have been found to reduce the risk of COPD.[17,18] However, many studies focus on isolated nutrients rather than overall dietary quality. Therefore, the Healthy Eating Index-2015 (HEI-2015), a widely used measure of overall dietary quality, is introduced here. HEI-2015 aligns with the Dietary Guidelines for Americans.[19,20] Higher HEI-2015 scores indicate healthier and more balanced diets. Current research has shown significant correlations between HEI and various disease risks, including obesity,[21] depression,[22] and diabetes.[23] However, the relationship between HEI-2015 and COPD, particularly in middle-aged and elderly populations, remains insufficiently studied.

Notably, previous studies have primarily focused on the individual effects of systemic immune-inflammation index and dietary quality on COPD, with few studies examining the potential interaction between these two factors. Therefore, this study plans to comprehensively analyze data from the National Health and Nutrition Examination Survey (NHANES) to explore how systemic immune-inflammation index and dietary quality jointly influence the development of COPD in middle-aged and elderly adults. The goal is to identify and quantify the potential benefits of systemic immune-inflammation index levels and dietary quality in preventing or managing COPD, thereby improving patient health outcomes and reducing disease burden.

Methods

Source of Data and Study Population

NHANES is a series of complex, multi-stage, probabilistic sampling surveys of the United States. NHANES data is released on a 2-year cycle. We analysed data from 2007–2018, which included the population aged 40 years and over. This database is collected and maintained by the National Center for Health Statistics (NCHS), which is under the Centers for Disease Control and Prevention (CDC) of the United States. NHANES aims to assess the health and nutritional status of adults and children in the United States.[24] It collects and analyzes data representative of the non-institutionalized US population by using a complex multi-stage. These datasets are aimed to research purposes, and NCHS provides authorization for researchers to use the data. NHANES participants first finish a household interview and are then invited to a mobile examination center (MEC) for comprehensive health examinations, including physical examinations, professional measurements, and laboratory tests. Therefore, the judgements for participants in NHANES database are comprehensive, reliable, and multidimensional, equivalent to population-level assessments.[25]

In our study, we selected 10,898 participants aged 40 and above from six consecutive NHANES cycles from 2007 to 2018. Exclusion criteria were as follows: participants without HEI-2015 data; participants without SII data; participants without COPD data; missing covariates; and missing survival status (Figure 1).

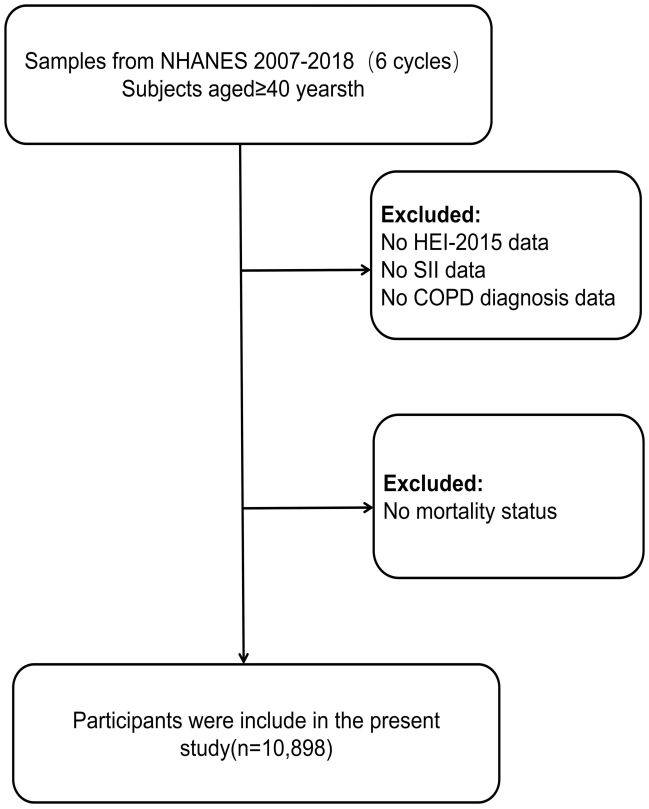

Figure 1 Flowchart of participants selection from NHANES 2007–2018.

Definition of HEI-2015

Dietary intake data were collected through two 24-hour recall interviews, which were conducted by professionally trained dietary interviewers.[24] The first interview was conducted face-to-face, and the second was conducted by phone 3 to 10 days after the initial interview. All participants were told to recall and report the types and quantities of foods and beverages consumed in the past 24 hours, and the average of these two reports was used to estimate dietary intake.[26] The energy and nutrient intake of all food items were calculated using the Food and Nutrient Database for Dietary Studies.[27] The dietary data from NHANES database used to construct the Healthy Eating Index-2015 (HEI-2015). HEI-2015 is a tool for evaluating an individual's diet quality, and this score reflects compliance with the 2015–2020 Dietary Guidelines for Americans (DGA).[28] HEI-2015 is not based on absolute intake amounts but on energy density per 1,000 kcal (Supplementary Table S1). It includes 13 components, among which 9 are adequacy components that encourage sufficient intake: total fruits, whole fruits, total vegetables, greens and beans, total protein foods, seafood and plant proteins (each 0–5 points), whole grains, dairy, and fatty acids (each 0–10 points). The higher the intake of these components, the higher the score. The remaining 4 components are moderation components, with recommended intake limits: sodium, refined grains, added sugars, and saturated fats (each 0–10 points). Lower intake of these components results in higher scores.[19] The HEI-2015 score was calculated using SAS code[29] The theoretical range for HEI-2015 scores is 0–100, with higher scores indicating better overall dietary quality. Participants with an average HEI score equal to or above the 60th percentile were considered to have a healthy diet (adhering to dietary guidelines or consuming healthy foods); otherwise, they were considered to have an unhealthy diet.[30]

Definition of SII

Whole blood samples were collected from eligible participants at the NHANES mobile examination center. The development and calculation of the SII have been reported in previous studies.[8] SII includes platelet count, neutrophil count, and lymphocyte count, calculated using the formula: SII = platelet count×neutrophil count/lymphocyte count.

Laboratory data from the 5-part differential complete blood count were assessed using the Beckman Coulter DxH 800 instrument, which quantifies neutrophils, lymphocytes, platelets, and monocytes, expressed as 1,000 cells/μL. Complete blood count parameters were measured and sized according to Beckman Coulter methods. SII was log10-transformed to ensure normal distribution.

Diagnostic Criteria of COPD

In the NHANES questionnaire (Medical Conditions dataset), physician-diagnosed COPD was determined by a positive response to any of the following questions: 1) After the participant inhaled a β2-adrenergic bronchodilator drug, the ratio of forced expiratory volume in 1 second to forced vital capacity (FEV_1/FVC) was < 0.70; 2) Participants who were told by a doctor or other health professional that they had emphysema or chronic bronchitis. This method of identifying COPD patients has been successfully implemented in many previous studies using NHANES data.[31,32] Participants who answered "yes" to any of these questions were coded as having COPD.

Covariates

Data on age, gender, race, poverty-income ratio (PIR), marital status, and educational level were collected by trained NHANES interviewers using the Household and Sample Person Demographic Questionnaire and the Computer - Assisted Personal Interviewing (CAPI) system (Rummit Corp., New York, USA). Data were weighted according to NHANES protocols. Body mass index (BMI) was calculated based on NHANES examination measurements as weight (kg) divided by height (m) squared. Hypertension was defined as an Yes response to at least one of the following questions: "Have you ever been told by a doctor or other health professional that you have hypertension, also called high blood pressure?" or "Have you ever been told to take prescription medication for your (high blood pressure)?" or an average systolic blood pressure (SBP) ≥140 mmHg or diastolic blood pressure (DBP) ≥90 mmHg over three consecutive measurements. Diabetes mellitus (DM) was identified based on an Yes response to at least one of the following questions: "Are you now taking insulin?" or "Has a doctor ever told you that you have diabetes?" or "Are you now taking medication to lower your blood sugar?" or laboratory data showing glycated hemoglobin (HbA1c) ≥6.5%, fasting blood glucose ≥126 mg/dL, or oral glucose tolerance test (OGTT) blood glucose ≥200 mg/dL.[33] Alcohol consumption was categorized as never drinker (no alcohol consumption in the past 12 months and fewer than 12 drinks in a lifetime), current drinker (alcohol consumption in the past 12 months), and former drinker (no alcohol consumption in the past 12 months but more than 12 drinks in a lifetime). Smoking status was categorized into three groups: never smoker (never smoked or smoked fewer than 100 cigarettes and quit), former smoker (smoked more than 100 cigarettes but currently does not smoke), and current smoker (smoked more than 100 cigarettes and answered "yes" to "Do you now smoke cigarettes?").[34] Cardiovascular disease history was defined based on a Yes response to questions about physician-diagnosed congestive heart failure, myocardial infarction, angina, coronary heart disease, or stroke. Laboratory data, including platelet, neutrophil, white blood cell, monocyte, lymphocyte, and red blood cell counts, were obtained from NHANES laboratory data files.

Statistical Analysis

This study data is weighted using NHANES dietary interview sample weights to account for the complex survey design. We used the dietary day one sample weight set (WTDRD 1) for weighted analysis. Summarize baseline characteristics using descriptive statistics, where Continuous variables are presented as means ± SD and categorical variables as percentages; between-group comparisons used weighted χ^2-tests. Multivariable logistic regression was performed in three sequential models: Model 1, unadjusted; Model 2, adjusted for age, sex, and race; and Model 3, further adjusted for education, poverty-to-income ratio, marital status, BMI, hypertension, and cardiovascular disease, with results reported as ORs (95% CIs). Non-linear relationships between log10-transformed SII and HEI-2015 with COPD were examined using four-knot restricted cubic splines, and ROC curves were used to compare the AUCs across the three models. Interaction effects were quantified with RERI, API, and the synergy index (S), and subgroup analyses were stratified by age, sex, race, and other covariates. All-cause mortality among COPD patients was assessed with Kaplan–Meier curves

and Cox regression. All tests were two-sided, with $P < 0.05$ considered significant, and analyses were conducted in R Studio (v4.3.1).

Results

Baseline Characteristics of the Population

This study data from NHANES were derived from the NHANES database (2007–2018), including 10,898 participants aged 40 and above. Participants were grouped based on the presence or absence of COPD, and detailed study characteristics are presented in Table 1. Among the participants, 1,764 were diagnosed with COPD, including 940 males (53.3%) and 824 females (46.7%). Compared to the non-COPD group, COPD participants were more likely to be older, non-Hispanic white, have higher education levels, be current or former smokers, be current or former alcohol and have hypertension. Significant differences were also observed between the two groups in terms of PIR, dietary quality, cardiovascular disease, and diabetes (all $p < 0.05$). Overall, except for sex, monocyte count, lymphocyte count, and red

Table 1 Basic Characteristics of Participants (n = 10898) in the NHANES 2007–2018

Demographic and Health Variables	Total 10.898	COPD Symptoms Status		P value
		No 9134	Yes 1764	
Age group (years), mean (SD)				<0·0001
40–60 years,n (weighted %)	5,828(53.5%)	5,067(55.5%)	761(43.1%)	
60–80 years, n (weighted %)	4,403(40.4%)	3,481(38.1%)	922(52.3%)	
Over 80 years, n (weighted %)	667(6.1%)	586(6.4%)	81(4.6%)	
Sex, n (weighted %)				0·70
Male	5,634(51.7%)	4,694(51.4%)	940(53.3%)	
Female	5,264(48.3%)	4,440(48.6%)	824(46.7%)	
Race or ethnicity, n (weighted %)				<0·0001
Non-Hispanic White	5,258(48.2%)	4,122(45.1%)	1,136(64.4%)	
Non-Hispanic Black	2,267(20.8%)	1,970(21.6%)	297(16.8%)	
Mexican American	1,309(12.0%)	1,204(13.2%)	105(6.0%)	
Other race (including multi-racial, other Hispanic)	2,064(18.9%)	1,838(20.1%)	226(12.8%)	
Education, n (weighted %)				<0·0001
<9th Grade	898(8.2%)	761(8.3%)	137(7.8%)	
9–11th Grade (Includes 12th grade with no diploma)	1,266(11.6%)	1,017(11.1%)	249(14.1%)	
High school graduate or GEDa or equivalent	2,501(22.9%)	2,022(22.1%)	479(27.2%)	
Some college or AAb	3,223(29.6%)	2,681(29.4%)	542(30.7%)	
College graduate or above	3,010(27.6%)	2,653(29.0%)	357(20.2%)	
Household income and PIRc, mean (SD)				<0·0001
Below poverty (< 1.0), n (weighted %)	1,722(15.8%)	1,376(15.1%)	346(19.6%)	
Above poverty (³ 1.0), n (weighted %)	9,176(84.2%)	7,758(84.9%)	1,418(80.4%)	

(*Continued*)

Table 1 (Continued).

Demographic and Health Variables	Total 10.898	COPD Symptoms Status		P value
		No 9134	Yes 1764	
Marital status, n (weighted %)				0·0070
Married or living with partner	6,995(64.2%)	5,964(65.3%)	1,031(58.4%)	
Widowed or divorced or separated	2,984(27.4%)	2,397(26.2%)	587(33.3%)	
Never married	919(8.4%)	773(8.5%)	146(8.3%)	
BMI (kg/m2), mean (SD)				0·016
Underweight (<18.5kg/m2), n (weighted %)	121(1.1%)	83(0.9%)	38(2.2%)	
Normal (18.5–24.9kg/m2), n (weighted %)	2,549(23.4%)	2,105(23.0%)	444(25.2%)	
Overweight (25–29.9kg/m2), n (weighted %)	3,943(36.2%)	3,347(36.6%)	596(33.8%)	
Obese (≥30kg/m2), n (weighted %)	4,285(39.3%)	3,599(39.4%)	686(38.9%)	
Smoking status, n (weighted %)				<0·0001
Nonsmoker	5,642(51.8%)	5,131(56.2%)	511(29.0%)	
Former smoker	3371(30.9%)	2,678(29.3%)	693(39.3%)	
Current smoker	1,885(17.3%)	1,325(14.5%)	560(31.7%)	
Alcohol use, n (weighted %)				0.023
Never	1,297(11.9%)	1,151(12.6%)	146(8.3%)	
Former	1,716(15.7%)	1,453(15.9%)	263(14.9%)	
Current	7,885(72.4%)	6,530(71.5%)	1,355(76.8%)	
Comorbidities, n (weighted %)				
Hypertension	5,571(51.1%)	4,532(49.6%)	1,039(58.9%)	0·0006
Diabetes	2,458(22.6%)	2,018(22.1%)	440(24.9%)	0·0007
Cardiovascular	1,411(12.9%)	1,033(11.3%)	378(21.4%)	<0·0001
Laboratory indicators, n (weighted %)				
Platelet	238±(61)	237±(60)	244±(64)	0·013
Neutrophils	4·21±(1·55)	4·15±(1·52)	4·51±(1·63)	<0·0001
WBC	7·08±(2·21)	7·01±(2·20)	7·43±(2·22)	<0·0001
Monocyte	0·56±(0·20)	0·56±(0·20)	0·57±(0·20)	0·19
Lymphocyte	2·06±(1·16)	2·06±(1·18)	2·07±(1·09)	0·40
RBC	238±(61)	237±(60)	244±(64)	0·28
HEI-2015, n (weighted %)				0.0004
Unhealthy diet	5,440(49.9%)	4,440(48.6%)	1000(56.7%)	
Healthy diet	5,458(50.1%)	4,694(51.4%)	764(43.3%)	

(*Continued*)

Table 1 (Continued).

Demographic and Health Variables	Total 10.898	COPD Symptoms Status		P value
		No 9134	Yes 1764	
SII, n (weighted %)	533±(312)	522±(294)	590±(383)	<0·0001
Log_SII, n (weighted %)	3,762(36%)	3,027(35%)	735(42%)	0·0006
HEI-SII				<0·0001
Low SII&Low HEI-2015	3,479(31.9%)	2,911(31.9%)	568(32.2%)	
Low SII&High HEI-2015	3,657(33.6%)	3,196(35.0%)	461(26.1%)	
High SII&Low HEI-2015	1,961(18.0%)	1,529(16.7%)	432(24.5%)	
High SII&High HEI-2015	1,801(16.5%)	1,498(16.4%)	303(17.2%)	

Notes: Continuous variables are presented as the means with SDs. Categorical variables are summarized as proportions. $p < 0.05$ was considered statistically significant.

Abbreviations: COPD, Chronic Obstructive Pulmonary Disease; PIR, Poverty Income Ratio; BMI, Body Mass Index; SII, Systemic Immune-Inflammation Index; WBC, White Blood Cell; HEI-2015, Healthy Eating Index.

blood cell count, significant differences in baseline characteristics were observed between the non-COPD and COPD groups. The grouping by different lifestyle patterns and gender is detailed in Supplementary Tables S2 and S3.

Relationship Between SII, Dietary Quality, and COPD

To further explore the relationship between dietary quality, SII, and COPD, Table 2 presents three weighted logistic regression models. From the three models, it can be seen that log-SII is positively associated with COPD, while HEI is

Table 2 Association Between SII and Dietary Quality and Prevalence of COPD Among 10,898 Study Participants

Variable	OR (95% CI)		
	Model 1	Model 2	Model 3
HEI-2015			
Low	1 [Reference]	1 [Reference]	1 [Reference]
High	0.96 (0.94 to 0.98)	0.96(0.94 to 0.98)	0.97(0.96 to 0.99)
Log-SII			
Low	1 [Reference]	1 [Reference]	1 [Reference]
High	1.04(1.02 to 1.06)	1.03 (1.01 to 1.06)	1.03(1.01 to 1.05)
Age group			
40–60 years		1 [Reference]	1 [Reference]
60–80 years		1.07(1.05 to 1.11)	1.05(1.03 to 1.08)
Over 80		0.97(0.95 to 1)	0.92(0.89 to 0.96)
Gender			
Female		1 [Reference]	1 [Reference]
Male		0.99(0.97 to 1.01)	1.00(0.98 to 1.02)
Race			
Non-Hispanic White		1 [Reference]	1 [Reference]
Mexican American		0.90(0.88 to 0.92)	0.87(0.85 to 0.90)
Non-Hispanic Black		O.94(0.92 to 0.97)	0.92(0.90 to 0.94)
Other race (including multi-racial, other Hispanic)		O.96(0/93 to 0.99)	0.95(0.92 to 0.97)

(*Continued*)

Table 2 (Continued).

Variable	OR (95% CI)		
	Model 1	Model 2	Model 3
Education			
Less than 9th grade			1 [Reference]
9–11th grade (Includes 12th grade with no diploma)			1.03(0.98 to 1.09)
Some college or AA degree			1.00(0.95 to 1.04)
High school graduate/GED or equivalent			1.02(0.97 to 1.06)
College graduate or above			0.95(0.91 to 0.99)
PIR			
Below poverty			1 [Reference]
Above poverty			0.94(0.90 to 0.98)
Marital			
Never married			1 [Reference]
Married or living with partner			0.98(0.94 to 1.02)
Widowed or divorced or separated			1.00(0.96 to 1.05)
BMI			
Normal			1 [Reference]
Underweight			1.15(1.02 to 1.30)
Overweight			0.99(0.96 to 1.01)
Obese			0.97(0.94 to 1.00)
Hypertension			
No			1 [Reference]
Yes			1.04(1.00 to 1.07)
Cardiovascular			
No			1 [Reference]
Yes			1.11(1.07 to 1.16)

Notes: The Log-SII and HEI-2015 was converted from a continuous variable to a categorical variable. Data are presented as OR (95% CI). Model 1: no covariates were adjusted. Model 2 age, sex, and race were adjusted. Model 3 age,sex, and race,education level, poverty, marital status, BMI, cardiovascular disease, and hypertension were adjusted. 95% $p < 0.05$ was considered statistically significant.
Abbreviations: CI, 95% confidence interval; OR, odds ratio.

negatively associated with COPD. Model 1 is a crude model, patients with high-scoring HEI-2015 have a 0.96-fold lower odds of COPD than patients with low-scoring HEI-2015 (OR: 0.96, 95% CI: 0.94 to 0.98).However, patients with high log-SII have a 1.04-fold higher odds of COPD compared to those in the low log-SII (OR = 1.04, 95% CI: 1.02 to 1.06). Model 2 adjusted for demographic characteristics (ie, sex, age group, race). Additionally, Model 3 further adjusted for education, poverty-to-income ratio, marital status, BMI, hypertension history, and cardiovascular disease history. In both Model 2 and Model 3, additional covariate adjustments did not materially attenuate the associations of SII and HEI-2015 with COPD. Moreover, Model 3 demonstrated that a higher HEI-2015 score remained significantly associated with decreased COPD prevalence (OR: 0.97; 95% CI: 0.96 to 0.99). Conversely, higher log-SII showed a significant association with increased COPD risk (OR: 1.03; 95% CI: 1.01 to 1.05). Besides, in the adjusted model, age,race,PIR, cardiovascular were significantly correlated with COPD statues.

To further investigate potential nonlinear associations between SII and HEI and COPD risk after adjusting for age and sex, restricted cubic spline (RCS) analyses were performed. As illustrated in Figure 2, the relationship between Log-SII and COPD risk demonstrated significant nonlinear characteristics, with a P-nonlinear value of 0.006 and P-overall < 0.001, indicating a statistically significant nonlinear correlation.Besides,it reveals an inverted L-shaped dose-response pattern between Log-SII and COPD risk, with an identified inflection point at 2.887 (P-nonlinear = 0.006). In contrast, no significant nonlinear relationship was observed between HEI and COPD risk, as evidenced by a nonsignificant P-nonlinear value of 0.786 (Figure 3).

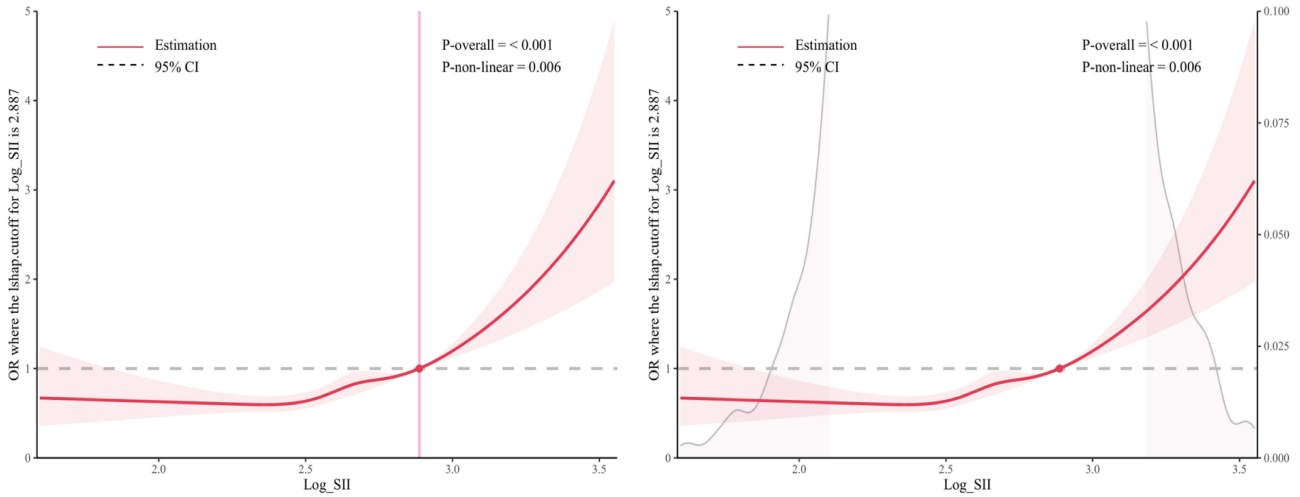

Figure 2 RCS analysis of the association between Log-SII and COPD.The association was adjusted for age, sex. p<0.05 was considered statistically significant.
Abbreviations: SII, Systemic Immune-Inflammation Index; RCS, restricted cubic spline.

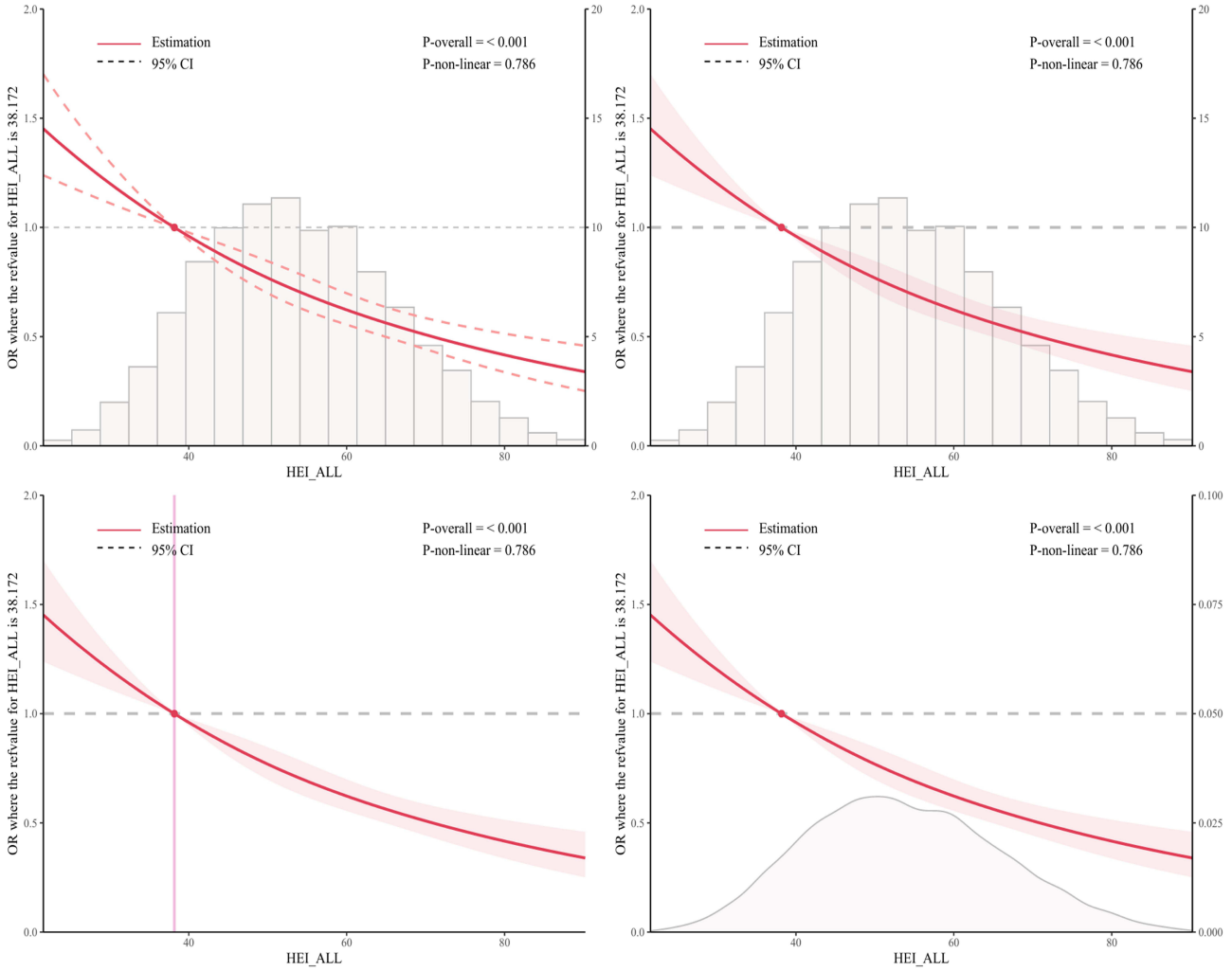

Figure 3 RCS analysis of the association between HEI and COPD. p<0.05 was considered statistically significant.
Abbreviations: HEI_ALL, Healthy Eating Index_ALL; RCS, restricted cubic spline.

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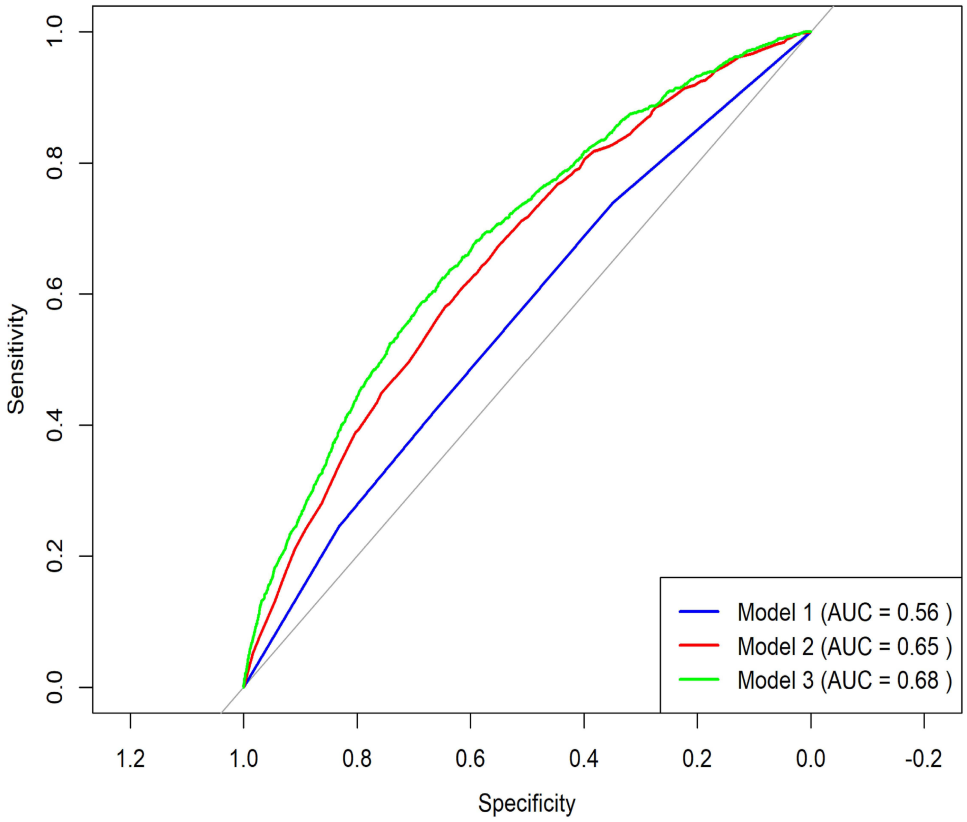

Figure 4 ROC curve analysis. ROC curves for predicting COPD risk by SII and HEI. Model 1: no covariates were adjusted. Model 2 age, sex, and race were adjusted Model 3 sex, and race,education level, poverty, marital status, BMI, cardiovascular disease, and hypertension were adjusted. $p < 0.05$ was considered statistically significant.
Abbreviations: 95% CI, 95% confidence interval; OR, odds ratio.

Additionally, we employed ROC curves to conduct a comparative analysis of Model 1, Model 2, and Model 3 (Figure 4). Compared to Model 1 (AUC = 0.56) and Model 2 (AUC = 0.65), Model 3 (AUC = 0.68) demonstrated a higher predictive value for COPD prevalence.

Interaction Analysis of SII and Dietary Quality Levels on Outcomes

Next, we further investigated the joint effects of HEI-2015 and SII under different conditions to analyze the interaction between these two variables. Details are shown in Table 3 and Table 4. The risk of COPD in individuals with high log-SII and low HEI-2015 was 1.28-fold higher (OR:1.28,95% CI: 1.11 to 1.49) than in those with low log-SII and low HEI-2015. In contrast, individuals with low log-SII and high HEI-2015 had a 0.81-fold lower risk of COPD (OR:0.81,95% CI: 0.70 to 0.93) compared to those with low log-SII and low HEI-2015. Thus, individuals with low inflammation index and high healthy eating index had a lower risk of COPD. Furthermore, when log-SII increased from low to high, the risk

Table 3 The Interaction of the HEI_2015 and Log_SII on the Prevalence of COPD

	Log_SII_Low	Log_SII_High	Effect of Log_SII_High Within the Strata of HEI_2015High
	OR [95% CI]	**OR [95% CI]**	**OR [95% CI]**
HEI_2015_Low	1 [Reference]	1.28 [1.11, 1.49]	1.28 [1.11, 1.49]
HEI_2015_High	0.81 [0.70, 0.93]	1.04 [0.89, 1.23]	1.29[1.11, 1.52]
Effect of HEI_2015_High within the strata of Log_SII_High	0.81 [0.70, 0.93]	0.81 [0.68, 0.96]	

Notes: Age, sex, and race, education level, poverty, marital status, BMI, cardiovascular disease, and hypertension were adjusted.
Abbreviations: SII, Systemic Immune-Inflammation Index; HEI-2015, Healthy Eating Index; 95% CI, 95% confidence interval; OR, odds ratio.

Table 4 Additive Interaction Between the HEI_2015 and Log_SII

Index	OR	95% CI
RERI	−0.05	[−0.28, 0.19]
API	−0.04	[−0.27, 0.18]
S	0.48	[0.01, 18.57]

Notes: Age, sex, and race, education level, poverty, marital status, BMI, cardiovascular disease, and hypertension were adjusted.
Abbreviations: RERI, Relative Excess Risk Of Interaction; API, Attributable Proportion Of TeractionS, Synergy Index; 95% CI, 95% confidence interval; OR, odds ratio.

increased (OR from 1 to 1.28) if HEI-2015 was low, but the risk change was minimal (OR from 1 to 1.04) if HEI-2015 was high. This suggests that a high level of healthy diet can improve the impact of inflammation on COPD risk. Additionally, the table shows that in individuals with high healthy eating index, high inflammation index was associated with a 29% increased risk of COPD (OR: 1.29; 95% CI: 1.11 to 1.52). Conversely, in individuals with high inflammation index, high healthy eating index was associated with a 19% reduced risk of COPD (OR: 0.81; 95% CI: 0.68 to 0.96).

To further investigate potential interactions between HEI-2015 and SII, interaction index-synergy index (S), relative excess risk of interaction (RERI), and attributable proportion of interaction (API)-were calculated (Table 4). Both RERI and API were −0.05, suggesting a potential negative correlation; however, their 95% confidence intervals (CIs) included 0, indicating that the interaction effects of these metrics were non-significant. The synergy index (S) was 0.48 (95% CI: 0.01 to 18.57), with a wide confidence interval spanning from 0.01 to 18.57, reflecting unstable effect estimates and statistical non-significance. These results demonstrate no significant interaction between HEI-2015 and SII, suggesting that both factors independently influence COPD risk.

Subgroup Analysis

Subgroup analyses stratified by age group, sex, race, marital status, education level, PIR, smoking, alcohol consumption, hypertension, cardiovascular disease, and diabetes were conducted to explore the relationship between lifestyle factors and COPD. Significant associations were observed in the 40–60 age group and among individuals with PIR ≥1 ($P < 0.05$). Notably, males with high HEI-2015 scores and low SII levels had reduced COPD risk (OR: 0.70, 95% CI: 0.54–0.91), as did non-Hispanic White individuals (OR: 0.66, 95% CI: 0.52–0.82) and those with hypertension (OR: 0.65, 95% CI: 0.50–0.86). Interaction analysis revealed significant effect modifications only for sex and race ($P < 0.05$) (Table 5).

Different Lifestyle Groups and COPD All-Cause Mortality

The joint relationship between HEI-2015, SII, and COPD mortality risk is presented in Table 6. Compared to individuals with low log-SII and low HEI-2015, those with high log-SII and high HEI-2015 had a 36% increased risk of all-cause mortality (HR: 1.36, 95% CI: 1.10–1.68). In contrast, combinations of high log-SII with low HEI-2015 or low SII with high HEI-2015 showed no significant association with COPD mortality. After further adjusting the age, gender, education and PIR, the results remained similar (Supplementary Tables S4 and S5). Kaplan-Meier survival curves (Figure 5) demonstrated a gradual decline in survival probability over time across all groups. The survival curves differed significantly among the four groups ($P < 0.0001$), indicating that distinct combinations of SII and dietary quality profoundly impacted survival outcomes in COPD patients. Specifically, the combination of low SII and high HEI-2015 was associated with higher survival probabilities at most time points, suggesting that healthier dietary habits and lower systemic inflammation levels may improve survival. Conversely, the combination of high SII and low HEI-2015

Table 5 Subgroup Analyses of the Association Between Log-SII and HEI-2015 and COPD

Lifestyle Group								
	High_SII and Low HEI-2015	Low SII and Low HEI-2015		Low SII and High HEI-2015		High_SII and High HEI-2015		P for Interaction
	OR (95% CI)	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P	
Age group								0.395
40–60 years	1[Reference]	0.67(0.53–0.87)	0.003	0.54(0.44–0.74)	<0.001	0.65(0.47–0.9)	0.012	
60–80 years	1[Reference]	0.87(0.63–1.19)	0.383	0.57(0.41–0.8)	0.002	0.79(0.56–1.11)	0.18	
Over 80	1[Reference]	1.68(0.66–4.29)	0.28	0.9(0.36–2.26)	0.822	1.03(0.41–2.59)	0.95	
Gender								0.038
Female	1[Reference]	0.78(0.59–1.02)	0.069	0.5(0.37–0.66)	<0.001	0.58(0.41–0.82)	0.002	
Male	1[Reference]	0.74(0.53–1.04)	0.084	0.7(0.54–0.91)	0.01	0.98(0.72–1.32)	0.885	
Race								0.046
Mexican American	1[Reference]	0.81(0.43–1.51)	0.504	0.91(0.49–1.71)	0.778	1.57(0.76–3.23)	0.224	
Non-Hispanic Black	1[Reference]	0.83(0.54–1.29)	0.41	0.6 8(0.43–1.08)	0.107	0.89(0.49–1.61)	0.7	
Non-Hispanic White	1[Reference]	0.81(0.63–1.05)	0.118	0.66(0.52–0.82)	0.0001	0.75(0.58–0.98)	0.039	
Other race	1[Reference]	0.52(0.28–0.97)	0.042	0.28(0.14–0.56)	<0.001	0.56(0.28–1.12)	0.103	
Marital								0.881
Married or living with partner	1[Reference]	0.75(0.58–0.97)	0.031	0.64(0.5–0.81)	<0.001	0.76(0.59–0.98)	0.039	
Never married	1[Reference]	0.57(0.27–1.19)	0.14	0.37(0.18–0.76)	0.009	0.69(0.28–1.73)	0.435	
Widowed or divorced or separated	1[Reference]	0.82(0.6–1.11)	0.205	0.55(0.39–0.77)	0.001	0.74(0.52–1.05)	0.091	
Education								0.939
9–11th grade (Includes 12th grade with no diploma)	1[Reference]	0.9(0.55–1.47)	0.673	0.45(0.24–0.86)	0.018	0.8(0.41–1.57)	0.518	
College graduate or above	1[Reference]	0.91(0.54–1.52)	0.713	0.83(0.56–1.25)	0.373	0.87(0.55–1.37)	0.546	
High school graduate/ GED or equivalent	1[Reference]	0.62(0.41–0.92)	0.021	0.59(0.37–0.93)	0.025	0.91(0.6–1.36)	0.635	
Less than 9th grade	1[Reference]	0.800.42–1.53)	0.504	0.53(0.26–1.08)	0.084	1.6(0.75–3.39)	0.228	
Some college or AA degree	1[Reference]	0.79(0.56–1.11)	0.182	0.68(0.5–0.93)	0.019	0.78(0.53–1.5)	0.214	
PIR								0.365
Above poverty	1[Reference]	0.71(0.58–0.88)	0.003	0.61(0.5–0.74)	<0.001	0.74(0.59–0.93)	0.01	
Below poverty	1[Reference]	0.92(0.63–1.33)	0.642	0.45(0.29–0.72)	0.001	0.95(0.54–1.65)	0.848	
Smoke								0.325
Non smoke	1[Reference]	0.82(0.59–1.15)	0.251	1.05(0.77–1.43)	0.773	1.02(0.79–1.41)	0.928	
Past smoke	1[Reference]	0.86(0.61–1.21)	0.378	0.67(0.48–0.92)	0.016	1.06(0.73–1.54)	0.747	
Now smoke	1[Reference]	0.73(0.53–1.00)	0.054	0.65(0.4–1.05)	0.081	0.73(0.45–1.17)	0.191	
Drink								0.747
Non drink	1[Reference]	0.71(0.31–1.62)	0.420	0.41(0.19–0.88)	0.024	0.74(0.37–1.46)	0.385	
Past drink	1[Reference]	0.65(0.4–1.07)	0.095	0.45(0.27–0.74)	0.002	0.31(0.18–0.54)	<0.001	
Now drink	1[Reference]	0.78(0.61–0.98)	0.037	0.64(0.52–0.79)	<0.001	0.85(0.66–1.1)	0.225	
Hypertension								0.964
No	1[Reference]	0.77(0.58–1.02)	0.075	0.55(0.41–0.73)	<0.001	0.73(0.52–1.03)	0.077	
Yes	1[Reference]	0.73(0.56–0.96)	0.026	0.65(0.5–0.86)	0.003	0.73(0.55–0.97)	0.035	
Cardiovascular								0.077
No	1[Reference]	0.77(0.62–0.96)	0.023	0.60(0.49–0.73)	<0.001	0.79(0.63–1)	0.05	
Yes	1[Reference]	0.74(0.46–1.19)	0.222	0.54(0.32–0.91)	0.023	0.53(0.33–0.85)	0.009	
Diabetes								0.573
No	1[Reference]	0.76(0.6–0.97)	0.031	0.57(0.46–0.70)	<0.001	0.77(0.58–1.01)	0.061	
Yes	1[Reference]	0.72(0.50–1.04)	0.087	0.69(0.44–1.08)	0.111	0.65(0.42–1.03)	0.067	

Notes: $p < 0.05$ was considered statistically significant.

Abbreviations: SII, Systemic Immune-Inflammation Index; HEI-2015, Healthy Eating Index; PIR, Poverty Income Ratio; BMI, Body Mass Index; 95% CI, 95% confidence interval; OR,odds ratio.

Table 6 Joint Relationship Between HEI-2105 and SII and Mortality

Lifestyle Group	HR	95% CI	P
Factor (HEI_SII)			**<0.001**
Low_SII and Low HEI_2015	1[Reference]	1[Reference]	
High_SII and High HEI_2015	1,36	1.10,1.68	
High_SII and Low HEI_2015	1.17	0.95,1.453	
Low_SII and High HEI_2015	0.87	0.70,1.08	

Notes: p < 0.05 was considered statistically significant.
Abbreviations: SII, Systemic Immune-Inflammation Index; HEI-2015, Healthy Eating Index; HR, Hazard Ratio; 95% CI, 95% confidence interval.

correlated with poorer survival outcomes, likely reflecting the detrimental effects of chronic inflammation and poor dietary quality.

Discussion

This study, based on NHANES data, highlights significant associations between systemic immune-inflammation index, dietary quality, and COPD in middle-aged and elderly adults. In this cross-sectional analysis of 10,898 participants, elevatedlogSII levels were associated with increased COPD risk, while higher HEI-2015 scores were linked to reduced risk in unadjusted models. And then the SII might exhibit a nonlinear dose-response relationship with COPD risk. Furthermore, the ROC curve analysis indicated that, compared to Model 1 and Model 2, Model 3 exhibited a higher predictive value for COPD prevalence. These associations remained robust after adjusting for covariates, aligning with prior evidence underscoring the roles of inflammation and dietary quality in COPD pathogenesis.[35,36] Notably, while the combination of high log-SII and low HEI-2015 markedly elevated COPD risk, interaction indices (S, RERI, API) were statistically non-significant, supporting the notion that SII and dietary quality act as independent risk factors. Subgroup analyses further revealed that specific populations, such as non-Hispanic White individuals and hypertensive patients, benefited most from the protective effects of low SII combined with high HEI-2015. Survival analyses reinforced these

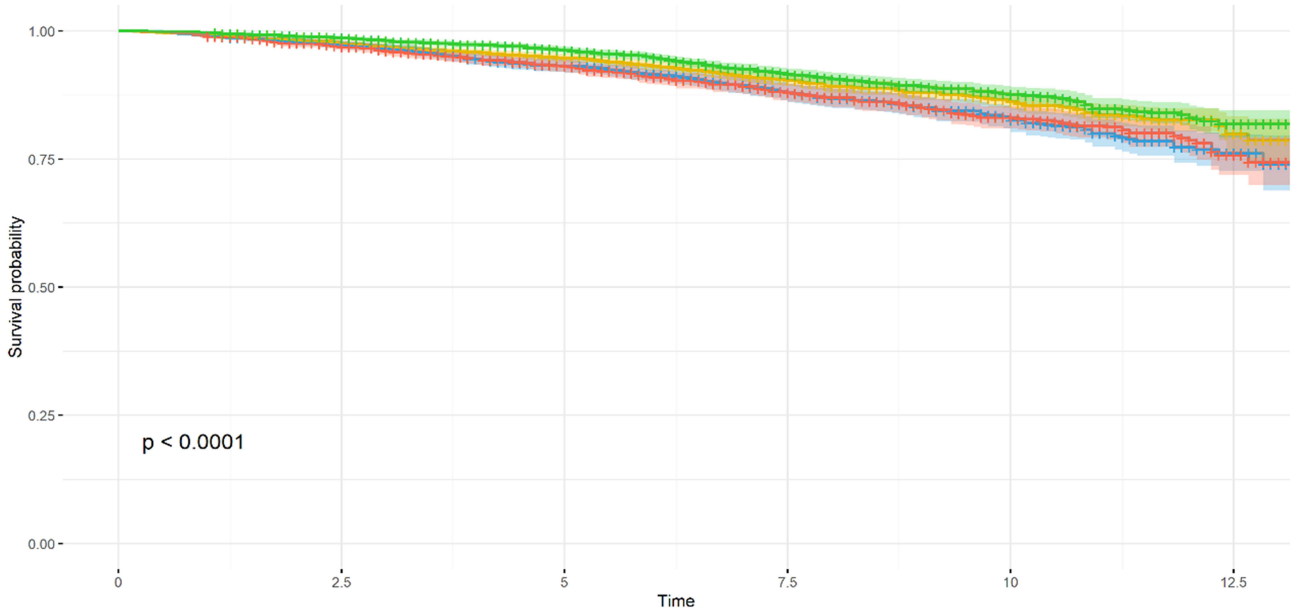

Figure 5 The Kaplan-Meier curves display the long-term all-cause mortality by different lifestyle group. factor(HEI_SII)=Low SII&Low HEI-2015: factor(HEI_SII)=High SII&High HEI-2015: factor(HEI_SII)=High SII&Low HEI-2015: factor(HEI_SII)=Low SII&High HEI-2015: .
Abbreviations: SII, Systemic Immune-Inflammation Index; HEI-2015: Healthy Eating Index-2015.

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findings, demonstrating that coexisting high systemic inflammation and poor dietary habits significantly increased mortality risk in COPD patients.

Our study further elucidates the crucial roles of inflammation and diet quality in the pathogenesis of COPD. COPD is a leading cause of morbidity and mortality globally. Nevertheless, the precise pathological and physiological mechanisms underlying the relationship between the SII and COPD remain elusive. From a biological standpoint, inflammation is one of the central factors in the development of COPD. As an indicator of the systemic inflammatory state, a high SII value reflects an intensification of the body's inflammatory response. An increasing body of research has demonstrated that inflammation and immune stress are associated with the risk and progression of COPD.[37,38] Stimuli such as cigarette smoke, bacteria, and viruses can trigger neutrophilic inflammation by promoting airway epithelial cells to release neutrophil mediators. Neutrophils are key inflammatory cells in the pathogenesis of COPD,[39] being particularly active during the acute exacerbation of COPD (AECOPD). They participate in pathogen clearance and release inflammatory mediators.[40,41] The inflammatory response damages lung tissue and disrupts the normal regulation of lung function, thereby further driving the progression of COPD. Additionally, previous studies have indicated that inflammatory markers have certain value in the treatment of COPD[7,42,43.] Meanwhile, HEI-2015 assesses the overall quality of an individual's diet. Existing research has shown that dietary factors play a significant role in ameliorating inflammation, oxidative stress, and regulating immunity and metabolism.[16,44] Previous reports have confirmed an inverse relationship between a healthy diet pattern and the incidence of COPD.[45–47] Besides,a case-control study in Iranian adults reported an inverse association between adherence to a low-carbohydrate diet (LCD) and the odds of COPD.[48] Similarly, Another study showed that a significant inverse association between adherence to the HEI-2010 recommendations and the odds of developing COPD in a Middle Eastern population.[49] Our findings, similar to those of these studies, suggest that adherence to the HEI-2015 recommendations reduces the risk of developing COPD and adverse outcomes, and therefore recommends that people with COPD consume more fruits, vegetables, and nuts while limiting their intake of sodium, refined grains, added sugars, and saturated fats.Thus, it can be inferred that a healthy dietary intake may be protective against COPD and its symptoms.[50]

SII, as a comprehensive inflammatory indicator, was first developed to predict the prognosis of patients after radical resection of liver cancer.[8] By incorporating the counts of neutrophils, lymphocytes, and platelets, SII can reflect the body's systemic inflammatory state more comprehensively. Compared with single inflammatory markers, it has a stronger ability to predict disease risks.[51] Multiple studies have shown that SII is associated with acute exacerbations in patients with stable-phase COPD and the all-cause mortality of COPD patients.[14,52] In addition, HEI-2015 quantifies diet quality by evaluating an individual's dietary pattern. It can reflect the nutritional intake status more effectively than single components of a healthy diet. Our study further highlights the advantages of SII and diet quality in assessing the risk of COPD and their potential application value. Although our study has observed that the combination of a high log-SII and a low HEI-2015 significantly increases the risk of COPD, the results of the interaction analysis suggest that SII and HEI-2015 may influence the risk of COPD as independent factors, each affecting the likelihood of COPD occurrence. Moreover, the results of the subgroup analysis indicate that in specific populations, such as non-Hispanic whites and individuals with hypertension, the combination of a low systemic inflammation index and a high healthy-diet-quality index is associated with a lower risk of COPD. This may provide a new perspective for the individualized prevention of COPD. More importantly, the survival curve analysis further strengthens this relationship, demonstrating a significant increase in the mortality of COPD patients in the context of co-existing chronic inflammation and poor dietary habits.However, NHANES provides representative data for the US population, and our results should consider the potential impact when applied to other populations, such as Asian populations with lower BMI thresholds.

It is worth noting that the selection of the NHANES database provides a solid data foundation and broad representativeness. Second, this is the first study to integrate SII and the HEI-2015 to examine their associations with COPD risk. By exploring the interplay between these two critical health indicators at a systemic level, this research offers a dual perspective on COPD pathogenesis, combining insights from inflammatory responses and dietary patterns. A "inflammation-nutrition" dual-path intervention framework was established for clinical practice, and individualized dietary intervention strategies were better specified for COPD patients. Rigorous adjustment for confounding factors further enhances the reliability and representativeness of the findings. Additionally, the interaction analysis provides evidence supporting the independent effects of SII and HEI-2015, while subgroup analyses offer valuable guidance for

developing personalized prevention and treatment strategies. Our study provides a new perspective for further mechanism exploration in the future, and at the same time, the integration of anti-inflammatory and dietary to establish the first "diet-inflammation" quantitative efficacy evaluation system will promote the update of clinical nutrition guidelines.

However, this study is not without limitations. It should be acknowledged that our analysis did not adjust for air pollution exposure—a known risk factor for COPD exacerbation and pulmonary function decline. Additionally, direct pulmonary function measurements were unavailable in NHANES, limiting our capacity to assess airflow limitation severity.Besides, Diagnosis of COPD relies on self-reported questionnaire data in NHANES and may be subject to recall bias; In particular, changes in the definition of COPD during the study may affect the uniformity of case identification. Survival analyses did not account for competitive risk and may overestimate COPD-specific mortality; At the same time, unadjusted for survivorship bias may lead to an underestimation of true risk.

Conclusions

In conclusion, higher SII levels and lower HEI-2015 scores were associated with increased COPD prevalence and mortality after statistical adjustment for potential confounders. Patients with COPD with elevated SIIs and suboptimal dietary quality are at higher risk of adverse outcomes. Although our study was unable to establish causal relationships, these associations highlighted the potential importance of inflammation and dietary quality in the management of COPD, and further statistical adjustments for potential confounders, taking into account non-significant variables, may help refine these associations and better understand their clinical relevance. In addition, the clinical significance of the results of this study is to suggest that clinicians should pay attention to the monitoring of inflammation level and the improvement of dietary quality in the management of COPD patients. From a public health perspective, this highlights the need for a combination of inflammatory management and nutritional interventions to reduce COPD morbidity and mortality in COPD prevention and management strategies.

Ethics Approval and Informed Consent

This study used the publicly available NHANES database, which was collected with patient informed consent and ethics approval from its source institution. Since this study only involves secondary analysis of existing and de-identified data, it meets the exemption conditions stipulated in Article 32 (1) and/or (2) of the "Measures for Ethical Review of Life Science and Medical Research Involving Human Subjects" issued by the National Health Commission on February 18, 2023, and has been approved by the First Affiliated Hospital of Nanjing Medical University.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

References

1. Christenson SA, Smith BM, Bafadhel M, et al. Chronic obstructive pulmonary disease. *Lancet*. 2022;399(10342):2227–2242. doi:10.1016/S0140-6736(22)00470-6
2. Croft JB, Wheaton AG, Liu Y, et al. Urban-rural county and state differences in chronic obstructive pulmonary disease - United States, 2015. *MMWR Morb Mortal Wkly Rep*. 2018;67(7):205–211. doi:10.15585/mmwr.mm6707a1
3. Fang L, Gao P, Bao H, et al. Chronic obstructive pulmonary disease in China: a nationwide prevalence study. *Lancet Respir Med*. 2018;6(6):421–430. doi:10.1016/S2213-2600(18)30103-6
4. Singh D, Agusti A, Anzueto A, et al. Global strategy for the diagnosis, management, and prevention of chronic obstructive lung disease: the GOLD science committee report 2019. *Eur Respir J*. 2019;53(5):1900164. doi:10.1183/13993003.00164-2019
5. Mannino DM, Buist AS. Global burden of COPD: risk factors, prevalence, and future trends. *Lancet*. 2007;370(9589):765–773. doi:10.1016/S0140-6736(07)61380-4
6. Hanson C, Rutten E, Wouters EFM, et al. Influence of diet and obesity on COPD development and outcomes. *Int J Chron Obstruct Pulmon Dis*. 2014;9:723–733. doi:10.2147/COPD.S50111
7. Benz E, Wijnant SRA, Trajanoska K, et al. Sarcopenia, systemic immune-inflammation index and all-cause mortality in middle-aged and older people with COPD and asthma: a population-based study. *ERJ Open Res*. 2022;8(1):00628–2021. doi:10.1183/23120541.00628-2021
8. Hu B, Yang X-R, Xu Y, et al. Systemic immune-inflammation index predicts prognosis of patients after curative resection for hepatocellular carcinoma. *Clin Cancer Res*. 2014;20(23):6212–6222. doi:10.1158/1078-0432.CCR-14-0442
9. Chen J-H, Zhai E-T, Yuan Y-J, et al. Systemic immune-inflammation index for predicting prognosis of colorectal cancer. *World J Gastroenterol*. 2017;23(34):6261–6272. doi:10.3748/wjg.v23.i34.6261
10. Miao Y, Yan Q, Li S, et al. Neutrophil to lymphocyte ratio and platelet to lymphocyte ratio are predictive of chemotherapeutic response and prognosis in epithelial ovarian cancer patients treated with platinum-based chemotherapy. *Cancer Biomark*. 2016;17(1):33–40. doi:10.3233/CBM-160614
11. Yang YL, Wu C-H, Hsu P-F, et al. Systemic immune-inflammation index (SII) predicted clinical outcome in patients with coronary artery disease. *Eur J Clin Invest*. 2020;50(5):e13230. doi:10.1111/eci.13230
12. Satis S. New inflammatory marker associated with disease activity in rheumatoid arthritis: the systemic immune-inflammation index. *Curr Health Sci J*. 2021;47(4):553–557. doi:10.12865/CHSJ.47.04.11
13. Xie R, Xiao M, Li L, et al. Association between SII and hepatic steatosis and liver fibrosis: a population-based study. *Front Immunol*. 2022;13:925690. doi:10.3389/fimmu.2022.925690
14. Ye C, Yuan L, Wu K, et al. Association between systemic immune-inflammation index and chronic obstructive pulmonary disease: a population-based study. *BMC Pulm Med*. 2023;23(1):295. doi:10.1186/s12890-023-02583-5
15. Fu Y, Wang Y, Wang Y, et al. Biomarkers (NLR,PLR, SII) for frequent COPD exacerbations: diagnostic and clinical management implications in a retrospective study. *Int J Chron Obstruct Pulmon Dis*. 2025;20:987–998. doi:10.2147/COPD.S510118
16. Scoditti E, Massaro M, Garbarino S, et al. Role of diet in chronic obstructive pulmonary disease prevention and treatment. *Nutrients*. 2019;11(6):1357. doi:10.3390/nu11061357
17. Shaheen SO, Jameson KA, Syddall HE, et al. The relationship of dietary patterns with adult lung function and COPD. *Eur Respir J*. 2010;36(2):277–284. doi:10.1183/09031936.00114709
18. Kaluza J, Larsson SC, Orsini N, et al. Fruit and vegetable consumption and risk of COPD: a prospective cohort study of men. *Thorax*. 2017;72(6):500–509. doi:10.1136/thoraxjnl-2015-207851
19. Krebs-Smith SM, Pannucci TE, Subar AF, et al. Update of the healthy eating index: HEI-2015. *J Acad Nutr Diet*. 2018;118(9):1591–1602. doi:10.1016/j.jand.2018.05.021
20. DeSalvo KB, Olson R, Casavale KO. Dietary guidelines for Americans. *JAMA*. 2016;315(5):457–458. doi:10.1001/jama.2015.18396
21. Guo X, Warden BA, Paeratakul S, et al. Healthy eating index and obesity. *Eur J Clin Nutr*. 2004;58(12):1580–1586. doi:10.1038/sj.ejcn.1601989
22. Wu PY, Lin MY, Tsai PS. Alternate healthy eating index and risk of depression: a meta-analysis and systemematic review. *Nutr Neurosci*. 2020;23(2):101–109. doi:10.1080/1028415X.2018.1477424
23. Schwingshackl L, Bogensberger B, Hoffmann G. Diet quality as assessed by the healthy eating index, alternate healthy eating index, dietary approaches to stop hypertension score, and health outcomes: an updated systematic review and meta-analysis of cohort studies. *J Acad Nutr Diet*. 2018;118(1):74–100.e11. doi:10.1016/j.jand.2017.08.024
24. National health and nutrition examination survey. Centers for Disease Controland Prevention; 2023. Available from: https://www.cdc.gov/nchs/nhanes/index.htm. Accessed August 28, 2025.
25. NHANES survey methods and analytic guidelines. Available from: https://wwwn.cdc.gov/nchs/nhanes//analyticguidelines.aspx. Accessed February 24, 2022.
26. Gress TW, Mansoor K, Rayyan YM, et al. Relationship between dietary sodium and sugar intake: a cross-sectional study of the National Health and Nutrition Examination Survey 2001–2016. *J Clin Hypertens*. 2020;22(9):1694–1702. doi:10.1111/jch.13985
27. Liu Q, Kang Y, Yan J. Association between overall dietary quality and constipation in American adults: a cross-sectional study. *BMC Public Health*. 2022;22(1):1971. doi:10.1186/s12889-022-14360-w
28. USDHH (2015) 2015–2020 Dietary Guidelines for Americans; 2022. Available from: https://odphp.health.gov/our-work/nutrition-physical-activity/dietary-guidelines/previous-dietary-guidelines/2015. Accessed August 28, 2025.
29. Healthy eating index SAS code; 2024. Available from: https://epi.grants.cancer.gov/hei/sas-code.html. Accessed August 28, 2025.
30. Ford ES, Bergmann MM, Boeing H, et al. Healthy lifestyle behaviors and all-cause mortality among adults in the United States. *Prev Med*. 2012;55(1):23–27. doi:10.1016/j.ypmed.2012.04.016
31. Schnell K, Weiss CO, Lee T, et al. The prevalence of clinically-relevant comorbid conditions in patients with physician-diagnosed COPD: a cross-sectional study using data from NHANES 1999–2008. *BMC Pulm Med*. 2012;12:26. doi:10.1186/1471-2466-12-26
32. Wang X, Wen J, Gu S, et al. Frailty in asthma-COPD overlap: a cross-sectional study of association and risk factors in the NHANES database. *BMJ Open Respir Res*. 2023;10(1).

33. American Diabetes Association. Classification and diagnosis of diabetes: standards of medical care in diabetes-2021. *Diabetes Care*. 2021;44(Suppl 1):S15–s33. doi:10.2337/dc21-S002
34. Centers for Disease Control and Prevention; National Center for Health Statistics. NHIS-adult tobacco use-glossary; 2022. Available from: https://www.cdc.gov/nchs/nhis/tobacco/tobacco_glossary. Accessed August 28, 2025.
35. Xu J, Zeng Q, Li S, et al. Inflammation mechanism and research progress of COPD. *Front Immunol*. 2024;15:1404615. doi:10.3389/fimmu.2024.1404615
36. van Iersel LEJ, Beijers RJHCG, Gosker HR, et al. Nutrition as a modifiable factor in the onset and progression of pulmonary function impairment in COPD: a systematic review. *Nutr Rev*. 2022;80(6):1434–1444. doi:10.1093/nutrit/nuab077
37. Barnes PJ. The cytokine network in asthma and chronic obstructive pulmonary disease. *J Clin Invest*. 2008;118(11):3546–3556. doi:10.1172/JCI36130
38. Cosio MG, Saetta M, Agusti A. Immunologic aspects of chronic obstructive pulmonary disease. *N Engl J Med*. 2009;360(23):2445–2454. doi:10.1056/NEJMra0804752
39. Barnes PJ. Inflammatory endotypes in COPD. *Allergy*. 2019;74(7):1249–1256. doi:10.1111/all.13760
40. Vaitkus M, Lavinskiene S, Barkauskiene D, et al. Reactive oxygen species in peripheral blood and sputum neutrophils during bacterial and nonbacterial acute exacerbation of chronic obstructive pulmonary disease. *Inflammation*. 2013;36(6):1485–1493. doi:10.1007/s10753-013-9690-3
41. Ge L, Wang N, Chen Z, et al. Expression of Siglec-9 in peripheral blood neutrophils was increased and associated with disease severity in patients with AECOPD. *Cytokine*. 2024;177:156558. doi:10.1016/j.cyto.2024.156558
42. Kumar P, Law S, Sriram KB. Evaluation of platelet lymphocyte ratio and 90-day mortality in patients with acute exacerbation of chronic obstructive pulmonary disease. *J Thorac Dis*. 2017;9(6):1509–1516. doi:10.21037/jtd.2017.05.77
43. Rahimirad S, Ghaffary MR, Rahimirad MH, et al. Association between admission neutrophil to lymphocyte ratio and outcomes in patients with acute exacerbation of chronic obstructive pulmonary disease. *Tuberk Toraks*. 2017;65(1):25–31. doi:10.5578/tt.27626
44. Wang YB, Page AJ, Gill TK, et al. The association between diet quality, plant-based diets, systemic inflammation, and mortality risk: findings from NHANES. *Eur J Nutr*. 2023;62(7):2723–2737. doi:10.1007/s00394-023-03191-z
45. Varraso R, Chiuve SE, Fung TT, et al. Alternate healthy eating index 2010 and risk of chronic obstructive pulmonary disease among US women and men: prospective study. *BMJ*. 2015;350:h286. doi:10.1136/bmj.h286
46. Neelakantan N, Koh W-P, Yuan J-M, et al. Diet-quality indexes are associated with a lower risk of cardiovascular, respiratory, and all-cause mortality among Chinese adults. *J Nutr*. 2018;148(8):1323–1332. doi:10.1093/jn/nxy094
47. Wen J, Gu S, Wang X, et al. Associations of adherence to the DASH diet and the Mediterranean diet with chronic obstructive pulmonary disease among US adults. *Front Nutr*. 2023;10:1031071. doi:10.3389/fnut.2023.1031071
48. Malmir H, Onvani S, Ardestani ME, et al. Adherence to low carbohydrate diet in relation to chronic obstructive pulmonary disease. *Front Nutr*. 2021;8:690880. doi:10.3389/fnut.2021.690880
49. Ghosn B, Onvani S, Ardestani ME, et al. The association between diet quality and chronic obstructive pulmonary disease: a case-control study. *BMC Public Health*. 2023;23(1):2071. doi:10.1186/s12889-023-16586-8
50. Ignacio Carlotto C, Bernardes S, Zanella P, et al. Dietary patterns and risk of Chronic Obstructive Pulmonary Disease (COPD) and clinical outcomes in diagnosed patients: a scoping review. *Respir Med*. 2024;233:107773. doi:10.1016/j.rmed.2024.107773
51. Çavuşoğlu Türker B, Ahbab S, Türker F, et al. Systemic immune-inflammation and systemic inflammation response indices are predictive markers of mortality in inpatients internal medicine services. *Int J Gen Med*. 2023;16:3163–3170. doi:10.2147/IJGM.S420332
52. Liu X, Ge H, Feng X, et al. The combination of hemogram indexes to predict exacerbation in stable chronic obstructive pulmonary disease. *Front Med Lausanne*. 2020;7:572435. doi:10.3389/fmed.2020.572435

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