Original Article

Dietary Inflammatory Index and all-cause mortality among asthma patients: a population-based cohort study

Yamei Yuan^, Weidong Ye^, Xiangming Fang^

College of Traditional Chinese Medicine, Anhui University of Chinese Medicine, Hefei, China

Contributions: (I) Conception and design: Y Yuan, X Fang; (II) Administrative support: X Fang; (III) Provision of study materials or patients: Y Yuan, W Ye; (IV) Collection and assembly of data: Y Yuan; (V) Data analysis and interpretation: Y Yuan; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

Correspondence to: Xiangming Fang. College of Traditional Chinese Medicine, Anhui University of Chinese Medicine, 103 Meishan Road, Hefei, China. Email: fxmactm@sina.com.

Background: Inflammatory response plays a crucial role in the development and deterioration of asthma. The aim of this study was to assess the association between Dietary Inflammatory Index (DII) score and all-cause mortality among patients with asthma in the United States (US).

Methods: We included participants aged ≥20 years and excluded those with no data relating to 24-hour dietary recall of dietary intake interview and asthma status. DII score was calculated based 25 food parameters. Multivariate Cox regression analysis was used to analyze the linear association between DII score and all-cause mortality, and a generalized additive model (GAM) with a spline smoothing function was used to analyze the nonlinear dose-response relationship.

Results: Among the 20,573 participants, 2,805 (13.63%) participants had a current or past diagnosis of asthma, and 17,768 (86.37%) participants had no history of asthma. DII score was associated with increased all-cause mortality among participants who had previously been diagnosed with asthma [hazard ratio (HR): 1.13; 95% confidence interval (CI): 1.04, 1.23; P=0.004] and participants who were currently diagnosed with asthma (HR: 1.13; 95% CI: 1.03, 1.24; P=0.010) after multivariable adjustment for age, sex, race, education, family income, body mass index, physical activity, smoking status, diabetes, hypertension, and coronary heart disease. When DII score was transformed into a categorical variable, participants with a current or past diagnosis of asthma who were in the DII score fifth quintile were associated with increased all-cause mortality when compared with participants in the lowest quintile (HR: 2.34; 95% CI: 1.34, 4.11). The association was stronger in these participants than in participants with no history of asthma (HR: 1.25; 95% CI: 1.04, 1.49). Furthermore, the GAM model showed a linear dose-response relationship, which indicated that increased DII score was associated with increased all-cause mortality among participants with a current or past diagnosis of asthma.

Conclusions: Our study is the first to provide evidence that DII score is associated with increased all-cause mortality among patients with asthma in the US. Due to potential bias and residual confounding, our findings require further investigation and confirmation in randomized controlled trials.

Keywords: Dietary Inflammatory Index (DII); asthma; all-cause mortality; National Health and Nutrition Examination Survey (NHANES)

Submitted Apr 29, 2022. Accepted for publication Jun 09, 2022.
doi: 10.21037/apm-22-615
View this article at: https://dx.doi.org/10.21037/apm-22-615

^ ORCID: Yamei Yuan, 0000-0003-0943-9821; Weidong Ye, 0000-0001-5483-7998; Xiangming Fang, 0000-0001-6752-7438.

Introduction

Asthma is a common chronic inflammatory disease of the airways that causes coughing, wheezing, shortness of breath, and chest tightness, leading to substantial effects on healthcare utilization, quality of life, and mortality (1). Asthma generally affects children and young people, although it also affects a significant proportion of the middle-aged and older population (2,3). From 1982 to 2018, asthma prevalence doubled from less than 4% to 7.7% in the United States (US) (4,5). Because asthma can be induced and aggravated by both allergic and nonallergic factors, asthma prevalence is partly attributable to changes in the environment and lifestyle, including diet (6,7). Furthermore, previous studies have shown that smoking, low physical activity, air pollution, psychosocial distress, and comorbidity are independent risk factor for mortality among patients with asthma (8-12).

Previous studies have reported that a proinflammatory diet may negatively affect lung function (13) and increase allergic airway inflammation by influencing innate and adaptive immune responses (14). By contrast, a Mediterranean diet characterized by a high intake of fruits, vegetables, and n-3 polyunsaturated fatty acids is associated with lower asthma risk (15). Given that the inflammatory response plays a crucial role in the development and deterioration of asthma, the consumption of proinflammatory foods may worsen the disease. The Dietary Inflammatory Index (DII) is a scoring system for evaluating the inflammatory potential of dietary components, and a higher score indicates a pro-inflammatory diet (16). It is well-known that the combined and interacting effects of dietary components may have greater effects on disease pathogenesis than individual nutrients. A cross-sectional study used the DII to demonstrate that a proinflammatory diet increased the risk of allergic wheezing in adults and children (17). Furthermore, a prior study including 99 patients with asthma and 60 healthy controls demonstrated that DII score was associated with increased systemic inflammation and decreased pulmonary function (18). However, to the best of our knowledge, no study has investigated the association between DII score and mortality among patients with asthma.

The present study aimed to assess the association between DII score and all-cause mortality among patients with asthma and participants with no history of asthma by analyzing data from the National Health and Nutrition Examination Survey (NHANES) database. We present the following article in accordance with the STROBE reporting checklist (available at https://apm.amegroups.com/article/view/10.21037/apm-22-615/rc).

Methods

Study population

We conducted a population-based cohort study to assess the association between DII score and all-cause mortality among patients with asthma. The data used in this study were pooled from 5 independent cycles (2005–2006 to 2013–2014) of the NHANES, a survey designed to assess the health and nutritional status of the US civilian noninstitutional population. A stratified, multistage probability design was used to select a representative sample. The NHANES interview includes s a wide range of topics including demographics, socioeconomic, medical histories, dietary, behavioral, and lifestyle factors. Detailed methodology, protocols, and definitions of NHANES can be found at https://www.cdc.gov/nchs/nhanes.htm. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013).

In the present study, we included patients aged ≥20 years and excluded those with no data relating to personal interviews or 24-hour dietary recall of dietary intake interviews. Furthermore, to be included in our study, participants had to provide complete information on asthma status. Finally, a total of 20,573 participants were included in this study.

Definition of asthma

The presence of asthma was defined as an affirmative response to the following questions: "Has a doctor or health professional ever told you that you have asthma?" and "Do you still have asthma?". The participants were divided into the following three groups based on their answers to these two questions: (I) patients with no history of asthma; (II) patients who had previously been diagnosed with asthma but no longer had asthma; and (III) patients who currently had asthma. The detailed description of the NHANES definition of asthma has been published elsewhere (19).

Assessment of DII

The development and validation of the DII has been described in previous publications (20). The calculation

of DII score was based on dietary intake data gathered by 24-hour dietary recall interviews including the types and amounts of foods and beverages consumed by participants. The intake of energy, nutrients, and other components of these foods and beverages was further estimated. In the present study, 25 food parameters were used to calculate the DII score, including energy, carbohydrates, total fat, protein, fiber, cholesterol, saturated fatty acids, monounsaturated fatty acids, polyunsaturated fatty acids, n-3 fatty acids, n-6 fatty acids, vitamin A, beta carotene, vitamin B6, vitamin B12, vitamin C, vitamin E, folic acid, alcohol, caffeine, iron, magnesium, zinc, selenium, and niacin. The z-score was calculated by subtracting the standard global mean (derived from a global database) from the individual intake of each food parameter and dividing the value by its standard deviation. The z-scores were converted to a percentile score, and symmetrical distribution was achieved by doubling each percentile score and subtracting "1". The derived value was multiplied by its respective overall food parameter-specific inflammatory effect score and summed to obtain the overall DII score.

Mortality data

The outcome for the present study was all-cause death that occurred from the date of survey participation through to December 31, 2015. Mortality information was determined by using a probability match between the NHANES and death certificate records from the National Death Index (NDI). Other sources of follow-up for mortality included the US Social Security Administration, the Centers for Medicare and Medicaid Services, and death certificates.

Covariates

The demographics questionnaires, including age, sex, ethnicity, educational status, family income per capita were asked, in the home, by trained interviewers using Computer-Assisted Personal Interview (CAPI) system. Body mass index was calculated as the weight (kg) divided by the square of the height (m^2). Current smoking was defined as smoking at least 100 cigarettes in life. For leisure-time physical activity, participants were asked "In a typical week, on how many days do the following: walking or bicycling for transportation, vigorous leisure-time physical activity, or moderate leisure-time physical activity?". Participants could select more than one activity and were asked to quantify their participation by frequency and duration. The intensity was expressed in terms of standardized metabolic equivalent of task (MET) values: 4.0 METs for walking or bicycling for transportation; 8.0 METs for vigorous leisure-time physical activity; 4.0 METs moderate leisure-time physical activity. The medical conditions section from the questionnaire data provides self-reported personal interview information on the diagnosed health status, including diabetes, coronary heart disease, and hypertension. The medical conditions were based on their answers to the question, "Has a doctor or other health professional ever told you that you had diabetes/coronary heart disease/hypertension?".

Statistical analysis

Our data analysis used recommended weighting methodology due to NHANES using a complex, multistage, and clustered probability design to select participants who represented the US civilian noninstitutional census population. Baseline characteristics were presented as weighted means and standard error (SE) for continuous variables and weighted percentages and SE for categorical variables. Univariate Cox regression analysis was used to identify the association between age, sex, ethnicity, educational status, family income per capita, body mass index, smoking status, leisure time physical activity, diabetes, coronary heart disease, hypertension, DII score, and all-cause mortality among participants with asthma. Multivariate Cox regression analysis was used to analyze the association between DII score and all-cause mortality among participants with asthma, which was adjusted for confounders on the basis of a change in effect estimate of more than 10% in the univariate analysis (21). Furthermore, we used a generalized additive model (GAM) with a spline smoothing function to identify the nonlinear dose-response relationship between DII score and all-cause mortality in participants with asthma. All statistical analyses were performed using R (https://www.r-project.org/; The R Foundation for Statistical Computing, Vienna, Austria). A two-sided P values less than 0.05 was considered statistically significant.

Results

Table 1 shows the weighted characteristics of the study participants stratified by self-reported asthma status. Among the 20,573 participants aged ≥20 years, 2,805 (13.63%) participants had been diagnosed with asthma in the past, and 1,655 (8.04%) participants reported current asthma. In

Table 1 Weighted characteristics of participants stratified by self-reported asthma status, NHANES [2005–2014]

Characteristics	Total population	No history of asthma	Prior history of asthma	Current asthma
Unweighted, n	20,573	17,768	1,150	1,655
Age (years), mean ± SE	46.88±0.27	47.27±0.28	41.37±0.55	46.90±0.60
Sex, %				
Male	48.30	49.43	49.47	35.46
Female	51.70	50.57	50.53	64.54
Ethnicity, %				
Non-Hispanic white	69.81	69.39	71.08	73.37
Black	10.57	10.32	11.32	12.58
Other	19.62	20.29	17.61	14.05
Educational status, %				
Under high school	17.16	17.49	13.64	16.28
High school	22.77	23.10	19.00	22.16
Above high school	60.07	59.41	67.36	61.56
Family income, %				
Under $20,000	16.76	16.09	17.37	23.35
$20,000 and $55,000	35.72	35.85	34.71	35.14
$55,000 and over	47.52	48.06	47.92	41.51
Body mass index (kg/m^2), mean ± SE	28.73±0.09	28.55±0.09	28.73±0.25	0.28
Smoking status, %				
No	54.35	54.92	51.56	50.36
Yes	45.65	45.08	48.44	49.64
Leisure time physical activity (MET/week), %				
<500	57.10	56.92	53.38	61.81
500–999	13.54	13.88	12.77	10.57
≥1,000	29.35	29.20	33.85	27.62
Diabetes, %	8.71	8.48	7.69	11.87
Hypertension, %	30.77	30.32	25.33	39.58
Coronary heart disease, %	3.22	3.14	2.48	4.66

NHANES, National Health and Nutrition Examination Survey; SE, standard error; MET, metabolic equivalent of task.

the entire sample, the mean age was 46.88 years, and 48.3% of the participants were male. At the mean follow-up of 5.66 years, 1,579 patients had died. Of these deaths, 157 were participants who had reported baseline asthma.

Table 2 summarizes the results of the univariate analysis of the association between all potential confounding variables, including demographic, socio-economic, and other risk factor variables, with all-cause mortality among participants stratified by self-reported asthma status. The results showed that age, obesity (body mass index ≥25 kg/m^2), smoking, diabetes, coronary heart disease, hypertension, and DII score were positively associated with all-cause mortality

Table 2 Univariate Cox regression of the relationship between demographic, socio-economic, and other risk factor variables and all-cause mortality among participants stratified by self-reported asthma status, NHANES [2005–2014]

Variables	No history of asthma		Prior history of asthma		Current asthma	
	HR (95% CI)	P value	HR (95% CI)	P value	HR (95% CI)	P value
Age (years)	1.08 (1.08, 1.09)	<0.001	1.08 (1.07, 1.09)	<0.001	1.08 (1.07, 1.09)	<0.001
Sex						
Male	Reference		Reference		Reference	
Female	0.66 (0.60, 0.74)	<0.001	0.62 (0.47, 0.81)	<0.001	0.52 (0.38, 0.72)	<0.001
Ethnicity						
Non-Hispanic white	Reference		Reference		Reference	
Black	0.76 (0.66, 0.88)	<0.001	0.81 (0.57, 1.14)	0.219	0.82 (0.56, 1.20)	0.300
Other	0.45 (0.40, 0.52)	<0.001	0.56 (0.38, 0.83)	0.004	0.57 (0.36, 0.90)	0.015
Educational status						
Under high school	Reference		Reference		Reference	
High school	0.72 (0.63, 0.82)	<0.001	0.83 (0.57, 1.19)	0.310	0.89 (0.59, 1.36)	0.590
Above high school	0.48 (0.43, 0.55)	<0.001	0.57 (0.41, 0.78)	<0.001	0.69 (0.48, 1.00)	0.049
Family income						
Under $20,000	Reference		Reference		Reference	
$20,000 and $55,000	0.68 (0.61, 0.77)	<0.001	0.84 (0.61, 1.15)	0.279	0.88 (0.61, 1.25)	0.468
$55,000 and over	0.34 (0.29, 0.40)	<0.001	0.43 (0.29, 0.63)	<0.001	0.50 (0.32, 0.77)	0.002
Body mass index (kg/m^2)						
<18.5	1.33 (0.91, 1.96)	0.142	2.66 (1.13, 6.26)	0.025	2.46 (0.95, 6.34)	0.063
18.5–24.9	Reference		Reference		Reference	
25–29.9	1.00 (0.88, 1.14)	0.996	1.21 (0.82, 1.80)	0.335	1.10 (0.69, 1.77)	0.681
≥30	0.84 (0.73, 0.96)	0.013	1.11 (0.76, 1.60)	0.592	1.04 (0.68, 1.59)	0.868
Smoking status						
No	Reference		Reference		Reference	
Yes	1.70 (1.52, 1.89)	<0.001	2.32 (1.72, 3.14)	<0.001	2.23 (1.58, 3.13)	<0.001
Leisure time physical activity (MET/week)						
<500	Reference		Reference		Reference	
500–999	0.58 (0.48, 0.71)	<0.001	0.74 (0.44, 1.24)	0.251	0.79 (0.43, 1.44)	0.443
≥1,000	0.39 (0.32, 0.46)	<0.001	0.30 (0.18, 0.49)	<0.001	0.36 (0.21, 0.64)	<0.001
Diabetes						
No	Reference		Reference		Reference	
Yes	3.12 (2.77, 3.52)	<0.001	2.25 (1.64, 3.08)	<0.001	1.82 (1.27, 2.61)	0.001

Table 2 (*continued*)

Table 2 (*continued*)

Variables	No history of asthma		Prior history of asthma		Current asthma	
	HR (95% CI)	P value	HR (95% CI)	P value	HR (95% CI)	P value
Hypertension						
No	Reference		Reference		Reference	
Yes	2.94 (2.64, 3.27)	<0.001	4.12 (3.04, 5.59)	<0.001	3.44 (2.42, 4.90)	<0.001
Coronary heart disease						
No	Reference		Reference		Reference	
Yes	4.20 (3.59, 4.92)	<0.001	5.38 (3.72, 7.78)	<0.001	3.57 (2.29, 5.56)	<0.001
DII						
Quintile 1	Reference		Reference		Reference	
Quintile 2	1.13 (0.93, 1.36)	0.220	1.81 (1.04, 3.15)	0.035	1.43 (0.78, 2.64)	0.248
Quintile 3	1.41 (1.18, 1.69)	<0.001	2.48 (1.46, 4.20)	<0.001	2.26 (1.28, 4.01)	0.005
Quintile 4	1.41 (1.18, 1.69)	<0.001	2.45 (1.45, 4.16)	<0.001	2.05 (1.16, 3.63)	0.014
Quintile 5	1.66 (1.40, 1.97)	<0.001	2.31 (1.35, 3.93)	0.002	2.08 (1.18, 3.69)	0.012

NHANES, National Health and Nutrition Examination Survey; MET, metabolic equivalent of task; DII, Dietary Inflammatory Index; HR, hazard ratio; CI, confidence interval.

among participants with no history of asthma, participants who had previously been diagnosed with asthma, and patients who had a current diagnosis of asthma. In contrast, female, other ethnic groups, education level more than high school, higher family income per capita, and leisure time physical activity were negatively associated with all-cause mortality.

Table 3 shows the multivariable adjusted association between DII score and all-cause mortality among participants stratified by self-reported asthma status. After adjustment for potential covariates, DII score was associated with increased all-cause mortality among participants with no history of asthma [hazard ratio (HR): 1.05; 95% confidence interval (CI): 1.01, 1.08; P=0.004], those who had previously been diagnosed with asthma (HR: 1.13; 95% CI: 1.04, 1.23; P=0.004), and those who had a current diagnosis of asthma (HR: 1.13; 95% CI: 1.03, 1.24; P=0.010). When DII score was transformed into a categorical variable, the HRs (95% CIs) for participants with a current diagnosis of asthma in the higher quintiles (Q2, Q3, Q4, and Q5) were 1.62 (0.86, 3.05), 2.38 (1.33, 4.29), 2.01 (1.11, 3.66), and 2.31 (1.27, 4.23), respectively. The association between DII score and all-cause mortality was stronger for participants who had a current or past diagnosis of asthma than for participants with no history of asthma. The results of the GAM model with a smoothing curve showed a linear dose-response relationship, which indicated that increased DII score was associated with increased all-cause mortality among participants who had a current or past diagnosis of asthma (*Figure 1*).

Discussion

Asthma prevalence has increased significantly in both developed and developing countries over the past few decades (22). According to the Global Burden of Disease statistics, 0.40 million people died of asthma in 2015, and asthma prevalence increased by 12.6% (9.0% to 16.4%) from 1990 to 2015 (23). To the best of our knowledge, this was the first study to explore the association between DII score and all-cause mortality among patients with asthma and participants with no history of asthma. Our results showed that DII score was positively associated with all-cause mortality among both participants with no history of asthma and participants with asthma. Among participants who had a current or past diagnosis of asthma, those in the highest DII score quintile had a more than 2.3-fold increase in all-cause mortality risk. Most importantly, the association was stronger for participants who had a current or past diagnosis of asthma than for those with no history

Table 3 HRs (95% CI) for the multivariate-adjusted association between DII score and all-cause mortality among participants stratified by self-reported asthma status, NHANES [2005–2014]

Variables	No history of asthma		Prior history of asthma		Current asthma	
	HR (95% CI)	P value	HR (95% CI)	P value	HR (95% CI)	P value
DII (continuous)	1.05 (1.01, 1.08)	0.004	1.13 (1.04, 1.23)	0.004	1.13 (1.03, 1.24)	0.010
DII (quintile)						
Quintile 1	Reference		Reference		Reference	
Quintile 2	1.02 (0.84, 1.23)	0.870	2.03 (1.16, 3.57)	0.014	1.62 (0.86, 3.05)	0.132
Quintile 3	1.17 (0.98, 1.41)	0.087	2.51 (1.47, 4.30)	<0.001	2.38 (1.33, 4.29)	0.004
Quintile 4	1.09 (0.91, 1.31)	0.366	2.12 (1.23, 3.66)	0.007	2.01 (1.11, 3.66)	0.021
Quintile 5	1.25 (1.04, 1.49)	0.017	2.34 (1.34, 4.11)	0.003	2.31 (1.27, 4.23)	0.006
P for trend	0.013	–	0.002	–	0.011	–

Adjust for: age, sex, race, education, family income, body mass index, physical activity, smoking status, diabetes, hypertension, and coronary heart disease. HR, hazard ratio; CI, confidence interval; DII, Dietary Inflammatory Index; NHANES, National Health and Nutrition Examination Survey.

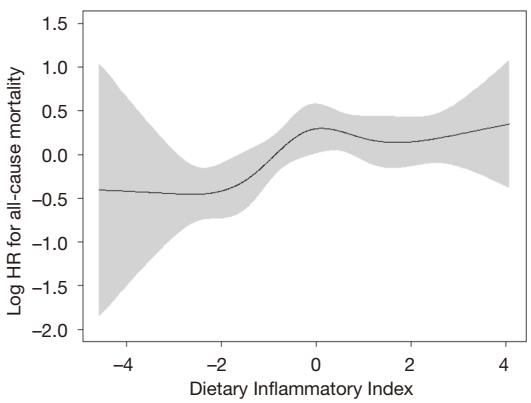

Figure 1 The dose-response relationship between DII score and mortality among participants with a current or past diagnosis of asthma. HR, hazard ratio; DII, Dietary Inflammatory Index.

of asthma. Furthermore, the GAM model suggested a linear increasing trend, with the risk of all-cause mortality increasing as the DII score increased.

Certain factors, such as environmental factors, genetic factors, and nutritional habits (relating to specific nutrients in the diet) contribute to the development and progression of asthma. A diet rich in fruits, vegetables, and anti-inflammatory components, such as the Mediterranean diet, can prevent asthma (24). By contrast, processed foods with a high fat content exacerbate asthma symptoms (25). Two earlier studies have demonstrated that a diet rich in sodium may exacerbate asthma and increase mortality in men with asthma (26,27). Furthermore, studies have demonstrated that the intake of anti-inflammatory nutrients, especially those rich in vitamins A, C, and E, reduces the inflammatory response and improves the clinical outcomes of asthma and lung function (28-30). In recent years, studies assessing the association between diet and chronic diseases have mainly focused on diet pattern and diet score, as the combined effect (synergistic or antagonistic) of dietary components may have a greater effect on disease pathogenesis than individual components. Recently, the DII was developed to evaluate the inflammatory potential of a diet (16,31). The DII score is not limited to micronutrients and macronutrients but also includes common daily dietary components, such as alcohol and caffeine.

Epidemiologic evidence for an association between DII score and asthma progression is scarce. In this study, participants who had a current or past diagnosis of asthma and were in the highest DII score quintile had a more than 2.3-fold increase in all-cause mortality risk, and this increase was significantly higher than that of participants with no history of asthma. This finding suggests that dietary intervention as a part of asthma treatment may improve the prognosis of patients with asthma. A past cross-sectional study demonstrated that an increase in the inflammatory potential of diet among asthmatics decreased pulmonary functions and asthma control (32). Another cross-sectional study demonstrated that a proinflammatory diet assessed using the DII increased the risk of allergic wheezing in adults and children (17). Interestingly, several recent

prospective longitudinal studies reported that a diet with a higher DII score during pregnancy was associated with a higher risk of offspring asthma (33,34).

Asthma symptoms are usually caused by airway inflammation, which can trigger mucus production, airway wall remodeling, and bronchial hyperresponsiveness (1). Previous studies have demonstrated that increased levels of inflammatory cytokines, such as tumor necrosis factor alpha (TNF-α), C-reactive protein (CRP), and interleukin-6 (IL-6), are associated with decreased asthma control (35,36). Because inflammation is crucial in asthma pathogenesis, the primary goal of asthma treatment is to control symptoms and inflammation to prevent exacerbation. Among the food parameters included in the DII score in our study, saturated fatty acids and cholesterol were the main proinflammatory factors; in contrast, n-3 and n-6 fatty acids have anti-inflammatory properties (16). Dietary fiber, beta carotene, and vitamins A, C, and E that are mainly derived from fruits and vegetables also have anti-inflammatory effects. Fruits and vegetables may protect against asthma by reducing the T helper 2 (Th2) immune response, airway inflammation, and oxidative stress (37). Furthermore, dietary selenium is incorporated into selenoprotein and is involved in regulating excessive immune responses and chronic inflammation. Selenium deficiency has long been considered to have a negative effect on the activation, differentiation, and proliferation of immune cells (38,39). A previous study demonstrated that zinc, a nutritionally fundamental trace element, decreases nuclear factor kappa B (NF-κB) activation, resulting in the decreased expression of its target genes, such as TNF-α and interleukin-1 beta (IL-1β); however, it also increases the expression of A20 and peroxisome proliferator-activated receptor alpha (PPAR-α), the 2 zinc finger proteins with anti-inflammatory properties (40).

The present study had several strengths. First, we used a large sample representative of the US population, and standardized procedures were performed by uniformly trained personnel. Second, to evaluate the role of DII score in the prognosis of patients with asthma, we examined the relationship between DII score and all-cause mortality among participants who had a current or past diagnosis of asthma and participants with no history of asthma. Third, we strictly adjusted for multiple covariates and used a GAM with a spline smoothing function to identify the dose-response relationship between DII score and all-cause mortality among participants with asthma. Our study had several limitations. First, asthma status was obtained using a questionnaire-based survey; therefore, the conclusions may be biased. Second, although our analyses adjusted for multiple potential confounders, residual confounding such as drug therapy cannot be excluded. Third, we did not assess the association between DII score and other subtypes of mortality such as injury, infectious disease, and chronic illness due to the limited number of deaths.

In summary, this study provides the first evidence of an association between DII score and increased all-cause mortality among patients with asthma in the US. Dietary intervention as a part of asthma treatment may improve the prognosis of patients with asthma. Due to potential bias and residual confounding, our findings require further investigation and confirmation in randomized controlled trials.

Acknowledgments

We express our appreciation to the staff from the National Health and Nutrition Examination Survey (NHANES).
Funding: This study was sponsored by the University Excellent Young Talent Support Project in Anhui (Grant No. gxyq2021186).

Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://apm.amegroups.com/article/view/10.21037/apm-22-615/rc

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://apm.amegroups.com/article/view/10.21037/apm-22-615/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013).

References

1. Hammad H, Lambrecht BN. The basic immunology of asthma. Cell 2021;184:1469-85.
2. Busse PJ, Kilaru K. Complexities of diagnosis and treatment of allergic respiratory disease in the elderly. Drugs Aging 2009;26:1-22.
3. Patel PO, Patel MR, Baptist AP. Depression and Asthma Outcomes in Older Adults: Results from the National Health and Nutrition Examination Survey. J Allergy Clin Immunol Pract 2017;5:1691-7.e1.
4. Moorman JE, Akinbami LJ, Bailey CM, et al. National surveillance of asthma: United States, 2001-2010. Vital Health Stat 3 2012;(35):1-58.
5. Centers for Disease Control and Prevention. Most Recent National Asthma Data. Available online: https://www.cdc.gov/asthma/most_recent_national_asthma_data.htm (Accessed on September 15, 2020).
6. Beasley R, Semprini A, Mitchell EA. Risk factors for asthma: is prevention possible? Lancet 2015;386:1075-85.
7. Han YY, Forno E, Holguin F, et al. Diet and asthma: an update. Curr Opin Allergy Clin Immunol 2015;15:369-74.
8. Liu Y, Pan J, Zhang H, et al. Short-Term Exposure to Ambient Air Pollution and Asthma Mortality. Am J Respir Crit Care Med 2019;200:24-32.
9. Bellia V, Pedone C, Catalano F, et al. Asthma in the elderly: mortality rate and associated risk factors for mortality. Chest 2007;132:1175-82.
10. Razvodovsky YE. Psychosocial distress as a risk factor of asthma mortality. Psychiatr Danub 2010;22:167-72.
11. Sumino K, O'Brian K, Bartle B, et al. Coexisting chronic conditions associated with mortality and morbidity in adult patients with asthma. J Asthma 2014;51:306-14.
12. Tupper OD, Andersen ZJ, Ulrik CS. Demographic, lifestyle and comorbid risk factors for all-cause mortality in a Danish cohort of middle-aged adults with incident asthma. BMJ Open 2021;11:e049243.
13. Han YY, Jerschow E, Forno E, et al. Dietary Patterns, Asthma, and Lung Function in the Hispanic Community Health Study/Study of Latinos. Ann Am Thorac Soc 2020;17:293-301.
14. Julia V, Macia L, Dombrowicz D. The impact of diet on asthma and allergic diseases. Nat Rev Immunol 2015;15:308-22.
15. Thorburn AN, Macia L, Mackay CR. Diet, metabolites, and "western-lifestyle" inflammatory diseases. Immunity 2014;40:833-42.
16. Shivappa N, Steck SE, Hurley TG, et al. Designing and developing a literature-derived, population-based dietary inflammatory index. Public Health Nutr 2014;17:1689-96.
17. Han YY, Forno E, Shivappa N, et al. The Dietary Inflammatory Index and Current Wheeze Among Children and Adults in the United States. J Allergy Clin Immunol Pract 2018;6:834-41.e2.
18. Wood LG, Shivappa N, Berthon BS, et al. Dietary inflammatory index is related to asthma risk, lung function and systemic inflammation in asthma. Clin Exp Allergy 2015;45:177-83.
19. Hong H, Hou W, Kaur S, et al. The association of co-morbid asthma and overweight/obese status with healthcare utilization and caregiver perception of health in children 4-17 years, a NHANES study. J Asthma 2022;59:1181-7.
20. Wang T, Jiang H, Wu Y, et al. The association between Dietary Inflammatory Index and disability in older adults. Clin Nutr 2021;40:2285-92.
21. Kernan WN, Viscoli CM, Brass LM, et al. Phenylpropanolamine and the risk of hemorrhagic stroke. N Engl J Med 2000;343:1826-32.
22. Odebeatu CC, Taylor T, Fleming LE, et al. Phthalates and asthma in children and adults: US NHANES 2007-2012. Environ Sci Pollut Res Int 2019;26:28256-69.
23. GBD 2015 Chronic Respiratory Disease Collaborators. Global, regional, and national deaths, prevalence, disability-adjusted life years, and years lived with disability for chronic obstructive pulmonary disease and asthma, 1990-2015: a systematic analysis for the Global Burden of Disease Study 2015. Lancet Respir Med 2017;5:691-706.
24. Del Chierico F, Vernocchi P, Dallapiccola B, et al. Mediterranean diet and health: food effects on gut microbiota and disease control. Int J Mol Sci 2014;15:11678-99.
25. Allan K, Devereux G. Diet and asthma: nutrition implications from prevention to treatment. J Am Diet Assoc 2011;111:258-68.
26. Burney P. A diet rich in sodium may potentiate asthma. Epidemiologic evidence for a new hypothesis. Chest 1987;91:143S-8S.
27. Burney PG, Neild JE, Twort CH, et al. Effect of changing dietary sodium on the airway response to histamine. Thorax 1989;44:36-41.
28. Wood LG, Garg ML, Smart JM, et al. Manipulating

antioxidant intake in asthma: a randomized controlled trial. Am J Clin Nutr 2012;96:534-43.
29. Li J, Li L, Chen H, et al. Application of vitamin E to antagonize SWCNTs-induced exacerbation of allergic asthma. Sci Rep 2014;4:4275.
30. McEvoy CT, Schilling D, Clay N, et al. Vitamin C supplementation for pregnant smoking women and pulmonary function in their newborn infants: a randomized clinical trial. JAMA 2014;311:2074-82.
31. Cavicchia PP, Steck SE, Hurley TG, et al. A new dietary inflammatory index predicts interval changes in serum high-sensitivity C-reactive protein. J Nutr 2009;139:2365-72.
32. Özbey Ü, Uçar A, Shivappa N, et al. The Relationship between Dietary Inflammatory Index, Pulmonary Functions and Asthma Control in Asthmatics. Iran J Allergy Asthma Immunol 2019;18:605-14.
33. Chen LW, Lyons B, Navarro P, et al. Maternal dietary inflammatory potential and quality are associated with offspring asthma risk over 10-year follow-up: the Lifeways Cross-Generation Cohort Study. Am J Clin Nutr 2020;111:440-7.
34. Venter C, Palumbo MP, Sauder KA, et al. Examining Associations Between Dietary Inflammatory Index in Pregnancy, Pro-inflammatory Cytokine and Chemokine Levels at Birth, and Offspring Asthma and/or Wheeze by Age 4 Years. J Acad Nutr Diet 2021;121:2003-12.e3.
35. Zhang X, Zheng J, Zhang L, et al. Systemic inflammation mediates the detrimental effects of obesity on asthma control. Allergy Asthma Proc 2018;39:43-50.
36. Monadi M, Firouzjahi A, Hosseini A, et al. Serum C-reactive protein in asthma and its ability in predicting asthma control, a case-control study. Caspian J Intern Med 2016;7:37-42.
37. Han YY, Blatter J, Brehm JM, et al. Diet and asthma: vitamins and methyl donors. Lancet Respir Med 2013;1:813-22.
38. Hariharan S, Dharmaraj S. Selenium and selenoproteins: it's role in regulation of inflammation. Inflammopharmacology 2020;28:667-95.
39. Huang Z, Rose AH, Hoffmann PR. The role of selenium in inflammation and immunity: from molecular mechanisms to therapeutic opportunities. Antioxid Redox Signal 2012;16:705-43.
40. Jarosz M, Olbert M, Wyszogrodzka G, et al. Antioxidant and anti-inflammatory effects of zinc. Zinc-dependent NF-κB signaling. Inflammopharmacology 2017;25:11-24.

(English Language Editor: C. Gourlay)

Cite this article as: Yuan Y, Ye W, Fang X. Dietary Inflammatory Index and all-cause mortality among asthma patients: a population-based cohort study. Ann Palliat Med 2022;11(6):2061-2070. doi: 10.21037/apm-22-615

