BMC Public Health

RESEARCH

Open Access

Associations of the planetary health diet index (PHDI) with asthma: the mediating role of body mass index

Shaoqun Huang[1†], Qiao He[2†], Xiaoxuan Wang[2], Seok Choi[3] and Hongyang Gong[3*]

Abstract

Background Given the global shifts in environmental conditions and dietary habits, understanding the potential impact of dietary factors and body mass index (BMI) on respiratory diseases, including asthma, is paramount. Investigating these relationships can contribute to the formulation of more effective prevention strategies. The Planetary Health Diet Index (PHDI), a dietary scoring system that balances human health with environmental sustainability, underscores the importance of increasing the consumption of plant-based foods while reducing the intake of red meat, sugar, and highly processed foods. The objective of this study was to assess the association between PHDI and the prevalence of asthma and the mediation effect of BMI in a US general population.

Methods This study utilized data from 32,388 participants in the National Health and Nutrition Examination Survey (NHANES) spanning from 2005 to 2018. Multivariate logistic regression and weighted quantile sum (WQS) regressions were employed to investigate the association between PHDI, individual nutrients, and asthma. Restricted cubic spline (RCS) analysis explored the linear or non-linear relationship between PHDI and asthma. Interaction analyses were conducted on subgroups to validate the findings. Mediation analysis was performed to examine the effect of BMI on the relationship between PHDI and asthma.

Results There was a significant negative association between PHDI and asthma. After adjusting for covariates, for every 10-point increase in PHDI, there was a 4% decrease in the prevalence of asthma ($P = 0.025$). Moreover, as PHDI increased, there was a trend towards lower asthma prevalence (P for trend < 0.05). WQS analyses showed consistent associations (OR = 0.93, 95%CI: 0.88, 0.98), with Fiber, Vitamin C, and Protein significant factors. The dose-response curve indicated a linear association between PHDI and asthma, with higher PHDI associated with lower asthma prevalence. Additionally, BMI is significantly positively associated with asthma ($P < 0.001$), and BMI decreases as the PHDI increases ($\beta = -0.64$, $P < 0.001$). Mediation analysis indicates that BMI significantly mediates the relationship between PHDI and asthma, with a mediation proportion of 33.85% ($P < 0.001$).

Conclusion The results of this study show a strong negative correlation between PHDI and the prevalence of asthma. In addition, BMI mediated this negative relationship.

†Shaoqun Huang, Qiao He contributed equally to this work.

*Correspondence:
Hongyang Gong
hygong@chosun.ac.kr

Full list of author information is available at the end of the article

Keywords Asthma, Planetary Health Diet Index, NHANES, Mediation analysis, Body mass index

Introduction

Asthma is a chronic pulmonary disease and a global health issue affecting individuals of all ages. It is prevalent among both children and adults and is characterized by airway inflammation and muscle tightening, leading to breathing difficulties [1]. Approximately 350 million people worldwide suffer from asthma [2], with a prevalence rate of 4.3%. The global crude prevalence is increasing, resulting in 455,000 deaths [3]. The Global Initiative for Asthma (GINA) predicts that the number of individuals with asthma will reach 400 million by 2025. Prevalence rates vary significantly across countries, with the highest rates observed in developed countries, likely due to aging populations and increased life expectancy [4]. Asthma is a major cause of severe disability, reduced quality of life, and poor utilization of healthcare resources [5]. It is a significant contributor to the global burden of non-communicable diseases and is a leading cause of death, second only to cardiovascular diseases and cancer [6]. Although most asthma patients can achieve control through treatment, the burden of managing the disease remains high, and asthma does not receive as much attention as other non-communicable diseases [7]. Current strategies for improving asthma prevention are targeted at different risk levels but are not fully utilized. To alleviate the global disease burden and promote universal health, finding effective methods to address this issue is crucial.

Diet is closely related to human health, with approximately 3 billion people worldwide suffering from malnutrition, of which about 2 billion are overnourished. Unhealthy diets increase the burden of diseases on the human body and affect the planet's carrying capacity, contributing to climate and environmental changes. To better balance the health of humans and the planet, the EAT-Lancet Commission introduced a new dietary metric in 2019—the Planetary Health Diet Index (PHDI) [8]. The PHDI provides guidelines for various food groups that collectively constitute the optimal diet for human health and environmental sustainability. It emphasizes a higher proportion of plant-based foods, with significant portions of whole grains, fruits, nuts, and vegetables.

The PHDI can potentially prevent 11.6 million premature deaths worldwide, reduce greenhouse gas emissions, decrease environmental pollution, and protect Earth's biodiversity. Asthma results from the interplay between genetic factors and environmental influences, making it particularly susceptible to climate and environmental changes. Research by Biesbroek S et al. [9]. indicates that dietary changes in specific contexts depend on the national disease burden (such as obesity or malnutrition), environmental challenges, and cultural traditions. High-income countries, which have a higher risk of chronic non-communicable diseases and a larger environmental footprint, should thus limit consumption. Another longitudinal study involving 1,050 children demonstrated that children living in environments with abundant greenery had a lower risk of developing asthma compared to those born in areas with a higher presence of animals [10].

Moreover, several studies have shown that a healthy diet may be associated with body mass index (BMI) [11–13]. BMI, as a proxy for body weight and obesity, is considered closely related to asthma [14, 15]. However, whether BMI mediates this relationship remains unknown. Currently, no studies have investigated the association between PHDI and asthma mediated by BMI. For the first time, we investigated the association between the PHDI and asthma prevalence mediated by BMI in this study using a large cross-sectional methodology. It is hypothesized that there may be a negative correlation between the PHDI and asthma prevalence and explored the mediating role of BMI in the correlation between PHDI and asthma prevalence, aiming to validate that an appropriate and healthy PHDI could prevent asthma.

Methods

Study population

The National Health and Nutrition Examination Survey (NHANES) is a nationally representative cross-sectional survey conducted through home interviews and mobile examination centers, aimed at assessing the health and nutritional status of the U.S. population. This survey utilized data from 70,190 participants over seven cycles of NHANES, spanning from 2005 to 2018. After excluding individuals younger than 20 years (n=30,441), pregnant women (n=708), and participants with missing or incomplete PHDI data (n=6,653), a total of 32,388 participants were included in the final analysis. Figure 1 displays a flowchart of the entire selection process. NHANES is approved by the Research Ethics Review Board of the National Center for Health Statistics, and all participants provided informed consent. The data used in this study are de-identified and publicly available (https://www.cdc.gov/nchs/nhanes/index.htm).

Measurement

Planetary health diet index

The measurement of the PHDI is based on the ranges provided in the EAT-Lancet Commission's scientific report. It comprises 14 food categories: whole grains, whole fruits, non-starchy vegetables, nuts and seeds,

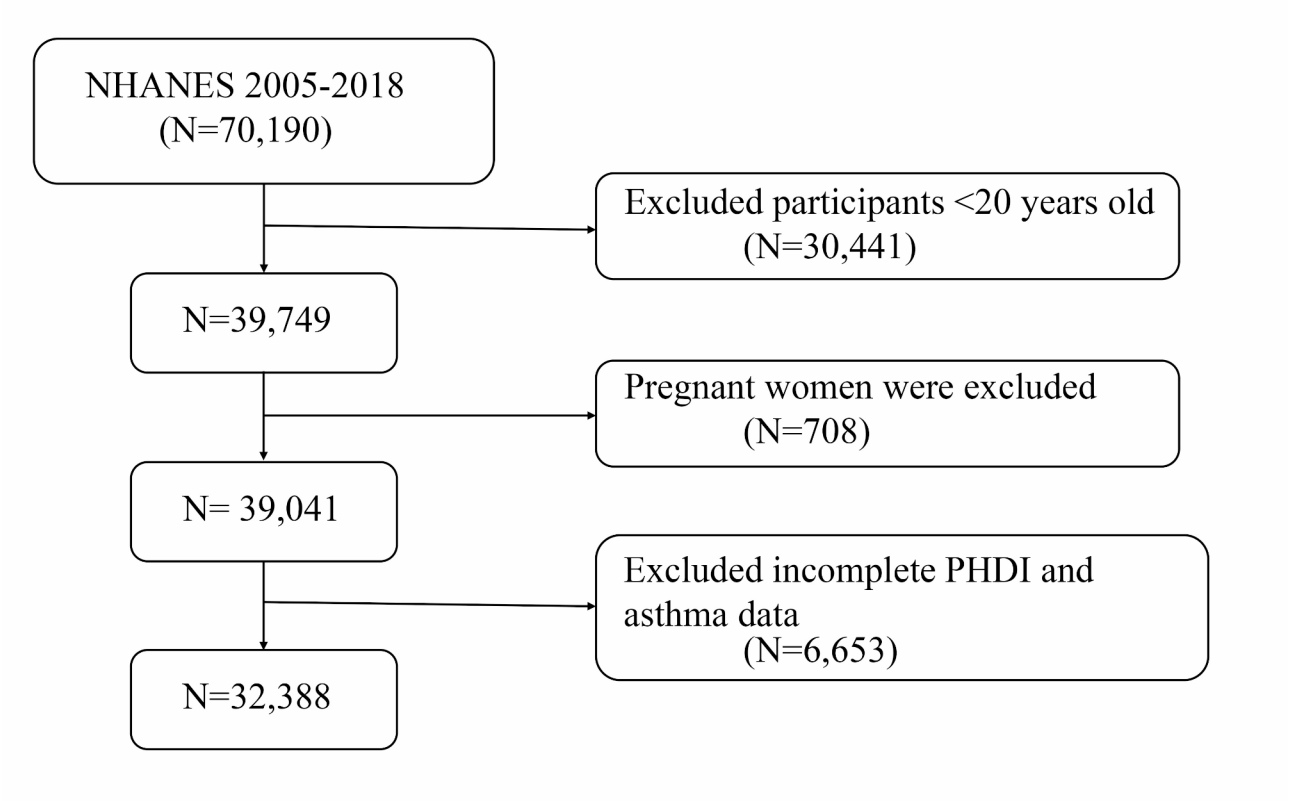

Fig. 1 A flow diagram of eligible participant selection in the National Health and Nutrition Examination Survey

legumes and unsaturated oils, starchy vegetables, dairy products, red and processed meats, poultry, eggs, fish, saturated fats and trans fats, added sugars, and fruit juices. The first six categories are adequacy components that are encouraged for consumption, while the latter eight are moderation components that are discouraged for consumption. Each food category is scored on a scale from 0 to 10, resulting in a theoretical PHDI range from 0 to 140. Further details on the PHDI can be found in previous studies and Table S1 [8, 16, 17].

Diagnosis of asthma

According to previous studies [18, 19], the criteria for diagnosing asthma were extracted from the questionnaire section of the NHANES database: (1) "Has a doctor or other health professional ever told you that you have asthma?" (2) "Do you still have asthma?" Participants who answered "yes" to both questions were classified as having asthma, while those who did not were excluded. For a detailed explanation, see the website (https://www.cdc.gov/nchs/nhanes/index.htm).

Covariables

We constructed a directed acyclic graph (DAG) [20] to visualize the hypothesized associations of the primary exposure (Planetary Health Diet Index) with the outcomes of interest (the prevalence of asthma), and potential covariates. According to previous studies [21–23], the covariates in this research include age, gender, race, marital status, education level, family poverty-to-income ratio (PIR), energy intake, smoking, alcohol consumption, hypertension, diabetes, and hypercholesterolemia. For detailed information on these covariates, please refer to Table S2. The resulting DAG is presented in Figure S1.

Statistical analyses

In this study, all data were statistically analyzed using R (version 4.3.1). To guarantee that the data in our investigation were nationally representative, we used the weights that the NCHS suggested. The weighting variable was the two-day dietary sample weight (WTDR2D), and the new weights for the years 2005–2018 were computed as 1/7 × WTDR2D. The data were weighted, with continuous variables presented as mean ± standard deviation, and p-values calculated using weighted Students T-test. Percentages for categorical variables (weighted N, %) and their p-values were calculated using weighted chi-square tests [24]. The association between PHDI and asthma was analyzed using multivariable logistic regression models, where PHDI was categorized into quartiles. Trend tests and p-values for linear trends were calculated

to determine the consistency of the relationship. Three models were constructed in this study: (1) an unadjusted crude model; (2) a model adjusted for age, gender, race, education level, marital status, and family poverty-to-income ratio (PIR); and (3) a model further adjusted for energy intake, smoking, alcohol consumption, hypertension, diabetes, and hypercholesterolemia. A smooth curve fitting was applied to further explore the potential linear relationship between PHDI and asthma. Additionally, odds ratios (ORs) were calculated for every 10-point increase in PHDI, with subgroup analyses conducted based on age, gender, race, marital status, education level, PIR, smoking, alcohol consumption, hypertension, diabetes, and hypercholesterolemia. In the subgroup analysis, we further adjusted for age, gender, race, marital status, education level, PIR, energy intake, smoking, alcohol consumption, hypertension, diabetes, and hypercholesterolemia.

The "mediation" package in R software was used to assess the indirect impact, direct effect, and total effect. A mediation analysis with 1000 bootstrap resamples and correction for variables was carried out to determine if BMI mediated the association between PHDI and asthma. The indirect effect/ (indirect effect + direct effect) ×100% was used to compute the mediated fraction [25]. The total effect of PHDI on asthma (path C), the direct effect of PHDI on asthma when BMI (mediator) is included in the model (path C′), the effect of PHDI on BMI (path A), the effect of BMI on asthma (path B), and the indirect effect of BMI on the association between PHDI and asthma (path A*B) are all represented by regression coefficients (Figure S2).

To evaluate the results obtained in this study from a food science perspective, we added detailed data on the daily consumption of individual nutrients, including energy, carbohydrates, dietary fiber, fats, proteins, vitamins, and minerals, and assessed their impact on asthma. In addition, we applied the weighted quantile sum (WQS) regressions to explore the overall effect of individual single nutrients on asthma. WQS is a new statistical tool for estimating both the collective and specific impacts of exposures [26]. The data was divided into training sets (40%) and validation sets (60%) at random. The training sets were then bootstrapped 1000 times. We ran the WQS model in the negative direction because metrics had an inverse relationship with asthma. Metric weights varied from 0 to 1 and added up to 1. Major contributors were determined to be the metrics (average of 10 metrics) with weights greater than 0.1. Given that diabetes, hypertension, and hypercholesterolemia can lead to changes in dietary habits, we excluded participants with these conditions and reanalyzed the data (Table S6). The significance was determined by p-values below 0.05.

Results

Characteristics of the participants

This study included 32,388 participants, with 52% being female and 48% male. There were 4,618 participants with a history of asthma and 27,770 without. Compared to other racial groups, non-Hispanic whites had a higher incidence of asthma (69%). The prevalence of asthma was higher among participants with at least a high school education (85%). The family poverty-to-income ratio (PIR) was inversely related to asthma prevalence. A higher prevalence of asthma was observed among those who were obese, smoked, consumed alcohol, had hypertension, diabetes, or hypercholesterolemia ($p<0.05$). Participants with the prevalence of asthma had lower PHDI scores ($p<0.05$). Baseline characteristics are detailed in Table 1.

Relationship between PHDI and asthma

Table 2 of the multivariable logistic regression model demonstrated an association between PHDI and asthma. In model 3, a negative association was found between PHDI and asthma prevalence (OR 0.96, 95% CI 0.92–0.99). This negative association was more pronounced in the higher quartiles of PHDI, specifically Q2 (OR 0.91, 95% CI 0.79–1.05), Q3 (OR 0.89, 95% CI 0.77–0.99), and Q4 (OR 0.86, 95% CI 0.75–0.98), compared to Q1. Trend tests in each model, using Q1 as the reference, also confirmed this finding ($p<0.05$). A smooth curve fitting was employed to assess the association between PHDI and asthma; as shown in Fig. 2, the correlation between PHDI and asthma was linearly negative (nonlinearity = 0.138).

Subgroup and WQS analysis

The subgroup analysis of the association between PHDI and asthma is shown in Fig. 3. This analysis explored the stability of the relationship and potential interactions by adjusting and stratifying based on age, gender, education level, marital status, income, race, smoking, alcohol consumption, hypertension, diabetes, and hypercholesterolemia. In the subgroup analysis, no significant interaction was found between PHDI and these stratified variables ($p>0.05$), and the negative correlation remained very stable.

In addition, the WQS index from the WQS regression was negatively associated with the risk of asthma (OR 0.93,95% CI 0.88 to 0.98) (Table S5). Figure 4 showed that all nutrients were negatively associated with asthma, with dietary fiber (weight = 0.440) identified as the most important factor influencing the presence of asthma, followed by Vitamin C and Protein (weights = 0.197 and 0.149).

Table 1 Baseline characteristics of all participants were stratified by asthma

Characteristic	Total, *N* = 32,388 (100%)	Non- asthma, *N* = 27,770 (85%)	asthma, *N* = 4,618 (15%)	*P* Value
Age (%)				**< 0.001**
20–40	11,020 (36%)	9,256 (35%)	1,764 (41%)	
41–60	10,935 (37%)	9,407 (37%)	1,528 (35%)	
> 60	10,433 (27%)	9,107 (27%)	1,326 (24%)	
Gender (%)				**< 0.001**
Male	15,853 (48%)	13,899 (49%)	1,954 (41%)	
Female	16,535 (52%)	13,871 (51%)	2,664 (59%)	
Race (%)				**< 0.001**
Non-Hispanic White	14,049 (69%)	11,871 (68%)	2,178 (70%)	
Non-Hispanic Black	7,036 (11%)	5,884 (11%)	1,152 (13%)	
Other	6,367 (12%)	5,497 (12%)	870 (12%)	
Mexican American	4,936 (7.8%)	4,518 (8.3%)	418 (4.9%)	
Married/live with partner (%)				**< 0.001**
No	13,043 (37%)	10,903 (36%)	2,140 (41%)	
Yes	19,345 (63%)	16,867 (64%)	2,478 (59%)	
Education level (%)				0.098
Below high school	7,769 (15%)	6,795 (16%)	974 (14%)	
High School or above	24,619 (85%)	20,975 (84%)	3,644 (86%)	
PIR (%)				**< 0.001**
Not Poor	20,571 (79%)	17,866 (80%)	2,705 (74%)	
poor	9,160 (21%)	7,595 (20%)	1,565 (26%)	
Obesity (%)				**< 0.001**
No	19,641 (63%)	17,247 (64%)	2,394 (56%)	
Yes	12,388 (37%)	10,211 (36%)	2,177 (44%)	
Smoking (%)				**< 0.001**
Never	17,845 (55%)	15,572 (56%)	2,273 (50%)	
Former	7,979 (25%)	6,789 (25%)	1,190 (26%)	
Current	6,564 (20%)	5,409 (19%)	1,155 (24%)	
Drinking (%)				0.103
former	4,936 (14%)	4,198 (14%)	738 (14%)	
heavy	5,839 (21%)	4,975 (20%)	864 (22%)	
mild	10,097 (37%)	8,650 (37%)	1,447 (37%)	
moderate	4,604 (17%)	3,899 (18%)	705 (17%)	
never	4,204 (11%)	3,691 (11%)	513 (9.6%)	
Hypertension (%)				**< 0.001**
No	17,771 (60%)	15,424 (61%)	2,347 (57%)	
Yes	14,057 (40%)	11,852 (39%)	2,205 (43%)	
Diabetes (%)				**0.009**
No	12,109 (74%)	10,418 (74%)	1,691 (71%)	
Yes	5,778 (26%)	4,830 (26%)	948 (29%)	
High cholesterol (%)				0.332
No	17,295 (62%)	14,858 (62%)	2,437 (61%)	
Yes	10,986 (38%)	9,280 (38%)	1,706 (39%)	
Energy, kcal/d (mean (SD))	2,087.61 (840.36)	2,087.78 (835.20)	2,086.61 (870.04)	0.553
Carbohydrate, g/d (mean (SD))	248.78 (107.91)	248.53 (107.39)	250.23 (110.92)	0.801
Fiber, g/d (mean (SD))	16.88 (9.08)	17.00 (9.05)	16.22 (9.27)	**< 0.001**
Total fat, g/d (mean (SD))	80.77 (39.26)	80.76 (39.03)	80.83 (40.61)	0.575
Protein, g/d (mean (SD))	81.88 (35.59)	82.20 (35.32)	79.99 (37.07)	**< 0.001**
Vitamin A, ug/d (mean (SD))	649.38 (564.74)	653.35 (571.87)	626.20 (520.51)	**0.002**
Vitamin C, mg/d (mean (SD))	82.73 (77.85)	83.00 (76.27)	81.15 (86.52)	**< 0.001**
Vitamin E, mg/d (mean (SD))	8.46 (5.60)	8.47 (5.56)	8.38 (5.81)	0.068
Zinc, mg/d (mean (SD))	11.58 (6.90)	11.61 (6.84)	11.39 (7.21)	**0.009**

Table 1 (continued)

Characteristic	Total, *N* = 32,388 (100%)	Non- asthma, *N* = 27,770 (85%)	asthma, *N* = 4,618 (15%)	*P* Value
Selenium, ug/d (mean (SD))	112.87 (53.06)	113.16 (52.18)	111.22 (57.91)	**0.004**
PHDI (mean (SD))	61.51 (15.00)	61.67 (14.97)	60.56 (15.12)	**< 0.001**

Mean (SD) for continuous variables: the P value was calculated by the weighted Students T-test

Percentages (weighted N, %) for categorical variables: the P value was calculated by the weighted chi-square test

Abbreviation: PIR, Ratio of family income to poverty; PHDI, Planetary Health Diet Index

Table 2 Adjusted odds ratios of PHDI and asthma, NHANES 2005–2018

Characteristic	Model 1 [OR (95% CI)]	*p*-value	Model 2 [OR (95% CI)]	*p*-value	Model 3 [OR (95% CI)]	*p*-value
PHDI - asthma						
Continuous (per 10 scores)	0.95 (0.92, 0.98)	0.002	0.96 (0.93, 0.98)	0.006	0.96 (0.92, 0.99)	0.025
Quartile						
Q1	1 (ref.)		1 (ref.)		1 (ref.)	
Q2	0.88 (0.79, 0.97)	0.011	0.89 (0.79, 0.99)	0.042	0.91 (0.79, 1.05)	0.200
Q3	0.83 (0.74, 0.93)	0.002	0.85 (0.75, 0.96)	0.010	0.89 (0.77, 0.99)	0.036
Q4	0.80 (0.71, 0.90)	< 0.001	0.81 (0.71, 0.92)	0.002	0.86 (0.75, 0.98)	0.032
P for trend	< 0.001		0.002		0.026	
Components - asthma						
Energy	1.000 (1.000, 1.000)	> 0.9	1.000 (1.000, 1.000)	0.018	1.000 (1.000, 1.000)	0.055
Carbohydrate	1.000 (1.000, 1.001)	0.500	1.001 (1.000, 1.001)	0.019	1.001 (1.000, 1.001)	0.026
Fiber	0.990 (0.984, 0.997)	0.003	0.998 (0.991, 1.004)	0.400	0.990 (0.982, 1.003)	0.500
Total fat	1.000 (0.999, 1.001)	> 0.9	1.001 (1.000, 1.003)	0.043	1.001 (0.999, 1.003)	0.600
Protein	0.998 (0.997, 1.000)	0.019	1.000 (0.999, 1.001)	> 0.9	0.999 (0.997, 1.003)	0.700
Vitamin A	0.999 (0.999, 1.000)	0.051	0.999 (0.999, 1.000)	0.400	0.999 (0.999, 1.000)	0.300
Vitamin C	0.999 (0.999, 1.000)	0.400	0.999 (0.999, 1.001)	> 0.9	0.999 (0.999, 1.001)	0.200
Vitamin E	0.997 (0.998, 1.006)	0.500	1.002 (0.994, 1.010)	0.600	0.989 (0.975, 1.004)	0.400
Zinc	0.995 (0.987, 1.003)	0.200	1.003 (0.995, 1.010)	0.400	0.998 (0.988, 1.012)	0.500
Selenium	0.999 (0.998, 1.000)	0.200	1.000 (0.999, 1.001)	0.500	0.999 (0.998, 1.001)	0.800

Model 1: no covariates were adjusted

Model 2: age, gender, education level, marital, PIR, and race were adjusted

Model 3: age, gender, education level, marital, PIR, race, smoking, drinking, energy intake, hypertension, diabetes, and high cholesterol were adjusted

Abbreviation: PHDI, Planetary Health Diet Index; PIR, Ratio of family income to poverty; OR, odds ratio; CI, confidence interval

Mediation effect

Table S3 displays the association between BMI and asthma. In model 3, Following adjustment for all covariates, compared to the first quartile (Q1) of BMI, the odds of asthma increased by 44% in the fourth quartile (Q4) [OR = 1.44, 95% CI:1.23, 1.68]. When BMI was considered as a continuous variable, the positive relationship between BMI and asthma remained statistically significant (OR = 1.02, 95% CI: 1.01, 1.03). Following adjustment for all covariates, there was a significant statistical association between PHDI and BMI (β=-0.64, 95% CI: -0.73, -0.56, $P<0.001$) (Table S4). Based on the above analysis, our study meets the prerequisites for conducting mediation analysis. Following adjustment for all covariates, we observed the mediation effect of BMI (Figure S2). BMI (indirect effect= $-2.36*10^{-3}$, $P<0.001$; direct effect= $-4.70*10^{-3}$, $P=0.036$) mediated 33.85% (mediation proportion = indirect effect / (indirect effect + direct effect) *100%, $P<0.001$) of the association between PHDI and asthma. Therefore, BMI can be considered a mediating factor in the relationship between PHDI and asthma.

Discussion

Based on the NHANES database, this study found that after adjusting for relevant covariates, there is a negative association between PHDI and the prevalence of asthma and that BMI mediated this relationship, thus validating our hypothesis. The results of the RCS and subgroup analyses reaffirmed that higher PHDI scores are beneficial in reducing the prevalence of asthma. Additionally, WQS analysis suggests that dietary components of the PHD, including fiber, vitamin C, and protein, play significant roles in the development of asthma.

An important pathogenic mechanism of asthma is airway hyperresponsiveness. Upon exposure to allergens, various inflammatory cells such as eosinophils, mast cells, T cells, neutrophils, airway epithelial cells, and macrophages release inflammatory mediators and cytokines. This release causes damage to airway epithelial

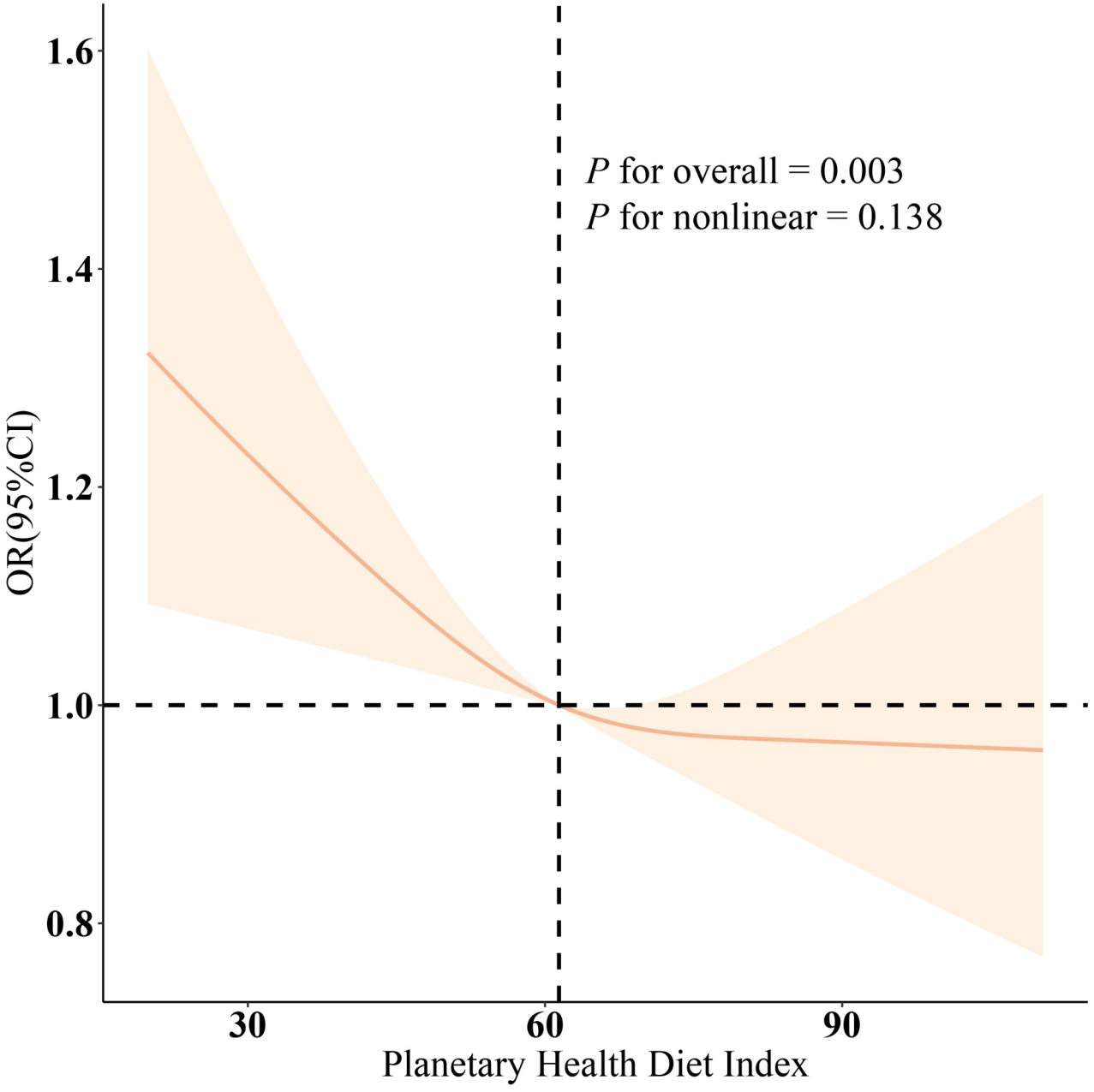

Fig. 2 Dose-response relationships between PHDI and asthma. OR (solid lines) and 95% confidence levels (shaded areas) were adjusted for age, gender, education level, marital, PIR, race, smoking, drinking, energy intake, hypertension, diabetes, and high cholesterol

cells and exposes epithelial nerve endings, leading to hyperresponsiveness. Studies [27] indicate that the underlying mechanisms of asthma are multifactorial, involving environmental factors, genetic predispositions, and lifestyle choices, with diet being a significant factor. Previous research [28] has found a positive correlation between asthma prevalence and gross domestic product (GDP). For instance, the prevalence of asthma in developed Western countries is typically around 10%, whereas in less developed countries, it is ≤ 1% [29].

The disparity in asthma prevalence between developed and developing countries can be attributed not only to underdiagnosis, high misdiagnosis rates, and inadequate treatment in low- and middle-income countries but also to differences in dietary patterns. Studies [30] have shown that Western dietary patterns emphasize animal-based foods while neglecting whole grains, fruits, vegetables, and legumes. For example, the intake of saturated fats in the U.S. population, which follows a predominantly Western diet, significantly exceeds recommended levels, while fruit and vegetable consumption falls below the

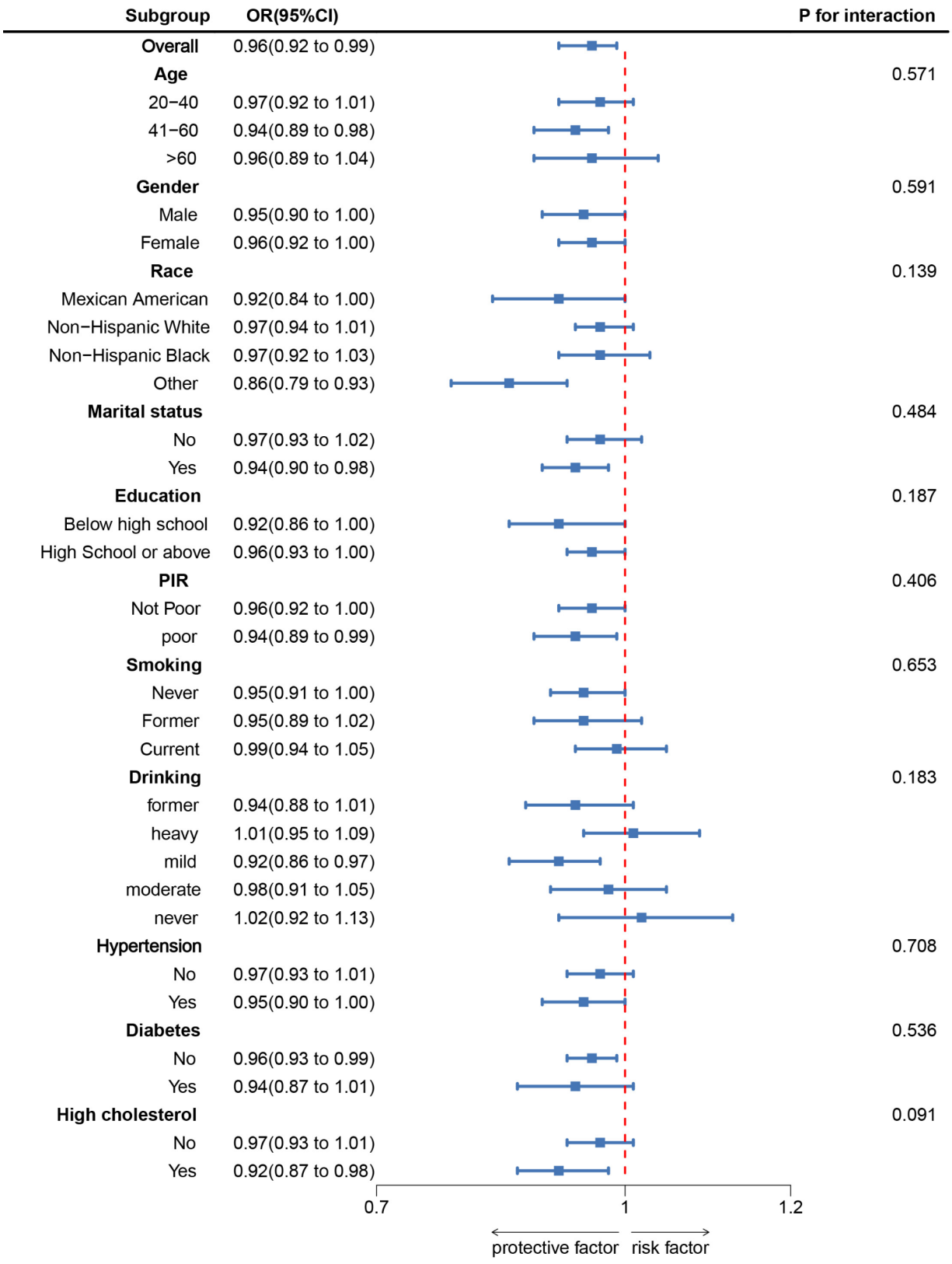

Fig. 3 Subgroup analysis between PHDI and asthma. ORs were calculated as per 10 scores increase in PHDI. Analyses were adjusted for age, gender, education level, marital, PIR, race, smoking, drinking, energy intake, hypertension, diabetes, and high cholesterol

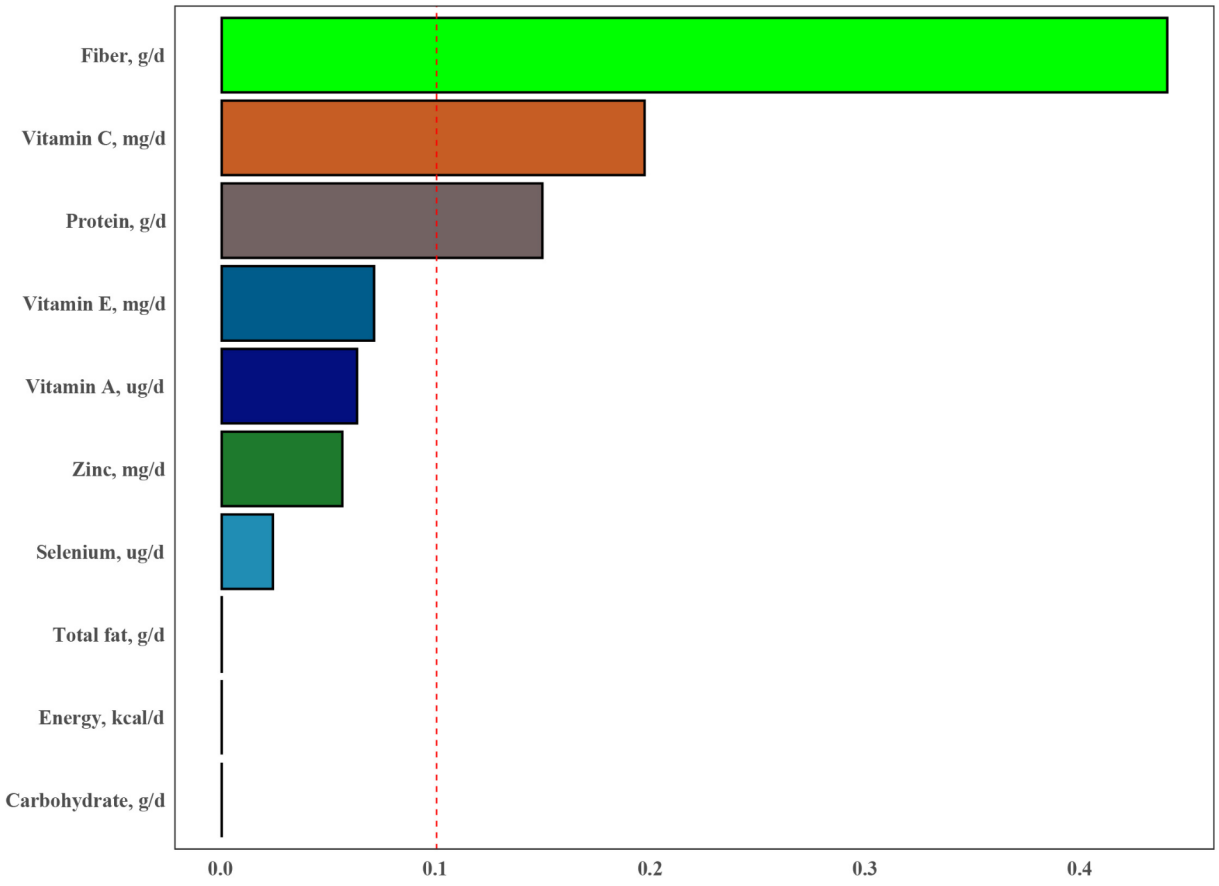

Fig. 4 Weights represent the proportion of partial effect for each PHDI metric in the WQS regression. Model adjusted for age, sex, race, PIR, educational level, and marital status

norm. As dietary patterns westernize, the risk of asthma increases [31]. In contrast, diets that emphasize whole grains, fruits, vegetables, and legumes, and de-emphasize high-fat meats and dairy products, are beneficial in reducing asthma risk. A case-control study involving 287 children aged 9–19 in Lima, Peru [32], used a modified Mediterranean Diet Score (MDS) to analyze dietary patterns. Preliminary analysis showed a negative correlation between adherence to the Mediterranean diet and the incidence of asthma in children. Similar plant-based dietary patterns have also been shown to reduce asthma risk [33, 34]. A meta-analysis and systematic review of 31 studies evaluated asthma outcomes such as prevalence, asthma-related quality of life, symptoms, lung function, frequency of asthma attacks, asthma control, and inflammatory markers associated with asthma. The results indicated a significant association between dietary patterns and asthma in 12 of the studies, with protective dietary patterns including components like black bread, nuts, and wine [35]. Adhering to a plant-based dietary pattern can result in higher PHDI scores. A multicenter cohort study conducted in Brazil with 14,779 participants using a 114-item Food Frequency Questionnaire (FFQ) found that the PHDI was positively correlated with carbohydrates, plant proteins, polyunsaturated fats, fiber, and micronutrients from fruits, vegetables, oilseeds, and whole grains. In the PHDI, nuts and peanuts, legumes, fruits, total vegetables, and whole grains are defined as adequacy components. Recent research [36] showed that PHDI scores are positively correlated with plant protein, fiber, polyunsaturated fats, vitamins A, E, K, C, and folic acid ($p<0.001$), and negatively correlated with animal protein, total fat, monounsaturated fats, and riboflavin ($p<0.001$).

This study indicates that dietary fiber, vitamin C, and protein play significant roles in the development of asthma. Meta-analyses [37] have long shown that high consumption of fruits and vegetables is associated with a reduced risk of asthma in both children and adults. Retrospective studies [38] have demonstrated that a high

intake of fruits and vegetables is correlated with higher forced expiratory volume in one second (FEV1), reducing the risk of asthma and the incidence of wheezing. Fruit and vegetable consumption can also effectively control and alleviate symptoms during asthma attacks. A cohort study involving 2,870 children [39] showed that habitual fruit consumption helps alleviate asthma symptoms (OR=0.93, 95% CI 0.85-1.00), and long-term fruit intake was negatively correlated with the frequency of asthma symptoms (OR=0.90, 95% CI 0.82–0.99) and allergen sensitization (OR=0.90, 95% CI 0.82–0.99). However, Willers et al. further noted that increased consumption of certain foods, whether early or late in life or over extended periods, does not have a consistent impact on asthma and atopy in 8-year-old children [39]. Consequently, fruit intake may be inversely related to asthma. A prospective cohort study [40] observing 520 children found that fruit intake was inversely associated with the incidence of asthma, rhinitis, and allergy symptoms. The incidence of asthma symptoms decreased from 33.3 to 28.3% to 14.3% across groups with increasing fruit intake (P for trend=0.01). Additionally, a Dutch study indicated a negative correlation between whole grain consumption and asthma incidence, which our study further supports.

Whole grains are essential components of a healthy diet, providing dietary fiber, B vitamins, minerals, and other nutrients. Early epidemiological studies have linked asthma prevalence to dietary habits. A study using questionnaires and clinical data to define asthma [41] found a negative correlation between whole grain intake and asthma (OR=0.46, 95% CI 0.19-10), suggesting that high consumption of whole grain products may reduce asthma attacks in children. A Danish study [42] used a disease-death multi-state model to evaluate the correlation between whole grain intake and life expectancy (defined as 20 years of follow-up without cancer, asthma, chronic obstructive pulmonary disease, etc.). It found that for every doubling of whole grain intake, the difference in disease-free life expectancy (without cancer, type 2 diabetes, ischemic heart disease, stroke, asthma, chronic obstructive pulmonary disease or dementia or with disease (any of the listed)) increased by 0.43 (95% CI: 0.33–0.52) in men and 0.15 (95% CI: 0.06–0.24) in women over an average follow-up period of 13.8 and 17.5 years for 22,606 men and 25,468 women, respectively. Furthermore, studies have shown that dietary fiber intake is positively correlated with improved lung function. Higher fiber intake was associated with higher average FEV1 and FVC measurements, with those consuming the most fiber having average FEV1 and FVC values that were 82 ml and 129 ml higher than those consuming the least fiber (P=0.05 and 0.01, respectively). Therefore, a fiber-rich diet may also play a role in improving asthma [43].

Current observational and clinical research evidence suggests that plant-based dietary patterns (primarily consisting of fruits, vegetables, and whole grains) are valuable in preventing asthma, whereas Western dietary patterns (emphasizing red meat, processed meats, refined grains, and added sugars) appear to increase asthma risk [44]. As a modifiable factor affecting lung health [45], the protective effects of a plant-based dietary model (corresponding to higher PHDI scores) may be associated with oxidative stress, inflammation, and gut microbiota. In contrast, the exacerbation of asthma by a Western dietary model (corresponding to lower PHDI scores) is likely linked to inflammation.

Oxidative stress and inflammation are key mechanisms by which diet influences asthma. Oxidative stress occurs when the balance between the production and elimination of free radicals is disrupted, leading to neutrophilic inflammatory infiltration and increased secretion of proteases, resulting in the production of a large amount of reactive oxygen species (ROS). Excessive ROS can also increase the expression of NLRP3, triggering the release of pro-inflammatory cytokines and inducing inflammation. During inflammation, mast cells and leukocytes accumulate at the damaged site, increasing oxygen intake and subsequently the release and accumulation of ROS at the site, thereby exacerbating oxidative stress. Oxidative stress and inflammation are associated with poor asthma outcomes. When the lungs are exposed to oxidative stress and an inflammatory environment, it can lead to pulmonary dysfunction such as asthma. Research by Wood LG et al. [46, 47]. confirmed that a diet rich in plant-based foods (high PHDI scores) can reduce inflammatory responses and enhance anti-inflammatory factors. Early studies [48] have shown that adopting a plant-based diet provides antioxidants and unsaturated fatty acids, which can alleviate oxidative stress and inflammation, thus mitigating asthma. Increased intake of fruits and vegetables is negatively correlated with the number of pro-inflammatory cytokines and airway neutrophils in asthma patients [49]. Whole grains also have antioxidant and anti-inflammatory properties. Studies [50] have shown that higher consumption of whole grains is associated with lower levels of serum C-reactive protein and tumor necrosis factor-α receptor-2. Conversely, Western dietary patterns exacerbate oxidative stress and inflammation. Research [51] indicates that dairy consumption is positively correlated with the concentration of pro-inflammatory interleukin (IL)-17 F (P<0.05), suggesting that the IL-17 F-dependent inflammatory pathway may mediate asthma development. Kim et al. [52]. found that a high-fat diet induces the production of numerous pulmonary cytokines, increasing airway hyperresponsiveness and inflammation, as evidenced by elevated IL-6 and IL-8 expression in sputum samples. A diet high in mixed

fats not only increases the release of TNF-α and IL-6 but also activates Toll-like receptors, triggering immune responses [53].

The composition and function of respiratory and gut microbiota interact, known as the "gut-lung axis," and are related to airway immune function. Dysfunction of the airway epithelial barrier and increased permeability contribute to antigen sensitization and the progression of asthma. Concurrently, gut microbiota dysbiosis can increase the risk of asthma. Gut microbiota is closely related to dietary patterns, with different diets influencing the production of various metabolites by gut microbiota, thereby affecting immune responses and modulating pulmonary pro-inflammatory reactions [54]. Studies have shown that plant-based diets (with high PHDI scores) can modulate gut immune responses to improve airway inflammation [55]. Intake of dietary fiber can lead to the production of short-chain fatty acids by gut microbiota, which have a regulatory effect on immune responses. Consuming yogurt can supplement the body with prebiotics, which are dietary supplements that regulate gut microbiota, affect blood lipid levels, and enhance immune system function. Research [56] indicates that women who take prebiotics during pregnancy and lactation may reduce the risk of allergen sensitization.

This study found that BMI mediates the association between PHDI and asthma, potentially through the following mechanisms: (1) The dietary patterns within PHDI may influence asthma risk through their impact on BMI. For instance, diets rich in vegetables, fruits, and whole grains help maintain a healthy BMI [57], potentially reducing asthma risk. Conversely, high-fat and high-sugar diets may increase BMI [58], thereby elevating asthma risk. (2) The gut microbiota in obese individuals typically differs significantly from that of healthy individuals, often characterized by a reduction in beneficial bacteria and an increase in harmful bacteria [59]. This dysbiosis can affect lung health via the "gut-lung axis," increasing airway susceptibility [54]. Elevated BMI may mediate the relationship between PHDI and asthma indirectly by altering gut microbiota composition, particularly when high-fat and high-sugar diets further disrupt the gut microbiome. (3) Increased BMI may influence immune system function [60], potentially affecting how certain dietary components in PHDI (such as high sugar or high fat) impact asthma risk through BMI's effects on the immune system. (4) Individuals with higher BMI are more likely to experience airway narrowing and airflow limitation [61], which may increase susceptibility to asthma or exacerbate asthma symptoms during an attack. The mechanical pressure of obesity on the airways could amplify the effects of unhealthy diets within PHDI. (5) Expansion of adipose tissue in obese individuals leads to macrophage infiltration and elevated levels of pro-inflammatory cytokines (e.g., TNF-α, IL-6, and CRP) [62]. These inflammatory markers can affect the lungs via the bloodstream, increasing airway inflammation and thereby worsening or triggering asthma symptoms [63]. Elevated BMI may enhance or accelerate the negative impact of unhealthy dietary components in PHDI on asthma risk through this systemic inflammation pathway.

In summary, the PHDI is a dietary metric representing both planetary and human dietary health. Asthma, beyond its genetic predispositions, is closely associated with diet and environmental factors. Current research predominantly focuses on the relationships between dietary patterns, dietary fiber, and nutrients with asthma. There has been no previous research on the association between PHDI and asthma risk in adults. This study is the first to investigate the role of BMI in the relationship between PHDI and asthma prevalence using the NHANES database. First, a multivariable logistic regression model demonstrated a negative correlation between PHDI and asthma. Smooth curve fitting assessed the relationship, confirming a negative and linear correlation (nonlinearity=0.138). Subgroup analyses adjusting for age, gender, education level, marital status, income, race, energy intake, smoking, alcohol consumption, hypertension, diabetes, and hypercholesterolemia showed no significant interaction between PHDI and these stratified variables ($p>0.05$), indicating a very stable relationship. Finally, the mediation analysis results indicate that BMI serves as a mediator in the association between PHDI and asthma. Literature review reveals that plant-based dietary patterns correspond to higher PHDI scores, while Western and high-fat dietary patterns correspond to lower PHDI scores. Previous studies have found that a high intake of fruits, vegetables, and grains can reduce the risk of asthma [37, 38], whereas a high intake of red meat, eggs, fish, saturated fats trans fats, added sugars and fruit juices increases the risk of asthma. The underlying mechanisms are closely related to oxidative stress, inflammation, and gut microbiota.

Additionally, this study has limitations: (1) The cross-sectional nature of the data limits causal inference, and although PHDI may have an impact on asthma through BMI, more comprehensive prospective cohort studies, randomized controlled trials, or animal studies are required in the future to determine the precise pathogenic processes. (2) The constraints of the NHANES database preclude us from eliminating the potential confounding factors' ultimate impact on the study results, even though we included a reasonably high number of covariates based on prior research to improve the robustness of our study results. As a result, it is important to interpret the study's findings carefully and impartially. (3) Given that the analysis of the relationship between dietary components and disease risk should adhere to

the isocaloric principle, incorporating all macronutrients, micronutrients, and energy within the same model may introduce bias [64]. Consequently, the WQS analysis results in this study should be interpreted with caution. (4) The development of the PHDI is based on the Food Frequency Questionnaire (FFQ). Although the FFQ is the most used method in epidemiological studies to investigate the relationship between diet and health outcomes, it has its limitations. These include the incompleteness of the food list and the potential for recall bias in the reports provided [65]. (5) It is well known that asthma is associated with socioeconomic status, urbanization, and local air pollution levels, all of which play a significant role in the pathogenesis of asthma [66]. In the future, we plan to pursue multicenter studies with larger sample sizes or explore alternative study designs to address this limitation.

Conclusion

In conclusion, our study indicates a linear inverse association between PHDI and asthma risk, with BMI mediating this relationship. Randomized controlled trials are necessary to further explore the connection between PHDI and asthma symptoms. Future prospective studies and fundamental experiments are needed to delve deeper into the mechanisms, which can then guide dietary recommendations for asthma patients while promoting both planetary and human health. Providing specific dietary structure recommendations can offer new perspectives for the comprehensive prevention and treatment of chronic non-communicable diseases like asthma, thus reducing the global disease burden. Integrating human dietary health, respiratory health, and planetary health into a unified approach can advance universal health coverage and alleviate the global disease burden.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12889-024-19856-1.

Supplementary Material 1

Acknowledgements
We are very grateful to the NHANES database for all the data provided.

Author contributions
S.H. contributed to the original draft, Methodology, Supervision, Project administration, and Formal analysis. Q.H. contributed to the original draft, Methodology, and Formal analysis. X.W. contributed to Conceptualization, Methodology, Validation, Formal analysis, Resources, and Data curation. S.C. contributed to Validation, Formal analysis, Resources, and Data curation. H.G. was involved in Writing – review & editing, Supervision, Project administration, and Investigation.

Funding
This study was not financially supported by any governmental, corporate, or non-profit entities.

Data availability
The study involved the analysis of publicly available datasets. The data can be accessed at the following URL: https://www.cdc.gov/nchs/nhanes/.

Declarations

Ethics approval and consent to participate
Data survey conducted by NHANES has been approved by the NCHS Research Ethics Review Board (ERB). All information from the NHANES program is available and free for public, so an individual investigator was not necessary to obtain approval from the institution's internal ethics review board.

Consent for publication
Not Applicable.

Competing interests
The authors declare no competing interests.

Author details
[1]Department of Oncology Surgery, Fuzhou Hospital of Traditional Chinese Medicine Affiliated to Fujian University of Traditional Chinese Medicine, Fuzhou City, Fujian Province, China
[2]Graduate School of Tianjin, University of Traditional Chinese Medicine, Tianjin City, China
[3]Department of Physiology, College of Medicine, Chosun University, Gwangju, Korea

Received: 10 July 2024 / Accepted: 22 August 2024
Published online: 26 August 2024

References

1. GBD 2019 Diseases and Injuries Collaborators. Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019: a systematic analysis for the global burden of Disease Study 2019. Lancet. 2020;396:1204–22.
2. García-Marcos L, Chiang C-Y, Asher MI, Marks GB, El Sony A, Masekela R, et al. Asthma management and control in children, adolescents, and adults in 25 countries: a global Asthma Network Phase I cross-sectional study. Lancet Glob Health. 2023;11:e218–28.
3. Papi A, Brightling C, Pedersen SE, Reddel HK, Asthma. Lancet. 2018;391:783–800.
4. To T, Stanojevic S, Moores G, Gershon AS, Bateman ED, Cruz AA, et al. Global asthma prevalence in adults: findings from the cross-sectional world health survey. BMC Public Health. 2012;12:204.
5. Raghavan D, Jain R. Increasing awareness of sex differences in airway diseases. Respirology. 2016;21:449–59.
6. GBD Chronic Respiratory Disease Collaborators. Prevalence and attributable health burden of chronic respiratory diseases, 1990–2017: a systematic analysis for the global burden of Disease Study 2017. Lancet Respir Med. 2020;8:585–96.
7. 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.
8. Willett W, Rockström J, Loken B, Springmann M, Lang T, Vermeulen S, et al. Food in the Anthropocene: the EAT-Lancet Commission on healthy diets from sustainable food systems. Lancet. 2019;393:447–92.
9. Biesbroek S, Kok FJ, Tufford AR, Bloem MW, Darmon N, Drewnowski A, et al. Toward healthy and sustainable diets for the 21st century: importance of sociocultural and economic considerations. Proc Natl Acad Sci U S A. 2023;120:e2219272120.
10. Cavaleiro Rufo J, Paciência I, Hoffimann E, Moreira A, Barros H, Ribeiro AI. The neighbourhood natural environment is associated with asthma in children: a birth cohort study. Allergy. 2021;76:348–58.
11. Gutiérrez-Pliego LE, Camarillo-Romero E, del Montenegro-Morales S, de Garduño-García LP. J. Dietary patterns associated with body mass index (BMI) and lifestyle in Mexican adolescents. BMC Public Health. 2016;16:850.

12. Platikanova M, Yordanova A, Hristova P. Dependence of body Mass Index on some Dietary habits: an application of classification and regression tree. Iran J Public Health. 2022;51:1283–94.
13. Newby P, Muller D, Hallfrisch J, Qiao N, Andres R, Tucker KL. Dietary patterns and changes in body mass index and waist circumference in adults123. Am J Clin Nutr. 2003;77:1417–25.
14. Kang M, Sohn S-J, Shin M-H. Association between Body Mass Index and Prevalence of Asthma in Korean adults. Chonnam Med J. 2020;56:62–7.
15. Hjellvik V, Tverdal A, Furu K. Body mass index as predictor for asthma: a cohort study of 118,723 males and females. Eur Respir J. 2010;35:1235–42.
16. Frank SM, Jaacks LM, Adair LS, Avery CL, Meyer K, Rose D, et al. Adherence to the Planetary Health Diet Index and correlation with nutrients of public health concern: an analysis of NHANES 2003–2018. Am J Clin Nutr. 2024;119:384–92.
17. Frank SM, Jaacks LM, Meyer K, Rose D, Adair LS, Avery CL, et al. Dietary quality and dietary greenhouse gas emissions in the USA: a comparison of the planetary health diet index, healthy eating index-2015, and dietary approaches to stop hypertension. Int J Behav Nutr Phys Act. 2024;21:36.
18. Tian Z, Li X, Han Y, Zhang X. The association between the composite dietary antioxidant index and asthma in US children aged 3–18 years: a cross-sectional study from NHANES. Sci Rep. 2024;14:17204.
19. Wang K, Chen Z, Wei Z, He L, Gong L. Association between body fat distribution and asthma in adults: results from the cross-sectional and bidirectional mendelian randomization study. Front Nutr. 2024;11:1432973.
20. Li Y, Liu X, Lv W, Wang X, Du Z, Liu X, et al. Metformin use correlated with lower risk of cardiometabolic diseases and related mortality among US cancer survivors: evidence from a nationally representative cohort study. BMC Med. 2024;22:269.
21. Wang Y, Han X, Li J, Zhang L, Liu Y, Jin R, et al. Associations between the compositional patterns of blood volatile organic compounds and chronic respiratory diseases and ages at onset in NHANES 2003–2012. Chemosphere. 2023;327:138425.
22. Sveiven SN, Bookman R, Ma J, Lyden E, Hanson C, Nordgren TM. Milk consumption and respiratory function in Asthma patients: NHANES Analysis 2007–2012. Nutrients. 2021;13:1182.
23. Cheng W, Bu X, Xu C, Wen G, Kong F, Pan H, et al. Higher systemic immune-inflammation index and systemic inflammation response index levels are associated with stroke prevalence in the asthmatic population: a cross-sectional analysis of the NHANES 1999–2018. Front Immunol. 2023;14:1191130.
24. Feng G, Huang S, Zhao W, Gong H. Association between life's essential 8 and overactive bladder. Sci Rep. 2024;14:11842.
25. Wang S, Shan T, Zhu J, Jiang Q, Gu L, Sun J, et al. Mediation effect of body Mass Index on the Association of Urinary Nickel Exposure with serum lipid profiles. Biol Trace Elem Res. 2023;201:2733–43.
26. Zhao S, Tang Y, Li Y, Shen H, Liu A. Associations between Life's essential 8 and depression among US adults. Psychiatry Res. 2024;338:115986.
27. Wen J, Wang C, Giri M, Guo S. Association between serum folate levels and blood eosinophil counts in American adults with asthma: results from NHANES 2011–2018. Front Immunol. 2023;14:1134621.
28. Wang D, Xiao W, Ma D, Zhang Y, Wang Q, Wang C, et al. Cross-sectional epidemiological survey of asthma in Jinan, China. Respirology. 2013;18:313–22.
29. Paciência I, Cavaleiro Rufo J. Urban-level environmental factors related to pediatric asthma. Porto Biomed J. 2020;5:e57.
30. Kim J-H, Ellwood PE, Asher MI. Diet and Asthma: looking back, moving forward. Respir Res. 2009;10:49.
31. Lee-Kwan SH, Moore LV, Blanck HM, Harris DM, Galuska D. Disparities in State-Specific Adult Fruit and Vegetable Consumption - United States, 2015. MMWR Morb Mortal Wkly Rep. 2017;66:1241–7.
32. Rice JL, Romero KM, Galvez Davila RM, Meza CT, Bilderback A, Williams DL, et al. Association between Adherence to the Mediterranean Diet and Asthma in Peruvian children. Lung. 2015;193:893–9.
33. Vassilopoulou E, Guibas GV, Papadopoulos NG. Mediterranean-Type diets as a protective factor for Asthma and Atopy. Nutrients. 2022;14:1825.
34. Bédard A, Northstone K, Henderson AJ, Shaheen SO. Mediterranean diet during pregnancy and childhood respiratory and atopic outcomes: birth cohort study. Eur Respir J. 2020;55:1901215.
35. Lv N, Xiao L, Ma J. Dietary pattern and asthma: a systematic review and meta-analysis. J Asthma Allergy. 2014;7:105–21.
36. Cacau LT, De Carli E, de Carvalho AM, Lotufo PA, Moreno LA, Bensenor IM, et al. Development and validation of an Index based on EAT-Lancet recommendations: the Planetary Health Diet Index. Nutrients. 2021;13:1698.
37. Uddenfeldt M, Janson C, Lampa E, Leander M, Norbäck D, Larsson L, et al. High BMI is related to higher incidence of asthma, while a fish and fruit diet is related to a lower- results from a long-term follow-up study of three age groups in Sweden. Respir Med. 2010;104:972–80.
38. Mendes F, de Paciência C, Cavaleiro Rufo I, Farraia J, Silva M, Padrão D. Higher diversity of vegetable consumption is associated with less airway inflammation and prevalence of asthma in school-aged children. Pediatr Allergy Immunol. 2021;32:925–36.
39. Willers SM, Wijga AH, Brunekreef B, Scholtens S, Postma DS, Kerkhof M, et al. Childhood diet and asthma and atopy at 8 years of age: the PIAMA birth cohort study. Eur Respir J. 2011;37:1060–7.
40. Kusunoki T, Takeuchi J, Morimoto T, Sakuma M, Yasumi T, Nishikomori R, et al. Fruit intake reduces the onset of respiratory allergic symptoms in schoolchildren. Pediatr Allergy Immunol. 2017;28:793–800.
41. Tabak C, Wijga AH, de Meer G, Janssen N, a. H, Brunekreef B, Smit HA. Diet and Asthma in Dutch school children (ISAAC-2). Thorax. 2006;61:1048–53.
42. Eriksen AK, Grand MK, Kyrø C, Wohlfahrt J, Overvad K, Tjønneland A, et al. Whole-grain intake in mid-life and healthy ageing in the Danish Diet, Cancer and Health cohort. Eur J Nutr. 2024. https://doi.org/10.1007/s00394-024-03357-3.
43. Hanson C, Lyden E, Rennard S, Mannino DM, Rutten EPA, Hopkins R, et al. The relationship between Dietary Fiber intake and lung function in the National Health and Nutrition Examination Surveys. Ann Am Thorac Soc. 2016;13:643–50.
44. Medina-Remón A, Kirwan R, Lamuela-Raventós RM, Estruch R. Dietary patterns and the risk of obesity, type 2 diabetes mellitus, cardiovascular diseases, asthma, and neurodegenerative diseases. Crit Rev Food Sci Nutr. 2018;58:262–96.
45. Visser E, de Jong K, van Zutphen T, Kerstjens HAM, Ten Brinke A. Dietary inflammatory index and clinical outcome measures in adults with moderate-to-severe asthma. J Allergy Clin Immunol Pract. 2023;11:3680–e36897.
46. Wood LG, Baines KJ, Fu J, Scott HA, Gibson PG. The neutrophilic inflammatory phenotype is associated with systemic inflammation in asthma. Chest. 2012;142:86–93.
47. Fu J, Baines KJ, Wood LG, Gibson PG. Systemic inflammation is associated with differential gene expression and airway neutrophilia in asthma. OMICS. 2013;17:187–99.
48. Schwingshackl L, Hoffmann G. Mediterranean dietary pattern, inflammation and endothelial function: a systematic review and meta-analysis of intervention trials. Nutr Metab Cardiovasc Dis. 2014;24:929–39.
49. Wood LG, Garg ML, Smart JM, Scott HA, Barker D, Gibson PG. Manipulating antioxidant intake in asthma: a randomized controlled trial. Am J Clin Nutr. 2012;96:534–43.
50. Slavin JL, Jacobs D, Marquart L, Wiemer K. The role of whole grains in disease prevention. J Am Diet Assoc. 2001;101:780–5.
51. Han Y-Y, Forno E, Brehm JM, Acosta-Pérez E, Alvarez M, Colón-Semidey A, et al. Diet, interleukin-17, and childhood asthma in Puerto ricans. Ann Allergy Asthma Immunol. 2015;115:288–e2931.
52. Kim HY, Lee HJ, Chang Y-J, Pichavant M, Shore SA, Fitzgerald KA, et al. Interleukin-17-producing innate lymphoid cells and the NLRP3 inflammasome facilitate obesity-associated airway hyperreactivity. Nat Med. 2014;20:54–61.
53. Simpson JL, Grissell TV, Douwes J, Scott RJ, Boyle MJ, Gibson PG. Innate immune activation in neutrophilic asthma and bronchiectasis. Thorax. 2007;62:211–8.
54. Alwarith J, Kahleova H, Crosby L, Brooks A, Brandon L, Levin SM, et al. The role of nutrition in asthma prevention and treatment. Nutr Rev. 2020;78:928–38.
55. Vutcovici M, Brassard P, Bitton A. Inflammatory bowel disease and airway diseases. World J Gastroenterol. 2016;22:7735–41.
56. Adjibade M, Vigneron L, Delvert R, Adel-Patient K, Divaret-Chauveau A, Annesi-Maesano I et al. Characteristics of infant formula consumed in the first months of life and allergy in the EDEN mother-child cohort. Matern Child Nutr. 2024;:e13673.
57. Mellendick K, Shanahan L, Wideman L, Calkins S, Keane S, Lovelady C. Diets Rich in Fruits and vegetables are Associated with Lower Cardiovascular Disease Risk in adolescents. Nutrients. 2018;10:136.
58. Moreno-Fernández S, Garcés-Rimón M, Vera G, Astier J, Landrier JF, Miguel M. High Fat/High glucose Diet induces metabolic syndrome in an experimental rat model. Nutrients. 2018;10:1502.
59. Liu B-N, Liu X-T, Liang Z-H, Wang J-H. Gut microbiota in obesity. World J Gastroenterol. 2021;27:3837–50.

60. Morąg B, Kozubek P, Gomułka K. Obesity and selected allergic and immunological Diseases—Etiopathogenesis, Course and Management. Nutrients. 2023;15:3813.
61. Franssen FM. Obesity, airflow limitation, and respiratory symptoms: does it take three to tango? Prim Care Respir J. 2012;21:131–3.
62. Kunz HE, Hart CR, Gries KJ, Parvizi M, Laurenti M, Dalla Man C, et al. Adipose tissue macrophage populations and inflammation are associated with systemic inflammation and insulin resistance in obesity. Am J Physiol Endocrinol Metab. 2021;321:E105–21.
63. Canöz M, Erdenen F, Uzun H, Müderrisoglu C, Aydin S. The relationship of inflammatory cytokines with asthma and obesity. Clin Invest Med. 2008;31:E373–379.
64. Willett WC, Howe GR, Kushi LH. Adjustment for total energy intake in epidemiologic studies. Am J Clin Nutr. 1997;65 4 Suppl:1220S-1228S; discussion 1229S-1231S.
65. Thompson FE, Kirkpatrick SI, Subar AF, Reedy J, Schap TE, Wilson MM, et al. The National Cancer Institute's Dietary Assessment primer: a resource for Diet Research. J Acad Nutr Diet. 2015;115:1986–95.
66. Asthma. https://www.who.int/news-room/fact-sheets/detail/asthma. Accessed 18 Aug 2024.

Publisher's note