Journal of Health, Population and Nutrition

RESEARCH

Open Access

Association between the dietary inflammatory index and allergic rhinitis results from the National health and nutrition examination survey (2005–2006)

Hanchao Shen[1,2], Jie Liao[3], Lan Zhang[4], Peijia Li[1], Luyun Jiang[1], Tao Guo[1], Ya Yu[1] and Hui Xie[1,2*]

Abstract

Background Common chronic inflammatory condition known as allergic rhinitis(AR) has a major negative influence on people's health and puts a heavy strain on the world's healthcare systems. Despite the significant incidence of AR. This study aims to assess the potential link between the Dietary Inflammatory Index (DII) and the risk of developing AR.

Methods This study involved the analysis of data from 3,938 adult participants in the National Health and Nutrition Examination Survey (NHANES) conducted in 2005–2006. The DII score was used to evaluate the inflammatory potential of the participants' diets, and Multivariable logistic regression models were used to assess the association between DII (in tertiles) and having AR, adjusting for potential confounders. Subgroup analyses stratified by sex and Body Mass Index (BMI) were conducted to evaluate effect modification.

Results Our study demonstrated a positive correlation between the DII and the odds of AR prevalence. After adjusting for potential confounders, compared to individuals in the lowest tertile, those in the highest DII tertile had a 34% higher odds of AR prevalence.(OR 1.34, 95% CI 1.09–1.65). Furthermore, the subgroup analysis revealed a significant interaction ($P < 0.05$ for interaction) when stratified by sex and BMI.

Conclusion These results show that a higher DII score corresponds to the odds of AR prevalence, emphasizing the possible reduction of AR risk that can be achieved by eating a diet strong in anti-inflammatory nutrients and low in pro-inflammatory foods. This study emphasizes the role that dietary choices play in managing the risk of developing AR.

Clinical trial number Not applicable.

Keywords Allergens, Anti-inflammatory nutrients, Epidemiology, Inflammation, Dietary patterns, Cross-sectional study

*Correspondence:
Hui Xie
Wangxie-ctu@163.com
[1]Hospital of Chengdu University of Traditional Chinese Medicine, Chengdu 610032, China
[2]Chengdu University of Traditional Chinese Medicine, Chengdu 610075, China
[3]The TCM Hospital of Longquanyi, Chengdu 610100, China
[4]Jiangxi Provincial Traditional Chinese Medicine Hospital, Nanchang 330006, China

Introduction

AR is an escalating public health concern globally and has garnered increasing attention. Epidemiology shows significant geographical variation in the prevalence of allergic rhinitis, which affects 20% of adults in the UK [1]. In the United States, population-based studies have documented a prevalence of 15% [2], while nationally representative surveys in China report a lower prevalence of 8.1% [3]. The symptoms of AR include itching, nasal obstruction, excessive nasal discharge, and sneezing. This chronic nasal symptom may further lead to olfactory dysfunction, anxiety and depression, cognitive impairment, memory loss and other brain-related symptoms, which impose a great burden on patients' sleep and quality of life [4]. The economic burden and loss of productivity due to work absenteeism caused by the above symptoms are seriously underestimated [5].

Although the pathophysiology and treatment mechanisms of AR have been widely studied, the associated risk factors and protective factors are still not fully understood and need to be further investigated. Numerous studies have demonstrated that various dietary patterns contribute to the development of AR. From the "dietary hypothesis",nutrients and food components can significantly impact the pathogenesis of allergic diseases. They might achieve this by directly influencing the immune system and the pathways related to allergic inflammation, or diet can influence AR by modulating the composition of the gut microbiota, which may either promote or prevent the development of allergic conditions [6]. Lim et al. found that varying levels of dietary fat intake, from moderate to high, were linked to the development of AR [7]. The Mediterranean diet, known for its high content of vegetables and fruits, preference for seafood, moderate alcohol consumption, abundant use of olive oil, and low intake of red meat and butter, is frequently emphasized for its potential anti-inflammatory properties and has been shown to lower the incidence of asthma and other allergic conditions [8]. Another study revealed that high dairy product consumption was inversely associated with symptoms of AR [9]. The DII is a tool created to evaluate the inflammatory potential of a diet by giving each food item a score between + 1 and − 1. Scores on the positive end indicate a greater chance of promoting inflammation, Negative scores, on the other hand, suggest that inflammation may be reduced. A higher DII score denotes a dietary pattern that may make inflammatory conditions worse right now, whereas a lower score implies a dietary pattern that might aid in alleviating inflammation [10].

Emerging evidence has linked higher DII scores to an increased risk of various chronic conditions, such as cardiovascular disease and cancer. While the relationship between the Energy-Adjusted DII (E-DII) and AR has been studied in Asian populations [11]. However, there has been no research to date that investigates the link between the DII and AR in American adults, making it crucial to investigate this specific relationship. Identifying a dietary pattern associated with AR could offer new insights into preventive strategies through diet modification. Consequently, we analyzed a representative sample of adults from the database of NHANES.

Materials and methods

Survey description and study subjects

This study employed a cross-sectional design and utilized data from the NHANES database (https://www.cdc.gov/nchs/nhanes/index.htm) to obtain a nationally represent ative sample. Assessing the nutritional status and general health of people was the primary goal, thereby improving the external validity of the Study's finding. NHANES conducts extensive health-related interviews and examinations every two years to provide comprehensive data. The database includes a diverse range of samples from various regions, age groups, and ethnic backgrounds nationwide. The National Center for Health Statistics Institutional Review Board granted ethical approval for this study, and each participant provided informed permission.

The analysis focused on data from the 2005–2006 NHANES cycle, which uniquely included complete allergy-related information, encompassing a total of 10,348 participants. However,1,180 participants were excluded due to missing dietary data, and another 2,226 were excluded because of the absence of allergen-specific immunoglobulin E (sIgE) test results in serum samples. Additionally,2,717 children and 287 participants with missing covariates were excluded. Ultimately, the study included 3,938 adults aged 18 and older with available data on dietary intake and allergy-related outcomes. A systematic screening process flowchart is provided in Fig. 1.

Dietary inflammatory index

Utilizing a 24-hour dietary recall questionnaire, nutritional data was gathered in order to assess the individuals' dietary intake. This process typically involved two stages: The first dietary recall interview was conducted at a mobile examination center, followed by a second interview over the phone, which took place three to ten days later. We calculated the DII based on the average of the two dietary data. The DII was calculated according to the definition proposed by Shivappa [10], which mainly calculates the inflammatory score (in favor of inflammation or anti-inflammatory) of 45 nutrients. The specific calculation formula is: (daily intake of each dietary component - global average daily intake)/standard deviation (SD) of the global average daily intake of that specific dietary component the overall inflammatory effect score of that dietary component. Previous studies have found

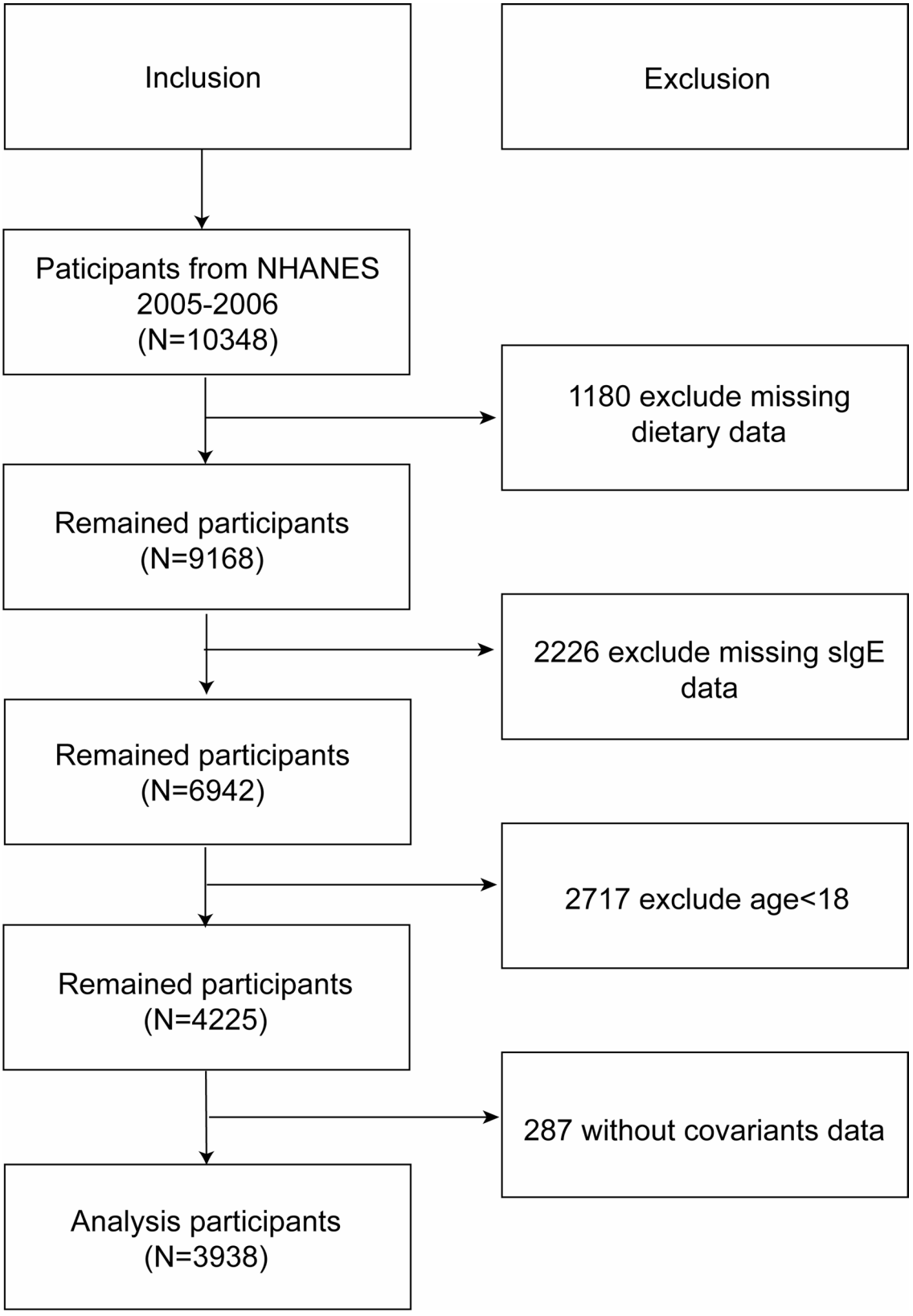

Fig. 1 Flow chart of study participant selection

that even though the NHANES program only records 26 foods, the DII score based on less than 30 foods still has reliable predictive ability [12]. In this study, we selected 26 nutrients from the DII calculation, including protein, carbohydrates, fiber, cholesterol, total fat, alcohol, caffeine, beta-carotene, vitamins A, C,E, monounsaturated fatty acids (MUFA), folic acid, iron, magnesium, zinc, selenium, niacin, omega-3 and omega-6 fatty acids, polyunsaturated fatty acids (PUFA), riboflavin, saturated fats, thiamine, and B6,B12.

Assessment of AR

The evaluation of AR-related results consists of two parts. The first step involves collecting blood samples at the mobile examination station. Serum antigen-specific IgE levels were measured using the Pharmacia Diagnostics ImmunoCAP 1000 system in Kalamazoo, MI. Allergens included D. farinae, D. pteronyssinus, Alternaria and Aspergillus, ragweed, ryegrass, Bermuda grass, oak, birch and thistle, cat, dog, mouse, cockroach, as well as peanut, egg, milk and shrimp. Serum sIgE was considered positive if the sIgE result for at least one allergen was ≥0.35 kU/L. The second consisted of two questionnaire questions: (1) "Have you had hay fever in the past 12 months? (2) "Have you had problems with sneezing, a runny or blocked nose in the past 12 months without having a cold or flu? Participants who tested positive for serum sIgE and responded "yes" to either of the two related questions were classified as having AR. Those who did not meet these criteria were classified as having non-AR [13].

Covariate assessment

In constructing the model, the researchers selected covariates based on a preliminary screening of variables from the NHANES database and a thorough review of the literature from previous studies: demographic variables such as age, gender, race, BMI, and income-to-poverty ratio(PIR); lifestyle variables, comprising one's smoking status (defined as having smoked more than 100 cigarettes in one's lifetime), diabetes, hypertension, and physical activity. Detailed procedures for measuring these variables are available on the NHANES website.

Covariate inclusion criteria

Hypertension

If a participant's diastolic blood pressure was ≥90 mmHg or their systolic blood pressure was greater than 140 mmHg, and they self-reported having hypertension, or they were taking antihypertensive medication they were considered hypertensive [14].

Diabetes

Diabetes was identified based on self-reported diabetes, a HbA1c level of more than 6.5%, a fasting blood glucose level of ≥7.0 mmol/L, or the use of antidiabetic medication [15].

Body mass index

BMI is calculated as follows: kg/m^2, or weight in kilograms divided by height in meters squared. Participants were split into three groups according to their BMI: underweight or normal weight (BMI less than 25 kg/m^2), overweight ($25 < BMI \leq 30$ kg/m^2), and obese (BMI more than 30 kg/m^2).

Statistical analysis

In the descriptive analysis, data characterization and statistical evaluation relied on intricate weighting. To determine statistical significance between the AR and non-AR groups, T-tests, Wilcoxon rank sum tests, or Chi-square tests were conducted as appropriate. Percentages were used to represent categorical data, while averages with standard deviations (SD) were used to depict continuous variables. Through multiple logistic regression analysis, the link between DII tertiles and AR was evaluated using odds ratios (OR) and 95% confidence intervals (CI), with trend *P* values computed for the various tertiles. Three regression models were constructed to control potential confounding variables. Model 1 did not take any confounding factors into account, while Model 2 adjusted for gender, age, and race; and model 3 further adjusted for PIR, BMI, smoking, hypertension, diabetes, and physical activity on the basis of model 2. A multivariate regression model with stratification by gender, age, race, household income-poverty ratio, BMI, smoking, hypertension, diabetes, and physical activity was used to conduct subgroup analysis and interaction testing. To further explore the nature of this relationship, smoothing curve fitting was employed to determine if the relationship between DII and AR was linear. For all statistical studies, Empowerstats(X&Y Solutions, Inc., Boston, MA, USA) were utilized [16, 17], and statistical significance was defined as $P < 0.05$.

Results

Study population description

The study included 3938 eligible participants, who were divided into an AR group (702) and a non-AR group (3236). In terms of all individuals(Table 1), the AR group was younger, had a higher PIR, higher IgE, and a higher proportion of smokers($P < 0.05$). It is noteworthy that the DII score was lower in AR patients than in non-AR patients ($P < 0.05$). Although the mean DII score was lower in the AR group in unadjusted analyses, after adjusting for potential confounders, higher DII

Table 1 Characteristics of eligible adults

Characteristics	AR(*n* = 702)	Without AR(*n* = 3236)	*P* value
AGE(years)	43.61 ± 15.13	47.06 ± 16.85	< 0.0001
PIR	3.29 ± 1.60	3.12 ± 1.56	0.0074
BMI, kg/m^2	28.93 ± 7.04	28.47 ± 6.63	0.0875
IgE, IU/mL	237.25 ± 464.90	104.95 ± 334.47	0.0157
DII(SE)	1.24 ± 1.75	1.41 ± 1.75	0.0049
Gender(%)			0.1057
Male	50.57	47.32	
Female	49.43	52.68	
RACE(%)			0.2478
Mexican American	5.87	8.27	
Other Hispanic	3.84	3.36	
Non-Hispanic White	74.55	72.42	
Non-Hispanic Black	10.68	10.80	
Other Race - Including Multi-Racial	5.06	5.15	
Hypertention(%)			0.1386
YES	16.59	18.90	
NO	83.41	81.10	
Diabetes(%)			0.8013
YES	7.74	8.02	
NO	92.26	91.98	
Physical activity(%)			0.1305
YES	25.08	22.53	
NO	74.92	77.47	
Smoked at least 100 cigarettes in life(%)			< 0.0001
YES	42.35	50.72	
NO	57.65	49.28	

Table 2 Association of dietary inflammatory index and AR

	Model I, OR(95%CI)	Model II, OR(95%CI)	Model III, OR(95%CI)
DII	1.07 (1.03, 1.12)	1.08 (1.03, 1.13)	1.08 (1.03, 1.13)
T1(≤ 0.850)	Reference	Reference	Reference
T2 (0.850–2.453)	1.17 (0.96, 1.43)	1.18 (0.97, 1.44)	1.19 (0.98, 1.46)
T3(≥ 2.454)	1.33 (1.09, 1.63)	1.35 (1.09, 1.65)	1.34 (1.09, 1.65)
P for trend	0.0049	0.0045	0.0046

Model 1: unadjusted for variables; Model II: adjusted for age, sex, race; Model III: adjusted for age, sex, race, PIR, BMI, hypertension, diabetes, physical activity, smoking

values were associated with increased odds of AR(T3: OR = 1.34)(Table 2).

Association of DII and AR

As presented in Table 2, we conducted multivariate logistic regression analyses using DII as a categorical variable (tertiles) to examine the relationship between DII and (AR). The results showed a positive link between DII and the odds of AR prevalence, a result that was also stable when DII was a continuous value. In Model I (unadjusted), there was no statistical significance in the T2 group compared with participants in T1 tertiles (T2: OR = 1.17, 95%CI: 0.96, 1.43), whereas in the T3 group, the incidence of AR increased by 33% for each unit increase in DII (T3: OR = 1.33, 95%CI:1.09, 1.63; *P* for trend = 0.0049). In Model II, there was no statistically significant difference in the T2 group (T2: OR = 1.18, 95%CI:0.97, 1.44) compared to the first tertiles group (T1 group), while the T3 group had a higher odds of AR prevalence (T3: OR = 1.35, 95%CI:1.09, 1.65; *P* for trend = 0.0045). In the fully adjusted model (Model III), each unit increase in DII in the T3 group was associated with a 34% increase in the incidence of AR compared with participants in the T1 tertiles (T3: OR = 1.34, 95%CI:1.09, 1.65; *P* for trend = 0.0046).

Subgroup analysis

To assess whether the relationship between DII and AR prevalence is consistent across different populations, we performed subgroup analyses and interaction tests by age, sex, race, BMI, PIR, hypertension, diabetes, physical activity, and smoking. The results in Fig. 2 show that AR

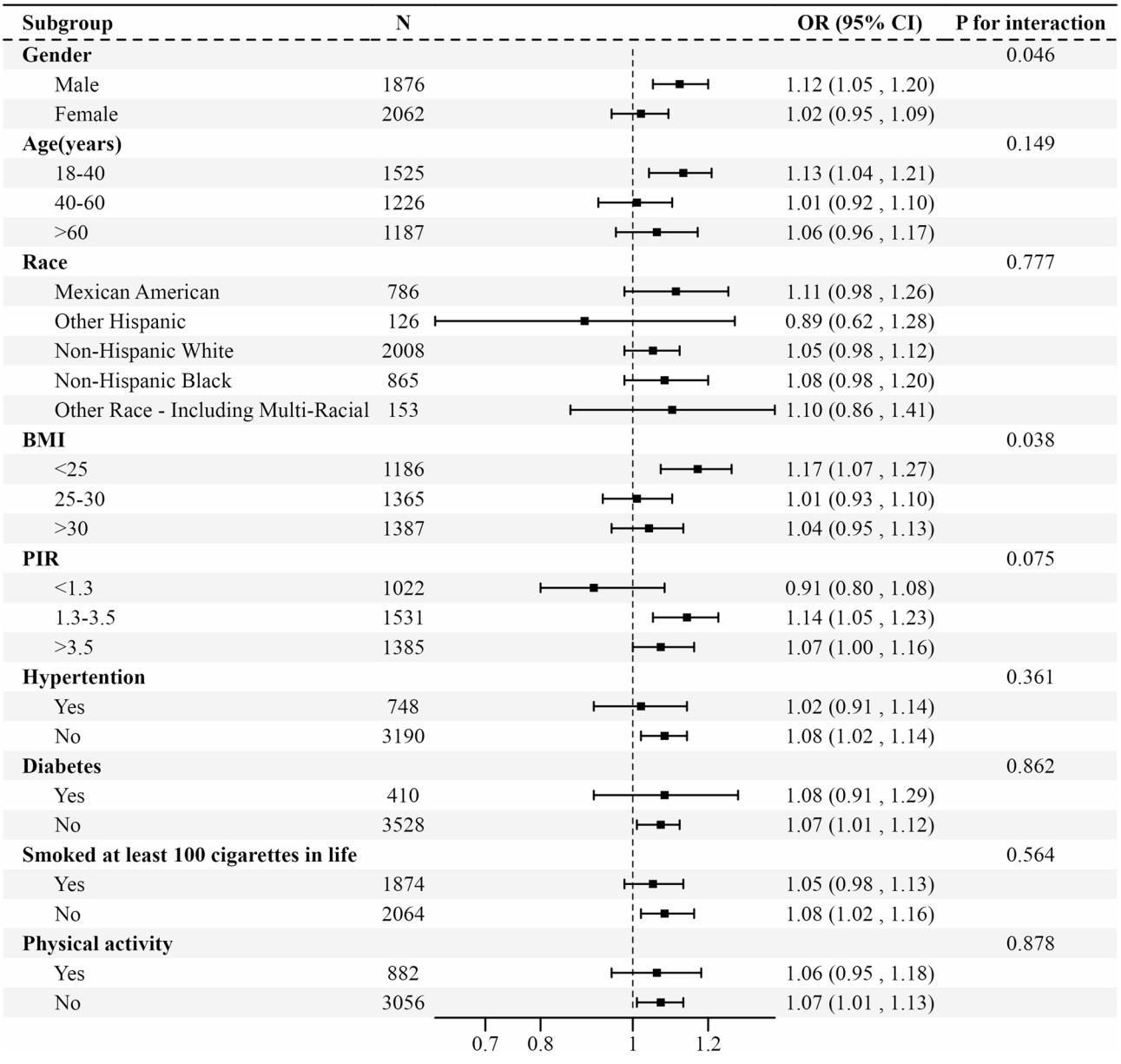

Fig. 2 Subgroup analysis[1]

[1] Age, gender, race, PIR, BMI, hypertension, diabetes, physical activity, and smoking were adjusted

was positively associated with DII in all groups except for Hispanics and those with a PIR < 1, where AR was negatively associated with DII. Subgroup analysis by gender showed that men were more sensitive to dietary inflammation than women (p for interaction = 0.046). We also found that the odds of AR prevalence was more closely associated with normal weight than with underweight, overweight, and obesity (p for interaction < 0.01).

Curve fitting analysis

The curve fitting analysis (Fig. 3) supports the positive relationship between DII and AR, with the odds of AR prevalence increasing as the DII score increases.

Discussion

This study included 3938 participants from the 2005–2006 NHANES and investigated the relationship between DII and AR. Our findings showed that AR was statistically associated with younger age, higher PIR, higher IgE levels, and a higher proportion of smokers. A significant correlation between DII and AR was found using

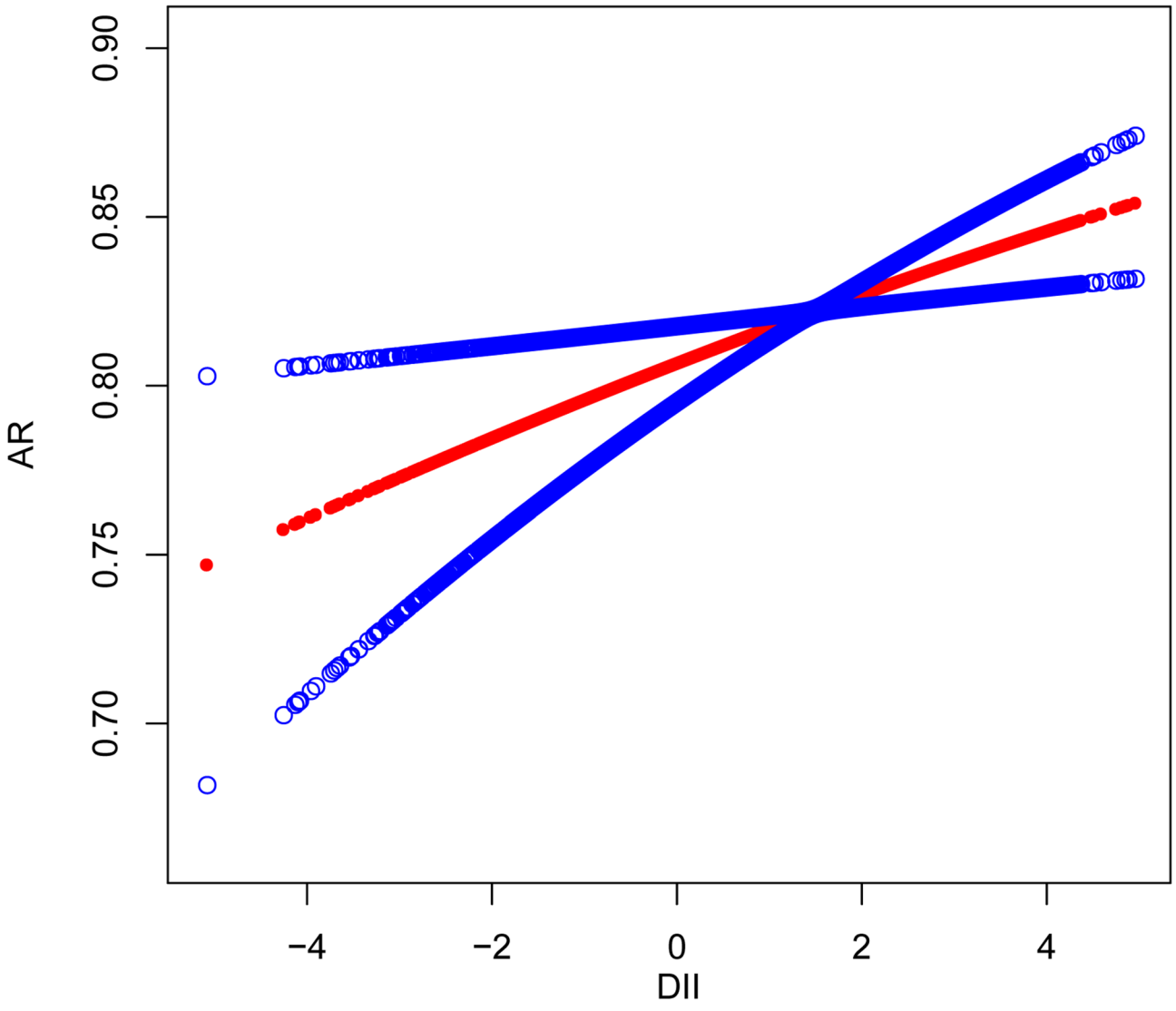

Fig. 3 Curve fitting analysis[1]

[1] Model 3-based link between DII levels and AR. The solid red line shows the smoothed fitted curve between DII levels and AR, and the blue dashed line shows the fitted 95% confidence intervals. Each red dot represents a separate sample, and the blue dots above and below the red dots represent the 95% confidence intervals

multivariate logistic regression and smoothing curve fitting. To further go into the details of the link between DII and AR, additional subgroup analyses were carried out, which consistently reaffirmed our findings. According to research, eating foods that promote inflammation increases the odds of AR prevalence. This aligns with the conclusions drawn by Wang and colleagues [11], who proposed that adopting a diet rich in anti-inflammatory nutrients while minimizing pro-inflammatory options can be an effective approach to preventing AR. In particular, the odds of AR prevalence was positively associated with DII in the following subgroups: male, 18–40 years old, BMI < 25, 1.3 ≤ PIR < 3.5, no hypertension, no diabetes, smoking less than 100 cigarettes in a lifetime, and no physical activity.

The findings from the subgroup analysis that took gender into account revealed a positive correlation between the prevalence of AR and the DII across all demographics. Furthermore, the results indicated that men exhibited a greater sensitivity to dietary inflammation compared to their female counterparts is consistent with the results of two investigations carried out in South Korea [18, 19] and a cross-sectional research carried out in Japan [20]. This could stem from gender-specific genetic influences alongside exposure to environmental allergens. These factors might help explain the disparities in the prevalence of allergic diseases between the sexes, as there are

likely variations in how men and women respond to different allergens through their inflammatory pathways [18]. In addition, the underlying mechanism for differences in allergy presentation between women and men may be the result of sex-specific genetic differences associated with the X chromosome and the effects of sex hormones on immune system response regulation and inflammation [21]. Therefore, further research is needed to investigate the role of gender differences in the occurrence of AR.

We might speculate that an association between BMI and the effect of DII on AR exists based on this study, and the association between them is more significant in the normal BMI group ($P<0.001$) than in the obese group. This aligns with the findings of a research conducted in Japan [19]. In contrast to these results, several studies [22–24]have shown that AR is associated with an increased body mass index (BMI) and that obesity has various effects on the immune system. New evidence suggests that BMI influences the occurrence and development of AR through multiple pathways, including chronic inflammation, metabolism-immune axis disorders, hormone level changes, and genetic susceptibility [25–28]. To properly understand the role of DII in AR, more research is needed, taking into account the different effects of BMI in men and women. However, we believe that further prospective and mechanistic studies are needed to better understand this important topic.

There are numerous ways that diet might impact allergy illnesses, and most of the research done in the past has focused on the contribution of particular food ingredients to the onset of allergic reactions. An increasing number of cellular and molecular mechanisms explaining the regulation of allergic inflammation by individual food ingredients or particular nutrients have been revealed. Certain allergic nutrients, like saturated fatty acids, trigger epithelial and stromal cells to secrete TSLP, IL-25, and IL-33, which, in turn, stimulate ILC2 cells to generate cytokines such as IL-4, IL-5, IL-9, and IL-13, thereby creating a cytokine environment that is conducive to allergic inflammation [29]. Unsaturated fatty acids mainly include monounsaturated fatty acids (MUFAs) and polyunsaturated fatty acids (PUFAs), of which PUFAs contain many essential fatty acids that the human body cannot synthesize. These fatty acids have anti-inflammatory and triglyceridemic effects, while their dietary counterparts, n-6 PUFAs, have pro-inflammatory properties. These fatty acids can produce a variety of biological effects by changing the composition of cell membranes, regulating cell signal transcription and conduction, and other pathways [30]. A high intake of n-6 PUFAs and a low intake of n-3 PUFAs may lead to a higher risk of inflammation [31]. Miyake et al. reported that the direction of the association between n-6 PUFAs and allergic diseases may change depending on the time of exposure: from the womb to childhood there may be an inverse association, whereas in adulthood there may be a positive association [32]. Another study found that n-3 PUFAs help to create a favorable environment for immune maturation and may help to prevent allergies [33]. The antioxidant, anti-inflammatory and immunomodulatory effects of vitamins and minerals are well known, and important dietary components that affect the development of allergic inflammation and allergic disorders include the trace minerals zinc and iron, as well as the vitamins A, D, and E [29]. To reduce oxidative damage, vitamins A and C can exert an antioxidant defense during allergic inflammatory reactions [34]. Vitamin E and selenium not only reduce the production of IL-13, but also affect the immune response to AR by reducing serum IgE and histamine [35]. Compared to normal zinc intake, zinc deficiency leads to increased airway hyperresponsiveness, while zinc supplementation reduces inflammatory cell infiltration and has a positive effect on regulating the immune system and reducing allergy symptoms [36, 37]. Dietary fiber helps maintain a tolerogenic mucosal environment, promotes epithelial barrier function, induces T regulatory cells, prevents TH2 polarization and inhibits mast cell degranulation, and may prevent allergic diseases [38]. In addition, Chang and colleagues [39]found that the intake of probiotics reduced the incidence of AR, especially in men and in people aged 65 and ≥ 80 years. In addition, polyphenols such as resveratrol and curcumin and cinnamon have been shown to be beneficial for AR symptoms and the reduction of inflammatory cells. In particular, oral curcumin has been suggested as a potential substance for reducing allergic symptoms in AR [40]. According to these results, taking supplements of unsaturated fatty acids, certain vitamins, trace minerals, and phytochemicals may help avoid or lessen the likelihood that AR will become prevalent.

It has also been demonstrated that dietary patterns are linked to the development of AR, for example, high-fat diets are considered to be detrimental to health, and over-nutrition alters the gut microbiota, leading to activation of pro-inflammatory pathways, increased intestinal permeability, and systemic inflammation [41], Numerous studies indicate that diets rich in fats could heighten the likelihood of developing AR. It is noteworthy that an essential component of a healthy diet is consuming a lot of extra virgin (cold-pressed) olive oil, along with fruits, grains, nuts, legumes, and vegetables, especially leafy greens [7, 11, 42, 43]. Moreover, moderate portions of fish, meats, dairy, and red wine are also recommended, as well as a low intake of eggs and sweets, Mediterranean-style diets contain a high concentration of phytochemicals, which have been shown to have positive biological effects, such as anti-aging,

immunomodulatory, antioxidant, and anti-inflammatory properties [44]. In contrast to Western diets that are abundant in pro-inflammatory elements, the Mediterranean diet is enhanced by plant-based meals linked to strong anti-inflammatory and anti-allergic benefits [29].

As far as we know, this is the first significant cohort study examining the link between AR and DII scores in a predominantly non-Asian US population. Because we used a nationally representative sample, our findings are highly relevant to the general population. But we also need to be aware of this study's shortcomings. First off, since this study's subjects were adults, it's possible that the findings cannot be applied to smaller age groups. Second, it is challenging to establish a causal link between DII and AR because our study was cross-sectional. What's more, The sample size for this study was based on available data from the NHANES database, and no prospective sample size calculations were made. Future studies ought to focus on confirming these findings through comprehensive, forward-looking cohort research to strengthen the link between dietary inflammation and the risk of AR. Finally, we defined participants as AR and non-AR based on serum test results and questionnaire survey results. Because the questionnaire survey is subjective, it is inevitably biased.

Conclusion

According to the study's findings, there is a tiny increase in the odds of AR prevalence with a slightly higher DII score, or a pro-inflammatory diet, but not a significant rise with a lower score, or an anti-inflammatory diet. Reducing the amount of foods high in inflammatory compounds may help lower the odds of AR prevalence. Therefore, managing the consumption of pro-inflammatory foods could potentially diminish the prevalence of AR, suggesting that dietary changes might be an effective strategy for its prevention and management. These elements encompass gender and body mass index, both of which could serve as significant risk factors for AR. Therefore, we recommend that a DII assessment be carried out on AR high-risk groups. These findings serve as a wake-up call, indicating that a diet rich in inflammatory foods might heighten the likelihood of AR's occurrence, though further studies are necessary to validate this conclusion. However, this study provides important perspectives on how to prevent AR and its related health effects.

Author contributions

H.S and H.X equally contributed to the conception and design of the research; J.L and L.Z contributed to the design of the research; P.L and L.J contributed to the acquisition and analysis of the data; T.G and Y.Y contributed to the interpretation of the data; and H.S drafted the manuscript. All authors critically revised the manuscript, agree to be fully accountable for ensuring the integrity and accuracy of the work, and read and approved the final manuscript.

Funding

No specific funding for this work.

Data availability

All data analyzed in the current study are freely accessible on the NHANES website (https://www.cdc.gov/nchs/nhanes/).

Declarations

Ethical approval and consent to participate

Data collection for the NHANES was approved by the NCHS Research Ethics Review Board (ERB). An individual investigator utilizing the publicly available NHANES data do not need to file the institution internal review board (IRB).

Competing interests

The authors declare no competing interests.

Received: 13 March 2025 / Accepted: 19 May 2025
Published online: 29 May 2025

Refrences

1. Siddiqui ZA, Walker A, Pirwani MM, Tahiri M, Syed I. Allergic rhinitis: diagnosis and management. Br J Hosp Med (Lond). 2022;83(2):1–9.
2. Bernstein JA, Bernstein JS, Makol R, Ward S. Allergic rhinitis: A review. JAMA. 2024;331(10):866–77.
3. Zhang X, Zhang M, Sui H, Li C, Huang Z, Liu B, Song X, Liao S, Yu M, Luan T, et al. Prevalence and risk factors of allergic rhinitis among Chinese adults: A nationwide representative cross-sectional study. World Allergy Organ J. 2023;16(3):100744.
4. Wang Y, Song XY, Wei SZ, Wang HR, Zhang WB, Li YM, Mou YK, Ren C, Song XC. Brain response in allergic rhinitis: profile and proposal. J Neurosci Res. 2023;101(4):480–91.
5. Patel KB, Mims JW, Clinger JD. The burden of asthma and allergic rhinitis: epidemiology and health care costs. Otolaryngol Clin North Am. 2024;57(2):179–89.
6. Dębińska A, Sozańska B. Dietary Polyphenols-Natural bioactive compounds with potential for preventing and treating some allergic conditions. Nutrients 2023;15(22).
7. Lim JJ, Reginald K, Say YH, Liu MH, Chew FT. A dietary pattern for high estimated total fat amount is associated with enhanced allergy sensitization and atopic diseases among Singapore/Malaysia young Chinese adults. Int Arch Allergy Immunol. 2023;184(10):975–84.
8. Vassilopoulou E, Guibas GV, Papadopoulos NG. Mediterranean-Type diets as a protective factor for asthma and atopy. Nutrients 2022;14(9).
9. Antonogeorgos G, Priftis KN, Panagiotakos DB, Ellwood P, García-Marcos L, Liakou E, Koutsokera A, Drakontaeidis P, Moriki D, Thanasia M et al. Exploring the relation between atopic diseases and lifestyle patterns among adolescents living in Greece: evidence from the Greek global asthma network (GAN) Cross-Sectional study. Child (Basel) 2021;8(10).
10. Shivappa N, Steck SE, Hurley TG, Hussey JR, Hébert JR. Designing and developing a literature-derived, population-based dietary inflammatory index. Public Health Nutr. 2014;17(8):1689–96.
11. Wang Q, Dong N, Feng Y, Ning Y, Zhu R, Han S. The association between the dietary inflammatory index and allergic rhinitis: a case-control study. Front Nutr. 2024;11:1418305.
12. Liu X, Chen TY, Gao TY, Shi KQ, Yin FQ, Yu YX, Zhang C. Pro-inflammatory diets promote the formation of hyperuricemia. Front Endocrinol (Lausanne). 2024;15:1398917.
13. Jiao WE, Xi Y, Li D, Xu S, Kong YG, Deng YQ, Yang R, Tao ZZ, Hua QQ, Chen SM. Association of dietary polyunsaturated fatty acid intake with allergic rhinitis in adults: A Cross-Sectional study of NHANES 2005–2006. Int Arch Allergy Immunol. 2024;185(2):124–32.
14. Cheng X, Ma T, Ouyang F, Zhang G, Bai Y. Trends in the prevalence of cardiometabolic Multimorbidity in the united States, 1999–2018. Int J Environ Res Public Health 2022;19(8).
15. Association AD. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2010;33(Supplement1):S62–9.

16. Huang J, Rozi R, Ma J, Fu B, Lu Z, Liu J, Ding Y. Association between higher triglyceride glucose index and increased risk of osteoarthritis: data from NHANES 2015–2020. BMC Public Health. 2024;24(1):758.
17. Zhang Y, Liu M, Xie R. Associations between cadmium exposure and whole-body aging: mediation analysis in the NHANES. BMC Public Health. 2023;23(1):1675.
18. Acharya D, Bajgain BB, Yoo SJ. Factors associated with atopic dermatitis and allergic rhinitis among residents of two municipal areas in South Korea. Med (Kaunas) 2019;55(5).
19. Matsumoto M, Konno S, Kimura H, Takei N, Kimura H, Shimizu K, Goudarzi H, Suzuki M, Hashino S, Nishimura M. Associations of current wheeze and body mass index with perennial and seasonal allergic rhinitis in young adults. Int Arch Allergy Immunol. 2018;176(2):143–9.
20. Nishijima H, Suzuki S, Kondo K, Yamasoba T, Yanagimoto S. Environmental factors associated with allergic rhinitis symptoms in Japanese university students: A cross-sectional study. Auris Nasus Larynx. 2018;45(5):1006–13.
21. De Carli M, Capezzali E, Tonon S, Frossi B. Mechanism and clinical evidence of immunotherapy in allergic rhinitis. Front Allergy. 2023;4:1217388.
22. Ciprandi G, Ricciardolo FL, Signori A, Schiavetti I, Monardo M, Ferraro MR, Cirillo I. Increased body mass index and bronchial impairment in allergic rhinitis. Am J Rhinol Allergy. 2013;27(6):e195–201.
23. Morąg B, Kozubek P, Gomułka K. Obesity and selected allergic and immunological Diseases-Etiopathogenesis, course and management. Nutrients 2023, 15(17).
24. Harugop AS, Walia A, Havaldar RR, Mudhol RS. Correlation between allergic rhinitis and body mass index: an observational study. Indian J Otolaryngol Head Neck Surg. 2022;74(Suppl 2):1033–6.
25. Yeo BSY, Guan EJ, Ng K, Lim YS, Goh RTH, Liu X, Phua CQ, Tay K, Png LH, Xu S, et al. Association of abnormal body weight and allergic Rhinitis-A systematic review and Meta-Analysis. Clin Exp Allergy. 2025;55(2):142–65.
26. de Sá Pittondo M, Migueis DP, Fujita RR, Thamboo A, Tepedino MS, Pezato R. Effect of body weight on response to nasal glucocorticoid treatment in allergic rhinitis. Indian J Otolaryngol Head Neck Surg. 2024;76(1):1002–9.
27. Liu J, Ma T, Wang X, Bai W, Wang X. Associations between HT, BMI, and allergic rhinitis in perimenopausal women. Allergy Asthma Clin Immunol. 2023;19(1):107.
28. Lin C, Li J, Deng Y, Li X, Li S. Effect of obesity, lipids and adipokines on allergic rhinitis risk: a Mendelian randomization study. Braz J Otorhinolaryngol. 2023;89(5):101306.
29. Zhang P. The role of diet and nutrition in allergic diseases. Nutrients 2023;15(17).
30. D'Angelo S, Motti ML, Meccariello R. ω-3 and ω-6 polyunsaturated fatty acids, obesity and Cancer. Nutrients 2020;12(9).
31. Magnusson J, Kull I, Westman M, Håkansson N, Wolk A, Melén E, Wickman M, Bergström A. Fish and polyunsaturated fat intake and development of allergic and nonallergic rhinitis. J Allergy Clin Immunol. 2015;136(5):1247–e12531241.
32. Miyake Y, Tanaka K, Sasaki S, Arakawa M. Polyunsaturated fatty acid intake and prevalence of eczema and rhinoconjunctivitis in Japanese children: the Ryukyus child health study. BMC Public Health. 2011;11:358.
33. Willemsen LEM. Dietary n-3 long chain polyunsaturated fatty acids in allergy prevention and asthma treatment. Eur J Pharmacol. 2016;785:174–86.
34. Barrera-Mendoza CC, Ayala-Mata F, Cortés-Rojo C, García-Pérez ME. Rodríguez-Orozco AR: [Antioxidant vitamins in asthma]. Rev Alerg Mex. 2018;65(1):61–77.
35. Jiang J, Mehrabi Nasab E, Athari SM, Athari SS. Effects of vitamin E and selenium on allergic rhinitis and asthma pathophysiology. Respir Physiol Neurobiol. 2021;286:103614.
36. Truong-Tran AQ, Ruffin RE, Foster PS, Koskinen AM, Coyle P, Philcox JC, Rofe AM, Zalewski PD. Altered zinc homeostasis and caspase-3 activity in murine allergic airway inflammation. Am J Respir Cell Mol Biol. 2002;27(3):286–96.
37. Maywald M, Rink L. Zinc deficiency and zinc supplementation in allergic diseases. Biomolecules 2024;14(7).
38. Venter C, Meyer RW, Greenhawt M, Pali-Schöll I, Nwaru B, Roduit C, Untersmayr E, Adel-Patient K, Agache I, Agostoni C, et al. Role of dietary fiber in promoting immune health-An EAACI position paper. Allergy. 2022;77(11):3185–98.
39. Chang C, Wang Q, Li X, Tan H, Huang G. The relationship between prebiotic intake and allergic rhinitis. Laryngoscope Investig Otolaryngol. 2023;8(5):1146–53.
40. Kaag S, Lorentz A. Effects of dietary components on mast cells: possible use as nutraceuticals for allergies?? Cells 2023;12(22).
41. Bojková B, Winklewski PJ, Wszedybyl-Winklewska M. Dietary fat and Cancer-Which is good, which is bad, and the body of evidence. Int J Mol Sci 2020;21(11).
42. Kim SY, Sim S, Park B, Kim JH, Choi HG. High-Fat and Low-Carbohydrate diets are associated with allergic rhinitis but not asthma or atopic dermatitis in children. PLoS ONE. 2016;11(2):e0150202.
43. Lin YP, Kao YC, Pan WH, Yang YH, Chen YC, Lee YL. Associations between respiratory diseases and dietary patterns derived by factor analysis and reduced rank regression. Ann Nutr Metab. 2016;68(4):306–14.
44. Nani A, Murtaza B, Sayed Khan A, Khan NA, Hichami A. Antioxidant and Anti-Inflammatory potential of polyphenols contained in mediterranean diet in obesity: molecular mechanisms. Molecules 2021;26(4).

Publisher's note