BMC Pediatrics

RESEARCH

Open Access

The relationship between energy intake and asthma in Americans aged 1–18 years: a cross-sectional study

Lin jun Du[1†], Chao Che[2†], Qian Liu[3], Xiaolan Zhang[1], Ning Feng[1], Lifang Chen[1] and Lili Wang[4*]

Abstract

Objective The objective of this study was to investigate the effects of total dietary energy intake on asthma.

Study selection.

The study was a retrospective cross-sectional study of Americans aged 1–18 year. Comprehensive demographic, dietary, examination, laboratory, and asthma questionnaire data were collected for each participant. Multivariate logistic regression, restricted triple spline curves, threshold effects, and stratified analyses were used for analysis.

Results Of 12,090 participants, 1,893 (15.66%) had a diagnosis of asthma. After accounting for potential confounders, compared with the group with the lowest energy intake (Q1), groups 2 (Q2), groups 3 (Q3), and groups 4 (Q4) had adjusted odds ratios (ORs) of 0.72 (0.62–0.85), 0.63 (0.51–0.77) and 0.55 (0.43–0.7) for asthma. The relationship between total energy intake and asthma showed an L-shaped curve ($p = 0.001$). The results were further verified by stratification and sensitivity analyses. In the threshold analysis, we found that the saturation effect was reached at a total energy intake of 56.442 kcal/kg/day with an OR of 0.981 (0.973–0.989).

Conclusion The prevalence of asthma in Americans aged 1–18 years was associated with total dietary energy intake in an L-shaped curve, with a significant turning point found at approximately 56.442 kcal/kg/day.

Keywords L-shaped relationship, Cross-sectional study, Asthma, Children, Diet, Energy intake

Introduction

Asthma is one of the most common chronic childhood diseases, characterized by recurrent attacks and reversible bronchial obstruction [1]. The Centers for Disease Control and Prevention (CDC) estimated that approximately 25 million people in the United States had asthma in 2021, of which 4.67 million were children under the age of 18 [2], placing a heavy economic burden on the healthcare system and society [3]. A growing body of evidence suggests that diet plays an important role in both chronic and allergic diseases [4].

Numerous factors contribute to the development of asthma. Past research has demonstrated a negative correlation between asthma-related outcomes and the consumption of fish and whole grain products.[5]On the other hand, children in developed nations consume a diverse range of foods that include intricate combinations of nutrients that could potentially interact or work in concert. Consequently, examining dietary patterns rather than individual food associations may yield more

†Lin jun Du and Chao Che are the co-first authors.

*Correspondence:
Lili Wang
wll15315797270@163.com
[1] The Third People's Hospital of Liaocheng City, Liaocheng, Shandong Province, China
[2] Qilu Hospital of Shandong University, Jinan, Shandong, China
[3] Jinan Fifth People's Hospital, Jinan, Shandong Province, China
[4] Maternal and Child Health Center, Chiping District, Liaocheng City, Shandong Province, China

accurate predictions of disease risk. The Western diet and the Mediterranean diet have received the most attention in studies on the general health impacts of childhood nutrition [6]. The prevalence of wheeze, a high-pitched, musical, adventitious lung sound produced by airflow through an abnormally narrowed or compressed airway, appears to be negatively correlated with a children Mediterranean diet, according to systematic reviews and meta-analyses of observational research [7]. In contrast, children who consume a Western diet may be more susceptible to respiratory ailments [8]. This correlation could be partially explained by obesity resulting from a high-energy diet, which has been shown to play a significant role in the higher incidence of asthma [9]. However, research on the connection between calorie intake and asthma is still scarce.

Therefore, we conducted a cross-sectional investigation for which the National Health and Nutrition Examination Survey (NHANES) provided a representative sample. The purpose of our study was to investigate the relationship between total energy intake and the risk of developing asthma in Americans aged 1–18 years and to assess the dose–response relationship between total energy intake and the risk of developing asthma in this population. This study contributes to our understanding of asthma management and prevention.

Materials and methods

Data sources and study population

The NHANES provided data collected between 2009 and 2018 for this cross-sectional investigation [10]. NHANES is a multistage probability survey that evaluates the health and nutritional status of the civilian population in the United States [11]. It is conducted by the National Center for Health Statistics (NCHS), a branch of the U.S. Centers for Disease Control and Prevention. Comprehensive health and demographic data were gathered through household-based healthcare, socioeconomic, and demographic interviews, followed by physical examinations, interviews, and laboratory evaluations of mobile examination centers (MECs). All participants were provided written informed consent prior to participation in the study, which followed the ethical guidelines established by the National Center for Health Statistics (NCHS) Ethics Review Committee [12]. Additional institutional review board approval was not required for this secondary analysis [12]. The official NHANES website (http://www.cdc.gov/nchs/nhanes.htm) will be accessible with the study's data starting March 1, 2024. All study participants were aged 1–18 years. The study excluded participants with missing body weight, total energy consumption, or asthma data. Figure 1 illustrates the exclusion procedure.

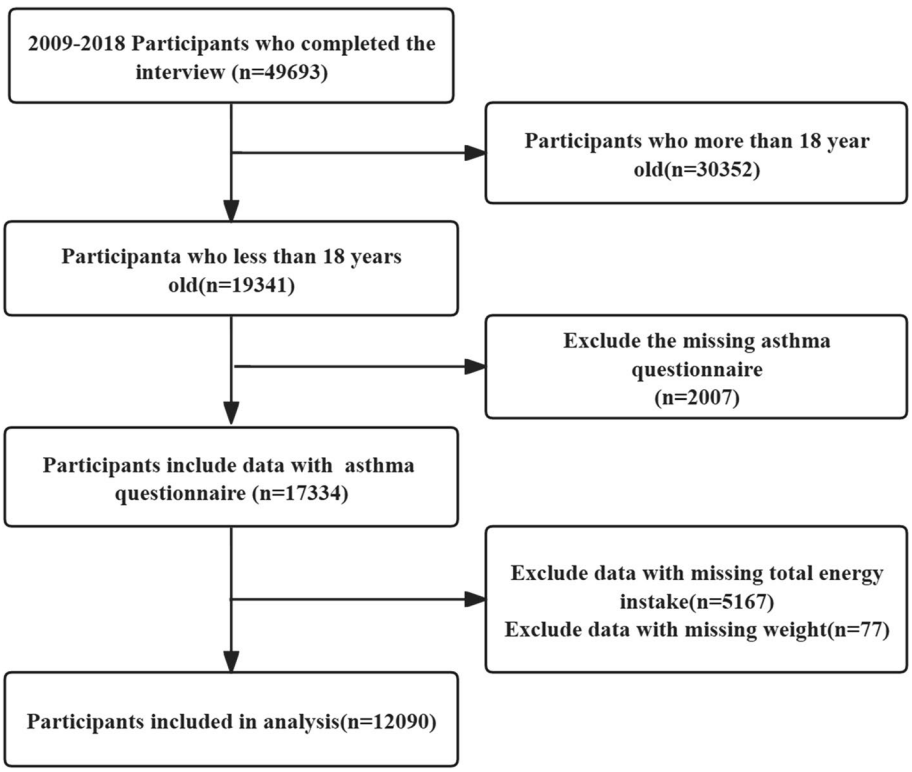


Fig. 1 Study's flow diagram

Ultimately, 12,090 participants were included in the analysis.

Asthma

By answering the question, "Has a doctor or other health professional ever told you that you have asthma?" we determined whether the participants had asthma. A participant's response of "yes" indicated he or she had asthma, whereas his response of "no" indicated he or she did not [13].

Total energy

A completely computerized recall system with standardized questions and alternative answers was used to obtain the NHANES dietary intake data. The system combined an automated multiple-pass method (AMPM) and a personal food intake survey (CSFII) to obtain precise nutritional values for each participant [11]. The Dietary Survey's methodology is thoroughly explained in the NHANES Dietary Survey Manual [14]. We determined total energy intake (kcal/kg/day) based on data from two 24-h dietary recall interviews, corrected for body weight based on previous literature [13]. Using total energy consumption as a guide, participants were divided into four groups, with the first group (Q1) representing an energy intake below the 25th percentile. Participants in the second group (Q2) had intakes that fell between the 25th and 50th percentiles. Those with intakes between the 50th and 75th percentiles were placed in the third group (Q3), and those with intakes beyond the 75th percentiles were placed in the fourth group (Q4).

Covariates

Based on previous research [15], several potential covariates were evaluated, including age, sex, race/ethnicity, BMI category (BMIC), poverty-to-income ratio (PIR), maternal smoking during pregnancy, maternal age at delivery, birth weight, dietary fiber, vitamin E, vitamin C, vitamin D, iron, zinc, selenium, n-3 polyunsaturated fatty acids (PUFA), white blood cells (WBC), Percentage of eosinophils (EOPC), lymphocyte count, monocyte count, hemoglobin (HGB), and 25-hydroxyvitamin D3 levels. Race/ethnicity was further classified as non-Hispanic white, non-Hispanic black, Mexican American, and other races [16, 17]. The following BMI classification categories were established based on weight classification criteria: underweight (BMI < 5th percentile), normal weight (BMI 5th to 85th percentile), overweight (BMI 85th to 95th percentile), and obese (BMI ≥ 95th percentile) [18]. PIR is computed as household (or individual) income divided by the survey year's poverty criterion. A 2015 U.S. government study states that household incomes are divided into three PIR-based groups: low (PIR ≤ 1.3), moderate (PIR 1.3–3.5), and high (PIR > 3.5) [19]. Mothers' smoking status during pregnancy was determined by answering the questionnaire "Did the biological mother smoke during pregnancy?" If the answer was "yes," the mother was considered to have smoked; otherwise, she was considered to have not smoked. The mother's age at birth was measured in years. Birth weight was defined as the participant's weight at birth in pounds. Daily intake of nutritional information, including vitamin D, vitamin A, vitamin E, vitamin C, n-3PUFAs, dietary fiber, dietary iron, and dietary zinc, was calculated using 24-h dietary recall interviews. Complete blood count (CBC) and blood cell distribution, including WBC count, EOPC, lymphocyte count, monocyte count, and HGB level, were measured in blood samples from each participant at the MEC using a Beckman Coulter DxH 800 device (California, USA). A Thermo Fisher TSQ Vantage system triple quadrupole tandem mass spectrometer (Thermo Fisher Scientific, Massachusetts, USA) was used to quantify the serum 25-hydroxyvitamin D3 levels. Comprehensive details of measures that consider covariates can be found at www.cdc.gov/nchs/nhanes/.

Analytical statistics

The publicly available dataset was subjected to quadratic analysis, whereby categorical variables were expressed as percentages (%) and continuous variables as either mean (SD) or median (IQR), contingent upon their distribution. One-way ANOVA (normal distribution), Kruskal–Wallis test (skewed distribution), and chi-square test (categorical variables) were used to compare the differences among the groups. The association between total energy intake and asthma was examined using logistic regression models that produced OR and 95% confidence intervals (95% CI). Three adjusted models in the multivariate logistic regression analysis were created: age, sex, race/ethnicity, BMIC, PIR, mother's smoking status throughout pregnancy, mother's birth weight, and mother's age at delivery, all of which were included in Model 1. Dietary fiber, vitamin E, vitamin C, vitamin D, iron, zinc, selenium, and n-3 polyunsaturated fatty acids were further corrected in Model 2; WBC, EOPC, lymphocyte count, monocyte count, HGB, and 25-hydroxyvitamin D3 were entirely adjusted in Model 3. After adjusting for variables in accordance with logistic regression Model 3, we examined the nonlinear relationship between total energy intake and asthma using a restricted cubic spline (RCS) with four nodes. Furthermore, we adjusted for variables consistent with Model 3 and examined the threshold connection between total energy intake and asthma using a two-stage logistic regression model. Multivariate logistic regression analysis and likelihood ratio tests for interactions were used to investigate the relationship

between total energy intake and asthma across the subgroups. Additionally, to further evaluate the robustness of the data, we eliminated individuals whose daily energy expenditure of < 500 and > 5000 kcal [16], and adjusted for body weight to perform sensitivity tests. R statistical software 4.2.2 (http://www.R-project.org, The R Foundation) and Free Statistics software 1.9.2 were used for all analyses. Statistical significance was set at $p < 0.05$.

Results

Study population and baseline characteristics

A total of 49,693 participants were interviewed between 2009 and 2018. Data from a total of 12,070 participants was included in the analysis (Fig. 1), of whom 1,893 (15.66%) had asthma and 50.6% were male. We excluded participants who were aged ≥ 18 years ($n = 30,352$), had missing data regarding asthma questionnaires ($n = 2007$), energy intake ($n = 5177$), and/or weight ($n = 77$). The median patient age was 9 years. We divided the total energy into four groups, Q1 (< 33.98 kcal/kg/day), Q2 (33.98–55.41 kcal/kg/day), Q3 (55.41–81.6 kcal/kg/day), and Q4 (> 81.6 kcal/kg/day). The four groups were statistically different ($p < 0.05$) with respect to age, sex, race/ethnicity, BMIC, PIR, mother's age at birth, birth weight, dietary fiber, vitamins E, C, and D, iron, zinc, selenium, n-3 PUFA, WBC, EOPC, lymphocyte count, monocyte count, hemoglobin, and 25-hydroxyvitamin D3 levels. There were no statistically significant differences among the four groups regarding maternal smoking during pregnancy (Table 1). The Supplementary Material describes the demographic characteristics according to the asthma status (Table S1).

Relationship between total energy intake and asthma

One-way logistic regression analysis showed, age, sex, race/ethnicity, PIR, mother's smoking status throughout pregnancy, birth weight, and mother's age at delivery, vitamin E, vitamin D, iron, zinc, selenium, and n-3 polyunsaturated fatty acids, EOPC, lymphocyte count, HGB, and 25-hydroxyvitamin D3 were significantly associated with asthma (all $P < 0.05$) (Table S2). In a multifactorial logistic regression analysis, total energy was categorized into four groups, and total energy intake was negatively associated with asthma after excluding potential confounders. Compared with individuals with the lowest energy intake Q1 (< 33.98 kcal/kg/day), the asthma-adjusted odds ratio (OR) of the Q2 (33.98–55.41 kcal/kg/day), Q3 (55.41–81.6 kcal/kg/day), and Q4 (> 81.6 kcal/kg/day) groups were 0.72 (0.62–0.85, $p = 0.001$), 0.63 (0.51–0.77, $p < 0.001$), and 0.55 (0.43–0.7, $p < 0.001$), respectively (Table 2). Notably, the restricted cubic spline analysis revealed an L-shaped curve (nonlinear, $p = 0.001$) between total energy intake and asthma (Fig. 2). We found an odds ratio (OR) of 0.981,95% CI: 0.973–0.989, $p < 0.001$) among people with a total energy intake < 56.442 kcal/kg/day in the threshold analysis. For every unit increase in total energy intake (kcal/kg/day), the risk of asthma decreased by 1.9%. Conversely, we discovered that there was no longer a negative correlation between total energy intake and asthma, with a computed OR of 0.997 (95% CI: 0.993–1) and a p-value of 0.0906 for participants who consumed ≥ 56.442 kcal/kg/day (Table 3).

Stratification and sensitivity analysis based on additional variables

Stratified analyses of the subgroups revealed no significant interactions in any category when stratified by age, sex, ethnicity/race, or PIR (Fig. 3). Data from 12,046 people remained after eliminating those with extremely high total energy intakes, and the correlation between total energy intake and asthma did not change (Table S3).

Discussion

Our cross-sectional survey found an L-shaped association between total energy intake and asthma among Americans aged 1–18 years. A 1.9% decrease in the probability of asthma was linked to every unit increase in total energy intake (kcal/kg/day) when it was less than 56.442 kcal/kg/day. No significant relationship was found between total energy intake and the risk of asthma when intake surpassed 56.442 kcal/kg/day. These results show that there may be a saturation effect and that consuming more overall energy may not further lower the incidence of asthma. The subgroup and sensitivity analysis results were consistent. The clinical consequences of these findings are significant.

Numerous studies have examined the connection between diet and asthma in various life phases, including infancy [8, 20], adolescence [21–23], and adulthood [24], but the findings have been inconsistent. A 2016 study conducted in the United States found that children between the ages of 2 and 9 who consumed excessive amounts of free fructose beverages had a higher chance of developing asthma [25]. This study may have been biassed and produced different results because the exposure variable was derived using a food intake frequency questionnaire. In contrast, the consumption of whole grains and fish was found to be negatively correlated with asthma in a study on diet and asthma in Dutch students [5], whereas the consumption of (citrus) fruits, vegetables, and dairy products was not correlated with asthma. However, as foods are ingested in combination, a more comprehensive approach to illness prevention may be suggested by viewing the diet as a whole rather than focusing on particular ingredients or

Table 1 Baseline information on the population

Variables	Total energy(kcal/kg/day)					*p*
	Total (*n* = 12,090)	Q1(*n* = 3023)	Q2 (*n* = 3022)	Q3 (*n* = 3022)	Q4 (*n* = 3023)	
Age (years)	9.0 (4.0, 13.0)	14.0 (12.0, 16.0)	11.0 (8.0, 14.0)	7.0 (4.0, 9.0)	3.0 (2.0, 5.0)	< 0.001
Sex, n (%)						< 0.001
Male	6114 (50.6)	1355 (44.8)	1507 (49.9)	1604 (53.1)	1648 (54.5)	
Females	5976 (49.4)	1668 (55.2)	1515 (50.1)	1418 (46.9)	1375 (45.5)	
Race and ethnicity, n (%)						< 0.001
Non-Hispanic white	3519 (29.1)	740 (24.5)	865 (28.6)	956 (31.6)	958 (31.7)	
Non-Hispanic black	2883 (23.8)	841 (27.8)	695 (23)	660 (21.8)	687 (22.7)	
Mexican American	2657 (22.0)	727 (24)	685 (22.7)	627 (20.7)	618 (20.4)	
Others	3031 (25.1)	715 (23.7)	777 (25.7)	779 (25.8)	760 (25.1)	
PIR, n (%)						< 0.001
Low	5427 (44.9)	1361 (45)	1288 (42.6)	1350 (44.7)	1428 (47.2)	
Medium	4230 (35.0)	1116 (36.9)	1083 (35.8)	1028 (34)	1003 (33.2)	
High	2433 (20.1)	546 (18.1)	651 (21.5)	644 (21.3)	592 (19.6)	
BMI Category, n (%)						< 0.001
Underweight	344 (2.8)	28 (0.9)	76 (2.5)	130 (4.3)	110 (3.6)	
Normal weight	7314 (60.5)	1159 (38.3)	1869 (61.8)	2175 (72)	2111 (69.8)	
Overweight	1968 (16.3)	655 (21.7)	546 (18.1)	395 (13.1)	372 (12.3)	
Obese	2464 (20.4)	1181 (39.1)	531 (17.6)	322 (10.7)	430 (14.2)	
Asthma, n (%)						< 0.001
No	10,197 (84.3)	2378 (78.7)	2523 (83.5)	2610 (86.4)	2686 (88.9)	
Yes	1893 (15.7)	645 (21.3)	499 (16.5)	412 (13.6)	337 (11.1)	
Mother smoked when pregnant, n (%)						0.141
No	10,703 (88.5)	2666 (88.2)	2669 (88.3)	2710 (89.7)	2658 (87.9)	
Yes	1387 (11.5)	357 (11.8)	353 (11.7)	312 (10.3)	365 (12.1)	
Mother's age when born(years)	27.0 ± 6.3	26.5 ± 6.3	26.9 ± 6.3	27.4 ± 6.2	27.3 ± 6.2	< 0.001
Weight at birth (pounds)	6.8 ± 1.4	6.9 ± 1.4	6.8 ± 1.4	6.8 ± 1.3	6.6 ± 1.4	< 0.001
HGB (g/dL)	13.1 ± 1.2	13.6 ± 1.3	13.3 ± 1.2	12.9 ± 1.0	12.5 ± 0.9	< 0.001
WBC (1000 cells/μL)	7.0 (5.7, 8.5)	6.9 (5.6, 8.2)	6.7 (5.4, 8.1)	7.0 (5.7, 8.5)	7.6 (6.3, 9.3)	< 0.001
EOPC (%)	2.6 (1.6, 4.3)	2.3 (1.4, 3.9)	2.6 (1.6, 4.4)	2.8 (1.7, 4.7)	2.7 (1.7, 4.3)	< 0.001
Lymphocyte number (1000 cells/μL)	2.7 (2.1, 3.4)	2.3 (1.9, 2.8)	2.4 (2.0, 3.0)	2.8 (2.3, 3.6)	3.5 (2.7, 4.5)	< 0.001
Monocyte number (1000 cells/μL)	0.6 (0.4, 0.7)	0.5 (0.4, 0.7)	0.5 (0.4, 0.6)	0.6 (0.4, 0.7)	0.6 (0.5, 0.8)	< 0.001
Dietary fiber (gm/day)	12.4 (9.1, 16.6)	10.9 (8.1, 14.4)	13.5 (10.0, 17.8)	12.7 (9.2, 17.0)	12.8 (9.5, 17.0)	< 0.001
Vitamin E(mg/day)	5.6 (4.0, 7.8)	5.0 (3.6, 6.8)	6.2 (4.5, 8.4)	5.7 (4.0, 8.1)	5.8 (4.2, 7.9)	< 0.001
Vitamin C (mg/day)	64.3 (34.7, 104.2)	48.0 (22.9, 86.2)	63.5 (33.0, 101.8)	68.1 (39.1, 103.3)	79.4 (47.1, 121.9)	< 0.001
Vitamin D (D2 + D3) (mcg/day)	5.1 (3.0, 7.6)	3.5 (1.8, 5.6)	4.8 (3.0, 7.2)	5.4 (3.5, 7.7)	6.8 (4.5, 9.4)	< 0.001
Iron (mg/day)	12.2 (8.8, 16.4)	10.7 (7.9, 14.4)	13.3 (9.9, 17.5)	12.5 (9.0, 16.7)	12.3 (8.9, 16.6)	< 0.001
Zinc (mg/day)	8.6 (6.4, 11.5)	7.7 (5.6, 10.3)	9.3 (7.0, 12.4)	8.6 (6.3, 11.8)	8.9 (6.8, 11.6)	< 0.001
Selenium (mcg/day)	84.0 (62.2, 109.2)	78.4 (59.0, 101.1)	93.2 (69.8, 121.4)	81.8 (59.4, 108.1)	82.2 (63.5, 106.6)	< 0.001
25-hydroxyvitamin D3 (nmol/L)	63.7 (51.4, 76.7)	53.8 (41.9, 65.7)	61.7 (50.1, 73.4)	67.6 (56.4, 79.8)	71.0 (59.9, 83.0)	< 0.001
n-3 PUFAs (mg/day)	1.2 (0.8, 1.6)	1.1 (0.7, 1.5)	1.3 (0.9, 1.8)	1.2 (0.8, 1.6)	1.2 (0.9, 1.6)	< 0.001
Total energy (kcal/kg/day)	55.4 (34.0, 81.6)	24.6 (19.1, 29.3)	44.2 (38.9, 49.6)	67.9 (61.3, 74.7)	102.7 (91.2, 119.9)	< 0.001

Abbreviations: PIR ratio of income to poverty, *BMI* body mass index, *WBC* white blood cell count, *EOPC* eosinophils percent, *HGB* hemoglobin, n-3 PUFAs, n-3 polyunsaturated fatty acids, *Q* quartile, Q1 (< 33.98); Q2 (33.98–55.41); Q3 (55.41–81.6); Q4 (> 81.6)

nutrients. According to certain research, a "Western" diet may make children more likely to experience respiratory symptoms frequently between the ages of 3 and 4 [8]; in contrast, a Mediterranean diet may help prevent asthma attacks [7, 26]. This correlation can be partially explained by energy intake. According to systematic reviews and meta-analyses of observational studies [27] found in the Cochrane Database, reducing dietary caloric intake may

Table 2 Association between total energy intake and asthma

Total energy intake	OR (95%CI)									
	No	N (%)	Unadjusted	*P*-value	Model 1	*P*-value	Model 2	*P*-value	Model 3	*P*-value
Quartile(kcal/kg/day)										
Q1(< 33.98)	3023	645 (21.3)	1(Ref)		1(Ref)		1(Ref)		1(Ref)	
Q2 (33.98–55.41)	3022	499 (16.5)	0.73 (0.64 ~ 0.83)	< 0.001	0.86 (0.74 ~ 0.99)	0.038	0.72 (0.62 ~ 0.84)	< 0.001	0.72 (0.62 ~ 0.85)	0.001
Q3(55.41–81.6)	3022	412 (13.6)	0.58 (0.51 ~ 0.67)	< 0.001	0.79 (0.66 ~ 0.94)	0.009	0.62 (0.51 ~ 0.75)	< 0.001	0.63 (0.51 ~ 0.77)	< 0.001
Q4 (> 81.6)	3023	337 (11.1)	0.46 (0.4 ~ 0.53)	< 0.001	0.64 (0.52 ~ 0.78)	< 0.001	0.49 (0.39 ~ 0.62)	< 0.001	0.55 (0.43 ~ 0.7)	< 0.001
Trend. test	12,090	1893 (15.7)		< 0.001		< 0.001		< 0.001		< 0.001

Model1: adjusted for age, sex, race/ethnicity, BMI Category, PIR; Mother smoked when pregnant; Mother's age when born; Weight at birth;

Model2:Model1 + Dietary fiber; Vitamin E; Vitamin C; Vitamin D; Iron; Zinc; Selenium; n-3 PUFAs;

Model3:Model2 + WBC; EOPC; Lymphocyte number; Monocyte number; HGB; 25-hydroxyvitamin D3;

Abbreviations: CI confidence interval, *OR*, odds ratio, *Ref* reference, *PIR* ratio of income to poverty, *BMI* body mass index, *WBC* white blood cell count, *EOPC* eosinophils percent, *HGB* hemoglobin; n-3 PUFAs, n-3 polyunsaturated fatty acids, *Q* quartile

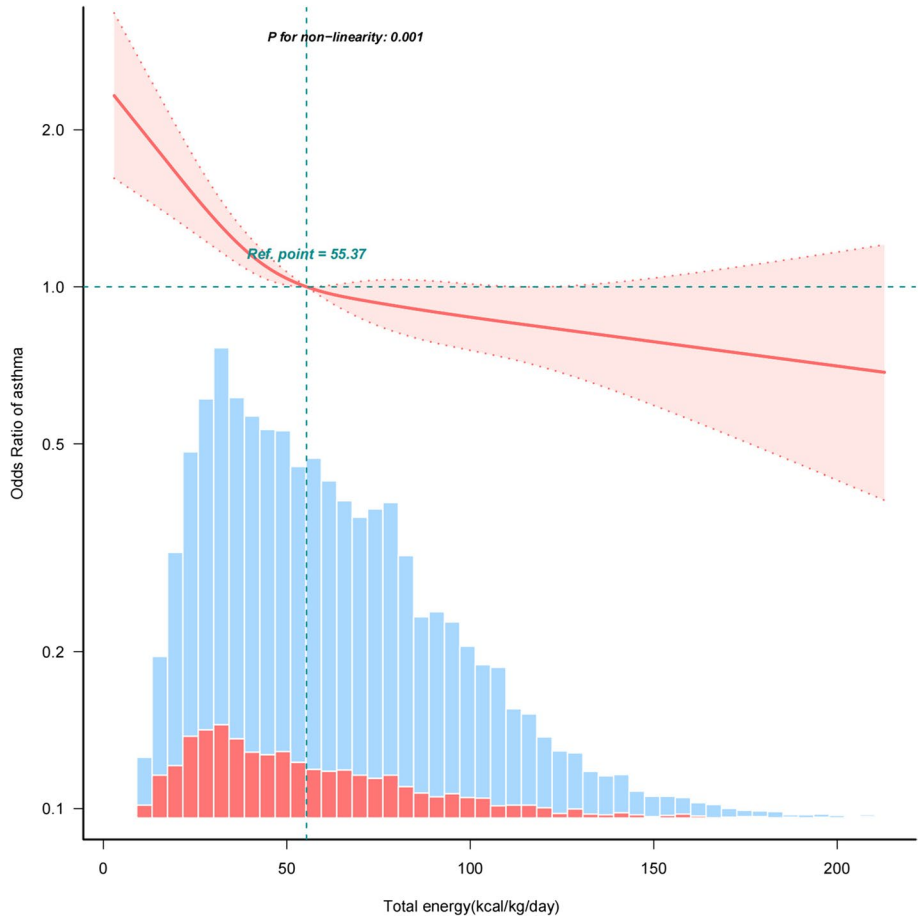


Fig. 2 Odds ratios for the association between total energy intake and asthma. Note: Solid and dashed lines represent predicted values and 95% confidence intervals. Adjusted for age, sex, race/ethnicity, BMI category, PIR, mother's smoking during pregnancy, mother's age at birth, birth weight, dietary fiber, vitamin E, vitamin C, vitamin D, iron, zinc, selenium, n-3 PUFA, WBC, EOPC, lymphocyte counts, monocyte counts, HGB, and 25-hydroxyvitamin D3; Only 99.9% of the data is shown. Abbreviations: PIR, ratio of income to poverty; BMI, body mass index; WBC, white blood cell count; EOPC, eosinophil percentage; HGB, hemoglobin; n-3 PUFAs, n-3 polyunsaturated fatty acids

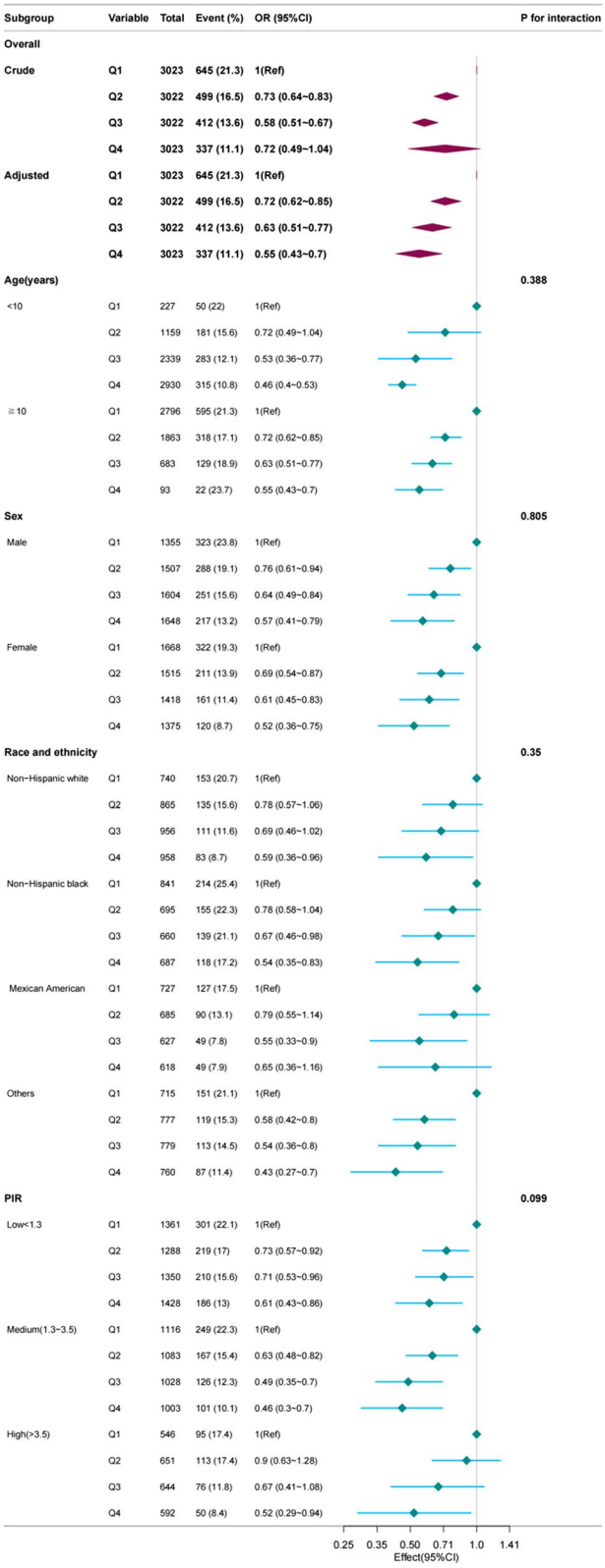
Table 3 Threshold analysis of the relationship between total energy intake and asthma

Total energy(kcal/kg/day)	No	Adjusted Model	
		OR (95%CI)	*P*-value
< 56.442	6177	0.981 (0.973 ~ 0.989)	<0.001
≥ 56.442	5913	0.997 (0.993 ~ 1)	0.0906
Likelihood ratio test			<0.001

Adjusted for age, sex, race/ethnicity, BMI category, PIR, mother's smoking during pregnancy, mother's age at birth, birth weight, dietary fiber, vitamin E, vitamin C, vitamin D, iron, zinc, selenium, n-3 PUFA, WBC, EOPC, lymphocyte counts, monocyte counts, HGB, and 25-hydroxyvitamin D3; Only 99.9% of the data is displayed. *Abbreviations: CI* confidence interval, *OR* odds ratio, *PIR* ratio of income to poverty, *BMI* body mass index, *WBC* white blood cell count, *EOPC* eosinophil percentage, *HGB* hemoglobin, n-3 PUFAs, n-3 polyunsaturated fatty acids

help reduce asthma symptoms. Dietary caloric restriction is crucial for both adults and children with chronic asthma. However, the review was constrained by a small sample size and the fact that the study participants were obese.

Different populations, ages, study methods, and follow-up durations may account for the differences between our results and those of other studies. Unlike these earlier studies, our study selected Americans aged 1–18 years and appropriately controlled for factors that may influence asthma attacks. Once the total energy intake exceeded a specific threshold, we did not find a significant correlation between asthma and total energy intake. However, within the threshold range, the asthma risk decreased as the total energy intake increased. For example, for a 20 kg child, it is better to maintain the total energy intake within 1100 kcal per day to decrease asthma risk. Notably, our findings are supported, to



◀ **Fig. 3** Forest plot of multivariable logistics analysis between total energy intake and asthma. Abbreviations: Energy intake quartile (kcal/kg/day): Q1 (< 33.98); Q2 (33.98–55.41); Q3 (55.41–81.6), Q4 (> 81.6), CI, confidence interval; OR, odds ratio; PIR, ratio of income to poverty

some extent, by those of other studies. For example, Maffei et al. [21] found that children with mild-to-moderate asthma may benefit from using their dietary history to estimate their energy needs. In a review of weight loss interventions for chronic asthma [28], it was found that weight loss may help improve asthma control in overweight and obese patients; however, an appropriate range of energy intake was not specified. Multicenter randomized controlled trials are required to confirm our findings.

Despite the need for further research into the potential mechanisms underlying the negative correlation between energy intake and asthma, the results of this study are already biologically plausible based on existing evidence. First, excessive energy intake, particularly from high-sugar and high-fat diets, may indirectly increase the risk of asthma by promoting chronic low-grade inflammation and the development of obesity. [29] In contrast, moderate energy intake helps maintain normal immune system function, reduces excessive immune responses, and thereby lowers the incidence of asthma [30]. Second, obesity is one of the key risk factors for asthma [31, 32]. Excessive energy intake leads to fat accumulation, and cytokines secreted by adipose tissue (such as tumor necrosis factor-alpha and interleukin-6) can promote systemic inflammatory responses [33]. An increase in these cytokines may exacerbate asthma symptoms. Furthermore, energy intake may influence asthma susceptibility by modulating cytokine levels, particularly the secretion of pro-inflammatory cytokines. For example, excessive energy intake may increase levels of pro-inflammatory cytokines such as interleukin-4 (IL-4) and interleukin-13 (IL-13), which play a critical role in the pathogenesis of asthma [34]. In contrast, moderate energy intake helps maintain immune system balance, reduces the release of pro-inflammatory cytokines, and thus decreases the incidence of asthma [35]. Third, excessive energy intake, particularly from high-sugar and high-fat diets, may increase oxidative stress levels in the body. Oxidative stress refers to an imbalance between reactive oxygen species and antioxidant defenses, where excess reactive oxygen species can damage cells and activate inflammatory pathways, thereby worsening asthma pathogenesis [36, 37]. On the other hand, lower energy intake (especially reduced sugar intake) may help decrease oxidative stress levels, thereby reducing lung damage reduce [38]. Fourth, energy intake may also affect the development

of asthma by altering the gut microbiota composition [39]. High-energy diets can alter gut microbiota, increasing the abundance of pro-inflammatory bacteria while decreasing anti-inflammatory bacteria. This microbial imbalance may promote systemic inflammation, increase airway sensitivity, and thus contribute to asthma development [40]. Studies suggest an L-shaped relationship between energy intake and asthma prevalence, indicating that moderate energy intake (approximately 56.442 kcal/kg/day) may provide the best protective effect. Therefore, appropriate energy intake is of significant importance in asthma management. The relationship between energy intake and asthma likely involves multiple interacting mechanisms, including immune modulation, inflammatory responses, obesity development, and oxidative stress. Future research needs to further explore the specific effects of energy intake on different asthma subtypes and whether moderate energy intake can serve as a preventive and intervention strategy to reduce asthma prevalence. Additionally, further clinical studies will help clarify the specific thresholds of energy intake and the underlying biological mechanisms.

The use of data spanning ten years to obtain a large sample size is one of the strengths of our study. To examine the dose–response relationship between total energy intake and asthma risk in Americans aged 1–18 years, we used robust methods including regression modeling, restricted triple spline curves, and threshold analysis. However, there are some limitations of this study that need to be considered. First, total energy intake data were derived from 24-h self-reported dietary recall, which may lead to bias. Second, despite adjusting for various confounders, the association between total energy intake and asthma may have been modified by other potential confounding variables. In addition, because of the discontinuity in child growth and development, with higher energy requirements in infancy and adolescence, we will need to examine the association between energy intake and asthma at different ages in the future. Finally, due to the inherent limitations of cross-sectional studies, a causal relationship between total energy intake and asthma cannot be demonstrated, and long-term studies are still needed.

Conclusion

We found an L-shaped relationship between total energy intake and asthma among Americans aged 1–18 years. An important inflection point was located at an intake of approximately 56.442 kcal/kg/day. Larger prospective studies using more precise dietary assessment techniques are needed to confirm and expand our findings.

Abbreviations

NHANES	National Health and Nutrition Examination Survey
BMI Category	Body mass index category
PIR	Ratio of income to poverty
WBC	White blood cell count
EOPC	Eosinophils percent
BMI Category	Body mass index category
HGB	Hemoglobin
RCS	Restricted cubic spline

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12887-025-05552-5.

Supplementary Material 1.

Supplementary Material 2.

Supplementary Material 3.

Acknowledgements

We thank Jie Liu of the Department of Vascular and Endovascular Surgery, Chinese PLA General Hospital, for his contribution to statistical support, study design consultations, and comments regarding the manuscript.

Authors' contributions

Linjun Du, Chao Che, Qian Liu, Xiaolan Zhang, Ning Feng, Lifang Chen, Lili Wang contributed to the study design, data collection, analysis, interpretation of results, and manuscript preparation. All the authors have read and approved the final version of the manuscript. Conceptualization, L. J., C., and Q.; Data curation, L. J., C, X.L. and L. F.; Formal analysis, N., L. F. and L.J. Methodology: L.F., L.J., and Q.; Writing original draft: L.J. and C; Writing review and editing: L.L., N, Q.,and L.F. All authors have read and agreed to the published version of the manuscript.

Funding

This research did not receive any specific grants from funding agencies in the public, commercial, or not-for-profit sectors.

Data availability

Publicly available datasets are available online for this study. The repository names and accession numbers are available online at http://www.cdc.gov/nchs/nhanes.htm (accessed on 11 November 2024).

Declarations

Ethics approval and consent to participate

Ethical review and approval for this study were not required as it involved a secondary analysis and did not require additional institutional review board approval. The National Health and Nutrition Examination Survey (NHANES) was approved by the Ethics Review Committee of the National Center for Health Statistics (NCHS). Written informed consent was obtained from all participants before their participation in the survey. Participants under 16 years of age, with informed consent provided by their parents or legal guardians.

Consent for publication

Not Applicable.

Competing interests

The authors declare no competing interests.

Received: 7 November 2024 Accepted: 26 February 2025
Published online: 11 March 2025

References

1. Gans MD, Gavrilova T. Understanding the immunology of asthma: Pathophysiology, biomarkers, and treatments for asthma endotypes. Paediatr Respir Rev. 2020;36:118–27.

2. Most recent national asthma data | CDC. 2023. https://www.cdc.gov/asthma/most_recent_national_asthma_data.htm. Accessed 29 Mar 2024.
3. Perry R, Braileanu G, Palmer T, Stevens P. The economic burden of pediatric asthma in the united states: literature review of current evidence. Pharmacoeconomics. 2019;37:155–67.
4. Reis WP, Chai E, Gaio J, Becerra MB, Banta JE, Dos Santos H. Dietary factors associated with asthma prevalence among children in california. Pediatr Allergy Immunol Pulmonol. 2020;33:85–91.
5. 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.
6. Brustad N, Bønnelykke K, Chawes B. Dietary prevention strategies for childhood asthma. Pediatr Allergy Immunol. 2023;34:e13984.
7. Zhang Y, Lin J, Fu W, Liu S, Gong C, Dai J. Mediterranean diet during pregnancy and childhood for asthma in children: a systematic review and meta-analysis of observational studies. Pediatr Pulmonol. 2019;54:949–61.
8. Tromp IIM, Kiefte-de Jong JC, De Vries JH, Jaddoe VWV, Raat H, Hofman A, et al. Dietary patterns and respiratory symptoms in pre-school children: the generation R study. Eur Respir J. 2012;40:681–9.
9. Oland AA, Booster GD, Bender BG. Psychological and lifestyle risk factors for asthma exacerbations and morbidity in children. World Allergy Organ J. 2017;10:35.
10. NHANES - national health and nutrition examination survey homepage. 2024. https://www.cdc.gov/nchs/nhanes/index.htm. Accessed 26 Mar 2024.
11. Ahluwalia N, Dwyer J, Terry A, Moshfegh A, Johnson C. Update on NHANES dietary data: focus on collection, release, analytical considerations, and uses to inform public policy. Adv Nutr. 2016;7:121–34.
12. NHANES - NCHS research ethics review board approval. 2022. https://www.cdc.gov/nchs/nhanes/irba98.htm. Accessed 25 Jul 2024.
13. Zhang 等 - 2023 - The association between n-3 polyunsaturated fatty acid intakes and asthma in span style=font-varia.pdf.
14. NHANES survey methods and analytic guidelines. https://wwwn.cdc.gov/Nchs/Nhanes/AnalyticGuidelines.aspx. Accessed 28 Mar 2024.
15. Nurmatov U, Nwaru BI, Devereux G, Sheikh A. Confounding and effect modification in studies of diet and childhood asthma and allergies. Allergy. 2012;67:1041–59.
16. Liu H, Wang L, Chen C, Dong Z, Yu S. Association between dietary niacin intake and migraine among american adults: national health and nutrition examination survey. Nutrients. 2022;14:3052.
17. Frey T. Updated guidance on the reporting of race and ethnicity in medical and science journals. AMWA J. 2023;38.
18. Growth charts - 2000 CDC growth charts - united states. 2022. https://www.cdc.gov/growthcharts/cdc_charts.htm. Accessed 28 Mar 2024.
19. What we eat In america - food commodity intake database. https://fcid.foodrisk.org/. Accessed 7 Mar 2025.
20. Kim SY, Sim S, Park B, Kim J-H, 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:e0150202.
21. Maffeis C, Chiocca E, Zaffanello M, Golinelli M, Pinelli L, Boner A. Energy intake and energy expenditure in prepubertal males with asthma. Eur Respir J. 1998;12:123–9.
22. Berntsen S, Carlsen KCL, Anderssen SA, Mowinckel P, Hageberg R, Bueso AK, et al. Norwegian adolescents with asthma are physical active and fit*. Allergy. 2009;64:421–6.
23. Huang S, Pan W. Dietary fats and asthma in teenagers: analyses of the first nutrition and health survey in taiwan (NAHSIT). Clin Exp Allergy. 2001;31:1875–80.
24. Park S, Akinbami LJ, McGuire LC, Blanck HM. Association of sugar-sweetened beverage intake frequency and asthma among US adults, 2013. Prev Med. 2016;91:58–61.
25. DeChristopher LR, Uribarri J, Tucker KL. Intakes of apple juice, fruit drinks and soda are associated with prevalent asthma in US children aged 2–9 years. Public Health Nutr. 2016;19:123–30.
26. Morales E, Strachan D, Asher I, Ellwood P, Pearce N, Garcia-Marcos L. Combined impact of healthy lifestyle factors on risk of asthma, rhinoconjunctivitis and eczema in school children: ISAAC phase III. Thorax. 2019;74:531–8.
27. Cheng J, Pan T. Calorie controlled diet for chronic asthma. Cochrane Database Syst Rev. 2003. https://doi.org/10.1002/14651858.CD004674.pub2.
28. Adeniyi FB, Young T. Weight loss interventions for chronic asthma. Cochrane Database Syst Rev. 2012. https://doi.org/10.1002/14651858.CD009339.pub2.
29. Zhang P. The Role of Diet and Nutrition in Allergic Diseases. Nutrients. 2023;15:3683.
30. Cao X, Lu T, Tu Y, Zhou R, Li X, Du L. The association between adult asthma in the United States and dietary total energy intake: a retrospective cross-sectional analysis from NHANES. BMC Nutr. 2024;10:128.
31. Peters U, Dixon AE, Forno E. Obesity and asthma. J Allergy Clin Immunol. 2018;141:1169–79.
32. Wood LG. Diet, obesity, and asthma. Ann Am Thorac Soc. 2017;14(Supplement_5):S332-8.
33. 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.
34. IL-4/IL-13 axis as therapeutic targets in allergic rhinitis and asthma - PubMed. https://pubmed.ncbi.nlm.nih.gov/35663523/. Accessed 7 Feb 2025.
35. Zimmermann N, Hershey GK, Foster PS, Rothenberg ME. Chemokines in asthma: cooperative interaction between chemokines and IL-13. J Allergy Clin Immunol. 2003;111:227–42; quiz 243.
36. Gozzi-Silva SC, Teixeira FME, Duarte AJ da S, Sato MN, Oliveira L de M. Immunomodulatory Role of Nutrients: How Can Pulmonary Dysfunctions Improve?. Front Nutr. 2021;8:674258.
37. Ik I, A L, F D, I T, Mm A, V S, et al. Oxidative Stress and Antioxidants in Pediatric Asthma's Evolution and Management. Antioxid Basel Switz. 2024;13:1331
38. Ferreira CA, de Souza FIS, Melges APB, Fonseca FA, Solé D, Sarni ROS. Retinol, beta-carotene, oxidative stress, and metabolic syndrome components in obese asthmatic children. Pediatr Allergy Immunol Off Publ Eur Soc Pediatr Allergy Immunol. 2014;25:292–4.
39. Alsharairi NA. The Role of Short-Chain Fatty Acids in the Interplay between a Very Low-Calorie Ketogenic Diet and the Infant Gut Microbiota and Its Therapeutic Implications for Reducing Asthma. Int J Mol Sci. 2020;21:9580.
40. Dysbiosis of the gut and lung microbiome has a role in asthma - PubMed. https://pubmed.ncbi.nlm.nih.gov/32072252/. Accessed 7 Feb 2025.

Publisher's Note