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The association between body mass index and asthma in children: a cross-sectional study from NHANES 1999 to 2020

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The relationship between body mass index (BMI) and the risk of asthma in the pediatric population is not fully understood. This study aimed to investigate the association between BMI and asthma in a large nationally representative sample. The study included 35,603 pediatric participants from the National Health and Nutrition Examination Survey (NHANES) from 1999 to 2020. The association between BMI and asthma was examined using various statistical models, including logistic regression, piece-wise linear regression, and subgroup analyses, adjusting for potential confounding factors. When analyzing BMI as a continuous variable, a one-unit increase in BMI was associated with a 4% higher odds of asthma. A clear dose-response relationship was observed, where individuals in the higher BMI quartiles had progressively higher odds of asthma compared to those in the lowest quartile. Smooth curve fitting revealed a not entirely linear relationship, with a steeper increase in asthma risk at lower BMIs (below an inflection point of 21 kg/m²) compared to higher BMIs. Subgroup analyses consistently showed a positive association between BMI and asthma across different age, gender, race, socioeconomic, and smoking-related factors. Sensitivity analyses, including multiple imputation for missing data and alternative BMI metrics, confirmed the stability of the results. This study provides robust evidence for a positive and not entirely linear association between BMI and the risk of asthma in the pediatric population. These findings enhance the existing literature and underscore the necessity of considering BMI in both asthma research and clinical practice.

Keywords Body mass index, Asthma, Children, Cross-sectional study, NHANES

Asthma is a chronic respiratory disease characterized by airway inflammation and narrowing, resulting in recurrent episodes of wheezing, shortness of breath, chest tightness, and coughing[1]. The prevalence of asthma varies across populations, with higher rates observed in urban and developed regions, such as 7.2% in the United States [2] and 4.2% in China[3]. Asthma is estimated to impact 262 million people, equating to an age-standardized prevalence rate of 3,416 cases per 100,000 individuals[4]. Exposure to triggers like allergens, respiratory infections, air pollutants, and occupational irritants can lead to airway inflammation and hyperresponsiveness in susceptible individuals[5,6]. The immune response is characterized by T-helper 2 (Th2) cell-mediated inflammation, involving the release of cytokines that promoting immunoglobulin (Ig) E production, eosinophil recruitment, and mucus secretion[7]. Early-life factors, including exposure to secondhand smoke, respiratory infections, and a family history of asthma or allergies contribute to asthma development[8]. Asthma treatment strategies involve a combination of medications, such as inhaled corticosteroids, bronchodilators, and leukotriene modifiers, tailored to the individual's disease severity and triggers. Additionally, biological therapies have demonstrated promising results in severe, treatment-resistant asthma cases[9,10].

Obesity is a complex, multifactorial chronic disease characterized by an excessive accumulation of body fat that poses significant health risks. The prevalence of obesity has surged globally, becoming a major public health concern linked to numerous comorbidities, including type 2 diabetes, cardiovascular diseases, and cancers[11]. Obesity also contributes to impaired respiratory function, with evidence indicating a relationship between increased body weight and exacerbated respiratory conditions, such as asthma[12]. Obesity impacts asthma pathogenesis through various mechanisms, such as reducing immune tolerance by releasing pro-inflammatory mediators like IL-6, leptin, and tumor necrosis factor alpha (TNFα), thereby promoting asthma development[13]. Additionally, obesity may alter the clinical presentation of asthma by modifying airway inflammation, for

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instance, by decreasing the proportion of eosinophils[14], and affecting lung mechanical properties[15]. Research has shown that obese individuals with asthma may exhibit more nonspecific inflammatory characteristics, such as neutrophilia[16]. Genetic studies suggest shared pathophysiological mechanisms between obesity and asthma, while a high-fat diet may also contribute to asthma development[17]. Obesity can also impact the efficacy of asthma treatment, particularly by reducing responsiveness to inhaled glucocorticoids[18].

Body mass index (BMI) is the most widely used metric for evaluating the degree of obesity. While BMI can be readily calculated and measured to determine obesity in adults, defining overweight and obesity in children is more controversial[19]. Due to variations in classification methods and research approaches, prior studies on the association between obesity and asthma have sometimes yielded inconsistent findings[19–21]. Therefore, it remains necessary to examine the association between childhood BMI and asthma using large-scale data. This study employed the National Health and Nutrition Examination Survey (NHANES) 1999–2020 data on children and adolescents to perform a comprehensive analysis of the association between BMI and asthma, aiming to provide the latest robust evidence in this domain.

Methods and materials

Study population

NHANES, a program of the Centers for Disease Control and Prevention (CDC), has been assessing the health and nutritional status of the American population since the early 1960s. NHANES employs a well-designed survey and sampling methodology to ensure national representation, collecting comprehensive data on diverse health indicators for both adults and children through in-depth interviews, physical examinations, and laboratory tests[22]. Eligible participants aged 16 and older were interviewed directly, while those under 16 were interviewed with a proxy. All participants who completed the household interview were invited to the Medical Examination component. This integrated approach has enabled NHANES to uncover critical insights into chronic diseases, dietary patterns, physical activity, and environmental exposures, informing evidence-based public health policies and clinical guidelines. The publicly available NHANES data serves as an invaluable resource for researchers investigating complex health phenomena and addressing pressing public health issues. Scholars can customize their data retrieval to align with their specific research objectives, selectively extracting relevant variables related to epidemiology, nutrition, and various other health metrics, while adhering to strict guidelines for data use and dissemination. For this study, we accessed data from the 1999–2020 NHANES cycles and collected demographic, examination, laboratory, and questionnaire data on relevant variables.

Body mass index

In NHANES, BMI data are derived from the body measurement section of the examination data. Body measurements are obtained through standardized procedures carried out by trained health technicians in specially equipped mobile examination centers. Height is measured using a stadiometer and weight is measured using a digital scale. These measurements follow strict protocols to ensure accuracy and consistency across survey participants. BMI is calculated as weight in kilograms divided by the square of height in meters, and NHANES provides BMI data for individuals aged 2 years and above.

Asthma

The assessment of asthma status in NHANES is typically based on responses to specific questionnaire items related to medical conditions. The presence of asthma is determined by the response to the question "Has a doctor or other health professional ever told you that you have/s/he has asthma?". The NHANES website provides a more comprehensive description of the variables used to assess asthma.

Covariates

Based on a review of prior research[23], the potential confounders for the current study included demographic and home environment factors: age, gender, race, poverty income ratio (PIR), and household smokers; as well as laboratory tests: serum lead, serum cadmium, and cotinine. PIR is defined as the ratio of household income to the poverty threshold. Previous studies have shown that children born in areas with higher proportions of low-income families, population density, and poverty have higher asthma prevalence rates[24]. The presence of a smoker in the home was determined by the participant's response to the interview question, "Does anyone who lives here smoke cigarettes, cigars, or pipes anywhere inside this home?". Cotinine, a major metabolite of nicotine, can be used as a marker of active smoking and an indicator of environmental tobacco smoke exposure, also known as "passive smoking". Numerous studies have reported that exposure to environmental tobacco smoke, or "secondhand smoke," increases the incidence and severity of asthma[25,26]. In addition, serum levels of lead, and cadmium have also been implicated in asthma, allergies, and other diseases[27].

Inclusion and exclusion criteria

Individuals aged 2 to 19 years who answered yes or no to the asthma assessment questions and had BMI data were included in this study. Participants with missing data for any of the study variables were excluded.

Ethics statement

The human subjects used in this study were acquired from the NHANES protocol which was approved by the Institutional Review Board of the National Center for Health Statistics (https://wwwn.cdc.gov/Nchs/Nhanes/). All participants or their legal guardians provided written informed consent.

Statistical analysis

Appropriate sampling weights (NHANES MEC weights) were applied to the data to account for oversampling, nonresponse, and noncoverage, allowing for nationally representative estimates. Continuous variables were summarized as means ± standard deviations, while categorical variables were presented as percentages. Participants were categorized into asthma and non-asthma groups based on the presence or absence of asthma. Weighted chi-square tests were used to calculate p-values for categorical variables, while weighted linear regression models were used for continuous variables. The association between BMI and asthma was evaluated using three logistic regression models. Model 1 was unadjusted, Model 2 was partially adjusted for age, gender, and race, and Model 3 was fully adjusted for all covariates, including age, gender, race, PIR, serum lead, serum cadmium, cotinine, and the presence of a smoker in the home. Trend tests were used to confirm the validity of the regression analysis results. Smooth curve fitting was employed to explore potential nonlinear relationships between BMI and asthma, and piecewise linear regression was used to analyze threshold effects if a nonlinear relationship was identified. The smooth curve fitting was conducted using generalize additive models. Stratified analyses by gender, age, race, PIR (trisection by percentage), cotinine (trisection by percentage), and the presence of smoker in the home were conducted to determine the association between BMI and asthma in specific subgroups. An interaction test was performed to assess whether individual characteristics influenced the association between BMI and asthma. Sensitivity analyses were performed using several approaches: BMI with 1% lowest and 1% highest values removed, BMI quintiles, and BMI z-score. A p-value less than 0.05 (two-sided) was considered statistically significant. In addition, in order to maximize statistical power and minimize bias, we also used the chained equation method based on 5 repetitions and the R MI procedure to compute missing data for missing values of covariates. All statistical analyses were performed with the statistical software R and EmpowerStats software.

Results

Baseline characteristics

Out of 107,622 NHANES participants enrolled from 1999 to 2020, 35,603 pediatric participants were included in the present study, as illustrated in the participant screening flowchart (Fig. 1).

Table 1 presents the weighted basic characteristics of participants categorized by their asthma status. Participants with asthma had a significantly higher mean age of 11.59 ± 4.67 years compared to those without asthma, who had a mean age of 10.97 ± 4.89 years ($p < 0.001$). The gender distribution also differed significantly between the two groups, with a greater percentage of males in the asthma group than the non-asthma group (56.76% vs. 49.59%, $p < 0.001$). Significant differences were observed in racial demographics, lead, cadmium,

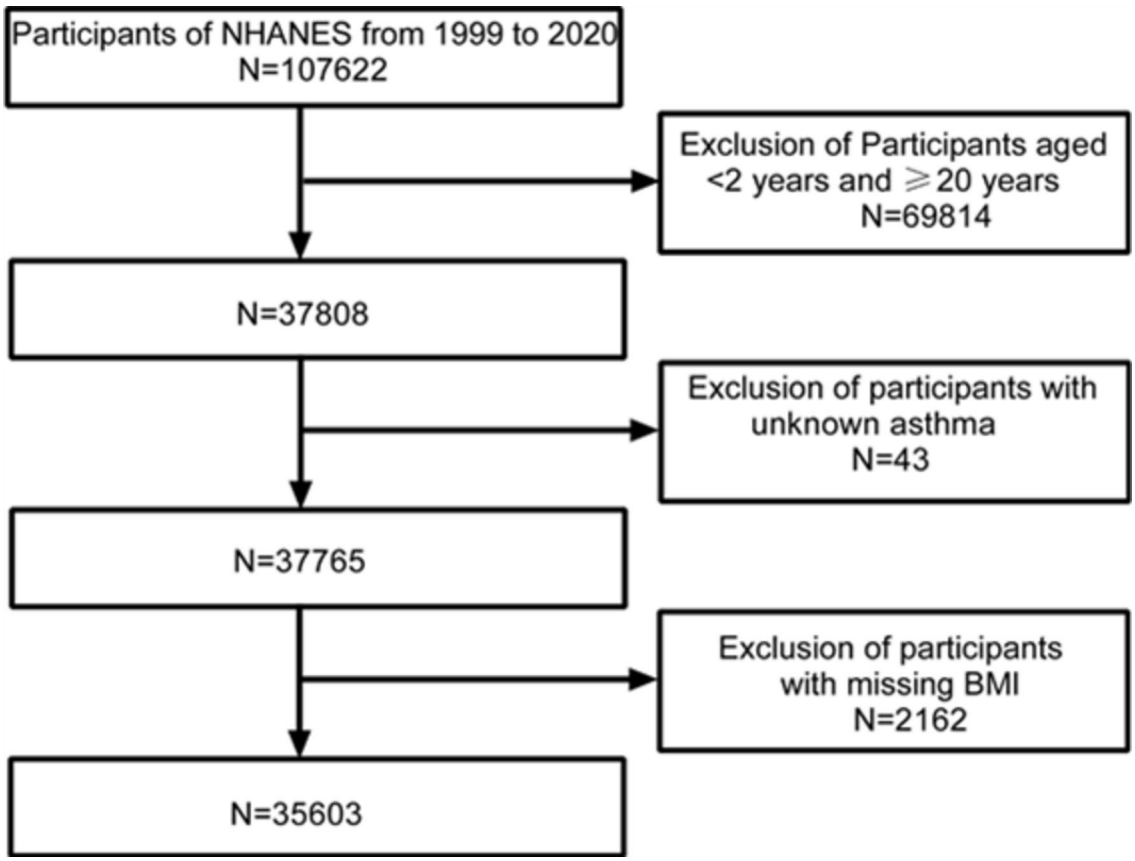

Fig. 1. Flowchart of the participants screening process for this study.

	Non-asthma (29595)	Asthma (6008)	*P*-value
Age (years)	10.97 ± 4.89	11.59 ± 4.67	< 0.001
Gender (%)			< 0.001
Male	49.59	56.76	
Female	50.41	43.24	
Race (%)			< 0.001
Mexican American	28.31	19.17	
Other Hispanic	7.71	9.60	
Non-Hispanic White	27.72	26.23	
Non-Hispanic Black	26.50	35.60	
Other Race	9.75	9.39	
PIR	2.02 ± 1.53	1.99 ± 1.53	0.066
Lead (umol/L)	0.06 ± 0.06	0.06 ± 0.06	< 0.001
Cadmium (umol/L)	1.88 ± 1.97	1.97 ± 2.27	< 0.001
Cotinine (ng/mL)	7.68 ± 39.42	10.80 ± 46.73	< 0.001
Smoker in the home (%)			< 0.001
No	78.28	75.74	
Yes	20.72	24.26	

Table 1. Weighted basic characteristics of participants. Continuous variables are expressed as weighted mean ± SD. Categorical variables are expressed as weighted %. *SD* standard deviation, *BMI* body mass index, *PIR* Poverty income ratio.

	Variant (kg/m^2)	Model 1 (35603)	Model 2 (35603)	Model 3 (25452)
	Mean ± SD (min–max)	OR (95%CI) P-value	OR (95%CI) P-value	OR (95%CI) P-value
BMI	20.96 ± 6.00 (7.99–72.60)	1.04 (1.03,1.04) < 0.001	1.04 (1.04, 1.04) < 0.001	1.03 (1.04, 1.04) < 0.001
BMI quartile				
Q1	15.19 ± 0.85 (7.99–16.43)	Reference	Reference	Reference
Q2	17.87 ± 0.89) (16.44–19.49)	1.25 (1.15, 1.37) < 0.001	1.28 (1.17, 1.40) < 0.001	1.25 (1.12, 1.40) < 0.001
Q3	21.41 ± 1.18 (19.50-23.69)	1.49 (1.37, 1.62) < 0.001	1.57 (1.43, 1.74) < 0.001	1.60 (1.42, 1.80) < 0.001
Q4	29.25 ± 5.37 (23.70–72.60)	1.85 (1.71, 2.01) < 0.001	2.00 (1.81, 2.22) < 0.001	1.97 (1.75, 2.23) < 0.001
P for trend		< 0.001	< 0.001	< 0.001

Table 2. Association between BMI and asthma in different models. *BMI* body mass index, *SD* standard deviation, *OR* odds ratio, *CI* confidence interval. Model 1 unadjusted. Model 2 adjusted for age, gender and race. Model 3 adjusted for age, gender, race, poverty income ratio, lead, cadmium, cotinine, and smoker in the home.

cotinine, and the presence of smokers in the home (all $p < 0.001$). However, no significant disparities were found in PIR between the two groups.

Association between BMI and asthma

The current study examined the association between BMI and the risk of asthma using various statistical models (Table 2). When analyzing BMI as a continuous variable, the results consistently showed a significant positive association across all models. Specifically, a one-unit increase in BMI was associated with a 4% higher odds of asthma in Model 1 (OR 1.04, 95% CI 1.03–1.04, $p < 0.001$), and this association remained unchanged after adjusting for demographics in Model 2 (OR 1.04, 95% CI 1.04–1.04, $p < 0.001$). Further adjusting for additional confounding factors was made in Model 3 (OR 1.03, 95% CI 1.02–1.04, $p < 0.001$). The analysis based on BMI quartiles revealed a clear dose-response relationship, where individuals in the higher BMI quartiles had progressively higher odds of asthma. Compared to those in the first quartile (Q1), participants in the second (Q2), third (Q3), and fourth (Q4) quartiles had 25%, 49%, and 85% increased odds of asthma, respectively, in the non-adjusted model. This trend remained statistically significant across all three models (p for trend < 0.001).

Smooth curve fitting revealed that the relationship between BMI and asthma was not entirely linear after adjusting for relevant confounders (Fig. 2). Table 3 demonstrates the results of the piece-wise linear regression analysis, which identified an inflection point at a BMI of 21 kg/m^2. Below this inflection point, the odds ratio

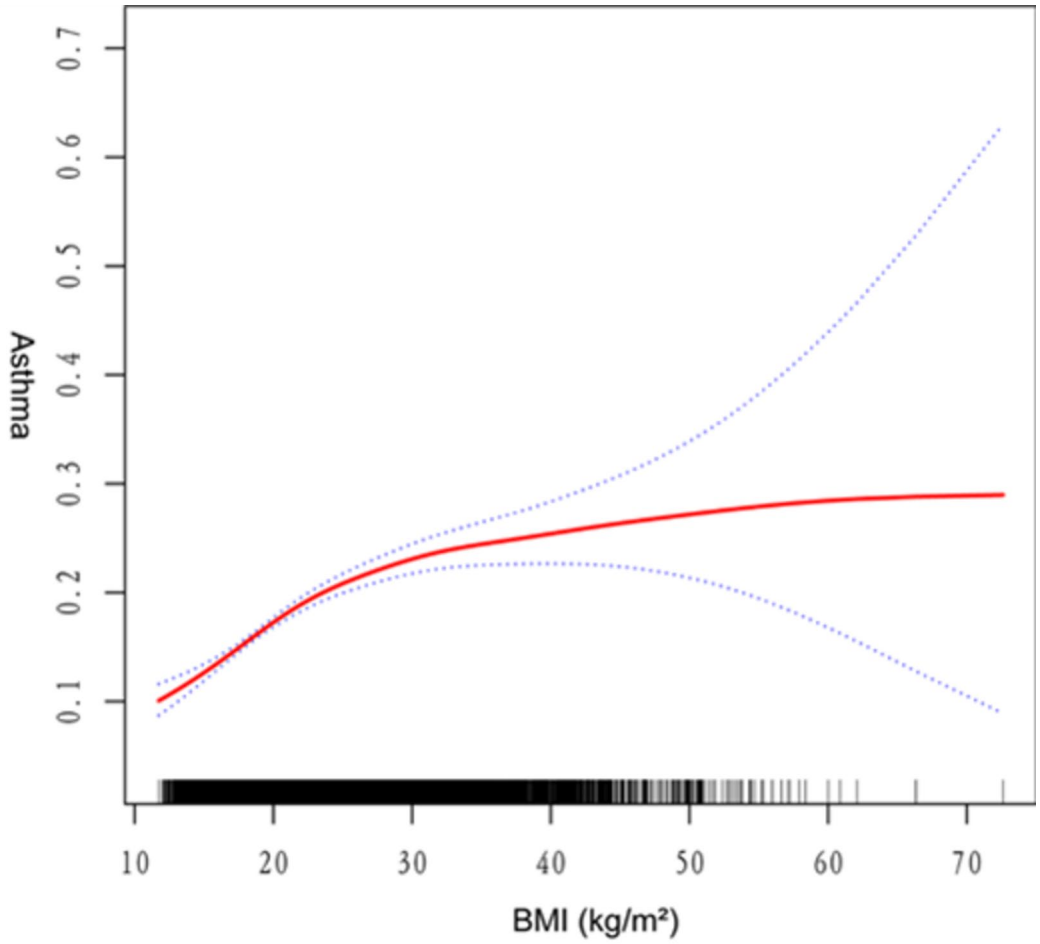

Fig. 2. Smooth curve fitting for BMI and asthma. The solid line and dashed line represent the estimated values and their corresponding 95% confidence interval, respectively. Adjusted for age, gender, race, poverty income ratio, lead, cadmium, cotinine, and smoker in the home.

	OR (95% CI)	*P* value
Model I		
Linear effect	1.03 (1.03, 1.04)	< 0.001
Model II		
Inflection point (K)	21	
< K	1.10 (1.07, 1.12)	< 0.001
> K	1.02 (1.01, 1.03)	< 0.001
Log likelihood ratio		< 0.001

Table 3. Threshold effect analysis of BMI and asthma using piece-wise linear regression. *BMI* body mass index, *OR* odds ratio, *CI* confidence interval. Model I, linear analysis; Model II, non-linear analysis. Adjusted for age, gender, race, poverty income ratio, lead, cadmium, cotinine, and smoker in the home.

for asthma was markedly higher (OR 1.10, 95% CI 1.07–1.12, $p < 0.001$), indicating a steeper increase in asthma risk at lower BMIs. In contrast, above the inflection point, the odds ratio decreased (OR 1.02, 95% CI 1.01–1.03, $p < 0.001$), suggesting a weaker association between BMI and asthma in higher BMI ranges.

Subgroup analysis

To further explore the relationship between BMI and asthma risk, a comprehensive subgroup analysis was conducted, adjusting for a range of potential confounding factors (Fig. 3). Our analysis revealed a consistent positive association between BMI and asthma across all examined subgroups, with ORs ranging from 1.025 to 1.059. Stratified by age, the association was stronger in participants aged ≤ 11 years (OR = 1.06, 95%CI: 1.05, 1.07) compared to those > 11 years (OR = 1.03, 95%CI: 1.02, 1.03), with a statistically significant interaction ($p < 0.001$).

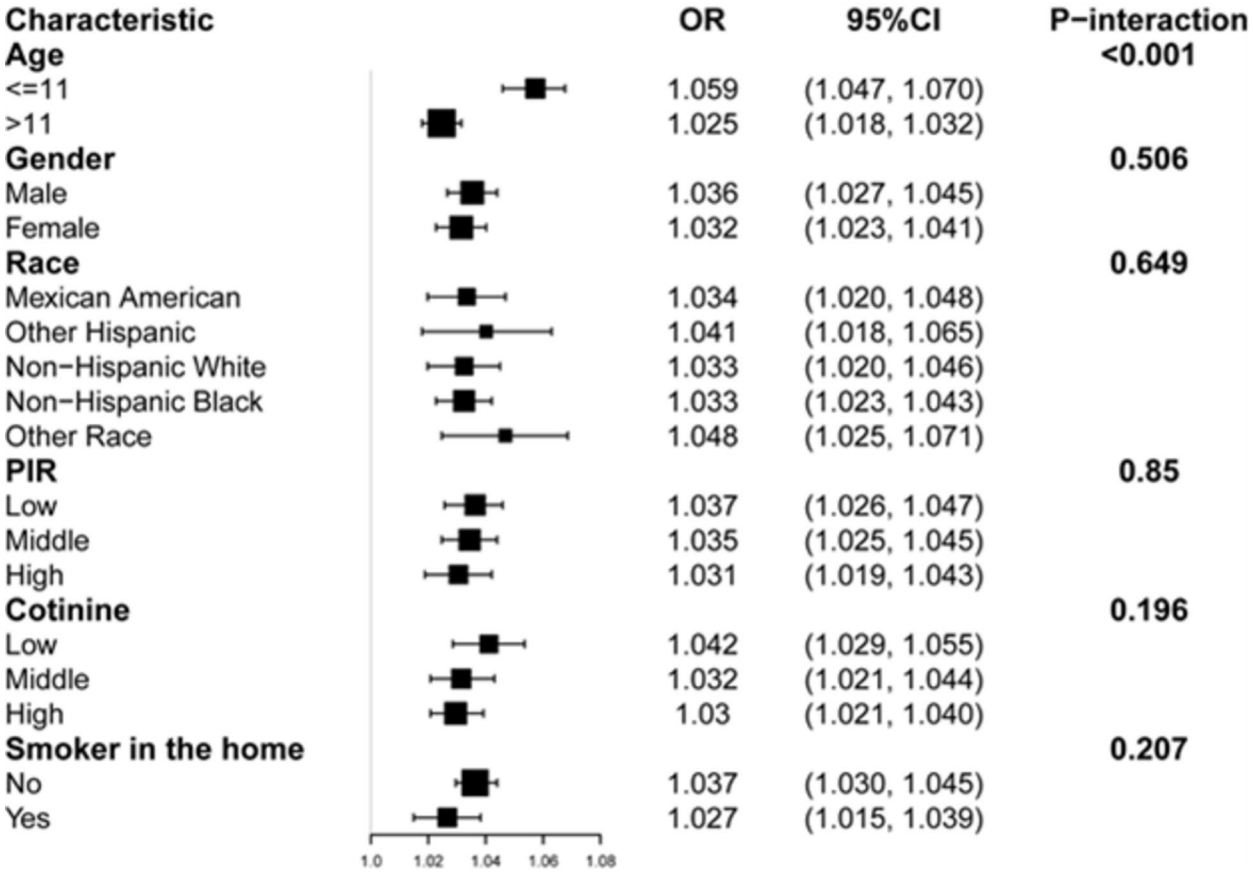

Fig. 3. Subgroup analysis of the association between BMI and asthma. Adjusted for age, gender, race, poverty income ratio, lead, cadmium, mercury, and smoker in the home except the subgroup variable.

The subgroup analyses, stratified by gender, race, poverty income ratio, cotinine levels, and the presence of smoker in the home, all showed consistent positive associations between BMI and asthma.

Sensitivity analyses

Of the 35603 cases, 25452 (71.5%) had complete data for all variables. The amount of missing data was counted for each covariate and the results are displayed in Table S1. No covariates had a missing amount of more than 30%. in order to maximize statistical power and minimize bias, we also used the chained equation method based on 5 repetitions and the R MI procedure to compute missing data for missing values of covariates. The result of a fully adjusted regression analysis on the dataset obtained by multiple interpolation estimation (OR = 1.04, 95%CI: 1.03, 1.04), $P < 0.001$)) showed similar result to that with complete cases data. The association between asthma and BMI was further examined using BMI quintiles, BMI z-score, BMI with 1% lowest and 1% highest values removed. The results, presented in Table S2, showed that BMI quintiles, BMI z-score, and BMI with extreme values removed remained significantly and positively associated with asthma across different regression models. Additionally, a smoothed curve fitting analysis was conducted using BMI with 1% lowest and 1% highest values removed. The result, shown in Figure S1, remained not entirely linear. Piece-wise linear regression analysis identified an inflection point of 20.9 kg/m^2, as presented in Table S2. In summary, the sensitivity analyses confirmed the stability of the results and the not entirely linear positive correlation between BMI and asthma.

Discussion

The study investigated the correlation between BMI and asthma in children, utilizing data from NHANES spanning 1999 to 2020. Our findings demonstrate a significant positive correlation between BMI and the risk of asthma. This relationship was consistent across different statistical models and remained significant after adjusting for demographic factors and potential confounders. Notably, a not entirely linear relationship was identified, with an inflection point at a BMI of 21 kg/m^2. Below this inflection point, the odds ratio for asthma was markedly higher (OR 1.10, 95% CI 1.07–1.12, $p < 0.001$), indicating a steeper increase in asthma risk at lower BMIs. In contrast, above the inflection point, the odds ratio decreased (OR 1.02, 95% CI 1.01–1.03, $p < 0.001$), suggesting a weaker association between BMI and asthma in higher BMI ranges. The comprehensive subgroup analysis, adjusting for potential confounding factors, revealed a consistent positive association between BMI and asthma across all examined subgroups. The result underscores the uniformity of this relationship across diverse demographic and environmental subgroups, suggesting the observed association is generalizable.

The concurrent rise in childhood obesity and asthma prevalence has become a significant public health concern, prompting an urgent need to understand their interplay[28]. As obesity is often measured by BMI, this research delves into the relationship between BMI and asthma risk in pediatric populations. The findings are consistent with several studies that have reported similar observations[23,29]. In our study, this association was observed across different logistic regression models, showing that each unit increase in BMI was associated with a 3 − 4% increase in the odds of developing asthma. This trend persisted even after adjusting for demographic factors and potential confounders. However, a study by Guibas et al. found BMI may not be as effective an indicator in preschool children, highlighting the complexity of the relationship and the need for age-specific assessment[19].

While BMI has been widely adopted as a practical tool for identifying overweight and obesity in children and adolescents, it has limitations. It is a relative measure that does not account for differences in muscle mass, bone density, and overall body composition[30]. Moreover, BMI does not distinguish between fat-free mass and fat mass, which can be problematic when assessing obesity in children, given their ongoing growth and development[31]. Additionally, the use of BMI percentiles based on population data may not be universally applicable across different countries and regions due to variations in growth patterns and standards of living[32]. Relying solely on BMI can also neglect other crucial factors such as diet, physical activity, and genetic predispositions that contribute to obesity and related health risks[33]. Therefore, this study examines BMI as a continuous variable to reduce the impact of different categorization methods on the accuracy of the results.

Our analysis revealed a not entirely linear association between BMI and asthma, identified through smoothed curve fitting. A segmented linear regression model showed a threshold effect, with an inflection point at a BMI of 21 kg/m^2. This indicates a steeper increase in asthma risk at lower BMIs and a weaker association as BMI rises beyond this threshold, a finding not previously reported. Interestingly, a prospective study by Ulrik et al. emphasizes a similar nonlinear link between childhood BMI and asthma admissions in early adulthood, with gender-specific associations[34]. For females, a higher childhood BMI is linked to increased asthma admission risks, while in males, it is the lower BMI category that is associated with greater risks in early adulthood[34]. This finding challenges the conventional understanding of the obesity-asthma link, suggesting that both ends of the BMI spectrum may confer different risks for asthma severity, and these risks are modulated by gender. But there is also evidence suggesting that obesity in childhood and adulthood is an independent risk factor for asthma at each of the corresponding time points in its life course[35]. In summary, these findings suggest a more complex relationship between BMI and asthma. The age-stratified analysis revealed a statistically significant interaction, with a stronger association between BMI and asthma in participants aged ≤ 11 years compared to those > 11 years. This suggests that BMI may have a more pronounced impact on asthma risk during early childhood. Potential explanations include the critical period of immune and pulmonary development in younger children, during which adiposity-related inflammation and metabolic dysregulation may significantly influence airway hyperresponsiveness and asthma pathogenesis[36,37]. Although our study did not stratify by gender within age groups, existing evidence indicates that prepubertal boys may be more susceptible to the effects of adiposity on asthma risk, possibly due to differences in airway geometry and hormonal factors[38,39]. The weaker association in older children may reflect the modulating effects of pubertal hormonal changes on the relationship between adiposity and asthma[40]. Further research is needed to clarify the mechanisms underlying this age-dependent association. The comprehensive subgroup analysis, adjusting for potential confounding factors, revealed a consistent positive association between BMI and asthma across all examined subgroups. The result underscores the uniformity of this relationship across diverse demographic and environmental factors.

However, our study has its limitations. The cross-sectional nature of our research restricts our ability to establish causality between BMI and asthma. Additionally, reliance on self-reported or proxy-reported data for asthma diagnosis could introduce bias, potentially impacting the accuracy of our estimates. The use of BMI as an adiposity measure is also a limitation, as it does not differentiate between fat and lean mass, which may have distinct associations with asthma[36]. The potential role of adipose tissue distribution, particularly central obesity[37], which could be more relevant to asthma risk, was not directly evaluated in our study. Moreover, our analysis did not consider all possible confounding factors, such as diet, physical activity, sleep patterns, and genetic predispositions, that could affect the BMI-asthma relationship. The generalizability of our findings is further constrained by the predominantly Caucasian demographic of our sample, as the prevalence and relationship between obesity and asthma may differ among diverse racial and ethnic groups, suggesting that our results may not be universally applicable. Smooth curve fitting and inflection point testing were not conducted separately for subgroups such as boys and girls or younger and older children, which limits our ability to determine whether the observed not entirely linear association between BMI and asthma risk varies across these groups. Future studies with larger sample sizes and more detailed phenotypic characterization are needed to explore these potential subgroup differences.

Conclusion

In conclusion, our study confirms a significant positive and not entirely linear association between BMI and the risk of asthma in children. Despite the robustness of our findings, the study's limitations, including its cross-sectional design and reliance on self-reported data, warrant further investigation. Future research should utilize longitudinal approaches and incorporate a wider array of obesity metrics, alongside lifestyle and genetic factors, to enhance our comprehension of this relationship. Our study's results enhance the existing literature by providing a comprehensive understanding of the BMI-asthma relationship in pediatric populations, underscoring the necessity of considering BMI in both asthma research and clinical practice.

Data availability

The study data were derived from NHANES which can be freely obtained online (https://www.cdc.gov/nchs/nhanes/index.htm). All data generated or analyzed during this study are included in this published article.

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Author contributions

XS and WF contributed to the study design and critically revised the manuscript for important intellectual content. CF and JZ contributed to the data collection. CF, JZ and WF contributed to the data analysis and drafted the manuscript. All authors have participated sufficiently in the work to take public responsibility for the content and approved the final version of the manuscript to be published.

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Declarations

Competing interests

The authors declare no competing interests.

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