

Biomolecules and Biomedicine

ISSN: 2831-0896 (Print) | ISSN: 2831-090X (Online)

Journal Impact Factor® (2024): 2.2

CiteScore® (2024): 5.2

www.biomolbiomed.com | blog.bjbms.org

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#### **META-ANALYSIS**

Xing et al: Obesity and allergic rhinitis in children

# Childhood obesity and allergic rhinitis: A meta-analysis

## Xinxin Xing, Sihao Zhu, Guang Zhou, Yubo Ma, Hai Wang\*

Department of Pediatrics II, the First Affiliated Hospital of Heilongjiang University of Chinese Medicine, Harbin, China

\*Correspondence to Hai Wang: drwanghaihlj23@hotmail.com

DOI: https://doi.org/10.17305/bb.2025.12982

#### **ABSTRACT**

Allergic rhinitis (AR) is a prevalent chronic condition in childhood, and its increasing incidence has prompted research into potential associations with modifiable factors such as obesity. This meta-analysis aimed to assess the multivariate-adjusted relationship between childhood obesity and AR. A systematic search was conducted across PubMed, Embase, and Web of Science for observational studies that reported on the association between obesity and AR in children. Only studies that included multivariate adjustments for at least age and sex were considered. Random-effects models were employed to pool odds ratios (ORs) with 95% confidence intervals (CIs), accounting for heterogeneity. Fifteen cross-sectional studies comprising 23 datasets involving a total of 569,856 children were included in the analysis. The overall results indicated that obesity was not significantly associated with AR (adjusted OR: 1.04, 95% CI: 1.00–1.09; p = 0.08;  $I^2 = 24\%$ ). However, subgroup analyses revealed a significant association in Western countries (OR: 1.12, 95% CI: 1.00–1.24; p = 0.04;  $I^2 = 0\%$ ), while no significant association was found in Asian countries (OR: 1.04, 95% CI: 0.97–1.12; p = 0.27;  $I^2 = 52\%$ ). Notable associations were identified in studies utilizing national or international BMI cutoffs (OR: 1.06, 95% CI: 1.01–1.10; p = 0.02) and those with physician-diagnosed AR (OR: 1.07, 95% CI: 1.02–1.13; p =0.006), but not in studies employing the 95th percentile BMI definition or ISAACbased AR diagnosis. No significant differences were observed based on age or sex. Meta-regression analysis indicated that age, sex, and study quality score did not significantly influence the results (p all > 0.05). Egger's test revealed no evidence of publication bias (p = 0.43). In conclusion, while no significant overall association between childhood obesity and AR was found, subgroup analyses suggest potential links within specific populations and under particular methodological definitions. These findings should be interpreted with caution, and further longitudinal studies are necessary to determine whether preventive strategies aimed at reducing childhood obesity may also impact allergic outcomes.

Keywords: Allergic rhinitis, obesity, children, risk factor, meta-analysis.

#### INTRODUCTION

Allergic rhinitis (AR) is a prevalent chronic inflammatory disease of the upper airway in children, characterized by sneezing, nasal congestion, rhinorrhea, and nasal itching, often accompanied by ocular symptoms (1, 2). It affects up to 40% of children worldwide and its prevalence has been rising, particularly in urbanized and industrialized regions (3, 4). Although not life-threatening, AR imposes a significant burden on children's quality of life, leading to sleep disturbances, impaired cognitive performance, reduced school attendance, and behavioral problems (5, 6). AR frequently coexists with other atopic conditions such as asthma and eczema, further amplifying its clinical impact (7). While genetic predisposition plays a critical role, environmental exposures, air pollution, secondhand smoke, and socioeconomic factors have all been implicated in its development (8, 9). Identifying modifiable risk factors for AR is essential to support early prevention and reduce long-term health consequences in children.

Obesity, typically defined as a body mass index (BMI) at or above the 95th percentile for age and sex, has become an increasing concern in the pediatric population, with over 340 million children and adolescents affected globally (10, 11). Childhood obesity is associated with adverse physical and psychosocial outcomes and often tracks into adulthood, increasing the risk of chronic diseases such as type 2 diabetes and cardiovascular disorders (12, 13). In recent years, obesity has also been hypothesized to contribute to allergic diseases. Adipose tissue acts as an endocrine organ releasing pro-inflammatory cytokines and adipokines, which may promote systemic inflammation, alter T-helper cell balance, and impair epithelial barrier integrity in the airways, thereby increasing susceptibility to allergic responses (14, 15). Despite the biological plausibility, epidemiological findings on the relationship between obesity and AR in children remain inconsistent. Some studies suggest a positive association (16, 17), while others report no significant link (18-30), potentially due to differences in population characteristics, obesity definitions, or AR diagnostic methods. Notably, obesity in children can be defined either by age- and sex-specific ≥ 95th percentile BMI thresholds or by fixed international BMI cut-offs (11), which may capture different populations and contribute to variability across studies. To address these uncertainties, we performed a systematic review and metaanalysis to assess the association between obesity and AR in children, and to explore possible effect modifiers through predefined subgroup analyses.

#### **MATERIAL AND METHODS**

This study followed the PRISMA 2020 (31, 32) and Cochrane Handbook guidelines (33) for conducting systematic reviews and meta-analyses, covering study design, data collection, statistical methods, and interpretation of results. The protocol was also registered in PROSPERO under the ID CRD420251108821.

#### **Database search**

To identify studies pertinent to this meta-analysis, we searched PubMed, Embase, and Web of Science databases using an extensive array of search terms, which involved the combined terms of (1) "obesity" OR "obese" OR "overweight" OR "body mass index" OR "BMI"; (2) "allergic rhinitis" OR "atopic rhinitis" OR "allergic rhinitides" OR "atopic rhinitides"; and (3) "children" OR "pediatric" OR "paediatric" OR "adolescents". The search was restricted to studies on human subjects and included only full-length articles published in English in peer-reviewed journals. Grey literature was not included, as most such reports are not peer-reviewed and may reduce the reliability of findings. We also manually checked the references of related original and review articles to find additional relevant studies. The search covered all records from database inception up to May 26, 2025. The detailed search strategy for each database is shown in **Supplemental File 1**.

## Study eligible criteria

We applied the PECO framework to define the inclusion criteria:

P (patients): Children and adolescents under the age of 18 years. Eligible participants were those from the general pediatric population or specific subgroups (e.g., schoolaged children, adolescents) who have been assessed for both body weight status (obesity) and AR.

E (exposure): The exposure to be reviewed in this meta-analysis was childhood obesity, defined using standardized criteria such as BMI at or above the 95th percentile for age and sex, or equivalent fixed cut-off values based on national or international references. Only studies that clearly distinguish and report on obesity as a separate category were included.

C (comparison): The comparator group of the meta-analysis consists of children with normal weight, defined according to BMI criteria as falling below the 85th percentile for age and sex or within the normal BMI range based on national or international growth references.

O (outcome): The main outcome of interest was the presence of AR in children, as defined by physician diagnosis, validated questionnaires (e.g., the International Study of Asthma and Allergies in Childhood [ISAAC] criteria), or self-reported diagnosis confirmed by symptoms. Only studies reporting the multivariate-adjusted association between obesity and AR in children were included, at least for age and sex.

S (study design): Observational studies, such as cohort studies, case-control studies, and cross-sectional studies.

We excluded reviews, editorials, other meta-analyses, studies including adult population, studies evaluating influence of overweight only or combined overweight with obesity, studies not reporting AR as outcome, or studies reporting univariate data only. If studies had overlapping populations, we included the one with the largest sample size in the meta-analysis.

## Study quality evaluation

Two authors independently performed the literature search, study selection, quality assessment, and data extraction. Disagreements were resolved by discussion with the corresponding author. Study quality was assessed using a modified version of the Newcastle–Ottawa Scale (NOS) adapted for cross-sectional studies (34), as applied in prior meta-analyses (35, 36). The rubric covered selection (4 items), comparability (2 items), and outcome (3 items), with a maximum of 9 points. Details of the specific items and scoring criteria are provided in **Supplemental File 3**. Studies scoring ≥7 were considered high quality.

#### **Data collection**

The data collected for analysis included the study details (author, year, study country, and design), participant characteristics (source of the children, age range, mean age, and the proportion of boys), definition and the number of children with obesity, methods for the diagnosis of AR, numbers of children with AR, and covariates adjusted in the regression models.

## Statistical analysis

We used odds ratios (ORs) and 95% confidence intervals (CIs) to assess the association between obesity and AR in children, comparing between children with obesity and normal weight. ORs and standard errors were directly extracted or calculated from 95% CIs or p values, then log-transformed to stabilize variance and normalize the data (33). If multiple ORs were reported from different models, we used the one with the most complete adjustment. Heterogeneity was assessed using the Cochrane Q test and I<sup>2</sup> statistic (37), with a p value < 0.10 suggesting significant heterogeneity and I<sup>2</sup> values of < 25%, 25–75%, and > 75% indicating low, moderate, and high heterogeneity. A random-effects model was used to pool the data, accounting for heterogeneity between studies (33). The primary analyses applied the DerSimonian-Laird (DL) method, and sensitivity analyses were also performed using the restricted maximum likelihood (REML) estimator (33). In addition, a sensitivity analysis was conducted by collapsing sex-stratified datasets into one effect size per study to account for potential within-study clustering. Sensitivity analysis by excluding one dataset at a time was performed to evaluate the robustness of the finding (33). Predefined subgroup analyses were conducted based on study countries (Asian vs. western countries), average age of the children, sex distribution, definition of obesity (national/international BMI cutoffs vs. BMI ≥ the 95th percentile for age and sex), methods for diagnosis of AR (ISAAC criteria or physician diagnosed), and NOS scores of the included studies. Medians of continuous variables were used to divide subgroups evenly. Further univariate meta-regression analysis was performed to evaluate if mean age, proportion of male, or NOS could affect the association between obesity and AR (37). Publication bias was assessed using funnel plots and visual inspection for asymmetry, along with Egger's test (38). The certainty of the evidence was assessed using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach, considering risk of bias, inconsistency, indirectness, imprecision, and publication bias (33). All analyses were performed using RevMan (Version 5.3; Cochrane Collaboration, Oxford, UK) and Stata (Version 17.0; Stata Corporation, College Station, TX, USA).

## **RESULTS**

## **Study inclusion**

The study selection process is shown in **Figure 1**. We first identified 1129 records from the three databases. After removing 311 duplicates, 818 articles were screened by title and abstract. Of these, 783 were excluded for not meeting the aims of the meta-analysis. The full texts of the remaining 35 articles were reviewed by two independent authors, and 20 were excluded for various reasons (see **Figure 1**). In the end, 15 studies were included in the quantitative analysis (16-30).

## **Summary of study characteristics**

Table 1 summarizes the characteristics of the 15 cross-sectional studies included in this meta-analysis, which were published between 2007 and 2025 and conducted across diverse regions including Spain, Japan, Canada, Taiwan (China), the United States, Korea, Poland, China, and multiple international centers. These studies involved a combined total of 569,856 children and adolescents. Participants ranged in age from 2 to 18 years, with mean ages between 6.5 and 15.5 years. All included studies adopted a cross-sectional design, and obesity was primarily defined using the age- and sex-specific  $\geq$  95th percentile BMI cutoffs (19, 21, 24, 26, 27, 29, 30), international BMI standards (18, 20, 22, 23, 25), or country-specific thresholds (e.g.,  $\geq$  25 kg/m<sup>2</sup> in Korea,  $\geq$  23 kg/m<sup>2</sup> in Taiwan) (16, 17, 28). The number of obese children in each study varied widely, ranging from 130 to 14,452. AR was most frequently diagnosed based on the ISAAC questionnaire in 8 studies (18-23, 25, 26), with additional methods including physician diagnosis in six studies (16, 17, 27-30), and via International Classification of Disease codes in another study (24). Some studies also incorporated symptom criteria and/or objective measures such as positive IgE tests (27, 30). The number of AR cases ranged from 295 to over 25,000 across studies. All studies adjusted for at least age and sex in their analyses. Many studies additionally accounted for various confounders such as household smoking, parental education, physical activity, environmental exposures (e.g., traffic or secondhand smoke), socioeconomic indicators, and other allergic conditions. As shown in **Table 2**, the methodological quality of included studies, assessed using the NOS scale, was high, with NOS varying from seven to nine. Six studies achieved a full score of 9, reflecting appropriate case and control selection, adequate control for age, sex, and

other confounders, and consistent exposure and outcome ascertainment methods (16, 18-21, 27). Other nine studies scored 7 or 8 (17, 22-26, 28-30), mainly due to limitations in case definition, incomplete adjustment for additional confounders, or lack of representativeness. Despite variability in design detail, all studies met criteria for valid AR ascertainment and comparable assessment methods between groups, supporting the reliability of the pooled estimates.

## Association between obesity and AR in children

Because five studies reported the association between obesity and AR in boys and girls separately (18, 19, 23, 27, 28), and two studies reported the association in two independent age groups (23, 26), these stratified datasets were independently included in the meta-analysis, resulting in 23 datasets. This approach allowed more detailed subgroup analyses. Pooled results showed that overall, obesity was not associated with AR in children (adjusted OR: 1.04, 95% CI: 1.00 to 1.09, p = 0.08; Figure 2) with mild heterogeneity observed (p for Cochrane Q test = 0.15;  $I^2 = 24\%$ ). In addition, a sensitivity analysis using the REML model also showed similar results (adjusted OR: 1.04, 95% CI: 1.00 to 1.09, p = 0.07;  $I^2 = 28\%$ , Supplemental Figure 1). We further collapsed sex-stratified datasets into a single effect size per study, which produced results similar to the main analysis (adjusted OR: 1.05, 95% CI: 1.00 to 1.09, p = 0.05;  $I^2 = 14\%$ , Supplemental Figure 2), indicating that potential withinstudy clustering did not materially influence our conclusions. In addition, sensitivity analysis omitting one dataset at a time showed consistent results (OR: 1.03 to 1.06, p all > 0.05). Moreover, sensitivity analysis limited to studies with NOS  $\ge 8$  showed similar results (adjusted OR: 1.03, 95% CI: 0.97 to 1.08, p = 0.36;  $I^2 = 36\%$ ). Further subgroup analyses suggested a significant association between obesity and AR in children from Western countries (OR: 1.12, 95% CI: 1.00 to 1.24, p = 0.04;  $I^2 = 0\%$ ), but not in children from Asian countries (OR: 1.04, 95% CI: 0.97 to 1.12, p = 0.27;  $I^2$ = 52%; Figure 3A). However, similar results were observed in studies of children with mean ages < or  $\ge 11$  years (p for subgroup difference = 0.82; Figure 3B) and between girls and boys (p for subgroup difference = 0.95; Figure 4A). Moreover, a significant association between obesity and AR was observed in studies with obesity defined according to the national/international BMI cutoffs (OR: 1.06, 95% CI: 1.01 to 1.10, p = 0.02;  $I^2 = 17\%$ ), but not in studies with obesity defined as BMI  $\geq$  age- and sex-specific 95th percentile (OR: 1.02, 95% CI: 0.92 to 1.14, p = 0.68;  $I^2 = 28\%$ ;

**Figure 4B**). Interestingly, a significant association between obesity and AR was observed in studies with physician-diagnosed AR (OR: 1.07, 95% CI: 1.02 to 1.13, p = 0.006;  $I^2 = 13\%$ ), but not in studies with AR diagnosed according to the ISAAC questionnaire (OR: 1.01, 95% CI: 0.95 to 1.08, p = 0.74;  $I^2 = 25\%$ ; **Figure 5A**). Moreover, a significant association between obesity and AR was observed in studies with NOS = 7 (OR: 1.16, 95% CI: 1.02 to 1.31, p = 0.02;  $I^2 = 0\%$ ), but not in studies with NOS = 8 (OR: 1.02, 95% CI: 0.95 to 1.10, p = 0.52;  $I^2 = 30\%$ ) or 9 (OR: 1.02, 95% CI: 0.93 to 1.12, p = 0.68;  $I^2 = 44\%$ ; **Figure 5B**). Finally, results of univariate meta-regression analysis did not support that mean age, proportion of male, or NOS have significant influence on the association between obesity and AR (p all > 0.05; **Table 3**). According to the GRADE framework, the certainty of the evidence was rated as low, reflecting the cross-sectional study design and imprecision of effect estimates, although heterogeneity, indirectness, and publication bias were not considered serious concerns (**Supplemental Table 1**).

#### **Publication bias**

Funnel plots for the meta-analyses of the association between obesity and AR in children are shown in **Figure 6**. The plots appeared symmetrical, suggesting a low risk of publication bias. Egger's test also showed no evidence of publication bias (p = 0.43).

## **DISCUSSION**

This meta-analysis of over half a million children across 15 studies found no significant overall association between obesity and AR after adjusting for age, sex, and other relevant covariates. However, subgroup analyses revealed a stronger association in children from Western countries, in studies using national/international BMI cutoffs, in studies with physician-diagnosed AR, and in those with a NOS score of 7. These findings suggest that the relationship between obesity and AR in children may vary by geographic region, obesity and AR definitions, and study design quality. Our results refine and add important context to previous syntheses. An early meta-analysis published in 2020 reported a modest but significant association between obesity or overweight and AR in children (OR: 1.09; 95% CI: 1.04–1.14), although that analysis combined children and adults, and included both overweight and obese categories, which may have inflated the pooled estimates (39). More recently, Yeo et

al. (40) conducted a large-scale meta-analysis including both children and adults with abnormal BMI (overweight and obesity) and found no significant association with AR in any subgroup. Our meta-analysis differs from these meta-analyses by strictly including children and adolescents, focusing exclusively on obesity (not overweight), requiring multivariate adjustment (at least for age and sex), and performing detailed subgroup analyses to explore sources of heterogeneity. Compared to prior work, this study provides more nuanced insights into specific subgroups where the association may be more pronounced or absent.

The stronger association observed in Western countries may be explained by several factors. Lifestyle and dietary patterns in Western populations, including higher consumption of processed foods, sedentary behaviors, and greater prevalence of obesity-related metabolic inflammation, could amplify immune dysregulation and increase susceptibility to AR (41). Environmental exposures also differ: Western children may have greater exposure to indoor allergens, pollutants, or urban environments that interact with obesity-related inflammation to exacerbate airway responses. By contrast, Asian countries may report weaker associations partly due to stricter diagnostic thresholds for AR, differences in healthcare access, and cultural variations in healthcare-seeking behavior, which can lead to underdiagnosis (42). Genetic differences, distinct microbiome profiles, and protective early-life exposures (e.g., traditional diets, lower prevalence of early-life obesity) may also attenuate the observed association in Eastern populations. These factors together may explain the regional heterogeneity observed in our subgroup analysis. The definition of obesity appeared to impact the observed relationship with AR. Studies using fixed national or international BMI cutoffs (e.g.,  $\geq 25 \text{ kg/m}^2$ ) showed a significant association, while those using the age- and sex-specific  $\geq$  95th percentile did not. This may reflect measurement inconsistencies or differences in the populations captured by each approach. The fixed cutoffs may classify more children as obese in certain populations, particularly in adolescents where BMI can plateau, thereby increasing statistical power to detect associations (43). Conversely, the percentile-based method may misclassify children near the threshold and dilute the effect (43). Our findings also suggest that AR diagnostic methods influence observed associations. Studies using physician-diagnosed AR reported a significant association with obesity, while those using the ISAAC questionnaire did not. Physician diagnosis may incorporate clinical judgment, symptom severity, and objective evidence (e.g., IgE testing),

providing a more accurate AR case definition (44). The ISAAC questionnaire, while validated, may be more susceptible to recall bias and variability in parental reporting (44). This discrepancy underscores the importance of standardized and objective AR diagnosis in future epidemiologic studies. Interestingly, the association was strongest in studies with an NOS score of 7 but not significant in studies with scores of 8 or 9. However, the between-subgroup p value was not significant, and meta-regression did not support NOS score as a moderator. Given that all included studies were rated as high quality (NOS  $\geq$  7), this apparent paradox is unlikely to reflect a genuine quality gradient and may instead arise from chance variation or sample characteristics. Thus, while subgroup patterns are noted, our overall conclusion rests on consistently high-quality evidence across all studies.

Several biological mechanisms may underlie the potential link between obesity and AR in children. Adipose tissue functions as an endocrine organ that secretes proinflammatory cytokines such as tumor necrosis factor-alpha, interleukin-6, and leptin, which can amplify systemic inflammation and immune dysregulation (45). These mediators may alter the balance between T-helper 1 and T-helper 2 immune responses, promote eosinophilic inflammation, and compromise epithelial barrier integrity in the airway mucosa (46). Obesity-related changes in lung mechanics, such as reduced functional residual capacity and altered nasal airflow, may further predispose obese children to airway inflammation and allergen sensitization (47). In addition, recent experimental work demonstrated that modulation of the toll-like receptor 4 /mitogenactivated protein kinase/nuclear factor kappa B signaling pathway plays a key role in AR pathogenesis, with chlorogenic acid shown to exert therapeutic effects through this mechanism (48). These immunologic and physiologic pathways provide a plausible foundation for the observed association between obesity and AR, especially in children with coexisting atopic conditions.

This study has several strengths. It represents a comprehensive and up-to-date synthesis focusing exclusively on childhood obesity and AR in children. By requiring multivariable adjustment (at least for age and sex), our analysis minimizes confounding and complements recent broader meta-analyses that combined children and adults. We applied stringent inclusion criteria, conducted extensive subgroup analyses, and assessed heterogeneity and publication bias using established methods. Nonetheless, several limitations should be acknowledged. First,

although our protocol was registered in PROSPERO, this occurred after the completion of the database search, meaning the registration was not strictly prospective. While the review protocol was defined in advance and adhered to, posthoc registration may raise concerns about selective reporting and should be considered when interpreting the subgroup findings. Second, the cross-sectional design of all included studies precludes causal inference; reverse causation remains possible, where AR or its treatment influences physical activity and weight (49). Although our inclusion criteria permitted longitudinal designs, no eligible cohort or case-control studies were identified, and the evidence base is therefore limited to cross-sectional studies. Third, the lack of individual participant data limited our ability to explore the effects of dose-response relationships, detailed age stratification (e.g., preschoolers vs. adolescents), or coexisting conditions such as asthma. In addition, heterogeneity in obesity and AR definitions may have influenced the findings, and residual confounding from unmeasured factors—such as diet, vitamin D status, genetic susceptibility, or allergen exposure—cannot be excluded. Also, the majority of studies relied on self-reported or questionnaire-based data, which may be subject to recall and misclassification biases. Another limitation is that some included studies reported stratified results by age or sex, which we treated as independent datasets to enable more detailed subgroup analyses. While this is a common approach in meta-analyses, it may not fully account for within-study clustering. Nevertheless, as only a few studies contributed stratified data and leave-one-out sensitivity analyses showed stable findings, the potential influence on variance estimates is likely minimal. Moreover, the subgroup and meta-regression analyses were based on study-level covariates rather than individual participant data, which may introduce ecological bias. Therefore, the absence of significant moderator effects should be interpreted with caution. In addition, we could not evaluate adjustment depth (e.g., number or type of covariates included) through meta-regression because the covariates varied substantially across studies. This variability limits our ability to formally assess whether differences in confounder adjustment contributed to heterogeneity. Also, the exclusion of overweight individuals may limit the generalizability of findings, though it strengthens the specificity of our exposure definition. Finally, our restriction to English-language, peer-reviewed publications and the exclusion of grey literature may have introduced language or publication bias. Although Egger's test did not indicate significant publication bias (p = 0.43), this possibility cannot be entirely excluded.

Clinically, these findings suggest that obesity may contribute to AR risk in certain pediatric populations, particularly in Western countries or when using standardized diagnostic criteria. However, given the non-significant overall association and the cross-sectional design of all included studies, causal inferences cannot be made. Thus, while preventive strategies against childhood obesity remain important for many health outcomes, their potential to reduce allergic burden requires confirmation in longitudinal and interventional studies. In addition, the modest effect sizes observed may underscore the need for multifactorial approaches in AR prevention and management. Future research should prioritize large-scale, prospective cohort studies with standardized definitions of both obesity and AR, detailed phenotyping of allergic disease, and adjustment for a broader range of confounders. Investigating the role of obesity interventions in AR prevention or symptom control, as well as exploring underlying molecular mechanisms through systems biology or biomarker studies, could offer novel insights.

#### **CONCLUSION**

In conclusion, childhood obesity was not significantly associated with overall AR risk; however, subgroup analyses indicated significant associations in studies using national/international BMI cut-offs and physician-diagnosed AR. These findings suggest that methodological differences and population context may influence observed associations, underscoring the need for longitudinal confirmation.

### **GAMER** statement

The authors declare that no generative AI tools were used in the writing, editing, figure/table preparation, or reference management of this manuscript.

**Conflicts of interest:** The authors declare no competing interests.

**Funding:** This study is supported by Research Project of Heilongjiang Provincial Health Commission (No. 20240606010049).

**Data availability:** All data generated or analyzed during this study are included in this published article.

Submitted: July 21, 2025

Accepted: September 16, 2025

Published online: September 24, 2025

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## TABLES AND FIGURES WITH LEGENDS

**Table 1. Characteristics of the included studies** 

Study	Country	Design	Participant characterist ics	No. of participa nts	Age range s (years	Mean age (years	Boy (%)	Definition of obesity	No. of childre n with obesity	Diagnosis of AR	No. of children with AR	Variables adjusted
Garcia- Marcos 2007	Spain	CS	Schoolchild ren	20160	6~7	6.5	NR	Internatio nal BMI cut-offs	NR	ISAAC questionnair e (sneezing/ru nny nose + itchy/watery eyes)	1446	Age, sex, maternal smoking, siblings, exercise, Mediterranean diet score
Kusunoki 2008	Japan	CS	Schoolchild ren	45520	7~15	10.9	50.3	BMI ≥95th percentile (age/sex- specific)	NR	ISAAC- based questionnair e (sneezing/ru nny nose +	9888	Age, sex, birth order, other allergic diseases

Wang 2010	Canada	CS	Schoolchild	8334	13~1	13.4	46.7	Internatio nal BMI	330	nasal obstruction/i tching)  ISAAC criteria (current rhinoconjun	1572	Age, sex, region, birthplace, ethnicity, maternal education, siblings, smoking,
			ren		4			cut-offs		ctivitis: sneezing/ru nny nose + itchy eyes)		traffic exposure, pet ownership, acetaminophe n use,
		4										physical activity, TV time
Tanaka 2011	Japan	CS	Schoolchild ren	24399	6~15	10.0	49.2	BMI ≥95th	NR	ISAAC criteria	1854	Age, sex, region,

								percentile		(sneezing/na		siblings,
								(age/sex-		sal		household
								specific)		symptoms +		smoking,
										itchy-watery		physical
										eyes)		activity,
												parental
												history of
												allergies,
												parental
												education
										ISAAC		
										criteria		
				,				Internatio		(current		
Yao 2011	Taiwan	CS	Schoolchild	5351	4~18	10.4	48.9	nal BMI	435	rhinitis =	2365	Age and sex
1 40 2011	(China)	CS	ren	3331	₹~10	10.4	70.7	cut-offs	733	sneezing/ru	2303	Age and sex
								Cut-0118		nny/blocked		
										nose in past		
										12 months)		
Mitchell	Multi-		Schoolchild		6~7	6.5		Internatio		ISAAC		Age, sex,
2013	national	CS	ren	277534	and	and	NR	nal BMI	NR	criteria	NR	region,
2013	(29		ICII		13~1	13.5		cut-offs		(current		language,

	centers,				4					rhinoconjun		income,
	17									ctivitis)		physical
	countries											activity, TV
	)											viewing, BMI
												measurement
												type
								BMI				Age, sex,
								≥95th				race/ethnicity,
Sidell 2013	USA	CS	Schoolchild	10623	6~17	12.2	51	percentile	2776	ICD-9 code	NR	insurance
Sideli 2013	OSA	CS	ren	10023	0.217	12.2	31	(age/sex-	2770	1CD-7 code	INIC	status,
								specific)				geographic
								specific)				region
										ISAAC		
Weinmayr	Multi-	CS	Schoolchild	10652	8~12	9.5	NR	Internatio	1925	questionnair	NR	Age and sex
2014	country		ren					nal BMI		e		
								cut-offs				
					6~7			BMI				
Sybilski			Schoolchild		and	6.5		≥95th		ISAAC		Age, sex, and
2015	Poland	CS	ren	9231	13~1	and	51.3	percentile	439	questionnair	2216	urban/rural
					4	13.5		(age/sex-		e		residence
								specific)				

Lin 2015	Taiwan (China)	CS	High- school children	74688	13~1	14.0	50.7	BMI >23 kg/m²	14452	Physician- diagnosed + symptoms	16720	Age, sex, parental education, environmental tobacco smoke
Lei 2016	China	CS	Cluster- stratified random sampling from community	3327	2~14	8.0	50	BMI  ≥95th  percentile  (age/sex-  specific)	417	Physician- diagnosed (ARIA criteria)	588	Age and sex
Lee 2016	Korea	CS	Nationally representati ve survey (KYRBWS -VII)	75643	13~1	15.5	50	BMI ≥ 25 kg/m2 (Korea standard)	8926	Self- reported physician diagnosis	25643	Age, sex, residence, family affluence scale, parental education, academic achievement, smoking,

												drinking
Han 2016	USA	CS	Nationally representati ve survey	2358	6~17	11.8	49.8	BMI ≥95th percentile (age/sex- specific)	825	Physician- diagnosed + symptoms + ≥1 positive IgE	295	Age, sex, race, income, cotinine, asthma, CRP
McArdle 2024	USA	CS	Children attending otolaryngol ogy clinic	406	2~18	10.7	55.5	BMI ≥95th percentile (age/sex- specific)	130	Physician- diagnosed	NR	Age, sex, and comorbidities
Lee 2025	Korea	CS	Nationally representati ve survey (KNHNES)	1630	13~1 8	15.5	54.5	BMI ≥ 25 kg/m2 (Korea standard)	234	Self- reported physician diagnosis	374	Age and sex

Abbreviations: AR: Allergic rhinitis; ARIA: Allergic rhinitis and its impact on asthma; BMI: Body mass index; CRP: C-reactive protein; CS: Cross-sectional study; ICD-9: International Classification of Diseases, Ninth Revision; IgE: Immunoglobulin E; ISAAC: International Study of Asthma and Allergies in Childhood; KNHNES: Korea National Health and Nutrition Examination Survey; KYRBWS: Korea Youth Risk Behavior Web-Based Survey; NR: Not reported in the original publication; values were not available and were not imputed by the authors.

Table 2. Study quality evaluation via the modified Newcastle-Ottawa Scale

Study	Adequat e definitio n of cases	Representativen ess of cases	Selection of controls	Definitio n of controls	Contro 1 for age and sex	Control for other confounders	Exposure ascertainme nt	Same methods for events ascertainme nt	Non- response rates	Total
Garcia- Marcos 2007	1	1	1	1	1	1	1	1	1	9
Kusunoki 2008	1	1	1	1	1	1	1	1	1	9
Wang 2010	1	1	1	1	1	1	1	1	1	9
Tanaka 2011	1	1	1	1	1	1	1	1	1	9
Yao 2011	1	1	1	1	1	0	1	1	1	8
Mitchell 2013	1	1	1	ì	1	1	1	1	0	8
Sidell 2013	0	1	1	1	1	1	1	1	0	7
Weinmayr 2014	1	1	1	1	1	0	1	1	0	7
Sybilski 2015	1	1	1	1	1	0	1	1	0	7
Lin 2015	1	1	1	1	1	1	1	1	1	9

Lei 2016	1	1	1	1	1	0	1	1	1	8
Lee 2016	0	1	1	1	1	1	1	1	1	8
Han 2016	1	1	1	1	1	1	1	1	1	9
McArdle 2024	1	0	1	1	1	1	1	1	0	7
Lee 2025	0	1	1	1	1	0	1	1	1	7

Table 3. Results of univariate meta-regression analysis

Variables	OR for the association be	OR for the association between obesity and AR									
	Coefficient	Coefficient 95% CI p values Adjusted R <sup>2</sup>									
Mean age (years)	-0.0022	-0.0195 to 0.0152	0.80	0%							
Male (%)	-0.0015	-0.1665 to 0.1636	0.99	0%							
NOS	-0.047	-0.126 to 0.033	0.24	0%							

Abbreviations: OR: odds ratio; CI: confidence interval; AR: allergic rhinitis; NOS: Newcastle-Ottawa Scale.

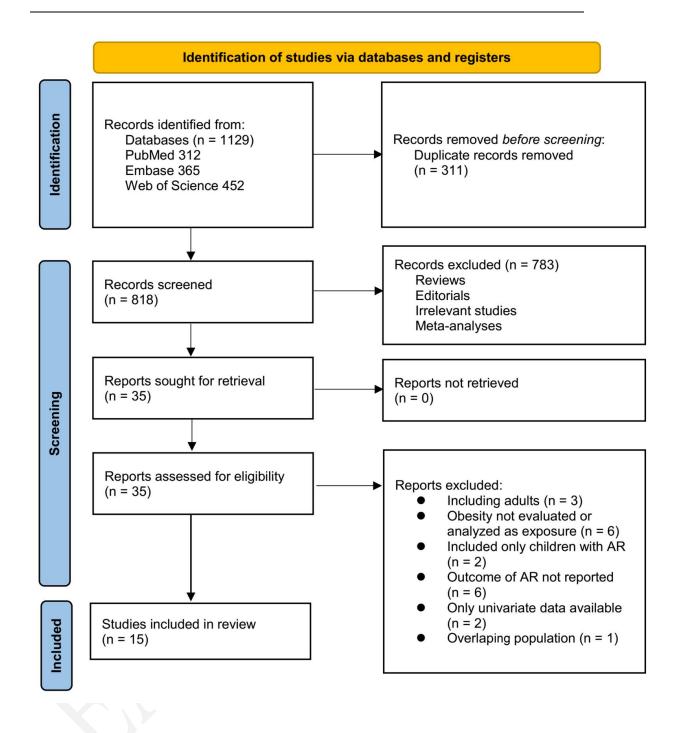


Figure 1. Flowchart of database search and study inclusion

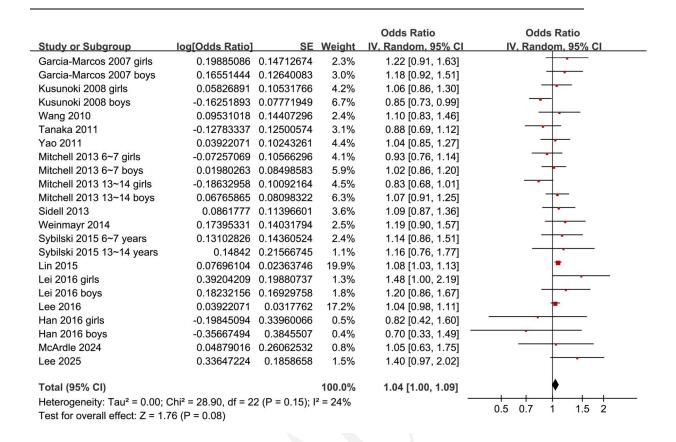


Figure 2. Association between obesity and allergic rhinitis (AR) in children.

Forest plot of 23 datasets assessing the association between obesity and AR in children. Pooled analysis showed no significant association (OR: 1.04, 95% CI: 1.00– 1.09, p = 0.08), with low heterogeneity ( $I^2 = 24\%$ ). Abbreviations: OR: Odds ratio; CI: Confidence interval; SE: Standard error; IV: Inverse variance; df: Degrees of freedom.

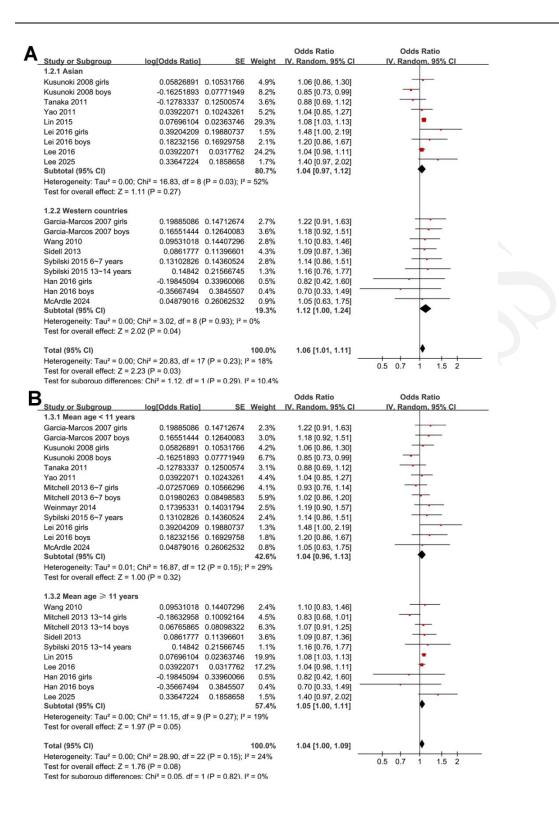


Figure 3. Subgroup analyses of the association between obesity and allergic rhinitis (AR) in children by region and age. (A) Forest plot of studies stratified by geographic region (Asian vs. Western countries). Obesity was associated with AR only in children from Western countries (OR: 1.12, 95% CI: 1.00–1.24, p = 0.04;  $I^2 = 0.09$ ), but not in Asian children (OR: 1.04, 95% CI: 0.97–1.12, p = 0.27;  $I^2 = 52\%$ ). (B)

Forest plot of studies stratified by mean age (< 11 vs. ≥ 11 years), showing no significant subgroup differences (p for subgroup difference = 0.82). Note: the regional subtotal in Figure 3A excludes multinational datasets that could not be uniquely assigned to either Asian or Western categories, and therefore does not equal the overall total reported in Figure 2. Abbreviations: OR: Odds ratio; CI: Confidence interval; SE: Standard error; IV: Inverse variance; df: Degrees of freedom.

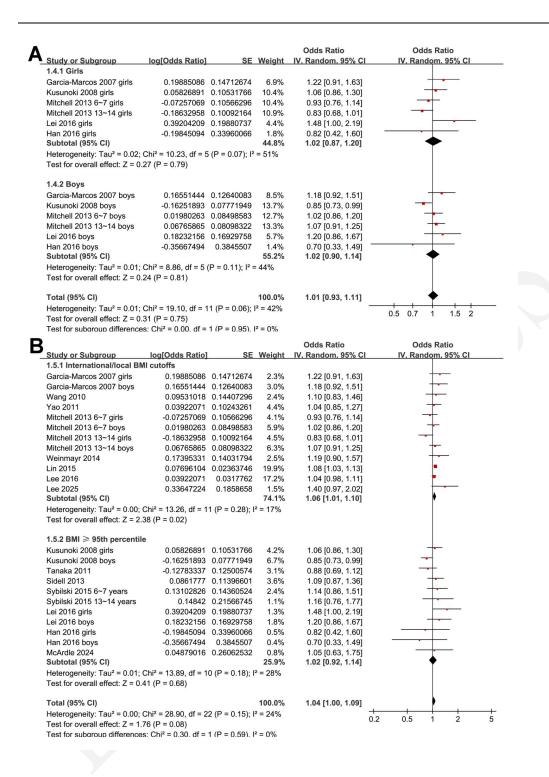
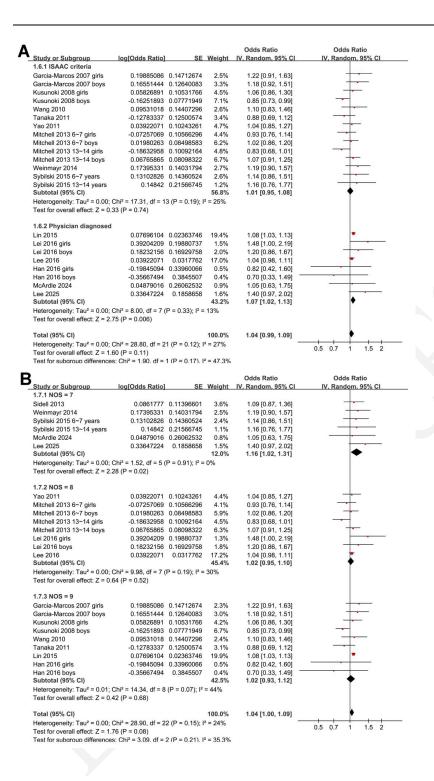


Figure 4. Subgroup analyses of the association between obesity and allergic rhinitis (AR) in children by sex and obesity definition. (A) Forest plot of studies stratified by sex, showing no significant subgroup differences between girls and boys (p = 0.95). (B) Forest plot of studies stratified by obesity definition. A significant association was observed in studies using national/international BMI cutoffs (OR: 1.06, 95% CI: 1.01-1.10, p = 0.02;  $I^2 = 17\%$ ), but not in those using BMI  $\geq 95$ th

percentile (OR: 1.02, 95% CI: 0.92–1.14, p = 0.68;  $I^2 = 28\%$ ). Abbreviations: OR:

Odds ratio; CI: Confidence interval; SE: Standard error; IV: Inverse variance; df:

Degrees of freedom; BMI: Body mass index.



**Figure 5. Subgroup analyses of the association between obesity and allergic rhinitis (AR) in children by diagnostic method and study quality.** (A) Obesity was significantly associated with physician-diagnosed AR (OR: 1.07, 95% CI: 1.02–1.13), but not with AR defined by the ISAAC questionnaire. (B) A significant association was observed in studies with NOS = 7, but not in those with NOS = 8 or 9. Abbreviations: OR: Odds ratio; CI: Confidence interval; SE: Standard error; IV:

Inverse variance; df: Degrees of freedom; BMI: Body mass index; ISAAC: International Study of Asthma and Allergies in Childhood; NOS: Newcastle-Ottawa Scale.

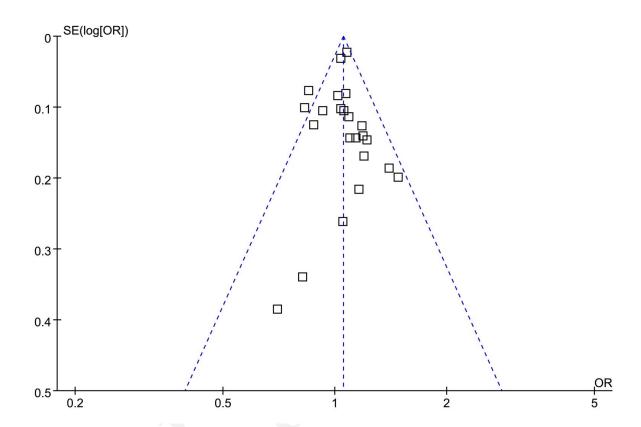


Figure 6. Funnel plot of the association between obesity and allergic rhinitis (AR) in children. The plot appeared symmetrical, and Egger's test showed no evidence of publication bias (p = 0.43).

## SUPPLEMENTAL DATA

Supplemental data are available at the following link:

 $\underline{https://www.bjbms.org/ojs/index.php/bjbms/article/view/12982/4010}$