

Article

Associations Between Residential Indoor Environmental Indicators and Respiratory Allergic Symptoms Among Children in Shenzhen, China

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Abstract

Children are vulnerable to indoor environmental exposures, yet evidence from direct measurements in residential environments remains limited. This study included 115 observations from 74 children in Shenzhen during 2023–2024, with 111 observations available for regression-based analyses. Indoor environmental indicators were evaluated using single-indicator generalized estimating equation (GEE) models, restricted cubic spline (RCS) analyses, and weighted quantile sum (WQS) regression. The primary fully adjusted GEE models included age, sex, body mass index, family history of allergy, indoor temperature, district, and survey year. Asthma-related and allergic rhinitis-related symptoms were reported in 35.7% and 47.8% of the 115 observations, respectively. In the fully adjusted models, illuminance was nominally positively associated with both outcomes, whereas NO₂ was nominally inversely associated with asthma-related symptoms. For allergic rhinitis-related symptoms, NH₃ showed a nominal positive association, whereas O₃ and total volatile organic compounds showed nominal inverse associations. However, none of these associations remained statistically significant after false discovery rate correction. Exploratory RCS analyses suggested potential nonlinear associations for several indicators. Exploratory WQS regression did not identify a significant overall mixture association. Overall, the associations were sensitive to adjustment and multiple-testing correction and should be considered exploratory. Longitudinal studies with repeated exposure measurements are needed to confirm these findings.

Keywords: residential indoor environmental indicators; children; asthma-related symptoms; allergic rhinitis-related symptoms; generalized estimating equations; repeated cross-sectional study



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1. Introduction

Allergic diseases are among the most common chronic diseases in children worldwide and contribute substantially to pediatric morbidity, hospitalization, and healthcare burden [1]. In recent decades, their prevalence has remained high in developed countries and has increased rapidly in many developing countries. These diseases have therefore

become a major public health concern, adversely affecting children's health and quality of life while imposing substantial economic burdens on families and society [2]. Previous studies have indicated that the residential indoor environment may play an important role in the development and exacerbation of allergic diseases in children. Multiple physical, chemical, and biological factors may contribute, making indoor environmental exposure a potentially modifiable determinant of childhood allergic health [2].

Compared with adults, children spend more time indoors and may therefore be more vulnerable to adverse indoor environmental conditions. Previous studies have shown that both short- and long-term exposure to indoor environmental factors may adversely affect lung function development in children [3]. This increased vulnerability may be explained by two main factors. First, children's respiratory and immune systems are still developing, which may increase their susceptibility to environmental stimuli and reduce their ability to respond effectively to such exposures [4]. Second, children have higher ventilation rates per unit body weight and a relatively higher respiratory rate than adults, resulting in a higher inhaled dose relative to body weight under the same environmental conditions [5].

Previous epidemiological studies of indoor environments and childhood allergic diseases have often relied on questionnaires or residential characteristics as proxy measures of indoor environmental conditions. Although these approaches can provide useful information, they have limitations in accurately quantifying actual exposure levels and may reduce the precision of estimated exposure-health associations [6]. In contrast, on-site monitoring provides a more direct characterization of residential exposures. However, previous residential studies based on measured indoor environmental data have largely focused on individual pollutants or a limited range of exposure indicators [7,8]. For example, a systematic review of residential indoor exposure studies identified particulate matter, NO₂, VOCs, and PAHs as the most frequently investigated pollutants [7], while a previous on-site residential study in China evaluated a relatively limited set of physical, chemical, and biological indicators [8]. Consequently, residential indoor environments have rarely been characterized using a broader range of simultaneously measured physical, chemical, and biological indicators. In addition, the health implications of co-occurring indoor environmental exposures remain insufficiently understood, and recent evidence has highlighted the need for further investigation of indoor pollutant mixtures in relation to childhood asthma [9].

Shenzhen is a rapidly urbanizing megacity characterized by high population density, widespread high-rise residential buildings, and substantial traffic- and industry-related emissions [10]. These characteristics may contribute to complex sources of indoor environmental pollutants and distinct exposure patterns. Shenzhen also has a warm and humid subtropical monsoon climate, with long summers and abundant precipitation [11]. Such climatic conditions may influence the accumulation, transformation and dispersion of indoor pollutants [12]. However, evidence on the association between directly measured indoor environmental indicators and childhood allergic diseases in this region remains limited [13].

Indoor environmental quality encompasses multiple dimensions, including physical conditions, chemical pollutants, and biological contaminants, which may jointly influence respiratory health through different pathways. Therefore, using on-site residential monitoring data collected from three districts of Shenzhen between 2023 and 2024, this study systematically evaluated multiple dimensions of indoor environmental quality and their associations with respiratory allergic symptoms in children. Single-indicator generalized estimating equation (GEE) models, restricted cubic spline (RCS) analyses, and weighted quantile sum (WQS) regression were used to address three research questions: (1) which indoor environmental indicators are associated with childhood respiratory allergic symp-

toms; (2) whether the exposure–response relationships show evidence of nonlinearity; and (3) whether co-occurring indoor environmental exposures show an overall mixture association with childhood respiratory allergic symptoms. These analyses were intended to characterize potential associations rather than establish causal relationships.

2. Materials and Methods

2.1. Study Design and Participants

This study was based on a residential indoor environment and child health survey conducted in Luohu District, Bao'an District, and the Shenshan Special Cooperation Zone, China, from 2023 to 2024. A repeated cross-sectional household survey design was adopted, with repeated observations obtained from a subset of households across survey years. Children aged 6–12 years were eligible for participation. Each household contributed one eligible child. To minimize potential information bias related to questionnaire comprehension, when multiple eligible children were present in household, the older child was selected according to the survey protocol. The same children were reassessed when their households participated in both survey years. Symptom questionnaires and indoor environmental measurements included in the present analysis were collected during November–December 2023 and November–December 2024. Indoor environmental measurements were conducted during scheduled daytime household visits, with the exact clock time varying across households.

The sampling framework included three geographic study areas selected according to the survey protocol to represent an upwind urban area, a downwind urban area, and a rural area. Within each area, volunteer households were initially recruited and screened using a standardized questionnaire. Households were subsequently selected according to predefined eligibility criteria, including residence in the current home for at least two years, absence of recent indoor renovation, absence of smoking in the household or continuous incense burning, no household members engaged in predefined high-risk occupations, and provision of written informed consent.

2.2. Exposure Measurement and Health Outcomes

Indoor environmental indicators were assessed through on-site monitoring. The monitoring protocol was developed with reference to the Chinese standard GB/T 18883-2022 Indoor Air Quality Standard [14]. Specific sampling and measurement methods are presented in Table 1. Indoor environmental measurements were conducted in both the children's bedrooms and living rooms, and the arithmetic mean of measurements from the two rooms was used to represent household-level indoor environmental exposure. During monitoring, doors and windows were kept closed, and air-conditioning and mechanical ventilation systems were turned off. The monitored rooms were unoccupied during measurement, and no cooking, cleaning, or other indoor activities were conducted during the monitoring period. Additional information on indoor environmental exposure assessment is provided in Supplementary Table S1. A total of 22 indoor environmental indicators were prespecified for exposure characterization based on GB/T 18883-2022. Indicators meeting the predefined detection-frequency criteria were included in the primary single-indicator GEE analyses.

Table 1. Sampling and measurement methods for indoor environmental indicators.

Indicator	Sampling/Monitoring Method	Sampling Duration	Measurement Method
Temperature, relative humidity (RH), air velocity, illuminance, noise, carbon dioxide (CO ₂), carbon monoxide (CO), particulate matter with an aerodynamic diameter $\leq 10 \mu\text{m}$ (PM ₁₀), and particulate matter with an aerodynamic diameter $\leq 2.5 \mu\text{m}$ (PM _{2.5})	Real-time monitoring instruments	Recorded at 5-min intervals over 45 min	Direct-reading measurement
Ozone (O ₃), nitrogen dioxide (NO ₂), sulfur dioxide (SO ₂), ammonia (NH ₃), and formaldehyde (HCHO)	Gas sampler with absorption solution	45 min	Measured according to the corresponding GB/T or HJ standard methods
Benzene (C ₆ H ₆), toluene (C ₇ H ₈), xylenes (C ₈ H ₁₀), total volatile organic compounds (TVOC), trichloroethylene (C ₂ HCl ₃), and tetrachloroethylene (C ₂ Cl ₄)	Gas sampler with absorption tubes	45 min	Measured according to the corresponding GB/T standard methods
Total bacterial count (TBC) and total fungal count (TFC)	Impactor air microbial sampler	10 min	Culture-based enumeration

GB/T denotes Chinese national standards, and HJ denotes Chinese environmental protection industry standards. Sampling media, absorption solutions, and adsorption materials were selected according to the physicochemical properties of each indicator and the requirements of the corresponding standard methods. Sampling and measurement procedures followed the applicable GB/T or HJ standard methods.

Information on health outcomes and covariates was collected using a structured questionnaire completed by the children's guardians. The questionnaire was developed with reference to the International Study of Asthma and Allergies in Childhood (ISAAC) questionnaire, which has been widely used in epidemiological studies of childhood allergic diseases. The questionnaire assessed symptoms occurring during the preceding 3 months. Asthma-related symptoms and allergic rhinitis-related symptoms were prespecified as the two primary health outcomes. Asthma-related symptoms were defined as the presence of any of the following symptoms: breathing difficulty during sleep, nocturnal cough, loud snoring, or wheezing. Allergic rhinitis-related symptoms were defined as the presence of any of the following symptoms: nasal itching, nasal congestion, runny nose, or sneezing. The questionnaire did not specifically distinguish symptoms caused by respiratory infections from other causes. Covariates considered in the regression analyses included children's age, sex, body mass index (BMI), family history of allergy, indoor temperature, district, and survey year, as appropriate.

2.3. Statistical Analysis

No imputation was performed. Observation records with substantial missing indoor environmental or questionnaire data were excluded before construction of the initial analytical dataset. Regression analyses were subsequently conducted using complete cases for all variables included in each model.

2.3.1. Descriptive Analysis

Descriptive statistics were calculated separately for participant characteristics and indoor environmental indicators. For participant characteristics, continuous variables are summarized as mean $\pm$ standard deviation, and categorical variables as frequencies and percentages. Comparisons between symptom groups were performed using GEE models, with the household identifier specified as the clustering variable to account for repeated

observations across survey years. Gaussian GEE models with an identity link were used for continuous variables, whereas binomial GEE models with a logit link were used for binary variables. Indoor environmental indicators are summarized using the mean $\pm$ standard deviation, minimum, 25th percentile (P25), median (P50), 75th percentile (P75), maximum, and interquartile range (IQR). Spearman's rank correlation coefficients were calculated to assess pairwise correlations among indoor environmental indicators.

2.3.2. Primary Association Analysis

Because some households contributed repeated observations across survey years, observations within the same household could not be considered independent. Therefore, GEE models with a binomial distribution and logit link were applied to account for within-household correlation. The household identifier was specified as the clustering variable because each household contributed only one child participant, and the same household identifier was used to link repeated observations across survey years. An independence working correlation structure was specified because it provided stable convergence across all exposure-outcome models. Robust (sandwich) standard errors were used to reduce sensitivity of statistical inference to misspecification of the working correlation structure [15]. Sensitivity analyses were also conducted using an exchangeable working correlation structure with the same model specifications to evaluate the robustness of the findings.

Single-indicator GEE models were used to examine the associations between eligible indoor environmental indicators and asthma-related symptoms and allergic rhinitis-related symptoms. Indicators were processed according to their detection rates and distributional characteristics [16], as summarized in Table 2. Indicators with detection rates $\leq 30\%$ were excluded from regression analyses because the limited number of detected observations did not support reliable effect estimation. Values below the limit of detection (LOD) were replaced with LOD/2 [17].

Table 2. Processing methods for indoor environmental indicators included in regression analyses.

Indicator	Processing Method
Temperature, RH, and noise	Original values
Illuminance, PM ₁₀ , PM _{2.5} , TBC, and TFC	Natural log-transformed values
Air velocity, O ₃ , CO ₂ , CO, NH ₃ , HCHO, and TVOC	IQR-standardized log-transformed values
NO ₂ , SO ₂ , toluene, and xylenes	Categorical variable (detected vs. non-detected)

IQR, interquartile range. For IQR-standardized log-transformed indicators, natural logarithm-transformed values were scaled by their corresponding IQRs.

Two covariate adjustment sets were evaluated. The primary fully adjusted GEE model included children's age, sex, BMI, family history of allergy, indoor temperature, district, and survey year. District and survey year were included to account for potential spatial and interannual differences in residential and environmental conditions across the study areas and survey periods. When indoor temperature was analyzed as the exposure, it was not simultaneously included as a covariate. A reduced adjustment model, excluding district and survey year, was additionally fitted to evaluate the sensitivity of the exposure–outcome associations to spatial and temporal adjustment.

Results are presented as odds ratios (ORs) with 95% confidence intervals (CIs). To account for multiple comparisons, Benjamini–Hochberg false discovery rate (FDR) correction was applied separately for each primary outcome across the indoor environmental indicators included in the GEE analyses. Both nominal and FDR-adjusted *p* values were reported. All statistical tests were two-sided. Nominal *p* values < 0.05 were used to identify

nominal associations, whereas FDR-adjusted p values < 0.05 were considered statistically significant after correction for multiple comparisons.

2.3.3. Sensitivity Analyses

To assess the robustness of the main findings, sensitivity analyses were conducted to examine the influence of indoor temperature adjustment and alternative exposure categorization. First, for indicators other than indoor temperature, a temperature-unadjusted model was fitted by removing indoor temperature from the primary fully adjusted GEE model to evaluate the sensitivity of the estimated associations to temperature adjustment. Second, indoor environmental indicators with detection rates $> 60\%$ were categorized into low-, medium-, and high-exposure groups based on tertiles. Using the low-exposure group as the reference category, categorical GEE models were fitted using the same covariate structure as the primary fully adjusted GEE model. Additional comparison models including district or survey year separately were also fitted to examine the relative influence of spatial and interannual adjustment.

2.3.4. Nonlinear Analysis

Restricted cubic spline (RCS) models with three knots were used to explore potential nonlinear exposure–response relationships between indoor environmental indicators and asthma-related and allergic rhinitis-related symptoms. Knots were placed at the 10th, 50th, and 90th percentiles of each exposure distribution, with the median value used as the reference. Three knots were selected to limit model complexity while allowing exploratory assessment of nonlinearity [18,19]. A three-knot RCS term contributed two parameters for each exposure. Wald chi-square tests were used to obtain an overall p value for the exposure–response association and a p value for nonlinearity.

Given the modest number of outcome events, a reduced adjustment set was used for the RCS analyses to limit model complexity. The models were adjusted for children's age, sex, BMI, family history of allergy, and indoor temperature. When indoor temperature was analyzed as the exposure, it was not simultaneously included as a covariate. For indicators other than indoor temperature, a temperature-unadjusted sensitivity model was additionally fitted by removing indoor temperature from the covariate set. For most exposures, the RCS model contained seven predictor parameters, including two spline parameters and five covariate parameters, excluding the intercept; models with indoor temperature as the exposure contained six predictor parameters.

2.3.5. Mixture Exposure Analysis

An exploratory WQS feasibility analysis was conducted to assess potential overall mixture associations among selected indoor environmental indicators. Indicators were selected based on detection rates and pairwise Spearman correlation patterns. Physical environmental indicators, indicators with low detection rates, indicators modeled as categorical variables, and one indicator from each highly correlated pair ($|\rho| \geq 0.70$) were excluded from the mixture analysis.

Because no strong a priori directional assumption was available and the individual-indicator analyses showed heterogeneous association directions, WQS component weights were estimated separately under positive and negative directional constraints. All WQS models were adjusted for children's age, sex, BMI, family history of allergy, and indoor temperature.

WQS regression was performed using four quantiles ($q = 4$), with 60% of observations randomly assigned to the training set and 40% to the validation set. A total of 1000 bootstrap samples were used to estimate the WQS component weights, and the random seed was set

to 123 for reproducibility. The WQS implementation used in this study did not account for within-household clustering of repeated observations.

2.3.6. Simulation-Based Power Analysis

A simulation-based power analysis was conducted using the observed analytical structure of 111 complete observations. The simulated datasets retained the observed covariate values for age, sex, BMI, family history of allergy, indoor temperature, district, and survey year, and were analyzed using the same fully adjusted GEE structure as the primary analysis. A standardized continuous exposure was simulated from a normal distribution, such that the specified OR represented the association per 1-SD increase in exposure. Outcome prevalences were set to the observed values of 36.9% for asthma-related symptoms and 48.6% for allergic rhinitis-related symptoms. An intraclass correlation coefficient (ICC) of 0.10 was used as the primary assumption for within-household correlation, with ICC values of 0, 0.05, 0.10, and 0.20 examined in sensitivity analyses. Power was estimated using two-sided $\alpha = 0.05$. A total of 5000 simulations per effect size were used around the 80% power threshold, and 2000 simulations per scenario were used for the ICC sensitivity analyses.

2.3.7. Statistical Software

All statistical analyses were performed using R software (version 4.5.0; R Foundation for Statistical Computing, Vienna, Austria). The geepack (version 1.3.13), rms (version 8.1.1), and gWQS (version 3.0.5) packages were used for GEE, RCS, and WQS analyses, respectively. Figures were generated using the ggplot2 (version 3.5.2) package.

3. Results

3.1. Characteristics of Study Participants

The participant selection process and construction of the final analytical dataset are presented in Figure 1. In 2024, 45 children returned from the 2023 survey and 20 were newly recruited. Before statistical analysis, 10 observation records were excluded because of missing indoor environmental measurements ($n = 8$) or questionnaire data ($n = 2$). Of these, four records were from returning children and six were from children who contributed only one survey-year record.

A total of 74 children from 74 households were included in the final analytical dataset. Of these, 41 children participated in both the 2023 and 2024 surveys, contributing 82 observation records, whereas 33 children participated in only one survey year (19 in 2023 only and 14 in 2024 only), contributing 33 observation records. The final analytical dataset therefore comprised 115 observation records. Four observations with missing BMI or family history of allergy were excluded from regression analyses, resulting in 111 complete observations. All GEE, RCS, and WQS analyses were based on these 111 complete observations. The WQS sample was subsequently divided into training and validation subsets.

Among the 115 observations, asthma-related symptoms were reported in 41 (35.7%) and allergic rhinitis-related symptoms in 55 (47.8%). The mean age was 9.3 years, and males accounted for a slightly higher proportion than females. No statistically significant differences in age, sex, family history of allergy, or BMI were observed between groups defined by asthma-related symptoms or allergic rhinitis-related symptoms. Details are presented in Table 3.

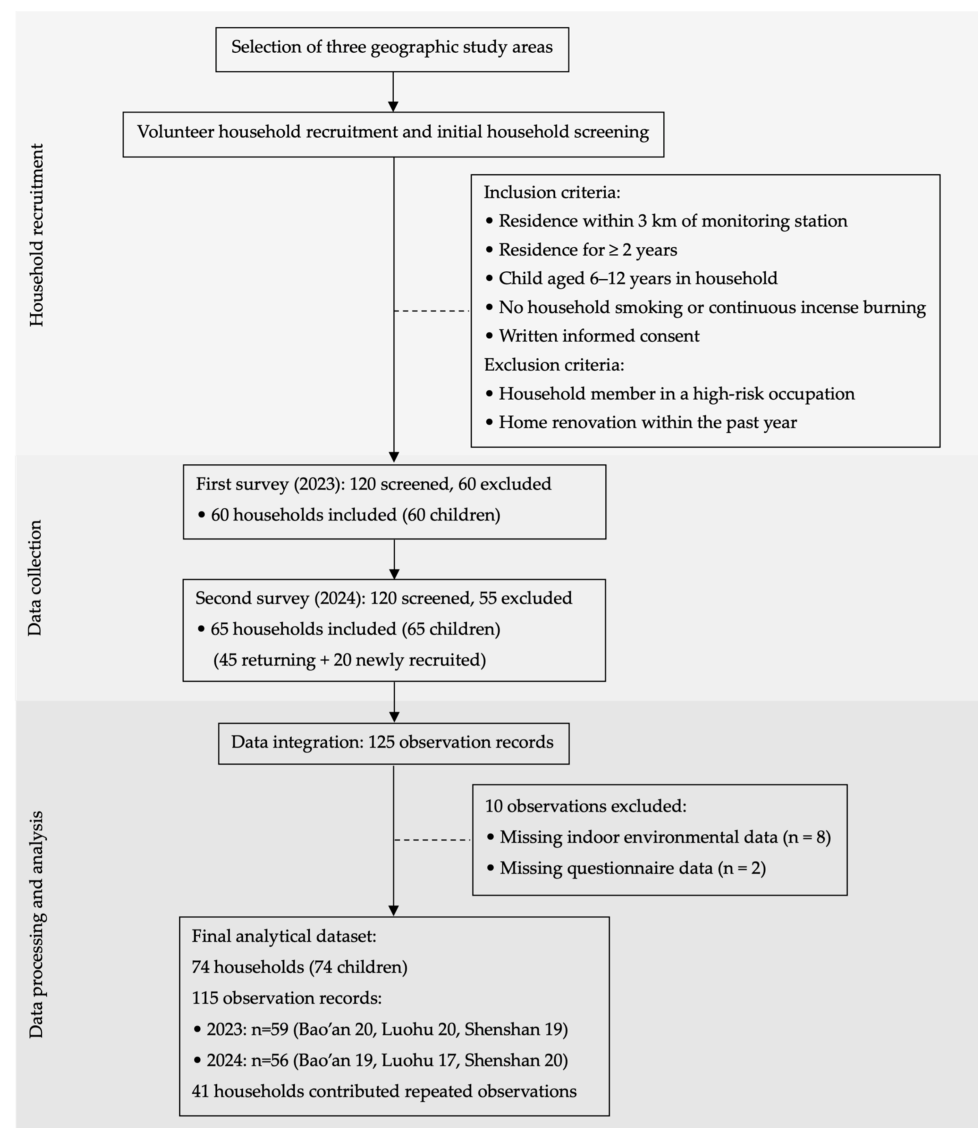


Figure 1. Flow diagram of household recruitment, data integration, and selection of the final analytical dataset.

Table 3. Characteristics of study participants.

Variable	Asthma-Related Symptoms	Without Asthma-Related Symptoms	<i>p</i> Value	Allergic Rhinitis-Related Symptoms	Without Allergic Rhinitis-Related Symptoms	<i>p</i> Value
Age, years, mean $\pm$ SD	9.29 $\pm$ 2.05	9.27 $\pm$ 2.05	0.956	9.38 $\pm$ 1.86	9.18 $\pm$ 2.21	0.615
Sex			0.492			0.654
Male, n (%)	25 (61.0)	39 (52.7)		32 (58.2)	32 (53.3)	
Female, n (%)	16 (39.0)	35 (47.3)		23 (41.8)	28 (46.7)	
Family history of allergy			0.836			0.523
Yes, n (%)	10 (24.4)	16 (22.5)		11 (20.4)	15 (25.9)	

Table 3. Cont.

Variable	Asthma-Related Symptoms	Without Asthma-Related Symptoms	<i>p</i> Value	Allergic Rhinitis-Related Symptoms	Without Allergic Rhinitis-Related Symptoms	<i>p</i> Value
No, n (%)	31 (75.6)	55 (77.5)		43 (79.6)	43 (74.1)	
BMI, mean $\pm$ SD	18.80 $\pm$ 3.59	18.50 $\pm$ 5.91	0.726	18.43 $\pm$ 3.91	18.77 $\pm$ 6.16	0.705

This study used a repeated cross-sectional design and included 115 observation records, with some households participating in both survey years. Asthma and allergic rhinitis refer to asthma-related symptoms and allergic rhinitis-related symptoms, respectively. The two outcomes were analyzed separately in the between-group comparisons. Percentages were calculated based on the available observation records, with missing values excluded where applicable. Missing data: BMI, $n = 1$; family history of allergy, $n = 3$; all other variables had no missing values.

3.2. Distribution and Correlation of Indoor Environmental Indicators

Indoor environmental monitoring indicated relatively limited variability in physical environmental indicators across households, whereas chemical and biological indicators exhibited greater variability. Among the 22 monitored indoor environmental indicators, benzene, trichloroethylene, and tetrachloroethylene had detection rates $\leq 30\%$ and were therefore excluded from regression analyses because the limited number of detected observations did not support reliable effect estimation.

For descriptive comparison with GB/T 18883-2022 Indoor Air Quality Standard, SO₂, CO₂, NH₃, formaldehyde (HCHO), benzene, toluene, and total bacterial count (TBC) were descriptively compared with the corresponding reference limits because their sampling durations were generally consistent with the assessment periods specified in the standard. Several observations exceeded the corresponding reference limits for these indicators. In contrast, PM_{2.5}, PM₁₀, total volatile organic compounds (TVOC), trichloroethylene, and tetrachloroethylene were not compared with the standard limits because their sampling durations did not match the specified assessment periods; their measured concentrations were retained for descriptive characterization. Although GB/T 18883-2022 also specifies requirements for some physical indicators, these were not included in the standard-limit comparison because the monitoring conditions could not be confirmed to fully meet the prescribed assessment conditions. Detailed descriptive statistics are presented in Table 4.

Spearman's rank correlation analysis identified several statistically significant pairwise correlations among the indoor environmental indicators ($p < 0.05$). Among the physical indicators, temperature was strongly positively correlated with relative humidity (RH) ($\rho = 0.78$). Among the chemical indicators, PM₁₀ was strongly positively correlated with PM_{2.5} ($\rho = 0.79$), while TVOC was positively correlated with toluene ($\rho = 0.76$) and O₃ ($\rho = 0.69$). In addition, NO₂, CO₂, HCHO, and total fungal count (TFC) were significantly correlated with several other indoor environmental indicators, although the directions of these correlations varied. Detailed correlation results are presented in Figure 2.

Table 4. Descriptive statistics of indoor environmental indicators.

Category	Indicator (Unit)	Mean $\pm$ SD	Min	P25	P50	P75	Max	IQR
Physical	Temperature ($^{\circ}$ C)	22 $\pm$ 3.7	13.5	19.4	22.3	24.8	33.6	5.4
	RH (%)	58.3 $\pm$ 12.5	28.1	51.6	58.5	67.2	89.6	15.6
	Air velocity (m/s)	0.04 $\pm$ 0.06	0	0.02	0.03	0.05	0.56	0.03
	Illuminance (lx)	380 $\pm$ 292	86	195	292	450	1404	255
	Noise (dB(A))	50.8 $\pm$ 5.6	39.3	46.5	50.7	55.1	63.3	8.6
Chemical	O ₃ (mg/m ³)	0.040 $\pm$ 0.043	0.003	0.008	0.021	0.056	0.148	0.048
	NO ₂ (mg/m ³)	0.033 $\pm$ 0.025	0.005	0.010	0.027	0.053	0.162	0.043
	SO ₂ (mg/m ³)	0.023 $\pm$ 0.051	0.005	0.008	0.012	0.027	0.538	0.019
	CO ₂ (%) ¹	0.056 $\pm$ 0.033	0.039	0.046	0.050	0.055	0.358	0.009
	CO (mg/m ³)	0.621 $\pm$ 0.963	0.050	0.222	0.235	0.638	5.402	0.415
	NH ₃ (mg/m ³)	0.095 $\pm$ 0.114	0.004	0.024	0.049	0.116	0.760	0.092
	HCHO (mg/m ³)	0.029 $\pm$ 0.044	0.005	0.005	0.012	0.037	0.329	0.032
	TVOC (mg/m ³)	0.551 $\pm$ 0.752	0.014	0.090	0.276	0.634	3.175	0.544
	benzene (mg/m ³)	0.0019 $\pm$ 0.0038	0.0002	0.0002	0.0006	0.0019	0.0327	0.0018
	toluene (mg/m ³)	0.0901 $\pm$ 0.2020	0.0003	0.0014	0.0057	0.0163	0.9045	0.0149
	xylenes (mg/m ³)	0.0078 $\pm$ 0.0102	0.0001	0.0028	0.0045	0.0085	0.0765	0.0057
	trichloroethylene (mg/m ³)	0.0014 $\pm$ 0.0019	0.0006	0.0008	0.0008	0.0010	0.0153	0.0002
	tetrachloroethylene (mg/m ³)	0.0112 $\pm$ 0.0282	0.0010	0.0013	0.0013	0.0025	0.1877	0.0012
	PM ₁₀ (μ g/m ³)	52.6 $\pm$ 28.8	4.9	40.4	51.0	61.9	148.0	21.5
	PM _{2.5} (μ g/m ³)	34.1 $\pm$ 22.8	2.6	22.8	27.5	43.5	126.0	20.7
Biological	TBC (CFU/m ³)	200 $\pm$ 237	24	77	104	234	2092	157
	TFC (CFU/m ³)	296 $\pm$ 496	13	73	122	321	4141	248

¹ Expressed as volume fraction (v/v). Values below the limit of detection (LOD) were replaced with LOD/2. P25, 25th percentile; P50, median; P75, 75th percentile; IQR, interquartile range.

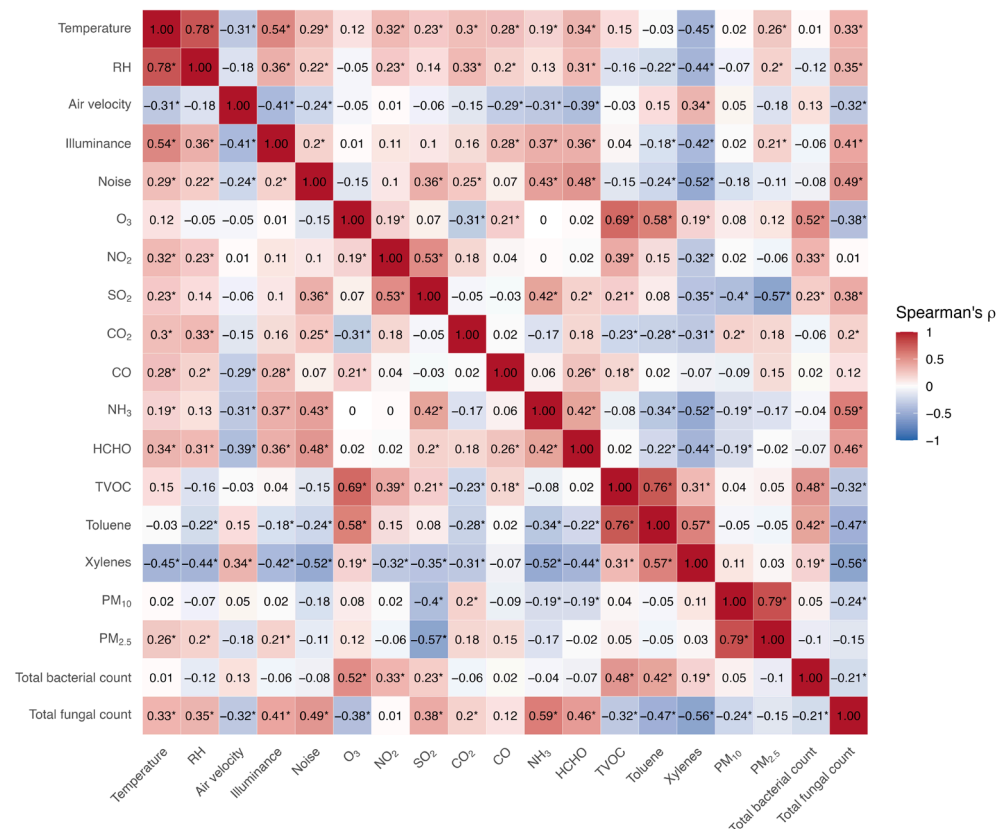


Figure 2. Spearman's rank correlation heatmap of indoor environmental indicators. * $p < 0.05$. Values below the limit of detection (LOD) were replaced with LOD/2.

3.3. Associations Between Indoor Environmental Indicators and Respiratory Allergic Symptoms

GEE models were used to evaluate associations between eligible indoor environmental indicators and asthma-related symptoms and allergic rhinitis-related symptoms. Results from the fully adjusted and reduced adjustment models are presented in Table 5. Benjamini–Hochberg false discovery rate (FDR) correction was applied separately for each primary outcome across the eligible indicators, with nominal and FDR-adjusted *p* values provided in Supplementary Table S2.

Table 5. Associations between eligible indoor environmental indicators and respiratory allergic symptoms in the primary fully adjusted and reduced adjustment GEE models. Panel (A). Asthma-related symptoms. Panel (B). Allergic rhinitis-related symptoms.

(A)					
Indicator	Exposure Unit	Primary Fully Adjusted Model		Reduced Adjustment Model	
		OR (95% CI)	<i>p</i> Value	OR (95% CI)	<i>p</i> Value
Temperature	per 1 °C increase	1.08 (0.95–1.23)	0.243	1.14 (1.02–1.28)	0.020
RH	per 1% increase	0.96 (0.92–1.00)	0.067	0.97 (0.93–1.01)	0.150
Air velocity	per IQR increase in ln(X)	1.03 (0.82–1.28)	0.813	1.05 (0.84–1.32)	0.667
Illuminance	per 1-unit increase in ln(X)	2.42 (1.17–4.99)	0.017	2.46 (1.24–4.88)	0.010
Noise	per 1 dB(A) increase	1.03 (0.95–1.11)	0.539	1.04 (0.96–1.12)	0.387
O ₃	per IQR increase in ln(X)	0.55 (0.29–1.05)	0.072	0.65 (0.40–1.05)	0.079
NO ₂	detected vs. not-detected	0.33 (0.12–0.93)	0.036	0.42 (0.15–1.14)	0.088
SO ₂	detected vs. not-detected	0.71 (0.12–4.31)	0.712	0.76 (0.28–2.09)	0.601
CO ₂	per IQR increase in ln(X)	0.98 (0.88–1.08)	0.681	1.00 (0.89–1.12)	0.957
CO	per IQR increase in ln(X)	1.17 (0.78–1.73)	0.448	1.25 (0.85–1.84)	0.262
NH ₃	per IQR increase in ln(X)	1.30 (0.74–2.28)	0.357	1.27 (0.87–1.88)	0.219
HCHO	per IQR increase in ln(X)	0.81 (0.60–1.09)	0.162	0.86 (0.66–1.13)	0.283
TVOC	per IQR increase in ln(X)	0.82 (0.51–1.34)	0.436	0.81 (0.53–1.24)	0.334
Toluene	detected vs. not-detected	0.53 (0.24–1.15)	0.109	0.49 (0.24–0.99)	0.045
Xylenes	detected vs. not-detected	0.62 (0.31–1.25)	0.181	0.55 (0.29–1.05)	0.070
PM ₁₀	per 1-unit increase in ln(X)	1.34 (0.74–2.43)	0.334	1.22 (0.64–2.30)	0.545
PM _{2.5}	per 1-unit increase in ln(X)	1.37 (0.74–2.55)	0.321	1.26 (0.64–2.50)	0.504
TBC	per 1-unit increase in ln(X)	0.74 (0.43–1.29)	0.296	0.71 (0.43–1.17)	0.179
TFC	per 1-unit increase in ln(X)	1.36 (0.81–2.26)	0.244	1.27 (0.88–1.82)	0.205
(B)					
Indicator	Exposure Unit	Primary Fully Adjusted Model		Reduced Adjustment Model	
		OR (95% CI)	<i>p</i> Value	OR (95% CI)	<i>p</i> Value
Temperature	per 1 °C increase	1.03 (0.90–1.17)	0.700	1.12 (1.00–1.25)	0.050
RH	per 1% increase	1.00 (0.96–1.04)	0.913	1.02 (0.98–1.06)	0.336
Air velocity	per IQR increase in ln(X)	0.85 (0.65–1.10)	0.216	0.90 (0.61–1.35)	0.616
Illuminance	per 1-unit increase in ln(X)	2.00 (1.04–3.84)	0.037	2.13 (1.12–4.05)	0.021
Noise	per 1 dB(A) increase	0.97 (0.90–1.05)	0.434	1.00 (0.93–1.08)	0.981
O ₃	per IQR increase in ln(X)	0.49 (0.25–0.95)	0.035	0.49 (0.30–0.79)	0.004
NO ₂	detected vs. not-detected	0.37 (0.12–1.10)	0.074	0.38 (0.13–1.09)	0.072
SO ₂	detected vs. not-detected	0.49 (0.09–2.74)	0.418	0.42 (0.16–1.08)	0.073
CO ₂	per IQR increase in ln(X)	0.95 (0.87–1.04)	0.299	1.00 (0.90–1.11)	0.992
CO	per IQR increase in ln(X)	0.96 (0.66–1.39)	0.819	1.02 (0.71–1.46)	0.920
NH ₃	per IQR increase in ln(X)	1.77 (1.10–2.83)	0.018	1.36 (0.99–1.85)	0.054
HCHO	per IQR increase in ln(X)	0.75 (0.54–1.04)	0.088	0.80 (0.60–1.06)	0.117
TVOC	per IQR increase in ln(X)	0.60 (0.37–0.99)	0.044	0.56 (0.38–0.83)	0.004

Table 5. Cont.

(B)					
Indicator	Exposure Unit	Primary Fully Adjusted Model		Reduced Adjustment Model	
		OR (95% CI)	<i>p</i> Value	OR (95% CI)	<i>p</i> Value
Toluene	detected vs. not-detected	0.46 (0.19–1.08)	0.073	0.36 (0.16–0.82)	0.015
Xylenes	detected vs. not-detected	1.06 (0.51–2.23)	0.871	0.71 (0.35–1.46)	0.351
PM ₁₀	per 1-unit increase in ln(X)	0.79 (0.42–1.51)	0.478	1.11 (0.64–1.94)	0.714
PM _{2.5}	per 1-unit increase in ln(X)	0.87 (0.44–1.75)	0.704	1.32 (0.72–2.39)	0.367
TBC	per 1-unit increase in ln(X)	0.58 (0.33–1.00)	0.051	0.48 (0.30–0.78)	0.003
TFC	per 1-unit increase in ln(X)	1.37 (0.81–2.32)	0.245	1.32 (0.93–1.87)	0.118

ln(X) denotes the natural logarithm of the corresponding indicator, and IQR denotes the interquartile range. For indicators modeled per 1-unit increase in ln(X), a 1-unit increase corresponds to an approximately 2.72-fold increase in the original exposure level. For indicators modeled per IQR increase in ln(X), ORs represent the change in odds associated with an increase of one interquartile range on the log-transformed scale. All statistical tests were two-sided. The primary fully adjusted model was adjusted for children's age, sex, BMI, family history of allergy, indoor temperature, district, and survey year, except when indoor temperature was analyzed as the exposure. The reduced adjustment model used the same covariate structure as the primary fully adjusted model but excluded district and survey year. For categorical indicators, the non-detected group served as the reference category.

For asthma-related symptoms, the fully adjusted model showed a nominal positive association with illuminance (OR = 2.42, 95% CI: 1.17–4.99) and a nominal inverse association with NO₂ (OR = 0.33, 95% CI: 0.12–0.93). Neither association remained statistically significant after FDR correction. In the reduced adjustment model, indoor temperature and illuminance showed nominal positive associations, whereas toluene showed a nominal inverse association.

For allergic rhinitis-related symptoms, the fully adjusted model showed nominal positive associations with illuminance (OR = 2.00, 95% CI: 1.04–3.84) and NH₃ (OR = 1.77, 95% CI: 1.10–2.83), and nominal inverse associations with O₃ (OR = 0.49, 95% CI: 0.25–0.95) and TVOC (OR = 0.60, 95% CI: 0.37–0.99). None of these associations remained statistically significant after FDR correction. In the reduced adjustment model, illuminance showed a nominal positive association, whereas O₃, TVOC, toluene, and TBC showed nominal inverse associations; the association with indoor temperature was borderline.

Overall, several nominal associations were observed in both adjustment models, but no association remained statistically significant after FDR correction in the fully adjusted model.

3.4. Sensitivity Analysis Results

Sensitivity analyses were conducted using GEE models to evaluate the robustness of the associations observed in the primary fully adjusted analyses. In the temperature-unadjusted model, the directions and magnitudes of most associations were generally similar to those observed in the primary fully adjusted model. The nominal associations of illuminance and NO₂ with asthma-related symptoms, and those of illuminance, O₃, NH₃, and TVOC with allergic rhinitis-related symptoms, were generally consistent in direction after removal of indoor temperature from the covariate set.

Additional comparison models adjusting separately for district and survey year were examined. Effect estimates from the year-adjusted models were generally similar to those from the reduced adjustment model, whereas adjustment for district produced larger changes in several estimates.

In the categorical exposure analysis, the high-exposure O₃ group showed nominal inverse associations with both asthma-related symptoms (OR = 0.16, 95% CI: 0.03–0.86) and allergic rhinitis-related symptoms (OR = 0.12, 95% CI: 0.02–0.65), compared with the low-exposure group. The medium-exposure HCHO group showed a nominal positive

association with asthma-related symptoms (OR = 5.10, 95% CI: 1.52–17.10). For allergic rhinitis-related symptoms, the medium-exposure CO group showed a nominal positive association (OR = 20.19, 95% CI: 3.22–126.59), although the wide confidence interval indicated substantial imprecision. No other categorical exposure associations reached nominal statistical significance. Detailed results of the sensitivity analyses are presented in Supplementary Tables S3–S5.

3.5. RCS Analysis Results

RCS analyses were conducted as an exploratory assessment of potential nonlinear exposure–response relationships. The analyses included 41 asthma-related symptom events and 54 allergic rhinitis-related symptom events. Several indoor environmental indicators showed evidence of nonlinearity, with generally similar patterns observed in the reduced adjustment and temperature-unadjusted models.

For asthma-related symptoms, the most consistent nonlinear pattern was observed for air velocity, while potential nonlinear patterns were also suggested for PM₁₀ and PM_{2.5}. For allergic rhinitis-related symptoms, potential nonlinear patterns were observed for RH, air velocity, HCHO, and TFC. Overall and nonlinear *p* values for all RCS models are reported in Supplementary Table S6. Representative exposure–response curves with 95% confidence bands are shown in Supplementary Figure S1, with rug marks along the x-axis indicating the distribution of observed exposure values.

3.6. Exploratory WQS Analysis Results

Before the WQS analysis, physical environmental indicators (temperature, relative humidity, air velocity, illuminance, and noise) were excluded. Indicators with low detection rates (benzene, trichloroethylene, and tetrachloroethylene), indicators modeled as categorical variables (NO₂, SO₂, toluene, and xylenes) were also excluded. Among highly correlated indicators, PM₁₀ was excluded because of its strong correlation with PM_{2.5} ($|\rho| \geq 0.70$). Therefore, nine indoor environmental indicators (O₃, CO, CO₂, NH₃, PM_{2.5}, HCHO, TVOC, TBC, and TFC) were included in the WQS analysis.

For asthma-related symptoms, the overall mixture ORs were 1.29 (95% CI: 0.44–3.77) and 2.36 (95% CI: 0.63–8.82) under positive- and negative-direction weight estimation, respectively. For allergic rhinitis-related symptoms, the corresponding ORs were 2.21 (95% CI: 0.79–6.21) and 0.82 (95% CI: 0.24–2.86), respectively. None of the overall mixture associations was statistically significant. Component weights and detailed WQS results are provided in Supplementary Figure S2 and Table S7.

3.7. Simulation-Based Power Analysis

Under the primary assumption of an intraclass correlation coefficient (ICC) of 0.10, the simulation-based power analysis indicated approximately 80% power to detect an OR of 1.90 per 1-SD increase in exposure for asthma-related symptoms and an OR of 1.85 per 1-SD increase for allergic rhinitis-related symptoms. Across ICC values ranging from 0 to 0.20, the minimum tested ORs achieving at least 80% power ranged from 1.8 to 2.1.

4. Discussion

Based on field measurements of residential indoor environments in Shenzhen, this study evaluated associations between multiple indoor environmental indicators and respiratory allergic symptoms in children. In the fully adjusted GEE analyses, several nominal associations were observed, including positive associations of illuminance with both outcomes, an inverse association of NO₂ with asthma-related symptoms, and positive or inverse associations of NH₃, O₃, and TVOC with allergic rhinitis-related symptoms. However, none remained statistically significant after FDR correction. Several estimates were

attenuated after adjustment for district and survey year, suggesting the observed associations were sensitive to spatial and interannual adjustment. Sensitivity analyses also showed that some estimates changed after removal of indoor temperature from the covariate set, while categorical exposure analyses produced several nominal but imprecise associations. Exploratory RCS analyses suggested potential nonlinear exposure–response patterns for several indicators, whereas exploratory WQS regression did not identify a statistically significant overall mixture association. Taken together, these findings were sensitive to model specification and multiple-testing correction and should therefore be interpreted as exploratory rather than confirmatory.

Evidence regarding the association between indoor illuminance and respiratory allergic symptoms in children remains limited, and the underlying mechanisms are not well understood. In the present study, illuminance showed nominal positive associations with both asthma-related and allergic rhinitis-related symptoms in the fully adjusted model, but neither association remained statistically significant after FDR correction. Indoor illuminance may reflect several residential characteristics, including daylight availability, building design, window characteristics, and occupant behavior, and may therefore serve as a proxy for broader features of the indoor residential environment [20]. Although indoor illuminance is not directly comparable to personal light exposure, previous studies have suggested that light exposure may influence physiological processes through circadian-related pathways, including the regulation of sleep and immune function [21]. However, because detailed information on housing structure and light sources was not available, these potential explanations remain speculative and should be considered hypothesis-generating.

In addition to illuminance, indoor temperature and RH jointly characterize the indoor hygrothermal environment and may influence respiratory health through multiple pathways, including changes in ventilation behavior, microbial growth, and the emission and accumulation of volatile pollutants [22,23]. Previous experimental studies have shown that variations in temperature and humidity may influence allergic responses, symptom expression, and levels of inflammatory mediators [24]. In the present study, nominal positive associations of indoor temperature with both outcomes were observed in the reduced adjustment model but were attenuated after additional adjustment for district and survey year. This attenuation suggests that spatial, interannual, or correlated residential characteristics may partly account for the associations observed in the reduced adjustment model. The association between RH and allergic rhinitis-related symptoms also varied according to whether indoor temperature was included as a covariate, indicating that the independent contributions of individual hygrothermal indicators may be difficult to disentangle because of their interrelated nature. Overall, the observed hygrothermal associations were sensitive to model specification and should therefore be interpreted cautiously.

Previous systematic reviews and epidemiological studies have generally reported associations between residential dampness and mold exposure and asthma or allergic rhinitis in children [25–28]. Mold spores and their metabolites may act as inhaled allergens or inflammatory stimuli, potentially contributing to allergic responses and airway mucosal inflammation [29–31]. Because children’s immune and respiratory systems are still developing, they may be particularly susceptible to indoor biological allergens and inflammatory stimuli [5,32]. Temperature and humidity may also jointly influence mold growth, proliferation, and spore release. In the present study, TFC was not robustly associated with either outcome in the fully adjusted GEE analyses, although exploratory RCS analyses suggested a potential nonlinear pattern with allergic rhinitis-related symptoms. This nonlinear finding should be interpreted cautiously because it was derived from an exploratory spline analysis in a modest sample, and measured fungal concentrations may

reflect broader indoor hygrothermal conditions and correlated residential characteristics rather than an isolated biological exposure.

TBC was not statistically significantly associated with allergic rhinitis-related symptoms in the fully adjusted model, although a nominal inverse association was observed in the reduced adjustment model. Previous studies have suggested that exposure to diverse microbial environments may influence immune development [33–35]. However, TBC does not directly characterize microbial diversity or the allergenic or pathogenic potential of the bacterial community. Indoor airborne bacterial communities are shaped by multiple factors, including ventilation conditions, occupant activities, and outdoor air sources [36]. In the present study, only the total count of culturable bacteria was measured, without characterization of bacterial community composition or diversity. Therefore, the observed inverse pattern should not be interpreted as evidence of a protective effect.

The association between HCHO and respiratory allergic symptoms was not consistent across exposure parameterizations and model specifications. In the fully adjusted continuous-exposure GEE models, HCHO was not significantly associated with either outcome, whereas the categorical sensitivity analysis showed a nominal positive association with asthma-related symptoms. Previous systematic reviews have reported that indoor HCHO exposure may be associated with increased risks of asthma and related respiratory symptoms [37,38]. Indoor humidity may also enhance HCHO emissions from building and renovation materials, furniture, and engineered wood products, thereby increasing indoor exposure levels [23,39,40]. Given the inconsistency across specifications and the imprecision of the categorical estimate, the present findings do not provide robust evidence of an association between HCHO and respiratory allergic symptoms.

Similar caution is warranted when interpreting the chemical indicators. In the fully adjusted model, O₃ and TVOC showed nominal inverse associations with allergic rhinitis-related symptoms, but neither remained statistically significant after FDR correction. These findings should therefore not be interpreted as evidence of protective effects. Spearman's rank correlation analysis showed that O₃ concentrations were negatively correlated with CO₂ concentrations. Because indoor CO₂ is commonly used as an indicator of ventilation conditions, this correlation may partly reflect differences in ventilation and indoor–outdoor air exchange among households rather than a beneficial effect of O₃ exposure [41–45]. In addition, health responses to O₃ may occur with temporal lags that cannot be adequately captured by a single cross-sectional indoor measurement [46]. Consequently, the measured O₃ concentrations may not correspond to the exposure window most relevant to symptom occurrence.

TVOC showed a nominal inverse association with allergic rhinitis-related symptoms in the fully adjusted model, but this association did not remain statistically significant after FDR correction. TVOC represents the aggregate concentration of multiple volatile organic compounds that may differ substantially in their sources, toxicological properties, and potential health effects [47,48]. Indoor TVOC may originate from building materials, furnishings, cleaning products, and biogenic sources, and the total concentration may therefore not accurately reflect exposure to specific harmful compounds [48–51]. Because the chemical composition of TVOC was not characterized in the present study, the observed pattern may have reflected differences in component profiles, emission sources, ventilation conditions, or indoor–outdoor air exchange. Accordingly, this finding should not be interpreted as evidence of a protective effect.

The sensitivity analyses further supported a cautious interpretation of the main findings. Several estimates changed across alternative covariate specifications, particularly after adjustment for district, whereas adjustment for survey year generally produced smaller changes. Associations involving the indoor hygrothermal environment were also sensi-

tive to adjustment for indoor temperature, suggesting that the contributions of correlated environmental conditions may be difficult to disentangle. Categorical exposure analyses yielded several nominal associations, but some estimates were highly imprecise, further supporting an exploratory interpretation of these findings.

The associations between indoor environmental indicators and respiratory allergic symptoms may not necessarily follow simple linear dose–response patterns [52,53]. Exploratory RCS analyses suggested potential nonlinear patterns for several indicators, including air velocity, RH, HCHO, and TFC. However, these findings should be interpreted cautiously because the number of outcome events was limited relative to the number of spline and covariate parameters, raising the possibility of overfitting. In addition, confidence intervals widened at the extremes of several exposure distributions, where observations were relatively sparse. Therefore, the RCS findings should be regarded as exploratory and should not be interpreted as evidence of specific exposure thresholds or causal relationships.

Indoor environmental factors typically occur as co-exposures, and single-indicator analyses may not fully capture their joint relationships with health outcomes [54–56]. Evidence on the combined associations of physical, chemical, and biological indoor environmental indicators with respiratory health in children remains limited [57,58]. In the present study, the exploratory WQS analysis did not identify a statistically significant overall mixture association. Because the WQS analysis did not account for within-household clustering, the findings should be regarded as preliminary rather than confirmatory. In addition, component weights from the non-significant mixture models should not be interpreted as evidence of dominant or relatively important contributors. The modest analytical sample size and limited variability of some exposure components may also have reduced the ability to detect overall mixture associations.

A major strength of this study is the use of field measurements of multiple indoor environmental indicators rather than exclusive reliance on questionnaire-based proxy measures, providing a more objective characterization of household-level indoor environmental conditions. Studies simultaneously assessing physical, chemical, and biological indoor environmental indicators remain relatively limited, particularly in subtropical regions characterized by high temperature and humidity, such as Shenzhen. This study therefore provides additional descriptive and exploratory evidence on the relationships between residential indoor environmental conditions and respiratory allergic symptoms in children. In addition, GEE models were used to account for repeated observations within households, while exploratory RCS and WQS analyses were used to examine potential nonlinear and mixture patterns. These complementary approaches provided a broader characterization of indoor environmental exposures and their associations with respiratory allergic symptoms.

This study also has several limitations. First, in households with more than one eligible child, the older child was selected according to the survey protocol to minimize information bias. This non-random selection may have introduced age-related selection bias and under-represented younger eligible children. Second, most indoor environmental indicators were based on a single on-site measurement, which may not adequately represent children's long-term exposure or temporal variability and may therefore have resulted in exposure misclassification. Household-level exposure was also estimated using the arithmetic mean of bedroom and living room measurements without time-activity weighting, which may not fully reflect individual exposure patterns. In addition, all measurements were conducted during winter, limiting the assessment of seasonal variation. Third, although several individual, spatial, and temporal covariates were included in the fully adjusted models, other residential characteristics could not be comprehensively incorporated because of the modest sample size, leaving the possibility of residual confounding. Fourth, the outcomes

were based on parent-reported allergic symptoms rather than clinically diagnosed diseases, and the cross-sectional design precluded establishing temporal sequence or causal relationships. The modest number of outcome events relative to the number of covariates in the fully adjusted models may have reduced precision and increased uncertainty in some estimates. Simulation-based power analyses indicated approximately 80% power to detect ORs of about 2.0 per 1-SD increase in exposure, suggesting that smaller associations may have remained undetected. Finally, multiple environmental indicators and exploratory modeling approaches were evaluated, increasing the possibility of chance findings despite FDR correction in the primary analyses. Therefore, the overall findings should be interpreted as exploratory and require confirmation in larger longitudinal studies with repeated exposure assessments and more comprehensive residential information.

From a public health perspective, the present findings do not provide robust evidence that any specific indoor environmental indicator was independently associated with childhood respiratory allergic symptoms after full covariate adjustment and correction for multiple comparisons. Nevertheless, the nominal and exploratory patterns observed across the analyses suggest that residential indoor environments warrant further investigation alongside outdoor environmental exposures. Given the observational design and uncertainty of the present estimates, no specific indoor environmental intervention can be recommended on the basis of these findings alone. Future longitudinal and intervention studies incorporating repeated exposure assessments and more detailed characterization of residential, chemical, and biological conditions are needed to clarify whether specific indoor environmental factors are associated with childhood respiratory allergic symptoms.

5. Conclusions

This study identified several nominal associations between indoor environmental indicators and asthma-related or allergic rhinitis-related symptoms in children. However, no association remained statistically significant after full adjustment for individual, spatial, and interannual covariates and FDR correction. The observed associations were sensitive to model specification and should therefore be considered exploratory rather than confirmatory. Given the cross-sectional design, modest sample size, and reliance on single on-site exposure measurements, causal inference is not possible. Larger longitudinal studies with repeated exposure assessments and more comprehensive residential information are needed to further evaluate these associations.

Supplementary Materials: The following supporting information can be downloaded at <https://www.mdpi.com/article/10.3390/buildings16183607/s1>, Table S1: Indoor environmental exposure information; Table S2: Nominal and FDR-adjusted *p* values; Table S3: Temperature-unadjusted GEE results; Table S4. District- and year-adjusted comparison models; Table S5. Categorical exposure analysis results; Table S6: Results of RCS analyses; Table S7: Exploratory WQS regression results; Figure S1: Restricted cubic spline analyses of indoor environmental indicators and respiratory allergic symptoms in children. Figure S2: Exploratory WQS analyses for asthma-related and allergic rhinitis-related symptoms.

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Institutional Review Board Statement: All subjects gave their informed consent for inclusion before they participated in the study. The study was conducted in accordance with the Declaration of Helsinki, and the protocol was approved by the Ethics Committee of National Institute of Environmental Health, Chinese Center for Disease Control and Prevention (NIEH202320, NIEH202430).

Informed Consent Statement: Informed consent was obtained from all subjects involved in the study.

Data Availability Statement: The data presented in this study are available from the corresponding author upon reasonable request. The data are not publicly available because they contain information involving child participants and residential environments and are subject to privacy and ethical restrictions.

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Abbreviations

The following abbreviations are used in this manuscript:

RH	Relative Humidity
O ₃	Ozone
NO ₂	Nitrogen Dioxide
SO ₂	Sulfur Dioxide
CO ₂	Carbon Dioxide
CO	Carbon Monoxide
NH ₃	Ammonia
HCHO	Formaldehyde
TVOC	Total Volatile Organic Compounds
PM ₁₀	particulate matter with an aerodynamic diameter $\leq 10 \mu\text{m}$
PM _{2.5}	particulate matter with an aerodynamic diameter $\leq 2.5 \mu\text{m}$
TBC	Total bacterial count
TFC	Total fungal count
FDR	False discovery rate
RCS	Restricted Cubic Spline
WQS	Weighted Quantile Sum
GEE	Generalized Estimating Equation
ICC	Intraclass Correlation Coefficient
LOD	Limit of Detection

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