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Ambient Humidity and Childhood Food Allergy in a Tropical Climate: Insights from a Population-Based Study

Yihao Xue
Hainan Branch, Shanghai Children's Medical Center, School of Medicine, Shanghai Jiao Tong University
Huike Feng
Hainan Branch, Shanghai Children's Medical Center, School of Medicine, Shanghai Jiao Tong University
Wangyang Gu
Hainan Branch, Shanghai Children's Medical Center, School of Medicine, Shanghai Jiao Tong University
Qingpeng Li
Hainan Branch, Shanghai Children's Medical Center, School of Medicine, Shanghai Jiao Tong University
Linling Xu
Hainan Branch, Shanghai Children's Medical Center, School of Medicine, Shanghai Jiao Tong University
Yang Chen
Shanghai Municipal Hospital of Traditional Chinese Medicine, Shanghai University of Traditional Chinese Medicine
Shilu Tong
Queensland University of Technology
Dan Wang
Hainan Branch, Shanghai Children's Medical Center, School of Medicine, Shanghai Jiao Tong University
Shijian Liu
shijianliu@shsmu.edu.cn

Shanghai Municipal Hospital of Traditional Chinese Medicine, Shanghai University of Traditional Chinese Medicine

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Abstract

Background

Childhood food allergy (FA) is an increasing global public health concern; however, the role of tropical climate-related environmental exposures in childhood FA remains poorly understood.

Objective

We aimed to investigate the associations between long-term meteorological exposures and childhood FA prevalence in a tropical population.

Methods

This population-based cross-sectional study (n = 9,130; aged 2–10 years) was conducted in Sanya, China, in 2022. We estimated individualized, time-weighted meteorological exposures by integrating high-resolution grid-based data with geocoded residential and school addresses. Annual mean temperature, relative humidity (RH), ultraviolet B (UVB) radiation, and precipitation were evaluated. Associations were assessed using multivariate logistic regression and restricted cubic spline (RCS) models, adjusting for sociodemographic and household environmental factors.

Results

The prevalence of doctor-diagnosed FA was 5.2%. Ambient RH and temperature were independently and positively associated with FA. Each interquartile range (IQR) increase in RH and temperature corresponded to elevated FA risk, with adjusted odds ratios of 1.485 (95% CI: 1.245–1.773) and 1.916 (95% CI: 1.281–2.864), respectively. RCS analyses demonstrated near-linear exposure-response relationships for both parameters, whereas UVB radiation and precipitation exhibited no significant associations.

Conclusions

High ambient humidity and temperature serve as critical environmental determinants for childhood FA in tropical climates. These findings emphasize the necessity of integrating indoor humidity control, healthy building design, and optimized ventilation strategies to mitigate climate-related pediatric health vulnerabilities.

Contributions to the literature

● Research on environmental determinants of childhood food allergy has predominantly focused on temperate populations; this study extends the evidence to children living in a tropical climate.

● Higher ambient relative humidity and temperature were independently associated with childhood food allergy, suggesting that climate-related environmental conditions may contribute to variation in childhood allergic health.

● Individualized time-weighted meteorological exposure estimates, derived from high-resolution gridded data and geocoded residential and school locations, enabled a more spatially refined assessment of meteorological exposures at the population level.

● The findings underscore the importance of considering climatic context in environmental health research on childhood allergy and highlight the need for longitudinal and multisite studies to clarify these associations.

Introduction

Food allergy has become an increasingly serious global public health challenge. As an abnormal immune response to specific food antigens, FA can affect multiple target organs and, in severe cases, even lead to life-threatening anaphylactic shock [1, 2]. In recent decades, the prevalence of food allergies in children has continued to rise, with a trend toward younger onset [3]. Given that genetic factors cannot undergo drastic changes in the short term, identifying modifiable environmental determinants during early life has become a central scientific issue in the global prevention and control of allergic diseases [4, 5]. Therefore, identifying modifiable environmental risk factors is of great significance for the development of targeted allergy prevention strategies.

In recent years, the impact of macro-meteorological factors (such as temperature, relative humidity, ultraviolet radiation, and precipitation) on immune-mediated diseases has gradually attracted much research attention [6, 7]. Previous studies have shown that changes in meteorological conditions not only directly affect the abundance and spatial distribution of environmental allergens such as pollen, mold, and house dust mites, but also can profoundly influence an individual's immune tolerance status by modulating skin barrier integrity or vitamin D synthesis pathways [8, 9]. Currently, a substantial body of epidemiological evidence has confirmed that meteorological factors are closely associated with respiratory and cutaneous allergic diseases such as asthma, allergic rhinitis, and atopic dermatitis [10–13]. However, although "percutaneous sensitization" is widely recognized as one of the key pathways for the development of food allergy [14], epidemiological evidence regarding whether meteorological factors directly influence the risk of food allergies in children remains limited, and existing conclusions are inconsistent.

First, the vast majority of existing evidence originates from temperate climates in North America, Europe, and East Asia, while studies on tropical and subtropical populations are extremely rare [15–19]. Tropical

regions are characterized by year-round high temperatures, high humidity, and high solar radiation. These unique ecological features may shape specific allergen exposure profiles and immune adaptation mechanisms, making it difficult to directly extrapolate conclusions from studies conducted in temperate climates to tropical populations[20]. Second, previous studies have largely failed to adequately control for complex spatiotemporal confounders, making it difficult to quantify the independent effects of individual meteorological variables. Furthermore, current epidemiological studies examining the association between meteorological factors and allergies still exhibit significant geographical bias and methodological limitations. More critically, the actual health effects of macroclimate are often modified by micro-environmental factors, yet few large-scale studies have systematically assessed the combined impact of “macroclimate–micro-indoor environment” interactions on the risk of FA.

Based on the aforementioned knowledge gaps in the evidence, we conducted a large-scale, population-based epidemiological study in Sanya, a typical tropical coastal city in southern China. Combining the “cutaneous sensitization” hypothesis with tropical ecological and climatic characteristics, we propose the following core hypothesis: in a tropical environment characterized by minimal seasonal variation and year-round high humidity, environmental relative humidity is an independent risk factor driving increased risk of FA in children; furthermore, the main effect of this macro-meteorological factor is significantly modified and amplified by specific indoor microenvironmental characteristics.

Combining the “transdermal sensitization” hypothesis with the ecological and climatic characteristics of the tropics, we propose the following core hypothesis: in tropical environments characterized by minimal seasonal variation and year-round high humidity, environmental relative humidity is an independent risk factor driving an increased risk of FA in children; furthermore, relative humidity may be modified by specific indoor microenvironmental characteristics. To test this hypothesis, this study aims to: (1) elucidate the independent association between tropical meteorological exposure and the risk of childhood FA, as well as the potential nonlinear exposure-response relationship; and (2) systematically evaluate the potential effect-modifying roles of individual characteristics and indoor microenvironmental factors on this meteorological factors and FA association. This study provides new insights into the etiological mechanisms of childhood FA from both environmental and climatic perspectives, and offers robust empirical evidence for the development of precise, climate-adaptive public health policies for allergy prevention and control in tropical regions.

Methods

Study Design and Study Population

This study is a population-based cross-sectional study conducted in Sanya, Hainan Province, China. Sanya is located in a low-latitude tropical monsoon climate zone and is characterized by high temperatures, high humidity, and high ultraviolet radiation throughout the year. The study was conducted from October to December 2022. A stratified cluster sampling method was used to recruit participants from kindergartens and elementary schools across different administrative districts of the city. Data

were collected using a standardized parent questionnaire completed by the legal guardians of the participating children[21].

Inclusion criteria included: (1) children aged 2–10 years; (2) having resided at the current address for ≥ 1 year; (3) complete information on key variables. Exclusion criteria included: (1) missing data on primary outcome variables; (2) missing residential address information; (3) age outside the inclusion range. Of the 10,000 children invited to participate, 9,130 were ultimately included in the analysis.

Definition of Outcome Variables

This study collected data using a standardized questionnaire based on the core questionnaire of the International Study of Asthma and Allergies in Childhood (ISAAC), which had been localized, revised, and cross-culturally validated [21]. The primary outcome was doctor-diagnosed childhood food allergy. To ensure the specificity of outcome classification, the case definition for food allergy was based on a history of explicit medical diagnosis reported by parents (i.e., respondents who answered “Yes” to the questionnaire item: “Has your child ever been explicitly diagnosed with a food allergy by a doctor?”).

Environmental Exposure Assessment

This study integrated high-resolution spatial grid data with individual activity patterns to conduct precise time-weighted exposure assessments. Meteorological variables included annual average temperature (°C), relative humidity (%), ultraviolet B (UVB) radiation (kJ/m²), and precipitation (mm) for the year 2022. All meteorological grid data were extracted from the National Qinghai-Tibet Plateau Scientific Data Center (TPDC) and the ERA5 reanalysis dataset, with a spatial resolution of 1 km × 1 km.

Using geocoding technology, the home addresses and school addresses of all participants were converted into precise latitude and longitude coordinates and matched to spatial meteorological grids [22]. Taking into account the daily activity patterns of preschool and school-aged children, we constructed a time-weighted personal exposure model based on the participants’ time allocation between school and home, calculated as follows:

$$E_{\text{weighted_avg}} = \frac{(C_{\text{school}} \times t_{\text{school}}) + (C_{\text{home}} \times t_{\text{home}})}{24}$$

In this context, Eweighted-avg represents the individual’s daily time-weighted average meteorological exposure; Cschool denotes the concentration of the meteorological indicator at the grid cell where the school is located; Chome denotes the concentration of the meteorological indicator at the grid cell where the residence is located; and tschool (set to 8 hours) and thome (set to 16 hours) represent the daily time spent at school and at home, respectively.

Given the high temporal stability of tropical climates, this study primarily utilized spatial variation/contrast among participants in the model to quantitatively assess the epidemiological effects of meteorological factors on health. All meteorological exposure indicators were included as continuous variables in the subsequent statistical models.

Measurement and Definition of Covariates

Drawing on prior knowledge from the literature and the causal inference framework based on directed acyclic graphs (DAGs)[22, 23], this study comprehensively identified and defined potential spatiotemporal confounders. The included covariates were categorized into the following four dimensions:

1) Demographic and socioeconomic characteristics: Child's age (continuous variable), gender (male/female), household monthly income (< 3,000, 3,000–5,999, 6,000–8,999, 9,000–11,999, ≥ 12,000 CNY). 2) Prenatal and Early-Life Exposure: Antibiotic use during pregnancy (yes/no), history of miscarriage (yes/no), antibiotic use during infancy (yes/no), feeding method (exclusive breastfeeding/mixed feeding/formula). 3) Family and Living Environment: Family history of allergies (Yes/No), Presence of pets (Yes/No), Exposure to secondhand smoke (Yes/No), Exposure to traffic pollution (Residence within 50 m of traffic noise), Use of household insecticides (Yes/No), Cleaning frequency (Daily, 4–6 times per week, 1–3 times per week, 1–3 times per month, Less than once per month). 4) Indoor ventilation methods: Natural ventilation / Standard air conditioning / Central air conditioning / Fans.

Statistical Analysis

Descriptive and Correlational Analysis

Continuous variables were described using the mean ± standard deviation (SD) or the median and interquartile range (IQR), depending on the normality of their distribution; categorical variables were presented as frequencies and percentages (%). Differences between groups were compared using Student's t-test or the Mann-Whitney U test. The correlation between meteorological variables and potential air pollutants was assessed using Spearman's rank correlation matrix, and the Variance Inflation Factor (VIF) was calculated. To avoid multicollinearity, variables with a VIF > 5 were excluded from the main model.

Multivariate Logistic Regression Model

We used a multivariate logistic regression model to assess the independent association between meteorological exposure and the risk of food allergies in children. To test the robustness of the results, we constructed stepwise models adjusting for different sets of confounders: Model 1: Crude model (no

adjustment for any confounding factors). Model 2: Model 1 adjusted for core demographic variables (age, sex). Model 3: Model 2 further adjusted for perinatal factors and socioeconomic status (e.g., antibiotic use during pregnancy, household income). Model 4 (Fully Adjusted Model): Includes all family microenvironmental characteristics (family history of allergies, pets, secondhand smoke, traffic exposure, pesticides, and cleaning frequency) and other non-collinear co-exposure meteorological variables on top of Model 3. To control for multicollinearity, the Variance Inflation Factor (VIF) was calculated, and variables with a VIF > 5 were excluded. Effect estimates are presented as odds ratios (ORs) and 95% confidence intervals (95% CIs).

Nonlinear Exposure-Response Relationship

Restricted cubic spline (RCS) functions were used to model continuous meteorological variables in order to explore potential nonlinear relationships[24].The number of knots was set to 4, located at the 5th, 35th, 65th, and 95th percentiles of the continuous variables' distribution. The goodness of fit between the linear and nonlinear models was compared using a likelihood ratio test, and the p-value for nonlinearity was reported. Results were visualized as exposure–response curves.

Subgroup Analysis and Interaction Analysis

To identify potential effect modifiers, stratified analyses were conducted in predefined subgroups (e.g., sex, family history of allergies, indoor ventilation methods) based on biological plausibility. Multiplicative interaction terms for “meteorological exposure × stratification variable” were included in the fully adjusted models, and the statistical significance of these interactions was assessed using the Wald test.

Handling Missing Values and Sensitivity Analysis

To address missing covariates, we used the Multiple Imputation by Chained Equations (MICE) method to generate five imputed datasets and combined the effect estimates based on Rubin’s rules[25, 26]. To rule out potential confounding factors and multicollinearity arising from simultaneous exposure to meteorological factors, we conducted sensitivity analyses using a series of single-factor and multi-meteorological-factor models. Model 1 is a single-factor model that estimates the effect of relative humidity alone, while adjusting only for individual and environmental covariates. Models 2, 3, and 4 are two-factor models that additionally control for temperature, precipitation, and UVB, respectively. Model 5 is our fully configured core model, which includes all three meteorological indicators. We assessed the robustness of the independent effects by comparing the stability of the odds ratios (ORs) and variance inflation factors (VIFs) across these models. All analyses were performed using R software (version 4.5.1), with commonly used packages including rms, splines, mice, and ggplot2. Statistical tests were two-sided, with a significance level set at $p < 0.05$.

This study was approved by the Ethics Committee of Sanya Maternal and Child Health Hospital (Approval No. : SYFYIRB20220063), and the legal guardians of all participants signed written informed

consent forms prior to the survey. The reporting of this cross-sectional study followed the STROBE guideline[27].

Results

Baseline Characteristics of the Study Population

This study ultimately included 9,130 children with complete core data. Among them, 478 (5.2%) had a history of clinically diagnosed food allergy (FA). The median age of the study population was 6.75 years (interquartile range [IQR]: 2.8), and 54.9% were boys. Comparisons of baseline characteristics revealed that, compared with children without FA, those with FA exhibited distinct patterns in sociodemographic factors and early-life exposures: their household monthly income was significantly higher, and the proportions of maternal history of miscarriage, antibiotic exposure during pregnancy and infancy, and family history of allergies were all significantly increased (all $P < 0.001$). Regarding household microenvironmental characteristics, children with FA had significantly higher rates of pet exposure since pregnancy, household pesticide use, and frequent household cleaning compared to the control group (all $P < 0.05$). No statistically significant differences were observed between the two groups in terms of age, gender distribution, or rates of secondhand smoke exposure (Table 1).

Table 1
The basic characteristics of participants with and without food allergy in children.

Characteristics	Total (n = 9130)	Food allergy group (n = 478)	Non-food allergy group (n = 8652)	*p*
Child age, years, median (IQR)	6.75 (2.8)	6.43(2.1)	6.51(3.1)	0.289
Sex, n (%)				**0.74**
Boys	5014(54.9)	266(55.6)	4748(54.9)	
Girls	4116(45.1)	212(44.4)	3904(45.1)	
Household income per month, CNY, n (%				**< 0.001**
< 3000	2861 (31.3)	100(20.9)	2761(31.9)	
3000–5999	2665 (29.2)	113(23.6)	2552(29.5)	
6000–8999	1465 (16.1)	97(20.3)	1368(15.8)	
9000–11,999	905 (9.9)	48(10)	857(9.9)	
> 12,000	1234 (13.5)	120(25.1)	1114(12.9)	
History of miscarriage, n (%)				**< 0.001**
Yes	2114 (23.2)	150(31.4)	1964(22.7)	
No	7016 (76.9)	328(68.6)	6688(77.3)	
Antibiotic use during pregnancy, n (%)				**< 0.001**
Yes	174 (1.9)	27(5.6)	147(1.7)	
No	8956 (98.1)	451(94.4)	8505(98.3)	
Environmental tobacco smoke (ETS),n (%)				**0.525**
Yes	6821(74.7)	363(75.9)	6458(74.6)	
No	2309(25.3)	115(24.1)	2194(25.4)	
Feeding patterns (< 4 months), n (%)				**0.127**
Exclusive breastfeeding	5178 (56.7)	289(60.5)	4889(56.6)	
Mixed	2713 (29.7)	137(28.7)	2676(29.8)	

P-Value for sociodemographic between two groups was calculated by student-t-test or chi-square test appropriately. *P-Value* for environmental exposures between two groups was calculated by Mann-Whitney U test. Statistically significant results (*P-value < 0.05*) are in bold.

Characteristics	Total (n = 9130)	Food allergy group (n = 478)	Non-food allergy group (n = 8652)	*p*
Formula	1239 (13.6)	52(10.9)	1187(13.7)	
Antibiotic use within the first year, n (%)				**< 0.001**
Yes	1518 (16.6)	149(31.2)	1369(15.8)	
No	7612 (83.4)	329(68.8)	7283(84.2)	
Family history of allergy, n (%)				**< 0.001**
Yes	3089 (33.8)	291(60.9)	2798(32.3)	
No	6041 (66.2)	187(39.1)	5854(67.7)	
Traffic within 50 m, n (%)				**< 0.001**
Much	2398 (26.3)	152(31.8)	2246(26.0)	
Not much	3814 (41.8)	208(43.5)	3606(41.7)	
Less	2918 (32.0)	118(24.7)	2800(32.4)	
Having a pet since conception, n (%)				**< 0.001**
Yes	1,066 (11.7)	90(18.8)	976(11.3)	
No	8,064 (88.3)	388(81.2)	7676(88.7)	
Insecticide use, n (%)				**< 0.001**
Yes	3,459 (37.9)	239(50.0)	3220(37.2)	
No	5,671 (62.1)	239(50.0)	5432(62.8)	
Ventilation or refrigeration is used, n (%)				**0.004**
natural ventilation	1565(17.1)	70(14.6)	1495(17.3)	
General air conditioning	5630(61.7)	315(65.9)	5315(61.4)	
Central air-conditioning	329(3.6)	27(5.6)	302(3.5)	
Fanner	1606(17.6)	66(13.8)	1540(17.8)	
Household cleaning frequency,times,n(%)				**0.003**

P-Value for sociodemographic between two groups was calculated by student-t-test or chi-square test appropriately. *P-Value* for environmental exposures between two groups was calculated by Mann-Whitney U test. Statistically significant results (*P-value < 0.05*) are in bold.

Characteristics	Total (n = 9130)	Food allergy group (n = 478)	Non-food allergy group (n = 8652)	*p*
Everyday	1572(17.2)	112(23.4)	1460(16.9)	
4–6 times a week	6214(68.1)	305(63.8)	5905(68.3)	
1–3 times a week	1197(13.1)	53(11.1)	1144(13.2)	
1–3 times a month	99(1.1)	7(1.5)	92(1.1)	
Less than once a month	48(0.5)	1(0.2)	47(0.5)	
Redecoration, n (%)				**0.019**
Yes	2626(28.8)	160(33.5)	2466(28.5)	
No	6504(71.2)	318(66.5)	6186(71.5)	

P-Value for sociodemographic between two groups was calculated by student-t-test or chi-square test appropriately. *P-Value* for environmental exposures between two groups was calculated by Mann-Whitney U test. Statistically significant results (*P-value < 0.05*) are in bold.

Meteorological Exposure and Environmental Characteristics

The results of the time-weighted individual exposure assessment indicate that meteorological conditions in the study area exhibit typical tropical characteristics, namely high temperatures, high humidity, and low temporal variability. The median annual temperature exposure for the children was 26.14°C (IQR: 0.18°C), and the median relative humidity was 82.48% (IQR: 1.74%). The median UVB radiation exposure was 528.19 kJ/m² (IQR: 5.65), and the median annual precipitation was 1297.89 mm (IQR: 29.73). Furthermore, concentrations of ambient air pollutants in the study area were generally low, with median concentrations of O_3, NO_2, PM_{10}, $PM_{2.5}$, and SO_2 at 87.63, 10.92, 30.38, 16.22, and 14.95 μg/m³, respectively (Table S1).

Assessment of Correlations and Multicollinearity Among Environmental Variables

Spearman's rank correlation analyses indicate weak univariate correlations between individual meteorological exposure indicators and the prevalence of FA, with relative humidity exhibiting the highest correlation coefficient (r_S= 0.050). Regarding the intercorrelations among environmental variables, relative humidity showed strong negative correlations with both air temperature (r_S = − 0.704) and UVB radiation (r_S = − 0.899). Furthermore, there were extremely strong positive correlations among various air pollutants (e.g., $r_S \geq 0.70$ between NO_2, $PM_{2.5}$, PM_{10}, and SO_2). This highly correlated exposure matrix suggests that including multiple environmental indicators simultaneously in the same multivariate model poses a significant risk of multicollinearity, providing a methodological basis for implementing dimension reduction strategies and residual control in subsequent main models (Fig. 2).

Independent Associations between Meteorological Factors and Food Allergy

Figure.3 presents the results of multivariate logistic regression analyses after stepwise adjustment for potential confounding factors. In the fully adjusted model (Model 4), which accounted for demographic characteristics, early-life exposures, socioeconomic status, and multidimensional microenvironmental factors, environmental relative humidity was significantly and independently associated with the risk of childhood food allergy: For every 1 IQR increase in relative humidity, the odds ratio (OR) for food allergy increased (OR = 1.485, 95% CI: 1.245–1.773, P < 0.001). Similarly, air temperature also showed a significant positive association in the fully adjusted model (OR = 1.916, 95% CI: 1.281–2.864, P = 0.002). In contrast, after fully adjusting for confounding factors, no statistically significant association was observed between UVB radiation or precipitation and food allergy.

Non-linear Exposure-Response Relationships

To characterize the continuous effect trajectories of meteorological exposure, this study employed a restricted cubic spline (RCS) model to assess the potential nonlinear association between meteorological factors and the risk of FA. The RCS curves indicate that relative humidity exhibits a near-monotonic increasing exposure-response pattern with FA risk; specifically, the risk of developing the disease shows a steady upward trend in the high-humidity range. Regarding temperature exposure, although a slight upward trend in risk was observed in the high-temperature range, the 95% confidence interval was wide due to the distribution of extreme values in the sample. Non-linearity tests confirmed that the exposure-response relationships between temperature and relative humidity and FA both better fit the linear assumption (P for non-linearity > 0.05), whereas UVB and precipitation did not exhibit any statistically significant linear or non-linear trends (Fig.S1).

Subgroup Stratification and Sensitivity Analysis

Stratified analyses demonstrated that the hazardous effect of ambient relative humidity on childhood FA remained highly consistent across most demographic and environmental strata (Fig. 4), including sex (Boys: OR = 1.31, 95% CI: 1.10–1.55; Girls: OR = 1.33, 95% CI: 1.14–1.55) and maternal history of miscarriage (Yes: OR = 1.27; No: OR = 1.33). Concurrently, tests for multiplicative interaction revealed no statistically significant effect modification between ambient humidity and any individual or indoor environmental attributes (all P-interaction > 0.05; Table S5), indicating a homogeneous risk profile across the study population.

Sensitivity Analyses

To verify the independent effect of relative humidity, multi-exposure models were configured (Table 2). The positive association between humidity and FA remained robustly significant across all models after adjustment for a range of confounders, spanning from the single-exposure model (OR = 1.307, 95% CI: 1.157–1.477, P < 0.001) to models co-adjusting for temperature (OR = 1.502, P = 0.015), precipitation

(OR = 1.305, P < 0.001), UVB radiation (OR = 1.420, P < 0.001), and both temperature and UVB simultaneously (OR = 1.464, 95% CI: 1.234–1.737, P < 0.001).

Table 2
Sensitivity analysis of humidity and food allergy under different meteorological models.

Model	Meteorological Co-exposure	OR (95% CI)	*P-value*
Model 1	Humidity	1.307 (1.157–1.477)	**< 0.001**
Model 2	Humidity + Temperature	1.502 (1.083–2.083)	**0.015**
Model 3	Humidity + Precipitation	1.305 (1.154–1.475)	**< 0.001**
Model 4	Humidity + UVB	1.420 (1.200–1.680)	**< 0.001**
Model 5	Humidity + Temperature + UVB	1.464 (1.234–1.737)	**< 0.001**

Note: OR, odds ratio; CI, confidence interval; UVB, ultraviolet radiation B. [a]All four models were consistently adjusted for the full set of individual and environmental covariates, including child's age, sex, delivery mode, feeding practices, family history of allergies, household cleaning frequency, home redecoration, pet ownership, maternal history of miscarriage, and antibiotic use during pregnancy.

Discussion

Based on large-scale, representative population data from a typical tropical coastal city in southern China, this study comprehensively assessed the epidemiological association between macro-meteorological exposure and food allergy (FA) in children. Key findings indicate that, after fully adjusting for demographic characteristics, socioeconomic factors, household microenvironmental exposures, and air pollutants, environmental relative humidity was significantly and independently positively associated with the risk of childhood food allergy; furthermore, restricted cubic spline (RCS) analysis suggested that this exposure–response relationship was approximately linear. Furthermore, air temperature exhibited a positive association at higher exposure levels, whereas no independent effects were observed for ultraviolet radiation (UVB) or precipitation. This study fills the evidence gap in current environmental epidemiological research on allergies, which has primarily focused on temperate regions, and provides important population-based data for understanding the environmental determinants of food allergies in tropical climates[3, 28, 29].

Possible explanations for the observed association

The positive association between “high humidity and food allergies” observed in this study is biologically plausible, and its underlying mechanisms align perfectly with the “Epithelial Barrier Hypothesis”[6, 30] and the “Dual-Allergen Exposure Hypothesis”[31], which represent the current state-of-the-art in allergy pathogenesis. Relative humidity is a key factor in regulating the environment of indoor biological allergens, particularly influencing the survival and proliferation of house dust mites and fungi. When relative humidity remains at high levels over an extended period, the indoor biological allergen load

increases significantly [32–34]. Experimental studies have shown that major house dust mite antigens (such as Der p 1) possess proteolytic activity, which can directly disrupt the tight junctions between epidermal and mucosal epithelial cells, thereby compromising barrier integrity and inducing a Th2-dominant immune response[35, 36]. In this pathological microenvironment, trace amounts of food antigens present in the home environment can easily cause “percutaneous sensitization”[37] through the compromised skin barrier, thereby bypassing the normal oral immune tolerance mechanisms of the gastrointestinal tract and ultimately triggering systemic food allergies.

The role of temperature in childhood food allergy

In addition to relative humidity, this study also observed a positive association between air temperature in the higher exposure range and the risk of food allergies. This finding can be explained from both physiological and environmental behavioral perspectives: physiologically, prolonged heat stress leads to increased sweating, and prolonged exposure to sweat significantly increases the permeability and fragility of the stratum corneum [38, 39]; At the same time, heat-induced vasodilation of the skin may accelerate the entry of food antigens that have penetrated the skin into the systemic circulation. Behaviorally, persistently high outdoor temperatures and humidity compel people to spend significantly more time in enclosed, air-conditioned environments. This modern indoor microenvironment—characterized by “enclosed spaces with inadequate microcirculation”—easily becomes a breeding ground for the prolonged accumulation of allergens and volatile organic compounds (VOCs), thereby amplifying the adverse effects of the macroclimate[40, 41].

Comparison with findings from temperate regions

The findings of this study differ somewhat from those of previous studies conducted in temperate regions in terms of meteorological risk patterns[28, 42]. In temperate regions, cold and dry environments (particularly the dry winter climate) are generally considered major risk factors for the development of atopic dermatitis and food allergies [43–45]. These differences across climatic zones are not contradictory; rather, they suggest the possible existence of “climate-specific pathways of sensitization” in the development of allergies. In dry, cold temperate environments, skin barrier damage is primarily driven by stratum corneum dehydration and the formation of microcracks caused by low humidity[46]; whereas in hot, humid tropical environments, barrier dysfunction is more likely mediated by high levels of biological allergens and their protease activity [36, 47]. Therefore, sensitization processes in different climatic settings may converge on similar immunological outcomes via distinct pathways[48]. This framework of “heterogeneous pathways but consistent outcomes” helps integrate seemingly inconsistent epidemiological evidence from different regions and underscores the need to fully consider regional climatic characteristics when formulating allergy prevention and control strategies.

Public health implications

Against the backdrop of climate change and rapid urbanization, this study holds significant public health implications [49, 50]. The findings suggest that, during the critical early-life windows when immune

tolerance is established, simply avoiding specific foods is no longer sufficient to prevent food allergies (FA). For susceptible families and childcare facilities located in tropical and high-humidity regions, public health interventions should prioritize “environmental source control”: specifically, the routine monitoring of indoor temperature and humidity, scientific physical dehumidification, and the improvement of natural ventilation in enclosed buildings. This serves as an economical and feasible primary health prevention strategy aimed at controlling the percutaneous sensitization [51].

Strengths and Limitations

The strengths of this study are primarily reflected in the following aspects: First, the large sample size drawn from a representative population provides robust statistical power for effect estimation. Second, regarding exposure measurement, this study overcomes the limitations of traditional research that relies excessively on a single weather station. Instead, it employs high-spatial-resolution grid data and innovatively constructs an individualized time-weighted exposure model, significantly reducing spatial exposure misclassification bias. Furthermore, this study employed restricted cubic spline models to characterize nonlinear relationships and, through systematic multivariate adjustment and multicollinearity control strategies, effectively disentangled the correlations between meteorological factors and air pollutants, thereby enhancing the robustness of the results.

This study also has several limitations. First, due to the cross-sectional study design, the concurrent assessment of exposure and outcome data inherently limits the inference of clear causal sequences (temporal ambiguity). Although Sanya’s climate exhibits extremely high interannual stability and this study strictly limited the duration of residence to approximate long-term exposure, there remains an urgent need to rely on prospective birth cohorts to precisely identify key exposure windows for environmental sensitization during early life. Second, given the practical feasibility of large-scale population-based epidemiological studies, the FA outcomes in this study relied primarily on parent-reported physician diagnoses and did not fully adopt Oral Food Challenge tests (OFC), the clinical gold standard. Such questionnaire-based measurements inevitably carried a risk of non-differential misclassification; however, it is worth emphasizing that, in statistical terms, this type of bias typically tends to shift effect estimates toward the null hypothesis, implying that the positive association between meteorological factors and allergies observed in this study may be even stronger in the real world. Finally, meteorological exposure in this study was based on outdoor grid data and did not directly measure indoor humidity or biological allergen loads at the individual level. Future studies could integrate indoor environmental monitoring and exposureomics methods to more accurately elucidate the mechanisms underlying the relationship between macroclimate and micro-exposure.

Conclusions

Overall, this study, adopting a climate-environmental perspective, provides important evidence from tropical populations supporting the potential key role of relative environmental humidity in the development of food allergies in children. This finding not only expands the current epidemiological

framework, which is primarily based on temperate regions, but also further highlights the possibility of climate-specific pathways in the sensitization process.

From a public health perspective, this study suggests that regional climatic characteristics should be incorporated into risk assessments and intervention designs when formulating allergy prevention and control strategies. Future research should further integrate prospective longitudinal data, mechanistic experimental studies, and policy interventions at the climate–health interface to establish a more targeted comprehensive food allergy prevention and control system.

Abbreviations

FA
Food Allergy
IgE
Immunoglobulin E
Th2
T helper type 2 cell
TEWL
Transepidermal Water Loss
HDM
House Dust Mite
$PM_{2.5}$/PM_{10}
Particulate Matter
UVB
Ultraviolet B radiation
ISAAC
International Study of Asthma and Allergies in Childhood
DAGs
Directed Acyclic Graphs
OR/aOR
(Adjusted) Odds Ratio
CI
Confidence Interval
IQR
Interquartile Range
RCS
Restricted Cubic Spline
MICE
Multiple Imputation by Chained Equations
VIF
Variance Inflation Factor

OFC
Oral Food Challenge

Declarations

Acknowledgements

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CRediT authorship contribution statement

YX and SL conceived and designed the study. YX conducted the data curation and formal analysis and drafted the manuscript. HF, QL, WG, LX, and YC contributed to data investigation, validation, and methodology. YC also contributed to data curation. ST, DW, and SL supervised the study and contributed to its conceptualization, methodology, and resources. ST, DW, and SL also contributed to funding acquisition. All authors contributed to the interpretation of the findings, critically revised the manuscript, and approved the final manuscript.

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Data availability

Environmental data are publicly available from the National Tibetan Plateau/Third Pole Environment Data Center (http://data.tpdc.ac.cn). Individual-level participant data are available from the corresponding author (shijianliu@shsmu.edu.cn) upon reasonable request.

Ethics approval

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Sanya Women and Children’s Hospital (Approval No. SYFYIRB20220063). Written informed consent was obtained from the parents or legal guardians of all participating children prior to the survey.

Competing interests

The authors declare no conflicts of interest.

Consent for publication

Not applicable.

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Figures

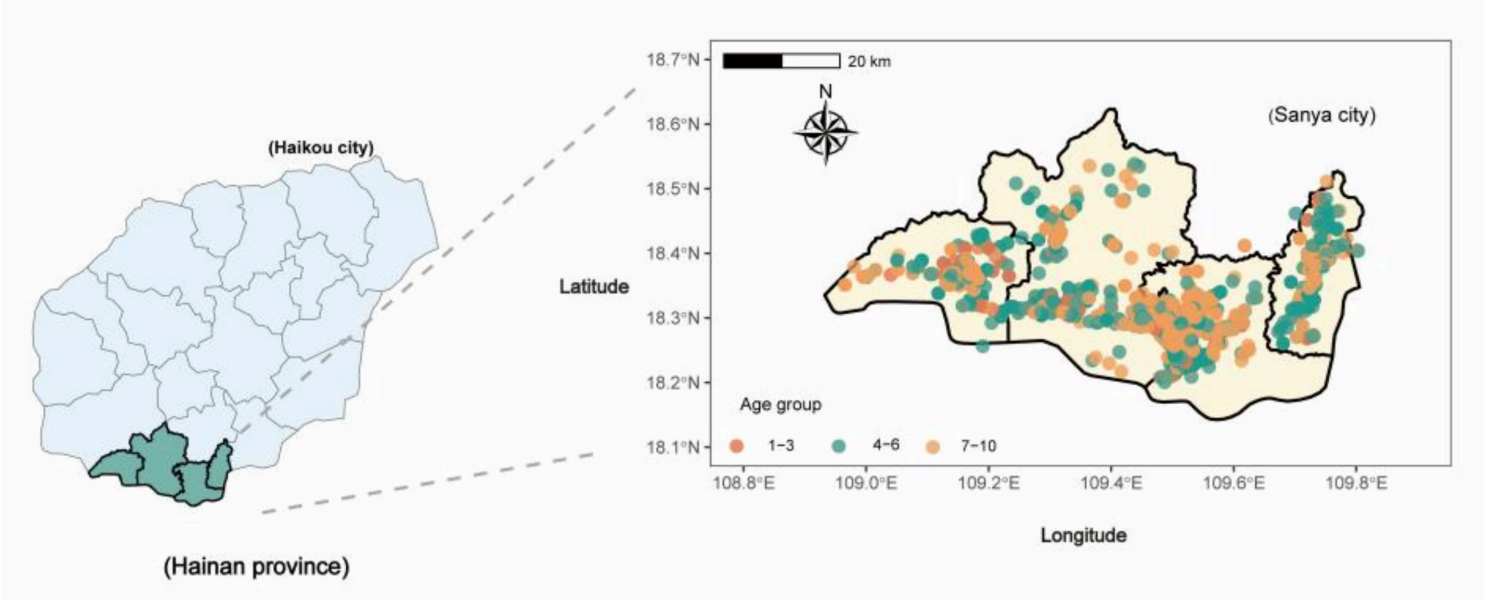


Fig.1. The geographical distribution of the study location and the participants' residential addresses.

Figure 1

See image above for figure legend

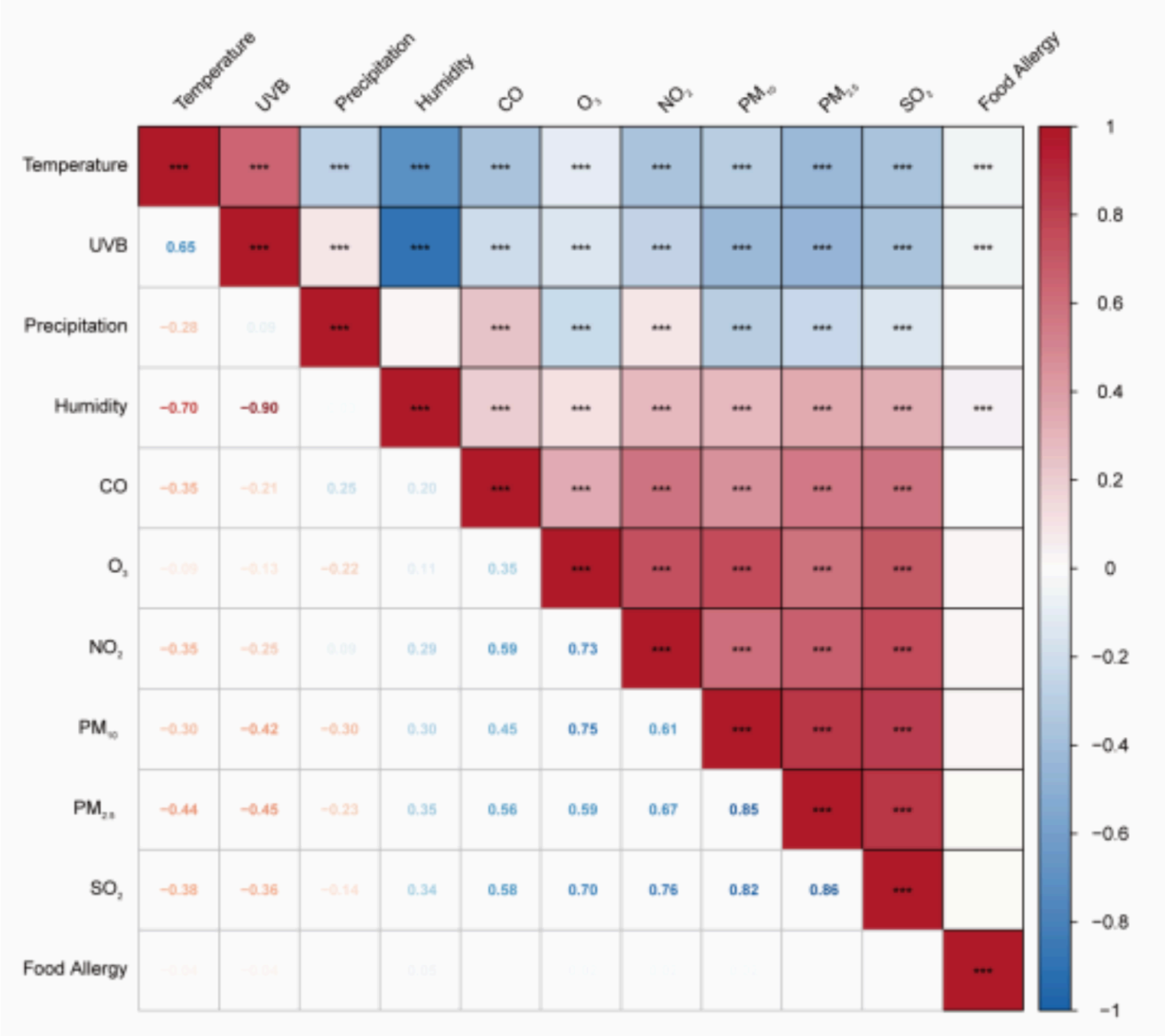


Fig.2. Spearman correlation of single meteorological factors and air pollutants withd food allergy risk.

Figure 2

See image above for figure legend.

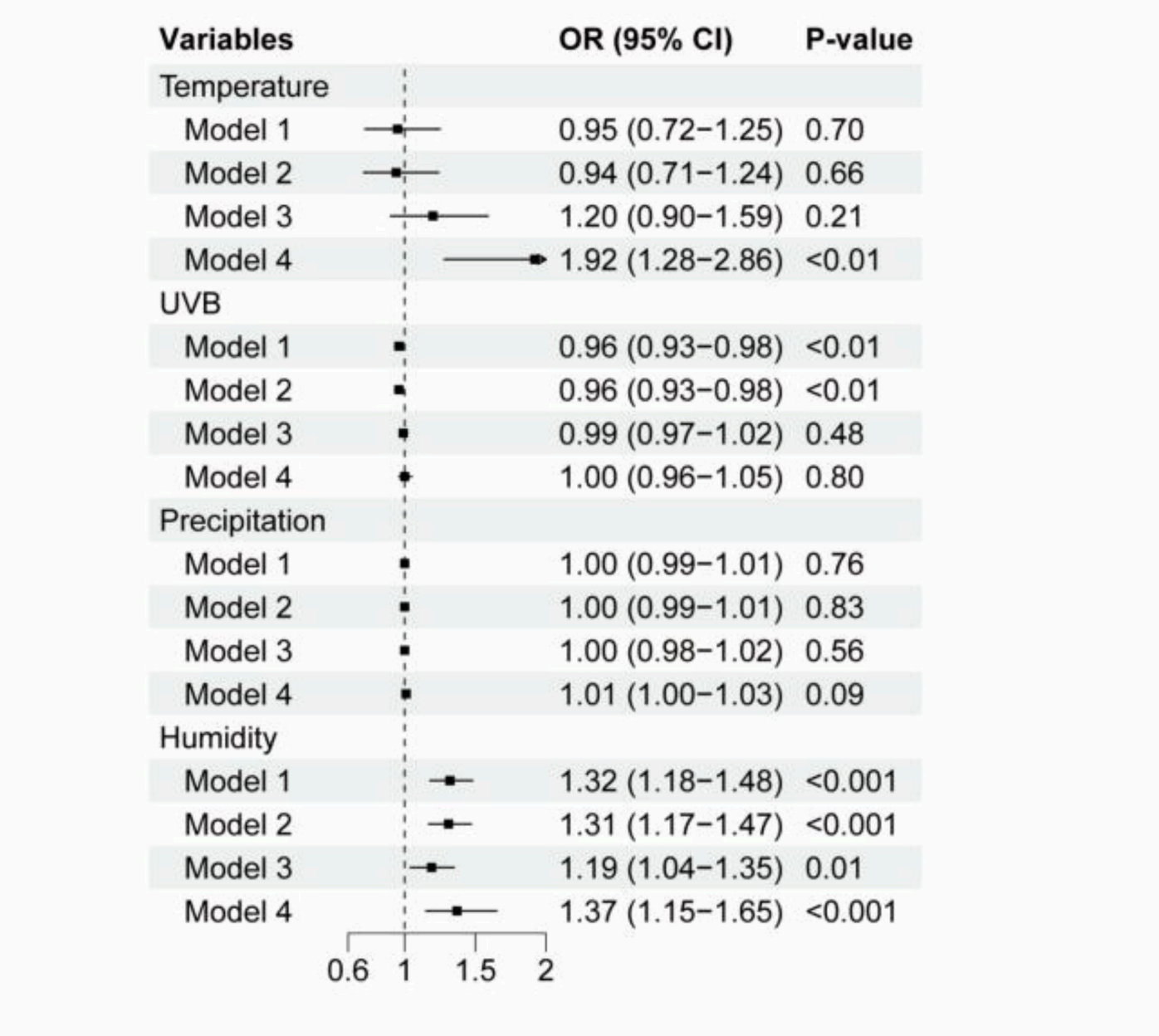


Fig. 3. Forest map of meteorological factors and food allergy using multivariate regression.Model 1 : Unadjusted univariate model. Model 2 : Based on Model 1,further adjusted demographic factors: Age, gender, history of abortion, early antibiotic use.Model 3 : Based on Model 2,further adjusted Sociological factors: prenatal antibiotic use, family history of allergies, pet feeding, traffic exposure, pesticide use, economic status, ventilation patterns, redecoration,cleaning frequency. Model 4 : Based on Model 3,further adjusted temperature, precipitation, UVB, humidity, and pollutant factors.

Figure 3

See image above for figure legend.

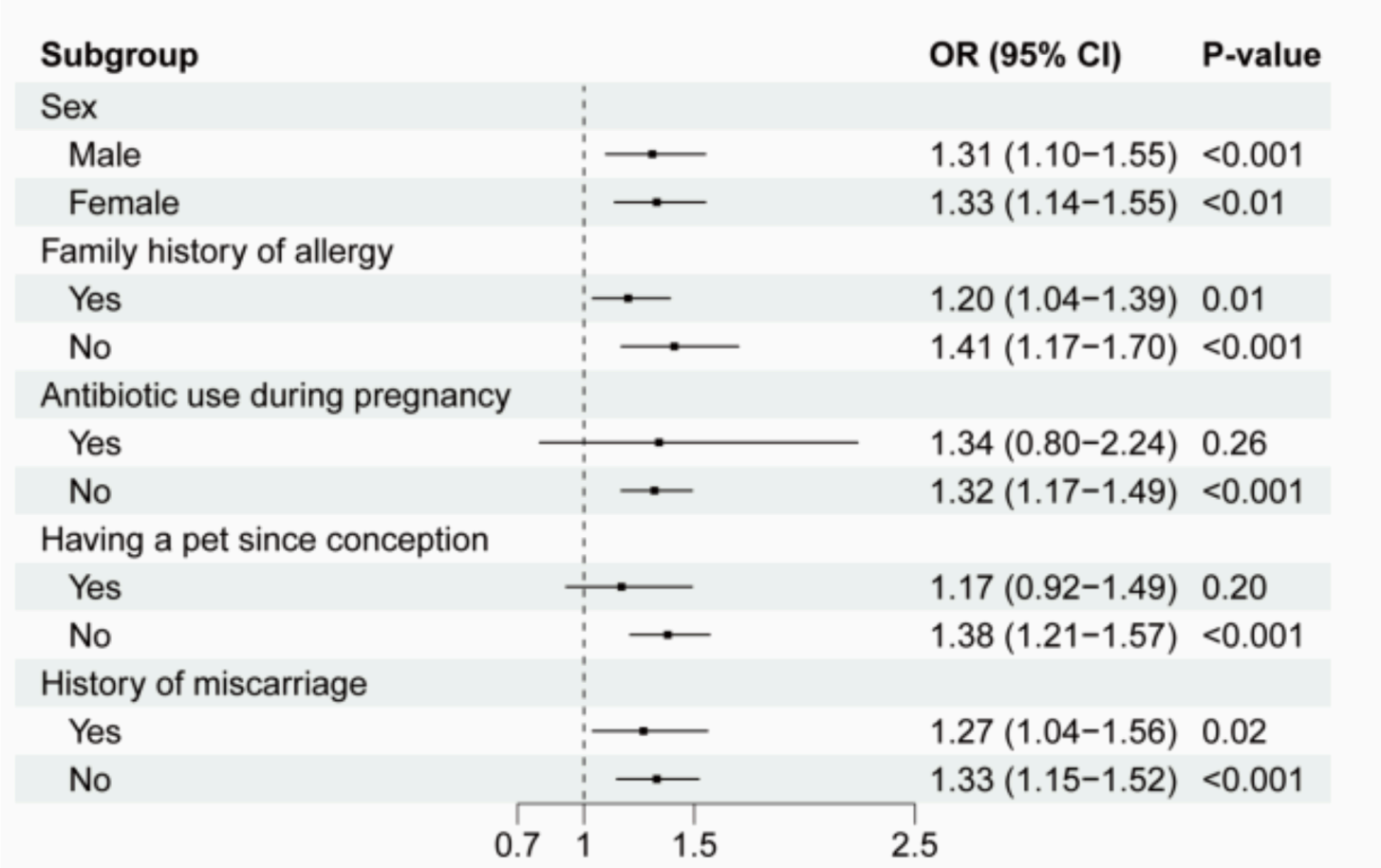


Fig.4. Subgroup analysis of the association between humidity and food allergy.

Figure 4

See image above for figure legend.

Supplementary Files

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