



Article

Outdoor Air Pollution and Indoor Window Condensation Associated with Childhood Symptoms of Allergic Rhinitis to Pollen

Yingjie Liu ^{1,†} , Chan Lu ^{2,†} , Yuguo Li ³, Dan Norbäck ⁴ and Qihong Deng ^{5,*}

¹ School of Energy Science and Engineering, Central South University, Changsha 410083, China; yingjieliu@csu.edu.cn

² XiangYa School of Public Health, Central South University, Changsha 410078, China; chanlu@csu.edu.cn

³ Department of Mechanical Engineering, The University of Hong Kong, Hong Kong 999077, China; liyg@hku.hk

⁴ Department of Medical Sciences, Uppsala University, 752 36 Uppsala, Sweden; dan.norbäck@medsci.uu.se

⁵ School of Public Health, Zhengzhou University, Zhengzhou 450001, China

* Correspondence: qhdeng@zzu.edu.cn

† These authors contributed equally to the work.

Abstract: Pollen is the main factor causing asthma and allergic rhinitis (AR). However, the key indoor and outdoor factors associated with childhood symptoms of allergic rhinitis (SAR) to pollen are unclear. We investigate the association of exposure to outdoor air pollution and indoor environmental factors with childhood SAR to pollen and consider SAR to pollen in different seasons. A cross-sectional study of 2598 preschool children aged 3–6 was conducted in Changsha, China (2011–2012). The prevalence of SAR to pollen in children and information on indoor environmental factors were obtained by questionnaire. Children’s exposure to outdoor air pollutants (PM₁₀, SO₂, and NO₂) was estimated from the monitored concentrations. The association of exposure to indoor environmental factors and outdoor air pollution with childhood SAR to pollen was estimated by multiple logistic regression models using odds ratio (OR) and a 95% confidence interval (CI), and the relationship between outdoor air pollutants and childhood SAR to pollen was investigated using restricted cubic splines. We found that early-life and current exposure to outdoor air pollution were significantly associated with childhood SAR to pollen in autumn, including exposure to SO₂ one year before conception (OR = 1.60, 95% CI = 1.08–2.37) and during entire pregnancy (OR = 1.49, 95% CI = 1.01–2.20) periods, exposure to PM₁₀ during the current period (OR = 1.78, 95% CI = 1.07–2.96), and exposure to NO₂ during the early-life (one year before conception and entire pregnancy) and current periods with ORs (95% CI) of 1.72 (1.10–2.71), 1.82 (1.17–2.83), and 1.94 (1.11–3.40), respectively. Further, we found significant associations of both prenatal and postnatal exposure to window condensation with childhood SAR to pollen, with ORs (95% CI) = 1.37 (1.05–1.77) and 1.38 (1.02–1.88), respectively. We encourage SAR to pollen sufferers to stay indoors due to outdoor air pollution and higher pollen concentration outdoors, but indoor ventilation should be maintained.

Keywords: seasonal allergic rhinitis; pollen allergy; air pollution; window condensation; indoor ventilation



Citation: Liu, Y.; Lu, C.; Li, Y.; Norbäck, D.; Deng, Q. Outdoor Air Pollution and Indoor Window Condensation Associated with Childhood Symptoms of Allergic Rhinitis to Pollen. *Int. J. Environ. Res. Public Health* **2022**, *19*, 8071. <https://doi.org/10.3390/ijerph19138071>

Academic Editor: Paola Palestini

Received: 20 May 2022

Accepted: 25 June 2022

Published: 30 June 2022

Publisher’s Note: MDPI stays neutral with regard to jurisdictional claims in published maps and institutional affiliations.



Copyright: © 2022 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (<https://creativecommons.org/licenses/by/4.0/>).

1. Introduction

As one of the most common chronic diseases [1], allergic rhinitis (AR) affects more than 40% of the world’s population [2], and its prevalence is rising rapidly in low- and middle-income countries [3]. AR in children receives considerable attention due to its high economic burden on families and substantial impairment of children’s quality of life and their learning and performance at school [4]. Plant pollen is considered a major cause of asthma and AR [5,6]. The rapid development of information and communication technologies and the urban economy have contributed to an accelerating urbanization

process in the last few decades [7]. Urban green spaces are shrinking due to accelerated urbanization [8]. However, symptoms of allergic rhinitis (SAR) to pollen and respiratory allergy symptoms are still increasing, and the prevalence of pollen-induced respiratory allergies in urban areas is higher than that in rural areas [9], which is mainly due to the role of outdoor air pollution [10,11] and indoor environmental factors.

Recent studies have suggested that exposure to air pollution may cause the development and/or symptom exacerbation of AR in children. Both epidemiological studies and toxicological experiments have consistently found that exposure to air pollution can contribute to AR [12–16]. An increasing number of studies have shown that air pollution modifies the effect of pollen on allergic respiratory symptoms and diseases [17,18]. Experimental studies have provided evidence that air pollution increases pollen allergen release and modifies the allergenic potential of pollen grains [11,19]. Many epidemiological studies have shown that the interaction of pollen and air pollutants increases the risk of allergic reactions [20–23]. However, there are very few studies that investigate the effect of exposure to early-life and current air pollution on childhood SAR to pollen, especially in different seasons.

Indoor air pollutant levels have increased due to rapid urbanization [24], and we spend more than 90 percent of our time indoors [25]. Many epidemiological studies have shown that indoor environmental factors are associated with AR in children and young adults, including indoor visible mold/damp stains [25–29] and window condensation [26,28]. There are studies that have found that moldy odor [30] and window condensation [31] can increase the risk of hay fever. However, the key indoor environmental risk factors for the development of SAR to pollen in children are unclear.

With the rapid urbanization and development of the economy during the past few decades, China has witnessed serious indoor and outdoor air pollution [32–34]. China's air quality has improved recently [35], but the impact of air pollution on health is irreversible. We aimed to explore whether exposure to indoor environmental factors (in the prenatal and postnatal periods) and outdoor air pollution (in the early-life and current periods) has increased the risk of SAR to pollen in children, especially the risk of SAR to pollen in different seasons.

2. Materials and Methods

2.1. Study Protocol and Questionnaire

A cross-sectional study was carried out in Changsha during the period from September 2011 to January 2012 as part of the nationwide "China-Children-Homes-Health (CCHH)" study [3]. The cross-sectional study protocol was approved by the Ethics Committee of Central South University and has been described in detail elsewhere [36]. In short, we used a standard questionnaire from the International Study of Asthma and Allergies in Childhood (ISAAC) [37] to obtain information about allergic diseases in children, and a Swedish questionnaire about dampness in buildings and health (DBH) [38] was used to survey the lifestyle and indoor environment of the children and their families. A total of 4988 questionnaires were distributed to children from 36 randomly selected kindergartens. We asked the parents to fill out the questionnaire and return them to the kindergarten within a week. A total of 3897 completed questionnaires were received, of which 2598 from children 3–6 years old were finally used in this work.

2.2. Exposure Windows

We divided the timing windows for indoor environmental factor exposure into prenatal and postnatal periods. The prenatal period was defined as the period from the first month to the last month of pregnancy. We divided timing windows for outdoor air pollution exposure into the early-life (one year before conception and prenatal) and current periods. The prenatal period can be divided into three gestational periods: months 1–3 (the first trimester), months 4–6 (the second trimester), and months 7–last (the third trimester), respectively. The current period was defined as the past 12 months or recent year(s).

2.3. Exposure to Indoor Environmental Factors

Both prenatal and postnatal exposure to indoor environmental factors included environmental tobacco smoke (ETS), new furniture, house redecoration, visible mold/damp stains at home, and window condensation. Detailed information on these factors is given in Table S1.

2.4. Exposure to Outdoor Air Pollution

Three types of air pollutants, sulfur dioxide (SO₂), nitrogen dioxide (NO₂), and particulate matter with a diameter $\leq 10 \mu\text{m}$ (PM₁₀), were selected as indicators of industry-related air pollution (IRAP), traffic-related air pollution (TRAP), and mixed air pollution, respectively [39]. The daily (24 h) average concentration of air pollutants was obtained from seven monitoring stations, and using these data, each child's daily exposure was estimated using the inverse distance weighted (IDW) method [39]. The prenatal exposure of children was calculated as the average monthly concentrations of PM₁₀, SO₂, and NO₂ during the early-life (one year before conception and prenatal) and current periods.

2.5. Health Outcome

The health outcome in this study was the symptoms of allergic rhinitis (SAR) to pollen, which was based on the answer to the question: "During past 12 months, whether your child had the rhinitis-like and eyes symptoms including sneezing, run-ning nose, stuffy nose, eyes' pain and tears after contacting with pollen or plants? If so, which month?" It was a modification of the original ISACC question for investigating SAR to pollen, especially in different seasons. The original question from ISAAC reads as follows: "In the past 12 months, has your child had a problem with sneezing, or a runny, or a blocked nose when he/she DID NOT have a cold or the flu?". SAR to pollen in different seasons was divided into spring (March to May), summer (June to August), autumn (September to November), and winter (December to February).

2.6. Confounding Covariates

Potential confounding variables were obtained from the questionnaire filled in by parents, which included personal factors (child's sex, age, birth season, breast-feeding, antibiotics use, and parental atopy) and indoor environmental factors (ETS at home, incense used, visible mold/damp stains, window condensation in winter, air humidifiers, cockroaches noted, and household pets) (Table S1) [25,26].

2.7. Statistical Analysis

The associations between childhood SAR to pollen and indoor environmental factors as well as outdoor air pollution were evaluated using multiple logistic regression models in terms of the odds ratio (OR) and 95% confidence interval (95% CI). The association between childhood SAR to pollen and exposure to indoor environmental factors was estimated with the classification model, and the OR was estimated in comparison with no exposure to indoor environmental factors. The relationship between childhood SAR to pollen and exposure to outdoor air pollutants was examined with continuous models. The OR (95% CI) in the continuous model was estimated with the interquartile range (IQR) increase in each pollutant exposure. We used restricted cubic spline regressions with four knots based on multiple logistic regression models for outdoor air pollution to model concentration–response curves. The association was considered statistically significant if the value of p was less than 0.05 ($p < 0.05$). All statistical analyses were performed using SPSS software (version 16.0, SPSS Inc., Chicago, IL, USA) and R software (version 4.0.5; RStudio, Boston, MA, USA).

3. Results

Of the 2598 children, 318 (12.2%) had symptoms of allergic rhinitis (SAR) to pollen in the previous 12 months. Table 1 shows the prevalence of SAR to pollen stratified by covariates. We found that children with parental atopy (21.1%) had significantly higher prevalence than those without parental atopy (11.2%) ($p < 0.05$). The prevalence of SAR to pollen was significantly higher among children who lived in a dwelling with mold/damp stains, window condensation, and incense used than among those living in a dwelling without mold/damp stains, window condensation, and incense used ($p < 0.05$). We further observed a higher prevalence of SAR to pollen in children with antibiotics used (12.9%) compared to children without antibiotics used (9.7%). Some families kept household pets that were found to have a significant effect on SAR to pollen. However, no difference in prevalence was observed among children stratified by the other covariates, including sex, age, birth season, breast-feeding, house size, ETS at home, house redecoration, and window condensation in winter.

Table 1. Demographic information and prevalence of SAR to pollen among children ($n = 2598$).

	<i>n</i>	Case	Prevalence (%)	<i>p</i> -Value
Total	2598	318	12.2	—
Sex				
Boys	1399	181	12.9	0.246
Girls	1199	137	11.4	
Age (years)				
3	665	82	12.3	0.898
4	952	114	12.0	
5	815	104	12.8	
6	166	18	10.8	
Birth season				
Warm (May–September)	1152	149	12.9	0.327
Cold (October–April)	1446	169	11.7	
Breast-feeding				
No	222	26	11.7	0.807
Yes	2376	292	12.3	
Antibiotics used				
No	432	42	9.7	0.059
Yes	2115	273	12.9	
Parental atopy				
No	2332	262	11.2	<0.001
Yes	266	56	21.1	
Environmental tobacco smoke (ETS) at home				
No	864	96	11.1	0.217
Yes	1734	222	12.8	
Incense used				
No	1845	204	11.1	0.005
Yes	705	107	15.2	
Visible mold/damp stains				
No	1985	227	11.4	0.027
Yes	606	90	14.9	
Window condensation in winter				
No	1175	116	9.9	<0.001
Yes	1369	201	14.7	
Air humidifier				
No	2095	263	12.6	0.316
Yes	277	41	14.8	
Cockroaches				
No	827	97	11.7	0.487
Yes	1653	210	12.7	
Household pets				
No	2300	292	12.7	0.061
Yes	294	26	8.8	

Sum of number is not 2598 due to the missing data. The p -values < 0.05 are in bold.

The associations between children with SAR to pollen and exposure to outdoor air pollution are shown in Table 2. Exposure to SO₂ in the first trimester of pregnancy was significantly associated with SAR to pollen in children, with an adjusted OR (95% CI) = 1.18 (1.02–1.37).

Table 2. Odds ratio (95% CI) of childhood annual SAR to pollen for exposure to outdoor air pollution during 1 year before conception, prenatal, and current periods (*n* = 2598).

	Crude OR	Adjusted OR #
1 year before conception		
PM ₁₀	0.97 (0.82–1.16)	1.01 (0.72–1.42)
SO ₂	1.02 (0.85–1.22)	1.11 (0.91–1.36)
NO ₂	1.02 (0.84–1.23)	1.10 (0.88–1.39)
Prenatal		
1st trimester		
PM ₁₀	1.08 (0.93–1.24)	1.09 (0.89–1.33)
SO ₂	1.11 (0.98–1.26)	1.18 (1.02–1.37) *
NO ₂	1.07 (0.90–1.27)	1.09 (0.87–1.37)
2nd trimester		
PM ₁₀	0.92 (0.80–1.06)	0.91 (0.77–1.07)
SO ₂	1.05 (0.94–1.18)	1.08 (0.95–1.24)
NO ₂	0.98 (0.84–1.16)	0.97 (0.79–1.20)
3rd trimester		
PM ₁₀	0.92 (0.81–1.05)	0.89 (0.74–1.06)
SO ₂	0.93 (0.81–1.07)	0.99 (0.83–1.17)
NO ₂	0.94 (0.79–1.11)	0.98 (0.80–1.20)
Entire pregnancy		
PM ₁₀	0.94 (0.83–1.08)	0.89 (0.72–1.09)
SO ₂	1.05 (0.89–1.23)	1.16 (0.95–1.41)
NO ₂	0.99 (0.83–1.19)	1.02 (0.82–1.28)
Current		
PM ₁₀	0.94 (0.83–1.08)	1.16 (0.90–1.51)
SO ₂	1.05 (0.89–1.23)	1.24 (0.99–1.56)
NO ₂	0.99 (0.83–1.19)	1.07 (0.81–1.41)

OR (95% CI) was estimated for an IQR increase in PM₁₀, SO₂, and NO₂. # Models were adjusted for all the covariates in Table 1. * *p* ≤ 0.05.

Table 3 provides the association between exposure to outdoor air pollution during the early-life and current periods and children with SAR to pollen in different seasons. We found a significant association between current exposure to PM₁₀ and children with autumn SAR to pollen, with an OR (95% CI) = 1.78 (1.07–2.96). Early-life exposure to SO₂ was significantly associated with autumn SAR to pollen in children, with an OR (95% CI) = 1.60 (1.08–2.37) during one year before conception, an OR (95% CI) = 1.42 (1.02–1.96) during the first trimester, an OR (95% CI) = 1.35 (1.03–1.77) during the second trimester, and an adjusted OR (95% CI) = 1.49 (1.01–2.20) during the entire pregnancy period. We further found that both early-life and current exposure to NO₂ were significantly associated with childhood autumn SAR to pollen, with ORs (95% CI) = 1.72 (1.10–2.71), 1.60 (1.02–2.49), 1.90 (1.22–2.96), 1.82 (1.17–2.83), and 1.94 (1.11–3.40) during one year before conception, the second and third trimester, the entire pregnancy, and current period, respectively. However, the third trimester and entire pregnancy exposure to PM₁₀ were significantly associated with winter SAR to pollen in children, with ORs (95% CI) = 0.45 (0.25–0.82) and 0.48 (0.26–0.90), respectively. Associations between pregnancy exposure to outdoor air pollution and childhood SAR to pollen in the multi-pollutant model and multi-pollutant/window model were the same as that in the single-adjusted model (Tables S3 and S4).

Table 3. Odds ratio (95% CI) of childhood seasonal SAR to pollen for exposure to outdoor air pollution during 1 year before conception, prenatal, and current periods (*n* = 2598).

	SAR to Pollen			
	Spring	Summer	Autumn	Winter
1 year before conception				
PM ₁₀	1.32 (0.86–2.03)	0.88 (0.43–1.81)	1.06 (0.55–2.06)	0.67 (0.27–1.65)
SO ₂	0.98 (0.76–1.26)	0.93 (0.61–1.43)	1.60 (1.08–2.37) **	1.16 (0.65–2.07)
NO ₂	0.91 (0.68–1.21)	1.10 (0.68–1.77)	1.72 (1.10, 2.71) **	1.28 (0.66–2.45)
Prenatal				
1st trimester				
PM ₁₀	1.02 (0.78–1.33)	1.33 (0.87–2.05)	1.05 (0.70–1.58)	0.98 (0.56–1.72)
SO ₂	1.06 (0.85–1.33)	1.22 (0.86–1.73)	1.42 (1.02–1.96) **	1.57 (0.97–2.54)
NO ₂	0.85 (0.62–1.17)	1.01 (0.60–1.70)	1.56 (0.96, 2.54)	1.22 (0.58–2.55)
2nd trimester				
PM ₁₀	0.93 (0.73–1.19)	0.91 (0.61–1.37)	0.98 (0.67–1.44)	0.82 (0.48–1.42)
SO ₂	1.13 (0.93–1.38)	1.05 (0.77–1.45)	1.35 (1.03–1.77) **	1.30 (0.87–1.92)
NO ₂	0.86 (0.64–1.15)	0.81 (0.49–1.34)	1.60 (1.02–2.49) **	1.20 (0.62–2.33)
3rd trimester				
PM ₁₀	0.89 (0.70–1.13)	0.93 (0.63–1.39)	0.83 (0.57–1.22)	0.45 (0.25–0.82) **
SO ₂	1.07 (0.83–1.38)	0.78 (0.49–1.23)	1.17 (0.81–1.69)	0.96 (0.58–1.60)
NO ₂	0.90 (0.67–1.21)	0.74 (0.45–1.21)	1.90 (1.22–2.96) **	0.88 (0.46–1.69)
Entire pregnancy				
PM ₁₀	0.87 (0.66–1.15)	1.04 (0.65–1.65)	0.91 (0.59–1.40)	0.48 (0.26–0.90) **
SO ₂	1.13 (0.87–1.47)	1.06 (0.69–1.62)	1.49 (1.01–2.20) **	1.33 (0.76–2.33)
NO ₂	0.86 (0.64–1.14)	0.87 (0.54–1.41)	1.82 (1.17–2.83) **	1.02 (0.53–1.95)
Current				
PM ₁₀	0.99 (0.71–1.37)	1.14 (0.66–1.96)	1.78 (1.07–2.96) **	1.11 (0.53–2.33)
SO ₂	1.13 (0.86–1.51)	1.37 (0.85–2.22)	1.49 (0.94–2.37)	0.96 (0.51–1.82)
NO ₂	0.86 (0.60–1.23)	1.06 (0.59–1.92)	1.94 (1.11–3.40) **	1.21 (0.54–2.72)

OR (95% CI) was estimated for an IQR increase in PM₁₀, SO₂, and NO₂. Models were adjusted for all the covariates in Table 1. ** *p* ≤ 0.01.

We assessed the continuous association between exposure to SO₂ in the first trimester and the risk of childhood SAR to pollen using a restricted cubic spline curve (Figure 1). A relatively linear relationship was observed between the risk of childhood SAR to pollen and first trimester SO₂ exposure. Figure 2 illustrates concentration–response curves between autumn SAR to pollen in children and SO₂ exposure during the early-life period, PM₁₀ exposure during the current period, and NO₂ exposure during both the early-life and current periods. All figures showed linear increases in risks associated with in PM₁₀, SO₂, and NO₂ exposure during the early-life and current periods.

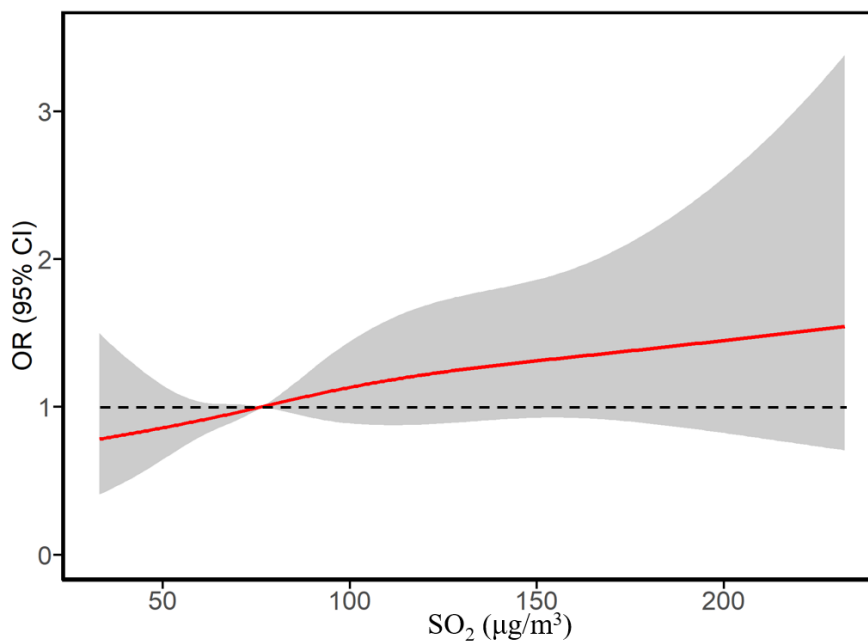


Figure 1. Concentration–response curves between exposure to SO₂ in the 1st trimester and risks of childhood annual SAR to pollen. OR (95% CI) was adjusted for all the covariates in Table 1.

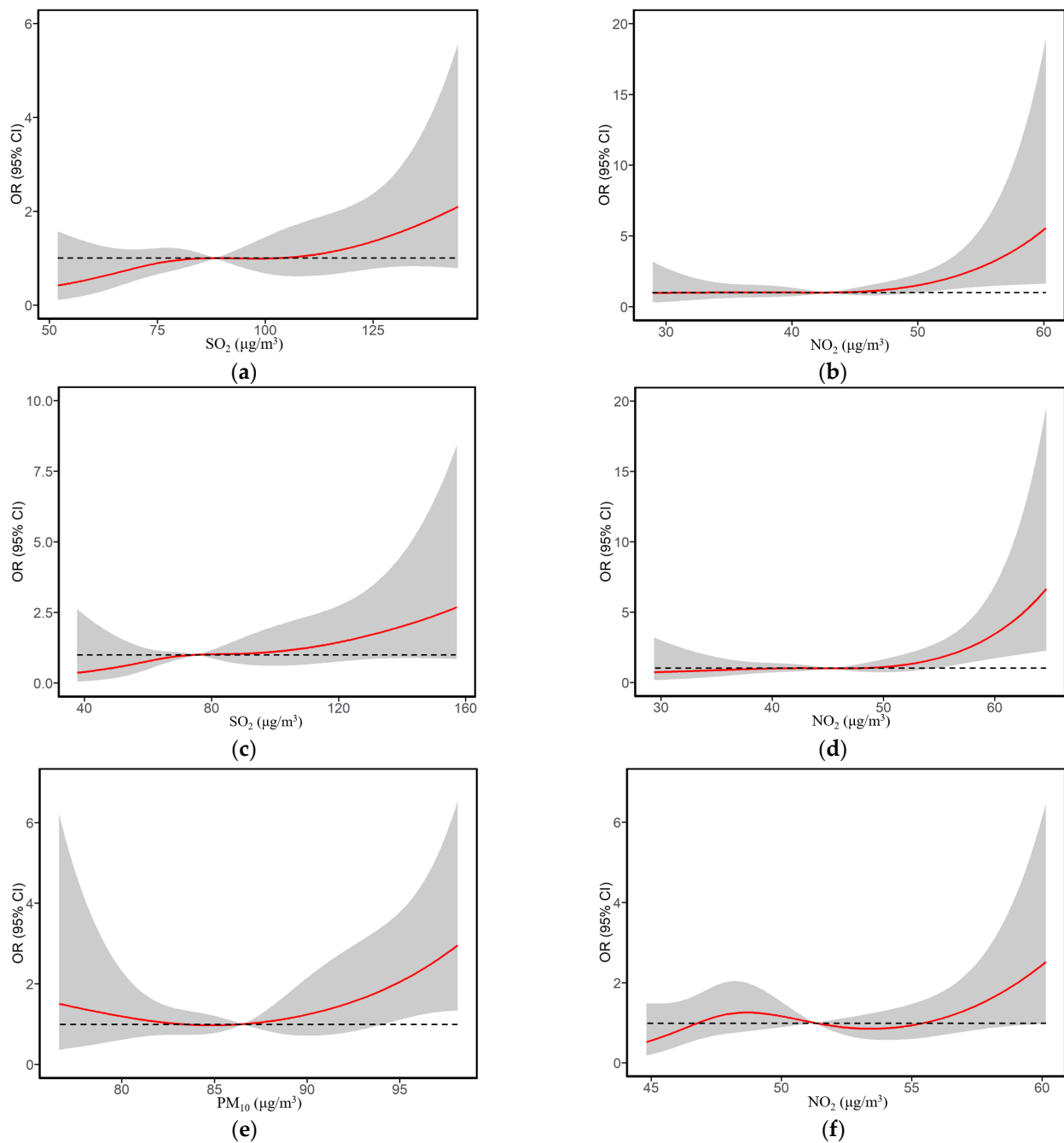


Figure 2. Concentration–response curves between exposure to PM_{10} , SO_2 , and NO_2 during time windows and risks of childhood autumn SAR to pollen. OR (95% CI) was adjusted for all the covariates in Table 1. (a) One year before conception SO_2 . (b) One year before conception NO_2 . (c) Entire pregnancy SO_2 . (d) Entire pregnancy NO_2 . (e) Current PM_{10} . (f) Current NO_2 .

We found that prenatal and postnatal exposure to window condensation were significantly associated with childhood SAR to pollen, with adjusted ORs (95% CI) = 1.37 (1.05–1.77) and 1.38 (1.02–1.88) (Table 4), respectively. We further found a significant association between exposure to visible mold/damp stains during prenatal period and SAR to pollen in children, with a crude OR (95% CI) = 1.41 (1.01–1.96).

Table 4. Odds ratio (95% CI) of childhood annual SAR to pollen for exposure to indoor environmental factors during prenatal and postnatal periods ($n = 2598$).

	<i>n</i>	Case	(%)	Crude OR	Adjusted OR #
Prenatal					
ETS at home	1252	157	(12.5)	1.04 (0.83–1.32)	1.10 (0.85–1.42)
New furniture	338	46	(13.6)	1.17 (0.83–1.65)	1.07 (0.73–1.56)
House redecoration	139	18	(12.9)	1.07 (0.64–1.80)	1.01 (0.58–1.77)
Visible mold/damp stains	435	63	(14.5)	1.26 (0.93–1.69)	1.19 (0.85–1.65)
Window condensation	1222	181	(14.8)	1.54 (1.21–1.96) ***	1.37 (1.05–1.77) *
Postnatal					
ETS at home	1734	222	(12.8)	1.17 (0.91–1.52)	1.32 (0.99–1.74)
New furniture	980	136	(13.9)	1.26 (0.99–1.60)	1.22 (0.94–1.59)
House redecoration	499	67	(13.4)	1.14 (0.85–1.52)	1.13 (0.82–1.55)
Visible mold/damp stains	308	49	(15.9)	1.41 (1.01–1.96) *	1.23 (0.85–1.79)
Window condensation	869	139	(16.0)	1.52 (1.15–2.02) **	1.38 (1.02–1.88) *

Models were adjusted for all the covariates in Table 1. * $p \leq 0.05$. ** $p \leq 0.01$. *** $p \leq 0.001$.

4. Discussion

This is the first cross-sectional study to examine the risk of prenatal exposure to outdoor air pollution and indoor environmental factors on the symptoms of allergic rhinitis (SAR) to pollen in children. We found that early-life and current exposure to outdoor air pollution (PM₁₀, SO₂, and NO₂) were significantly associated with childhood SAR to pollen in autumn. We further found a significant association of exposure to window condensation during prenatal and postnatal periods with SAR to pollen in children. Studies have reported that there is less condensation on the windows of rooms with a higher air change rate [40,41]. Therefore, we suggested that ventilation indoors may help reduce the risk of childhood SAR to pollen.

We found that exposure to SO₂ during the third trimester of pregnancy was significantly associated with SAR to pollen in children, and in particular, we found a significant relationship between early-life and current exposure to outdoor air pollution (PM₁₀, SO₂ and NO₂) and childhood SAR to pollen in autumn. There is increasing evidence of a causal relationship between outdoor air pollution and the risk of pollen-related allergic diseases, especially in children. A study found that exposure to both PM_{2.5} and NO₂ in highway tunnels may significantly enhance asthmatic reactions to inhaled pollen allergens [23]. A prospective birth cohort study reported that exposure to PM_{2.5} significantly increased the risk of hay fever (OR, 1.59; 95% CI, 1.11–2.27) and allergic sensitization to pollen (OR, 1.40; 95% CI, 1.20–1.64) in children [21]. A European birth cohort study observed that sensitization to inhaled pollen allergens may be related to PM_{2.5} and NO₂ [20]. Many studies have shown that the interaction between air pollutants and pollens might promote the release of pollen allergens, modify the potential of allergens, and enhance the expression of some allergens in pollen grains [11]. Moreover, studies have found that autumn is one of the two peaks of AR [42–44]. Symptomatic AR in autumn may be caused by weed pollen allergens [45] and the increasing prevalence of weed pollen allergy in children [46]. One study observed a significant positive association between air pollutants (SO₂), pollens, and the number of outpatients for AR during autumn (43.00967 ± 0.11284 , $p < 0.001$) [47]. The different association of exposure to PM₁₀ during the third trimester and the entire pregnancy with winter SAR to pollen in children may be because of the low case numbers of childhood SAR to pollen in winter.

We observed a significant association between exposure to window condensation during the prenatal and postnatal periods and childhood SAR to pollen. Although we have not found identical studies to ours, a large number of studies support our findings. Studies have observed that window condensation may increase the risk of childhood SAR [26,28]. One study indicated that window condensation may increase the risk of airway infection

during the past 12 months (OR = 1.66; 95% CI 1.17–2.37) and allergy to pollen (OR = 1.54; 95% CI 1.15–2.06) [31]. Window condensation is a proxy for less ventilation indoors [40,41]. Thus, our findings showed that indoor ventilation might be associated with SAR to pollen in children.

When staying outdoors, high levels of pollen and outdoor air pollution exposure might increase the risk of SAR to pollen. However, staying indoors without ventilation may also be a risk factor. The possible reason is that high pollen levels may increase the risk of SAR to pollen, while appropriate ventilation indoors might enrich indoor air microbiomes that reduce pollen allergy response. It is known to all that the allergic symptoms due to pollen become more frequent and serious with an increase in pollen counts that exceed a certain threshold [48–50]. Studies have shown that ventilation is the key factor in controlling indoor microbiomes [51] and that outdoor air is one of the main contributors to indoor airborne microbiomes [52]. The biodiversity hypothesis states that the abundance of human microbiota promotes immune balance and protects from allergic diseases [53,54].

There are some limitations that should be acknowledged in this study. On the one hand, SAR to pollen was not assessed by skin prick testing or measuring serum IgE. Therefore, some symptomatic responses to AR due to pollen in children may have been missed. On the other hand, we did not distinguish between specific types of pollen in our questionnaire, and thus we could not analyze which factors play key roles in SAR due to a specific type of pollen. We will consider the above limitations in a future study on SAR to pollen in children.

5. Conclusions

Our study found that exposure to outdoor air pollution during the early-life and current periods was significantly associated with the prevalence of childhood SAR to pollen in autumn. Meanwhile, prenatal and postnatal exposure to window condensation significantly increased the risk of SAR to pollen in children. Due to global climate change and air pollution, the harm of pollen sensitization has been aggravated. Meanwhile, outdoor air pollution in developing countries is still serious. We provide a measure to reduce the risk of SAR to pollen during pollen season when outdoor air pollution is high, which is to stay indoors and maintain good ventilation.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/ijerph19138071/s1>, Table S1. Descriptions and questions about confounding variables related to childhood symptoms of allergic rhinitis (SAR) to pollen in this study. Table S2. Descriptive statistics for air pollution levels attributed to the children during 1 year before conception, prenatal and current periods ($n = 2598$). Table S3. Odds ratio (95%CI) of childhood SAR to pollen for exposure to outdoor air pollution during different trimesters of pregnancy ($n = 2598$). Table S4. Odds ratio (95%CI) of childhood autumn SAR to pollen for exposure to outdoor air pollution during different trimesters of pregnancy ($n = 2598$). Table S5. Pearson correlations between outdoor air pollutants during different time windows ($n = 2598$).

Author Contributions: Conceptualization, Y.L. (Yuguo Li) and Q.D.; methodology, Y.L. (Yingjie Liu) and C.L.; software, Y.L. (Yingjie Liu); validation, Y.L. (Yingjie Liu); formal analysis, Y.L. (Yingjie Liu); investigation, Y.L. (Yingjie Liu), C.L. and D.N.; resources, Y.L. (Yuguo Li) and Q.D.; data curation, Y.L. (Yingjie Liu) and C.L.; writing—original draft preparation, Y.L. (Yingjie Liu); writing—review and editing, Y.L. (Yingjie Liu), C.L. and Q.D.; supervision, Y.L. (Yuguo Li) and Q.D.; project administration, D.N.; funding acquisition, Q.D. All authors have read and agreed to the published version of the manuscript.

Funding: This research was supported by the National Natural Science Foundation of China (No. 42007391 and 41977369) and the Natural Science Foundation of Hunan Province (Grant No. 2021JJ30813).

Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of the School of Public Health, Fudan University (protocol code #IRB-2013-01-0390 and date of approval 23 January 2013).

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 on request from the corresponding author.

Acknowledgments: We are particularly indebted to the children, their parents, and the kindergartens for their time and enthusiastic participation.

Conflicts of Interest: The authors declare no conflict of interest.

References

1. Li, R.-L.; Ho, Y.-C.; Luo, C.-W.; Lee, S.-S.; Kuan, Y.-H. Influence of PM_{2.5} Exposure Level on the Association between Alzheimer's Disease and Allergic Rhinitis: A National Population-Based Cohort Study. *Int. J. Environ. Res. Public Health* **2019**, *16*, 3357. [[CrossRef](#)]
2. Izquierdo-Domínguez, A.; Valero, A.L.; Mullol, J. Comparative analysis of allergic rhinitis in children and adults. *Curr. Allergy Asthma Rep.* **2013**, *13*, 142–151. [[CrossRef](#)] [[PubMed](#)]
3. Zhang, Y.; Li, B.; Huang, C.; Yang, X.; Qian, H.; Deng, Q.; Zhao, Z.; Li, A.; Zhao, J.; Zhang, X.; et al. Ten cities cross-sectional questionnaire survey of children asthma and other allergies in China. *Chin. Sci. Bull.* **2013**, *58*, 4182–4189. [[CrossRef](#)]
4. Greiner, A.N.; Hellings, P.W.; Rotiroli, G.; Scadding, G.K. Allergic rhinitis. *Lancet* **2011**, *378*, 2112–2122. [[CrossRef](#)]
5. Yin, S.; Shen, Z.; Zhou, P.; Zou, X.; Che, S.; Wang, W. Quantifying air pollution attenuation within urban parks: An experimental approach in Shanghai, China. *Environ. Pollut.* **2011**, *159*, 2155–2163. [[CrossRef](#)] [[PubMed](#)]
6. Saif, N.T.; Kleiner, G.I.; Forster, L.Q.; Hershorin, E.R.; Colin, A.A.; Mirsaiedi, M.; Kumar, N. Allergies, Allergic Comorbidities and the Home Environment in Pediatric Asthma in Southern Florida. *Int. J. Environ. Res. Public Health* **2021**, *18*, 4142. [[CrossRef](#)]
7. Sun, L.; Chen, J.; Li, Q.; Huang, D. Dramatic uneven urbanization of large cities throughout the world in recent decades. *Nat. Commun.* **2020**, *11*, 5366. [[CrossRef](#)] [[PubMed](#)]
8. Basu, S.; Nagendra, H. Perceptions of park visitors on access to urban parks and benefits of green spaces. *Urban For. Urban Green.* **2021**, *57*, 126959. [[CrossRef](#)]
9. D'Amato, G.; Cecchi, L.; D'Amato, M.; Liccardi, G. Urban Air Pollution and Climate Change as Environmental Risk Factors of Respiratory Allergy: An Update. *J. Investig. Allergol. Clin. Immunol.* **2010**, *20*, 95–102.
10. Gisler, A. Allergies in Urban Areas on the Rise: The Combined Effect of Air Pollution and Pollen. *Int. J. Public Health* **2021**, *66*, 1604022. [[CrossRef](#)]
11. Sedghy, F.; Varasteh, A.-R.; Sankian, M.; Moghadam, M. Interaction between Air Pollutants and Pollen Grains: The Role on the Rising Trend in Allergy. *Rep. Biochem. Mol. Biol.* **2018**, *6*, 219–224. [[PubMed](#)]
12. Lin, L.S.; Li, T.Y.; Sun, M.Q.; Liang, Q.Q.; Ma, Y.X.; Wang, F.H.; Duan, J.C.; Sun, Z.W. Effect of particulate matter exposure on the prevalence of allergic rhinitis in children: A systematic review and meta-analysis. *Chemosphere* **2021**, *268*, 128841. [[CrossRef](#)] [[PubMed](#)]
13. Lu, C.; Deng, Q.H.; Ou, C.Y.; Liu, W.W.; Sundell, J. Effects of ambient air pollution on allergic rhinitis among preschool children in Changsha, China. *Chin. Sci. Bull.* **2013**, *58*, 4252–4258. [[CrossRef](#)]
14. Saxon, A.; Diaz-Sanchez, D. Air pollution and allergy: You are what you breathe. *Nat. Immunol.* **2005**, *6*, 223–226. [[CrossRef](#)] [[PubMed](#)]
15. Zou, Q.Y.; Shen, Y.; Ke, X.; Hong, S.L.; Kang, H.Y. Exposure to air pollution and risk of prevalence of childhood allergic rhinitis: A meta-analysis. *Int. J. Pediatric Otorhinolaryngol.* **2018**, *112*, 82–90. [[CrossRef](#)]
16. Mariani, J.; Iodice, S.; Cantone, L.; Solazzo, G.; Marraccini, P.; Conforti, E.; Bulsara, P.A.; Lombardi, M.S.; Howlin, R.P.; Bollati, V.; et al. Particulate Matter Exposure and Allergic Rhinitis: The Role of Plasmatic Extracellular Vesicles and Bacterial Nasal Microbiome. *Int. J. Environ. Res. Public Health* **2021**, *18*, 10689. [[CrossRef](#)]
17. Fuertes, E.; Markevych, I.; Bowatte, G.; Gruzieva, O.; Gehring, U.; Becker, A.; Berdel, D.; von Berg, A.; Bergstrom, A.; Brauer, M.; et al. Residential greenness is differentially associated with childhood allergic rhinitis and aeroallergen sensitization in seven birth cohorts. *Allergy* **2016**, *71*, 1461–1471. [[CrossRef](#)]
18. Markevych, I.; Ludwig, R.; Baumbach, C.; Standl, M.; Heinrich, J.; Herberth, G.; de Hoogh, K.; Pritsch, K.; Weikl, F. Residing near allergenic trees can increase risk of allergies later in life: LISA Leipzig study. *Environ. Res.* **2020**, *191*, 110132. [[CrossRef](#)]
19. Senechal, H.; Visez, N.; Charpin, D.; Shahali, Y.; Peltre, G.; Biolley, J.-P.; Lhuissier, F.; Couderc, R.; Yamada, O.; Malrat-Domenge, A.; et al. A Review of the Effects of Major Atmospheric Pollutants on Pollen Grains, Pollen Content, and Allergenicity. *Sci. World J.* **2015**, *2015*, 940243. [[CrossRef](#)]
20. Melen, E.; Standl, M.; Gehring, U.; Altug, H.; Anto, J.M.; Berdel, D.; Bergstrom, A.; Bousquet, J.; Heinrich, J.; Koppelman, G.H.; et al. Air pollution and IgE sensitization in 4 European birth cohorts—the MeDALL project. *J. Allergy Clin. Immunol.* **2021**, *147*, 713–722. [[CrossRef](#)]
21. Morgenstern, V.; Zutavern, A.; Cyrys, J.; Brockow, I.; Koletzko, S.; Kramer, U.; Behrendt, H.; Herbarth, O.; von Berg, A.; Bauer, C.P.; et al. Atopic diseases, allergic sensitization, and exposure to traffic-related air pollution in children. *Am. J. Respir. Crit. Care Med.* **2008**, *177*, 1331–1337. [[CrossRef](#)] [[PubMed](#)]

22. Parker, J.D.; Akinbami, L.J.; Woodruff, T.J. Air Pollution and Childhood Respiratory Allergies in the United States. *Environ. Health Perspect.* **2009**, *117*, 140–147. [[CrossRef](#)] [[PubMed](#)]
23. Svartengren, M.; Strand, V.; Bylin, G.; Jarup, L.; Pershagen, G. Short-term exposure to air pollution in a road tunnel enhances the asthmatic response to allergen. *Eur. Respir. J.* **2000**, *15*, 716–724. [[CrossRef](#)]
24. Babu, P.; Suthar, G. *Indoor Air Quality and Thermal Comfort in Green Building: A Study for Measurement, Problem and Solution Strategies*; Indoor Environmental Quality; Sharma, A., Goyal, R., Mittal, R., Eds.; Springer: Singapore, 2020; pp. 139–146.
25. Norback, D.; Lu, C.; Wang, J.; Zhang, Y.P.; Li, B.Z.; Zhao, Z.H.; Huang, C.; Zhang, X.; Qian, H.; Sun, Y.X.; et al. Asthma and rhinitis among Chinese children—Indoor and outdoor air pollution and indicators of socioeconomic status (SES). *Environ. Int.* **2018**, *115*, 1–8. [[CrossRef](#)] [[PubMed](#)]
26. Deng, Q.; Lu, C.; Ou, C.; Chen, L.; Yuan, H. Preconceptional, prenatal and postnatal exposure to outdoor and indoor environmental factors on allergic diseases/symptoms in preschool children. *Chemosphere* **2016**, *152*, 459–467. [[CrossRef](#)]
27. Tischer, C.; Chen, C.M.; Heinrich, J. Association between domestic mould and mould components, and asthma and allergy in children: A systematic review. *Eur. Respir. J.* **2011**, *38*, 812–824. [[CrossRef](#)] [[PubMed](#)]
28. Wang, J.; Zhao, Z.; Zhang, Y.; Li, B.; Huang, C.; Zhang, X.; Deng, Q.; Lu, C.; Qian, H.; Yang, X.; et al. Asthma, allergic rhinitis and eczema among parents of preschool children in relation to climate, and dampness and mold in dwellings in China. *Environ. Int.* **2019**, *130*, 104910. [[CrossRef](#)]
29. Ellie, A.S.; Sun, Y.; Hou, J.; Wang, P.; Zhang, Q.; Sundell, J. Prevalence of Childhood Asthma and Allergies and Their Associations with Perinatal Exposure to Home Environmental Factors: A Cross-Sectional Study in Tianjin, China. *Int. J. Environ. Res. Public Health* **2021**, *18*, 4131. [[CrossRef](#)]
30. Norbäck, D.; Lampa, E.; Engvall, K. Asthma, allergy and eczema among adults in multifamily houses in Stockholm (3-HE study)—Associations with building characteristics, home environment and energy use for heating. *PLoS ONE* **2014**, *9*, e112960. [[CrossRef](#)]
31. Takaoka, M.; Suzuki, K.; Norbäck, D. The home environment of junior high school students in Hyogo, Japan—associations with asthma, respiratory health and reported allergies. *Indoor Built Environ.* **2016**, *25*, 81–92. [[CrossRef](#)]
32. Hu, M.M.; Wang, Y.F.; Wang, S.; Jiao, M.Y.; Huang, G.H.; Xia, B.C. Spatial-temporal heterogeneity of air pollution and its relationship with meteorological factors in the Pearl River Delta, China. *Atmos. Environ.* **2021**, *254*, 118415. [[CrossRef](#)]
33. Tao, M.H.; Gui, L.; Li, R.; Wang, L.L.; Liang, S.W.; Li, Q.J.; Wang, L.C.; Yu, C.; Chen, L.F. Tracking prevailing dust aerosol over the air pollution in central China with integrated satellite and ground observations. *Atmos. Environ.* **2021**, *253*, 118369. [[CrossRef](#)]
34. Lopes, D.; Ferreira, J.; Hoi, K.I.; Yuen, K.-V.; Mok, K.M.; Miranda, A.I. Emission Inventories and Particulate Matter Air Quality Modeling over the Pearl River Delta Region. *Int. J. Environ. Res. Public Health* **2021**, *18*, 4155. [[CrossRef](#)] [[PubMed](#)]
35. Zeng, Y.Y.; Cao, Y.F.; Qiao, X.; Seyler, B.C.; Tang, Y. Air pollution reduction in China: Recent success but great challenge for the future. *Sci. Total Environ.* **2019**, *663*, 329–337. [[CrossRef](#)]
36. Deng, Q.; Lu, C.; Li, Y.; Sundell, J.; Norbäck, D. Exposure to outdoor air pollution during trimesters of pregnancy and childhood asthma, allergic rhinitis, and eczema. *Environ. Res.* **2016**, *150*, 119–127. [[CrossRef](#)]
37. Asher, M.I.; Montefort, S.; Björkstén, B.; Lai, C.K.; Strachan, D.P.; Weiland, S.K.; Williams, H.; the ISAAC Phase Three Study Group. Worldwide time trends in the prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and eczema in childhood: ISAAC Phases One and Three repeat multicountry cross-sectional surveys. *Lancet* **2006**, *368*, 733–743. [[CrossRef](#)]
38. Bornehag, C.-G.; Sundell, J.; Sigsgaard, T. Dampness in buildings and health (DBH): Report from an ongoing epidemiological investigation on the association between indoor environmental factors and health effects among children in Sweden. *Indoor Air* **2004**, *14*, 59–66. [[CrossRef](#)]
39. Deng, Q.; Lu, C.; Norbäck, D.; Bornehag, C.-G.; Zhang, Y.; Liu, W.; Yuan, H.; Sundell, J. Early life exposure to ambient air pollution and childhood asthma in China. *Environ. Res.* **2015**, *143*, 83–92. [[CrossRef](#)]
40. Bekö, G.; Toftum, J.; Clausen, G. Modeling ventilation rates in bedrooms based on building characteristics and occupant behavior. *Build. Environ.* **2011**, *46*, 2230–2237. [[CrossRef](#)]
41. Sun, Y.; Hou, J.; Sheng, Y.; Kong, X.; Weschler, L.B.; Sundell, J. Modern life makes children allergic. A cross-sectional study: Associations of home environment and lifestyles with asthma and allergy among children in Tianjin region, China. *Int. Arch. Occup. Environ. Health* **2019**, *92*, 587–598. [[CrossRef](#)]
42. Pfaar, O.; Karatzas, K.; Bastl, K.; Berger, U.; Buters, J.; Darsow, U.; Demoly, P.; Durham, S.R.; Galan, C.; Gehrig, R.; et al. Pollen season is reflected on symptom load for grass and birch pollen-induced allergic rhinitis in different geographic areas—An EAACI Task Force Report. *Allergy* **2020**, *75*, 1099–1106. [[CrossRef](#)] [[PubMed](#)]
43. Wang, J.; Li, B.Z.; Yu, W.; Yang, Q.; Wang, H.; Huang, D.C.; Sundell, J.; Norback, D. Rhinitis Symptoms and Asthma among Parents of Preschool Children in Relation to the Home Environment in Chongqing, China. *PLoS ONE* **2014**, *9*, e94731. [[CrossRef](#)] [[PubMed](#)]
44. Zhang, Y.; Zhang, L. Prevalence of Allergic Rhinitis in China. *Allergy Asthma Immunol. Res.* **2014**, *6*, 105–113. [[CrossRef](#)] [[PubMed](#)]
45. Forkel, S.; Beutner, C.; Heetfeld, A.; Fuchs, T.; Schoen, M.P.; Geier, J.; Buhl, T. Allergic Rhinitis to Weed Pollen in Germany: Dominance by Plantain, Rising Prevalence, and Polysensitization Rates over 20 Years. *Int. Arch. Allergy Immunol.* **2020**, *181*, 128–135. [[CrossRef](#)]

46. Kim, J.-H.; Oh, J.-W.; Lee, H.-B.; Kim, S.-W.; Kang, I.-J.; Kook, M.-H.; Kim, B.-S.; Park, K.-S.; Baek, H.-S.; Kim, K.-R.; et al. Changes in Sensitization Rate to Weed Allergens in Children with Increased Weeds Pollen Counts in Seoul Metropolitan Area. *J. Korean Med. Sci.* **2012**, *27*, 350–355. [[CrossRef](#)]
47. Kim, H.; Park, Y.; Park, K.; Yoo, B. Association between Pollen Risk Indexes, Air Pollutants, and Allergic Diseases in Korea. *Osong Public Health Res. Perspect.* **2016**, *7*, 172–179. [[CrossRef](#)]
48. Burr, M.L.; Emberlin, J.C.; Treu, R.; Cheng, S.; Pearce, N.E.; the ISAAC Phase One Study Group. Pollen counts in relation to the prevalence of allergic rhinoconjunctivitis, asthma and atopic eczema in the International Study of Asthma and Allergies in Childhood (ISAAC). *Clin. Exp. Allergy* **2003**, *33*, 1675–1680. [[CrossRef](#)]
49. D’Amato, G.; Cecchi, L.; Bonini, S.; Nunes, C.; Annesi-Maesano, I.; Behrendt, H.; Liccardi, G.; Popov, T.; van Cauwenberge, P. Allergenic pollen and pollen allergy in Europe. *Allergy* **2007**, *62*, 976–990. [[CrossRef](#)]
50. de Weger, L.A.; Bergmann, K.C.; Rantio-Lehtimäki, A.; Dahl, Å.; Buters, J.; Déchamp, C.; Belmonte, J.; Thibaudon, M.; Cecchi, L.; Besancenot, J.-P.; et al. Impact of Pollen. In *Allergenic Pollen: A Review of the Production, Release, Distribution and Health Impacts*; Sofiev, M., Bergmann, K.-C., Eds.; Springer: Dordrecht, The Netherlands, 2013; pp. 161–215.
51. Peccia, J.; Kwan, S.E. Buildings, Beneficial Microbes, and Health. *Trends Microbiol.* **2016**, *24*, 595–597. [[CrossRef](#)]
52. Nazaroff, W.W. Indoor bioaerosol dynamics. *Indoor Air* **2016**, *26*, 61–78. [[CrossRef](#)]
53. Bloomfield, S.F.; Rook, G.A.W.; Scott, E.A.; Shanahan, F.; Stanwell-Smith, R.; Turner, P. Time to abandon the hygiene hypothesis: New perspectives on allergic disease, the human microbiome, infectious disease prevention and the role of targeted hygiene. *Perspect. Public Health* **2016**, *136*, 213–224. [[CrossRef](#)]
54. Hahtela, T. A biodiversity hypothesis. *Allergy* **2019**, *74*, 1445–1456. [[CrossRef](#)]