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ORIGINAL RESEARCH

Parental Smoking and Risk of Allergic Conjunctivitis in Chinese Pediatric Population: A Cross-Sectional Study

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Purpose: Evidence on the link between parental smoking and allergic conjunctivitis (AC) is limited, particularly in Chinese children. This study aimed to examine the association between parental smoking and the risk of AC in children.

Patients and Methods: The cross-sectional study was conducted in the ophthalmology department at Tianjin Children's Hospital from 2021 to 2022. We used logistic regression to explore the association between parental smoking and AC. The stability of the results was ensured using subgroup analysis and propensity score matching (PSM).

Results: A total of 4249 participants met the inclusion criteria and were analyzed. After adjusting for all covariates, parental smoking was significantly associated with AC. The adjusted odds ratio was 1.17 (1.03–1.34). Significant interactions were observed for mode of delivery and multiple pregnancies, in relation to the prediction of AC ($P < 0.05$). Further exploratory subgroup analyses in children with myopia, hyperopia, and astigmatism revealed no significant interactions (all P values for interaction were > 0.05). After adjusting for potential confounders using PSM, the results remained stable.

Conclusion: This cross-sectional study showed that influence of inappropriate parental smoking on the risk of incident AC. Parental smoking was associated with increased risk of AC in children. Reducing parental smoking may help lower this risk. These findings underscore the importance of public health interventions to reduce children's exposure to secondhand smoke.

Keywords: parental smoking, allergic conjunctivitis, pediatric

Introduction

Allergic conjunctivitis (AC) is one of the most common eye diseases in children. AC can cause a range of ocular surface symptoms, including dry eyes, itching, and foreign body sensation. Vernal keratoconjunctivitis and atopic keratoconjunctivitis are the most aggressive forms that can lead to visual impairment and even blindness. If not treated promptly, it may lead to visual impairment.[1] One study showed that AC affects 40% of the general population, and its incidence is increasing annually,[2] especially in children and developing countries.[3] It has been suggested that environmental factors contribute to disease onset.

Passive smoking, most commonly from parental smoking, has significant adverse health effects in children.[4] Parental smoking is the most common cause of passive smoking in children and significantly impacts their health. It has been linked to conditions such as attention-deficit hyperactivity disorder,[5] bronchiolitis,[6,7] and sudden infant death syndrome[8] and is also associated with allergic diseases, including asthma, atopic dermatitis, and allergic rhinitis.[9,10] A review reported that the risk of developing allergic rhinoconjunctivitis may be increased by passive smoking.[11] However, most previous studies have focused on parental smoking and allergic diseases such as atopic dermatitis, asthma, and allergic rhinitis, and, to our knowledge, no study has examined AC specifically in Chinese children. Research evidence from Chinese pediatric populations regarding the relationship between parental smoking and AC remains limited.

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This study investigated the association between parental smoking and AC in Chinese children, addressing a critical evidence gap.

Materials and Methods

Study Design and Participants

A cross-sectional study was conducted at the Department of Ophthalmology, Tianjin Children's Hospital, between October 2021 and September 2022. All pediatric patients underwent comprehensive ophthalmic examinations and were classified into the AC group (n = 2,101) or the non-AC group (n = 2,148) based on clinical findings.

This study was conducted in accordance with the Declaration of Helsinki and approved by the institutional ethics committee. Parents or legal guardians were informed of the study purpose and provided oral informed consent before enrollment.

Study Variables and Outcome

All pediatric participants in this study underwent a routine ophthalmic examination by the same physician and were diagnosed with AC according to the Japanese guidelines for allergic conjunctival diseases (2017).[12] Myopia was defined as a spherical equivalent (SE) of −0.50 diopters (D) or less, hyperopia as an SE of 2.00 D or more, and astigmatism as an absolute value of SE of 1.00 D or more. Strabismus was diagnosed based on synoptophore results.

A structured questionnaire collected demographic data, maternal and pregnancy-related factors, and parental smoking history. These data were obtained for all patients. Demographic information included age, sex (male or female), race (Han or minority), season of birth (spring, summer, autumn, or winter), season of visit (spring, summer, autumn, and winter), mother's educational background (less than high school, high school or equivalent, college or above, or other), father's educational background (less than high school, high school or equivalent, college or above, or other), and parental high myopia (neither, one parent, or both parents). Maternal history and pregnancy-related factors included preterm birth (no or yes), mode of delivery (natural birth or cesarean section), multiple pregnancies (no or yes), and feeding method (breastfeeding, formula feeding, or mixed feeding). Parental smoking status was obtained from the questionnaire.

Statistical Analysis

We performed all analyses using the statistical software packages R (http://www.R-project.org, Te R Foundation) and Free Statistics software version 2.1. All normally distributed continuous variables were expressed presented as mean ± standard deviation, and skewed continuous variables as median interquartile range. Categorical variables were presented as frequencies (%). Comparisons of continuous variables among groups were performed using the independent samples Student's *t*-test or Mann–Whitney *U*-test, depending on the normality of the distribution. Categorical data were compared using the chi-square or Fisher's exact test.

We used logistic regression to examine the association between parental smoking and AC. Confounders were selected based on clinical relevance, prior literature, statistical significance in univariate analysis, a change in effect estimate >10%, or known associations with the outcomes of interest.

Two multivariate logistic regression models were constructed: Model 1 adjusted for age, sex, season of visit, and parental educational background; Model 2 additionally adjusted for strabismus, refractive errors (myopia, hyperopia, astigmatism), and parental high myopia.

A series of sensitivity analyses was conducted to evaluate the robustness of the findings and how conclusions were affected by applying various association inference models. Subgroup analyses were performed, and the interaction across subgroups was tested using the likelihood ratio test. Additional association inference models were applied, including propensity score adjusted (PSA), propensity score matching (PSM), inverse probability of treatment weighting (IPTW), standardized mortality ratio weighting (SMRW), pairwise algorithmic (PA), overlap weight (OW), and double robust analysis. Effect sizes and p-values from all these models were reported and compared.

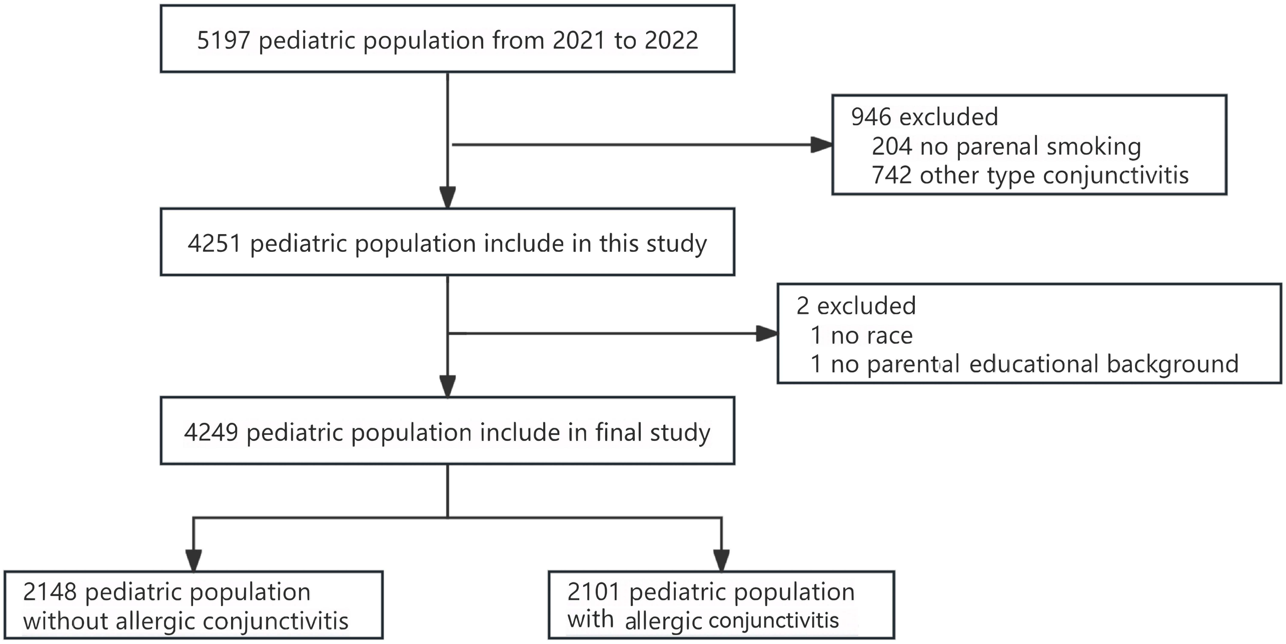

Figure 1 The flow chart.

Results

Baseline Characteristics of the Participants

We identified 5,197 potential participants. A total of 204 participants without a history of parental smoking and 742 participants with other types of conjunctivitis were excluded. Participants with missing data on race (n = 1) and parental educational background (n = 1) were also excluded. A total of 4,249 participants met the inclusion criteria and were analyzed. We presents the inclusion and exclusion flowchart (Figure 1).

Table 1 shows the baseline characteristics of participants with and without AC. AC was present in 2,101 participants (49.4%). Compared with the non-AC group, the AC group had a higher proportion of females ($P < 0.001$), higher

Table 1 Baseline Characteristics of Participants

Covariate	Total n = 4249	Allergic Conjunctivitis		P-values
		No	Yes	
		n = 2148	n = 2101	
Age, Mean ± SD	5.1 ± 3.1	5.1 ± 3.6	5.0 ± 2.5	0.260
Sex, n (%)				< 0.001
Male	2601 (61.2)	1172 (54.6)	1429 (68.0)	
Female	1648 (38.8)	976 (45.4)	672 (32.0)	
Season of Birth, n (%)				0.674
Spring	1087 (26.0)	538 (25.8)	549 (26.3)	
Summer	1055 (25.2)	543 (26.0)	512 (24.5)	
Autumn	859 (20.6)	419 (20.1)	440 (21.0)	
Winter	1179 (28.2)	589 (28.2)	590 (28.2)	
Season for visiting, n (%)				< 0.001
Spring	1099 (25.9)	400 (18.6)	699 (33.3)	
Summer	1087 (25.6)	684 (31.8)	403 (19.2)	
Autumn	1140 (26.8)	549 (25.6)	591 (28.1)	
Winter	923 (21.7)	515 (24)	408 (19.4)	

(*Continued*)

Table 1 (Continued).

Covariate	Total n = 4249	Allergic Conjunctivitis		P-values
		No n = 2148	Yes n = 2101	
Race, n (%)				0.682
Han	4142 (97.5)	2096 (97.6)	2046 (97.4)	
Minor race	107 (2.5)	52 (2.4)	55 (2.6)	
Preterm, n (%)				0.665
No	4129 (97.2)	2085 (97.1)	2044 (97.3)	
Yes	120 (2.8)	63 (2.9)	57 (2.7)	
Mode of delivery, n (%)				0.253
Natural birth	2009 (47.3)	997 (46.4)	1012 (48.2)	
Caesarean section	2240 (52.7)	1151 (53.6)	1089 (51.8)	
Multiple pregnancies, n (%)				0.671
No	4158 (97.9)	2104 (98)	2054 (97.8)	
Yes	91 (2.1)	44 (2)	47 (2.2)	
Feeding method, n (%)				0.997
Breastfeeding	2690 (63.3)	1361 (63.4)	1329 (63.3)	
Artificial feeding	759 (17.9)	383 (17.8)	376 (17.9)	
Mixed feeding	800 (18.8)	404 (18.8)	396 (18.8)	
Mother's educational background, n (%)				< 0.001
Less than high school	608 (14.3)	387 (18)	221 (10.5)	
High school or equivalent	781 (18.4)	421 (19.6)	360 (17.1)	
College or above and other	2860 (67.3)	1340 (62.4)	1520 (72.3)	
Father's educational background, n (%)				< 0.001
Less than high school	601 (14.1)	379 (17.6)	222 (10.6)	
High school or equivalent	789 (18.6)	436 (20.3)	353 (16.8)	
College or above and other	2859 (67.3)	1333 (62.1)	1526 (72.6)	
Parental high myopia, n (%)				0.015
No	3489 (82.1)	1786 (83.1)	1703 (81.1)	
Only one	705 (16.6)	344 (16.0)	361 (17.2)	
Both	55 (1.3)	18 (0.8)	37 (1.8)	
Strabismus, n (%)				< 0.001
No	3862 (90.9)	1883 (87.7)	1979 (94.2)	
Yes	387 (9.1)	265 (12.3)	122 (5.8)	
Myopia, n (%)				< 0.001
No	3173 (74.7)	1452 (67.6)	1721 (81.9)	
Yes	1076 (25.3)	696 (32.4)	380 (18.1)	
Hyperopia, n (%)				< 0.001
No	4137 (97.4)	2054 (95.6)	2083 (99.1)	
Yes	112 (2.6)	94 (4.4)	18 (0.9)	
Astigmatism, n (%)				< 0.001
No	3561 (83.8)	1712 (79.7)	1849 (88.0)	
Yes	688 (16.2)	436 (20.3)	252 (12.0)	

parental educational attainment ($P < 0.01$), and a greater prevalence of strabismus, myopia, hyperopia, and astigmatism ($P < 0.001$). Significant differences in the season of visit were observed between the groups ($P < 0.05$). No significant differences were observed between groups for age, season of birth, race, preterm birth, mode of delivery, multiple pregnancies, or feeding method (all $P > 0.05$).

Table 2 Multivariate Analysis of the Association Between Parental Smoking and Allergic Conjunctivitis

Covariate	Allergic Conjunctivitis (n=2101)					
	Model 1		Model 2		Model 3	
	OR (95% CI)	P-value	OR (95% CI)	P-value	OR (95% CI)	P-value
Parental smoking	1(Ref) 1.03 (0.91–1.16)	0.649	1(Ref) 1.17 (1.03–1.33)	0.02	1(Ref) 1.17 (1.03–1.34)	0.020

Notes: Adjusted covariates: Model 1: unadjusted; Model 2: adjusted by age, sex, season for visiting, mother's educational background, father's educational background; Model 3: Model 2+strabismus, myopia, hyperopia, astigmatism, parental high myopia.
Abbreviations: OR, odds ratio; 95% CI, 95% confidence interval.

Association Between Parental Smoking and Allergic Conjunctivitis

Based on the univariate logistic regression analysis (Supplementary Table 1), sex, season of visit, mother's educational background, father's educational background, strabismus, myopia, hyperopia, astigmatism, and parental high myopia were all significantly related to AC (all $P < 0.05$).

In the analysis of the association between parental smoking and AC (Table 2), the unadjusted model (Model 1) showed an increased risk of AC in patients with parental smoking (odds ratio (OR): 1.03, 95% confidence interval (CI): 0.91–1.16). In Model 2, adjusted for age, sex, season of visit, and parental educational background, the OR was 1.17 (95% CI: 1.03–1.33). After further controlling for Model 2 covariates along with strabismus, myopia, hyperopia, astigmatism, and parental high myopia, AC was still significantly associated with parental smoking (OR: 1.17, 95% CI: 1.03–1.34). These findings are detailed in Table 2.

Subgroup Analysis

Subgroup analyses of the association between parental smoking and AC were presented (Figure 2). Subgroups based on myopia, hyperopia, and astigmatism showed no statistically significant interactions (all P for interaction > 0.05), and the association between parental smoking and AC remained consistent across all subgroups. Significant interactions were observed for mode of delivery and multiple pregnancies. The association between parental smoking and AC was stronger among children born via cesarean section compared to natural delivery, and among those from multiple pregnancies compared to singletons.

PSM Analysis

Furthermore, PSM analysis, adjusted for the primary confounding covariates between the parental smoking groups, was performed to evaluate the consistency of our results. After PSM, baseline characteristics between the two groups were well balanced (Supplementary Table 2 and Supplementary Figures 1–2).

Among the 1,566 propensity-matched pairs, AC was associated with parental smoking. The ORs were 1.03–1.26 calculated by univariate, multivariate logistic regression analyses, PSM, PSA, IPTW, SMRW, PA, OW, and doubly robust estimate (Figure 3).

Discussion

In this cross-sectional study of a Chinese pediatric population, this study demonstrates that parental smoking is associated with a 17% higher odd of AC in children. Moreover, the association between parental smoking and AC was stronger in participants born by cesarean section than in those born by natural delivery, and in multiple pregnancies than in single pregnancies. These findings may have important clinical and public health implications.

Previous reports have shown that parental smoking increases the risk of allergic diseases in children. Exposure to passive smoking has been reported to significantly increase the incidence of childhood asthma, eczema, and allergic rhinitis.[13,14] Yamada et al Suggested that there is a positive association of perinatal smoking and the prevalence of asthma and wheezing among girls.[15] A Danish birth cohort showed that children with prenatal smoking exposure had double the risk of developing asthma.[16] However, few studies have focused on AC. Our study is the first, to our knowledge, to

Subgroup	Parental smoking	Event (%)	OR (95%CI)	P for interaction
Overall				
Crude	**1775**	**885 (49.9)**	**1.03 (0.91−1.16)**	**0.649**
Adjusted			**1.17 (1.03−1.34)**	**0.020**
Mode of production				0.008
Natural birth	793	380 (47.9)	0.99 (0.82−1.21)	
Caesarean section	982	505 (51.4)	1.36 (1.13−1.63)	
Multiple pregnancies				0.029
No	1742	862 (49.5)	1.15 (1.00−1.32)	
Yes	33	23 (69.7)	7.01 (1.85−26.60)	
Myopia				0.776
No	1308	716 (54.7)	1.13 (0.97−1.32)	
Yes	467	169 (36.2)	1.32 (0.99−1.76)	
Hyperopia				0.256
No	1729	876 (50.7)	1.16 (1.01−1.33)	
Yes	46	9 (19.6)	2.35 (0.58−9.49)	
Astigmatism				0.110
No	1481	761 (51.4)	1.12 (0.97−1.30)	
Yes	294	124 (42.2)	1.62 (1.13−2.31)	

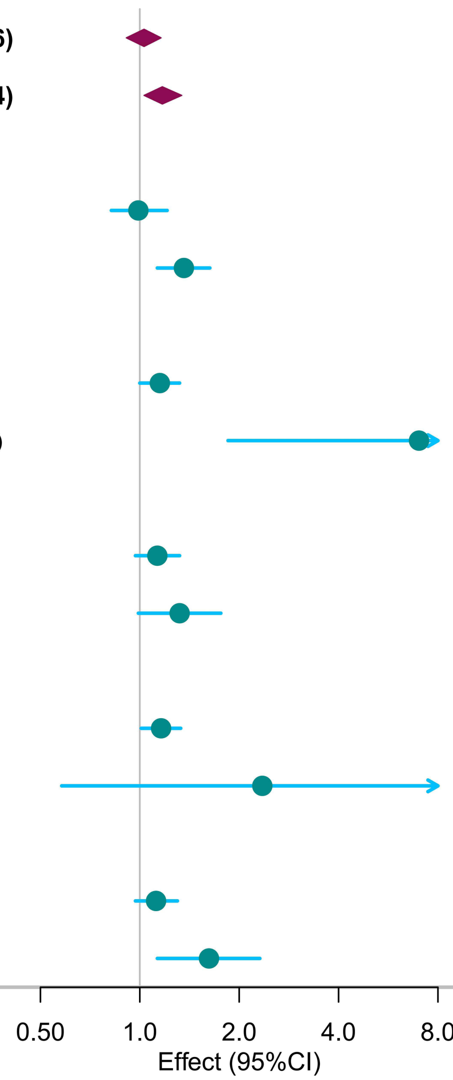

Figure 2 Effect size of parental smoking on the presence of allergic conjunctivitis in the mode of delivery, multiple pregnancies, myopia, hyperopia, and astigmatism subgroup.
Abbreviations: OR, odds ratio; 95% CI, 95% confidence interval.

identify a positive association between parental smoking and AC in a Chinese pediatric population. A study in Italy showed that tobacco exposure increased the incidence of AC in females.[17] Lee et al found that passive smoking in adolescents was associated with asthma, allergic rhinitis, and atopic dermatitis.[18] While prior studies have examined maternal smoking during pregnancy or active smoking and allergic diseases, our study focused on the effects of parental smoking in children's living environments.

A European birth cohort suggested that tobacco smoke exposure during infancy or fetal life increases the risk of asthma in adolescence and childhood, and high exposure also increases the risk of persistent rhinoconjunctivitis.[19] A study on allergic diseases in children reported that maternal smoking during infancy increased the risk of developing AC and allergic rhinitis in children.[10] Mitchell et al suggested that parental smoking was associated with an increased risk of eczema, asthma and rhinoconjunctivitis symptoms in children and adolescents.[20] These studies did not distinguish

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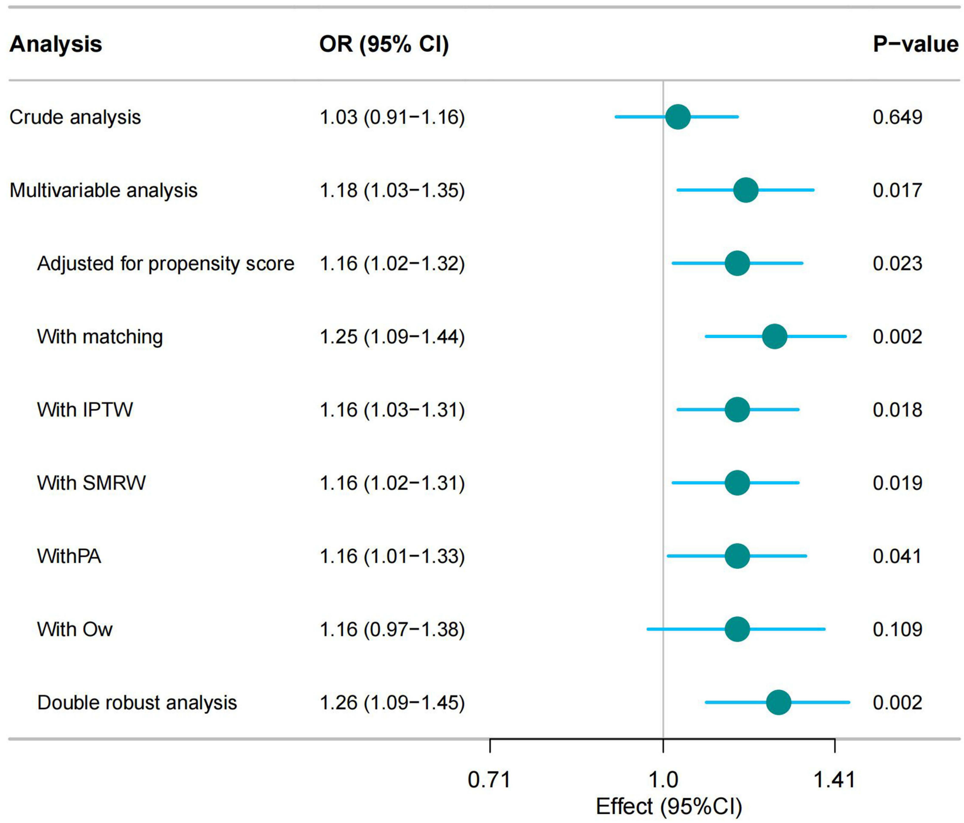

Figure 3 Forest plot shows ORs of allergic conjunctivitis using a variety of models.
Abbreviations: OR, odds ratio; 95% CI, 95% confidence interval; IPTW, inverse probability of treatment weighting; SMRW, standardized mortality ratio weighting; PA, pairwise algorithmic; OW, overlap weight.

between allergic rhinitis and AC, and were unable to only analyze the risk of developing AC. Our study specifically explored the association between AC and parental smoking status.

A systematic review on ocular health and secondhand smoke exposure showed that such smoke exposure was related to allergic changes and inflammatory of the eyes.[21] A previous study showed that AC in Russian children was associated with maternal smoking during infancy and at the time of study.[22] These findings were consistent with those from our study. Ocular surface inflammation may be induced by smoking. One report suggested that smoking may be a risk factor for chalazion, a common eyelid inflammation disorder.[23] Another study showed that current smoking habits may be independent risk factors for conjunctivitis after COVID-19.[24] However, the underlying mechanisms by which exposure to smoke might be associated with the development of AC remain unclear and warrant further investigation.

Many evidence supports the link between parental smoking and markers of atopy in children, such as serum eosinophilia, immunoglobulin E (IgE) levels and positive skin-prick tests.[25] One study reported that specific IgE antibodies to the peanut-related allergens were significantly increased at 16 years of age with exposed to passive smoking during infancy.[26] A review and some laboratory studies using animal models both showed that inflammatory processes can be affected by tobacco smoke, leading to an increase in collagen production, eosinophils levels, and airway remodeling.[27,28] It may also alter the inflammatory responses generated by respiratory epithelial cells and modulating the production of chemokines and potent proinflammatory cytokines, promoting the recruitment of neutrophils and macrophages, and contributing to lung tissue damage.[28] Bozinovski et al recently showed that cigarette smoke exposure promotes the release of a proinflammatory cytokine from nonconventional T cell sources, interleukin (IL)-17A implicated in the pathogenesis of asthma.[29] Cigarette smoke exerts cytotoxic; there are both proinflammatory and anti-inflammatory effects on nasal epithelial cells. It leads to increased reactive oxygen species production, IL-17A synthesis, lipopolysaccharide, and Toll-like receptor 4 expression.[30,31]

The pathological conditions of AC, which involves lesions in the conjunctiva, may be caused by interactions between various resident cells and immune system cells mediated by physiologically active substances (eg, leukotriene and histamine), chemokines, and cytokines. The main effector cells in AC are eosinophils. Keratoconjunctival disorders may be caused by cytotoxic proteins which released from eosinophils infiltrating the conjunctiva.[12] Smoking had been shown to inhibit the expression of proinflammatory cytokines and T cell activation, and reduce serum levels of immunoglobulins.[32,33] It has been also demonstrated to inhibit multiple inflammatory mediators, leading to increased susceptibility to infections.[34] Exposure to tobacco smoke can plausibly skew immune responses and alter the immune functions of various immune cells, thereby aggravating allergic inflammation and sensitization.[28] These mechanisms may help explain the associations observed between smoking exposure and AC in our study.[35] Further investigations are warranted to validate our findings and explore the detailed relationships and underlying mechanisms.

This study had some limitations. First, the cross-sectional design precludes causal inference between parental smoking and AC, despite the large, representative sample. Therefore, it requires longitudinal studies to determine whether the observed relationship between parental smoking and AC is causal. Second, despite adjusting for available demographic factors, residual confounding by unmeasured variables such as family atopy history and detailed socio-economic status indicators may still be present. Third, the diagnosis of allergic conjunctivitis was based on clinical signs and symptoms rather than objective laboratory confirmatory tests, such as conjunctival eosinophil count or tear IgE levels. Although this approach is consistent with common practice in large-scale epidemiological research and was conducted by specialist ophthalmologists, it may potentially lead to misclassification bias. Future studies incorporating specific IgE testing or conjunctival provocation tests could help to validate our findings in a more definitively diagnosed cohort. Forth, our analysis did not differentiate based on whether one or both parents smoked. We should investigate the distinct effects of paternal, maternal, and biparental smoking on allergic conjunctivitis in future research with larger sample sizes. Finally, as some data were self-reported via questionnaire, recall and reporting biases are possible. Residual measured or unmeasured confounding may still influence the findings. Future studies should address these limitations to validate and expand on our results.

Conclusion

This study highlights the influence of inappropriate parental smoking on the risk of incident AC. Parental smoking was associated with increased risk of AC in children. Reducing parental smoking may help lower this risk. These findings underscore the importance of public health interventions to reduce children's exposure to secondhand smoke.

Abbreviations

AC, allergic conjunctivitis; SE, spherical equivalent; D, diopters; PSA, propensity score adjusted; PSM, propensity score matching; IPTW, inverse probability of treatment weighting; SMRW, standardized mortality ratio weighting; PA, pairwise algorithmic; OW, overlap weight; OR, odds ratio; 95% CI, 95% confidence interval; IgE, immunoglobulin E; IL, interleukin, SMD, Standardized Mean Difference.

Ethics Approval and Informed Consent

This study was performed in line with the principles of the Declaration of Helsinki. This study was approved by the Institutional Review Board of the Tianjin Children's Hospital (No. 2023-TYXWKY-003).

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The author(s) report no conflicts of interest in this work.

References

1. Zhou J, Fan L, Lin H, et al. Size-specific particulate matter and outpatient visits for allergic conjunctivitis in children: a time-stratified case-crossover study in Guangzhou, China. *Environ Sci Pollut Res Int*. 2022;30(12):33949–33959. doi:10.1007/s11356-022-24564-7
2. Wang J, Sun M, Li G, Yin D, Hu C, Sun J. The Spectrum and Age-Sex Patterns Among Outpatients with Allergic Diseases - Yichang City, Hubei Province, China, 2018-2021. *China CDC Wkly*. 2023;5(31):681–686. doi:10.46234/ccdcw2023.131
3. Vazirani J, Shukla S, Chhawchharia R, Sahu S, Gokhale N, Basu S. Allergic conjunctivitis in children: current understanding and future perspectives. *Curr Opin Allergy Clin Immunol*. 2020;20(5):507–515. doi:10.1097/aci.0000000000000675
4. Sureda X, Fernández E, López MJ, Nebot M. Secondhand tobacco smoke exposure in open and semi-open settings: a systematic review. *Environ Health Perspect*. 2013;121(7):766–773. doi:10.1289/ehp.1205806
5. Huang A, Wu K, Cai Z, Lin Y, Zhang X, Huang Y. Association between postnatal second-hand smoke exposure and ADHD in children: a systematic review and meta-analysis. *Environ Sci Pollut Res Int*. 2021;28(2):1370–1380. doi:10.1007/s11356-020-11269-y
6. Lanari M, Vandini S, Adorni F, et al. Prenatal tobacco smoke exposure increases hospitalizations for bronchiolitis in infants. *Respir Res*. 2015;16:152. doi:10.1186/s12931-015-0312-5
7. Akar-Ghibril N, Phipatanakul W. The indoor environment and childhood asthma. *Curr Allergy Asthma Rep*. 2020;20(9):43. doi:10.1007/s11882-020-00941-5
8. Prescott SL. Effects of early cigarette smoke exposure on early immune development and respiratory disease. *Paediatr Respir Rev*. 2008;9(1):3–9. doi:10.1016/j.prrv.2007.11.004
9. Tanaka K, Miyake Y, Furukawa S, Arakawa M. Pre- and postnatal smoking exposure and risk of atopic eczema in young japanese children: a prospective prebirth cohort study. *Nicotine Tob Res*. 2017;19(7):804–809. doi:10.1093/ntr/ntw299
10. Shigehara K, Matsumoto N, Tsuge M, et al. Maternal smoking during infancy increases the risk of allergic diseases in children: a nationwide longitudinal survey in Japan. *Allergy Asthma Clin Immunol*. 2025;21(1):4. doi:10.1186/s13223-025-00952-9
11. Thong BY. Allergic conjunctivitis in Asia. *Asia Pac Allergy*. 2017;7(2):57–64. doi:10.5415/apallergy.2017.7.2.57
12. Takamura E, Uchio E, Ebihara N, et al. Japanese guidelines for allergic conjunctival diseases 2017. *Allergol Int*. 2017;66(2):220–229. doi:10.1016/j.alit.2016.12.004
13. Thacher JD, Gruzieva O, Pershagen G, et al. Pre- and postnatal exposure to parental smoking and allergic disease through adolescence. *Pediatrics*. 2014;134(3):428–434. doi:10.1542/peds.2014-0427
14. Yuan X, Liu L, Zhang B, et al. Unveiling the impact of smoking on allergic rhinitis: disease severity and efficacy of subcutaneous immunotherapy. *Otolaryngol Head Neck Surg*. 2024;171(6):1887–1896. doi:10.1002/ohn.937
15. Yamada M, Tanaka K, Nagata C, Arakawa M, Miyake Y. Sex differences in the association between smoking exposure and prevalence of wheeze and asthma in 3-year-old children. *J Asthma*. 2023;60(7):1369–1376. doi:10.1080/02770903.2022.2147081
16. Sunde RB, Thorsen J, Pedersen CT, et al. Prenatal tobacco exposure and risk of asthma and allergy outcomes in childhood. *Eur Respir J*. 2022;59(2). doi:10.1183/13993003.00453-2021
17. Simoni M, Baldacci S, Puntoni R, et al. Respiratory symptoms/diseases and environmental tobacco smoke (ETS) in never smoker Italian women. *Respir Med*. 2007;101(3):531–538. doi:10.1016/j.rmed.2006.06.021
18. Lee A, Lee SY, Lee KS. The use of heated tobacco products is associated with asthma, allergic rhinitis, and atopic dermatitis in korean adolescents. *Sci Rep*. 2019;9(1):17699. doi:10.1038/s41598-019-54102-4
19. Thacher JD, Gehring U, Gruzieva O, et al. Maternal smoking during pregnancy and early childhood and development of asthma and rhinoconjunctivitis - a MeDALL project. *Environ Health Perspect*. 2018;126(4):047005. doi:10.1289/ehp2738
20. Mitchell EA, Beasley R, Keil U, Montefort S, Odhiambo J. The association between tobacco and the risk of asthma, rhinoconjunctivitis and eczema in children and adolescents: analyses from Phase Three of the ISAAC programme. *Thorax*. 2012;67(11):941–949. doi:10.1136/thoraxjnl-2011-200901
21. Zhang Y, Zhang XJ, Yuan N, et al. Secondhand smoke exposure and ocular health: a systematic review. *Surv Ophthalmol*. 2023;68(6):1166–1207. doi:10.1016/j.survophthal.2023.07.001
22. Hugg TT, Jaakkola MS, Ruotsalainen RO, Pushkarev VJ, Jaakkola JJ. Parental smoking behaviour and effects of tobacco smoke on children's health in Finland and Russia. *Eur J Public Health*. 2008;18(1):55–62. doi:10.1093/eurpub/ckm053
23. Patel S, Tohme N, Gorrin E, Kumar N, Goldhagen B, Galor A. Prevalence and risk factors for chalazion in an older veteran population. *Br J Ophthalmol*. 2022;106(9):1200–1205. doi:10.1136/bjophthalmol-2020-318420
24. Tajima A, Sassa Y, Ishio D, et al. Clinical features of 26 cases of COVID-19-associated conjunctivitis. *Jpn J Ophthalmol*. 2024;68(1):57–63. doi:10.1007/s10384-023-01033-3
25. Ronchetti R, Macri F, Ciofetta G, et al. Increased serum IgE and increased prevalence of eosinophilia in 9-year-old children of smoking parents. *J Allergy Clin Immunol*. 1990;86(3 Pt 1):400–407. doi:10.1016/s0091-6749(05)80104-6
26. Feldman LY, Thacher JD, van Hage M, et al. Early-life secondhand smoke exposure and food hypersensitivity through adolescence. *Allergy*. 2018;73(7):1558–1561. doi:10.1111/all.13463

27. Botelho FM, Llop-Guevara A, Trimble NJ, et al. Cigarette smoke differentially affects eosinophilia and remodeling in a model of house dust mite asthma. *Am J Respir Cell Mol Biol*. 2011;45(4):753–760. doi:10.1165/rcmb.2010-0404OC
28. Strzelak A, Ratajczak A, Adamiec A, Feleszko W. Tobacco smoke induces and alters immune responses in the lung triggering inflammation, allergy, asthma and other lung diseases: a mechanistic review. *Int J Environ Res Public Health*. 2018;15(5). doi:10.3390/ijerph15051033
29. Bozinovski S, Seow HJ, Chan SP, et al. Innate cellular sources of interleukin-17A regulate macrophage accumulation in cigarette- smoke-induced lung inflammation in mice. *Clin Sci*. 2015;129(9):785–796. doi:10.1042/cs20140703
30. Comer DM, Elborn JS, Ennis M. Inflammatory and cytotoxic effects of acrolein, nicotine, acetylaldehyde and cigarette smoke extract on human nasal epithelial cells. *BMC Pulm Med*. 2014;14:32. doi:10.1186/1471-2466-14-32
31. Huang CC, Wang CH, Fu CH, et al. Association between cigarette smoking and interleukin-17A expression in nasal tissues of patients with chronic rhinosinusitis and asthma. *Medicine*. 2016;95(47):e5432. doi:10.1097/md.0000000000005432
32. Ouyang Y, Virasch N, Hao P, et al. Suppression of human IL-1beta, IL-2, IFN-gamma, and TNF-alpha production by cigarette smoke extracts. *J Allergy Clin Immunol*. 2000;106(2):280–287. doi:10.1067/mai.2000.107751
33. Sopori M. Effects of cigarette smoke on the immune system. *Nat Rev Immunol*. 2002;2(5):372–377. doi:10.1038/nri803
34. Chen H, Cowan MJ, Hasday JD, Vogel SN, Medvedev AE. Tobacco smoking inhibits expression of proinflammatory cytokines and activation of IL-1R-associated kinase, p38, and NF-kappaB in alveolar macrophages stimulated with TLR2 and TLR4 agonists. *J Immunol*. 2007;179(9):6097–6106. doi:10.4049/jimmunol.179.9.6097
35. Kum-Nji P, Meloy L, Herrod HG. Environmental tobacco smoke exposure: prevalence and mechanisms of causation of infections in children. *Pediatrics*. 2006;117(5):1745–1754. doi:10.1542/peds.2005-1886