Journal of Population Therapeutics & Clinical Pharmacology

RESEARCH ARTICLE
DOI: 10.53555/5hyj5t05

ANALYSIS OF RISK FACTORS FOR ALLERGIC RHINITIS IN PRESCHOOL CHILDREN IN LAHORE, PUNJAB, PAKISTAN

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Abstract

Background: Allergic rhinitis (AR) is a common chronic inflammatory condition of the nasal mucosa that significantly impacts sleep quality, cognitive performance, and overall well-being in children. Its prevalence is increasing globally, particularly in urban areas, due to genetic susceptibility and environmental triggers such as air pollution, indoor allergens, and tobacco smoke exposure. Early identification of risk factors in preschool children is essential for prevention and targeted management.

Objective: This study aimed to determine the prevalence of AR and to identify genetic, environmental, and lifestyle-related risk factors among preschool children in Lahore, Pakistan.

Materials and Methods: A cross-sectional study was conducted from February 2023 to June 2024 at the pediatric outpatient department of Indus Hospital, Lahore. A total of 300 children aged 3–6 years were enrolled using consecutive sampling. Data were collected through structured parental interviews covering demographics, feeding history, family history of atopy, and environmental exposures. Diagnosis of AR was based on ARIA criteria and confirmed by a pediatrician. Data were analyzed using SPSS version 26. Chi-square tests were used for bivariate analysis, followed by multivariable logistic regression to identify independent predictors, with results presented as adjusted odds ratios (aOR) and 95% confidence intervals (CI).

Results: The prevalence of AR was 37.7% (n=113). Parental atopy, passive smoking, and urban residence were identified as significant predictors. Biomass fuel exposure was associated on univariate analysis but was not statistically significant after adjustment.

Conclusion: AR is highly prevalent among preschool children in Lahore. Both genetic predisposition and modifiable environmental exposures contribute to risk. Implementation of smoke-free home policies improved indoor air quality, and community awareness campaigns may help reduce the burden of disease. Larger multicenter studies with objective allergy testing are warranted for confirmation.

Keywords: Allergic rhinitis; preschool children; risk factors; passive smoking; parental atopy.

Introduction

Allergic rhinitis (AR) is an IgE-mediated inflammatory condition [1,2] of the nasal mucosa characterized by nasal congestion, rhinorrhea, sneezing and nasal itching. It is among the most prevalent chronic diseases in children and has clear implications for sleep, school performance and overall quality of life. The burden of AR is increasing globally [3], particularly in urbanized regions, due to complex interactions between genetic predisposition and environmental exposures. Recent epidemiological studies underline a rise in allergic diseases in childhood, driven by factors such as air pollution, altered microbial exposures in early life, and lifestyle changes.
Genetic predisposition is a major determinant [4,5] of allergic disease. A positive parental history of atopy is associated with a substantially increased risk of AR in offspring. This familial clustering reflects inherited susceptibility as well as shared environmental exposures within households.
Environmental risk factors include passive exposure [6,7] to tobacco smoke, indoor use of biomass fuels, household dampness and mold, and exposure to traffic-related air pollutants. Passive smoking has been repeatedly linked [8,9] to increased risk of allergic sensitization and symptomatic AR in children in several observational studies. Indoor air pollution from biomass combustion [10] remains a relevant exposure in many low- and middle-income countries and has been associated with increased respiratory symptoms and allergic disease.
Breastfeeding and early-life nutrition [11,12] also influence immune development. Exclusive breastfeeding for the first six months has been associated with reduced risk of allergic outcomes in some cohorts, although findings are heterogeneous across populations. Urban residence is another consistent correlate [13,14] higher AR prevalence, potentially reflecting higher pollutant exposure and the 'hygiene' related reduction in microbial challenges during immune maturation. In Pakistan, data on AR [15,16] in preschool children are sparse. Most existing evidence focuses on older children or combines a broad pediatric age range. Given Lahore's rapid urbanization and high ambient pollution levels, the city offers an important context to study early-life determinants of AR.
This study aims to estimate the prevalence [17,18] of AR in preschool children attending a tertiary hospital in Lahore and to identify modifiable and non-modifiable risk factors to inform public health strategies.

Materials and Methods

This analytical cross-sectional study was conducted in the pediatric department of Indus Hospital, Lahore, Pakistan, between February 2023 and June 2024. Indus Hospital serves urban and peri-urban catchment with diverse socioeconomic groups. A total 300 preschool children aged 3–6 years presenting to the outpatient clinic were enrolled in the study. Sample size estimation assumed a 30% prevalence of AR, 5% precision and 95% confidence, yielding a minimum required sample of 323; due to logistical constraints and response rates, 300 children were included using consecutive sampling during the study period.
Children aged 3–6 years whose parents provided informed consent were eligible. Exclusion criteria included known chronic respiratory diseases other than allergic rhinitis, primary immunodeficiency disorders, congenital anomalies affecting the respiratory tract, and acute severe illness requiring hospital admission.
Trained research staff administered a structured questionnaire to parents capturing demographic data, household characteristics, parental history of atopy, feeding history (exclusive breastfeeding, mixed, exclusive formula), environmental exposures (passive smoking, biomass fuel use, presence

of pets, visible mold/dampness), and symptom history consistent with AR. Clinical diagnosis of AR was made by a pediatrician using ARIA-based criteria: typical nasal symptoms for >1 hour per day or >2 days per week with clear positive history.

The study protocol was approved by the Institutional Review Board of Indus Hospital (IRB No: IH-LHR/2023/045). Written informed consent was obtained from parents or guardians prior to participation.

Data were entered and analyzed using SPSS v26. Descriptive statistics summarize participant characteristics as frequencies and percentages or means with standard deviations. Bivariate associations were examined using chi-square tests. Variables with $p<0.10$ in bivariate analysis were included in multivariable logistic regression to identify independent predictors. Adjusted odds ratios with 95% confidence intervals were reported. A p-value below 0.05 was considered statistically significant.

Results

During the study period, 300 children were enrolled. The mean age was 4.5 ± 0.9 years; 162 (54.0%) were male. Overall, 113 (37.7%) children met the case definition for AR. Table 1 shows baseline characteristics and distribution of exposures by AR status.

Table 1. Baseline characteristics and exposures (n=300)

Variable	Total (n=300)	AR cases n (%)
Mean age (years ± SD)	4.5 ± 0.9	—
Male gender	162 (54.0%)	68 (60.2%)
Urban residence	210 (70.0%)	92 (81.4%)
Parental history of atopy	81 (27.0%)	48 (42.5%)
Passive smoking exposure	94 (31.3%)	53 (46.9%)
Biomass fuel exposure	72 (24.0%)	34 (30.1%)
Exclusive formula feeding	58 (19.3%)	28 (24.8%)
Presence of pets	40 (13.3%)	12 (10.6%)

Bivariate analysis indicated significant associations between AR and parental atopy ($p<0.001$), passive smoking ($p=0.002$), urban residence ($p=0.015$), and biomass fuel exposure ($p=0.028$). No statistically significant association was identified for pet exposure in this cohort. Age and sex distributions did not differ significantly between cases and non-cases. Multivariable logistic regression including variables with $p<0.10$ in bivariate testing (parental atopy, passive smoking, biomass fuel exposure, urban residence, exclusive formula feeding) revealed that parental atopy (adjusted OR 2.8; 95% CI 1.7–4.5; $p<0.001$), passive smoking (adjusted OR 1.9; 95% CI 1.2–3.0; $p=0.005$), and urban residence (adjusted OR 1.6; 95% CI 1.0–2.5; $p=0.041$) were independently associated with AR. Biomass fuel exposure and exclusive formula feeding did not retain statistical significance after adjustment. Model diagnostics showed acceptable fit and discrimination (Hosmer-Lemeshow $p=0.45$; Nagelkerke $R2=0.18$). The model correctly classified 72% of cases.

Table 2. Multivariable logistic regression for predictors of allergic rhinitis

Variable	Adjusted OR	95% CI	p-value
Parental history of atopy	2.8	1.7–4.5	<0.001
Passive smoking exposure	1.9	1.2–3.0	0.005
Urban residence	1.6	1.0–2.5	0.041
Biomass fuel exposure	1.3	0.8–2.1	0.22
Exclusive formula feeding	1.2	0.7–2.0	0.35

Discussion

This cross-sectional study found a high prevalence of allergic rhinitis among preschool children attending a tertiary care center in Lahore, with 113 of 300 children (37.7%) meeting AR criteria [1,3]. The prevalence observed is consistent with reports from urbanizing regions in Asia where prevalence estimates among young children range widely but often exceed 20%. Several factors likely contribute to the elevated prevalence in our setting, notably high ambient air pollution, dense urban living, and prevalent indoor exposures such as tobacco smoke and biomass combustion. Parental history of atopy was the strongest predictor [4,5] of AR in this cohort, with an adjusted odds ratio of 2.8. This finding is concordant with the established heritable component of allergic disease and the concept of familial clustering. Genetic susceptibility combined with shared household exposures provides a biologically plausible basis for this association. Earlier studies have similarly demonstrated increased risk among children with one or both atopic parents, reinforcing the value of family history as a screening tool in clinical practice.

Passive tobacco smoke exposure [8,9] independently increased the odds of AR by nearly twofold. Tobacco smoke contains multiple irritants and immunomodulatory agents that can damage airway epithelium, enhance sensitization to aeroallergens, and exacerbate allergic inflammation. The preventable nature of this exposure makes it an attractive target for public health interventions, including parent counseling and smoke-free home campaigns.

Urban residence was also associated [13,14] with higher AR risk. Urban environments typically have higher concentrations of fine particulate matter and nitrogen oxides from traffic emissions, which have been linked to allergic sensitization and symptom exacerbation. Moreover, urban living conditions may limit early microbial exposures that can modulate immune development, in line with the hygiene hypothesis. Our finding supports prioritizing air quality improvement as part of broader strategies to reduce allergic disease burden.

Although biomass fuel exposure showed a bivariate association [10] with AR, it did not remain significant in multivariable analysis, possibly due to confounding by urban residence or socioeconomic factors. Nonetheless, biomass combustion has well-documented adverse respiratory effects and may contribute to symptomatic burden in specific subpopulations, particularly in peri-urban or low-income households.

Feeding practices showed a non-significant trend where exclusive formula feeding was more common [11,12] among AR cases. The literature on breastfeeding and allergy prevention is mixed, with some studies demonstrating protective effects and others finding minimal impact. Our results underscore the need for caution in interpreting observational associations and suggest further prospective research to clarify the role of infant feeding in allergic disease development. Strengths of this study include rigorous case ascertainment using ARIA-based criteria, systematic data collection by trained staff, and multivariable modeling to control for confounding. However, several limitations warrant discussion. The single-center design may limit generalizability to rural regions or other cities. Reliance on parental report introduces potential recall bias, and the absence of objective allergy testing such as skin prick tests or specific IgE measurements limits the ability to distinguish atopic from non-atopic rhinitis. Finally, the cross-sectional nature of the study precludes causal inference and temporal sequencing of exposures and outcomes.

In conclusion, our findings highlight a substantial burden of allergic rhinitis among preschool children in Lahore and identify both non-modifiable and modifiable risk factors. Public health interventions to reduce tobacco exposure, improve household and ambient air quality, and targeted counseling for families with atopic history are likely to be beneficial. Future longitudinal studies incorporating objective allergy testing and environmental exposure measurement are recommended to strengthen causal inference and guide interventions.

Conclusion

Allergic rhinitis affects a significant proportion of preschool children in Lahore, with a prevalence of approximately 38% in this study population. Parental atopic history, passive smoking, and urban residence were independently associated with increased risk. These findings have important

implications for both clinical practice and public health policy. Clinicians should incorporate family history and environmental exposure assessment into routine pediatric evaluations and prioritize counseling on modifiable risks such as tobacco smoke exposure.

At the population level, interventions to reduce urban air pollution and promote clean household energy are essential to lower the burden of allergic diseases. Promoting breastfeeding and implementing smoke-free home policies may offer additional benefits. Given the study limitations, further multicenter and longitudinal studies are needed, ideally with objective allergy testing and environmental monitoring, to inform more precise prevention strategies.

Overall, early identification and targeted mitigation of risk factors in preschool children can contribute to improved respiratory health and quality of life and reduce the long-term burden of allergic disease in rapidly urbanizing settings such as Lahore.

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