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RISK FACTORS ASSOCIATED WITH CHILDHOOD ASTHMA

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Abstract

Objective: To identify and evaluate risk factors associated with childhood asthma among children attending a tertiary-care hospital in Lahore, Pakistan.

Study Design: A case–control study design was employed.

Place and Duration of Study: The Study was conducted at the Department of Pediatrics, Gulab Devi Chest Hospital, Lahore, during the period from May 2025 to September 2025.

Methodology: A cohort of 200 pediatric subjects was recruited, comprising 100 clinically diagnosed cases of asthma and 100 controls matched for age, sex, and residential background, who were free from asthmatic conditions. Comprehensive data pertaining to familial history, parental atopy, exposure to passive tobacco smoke, environmental allergens, early weaning practices, pet ownership, allergic rhinitis, and the frequency of respiratory tract infections were procured utilizing a structured questionnaire following the acquisition of informed consent. The calculation of odds ratios (ORs) was conducted, with statistical significance evaluated through Pearson's chi-square test.

Results: Identified significant risk factors comprised a familial history of asthma (OR 1.8, $p < 0.0001$), the presence of allergic rhinitis (OR 2.7, $p < 0.0001$), exposure to smoking (OR 2.2, $p = 0.0001$), environmental allergens (OR 1.6, $p = 0.0067$), parental atopy (OR 2.0, $p = 0.0034$), and a high frequency of respiratory infections (OR 3.5, $p < 0.0001$). Conversely, early weaning practices and exposure to pets did not exhibit any statistically significant correlation with asthma.

Conclusion: Childhood asthma exhibits a robust association with genetic predisposition, allergic comorbidities, exposure to tobacco smoke, and recurrent respiratory infections. Preventative measures should prioritize the mitigation of environmental risks and the early identification of children at heightened risk.

Keywords: Pediatric asthma, Risk factors, Case–control study, Passive smoking, Allergic rhinitis

INTRODUCTION

Asthma is a chronic inflammatory condition of the airways, characterized by bronchial hyperresponsiveness, airflow obstruction, and recurrent symptoms including wheezing, dyspnea, chest constriction, and cough. It persists as one of the most prevalent chronic respiratory disorders affecting the pediatric population globally and constitutes a significant public health challenge due to its ramifications on quality of life, educational attendance, and healthcare resource utilization [1]. The pathophysiology of asthma is multifaceted, encompassing a complex interplay between genetic susceptibility and environmental exposures. The familial aggregation of asthma and atopic disorders strongly indicates a hereditary component, while environmental factors such as tobacco smoke, indoor allergens, respiratory infections, and early-life exposures further influence the manifestation of the disease [2]. It is estimated that respiratory allergy accounts for nearly half of all asthma cases, although non-atopic asthma phenotypes are also gaining recognition. On a global scale, asthma impacts approximately 334 million individuals, with a substantial prevalence in low- and middle-income nations. In Pakistan, the prevalence of asthma remains elevated, with around 15 million children affected; however, local research investigating modifiable risk factors is notably scarce [3]. Most regional investigations have concentrated on prevalence rather than on etiological determinants, thereby creating a deficit in the evidence required for targeted prevention. Early-life exposures are crucial in shaping lung development and immune system maturation. Prenatal and postnatal exposure to tobacco smoke, environmental pollutants, early infant feeding practices, and recurrent respiratory infections may predispose children to chronic airway inflammation and the development of asthma [4–7]. Identifying these risk factors is paramount for the formulation of preventive strategies, especially within resource-constrained environments. Numerous studies have documented associations between childhood asthma and familial history, allergic rhinitis, parental atopy, passive smoking, and recurrent respiratory infections, while the evidence concerning early weaning practices and pet exposure remains ambiguous [8–10].this study has examined the following risk factors to investigate their link to childhood asthma

MATERIALS AND METHODS

Study Design
This was a hospital-based case–control study conducted to identify risk factors associated with childhood asthma.
Study Setting
The study was carried out in the Pediatrics Outpatient Department of Gulab Devi Chest Hospital, Lahore from May 2025 to September 2025.
Sample Size
A total sample size of 200 participants (100 cases and 100 controls) was calculated using 80% power and a 5% level of significance, based on previously reported prevalence of allergic rhinitis among asthmatic and non-asthmatic children.
Selection Criteria
Inclusion Criteria
- Children aged 5–14 years
- Cases: clinically diagnosed asthma based on physician evaluation, supported by spirometry where feasible
- Controls: children without asthma, matched for age, sex, and geographical location
- Written informed consent from parents or guardians

Exclusion Criteria
- History of a single wheezing episode
- Requirement for mechanical ventilation
- Preterm infants requiring oxygen therapy for more than 28 days
- Congenital lung disorders

Data Collection
Data were collected using a structured questionnaire covering:
- Demographic characteristics
- Family history of asthma and allergic diseases
- Parental atopy
- Exposure to passive smoking
- Environmental allergens (dust, indoor dampness, biomass fuel, mold)
- Pet exposure
- Early feeding and weaning practices
- Frequency of respiratory tract infections

Exposure Definitions
Passive smoking is operationally defined as the presence of at least one household member who engages in daily smoking activities within the domestic environment. Environmental allergen exposure encompasses factors such as dust, smoke from biomass fuels, damp living conditions, or the visible presence of mold. Frequent respiratory infections are characterized as the occurrence of three or more physician-diagnosed infections of the lower respiratory tract within a single year.
Statistical Analysis
Data were subjected to examination through descriptive statistical methods. Odds ratios accompanied by 95% confidence intervals were derived utilizing 2×2 contingency tables. The application of Pearson's chi-square test was conducted, with a p-value threshold of <0.05 being designated as statistically significant. It is acknowledged that multivariate analysis was not executed, thereby constituting a limitation of the study.

RESULTS

A total of 200 pediatric subjects were incorporated into the investigation, consisting of 100 individuals diagnosed with asthma and 100 age- and sex-matched control participants. The distribution of evaluated risk factors and their correlations with pediatric asthma are encapsulated in Table 1, whereas the corresponding chi-square test results and their statistical significance are delineated in Table 2.As illustrated in Table 1, a familial history of asthma was documented in 71% of asthmatic children in contrast to 40% of control subjects, thereby evidencing a significant correlation with asthma (OR 1.8; 95% CI 1.3–2.5; $p < 0.0001$). Correspondingly, a history of allergic rhinitis was substantially more prevalent among the asthmatic cohort (54%) than the control group (20%), indicating a robust association with pediatric asthma (OR 2.7; 95% CI 1.8–4.0; $p < 0.0001$).Environmental exposures also revealed significant correlations. The incidence of passive smoking exposure was recorded in 51% of asthmatic children as opposed to 23% of controls, imparting a greater than two-fold elevation in asthma risk (OR 2.2; 95% CI 1.5–3.3; $p < 0.0001$). The combined exposure to passive smoking and environmental allergens was similarly significantly correlated with asthma (53% vs. 33%; OR 1.6; 95% CI 1.1–2.3; $p = 0.0067$), as outlined in Tables 1 and 2.Genetic predisposition was further corroborated by the markedly elevated prevalence of parental atopy among asthmatic cases in comparison to controls (40% vs. 20%), resulting in an odds ratio of 2.0 (95% CI 1.4–2.9; $p = 0.0034$) (Table 1).The most pronounced association identified in this investigation was associated with a history of recurrent respiratory tract infections, reported in 49% of asthmatic children compared to only 14% of control subjects. This particular factor exhibited the highest odds ratio (OR 3.5; 95% CI 2.5–5.0; p <

0.0001), signifying a considerable augmentation in asthma risk (Table 1; Table 2).In contrast, early weaning prior to four months of age did not exhibit a significant association with asthma (18% in cases vs. 16% in controls; OR 1.1; 95% CI 0.7–1.6; p = 0.8507). Likewise, exposure to domestic pets was not significantly correlated with pediatric asthma and demonstrated a non-significant inverse trend (38% vs. 45%; OR 0.8; 95% CI 0.5–1.2; p = 0.3892). These non-significant findings are recorded in Tables 1 and 2.In summary, the findings suggest that pediatric asthma within this demographic is significantly associated with genetic predisposition, allergic comorbidities, exposure to environmental tobacco smoke, and frequent respiratory infections, whereas early feeding practices and pet exposure did not yield significant associations.

Table 1. Risk Factors Associated with Childhood Asthma (n = 200)

Risk Factor	Asthmatic Group (n = 100)	Control Group (n = 100)	Odds Ratio (OR)	95% Confidence Interval (CI)
Family history of asthma	71 (71.0%)	40 (40.0%)	1.8	1.3 – 2.5
History of allergic rhinitis	54 (54.0%)	20 (20.0%)	2.7	1.8 – 4.0
Early weaning (<4 months)	18 (18.0%)	16 (16.0%)	1.1	0.7 – 1.6
Exposure to smoking **and** environmental allergens	53 (53.0%)	33 (33.0%)	1.6	1.1 – 2.3
Exposure to passive smoking	51 (51.0%)	23 (23.0%)	2.2	1.5 – 3.3
Exposure to pets at home	38 (38.0%)	45 (45.0%)	0.8	0.5 – 1.2
History of parental atopy	40 (40.0%)	20 (20.0%)	2.0	1.4 – 2.9
Frequent respiratory tract infections	49 (49.0%)	14 (14.0%)	3.5	2.5 – 5.0

Odds ratios were calculated using 2×2 contingency tables. Values are expressed as number (%). Comparison of demographic, genetic, environmental, and clinical risk factors between children with asthma (n = 100) and age- and sex-matched controls without asthma (n = 100). Data are presented as number (percentage). Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated using 2 × 2 contingency tables to estimate the strength of association between each risk factor and childhood asthma.

Table 2. Association Between Risk Factors and Childhood Asthma Using Chi-Square Test

Risk Factor	Children with Asthma (n = 100)	Children without Asthma (n = 100)	p-value
Family history of asthma	71 (71.0%)	40 (40.0%)	<0.0001
History of allergic rhinitis	54 (54.0%)	20 (20.0%)	<0.0001
Early weaning (<4 months)	18 (18.0%)	16 (16.0%)	0.8507
Exposure to smoking and environmental allergens	53 (53.0%)	33 (33.0%)	0.0067
Exposure to passive smoking	51 (51.0%)	23 (23.0%)	<0.0001
Exposure to pets at home	38 (38.0%)	45 (45.0%)	0.3892
History of parental atopy	40 (40.0%)	20 (20.0%)	0.0034
Frequent respiratory tract infections	49 (49.0%)	14 (14.0%)	<0.0001

Distribution of assessed risk factors among asthmatic and non-asthmatic children and their statistical association with childhood asthma. Pearson's chi-square test was applied to evaluate significance. A p-value < 0.05 was considered statistically significant.

DISCUSSION

This investigation elucidates that childhood asthma is significantly correlated with genetic predisposition, the presence of allergic comorbidities, exposure to environmental tobacco smoke, and recurrent respiratory infections. The outcomes align with antecedent studies that identify family history and allergic rhinitis as substantial contributors to the onset of asthma [11–14]. The emergence of passive smoking as a critical environmental risk factor is noted, effectively doubling the probability of developing asthma. It is well-established that exposure to tobacco smoke adversely affects lung maturation and exacerbates airway inflammation in pediatric populations, especially during early developmental stages. Similarly, the compounded exposure to indoor allergens and tobacco smoke further escalates the risk of asthma, thereby underscoring the cumulative impact of environmental adversities. Recurrent respiratory infections exhibit the strongest correlation with asthma; such infections may facilitate airway remodeling or signify an underlying immune susceptibility that predisposes children to asthma, corroborated by previous cohort studies [15–17]. The presence of parental atopy further substantiates the genetic and immunological underpinnings of asthma vulnerability. Conversely, early weaning and pet exposure did not reveal significant associations with asthma within this cohort. The limited evaluation of breastfeeding duration and exclusivity may elucidate the lack of observed association. Moreover, emerging evidence posits that early exposure to pets might confer a protective or neutral influence, contingent upon timing and genetic predisposition [18–19]. Notwithstanding these findings, the study is subject to significant limitations. The case–control design

inherently restricts causal interpretations. The dependence on self-reported exposure data may foster recall bias. The single-center nature of the study constrains its generalizability, and the absence of multivariate analysis limits the capacity to adjust for potential confounders such as socioeconomic status and environmental pollution.

LIMITATIONS

This single-center case–control study cannot establish causality. Exposure data were self-reported, introducing potential recall bias. The sample size limited detection of weaker associations and subgroup analyses. Absence of multivariate analysis prevented adjustment for confounding variables. Important factors such as air pollution, socioeconomic status, dietary habits, and prenatal exposures were not assessed.

CONCLUSION

Childhood asthma is significantly associated with family history, allergic rhinitis, parental atopy, exposure to tobacco smoke, and recurrent respiratory infections. Early weaning and pet exposure were not significant determinants in this population. Larger multicenter studies incorporating objective exposure measurements and multivariate analyses are required to strengthen evidence and guide preventive strategies.

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Conflict of Interest: Nil

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