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Original Article

The Influence of Environmental Pollution on the Incidence of Allergic Rhinitis in Urban Populations: A Case-Control Study

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ABSTRACT

Background: Allergic rhinitis is an inflammatory upper-airway disorder that may be aggravated by traffic emissions, particulate pollution, construction dust, and household environmental exposures. Objective: To examine the association between categorized environmental pollution exposure and allergic rhinitis among adults in Lahore, Pakistan. Methods: This case-control study included 240 adults comprising 120 clinically diagnosed allergic rhinitis cases and 120 controls. Demographic, residential, household, hereditary, and environmental exposure data were collected using a structured questionnaire. Group differences were assessed using appropriate comparative tests, and binary logistic regression was used to estimate adjusted odds ratios with 95% confidence intervals. Results: High pollution exposure was reported by 68.3% of cases and 36.7% of controls. High pollution exposure was independently associated with allergic rhinitis (adjusted OR: 3.18; 95% CI: 1.83–5.53), as were residence near a major road (adjusted OR: 2.12; 95% CI: 1.24–3.63), industrial or construction exposure (adjusted OR: 1.89; 95% CI: 1.07–3.34), indoor dust exposure (adjusted OR: 1.74; 95% CI: 1.02–2.97), and family history of allergy (adjusted OR: 2.03; 95% CI: 1.10–3.75). Passive smoking was not statistically significant. Conclusion: Categorized environmental pollution and related residential exposures were associated with higher odds of allergic rhinitis among adults in Lahore. Prospective studies using objective pollutant measurements are required. Keywords: Allergic rhinitis; air pollution; environmental exposure; particulate matter; traffic emissions; Lahore.

INTRODUCTION

Allergic rhinitis is a chronic, immunoglobulin E–mediated inflammatory disorder of the nasal mucosa characterized by sneezing, nasal itching, rhinorrhoea, and nasal obstruction, often accompanied by ocular symptoms. Although it is not generally associated with mortality, persistent or poorly controlled allergic rhinitis can impair sleep, concentration, academic and occupational performance, emotional well-being, and overall quality of life. It also commonly coexists with asthma and other allergic disorders, supporting the concept of a unified inflammatory airway and highlighting the need to consider allergic rhinitis as a clinically and socially important health condition rather than a minor seasonal complaint (1–5).

The development and exacerbation of allergic rhinitis reflect interactions between individual susceptibility and environmental exposures. Genetic predisposition, particularly a family history of

allergic disease, may increase vulnerability, whereas ambient and household exposures may initiate or intensify nasal inflammation. Outdoor air pollutants, including particulate matter with aerodynamic diameters of ≤2.5 μm and ≤10 μm, nitrogen dioxide, sulfur dioxide, carbon monoxide, and ozone, can interact directly with the nasal epithelium during inhalation. These exposures may impair epithelial-barrier function, increase oxidative stress, alter local immune responses, and facilitate allergen penetration into the nasal mucosa. Indoor dust, dampness, mold, tobacco smoke, and inadequate ventilation may further sustain or amplify inflammatory responses, particularly among individuals with an underlying allergic predisposition (6–8).

Evidence from systematic reviews, meta-analyses, and population-based studies suggests that exposure to ambient air pollution is associated with an increased occurrence of allergic rhinitis. Fine particulate matter has been identified as an important environmental exposure, although the magnitude and consistency of reported associations vary according to age, pollutant type, exposure duration, geographic setting, outcome definition, and exposure-assessment method (6,7). Studies among children have reported associations between allergic rhinitis and traffic-related air pollution, including particulate matter and nitrogen dioxide, while prospective evidence among adults has similarly suggested that long-term exposure to PM2.5, PM10, nitrogen dioxide, and nitrogen oxides may increase the likelihood of developing allergic rhinitis (9–12). Short-term fluctuations in ambient pollution have also been associated with increased outpatient attendance and worsening of allergic rhinitis symptoms, indicating that both cumulative and episodic exposures may be clinically relevant (13,14).

Traffic-related pollution is particularly important in densely populated cities because residents living near major roads may experience repeated exposure to exhaust emissions, resuspended road dust, and combustion-derived particulate matter. Previous research has demonstrated associations between traffic exposure and allergic rhinitis in both adult and paediatric populations (12,15). Industrial activity and construction may provide additional sources of fine and coarse particles, whereas household dust may combine outdoor particles with indoor allergens such as dust mites and mold. These overlapping exposures make it difficult to attribute allergic rhinitis to a single pollutant and support the use of an integrated environmental-exposure assessment when direct personal monitoring is unavailable.

Lahore represents an important setting in which to investigate these relationships because the city experiences substantial traffic congestion, construction activity, industrial emissions, seasonal crop-residue smoke, resuspended dust, and recurrent winter smog. Studies conducted in Lahore have documented elevated PM2.5 concentrations, marked seasonal variation, and the presence of chemically reactive particulate components capable of generating oxidative stress (16–22). These environmental conditions may have important implications for upper-airway health, particularly among residents who live near major roads, industrial areas, or construction zones or who experience substantial indoor dust exposure.

Despite the recognised burden of air pollution in Lahore, locally generated evidence has focused more frequently on general respiratory morbidity, particulate-matter composition, or lower-airway disease than on clinically diagnosed allergic rhinitis. Pakistani studies have described the demographic and clinical characteristics of patients with allergic rhinitis and have documented associations with impaired sleep and psychological well-being, but comparatively limited case-control evidence has examined whether environmental exposure patterns differ between affected and unaffected adults from the same urban setting (23–25). This gap limits the ability of clinicians and public-health professionals to identify locally relevant environmental risk indicators and to integrate exposure reduction into patient counselling and urban-health planning.

The present study therefore examined the association between categorized environmental pollution exposure and clinically diagnosed allergic rhinitis among adult residents of Lahore. It specifically compared cases and controls with respect to high overall pollution exposure, residence near a major road, exposure to industrial or construction environments, indoor dust, passive smoking, and family

history of allergy. It was hypothesized that adults with greater environmental pollution exposure would have higher odds of allergic rhinitis than adults with lower exposure after adjustment for relevant individual and household factors.

MATERIALS AND METHODS

A case-control study was conducted among adult residents of Lahore, Pakistan, to examine the association between environmental pollution exposure and allergic rhinitis. Participants were recruited from outpatient services of tertiary-care hospitals and ear, nose, and throat clinics serving the urban population of Lahore. Cases and controls were selected from the same underlying geographic population to improve comparability between the groups and reduce differences arising solely from place of residence or access to healthcare.

The study included 240 participants, comprising 120 cases with clinically diagnosed allergic rhinitis and 120 controls without allergic rhinitis. Cases were recruited consecutively from patients presenting to participating outpatient departments and clinics. Eligible cases were adults who had lived in Lahore for at least one year and had clinical features consistent with allergic rhinitis, including recurrent sneezing, nasal itching, watery rhinorrhoea, nasal obstruction, or associated ocular itching or watering. The diagnosis was established by an ear, nose, and throat clinician on the basis of clinical history and physical examination. Controls were recruited from adult attendants and general outpatient visitors drawn from the same urban catchment population. Eligible controls had lived in Lahore for at least one year and reported no current or previous allergic rhinitis, asthma, chronic rhinosinusitis, or other major allergic airway disease. Controls were selected to achieve group comparability with respect to age, sex, and residential area.

Participants were excluded if they had an acute upper respiratory tract infection, nasal polyps, severe obstructive deviation of the nasal septum, chronic rhinosinusitis, recent nasal surgery, long-term systemic or intranasal corticosteroid use, or substantial occupational exposure to chemicals that could independently cause chronic nasal irritation. Individuals who were not permanent residents of Lahore were also excluded because their environmental exposure could not be reasonably assigned to the study setting.

Data were collected through a structured interviewer-administered questionnaire and clinical assessment. The questionnaire recorded age, sex, residential area, duration of residence in Lahore, occupation, active and passive smoking exposure, family history of allergic disease, household dust exposure, pet exposure, biomass-fuel use, time spent outdoors, and residence or routine activity near major roads, industrial sites, or active construction areas. For cases, additional clinical information was recorded regarding sneezing, nasal itching, rhinorrhoea, nasal obstruction, ocular symptoms, symptom duration, frequency, and seasonal worsening.

The primary outcome was allergic rhinitis status, categorized as a clinically diagnosed case or a control without a history of allergic rhinitis. The principal exposure was categorized environmental pollution exposure. Exposure classification incorporated the participant's residential proximity to major traffic routes, industrial or construction environments, reported indoor dust exposure, time spent outdoors, and ambient air-quality information available for the relevant residential area. Participants with greater combined exposure to these environmental sources were classified as having high pollution exposure, whereas the remaining participants formed the lower-exposure comparison category used in the principal analysis.

Residence near a major road was treated as an indicator of repeated exposure to traffic emissions and resuspended road dust. Industrial or construction exposure represented regular residential or environmental contact with emissions or dust generated by industrial activity or active construction. Indoor dust exposure referred to recurrent household exposure reported by the participant. Passive

smoking exposure was defined as regular exposure to tobacco smoke generated by another household member or close contact. Family history of allergy was recorded when allergic rhinitis, asthma, eczema, or another clinically recognised allergic condition was reported in a first-degree relative.

Ambient air-quality information was reviewed to provide contextual support for the environmental-exposure classification. The pollutants considered included PM2.5, PM10, nitrogen dioxide, sulfur dioxide, carbon monoxide, ozone, and the overall air quality index. Because participant-level pollutant concentrations were not available for every individual, these indicators were used as contextual environmental information rather than as independently quantified personal exposure measurements. Accordingly, the primary statistical analysis evaluated categorized pollution exposure and specific environmental indicators rather than attributing individual disease status to measured concentrations of a single pollutant.

The planned sample comprised equal numbers of cases and controls to improve statistical efficiency and facilitate group comparison. The sample size was based on a case-control framework using a 95% confidence level, 80% statistical power, and an anticipated difference in environmental pollution exposure between cases and controls. Cases were enrolled through non-probability consecutive sampling, while eligible controls were recruited from the same healthcare settings and urban catchment areas.

Potential sources of selection and information bias were addressed by recruiting cases and controls from the same geographic population, applying predefined eligibility criteria, using the same structured questionnaire for both groups, and collecting exposure information through a standardized procedure. Group comparability was assessed for age and sex. Variables considered as potential confounders included age, sex, family history of allergy, passive smoking, indoor dust exposure, and socioeconomic characteristics. The interpretation of the exposure estimates was restricted to associations because the retrospective case-control design did not establish temporal or causal relationships between environmental exposure and allergic rhinitis.

Data were entered and analysed using SPSS 26.0. Continuous variables were summarized using means and standard deviations, while categorical variables were presented as frequencies and percentages. Age was compared between cases and controls using the independent-samples t test. Categorical characteristics and environmental exposures were compared using the chi-square test. Crude exposure distributions were initially examined between the two groups, after which binary logistic regression was used to estimate adjusted odds ratios and 95% confidence intervals for the association of allergic rhinitis with high pollution exposure, residence near a major road, industrial or construction exposure, indoor dust exposure, family history of allergy, and passive smoking exposure. Allergic rhinitis status was entered as the binary dependent variable. Statistical adjustment was undertaken to reduce confounding by relevant individual and environmental characteristics. A two-sided p-value of less than 0.05 was considered statistically significant.

Data were reviewed for completeness and internal consistency before analysis. Frequencies, ranges, and denominators were checked against the original records, and all analyses were based on the available complete observations. Results were reported as associations rather than causal effects, with odds ratios interpreted in relation to their corresponding confidence intervals and the limitations of clinic-based recruitment and categorized exposure assessment.

RESULTS

A total of 240 participants were included in the analysis, comprising 120 adults with clinically diagnosed allergic rhinitis and 120 controls without allergic rhinitis. The two groups had comparable age and sex distributions. The mean age was 34.7 ± 11.2 years among cases and 33.9 ± 10.8 years among controls,

corresponding to a mean difference of 0.8 years (95% CI: −2.0 to 3.6; p = 0.580). Men constituted 55.8% of cases and 52.5% of controls (p = 0.610).

Table 1. Demographic and Environmental Characteristics of Cases and Controls

Characteristic	Cases, n = 120	Controls, n = 120	p-value
Age, years, mean ± SD	34.7 ± 11.2	33.9 ± 10.8	0.580
Male sex, n (%)	67 (55.8)	63 (52.5)	0.610
Residence near a major road, n (%)	85 (70.8)	51 (42.5)	<0.001
High pollution exposure, n (%)	82 (68.3)	44 (36.7)	<0.001
Industrial or construction exposure, n (%)	54 (45.0)	29 (24.2)	0.001
Indoor dust exposure, n (%)	61 (50.8)	39 (32.5)	0.004
Family history of allergy, n (%)	42 (35.0)	24 (20.0)	0.009
Passive smoking exposure, n (%)	38 (31.7)	30 (25.0)	0.250

SD: standard deviation. The independent-samples t test was used for age, and the chi-square test was used for categorical variables.

Environmental exposures were consistently more frequent among allergic rhinitis cases than controls. High pollution exposure was identified in 82 cases (68.3%) compared with 44 controls (36.7%; p < 0.001). Similarly, 85 cases (70.8%) lived near a major road compared with 51 controls (42.5%; p < 0.001). Industrial or construction exposure was reported by 54 cases (45.0%) and 29 controls (24.2%; p = 0.001), while indoor dust exposure was reported by 61 cases (50.8%) and 39 controls (32.5%; p = 0.004). A family history of allergy was also more frequent among cases than controls, at 35.0% and 20.0%, respectively (p = 0.009). Passive smoking exposure was reported by 31.7% of cases and 25.0% of controls, but the difference was not statistically significant (p = 0.250).

Table 2. Crude and Adjusted Associations Between Environmental Exposures and Allergic Rhinitis

Exposure	Crude OR	95% CI	p-value	Adjusted OR	95% CI	p-value
High pollution exposure	3.73	2.18–6.36	<0.001	3.18	1.83–5.53	<0.001
Residence near a major road	3.29	1.92–5.61	<0.001	2.12	1.24–3.63	0.006
Industrial or construction exposure	2.57	1.48–4.46	0.001	1.89	1.07–3.34	0.028
Indoor dust exposure	2.15	1.27–3.62	0.004	1.74	1.02–2.97	0.041
Family history of allergy	2.15	1.20–3.86	0.009	2.03	1.10–3.75	0.023
Passive smoking exposure	1.39	0.79–2.44	0.252	1.32	0.73–2.39	0.350

CI: confidence interval; OR: odds ratio. The absence of each listed exposure constituted the reference category. Crude ORs and confidence intervals were calculated directly from the reported 2×2 frequencies. Adjusted estimates were retained from the reported binary logistic-regression analysis.

High pollution exposure demonstrated the largest association with allergic rhinitis. Before adjustment, participants classified as having high pollution exposure had 3.73 times the odds of allergic rhinitis compared with those in the lower-exposure category (95% CI: 2.18–6.36; p < 0.001). The association remained statistically significant after adjustment, although its magnitude was attenuated to an adjusted OR of 3.18 (95% CI: 1.83–5.53; p < 0.001).

Residence near a major road was associated with more than threefold higher crude odds of allergic rhinitis (OR: 3.29; 95% CI: 1.92–5.61; p < 0.001). After adjustment, the association remained significant, with an adjusted OR of 2.12 (95% CI: 1.24–3.63; p = 0.006). Industrial or construction exposure was similarly associated with allergic rhinitis in both the crude analysis (OR: 2.57; 95% CI: 1.48–4.46; p = 0.001) and the adjusted model (adjusted OR: 1.89; 95% CI: 1.07–3.34; p = 0.028).

Indoor dust exposure was associated with approximately twofold higher crude odds of allergic rhinitis (OR: 2.15; 95% CI: 1.27–3.62; p = 0.004). The adjusted estimate remained statistically significant but was reduced to 1.74 (95% CI: 1.02–2.97; p = 0.041), with the lower confidence limit close to the null value. A family history of allergy was associated with increased odds in both the crude analysis (OR: 2.15; 95% CI: 1.20–3.86; p = 0.009) and the adjusted model (adjusted OR: 2.03; 95% CI: 1.10–3.75; p = 0.023).

Passive smoking exposure was associated with a modest increase in the estimated odds of allergic rhinitis, but the association was not statistically significant in either the crude analysis (OR: 1.39; 95% CI: 0.79–2.44; p = 0.252) or the adjusted model (adjusted OR: 1.32; 95% CI: 0.73–2.39; p = 0.350). The relatively wide confidence interval indicates that the available data were compatible with both little or no association and a potentially clinically relevant increase in odds.

Overall, the adjusted analysis identified high pollution exposure, residence near a major road, industrial or construction exposure, indoor dust exposure, and family history of allergy as independently associated with allergic rhinitis. High pollution exposure remained the most prominent reported correlate, while passive smoking did not demonstrate an independent statistically significant association in the available sample.

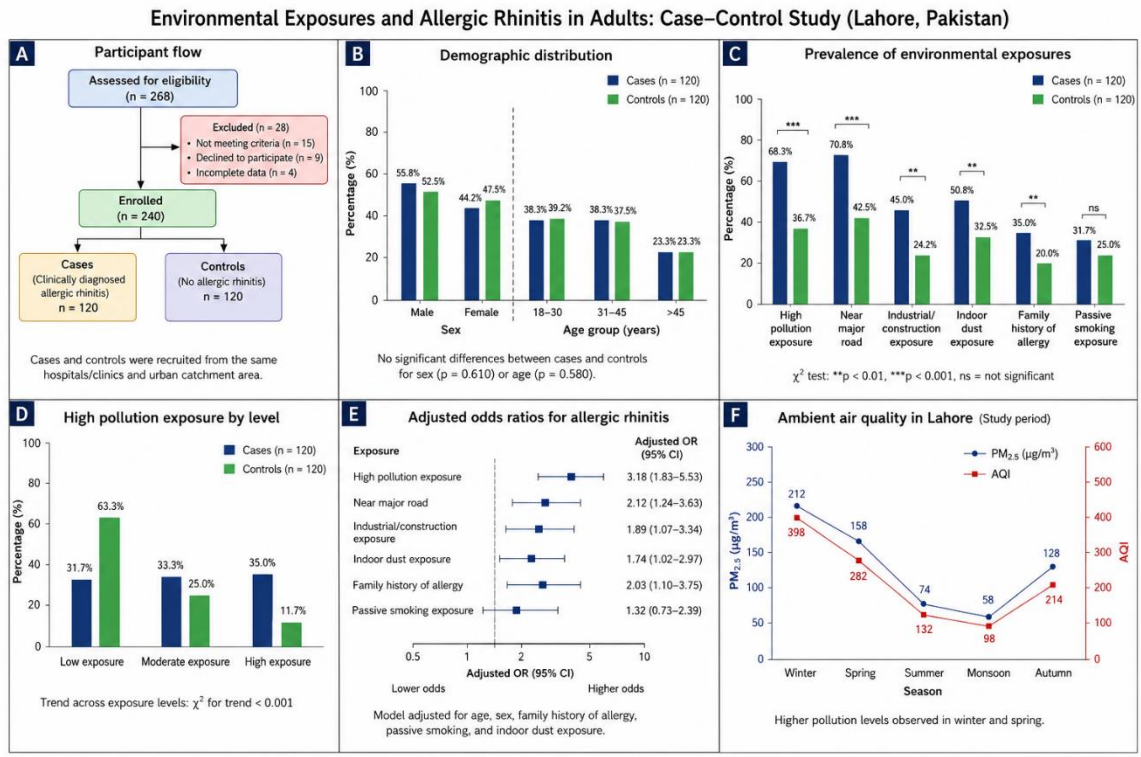

Figure 1 The multipanel figure summarizes participant allocation, demographic comparability, environmental exposure patterns, adjusted associations, and seasonal air-quality variation. Cases showed substantially higher exposure to overall pollution, major roads, industrial or construction activity, indoor dust, and family history of allergy than controls, while high pollution exposure demonstrated the strongest adjusted association with allergic rhinitis (adjusted OR 3.18, 95% CI 1.83–5.53); passive smoking was not statistically significant.

DISCUSSION

This case-control study identified a consistent association between categorized environmental pollution exposure and allergic rhinitis among adults recruited from healthcare settings in Lahore. High pollution exposure was reported by 68.3% of cases compared with 36.7% of controls and was associated with more than threefold higher adjusted odds of allergic rhinitis (adjusted OR: 3.18; 95% CI: 1.83–5.53). Residence near a major road, industrial or construction exposure, indoor dust exposure, and family history of allergy also remained independently associated with allergic rhinitis after adjustment. In contrast, passive smoking exposure showed a modest increase in the estimated odds but was not statistically significant. These findings suggest that allergic rhinitis in this population was associated with a combination of ambient, residential, household, and hereditary factors rather than with a single isolated exposure.

The observed association between high pollution exposure and allergic rhinitis is consistent with evidence from systematic reviews and meta-analyses examining ambient air pollution and allergic

upper-airway disease. Li et al. reported that exposure to several outdoor air pollutants was associated with an increased risk of allergic rhinitis, although the strength of association varied by pollutant, population, and exposure-assessment method (6). Similarly, a meta-analysis focused on fine particulate matter supported an association between PM2.5 exposure and allergic rhinitis (7). The adjusted estimate in the present study remained substantial after accounting for relevant covariates, although it should be interpreted as an association with a composite exposure category rather than as a pollutant-specific effect. Direct comparisons with studies using measured PM2.5 or nitrogen dioxide concentrations should therefore be made cautiously.

The association with residence near a major road further supports the possible relevance of traffic-related environmental exposure. Participants living near major roads had an adjusted OR of 2.12 for allergic rhinitis compared with those who did not report this exposure. Traffic environments may involve repeated exposure to exhaust emissions, nitrogen oxides, fine and ultrafine particles, tire and brake wear, and resuspended road dust. Previous studies have similarly reported associations between traffic-related pollution and allergic rhinitis in adults and children (12,15). However, the present study did not quantify residential distance, traffic density, or individual pollutant concentrations, and the observed association may also reflect correlated characteristics such as socioeconomic conditions, housing quality, occupational mobility, noise, or reduced indoor-air quality.

Industrial or construction exposure was independently associated with allergic rhinitis, with an adjusted OR of 1.89. This finding is relevant to Lahore, where construction activity, industrial emissions, fuel combustion, and resuspended dust contribute to ambient particulate pollution. Local environmental studies have documented elevated particulate-matter concentrations and chemically reactive components in Lahore's air, including compounds capable of promoting oxidative activity (17–22). Nevertheless, the current study did not directly measure the composition, dose, or duration of industrial and construction-related exposure. The result should therefore be interpreted as an association with reported environmental proximity rather than proof that a particular industrial contaminant caused allergic rhinitis.

Several biological pathways may plausibly explain the associations observed in this study. Inhaled particulate matter and gaseous pollutants may impair the integrity of the nasal epithelial barrier, generate reactive oxygen species, activate inflammatory signalling pathways, and increase mucosal responsiveness to aeroallergens. Pollutants may also act as adjuvants that intensify immunoglobulin E–mediated responses in susceptible individuals. These mechanisms are consistent with current clinical and environmental literature describing allergic rhinitis as an inflammatory airway disorder influenced by both allergen exposure and non-allergic irritants (1–3,8). However, oxidative stress, epithelial injury, allergen sensitization, and inflammatory biomarkers were not measured in the present study; these mechanisms therefore remain explanatory hypotheses rather than direct study findings.

Indoor dust exposure was reported by 50.8% of cases and 32.5% of controls and remained associated with allergic rhinitis after adjustment (adjusted OR: 1.74; 95% CI: 1.02–2.97). Indoor dust may contain house-dust mites, fungal particles, animal-derived allergens, textile fibres, combustion products, and particulate matter transported from outdoor environments. In houses located near busy roads or construction sites, outdoor particles may enter through windows, doors, ventilation systems, footwear, and clothing. The confidence interval for indoor dust exposure was relatively close to the null value, indicating greater uncertainty around the magnitude of this association. Moreover, indoor dust was based on reported exposure rather than standardized household sampling, allergen measurement, or environmental inspection.

A family history of allergy was associated with approximately twofold higher adjusted odds of allergic rhinitis. This result is consistent with the established role of hereditary susceptibility in allergic disease. Individuals with a family history of allergic rhinitis, asthma, or eczema may have an underlying predisposition toward immunoglobulin E–mediated sensitization and mucosal hyperresponsiveness

(1,5). The simultaneous associations of family history and environmental exposure support a multifactorial model in which inherited susceptibility and environmental triggers may both contribute to disease occurrence. The current analysis did not include a formal interaction term between family history and pollution exposure, however, and it cannot establish that the effect of pollution differed according to genetic or familial susceptibility.

Passive smoking exposure was more frequent among cases than controls, but the adjusted association was not statistically significant (adjusted OR: 1.32; 95% CI: 0.73–2.39). The confidence interval was wide and included both little or no association and a potentially meaningful increase in odds. The absence of statistical significance should therefore not be interpreted as evidence that passive smoking is harmless. The estimate may have been affected by limited statistical power, exposure misclassification, variation in the duration or intensity of exposure, or correlation with other household and environmental factors. More detailed assessment using household smoking frequency, number of smokers, duration of exposure, and objective biomarkers would be necessary to evaluate this relationship more precisely.

The attenuation of several odds ratios after adjustment indicates that the measured exposures shared some degree of confounding or overlap. For example, the crude OR for residence near a major road decreased from 3.29 to an adjusted OR of 2.12, while the estimate for industrial or construction exposure decreased from 2.57 to 1.89. Such attenuation is expected when environmental and household exposures cluster within the same individuals or neighbourhoods. Residents near major roads may also experience higher indoor dust, greater outdoor particulate exposure, lower-quality housing, and different occupational or socioeconomic conditions. Because the exact regression covariates and model diagnostics were not available in the supplied record, the independence and stability of the adjusted estimates should be confirmed from the original statistical output.

The findings have potential clinical and public-health relevance. Clinicians assessing patients with persistent allergic rhinitis may benefit from asking about residential proximity to major roads, construction or industrial activity, indoor dust, household smoking, seasonal worsening, and family history of allergy. Exposure reduction may complement pharmacological management, although the present study did not evaluate the effectiveness of any preventive intervention. At the population level, the findings support continued attention to traffic emissions, construction-dust control, industrial pollution, indoor ventilation, and public risk communication during high-pollution periods. These implications should be regarded as evidence-informed recommendations rather than direct intervention effects demonstrated by this study.

This study should be interpreted in light of several limitations. First, cases and controls were recruited from healthcare settings, which may have introduced selection bias and may restrict generalizability to the wider adult population of Lahore. Controls recruited from attendants and general outpatient visitors may differ from cases in healthcare-seeking behaviour, occupation, socioeconomic circumstances, and environmental exposure. Second, environmental exposures were categorized using reported residential and household characteristics rather than personal monitoring or validated geospatial exposure models, creating the possibility of non-differential or differential exposure misclassification. Third, the retrospective case-control design limits assessment of temporality, and the study cannot determine whether exposure preceded the onset of allergic rhinitis. Fourth, recall bias may have occurred if cases were more likely than controls to report pollution, dust, or traffic exposure. Fifth, residual confounding may remain because pollen exposure, mold, household ventilation, active smoking, occupation, socioeconomic status, residential mobility, cooking fuel, and seasonal recruitment were not fully reported in the available analysis.

Additional limitations include the absence of pollutant-specific participant-level concentrations, lack of objective allergic sensitization testing, and uncertainty regarding the matching strategy and regression specification. The study also did not report formal interaction analyses, model-fit statistics, multicollinearity assessment, or sensitivity analyses using alternative exposure definitions. These

limitations mean that the adjusted odds ratios should not be interpreted as causal effects or as estimates attributable to individual pollutants such as PM2.5 or nitrogen dioxide. Future studies should use prospective or population-based designs, standardized diagnostic criteria, validated questionnaires, geocoded residential histories, repeated seasonal exposure measurements, personal or area-level pollutant monitoring, and prespecified multivariable and interaction analyses.

Despite these limitations, the study contributes locally relevant evidence regarding the environmental context of allergic rhinitis in Lahore. The consistent direction of associations across high pollution exposure, major-road residence, industrial or construction exposure, and indoor dust suggests that multiple environmental sources may contribute to the observed burden of allergic rhinitis. The findings provide a basis for more rigorous longitudinal research integrating clinical diagnosis with objective exposure assessment and spatially resolved air-quality data.

CONCLUSION

Among adults recruited from healthcare settings in Lahore, high categorized pollution exposure, residence near a major road, industrial or construction exposure, indoor dust exposure, and family history of allergy were independently associated with higher odds of clinically diagnosed allergic rhinitis, while passive smoking was not statistically significant after adjustment. High pollution exposure demonstrated the largest adjusted association, but the findings should be interpreted as non-causal because exposure was categorized, participant-level pollutant concentrations were unavailable, and the case-control design could not establish temporality. The results support incorporating environmental-exposure histories into clinical assessment and justify future population-based studies using standardized allergic-rhinitis definitions, geocoded residential data, seasonal monitoring, and objective measurement of individual pollutants.

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