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Air Pollution and Immune Disorders in Women from Urban Punjab: A Clinical and Epidemiological Study

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Abstract

Urban Punjab, particularly cities like Ludhiana, Amritsar, and Jalandhar, experiences some of the highest air pollution levels in India due to industrial activity, vehicular emissions, and agricultural practices. Despite this, gender-specific health impacts—especially related to immune function—remain inadequately studied. Objective of the study is to investigate the relationship between air pollution exposure and immune-related disorders in women residing in urban Punjab. A cross-sectional study was conducted from 2022 to 2023, involving 450 women aged 18–60 years from three major cities in Punjab. Air quality data ($PM_{2.5}$, PM_{10}, and NO_2) were compared with WHO guidelines. Clinical evaluations identified allergic conditions, autoimmune diseases, and recurrent infections. Biomarkers including IgE, C-reactive protein (CRP), and interleukin-6 (IL-6) were measured. Statistical analysis was used to assess associations and odds ratios (ORs). Results show that air pollution levels were 5–10 times above WHO recommendations, with 98–100% of readings exceeding safe thresholds. Health assessments revealed: (a) 32% had allergic conditions, (b) 20% had autoimmune disorders, (c) 16% experienced recurrent infections. Significant associations were observed for nutritionally modified autoimmune hypothyroidism with: (a) Allergies (OR = 2.5), (b) Autoimmune disorders (OR = 1.8), (c) Infections (OR = 1.6). $PM_{2.5}$ levels were positively correlated with elevated IgE (250 ± 45 IU/mL), CRP (6.2 ± 1.8 mg/L), and IL-6 (12.5 ± 3.1 pg/mL), indicating systemic inflammation. NO_2 was associated with raised IgE but not with generalized inflammation. It is concluded that high levels of air pollution in urban Punjab are significantly associated with immune disorders in women, particularly those involving systemic inflammation. Immediate mitigation efforts and policies incorporating gender-sensitive health frameworks are urgently needed to address these findings.

Keywords: Air pollution, immune disorders, women's health, particulate matter, Punjab, biomarkers, epidemiology

INTRODUCTION

The last two decades have seen increased economic activities, along with the growth of urban centers in Punjab, India, adversely affecting the air quality of the region. Major contributors to the pollution include exhaust fumes from vehicles, industrial waste, burning of agricultural waste, and construction activities [1]. The major pollutants are particulate matter ($PM_{2.5}$ and PM_{10}), nitrogen dioxide (NO_2), and sulfur dioxide (SO_2), which are associated with chronic inflammation, oxidative damage, and immune system disruption [2]. Women, who are routinely exposed to indoor and outdoor pollutants, in most cases, carry an additional risk of immune dysfunction disorders like allergies and autoimmune disorders or recurrent infections.

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There is abundant literature documenting the associations of air pollution with respiratory and cardiovascular disorders; however, the literature on the immune system correlates, especially with respect to gender, is scarce [3]. This understanding becomes imperative in Punjab, India, where cultural and occupational activities heighten women's risks of exposure to both indoor and outdoor pollution. It is this relationship that this study seeks to analyze the clinical and epidemiological correlations of air pollution and immune dysfunction in women in urban Punjab with environmental data and biomarkers.

METHODOLOGY

Study Design and Population

A cross-sectional study was undertaken in three major urban centers of Punjab (Ludhiana, Amritsar, Jalandhar) from January 2022 to December 2023. The study targeted a total of 450 women between 18 and 60 years of age and enrolled them randomly from the outpatient departments of tertiary care hospitals. Patients with a history of smoking, any chronic illness like diabetes or cancer, or having occupational exposure to chemicals were not included in the study.

Data Collection

Exposure Assessment

- *Air Quality Data:* Levels of $PM_{2.5}$, PM_{10}, NO_2 and SO_2 were collected from government monitoring stations. Averaged values for a three-year period (2020–2023) were calculated.
- *Residential Proximity:* The participants' addresses were geocoded to estimate longitude and latitude with regard to relevant highways, industries and construction sites, using GIS mapping.
- *Questionnaire:* Self-reported data related to exposure to indoor pollutants such as cooking fuels, duration of residency and related metrics were gathered.

Clinical Evaluation

- *Blood Tests:* Levels of IgE, C-reactive protein (CRP) and the following cytokines: IL-6 and TNF-α were evaluated.
- *Immune Disorders:* Allergic conditions (rhinitis, asthma), autoimmune disorders (rheumatoid arthritis, lupus), and recurrent infections (respiratory, urinary) were validated through the medical history files.

Covariates

Age, social economic status (SES), body mass index (BMI) and the diet pattern were captured.

Statistical Analysis

Evaluation of association was conducted using chi-square tests, logistic regression models (accounting for covariates), and descriptive statistics. ORs and 95% CIs were estimated through SPSS v26.

RESULTS

The demographic data of the 450 participants suggests that they were representative of women in their reproductive and post-reproductive ages with a mean calculated age was 35.2 years (±8.6) (Table 1).

Table 1. Demographic characteristics of participants (N=450).

Variable	Category	Frequency (%)
Age (Mean ± SD)	35.2 ± 8.6 years	-
	Middle	225 (50%)
	High	113 (25%)
BMI	Underweight (<18.5)	45 (10%)
	Normal (18.5–24.9)	270 (60%)
	Overweight (≥25)	135 (30%)

The distributions of socioeconomic status (SES) were balanced at 25% low income, 50% middle, and 25% high income, reflecting the economic stratification of urban Punjab. Analysis of body mass index (BMI) showed that 60% of participants were within the normal range (BMI: 18.5–24.9 kg/m²), 30% were categorized as overweight (BMI ≥25), and 10% as underweight (BMI <18.5). This distribution indicates an increased risk of malnutrition and highlights the fact that BMI needs to be treated as a potential confounding factor in studies analyzing immune health for the reasons that both underweight and overweight individuals may be more prone to ailments stemming from pollution [1].

Severe degradation of air quality was observed in the study areas through particulate matter (PM) concentration which was well beyond the limits established by the WHO. The average $PM_{2.5}$ and PM_{10} concentrations were 75 and 150 μg/m³, respectively, which is approximately 5 to 10 times greater than the limits set by WHO (15 and 45 μg/m³). Alarmingly, 98–100% of these measurements were beyond the limits indicating widespread exposure to dangerous levels of particulates. NO_2 levels (40 μg/m³) also exceeded the limit set by WHO (25 μg/m³) in 82% of cases and were likely due to road traffic emissions. On the other hand, sulfur dioxide concentrations (SO_2) had a value of 15 μg/m³, which remains below the WHO threshold limit of 40 μg/m³, indicating either stricter industrial regulation or cleaner fuels employed (Table 2). These noted levels of chronic PM and NO_2 exposure pose significant concerns, particularly for women, due to the associated respiratory and immune dysfunction risk patterns [1].

The prevalence of immune disorders among participants was substantial, with the most common being allergies at 32%, followed by autoimmune diseases at 20% and recurrent infections at 16%. After controlling for age, socioeconomic status, and BMI, women exposed to high pollution levels demonstrated significantly increased risk: allergies exhibited the highest correlation with exposure (odds ratio (OR) = 2.5). This indicates that affected individuals were 2.5 times more likely to develop allergies compared to individuals living in a cleaner environment. There was also a notable association with autoimmune diseases (OR = 1.8) and recurrent infections (OR = 1.6), but to a lesser extent. The precision of these estimates is reinforced by narrow confidence intervals—for example, 1.6 to 3.9 for allergies. This supports relevant data suggesting that $PM_{2.5}$, among other pollutants, triggers immune dysregulation, likely increasing the risk for chronic inflammation and hypersensitivity in vulnerable populations [1]. The gradient in ORs indicates that particulate matter exposure may drive stronger responses in allergic outcomes than other immune responses (Table 3).

Strongly correlated were the exposure to $PM_{2.5}$ and PM_{10} particulates with elevated immune biomarkers. Women with exposure to $PM_{2.5}$ had the highest mean serum IgE (250 ± 45 IU/mL) as well as increased C-reactive protein (6.2 ± 1.8 mg/L) and Interleukin-6 (12.5 ± 3.1 pg/mL) suggesting systemic inflammation.

Table 2. Air pollution levels in study areas.

Pollutant	Mean Concentration	WHO Guideline	% Exceeding WHO Limits
$PM_{2.5}$	75 μg/m³	15 μg/m³	98%
PM_{10}	150 μg/m³	45 μg/m³	100%
NO_2	40 μg/m³	25 μg/m³	82%
SO_2	15 μg/m³	40 μg/m³	10%

Table 3. Prevalence of immune disorders.

Disorder	Frequency (%)	Adjusted OR (95% CI)
Allergies	144 (32%)	2.5 (1.6–3.9)*
Autoimmune Diseases	90 (20%)	1.8 (1.1–3.0)*
Recurrent Infections	72 (16%)	1.6 (1.0–2.6)*
*Adjusted for age, SES, and BMI; p<0.05.		

Table 4. Association between pollutants and immune markers.

Pollutant	IgE (IU/mL)	CRP (mg/L)	IL-6 (pg/mL)
$PM_{2.5}$	250 ± 45*	6.2 ± 1.8*	12.5 ± 3.1*
PM_{10}	230 ± 40*	5.8 ± 1.6*	11.0 ± 2.8*
NO_2	210 ± 35*	4.5 ± 1.2	9.5 ± 2.4
*Significant elevation compared to controls ($p<0.05$).			

Exposure to PM_{10} similarly increased IgE levels (230 ± 40 IU/mL) alongside inflammation mediators, albeit to a lesser degree than $PM_{2.5}$. NO_2 was associated with higher IgE levels (210 ± 35 IU/mL) although there was no change in CRP or IL-6, indicating that NO_2 is an underlying factor for triggering allergic responses, but not for inflammation responses. These discoveries highlight fine particulate matter ($PM_{2.5}$) as a possible substantial contributor to allergy and inflammation-driven immune response pathways due to its capacity of penetrating deep into lung tissues and bloodstream [1]. The absence of SO_2 data suggests little relevance to immune activation in this group. There was a strong correlation between $PM_{2.5}$ and PM_{10} particulates with elevated markers of IgE and inflammation, indicating immune activation (Table 4).

DISCUSSION

The investigation indicates concerning links to air pollution and immune system diseases for women residing in urban Punjab. Airborne pollutants with the greatest impact in the region included $PM_{2.5}$ and PM_{10} as they measurably surpassed WHO recommendations by more than 10 folds. These levels are considerably greater than those documented in other Indian cities such as Delhi [4], presumably as a consequence of Punjab's distinctive combination of crop residue burning, industrial activity, and motor vehicle emissions. High correlational values between $PM_{2.5}$ and heightened IgE levels validate the association between fine particulates and allergic responses mediated by Th2 cells [5]. Due to its small diameter, $PM_{2.5}$ can gain access to the deep alveoli region of lungs where it can induce oxidative stress and cytokine production, such as IL-6 and CRP, thereby aggravating chronic inflammation and increasing the risk of autoimmune diseases [3]. Noteworthy, autoimmune diseases yielded weaker correlations (OR=1.8) than allergies (OR=2.5). This may illustrate different pathogenic mechanism modulating the correlation; while allergens can directly engage with mast cells, autoimmune mechanisms demand intricate gene-environment interplay across protracted latency duration [6]. The high incidence of recurrent infections (16%) infers a state of pollution driven immunosuppression most likely via disrupted mucosal immunity or neutrophil activity [7].

The immune inactivation symptoms contrasting with other findings from high coal use areas, like the ones from Chen et al. study [8], suggests that SO_2 emissions and controls in Punjab such as burning cleaner fuels in industries, does tend to mitigate the adverse effect. On the other hand, congested traffic tends to be a controllable negative contributor due to the elevation of lower respiratory contaminants such as NO_2 and its resultant impact on IgE [9, 10].

LIMITATIONS

Dependence on fixed location monitors may lead to overestimation of exposure range at the individual level. Self-reported data on indoor pollution may suffer from memory lapse. Cross-sectional data do not allow for relationships for causal reasoning.

CONCLUSION

This study strongly asserts that air pollution, and more so $PM_{2.5}$, exponentially increases the risk of immune disorders for women in urbanized regions of Punjab. Increased prevalence of allergy and inflammatory indicators suggest that immediate policy action is required to control emissions from agriculture, industrial activities, and transportation. Public health policy needs to focus on women by way of increasing access to fresh air indoors, clean subsidized fuel-less cooking, and through health system education on screened pollutants. There is a need for long-term research to establish causative

relations between pathways and measure results of these proposed interventions. Prevention of vulnerable demographic groups from exposure to air pollution is not only an environmental responsibility but also a step towards creating fairness in women's health.

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