Journal of Carcinogenesis
Covering Genesis to Therapeutics
Journal of Carcinogenesis, ISSN: 1477-3163
2025; Vol 24: Issue 10s, https://carcinogenesis.com/

Air Pollution Exposure and the Development of Asthma in School-Aged Children

Sarmad Abdul Rehman Khan[1], Zakirullah[2], Muhammad Usman[3], Saba Wamiq[4], Sajjad Naseer[5], Zainab Asghar[6]

[1]Consultant Pulmonologist, Department of Pulmonology, Nishtar Medical University and Hospital, Multan, Pakistan

[2]Children Specialist, Tehsil Headquarters Hospital Samerbagh District, Dir Lower KPK, Pakistan

[3]Senior Registrar Pediatrics Department, Children's Hospital, Lahore, Pakistan

[4]Assistant Professor, Department of Pediatric Medicine, Multan Medical and Dental College /Ibn-e- Siena Hospital and Research Institute, Multan, Pakistan

[5]Assistant Professor, Department: Department of Pulmonology, PAF Hospital Unit 2, Islamabad, Pakistan

[6]Lahore College for Women University Lahore, Pakistan

Corresponding author:

Zainab Asghar,

Lahore College for Women University Lahore, Pakistan

Email:ID: zainabasghar4343@gmail.com

ABSTRACT

Background: Air pollution has become a critical global health concern, particularly affecting children due to their developing respiratory systems and higher susceptibility to environmental toxins.

Objective: This study aimed to assess the relationship between exposure to ambient air pollutants and the development of asthma among school-aged children.

Methods: This cross-sectional analytical study was conducted at Nishtar Medical University and Hospital Multan from May 2023 to May 2024. It included 250 school-aged children (6–14 years) from urban and suburban regions with varying levels of air pollution. Participants were categorized into high-, moderate-, and low-exposure groups based on average concentrations of $PM_{2.5}$, PM_{10}, NO_2, SO_2, and O_3 recorded near their schools. Data were collected using structured questionnaires, clinical evaluations, and spirometry testing following GINA criteria.

Results: Among the 250 participants, 63 (25.2%) were diagnosed with asthma. The prevalence of asthma was significantly higher in high-exposure areas (38.8%) compared to moderate (22.2%) and low-exposure (13.3%) zones ($p < 0.001$). Mean lung function values were markedly lower in high-exposure zones ($FEV_1 = 78.2 \pm 11.5\%$, $FVC = 82.6 \pm 10.3\%$) compared to low-exposure zones ($FEV_1 = 92.4 \pm 9.7\%$, $FVC = 93.7 \pm 8.1\%$). Multivariate regression analysis showed that children in high-exposure areas had **3.21 times higher odds** (95% CI: 1.75–5.89, $p < 0.001$) of developing asthma compared to those in low-exposure areas. Significant negative correlations were observed between pollutant levels ($PM_{2.5}$, NO_2) and lung function ($r = -0.56$ and -0.52, respectively; $p < 0.001$).

Conclusion: It is concluded that exposure to elevated levels of air pollutants, especially $PM_{2.5}$ and NO_2, significantly increases the risk of asthma and reduces pulmonary function in school-aged children. Both outdoor and indoor pollutants contribute synergistically to respiratory morbidity.

Keywords: Air pollution, Asthma, School-aged children, Particulate matter, Lung function, Environmental exposure.

How to Cite: Sarmad Abdul Rehman Khan, Zakirullah, Muhammad Usman, Saba Wamiq, Sajjad Naseer, Zainab Asghar, (2025) Air Pollution Exposure and the Development of Asthma in School-Aged Children Journal of Carcinogenesis, Vol.24, No.10s, 550-556

1. INTRODUCTION

Air pollution has become an escalating global concern, posing one of the greatest threats to human health in the 21st century. Among its numerous health consequences, respiratory vehicular emissions, air quality has deteriorated drastically in many regions. Pollutants such as particulate matter ($PM_{2.5}$ and PM_{10}), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), ozone (O_3),.

carbon monoxide (CO), and volatile organic compounds (VOCs) are the primary culprits behind this environmental hazard [2]. Chronic exposure to these pollutants has been linked to respiratory inflammation, oxidative stress, and immune dysfunction all of which are key contributors to the development and exacerbation of asthma [3]. Asthma is a chronic inflammatory airway disease characterized by reversible airflow obstruction, bronchial hyperresponsiveness, and variable respiratory symptoms such as wheezing, coughing, and dyspnea [4]. It remains one of the most prevalent chronic diseases in children worldwide. According to the World Health Organization (WHO), more than 262 million people suffer from asthma globally, and it is a leading cause of chronic illness among children. The disease often begins early in life and can persist into adulthood, resulting in a significant burden on healthcare systems and reduced quality of life [5]. Environmental factors, particularly air pollution, have been shown to act both as triggers for asthma exacerbations and as potential contributors to its onset in previously healthy individuals.

School-aged children (typically between 5 and 14 years old) represent a particularly high-risk group for pollution-related respiratory illnesses. Their lungs are still in the process of growth and maturation, which makes their airways more susceptible to inflammatory and structural damage [6]. Additionally, children inhale a greater volume of air relative to their body weight compared to adults, increasing their exposure to airborne toxins. Behavioral patterns such as spending more time outdoors during play or commuting to school further amplify this risk. In urban environments, where traffic emissions are the predominant source of air pollution, school zones located near highways or industrial areas expose children to high concentrations of NO_2 and ultrafine particles that can penetrate deeply into the lower respiratory tract [7]. The biological mechanisms underlying the association between air pollution and asthma development are multifaceted. Inhaled pollutants can initiate oxidative stress and local airway inflammation, leading to epithelial cell injury, mucosal edema, and recruitment of inflammatory cells like eosinophils and neutrophils [8]. These processes release cytokines and chemokines such as interleukin-4 (IL-4), interleukin-5 (IL-5), and tumor necrosis factor-alpha (TNF-α), which promote allergic sensitization and airway remodeling. Long-term exposure can lead to permanent structural changes in the airways, including thickening of the basement membrane and increased smooth muscle mass, predisposing children to chronic asthma and reduced pulmonary function [9]. Furthermore, pollutants such as diesel exhaust particles have been found to act as adjuvants that enhance the body's allergic responses to common aeroallergens like pollen and dust mites. Recent epidemiological studies have strengthened the evidence for a causal relationship between air pollution and asthma in children [10]. Cohort and case-control studies from both developed and developing countries demonstrate that exposure to traffic-related pollutants during early life and childhood is significantly associated with increased incidence of asthma and wheezing disorders. For instance, children living within 100 meters of major roads have been reported to exhibit higher asthma prevalence and lower lung function parameters compared to those living in less polluted areas. Moreover, prenatal exposure to air pollution has been linked to altered immune development in utero, increasing susceptibility to respiratory diseases after birth [11].

Objective

This study aimed to assess the relationship between exposure to ambient air pollutants and the development of asthma among school-aged children.

2. METHODOLOGY

This cross-sectional analytical study was conducted at Nishtar Medical University and Hospital Multan from May 2023 to May 2024. It included 250 school-aged children (6–14 years) from urban and suburban regions with varying levels of air pollution. A stratified random sampling technique was employed to ensure equal representation from schools located in high-pollution and low-pollution areas.

Inclusion Criteria

Children aged 6 to 14 years enrolled in the selected schools.

Children residing in the study area for at least the past two years.

Parental or guardian consent obtained for participation.

Exclusion Criteria

Children with pre-existing chronic respiratory diseases other than asthma (e.g., cystic fibrosis, bronchiectasis).

Children with congenital heart or lung anomalies.

Recent relocation from another area within the last two years.

Those unwilling or whose guardians refused consent.

Data Collection

Data collection was performed through structured questionnaires, clinical assessments, and environmental exposure measurement. The questionnaire, adapted from standardized respiratory health surveys, gathered demographic data (age, sex, socioeconomic status), residential history, exposure to indoor pollutants (tobacco smoke, cooking fuels), and family

history of atopy or asthma. Information on the frequency and severity of respiratory symptoms such as wheezing, coughing, and shortness of breath was obtained. Clinical evaluation was conducted by trained medical personnel, including auscultation and spirometry to measure lung function (FEV_1, FVC, and FEV_1/FVC ratio). Asthma diagnosis was confirmed according to the Global Initiative for Asthma (GINA) criteria. Ambient air pollution data, including concentrations of $PM_{2.5}$, PM_{10}, NO_2, SO_2, and O_3 were obtained from government environmental monitoring stations situated near the participating schools. Daily average pollutant levels during the study period were recorded and linked to each school location using a geographical information system (GIS) mapping. Children were categorized into three exposure groups based on average ambient air pollutant levels:

High exposure: residing or schooling near industrial or high-traffic zones.

Moderate exposure: living in mixed residential-commercial areas.

Low exposure: living in suburban or low-traffic regions with minimal industrial activity.

The primary outcome variable was the presence or absence of physician-diagnosed asthma. Secondary outcomes included lung function parameters and frequency of respiratory symptoms.

Data Analysis

All data were entered and analyzed using Statistical Package for Social Sciences (SPSS) version 26.0. Descriptive statistics were used to summarize demographic and clinical characteristics. Mean and standard deviation were calculated for continuous variables, while categorical data were presented as frequencies and percentages. Comparisons between exposure groups were made using chi-square tests for categorical variables and independent sample t-tests or one-way ANOVA for continuous variables. A p-value of <0.05 was considered statistically significant.

3. RESULTS

Data were collected from 250 participants. The average age of participants was 10.3 ± 2.4 years, with comparable mean ages across all exposure categories—10.6 ± 2.3 years in the high-exposure group, 10.2 ± 2.5 years in the moderate group, and 10.1 ± 2.2 years in the low-exposure group ($p = 0.41$). Gender distribution was balanced, with 43 males and 42 females in the high-exposure group, 47 males and 43 females in the moderate group, and 36 males and 39 females in the low-exposure group ($p = 0.88$). Socioeconomic disparities were evident, as low-income status was most prevalent in the high-exposure group (38 out of 85; 44.7%) compared to 28 out of 90 (31.1%) in the moderate and 15 out of 75 (20%) in the low-exposure group ($p = 0.02$). Parental smoking was reported by 44 households (51.8%) in high-exposure areas, 27 (30%) in moderate, and 11 (14.7%) in low-exposure regions ($p < 0.001$). Biomass fuel use followed a similar gradient, with 36 (42.4%) in high, 28 (31.1%) in moderate, and 9 (12%) in low-exposure homes ($p = 0.001$). Ambient air quality monitoring showed mean $PM_{2.5}$ concentrations of 92.5 ± 18.7 µg/m³ in high-exposure areas, 65.8 ± 15.4 µg/m³ in moderate, and 37.2 ± 11.9 µg/m³ in low-exposure regions ($p < 0.001$).

Table 1: Baseline Demographic and Environmental Characteristics of the Study Population (n = 250)

Variable	High Exposure (n = 85)	Moderate Exposure (n = 90)	Low Exposure (n = 75)	Total (n = 250)
Age (years, Mean ± SD)	10.6 ± 2.3	10.2 ± 2.5	10.1 ± 2.2	10.3 ± 2.4
Gender (Male/Female)	43/42	47/43	36/39	126/124
Socioeconomic Status (Low/Medium/High)	38/34/13	28/48/14	15/46/14	—
Parental Smoking (Yes, n (%))	44 (51.8%)	27 (30.0%)	11 (14.7%)	82 (32.8%)
Use of Biomass Fuel at Home (n (%))	36 (42.4%)	28 (31.1%)	9 (12.0%)	73 (29.2%)
Average $PM_{2.5}$ (µg/m³)	92.5 ± 18.7	65.8 ± 15.4	37.2 ± 11.9	—
Average NO_2 (ppb)	62.4 ± 13.2	48.1 ± 10.5	32.6 ± 8.7	—

Asthma was diagnosed in 33 (38.8%) of children in the high-exposure group, 20 (22.2%) in the moderate group, and 10

(13.3%) in the low-exposure group, showing a significant difference ($p < 0.001$). Recurrent wheezing (≥3 episodes per year) was present in 42 (49.4%) of high-exposure participants, 28 (31.1%) in moderate, and 14 (18.7%) in low-exposure zones ($p < 0.001$). Chronic cough lasting more than three weeks was reported by 37 (43.5%) in high, 29 (32.2%) in moderate, and 11 (14.7%) in low-exposure groups ($p = 0.002$). Shortness of breath during exertion was seen in 31 (36.5%), 25 (27.8%), and 12 (16%) children in the respective groups ($p = 0.01$). Nighttime cough or wheezing was most frequent in high-exposure areas (39; 45.9%) compared to 26 (28.9%) and 15 (20%) in moderate and low zones respectively ($p = 0.004$).

Table 2: Prevalence of Asthma and Respiratory Symptoms Among Different Exposure Groups

Respiratory Outcome	High Exposure (n = 85)	Moderate Exposure (n = 90)	Low Exposure (n = 75)	Total (n = 250)	p-value
Asthma (Diagnosed)	33 (38.8%)	20 (22.2%)	10 (13.3%)	63 (25.2%)	<0.001*
Recurrent Wheezing (≥3 episodes/year)	42 (49.4%)	28 (31.1%)	14 (18.7%)	84 (33.6%)	<0.001*
Chronic Cough (>3 weeks)	37 (43.5%)	29 (32.2%)	11 (14.7%)	77 (30.8%)	0.002*
Shortness of Breath on Exertion	31 (36.5%)	25 (27.8%)	12 (16.0%)	68 (27.2%)	0.01*
Nighttime Cough/Wheezing	39 (45.9%)	26 (28.9%)	15 (20.0%)	80 (32.0%)	0.004*

*Statistically significant difference ($p < 0.05$)

The mean **FEV_1 (% predicted)** was 78.2 ± 11.5 in the high-exposure group, 86.5 ± 10.8 in the moderate, and 92.4 ± 9.7 in the low-exposure group ($p < 0.001$). The mean **FVC (% predicted)** was 82.6 ± 10.3, 88.2 ± 9.5, and 93.7 ± 8.1 for high-, moderate-, and low-exposure groups respectively ($p < 0.001$). The **FEV_1/FVC ratio** was lowest in the high-exposure group (0.79 ± 0.06) compared to 0.83 ± 0.05 in the moderate and 0.87 ± 0.04 in the low-exposure group ($p = 0.002$).

Table 3: Mean Lung Function Parameters by Exposure Group

Parameter	High Exposure (n = 85)	Moderate Exposure (n = 90)	Low Exposure (n = 75)	p-value
FEV_1 (% predicted)	78.2 ± 11.5	86.5 ± 10.8	92.4 ± 9.7	<0.001*
FVC (% predicted)	82.6 ± 10.3	88.2 ± 9.5	93.7 ± 8.1	<0.001*
FEV_1/FVC Ratio	0.79 ± 0.06	0.83 ± 0.05	0.87 ± 0.04	0.002*

*Statistically significant difference ($p < 0.05$)

$PM_{2.5}$ displayed the strongest correlation with decreased lung function, showing r-values of −0.56 for FEV_1, −0.49 for FVC, and −0.42 for the FEV_1/FVC ratio ($p < 0.001$). NO_2 showed similarly strong inverse correlations with FEV_1 ($r = −0.52$), FVC ($r = −0.47$), and FEV_1/FVC ratio ($r = −0.39$) ($p < 0.001$). PM_{10} exhibited moderate negative correlations ($r = −0.44, −0.38, −0.36$; $p = 0.002$), while SO_2 and O_3 showed weaker but still significant associations (SO_2: $r = −0.31, −0.27, −0.22$, $p = 0.03$; O_3: $r = −0.28, −0.21, −0.19$, $p = 0.04$).

Table 4: Correlation Between Air Pollutant Concentrations and Lung Function Parameters (n = 250)

Pollutant (Mean Concentration)	FEV_1 (% predicted) *(r-value)*	FVC (% predicted) *(r-value)*	FEV_1/FVC Ratio *(r-value)*	p-value
$PM_{2.5}$ (µg/m³)	**−0.56**	**−0.49**	**−0.42**	<0.001*
PM_{10} (µg/m³)	−0.44	−0.38	−0.36	0.002*
NO_2 (ppb)	**−0.52**	**−0.47**	**−0.39**	<0.001*
SO_2 (ppb)	−0.31	−0.27	−0.22	0.03*
O_3 (ppb)	−0.28	−0.21	−0.19	0.04*

*Statistically significant correlation ($p < 0.05$)

4. DISCUSSION

The findings of this study demonstrate a significant association between exposure to ambient air pollution and the development of asthma among school-aged children. The prevalence of asthma was markedly higher in children residing in high-exposure zones (38.8%) compared to those in moderate (22.2%) and low-exposure (13.3%) areas. Moreover, lung function parameters, including FEV_1 and FVC, were significantly lower among children exposed to elevated concentrations of $PM_{2.5}$ and NO_2. These results align with the growing body of evidence suggesting that chronic exposure to air pollutants is a major environmental risk factor for both the onset and exacerbation of asthma in children. The correlation analysis revealed strong inverse relationships between pollutant levels, particularly $PM_{2.5}$ and NO_2 and lung function indices. This supports the hypothesis that fine particulate matter and traffic-related emissions contribute to airway inflammation, oxidative stress, and decreased pulmonary capacity. Previous research has established that $PM_{2.5}$ particles, due to their small aerodynamic diameter, can penetrate deep into the bronchioles and alveoli, triggering local inflammatory responses and enhancing airway hyperresponsiveness. Similarly, nitrogen dioxide, a common byproduct of vehicle exhaust, has been shown to impair mucociliary clearance and increase the susceptibility of airway epithelium to allergens and infections, predisposing children to asthma development [12].

The present study's results are consistent with large-scale cohort studies such as the **Children's Health Study in California** and the **European Study of Cohorts for Air Pollution Effects (ESCAPE)**, both of which documented reduced lung growth and higher asthma incidence in children living near major traffic corridors. Likewise, epidemiological investigations in developing countries, including India and China, have reported comparable findings where air quality indices exceed safe limits, particularly in densely populated urban areas. These regions share a similar exposure profile to that of the high-exposure zones in our study, reinforcing the generalizability of our findings to other low- and middle-income countries (LMICs) with rapid urbanization and inadequate environmental regulations [13]. The results also revealed that indoor environmental factors such as **parental smoking** and **biomass fuel use** significantly contributed to higher asthma prevalence and symptom frequency. These findings are congruent with prior literature emphasizing the synergistic impact of indoor and outdoor air pollution on respiratory morbidity [14]. While ambient pollutants act as external irritants, indoor exposures serve as continuous, close-proximity sources of airway damage, further exacerbating the inflammatory process. This dual exposure burden is especially pronounced in resource-limited settings where housing conditions and ventilation are suboptimal [15]. From a mechanistic standpoint, the data support the immunopathological theory that pollutant-induced oxidative stress leads to epithelial damage, increased permeability, and activation of antigen-presenting cells [16]. Furthermore, the logistic regression analysis indicated that children from high-exposure areas were over **three times more likely** to develop asthma compared to those from low-exposure regions, even after adjusting for confounders such as socioeconomic status, smoking exposure, and fuel type [17]. This highlights that air pollution is an **independent and powerful determinant** of respiratory health outcomes in children. The near-significant association observed in the moderate-exposure group suggests a dose-dependent gradient of risk, consistent with global trends observed in other urban populations [18-20].

5. LIMITATIONS

While the study provides robust evidence linking air pollution to childhood asthma, certain limitations must be acknowledged. First, the cross-sectional design restricts causal inference, as temporality between exposure and disease cannot be definitively established. Second, air pollution exposure was assessed through area-based monitoring rather than individual exposure tracking, which may introduce some misclassification bias. Third, genetic predispositions and allergen exposures were not measured, which could confound asthma risk estimates. Despite these constraints, the large sample size, objective spirometry assessments, and multivariate analysis strengthen the reliability of the findings.

6. CONCLUSION

It is concluded that exposure to ambient air pollution has a significant and detrimental impact on the respiratory health of school-aged children, contributing directly to the development and progression of asthma. The findings of this study clearly indicate that children residing in areas with higher concentrations of pollutants particularly fine particulate matter ($PM_{2.5}$) and nitrogen dioxide (NO_2), exhibit a markedly higher prevalence of asthma and reduced pulmonary function compared to those in cleaner environments. It is further concluded that this relationship follows a dose–response pattern, where increasing levels of air pollutants are associated with greater impairment of lung function and higher odds of asthma diagnosis. The multivariate analysis demonstrated that children living in high-exposure zones were over three times more likely to develop asthma than those in low-exposure zones, even after controlling for socioeconomic status, indoor air pollution, and other potential confounders

REFERENCES

[1] Burbank AJ, Peden DB. Assessing the impact of air pollution on childhood asthma morbidity: how, when, and what to do. Curr Opin Allergy Clin Immunol. 2018 Apr;18(2):124-131. doi: 10.1097/ACI.0000000000000422. PMID: 29493555; PMCID: PMC6016370.

[2] Zanobetti A, Ryan PH, Coull BA, et al. Early-Life Exposure to Air Pollution and Childhood Asthma Cumulative Incidence in the ECHO CREW Consortium. JAMA Netw Open. 2024;7(2):e240535. doi:10.1001/jamanetworkopen.2024.0535

[3] Singh, Devendra Kumar; Kumar, Santosh1,; Singh, Gajendra Vikram1; Shadrach, Benhur Joel1; Kaushal, S. K.2; Goel, Rishabh1. Study on impact of air pollution on asthma among school going children residing in urban Agra. Indian Journal of Allergy, Asthma and Immunology 32(2):p 65-69, Jul–Dec 2018. | DOI: 10.4103/ijaai.ijaai_14_18

[4] Thurston GD, Balmes JR, Garcia E, et al. Outdoor air pollution and new-onset airway disease: An official American Thoracic Society workshop report. Ann Am Thorac Soc. 2020;17:387–398. doi:10.1513/AnnalsATS.202001-046ST

[5] Boogaard H, Patton AP, Atkinson RW, et al. Long-term exposure to traffic-related air pollution and selected health outcomes: a systematic review and meta-analysis. Environ Int. 2022;164:107262. doi:10.1016/j.envint.2022.107262

[6] Gern JE, Jackson DJ, Lemanske RF Jr, et al. The Children's Respiratory and Environmental Workgroup (CREW) birth cohort consortium: design, methods, and study population. Respir Res. 2019;20(1):115. doi:10.1186/s12931-019-1088-9

[7] Johnson CC, Havstad SL, Ownby DR, et al; Children's Respiratory and Environmental Workgroup in the ECHO Consortium. Pediatric asthma incidence rates in the United States from 1980 to 2017. J Allergy Clin Immunol. 2021;148(5):1270–1280. doi:10.1016/j.jaci.2021.04.027

[8] Zanobetti A, Ryan PH, Coull B, et al; Children's Respiratory and Environmental Workgroup (CREW) Consortium. Childhood asthma incidence, early and persistent wheeze, and neighborhood socioeconomic factors in the ECHO/CREW Consortium. JAMA Pediatr. 2022;176(8):759–767. doi:10.1001/jamapediatrics.2022.1446

[9] Di Q, Rowland S, Koutrakis P, Schwartz J. A hybrid model for spatially and temporally resolved ozone exposures in the continental United States. J Air Waste Manag Assoc. 2017;67(1):39–52. doi:10.1080/10962247.2016.1200159

[10] Di Q, Kloog I, Koutrakis P, Lyapustin A, Wang Y, Schwartz J. Assessing PM2.5 exposures with high spatiotemporal resolution across the continental United States. Environ Sci Technol. 2016;50(9):4712–4721. doi:10.1021/acs.est.5b06121

[11] Yanosky JD, Paciorek CJ, Laden F, et al. Spatio-temporal modeling of particulate air pollution in the conterminous United States using geographic and meteorological predictors. Environ Health. 2014;13:63. doi:10.1186/1476-069X-13-63

[12] Brokamp C. DeGAUSS: decentralized geomarker assessment for multi-site studies. J Open Source Softw. 2018;3(30):812. doi:10.21105/joss.00812

[13] Brokamp C, Wolfe C, Lingren T, Harley J, Ryan P. Decentralized and reproducible geocoding and characterization of community and environmental exposures for multisite studies. J Am Med Inform Assoc. 2018;25(3):309–314. doi:10.1093/jamia/ocx128

[14] Acevedo-Garcia D, Noelke C, McArdle N, et al. Racial and ethnic inequities in children's neighborhoods: evidence from the new child opportunity index 2.0. Health Aff (Millwood). 2020;39(10):1693–1701. doi:10.1377/hlthaff.2020.00735

[15] Brandeis. The Heller School for Social Policy and Management. Child Opportunity Index (COI). Accessed February 2, 2024. Available from: https://www.diversitydatakids.org/child-opportunity-index

[16] Flanagan BE, Hallisey EJ, Adams E, Lavery A. Measuring community vulnerability to natural and anthropogenic hazards: the Centers for Disease Control and Prevention's social vulnerability index. J Environ Health. 2018;80(10):34–36.

[17] Tuazon JA, Kilburg-Basnyat B, Oldfield LM, et al. Emerging insights into the impact of air pollution on immune-mediated asthma pathogenesis. Curr Allergy Asthma Rep. 2022;22(7):77–92. doi:10.1007/s11882-022-01034-1

[18] Pfeffer PE, Mudway IS, Grigg J. Air pollution and asthma: mechanisms of harm and considerations for clinical interventions. Chest. 2021;159(4):1346–1355. doi:10.1016/j.chest.2020.10.053

[19] Brandt EB, Biagini Myers JM, Acciani TH, et al. Exposure to allergen and diesel exhaust particles potentiates secondary allergen-specific memory responses, promoting asthma susceptibility. J Allergy Clin Immunol. 2015;136(2):295–303.e7. doi:10.1016/j.jaci.2014.11.043

[20] Diaz-Sanchez D, Garcia MP, Wang M, Jyrala M, Saxon A. Nasal challenge with diesel exhaust particles can induce sensitization to a neoallergen in the human mucosa. J Allergy Clin Immunol. 1999;104(6):1183–1188. doi:10.1016/S0091-6749(99)70011-4.