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Original Research Article

Impact of Indoor Air Pollution Exposure on Asthma Control and Exacerbation Frequency in School-Aged Children

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Abstract

Background: Indoor air pollution represents a significant environmental health concern, particularly for children with asthma who spend considerable time in enclosed environments. Understanding the relationship between indoor pollutant exposure and asthma outcomes is essential for developing effective prevention strategies.

Methods: A prospective observational study was conducted among 248 children aged 6-14 years with physician-diagnosed asthma. Indoor air quality measurements including particulate matter (PM2.5), nitrogen dioxide (NO2), and volatile organic compounds (VOCs) were obtained from participants' homes using standardized monitoring equipment. Asthma control was assessed using the Childhood Asthma Control Test (C-ACT), and exacerbation frequency was documented over a 12-month follow-up period. Spirometry was performed to evaluate pulmonary function.

Results: Children exposed to high indoor PM2.5 levels (>35 μg/m³) demonstrated significantly poorer asthma control (C-ACT score: 16.2 ± 4.1 vs. 21.8 ± 3.6, $p<0.001$) compared to those with low exposure. The mean annual exacerbation frequency was 3.8 ± 1.9 in the high-exposure group versus 1.4 ± 1.2 in the low-exposure group ($p<0.001$). Elevated NO2 concentrations were associated with reduced FEV1/FVC ratio (78.3 ± 8.2% vs. 85.7 ± 6.4%, $p<0.001$). Multiple regression analysis revealed that PM2.5 exposure independently predicted exacerbation risk (OR: 2.84, 95% CI: 1.92-4.21, $p<0.001$).

Conclusion: Indoor air pollution exposure significantly compromises asthma control and increases exacerbation frequency in school-aged children, emphasizing the critical need for environmental interventions in residential settings.

Keywords: Indoor Air Pollution, Childhood Asthma, Asthma Control, Exacerbation, Particulate Matter, Nitrogen Dioxide, Pulmonary Function.

Introduction

Asthma remains the most prevalent chronic respiratory disease affecting children worldwide, with an estimated 262 million individuals affected globally [1]. The pathophysiology of asthma involves chronic airway inflammation, bronchial hyperresponsiveness, and variable airflow obstruction, which collectively contribute to recurrent episodes of wheezing, breathlessness, chest tightness, and coughing [2]. In pediatric populations, poorly controlled asthma significantly impacts quality of life, academic performance, and healthcare resource utilization [3].

Environmental factors play a crucial role in asthma development, progression, and exacerbation. Indoor air pollution has emerged as a particularly important exposure pathway, given that children spend approximately 80-90% of their time in indoor environments, including homes and schools [4]. Common indoor air pollutants include particulate matter (PM2.5 and PM10), nitrogen dioxide (NO2), volatile organic compounds (VOCs), carbon monoxide, and biological allergens [5]. These pollutants originate from various sources such as cooking appliances, heating systems, tobacco smoke, cleaning products, and building materials [6].

Previous investigations have established associations between indoor air pollution and respiratory health outcomes. Kanchongkittiphon and colleagues conducted a comprehensive review demonstrating sufficient evidence linking indoor dampness, environmental tobacco smoke, and

nitrogen dioxide exposure to asthma exacerbation [7]. Similarly, research by Diette and colleagues revealed that inner-city children with asthma experience significantly higher indoor pollutant exposures compared to non-asthmatic counterparts [8]. The mechanistic pathways underlying these associations involve oxidative stress induction, inflammatory cascade activation, and epithelial barrier dysfunction [9].

Despite growing evidence, significant research gaps persist regarding the dose-response relationships between specific indoor pollutants and asthma outcomes in pediatric populations. Furthermore, limited data exist concerning the combined effects of multiple indoor pollutants on asthma control metrics and exacerbation patterns [10]. Understanding these relationships is essential for developing evidence-based environmental interventions and clinical guidelines.

The present study aimed to investigate the impact of indoor air pollution exposure on asthma control levels, exacerbation frequency, and pulmonary function parameters in school-aged children. We hypothesized that higher indoor pollutant concentrations would be associated with poorer asthma control, increased exacerbation rates, and diminished pulmonary function.

Materials and Methods

Study Design and Setting: This prospective observational study was conducted at the Pediatric Clinic of a tertiary care hospital.

Study Population: A total of 248 children aged 6-14 years with physician-diagnosed asthma, according to Global Initiative for Asthma (GINA) criteria, were enrolled. Inclusion criteria comprised: (1) confirmed asthma diagnosis for at least 12 months; (2) current prescription of asthma controller medication; (3) residence in the same household for at least 6 months prior to enrollment; and (4) parental consent and child assent where applicable.

Exclusion criteria included: (1) concurrent chronic respiratory conditions other than asthma; (2) significant cardiovascular, neurological, or immunological disorders; (3) hospitalization for respiratory illness within 4 weeks prior to enrollment; (4) planned residential relocation during the study period; and (5) inability to perform spirometry adequately.

Indoor Air Quality Assessment: Indoor air quality monitoring was conducted in participants' homes using validated portable monitoring devices. PM2.5 concentrations were measured using laser-based particle counters (DustTrak II Model 8530, TSI Incorporated) placed in the main living area and the child's bedroom. Nitrogen dioxide levels were assessed using passive diffusion samplers (Ogawa & Company) deployed for 7-day sampling periods. VOC concentrations were measured using photoionization detectors (ppbRAE 3000, RAE Systems).

Measurements were obtained during two separate 7-day periods (winter and summer seasons) to capture seasonal variation. Mean values across both seasons were calculated for analysis. Based on PM2.5 concentrations, participants were categorized into low-exposure (<15 μg/m³), moderate-exposure (15-35 μg/m³), and high-exposure (>35 μg/m³) groups according to World Health Organization indoor air quality guidelines.

Clinical Assessments: Asthma control was evaluated using the Childhood Asthma Control Test (C-ACT) for children aged 6-11 years and the Asthma Control Test (ACT) for children aged 12-14 years. Scores were assessed at baseline and quarterly intervals throughout the 12-month follow-up period. Scores ≤19 indicated poorly controlled asthma.

Asthma exacerbations were defined as episodes requiring systemic corticosteroid administration for ≥3 days, emergency department visits, or hospitalizations for asthma. Exacerbation events were documented through medical record review and monthly telephone follow-ups with caregivers.

Pulmonary function testing was performed using a calibrated spirometer (MasterScreen Pneumo, CareFusion) according to American Thoracic Society/European Respiratory Society guidelines. Parameters included forced expiratory volume in one second (FEV1), forced vital capacity (FVC), FEV1/FVC ratio, and peak expiratory flow (PEF). Values were expressed as percentage of predicted based on age, height, sex, and ethnicity.

Covariates: Demographic and clinical information was collected through standardized questionnaires administered to caregivers. Variables included age, sex, body mass index, socioeconomic status, parental smoking history, presence of pets, type of cooking fuel, housing characteristics, and asthma medication use.

Statistical Analysis: Sample size was calculated based on an expected difference of 15% in exacerbation frequency between exposure groups, with 80% power and 5% significance level, yielding a minimum requirement of 220 participants. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Comparisons between groups were performed using one-way ANOVA with post-hoc Tukey tests for continuous variables and chi-square tests for categorical variables.

Pearson correlation coefficients were calculated to assess relationships between continuous variables.

Multiple logistic regression analysis was conducted to identify independent predictors of exacerbation risk, adjusting for potential confounders. Statistical significance was set at $p<0.05$. All analyses were performed using SPSS version 26.0 (IBM Corporation).

Results

Participant Characteristics: Of 278 children initially screened, 248 met eligibility criteria and completed the study protocol. The mean age was 9.4 ± 2.3 years, with 142 (57.3%) males and 106 (42.7%) females. Baseline demographic and clinical characteristics stratified by PM2.5 exposure level are presented in Table 1.

Table 1: Baseline Demographic and Clinical Characteristics by PM2.5 Exposure Level

Variable	Low Exposure (n=76)	Moderate Exposure (n=98)	High Exposure (n=74)	p-value
Age (years), mean ± SD	9.2 ± 2.4	9.5 ± 2.2	9.6 ± 2.3	0.528
Male sex, n (%)	42 (55.3)	58 (59.2)	42 (56.8)	0.847
BMI (kg/m²), mean ± SD	18.2 ± 3.1	18.6 ± 3.4	19.1 ± 3.6	0.234
Asthma duration (years), mean ± SD	4.1 ± 2.2	4.3 ± 2.4	4.5 ± 2.6	0.564
Parental smoking, n (%)	12 (15.8)	24 (24.5)	34 (45.9)	<0.001
Gas cooking fuel use, n (%)	58 (76.3)	82 (83.7)	68 (91.9)	0.028
Pet ownership, n (%)	18 (23.7)	22 (22.4)	14 (18.9)	0.752
ICS use, n (%)	68 (89.5)	86 (87.8)	62 (83.8)	0.563
Baseline C-ACT score, mean ± SD	21.4 ± 3.2	19.2 ± 3.8	17.8 ± 4.2	<0.001

BMI: body mass index; ICS: inhaled corticosteroid; C-ACT: Childhood Asthma Control Test

Indoor Air Quality Measurements: Mean indoor PM2.5 concentration across all households was 28.4 ± 18.6 μg/m³. The mean NO2 concentration was 32.8 ± 14.2 ppb, and mean total VOC concentration was 186.4 ± 78.3 μg/m³. Significant positive correlations were observed between PM2.5 and NO2 levels ($r=0.52$, $p<0.001$) and between PM2.5 and VOC levels ($r=0.41$, $p<0.001$).

Asthma Control and Exacerbation Outcomes: Table 2 presents asthma control parameters and exacerbation outcomes stratified by PM2.5 exposure level.

Children in the high-exposure group demonstrated significantly lower mean C-ACT scores and higher exacerbation frequencies compared to low-exposure counterparts.

Table 2: Asthma Control and Exacerbation Outcomes by PM2.5 Exposure Level

Outcome Variable	Low Exposure (n=76)	Moderate Exposure (n=98)	High Exposure (n=74)	p-value
Mean C-ACT score (12-month), mean ± SD	21.8 ± 3.6	18.6 ± 4.0	16.2 ± 4.1	<0.001
Poorly controlled asthma (C-ACT ≤19), n (%)	14 (18.4)	48 (49.0)	56 (75.7)	<0.001
Annual exacerbations, mean ± SD	1.4 ± 1.2	2.6 ± 1.6	3.8 ± 1.9	<0.001
≥2 exacerbations/year, n (%)	22 (28.9)	54 (55.1)	58 (78.4)	<0.001
ED visits for asthma, n (%)	8 (10.5)	26 (26.5)	32 (43.2)	<0.001
Hospitalizations for asthma, n (%)	2 (2.6)	8 (8.2)	14 (18.9)	0.002
Oral corticosteroid courses, mean ± SD	0.8 ± 0.9	1.8 ± 1.4	2.9 ± 1.7	<0.001
School absence days, mean ± SD	4.2 ± 3.8	8.6 ± 5.4	14.2 ± 7.6	<0.001

ED: emergency department; C-ACT: Childhood Asthma Control Test

Pulmonary Function Parameters: Pulmonary function measurements demonstrated significant differences across exposure groups. Table 3 summarizes spirometric parameters stratified by NO2 exposure level.

Table 3: Pulmonary Function Parameters by NO2 Exposure Level

Parameter	Low NO2 (<25 ppb) (n=82)	Moderate NO2 (25-40 ppb) (n=96)	High NO2 (>40 ppb) (n=70)	p-value
FEV1 % predicted, mean ± SD	92.4 ± 10.8	86.2 ± 11.4	79.6 ± 12.2	<0.001
FVC % predicted, mean ± SD	96.8 ± 9.6	94.2 ± 10.2	91.4 ± 11.8	0.008
FEV1/FVC %, mean ± SD	85.7 ± 6.4	81.8 ± 7.6	78.3 ± 8.2	<0.001
PEF % predicted, mean ± SD	88.6 ± 14.2	82.4 ± 15.6	74.8 ± 16.8	<0.001
FEF25-75 % predicted, mean ± SD	78.4 ± 18.6	68.2 ± 20.4	58.6 ± 22.8	<0.001
Bronchodilator reversibility ≥12%, n (%)	24 (29.3)	38 (39.6)	42 (60.0)	<0.001

FEV1: forced expiratory volume in one second; FVC: forced vital capacity; PEF: peak expiratory flow; FEF25-75: forced expiratory flow at 25-75% of FVC

Multivariable Analysis: Multiple logistic regression analysis, adjusting for age, sex, BMI, asthma duration, parental smoking, and medication adherence, revealed that high PM2.5 exposure independently predicted frequent exacerbations (≥2/year) with an odds ratio of 2.84 (95% CI: 1.92-4.21, $p<0.001$). Elevated NO2 exposure was associated with poorly controlled asthma (OR: 2.16, 95% CI: 1.48-3.15, $p<0.001$).

Discussion

The present study demonstrates significant associations between indoor air pollution exposure and adverse asthma outcomes in school-aged children. Our findings indicate that elevated concentrations of PM2.5 and NO2 in residential environments are independently associated with poorer asthma control, increased exacerbation frequency, and reduced pulmonary function parameters. These results have important implications for clinical management and public health interventions targeting pediatric asthma.

The observed relationship between PM2.5 exposure and asthma exacerbations aligns with previous investigations examining indoor environmental factors. Hansel and colleagues demonstrated that indoor NO2 levels were associated with increased respiratory symptoms and decreased pulmonary function in inner-city children with asthma over a longitudinal follow-up period [11]. Similarly, our study revealed a dose-response relationship, with children in the highest exposure category experiencing nearly three-fold greater exacerbation frequency compared to those with minimal exposure.

The mechanistic basis for pollution-induced asthma exacerbation involves multiple pathways. Particulate matter induces oxidative stress through reactive oxygen species generation, leading to airway epithelial damage and inflammatory mediator release [12]. Nitrogen dioxide, a product of combustion processes, directly irritates airway mucosa and enhances bronchial responsiveness to allergens [13]. These pathophysiological mechanisms explain the clinical observations of increased symptom burden and healthcare utilization among highly exposed children.

Our findings regarding pulmonary function decrements associated with NO2 exposure corroborate epidemiological evidence linking traffic-related air pollution to reduced lung growth in children [14]. The significant reductions in FEV1 and FEV1/FVC ratio observed in the high-exposure group suggest that chronic indoor pollutant exposure may contribute to airway obstruction beyond the acute inflammatory response. Long-term consequences may include accelerated lung function decline and increased risk of fixed airway obstruction in adulthood [15].

The association between gas cooking fuel use and elevated pollutant exposure underscores the importance of combustion sources in residential settings. Previous research has identified cooking activities as major contributors to indoor PM2.5 and NO2 concentrations, particularly in homes with inadequate ventilation [16]. Implementation of improved ventilation systems and transitioning to electric cooking appliances represent potential intervention strategies to reduce exposure [17].

Parental smoking emerged as a significant covariate associated with both higher pollutant levels and worse asthma outcomes. Environmental tobacco smoke contains numerous toxic constituents that synergistically enhance the effects of other indoor pollutants on respiratory health [18]. Smoking cessation counseling and enforcement of smoke-free home policies should remain integral components of comprehensive asthma management programs [19].

The substantial school absenteeism observed among highly exposed children highlights the broader societal impact of indoor air pollution on pediatric asthma. Academic disruption, reduced physical activity, and psychological stress associated with poorly controlled asthma may have lasting effects on child development and educational attainment [20].

Several limitations warrant consideration. The observational design precludes causal inference, and unmeasured confounders may influence the observed associations. Indoor air quality measurements, although conducted over two seasonal periods, may not fully capture temporal variability in pollutant concentrations. Additionally, reliance on caregiver-reported exacerbation data introduces potential recall bias.

Conclusion

This study provides compelling evidence that indoor air pollution exposure significantly compromises asthma control and increases exacerbation frequency in school-aged children. Children residing in homes with elevated PM2.5 and NO2 concentrations demonstrated substantially poorer clinical outcomes, including lower asthma control scores, more frequent exacerbations, increased emergency department visits, and reduced pulmonary function. These findings emphasize the critical importance of environmental assessment and intervention as integral components of pediatric asthma management. Healthcare providers should incorporate indoor air quality evaluation into routine asthma care and provide families with evidence-based recommendations for reducing household pollutant exposures. Public health initiatives targeting improved housing conditions, ventilation systems, and clean cooking

technologies may substantially reduce the burden of poorly controlled childhood asthma.

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