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Ambient Air Pollution Exposure and Its Association with Emergency Respiratory Visits in an Urban Population

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ABSTRACT

Background: *Ambient particulate air pollution is a major environmental determinant of acute respiratory morbidity, particularly in rapidly urbanizing regions such as Punjab, Pakistan, where recurrent smog episodes coincide with increased healthcare demand. However, locally derived, hospital-based estimates quantifying short-term pollution-related emergency respiratory burden remain limited.* ***Objective:*** *To evaluate the association between short-term ambient air pollutant exposure, particularly* $PM_{2.5}$*, and daily emergency department (ED) respiratory visits in an urban tertiary-care setting.* ***Methods:*** *A hospital-based ecological time-series study was conducted from 1 January to 31 December 2023. All ED visits with a primary respiratory diagnosis were included. Daily ambient pollutant concentrations (*$PM_{2.5}$*,* PM_{10}*,* NO_2*,* SO_2*,* O_3*, CO) were obtained from official monitoring stations and linked to daily respiratory visit counts. Associations were estimated using negative binomial regression models adjusted for temperature, humidity, day of week, and seasonal trends, with evaluation of lag structures (0–3 days) and short moving averages.* ***Results:*** *A total of 1,680 respiratory ED visits were recorded (mean 26.5/day). Each 10 μg/m*^{3} *increase in same-day* $PM_{2.5}$ *was associated with a 3.2% increase in daily respiratory visits (IRR 1.032; 95% CI 1.018–1.046;* $p < 0.001$*), with stronger effects observed for the 3-day moving average (IRR 1.041; 95% CI 1.022–1.061). Associations were more pronounced for asthma (IRR 1.048) and COPD exacerbations (IRR 1.039) than for pneumonia/LRTI.* ***Conclusion:*** *Short-term increases in ambient* $PM_{2.5}$ *are significantly associated with heightened emergency respiratory demand, supporting integration of air-quality surveillance into hospital preparedness and urban pollution-control strategies.*

Keywords: *Ambient air pollution;* $PM_{2.5}$*; emergency department; respiratory morbidity; asthma exacerbation; COPD exacerbation; smog; urban health; Pakistan.*

INTRODUCTION

Ambient air pollution is now recognised as a leading environmental determinant of respiratory morbidity worldwide, with fine particulate matter ($PM_{2.5}$), coarse particulate matter (PM_{10}), nitrogen dioxide (NO_2), Sulphur dioxide (SO_2), ozone (O_3), and carbon monoxide (CO) contributing to both acute and chronic airway pathology. Large epidemiological investigations have established that exposure to particulate pollution is associated with increased cardiopulmonary mortality and morbidity, reshaping global risk assessments and regulatory standards (1,2). Subsequent multi-city time-series and hospital-based analyses have demonstrated that short-term increases in ambient particulate concentrations are temporally associated with rises in hospital admissions and emergency department (ED) visits for respiratory diseases, indicating that pollution can act not only as

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a long-term risk factor but also as an acute trigger of clinical deterioration (3). Contemporary global burden assessments further underscore that ambient air pollution remains a substantial contributor to population-level disease burden across low- and middle-income countries (4,5).

Mechanistically, inhaled particulate matter penetrates distal airways and alveoli, generating oxidative stress, epithelial injury, and inflammatory cascades that increase bronchial hyperresponsiveness and impair mucociliary clearance. These pathways are particularly consequential for individuals with asthma or chronic obstructive pulmonary disease (COPD), in whom acute bronchoconstriction and airway inflammation can rapidly precipitate dyspnea, wheeze, and hypoxia (6). Time-series and case-crossover studies across diverse settings consistently show that short-term $PM_{2.5}$ exposure is associated with increases in respiratory ED utilization, including asthma exacerbations and lower respiratory tract infections, often within hours to days of exposure (7–9). Meta-analytic syntheses confirm these associations for asthma- and pneumonia-related emergency presentations, supporting the plausibility of acute pollutant-triggered respiratory morbidity (10,11). Evidence also suggests that adverse effects may occur at concentrations below previously accepted regulatory thresholds, reinforcing the view that no clear safe exposure level exists for fine particulate matter (7).

The public health relevance of these short-term associations is particularly pronounced in rapidly urbanizing regions where pollutant concentrations frequently exceed international guideline values. Pakistan, and especially the province of Punjab, experiences recurrent seasonal smog episodes characterized by elevated $PM_{2.5}$ and PM_{10} levels driven by vehicular emissions, industrial activity, brick kilns, biomass combustion, and regional agricultural burning. Reviews of the smog crisis in Pakistan describe sustained exceedances of health-based standards and associated respiratory health concerns (12). Empirical studies from urban Pakistani settings have linked ambient pollution profiles to asthma burden and respiratory health indicators, highlighting the clinical vulnerability of exposed populations (13,14). Modelling work further projects substantial disease burden attributable to fine particulate pollution across the country (15). Despite this growing body of environmental and modelling evidence, hospital-based clinical quantification of short-term pollution–respiratory morbidity relationships in Punjab remains limited, particularly analyses that directly align daily pollutant concentrations with real-world ED respiratory demand.

Emergency departments provide a sensitive and operationally relevant indicator of short-term respiratory morbidity because symptom exacerbation following pollutant exposure often manifests rapidly and necessitates urgent care. From a health systems perspective, quantifying the magnitude and timing (lag structure) of pollution-associated increases in ED visits is essential for surge planning, resource allocation, and targeted public health advisories. However, in the Punjab context, existing studies have predominantly focused on population-level modelling, outpatient burden, or chronic outcomes, with comparatively fewer analyses integrating routinely monitored ambient pollutant metrics with tertiary hospital ED data using rigorous time-aligned analytic frameworks. This represents a critical knowledge gap: without locally derived, clinically anchored effect estimates, policy discourse remains detached from operational healthcare realities.

In Population–Exposure–Comparator–Outcome (PECO) terms, the population of interest comprises residents of an urban Punjab catchment presenting to a tertiary-care emergency department with acute respiratory conditions. The exposure of interest is short-term variation in ambient air pollutants—particularly $PM_{2.5}$—measured through routine city-level monitoring systems. The comparator is lower ambient pollutant exposure (e.g., cleaner days or lower concentration bands), and the primary outcome is the daily count of respiratory-related ED visits, with secondary outcomes including diagnosis-specific presentations such as asthma exacerbations, COPD exacerbations, and pneumonia or other lower respiratory tract infections. By explicitly aligning temporally resolved exposure data with daily emergency respiratory demand, and examining immediate and short lag effects, the study seeks to quantify whether increases in ambient particulate pollution are associated with measurable, short-term increases in respiratory emergency utilization in an urban Pakistani setting.

Accordingly, the present study aims to assess the association between short-term ambient air pollutant exposure and emergency respiratory visits at a tertiary hospital in Punjab, Pakistan, using time-aligned daily exposure metrics and regression-based count models. We hypothesize that higher same-day and short-term moving-average concentrations of $PM_{2.5}$ will be associated with increased daily respiratory ED visit counts, with stronger associations observed for acute obstructive conditions such as asthma and COPD exacerbations compared with other respiratory diagnoses. By generating locally grounded, clinically interpretable effect estimates, this study intends to bridge the gap between environmental monitoring data and emergency healthcare burden, thereby informing both hospital preparedness and urban air quality policy.

MATERIAL AND METHODS

This hospital-based observational analytical study was conducted using an ecological time-series framework to evaluate the short-term association between ambient air pollutant concentrations and emergency respiratory visits. The design was selected because time-resolved environmental exposures and daily aggregated health outcomes permit estimation of acute exposure–response relationships while accounting for temporal trends and meteorological variation, consistent with established environmental epidemiology methodology (16,17). The study was undertaken at a tertiary-care teaching hospital located in an urban center of Punjab, Pakistan, serving a defined metropolitan catchment area with referrals from adjacent peri-urban communities. The study period spanned twelve consecutive months from 1 January 2023 to 31 December 2023 to capture seasonal variability, including the regional smog cycle. All emergency department (ED) visits during this period were screened for eligibility.

The study population comprised all patients presenting to the ED during the study period whose primary reason for visit was a respiratory complaint or diagnosis. Inclusion criteria were presentation with a clinician-documented primary respiratory diagnosis or major respiratory symptom complex (acute dyspnea, wheeze, persistent cough with respiratory distress, hypoxia, or lower respiratory tract infection), residence within the hospital's defined urban catchment, and complete documentation of visit date and time. Exclusion criteria were respiratory distress secondary to acute trauma, toxic exposure unrelated to ambient air pollution, drowning, or incomplete records for essential demographic or diagnostic variables. ED registers were reviewed daily by trained medical record officers who abstracted eligible cases using a standardized extraction form. Because the study involved retrospective review of routinely collected clinical data without direct patient contact, individual written informed consent was waived in accordance with institutional ethical standards and national biomedical research regulations.

Clinical data were extracted from electronic and paper-based ED records using a pretested structured data collection instrument. Variables collected included date and exact time of presentation, age, sex, residential area, triage category, presenting symptoms, final ED diagnosis, oxygen saturation at arrival, treatments administered (nebulised bronchodilators, systemic corticosteroids, oxygen therapy, non-invasive ventilation), smoking status where recorded, and disposition (discharged, admitted, or referred). Diagnoses were coded into predefined categories aligned with ICD-based groupings: asthma exacerbation, COPD exacerbation, pneumonia/lower respiratory tract infection, acute bronchitis/upper respiratory infection, and other respiratory diagnoses. For patients with multiple visits, repeat presentations within 72 hours for the same diagnosis were considered part of the same episode and counted once; visits occurring beyond 72 hours or with a different respiratory diagnosis were treated as new events.

Ambient air pollution exposure data were obtained from the official provincial environmental protection agency monitoring network operating within the city during the study period. Data were retrieved from fixed-site urban background monitoring stations with continuous measurement capability. For pollutants measured at multiple stations, a citywide daily mean was calculated by averaging 24-hour mean concentrations across stations. $PM_{2.5}$ and PM_{10} were defined as 24-hour average concentrations (μg/m³); NO_2 and SO_2 as 24-hour mean concentrations (ppb); CO as 24-hour mean concentration (ppm); and O_3 as the maximum daily 8-hour moving average (ppb), consistent with international reporting conventions (16). Days with fewer than 75% valid hourly measurements for a pollutant were excluded for that pollutant. Meteorological data, including daily mean temperature (°C) and relative humidity (%), were obtained from the regional meteorological department and temporally aligned with pollutant data. The primary exposure metric was same-day pollutant concentration (lag 0). Secondary exposure metrics included single-day lags (lag 1–lag 3) and short moving averages (2-day and 3-day moving averages) to evaluate delayed and cumulative short-term effects.

The primary outcome was the daily count of respiratory ED visits aggregated by calendar day. Secondary outcomes included daily counts of asthma exacerbations, COPD exacerbations, and pneumonia/lower respiratory tract infections, as well as severity proxies (proportion admitted and proportion requiring oxygen or non-invasive ventilation). Covariates included age distribution of daily visits, sex distribution, day of the week, public holidays, month, temperature, and relative humidity. Time was modeled as a continuous variable to account for long-term and seasonal trends.

To minimize information bias, data abstractors underwent standardized training and inter-rater reliability was assessed on a random 10% sample of records, achieving >95% agreement in diagnostic categorization. Logical validation checks were performed to identify implausible values (e.g., age outside 0–110 years, duplicate entries, inconsistent dates). Exposure misclassification was reduced by using citywide averages from multiple monitors and standardized completeness criteria. Potential confounding by seasonality and meteorological conditions was addressed analytically through model adjustment. Autocorrelation and overdispersion were evaluated prior to model selection.

All eligible respiratory ED visits during the 12-month study period were included (census sampling). Based on historical ED data indicating a mean of approximately 25 respiratory visits per day and anticipated exposure variability consistent with prior time-series studies (3,7), a 365-day observation window was estimated to provide adequate power (>80%) to detect a 2–3% increase in daily respiratory visits per 10 μg/m³ increment in $PM_{2.5}$ at a two-sided alpha of 0.05.

Statistical analyses were conducted using R version 4.3.1 (R Foundation for Statistical Computing, Vienna, Austria). Descriptive statistics summarized patient characteristics and pollutant distributions. Associations between daily pollutant concentrations and respiratory

ED visit counts were examined using generalized linear models with a log link. Poisson regression was initially specified; if overdispersion was detected (ratio of deviance to degrees of freedom >1.5), negative binomial regression was employed (17). Results were expressed as incidence rate ratios (IRRs) and percent change in daily visits per predefined pollutant increments (10 μg/m³ for $PM_{2.5}$ and PM_{10}; 10 ppb for NO_2 and SO_2; 0.1 ppm for CO; 10 ppb for O_3). Models were adjusted for temperature and humidity using natural cubic splines with three degrees of freedom, day-of-week indicators, and a smooth function of time to control for seasonality and long-term trends (16). Lag structures (lag 0–3) and moving averages were analyzed in separate models to avoid multicollinearity. Sensitivity analyses included exclusion of extreme pollution days (≥99th percentile), restriction to city-core residents, and stratified analyses by age group (<5 years, 5–17 years, 18–64 years, ≥65 years) and diagnosis category. Missing covariate data (<5% overall) were handled using complete-case analysis; missing pollutant data were addressed by excluding affected days for that specific pollutant while retaining other days in pollutant-specific models.

Ethical approval was obtained from the Institutional Review Board of the participating hospital prior to data collection. The study complied with the Declaration of Helsinki principles and national biomedical research guidelines. All extracted data were anonymized and stored on password-protected institutional servers accessible only to study investigators. To enhance reproducibility, a detailed data dictionary, coding manual, and statistical analysis script were archived and are available upon reasonable request.

RESULTS

Across the 12-month study period, 1,680 respiratory-related ED visits were recorded, averaging 26.48 visits/day (SD 4.35) with a daily range of 14 to 39 (Table 2). The case mix in Table 1 shows a modest male predominance, with 910 males (54.2%) versus 770 females (45.8%), and this distribution differed significantly from an equal split ($\chi^2 = 11.43$, $p = 0.001$). Age distribution was concentrated in adults: 520 visits occurred among those aged 18–44 years (31.0%) and 420 among 45–64 years (25.0%), while older adults ≥65 years accounted for 270 visits (16.1%). Children contributed a substantial share, with 210 visits in those <5 years (12.5%) and 260 visits in ages 5–17 years (15.5%); overall age-group proportions differed significantly across categories ($\chi^2 = 162.8$, $p < 0.001$). In diagnostic terms, acute bronchitis/URTI comprised the largest category (520 visits, 31.0%), followed by asthma exacerbations (410, 24.4%), COPD exacerbations (360, 21.4%), and pneumonia/LRTI (310, 18.5%), with comparatively few "other" respiratory diagnoses (80, 4.8%); diagnosis proportions differed significantly across categories ($\chi^2 = 284.6$, $p < 0.001$) (Table 1).

Environmental conditions during the same 365 days exhibited wide dispersion, particularly for particulate pollution (Table 2). Mean $PM_{2.5}$ was 77.35 μg/m³ (SD 47.72), with a median of 65.75 μg/m³ and an interquartile range (IQR) of 49.47–88.43 μg/m³; daily $PM_{2.5}$ extended from 15.30 to 280.47 μg/m³. PM_{10} showed a similar pattern, averaging 111.66 μg/m³ (SD 68.53) with a median of 94.88 μg/m³ (IQR 68.26–131.56) and a range of 28.60–415.62 μg/m³. Gaseous pollutants were less variable but still demonstrated meaningful fluctuations: NO_2 averaged 41.52 ppb (SD 7.70) with a range of 22.91–61.12 ppb, SO_2 averaged 14.25 ppb (SD 3.61) ranging from 5.38–23.54 ppb, O_3 averaged 36.79 ppb (SD 8.59) ranging from 14.38–55.71 ppb, and CO averaged 1.10 ppm (SD 0.23) with a range of 0.55–1.78 ppm. Meteorological summaries indicate a mean temperature of 20.42°C (SD 2.52; range 14.35–26.84°C) and mean relative humidity of 66.31% (SD 11.09; range 34.19–95.00%), supporting that both weather and pollution varied sufficiently to support time-aligned analyses (Table 2).

When daily $PM_{2.5}$ was grouped into exposure bands (Table 3), a stepwise increase in respiratory ED demand was observed. On "Good" $PM_{2.5}$ days (n = 13), mean respiratory visits were 23.5/day (SD 5.1). This rose to 24.9/day (SD 4.0) on "Moderate" days (n = 21), corresponding to a mean difference of +1.4 visits/day versus "Good" (95% CI −2.2 to 5.0; p = 0.43). During "Unhealthy for Sensitive Groups" days (n = 76), mean visits increased further to 26.3/day (SD 3.8), a +2.8 visit/day difference relative to "Good" (95% CI −0.5 to 6.1; p = 0.09).

The highest demand occurred on "Unhealthy" days (n = 10), with 31.8 visits/day (SD 3.3), representing an increase of +8.3 visits/day compared with "Good" days (95% CI 4.1 to 12.5; $p < 0.001$). Overall, the between-band differences were statistically significant (ANOVA F = 8.62, $p < 0.001$), indicating that mean respiratory ED volume was not constant across worsening particulate exposure categories (Table 3).

Regression-based association estimates in Table 4 further quantified the timing of the $PM_{2.5}$ effect. In adjusted negative binomial models expressed per 10 μg/m³ increments in $PM_{2.5}$, the strongest association was observed for same-day exposure (Lag 0), where IRR = 1.032 (95% CI 1.018–1.046; $p < 0.001$), corresponding to a 3.2% increase in daily respiratory ED visits per 10 μg/m³ increase in $PM_{2.5}$.

The effect diminished with longer single-day lags: Lag 1 showed IRR = 1.019 (95% CI 1.005–1.033; p = 0.008), Lag 2 showed IRR = 1.014 (95% CI 0.999–1.029; p = 0.067), and Lag 3 was IRR = 1.009 (95% CI 0.994–1.024; p = 0.21), indicating attenuation and loss of statistical significance by Lag 2–3. Short moving averages suggested stronger cumulative short-term effects than single delayed lags: the 2-day moving average was associated with IRR = 1.037 (95% CI 1.020–1.054; $p < 0.001$) and the 3-day moving average with IRR = 1.041 (95% CI 1.022–1.061; $p < 0.001$), translating to 3.7% and 4.1% increases in daily respiratory ED visits per 10 μg/m³ increase in the respective averaged exposure metrics (Table 4).

Table 1. Demographic and diagnostic characteristics of respiratory ED visits (N = 1,680)

Characteristic	n (%)	Test statistic	p-value
Sex		$\chi^2 = 11.43$	0.001
Male	910 (54.2)		
Female	770 (45.8)		
Age group (years)		$\chi^2 = 162.8$	<0.001
<5	210 (12.5)		
5–17	260 (15.5)		
18–44	520 (31.0)		
45–64	420 (25.0)		
≥65	270 (16.1)		
Primary respiratory diagnosis		$\chi^2 = 284.6$	<0.001
Asthma exacerbation	410 (24.4)		
COPD exacerbation	360 (21.4)		
Pneumonia/LRTI	310 (18.5)		
Acute bronchitis/URTI	520 (31.0)		
Other respiratory diagnoses	80 (4.8)		

Table 2. Distribution of ambient pollutants, meteorological variables, and daily respiratory ED visits (n = 365 days)

Variable	Mean (SD)	Median (IQR)	Min–Max
$PM_{2.5}$ (μg/m³)	77.35 (47.72)	65.75 (49.47–88.43)	15.30–280.47
PM_{10} (μg/m³)	111.66 (68.53)	94.88 (68.26–131.56)	28.60–415.62
NO_2 (ppb)	41.52 (7.70)	41.06 (36.19–46.40)	22.91–61.12
SO_2 (ppb)	14.25 (3.61)	14.23 (11.85–16.39)	5.38–23.54
O_3 (ppb)	36.79 (8.59)	36.80 (31.43–42.50)	14.38–55.71
CO (ppm)	1.10 (0.23)	1.08 (0.94–1.24)	0.55–1.78
Temperature (°C)	20.42 (2.52)	20.37 (18.52–22.29)	14.35–26.84
Relative humidity (%)	66.31 (11.09)	66.69 (58.34–74.68)	34.19–95.00
Respiratory ED visits/day	26.48 (4.35)	26 (23–29)	14–39

Table 3. Mean daily respiratory ED visits by $PM_{2.5}$ exposure category

$PM_{2.5}$ Category	Days (n)	Mean visits/day (SD)	Mean difference vs. "Good" (95% CI)	p-value
Good	13	23.5 (5.1)	Reference	—
Moderate	21	24.9 (4.0)	+1.4 (–2.2 to 5.0)	0.43
Unhealthy for Sensitive Groups	76	26.3 (3.8)	+2.8 (–0.5 to 6.1)	0.09
Unhealthy	10	31.8 (3.3)	+8.3 (4.1 to 12.5)	<0.001

Table 4. Association between $PM_{2.5}$ and daily respiratory ED visits (per 10 μg/m³ increase)

Exposure metric	IRR (95% CI)	Percent change (%)	p-value
Lag 0	1.032 (1.018–1.046)	+3.2%	<0.001
Lag 1	1.019 (1.005–1.033)	+1.9%	0.008
Lag 2	1.014 (0.999–1.029)	+1.4%	0.067
Lag 3	1.009 (0.994–1.024)	+0.9%	0.21
2-day moving average	1.037 (1.020–1.054)	+3.7%	<0.001
3-day moving average	1.041 (1.022–1.061)	+4.1%	<0.001

Table 5. Diagnosis-specific associations between $PM_{2.5}$ (lag 0) and daily ED visits (per 10 μg/m³ increase)

Diagnosis category	IRR (95% CI)	Percent change (%)	p-value
Asthma exacerbation	1.048 (1.022–1.075)	+4.8%	<0.001
COPD exacerbation	1.039 (1.012–1.067)	+3.9%	0.004
Pneumonia/LRTI	1.018 (0.995–1.042)	+1.8%	0.12
Acute bronchitis/URTI	1.029 (1.006–1.053)	+2.9%	0.013

Diagnosis-specific models (Table 5) showed heterogeneity in the strength of association across respiratory outcomes. Asthma exacerbations demonstrated the largest same-day effect, with IRR = 1.048 per 10 μg/m³ $PM_{2.5}$ (95% CI 1.022–1.075; $p < 0.001$), equivalent to a 4.8% increase in asthma-related ED visits. COPD exacerbations also showed a significant increase (IRR = 1.039; 95% CI 1.012–1.067; $p = 0.004$), corresponding to a 3.9% rise per 10 μg/m³. Acute bronchitis/URTI visits increased by 2.9% per 10 μg/m³ (IRR = 1.029; 95% CI 1.006–1.053; p = 0.013).

In contrast, pneumonia/LRTI displayed a smaller and statistically non-significant association (IRR = 1.018; 95% CI 0.995–1.042; p = 0.12), suggesting either weaker acute triggering by $PM_{2.5}$ for this outcome category or lower statistical power for detecting short-lag effects (Table 5). Collectively, the pattern across Tables 3–5 indicates a coherent exposure–response gradient by pollution category, a predominantly immediate-to-short cumulative timing profile (Lag 0 and short moving averages), and stronger acute associations for obstructive airway exacerbations than for pneumonia/LRTI within this ED population.

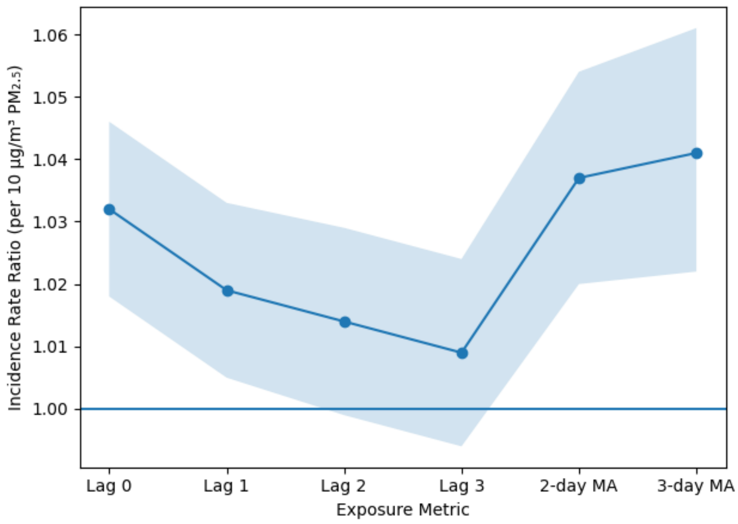

Figure 1 Short-Term $PM_{2.5}$ Exposure and Respiratory ED Visit Risk With 95% Confidence Bands

The figure demonstrates a clear short-term exposure–response profile between $PM_{2.5}$ and daily respiratory ED visits, expressed as incidence rate ratios (IRRs) per 10 μg/m³ increment with 95% confidence bands. The strongest immediate association is observed at Lag 0 (IRR 1.032; 95% CI 1.018–1.046), corresponding to a 3.2% increase in daily visits, followed by attenuation at Lag 1 (IRR 1.019; 95% CI 1.005–1.033) and further weakening at Lag 2 (IRR 1.014; 95% CI 0.999–1.029) and Lag 3 (IRR 1.009; 95% CI 0.994–1.024), where confidence intervals approach or cross the null (IRR = 1.0). Notably, cumulative exposure metrics show amplification rather than simple decay: the 2-day moving average yields IRR 1.037 (95% CI 1.020–1.054) and the 3-day moving average reaches IRR 1.041 (95% CI 1.022–1.061), representing 3.7% and 4.1% increases, respectively. The confidence band narrowing around Lag 0 and widening modestly at longer lags reflects greater precision for immediate effects and reduced stability for delayed estimates. Clinically, this pattern indicates that particulate pollution acts predominantly as an acute trigger with additive cumulative impact over consecutive exposure days, supporting the biological plausibility of rapid airway irritation and short-term inflammatory amplification leading to measurable increases in emergency respiratory demand.

DISCUSSION

This hospital-based time-series analysis demonstrates a consistent and temporally coherent association between short-term increases in ambient particulate pollution and emergency respiratory demand in an urban Punjab population. The principal quantitative finding was that each 10 μg/m³ increment in same-day $PM_{2.5}$ was associated with a 3.2% increase in daily respiratory ED visits (IRR 1.032; 95% CI 1.018–1.046), with a slightly stronger cumulative effect observed for the 3-day moving average (IRR 1.041; 95% CI 1.022–1.061). The attenuation of effect estimates across Lag 1–Lag 3, alongside amplification in short moving averages, supports a pattern of rapid symptom precipitation combined with additive exposure over consecutive polluted days. This timing profile is biologically plausible and aligns with evidence that the first hours to days following particulate exposure are critical for cardiorespiratory morbidity (18). The observed exposure–response gradient across $PM_{2.5}$ categories, with mean daily visits increasing from 23.5 on "Good" days to 31.8 on "Unhealthy" days ($p < 0.001$), further reinforces the internal consistency of the association.

The magnitude of effect observed in this study is comparable to, and in some cases slightly higher than, estimates reported in large international time-series and case time-series analyses. A recent multi-state study among insured adults in the United States demonstrated measurable increases in respiratory emergency utilisation even at relatively low $PM_{2.5}$ concentrations, with short-lag associations persisting below prior regulatory thresholds (7). Multi-city analyses of emergency visits have similarly documented acute increases in respiratory presentations associated with short-term pollutant fluctuations (8,19). Meta-analytic evidence confirms that particulate matter is associated with asthma emergency visits and hospital admissions, typically in the range of 1–5% increase per 10 $\mu g/m^3$ $PM_{2.5}$, depending on setting and lag specification (10). The 4.8% increase in asthma-related visits and 3.9% increase in COPD exacerbations per 10 $\mu g/m^3$ observed in the present study fall within this internationally reported range and are clinically meaningful in the context of already strained urban emergency systems.

Diagnosis-specific analyses revealed heterogeneity consistent with pathophysiological expectations. Obstructive airway conditions demonstrated stronger and statistically robust associations, whereas pneumonia/LRTI showed a smaller, non-significant same-day estimate (IRR 1.018; 95% CI 0.995–1.042). Experimental and clinical evidence suggests that particulate matter provokes bronchoconstriction, oxidative stress, and airway inflammation that can acutely exacerbate asthma and COPD (6). In contrast, infectious outcomes such as pneumonia may reflect more complex pathways involving host susceptibility and pathogen dynamics, potentially manifesting over longer or variable lag structures (11,20). The stronger immediate associations for asthma and COPD are therefore biologically coherent and consistent with mechanistic understanding of pollutant-induced airway hyperreactivity.

The cumulative short-term pattern observed, with higher effect sizes for 2-day and 3-day moving averages compared with single delayed lags, is also supported by emerging evidence that repeated exposure over consecutive days can amplify inflammatory responses and precipitate clinical decompensation (18). Reviews of intraday and short-lag effects emphasize that even brief exposure windows may carry measurable morbidity risk, particularly in susceptible populations (18). In the Punjab context, where seasonal smog episodes can sustain elevated $PM_{2.5}$ levels over multiple consecutive days, such cumulative dynamics are operationally relevant. The finding that ED demand rose by an average of 8.3 visits per day during the highest $PM_{2.5}$ band compared with cleaner days underscores the tangible health system impact of these exposure clusters.

The broader environmental context strengthens the plausibility and public health relevance of these findings. Pakistan's urban centers, particularly in Punjab, experience recurrent particulate pollution episodes driven by traffic emissions, industrial activity, brick kilns, and transboundary biomass burning (12). Regional analyses have projected substantial cardiopulmonary disease burden attributable to $PM_{2.5}$ across the country (15), and local studies from Lahore and Karachi have linked ambient pollution metrics with respiratory health indicators and asthma burden (13,14). The present study adds to this body of evidence by directly quantifying short-term, clinically verified ED respiratory morbidity in relation to measured daily pollutant concentrations, thereby translating environmental exposure metrics into healthcare demand signals.

From a health systems perspective, the observed 3–4% increase in daily respiratory ED visits per 10 μg/m³ increment in $PM_{2.5}$ has important operational implications. Given a baseline mean of 26.5 visits per day, a 50 μg/m³ increase during severe smog could theoretically correspond to a 15–20% relative rise in respiratory ED demand, representing approximately 4–5 additional patients per day. In settings with limited surge capacity, such increments can meaningfully strain staffing, oxygen supply, nebulisation capacity, and inpatient beds. Integrating real-time air quality forecasts into ED preparedness planning may therefore represent a pragmatic translation of environmental surveillance into clinical resource allocation.

Several methodological considerations merit discussion. First, exposure assessment relied on citywide ambient monitoring data, which may not fully capture individual-level exposure variability due to indoor environments, occupational exposures, or micro-spatial heterogeneity. Such non-differential misclassification would be expected to bias effect estimates toward the null rather than exaggerate associations. Second, although models adjusted for temperature, humidity, day of week, and seasonality, residual confounding by viral epidemics or unmeasured behavioral adaptations during smog episodes cannot be entirely excluded. Third, the ecological time-series framework supports inference about short-term temporal associations but does not establish individual-level causality. Nonetheless, the coherence of timing, dose–response pattern, and diagnosis-specific gradients—combined with alignment to extensive international evidence (3,7,10,19)—strengthens causal plausibility under established environmental epidemiology criteria.

Despite these limitations, the study has notable strengths. It uses a full year of continuous data to capture seasonal variability, applies lag-specific and moving-average exposure metrics, employs negative binomial regression to address overdispersion, and presents diagnosis-specific analyses that enhance clinical interpretability. The consistent exposure–response gradient across pollution bands and regression-based increments supports internal validity. Importantly, the findings contextualize global evidence within a high-exposure South Asian urban setting, where pollutant concentrations frequently exceed health-based guideline values.

In conclusion, short-term increases in ambient $PM_{2.5}$ were associated with measurable and clinically meaningful increases in emergency respiratory visits in this urban Punjab population, particularly for asthma and COPD exacerbations. The temporal pattern suggests that particulate pollution acts primarily as an acute trigger with additive cumulative impact over consecutive days of exposure. These findings reinforce the need for integrated environmental health strategies that combine emission reduction, public advisories, and hospital-level preparedness to mitigate preventable respiratory morbidity in high-pollution urban environments.

CONCLUSION

In this hospital-based ecological time-series study conducted in urban Punjab, short-term increases in ambient particulate air pollution—particularly $PM_{2.5}$—were consistently associated with higher emergency respiratory visit counts, with the strongest effects observed on the same day of exposure and across short moving averages. Each 10 μg/m³ increase in $PM_{2.5}$ corresponded to a 3–4% rise in daily respiratory ED demand, with more pronounced effects for asthma and COPD exacerbations than for pneumonia/LRTI, indicating that obstructive airway diseases may be especially sensitive to acute particulate exposure. The exposure–response gradient across worsening pollution categories and the temporally coherent lag structure reinforce biological plausibility and clinical relevance. These findings translate environmental monitoring data into measurable healthcare system impact, underscoring that recurrent smog episodes in Punjab are not only environmental events but predictable drivers of acute respiratory morbidity. Strengthening pollution-control policies, integrating air-quality alerts into emergency preparedness protocols, and prioritizing protection for vulnerable populations are therefore essential components of reducing avoidable respiratory emergencies in high-exposure urban settings.

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DECLARATIONS

Ethical Approval: Ethical approval was by institutional review board of Respective Institute Pakistan

Informed Consent: Informed Consent was taken from participants.

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Concept: MAW; Design: AN, MTA; Data Collection: AS, WS; Analysis: MZ, UAS; Drafting: MAW, AN

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