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Acute Respiratory Impacts of Fine Particulate Matter (PM2.5) in Kinshasa: Epidemiological Evidence and Pulmonological Implications

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Research Article

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Abstract

Background

Air pollution, particularly fine particulate matter ($PM_{2.5}$), constitutes an escalating threat to respiratory health worldwide. In African megacities such as Kinshasa, where systematic air quality monitoring remains limited, clinicians are increasingly confronted with respiratory conditions attributable to pollution exposure. The $PM_{2.5}$ concentrations in Kinshasa consistently exceed the World Health Organization (WHO) air quality guidelines; however, locally generated clinical evidence on associated health impacts remains markedly scarce.

Objective

To evaluate the relationship between acute exposure to $PM_{2.5}$ and respiratory emergency admissions among adults in Kinshasa from 2018–2022.

Methods

A hospital-based, time-stratified case-crossover study (unidirectional and bidirectional symmetric) was conducted using medical records from the Monkole Hospital Center. Daily $PM_{2.5}$ concentrations were derived from PurpleAir and QuantAQ Modulair sensors and corrected GEOS-CF (GEOS Composition Forecast) model outputs via the bias correction model (BCM). Associations between $PM_{2.5}$ and respiratory admissions were estimated via conditional logistic regression and generalized linear mixed models (GLMMs) while adjusting for temperature and relative humidity.

Results

Among the 13,049 adult admissions, acute respiratory diseases and infections accounted for 93.3%, whereas exacerbations of chronic respiratory diseases accounted for 6.7%. The mean daily $PM_{2.5}$ concentrations ranged from 46 to 50 µg/m³. In multivariate analyses (BI3), an increase of 10 µg/m³ in $PM_{2.5}$ was associated with a significant increase in respiratory admissions (adjusted OR = 1.01; 95% CI [1.00–1.03]) within 0–5 days post-exposure. The strongest associations were observed among adults aged 35–64 years and residents of the Tshangu and Funa districts.

Conclusion

The present study provides the first clinical and epidemiological evidence of the short-term impact of $PM_{2.5}$ on respiratory morbidity in Kinshasa. These findings underscore the increasing burden of pollution-related respiratory diseases and emphasize the imperative for pulmonologists to incorporate environmental exposure assessment into routine clinical practice. This should be accompanied by enhanced air quality surveillance systems and the implementation of comprehensive prevention strategies across sub-Saharan Africa.

Introduction

The respiratory system is a primary pathway of exposure to airborne contaminants, particularly chemical pollutants such as particulate matter (PM), which are widely recognized as indirect indicators of ambient air pollution [1]. Robust evidence links both short- and long-term PM exposure to adverse health outcomes [2]. The major PM constituents include sulfates, nitrates, ammonia, sodium chloride, black carbon, mineral dust, and water. Other air pollutants regulated by the WHO include carbon monoxide (CO), ozone (O_3), nitrogen dioxide (NO_2), and sulfur dioxide (SO_2) [3, 4].

A large body of evidence has shown that exposure to $PM_{2.5}$, both in the short term (spanning from hours to days) and in the long term (from months to years), contributes to adverse health effects [5]. Long-term exposure has been linked to cardiovascular and

respiratory diseases, metabolic disorders, various cancers, impaired renal and bone function, and adverse perinatal and neurodevelopmental outcomes [5–8].

Short-term effects include increased respiratory symptoms, decreased lung function, infectious episodes among otherwise healthy individuals, and the exacerbation of preexisting respiratory and/or cardiac conditions, which may ultimately lead to death [9]. Studies in North America, Europe, and Asia have documented associations between short-term $PM_{2.5}$ exposure and emergency visits, hospitalizations, and mortality related to respiratory diseases, including asthma, bronchitis, pneumonia, and chronic obstructive pulmonary disease (COPD) [10–14]. A recent study conducted in Vietnam provided robust evidence that short-term exposure to PM2.5 is associated with an increase in adverse cardiovascular and respiratory events among urban inhabitants of Ho Chi Minh City [15].

In Africa, few studies have examined the association between acute $PM_{2.5}$ exposure and respiratory health. Research conducted in South Africa, particularly child panel studies and local time series analyses, has highlighted short-term links between increases in $PM_{2.5}$ levels and the worsening of respiratory symptoms or hospital admissions [16–20]. Nevertheless, many African countries remain understudied, and regional reviews emphasize the need for locally conducted time series or case crossover analyses [21].

Kinshasa, the capital city of the Democratic Republic of the Congo in Central Africa, is currently the third most populous city in Africa after Lagos and Cairo, with an estimated population of 16 million inhabitants [22, 23]. Kinshasa is also affected by anthropogenic air pollution, which is driven mostly by road traffic with an aging vehicle fleet, waste incineration, and other sources [22]. A study conducted in Kinshasa in 2021 reported annual average $PM_{2.5}$ concentrations of 54, 43, and 35 µg/m³ in 2018, 2019, and 2020, respectively [24]. These values consistently exceed the WHO guideline value of 15 µg/m³ [24, 25]. Seasonal peaks were observed between June and September, corresponding to the near absence of rainfall, with daily concentrations exceeding 100 µg/m³ on some days [22]. Poor air quality was further confirmed in 2022, with the highest $PM_{2.5}$ levels occurring during the dry season [22]. To date, no study has examined the association between acute $PM_{2.5}$ exposure and respiratory health in the general population of Kinshasa, although an occupational study among public transport drivers has been conducted [26].

Given the growing concern of air pollution in Kinshasa and the paucity of epidemiological evidence in the general population, it is essential to undertake studies examining the relationship between short-term $PM_{2.5}$ exposure and the onset or exacerbation of respiratory diseases among adults residing in the city.

Materials and methods

Study Design and Methodological Framework

We conducted a retrospective, hospital-based case-crossover study using both unidirectional (UNI) and symmetric bidirectional (SBI) designs [15] in the city of Kinshasa (Democratic Republic of the Congo). The study period extended from March 4, 2018, to December 31, 2022.

In the unidirectional design (UNI), control days always precede the case day. For each case, four control days were selected at 7, 14, 21, and 28 days before the event. This structure controls for weekly cyclic variations (weekday vs. weekend effects).

In the symmetric bidirectional design (SBI), control days are selected symmetrically before and after the case day. Three configurations were tested:

- BI1 (bidirectional 1): ±7 and ± 14 days
- BI2 (bidirectional 2): ±14 and ± 21 days
- BI3 (bidirectional 3): ±21 and ± 28 days

The use of postevent control days is justified by the absence of any reverse causal relationship between $PM_{2.5}$ exposure and emergency admissions, thus reducing time-trend bias. The symmetric bidirectional design minimizes overlap bias while adjusting for seasonal and long-term pollution trends.

To explore the temporal dynamics of acute effects, we examined two types of lag structures: single-day lags (lag 0 to lag 7), which represent exposure on the day of the event and up to seven days prior, and moving averages (ma3 to ma7), which represent cumulative exposure over multiple days (day 0 to day 3, up to day 7).

In alignment with the methodological approach described by Tran et al. (2024) [15], the sample size was calculated via DuPont's formula for matched case–control designs. Assuming a $PM_{2.5}$ exposure prevalence of 0.5, a case–control correlation of 0.5, and an expected odds ratio (OR) of 1.1, the minimum required sample was 9,937 subjects (with four control days per case) to achieve 90% statistical power at $\alpha = 5\%$. Our final dataset comprised 13,049 subjects, thus ensuring more than sufficient power to detect small to moderate associations.

Study Setting

Kinshasa covers a total area of 9,965 km² and is located in the southwestern DRC (latitude 4.325°, longitude 15.322°) at an elevation of 281 m above sea level. Kinshasa has a tropical Aw_4 climate characterized by a long rainy season lasting approximately eight months (mid-September to mid-May, interrupted by a short dry spell in January–February), followed by a major dry season [27].

- Administratively, as shown in Table 1, Kinshasa is a decentralized entity comprising 24 communes grouped into 4 districts, Lukunga, Funa, Mont-Amba, and Tshangu, which differ in size, population density, urbanization level, living standards, and infrastructure quality [28].

Table 1
Districts of Kinshasa and their respective communities

Districts	Communes
Lukunga (northwest)	- Gombe (administrative and economic center), - Barumbu, - Kinshasa, - Kintambo, - Lingwala, - Ngaliema, - Mont-Ngafula, - Selembao.
Funa (central-south), densely populated with high commercial activity.	1. Bandalungwa 2. Bumbu 3. Kalamu 4. Kasa-Vubu 5. Makala 6. Ngiri-Ngiri
Mont-Amba (east-central), notable for academic and governmental institutions.	1. Kisenso 2. Lemba 3. Limete 4. Matete 5. Ngaba
Tshangu (easternmost), the largest district, including the airport and extensive rural zones	1. Kimbanseke 2. Masina 3. Ndjili 4. Nsele 5. Maluku

Air Pollution Data

Air pollution data were obtained from multiple sources by combining **in situ measurements** and satellite observations through collaboration with the *Lamont-Doherty Earth Observatory* (Columbia University, USA) and the *Health, Environment, and Policy Program* at the *NYU Marron Institute (New York University)*.

$PM_{2.5}$ Monitoring

The daily $PM_{2.5}$ concentrations in Kinshasa were obtained from measurement campaigns between 2018 and 2022 via several complementary devices:

- First dataset (March 2018–August 2020): Collected by a network of PurpleAir PA-II-SD sensors installed at four sites: the U.S. Embassy (Gombe), Cité de Bellevue (Kintambo), Université Pédagogique Nationale (Ngaliema), and ERAIFT/University of

Kinshasa (Lemba). These low-cost sensors continuously measure PM_1, $PM_{2.5}$, PM_{10}, and meteorological parameters (temperature, humidity, pressure). The data were calibrated against the reference BAM-1020 monitor via multiple linear regression (MLR) and random forest models, which improved the accuracy ($r^2 = 0.96$; MAE = 3.4 μg/m³) [24]. The mean daily $PM_{2.5}$ concentration was approximately 43.5 μg/m³.

- Second dataset (March–December 2022): Obtained via the QuantAQ Modulair device installed at the U.S. Embassy (Gombe). The Modulair combines a Plantower PMS5003 nephelometer and an Alphasense OPC-N3 optical particle counter to measure PM_1, $PM_{2.5}$, PM_{10}, and gases (CO, CO_2, NO, NO_2, O_3). Modulair data strongly correlated with the BAM-1020 measurements ($r^2 = 0.758$; MAE = 6.97 μg/m³; mean bias = 3.2%). After MLR correction, the MAE was reduced to 5.53 μg/m³, with a corrected mean daily concentration of 40.3 μg/m³ [22].
- Gap-filling data (October 2020–March 2022): $PM_{2.5}$ concentrations were obtained from the NASA GEOS Composition Forecast (GEOS-CF) model, which couples the GEOS meteorological system with the GEOS-Chem atmospheric chemistry module, providing near-real-time global forecasts at a 25 km resolution [29, 30]. Model outputs were adjusted via a bias correction model (BCM) based on local PurpleAir observations ($R^2 = 0.74$), ensuring the continuity and reliability of the $PM_{2.5}$ time series [31]. Figure 1 illustrates the visualization of our $PM_{2.5}$ data.

Meteorological Data

Daily temperature and relative humidity (RH) data for Kinshasa (October 2020–March 2022) were extracted from the ERA5 reanalysis dataset of the *Copernicus Climate Change Service (C3S)* (Hersbach et al., 2023). ERA5 provides hourly and daily reanalysis data with a horizontal resolution of 31 km. The variables used were 2-meter air temperature (T2m) and 2-meter dew point temperature, from which the RH was calculated via the standard psychrometric formula validated in ERA5 postprocessing. These high-quality datasets ensure temporal and spatial consistency with $PM_{2.5}$ measurements for integrated air pollution meteorology analysis.

Health Data

Initially, five tertiary emergency hospitals were selected: *Cliniques Universitaires de Kinshasa*, *Clinique Ngaliema*, *De Smet Hospital of Kinsuka (DHK)*, *Centre Hospitalier Initiative Plus d'Olive Lembe* (Gombe and Bibwa sites), and *Centre Hospitalier de Monkole*. However, only the Monkole Hospital Center provided consistent, high-quality, detailed electronic health records. The data used in this study were extracted from Monkole's hospital information system, which has been officially recognized as a general reference hospital since 2014.

Ethical approval

was obtained, and health data were retrospectively collected in June 2024. All the data were fully anonymized by the hospitals' data management office before being made available to us.

Acute respiratory events included both acute respiratory infections and acute exacerbations of chronic respiratory diseases, which were classified according to the International Classification of Diseases, 11th Revision (ICD-11, codes CA00–CB7Z) (WHO, 2022). The extracted variables included the patient's municipality of residence, admission and discharge dates, sex, age, and ICD-11 diagnostic codes.

Inclusion and exclusion criteria

Only Kinshasa residents (based on address) were included. Repeated visits for the same condition within four days after discharge were treated as readmissions and excluded to limit autocorrelation.

Statistical Analyses

Two analytical approaches were used to assess the linear association between daily mean $PM_{2.5}$ concentrations and emergency respiratory events.

1. Crude models: Conditional logistic regression (CLR) models were used to estimate the relationship between $PM_{2.5}$ levels (independent variable) and the occurrence of an acute respiratory event (dependent variable: no event = control day, event present = case day). The results are expressed as unadjusted odds ratios (UORs) and 95% confidence intervals (CIs) per 10 μg/m³ increase in $PM_{2.5}$.
2. Adjusted models: Associations were further adjusted for daily air temperature, relative humidity, and wind speed via generalized linear mixed models (GLMMs). These models provided adjusted odds ratios (AORs) and 95% CIs for the same $PM_{2.5}$ increment.

Meteorological parameters were systematically included as covariates. GLMMs account for repeated measures within individuals, modelling random and fixed effects to capture intersubject heterogeneity. Both models were applied under UNI and BI1-BI3 designs, using corresponding lag and control structures. Bidirectional designs reduce long-term time-trend bias by symmetrically integrating pre- and postevent control days, improving adjustment for seasonal and meteorological variations. Model selection for stratified analyses was based on the likelihood ratio test ($p < 0.05$) for crude models or the lowest Akaike information criterion (AIC) for adjusted models.

Stratified analyses were performed by age group (< 35, 35–64, ≥ 65 years), sex, type of respiratory event (acute infection vs. chronic disease exacerbation), and residential district (Funa, Mont-Amba, Lukunga, Tshangu). The sensitivity analyses were restricted to the dry and wet seasons.

A p value < 0.05 was considered statistically significant. Benjamini–Hochberg correction was applied to adjust for multiple comparisons (Benjamini & Hochberg, 1995). All analyses were performed via R software (version 4.4.3), with the clogit function from the survival package for CLR and the glmer function from the lme4 package for GLMM.

Results

Demographic characteristics of patients, daily air pollution, and meteorological conditions in the districts of Kinshasa between March 2018 and December 2022

Table 2
Patient demographic characteristics, daily air pollution levels, and weather conditions by district in Kinshasa (March 2018–December 2022)

Variables	**N (%) or Mean (S.D.)**	
Sex :		
- Female (n, (%))	6617	(50,7%)
- Male (n, (%))	6431	(49,3%)
Age group (years) :		
- < 35 (n, (%))	4743	(36,3%)
- 35–64 (n, (%))	6127	(47.0%)
- > 65 (n, (%))	2178	(16,7%)
Type of acute respiratory event:		
- Acerbation of chronic respiratory diseases (n, (%))	124	(6,7%)
- Acute respiratory illness or infection (n, (%))	1725	(93,3%)
District of residence :		
- FUNA (n, (%))	3025	(23,2%)
- LUKUNGA (n, (%))	2194	(16,8%)
- MONT-AMBA (n, (%))	7053	(54,0%)
- TSHANGU (n, (%))	776	(6.0%)
Season :		
- Dry (n, (%))	4479	(34,3%)
- Rainy (n, (%))	8569	(65,7%)
Air pollution and meteorological variables :		
Average daily $PM_{2.5}$ concentration ($\mu g/m^3$), (mean, SD):		
- FUNA District	48,2	(36,9)
- LUKUNGA District	46,3	(34,5)
- MONT-AMBA District	47,9	(37,2)
- TSHANGU District	50,2	(43,6)
Average daily air temperature (°C), (mean, SD):		
- FUNA District	25,9	(1,9)
- LUKUNGA District	25,8	(1,9)
- MONT-AMBA District	25,9	(1,9)
- TSHANGU District	25,8	(2,0)
Average daily Relative humidity (%), (mean, SD):		
- FUNA District	78,8	(5,4)
- LUKUNGA District	79,1	(5,5)
- MONT-AMBA District	78,9	(5,4)

Variables	N (%) or Mean (S.D.)	
- TSHANGU District	79,3	(5,3)

Table 2 presents the distributions of patients by sex, age group, type of acute respiratory event, and district of residence, as well as the mean daily $PM_{2.5}$ concentrations, temperature, and relative humidity by district. The data cover the period from March 3, 2018, to December 31, 2022, in Kinshasa.

The total sample was nearly evenly distributed between females (50.7%) and males (49.3%), reflecting a balanced sex ratio. Middle-aged adults (35–64 years) represented the majority (47%), followed by individuals under 35 years of age (36.3%) and those aged 65 years and above (16.7%), indicating that the working-age population was particularly affected.

Most acute pulmonary conditions were acute respiratory infections or diseases (93.3%), whereas exacerbations of chronic respiratory diseases were relatively uncommon (6.7%).

Regarding the distribution of acute respiratory events by district, the findings indicated that Mont-Amba accounted for the majority of cases (54%), followed by Funa (23.2%), Lukunga (16.8%), and Tshangu (6%).

Approximately two-thirds of hospital admissions occurred during the rainy season (65.7%), whereas acute episodes were less frequent during the dry season (34.3%), suggesting a notable climatic influence on the incidence of respiratory diseases.

The mean daily $PM_{2.5}$ concentrations were extremely high, ranging from 46.3 to 50.2 µg/m³ across districts. Tshangu recorded the highest level (50.2 µg/m³), followed by Funa and Mont-Amba. All these values far exceed the WHO annual guideline of 5 µg/m³, exposing the population to significant health risks.

The mean daily temperature was approximately 26°C, with little variation among districts, and the relative humidity remained relatively stable at approximately 79%, which is consistent with the city's tropical climate.

This analysis highlights persistent exposure to elevated particulate pollution across all districts of Kinshasa, with a worsening trend during the rainy season.

Trends of daily average fine particulate matter ($PM_{2.5}$) concentrations in Kinshasa between March 2018 and December 2022
Figure 2 shows the temporal evolution of daily mean fine particulate matter ($PM_{2.5}$) concentrations in Kinshasa between March 2018 and December 2022, which is based on three complementary data sources: blue represents measurements from PurpleAir PA-II-SD sensors (March 2018–August 2020), red represents estimates from the GEOS composition forecasting (GEOS-CF) model (October 2020–March 2022), and green represents measurements from the QuantAQ Modulair device (March-December 2022).

During the covered period, daily average $PM_{2.5}$ concentrations ranged between 10 and 300 µg/m³, with marked variability across seasons and years.

The blue series (PurpleAir period) shows alternating high peaks (> 100 µg/m³) and more stable phases at approximately 30–60 µg/m³. The highest peaks occurred during the dry months (June–August and December–February), periods known for increased atmospheric stagnation and pollutant accumulation.

The red series (GEOS-CF model) provides temporal continuity between the two measurement campaigns. The simulated concentrations reproduce the observed seasonal dynamics, with an average level slightly above 40 µg/m³, which is consistent with values recorded by the PurpleAir and QuantAQ sensors. The peak observed from late 2021 to early 2022 reached nearly 250 µg/m³, reflecting an episode of intense pollution likely linked to unfavourable meteorological conditions for dispersion or increased biomass burning.

The green series (QuantAQ Modulair) indicates concentrations generally between 20 and 60 µg/m³, which is slightly lower than those reported in previous periods. This apparent decline may be due to more favourable dispersion conditions, improved instrumental precision, or a temporary reduction in local emissions. Nevertheless, the values remain far above WHO recommendations, emphasizing a persistently high background pollution level.

Despite being derived from different instruments, the three datasets exhibit strong temporal coherence and methodological consistency. The transitions between sources (low-cost sensors, satellite-based modelling, and professional-grade instruments) show no major discontinuities, confirming the reliability of bias-corrected data integration.

Overall, the figure clearly illustrates the chronic prevalence of air pollution in Kinshasa, characterized by recurrent episodes of severe exceedance of health-based limits. This study underscores the importance of a hybrid air quality monitoring approach that combines local measurements (PurpleAir, QuantAQ) with atmospheric modelling (GEOS-CF) to address observational gaps in sub-Saharan African urban contexts. The results also highlight the urgent need for locally adapted air quality management policies to mitigate health risks associated with prolonged exposure to $PM_{2.5}$.

Analysis of Daily Emergency Admissions

Temporal analysis of daily emergency admissions: fluctuations, stability, and exceptional events (2018–2022)

Figure 3 presents the temporal evolution of the daily number of patients admitted for medical emergencies in Kinshasa between January 2018 and December 2022. The data show marked day-to-day variability, with rapid fluctuations and frequent peaks exceeding 15 admissions per day. The most common range of daily admissions lies between 5 and 10 patients, indicating a sustained baseline demand for emergency care.

Several distinct periods display heightened volatility in admissions, likely corresponding to epidemic waves, seasonal high-risk periods, or major public health events.

The overall pattern reflects the influence of short-term epidemiological factors, including seasonal viral epidemics, pollution episodes, and meteorological variations, on hospital admissions. The persistence of elevated daily admissions throughout the entire study period highlights the chronic structural burden of respiratory health risks in Kinshasa.

Recurrent fluctuations may also indicate a seasonal modulation linked to the rainy and dry seasons. Together, these findings suggest a cyclical and enduring pressure on emergency medical services, underscoring the need for strengthened preparedness and adaptive response capacity within hospital systems to cope with peaks in respiratory morbidity.

Distribution of acute respiratory events according to the ICD-11 classification among patients admitted to medical emergencies in Kinshasa

Table 3 presents the distribution of diagnostic categories for acute respiratory events according to the International Classification of Diseases, 11th Revision (ICD-11) and their frequency within the study cohort in Kinshasa.

Exacerbations of chronic respiratory diseases accounted for a minority of cases (6.7%), mainly asthma (4.1%), and, less frequently, chronic obstructive pulmonary disease (COPD, 0.3%) and diffuse interstitial pneumonias (2.3%). This pattern highlights the predominance of asthma among chronic respiratory conditions in the local context.

The vast majority of emergency admissions were related to acute respiratory diseases or infections (93.3%), including influenza (21.7%), COVID-19 (confirmed: 10.1%; unconfirmed: 20.4%), pneumonias (bacterial: 4.1%; unspecified: 5.7%; bronchopneumonia: 1.9%), acute respiratory distress syndrome (ARDS, 7.5%), and upper respiratory infections such as sinusitis (4.2%), tonsillitis (6.4%), and allergic rhinitis (3.5%).

Infectious diagnoses remained predominant, particularly during epidemic periods (e.g., influenza, COVID-19), although a proportion of cases were related to inflammatory or respiratory complications such as ARDS or pleurisy.

Lower respiratory tract infections (pneumonia, bronchitis, ARDS) are the leading causes of acute admissions, underscoring the impact of viral and bacterial infections on respiratory morbidity. While less frequent, upper respiratory infections represented a notable share of emergency cases, particularly sinusitis, allergic rhinitis, and tonsillitis.

Patients with isolated respiratory symptoms such as cough and dyspnea accounted for less than 1% of the total admissions.

Table 3
Distribution of acute respiratory events according to the ICD-11 classification among patients admitted to medical emergencies in Kinshasa.

Type of acute respiratory event	Chapiter	Main_Category	Main_Diagnosis_ICD-11	ICD-11 Code	number of cases	%	number of cases	%
Exacerbations of chronic respiratory illnesses:							**124**	**6.7**
	Diseases of the respiratory system (CA00 – CB7Z)	Certain lower respiratory tract diseases (CA20-CA2Z)	Asthma, unspecified	CA23.Z	76	4.1		
			Chronic obstructive pulmonary disease (COPD)	CA22	6	0.3		
		Respiratory diseases principally affecting the lung interstitium (CB00-CB0Z)	Diffuse interstitial lung disease, unspecified	CB03.Y	42	2.3		
Acute respiratory illness or infection:							**1725**	**93.3**
	Certain infectious or parasitic diseases (1A00–1H0Z):	Influenza (1E30–1E32)	Influenza, unspecified	1.00E + 32	401	21.7		
	Codes for special purposes (RA00 – RA26):	International provisional assignment of new diseases of uncertain aetiology and emergency use (RA00 – RA09):	COVID-19, confirmed viral infection	RA01.0	187	10.1		
			COVID-19, unconfirmed	RA01.1	377	20.4		
	Diseases of the ear or mastoid process (AA00 – AC0Z):	Diseases of middle ear or mastoid (AA80 - AB1Z):	Acute otitis media	AB00	20	1.1		
	Diseases of the respiratory system (CA00 – CB7Z):	Lung infections/Infections pulmonaires (CA40-CA4Z) :	Acute bronchitis	CA42	10	0.5		
			Acute bronchopneumonia	CA40.1	35	1.9		
			Bacterial pneumonia, unspecified	CA40.0	76	4.1		

Type of acute respiratory event	Chapiter	Main_Category	Main_Diagnosis_ICD-11	ICD-11 Code	number of cases	%	number of cases	%
Exacerbations of chronic respiratory illnesses:							**124**	**6.7**
			Pneumonia, unspecified	CA40.Z	106	5.7		
		Pleural, diaphragm or mediastinal disorders (CB20-CB2Z)	Pleural effusion	CB27	31	1.7		
		Respiratory diseases principally affecting the lung interstitium (CB00-CB0Z)	Acute Respiratory Distress Syndrome – ARDS	CB00	138	7.5		
		Upper respiratory tract disorders (CA00-CA0Z):	Acute nasopharyngitis	CA00	20	1.1		
			Acute pharyngitis	CA02	37	2.0		
			Acute sinusitis	CA01	78	4.2		
			Acute tonsillitis	CA03	118	6.4		
			Allergic rhinitis, unspecified	CA08.0	65	3.5		
	Symptoms, signs or clinical findings, not elsewhere classified (MA00 – MH2Y)	Symptoms, signs or clinical findings of the respiratory system (MD10-MD6Y):	Cough (symptom, unspecified)	MD12	16	0.9		
			Dyspnoea	MD11.5	10	0.5		

Effects of $PM_{2.5}$ Exposure on Emergency Visits for Acute Respiratory Events Across Lag Structures and Case-Crossover Designs

To evaluate the individual-level associations between short-term $PM_{2.5}$ exposure and respiratory morbidity in Kinshasa, we compared the performance of four case-crossover designs: unidirectional (UNI) and bidirectional (BI1, BI2, BI3). Figure 3 summarizes both unadjusted and adjusted odds ratios (OR_{10}, 95% CI) for a 10 µg/m³ increase in $PM_{2.5}$ under each design. Given that acute effects of $PM_{2.5}$ may occur immediately or with delay, both single-day lags and moving averages were analysed to assess the timing and cumulative magnitude of exposure effects.

The forest plot (Fig. 4) provides a synthesis of estimated associations between a 10 µg/m³ increase in $PM_{2.5}$ and the risk of emergency admission for acute respiratory events across statistical models and case-crossover configurations (UNI, BI1–BI3). Two analytical approaches are displayed: (1) the unadjusted conditional logistic regression (CLR) model and (2) the adjusted generalized linear mixed model (GLMM), which accounts for meteorological confounders.

Unidirectional case-crossover design (UNI)

As shown in Table 1A and Fig. 3, crude CLR models revealed a strong and statistically significant association between $PM_{2.5}$ exposure and acute respiratory events ($p < 0.05$ across all lags). The unadjusted OR_{10} values ranged from 1.06–1.08, with the strongest association observed for the 7-day moving average (MA7: OR_{10} = 1.08 [1.06–1.10]). This finding indicates that a 10 µg/m³

increase in the mean $PM_{2.5}$ over the previous 7 days was associated with an 8% increase in the risk of emergency admission compared with that on control days.

However, estimates from unidirectional designs may be inflated by temporal trends, such as gradual increases in $PM_{2.5}$ levels over time, which can lead to apparent risk overestimation. The attenuation observed in adjusted models (GLMMs) supports this hypothesis.

The adjusted GLMM models, which controlled for temperature, humidity, and interindividual variability, provided more robust and less confounded estimates. The adjusted OR_{10} values ranged from 1.02 to 1.03 across lag structures, with a value of 1.03 [1.02–1.04] for MA7, corresponding to a 3% increase in risk per 10 µg/m³ increase in $PM_{2.5}$ after adjustment.

Although attenuated, all associations remained highly significant even after Benjamini Hochberg correction (p_adj_BH). These findings confirm that short-term $PM_{2.5}$ exposure is a significant trigger of acute respiratory events, independent of meteorological variations.

In summary, while crude estimates suggested a 7–8% increase in risk, adjusted models yielded a more conservative yet consistent effect of 2–3%, reflecting the influence of confounding factors. The persistence of significant ORs across lags (lags 0–7) suggests both immediate and sustained effects of particulate pollution on respiratory morbidity.

To test the robustness of these findings and minimize the temporal bias inherent to the unidirectional design, we conducted additional analyses using bidirectional case-crossover designs (BI1–BI3), incorporating control days both before and after the event.

Symmetric Bidirectional Case-Crossover Design (BI)

Bidirectional Plan 1 (BI1)

As shown in Table 2A, the results from the first bidirectional design (BI1) indicate no statistically significant association between $PM_{2.5}$ exposure and the occurrence of acute respiratory events.

In this design, control days were selected 7 days before and 14 days after the case day, effectively controlling for slow temporal trends and gradually evolving confounders. Compared with the unidirectional (UNI) plan, the BI1 results clearly contrast: all raw p values were much higher than 0.05 (ranging from 0.22 to 0.98), and the Benjamini–Hochberg adjusted p values remained elevated (0.83–0.97), confirming the absence of a statistically significant association after multiple testing correction.

In the crude CLR models, the OR_{10} values ranged from 1.00 to 1.01, whereas in the adjusted GLMM models, all estimates were uniformly 1.00 across the lag structures. After adjusting for meteorological covariates (temperature and relative humidity), the $PM_{2.5}$ effect disappeared entirely, confirming that there was no residual association.

Thus, the significant association observed under the unidirectional design (UNI) is likely explained by temporal confounding, where concurrent variations in air pollution and respiratory admissions reflect a noncausal common trend. The BI1 design, which is more robust to such biases, consistently shows no evidence of a causal short-term relationship between a 10 µg/m³ increase in $PM_{2.5}$ and acute respiratory admissions.

Bidirectional Plan 2 (BI2)

The results from the second bidirectional design (BI2), shown in Table 3A, confirm and reinforce the previous observations. Like BI1, this design rigorously controls for long-term temporal bias, using symmetrical control days 14 days before and 21 days after the event date. This approach neutralizes slow seasonal variations or background exposure trends unrelated to the event while testing a slightly broader control window.

The crude CLR models indicated a weak association, which was not confirmed after statistical correction. The OR_{10} values ranged between 1.01 and 1.02, suggesting a marginal 1–2% increase in risk per 10 µg/m³ increase in $PM_{2.5}$.

After applying the Benjamini–Hochberg correction, all adjusted p values became nonsignificant, with the lowest being 0.157. This suggests that initially significant results were likely false positives arising from multiple testing (20 total comparisons: 10 lags × 2 models).

Adjusted GLMM models, accounting for temperature and humidity, confirmed this lack of association. The adjusted OR_{10} values remained between 1.00 and 1.01, with high p values (0.15–0.76), indicating that there was no statistically significant relationship after adjustment.

These findings imply that the significant effect observed under the UNI design was likely a statistical artifact related to temporal trend bias. The bidirectional designs (BI1 and BI2), recognized for their ability to control such bias, consistently show no robust independent associations between short-term $PM_{2.5}$ increases and acute respiratory events.

In summary, at this stage, no solid evidence supports a short-term triggering effect of a 10 µg/m³ $PM_{2.5}$ increase in acute respiratory admissions.

Bidirectional Plan 3 (BI3)

The results from the third bidirectional design (BI3), shown in Table 4A, are particularly important, as they reveal a significant association between $PM_{2.5}$ exposure and acute respiratory events, an association that was not detected in BI1 or BI2.

This bidirectional configuration, while effectively controlling long-term temporal trends, also preserves the ability to detect true short-term effects.

Unlike BI1 and BI2, the crude CLR model under BI3 identified a clear and highly significant association. The OR_{10} values ranged from 1.02–1.03 across all lags and moving averages. The strongest effects were observed for lag0 and for the 3-day and 7-day moving averages (ma3, ma7), all with $OR_{10} = 1.03$. All corresponding p values were very low ($p < 0.006$), indicating a statistically robust association.

After multiple testing correction, the adjusted p values (p_adj_BH) for key lags (Lag0, MA3, MA7) remained well below 0.05 (~ 0.0005), confirming the robustness and stability of the association.

Adjusted GLMM models incorporating temperature and relative humidity also confirmed an independent effect of $PM_{2.5}$ exposure. Although attenuated, the OR_{10} values decreased slightly (from 1.03–1.01), yet several lags (Lag0–Lag5) and the MA3 average remained statistically significant (p_adj_BH < 0.05). For Lag0 (GLMM), $OR_{10} = 1.01$ [1.00–1.03], indicating that a 10 µg/m³ increase in $PM_{2.5}$ on the same day was associated with an approximately 1% greater risk of emergency admission for an acute respiratory event.

Longer lags (Lag6, Lag7, MA7) lost significance after adjustment (p_adj_BH > 0.05), suggesting that the causal effect of $PM_{2.5}$ is primarily immediate, extending over approximately 0–5 days.

Stratified Analyses at Lag0 across Case-Crossover Designs

Analyses were stratified by age, sex, type of respiratory disease, residential district, and season to examine potential differences in susceptibility and temporal bias.

Table 4 presents both unadjusted (UOR) and adjusted (AOR) odds ratios with 95% confidence intervals for a 10 µg/m³ increase in $PM_{2.5}$ across the unidirectional (UNI) and bidirectional symmetric (BI1–BI3) designs.

Stratified analysis confirmed the presence of temporal confounding in the unidirectional design and revealed that the most credible causal effect, although small, was primarily detected by the BI3 plan. Overall, the general trend observed in the nonstratified analyses was consistent across all the subgroups (age, sex, disease type, residential zone, and season).

Interpretation is based primarily on adjusted estimates from BI3, which is considered the most robust, as it combines effective temporal bias control with adjustment for short-term meteorological confounding.

Age

As shown in Table 4, age-stratified analysis revealed notable variation in the association between $PM_{2.5}$ exposure and the risk of acute respiratory admission. Among individuals aged 35–64 years, the effect was statistically significant (AOR_{10} = 1.015 [1.005–1.025]), indicating a 1.5% increase in risk per 10 μg/m³ increase in $PM_{2.5}$. In contrast, the association was marginal among individuals < 35 years (AOR_{10} = 1.010 [0.999–1.020]) and nonsignificant among those ≥ 65 years (AOR_{10} = 1.006 [0.996–1.016]).

This pattern suggests that middle-aged adults (35–64 years) may be more susceptible to short-term particulate exposure, possibly reflecting greater cumulative exposure due to occupational and urban mobility factors, a higher prevalence of chronic comorbidities (e.g., asthma, chronic bronchitis), or behavioral/environmental differences (e.g., transport use, housing conditions, proximity to traffic).

In younger individuals, the lack of significance may indicate greater physiological resilience or more variable exposure, whereas in older adults, the result may reflect selective survival or reduced mobility, limiting exposure to pollution peaks.

Overall, these findings indicate that the health effects of $PM_{2.5}$ are not homogeneous across age groups and that the working-age population represents a key epidemiological target for air pollution health prevention strategies.

Table 4

Unadjusted odds ratios and adjusted odds ratios (95% confidence intervals) per 10 µg/m^3 increase in $PM_{2.5}$, unidirectional and symmetric bidirectional case crossover designs, at lag 0, stratified by age, sex, disease, residential district and season.

			Unidirectional case-crossover design		**Symmetric bidirectional case-crossover design**					
			UNI		**BI1**		**BI2**		**BI3**	
N°	**Strata**	**Subgroup**	**UOR_{10} (CI 95%)**	**AOR_{10} (CI 95%)**	**UOR_{10} (CI 95%)**	**AOR_{10} (CI 95%)**	**UOR_{10} (CI 95%)**	**AOR_{10} (CI 95%)**	**UOR_{10} (CI 95%)**	**AOR_{10} (CI 95%)**
1.	**Age** :									
		< 35 years	1.055 (1.035–1.075)	1.008 (0.998–1.018)	1.046 (1.026–1.066)	1.001 (0.991–1.011)	1.063 (1.043–1.083)	1.014 (1.004–1.024)	1.032 (1.012–1.052)	1.010 (0.999–1.020)
		35–64 years	1.044 (1.024–1.064)	1.013 (1.003–1.023)	1.029 (1.009–1.049)	1.010 (0.999–1.020)	1.060 (1.040–1.080)	1.001 (0.991–1.011)	1.046 (1.026–1.066)	1.015 (1.005–1.025)
		≥ 65 years	1.050 (1.030–1.070)	1.007 (0.997–1.017)	1.058 (1.038–1.078)	1.008 (0.998–1.018)	1.051 (1.031–1.071)	1.008 (0.998–1.018)	1.048 (1.028–1.068)	1.006 (0.996–1.016)
2.	**Sex** :									
		Male	1.044 (1.024–1.064)	1.013 (1.003–1.023)	1.046 (1.026–1.066)	1.013 (1.003–1.023)	1.056 (1.036–1.076)	1.014 (1.004–1.024)	1.048 (1.028–1.068)	1.006 (0.996–1.016)
		Female	1.036 (1.016–1.056)	1.013 (1.003–1.023)	1.041 (1.021–1.061)	1.001 (0.991–1.011)	1.056 (1.036–1.076)	1.012 (1.002–1.022)	1.071 (1.051–1.091)	1.022 (1.012–1.032)
3.	**Respiratories diseases group** :									
		Acute respiratory diseases and infections	1.033 (1.013–1.053)	1.006 (0.996–1.016)	1.059 (1.039–1.079)	1.005 (0.995–1.015)	1.070 (1.050–1.090)	1.006 (0.996–1.016)	1.065 (1.045–1.085)	1.033 (1.013–1.053)
		Exacerbations of chronic respiratory illnesses	1.034 (1.014–1.054)	1.007 (0.997–1.017)	1.050 (1.030–1.070)	1.007 (0.997–1.017)	1.041 (1.021–1.061)	1.021 (1.011–1.031)	1.056 (1.036–1.076)	1.034 (1.014–1.054)
4.	**Residence** :									
		TSHANGU District	1.061 (1.041–1.081)	1.015 (1.005–1.025)	1.057 (1.037–1.077)	0.997 (0.987–1.007)	1.069 (1.049–1.089)	1.004 (0.994–1.014)	1.065 (1.045–1.085)	1.016 (1.006–1.026)
		FUNA District	1.043 (1.023–1.063)	1.008 (0.998–1.018)	1.050 (1.030–1.070)	1.009 (0.999–1.019)	1.061 (1.041–1.081)	1.012 (1.002–1.022)	1.045 (1.025–1.065)	1.020 (1.010–1.030)
		LUKUNGA District	1.027 (1.007–1.047)	1.011 (1.001–1.021)	1.051 (1.031–1.071)	1.004 (0.994–1.014)	1.041 (1.021–1.061)	1.010 (1.000-1.020)	1.049 (1.029–1.069)	1.005 (0.995–1.015)
		MONT AMBA District	1.045 (1.025–1.065)	1.010 (0.999–1.020)	1.047 (1.027–1.067)	1.015 (1.005–1.025)	1.040 (1.020–1.060)	1.008 (0.998–1.0181)	1.059 (1.039–1.079)	1.010 (1.000-1.020)
5.	**Season** :									

			Unidirectional case-crossover design		Symmetric bidirectional case-crossover design					
			UNI		BI1		BI2		BI3	
N°	Strata	Subgroup	UOR_{10} (CI 95%)	AOR_{10} (CI 95%)	UOR_{10} (CI 95%)	AOR_{10} (CI 95%)	UOR_{10} (CI 95%)	AOR_{10} (CI 95%)	UOR_{10} (CI 95%)	AOR_{10} (CI 95%)
		Dry	1.04 (1.020–1.060)	1.001 (0.991–1.011)	1.028 (1.008–1.048)	1.019 (1.009–1.029)	1.052 (1.032–1.072)	1.006 (0.996–1.016)	1.058 (1.038–1.078)	1.007 (0.997–1.017)
		Rainy	1.051 (1.031–1.071)	1.008 (0.998–1.018)	1.037 (1.017–1.057)	1.002 (0.992–1.012)	1.043 (1.023–1.063)	1.001 (0.991–1.011)	1.072 (1.052–1.092)	1.012 (1.002–1.022)

UNI: unidirectional case-crossover design.

BI 1 to BI 3: Symmetric bidirectional case–crossover design with fixed-interval reference selections including 7 and 14 days (BI 1), 14 and 21 days (BI 2), and 21 and 28 days (BI 3) before and after the case day, respectively.

UOR (95% CI): unadjusted odds ratio (95% confidence interval) determined via conditional logistic regression (CLR) analyses.

AOR (95% CI): adjusted odds ratio (95% confidence interval) determined via generalized linear mixed model (GLMM) analyses adjusted for air temperature and relative humidity.

Bold indicates statistical significance after false discovery rate adjustments (adjusted Benjamini–Hochberg p values < 0.05).

Dry season: Mid-May - Mid-September

Rainy season: Mid-September - mid-May

Sex

As shown in Table 4, sex-stratified analysis revealed a marked difference in the response to $PM_{2.5}$ exposure. Among women, bidirectional plan 3 (BI3) showed a statistically significant association between a 10 µg/m³ increase in $PM_{2.5}$ and the risk of emergency admission for an acute respiratory event (AOR_{10} = 1.022 [1.012–1.032]), corresponding to an estimated 2.2% increase in hospital admission risk. In contrast, among men, the association was not significant (AOR_{10} = 1.006 [0.996–1.016]), indicating that there was no clear relationship between exposure and respiratory event risk in this subgroup.

This difference may reflect greater biological susceptibility among women, potentially related to anatomical and hormonal factors, as well as behavioral or environmental differences in exposure, such as time spent outdoors, indoor ventilation practices, or socioenvironmental factors influencing contact with air pollution.

Types of Respiratory Diseases

As shown in Table 4, analysis by disease type revealed a statistically significant association between $PM_{2.5}$ exposure and the risk of emergency admissions for respiratory events, regardless of the condition considered. For acute respiratory diseases, the association was AOR_{10} = 1.023 [1.013–1.053], indicating an ≈ 2.3% increase in risk per 10 µg/m³ increase in $PM_{2.5}$. For chronic respiratory diseases, the association was slightly stronger (AOR_{10} = 1.034 [1.014–1.054]), corresponding to an ≈ 3.4% increase in risk.

Although moderate in magnitude, these effects are consistent and robust, suggesting that fine particulate pollution contributes to both acute episodes and exacerbations of chronic respiratory diseases, with a slightly greater impact on the latter.

Residential Districts

As shown in Table 4, spatial analysis revealed marked heterogeneity in $PM_{2.5}$ effects across residential districts. Under the BI3 design, significant associations were found in FUNA (AOR_{10} = 1.020 [1.010–1.030]) and TSHANGU (AOR_{10} = 1.016 [1.006–1.026]), indicating stronger exposure response relationships in these areas. Conversely, LUKUNGA (AOR_{10} = 1.005 [0.995–1.015]) and MONT-AMBA (AOR_{10} = 1.010 [1.000-1.020]) showed weaker or borderline effects, suggesting a less consistent association.

This spatial variability may reflect local differences in pollution sources (industrial emissions, road traffic, domestic burning), unmeasured socioeconomic factors, or variations in the chemical composition and toxicity of $PM_{2.5}$, particularly in densely populated districts such as FUNA and TSHANGU.

Seasons

As shown in Table 4, seasonal analysis revealed a clear difference in the intensity and significance of the $PM_{2.5}$ health association. During the rainy season, the effect was statistically significant (AOR_{10} = 1.012; 95% CI: 1.002–1.022), indicating a measurable increase in the risk of respiratory admissions per 10 µg/m³ increase in $PM_{2.5}$. During the dry season, however, the relationship was not significant (AOR_{10} = 1.007; 95% CI: 0.997–1.017), suggesting a weaker or context-dependent association. This seasonal asymmetry may be explained by differences in atmospheric dispersion, seasonal variations in particle composition, or increased population vulnerability during the rainy season, when high humidity and concurrent respiratory infections could amplify pollutant effects.

Discussion

The objective of this study was to examine the association between short-term $PM_{2.5}$ exposure and the onset or exacerbation of respiratory diseases among adults living in cities. The analyses conducted provide strong evidence of an association between acute $PM_{2.5}$ exposure and respiratory morbidity among adults in Kinshasa. In both crude CLR models and adjusted GLMM models and across unidirectional and bidirectional approaches, daily $PM_{2.5}$ concentrations remained consistently associated with hospital admissions for acute respiratory conditions.

In the unidirectional design, while crude (CLR) estimates suggested a 7–8% increase in risk per 10 µg/m³ increase in $PM_{2.5}$, the adjusted GLMM models indicated a more moderate yet homogeneous effect of approximately 2–3% depending on the lag structure. In the bidirectional design, the comparative evaluation of the analytical configurations revealed that the BI3 approach provided the most methodologically robust balance, effectively controlling temporal bias while avoiding the overadjustment observed in BI1 and BI2. Estimates from BI3 suggested an approximately 1% increase in risk per 10 µg/m³ increase in $PM_{2.5}$, confirming an acute, immediate, and coherent association that strengthens the plausibility of a short-term causal link between fine particulate pollution and respiratory morbidity.

With respect to the stratified analyses, our results highlight substantial heterogeneity in susceptibility within the population. The estimates derived from the BI3 model, the most robust configuration, indicate that the impact of $PM_{2.5}$ is concentrated within specific subgroups, notably women, middle-aged adults (35–64 years), and residents of the FUNA and TSHANGU districts, with increased vulnerability during the rainy season.

To guide the interpretation of our findings, the discussion is organized into three analytical components. First, we address the epidemiological dimensions, focusing on the internal validity of the associations, the consistency of effect estimates across statistical frameworks (CLR, GLMM) and analytical designs (unidirectional, bidirectional), and the extent to which temporal confounding was controlled. Second, we examine the pulmonary and pathophysiological mechanisms that plausibly underpin the observed acute effects, drawing on established pathways involving inflammation, oxidative stress, and airway reactivity. Third, we consider the context of environmental epidemiology, including differential susceptibility across population subgroups, spatial heterogeneity, seasonal modifiers, and implications for exposure assessment in low-monitoring environments such as Kinshasa.

Epidemiological dimensions

The use of the symmetric bidirectional case–crossover design (BI3) minimized temporal bias and detected a short-term effect of $PM_{2.5}$ on respiratory morbidity. A 10 µg/m³ increase in $PM_{2.5}$ was associated with a 1% increase in the risk of admission for acute respiratory disease, mainly within five days following exposure.

These findings are consistent with the international literature. Studies conducted in China, Spain, and the United States have reported similar increases (0.8% to 1.2%) in respiratory hospitalizations for comparable variations in $PM_{2.5}$ [11–13]. Despite geographic variations, short-term particulate pollution effects on respiratory health are universal and quantifiable, even at relatively low exposure increments.

Moreover, at Lag 0 in the unidirectional analysis (after adjustment), our results revealed that a 10 µg/m³ increase in $PM_{2.5}$ was associated with a 3% increased risk (OR_{10}: 1.03 [1.02–1.04]) of admission for acute respiratory disease. These results align with those of a South African study where in a unidirectional design at Lag 0, hospitalizations rose by 2.7% (95% CI: 0.6–4.9) per 10 µg/m³ increase in total $PM_{2.5}$ [18]. Similarly, a meta-analysis by Zheng et al. across 37 studies revealed a 2.3% increase (95% CI: 1.5–3.1%) in asthma cases per 10 µg/m³ increase in $PM_{2.5}$ exposure [32]. While that meta-analysis focused exclusively on asthma, both our study and that of Howlett-Downing included a broader spectrum of respiratory conditions, encompassing acute respiratory infections (pneumonia, bronchitis, etc.) and other chronic respiratory diseases such as asthma, COPD, and interstitial lung diseases.

With respect to sex, our analyses (BI3) indicate a greater risk among women than Bryan et al.'s study in Lima, which reported no significant sex differences [33]. This discrepancy may reflect contextual factors. In a local study conducted among a sample of 1,154 households in Kinshasa, traditional fuels (wood and charcoal) remained overwhelmingly predominant compared with modern energy sources (gas and electricity) [34], and traditional sociocultural roles resulted in women being more exposed to indoor air pollution. This combined domestic and environmental exposure may heighten women's susceptibility to the acute respiratory effects of $PM_{2.5}$.

This study also investigated geographic variations among districts in Kinshasa. The findings revealed an overrepresentation of cases in Funa and Tshangu. This likely mirrors urban environmental inequities related to road traffic, domestic combustion, and population density [16, 21]. Overall, these results confirm the classic epidemiological profile of the effects of short-term air pollution on respiratory health in emerging African megacities.

Pulmonology and pathophysiology mechanisms

Our findings confirmed the deleterious effects of acute exposure to $PM_{2.5}$ on respiratory health, which is in line with the international literature. Most cases (93.3%) involved acute respiratory diseases, whereas exacerbations of chronic diseases accounted for a smaller proportion (6.7%). This profile highlights the preponderant role of acute respiratory infections, which are dominated by influenza, COVID-19, and pneumonia, in emergency admissions in Kinshasa. Asthma remains the leading chronic condition among exacerbations. The predominance of infectious diagnoses and the high frequency of acute respiratory distress syndrome (ARDS) reflect the vulnerability of the respiratory system in this urban context, which is likely exacerbated by environmental and socioeconomic factors.

The strongest association was observed for exacerbations of chronic respiratory diseases, which is consistent with Bryan et al. [33] and the meta-analysis by Zeng et al. [32], which also revealed significant effects of daily $PM_{2.5}$ increases in emergency visits for asthma and COPD. These studies highlight particularly pronounced effects among middle-aged adults (19–64 years), a pattern also observed in our sample. In Kinshasa, this increased vulnerability may be explained by more frequent occupational exposure, higher urban mobility, and a greater prevalence of chronic respiratory comorbidities. The high incidence of acute respiratory diseases among middle-aged adults likely reflects the combined influence of poor air quality and dense urban conditions.

Conversely, no significant effect was observed among older adults (≥ 65 years). This may reflect the underutilization of emergency services in this age group rather than true physiological resistance to $PM_{2.5}$ effects.

The association between $PM_{2.5}$ exposure and acute respiratory events is supported by several well-documented pathophysiological mechanisms.

Alveolar deposition and pulmonary penetration

Because their diameter is < 2.5 *μm*, fine particles penetrate deep into the airways, reaching terminal bronchioles and alveoli. They evade mucociliary clearance and deposit at the alveolar level, where they are phagocytosed by macrophages. This deposition triggers cellular activation, impairs alveolar macrophage function, and stimulates the production of proinflammatory mediators [35].

Inflammation and oxidative stress

$PM_{2.5}$ is rich in heavy metals, soot carbon, and organic compounds capable of generating reactive oxygen species (ROS). Oxidative stress activates the NF-κB pathway, increasing the production of proinflammatory cytokines (IL-1β, IL-6, and TNF-α) and chemokines (IL-8), leading to local and systemic inflammation. These processes promote lipid peroxidation, antioxidant depletion, and proinflammatory signalling, resulting in alveolar oedema and increased capillary permeability, thereby facilitating respiratory infections (pneumonia, acute bronchitis) and ARDS. These processes may extend beyond the lungs to affect the cardiovascular system and other organs [7, 36, 37].

Exacerbation of chronic respiratory diseases

In individuals with asthma or chronic obstructive pulmonary disease (COPD), $PM_{2.5}$ exacerbates symptoms via several mechanisms: (1) bronchial hyperresponsiveness induced by direct epithelial irritation; (2) increased parasympathetic tone leading to bronchoconstriction; and (3) stimulation of Toll-like receptors (TLR-2, TLR-4) and the interleukin-8 pathway, causing neutrophil influx into the bronchial lumen and wall thickening [7, 36, 37]. One publication suggested potential mechanisms whereby air pollution through oxidative stress, inflammation, and telomere shortening may trigger acute exacerbations or promote the progression of interstitial lung diseases in genetically susceptible individuals [36].

Systemic and extrapulmonary effects

A fraction of ultrafine particles crosses the alveolar–capillary barrier and enters the bloodstream, triggering systemic inflammation, endothelial dysfunction, and platelet activation [38, 39]. These effects can aggravate cardiovascular comorbidities and contribute to respiratory and cardiopulmonary mortality.

These pathophysiological data reinforce the biological plausibility of a causal link between $PM_{2.5}$ exposure and respiratory morbidity, which is consistent with the immediate effects observed in this study (lags of 0–5 days).

On the basis of these findings, we conclude that the following conclusions are clearly needed:

1. integrate environmental prevention measures into national strategies for managing chronic respiratory diseases; and
2. strengthen the training of healthcare professionals regarding the health impacts of air pollutants.

Environmental epidemiology context

The average $PM_{2.5}$ concentration observed in Kinshasa (48 μg/m³) far exceeds the WHO annual guideline value of 15 μg/m³ [25], confirming the magnitude of urban pollution. Peaks observed during the dry season result from biomass burning, vehicular traffic, and atmospheric stagnation, as shown in other African studies [19, 40]. The observed geographical and seasonal disparities underscore the need for targeted interventions both to reduce pollution sources and to improve healthcare accessibility, particularly in the most affected districts. From a public health standpoint, even a 1% increase in the risk of respiratory admissions per 10 μg/m³ increase in $PM_{2.5}$ translates into a considerable burden on hospital services, given the large size of the exposed population (16 million inhabitants). These findings underscore the need for the following:

1. Reinforce continuous air quality monitoring in Kinshasa;
2. implement policies aimed at reducing emissions from traffic and household combustion.

Limitations and future directions

The key limitations of this study include its monocentric design, reliance on data from a single hospital (Monkole), and the limited spatial coverage of air pollution monitoring sensors. In addition, individual exposure to both indoor and occupational air pollution, which are recognized contributors to respiratory morbidity, could not be assessed. Nevertheless, because the Monkole Hospital

Center serves emergency patients originating from all four districts of Kinshasa, the findings may reasonably reflect city-wide patterns of acute respiratory morbidity. Importantly, the case-crossover design relies on within-individual comparisons, whereby each patient serves as their own control, thereby ensuring high internal validity despite the monocentric study setting. Furthermore, the integration of ground-based $PM_{2.5}$ measurements with bias-corrected GEOS-CF model estimates helped to reduce gaps in environmental exposure assessment. The application of robust and complementary statistical approaches further strengthens the validity of the findings.

Future multicenter studies incorporating additional air pollutants (such as NO_2, O_3, and PM_{10}), broader sensor networks, and advanced spatiotemporal modelling frameworks are warranted to better characterize cumulative exposure and respiratory health risks in rapidly urbanizing tropical African settings.

Conclusion

This study provides the first clinical evidence in Kinshasa that short-term exposure to fine particulate matter ($PM_{2.5}$) is associated with an increased risk of emergency admissions for acute respiratory conditions among adults. By integrating hospital-based individual data with daily air pollution measurements, our findings demonstrate that ambient $PM_{2.5}$ acts as an acute trigger of respiratory morbidity, with effects occurring predominantly within the first five days following exposure.

Although the estimated increase in risk at the individual level is modest (approximately 1% per 10 µg/m³ increase in $PM_{2.5}$ under the most robust bidirectional case–crossover design), this effect is clinically meaningful in the context of persistently high pollution levels. In a large urban population chronically exposed to concentrations far exceeding international guidelines, small relative increases in risk can translate into a substantial number of additional emergency visits, placing sustained pressure on emergency departments and pulmonology services.

From a methodological standpoint, the use of a symmetric bidirectional case-crossover design allowed for rigorous control of temporal confounding while preserving sensitivity to immediate exposure effects. The consistency of the associations observed under the BI3 configuration supports the plausibility of a true short-term effect of $PM_{2.5}$ on respiratory health rather than an artifact of seasonal or long-term trends.

These findings highlight the importance of incorporating environmental exposure assessment into routine respiratory care. Pulmonologists and emergency physicians should be aware that peaks in ambient $PM_{2.5}$ may precipitate acute respiratory infections and exacerbate chronic respiratory diseases, particularly among middle-aged adults, women, and residents of highly polluted urban districts. Integrating air quality information into clinical decision-making, patient education, and anticipatory management strategies may help reduce preventable emergency admissions.

In conclusion, air pollution should be recognized as a modifiable risk factor for acute respiratory morbidity in urban African settings. Strengthening collaboration between clinicians, public health authorities, and environmental monitoring systems is essential to improve early detection of pollution-related health risks and to guide both clinical practice and preventive interventions aimed at reducing the respiratory disease burden.

Declarations

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Author contributions: MNK, DMM, FB, BOK, TTT, GKM, and JMKN contributed to the study conception and design. The materials were prepared via MNK., DMM, DMW, ZDT, NL, KC, PIK and PT. Formal data analysis and evaluation were performed via MNK and DMM. A critical data review was performed by MNK, DMM, DMW, ZDT, NL, KC, GKM and JMKN. The first draft of the manuscript

was written by MNK, and all the authors commented on the previous versions of the manuscript. All the authors have read and approved the final manuscript.

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Availability of data and material: The data used in this study are not publicly available due to institutional confidentiality policies, but they may be obtained from the corresponding author upon reasonable request and with authorization from the Monkole Hospital Center.

Ethical Statement: This study was conducted in accordance with the ethical principles of the Declaration of Helsinki, the CIOMS (Council for International Organizations of Medical Sciences) International Ethical Guidelines (2016), and applicable national regulations. This was an observational, noninterventional study based solely on retrospective individual-level data fully anonymized by the hospitals' data management office prior to transfer to the research team. Anonymization was performed via irreversible methods that removed all direct and indirect identifiers.

Given the absence of identifiable information and the noninterventional nature of the research, individual informed consent was waived, in line with CIOMS recommendations and local ethical requirements.
Ethical approval was obtained from the Ethics Committee of the *Centre de Formation et d'Appui Sanitaire (CEFA)*, affiliated with the Monkole Hospital Center (Approval No. N/Réf: 010/CEFA/MONKOLE/CEL/2024, 20 November 2024).

Consent to participate: This study used retrospective individual-level data that had been fully anonymized by the hospitals' data management office before being provided to the investigators. No identifiable information was accessible, and no patients were contacted or involved directly.
In accordance with the CIOMS International Ethical Guidelines (2016) and relevant national regulations, individual informed consent was waived.

Consent to Publish: The manuscript does not contain any identifiable personal data. All information used for the study was previously anonymized. Therefore, consent for publication was not needed.

Competing interests: The authors declare that they have no competing interests.

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Figures

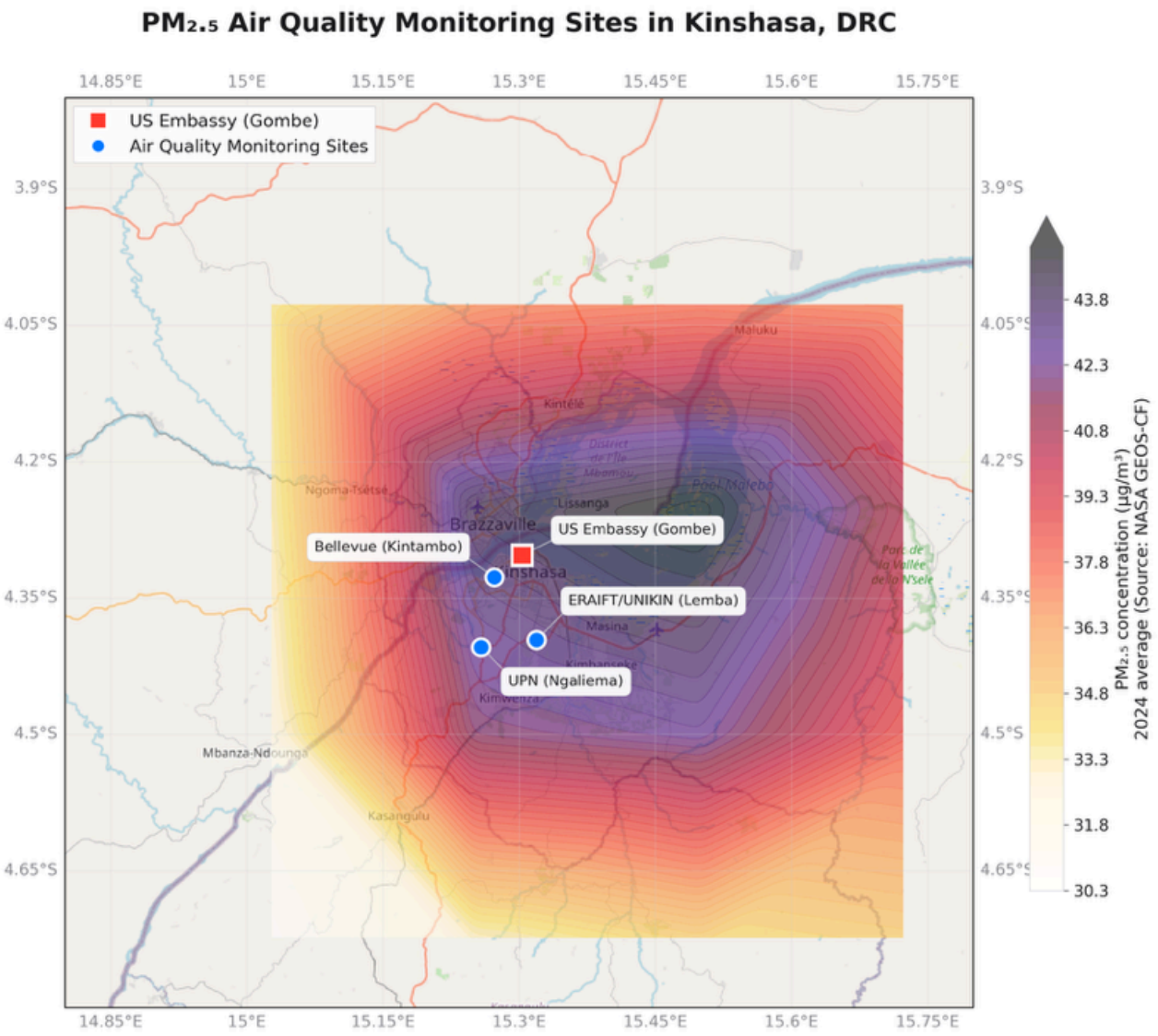


Figure 1

$PM_{2.5}$ air quality monitoring sites in Kinshasa, DRC, and overlay of GEOS-CF annual mean surface PM2.5 estimates

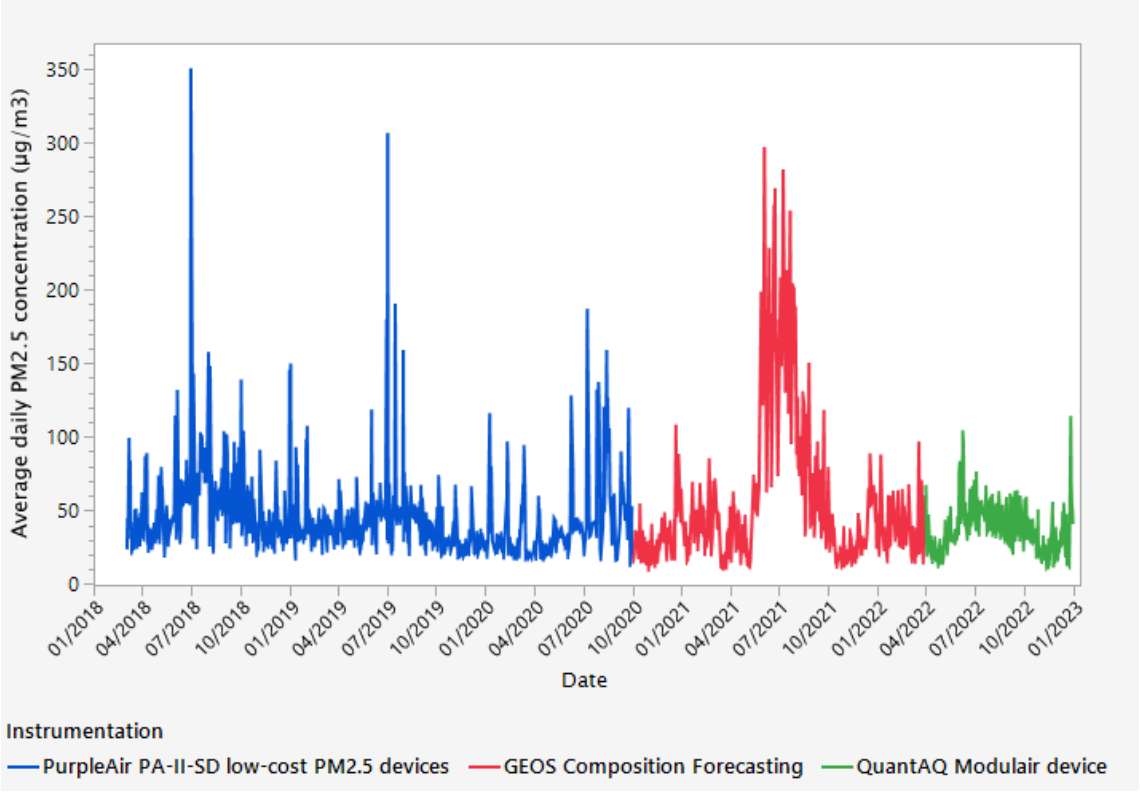


Figure 2

Trends of daily average $PM_{2.5}$ concentrations in Kinshasa (2018–2022). Sources: PurpleAir PA-II-SD, GEOS Composition Forecasting, and QuantAQ Modulair.

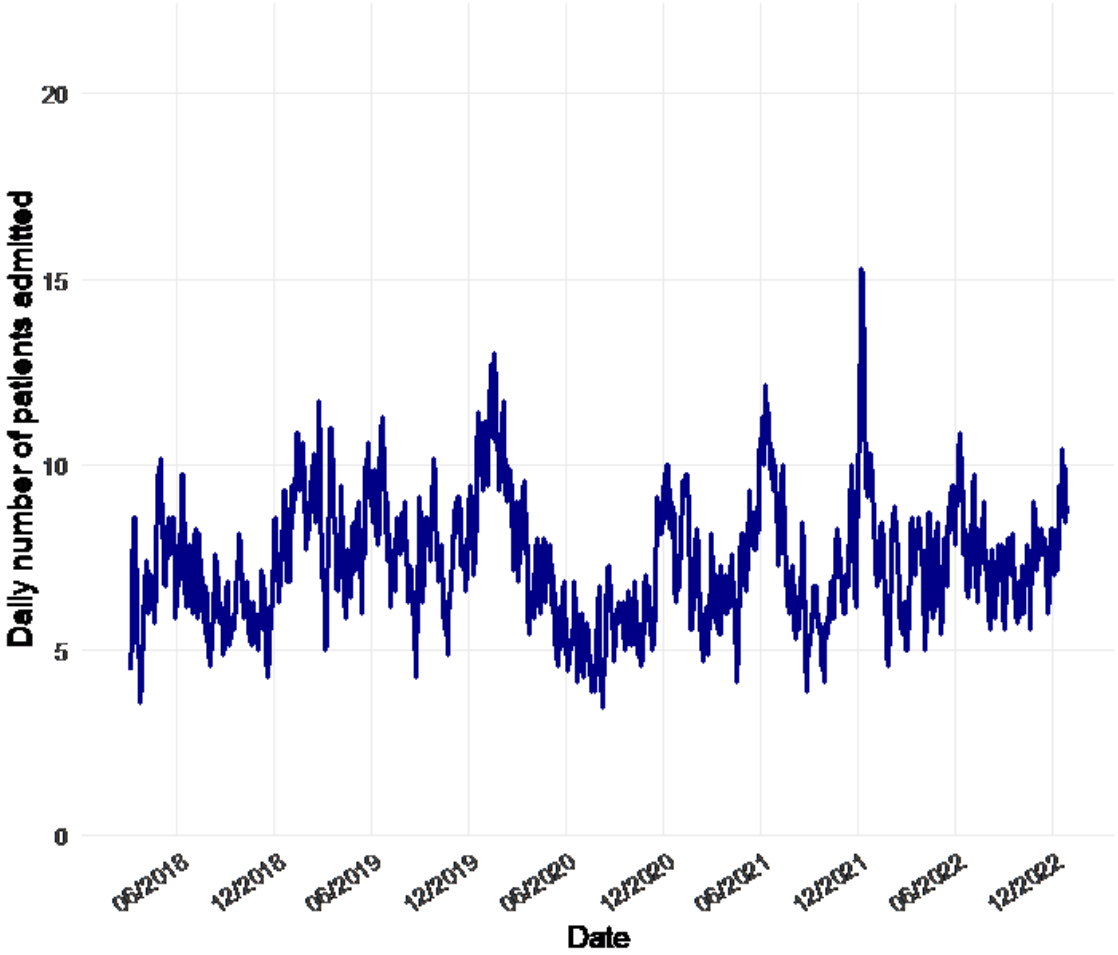


Figure 3

Daily variation in the number of patients admitted for acute respiratory events to medical emergency departments in Kinshasa (March 2018--December 2022).

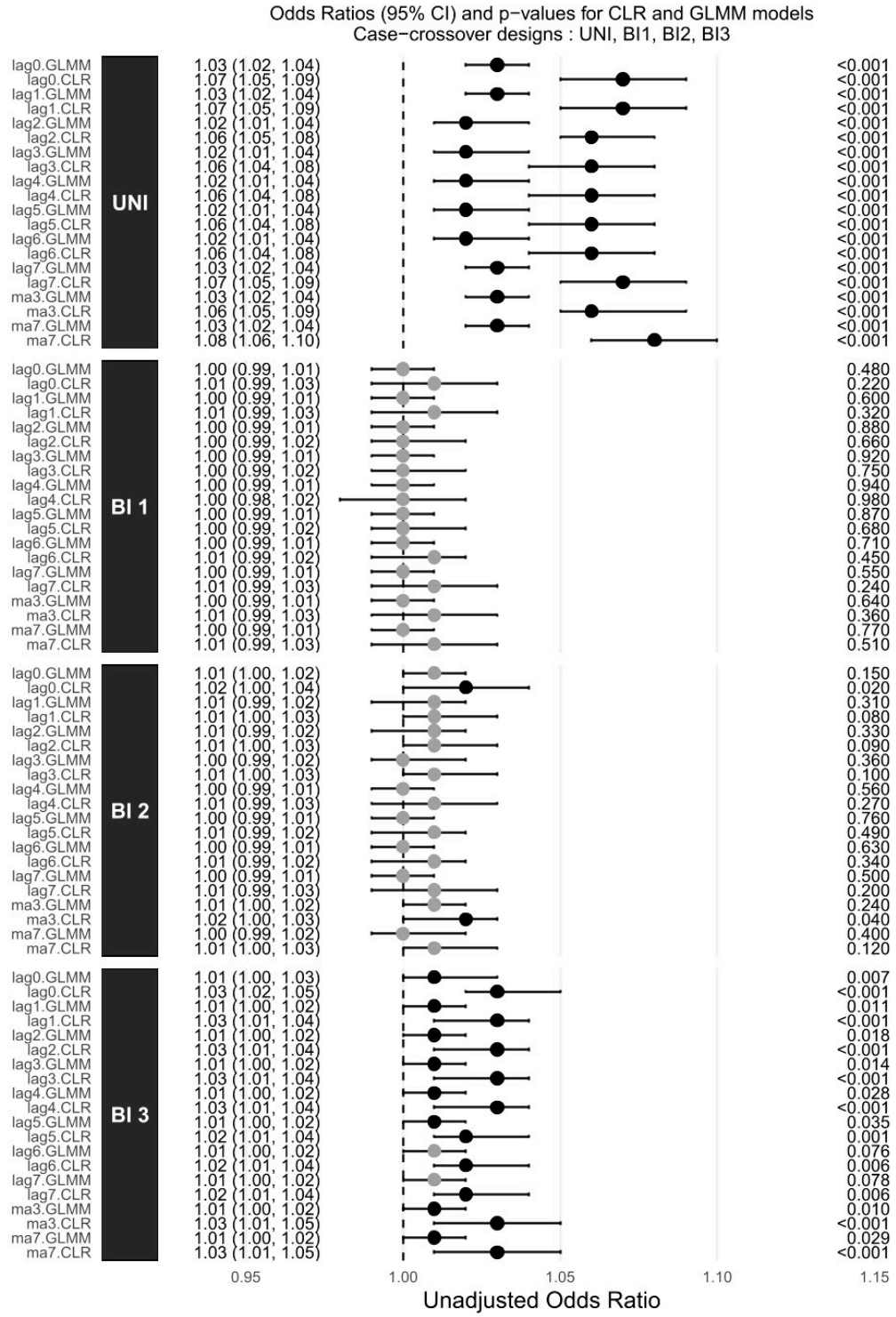


Figure 4

Adjusted and unadjusted odds ratios(95% confidence intervals) for a 10 μg/m³ increase in $PM_{2.5}$, estimated via conditional logistic regression (CLR, unadjusted) and generalized linear mixed models (GLMM, adjusted) within unidirectional and bidirectional symmetric case–crossover study designs, across single-day lags (lag0–lag7) and 3-day moving average lags (ma3–ma7).

Supplementary Files

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