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Short-term exposure to ambient $PM_{2.5}$ and PM_{10} and daily cardiopulmonary mortality in Sierra Leone: A time-series analysis using a generalized additive model.

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Abstract

Background: Ambient particulate matter (PM) remains a critical global public health challenge, especially for cardiovascular and respiratory diseases. The Sierra Leone Sample Registration System provides representative cause-specific mortality data, but little is known about the association between particulate matter exposure and cardiopulmonary mortality. To address this gap, we conducted a time-series analysis to assess the effect of short-term PM2.5 and PM10 exposure on cardiopulmonary mortality in Sierra Leone.

Method: We retrieved daily mortality data from 2016 to 2025 from the Health Enumeration and Assessment in Sierra Leone surveillance system and aggregated them into 3,279 daily observational counts. We used high-resolution satellite reanalysis datasets to estimate environmental exposures: MERRA-2 for $PM_{2.5}$ and PM_{10} and ERA5-Land for meteorological covariates. We estimated the relative increase in daily mortality for each 10 ug/m^3 rise in pollutant, with 95% confidence intervals. We used a quasi-Poisson distribution and Generalized Additive Models.

Results: Our findings show that, for every 10 μg/m^3 increase in $PM_{2.5}$ and PM_{10}, cardiopulmonary mortality risk was 3.24% (95% CI: 0.16, 6.43) and 1.12% (95% CI: 0.13, 2.11) higher, respectively. The strongest single-day effect was observed at lag 2 (i.e., a 48-hour delayed biological response). The associations remained robust after excluding the COVID-19 pandemic period. Stratified analyses show no significant effect modification by season, sex, or age category.

Conclusion: Short-term exposure to $PM_{2.5}$ and PM_{10} is associated with increased cardiopulmonary and cardiovascular mortality in Sierra Leone, underscoring the need for air quality monitoring, emission controls, and climate-sensitive public health measures.

Keywords: Air pollution; cardiovascular mortality; satellite remote sensing; acute exposure; time-series analysis; particulate matter.

Introduction

Ambient particulate matter (PM) remains a major global public health challenge, especially for cardiovascular and respiratory diseases [1]. PM with an aerodynamic diameter ≤ 2.5 μm ($PM_{2.5}$) and ≤ 10 μm (PM_{10}) are among the most harmful pollutants in the atmosphere, and the pathophysiological mechanisms have been documented: fine particles enter the pulmonary alveoli and translocate into the systemic circulation, causing a metaflammatory pathway that links pulmonary inflammation and autonomic dysfunction, significantly increasing the risk of acute cardiopulmonary events [1–3]. Evidence from global assessments shows that over 9 million premature deaths are attributed to air pollution in 2015, while exposure to ambient air pollution alone has contributed to more than 4 million premature deaths globally in 2019, with the burden concentrated among low- and middle-income countries [4, 5]. In Sub-Saharan Africa (SSA), air pollution alone is estimated to cause more than 1.1 million premature deaths each year [6, 7], making it a major driver of mortality in the region. Rapid urbanization, biomass burning as the main fuel for cooking and home heating, and a lack of air quality monitoring systems pose specific risks in Sub-Saharan Africa, yet the region remains among the most underrepresented in the epidemiological literature on short-term PM exposure and mortality [8].

Sierra Leone offers a compelling setting to explore air pollution's impact on cardiopulmonary mortality. The country has one of the highest mortality rates globally, with a life expectancy at birth of 54 years and over 60% of individuals dying before age 70 [9]. Currently, the nation is undergoing an epidemiological transition; a significant proportion of adult deaths is induced by non-communicable diseases (NCDs) [10, 11]. For instance, lower respiratory and cardiovascular diseases, including stroke and ischemic heart disease, have been reported as some of the leading causes of premature deaths in Sierra Leone [9]. Therefore, reliable cause-of-death data are crucial for evaluating the health burden of environmental exposures, yet Sierra Leone has historically lacked comprehensive mortality surveillance. The Sierra Leone Sample Registration System (SL-SRS) was the country's first nationally representative mortality survey to use electronic verbal autopsy (e-VAs) to ascertain cause of death [9]. However, the SL-SRS does not consider air pollution a cause of death. This is not a limitation of the survey design: several autopsy instruments assign

underlying causes of death according to the international classification of diseases (ICD), and environmental factors such as air pollution are not registered as underlying causes of death [4, 12]. Because air pollution is a risk factor rather than an underlying cause of death, it is not registered in the SL-SRS, and the verbal autopsy data alone cannot reveal the extent to which it contributed to these deaths.

To overcome the limitation of verbal autopsy data and accurately quantify the health burden of air pollution, this study needs precise daily environmental exposure data. Beyond gaps in mortality monitoring, Sierra Leone also lacks a comprehensive ground-based air-quality monitoring network across its 16 administrative districts. To bridge this dual data scarcity, this study integrates satellite remote sensing and atmospheric reanalysis models. These technologies can offer a high-resolution, powerful alternative to assessing environmental exposures at scale in data-limited regions [13, 14]. In particular, by combining NASA MERRA-2 aerosol reanalysis data with high-resolution observations from Copernicus Sentinel-5P, researchers can accurately estimate surface particulate matter concentrations and track the transport of atmospheric pollutants across large time and spatial scales [15, 16]. These multi-source satellite products can be combined with geospatial computational algorithms to monitor air-quality dynamics across space and time continuously. This enables the collection of essential data on pollution sources and transport from region to district, providing robust pollution exposure estimates that conventional, fragmented ground-based networks cannot provide.

To address our study's aim, we used an ecological time-series design, integrating SL-SRS cause-specific mortality data with satellite-derived ambient $PM_{2.5}$ and PM_{10} concentrations across Sierra Leone's 16 districts from 2016 to 2025. We specifically aimed to (1) quantify the exposure-response relationship for non-accidental mortality, (2) assess cause-specific cardiovascular and cardiopulmonary mortality, and (3) identify effect modification by sex, age, and season (Harmattan period and the wet monsoon period). This provides the first empirical evidence of the short-term mortality burden from PM2.5 and PM10 exposure in Sierra Leone and offers a transferable methodological framework for other West African locations without sufficient monitoring data.

Materials and methods

Study Design and Environment

This study linked population-wide mortality registries from the HEAL-SL surveillance system with high-resolution, satellite-driven environmental arrays to assess the acute and lagging impacts of atmospheric particulate matter on human survival. We utilized a retrospective ecological time-series analysis, a robust analytical approach for environmental epidemiological studies, to evaluate the acute effects of environmental exposures on mortality [17, 18]. Our study environment covered the 16 Districts in Sierra Leone. During the dry season, prevailing Harmattan trade winds transport fine mineral dust particles from the Saharan Desert, significantly affecting regional air quality [19]. Both anthropogenic and natural pollution sources impact air quality in Sierra Leone. Substandard, unpaved, and poorly maintained roads are a major source of resuspended road dust. In addition, other pollutants, such as carbon monoxide (CO), are higher in urban areas because of the growing fleet of older vehicles.

Data Sources

We obtained daily mortality data from January 2016 to June 2025 from the HEAL-SL project website (Level 2), which operates under the SRS in Sierra Leone and provides continuous vital statistics registration from 2016 to 2025 [20]. We chose the 9 years (2016-2025) to minimize Ebola's impact on data from 2014-2015 while providing sufficient daily observations for time-series analysis. Satellite-derived environmental data, the NASA MODERN-Era Retrospective Analysis for Research and Applications Version 2 (MERRA-2) aerosol reanalysis data, were used to obtain the daily concentrations of particulate matter ($PM_{2.5}$, PM_{10}), as the acquisition and use are described by Gelaro et al. (2017) [13]. We calculated $PM_{2.5}$ and PM_{10} by summing the mass concentrations of their components.

$PM_{2.5}$ = *BCSMASS* + *OCSMASS* + *SO4SMASS* + *DUSMASS25* + *SSSMASS25*……(1)

PM_{10} = *BCSMASS* + *OCSMASS* + *SO4SMASS* + *DUSMASS* + *SSSMASS*……....(2)

We converted the results from kg/m³ to µg/m³ by multiplying by 10^9. We computed daily means for $PM_{2.5}$ and PM_{10}.

We retrieved daily air temperature (2m temperature) and daily dew point temperature data from the ERA5-Land daily reanalysis dataset from the European Center for Medium-Range Weather Forecasts (ECMWF) [21]. We derived relative humidity from temperature and dew point using the magnus formula.

$$RH = 100\ x\ \frac{\exp((17.625Td)/(243.04+Td))}{\exp((17.625\ T)/(243.04+T))} \quad \text{..... (3)}$$

Where:

RH: Relative humidity in percent,

Td: The dew point temperature at 2 meters in °C, and

T: The air temperature at 2 meters (°C.

For each year, we derived the annual mean relative humidity, resulting in the humidity annual band.

We extracted all environmental data by district using zonal statistics in the Google Earth Engine tool, and downloaded the district boundaries from the Demographic and Health Survey (DHS) Spatial Data Repository [22]. The environmental parameters were then summarized to obtain national daily averages for the time-series analysis.

Statistical analysis

Data classification, aggregation, and descriptive statistics.

The de-identified 31,700 mortality records were categorized based on the ICD-10 codes: respiratory diseases (ICD-10 codes J00-J98), non-accidental causes (all-cause) (ICD-10 codes A00-R99), cardiopulmonary diseases- combined cardiovascular and respiratory causes (I00-I99, J00-J98), and cardiovascular diseases (ICD-10 codes I00-I99) [23].

Based on the study design, we aggregated individual-level mortality records into daily observational counts by date of death and specific cause-of-death categories. Similarly, we averaged district-level environmental data to obtain daily national means of PM2.5 and PM10 concentrations, humidity, and temperature. To analyze all cases, we joined the mortality and environmental datasets using an inner join, yielding a final analytical sample of 3,279 daily observations (see Fig. 1). We summarized daily distributions of mortality and environmental variables with descriptive statistics, including means, standard deviations, and percentiles. We also calculated Pearson correlation coefficients to assess linear associations and potential multicollinearity between air pollutants and meteorological parameters, ensuring the selected confounders were sufficiently independent for the regression models.

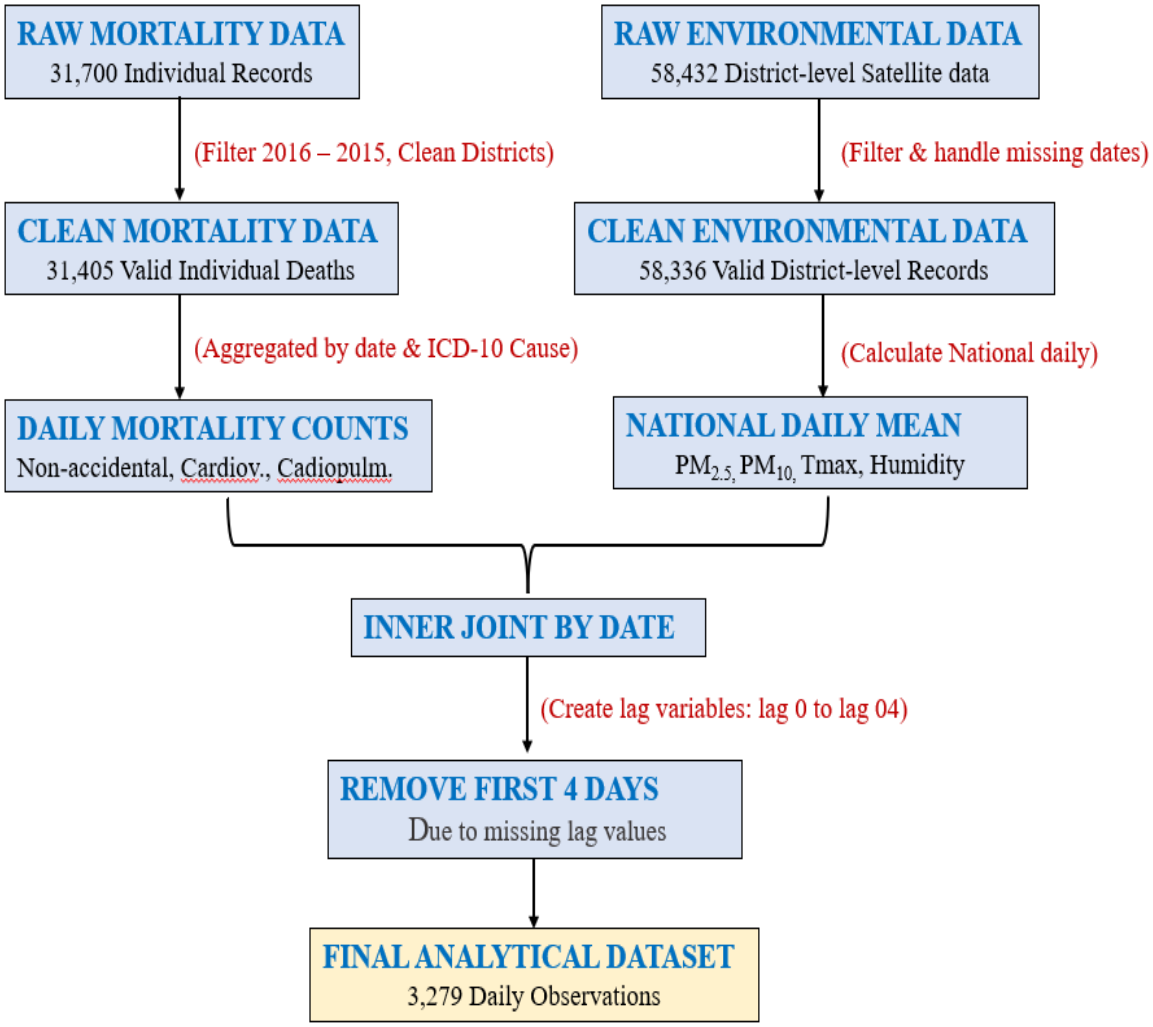


Fig. 1 Data screening and aggregation.

The diagram illustrates how we aggregated and merged individual-level mortality records with environmental data. The final analytical dataset consisted of 3,279 daily observation counts after accounting for the 4-day lag structure requirements

Lag structure and main time-series modeling

Because the physiological impacts of inhaling particulate matter may not manifest immediately, we constructed lag variables. We evaluated single-day lags (lag 0-lag 4, where lag 0 represents the current day and lag 4 represents four days prior) and multi-day moving averages (lag 01-lag 04). This approach captured both the acute, delayed effects and the cumulative burden of air pollution exposure over time.

To estimate the core association between daily mortality counts and daily ambient air pollutant concentrations, we applied generalized additive models (GAMs) with a quasi-Poisson distribution. Because overdispersion is common in daily mortality count data (variance exceeds the mean), we used a quasi-Poisson family to address it. We used penalized regression splines to control for potential confounding factors. We used a spline with 3 degrees of freedom (df) for same-day maximum temperature and relative humidity to flexibly adjust for their non-linear effects on mortality. We also applied a spline with 80 degrees of freedom to the continuous time index (days since the start of the study) to adjust for long-term trends and seasonal variation in mortality. We also added categorical variables for day of the week (DOW) to control for weekly behavioral and reporting trends. We present results as % change in mortality/day, with corresponding 95% confidence intervals (CIs), for each 10 $\mu g/m^3$ increase in $PM_{2.5}$.

Stratified analyses and effect modification:

We performed stratified analyses to identify potentially vulnerable subpopulations and assess whether environmental contexts modified air pollution's health impacts. We also dichotomized the data by season (Dry/Harmattan vs. Wet), Gender (Male/Female), and Age (0-35 years vs. 36+ years). This age division reflects distinct demographic and epidemiological patterns and provides sufficient statistical power for our time-series analysis. The 0-35 age group represents most of Sierra Leone's population and the main workforce, while the 36+ age group represents the aging population and the high burden of non-communicable diseases (NCDs) in the country,

which is on a rising trend. We defined the Dry/Harmattan season as November to April, when Saharan dust intrusion is elevated, and the Wet season as May to October. We tested effect modification using the Z-test for independent estimates.

$$Z = (Q1 - Q2)/\sqrt{SE1^2 + SE2^2} \quad (4)$$

Here, Q1 and Q2 are the effect estimates for the strata, and SE1 and SE2 are the strata standard errors. If the interaction term p-value is less than 0.05, the strata differ significantly in the association between pollution and mortality.

Sensitivity and Spatial analysis.

We conducted two sensitivity tests to assess the robustness of our results. First, to avoid potential undue influence from abnormal mortality patterns and potential behavioral shifts during the global pandemic, we re-ran the key models excluding the COVID-19 period (1st March 2020 to 31st December 2022). Second, we tested the stability of our effect estimates across lag structures by comparing the cumulative lag 04 model with the acute lag 0 model and the lag 2 model for all-cause mortality.

We also conducted a spatial analysis using the packages `sf` and `ggplot2` in R to visualize the spatial distribution of mortality burden across the country. We conducted all statistical analyses in RStudio version 4.5.3 and considered p-values < 0.05 (two-sided) statistically significant.

Ethical Consideration

In this study, we utilized secondary, publicly de-identified mortality data from the HEAL-SL project website, which operates under the SL-SRS. Because the data are secondary and anonymized, we deemed ethical approval unnecessary.

Results

Study population and baseline statistics

On average, the results show 8.8 non-accidental deaths per day, 1.1 each from cardiovascular disease and 1.7 each from cardiopulmonary diseases, as shown in **Table 1**. The mean daily concentrations of $PM_{2.5}$ and PM_{10} were 41.1 μg/m³ (SD: 30.6) and 120.7 μg/m³ (SD: 85.7), respectively. The mean daily maximum temperature was 30.5°C (SD: 2.8), while the mean relative humidity was 93.7% (SD: 4.0).

Table 1. Daily mortality and environmental parameters, Sierra Leone, 2016-2025

Section	Variable	Mean ± SD	Min	P25	P50	P75	Max
Daily death counts							
	Non-accidental	8.8 ± 14.5		3	6	11	416
	Cardiovascular	1.1 ± 2.2	0	0	1	1	62
	Cardiopulmonary	1.7 ± 3.1	0	0	1	2	94
Air pollutants (μg/m³)							
	$PM_{2.5}$	41.1 ± 30.6	2.9	16.2	35.3	56.3	207.3
	PM_{10}	120.7 ± 85.7	8.1	54.8	103.1	158.9	574.4
Weather							
	Temperature (°C)	30.5 ± 2.8	23.5	28.5	30.4	32.8	38.1
	Humidity (%)	93.7 ± 4.0	74.7	90.9	95.2	96.8	99.5

[a] $PM_{2.5}$ and PM_{10} concentrations exceeded WHO guideline limits on numerous days.
[b]PM25, P50 and PM75 denote the 25th, 50th (Median), and 75th percentiles, respectively.
[c]SD = standard deviation; Min = minimum; max = maximum.

Correlations between environmental variables

$PM_{2.5}$ and PM_{10} were highly correlated (r = 0.97, $P < 0.01$), suggesting that both pollutants have similar sources and/or formation mechanisms (Table 2**).** Both particulate matters were also positively correlated with temperature ($PM_{2.5}$: r = 0.70; PM_{10}: r = 0.63; both $P < 0.01$) and negatively correlated with humidity ($PM_{2.5}$: r = -0.70; PM_{10}: r = -0.63; both $P < 0.01$). Temperature and humidity showed a strong inverse relationship (r = -0.89, $P < 0.01$), reflecting the inverse relationship between these meteorological variables in Sierra Leone's tropical climate.

Table 2. Correlation matrix for $PM_{2.5}$, PM_{10}, and weather, Sierra Leone (2016-2025)

Variable	PM25	PM10	Temperature	Humidity
$PM_{2.5}$	1	0.97*	0.7*	-0.7*
PM_{10}	0.97*	1	0.63*	-0.63*
Temperature	0.7*	0.63*	1	-0.89*
Humidity	-0.7*	-0.63*	-0.89*	1

[a.] $PM_{2.5}$ and PM_{10} have a strong correlation, suggesting common emission sources.

[b.] Temperature and Relative humidity have an inverse correlation, reflecting reduced particulate concentrations during wet-season precipitation.

[c] * = strong correlations

Association of $PM_{2.5}$ and PM_{10} and cause-specific mortality

In single-pollutant generalized additive models, we observed significant positive associations between acute particulate-matter exposure and cause-specific mortality (Table 3) after adjusting for meteorological variables and temporal trends. A 5-day moving average (lag 04) showed that a 10 μg/m3 increase in $PM_{2.5}$ was associated with a 3.24% (95% CI: 0.16%, 6.43%) increase in cardiopulmonary mortality. Similarly, a 10 $\mu g/m^3$ increase in PM_{10} was associated with a 1.12% (95% CI: 0.13%, 2.11%) increase in cardiopulmonary mortality.

For cardiovascular mortality specifically, the effects were more pronounced: a 10 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 3.60% (95% CI: 0.16%, 7.16%) increase, while PM_{10} was associated with a 1.35% (95% CI: 0.25%, 2.45%) increase. For non-accidental (all-cause) mortality, PM_{10} showed a statistically significant association (0.98%, 95% CI: 0.08%, 1.90%), while $PM_{2.5}$ showed a positive but borderline significant association (2.74%, 95% CI: -0.11%, 5.68; $P = 0.0601$).

Table 3. Daily mortality increase (%) per 10 μg/m³ $PM_{2.5}$ and PM_{10} (Lag 0-4).

Outcome	Pollutant	Estimate (95% CI)	P Value
Non-Accidental			
	PM_{25}	2.74 (-0.11, 5.68)	0.0601
	PM_{10}	0.98 (0.08, 1.90)	**0.0325**
Cardiovascular			
	PM_{25}	3.60 (0.16, 7.16)	**0.0401**
	PM_{10}	1.35 (0.25, 2.45)	**0.0155**
Cardiopulmonary			
	PM_{25}	3.24 (0.16, 6.43)	**0.0393**
	PM_{10}	1.12 (0.13, 2.11)	**0.0261**

[a]The table shows % change in daily mortality per 10 μg/m^3 increase in pollutant concentrations using a 5-day moving average (lag 04).

[b]Bold p-values signify statistically significant differences ($P < 0.05$).

[c]$P < 0.05$ indicates statistical significance

To display the shape of the exposure-response curve for $PM_{2.5}$ and PM_{10} and the assessed potential non-linear thresholds, we used a generalized additive model. The concentration-response curves were largely linear and positive, with no evidence of a safe threshold or an exponential increase at very high exposures (Fig. 2). This supports the validity of the earlier linear effect estimates.

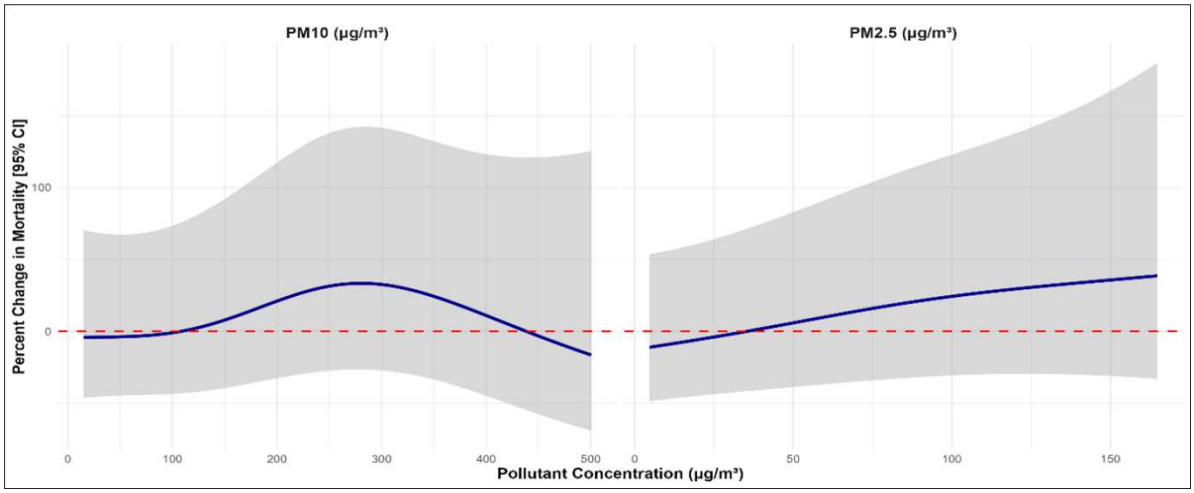


Fig. 2 Concentration-response curves for $PM_{2.5}$ and PM_{10} and cardiopulmonary mortality in Sierra Leone (2016-2025). The solid blue line represents the estimated percent change in cardiopulmonary mortality, and the shaded area represents the 95% confidence interval. The red dashed line indicates the reference point (median pollutant concentration). The curves demonstrate a largely linear exposure-response relationship without a clear threshold effect

Lag structure analysis for non-accidental mortality

The temporal pattern of air pollution effects varied across different lag structures for non-accidental mortality (**Fig. 3**). $PM_{2.5}$ showed its strongest single-day effect at lag 2 (4.10%, 95% CI: 1.81%, 6.44%; $P < 0.001$), while the cumulative 5-day moving average (lag 04) showed a 2.74% increase (95% CI: -0.11%, 5.68). Similarly, PM_{10} peaked at lag 2 (1.41%, 95% CI: 0.70%, 2.12%; $P < 0.001$), while the lag 04 moving average showed 0.98% (95% CI: 0.08%, 1.90%). This pattern suggests a delayed biological response, with health effects peaking about 48 hours after exposure and potentially lasting several days.

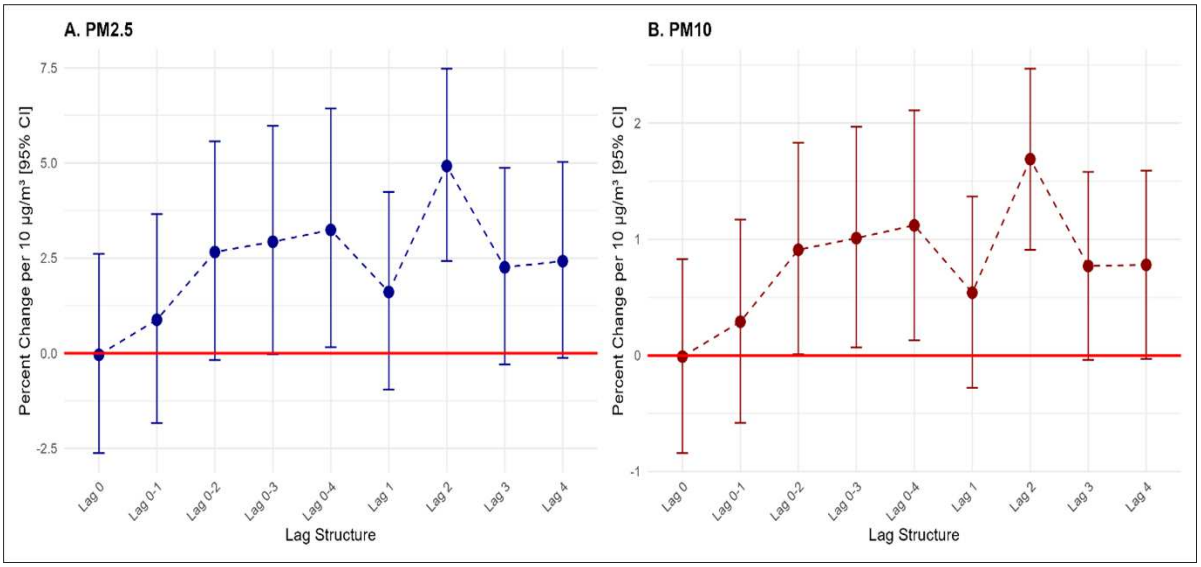


Fig. 3 Lag structure of $PM_{2.5}$ and PM_{10} on non-accidental mortality in Sierra Leone (2016-2025). Single-day lags (Lag 0-4) represent exposure on the day of death and up to 4 days prior. Multi-day lags (Lag 01-04) represent moving averages. The peak effect at Lag 2 indicates a 48-hour delayed response, with a cumulative effect at Lag 04

Seasonal variation analysis

The effect estimates for both pollutants were higher for cardiopulmonary mortality in the Dry (Harmattan) season than in the Wet season (Table 4, Fig. 4). Specifically, a 10 µg/m³ increase in $PM_{2.5}$ was associated with a 3.64% (95% CI: 0.00%, 7.42%; *P* = 0.050) increase in cardiopulmonary mortality during the Dry season, compared to a 1.11% (95% CI: -5.70%, 8.42%; *P* = 0.756) increase during the Wet season. Similarly, PM_{10} increased by 1.19% (95% CI: 0.02%, 2.37%; P = 0.047) in the Dry season, while the Wet season estimate was 0.47% (95% CI: -1.45%, 2.42%; *P* = 0.636) and not significant.

The associations were also greater for non-accidental (all-cause) mortality during the wet season. $PM_{2.5}$ was associated with a 6.83% (95% CI: 0.35%, 13.73%; *P* = 0.039) increase in non-accidental mortality during the Wet season, compared with 3.08% (95% CI: -0.48%, 6.75%; *P* = 0.091) in the Dry season. PM_{10} showed a similar pattern, with a 1.67% (95% CI: 0.07%, 3.31%; *P* = 0.041) increase with significance

during the Wet season and a 1.02% (95% CI: -0.13%, 2.17%; *P* = 0.082) increase during the Dry season.

The formal interaction test showed no statistically significant seasonal interaction for either cardiopulmonary mortality ($PM_{2.5}$: *P* = 0.537; PM_{10}: P = 0.533) or non-accidental mortality ($PM_{2.5}$: *P* = 0.328; PM_{10}: P = 0.516). Although the point estimates for cardiopulmonary mortality were higher in the Dry season, the interaction tests were not statistically significant, likely reflecting limited statistical power rather than a true absence of seasonal effect modification. However, these seasonal results should therefore be interpreted as exploratory.

Table 4. Seasonal variation in cardiopulmonary mortality associated with 10 μg/m³ increases in $PM_{2.5}$ and PM_{10}.

Outcome	Pollutants	Season	N days	Estimate (95% CI)	P value
Cardiopulmonary					
	PM_{25}	Dry (Harmattan)	1663	3.64 (0.00, 7.42)	0.050
	PM_{25}	Wet	1616	1.11 (-5.70, 8.42)	0.756
	PM_{10}	Dry (Harmattan)	1663	1.19 (0.02, 2.37)	**0.047**
	PM_{10}	Wet	1616	0.47 (-1.45, 2.42)	0.635
Total (non-accidental)					
	PM_{25}	Dry (Harmattan)	1663	3.08 (-0.48, 6.75)	0.090
	PM_{25}	Wet	1616	6.83 (0.35, 13.73)	**0.038**
	PM_{10}	Dry (Harmattan)	1663	1.02 (-0.13, 2.17)	0.081
	PM_{10}	Wet	1616	1.67 (0.07, 3.31)	**0.041**

[a]Effect estimates are in units per 10 μg/m³ increase in pollutant.

[b]P-values for interaction tests by season were: Cardiopulmonary $PM_{2.5}$ (0.5365); Cardiopulmonary PM_{10} (0.5326); non-accidental $PM_{2.5}$ (0.3284); non-accidental PM_{10} (0.5161).

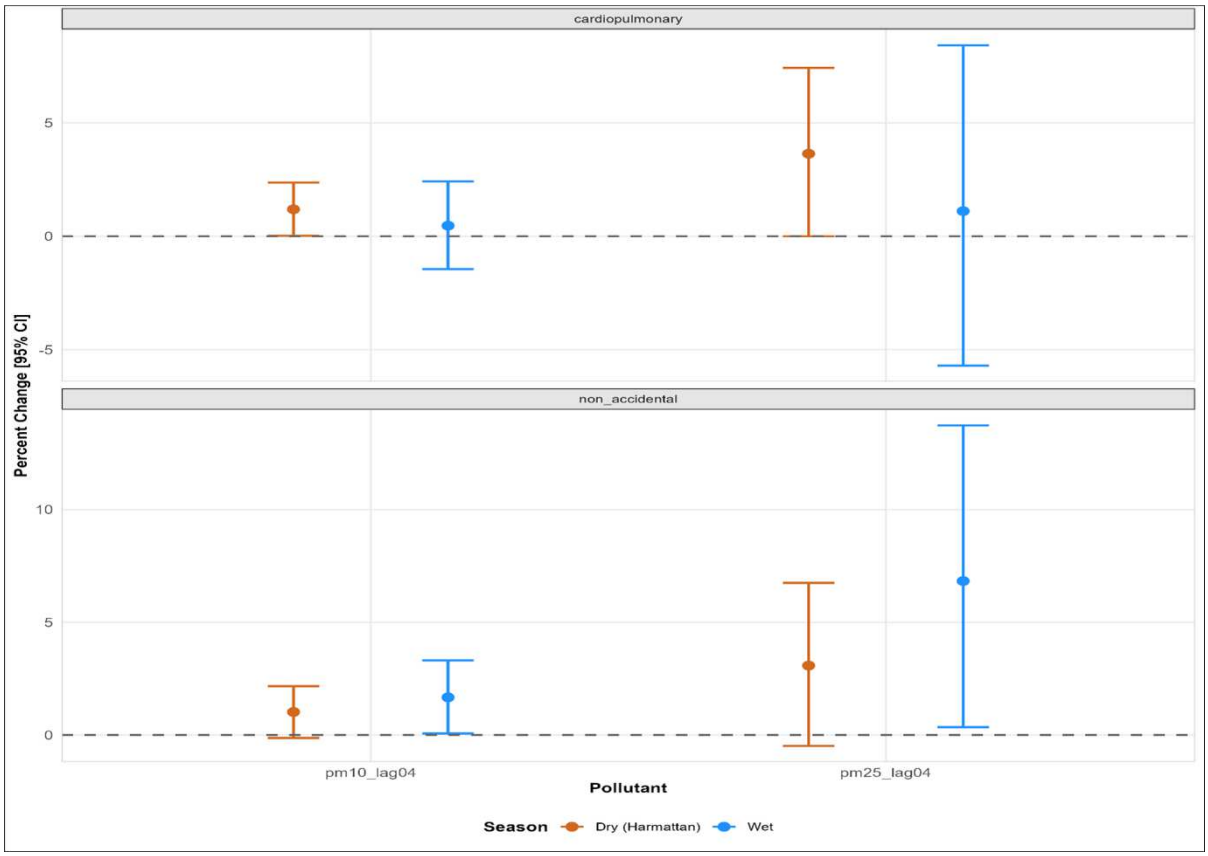


Fig. 4 Season-specific effects of $PM_{2.5}$ and PM_{10} on mortality in Sierra Leone (2016-2025). Forest plot showing the percent change in cardiopulmonary and non-accidental mortality per 10 μg/m³ increase in $PM_{2.5}$ and PM_{10}, stratified by Dry (Harmattan, orange) and Wet (blue) seasons. Error bars represent 95% confidence intervals. The dashed line represents the null effect (0% change). Although point estimates differ between seasons, overlapping confidence intervals and non-significant interaction tests suggest no significant seasonal modification

Stratified analysis by sex (male and female) and age group

Among the subgroup analyses (Table 5), a higher percent increase in cardiopulmonary mortality associated with $PM_{2.5}$ was observed in males (2.81%, 95% CI: -0.35%, 6.08) than in females (1.06%, 95% CI: -2.01%, 4.23), but neither was statistically significant. Similarly, for PM_{10}, males showed a 0.92% increase (95% CI: -0.08%, 1.93) while females showed 0.45% (95% CI: -0.52%, 1.44); again, there was no significant difference between sexes (*P* for interaction > 0.05) observed in the interaction tests.

For age-stratified analysis, we compared the 0-35 age group with the 36+ age group. The younger population (0-35 years) showed a 3.15% increase in cardiopulmonary mortality per 10 μg/m³ $PM_{2.5}$ (95% CI: -0.33%, 6.76), while the older population (36+ years) showed a 1.31% increase (95% CI: -1.74%, 4.45). For PM_{10}, the 0-35 years group showed a 1.00% increase (95% CI: -0.09%, 2.09), and the 36+ years group showed a 0.54% increase (95% CI: -0.43%, 1.51). Again, interaction tests showed no statistically significant age-based effect modification (*P* for interaction > 0.05).

Table 5. Cardiopulmonary mortality by sex and age per 10 μg/m³ of $PM_{2.5}$ and PM_{10}

Sex Label	Mean Daily Deaths	Pollutant	Estimate (95% CI)	P Value
Male	0.78	PM_{25}	2.81 (-0.35, 6.08)	0.0823
Female	0.73	PM_{25}	1.06 (-2.01, 4.23)	0.5013
Male	0.78	PM_{10}	0.92 (-0.08, 1.93)	0.0731
Female	0.73	PM_{10}	0.45 (-0.52, 1.44)	0.3627
0-35 years	0.32	PM_{25}	3.15 (-0.33, 6.76)	0.0762
36+ years	1.19	PM_{25}	1.31 (-1.74, 4.45)	0.4042
0-35 years	0.32	PM_{10}	1.00 (-0.09, 2.09)	0.0719
36+ years	1.19	PM_{10}	0.54 (-0.43, 1.51)	0.2771

[a]Males and younger populations (0-35 years) showed numerically higher effect estimates

[b]None of the stratum-specific associations reached statistical significance, and interaction tests revealed no significant effect modification.

Sensitivity analyses

We performed two sensitivity analyses to assess the robustness of our results: excluding the COVID-19 period and Using Different Lag Structures for all-cause mortality.

Exclusion of COVID-19 period

Excluding the COVID-19 period (March 1st 2020, to December 31st 2022), the effect estimates remain robust (see Table 6) and, in some cases, are even higher. For non-accidental mortality, the $PM_{2.5}$ association increased from 2.74% (95% CI: -0.11%, 5.68) in the main analysis (Table 3) to 3.78% (95% CI: 0.80%, 6.86) when excluding COVID-19 months. Similarly, for cardiopulmonary mortality, $PM_{2.5}$ increased from 3.24% (95% CI: 0.16%, 6.43) to 3.71% (95% CI: 0.50%, 7.03). These findings confirm that the observed association between air pollution and mortality is not just an artifact of pandemic-related mortality anomalies but represents a true environmental health risk.

Table 6. Comparison of main analysis with COVID-19 period exclusion (March 2020 - December 2022)

Outcome	Pollutant	Estimates (95% CI)	COVID Estimates
Non-Accidental	PM_{25}	2.74 (-0.11, 5.68)	3.78 (0.80, 6.86)
Non-Accidental	PM_{10}	0.98 (0.08, 1.90)	1.22 (0.28, 2.17)
Cardiopulmonary	PM_{25}	3.24 (0.16, 6.43)	3.71 (0.50, 7.03)
Cardiopulmonary	PM_{10}	1.12 (0.13, 2.11)	1.30 (0.28, 2.33)

[a]Estimates (95% CI) = main analysis with study period (January 2016 - June 2025)

[b]COVID Estimates = exclusion of the COVID-19 period (1 March 2020 - 31 December 2022).

[C]Adjusted for day of the week and long-term time trends (80 degrees of freedom), as well as same-day maximum temperature and relative humidity (3 degrees of freedom each); 95% CI = confidence interval.

Different Lag Structures for all-cause mortality

We checked the stability of our effect estimates by varying lag structures in the non-accidental mortality analysis (Table 7**).** The analysis confirmed that the strongest

single-day effect occurred at lag 2 (48-hour delay) for both pollutants: $PM_{2.5}$ showed a 4.10% increase (95% CI: 1.81%, 6.44; $P < 0.001$) and PM_{10} showed a 1.41% increase (95% CI: 0.70%, 2.12; $P < 0.001$). However, the cumulative 5-day moving average (lag 04) for $PM_{2.5}$ was not statistically significant (2.74%, 95% CI: -0.11%, 5.68), but it was significant for PM_{10} (0.98%, 95% CI: 0.08%, 1.90; $P = 0.0325$). The same-day exposure (lag 0) showed no significant associations for either pollutant, reinforcing the delayed nature of air pollution health effects. This sensitivity analysis shows that our selection of lag 04 for the main analysis in "Table 3" was suitable for capturing cumulative effects, and it also shows acute effects at lag 2.

Table 7. Percent increase in non-accidental mortality across different lag structures

Pollutant	Estimate (95% CI)	P Value
PM25 LAG0	-0.52 (-2.89, 1.90)	0.6704
PM25 LAG2	4.10 (1.81, 6.44)	**-0.001***
PM25 LAG04	2.74 (-0.11, 5.68)	0.0601
PM10 LAG0	-0.18 (-0.94, 0.59)	0.6441
PM10 LAG2	1.41 (0.70, 2.12)	**-0.001***
PM10 LAG04	0.98 (0.08, 1.90)	**0.0325**

[a]Single-day lags (Lag 0, Lag 2) represent exposure on the day of death and 2 days prior, respectively.

[b]Lag 04 represents the 5-day moving average.

[c]The peak effect at Lag 2 and significant cumulative effects at Lag 04 demonstrate the delayed and prolonged nature of air pollution health impacts.

[d]Bold p-values indicate statistical significance ($P < 0.05$); * = P < -0.001

Spatial distribution of cardiopulmonary mortality

To visualize the geographic burden of pollution-sensitive mortality, we generated a choropleth map showing the spatial distribution of cardiopulmonary (combined cardiovascular and respiratory causes) deaths across Sierra Leone's 16 districts (Fig. 5). The map showed that the absolute mortality burden differed substantially across districts. The darkest shading (Bo) indicates the district with the most cardiopulmonary deaths across the study period. This result is consistent with Bo being the second-largest urban center and a major regional population hub in Sierra Leone, where

higher population density translates to a higher absolute number of daily deaths. Darker orange and brown indicate other districts with a high mortality burden, including Koinadugu and Bonthe. In contrast, the lightest yellow districts show the lowest absolute counts: Falaba, Karene, and Port Loko. This spatial visualization highlights the need for targeted public health interventions, enhanced clinical capacity for cardiovascular and respiratory care, and localized air quality monitoring in high-burden districts like Bo.

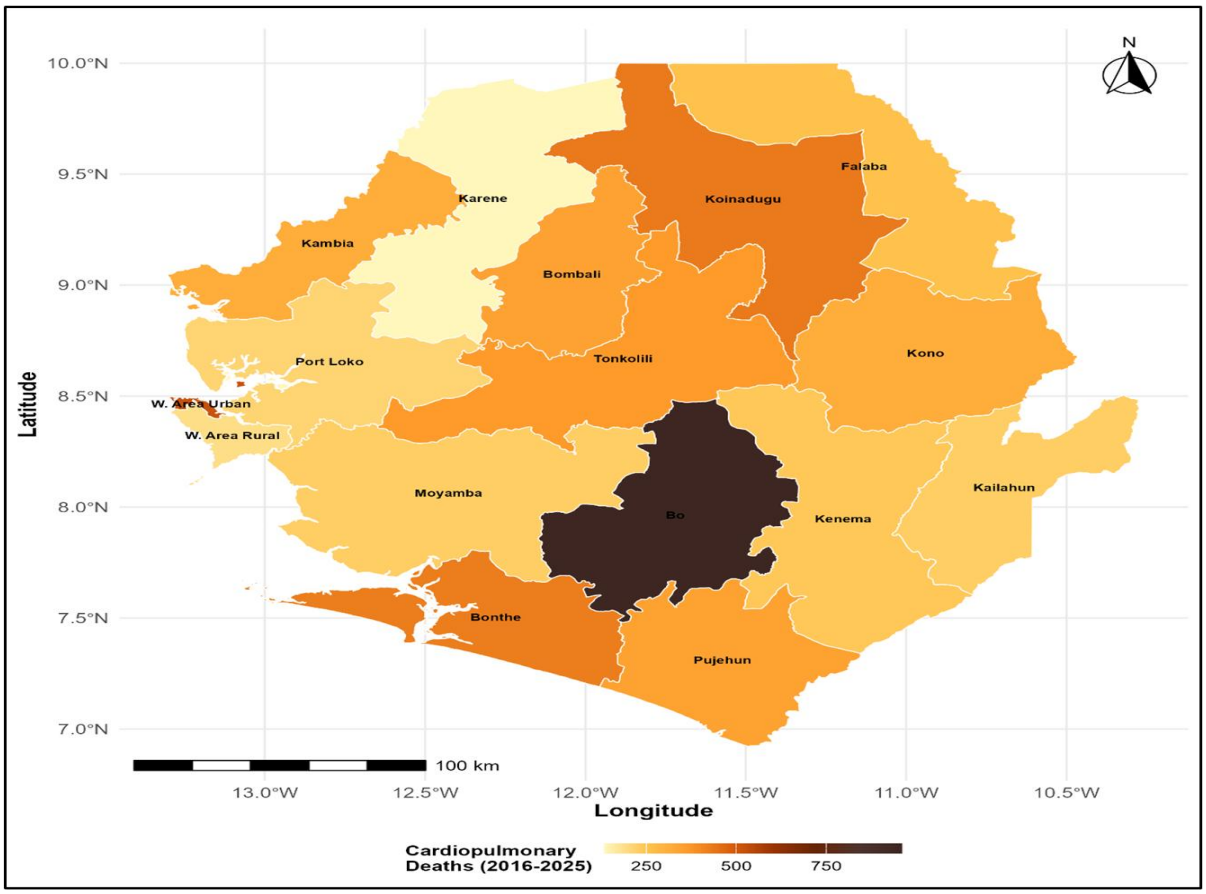


Fig. 5 Geographic distribution of cardiopulmonary mortality across Sierra Leone's 16 districts (2016-2025). The color gradient ranges from light yellow (lowest absolute mortality burden) to dark chocolate brown (highest absolute mortality burden). Bo district exhibited the highest total cardiopulmonary death count, reflecting its status as a major population center

Discussion

In this study, we analyzed a 9-year time series dataset linking acute exposure to ambient particulate matter and daily cardiopulmonary and cardiovascular mortality in Sierra Leone. Analysis of 3,279 daily observations from the HEAL-SL surveillance system shows that a 10 μg/m3 increase in $PM_{2.5}$ and PM_{10} was significantly associated with increased cardiopulmonary mortality, with peak acute effects at a 48-hour delay (lag 2) and significant cumulative effects over a 5-day window (lag 04). Moreover, our findings further reveal that the Sierra Leone population lives in a complex, multi-stressor environment where ambient $PM_{2.5}$ and PM_{10} levels exceed WHO guideline levels, with mean daily concentrations of 41.1 and 120.7 $\mu g/m^3$, respectively.

The higher concentrations of PM in our results can be attributed to local anthropogenic sources, including emissions from old vehicles, unpaved road dust, biomass burning, transboundary natural sources, and the seasonal transport of Saharan dust during the Harmattan season, as reported in previous studies [24–26]. Similarly, our findings align well with similar literature and are biologically plausible. Breathing fine and coarse particulate matter causes localized pulmonary oxidative stress and inflammation that can spread into the systemic circulation [2, 3]. This systemic inflammatory response is associated with endothelial dysfunction, autonomic imbalance, and activation of hemostatic pathways, ultimately promoting thrombosis, arrhythmias, and acute cardiovascular events [1, 27, 28]. In contrast, our national estimates are higher than previous Freetown bus-station measurements ($PM_{2.5}$ = 27.6-30.3 $\mu g/m^3$, PM_{10} = 31.4-37.2 $\mu g/m^3$), likely because it was a short time period and the Harmattan season was excluded [29]. However, our 9-year daily estimates included the Harmattan season, which drives PM_{10} much higher across the country.

Furthermore, the effect estimates for $PM_{2.5}$ and PM_{10} on Cardiopulmonary mortality (3.24% and 1.12% per 10 $\mu g/m^3$, respectively) are consistent with, and in some cases higher than, those seen in high-income countries and other low- and middle-income countries (LMICs) (Table 3). For instance, Liu et al. (2019) reported a 0.68% increase in cardiovascular mortality for every 10 μg/m3 increase in $PM_{2.5}$, and

Chen et al. (2017) reported a 0.27% increase in China [30, 31]. Likewise, region-specific studies in Africa have reported significant health impacts from particulate matter exposure. Ofoezie et al. (2022) found that urban air pollution is a major contributor to cardiovascular morbidity and premature mortality in South Africa, and Moro et al. (2024) found that ambient $PM_{2.5}$ is significantly associated with under-5 mortality in Ghana [32, 33]. The elevated effect estimates in our study may reflect very high PM concentrations in Sierra Leone and an aerosol mix of transboundary mineral dust and local biomass smoke. Moreover, there is a high prevalence of untreated cardiovascular risk factors, and access to advanced health services is limited, making the population highly vulnerable to adverse health outcomes [34, 35].

Our observed temporal dynamics of the air pollution-mortality relationship offer further biological validation. The strongest single-day effect at lag 2 (48 hours) is highly consistent with pathophysiological processes underlying acute air pollution-induced cardiovascular events [36]. The magnitude of the cumulative effect observed at lag 04 also suggests that the health impact of air pollution exposure in Sierra Leone is not merely an acute, single-day phenomenon but a long-term physiological stressor resulting from multi-day exposure windows that should be considered when designing public health interventions.

In our stratified results, although not statistically significant, we observed higher effect estimates among males and younger age groups (0-35 years) than among females and older age groups (36+ years) for $PM_{2.5}$ and PM_{10} on cardiopulmonary mortality**.** This insignificance may reflect diminished statistical power in analyses of already small daily cause-specific death series, as typically occurs in time-series studies [37]. Notably, the trend toward higher vulnerability in males may, however, be a biologically and socially plausible trend, as males in Sierra Leone are more likely to be associated with outdoor activities that involve physically strenuous work, like mining and farming, and thus have higher inhalation rates and higher cumulative exposure to ambient particulate matter. [38]. Larger studies or individual-level exposure data should confirm sex- and age-specific vulnerabilities.

The magnitude of air pollution effects varied by season, although the formal interaction tests were not statistically significant. The high effect estimates for $PM_{2.5}$

and PM_{10} during the dry season (Harmattan) and wet season may reflect high volumes of transboundary Saharan dust combined with local anthropogenic emissions, forming a particulate mixture that may adversely affect the cardiopulmonary system [24, 26]. However, for non-accidental mortality, the associations were stronger in the Wet season for $PM_{2.5}$ (6.83% vs. 3.08% in the Dry season) and PM_{10} (1.67% vs. 1.02%). This may reflect behavioral and environmental changes during the rainy season, such as spending more time indoors with inadequate ventilation and increased biomass burning for cooking, which may increase exposure to indoor and outdoor air pollution [25, 32]. Although point estimates vary by season, the lack of statistical significance in the formal interaction tests between seasons may reflect reduced statistical power when daily death counts are stratified by season for cause-specific mortality, rather than a true absence of seasonality. Nonetheless, all point estimates were positive in both the dry (Harmattan) and Wet seasons, suggesting that Sierra Leone's population may be vulnerable to particulate matter exposure year-round, whether from local anthropogenic sources or Saharan dust.

The robustness of our results is supported by the use of generalized additive models (GAMs) with penalized regression splines to account for non-linear confounding by temperature and humidity, as well as gradual temporal changes. The concentration-response analysis showed a highly linear, positive association between increased PM concentrations and cardiopulmonary mortality, with no evidence of safe thresholds. This is consistent with global evidence showing that health risks occur even at low PM concentrations [12, 39, 40]. The absence of a threshold in our data further supports the updated WHO air quality guidelines and emphasizes public health benefits of reducing ambient particulate matter, regardless of baseline concentration [41, 42].

This study has several strengths. First, the short-term exposure window is comparatively long at 9 years, providing sufficient statistical power to measure acute associations between $PM_{2.5}$ and PM_{10} variation and non-accidental, cardiopulmonary, and cardiovascular mortality. Second, we obtained mortality data from the HEAL-SL, which uses physician-confirmed causes of death, increasing the validity and reliability

of outcome classification. Third, to address the critical lack of ground-level air quality monitoring stations in all 16 districts of Sierra Leone, we used satellite-based, high-resolution reanalysis data from MERRA-2 and ERA5-Land to provide spatially and temporally resolved exposure estimates that would otherwise be unavailable. Fourth, a generalized additive model (GAM) let us flexibly control for nonlinear temporal trends and seasonality, potential confounders such as temperature and relative humidity, and lagged effects of PM exposure on mortality. Together, these methodological strengths support the internal validity of our results and provide strong, locally relevant evidence to inform air quality and public health policy, for which it has historically been difficult to gather.

However, a few limitations are worth noting. Using population-level ambient PM2.5 and PM10 concentrations as proxies for individual exposure may introduce non-differential exposure misclassification. This type of measurement error commonly deflates effect estimates in time-series studies, but does not inflate them [43]. Hence, our observed significant increases in cardiopulmonary and cardiovascular mortality risk per 10 $\mu g/m^3$ increment in $PM_{2.5}$ and PM_{10} exposure may represent conservative estimates of the true association. In addition, no data are available on individual exposure to other potentially important confounders such as lifestyle (socio-economic status and smoking) factors. Traditional chronic risk factors, however, are not expected to vary much day to day among individuals in a population and therefore are not believed to cause substantial confounding in time-series studies [44]. This limitation is common in time-series designs using aggregate mortality data and does not detract from the validity of the short-term associations observed.

Conclusion

To our knowledge, this is the first study to quantify the short-term association between ambient particulate matter and cause-specific cardiopulmonary mortality in Sierra Leone. Our findings show that short-term exposure to ambient $PM_{2.5}$ and PM_{10} is significantly associated with increased cardiopulmonary and cardiovascular mortality risk in Sierra Leone, with the greatest effect seen after a 48-hour (2-day) delay. The robustness of these associations after excluding the COVID-19 pandemic period strengthens the validity of our findings. Our findings underscore a public health

crisis that calls for air quality interventions in Sierra Leone and in similar data-constrained West African countries. We recommend that the government of Sierra Leone and regional bodies prioritize: (1) integrating air quality monitoring into Sierra Leone existing health surveillance infrastructure; (2) establishing a national ground-based air quality monitoring network across the 16 districts; (3) developing public health early-warning systems for high-pollution days; and (4) accelerating the transition to clean energy and improved cookstoves to reduce both ambient and household air pollution. Regional cooperation is also necessary to minimize the transboundary effects of Saharan dust. Future research should integrate individual-level exposure assessments and explore the specific toxicological components of Harmattan dust to refine public health interventions.

Declarations

All authors have read, understood, and complied, as applicable, with the statement on 'ethical responsibilities of authors' as found in the instructions for authors.

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Competing Interest

The authors declare no known competing financial interests or personal relationships that could have influenced the work reported in this paper.

Ethics approval

We utilized secondary, publicly de-identified mortality data from the HEAL-SL project website, which operates under the SL-SRS. Because the data are secondary and anonymized, we deemed ethical approval unnecessary. We also used open-access, publicly available satellite remote sensing and reanalysis data.

Consent to participate

Not applicable. The study used de-identified, population-level mortality data and did not involve direct contact with human subjects.

Data Availability

The datasets generated and/or analyzed during the current study are publicly available from the following sources:

- HEAL-SL. (2026). HEAL-SL mortality and cause-of-death surveillance data, Sierra Leone [Data set]. Healthy Sierra Leone, Ministry of Health and Sanitation (Sierra Leone), Njala University. https://healsl.org/ access under level 2.
- ERA5-Land reanalysis data (temperature and dewpoint temperature): Available from the Copernicus Climate Change Service (C3S) Climate Data Store at https://doi.org/10.24381/cds.e2161bac, and accessed via Google Earth Engine (ECMWF/ERA5_LAND/DAILY_AGGR).
- MERRA-2 aerosol reanalysis data ($PM_{2.5}$ and PM_{10}): Available from the NASA Global Modeling and Assimilation Office (GMAO) and accessed via Google Earth Engine.
- Administrative boundary shapefile (Sierra Leone, 16 districts): Available from the Demographic and Health Survey (DHS) Program Spatial Data Repository at https://spatialdata.dhsprogram.com/ under its terms of use (ICF, 2025).

The custom Google Earth Engine scripts for the environmental data extraction, the final dataset, and R code used for statistical analysis and figure generation are available from the corresponding author upon reasonable request.

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AI Declaration

During the preparation of this work, the authors used Grammarly solely for language editing purposes, including grammar correction, spelling checks, and improvements to sentence clarity. The authors used no other artificial intelligence (AI) tools or services for data analysis, interpretation, or scientific content generation. The authors reviewed and edited all content and took full responsibility for the final manuscript.

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