Journal of Air Pollution and Health (Summer 2026); 11(3): 307-324

Original Article

Available online at https://japh.tums.ac.ir

Detectability of short-term associations between ambient air pollution and cardiovascular morbidity: An ecological time-series analysis under heterogeneous atmospheric conditions

Sachin Admane[1,*], Tejas Admane[2], Aditi Admane[3], Sanket Kasture[3]

[1] Department of Civil Engineering, Sanjivani College of Engineering, Kopargaon, India
[2] School of Civil and Environmental Sciences, JSPM University Pune, Maharashtra, India
[3] Faculty of Medicine, Kasr Al Ainy, Cairo University, Cairo, Egypt

ARTICLE INFORMATION

Article Chronology:
Received 24 January 2026
Revised 19 July 2026
Accepted 10 August 2026
Published 29 September 2026

Keywords:
Ambient air pollution; Cardiovascular morbidity; Short-Term exposure; Generalized additive models; Particulate matter

CORRESPONDING AUTHOR:
sachinadmane@gmail.com
Tel: (+91) 91301 91301
Fax: (+91) 91301 91301

ABSTRACT

Introduction: Short-term associations between ambient air pollution and cardiovascular morbidity remain inconsistent across different settings. This study evaluated these associations using Generalized Additive Models (GAMs) and examined the robustness of the findings through lag and sensitivity analyses.

Materials and methods: An ecological time-series analysis was conducted using a publicly available environmental–health dataset comprising 5,811 daily observations of air pollutant concentrations, meteorological variables, and aggregated cardiovascular morbidity indicators. GAMs with penalized smoothing splines were fitted to assess potentially non-linear exposure–response relationships while adjusting for meteorological covariates. Lag structures (Lag 0–3 days), alternative spline dimensions, and multi-pollutant models were evaluated as sensitivity analyses.

Results: The final GAM demonstrated limited explanatory capacity (AIC=25,602.9; AICc=25,604.6; explained deviance=1.04%). Pollutant smooth terms showed shallow non-linear exposure–response patterns (effective degrees of freedom: 8.36-9.40), but none were statistically significant ($PM_{2.5}$: p=0.357; PM_{10}: p=0.950; NO_2: p=0.594; SO_2: p=0.339; O_3: p=0.530). Sensitivity analyses produced consistent results across alternative lag structures and spline dimensions, and model diagnostics indicated satisfactory fit without evidence of substantial over dispersion.

Conclusion: Only shallow, statistically non-significant short-term associations were identified. These findings should be interpreted within the limitations of the ecological design, potential exposure misclassification, and residual confounding.

Please cite this article as: Admane S, Admane T, Admane A, Kasture S. Detectability of short-term associations between ambient air pollution and cardiovascular morbidity: An ecological time-series analysis under heterogeneous atmospheric conditions. Journal of Air Pollution and Health. 2026;11(3):307-324.
Doi: https://doi.org/10.18502/japh.v11i3.22622

Introduction

Cardiovascular Diseases (CVDs) remain a leading cause of global morbidity and mortality, contributing substantially to the worldwide disease burden. In addition to established behavioral and metabolic determinants, ambient air pollution is recognized as a major environmental risk factor for cardiovascular health. A large body of evidence demonstrates that exposure to particulate matter and gaseous pollutants adversely affects cardiovascular function through mechanisms including systemic inflammation, oxidative stress, endothelial dysfunction, and autonomic imbalance [1, 2].

Although the long-term cardiovascular impacts of chronic air pollution exposure are well established, evidence for short-term exposure–response relationships remains heterogeneous. Time-series and case-crossover studies have linked short-term fluctuations in pollutant concentrations with acute cardiovascular events such as myocardial infarction, stroke, arrhythmias, and heart failure; however, reported effect sizes and temporal patterns vary considerably across regions and study designs [3-5]. This variability likely reflects differences in pollutant mixtures, baseline exposure levels, population susceptibility, and meteorological conditions, all of which influence the detectability of short-term effects [6, 7]. Particulate matter, particularly $PM_{2.5}$ and PM_{10}, plays a central role in cardiovascular risk due to its ability to penetrate the respiratory system and induce systemic responses [1, 8]. Gaseous pollutants including NO_2, SO_2, and O_3 have also been implicated, although evidence for their short-term cardiovascular effects is less consistent and frequently influenced by co-pollutant interactions and atmospheric chemistry [5, 9].

Despite extensive investigation, the shape and magnitude of short-term exposure–response relationships remain incompletely characterized. Many studies rely on linear or log-linear assumptions that may oversimplify complex biological processes and obscure potential nonlinearities or thresholds [10, 11]. This limitation is particularly relevant in high-exposure environments, where incremental short-term fluctuations may contribute limited additional physiological impact relative to sustained background exposure. Furthermore, relatively few studies have simultaneously examined multiple pollutants using a unified non-linear modeling framework while explicitly evaluating the robustness of observed associations through complementary lag and sensitivity analyses.

Generalized Additive Models (GAMs) provide a flexible and robust framework for evaluating nonlinear exposure–response relationships while accounting for temporal trends, seasonality, and meteorological confounding. These models are widely applied in environmental epidemiology and are well suited for analyzing count-based outcomes such as daily cardiovascular morbidity [12-14]. Their flexibility also enables characterization of complex exposure–response patterns without imposing restrictive functional assumptions, thereby providing a more comprehensive assessment of potential short-term health effects.

Meteorological conditions further complicate interpretation, as temperature, humidity, and wind speed influence both pollutant dynamics and cardiovascular physiology [15, 16]. Accordingly, rigorous adjustment for temporal structure, seasonality, and atmospheric variability is essential in short-term analyses. Recent evidence suggests that short-term air pollution effects are highly context dependent and may be weak or statistically undetectable in settings characterized by high background pollution, aggregated health outcomes, or limited exposure contrast [17-19]. Importantly, the absence of statistically significant associations should not be interpreted

as absence of biological effect but may reflect limitations in epidemiological detectability under specific data conditions.

Against this background, the present study investigates short-term associations between ambient air pollution and cardiovascular morbidity using an ecological time-series framework applied to a heterogeneous dataset. Concentrations of $PM_{2.5}$, PM_{10}, NO_2, SO_2, and O_3 are evaluated using generalized additive models that explicitly account for temporal structure, seasonality, nonlinear exposure–response relationships, alternative lag structures, and multiple sensitivity analyses to evaluate the robustness of the observed associations.

Unlike many previous ecological time-series studies that primarily focused on identifying statistically significant pollutant effects, the present analysis also emphasizes the stability, robustness, and detectability of short-term exposure–response relationships under varying modeling assumptions. By integrating multi-pollutant modeling, lag comparisons, sensitivity analyses, and quantitative exposure–response characterization within a unified GAM framework, this study provides a more comprehensive methodological evaluation of short-term air pollution–cardiovascular associations.

Crucially, this study focuses on the detectability and stability of short-term associations rather than presuming their presence. By explicitly examining how exposure variability, outcome aggregation, and atmospheric context influence observable epidemiological signals, this work provides a methodologically grounded perspective on interpreting short-term air pollution–cardiovascular relationships.

Materials and methods

Study design

An ecological time-series design was used to assess short-term associations between ambient air pollution and cardiovascular morbidity at the population level. Daily observations were analyzed to evaluate whether temporal variation in pollutant concentrations corresponded to variation in aggregated cardiovascular case counts. This design is widely applied in air pollution epidemiology when individual-level data are unavailable, provided that temporal structure and confounding are adequately addressed [12, 13]. The study focused on short-term associations using daily observations and evaluated the robustness of the findings through alternative lag structures and sensitivity analyses.

Data source

The analysis utilized a publicly available dataset from the Air Quality and Health Impact repository, comprising 5,811 daily observations linking air pollutant concentrations, meteorological variables, and aggregated cardiovascular case counts. Data were preprocessed prior to release and represent population-level indicators derived from multiple monitoring sources. The dataset includes daily measurements of $PM_{2.5}$, PM_{10}, NO_2, SO_2, O_3, temperature, humidity, wind speed, and total cardiovascular case counts. Air pollution measurements represent area-level monitoring data and therefore characterize average environmental exposure rather than individual exposure. The descriptive characteristics of all study variables are summarized in Table 1, while their distributional characteristics are illustrated in Fig. 1.

Table 1. Descriptive statistics of air pollutants, meteorological variables, and cardiovascular morbidity

Variable	Mean	SD	Median	Minimum	Maximum	IQR	Skewness
$PM_{2.5}$ (μg/m³)	100.224	58.097	100.506	0.032	199.985	101.905	−0.003
PM_{10} (μg/m³)	148.655	85.699	147.635	0.016	299.902	147.062	0.019
NO_2 (μg/m³)	102.293	57.713	102.988	0.01	199.98	98.12	−0.054
SO_2 (μg/m³)	49.457	28.53	49.53	0.011	99.97	48.459	0.026
O_3 (μg/m³)	149.312	86.534	149.56	0.002	299.937	149.38	0.011
Temperature (°C)	14.975	14.483	14.942	−9.991	39.963	24.984	0.005
Relative Humidity (%)	54.777	26.021	54.544	10.002	99.997	45.646	0.023
Wind Speed (m/s)	9.989	5.777	10.052	0.002	19.999	10.019	−0.011
Cardiovascular Cases (cases/day)	4.989	2.217	5	0	14	3	0.446

SD = standard deviation; IQR = interquartile range.

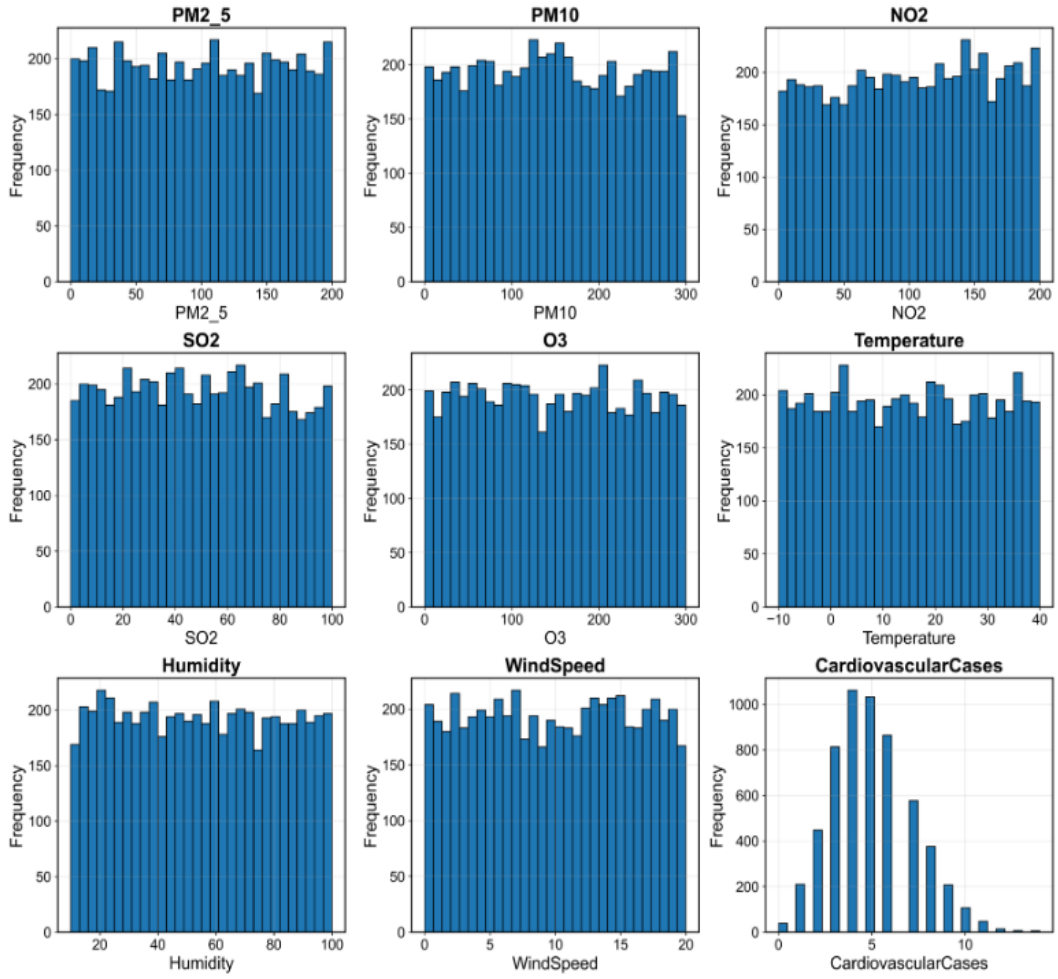

Fig. 1. Distribution of air pollutants, meteorological variables, and cardiovascular morbidity

Fig. 1. Distribution of air pollutants, meteorological variables, and cardiovascular morbidity. Histograms with density curves illustrate the distributions of $PM_{2.5}$, PM_{10}, NO_2, SO_2, O_3, temperature, relative humidity, wind speed, and daily cardiovascular case counts. Air pollutant concentrations exhibited broad temporal variability, whereas cardiovascular morbidity showed comparatively limited variation, providing important context for interpretation of subsequent exposure-response analyses.

Individual-level information on demographics, clinical diagnoses, and personal exposure was not available; therefore, analyses were conducted at the ecological level. Potential limitations, including exposure misclassification and outcome aggregation, were considered in study design and interpretation.

Outcome definition

The outcome was the daily aggregated count of cardiovascular cases. Diagnostic subcategories, inclusion and exclusion criteria, severity information, and diagnostic coding (e.g., ICD classification) were not available in the public dataset; therefore, the outcome represents a composite indicator of cardiovascular morbidity. Aggregation across heterogeneous conditions may attenuate short-term associations and reduce sensitivity to acute exposure effects [3, 4].

Exposure assessment

Exposure was characterized using daily mean concentrations of $PM_{2.5}$, PM_{10}, NO_2, SO_2, and O_3. These pollutants are associated with cardiovascular outcomes through inflammatory, oxidative, and vascular mechanisms [1, 7, 9]. All variables were treated as continuous and assumed to represent population-average exposure. Correlation among the air pollutants and meteorological variables was evaluated prior to model development to assess potential multicollinearity (Table 2).

Table 2. Pearson correlation coefficients among air pollutants and meteorological variables

Variable	$PM_{2.5}$	PM_{10}	NO_2	SO_2	O_3	Temperature	Humidity	Wind Speed
$PM_{2.5}$	1	−0.012	0.006	0.016	0.006	−0.002	0.007	0.009
PM_{10}	−0.012	1	0.008	−0.007	−0.004	−0.018	−0.017	−0.019
NO_2	0.006	0.008	1	−0.020	−0.015	0.007	−0.011	−0.001
SO_2	0.016	−0.007	−0.020	1	−0.004	−0.022	0.001	−0.002
O_3	0.006	−0.004	−0.015	−0.004	1	−0.001	0.004	−0.003
Temperature	−0.002	−0.018	0.007	−0.022	−0.001	1	−0.000	0.001
Humidity	0.007	−0.017	−0.011	0.001	0.004	−0.000	1	0.021
Wind Speed	0.009	−0.019	−0.001	−0.002	−0.003	0.001	0.021	1

Pearson correlation matrix used to assess multicollinearity among explanatory variables before model development.

Spatial variability, fixed-site monitoring limitations, and individual activity patterns could not be captured, introducing potential non-differential exposure misclassification. The Air Quality Index (AQI) was excluded from multivariable models to avoid collinearity with individual pollutant metrics [10].

Meteorological covariates

Daily mean temperature, relative humidity, and wind speed were included as covariates to account for atmospheric variability and potential confounding. These variables influence pollutant behavior and cardiovascular physiology and are routinely adjusted for in short-term epidemiological analyses [15, 16]. Penalized smoothing functions were used to flexibly model their potentially non-linear relationships with cardiovascular morbidity.

Statistical analysis

Short-term associations were estimated using Generalized Additive Models (GAMs) with a Poisson distribution and log link. GAMs were selected for their ability to flexibly model non-linear exposure–response relationships while accommodating count-based outcomes [12, 14]. Temporal trends and potential seasonal variation were controlled using smooth functions of calendar time, while day-of-week effects were incorporated as indicator variables. Penalized regression splines with a basis dimension of k=10 were adopted for the primary analysis to balance model flexibility and overfitting, and sensitivity analyses were conducted using alternative spline dimensions (k=5 and k=15). The principal GAM results are presented in Table 3 and visualized in Fig. 2.

Table 3. Generalized additive model results for pollutant-specific exposure–response relationships

Pollutant	EDF	Minimum Effect	Maximum Effect	Effect Range	Mean Effect	95% CI Width	Exposure–Response Pattern
$PM_{2.5}$	9.395	0.037	0.11	0.074	0.06	0.204	Shallow Increasing
PM_{10}	8.368	0.096	0.14	0.044	0.118	0.212	Shallow Increasing
NO_2	8.386	0.034	0.088	0.054	0.054	0.205	Shallow Decreasing
SO_2	8.358	0.123	0.217	0.093	0.174	0.211	Shallow Decreasing
O_3	8.366	0.116	0.189	0.073	0.161	0.208	Shallow Increasing

EDF = effective degrees of freedom; CI = confidence interval.

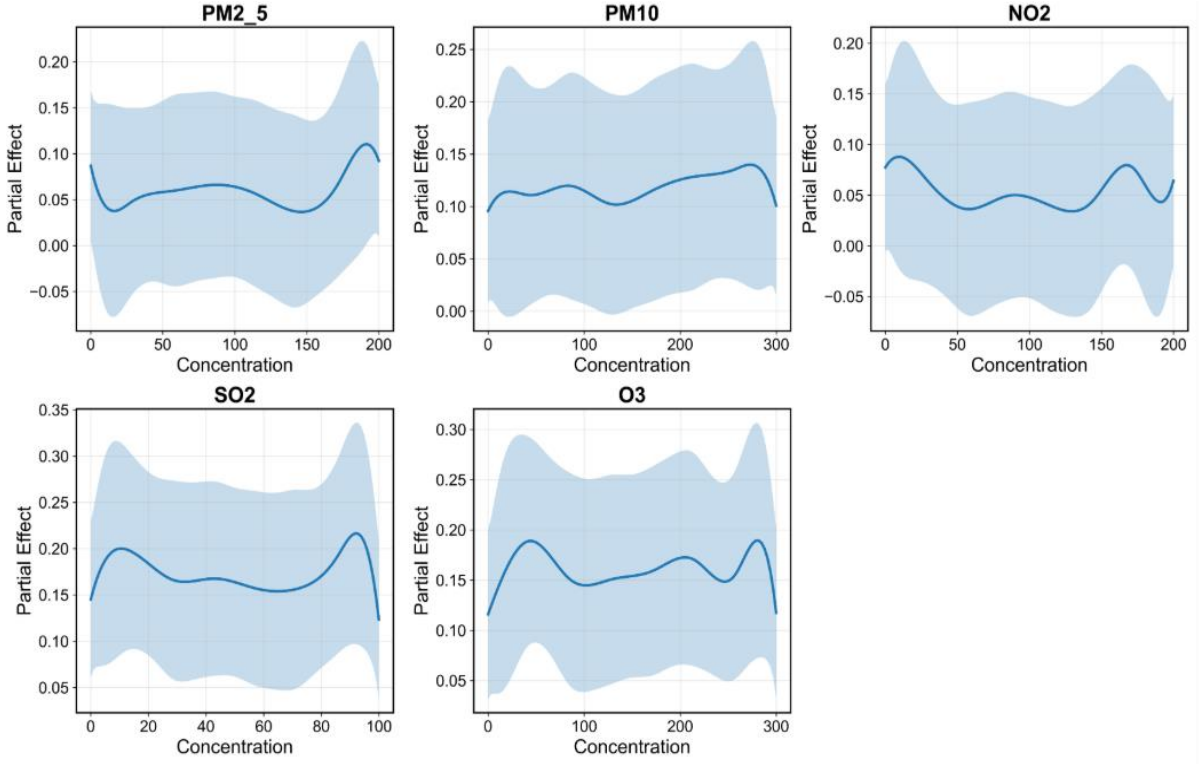

Fig. 2. Pollutant-specific exposure–response relationships estimated using generalized additive models

Fig. 2. Estimated non-linear exposure–response functions for $PM_{2.5}$, PM_{10}, NO_2, SO_2, and O_3 obtained from the final generalized additive model. Solid lines represent the estimated smooth functions, and shaded regions indicate the corresponding 95% confidence intervals. All pollutants exhibited shallow, non-linear exposure–response relationships with no statistically significant associations, pronounced thresholds, or saturation effects across the observed exposure ranges.

Single-day lag models (Lag 0–3 days) were evaluated separately to examine potential delayed effects. This lag window was selected because previous short-term cardiovascular epidemiological studies have consistently reported that acute air pollution effects, when present, are most frequently observed within the first few days following exposure [3-5]. In addition, multi-pollutant models and alternative smoothing specifications were evaluated to assess the robustness of the primary findings.

Overdispersion and statistical inference

Overdispersion was assessed using the dispersion statistic and residual diagnostics. The estimated dispersion value (0.204) indicated no evidence of substantial overdispersion, supporting the suitability of the Poisson modeling framework. Statistical significance was evaluated using the approximate smooth-term tests provided by the pyGAM framework. Because smoothing parameters are estimated during model fitting, these p-values were interpreted cautiously and considered supportive rather than definitive inferential measures. Interpretation emphasized effect magnitude, stability, and functional form rather than sole reliance on statistical significance [12, 13].

Model diagnostics and sensitivity analysis

Model adequacy was assessed using deviance residuals, residual distribution, fitted-versus-observed relationships, residual–fitted correlations, Root Mean Squared Error (RMSE), and Mean Absolute Error (MAE). Diagnostic statistics are summarized in Table 4, while graphical diagnostic assessments are presented in Fig. 3. Sensitivity analyses examined alternative spline dimensions, multi-pollutant models, and Lag 0–3 structures to evaluate the stability of exposure–response relationships and the robustness of model conclusions [11, 17].

Fig. 3. Diagnostic assessment of the final generalized additive model, including residual distribution, residual histogram, residual boxplot, and residuals versus fitted values. Residuals were approximately symmetric and centered near zero, with negligible residual–fitted correlation and no evidence of substantial overdispersion, supporting the adequacy of the fitted model.

Table 4. Diagnostic statistics for the final generalized additive model

Metric	Value	Interpretation
Residual Mean	−0.079	Near-zero residual bias
Residual SD	1.008	Residual variability
Residual Skewness	−0.058	Approximately symmetric residuals
Residual Kurtosis	0.087	No marked heavy tails
RMSE	2.204	Root mean squared prediction error
MAE	1.747	Average absolute prediction error
Dispersion	0.204	No evidence of substantial overdispersion
Residual–Fitted Correlation	0.009	Negligible residual dependence
Maximum Residual	3.368	Largest observed residual

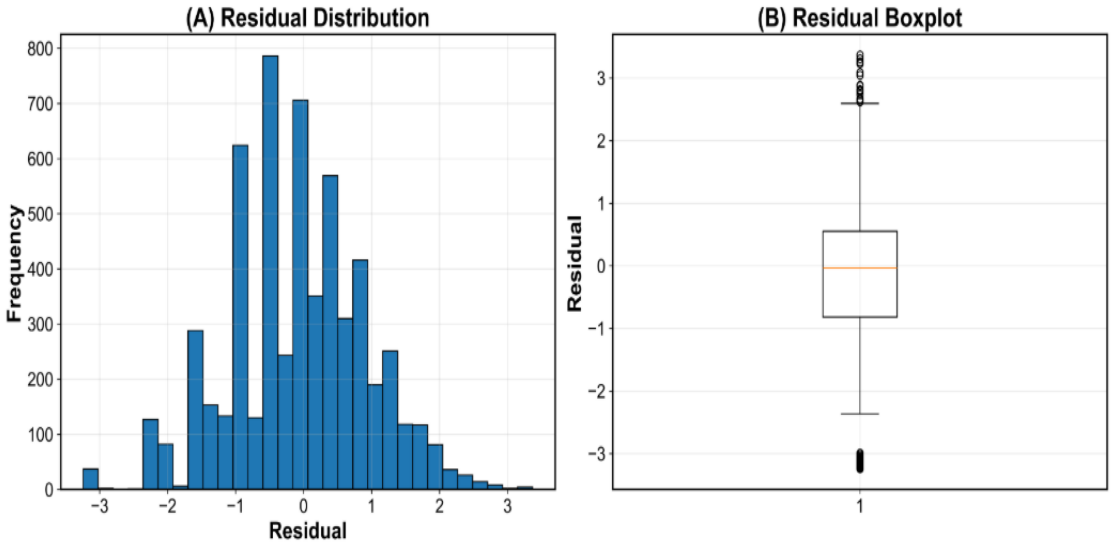

Fig. 3. Diagnostic evaluation of the final generalized additive model

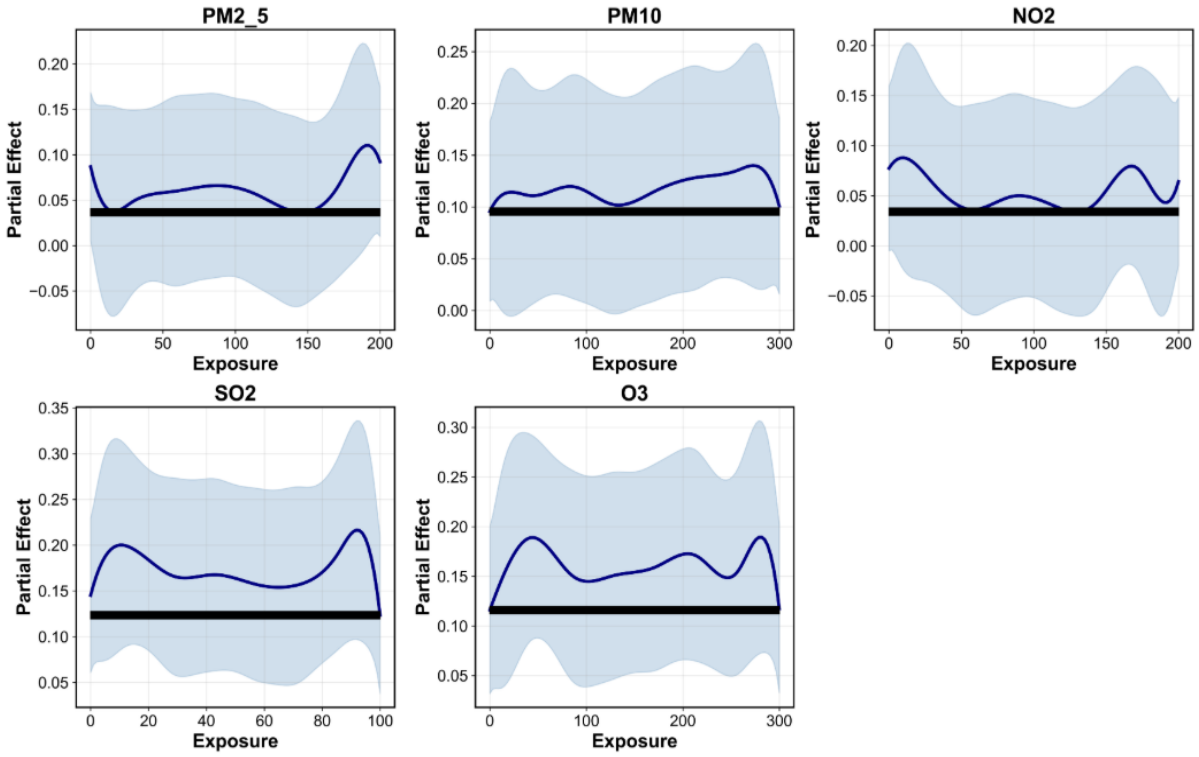

Fig. 4. Comparative exposure–response patterns across ambient air pollutants

Fig. 4. Comparative visualization of pollutant-specific exposure–response functions estimated using generalized additive models. The figure facilitates direct comparison of the magnitude, direction, and shape of the estimated smooth functions for $PM_{2.5}$, PM_{10}, NO_2, SO_2, and O_3. Consistent with the quantitative analyses, all pollutants demonstrated relatively shallow exposure–response relationships with only minor differences in estimated effect magnitude.

Software

All analyses were conducted in Python using pandas, NumPy, and the pyGAM library for generalized additive model estimation. Statistical summaries, diagnostic analyses, sensitivity analyses, and graphical visualizations were generated using standard scientific computing libraries.

Results and discussion

Descriptive characteristics

Descriptive statistics are presented in Table 1. Substantial day-to-day variability was observed across all air pollutants. Mean concentrations of $PM_{2.5}$ and PM_{10} were 100.22 and 148.65 µg/m³, respectively, with wide interquartile ranges (101.91 and 147.06 µg/m³), indicating pronounced exposure heterogeneity. Gaseous pollutants also exhibited broad distributions, particularly O_3 (IQR: 149.38 µg/m³). Meteorological variables showed moderate variability, with temperature ranging from −9.99 °C to 39.96 °C. In contrast, cardiovascular morbidity exhibited comparatively limited variation, with a mean of 4.99 cases/day (SD=2.22), an interquartile range of 3 cases, and a maximum of 14

cases/day. Overall, pollutant concentrations displayed substantially greater temporal variability than cardiovascular case counts, providing important context for interpretation of subsequent exposure–response analyses. The corresponding variable distributions are illustrated in Fig. 1.

Correlation structure

Pearson correlation coefficients among pollutants and meteorological variables are summarized in Table 2. Correlations were uniformly weak ($|r| \leq 0.03$), indicating minimal multicollinearity among explanatory variables. These findings support simultaneous inclusion of multiple pollutants and meteorological covariates in the generalized additive models without evidence of substantial collinearity. The near-independence of predictors likely reflects the aggregated nature of the dataset and preprocessing procedures, which may attenuate real-world co-pollutant relationships.

Distributional characteristics

Distributional characteristics are illustrated in Fig. 1. Pollutant concentrations were generally symmetric, whereas cardiovascular case counts showed mild positive skewness (skewness=0.45), consistent with aggregated count data. The combination of broad exposure distributions and relatively limited variability in cardiovascular morbidity suggests reduced statistical contrast for detecting short-term associations. These distributional characteristics are consistent with the descriptive statistics presented in Table 1.

Exposure-response relationships

Generalized additive model results are summarized in Table 3, while the estimated pollutant-specific exposure-response functions are presented in Figs. 2 and 4. The final model demonstrated limited explanatory capacity (AIC = 25,602.9; AICc = 25,604.6; explained deviance = 1.04%). None of the pollutant smooth terms reached statistical significance ($PM_{2.5}$: p = 0.357; PM_{10}: p = 0.950; NO_2: p = 0.594; SO_2: p = 0.339; O_3: p = 0.530). Effective degrees of freedom ranged from 8.36 to 9.40, indicating flexible but stable smooth functions. Exposure–response curves were consistently shallow, with no evidence of marked thresholds, inflection points, or saturation effects across the observed exposure ranges.

Model diagnostics and sensitivity analyses

Model diagnostics (Table 4 and Fig. 3) demonstrated satisfactory model performance. Residuals were centered near zero (mean = −0.079), with minimal residual–fitted correlation (0.009), RMSE of 2.20, MAE of 1.75, and a dispersion statistic of 0.204, indicating no evidence of substantial overdispersion. Sensitivity analyses using alternative spline dimensions (Table 5 and Fig. 6), single-day lag structures (Table 7 and Fig. 5), and alternative model specifications (Table 6 and Fig. 6) produced consistent findings. These analyses demonstrated that the observed shallow exposure–response relationships remained stable across alternative modeling assumptions, supporting the robustness of the primary results.

Table 5. Sensitivity analysis of generalized additive models under alternative spline dimensions

Spline Dimension (k)	AIC	AICc	ΔAIC	Explained Deviance	Δ Explained Deviance	Scale	Log-Likelihood	Interpretation
5	25559.65	25560	0	0.005	0	1	−12749.413	Comparable model fit
10	25602.92	25604.6	43.271	0.01	0.005	1	−12733.508	Higher model complexity
15	25641.51	25645.5	81.868	0.016	0.011	1	−12715.591	Higher model complexity

(k). ΔAIC represents the difference relative to the reference model.

Table 6. Comparison of alternative generalized additive model specifications

Model	Predictors	AIC	Explained Deviance (%)	Relative Performance
Meteorology Only	Temperature + Humidity + Wind Speed	25600.09	0.6	Best fit
Multi-pollutant	$PM_{2.5}$ + PM_{10} + NO_2 + SO_2 + O_3	25602.92	1	Comparable
$PM_{2.5}$ Only	$PM_{2.5}$	25613.98	1	Inferior
SO_2 Only	SO_2	25616.47	0.9	Inferior
NO_2 Only	NO_2	25618.17	0.9	Inferior
O_3 Only	O_3	25621.87	0.9	Inferior
PM_{10} Only	PM_{10}	25622.59	0.8	Inferior

Table 7. Comparison of lag-specific generalized additive models

Lag (days)	Observations	AIC	ΔAIC	Explained Deviance	Scale	Model Support
Lag 0	5811	25692.21	5.293	0.02	1	Moderate
Lag 1	5810	25693.46	6.546	0.02	1	Moderate
Lag 2	5809	25695.55	8.632	0.018	1	Lower
Lag 3	5808	25686.92	0	0.019	1	Best-supported

Fig. 5. Comparison of generalized additive model performance across Lag 0-Lag 3 exposure structures. Model comparison based on Akaike Information Criterion (AIC) and explained deviance demonstrates that the principal findings remain stable under alternative lag assumptions, indicating robust short-term exposure–response estimates.

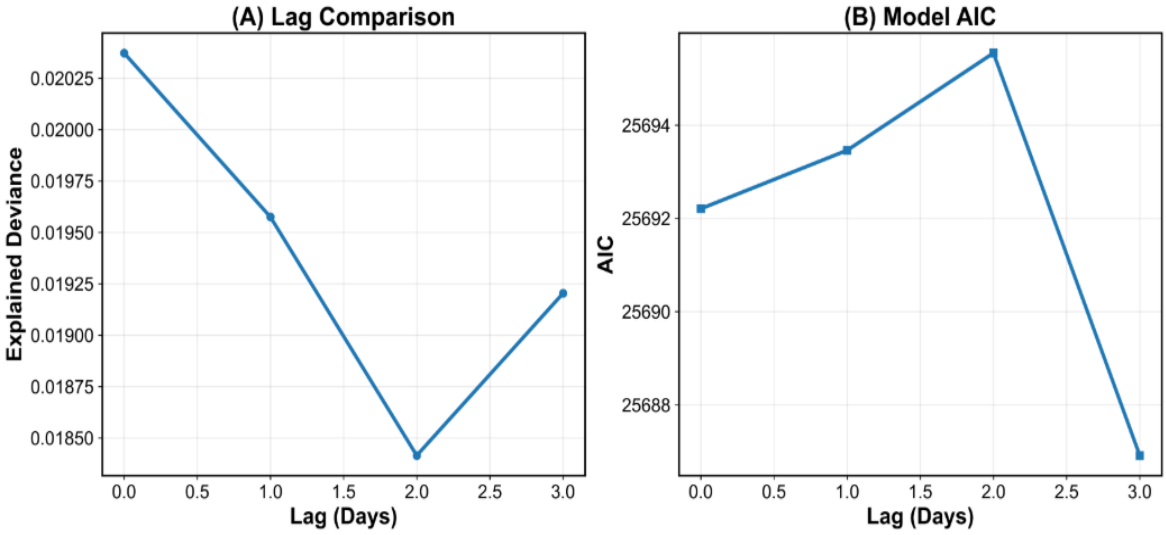

Fig. 5. Sensitivity analysis of lag-specific generalized additive models

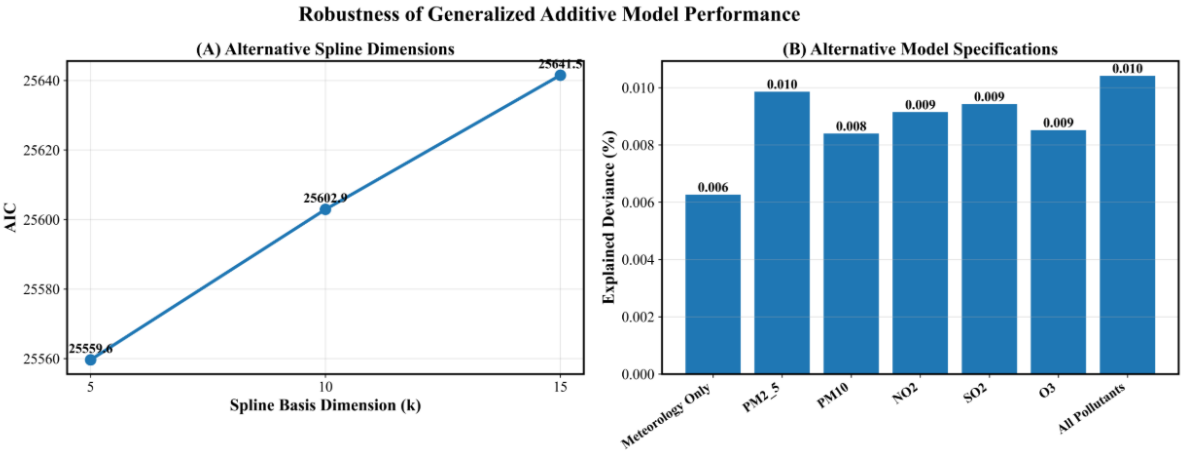

Fig. 6. Robustness of generalized additive model performance under alternative model specifications

Fig. 6. Combined sensitivity analysis illustrating the influence of alternative spline basis dimensions (k = 5, 10, and 15) and different model specifications (meteorology-only, single-pollutant, and multi-pollutant models). Model performance is compared using Akaike Information Criterion (AIC) and explained deviance. The consistent performance across alternative specifications indicates that the principal conclusions are robust to changes in model complexity and predictor selection.

Summary of findings

Despite substantial temporal variability in ambient air pollutant concentrations, the generalized additive models identified only shallow and statistically non-significant short-term associations with aggregated cardiovascular morbidity. These findings were consistent across alternative lag structures, spline specifications, and model formulations (Tables 3 and 5-7; Figs. 2 and 4-6). Accordingly, the absence of statistically significant associations in the present analysis should be interpreted within the context of the ecological study design, aggregated health outcomes, limited variability in daily cardiovascular case counts, and potential exposure measurement uncertainty, rather than as evidence that short-term biological effects do not exist.

This study evaluated the detectability of short-term associations between ambient air pollution and cardiovascular morbidity using an ecological time-series framework under heterogeneous exposure conditions. Despite substantial variability in pollutant concentrations and meteorological factors, exposure–response relationships were consistently shallow, statistically non-significant, and stable across alternative lag structures and sensitivity analyses (Tables 3 and 5-7; Figs. 2 and 4-6). Diagnostic results (Table 4 and Fig. 3) indicated satisfactory model performance, suggesting that the observed findings primarily reflect the characteristics of the available data and study design rather than model misspecification.

Interpretation of findings

The uniformly shallow exposure–response functions (Table 3; Figs. 2 and 4) indicate limited short-term sensitivity of aggregated cardiovascular morbidity to day-to-day fluctuations in ambient air pollution. Importantly, the absence of statistically significant associations should not be interpreted as absence of biological effect, but rather as limited epidemiological detectability under the available ecological data structure. Cardiovascular outcomes arise from complex and often cumulative processes influenced by long-term exposure, pre-existing disease, lifestyle, and healthcare access. Acute physiological responses to air pollution, including inflammation, oxidative stress, endothelial dysfunction, and autonomic imbalance, may not consistently translate into measurable short-term clinical events, particularly when daily variability in cardiovascular case counts is limited relative to exposure variability.

Exposure context and detectability

The dataset is characterized by persistently elevated and highly variable pollutant concentrations, as summarized in Table 1 and Fig. 1. In such settings, short-term fluctuations may contribute only modest incremental physiological stress relative to sustained background exposure. Previous studies have similarly reported weak or heterogeneous short-term associations under

high-exposure conditions, emphasizing the importance of exposure contrast in determining epidemiological detectability [5, 17, 18]. The absence of pronounced thresholds or saturation effects in the exposure–response curves (Figs. 2 and 4) further suggests that daily pollutant variation was not associated with substantial short-term changes in aggregated cardiovascular morbidity. These findings should therefore be interpreted as context-specific rather than generalized evidence against short-term air pollution effects [18-20].

Outcome aggregation and measurement constraints

A central limitation affecting detectability is the aggregated nature of the cardiovascular outcome. The composite case count includes heterogeneous cardiovascular conditions with differing etiologies, susceptibilities, and temporal responses, potentially diluting pollutant-specific short-term effects that may be more apparent for individual clinical endpoints [3, 4]. Furthermore, ecological studies are inherently susceptible to ecological fallacy because population-level associations cannot be directly translated into individual-level risk. Exposure assessment was based on area-level monitoring data rather than personal exposure, introducing potential exposure misclassification resulting from spatial variability, indoor exposure, and individual mobility. These factors generally bias associations toward the null and should be considered when interpreting the present findings.

Meteorological considerations

Meteorological variables were included to account for atmospheric and physiological confounding but did not demonstrate strong independent non-linear associations after adjustment for other predictors. This likely reflects outcome aggregation, temporal adjustment, and attenuation of underlying atmospheric relationships within the available dataset. Potential effect modification by season, extreme temperatures, age, sex, or other demographic characteristics could not be evaluated because these variables were unavailable in the public dataset. Consequently, differential susceptibility among population subgroups cannot be excluded. These findings should therefore be interpreted as dataset-specific and do not diminish the established role of meteorological conditions in cardiovascular risk.

Methodological implications

The use of generalized additive models with explicit adjustment for temporal trends, seasonality, meteorological covariates, and non-linear exposure–response relationships (Table 3; Figs. 2 and 4) reduces the likelihood that the observed findings resulted from model misspecification. The consistency of results across alternative lag structures, spline dimensions, and multi-pollutant analyses (Tables 5-7; Figs. 5 and 6) further supports the robustness of the primary conclusions. Nevertheless, residual confounding arising from unmeasured behavioral, socioeconomic, healthcare, or environmental factors remains possible and is an inherent limitation of ecological analyses. More broadly, these results highlight the importance of considering epidemiological detectability when interpreting weak or null findings in short-term air pollution studies. Transparent reporting of such findings is essential for reducing publication bias and

improving evidence synthesis.

Public health implications

The absence of statistically significant short-term associations in the present analysis should not be interpreted as evidence that ambient air pollution poses no cardiovascular risk. Long-term exposure remains a well-established determinant of cardiovascular morbidity and mortality, and susceptible individuals may still experience clinically relevant short-term effects that are not detectable using aggregated ecological data. These findings (Table 3; Figs. 2 and 4) reinforce the importance of sustained reductions in background pollution levels and continued public health efforts to minimize population exposure, particularly in regions experiencing persistently elevated pollutant concentrations.

Strengths and limitations

This study is strengthened by the large number of daily observations, substantial exposure variability (Table 1; Fig. 1), flexible generalized additive modeling, comprehensive diagnostic evaluation (Table 4; Fig. 3), and multiple sensitivity analyses examining lag structures, spline dimensions, and alternative model specifications (Tables 5-7; Figs. 5 and 6). Limitations include the ecological study design, potential ecological fallacy, exposure misclassification associated with area-level monitoring data, aggregated cardiovascular outcomes, absence of diagnostic subcategories and individual-level covariates, and the possibility of residual confounding. These limitations restrict causal interpretation at the individual level and may reduce sensitivity for detecting short-term associations. Future studies incorporating individual-level exposure assessment, clinically specific cardiovascular endpoints, distributed lag approaches, and extended temporal observations may provide a more comprehensive characterization of acute cardiovascular responses to ambient air pollution.

Conclusion

This study evaluated short-term associations between ambient air pollution and cardiovascular morbidity using an ecological time-series framework under heterogeneous exposure conditions. Despite substantial variability in pollutant concentrations and meteorological factors, the generalized additive models identified only shallow and statistically non-significant short-term associations (Table 3; Figs. 2 and 4). Exposure–response relationships remained stable across alternative lag structures and sensitivity analyses (Tables 5–7; Figs. 5 and 6), suggesting that the observed findings were robust within the adopted modeling framework and the characteristics of the available dataset.

These findings indicate that weak or statistically non-significant short-term associations may arise from limited exposure contrast, aggregated cardiovascular outcomes, exposure measurement uncertainty, and other methodological constraints, rather than necessarily indicating the absence of underlying biological effects. Accordingly, the present results should be interpreted within the context of the ecological study design, potential residual confounding, and the limitations of population-level exposure assessment, and should not be considered evidence against the established cardiovascular risks associated with long-term air pollution

exposure.

From a public health perspective, the findings reinforce the importance of continued efforts to reduce ambient air pollution and protect susceptible populations. Future studies incorporating individual-level exposure assessment, clinically specific cardiovascular endpoints, higher spatial resolution, and longer observation periods will be important for improving understanding of short-term cardiovascular responses to ambient air pollution.

Financial supports

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Competing interests

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

Acknowledgements

The authors gratefully acknowledge the contributors and maintainers of the publicly available dataset used in this study. The authors also thank academic colleagues who provided informal feedback during the development of earlier drafts. No external organization or individual influenced the study design, analysis, interpretation, or reporting of results.

Ethical considerations

This study involved secondary analysis of publicly available, anonymized data. No human participants were directly involved, and no identifiable personal information was accessed. Ethical issues (Including plagiarism, Informed Consent, misconduct, data fabrication and/or falsification, double publication and/or submission, redundancy, etc) have been completely ob-served by the authors

References

1. Brook RD, Rajagopalan S, Pope CA III, Brook JR, Bhatnagar A, Diez-Roux AV, et al. Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. Circulation. 2010;121(21):2331–78. doi:10.1161/CIR.0b013e3181dbece1

2. Rajagopalan S, Al-Kindi SG, Brook RD. Air pollution and cardiovascular disease: State-of-the-art review. J Am Coll Cardiol. 2018;72(17):2054–70. doi:10.1016/j.jacc.2018.07.099

3. Mustafic H, Jabre P, Caussin C, Murad MH, Escolano S, Tafflet M, et al. Main air pollutants and myocardial infarction: A systematic review and meta-analysis. JAMA. 2012;307(7):713–21. doi:10.1001/jama.2012.126

4. Shah ASV, Lee KK, McAllister DA, Hunter A, Nair H, Whiteley W, et al. Short-term exposure to air pollution and stroke: Systematic review and meta-analysis. BMJ. 2015;350:h1295. doi:10.1136/bmj.h1295

5. Atkinson RW, Kang S, Anderson HR, Mills IC, Walton HA. Epidemiological time-series studies of air pollution and health. Environ Res. 2014;132:334–43. doi:10.1016/j.envres.2014.03.021

6. Burnett R, Chen H, Szyszkowicz M, Fann N, Hubbell B, Pope CA III, et al. Global estimates of mortality associated with long-term exposure to outdoor fine particulate matter. Proc Natl Acad Sci U S A. 2018;115(38):9592–7. doi:10.1073/pnas.1803222115

7. Pope CA III, Bhatnagar A, McCracken JP, Abplanalp W, Conklin DJ, O'Toole T. Exposure to fine particulate air pollution is associated with endothelial injury and systemic inflammation. Circ Res. 2019;124(1):1–18. doi:10.1161/CIRCRESAHA.118.314186

8. Lelieveld J, Evans JS, Fnais M, Giannadaki D, Pozzer A. The contribution of outdoor air pollution sources to premature mortality. Nature. 2015;525(7569):367–71. doi:10.1038/nature15371

9. Yang BY, Qian Z, Li S, Chen G, Bloom MS, Elliott M, et al. Ambient air pollution in relation to cardiovascular disease incidence and mortality. Environ Res. 2018;164:1–9. doi:10.1016/j.envres.2018.02.036

10. Peng RD, Dominici F, Louis TA. Model choice in time-series studies of air pollution and mortality. J R Stat Soc Ser A. 2006;169(2):179–203. doi:10.1111/j.1467-985X.2006.00410.x

11. Schwartz J, Laden F, Zanobetti A. The concentration–response relationship between $PM_{2.5}$ and daily deaths. Environ Health Perspect. 2011;119(11):1614–9. doi:10.1289/ehp.1103440

12. Dominici F, Peng RD, Bell ML, Pham L, McDermott A, Zeger SL, et al. Fine particulate air pollution and hospital admission for cardiovascular and respiratory diseases. JAMA. 2006;295(10):1127–34. doi:10.1001/jama.295.10.1127

13. Samoli E, Touloumi G, Zanobetti A, Le Tertre A, Schindler C, Atkinson R, et al. Acute effects of air pollution on mortality: A European analysis. Environ Health. 2013;12:1–15. doi:10.1186/1476-069X-12-1

14. Wood SN. Generalized Additive Models: An Introduction with R. 2nd ed. Boca Raton (FL): CRC Press; 2017. (No DOI available)

15. Analitis A, Katsouyanni K, Biggeri A, Baccini M, Forsberg B, Bisanti L, et al. Effects of cold weather on mortality: Results from a multi-country study. Environ Health Perspect. 2014;122(11):1195–202. doi:10.1289/ehp.1306899

16. Gasparrini A, Guo Y, Hashizume M, Lavigne E, Zanobetti A, Schwartz J, et al. Mortality risk attributable to high and low ambient temperature: A multi-country observational study. Lancet. 2015;386(9991):369–75. doi:10.1016/S0140-6736(14)62114-0

17. He J, Gong S, Yu Y, Yu L, Wu L, Mao H, et al. Short-term effects of air pollution on cardiovascular hospital admissions in China. Environ Res. 2020;186:109545. doi:10.1016/j.envres.2020.109545

18. Chen K, Wolf K, Breitner S, Gasparrini A, Stafoggia M, Samoli E, et al. Air pollution and cardiovascular hospital admissions: A multicity time-series analysis. Environ Res. 2021;197:111158. doi:10.1016/j.envres.2021.111158

19. Liu C, Chen R, Sera F, Vicedo-Cabrera AM, Guo Y, Tong S, et al. Ambient air pollution and cardiovascular disease burden: A global assessment. Environ Res. 2022;204:112368. doi:10.1016/j.envres.2021.112368

20. Ren M, Li N, Wang Z, Liu Y, Chen X, Chu Y, et al. Short-term exposure to ambient air pollution and cardiovascular hospitalizations: A

multicity study. Environ Res. 2023;216:114552.
doi:10.1016/j.envres.2022.114552