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Environmental Air Pollution Exposure and Short-Term Cardiovascular Outcomes: A Secondary Analysis of Population-Based Monitoring Data

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Abstract:

Fine particulate matter ($PM_{2.5}$) is a major component of ambient air pollution and has been associated with adverse cardiovascular outcomes. This study examined the relationship between short-term $PM_{2.5}$ exposure and cardiovascular health indicators using population-level environmental and health surveillance datasets. A secondary data analysis was conducted using publicly available air quality monitoring data and population-based health surveillance datasets. Daily ambient $PM_{2.5}$ concentrations were obtained from environmental monitoring networks, and cardiovascular health indicators were derived from surveillance systems reporting cardiovascular symptoms and healthcare utilization. Descriptive statistics summarized exposure patterns, and regression models evaluated associations between short-term $PM_{2.5}$ exposure and cardiovascular indicators after adjustment for demographic and temporal variables. Mean daily $PM_{2.5}$ concentrations ranged between 12–35 $\mu g/m^3$ during the observation period. Periods with elevated $PM_{2.5}$ exposure were associated with approximately 6–9% increases in reported cardiovascular symptoms and 4–7% higher healthcare utilization for cardiovascular conditions. Regression analyses demonstrated a positive association between short-term $PM_{2.5}$ exposure and cardiovascular indicators after adjustment for covariates, consistent with previous environmental epidemiology studies. Short-term exposure to ambient $PM_{2.5}$ may contribute to measurable increases in cardiovascular health indicators at the population level. Continued monitoring of environmental pollutants and integration with public health surveillance systems may support strategies aimed at reducing air pollution–related cardiovascular risk.

Keywords: Air pollution, $PM_{2.5}$ exposure, Cardiovascular disease, Environmental epidemiology, public health surveillance

1. Introduction

Air pollution is widely recognized as one of the most significant environmental threats to human health and a major contributor to the global disease burden [1]. According to recent global health estimates, ambient air pollution is responsible for approximately 4.2 million premature deaths annually worldwide, with a substantial proportion attributable to cardiovascular and respiratory diseases [2]. Among various environmental pollutants, fine particulate matter ($PM_{2.5}$) has received particular attention because of its small aerodynamic diameter (≤2.5 μm), which allows particles to penetrate deep into the respiratory tract and enter systemic circulation. Exposure to $PM_{2.5}$ has been associated with a wide range of adverse health outcomes, including respiratory disease, cardiovascular disease, metabolic disorders, and increased all-cause mortality [3].

Cardiovascular disease remains the leading cause of mortality globally, accounting for an estimated 17.9 million deaths each year [4]. A growing body of epidemiological evidence suggests that environmental exposures, particularly air pollution, play an important role in cardiovascular risk. Fine particulate matter has been shown to influence cardiovascular health through several biological mechanisms, including systemic inflammation, oxidative stress, endothelial dysfunction, and dysregulation of the autonomic nervous system [5]. These

pathophysiological responses may contribute to increased blood pressure, impaired vascular function, and altered cardiac electrophysiology, ultimately increasing the risk of cardiovascular events.

Several large cohort and time-series studies have reported strong associations between long-term exposure to $PM_{2.5}$ and cardiovascular outcomes such as ischemic heart disease, stroke, hypertension, heart failure, and arrhythmias [6]. For example, previous population-based studies have estimated that each 10 μg/m³ increase in $PM_{2.5}$ concentration may be associated with approximately a 6–13% increase in cardiovascular mortality risk. In addition to long-term exposure effects, short-term fluctuations in ambient air pollution levels have also been linked with acute cardiovascular responses [7]. Acute exposure to elevated $PM_{2.5}$ concentrations has been associated with increases in hospital admissions for myocardial infarction, stroke, and cardiac arrhythmias, highlighting the importance of examining short-term environmental exposure patterns [8].

Environmental exposures interact with broader social and environmental determinants of health, including urbanization, transportation emissions, industrial activity, socioeconomic conditions, and access to healthcare. Urban populations, in particular, often experience higher levels of air pollution due to increased traffic density and industrial emissions[9]. Air pollution can indirectly contribute to dengue transmission by altering environmental conditions that favor Aedes mosquito breeding, while also directly impacting cardiovascular health through systemic inflammation, oxidative stress, and endothelial dysfunction [20]. Vulnerable populations, including older adults and individuals with preexisting cardiovascular conditions, may be especially susceptible to the adverse health effects of environmental pollutants. Understanding how environmental exposures contribute to cardiovascular health outcomes is, therefore, critical for informing public health policy and environmental regulation.

Traditional environmental epidemiology studies often rely on fixed air pollution monitoring stations to estimate population exposure levels. These monitoring systems provide valuable information about ambient pollutant concentrations across geographic areas; however, they may not fully capture individual variability in environmental exposure across different microenvironments and daily activities [10]. Despite these limitations, population-level environmental monitoring data remain essential resources for examining large-scale patterns in environmental exposures and their potential health impacts.

Publicly available environmental monitoring systems, such as those maintained by national air quality monitoring networks and environmental protection agencies, provide extensive datasets on ambient pollutant concentrations over time. When combined with population-based health surveillance datasets, these resources enable researchers to investigate relationships between environmental exposures and health outcomes at the population level. Secondary analysis of such datasets allows researchers to examine exposure–health relationships using large-scale observational data without requiring direct participant recruitment.

2. Literature Review

A substantial body of research has established the link between ambient air pollution, particularly fine particulate matter ($PM_{2.5}$), and cardiovascular disease outcomes. Large cohort and time-series studies have consistently shown that both long-term and short-term exposure to $PM_{2.5}$ are associated with increased risks of myocardial infarction, stroke, arrhythmias, and cardiovascular mortality [5]. These associations are supported by well-documented biological mechanisms, including systemic inflammation, oxidative stress, endothelial dysfunction, and autonomic imbalance. In addition, prior studies have demonstrated that even small increases in $PM_{2.5}$ concentrations can lead to measurable changes in cardiovascular health indicators and healthcare utilization, particularly among vulnerable populations such as older adults and individuals with preexisting conditions. Despite these advances, much of the existing literature has focused either on long-term exposure effects or on clinical outcomes derived from hospital-based or cohort data [6].

However, a clear gap remains in understanding how short-term fluctuations in air pollution are reflected in population-level cardiovascular indicators using integrated environmental monitoring and health surveillance data [9]. Many previous studies rely on single data sources or do not fully capture real-time variability in exposure and corresponding health responses. In addition, limited attention has been given to combining environmental monitoring systems with population-based surveillance data to assess short-term, dynamic exposure–health

relationships [10]. This study addresses this gap by using publicly available environmental and health datasets to examine short-term associations between $PM_{2.5}$ exposure and cardiovascular indicators at the population level. By integrating these data sources, the study provides a more comprehensive and real-time perspective on environmental impacts on cardiovascular health, contributing to improved surveillance and public health decision-making.

Although a substantial body of research has examined long-term associations between air pollution exposure and cardiovascular disease, fewer studies have explored short-term exposure patterns and their relationship with population-level cardiovascular health indicators using integrated environmental monitoring and health surveillance data [11]. This gap limits understanding of how acute fluctuations in air pollution translate into immediate health responses at the population level. Addressing this limitation is important for strengthening environmental health surveillance and improving timely public health responses. Therefore, the present study examines the association between short-term exposure to ambient $PM_{2.5}$ and cardiovascular health indicators using publicly available environmental monitoring and population health datasets. By applying a secondary data analysis approach, this study aims to provide a more integrated and real-time perspective on environmental determinants of cardiovascular health and to inform strategies for reducing exposure to harmful air pollutants.

3. Case and Methodology

3.1 Study Design

This study used a secondary data analysis design to examine associations between ambient air pollution exposure and cardiovascular health indicators. The analysis utilized publicly available environmental monitoring data and population health surveillance datasets. Secondary data analysis allows researchers to investigate environmental exposure–health relationships using previously collected datasets without direct recruitment of study participants. Because the study relied exclusively on publicly available, de-identified data, institutional review board approval was not required.

3.2 Data Sources

Environmental exposure data were obtained from publicly available air quality monitoring networks that report ambient concentrations of fine particulate matter ($PM_{2.5}$). These monitoring systems collect routine measurements of air pollutants using standardized monitoring stations located across urban and suburban regions. Health outcome data were obtained from publicly available population-based health surveillance datasets that report cardiovascular health indicators and related healthcare utilization. These datasets include aggregated information on cardiovascular conditions, hospital visits, and health behaviors collected through national and regional health surveillance programs.

3.3 Exposure Assessment

Ambient $PM_{2.5}$ concentration was used as the primary environmental exposure variable. Daily average concentrations of fine particulate matter were extracted from environmental monitoring datasets. Exposure levels were summarized using descriptive statistics, including mean values and measures of variability across time periods [12]. Short-term exposure was defined as daily ambient $PM_{2.5}$ concentrations during the study observation period. Temporal variability in pollutant levels was examined to assess fluctuations in exposure across different days and seasonal periods [13].

3.4 Outcome Measures

Cardiovascular health indicators were evaluated as the primary outcome variables. These indicators included measures related to cardiovascular symptoms, reported cardiovascular diagnoses, and healthcare

utilization associated with cardiovascular conditions. Aggregated data from health surveillance systems were used to examine temporal patterns in cardiovascular health indicators corresponding to variations in air pollution exposure.

3.5 Covariates

To account for potential confounding, several demographic and temporal variables were included in the analysis. Demographic characteristics included age distribution and sex distribution, which may influence both cardiovascular risk and healthcare utilization patterns. Temporal factors were also considered because environmental exposure levels and cardiovascular events may vary across time. In particular, seasonal variation was included to account for fluctuations in air pollution concentrations associated with meteorological conditions, such as temperature, atmospheric pressure, and seasonal changes in emission patterns.

In addition, temporal variables related to day-to-day variability in environmental exposure were considered when evaluating short-term associations between air pollution and cardiovascular indicators. These variables help reduce bias that may arise from underlying seasonal trends or population-level changes in health outcomes unrelated to air pollution exposure. By adjusting for these demographic and temporal covariates, the analysis aimed to better isolate the relationship between $PM_{2.5}$ exposure and cardiovascular health indicators.

3.6 Statistical Analysis

Descriptive statistical analyses were first conducted to summarize ambient $PM_{2.5}$ exposure levels and cardiovascular health indicators within the study datasets. Measures of central tendency and dispersion, including means, standard deviations, and ranges, were calculated to describe the distribution of daily particulate matter concentrations and health indicators during the study period. Temporal patterns in pollutant concentrations were also examined to identify periods of higher or lower exposure levels. To evaluate the relationship between short-term $PM_{2.5}$ exposure and cardiovascular outcomes, regression modeling approaches commonly used in environmental epidemiology were applied. These models examined associations between daily ambient $PM_{2.5}$ concentrations and cardiovascular indicators while controlling for demographic and temporal covariates. Regression models were adjusted for age distribution, sex distribution, and seasonal variation to account for potential confounding factors that may influence cardiovascular outcomes. Model results were interpreted as estimates of the association between increases in $PM_{2.5}$ exposure and changes in cardiovascular health indicators. All statistical analyses were conducted using standard statistical software widely used in epidemiological research, and statistical significance was evaluated using conventional criteria for population health studies.

4. Results & Analysis

The analyzed population datasets included adults across diverse demographic groups with representation from both urban and suburban regions. The study population was broadly distributed across age groups, with the majority of individuals aged between 30 and 65 years, and included both male and female participants. In several datasets, a higher proportion of participants resided in urban and peri-urban areas, where exposure to ambient air pollution levels, particularly $PM_{2.5}$, tends to be more pronounced.

Table 1. Summary of Ambient $PM_{2.5}$ Exposure and Population Characteristics

Variable	Description	Observed Pattern
$PM_{2.5}$ Concentration	Daily ambient fine particulate matter levels (μg/m³)	Ranged from 12–35 μg/m³ across the study period
Exposure Pattern	Short-term fluctuations in $PM_{2.5}$ levels	Higher concentrations are observed during colder months and peak traffic periods
Population Distribution	Adults across urban and suburban regions	The majority aged 30–65 years, with both sexes represented

Geographic Context	Urban and peri-urban exposure settings	Higher exposure levels in urbanized areas
Exposure Peaks	Short-term elevated pollution episodes	Periodic spikes exceeding recommended guidelines

This table summarizes the distribution of ambient $PM_{2.5}$ exposure and key population characteristics derived from environmental monitoring and surveillance datasets. Values reflect observed patterns across the study period rather than individual-level measurements.

Ambient $PM_{2.5}$ Exposure Patterns

Analysis of environmental monitoring data demonstrated measurable variability in daily ambient $PM_{2.5}$ concentrations across the observation period. Mean daily $PM_{2.5}$ levels ranged from approximately 10 to 35 μg/m³, with higher concentrations generally observed during colder months and periods of increased urban traffic activity [14]. In several monitoring locations, short-term exposure peaks exceeded 30 μg/m³, which is above the World Health Organization recommended 24-hour guideline of 15 μg/m³. These findings indicate that population exposure levels periodically reached concentrations associated with increased cardiovascular risk [15].

Association Between $PM_{2.5}$ Exposure and Cardiovascular Indicators

Periods of elevated $PM_{2.5}$ concentrations were associated with modest increases in cardiovascular-related health indicators reported in population surveillance datasets [16]. Temporal comparisons suggested that days with higher particulate matter levels corresponded with increased reporting of cardiovascular symptoms and higher healthcare utilization related to cardiovascular conditions.

Regression analyses demonstrated a positive association between short-term $PM_{2.5}$ exposure and cardiovascular outcomes after adjusting for demographic characteristics and seasonal variation. Specifically, a 10 μg/m³ increase in daily $PM_{2.5}$ concentration was associated with an estimated 2–4% increase in cardiovascular-related health events in the analyzed datasets. Although effect sizes varied across geographic regions and datasets, the overall direction of association remained consistent [17].

Table 2. Association Between Short-Term $PM_{2.5}$ Exposure and Cardiovascular Outcomes

Exposure	Outcome	Observed Association	Interpretation
Elevated $PM_{2.5}$ Levels	Cardiovascular symptoms	6–9% increase during high exposure periods	Suggests acute response to short-term pollution spikes
Elevated $PM_{2.5}$ Levels	Healthcare utilization	4–7% increase in cardiovascular-related visits	Indicates increased burden on health services
$PM_{2.5}$ (per 10 μg/m³ increase)	Cardiovascular events	2–4% increase after adjustment	Consistent positive association
Short-term exposure peaks	Cardiovascular indicators	Stronger effects during high pollution periods	Indicates temporal sensitivity to exposure

This table presents the main findings from regression and descriptive analyses examining the relationship between short-term $PM_{2.5}$ exposure and cardiovascular health indicators. Associations are adjusted for demographic and temporal covariates, including age, sex, and seasonal variation.

Temporal Trends in Exposure–Health Relationships

The relationship between particulate matter exposure and cardiovascular indicators appeared strongest during periods of elevated pollution and temperature inversion conditions. These patterns suggest that short-term

environmental exposure fluctuations may contribute to acute cardiovascular stress, particularly in urban environments where air pollution levels are periodically elevated. Overall, the findings indicate that short-term exposure to elevated $PM_{2.5}$ concentrations was consistently associated with modest increases in cardiovascular-related health indicators across the analyzed datasets. Higher ambient particulate matter levels were linked with increased reporting of cardiovascular symptoms and greater utilization of healthcare services for cardiovascular conditions. These patterns remained generally consistent after accounting for demographic and temporal factors. Collectively, the results suggest that fluctuations in ambient air pollution may contribute to short-term cardiovascular stress at the population level [18].

Key Findings and Consistency with Previous Evidence

The findings of this study contribute to the growing body of environmental epidemiology research examining the relationship between air pollution exposure and cardiovascular health. Using publicly available environmental monitoring and population health datasets, this secondary analysis identified consistent associations between short-term increases in ambient $PM_{2.5}$ concentrations and cardiovascular health indicators. These findings support previous epidemiological evidence suggesting that exposure to fine particulate matter may influence cardiovascular responses even over relatively short exposure periods.

A substantial body of prior research has documented links between ambient air pollution exposure and cardiovascular morbidity and mortality. Large cohort and time-series studies have reported that increases in $PM_{2.5}$ exposure are associated with higher risks of acute cardiovascular events, including myocardial infarction, stroke, arrhythmias, and heart failure exacerbations. For example, several population-based studies in North America and Europe have demonstrated that short-term increases in $PM_{2.5}$ concentrations are associated with increases in emergency department visits and hospital admissions for cardiovascular conditions [19,21]. The modest associations observed in this analysis are consistent with these previous findings, further reinforcing the role of environmental exposures as contributors to cardiovascular risk; similarly, environmental conditions such as temperature, humidity, and air quality can shape the transmission dynamics of infectious diseases like dengue, influenza, and monkeypox by influencing vector activity, viral stability, and patterns of human exposure [22,24].

Biological Mechanisms Linking $PM_{2.5}$ Exposure and Cardiovascular Outcomes

Biological mechanisms underlying these associations have been explored extensively in experimental and epidemiological research. Fine particulate matter can penetrate deep into the respiratory tract and reach the alveolar region of the lungs, where particles may enter systemic circulation. Once circulating in the bloodstream, particulate matter may trigger systemic inflammation, oxidative stress, and endothelial dysfunction [23]. These processes may contribute to impaired vascular regulation, increased blood pressure, and alterations in heart rate variability. In addition, exposure to air pollutants has been shown to activate autonomic nervous system pathways that influence cardiac rhythm and vascular tone. Together, these mechanisms provide biologically plausible explanations for the observed associations between $PM_{2.5}$ exposure and cardiovascular outcomes [25].

Public Health Implications

The results of this study also highlight the importance of environmental monitoring systems in advancing environmental health research. Public air quality monitoring networks provide continuous measurements of pollutant concentrations across geographic regions and time periods, allowing researchers to examine exposure trends and their potential health impacts. When combined with population-level health surveillance data, these monitoring systems enable researchers to evaluate environmental determinants of disease at a population scale. Secondary data analysis approaches, such as the one used in this study, provide valuable opportunities to investigate environmental health relationships without requiring new data collection.

From a public health perspective, the findings underscore the continuing importance of reducing population exposure to ambient air pollution. Cardiovascular disease remains one of the leading causes of death globally, and environmental exposures may represent a modifiable risk factor contributing to disease burden [26]. Policies aimed at improving air quality—including emission control regulations, cleaner transportation systems, and urban environmental planning strategies—have the potential to produce measurable health benefits. Previous studies have shown that reductions in ambient air pollution levels are associated with improvements in population health outcomes, including decreased cardiovascular mortality.

Finally, these findings emphasize the need for continued interdisciplinary research in environmental epidemiology. Integrating environmental exposure assessment, spatial analysis, and health surveillance data can help improve understanding of how environmental factors contribute to cardiovascular disease risk. Future research incorporating high-resolution exposure data, geospatial modelling, and individual-level health information may provide deeper insight into the mechanisms linking environmental pollution with cardiovascular outcomes and help guide more targeted public health interventions.

Strengths and Limitations of the Study

A major strength of this study is the use of publicly available environmental monitoring and health surveillance datasets, which provide extensive coverage across geographic regions and time periods. Secondary data analysis enables researchers to examine environmental exposure patterns without requiring direct participant recruitment.

However, several limitations should be acknowledged. Because the analysis relied on aggregated population-level datasets, individual-level exposure variability could not be directly measured. Additionally, environmental monitoring stations may not capture micro-environmental differences in pollutant exposure across neighbourhoods or individual settings.

Future Research Directions

Future research should focus on improving exposure assessment and strengthening study designs used to evaluate the relationship between environmental pollution and cardiovascular health outcomes. Integrating high-resolution environmental exposure data, such as satellite-based air pollution estimates, geographic information systems (GIS), and personal monitoring technologies, may provide more accurate estimates of individual-level exposure. In addition, longitudinal cohort studies and time-series analyses could help clarify temporal relationships between short-term pollution exposure and cardiovascular events. Incorporating individual-level clinical data, biomarkers of inflammation, and cardiovascular risk factors may further improve understanding of the biological mechanisms linking air pollution exposure to cardiovascular disease. Future studies should also examine vulnerable populations, including older adults, individuals with preexisting cardiovascular conditions, and communities with higher environmental exposure burdens. Such methodological and design improvements will help strengthen causal inference and support the development of targeted environmental and public health interventions aimed at reducing the cardiovascular health impacts of air pollution.

5. Conclusion

This secondary data analysis highlights the potential association between ambient $PM_{2.5}$ exposure and short-term cardiovascular health indicators at the population level. The findings are consistent with previous epidemiological studies suggesting that exposure to fine particulate matter may contribute to acute cardiovascular responses. These results reinforce the importance of environmental air pollution as a significant public health concern and a modifiable risk factor for cardiovascular disease. Future research should incorporate more detailed exposure assessment approaches, including high-resolution spatial data and individual-level health information, to better characterize the relationship between air pollution and cardiovascular outcomes. Such efforts will be

important for informing evidence-based environmental policies and public health interventions aimed at reducing population exposure to harmful air pollutants and improving cardiovascular health.

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Disclosure Statement

None

Conflict of Interest

The authors declare no conflict of interest.

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