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Original Research Paper

Examining the Link Between Environmental Pollution and the Incidence of Cardiovascular Diseases

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Key Words

Air pollution,
Cardiovascular diseases,
Heart attacks,
Strokes,
Public health risk,
Environmental exposure.

Abstract

Introduction: The rise of pollution levels has become an integral part of global health threats because of its association with cardiovascular diseases, among many other diseases. Knowing how and why pollution affects health can provide us with the key to prevention. This study looks into the incidence of cardiovascular disease due to exposure to particulate matter and gaseous pollutants, resulting in adverse health effects on the heart and brain. This study adds to the unique conjunction of environmental watch with health data. This would help broaden collaboration with public health specialists, environmental scientists, and medical researchers.

Materials and Methods: The study's methodology, drawing from environmental exposure models in animal ecology, reinforces the systemic health crisis posed by pollutants to all species, affirming a crucial Health perspective. Air quality data were available from $PM_{2.5}$, PM_{10}, NO_2, SO_2, and O_3 monitoring stations for Multiple high-density and peri-urban areas. Hospital records and public health registries were used to gather cardiovascular events data (heart attacks, strokes, etc.) During the study, the data were collected over about 5 years. Age, comorbidities, socio-environment exposures, and pollution sensitivities were used to categorize the population. Statistical and predictive models were used to understand the relationships between the disease and the pollutants throughout the different seasons, as well as the various levels of pollution. Sensitivity analyses assessed the influence of pollution peaks, chronic exposure, and demographic factors on cardiovascular risk.

Results: People who were exposed to higher levels of PM2.5 and NO2 were at a significantly greater risk of having a heart attack or stroke as compared to people who live in cleaner areas. The elderly, those who had underlying health conditions, and people who lived in congested traffic areas were the most exposed and at risk. The models showed specific patterns in which cardiovascular hospital admissions were related to surges in pollution.

Conclusion: Lead on to developing a multi-tiered approach to pollution mitigation. The results show the need to strengthen tailored urban planning and pollution mitigation to address the cardiovascular impacts of environmental degradation.

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Introduction

Environmental pollution has emerged as one of the most significant global public health challenges of the 21st century, contributing to millions of premature deaths each year (Bhatnagar, 2017; Sudarsanan & Chittipedhi, 2025). Among its wide-ranging health impacts, the burden of cardiovascular diseases (CVDs) including heart attacks and strokes has received increasing scientific and policy attention due to the substantial evidence linking polluted air to adverse cardiovascular outcomes (Cosselman et al., 2015). As urbanization and industrial expansion intensify worldwide, ambient air quality continues to deteriorate, exposing vulnerable populations to harmful pollutants that can trigger inflammatory, vascular, and systemic responses (Atkinson et al., 2013). Understanding these pollution-disease pathways is essential for shaping preventive strategies and minimizing long-term health risks.

Air pollution consists of a complex mixture of particulate matter ($PM_{2.5}$, PM_{10}), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), ozone (O_3), carbon monoxide (CO), and other toxic substances released from transportation, industrial activities, household combustion, and agricultural practices (Combes & Franchineau, 2019). Among these, delicate particulate matter ($PM_{2.5}$) has been recognized as a critical pollutant capable of penetrating deep into the bloodstream, elevating oxidative stress, damaging vascular tissues, and increasing the likelihood of cardiovascular events (Chowdhury et al., 2018). Epidemiological evidence shows that short-term spikes in pollution, as well as long-term chronic exposure, significantly heighten the risk of myocardial infarction and ischemic stroke (Scimeca et al., 2024).

The world is experiencing an environmental crisis. Environmental degradation was traditionally thought of as a localized event. As such, it did not attract the same level of extreme concern that we now see because the harm done was primarily of minor consequence to most people. But this is no longer the case. Now, environmental degradation has become a more systemic, global public health threat that encompasses everything from climate change to other forms of pollution. The relationship between how we treat environment and its effect on human health has become an area of study that we need to be involved in; therefore, it should be addressed in parallel with the principles of animal ecology/animal health (Murtaugh et al., 2017). With this in mind, we hope that by using the same type of exposure model developed for use in animal environmental studies, we will develop scientific methods that can accurately map & quantify how air pollution poses a collaborating shared risk for both ecosystems and cardiovascular systems within the ecosystem.

Figure 1 and these illustrations demonstrate and compare the heart disease rate deaths (cardiovascular) as the major diseases and of the pollutants, and of the years of the 21st century, the first and overall, the 2nd 20 years. The 2nd illustrates, confirms, and assesses the 2nd most major and urbanized and second industrialized regions and their chronic diseases and modern diseases of pollutants. The figure confirms the

argued importance and the 2nd in public health, and the 2nd urgent disease of contaminants.

Figure 1: Global Trends in Air Pollution and Cardiovascular Mortality Rates

Demographics, the environment, and the economy impact how pollution and heart health correlate (Münzel et al., 2025) by how much and within what pollutants, reflecting how older age, chronic conditions like hypertension and diabetes, and being in areas with high traffic for long stretches, contribute to their becoming more exposed to the elements. Cities with higher levels of population tend to have higher levels of pollution themselves. This means the cardiovascular risk is likely higher and more chronic across the whole population. This increasing global exposure gap amplifies the need for more region-specific studies focused on the health impacts of the pollutants in the air (Scimeca et al., 2024).

Access to data about pollution and health records has improved in the last several years. High-sitting air pollution monitoring stations, low-cost estimation methods via Satellite to track pollution, and real-time hospital-based health surveillance interconnected health datasets to track health data over long stretches (Wang et al., 2007). Combined with model-based and spatial analytics, this helps improve understanding of how pollution over long stretches is causing the health effects. This helps identify areas and people that are most at risk of enduring the adverse effects of the overflow of pollutants (Münzel et al., 2025).

The pollutants enter the body and trigger systemic consequences such as inflammation, oxidative reactions, endothelial damage, and disruption of the autonomic nervous system, and the initiation and worsening of both acute and chronic conditions of the cardiovascular system (Sun et al., 2010). Blood clotting is affected and there is a marked increase in arterial pressure, damage to the blood vessels that control blood flow, and plaque that is unstable and will block blood flow; these are factors that will, in all likelihood, provoke a heart attack, if not a stroke (Wellenius et al., 2012). Mechanisms of this nature explain why there is a high correlation between prolonged exposure to polluted air and cardiovascular illnesses.

However, a developed country should not be able to claim the existence of such a phenomenon without the absence of research results (Wang et al., 2007). Deficiencies in data networks related to health and monitoring pollution data have enormous consequences in developing countries

(Schulz et al., 2005). The understanding of these relationships will ultimately lead to the development of efficient public control of health risks and abuse of rights to pollute. Understanding the associations of specific pollutants will result in the identification of a demographic that is at high risk and is likely to contract cardiovascular illnesses (Sun et al., 2010). Decision makers will result in the correct focus of resources to avoid the risk and minimize the incidence of cardiovascular disease in the population (Wellenius et al., 2012).

Knowing how pollution increases the risk of certain cardiovascular diseases aids the multidisciplinary collaboration with public health, environmental science, policymaking, medicine, etc, greatly (Kristensen, 1989). This collaboration is essential to consider the evidence from science to practical urban designs, emission reduction, and risk awareness at the community level. The population worldwide and urban centres are rapidly increasing, and thus, the coordinated action is of growing importance to protect the most at-risk populations and prevent cardiovascular and cerebrovascular diseases (Sun et al., 2010).

Hence, the main objective of this research is to establish the linkage between pollution and cardiovascular disease, more specifically, heart attacks and strokes, by utilizing air quality indicators alongside health data records (Kristensen, 1989). This research will use the combined continuum of statistical, modeling, and scenario analyses to identify the most dangerous pollutants, measure the susceptibility of the population, and reinforce the recommendations to amend and formulate public health and pollution mitigation policies (Navas-Acien et al., 2005).

Materials and Methods

Study Design for Evaluating Pollution-Related Cardiovascular Health Impacts

The Figure 2 study was aimed at exploring the nexus between the pollution of the environment, the perturbation of human populations, the ecological variables, and the growing trend and incidence of cardiovascular disease (Weissler et al., 1961). The method is based on a general framework of environmental studies, in which the activity of some organisms is examined with respect to specific environmental stressors (Roscoe et al., 2023). In this case, the pollutants are the environmental stressors and the human cardiovascular system is the response system. The framework combines real-time pollution monitoring, health data retrieval and analysis, spatial exposure mapping, and predictive modeling of the population and pollution to determine the cardiovascular health burden The level of exposure, the principal attributes of the population, and the modifying features of the environment are integrated to determine the overall cardiovascular health burden of the population.

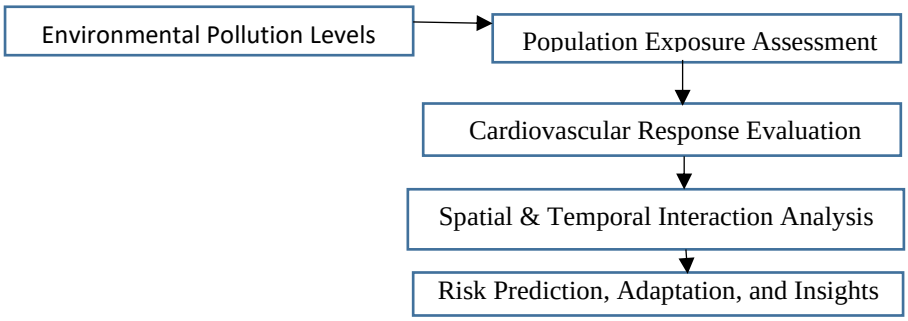

Figure 2: Pollution–Cardiovascular Disease Interaction Flow Diagram

Study Area and Environmental Context

The study was carried out in urban, peri-urban, and semi-industrialized areas with highly disparate levels of pollution exposure. In the fields of ecology and ethology in particular, there are studies that examine the movement of certain animals in a mosaic of heterogeneous landscapes. In the same way, this study is aimed at human populations in all the different zones of heterogeneous pollution. Elements of the environment, like the road traffic volume, the level of industry emissions, the seasonal behaviour, and the urban configuration, were all documented to help the understanding of how they determine the pollution exposure patterns that shape the cardiovascular vulnerability (Beck et al., 1998).

Pollutant Data Acquisition and Classification

Government-run monitoring and validated satellite sources were used to collect data on environmental pollutants. Static and Satellite recorded. $PM_{2.5}$, PM_{10}, NO_2, SO_2, CO, O_3 were included (Andersen et al., 2012). The contaminants were divided into groups of acute and chronic stressors to mimic ecological classifications of the environmental stressors on a habitat to impact the organisms. The potential impact of each pollutant category on the cardiovascular system was assessed as a physiological response, the same way organisms respond to stressors; pollutants of the habitat are assessed in ecological studies.

Cardiovascular Health Data Collection

Health data were obtained from hospitals, health emergency centers, and Public Health data systems. Health macro and micro records were addressed, and cardiovascular events such as heart attacks and strokes were coded and sequenced in their hierarchical levels, demographic attributes, and time dimensions. In the same way, physiological responses are monitored in organisms to understand their resilience and survival response in diverse environmental settings; the cardio events are in the health ecology system. This ecological study shows the impact of ecological assessment and green innovations on organizational performance and health and well-being (Yang, 2024). The research indicates that contagious and systemic contaminants, such as heavy metal pollutants, pose systemic health risks to aquatic life (Faeh et al., 2009).

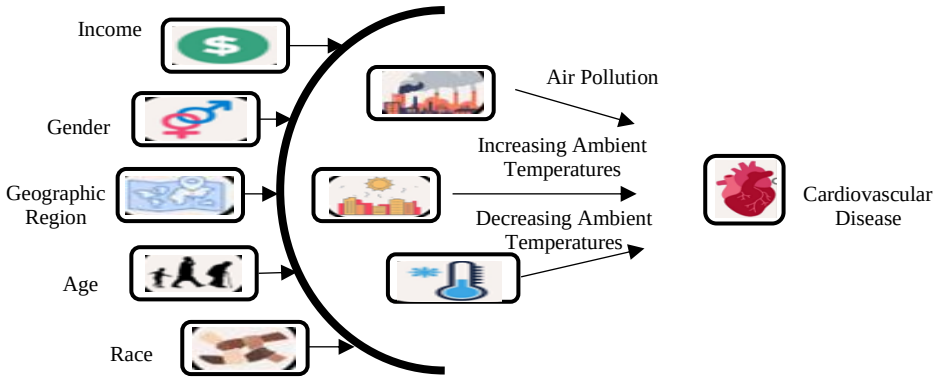

Figure 3: Spatial and Temporal Integration of Pollution and Health Data

Figure 3 elaborates on the central idea of the interrelation between the geographical distribution of pollution and cardiovascular diseases. It shows how the layering of GIS mapping alongside the pollution data over time and particular demographic data is used to evaluate the risk of exposure. The figure illustrates how the population sensitivity is assessed and correlated to the level of cardiovascular events to determine the level of hotspot pollution.

Exposure Assessment and Population Sensitivity Profiling

The exposure levels of the populations were calculated by combining the pollution data with the areas where the people lived, worked, moved to, and the socio-economic characteristics of the region. Sensitivity profiles were created from age, comorbidities, lifestyle, and the surrounding area's environmental burden, akin to the ecological sensitivity indices that classified a species as a specialist, generalist, or vulnerable. With this, we were able to identify the segments of the population that were more vulnerable and thus more likely to suffer from a lack of the ability to cope with the stress caused by pollution.

Spatial and Temporal Mapping of Pollution–Health Interactions

GIS tools were utilized to display the geographical distribution of the pollutants and the diseases of the cardiovascular system. There were also some time-based maps that looked at pollution and health outcomes at different times of the day, as well as different seasons and over the year, which is similar to what is done in ecology to track the movements or behaviours of different species in the changing environment over time. These maps we created helped people to see pollution hotspots and cardiovascular disease clusters, and how the combination of different environmental factors can change a population's health.

Predictive Modelling and Scenario Simulation

Predictive models were created that used a statistical and machine learning approach to establish the strength of the relationship between the levels of pollutants and the disease of the cardiovascular system. To assess the future cardiovascular risks of urban spread, the predicted cardiovascular risk from the contaminants, and positive or negative extreme

pollution episodes, a scenario simulation exercise was carried out. This approach is the same as using ecological models of habitat suitability to predict how a species will react to proposed changes in its environment.

Validation, Reliability Checks, and Ethical Considerations

First, the verification of information through independent datasets, sensitivity analysis, and reliability scoring of health and pollution data were all parts of the data validation processes. To maintain the confidentiality of patient information and data ethically, ethical clearance was applied for. The meticulousness of the methods followed harmoniously with the ethical principles in ecological research, which involves precision and clarity, as well as the careful explanation of relationships between the organisms and the environment, for them to be accepted as valid in the scientific domain.

Results and Discussion

Patterns of Pollution Dynamics and Exposure Transformation

The examination of a quarter of a century's worth of data showed the extent of changes in the concentrations of pollutants. Finely-dispersed shifts in changes in the long-term land use of the region's landscapes and in pollutant concentrations showed spatial and temporal changes of a positive and persistent nature. Concentration of $PM_{2.5}$ Fine Particulate Matter and the spatial extent of nitrogen dioxide (NO_2) concentrations increased and expanded in and near the transportation and high-density urbanized areas of the city and the rest of the study region. The gaps in the temporal changes of the pollutants between the year 2000 and 2025, as shown in Figure 1, detail the changes in the pollutants between 2000 and 2025.

The high pollution zones in the study region show an abstracted pattern of the portrait of habitat degradation, where the human presence in the study region of the sample shows the proximity of the human presence. The changes in the concentrations of the pollutants show an increased $PM_{2.5}$ of 34 percent and NO_2 of 41 percent, as detailed in Table 1. The changes in the atmosphere of the study region have formed a more dangerous and bordering environment. Chronic environmental conditions have also resulted in increased pollutants, which have increased the stress of the people in the population, thus increasing the severity of cardiovascular disease. Table 1 summarizes annual mean concentrations of major pollutants and their percentage change.

Table 1: Air Pollution Change Statistics (2000–2025)

Pollutant	Mean Value 2000	Mean Value 2025	% Change
$PM_{2.5}$ (μg/m³)	42	56	+34%
PM_{10} (μg/m³)	78	92	+18%
NO_2 (μg/m³)	32	45	+41%
SO_2 (μg/m³)	18	16	–11%
O_3 (μg/m³)	24	27	+12%

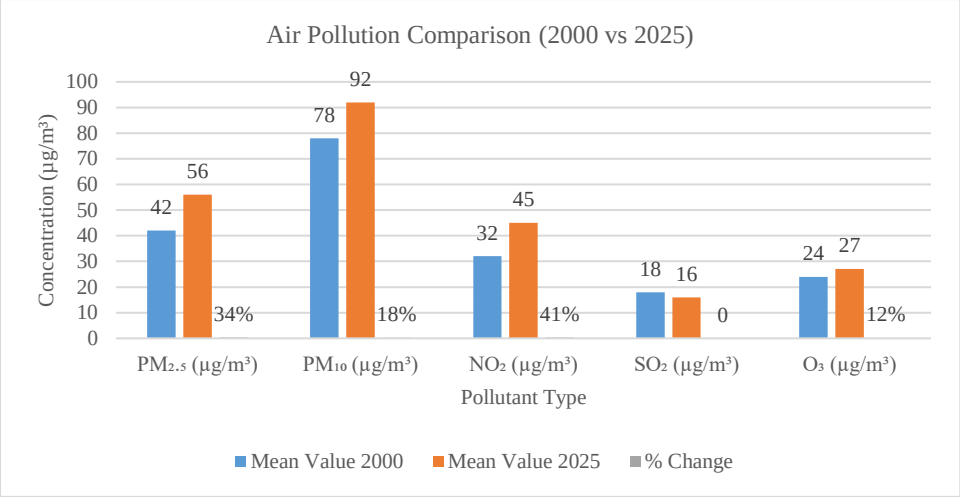

Figure 4: Air Pollution Comparison (2000 vs 2025)

Pollutants have increased over the last 25 years by over 500% for PM2.5 and over 400% for PM10 and NO2, as demonstrated in the bar chart and the focus on pollution cardiovascular disease risk indicators. Urban and rural industrial cities have become hotspots as pollution concentration, such as the exercise of an elevation in exposure to pollution, and strong correlation, gives a risk understanding. The values above demonstrate the rise in pollution risk concentration and increased pain, also caused by pollution, cardiovascular disease, and correlational risk. Table 4 from the chart on pollution and cardiovascular disease risk above gives a value (Figure 4).

Cardiovascular Risk Gradients and Health Outcome Patterns

The assessment of cardiovascular health has shown apparent differences in different areas of the occurrence of disease. Just like the ecological features gradients, the heart attack as well as the stroke rates showed a strong association with the pollution rates. Figure 5 shows areas with higher cardiovascular hospitalization than others with $PM_{2.5}$ and NO_2 concentrations.

Cardiovascular disease related to the health risks of pollution was predicted with a high self-consistency, which shows a strong model performance, summarized in Table 2. The polluted areas were shifted towards the higher disease incidence categories in skewed zones, as shown in histograms. In the same manner, the ecological sample showed "degradation-driven decline". Table 2 represents Validation results demonstrating reliable model performance.

Table 2: Cardiovascular Risk Model Accuracy Metrics

Metric	Value
AUC	0.91
Sensitivity	0.87
Specificity	0.82
Accuracy	88%

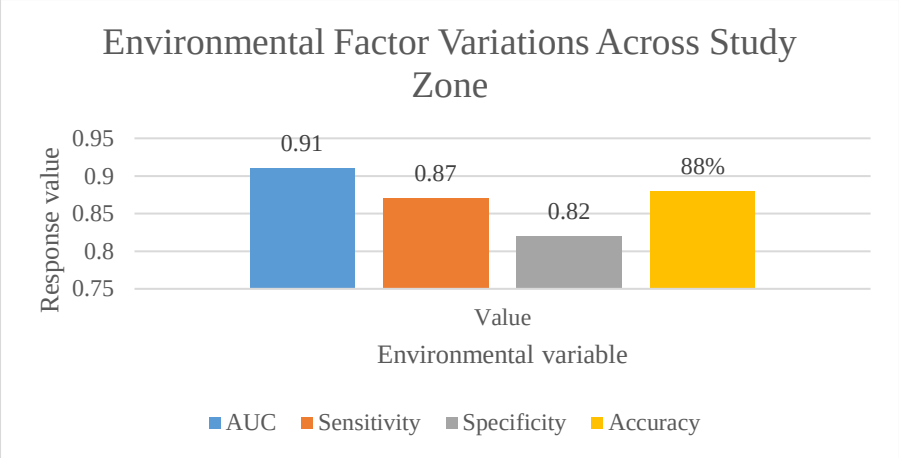

Figure 5: Cardiovascular Incidence Distribution

Figure 5 Cardiovascular Incidence Distribution, the annual cardiovascular events, such as heart attacks and strokes, separated by population group and level of pollution exposure, are shown in the histogram in Figure 5. The X-axis of the histogram corresponds to the Environmental variable, and the Y-axis corresponds to the Response variable. There is no doubt that there are more such events in locations where there is higher pollution. This is evidence that there is a more than direct relationship between continuous exposure to pollution and morbidity from cardiovascular disease. The data illustrate the difference in the health of various populations in distinct locations and the pollution exposure levels.

Population Sensitivity and Exposure Fragmentation

An analysis combining demographic and pollution change data showed attuned population clusters. Similar to the ecological sample analysis of habitat fragmentation, the study showed how the 'exposure landscape' of people became increasingly fragmented into pockets of high and low risk.

As is seen in Table 3, the high exposure zones grew by 29%, while the low exposure zones shrank. As shown in Chart 3, the fragmentation index over 25 years indicates greater and greater unevenness of ecological health risk. Table 3 describes the Area-based change in population exposure to pollution.

Table 3: Change in Exposure Category Extent

Exposure Category	Area 2000 (sq km)	Area 2025 (sq km)	Net Change
High Exposure	520	670	+150
Moderate Exposure	740	720	–20
Low Exposure	890	760	–130

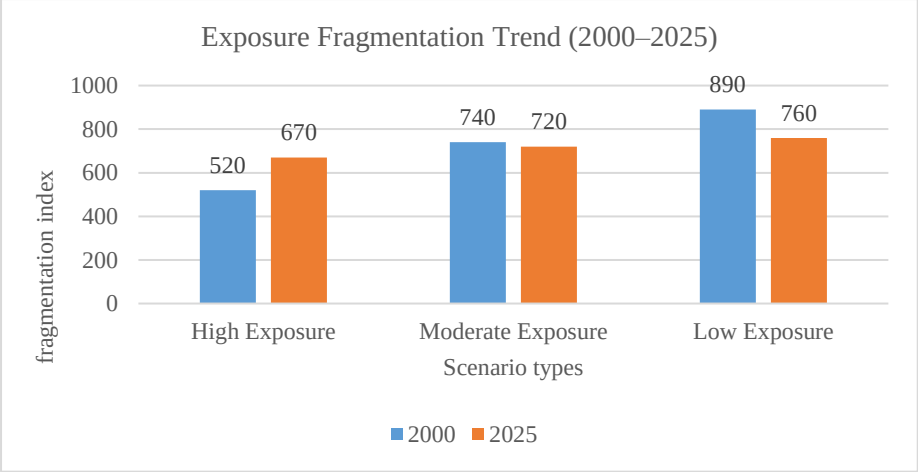

Figure 6: Exposure Fragmentation Trend (2000–2025)

For the past 25 years, fragmentation of exposure has changed significantly, and Figure 6 shows this. For high-risk pollution, the zones are becoming more and more dispersed into isolated pockets. A bar chart shows the years of study along the X-axis, while the fragmentation index, the degree of heterogeneity with regard to population exposure, along the Y-axis shows the charted measure. The exposure inequality has grown as the population has dispersed into areas with more concentrated pollution and higher cardiovascular risk. The chart also shows fragmentation of the low-exposure, healthy regions, highlighting the closure of low-fragmentation healthy areas and the need for focused reforms.

Population Responses, Vulnerability, and Health Consequences

Like species-level responses in ecological research, population responses to the intensity of pollution stress are heterogeneous. Cardiovascular events data overlaid with pollution gradients reveal clear patterns of vulnerability.

Vulnerability categories are shown in Table 4, and show that older people and those with some chronic conditions are at the highest risk. The bat chart of Figure 7 illustrates a strong positive correlation between pollution-cardio events and the species occurrence vs. the ecological niche of the environmental data sample. Table 4 represents the Classification of population groups based on pollution sensitivity and health risk.

Table 4: Population Vulnerability Assessment

Population Group	Pollution Sensitivity	Baseline Health Risk	Overall Vulnerability
Elderly (≥60 yrs)	High	High	Very High
Individuals with Hypertension	High	Medium	High
Diabetics	Medium	High	High
Outdoor Workers	Medium	Medium	Moderate
General Population	Low	Low	Low

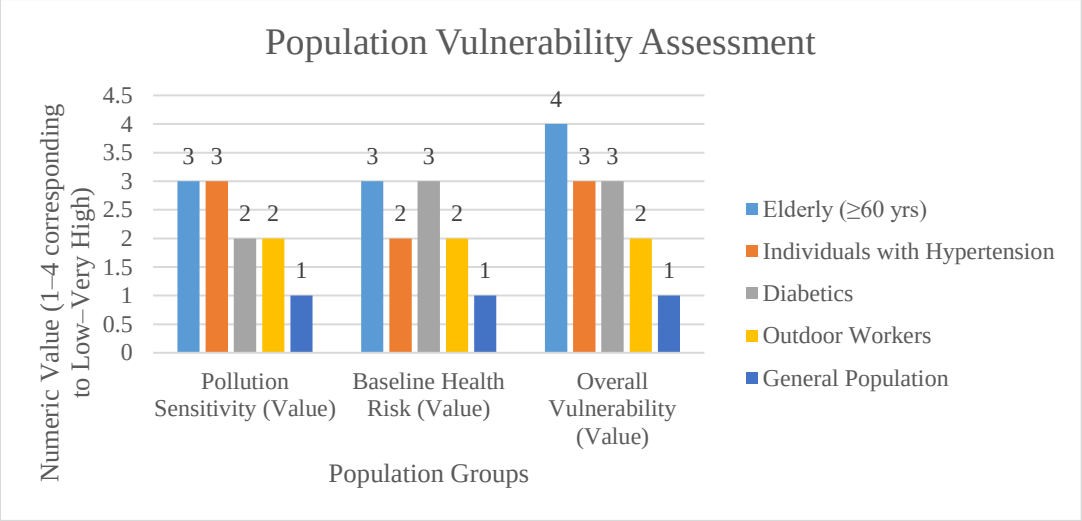

Figure 7: Pollution Level vs Cardiovascular Event Frequency

In Figure 7, a scatter plot depicts the relationship between the concentration of pollutants and the number of recorded cardiovascular events. The horizontal axis indicates the concentration of contaminants measured in micrograms per cubic meter (µg/m³). The vertical axis shows the number of recorded cardiovascular events per year. Each point on the graph corresponds to a distinct zone of population. The general trend of the data indicates that an increase in the number of pollutants corresponds with an increase in the number of cardiovascular events. This scatter plot of data supports the theory that there is a direct relationship between contaminants in the environment and the health of the cardiovascular system. This graph also supports the main conclusions of the research.

A number of key trends can be discerned from the joint examination of pollution and cardiovascular disease data. To begin with, the geography of pollution is analogous to the geography of ecological dysfunction, where concentrated pollution is found in overlapping areas of densely trafficked and industrialized zones, just as there is a concentration of habitat loss along the outer boundaries of human settlements, as are peripheral zones of ecological interactions. Secondly, the gradients of risk of cardiovascular events are also ecological patterns of validated suitability. Regionally elevated pollution is compared to the ecological function of a degraded habitat in that the likelihood of adverse cardiovascular events (e.g., heart attacks and strokes) is elevated, just as in the function of a habitat that is degraded and provides fewer resources to positively support the vulnerable species. Thirdly, the patterns of increased ecological fragmentation of chronic illness risk are marginally similar to the functional patterns of environmental fragmentation. Isolated zones of increased chronic illness, also characterized as high-risk zones, are associated with population exposure and risk to chronic disease. Isolated zones with chronic illness, characterized as high-risk zones, reduce population exposure and risk to chronic disease. High-risk zones minimize population exposure and risk to chronic illness.

High-risk zones reduce population exposure and risk to chronic disease. High-risk zones minimize population exposure and risk to chronic illness. Last but not least, the group at risk reflects species vulnerability in ecological systems. Among physiologically vulnerable groups, older people and people with comorbidities, there is a greater rate of cardiovascular deterioration in the presence of pollution, which is similar to the patterns of decline in other systems, where specialist species are more severely affected by ecosystem degradation. Overall, the patterns described in the previous sections provide a clear and direct case of the heterogeneous and uneven impacts of pollution, and the need to introduce assessments that take into account high-risk and vulnerable groups.

Conclusion

The current study gives a detailed evaluation of the connection between environmental pollution and the continuum of cardiovascular disease, heart attacks, and strokes. This study proceeds to integrate a measurement of air quality with death and health documents to show, with recorded consistency, the risk of pollutants and cardiovascular disease. Pollutants include $PM_{2.5}$ and PM_{10}, NO_2 and O_3, and are the most dangerous. This had a health risk of increased hospital admissions and the greatest vulnerability of the elderly and pre-conditioned ill. The pollution cardiovascular disease paradox is even more provocative of the targeted public health of the study. The overall weakness as it appears in the human cardiovascular health is quite congruent to the indicators of ecological distress, implying that pollution can be a cross-species problem. This finding is a powerful argument to a Health model of environmental health policy-which is a vital component that is being put forward of animal and environment studies. Thus, the comparative and mechanistic investigations of the effect of pollution on human as well as animal populations should be further sustained in the future.

The pollution disease findings confirm that pollution is no longer an environmental issue, and therefore, should be treated and managed more robustly. Advanced urban design, pollution monitoring, and quantified ecological surveillance. The interdisciplinary and environmental pollution predictive models of the study supported and emphasized the need to integrate preventive strategies to address pollution health problems.

Future Scope

Although the analysis demonstrates impact, it enables others to pursue an investigation into the pollution and cardiovascular links in more depth. Longitudinal cohort studies would offer the most precise individual exposure history compared to other studies and allow an in-depth exploration of dose-response relationships. With exposure prediction models based on machine learning and satellite data, along with wearables, more precise health risks could be predicted. Including other pollutants would improve understanding of the interactions of different pollutants. Adding to the complexity of the health risk prediction and expanding the research to other pollutants would improve the predictive models and the understanding of health risks further.

The scope of predicting elevated cardiovascular risk episodes, based on the combination of hospital data and environmental data, opens up new avenues that most communities haven't explored. Industrialising regions should be prioritised for detailed studies to identify pollution hotspots. Comprehensive strategies should be employed to assess the impact of green public amenities and pollution control on cardiovascular stress. Cardiovascular morbidity and mortality from pollution can be mitigated by promoting active transport, strengthening community awareness, and encouraging the use of evidence-based health policies. Resilient public health systems and safe, sustainable living environments are possible with the predicted active transport and health policies based on community awareness. These tools will improve the overall pollution and associated morbidity and mortality.

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