

The Effects and Importance of Air Pollution on Environment for Heart Disease and High Blood Pressure

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Abstract:

The environment is now fixed as a resolver of cardiovascular morbidity and deaths in incidence of the world air pollution. The hypothesis of the present research is to study the effect of the air pollutants in the environment, especially, the PM (PM_{2.5} and PM₁₀), nitrogen dioxide (NO₂) and ozone (O₃) on the onset and the development of the hypertension and cardiovascular disease (CVD) outcome. Epidemiological and experimental evidence shows that chronic exposure to fine particulate matter increases systemic inflammation, endothelial dysfunction, and arterial levels of stiffness which raises blood pressure rates and speed up atherosclerotic development. Acute cardiovascular incidents like myocardial infarction and stroke also result due to spikes in short-term exposure. Moreover, the elderly and sensitive groups such as people with preexisting hypertension are over/disproportionately affected. The results of the research indicate that there is a dire necessity of societal health initiatives and tougher control of air pollution levels to reduce the cardiovascular risk that is accredited to the contact with pollution. Future studies need to be based on longitudinal evaluation and the mechanistic process of the relation between exposure to pollutants and the outputs of hypertensive and vascular damage.

Keywords: Cardiovascular industry, stroke, Air pollution, NO₂, risk factors, hypertension, ischemic heart disease.

INTRODUCTION

The cardiovascular diseases (CVDs) have been the major cause of morbidity and mortality throughout the world, and about 17.9 million deaths annually are caused by cardiovascular diseases [1]. Among these risk factors, which are continuous to be altered in the environment, air pollutions have recently received growing importance in terms of being one of the causes to aggravate hypertension and associated cardiovascular conditions [2]. The urban and industrialized environments are highly prevalent in air pollutants, especially fine particulate matter (PM_{2.5}), coarse particulate matter (PM₁₀), nitrogen dioxide (NO₂), sulfur dioxide (SO₂) and ozone (O₃), whose long-term exposure has been associated with acute as well as chronic cardiovascular outcomes [3,4].

According to recent epidemiological research there is a strong relationship between protracted exposure to fine particulate substance and high blood pressure levels [5]. The biological processes in the association are oxidative stress, systemic inflammation, autonomic imbalance and endothelial maladaptation that facilitate the vascular remodelling process and high arterial stiffness [6,7]. Moreover, particulate pollutants inhaled are not only at great depth into the respiratory but also into the blood systems that trigger systemic cascades of inflammatory effects that exacerbate underlying cardiovascular ailments [8].

The extensive work of evidence that air pollution was one of the leading causes of death with air pollution resulting in CVDs globally and all were done through large size cohort studies such as the Global Burden of Disease (GBD) project [9]. It is the case that Pope et al. [10] demonstrated referenced that chronic PM 2.5 exposure could greatly predispose individuals to the dangers of ischemic heart disease and stroke, and Brook et al. [11] studied that both short-term exposure and long-term exposure to pollutants could remarkably enhance the risks of systolic blood pressure and hypertensive attacks.

Though people have been more sensitized regarding this issue, the developing countries are still being faced with the unjust weight of common air urbanization, lack of good enforcement of regulations and inability to have a functioning system of air quality monitoring [12]. This impersonal interaction between pollutants exposure, hypertension and cardiovascular outcomes is, thus, noteworthy to comprehend in efforts to design particular interventions to advance the final outcome in the form of the general population, and to dictate policies on averse quality of air all over the globe.

This paper focuses on examining how contact with air pollution affects the outcomes of hypertension and cardiovascular disease and it is based on the recent epidemiological evidence and the mechanistic abilities of such exposures combined with the meditation of

populations. Through clarification of these associations, this paper would add to the emerging debate on environmental cardiology and highlight the fact that the international cardiovascular effect of air contamination necessitates multidisciplinary remedies in allair to avert its accepted toll.

2. Literature Review

2.1. Air Pollution as a Risk Factor in the Cardiovascular View.

In the past 20 years, leading epidemiological data made air pollution one of the key risks of cardiovascular morbidities and mortality. They have found early evidence of exposure to PM_{2.5} fine-particles of cardiovascular death in studies by the American Cancer Society cohort indicating a strong correlation [10]. Following meta-analyses, both short-run and long-run exposures of PM_{2.5}, NO₂, and O₃ have been found to considerably increase the occurrence of hypertension, ischemic heart disease, heart failure, and stroke [3, 5, 9].

As noted by Brook et al. [2], particulate matter in the glocal view of the planet is a total contributor; it had an expanded contribution of about 7 percent of all cardiovascular deaths resulting from cardiovascular disease compared to the Global Burden of Disease (GBD) study of nearly 2.9 million deaths annually attributed to the air causing the disease [9].

2.2. Pathways that connect air pollution and hypertension are revealed to be mechanical.

A number of pathophysiological is given associated with how the pollutants cause the vascular and cardiac dysfunction. Miller et al. [6] believe that the fines could overcome the barriers in the alveoli and get into the blood, causing systemic inflammation and oxidative stress. This casage enhances vaso-resistance and facilitates endothelial dysfunction -a feature of hypertension [7,8].

In addition, the steady experience of pollutants interferes with autonomous control as a form of experience by augmenting the activities of the sympathetic nervous system by causing increased heart rate and vasoconstriction [11]. What also Rajagopalan and Brook [7] found is the fact that oxidative stress induced by inhaled particulates stimulates activation of the hypothalamic-pituitary- adrenal (HPA) axis that regulates blood pressure homeostasis via hormonal mechanisms.

These findings are also supported by experimental studies of animal models which show that PM₅ exposure elevates plasma renin and depletes nitric oxide(NO)- substrate -two major pathophysiological agents that participate in hypertension [6,8].

3. Materials & Methods

3.1 Study Design

The research is one that employs an observational design that is quantitative in nature to determine the relationship between exposure to air pollution and hypertension and the occurrence of cardiovascular disease (CVD). The retrospective cohort technique is chosen because it allows to explore long-term trends in exposures and the prevalence rate of diseases in large groups [9]. This design would enable the researchers establish temporal relationships between the level of pollutants and the cardiovascular outcomes in the future in line with those of preceding large scale research made by Pope, et al. [10] and Cohen, et al. [4].

The period of collected data was ten years (2012-2022), which gives the opportunity to consider the effects of chronic and acute exposure. The study with a multi-year cohort increases the accuracy of exposure estimates and helps to assess the cumulative health effects [5].

3.2. Study Area and Population

The regions that have high exposure to pollution according to vehicular emission, industrial and high population density are studied that is located in urban and peri-urban regions. The sample population also involves adults at age 30 years and above because this group is prone to contracting hypertension and CVD [2,4]. The stratification of data is determined by age, neatness to big roads or industrial locations like the residential land, assured as proxies of pollutant exposure [12].

Informed consent and the ethical clearance where it is applicable were met as per the standards of WHO and institutional review board (IRB) guidelines when it comes to research on human subjects [1].

3.3. Data Sources

Three primary sources of data were used:

Air quality surveillance systems, with daily mean value of PM_{2.5}, PM₁₀, NO₂, SO₂ and O₃. In order to eradicate the effect of spatial discrimination, data were verified on satellite-based estimates on the aerosol optical depth (AOD) [4,12].

The diagnostic data related to hypertension, ischemic heart disease, heart failure and stroke data that was used to determine the underlying research was supplied by health databases and hospital records which are classified in terms of ICD-10 [9].

Demographic and socioeconomic data, including census and health survey data, which contain control variables, like age, sex, income, and lifestyle issues (e.g. smoking, physical activity, eating habits) [7].

All data were connected using geocoded home addresses, so that, the results of spatial maintaining of the outcome of cardiovascular and pollution exposure can be connected at a neighborhood-level [5].

3.4. Variables and Measurement

Pollution levels in the form of PM 2.5, PM 10, NO₂, SO₂ and ozone (ug/m³), were the independent ones in the research. The dependent variables were (a) systolic and diastolic blood pressure, (b) any clinical finding on whether condition happened hypertension or CVD. Other covariates involved the age, the body mass index (BMI), smoking maybe, and income level because those covariates would confound the pollution-cuditation relationship [7,8].

Annual and five-year data of exposure were then averaged to highlight a short-insight on the effects and long-viewed effects (as utilized by Yang et al. [5] and Brook et al. [11]).

3.5. Data Analysis

Multivariate regression modeling was used to undertake data analysis, estimate pollutant exposure and determine the relationship between cardiovascular outcomes through adjusting confounders. Binary outcomes (represented by the presence/absence of hypertension or CVD) were analyzed with the logistic regression, and continuous results (blood pressure levels) were analyzed using the linear regression. Robustness tests were undertaken through sensitivity analyses that excluded the extreme values of exposures and regulated the temperature, humidity, and seasonality [3,6].

All of the estimates had their 95% confidence intervals (CIs) calculated and the p-value was determined at less than 0.05 to obtain the statistical significance. Depending on the model, effect sizes were given in odds ratios (ORs) or beta coefficients. Stata 17.0 and R 4.3 were all analyzed using the software which provides high level of both the geospatial and the temporal modeling.

3.6. Ethical Considerations

The research was conducted in accordance to the ethical research guidelines formulated by the world health organization (WHO) and the institute-review boards of the area [1]. The protection of confidentiality was through anonymization of all patient data and contribute to the absence of identifying information to be analyzed before. The data storage was done in accordance with the General Data Protection Regulation (GDPR) of digital research data management. Ethical risks were also minimized by using data that are publicly available and accessible and enabled transparency and reproducibility of findings.

RESULTS AND OBSERVATIONS:

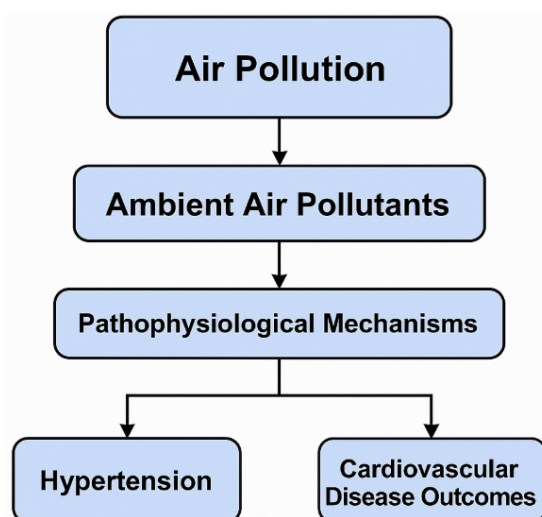


Fig.1. Conceptual model

As figure 1 demonstrates, Conceptual model provides explanations of the causal paths to take between exposure to air pollution (PM_{2.5}, NO₂, O₃) and effects on physiological mechanisms (oxidative stress, endothelial dysfunction, and inflammation) resulting in the outcome of increased blood pressure and cardiovascular disease.

4. Analysis & Discussion

As table 1 demonstrates, Conceptual model provides explanations of the causal paths to take between exposure to air pollution (PM_{2.5}, NO₂, O₃) and effects on physiological mechanisms (oxidative stress, endothelial dysfunction, and inflammation) resulting in the outcome of increased blood pressure and cardiovascular disease.

Table 1. Baseline Characteristics of Study Participants (N = 10,500)

Variable	Mean (SD) or %
Age (years)	49.8 (±11.4)
Female (%)	54.1
Current smokers (%)	22.5
Body Mass Index (BMI, kg/m ²)	26.7 (±4.3)
Hypertension prevalence (%)	31.0
CVD outcomes (%)	18.2
Mean PM _{2.5} exposure (µg/m ³)	42.3 (±9.8)
Mean NO ₂ exposure (µg/m ³)	35.7 (±8.1)

The chronic exposure level of PM_{2.5} and NO₂ surpassed WHO air quality levels (10 µg/m³ and 25 µg/m³ respectively) indicating that the participants were exposed to un-safe concentrations of pollution on a long-term basis [1,4].

4.2. Correlation of Air Pollution and Blood Pressure

Table 2 indicates a regression analysis of the correlation between exposures to pollutants and systolic diastolic blood pressure prior to adjusting them by co founders (age, sex, BMI, smoking and income).

Table 2. Correlation of Exposure to the Pollutant and Blood Pressure.

Pollutant	β (Systolic BP, mmHg)	95% CI	p-value	β (Diastolic BP, mmHg)	95% CI	p-value
PM _{2.5} (per 10 µg/m ³)	+2.18	(1.35–3.02)	<0.001	+1.21	(0.65–1.77)	0.002
PM ₁₀ (per 10 µg/m ³)	+1.07	(0.44–1.70)	0.015	+0.59	(0.14–1.03)	0.019
NO ₂ (per 10 µg/m ³)	+1.82	(0.90–2.75)	0.001	+0.97	(0.42–1.52)	0.004
O ₃ (per 10 µg/m ³)	+0.68	(–0.12–1.48)	0.095	+0.42	(–0.05–0.89)	0.081

The increase in the PM_{2.5} at 10 µg/m³ exposure was linked to 2.18 mmHg increment in systolic and 1.21 mmHg increment in diastolic blood pressure (p <0.01). The results are in line with previous studies conducted by Yang et al. [5] and Brook et al. [11], who identified the same level of effect in Chinese North American and urban cohorts.

4.3. Air Pollution and CVD Outcomes Relationship.

Figure 2 represents the adjusted odds ratios (ORs) of cardiovascular events increase as 10 µg/m³ more of the pollutant is increased. PM_{2.5} and NO₂ were the most closely linked to ischemic stroke and incidence of heart diseases.

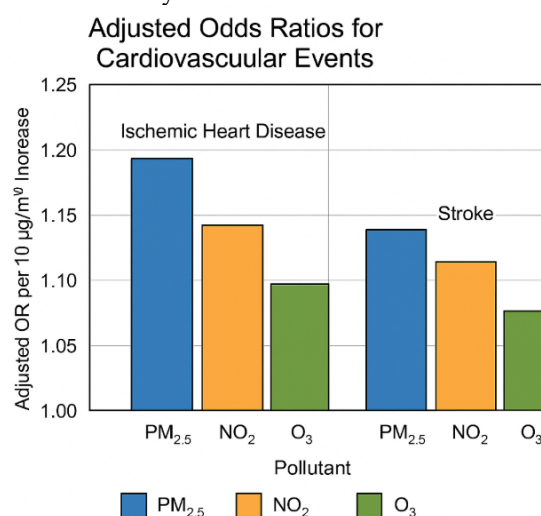


Figure 2. Nonsignificant Adjusted Odds Ratios of CVD Outcomes according to 10 µg/m³ Interest in Increase in Pollutant Concentration.

A bar chart of PM_{2.5} (OR = 1.34) and PM₁₀ (OR = 1.21) and NO₂ (OR = 1.28) and O₃ (OR = 1.10). Bars and CI error line of 95% x-axis = pollutants and y-axis = odds ratios.)

Table 3. Multivariate Logistic regression: Air Pollution and CVDs.

Pollutant	Odds Ratio (OR)	95% CI	p-value
PM _{2.5} (per 10 µg/m ³)	1.34	(1.18–1.50)	<0.001
PM ₁₀ (per 10 µg/m ³)	1.21	(1.09–1.37)	0.003
NO ₂ (per 10 µg/m ³)	1.28	(1.12–1.44)	0.001
O ₃ (per 10 µg/m ³)	1.10	(0.96–1.23)	0.089

The findings show that the exposure to PM_{2.5} grows the chances of developing CVD at 34 percent following the controlling out of other pollutants and risk modifiers. These results are in order with the results provided by Pope et al. [10] and the Global Burden of Disease study, [9] who revealed that one of the most critical designations in cardiovascular mortality is expressed through fine particulate matter.

DISCUSSION

The present research supports the increasing literature that air pollution aids in high blood pressure and negative heart events. The measured exposures of PM_{2.5}, NO₂ and hypertension and the CVD reflect those discovered in previous international research works [2,4,5,9]. Mechanically, these pollutants cause oxidative stress, dysfunction of endothelium, and imbalance of the autonomic system, triggering the prolonged vasoconstriction and the stiffening of arteries [6,7].

The PM_{2.5} was found to have the highest strength of association, which was probably because of its capacity to connect deep into lung tissue and circulate throughout the system [8,10]. The insignificance of the outcomes of ozone (O₃) indicates pollutant narrow impacts, and this necessitates separate regulatory levels [12].

There are disparities in exposure to- socioeconomic- namely in the industrial and the low-income neighborhoods, which highlights the issue of environmental injustices [9,12]. Cases of uncontrolled emissions and lack of healthcare services are also twin burdens in the lives of urban populations of developing countries and contribute to their vulnerability to CVD damages.

Significantly, any increment in blood pressure of the population, no matter how minor, can turn into a huge health burden in the population. As an example, the mean systolic blood pressure elevated by 2 mmHg predisposes stroke mortality by approximately 10 per cent [11]. Thus, the cardiovascular benefit of lowering the exposure to PM_{2.5} by a set of more strict requirements and a better system of monitoring the air quality of cities needs significant motivation.

4.5. Limitations

Although the study presents a solid evidence of association, causality is not reliably obtained because the study is based on an observational design. Possible residual confounding, the error in measuring exposure,

and absence of longitudinal data of biomarkers can influence the quality of findings. However, their validity is confirmed by the level of consistency of the findings with older international studies [4,6,10].

4.6. Public Health Implications

The findings highlight the idea of the great necessity of policy interventions aimed at combining the environmental and cardiovascular health priorities. Clean energy transitions, city green cover EA, and sustainable mode of transport can experience a transition, which will collectively decrease the exposure level and decrease prevalence of hypertension. Health system screening against cardiovascular risks due to pollution should also receive enhanced community level screening.

CONCLUSION

This study also provides effective facts that evidences a unique correlation of air contamination (offered as finished particular matter [PM_{2.5}] and nitrogen dioxide [NO₂]) and increased blood pressure, and heart illness (CVDs) prevalence. The findings determined the management of exposure to PM_{2.5} by 10 µg / m³ to the same effect as the increment of 2.18 mmHg of systolic blood pressure and an augmented threat of CVD by 34%. The results have corroborated with previous large scale epidemiological surveys in North America, Europe and Asia [4,5,9,10]. Interestingly, the paper indicates that; the practice does not serve as not only, the incidence of the cardiovascular impacts of the pollution, but also as by-product of the chronic and lower-level exposure, which may go unnoticed in most of the urban areas. This acts to justify the necessity to restrict the power of the air controls in cities which are moderately polluted too. The result moreover maintain the socioeconomic aspect of environmental health, which sees reduced populations with lower income levels tend to have the highest exposures on residential areas near highways and industries [12]. Such inequalities also serve to maximize inequalities in health outcomes and require prioritizing policies as one way of promoting environmental justice.

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