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Health effects of outdoor air pollutants

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Abstract

One of the most common causes of death and sickness worldwide is cardiovascular disease. A growing body of research suggests that exposure to air pollution increases the risk of cardiovascular disease. Especially children, the elderly, pregnant women, and those from lower socioeconomic backgrounds are more susceptible to the impacts of air pollution. This study aims to explore the health effects of pollutants on the cardiovascular system and respiratory systems. This study follows a literature study method. The study found that exposure to outdoor air pollution is related to immediate and chronic health consequences, from mild physiologic abnormalities to death, especially from respiratory and cardiovascular illnesses. Chemical air pollutants harm patients, especially with chronic respiratory and cardiac disorders, but also asthmatic youngsters. Short-term exposure worsens chronic diseases, and increases symptoms, medical care needs, and mortality, while long-term, repetitive exposure enhances the cumulative risk of these diseases and death.

Keywords: Air pollutants, Cardiovascular system, Respiratory system.

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1. Introduction

It is now commonly known that exposure to outdoor air pollution is associated with a wide range of acute and chronic health impacts, ranging from modest physiologic abnormalities to death, particularly from respiratory and cardiovascular disorders [1,2]. Human exposure to high amounts of ambient particles has been demonstrated to stimulate bone marrow and the production of neutrophils, band cells, and monocytes into circulation. The cytokines IL-1, IL-6, IL-8, the hematopoietic growth factor, and the granulocyte-macrophage colony-stimulating factor are enhanced in the circulation of individuals exposed to elevated amounts of ambient particles (GM-CSF).

Alveolar macrophages and bronchial epithelial cells play a significant role in the secretion of these circulating cytokines, which are plausible candidates for the bone marrow response associated with lung inflammation [1-3]. Stimulation of bone marrow is an essential part of the systemic response characterized by an increase in circulating leukocytes, platelets, pro-inflammatory, and prothrombotic proteins. As one of the known indicators of overall cardiovascular morbidity and mortality, systemic inflammation stimulates the liver to create acute phase proteins such as CRP and fibrinogen that may enhance blood coagulability [4-6].

1.1. Purpose of study

A growing body of research suggests that exposure to air pollution increases the risk of cardiovascular disease. This study aims to explore the health effects of pollutants on the cardiovascular system and respiratory systems.

2. Materials and Methods

This study used a literature review method to arrive at the conclusions for this study. The study used data from related research conducted by the previous researchers. The findings are discussed and the effects of air pollutants are explained. The process of conducting the research and the results of the research did not harm the environment, humans, or living organisms.

3. Results

In their study, Chuang *et al*. [7] found that urban air pollution is related to inflammation, oxidative stress, blood coagulation, and autonomic dysfunction simultaneously in young, healthy adults, with sulfate and ozone as the two major pollutants contributing to these effects. Increases in high sensitivity-C-reactive protein (hs-CRP), 8-hydroxy-2'-deoxyguanosine (8-OHdG), fibrinogen, plasminogen activator inhibitor-1 (PAI-1) and decreases in heart rate variability, which measures alterations in cardiac autonomic control, were found to be associated with increases in levels of PM_{10}, $PM_{2.5}$, sulfate, nitrate, and ozone in single-pollutant models.

One of the additional mechanisms responsible for the cardiovascular consequences of air pollution is connected with the automatic cardiac tone, which may lead to the instability of a vascular plaque, an increase in heart rate, or the onset of dysrhythmias [8-10]. Lipsett *et al*. [11] demonstrated that elevated levels of ambient PM_{10} and $PM_{2.5}$ may hurt heart rate variability in older individuals with coronary artery disease. Berger *et al*. [12] revealed that elevated concentrations of ultrafine particles, accumulation mode particles, $PM_{2.5}$, and nitrogen dioxide enhanced the risk for supraventricular and ventricular runs at practically all delays. Another mechanism is linked to cardiovascular risk factors such as myocardial infarctions and hypertension [13,14].

Short-term inhalation of fine particles and ozone at normal concentrations (120 ppb) in an urban environment causes acute conduit artery vasoconstriction, as measured by high-resolution vascular ultrasonography (exposure to fine particles plus ozone caused significant brachial artery vasoconstriction compared to filtered air inhalation) [15]. PM_{10} was significantly favorably linked with flow-mediated vasodilation and thiobarbituric acid reactive substances (TBARS), but negatively associated with end-systolic basal brachial artery diameter (p<0.05) [16].

3.1. Health effects of pollutants on the Respiratory System

The presence of chemical pollutants in the air has detrimental consequences on people with pre-existing chronic respiratory diseases (CRD) [17], more so in children with asthma [18]. Bronchitis symptoms in asthmatic children were associated with increasing concentrations of $PM_{2.5}$, organic carbon, NO_2, and O_3 in a 4-year cohort study [19].

Exposure to a short-term substance increases the risk of symptoms, medical attention, and mortality while long-term, repetitive exposure increases the cumulative risk of CRD and death. Numerous studies have demonstrated correlations between air pollution and hospital admissions for CRD in the short-term [20-25]. Several writers have investigated the long-term impact of air pollution by comparing the prevalence of asthma or bronchial hyperresponsiveness in regions with varying levels of air pollution [26-28]. According to the findings of Jorres' study, O_3 exposure may increase the development of bronchial allergic responses in previously non-asthmatic individuals with solely allergic rhinitis [29]. With the highlights of these studies, we now know that pollutants are not only associated with the worsening of asthma but may also play a role in the etiology of asthma and that further analysis of common characteristics of pollutants may help elucidate mechanisms linking exposures to the genesis of asthma [30-32].

It is stated that excessive PM_{10} pollution levels and moderately high O_3 levels are associated with a reduction in lung function as evaluated by peak expiratory flow (PEF), an increase in asthma symptoms, and an increase in the usage of asthma drugs [33-35]. After short-term exposure to O_3, patients with extrinsic asthma were found to be more sensitive to inhaled pollen allergen [36]. Increases in PM_{10} and NO_2 concentrations were related to an increase in nighttime chest symptoms and the use of relief inhalers and nebulizers among COPD patients. With greater SO_2 and O_3 concentrations, COPD patients have reported significant increases in PEFR variability, bronchodilator use, and respiratory symptoms [37-39].

Pollutants were also related to impaired pulmonary function, both incident and prevalent respiratory infections, and acute respiratory symptoms in healthy people, particularly youngsters [40-45]. The Children's Health Study [46], which examined the lung function of 10–18-year-old schoolchildren and evaluated their cumulative exposure to various contaminants over 8 years, was a noteworthy 1990s-era air quality research endeavor. In this study, it was found that lung function growth deficits were connected with a correlated set of pollutants, including $PM_{2.5}$, NO_2, acid vapor, and elemental carbon. In a study including schoolchildren, the relationship between bronchial hyperreactivity, IgE levels, and the prevalence of lower airway symptoms with levels of PM, SO_2, and NO_2 was examined [47]. In the study of Nicolai *et al*, increases in reported cough and wheezing symptoms and asthma prevalence were linked to traffic pollution [48].

More than 65 years have passed since the effect of air pollution on lung cancer was first hypothesized [49-51]; the evidence about this connection has been the subject of multiple reviews [52-56]. Despite controlling for key individual variables such as smoking and occupational exposure, cohort studies revealed a robust association between smoking and lung cancer [57-59]. Publications, such as the American Cancer Society cohort research, have finally confirmed initial fears [60]; each 10 g/m3 rise in $PM_{2,5}$ was associated with a significantly higher mortality risk ratio for lung cancer (RR=1.14, 95% CI: 1.04 to 1.23).

3.2. Health effects of pollutants on the Cardiovascular System

Cardiovascular disorders are among the world's leading causes of illness and mortality. There is mounting evidence that air pollution plays a significant role in triggering cardiovascular impairment [61-62]. Numerous air contaminants (such as O_3, SO_2, and NO_2) can individually or in combination cause cardiovascular disease, and PM is the most investigated [61]. Several studies have also linked long-term PM exposure to poor cardiovascular outcomes [63-67]. Patients with underlying cardiac or respiratory disease, socioeconomically disadvantaged groups, and diabetics may be at a higher risk

[61]. Furthermore, short-term increases in PM and other air pollutants are connected with daily cardiovascular morbidity and mortality [67-84].

Miller *et al*.'s [66] study conducted in postmenopausal women found that long-term exposure to PM2.5 increased the risk of cardiovascular disease and mortality. Each increase of 10 micrograms per cubic meter was linked with a 24% increase in the risk of a cardiovascular event (hazard ratio:1.24; 95%CI: 1.09-1.41) and a 76% increase in the risk of cardiovascular disease-related mortality (hazard ratio:1.76; 95% CI:1.25-2.47). The risk of cerebrovascular incidents was likewise linked to higher $PM_{2.5}$ levels (hazard ratio: 1.35; 95% CI: 1.08-1.68).

Short-term increases in the levels of certain ambient particles are associated with cardiac arrhythmias [77–79], worsening heart failure [80,81], and a rise in acute ischemic heart disorders [75, 76, 82-85]. The EMECAM study, which examined the short-term impact of air pollution on the death rate in 14 Spanish cities between 1990 and 1995, revealed a correlation between air pollution levels and hospital admissions for cardiovascular illnesses.

Despite great breakthroughs in surgical treatment, congenital cardiovascular abnormalities continue to be a serious public health concern, and they account for a significant fraction of the newborn death rate [86]. It is a cardiac abnormality that is primarily present at birth and can refer to a wide range of disorders affecting the heart. The incidence of congenital heart disease is the rate of new cases of congenital heart disease, which is typically represented as the number of infants born with congenital heart disease per one thousand live births. Variable estimates range from 4.1/1000 to 12.3/1000 for the incidence of congenital cardiac disease in infants [87].

Environmental factors, such as chemicals, medications, radiation, or infection (e.g., rubella), and genetic factors, have been identified as possible causes of congenital cardiac disease [61,88-94]. Ritz *et al*. [95] analyzed the impact of air pollution on the incidence of birth abnormalities identified by the California Birth Defects Monitoring Program in neonates and fetuses born in southern California between 1987 and 1993. The odds ratios (ORs) for cardiac ventricular septal abnormalities increased dose-dependently with increasing carbon monoxide exposure (OR 2^{nd} quartile 1.62 [95% CI 1.05–2.48], OR3rd quartile 2.09 [95% CI 1.19–3.67], and OR4th quartile 2.95 [95% CI 1.44–6.05]). Although aortic artery and valve anomalies, pulmonary artery and valve abnormalities, and conotruncal malformations increased with 2^{nd}-month ozone exposure, there was no connection between PM and other assessed air pollutants and these anomalies.

Even though the majority of research has focused on the consequences of short-term exposures, it has been proposed that long-term exposure may be more significant in terms of overall public health, and two prospective cohort studies imply the new trend for long-term exposure studies [96-102]. The first investigation, the 1993 Harvard Six Cities study by Dockery *et al*. [63], a cohort of 8111 people with 14 to 16 years of follow-up, revealed that the overall death rate ratio for the most polluted city relative to the least polluted city was 1.26. (95% CI: 1.08-1.47). Cardiovascular events were the leading cause of this. This study also demonstrated that chronic air pollution exposure is an independent cardiovascular mortality risk factor. $PM_{2.5}$ and sulfates exhibited the most significant association with the illnesses.

The second study, an ACS study by Pope *et al*. [64] with the largest study population in chronic air pollutants exposure studies, revealed that the adjusted relative risk ratios for all-cause mortality in the most polluted areas relative to the least polluted areas were 1.15 (95% CI: 1.09-1.22) and 1.17 (95% CI: 1.09-1.26) when using sulfate and fine particulate measures, respectively. After this research reported an association between air pollution and mortality from lung cancer and cardiovascular disease, numerous others verified this result [65]. Schikowski *et al*. [67] aimed to determine if baseline respiratory health contributes to the impact of long-term exposure to high levels of air pollution on cardiovascular mortality in a cohort of 4750 older women. They demonstrated that women with reduced lung function or preexisting respiratory disorders had an increased risk of cardiovascular-related death. The risk ratio (RR) of women with an FEV_1 of < 80%

anticipated to die from cardiovascular reasons was RR=3.79 (95%CI: 1.64-8.74) at 5 years survival time and RR=1.35 (95% CI: 0.66-2.74) after 12 years.

On the other hand, the short-term impacts of air pollutants on cardiopulmonary mortality were illustrated by showing the correlation between the increase in pollutants and the rise in daily mortality rates; the EMECAM project (Spanish multicentric study on the relationship between air pollution and mortality) has analyzed and reported the short-term link between air pollution levels and mortality in Spain by analyzing series from the early 1990s in13 Spanish cities [61-64].

4. Conclusion

Children, the elderly, pregnant women, and those from lower socioeconomic backgrounds are more susceptible to the impacts of air pollution, and a 10 μg/m3 rise in PM_{10} increases mortality by 0.4% to 0.8% in short-term exposures, according to recent studies. Moreover, outdoor air pollution produced by uneven urbanization and industrialization kills 3.2 million people year worldwide. Although there is substantial data about the impact of short-term exposure to PM_{10} on respiratory health, it is well established that $PM_{2.5}$ is a more significant risk factor for death, particularly when compared to PM_{10}'s potential for long-term exposure.

Air pollution can result in both immediate and chronic health effects, such as mild physiologic abnormalities and death, particularly from respiratory and cardiovascular maladies. Patients with chronic respiratory and cardiovascular diseases, as well as asthmatic children, are particularly vulnerable to the effects of chemical air pollutants. It is known that short-term exposure worsens chronic diseases and increases symptoms, medical care needs, and mortality, whereas long-term, repetitive exposure increases the cumulative risk of these diseases and death. The study recommends that exposure to air pollutants be reduced if possible, with the intervention of the right authorities.

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