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Cardiovascular Implications Due To Air Pollution: A Review Paper

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ABSTRACT: ***Particulate matter (PM) and gaseous pollutants like nitrogen dioxide and ozone make up air pollution. Coarse particles (PM10), fine particles (PM2.5), and ultrafine particles (PM100) are the three sizes of PM. We want to offer a unique overview of the scientific data from epidemiological and experimental research looking at the impact of outdoor air pollution on the cardiovascular system. According to a meta-analysis of epidemiological research, a 10g/m3 increase in long-term PM2.5 exposure was linked to an 11% increase in cardiovascular death. Long-term and short-term nitrogen dioxide exposure were both linked to an increase in cardiovascular mortality. As shown by early aortic and coronary calcification, exposure to air pollution and road traffic was linked to an elevated risk of arteriosclerosis. Myocardial infarction, stroke, and abrupt heart failure have all been linked to short-term increases in air pollution. Even when pollutant concentrations were below European limits, the danger was raised. Numerous experimental investigations have shown that air pollution induces a systemic vascular oxidative stress response, corroborating the findings from epidemiological studies. Endothelial dysfunction, monocyte activation, and certain proatherogenic alterations in lipoproteins are all caused by reactive oxygen species, which lead to plaque development. Furthermore, because of a rise in coagulation factors and platelet activation, air pollution promotes thrombus formation. Experiments have also shown that certain pollutants, such as combustion-derived PM2.5 and ultrafine particles, have greater detrimental cardiovascular effects. Cardiovascular disorders are exacerbated by air pollution. Promoting better air quality seems to be a new issue in the prevention of cardiovascular disease.***

KEYWORDS: ***Air Pollution, Cardiovascular Mortality, Myocardial Infarction, Oxidative Stress, Particulate Matter.***

1. INTRODUCTION

Air pollution is a significant public health problem that causes millions of deaths prematurely across the globe. Cardiovascular diseases account for 60–80 percent of deaths caused by air pollution [1]. We conducted a study of articles reporting on the cardiovascular consequences of air pollution in order to support the European Society of Cardiology's campaign, which seeks to increase awareness of the harmful impact that the environment may have on the heart. We focused our study on indoor and outdoor air pollution since both indoor and outdoor air pollution have a significant effect on cardiovascular illnesses. We start with some definitions and sources of air pollution, which are necessary for presenting evidence from long and short-term epidemiological studies. Following that, we provide some pathophysiology data and offer a schematic summary of the molecular pathways that connect air pollution exposure to clinical outcomes. We also provide current statistics on the benefits of mitigation strategies, as well as potential future possibilities for overcoming environmental research constraints.

1.1. Air Pollution Basics:

Particulate Matter (PM) and gaseous components make up air pollution. Based on their dimension, PM is categorized as coarse particles, fine particles, ultrafine particles, or nanoparticles [2]. Depending on the source, PM has a varied makeup. Carbonaceous particles come from combustion sources like traffic emissions or home heating, while inorganic particles come from sources like desert dust and agricultural mineral dust. Carbonaceous

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particles are carbon-based, but they also include organic compounds like polycyclic aromatic hydrocarbons and reactive metals on their surfaces. Nitrogen oxides, such as nitrogen dioxide (NO_2) and nitric oxide (NO), ozone, sulphur dioxide (SO_2), volatile organic compounds, and carbon monoxide are examples of gaseous pollutants (CO). SO_2 and nitrogen oxides contribute to particle production via complicated atmospheric photochemical processes including ammonia from agriculture, in addition to their inherent toxicity. Secondary particles are formed as a consequence of gaseous transition and are mostly comprised of inorganic chemicals such as ammonia, sulphates, and nitrates. Ozone is a secondary gaseous pollutant that results from a photochemical interaction between sunlight and gaseous precursors like nitrogen oxides or volatile organic compounds.

1.1.1. Indoor Air Pollution:

In 2010, air pollution caused 7 million premature deaths globally, with both indoor and outdoor air pollution playing a role. Indoor air pollution, on the other hand, is more diverse, with significant differences in contaminants and sources between nations [3]. Secondhand smoking is a significant cause of indoor air pollution all around the world. In Asia, solid-fuel cooking and heating are the primary causes of interior air pollution, while in Europe, a variety of sources contribute to indoor pollution, including volatile organic compounds from organic solvents, household goods, and particulates from cooking and wood burning. Nonetheless, a recent European research found that outdoor PM2.5 entering inside through air exchange is responsible for 60% of the worldwide burden of indoor air pollution-related illness. As a result, reducing the illness burden caused by interior air pollution necessitates changes to indoor air pollution sources, ventilation, and outdoor air filtering.

1.1.2. Outdoor Air Pollution:

Agriculture is a significant generator of PM2.5 in Europe. Agricultural particles, on the other hand, are mostly inorganic particles, which are generally less hazardous than carbonaceous particles from combustion sources like road traffic [4]. In Europe, road traffic and home heating have the greatest effect on outdoor air pollution-related mortality, assuming this difference in toxicity. In North America, industry and fossil-fuel-based electricity production are also significant producers of PM. Natural causes such as desert dust and biomass burning contribute significantly to ambient air pollution levels in Africa. In Asia, the major sources of particles in both outdoor and indoor emissions are home heating and cooking. Aside from variations across nations and continents, there are significant disparities among the major sources of pollutants within a single country, depending on local sources. Road traffic in big cities is a significant source of worldwide pollution emissions, as well as the primary source of NO_2, which comes mostly from diesel cars. Road traffic, for example, contributes for 30% of PM emissions in Paris, almost as much as the residential sector, and almost 60% of nitrogen oxide emissions. SO_2 is mostly produced by industrial emissions and marine transport, although its contribution to air pollution has been decreasing over time.

2. DISCUSSION

2.1. Epidemiological Evidence:

Long-term exposure research looked at the cardiovascular consequences of air pollution following yearly changes in pollutant concentrations, while short-term exposure studies mostly looked at daily or hourly changes in pollutant concentrations. The majority of the research inferred the amount of person exposure to air pollution based on background pollutant concentrations or traffic exposure at the residence.

2.1.1. Cardiovascular Mortality:

- *Short-Term Exposure:*

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In a recent meta-analysis, a daily increase in PM2.5 exposure of 10g/m3 was linked to a 0.84 percent increase in cardiovascular mortality. A research that looked at short-term changes in NO_2 found that a 10g/m3 daily rise in NO_2 resulted in a 0.4—0.88% increase in cardiovascular mortality. The effects of short-term ozone fluctuations on cardiovascular mortality are still being studied.

- *Long-Term Exposure:*

According to a 2013 meta-analysis, a 10g/m3 increase in yearly PM2.5 concentration was linked with an 11 percent increase in cardiovascular mortality [5]. Coronary artery disease-related mortality had the greatest correlations, which remained after controlling for cardiovascular variables and socioeconomic level. Among other research, however, PM2.5 harmful effects were shown to be greater in those with the lowest education, which was likely due to a lack of antioxidants due to a lack of fruit consumption. When compared to coarse particles, fine and ultrafine particles had the greatest effect on cardiovascular mortality. Furthermore, the composition of PM is an essential factor to consider, with some research indicating that carbonaceous particles from combustion-derived sources, such as road traffic, fossil fuels, and wood burning, have a greater cardiovascular toxicity. NO_2 is mostly produced by combustion sources. According to a meta-analysis of the cardiovascular consequences of long-term NO_2 exposure, a 10g/m3 rise in yearly NO_2 concentrations resulted with a 13 percent increase in cardiovascular mortality. Ozone's impact seems to be less significant, with some long-term exposure studies indicating a modest increase in cardiopulmonary causes of mortality, although this was only seen during the warm season and not in a yearlong study. This may be explained by the fact that, unlike NO_2, ozone pollution is more common on hot, sunny days because it needs photochemical interactions with sunlight to produce. Long-term SO_2 exposure has been linked to a higher risk of respiratory death, but its effect on cardiovascular death is unknown.

Long-term exposure to road traffic was significantly related to cardiovascular mortality, regardless of air pollutant background concentrations. Living within 50 meters of a major highway raised the risk of sudden cardiac death by 38% compared to living more than 500 meters away in a large-scale prospective study of women, and this impact persisted after controlling for possible confounders and cardiovascular risk factors.

2.1.2. Coronary Artery Disease:

- *Short-Term Exposure:*

Acute coronary syndromes have been demonstrated to be triggered by a short-term increase in air pollution, with the greatest correlations seen with contemporaneous day or last two days' mean air pollutant exposure. Exposure to air pollution and road traffic were the most significant variables at the population level in a meta-analysis of non-fatal myocardial infarction triggers, given that a high number of individuals are exposed [6]. In terms of the risk of ST-segment elevation myocardial infarction, a recent research found that each 10g/m3 rise in PM2.5 in the 24 hours leading up to the event was linked with a 2.8 percent increase in risk, while a comparable increase in NO_2 was associated with a 5.1 percent increase in risk. The elderly seemed to be at higher risk from PM, whereas younger patients tended to be more vulnerable to NO_2 exposure. In middle-aged individuals without prior cardiovascular illness, a short-term rise in ozone was similarly linked to acute coronary events.

- *Long-Term Exposure:*

Several studies have shown a significant link between long-term air pollution exposure and acute myocardial infarction. Annual increases of 10g/m3 in PM10 and 5g/m3 in PM2.5 were linked to higher risks of myocardial infarction of 12 percent and 13 percent, respectively, in a

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large-scale prospective European research. Above importantly, these beneficial correlations were found despite air pollution concentrations being below current European policy recommendations. Long-term exposure to PM2.5 has been linked to the development of early arteriosclerosis in many studies. Exposure to PM2.5 and traffic-related air pollution were shown to be substantially linked to an elevated coronary artery calcium score in a prospective cohort study. A similar finding was found in the case of road traffic exposure, with higher chances of a high coronary artery calcium score of 63 percent and 34 percent for individuals living within 50 meters and 50—100 meters of a major road, respectively, compared to people living 200 meters away.

2.1.3. Other Cardiovascular Outcomes:

- *Heart Failure:*

A meta-analysis found a link between short-term increases in PM and gaseous components (NO_2, SO_2, CO) with an increased risk of congestive heart failure hospitalization or death, with the greatest correlations occurring on the day of exposure and more lasting effects for PM2.5 [7].

- *Stroke:*

Air pollution contributes to 29 percent of the burden of stroke, according to data from the Global Burden of Diseases 2013, which was gathered in 188 countries between 1990 and 2013 [8]. Several studies have shown favorable links between long- and short-term air pollution exposure and stroke incidence and death. In a prospective European research, a 5g/m3 rise in yearly PM2.5 was linked to a 19% higher risk of stroke, with the greatest correlations observed among individuals who had never smoked. At pollution levels below current European air quality regulations, the risk of myocardial infarction was nevertheless elevated, as previously reported. Road traffic exposure significantly raised the risk of stroke, with substantial links to ischemic stroke seen in those who lived within 75 meters of a major road. Changes in pollutant concentrations over time were also linked to an increased risk of stroke and stroke death, with ultrafine particles having a particularly significant link to stroke mortality. Increases in gaseous chemicals such as NO_2, SO_2, and CO enhanced the risk of stroke in the short term. In the case of ozone, some studies found a link between a short-term rise in ozone and an increased risk of stroke, while others found no link.

2.2. Pathophysiological Evidence:

The findings of many animal and human interventional investigations are combined in environmental research on air pollution. In humans, these investigations are primarily randomized double-blind crossover experiments carried out in specialized chambers, allowing for complete uniformity of air pollutant exposure [9]. Healthy volunteers, as well as patients with stable coronary artery disease, are typically exposed to regulated quantities of dilute diesel exhaust or filtered air while exercising on a bicycle ergometer in these exposure chambers. The exposure time is usually restricted to one or two hours, and the PM concentrations obtained are similar to those seen during a severe air pollution event. These investigations support the evidence of air pollution-mediated cardiovascular damage, despite the limitations of epidemiological research previously discussed.

2.2.1. From Oxidative Stress to Endothelial Dysfunction:

Numerous studies have shown that air pollution causes a severe oxidative stress response in the lungs, which is triggered by PM entering the lungs. However, by stimulating several enzymatic pathways, this lung oxidative response is increased, eventually leading to a systemic vascular oxidative stress reaction. A recent research found that individuals who

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were exposed to diesel exhaust had an acquired oxidative capacity in their blood. Superoxide anion generation was found after incubating endothelial cell cultures with blood from individuals who voluntarily exposed themselves to air pollution, with a dose-response pattern closely proportional to the quantity of inhaled PM2.5. In vitro experiments showed that superoxide dismutase reverses the negative vascular impact caused by diesel exhaust exposure, indicating that ROS generation plays a significant role. Several studies have shown that the surface chemicals covering diesel particles, such as transition metals, polycyclic aromatic hydrocarbons, and quinones, are mostly responsible for the oxidative stress response. Although particles are the primary cause of oxidative stress, gaseous molecules such as NO_2 play a role in ROS production as peroxynitrite.

2.2.2. From Endothelial Dysfunction to Atherogenesis:

Air pollution causes oxidative stress, which is followed by changes in circulating lipids, in addition to endothelial dysfunction. Pollution encourages the formation of oxidized low-density lipoproteins and the release of additional highly oxidized phospholipids. These proatherogenic chemicals diffuse into the subendothelial region, causing endothelial cells to become more activated. The release of proinflammatory adhesion molecules such as vascular cell adhesion molecule-1 and monocyte chemotactic protein-1, which ensure monocyte recruitment and differentiation into macrophages in the subendothelial space, characterizes endothelial cell activation following air pollution exposure. Furthermore, air pollution has been linked to the depletion of anti-atherogenic substances such high-density lipoprotein. In an animal research, ultrafine particles were shown to have a greater impact than PM2.5 on high-density lipoprotein antioxidant capacity. As a result, air pollution increases vascular inflammation, foam cell lipid accumulation, and plaque development.

2.2.3. From Atherogenesis to Atherothrombosis:

Platelet activation and enhanced thrombus formation have been seen in healthy individuals exposed to diesel exhaust. Similar results were shown in individuals with coronary artery disease who were exposed to diesel exhaust, as well as a suppression of endogenous fibrinolytic ability, as seen by a decrease in acute tissue activator plasminogen release [10]. Increased endothelial cell death, a lower circulating number of endothelial progenitor cells, and tight junction protein degradation are all signs of air pollution-induced endothelial damage. Increased fibrinogen, factor VIII, and tissue factor release are caused by an elevation in interleukin-6 following PM inhalation. Platelet activation is also triggered by interleukin-6 and other proinflammatory chemicals. Air pollution creates all of the prerequisites for thrombus formation, including a damaged endothelial cell barrier, an increase in coagulation factors, a decrease in fibrinolytic ability, and platelet activation.

3. CONCLUSION

According to epidemiological research, air pollution raises long- and short-term cardiovascular mortality through increasing myocardial infarction, stroke, and heart failure occurrences. Despite the fact that cardiovascular risk rises with exposure amount and time, all investigations found that there is no safe threshold beyond which no impact occurs. Interventional controlled research may help explain the processes at work. Air pollution, especially diesel exhaust, causes a significant increase in reactive oxygen species production, impairing nitric oxide-mediated vasodilation and promoting vascular inflammation. Air pollution has been shown to have acute functional effects in myocardial and pulmonary blood flow control, as well as coagulation function. Because of their tiny size and the polycyclic aromatic hydrocarbons and metals that they carry on their surface, combustion-derived particles have significant negative impacts. While ultrafine particles have a significant

cardiovascular impact, current standards and tests greatly underestimate their amounts in ambient air. Furthermore, transportation is a major source of NO_2, which has recently been linked to an increased risk of ST-segment elevation myocardial infarction in metropolitan settings. The existing scientific data supports the proposal that efforts to decrease exposure to air pollution should be immediately increased and backed by suitable and effective legislation, as recently approved by the European Society of Cardiology.

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