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Air pollution and cardiovascular diseases

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Abstract

This comprehensive literature review explores the intricate relationship between air pollution and cardiovascular diseases, emphasizing the global significance of this environmental concern. The study delves into key air pollutants, including Particulate Matter (PM), Nitrogen Dioxide (NO2), Ozone (O3), and Sulfur Dioxide (SO2), revealing their associations with cardiovascular morbidity and mortality. The detrimental effects involve systemic inflammation, oxidative stress, endothelial dysfunction, and atherosclerosis.

The review meticulously analyzes epidemiological studies, such as the UK Biobank study, linking long-term exposure to ambient air pollution, specifically PM2.5, PM10, NO2, and NOx, with increased risks of all-cause and cardiovascular mortality. Specific populations, characterized as susceptible or vulnerable, are identified, including adults over 60 and individuals with pre-existing health conditions.

Studies examining genetic variations, blood pressure changes, and meteorological variables contribute to understanding individual susceptibility. The review highlights the role of air pollution in influencing sleep patterns, physical activity, and temperature-related cardiovascular strain. Furthermore, it emphasizes the importance of recognizing air pollution as a modifiable risk factor, providing evidence of its association with cardiovascular risk factors and Dyslipidemia.

The impact of air pollution on diverse cardiovascular outcomes, including heart failure, stroke, and ischemic heart disease, is discussed. The review underscores the urgent need for public health interventions and policy recommendations to mitigate air pollution's adverse effects. Recognizing air pollution as a significant risk factor for cardiovascular diseases, the study advocates for proactive measures to reduce pollution, promoting cardiovascular health and creating a cleaner environment for future generations.

Keywords: PM: Particulate matter; PM10: Particulate matter with a diameter <10 µm; PM2.5: Particulate matter with a diameter <2.5 µm; SO_2: Sulphur dioxide CO: Carbon monoxide CO_2: Carbon dioxide; NO: Nitric oxideO3:Ozone; NO_2: Nitrogen dioxide; CVD: Cardiovascular Disease; sLOX-1: (soluble lectin-like oxidized low-density lipoprotein receptor-1) is a biomarker that refers to the soluble form of LOX-1, a receptor involved in the uptake of oxidized low-density lipoprotein (LDL) particles by endothelial cells; ER: Emergency Room (of Hospital)

1. Introduction

Air pollution is a significant environmental concern worldwide, with detrimental effects on human health. It has been linked to various adverse health outcomes, including respiratory illnesses, cancer, and cardiovascular diseases (CVD). Among these, the association between air pollution and cardiovascular diseases has gained considerable attention in recent years. Cardiovascular diseases, such as heart attacks, strokes, and heart failure, are leading causes of morbidity

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and mortality globally. Understanding the impact of air pollution on cardiovascular health is crucial for developing effective preventive measures and public health policies. Air pollution is a global issue that poses significant risks to human health, with its detrimental effects extending beyond respiratory ailments. Some major air pollutants are:

1.1. Particulate Matter (PM)

Particulate matter, consisting of fine and ultrafine particles suspended in the air, is a major component of air pollution. Numerous studies have demonstrated a robust link between PM exposure and increased cardiovascular morbidity and mortality rates. PM can penetrate deep into the respiratory system, entering the blood-stream and causing systemic inflammation, oxidative stress, endothelial dysfunction, and atherosclerosis. Particular attention has been given to PM2.5 (particles with a diameter of 2.5 micrometers or smaller), which has shown the strongest association with CVDs.

1.2. Nitrogen Dioxide (NO_2)

NO_2, primarily emitted from vehicles and industrial sources, is a key marker of traffic-related air pollution. Several epidemiological studies have consistently associated NO_2 exposure with adverse cardiovascular outcomes, including hypertension, myocardial infarction, and stroke. NO^2 can induce oxidative stress, inflammation, and endothelial dysfunction, contributing to the development of atherosclerosis and plaque instability.

1.3. Ozone (O_3)

Ground-level ozone is formed through chemical reactions involving volatile organic compounds (VOCs) and nitrogen oxides (NOx) in the presence of sunlight. While ozone is a vital component of the Earth's upper atmosphere, high concentrations at ground level have detrimental effects on human health. Long-term exposure to O_3 has been linked to increased cardiovascular mortality, arrhythmias, and ischemic heart diseases. O^3 induces oxidative stress, inflammation, and alters autonomic function, leading to adverse cardiovascular events.

1.4. Sulfur Dioxide (SO_2)

SO_2, primarily emitted from fossil fuel combustion, is associated with industrial activities and power generation. Prolonged exposure to SO_2 has been shown to increase the risk of cardiovascular events, including heart failure and arrhythmias. SO_2 can trigger endothelial dysfunction, oxidative stress, and systemic inflammation, all of which contribute to the development of CVDs.

Cardiovascular diseases pose a significant burden on individuals, families, and healthcare systems. It is well-established that traditional risk factors, such as smoking, unhealthy diet, sedentary lifestyle, and obesity, contribute to the development of cardiovascular diseases. However, emerging evidence suggests that air pollution, particularly fine particulate matter (PM2.5) and ambient air pollutants, play a substantial role in increasing the risk of cardiovascular diseases. Air pollution is a complex mixture of particulate matter (PM), nitrogen dioxide (NO_2), ozone (O_3), sulfur dioxide (SO_2), and volatile organic compounds (VOCs) emitted from various sources, including industrial activities, vehicle emissions, and power generation. These pollutants have been extensively studied for their detrimental effects on respiratory health. However, growing evidence suggests that exposure to air pollution also plays a significant role in the development and progression of cardiovascular diseases (CVDs).

Numerous epidemiological studies have demonstrated a strong association between air pollution exposure and increased cardiovascular morbidity and mortality. For instance, a large-scale study conducted by Brook et al. (2010) found that short-term exposure to PM2.5 (particulate matter with a diameter of 2.5 micrometers or less) was associated with an increased risk of myocardial infarction. (R. D. Brook, et al. , 2010) Similarly, a systematic review reported that long-term exposure to PM2.5 was significantly associated with an elevated risk of stroke.(Mustafic, 2012)

Several epidemiological studies have investigated the relationship between air pollution and cardiovascular diseases, providing compelling evidence of their association. These studies have observed higher rates of cardiovascular events and mortality in areas with higher levels of air pollution. The mechanisms through which air pollution affects cardiovascular health are complex and multifaceted, involving systemic inflammation, oxidative stress, endothelial dysfunction, autonomic nervous system imbalance, and accelerated atherosclerosis.

While the adverse effects of air pollution on respiratory health have long been recognized, the impact on cardiovascular health has garnered increasing attention. The World Health Organization (WHO) estimates that air pollution contributes to millions of premature deaths annually, with a significant proportion attributed to cardiovascular diseases. This has led to heightened awareness among researchers, healthcare professionals, and policymakers regarding the need to address air pollution as a public health concern.

This literature review aims to provide a comprehensive analysis of the existing research on the relationship between air pollution and cardiovascular diseases. It will explore the various types of air pollutants implicated in cardiovascular health effects, the underlying mechanisms involved, and the epidemiological evidence supporting the association. Additionally, it will discuss the implications for public health interventions, policy recommendations, and future research directions in this field.

By understanding the impact of air pollution on cardiovascular diseases, we can develop strategies to mitigate exposure, implement preventive measures, and advocate for cleaner air policies. Such efforts are essential for promoting cardiovascular health and reducing the burden of cardiovascular diseases on individuals and societies.

1.5. Inclusion/Exclusion Criteria

Inclusion/Exclusion Criteria for the literature review entitled "Air Pollution and Cardiovascular Diseases" are as follows

1.5.1. Inclusion Criteria

- Studies published in prestigious journals within the last 15 years (2000-2023), at least one third articles published within last 5 years.
- Studies focusing on the association between air pollution and cardiovascular diseases.
- Research articles that investigate the effects of various types of air pollutants on cardiovascular health outcomes.
- Studies examining the mechanisms and pathways through which air pollution influences cardiovascular diseases.
- Epidemiological studies investigating the relationship between air pollution and cardiovascular events, mortality, or prevalence.
- Studies that provide quantitative data, such as effect sizes, odds ratios, relative risks, or hazard ratios.

1.5.2. Exclusion Criteria

- Studies examining indoor air pollution without a specific focus on outdoor air pollution or ambient air pollutants.
- Studies not available in English or without full-text accessibility.
- Studies before 2000 AD.

2. Search Strategy

- Identified the main keywords and phrases related to the research topic.
- Combined the keywords using Boolean operators (AND, OR) to create search queries: ("Air pollution" OR "Outdoor air pollution" OR "Ambient air pollutants") AND ("Cardiovascular diseases" OR "Heart diseases" OR "Stroke" OR "Hypertension" OR "Atherosclerosis")
- Determined the appropriate databases to search:
  - Pub Med
  - Web of Science
  - Google Scholar
- Applied the search query to each selected database, using the advanced search options when available.
- Set the time frame for the search, typically within the last 10 years (2013-2023), butas per the specific requirements of the literature review I took the 1/3rd of articles published within last 5 years.
- Screened the search results based on the inclusion/exclusion criteria, reviewing titles and abstracts to determine relevance.
- Retrieved full-text articles for the relevant studies and review them thoroughly.
- Utilized the reference lists of selected articles to identify additional relevant studies (backward citation searching).

3. Synthesis of findings/ Review

Major sources of air pollution include the emission of pollutants from power stations, refineries, and petrochemicals, the chemical and fertilizer industries, metallurgical and other industrial plants, and, finally, municipal incineration. Indoor area sources include domestic cleaning activities, dry cleaners, printing shops, and petrol stations. Mobile sources entail automobiles, cars, railways, airways, and other types of vehicles. Finally, natural sources include physical

disasters (Stover, 1979) such as forest fire, volcanic erosion, dust storms, and agricultural burning. However, many other classification systems have been proposed. Another type of classification is a grouping according to the recipient of the pollution, as follows: Air pollution is determined as the presence of pollutants in the air in large quantities for long periods. Air pollutants are dispersed particles, hydrocarbons, CO, CO_2, NO, NO_2, SO_3, etc.

Particulate matter (PM) is formed in the atmosphere as a consequence of chemical reactions between the different pollutants. The penetration of particles seems to be closely dependent on their size. Particulate Matter (PM) was defined as a term for particles by the United States Environmental Protection Agency. Particulate matter (PM) pollution includes particles with diameters of 10 micrometers (μm) or smaller, which is called PM10, and extremely fine particles with diameters that are generally 2.5 micrometers (μm) and smaller. Particulate matter consists of tiny liquid or solid droplets that could be inhaled and lead to serious health effects (Agency), 2018).

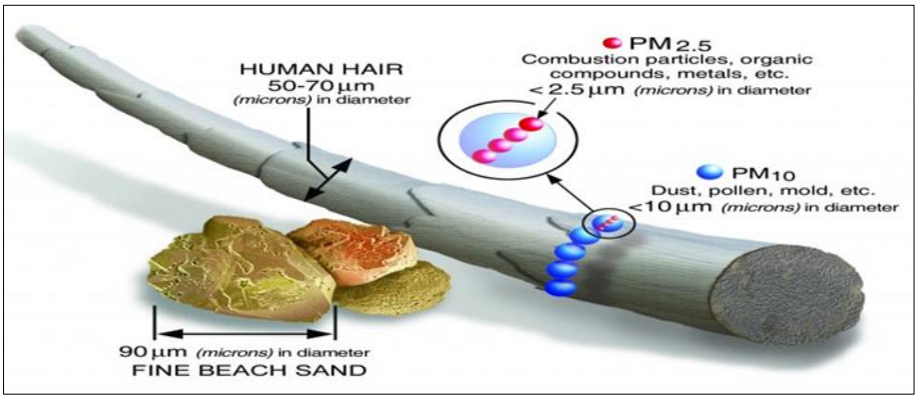

Figure 1 Sizes of Particulate matter

A prospective UK Biobank study conducted in UK found that long-term exposure to ambient air pollution might be associated with the risk of all-cause, CVD and CHD mortality. The study identified 11881 deaths [2426 from cardiovascular diseases (CVD), 1211 from coronary heart disease (CHD) and 466 from stroke] during a median follow-up of 8.9 years. It found that PM2.5 [hazard ratio (HR), 1.27; 95% CI, 1.05–1.55], PM10 (HR, 1.18; 95% CI, 1.04–1.34), NO_2 (HR, 1.05; 95% CI, 1.01–1.08), and NOx (HR, 1.02; 95% CI, 1.01– 1.03) were associated with all-cause mortality. PM2.5 was found mainly associated with increased risks of CVD (Wang et al., 2022).

Air pollution is an important factor threatening human health. Inhalation of pollutants can cause damage to the cardiovascular system, leading to increased morbidity and mortality of cardiovascular diseases. One research done in Shanghai, China concluded that PM2.5 and PM10 stimulate the respiratory tract, aggravate asthma, increase the heart rate, reduce lung function, and so on, and tend to affect the respiratory system and cardiovascular system adversely and in severe cases will cause premature death of heart disease patients.(Lu & Kang, 2022)

It is significant to note that specific populations are at higher risk from air pollution. These populations can be categorized as susceptible, vulnerable, or both. Susceptible groups are those at higher risk of cardiovascular events for a given level of pollution exposure. Majority of cohort studies demonstrate that more susceptible populations include adults over 60, socio-economically disadvantaged and minority groups, and individuals with obesity, diabetes, hypertension, pulmonary disease, or atherosclerotic cardiovascular disease. (Rajagopalan et al., 2020). On other hand, vulnerable individuals are those exposed to elevated levels of air pollution. Important factors entail living in areas of high pollution and close to any of the following: urban industrial emissions, heavy traffic, wildfires, seasonal agricultural burning, or burning of solid fuels for cooking or heating.(Brauer, Casadei, Harrington, Kovacs, & Sliwa, 2021)

Cardiovascular morbidities have been estimated to be responsible for more than two thirds of the considerable mortality attributed to air pollution. There is now a substantial foundation of research demonstrating that exposure to air pollution has many detrimental effects over the cardiovascular system. Multiple biological mechanisms are responsible; though, oxidative stress is known as a prominent observation at many levels of the cardiovascular impairment induced by pollutant exposure. Oxidative stress contributes to the mechanisms by which inhaled PM induces cardiovascular dysfunction. A complex series of interconnecting mechanisms induces the effects of inhaled PM on cardiovascular morbidity and mortality.(M. R. Miller, 2020)

A cohort study conducted in Californian teachers in 2011 provided the evidence linking long-term exposure to $PM_{2.5}$ and PM_{10} with increased risks of incident stroke as well as ischemic heart disease mortality; exposure to nitrogen oxides was also related to death from cardiovascular diseases. The study was done based on the long-term residential air pollution exposure over more than 100,000 participants in the California; a prospective cohort of female public school professionals was taken to conclude the finding.(Lipsett et al., 2011)

A 2013 meta-analysis found that an average increase of 11% in cardiovascular mortality was pertaining to 10 μg/m3 increase in annual PM2.5 concentration (Hoek et al., 2013). The strongest associations were witnessed for coronary artery disease associated mortality (K. A. Miller et al., 2007), and were persistent even after adjustment for cardiovascular issues and socioeconomic conditions. However, PM2.5 adverse effects were relatively higher for individuals with the lowest educational status in some studies, probably related to low antioxidant intake because of low fruit intake. Fine and ultrafine particles found to have the most important impact on cardiovascular mortality, compared with coarse particles. Moreover, the composition of PM is an significant issue to consider, with some findings showing increasing cardiovascular toxicity of carbonaceous particles from combustion-derived sources, such as road traffic, fossil fuels and wood burning (Laden, Neas, Dockery, & Schwartz, 2000). Combustion sources are also found to be the leading source of NO_2. A meta-analysis of cardiovascular effects of long-term exposure to NO_2has reported a 13% increment in cardiovascular mortality after a 10 μg/m3 increase in annual NO_2concentrations (Zeng et al., 2023). The effect of ozone seems to be less pronounced, with some long-term exposure studies indicating a small increase in cardiopulmonary causes of death, but this was only observed during the warm season, and not in annual based analysis. This can be explained by the fact that, unlike NO_2, ozone pollution typically occurs during warm and sunny days, because its formation requires photochemical reactions involving sunlight. Long-term exposure to SO_2 has been strongly associated with respiratory mortality, although its impact on cardiovascular mortality remains unclear (Atkinson et al., 2016). Constant air pollutant background concentration, long-term exposure to road traffic was robustly linked to cardiovascular mortality (Hoek, Brunekreef, Goldbohm, Fischer, & van den Brandt, 2002). In a large-scale prospective study in women, living within 50 meter of a major roadway showed to increase the risk of sudden cardiac death by 38% compared with living ≥ 500 m away, and this consequence remained, even after adjusting for potential confounders and cardiovascular risk factors (Hart, Chiuve, Laden, & Albert, 2014).

Individual susceptibility to the cardiovascular effects of air pollution has been recognized as an important factor. Recent studies have investigated the influence of genetic variations and co-morbidities on susceptibility. For example, a study explored the interaction between air pollution and genetic polymorphisms related to inflammation pathways and found that individuals with specific genetic variants were more susceptible to the adverse cardiovascular effects of air pollution (Ward-Caviness, 2019).

Abundant studies assessing the relationship between air pollution and cardiovascular disease have also scrutinized the changes in blood pressure. It is well known fact that both air pollution and increased blood pressure contribute to an elevated risk of cardiovascular disease. The study strongly showed that particle pollution was associated with a remarkable rise in blood pressure. These studies revealed that for every 10.5 μg/ m3 increase in PM2.5 levels, there was a 2.8 mmHg increase in systolic blood pressure (SBP) and 2.7 mmHg increase in diastolic blood pressure (DBP) among patients over five days in Boston. Some other identical studies also showed that increases in SBP (5.2 mmHg) were observed with increased PM2.5 levels in Detroit. As per these studies, there was a close correlation between increased blood pressure and PM (R. D. Brook, 2007). A study of 23 normotensive patients showed significant rises Diastolic BP (6 mmHg) after a two hour exposure to PM2.5 and O3 (Urch et al., 2005). According to this study, carbon content in air pollution PM was highly associated with the rise in blood pressure. Furthermore, increases in SBP (2.6 mmHg) and Diastolic BP (2.4 mmHg) are significantly influenced by Carbon monoxide levels (de Paula Santos et al., 2005). Notably, air pollution doubled the risk of obesity, hypertension, chronic pulmonary disease, and cardiovascular disease in geriatric population (Dubowsky, Suh, Schwartz, Coull, & Gold, 2006; Metzger et al., 2004).Air pollution and blood pressure have been seen directly associated. A rapid and a statistically significant increase of diastolic BP among individuals exposed to ambient fine particles and O_3 has been seen in the studies.(Urch et al., 2005)

The study conducted in Beijing, Chinaamong 8377 hospital ER visits of CVD to explore the association between $PM_{2.5}$ and the hospital ER visits for CVD revealed that after adjusting the temperature and the relative humidity, the associations for 10 $\mu g/m^3$ increases in levels of $PM_{2.5}$, SO_2, or NO_2 and hospital ER visits for cardiovascular diseases were statistically significant with odds ratios (ORs) of 1.005{95% confidence interval suggesting that elevated levels of ambient air pollutants are associated with the increase in hospital ER visits for CVD in Beijing, China.(Guo, Jia, Pan, Liu, & Wichmann, 2009). Similar study conducted in Bangladesh among ER patients revealed that increases in the daily concentrations of $PM_{2.5}$ may lead to more cardiovascular emergency room visits in Dhaka, Bangladesh. However, response times from ambient exposure to CVD emergency visits may differ by season and the nutritional status of susceptible individuals, necessitating further research.(Khan et al., 2019)

Cardiovascular disease (CVD) has been associated with meteorological variables such as temperature, relative humidity, and wind along with pollutant levels. Study shows that meteorological variables play an important role in the causation and aggravation of CVDs. The air pollutants, PM_{10} and SO_2, except ozone, presented positive loadings with CVD.(Gonçalves, Braun, Silva Dias, & Sharovsky, 2007)

Heart failure (HF) is the sole largest cause of increased hospitalization after fine particulate matter (PM2.5) exposure. Patients with left heart failure often progress to right ventricular (RV) failure even with optimal medical care. An increase of PM2.5 of 10 μg per cubic meter is associated with a 76% increase in the risk of death from cardiovascular disease over 4 years' period.

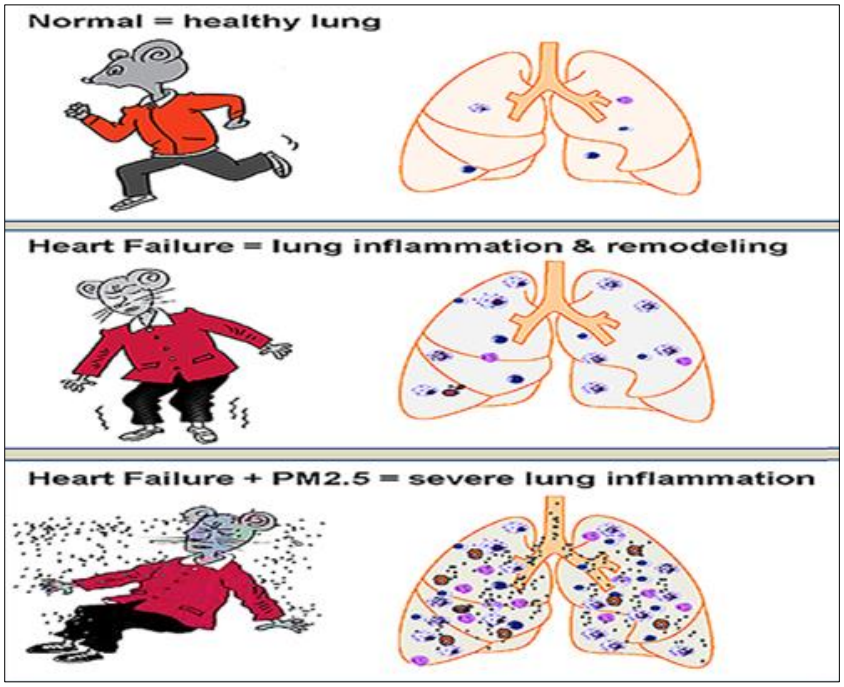

Figure 2 Association of PM and Heart Failure in Lung Pathology

The findings notify that a short and moderate PM2.5 exposure is enough to cause multiplicative lung inflammation, vascular remodeling and fibrosis, as well as significant RV hypertrophy in the mice with existing LV failure.(Yue et al., 2019).

One study found that increments of $PM_{2.5}$ levels seem to be associated with stroke morbidity and stroke mortality. It might be due to alterations in the ANS pathway and oxidative stress. Many in vitro and in vivo studies confirmed that exposure to PM can induce neuronal loss leading to cerebral ischemia or influence oxidative stress which may further disrupt the blood–brain barrier and increasing the risk of stroke. However, composite CVD events, CVD mortalities, and all-cause mortalities were associated with NO_2 and $PM_{2.5}$. Exposure to NO_2 or $PM_{2.5}$ could stimulate multiple primary and secondary pathways influencing inflammation, oxidative stress, autonomic nervous imbalance, and hypothalamic and pituitary, endothelial, and thrombotic functions. Through activation of these pathways, multiple studies showed that exposure to air pollution could lead to arterial stiffness, atherosclerosis, and heart rate variability that result in CVD outcomes (Hemmingsen et al., 2015). Higher PM_{10} and O_3 exposures were reported to be associated with HF and HF mortality, respectively. Although limited evidence, studies have reported even short-term inhalation of O_3 and PM can affect vasoconstriction, arterial hypertension, heart rate, and arrhythmias that can elevate the risk of HF and other CVDs, especially in at-risk individuals (Dwivedi, Vishwakarma, Dubey, & Reddy, 2022)

Ambient air pollution is clearly a modifiable risk factor for cardiovascular diseases, still uncertainty remains about the size of risks at lower levels of fine particulate matter (PM2.5) exposure which now occur in the USA and elsewhere. Long-term exposure to fine particulate air pollution remains associated with ischemic heart disease/heart attack and stroke death, with an excess risks occurring in the range of and below the present US long-term standard for ambient exposure to PM2.5 (12 mu g/m(3)), strongly indicating the need for continued improvements in air pollution for CVD prevention.(Hayes et al., 2020)

Some studies also have revealed indirect relationship with air pollution to CVDs since air pollution is responsible for climate change which is detrimental to CVDs in following ways (Münzel et al., 2021):

- The higher the temperature the higher the surface blood circulation and sweating, all of which contributes to higher cardiac strain, blood viscosity, plasma cholesterol, and interleukin-6 levels.
- Warmer temperature is responsible for sleep disturbance such as too short sleep (<6 h) or fragmentation of sleep, which inversely increases the risk of CVD.
- Very high temperature reduces physical activity, which is inversely associated with higher cardiovascular risk. Vice versa, physical activity at very high temperature may represent a risk factor of its own.

A prospective cohort study aiming to examine the progression of subclinical and clinical cardiovascular disease in an elderly population initially free of clinical CVD conducted in six U.S. communities, including Baltimore, MD, Chicago found that short-term increases in $PM_{2.5}$ concentrations were associated with elevated blood concentrations of sLOX-1 without increment in blood nitrite.In the light of its known mechanistic role in promoting vascular disease, sLOX-1 may be a suitable translational biomarker linking air pollutant exposures and cardiovascular outcomes.(Chen et al., 2015; Ni et al., 2021) As we know, elevated levels of sLOX-1 have been found in individuals with various cardiovascular conditions, including acute coronary syndrome, myocardial infarction (heart attack), and heart failure. Studies suggest that sLOX-1 may serve as a useful biomarker for assessing the severity of cardiovascular disease and predicting future cardiac events.

The population-based cross-sectional study conducted on participants aged 18 to 74 years who had lived in study area for 5 years or longer in Northeastern China gave comprehensive evidence of associations between long-term exposure to ambient air pollution and cardio-metabolic risk factors and CVD prevalence. Dyslipidemia, especially hyperbetalipoproteinemia, revealed the strongest associations with air pollutants, particularly in relation to $PM_{1.0}$ and NO_2. Moreover, participants with cardio-metabolic risk factors may be more vulnerable to associations of cardio-metabolic risk factors and CVD with air pollution.(Yang et al., 2019)

The associations between daily changes in ambient fine particulate matter (PM2.5) and cardiovascular diseases have been established robustly in mechanistic, epidemiologic and exposure studies. A study of Tainan, Taiwan demonstrated that transient and low concentrations of ambient PM2.5 trigger adult vascular disease events, especially cerebrovascular disease, regardless of age, sex, and exposure timing. Warning and delivery systems should be developed and employed to protect people from these prompt adverse health impacts.(Wu & Cheng, 2020)

New evidence now suggests that the co-emissions from human activity which produce compounds that are shorter-lived than carbon dioxide such as black carbon and organic carbon aerosols, carbon monoxide, volatile organic compounds, sulfur dioxide and others, are not well mixed and will exert geographically heterogeneous effects on atmospheric composition, and climatic systems.12 Not only are these co-emitted aerosols associated with poor health outcomes when inhaled by humans, but are also disturbing the balance of climate by influencing precipitation patterns and temperatures.(Burney et al., 2022)

Some minute pollutant particles can cross the alveolar-capillary membrane and circulate in the blood affecting the cardiovascular system directly. PM exposure is linked to abnormal activation of the haemostatic system, leading to a pro-coagulant and antifibrinolytic state.(Panasevich et al., 2009; Robertson & Miller, 2018) Exposure to air pollutants also causes some endothelial injuries, leading to an increase of endothelial cell apoptosis, a decreased circulating level of endothelial progenitor cells and tight junction protein degradation. After PM inhalation, we find a rise in interleukin-6, which leads to an increased fibrinogen, factor VIII and tissue factor release. A disrupted endothelial cell barrier, an increase in coagulation factors, a reduction in fibrinolytic capacity and platelet activation, all represent clear patho-physiological mechanisms to promote thrombus formation. Recent studies have also showed that inhaled nanoparticles can go into systemic circulation and accumulate in common sites of atherosclerosis; a direct toxic interaction is plausible.(Mark R. Miller et al., 2017)

The association of airborne particulate matter exposure with the deteriorating function of the cardiovascular system is basically driven by the impairment of mitochondrial-nucleus. The loss of delicate balance in retrograde communication from mitochondria to the nucleus often culminates in the methylation of the newly synthesized strand of mitochondrial DNA. A vivid alteration in mtDNA methylation leads to systemic inflammation, an etiological determinant for several co-morbidities, including vascular endothelial dysfunction and myocardial injury.(Rehman et al., 2023)

4. Conclusion

The mechanisms by which air pollutants contribute to cardiovascular diseases are complex and multifaceted. PM, especially fine particles (PM2.5), can penetrate deep into the respiratory system, entering the bloodstream and triggering systemic inflammation, oxidative stress, endothelial dysfunction, and atherosclerosis. NO_2, primarily emitted by traffic-related sources, promotes oxidative stress, inflammation, and endothelial dysfunction, leading to the development and progression of cardiovascular diseases. O_3, formed through chemical reactions involving sunlight and air pollutants, induces oxidative stress, inflammation, and autonomic dysfunction, all of which contribute to adverse cardiovascular events. SO_2, primarily emitted from fossil fuel combustion, triggers endothelial dysfunction, oxidative stress, and systemic inflammation, increasing the risk of cardiovascular events

In conclusion, the evidence presented in this literature review underscores the urgent need to recognize air pollution as a significant risk factor for cardiovascular diseases. By taking proactive measures to reduce air pollution, we can mitigate the burden of cardiovascular diseases, improve public health, and create a cleaner and healthier environment for future generations.

Compliance with ethical standard

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I would like to express my sincere gratitude to the researchers, scientists, and authors whose valuable studies and publications formed the foundation of this review. Their dedication to advancing knowledge in the field of public health has significantly enriched the content and insights presented in this paper.

Disclosure of Conflict of Interest

There is no any conflict of interest that could have influenced the impartiality and objectivity of this literature review on air pollution and cardiovascular diseases.

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