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# Risk of Cardiovascular Disease Associated with the Exposure of Particulate Matter (PM<sub>2.5</sub>): Review

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## **Abstract**

Association of particulate matter ( $PM_{2.5}$ ) increases the risk of cardiovascular disease. Experimental and clinical evidences suggested that  $PM_{2.5}$  is directly linked with cardiovascular disease.  $PM_{2.5}$  also plays an important role in the biological mechanisms influencing in the cardiovascular system. Here in this review, we tried to discuss that  $PM_{2.5}$  is associated with the increase risk of cardiovascular disease in which  $PM_{2.5}$  air pollutants enter through the alveolar of the lungs through the systemic circulation inducing cardiovascular disease. More studies need to be done for further understanding to clarify the interactions of  $PM_{2.5}$  components associated with cardiovascular disease.

# Keywords

Particulate Matter, Air Pollution, Cardiovascular Disease, Risk

#### 1. Introduction

Previous studies have evidenced particulate air pollution with adverse health effects relating with cardiovascular disease, and the ambient air pollution effect is linked with different diseases of cardiovascular with the upregulated cardiovascular mortality and morbidity [1] [2] [3]. The particulate matter (PM) with aerodynamic diameter  $\leq 2.5~\mu m$  can penetrate deep into the alveolar regions of the lung, which results in cardiovascular and other health effects. Many studies have been done for the better understanding the PM characteristics to observe the associated cardiovascular diseases [4] [5] [6]. The toxicological and epidemiological studies evidenced that PM<sub>2.5</sub> component plays an important role in cardiovascular responses [2] [6]. Many studies done before have evidenced that the

sources of air pollution which are responsible for the cardiovascular and other health effects are directly linked with the traffic-related particles [7]. It has been evidenced that the air pollutants PM, which enters the circulatory system easily induces systemic inflammation and hypercoagulability [8] [9], alters cardiac autonomic nervous system [10], induces in significant increased blood pressure [11] [12], and promotes cardiovascular diseases through oxidative pathway [13] [14]. Epidemiological studies reported that the air pollution exposure evidenced in the growth of cardiovascular health effects [15] [16] [17].

Cardiovascular disease in respect with cerebral vascular disease, coronary artery disease and peripheral artery disease are the leading cause of death worldwide [18]. Cardiovascular disease is associated with risk factors including family history, ageing, smoking, diabetes, hyperlipidemia and hypertension [19]. Importantly, the healthy life style may decrease the risk of cardiovascular disease in which one study suggested that Mediterranean diet helps in deceasing risk of cardiovascular disease, while the incidence of cardiovascular disease can be raised taking excessive amount of carbohydrate and high fat diets [20]. Thus, it became a global and socio-economic burden with significant increased populations is exposed to particulate matters [21]. PM components play an important role in the biological mechanisms associated with air pollution but the adverse effects in the cardiovascular outcomes are not fully understood till now. There were only limited number of studies done in this; where more studies are need to be done to better understand the features associated to PM<sub>2.5</sub> and cardiovascular disease in a large population so that it could help in deteriorating the risk of cardiovascular disease.

# 2. Biomarkers Associated with Exposures to PM<sub>2.5</sub> and CVD

Experimental data have demonstrated that PM<sub>2.5</sub> may mediate a systemic cellular inflammatory response through TLR4/NADPH oxidase-dependent mechanisms [22]. The human studies done previously suggested that increase in circulating biomarkers of inflammation, coagulation, and vasoconstriction following PM exposure can be one of the main pathways [2]. In the study by Chen R *et al.*, they found positive associations of PM<sub>2.5</sub> CVD biomarkers, which include six inflammatory markers fibrinogen, CRP, ICAM-1, VCAM-1, P-selectin, MCP-1; three coagulation markers PAI-1, CD40-L, vWF; and the vasoconstrictor ET-1 [23]. This study evidenced vital insights in the characteristics of PM<sub>2.5</sub> that could implicate the CVD prevention.

One another study suggested that circulating inflammatory biomarkers may be increased by short term inhalation of particulate matters [2]. These biomarkers included CRP, fibrinogen, ICAM-1, VCAM-1. P-selectin and MCP-1. Another panel study done by Wu S *et al.* suggested that the PM2.5 constituents associated with the biomarkers of inflammation, coagulation and homocysteine in the context of traffic related air pollution play the vital role influencing the CVD [24]. They also evidenced that the circulatory biomarkers of TNF- $\alpha$ , fibri-

nogen, PAI-1 and P-selectin more likely responsive to  $PM_{2.5}$  metal contents and Zn, Co, Mn and NO<sub>3</sub> were more likely associated with these markers.

These biomarkers are directly related with CVD which are associated with short term air pollution exposure and these biomarkers are predicted to be the risk of CVD [25] [26] [27]. A panel study in patients with chronic pulmonary disease found the increased concentration of air pollution are directly associated with the biomarkers of inflammation and coagulation [28]. Growing evidence suggested that ambient air pollution is associated with exacerbation of the chronic diseases. These biomarkers of systemic inflammation and traffic related air pollutants are directly associated with the risk of CVD morbidity and mortality leading to platelet activation and decreased antioxidant enzyme activity in elderly people [16] [17].

# 3. Exposure to PM<sub>2.5</sub> Components or Constituents

The associations of exposures to PM components were mostly found as organic carbon (OC) and transition metals (iron, manganese, zinc, etc.) [29] [30]. Airborne PM is a constituent of different types of chemical which induce in the biological effects of total PM [2] [31]. In addition, PM related to different sources (e.g., traffic, secondary) also has been associated with cardiovascular effects in recent studies [15] [17] [32]. The effects of different PM chemical properties in the above mentioned biological mechanisms connecting air pollution to adverse the cardiovascular diseases are not well understood till now. PM<sub>2.5</sub> contains different types of chemical components, and some studies have reported whether these chemical components may have differential adverse effects on cardiovascular system [33] [34] but it still remains unclear if these components are responsible in the cardiovascular diseases effected by PM<sub>2.5</sub>.

Heavy haze-fog episodes have frequently occurred in recent years [35] [36]. For example, starting in early January 2013, Beijing experienced multiple prolonged periods of severe smog; the peak hourly concentration of ambient  $PM_{2.5}$  soared to 800  $\mu$ g/m³, and the annual ambient  $PM_{2.5}$  concentration in 2013 reached 89.5  $\mu$ g/m³ [37]. The extremely high ambient  $PM_{2.5}$  concentrations have attracted extensive public attention because of the adverse health effects. However, evidences by the previous studies suggested that the effects of different particle constituents in the air pollution-related cardiovascular effects is generally lacking which might be limited number studies done in the particle constituents [35].

Among the  $PM_{2.5}$  components, metals Fe, Zn, Cd, Mn and Pb come from the traditional source of industrial wastages, and metals Al, Mg, Ba and Ti (and part of Fe and Mn) originating from mineral aerosols that would be likely from re-suspended road dust and long-range transported dust;  $SO_4^{2-}$ ,  $NO_3^{-}$  and Se are mainly generated from coal burning whereas the chemical transformation that formed the secondary aerosols can also produce a lot of  $SO_4^{2-}$  and  $NO_3^{-}$  [38]; Cl<sup>-</sup> and F<sup>-</sup> generally indicate the waste incineration and part of coal burn-

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ing in the rural areas; additionally, some metals Fe, Zn, V, Mn and Pb may also be contained in traffic-related emissions in addition to the carbonaceous fractions and gases [39].

## 4. The Health Effects of Indoor Air Pollution

Indoor air pollution from the past can be described when humans first moved to temperate climates during winter season. People used to fire inside the house for different purpose like cooking and warmth light. As a result, 2 million people deaths were estimated mostly in developing countries [40]. House hold biomass fuel combustion led to chronic bronchitis with the high exposure of cooking stoves worldwide being used by the people [41]. They cook inside the house typically or poorly functioned stoves which leads the air pollution and increase the risk of respiratory disease and other health problems. It may expose to infant, women, men, and elderly. Person who spends more time in kitchen, they may have higher risk of getting the chance of cardiovascular and respiratory diseases. Open wooden fires or poorly functioned stoves were typically used which leads to the increased air pollution [42].

Cultural practice common in developing countries may promote exposure of infants, women, the elderly and the sick. Since it is the woman who generally cooks, their exposure is much higher than men [43]. Most mothers carry their children on their back while she cooks. During those time, their infants spent most of the times breathing smokes. Some of the most vulnerable people typically spend more than 95% of their time indoor. The gender difference was not easy to explain because men may have an increased risk of cardiovascular events [44], but increased depositions of particles in the lung and higher airway responsiveness could make women more vulnerable to PM<sub>2.5</sub> pollution [45]. Air quality and human health are strongly linked.

The health related compounds emitted from solid fuel cook stoves include: carbon monoxide (CO), sulfur dioxide (SO<sub>2</sub>, coal burning stove), PM and some hydrocarbon constituents. Solid fuels which are used as household, led to high exposures to unhealthy indoor air pollution, particularly for those women working and cooking indoors [43]. Significant long-term exposure in environments containing the products of incomplete combustion and particulates found in solid-fuels moke can cause chronic diseases as well as other adverse health effects. A recent study conducted in Yunnan province of China evidenced strong correlation between residential coal use and lung cancer occurrence [46] [47].

## 5. Hospital Emergency Visits with the Exposure to PM<sub>2.5</sub>

PM<sub>2.5</sub> leads to more than 1.2 million deaths ranking at number 4 among all those risk factors conducing the health burden in China [21]. The main reasons that were associated with emergency hospital visit are related with the burning of fossil fuel (e.g., coal and oil). These lead in cardiovascular and respiratory diseases which are the major causes of emergency hospital visits. Previously, the

studied evidenced that the exposures to  $PM_{2.5}$  leading cardiovascular diseases lead to hospital emergency visits and hospital admissions, have been extensively reported in the U.S. and Europe [48] [49] [50].  $PM_{2.5}$  exposure and cardiovascular morbidity is more crucial in China than in other developed countries [51]. Moreover, only a few studies have been performed in other large Asian countries [52] [53] [54].

It is clear that epidemiological evidence reported in Western countries cannot be directly generalized to Asian populations [53]. Therefore, the cardiovascular effects of PM<sub>2.5</sub> exposure require further study in Asia. With rapid urbanization and industrialization during the past three decades, PM<sub>2.5</sub> air pollution in China has become a serious public health problem [35] [37]. There are a few studies that have examined the associations between PM<sub>2.5</sub> and cardiovascular morbidity in Beijing [1] [55] [56]. However, these studies only used emergency hospital visits data from one hospital and PM<sub>2.5</sub> data from a fixed monitoring station, which might make the results insufficiently representative due to Beijing's large population size and wide residential distribution. Another study conducted in Beijing only evaluated the effects of PM<sub>2.5</sub> on ischemic heart disease and did not include other cardiovascular diseases, such as cerebrovascular disease [57].

Moreover, the PM<sub>2.5</sub> monitoring data have been released by the Beijing Environmental Protection Bureau (BEPB) since October 2012 and can be accessed by the general population [58]. The availability of data from this monitoring network provided us with an opportunity to assess the short-term effects of PM<sub>2.5</sub> on cardiovascular morbidity. However, few reports have directly evaluated the effects of PM<sub>2.5</sub> on total and cause-specific respiratory diseases using hospital emergency visits as a morbidity indicator. In addition, most of these studies were conducted in Western developed countries; however, because of the different level of effects, such as ambient PM<sub>2.5</sub> levels, characteristics of ambient PM<sub>2.5</sub>, population susceptibility, and weather patterns, there is still a need to assess the health effects of PM<sub>2.5</sub> exposures in developing countries.

# 6. Conclusion

Our review evidenced that particulate matter ( $PM_{2.5}$ ) is directly associated with the risk factors of cardiovascular disorders.  $PM_{2.5}$  air pollution is linked with the increasing emergency hospital visits inducing cardiovascular diseases. More precisely, experimental and clinical evidences suggested that  $PM_{2.5}$  increases the pathogenesis of the cardiovascular diseases. Hence, more studies need to be done to elucidate the risks of  $PM_{2.5}$  associated with cardiovascular diseases.

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# **Abbreviation**

Al Aluminium, Ba Barium, Cd Cadmium, Cl Chlorine, Co Cobalt, CRP C-reactive protein, ET-1 Endothelin 1, FFluorine, Fe Iron, ICAM-1 Intercellular adhesion molecule 1, MCP-1 Monocyte chemoattractant protein 1, Mg Magnesium, Mn Manganese, NADPH Nicotinamide adenine dinucleotide phosphate, NO $_3$  Nitrate, PAI-1 Plasminogen activator inhibitor-1, PAI-1 Plasminogen activator inhibitor-1, Pb Lead, SO $_4$  Sulfate, Ti Titanium, TLR4 Toll-like receptor 4, TNF- $\alpha$  Tumor necrosis factor alpha, V Vanadium, VCAM-1 Vascular cell adhesion protein 1, vWF Von Willebrand factor, Zn Zinc.