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Research on the effects of PM2.5 exposure on respiratory and cardiovascular diseases in an aging population

Haocheng Li

Earth Science Department, University Collage London, WC1E 6BT, USA

zcfbhli@ucl.ac.uk

Abstract. In contemporary times, there is an observable inclination towards the exacerbation of air pollution, which has emerged as a prominent global environmental issue. Among the various factors contributing to this issue, PM2.5 emerges as a key determinant. The geriatric demographic exhibits a heightened vulnerability to respiratory and cardiovascular disorders. This study offers a thorough analysis of the correlation between PM2.5 and human pathology and epidemiology. It also investigates the many pathophysiological pathways that establish a connection between exposure to PM2.5 and the development of respiratory and cardiovascular disorders. The results of this study suggest that PM2.5 has a substantial impact on the occurrence and fatality rates of several common illnesses within the senior demographic. Furthermore, this paper offers recommendations for potential public health interventions intended to alleviate the adverse effects of this avoidable determinant on the development of diseases and death rates.

Keywords: aging people, respiratory disorders, cardiovascular disorders, pathophysiological pathways, public health.

1. Introduction

The WHO reported 3.7 million premature deaths from outdoor air pollution in 2012 [1]. The worldwide Burden of Disease Study reported poor indoor air quality and ambient air pollution as the top seven and twelve worldwide health hazards [2]. Air pollution is significant due to PM. PM consists of solid and liquid particles suspended in the atmosphere. Particles of nitrates, sulphates, elemental and organic carbon, organic molecules, biological compounds, and metals vary in size and chemical content [3]. After the 1952 Great London Haze, which killed over 12,000 people, PM, especially PM2.5, is a risk [4]. Small solid particles and liquid droplets under 2.5 micrometers are called PM2.5. PM2.5 was estimated to kill 399,000 EU citizens prematurely in 2014 [5]. Fast urbanization, industry, and high population density increase PM2.5 levels globally [6]. Income, urbanization, and services greatly impact PM2.5 pollution [7]. Chemical combustion in industrial and motor vehicle environments produces most PM2.5 carbonaceous compounds [8]. These species are characterized by quantifying elemental and organic carbon [9].

PM2.5 particles can slip through nasal hair filters and contain harmful compounds due to their vast surface area and tiny diameter [10]. Thus, airflow can convey these particles to the terminal respiratory tract for collection. Air exchange in the lungs can harm many body components [11]. PM2.5 damages the respiratory system by causing asthma, respiratory tract inflammation, lung function loss, and carcinogenic promotion [12]. Inhaling fine particulate matter can cause cardiovascular disease (CVD),

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including arrhythmias, coronary vascular disease, heart failure, peripheral arterial disease, and venous thrombosis, especially brain blood supply issues like ischaemic and hemorrhagic stroke. CVD is associated with 2.5-micron particles [13]. Most cardiovascular and respiratory illness deaths occur in elderly people [14, 15]. This paper aims to present a comprehensive analysis and synthesis of the evidence indicating that PM2.5 has a significant role in causing respiratory and cardiovascular fatalities among the older demographic. Additionally, it will offer practical suggestions for mitigating PM2.5 exposure among this vulnerable population.

2. Analysis on the effects of PM2.5 exposure

2.1. Mechanistic study of PM2.5 and human respiratory system

2.1.1. Free radical peroxidation damage. PM2.5 molecules contain free radicals, metals, and chemical compounds that oxidize lung cells, which may cause the most harm [16]. The surface of ambient particulate matter produces free radicals. Transitional elements like iron, copper, zinc, and manganese, as well as polycyclic aromatic hydrocarbons and lipopolysaccharides, are found in PM2.5, which can increase lung free radical production, deplete antioxidants, and cause oxidative stress [17]. Reactive oxygen species (ROS) from water-soluble particles activate metals to produce hydroxyl radicals and DNA oxidation is mostly caused by hydroxyl radicals [18]. If not repaired quickly, damaged DNA can cause teratogenesis, carcinogenesis, and mutation [19]. Particles damage DNA, hinder DNA repair, and encourage DNA fragment replication, causing carcinogenesis [19].

2.1.2. Imbalance in intracellular calcium homeostasis. Calcium has a crucial role as a secondary messenger in the regulation of various cellular physiological and pathological processes. Elevated calcium levels have been found to induce inflammation and result in cellular damage. The presence of PM2.5 leads to an elevation in free radicals or reactive oxygen species (ROS), resulting in a reduction in cellular antioxidant capacity. This, in turn, induces lipid peroxidation of the cell membrane and an increase in intracellular calcium ion concentration. Furthermore, an increase in intracellular calcium ion (Ca^{2+}) levels has the potential to enhance the production of reactive oxygen species (ROS) [20]. The upregulation of calcium-sensitive receptors leads to the induction of programmed cell death (apoptosis) and uncontrolled cell death (necrosis) [21]. Exposure to PM2.5 particles has the potential to cause cellular harm through the modulation of intracellular calcium ion (Ca^{2+}) levels by reactive oxygen species (ROS) [22].

2.1.3. Inflammatory damage. Multiple transcription factor genes and inflammation-associated cytokine genes are overexpressed in PM2.5, causing inflammatory damage [23]. Increased PM2.5-induced inflammation increases neutrophils and pine dust raises bronchoalveolar lavage fluid eosinophils, T cells, and mast cells [24]. Both types of alveolar macrophages are affected by PM2.5 and its microenvironment. Thl-type cytokines (IL-12, IFN-γ) and infections activate M1-polarized alveolar macrophages, which increase inflammation in vivo. M2-polarized alveolar macrophages work with Th2-type cytokines (IL-4 and IL-13) and immunomodulatory cytokines (IL-10), suppressing inflammation [25]. This suggests that cytokines can stimulate neutrophils, T cells, and eosinophils to move to the lungs and other tissues and migrate there on their own, increasing cellular activity and producing more inflammatory cytokines and chemokines. Interactions between inflammatory cells and cytokines can damage lung cells [10].

2.2. Mechanistic study of PM2.5 and human cardiovascular diseases

2.2.1. Oxidative stress and inflammation. Respiratory inflammation can result from particle inhalation. Systemic inflammation and cardiovascular discomfort result after PM 2.5 inhalation [26]. PM 2.5 enters the alveolar epithelium, causing inflammation and oxidative damage. Lung cells emit inflammatory

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mediators such as IL-6, IL-8, TNF-α, and interferon into the bloodstream and bronchial fluid [27]. These substances spread and cause systemic effects after entering the bloodstream [28]. IL-6 accelerates air pollution response and CRP generation. CRP, a major protein in the Acute Phase Reactant (APR) system, indicates inflammation and cardiovascular disease vulnerability [29]. A positive correlation was established between PM 2.5 and blood CRP. Research shows that a 100 μg/m3 increase in PM 2.5 concentrations causes an 8.1 mg/L rise in blood CRP levels [30]. Older people with elevated TLR2 methylation may be more vulnerable to PM 2.5's cardiac autonomic neuron effects [31].

2.2.2. Abnormal activation of the haemostatic system. PM 2.5 inhalation can cause venous thrombosis and slow plasma plasminogen blood clotting. Inhaling PM 2.5 particles can cause widespread lung inflammation and circulatory system irritation [32]. This may then produce abnormal hemostatic activation. In animal models and in vitro cellular research, PM 2.5 exposure increases fibrinogen and tissue factor. Fine particulate particles in blood vessels may directly activate platelets [33]. Platelets maintain blood hypercoagulability, which aids thrombosis. PM 2.5 exposure increases the risk of acute thrombosis, including myocardial infarction and stroke. Moreover, fibrinogen increases stroke risk [34].

2.2.3. Autonomic nervous system disorder. PM causes autonomic nerve damage [35]. The vagus nerve controls sinus node autoregulatory cells, which regulate heart rhythm. Autonomic nervous system (ANS) responses to acute PM 2.5 exposure increase arrhythmias and acute cardiovascular events [36]. These unfavorable effects are substantial, especially for seniors. Heart rate variability (HRV) in the elderly is associated with PM 2.5 exposure. Exposure to PM 2.5 at 21.2-80.3 μg/m3 significantly lowered HRV by 35.7% compared to a 2-hour clean air control condition [37]. PM 2.5 affects hypertensives more than others. The reduction in heart rate variability (HRV) shows cardiac autonomic nerve system disruption, which may indicate early cardiovascular illness [38].

2.2.4. Endothelial cell injury. PM 2.5 may cause cardiovascular illness by damaging vascular endothelial cells, which is a pathophysiological underpinning of the disease. Adhesion molecules and apoptosis were seen in PM 2.5-treated cardiovascular endothelial cells after 24 h. PM 2.5 increased cell mortality, suggesting it may harm the vascular endothelium and induce cardiovascular disease [39]. Several investigations have revealed that certain metals and decreased endothelium repair ability may be essential components of PM 2.5-induced cardiovascular damage [40].

3. Recommendation

The elderly population should take social and personal measures to reduce the risk factors for contracting or aggravating respiratory and cardiovascular diseases due to PM2.5 exposure [10, 41]:

For older individuals, the first step is to take antioxidant supplements or eat a healthy diet (e.g., fish oil w-3 fatty acids). Secondly, older adults can watch the weather forecast and if PM 2.5 levels are too high, they should stay home and close all windows and doors. If they must go outside, they should wear certified masks and limit outdoor activities. Finally, elderly people should reduce smoking and alcohol consumption as well as other cardiovascular and respiratory risk factors.

For the community, environmental protection authorities should monitor pollutant emissions from factories. Secondly, the government should urge people to minimise driving and reduce tailpipe emissions.

4. Conclusion

This research delves into the pathomechanisms and literature on PM2.5 exposure and geriatric respiratory and cardiovascular illnesses. PM2.5 damages the respiratory system by free radical peroxidation and intracellular calcium imbalance. PM2.5 damages the aged respiratory system by free radical peroxidation, intracellular calcium imbalance, and Damage from inflammation, Oxidative stress, inflammation, and haemostatic system abnormalities from PM2.5 can affect the old respiratory system. In the elderly, autonomic nervous system malfunction and endothelial cell injury influence

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cardiovascular health. This document offers helpful individual and societal suggestions. To start, elderly adults can take antioxidant supplements or consume a nutritious diet including fish oil w-3 fatty acids. Second, elderly individuals can watch the weather and stay inside with all windows and doors closed if PM 2.5 levels are high. If they must go outside, wear certified masks and minimize activities. Finally, seniors should cut smoking, drinking, and other cardiovascular and respiratory risk factors. Environmental protection agencies should monitor factory pollutant emissions for the community. Second, the government should encourage driving less and reducing tailpipe emissions. This research does not examine the impacts of PM2.5 on the elderly beyond cardiovascular and pulmonary systems, such as mental health, and its suggestions lack validation, feasibility, and efficiency. Explore feasibility and efficiency. Future PM2.5 research could include newborns.

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