REVIEW ARTICLE

PM2.5 Air Pollution: Molecular Pathogenesis and Global Disease Burden in Cancer and Chronic Non-Communicable Diseases

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Abstract. Fine particulate matter (PM2.5) air pollution significantly increases illness and death, especially among vulnerable groups like children and the elderly in low- and middle-income countries. Epidemiological data strongly associates PM2.5 exposure with higher rates of morbidity and mortality across a range of health issues, including respiratory and cardiovascular diseases, metabolic and neurological disorders, as well as different types of cancer. This review synthesizes current research on the health impacts of PM2.5, including its association with respiratory diseases, cardiovascular conditions, and various cancers. The review also presents the global disease burden linked to PM2.5, incorporating various epidemiological studies. It also highlights the critical need for regulations, innovative technology, and public health strategies to reduce exposure and protect human health. Despite significant progress, important gaps persist in understanding the specific molecular mechanisms of PM2.5 toxicity across organ systems and the relative contributions of distinct PM components. Future research is essential to better understand the molecular mechanisms of PM2.5 and to develop effective strategies to protect public health. Coordinated efforts across various sectors are necessary to combat the health risks posed by PM2.5

Keywords: PM2.5, Carcinogenesis, Respiratory Disease, Cardiovascular disease, Chronic non-communicable diseases

1. Introduction

Currently, ambient air pollution from fine particulate matter (PM2.5) is a widespread environmental threat that significantly increases the global disease burden (Cohen et al. 2017). This common environmental pollutant, known as PM2.5, is defined as particulate matter suspended in the air with an aerodynamic diameter of 2.5 micrometers or smaller, allowing it to enter the respiratory system and spread throughout the body (Xing et al. 2016). This prevalent pollutant has been linked to numerous health problems, including respiratory and cardiovascular issues, neurological disorders, and multiple types of cancer (Pun et al. 2017; Thangavel, Park, and Lee 2022). Its microscopic size allows it to bypass the body's natural defense mechanisms, facilitating its entry into the bloodstream and subsequent deposition in various organs (Thangavel, Park, and Lee 2022). The World Health Organization attributes approximately one million premature deaths annually specifically to PM2.5 exposure, establishing it as a significant environmental health risk (Yu et al. 2024). Indeed, outdoor air pollution alone accounted for an estimated 4.2 million deaths worldwide in 2019, highlighting the critical need to further elucidate the intricate molecular mechanisms by which PM2.5 exposure instigates disease and quantify its extensive global burden (Liu, Dajnak, et al. 2024). This review aims to synthesize current knowledge regarding the molecular pathogenesis linking PM2.5 exposure to chronic non-communicable diseases and various cancers, while also quantifying its substantial global disease burden. This includes an examination of its impact on respiratory, cardiovascular, and cancers. Furthermore, we aim to consolidate existing knowledge and evidence, providing a comprehensive overview of the mechanisms through which PM2.5 contributes to systemic pathology. This understanding may reveal future therapeutic targets and shape public health strategies.

2. Fine Particulate Matter (PM 2.5)

Fine particulate matter, especially PM2.5, serves as a major indicator of air pollution arising from both natural events and human activities, profoundly impacting air quality, regional and global climate trends, and, importantly, human health (Kelly and Fussell 2015; Bhattarai et al. 2024). These minute particles, composed of a heterogeneous mixture of solid fragments, liquid droplets, and chemical species (Sulfates, Nitrates, Ammonium, Organic and elemental carbon, Metals such as lead, cadmium and arsenic), can penetrate deep into the respiratory system, with the smallest fractions even reaching the brain (Xing et al. 2016). The International Agency for Research on Cancer (IARC) recognizes PM2.5 as a Group 1 carcinogen, further emphasizing the need to understand and mitigate its impact on human health (Stewart et al. 2003). The size range of PM2.5 allows for their efficient deposition in the alveoli, facilitating the systemic absorption of associated toxicants (Thangavel, Park, and Lee 2022). Their pervasive presence in the atmosphere is largely attributed to emissions from on-road vehicles and industrial processes, contributing significantly to widespread human exposure (Li and Managi 2021). The physicochemical properties of these suspended particles are defined by their size and morphological features, influencing their dispersion duration in the atmosphere. These characteristics further dictate their capacity for long-range atmospheric transport and their potential to induce systemic inflammatory and oxidative stress responses upon inhalation (Zhai et al. 2022; Wang et al. 2023).PM0.1 (an aerodynamic diameter of less than 0.1 μm), though less characterized, pose an even greater health risk due to their enhanced ability to traverse biological barriers and their disproportionately high surface area, which facilitates the adsorption and transport of harmful chemical species (Larionov et al. 2022). The health impact of airborne particulate matter has long been a concern for clinicians, biologists, and the public, with epidemiological studies consistently linking PM2.5 to increased cardiopulmonary morbidity and mortality (Krittanawong et al. 2023). With many epidemiological studies confirming the association of PM with allergic respiratory diseases, an increasing number of follow-up empirical studies are being conducted to investigate the mechanisms underlying the toxic effects of PM on asthma and allergic rhinitis (Wu et al. 2018). PM with larger size, such as PM10 (diameter less than 10 μm), primarily impact the upper airways, whereas the smaller PM2.5 (diameter less than 2.5 μm) particles can access the deeper regions of the lungs. (Xing et al. 2016).

3. Disease Burden and Epidemiological Evidence

Given that PM2.5 can reach the deepest regions of the lungs, its impact on respiratory health is particularly pronounced, with exposure linked to a substantial increase in respiratory disease hospitalization and mortality (Pryor, Cowley, and Simonds 2022; Feng et al. 2022). This heightened risk is evident globally, but particularly in low- and middle-income countries where the burden of respiratory mortality linked to particulate matter is significantly higher (Guo et al. 2023). Moreover, short-term exposure to elevated PM2.5 concentrations has been definitively linked to acute adverse cardiovascular events, including increased risks of heart attack and stroke (Pope et al. 2006; Anyachebelu et al. 2023). This is further compounded by evidence suggesting that long-term exposure to PM2.5 is associated with severe respiratory illnesses and accelerated decline in lung function (Schwartz 2000). The intricate interplay between PM2.5 exposure and the development of chronic bronchitis in adults, as well as the prevalence of bronchitis in children, further underscores the extensive public health ramifications. This includes an increased risk of asthma, chronic obstructive pulmonary disease, and bronchitis, with vulnerable populations like children and the elderly experiencing particularly severe and irreversible health issues from prolonged exposure (Zhang et al. 2023; Marcon 2024). Furthermore, global analyses indicate that PM2.5 exposure contributes to millions of premature deaths annually, establishing it as a leading environmental risk factor for global

disease burden (Fang et al. 2025). Exposure to PM2.5 is also associated with increased daily mortality rates from non-accidental, circulatory, hypertensive, and chronic lower respiratory causes (Parasin and Amnuaylojaroen 2024; Gutierrez-Avila et al. 2023). While the mass concentration of PM is often regulated, the detrimental effects are more strongly associated with the PM's composition, surface area, and size (Samake et al. 2017). Beyond respiratory and cardiovascular ailments, PM2.5 exposure is also implicated in neurological disorders, metabolic dysregulation, and various cancers, highlighting its pervasive systemic effects (Pryor, Cowley, and Simonds 2022). Moreover, global analyses indicate that outdoor air pollution, encompassing PM2.5 and other particulate matter, contributes to an estimated 3 to 9 million deaths annually (Collaborators 2018; Cohen et al. 2017). The long-term exposure to ambient PM2.5 has been consistently associated with an elevated risk of premature mortality across various populations and geographical regions, even at concentrations below established national guidelines (Vodonos, Awad, and Schwartz 2018; Ko and Kyung 2022). Numerous epidemiological studies, including those conducted in China, have robustly demonstrated a direct correlation between short-term PM2.5 exposure and increased mortality rates from various cardiopulmonary diseases (Chen et al. 2017). A robust body of evidence also indicates that both short-term and long-term exposure to airborne pollutants, including PM2.5, significantly exacerbates conditions such as chronic obstructive pulmonary disease, asthma, and various other respiratory ailments, leading to higher rates of hospitalization (Manisalidis et al. 2020). This is particularly concerning given that air pollution is a non-threshold pollutant, meaning that even low levels of exposure are associated with excess deaths (Chen et al. 2019). This makes air pollution the leading environmental health risk factor in the United States, contributing to an estimated 100,000 to 200,000 excess deaths per year (Castillo et al. 2021). PM2.5 exposure correlates significantly with bladder cancer mortality, demonstrating spatial variability across gender and regional demographics in Taiwan (Yeh et al. 2017). The extensive U.S. cohort study found that PM2.5 exposure is linked to increased risk of estrogen receptor-positive breast cancer, highlighting the need for further research on regional associations and PM2.5 chemical composition (White et al. 2024). Furthermore, the meta-analysis identified a significant association between PM2.5 exposure and colorectal cancer risk, notably higher in North America (Fu et al. 2024). Inevitably, there have been studies establishes a connection between PM2.5 exposure and lung cancer incidence, highlighting the necessity for targeted public health interventions and further research on environmental risk factors to mitigate lung cancer risk (Neupane et al. 2024; Wei et al. 2025; Zhu et al. 2025). In 2018, Babatola and colleagues reported that globally, air pollution is responsible for an estimated 3.1 million premature deaths each year, with PM2.5 contributing significantly to this figure. This encompasses various health outcomes attributed to outdoor air pollutants (Babatola 2018). The persistent exposure to fine particulate matter necessitates a deeper understanding of its molecular pathogenesis to inform targeted interventions and policy reforms.

4. Molecular Pathogenesis of PM2.5-Mediated Toxicity

The toxicological mechanisms involve not only direct cellular damage but also complex interactions with cellular machinery, leading to chronic inflammation, oxidative stress, and DNA damage (Pryor, Cowley, and Simonds 2022). These processes are fundamental to the development and progression of various PM2.5-associated diseases, including cardiovascular diseases, respiratory illnesses, and various forms of cancer. The interaction of PM2.5 with biological systems initiates a cascade of molecular events, beginning with the particles' deposition in the respiratory tract and subsequent translocation into the bloodstream (Pryor, Cowley, and Simonds 2022). Once internalized, these fine particles, laden with diverse chemical components, induce systemic inflammation and oxidative stress, thereby compromising cellular integrity and function across multiple organ systems. This widespread exposure to PM2.5 contributes to approximately 5.2 million

deaths each year, encompassing a range of health issues from respiratory and cardiac complications to a higher incidence of various chronic diseases (Huang et al. 2024). This systemic inflammatory response is further exacerbated by the activation of various cellular signaling pathways, including those involving nuclear factor-kappa B (NF-kB) and mitogen-activated protein kinases (MAPK), which collectively promote the transcription of pro-inflammatory cytokines and chemokines (Zhang et al. 2018; Kim et al. 2022). Furthermore, PM2.5 can directly interact with genetic material, leading to DNA adduct formation and chromosomal aberrations, which are critical steps in mutagenesis and carcinogenesis (Longhin et al. 2013). While the precise mechanisms underlying PM toxicity remain an active area of research, the generation of reactive oxygen species is widely recognized as a pivotal pathway through which PM induces both chronic and acute adverse health effects (Park et al. 2018). Particle surface reactivity, metallic components, and redox-cycling organic compounds within PM2.5 are critical properties contributing to this generation of reactive oxygen species (Rimauro et al. 2025).

4.1 respiratory disease

The interaction of PM2.5 with biological systems triggers a series of molecular events, mainly involving the production of reactive oxygen species and the activation of inflammatory pathways (Zhang et al. 2024). The oxidative stress can cause immediate cellular injuries and instigate ongoing inflammatory reactions in the lungs, playing a significant role in the development of conditions like asthma and chronic obstructive pulmonary disease (COPD). This disruption can impair airway epithelial barrier function, leading to exaggerated inflammatory responses and airway remodeling through the Wnt5a/β-Catenin Pathway, which are hallmark features of obstructive lung diseases (Zou et al. 2021). Additionally, this can result in the activation of signaling pathways that promote fibroblast proliferation and extracellular matrix deposition, contributing to pulmonary fibrosis and irreversible lung damage (Liu, Han, et al. 2024). The chronic inflammatory state induced by PM2.5 can also prime the lungs for increased susceptibility to infections and perpetuate tissue damage, accelerating the progression of respiratory pathologies (Luo et al. 2024). PM2.5 exposure can further alter the airway microbiota, inducing dysbiosis that compromises respiratory immunity and increases susceptibility to viral infections, including SARS-CoV-2 by altering the expression of angiotensin converting enzyme II (Zhou et al. 2023; Lin et al. 2025). This disruption can impair airway epithelial barrier function, leading to exaggerated inflammatory responses and airway remodeling, which are hallmark features of obstructive lung diseases. Studies indicate that sustained exposure to high levels of particulate matter can lead to chronic airflow obstruction and structural changes in the small airways, including muscle hyperplasia, driven by the intrinsic properties of the particles themselves (Zhao et al. 2018). This damage can cause injury and narrow peripheral channels, ultimately leading to severe airway obstruction (Zou et al. 2021). Inhalation of particulate air pollutants directly induces lung inflammation by acting as an adjuvant for allergens or respiratory viral infections, primarily through the activation of macrophages and epithelial cells (Zou et al. 2021; Chen et al. 2020; Quek et al. 2024). Exposure to particulate matter can worsen existing asthma conditions and may play a role in the development of asthma, allergic rhinitis, and heightened sensitivity to aeroallergens (Chen et al. 2018; Tsai et al. 2025).

4.2 cardiovascular disease

Inhaling PM2.5 leads to the activation of pro-oxidative and pro-inflammatory mediators (interleukin-6 and tumor necrosis factor), as well as acute-phase reactants like C-reactive protein and vasoactive hormones (endothelins) that are subsequently discharged into the bloodstream and deposited on the walls of blood vessels (Mannucci, Harari, and Franchini 2019). These mediators induce systemic inflammation, endothelial dysfunction, and autonomic nervous system imbalance, all of which contribute to the increased risk of acute

cardiovascular events such as myocardial infarction and stroke (Breitner et al. 2019). Cardiovascular disease caused by particulate matter exposure has been strongly correlated with long-term exposure to PM2.5, which instigates complex molecular mechanisms contributing to atherosclerosis and adverse cardiovascular outcomes. These changes can manifest as alterations in heart rate variability, reflecting an impaired cardiac autonomic control (Magari et al. 2002). Exposure to PM2.5 exacerbates angiotensin II-induced hypertension by increasing oxidative stress and activating the Rho-kinase pathway, leading to abnormal vascular constriction, as confirmed in both animal and laboratory studies (Sun et al. 2008). In mice, PM2.5 exposure induces a prothrombotic state of enhanced platelet reactivity and faster clotting. These effects are largely preventable by blocking IL-6 or TNF-α, demonstrating a primary role for inflammatory signaling (Robertson and Miller 2018). Furthermore, PM2.5 exposure accelerated atherosclerosis in mice by promoting inflammation and oxidative stress, resulting in larger plaques and increased blood vessel damage. This occurred through activation of platelets and immune cells, driving disease progression (Zhu et al. 2019).

4.3 cancers

Particulate matter has been linked to various types of cancer , with particular emphasis on lung cancer, where its carcinogenic potential is mediated by inducing DNA damage, promoting chronic inflammation, and altering cellular signaling pathways. The International Agency for Research on Cancer (IARC) has classified outdoor air pollution, specifically particulate matter, as carcinogenic to humans, affirming its causal role in lung cancer development (Bhui et al. 2023). The chronic exposure to particulate matter can result in the activation of oncogenic pathways and epigenetic modifications, further contributing to uncontrolled cell proliferation and tumor progression. The oxidative potential of PM2.5, influenced by its diverse chemical composition, has been identified as a critical determinant of its toxicity, driving a range of adverse health outcomes (Daellenbach et al. 2020). This intrinsic characteristic facilitates the excessive generation of reactive oxygen species, leading to DNA damage, cellular dysfunction, and chronic inflammatory responses (Lim and Kim 2024). Exposure to PM2.5 in 16HBE (human bronchial epithelial cells) cell lines has been shown to induce DNA strand breaks, the formation of DNA adducts such as 8-hydroxydeoxyguanosine, and altered expression of DNA repair genes like OGG1(8-oxoguanine DNA glycosylase) and XRCC1(X-ray repair cross-complementing protein 1) (Niu et al. 2020). Similarly, the components of PM2.5 have been observed to inhibit the nucleotide excision repair process, thereby suppressing DNA repair mechanisms. The resultant accumulation of DNA damage is implicated in lung carcinogenesis (Mehta et al. 2008). Yang et al. found that PM2.5 exposure altered the expression of 143 genes in NSCLC cells, enhancing their proliferation and invasion (Yang et al. 2017).

5. Emerging Approaches for Health Protection

There are numerous approaches that can be utilized to protect human health by minimizing the adverse health consequences of PM2.5 exposure (Schulze et al. 2017). The effective protective strategy can result in notable decreases in heart rate variability, especially among individuals with pre-existing conditions like ischemic heart disease, hypertension, or diabetes (Park et al. 2005). One critical approach involves implementing stringent air quality regulations and promoting cleaner energy sources, which have been shown to significantly reduce ambient PM2.5 levels and subsequently decrease morbidity and mortality rates (Li et al. 2017; Yang 2025). This proactive strategy involves the adoption of renewable energy technologies and the enforcement of stricter emission standards for industrial and mobile sources (Ross, Chmiel, and Ferkol 2012). Furthermore, advancements in filtration technologies and air purification systems are pivotal in reducing indoor exposure to PM2.5, especially in vulnerable populations. These interventions directly address the source of pollution, decreasing overall population exposure and alleviating the burden on healthcare systems (Niu, He, and Chen 2024; Chen et al. 2022).

Table 1: Summary of Disease Outcomes Associated with PM2.5 Exposure

Disease category	Health outcome	Reference
Respiratory	- Trigger sustained inflammatory responses within the pulmonary system (through the Wnt5a/β-Catenin Pathway)	L. Zhang et al. 2024 Zou et al. 2021
	- Aggravate asthma and chronic obstructive pulmonary disease - Initiate the onset of asthma and allergic rhinitis	Chen et al. 2018 Tsai et al. 2025
	- Promote pulmonary fibrosis and irreversible lung damage	B. Liu et al. 2024
	- Increased susceptibility to lung infections	Luo et al. 2024
	- Alter the airway microbiota, inducing dysbiosis	Zhou et al. 2023 Lin et al. 2025
	- Lead to chronic airflow obstruction and muscle hyperplasia in small airways	Zhao et al. 2018
Cardiovascular	- Activate the release of inflammatory markers (interleukin-6 and tumor necrosis factor) that impact the walls of blood vessels	Mannucci, Harari, and Franchini 2019
	- Instigates complex molecular mechanisms contributing to atherosclerosis and adverse cardiovascular outcomes	Zhu et al. 2019
	- Induces a prothrombotic state of enhanced platelet reactivity and faster clotting	Robertson and Miller 2018
Cancers	- Generate reactive oxygen species, leading to DNA damage, cellular dysfunction, and chronic inflammatory responses	Lim and Kim 2024
	- Induce DNA strand breaks, formation of DNA adducts and altered expression of DNA repair genes (OGG1 and XRCC1) in human bronchial epithelial cells (16HBE)	B.-Y. Niu et al. 2020
	- Inhibit the nucleotide excision repair process and suppress DNA repair mechanisms that are implicated in lung carcinogenesis	Mehta et al. 2008
	- Enhance lung cancer proliferation and invasion	B. Yang et al. 2017

Beyond these technological and regulatory interventions, public health campaigns are vital for raising awareness about the risks associated with PM2.5 and for advocating for behavioral changes that can minimize personal exposure (Peng et al. 2025). The development and deployment of low-cost, high-resolution air quality monitoring networks could significantly improve the accuracy of exposure assessments and inform targeted interventions (Blaser et al. 1987). An integrated approach combining policy, technology, and public engagement is essential for effective mitigation of PM2.5-related health risks.

6. Conclusion

While these efforts are significant, it is equally vital to enhance notification systems for susceptible patients and to effectively control PM production to help lower the rates of chronic respiratory diseases, hospitalizations, and deaths due to acute exacerbations (Kyung and Jeong 2020). Further research is essential to investigate the specific molecular mechanisms by which different PM2.5 components contribute to various chronic diseases. This understanding will facilitate the development of targeted therapeutic and preventive strategies. (Hime, Marks, and Cowie 2018). The implementation of engineering solutions to drastically reduce emissions from sources such as cooking stoves and vehicles can significantly improve air quality and human health outcomes, as demonstrated by trials during the Beijing Olympics (Sierra-Vargas and Teran 2012). The complexity of atmospheric systems and the multifactorial nature of emission sources necessitate advanced air quality models and decision-support tools to identify the most impactful interventions for reducing population exposure and improving public health (Tessum et al. 2022). Such models can inform policy decisions aimed at curbing the burden of disease attributable to air pollution, which disproportionately affects low- and middle-income countries (Cohen et al. 2017). In summary, ambient air pollution is a significant global health concern, necessitating a comprehensive approach that integrates environmental, economic, and social policies to achieve sustainable improvements in air quality and public health outcomes (Schraufnagel et al. 2019). A critical step towards mitigating this global health crisis involves a concerted effort to enhance public awareness regarding the pervasive health threats posed by air pollution, alongside promoting effective communication strategies to foster community engagement in pollution reduction initiatives (Ramirez et al. 2019).

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