Literature Review

Effects of PM2.5 Exposure on the Respiratory System

Veghasanah Tanlie*, Indi Esha

Department of Pulmonology and Respiratory Medicine, Faculty of Medicine, Riau University, Pekanbaru

Corresponding Author:
Veghasanah Tanlie | Department of Pulmonology and Respiratory Medicine, Faculty of Medicine, Riau University, Pekanbaru | veghatanlie13@gmail.com

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Abstract

Fine particulate matter (PM2.5) is recognized as one of the most harmful air pollutants, contributing substantially to the global burden of respiratory diseases. Because of its small aerodynamic diameter, PM2.5 can penetrate deep into the lower respiratory tract and alveoli, inducing oxidative stress, persistent inflammation, immune dysregulation, and epithelial injury. These biological responses increase the risk of developing chronic respiratory diseases, including asthma, chronic obstructive pulmonary disease (COPD), pulmonary infections, and lung cancer. This narrative literature review summarizes current evidence on the pathophysiological mechanisms and respiratory health effects associated with PM2.5 exposure. The literature review was conducted using PubMed, Scopus, Web of Science, and Google Scholar, focusing on publications related to PM2.5 and respiratory diseases. Recent evidence consistently indicates that prolonged PM2.5 exposure is associated with impaired pulmonary function and increased respiratory morbidity and mortality. Indonesia's PM2.5 air quality standards remain less stringent than the World Health Organization (WHO) Air Quality Guidelines (AQGs), emphasizing the need for stronger air pollution control policies and effective mitigation strategies. Comprehensive interventions involving emission reduction, air quality monitoring, cleaner transportation, and public education are essential to minimize PM2.5 exposure and protect respiratory health.

Keywords: inflammation, mitigation, oxidative stress

INTRODUCTION

Air pollution is one of the leading contributors to illness and premature death across the globe. It can arise from both indoor and outdoor sources, affecting people in homes, workplaces, and cities alike. Even short-term exposure—ranging from a few hours to several days—to certain pollutants has been associated with an increased risk of early mortality.[1,2]

Data from the State of Global Air in 2020 estimated that air pollution was responsible for around 6.67 million deaths worldwide in 2019.[3] In addition, the World Health Organization (WHO) also estimates that each year, air pollution may contribute to between four and nine million deaths, with low- and middle-income countries bearing the heaviest burden.[4]

Air pollution is made up of various harmful substances, including gases,

persistent organic compounds, heavy metals, and tiny particles suspended in the air. Among these, particulate matter (PM) is particularly concerning. These are microscopic dust particles smaller than 10 µm that can be inhaled and settle in the lungs. Of special concern are fine particles, those under 2.5 µm in diameter, which can penetrate deep into the respiratory system, bypassing the body's natural defenses from the cilia all the way to the alveoli.[1,2]

The global issue of air pollution, largely driven by PM, has been intensifying, particularly in industrial areas where PM levels continue to rise. Growing evidence from numerous studies consistently points to a strong connection between exposure to fine particulate matter (PM2.5) and increased risks of death from respiratory diseases, chronic obstructive pulmonary disease (COPD), ischemic heart disease, lung cancer, and stroke.[5] Exposure to PM2.5 can affect the respiratory system by interfering with bronchial mucociliary clearance, which can allow infectious pathogens to enter and damage the epithelial cells lining the airways.[6]

Recent systematic reviews further demonstrate that oxidative stress, chronic inflammation, immune dysregulation, DNA damage, and epigenetic alterations constitute the principal biological pathways through which PM2.5 also contributes to respiratory diseases and lung carcinogenesis. This emphasizes the need for continuous updates of current evidence.[6] Therefore, this literature review provides updated insights and knowledge that can help guide strategies for preventing and managing these health impacts.

This narrative review was prepared by synthesizing evidence from peer-reviewed articles retrieved from PubMed, Scopus, Web of Science, and Google Scholar. The literature search focused primarily on publications from 2022–2026 using keywords including PM2.5, fine particulate matter, air pollution, respiratory diseases, lung cancer, pulmonary infection, oxidative stress, and COPD. Several landmark studies and international guidelines published before 2022 were also included to provide essential scientific background. The selected literature was critically reviewed and synthesized to summarize the current understanding of the respiratory health effects associated with PM2.5 exposure.

PARTICULATE MATTER

Particulate matter refers to tiny particles suspended in the air, made up of a complex mix of solid and liquid substances. These particles vary in size, chemical composition, and origin. Commonly, PM is classified by its aerodynamic diameter, including PM10 (particles smaller than 10 µm), PM2.5 (particles smaller than 2.5 µm), and ultrafine particles (smaller than 0.1 µm), as illustrated in Figure 1.[1,7]

The main components of PM include sulfates, nitrates, ammonium, various inorganic ions, black carbon, crystalline minerals, and trace metals such as copper, cadmium, nickel, zinc, and vanadium.

Additionally, organic matter, microbial material, and allergens significantly contribute to the overall makeup of particulate matter.[1,7]

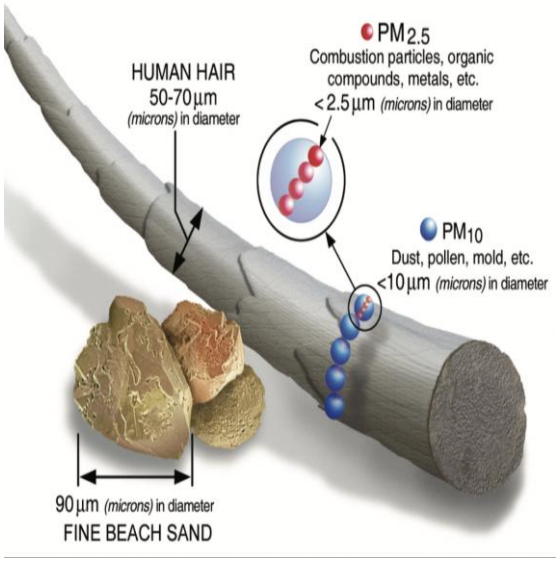


Figure 1. Comparison of Particle Sizes[7]

Specifically, PM2.5 originates from several natural sources, including biomass, sea aerosols, coal combustion, soil and road dust, volcanic dust, and wildfire smoke. Each source contains different chemical components, such as potassium (K), sodium (Na), aluminum (Al), silicon (Si), calcium (Ca), selenium (Se), cobalt (Co), arsenic (As), zinc (Zn), and lead (Pb).[8]

Furthermore, PM exposure can come from diesel emissions, residual agricultural dust, and various industrial processes. The behavior of these particles, shaped by their size, density, temperature, and environmental conditions such as wind speed, can allow them to remain suspended in the air for long periods, worsening overall air pollution.[7,8]

Based on the location, other contributors to PM2.5 exposure may include resuspended dust, emissions from power plants, biomass burning, industrial discharges, sea spray, and exhaust from ships and aircraft.[9] A detailed overview of PM sources, categorized by particle size and origin, is presented in Table 1.

In addition, based on particle types, PM2.5 particles consist of both primary particles, which are emitted directly into the air, and secondary particles, which form through chemical transformations of precursor gases. Primary particles come from natural events like dust storms and wildfires, as well as human activities such as burning fossil fuels, smoking, and biomass combustion. Secondary particles, on the other hand, are created when natural and human-made emissions interact chemically in the atmosphere.[9]

Table 1. Classification of Particles by Size and Source[10]

Particle	Diameter	Source	Formation Origin
Large Particles	<10 µm	---	---
Coarse Particles	2.5–10 µm	Soil suspension (agriculture, mining, unpaved roads) and construction	Mechanical processes such as crushing, grinding, and surface abrasion, as well as spray evaporation and dust suspension
Fine Particles	<2.5 µm	Power plants, oil refineries, forest fires, household fuel combustion, and vehicle emissions	Gas-to-particle conversion through condensation, coagulation (accumulation mode)
Ultrafine Particles	<0.1 µm	Fuel combustion (diesel, gasoline) and exhaust emissions from mobile sources (motor vehicles, aircraft, ships)	Fresh emissions, secondary photochemical reactions (nucleation mode)

Measuring particle mass is the most common method for assessing the impact of PM on respiratory health. Evaluating particle number and size distribution, meanwhile, is often used to study affects PM to the climate. On the other hand, surface area is particularly important for characterizing ultrafine particles, whose mass is usually so small that it is almost negligible.[9]

In terms of size, PM can be measured using four main approaches: particle mass, particle number, surface area, and particle size distribution. Measuring particle mass is the most common method for assessing the impact of PM on respiratory health. For PM10 and PM2.5, the gravimetric method is usually used, which is a quantitative technique for determining particle concentrations in the air.[9,11]

Essentially, the method measures the difference in weight of PM samples before and after collection. These measurements are conducted under controlled conditions, with temperatures maintained between 18–23°C and humidity levels around 45–55 g/m^3. With certain adjustments, the gravimetric approach can also be adapted to evaluate particle size distribution.[9,11]

Meanwhile, particle number and size measurement is commonly used to study affects PM to the climate and, evaluate and classify emissions from vehicles. This method is especially effective for detecting particles in the 0.5–18 µm range, as well as smaller ultrafine particles. One of the most widely used techniques employs Optical Particle Counters (OPCs). These devices work by shining a light, usually from a laser diode, through the air sample and measuring the light scattered by the particles with a photodetector. The method relies on the principle of light scattering to quantify particle numbers.[9]

On the other hand, the surface area of airborne particles is particularly important for characterizing ultrafine particles, whose mass is usually so small that it is almost negligible. These particles can be measured using Nanoparticle Surface Area Monitors (NSAM), which estimate particle surface area by detecting the charge transferred from ions to particles through an electrometer. NSAM is considered a highly reliable method because it can gauge the surface area of particles that are likely to deposit in the lungs.[9]

During the measurement process, the charge transferred from ions to particles generates an electrical current that the electrometer detects. The air sample is divided into two paths, one passing through a filter and the other through an ionizer, allowing the instrument to measure the interaction between charged particles and ions.[9]

EXPOSURE TO PM2.5 AND PATHOPHYSIOLOGICAL RESPONSES IN THE RESPIRATORY SYSTEM

The impact of PM exposure is shaped by local conditions, including topography, meteorological factors, emission levels, particle sources, and environmental characteristics. Smaller particles, especially PM2.5, exhibit greater mobility and can

reach the deeper alveolar regions of the lungs.[7,12]

In contrast, particles larger than 10 µm are typically captured by the body's natural defense systems, such as nasal structures, ciliary action, and mucus filtration. Particles with aerodynamic diameters between 5 and 10 µm typically deposit in the tracheobronchial region, while those ranging from 1 to 5 µm reach the bronchioles and alveoli, the primary sites of gas exchange.[7,12]

Exposure to PM2.5 makes the lungs a primary target organ of toxicity. Its deposition has the potential to cause structural damage and impaired pulmonary function. This has been associated with a heightened risk of respiratory infections, particularly in children, the elderly, and other susceptible populations.[7,8,12]

The adverse effects develop cumulatively because of damage to airway epithelial barriers, impaired mucociliary clearance, altered respiratory microecology with reduced normal airway flora that facilitates pathogen entry, and impaired immune cell function. Such exposure can induce airway inflammation, compromise immune responses, and heighten vulnerability to pulmonary infections.[8,12]

A study by Yang et al demonstrated in a rat model that PM2.5 exposure can impair macrophage activity and slow ciliary movement in the respiratory tract.[13] Val et al also reported that PM2.5 exposure increased the expression of Mucin 5AC (MUC5AC), the primary mucin in the respiratory epithelium, resulting in excessive mucus production and secretion.[14] Furthermore, Yang et al found evidence suggesting that PM2.5 exposure can increase the likelihood of developing respiratory tract infections within a period of 2 to 4 weeks.[12]

As is known, in the respiratory system there exist tight junctions (TJs), which are known as protein complexes that connect adjacent cells and form a pulmonary epithelial barrier that wards off invading pathogens. Maintaining the integrity of these junctions is vital for the airway's natural defense mechanisms, as any disruption can make it easier for pathogens to colonize and increase the risk of lung infections.[8]

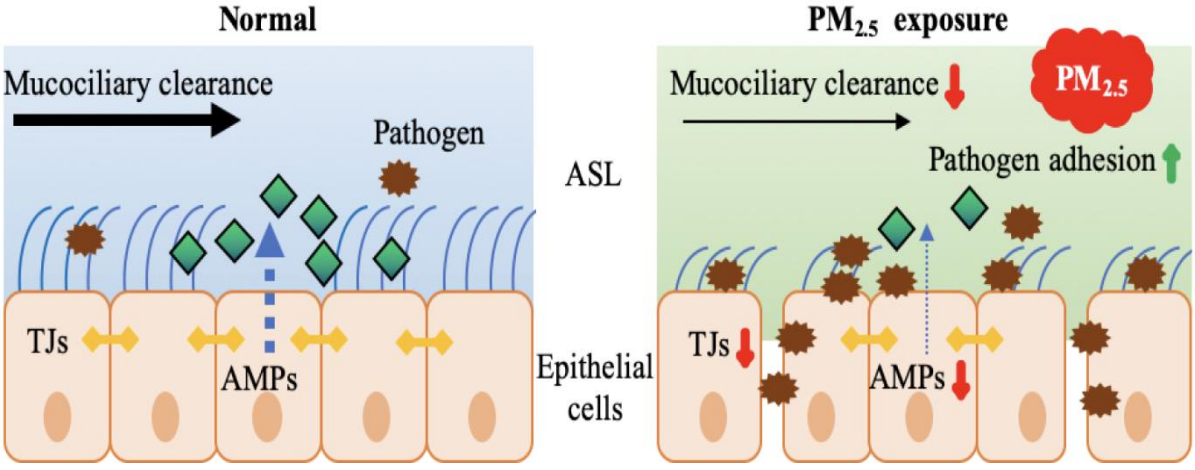


Figure 2. Normal and Abnormal Responses to PM2.5 Exposure[12]

Covering the airway epithelium is a fluid known as Airway Surface Liquid (ASL), which is essential for supporting the respiratory tract's innate immune defenses. ASL's ability to fight off microbes largely depends on antimicrobial peptides (AMPs), such as Salivary Agglutinin (SAG), beta-defensins, lactoferrin, IgA, and surfactant protein D (SP-D). The expression of SAG and beta-defensins through oxidative stress can be reduced, compromising airway defense due to PM2.5 exposure, as illustrated in Figure 2.[8]

Exposure to PM2.5 that induces oxidative stress can impair normal cellular functions and trigger inflammatory responses, activating inflammatory signaling pathways. This is evidenced by elevated levels of T-helper 2 (Th2) cytokines—including interleukin-4 (IL-4), IL-5, and IL-13—in bronchoalveolar lavage fluid, along with increased expression of IL-8, histamine, and leukotrienes.[15]

In addition, PM2.5 also promotes eosinophil infiltration, contributing to airway hyperresponsiveness, and stimulates the release of IL-33, enhancing Th2 immune activity. Furthermore, PM2.5 upregulates IL-6 and IL-1β expression in bronchial epithelial cells, leading to lung injury via modulation of lipid mediators and sphingosine-1-phosphate.[15]

Besides that, PM2.5 exposure has also been shown to disrupt macrophage polarization by promoting type 1 (M1) macrophage responses over type 2 (M2), resulting in a higher M1/M2 ratio. This shift, along with an imbalance between Th1 and Th2 immune responses, favors Th2 dominance and leads to increased production of interferon-gamma (IFN-γ), IL-4, and IL-13, causing immune dysregulation. It is also known that PM2.5 inhibits the differentiation of regulatory T cells (T-reg) and enhances Th17 cell differentiation.[7,12]

These processes impair lung function and may exacerbate obstructive airway diseases by inducing persistent inflammation and oxidative stress. Prolonged exposure is linked to the onset of conditions such as chronic bronchitis, chronic obstructive pulmonary disease (COPD), and asthma.[7,12]

The Impact of PM2.5 Exposure on Obstructive Pulmonary Diseases

Exposure to PM2.5 can negatively affect lung function and accelerate the development of obstructive airway diseases through inflammatory pathways. Long-term exposure leads to ongoing inflammation and oxidative stress, which contribute to conditions such as chronic bronchitis, COPD, and occupational asthma. Research indicates that even a modest rise in PM2.5 levels—about 2.4 $\mu g/m^3$—can reduce forced expiratory volume in 1 second (FEV1) by as much as 101.7 mL. Moreover, a meta-analysis by DeVries et al revealed that for every 10 $\mu g/m^3$ increase in PM2.5, the risk of COPD exacerbations and related hospitalizations rises by 1.4–2.5%.[16,17]

In individuals with asthma, the production of reactive oxygen species (ROS) tends to rise over time, while the lungs' natural antioxidant defenses become

disrupted, leading to an imbalance that can worsen airway inflammation.[18]

Endogenous ROS are produced via mitochondrial respiration, the nicotinamide adenine dinucleotide phosphate (NADPH) oxidase pathway, and the xanthine oxidase system, while external sources of ROS include environmental exposures such as cigarette smoke, ozone, particulate matter, and ionizing radiation. These factors can increase ROS production and trigger the release of proinflammatory cytokines, causing damage to the airway epithelium and pulmonary capillaries.[18]

In addition, the activity of key antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPX), along with levels of non-enzymatic antioxidants such as vitamins C and E, gradually decreases over time in people with asthma. This condition weakens the lungs' defense mechanisms and promotes the release of inflammatory mediators, triggers inflammatory responses, and contributes to the development and advancement of asthma, as depicted in Figure 3.[18]

Meanwhile, in COPD, the inflammatory profile and oxidative stress represent two concurrent biological processes that lead to tissue damage. Short-term PM2.5 exposure can increase inflammatory biomarkers such as Th1 and Th17 cytokines, as well as elevate Th2 cytokines that activate inflammatory responses.[16]

Exposure to PM2.5 also activates dendritic cells, which subsequently migrate to lymph nodes and stimulate specific T cells. The upregulation of proinflammatory cytokines mediated by ROS, including IL-6, IL-8, monocyte chemoattractant protein-1 (MCP-1), and tumor necrosis factor-alpha (TNF-α), promotes inflammation and contributes to pulmonary fibrosis.[16]

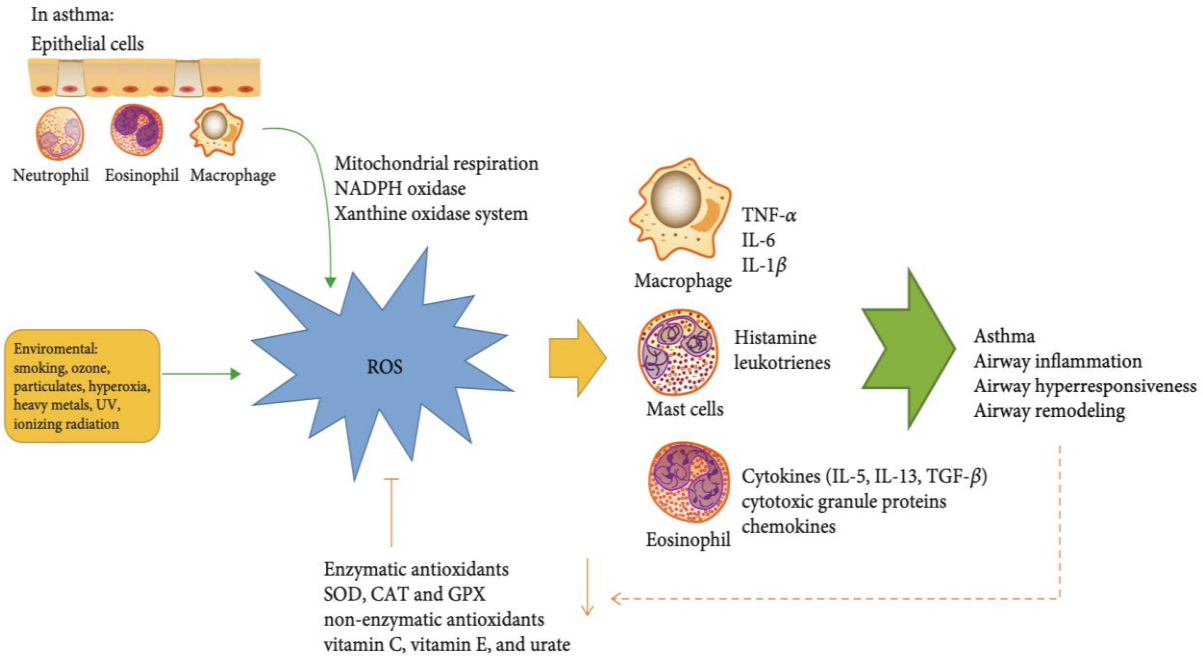


Figure 3. Oxidative and Antioxidant Imbalance in Asthma[18]

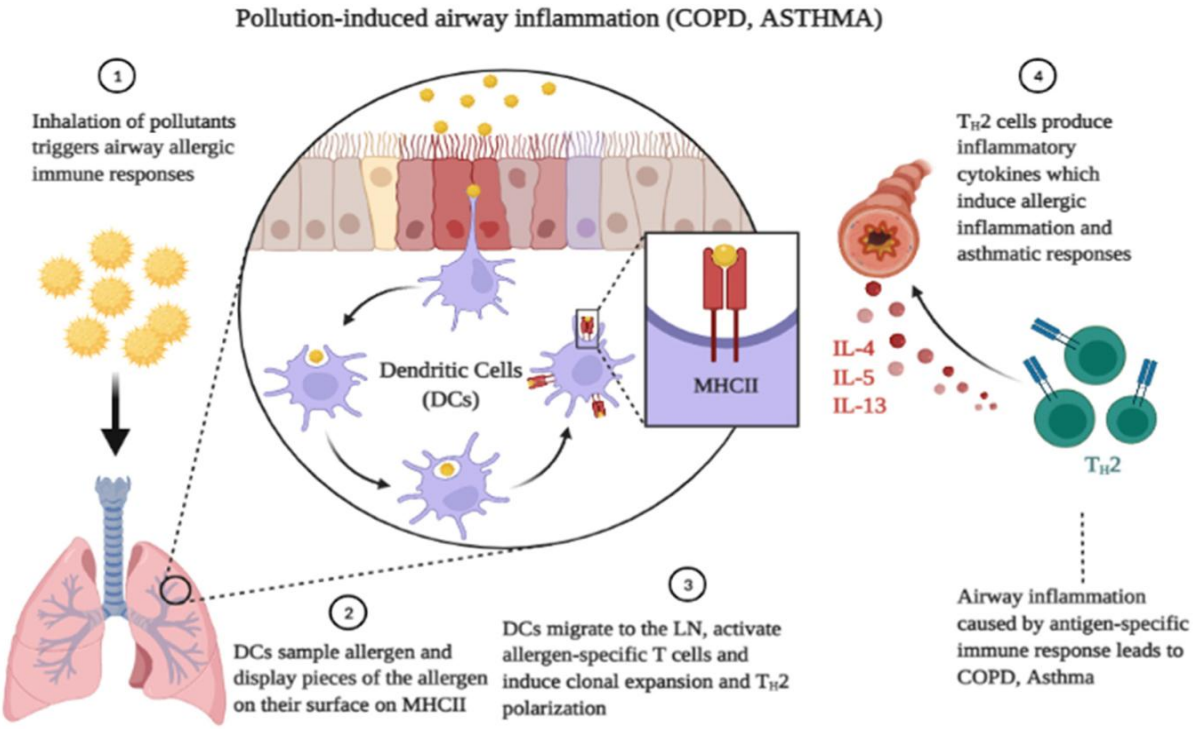


Figure 4. Illustration of Mechanisms Underlying COPD Induced by PM2.5 Exposure[16]

Moreover, PM2.5 exposure also reduces the expression of the 8-oxoguanine DNA glycosylase 1 (OGG1) enzyme in alveolar epithelial cells, thereby impairing the regeneration of damaged type II alveolar epithelial cells, decreasing the number of multilamellar bodies within type II alveolar cells, and leading to collagen accumulation and pulmonary inflammation. This mechanism is illustrated in Figure 4.[16]

Overall, the available evidence consistently demonstrates that long-term PM2.5 exposure plays a significant role in the development and progression of obstructive pulmonary diseases, particularly asthma and COPD. Experimental studies have consistently shown that PM2.5 induces oxidative stress, airway epithelial injury, and chronic inflammation.[16]

Epidemiological studies have also reported associations between increasing PM2.5 concentrations and reduced lung function, higher COPD exacerbation rates, and increased asthma-related hospital admissions. Nevertheless, the magnitude of these associations varies across studies, which may be attributed to differences in PM2.5 composition, exposure duration, population characteristics, smoking prevalence, and methods of exposure assessment.[16]

The Impact of PM2.5 Exposure on Lung Cancer

Both the WHO and the International Agency for Research on Cancer (IARC) identified PM2.5 exposure as a carcinogen in 2013. Elevated levels of particulate matter in the air are linked to a greater likelihood of lung cancer development. Results from the European Study of

Cohorts for Air Pollution Effects (ESCAPE) showed that increases of 10 μg/m^3 in PM10 and 5 μg/m^3 in PM2.5 were associated with 1.22-fold and 1.18-fold elevated lung cancer risks, respectively.[19] Furthermore, a 2015 Korean meta-analysis revealed that each 10 μg/m^3 rise in PM2.5 was related to a 1.09-fold higher risk of developing lung cancer.[20]

In this case, air pollution is linked to carcinogenesis through complex and not yet fully understood biological mechanisms involving abnormal cell proliferation and exposure to mutagenic compounds such as polycyclic aromatic hydrocarbons (PAHs), dioxins, sulfur-containing substances, and 3-nitrobenzanthrone. PAHs can form DNA adducts that, if not repaired by cellular mechanisms, lead to genetic mutations, while TP53 gene mutations caused by PM2.5 exposure affect cell proliferation, apoptosis, and DNA repair.[21]

Exposure to PM2.5 in the long term promotes excessive production of ROS, resulting in epigenetic modifications, including abnormal DNA methylation, histone modification, and dysregulation of microRNA expression, which alter the transcription of genes involved in cell proliferation, apoptosis, angiogenesis, and immune surveillance. Persistent oxidative stress also activates several oncogenic signaling pathways, particularly NF-κB, PI3K/Akt, MAPK, and STAT3, creating a chronic inflammatory microenvironment that promotes tumor initiation and progression.[6,22]

Recent epidemiological evidence consistently supports the association between long-term PM2.5 exposure and increased lung cancer incidence, particularly among never-smokers and individuals living in highly urbanized regions. Nevertheless, the magnitude of risk varies across studies due to differences in PM2.5 chemical composition, exposure duration, population characteristics, smoking status, occupational exposure, and methods used for exposure assessment.[22,23]

PM2.5 should not be regarded as an isolated carcinogenic factor but rather as an important environmental contributor that interacts with genetic susceptibility, lifestyle factors, and co-exposure to other pollutants. Collectively, these findings reinforce the importance of reducing ambient PM2.5 concentrations through comprehensive environmental policies while simultaneously improving individual preventive strategies to reduce cumulative lifetime exposure.[22,23]

The Impact of PM2.5 Exposure on Pulmonary Infections

Exposure to PM2.5 has been associated with the development of pneumonia and with increased incidence and mortality in both children and adults. A meta-analysis reported that each 10 μg/m^3 rise in PM10 and PM2.5 concentrations increases pneumonia incidence by 1.5% and 1.8%, respectively. Another meta-analysis also found that a 10 μg/m^3 increase in PM2.5 significantly raises pneumonia-related mortality in children. However, studies that adjusted for factors such as smoking and bacterial infections

reported no significant association, suggesting that bacterial components in PM2.5 may contribute to pneumonia development.[24,25]

Recent experimental and clinical studies have provided further insight into the mechanisms by which PM2.5 increases susceptibility to pulmonary infections. As is known, fine particulate matter disrupts the integrity of the airway epithelial barrier by reducing the expression of TJs proteins, including zonula occludens-1 (ZO-1) and occludin, thereby facilitating the invasion of pathogenic microorganisms into the lower respiratory tract. In addition, PM2.5 exposure also impairs mucociliary clearance and suppresses the production of antimicrobial peptides such as human β-defensin-1 (HBD1), weakening the first line of innate immune defense.[26]

Fine particulate matter alters macrophage polarization, suppresses phagocytic activity, disrupts airway microbial homeostasis, and enhances the production of pro-inflammatory cytokines.[27] Excessive oxidative stress induced by PM2.5 also activates the NF-κB and MAPK signaling pathways, leading to persistent inflammatory responses while simultaneously reducing the antimicrobial capacity of airway epithelial cells. These alterations increase host susceptibility to bacterial pathogens, including *Pseudomonas aeruginosa*, and contribute to the development of more severe pulmonary infections.[26]

Nevertheless, variations in the magnitude of infection risk among epidemiological studies may be attributed to differences in PM2.5 composition, exposure duration, climatic conditions, population characteristics, smoking status, vaccination coverage, and pre-existing respiratory diseases. Overall, the available evidence supports the conclusion that PM2.5 functions as an important environmental modifier that amplifies host susceptibility to pulmonary infections rather than acting as an independent infectious agent.[28]

PREVENTION AND MITIGATION OF PM2.5

As a preventive measure for air pollution, regulation of PM2.5 concentrations in Indonesia has been established through several government policies that set ambient air quality standards. As stated in Annex VII of Government Regulation of the Republic of Indonesia No. 22 of 2021, the 24-hour standard for PM2.5 is established at 55 $\mu g/m^3$, whereas the annual standard is set at 15 $\mu g/m^3$.[29] Meanwhile, according to the Global Air Quality Guidelines published by the WHO in 2021, the recommended annual average concentration of PM2.5 is 5 $\mu g/m^3$, with a 24-hour mean set at 15 $\mu g/m^3$.[4]

The discrepancy between Indonesia's national standards and the WHO recommendations indicates that Indonesian regulations are relatively more lenient than international guidelines. Therefore, mitigation efforts are necessary to enhance protection of public health. The WHO has recommended a stepwise approach to reduce air pollution, including

PM2.5, through the Interim Target scheme, which provides more realistic goals for countries with high population density. This approach also contributes to the progressive reduction of health impacts and serves as a reference for policymakers to improve air quality.[4]

Prevention and mitigation of PM2.5 require a comprehensive approach encompassing both short-term and long-term measures. One of the primary strategies is strengthening air quality management programs, which includes enhancing air quality monitoring systems and utilizing sensors to identify major sources of PM2.5.[30]

Additionally, modernization of air quality monitoring systems is necessary to ensure real-time detection of PM2.5. Low-cost monitoring networks with calibrated low-cost sensors, combined with advanced correction algorithms, can accurately track PM2.5 concentrations. This is improving exposure assessment by providing approximate reference-grade instruments and expanding routine surveillance capacity, enabling community-level interventions.[30,31]

Control measures are also crucial to reduce atmospheric PM2.5 concentrations and can be achieved through the reduction of fossil fuel use and the shift toward alternative energy sources, including solar, wind, and nuclear power. For instance, projections for the United States indicate that phasing out fossil fuels and tightening air pollution controls could prevent over 6,000 premature deaths annually by 2030.[32]

The WHO further recommends promoting the adoption of clean cooking fuels to reduce household air pollution. According to a joint policy brief issued by the WHO and the University of Liverpool, transitioning from traditional biomass stoves to clean cooking fuels can reduce indoor PM2.5 emissions by approximately 85%, representing a substantially greater reduction than that achieved by improved biomass stoves.[33]

Meanwhile, vulnerable groups, including children, the elderly, and individuals with comorbidities, still require heightened protection against PM2.5 exposure. Health education regarding the prolonged impacts of air pollution, along with early warning systems for deteriorating air quality, constitutes an essential step in mitigating health risks. The use of air purifiers can also help reduce PM2.5 exposure in settings such as homes, schools, and offices.[34]

Furthermore, the use of personal protective equipment, such as N95 or KN95 masks, is effective in reducing inhalation of fine particulate matter when air quality reaches hazardous levels.[34] As is known, masks are an effective form of personal protection against PM, which is divided into procedural/surgical masks and respirators. Surgical masks are designed to filter air exhaled from the wearer's mouth or nose, but their effectiveness is comparable to industrial paper masks used to filter larger particles. Meanwhile, respirators are personal protective equipment (PPE) that reduce the risk from airborne particles, gases, and vapors.[35]

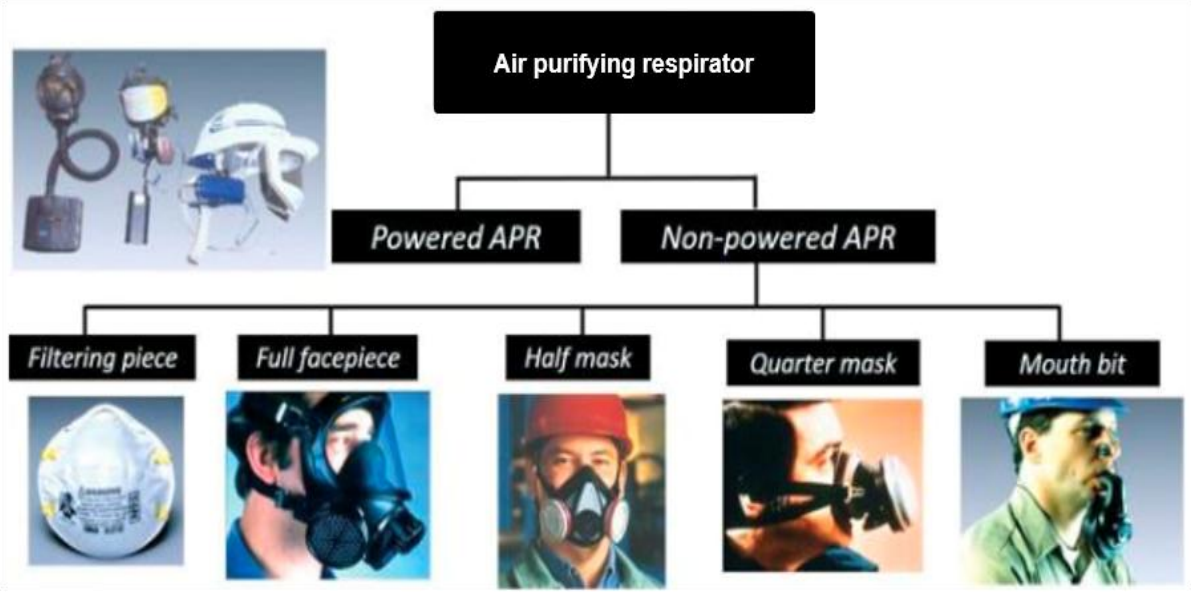


Figure 5. Classification of Air-Purifying Respirator Types[35]

Respirators can be categorized by fit into two types: tight-fitting and loose-fitting. Tight-fitting respirators cover either half or the entire face, with edges designed to create an airtight seal, whereas loose-fitting respirators consist of helmets or hoods that cover the entire face.[35]

Respirators can also be classified according to their mechanism of operation, into air-purifying types and those that deliver air supplied from an external source. Respirators that purify incoming air, such as N95 masks, function by filtering harmful particles from the surrounding air, whereas supplied-air respirators deliver fresh air drawn from an external system, such as an oxygen tank.[35]

Then, air-purifying respirators are divided into two types: powered air-purifying respirators (PAPR) and non-powered air-purifying respirators (non-PAPR). PAPRs use electrical power, such as batteries, to create positive pressure within the mask by drawing, filtering, and delivering outside air into the mask. Non-PAPR types do not have electrical power for air filtration, and the pressure inside the mask is negative. Non-PAPR types include filtering facepieces, full facepieces, half masks, quarter masks, and mouthbits, as shown in Figure 5.[35]

Specifically, the N95 mask, a type of air-purifying respirator (filtering facepiece), can filter 95% of 0.3 μm particles and offers advantages over other respirators in cost and performance. Unlike procedural masks, its filtration is certified by the National Institute for Occupational Safety and Health (NIOSH). The use of N95 masks as PPE is a primary prevention measure against PM2.5 exposure, and proper fit testing is essential for optimal performance.[35]

Beyond individual protective measures, effective mitigation of PM2.5 exposure requires coordinated multisectoral actions involving governments, industries, healthcare professionals, researchers, and local communities. Strengthening environmental regulations, expanding air

quality monitoring systems, promoting renewable energy, and increasing public awareness are essential to achieve sustainable reductions in PM2.5 concentrations. These integrated strategies not only reduce the burden of respiratory diseases but also contribute to broader public health and environmental sustainability goals.[4,30,31,33,36]

CONCLUSION

PM2.5 is one of the most hazardous air pollutants affecting respiratory health because of its ability to penetrate deeply into the lower respiratory tract and induce oxidative stress, chronic inflammation, immune dysregulation, and genetic alterations. The current literature consistently demonstrates that prolonged exposure to PM2.5 contributes to the development and progression of obstructive pulmonary diseases, pulmonary infections, and lung cancer through multiple interconnected biological mechanisms. Although variations exist among epidemiological studies due to differences in exposure characteristics and population factors, the overall evidence strongly supports PM2.5 as a major environmental determinant of respiratory morbidity and mortality. Strengthening air quality management, reducing emissions, promoting clean energy, and implementing effective public health interventions are essential to minimize PM2.5 exposure and improve respiratory health outcomes worldwide.

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