LITERATURE REVIEW

The Impact of Particulate Matter on the Respiratory System

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ABSTRACT

Nowadays, around 50% of the world's population lives in cities and urban areas and is exposed to increasingly higher levels of air pollutants including gaseous pollutants and particulate matter (PM). The latter is a key indicator of air pollution transported into the air by a variety of natural processes and human activities. The particles' size has been directly related to their potential impact on health problems. Particles of concern include small particles with diameters of 2.5 to 10 µm (coarse particles) and smaller than 2.5 µm (fine particles). Exposure to air pollutants over a long period of time not only decompensates pre-existing diseases but also leads to pulmonary and respiratory health problems such as asthma, chronic obstructive pulmonary disease (COPD), and lung cancer even in rural areas. A thorough analysis has to be provided to address the implications for policymakers. Hence, more stringent strategies can be implemented to control air pollution and prevent its health effects.

INTRODUCTION

Globally, air pollution has a detrimental impact on health. Either long-term or short-term air pollution exposures play a role in declining cardiopulmonary functions as well as have a significant impact on quality of life and life expectancy. Studies of air pollution's adverse health effects show that air pollution is currently the biggest environmental risk factor. The Global Burden of Diseases 2017 stated that annually, approximately 5 million deaths are caused by exposure to indoor and outdoor air pollution. Reliable predictions of air pollution exposure and related impacts on health are the keys to informing governments in policymaking and other health and development partners. These are important to implement, monitor, and evaluate policies that help control air pollution and protect health.[1,2] The sustainable development goals (SDGs) are a global plan with 17 goals and 169 objectives to exterminate poverty, inequality, and preserve the environment by 2030. Reducing air pollution is crucial as pollutants and their sources affect climate change. Regulations on air pollution improve air quality and have multiple health benefits. The eleventh SDG goal emphasizes reducing air pollution in urban areas and access to clean fuel energy and household technologies for sustainable development. The third goal uses mortality rates from air pollution as indicators to track progress in improving air quality and safeguarding health. These data indicators are necessary to monitor improvements.[3,4]

Widespread air pollutants encompass carbon monoxide (CO), particulate matter (PM), lead (Pb), ozone (O_3), nitrogen dioxide (NO_2), and sulphur dioxide (SO_2). Particulate PM) is often classified by size—particles measuring 10 µm or smaller are denoted as PM_{10}, while those with a diameter of 2.5 µm or below are termed $PM_{2.5}$. Furthermore, ultrafine particles possessing a diameter under 0.1 µm can be distinguished.[1] PM has various consequences on human health. This review discusses PM's impact on health in depth.

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AIR POLLUTION

Air pollution is defined as the presence of obnoxious materials in the air that can affect humans, animals, and plants. Air pollutants come from primary sources such as industrial and transportation emissions, and secondary sources such as chemical reactions with other pollutants. Primary pollutants include NO_2, SO_2, and CO, while secondary pollutants include O_3, sulphuric acid, and ammonium nitrate aerosols.[5,6]

The harmful impact of gases on the human body is reliant on their solubility in water, concentration, oxidizing capacity, and the body's immunity. Sulphur dioxide has a high solubility in water and primarily affects the skin and upper airways. On the other hand, NO_2 and O_3 are less water-soluble, which allows them to penetrate deeper into the respiratory tract. CO is a non-irritating compound that readily enters the bloodstream, where it competes with oxygen (O_2) binding to hemoglobin, leading to tissue hypoxia. Exposure to an average CO level of 1 mg/m^3 over two days was related to a 1.2% rise in mortality. Nitric oxide (NO) too can bind to hemoglobin and other iron-containing proteins but only for a short period due to its low affinity. Table 1 illustrates the classification of air pollutants.[5]

Table 1. The classifications of air pollutants[5]

Air Pollution	Source
A. Type	
Primary pollutants	Pollutants released directly into the atmosphere such as SO_2, Nitric oxide, CO, and PM.
Secondary pollutants	pollutants that are formed in the air as a result of chemical reactions with other pollutants such as ozone, nitric oxide.
B. Location	
Indoor pollutants	Cooking, particle resuspension, building materials, air conditioning, smoking, heating, biological agents Examples: tobacco, smoked wood, CO, CO_2, aldehydes, alcohols, alkanes, ketones, microbial agents, organic dust, radon, artificial vitreous fibers.
Outdoor pollutants	Industrial, urban, regional, agricultural, natural Examples: SO_2, Ozone, Nitric oxide, CO, PM.
C. Form	
Gas	SO_2 gas, Nitric oxide, Ozone, dioxin, benzene, aldehyde, 1,3-butadiene
Particle	PM

Particulate Matter

PM is an air pollutant composed of solid and fluid particles floating in the air, which varies in composition by location. PM includes sulphate, nitrate, ammonium, inorganic ions, carbon black, crustal materials, and metal particles such as copper, cadmium, nickel, zinc, and vanadium, as well as organic components such as microbial substrate and allergen. In a meta-analysis by Levy, *et al*. (2010), it was approximated that an increase of $PM_{2.5}$ concentration by 10 µg/m^3 was correspondent with a 0.7% increase in mortality rate. Further research is needed to identify the factors influencing the relationship between PM-related deaths.[1]

PM is a complex mix of suspended particles which are known as suspended particulate pollutants. Based on their aerodynamic diameter, PM is categorized into two groups, namely coarse particles (diameter >2.5 µm) and fine particles (diameter <2.5 µm) (Figure 1). The smaller particles are formed by the conversion of gases to particles and contain aerosols, organic vapours, and condensed metals. Conversely, the coarse particles include material from the earth's crust, road, and industrial dust. Fine particles have an acidic property and mutagenic activity. The dimension of the particles suspended in the atmosphere varies from nanometres to tens of micrometres. The coarse fraction, which is the largest particle, is mechanically produced by breaking down larger solid fractions. These particles are generated from farming processes, uncovered soil, unpaved roads, or mining activities. Air turbulence from road traffic allows the recirculation of dust on the highway, and evaporation of seawater on beaches can generate large particles. Plant and insect fragments, mould spores, and pollen also contribute to the formation of large particles. The forces required to disintegrate particles into smaller sizes increase with lesser size.[7]

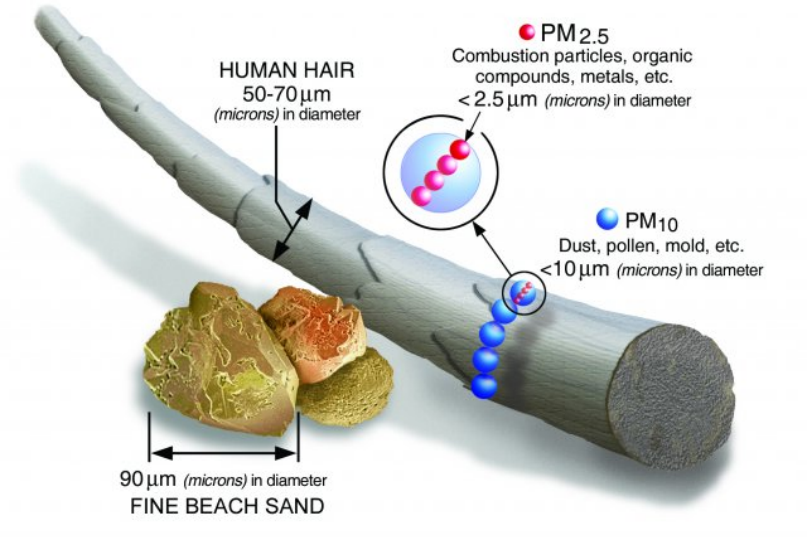

Figure 1. Comparison of PM size[8]

Particles with an aerodynamic diameter <0.1 µm are generated through nucleation and condensation from low-pressure vapour materials created by intense heat evaporation or chemical reactions in the atmosphere. On the surface of previously existing particles, resultant particles will coagulate and form larger particles, condense gas, or vapour molecules. Greater particle concentration enhances coagulation efficiency, while a larger surface area is necessary for efficient condensation of particles. However, when particle size exceeds about 1 µm, the efficiency of particle coagulation and condensation decreases since these processes cannot form particles beyond that size.[7]

There are four methods for measuring PM, the gravimetric principle, the microbalance principle, the beta light attenuation principle, and the light scattering principle (Figure 2). The gravimetric principle involves collecting particles on a filter by drawing air through it using a pump. Centrifugal force impaction is used to eliminate larger particles before filtration. Weighing of filters is conducted before and after sampling under defined situations. Mass concentration is determined by dividing particle mass by the volume of air that was drawn through the pump. A stable room temperature and humidity are required for weighing. A device with high resolution which is 1 or 10 µg is needed for measurements.[9]

The microbalance principle provides sustained data by measuring the mass build-up of PM on a filter placed on a perforated element that oscillates as air is drawn through it. Turnover in oscillation frequency determines mass concentration, which is stored in memory (Figure 2). However, the conventional configuration of the microbalance principle heats the air sample to 50°C, causing the PM material to evaporate and leading to an underestimation of the true PM concentration. Both methods have their own advantages and limitations and should be chosen based on specific research requirements.[9]

To determine the concentration of particles in the air, different devices use various principles, such as light scattering and beta-ray attenuation. In light scattering, a laser beam illuminates the air sample and a detector measures the level of light dispersion due to the particles present. The signal is converted into mass concentration using calibration constants. This device has an internal pump, data storage, and a battery for continuous data collection. Some devices can measure multiple mass portions simultaneously or count particles in different size channels. Beta-rays emitted from radioactive (Kr gas or ^{14}C) will be filtered and collected with a beta-ray attenuation device. The attenuation of the beam passing through the filter is a measure of the PM mass build-up, which is stored in the device's memory. Overloading can occur while separating PM_{10} and $PM_{2.5}$ based on impaction, resulting in an overestimation of actual concentrations. The measurement tool for PM is shown in Figure 2.[9]

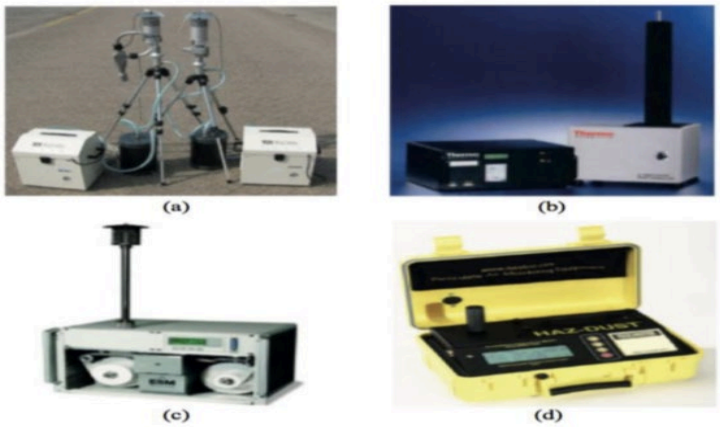

Figure 2. (a) Gravimetric principle, (b) Microbalance principle, (c) Beta-ray attenuation principle, (d) Light scattering principle[9]

The government of the Republic of Indonesia shows concern about indoor air sanitation by regulating pollution threshold via the Minister of Health of the Republic of Indonesia which regulates PM through 1077/MENKES/PER/V/2011, stating $PM_{2.5}$ threshold values of 35 μg/m^3 in 24 hours and PM_{10} ≤70 μg/m^3 in 24 hours.[10] The threshold value for the permissible concentration of air pollution in outdoor air for $PM_{2.5}$ is 65 μg/m^3 and for PM_{10} is 150 μg/m^3. European regulations regarding the PM threshold value for outdoor air concentration mean PM_{10} limit per day should not exceed 50 μg/m^3, the value found maximally in 35 days/year. Daily average outdoor PM_{10} concentration annually should not be beyond 40 μg/m^3. Belgian regulations state the outdoor exposure limits for $PM_{2.5}$ and PM_{10} are 3 and 10 μg/m^3 over an 8-hour period.[10] Difference in the regulation between Indonesia and other countries is pertinent to the years in which the laws were ratified and scientific evidence up to that date.

$PM_{2.5}$ and PM_{10} were studied by Chow, *et al*. (2022) in California.[11] Carbon and organic elements make up 70-80% of the total mass of $PM_{2.5}$ together with nitrate, sulphate, and ammonium ions which make up about 10-20% of $PM_{2.5}$ and PM_{10}.[6] Approximately 40-50% of coarse particles are made up of aluminium, silicon, sulphur, potassium calcium, and iron. The composition of $PM_{2.5}$ and PM_{10} was studied by Israel, *et al*., in Berlin and control sites in rural areas. The average concentration of $PM_{2.5}$ and PM_{10} were 39 μg/m^3 and 58 μg/m^3, respectively. The same component found in California was 78% for fine particles as in a study conducted by Israel, *et al*., while for coarse particles the proportion of 50% was found to be similar. Similar particle composition data in the air were also reported from the UK.[11,12]

PM components exhibit temporal and spatial variability, which makes it uncertain whether research data from different countries can be used universally. In Indonesia, the dry season from April to September is characterized by cold and dry air with minimal rainfall, while the rainy season from October to March is marked by warm and wet air brought by winds. In January and February, the rainfall is maximum with an average total of over 400 mm, while the minimum total rainfall of less than or equal to 70 mm occurs in August and September. Studies in Indonesia and Malaysia have shown that PM concentrations are higher during the dry season than the rainy season. In Malaysia, PM concentrations are higher from May to September due to the southwest winds, while during the rainy season from December to March, the PM concentration is at a minimum level. High rainfall and small soil-based inversion have been identified as the main causes of low PM concentrations during the rainy season. Therefore, seasonal variability needs to be considered while analyzing PM components in different regions.[13]

In a 10-year study of PM components in Jakarta, Muhayatun, *et al*. (2020) found that sulphur and black carbon had the highest concentrations in $PM_{2.5}$, while $PM_{2.5-10}$ was dominated by earth's crust elements.[14] Bogor and Tangerang had high concentrations of lead in $PM_{2.5}$ due to lead rod production and battery recycling. In Surabaya, the earth's crust elements, especially silica from volcanic eruptions, were found at high levels in PM. Additionally, lead levels in $PM_{2.5}$ and $PM_{2.5-10}$ in Surabaya were significantly higher compared to other cities in East Java.[14,15]

Impact of Particulate Matter on Health

PM exposure is associated with various adverse health effects, including exacerbation of cardiopulmonary disease, decreased lung function, and premature death. Studies suggest that it may also lead to low birth weight, preterm delivery, and fetal/infant mortality. Inhalation of $PM_{2.5}$ may cause symptoms such as chest pain, coughing, wheezing, and breathing difficulties. Studies from America have also shown an association between exposure to PM and diabetes incidence in adults with specific ethnic and obesity risk

factors. Elderly, children, and individuals with pulmonary heart disease are specifically susceptible to the detrimental effects of PM exposure.[16,17]

Studies have shown that exposure to PM can have negative effects on children's lung development. Exposure to $PM_{2.5}$ levels exceeding 65 μg/m^3 for 24 hours increases children's risk of developing respiratory symptoms, frequent use of asthma medications, and declining lung function. Children exposed to PM_{10} at the threshold of 150 μg/m^3 have been shown to experience a 3-6% decline in lung function. Additionally, asthmatic children in Guadeloupe have a higher risk of emergency room visits due to Saharan dust containing PM_{10} and $PM_{2.5}$ Saharan dust. A study in Baltimore found a statistically significant association between PM exposure and asthma symptoms in either atopic or non-atopic asthmatic children aged 2 to 6 years old.[16]

Exposure to PM is related to an increased risk of hospitalization due to heart failure exacerbation and myocardial infarction. Chow, *et al.* (2022) stated that PM exposure increases plasma viscosity, causes endothelial dysfunction, and triggers acute phase reactions, resulting in changes in cardiac autonomic control.[11] PM also promotes atherosclerotic progression, increasing the risk of cardiac events. A study conducted in Los Angeles discovered that an increase of 10 μg/m^3 in $PM_{2.5}$ was associated with a rise in carotid intima-media thickness. Bell, *et al*. showed short-term exposure to $PM_{2.5}$ significantly influenced cardiovascular and respiratory hospital admission rates. Another study in Boston revealed that $PM_{2.5}$ exposure reduced vagal tone and variability of heart rate.[16]

Many studies have recently explored the relationship between air pollution and cognitive function decline by evaluating cognitive scores in adults aged 50 years old and above who are regularly exposed to air pollution. Ailshire and Clarke discovered that elderly Americans who lived in areas with high $PM_{2.5}$ levels had a 1.5 times greater error rate on memory and orientation tests than those living in areas with lower $PM_{2.5}$ levels. Weuve, *et al*. also reported that elderly women exposed to $PM_{2.5}$ were more likely to experience a greater decline in global cognitive function than those with less exposure. They concluded that a 10 μg/m^3 rise in long-term PM exposure was equivalent to around two years of cognitive aging.[18]

Impact of Particulate Matter on the Respiratory System

The efficacy of PM exposure is influenced by various local factors like topography, weather, emitted concentrations, source of particles, and environment. Smaller particles travel faster and penetrate deeper into the respiratory tract. The human nose, cilia, and mucus filter properties trap particulates larger than 10 μm diameter. Coarse particles tend to settle in the trachea or bronchi and trigger the body to sneeze and cough. A study by Phipps, *et al*. observed two sets of mice exposed to air pollution and cigarette smoke, then injected with *Streptococcus pneumoniae* intratracheally five weeks later. The bacterial count in the lungs of the exposed mice was four times higher within 24 hours and 35 times higher within 48 hours compared to the control group. Similarly, a study in China revealed that air pollution damages, erases, and interferes with the tracheal cilia of mice, making them susceptible to secondary infection. Jalava, *et al*. cultured airborne particles retrieved from six European cities in macrophages from rats for 24 hours and found reduced viability of alveolar macrophages significantly with exposure of $PM_{0.2\text{-}2.5}$ in the range 300 pg/mL to 150 g/mL. [16,19]

Particles with a diameter of less than 10 μm provide the most harmful consequence on human wellbeing. These particles can penetrate the alveoli due to their high penetration power. Figure 3 shows particles with aerodynamic diameter 5-10 μm settle in the tracheobronchial while particles with aerodynamic diameter 1-5 μm deposit in bronchioles and alveoli where gas exchange occurs. These particles participate in the ventilation, diffusion, and perfusion processes causing substantial health problems. In general, particles with a diameter <1 μm have similar properties to gas molecules. They allow penetration to alveoli and can move distally into the cell tissue or circulatory system.[20]

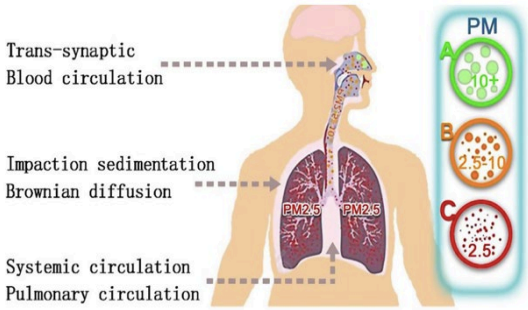

Figure 3. Particle size and sedimentation[20]

Metals from PM are mediators of airway injury through the Fenton reaction, i.e., a catalytic process altering the hydrogen peroxide products of mitochondrial oxidative respiration into highly toxic hydroxyl free radicals.[21] Metals from PM, especially iron, increase free radicals in cells. Free radicals lead to cell and tissue damage and exacerbate inflammatory reactions. An animal study revealed a relationship role of dissolved transition metals in PM-induced lung injury in vivo.[16]

Lung Functions and Particulate Matter Exposure

Lung function is a key indicator of respiratory health, which starts to decline after the mid-20s despite improving from birth. Exposure to air pollution, particularly PM, has been relaed to a decline in lung function in adults. The study on air pollution and lung disease in adults (SAPALDIA) monitored 9,651 individuals aged 18-60 years old over 11 years and found that exposure to PM was associated with a decline in forced vital capacity (FVC) by 3.4% and forced expiratory volume in 1 second (FEV1) by 1.6%. Similarly, the study on the influence of air pollution on lung function, inflammation and aging (SALIA) found that an increase in 10 $\mu g/m^3$ PM_{10} concentration led to a 4.7% and 3.4% decrease in FVC and FEV1, respectively. The impact of PM exposure on lung function is significant, but other factors such as age, individual susceptibility, other pollutants, and genetics still need further investigation.[22]

Immunopatophysiology of Particulate Matter

The respiratory system is adversely affected by $PM_{2.5}$ due to the oxidation of free radicals that cause damage to lung cells. $PM_{2.5}$ is rich in manganese, copper, iron, zinc, lipopolysaccharides, and polycyclic aromatic hydrocarbons, which can increase free radical production and cause oxidative stress in the lungs. The organic components, metals, and reactive oxygen compounds present in $PM_{2.5}$ trigger the production of free radicals, which oxidize the lung cells leading to lung damage. Hydroxyl radicals are produced by activating metals, and these radicals cause oxidative damage to deoxyribonucleic acid (DNA), which can lead to carcinogenesis, teratogenesis, mutagenesis, and other permanent damage.[19]

In addition, $PM_{2.5}$ can interfere with calcium balance in the respiratory system. Calcium is an important messenger regulating cell function. High levels of calcium activate an inflammatory cascade, leading to cell damage. $PM_{2.5}$ produces free radicals excessively and reduces cells' antioxidant capacity, resulting in lipid peroxidation in cell membranes and an increase in intracellular calcium concentration. This increase can further increase the production of free radicals. Xing, *et al*. have demonstrated that overexpression of calcium-sensitive receptors is associated with cell apoptosis and necrosis. Mechanism of cell damage induced by $PM_{2.5,}$ postulated to be free radical-mediated regulation of intracellular calcium concentrations.[19]

$PM_{2.5}$ induces the overexpression of genes responsible for inflammatory injury and cytokine production. Studies have shown that exposure to $PM_{2.5}$ leads to an increase in T cells, neutrophils, mastocytes, eosinophils, and mastocytes in the bronchoalveolar fluid. $PM_{2.5}$ also influences two types of alveolar macrophages, M1 and M2. M1 macrophages are associated with inflammation, while M2 macrophages impede inflammation. Human alveolar macrophages associated with $PM_{2.5}$ produce more M1 macrophages than M2, inducing the migration of immune cells to the lungs and other tissues and releasing more inflammatory cytokines and chemokines. This suggests that cytokines can induce the migration of neutrophils, T cells, and eosinophils to the lungs and other tissues, thereby releasing more inflammatory cytokines and chemokines, which leads to the synergistic damage of lung cells. The role of $PM_{2.5}$ in damaging human health is currently a focus of several ongoing studies.[19]

Neutrophils and macrophages in alveolar cells are stimulated by cytokines and chemokines to envelop foreign molecules through phagocytosis, which are then transported by the mucociliary system. PM activates the distribution of inflammatory mediators, such as IL-6, GM-CSF, TNF-α, and IL-8, leading to oxidative stress, innate immunity, and respiratory disease exacerbation. Exposure to PM increases cell death due to reactive oxygen compounds, mitochondrial membrane potential disruption, and human endothelial cell activation of NF-kB. Oxidative stress and cytokine stimulation activate signal transmission pathways, leading to the activation of transcription factors such as NF-kB and AP-1. AP-1 is a protein dimer that plays a role in immune and inflammatory genes in oxidative stress-mediated disease.[23]

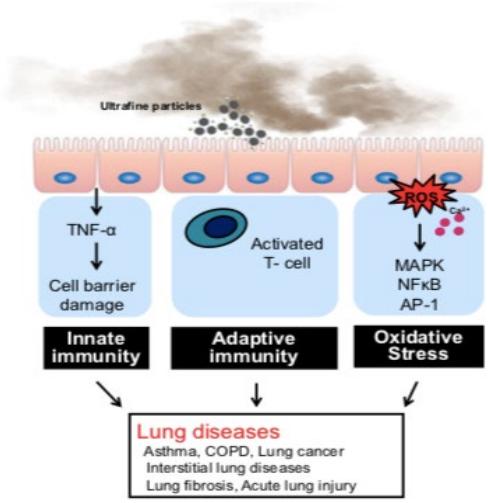

Figure 4. Immunomechanism of PM[23]

Exposure to PM activates NF-kB and activation protein 1, leading to cytotoxicity through apoptosis. Ex vivo smoke exposure decreases phagocytosis and increases IL-6 and IL-8 cytokine production in human macrophages. PM releases protein kinase B molecular mediators, signal transducers, mitogen-activated protein kinase (MAPK), and transcription-1 activators that cause chronic inflammation in cells, tissues, and the respiratory system. It also alters T cell response, inducing Th-2 and excess production of IL-4 and IL-13, leading to asthma.[20]

Particulate Matters and Asthma-Chronic Obstructive Pulmonary Disease

Various studies have investigated the relationship between PM exposure and obstructive pulmonary disease (asthma and chronic obstructive pulmonary disease/COPD). In patients with COPD, exposure to PM was related to increased hospitalization rates and mortality. A meta-analysis conducted in Korea found that incremental PM_{10} concentration by 10 $\mu g/m^3$ increased the hospitalization rate of COPD patients by 2.7%. Similarly, a PM_{10} increase of 10 $\mu g/m^3$ was found to elevate the COPD mortality rate by 1.1%. Exposure to PM is also related to allergic sensitization and exacerbation of asthma. Asthmatic patients exposed to PM have increased airway hyperresponsiveness and increased exhaled nitric oxide fraction (FeNO), which leads to decreased lung volume and pulmonary diffusion capacity. Exposure to $PM_{2.5}$ that leads to plasma levels of 10 $\mu g/m^3$ increases the incidence of emergency room, outpatient, and hospital visits. These results emphasize the importance of reducing exposure to PM to prevent and manage COPD and asthma.[22]

Particulate Matter and Interstitial Lung Disease

An international study in 2014 found a relationship between PM and idiopathic pulmonary fibrosis (IPF), with PM containing O_3 and NO_2 associated with an increased risk of acute IPF exacerbation six weeks after first exposure. Another investigation found a 5 $\mu g/m^3$ increase in PM_{10} exposure level reduced the FVC of IPF patients by 46 ml. The 2018 Cohorte Fibrose (COFI) study in France demonstrated that 10 $\mu g/m^3$ concentrations increase in $PM_{2.5}$ and PM_{10} increased IPF mortality rates by 7.93 and 2.01-fold, respectively. These findings indicate exposure to higher levels of PM significantly reduces pulmonary function, leading to increased fatality rates and acute exacerbation of IPF.[22] A study observing the effect of $PM_{2.5}$ on interstitial lung disease (ILD) showed that $PM_{2.5}$ exposure of 8 $\mu g/m^3$ increased the risk of mortality (HR 4.40 (95% CI, 3.51-5.51)).[24] Exposure to PM induces ILD through various mechanisms, such as increased production of oxidative stress, shortening of telomere, and parenchymal inflammation involving TGF-ß.[25]

Particulate Matter and Lung Cancer

In 2013, the World Health Organization (WHO) and the International Agency for Research on Cancer (IARC) declared PM as a carcinogenic agent and associated increased levels of PM in the atmosphere with elevated risk of lung cancer. The European Study of Cohorts of Air Pollution Effects (ESCAPE) found that every increase of PM_{10} and $PM_{2.5}$ levels by 10 $\mu g/m^3$ and 5 $\mu g/m^3$ increased risk for lung cancer by 1.22-fold and 1.18-fold, respectively. A 2015 meta-analysis conducted in Korea revealed that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ concentration led to a 1.09-fold increase in the risk of lung cancer. The association between PM_{10} levels and lung cancer events was weaker compared to $PM_{2.5}$ levels. Smoking, in combination with exposure to higher levels of $PM_{2.5}$, was found to increase the risk of developing lung cancer. PM pollution is estimated as responsible for around 500,000 lung cancer deaths internationally. Preventive measures such as controlling PM formation, reducing PM exposure, and smoking cessation are essential in reducing lung cancer incidence and mortality rates.[22]

Particulate Matter and Pneumonia

Airway inflammation is increased by PM exposure, which leads to an elevation in inflammatory mediators and neutrophils. In addition, elevation of serum 8-isoprostane in bronchoalveolar lavage fluid or sputum was observed. In individuals, either children or adults, who are exposed to PM, the incidence and mortality rate due to pneumonia is increased. A meta-analysis demonstrated that an additional 10 $\mu g/m^3$ rise in PM_{10} and $PM_{2.5}$ concentration increased the rate of pneumonia in children by 1.5% and 1.8%, respectively.

Other meta-analysis reported that every increase of 10 $\mu g/m^3$ in $PM_{2.5}$ significantly increased overall mortality rate for respiratory disease.[22]

SUMMARY

Air pollution has an adverse impact on the health sector around the world. Suspended particulate pollutants are referred to as PM. PM measurement can be performed in four ways, using the gravimetric principle, the microbalance principle, the beta light attenuation principle, and the light scattering principle. The mass and composition of PM are divided into two main groups, namely coarse particles, and fine particles. Coarse particles settle quickly and tend to settle in the trachea or bronchi and fine particles enter the alveoli. PM increases the risk of pneumonia in children, risk of hospitalization and mortality in COPD, risk of cancer, risk of asthma exacerbation, and risk of developing coronary heart disease and stroke. PM also affects respiratory symptoms, particularly in patients with a history of respiratory diseases. WHO sets the daily pollution threshold (24 hours) for $PM_{2.5}$ to be a maximum of 15 $\mu g/m^3$ in a 24-hour period and for PM_{10} of 45 $\mu g/m^3$.

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Conflict of Interest
The authors declared there is no conflict of interest.

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Authors' Contributions
All authors contributed and approved the final version of the manuscript.

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