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Exposure to Particulate Matter Affects Lung Function and Performance: A Literature Review

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ABSTRACT: Air pollution has emerged as a significant peril to public health on a global scale, with around five million deaths occurring each year as a direct result of air pollution. The respiratory system mostly absorbs air contaminants, which then enter the alveoli and circulate in the bloodstream. Exposure to particulate matter (PM), the primary pollutant, can lead to acute inflammation of the respiratory tract and exacerbate pre-existing chronic lung inflammation. The extent of damage produced by particulate matter (PM) is determined by the manner in which the particles enter the lungs, including factors such as their aerodynamic diameter, ambient air velocity, and respiratory rate. Additionally, the manner in which the particles depart the lungs, involving processes such as nostril hair, mucociliary clearance, and macrophage phagocytosis, also plays a role in determining the damage caused. During periods of low air quality, particulate matter (PM) can negatively impact the immune system by causing damage to the bronchial mucociliary system. This damage makes it harder for the body to eliminate pathogens and leads to the release of inflammatory cytokines, which can harm lung epithelial cells and fibroblasts. Additionally, it inhibits intercellular communication within the gaps of the epithelial barrier, hence reducing its efficacy as a protective barrier for the lungs. The detrimental effects of PM can be attributed to oxidative stress caused by free radicals. PM has the ability to directly stimulate macrophages, leading to inflammation and an increase in the production of reactive oxygen species (ROS). Reactive oxygen species (ROS) impair antioxidant defenses and inflict damage on DNA, proteins, carbohydrates, and lipids. Lung function will decline as a result of bronchoconstriction, pulmonary edema (which leads to thickening of the alveolar capillaries), and fibrosis (which restricts lung expansion) caused by epithelial thickening. Based on the findings of many research, it may be inferred that an excessive influx of particles can lead to an excessive burden on the lungs, consequently impairing the respiratory system's defense capability and potentially resulting in lung damage. These investigations have established that higher levels of exposure to particulate matter are linked to a larger likelihood of reduced lung function. On the other hand, decreasing the amount of these particles that one is exposed to will reduce the danger.

INTRODUCTION

Air pollution is a significant threat to global public health, reducing the average human lifetime worldwide by 20 months[1]. Exposure to air pollution can heighten the likelihood of developing a range of illnesses, including heart disease, stroke, asthma, bronchitis, pneumonia, chronic obstructive pulmonary disease (COPD), lung cancer, and other health issues. Additionally, the average human lifespan can be shortened due to the combined impact of outdoor pollution and fine particle pollution (PM2.5) from household activities such as cooking with wood or other solid fuels. The effect on human life expectancy is substantial in comparison to other significant illnesses. In 2015, air pollution-related diseases resulted in almost nine million premature deaths, which accounted for 16% of all global deaths. The majority of health consequences resulting from air pollution are observed in fast advancing poor and medium income nations, such as India and China. The results demonstrate that enhancements in worldwide air quality can result in extended and more robust lifespans in numerous regions across the globe[2,3].

A variety of natural phenomena, such as volcanic eruptions and forest fires, have the ability to generate air pollution. In addition to these natural processes, human combustion activities and industrial machinery can generate air pollution. The swift growth of industry and the utilization of technology have generated pollution sources that can diminish air quality and have an adverse effect on our well-being. The extensive urban development involving activities such as land clearance, earth excavation, operation of diesel engines, use of hazardous chemicals, and vehicular movement generates particles on a daily basis. These particles result in the release of carbon monoxide, carbon dioxide, nitrogen oxides, and hydrocarbons through combustion, all of which significantly contribute to air pollution. Research conducted in the industry has shown that poor air quality leads to

significant healthcare expenses, material and equipment damage, and decreases in productivity. Several air pollutants that have an impact on human health include carbon monoxide (CO), nitrogen oxides (NOx), sulfur oxides (SOx), hydrocarbons, and particle matter (PM).

Particulate matter (PM) is a type of air pollutant that originates from natural sources including volcanoes and forest fires, as well as human activity include office spaces, industrial operations, and cars. Particulate matter (PM) refers to solid and liquid particles that are present in the air. These particles can be classified based on their size, specifically their aerodynamic diameter. PM2.5 refers to particles with a diameter of less than 2.5 µm, PM10 refers to particles with a diameter of less than 10 µm, and ultrafine particles or nanoparticles are particles with a diameter of less than 100 nm. In 2017, 2.9 million deaths were attributed to ambient PM2.5, 1.6 million deaths were caused by home air pollution, and 472,000 premature deaths were linked to ozone[1]. Cardiovascular disorders like ischemic heart disease and stroke, along with other conditions such as respiratory infections, lung infections, and chronic obstructive pulmonary disease, are responsible for a higher global mortality rate compared to parasitic diseases, vector-borne diseases, HIV infection, smoking, and diseases caused by violence[4].

The respiratory system takes in these airborne contaminants, which subsequently traverse the alveoli and enter the bloodstream. Several research studies have consistently found a significant association between air pollution and respiratory diseases. Exposure to PM can lead to immediate inflammation and worsen long-term lung inflammation, such as pulmonary hypertension, cardiovascular problems, and autoimmune disorders. Scientists have identified multiple mechanisms by which particulate matter (PM) impacts the respiratory system. This encompasses the secretion of molecules such as extracellular protein kinase/protein kinase B, mitogen-activated protein kinase (MAPK), signal transducer and activator of transcription (STAT)-1, and pathogenic antibodies that attach to proinflammatory cell receptors, exacerbating chronic inflammation[5]. Analysts forecast a rise in the adverse effects of PM in the forthcoming years. Particulate matter (PM) toxicity specifically affects the lungs, which are vital organs in the respiratory system. This leads to inflammation of the respiratory system, which disrupts the usual immune response and increases vulnerability to respiratory infections[4]. Lung function serves as a measure of respiratory well-being, and air pollution, particularly exposure to PM, is linked to a decline in lung function. Hence, it is imperative to examine the impact of particulate matter on lung function in order to potentially mitigate or surmount the diverse health consequences associated with it.

DISCUSSION

Air pollution, which includes ambient PM2.5, home ozone, and ozone, has emerged as a worldwide health concern. In 2017, air pollution was responsible for approximately 4.9 million fatalities worldwide, as estimated. The ten countries with the largest death load in 2017 are China (1.2 million), India (1.2 million), Pakistan (128,000), Indonesia (124,000), Bangladesh (123,000), Nigeria (114,000), the United States of America (108,000), Russia (99,000), Brazil (66,000), and the Philippines (64,000). There exists compelling scientific evidence that inhaling PM2.5 can result in ischemic heart disease, cerebrovascular disease (including ischemic and hemorrhagic strokes), lung cancer, COPD, and lower respiratory tract infections, particularly pneumonia. PM2.5 exposure accounts for 5.2% of total global mortality. Since 1990, there has been a 68% rise in PM2.5-related deaths, with significant increases occurring in 1990 and 2010, followed by a slow increase thereafter[1].

Particulate Matter (PM)

The United States Environmental Protection Agency (US EPA) employs the term "particulate matter" (PM) to refer to solid particles and liquid droplets present in the air, which exhibit diverse composition and size. These particles consist of dust, dirt, soot, smoke, and droplets. PM10 refers to particles that have a diameter of 10 micrometers or less, while PM2.5 refers to particles that have a diameter of 2.5 micrometers or smaller. These particles can enter the lungs and travel through the bloodstream. They are commonly used as indicators of air pollution. PM2.5, also known as fine particles, exhibit prolonged atmospheric persistence and can travel extensive distances, spanning hundreds of kilometers. Conversely, PM10, or larger coarse particles, lack the ability to endure in the air and are prone to deposition on the ground[6].

These air pollutants can induce acute inflammation in the human respiratory system and worsen pre-existing chronic lung inflammation, such as pulmonary hypertension, cardiovascular illness, and autoimmune diseases. The respiratory system is divided into two functional zones: the conduction zone and the respiratory zone. The conduction zone is responsible for delivering air to the lower respiratory tract without any diffusion process. It includes the nose, pharynx, larynx, trachea, bronchi, and terminal bronchioles. On the other hand, the respiratory zone is where gas diffusion takes place. It includes the respiratory bronchioles, alveolar ducts and sacs, as well as the alveoli. The primary role of the respiratory system is to supply oxygen for cellular requirements and eliminate carbon dioxide generated by bodily cells. This function is executed through the mechanisms of ventilation, oxygen and carbon dioxide diffusion, perfusion, gas transportation, and respiratory regulation. During the ventilation

process, we extract not only oxygen but also any substances present in the air. Inhaled air comprises several components present in the atmosphere, including harmful substances in the form of particulate matter (PM)[7,8].

Particulate matter (PM) toxicity predominantly affects the respiratory system, specifically the lungs. Multiple investigations have demonstrated that particulate matter (PM) is responsible for inducing respiratory impairments in human lung function. Individuals with inflammatory respiratory disorders such as COPD and asthma may experience exacerbation of respiratory symptoms, including wheezing, coughing, and shortness of breath, as a result of brief exposure to pollution. The research findings indicated a notable increase in wheezing, shortness of breath, bronchitis symptoms, and reduced lung function among individuals residing within a 100-meter radius of a heavily trafficked main road. A study conducted on a group of 224 individuals from a Chinese community revealed a notable correlation between the overall short-term exposure to PM2.5 and long-term exposure to polycyclic aromatic hydrocarbons (PAHs), resulting in a decline in lung function[9,10].

Host Defense System

The level of damage caused by exposure to particulate matter (PM) is determined by the process of particle deposition and particle clearance in the respiratory tract. The deposition of particles is influenced by the aerodynamic force and the properties of the particles, with the effectiveness of deposition being determined by their size. The particle's size determines its shape and weight, which in turn influence coagulation, dispersion, sedimentation, and implication. The velocity of the air and the frequency of respiration both impact the deposition. Changes in the diameter of the respiratory tract can potentially enhance the deposition of particles. Exposure to irritants in patients with chronic bronchitis and pneumonia leads to bronchoconstriction, resulting in increased deposition of particles[11]. Particle deposition occurs by several methods, including interception, inertia impaction, diffusion, sedimentation, and electrostatic forces. Airflow waves induce inertia impaction, a phenomenon in which airborne particles settle within the airway lumen. Interception is the occurrence when particles move in close proximity to the lumen and adhere to the airway lumen. Interception occurs exclusively in narrow tracks, allowing particles to come into contact with the respiratory lining. Interception is a significant process for the deposition of particles that have irregular forms and fiber-like structures. Sedimentation is the process by which particles are deposited as a result of gravitational forces, causing them to migrate downward continuously. This typically occurs in the narrow airways of the respiratory system where the flow of air is sluggish, particularly in the bronchioles and alveoli. The process of sedimentation has a negligible impact on the settling of particles that have aerodynamic dimensions smaller than 0.5 µm. Deposition in particles of extremely small sizes occurs by diffusion. The deposition mechanism occurs by diffusion, which is caused by the random motion of particles known as Brownian motion[8].

The respiratory system employs various defense mechanisms to remove particles, including physical defenses such as upper respiratory tract filtration, sneezing reflex, coughing, and mucociliary movement, as well as cellular defenses such as phagocytosis by alveolar macrophages and immunological neutrophils. When particles are transported by fast-moving air, they typically collide with the nasal cavity and become trapped in the upper respiratory tract, particularly particles larger than 10 µ. The sneeze and cough reflex serve to expel particles from the upper respiratory tract through forced expiration, preventing them from reaching the lungs. Certain particles, particularly those with a size less than 2u, typically have the ability to bypass these protective barriers and enter the lower respiratory tract. Multiple pathways are activated as particles reach the lower respiratory tract. Initially, the mucociliary escalator will transport particles that are stuck in the fluid of the conduction and impaction tract upwards into the tracheobronchial tree. Furthermore, macrophages have the ability to ingest particles and eliminate them through the mucociliary escalator. If a particle is able to enter the alveoli, it will be eliminated by alveolar macrophages via the lymph or blood circulation. Macrophages exhibit fast phagocytosis, with over 50% of particles being ingested within three hours and close to 100% within 24 hours. Alveolar macrophages have the ability to engulf and eliminate certain tiny, insoluble particles by phagocytosis, and then eliminate them through the lymphatic system. In the lymphatic system, certain epithelial and interstitial cells may retain particles that have not been eliminated by macrophages. These particles might persist for months to years before being cleaned[8,12].

Mechanism of Lung Damage

When the air quality is poor, particulate matter (PM) can adversely affect the immune system through many mechanisms. For instance, smoking disrupts the bronchial mucociliary system, impeding the bacteria's ability to eliminate itself. It triggers the release of inflammatory cytokines, which can lead to the death of lung epithelial cells. Additionally, it hinders intercellular communication within the epithelium, weakening its barrier function and compromising the lungs' innate defenses. In addition, there are several mechanisms that can interfere with the normal physical and immunological function of lung surfactant, hinder the natural killer (NK) cell responses, impair the ability to fight bacteria, disrupt the expression of toll-like receptors (TLRs), and affect the structure of microtubules, all of which can inhibit the process of alveolar macrophage phagocytosis. These many pathways will diminish lung immunity and render it vulnerable to lung illness[4].

The detrimental effects of PM can be attributed to oxidative stress caused by free radicals. Oxidative stress refers to a state of imbalance between oxidants and antioxidants, resulting in harmful effects on lipids, proteins, and DNA. Oxidative stress occurs when there is an unequal level of reactive oxygen species (ROS) generation and antioxidants, which serve as the defense mechanism that restricts harm caused by free radicals. Environmental stressors, including as UV radiation, ionizing radiation, pollutants (PM), heavy metals, xenobiotics, and antibiotics, contribute to the generation of reactive oxygen species (ROS), which are byproducts of oxygen metabolism. Exposure to PM will directly stimulate macrophages, leading to inflammation. Additionally, it will enhance the generation of reactive oxygen species (ROS) via NOX and mitochondria, leading to impairment of your antioxidant defense and disruption of your mitochondria. Oxidative stress leads to the impairment of DNA, proteins, carbohydrates, and lipids. Before the identification of lipid peroxidation and protein damage, DNA is the main focus of attention. DNA is a resilient molecule that can undergo spontaneous degradation at a specific point in time. Nevertheless, the presence of reactive oxygen species (ROS) and reactive nitrogen species (RNS) will expedite the occurrence of DNA damage through oxidative stress. Excessive exposure to oxidative stress can lead to cellular apoptosis. Elevated activation of poly (ADP-ribose) polymerase (PARP) can lead to a depletion of intracellular NAD+/NADP+, resulting in the cell's inability to produce ATP and subsequent cell death[7,14].

Respiratory tract injury reaction

The respiratory system's health is primarily assessed by lung function, which serves as the most crucial sign. Studies indicate that air pollution, specifically exposure to particulate matter (PM), might cause a decline in lung function, resulting in a higher occurrence of compromised lung function in adults. A cohort study conducted in Switzerland examined the effects of fine dust on adult respiratory illness. The study involved 9,651 adults aged 18-60 over a span of 11 years. The researchers observed a 3.4% decrease in the average force vital capacity (FVC) and a 1.6% reduction in the average force expiration volume in one second (FEV1). Lower concentrations of PM10 over the research period were linked to a reduced occurrence of impaired lung function. A study conducted in Germany examined the impact of a 10 µg/m3 increase in PM10 levels between 1985 and 1994 on lung function, inflammation, and aging. The study revealed a decrease of 4.7% in FEV1, a decrease of 3.4% in FVC, and a decrease of 1.1% in FEV1/FVC. The preceding explanation illustrates that being exposed to PM can have an adverse impact on lung function. Nevertheless, implementing environmental enhancements to minimize exposure to PM should decelerate this process.

Particulate matter can induce damage to the respiratory tract, leading to either an immediate or long-term reaction. Activation of the irritant receptor (TRPA1) can elicit a trigeminal nerve reaction, leading to an immediate airway reflex characterized by sensations like tickling, itching, or discomfort. In addition, particles that enter the respiratory system will induce bronchoconstriction in the airway as a result of the autonomic nerve response that controls the tone of the bronchial smooth muscles. Irritants also trigger an autonomic response by lowering the threshold for bronchoconstriction. Bronchoconstriction induces the tightness of the airway, resulting in a reduction in airflow. Bronchoconstriction can also be linked to excessive mucus secretion, resulting in symptoms such as coughing, wheezing, quick and shallow breathing, difficulty breathing, soreness below the sternum, and shortness of breath. In addition to causing bronchoconstriction, irritants also activate TRPA1 and TRPV1, leading to the development of neurogenic inflammation. The rupture of alveolar endothelium and epithelial cells results in acute lung damage and the accumulation of fluid in the lungs, known as lung edema. Additionally, the influx of inflammatory cells leads to the breakdown of surfactant, further contributing to lung edema and the collapse of lung tissue, known as atelectasis. Acute lung injuries will impair the process of breathing, where the circulation of blood through the lungs is crucial for effective exchange of gases. Pulmonary edema can lead to the thickening of the alveolar capillaries, so restricting the exchange of oxygen and carbon dioxide[11].

Chronic responses, such as chronic obstructive pulmonary disease (COPD), can be triggered by respiratory tract trauma. COPD is a condition characterized by progressive airflow restriction resulting from thermal, osmotic, and chemical responses. Disruption arises from an augmentation in goblet cells, a reduction in cilia cells, and a substantial enlargement of the submucosa gland. These processes result in an augmentation of mucus production, a reduction in the ability of the cilia to clear the airways, causing blockage, worsening viral or bacterial infections, and a fall in the FEV/FVC ratio, which cannot be reversed by a bronchodilator. COPD is a chronic inflammatory illness of the respiratory tract that specifically impacts the small airways and is characterized by a gradual and permanent restriction of airflow. Chronic Obstructive Pulmonary Disease (COPD) currently ranks as the fourth most common cause of death globally. Projections indicate that by 2030, it will rise to become the third leading cause of death, making it a significant public health concern. The occurrence of this can be attributed to the fact that the likelihood of developing COPD is influenced by the expansion of the lungs during childhood and adolescence, until the lungs reach their maximum capacity in early adulthood. Exposure to air pollution throughout childhood and adolescence is linked to a decline in lung function in older individuals[15,16].

Doiron et al. (2017) investigated the correlation between exposure to air pollution, lung function, and chronic obstructive pulmonary disease (COPD) in persons between the ages of 40 and 69. The study found that increased levels of air pollutants were linked to a decrease in FEV1 and FVC, a lower FEV1/FVC ratio, and an elevated risk of COPD. The study's results corroborated the findings of prior research, demonstrating that adults who are exposed to elevated levels of air pollution experience a notable deterioration in lung function and are more susceptible to developing chronic obstructive pulmonary disease (COPD). Prolonged exposure to particulate matter (PM) can result in bronchiolitis obliterans, a medical disorder characterized by the obstruction of airways due to the thickening of injured epithelial tissue. Bronchiolitis obliterans can manifest with symptoms like cough, wheezing, difficulty breathing, and exhaustion, along with fever, weight loss, night sweats, and a persistent decline in FEV1 lasting for at least three weeks. Lung fibrosis is a condition when there is an excessive accumulation of collagen in the interstitial alveolar, centriacinar, and pleura, resulting in a reduced capacity of the lungs to expand and contract. Consequently, this diminishes the capacity of the lungs by decreasing Forced Vital Capacity (FVC), with or without alterations in Forced Expiratory Volume in one second (FEV1). Multiple studies have also documented instances of industrial dust exposure leading to respiratory issues. The study conducted by Younes revealed that the average frequency of silica dust among workers exceeded the maximum limit by 6.3 times. Furthermore, all indices of lung function were considerably reduced among workers who were exposed to silica. The study conducted by Lakhwinder revealed that the exposed group had significantly reduced spirometry measures, including FVC, FEV1, FEV1/FVC ratio, FEF25–75, PEFR, PIFR, and FIVC, compared to the control group[17,18,19].

Chi-Hsien Chen's study revealed a negative correlation between FVC and fine particles (PM2.5), indicating a decrease in lung function. Additionally, the study discovered that FEV1, FEF25–75, FEF25, and FEF50 were also declining, which was associated with the presence of coarse particles (PM2.5–10). An interquartile range (IQR) rise of 10μg/m3 in PM2.5 resulted in a drop of forced vital capacity (FVC) by about 106.38 ml, which corresponds to a reduction of 4.47%. An interquartile range (IQR) rise of 7.29μg/m3 in PM2.5–10 resulted in a drop of 91.23 ml (4.85%) in forced expiratory volume in one second (FEV1) and a decrease of 104.44 ml/second (5.58%) in forced expiratory flow between 25% and 75% of the forced vital capacity (FEF25–75%). Elderly individuals who were exposed to PM2.5 over an extended period of time had decreased lung strength and vital capacity. Furthermore, PM2.5–10 exerts a more pronounced detrimental impact on the conduction airway compared to PM2.5[22]. According to a study conducted by Giulia Paolocci, there is a correlation between the emission of PM10 particles from a port and a decrease in lung function (specifically FEV1 and FEV1/FVC) in individuals who are healthy but have little exposure to these particles[3]. The study conducted by Siti Aisyah revealed that the process of installing bricks resulted in the emission of PM2.5 particles, with the highest average concentration recorded at 79.98 μg/m3. This was followed by concrete mixing and wall plastering, which produced concentrations of 78.42 μg/m3 and 72.57 μg/m3, respectively. The individuals who put wall plaster exhibited the greatest mean Peak Expiratory Flow Rate (PEFR), which measured 343.65 L/minute. The average PEFR (Peak Expiratory Flow Rate) for brick installation was 329.26 L/minute, whereas for concrete mixing it was 298.62 L/minute[23].

Thet Wai Oo found that textile workers in the yarn twist department had the greatest level of dust exposure, resulting in a 40.1% decrease in lung function overall. The occurrence of decreasing Forced Vital Capacity (FVC), Forced Expiratory Volume in one second (FEV1), and the ratio of FEV1 to FVC was 36.7%, 34.3%, and 3.9%, respectively. Workers in the twisting department who had more than 5 years of work experience and were exposed to dust showed a decrease in Forced Vital Capacity (FVC) and Forced Expiratory Volume in one second (FEV1)[24]. Helmy's study revealed variations in lung function between traders in the industrial region of Manyar sub-district and the Sunan Giri religious tourism sector of Gersik Regency. In the expansive industrial zone, characterized by a dust concentration of 0.86, it was discovered that a significant proportion of traders (76.9%) had limitations, while only 15.4% of traders had an unimpeded functional condition. Meanwhile, in areas of religious tourism with dust levels measuring 0.017, it was shown that the majority of traders (90.9%) had a normal physical condition, while just 9.1% had limitations[28]. In a study conducted by Cintya et all, it was discovered that traders who were exposed to inhaled dust levels of 3 mg/m3 or higher had a significantly elevated risk, 1,757 times larger, of developing lung function abnormalities[29]. Anjani discovered a significant correlation (p=0.014) between the amounts of breathed dust and the occurrence of pulmonary function abnormalities in workers at PT Marleny Jepara furniture industry. A study revealed that 82.6% of participants who were exposed to dust levels over 1 mg/m3 suffered from respiratory impairments. This figure exceeds the percentage of respondents, specifically 28.6%, who had lung function abnormalities and were exposed to inhaled dust levels of ≤1 mg/m3. If the spirometry results indicate a %FVC value below 80% or a %FVC value below 75%, it is considered problematic[30]. Downs were discovered during the 11-year follow-up period, there was a decrease in the average exposure to individual house outdoor PM10, with a median fall of -5.3 micrograms per cubic meter and an interquartile range of -7.5 to -4.2. Using mixed-model regression analyses, while accounting for confounders, baseline PM10 concentrations, and clustering within areas, we observed significant negative associations between the reduction in PM10 and the rate of decline in FEV1 (P=0.045), FEV1 as a percentage of FVC (P=0.02), and FEF25-75 (P=0.001). Over an 11-year period, a decrease of 10 micrograms of PM10 per cubic meter resulted in a 9% reduction in

the annual rate of decline in FEV1 and a 16% reduction in the yearly rate of decline in FEF25-75. The relationships between cumulative exposure during the interim between the two assessments were similar[31].

Based on the findings of these many research, it may be inferred that an excessive influx of particles can lead to an excessive burden on the lungs, consequently impairing the respiratory system's defense capability and potentially resulting in lung damage. These investigations have established that higher levels of exposure to particulate matter are linked to a larger likelihood of reduced lung function. On the other hand, decreasing the amount of these particles that one is exposed to will reduce the danger. In this case, it is crucial to take into account the presence of threshold values that are determined based on scientific evidence and health-related criteria.

SUMMARY

Lung function serves as a reliable measure of the state of our respiratory system. Exposure to particulate matter (PM) in the air has been linked to a decrease in lung function. Inhaled particles can induce acute inflammation and exacerbate chronic inflammation in the respiratory system. Exposure to particulate matter (PM) can disrupt the respiratory defense system by impairing the bronchial mucociliary system, so hindering the ability of germs to eliminate themselves. The release of the inflammatory cytokine will induce apoptosis in both epithelial cells and fibroblasts in the lungs, disrupt intercellular communication, increase the permeability of the epithelial layer, and impair its ability to defend the lungs' innate immune system. Particulate matter (PM) also obstructs the process of phagocytosis carried out by alveolar macrophages by interfering with the physical functioning and normal immune response of the lungs, particularly the surfactant system. The substance hinders the response of natural killer (NK) cells and reduces their ability to fight against microbes by interfering with the transport of Fe3+ through transferrin. Additionally, it alters the expression of toll-like receptors (TLRs) and the structure of microtubules. Exposure to particulate matter (PM) will directly stimulate macrophages, leading to the induction of inflammation and increased production of reactive oxygen species (ROS) through the activation of NOX and mitochondria. This can disrupt the body's antioxidant defense mechanism and impair the proper functioning of mitochondria. This leads to harm to the targets of DNA, protein, carbohydrate, and fat, resulting in oxidative stress and various health issues. Exposure to PM (particulate matter) can result in either an acute or chronic reaction, resulting in a decrease in lung function over time. Multiple studies have demonstrated that exposure to particulate matter (PM) can impair the lungs' capacity to expand and contract, leading to a decrease in lung function as indicated by a reduction in forced vital capacity (FVC), with or without alterations in forced expiratory volume in one second (FEV1). Multiple studies have shown that deteriorating lung function is negatively affected, although the reduction of exposure to particulate matter (PM) can help minimize this decrease. These findings suggest that implementing various measures to decrease exposure to PM will aid in safeguarding our respiratory well-being.

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