# PARTICULATE MATTER

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## I. INTRODUCTION

Particulate Matter (PM) is one of the six criteria pollutants, and the most important in terms of adverse effects on human health. The marked increase in mortality that occurred during air pollution episodes

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in the small town of Donora, Pennsylvania, in 1948 (20 deaths) and the London fog of 1952 (4000 deaths), provides evidence of the impact that this pollutant has on human health. Since this date, many epidemiological studies of PM health effects have been completed. These studies showed that particulate matter, especially particles that are smaller than 10 microns ($PM_{10}$), are likely to cause adverse health effects including increasing morbidity and mortality in susceptible individuals. These results influenced governments to establish strategies to control air pollution. Air quality guidelines and standards were developed in an attempt to reduce adverse impacts on human health and the environment. The US National Ambient Air Quality Standards (NAAQS) established in 1971 included a the first standard for Total Suspended Particulate Matter (TSP). In 1987, the TSP standard was replaced with $PM_{10}$ standard (150mg/$m^3$ 24-h average and 50mg/$m^3$ averaged annually), and in 1997, a standard for particles less than 2.5 micrometers in diameter ($PM_{2.5}$) was established (65mg/$m^3$ 24-h and 16mg/$m^3$ annual). Despite the significant improvements made in air quality over the last three decades, PM continues to exert a public health impact.

## II. DEFINITION

Particulate matter (PM) is the term used for a mixture of solid particles and liquid droplets suspended in the air. These particles originate from a variety of sources, such as power plants, industrial processes, and diesel trucks, and they are formed in the atmosphere by transformation of gaseous emissions. Their chemical and physical compositions depending of location, time of year, and weather.(Figure 1). Particulate matter is composed of both coarse and fine particles.

Coarse particles ($PM_{10}$) have an aerodynamic diameter between 2.5µ m and 10µ m. They are formed by mechanical disruption (e.g. crushing, grinding, abrasion of surfaces); evaporation of sprays, and suspension of dust. $PM_{10}$ is composed of aluminosilicate and other oxides of crustal elements, and major sources including fugitive dust from roads, industry, agriculture, construction and demolition, and fly ash from fossil fuel combustion. The lifetime of $PM_{10}$ is from minutes to hours, and its travel distance varies from <1km to 10 km.

Fine particles have an aerodynamic diameter less than 2.5µ m ($PM_{2.5}$). They differ from $PM_{10}$ in origin and chemistry (Table 1). These particles are formed from gas and condensation of high-temperature vapors during combustion, and they are composed of various combinations of sulfate compounds, nitrate compounds, carbon compounds, ammonium, hydrogen ion, organic compounds, metals (Pb, Cd, V, Ni, Cu, Zn, Mn, and Fe), and particle bound water. The major sources of PM2.5 are fossil fuel combustion, vegetation burning, and the smelting and processing of metals. Their lifetime is from days to weeks and travel distance ranges from 100s to >1000s km.. In addition, fine particles are associated with decreased visibility ( haze) impairment in many cities of the U.S

Table 1. Comparisons of fine and coarse particle matter

| | Fine Particle | Coarse Particle |
|---|---|---|
| Formed from | Gases | Large solids/droplets |

| | | |
|---|---|---|
| Formed by | Chemical reaction; nucleation; condensation, coagulation; evaporation of fog and cloud droplets in which gases have dissolved and reacted | Mechanical disruption (e.g. crushing, grinding, abrasion of surfaces); evaporation of sprays; suspension of dusts |
| Composed of | Sulfate ($SO_{42}^{-}$); Nitrate ($NO^{-}_{3}$); ammonium ($NH_{4+}$); hydrogen ion ($H+$); elemental carbon; organic compounds (e.g. PAHs, PNAs); metals (Pb, Cd, V, Ni, Cu, Zn, Mn, Fe); particle bound water | Resuspended dust (e.g. soil dust, street dust); coal and oil fly ash; metal oxides of crustal elements (Si, Al, Ti, Fe); CaCO3, NaCl, sea salt; pollen, mould spores; plant/animal fragments; tyrewear debris |
| Solubility | Largely soluble, Hygroscopic and deliquescent | Largely insoluble and non-hygroscopic |
| Sources | Combustion of coal, oil, gasoline, diesel, wood; atmospheric transformation products of NOx, $SO_2$ and organic compounds including biogenic species (e.g. terpenes); high temperature processes, smelters, steel mills, etc. | Resuspension of industrial dust and soil tracked onto roads; suspension from disturbed soil (e.g. farming, mining, unpaved roads); biological sources; construction and demolition; coal and oil combustion; ocean spray |
| Lifetimes | Days to weeks | Minutes to Hours |
| Travel distance | 100s to 1000s of kilometers | <1 to 10s of kilometers |

Source: EPA (1996)

## III. PARTICULATE MATTER STANDARDS

In 1971, the United States Environmental Protection Agency (EPA) established the first PM National Ambient Air Quality Standard. The original PM standard was Total Suspended Particulate (TSP). This standard was replaced in 1987 with particulate matter less than 10 µ m in aerodynamic diameter ($PM_{10}$) and specified an annual average concentration of 50µ g/$m^3$ and a 24-h maximum of 150µ g/$m^{3,}$ based on the highest value over 3-year period. In 1997, after reviewing scientific studies, the EPA concluded that particles with aerodynamic diameter less than 2.5µ m ($PM_{2.5}$) had a greater association with mortality and morbidity rates than $PM_{10}$. On this bases, the EPA established an annual $PM_{2.5}$ standard level of 15µ g/$m^{3,}$, and a 24-h $PM_{2.5}$ standard level of 65µ g/m. Also, the EPA established the Pollutant Standard Index (PSI) as an air pollution alert system to communicate to the general public information about the health risks of PM and other pollutants.

## IV. PHYSICAL PROPERTIES AND PHYSIOLOGICAL EFFECTS

The capacity of particulate matter to produce adverse health effects in humans depends on its deposition in the respiratory tract. Particle size, shape, and density affect deposition rates. The most important characteristics influencing the deposition of particles in the respiratory system are size and aerodynamic properties. The aerodynamic diameter of a particle is the diameter of a unit density sphere having the same settling velocity as the particle in question, whatever its size, shape or density. Particles between 2.5 and 10µ m in aerodynamic diameter correspond to the inhalable particles capable to be deposited, in the upper respiratory tract. Particles with aerodynamic diameter smaller than 2.5µ m (PM2.5), called fine particles, correspond to the respirable particle fraction capable of penetrating the alveolar region of the lung. Inhaled particles come in contact with surface of the respiratory system. These particles pass the proximal airway (throat and larynx) of the respiratory tract, and deposit in the tracheobronchial conductive airway of the lungs (bronchial and bronchiolar airway) or in the gas exchange region (respiratory bronchioles, alveolar ducts, and alveoli of the lung parenchyma).

There are five mechanisms that influence particle deposition within the respiratory tract. The primary mechanisms are gravitational settling, impaction, and Brownian diffusion. Secondary mechanisms are electrostatic attraction and interception. These last two processes have minimal importance for inhalation and deposition of particulate matter. Deposition by gravitational settling occurs as a result of the influence of gravity on particles suspended in the air. The settling rate of particles is directly proportional to particle size. This process is most important in the distal region of the bronchial airway and in proximal portions of the gas exchange region. Another mechanism of particle deposition is impaction. Due to inertia airborne particles do not follow changes in direction or speed of airflow and they may impact on the wall of the airway. This mechanism occurs primarily in the throat and larynx with particles larger than 3µ m and increases with increasing particle size. Brownian diffusion involves collision between gas molecules and micrometer-sized particles, which push the particle in an irregular manner. It depends on the diffusive or thermodynamic diameter of the airborne particle rather than on the aerodynamic diameter. Due to this, Brownian diffusion increases with decreasing particle size. This mechanism is predominant in the gas exchange alveolar region of the lung for particles smaller than 0.5µ m.

There are other factors that also influence particle deposition, including mode of breathing (oral breathing permit the passage of particles greater than 10µ m to the lung), physical activity (exercise), age, lung diseases (chronic obstructive lung disease), and ambient conditions (increase in temperature or the presence of other pollutants).

The ability of the lung trying to protect itself against inhaled particles, clearance, will determine the adverse health effects of particulate matter. There are two clearance mechanisms: the mucociliary system and the alveolar macrophages. Particles deposited in the ciliated region of the tracheobronchial airway, rise on the mucociliary ladder to be expelled by coughing or swallowing. Particles deposited on the terminal bronchioles are cleared by lung macrophages. An early cellular response to an acute particulate exposure is damage to epithelial cells of respiratory tract, which also produce many different types of inflammatory mediators. The local pulmonary inflammation induced by $PM_{10}$ could impact on the cardiovascular system via the local production of procoagulant factors in the lung or as a result of the effects of mediators released from the lungs which act on the liver, to increase the levels of procoagulant factors which could promote myocardial infarction.

## V. HEALTH EFFECTS

Several epidemiological studies have linked both $PM_{10}$ and specially $PM_{2.5}$ with significant health problems, including: premature mortality, chronic respiratory disease, respiratory emergency room visits and hospital admissions, aggravated asthma, acute respiratory symptoms, and decreased lung function. Like the other criteria pollutants, the elderly, whose physiological reserves decline with age and who have higher prevalence of cardiorespiratory conditions, and children, whose respiratory systems' are still developing and who spend more time outdoors, are most at risk from exposure to particulate matter. Also, individuals with preexisting heart or lung disease and asthmatics are sensitive to PM effects. Fine particulate pollution ($PM_{2.5}$), is of specific concern because it contains a high proportion of various toxic metals and acids, and aerodynamically it can penetrate deeper into the respiratory tract.

PREMATURE MORTALITY

Historically, the association between $PM_{10}$ and mortality has been manifested in many air pollution episodes such as those which occurred in Belgium (1930), Pennsylvania (1948), London (1952), New York (1953), and London (1962), where the number of deaths attributed to air pollution was 63, 20, 4000, 200 and 700, respectively. Several studies have demonstrated the relationship between low concentrations of $PM_{10}$ and $PM_{2.5}$ and increase in daily mortality. A study conducted by Pope, et al., 1996, demonstrated the association between $PM_{10}$ air pollution and cardiopulmonary and lung cancer mortality. The relationship was stronger for $PM_{2.5}$ than $PM_{10}$. $PM_{2.5}$ was associated with a 36% increase in death from lung cancer and 26% in cardiopulmonary deaths, the risk being higher for people over the age of 65. $PM_{10}$ was not associated with lung cancer death (3,8,9,18,19). In addition, another study conducted by Ostro demonstrated the association between $PM_{10}$ levels with Sudden Infant Disease Syndrome (SIDS) (34).

CHRONIC RESPIRATORY DISEASE

Epidemiological studies have showed the relationship between $PM_{10}$ exposure and an increase in bronchitis, chronic cough, and respiratory symptoms in persons with COPD (1,23,36).

RESPIRATORY AND HOSPITAL ADMISSION

In a number of studies, investigators have observed an increased incidence of respiratory diseases in association with $PM_{10}$ air pollution. For example, in a study conducted in the United Kingdom, an association between emergency hospital admissions for respiratory and cardiovascular disease and $PM_{10}$ was found (2). Another study conducted in Seattle, Washington, demonstrated association with emergency room visits for asthmatics and $PM_{10}$ air pollution (27). Also, $PM_{10}$ was associated with an increase in hospital admission of the elderly for COPD and asthma and lower respiratory tract infections including bronchitis and pneumonia (2,4,28). In addition, a study conducted in Canada by Burnett et al., in 1980, found that increases of 10mg/$m^3$ in $PM_{10}$ and $PM_{2.5}$ were associated with 1.9% and 3.3% respective increases in respiratory and cardiac hospital admission. The relationship was strongest between $PM_{2.5}$ and cardiac disease (5,7,8,14).

AGGRAVATED ASTHMA

Persons with asthma, especially children, are more susceptible to PM air pollution. Recent studies have associated $PM_{10}$ at low concentrations with an increase in bronchodilator and asthma medication use (27,36). The relationship between $PM_{10}$ air pollution and asthma is stronger than for $PM_{2.5}$ (6).

RESPIRATORY SYMPTOMS

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A series of analyses adults and children demonstrated an association between exposure to PM and respiratory symptoms severe to restrict their activities. Respirable particulate matter from combustion sources ($PM_{2.5}$) was associated with increased respiratory symptoms, including cough, wheeze and shortness of breath (12,16,24). In two studies in Utah Valley, upper and lower respiratory symptoms increased with $PM_{10}$ concentrations (20,22,23). $PM_{2.5}$ levels and H+ were associated with moderate to severe cough and shortness of breath (14).

CHANGE IN LUNG FUNCTION

An association between $PM_{10}$ air pollution and pulmonary function reduction was reported in several epidemiological studies. In a study conducted in Utah Valley in 1989, Pope et al., found a relationship between elevated $PM_{10}$ levels and reduction in lung function as measured by peak expiratory flow (PEF) (20, 22,23).

According to the NAAQS and the AQI, $PM_{10}$ levels of 150µ g/m³ increase the likelihood of respiratory symptoms and aggravation of lung disease. However, numerous studies have showed that $PM_{10}$ health effects can be observed with $PM_{10}$ levels below give values, although the PSI does not describe health effects or offer cautionary statements at these levels (Table 3 and 4).

## $PM_{10}$ NAAQS RANGES AND HEALTH EFFECTS

### NAAQS: 0.0 TO 54µ g/m³ 24-H AIR QUALITY CATEGORY: GOOD

**The PSI does not describe specific health effects and cautionary statements for these $PM_{10}$ concentrations. However, numerous studies have demonstrated an association between $PM_{10}$ exposure at 10µ g/m³ and increased daily mortality for cardiorespiratory diseases. The risk is higher in individuals over 65 years old of age (3,10,13,17,18,19). Another study conducted in the U.S. demonstrated that for each 10µ g/m³ rise in $PM_{10}$ postneonatal general mortality, sudden infant death syndrome and postneonatal respiratory mortality increased 4%, 12%, and 20% respectively (34). Other studies observed a relationship between $PM_{10}$ exposure at 20µ m/m³ and changes in PEFR in children with chronic symptoms or asthmatics (16,24); between $PM_{10}$ levels of 30µ g/m³ and increased total mortality (4.6%), decreased peak expiratory flow (PEF) and increased symptoms and medication use in asthmatic children (7,10,27,34,35); and between $PM_{10}$ levels of 40µ g/³ an increased cardiovascular mortality in individuals under 65 years old and mortality by Sudden Infant Death Syndrome (19,34). In addition, several epidemiological studies observed with $PM_{10}$ levels of 50µ g/m³ an increase in respiratory diseases such as pneumonia, chronic obstructive pulmonary disease (COPD), and asthma. Also, a reduction in pulmonary function, an increase in hospital admission, and increased cardiorespiratory mortality were found at these $PM_{10}$ concentrations.**

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**NAAQS: 55 TO 154µ g/m³ 24-H AIR QUALITY CATEGORY: MODERATE**

**In the EPA PSI no health effects are described in this range. However, in a study conducted in Coachella Valley, California, at $PM_{10}$ levels of 57µ g/m³, deaths due to cardiovascular and respiratory diseases increased in individuals older than 50 (18). Another study conducted in Coachella associated $PM_{10}$ levels of 87µ g/m³ with an increase in hospital admissions of children with respiratory disease (36). Health effects are aggravated with concentrations approaching 100µ g/m³. Schwartz et al quoted that 100µ g/m³ $PM_{10}$ concentrations increased the relative risk (1.07, 95% CI: 1.02, 1.12) for ischemic heart disease admissions (2). Other studies have shown a relationship between 100µ g/m³ $PM_{10}$ and hospital admissions for respiratory diseases including pneumonia, COPD and asthma in all ages. Children report cough, wheeze and asthma medication use. Also, smokers with mild to moderate COPD demonstrated a decline in $FEV_1$ and PEF (2,3,7,18,19,21,27,28,30,33). $PM_{10}$ exposure to 100µ g/m³ is also associated with a 16% increase in deaths per day, primarily respiratory death and cardiovascular death (7,21,26). In a study conducted in Utah Valley, elevated $PM_{10}$ levels (150µ g/m³) were associated with a statistically significant reduction in lung function measured by reduction in peak expiratory flow (PEF) and increased symptoms of respiratory disease (20,22,23). In addition, another study found $PM_{10}$ association with a 26% increase in upper respiratory symptoms; a 50% increase in lower respiratory symptoms and 217% increase in the use of asthma medication and bronchodilator. (27).**

**NAAQS: 155 TO 254µ g/m³ 24-H AIR QUALITY CATEGORY: UNHEALTHY**

**FOR SENSITIVE GROUP**

**In this range, in a study conducted in Mexico demonstrated a relationship between $PM_{10}$ exposure to 166µ g/m³ and cough and difficulty breathing in children (25). In another study, Pope et al found an association between 195µ g/m³ $PM_{10}$, reduction in EF, and an increase in symptoms of respiratory disease and asthma medication use (20,22).**

**NAAQS: 255 TO 354µ g/m³ 24-H AIR QUALITY CATEGORY: UNHEALTHY**

**No information available.**

**NAAQS: 355 TO 424µ g/m³ 24-H AIR QUALITY CATEGORY: VERY UNHEALTHY**

**No information available.**

## PM2.5 NAAQS RANGES AND HEALTH EFFECTS

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**NAAQS: 0.0 TO 15.4μ g/m³ 24-H AIR QUALITY CATEGORY: GOOD**

According to the NAAQS, at $PM_{2.5}$ levels from 0.0 to 15.4 no health effects are expected. However, the Six U.S. Cities study found a relationship between exposure to $PM_{2.5}$ at 11μ g/m³ and daily mortality. A study conducted in Canada indicated that an increase of 10μ g/m³ in $PM_{2.5}$ was associated with an 3.3% increase in respiratory and cardiac hospital admission (5).

**NAAQS: 15.5 TO 65.4μ g/m³ 24-H AIR QUALITY CATEGORY: MODERATE**

In contrast with $PM_{10}$, in this range the PSI includes a $PM_{2.5}$ health effect cautionary statement where it mentions the possibility of aggravation of heart or lung disease among persons with cardiopulmonary disease and the elderly. A few epidemiological studies found that $PM_{2.5}$ exposure from 18μ g/m³ to 30μ g/m³ increased daily mortality due to cardiorespiratory mortality in the elderly and persons with cardiovascular and respiratory disease. (7,8,9,14). In addition, other studies demonstrated a relationship between 45μ g/m³ and increased hospital admission and chronic bronchitis (3,7).

**NAAQS: 65.5 TO 100.4μ g/m³ 24-H AIR QUALITY CATEGORY: UNHEALTH**

**FOR SENSITIVE GROUP MPDERATE**

No information available

**NAAQS: 100.5 TO 150.4μ g/m³ 24-H AIR QUALITY CATEGORY: UNHEALTHY**

No information available

**NAAQS: 150 TO 250.4μ g/m³ 24-H AIR QUALITY CATEGORY: VERY UNHEALTHY**

No information available

**NAAQS: 250.5 TO 500.4μ g/m³ 24-H AIR QUALITY CATEGORY: HAZARDOUS**

**No information available**

# VI. CONCLUSION

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Extensive epidemiological evidence demonstrated that increases in ambient particulates concentrations are associated with increases in total mortality from respiratory and cardiac diseases, increases in hospitalizations and emergency room visits for respiratory and cardiac diseases, increases in daily respiratory symptoms and decreases in pulmonary function. Sensitive groups including the elderly, children and individuals with cardiopulmonary diseases such as asthma and COPD, are at greater risk of developing adverse health effects from particulate exposure. Several recently published health studies indicate that increases in cardiorespiratory mortality and morbidity can be expected with particle levels below the particulate matter standard.

Numerous epidemiological studies have mentioned that the relationship between $PM_{2.5}$ and adverse health effects is stronger than $PM_{10}$. Fine particulate pollution is of specific concern because it contains a higher proportion of various toxic metals and acid species, and aerodynamically it can penetrate deeper into the respiratory tract. However, the number of studies that demonstrating $PM_{10}$ adverse health effects is greater than PM2.5.

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