International Journal of Innovative Research in Engineering and Management (IJIREM)
ISSN: 2350-0557, Volume-9, Special Issue-6, June-2022
Innovative Projects 2022
Organized by Presidency University, Bangalore, India

Determination of Particulate Matter

Mr. Venkatesh Ashokababu
Assistant Professor,
Masters In Business Administration, Presidency University, Bangalore, India
Email Id:ashokababu@presidencyuniversity.in

Particulate matter PM, a combination of solid particles and liquid droplets present in the air, may be either natural or man-made. Natural particle sources include crustal or surface particles spread by wind, plant pollen, and sea spray. Anthropogenic sources include combustion products such as mobile sources, industrial power plants, industries, refineries, forest fires and/or agricultural sources, as well as secondary sources such as aerosol condensation.

Characteristics and Deposition

The particle size is significant because particle size influences exposure. Particles larger than 10 microns are often undesirable. Particles 10 to 2.5 microns in size are coarse and are inhaled into the major airways; particles 2.5 microns in size may reach the lungs alveolar spaces; and particles 0.1 microns in size can reach the distal lung and have an attraction for bacteria. in order to absorb. The respiratory systems defences against inhalable particles are divided into three levels of defences that are confronted sequentially by particles that enter the airways. The lower respiratory tracts first line of defense is impaction, sedimentation, and diffusional deposition of particles suspended in inspired air as it travels via the nose, nasopharynx, pharynx, and larynx, as well as the conducting airways, or tracheobronchial tree. Particle accumulation throughout the airway's limits particle penetration into the more sensitive gas-exchanging structures, such as the respiratory bronchioles, alveolar ducts, and alveoli in the lung's periphery.

The fluids that line the airways and gas exchange structures, as well as the clearance processes that physically remove particles from their surfaces, offer the second line of defences. The respiratory tract fluids operate as a physical barrier between particles on airway surfaces and the bronchial and alveolar epithelia; these fluids may also act as a chemical buffer if they include chemicals with detoxifying and antibacterial properties. Moreover, the secretions that cover the ciliated epithelia of the upper and lower airway carrying air passageways generate a viscoelastic fluid. Residents alveolar macrophages scavenge particles from the surfaces of the alveoli, digest them, and/or remove them via the mucociliary escalator [1]–[3].

Particles enter the respiratory system by numerous methods, including impaction, gravitational sedimentation, Brownian diffusion, and interception. Deposition by impaction happens at airway bifurcations when a particle fails to make the turn into either of the daughter branches due to its velocity and the aerodynamic forces placed on it by the stream of air in which it is transported. Gravitational sedimentation refers to the settling of particles onto airway surfaces caused by gravity. Gravitational factors that induce sedimentation and impaction have less influence on particles 2.5 microns in diameter, and they are more impacted by the random thermal kinetic buffeting of the gas molecules in the air surrounding them. Sedimentation increases with airway length and is unaffected by branching angle; impaction deposition increases with branching angle and is unaffected by airway length. Hacking occurs when long fibres penetrate the lower respiratory tract like a spear.

from combustion processes, generated from coagulation of smaller particles from the nucleation and Aitken modes aerosol droplets <100 nm, or created subsequently in atmospheric reactions. The nucleation mode is mostly composed of combustion-related gases that condense forming particles that comprise the Aitken mode. The coarse mode comprises particles with diameters ranging from 2 to 100 mm and generally provides the greatest mass. Particles in the accumulation phase have the largest surface area and vary in diameter from 0.1 to 1.0 mm. The coarse and accumulation modes include virtually all of the PM mass. Particles in the ultrafine region are smaller than 0.1 mm in diameter with nucleation mode particles ranging from 0.01 to 0.10 mm in diameter and Aitken mode particles ranging from 0.01 to 0.10 mm in diameter and contain the most particles; the ultrafine mode contributes only a negligible amount to PM mass.

There might also be significant concentration discrepancies between urban and nonurban areas. Annual average PM 2.5 concentrations in mostly metropolitan regions in the United States have recently varied from 4 to 28 mg/m3 median 13 mg/m3. Except for California, urban PM concentrations in the United States are generally greater in the east than in the west. From 1999 to 2003, urban concentrations fell 10% and rural concentrations fell 20% countrywide; only the northeast region had no decline in annual PM2.5 concentrations during that time period.

Epidemiological Studies

Many studies have shown links between higher PM concentrations and greater cardiopulmonary mortality. They discovered that each 10 mg/m3 rise in PM10 increased mortality by a tenth of a fraction 2, 3, 4, 5, 6, 7, 8, 9. Despite the minor effect of PM rises, the direct outcome might be significant if seen over large populations. As a result, rising ambient PM concentrations pose a pretty high risk of death. Similar studies have shown significant health risks connected with greater PM, such as an increased risk of lung cancer and heart disease. Arden Pope and colleagues launched the American Cancer Society Air Pollution Study in the autumn of 1982, with 1.2 million people participating. A subset of 552,138 persons resided in 151 US urban regions that could be linked to air quality statistics gathered by the Environmental Protection Agency EPA.

In this subgroup, the links between sulphate and particulate matter air pollution and all-cause, lung cancer, and cardiovascular mortality were investigated using multivariate analysis that controlled for smoking, education, and other risk variables up to 1989. Deaths from air pollution were 15%-17% higher in the most contaminated neighborhoods than in the least polluted. PM2.5 data were gathered and assessed using mortality risk ratios derived by a Cox proportional hazard regression model in a follow-up of this cohort until 1998, when 22.5% of the cohort died. Each 10 mg/m3 rise in PM 2.5 was associated with a significant increase in mortality for ischemic heart disease, dysrhythmias, heart failure, and cardiac arrest, as well as pneumonia and influenza among nonsmokers. Each 10 mg/m3 increase in fine particle air pollution was related with a higher risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively.

Since PM levels have decreased over the last two decades, Pope and colleagues compiled data on life expectancy, socioeconomic status, and demographic characteristics for 51 U.S. metropolitan areas, as well as data on fine particulate air pollution for the late 1970s and early 1980s and the late 1990s and early 2000s. They discovered that a reduction in PM2.5 of 10 mg/m3 was linked with an estimated improvement in mean SE life expectancy of 0.610.20 year p 14 0.004. Air pollution reductions contributed for up to 15% of the total improvement in life expectancy in the studied locations. At the same time that the American Cancer Society cohort was being created, researchers at Harvard School of Public Health began a six-city longitudinal study on the health impacts of air pollution. Beginning in the 1970s, the Harvard Six Cities Study was a sixteen-year prospective cohort study of 8,111 people residing in the northeastern and midwestern United States. The research found a link between PM2.5 and total mortality, cardiovascular causes, and lung disease. There was a 26% increase. Total mortality disparity between the most congested city and the least polluted city Portage, Wisconsin. An eight-year extended follow-up

confirmed the higher mortality rate ratios of 1.27 for lung cancer and 1.28 for cardiovascular fatalities, with lower PM2.5 levels in more polluted areas linked with lower mortality.

These findings revealed that the mortality impact was not cumulative and that reducing air pollution had a public health benefit. This was more noticeable in cardiopulmonary mortality than in lung cancer mortality. Following these studies, Jonathan Samet, M.D., and colleagues published the National Morbidity and Mortality Air Pollution Study NMMAPS, in which they assessed the effects of five major outdoor air pollutants on daily mortality rates in twenty U.S. metropolitan areas from 1987 to 1994. PM10, ozone, sulphur dioxide, carbon monoxide, and nitrogen dioxide were the contaminants. They discovered a 0.5% increase in all-cause mortality for every 10 mg/m3 rise in PM10. After adjustments to the statistical modelling, this modest gain was eventually corrected downward to 0.2%. The data were examined using a generalised additive model GAM in S-plus with previously used default convergence criteria and with more strict criteria and a generalised linear model GLM using natural cubic splines. The initial technique indicated a 0.41% increase in overall mortality from nonexternal causes per 10 mg/m3 rise in PM10; the more rigorous criterion projected a 0.27% increase; and GLM estimated a 0.21% increase. The risk was greatest in the Northeast, as well as for cardiovascular and respiratory diseases. Seasonal ozone June, July, and August increased total mortality by 0.41% per 10 ppb. CO, SO2, and NO2 had no significant relationships. PM levels fell by 20% throughout these years, according to 799 monitoring sites. In linear regression models, key socioeconomic characteristics had no effect on the link between PM10 levels and the risk of mortality [4]–[6].

These NMMAPS data were then connected with Medicare hospitalizations from 1999 to 2002 for 204 U.S. metropolitan counties including 11.5 million Medicare patients age >65 years who lived an average of 5.9 miles from a PM2.5 sensor. They found a short-term increase in hospitalisations for stroke, ischemic heart disease, cardiac rhythm, heart failure, COPD exacerbations, and respiratory tract infections per 10 mg/m3 PM2.5, but no increase in injuries a negative control group with no evident association to air pollution. Cardiovascular risks were shown to be greater in counties situated in the eastern United States, particularly the Northeast, South, and Midwest. The East has a higher sulphate content of PM, whereas California has a higher nitrate concentration, which likely reflects power plant emissions in the East and transportation in California. The reasons behind these negative results are assumed to be that PM2.5 causes inflammatory reactions in the lower respiratory tract, resulting in the production of cytokines with local and systemic implications. Particulate matter causes inflammation, worsens underlying lung illness, and lowers the effectiveness of lung defences systems.

Moolgavkar studied data from Los Angeles County, California, from 1987 to 1995 and discovered that PM2.5 was substantially related with hospital admissions for cardiovascular illness in those aged 65 and above. Air Pollution and Health: A European Approach APHEA 1 and 2 across Europe discovered epidemiological evidence that short-term exposure to PM and ozone increased daily hospital admissions and emergency room ER visits for asthma higher effect in children than adults and COPD in eight cities. 10 During an eleven-year period, the Swiss Cohort Study on Air Pollution and Lung Disorders in Adults SAPALDIA connected PM10 readings to lung function in 4,742 people. They discovered a linear connection with FEV1 ranging from 5 to 45 mg/m3, supporting World Health Organization guidelines for yearly PM10 limits of 20 mg/m3 and a decrease in PM10 over the course of eleven years. 5.3 mg was linked to substantial decreases in yearly levels of lung function decline FEV1. The California Air Resources Board has supported air pollution research in order to give data on the particular conditions of California smog and to establish a justification to justify expenditures and emission-reduction initiatives

The Health Study, which began at the Department of Preventive Medicine at the University of Southern California, recruited fourth-grade students in twelve neighborhoods in the Los Angeles basin. In 1993 and 1996, two cohorts were recruited total n 14 3840, and a quarterly questionnaire and lung function test were acquired. Hourly ozone, NO2, PM10, and two-week integrated samples of PM2.5 and acid vapour were all measured. Galderma and colleagues studied 1,759 children with eight years of lung function data

to compare living in high- and low-polluted neighborhoods. NO2, acid vapour, PM2.5, and elemental carbon were linked to deficits in the increase of forced expired capacity in the first second FEV1. The percentage of projected FEV1 at age 18 that was 80% of normal values was 4.9 times higher in children from the highest PM2.5 areas vs those from the lowest 7.9% versus 1.6%, respectively.

The Health Study, which began at the Department of Preventive Medicine at the University of Southern California, recruited fourth-grade students in twelve neighborhoods in the Los Angeles basin. In 1993 and 1996, two cohorts were recruited total n 14 3840, and a quarterly questionnaire and lung function test were acquired. Hourly ozone, NO2, PM10, and two-week integrated samples of PM2.5 and acid vapour were all measured. Galderma and colleagues studied 1,759 children with eight years of lung function data to compare living in high- and low-polluted neighborhoods. NO2, acid vapour, PM2.5, and elemental carbon were linked to deficits in the increase of forced expired capacity in the first second FEV1. The percentage of projected FEV1 at age 18 that was 80% of normal values was 4.9 times higher in children from the highest PM2.5 areas vs those from the lowest 7.9% versus 1.6%, respectively.

Those who resided closest to a motorway had lower FEV1 compared to people who lived at least 1,500 metres away from a motorway. Dwelling within 75 metres of a major road raised asthma risk by 1.29 95% confidence range 1.01- 1.86, prevalent asthma, and wheeze; background values were seen 150-200 metres from a major road. 14 Those who relocated to a less polluted place after leaving the Los Angeles area showed less loss of lung function, according to assessments of persons who had left the area. Several studies have sought to uncover detrimental health consequences among those exposed to traffic since it is a significant source of pollutants. Beginning in 1986, environmental scientists in the Netherlands investigated a random sample of 5,000 people enrolled in a national diet and cancer study ages 55-69 years with their home addresses and plotted distance to a major road using Coxs proportional hazard models, with follow-up until 1994. Living near a major road increased the relative risk of cardiopulmonary death by 1.95 95% confidence interval [CI] 1.09-3.52.

After controlling for age, gender, education, employment, and active and passive smoking, the all-cause death rate for living near a major road was 1.41. Asthmatics would seem to be more vulnerable to traffic-related exposure, particularly black smoke from diesel engines. McCreanor et al. devised such a trial by enrolling sixty asthmatics in a randomised crossover study that compared walking for two hours on polluted Oxford Street confined to diesel-powered buses and taxicabs against walking for two hours in lovely Hyde Park. After walking on Oxford Street, their FEV1 was dropped by 6.1% and their FVC was reduced by 5.4% both p0.05, with moderate asthmatics seeing higher decreases. Sputum was collected after the walk, and the inflammatory mediator, myeloperoxidase, was measured to be 24.5 ng/ml after walking on Oxford Street vs 4.2 ng/ml after walking in Hyde Park. These relationships were significant for ultrafine particles, elemental carbon, and NO2, but merely trended towards significance for PM 2.5. Another significant research of children exposed to traffic using pulmonary function and induced sputum measures discovered tiny carbonaceous particles inside alveolar macrophages that linked with greater PM10 exposure and decreased FEV1. For example, each 1.0 mm2 increase in carbon in the alveolar macrophage was related with a 17% reduction in projected FEV118

Air pollution intervention studies have shown reductions in pollutant levels as well as better health outcomes. A thirteen-month worker strike at the local steel plant occurred in Utah Valley, Utah, in 1987. PM10 levels fell by around 15 mg/m3, and overall fatalities fell by 3.2%. 19 Compared to before the strike, acute bronchitis and asthma exacerbations in preschool-aged children decreased by half. Throughout the 1980s, Dublin, Ireland, saw decreasing air quality due to a shift from oil to the much cheaper and more readily accessible bituminous coal for household space and water heating. High levels of air pollution were linked to an increase in in-hospital respiratory mortality. The Irish government prohibited coal inside Dublin on September 1, 1990. Black smoke was reduced by two-thirds, and SO2 by one-third. The mortality rate reduced by 5.7% when comparing the six years after the intervention to the six years before; cardiovascular deaths fell by 10.3% and respiratory deaths declined by 15.5%.

Ghio and Devlin explored the continuing theory that PM promotes inflammation in the lower respiratory tract directly by introducing PM into the lung using a fiberoptic bronchoscope. Twenty-four nonsmokers were given a left lavage bal lavage BAL followed by 10 ml of PM10 saline extract. Rats exposed to PM2.5 for five hours had higher levels of reactive oxidants and antioxidant enzymes in their lung tissues. Using ApoE-/- transgenic mice, atherosclerosis and vascular inflammation developed on high-fat chow with PM2.5 exposure for six hours per day, five days per week, for six months, as evidenced by increased thoracic and abdominal plaque: PM2.5 41.5% versus air 26.2% p 0.001 or normal chow PM2.5 19.2% versus 13.2%. 30 Animals given high-fat chow and exposed to PM2.5 showed enhanced macrophage infiltration, elevated nitric oxide synthase expression, increased reactive oxidants, and had higher lipid content in the aortic arch. Ultrafine particles may have an even bigger impact on the development of atherosclerotic plaques in these animals.

Human clinical and epidemiological research of air pollution and cardiovascular disease have become more difficult. Increases in PM10 and PM2.5 particles were strongly related with increases in C-reactive protein, a marker of systemic inflammation collected on many occasions in a German panel study of 57 male coronary heart disease patients. Peters and colleagues investigated whether exposure to air pollution while stalled in traffic increased the chance of a heart attack heart attack. 33 They gathered case histories of 691 people who had heart attacks in Germany and survived for 24 hours, as well as diaries describing their activities in the four days before the heart attack. They discovered an elevated odds ratio OR of being in traffic in a car, using public transportation, or riding a bicycle at least one hour before having a heart attack OR 2.92 95% CI 2.22-3.83; P 0.001. They conducted comprehensive correlations with PM10 and gaseous air contaminants, however the results of these studies were insignificant. In a case-control study conducted in Worcester, Massachusetts, long-term exposures to traffic within 100 metres of dwelling near a road was analyzed.

Heart Attack Research.34 Between 1995 and 2003, they had 5,049 confirmed cases; demographic controls were collected from Massachusetts resident records. They discovered a 5% increase in the probability of having an acute myocardial infarction near a major highway using logistic regression 95% CI 3-6%. Correlations for housing, job, and air pollution variables were created in a cohort of 3,239 nonsmoking people observed in California throughout twenty-two years. 35 Only females reported statistically significant correlations between coronary heart disease and PM2.5, with the relative risk RR rising to 1.42 95% CI 1.06-1.90. In a two-pollutant model, ozone raised the relative risk to 2.00 95% CI 1.51-2.64. From 1994 to 1998, the NIHs Womens Health Initiative recruited 65,893 postmenopausal women without prior cardiovascular illness from 36 U.S. metropolitan regions and followed them for a median of six years.

There were 1,816 women who had one or more fatal , nonfatal cardiovascular events, such as death from coronary or cerebrovascular disease, coronary revascularization, myocardial infarction, or stroke. Each 10 mg/m3 increase in PM2.5 exposure was linked with a 24% increase in the probability of a cardiovascular event Hazard Ratio 1.24; 95% CI 1.09-1.41 and a 76% increase in the risk of mortality from cardiovascular disease Hazard Ratio 1.24; 95% CI 1.09-1.41. Hazard Ratio 1.76; CI 1.25–2.47. There was a greater within-city variation in exposure differences than there was across cities. Twenty men with past heart attacks but now stable were exposed to dilute diesel exhaust 300 mg/m3 of filtered air for one hour with moderate activity in a controlled, double-blind, randomised crossover research.

37 Exercise-induced ST-segment drop an electrocardiographic indication of heart muscle oxygen deprivation was seen in all individuals, although there was a larger increase in the ischemia load following diesel exhaust exposure. Exposure to diesel exhaust did not worsen preexisting vasomotor dysfunction, but it did lower acute endothelial plasminogen activator release a mediator of fibrinolysis that dissolves blood clots that diesel particulate aggravated electrocardiographic signs as compared to filtered air. Carotid artery intima-media thickness was measured by ultrasonography in 798 patients from vitamin E and B atherosclerosis intervention studies as another indicator of atherosclerosis due to air pollution. 38

Annual PM2.5 concentrations were geocoded in their residential regions. Carotid intima-media thickness rose 5.9% 95% CI 1- 11% with a cross-sectional exposure contrast of 10 mg/m3 PM2.5. The greatest link between PM2.5 and carotid intima/media thickness was seen in women over 60, with a 15.7% correlation. There may be a genetic predisposition to air pollution exposures; for example, persons missing the gene glutathione S-transferase M1, which encodes an enzyme scavenger of oxygen-free radicals, are more sensitive to inhaled particulate matter, as seen by higher alterations in heart rate variability.

Patients fitted with automated implanted cardioverter defibrillators AICDs were utilised to research the short-term effects of PM and other atmospheric pollutants on arrhythmias in a demographic subset that may be predicted to be more vulnerable. AICDs keep track of past arrhythmias, which may be used to assess the link between arrhythmia initiation and PM concentration. Research from Boston showed the first evidence that short-term increases in PM and NO2 concentrations were related with arrhythmias. Current research has also looked at links with pregnancy outcomes. Just a few of them have concentrated on the short-term relationships with PM. Associations between short-term increases in PM concentrations and newborn mortality, particularly respiratory mortality and death from sudden infant death syndrome, have been observed in time-series investigations of overall and more specific causes of mortality. 41 In a recent time-series analysis, PM10 averaged over the preceding six weeks, or at a few particular days before delivery, was linked to premature birth. A statewide study conducted by the New Jersey Department of Health linked birth certificate and maternal/newborn hospital discharge summaries for all singleton births from 1999 to 2003 with a gestational age of 37-42 completed weeks and a birth weight >500 grammes with air pollution data for PM2.5, NO2, CO, and SO2 from a monitoring station within 10 kilometers of residence [7]–[9].

43 There were significant increased risks for small for gestational age fetal growth ratio >0.75 and 0.85, 88,678 births and first and third trimester PM2.5 exposure, and for very small for gestational age foetal growth ratio 0.75, 114,411 births and NO2 concentrations in the first, second, and third trimesters. After adjusting for known risk variables, this implies that traffic has a negative influence on birth outcomes. At least two time-series studies suggest that crustal PM may not cause mortality. High-PM concentrations due to windy circumstances that result in the suspension of crustal PM in air in Spokane, Washington44 and Salt Lake City, Utah45 were not linked with mortality, although PM on other days did.

Rather of concentrating on the impacts of individual PM components, concentrating on the effects of particles released from certain sources might possibly aid in the identification of especially dangerous kinds of PM. In a few health investigations, 46 Factor analysis methodologies were employed to divide daily PM concentrations into their respective source contributions. Data from the Six Cities Study in the United States, which included measurements of elemental chemical species of fine PM, were used to determine daily mortality effects associated with different sources of ambient PM. 47 PM from motor vehicle exhaust was consistently related with higher daily mortality than PM from coal burning. Crustal PM was not linked to mortality. Independent of chemical content, particle size might potentially have an impact in toxicity. In epidemiological research, PM has traditionally been classified by size into total suspended particles TSP and its inhalable fraction PM10, which has been further classified into coarse PM10-2.5 and fine PM2.5 fractions, owing in part to the use of PM data from regulatory monitoring networks.

Respiratory Effects of Concentrated Ambient Particles

At particle concentrations of up to 311 mg/m3, exposure to concentrated ambient particles CAPs in healthy and asthmatic persons has shown a lack of respiratory symptoms or changes in spirometry. Healthy subjects in these investigations demonstrated a slight increase in bronchoalveolar lavage microorganisms 18 hours after CAPs exposure compared to filtered air. In one investigation, there was no significant impact on spirometry or induced sputum cell counts in older people with and without COPD who were exposed to CAPs and NO2. Nonetheless, there were decreases in maximum mid-expiratory flow and arterial saturation related with CAPs exposure, indicating an impact on small airways.

Interestingly, the healthy persons had a higher influence. After CAPs exposure, induced sputum in asthmatics, young healthy individuals, and elderly participants with and without COPD included less columnar epithelial cells than filtered air, indicating a possible impact on bronchial epithelial cells.

Diesel exhaust enhances airway hyper responsiveness to methacholine and airway resistance in individuals with moderate asthma, according to controlled exposure studies in healthy and asthmatic volunteers 50,51 raises sputum neutrophil numbers as well as mast cell, neutrophil, and lymphocyte counts in bronchial tissue. Moreover, diesel particulate matter DE exposure enhances the expression of IL-6, IL-8, and the stickiness molecules ICAM-1 and VCAM-1. Simultaneous allergen and DE exposure have been proposed as a possible cause of increased allergic sensitivity and the incidence of allergic diseases. Researchers have proven the capacity of DE or traffic exposure to amplify subsequent allergic symptoms antibody class switching, Th2 skewing of cytokines, and neo-antigen effects in people using high-level exposures and nasal instillations. Recent research suggests that the diesel particle is to blame for the allergy sensitivity.

Particulate Matter and Public Policy

The EPA has developed six criterion pollutants: ozone, particulate matter, carbon monoxide, nitrogen oxides, sulphur dioxide, and lead. PM is unusual among them in that it is not a particular chemical molecule or element. Each NAAQ is made up of an indicator, average time, a form, and a level. In 1987, PM10 replaced TSP as the indication. In 1997, PM2.5 was included as an indicator. The periods for PM averaging include both a 24 hr average and a yearly average. The standards forms are related to the particular data that are computed for assessing area attainment with the NAAQS. Different NAAQS are established to safeguard public health the main standard and welfare the secondary standard; welfare impacts include visibility and flora effects. The main and secondary PM NAAQS have been the same since 1987. The NAAQS are required to be reviewed every five years.

The most recent EPA PM review was done in 1997, when the 24-hour and yearly thresholds for PM10 were maintained at 150 mg/m3 and 50 mg/m3, respectively. At the time, the initial 24-hour and yearly PM2.5 values were established at 65 mg/m3 and 15 mg/m3, respectively. Because of the plethora of scientific health evidence that has subsequently gathered suggesting that impacts may occur much below such concentrations, it became critical that the next assessment propose reductions in these levels. The California Air Resources Board authorised new ambient air quality guidelines in 2005 that were far stricter than the EPAs, with 24-hour and annual PM10 values of 50 and 20 mg/m3, respectively, and an annual PM2.5 concentration of 12 mg/m3. The European Union proposed maximum values for 24-hour and annual PM10 to be attained in 2005 were 50 and 40 mg/m3, respectively, with a further decrease in the annual level to 20 mg/m3 by 2010.

The American Trucking Association sued the EPA over the 1997 PM regulations, and the D.C. Circuit Court of Appeals ruled in a 2-1 vote that the new public health air quality criteria for PM were unconstitutional and an unlawful transfer of legislative power to the EPA. The EPA took the courts ruling all the way to the United States Supreme Court. The Supreme Court maintained the EPAs jurisdiction to establish national air quality standards in a historic decision in February 2001, protecting millions of people from the damaging consequences of air pollution. The Supreme Court also affirmed that the Clean Air Act does not permit the EPA to consider cost when setting national ambient air quality standards, but rather requires the EPA to set those standards at levels sufficient to protect public health with an adequate margin of safety and to protect public welfare from adverse effects. This was a significant case in terms of interpreting the constitutional foundation for the EPA to enforce low emission standards in order to safeguard the publics health.

The EPA staff document suggested two alternatives for the administrator to consider an annual PM2.5 standard of 15 mg/m3 with a revised 24-hour PM2.5 standard in the range of 35-25 mg/m3; or an annual standard of 14-12 mg/m3 with a revised 24-hour PM2.5 standard in the range of 40 to 35 mg/m3 to offer

supplementary protection against episodic localised or seasonal peaks. The Clean Air Scientific Advisory Committee CASAC evaluated the EPA criteria document for PM2.5, including health effects, exposure trends, and policy considerations, and recommended that the administrator strengthen the 24-hour standard from 65 to 35 mg/m3 and the annual standard from 15 mg/m3 to the range of 13-14 mg/m3. Yet, the CASAC recommendations were not unanimous. The American Thoracic Societys ATS and American Lung Associations ALA Environmental Health Policy Committees collaborated to decrease the 24-hour threshold to 25 mg/m3 and the yearly level to 12 mg/m3.

To achieve these levels, the ATS wrote a letter to the administrator and enlisted the support of other professional societies, along with the American College of Cardiology, the American Academy of Pediatrics, the American Association of Cardiovascular and Pulmonary Rehabilitation, and the National Association for the Medical Direction of Respiratory Care. The American Medical Association House of Delegates presented legislation and approved the ATS recommended levels. Separately, the American College of Chest Physicians, the American College of Preventive Medicine, and the American Public Health Association all endorsed reducing the criterion. The ATS then expressed verbal support for reducing the bar during a phone conversation during the CASAC public hearing. The EPA convened public hearings around the nation, and the ATS coordinated academic pulmonologists who spoke so that each one had a public record of support.

Finally, the EPA administrator granted the ATS and ALA a personal interview to present their data and opinions. They met with Administrator Steven Johnson and his advisers in the Rachel Carson Great Room and argued vehemently that there was scientific agreement supporting a lesser requirement. The EPA did not follow the CASAC recommendations by maintaining the yearly guideline, but did cut the 24-hour threshold to 35 mg/m3. PM10 was still under control. From the industry side, the American Farm Bureau promptly sued the EPA, claiming that PM2.5 was being controlled twice by overlapping PM10 and PM2.5 limits and that farms should only have a coarse threshold of PM10-2.5 that was also nontoxic [10]–[12]. On the other hand, thirteen states, the District of Columbia, the American Library Association, Environmental Defense, and the National Parks Conservation Council all filed briefs arguing that the EPA illegally used uncertainty to justify ignoring scientific consensus recommending a more protective standard.

REFERENCES

[1] Doreswamy, K. S. Harishkumar, Y. Km, and I. Gad, Forecasting Air Pollution Particulate Matter PM2.5 Using Machine Learning Regression Models, 2020. doi: 10.1016/j.procs.2020.04.221.

[2] N. Hamdan, H. Alawadhi, and M. Shameer, SEM/EDS as complementary techniques to XRD and XRF for structural determination of particulate matter pollution, *Microsc. Microanal.*, 2020, doi: 10.1017/S1431927620016591.

[3] A. A. Kokhanovsky, A. S. Prikhach, I. L. Katsev, and E. P. Zege, Determination of particulate matter vertical columns using satellite observations, *Atmos. Meas. Tech.*, 2009, doi: 10.5194/amt-2-327-2009.

[4] M. Szewczyńska, M. Pośniak, and E. Dobrzyńska, Determination of phthalates in particulate matter and gaseous phase emitted into the air of the working environment, *Int. J. Environ. Sci. Technol.*, 2020, doi: 10.1007/s13762-019-02435-y.

[5] M. Ahmed, X. Guo, and X. M. Zhao, Determination and analysis of trace metals and surfactant in air particulate matter during biomass burning haze episode in Malaysia, *Atmos. Environ.*, 2016, doi: 10.1016/j.atmosenv.2016.06.066.

[6] C. D. Simpson, R. L. Dills, B. S. Katz, and D. A. Kalman, Determination of levoglucosan in atmospheric fine particulate matter, *J. Air Waste Manag. Assoc.*, 2004, doi:

10.1080/10473289.2004.10470945.

[7] R. S. Picoloto, S. M. Cruz, P. A. Mello, E. I. Muller, P. Smichowski, and E. M. M. Flores, Combining pyrohydrolysis and ICP-MS for bromine and iodine determination in airborne particulate matter, *Microchem. J.*, 2014, doi: 10.1016/j.microc.2014.05.002.

[8] M. Bełdowska, D. Saniewska, K. Gębka, U. Kwasigroch, E. Korejwo, and J. Kobos, Simple screening technique for determination of adsorbed and absorbed mercury in particulate matter in atmospheric and aquatic environment, *Talanta*, 2018, doi: 10.1016/j.talanta.2018.01.082.

[9] D. Jandacka and D. Durcanska, Differentiation of particulate matter sources based on the chemical composition of PM10 in functional urban areas, *Atmosphere Basel.*, 2019, doi: 10.3390/atmos10100583.

[10] R. G. O. Araujo *et al.*, Determination of silver in airborne particulate matter collected on glass fiber filters using high-resolution continuum source graphite furnace atomic absorption spectrometry and direct solid sampling, *Microchem. J.*, 2013, doi: 10.1016/j.microc.2012.05.009.

[11] J. Klyta and M. Czaplicka, Determination of secondary organic aerosol in particulate matter – Short review, *Microchemical Journal*. 2020. doi: 10.1016/j.microc.2020.104997.

[12] Determination of Particulate Matter and Toxic Gaseous in Ambient Air Adjacent to Industrial Area, *Iran. J. Energy Environ.*, 2017, doi: 10.5829/ijee.2017.08.02.11.