

Health effects of airborne particulate matter and the Indian scenario

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Airborne suspended particulate matter (SPM) is a serious worldwide concern since it is linked with adverse health effects. Several epidemiological studies have been made across the world revealing the association of SPM in air with acute and chronic respiratory disorders, lung cancer, morbidity and mortality. Odds ratio estimated by several studies of the dose–response relationship for particulate matter (PM)-associated respiratory sickness and premature mortality, increased with rise in PM levels. Associations have been found with cardiovascular deaths, with myocardial infarctions and ventricular fibrillation. PM is also associated with autonomic function of the heart, including increased heart-rate, decreased heart-rate variability and increased cardiac arrhythmias. Such health disorders are widely seen in urban areas worldwide that suffer from serious air-quality problems due to increasing population, combined with change in land use and vehicular traffic. In India, haphazard urbanization, unprecedented vehicular emissions and inadequate infrastructure development are supplementary factors for the fall in air-quality. Challenge for the future generation in India lies in grappling the menace of air pollution-induced diseases, where already the public health is in a worrying state with a variety of diseases. This article attempts a brief review of atmospheric PM, its inhalation, deposition and toxicity, with experiences from the western countries and the current Indian scenario.

AMONG the variety of factors influencing health of an individual, natural elements (the air we breathe, the water we drink, the radiation we are exposed to, etc.) and man-made environmental modifications (habitat, place of work, transport, industry and other development activities) play a crucial role. Chemical agents that are released into the environment from various anthropogenic activities impact human health seriously. The respiratory system is one major route whereby these chemicals and toxic agents enter the body and cause disorders, including mortality. On a global scale, millions suffer from respiratory ailments and other diseases attributed to the presence of toxic chemicals and biological agents in the air¹. Although concentration of any pollutant in the environment is a quantitative expression of the presence of the pollutant, there is no exposure unless there is physical contact with human beings². Exposure denotes the event when a person comes into contact with a pollutant for a particular time. On the other hand, dose refers to the actual quantity of pollutant that crosses the barrier of a body. Airborne particulate matter (PM) is the recent focus of the world community as it penetrates the respiratory system of human beings

and causes many disorders. In the case of PM it is believed that aerodynamic size, number and quantity of PM in the atmosphere play a vital role in impacting human health. It has also been shown through worldwide studies that the urban population is at risk due to elevated levels of PM in the urban atmosphere. Several time series and cohort studies have shown that children, elderly and asthmatic people are at higher risk due to air pollution. This article is an overview of outdoor air particulates and their impact on human health effects.

Particulate matter

Suspended particulate matter (SPM) refers to the mixture of solid and liquid particles in air. In a broader sense the term applies to matter in the atmosphere classed into particles having a lower size limit of the order of 10^{-3} μm and an upper limit of 100 μm . SPM, a complex mixture of organic and inorganic substances, is a ubiquitous air-pollutant, arising from both natural and anthropogenic sources. Ever since the advent of the industrial era, anthropogenic sources of PM have been increasing rapidly. PM that is 10 μm or less in diameter is called as respirable suspended particulate matter (RSPM) or PM₁₀, since it penetrates the respiratory system. RSPM is generally grouped into three modes: ultra fine (size range less than

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0.1 μm), fine (0.7–1 μm) and coarse (1–10 μm)^{3–5}. Major components of fine particles often consist of sulphates, carbonaceous materials, nitrates, trace elements and water^{6,7}. Next to inorganic substances, organic substances⁸ are the second major constituents of the fine PM, representing 26–47%. The coarse fraction⁹ is mostly dominated by aluminum, silicon, sulphur, potassium, calcium and iron, which make up 40–50%, while components such as nitrate, sulphate and ammonium ions, elemental and organic carbon make up only 10–20%.

Sources for PM

The ultrafine particles of size range less than 0.1 μm are formed by nucleation, that is condensation of low-vapour-pressure substances formed by high-temperature vaporization or by chemical reactions in the atmosphere to form new particles (nuclei). They are mainly of anthropogenic origin such as from automobile exhaust, wood smoke and emission from diesel engines and generators^{10–12}. Fine particles of size range 0.7–1 μm are formed by accumulation or coagulation of ultra fine particles. In close vicinity of the road, contribution of traffic to fine particle concentration¹³ was 58–68%.

Biomass burning is another important source of fine organic aerosols¹⁴. Coarse particles ranging from 1 to 200 μm are predominantly rock or soil material of natural origin emitted into the atmosphere by mechanical grinding or spraying. These particles can include wind-blown dust from agricultural processes, uncovered soil, unpaved roads or mining operations. Traffic produces road dust and air turbulence that can re-entrain road dust. Near the coast, evaporation of sea spray can produce copious particles. In the urban atmosphere, dust arises due to agitation of soil through activities such as vehicular movement¹⁵, construction and earth-moving⁷. An estimated 80% of coarse particles from traffic in the urban environment¹³ settles within 150 m distance from the road, ~40% at 200–270 m, and ~20% at about 1500 m.

According to Harrison *et al.*¹⁶, PM10 fraction that causes significant health impacts is dominated by particles from three sources. (i) Primary fine particles from industrial and combustion sources, predominantly road traffic. (ii) Secondary aerosol, mostly ammonium sulphate and ammonium nitrate formed through photochemical reactions. (iii) Wind-blown soil and resuspended street dust present largely in coarse fraction (2.5–10 μm). The coarse particles make a significant contribution to the particle mass, with coarse particles showing seasonal variation from about 20% of the total PM10 mass in winter to 50% in summer, reflecting the impact of drier summer climate on the re-suspension process. In the urban atmosphere, Miguel¹⁷ found re-suspension of paved-road dust contributing up to 25–63% of the PM10.

Deposition and clearance mechanisms of PM in respiratory system

On achieving entry into the respiratory system, particles of size range 4.6–9 μm normally deposit in the region of tracheo-pharynx; 1.1–4.6 μm in the bronchi and 0–1.1 μm in the alveoli. Penetration of these particles into the lung airways is determined primarily by convective flow, i.e. motion of the air in which particles are suspended¹⁸. Particles deposit within the respiratory tract by five mechanisms: inertial impaction, sedimentation, diffusion, electrostatic precipitation and interception. Particles <0.5 μm are deposited in small airways. For particles 0.5–2 μm , deposition occurs in small to mid-sized airways by sedimentation. For particles >2 μm , inertia causes the particle motion to deviate from the flow stream lines, resulting in deposition by impaction in mid- to large-sized airways. Interception is deposition by physical contact with airway surfaces. The interception potential of any particle depends on its physical size, and fibres are of chief concern in relation to the interception process. Their aerodynamic size is determined predominantly by their diameter, but their length is the factor that influences probability of interception deposition. Electrostatic precipitation is deposition related to particle charge.

Particles deposited inside the lungs are cleared by several mechanisms¹⁹. Particles deposited in the bronchi and bronchioles (ciliated airways) are captured on the layer of the mucus lining and are carried out of the lungs on the mucociliary ladder for expulsion through coughing or they are swallowed. Particles deposited deeper, in the nonciliated airways, are engulfed by lung macrophages and cleared more slowly, as the macrophages transport the particles onto the mucociliary ladder or into the lymphatic system. In human beings, chronically inhaled PM was retained mainly in interstitium of lungs (57–91%) and its percentage increased with increase in dose²⁰. Overall, clearance of insoluble particles deposited in the pulmonary region of the lung has half-times that are measured in weeks to months or even years⁹. Clearance mechanisms themselves may be adversely affected by inhaled toxicants, so that clearance may take even longer because of the influence of the particles and co-pollutants such as ozone. There are large differences between species in the clearance rates of particles from the lung. The clearance rates in dogs and humans seems to be comparable, but clearance of a variety of particles from the lung of rats was seen to be much faster than from either humans or dogs¹⁹. As a result, the long-term retention of particles deposited in the lung can be much greater in humans than in rats.

Toxic components of PM

Biological or symptomatic effects of the deposited particles are determined by the chemical nature of the particles,

the site of deposition within the lungs and physiologic response to the particles. Nearly 3000 different anthropogenic air pollutants have been identified, of which only for about 200 such compounds, the impact on environment and human beings has been investigated to a notable extent³. Possible fine particulate components that may be responsible for observed health effects include the entire PM_{2.5} fraction, particulate-associated sulphate, nitrate, acidity, soot, transition metals, organic contaminants and total ultra fine particles. Organic carbon (OC) and elemental carbon (EC) are the major carbon fractions in the PM responsible for many deleterious health effects²¹. OC is suspected as mutagenic and carcinogenic, while EC is linked with a range of adverse effects, including interference in the lung-clearance mechanisms. Among OC compounds, poly aromatic hydrocarbons (PAHs) are likely to be carcinogenic and interfere with the hormone systems, reproduction and immune function²². PAHs emitted into the atmosphere by various combustion processes are present in gaseous phase or bound to PM. Several PAHs such as benzo[a]anthracene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, benzo[a]pyrene, associated with PM are direct-acting mutagens, while some others are indirect-acting mutagens in *Salmonella*/microsome assay²². Since vehicular emissions are often cited as an important source of PAH in air, people living in urban areas characterized by high levels of petrochemical pollution are apparently at a greater risk of developing lung cancer than those in the areas of low air-pollution²³. The toxicity and properties of PAHs have been well explained^{22,24}. Reactive oxygen radicals generated by the particles have also been reported as an important factor for acute and chronic toxic effects in the respiratory system²⁵.

A number of experimental studies have demonstrated the physiological effects of PM. For instance, significant increase in pulmonary inflammation was observed in rats when exposed to concentrated air particles ranging from 207 to 733 $\mu\text{g}/\text{m}^3$. Simultaneously, a stress-type pulmonary function response marked by increased deeper breathing and acute response marked by cellular influx (neutrophils, lymphocytes) was seen²⁶. Particularly, diesel exhausts are found to stimulate hormonal secretion of the adrenal cortex, depress gonadotropin-releasing hormone and inhibit spermatogenesis in rats²⁷. Especially, serum levels of testosterone and estradiol were significantly higher in rats exposed to diesel exhaust (5.63 $\mu\text{g}/\text{m}^3$) compared to the controls. However, detailed animal studies on single, specific compounds are not available since the health disorders of PM are mainly due to synergistic effects of a cocktail of compounds.

Assessment techniques for air pollution-related human health effects

Health impacts of air pollution are normally assessed by dose-response studies in a given area, which link the

concentration of air pollutants to the observed health effects. A number of models are attempted for such dose-response extrapolation²⁸⁻³⁰. Through regression analysis, coefficients are estimated that are then multiplied by changes in ambient pollution concentrations and the population exposed. Most of the epidemiological studies have used logistic regression models after controlling other confounders such as age, climate, demography and environmental factors. After adjustment for covariates, the association between air pollution and mortality/morbidity is expressed in Odds ratio and 95% confidence interval.

One of the methods commonly used to estimate health impacts by air pollution is Ostro's approach²⁸: $dHi = bi * POPi * dA$, where dHi is the change in population risk of health effect i ; bi the slope from the dose-response curve for health impact i ; $POPi$ the population at risk of health effect i and dA the change in ambient air pollutant under consideration.

The association between exposure to air pollution and mortality (cause-specific) was assessed with Cox's proportional hazards models, with adjustment for potential confounders³¹. Zuidema and Nentjes³² have reviewed various techniques for estimating dose-response relationship for air pollution-related health damage, morbidity and work-loss days. The review discusses a number of techniques, including ordinary least square method, one-way fixed effect method, Logit linear models and models using Poisson regression analysis. A method suggested by Burnett *et al.*³³ is often used for estimating air pollution and its health effects. The model incorporates risk factors measured at the individual level, such as smoking, and at the spatial level, such as air pollution. It demonstrates that the spatial autocorrelation in community mortality rates, an indication of not fully characterizing potentially confounding risk factors to the air pollution-mortality association, can be accounted for, through the inclusion of location in the model assessing the effects of air pollution on mortality. Exposure assessment studies using personal samplers and recording the levels of particulate exposure throughout the day and accounting for corresponding variations in the health parameters are another method for assessing the health risk posed by air pollutants.

Human health effects of PM (morbidity and mortality)

Notorious acute air pollution episodes such as London smog episodes (1952, 1962), the Donora smog (1948), and Muese valley smog (1930) that resulted in heavy mortality clearly indicate that short-term elevated levels of PM and sulphur dioxide are associated with a variety of pulmonary disorders, including mortality³⁴. Since then, whether such analogous health effects and mortality would occur from long-term exposure to low-level concentrations was a hypothesis. Interestingly, many epidemiologi-

cal studies across the world found association between SPM and acute and chronic respiratory disorders, lung cancer, morbidity and mortality^{29,34-42}. Associations have been found with cardiovascular deaths, myocardial infarctions and ventricular fibrillation⁴³. PM is also associated with autonomic function of heart, including increased heart-rate, decreased heart rate variability and increased cardiac arrhythmias. The hypothesis was also strengthened by observations such as urban-rural differences in the case of mortality from lung cancer and chronic obstructive pulmonary disease. Although lung cancer was rare in the beginning of the 20th century, today it has become one of the leading causes of death in males of the industrialized world. Internationally, lung cancer accounts for up to 13% of all deaths above 45 years of age. The global incidence of lung cancer is increasing at a rate of 0.5% a year.

Diseases caused by air pollution may primarily involve three target tissues⁴⁴. These are the respiratory passage, blood vessels and respiratory membrane. The most prevalent pulmonary disorder is asthma in which the target tissues are the walls of the bronchi and bronchioles. In 1987, there were approximately 25 million individuals in the US who had asthma. Illness associated with asthma accounted for 27 million patient visits and 470,000 hospital admissions annually⁴⁵. The World Health Organization assessed that about 460,000 people die each year because of SPM, among which 135,000 are victims of chronic asthma and the rest die of cardiovascular or heart diseases¹.

Acute infections involving upper respiratory damages causing major morbidity from respiratory illnesses such as influenza, bronchitis (acute and chronic) pneumonia, all types of asthma, sinusitis and other respiratory disorders were reported⁴⁶⁻⁴⁸ since late 1950s. Particulate-associated mortality had been well established across the world by several studies^{41,49}. A possible association between heart disease mortality/morbidity and the same day particulate levels, was reported at Pittsburgh, USA⁵⁰. Dockery *et al.*⁵⁵ found PM10 with stronger associations for cardiovascular mortality and considerably stronger associations for respiratory mortality. Cardiovascular mortality was also found to be significantly associated with other pollutants⁵¹, including carbon monoxide, nitrogen dioxide, sulphur dioxide, PM10, PM2.5 and PM10 minus PM2.5. When the SPM is dominated by 68% of PM10, it is associated with 9.5% increase in mortality (CI = 1.2-18.5%). Particularly, an increase in black smoke by 50 $\mu\text{g}/\text{m}^3$ was associated with 2.2% and 3.1% increase in mortality⁵², when the analysis was restricted to days with < 200 $\mu\text{g}/\text{m}^3$ and < 150 $\mu\text{g}/\text{m}^3$. PM2.5 and PM10 have also significant effect on hospital admission rates for a subset of respiratory diagnoses (asthma, bronchitis, chronic obstructive pulmonary disease, pneumonia, upper respiratory and lower respiratory tract infections), with a relative risk of 1.24 for a log₁₀ increase in exposure⁵³. All age groups are found to be affected, although children and elderly are most

prone to chronic obstructive pulmonary diseases and asthma. Creason *et al.*⁵⁴ found that outdoor PM2.5 is negatively associated with diminished heart rate variability (HRV) response in the elderly [-0.03 change in log (high frequency HRV) for 10 $\mu\text{g}/\text{m}^3$ increase in PM2.5], after adjustment of age, sex, cardiovascular status, trend, maximum temperature, average dew point temperature, random subject intercepts and autocorrelated residuals. The results of the study also suggested that decrease in HRV with higher concentrations of PM2.5 might be a consequence of greater loss of parasympathetic control of heart rate. Recently, Pope *et al.*⁴⁰ reported that each 10 $\mu\text{g}/\text{m}^3$ elevation in fine particulate air pollution was associated with approximately a 4, 6, and 8% increased risk of all-cause, cardiopulmonary and lung cancer mortality respectively. But coarse particle fraction and total suspended particles were not consistently associated with vital status.

Indian scenario

Air quality in most of the Indian cities is deteriorating at a fast pace due to increasing vehicle fleet, with the present figure crossing 35 million⁵⁵. To monitor the air quality changes, a nationwide programme, National Ambient Air Quality Monitoring (NAAQM) was initiated in 1984. As on 31 March 1995, the network comprised 290 stations covering over 90 towns/cities distributed over 24 States and 4 Union Territories. The network is operated through the respective State Pollution Control Boards, the National Environmental Engineering Research Institute (NEERI), Nagpur and also through the Central Pollution Control Board (CPCB). The pollutants monitored under NAAQM are sulphur dioxide, nitrogen dioxide and SPM, besides meteorological parameters like wind speed and direction, temperature and humidity. Recently, in certain major cities, regular PAHs in ambient air are also being included in the assessment. Annual data on air-quality parameters of various places are available at the CPCB website⁵⁶. Station-wise reports on air quality parameters such as SPM, SO₂ and NO₂ published by NAAQM are reproduced in 'The citizens fifth report - Part II: Statistical database'⁵⁷. SPM data of some important cities are reproduced in Table 1. Levels of RSPM are not available in the NAAQM data until 1995.

According to a recent UNEP report⁵⁸, in most of the Indian cities the mean of average values of SPM for nine years (1990-98) ranged between 99 and 390 $\mu\text{g}/\text{m}^3$ in residential areas and between 123 and 457 $\mu\text{g}/\text{m}^3$ in industrial areas, exceeding the annual average limit of SPM for residential areas 140 $\mu\text{g}/\text{m}^3$ and for industrial areas 360 $\mu\text{g}/\text{m}^3$. Other reports on particulate levels are given in Table 2.

Carcinogenic PAH compounds in Delhi ranged between 9.4 and 60.9 ng/m³ during 1999-2000 with higher values

Table 1. Air quality in certain cities of India

City	Population (urban)	Year	SPM level ($\mu\text{g}/\text{m}^3$)			
			Minimum	Average	Maximum	
Metropolitan city	Mumbai	16,368,084	1990–95	48.5	225.7	573
Metropolitan city	Kolkata	13,216,546	1990–95	31.5	353.6	1278
Metropolitan city	Delhi	12,791,458	1987–95	62.5	404	1609
Metropolitan city	Chennai	6,424,624	1987–95	26.2	122.3	516.1
State capital city	Bangalore	5,686,844	1988–91	18.5	146	632
State capital city	Hyderabad	5,533,640	1990–95	20.6	150.4	582.5
State capital city	Ahmedabad	4,519,278	1989–95	37.5	252.5	939
Class II city	Pune	3,755,525	1987–95	50	202	533
State capital city	Jaipur	2,324,319	1990–95	37	278	1067
State capital city	Lucknow	2,266,933	1992–95	224.7	374.5	535
State capital city	Thiruvananthapuram	< 1,300,000	1991–95	24.4	107.8	317.2
Class II city	Shimla	< 1,300,000	1987–95	18.8	197.5	666
Class II city	Coimbatore	1,446,034	1993–95	10	50	153
Class II city	Cochin	1,355,406	1988–95	10.5	97.15	363.2

Source: Agarwal and Narain⁵⁷.

Table 2. Research reports on SPM level in Indian cities

City	Total SPM ($\mu\text{g}/\text{m}^3$) range/mean	Observations	Reference
Delhi	454.70–658.4 of PM10	Values exceeded the CPCB permissible limits. Vehicular emission, industrial and soil resuspension were major sources	69
Shimla	53–322	Residential areas witnessed high SPM in spite of no notable industrial activity	70
Mumbai	180–270 (1981–90)	Annual emissions of TSP and PM10 were 32,000 and 16,000 tones respectively	71
Kolkata	982–1181 (1993–94)	Higher SPM values, high benzene values	72
Chennai	163–1835	Urban areas with highest SPM values followed by industrial and residential areas	73
Bangalore	77–787	SPM levels high at traffic junctions. Physical obstacles and topographical features minimized dispersal of SPM	74
Pune	99–122	TSP concentrations in winter were less compared to the beginning of summer	75
Lucknow	200	–	76
Mysore	79.5–266	–	77
Indore	285–465	–	78
Udagamandalam	22–96 of PM10	RSPM values higher in commercial area where traffic flow is high	79
Urban and suburban Coimbatore	30–149 of PM10	Urban samples exceeded CPCB limit	80

recorded in winter⁵⁶. At Kolkata⁵⁹, the sum of 12 PAHs ranged from 22.91 to 190.96 ng/m^3 in SPM during 1994. In Coimbatore⁶⁰, a total of 13 PAHs in PM10 ranged between 20 and 172 ng/m^3 , with an average $90.37 \pm 57.4 \text{ ng}/\text{m}^3$ during 2001. These levels are apparently manifold higher than the European standards.

In India, except for a few reports no detailed epidemiological studies are available on health effects of PM. It was reported⁶¹ that the premature death of 51,779 people in 33 Indian cities is likely to be attributed to the high levels of PM in 1995, a rise of 28% compared to 40351 in 1991–92. The number of respiratory system-related ailments requiring medical assistance and hospital admissions in these cities due to elevated levels of PM is estimated to have increased from 19 million in 1991 to 25 million in 1995 (Table 3). In Kolkata and Delhi, cases of

hospital admissions and sickness requiring medical treatment suspected to have been caused by air pollution doubled in a span of three years, crossing the five million mark in 1995. Premature deaths in Kolkata during 1991–92 were 5726, while within a span of three years, they almost doubled to 10,647. Such sharp increase was also noticed in other metropolitan cities like Mumbai and Delhi, where premature deaths during 1991–92 were 4477 and 7491 respectively, which rose to 7023 and 9859 in 1995. During 2000, in Bandra, Parel and Kalbadevi areas of Mumbai, the premature mortality caused by PM10 estimated by Joseph *et al.*⁶² using Ostro's approach²⁸ was 64, 169 and 221 respectively.

In Bangalore, respiratory problems among children have risen threefold during the last 20 years⁶³. The incidence of respiratory ailments such as asthma during 1979,

was only 9% in the children population of the district. By 1999, it had risen to 29.5% (Table 4). Corresponding increase in the number of industries and automobiles was also witnessed. Globally, over 180,000 people die from asthma each year. India has approximately 15–20 million asthmatics and the prevalence and incidence is more amongst the affluent⁶⁴.

An association between ambient air pollutants and respiratory symptoms complex (RSC) in preschool children, a cohort of 664 children between the ages of 1 month to 4.5 years, was found at Lucknow⁶⁵. Exposure to ambient air sulphur dioxide, oxides of nitrogen and SPM on the day of the interview or in the week prior to it, was assessed by ambient air monitoring at nine centres in the city. The cumulative incidence of RSC was observed to be 1.06 and the incidence density per 100 days of follow-up was 1.63. Health costs due to air pollution in India are alarming (Table 5).

In Coimbatore, a preliminary attempt was made to know the status of respiratory disorders among people residing there through a preliminary questionnaire survey and hospital records⁶⁶. It was found that 53% of respondents suffered from respiratory disorders and other respiratory system-associated ailments. Nearly 90% of the people who are suffering from respiratory problems live in urban, suburban or industrial environment. A substantial per-

centage of respondents with respiratory problems seem to be associated with roadside shops. In certain dispensaries, the number of hospital visits made by people for respiratory problems showed positive correlation with SPM values.

In India, despite high levels of air pollution and health risks caused by these air pollutants, there is a dearth in understanding the nature of air pollution-associated diseases. A recent Indian Council of Medical Research study showed that medical colleges have low rates of publication; 20% of the 156 medical colleges had not published a single research paper during 1990–94. In addition, those who did publish managed to do so in low impact journals⁶⁷. In seven years (November 1987 – December 1994), Indian researchers had published only 19,952 papers in 1440 journals. Among these 19,916 were journal articles (as classified by Medline), nine were letters and eight clinical trials⁶⁸. Nearly three-fourths of these articles (14,822) were published in journals with impact factor less than 1. Only 58 papers were published in journals having impact factor higher than 8. Indian researchers have used just one epidemiology journal to publish the only two papers.

This would reflect the quality and quantity of research and development in medical science. Environmental epidemiological research is in a more dismal state. A brief search of the Indian journals and magazines yielded no article elaborating a cohort study or personal exposure study related to air pollution. As discussed earlier, lack of R&D in medical research, especially in the field of environmental epidemiology, is a discouraging factor in India. Steps are essential to streamline recording the morbidity in a more detailed fashion. All private hospitals and medical practitioners need to be aware of the valuable knowledge that can be generated from such records. Data should be made easily available to the research community by publishing on the web or making available statistical documents in public domain, possibly by hiding the patients' personal identification details. Such a reform would give great impetus to studies on air pollution and health, apart from many valuable contributions to medical epidemiology in a developing tropical country such as India.

Conclusion

Airborne PM is reportedly known to cause wide-ranging health effects. PM (liquid or solid particles) dispersed in air is generally classified as ultra fine (size range less than 0.1 µm), fine (0.7–1 µm) and coarse (1–200 µm). Apart from the natural sources such as forest fire, volcanic eruptions and wind-blown anthropogenic emissions from industries, vehicles, incomplete combustion of fossil fuel, careless waste treatment and disposal, commercial and residential combustion plants, and industrial combustion plants contribute to elevated levels of atmospheric PM. Many epidemiological studies published between 1989 and 2003 strongly indicate that long-term exposure to even low levels of PM are linked with deleterious health

Table 3. Estimates of health effects due to air pollution

Total	Premature death		Number of sick cases	
	1991–92	1995	1991–92	1995
Thirty-three cities	40,351	51,779	19,098,127	25,645,721

Source: <http://www.iglindia.com/whyigl/humans1>.

Table 4. Asthma cases in Bangalore

Year	Percentage of child asthma cases	Number of industries	Number of automobiles (in millions)
1979	9	4700	0.146
1984	10.5	7887	0.236
1989	18.5	14384	0.460
1994	24.5	25758	0.715
1999	29.5	40146	1.223

Table 5. Health costs due to air pollution in Indian cities

Nature of effect	Number	Cost valuation (US\$ millions)
Premature deaths	40,351	170–1615
Hospital admissions and sickness requiring medical treatment	19,800,000	25–50
Minor sickness (including restricted activity days and respiratory symptom days)	1,201,300,000	322–437
Total		517–2102

problems, including asthma, bronchitis, chronic obstructive pulmonary disease, pneumonia, upper respiratory tract and lower respiratory tract disorders. Recent reports suggest that fine PM is a risk factor for premature mortality, cardiopulmonary and lung cancer mortality.

Air quality in India is deteriorating at a fast pace ever since the advent of the industrial era, urbanization and the fast-growing vehicular fleet. Alarming levels of PM are reported in all metropolitan cities and other urban areas. Despite such high levels, lack of studies especially in the field of environmental epidemiology, is discouraging. Immediate measures are essential to streamline recording the morbidity in a more detailed fashion. Data should be made easily available to the research community by publishing on the web or making available statistical documents in public domain. Such a reform would give great impetus to studies on air pollution and health. Ambient air quality standards for particulate matter (PM10 and PM2.5) might be newly promulgated after serious consideration of existing data on public and environmental health in India and elsewhere.

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