Health Risk Assessment of Urban Suspended Particulate Matter with Special Reference to Polycyclic Aromatic Hydrocarbons: A Review

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ABSTRACT

Airborne suspended particulate matter is an important marker of air quality. The term 'particulates' includes organic and inorganic matter, nitrogen compounds, sulphur compounds, polycyclic aromatic hydrocarbons (PAHs), several heavy metals, and radionuclides. The health risks from the 'classic' pollutants sulfur dioxide, nitrogen dioxide, ozone, carbon monoxide, and particulates have been comprehensively reviewed. Alarming levels of non-classic pollutants like the PAHs have been reported globally. PAHs have been found in placental tissues of women and in umbilical cord blood samples from newborn babies. The damaged DNA in cord blood is a indication of the fate of these pollutants in the environment. Hence, a need exists for a comprehensive investigation of the human health-related aspects of exposure to particulates and PAHs in the urban environment. This paper reviews the literature on PAHs in conjunction with particulate matter on a global perspective.

KEYWORDS

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INTRODUCTION

Air quality in urban areas is of great concern these days, especially in connection with human health risks. The rate of population growth continues to increase, leading to a doubling of global population by the middle of 21st century. In parallel, the process of urbanization also continues, such that the proportion of the global population living in cities will increase from around 45% to 62% by the year 2005, creating dense centers of anthropogenic emission.

Poor control of industrial emissions and a rapid increase in ill-maintained vehicles implies that most cities in developing countries suffer from serious outdoor air pollution. The burden of ill health caused by particulate pollution, either on its own or in combination with gaseous pollutants, is enormous. At least 500,000 premature deaths and 4 to 5 million new cases of chronic bronchitis are reported each year /1/. The increase in daily mortality shows that on a global scale, 4% to 8% of premature deaths are due to exposure to particulate matter in the ambient and indoor environment /2/.

A.   1

In India, suspended particulate matter (SPM), defined as particles of various sizes and chemical composition, exceeded the annual mean guidelines of the World Health Organization (WHO) for 294 days in Delhi and 268 days in Kolkata. About 40,000 Indians die early every year because of air pollution—7,500 in Delhi,   5,700   in   Mumbai,   and
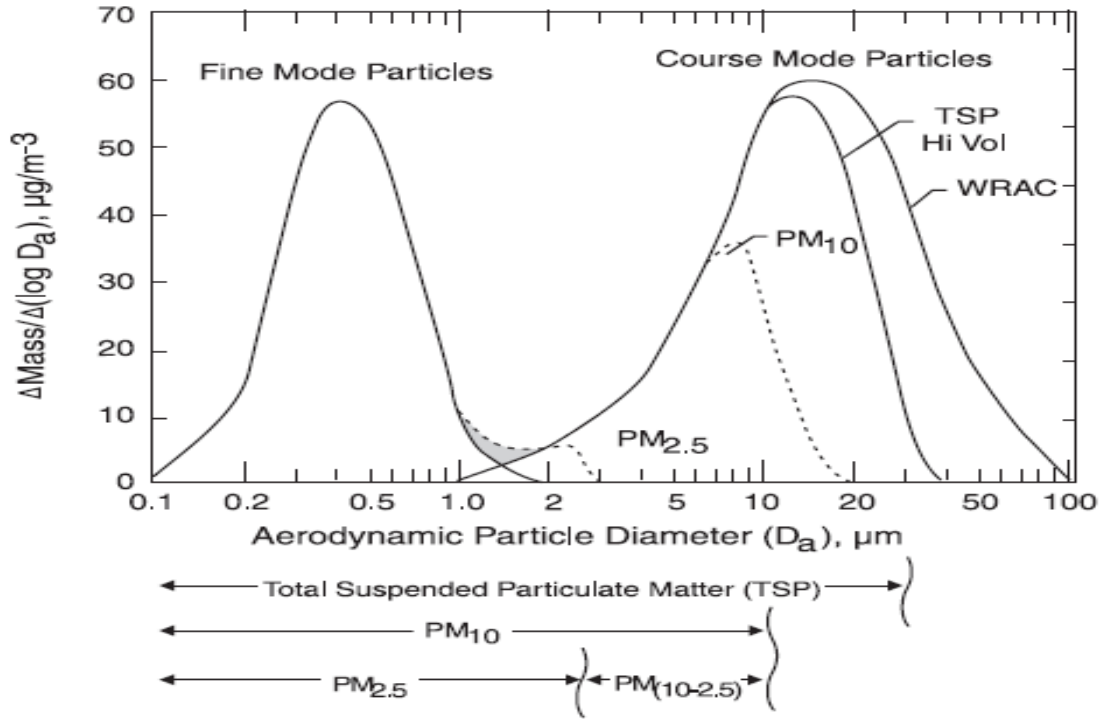

Fig 1: Representative example of mass distribution of ambient PM as function of aerodynamic particle diameter. A wide ranging of aerosol collector (WARC) provides an estimate of the full coarse mode distribution. Inlet restrictions of the high volume sampler for TSP, the PM10 sampler, and the PM2.5 sampler reduce the total mass reaching the sampler filter (source: US EPA, 1996).

4,500 in Kolkata. The World Bank estimates that Indians spend Rs. 4.55 billion every year on the treatment of diseases that are caused by ambient air pollution /3/.

Indoor air pollution in developing countries is a major environmental and public health challenge. Around 50% of people, almost all in developing countries, rely on coal and biomass in the form of wood, dung and crop residues for domestic energy. Exposure to indoor air pollution may be responsible for nearly 2 million excess deaths in developing countries and for some 4% of the global burden of disease.

PARTICULATE MATTER

Characterization

Particulate matter varies in size, ranging from a diameter of 0.0002 μm to 500 μm. Figure-1 shows the size distribution of urban particulate matter /4/. Particles having an aerodynamic diameter larger than 2.5 μm form the coarse particle fraction, particles having an aerodynamic diameter less than 2.5 μm form the fine particle fraction, and particles having <100 nm in aerodynamic diameter are classified as ultrafine particles. Table 1 presents a comparison of ambient fine and coarse mode particles /4–6/. The lifetime of particulates varies from a few seconds to several months, depending upon their settling rate, size, density of particles, and air turbulence. For example, clouds of very fine particles may drift for many miles and cause pollution at large distances from where they were emitted (Table 1).

The density of particulates in the atmosphere varies from several hundred per cm^3 in clean air to more than 100,000 per cm^3 in densely polluted air. In urban areas, the concentration may range from 60 to 2000 μg/cm^3, whereas in remote areas, particulate content can be as low as 10 μg/cm^3. Although air pollution by SPM is usually considered an urban phenomenon, in many areas of developed countries urban-rural differences in PM_{10} are either small or absent. Conversely, in developing countries, pollution by particulates in indoor environments is a serious problem in rural areas where biomass fuels are used for open-stove cooking and heating.

Particulate matter can be characterized by a range of different parameters, including the following:

* total mass or number concentration,
* mass or number size distribution,
* modality of size distribution,
* mass or count median diameter (MMD or CMD) of the distribution or as the mode in the distribution /7–9/.

Particulates have a large surface area and hence are attractive sites for the adsorption of various organic and inorganic matter. Air quality standards are expressed in terms of particles mass concentration as PM_{10} (particles below 10 μm in aerodynamic diameter) or $PM_{2.5}$ (particles below 10 μm in aerodynamic diameter). Yet, the mechanism of toxic action of airborne particles does not inform us of whether the most important variable is particle number, surface area, or mass /10–13/. Although the number size distribution of the urban aerosol is dominated by ultrafine particles, these particles contribute very little to the total mass concentration /14/. Thus. the population of particles that is responsible for the major part of the total particle count contribute little to the mass, whereas the particles contributing most to the total mass contribute little to the total particle count /10–15/. Thus, for health risk assessment, knowledge of the particle number concentrations could be more important than knowledge of the particle mass /16–18/.

TABLE 1

Properties of ambient fine and coarse mode particles

Property	Fine particles (0.1–2.5 μm aerodynamic diameter, $PM_{2.5}$)	Coarse particles (2.5–10 μm aerodynamic diameter, PM_{10})

Formed from:	Gases	Large solids/droplets
Formed by:	Chemical reactions; nucleation; condensation; coagulation; evaporation of fog and cloud droplets in which gases have dissolved and reacted	Mechanical disruption (crushing, grinding, abrasion of sprays; suspension of dusts.
Composition (examples)	Sulfate, nitrate; ammonium; hydrogen ion Elemental carbon Organic compounds (PAHs and others) Heavy metals (lead, cadmium, vanadium, nickel, iron, copper, zinc, manganese) Particle-bound water	Resuspended dusts (soil dusts, street dust) coal and oil, fly ash, metal oxides of crystal elements (Si, Al, Ti, Fe); $CaCO_3$, NaCl, sea salt; pollen, mould spores; plant/animal fragments; tire wear debris
Solubility	Largely soluble, hygroscopic and deliquescent	Largely insoluble and nonhygroscopic
Sources	Combustion of coal, oil, gasoline, diesel, wood; atmospheric transformation products of NO_X, SO_2 and organic compounds, including biogenic species (terpenes, etc) high-temperature processes, smelters, steel mills, and others.	Resuspension of industrial dust and soil tracked onto roads; suspension from disturbed soil (farming, mining, unpaved roads, etc.); biological sources; construction and demolition; coal and oil combustion; ocean spray
Lifetimes	Days to weeks	Minutes to hours
Travel distance	Hundreds to thousands of kilometers	Less than one to tens of kilometers

Suspended particulate matter can be categorized into primary and secondary aerosols.

- Primary aerosols include automobile exhaust, sea spray, and dust, and are emitted into the atmosphere directly from the source.
- Secondary aerosols, generally having a diameter, dp >2.5 μm are produced in the atmosphere from reactions involving primary or secondary gases.

Secondary components represent a significant fraction of both PM_{10} and $PM_{2.5}$ /19–21/. The dominant inorganic secondary aerosols species are sulfate, nitrate, and ammonia salts, primarily produced in a reaction of nitric acid or dinitrogen pentoxide with ammonia, sodium chloride, or dust. Sodium and chloride are also present in small quantities in the atmosphere over continental areas /19–20; 22–23/

Particulate contaminants are estimated to contain about 22 metallic elements /24/. The most abundant elements are calcium, sodium, silicon, aluminum, and iron. Considerable quantities of zinc, lead, copper, magnesium, and manganese are also present in air /25/. Iron is one of the metals that can catalyze oxidative damage in pulmonary systems /26/. Size distribution of metals in urban aerosols in Seville, Spain has shown that potentially toxic metals, such as nickel, lead, and cadmium, accumulate mainly in smaller particles ($PM_{2.5}$), with respective percentages of 72.6, 69.4, and 63.8. The maximum concentration of lead (63.7 ng/m^3) is higher than that of copper (26.7 ng/m^3), manganese (16.57 ng/m^3), nickel (1.977 ng/m^3), cobalt (0.547ng/m^3), or cadmium (0.327 ng/m^3). /27/

Organic compounds found in urban areas can be classified as hydrocarbons, thiols, mercaptants, ketones, aldehydes, and polycyclic aromatic hydrocarbons, which contribute from 10% to 40% of the $PM_{2.5}$ and PM_{10} mass in polluted urban areas. In rural areas, such compounds represent 30% to 50% of the PM_{10} mass /19–20/.

Particulate organic carbon originates from man-made activities, including petroleum residues and vehicular emissions, as well as from natural sources like plant epicular waxes and resin residues /28–31/.

Polycyclic Aromatic Hydrocarbons

The term polycyclic aromatic hydrocarbons (PAHs) covers a large group of organic compounds whose molecular structure contains two or more fused benzene rings. PAHs are widely distributed in the atmosphere, formed during the incomplete combustion of coal, oil, gas, wood, garbage, or other organic substances like tobacco and charbroiled meat. PAHs generally occur as complex mixtures like soot or environmental tobacco smoke (ETS), not as single compounds. PAHs are components of petroleum and, as such, are present in the environment from both natural and anthropogenic sources /29–33/. These pollutants are ubiquitous in ambient air, water, and soil and /29–38/. Because of their low vapor pressure, PAHs are present at ambient temperature in air, either as gases or in association with particles /38, 40/. Although certain concentrations detected in ambient air may not be directly toxic, PAHs participate in secondary chemical reactions that, in turn, produce photochemical air pollution. In Sweden, secondary harmful components like benzene and PAHs were shown to constitute a major portion of organic emissions from a recently introduced energy-efficient wood stove for residential heating and hot-water supply /39/.

The United States Agency for Toxic Substances and Disease Registry (ATSDR)[1] published a toxicology profile of 17 priority PAHs (Table 2), chosen from among more than 100 different compounds because they are suspected to be more harmful than some of the others and exhibit harmful effects that are representative of the PAHs. According to the ASDSR, background levels of some representative PAHs in the air are reported to be 0.02–1.2 ng/m³ in rural areas and 0.15–19.3 ng/m³ in urban areas.

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[1]http://www.atsdr.cdc.gov/toxprofiles/phs69.html

PAH Structures

Pericondensed

Pyrene
$C_{16}H_{10}$

Coronene
$C_{24}H_{12}$

Perylene
$C_{20}H_{12}$

Benzo[ghi]perylene
$C_{22}H_{12}$

Antanthrene
$C_{22}H_{12}$

Ovalene
$C_{32}H_{14}$

Catacondensed

Naphthalene
$C_{10}H_8$

Phenanthrene
$C_{14}H_{10}$

Tetraphene
$C_{18}H_{12}$

Chrysene
$C_{18}H_{12}$

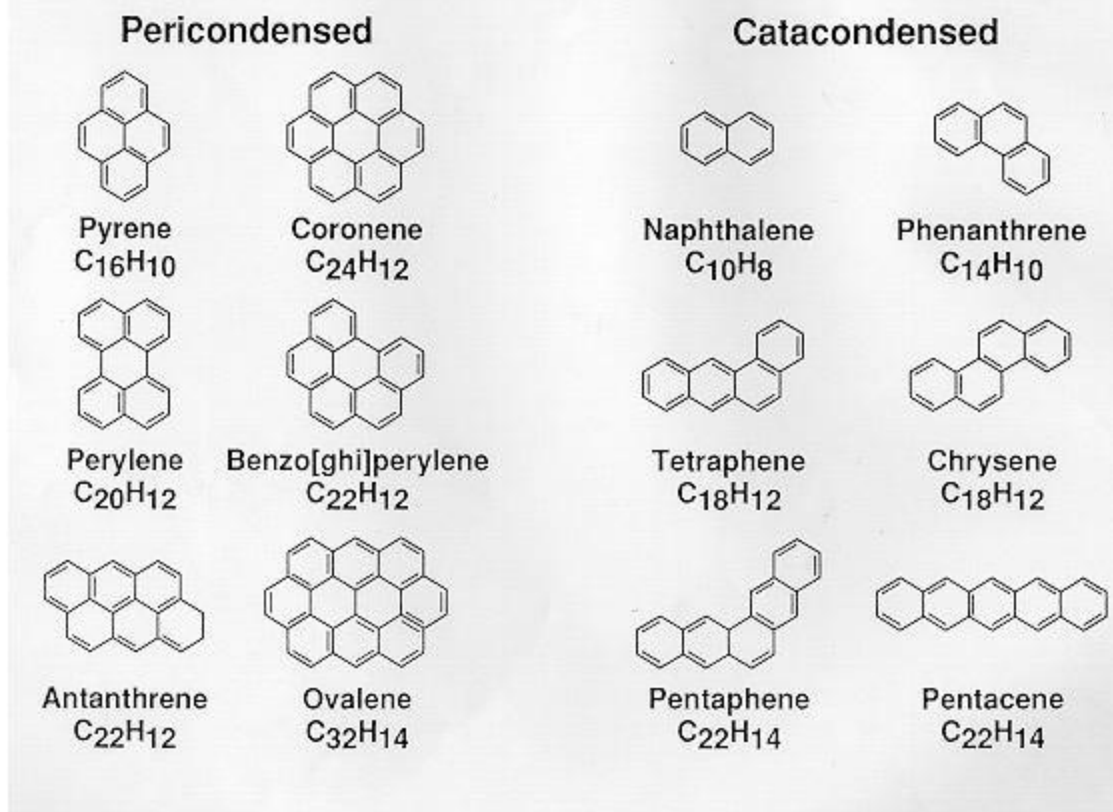Pentaphene
$C_{22}H_{14}$

Pentacene
$C_{22}H_{14}$

Fig 2: Representative PAH structures. (Source: www.kepler.arc.nasa.gov/~astrochem/PAHs.jpg)

Exposure Studies

Ambient exposure. The prevalence of different PAHs in ambient air at selected sites of world are shown in Table 3 /36, 41-46/. Among the most studied PAH is benzo(a)pyrene (BaP), a five ring ($C_{20}H_{12}$) compound that is mutagenic for human cells in culture /47/ and carcinogenic in whole animal assays /48/. Although BaP accounts for a significant fraction of the total mutagenic activity of urban aerosol extracts, several investigators have suggested that several other PAHs may be important mutagens in urban aerosol /49–52/.

TABLE 2

ATDSR priority polycyclic aromatic hydrocarbons and their emission sources

INSERT TABLE 3 HERE

Carcinogenic PAHs are present in air as vapors or adsorbed to the surfaces of particulate matter /38, 53–54/. The cumulative fractions of total PAHs in different particle size ranges in the ambient air of traffic intersections in Southern Taiwan and at rural sites are presented in Table 4 /55/. The results indicate that PAH exposure is widespread in Taiwan. Atmospheric concentrations of PAHs are strongly dependent upon the size of the carbon particulate matter, with the highest concentration being in the respirable size range. About 95% of total PAH is associated with a size class less than 3 μm in diameter /56–58/.

TABLE 4

Particle size distribution of PAHs in ambient air in Taiwan

Particle size	Fraction of total PAH (percent)	
	Traffic intersections	Rural sites
<1.0 μm	50.9%	38.3%
<2.5 μm	74.2%	56.4%
<10 μm	90.8%	85.75
<35 μm	95.9%	94.0%

In several European cities, PAH levels are higher on busy urban roads than on quiet rural roads with a lower traffic density (reviewed in /59/). A recent study of PAHs in the ambient air of six towns in Northern Greece found that in the warm months, PAHs significantly correlated with vehicular pollutants, whereas during the winter the contribution of residential heating was significant /60/. A pilot investigation in Roxbury, Massachusetts, USA, found that both $PM_{2.5}$ and PAH concentrations were greater during morning rush hours and on weekdays and significantly higher with closer proximity to the bus terminal /61/.

In Burundi, where wood combustion is used as a major energy source, mean airborne concentration of four volatile PAHs, naphthalene, fluorene, phenanthrene and acenaphthene, exceeded 1 $\mu g/m^3$, and that of benzo(a)pyrene was 0.07 $\mu g/m^3$. Naphthalene was by far the main PAH contaminant, with a mean concentration of 28.7± 23.4 $\mu g/m^3$, representing on average 60% to 70% of the total PAH concentration /62/. In Oviedo, Spain, total concentration of PAHs in air samples analyzed by GC-MS ranged from 28 to 76 ng/m^3 /63/.

In a characterization of the extent of the air toxics problem in California for the base year of 1990, risk analysis estimated a median excess individual cancer risk of 2.7E-4 for all air toxics concentrations and 8600 excess lifetime cancer cases, 70% of which were attributable to four pollutants: polycyclic organic matter, 1,3 butadiene, formaldehyde, and benzene. Although hazardous air pollutants are ubiquitous in the environment, potential cancer and noncancer health hazards posed by ambient exposures were geographically concentrated in three urbanized areas and in a few rural counties /64/.

A Romanian study on occupational versus urban environmental air yielded surprising results. Contrary to expectations, the results showed low levels of particle-bound PAHs in the occupational environment (<1 ng benzo(a)pyrene/m^3 air) and high levels in urban air (range 80-1250 ng benzo(a)pyrene/m^3). The SPM collected from the power plant exhibited non-respirable characteristics (particles >10 μm), whereas urban SPM almost exclusively contained respirable airborne particles (<3 μm). The PAH burden, combined with the enhanced probability of respiratory absorption, confers a much greater hazard potential to the urban SPM. /65/

Personal exposure. The focus of air quality assessment on ambient environmental levels has ignored

state of indoor air. Personal exposure to nine particulate-phase atmospheric PAHs was assessed among adult non-smoking volunteers in the Grenoble, France, metropolitan area, and the associated total atmospheric PAHs lifelong cancer risk was estimated. The predominant PAHs found were fluoranthene and indeno pyrene. According to the compound, the personal exposure estimates ranged from 0.13 to 1.67 ng/m^3 (yearly means). The average benzo(a) pyrene value is 0.67 ng/m^3 (95% confidence interval=0 to 2.1 ng/m^3). The total PAHs lung cancer lifelong risk is 7.8 10$^{(-5)}$ and is driven by exposure to benzo(a) pyrene. The authors concluded that although these risk estimates are 2 to 3 orders of magnitude lower than those associated with specific occupational exposures, they are of public health concern because they are spread over large urban populations /66/.

In Berlin, Germany, PAHs and diesel motor emission (estimated as elemental carbon (EC)) were determined in the interior of a car and in the passenger compartment of a subway train (below ground). The sampling represented a realistic exposure model for driving in a high traffic and polluted urban area. The respective mean values obtained during summer/winter inside the car were 1.0 and 3.2 ng/m^3 for benzo[a]pyrene, 10.2 and 28.7 ng/m^3 for total-measured-PAHs, 14.1 and 8.2 μg/m^3 for EC and in the subway 0.7 and 4.0 ng/m^3 for benzol[a]pyrene, 30.2 and 67.5 ng/m^3 for total PAHs, 109 and 6.9 μg/m^3 for EC. The significantly higher concentrations of total PAHs in the subway train were explained by the relatively high concentrations of fluoranthene and pyrene in the subway /67/.

Environmental tobacco smoke (ETS), also called passive smoking, contains over 40 known or suspected human carcinogens, including a variety of PAHs. Passive smoking is defined as an involuntary exposure to a combined but diluted cigarette sidestream smoke (gas and particle phases that are evolved from the smoldering end of a cigarette while the smoker is not puffing) and the smoke exhaled directly from smokers (mainstream smoke). Sidestream smoke contains PAHs and other cytotoxic substances in quantities much higher than those found in mainstream smoke /68-69/. The results of a recent molecular epidemiology study in Greece demonstrated that the differences in PAH-exposure profiles of subjects exposed to relatively low levels of urban air pollution are attributable to personal individual exposure to ETS. Exposures to PM$_{2.5}$ were lower in Athens (an area with moderately high levels of air pollution) during the winter than in Halkida (lower air pollution levels). Although exposures to eight particulate-bound carcinogenic PAHs were significantly higher in Athens than in Halkida, the proportion of lighter PAHs found in ETS (benzo [a]anthracene, chrysene, benzo[k]fluoranthene, and benzo[b]fluoranthene) was higher in Halkida than in Athens /70/. In Hangzhou, China, the finding that concentrations of eight PAHs in indoor air generally exceeded those in simultaneously measured outdoor air was attributed to ETS. They calculated that smoking indoors could contribute 67% of the total benzo[a]pyrene in homes /71/. Conversely, a German study in persons not occupationally exposed to PAHs found no significant difference in the PAH biomarkers between nonsmokers exposed to ETS and those not or rarely exposed to ETS /72/.

HEALTH EFFECTS

Airborne pollutants affect health in varying degrees of severity, ranging from serious illness to premature death in extreme cases. Such pollutants may produce immediate (acute), as well as long term (chronic), symptoms. Air pollution causes serious health problems, even when the levels are much lower than WHO limits /2/. Exposure to particulate matter can aggravate chronic respiratory and cardiovascular diseases, alter

host defenses, damage lung tissue, lead to premature death, and possibly contribute to cancer. Particle shape and size are critical factors controlling the extent to which particles can penetrate into the respiratory tract, how and where particles are deposited, and at what rate particles are cleared form respiratory tract. Furthermore, a large number of smaller particles have a greater reactive surface area than an equivalent mass of larger particles and have a higher likelihood of reaching the deepest regions of the lungs, namely the alveolar region. Ultrafine airborne particles below 1 μm in diameter have been related to premature death, aggravated asthma, increased hospital admissions, and increased respiratory problems /18, 73-75/.

Most of the available information on the health effects of PAHs in humans must be inferred from studies that report the effects of exposure to complex mixtures that contain PAHs. In the environment, individuals are most likely to be exposed to PAH vapors or PAHs that are attached to dust and other particles in the air. Sources include cigarette smoke, vehicle exhausts, asphalt roads, coal, coal tar, wildfires, agricultural burning, residential wood burning, municipal and industrial waste incineration, and hazardous waste sites. Several epidemiologic studies have shown increased mortality due to lung cancer in humans exposed to coke oven emissions, roofing-tar emissions, and cigarette smoke. Each of these mixtures contains benzo[a]pyrene, chrysene, benz[a]anthracene, benzo[b]fluoranthene, and dibenz[a,h]anthracene as well as other potentially carcinogenic PAHs.[2]

The impacts of air pollution on health are diverse. Indicators of health impacts include the following effects:
- daily mortality (excluding accidents/suicides);
- hospital admissions on a daily basis;
- hospital emergency visits on a daily basis;
- changes in lung function on a daily basis,
- increased medication use by asthmatics;
- respiratory symptoms in children (daily diary entries);
- school and kindergarten absence;
- longitudinal survival data;
- development of asthma in longitudinal population studies, and
- cross-sectional comparison of symptoms and lung function.

The interrelationships among these indices are seldom clear, but some aggregation is possible /76-78/.

TABLE 5

Combined effect estimates of daily mean particulate pollution[1]

Health impact	Percent change	Health impact	Percent change

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[2] http://www.atsdr.cdc.gov/toxprofiles/phs69.html; Public Health Statement for Polycyclic Aromatic Hydrocarbons (PAHs).

Increase in daily mortality		Increase in respiratory symptom report	
Total deaths	1.0	Lower respiratory	3.0
Respiratory deaths	3.4	Upper respiratory	0.7
Cardiovascular deaths	1.4	Cough	2.5
Increase in hospital usage			
(all respiratory diagnoses		Exacerbation of asthma	
Admissions	1.4	Asthmatic attacks	3.0
Emergency department visits	0.9	Bronchodilator use	12.2
Decrease in lung function		Emergency department visit	3.4
Forced expiratory	0.15	Hospital admissions	1
Peak expiratory flow	0.08		.9

[1]Percent change in health indicator per each 10 $\mu g/m^3$ increase in PM_{10}. Source: Am J Respir Crit Care Med 1996; 153: 13–50.

Mortality

Reviews and analyses of the recent epidemiological literature for acute adverse effects following exposure to particulates presented by Dockery, Pope, Schwartz, and others /79-85/ estimated these effects as percent increase in mortality associated with each incremental increase of PM_{10} by 10 $\mu g/m^3$ (Table 5). Individuals who are elderly or have preexisting lung or heart disease appear to be more susceptible than others to the adverse effects of PM_{10}.

TABLE 6
Comparison of daily mortality studies for selected cities

City	Percent change[1]	Particulate matter used in analysis	Mean Daily TSP* ($\mu g/m^3$)	Ref
United States				
Steubenville, OH	4	TSP(-1)	111	/69/
Birmingham, AL	6	PM10	87	/10/
Detroit, MI	6	TSP(-1)		/67/
Utah Valley	9	PM10 (5-Days Mean)	87	/65/
Philadelphia, PA	7	TSP (2-Days Mean)	85	/68/
St. Louis, MO	8	PM10(-1)	50	/63/
Kingston/Harriman, TN	9	PM10(-1)	55	/63/
Europe				
Athens, Greece	3 (winter)	BS (-1)	153	/71/
Paris, France	4 / 9	ln(BS) / Ln(PM13)	58 / 93	/72/
Erfurt, Germany	7	Ln(BS)	106(median)	/73/
Barcelona, Spain	4	BS	64-914 (median)	/74/)
Developing countries				
Santiago, Chile	4	Ln(PM10)	210	/75/
Bankok, Thailand	6	PM10 (-3)	118	/76/

| Beiging, China | 4 (summer) | TSP(-1) | 375 | /77/ |
| Delhi, India | 2 | TSP(-2) | 375 | /78/ |

[1]Daily mortality per 100 g/m^3 increase in TSP; *PM_{13}, PM_{10}, Pm_7, and BS values were converted to TSP using the following relationship: $PM_{13}=PM_{10}=PM_7=BS=0.55TSP$

Table 6 shows the results of daily mortality studies conducted in a number of cities in the U.S., Europe, and other developing countries /79-93/. These studies found a significant and positive relation between particulate pollution and daily mortality, with an average impact on daily mortality (excluding Delhi) of 6% for a 100 µg/m^3 in total suspended particles (TSP). In Delhi, the increase in TSP is associated with a 2.3% increase in deaths /93/. The results of sequential studies of six eastern U.S. cities indicate that the fine particle mass ($PM_{2.5}$) fraction from mobile and coal combustion sources, but not fine crustal particles, are associated with increased mortality /85, 94/. Other studies conducted by Bacharova et al. /95/ in Slovak Republic and Wojtyniak et al. /96/ in Poland did not find a significant relation between particulate pollution and daily mortality. In addition, Wojtyniak reported mixed results for four cities in his study. Conversely, Zimirou et al. /97/ reported significant respiratory deaths from particulates exposure in the city of Lyon, France.

Environmental tobacco smoke contributes to mortality from cancer and cardiac disease. Furthermore, it damagingly involves reproductive organs, the nervous system, genetic materials, and is particularly hazardous to mother and child during pregnancy and to those with a history of asthma, chronic infections, induced or earned immune deficiency, or predisposed susceptibility (reviewed in 67, 68).

Respiratory Illness

Countless respiratory problems are related to air pollution. The main pollutants that are responsible for this phenomenon are sulfur dioxide, the nitrogen oxides, ozone, and the particulates PM_{10}, and $PM_{2.5}$ /98–100/.

Respiratory tract infections. The results of many studies indicate that exposure to air pollutants increase respiratory morbidity in children /77-78, 101/. A rise in PM_{10} level by10 µg/m^3 augments the prevalence of bronchitis and chronic cough by as much as 25%, as well as causing relatively smaller increases in respiratory deaths and reduced lung function /73/. An annual average rise in PM_{10} levels by 25 µg/m^3 every year in Delhi implies an increase of 25% to 65% in bronchitis and chronic cough every year /102-103/. Consistent evidence indicates that indoor air pollution increases the risk of COPD and of acute respiratory infections in childhood, the most important cause of death among children under 5 years of age in developing countries /101/.

A growing body of epidemiological data indicates that fine particles, either alone or as part of a complex mixture of air pollutants, increase asthmatic symptoms (for review see /75, 104/). In asthmatics, epidemiological studies generally show a positive correlation between the particulate fraction of air pollution and increased morbidity, although roles for other co-pollutants (for example, ozone) are implicated as well. A rise in PM_{10} by 10 µg/m^3 increased asthma attacks by 3% /73/. Experimental studies in predisposed animals and humans have shown that PAH associated with diesel exhaust particulates can modulate the immune response by increasing allergen-specific IgE synthesis and the cytokines involved in IgE synthesis. Conflicting evidence exists with regard to air pollution and asthma in humans. Studies from several countries found no association between particle exposure and exacerbation of asthma symptoms. Such discrepancies

might be attributable to differences in methodology.

Cardiopulmonary Illness

In many parts of the world, air pollution is associated with increased mortality and morbidity rates not only for respiratory illnesses but also for cardiovascular illnesses. Epidemiological studies have shown a rather consistent relation between PM_{10} mass and cardiorespiratory illness, but no mechanism has been proposed that can fully explain this relation in terms of the toxic component of the aerosol /11–12/. Moreover, exactly what aspect(s) of mass, size, composition, or combination of these factors might be contributing to the observed adverse health effects is not known. Recent research has been directed toward gaining a better understanding of the composition of particle surface; one popular theory is that surface contaminants, such as transition metals, contribute toward ill health /105-107/. Another popular theory is that the physical characteristics (for example, number, size, shape, and aggregation properties) are important in producing health effects /16, 108/.

Average sulfur dioxide and PM_{10} levels in the USA and Europe are much lower than in many residential areas in India—for example, Delhi, Ghaziabad, Kolkata, and Ahmedabad. Certain areas in India have sulfur dioxide and particulate concentrations as high as 140 $\mu g/m^3$ and 435 $\mu g/m^3$, respectively /102-103/. A rise in sulfur dioxide or PM_{10} increases the incidence of cardiovascular deaths by 1.5% /83/. In Delhi, the annual increase in the average level of PM_{10} of 25 $\mu g/m^3$ translates to a 5% rise in cardiovascular deaths every day /102-103/.

Preliminary epidemiologic evidence suggests that a systemic response to fine particle-induced pulmonary inflammation, including cytokine release and altered cardiac autonomic function, may be part of the pathophysiologic mechanisms or pathways linking particulate pollution with cardiopulmonary disease /109/. Air-pollution-associated increase in hospital admissions for cardiovascular diseases was almost doubled in subjects with concurrent respiratory infections. For chronic obstructive pulmonary disease (COPD) and pneumonia admissions, diagnosis of conduction disorders or dysrhythmias increased the risk of PM_{10}-associated admissions. Persons with heart failure had twice the risk of PM_{10}-induced COPD admissions., suggesting that patients with acute respiratory infections or defects in the electrical control of the heart are a risk group for particulate matter effects.

Cancer

The estimated range of lung cancer risk (upper 95% confidence interval) from human epidemiological data is 1.3×10^{-4} to 2.4×10^{-3} $\mu g/m^3$. Individuals who are exposed by breathing or skin contact for long periods to mixtures containing PAHs can develop cancer. The U.S. EPA has concluded that ETS or passive smoking, a common source of PAHs, causes lung cancer in adult nonsmokers. Recent evidence has established the uptake and metabolism by nonsmokers of three major classes of carcinogens found in ETS: aromatic amines, nitrosamines, and PAHs (reviewed in /69/).

According to an ATSDR Public Health Statement,[3] the International Agency for Research on Cancer (IARC) has determined that benz[a]anthracene and benzo[a]pyrene are probably carcinogenic to humans and that benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k]fluoranthene, and indeno[1,2,3-c,d]pyrene are

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[3] http://www.atsdr.cdc.gov/toxprofiles/phs69.html; Public Health Statement for Polycyclic Aromatic Hydrocarbons (PAHs).

possibly carcinogenic to humans. The U.S. EPA has determined that benz[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-c,d]pyrene are probable human carcinogens.

Exposure to diesel exhaust also contributes to lung cancer in humans. Diesel is the main source of PAH and particulate emissions in outdoor air /7, 29–31, 110-111/. The results of a recent study on inhaled diesel soot-adsorbed benzo[a]pyerene, a model PAH, indicate that direct absorption through the alveolar epithelium is an important route of entry into the circulation of unmetabolized PAHs /112/. Chronic exposure to 1 $\mu g/m^3$ of diesel exhaust can lead to 300 additional cases of lung cancer per million individuals /113/. The direct effect of PAH and other mutagens is considered to be a maximum of five lung cancer cases each year per one million individuals /114/.

Occupational exposures to PAHs are strongly suspected to increase lung cancer risk. Two case-control studies in Germany, with 3,498 male cases and 3,541 male population controls, found elevated odds ratios (OR) (95% confidence intervals (CI)) for lung cancer risk adjusted for smoking and asbestos exposure for diesel engine exhaust (OR=1.43, 95% CI 1.23, 1.67) and PAHs (OR = 1.53, 95% CI 1.14, among others. /115/.

Cancer results from alterations in cell structure (mutation) that result in abnormal and uncontrolled cell growth. Exposure to PAHs and SPM can cause cell mutation. The estimates of unit risk for several PAHs are presented in Table 6. In Washington, DC, USA, analysis of an organic extract of an airborne particle sample for human cell mutagens indicated that ~20% of the total mutagenic fraction was accounted for in two fourth-order PAH-containing fractions that accounted for ~3% of the total extract mass. A total of 13 PAHs were identified that accounted for ~15% of total mutagenicity in the extract. Of these, the most important mutagens were cyclopental[cd] pyrene, benzo[a]pyrene, and benzo[b]fluoranthene, which respectively accounted for ~7%, ~4%, and ~2%, of the extract mutagenicity. Two previously unknown human lymphoblast mutagens, naphtha (2,1-a)pyrene and naptho(2,3-a)pyrene were also identified in the sample /49–50/.

DNA adduct formation is one indicator of genotoxicity /116/. A study in Greece found high levels of PHA-DNA adducts in non-smoking students living in the Halkida Institute campus, which has minimal urban air pollution. Conversely, students living in Athens, where personal PAHs exposures are significantly higher that those in Halkida, showed the lowest DNA adduct levels. In the group suffering minimal exposure to urban air pollution, three bio markers of exposure signaled that ETS was a significant determinant of DNA damage /117/.

Neonatal Health Hazards

Human and experimental evidence indicates that the developing fetus may be more susceptible than the adult to the effects of certain carcinogens, including some PAHs. Factors that can modulate susceptibility include proliferation rates, detoxification capabilities, and DNA repair capacity. In India, human placenta, umbilical cord blood (UCB), maternal blood, and breast milk from mothers were analyzed for the presence of selected PAHs. Benzo(a)pyrene, dibenzo(a,c) anthracine, and chrysene were detected in all four sample types. The relatively high concentrations of all three PAHs in both UCB and breast milk samples implies that the developing fetus /newborn can be exposed to these contaminants in utero /118/. Chrysene, benz[a]anthraxcene, benzo[a]pyrene, benzo[b]fluoranthene, indeno[1,2,3c,d]pyrene, dibenz[a,h]anthracene,

and benzo[g,h,I]perylene were also detected in placentas from 200 women in Ukraine /119/.

Analysis of UCB from 163 babies revealed that the blood DNA of each child had been damaged by PAH /120/. Exposure to PAH and PM_{10} can result in the birth of babies who are 160 grams lighter and 1.04 cm shorter than normal. If mothers are simultaneously exposed to $100\mu g/m^3$ of sulfur dioxide and 400 mg/m³ of particulates, then an average of two of every ten babies will be premature /92/.

A recent molecular epidemiologic study analyzed PAH-DNA adducts (as indicators of DNA damage from PAHs and other aromatics) and plasma cotinine (as an internal dosimeter of cigarette smoke) in paired blood samples collected at birth from Polish mothers and newborns /121/. In mother/newborn pairs from whom the blood sample was drawn concurrently (within 1 h of each other), levels of all of the three biomarkers were significantly higher in the newborn than in the paired maternal blood samples. The results suggest reduced detoxification capabilities and increased susceptibility of the fetus to DNA damage, especially in light of experimental evidence that transplacental exposures to PAHs are 10-fold lower than paired maternal exposures. The results have implications for risk assessment, which currently does not adequately account for sensitive subsets of populations.

Airborne lead is associated with particles in size range below 1 μm /122/. In urban areas of developing countries where most fuel is unleaded, concentrations of lead range from less then 0.1 to 1 μg/m³. Traffic-related lead levels range between 0.3 and 1 μg/m³, with extreme annual mean values between1 2–5 μg/m³ /2/. Lead is a well known cause of encephalopathy (a disease of the brain) in children, which often results in death or permanent brain damage. In children, the inhalation of lead can permanently lower IQ, damage emotional stability, cause hyperactivity and reduce the ability to concentrate /123-124/.related lead levels range between 0.3 and 1 μg/m³, with extreme annual mean values between1 2–5 μg/m³ /2/. Toxic effects of air pollution can also lead to mental problems, including anxiety and change in mood, cognition, and behavior. Toxic air pollutants also interfere with the development and adult functioning of the nervous system /125/.

TABLE 7

Estimate of unit risks for several polycyclic aromatic hydrocarbons

Compounds	Relative potency range compared with BaP	Unit risk $[\mu g/m^3]^{-1}$
Anthracene	0.28-0.32	(2.4 - 2.8) x 10^{-2}
Benz(a)anthracene	0.14-0.145	(1.2 – 1.3) x 10^{-4}
Benzo(a)pyrene	1	(8.7) x 10^{-2}
Benzo(b)fluoranthene	0.1-0141	(0.87 - 1.2) x 10^{-2}
Benzo(j)fluoranthenecor	0.045-0.1	(0.4 – 0.87) x 10^{-2}
Benzo(k)fluoranthene	0.01-0.1	(8.7 - 87) x 10^{-4}
Chrycene	0.001-0.10	(8.7 – 870) x 10^{-5}
Cyclopental(cd)pyrene	0.012-0.1	(1.0 – 8.7) x 10^{-3}

Dibenzo(a,e)pyrene	1	$(8.7) \times 10^{-2}$
Dibenzo(a,c)anthracene	0.1	$(8.7) \times 10^{-3}$
Dibenzo(a,h)anthracene	0.89-5	$(7.7 - 43.5) \times 10^{-2}$
Dibenzo(a,l)pyrene	100	$(8.7) \times 10^{-0}$
Dibenzo(a,e)fluoranthene	1	$(8.7) \times 10^{-2}$
Dibenzo(a,h)pyrene	1-1.2	$(8.7 - 10.4) \times 10^{-2}$
Dibenzo(a,i)pyrene	0.1	$(8.7) \times 10^{-3}$
Fluoranthene	0.001-0.010	$(8.7 - 87) \times 10^{-5}$
Indeno(1,2,3,-cd)pyrene	0.067-0.232	$(5.8 - 20.2) \times 10^{-3}$

Overall, the evidence presented here supports the view that suspended particulate matter (SPM) is associated with adverse health effects, depending upon the type and amount of pollutant present, the duration of exposure, and the state of health, age, and level of the person exposed. Respirable particulate matter (fine and coarse) contains high levels of carcinogenic PAHs. A World Bank study on the health effects of air pollution in Delhi reveled that SPM in Delhi alone led to the premature death of 7,491 people in 1991 and 1992. A repeat of the study for the year 1995 revealed an increase to about 10,000 in just 3 years, meaning a death rate of one person per hour due to air pollution /3/.

CONCLUSIONS

Polycyclic aromatic hydrocarbons are found globally, both in developing and developed countries. Evaluating the contribution of any individual PAH to the total carcinogenicity of these mixtures in humans It is difficult because of the complexity of the mixtures and the presence of other carcinogens. Despite these limitations, the U.S. ATSDR has concluded that reports of this nature provide qualitative evidence of the potential for mixtures containing PAHs like benzo[a]pyrene, chrysene, benz[a]anthracene, benzo[b] fluoranthene, and dibenz[a,h]anthracene to cause cancer in humans. The presence of PAHs in placental tissue and the blood of newborn babies indicates that the chemical characteristics of airborne particles is deteriorating worldwide from the public health point of view. Hence, the prevention, control, and abatement of PAH contamination, along with particulates, have become an urgent need requiring the knowledge of the nature, source, and extent of pollution. Despite the ever increasing amount of information emerging from a wealth of individual air pollution studies, a lack of consistent environmental monitoring data on air toxics makes it difficult to assess the extent of low-level, chronic, ambient exposures to priority PAHs. Because reports on the health effects of particulates are divergent, conducting more epidemiological studies is imperative to establish and enforce preventive policies in air pollution. Indoor air pollution is a major global public health threat. Most governmental regulations and standards focus on ambient environmental levels of pollutants rather than on personal exposure. This approach fails to evaluate the state of indoor air. The existing evidence suggests, however, that the regulation of indoor air and the implementation of preventive

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measures should be put into practice.

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