The World Health Organization (WHO) estimates that every year 800,000 people die prematurely from lung cancer, cardiovascular and respiratory diseases caused by outdoor air pollution. Other adverse health effects include increased incidence of chronic bronchitis and acute respiratory illness, exacerbation of asthma and coronary disease and impairment of lung function. This note outlines how cities can estimate health gains to their residents as they take steps to reduce outdoor air pollution. The most significant health effects of air pollution have been associated with particulate matter (PM) and to a lesser extent with ground-level ozone. PM is a mixture of many subclasses of pollutants which vary in size and chemical composition. Most studies have examined the health effects based on particle size. Much less is known about the impact on health of varying PM chemical composition. The largest health impacts have been associated with particles small enough to penetrate deep into the respiratory tract: fine particles (PM$_{2.5}$, smaller than 2.5 microns or 2.5 x $10^{-6}$ meters in diameter) and PM$_{10}$ (smaller than 10 microns). Combustion, metallurgical processes, automobile exhaust and secondary sulfate and nitrate particles formed by the atmospheric transformation of sulfur dioxide (SO$_2$) and oxides of nitrogen (NO$_x$) are the main sources of these smaller particles. Elevated levels of NO$_x$ and SO$_2$ also result in higher hospital admissions and emergency room visits, but these effects are small compared to those of PM. Similarly the health effects of particles larger than 10 microns, arising primarily
from resuspended dust and soil, are also small. Since the health damage caused by elevated fine particulate concentrations in South Asia is much higher than that of all other pollutants combined, the remainder of this note focuses on studies on PM\(_{10}\) and PM\(_{2.5}\).

Air pollution is the presence of pollutants in the atmosphere from anthropogenic or natural. Substances in quantities likely to harm human, plant or animal life; to damage human-made materials and structures; to bring about changes in weather or climate; or to interfere with the enjoyment of life or property (Cooper and Alley, 1986; Elsom, 1987).

**Air pollutants**

**Suspended Particulate Matter**

Principal sources of contribution to particulate matter include road dust, emissions from foundaries, testing of diesel engines and automobile exhausts. (Khare *et al.*, 1985). Environments of India and of many tropical developing countries are characterized by high levels of suspended particulate matter (SPM). This is largely due to thin forest cover and prevalence of turbulence for prolonged periods resulting in resuspension of ground dust. Both these factors contribute to higher SPM with large size particles. Abundance of sunlight promotes photochemical reactions in the atmosphere and should normally increase the probability of occurrence of smog. The reasons for this are also related to the composition of the atmospheric particulates, even though in this case the leading role is played by gaseous components. Still we do not find frequent smog formation except during winters. Health hazards due to inhalation of aerosols depend not only on their total concentration in the ambient atmosphere but also on their size distribution and composition (Mishra, 1988).
PM-2.5 can also form as a result of solidification of volatile metal salts as crystals following cooling of hot exhaust gases from vehicles in ambient air (Winchester et al., 1993). Schwartz (1994a) study on acute exacerbations of chronic obstructive pulmonary disease (COPD), chronic bronchitis or emphysema has been associated with short-term exposure to air pollution.

PM-2.5 can remain suspended in the air and travel long distances. Sulfates, which are commonly generated by conversion from primary sulfur emissions, make up the largest fraction of PM-2.5 by mass (Pope et al., 1995). For vehicles fueled by leaded gasoline, lead compounds account for a major portion of PM emissions. PM emitted by diesel-fueled vehicles consists of soot formed during combustion, heavy HC condensed or absorbed on the soot, and sulfates. These emissions contain polycyclic aromatic hydrocarbons (PAH). In older diesel-fueled vehicles the contribution of soot to PM emissions is between 40 and 80 percent. With the advance of emission control measures in engines, however, the contribution of soot has been reduced considerably. Heavy HC referred to as the soluble organic fraction of PM, originate from lubricating oil, unburned fuel, and compounds formed during combustion (Walsh, 1995). Black smoke, associated with the soot portion of PM emitted by diesel-fueled vehicles, is caused by oxygen deficiency during the fuel combustion or expansion phase. Blue or gray smoke results from vaporized lubricating oil, and white smoke occurs during engine start-up in cold weather. Gasoline-fueled vehicles have lower PM emission rates than diesel-fueled vehicles. PM emissions from gasoline-fueled vehicles result from unburned lubricating oil and ash-forming fuel and oil additives (Faiz et al., 1995).

The World Bank (1996b) study showed that pollution is concentrated among a few industrial sub-sectors and that a sector’s contribution to pollution is often disproportionate to its contribution to industrial output. For example, petroleum refineries, textiles, pulp and paper, and industrial chemicals produce
27% of the industrial output but contribute 87% of sulphur emissions and 70% of nitrogen emissions from the industrial sector. Likewise, iron and steel, and non-metallic mineral products, produce about 16% of the industrial output but account for 55% of the particulate emissions.

Sulphur dioxide, nitrogen dioxide and suspended particulate matter (SPM) are regarded as major air pollutants in India (Agrawal and Singh, 2000). The major anthropogenic sources of particulate matter (coarse and fine particulates) are re-suspension of industrial dust and soil, suspension of soil (farming, mining, unpaved roads), combustion of coal, oil, wood and diesel exhaust particulate. In urban areas, about 50% of the particulate matter is estimated to be from traffic generated emissions (Worbel et al., 2000). Studies on particle size distribution and its elemental composition in the ambient air of Delhi indicated that metal concentration in fine fraction (<2.1µm) exceeds by a factor up to 6, as compared to that in coarse fraction (>2.1µm-10µm) (Balachandran et al., 2000). The level of particulate matter in the urban environment is of great concern; as several studies indicate that particulates may induce severe effects on public health (Khillare et al., 2004).

**Sulphur dioxide**

The transport sector's contribution to global SO$_2$ emissions is estimated at 2 to 6 percent. On a global scale, about 60% of the SO$_2$ was estimated from coal combustion (Pitts-Finlayson and Pitts, 1986). Since coal and petroleum often contain sulfur compounds, their combustion generates sulfur dioxide. Further oxidation of SO$_2$, usually in the presence of a catalyst such as NO$_2$, forms H$_2$SO$_4$, and thus acid rain. This is one of the causes for concern over the environmental impact of the use of these fuels as power sources.

Sulfur dioxide is produced by combustion of sulfur-containing fuels, such as coal and fuel oils. Power plants and factories emit 90% to 95% of the sulfur
dioxide and 57% of the nitrogen oxides in the United States. Almost 60% of the SO\textsubscript{2} emissions are released by tall smoke stakes, enabling the emissions to travel long distances. (Miller, 1990) As emissions of sulfur dioxide and nitric oxide from stationary sources are transported long distances by winds and form secondary pollutants such as nitrogen dioxide, nitric acid vapor, and droplets containing solutions of sulfuric acid, sulfate and nitrate salts. These chemicals descend to the earth's surface in wet form as rain or snow and in dry form as a gases fog, dew, or solid particles and is known as acid deposition or acid rain. Annual global emissions of SO\textsubscript{2} are estimated at 294 million tons and out of this, 160 million tons are from anthropogenic sources. About 90 percent of these emissions originate from the northern hemisphere; the United States and the republics of the former Soviet Union are two biggest sources (UNEP, 1991).

**Nitrogen dioxide**

Motor vehicles are the main contributors to anthropogenic NO\textsubscript{x} emissions. Worldwide anthropogenic NO\textsubscript{x} emissions for 1991 are estimated at 93 million tons. 43 percent of which were contributed by the transport sector, 32 percent by the power sector, 12 percent by the industrial sector, 8 percent by the residential and commercial sectors, and 5 percent by other sources. Developing countries contribute 26 percent of global NO\textsubscript{x} emissions among which the transport sector accounts for 49 percent, the power sector, 25 percent; the industrial sector, 11 percent; the residential and commercial sectors, 10 percent; and other sources, 5 percent (OECD/IEA, 1991) of NO\textsubscript{x} emissions.

The main anthropogenic source of NO\textsubscript{x} in ambient air is fossil fuel combustion in motor vehicles and industrial processes, particularly in power generation. High-temperature combustion results in the oxidation of atmospheric N\textsubscript{2}, NO and then to NO\textsubscript{2}. Motor vehicle emissions near busy streets can result in high local NO\textsubscript{x} concentrations. The typical diurnal NO\textsubscript{x} pattern
consists of a low background concentration, with morning and late afternoon peaks resulting from rush-hour traffic. Nitrogen in fossil fuels such as coal can be oxidized to NO\textsubscript{2} under oxygen-rich combustion conditions. NO\textsubscript{2} and NO are both formed naturally as a result of bacterial metabolism of nitrogenous compounds and to a lesser extent, from fires, volcanoes, and fixation by lightning. The generation of troposphere ozone and other photochemical oxidants is initiated with photolysis of NO\textsubscript{2}, whereas NO acts as an ozone scavenger (Lipsett, 2001).

**Ozone**

O\textsubscript{3} has been recognized since the 1950s as the principal component of photochemical smog. In the troposphere, it is formed by the action of solar UV radiation on nitrogen oxides and reactive hydrocarbons, both of which are emitted by motor vehicles and many industrial sources. Photochemical O\textsubscript{3} formation tends to be greatest on warm, sunny days. The typical profile of troposphere O\textsubscript{3} formation in populated areas is characterized by a broad peak that lasts from the late morning until the late afternoon or early evening. However, large-scale transport may result in elevated O\textsubscript{3} concentrations that extend over thousands of square miles to include rural areas far removed from the precursor sources. Thermal inversion height, wind speed and direction, addition of other O\textsubscript{3} precursors along an air mass trajectory, and other factors affect the temporal O\textsubscript{3} patterns downwind, so that peak concentrations may occur anytime from noon until late in the evening. Human activities are major sources of O\textsubscript{3} precursors, although the latter are also generated by non-anthropogenic processes that include the intrusion of stratospheric O\textsubscript{3}, the action of lightning on molecular oxygen, and chemical reactions involving naturally occurring nitrogen oxides and organic compounds, such as biogenic methane and other volatile organic compounds (Lioy and Dyba, 1989). Ground-level
ozone concentrations depend on the absolute and relative concentrations of its precursors and the intensity of solar radiation, which exhibits diurnal and seasonal variations. Thermal inversions increase ground-level ozone concentrations (World Bank, 1996a).

**Hydrocarbons**

Nonmethane hydrocarbons (NMHC) are reactive in forming secondary air pollutants; they are focus of air quality studies (Horowitz, 1982). Hydrocarbons (HC) are emitted from natural and anthropogenic sources. Natural sources include anaerobic decomposition of plants in swamps and marshes, seepage from natural gas and oil fields, and emissions from trees. The first two sources mainly produce methane, and the third source produces photochemically reactive HC (Horowitz, 1982). Anthropogenic emission sources include motor vehicles, gasoline and solvent storage tanks and transfer stations, petroleum refineries, and chemical and petrochemical plants. HC emissions from motor vehicles occur from unburned fuel or from partial combustion of fuels. About 55 per cent of HC emissions from gasoline-fueled vehicles with no emission controls originate in the exhaust system, 13 to 25 % come from the crankcase blow-by, and 20 to 32 per cent evaporate in the fuel lines, fuel tank, and carburetor. Methane constitutes 5 to 15 per cent of HC emissions from vehicles not equipped with catalytic converters and up to 40 percent of exhaust HC from catalyst-equipped vehicles. This is because the catalysts are less effective in oxidizing methane than other hydrocarbons. In the presence of ultraviolet radiation, NMHC and NOx react with oxygen to form ozone in the troposphere. The reaction time varies from less than an hour to several days depending on the reactivity of the NMHC. Motor vehicles emit toxic HC, including benzene, 1,3-butadiene, aldehydes, and polycyclic aromatic hydrocarbons (PAH). PAH are emitted at a higher rate in the exhaust of diesel-fueled vehicles than gasoline-
fueled vehicles. The reactions that form ground-level ozone also produce small quantities of other organic and inorganic compounds such as peroxyacetyl nitrate (PAN) and nitric acid (Horowitz, 1982).

Benzene constitutes 63 to 85 per cent of the toxic emissions in exhaust from gasoline-fueled cars equipped with fuel-injected engines and new technology, and 36 to 65 per cent from older model-year cars equipped with carbureted engines and catalytic converters (AQIRP, 1991). Aldehydes and 1,3-butadiene are not present in gasoline, diesel fuel, ethanol, or methanol but are present in their exhaust emissions as partial combustion products. Aldehydes are also formed in the atmosphere from other mobile source pollutants and have a high photochemical reactivity in ozone formation. The major types of aldehydes formed include formaldehyde and acetaldehyde. Combustion of ethanol favors acetaldehyde emissions and combustion of methanol favors formaldehyde emissions. Controlled gasoline-fueled cars have higher emissions of formaldehyde than acetaldehyde. Uncontrolled gasoline-fueled vehicles emit 0.6 to 2.3 grams of aldehydes per litre and uncontrolled diesel-fueled vehicles emit 1 to 2 grams of aldehydes per liter (Wijetilleke and Karunaratne, 1995).

Benzene is an aromatic HC present in gasoline. About 85 to 90 per cent of benzene emissions come from exhaust and the remainder comes directly from gasoline evaporation and through distribution losses. The benzene in exhaust originates both from partial combustion of other aromatic HC compounds in gasoline such as toluene and xylene; and from the benzene already in gasoline (Faiz et al., 1996).

**Heavy Metals**

Inorganic lead in ambient air also originates from emissions from coal combustion and various lead-based industries such as lead smelters and lead battery plants. Although lead in gasoline accounts for less than 10 per cent of all
refined lead production, about 80 to 90 per cent of lead in global ambient air originates from combustion of leaded gasoline (GEMS, 1988). Diesel fuel additives such as barium, calcium, or magnesium reduce smoke emissions, but increase sulfate emissions. These additives may also increase polycyclic aromatic hydrocarbons emissions. Copper based additives can reduce PM emissions, but may catalyze the reaction between HC and trace amounts of chlorides in diesel fuel to form dioxins, which are emitted in the exhaust (Faiz et al., 1996). The use of barium and copper in diesel fuel additives is of concern because of their toxic properties

Reports suggest that air in industrial and metropolitan areas in more heavily contaminated with heavy metals than air in rural areas (Meyer et al., 1999). Trace metals are emitted into the atmosphere mainly as a consequence of high temperature process such as combustion, roasting and metallurgical operations. Air borne toxic trace metals are found mainly in the suspended particulate matter dispersed in air (Dwivedi and Seth, 2001). Particulate matter of a diameter under 10um (PM_{10}) is a mixture of solid and liquid particles in the air and major source of these fine particles are diesel engine exhaust, food cooking operations, and dust from wood burning (Zheng et al., 2002). Also, the abrasion of tyres on the roads adds Cd to street-dust. Respirable dust is an important air pollutant of concern on account of its ability to reach alveoli of human lungs. In general, air pollution is injurious to humans and plants, since it unreasonably interferes with the comfortable mode of life and property. In particular, heavy metal contamination causes serious manifestation on the reproductive and nervous systems. A study on concentration and distribution of heavy metals in urban airborne particulate matter in Frankfurt am Main, Germany revealed that the highest values occurred at the main street with a large volume of traffic (Zereini et al., 2005). WHO/UNEP report (1992) reveals that the metropolitan cities of India are some of the most polluted cities
Air pollution has long been recognized as a potentially lethal form of pollution. Entry of pollutants into the atmosphere occurs in the form of gases or particles. Continuous mixing, transformation, and trans-bound transportation of air pollutants make air quality of a locality unpredictable. The growth of population, industry, and number of vehicles and improper implementation of stringent emission standards make the problem of air pollution still worse (Ravindra et al., 2001). India has 23 major cities of over 1 million people and ambient air pollution levels exceed the WHO standards in many of them (Gupta et al., 2002). The single most important factor responsible for deterioration of air quality in the cities is the exponential increase in the number of vehicles. Vehicular pollution contributes to 70% of total pollution in Delhi, 52% in Mumbai, and 30% in Calcutta (C.P.C.B., 2003 and Gokhale and Patil, 2004). In the developing countries, air quality crisis in cities is attributed to vehicular emission which contributes to 40-80% of total air pollution (Ghose et al., 2005). Vehicular traffic is the main source of particulate air pollution in Lucknow city (Sharma et al., 2006).

Anthropogenic air pollutants enter the atmosphere from fixed and mobile sources. Primary air pollutants like carbon monoxide, hydrocarbons, sulphur dioxide, nitrogen oxides, etc. enter the atmosphere directly from various sources. Adverse health effects of air pollutants on human beings have been observed by Carnow et al. (1969). The respiratory tract can also be affected (Gargil, 1978). Air pollution is strongly associated with the aggravation of asthma (Whittemore and Korn, 1980). The animal study of Gross (1981) has also demonstrated a consistent association between the air pollutants and the altered lung function. The decrease of pulmonary function is due to exposure to polluted air (Bedi et al., 1984).
It has been reported that high levels of pollution affect mental and emotional health too. Amongst the symptoms, feeling of fatigue, exhaustion, low mood, nervousness, irritation of eyes and stomach aches have shown a significant association with air quality. Typically, fine particles originate by condensation of materials produced during combustion or from atmospheric reactions. Suspended particulate matter comprises a wide variety of substances, which include inorganic and organic carbon (containing polycyclic aromatic hydrocarbons), acidic or neutral sulfates and nitrates, fine soil dust, residues of lead and other metals, asbestos and other fibers. These particulates act as nuclei or substrates on which many other pollutant gases could be adsorbed and form secondary pollutants and result in synergistic health effects (Krishna Murthy and Shanmukhappa, 1987). The pollutants in air namely SO$_2$, NO$_x$ and Suspended particulate matter (SPM) damage the human respiratory and cardio respiratory systems in various ways. The elderly, children, smokers and those with chronic respiratory diseases are most vulnerable. Elevated lead levels in children have been associated with impaired neurological development as measured by lowered IQ, poor school performance and behavioral difficulties (Sapru, 1987). Moweli and Subbayya (1989) observed harmful effects of the air pollutants on the health of human beings.

Experimental human exposure studies have mainly been carried out using exposure chamber set-ups with controlled diesel exhaust challenges. It is critical to ensure that the method is designed so as to maintain a certain relationship between the particulate and gaseous components and to obtain particles of the same size and chemical properties throughout the exposure series. A unique and carefully validated system for exposures has been particularly useful (Rudell et al., 1990). Inhalation of lead from leaded petrol emissions is an important source of lead exposure. Few studies have related lead to structural damage or impaired functions of the lungs (Lippman, 1990).
PAHs are highly lipid-soluble and are absorbed by the lungs and gut of mammals. PAHs may penetrate into the bronchial epithelium cells where metabolism takes places. The carcinogenicity of individual PAH requires metabolic activations and conversion into their corresponding ultimate carcinogenic metabolites which are responsible for DNA alkylations and the initiation step in the complex mechanism, associated with chemically induced cancer (Harris, 1988; Dipple and Bigger, 1991). Some studies have suggested that lung function decline and respiratory diseases are associated with proximity to roads with heavy traffic, traffic density or exposure to traffic-related air pollution (Nitta et al., 1993). Few studies have been conducted in India (Tyagi, 1993) and methodological aspects of measuring polycyclic hydrocarbons (PAHs) in urban atmosphere (Tyagi, 1994).

Deterioration of air quality is a major environmental problem in many large urban centers in both industrial and developing countries. Although urban air quality in industrial countries has been controlled to some extent during the past two decades, in many developing countries it is worsening and a major threat to the health and welfare of people and the environment (WHO/UNEP, 1992). The health effects of pollutants depend on many factors, including the number and age group of exposed people and their health status, ambient concentrations and types of pollutants and dose response functions. Increasing urbanization and industrialization result in more energy demand, which generally leads to higher emissions of air pollutants. Emissions from fixed sources such as refineries, power and industrial plants, commercial and residential buildings, chemical and fuel storage facilities, and gasoline stations are the main sources of air pollution in some cities of the world. But motor vehicles are the main contributor to deteriorating ambient air quality.

Irrespective of whether or not vehicle generated pollutants contribute to the increased numbers of sensitized individuals in urban areas, patients with
airway diseases such as asthma have been found more adversely affected than
the normal population to inhalation of air pollution components and in
particular acid aerosols (Hobbs and Mauderly, 1991).

Brunekreef et al. (1991) analyzed data from three studies of children
exposed to air pollution and pulmonary function responses to investigate
whether the observed variability in pulmonary function indicates a difference in
sensitivity or is due to random inter-occasion variability among. Many
epidemiological studies of the acute effects of ambient air pollution on
respiratory health using repeated measurements of pulmonary function have
been reported (Pope et al., 1991; Braun-Fahrlander et al., 1992). These studies
demonstrated that daily increases in air pollution, especially particulate air
pollution, are negatively associated with pulmonary function. Many studies
have also documented that some children are more sensitive to air pollution than
others (Pope and Dockery, 1992). Children, who have a history of symptomatic
asthma or chronic lung diseases screened by questionnaire, or clinical cases of
asthma, have been reported to be more susceptible to air pollution than
asymptomatic children.

Mohan et al. (1992) also observed that vehicular traffics are responsible
for indoor and outdoor air pollution. At least 500,000 premature deaths and 4 to
5 million new cases of chronic bronchitis are reported each year (World Health
found consistent and coherent associations between air pollution and various
outcomes (e.g. respiratory symptoms, reduced lung function, chronic bronchitis,
and mortality). Winchester et al. (1993) study reported that particles are
responsible for the aggravated asthma. The risks of adverse health effects are
reported greater when particles enter the tracheo-bronchial and alveolar portions
of the respiratory system. PM10 particles are small enough to penetrate deep into
the lungs and so potentially pose significant health risks (Folinsbee, 1992;
Studies have found significant associations between concentrations of air pollutants and health outcomes, particularly in children, with and without asthma (Koenig et al., 1993). Inhalation of fumes is associated with recurrent colds, chronic bronchitis and hyperactive airways (Smith, 1993). Despite the increasing evidence of negative impact of air quality on human health (Dockery et al., 1993a, b; Pope et al., 1995), not much data on ambient air quality, a prerequisite for health studies, is available for most of the medium size cities or towns in India, although a large population lives in these cities or towns.

Gupta and Vidya (1994) considered that increased vehicular traffic during tourism periods are one of the major activities which contribute SPM in the atmosphere. Air pollution has been of great concern since the major industrial events associated with air pollution happened in Europe. Dockery and Pope (1994) reviewed studies of the acute respiratory effect of particulate air pollution. They found the observed health effects on pulmonary function changes were modest, approximately 0.15 percent decrease in FEV1 or FEV0.75 and a 0.08 percent decrease in peak flow per\(10\mu g/m^3\). Although the magnitude of the lung function change estimates were relatively small, there might be persons with responses much larger than average. Total suspended particulate matter was as high as 3000 \(\mu g/m^3\). Similar patterns of elevated morbidity and mortality rates, primarily in patients with preexisting lung disease, have been documented in other acute episodes of air pollution in the past (Schwartz, 1994b).

Environmentalists claim that living in an Indian metropolitan city is like smoking 10-20 cigarettes every day. More than 40,000 people die prematurely every year because of air pollution, says a World Bank report, of which Delhi’s share is the highest (19%) (David, 1994). Several studies have shown the adverse effects of ambient air pollution on respiratory health, and exposure to components of air pollution enhances the airway response to inhaled allergens in
susceptible subjects. Indeed, in most industrialised countries, people who live in urban areas tend to be more affected by allergic respiratory diseases than those of rural areas (UNEP and WHO, 1994).

WHO, 1995 has attempted the evaluation of the exposure and its impact in developed countries. 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 Lucknow. 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. The concentration of ambient particulate matter with aerodynamic diameter ≥ 10µm (PM$_{10}$), primarily in combination with high sulphate particulate matter, has been associated with increased hospitalization for asthma (Koren, 1995). Ambient air levels exceeding the WHO levels in 36 major Indian cities and towns result in 40 thousand premature deaths, around 19 million respiratory hospital admissions and sickness requiring medical treatment and also 1.2 billion incidences of minor sickness annually (Brandon and Homman, 1995). The effects of exposure to ambient air pollution on lung function have been the focus of a great deal of epidemiologic and experimental research, especially over the last two decades (American Thoracic Society, 1996). The vast majority of epidemiologic and controlled exposure studies have investigated the effects of acute exposure on short-term responses for a variety of measures of lung function derived from forced expiratory maneuvers. Most of these studies find small effects of daily fluctuations of concentrations of ambient air pollutants on daily changes in lung function (American Thoracic Society (ATS), 1996). The polluted air increases the respiratory symptoms in peoples (Vigotti et al., 1996).
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 Lucknow (Awasthi et al., 1996). 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. Air pollutants are responsible for increase in the number of patients of asthma (Timonen and Pekkanen, 1997 and Lipsett et al., 1997).

Similar projects for developing countries have not yet been performed systematically. In urban areas of developing countries, human exposures to outdoor gaseous air pollutants often exceed WHO guideline values by factor of 5-10 (WHO, 1997). Recent attempts to estimate the global burden of disease due to air pollution from suspended particulate matter (Schwela, 1996 and 1998). Peden et al. (1996) found increased neutrophils, cytokine levels, and evidence of epithelial permeability in BALF 18 h after short-term O₃ exposure, in subjects with mild asthma versus healthy subjects. In addition to increased neutrophils, Peden et al. (1996) also found increased eosinophils in BALF 18 h after O₃ exposure. The higher post-O₃ cytokine levels, in asthmatic subjects, are consistent with the possibility that pre-existing airway inflammation in these subjects primes the inflammatory response to O₃. Because O₃-induced airway inflammation may last several days and O₃-related asthma exacerbations often occur several days after exposure, it seems feasible that O₃-induced enhancement of pre-existing airway inflammation enhances susceptibility to asthma exacerbations. Devalia et al. 1998 investigated the effect of previous exposure to O₃ and NO₂ on subsequent allergen-induced changes in the nasal mucosa of patients with seasonal allergic rhinitis or perennial allergic asthma.
They found that exposure to these pollutants significantly increased the allergen-induced release of eosinophil cationic protein in nasal lavage. These results suggest that exposure to O₃ and NO₂ may "prime" the eosinophils to subsequent activation by inhaled allergen in atopic patients. Taken together, the results of the studies described above are consistent with a dose-dependent effect and they indicate that the concentration of O₃ and length of exposure is critical, with a possible threshold in the region of 0.1-0.2 ppm.

Particles derived from mobiles and stationary combustion sources are more likely to be of the PM₁₀ and decrements in pulmonary function test (PFT) values for in respirable range. The decrease of peak expiratory flow (PEF) in children with asthma has been noted in these places where the density traffic is heavy (Pekkanen et al., 1997). The technology for measuring PM₂.₅ levels in air was not widely available until the mid-1990s, so some studies report only on TSP, BS and other particulates. Pollution in the cities has associated serious to moderate health problems due to high levels of total suspended particulate matter (TSPM), sulphur dioxide (SO₂) and lead (The world Bank report, 1997). At elevated concentrations all the metals are harmful to living beings including human (Yasutake and Hirayama, 1997). Particles ≤0.1 μm diameters are referred to as ultratine and aerodynamically are too small to sediment out during normal breathing; however, they are deposited on alveolar walls by diffusion or are breathed back out again without deposition. They are rapidly removed by a combination of phagocytosis, lymphatic flow toward hilar nodes, and capillary blood flow, and they appear to not be retained in large numbers in the lung parenchyma (Churg and Brauer, 1997).

Srinivasan and Sukumar (1998) studied the impact of air pollutants on flora and human population in Neyveli Lignite Corporation area, and Sharma and Debarati (1998) determined the incidence acute lower respiratory infection in 642 infants in two urban slums in Delhi to examine the potential relationship.
of this condition with indoor air pollution. Leem et al., 1998 studied increased number of patients with respiratory or cardiovascular diseases at outpatients departments or emergency rooms. Vedal et al., 1998 study have shown that an increase in number of patients of asthma due to air pollution.

Brunekreef et al., 1999 reviewed that lung function was associated with truck traffic density but had a lesser association with automobile traffic density. The association was stronger in children living closest (< 300 m) to the motorways. Lung function was also associated with the concentration of black smoke, measured inside the schools, as a proxy for diesel exhaust particles. The associations were stronger in girls than in boys. The results indicate that exposure to traffic-related air pollution, in particular diesel exhaust particles, and may lead to reduced lung function in children living near major motorways.

Parallel to the increase in air pollution, there has also been a rapid increase in the global incidence of allergic diseases such as asthma and rhinitis in the last two decades, which cannot be attributed to genetic changes, and is assumed to be related to changes in environmental factors. Observations in Japan have suggested that children living close to roads with heavy traffic are more likely to develop allergies. Recent epidemiological data support the theory that atopic children may constitute a group of individuals that run a heightened risk of developing negative health effects following exposure to airborne particles (Boezen et al., 1999). At high ambient concentrations, well defined and marked systemic pulmonary inflammatory response is also observed (Salvi et al., 1999). Diesel exhaust contains various harmful substances (Halankar, 1999) and in association with air pollution cause asthma in children (Van der et al., 1999). PM_{10} is often associated with asthma and chronic cardiovascular and respiratory health problems. The PM_{10} particulate matter in particular has a potential to induce acute respiratory morbidity (Pope, 1999). The review has identified several effects of DEPs on immunological or inflammatory systems that may
potentially have particular relevance for a role of chronic diesel exhaust exposure in the pandemic of allergic disease.

Frischer et al. (1999) present an interesting study that concludes that exposure to increasing levels of ambient ozone \( (O_3) \) is associated with reduced growth in FVC, FEV\(_1\), and maximum expiratory flow at 50% of vital capacity (MEF\(_{50}\)) in children who were observed prospectively from age 8 to 11 yr (approximately). Roemer et al. (1999) reported the results of a multicenter panel study of the acute effect of particles (PM\(_{10}\)), black smoke, SO\(_2\), and NO\(_2\) on respiratory health of children with chronic respiratory diseases in Europe. They evaluated whether the potentially more sensitive subgroups were associated with the variations in air pollution. The predefined potentially sensitive groups were the presence of chronic respiratory symptoms, the use of respiratory medication, atopy, sex, and baseline lung function. They did not find a strong association between respiratory morbidity and air pollution among these groups of children. The general body of literature on lead toxicity indicates that, depending on the dose, lead exposure in children and adults can cause a wide spectrum of health problems, ranging from convulsions, coma, renal failure, and death at the high end to subtle effects on metabolism and intelligence at the low end of exposures.

An epidemiological study of children strongly suggests that increased respiratory morbidity and mortality are related to chronic exposure to photochemical pollutants and particulate matter (PM). Further 4% to 8% of premature deaths on a global scale are due to exposure to high levels of particulate matter in ambient air (WHO, 2000). Thus monitoring of PM\(_{10}\) from air reflects respiratory morbidity (American Thoracic Society (ATS), 2000) and hence considered an index of health. There are several reports that high level of Pb can induce severe neurological and hematological effects on the exposed population especially children, whereas Cd and Ni are known for inducing
carcinogenic effects in humans through inhalation, occupational level of Cd exposure is a risk factor for chronic lung diseases (Benoff et al., 2000). The health implications of cadmium exposure are exacerbated by the relative inability of human beings to excrete cadmium. Acute high-dose exposures can cause severe respiratory irritation. Occupational levels of cadmium exposure are a risk factor for chronic lung disease (through airborne exposure) and testicular degeneration (Benoff et al., 2000) and are still under investigation as a risk factor for prostate cancer. Lower levels of exposure are mainly of concern with respect to toxicity to the kidney. Cadmium damages a specific structure of the functional unit of the kidney (the proximal tubules of each nephron) in a way that is first manifested by leakage of low molecular weight proteins and essential ions, such as calcium, into urine, with progression over time to frank kidney failure.

Health problems posed by the pollutants at the work environment of an individual are closely linked to the nature and level of exposure to these hazardous chemicals. It has been known for quite sometime that air pollution from diesel exhaust is a major respiratory hazard for workers exposed to it in enclosed space (Kilburn, 2000). Particles >PM_{10} seldom reach the lung acinus; they are filtered out during inspiration by wall impact due to inertial forces at sites of turbulence in the nose or larynx or at branch points of conducting airways and ultimately are removed by ciliary action. Particles with size <2.5 \mu m and >0.1 \mu m are called fine particulates and are sedimented out in the gas exchange region of the lung (lung acini), where air movement is slow. These particles tend to be retained in respiratory bronchioles within the central part of the acinus (Pinkerton et al., 2000). Their removal from respiratory bronchioles is inefficient for lack of cilia and lack of an appropriate surface for efficient removal by macrophages. Ultrafine particles probably are capable of causing much greater tissue damage than larger particles when deposited and may be the
greatest source of elevated risk for death from ischemic heart disease or fatal arrhythmia. Air pollution is associated convincingly with many signs of asthma exacerbation, e.g. increased bronchial hyperresponsiveness, visits to emergency departments, hospital admissions, and increased medication use. Moreover, time-series data clearly show that traffic-related air pollution in urban areas has adverse effects on mortality from respiratory and cardiovascular disease. In a study of six USA cities, after adjusting for smoking, the mortality rate ratio increased in the most polluted areas compared with the least polluted city (Schwartz, 2000). This observation, which initially met with some scepticism, has been confirmed in a wide range of settings and in different countries. Diesel exhaust particulate (DEP) constitutes a large proportion of the PM in ambient air. In particular, diesel exhaust fumes cause bronchoconstriction, neutrophilic inflammation and dysfunction of alveolar macrophage phagocytosis, together with histamine release from mast cells in healthy individuals. Study of Sydbom et al. (2001) have shown the effect of DEP inhalation in asthmatic subjects. An increased bronchial hyperresponsiveness and an enhanced allergen response were demonstrated after local diesel exhaust particle instillation in the nose. The increase in the volume of road traffic and in air pollution shows a parallel increase in allergic disease, such as asthma and rhinitis. In fact, after inhaling DEP, the normal defence mechanism of the lung may be overwhelmed by the quantity or the toxicity of those particles. More specifically, the bronchial epithelium and cilia are damaged and the allergens remain on the epithelial surface for long time. Likewise, DEP may bind pollen or other allergens, thereby sensitising the airways to successive allergen exposure. Epidemiological studies have shown a significant association between a PM$_{10}$ exposure and exacerbation of asthma in children and adults.

The effect of ambient O$_3$ concentrations on human health has been heavily studied. There is now overwhelming evidence showing associations between the
levels of this pollutant and adverse respiratory effects, such as decrements in lung function, aggravation of pre-existing respiratory disease, increase in hospital admission and premature respiratory deaths (MacNee and Donaldson, 2000 and 2003).

Diesel exhaust, in addition generating pollutants like hydrocarbons, oxides of nitrogen and carbon is a major contributor to particulate matter in most places of the world. Symptoms like chronic cough, wheezing and breathlessness have been reported on exposure to these pollutants (Chhabra et al., 2001).

The relationship between daily levels of air pollutants and respiratory function in patients with chronic respiratory diseases has been analysed in various panel studies, with inconsistent results (Sunyer, 2001). The toxicology of air pollution is exceedingly complex as there are different types of pollutants affecting individual differently (Yassi et al., 2001). Chhabra et al. (2001) conducted a cross-sectional study among residents of Delhi to determine the role of ambient air pollution in chronic respiratory morbidity in Delhi. Air quality data for the past 10 year were obtained; data were based on the differences in total suspended particulates, and the study areas were categorized into lower and higher pollution zones. Lung function of asymptomatic nonsmokers was consistently and significantly better among both male and female residents of the lower pollution zone. Ambient air pollution was found to be important for chronic respiratory morbidity in Delhi (Chhabra et al., 2001). A large number of studies from the West do strongly implicate air pollution with chronic obstructive pulmonary disease (Sunyer, 2001).

The lungs seem to provide rapid transport of these potentially damaging particles from inspired air to circulating blood. This is consistent with the significant increase in risk of triggering a myocardial infarction within 1 to 3 hours of a sudden increase in fine particulate pollution, and the increased risk
may persist several days after the exposure (Peters et al., 2001). Ultrafine particles probably are capable of causing much greater tissue damage than larger particles when deposited and may be the greatest source of elevated risk for death from ischemic heart disease or fatal arrhythmia. The lungs seem to provide rapid transport of these potentially damaging particles from inspired air to circulating blood. Compared with other particle fractions, ultrafine particles have better deposition efficiency and higher number and area concentration (Frampton, 2001). Ultrafine particles also have ability to enter directly from lungs into systemic circulation (Nemmar et al., 2002). Brunckreef and Holgate (2002) epidemiological study consistly reported adverse health effects at unexpectedly low levels of ambient air pollution.

It has been assumed that damage from exposure to particulate air pollutants and gaseous pollutants is imposed primarily on the lungs and is associated with increased morbidity and mortality rates in patients with preexisting lung disease. This is supported by a considerable amount of previous data, such as the mortality data from the December 1952 London smog disaster, which may have caused as many as 12,000 deaths, almost all in patients with lung disease (Davis et al., 2002)

As children grow, their lungs become larger and the numerical values for tests of lung function also increase. Several studies involving three to eight years of follow-up have shown that deficits in the growth of lung function, as assessed by lung function tests, are related to exposure to ozone, fine particulates, NOx and acid vapor (Gauderman et al., 2002 and 2004). Follow-up study of Horak et al. (2002) assessed that decrease in lung function due long term exposure to ozone, particulate matter and nitrogen oxides.

Inhalation of dusts is an important cause of interstitial lung disease in the tropical countries such as India, while dusts of organic origin, such as the cotton
dust causing byssinosis, generally cause bronchial or bronchiolar involvement and hypersensitivity pneumonitis, inorganic metallic dusts cause progressive pulmonary fibrosis (Jindal et al., 2001 and Bayram et al., 2001) demonstrated that O\textsubscript{3} and NO\textsubscript{2} modulate the airway inflammation of diseases, such as bronchial asthma, by increasing the release of inflammatory mediators from bronchial epithelial cells, and that the cells of asthmatic subjects may be more susceptible to the adverse effects of these pollutants. It has also been observed that O\textsubscript{3} exposure has a priming effect on allergen-induced responses as well as an intrinsic inflammatory effect in the airways of allergic asthmatics. The demand of the risk management efficiency requires the quantification of the exposure and its health impact. Studies have been and are being performed at World Health Report, (2002) to estimate the global burden of disease and to assess the causes of this disease burden. This study aims at providing a better understanding of the more important environmental factors that affect the health of the general population and of potentially vulnerable groups. The main issue is the identification of environmental causes of ill-health using the tools of risk assessment and to assess the global burden of diseases from air pollution.

Dhara and Dhara (2002) have reviewed studies of human health effects that resulted from exposure to methyl isocyanate gas that leaked from the Union Carbide plant in Bhopal, India, in 1984. The studies were conducted during both the early and late recovery periods. Major organs exposed were the eyes, respiratory tract, and skin. Although mortality was initially high, it declined over time, but remained elevated among the most severely exposed population. The concentrations of the different types of particulate matter in air tend to vary up and down together. Black smoke concentration is easily determined by absorption of light by particulate matter, and the Black smoke level can be used as an indicator of diesel exhaust emissions (Gotschi et al., 2002). The concentration of black smoke (BS) in the air fell by 70%. Death rates from all
causes except trauma fell by 5.7%, respiratory deaths fell by 15.5 % and cardiovascular deaths fell by 10.3%. Approximately 75 deaths per year per 100,000 populations could be attributed to air pollution (Clancy et al., 2002).

The ability of particulate matter to induce oxidative stress in the airways has been proposed as an important biological mechanism (Kelly, 2003). In healthy and asthmatic volunteers, airborne particles increase bronchial responsiveness, airways resistance, and bronchial tissue mast cell, neutrophil, and lymphocyte counts (Holgate et al., 2003). Diesel exhaust fumes are made up largely of fine and ultrafine carbonaceous particulates generated by incomplete combustion. Experimental intratracheal instillation of these particles in hamsters caused platelet activation in blood perfusing the lung and enhanced peripheral thrombosis in an experimental arterial and venous thrombosis model (Nemmar et al., 2003). Pollutant particulates have been collected from the Los Angeles basin by Li et al., 2003, and separated into coarse, fine, and ultrafine particulates to compare their independent in vitro effects on macrophages. Ultrafine particles caused significantly greater oxidative stress and mitochondrial damage per microgram of particles—probably because of their smaller size, larger surface-to-volume ratio, and ability to penetrate into the cell interior and localize near mitochondria.

U.S. Environmental Protection Agency (EPA) in 2004 stimulated numerous studies of its chemical/physical properties of the sources that contribute the most hazardous components. Ozone is a highly reactive substance that reacts with biological compounds to form oxygen free radicals. These radicals are also highly reactive, promoting inflammation and damaging living tissues. Fine particulate matter contains heavy metals and endotoxin which can also initiate inflammation. Instillation of fine particles into the lungs of human volunteers evokes an inflammatory response characterized by the appearance of inflammatory cells and substances called cytokines in the lungs (Ghio and
Devlin, 2001). Sharma et al. (2004) assessed the relationship between daily changes in respiratory health and particulate levels with diameters of (a) less than 10 micron (PM$_{10}$) and (b) less than 2.5 micron (PM$_{2.5}$) in Kanpur, India. Sharma et al. (2004) used a statistical model to estimate that an increase of 100 microg/m$^3$ of the pollutant PM$_{10}$ could reduce the mean peak expiratory flow rate of an individual by approximately 3.2 l/min. Cardiovascular mortality related to air pollution is thought to be mediated by inflammation. Evidence from the past 10 years shows that sudden increases in ambient air pollution can also rapidly raise morbidity and mortality rates in patients with existing cardiovascular disease, as much or more than the rise associated with lung disease. In the present issue of Circulation, Pope et al. (2004) reported interesting new data on the effects on mortality rate of long-term differences, as opposed to sudden transient increases, in levels of air pollution. Diesel exhaust is considered to contain those substances, which are hazardous to human health (Gurjar et al., 2004). Heavy metals in air are responsible for the alveolar inflammation (Schaumann et al., 2004).

Ozone is a highly reactive substance that reacts with biological compounds to form oxygen free radicals. These radicals are also highly reactive, promoting inflammation and damaging living tissues. Fine particulate matter contains heavy metals and endotoxin which can also initiate inflammation. Instillation of fine particles into the lungs of human volunteers evokes an inflammatory response characterized by the appearance of inflammatory cells and substances called cytokines in the lungs (Schaumann et al., 2004). SO$_2$ effects have been related to decreases in pulmonary function in controlled human exposure studies but may also contribute to acid sulfate aerosol formation. Diesel exhaust particles have been found to increase airway inflammation and exacerbate asthma, and there have been a number of studies on the inflammatory impacts of O$_3$ on respiratory disease (Bernstein et al.,
2004). When emissions of air pollutants cease, air pollution levels drop rapidly. In a 2003 power outage that affected mid-Atlantic states there were 50-90% reductions in SO$_2$, ozone and light scattering particles within 24 hours (Marufu et al., 2004).

Becker et al., (2005) examined the in vitro effect of particulate matter and an epidemiological study examined seasonal differences across 100 US cities. Nanoparticles (particiles < 0.1 μm) in mass median aerodynamic diameter) have been postulated to affect cardiopulmonary system (Nel, 2005). Worldwide, PM$_{2.5}$ causes about 3% of mortality from cardiopulmonary disease, 5% of mortality from cancer of trachea, bronchus and lung, and about 1% of mortality from acute respiratory infections in children under age 5 years. It amounts to 0.8 million premature deaths (1.2 %) and 6.4 million years of life lost (Cohen et al., 2005). Even short-term exposure to particulates increases the mortality rates beyond the effect of hastening the death of the most vulnerable people.

Most of the reports that deal with health effects of particulate air pollution concern PM10 and PM2.5. These are the particulates that are most harmful to human health, especially those produced in motor vehicles (Laden et al., 2000; Lanki et al., 2006). Suspended particulate matter in ambient air is a complex, multi-phase system consisting of particle sizes ranging from <.01μm to 100μm (Wan-Kuen et al., 2005 and 2006). When breathed in, these particles can reach the deepest regions of the lungs and lead to many significant health problems ranging from aggravated asthma to premature death (Freiman et al., 2006; Benjamin et al., 2006). Human beings who inhaled fine particulate matter developed biochemical markers of inflammation in their blood and urine (Ruckeri et al., 2006 and Rabinovitch et al., 2006).

Agarwal et al. (2006) focused on assessing the status of respiratory morbidity in Delhi over a four years period from 2000-2003. The study showed
that winter months had greater exposure risk as pollutants often get trapped in the lower layers of atmosphere resulting in high concentrations. The partial correlation of SPM and relative humidity on COPD was higher than any other combination and therefore they can be regarded as important contributing variables on COPD. The urban population is mainly exposed to high levels of air pollution including metals because of motor vehicle emissions, which is also the main source of fine and ultrafine particles (Morawska \textit{et al.}, 2002 and Morawska and Shang 2002; Ristovski \textit{et al.}, 1998; Fang \textit{et al.}, 2005; Sharma \textit{et al.}, 2006), which influence the air quality. These particles can penetrate deep into the respiratory system, and studies indicate that the smaller the particle, more severe the health impacts (Schwartz \textit{et al.}, 1996). Ambient particulate matter may be carriers of acidic or toxic species (e.g., heavy metals, acids and carcinogenic organic compounds) and may have detrimental effects on human health and ecosystems. Beside particulate matter, there is a strong relationship between higher concentration of SO$_2$ and NO$_x$ and several health effects (Curtis \textit{et al.}, 2006), like cardiovascular diseases (Peters \textit{et al.}, 2004), respiratory health effects such as asthma and bronchitis (Barnett \textit{et al.}, 2005), reproductive and developmental effects such as increased risk of preterm birth (Liu \textit{et al.}, 2003) Exposure can occur through a variety of routes; inhalation of particles (<10$\mu$m) is one of the important routes. The inorganic components constitute a small portion by mass of the particulates; however, it contains some trace elements such as As, Cd, Co, Cr, Ni, Pb and Se which are human or animal carcinogens even in trace amounts (ATSDR, 2003; Wang \textit{et al.}, 2006). Cr (VI) is known to have toxic and carcinogenic effect on the bronchial tree (Manalis \textit{et al.}, 2005 and Hu, 2002). The increased level of Cu can lead to respiratory irritance (ATSDR, 2002). The overall burden of asthma in India is estimated at more than 15 million patients (Viswanathan \textit{et al.}, 1996).
Kumar et al. (2007) conducted this prospective study at the Shahdara industrial area of Delhi, India. They examined the effects of indoor and outdoor air pollutant levels on respiratory health in 394 children aged 7 to 15 years. The majority of children had a history of respiratory problems, including cough, sputum production, and shortness of breath, wheezing, common cold and throat congestion. The association of indoor and outdoor air pollutant levels showed that outdoor SO$_2$ and NO$_2$ was significantly higher than indoor SO$_2$ and NO$_2$ levels, whereas the mean indoor level of suspended particulate matter (SPM) was significantly higher than outdoor SPM level. Indoor SPM level also was significantly higher in homes of children with a history of respiratory illness than homes of children having no history of respiratory illness. Results suggest that both indoor and outdoor particulate exposure may be important risk factors in the development of respiratory illness in children.

Particulate matter less than PM$_{10}$ and aromatic chemicals formed during incomplete combustion of organic matter are major environmental pollutants because of their toxic potential. Chattopadhyay et al., (2007) studied reports on the respiratory morbidity pattern of people exposed to auto exhaust as a result of the traffic load consisting of three varieties of vehicles (heavy, medium and light) at three different points: North (B), South (E), and Central(c), regions of Kolkata, India. Particle size distribution was analyzed by an Anderson cascade impactor and volatile organic compounds (VOCs) were analyzed by sorbent tube and capillary gas chromatography with flame ionization detector. The traffic load in the vicinity supported the occurrence higher respiratory functional deterioration. PFT status showed restrictive, obstructive and combined restrictive and obstructive types impairment. Higher restrictive impairments in males might be due to their combined occupational and environmental exposures. The rate of increase of the numbers of vehicles on the roads of the city adds to the risk of greater problems due to exposure to hazardous
substances that are less than PM$_{10}$, in particular, polycyclic aromatic hydrocarbons and VOCs. Jayaraman and Nidhi (2008) study determine the association, if any, between the air pollutants, sulphur dioxide, nitrogen dioxide, carbon monoxide, ozone, suspended particulate matter and respiratory suspended particulate matter and daily variations in respiratory morbidity in Delhi during the years 2004-2005. Data analysis was based on the Generalized Additive Poisson regression model including a smoothing function for the entire patient population and subgroups defined by season. The best fitting lag period for each pollutant was found by testing its concentration at varying lags. The model demonstrated associations between daily visits and some of the pollutants (O$_3$, NO$_2$ and RSPM), but their strongest components were observed at varying lags.
Map of Rohtak city showing selected sites
- Hissar Road
- Bhiwani Stand
- Delhi Bye Pass
- Medical Mor
- University Campus
- New Bus Stand

Major Road

Municipal Boundary