ENVIRONMENTAL HEALTH MANAGEMENT SERIES:
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STUDY ON AMBIENT AIR QUALITY, RESPIRATORY SYMPTOMS AND LUNG FUNCTION OF CHILDREN IN DELHI

CENTRAL POLLUTION CONTROL BOARD
MINISTRY OF ENVIRONMENT & FORESTS
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FOREWORD

Rapid urbanization and economic growth has led to growth of vehicles in India, which has serious implication on the air quality. The substantial increase in number of vehicles has resulted in increased emission of air pollutants, specially the particulate matter which exceeds the prescribed standards in many cities. Particulate matter is associated with mortality and morbidity. Fine particles on their own or in combination with other air pollutants are linked to number of health problems. For rational planning of pollution control strategies, scientific information is needed on nature, magnitude and adverse health effects of air pollution.

To assess the impact of air pollution on human health (children), CPCB initiated an Epidemiological study in Delhi in 2003 with the help of Chittaranjan National Cancer Institute, Kolkata. The study was carried out over a period of three years during which several health camps were organized in different seasons, covering different parts of the city. The study included questionnaire survey as well as clinical examination.

The findings of the study were Peer reviewed by Indian Council for Medical Research (ICMR), New Delhi and All India Institute of Medical Sciences (AIIMS), New Delhi. The report was updated based on Peer review comments. Dr. Sanghita Roychoudhury, Research Associate, Sh. Tarun Darbari, Scientist B, Dr. Sanjeev Agrawal, Scientist D, have finalized the report under the supervision of Dr. D.D. Basu, Scientist E, and guidance Sh. J.S. Kamyotra, Member Secretary.

I hope the findings of the Report would be useful to all concerned.

(Mira Mehrishi)

3rd July, 2012

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EXECUTIVE SUMMARY

Central Pollution Control Board had sponsored the epidemiological studiy '**Study on Ambient Air Quality, Respiratory Symptoms and Lung Function of Children in Delhi**' carried out during March 2003–August 2005 and conducted by Chittranjan National Cancer Institute, Kolkata. The findings of these studies are as follows:

A. Objectives

- Assessment of the respiratory health status of school children chronically exposed to ambient air pollution of Delhi
- Assessment of degree of lung function impairment among children of Delhi

B. Study details

- 11,628 school-going children (7757 boys and 3871 girls) from 36 schools in different parts of Delhi in different seasons were included in the study
- Control: 4536 children, boys 2950 and girls 1586, from 15 schools of rural West Bengal and 2 schools from Khirsu and Kotdwar in Uttaranchal.
- Overall, the age of the children was between 4 to 17 years.
- Study was carried out between December 2002 – August 2005
- Pulmonary function tests (PFT) was conducted in 5718 participants of Delhi and 2270 control children by electronic, battery-operated spirometer

C. Study protocol

- Assessment of respiratory health by questionnaire survey and clinical examination
- Pulmonary function test (PFT) by spirometry
- Assessment of childhood obesity
- Examination of cellular lung reaction to inhaled pollutants by sputum cytology and cytochemistry
- Assessment of hematological and vascular changes associated with air pollution exposure following standard hematological procedure
- Assessment of behavioral characteristics

D. Findings

1. Respiratory and associated symptoms

- Compared to control, Delhi's children had 1.80 times more Upper respiratory symptoms (sinusitis, running or stuffy nose, sneezing, sore throat and common cold with fever) and two times more Lower respiratory symptoms (frequent dry cough, sputum-producing cough, wheezing breath, breathlessness on exertion, chest pain or tightness and disturbed sleep due to breathing problems) suggesting higher prevalence of underlying respiratory diseases.
- Respiratory and associated symptoms were most prevalent in children from low socio- economic status, and least in children from families with high socio-economic background.

- The symptoms were more prevalent in children during winter when PM_{10} level in air is highest in a year, and lowest during monsoon when particulate air pollution level is lowest, suggesting a positive association with particulate air pollution.

2. Lung function

- The results showed reduction of lung function in 43.5% schoolchildren of Delhi compared with 25.7% in control group. Delhi's children had increased prevalence of restrictive (20.3% vs 14.3% in control), obstructive (13.06% vs. 8% in control), as well as combined (both restrictive and obstructive) type of lung functions deficits (9.6% vs. 3.5% in control). After controlling potential confounders like season, socioeconomic conditions and ETS, PM_{10} level in ambient air was found to be positively associated with types of lung function

- Lung function reduction was more prevalent in girls than the boys both in rural and urban settings.

- Based on BMI data, 5.4% children of Delhi enrolled in this study were overweight against 2.4% children in control ($p<0.001$). Overweight and underweight children had poor lung function than children with normal weight.

3. Cellular lung reaction to air pollution

- The mean number of alveolar macrophages (AM) per high power field in Delhi's Children was 5.2 in contrast to 1.7 AM per hpf in control. Hence, school children of Delhi had 3.1 times more AM in their sputum. Marked increase in AM number signifies greater exposure to particulate pollution as AM represents the first line of cellular defence against inhaled pollutants.

- Sputum of Delhi's children contained 4-times more iron-laden macrophages (siderophages) than controls indicating convert pulmonary hemorrhage.

- Changes in the sputum cytology among the school children of Delhi positively correlated with ambient PM_{10} level.

4. Hematological and vascular changes

- The prevalence of hypertension in children was 6.2% in Delhi compared with 2.1% in control. Hypertension was more prevalent among girls than the boys and increased progressively with age, highest being in the age group of 15 – 17 years.

- 'Target' cells in 9.8% of Delhi's children against 4.3% of controls, implying a greater risk of liver problem.

- Higher prevalence of toxic granulation in neutrophils (21.0% vs. 8.7%) and circulating immature neutrophils (11.3% vs. 6.5%) was found among the children of Delhi, which suggests greater risk of infection and inflammation.

5. Behavior

- Delhi's schoolchildren had 2.5-times more Attention-Deficit Hyperactivity Disorder (ADHD) prevalence than age-and sex-matched controls (6.7% vs.2.7%, $p<0.05$). Boys had a remarkably higher prevalence than the girls. Besides air pollution, the stress of urban living could have played a role in eliciting greater prevalence of ADHD among the schoolchildren of Delhi

CHAPTER-1.0

BACKGROUND AND OBJECTIVE OF THE STUDY

1.1 BACKGROUND OF THE STUDY

Epidemiological studies have established a close relationship between exposure to ambient air pollution and morbidity and mortality from cardio-pulmonary diseases (Dockery et al., 1993, Samet et al., 2000, Schwela, 2000). Air pollution is a complex mixture of various gases, particulates, hydrocarbons, and transition metals. Of all these pollutants, the association between air pollution and adverse health conditions was the strongest and most consistent for respirable suspended particulate matters (RSPM) with an aerodynamic diameter of less than 10 micrometer (PM10). Health risk from particulate pollution is especially high for some susceptible groups such as the children and the elderly persons, and those with diseases of the heart and lungs (Ulrich et al., 2002).

(a) Children, the soft target

Children are more susceptible to environmental pollutants than the adults. The special vulnerability of children to air pollution exposure is related to several differences between children and the adults (Gilliland et al., 1999):

1. Children generally spend more time and are also more active outdoors than the adults. They are active outdoors during midday when air pollution levels tend to be higher. They have significantly higher oxygen demands so their respiration rates are higher and they inhale more air per unit of body weight than adults.

2. Because of their smaller stature their breathing zone is lower, so they inhale air loaded with more particles.

3. Diameters of their airways are smaller and therefore more likely to be affected by inflammation produced by air pollution

4. Their lungs are still developing and hence are more vulnerable to airborne insults. The efficiency of detoxification system of the body develops in time-dependent pattern. This in part accounts for increased susceptibility of children at critical points of time, and finally

5. Their immune defense is immature and hence less active against inhaled pathogens.

In essence, children represent the largest subgroup of the population susceptible to the adverse health effects of air pollution (Dockery et al., 2005). Damage to the respiratory system in children can be devastating and permanent and the adverse effects of air pollution may be obvious in adult life owing to the long latent periods for several chronic diseases.

Some children are more susceptible than others. Individuals with underlying chronic lung disease are at a greater risk than those not having such conditions. Polymorphic variations in genes involved in protecting against tissue injury or regulating tissue repair may explain some of the variations in individual susceptibility to adverse effects of air pollution on health. Furthermore, pattern and magnitude of exposure to indoor air pollution vary among children; those receiving higher exposures

indoor for example from parental smoking and cooking fuel emissions are at greater risk of being affected by outdoor pollutants.

(b) Health effects of air pollution in children

In the early part of this century, severe air pollution episodes of Meuse Valley, Belgium in 1930, Donora, Pennsylvania in 1948, New York in 1953 and 1962 and in London 1948, 1952 and 1956 were associated with excess deaths primarily from pneumonia and cardiovascular diseases (reviewed by Graham, 1990). Since 1960s studies on health effects of air pollution had shifted focus from mortality to morbidity as more and more emphasis were given to the effects of chronic, low level exposures of air pollution that the people in general experience in real life. Low-level air pollution in Japan was found to be associated with higher respiratory morbidity in all ages, and lung function were poorer in children residing in highly polluted cities compared with non-polluted rural areas (Toyama, 1964). A subsequent study in the United Kingdom revealed a relationship between exposure to high levels of particulates and sulphur dioxide in air and repeated episodes of acute upper and lower respiratory tract illness in children, after adjusting for socio-economic status (Lunn et al., 1967, 1970).

Acute exacerbations of chronic bronchitis were related to daily variations in smog and sulphur dioxide (Lawther et al., 1970). However, these adverse effects are only apparent above a moderately high threshold level (120 $\mu g/m^3$ of smoke in children). Successive studies have examined the health effects of air pollution at much lower levels than earlier works and they specifically addressed the question as to which component(s) of air pollution is the most important. It was found that a mean annual difference of 80 $\mu g/m^3$ of particulates between the cities doubled the risk of acute cough, bronchitis and other lower respiratory illness in children (Ware et al., 1986). In another study, 24-hour fine particulate levels as low as 15$\mu g/m^3$ were associated with significantly increase rates of hospitalization both in children and adults for respiratory diseases (Pope, 1989). The association between particulates and hospitalization was stronger for bronchitis and asthma than for pneumonia and pleurisy, and the association persisted after adjustment for meteorological variables. Other studies have confirmed the importance of the association between particulates and respiratory symptoms in children (Dockery et al., 1989, Dales et al., 1989). Ozone exposures below the US ambient air quality standards have been found to be associated with increased risk of cough and lower respiratory illness (Schwartz et al., 1989), and reduction in lung function (Kinney, 1989). Overall, the available evidence supports the hypothesis that particulate air pollution significantly increases the risk of morbidity from acute respiratory illness in children as well as in adults. However, it is not clear whether this morbidity is caused primarily by bronchial reactivity and respiratory tract irritation or by the effects of infection.

In both children and adults, upper respiratory illness is mainly caused by viral agents especially the rhinoviruses, coronaviruses, influenza and parainfluenza virus, adenovirus and respiratory syncytial virus. Viral infection often leads to bacterial infection such as pneumonia in the developing countries (Graham, 1990). On the other hand, respiratory syncytial virus, parainfluenza virus type I, II and III, influenza virus type A and B, adenoviruses and enteroviruses mainly cause lower respiratory illness in children, (Graham, 1990). In the developed world most causes of lower respiratory infection in

children are viral, but in developing countries mortality from bacterial pneumonia is the major problem (Glezen et al., 1973). Respiratory infection by agents other than bacteria and virus can be associated with respiratory infection in children. For example, *Mycoplasma pneumonae* and *Pneumocystis carinii* are important cause of pneumonia and acute bronchitis in both children and adults (Denny and Clyde, 1986). A large number of studies have shown that the incidence of viral respiratory illness peaks in infancy and early childhood and steadily reduces with age (Fox et al., 1972). This trend is generally attributed to changing pattern of exposure and the acquisition of specific immunity to the increasingly large array of virus types occurring with age. Respiratory illness have increased in recent years and the change has been attributed to changing virulence of respiratory pathogens and increased cumulative exposures to air pollution (Graham, 1990). Incident rates of acute respiratory infections also vary between sexes, higher rates have been found in males under the age of nine years, and the reverse pattern over that age (van Volkenburgh and Froast, 1933). In support of this, Gwaltney et al. (1966) reported more upper respiratory tract illness in females aged 16 to 24 years than males of similar age even after controlling confounders.

The accumulated evidence indicates that children's health is adversely affected by air pollution levels currently experienced in Europe and the United States. Evidence is sufficient to infer a causal relationship between exposures to ambient air pollution and adverse effects on lung function development in children. Chronic exposure to particulate matter (PM) in ambient air has been shown to cause lung function impairment (Hoek and Brunekreef, 1994, Schwartz and Neas, 2000), diminished birth weight and reduced growth of airways in children (Avol et al., 2001; Horak et al., 2002; Gauderman et al., 2002; Yang et al., 2003).

Studies have also revealed that diminished growth of airways in childhood increases the probability of subnormal lung function and risk of cardio-pulmonary diseases in adulthood (Gilliland et al., 1999; Schunemann et al., 2000).

1.2 AIR POLLUTION AND ADVERSE HEALTH EFFECTS: MODIFYING FACTORS

(a) Indoor air pollution

Environmental tobacco smoke (ETS) i.e. passive smoking, nitrogen dioxide from gas cooking / heating and smoke from biomass fuels are the three potential sources of indoor air pollution that may modify health effects of ambient air pollution. ETS increases the risk of respiratory symptoms and lung function reduction in children (Ware et al., 1984). Natural gas cooking and heating stoves increase exposure of family members to nitrogen dioxide (Melia et al., 1978). Children who are exposed to gas heating in their homes are more likely to be prone to respiratory illness than those with electric heating, but the level of significance was only marginal (Graham, 1990). In a study in Nepal, Pandey et al. (1989), found a relation between hours per day spent near a stove and acute lower respiratory illness in children.

(b) Housing and family size

Respiratory illnesses caused by respiratory infections are contagious diseases. Overcrowding favor their propagation. As early as in 1927, Woods reported a highly significant correlation between

overcrowded houses and pneumonia mortality in England and Wales. Payling-Wright, and Payling-Wright (1945) confirmed this finding by reporting a strong correlation between person per room and number of children per family and mortality from broncho pneumonia in children. Pneumonia epidemics have also been observed in crowded living conditions in South African mining camps, and during the construction of the Panama Canal (Finland, 1982).

(c) Nutrition

Malnutrition is generally regarded as a risk factor for respiratory infection. However, malnutrition is closely correlated with crowding, poverty, poor education and poor housing in developing countries. Its independent effect on risk of respiratory infection is rather difficult to assess. Malnourished children have been shown to experience 2.7 times more bronchitis and 19 times more pneumonia than normal-weight properly nourished children (James, 1972). A significant relation between malnutrition and pneumonia but not bronchitis has been reported (Berman et al., 1983). Vitamin A deficiency in children is associated with increased morbidity from respiratory infection and increased overall mortality. Breast-feeding reduce mortality in children in the developing countries (Victora et al., 1987). Whether the protective effect from breast milk is from its conferred anti-infective properties (Saarinen, 1982) or from nutritional factors *per se* is not clear. Conversely, obesity was reported to be associated with increased incidence of respiratory illness in infants (Tracey, 1971).

(d) Age

Some studies have observed a relationship between acute lower respiratory tract infection in the first two years of life and chronic respiratory disease in later life. For example, acute lower respiratory infection in childhood has been related to chronic cough in young adults (Colley et al., 1973), adult mortality from bronchitis (Barker and Osmond, 1986), reduced lung function and increased bronchial reactivity (Mok and Simpson, 1984).

(e) Psychosocial factor

Early cross sectional studies reported relations between anxiety and upper respiratory illness (Belfer et al., 1968), and between life changes, maladaptive coping, social isolation, unresolved role crises with respiratory infections (Jacobs et al., 1970). Other cross sectional studies have found relations between maternal stress and bronchitis in children (Hart et al., 1984); and poor family functioning with doctor visits for respiratory infection in children (Foulke et al., 1988). Stressful life events in families are four times more likely to precede an episode of streptococcal pharyngitis (Meyer and Haggerty, 1962). Stress and anxiety might predispose to respiratory infection by two mechanism: first, high stress levels may lead to disruption of normal hygiene measures that reduce transmission of respiratory viruses; second, since psychological stress and other psychological factors suppress body's defense against infection (Kieolt-Glaser and Glaser, 1986), this may lead to increased susceptibility to increased respiratory infection.

(f) Socio-economic status

Socio-economic status (SES) is usually measured in terms of level of income, education and occupational prestige. Low SES has been associated with increased mortality from bronchitis and

pneumonia in children (Collins et al., 1971). Social class is also related to respiratory morbidity from predominantly lower respiratory tract infections (Colley and Reid, 1970, Colley et al., 1973). Tupasi et al., (1988) confirmed that SES within developing countries strongly predicts risk of acute respiratory infection. Question has been raised about the key component of the low SES that increases the risk of respiratory infection. Poverty and lower social status are associated with large family size, crowded living conditions, poorer access to medical care, higher smoking rates, nutritional deficits and exposure to environmental pollutants including urban air pollution and stressful living environments. These factors may contribute individually or perhaps interact between themselves to increase the susceptibility to respiratory diseases.

(g) Meteorological factors

Low temperatures are usually associated with increase in mortality from pneumonia and bronchitis (Yang, 1924). However, the association could be explained by high PM level because peak levels of respirable particles occurred in mid winter presumably due to condensation, cloud cover and precipitation that prevent dispersal (Graham, 1990). Humidity might play a role in respiratory illness; for example, rhinoviruses survive better at higher humidity implying greater transmission during high humidity periods (Gwaltney, 1980). In temperate and warm climates, however, high humidity is often associated with the monsoon when people spent more time indoors. Therefore it remains a matter of conjecture whether the association was due to humidity or indoor air pollution.

(h) Low birth weight

It has been hypothesized that low birth weight could lead to more respiratory infections (Pio et al., 1985). Low birth weight (< 2 kg) is associated with chronic cough but not wheeze (Chan et al., 1989). A study in India by Datta et al., (1987) revealed that low birth weight infants (<2.5 kg) experienced the same respiratory illness prevalence as normal weight infants in the first year of life (4.65 vs. 4.56 episodes), but had a much higher death rate (24.6 vs. 3.2 per 100 episodes of moderate to severe respiratory illness). Increased mortality from respiratory infection in low birth weight children has also been reported by Victora et al., (1989) and this relationship persisted after adjustment for parental income and education. These studies suggest that low birth weight children do not experience higher rates of respiratory illness, but do experience more severe infections. Confounding factors for low birth weight such as overcrowding, poverty and poor nutrition make it difficult to ascertain whether the association is causal or not.

(i) Particle size, chemical composition and source

It is now well recognized that particulate matter (PM) with aerodynamic diameter of less than 10 mm (PM_{10}) and less than 2.5 mm ($PM_{2.5}$) are the primary mediators of toxicity in the lungs and the airways, while fine ($PM_{2.5}$) and ultrafine particles (UFP, aerodynamic diameter less than 0.1 mm) generally mediate toxicity on the heart and blood vessels (Pope 2004, Brook et al., 2004). It was also observed that exposures to fine particles from outdoor sources of combustion and from tobacco smoke invoke similar pathophysiological processes. Indeed, airway inflammation, an important factor in mediating air pollution effects on the lungs, is a common finding among smokers as well as in persons who have lived for long in a polluted environment (Gauderman et al., 2004).

There are increasing evidences that mass concentration of PM may not be the most relevant measure of PM standards to protect respiratory health. In general, relatively large and therefore heavier particles contribute more than smaller particles to ambient PM concentration. However, the smaller particles, such as PM2.5 and UFP are considered more harmful than larger particles (Peters et al., 1997; Schwartz and Neas, 2000; Diociaiuti et al., 2001; Huang et al., 2003; Li et al., 2003). Besides size, the chemical composition of the PM may determine its harmfulness. Several PM components like transition metals such as iron, copper, nickel and chromium (Campen et al., 2001; Aust et al., 2002; Gavett et al., 2003; Okesen et al., 2003), quinoid structures (Li et al., 2000), and aromatic hydrocarbons (Pacheco et al., 2001; Vassilev et al., 2001) are known to exert severe toxicity on exposed tissues. Moreover, PM from specific sources such as diesel exhaust (Hiura et al., 1999; Li et al., 2002) and residual oil fly ashes (ROFA) (Lambert, 2000) are highly toxic for airway cells probably because of their high organic aromatic and metal contents respectively (Hogervorst et al., 2006).

(j) Toxic effects of airborne pollutants

Based on findings of laboratory investigations, the consensus opinion is that exposures to particulate air pollution elicit adverse health effects through generation of reactive oxygen species (ROS) (Marano et al., 2002; Roberts et al., 2003). ROS can induce airway cell damage by inducing apoptosis (programmed cell death) and necrosis of airway cells and thereby increasing airway permeability which allows airborne allergens and pathogens to penetrate the airways more effectively, and by enhancing transcription of pro-inflammatory mediators including several cytokines and chemokines including tumor necrosis factor-alpha (TNF-±) and interleukin-8 (IL-8) (Samet et al., 1998; van Eeden and Hogg, 2002). These processes can ultimately lead to lung function reduction in children (Hogervorst et al., 2006). More importantly, it can lead to reduction in lung growth if the exposure to high level of air pollution is chronic (Hogervorst et al., 2006).

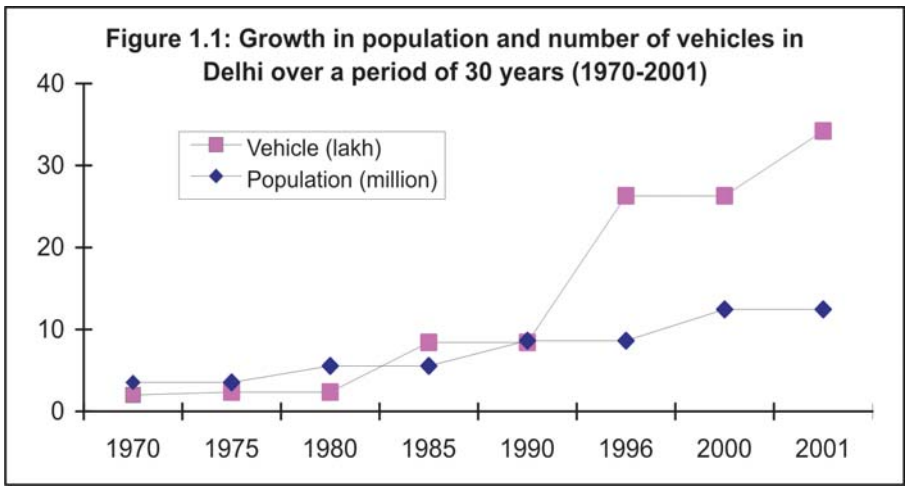
1.3 AIR POLLUTION AND ITS SOURCES IN DELHI

According to 2001 census, 13.8 million people lived in the Delhi within an area of 1483 km^2. Due to relatively high employment opportunities and better living conditions, Delhi has attracted millions of people from rural areas in neighbouring states. Currently Delhi and its surrounding suburbs is the third largest metropolitan area in the country after Mumbai and Kolkata. There are 827 women per 1000 men, and the literacy rate is 78.5%. Approximately 90% of the population is urban.

(a) Vehicular source of air pollution: motor vehicles in Delhi

Motor vehicles are responsible for a substantial part of Delhi's air pollution. The motor vehicle fleet of Delhi presently stands at 4.2 million, which is more than Mumbai, Kolkata and Chennai put together (Badami, 2005). Delhi alone with only a little over 1% of India's population accounts for 1/8th of national vehicle population (Badami, 2005). In 1975, the number of vehicles in Delhi and Mumbai was almost the same. Today Delhi has 3 times more vehicles than Mumbai, although Mumbai has 4 million more inhabitants than Delhi. While Delhi's population has grown about 5% per annum over the last three decades motor vehicles grew 20% per annum in the 1970s and 1980s and 10% per annum in the 1990s (Fig.1.1). They are still growing at a current rate of 7% per

annum (DDA 1996; Mohon et al., 1997). Vehicular particulate emissions are especially harmful for human health, because they are small and numerous, and occur near ground level where people live and work.

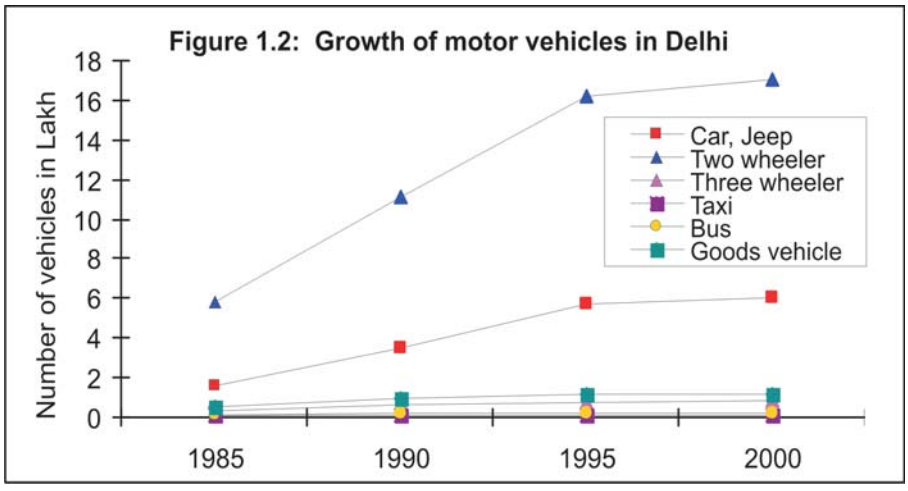


(i) Road transportation in Delhi

Delhi's road transport includes private vehicles such as 2-wheelers, cars, Jeeps etc.; public transport vehicles, such as bus, taxi, and autorickshaws; and goods transport vehicles such as trucks and tempos.

(ii) Bus

Delhi's buses constitute only small percentage of city's vehicular population, but they cater to maximum of the total traffic load. Although personal vehicles such as cars and two wheelers represent nearly 94% of the total number of vehicles of the city, they cater to only 30% of the travel demand (Deptt. of Transport, Govt. of Delhi). Growth of motor vehicles in Delhi is depicted in Fig. 1.2. Delhi Transport Corporation operates large fleet of compressed natural gas (CNG)-fueled buses. Besides, there are a large number of private-owned CNG-fueled buses plying in Delhi. Delhi's buses pollute much less than diesel-fueled buses of most other cities in India.



(iii) Taxi and Auto rickshaw

Compared to Mumbai and Kolkata, taxis are fewer in Delhi as people rely more on their own cars. However, Delhi's auto rickshaws provide a very important contribution in mass transport. Because they are CNG-powered, emissions are much less compared to pre-CNG era.

(iv) Goods transport vehicles

Lorries and trucks plying on Delhi roads including those coming with goods from adjoining states constitute small percentage of city's vehicular population. They are a significant source of air pollution, because the vehicles are run by diesel-powered engines.

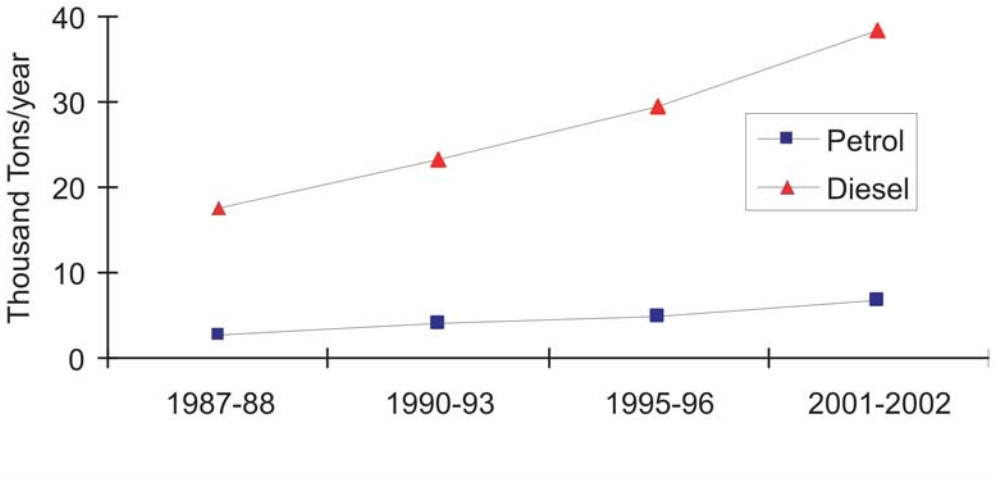
(v) Private vehicles

Private vehicles, such as cars, scooters and motor bikes, constitute 94% of Delhi's vehicular population. The numbers of 2-wheelers and cars have increased exponentially in recent times in Delhi, although the number of public transport vehicles has increased only marginally. About two-third of the total vehicular population of Delhi is two wheelers. Currently the city has 2.7 million two wheelers which was 1.4 million in 1993 - 1.5 times more than the combined number of two wheelers in three other metros of India – Mumbai 2.4 lakh, Kolkata 2.2 lakh and Chennai 4.6 lakh.

(vi) Automotive fuel

Diesel and petrol (gasoline) were the principal automotive fuels till the introduction of compressed natural gas (CNG) for public transport vehicles in 2001. There has been a steady increase in vehicle number and automotive fuel consumption, especially diesel, in Delhi in the last three decades. Estimated use of automotive use is depicted in Fig.1.3. Diesel is considered more polluting fuel than petrol, because it emits more particulates, especially of fine and ultrafine categories, than petrol during combustion. Petrol, on the other hand, releases more volatile organic compounds, particularly benzene, during combustion and also due to evaporation.

Figure 1.3: Estimated use of automotive fuel in India



Compared with diesel and petrol, CNG is a cleaner fuel as it emits substantially lower amount of particulates carbon monoxide and oxides of nitrogen as detailed in Table 1.1. Moreover, these vehicles have low vibrations and fewer odors than diesel engines.

Table 1.1: Emission comparison of diesel and CNG-powered buses

	Diesel	CNG	% reduction
CO	2.4 g/km	0.4 g/km	83
NOx	21 g/km	8.9 g/km	58
PM	380 mg/km	12 mg/km	97

Source: Frailey et al., 2000

(b) Industrial source of air pollution in Delhi

There are three thermal power plants at Indraprastha, Badarpur and Rajghat. The industries emit suspended particulate matter, hydrocarbons, sulphur dioxide, oxides of nitrogen and carbon monoxide. The emissions from power plants include sulphur dioxide, oxides of nitrogen and suspended particulate matter.

(c) Domestic source

Pollution from household sources is mainly due to the use of coal, kerosene and unprocessed solid biomass like firewood, cow dung and agricultural refuse like hay, husk, dried leaves etc.

(d) Climate and Natural Sources

Climate and natural sources also play an important role in contributing to the pollution levels of Delhi in addition to man-made sources. The region has a semi-arid climate. A sporadic pre-monsoon feature is dust storms when winds from the west deposit large concentrations of suspended particulate matter in the atmosphere of Delhi. Pre-monsoon calms contribute to increased pollution loads due to lack of mixing between different atmospheric levels. In winter, ground-based temperature inversions constrain dispersal of pollutants.

1.4 ECONOMIC ASPECT OF AIR POLLUTION IN INDIA

Improvement of air quality is associated with reduction in the number of premature deaths, episodes of acute illness such as asthma attacks and the number of chronic respiratory illness cases. The economists evaluate the value of avoiding an illness episode as

a. The value of work time lost due to the illness by the patient or the caregiver, or both
b. The medical cost of treatment
c. The amount paid to avoid the pain and suffering associated with the illness, and
d. The value of leisure time lost due to the illness by the patient or caregiver.

1.5 SCOPE OF THE WORK AND OBJECTIVES

Air pollution is considered as the most important contributing factor for respiratory illnesses. Considering these, it is important to assess the respiratory health of children in Delhi. Accordingly, the present study was undertaken in 2003 to study the respiratory health of children in Delhi.

Objective of the Study

The objective of this study was as follows:

- Assessment of the respiratory health status of school children chronically exposed to ambient air pollution of Delhi
- Establishment of a database relating to pollution related respiratory problems among children of the city.

This will serve as the reference data for all future monitoring studies of city's air quality with respect to health. Also, it will help to adopt and monitor intervention policies for betterment of the situation.

CHAPTER-2.0

MEASUREMENT OF AMBIENT AIR QUALITY OF DELHI

2.1 AMBIENT AIR QUALITY MONITORING IN DELHI

Data on the concentration of ambient air pollutants with respect to RSPM (respirable particulate matter with an aerodynamic diameter of less than 10 µm, i.e. PM_{10}), carcinogenic organic compounds like polycyclic aromatic hydrocarbons (PAHs) and volatile organic compounds (VOCs), oxides of nitrogen (NOx), sulfur dioxide (SO_2), suspended particulate matter (SPM) and ozone in study areas during and preceding months of this study were obtained from different air quality monitoring stations operated under National Air Quality Monitoring Programme (NAMP). Central Pollution Control Board and National Environmental Engineering Research Institute (NEERI) operate these stations under NAMP at the following locations as mentioned in Table 2.1.

Table 2.1: Air Quality Monitoring Stations in Delhi

	Name	Area	Operated by
1	Ashok Vihar	North	CPCB
2	ITO	Central	CPCB
3	Nizamuddin	South-east	CPCB
4	Shahadara	North-east	CPCB
5	Janak Puri	West	CPCB
6	Shahzada Bagh	North	CPCB
7	Siri Fort	South	CPCB
8	Sarojini Nagar	South	NEERI
9	Town Hall Library	North	NEERI
10	Mayapuri Industrial Area	West	NEERI

(a) Air quality measurements in control areas

Air quality data of rural areas of West Bengal were obtained from monitoring stations of the West Bengal State Pollution Control Board. Additionally, real-time particulate pollutant concentration in air by portable, battery-operated laser photometer (DustTrak™ Aerosol monitor, model 8520, TSI Inc., MN, USA) were measured, particularly for those areas where monitoring stations were absent. The instrument contains 10-mm nylon Dor-Oliver cyclone, operates at a flow rate of 1.7 liter/min and measures particle load in the concentration range of 1µg-100 µg/m^3. PM was measured with the aerodynamic diameter of less than 10 µm (PM_{10}), less than 2.5µm ($PM_{2.5}$) and les than 1.0 µm (PM_1). The monitor was calibrated to the standard ISO 12103-1 A1 test dust. The monitor was placed in open space at least 30 feet away from the roads, 3 feet above the ground level on a wooden stool. The monitoring was carried out 8 hours/day (07:30 –15:30 hours) for three consecutive days in a week and 3 alternate weeks in a season (summer, monsoon, and winter seasons). Monitor could not be used for longer periods due to the limitation of battery power.

(b) Efficiency of real-time aerosol monitor and reliability of data

Real-time Dust Trak monitors of TSI, USA was experimentally used in California, USA in late 90's simultaneously with traditional monitors using filter-based gravimetric method. Researchers of Harvard School of Public Health have reported satisfactory performance (Kim et al., 2004). They measured $PM_{2.5}$ simultaneously by DustTrak direct-reading aerosol monitor Model 8520 (which was used in this study) and filter-based gravimetric method. Spearman correlation proved the two methods as highly correlated. A subsequent study in Canada by Zhu et al., (2005) confirmed the reliability of data generated by real-time monitor. They monitored concentrations of fine particles in diesel exhausts with Dust Trak real-time monitors, and recorded the measured data every 5 seconds. Test variation of real-time monitoring in different test days was found similar to that measured by filter-based traditional gravimetric method, whereas the repeatability of the monitored data within the same day was better than that of gravimetric method (Zhu et al., 2005).

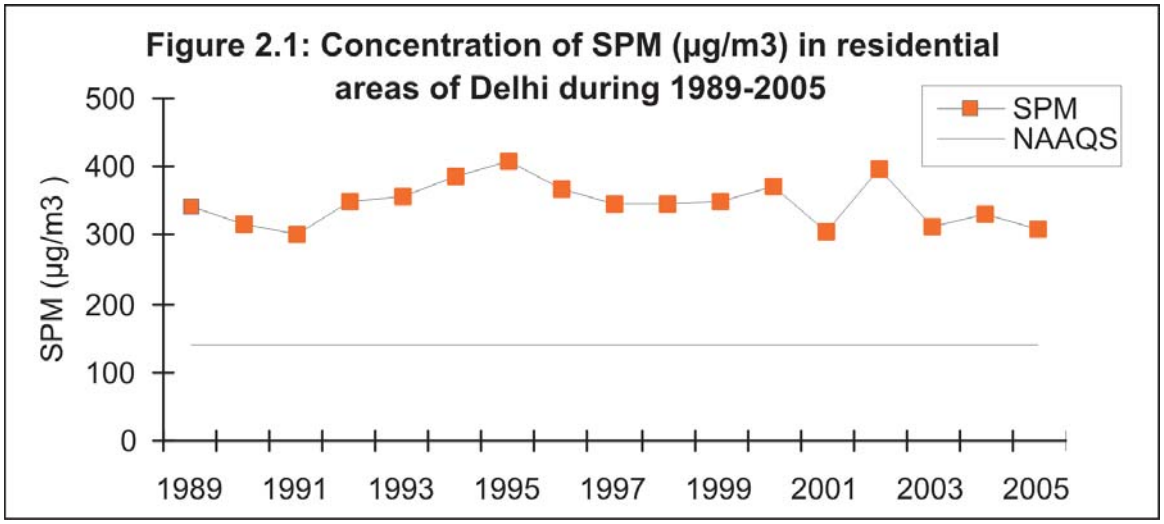
(c) Statistical analysis

The results are expressed as mean ± Standard Deviation (SD). The data were statistically analyzed by student 't' test and $p<0.05$ was considered significant.

2.2 RESULTS

(a) Suspended Particulate Matter (SPM)

The data shows that the concentration of SPM in Delhi's air had varied between 300 and 409 µg/m^3 from 1989 to 2005. All through these years the SPM level exceeded National Ambient Air Quality Standards (NAAQS) in residential areas as depicted Figure 2.1 and 2.2. SPM levels at various monitoringstations in Delhi is given in Table 2.2.



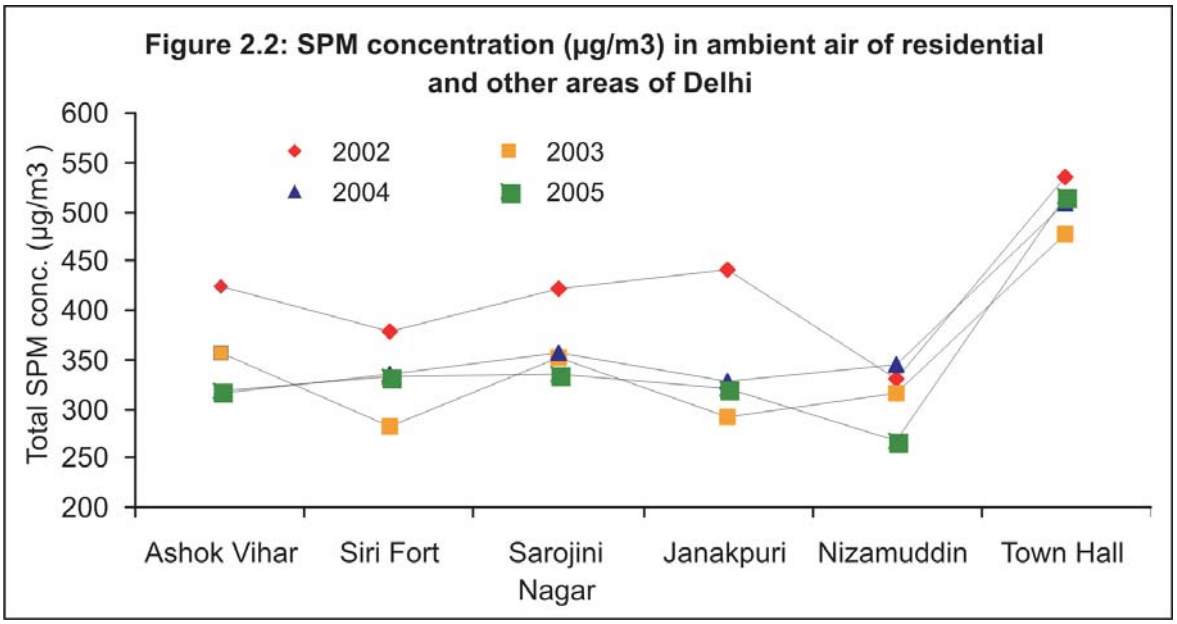
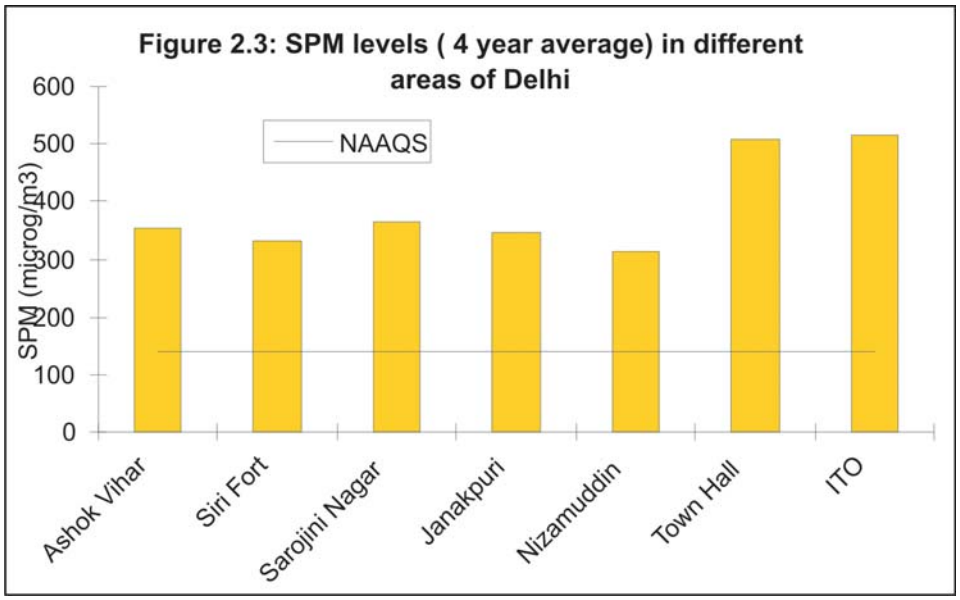


Table 2.2: Suspended Particulate Matter concentrations (in µg/m³) in Delhi's air during 2002-2005

Area	2002	2003	2004	2005	4-yr (Mean ± SD)
Residential areas					
Ashok Vihar	425	356	315	318	354 ± 51
Siri Fort	378	281	334	333	332 ± 40
Sarojini Nagar	421	352	356	335	366 ± 38
Janakpuri	442	291	328	320	345 ± 66
Nizamuddin	329	315	345	268	314 ± 33
Town Hall	534	478	508	516	509 ± 23
Mean ± SD	422 ± 69	346 ± 72	364 ± 72	348 ± 86	370 ± 35
Industrial area					
Mayapuri	NA	425	484	523	477 ± 49
Shahzada Bagh	468	354	338	308	367 ± 70
Shahdara	415	343	357	300	354 ± 47
Mean ± SD	442 ± 37	374 ± 45	393 ± 79	377 ± 127	396 ± 31
Traffic intersection					
ITO	533	509	500	512	514 ± 14
Overall	438 ± 67	370 ± 77	387 ± 78	373 ± 101	392 ± 32

*Source, CPCB, Delhi ; NA, data not available; *p<0.05 compared with residential area*

During 2002-2005, Delhi had an average concentration of 392 ± 32 µg/m³ (mean ± SD) of SPM in ambient air. Average SPM concentration in residential areas was 370 ± 35 µg/m³, which was higher than the National Standard for SPM in residential areas. Among the residential areas monitored during this period, highest SPM level was found in Town Hall (4-year average 509 µg/m³) in northeast Delhi, followed by Sarojini Nagar (366 µg/m³) and Ashok Vihar (354 µg/m³) in northern part of the city. During this period, the average concentration of SPM n residential areas declined from 422 to 348 µg/m³, showing a reduction of 18%. SPM levels (4 year average) at various monitoring statons in Delhi is deopicted in Figure 2.3.



In Industrial area, mean SPM concentrations (4 year average) was 396 µg/m³. However, highest SPM level of the city was found in traffic intersection point at ITO (514 ± 14 µg/m³, 4-year average).

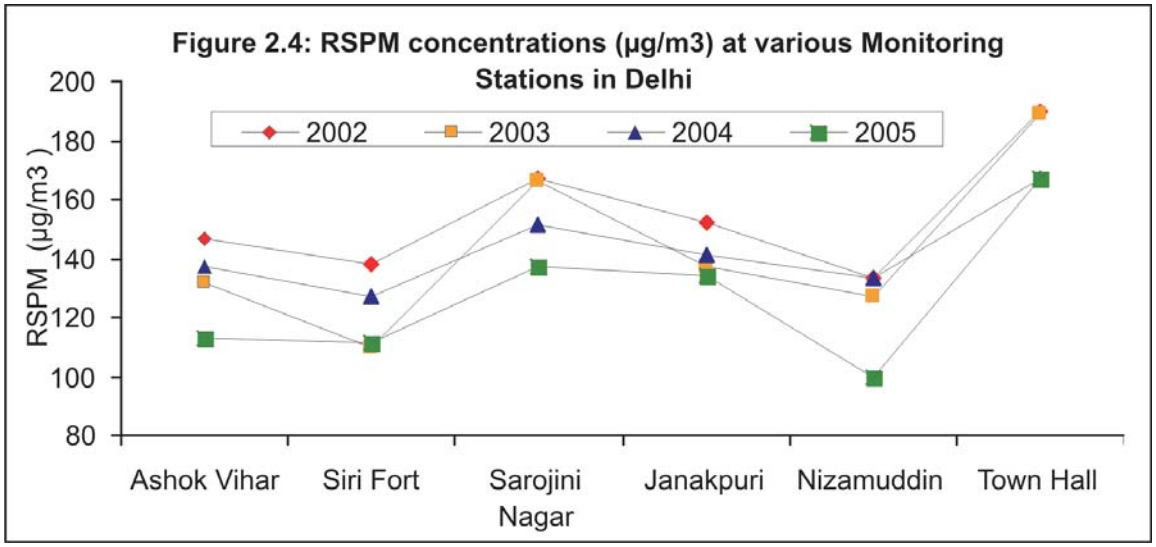
(b) Respirable Suspended Particulate Matter (RSPM/PM_{10})

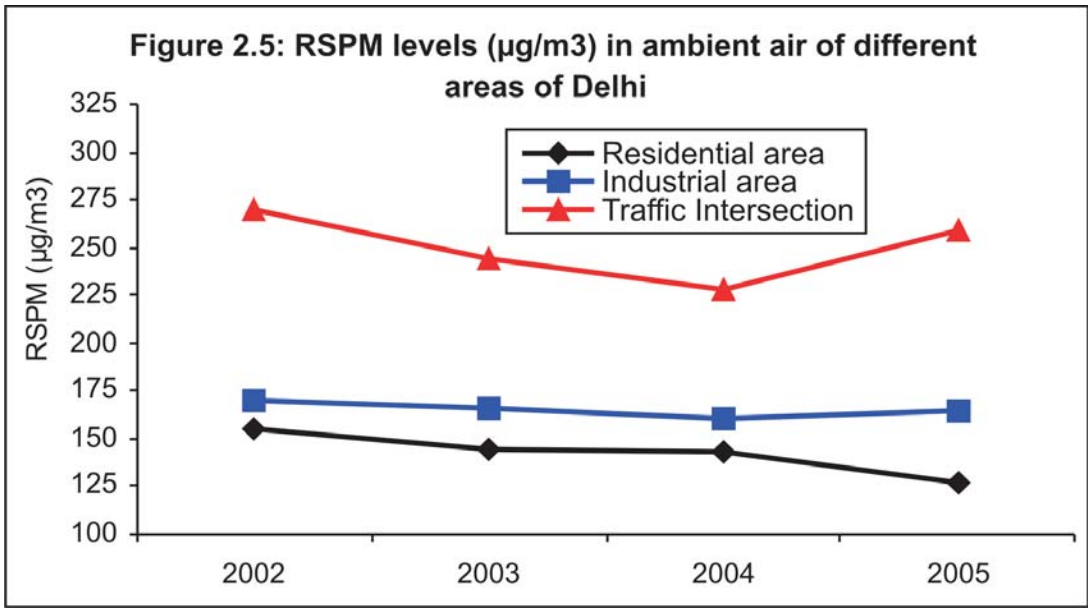
RSPM levels at various monitoring stations are given in Table 2.3. Out of the 10 air quality monitoring stations operative in Delhi, 6 were located in residential areas. The mean annual average respirable suspended particulate matter (RSPM) level during 2002-2005 in these areas of Delhi was 142 µg/m^3, which exceeded NAAQS. RSPM levels at various monitoring stations and areas are depicted in Figure 2.4 and 2.5 respectively. Among the residential areas, highest RSPM concentration was reported at Town Hall (4-year mean 178 µg/m^3), followed by Sarojini Nagar (155 µg/m^3) and Janakpuri (141 µg/m^3).

Table 2.3: Respirable suspended particulate matter (RSPM) concentrations (µg/m^3) in Delhi's air during 2002-2005

Area	2002	2003	2004	2005	4-yr (Mean ± SD)
Residential and other areas					
Ashok Vihar	147	132	137	113	132 ± 14
Siri Fort	138	110	127	111	122 ± 13
Sarojini Nagar	167	166	151	137	155 ± 14
Janakpuri	152	137	141	134	141 ± 8
Nizamuddin	133	127	133	100	123 ± 16
Town Hall	190	189	167	167	178 ± 13
Mean ± SD	155 ± 21	144 ± 29	143 ± 14	127 ± 24	142 ± 11
Industrial area					
Mayapuri	NA	212	213	233	219 ± 12
Shahzada Bagh	186	151	138	130	151 ± 25
Shahdara	153	136	131	131	138 ± 10
Mean ± SD	170 ± 23	166 ± 40	161 ± 45	165 ± 59	165 ± 4
Traffic intersection					
ITO	270	244	228	259	250 ± 18
Overall	171 ± 42	160 ± 42	157 ± 36	152 ± 53	160 ± 8

*Note:- NA- Data not available; *p<0.05 compared with residential area*





There was a declining trend in RSPM level in residential areas in the preceding four years. Compared with 2002 average (155 µg/m^3), the RSPM level has been decreased by 18% in 2005 (127µg/m^3).Industrial areas had a 4-year mean of 165 µg/m^3 of RSPM, which was higher than the NAAQS. At ITO crossing, the 4-year annual average RSPM concentration was 250 µg/m^3, the highest in Delhi. Taking 2000 data as the baseline, a sharp decline in RSPM level was recorded in 2001, the year in which CNG was introduced for public transport vehicles in Delhi. However, in the subsequent years the benefit was diluted perhaps due to the rise in the number of personal vehicles. The concentration was highest in traffic intersection point at ITO, followed by industrial areas and residential areas. RSPM level was lowest in residential areas. PM_{10} distribution in Delhi is depicted in Figure 2.6.

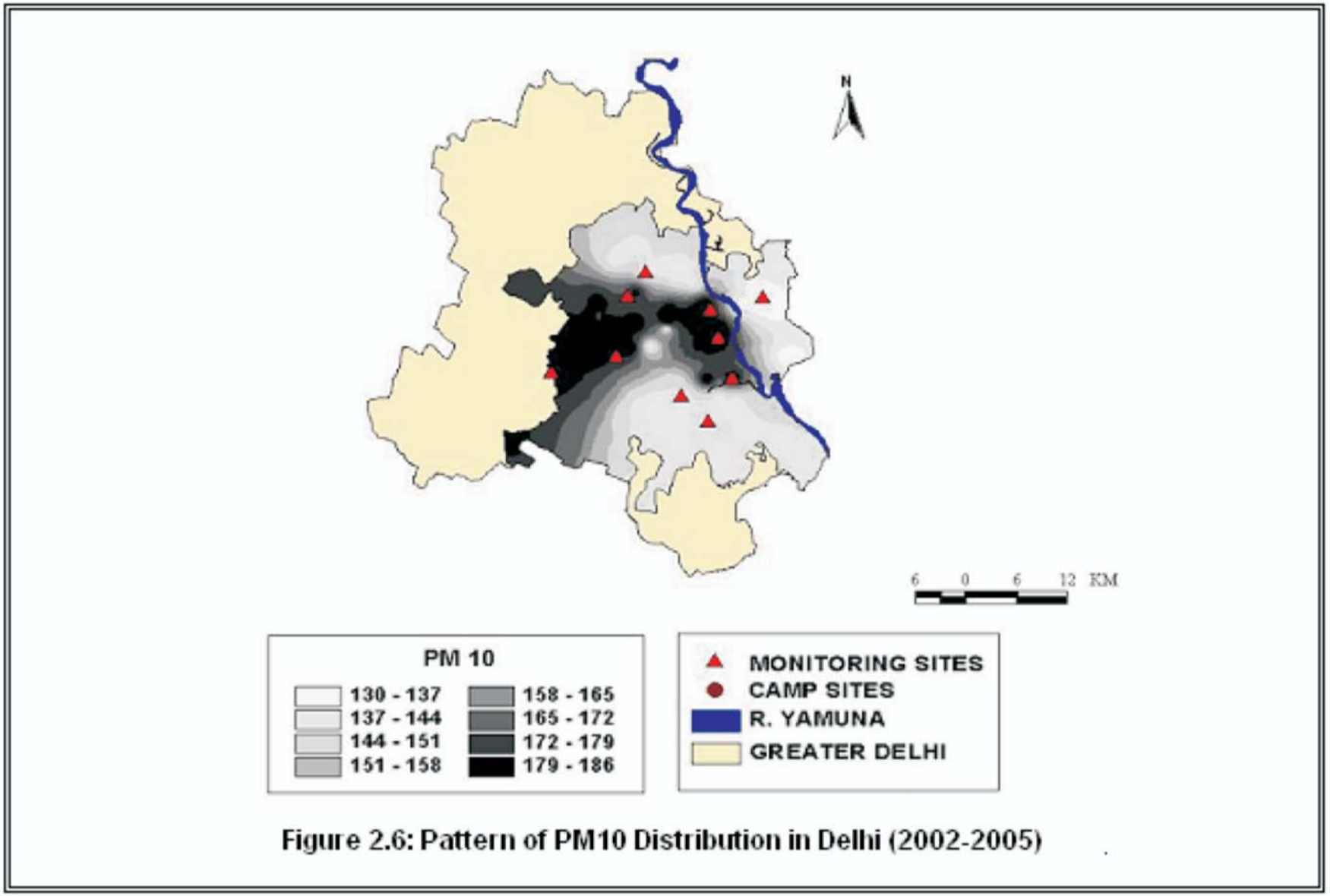


Figure 2.6: Pattern of PM10 Distribution in Delhi (2002-2005)

RSPM to SPM ratios are given in Table 2.4. RSPM:SPM ratio was appreciably higher at traffic intersection areas (0.49) than that of residential (0.39) and industrial areas (0.39), suggesting that the proportion of respirable particles in SPM was higher at traffic intersection.

Table 2.4: Four-year (2002-05) average SPM and RSPM concentrations at different areas of Delhi

Area	SPM (µg/m³)	RSPM (µg/m³)	RSPM/SPM ratio
Residential and other areas			
Ashok Vihar	354	132	0.37
Siri Fort	332	122	0.37
Sarojini Nagar	366	155	0.42
Janakpuri	345	141	0.41
Nizamuddin	314	123	0.39
Town Hall	509	178	0.35
Industrial area			
Mayapuri Industrial Area	477	219	0.46
Shahzada Bagh	367	151	0.41
Shahdara	354	138	0.39
Traffic intersection			
ITO	514	250	0.49

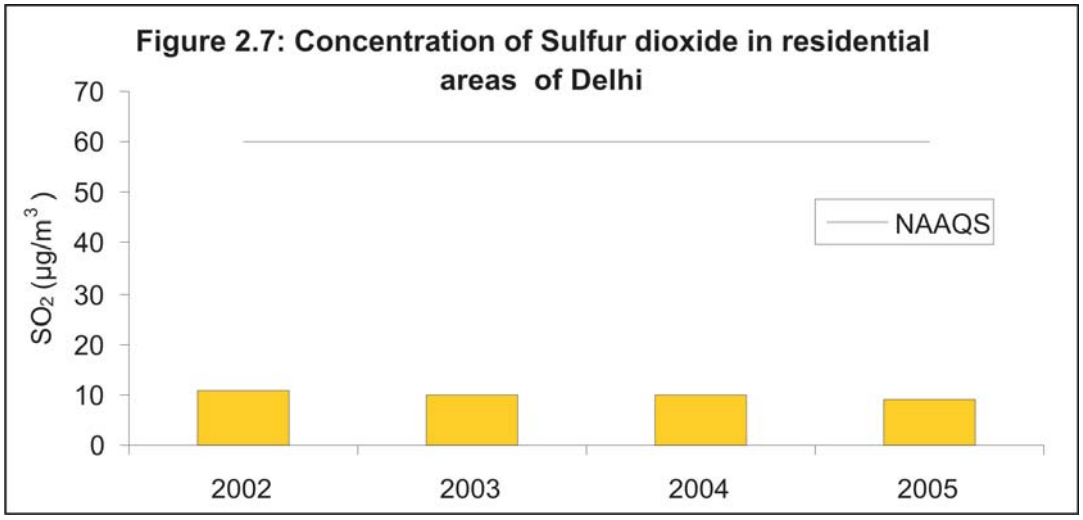
(c) Sulfur dioxide (SO_2)

The concentrations of SO_2 in Delhi's air were within the NAAQS during 2002-2005. The ambient SO_2 levels are given in Table 2.5. The 4-year (2002-05) average concentration of SO_2 in residential areas was 10µg/m³. SO_2 levels at ITO and industrial areas were 9 and 11 µg/m³ respectively. Trend in ambient SO_2 (4 year average) levels in residential areas is depicted in Figure 2.7. SO_2 levels were within the prescribed NAAQS during all the years.

Table 2.5: Concentration of Sulfur dioxide (µg/m³) in ambient air in different areas of Delhi during 2002- 2005

Area	2002	2003	2004	2005	4-yr mean±SD
Residential and other areas					
Ashok Vihar	6	6	10	8	8 ± 2
Siri Fort	12	9	8	9	10 ± 2
Sarojini Nagar	7	7	7	5	7 ± 1
Janakpuri	14	12	10	11	12 ± 2
Nizamuddin	13	12	11	10	12 ± 1
Town Hall	12	12	11	8	11 ± 2
Mean ± SD	11 ± 3	10 ± 3	10 ± 2	9 ± 2	10 ± 1
Industrial area					
Mayapuri	NA	13	12	14	13 ± 1
Shahzada Bagh	10	7	10	8	9 ± 2
Shahdara	17	11	10	9	12 ± 4
Mean±SD	14 ± 5	10 ± 3	11 ± 1	10 ± 3	11 ± 2
Traffic intersection					
ITO	10	9	8	9	9 ± 1
Overall	11 ± 3	10 ± 3	10 ± 2	9 ± 2	10 ± 1

Note:- NA- data not available



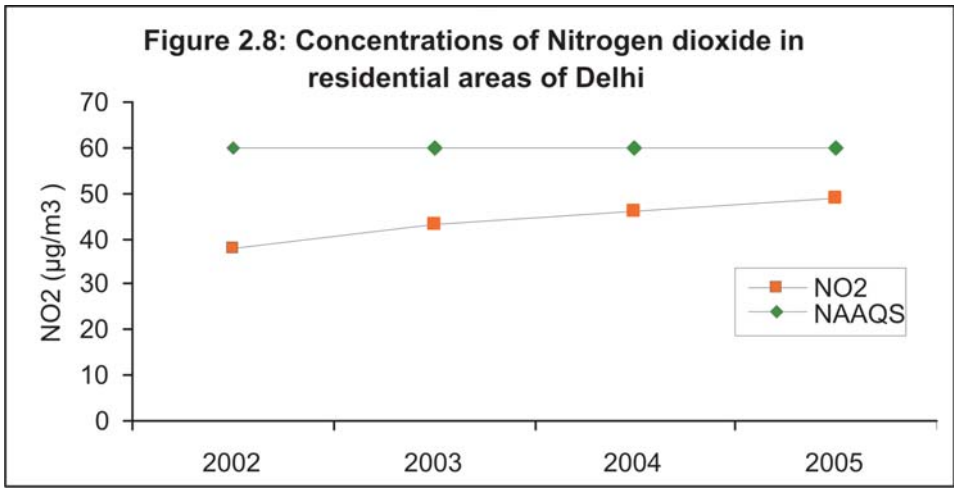
(d) Nitrogen dioxide (NO_2)

The ambient NO_2 levels are given in Table 2.6. NO_2 levels in Delhi's air in residential areas varied between 32 and 59 µg/m³ during 2002-2005. The 4-year average in residential areas was 44 µg/m³. NO2 levels at ITO was higher than other locations. NO_2 levels at ITO ranged from 75 µg/m³ in 2002 and 83 µg/m³ in 2005. Trend in ambient NO_2 (4 year average) levels inresidential areas is depicted in Figure 2.8.

Table 2.6: Concentrations of nitrogen dioxide (µg/m³) in ambient air of different areas of Delhi during 2002- 2005

Area	2002	2003	2004	2005	4-yr (Mean±SD)
Residential and other areas					
Ashok Vihar	26	32	39	49	37± 10
Siri Fort	27	32	35	35	32 ± 4
Sarojini Nagar	43	46	53	54	49 ± 5
Janakpuri	40	44	41	48	43 ± 4
Nizamuddin	39	43	45	45	43 ± 3
Town Hall	53	59	60	64	59 ± 5
Mean±SD	38 ± 10	43 ± 10	46 ± 9	49 ± 10	44 ± 5
Industrial area					
Mayapuri	NA	45	56	49	50 ± 6
Shahzada Bagh	34	39	47	46	42 ± 6
Shahdara	36	33	39	36	36 ± 2
Mean±SD	35 ± 1	39 ± 6	47 ± 9	44 ± 7	41 ± 5
Traffic intersection					
ITO	75	94	89	83	85 ± 8
Overall	41 ± 15	47 ± 19	50 ± 16	51 ± 14	47 ± 4

Source, CPCB and NEERI, Delhi; NA, data not available



The annual average concentration of NO_2 in ambient air of Delhi's was within the NAAQS at most of the locations.

(e) Polycyclic aromatic hydrocarbons (PAHs)

(i) Total PAHs

SPM-laden PAH concentrations in air of Delhi in winter (December 2004 and January 2005) are presented in Table 2.7. The mean value of PAHs in residential areas of the city during this period was 23.8ng/m^3. PAH concentration was highest in ITO traffic intersection area (54.4 ng/m^3), followed by Shahdara (44.4 ng/ m^3) and Nizamuddin (42.6 ng/ m^3). Lowest PAH level (10.8 ng/ m^3) was recorded in Siri Fort in South Delhi. Ashok vihar and Shahzada Bagh both in North Delhi had 18.0 and 17.0 ng / m^3 of PAHs (2-month average) respectively.

Table 2.7: Concentration of SPM-laden total polycyclic aromatic hydrocarbons (ng/m^3) in Delhi's air

Location	December 2004	January 2005	Mean
Residential Areas			
Siri Fort	6.6	14.9	10.8
Ashok Vihar	27.9	8.1	18.0
Nizamuddin	65.2	20.0	42.6
Residential areas, mean	33.2	14.3	23.8
Industrial Areas			
Shahzada Bagh	22.7	11.3	17.0
Shahdara	72.3	16.5	44.4
Industrial areas , mean	47.5	13.9	30.7
Traffic Intersection			
ITO	41.8	66.9	54.4
Overall	39.4	23.0	31.2

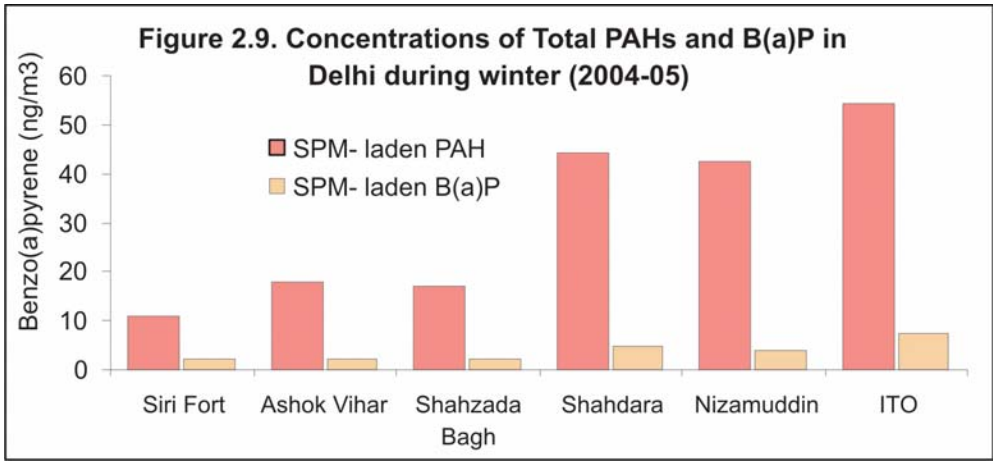
(ii) Benzo(a)pyrene (B(a)P)

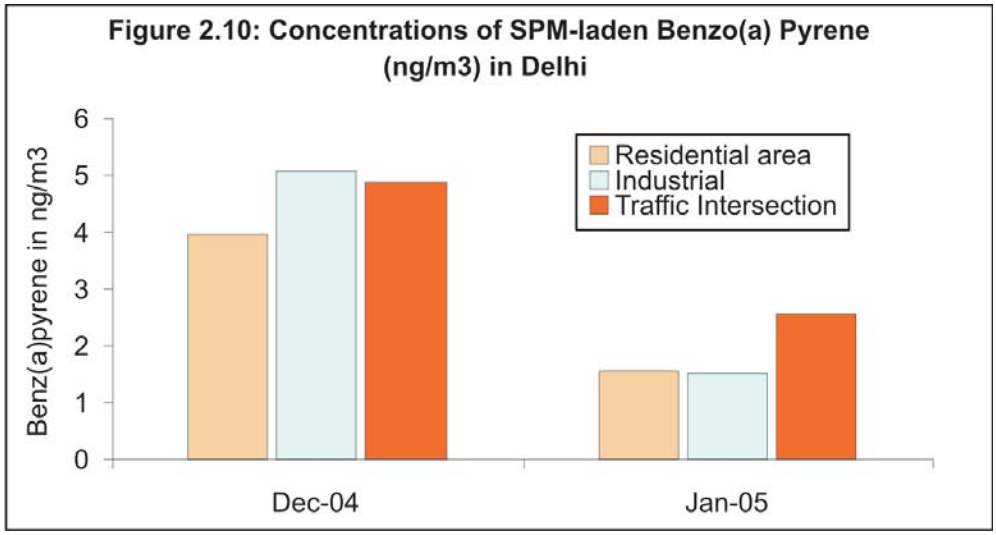
B(a)P is a potential carcinogenic PAH. B(a) P levels in Delhi are presented in Table 2.8. Its concentration was highest in traffic intersection area of ITO (7.3 ng/m^3), and mean value in residential area was 2.77 ng/ m^3 in three residential areas monitored. Among the residential areas, Nizamuddin had highest level of this carcinogen in its air (3.96 ng/m^3), followed by Siri Fort (2.30 ng/m^3) in southeast. The lowest B(a)P level (1.98 ng/m^3) was found in Shahzada Bagh in north Delhi.

Concentration of Total PAHs and B(a)P is depicted in Figure 2.9. B(a)P levels in residential and industrial areas are depicted in Figure 2.10. Pattern of B(a)P distribution in Delhi is depicted in Figure 2.11.

Table 2.8: Concentrations of SPM-laden Benzo(a)pyrene (ng/m³) in Delhi's air

Location	December 2004	January 2005	Mean
Residential areas			
Siri Fort	2.74	1.87	2.30
Ashok Vihar	3.34	0.78	2.06
Nizamuddin	5.86	2.07	3.96
Residential areas, mean	3.98	1.57	2.77
Industrial areas			
Shahzada Bagh	2.74	1.22	1.98
Shahdara	7.41	1.80	4.60
Industrial areas, mean	5.08	1.51	3.29
Traffic intersection			
ITO (SPM)	7.04	7.58	7.31
Overall	4.86	2.55	3.70





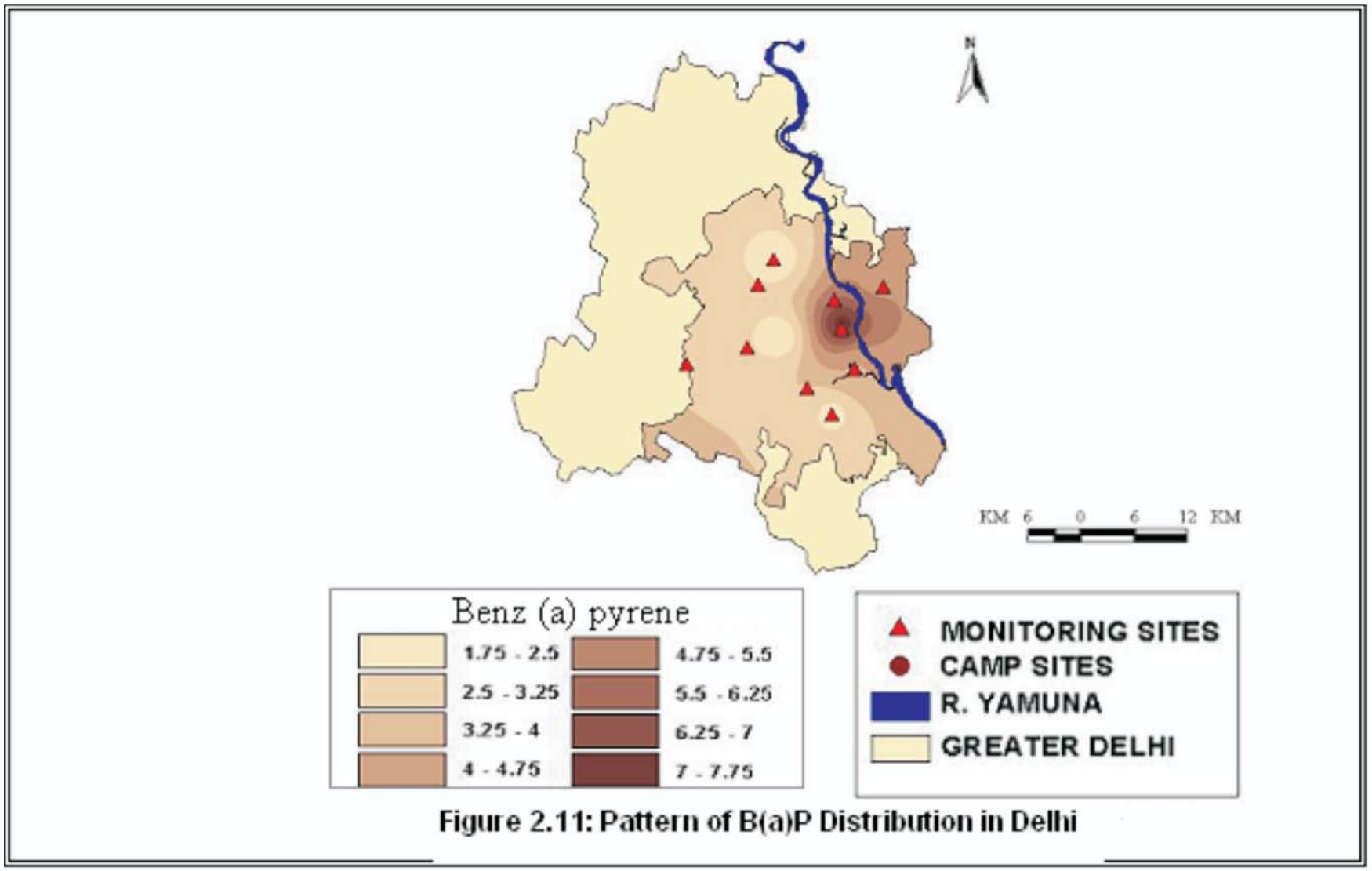


Figure 2.11: Pattern of B(a)P Distribution in Delhi

(f) Volatile organic compounds (VOCs): Benzene and toluene

VOCs contain several organic chemicals like benzene, toluene, xylene (BTX), formaldehyde etc. Out of these, benzene and toluene levels have been measured in some selected areas of Delhi by CPCB. Benzene is a highly toxic compound for human health. Sustained exposure to benzene may cause damage to the bone marrow that may lead to life-threatening diseases like aplastic anemia and leukemia.

(i) Benzene

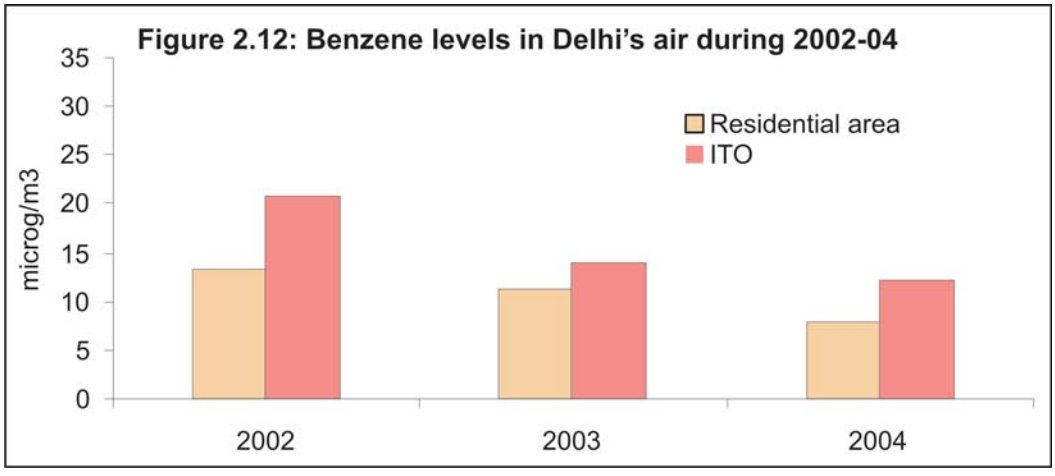
Benzene (mg/m^3) level in Delhi during 2002-2004 are presented in Table 2.9. Analysis of benzene data for the past four years (2002-05) shows a slow but steady decline in airborne benzene level till 2004 in residential localities and traffic intersection area of ITO. Benzene data of residential areas shows that north Delhi (Ashok Vihar and Moti Nagar) had higher benzene level in air than that of east (East Arjun Nagar) and JNU, south Delhi. JNU area had the lowest benzene level in the city. Benzene levels in residential areas and ITO are presented in Figure 2.12.

Table 2.9: Benzene (mg/m^3) level in Delhi during 2002-2004

Area	2002	2003	2004
Traffic intersection area			
ITO crossing	20.7	14.0	12.3
Residential and other areas			
Siri Fort	10.9	11.0	8.1
Ashok Vihar	NA	13.0	8.1
Moti Nagar	NA	13.0	9.5
East Arjun Nagar	NA	11.0	7.3
JNU	NA	6.0	4.7
Town Hall	15.8	13.0	8.8
Residential and other area, mean	13.4	11.2	7.8
Overall	**15.8**	**11.6**	**8.4**

Note: NA- Data not available

Compared with the residential areas, however, benzene concentration was higher in traffic intersection area of ITO. Pattern of Bezene distribution in Delhi is depicted in Figure 2.13.



(ii) Toluene

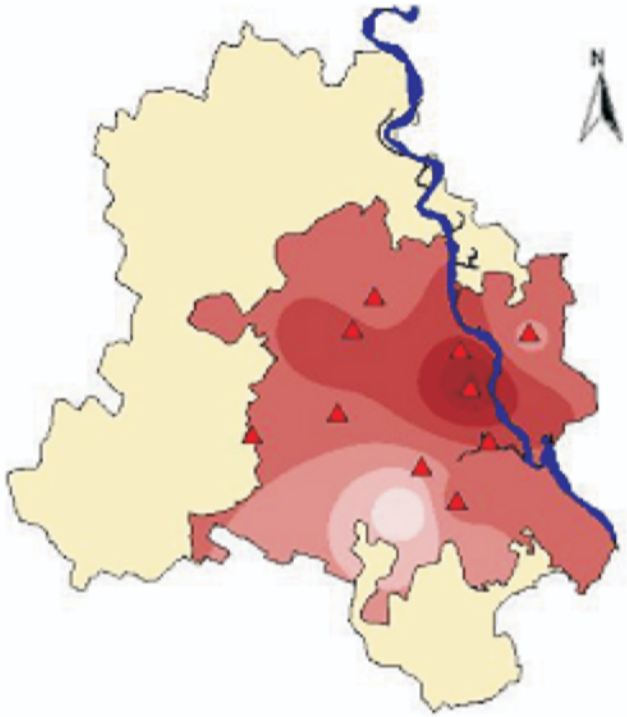
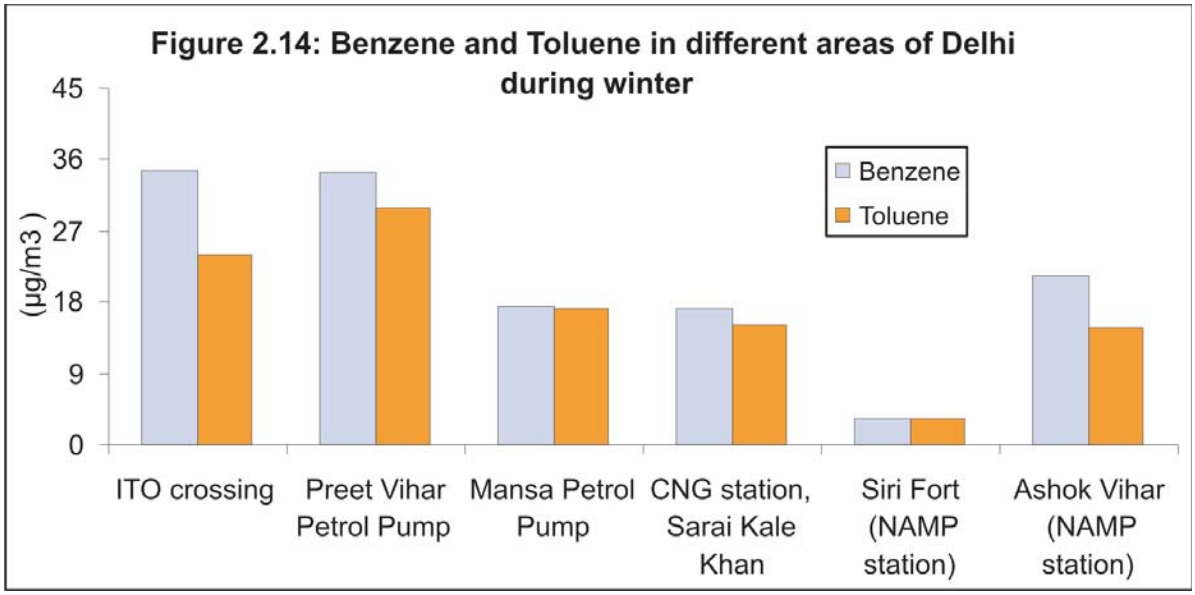


Figure 2.13: Pattern of Benzene Distribution in Delhi

Benzene and Toluene levels in Delhi are presented in Table 2.10. Toluene level was relatively low in residential areas, and higher in traffic intersection area of ITO, and petrol/CNG refueling stations of the city. The petrol pump and CNG refueling stations had benzene: toluene ratio of 1.01-1.15. In contrast, roadside measurements at ITO showed a higher ratio of 1.44. Benzene and Toluene levels in different areas of Delhi are presented in Figure 2.14.

Table 2.10: Volatile organic compound in ambient air in Delhi during winter (Feb 3-10, 2005)

Location	Benzene(mg/m^3)	Toluene(mg/m^3)	Benzene: Toluene
Residential areas			
Siri Fort (NAMP station)	3.39	3.13	1.08
Ashok Vihar (NAMP station)	21.44	14.82	1.45
Petrol Pump			
Preet Vihar Petrol Pump	34.38	29.81	1.15
Mansa Petrol Pump	17.40	17.21	1.01
CNG refueling station			
Sarai Kale Khan	17.15	15.15	1.13
Traffic intersection area			
ITO crossing	34.57	24.02	1.44



(g) Meteorological Data

Meteorological data of Delhi such as temperature, relative humidity, wind speed, wind direction and visibility during the study period was obtained from Indian Meteorological Department at Mausam Bhawan, Lodhi Road, New Delhi and the website wunderground.com and the meteorological data are presented in Table 2.11. For the rural areas of West Bengal data supplied by the regional Meteorological office at Alipore, Kolkata was used.

Table 2.11: Meteorological data of Delhi during 2004-2005.

	Summer(Mar-Jun)	Monsoon(Jul-Oct)	Winter(Nov-Feb)
Temperature (^{0}C)			
Minimum	17.5	22.2	9.7
Maximum	42.6	36.5	26.0
Average	31.4	28.2	18.3
Relative humidity (%)			
Minimum	14.2	47.6	29.0
Maximum	72.9	94.8	97.0
Average	38.7	63.6	64.2
Wind speed (km/h)			
Maximum	29.3	31.5	38.3
Average	5.9	4.6	3.4
Wind direction	WNW / W / WSW	WSW /ENE/ ESE	WSW / ENE
Visibility (km)	3.1	3.3	2.1

Source: wunderground.com

(h) Air Quality of Control Areas

The mean (SD) concentrations of SPM and PM_{10} in air of the control areas of West Bengal were 179.8±24.7 and 82.5±14.2 µg/m^3 respectively. Mean $PM_{2.5}$ concentration of these areas was 45.8±5.9 µg/m^3. Concentrations of SO_2 and NO_2 in these areas during the study period were 5.6± 2.2 and 30.3±5.2 µg/m^3 respectively.

Comparison of the air quality of residential areas of Delhi with that of control areas during 2002-2005 revealed significantly higher levels of all the pollutants in residential areas as compared to control areas. Concentrations of SO_2 and NO_2 were within standards in Delhi as well as in control areas, but Delhi had a substantially higher level as compared with control.

2.3 FINDINGS

1. Mean concentration of total suspended particular matter (SPM) in Delhi's air during 2002-2005 was 370 µg/m^3 in residential areas, 396 µg/m^3 in industrial areas, and 514 µg/m^3 in traffic intersection point at ITO.
2. Mean concentrations of the respirable suspended particulate matter (RSPM, particulate matter with less than 10 µm diameter, PM_{10}) during this period were 142, 165, and 250 µg/m^3 in residential, industrial, and traffic intersection point respectively.
3. Mean concentrations of sulfur dioxide (SO_2) and nitrogen dioxide (NO_2) in Delhi's air during 2002-2005 were 10 and 47 µg/m^3 respectively. In the control areas the concentrations of SO_2 and NO_2 were 5.6 and 30.3 µg/m^3 respectively. The levels of these two pollutants were within the Standard in Delhi as well as in control areas.
4. A small decline in the concentrations of SPM and RSPM in ambient air has been recorded in residential areas of Delhi during 2002-05.
5. The average concentration of benzo(a)pyrene in ambient air of Delhi was 3.70 ng/m^3 during December 2004 and January 2005. The concentration was highest at ITO (7.31 ng/m^3).
6. Benzene levels were 7.8 µg/m^3 in residential areas of Delhi in 2004. Highest concentration was found in traffic intersection point at ITO.

CHAPTER-3.0

PREVALENCE OF RESPIRATORY AND ASSOCIATED SYMPTOMS

3.1 INTRODUCTION

Air pollution is recognized as a major contributor to several respiratory problems. Diseases of the lungs and the airways are often manifested by one or more symptoms that can be easily recognized. Thus, the presence of a particular symptom or a group of symptoms can confirm the presence of an underlying disease in the upper or lower airways. This has been utilized by several epidemiological studies in which the prevalence of respiratory symptoms has been assessed in order to get an insight into the occurrence of a disease in the airways and the lungs. In view of this, prevalence of upper and lower respiratory symptoms in children was assessed through questionnaire survey. Questionnaires are the most commonly used subjective instrument of measurement in respiratory epidemiology. They represent a convenient tool of investigating large sample population owing to low cost, easy to use by the investigator, and good compliance of the subjects (Liard and Neukirch, 2000).

3.2 MATERIALS AND METHODS

(a) Subjects

The study was conducted between December 2002 and August 2005. The sample areas were divided into different homogeneous strata based on air pollution level. Then sampling was done from each stratum randomly. Thus stratified random sampling procedure was followed under the general plan of Simple Random Sampling Without Replacement method (Rao, 1989). A total number of 16,164 children aged between 4-17 years participated in this study.

(i) Urban children

Among the participants, 11,628 school-going children were enrolled from 36 schools situated in different parts of Delhi. Out of the 11,628 participants from Delhi, 7757 (66.7%) were boys and 3,871 (33.3%) were girls. Thus, boys: girls ratio was 2.00.

The schools were selected on the basis of following considerations:

- Located within 3 km radius of the air quality monitoring stations of Central Pollution Control Board (CPCB) and National Environmental Engineering Research Institute (NEERI), so that health data of the children could be analyzed vis-à-vis air quality.

- Represent every section of the people with comparable representations of children from low, medium and high socio-economic status.

The list of the schools and number of children examined are presented in Table 3.1.

Table. 3.1: Number of children and Names of the Schools of Delhi where the study has been conducted

S.No.	Name of the School	Area	No.
1	Sarvodaya Vidayalaya, Shakti Nagar	North	64
2	Andhra Education Society, New Ashok Nagar *	North	127
3	Mahaveer Sr. Model School, Sangam Park Ext. *	North	240
4	Montfort S.S. School, Ashok Vihar *	North	602
5	Bengali S.S. School, Civil Lines *	North	670
6	Mata Saheb Kaur Public Shool, Kalyan Vihar *	North	65
7	Siwan Public School, Jawahar Nagar *	North	21
8	Nagar Nigam School, Sec. 7, Rohini *	North	25
9	Maharaja Agrasain Public School, Ashok Vihar *	North	385
10	N.P.Bengali Girls' S.S. School, Gol Market	Central	345
11	D. A. V. Boys S.S. School, Chitragupta Road	Central	303
12	S.G.T.B. Khalsa Boys School, Dev Nagar	Central	446
13	Govt. Boys S.S. School, Rani Jhansi Road	Central	246
14	DAV Sr. Secondary School, Pusa Road	Central	364
15	Ramjas School, Pusa Road *	Central	284
16	Guru Harkishan S.S. School, Karol Bagh *	Central	335
17	Ramjas Girls' School, Daryaganj	Central	133
18	Ramjas Boys', School, Daryaganj	Central	408
19	Bal Bharati Public School, Sir Ganga Ram Road *	Central	449
20	M.B.D.AV. S.S. School, Yusuf Sarai *	South	666
21	Navyug School, Sarojini Nagar *	South	362
22	St. Pauls School, Hauz Khas *	South	103
23	Cambridge School, Srinivas Puri *	South	117
24	St. Mary's School, Safdarjung Enclave *	South	213
25	Delhi Police Public School, Safdarjung Enclave *	South	256
26	Bidhan Chandra Vidyalaya, Moti Bagh-I *	South	349
27	Ramjas School, R.K. Puram *	South	312
28	Gyan Bharati School, Saket *	South	435
29	Kendriya Vidyalaya, East Arjun nagar *	East	730
30	Sarvodaya Vidyalaya, Gandhi Nagar *	East	37
31	Lakshmi Public School, East Arjun Nagar *	East	971
32	Tagore Public School. Naraina Vihar *	West	202
33	J.L.D.A.V. Public School, Paschim Vihar *	West	410
34	Blooming Buds Public School, New Moti Nagar *	West	245
35	Shivmandir S.B Vidyalaya, Jaidev Park *	West	106
36	Sukho Khalsa S.S. School, Janak Puri *	West	602
	Total	**Delhi**	**11, 628**

** Co-educational school*

(ii) Rural children (control)

A total number of 4536 children from 17 schools of rural areas of Uttaranchal and West Bengal where the level of ambient air pollution was much less for the absence of air polluting industries and lesser number of motor vehicles were enrolled in this study as control. There were 2 schools in Khirsu and Kotdwar in Pauri-Garhwal district of Uttaranchal, and 15 schools in Cooch Behar, South Dinajpur, Maldah, North and South 24-Parganas, and West Medinipur districts of west Bengal. To make the urban (Delhi) and control children comparable with respect to fuel use at home,

children from LPG-using homes were preferred. Boys constituted 65.0% (2950 of 4536) and girls 35.0% (1586 of 4536) of the control group. The boys: girls' ratio was 1.86. Name of the schools are presented in Table 3.2.

Table 3.2: Names of the schools of Uttaranchal (UT) and West Bengal (WB) from where the children of control group were examined.

S.No.	Name of the School	District	State	No.
1	Govt. Inter College, Khirsu *	Pauri-Garhwal	UT	171
2	Motherland Academy, Kotdwar *	Pauri-Garhwal	UT	127
3	Manikchak Shiksha Niketan, Manikchak *	Maldah	WB	450
4	Gourio High School, Gour *	Maldah	WB	343
5	Kaliaganj Parbati Sundari High School *	North Dinajpur	WB	438
6	Prachya Bharati Vidyapith, Balurghat *	South Dinajpur	WB	145
7	Khadimpur Girls High School, Balurghat	South Dinajpur	WB	173
8	Sunity Academy, Cooch Behar	Cooch Behar	WB	256
9	Maharaja N.N.High School, Cooch Behar	Cooch Behar	WB	179
10	Toofanganj Ila Devi Girls' High Schol	Cooch Behar	WB	200
11	Dinhata Girls' High School, Dinhata	Cooch Behar	WB	257
12	Dinhata High School, Dinhata	Cooch Behar	WB	167
13	Taki Govt High School, Taki *	N. 24-Parganas	WB	190
14	Taki S.L. Girls' High School, Taki	N. 24-Parganas	WB	200
15	Sundarban JKS School, Rudranagar, Sagar Island*	S. 24-Parganas	WB	315
16	N.N. High School, Natendrapur, Sagar Is*	S. 24-Parganas	WB	488
17	Mohar Brahmamoyee H.S., Sabang *	West Medinipur	WB	437
	Total	**Control**		**4536**

** Co-educational school*

Photographs of schools where health camps were conducted are presented in Figure 3.1 and 3.2.

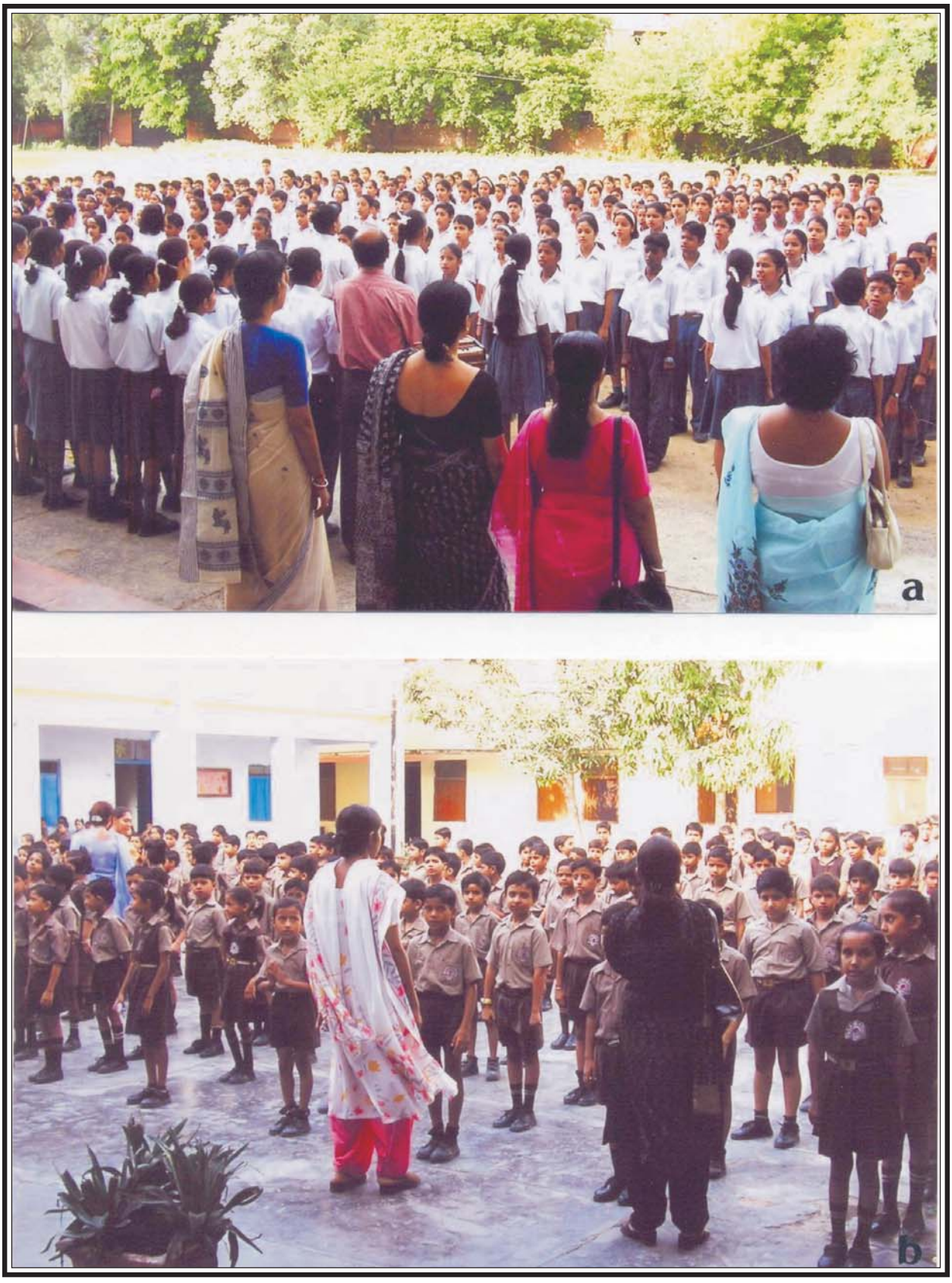


Figure 3.1: Chilren Health Camp in (a) Bidhan Chandra Vidyalaya, Moti Bagh, New Delhi and (b) D.A.V. Senior Secondary School, Pusa Road, New Delhi

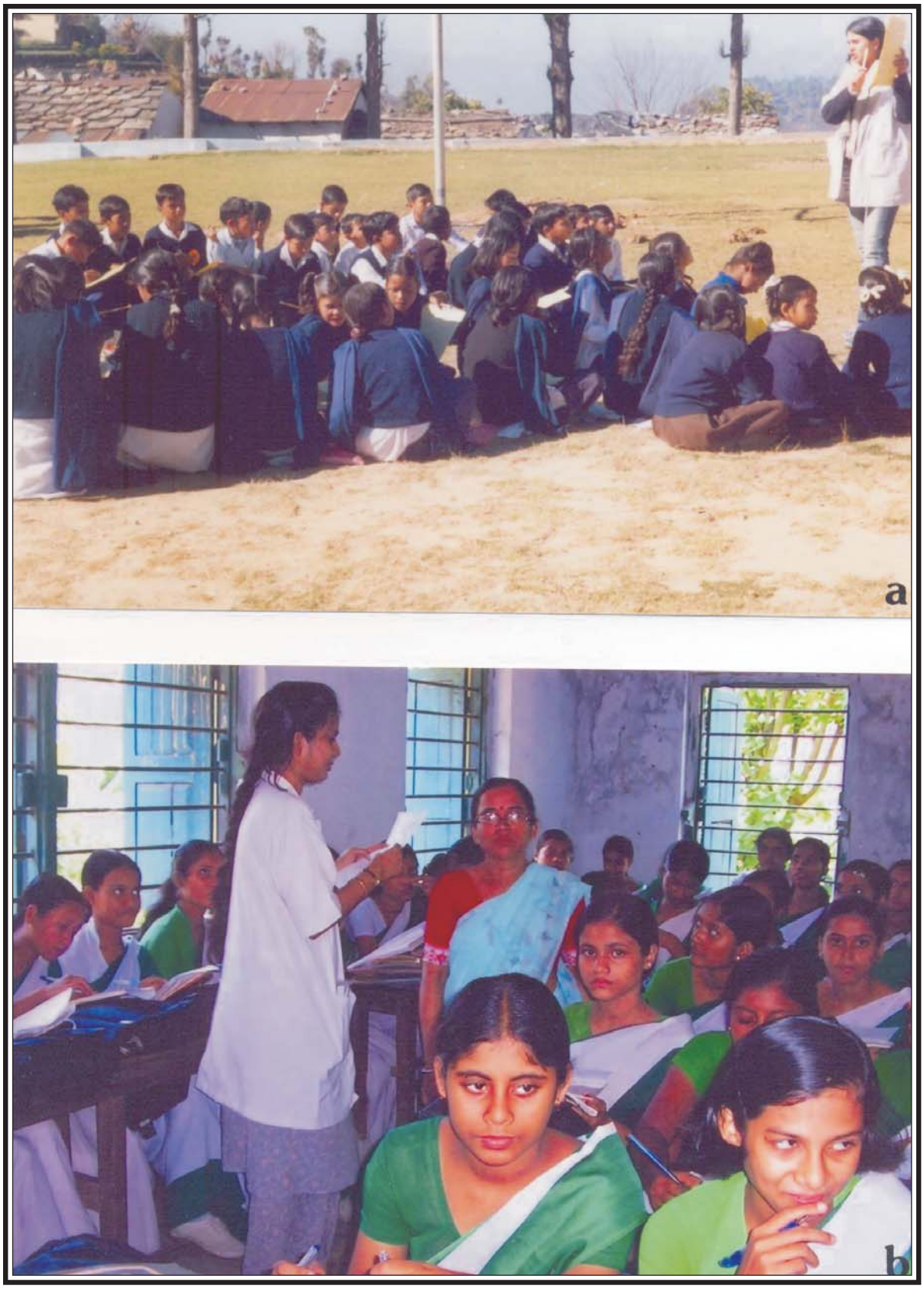


Figure 3.2: Children Health Camp at (a) Government Inter College, Khirsu, Uttaranchal and at (b) Taki S.L. Girls School, 24 Paraganas (N), West Bengal.

(b) Questionnaire Survey for Respiratory Symptoms

Respiratory health of the children has been assessed by idetermining the prevalence of respiratory symptoms; by measuring the lung function and by evaluating the cellular lung response through sputum cytology and cytochemistry. Respiratory symptoms prevalence was determined through a structured respiratory symptomlogy questionnaire based on the questionnaire developed by the British Medical Research Council (BMRC) Cotes, 1987, American Thoracic Society (ATS) and National Heart and Lung Institute (NHLI) Division of Lung Diseases (DLD) questionnaire (ATS-DLD-78-C; Ferris, 1978), and the International Union Againt Tuberculosis and Lung Disease (IUATLD) bronchial symptoms questionnaire (1984) [Burney et al., 1989]. The questions mainly addressed respiratory symptoms, such as attacks of shortness of breath, wheezing, dry or wet cough and chest tightness. The questions were generally formulated to elicit information covering the last 12-month period of life of the child. It also included extended items on asthma, asthma-like symptoms, and allergies.

After obtaining consent from the school Principal, respiratory health questionnaires were distributed among the school children. The children were instructed through class teachers to take home the questionnaire and ask their parents or guardians to fill these up. Filled up questionnaire forms signed by the parent or guardian and countersigned by the class teacher were collected from the respective schools. Information was obtained regarding:

- Age, sex, height and weight, calculated body mass index in kg/m^2
- Prevalence of upper respiratory symptoms (URS) like sinusitis, rhinitis (running or stuffy nose), common cold and fever and sore throat and in past 3 months and one year
- Prevalence of lower respiratory symptoms (LRS) like chronic wet or dry cough, wheeze, heaviness in chest or chest pain, disturbed sleep due to breathing problem in past three months and one year
- Prevalence of asthma symptoms such as history of dispend attacks associated with wheezy breathing at any time in the last twelve months (Golshan et al., 2002) and medically diagnosed asthma established from parents' answer to the questions "Has a doctor ever told you that your child had asthma?" (Eisner, 2002).
- Prevalence of symptoms related to carbon monoxide exposure like headache, dizziness and eye irritation
- Information was also collected for congenital abnormalities, recent illness and history of medication, if any.
- Schools' proximity to main road
- Type of cooking fuel use at home, education level of the parents, average family income, passive smoking as defined by number of smokers at home
- The amount of time the child spends outside, their overall activity and behavior like responsiveness, physical activity, memory, aptitude for sports, music, and reading; personality, hobby etc.

(i) Establishment of socio-economic status

Socio-economic status (SES) of the child's family was ascertained following the procedure of Srivastava (1978) and Tiwari et al., (2005) by scoring 0 to 10 of seven indicators: house, material possession (household gadgets, conveyance etc.), education of the parent or guardian (score 0

for illiterate, 10 for Ph.D., M.D, M.E. etc.), occupation of the parents/guardian (0 for no gainful employment, and 10 for Class I or equivalent jobs), monthly income of the family (per capita income of the family Rs.500/- and below got a score of 2, and >15,000 in urban and >10,000 in rural got a score of 10), cost of the land/house possessed by the family (0 for no land/house, 10 for costing >50 lakh), social participation and understanding. Scores of seven profiles were added and classified into 3 categories-low, medium and high.

(ii) Statistical analysis

The collected data were processed and analyzed in EPI info 6.0 and SPSS (Statistical Package for Social Sciences) software. Chi-square test was done for dichotomous or multinomial qualitative variables, and the Student's t-test for quantitative variables of normal distribution and homogeneous variances. A descendant stepwise logistic regression adjusted over potential confounding variables was carried out for multivariate analysis.

(iii) Ethical clearance

Institutional Ethics Committee of Chittaranjan National Cancer Institute (CNCI), Kolkata approved the study protocol.

3.3 RESULTS

(a) Response to questionnaire survey

12,754 questionnairs were filled by the parents, but 1126 were not accepted due to incomplete filling, resulting in 11,628 completely filled in questionnaires.

For the control group of children in rural areas of West Bengal and Uttaranchal, altogether 4875 questionnaires were filled but 339 were excluded due to incomplete filling, resulting in 4536 completely filled in questionnaires.

(b) Demographic characteristics

The characteristics of both groups of children are presented in Table 3.3. It is evident that the children of Delhi (case) and rural areas of Uttaranchal and West Bengal (control) were comparable ($p>0.05$) with respect to age, gender, BMI, parental smoking, religion and food habit. However, the control group had lower parental education and family income than that of Delhi's ($p<0.05$). Moreover, a substantially increased number of households of control children ($p<0.05$) used biomass fuel such as dung, firewood and agricultural refuse (dried leaves, hay, jute stick etc.) for domestic cooking

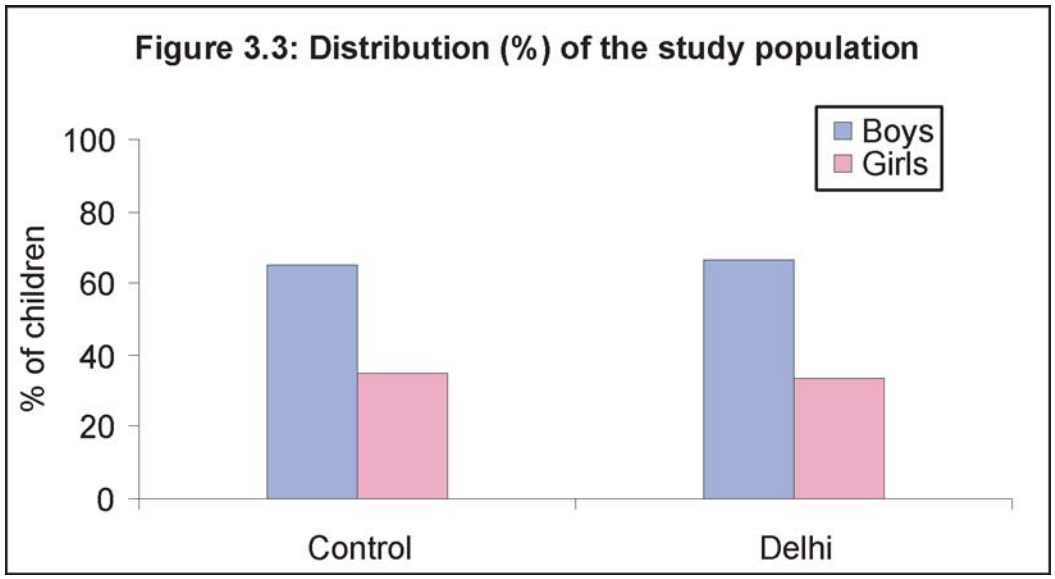
Table 3.3: Demographic Characteristics of the Children.

Characteristics	Control(n=4536)	Delhi (n=11628)	p-value
Median age in years	13.2	13.0	NS
Boys: Girls	1.86	2.0	NS
Height in cm ± SD	149.5 ± 10.5	151.5 ± 11.3	NS
Mean body weight in kg ± SD	38.0 ± 10.3	42.5± 12.9	NS
Mean BMI in kg/m^2 ± SD	16.8 ± 3.8	18.2± 3.9	NS
Parental smoking (%)	28.2	26.8	NS
Parents' education (%)			
Up to 5 years of schooling	11.8	9.8	<0.05
10 years of schooling	37.1	30.8	<0.05
Graduate	47.0	52.7	<0.05
Postgraduate	3.6	6.1	<0.05
Professional	0.5	0.6	NS
Religion (%)			
Hindu	88.6	87.8	NS
Muslim, Sikh, Jain and others	11.4	12.2	NS
Food habit (%)			
Vegetarian	4.7	6.4	NS
Mixed	95.3	93.6	NS
Cooking fuel use at home (%)			
LPG	94.8	99.4	<0.05
Kerosene	0.5	0.4	NS
Biomass	4.7	0.2	<0.05
Average family income/month (Rs.)	4700	9800	<0.05

NS, statistically not significant

(c) Gender

Among the 11,628 school-going children participated in this study, 7757 were boys (66.7%) and 3871 were girls (33.7%). In 4536 control children, 2950 (65%) were boys and 1586 (35%) were girls as depicted in Figure 3.3.



(d) Age

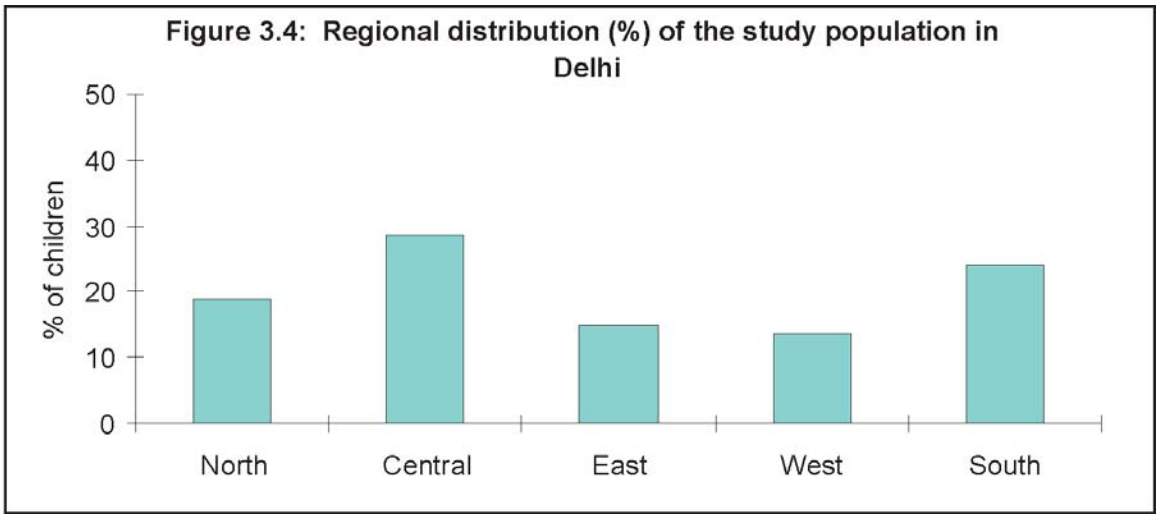
The age of the children varied between 4 and 17 years. Majority of these children (around 55%) both in urban and rural groups were 12 to 14 years old as presented in Table 3.4.

Table 3.4: Distribution (%) of children in different age groups

Age in years	Control	Delhi
4-5	0.1	0.1
6 - 8	0.9	1.2
9 - 11	14.0	15.3
12 – 14	55.7	54.2
15 – 17	29.2	29.2

(e) Regional distribution in Delhi

About 28% of children were from central Delhi, and 24% was from southern part of the city. Children from north, east and west Delhi constituted 18.9%, 14.9%, and 13.5% respectively of the study group as depicted in Figure 3.4.



(f) Seasonal distribution

The study was conducted in all three seasons i.e. summer, monsoon and winter. Out of the total 11628 participants in Delhi, 26.1% children were examined in winter, 19.6% in summer and 54.3% in monsoon. To minimize the impact of seasonal variation on health outcome, the rural children were examined simultaneously and the proportion of children examined in each season was made comparable with that of urban group. For instance, 20.8% of rural children were examined in summer, 27% in winter, and 51.1% in monsoon.

(g) Socio-economic status (SES)

About 32% of urban children participated in this study (3702 out of 11628) belonged to families of high SES. In contrast, only 14% rural children (647 out of 4536) had high SES ($p<0.05$). On the other hand, 35.3% (1604 of 4536) of control children were from families that had low social and

economic status against 21.2% participants in Delhi (2463 out of 11628) with similar background. About half of the children in control and 47% in Delhi belonged to medium SES.

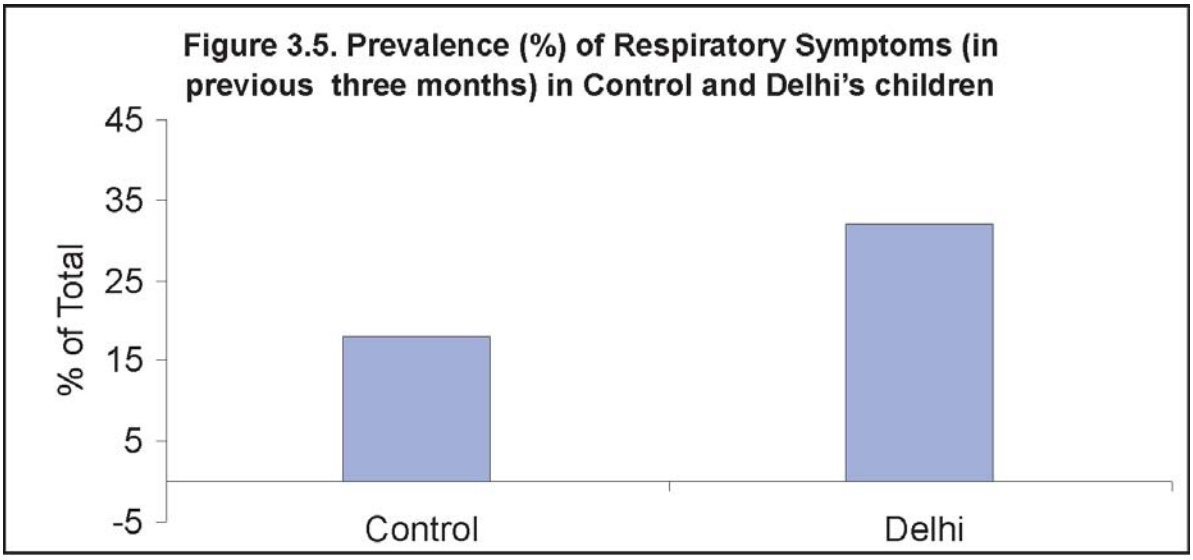
3.3.1 Prevalence of Respiratory Symptoms

The prevalence of respiratory symptoms was recorded through questionnaire survey and personal interview by the researchers. One or more respiratory symptoms were present in the past 3 months in nearly one-third (32.1%) of Delhi's children (3732 out of 11628). In contrast, 18.2% of rural children (827 out of 4536) had experienced respiratory symptoms (Table 3.5, Fig. 3.5). The difference in period prevalence (in 3 months) of respiratory symptoms between urban (Delhi) and rural (Control) children was highly significant ($p<0.05$) in Chi- square test.

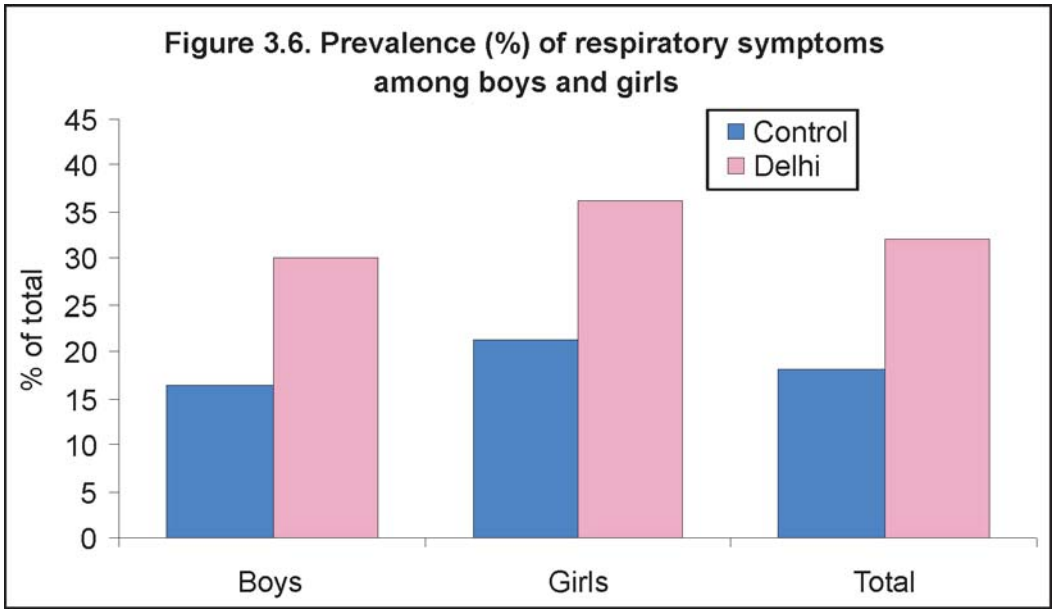
Table 3.5: Prevalence (%) of respiratory symptoms in children in past three months

Group	Control	Delhi
Boys	16.3	30.0*
Girls	22.4	36.3*
Overall	18.2	32.1*

*Many children had more than one symptom; *p<0.05 compared with control in Chi- square test*



The respiratory symptoms were more prevalent in girls than in boys. This is true both in rural and urban settings. For example, respiratory symptoms were present in 36.3% of girls in Delhi against 30% of the city's boys. Similarly, 21.2% of girls from rural areas had respiratory symptoms compared with 16.3% of rural boys (Fig. 3.6). The girls: boys ratio in symptom prevalence was 1.21 in Delhi and 1.3 in rural areas included in this study. Thus, the gender difference in the prevalence of respiratory symptoms was wider in rural areas. The prevalence of respiratory symptoms in children of Delhi also correlated with the PM_{10} level. In summary, respiratory symptoms were significantly more prevalent among the children of Delhi compared with their rural counterparts, and girls suffered more than the boys in both rural and urban settings.



Respiratory symptoms were broadly classified into two: upper respiratory symptoms (URS) and lower respiratory symptoms (LRS). Delhi's children exhibited greater prevalence of both URS (23.1 vs. 14.6%) and LRS (17.0 vs. 8.0%) than the controls (Fig. 3.7). The prevalence of respiratory symptoms (RSC) varied in different parts of the city. It was maximum in the residents of Central Delhi where the level of particulate pollution was also high. The concentration of PM_{10} was least in South Delhi and this reflected in the prevalence of RSC in the residents of this part of the city; prevalence being the lowest. Respiratory symptoms, both URS as well as LRS, was more prevalent during winter followed by summer and monsoon, corresponding with the concentration of particulate matter, concentration being highest during winter and lowest during monsoon (Table 3.6; Fig. 3.8).

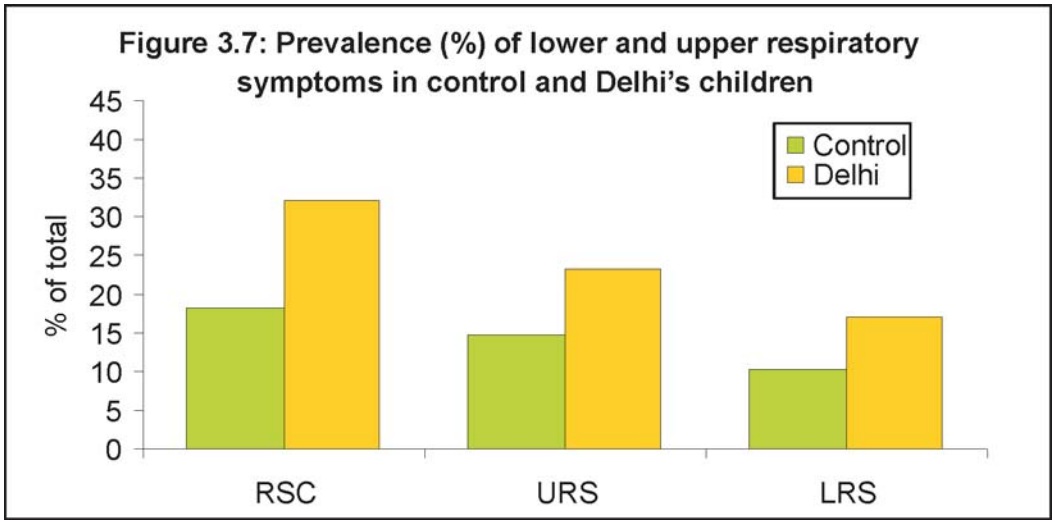
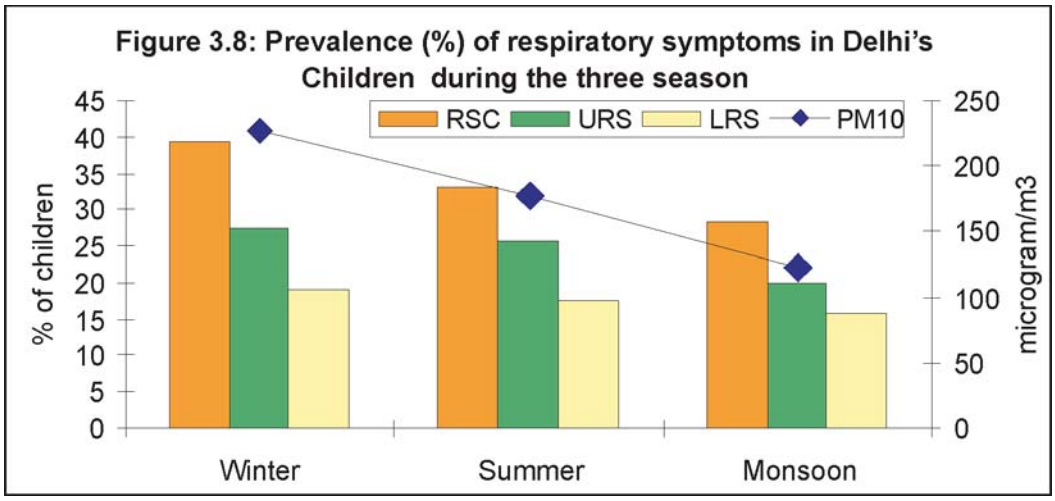


Table 3.6: Prevalence (%) of respiratory symptoms in different seasons

	Winter(3035)	Summer(2279)	Monsoon(6314)
Respiratory symptoms (RSC)	39.3	33.0	28.3
Upper respiratory symptoms (URS)	27.5	25.5	20.1
Lower respiratory symptoms (LRS)	19.2	17.5	15.8



(a) Prevalence of Upper Respiratory Symptoms (URS)

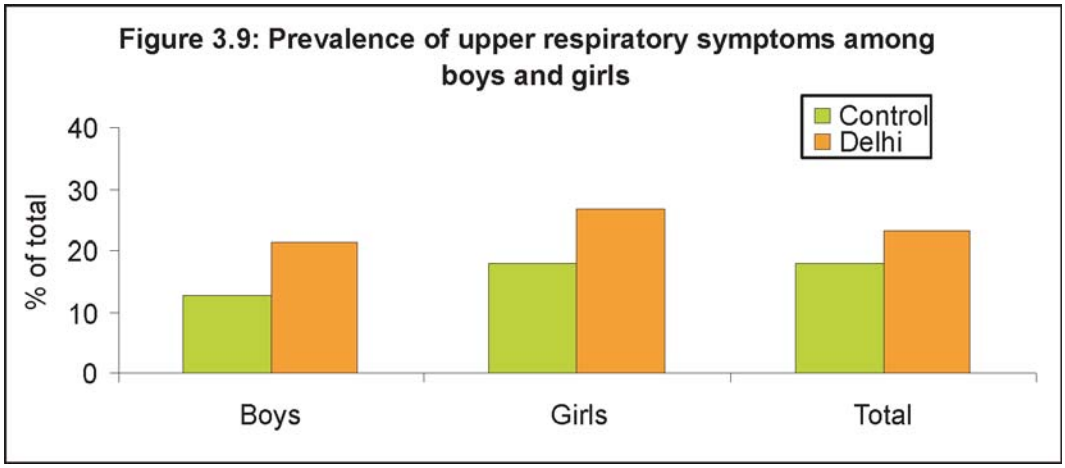
The prevalence of five URS -sinusitis, rhinitis (running or stuffy nose), sneezing, sore throat and common cold with fever were the most prevalent examined in this study. In general, 23.1% children of Delhi had experienced one or more of these URS in the past 3 months. In comparison, 14.6% of control children had URS. The difference in the prevalence of URS between Delhi and control group was significant ($p < 0.05$) in Chi-square test (Table 3.7).

Table 3.7: Prevalence (%) of upper respiratory symptoms in past three months

Group	Control	Delhi	P value
Boy	12.8	21.4	<0.001
Girl	18.0	26.5	<0.01
Overall	14.6	23.1	<0.001

Many children had more than one symptom

URS was more prevalent in girls than in boys in Delhi (26.5 vs. 21.4%) as well as in control group (18.0 vs. 12.8%, $p<0.05$). The boys: girls ratio of URS prevalence was 1.24 in Delhi and 1.40 in control, implying that URS had female predominance especially in rural areas (Fig. 3.9).



(i) Prevalence of Sinusitis

Sinusitis is an inflammation of one or more of the paranasal sinuses. The symptoms are nasal congestion, facial pain, headache, fever, cough and purulent discharge. Sinusitis can be subdivided into acute (symptoms present for less than 4 weeks), sub acute (symptoms for 4-8 week), chronic (symptoms for 8 week or longer of varying severity) and recurrent (3 or more episodes of acute sinusitis per year). The diagnosis is based on symptoms, clinical history and physical examination of the child. Sinusitis was present in 3.4% children of Delhi compared with 3.2% of control children (Table 3.8), suggesting little difference between Delhi and control groups.

Table 3.8: Prevalence of sinusitis among schoolchildren

Group	Prevalence in past 3 months (%)		
	Boys	Girls	Total
Control (n=4536)	3.2	3.1	3.2
Delhi (n=11628)	3.5	3.2	3.4

Sinusitis prevalence was slightly higher in boys than in girls both in Delhi and control groups, but the difference were not significant. Children from west Delhi showed remarkably higher prevalence of sinusitis (5.8%) compared with children from north (3.0%), south (2.9%), east (3.0%) and central Delhi (3.6%). The symptom was more common during monsoon (3.9% against 3.0% in winter and 2.6% during summer). Children from middle (3.8%) and low SES (3.7%) had greater prevalence of sinusitis than children from high SES (2.6%). The prevalence of sinusitis increased progressively with age (Table 3.13, Fig. 3.13).

(ii) Running or stuffy nose

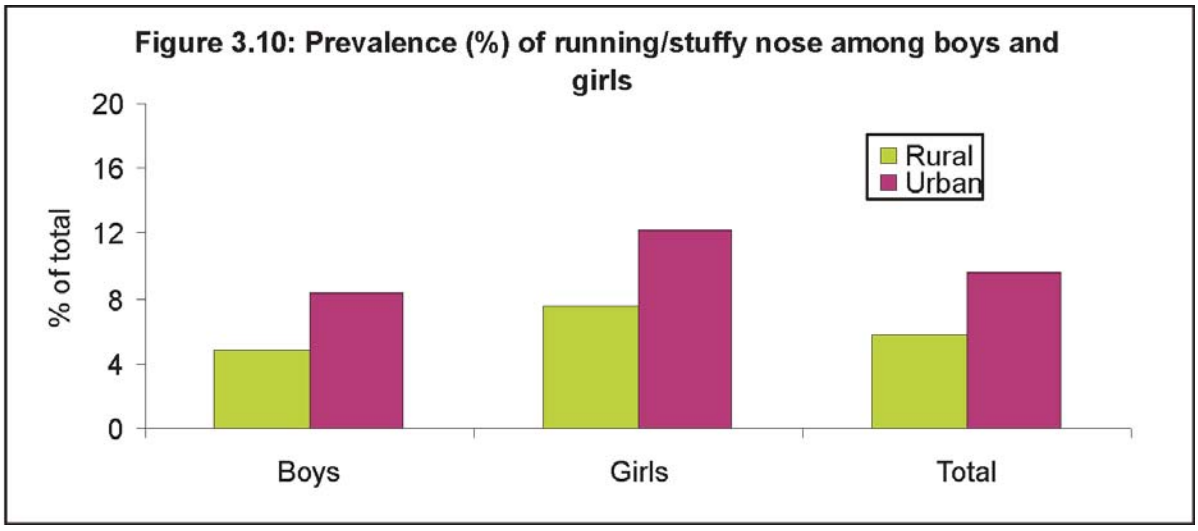
Running or stuffy nose is associated with rhinitis. In nearly 90% cases rhinitis in children is caused by hypersensitivity i.e. allergic reactions to a host of environmental allergens. The symptom was present in 9.6% children of Delhi (1116 out of 11628) compared with 5.8% (263 out of 4536) in control, and the difference was highly significant ($p<0.05$, Table 3.9).

Table 3.9: Prevalence of running or stuffy nose among children in past three months

Group	Prevalence (%)		
	Boys	Girls	Total
Control	4.8	7.5	5.8
Delhi	8.3*	12.2*	9.6*

**p<0.05 compared with respective control in Chi-square test*

Girls suffered more from this problem than the boys both in Delhi and in rural areas. The affected girls: boys ratio was 1.56 in control and 1.47 in Delhi (Fig. 3.10).



Children from west (12.9%), east (12.5%) and central Delhi (11.2%) suffered more from running or stuffy nose than children from north (8.5%) and south Delhi (4.8). The symptom was most prevalent during winter (10.3%) but the prevalence declines in summer (9.2%) and especially during monsoon (7.2%; OR= 1.85 and 95% CI, 1.28 – 2.46 for winter vs. summer). Children from low SES had highest prevalence of running nose both in Delhi (14.6%) and in control group (7.0%), while children from high SES had lowest prevalence (6.5% in Delhi and 3.8% in control). Its prevalence was more in younger age group especially in 6-8 (12.5%) and 9-11 years (11.3%) of age (Table 3.13, Fig. 3.13).

(iii) Sneezing

Sneezing, a respiratory allergy was found in 1058 children of Delhi (9.1%) and 236 in control group (5.2%). Thus, Delhi's children had 1.7-times more prevalence of sneezing than control, and the difference was highly significant ($p<0.001$). Like running or stuffy nose, sneezing was more prevalent in girls both in rural and urban settings (Table 3.10). In control group, 6.4% of the girls (102/1586) had this symptom compared with 132 (4.5%) of the boys ($p<0.05$). In Delhi, 426 girls (11%) and 632 boys (8.1%) had this symptom ($p<0.05$).

Table 3.10: Prevalence of sneezing among children in past three months

Group	Prevalence (%)		
	Boys	Girls	Total
Control	4.5	6.4	5.2
Delhi	8.1*	11.0*	9.1*

**p<0.05 compared with respective control in Chi-square test*

Prevalence of sneezing was highest in children of the age group age group of 9 – 11 (11.2%) and 6-8 years (9.3%, Table 3.13, Fig. 3.13), those residing in west Delhi, and during winter (9.4%) and monsoon (9.0%, Fig. 3.14). Frequency of the symptom was identical in children belonging to low and medium SES (9.2%) and lightly lowers in high SES (8.9%, Figure 3.14).

(iv) Sore throat

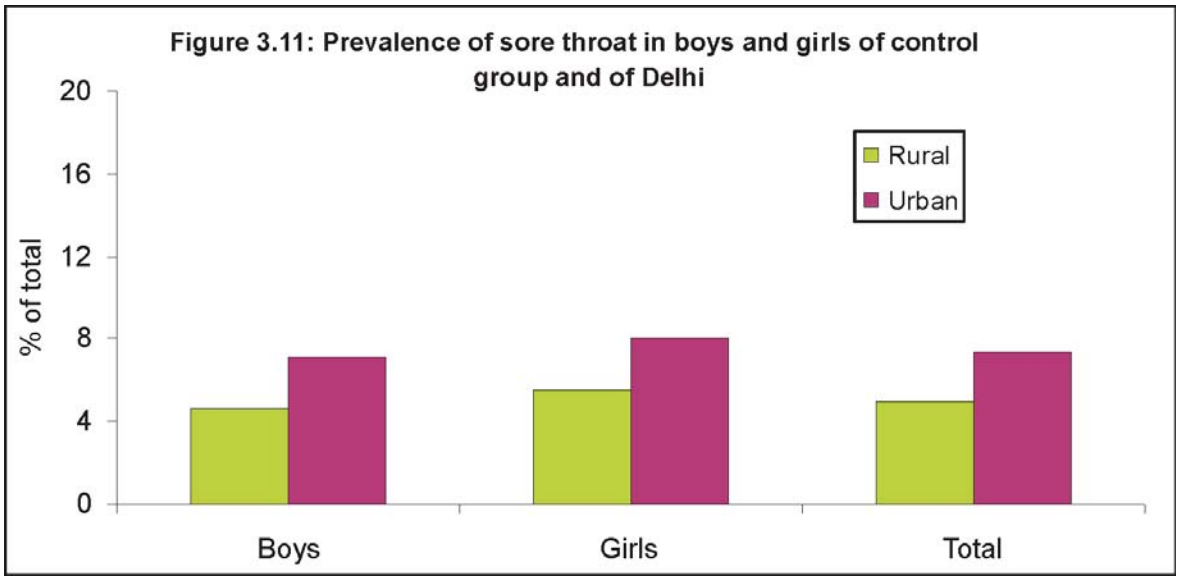
Sore throat was reported by 861 (7.4%) children of Delhi compared with 223 (4.9%) of controls (Table 3.11). The difference between Delhi and control group in this regard was significant ($p<0.05$).

Table 3.11: Prevalence of sore throat among children in past three months

Group	Prevalence (%)		
	Boys	Girls	Total
Control	4.6	5.5	4.9
Delhi	7.1*	8.0*	7.4*

**p<0.05 compared with respective control in Chi-square test*

Sore throat was slightly more prevalent among girls, but the difference was not significant (Fig. 3.11). The prevalence was highest during winter (9.3%), followed by summer (7.2%) and monsoon (5.9%). and children from west (6.7%) and east Delhi (6.3%) had higher prevalence than did children from rest of the city.



Children belonging to the age group of 12 – 14 years showed highest prevalence (8.6%) of sore throat (Table 3.13, Fig. 3.13). The symptom was most prevalent in children from low SES both in Delhi (9.1%) and control group (6.4%), while lowest prevalence was found in high SES (2.0% in control and 5.8% in Delhi).

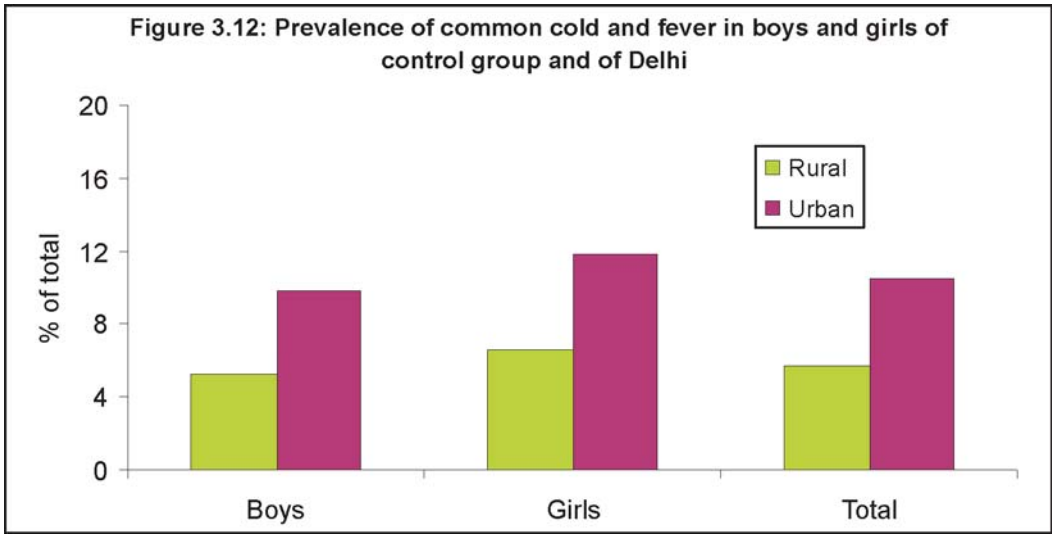
(v) Common cold and fever

More than ten per cent children of Delhi (1221/11628) suffered from common cold and fever in the past 3 months compared with 259/4536 (5.2%) in control ($p<0.01$, Table 3.12). The symptom was more prevalent among girls than in boys (Fig. 3.12). Children from west (14.4%) and East Delhi (11.4%) had relatively higher prevalence while the symptom was least prevalent among the children from Central Delhi (4.5%).

Table 3.12: Prevalence of common cold and fever among children in past three months

Group	Prevalence (%)		
	Boys	Girls	Total
Control	5.2	6.6	5.2
Delhi	9.8*	11.8*	10.5*

**p<0.05 compared with respective control in Chi-square test*



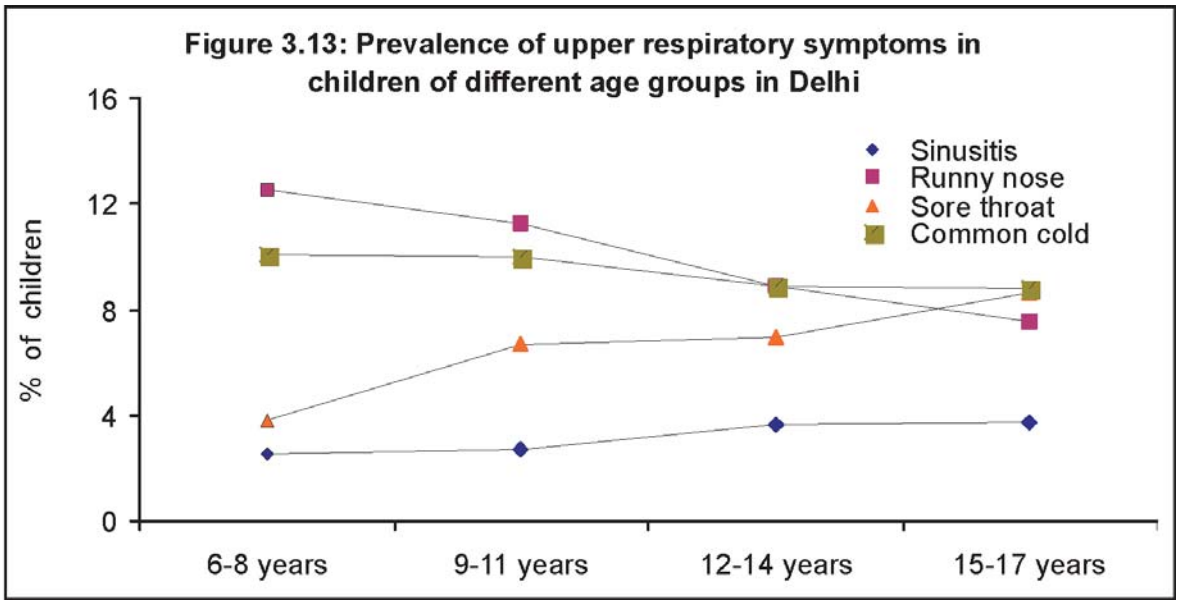
The symptom was highest during the winter months (12.2%) when the PM_{10} level in ambient air was highest. The prevalence declined to some extent in summer (8.9%) and more so in monsoon (7.7%, Fig. 3.14). Children of younger age group (6-11 years) had more symptoms than 12-17 year-old children. For example, 13.2 and 12.5% children had common cold in previous three months compared with 9.9 and 9.4% children aged 12-14 and 15-17 years respectively (Table 3.13, Fig. 3.13). Prevalence of common cold and fever was inversely associated with socio-economic conditions as children from low (10.7%) and medium SES in Delhi (10.0%) suffered more than children belonging to high SES (8.1%; Fig. 3.15). Similarly, in the control group, the symptom was highest in low (6.6%) and lowest in high (2.6%) SES.

(vi) URS in different age groups among the children of Delhi

In general, children belonging to younger age groups (6-11 yr) suffered more from running/stuffy nose and common cold, while children aged more than 11 years had greater prevalence of sinusitis and sore throat (Fig. 3.13, Table 3.13).

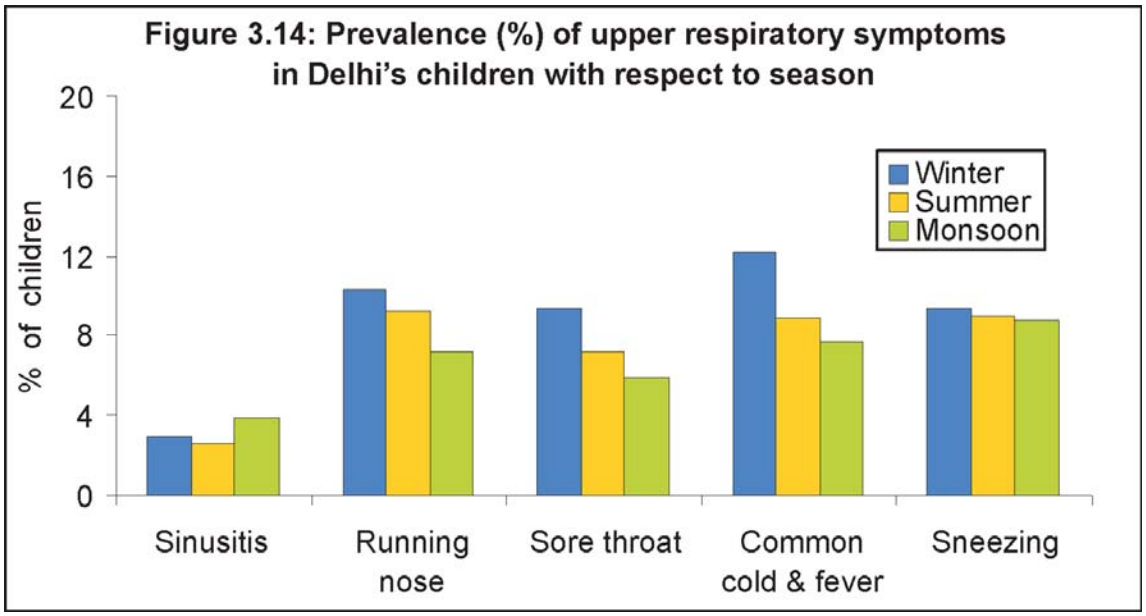
Table 3.13: Prevalence (%) of URS in different age groups of Delhi's children

Symptoms	6-8 years	9-11 years	12 – 14 years	15 – 17 years
Sinusitis	2.5	2.7	3.6	3.7
Running nose	12.5	11.3	8.9	7.5
Sore throat	6.7	6.9	8.6	7.3
Sneezing	9.3	11.2	8.3	5.5
Common cold	13.2	12.5	9.9	9.4



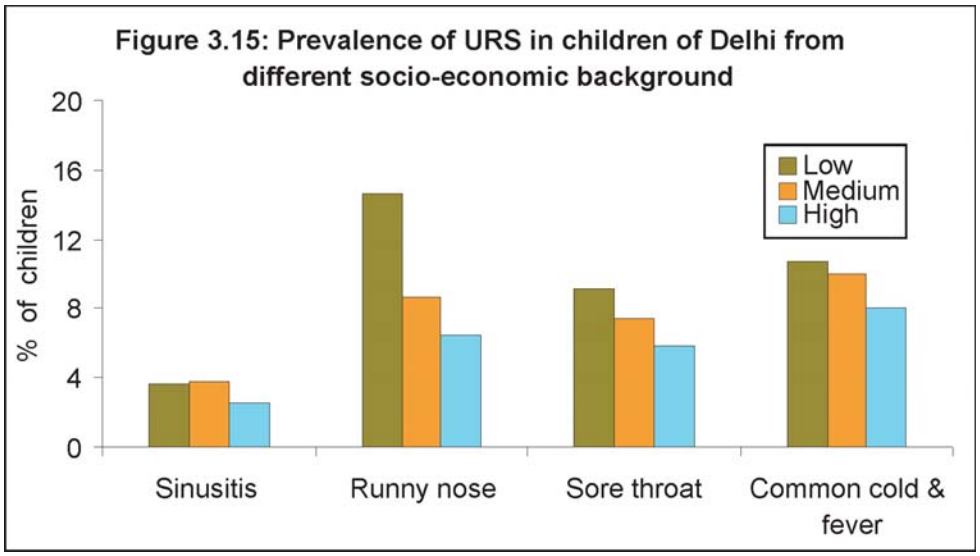
(vii) Prevalence of URS in Delhi in relation to season

Except for sinusitis which was most prevalent during monsoon, the remaining four symptoms under URS viz. running/stuffy nose, sore throat, sneezing and common cold were more prevalent in children during winter (Fig. 3.14).



(viii) Prevalence of URS in Delhi in relation to socio-economic status

URS was more prevalent in children from families with low SES both in Delhi and control group (Fig. 3.15).



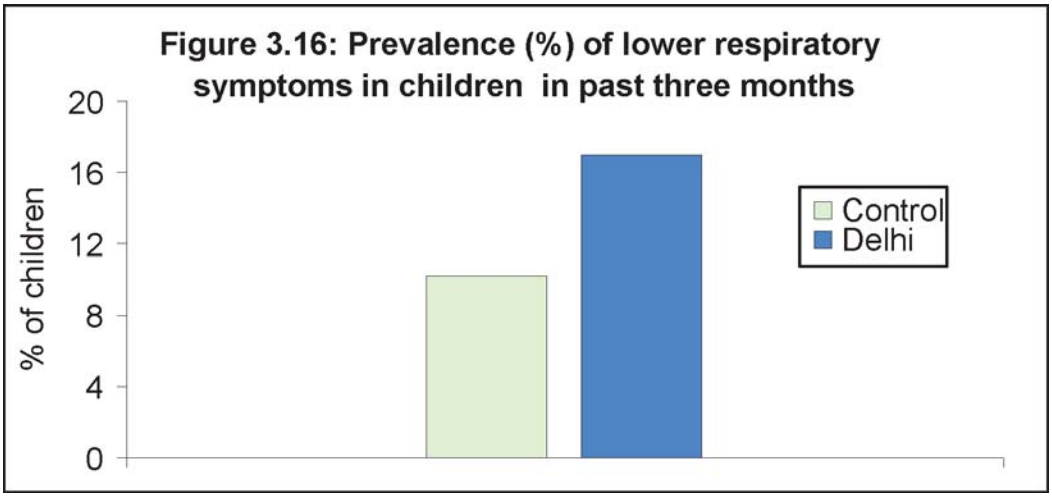
(b) Prevalence of lower respiratory symptoms (LRS)

The prevalence of six LRS, viz. dry cough, cough with phlegm, wheeze, breathlessness on exertion, chest discomfort and sleep disturbance due to breathing problem in past three months was examined by questionnaire survey.

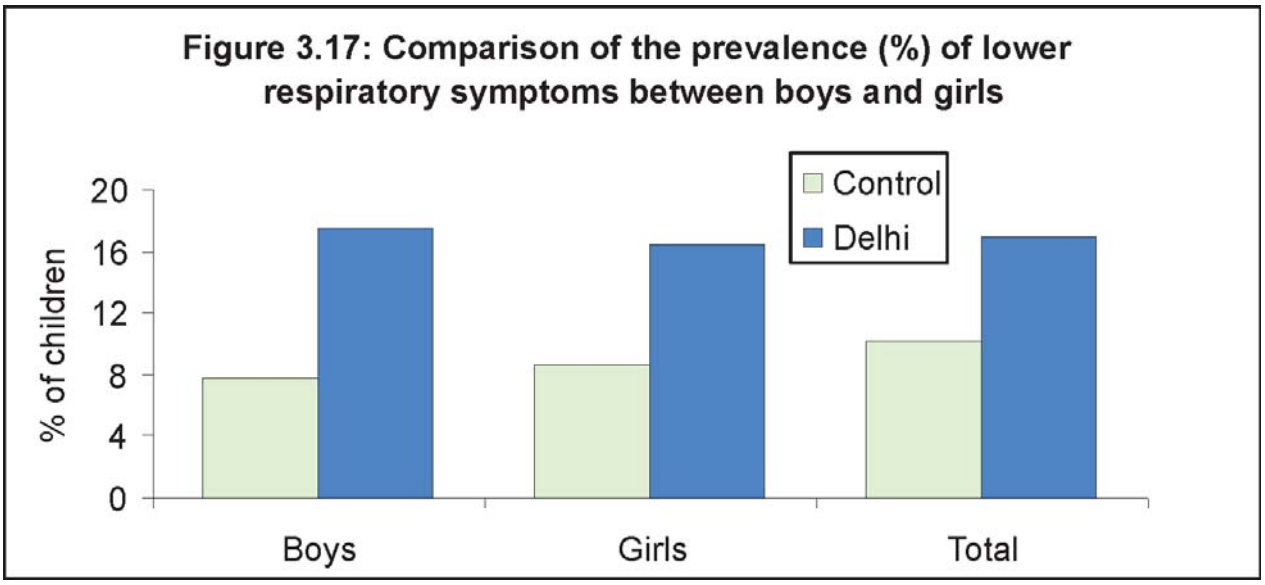
The overall prevalence of LRS was significantly higher ($p<0.001$) among children residing in Delhi when compared with their rural counterparts (control). For example, 17% participants from Delhi (1986 out of 11628) had one or more LRS in contrast to 8% children in control (363 out of 4536, Table 3.14, Fig. 3.16).

Table 3.14: Prevalence (%) of lower respiratory symptoms in children in past three months

Group	Control (n=4536)	Delhi (n=11628)	p
Boy	7.7	17.4	<0.001
Girl	8.5	16.4	<0.001
Overall	8.0	17.0	<0.001



Delhi's boys had a slightly greater prevalence of LRS than city's girls (17.4 vs. 16.4%), whereas it was just the opposite in control: the girls had greater prevalence of LRS than the boys (8.5 vs. 7.7%) in control group (Table 3.14, Fig. 3.17).



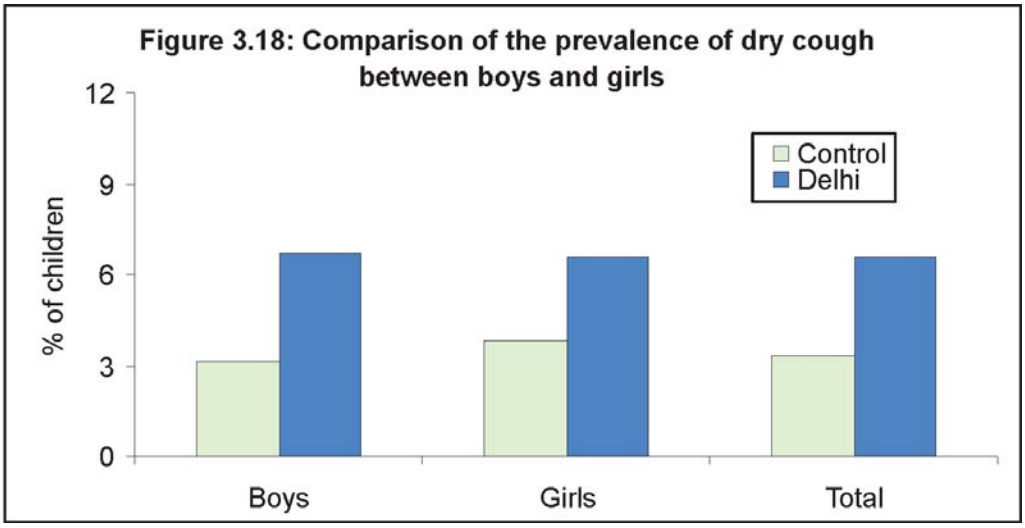
(i) Prevalence of dry cough

Dry cough was present in 773 (6.6%) children of Delhi and 3.3% children of control group ($p<0.05$, Table 3.15). The symptom was more or less similar among boys (6.7%) and girls (6.6%) of Delhi, but girls in the control group had greater prevalence than the boys (3.8 vs. 3.1%, Table 3.15, Fig. 3.18).

Table 3.15: Prevalence (%) of dry cough

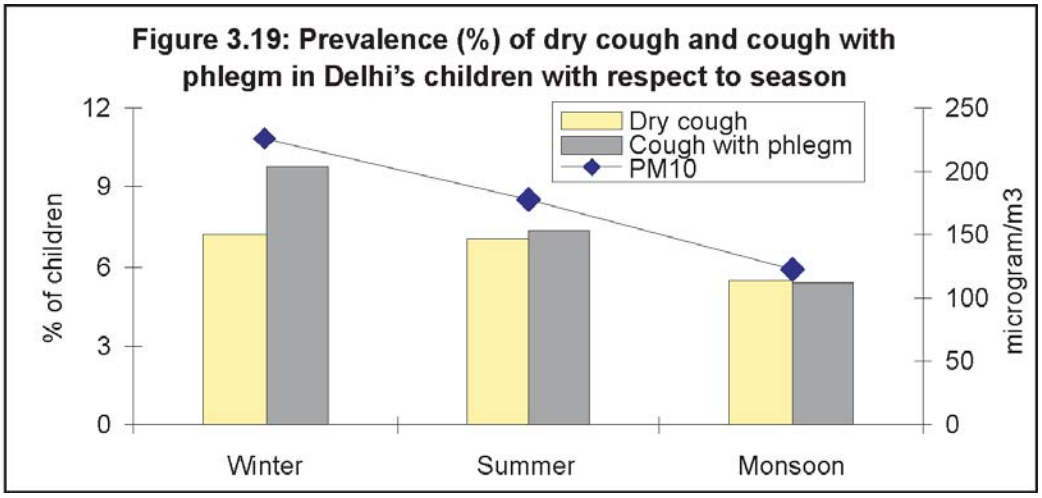
Group	Boys	Girls	Total
Control	3.1	3.8	3.3
Delhi	6.7*	6.6*	6.6*

**p<0.05 compared with control in Chi-square test*



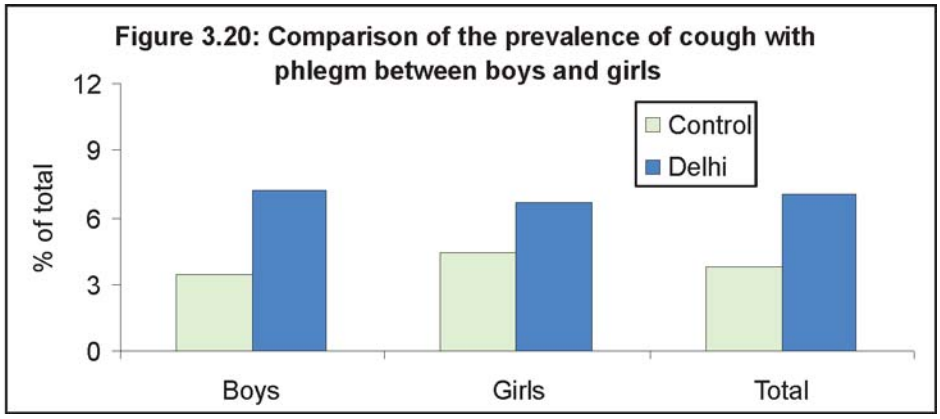
Children from north and central Delhi (7.2 % each) suffered most from this problem while the prevalence was lowest in south Delhi (4.1%).

The problem of dry cough was more frequent in children of Delhi during winter (7.2%) and summer (7.0%) than in monsoon (5.5%; Fig. 3.19). In control group also, dry cough was most prevalent during winter. Children from families with low SES (8.2% in Delhi and 4.5% in control) and in the 6-8 year age group (7.8% and 4.2% in Delhi and control group respectively) suffered most both in urban and rural settings. After controlling potential confounders, dry cough was found to be positively associated with Delhi's air pollution (OR=1.48, 95% CI 1.24 – 1.67).

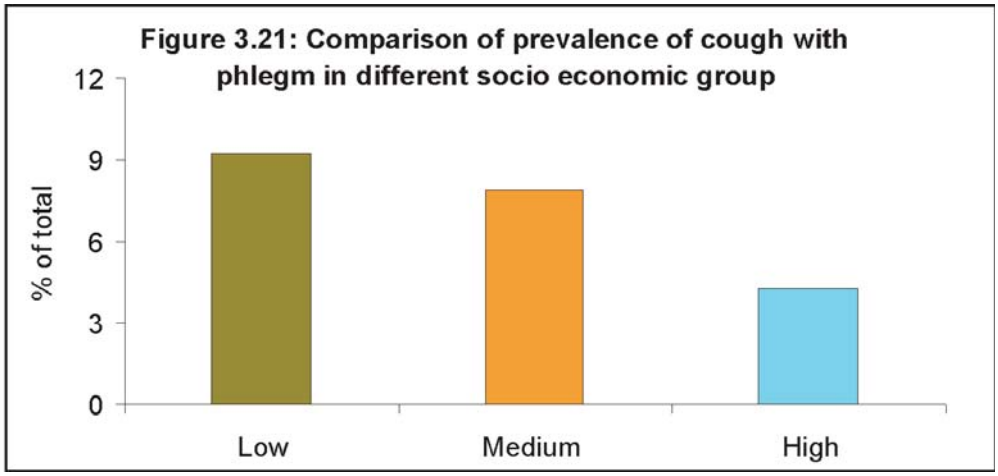


(ii) Prevalence of cough with phlegm: 7% in Delhi compared with 3.8% in control

Cough with phlegm (sputum production) or wet cough was present in 7.0% (817 0f 11628) of Delhi's children. In contrast, only 3.8% of children in control group had this symptom ($p<0.001$). Wet cough was more prevalent in boys (7.2%) than in girls (6.7%) in Delhi, whereas girls experienced more symptoms than the boys in control group (4.4 vs. 3.4%, Fig. 3.20).



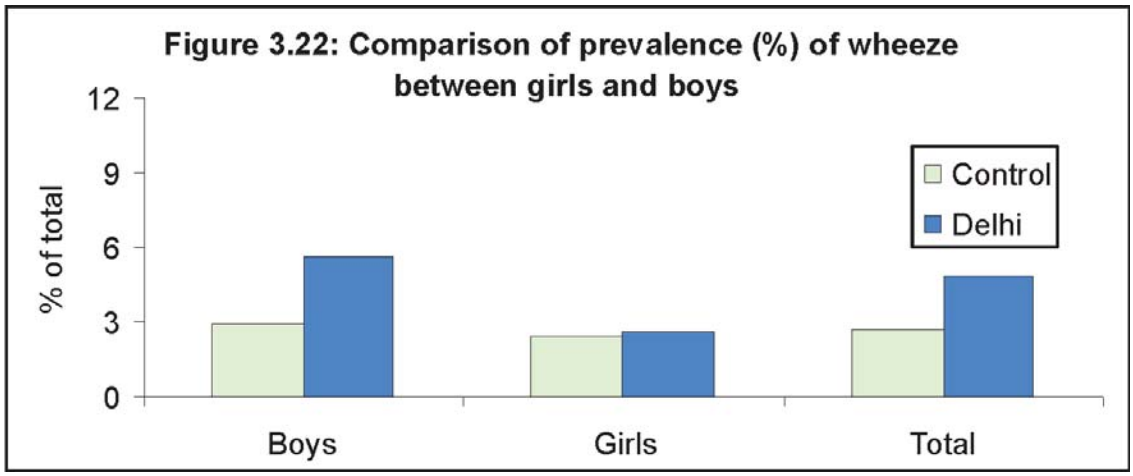
Children from north (8.9%), west (8.7%) and central (8.0%) Delhi suffered more from this symptom did children from east (6.0%) and south (4.0%) Delhi. The problem was more prevalent during winter (9.8%) than in summer (7.4%) and monsoon (5.4%, Fig. 3.19). The differences in prevalence between winter vs. summer (OR = 1.98; 95% CI: 1.34 – 2.71) and winter vs. monsoon (OR= 2.23, 95%CI: 1.55-2.95) were significant. The symptom was less common in children aged 6-8 years (5.8%) compared with those aged 9 years or more (7.6%). Children with low and middle SES were more susceptible to cough with phlegm both in Delhi and control group (Table 3.20). For example, the symptom was present in 9.2% children of Delhi with low SES, 7.9% in medium against 4.3% children from high SES (Fig. 3.21). After controlling potential confounders, cough with phlegm was found to be positively associated with urban life (OR=1.33, 95% CI 1.12 – 1.56) and PM_{10}.

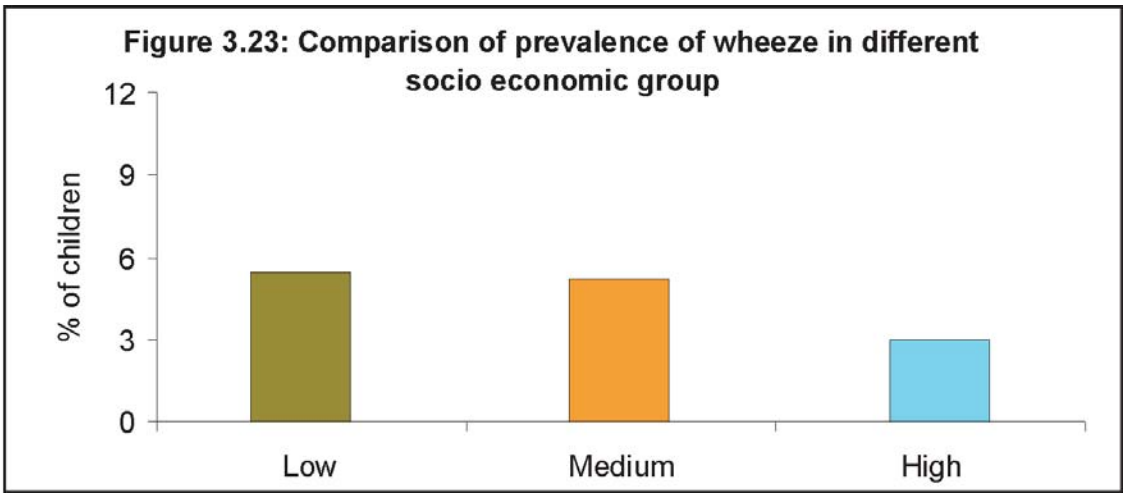


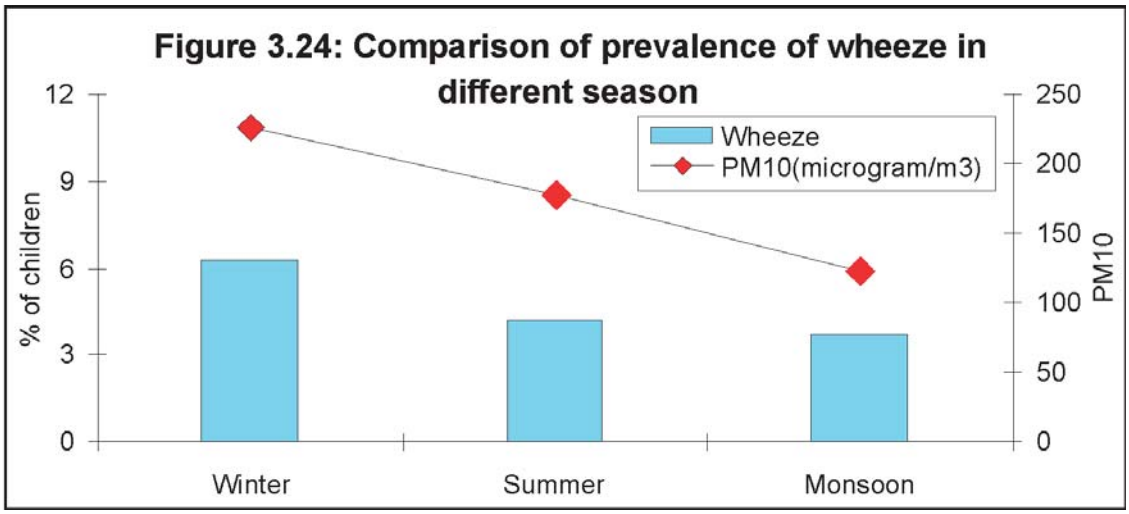
(iii) Wheezing breath

Wheeze i.e. whistling sound during breathing was present in 4.8% of Delhi's children (535 of 11628) in past three months compared with 2.7% children with this symptom in control (123/4536, Fig. 3.22). Thus, Delhi's children had 1.8-times more wheeze prevalence than the controls, and the difference was highly significant ($p<0.01$). We found marked difference in the prevalence of wheeze in past three months between boys and girls both in rural and urban settings. In both cases, boys had greater prevalence than the girls. For example, 5.6% of the boys experienced wheeze in Delhi compared with 2.6% of the girls ($p<0.05$). In controls also, boys suffered more from wheezing breath than the girls (2.9 vs. 2.4 %, Fig. 3.22). It was also seen that children from

the lower socio economic status suffered most from this problem (Fig. 3.23). Prevalence of wheeze was higher during winter and lowest during monsoon (Fig. 3.24). After controlling potential confounders, wheeze was found to be positively associated with particulate air pollution (OR= 1.87, 95% CI, 1.52-2.31).

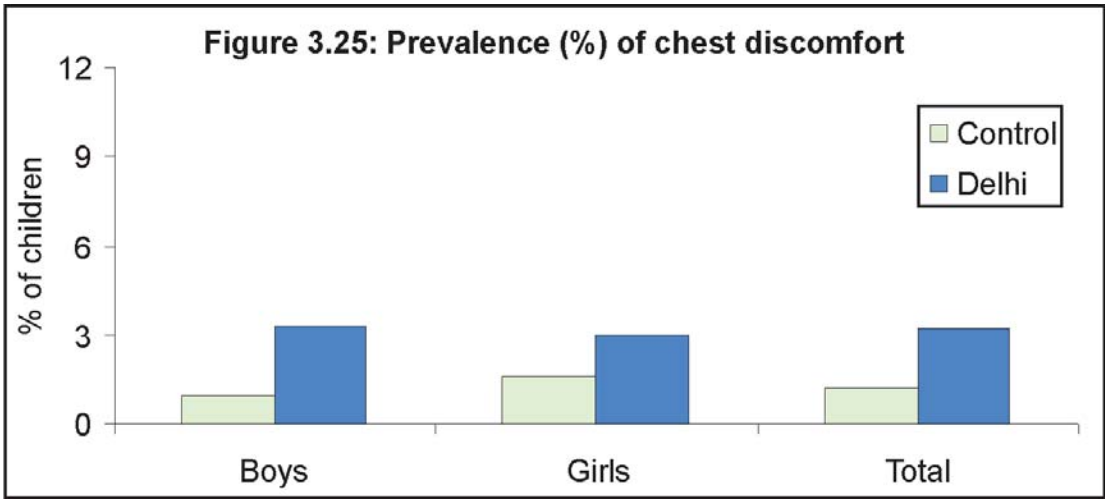






(iv) Chest discomfort and pain: 3.2% in Delhi against 1.2% in control

Pain or tightness of the chest was present in 3.3% of the boys and 3% of the girls of Delhi in previous three months, with an overall prevalence of 3.2%. In contrast, 1.2 % of control children, 1% of boys and 1.6% of girls, had this symptom (Fig. 3.25). The difference between Delhi's and control children in this regard was highly significant ($p<0.001$). The symptom was more frequent in Delhi's children belonging to 9 – 11 year age group. The prevalence was highest during winter when the PM_{10} level in air is highest and lowest during monsoon when the PM_{10} level is lowest. After controlling potential confounders, logistic regression analysis showed that chest discomfort and pain was positively associated with PM_{10} level in breathing air (OR=1.44, 95% CI 1.18 – 1.76).

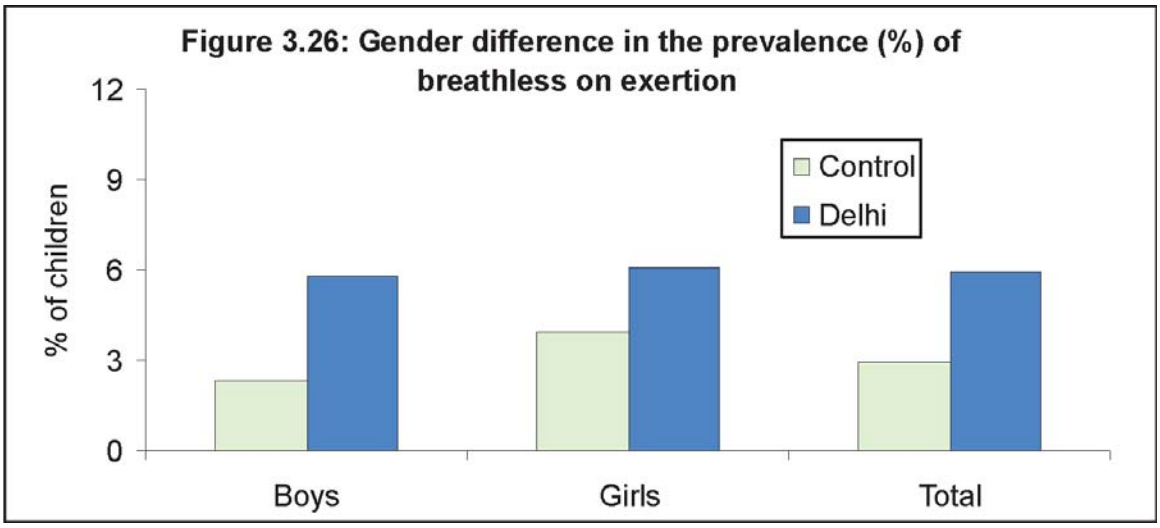


(v) Shortness of breath on exertion: 5.9% in Delhi compared with 2.9% in control

Out of breathe or breathless on exertion was reported by 5.9% children of Delhi compared with 2.9% children in control group ($p<0.001$; Table 3.16). Its prevalence was higher in girls than in boys both in urban and control groups (Fig. 3.26). Children from north and west Delhi suffered most from this problem, and it was more common in 9 – 11 year age group, As in the case of other symptoms, breathlessness was most prevalent during winter and least common during monsoon (OR = 1.93; 95% C.I.: 1.71 – 2.19, winter vs. monsoon), suggesting a positive association between particulate pollution and breathlessness. Indeed, logistic regression analysis has shown such an association between PM_{10} level in breathing air and breathlessness (OR=1.37, 95% CI 1.15 – 1.63). Children from lower SES suffered most from this symptom (Table 3.21).

Table 3.16: Prevalence (%) of breathless on exertion

Group	Boys	Girls	Total
Control	2.3	3.9	2.9
Delhi	5.8*	6.1*	5.9*



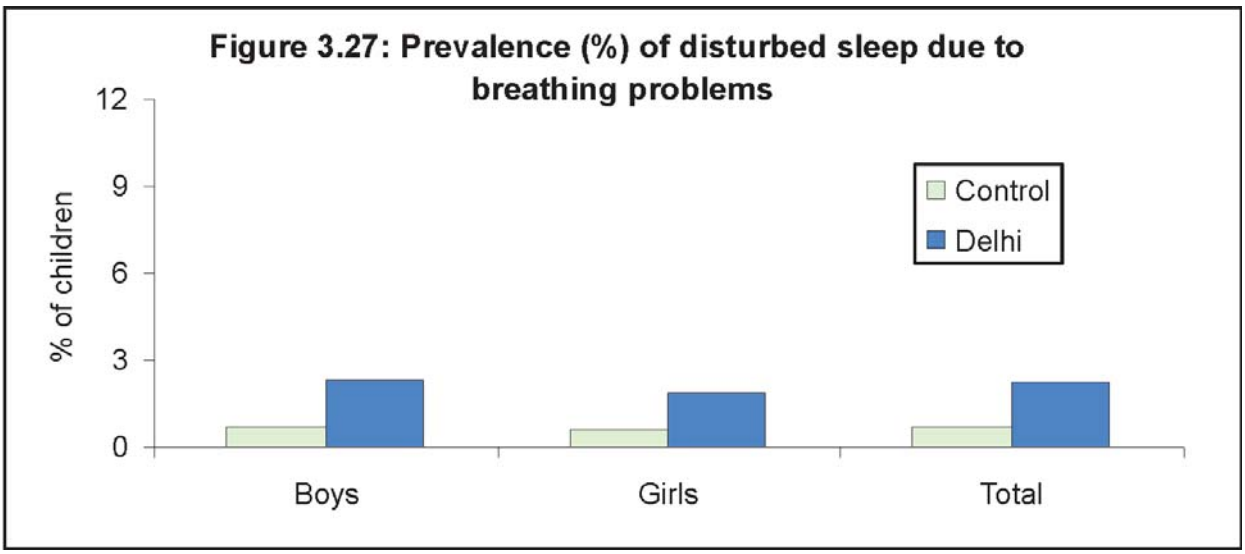
(vi) Disturbed sleep due to breathing problem

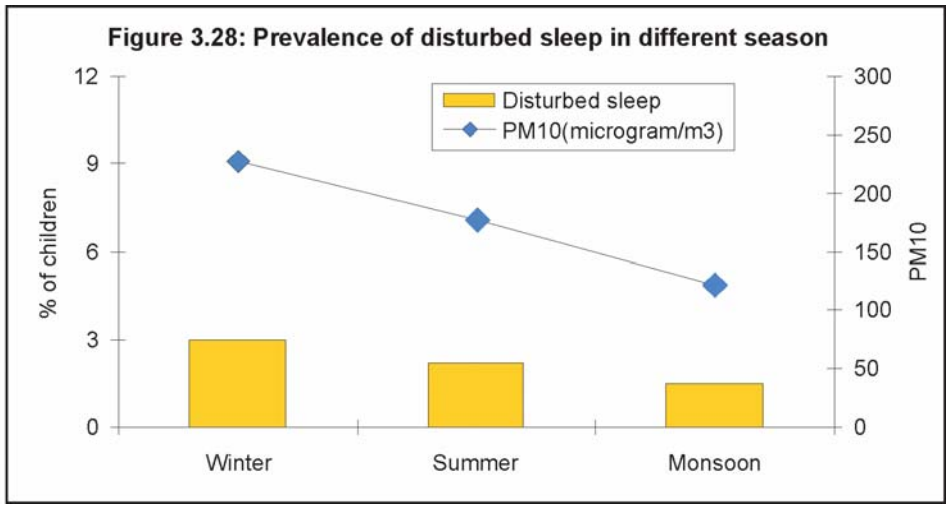
Disturbed sleep due to breathing problem was reported by 2.2% children of Delhi compared with 0.7% of rural children ($p<0.001$, Table 3.17). The prevalence was slightly higher in boys than in girls both in Delhi and control group, but the difference was not statistically significant ($p>0.05$, Fig. 3.27). The problem was most prevalent during winter and least in monsoon, implying a positive association with air pollution (Fig. 3.28). After controlling potential confounders, the symptom was indeed found to be positively associated with PM_{10} level (OR=1.68, 95% CI 1.35 – 2.26).

Table 3.17: Prevalence (%) of disturbed sleep due to breathing problems

Group	Boys	Girls	Total
Control	0.7	0.6	0.7
Delhi	2.3*	1.9*	2.2*

**, p<0.001 compared with control in Chi-square test*





Children in the age group of 9 – 11 years and those belonging to low SES suffered most from disturbed sleep owing to breathing problems both in rural and urban settings (Table 3.18).

Table 3.18: Prevalence (%) of disturbed sleep in children from different socio-economic background

Group	Low SES	Medium SES	High SES
Control	1.0	0.6	0.3
Delhi	3.1*	2.1*	1.7*

**, p<0.05 compared with corresponding control*

(c) Statistical evaluation of the data

(i) Inverse relationship between SES and prevalence of URS and LRS

We found an inverse relationship between SES and the prevalence of all the respiratory symptoms: lower the SES greater the prevalence. Keeping the prevalence of individual symptoms in high SES constant (OR=1), conditional logistic regression analysis showed the prevalence of URS and LRS increased remarkably in children from families belonging to low SES (Table 3.19, 3.20).

Table 3.19: Conditional logistic regression analysis for association between upper respiratory symptoms and socioeconomic status (SES)

SES	Sinusitis	Running nose	Sneezing	Common cold	Sore throat
High	1	1	1	1	1
Medium	1.26* (1.05-1.52)	1.82* (1.56-2.12)	1.65* (1.35-2.18)	2.06* (1.84-2.34)	1.44* (1.25-1.66)
Low	1.22* (1.07-1.65)	2.34* (2.01-2.73)	2.16* (1.52-2.80)	2.07* (1.82-2.34)	1.93* (1.67-2.23)

*Results are expressed as odds ratio with 95% confidence interval in parentheses; *, p<0.05*

Table 3.20: Conditional logistic regression analysis for association between lower respiratory symptoms and socioeconomic status (SES)

SES	Dry cough	Wet cough	Wheeze	Chest discomfort	Breathless-ness	Disturbed sleep
High	1	1	1	1	1	1
Medium	2.31* (1.96-2.73)	1.72* (1.48-2.01)	0.97 (0.79-1.21)	1.89* (1.55-2.30)	1.80* (1.52-2.13)	1.85* (1.44-2.24)
Low	3.34* (2.83-3.95)	2.20* (1.89-2.57)	1.24* (1.04-1.64)	2.43* (1.99-2.96)	2.09* (1.76-2.49)	2.05* (1.56-2.81)

*Results are expressed as odds ratio with 95% confidence interval in parentheses; *, p<0.05*

3.3.2 Prevalence of childhood asthma

(a) Asthma: more prevalent in Delhi

The prevalence of current asthma (dyspnea and wheeze at any time in the last twelve months), and physician-diagnosed asthma among the children of Delhi were 4.6% and 1.7 % respectively, which were significantly higher than the corresponding values in control group- 2.5% 1.1% respectively (p<0.001 in Chi-square test, Table 3.21).

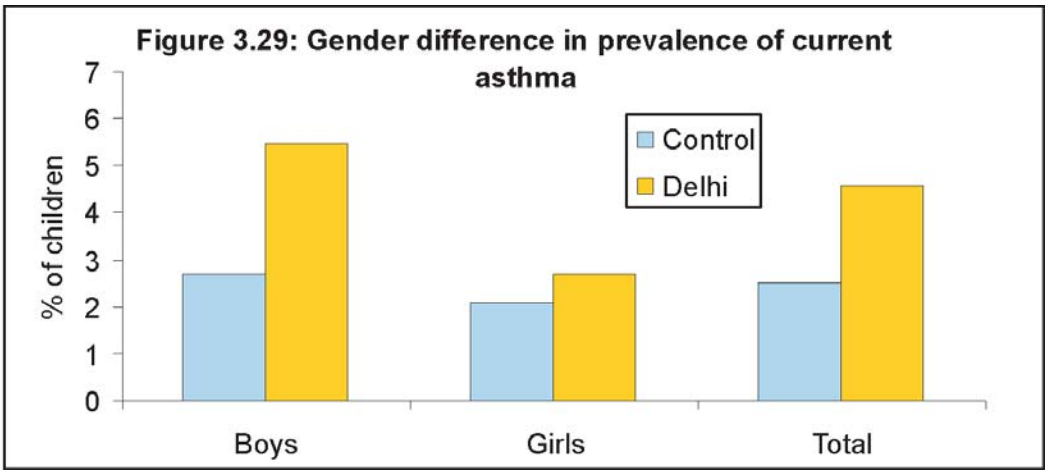
Table 3.21: Prevalence of bronchial asthma in rural and urban children

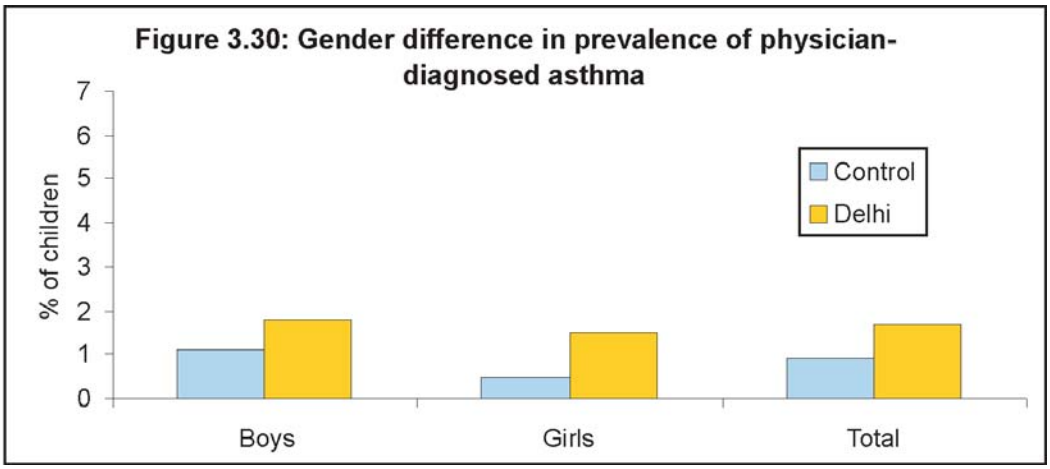
Type of asthma	Control(n=4536)	Delhi(n=11628)	p
Current asthma			
Boys	2.7	5.5	<0.001
Girls	2.1	2.7	>0.05
Total	2.5	4.6	<0.001
Physician-diagnosed asthma			
Boys	1.1	1.7	<0.05
Girls	0.5	1.5	<0.01
Total	0.9	1.7	<.001

Results are expressed as number of affected children with the percentage in parentheses

(b) Boys suffer more than the girls

Physician-diagnosed asthma and current asthma were more prevalent in boys than in girls both in rural and urban areas. The prevalence of asthma symptoms was 2-times more in boys than age-matched girls (5.5 vs.2.7%) in Delhi, and the gender difference was highly significant (p<0.001, Fig. 3.29). Delhi's boys also had greater prevalence of physician-diagnosed asthma than the girls (1.8 vs. 1.5%). Like the urban children, boys in the control group had greater prevalence of current asthma (2.7 vs. 2.1, p<0.05), and physician-diagnosed asthma (1.1 vs. 0.5%, p<0.001) than the girls (Fig. 3.30).





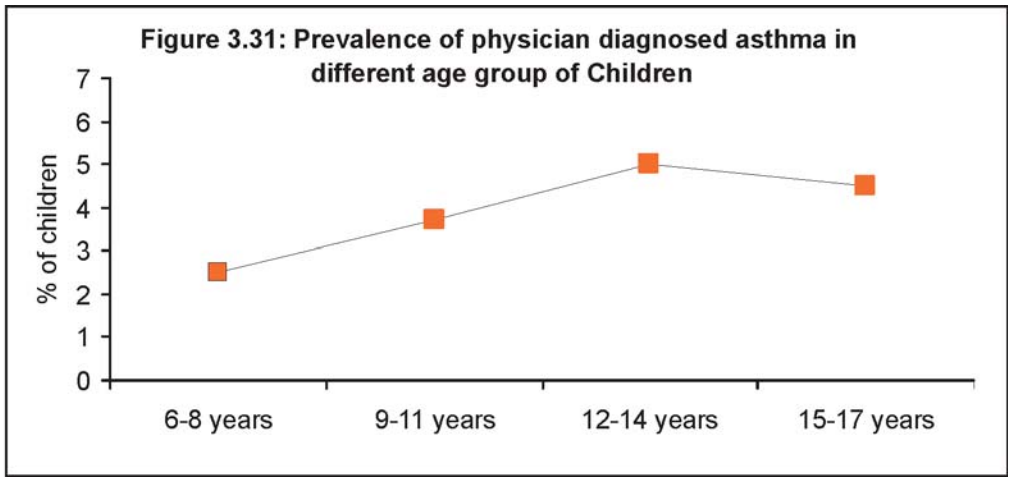
(c) Highest asthma prevalence in 12-14 year old children

Prevalence of current asthma in control children increased progressively up to the age of 14 years, and then a substantial fall in asthma prevalence was observed. A similar pattern was recorded in case of children of Delhi for current asthma, as well as for physician-diagnosed asthma. In essence, the prevalence of asthma is high in childhood, and then it declines substantially when the children enter their late teens and adolescence (Table 3.22; Fig. 3.31).

Table 3.22: Prevalence (%) of current asthma and physician-diagnosed asthma in relation to age

Age (yr)	Control		Delhi	
	Current asthma	Physician-diagnosed asthma	Current asthma	Physician-diagnosed asthma
6-8	2.0	2.0	2.5	1.3
9-11	2.5	1.1	3.7*	1.5
12-14	3.1	1.0	5.0*	1.9*
15-17	1.3	0.6	4.5*	1.4*

**, p<0.05 compared with respective control group in Chi-square test*



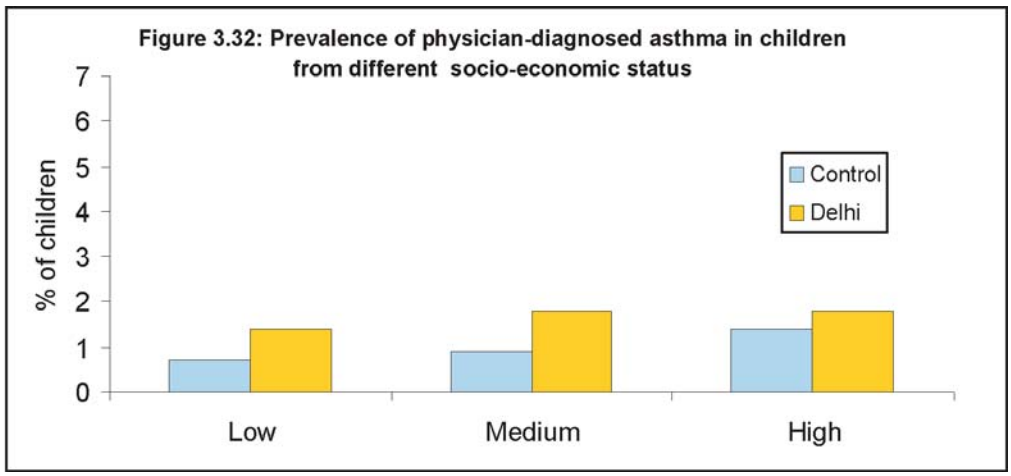
(d) Prevalence of asthma in children from different socio-economic background

Prevalence of asthma in different SES is presented in Table 3.23. In control group, asthma was slightly more prevalent in children from medium and high SES, especially in case of physician-diagnosed asthma (Fig. 3.31). In Delhi, children from lower and medium SES had a greater prevalence of asthma symptoms, while children from medium and high SES had higher prevalence of physician-diagnosed asthma (Fig. 3.32).

Table 3.23: Prevalence (%) of current asthma in different socio-economic status (SES)

Group	Low SES	Medium SES	High SES
Current asthma			
Control	2.4	2.6	2.5
Delhi	4.6*	4.8*	4.3*
Physician-diagnosed asthma			
Control	0.7	0.9	1.4
Delhi	1.4*	1.8*	1.8

**, p<0.05 compared with corresponding control group*



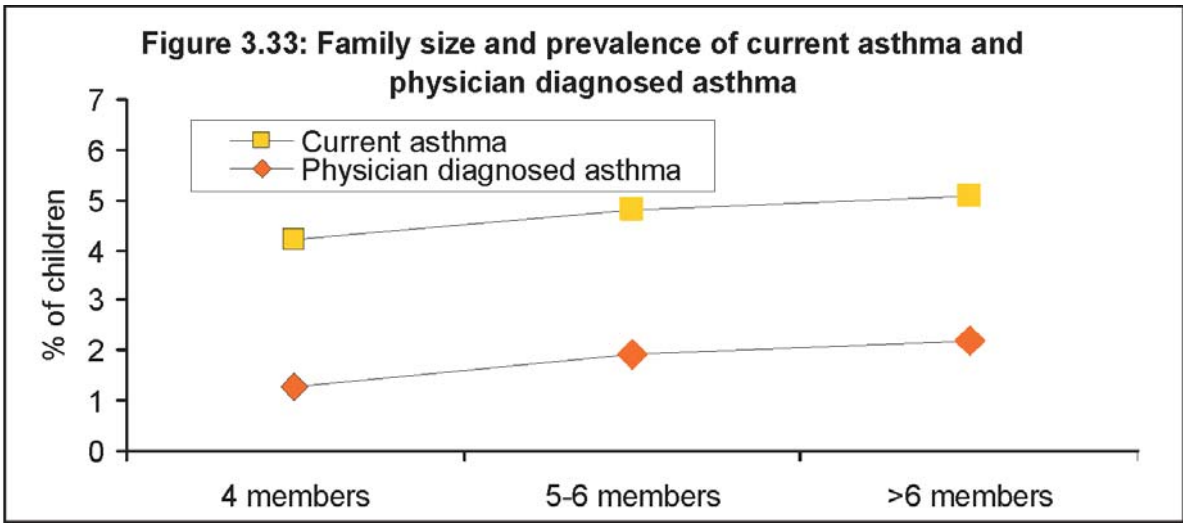
(e) Higher prevalence of asthma in large-sized families

Numbers of children from small (upto 4 members), medium (5-6 members) and large (>6 members) families were 5481, 4007, 2203 respectively in Delhi and 841, 1843 and 1852 in control. Asthma was found more prevalent in children from medium- and large-sized families particularly the latter, compared with children from small families. This was valid both for rural and urban settings (Table 3.24; Fig. 3.33). For example 2.2% from large families had physician-diagnosed asthma compared with 1.3% children from small families in Delhi, and the difference was statistically significant ($p<0.05$). Similarly 1.1% of control children from large families had physician-diagnosed asthma in contrast to 0.6% children from small sized families ($p<0.05$). In essence, large family size was more common among asthmatic children and the association was significant ($p<0.05$).

Table 3.24: Family size and asthma prevalence (%)

Members in family	Current asthma		Physician-diagnosed asthma	
	Control	Delhi	Control	Delhi
Up to 4	1.7	4.2*	0.6	1.3*
5-6	2.6	4.8*	0.9	1.9*
>6	2.8	5.1*	1.1	2.2*

**, p<0.05 compared with control in Chi-square test*



(f) Risk factors for asthma

There was a strong association between current asthma in children and parental smoking, and similar illness in father and / or other members of the family. History of bronchial asthma was present in families of 40.4% of asthmatic children in Delhi, and 46.4% in rural areas. Asthma attacks were most prevalent in children during winter (2.0%) when the air pollution level was highest. Controlling potential confounders, logistic regression analysis suggests positive association between particulate pollution and asthma attacks (OR=1.28; 95% CI 1.07-1.42), but not the prevalence of asthma (OR = 1.05, 95% CI 0.87-1.15, Fig. 3.34).

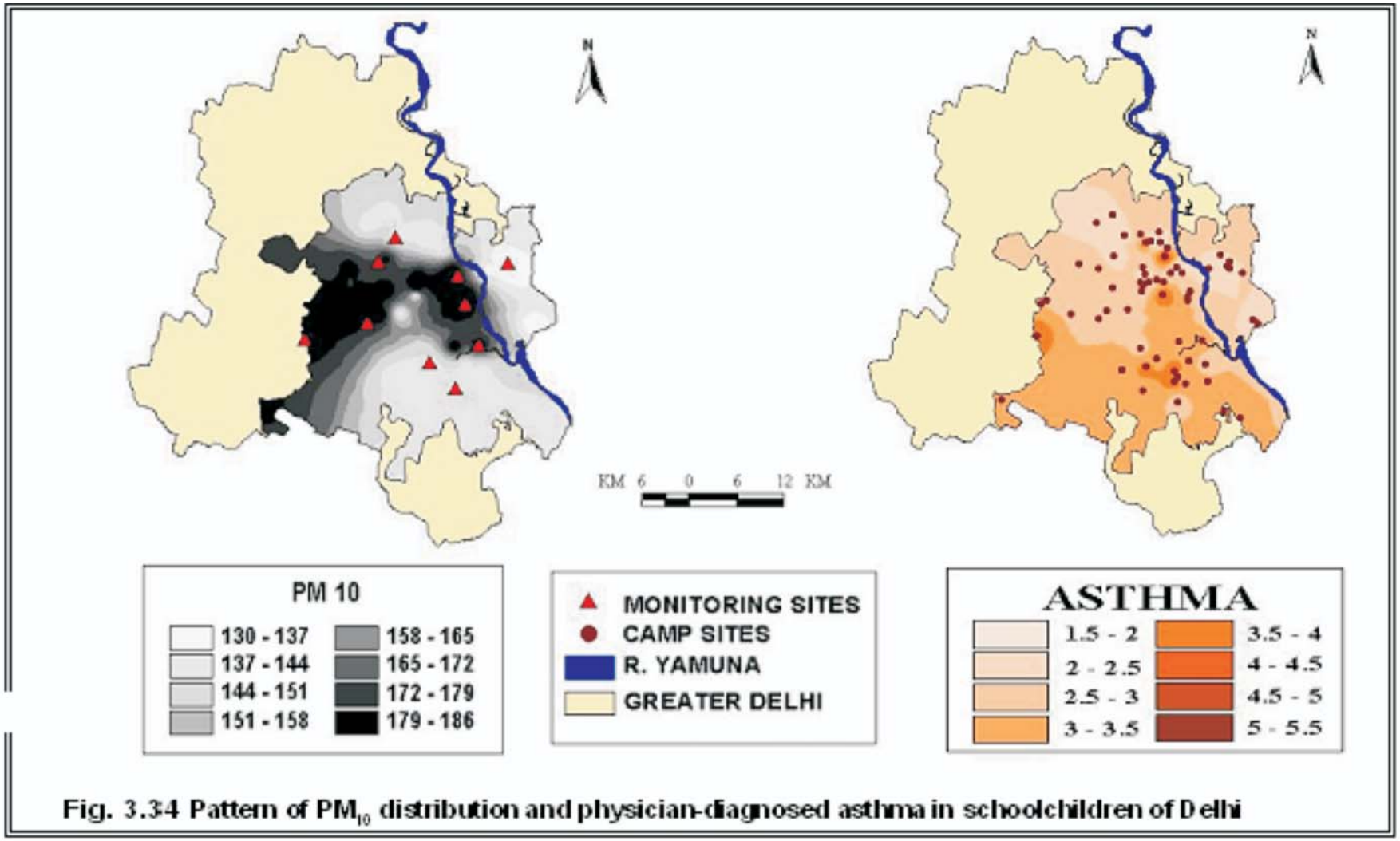


Fig. 3.34 Pattern of PM_{10} distribution and physician-diagnosed asthma in schoolchildren of Delhi

3.3.3 Prevalence of associated symptoms

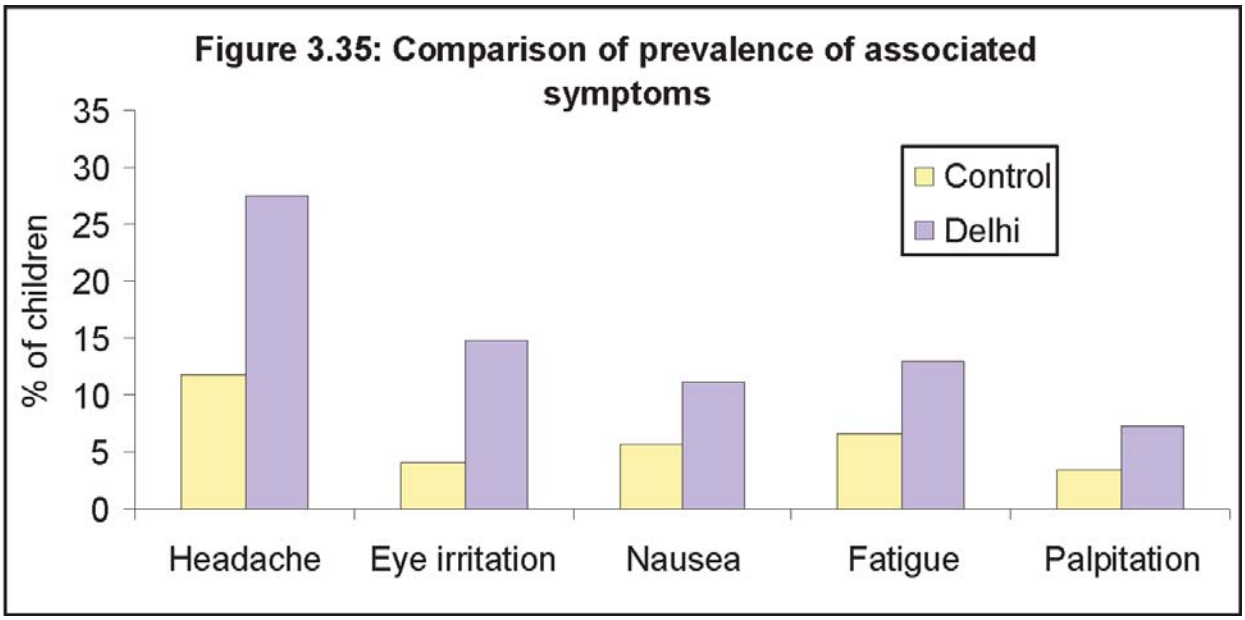
(a) Recurrent headache

Recurrent headache during past three months was reported by 27.4% children of Delhi in contrast to 11.8% children of control group. The difference between these two groups was highly significant ($p<0.001$, Table 3.25, Fig. 3.35). Headache was more prevalent in girls than in boys in control (19.0 vs. 7.9% in boys, $p<0.001$) as well as in Delhi (36.0 vs. 23.1%, $p<0.01$). Highest prevalence of headache was found in adolescents in the age group of 15-17 years (33.4%; Table 3.25, Fig. 3.36). Like respiratory symptoms, headache was most prevalent among children and adolescents with low SES (Fig. 3.37). The symptom among the children of Delhi was most prevalent during winter months (40.8%) followed by summer (29.6%) and monsoon (20.2%, Fig. 3.38).

Table 3.25: Prevalence (%) of respiratory-associated symptom in children

Symptom	Control	Delhi
Recurring headache	11.8	27.4*
Eye irritation	4.2	14.7*
Nausea	5.6	11.2*
Palpitation	3.3	7.2*
Fatigue	6.7	12.9*

**, p<0.05 compared with control in Chi-square test*



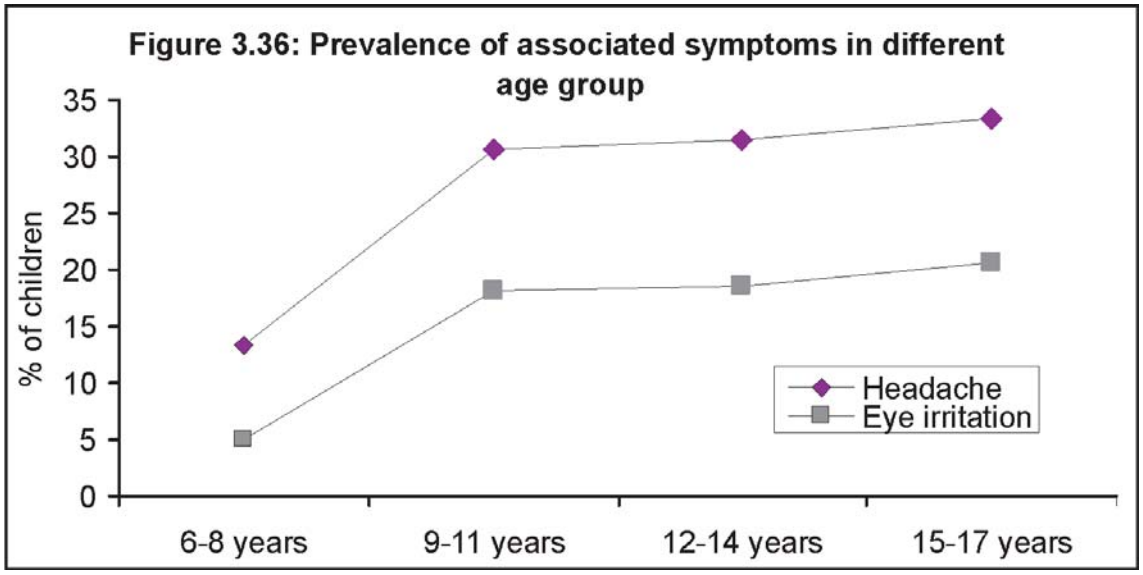


Figure 3.36: Prevalence of associated symptoms in different age group
% of children
35
30
25
20
15
10
5
0
6-8 years
9-11 years
12-14 years
15-17 years
Headache
Eye irritation

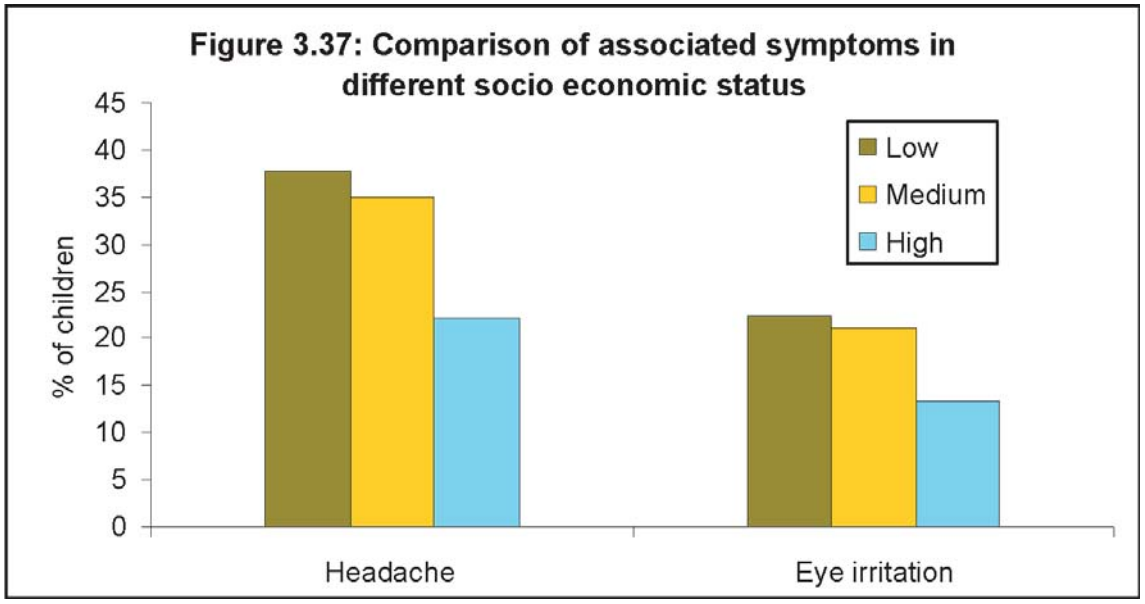


Figure 3.37: Comparison of associated symptoms in different socio economic status
% of children
45
40
35
30
25
20
15
10
5
0
Low
Medium
High
Headache
Eye irritation

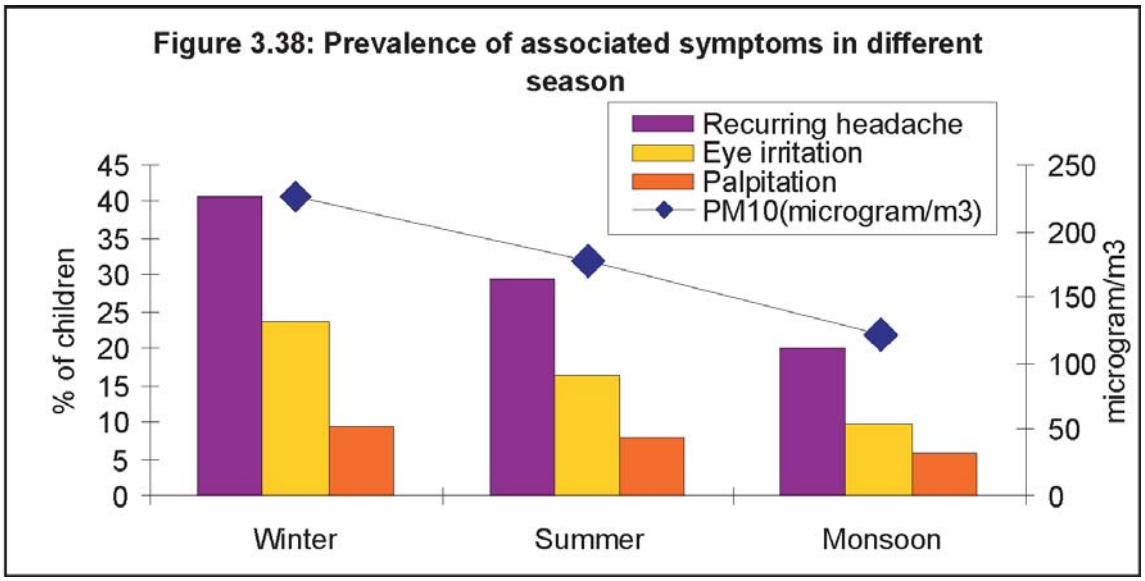


Figure 3.38: Prevalence of associated symptoms in different season
% of children
45
40
35
30
25
20
15
10
5
0
Recurring headache
Eye irritation
Palpitation
PM10(microgram/m3)
250
200
150
100
50
0
microgram/m3
Winter
Summer
Monsoon

(b) Eye irritation

Irritation and watering of the eyes were reported by 14.7% of the children of Delhi, compared with 4.2% of controls ($p<0.001$, Table 3.25, Fig. 3.35). Its prevalence in Delhi was marginally higher in girls (15.8% vs. 14.2% in boys), and the problem was more frequent during winter as 23.8% of examined children during November-February complained of eye irritation compared with 9.8% in monsoon (Fig. 3.38). Adolescents in the age group of 15-17 years (20.6%, Fig. 3.36) and those from North, Central and West Delhi showed higher prevalence of eye problem. Compared with children from high SES, the prevalence eye irritation was found doubled in children from low SES (Fig. 3.38).

(c) Nausea

Nausea, i.e. vomiting tendency, was present in nearly 11.2 % children of Delhi against 5.6% of controls (Table 3.25, Fig. 3.35). The prevalence was similar in girls and boys. Children suffered from this problem most during winter (15.1%), compared with 6.5% in summer. Its incidence was highest in children of the age group of 9 – 11 years (13.4%; Table 3.26, Fig. 3.36).

Table 3.26: Prevalence (%) of respiratory-associated symptom in different age group

Symptoms	6-8 years	9-11 years	12-14years	15-17years
Recurring headache	13.3	30.7	31.4	33.4
Eye irritation	5.1	18.2	18.5	20.6
Nausea	12.7	13.4	8.6	6.6
Palpitation	1.9	13.1	10.2	7.2
Fatigue	15.8	18.9	13.6	12.7

(d) Palpitation

Palpitation was prevalent among 8.0% of the boys and 5.6% of the girls of Delhi, with an overall prevalence of 7.2%. In contrast, 3.3% of control children had this problem (Table 3.25, Fig. 3.35). Its prevalence was highest in children of North (8.6%) and Central Delhi (8.5%) and lowest in children of South Delhi (4.5%). The prevalence of palpitation was greatest in winter (9.4 %; Table 3.27, Fig. 3.38) and in children of 9 – 11 year age group (13.1%; Table 3.26, Fig. 3.36).

Table 3.27: Prevalence (%) of respiratory-associated symptom in different season

Symptom	Summer	Monsoon	Winter
Recurrent headache	29.6	20.2	40.8
Eye irritation	16.3	9.8	23.8
Nausea	6.5	11.0	15.1
Palpitation	8.0	5.9	9.4
Fatigue	13.2	9.0	20.7

(e) Fatigue

About 12.9% of the examined children of Delhi reported of getting tired easily, of which 12.5% were girls and 13.2% were boys. Fatigue was found in 6.7% of control children ($p<0.001$, Table 3.25, Fig. 3.35). Its prevalence was highest during winter (20.7%) followed by summer (13.2%) and monsoon (9.0%; Table 3.26), and in children of the 9 – 11 year age group (18.9%; Table 3.27, Fig. 3.36). Children from the higher socio economic status suffered least from this problem as only 10.3% of children complained of getting tired easily as compared to 17.1% of children coming from the low socio economic status (Table 3.28; Fig. 3.37).

Table 3.28: Prevalence (%) of respiratory-associated symptom in different socio economic status (SES)

Symptom	Low SES	Medium SES	High SES
Recurring headache	37.8	34.9	22.0
Eye irritation	22.4	21.0	13.2
Nausea	8.2	11.1	6.5
Palpitation	11.7	11.2	5.8
Fatigue	17.1	15.3	10.3

3.3.4 Association between ambient air pollution (PM_{10} level) and prevalence of respiratory symptoms

(a) Weak positive association between PM_{10} level in breathing air and prevalence of URS

In univariate analysis the prevalence of upper respiratory symptoms excepting sinusitis were significantly elevated ($p<0.05$) in Delhi's children when the potential confounders like socio economic conditions, parental smoking, age and gender were controlled in multivariate logistic regression analysis, only sore throat (OR=1.25, 95% CI 1.03-1.52, $p<0.05$, Table 3.29) and common cold with fever (OR=1.35, 95% CI 1.12 – 1.65, $p<0.05$, Table 3.30) were found to be positively associated with particulate pollution (PM_{10}). PM_{10} level was also found to have a significant positive association with URS prevalence in general (OR=1.24, 95% CI 1.02-1.47). The association between PM_{10} and URS was further strengthened by conditional logistic analysis. The association was weakly positive and non-significant upto a PM_{10} level of 125 $\mu m/m^3$. A strong positive association between PM_{10} and sore throat and common cold was found thereafter. All symptoms except sinusitis positively correlated with PM_{10} when the ambient level reaches above 150 $\mu m/m^3$ (Table 3.29).

Table 3.29: Conditional logistic regression analysis of the association between particulate air pollution and upper respiratory symptoms

PM_{10} level(μg/m³)	Sinusitis	Running nose	Sneezing	Common cold	Sore throat
50-75	1	1	1	1	1
76-100	1.03 (0.75-1.43)	0.43 (0.31-0.61)	1.03 (0.81-1.21)	0.34 (0.26-0.44)	1.02 (0.57-1.26)
101-125	1.28 (0.94-1.60)	1.06 (0.81-1.20)	1.06 (0.78-1.33)	1.01 (0.88-1.16)	1.22 (0.69-1.43)
126-150	1.15 (0.91-1.45)	1.12 (0.83-1.20)	1.33 (0.87-1.53)	1.23 (1.07-1.43)*	1.33 (1.03-1.62)*
>150	1.09 (0.87-1.37)	2.60* (2.19-3.07)	2.60 (2.19-3.07)*	2.09 (1.82-2.40)*	1.74 (1.48-2.05)*

*Results are presented as odds ratio with 95% confidence interval in parentheses; *, p<0.05*

Table 3.30: Logistic regression analysis of the association between particulate air pollution (PM_{10}) and URS after adjustment for potential confounders.

Symptom	Control(n=4536)	Delhi(n=11628)	Odds Ratio (95% CI)
Sinusitis	3.2	3.4	1.06 (0.87 – 1.34)
Running or stuffy nose	5.8	9.6	1.22 (0.95 – 1.49)
Sore throat	4.9	7.4	1.25 (1.03 – 1.52)*
Sneezing	5.2	9.1	1.09 (0.86-1.22)
Common cold and fever	5.7	10.5	1.35 (1.12 – 1.65)*
Total URS	**14.6**	**23.1**	1.24 (1.02-1.47)*

*The results are expressed as percentage of affected children; *p<0.05*

(b) Strong positive association between PM_{10} level in breathing air and prevalence of LRS in children

Unlike URS, a strong, statistically significant positive association was observed between PM_{10} level in Delhi's air and the prevalence of six symptoms under LRS in children in past three months. Taking the prevalence of individual symptoms during low pollution level (PM_{10} 50-70μg/m³) as constant (OR=1) in conditional regression analysis (Table 3.31), prevalence of LRS symptoms was found doubled or tripled when particulate pollution level was elevated (PM_{10} >125 μg/m³).

Logistic regression analysis after controlling potential confounders like parental smoking and SES particulate pollution was found to be positively associated with all six symptoms of LRS (Table 3.32). The association was strongest for sleep disturbance due to breathing trouble and dry cough, followed by chest pain or discomfort, breathlessness on exertion, cough with sputum production, and wheeze. In essence, the prevalence of LRS increases with increasing concentration of particulate pollution in breathing air.

Table 3.31: Conditional logistic regression analysis of the association between chronic exposure to PM_{10} and lower respiratory symptoms

PM_{10} ($\mu g/m^3$)	Dry cough	Wet cough	Wheeze	Breathless-ness	Chest discomfort	Sleep disturbance
50-75	1	1	1	1	1	1
76-100	1.22* (1.04-1.47)	1.06 (0.84-1.29)	0.97 (0.76-1.14)	1.12* (1.02-1.44)	1.13* (1.02-1.34)	1.26* (1.10-1.48)
101-125	1.86* (1.54-2.24)	1.29* (1.06-1.57)	1.04 (0.72-1.38)	1.34* (1.14-1.83)	1.66* (1.27-2.14)	1.64* (1.25-2.13)
126-150	2.20* (1.81-2.68)	1.30* (1.09-1.56)	1.43* (1.12-1.77)	1.51* (1.22-1.78)	1.94* (1.53-2.44)	1.78* (1.33-2.27)
>150	3.12* (2.36-3.75)	3.03* (2.53-3.62)	1.67* (1.19-2.36)	2.84* (2.31-3.47)	2.65* (2.09-3.37)	2.73* (1.89-4.32)

*Results are presented as odds ratio with 95% confidence interval in parentheses; *, p<0.05*

Table 3.32: Logistic regression analysis of association between PM_{10} and lower respiratory symptoms in children

LRS	Control	Delhi	OR (95% CI)
Dry cough	3.3	6.6	1.48 (1.24 – 1.67)*
Cough with phlegm	3.8	7.0	1.33 (1.12 – 1.56)*
Wheeze	2.7	4.8	1.23 (1.04 – 1.45)*
Breathlessness on exertion	2.9	5.9	1.37 (1.15 – 1.63)*
Chest discomfort	1.2	3.2	1.44 (1.18 – 1.76)*
Sleep disturbance due to breathing problem	0.7	2.2	1.68(1.35– 1.96)*
LRS, total	**8.0**	**17.0**	**1.42 (1.19 – 1.83)***

*The results are expressed as percentage of children with symptom; *p<0.05*

(c) Strong positive association between PM_{10} and eye irritation, but not with asthma or headache

Conditional and multivariate logistic regression analyses showed a weak association between PM_{10} level in Delhi's air and the prevalence of medically diagnosed asthma and headache in city's children. However, a strong, statistically significant ($p<0.05$) positive association was found between PM_{10} level and eye irritation (Table 3.33). Nausea, palpitation and fatigue were also found to be significantly associated with particulate air pollution exposure (Table 3.34).

Table 3.33: Conditional logistic regression analysis for medically-diagnosed asthma, headache and eye irritation

PM_{10} level(μg/m^3)	Medically- diagnosed asthma	Headache	Eye irritation
50-75	1	1	1
76-100	0.31 (0.09-1.04)	0.59 (0.47-0.73)	1.18* (1.05-1.32)
101-125	1.41 (0.88-2.27)	1.19 (1.05-1.35)	1.27* (1.12-1.56)
126-150	1.22 (0.72-2.07)	1.11 (0.96-1.27)	1.30* (1.11-1.51)
>150	1.55* (1.05-2.56)	1.89* (1.66-2.16)	2.35* (1.54-2.82)

*Results are expressed as odds ratio with 95% CI in parenthesis; * p<0.05*

Table 3.34: Logistic regression analysis of association between air pollution PM_{10} and asthma and other symptoms

	Control	Delhi	OR (95% CI)
Medically-diagnosed asthma	0.9	1.7	1.08 (0.87-1.42)
Recurring headache	11.8	27.4	1.22 (0.98-1.65)
Eye irritation	4.2	14.7	2.45 (1.56-3.74)*
Nausea	5.6	11.2	1.44 (1.12-2.25)*
Palpitation	3.3	7.2	1.20 (1.07-1.73)*
Fatigue	6.7	12.9	1.21 (1.07-1.53)*

*The results are expressed as percentage of affected children; *p<0.05*

3.4 FINDINGS/

(a) The prevalence of respiratory and associated symptoms was investigated in 11,628 children from 36 schools in Delhi and 4536 control children from two schools in Uttaranchal and 15 from rural West Bengal. The children were aged between 4 and 17 years, and 55% of them were 12-14 year old. Two-third of the children was boys, and one-third was girls. Respiratory symptom data were collected through specially designed structured questionnaire based on three validated questionnaires of BMRC, ATS-DLD-78-C and IUATALD.

(b) Upper respiratory symptoms (URS) like sinusitis, running or stuffy nose, sneezing, sore throat and common cold with fever were 1.8-times more (23.1% vs. 14.6%) prevalent in Delhi than in controls, and the girls suffered more than the boys.

(c) Children in Delhi had 2-times more (17 % vs. 8%) lower respiratory symptoms (LRS) such as frequent dry cough, sputum-producing cough, wheezing breath, breathlessness on exertion, chest pain or tightness and disturbed sleep due to breathing problems. Thus, compared with

control, Delhi's children had 1.8- times more URS and 2-times more LRS suggesting higher prevalence of underlying respiratory diseases.

(d) Prevalence of current asthma was present in 4.6% children of Delhi against 2.5% of controls. Similarly, the instance of physician-diagnosed asthma was 2 times more in Delhi (1.7 vs. 0.9%). The difference in asthma prevalence between control and Delhi's school children was significant ($p<0.001$).

(e) About 15% of Delhi's children had frequent eye irritation compared with only 4% in controls. Similarly, Delhi's children had significantly higher prevalence of frequent headache (27.4 vs. 11.8%), nausea (11.2 vs. 5.6%), and palpitation (7.2 vs. 3.3%) and fatigue (12.9 v. 6.7%).

(f) Respiratory and associated symptoms were most prevalent in children from low socio-economic status, and least in children from families with high socio-economic background.

(g) The symptoms were more prevalent in children during winter when PM_{10} level in air is highest in a year, and lowest during monsoon when particulate air pollution level is lowest, suggesting a positive association with particulate air pollution. Conditional and multivariate logistic regression analysis confirmed the association between exposure to particulate air pollution and the prevalence of respiratory and associated symptoms in school children, excepting sinusitis, running nose, sneezing, asthma and headache.

CHAPTER-4.0

EFFECT OF DELHI'S AIR POLLUTION ON CHILDREN'S LUNG FUNCTION

4.1 INTRODUCTION

Since lung is the primary target organ for the health effects of air pollution, respiratory health is commonly assessed by measurements of mechanics of breathing referred to collectively as lung function. Pulmonary function test is one of the basic tools for evaluating a person's respiratory status.

Rapid lung growth begins *in utero* and continues until the late teens in girls and early 20s in boys. Lung function reaches a maximum by 18-20 years of age in females and 22-25 years in males (Tager et al., 1988). Some males may show a small increment in lung function into their mid –20's. Lung function varies widely among adults. The big difference in lung function in adults are due to attained lung function at maturity, which can differ by a factor of two for individuals of the same age, sex, height, weight and race (Dockery et al., 2005). Thus, factors that can affect growth of lung function in childhood are important in determining the level of lung function in adulthood.

Several factors can be responsible for disruption of normal lung development and growth, leading to reduced lung function. These include intrauterine growth retardation, viral infections, premature birth, inflammatory conditions, genetic changes and environmental toxicants. A child inhales large volumes of air daily, and in polluted environments substantial amounts of air toxics are deposited into the airways and air exchange surfaces. This may impair the lung defense. If lung defense is compromised, normal developmental and homeostatic process can be disrupted, leading to disturbances in lung development, organ damage, and consequent chronic reduction in lung function (Dockery et al., 2005).

Lung function of a child at any age is the result of cumulative lung growth up to that age. It is generally believed that children with highest lung function had the highest lung growth (Dockery et al., 2005). Exposure to environmental toxicants, such as ambient air pollutants, can handicap a child prenatally (before birth), because these pollutants can impair lung development pre- as well as postnatally. Even maternal smoking during pregnancy reduces lung development of the fetus *in utero* (Tager et al., 1995). Reduced prenatal or postnatal growth rates prevents lung from reaching their optimum developmental potential. Thus, exposure to air pollution even when the baby is in mother's womb may affect the development of the lungs, which are the last organs to develop and are not fully developed at birth.

A large number of pollutants have the potential to impair growth of the lung, and impairment of lung function. In this context, time of exposure is most critical, because susceptibility is highest during the fetal and childhood periods, than in adulthood when lung activity is declining. Abient air pollution in California increase the risk of low birth weight and preterm birth (Ritz and Fu, 1999). Premature birth can result in severe lung damage in neonatal periods.

A summary of the literature on epidemiological studies of air pollution and children's lung function (Dockery et al. 2005) reveals that: i. living in areas of high air pollution is associated with lower lung function, ii. chronic exposure to elevated level of air pollution is associated with lower rates of

lung function growth, iii. improvement in air quality leads to improvement in lung function level and/or growth rate, and iv. children who spend a significant amount of time outdoors in polluted environments or those with poor nutrition may be more strongly affected by air pollution.

Lung function is assessed using a broad array of tests that measure lung volume, airflow and gas diffusion. The most convenient test procedure is spirometry that measures how well the lung functions in exhaling air. Maximal forced expiratory volume maneuvers and spirometers are used to assess forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), maximum mid-expiratory flow (MMEF), and peak expiratory flow rate (PEFR). The information gathered using spirometry is useful in assessing airway obstruction and functional lung capacity. Poor cooperation limits the use of these tests in children less than five years of age.

In view of these reports, is necessary to examine the level of lung function and its growth rate among the school children of Delhi who were chronically exposed to elevated levels of air pollution relative to control children.

4.2 MATERIALS AND METHODS

(a) Subjects

Out of the 11,628 children enrolled for this study in Delhi, pulmonary function test (PFT) was carried out in 5718 children, of which 3730 (65.2%) were boys and 1988 ((34.8%) were girls. In control, 2270 children participated for PFT by spirometry: 1449 (63.8%) were boys and 821 (36.2%) were girls. The age of the children who participated in PFT in Delhi and in control group ranged between 9 and 17 years, with a median of 13.5 years. Altogether, PFT was done on a total number of 7988 children (5179 boys and 2809 girls).

(b) Pulmonary function test (PFT) procedure

PFT was carried out following the guideline of American Thoracic Society (ATS, 1995) using a portable, electronic spirometer (Spirovit SP-1, Schiller, Switzerland) with disposable filters (SP-150), designed for ambulatory pulmonary function measurements. The device measures actual respiratory flow at a precision of 2%, in addition to predicted values according to age, sex, height, weight and race.

Trained research scholars with the permission of school authority did lung function tests at the school premises usually in the medical room. Before performing the PFT, the height and weight of the child was measured with shoes removed (Fig. 4.1). Each child performed forced expiratory maneuvers while sitting with free mobility and nose closed with a nose clip to prevent passage of air through the nose (Fig. 4.2, 4.3). Each spirometric test was repeated 3 times to allow the choice of the best values, and 2 values of FEV_1 should not differ by more than 5% according to the ATS criteria. Using a computer assisted quantitative assessment the best maneuver for acceptance was determined.

Figure 4.1: Students lined up with shoes removed for height and weight measurement at Sukho Khalsa Senior Secondary School, Janak Puri, New Delhi (a) and Kendriya Vidyalaya, East Arjun Nagar, New Delhi (b)

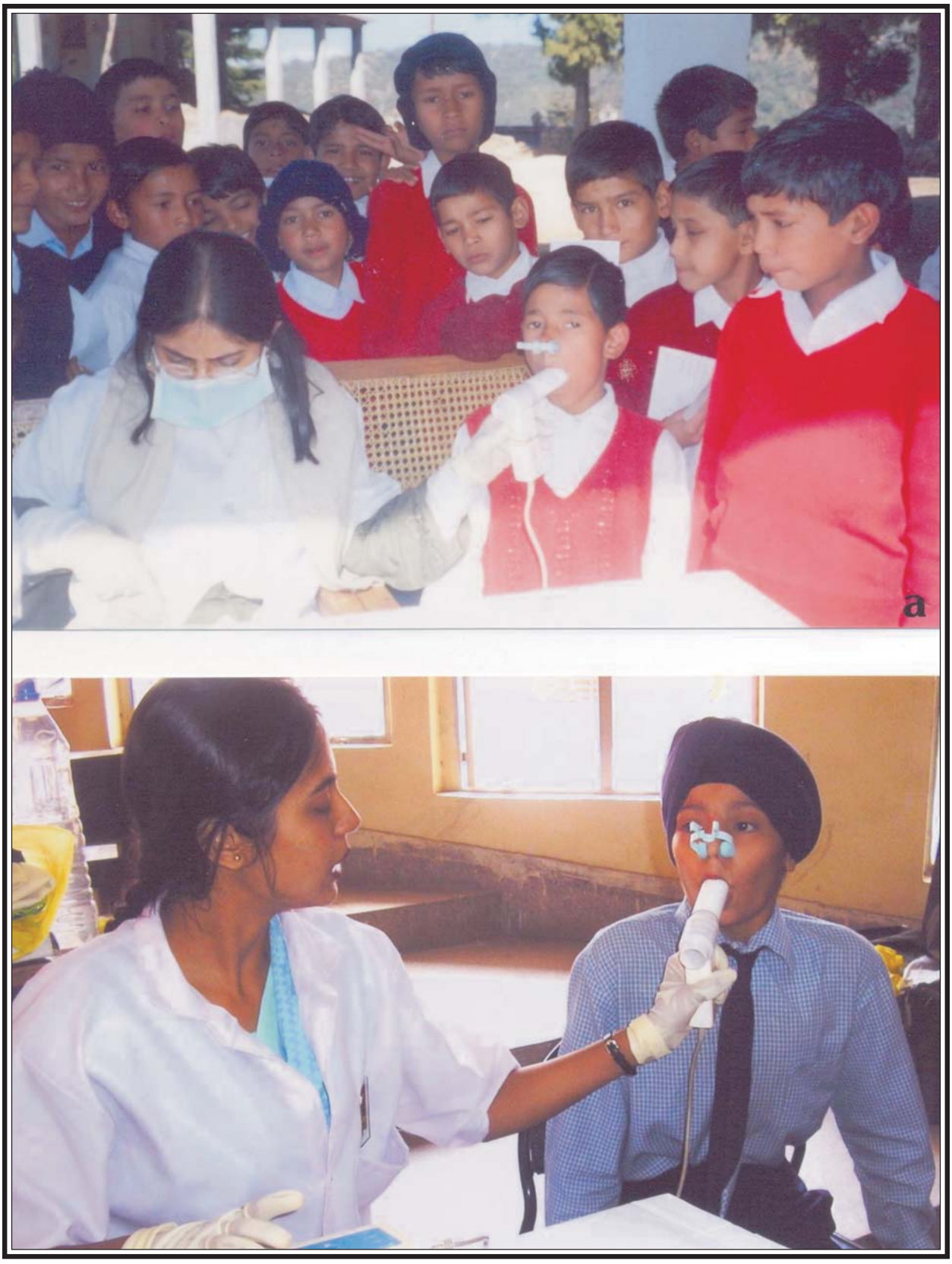


Figure 4.2: Pulmonary function test of students at Government Inter College, Khirsu, Uttaranchal (a) and Sukho Khalsa Senior Secondary School, Janak Puri, New Delhi (b)

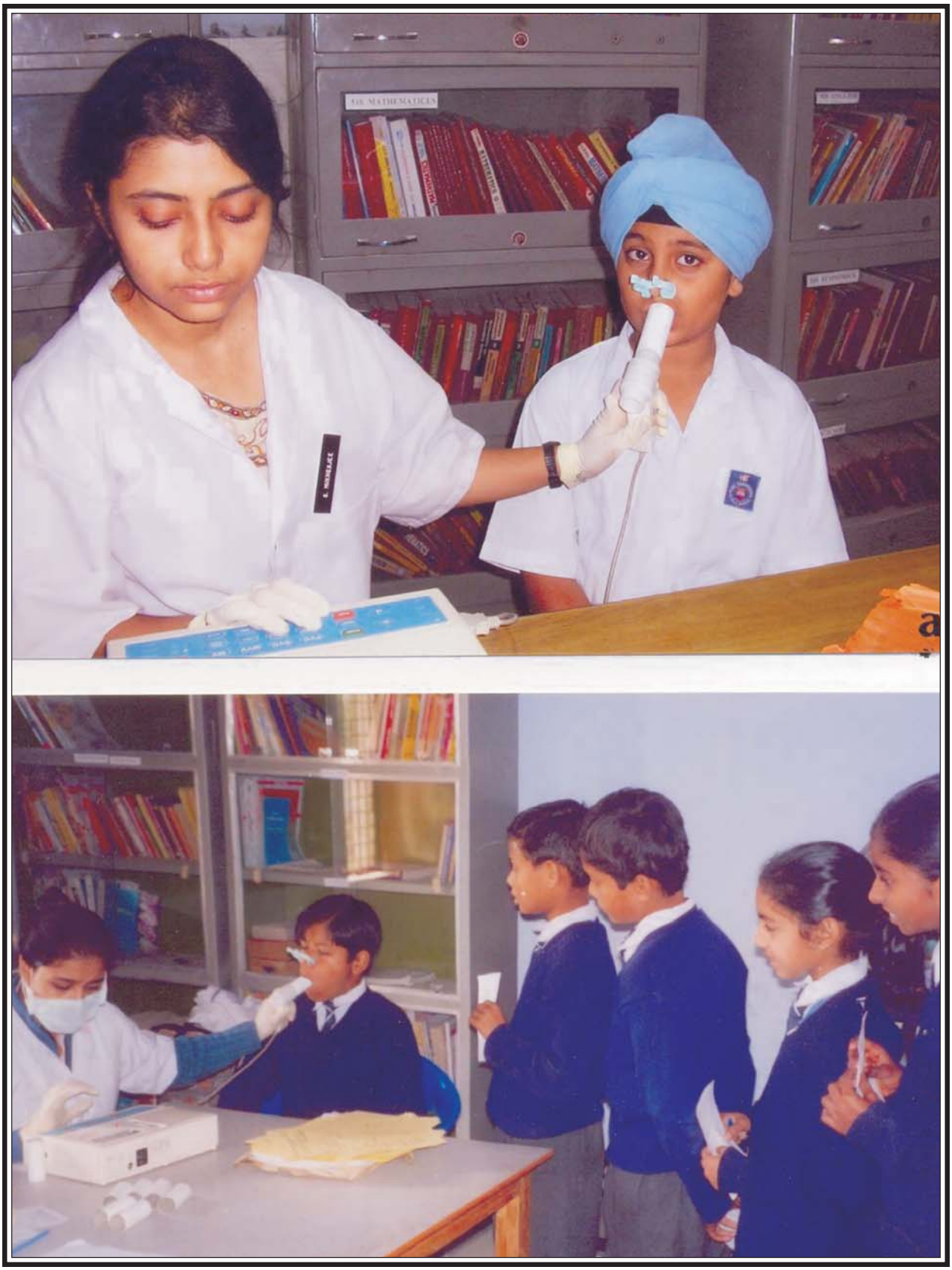


Figure 4.3: Lung function test of a student of (a) New Delhi's Guru Harkrishan Public School at Karol Bagh and (b) Motherland Academy, Kotdwar, Uttaranchal

The data were compared with predictive values based on age, sex, height and ethnic group. Flow was plotted against volume to display a continuous loop from inspiration to expiration, as the overall shape of the flow volume loop is important for interpreting spirometric results. The following spirometric parameters (absolute and relative values such as ratio of actual and predicted values) were recorded for analysis:

- Forced vital capacity (FVC), i.e. the volume of air in liters that can be maximally forcefully exhaled
- Forced expiratory volume at 1 second (FEV_1), i.e. volume of air (in liter) that is forcefully exhaled in one second
- Ratio of FEV_1 to FVC (FEV_1/FVC), expressed as percentage
- Forced expiratory flow at 25-75% ($FEF_{25-75\%)}$ or maximal mid-expiratory flow rate (MMFR), which is the average expiration flow rate during the middle 50% of the FVC.
- Peak expiratory flow rate (PEFR) – the peak flow rate during expiration

(i) Diagnosis of functional impairment of the lung

Decrement of lung function detected by spirometry could be generally of two types: Obstructive type and Restrictive types of impairment. In some cases combined (both obstructive and restrictive) type of lung function impairment could be encountered.

Obstructive type of lung function impairment

In obstructive type of lung function deficits such as emphysema or chronic bronchitis the FEV_1 is reduced disproportionately more than the FVC, resulting in an FEV_1/FVC ratio less than 70%. Thus, FEV_1/FVC <70% diagnoses airway obstruction. Subjects with obstructive lung had a rapid peak expiratory flow but the curve descends more quickly than normal and takes on a concave shape, reflected by a marked decrease in the $FEF_{25-75\%.}$ With more severe obstruction, the peak becomes sharper and the expiratory flow rate drops precipitously.

Restrictive type of lung function impairment

In restrictive lung type of lung function decrement, the FVC is reduced below 80% of predicted value. The shape of the flow volume loop is relatively unaffected in restrictive disease, but the overall size of the curve appears smaller when compared to normal on the same scale.

Combined type of lung function impairment

In combined type of lung function impairment, both FVC and FEV_1/FVC ratio are appreciably decreased. Subjects having this problem had FVC less than 80% of predicted value and FEV_1/FVC ratio < 70%.

(ii) Age for spirometry

It has been reported that schoolchildren who are 9 years of age can perform forced expiratory spirometric maneuvers well enough to meet currently established adult-based maneuver acceptability criteria (Enright et al. 2000). Accordingly, children aged 9 years and above were enrolled for PFT.

(iii) Calibration and quality control

In epidemiological studies in which spirometry results are primary outcome measurements, the results depend not only on the true lung function of the subject population, but also on quality of their test performance. Factors related to age, gender, size, ethnicity, and subject-technician affinity can influence the performance. But their overall effect is small with well-trained, experienced technicians/researchers (Enright et al., 2000). The spirometers were calibrated using a 2.0 liter syringe in each morning before the tests. The instrument was calibrated again after measurements in 50 children.

(c) Calculation of body mass index (BMI)

BMI is calculated by dividing the body weight in kilogram by the square of the standing height in meter. The value is expressed a kg/m^2. It is a reliable indicator of body fatness for most children. Although BMI does not measure body fat directly, but it correlates to body fat (Mei et.al, 2002). Moreover, BMI is an inexpensive and easy to perform method of screening for weight categories that may lead to health problems.

Although the BMI number is calculated the same way for children and adults, the criteria used to interpret the meaning of the BMI number for children are different from those used for adults. For children, BMI age- and sex-specific percentiles are used, because the amount of body fat changes with age, and the amount of body fat differs between girls and boys of the same age. Separate BMI- for- age growth charts for girls or boys formulated by the Center for Disease Control (CDC) take into account these differences and allow translation of a BMI number into a percentile for a child's sex and age (Fig. 4.4, 4.5).

After BMI calculation, the value of each child was plotted on to obtain a percentile ranking. The percentile indicates the relative position of the child's BMI number among children of the same sex and age. The body weight status of individual child was ascertained as belonging to either of the four categories 'underweight', 'healthy', 'at risk of overweight', or 'overweight' from the BMI percentile by the following procedure (Table 4.1):

Table 4.1: BMI calculation of children

Weight status category	BMI Percentile
Underweight	< 5th percentile
Healthy weight	5th to 85th percentile
At risk of overweight	85th to < 95th percentile
Overweight	≥95th percentile

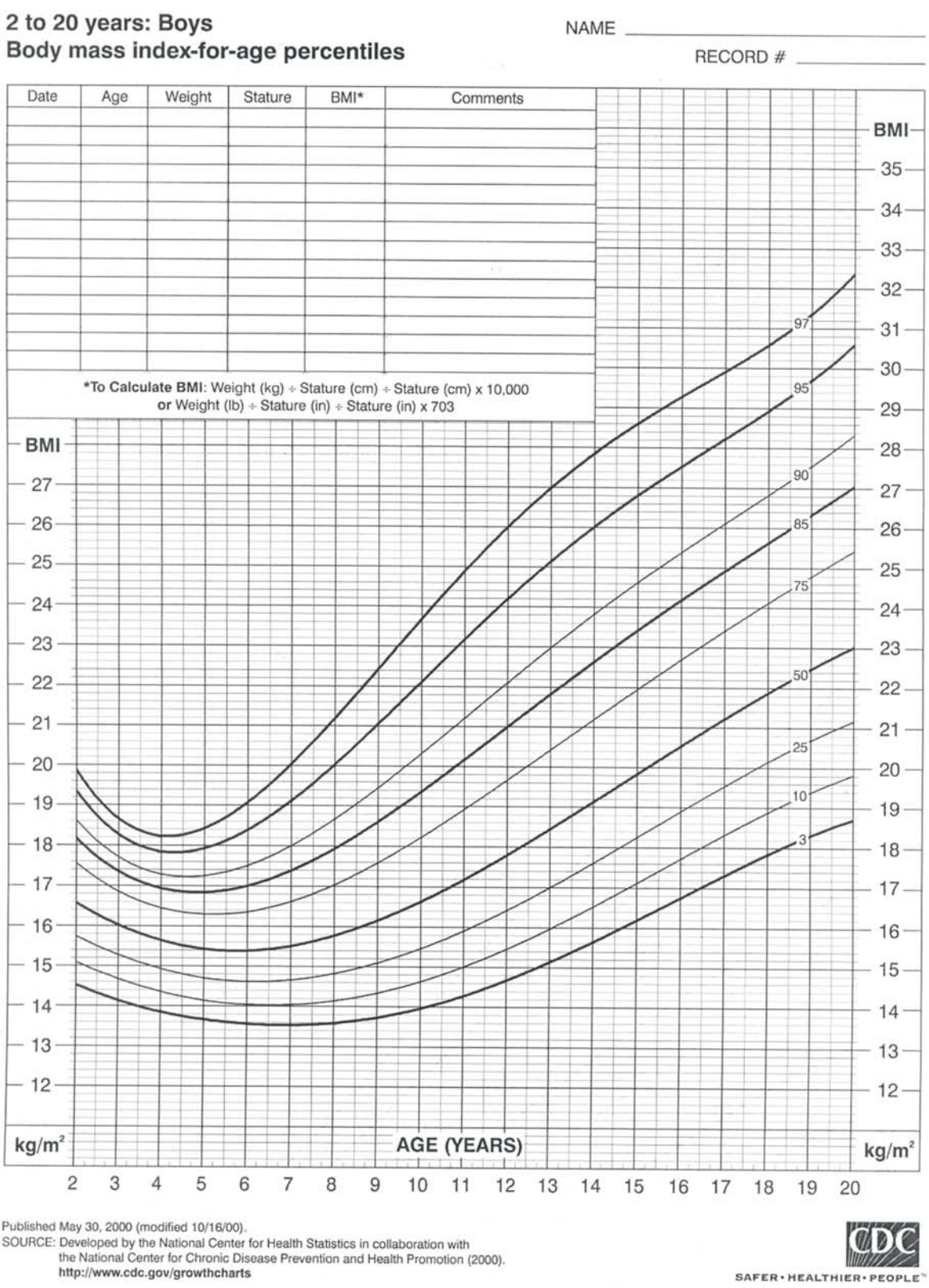


Figure 4.4: BMI- for- age growth charts for boys formulated by the Center for Disease Control (CDC)

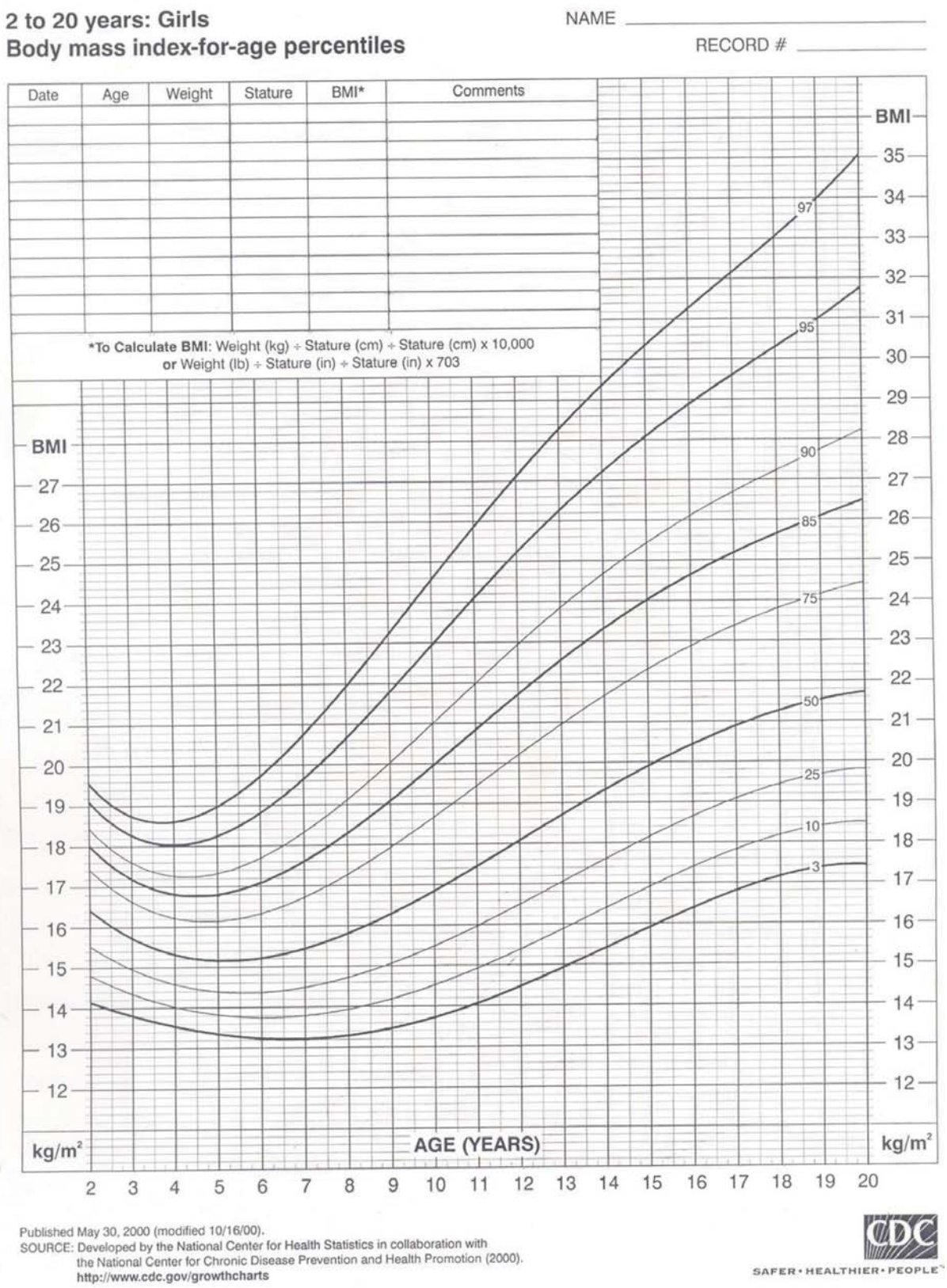


Figure 4.5: BMI- for- age growth charts for girls formulated by the Center for Disease Control (CDC)

(d) Statistical analysis

Bilateral statistical tests were used as follows: the Chi-square test for dichotomous or multinomial qualitative variables, the Mann Whitney U-test for quantitative variables with non-homogeneous variances or non-normal distribution and the Student's t-test for quantitative variables of normal distribution and homogeneous variances. A descendant stepwise logistic regression adjusted over potential confounding variables was carried out for multivariate analysis.

The collected data were processed and analyzed in EPI info 6.0 and SPSS (Statistical Package for Social Sciences) software. Logistic regression analysis using generalized estimating equations (GEEs) was used to examine the relationship between respiratory symptoms and RSPM levels.

4.3 RESULTS

(a) Successful pulmonary function tests (PFT): 5671 in Delhi and 2245 in controls

Forty-five children of Delhi (0.8%), 22 boys and 25 girls, failed to do PFT satisfactorily after repeated attempts (Table 4.2). In the control group 25 participants (1.1%), 11 boys and 14 girls failed to do the tests. Hence, these 70 children were excluded from the study. Therefore, lung function was successfully measured in 7916 children-5671 in Delhi and 2245 in control group. Out of these 5671 children of Delhi who successfully performed pulmonary function test, 3708 (65.4%) were boys and 1963 (34.6%) were girls. In the control group of 2245 successful performers, 1438 (64.1%) were boys and 807 (35.9%) were girls (Table 4.2).

Table 4.2: Participants in pulmonary function test (PFT) by spirometry

PFT	Boys	Girls	Total
Number of participants	5179	2809	7988
Delhi	3730	1988	5718
Control	1449	821	2270
Unsuccessful performers	33	39	70
Delhi	22	25	47
Control	11	14	25
Successful performers	5146	2770	7916
Delhi	3708	1963	5671
Control	1438	807	2245

(b) Age of the examined children

Age distribution of successful performers of spirometry in Delhi showed that 28.6% children belonged to 9-11 year of age, 38.7% aged 12-14 years and 32.7 % children were 15-17-year old. In control, 23.5% children were 9-11 years of age, 48.8% belonged to 12-14 year age group and 27.7% children were 15-17 years of age (Table 4.3).

Table 4.3: Age distribution of children whose lung function was measured by spirometry

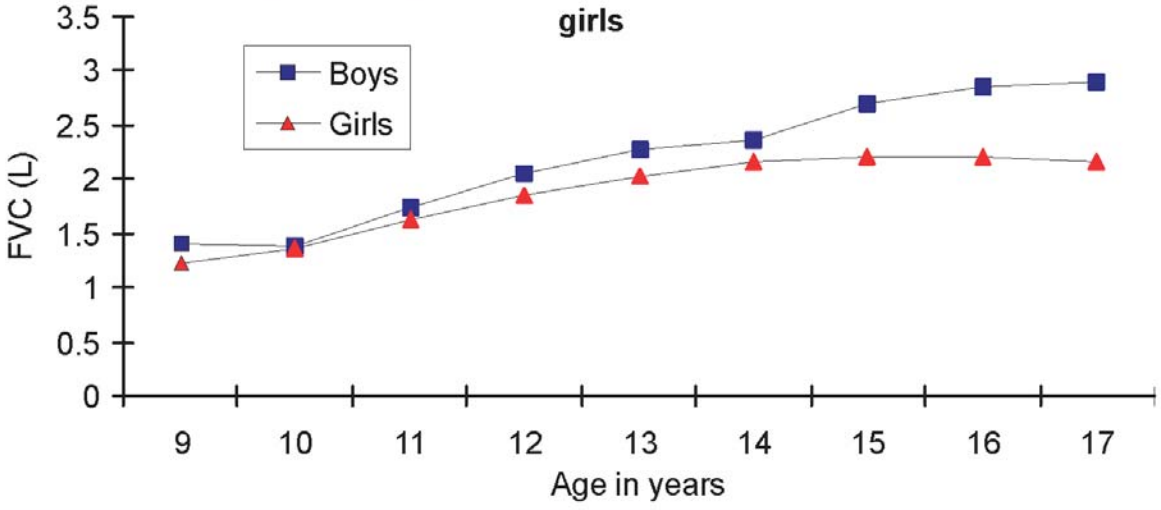
Age	Control(n=2245)	Delhi(n=5671)
9-11 year	527 (23.5)	1623 (28.6)
12-14 year	1095 (48.8)	2195 (38.7)
15-17 year	623 (27.7)	1853 (32.7)

The results are expressed as number of children in each age group with percentage in parentheses

4.3.1. Changes in forced vital capacity (FVC) with age in control and Delhi's children

The mean FVC value in boys aged 9 years was 1.41 liters in Delhi and 1.68 liters in control group. Girls of similar age in control and in Delhi had FVC values of 1.32 and 1.22 liters respectively. FVC values increased progressively with age reaching a plateau at the age of 16-17 years in girls (Fig. 4.6), but the increase continued till 19-20 years in boys (Fig. 4.6). Comparison of rising trend of FVC in relation to age between control and Delhi boys revealed a lower value in the latter group throughout (Fig.4.7). On the other hand, control girls had appreciably higher FVC till 16 years of age, thereafter a modest fall in FVC was recorded (Fig. 4.8).

Figure 4.6. Comparison of FVC (L) between girls and boys of Delhi in relation to age showing consistently lower value in the girls



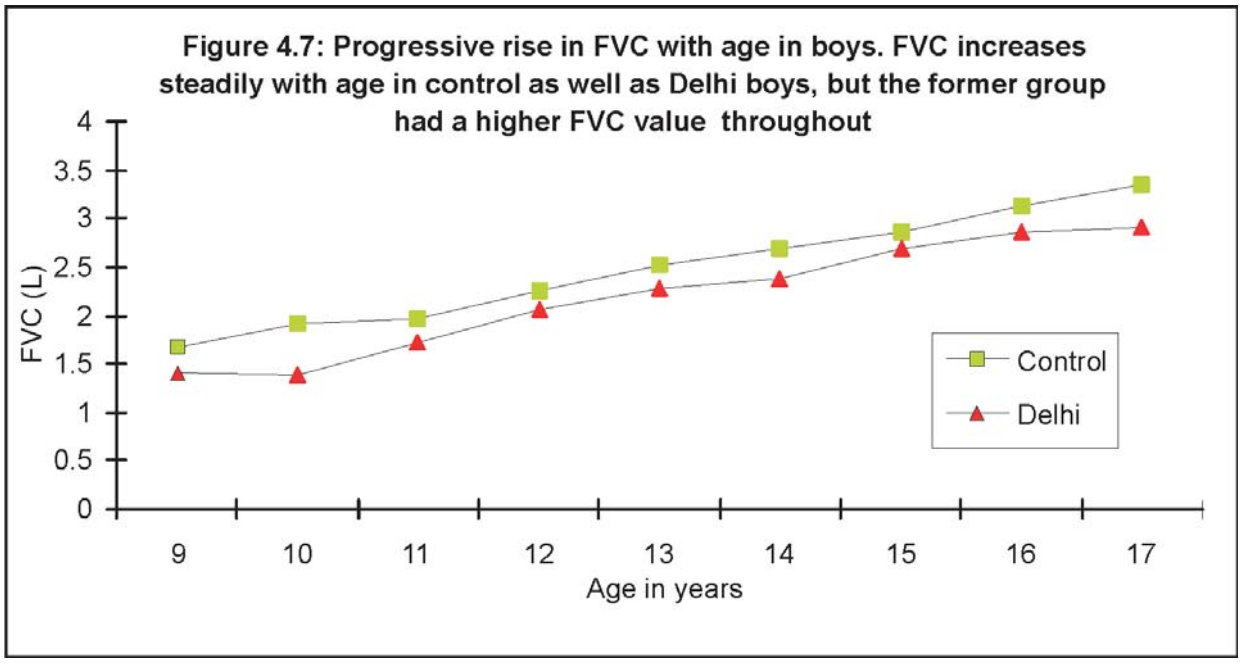


Figure 4.7: Progressive rise in FVC with age in boys. FVC increases steadily with age in control as well as Delhi boys, but the former group had a higher FVC value throughout

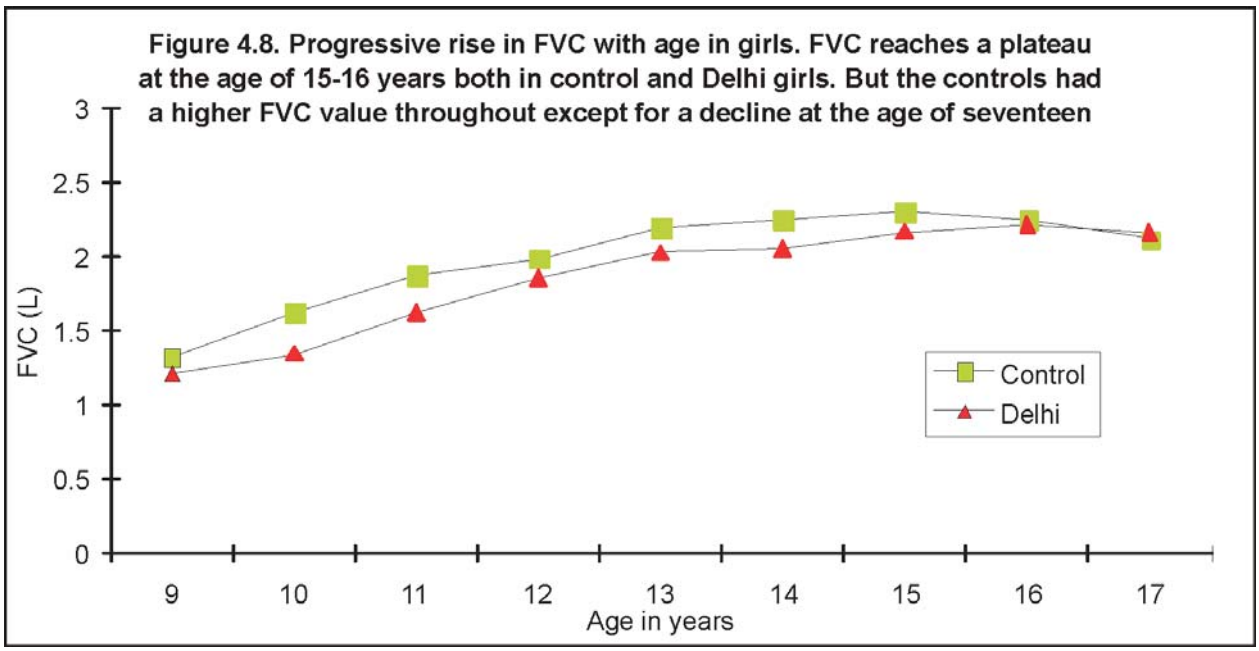


Figure 4.8. Progressive rise in FVC with age in girls. FVC reaches a plateau at the age of 15-16 years both in control and Delhi girls. But the controls had a higher FVC value throughout except for a decline at the age of seventeen

(a) Lower FVC values in girls

FVC differed significantly between the girls and the boys. The measured as well as the predicted value of FVC was significantly lower ($p<0.05$) in the girls (Table 4.4). The male: female ratio of measured FVC was 1.28:1 in control and 1.26:1 in Delhi. A comparison of FVC between boys and girls of Delhi of similar age revealed a consistently lower value in girls.

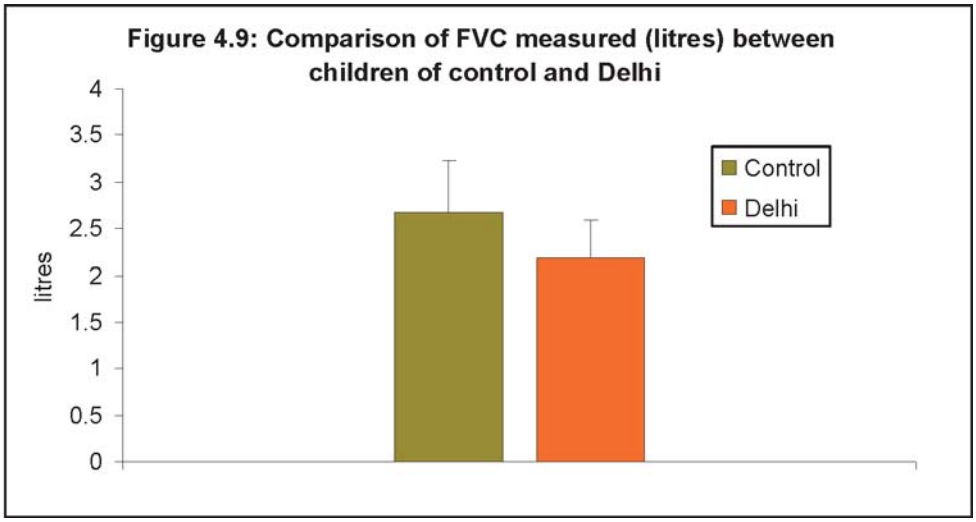
Table 4.4: Comparison of FVC between girls and boys

FVC (liter)	Boys		Girls		Total	
	Control (n=1438)	Delhi (n=3708)	Control (n=807)	Delhi (1963)	Control (n=2245)	Delhi (n=5671)
Measured	2.90 ± 0.73	2.38 ± 0.48*	2.24± 0.48	1.85 ± 0.35*	2.67± 0.55	2.19 ± 0.45*
Predicted	2.76± 0.49	2.81± 0.69	2.19 ± 0.24	2.26 ± 0.35	2.55± 0.42	2.62 ± 0.39
% predicted	105.1	84.7**	102.2	81.8**	104.7	83.6**

**, p<0.05 compared with respective control in Student's 't'-test; **p<0.05 compared with control in Chi-square test*

(b) Reduction of FVC in Delhi

Overall, the school children examined in Delhi had 2.19 ± 0.45 (SD) liters FVC that was 83.6 % of the predicted value based on age, weight, height and ethnicity. In contrast, school children in control group had 2.67± 0.55 liter FVC, which was 104.7% of the predicted value. Thus, Delhi's children showed a significant 480 ml reduction (p<0.001) in mean FVC (Table 4.4, Fig. 4.9). Compared with controls, boys in Delhi illustrated 520 ml reduction (-18%) in mean FVC, while in case of girls the deficit was 390 ml (-17%).



(c) Restrictive type of lung function deficits: 17.8% in control and 29.9% in Delhi's children

Restrictive type of lung function decrement (FVC<80% of predicted value) was recorded in 247 boys (17.2%) and 152 girls (18.8%) of control group (Table 4.3). Overall, 399 control children (17.8%) had restrictive type of lung function deficits. In contrast, 34.8% of the girls of Delhi (683 out of 1963) and 27.3% of the boys (1013 out of 3708) of the city had FVC less than 80% of predicted value, confirming the diagnosis of restrictive type of lung function impairment. Overall, 29.9% children of Delhi (1696 of 5671) had restrictive type of lung function decrement compared with 17.8% of controls, and the difference was highly significant (p<0.001, Table 4.5).

Table 4.5. Gender difference in the prevalence (%) of restrictive type of lung function deficits

Group	Boys	Girls	Total
Control	17.2	18.8	17.8
Delhi	27.3*	34.8*	29.9*

**, p<0.05 compared with respective control in Chi-square test*

(d) Girls suffered more than the boys

Girls had greater prevalence of restrictive type of lung function deficits than the boys both in control group and in Delhi (Table 4.5). For example, 18.8% and 34.8% of the girls in control group and Delhi respectively had restrictive type of lung function reductions compared with 17.2% and 27.3% of the boys in these two groups respectively, and the gender difference in this regard was significant ($p<0.05$).

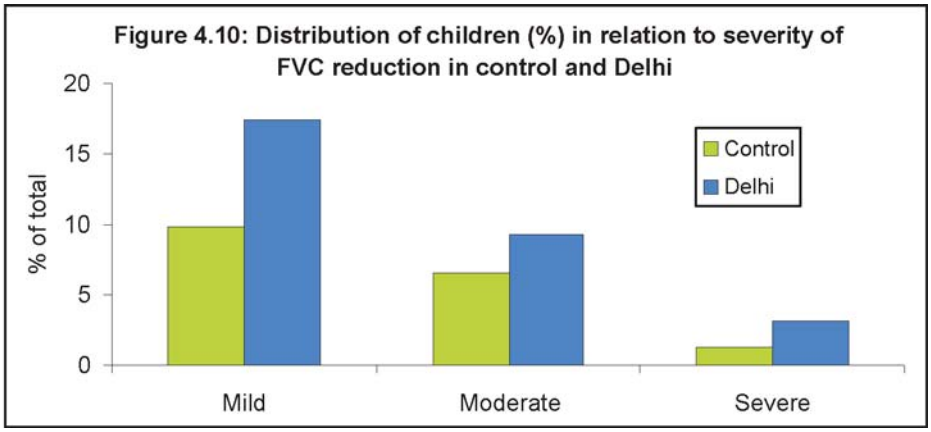
(e) Severe lung restriction in 3.1% children of Delhi compared with 1.3% of control

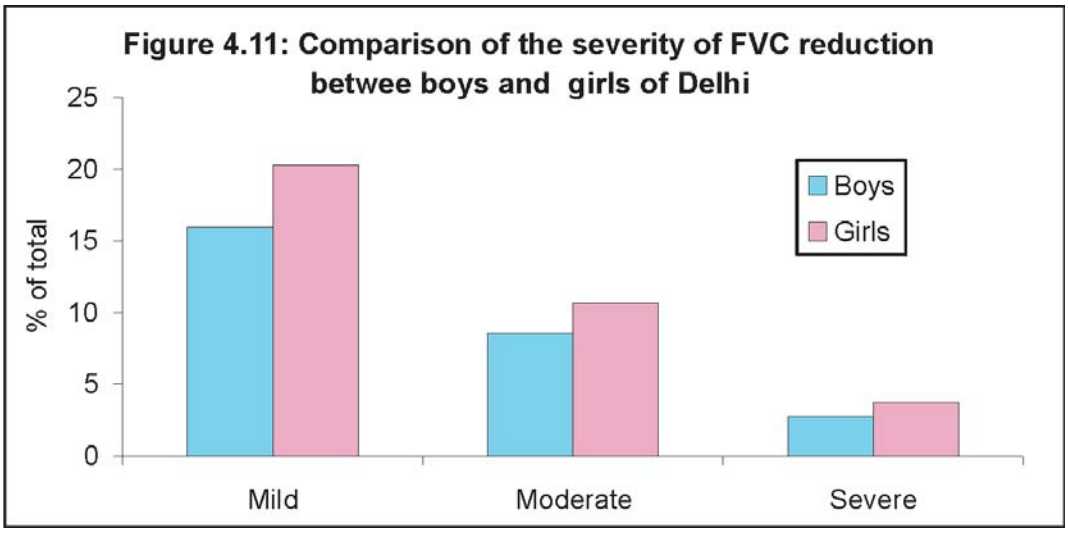
The magnitude of FVC decrement was classified into 3 categories: mild (FVC 60-79% of predicted value), moderate (FVC 40-59% of predicted) and severe (FVC<40% of predicted). A majority of children with restrictive type of lung function deficits had mild restriction both in control and Delhi. For example, 9.8% control and 17.5% Delhi children had mild restriction, 6.6% and 9.3% had moderate restriction, and 1.3% and 3.1% children of control group and Delhi respectively had severe restriction (Table 4.6; Fig. 4.10 & 4.11). The difference in the prevalence of mild, moderate and severe restriction between control and Delhi children was significant ($p<0.001$). The prevalence of severe restriction was more in girls both in control areas and in Delhi.

Table 4.6. Comparison of the severity of FVC reduction between boys and girls

FVC (% predicted)	Boys		Girls		Total	
	Control (n= 1438)	Delhi (n=3708)	Control (n=807)	Delhi (1963)	Control (n=2245)	Delhi (n=5671)
60-79 (Mild reduction)	8.0	15.9*	13.1	20.4*	9.8	17.5*
40-59 (Moderate)	8.0	8.6*	4.2	10.7*	6.6	9.3*
<40 (Severe)	1.2	2.8*	1.5	3.7*	1.3	3.1*

*Results are expressed as percentage of children; *, p<0.05 compared with respective control group in Chi-square test*





(f) Combined type of lung function reduction: 9.6% in Delhi against 3.5% in control

A section of children with restrictive lung also had obstructive type of lung function reduction (FEV_1/FVC <70%). These children are groped under 'combined' type (both restrictive and obstructive) of lung function reduction. The number of such children was 79 (3.5%) in control and 9.6% in Delhi. In the control group, 34 girls (4.2%) and 45 boys (3.1%) had combined type of lung function deficits. Similar problem was recorded in 200 girls (10.2%) and 344 boys (9.3%) of Delhi who participated in this study. Thus, combined type of lung function deficits was also more common in girls.

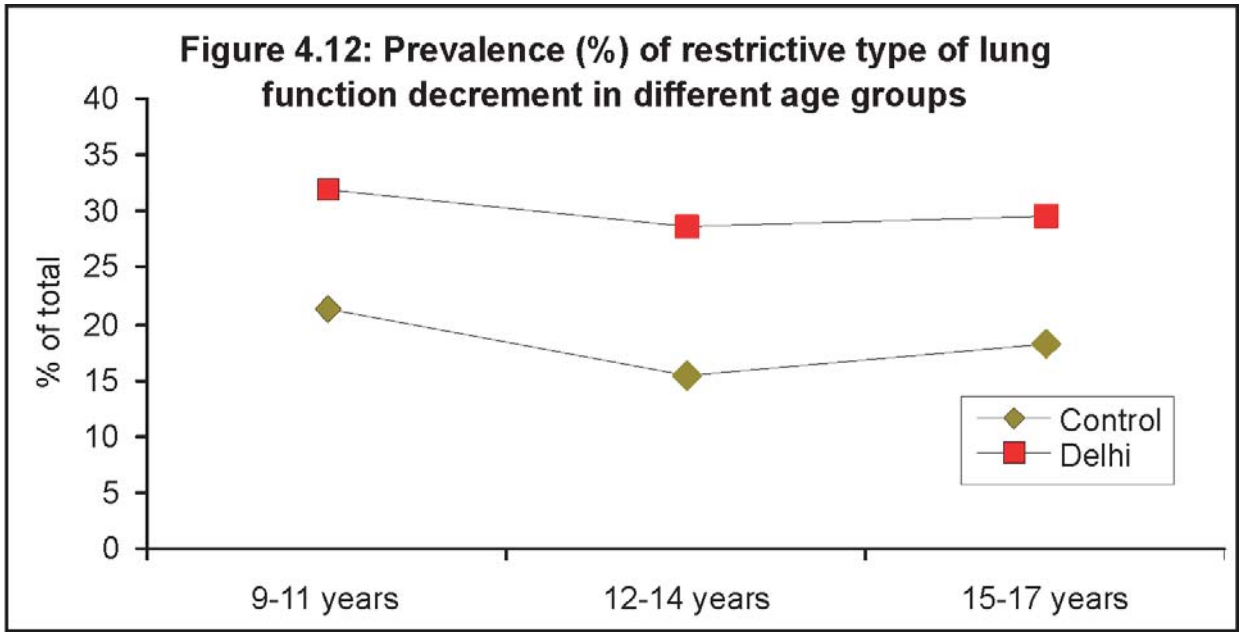
(g) 9-11 year-old children suffered most

The fall in FVC below 80% of predicted value was found in 32.0% of 9-11 year-old children of Delhi compared with 29.5% and 28.7% children in 15-17 year and 12-14 year age groups respectively. Similarly, 21.4% of control children aged 9-11 years had reduced lung function of restrictive type, compared with 15.3% and 18.3% children in 12-14 year and 15-17 year age groups respectively. Therefore, restrictive type of lung function decrement was most prevalent among children aged 9-11 years, and least prevalent in 12-14 year-old children (Table 4.7; Fig. 4.12).

Table 4.7: Prevalence (%) of restrictive type of lung function decrement in different age groups

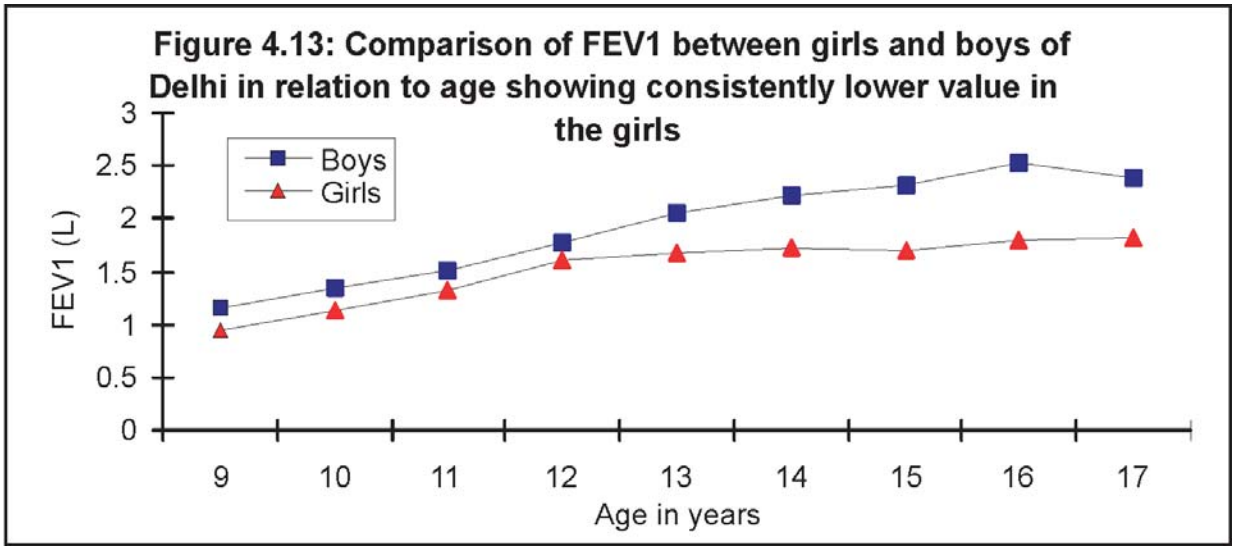
Age (year)	Percentage of children with FVC <80% of predicted value	
	Control	Delhi
9-11	21.4	32.0*
12-14	15.3	28.7*
15-17	18.3	29.5*

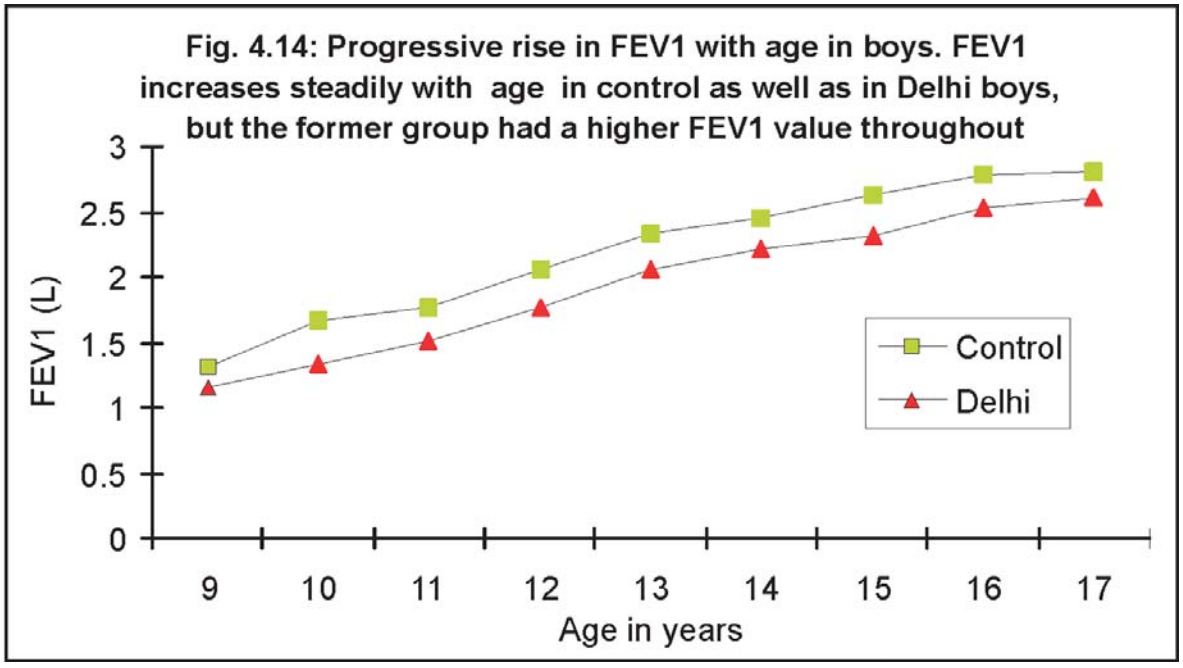
**, p<0.05 compared with respective control group in Chi-square test*

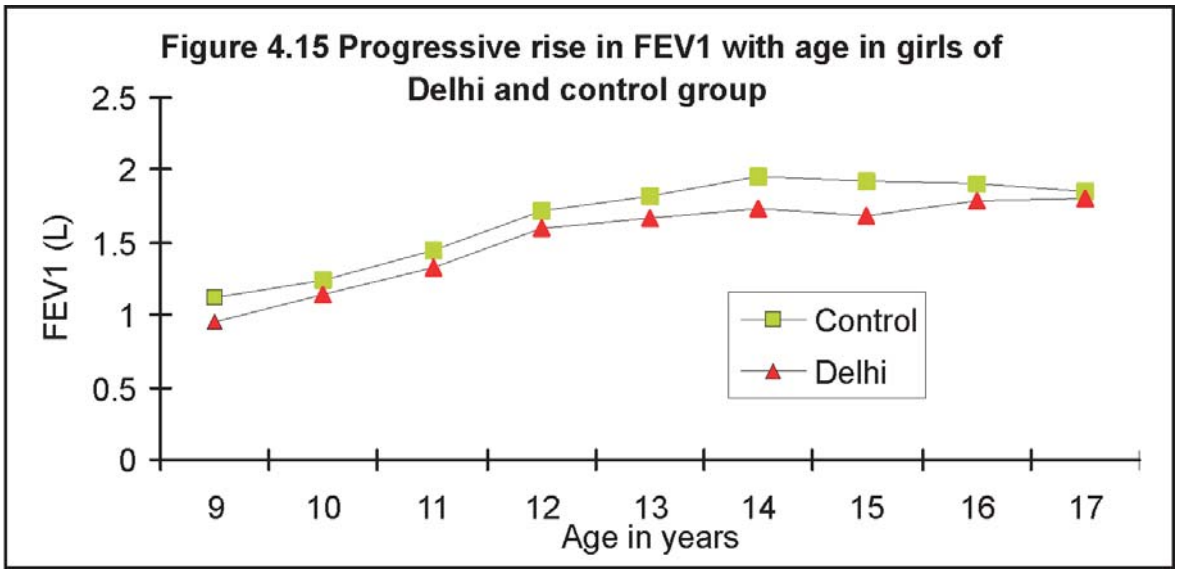


4.3.2. Reduction in FEV_1 in Delhi's children

The mean FEV_1 value in boys aged 9 years was 1.15 liters in Delhi and 1.32 liters in control group. Girls of similar age in Delhi and control had FVC values of 0.95 and 1.13 liters respectively. FEV_1 values increased progressively with age reaching a plateau at the age of 16-17 years in girls (Fig. 4.13), but the increase continued till 19-20 years in boys (Fig. 4.13), as in case of FVC. Comparison of the rising trend in FEV_1 in relation to age between control and Delhi boys and girls revealed a lower value in Delhi in all the age groups (Fig. 4.14 & 4.15).







(a) Lower FEV_1 values in girls

FEV_1 differed significantly between the girls and the boys. The measured as well as the predicted value of FEV_1 was significantly lower ($p<0.05$) in the girls.

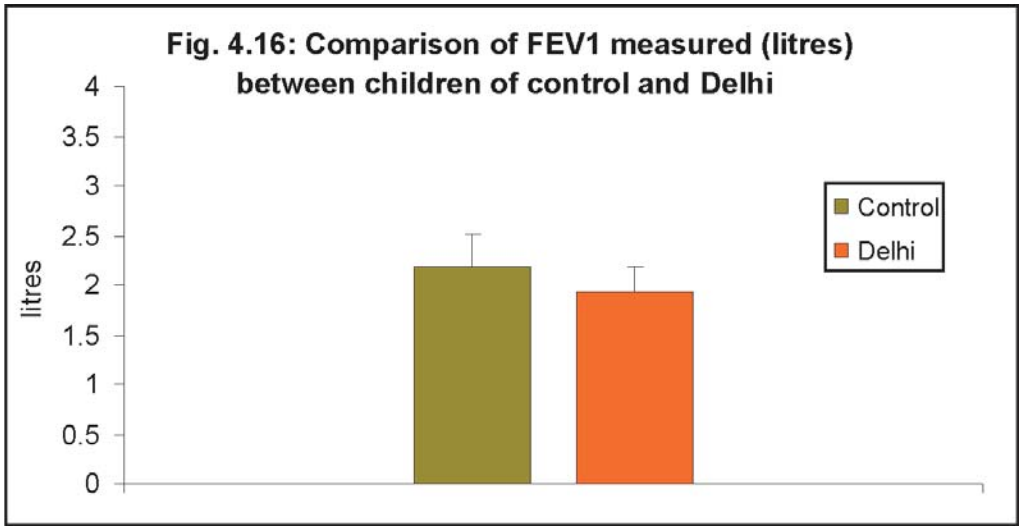
(b) Mean FEV_1

Mean measured forced expiratory volume in one second (FEV_1) was 1.93± 0.25 liter in Delhi compared with 2.19± 0.36 in control group, thereby showing a reduction of 12% in mean FEV_1 in Delhi's children ($p<0.05$, Table 4.8, Fig. 4.16). Compared with respective controls, the decline in mean FEV_1 in Delhi's children was 11% in boys and 14% in girls.

Table 4.8. Comparison of FEV_1 between control and Delhi's children

FEV_1 (liter)	Boys		Girls		Total	
	Control (n=1438)	Delhi (n=3708)	Control (n=807)	Delhi (1963)	Control (n=2245)	Delhi (n=5671)
Measured	2.31 ± 0.58	2.05 ± 0.44*	2.01± 0.41	1.72± 0.37*	2.19± 0.36	1.93± 0.25*
Predicted	2.36± 0.38	2.38 ± 0.45	2.01± 0.22	2.03 ± 0.40	2.23± 0.38	2.25 ± 0.33
% predicted	97.9	86.1**	100.0	84.7**	98.2	85.8**

**, p<0.05 compared with respective control in Student's 't'-test; **p<0.05 compared with control in Chi-square test*



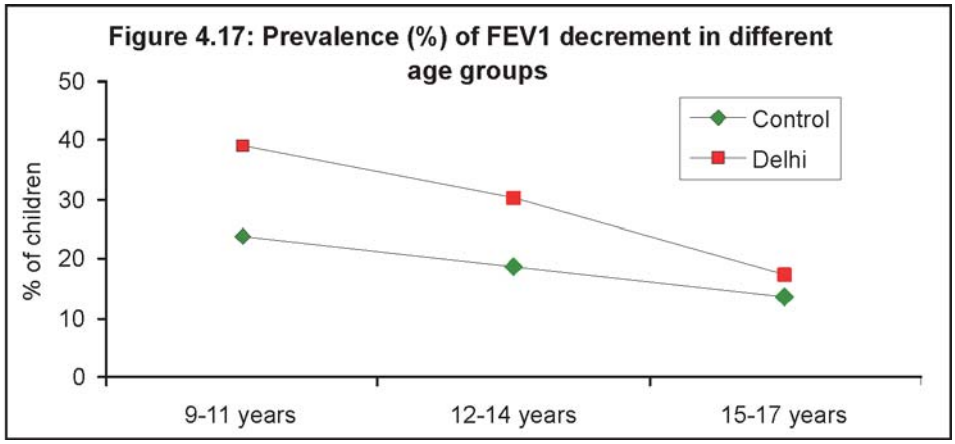
(c) Reduction of FEV_1 in younger age groups

The reduction in FEV_1 below 80% of predicted value was most prevalent among children in 9-11 year age group, and least prevalent in adolescents aged 15-17 years both in rural and urban settings (Table 4.9; Fig. 4.17). However, Delhi's children had reduced FEV_1 in all the age groups when compared with that of controls. Altogether, 28.6% children of Delhi had FEV_1<80% of predicted compared with 18.5% of controls (p<0.001).

Table 4.9: Prevalence of FEV_1 decrement in different age groups

Age group (yr)	% of children with FEV_1 <80% of predicted	
	Control (n=2245)	Delhi (n=5671)
9-11	23.7	39.1*
12-14	18.7	30.4*
15-17	13.6	17.4*
Total	18.5	28.6*

**p<0.05 compared with respective control groups in Chi-square test*



Like FVC, the magnitude of FEV_1 reduction was classified into 3 categories. Mild decrement (FEV_1 60-79% of predicted value) was present in 9.8% of control and 14.9% of Delhi's schoolchildren. Moderate (FEV_1 40-59% of predicted value) and severe (FEV_1<40% of predicted value) reductions were present in 6.6% vs. 9.8% and 2.0% vs. 3.9% schoolchildren of control and Delhi respectively ($p<0.05$). Therefore, the magnitude of FEV_1 reduction was much more in Delhi's schoolchildren compared with rural controls.

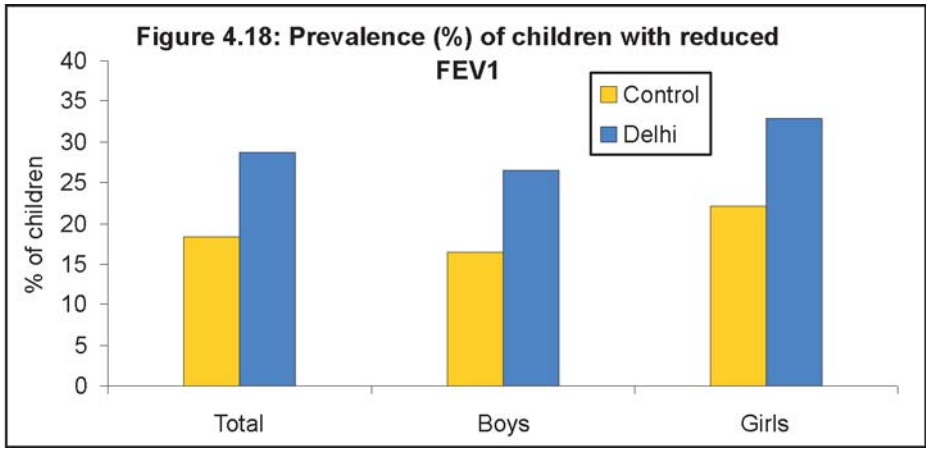
(d) Girls had greater prevalence of reduced FEV_1

Girls showed greater prevalence of reduced FEV_1 than did the boys. For example, 22% of control girls had FEV_1<80% predicted against 16.5% of boys. Similarly, 33% of girls in Delhi had lowered FEV_1 compared with 26.4% of the city's boys (Table 4.10; Fig. 4.18).

Table 4.10: Percentage of children with reduced FEV_1

FEV_1 < 80% predicted	Boys	Girls	Total
Control	16.5	22.1	18.5
Delhi	26.4*	32.9*	28.6*

**, p<0.05 compared with respective control in Chi-square test*



4.3.3. Reduction in FEV_1 /FVC ratio in Delhi's children

The FEV_1/FVC ratio was reduced in Delhi's schoolchildren compared with that of controls. The city's children had a mean FEV_1/FVC ratio of 91.4% (92% in boys and 90.4% in girls) in contrast to 94.3% (95.6% n boys and 92.1% in girls) in controls.

(a) Obstructive type of lung function reduction: 23.2% in Delhi and 11.5% in controls

The hallmark of obstructive type of lung function decrement is reduction in FEV_1/FVC ratio to less than 70%. This type of lung function deficits was found in 1316 (23.2%) schoolchildren of Delhi and 258 (11.5%) children of control group (Table 4.11), and the difference between these two groups in this regard was highly significant ($p<0.001$).

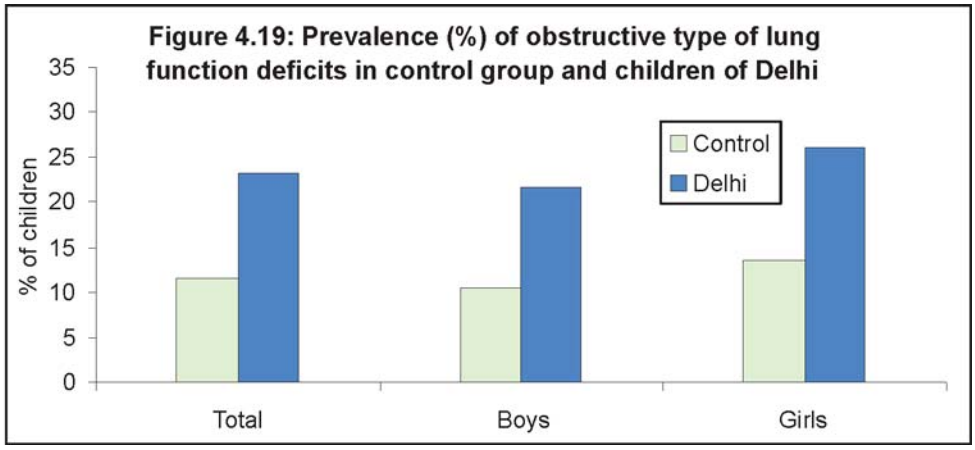
Table 4.11: Prevalence of obstructive type of lung function deficits in children

Group	% of children with FEV_1/FVC <70%	
	Control	Delhi
Boys	10.4	21.7*
Girls	13.5	26.0*
Total	11.5	23.2*

**, p<0.05 compared with control in Chi-square test*

(b) Girls are more affected than the boys

Girls in general had greater prevalence of obstructive lung than the boys both in Delhi and in control group. For example, 26.0% girls of Delhi (510 out of 1963) had obstructive lung compared with 21.7% of city's boys (806/3708). Similarly, 13.5% of control girls (109/807) had lung obstruction compared with 10.4% of control boys (149/1438, Table 4.11; Fig. 4.19).



(c) More severe obstruction in lungs of Delhi' children

After establishing greater prevalence of obstructive type of lung function impairment in Delhi's children the severity of the problem was examined. The magnitude of lung obstruction was classified into three categories on the basis of FEV_1/FVC values: mild (FEV_1/FVC 50-69%), moderate (FEV_1/

FVC 30-49%) and severe (FEV_1/FVC <30%). It was found that Delhi's schoolchildren had 5-times more severe type of lung obstruction than the controls (5.2% in Delhi vs. 1% in control, p<0.001, Fig. 4.20). The change was more noticeable in boys (8-times more than control boys) than in girls (3-time more than control girls, Table 4.12; Fig. 4.21).

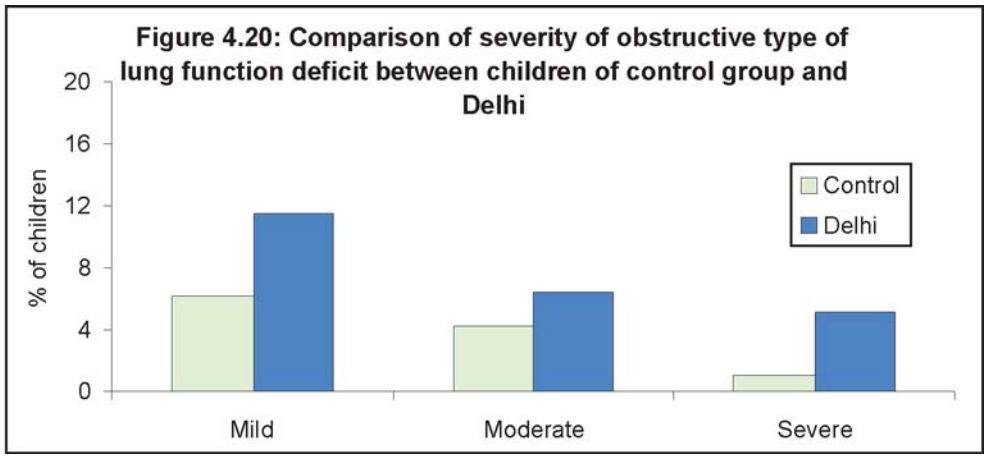
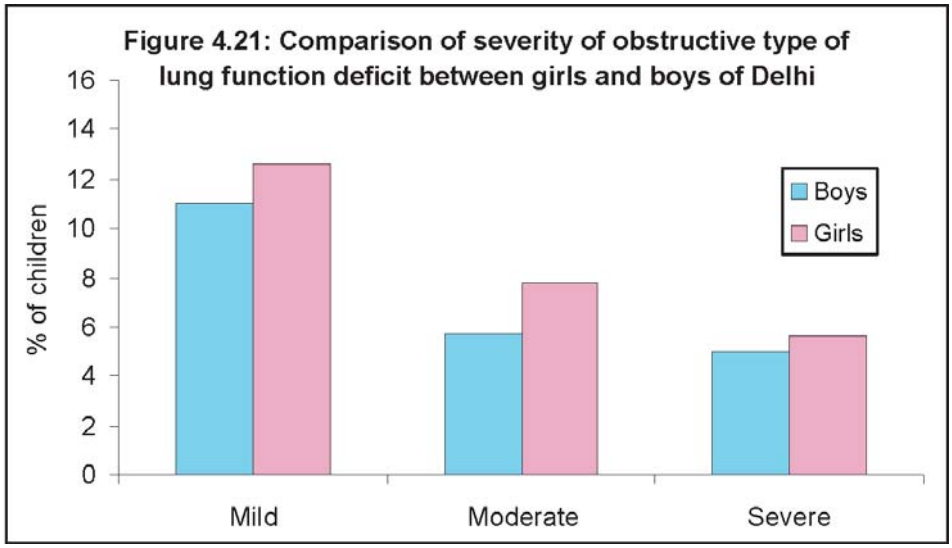


Table 4.12: Severity of obstructive type of lung function deficit in children

FEV_1/FVC (%)	Boys		Girls		Total	
	Control	Delhi	Control	Delhi	Control	Delhi
50-69 (Mild)	6.0	11.0*	6.6	12.6*	6.2	11.5*
30-49 (Moderate)	3.8	5.7	5.1	7.8*	4.3	6.4*
<30 (Severe)	0.6	5.0*	1.8	5.6*	1.0	5.2*

Results are expressed as percentage of children, p<0.001 compared with control in Chi-square test*



The prevalence of obstructive type of lung function deficiency in Delhi was highest (26.3%) in the age group of 15-17 years, followed by children aged 9-11 years (23.6%), while 12-14 year-old children had the lowest (20.8%) prevalence (Table 4.13). In control group also, children belonging

to 12-14 year age group had the lowest prevalence of obstructive lung (10.4%), but greatest prevalence was found in younger age group (13.3% in 9-11 year-old children).

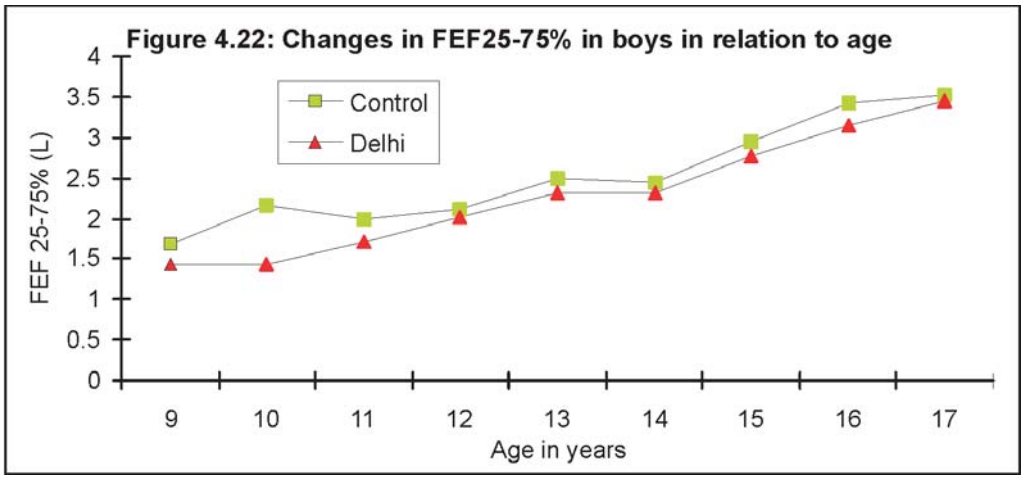
Table 4.13: Prevalence of obstructive type of lung function deficits in children in relation to age

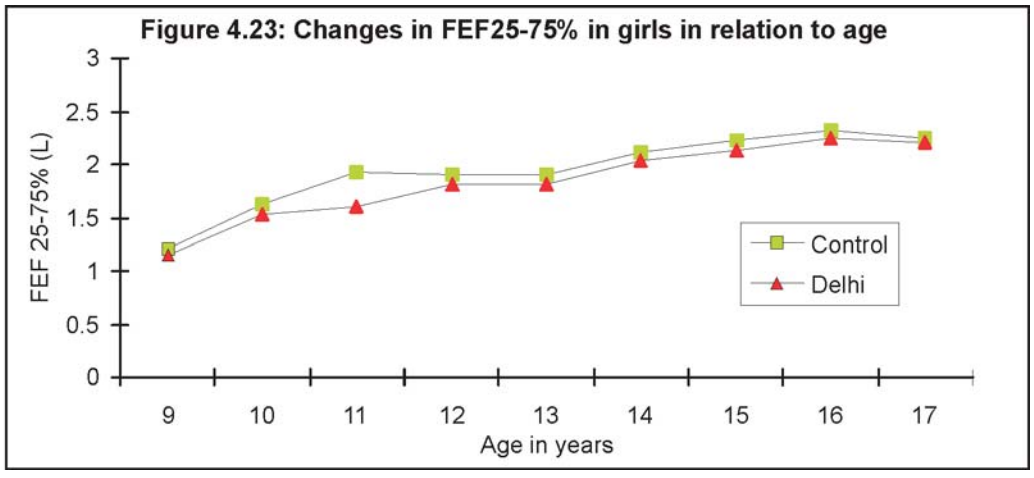
Age of the children	% of children with obstructive lung	
	Control	Delhi
9-11 year	13.3	23.6*
12-14 year	10.4	20.8*
15-17 year	11.9	26.3*

**, p<0.05 compared with control in Chi-square test*

4.3.4 Reduction in $FEF_{25-75\%}$ in Delhi

The mean $FEF_{25-75\%}$ value in boys aged 9 years was 1.44 liters in Delhi and 1.68 liters in control group. Girls of similar age in Delhi and control had $FEF_{25-75\%}$ values of 1.15 and 1.20 liters respectively. $FEF_{25-75\%}$ values increased progressively with age reaching a plateau at the age of 16 years in girls, but the increase continued beyond that age in boys Fig. 4.22 & 4.23).



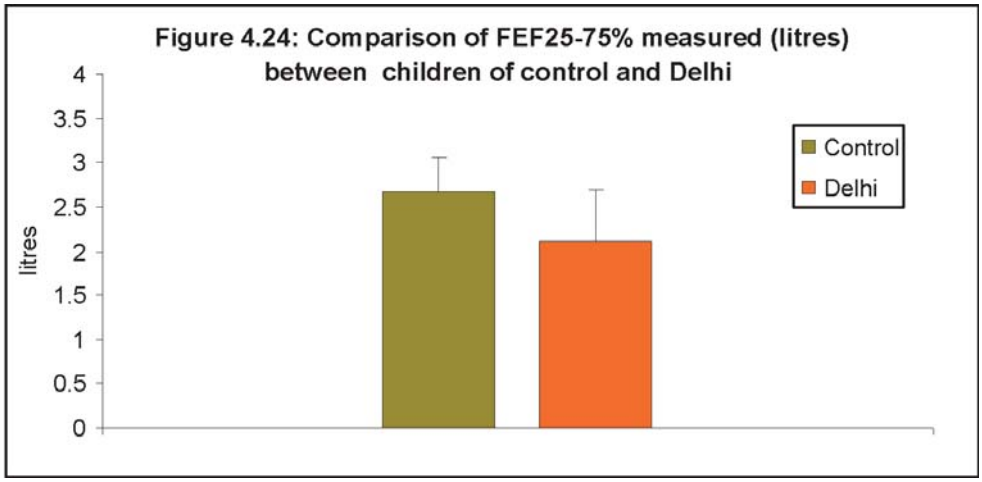


Comparison of $FEF_{25-75\%}$ in relation to age between control and Delhi boys and girls revealed a lower value in Delhi in all the age groups Overall, school children of Delhi demonstrated 21% reduction in $FEF_{25-75\%}$ ($p<0.001$) than the controls, suggesting greater prevalence of underlying small airway obstruction. Compared with respective controls, the reduction of $FEF_{25-75\%}$ was 24.4% in boys, and 21.6% in girls of Delhi. It was observed that the measured value of $FEF_{25-75\%}$ was much lower in both boys and girls of Delhi than the control group (Table 4.14, Fig. 4.24).

Table 4.14: Mean $FEF_{25-75\%}$ in control and Delhi's children

$FEF_{25-75\%}$	Control	Delhi
Boys	2.99 ± 0.65	2.26 ± 0.79*
Girls	2.45± 0.54	1.92± 0.80*
Total	2.66± 0.39	2.10± 0.59*

**, p<0.05 compared with control*



A total of 70.3% children of Delhi had small airway obstruction as evidenced by reduction of $FEF_{25-75\%}$ below 80% of predicted value. In contrast, 32.6% control children had $FEF_{25-75\%}$ < 80% predicted ($p<0.001$). In 17.8% of children of Delhi the reduction in $FEF_{25-75\%}$ was <40% of predicted value, implying severe obstruction of the small airways (Table 4.15). Severe small airway obstruction was found in 9.2% control children, suggesting a 2-fold rise in the prevalence of this problem among the children of Delhi.

Table 4.15: Percentage of children with reduced $FEF_{25-75\%}$

$FEF_{25-75\%}$ (% predicted)	Boys		Girls		Total	
	Control	Delhi	Control	Delhi	Control	Delhi
60-79	11.5	25.4*	15.0	25.5*	12.7	25.5*
40-59	9.9	27.8*	12.1	25.6*	10.7	27.0*
<40	9.2	17.9*	9.2	17.7*	9.2	17.8*

**, p<0.05 compared with respective control*

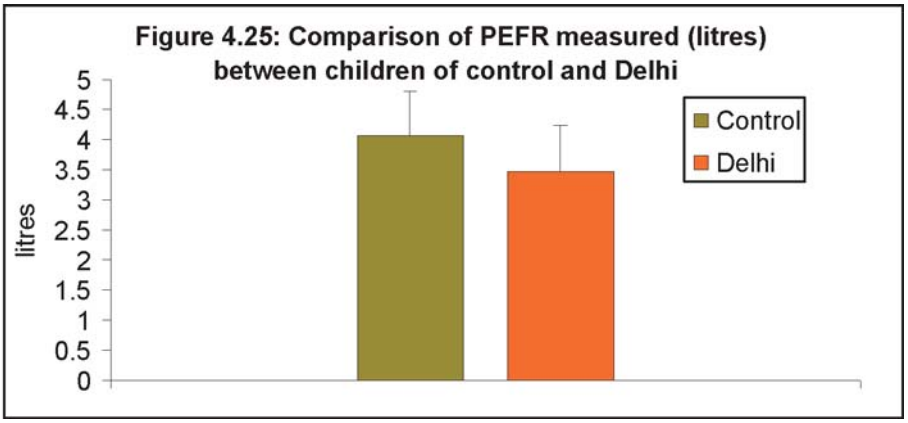
4.3.5. Reduction in PEFR

PEFR was decreased by 15% in Delhi's children when compared with that of control (Table 4.16; Fig. 4.25). The former group had a mean PEFR of 3.46 l/s compared with 4.07 l/s in control (Fig. 4.16). The decline was 17% in boys, and 11% in girls ($p<0.05$ in both cases).

Table 4.16: Comparison of PEFR between control and Delhi's children

PEFR (liter/second)	Control	Delhi
Boys	4.39 ± 0.62	3.63 ± 0.79*
Girls	3.51± 0.78	3.14± 0.81*
Total	4.07± 0.74	3.46± 0.77*

**, p<0.05 compared with control*



A total of 48.4% children of Delhi and 29.4% control children had PEFR value reduced to less than 80% of predicted values based on age, gender and BMI. In 14.2% children of Delhi the reduction in PEFR was <40% of predicted value, against 7.1% in controls ($p<0.001$, Table 4.17). PEFR reduction was more prevalent in girls (54%) than in boys (45.5%) of Delhi.

Table 4.17: Percentage of children with reduced PEFR

PEFR % predicted	Boys		Girls		Total	
	Control	Delhi	Control	Delhi	Control	Delhi
60-79	7.9	16.0*	13.0	17.8*	9.7	16.6*
40-59	11.7	16.3*	14.2	20.0*	12.6	17.6*
<40	6.5	13.2*	8.3	16.2*	7.1	14.2*

**, p<0.05 compared with control in Chi-square test*

4.3.6 Overall prevalence of lung function deficits in school children: 43.5% in Delhi against 25.7% in control

Out of 258 control children who had obstructive type of lung function, 79 children (3.5% of total) had combined type of lung function reduction. Thus, 179 control children (8.0%) had only obstructive type of lung function deficits. Similarly in Delhi, 772 children (13.6%) had obstructive type of lung

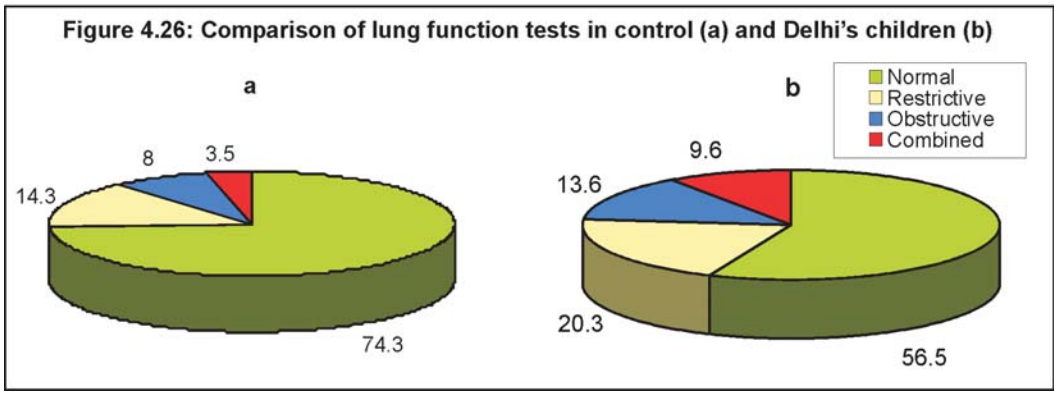
function deficits only. Likewise, only restrictive type of deficit was present in 14.5% control and 20.1% Delhi's children. Overall, lung function was reduced in 2468 (43.5%) schoolchildren of Delhi compared with 578 (25.7%) children of control group (Table 4.18). Therefore, the prevalence of reduced lung function in school-going children was 1.7-times more in Delhi. Delhi's schoolchildren had significantly increased prevalence ($p<0.05$) of restrictive (20.3% v. 14.3%), obstructive (13.6% vs. 8%), as well as combined type of lung functions deficits (9.6% vs. 3.5%; Fig. 4.26).

Table 4.18: Prevalence of lung function deficits in schoolchildren

Type of deficit	Number of children with percentages in parentheses	
	Control (n=2245)	Delhi (n=5671)
Restrictive	320 (14.3)	1152 (20.3)*
Obstructive	179 (8.0)	772 (13.6)*
Combined	79 (3.5)	544 (9.6)*
Overall	578 (25.7)	2468 (43.5)*

**, p<0.05 compared with control in Chi-square test*

Figure 4.26: Comparison of lung function tests in control (a) and Delhi's children (b)

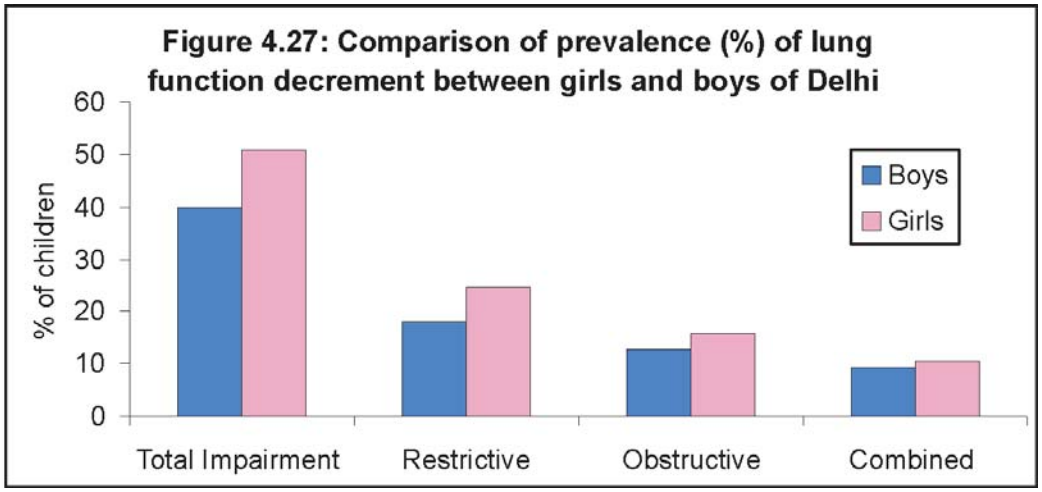


Lung function reduction was more prevalent in girls than the boys both in rural and urban settings. For example, 993 girls of Delhi (51.0%) had reduced lung function compared with 1475 boys (39.8%), and the difference was statistically significant ($p<0.05$). Similarly, 227 girls in controls group (28.1%) had reduced lung function compared with 351 control boys (24.2%). Moreover, the girls had increased prevalence of all three types of lung function deficits (Table 4.19; Fig. 4.27).

Table 4.19: Gender difference in the prevalence of lung function deficits in school children

Type of deficit	Number of children with percentages in parentheses			
	Control		Delhi	
	Boys	Girls	Boys	Girls
Restrictive	202 (14.0)	118 (14.6)	669 (18.0)	483 (24.6)*
Obstructive	104 (7.2)	75(9.3)	462 (12.5)*	310(15.8)*
Combined	45 (3.1)	34(4.2)	344(9.3)*	200(10.2)*
Overall	351 (24.4)	227(28.1)	1475(39.8)*	993(51.0)*

**, p<0.05 compared with control in Chi-square test*

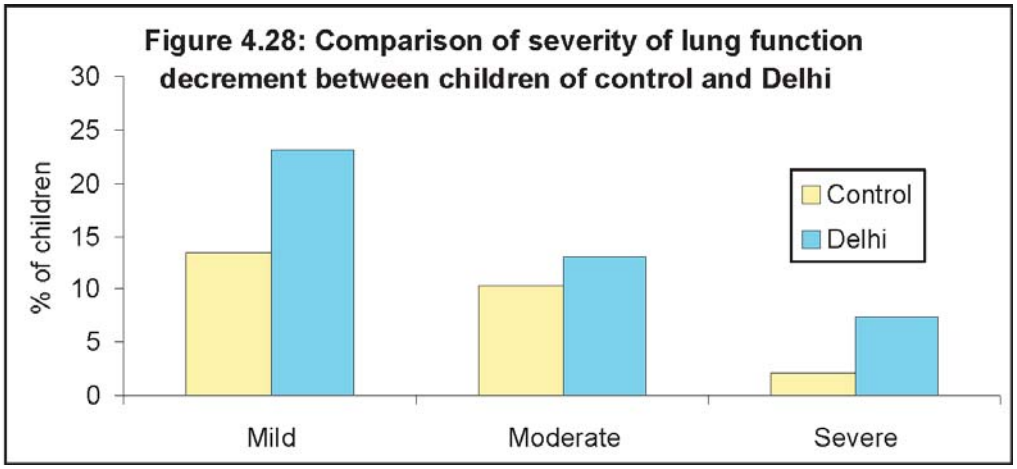


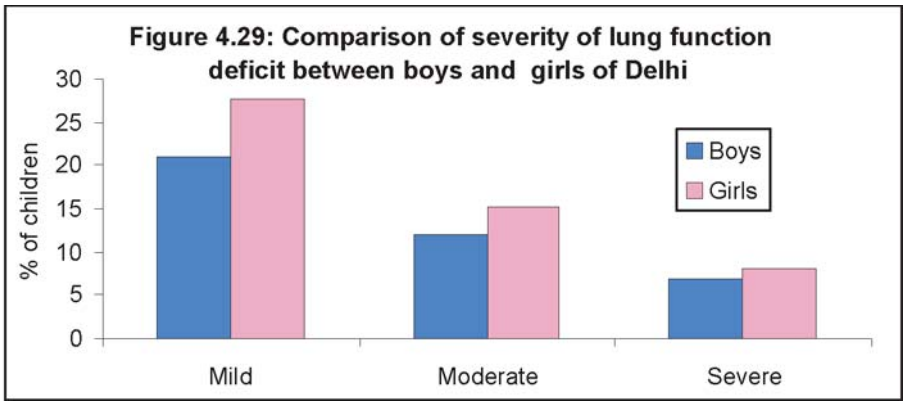
Besides higher prevalence of lung function deficits, the magnitude of the problem was greater in Delhi. For example, 7.3% schoolchildren of Delhi had severe lung function deficits compared with 2.2% children in control group with severe lung function decrement ($p<0.05$, Table 4.20; Fig. 4.28). Moderate lung function deficit was present in 13.1% children of Delhi compared with 10.2% of control, while mild deficit in lung function was present in 23.1% and 13.4% children of Delhi and control group respectively. Compared with boys, severe reduction in lung function was more prevalent among the girls both in Delhi and in control group (Table 4.20, Fig. 4.29).

Table 4.20: Magnitude of lung function reduction in schoolchildren of Delhi compared with rural control

Magnitude of deficits in PFT	Boys		Girls		Total	
	Control	Delhi	Control	Delhi	Control	Delhi
Mild	11.5	20.9*	17.0	27.7*	13.4	23.1*
Moderate	11.3	12.0	8.1	15.2*	10.2	13.1
Severe	1.7	6.9*	3.1	8.0*	2.2	7.3*

*Results are expressed as percentage of total children; *, $p<0.05$, compared with respective control in Chi-square test*





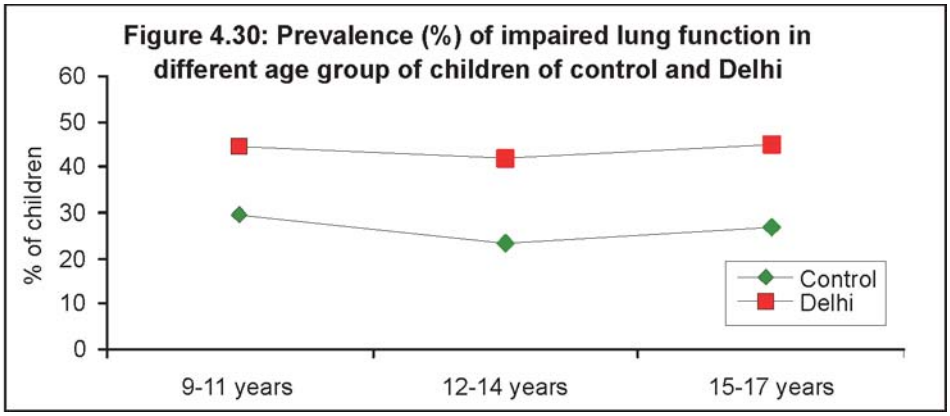
(a) Lung function reduction in different age groups

In Delhi, the prevalence of lung function reduction was highest (44.9%) among schoolchildren in the age group of 15 – 17 years, followed by 44.5% prevalence in 9-11 year-old children and 41.7% in 12-14 year-old children (Table 4.21; Fig. 4.30). On the other hand, highest prevalence of lung function reduction in control group (29.6%) was found in 12-14 year age group, followed by 26.8% in 15-17 year-old, and 23.2% in 12-14 year-old children.

Table 4.21: Prevalence of obstructive type of lung function deficits in school children in relation to age

Age	% Children with lung function deficits	
	Control	Delhi
9-11 year	29.6	44.5*
12-14 year	23.2	41.7*
15-17 year	26.8	44.9*

**, p<0.001 compared with control in Chi-square test*



(b) Seasonal variation in lung function

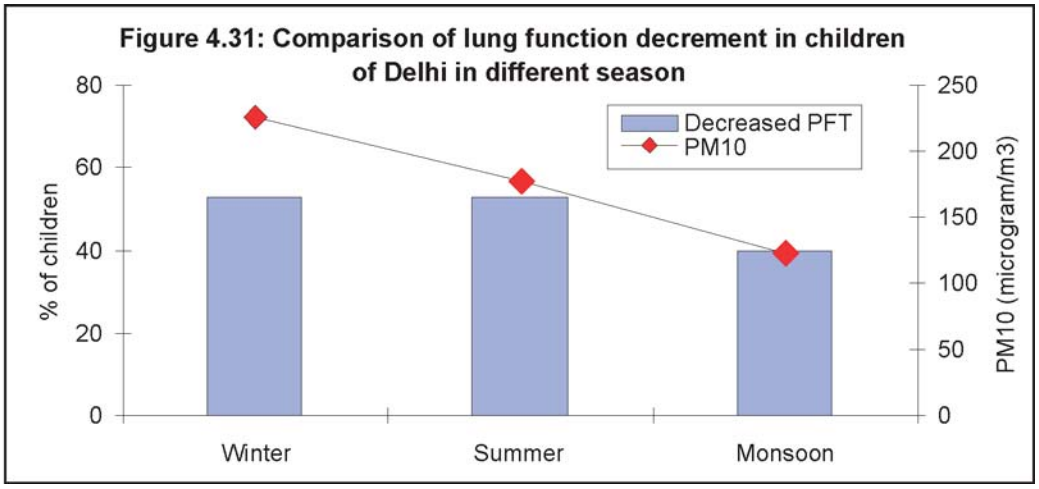
Lung function reduction was found to be associated with seasonal variations. In control group, prevalence of lung function reduction was highest during winter (32.9%) when the particulate pollution level in ambient air was highest. Conversely, lowest prevalence of lung function deficits in

schoolchildren was recorded in monsoon (19.9%) when the breathing air is cleanest. In Delhi, however, high prevalence of lung function deficits was observed both in winter and summer (52.7% in both seasons, Table. 4.22, Fig. 4.31), while a much lower prevalence (39.9%) was observed in monsoon. The difference in the prevalence of lung function deficits between winter/summer and monsoon in Delhi was significant in Chi–square test ($p<0.05$).

Table 4.22: Seasonal variation in the prevalence (%) of lung function decrement

Season	Control	Delhi
Summer	25.5	52.7*
Monsoon	19.9	39.9*
Winter	32.9	52.7*

**, p<0.001 compared with control in Chi-square test*



(c) Inverse relationship between SES and lung function deficits

An inverse relationship was found between SES and the prevalence of lung function deficits: lower the SES greater the percentage of children with reduced lung function. Keeping the prevalence of lung function deficit in high SES constant (OR=1), conditional logistic regression analysis showed that the prevalence of reduced lung function increased significantly in children from families belonging to medium and low SES both in rural and urban settings (Table 4.23).

Table 4.23: Conditional logistic regression analysis for association between lung function deficits and socio economic status (SES)

SES	Lung function deficits	
	Control	Delhi
High	1	1
Medium	1.34 (1.12-1.89)*	1.47 (1.29 – 1.74)*
Low	1.98 (1.25-2.67)*	1.80* (1.56 – 2.54)*

*The results are expressed as odds ratio with 95% CI in parentheses. *, p<0.05 compared with high SES*

(d) Parental smoking and children's lung function

About 28% of control and 27% of Delhi's children were exposed to environmental tobacco smoke (ETS) at home due to smoking habit of their fathers or some other male member of the family. These children had lower lung function than those unexposed to ETS. For example, 50.6% of Delhi's children exposed to ETS (768/1517) had lung function deficits, compared with 40.9% non-exposed children (1700/4154). Similarly in control group, 27% children exposed to ETS and 21.3% non-exposed children had lung function deficits.

(e) Relationship between PM_{10} and the prevalence of lung function deficits

After controlling potential confounders like season, socioeconomic conditions and ETS, PM_{10} level in ambient air was found to be associated with restrictive (OR= 1.35, 95%CI 1.07-1.58), obstructive (OR=1.45, 95% CI 1.16-1.82), and combined type of lung function deficits (OR= 1.74, 95%CI 1.37-2.71) in children.

Spearman's rank correlation test reaffirmed the association. It was found that the decrease in all the lung function measurements was correlated with PM_{10} level in ambient air (Fig. 4.32). The correlation was strongest for FEV_1/FVC ratio (rho value –0.986, $p<0.0005$), followed by $FEF_{25-75\%}$ (rho value –0.944, $p<0.0005$), FVC (rho = -0.912, $p<0.0005$), PEFR (rho = -0.542, $p<0.001$), and FEV_1 (-0.472, $p<0.001$).

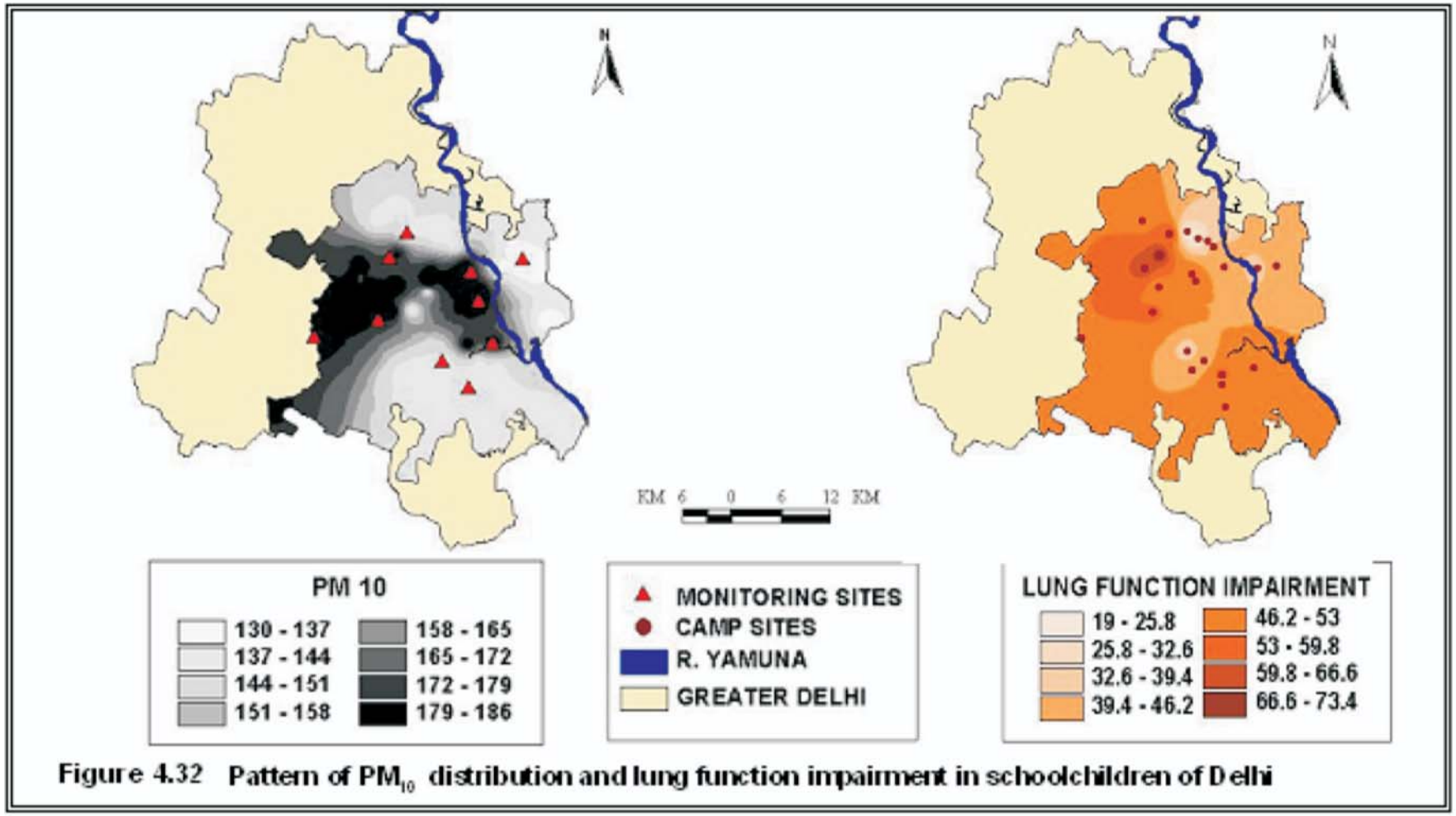


Figure 4.32 Pattern of PM_{10} distribution and lung function impairment in schoolchildren of Delhi

Similarly, the existence of a direct relationship between PM_{10} level in ambient air and lung function deficits was observed in conditional logistic regression analysis. Increasing levels of PM_{10} were found to be associated with increased lung function deficits of obstructive and restrictive type. In nearly all the cases it was found that as PM_{10} increased lung function deficit also increased (Table 4.24).

Table 4.24: Conditional logistic regression analysis of the relationship between PM_{10} level in ambient air and children's lung function.

PM_{10} (µg/m^3)	Reduced lung function	Restrictive type (FVC<80%)	Obstructive type (FEV_1/FVC<70%)
50-75	1	1	1
76-100	1.34* (1.06 – 1.68)	1.48* (1.15 – 1.91)	0.96 (0.70 – 1.23)
101-125	1.62* (1.40 – 1.87)	1.53* (1.30 – 1.81)	1.42* (1.12 – 1.80)
>125	3.75* (3.50 – 4.60)	4.82* (3.90 – 5.97)	1.59* (1.28 – 1.20)

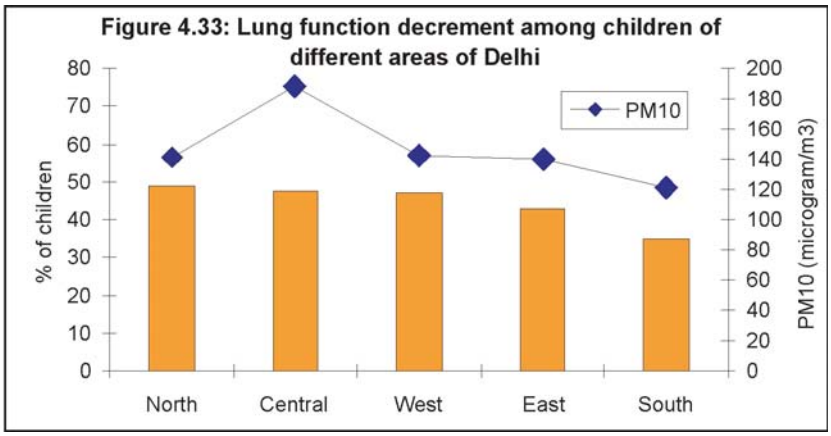
*The results are expressed as odds ratio with 95% CI in parentheses; *, p<0.05*

(f) Lung function in schoolchildren of different areas of Delhi

The prevalence of lung function decrement among school-age children varied greatly in different areas of the city. It was 48.5% in north Delhi, highest in the city, and 34.3% in south Delhi, the lowest in Delhi (Table 4.25; Fig. 4.33). Children from central (47.2%) and west Delhi (46.8%) also had higher prevalence of lung function reductions.

Table 4.25: Lung function in children of different areas of Delhi

Area	No. of children examined	Children with reduced PFT	Prevalence (%)	% Change from city's overall prevalence
East	602	259	43.0	-1
West	511	241	47.2	+9
North	872	426	48.9	+12
South	1687	589	35.0	-20
Central	1999	953	47.7	+10
Overall	5671	2468	43.5	-



Conditional logistic regression analysis revealed that compare with school children from the south, school children from west, north and central had significantly greater prevalence ($p<0.05$) of reduced lung function (Table 4.26).

Table 4.26: Conditional logistic regression analysis of the relationship between lung function of the children and residential area

Residential Area	Reduced lung function	Restrictive type	Obstructive type
South	1	1	1
West	1.47(1.20 – 1.80)*	1.48 (1.21 – 1.83)*	1.47(1.16 – 1.85)*
North	1.72 (1.46 – 2.03)*	1.93 (1.63 – 2.29)*	1.29 (1.05 – 1.57)*
Central	1.51 (1.25 – 1.82)*	1.22 (0.99 – 1.49)	1.62 (1.30 – 2.01)*
East	1.04 (0.92 - 1.20)	0.78 (0.68 – 0.91)	1.23 (1.08 – 1.44)*

*The results are expressed as odds ratio with 95% CI in parentheses; * p<0.05 compared with South*

The magnitude of decrement in lung function also varied in different areas. Severe reduction in lung function was found among 10% (87/872) and 9.2% (183/1999) schoolchildren of North and Central Delhi respectively, whereas only 4.3% schoolchildren of South Delhi (72/1687) had severe reduction in lung function. In essence, greatest impairment of lung function was found in children of North, Central and West Delhi while best lung activity was found in children of South Delhi (Table 4.27).

Table 4.27: Comparison of the magnitude of lung function decrement in school children of different areas of Delhi

Magnitude of lung function decrement	East (n=602)	West (n=511)	North (n=872)	South (n=1687)	Central (n=1999)
Mild	23.4	23.9	23.0	21.8	24.0
Moderate	14.0*	15.8*	16.0*	8.8	14.6*
Severe	5.6	7.4*	10.0*	4.3	9.2*

*The results are expressed as percentage of school children with reduced lung function of variable severity; * p<0.05 in Chi square test compared with South*

4.3.7. Body mass index (BMI) and lung function

(a) Prevalence of overweight in school children

It is evident from Table 4.26 and figure 48 that 5.4% school children of Delhi enrolled in this study were overweight on the basis of their BMI data. In contrast, 2.4% children of the control group were overweight (Table 4.28), and the difference between these two groups in this regard was highly significant ($p<0.001$).

Moreover, 9% of Delhi's school children were at risk of being overweight compared with 4.4% children of control group ($p<0.001$). On the other hand, the prevalence of

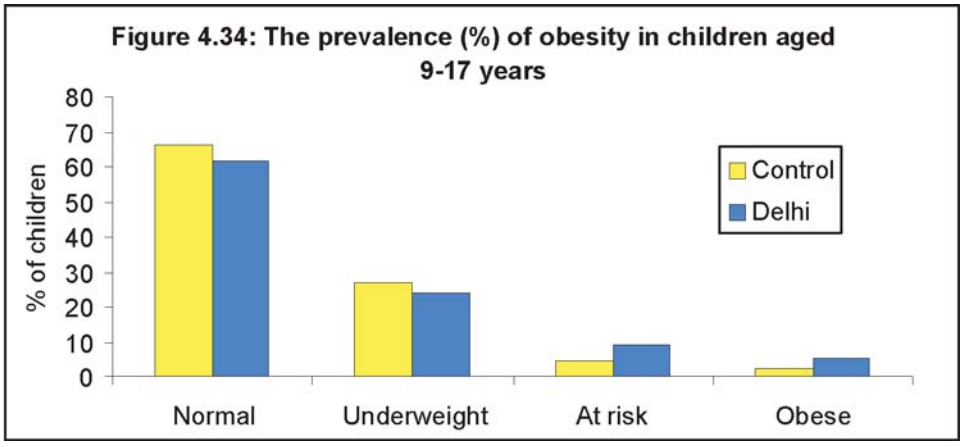
underweight children was greater in control group (26.8 vs. 23.9% in Delhi Tables 4.28 & 4.29, Fig. 4.34).

Table 4.28: Body mass index of boys and girls of Delhi

	Underweight (%)	Normal (%)	At risk (%)	Overweight (%)
Total (n=5671)	23.8	61.8	9.0	5.4
Boy (n=3708)	27.5	58.6	8.6	5.3
Girl (n=1963)	17.1	67.9	9.6	5.5

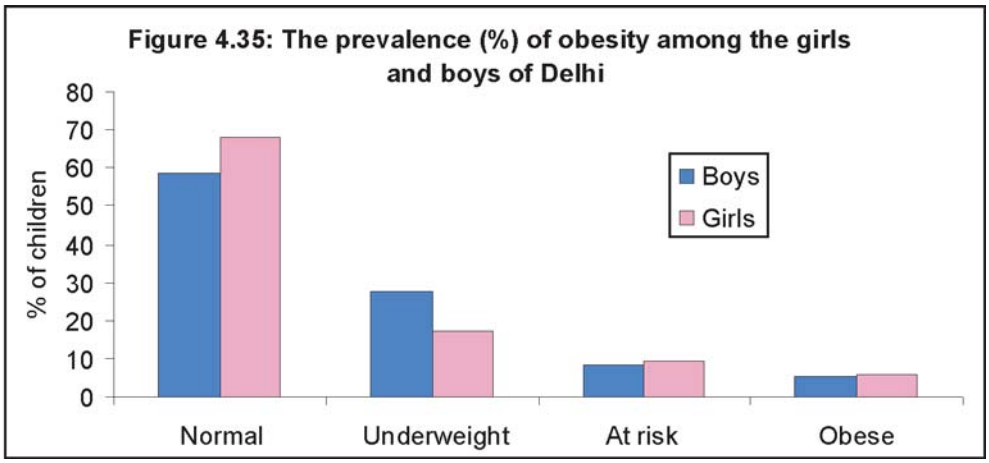
Table 4.29: Body mass index of boys and girls of controls

	Underweight (%)	Normal (%)	At risk (%)	Overweight (%)
Total (n=2245)	26.8	66.4	4.4	2.4
Boy (n=1438)	30.0	63.5	4.0	2.4
Girl (n=807)	20.9	71.6	5.0	2.3



(b) Difference between boys and girls

Girls had a slightly higher prevalence of overweight or at risk of being overweight than the boys both in Delhi and the control group (Tables 4.28 & 4.29, Fig. 4.35).



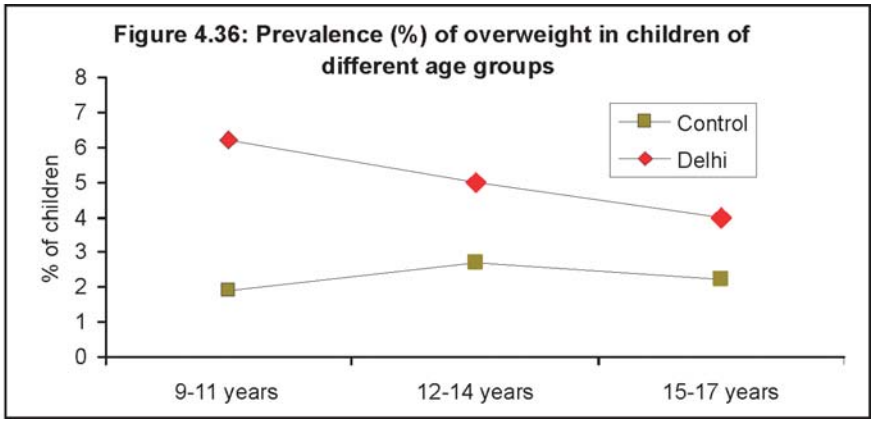
(c) Prevalence of overweight in relation to age

The problem of overweight was most prevalent (6.2%) in Delhi's school children aged between 9 and 11 years. Thereafter the prevalence progressively declined with age. In control group, overweight children were most prevalent (2.7%) in 12-14 year age group, and a modest decline in the prevalence of overweight was recorded thereafter (Table 4.30, Fig. 4.36).

Table 4.30: Prevalence of overweight among school children in relation to age

Age	At risk of overweight (%)Overweight (%)			
	Control	Delhi	Control	Delhi
9-11 years	3.4	9.1*	1.9	6.2*
12-14 years	4.6	9.1*	2.7	5.9*
15-17 years	4.8	8.8*	2.2	4.0*

**p<0.05 in Chi square test compared with control*



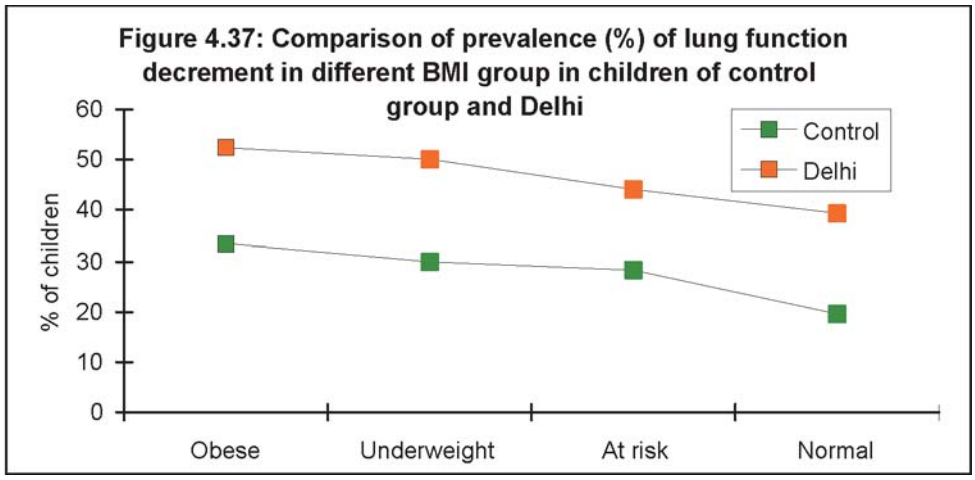
(d) BMI and lung function

BMI of the children had profound influence on their lung function. It was observed that overweight and underweight children had poor lung function than children with normal weight. For instance, 170 out of 306 overweight children (55.6%) of Delhi had decreased lung function compared with 39.3% children (1377/3505) with normal body weight (Table 4.31; Fig. 4.37). Children at risk of being overweight had 48% prevalence of lung function deficits in Delhi. Underweight children also had high incidence of lung function deficits in Delhi (50.1%) as well as in control group (30.0%). Thus, children with excess or subnormal body weight appeared to be more prone to lung function deficits.

Table 4.31. Lung function deficits in children with abnormal body weight

BMI status	% children with reduced lung function	
	Control	Delhi
Underweight	30.0	50.1*
Normal	19.4	39.3*
At risk	28.3	48.0*
Overweight	33.3	55.6*

**p<0.05 in Chi square test compared with control*



4.4 FINDINGS

1. Lung function test was conducted in schoolchildren aged 9-17 years by portable spirometer following the protocol of American Thoracic Society. A total number of 2245 control and 5671 Delhi's children successfully completed the test.

2. The results showed reduction of lung function in 43.5% schoolchildren of Delhi compared with 25.7% in control group. Delhi's children had increased prevalence of restrictive (20.3% vs 14.3%8 in control), obstructive (13.6% vs. 8%), as well as combined (both restrictive and obstructive) type of lung functions deficits (9.6% vs. 3.5%).

3. Lung function reduction was more prevalent in girls than the boys both in rural and urban settings. In Delhi, 51% of the girls had reduced lung function compared with 39.8% of age-matched boys. In control group, 28.1% of the girls had lung function deficits compared with 24.4% of the boys.

4. The prevalence of lung function reduction in Delhi was highest (44.9%) among schoolchildren in the age group of 15 – 17 years. Children belonging to 9-11 year and 12-14 year age group had 44.5% and 41.7% prevalence of lung function deficits respectively. On the other hand, highest prevalence of lung function reduction in control group (29.6%) was found in 12-14 year age group, followed by 26.8% in 15-17 year-old, and 23.2% in 12-14 year-old children.

5. Besides higher prevalence, the magnitude of lung function impairment was much more in Delhi. For example, 7.3% schoolchildren of Delhi had severe lung function deficits compared with 2.2% children in control group.

6. Prevalence of lung function reduction in schoolchildren varied considerably with season. In control group, prevalence of lung function reduction was highest during winter (32.9%) when the particulate pollution level in ambient air was highest. Conversely, lowest prevalence of lung function deficits in schoolchildren was recorded in monsoon (19.9%) when the breathing air is cleanest. In Delhi, however, high prevalence of lung function deficits was observed both in winter and summer (52.7% in both seasons), while a much lower prevalence (39.9%) was observed in monsoon. The difference in the prevalence of lung function deficits between winter/summer and monsoon in Delhi was significant ($p<0.05$).

7. An inverse relationship exists between socio-economic status (SES) and the prevalence of lung function deficits: lower the SES, greater the percentage of children with reduced lung function. Conditional logistic regression analysis showed that the correlation was significant.

8. Exposure to environmental tobacco smoke (ETS) at home due to smoking habit of any member of the family increases the possibility of lung function deficits in children. For example, 50.6% of Delhi's children exposed to ETS had lung function deficits compared with 40.9% non-exposed children. Similarly in control group, 27% children exposed to ETS and 21.3% non-exposed children had lung function deficits.

9. After controlling potential confounders like season, socioeconomic conditions and ETS, PM_{10} level in ambient air was found to be positively associated with restrictive (OR= 1.35, 95%CI 1.07-1.58), obstructive (OR=1.45, 95% CI 1.16-1.82), and combined type of lung function deficits (OR= 1.74, 95%CI 1.37-2.71) in children. Spearman's rank correlation test reaffirmed the association. It was found that the decrease in all the lung function measurements was correlated with PM_{10} level in ambient air. The correlation was strongest for FEV_1/FVC ratio (rho value –0.986, $p<0.0005$), followed by FEF 25-75% (rho value –0.944, $p<0.0005$), FVC (rho = -0.912, $p<0.0005$), PEFR (rho = -0.542, $p<0.001$), and FEV_1 (-0.472, $p<0.001$). Similarly, the existence of a direct relationship between PM_{10} level in ambient air and lung function deficits was observed in conditional logistic regression analysis. Increasing levels of PM_{10} were found to be associated with increased prevalence of lung function deficits.

10. Based on BMI data, 5.4% children of Delhi enrolled in this study were overweight against 2.4% children in control ($p<0.001$). Besides, 9% of Delhi's children were at risk of being overweight compared with 4.4% children in controls ($p<0.001$). On the other hand, the prevalence of underweight children was greater in the control group. The problem of overweight was most prevalent in children aged between 9 and 11 years in Delhi and 12-14 years in control.

11. BMI was shown to have profound influence on lung function. Overweight and underweight children had poor lung function than children with normal weight. For instance, 55.6% of overweight children of Delhi had decreased lung function compared with 39.3% children with normal body weight. Delhi's children who were at risk of being overweight demonstrated 48% prevalence of lung function deficits. Underweight children also had higher rate of reduced lung function in Delhi as well as in control group. Thus, children with excess or subnormal body weight appeared to be more prone to lung function deficits.

CHAPTER-5.0

ASSESSMENT OF CELLULAR LUNG REACTION TO DELHI'S AIR POLLUTION

5.1 INTRODUCTION

Adverse effect of Delhi's air pollution on lung function of the children has been shown in the previous Chapter. It may be emphasized that functional impairment is the ultimate manifestations of a complex pattern of cellular changes that took place for a long time inside the lung. Therefore chronic exposures to city's air pollution are probably impairing the lung at the cellular level, and it was of interest to explore this possibility.

The lung defense against inhaled particles and gaseous pollutants include innate mechanism such as aerodynamic filtration, muco-ciliary clearance, particle transport and detoxification by alveolar macrophages, as well as local and systemic innate and acquired anti viral immunity. In particular, alveolar macrophages provide an innate defense mechanism against bacteria and viruses. Virus particles are ingested by phagocytosis and macrophages, like epithelial and other virus infected cells, produce interferons that potentially inhibit viral replication. AM also contribute to the neutralization of viral infection by removing the debris of the destroyed virus-containing cells and by presenting viral antigen to T lymphocytes. In addition cell mediated immunity such as the development of cytotoxic T lymphocytes which are capable of destroying cells infected with virus, play an important role in the control of many viral infections of the respiratory tract. Many of these functions can be modulated by exposure to PM_{10}, NO_2 and other air pollutants (Chauhan and Johnston, 2003; Chauhan et.al., 2005)

Objective

The objective of this study was to examine whether sustained exposures to ambient air pollution of the city are causing i) inflammatory and allergic changes in the lung, ii) impairment of lung defense with special reference to the activity of alveolar macrophages, the first line of cellular defense in the lung, iii) damage to the alveolar wall, and iv) microscopic hemorrhages inside the lung that may impair lung activity.

5.2 MATERIALS AND METHODS

(a) Subjects and sputum collection

A total number of 250 school children, 100 (boys 65, girls 35) from control group and 150 from Delhi (boys 98, girls 52) participated in sputum analysis study. The children were aged between 13-15 years. They were instructed to cough vigorously and the produced expectorate was collected in a sterile plastic container as sputum. Sputum samples, collected during 8-30 to 11-00 A.M. at the school premises, were smeared on clean glass slides. Three smears were made from the non-transparent part of each sample. The slides were fixed immediately at the site of collection, and brought to the laboratory at Kolkata for staining and evaluation.

(b) Fixation and staining

The slides were semi-dried in air, and fixed for i) 30 minute in ethyl alcohol for Papanicolaou staining, ii) 20 min in buffered formalin (40% formaldehyde in 0.1M phosphate buffer, pH 7.4, 3:1, v/

v) for non-specific esterase staining and iii) 10 min in 10% formalin for Perl's Prussian blue reaction.

(c) Papanicolaou (Pap) staining for cytology

One slide was stained for cytology by Papanicolaou method (Hughes and Dodds, 1966). The staining mixture contained: i) Harris' hematoxylin (Sigma Chem, USA); ii) Orange G (Gurr, Germany); iii) EA 50 containing 0.5% solution of Light green (Gurr, Germany) in 95% alcohol, 0.5% alcoholic solution of Bismarck brown (Gurr, Germany), 0.5% alcoholic solution of Eosin yellow (Sigma Chem, USA), 200 mg of phosphotungstic acid (Sigma, USA) and 0.05 ml of saturated aqueous solution of lithium carbonate. Following staining the slides were dehydrated in graded ethanol, cleared in xylene and mounted in DPX mountant, and observed under light microscope (Leitz, Germany).

Observation and evaluation

The slides were examined thoroughly under light microscope (Leitz, Germany) at 400 and 1000x magnification for cytology (Grubb, 1994). All the cell types present in the sputum except squamous epithelial cells were taken into account. The sputum samples were considered as representative of the lower airways if either cylindrical epithelial cells or AM or both are observed. Children who had delivered non-representative samples were excluded from the study for further investigation.

(d) Staining for non-specific esterase (NSE)

NSE, a marker enzyme for macrophages, was localized by Fast Blue B method (Oliver et al., 1991). The reaction mixture contained 10 mg of alpha naphthyl acetate (Sigma Chem, USA), 100 mg of Fast Blue B (BDH, UK), 0.25 ml acetone, 20 ml phosphate buffer, pH 7.4. NSE-positive cells appeared golden brown in color.

(e) Perl' Prusian blue reaction for ferric iron

Perl's Prusian blue reaction was done in sputum of 205 control and 411 children of Delhi by the method of Pearse (1985). Iron (hemosiderin)-laden AM, known as siderophage, is suggestive of pulmonary hemorrhage. Staining was done using 2% potassium ferrocyanide (SD Fine Chem, India) and 2% hydrochloric acid (Merck, India) in iron-free distilled water. Iron-containing cells appeared blue under microscope

(f) Statistical analysis

All data are expressed as mean± standard deviation. The collected data were processed and analyzed in EPI info 6.0 and SPSS (Statistical Package for Social Sciences) software. Logistic regression analysis using generalized estimating equations (GEEs) was used to examine the relationship between cellular lung reaction and possible confounders such as RSPM levels. Spearman's rank test for continuous variables and Chi-square test for categorical variables were done. $P<0.05$ was considered as significant.

5.3 RESULTS

(a) Rate of acceptance of sputum samples

Altogether, 46 spontaneously expectorated sputum samples of control children (46% of total) and 35 from Delhi's children (25% of total) were discarded as they were not representative of the airways because of the absence of cylindrical epithelial cells and/or AM. Thus, more children in Delhi (75%) expectorated sputum samples than age- and gender-matched controls (54%, $p<0.05$). The findings of the remaining 166 samples (112 from Delhi and 54 from control) are presented in Table 5.1.

Table 5.1: Sputum cytology of the children

Cell type	**Control**(n=54)	**Delhi**(n=112)
Total cells/hpf	37.2± 9.9	58.8± 17.2*
Neutrophil		
%	82.4± 7.6	71.8±8.6*
per hpf	30.6± 2.8	42.2±4.6*
Eosinophil (%)		
%	1.7±0.8	3.2±1.2
per hpf	0.6± 0.3	1.9±0.7*
Lymphocyte (%)		
%	6.5±2.5	8.9±2.5*
per hpf	2.4±0.8	5.2±1.5*
AM (%)		
%	4.6±3.5	8.9±3.5*
per hpf	1.7±1.0	5.2±1.4*
Epithelial cells (%)		
%	4.8±1.6	7.2±3.6*
per hpf	1.8± 1.3	4.2±1.6*

**, p<0.05 compared with control*

(b) Total and differential counts of sputum cells

The total number of cells present in sputum per high power field (hpf) of microscope (10x eye piece x 40x objective) was 58.8 in Delhi against 37.2 in controls, establishing a significant rise of 58% in the former ($p<0.05$, Table 5.1). Thus, the sputum of Delhi's children was more cellular than that of controls.

Neutrophils were the major cell type in sputum of both control and Delhi's children, representing 82.4% and 71.8% of the total cells in these two groups respectively. Due to higher total cell count, however, the number of neutrophil per hpf was more in Delhi than in controls (42.2 vs. 30.6/hpf, $p<0.05$, Table 5.1; Fig. 5.1, 5.2).

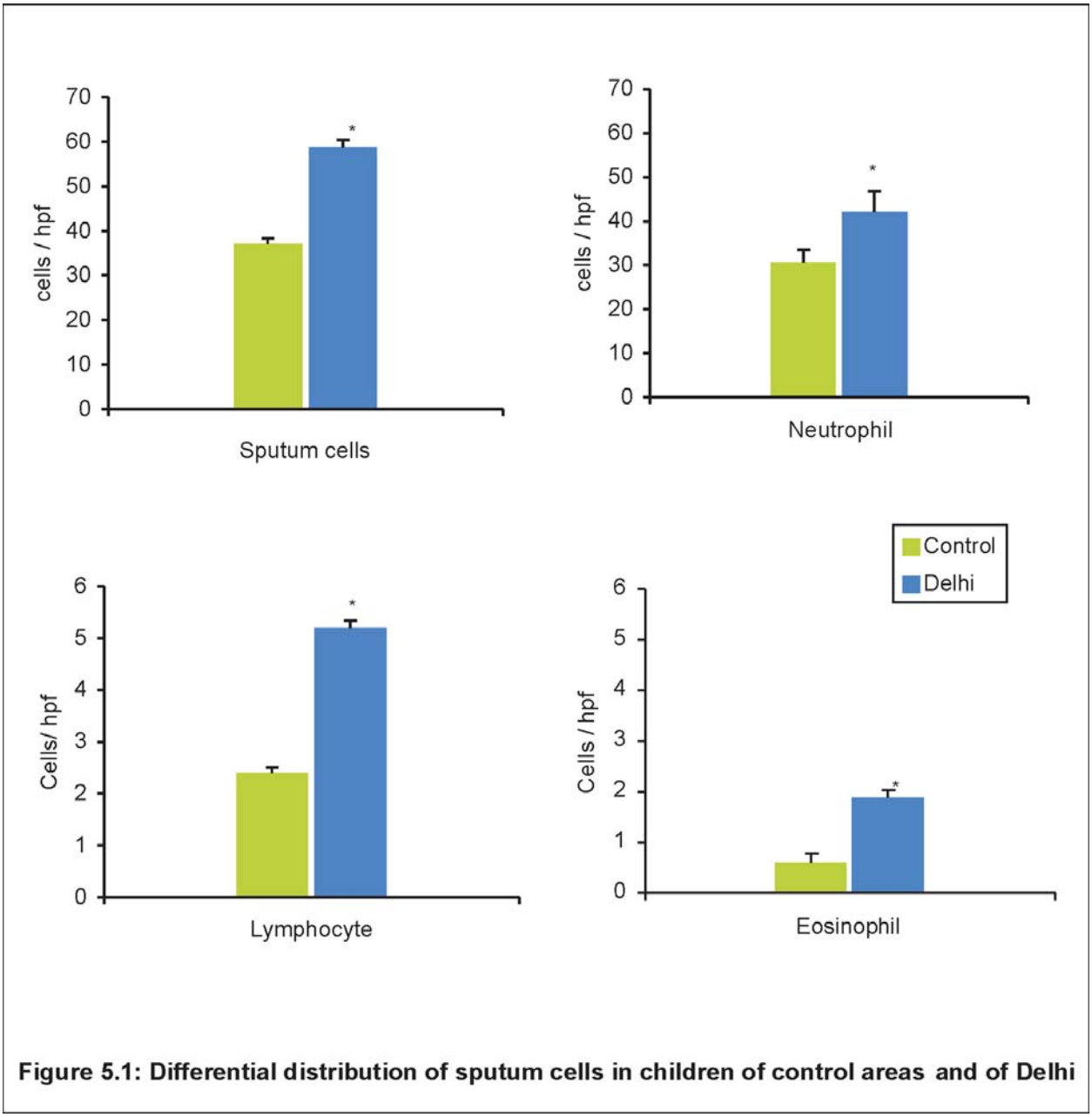


Figure 5.1: Differential distribution of sputum cells in children of control areas and of Delhi

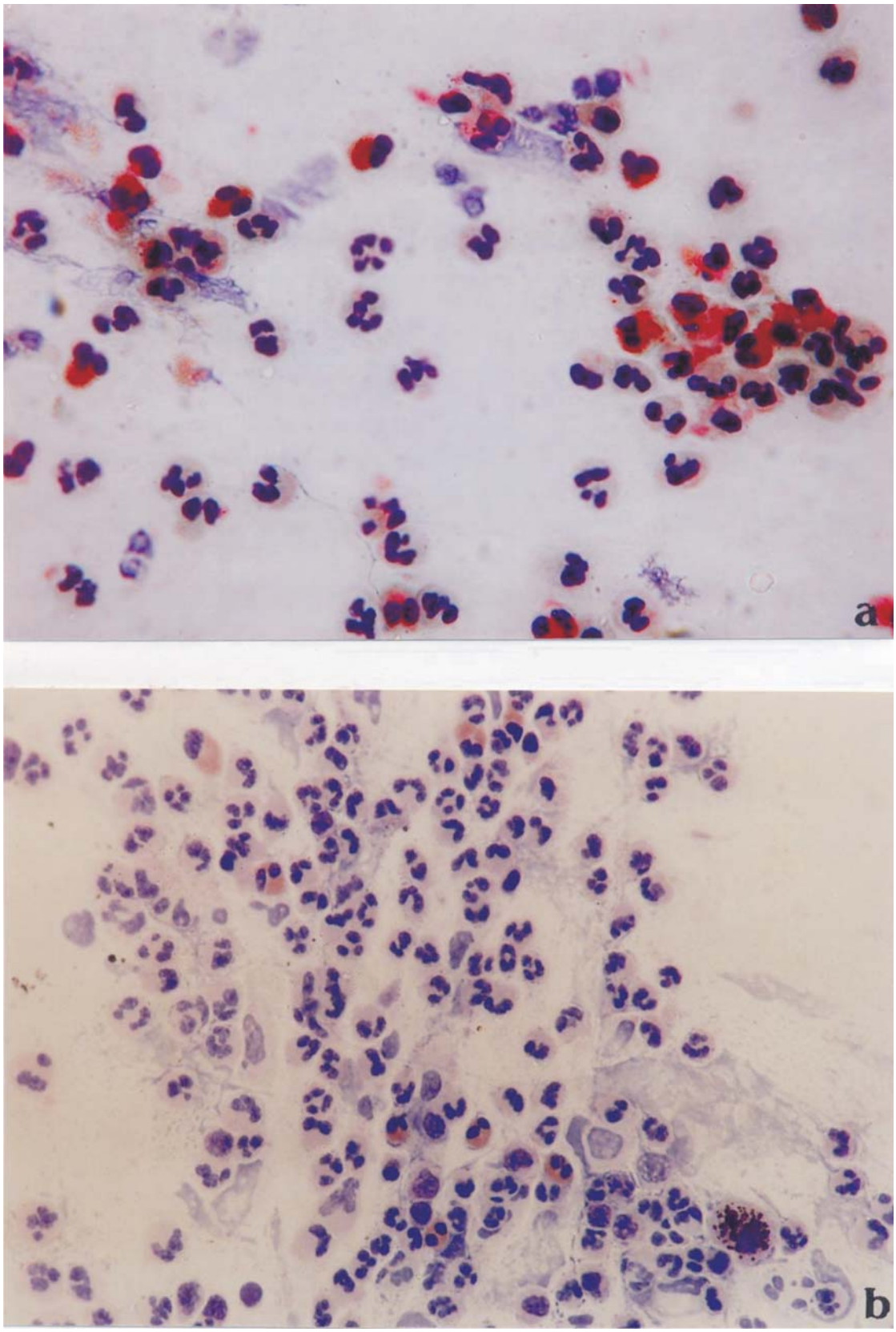


Figure 5.2: Sputum of children chronically exposed to Delhi's air pollution showing increase in the number of eosinophils (a) and neutrophils (b) indicating allergic and inflammatory reactions in the airways. Papanicolaou-stained, x400

Sputum samples of Delhi's school children also contained significantly greater percentage and absolute number ($p<0.05$) of eosinophils, lymphocytes, alveolar macrophages and airway cells such as basal and parabasal cells and ciliated columnar epithelial cells (Table 5.1, Fig. 5.3). Rise in the inflammatory cells like neutrophils and lymphocytes in sputum of Delhi's school children may suggest greater prevalence of pulmonary infection and inflammation in the city. The rise in eosinophil number in sputum of urban children may indicate underlying allergy and hypersensitivity response (Fig. 5.2). Marked rise in AM number with high particle load in these children signifies greater exposure to particulate pollution, because AMs represent the first line of cellular defense against inhaled particles and pathogens (Fig. 5.4, 5.5, 5.6, 5.7). Presence of basal and parabasal epithelial cells in clusters or sheets, that has been found in a number of sputa of children of Delhi, may indicate cellular damage to the airway walls.

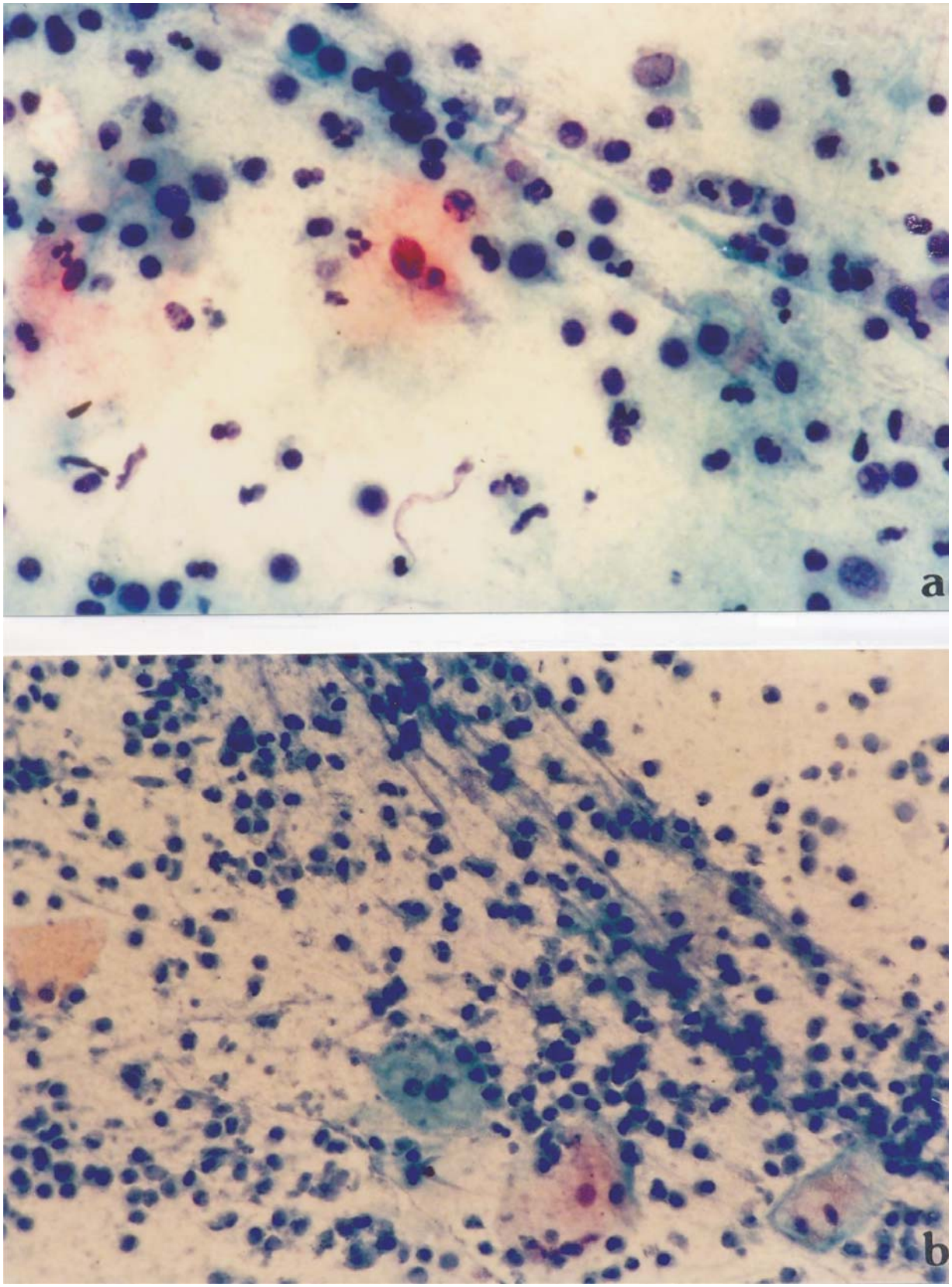
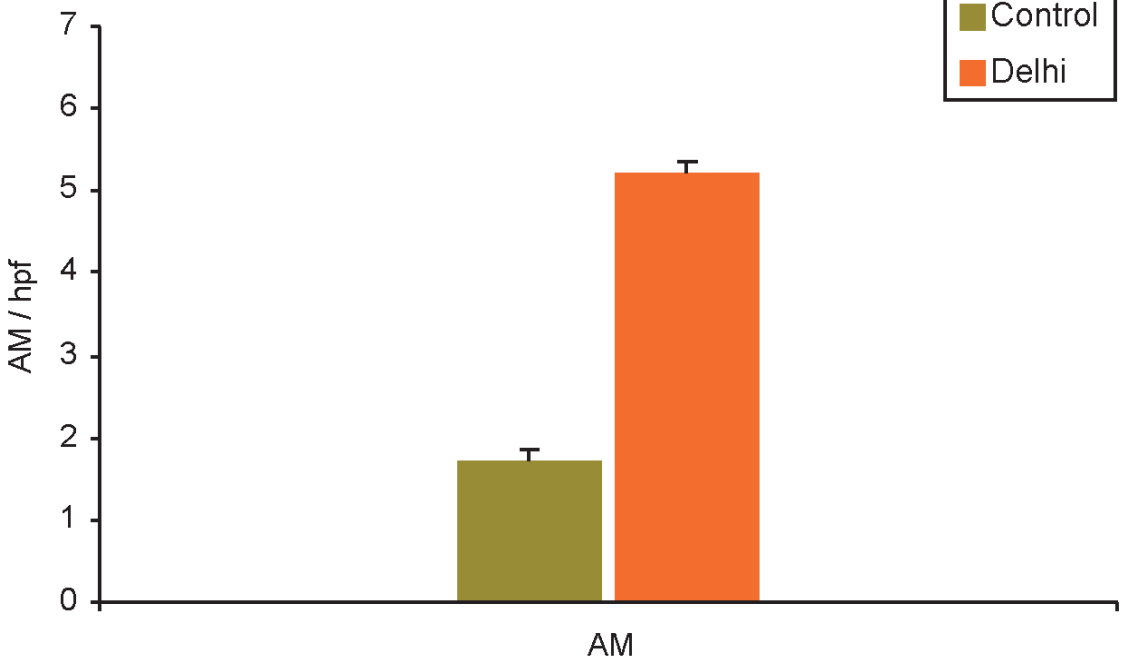


Figure 5.3: Photomicrographs of sputum of school children of Delhi (a, b) showing increase in the number of lymphocytes suggestive of respiratory viral infection. Papanicolaou-stained, x 400

Figure 5.4: Comparison of alveolar macrophage (AM/hpf) in children of control areas and of Delhi



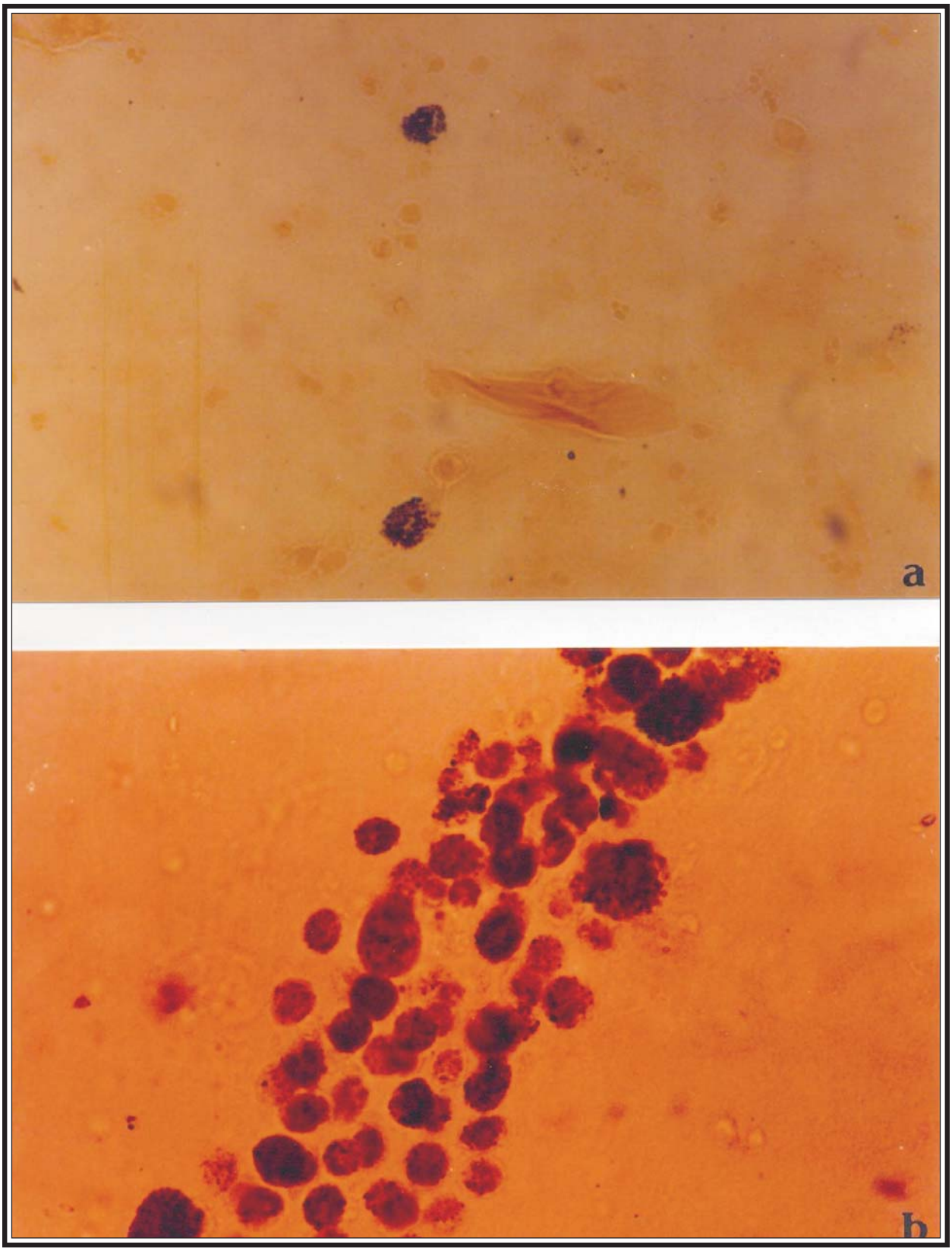


Figure 5.5: Photomicrographs showing particle-laden alveolar macrophages in sputum samples of control (a) and Delhi's school children (b). Note the remarkable increase in number and size of cells in sputum of the urban child. Non-specific esterase-stained, x 400

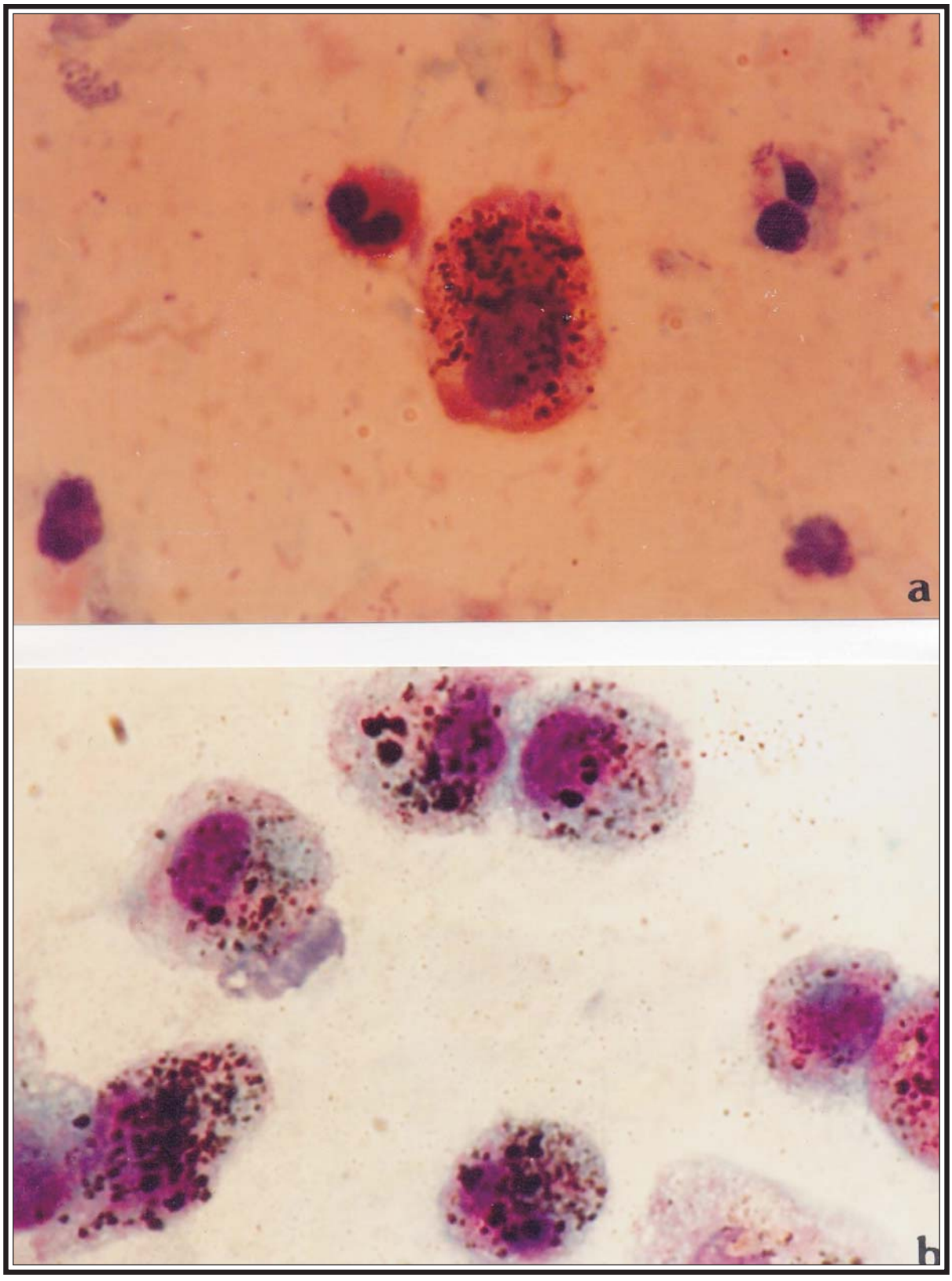


Figure 5.6: Sputum cytology of a 14-year old girl of Delhi (b) and age-, sex-matched control (a) showing abundance of particle-laden alveolar macrophages in former. Papanicolaou- stained, x 1000

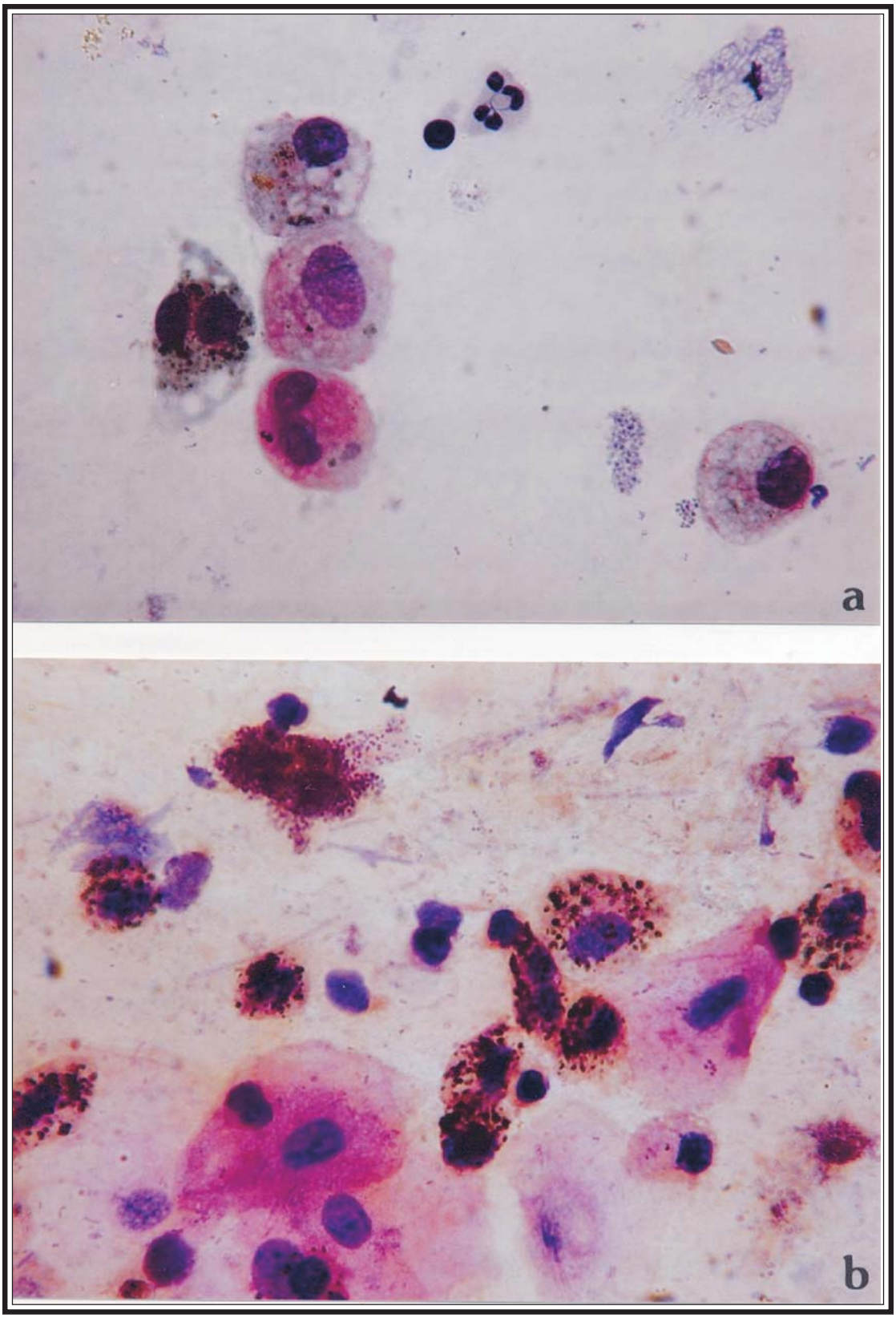


Figure 5.7: Comparison of sputum cytology between children of Delhi (b) and their matched rural control (a). Alveolar macrophages in the former are heavily loaded with particles suggesting greater exposure to particulate air pollution. Papanicolaou- stained, x 1000

(c) Association between particulate air pollution and cellular changes in sputum

The inflammatory changes in sputum of Delhi's children could be due to exposure to higher level of air pollution, because the changes were positively correlated with ambient PM_{10} level in Spearman' rank correlation test ($p<0.001$, Table 5.2).

Table 5.2: Spearman's rank correlation between PM_{10} level and sputum cell count

			Rho (r) value	p value
PM_{10}	with	Cells/hpf	0.643	<0.001
,,	with	Neutrophil/hpf	0.461	<0.001
,,	with	Eosinophil/hpf	0.644	<0.001
,,	with	Lymphocyte/hpf	0.462	<0.001
,,	with	Epithelial cells/hpf	0.448	<0.001
,,	with	AM/hpf	0.581	<0.001

On the other hand, a significant ($p<0.05$) negative correlation was found between total cells in sputum and FVC, FEF_{25-75} and PEFR values (Table 5.3). Likewise, AM number in sputum was negatively correlated with FVC and PEFR ($p<0.05$). It implies that the increase in inflammatory cell population in the airways following exposure to urban air pollution results predominantly in restrictive type of lung function deficits.

Table 5.3: Spearman's rank correlation test between sputum cell count and lung function

			Rho (r) value	p value
Total cells in sputum /hpf	with	FVC	-0.317	<0.05
"	with	FEV_1	-0.182	NS
"	with	$FEF_{25-75\%}$	-0.324	<0.05
"	with	PEFR	-0.241	<0.05
AM/hpf	with	FVC	-0.264	<0.05
"	with	FEV_1	-0.203	NS
"	with	$FEF_{25-75\%}$	-0.209	NS
"	with	PEFR	-0.364	<0.01

NS, not significant in statistical test

(d) Changes in the air way epithelial cells

Sputum samples of Delhi's children often displayed several qualitative and quantitative changes in airway epithelial cells. For example, goblet cell hyperplasia was found in 14.3% children against 3.7% of controls, and the difference was highly significant ($p<0.001$, Fig. 5.8). The change indicates hypersecrertion of mucous that could be a protective measure to contain inhaled pollutants (Fig. 5.9). Ciliocytophthoria, an indication of influenza virus infection in the airways was found in 4 (3.6%) samples against none in controls (Fig.5.10). Multinucleated columnar epithelial cells, suggestive of disregulation of cell division was found in 6.2% samples of Delhi's children compared with none in controls (Fig. 5.9). Metaplasia of airway epithelial cells was present in 16.1% in Delhi's children compared with 3.7% of controls (Fig. 5.11). Similarly, dysplasia of airway epithelial cells was present in 2 samples (1.8%) of Delhi against none in controls. (Fig. 5.12)

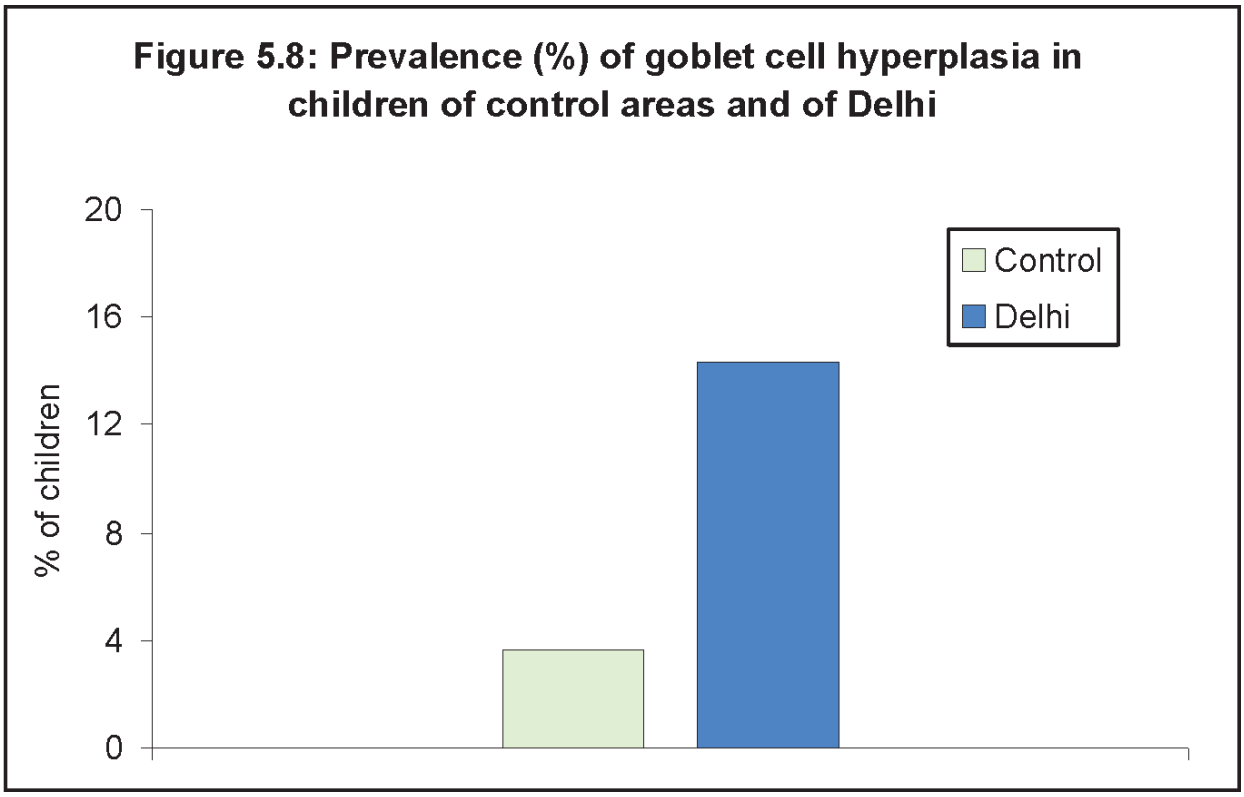


Figure 5.8: Prevalence (%) of goblet cell hyperplasia in children of control areas and of Delhi
20
16
12
8
4
0
% of children
Control
Delhi

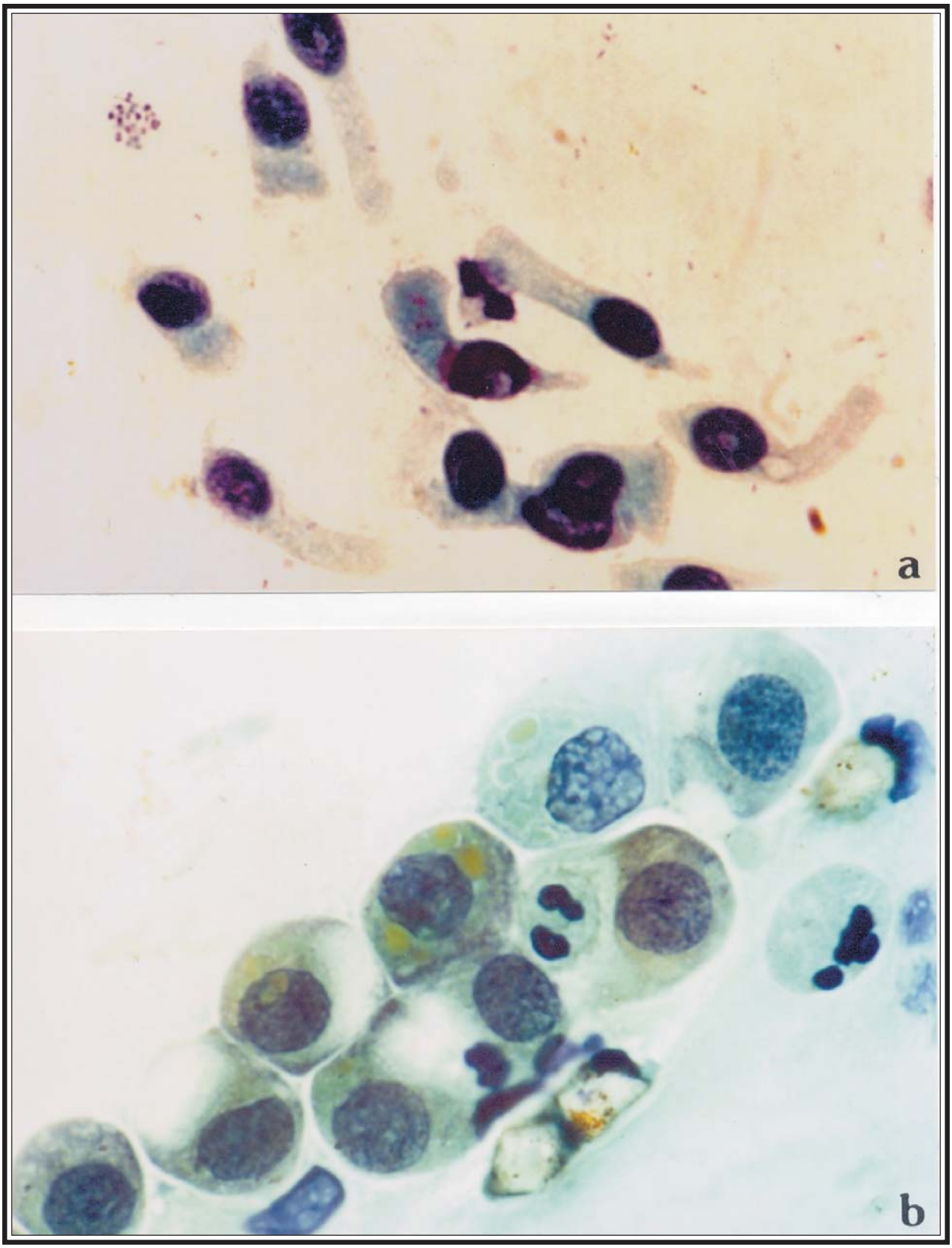


Figure 5.9: Photomicrograph of sputum of children of Delhi illustrating aggregates of columnar epithelial cells (a) suggesting airway injury and cluster of mucus producing goblet cells (b) indicating hyper production of mucus presumably to contain inhaled pollutants. Papanicolaou-stained, x 1000

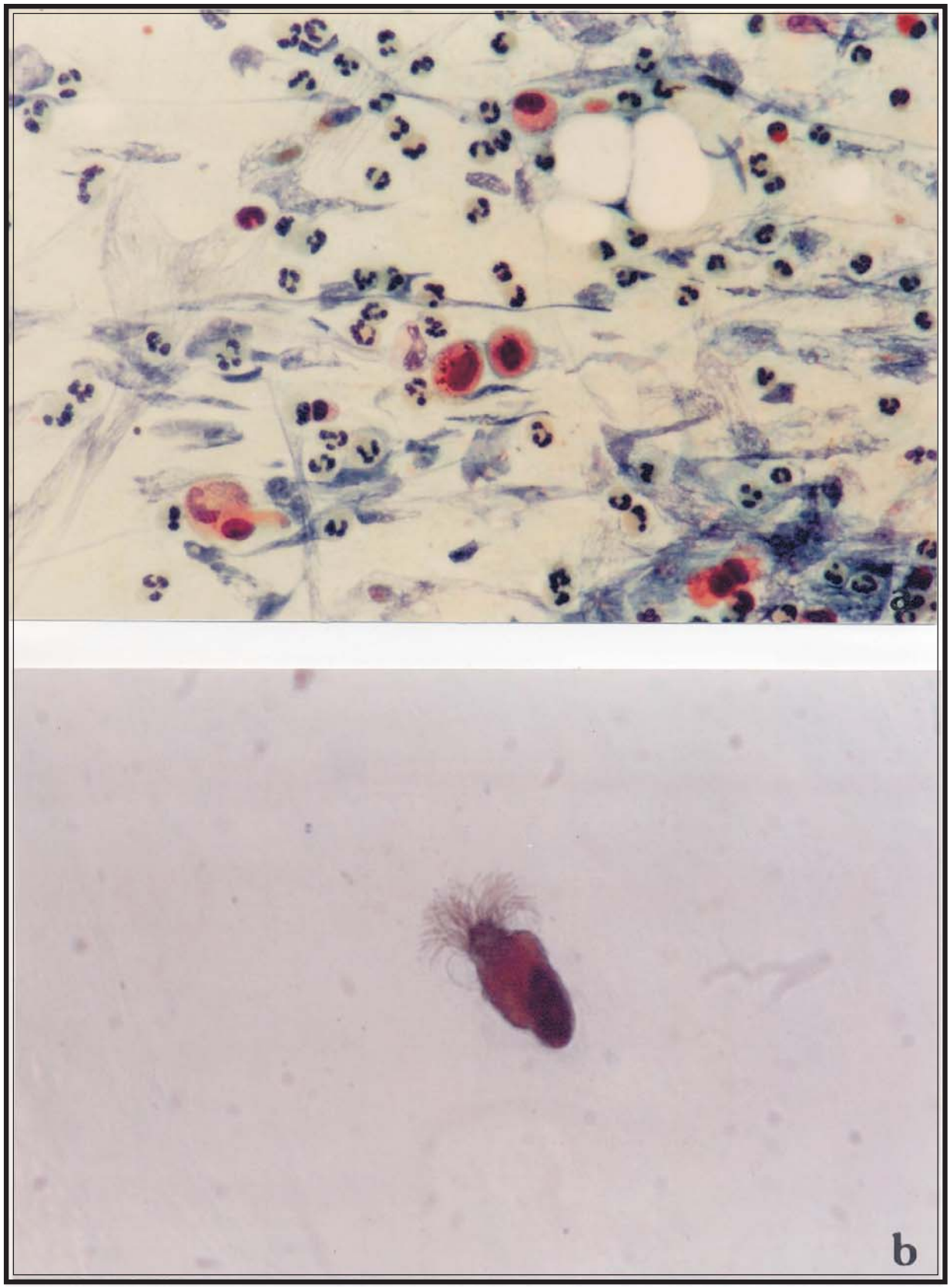
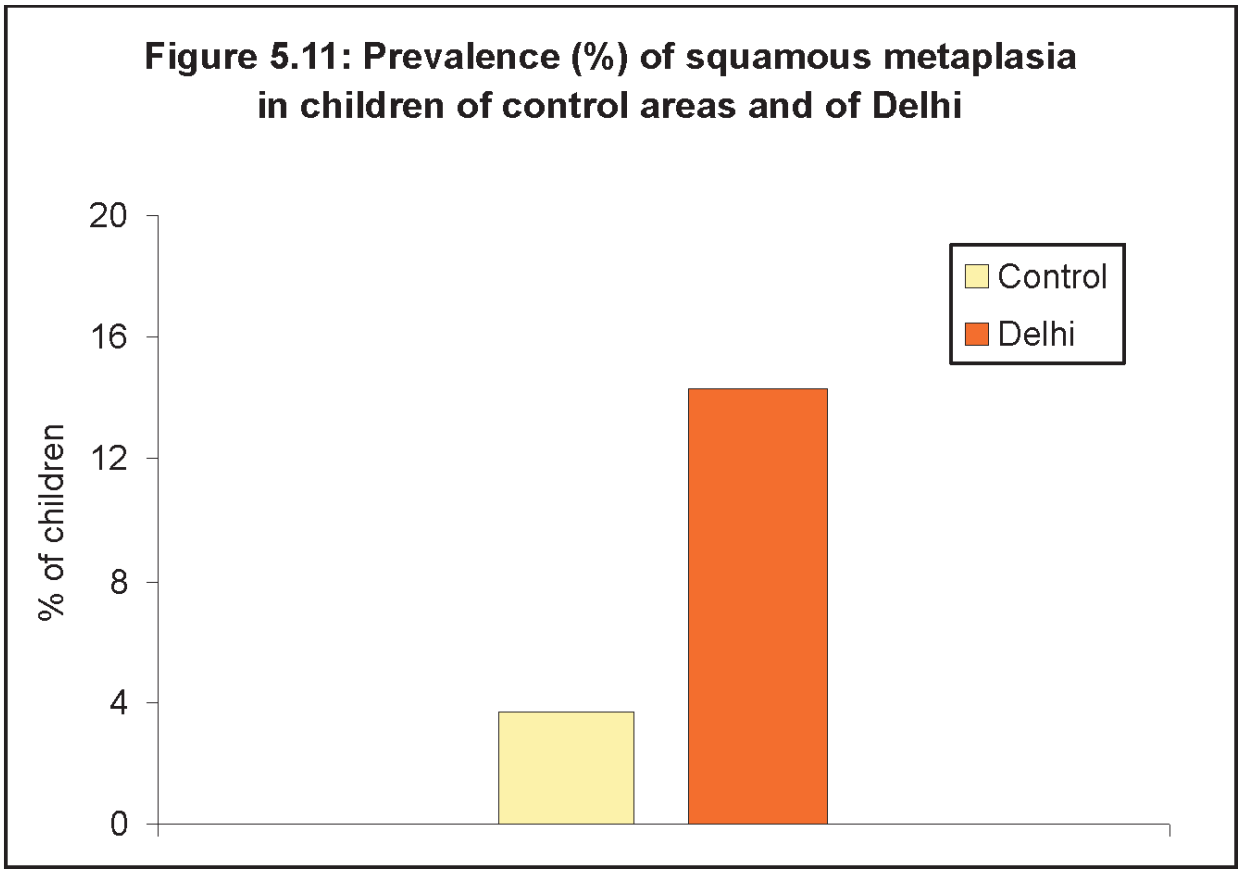


Figure. 5.10: Sputum cytology of school children chronically exposed to Delhi's pollution showing abnormal cells such as highly keratinized alveolar macrophages (a) and ciliocytopthoria (b) which is commonly associated with infection of influenza virus. Papanicolaou-stained, x 1000

Figure 5.11: Prevalence (%) of squamous metaplasia in children of control areas and of Delhi



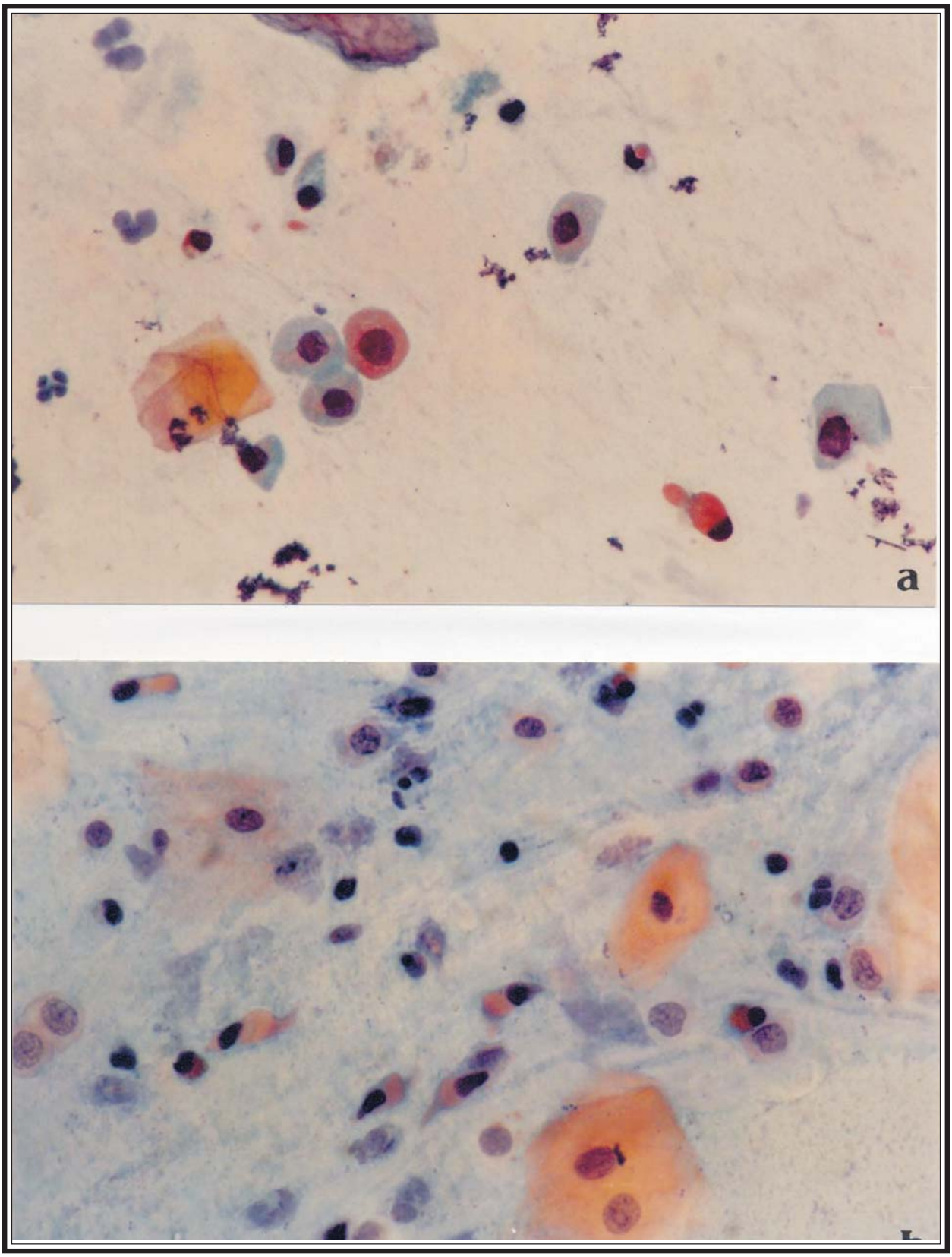
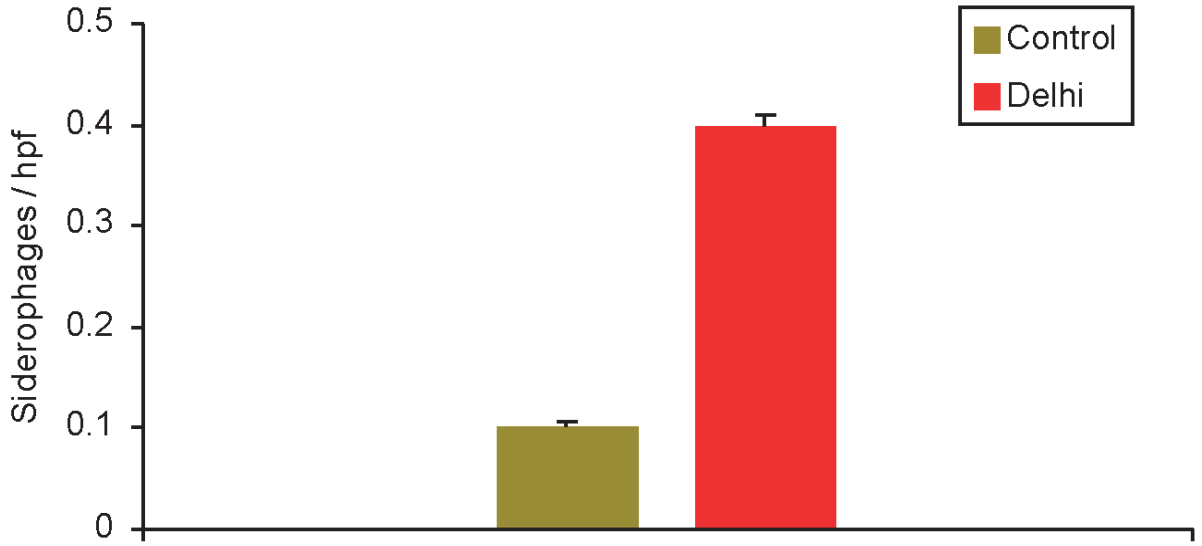


Figure 5.12: Sputum cytology of Delhi's children showing metaplastic changes in squamous epithelial cells of the airway tract (a) and exfoliation of ciliated columnar epithelial cells in clusters (b) suggesting damage to the airway walls. Papanicolaou-stained, x 1000

(e) Iron deposition in AM

Iron-laden AM (siderophages) were rare in control children, as less than 5% (average 3.7%) of AM present in sputum contained Prussian blue-positive hemosiderin iron. Moreover, the intensity of staining, as evaluated by subjective grading, was low (1+). In contrast, 8% of the AM present in sputum of the children of Delhi contained hemosiderin iron, and the intensity of Prussian blue staining was moderate (2+). On an average, 0.4 ± 0.1 (SD) siderophages were recorded per high power field of microscope (400 x magnification) in sputum of Delhi's children against 0.1 ± 0.05 per high power field in control children, indicating a 4-fold rise in the number of siderophages in the former group ($p<0.001$; Fig. 5.13, 5.14).

Figure 5.13: Number of iron-laden macrophages (siderophages/hpf) in sputum samples of children of Delhi



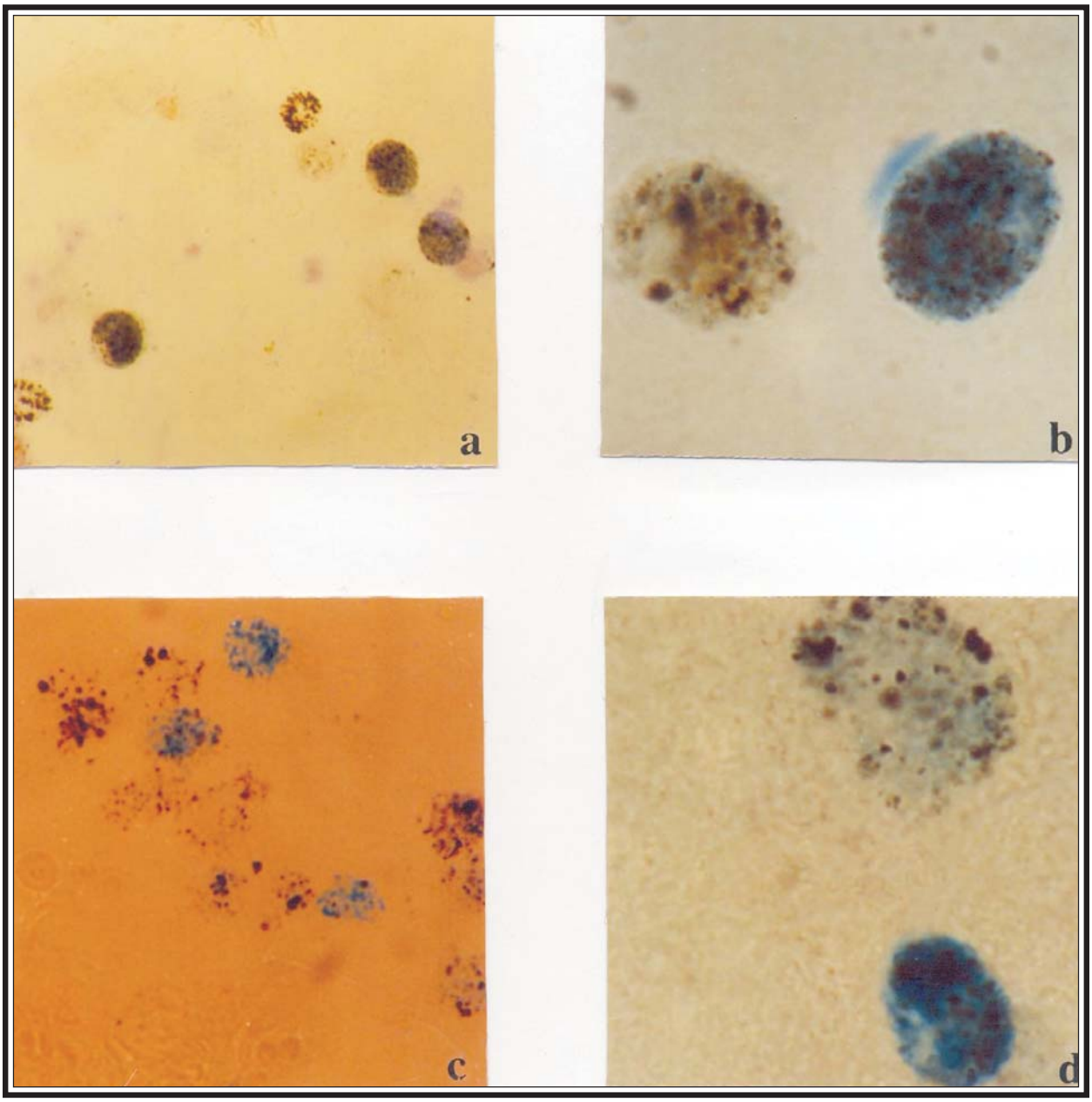


Figure 5.14: Photomicrographs showing iron-laden alveolar macrophages (siderophages) in sputum. Iron deposition in AM is negligible in control children (a) but fairly abundant in student of a school in Central Delhi (b, c, d). Perl's Prussian blue reaction, x 400 (a,c), x 1000 (b,d)

5.4 FINDINGS

1. Sputum samples from 250 school children aged between 13-15 years, 100 (boys 65, girls 35) from control group and 150 from Delhi (boys 98, girls 52) were analyzed. Altogether, 46 sputum samples of control children (46% of total) and 35 from Delhi's children (25% of total) were discarded because they were not representative samples of the airways and alveoli. Finally, 54 samples from control and 125 from Delhi's school children were cytologically and cytochemically analyzed and the results were compared.

2. Microscopical analysis revealed that the sputum samples of Delhi children were more cellular than that of controls: 58.8 cells/hpf were found in the sputum samples of Delhi's children in contrast to 37.2 in the control group. Compared with control children, therefore, Delhi's children had 58% more cells in their sputum ($p<0.05$).

3. The major cell type in sputum of both control and Delhi children were neutrophils, the number being higher in Delhi. The number of neutrophils per hpf was 42.2 in Delhi and 30.6 in the control group ($p<0.05$), which may suggest greater prevalence of pulmonary infection and inflammation in the city.

4. The percentage of eosinophils was also higher in the sputum samples of Delhi's children (3.2% vs. 1.7%) than in control. The rise in eosinophil number is an indication of underlying allergy and hypersensitivity response. Therefore, a greater prevalence of airway allergy may be envisioned among the school children of Delhi.

5. The mean number of alveolar macrophages (AM) per high power field in Delhi's children was 5.2 in contrast to 1.7 AM per hpf in control. Hence, school children of Delhi had 3.1 times more AM in their sputum. Marked increase in AM number signifies greater exposure to particulate pollution as AM represents the first line of cellular defence against inhaled pollutants.

6. Sputum of Delhi's children contained 4-times more iron-laden macrophages (siderophages) than controls (0.4 vs. 0.1 siderophage per high power field, $p<0.001$). Abundance of siderophages in lungs may indicate covert pulmonary hemorrhage among a section of school-going children of Delhi.

7. Changes in the sputum cytology among the school children of Delhi positively correlated with ambient PM_{10} level in Spearman's Rank Correlation, suggesting a close relationship between chronic exposure to Delhi's particulate pollution and cellular changes in the lung ($p<0.001$).

8. Negative correlation ($p<0.05$) was found between total cells in sputum and spirometric lung measurements like FVC, FEF_{25-75} and PEFR values, thereby indicating that the increase in inflammatory cell population in the airways plays a key role in the development of restrictive type of lung function deficits, and small airway obstruction.

9. In essence children chronically exposed to high level of ambient air pollution in Delhi are at a higher risk of inflammation and covert hemorrhage in the lungs that may be lead to lung function deficits.

CHAPTER-6.0

HEMATOLOGICAL AND VASCULAR CHANGES ASSOCIATED WITH AIR POLLUTION EXPOSURE

6.1 INTRODUCTION

Chronic exposures to high particulate air pollution, particularly in the ultrafine range, are known to provoke alveolar inflammation that release mediators capable of exacerbating lung disease and increased blood coagulability in susceptible individuals (Seaton, 1995). The heart can be affected by air pollution exposure, because animal studies by Godleski and coworkers have shown that inflamed lung, as evidenced by neutrophil accumulation in bronchoalveolar lavage fluid, release mediator that alter the autonomic nervous system control of cardiac rhythm. Subsequent studies in human subjects have indicated a rise in pulse rate in association with exposures to PM_{10} (Pope et al., 1999a). Air pollution is associated with lower heart rate variability, implying poor autonomic control (Liao et al., 1999; Pope et al., 1999b). Impairment of autonomic control of the blood has also been reported in association with air pollution exposure (Gold et al., 2000), and PM2.5 has been implicated for these changes (Liao et al., 1999).

Cropper et al., (1997) conducted a pioneering, time-series study on the impact of particulate air pollution of Delhi on daily mortality in early 1990's.They found a significant relationship between particulate air pollution and daily deaths from respiratory and cardiovascular problems.

Pande and his co-workers (2002) conducted a 2-year (January 1997-December 1998) time-series analysis in Delhi. Emergency room visits for acute asthma, acute exacerbation of chronic obstructive airway disease, and acute coronary events at All India Institute of Medical Sciences (AIIMS) increased by 21.3%, 24.9% and 24.3% respectively on account of higher than acceptable level of air pollutants (CO, NOx, SO_2). It was concluded that there is a considerable burden of cardiopulmonary diseases in Delhi due to high level of ambient air pollution (Pande et al., 2002).

Studies on laboratory animals and in human volunteers documented abnormal red cell, neutrophil and platelet levels (Salvi et al., 1999), increase in blood viscosity (Schwartz, 2001), and changes in the number of T-lymphocytes, B-lymphocytes, and NK cells (Salvi et al., 1999) in response to air pollution exposure. In addition, neurotoxicity (Anderson et al., 1983; Kilburn, 2000), change in antioxidant defense of the body (Georgieva et al., 2002), and genotoxicity at the level of chromosomes (Zhang et al., 2005) and DNA (Eastman and Barry, 1992; Moller and Wallin, 1998; Don Porto et al., 2001) have been reported.

6.2 METHODOLOGY

(a) Clinical examination and blood pressure measurement

A physician clinically examined the children for general health problems at the school premises. Arterial blood pressure (BP) was measured in 1808 school children (1082 in Delhi and 726 in control) by a sphygmomanometer (Fig. 6.1). An inflatable cuff with a meter attached was placed around the child's arm over the artery, while the child was seated. Systolic (the force that blood exerts on the artery walls as the heart contracts to pump out the blood) and diastolic (force as the

heart relaxes to allow the blood to flow into the heart) blood pressures were expressed in millimeters of mercury (mm Hg). In general, two BP measurements were done at a minimum of two minutes' interval. BP reading tends to decrease with repeated measurement because of an accommodation of the child to the measurement procedure or relaxation (Sinaiko, 1996).

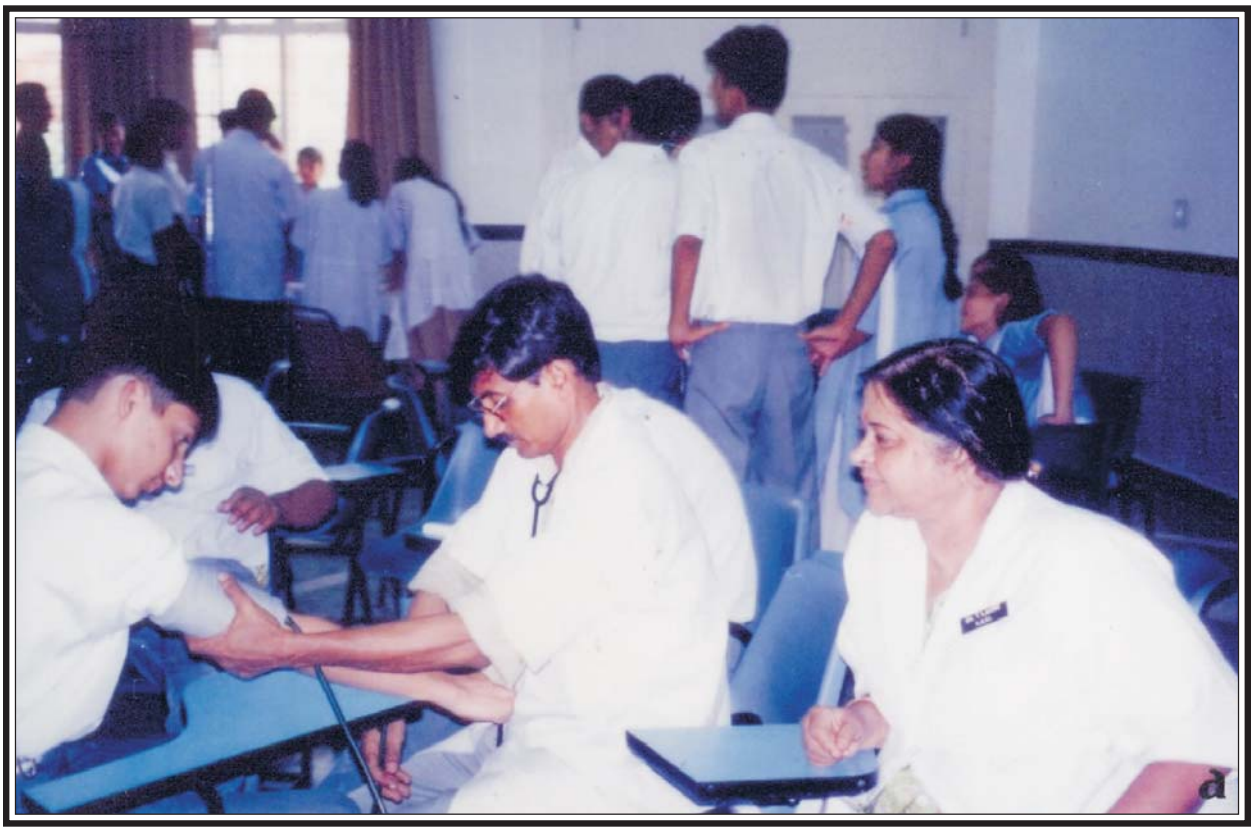


Figure 6.1: Measurement of blood pressure of the children at Mahaveer Senior Secondary School, G.T. Karnal Road, New Delhi

(b) Diagnosis of hypertension

A child's blood pressure is normally lower than an adult's. Children are at risk for hypertension if they exceed the 95th percentile of the age-specific height percentile according to the guideline of National Heart, Lung and Blood Institute's Task Force on Blood Pressure Control in Children (Task Force on High Blood Pressure in Children and Adolescent, updated in 1996, Table 6.1, 6.2).

Table 6.1: Blood pressure of children in relation to height

	Age(yr)	Height Percentile for Boys*				Height Percentile for Girls			
		5 th	25 th	75 th	95 th	5 th	25 th	75 th	95 th
Systolic	3	104	107	111	113	104	105	108	110
	6	109	112	115	117	108	110	112	114
	10	114	117	121	123	116	117	120	122
	13	121	124	128	130	121	123	126	128
	16	129	132	136	138	125	127	130	132
Diastolic	3	63	64	66	67	65	65	67	68
	6	72	73	75	76	71	72	73	75
	10	77	79	80	83	77	77	79	80
	13	79	81	83	84	80	81	82	84
	16	83	84	86	87	83	83	85	86

** The height percentile was determined with standard growth curves*
Ref. Task Force on High Blood Pressure in Children and Adolescent, updated in 1996

Table 6.2: Diagnosis of hypertension in children according to Task Force on High Blood Pressure in Children and Adolescent, 1996

Diagnosis	Systolic /diastolic blood pressure
Normal	Below 90th percentile
High normal	90-95th percentile
Hypertension	>95th percentile

(c) Blood cell counts

Blood samples (100 µl) were collected from 117 schoolchildren (control 46, Delhi 71) aged 13-15 years after informed consent of the parent/class teacher by finger prick by sterile, 21-gauge needle. Blood smears were prepared on glass slides, and routine hematology (hemoglobin measurement, red blood cell, white blood cells and platelets counts) was done following the procedures of Dacie and Lewis (1975). Differential counts of WBC and examination of blood cell morphology were done from Leishman-stained smears under light microscope (Leitz, Germany) following standard procedure (Dacie and Lewis, 1975).

(d) Statistical analysis

The results, expressed as mean ± standard deviation (SD), were statistically analyzed by Student's 't' test and $p<0.05$ was considered as significant.

6.3 RESULTS

(a) Prevalence of hypertension

Arterial blood pressure was measured in 1082 school children of Delhi (boys 604, girls 478) and 726 of rural control (boys 422, girls 304). Overall, hypertension was found in 6.2% participants of

Delhi (67/1082), while only 2.1% of control children (15/726) had hypertension (Table 6.1, Fig. 6.2). The difference in the proportion of hypertensive children in rural and urban areas was highly significant (p<0.001). Hypertension was more prevalent among girls than the boys both in Delhi and in control group. For instance, 7.9% of Delhi's girls (38/478) had hypertension compared with 4.8% (29/604) of the boys (Table 6.3). Similarly, 2.3% of control girls (7/304) had hypertension compared with 1.9% (8/422) of the boys. The difference in hypertension prevalence between girls and boys was statistically significant (p<0.05) in Delhi, but not in control group. Quite contrary to hypertension, prehypertension was found to be more in boys than in girls of Delhi (31.8% vs. 26.4%; Table 6.3, Fig. 6.3).

Figure 6.2: Prevalence (%) hypertension in girls and boys of control areas and of Delhi

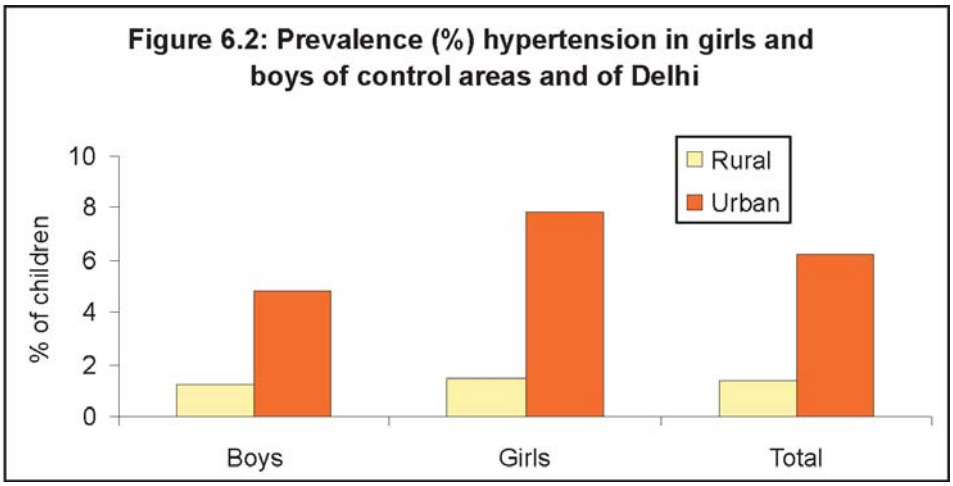
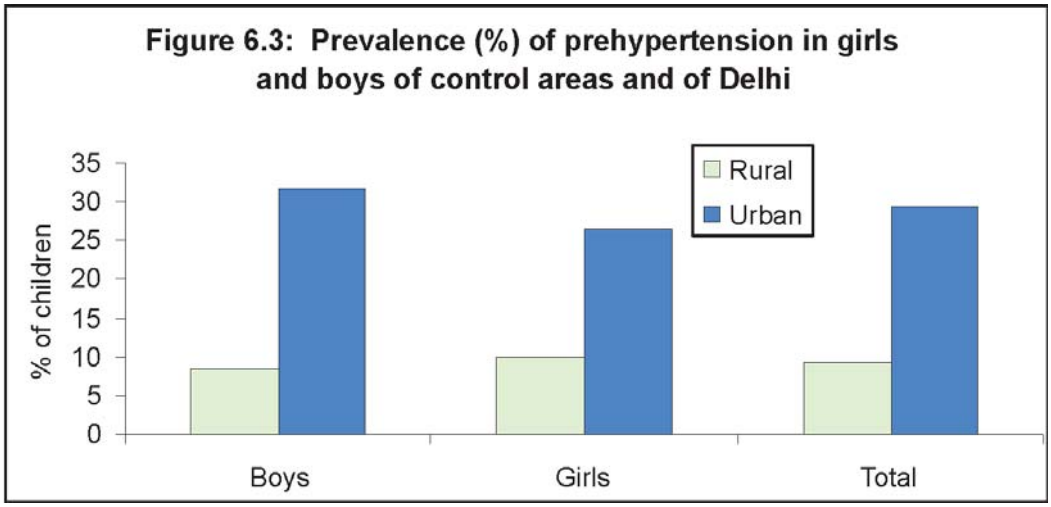


Table 6.3: Prevalence (%) of pre-hypertension and hypertension in school children aged 9-17 years

	Boys		Girls		Total	
	Control (n=422)	Delhi (n=604)	Control (n=304)	Delhi (n=478)	Control (n=726)	Delhi (n=1082)
Pre-hypertension	8.6	31.8*	9.8	26.4*	9.3	29.4*
Hypertension	1.9	4.8*	2.3	7.9*	2.1	6.2*

Figure 6.3: Prevalence (%) of prehypertension in girls and boys of control areas and of Delhi

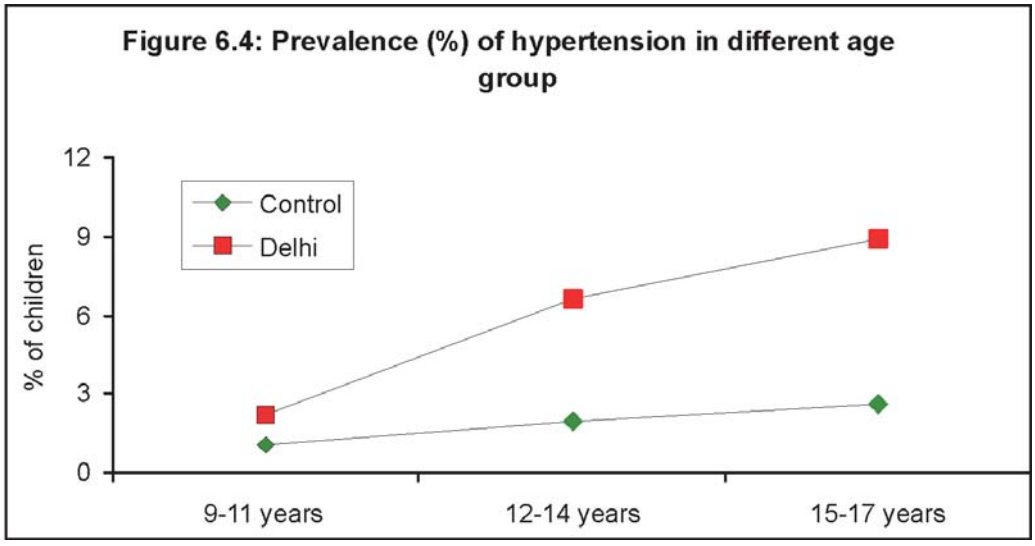


The prevalence of hypertension increased progressively with age. Highest prevalence was recorded in children aged 15-17 years both in rural (2.6%; 8/304) and urban settings (8.9%; Table 6.4, Fig. 6.4).

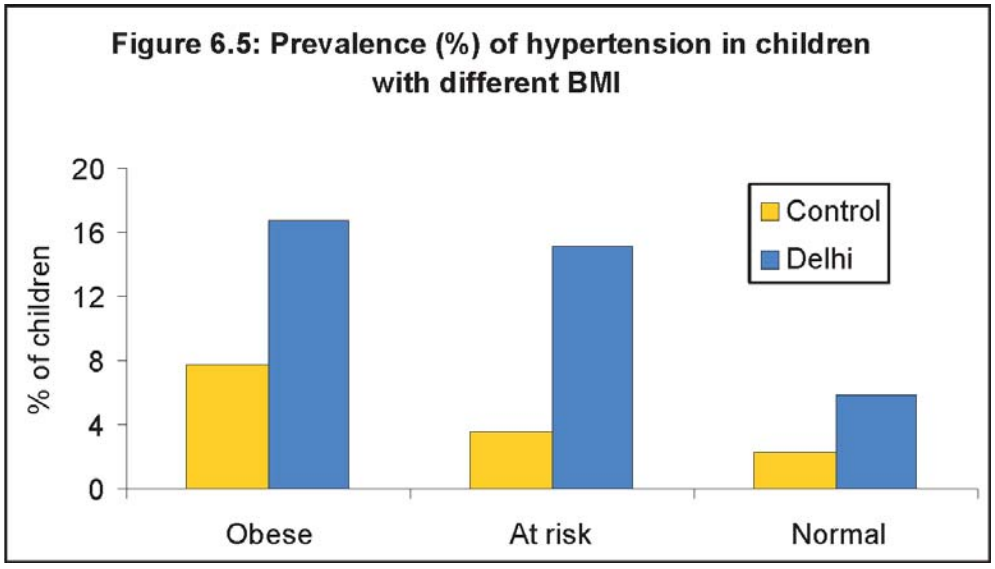
Table 6.4: Prevalence (%) of hypertension in school children

	Control(n= 726)	Delhi(n=1082)
Gender		
Boys Girls	1.9 2.3	4.8* 7.9*
Age (yr)		
9-11 12-14 15-17	1.1 2.0 2.6	2.2* 6.6* 8.9*
BMI		
Underweight Normal At risk Obese	0 2.3 3.6 7.7	1.8* 5.9* 15.1* 16.7*
Socio-economic status (SES)		
Low Medium High	1.3 1.8 3.7	5.8* 5.1* 7.0*

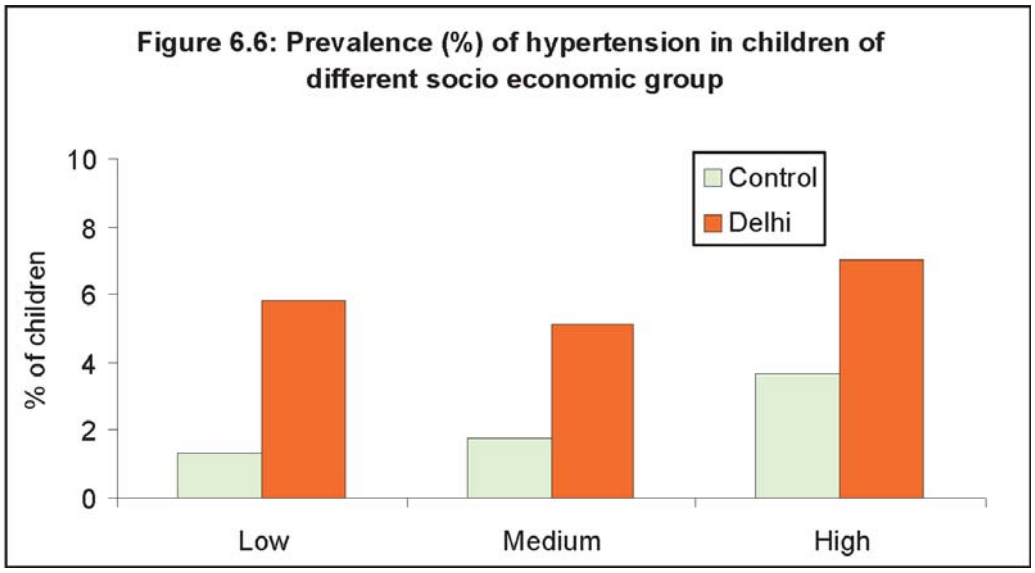
**, p<0.05 compared with control*



BMI was positively associated with the prevalence of hypertension. The prevalence was least (1.8%) in underweight children and highest (16.7%) in obese group (Table 6.4; Fig. 6.5).



Children from high SES had highest prevalence of hypertension (Table 6.4; Fig. 6.6). However in Delhi children from medium SES showed lower prevalence of hypertension (5.1%) than that of children from low SES (5.8%).



(b) Hematological changes

Hemoglobin and total count of RBC, WBC, and platelets

Hematological studies were conducted in 46 control and 71 children of Delhi. The results shown in Table 6.5. Increase in hemoglobin concentration and platelet count correlated significantly with PM_{10} level (rho values –0.295 and 0.354 respectively, $p<0.001$), while correlation between PM_{10} level and other hematological changes were not significant in Spearman's rank correlation test ($p>0.05$).

Table 6.5: Hematological values of the school children

Parameter	Control (n=46)	Delhi(n=71)
Hemoglobin (g/l),	12.2 ± 0.6	13.2±0.7
RBC (x106/ µl)	4.1±0.4	4.5±0.6
WBC (x103/ µl)	5776± 521	6682± 973
Platelet x106/ µl	1.9±0.8	2.2± 0.6

Results are expressed as mean ± S.D ; none of the hematological changes in Delhi's children was statistically significant (p>0.05)

Differential count of WBC showed significantly increased number of monocytes and basophils in peripheral blood of the children of Delhi when compared with that of controls (p<0.05; Table 6.6).

Table 6.6: Absolute numbers of leukocytes in peripheral blood

Cell type	Control (n=46)	Delhi(n=71)
Neutrophil / µl	3228± 322	3616±432
Lymphocyte / µl	2254± 208	2687±218
Monocyte / µl	112± 34	146± 37*
Eosinophil / µl	132± 32	153± 64
Basophil /µl	24± 12	70± 34*

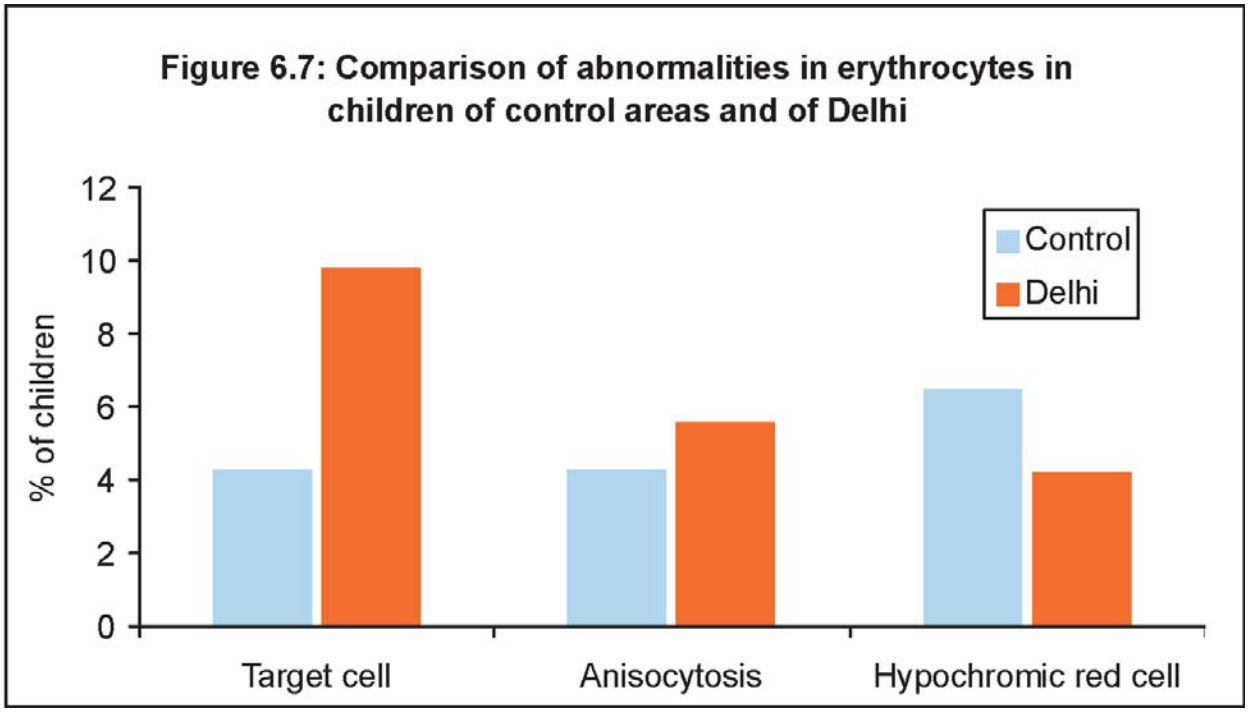
*Results are expressed as mean ± SD; *, p<0.05 compared with respective control value in Student's 't' test*

The children of Delhi had greater prevalence of several morphological abnormalities in blood cells. For example, 9.8% had abundance of 'target' cells in their peripheral blood compared with 4.3% of controls (p<0.001; Table 6.7; Fig. 6.7). Target cells are erythrocytes with higher surface-to-volume ratio. Their presence in circulation in excess signifies liver problem. It implies that a number of the Delhi's children might have liver problem (Fig. 6.8). Children of Delhi and the control group had similar frequencies of aniso-poikilocytosis (changes in red cell shape and size), but the urban subjects had lesser prevalence of hypochromic (RBC with lowered hemoglobin content) cells. The schoolchildren of Delhi had greater prevalence of giant platelets in circulation (5.9% vs. 1.5%, p<0.05) indicating platelet activation (Fig. 6.9). Higher prevalence of circulating immature neutrophil (11.3 vs. 6.5%, p<0.05) and toxic granulation in neutrophil (21.0% vs. 8.7%, p<0.05; Table 6.7; Fig. 6.10) were found in Delhi's school children, suggesting greater risk of infection and inflammation.

Table 6.7: Prevalence (%) of abnormal cell types in peripheral blood

Cell type	Control (n=46)	Delhi(n=71)
Changes in RBC		
'Target' cell	4.3	9.8*
Anisocytosis	4.3	5.6
Poikilocytosis	6.5	5.6
Hypochromic RBC	6.5	4.2*
Changes in WBC		
Toxic granulation in neutrophil	8.7	21.0*
Metamyelocyte/band cell >20%	6.5	11.3*
Change in Platelet		
Giant platelets	1.5	5.9*

**, p<0.05 compared with respective control value in Chi-square test*



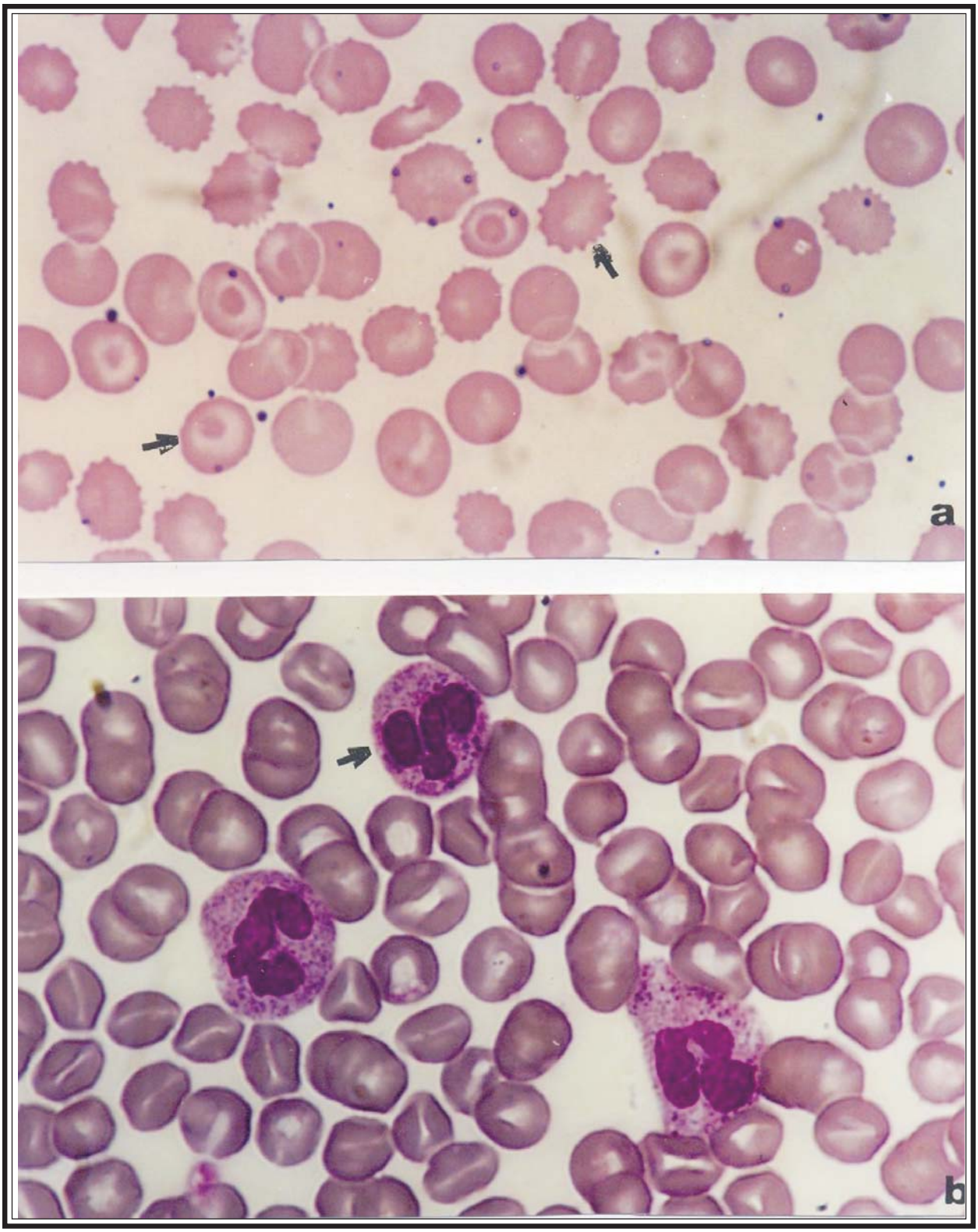


Figure 6.8: Photomicrographs of peripheral blood smear of Delhi's school children showing poikilocytosis and abundance of 'target' cells (a) along with toxic granulation in neutrophils (b). Leishman's stained x 1000

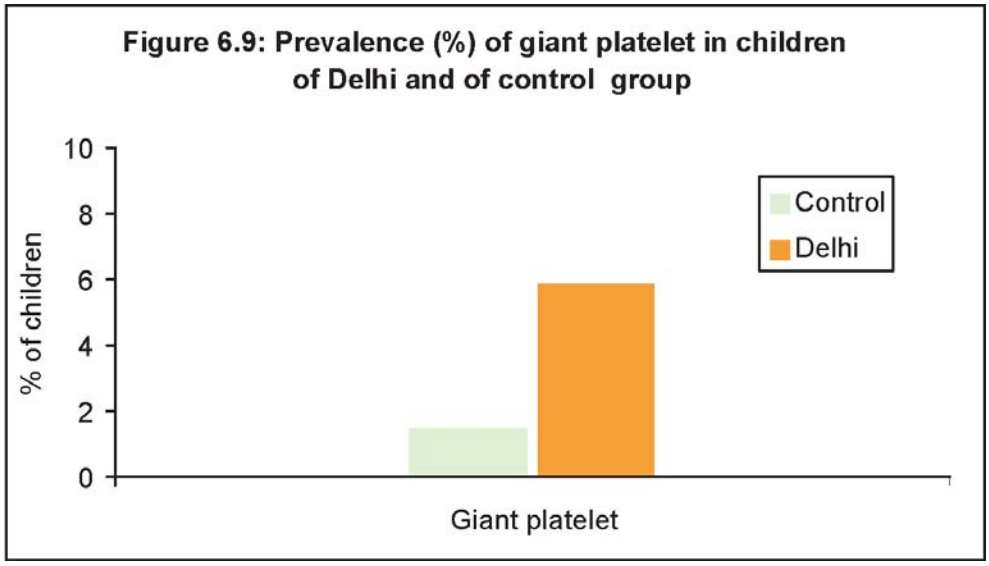


Figure 6.9: Prevalence (%) of giant platelet in children of Delhi and of control group

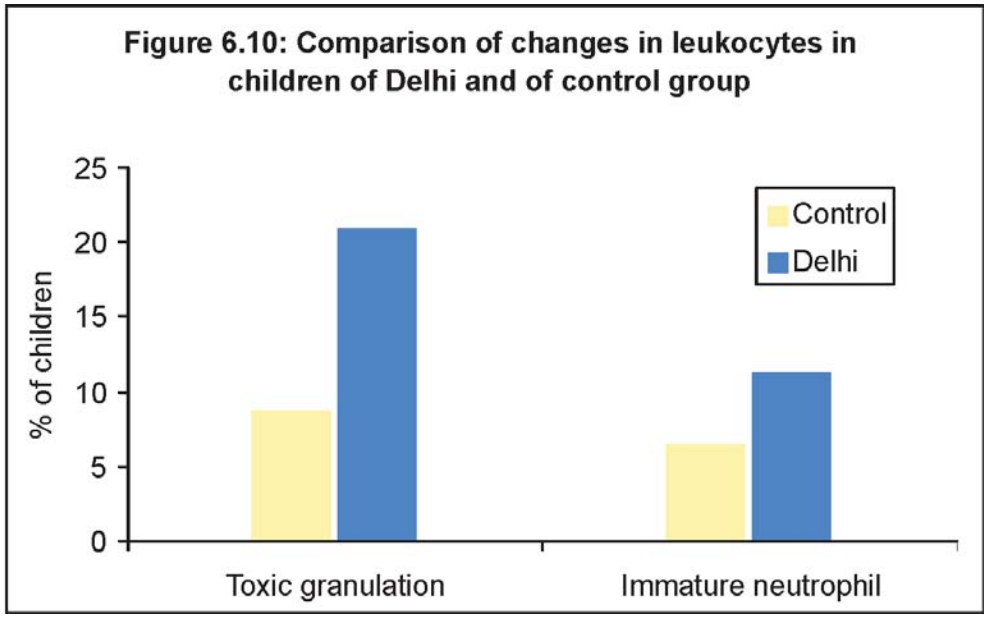


Figure 6.10: Comparison of changes in leukocytes in children of Delhi and of control group

6.4 FINDINGS

1. Arterial blood pressure was measured in 1082 school children of Delhi and 726 of controls. The prevalence of hypertension in children was 6.2% in Delhi compared with 2.1% in control. Thus, Delhi's school children have 3-times more incidence of high blood pressure.

2. Hypertension was more prevalent among girls than the boys: 7.9% of Delhi's girls had hypertension against 4.8% of boys. The prevalence of hypertension increased progressively with age, highest being in the age group of 15 – 17 years.

3. BMI was positively associated with hypertension: the prevalence of hypertension was lowest (1.8%) in underweight children and highest (16.7%) in obese group.

4. The absolute numbers of monocytes and basophils were significantly ($p<0.05$) increased in peripheral blood of Delhi's children as compared to that of control.

5. Rise in hemoglobin concentration and platelet number in Delhi's children were positively correlated with PM_{10} levels.

6. Examination of peripheral blood smears revealed abundance of 'target' cells in 9.8% of Delhi's children against 4.3% of controls, implying a greater risk of liver problem in the former.

7. Higher prevalence of toxic granulation in neutrophils (21.0% vs. 8.7%) and circulating immature neutrophils (11.3% vs. 6.5%) was found among the children of Delhi, which suggests greater risk of infection and inflammation.

8. In essence school children of Delhi had three times more prevalence of hypertension along with several quantitative and qualitative in peripheral blood that indicates greater possibility of infection and inflammation.

CHAPTER-7.0

BEHAVIOR AND ACTIVITIES OF THE CHILDREN

7.1 INTRODUCTION

Studies in laboratory animals have shown that that air pollution may cause brain damage (Calderon-Garciduenas et al., 2002). Investigations in humans have illustrated brain lesions and impairment of olfactory function among residents of highly polluted Mexico City (Hudson et al., 2006).

These reports indicate that chronic exposure to high level of urban air pollution may cause changes in the brain activity that may manifest themselves in behavioral alterations. In children a frequently diagnosed psychiatric problem is Attention-Deficit Hyperactivity Disorder (ADHD), previously known as attention deficit disorder and characterized by inattentiveness, over activity and impulsiveness. Children with ADHD are impulsive, forgetful, restless, prone to fail, unable to follow tasks, unpredictable and moody. Against this background it was decided to find whether the chronic exposure to Delhi's high level of particulate pollution can cause behavioral problems in children. To accomplish this objective the prevalence of ADHD and other behavioral activities was examined in this study in a cross sectional population of schoolchildren of Delhi and compared the results with that of controls.

Objective

The goal of this study was to explore whether sustained exposure to high level of urban air pollution has any impact on the behavior and activities of the in children.

7.2 SUBJECTS AND METHODOLOGIES

(a) Participants

A total number of 4275 children in the age group of 9-14 years were enrolled for ADHD screening. The control group was represented by 1645 children, boys 1220 and girls 425. In Delhi, a total number of 2630 children were enrolled, of which 1750 were boys and 880 were girls. The enrolled children did not have history of neurological problems such as epilepsy, autism, mental retardation, or sensory deficits.

(b) Diagnosis of ADHD

Attention-Deficit Hyperactivity Disorder (ADHD) was screened following the criteria prescribed in the Diagnostic and Statistical Manual of conduct disorders (DSM-IV) of American Pediatric Association (APA, 2000). In evaluating ADHD symptomatology, it is important to obtain independent reports about the child's behavior at school from the teacher and about the child's behavior at home from the parents (de Nijs et al., 2004). Accordingly, reports from the parent as well as the class teacher of the school had been obtained

DSM-IV manual emphasizes that presence of 6 or more symptoms of either category # 1 or 6 or more symptoms of category #2 confirms the diagnosis of ADHD.

#1. Six or more of the following symptom of *Inattention* have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Inattention

a. often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities
b. often has difficulty sustaining attention in task or play activities
c. often does not seem to listen when spoken to directly
d. often does not follow through on instruction and fail to finish schoolwork, chores or duties in the workplace not due to oppositional behavior or failure to understand instructions
e. often has difficulty organizing tasks or activities
f. often avoids, dislikes, or is reluctant to engage in tasks or activities such as school work or home work
g. often loses things necessary for tasks or activities (e.g. pencil, pen, books)
h. is often easily distracted by extraneous stimuli
i. is often forgetful in daily activities

 # 2. Six or more of the following symptoms of *Hyperactivity-Impulsivity* have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Hyperactivity

a. often fidgets with hands or feet or squirms in seat
b. often leaves seat in classroom or in other situations in which remaining seated is expected
c. often runs about or climbs excessively in situations in which it is inappropriate
d. often has difficulty in playing or engaging in leisure activities quietly
e. is often 'on the go' or often acts as if 'driven by a motor'
f. often talks excessively

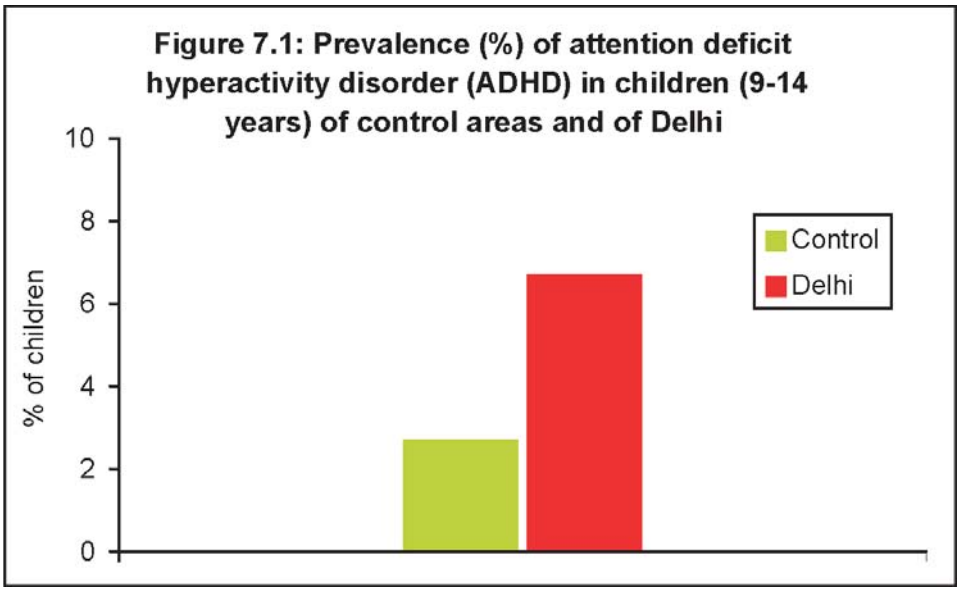
Impulsivity

g. often blurts out answer before question have been completed
h. often has difficulty awaiting turn
i. often interrupts or intrudes on others

7.3 RESULTS

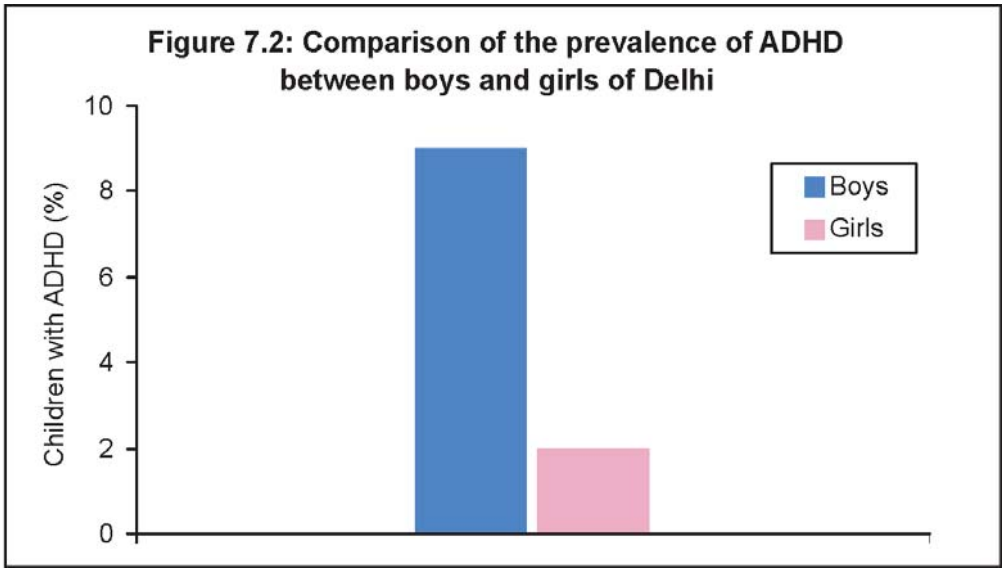
(a) Prevalence of ADHD

Out of 2630 children enrolled in Delhi for this study during 2003-2005, 176 children (6.7%) had ADHD. In contrast, 44 of 1645 control children (2.7%) examined during this period had ADHD (Fig. 7.1). Thus, Delhi's children had 2.5-times more ADHD prevalence than the controls.



(i) Difference between boys and girls

Of the 176 children with ADHD in Delhi, 158 were boys and 18 were girls. Thus, 9% of the boys enrolled in Delhi had ADHD against 2% of the girls, giving a male: female ratio of 4.5:1 in ADHD prevalence in the city (Fig. 7.2). In controls, 41 boys (3.4%) and 3 girls (0.7%) had this problem, giving a male: female ratio of 5:1. Thus, the boys were about 5-times more prone to ADHD than the girls of similar age both in control group and in Delhi. But, Delhi's boys and girls had 2.6- and 2.9-times greater prevalence of ADHD than control boys and girls respectively.



(ii) Type of ADHD

Inattentive type was predominant in Delhi as well as in control group. Among the ADHD children of Delhi, 3.6% had inattentive type, 2.4% had hyperactive-impulsive type, and 0.7% had combined

type of ADHD. In the control group, the relative proportions of these three categories were 2.1%, 0.4%, and 0.2% respectively (Table 7.1).

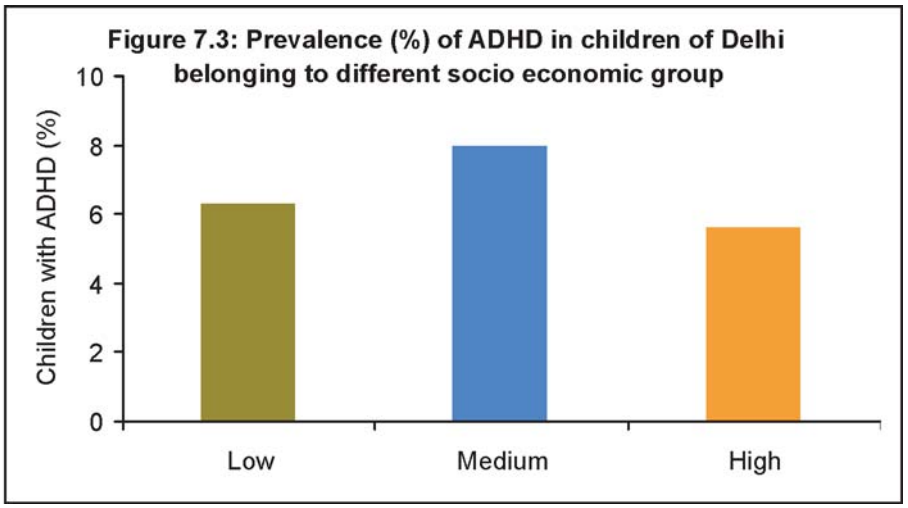
Table 7.1: Prevalence of ADHD and its subtypes

ADHD category	Control (n=1645)		Delhi (n=2630)	
	Number	% of total	Number	% of total
Inattentive	35	2.1	96	3.6*
Hyperactive-impulsive	6	0.4	62	2.4*
Combined	3	0.2	18	0.7*
ADHD, total	44	2.7	176	6.7*

*, p<0.05 compared with control in Chi-square test

(iii) ADHD and socio-economic status

The prevalence of ADHD in Delhi was more in children from medium (8%) and low (6.3%) SES compared with high SES (5.6%, Fig. 7.3).



(iv) Air pollution and ADHD

After controlling potential confounders like age, gender, socioeconomic status and parental smoking, PM_{10} was found to be positively associated with ADHD prevalence in children (OR=1.32, 95% CI 1.08-1.56).

(b) Comparison of physical and creative activities between urban and rural children

(i) Participation in sports

Active participation in sports was more prevalent in control group than in Delhi. About 80 % of control children participated in sports compared with 58% children of Delhi (p<0.05). Boys preferred outdoor games to indoor games, while girls showed an inclination towards indoor games.

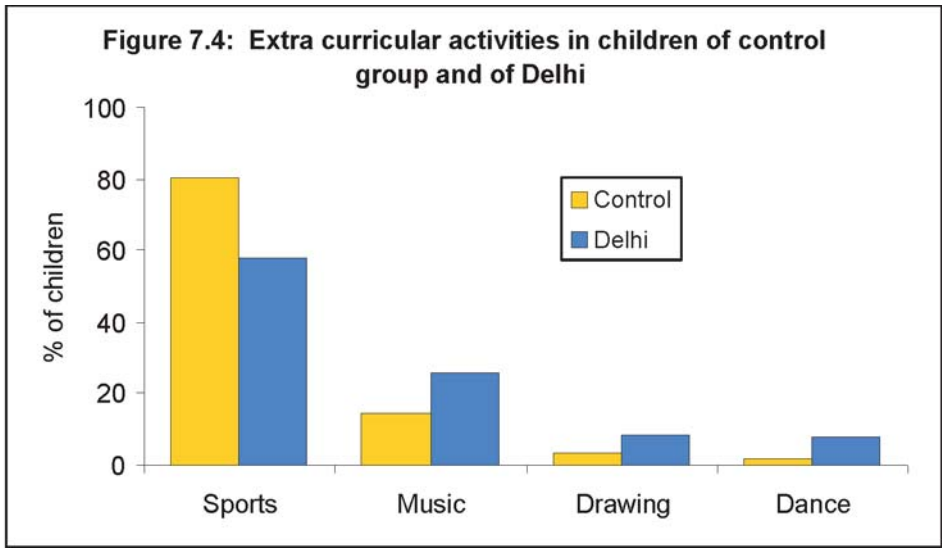
(ii) Interest in music, painting and dance

Delhi' children showed more interest in creative activities such as participation in vocal and instrumental music, drawing or painting and dance than children of the control group, and the difference were statistically significant (Table 7.2, Fig. 7.4).

Table 7.2: Percentage of children with different areas of interest

	Boys		Girls		Total	
	Control	Delhi	Control	Delhi	Control	Delhi
Sports (%)	88.3	61.4*	66.4	51.1*	80.4	58.0*
Indoor (%)	11.4	22.3*	40.6	20.0*	21.9	21.5
Outdoor (%)	76.9	39.1*	25.8	31.2*	58.5	36.5*
Music (%)	8.7	25.0*	24.5	28.1	14.4	26.0*
Instrumental (%)	4.2	13.3*	3.3	13.4*	3.9	13.3*
Vocal (%)	4.5	11.7*	21.2	14.6*	10.5	12.6
Drawing/painting (%)	2.5	7.2*	5.6	9.9*	3.6	8.2*
Dance (%)	0.5	6.4*	3.5	10.9*	1.6	7.9*

**, p<0.05 compared with controls in Chi-square test*



(iii) Pastimes: watching television

In general, the most preferred pastime of children while at home was watching television, especially in Delhi. About 54% of Delhi's schoolchildren watch television 1-3 hours per day regularly including school days compared with 45% of control (Table 7.3). The boys watched television more than the girls (60.0 vs. 43.1%, p<0.05) in Delhi. In contrast, girls of the control group were more addicted to television than the boys (48.0% vs. 43%). Another 12% of Delhi's children spent 1-2 hours per day with the computers and video games. The percentage of computer/video games users was negligible (0.7%) in the control group.

Table 7.3: Pastime of the children

Pastime	Boys		Girls		Total	
	Control	Delhi	Control	Delhi	Control	Delhi
Reading books and periodicals etc.	39.5	21.4*	37.8	28.4*	38.9	23.8*
Watching television	42.8	60.0*	47.6	43.1*	44.5	54.4*
Listening to music	17.6	8.2*	14.6	12.7	16.6	9.8*

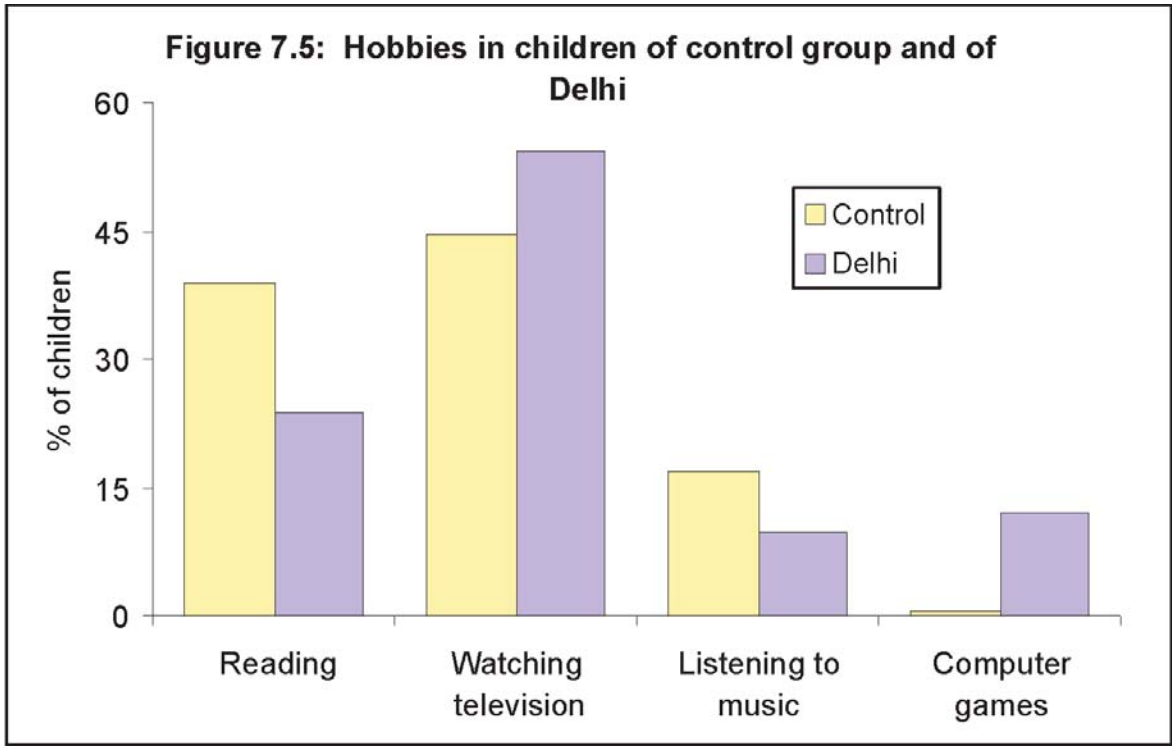
*Results are expressed as percentages of children; *, p<0.05 compared with control in Chi-square test*

(iv) Reading books and periodicals

Reading of books other than schoolbooks, and periodicals was the next preferred pastime for the children. Reading habit was much more prevalent in control group where 40% children had the habit against 24% of city's schoolchildren (p<0.001). Girls were more avid readers than the boys in Delhi (28.4 vs. 21.4%, p<0.05), but not in controls (Table 7.3).

(v) Listening to music

About 10% of Delhi's children and 17% of control had pastime other than watching television and reading books, of which listening to music was most important (Fig. 7.5).



7.4 FINDINGS

1. Delhi's schoolchildren had 2.5-times more ADHD prevalence than age-and sex-matched controls (6.7% vs.2.7%, $p<0.05$). Boys had a remarkably higher prevalence of ADHD than the girls both in Delhi and in control. Boys: girls ratio of ADHD prevalence was 4.5:1 in Delhi (9% vs. 2%) and 5:1 in control (3.4% vs. 0.7%), and children from medium socio-economic status had the greatest prevalence (8.0%) of ADHD. Among the ADHD children of Delhi, 3.6% had inattentive type, 2.4% had hyperactive-impulsive type, and 0.7% had combined type of ADHD. In the control group, the relative proportions of these three categories were 2.1%, 0.4%, and 0.2% respectively.

2. Active participation in sports was more prevalent among control children than in Delhi as 80 % of control children regularly participated in sports compared with 58% schoolchildren of Delhi ($p<0.05$). Boys preferred outdoor games to indoor games, while girls showed an inclination towards indoor games.

3. Delhi' children showed more interest in creative activities such as participation in music (26% vs. 14.4%), drawing or painting (8.2% vs. 3.6%) and dance (7.9% vs. 1.6%) than children of the control group, and the differences were statistically significant ($p<0.05$).

4. The most preferred pastime of children while at home was watching television. About 54% of Delhi's schoolchildren watched television 1-3 hours per day regularly including school days compared with 45% of control (Table 90). The boys watched television more than the girls (60.0% vs. 43.1%, $p<0.05$) in Delhi. In contrast, girls of the control group were more addicted to television than the boys (48.0% vs. 43%). Another 12% of Delhi's children spent 1-2 hours per day with the computers and video games. The percentage of computer/ video games users was negligible (0.7%) in the control group.

5. Reading of books other than schoolbooks, and periodicals was the next preferred pastime for the children. Reading habit was much more prevalent in control group where 40% children had the habit against 24% of city's schoolchildren ($p<0.001$). Girls were more avid readers than the boys in Delhi (28.4% vs. 21.4%, $p<0.05$), but the reverse was true in controls. About 10% of Delhi's children and 17% of control had pastime other than watching television and reading books, of which listening to music was most important (9.8% in Delhi and 16.6% in control).

CHAPTER-8.0

DISCUSSION

Compared with age-and sex-matched rural controls, remarkably higher prevalence of upper and lower respiratory symptoms and lung function deficits were found in Delhi's school-age children. The findings could be linked to city's high level of ambient air pollution, because a large number of studies have shown positive associations between air pollution and respiratory symptoms and lung function impairment (Samet, 2002). Upper and lower respiratory symptoms, which were found in excess in children of Delhi, could be a fall-out of greater interaction of the airways with allergens and pathogens and/or impairment in lung defense. A review of the literature on possible association between air pollution and respiratory symptoms in children show that outdoor and indoor air pollution are positively associated with upper and lower respiratory tract infections in children (Chauhan et al., 2005). Available evidence conclusively suggest that outdoor air pollution, including PM_{10}, $PM_{2.5}$, NO_2, SO_2 and ozone, is associated with increased upper and lower respiratory symptoms in children. Many of these symptoms are related to infections. Viral and bacterial infections can cause severe pathological abnormalities in both the upper and lower respiratory tract, the extent of epithelial damage varied with the pathogen type. The lower airway epithelium between the bronchial and alveolar regions is particularly susceptible compared with nasal, laryngeal and tracheal regions. It is possible that the penetration of pathogens into the epithelium could be facilitated both by epithelial shedding and by reduced ciliary clearance, resulting in easier access of pathogen to antigen presenting cells leading to increased inflammation. Acute respiratory infections (ARIs) are divided into two main groups; upper respiratory tract infections (URTIs) and lower respiratory tract infections (LRTIs). The risk of severe ARIs is greatest in very young children and in the elderly. WHO has defined URTIs as a combination of symptoms including one or more of the following; cough with or without fever, blocked or running nose, sore throat and ear discharge (WHO, 1997). URTIs are usually viral in origin and include the common cold viruses such as rhinoviruses and corona viruses. In contrast, LRTIs involve lung infection with symptoms like cough and dyspnea, bronchitis and pneumonia, which are usually presented in serious forms. URTIs and LRTIs are not mutually exclusive; they frequently coexist during the same episode of respiratory infections. Bacteria and viruses cause infection in both the upper and lower respiratory tracts, and upper respiratory infections is often followed or accompanied by lower respiratory infections (Chauhan et al., 2005).

There has been a series of studies investigating the link between URTIs and air pollution. Traffic related air pollution and the risk of respiratory diseases in a cohort of 4000 children were investigated in Netherlands, and a positive association was observed between air pollution and URTIs. A study in the United Kingdom investigated general practitioner consultation for upper respiratory tract diseases excluding allergic rhinitis with outdoor air pollution. In children aged 0-14 years, a 3.5-fold increase in such consultation was observed for an increase of SO_2 levels between 13-31 $\mu g/m^3$ (Hajat et al., 2002). Another study in Taiwan, China observed 0.3-1.3% increase in consultations with physicians for a 10% increase in levels of NO_2, SO_2 and PM_{10} (Hwang and Chan, 2002). In a cross sectional study in 10 different areas of Switzerland in 6-15 years old children, the risk of bronchitis between the most and least polluted areas was found significant with an odds ratio of 2.17 for PM_{10} (Braun-Fahrlander et al., 1997). A recent study from the UK investigated the association between lower respiratory conditions and primary care consultations in London (Hajat et al., 1991). For a 10-90th percentile increase in pollutant level in winter increase in consultations for respiratory illness in children were observed for NO_2 (7.2%), CO (6.2%) and SO_2 (5.8%), whereas in adults the only consistent association was with PM_{10}. It is possible that air pollution may modify symptoms in children who are already infected. Exposure to NO_2 at hourly peak levels of the order of less than 160 $\mu g/m^3$ compared with background level of 40$\mu g/m^3$ was associated with a significant increase

in sore throat, colds and school absenteeism although no infection was confirmed (Pilotto et al., 1997).

Allergy and asthma

Allergic rhinitis (running or stuffy nose), wheeze, cough and dyspnea were present in a substantial number of children of Delhi. Allergic rhinitis is one of the most common chronic diseases in children (Meltzer, 2006). It can significantly impact a child's health, as it impairs quality of life, and can predispose to the development of co-morbidities such as asthma (Meltzer, 2006). In addition, exposure to fungal spores, such as spores of *Alternaria* could elicit these symptoms (Delfino, 2002). Exposure to airborne bioaerosols including fungus (*Aspergillus* and *Alternaria;* Ponikau et al., 2005), bacteria (*Streptococcus pneumoniae*; Peltola et al., 2006), and viruses (*rhinoviruses*; Osur, 2002) are leading causes of sinusitis. The nose collects bioaerosols that may lead to several human diseases. For instance, living in a moldy house usually increases the risk of respiratory symptoms (Kostamo et al., 2005) and diseases like asthma, allergic rhinitis, sinusitis, and hypersensitivity pneumonitis (Bush et al., 2006). Incidentally, fungal spores are abundant and the prevalence of fungal sinusitis is high in north India (Chakrabarti and Sharma, 2000). Besides bioaerosols, other air pollutants can promote airway sensitization by acting as adjuvant. The prevalence of allergic rhinitis was positively correlated with traffic-related air pollutants after controlling potential confounding and climatic factors (Lee et al., 2003). However, a European study has shown that ambient particles (PM_{10}) from combustion sources act as carrier of allergens (such as pollen) that could elicit allergic reactions in the airways following inhalation (Namork et al., 2006).

Asthma is a chronic respiratory disease characterized by the inflammation of the airways and variable airflow obstruction (Holgate, 1999). The underlying mechanism of asthma is not fully understood. Clinical symptoms include wheeze, shortness of breath, cough, chest tightness and reduction in FEV_1, PEFR and FEF_{50}. It has been proposed that asthma probably encompasses several disparate groups of disorders that produce similar clinical effects (Pearce et al., 1999). In the present study, current asthma and medically diagnosed asthma was in recorded in 4.6% and 1.7% of Delhi's children respectively. Although asthma prevalence was significantly higher in Delhi than in rural areas, prevalence of asthma in Delhi was low compared with urban children of several other countries. For example, doctor-diagnosed asthma was found in 2.6% children aged 15-17 years in Lebanon (Salameh et al., 2003). Contrary to the common belief (Kjellman and Gustafson, 2000), the prevalence of asthma did not decline with advancing age among the children of Delhi. Similar finding has been rmported from some Asian cou~tries including Iran (Golshan et al., 2002) and Thailand (Trakultivakorn, 1999).

Information on the prevalence, magnitude of the problem and risk factors of childhood aóthma in India is scanty. A questionnaire-based survey was carried out in 18,955 children aged 5-17 years from 9 randomly selected schools of Delhi by Chhabra et al., (1999). The prevalence of current asthma was 11.9% while past asthma was reported by 3.4% of children. Boys had a significantly higher prevalence of current asthma as compared to girls (12.8 vs 10.7%). Multiple logistic regression showed that the risk factors were male sex, family history of atopic disorders, and presence of smokers in family, but not the economic class, air pollution, and fuel use at home (Chhabra et al., 1999). In agreement with this study, no causative role of particulate air pollution with asthma was found. However, a lower prevalence of asthma among the schoolchildren of

Delhi compared with earlier report was recorded. Whether this is due to difference in sample size and procedure between these two studies or a genuine decline in asthma prevalence in the intervening years after the introduction of CNG, is yet to be ascertained. In another study in Delhi, univariate analysis showed that the risk factors for asthma in children are family history of asthma, lack of exclusive breast-feeding, and associated allergic rhinitis and atopic dermatitis (Ratageri et al., 2000). Early onset of symptoms, asthma in grandparents and more than10 cigarettes smoked per day by any member of the family were found to be positively associated with severe asthma in 5-15 year old children. Like the previous study of Chhabra et al., (1999) and the current one, Ratageri et al., (2000) found no significant effect of air pollution on asthma development or its severity. On the other hand, the authors reported exclusive breast-feeding for more than 4 months as the most protective factor for development of asthma. The ISAAC questionnaire study in Chandigarh, on the other hand, has established a 12.5% prevalence of wheeze in 13-14 year-old children (Mistry et al., 2004).

There was no association between air pollutants and prevalence of asthma (Ware et al., 1986). A subsequent follow up study confirmed lack of association between particulate matter and the prevalence of asthma symptoms and diagnoses, although a positive association was found between exposures to particulate air pollution and the prevalence of bronchitis and cough (Dockery et al., 1989). Weiland and Forastiere, (2005) have recently reviewed the available data on impact of air pollution on asthma and allergies in children. They found little evidence in favor of a causal association between the prevalence/incidence of asthma and air pollution in general, although there are some suggestive evidence for a causal association between the prevalence/incidence of asthma symptoms and living in close proximity to traffic. Similarly the evidence for an association between the prevalence/incidence of hay fever and exposure to traffic-related air pollution is only suggestive. Among children with asthma, air pollutants cause increase in the frequency of respiratory symptoms, increase in medication use and transient deficit in lung function. The evidence for an association between air pollution exposure and the frequency of hospital admissions or emergency department visits for asthma was sufficient to infer causality. Overall the authors have concluded that air pollution contributes to asthma aggravation, leading to an increase in symptoms, greater use of relief medication and transient decline in lung function (Weiland and Forastiere, 2005). After adjusting potential confounders like ETS and atopy exposure to vehicular pollution before the age of three was found to be significantly associated with asthma while life long exposure was not (Zmirou et al., 2004).

The risk of developing asthma has a genetic basis, as many patients with asthma are atopic, i.e. they have a genetically determined immune reactivity that favors IgE response to multiple environmental antigens (Harris et al., 1997). Still, air pollution could increase the prevalence of asthma symptoms and their severity. A few reports have recorded an association between traffic-related air pollution and symptoms of asthma. For example, vehicular pollution from combustion of diesel has been shown to increase the prevalence of wheeze (Hoppin et al., 2004). After adjustment for age, history of atopy, and parental education, asthma prevalence in 13-15 years old children of Taiwan, China was reported to be associated with traffic-related air pollution, especially CO and NOx (Guo et al., 1999). Long-term exposure to traffic related air pollutants increase the risk of asthma in children (Hwang et al., 2005). In general, 3% increase in asthma symptoms (wheeze and dyspnea) has been observed for every 10 $\mu g/m^3$ rise in PM_{10} (Donaldson, 2000). Besides PM_{10}, a positive and consistent association between asthma outcome and ozone has been reported

(Delfino, 2002). Ozone (O_3), a product of volatile hydrocarbon degradation to nitrogen oxides, is a non-radical oxidant and a major component of photochemical smog. Exposure to O_3 concentration that exceeds current US EPA NAAQS (120 ppb) is a daily occurrence for millions of people throughout the world (e.g. Mexico City ozone >0.12 ppm). School children are especially highly exposed group because they engage in play and competitive outdoor physical activities in the afternoon, when ozone levels are at their peak (Calderon-Garciduenas et al., 1997). Delfino et al., (1998) found that asthma symptoms were associated with both PM_{10} and ozone with a greater relative effect from PM_{10}. Ostro and his group (2001) reported positive association between PM_{10} and $PM_{2.5}$ levels, particularly the former, and the prevalence of cough, shortness of breath, wheeze, and asthma episodes. But the authors could not find any association between these symptoms and ozone.

Some investigators have examined the relationship between socio-economic conditions and asthma. Low SES, expressed in terms of low parental education, was found to be associated with wheeze and nocturnal dry cough, but not with doctor-diagnosed asthma and bronchitis in children aged 6-12 years over 13 countries in Europe and America (Gehring et al., 2006). The authors suggested that crowding and mold and moisture in home could be the connection between low SES and higher prevalence of wheeze in children. It may be emphasized that living in a moldy house or working in a similar environment increases the risk of respiratory symptoms and infections (Kostamo et al., 2005). Increased or subnormal body weight can be important risk factors for asthma and other respiratory complications. For instance, emphysema is significantly associated with underweight (BMI <18.5 kg/m^2) while asthma and chronic bronchitis are more prevalent in overweight (BMI e”28 kg/m^2) persons (Guerra et al., 2002).

Vehicular pollution and respiratory symptoms

The combustion of automotive fuels produces a variety of outdoor pollutants such as NO_2, SO_2 and PM_{10}. There is increasing evidence to suggest that exposure to these pollutants is positively associated with the genesis of several respiratory diseases in children as well as in adults. In many epidemiological studies of outdoor NO_2 and PM_{10} exposure, an association has been found between exposure to these pollutants and adverse health effects often at levels well below the current WHO guidelines. Many of these functions can be modulated by exposure to PM_{10}, NO_2 and other air pollutants (Chauhan and Johnston, 2003; Chauhan et al., 2005). Acute exposure to oxidant pollutants results in ciliostatis in both upper and lower airways, which may prevent the nasal and bronchial mucosal from filtering inhaled particles such as aeroallergens, bacteria and viruses (Devalia et al., 1993). NO_2 reduces ciliary beat frequency of human bronchial epithelial cells and causes ciliary dyskinasis (Helleday et al., 1995). Repeated exposures to NO_2 impair bronchial immunity by reducing total lymphocytes in peripheral blood and CD4+ and CD8+ cell ratio in bronchoalveolar lavage fluid (Sandstrom et al., 1992). Particulate air pollutants are able to penetrate the lower airways in children (Bunn et al., 2001). The percentage of particle-containing AM was found increased (median 10% vs. 3%) in children who lived on a main road compared with those living on a quiet residential road (Bunn et al., 2001).

Several studies have described a close association between traffic-related air pollution and respiratory symptoms (Wjst et al., 1993) and decreased lung function (Brunekreef et al., 1997) in children. Unlike the concentration of RSPM (PM_{10}), the levels of SOx and NOx in Delhi’s air were within the standards. Therefore it seems possible that particulate matter, rather than SOx or NOx,

was primarily responsible for the adverse respiratory changes recorded in city's children. In agreement with this, epidemiological studies have shown that particulate matter, but not SO_2, is associated with increased prevalence of URS and LRS in urban children (Aekplakorn et al., 2003), adolescents (Pierce et al., 2006), and adults (Kamat et al., 1992). However, high level of NO_2 in some Korean cities has been shown to increase URS and LRS in children (Lee et al., 2005). The American 'six cities study' examined the effects of air pollution on the respiratory health of pre-adolescent children living in communities with different levels of air pollution between 1974 and 1977. Questionnaire survey revealed that the frequency of cough and bronchitis was significantly associated with average 24-hr mean concentrations of TSP, TSO_4 and SO_2 during the year preceding the health examination. In another study, no association was found between the prevalence of respiratory symptom in 8-11 year-old children and last year' mean PM_{10} level, suggesting that current air quality is important in relation to respiratory symptoms (Horak et al., 2000).

Exposure of human AM to NO_2 for short durations resulted in a functional impairment of the AM (Kienast et al., 1996). Exposure of AM to PM_{10} significantly reduced respiratory syncitial virus (RSV)-induced production of IL-6 and IL-8. This suggests that AM-related inflammatory responses to viral infection are altered by exposure to PM (Baker and Soukup, 1999). Prior exposure of laboratory animals to diesel particulates has been shown to increase viral replication, lung inflammation and impairment in host defense mechanism (Harrod et al., 2003). Diesel exhaust particles synergistically enhance neutrophilic lung injury related to endotoxin from gram-negative bacteria. In the presence of endotoxin DEP further activated the p65 subunit of NF-kB in the lung and the expression of ICAM-1, MCP-1 and MIP-1 (Takano et al., 2002). Therefore it is likely that alterations in AM function by airborne pollutants are important risk factors for potentially fatal infections in the airways and the alveoli.

Influence of SES

More respiratory symptoms were found in children from low socio-economic status. In agreement with this, poor sanitation, low birth weight, vitamin A and zinc deficiency, and poverty which are usually the hallmark of low socio-economic conditions, have been shown to be significantly associated with 4 million deaths globally due to respiratory infections between 1997 and 1999 (WHO, 2000). Acute respiratory infections were responsible for 25% of all deaths in children under five in Europe in 2001. Besides social and economic conditions, indoor and outdoor air pollution, which itself is intimately related to social and economic fabric of the society, is emerging as a major contributor to this problem. Low birth weight, under nutrition, vitamin A and zinc deficiency, lack of breastfeeding, overcrowding and air pollution increase the risk of pneumonia in children that cause 2 million death each year, 70% of which in Africa ad South-East Asia (Singh, 2005). A study conducted in Delhi has demonstrated lack of breast-feeding, URS in mother or sibling and severe malnutrition as important risk factors for acute lower respiratory symptoms in children below five years of age (Broor et al., 2001). Low socio-economic conditions in childhood may be a risk factor for respiratory illness in adulthood. A study in human volunteers in USA demonstrated a close association between low socio-economic status in childhood and decreased resistance to upper respiratory infections from rhinoviruses in adulthood (Cohen et al., 2004).

Air pollution and lung function deficits in children

Compared with rural controls, reduced lung function growth rate as well as lung function deficits was found at a given age both in boys and girls of Delhi. Lung development and lung function are

influenced by several factors. Most important among these are birth weight, infections, nutrition and environmental factors such as air pollution. It is important to mention in this context that air pollution hampers teenagers' lung development (Khan, 2004). Lung is affected by chronic exposures to high level of NO_2, SO_2 and PM, of which the effect is strongest for PM (Ackerman-Liebrich et al., 1997). Recent study has identified $PM_{2.5}$ as the most dangerous particulate fraction in this regard (Bernstein and Abelson, 2005). Fine particulates ($PM_{2.5}$) are ubiquitous because they are largely derived from common combustion processes such as engines of motor vehicles, power generation, burning of biomass, and manufacturing, and they are transported over long distances and readily penetrate indoors (Pope, 2004b). Exposure to fine particulate matter may be an important public health concern (Pope, 2004b). Such matters that can be breathed deeply into the lungs include sulfates, nitrates, acids, metals, and carbon particles with various chemicals adsorbed onto their surfaces (Pope, 2004b). Chemical composition and radical-generating capacity of the PM depends on the source of emission. Therefore, chemico-toxicological characteristics of PM vary at locations with high traffic emissions from that of PM at low traffic locations, regardless of the mass concentration of PM (Hogervorst et al., 2006). It should be emphasized in this context, that NAAQS is not the safe level. For example, NO_2 below the current European limit value significantly impairs lung function. It has been shown that for every increase of 10 µg/m^3 of PM_{10} and NO_2 lung function parameters are reduced in general, by 1% in school children aged 7-10 years (Moshammer et al., 2006).

The association between air pollution and children's lung function deficits was unveiled by the epidemiologic studies conducted in Europe and the United States. The Second National Health and Nutrition Examination Survey (NHANES II) in the United States demonstrated significant negative correlations between annual concentrations of TSP, NO_2 and ozone and FVC and FEV_1 in children, adolescents and young adults aged 6-24 years (Schwartz, 1989). Studies in young adults have shown that lung function decrement is associated with long- term elevated levels of particulates, and lung function decrement can be further worsened by concomitant exposure of PM_{10} with ozone (Abbey et al., 1998). Indeed, the 24-cities study in North America has shown a strong association of annual mean PM_{10}, ozone and acid aerosols with lung function of elementary school children (Raizenne et al., 1996). The authors reported 2.4% decrement in FVC and 2.1% decrement in FEV_1 for a rise of 17.3 µg/m^3 in annual mean PM_{10} level. Similarly, a 3.4% decrease in FVC has been recorded for every rise of 10 µg/m^3 in PM_{10}, (Ackerman-Liebrich et al., 1997). In general agreement with these studies, significant reduction in FVC (7.6% in medium and 9.9% in high pollution zones), but not in FEV_1, has been reported in school children of Moscow in response to varying ambient air pollution levels (Eroshina et al., 2004). Even in countries with cleaner air such as in lower Austria where annual PM_{10} level varies between 15.8-26.9 µg/m^3 in 1996, a 10 µg increase in last two week's mean PM_{10} was associated with appreciable decrease in FVC, FEV_1, and especially $FEF_{25-75\%}$ (MMEF) in children aged between 8 and 11 years (Horak et al., 2000). Most sensitive indicator for acute effects of combustion-related pollutant was a change in MMEF in small airways (Moshammer et al., 2006).

Measurement of children's lung function over several weeks or months (called Panel studies) before, during and after air pollution episodes in Ohio, USA (Dockery et al., 1982) and Netherlands (Dassen et al., 1986) have shown that lung function decreased after episodes of very high particulates and SO_2, but returned to pre-episode level within a few weeks Similarly, ozone (Kinney et al., 1996; Lippmann and Spektor, 1998) and PM_{10} concentrations (Pope et al., 1991) have been linked with fall in PEFR. Repeated measurements of lung function over many years (called

Prospective Cohort Study) in Poland in preadolescent children demonstrated significantly lower mean lung function growth rate adjusted to height in children living in more polluted areas of Krakow (Jedrychowski et al., 1999). Similar study in Austria for 3 years has found adverse effect of ozone on lung function growth in children (Fischer et al. 1999). In a large prospective cohort study conducted in Los Angeles, California with 3000 children showed a strong association between exposures to PM_{10}, $PM_{2.5}$, NO_2 and inorganic acid vapor with deficits in growth of lung function, as measured by changes in FVC, FEV_1 and MMEF (Gauderman et al., 2000). Compared with children living in the least polluted community, those living in most polluted community of the above study had a cumulative reduction of 3.4% in FEV_1 and 5% in MMEF over the four-year study period, and the deficits were more in children spending longer time outdoors (Gauderman et al., 2000). The latest report from the Children's Health Study in the US with 1759 children aged 10 to 18 years for a long 8-year follow up period has illustrated deficits in FEV_1, FVC, and MMEF in association with exposure to a variety of air pollutants including NO_2, acid vapor, $PM_{2.5}$ and elemental carbon (Gauderman et al., 2004). Impairment of lung function was found in apparently healthy student at levels below current European standards for NO_2- FEV_1 by 1%, MMEF 25% by 1.96% (Moshammer et al., 2006). Peripheral resistance increased by 1.03% per 10 µg/m^3 of $PM_{2.5}$ (Moshammer et al., 2006). Hence NO2 emissions should be strictly controlled. Collectively, these studies suggest that long-term exposure to elevated levels of air pollution during childhood can produce deficits in lung function growth. Therefore, poor air quality of the city can account for deficits in lung function growth in Delhi's schoolchildren. The level of lung function reflects cumulative effects of air pollution over a lifetime. Children with the highest level of lung function at any given age must have been growing faster. This is the 'horse racing effect' described by Fletcher and Peto (1977). This assumes that all children start at the same point in their lung function development. Yet, factors including air pollution may have affected lung function prenatally, and thus influence lung function development from birth onwards. Therefore, the decrement of FVC, FEV_1, MMEF and PEFR in Delhi's children in comparison with that of rural controls may suggest inhibition of expansion of the bronchioles and alveoli in the face of sustained exposure to high particulate pollution of the city.

Greater prevalence of restrictive type of lung function deficit in Delhi

Significant reductions of FVC in Delhi's children relative to age- and sex-matched children from rural areas provide clear signs of pulmonary function deficits in the city. Fall in FVC is associated with fall in total lung capacity and development of restrictive type of lung function decrement that has been seen more often than not in Delhi's children. Lung function and its growth during fetal and neonatal development, infancy, childhood and adolescence are influenced by many factors. These include genes, nutrition and a large number of environmental factors including air quality (Dockery et al., 2005). A recent study has shown that from childhood to adulthood a constant number (300-480 million) of respiratory units or alveoli is maintained in the lungs while both the smallest bronchioles and alveoli expand in size (mean linear diameter of alveoli increases from 184 to 231 µm) to produce the increased lung volume with increasing age and height. Males generally have more alveoli than females at all ages over 1 year, independent of weight (Thurlbeck, 1975). Lung development is essentially complete in girls by the age of 18 years, whereas in boys it continues till early twenties (Schwartz et al., 1988). It is therefore unlikely that the deficits in lung function at the age of 17 years that has been found in a large number of schoolchildren of Delhi will be reversed as they complete the transition into adulthood.

In addition to restrictive lung, Delhi's children had greater prevalence of obstructive type of lung function deficits. They had significantly reduced levels of FEV_1, FEV_1/FVC and $FEF_{25-75\%,}$ suggesting airway obstruction. As high as 70% of Delhi's children had $FEF_{25-75\%}$ value less than 80% of predicted, and in about 18% children it was reduced below 40% of predicted value. Reduction in $FEF_{25-75\%}$ is associated with peripheral (small) airway obstruction. In fact, it is a more sensitive index of airway obstruction than the FEV_1, especially for the small airways that are dependent on the elastic and resistant properties of the distal airways (Seaton and Crompton, 2000). Thus, a 2-fold rise in the prevalence of reduced $FEF_{25-75\%}$ in Delhi relative to control suggests a remarkable increase of small airway obstruction in Delhi that could be attributed to sustained exposure of these children to city's air pollution. COPD is not a well-defined entity in children (Kabra et al., 2001). A child presenting with chronic cough and wheeze should be investigated for asthma, recurrent aspiration airway compressions, chronic infection, cystic fibrosis and immune deficiency. In absence of these causes, environmental factors such as ETS and air pollution could be implicated (Kabra et al., 2001).

Road traffic and breathing problems

Delhi possesses 4.2 million motor vehicles, which is more than the combined number of automobiles possessed by country's other three metros-Mumbai, Kolkata and Chennai. A large number of investigations carried out in Europe and elsewhere support this hypothesis. For instance, a positive correlation between exposures to car traffic and the prevalence of cough, recurrent wheeze, recurrent dyspnea, and reduced lung function particularly PEFR and $FEF_{25-75\%}$ has been reported in Germany (Wjst, 1993). Road traffic also increases the risk of recurrent bronchitis, broncholitis and pneumonia among children in Italy (Ciccone et al., 1998). In the metropolitan areas of Turin, Milan and Rome in Italy the authors reported a positive association between exposures to road traffic and current respiratory symptoms, such as speech-limiting wheeze, persistent cough and persistent phlegm (Ciccone et al., 1998). The prevalence of respiratory symptoms in children aged 0-15 years living in streets with a high traffic density in the Dutch city of Harlem was much more than children living in quiet streets (Oosterlee et al., 1996). A group of Dutch investigators examined the prevalence of respiratory and allergic disorders among children aged 7-12 years who went to schools located less than 1 km from a major highway. Respiratory symptoms including cough, wheeze, and stuffy or runny nose were more prevalent among children attending schools located close (within 100 m) to a highway with high density of truck traffic, and the symptoms were more prevalent in girls (van Vliet, 1997). Lung function measurements of children attending schools located close to the highways were inversely related to truck traffic and concentrations of black smoke (Brunekreef et al., 1997).

Long-term periodic exposure to air pollution (SO_2, NO_2.CO, RSPM) of Jaipur was associated with increased PEF variability even in healthy subjects (Singh et al., 2003). Even brief exposures to particulate air pollutants have been associated with acute decrease in lung function. In an early meta-analysis, Dockery and Pope (1994) reported a 1.5% decrease in FEV_1 and a 0.8% decrease in PEFR for each 100µg/m^3 increase in PM_{10}. Similarly, Zmirou et al., (1997) reported 2.2% decline in FEV_1 and 0.7% decline in PEFR for each 100µg/m^3 rise in PM_{10}. In a meta-analysis, Ward and Ayres (1994) reported a 3.3 liter/min decrease in PEFR in association with 100µg/m^3 increase in PM_{10}. PM_{10} and $PM_{2.5}$ levels have been associated with decreased lung function in children in Utah (Pope, 1996) and southern California (Gauderman et al., 2000, 2002). The effect of particulates may be partially due to particle acidity. Particle strong acidity, categorized by SO_2- derived acidic sulphate particles has been associated with lung function decrement (Raizenne et al., 1996). In a

separate study in college students, it was observed that living for four or more years in regions of the country with high levels of ozone and related co-pollutants was associated with lower lung function, but the effects were more strongly associated with PM_{10} levels than with ozone (Galizia & Kinney, 1999; Kinney & Chae, 2005). Cross sectional analysis of the Children Health Studies also found a negative impact of ozone on PEFR ($r = - 0.75$, $p< 0.005$), and that of $PM_{2.5}$ on MMEF ($r = - 0.80$, $p< 0.005$) [Gauderman et al., 2000]. In the First Children Health Studies Cohort, ozone was not significantly associated with growth of FVC, FEV_1 or MMEF amongst school-aged children (Gauderman, 2000). In the second cohort of fourth grade student, however, ozone was associated with reduced growth of PEFR in children who spent more time outdoors (Gauderman et al., 2002). Overall, the result suggests age-dependent effects of ozone on the growth of small airway function that is most acute during pre-school ages.

In a prospective study of Dutch children followed over a two-year period with serial lung function measurement NO_2 was found negatively associated with MMEF, but the association was not consistent (Dijkstra et al., 1990). Other studies of the effect of NO_2, mainly from indoor sources, on lung function in children have been inconsistent (CEOHA, ATS, 1996). Taken together, the available report suggests that NO_2 at ambient levels may not have an independent effect on lung function level or growth. It is possible, however, that ambient NO_2 level may be associated with lung function growth in combination with other pollutants present in air.

Deficits in lung function have been observed in 1991 Dutch children living within 300 meter of busy roads, and the deficits were larger for traffic counts of diesel powered trucks than for petrol powered cars (Brunekreef et al., 1997). It was found in this study that the girls are affected more severely than the boys. In another study on 4320 fourth grade children in Munich traffic density was associated with diminished lung function (Wjst et al., 1993). Similar study in Leipzig in pre school children exposure to heavy traffic was associated with lower FVC and FEV_1 (Fritz and Herbarth, 2001). Although the effects of living near high-traffic roads may be related to NO_2 exposure, a number of other pollutants that are emitted in exhaust are of interest including diesel exhaust particles and ultra fine particles. Diesel exhaust contains high levels of NO_2 fine and ultra fine particles and organic compounds. Diesel exhausts appear to have acute and chronic effects on lung function (Sydbom et al., 2001). Elemental carbon, a marker of diesel exhaust, has been shown to be associated with reduced lung function in children (Gauderman et al., 2002).

Urban particles exist in three sizes: ultra fine particles < 0.1μm in diameter, accumulation mode particles between 0.1 μm and 2.5 μm in diameter, and coarse particles between 2.5 μm and 10μm in diameter. The fine particle mass $PM_{2.5}$ includes both the ultra fine and accumulation mode particles. UFP contribute very little to the overall mass of fine particles but are very high in number, especially within 100 m of road. These particles are of special interest because they have high deposition in the distal lungs, have larger surface areas coated with organic compound and transitional metals and have the ability to induce oxidative stress and inflammation (Oberdorster, 2001). However, no investigation of the chronic effect of UFP on lung function growth have been reported till date.

The impact of urban air pollution on the respiratory health of the children was studied cross-sectionally in Dresden, Germany (Hirsch, 1999). After adjustment for potential confounders, positive and statistically significant association between air pollutants and respiratory symptoms and reduced lung function was observed. Benzene was found to be associated with asthma and reduce FEF_{25-}

$_{75\%}$. A consistent association between particulate matter and respiratory symptoms has been reported by several panel studies. A majority of the 21 studies, 10 in Europe and 11 elsewhere, on PM_{10} have found an association between particulate pollution and respiratory symptoms. With a notable exception of the large PEACE study which did not find a clear effect of particulates. Six studies have been conducted with $PM_{2.5}$ and all showed a statistically significant effect. There are only a few studies available for $PM_{10\text{-}2.5}$ and for UFP so that no conclusion can be drawn for these particle ranges.

Fuel adulteration and children's health

In most cases adulteration involves i. blending of diesel or kerosene with petrol, ii. mixing of up to 30% of industrial solvents such as toluene, xylene and hexane with petrol, iii. mixing of used lubricants with diesel and petrol, and iv. blending as much as 20-30% of kerosene with diesel. Because kerosene is more difficult to burn than petrol, its addition results in higher levels of tailpipe emissions of hydrocarbons (HC), carbon monoxide (CO), oxides of nitrogen (NOx), particulate matter (PM) and known carcinogen like benzene and polyaromatic hydrocarbons (ESMAP, 2002). Moreover, kerosene has high sulfur level that can deactivate the catalyst of the vehicle leading to lower conversion of engine-out pollutants. If too much kerosene is added, octane quality will fall below the minimum requirement of the engines, causing engine knocking, mechanical damage of the engine besides abnormally higher emissions of PM, HC and NOx.

Impact of specific air pollutants

There are many studies on the effect of mixtures of ambient air pollutants on lung function development, but the specific pollutant responsible for reduced lung function is not clearly understood. Among the large number of chemical species present in ambient air ozone, NO_2, acid vapours, respirable particulates (PM_{10} and $PM_{2.5}$), SO_2 and acid aerosols have been identified as candidate pollutants for adverse effects on lung function (CEOHA, ATS, 1996). Living in regions with high levels of ambient ozone is associated with chronic deficits in lung function caused by reduced growth and faster decline in lung function (Lippmann, 1989). Short-term exposures to ozone are associated with reduced lung function, particularly fall in FEV_1 and PEFR (Kinney et al., 1996). In a meta-analysis of 29 panel studies on children, each 100µg/ m^3 increase in ozone was found to be associated with a 2.2% decrease in FEV_1 and a 3.4% decrease in PEFR (Zmirou et al., 1997). Community ozone level was associated with decrease in both FVC and FEV_1 (Schwartz, 1989). In contrast, no association of ozone with FVC or FEV_1 was found in college students although MMEF was reduced, suggesting that chronic exposure to ozone adversely affect the small airways (Kuenzli et al., 1997). In agreement with this, high lifetime ozone exposure was found to be associated with deficits in small airway function, and the effect was more acute if the exposure occurred earlier in life (Dockery et al., 2005).

Susceptibility

A number of host and genetic factors may contribute to variable response of children's lung to air pollutants. Asthma and other respiratory conditions, time-activity patterns and sex are important determinants of lung function growth. Dietary factors may also affect both lung growth and responses to air pollutants (Schunemann et al., 2002; Romieu et al., 2002; Romieu and Trenga, 2001). A growing number of susceptibility genes have been identified as participants in the

pathogenesis of persistent lung damage (Sanford et al., 2002; He et al., 2002). Genotypes that result in a higher-intensity oxidative stress, inflammatory responses or altered tissue response to damage appear to be associated with increase susceptibility to respiratory effects from acute and chronic exposure to air pollutants (Dockery et al., 2005). Overall lung function studies in children have suggested that air pollution adversely affects lung function growth. These effects of air pollution are generally modest. Nevertheless the effects can be cumulative over a 20- year growing period, and there is uncertainty over whether the chronic effects are reversible. Furthermore, even a small shift in average lung function can yield a substantial increase in the fraction of children with 'abnormally' low lung function. That is small changes in the population mean can reflect large changes in the susceptible subgroup of the population.

Other risk factors: malnutrition and low socioeconomic status

Underweight children from low SES of this study had greater prevalence of lung function deficits than that of children with normal weight, suggesting a role of nutrition on lung function. A recent study has documented that social class and poor air quality are independently associated with decreased lung function after controlling potential confounding factors (Wheeler and Ben-Shlomo, 2005). In agreement with the present finding, significant deficits in FVC, FEV_1 and PEFR were found in 5 to18-year-old semi-nomadic Fulani children and adolescents of Nigeria who were underweight due to poor nutrition (Glew et al., 2004). Diminished lung function in those underweight, malnourished children was thought to be due to respiratory muscle weakness and overall energy deficiency (Glew et al., 2004). In an earlier study with 50 boys aged 12-15 years, decreased lung function was found in boys from slums of East Delhi, compared with control boys studying in public school in east Delhi, indicating that environmental pollution and poor nutritional status contribute in unison to lung function deficits in children (Gupta, 1997).

Abnormal BMI

A significantly increased prevalence of overweight was observed among the schoolchildren of Delhi as compared with their control counterparts.This could be an important contributor to poor lung function, because lung function is adversely affected in young people by obesity (Perez-Padilla et al., 2006). A recent study has documented a strong negative correlation between BMI and FVC, FEV_1 and PEFR values (Ulger et al., 2006).

Fuel use at home, location of residence and population density

A study in north India on 200 schoolchildren examined the effect of fuel use at home for cooking on lung function of the children. It was found that lung function was mostly affected in children from biomass fuel and kerosene-using homes compared with LPG using households (Behera et al., 1998). A study in Ecuador supported these findings (Rinne et al., 2005). Duration of cooking hours also influences respiratory symptoms. For example, respiratory illness is more in children from households cooking with LPG two meals a day than one meal a day (Wong et al., 2004). A recent study has shown that children living near busy roads are 35% more exposed to 'soot' than children living in low traffic areas, although they attended the same school that was located far away from busy road (Van Roosbroeck et al., 2006). A major difference between the rural and urban areas covered in this study is the difference in population density, which was several times higher in Delhi. This could have influenced the prevalence of respiratory symptoms and lung function deficits

in city's children, as population density is associated with elevated prevalence of respiratory diseases (Holland and Reid, 1965).

Parental smoking and respiratory illness

Presence of smoker in family increases the risk of respiratory symptom and lung function deficits in children. In agreement with this, a study in Ecuador has shown that children living in homes where the parent or a family member is smoker had lower FVC and FEV_1 (Rinne et al., 2005). On the other hand, parental history of respiratory disease is a risk factor for the development of impaired lung function and airborne diseases in children, especially the boys (Lebowitz, 1989). It has been shown that increase of PM_{10} level over 100 $\mu g/m^3$ for 54 days per year is associated with a 7.2% decrease in FEV_1 as percent predicted in males whose parents had asthma, COPD or hay fever (Abbey et al., 1998).

Diet and respiratory health

Apart from difference in air pollution levels, urban and rural children of the current study have difference in food habit in relation to consumption of fish. While fish intake is high in children of West Bengal, which constitute bulk of the control group, the intake is low in Delhi. This could have influenced the prevalence of respiratory symptoms, because low fish intake is an important predictor of poor respiratory health (Antova et al., 2003).

Alteration of lung defense: changes in sputum cytology

The elevated number of alveolar macrophages in sputum of children in Delhi compared to their control counterparts is consistent with the relatively high level of air pollution in the city. The findings corroborate the observation of Mylius and Gullvag (1986) that the numbers of AM correlate with the level of particulate air pollution. Another significant finding of the study is the prevalence of iron-laden macrophages in the sputum of children of Delhi. Since Prussian blue positive ferric ion in AM generally comes from the disintegration of circulating erythrocytes, greater deposition of iron in AM may suggest chronic pulmonary irritation leading to microscopic hemorrhage (Grubb, 1994). Since the pulmonary tract of growing children is more susceptible to insults by air pollution (WHO, 1986), hemorrhage in lung capillaries of the children chronically inhaling polluted air in Delhi is not unlikely. Indeed, similar changes have been observed in schoolchildren of Kolkata who were chronically exposed to high level of particulate air pollution (Lahiri et al., 2000). In fact, abundance of iron-laden macrophages has been recognized as an adverse lung reaction to air pollution (Roy et al., 2001).

Greater presence of neutrophils, eosinophils and lymphocytes in sputum is indicative of underlying inflammatory and allergic reactions (Nobutomo, 1978). Therefore, the increased number of these inflammatory cells in sputum of Delhi's children underscores inflammatory response of the lungs to air pollution. Sputum neutrophilia in a large number of children in Delhi is suggestive of allergic reactions to air borne pollutants. A negative correlation was also found between sputum neutrophil number and spirometric lung function measurements of the children.

Goblet cell hyperplasia in sputum samples of Delhi's children underscores hypersecretion of mucous in response to chronic air pollution exposures. This could be a defense mechanism to restrict the airborne particle and pathogens and their speedy disposal through sputum expectoration. But it seems the defense is not adequate to contain the high load of particulates because abnormal

cellular characteristic such as multinucleation, metaplasia and dysplasia have already set in. Moreover, the chances of viral infection have apparently increased as recorded by the presence of ciliocytophthoria. The observation suggest that although the lungs have tried its best to resist the incoming pathogens and pollutants by secreting more mucous it has only fulfilled the objective partially and the cells are facing the burnt of toxic effects of air pollutants. In agreement with the present observation, squamous metaplasia was found in nasal epithelial cells of more than 30% children aged 9-12 years from highly polluted urban areas of Mexico City (Calderon-Garciguenas et al., 1997). They all had URS and damaged DNA in nasal cells. In essence, sputum cytology of the children of Delhi has illustrated adverse lung reaction at the cellular level. The inflammatory changes in the lung could be harmful because they exert oxidative stress, which is injurious for the airway lining cells leading to opportunistic infections.

Consequence of respiratory illness and lung function impairment

Childhood history of respiratory symptoms increases the risk of reduced lung function in adulthood, particularly in case of females (Lebowitz, 1989; Abbey et al., 1998). Respiratory symptoms in childhood are considered as significant predictors of all-cause mortality above the age of 30 years in urban subjects (Frostad et al., 2006). Deficits in lung function in childhood, on the other hand, is a strong risk factor for lung function impairment in adulthood (Dockery et al., 1985, Redline and Weiss, 1989), and all-cause mortality in adult life (Schroeder et al., 2003; Schnemann et al., 2000; Knuiman et al., 1999; Ryan et al., 1999; Neas and Schwartz, 1998; Hole et al., 1996; Rodriguez et al., 1994; Bang et al., 1993; Dockery et al. 1993; Krzyzanowski and Wysocki, 1986; Beaty et al., 1985; Kannell et al., 1983; Friedman et al., 1976; Asley et al., 1975). Of all the lung function measurements, depletion of FEV_1 percent predicted is a significant risk factor for mortality in both sexes, while reduced FEV_1/FVC ratio is associated with all-cause mortality only in males (Bang et al., 1993).

Is lung function deficit irreversible?

An important question is whether deficits in lung function growth related to air pollution are permanent or reversible. Cross-sectional prospective studies have shown that lung function may recover if an individual breathes cleaner air, either because improvement of air quality or because the person moves to an area with cleaner air (Dockery et al., 2005). Repeated measurement of children's lung function before, during and after air pollution episodes in Ohio, USA (Dockery et al., 1982) and Netherlands (Dassen et al., 1986) have shown that lung function decreased after episodes of very high particulates and SO_2, but returned to pre-episode level within a few weeks. In an earlier study Arossa and co-workers (1987) have shown decreased FEV_1 and MMEF values in Italian schoolchildren who lived in urban areas compared to their suburban controls. Following improvement in air quality of the city, the lung function of the urban children was so improved that it was hardly different from that of controls. Beneficial effects of reducing air pollution have been described from Dublin, Hong Kong, Atlanta, Schefaild (Chauhan et al., 2005). Lung function of children from more polluted areas who had moved to less polluted areas showed improvement in lung function growth rates (FEV_1, MMEF PEFR) [Avol et al., 2001].

Overall, the available reports suggest that reduction in air pollution in a short period improves lung function of the children. Thus, data from current available studies on responses to improving air quality or migration from areas of high concentrations of air pollutants to ones with lower concentrations suggest that recovery of lung function growth is possible.

Protective effects of antioxidants

Several lung diseases are associated with oxidative stress evoked by environmental pollutants. Dietary factors and nutrients including fruits and vegetables, antioxidant vitamins such as vitamin C, vitamin E, beta carotene and other carotenoids, vitamin A, fatty acids and some minerals such as sodium, magnesium and selenium confer protection against oxidative process and inflammatory response responsible for the genesis of these diseases (Romeiu, 2005). Vitamin E was found to be positively associated with FEV_1 while carotenoids increase FVC (Schunemann et al., 2002). Supplementation of Vitamin E (15 mg /day) and Vitamin C (250 mg/day) in asthmatic children improved MMEF and PEFR (Romieu et al., 2002).

Greater prevalence of hypertension in Delhi

Hypertension is emerging as a major health problem in India, and the magnitude of the problem is much more in urban than in rural subjects (Gupta, 1997). Our study has shown that even the children are not spared from this problem. About 6% children of the city and 1.4% of controls were suffering from hypertension, and the girls were particularly vulnerable to this problem as 7.9% of city's girls had hypertension compared with 4.8% of the boys. Arterial hypertension is influenced by some non-modifiable and modifiable factors. Non-modifiable factors such as age, female gender, and family history of hypertension increase the risk of increased systolic and diastolic blood pressure. Among the modifiable factors, lower physical activity, body weight gain, excess sodium chloride intake (> 5-7 g/day), and alcohol consumption increase BP. Higher prevalence of hypertension has been noted in upper socio-economic groups in countries at a transitional stage of economics.

In the past few years there has been a perceptible change in the lifestyle of the urban children in India. They are depending more and more on fast food and aerated beverages, and their participation in sports and physical activities is declining. It is conceivable that all these factors are leading to greater prevalence of obesity and to hypertension in urban children especially in those coming from affluent section of the society. Besides lifestyle changes, high level of air pollution in Indian cities could be partially responsible for increased prevalence of hypertension in urban children. Study in Brazil has shown that increase in air pollution from vehicular traffic, especially of CO and SO_2, increases systolic, diastolic and mean ambulatory blood pressure and reduces heart rate variability (de Paula Santos et al., 2005). Similarly, exposure to fine particulate pollution ($PM_{2.5}$) has been shown to increase cardiovascular morbidity and mortality (Samet et al., 2000; Pope et al., 2002). The mechanism of air pollution-mediated hypertension is not clearly understood, but exposure to airborne pollutants has been shown to cause arterial vasoconstriction and changes in autonomic balance (Gold et al., 2000; Devlin et al., 2003). In essence, greater prevalence of high blood pressure among the children of Delhi could be attributed to changes in lifestyle along with high level of ambient air pollution.

One of the important fall- out of exposure during pregnancy to high air pollution level is reduced birth weight of the baby. Interestingly, SBP correlates inversely with birth weight, beginning in the first decade of life, and the relation becomes stronger with increasing age in adulthood (Law et al., 1993).

Prevalence of childhood obesity

Obesity during childhood is a matter of growing concern. The global prevalence of overweight in school children is 3.3%. Some countries and regions had considerably higher rates. Highest

prevalence of overweight in the developing world is found mainly in the Middle East, North Africa and Latin America. However, Africa and Asia had 2.5 – 3.5 times more underweight (wasting) children than overweight (deOnis and Blossner, 2000). The problem of overweight amongst preschool children in Asia was 2.9% in 1995. In the cities, however, the prevalence varied from 5% to 9% (Tee, 2000).

Obesity in school-going children is a problem of the more affluent, Western countries. For example, 20.7 % of the boys and 17.2% of the girls in third grade at Baltimore City, USA were obese (Jehn et al., 2006). In contrast, only 2.2% of adolescents aged 10-15 years of Sri Lanka were obese (Jayatissa and Ranbanda, 2006). In fact, underweight children are a major problem of the developing countries, particularly in the villages. Every second child under 3 years of age is underweight in West Bengal, India (Mustaphi and Dobe, 2005). The main cause is malnutrition and poor hygiene. Similarly, the prevalence of underweight among adolescents of Sri Lanka aged 10-15 years was 47.2% (Jayatissa and Ranbanda, 2006).

Perez-Padilla and co-workers (2006) have reported 9.7% of children and adolescents aged 8-20 years of Mexico City as overweight, as they had BMI >95th percentile of CDC growth charts. Obesity was related to male gender, asthma and passive smoking. Lacar et al., (2000) reported that 22.1% of Mexican-Americans, aged between 12-17 years were overweight (>95th percentile). These reports provide example of the influence of better life conditions on body weight: compared with only 9.7% of overweight children in Mexico city, 22.1% of Mexican children in the United States were overweight.

Health consequences of obesity in children

Children who are overweight may begin to experience health consequences during their youth as well as put themselves at risk for weight-related health problems later in life. Lung function is affected adversely by obesity especially in young people (Perez-Padilla et al., 2006). Overweight children have been found to have risk factors for cardiovascular disease, including high cholesterol, elevated insulin levels, and elevated blood pressure during childhood. One study showed that approximately 60% of overweight children had at least one cardiovascular risk factor, such as high cholesterol or high blood pressure; in comparison, only 10% of children with healthy weight had at least one risk factor. Additionally, 25% of overweight children had two or more risk factors (Freedman, 1999).

Other health consequences include Type 2 diabetes, sleep apnea (not breathing for at least 10 seconds during sleep), social consequences including poor self-esteem and social discrimination (Must and Anderson, 2003).

In addition to the health problems that they may experience during their youth, overweight children are at increased risk for various chronic diseases as adults (including hypertension, type 2 diabetes, and coronary heart disease). Overweight adolescents are at greater risk of becoming overweight or obese as adults (Whitaker, 1997). It has been seen that about one third of all severely obese adults were overweight children (Ferraro, 2003).

Maintaining a healthy weight during childhood and adolescence may reduce the risk of becoming overweight or obese as an adult. Therefore, children should be encouraged to keep up healthy eating habits, participate in physical activity on most (preferably all) days of the week.

TV watching and obesity

Hours of TV watching were found to be positively correlated with BMI, and the risk of overweight (Eisenmann et al., 2002). Logistic regression analysis has shown lower precedence of overweight among adolescents who watched TV for lesser period per week combined with frequent moderate to vigorous physical activity compared to those more addicted to television and less inclined to physical activity (Gordon- Larsen et al., 2002). The association between TV viewing and overweight was stronger in girls (OR = 2.45, 95% C.I. 1.51 – 3.97) than in boys (OR = 1.52 95% C.I. 1.08 – 2.14) (Gordon-Larsen et al., 2002). It is important to mention in this context that about 27.5% of adolescents aged 10-15 years of Sri Lanka watched television for more than 2 hours per day (Jayatissa and Ranbanda, 2006). One fourth of US teenagers (14 – 18 years old) watch television e" 4-hrs/school day.

Air pollution and child development

Exposure of pregnant women to elevated levels of PAHs including benz(a)pyrene is thought to be associated with reduced fetal growth (Perera et al., 2005). A comparative study of vitamin D status in 9-24 month old children of more polluted Mori Gate area of Delhi and less polluted Gurgaon was carried out by Agarwal et al., (2002). They found 54% reduction in vitamin D level in blood of the children residing at more-polluted Mori Gate, as compared with less-polluted Gurgaon. Absorption of ultraviolet –B (UVB) photons of sunlight by skin is required for vitamin D synthesis by converting 7-dehydrocholesterol to cholecalciferol (vitamin D_3).

Air pollution and the nervous system

About half of the world's children reside in the South and Southeast Asia. These regions are rapidly undergoing industrialization. It is generally believed that threats to children's health, both physical and mental, are increasing in the process (Suk et al., 2003). Human and animal studies have confirmed that brain is a target organ for several environmental pollutants. Developing brain is particularly vulnerable to toxic chemical insults, and such insults may have long-lasting or even irreversible developmental consequences (Winneke, 2005). Prominent among these neurotoxic chemicals are some heavy metals like lead and mercury, and polyhalogenated aromatic hydrocarbons (PHAHs) such as polychlorinated biphenyls (PCBs) and dioxins (Kilburn, 2000).

Air pollution is also known to cause brain damage (Calderon-Garciduenas et al., 2004). Central nervous system (CNS) develops in orderly, synchronized fashion in four steps viz. proliferation and migration of neurons and glial cells, neuronal differentiation, and myelinization. By the end of 12th week of gestation, brain development of the child goes in full swing. Brain damage during these early stages of CNS development gives rise to gross structural anomalies. The regulation of brain development is partly under hormonal control, and hypothalamic-pituitary-thyroid axis plays an important role in this regard (Porterfield, 1994). Hypothyroid conditions during pregnancy or in the neonatal stage, if left untreated, often results in cretinism associated with mental retardation. This is one of the mechanism by which chemicals interacting with the endocrine system may interfere with brain development and associated neurobehavioral dysfunction in the child.

Lead is an important neurotoxic substance found in abundance in urban air. In Europe, air lead level varies between 0.15 to 0.5 $\mu g/m^3$. It has been estimated that for each 1 $\mu g/m^3$ of lead in air

blood lead level becomes 19 µg/liter (WHO, 2000). Lead is present in urban house dust (5.9-127.9 µg/g in Germany), and surface soil and road/pavement dust (100-600 µg/g) [Brunekreef et al., 1981]. In addition to inhalation, diet is the single most important route exposure in children. Lead in drinking water from lead pipes in old houses may add substantially to the dietary lead exposure.

Children are more sensitive to lead because, on a body weight basis, their dietary intake, absorption, and total retention are markedly higher (Winneke, 2005). In addition, the pollutants can reach the brain easily from circulation, because the blood-brain barrier is not yet fully developed in children. Cross-sectional studies in 6-16-year-old children from six countries-China (Wang et al., 2002), Croatia (Prip-Majic et al., 2000), Mexico (Calderon et al., 2001), Pakistan (Rahaman et al., 2002), Saudi Arabia (Al Saleh et al., 2001), and the United States (Lanphear et al., 2000), however, have elicited conflicting results: four found significant and borderline negative associations between blood lead level and IQ and two found no effect. In contrast, significant negative association between blood lead and children's IQ has been obtained in more recent prospective studies (Wasserman et al., 2000; Schnass et al., 2006). A study in Rochester, New York is important in this context because it has demonstrated lead-related IQ deficits below the presumed critical level of 100µg per liter of blood (Canfield et al., 2003). The study found an average of 0.46-point deficit in IQ evaluated by Stanford-Binet intelligence test for each 10µg/liter of blood lead.

Besides IQ, an association between bone lead level and antisocial behavior and delinquency has been reported (Needleman et al., 1979). Like lead, mercury has been linked to neurobehavioral disorders in children, but the strength of evidence in favor the association is weak than that for lead (Winneke, 2005). Lead emissions from gasoline combustion have been a real threat for children in Southeast Asian countries. However, decline in blood lead level in children has been achieved in Thailand, Philippines, India and Pakistan after the introduction of unleaded gasoline in a few years back (Suk et al., 2003).

Prevalence of attention-deficit hyperactivity disorder

In the present study ADHD was diagnosed in 6.7% of schoolchildren of Delhi against 2.7% in controls, suggesting 2.5-times higher prevalence in Delhi. However, factors like stress which I smore prevalent inurban areas also needs to be considered. To our knowledge this is the first cross-sectional as well as comparative study on the instance of ADHD in school-going children of rural and urban India. In two previous reports in Delhi in the 1990's, 5% (Sidana et al., 1998) and 11.2% (Bhatia et al., 1991) children attending pediatric outpatients' clinics had been diagnosed with ADHD. Similar study in Kolkata found 15.5% ADHD cases in referred children attending pediatric hospital (Mukhopadhyay et al., 2003). The authors found 4-times more ADHD prevalence in boys than the girls, and the 'inattentive' subtype was predominant. These observations are consistent with the present findings. Compared to these studies on referred cases, however, our study was cross-sectional, and still we got ADHD prevalence as high as 6.7%. It implies that the problem has probably increased in the intervening years. Present findings are also compatible with the reports from USA, where ADHD prevalence in school-aged varies between 2% to 18% in a community (CDC, 2005). A recent study in Texas has reported 5.44% ADHD in elementary schoolchildren (Schneider and Eisenberg, 2006). CDC (2005) has estimated that 4.4 million U.S. children aged 4-17 years had a diagnosis of ADHD in 2003; of these 2.5 million were under medication for the disorder. In Taiwan, 9.9% primary schoolchildren had this problem and 3.3-times higher prevalence was found in boys than in girls (Wang et al., 1993). Besides air pollution, the stress of urban living

could have played a role in eliciting greater prevalence of ADHD among the schoolchildren of Delhi.

Health impact of ADHD

ADHD has a significant impact on multiple domains of health-related quality of life (HRQL) in children and adolescents. They have impaired learning ability, decreased self-esteem, social problems, family difficulty and potential long-term effects. In addition, the problems of children with ADHD have a significant impact on parents' emotional health and parents' time to meet their own needs, and they interfered with family activities and family cohesion (Klassen et al., 2004). Recent study has shown that adolescent with ADHD are more prone to all kind of accidents (Brook and Boaz, 2006).

Etiology and possible association with air pollution

It has been hypothesized that ADHD is due to noncortical dysfunction that manifests early in ontogeny (Halperin and Schulz, 2006), but the precise etiology of ADHD is currently unknown. Stress could be a risk factor, because increased pressure for school performance is an important contributing factor for ADHD (Schneider and Eisenberg, 2006). Maternal smoking is another independent risk factor for ADHD in offspring (Linnet et al., 2003; Laucht and Schmidt, 2004; Button et al., 2005). Children of these mothers also exhibit antisocial behavior (Button et al., 2005). By analogy, chronic inhalation of highly polluted air of Delhi by the would-be mothers appears to be a risk factor for ADHD in their offspring.

Importance and significance of the study

Since most of the airway diseases take a long latent period to develop, prevalence of respiratory symptoms, lung function tests, and identification of cellular changes in the airways through sputum cytology are immensely helpful to identify the children at risk so that the disease can be diagnosed at an early stage and medical intervention can be initiated for better control of the disease. The tests that have been used in this study are simple, non-invasive, cost-effective and highly sensitive. Therefore, they are ideally suited for biomonitoring of health effects of air pollution in the developing countries like India.

One of the limitations of this study is that the pollution exposed and control population had a major difference of being urban and rural background respectively. Besides air pollution, there could be several other factors related to lifestyle that could have influenced the observed changes in outcome variables such as obesity, hypertension, blood profile and behavior of the children. These possibilities need to be taken into consideration while exploring the health impact of air pollution. Therefore the findings of the present study and the conclusions made thereof require further confirmation by follow up studies with appropriate study design.

CHAPTER-9.0

SUMMARY AND RECOMMENDATIONS

9.1 SUMMARY

Health hazards associated with chronic exposures to urban air pollution are now well recognized. Although air pollution affects the population in general, children are particularly vulnerable to toxic effects of air pollution because of more outdoor exposures, greater intake of air in relation to body weight, lower breathing zone where particulate concentration is higher and, most importantly, a child's underdeveloped immunity. Air pollution-related health effects ranged from low birth weight, reduced lung development and lung function to behavioral alterations. Compared with the developed nations, however, very little is known about the health effects of urban air pollution on children's health in developing countries including India. One of the main objectives of Air Act is to protect human health from air pollution. Keeping this in mind Central Pollution Control Board initiated a detailed study on health impact of air pollution on children with Chittaranjan National Cancer Institute. The study was carried out through questionnaire survey and study of detailed physiological parameters. The major findings of the study are summarized as below:

9.1.1 Measurement of ambient air quality of Delhi

(a) Mean concentration of total suspended particular matter (SPM) in Delhi's air during 2002-2005 was 370 µg/m3 in residential areas, 396 µg/m3 in industrial areas, and 514 µg/m3 in traffic intersection point at ITO.

(b) Mean concentrations of the respirable suspended particulate matter (RSPM, particulate matter with less than 10 µm diameter, PM10) during this period were 142, 165, and 250 µg/m3 in residential, industrial, and traffic intersection point respectively.

(c) Mean concentrations of sulfur dioxide (SO2) and nitrogen dioxide (NO2) in Delhi's air during 2002-2005 were 10 and 47 µg/m3 respectively. In the control areas the concentrations of SO2 and NO2 were 5.6 and 30.3 µg/m3 respectively. The levels of these two pollutants were within the Standard in Delhi as well as in control areas.

(d) A small decline in the concentrations of SPM and RSPM in ambient air has been recorded in residential areas of Delhi during 2002-05.

(e) The average concentration of benzo(a)pyrene in ambient air of Delhi was 3.70 ng/m3 during December 2004 and January 2005. The concentration was highest at ITO (7.31 ng/m3).

(f) Benzene levels were 7.8 µg/m3 in residential areas of Delhi in 2004. Highest concentration was found in traffic intersection point at ITO.

9.1.2 Prevalence of respiratory and associated symptoms

(a) The prevalence of respiratory and associated symptoms was investigated in 11,628 children from 36 schools in Delhi and 4536 control children from two schools in Uttaranchal and 15 from rural West Bengal. The children were aged between 4 and 17 years, and 55% of them were 12-14 year old. Two-third of the children was boys, and one-third was girls. Respiratory symptom data were collected through specially designed structured questionnaire based on three validated questionnaires of BMRC, ATS-DLD-78-C and IUATALD.

(b) Upper respiratory symptoms (URS) like sinusitis, running or stuffy nose, sneezing, sore throat and common cold with fever were 1.8-times more (23.1% vs. 14.6%) prevalent in Delhi than in controls, and the girls suffered more than the boys.

(c) Children in Delhi had 2-times more (17 % vs. 8%) lower respiratory symptoms (LRS) such as frequent dry cough, sputum-producing cough, wheezing breath, breathlessness on exertion, chest pain or tightness and disturbed sleep due to breathing problems. Thus, compared with control, Delhi's children had 1.8- times more URS and 2-times more LRS suggesting higher prevalence of underlying respiratory diseases.

(d) Prevalence of current asthma was present in 4.6% children of Delhi against 2.5% of controls. Similarly, the instance of physician-diagnosed asthma was 2 times more in Delhi (1.7 vs. 0.9%).

(e) About 15% of Delhi's children had frequent eye irritation compared with only 4% in controls. Similarly, Delhi's children had significantly higher prevalence of frequent headache (27.4 vs. 11.8%), nausea (11.2 vs. 5.6%), and palpitation (7.2 vs. 3.3%) and fatigue (12.9 v. 6.7%).

(f) Respiratory and associated symptoms were most prevalent in children from low socio-economic status, and least in children from families with high socio-economic background.

(g) The symptoms were more prevalent in children during winter when PM_{10} level in air is highest in a year, and lowest during monsoon when particulate air pollution level is lowest, suggesting a positive association with particulate air pollution.

9.1.3 Effect of Delhi's air pollution on children's lung function

(a) Lung function test was conducted in schoolchildren aged 9-17 years by portable spirometer following the protocol of American Thoracic Society. A total number of 2245 control and 5671 Delhi's children successfully completed the test.

(b) The results showed reduction of lung function in 43.5% schoolchildren of Delhi compared with 25.7% in control group. Delhi's children had increased prevalence of restrictive (20.3% vs 14.3% in control), obstructive (13.6% vs. 8%), as well as combined (both restrictive and obstructive) type of lung functions deficits (9.6% vs. 3.5%).

(c) Lung function reduction was more prevalent in girls than the boys both in rural and urban settings. In Delhi, 51% of the girls had reduced lung function compared with 39.8% of age-matched boys. In control group, 28.1% of the girls had lung function deficits compared with 24.4% of the boys.

(d) The prevalence of lung function reduction in Delhi was highest (44.9%) among schoolchildren in the age group of 15 – 17 years. Children belonging to 9-11 year and 12-14 year age group had 44.5% and 41.7% prevalence of lung function deficits respectively. On the other hand, highest prevalence of lung function reduction in control group (29.6%) was found in 12-14 year age group, followed by 26.8% in 15-17 year-old, and 23.2% in 12-14 year-old children.

(e) Besides higher prevalence, the magnitude of lung function impairment was much more in Delhi. For example, 7.3% schoolchildren of Delhi had severe lung function deficits compared with 2.2% children in control group.

(f) Prevalence of lung function reduction in schoolchildren varied considerably with season. In control group, prevalence of lung function reduction was highest during winter (32.9%)

when the particulate pollution level in ambient air was highest. Conversely, lowest prevalence of lung function deficits in schoolchildren was recorded in monsoon (19.9%) when the breathing air is cleanest. In Delhi, however, high prevalence of lung function deficits was observed both in winter and summer (52.7% in both seasons), while a much lower prevalence (39.9%) was observed in monsoon. The difference in the prevalence of lung function deficits between winter/summer and monsoon in Delhi was significant ($p<0.05$).

(g) Lower SES had greater percentage of children with reduced lung function.

(h) Exposure to environmental tobacco smoke (ETS) at home due to smoking habit of any member of the family increases the possibility of lung function deficits in children. For example, 50.6% of Delhi's children exposed to ETS had lung function deficits compared with 40.9% non-exposed children. Similarly in control group, 27% children exposed to ETS and 21.3% non-exposed children had lung function deficits.

(i) After controlling potential confounders like season, socioeconomic conditions and ETS, PM_{10} level in ambient air was found to be positively associated with restrictive (OR= 1.35, 95%CI 1.07-1.58), obstructive (OR=1.45, 95% CI 1.16-1.82), and combined type of lung function deficits (OR= 1.74, 95%CI 1.37-2.71) in children.

(j) Based on BMI data, 5.4% children of Delhi enrolled in this study were overweight against 2.4% children in control ($p<0.001$). Besides, 9% of Delhi's children were at risk of being overweight compared with 4.4% children in controls ($p<0.001$). On the other hand, the prevalence of underweight children was greater in the control group. The problem of overweight was most prevalent in children aged between 9 and 11 years in Delhi and 12-14 years in control.

(k) BMI was shown to have profound influence on lung function. Overweight and underweight children had poor lung function than children with normal weight. For instance, 55.6% of overweight children of Delhi had decreased lung function compared with 39.3% children with normal body weight. Delhi's children who were at risk of being overweight demonstrated 48% prevalence of lung function deficits. Underweight children also had higher rate of reduced lung function in Delhi as well as in control group. Thus, children with excess or subnormal body weight appeared to be more prone to lung function deficits.

9.1.4 Assessment of cellular lung reaction to Delhi's air pollution

(a) Sputum samples from 250 school children aged between 13-15 years, 100 (boys 65, girls 35) from control group and 150 from Delhi (boys 98, girls 52) were analyzed. Altogether, 46 sputum sasmples of control children (46% of total) and 35 from Delhi's children (25% of total) were discarded because they were not representative samples of the airways and alveoli. Finally, 54 samples from control and 125 from Delhi's school children were cytologically and cytochemically analyzed and the results were compared.

(b) Microscopical analysis revealed that the sputum samples of Delhi children were more cellular than that of controls: 58.8 cells/hpf were found in the sputum samples of Delhi's children in contrast to 37.2 in the control group. Compared with control children, therefore, Delhi's children had 58% more cells in their sputum ($p<0.05$).

(c) The major cell type in sputum of both control and Delhi children were neutrophils, the

number being higher in Delhi. The number of neutrophils per hpf was 42.2 in Delhi and 30.6 in the control group ($p<0.05$), which may suggest greater prevalence of pulmonary infection and inflammation in the city.

(d) The percentage of eosinophils was also higher in the sputum samples of Delhi's children (3.2% vs. 1.7%) than in control. The rise in eosinophil number is an indication of underlying allergy and hypersensitivity response. Therefore, a greater prevalence of airway allergy may be envisioned among the school children of Delhi.

(e) The mean number of alveolar macrophages (AM) per high power field in Delhi's children was 5.2 in contrast to 1.7 AM per hpf in control. Hence, school children of Delhi had 3.1 times more AM in their sputum. Marked increase in AM number signifies greater exposure to particulate pollution as AM represents the first line of cellular defence against inhaled pollutants.

(f) Sputum of Delhi's children contained 4-times more iron-laden macrophages (siderophages) than controls (0.4 vs. 0.1 siderophage per high power field, $p<0.001$). Abundance of siderophages in lungs may indicate covert pulmonary hemorrhage among a section of school-going children of Delhi.

(g) Changes in the sputum cytology among the school children of Delhi positively correlated with ambient PM_{10} level in Spearman's Rank Correlation, suggesting a close relationship between chronic exposure to Delhi's particulate pollution and cellular changes in the lung ($p<0.001$).

(h) Negative correlation ($p<0.05$) was found between total cells in sputum and spirometric lung measurements like FVC, FEF_{25-75} and PEFR values, thereby indicating that the increase in inflammatory cell population in the airways plays a key role in the development of restrictive type of lung function deficits, and small airway obstruction.

(i) In essence children chronically exposed to high level of ambient air pollution in Delhi are at a higher risk of inflammation and covert hemorrhage in the lungs that may be lead to lung function deficits.

9.1.5 Hematological and vascular changes associated with air pollution exposure

(a) Arterial blood pressure was measured in 1082 school children of Delhi and 726 of controls. The prevalence of hypertension in children was 6.2% in Delhi compared with 2.1% in control. Thus, Delhi's school children have 3-times more incidence of high blood pressure.

(b) Hypertension was more prevalent among girls than the boys: 7.9% of Delhi's girls had hypertension against 4.8% of boys. The prevalence of hypertension increased progressively with age, highest being in the age group of 15 – 17 years.

(c) BMI was positively associated with hypertension: the prevalence of hypertension was lowest (1.8%) in underweight children and highest (16.7%) in obese group.

(d) The absolute numbers of monocytes and basophils were significantly ($p<0.05$) increased in peripheral blood of Delhi's children as compared to that of control.

(e) Rise in hemoglobin concentration and platelet number in Delhi's children were positively

correlated with PM_{10} levels.

(f) Examination of peripheral blood smears revealed abundance of 'target' cells in 9.8% of Delhi's children against 4.3% of controls, implying a greater risk of liver problem in the former.

(g) Higher prevalence of toxic granulation in neutrophils (21.0% vs. 8.7%) and circulating immature neutrophils (11.3% vs. 6.5%) was found among the children of Delhi, which suggests greater risk of infection and inflammation.

(h) In essence school children of Delhi had three times more prevalence of hypertension along with several quantitative and qualitative in peripheral blood that indicates greater possibility of infection and inflammation.

9.1.6 Behavior and activities of the children

(a) Delhi's schoolchildren had 2.5-times more Attention-Deficit Hyperactivity Disorder (ADHD) prevalence than age-and sex-matched controls (6.7% vs.2.7%, $p<0.05$). Boys had a remarkably higher prevalence of ADHD than the girls both in Delhi and in control. Boys: girls ratio of ADHD prevalence was 4.5:1 in Delhi (9% vs. 2%) and 5:1 in control (3.4% vs. 0.7%), and children from medium socio-economic status had the greatest prevalence (8.0%) of ADHD. Among the ADHD children of Delhi, 3.6% had inattentive type, 2.4% had hyperactive-impulsive type, and 0.7% had combined type of ADHD. In the control group, the relative proportions of these three categories were 2.1%, 0.4%, and 0.2% respectively.

(b) Active participation in sports was more prevalent among control children than in Delhi as 80 % of control children regularly participated in sports compared with 58% schoolchildren of Delhi ($p<0.05$). Boys preferred outdoor games to indoor games, while girls showed an inclination towards indoor games.

(c) Delhi' children showed more interest in creative activities such as participation in music (26% vs. 14.4%), drawing or painting (8.2% vs. 3.6%) and dance (7.9% vs. 1.6%) than children of the control group, and the differences were statistically significant ($p<0.05$).

(d) The most preferred pastime of children while at home was watching television. About 54% of Delhi's schoolchildren watched television 1-3 hours per day regularly including school days compared with 45% of control. The boys watched television more than the girls (60.0% vs. 43.1%, $p<0.05$) in Delhi. In contrast, girls of the control group were more addicted to television than the boys (48.0% vs. 43%). Another 12% of Delhi's children spent 1-2 hours per day with the computers and video games. The percentage of computer/ video games users was negligible (0.7%) in the control group.

(e) Reading of books other than schoolbooks, and periodicals was the next preferred pastime for the children. Reading habit was much more prevalent in control group where 40% children had the habit against 24% of city's schoolchildren ($p<0.001$). Girls were more avid readers than the boys in Delhi (28.4% vs. 21.4%, $p<0.05$), but the reverse was true in controls. About 10% of Delhi's children and 17% of control had pastime other than watching television and reading books, of which listening to music was most important (9.8% in Delhi and 16.6% in control).

9.2 RECOMMENDATIONS

1. Children's health is most acutely affected by air pollution exposures. Therefore, potential adverse effects of air pollution on fetus, infant and child should be the main consideration while setting up standard for an air pollutant as well as during revision of existing standards, and there should be a sufficient margin of safety.

2. Epidemiological studies have identified particulates, especially $PM_{2.5}$, as the most harmful air pollutant in South Asia. It is recommended that regular monitoring of this pollutant may be carried out in the country because of its harmful effect on human health.

3. Children generally spend two-third of their time indoor either at home or at school. Therefore, indoor air quality seems to have significant influence on children's health.

4. Ozone is one of the most harmful air pollutants. Children are particularly susceptible to adverse health effects of ozone. Considering its toxicity, regular monitoring of ozone in Delhi and elsewhere in the country is recommended.

5. Ozone is a secondary pollutant. It is not directly emitted from the sources, but formed via complex chemical reactions between oxides of nitrogen, hydrocarbons and sunlight. As a result, ozone levels tend to be highest in the afternoon. Teachers and parents/guardians should be enlightened in the matter.

6. Considering the high prevalence of lung function deficits among schoolchildren of the city, all children aged 9 years and above in Delhi should undergo lung function test at least once in a year, and those having lung function deficits should be monitored at regular intervals. The medical facilities at the schools should include a separate unit for respiratory health, and every unit should be equipped with a spirometer and trained technicians. Where the schools' own medical facilities are absent, the appropriate authority should make alternative arrangements.

7. In view of the high percentages of children with hypertension in Delhi, blood pressure of the children should be checked regularly at the schools' medical facilities or elsewhere at least once in a year. More frequent follow up should be undertaken for those children with hypertension and/or obesity. Since hypertension affects some organ functions, medical conditions such as enlarged heart and abnormalities in kidney and eye functions should also be checked.

8. Since parental smoking has been found to be associated with respiratory problems in their children, the school authority should impress upon the parents/guardians to quit smoking for the sake of the child during parent-teacher meetings.

9. Diesel exhausts contain fine and ultrafine particles that are very harmful particularly for the children. Therefore a child's exposure to diesel fumes should be avoided as far as practicable. In view of this, school buses and other vehicles used for transportation of the school children everywhere in the country should be changed from diesel to cleaner fuel like CNG or LPG.

10. The Government should recognize childhood obesity as a significant problem in Indian cities. It requires intervention by family-based and school-based multi component programs

that include promotion of physical activity, parents' awareness, behavioral counseling, and nutrition education in the line of recommendations of different dietic associations.

11. Fast food and aerated beverages add excess calorie to the body. Delhi already has a large number of overweight and obese children. Obesity is a risk factor for several diseases including problems of the heart and lung.

12. High intake of fresh fruit and vegetables has beneficial effect on overall health as well as functioning of the lungs.

13. In general, city and land use pattern should encourage designing and redevelopment of communities to promote mass transit, carpooling, pedestrian walkways, and bicycle use.

14. While allotting sites for new schools and child care facilities, due considerations should be given on their distance from roads with heavy traffic and other sources of air pollution. In essence, they should be away from hot spots of localized pollution.

15. Information about air pollution in the country- its potential sources, health consequences and possible ways of abatement should be disseminated among the children. They are quick learners and their youthful exuberance can be utilized for spreading the message of environmental safety among the masses to garner public support that holds the key to success for every pollution mitigation projects.

CHAPTER-10.0

REFERENCES

Abbey DE, Burchette RJ, Knutsen SF, MacDonnel WF, Lebowitz MD, Enright TL. Long-term particulate and other air pollutants and lung function in non-smokers. *Am J Respir Crit Care Med* 158, 289-298, 1998.

Ackermann-Liebrich U, Leuenberger P, Schwartz J, Schindler C, Monn C, Bolgnini J, Bongard JP, Brandli O, Domenigatti G, Elsasser S, Grize L, Carrer W, Keller R, Keller-Wossidol H, Kunzli N, Martin BW, Medici TC, Perruchoud AP, Schoni MH, Taschopp JM, Villiger B, Wuthrich B, Zellweger JP, Zemp E. Lung function and long term exposure to air pollutants in Switzerland. Study on air pollution and lung diseases in adults (SATILDIA) Team. *Am J Respir Crit Care Med* 155, 122-129, 1997.

ADAM Inc. Blood pressure in children.

Aekplakorn W, Loomis D, Vichit-Vadakan N, Shy C, Plungchuchon S. Acute effects of SO2 and particles from a power plant on respiratory symptoms of children, Thailand. *South East Asian J Trop Med Public Health* 34, 906-914, 2003.

Aekplakorn W, Loomis D, Vichit-Vadakan N, Shy C, Wongtim S, Vitayamon P. Acute effect of SO2 from a power plant on pulmonary function of children, Thailand. *Int J Epidemiol* 32, 854-861, 2003.

Agarwal KS, Mughal MZ, Upadhyay P, Bury JL, Mawer EB, Puliyel JM. The impact of atmospheric pollution on vitamin D status of infant and toddlers in Delhi, India. *Arch Dis Childhood* 87, 111-113, 2002.

Al Saleh I. Nester M, DeVol E, Shinwari N, Muchari L, al-Shahria S. Relationships between blood lead concentrations, intelligence, and academic achievement of Saudi Arabian schoolgirls. *Int J Hyg Environ Health* 204, 165-174, 2001.

American Dietic Association. Position of the American Dietic Association: Individual-, family-, school-, and community- based interventions for pediatric overweight. *J Am Diet Assoc* 106, 925-945, 2006.

Aneja VP, Agarwal A, Roelle PA, Phillip SB, Tong Q, Watkins N, Yablonsky R. Measurements and analysis of criteria pollutants in New Delhi, India. *Environ Int* 27, 35-42, 2001.

Antova T, Pattenden S, Nikiforov B, Leonardi GS, Boeva B, Fletcher T, Rudnai P, Slachtova H, Tabak C, Zlotkowska R, Houthuijs D, Brunekreef B, Holikova J. Nutrition and respiratory health in children in six Central and Eastern European countries. *Thorax* 58, 231-236, 2003.

APA. American Pediatric Association. Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV), Text Revision, Washington DC, American Pediatric Association, 2000.

Arossa W, Spinaci S, Bugiani M, Natale P, Bucca C, de Candussio G. Changes in lung function of children after an air pollution decrease. *Arch Environ Health* 42, 170-174, 1987.

Ashley F, Kannell WB, Sorlie PD, Masson R. Pulmonary function: relation to aging, cigarette habit, and mortality. *Ann Intern Med* 82, 739-745, 1975.

ATS, Standardization of spirometry-1987 update. Statement of the American Thoracic Society. American review of respiratory diseases, 136, 1285-1298, 1987.

Aust AE, Ball JC, Hu AA, Lighty JS, Smith KR, Straccia AM, Veranth JM, Young WC. Particle characteristics responsible for effects on human lung epithelial cells. *Res Rep Health Eff Inst* 110, 1-65, 2002.

Avol EL, Gauderman WJ, Tan SM, Loudon SJ, Peters JM. Respiratory effects of relocating to areas of different air pollution levels. *Am J Respir Crit Care Med* 164, 2067-2072, 2001.

Badami MG. Transport and urban air pollution in India. *Environ Menage* 36, 195-204, 2005.

Balachandran S, Meena BR, Khillare PS. Particle size distribution and its elemental composition in the ambient air of Delhi. *Environ Int* 26, 49-54, 2000.

Bang KM, Gergen PJ, Kramer R, Cohen B. The effect of pulmonary impairment on all-cause mortality in a national cohort. *Chest* 103, 536-540, 1993.

Barker DJP, Osmond C. Childhood respiratory infection and adult chronic bronchitis in England and Wales. *BMJ* 293, 1271-1275, 1986.

Beaty TH, Newill CA, Cohen BH, Tockman MS, Bryant SH, Spurgeon HA. Effects of pulmonary function on mortality. *J Chronic Dis* 38, 703-710, 1985.

Becker S, Soukup J. Coarse (PM(2.5-10)), fine (PM(2.5)), and ultrafine air pollution particles induce/increase immune costimulatory receptors on human blood-derived monocytes but not on alveolar macrophages. *J Toxicol Environ Health Part A* 66, 847-859, 2003.

Becker S, Soukup JM. Exposure to urban air particulates alters the macrophage – mediated inflammatory response to respiratory viral infection. *J Toxicol Environ Health* 57, 445-457, 1999.

Behera D, Sood P, Singhi S. Respiratory symptoms in Indian children exposed to different cooking fuels. *J Asso Phy Ind* 46, 182-184, 1998.

Behera D, Balamugesh T. Lung cancer in India. *Ind J Chest Dis Allied Sci* 46, 269-281, 2004.

Belfer ML, Shader RI, Di Mascio A, Harmatz JS, Nahum JP. Stress and bronchitis. *BMJ* 3, 805-806, 1968.

Berman S, Duenas A, Bedoya A, Constain V, Leon S, Borrero I, Murphy J. Acute lower respiratory tract illness in Cali, Columbia: a two year ambulatory study. *Pediatrics* 71, 210-218, 1983.

Bernstein AS, Abelson HT. PM2.5- A killer in our midst. *Arch Pediatr Adolesc Med* 159: 786-786, 2005.

Bhatia MS, Nigam VR, Bohra N, Malik SC. Attention deficit disorder with hyperactivity among pediatric outpatients. *J Child Psychol Psychiatry* 32, 297-306, 1991.

Braun-Fahrlander C, Vuille JC, Sennhauser FJ, Keu U, Kunzle T, Grize L, Gasser M, Minder C, Schindler C, Varonier HS, Wuthrich B. Respiratory health and long-term exposure to air pollutants in Swiss school children. SCARPOL team. Swiss study on childhood allergy and respiratory symptoms with respect to air pollution, climate and pollen. *Am J Respir Crit Care Med* 155, 1042-1049, 1997.

Brauer M, Hoek G, Van Vliet P, Meliefste K, Fischer PH, Wijg A, Koopman LP, Neijens HJ, Kerkhif M, Heinrich J, Bellander T, Brunekreef B. Air pollution from traffic and the development of respiratory infections and asthmatic and allergic symptoms in children. *Am J Respir Crit Care Med* 166, 1092-1098, 2002.

Brunekreef B, Veenstra SJ, Biersteker K, Boleij JS. The Arnhem Lead Study I. Lead uptake by 1- to 3- year old children living in the vicinity of a secondary lead smelter in Arnhem, The Netherlands. *Environ Res* 25, 441-448, 1981.

Brunekreef B, Janssen NA, de Hartog J, Harssema H, Knape M, van Vliet P. Air pollution from truck traffic and lung function in children living near motorways. *Epidemiol* 8, 298-303, 1997.

Brook V, Boaz M. Adolescents with attention-deficit hyperactivity disorder/learning disability and their proneness to accidents. *Ind J Pediatr* 73, 299-303, 2006.

Brook RD, Franklin B, Cascio W, Hong Y, Howard G, Lipsett M, Luepker R, Mittleman M, Samet J, Smith SC Jr, Tager I. Expert Panel on Population and Prevention Science of the American Heart Association. Air pollution and cardiovascular disease: a statement for healthcare professionals from the Expert Panel on Population and Prevention Science of the American Heart Association. *Circulation* 109, 2655-2671, 2004.

Broor S, Pandey RM, Ghoh M, Maitreyi RS, Lodha R, Singhal T, Kabra SK. Risk factors for severe acute lower respiratory tract infection in under-five children. *Indian Pediatr* 38, 1361-1369, 2001.

Bunn HJ, Dinsdale D, Smith T, Grigg J. Ultrafine particles in alveolar macrophages from normal children. *Thorax* 56, 932-934, 2001.

Burney PG, Laities LA, Perdrizet S, Huckauf H, Tattersfield AE, Chinn S, Poison N, Heeren A, Britton JR, Jones T. validity and repeatability of the IUATLD (1984) Bronchial Symptom Questionnaire: an international comparison. *Eur Respir J* 2, 940-945, 1989.

Bush RK, Portnoy JM, Saxon A, Terr AI, Wood RA. The medical effects of mold exposure. *J Allergy Clin Immunol* 117, 326-333, 2006.

Button TM, Thapar A, McGuffin P. Relationship between antisocial behavior, attention-deficit hyperactivity disorder and maternal prenatal smoking. *Br J Psychiatry* 187, 155-160, 2005.

Calderon J, Navarro ME, Jimenez-Capdeville ME, Santos-Diaz MA, Golden A, Rodriguez-Leyva I, Borja-Aburto V, Diaz-Barriga F. Exposure to arsenic and lead and neuropsychological development in Mexican children. *Environ Res* 85, 69-76, 2001.

Calderon-Garciduenas L, Osnaya N, Rodriguez-Alcaraz A, Villarreal-Calderon A. DNA damage in nasal respiratory epithelium from children exposed to urban pollution. *Environ Mol Mutagenesis* 30, 11-20, 1997.

Calderon-Garciduenas L, Reed W, Maronpot RR, Henriquez-Roldan C, Delgado-Chavez R, Calderon-Garciduenas A, Dragustinovis I, Franco-Lira M, Aragon-Flores M, Solt AC, Altenburg M, Torres-Jardon R, Swenberg JA. Brain inflammation and Alzheimer's-like pathology in individuals exposed to severe air pollution. *Toxicol Pathol* 32, 650-658, 2004.

Campen MJ, Nolan JP, Schladweiler MC, Kodavanti UP, Evansky PA, Costa DL, Watkinson WP.

Cardiovascular and thermoregulatory effects of inhaled PM-associated transition metals: a potential interaction between nickel and vanadium sulfate. *Toxicol Sci* 64, 243-252, 2001.

Canfield RL, Henderson CR Jr., Cory-Slechta DA, Cox C, Jusko TA, Lanphear BP. Intellectual impairment in children with blood lead concentration below 10 μ per deciliter. *N Eng J Med* 348, 1517-1526, 2003.

Cassel EJ, Lebowitz M, McCarroll JR. The relationship between air pollution, weather and symptoms in an urban population. *Am Rev Respir Dis* 106, 677-683, 1972.

CDC. Centers for disease control and prevention. Mental health in the United States. Prevalence of diagnosis and medication treatment for attention-deficit/hyperactivity disorder-United States, 2003. *MMWR Morb Mortal Wkly Rep* 54, 842-847, 2005.

Chakrabarty A, Sharma SC. Paranasal sinus mycoses. *Ind J Chest Dis Allied Sci* 42, 293-304, 2000.

Chakrabarty A, Sharma SC. Paranasal sinus mycoses. *Ind J Chest Dis Allied Sci* 42, 293-304, 2000.

Chan KN, Elliman A, Bryan E, Silverman M. Respiratory symptoms in children of low birth weight. *Arch Dis Child* 64, 1294-1304, 1989.

Chauhan A, Chatterjee A, Johnston SL. Acute Respiratory Infections. In; Effects of Air Pollution on Children's Health and Development, A review of the evidence. World Health Organization; Special Programme on Health and Environment, European Center for Environment and Health, Bonn pp 44-69, 2005.

Chauhan A, Johnston SL. Air pollution and infection in respiratory illness. *Brit Med Bull* 68, 95-112, 2003.

Chhabra SK, Gupta CK, Chhabra P, Rajpal S. Risk factors for development of bronchial asthma in children in Delhi. *Ann Allergy Asthma Immunol* 83, 385-390, 1999.

Chung A, Chang DP, Kleeman MJ, Perry KD, Cahill TA, Dutcher D, McDougall EM, Stroud K. Comparison of real-time instruments use to monitor airborne particulate matter. *J Air Waste Manag Assoc* 51, 109-120, 2001.

Ciccone G, Forastiere F, Agabiti N, Biggeri A, Bisanti L, Chellini E, Corbo G, Dell'Orco V, Dalmasso P, Volante TF, Galassi C, Piffer S, Renzoni E, Rusconi F, Sestini P, Viei G. Road traffic and adverse respiratory effects in children. SIDRIA Collaborative Group. *Occup Environ Med* 55, 771-778, 1998.

Clancy L, Goodman P, Sinclair H, Dockery DW. Effect of air pollution control on death rates in Dublin, Ireland: an intervention study. *Lancet* 360, 1210-1214, 2002.

Cohen S, Doyle WJ, Turner RB, Alper CM, Skoner DP. Childhood socioeconomic status and host resistance to infectious illness in adulthood. *Psychosom Med* 66, 553-558, 2004.

Colley JRT, Reid DD. Urban and social origins of childhood bronchitis in England and Wales. *BMJ* 2, 213-217, 1970.

Colley JRT, Douglas JWB, Reid DD. Respiratory disease in young adults: influence of early childhood lower respiratory tract illness, social class, air pollution and smoking. *BMJ* 3, 195-198, 1973.

Collins JJ, Kasap HS, Holland WW. Environmental factors in child mortality in England and Wales. *Am J Epidemiol* 93, 10-22, 1971.

Committee of the Environmental and Occupational Health Assembly (CEOHA) of the American Thoracic Society (ATS). Health effects of outdoor air pollution. *Am J Respir Crit Care Med* 153, 3-50, 1996.

Cotes JE. Medical Research Council Questionnaire on Respiratory Symptoms (1986). *Lancet* 2 (8566), 1028, 1987.

Cropper ML, Simon NB, Alberini A, Sharma PK. The health effects of air pollution in Delhi, India. Policy Research Working Paper. The World Bank Development Research Group, 1860, 1-42, 1997.

CSE. Center for Science and Environment. A report on the independent inspection of fuel quality at fuel dispensing stations, oil tanks and tank lorries. March 2002. Available at <http://www.cseindia.org/html/cmp/air/Fnladul.pdf.

Dales RE, Spitzer WO, Suissa S, Schechter MT, Tousignant P, Steinmetz N. Respiratory health of a population living downwind from natural gas refineries. *Am Rev Respir Dis* 139, 595-600, 1989.

Damstra T. Potential effects of certain persistent organic pollutants and endocrine disrupting chemicals on the health of children. *J Toxicol Clin Toxiol* 40, 457-465, 2002.

Daniels MJ, Dominici F, Samet JM, Zeger SL. Estimating particulate matter-mortality dose-response curves and the threshold levels: an analysis of daily time-series for the 20 largest US cities. *Am J Epidemiol* 152, 397-406, 2000.

Dassen W, Brunekreef B, Hoek G, Hofschreuder P, Staatsen B, de Groot H, Schouten E, Biersteker K. Decline in children's pulmonary function during an air pollution episode. *J Air Poll Con Asso* 36, 1223-1227, 1986.

Datta N, Kumar V, Kumar L, Singhi S. Application of a case management approach to the control of acute respiratory infections in low birth weight infants: a feasibility study. *Bull World Health Organ* 65, 77-82, 1987.

De Nijs PF, Ferdinand RF, de Bruin EI, Dekker MC, van Duijn CM, Verhulst DC. Attention-deficit / hyperactivity disorder (ADHD): parents' judgment about school, teachers' judgment about home. *Eur Child Adolesc Psychiatry* 13, 315-320, 2004.

Devalia JL, Campbell AM, Sapsford RJ, Rusznak C, Quint D, Godard P, Bousquet J, Davies RJ. Effect of nitrogen dioxide on synthesis of inflammatory cytokines expressed by human bronchial epithelial cells in vitro. *Am J Respir Cell Mol Biol* 9, 271-278, 1993.

Dockery DW, Ware JH, Ferris BG Jr, Speizer FE, Cook NR, Herman SM. Changes in pulmonary function in children associated with air pollution episodes. *J Air Poll Control Asso* 32, 937-942, 1982.

Dockery DW, Ware JH, Ferris BG Jr., Glicksberg DS, fay ME, Spiro A 3rd, Speizer FE. Distribution of forced expiratory volume in one second and forced vital capacity in healthy, white, adult never-smokers in six U.S. cities. *Am Rev Respir Dis* 131, 511-520, 1985.

Dockery DW, Speizer FE, Stram DO, Ware JH, Spengler JD, Farris BG Jr. Effects of inhalable particles on respiratory health of children. *Am Rev Res Dis* 139, 587-594, 1989.

Dockery DW, Pope CA III, XU X . An association between air pollution and mortality in six U.S. cities. *N Eng J Med* 329, 1753-1759, 1993.

Dockery DW, Skerrett PJ, Walters D, Gilliland F. Development of lung function. In: *Effects of air pollution on children's health and development : A review of the evidence*. World Health Organization Special Programme on Health and Environment . European Centre for Environment and Health. Bonn, pp. 108-133, 2005.

De Paula Santos U, Braga AL, Giorgi DM, Pereira LA, Grupi VJ, Lin CA, Bussacos MA, Zanetta DM, do Nascimento Saldiva PH, Filho MT. Effect of air pollution on blood pressure and heart rate variability: a panel study of vehicular traffic controllers in the city of Sao Paulo, Brazil. *Eur Heart J* 26, 193-200, 2005.

Delfino RJ, Zeiger RS, Seltzer JM, Street DH. Symptoms in pediatric asthmatics and air pollution: differences in effects by symptom severity, anti-inflammatory medication use and particulate averaging time. *Environ Health Perspect* 106, 751-761, 1998.

Delfino RJ, Zeiger RS, Seltzer JM, Street DH, McLaren CE. Association of asthma symptoms with peak particulate air pollution and effect modification by anti inflammatory medication use. *Environ Health Perspect* 110A, 607-617, 2002.

Denny FW, Clyde WA. Acute lower respiratory tract infections in non-hospitalized children. *J Pediatr* 108, 635-646, 1986.

deOnis M, Blossner M. Prevalence and trends of overweight among preschool children in developing countries. *Am J Clin Nutr* 72, 1032-1039, 2000

Devlin RB, Ghio AJ, Kehrl H, Sanders G, Cascio W. Elderly humans exposed to concentrated air pollution particles have decreased heart rate variability. *Eur Respir J Suppl* 40, 76S-80S, 2003.

Dijkstra L, Houthuijs D, Brunekreef B, Akkerman I, Boleij JS. Respiratory health effects of the indoor environment in a population of Dutch children. *Am Rev Res Dis* 142, 1172-1178, 1990.

Diociaiuti M, Balduzzi M, deBerardio B, Cattani G, Stacchini G, Ziemacki G, Marconi A, Paoletti L. The two PM(2.5) (fine) and PM (2.5 – 10) (Coarse) fractions: evidence of different biological activity. *Environ Res* 86, 254-262, 2001.

Donaldson K, Gilmour MI, MacNee W. Asthma and PM10. *Respir Res* 1, 12-15, 2000.

Don Porto Carero A, Hoet PH, Verschaeve L, Schoeters G, Nemery B. Genotoxic effects of carbon black particles, diesel exhaust particles and urban air particulates and their extracts on a human alveolar epithelial cell line (A549) and a human monocytic cell line (THP-1). *Environ Mol Mutagen* 37, 155-163, 2001.

Eastman A, Barry MA. The origin of DNA breaks: a consequence of DNA damage, DNA repair or apoptosis? *Cancer Invest* 10, 229-240, 1992.

Eisner MD. Environmental tobacco smoke exposure and pulmonary function among adults in NHANES III: Impact of the general population and adults with current asthma. *Environ Health Perspect* 110, 765-770, 2002.

Eisenmann JC, Bartee RT, Wang MQ. Physical activity, TV viewing and weight in US youth: 1999 Youth risk behavior survey. *Obes Res* 10, 379-385, 2002.

Enright PL, Linn WS, Avol EL, Margolis HG, Gong H Jr. Peters JM. Quality of spirometry test performance in children and adolescents. *Chest* 118, 665-671, 2000.

EPCA. Environmental Pollution (Prevention and Control) Authority for the National Capital Region. The imperative of restricting vehicle numbers in Delhi: designing a parking policy to manage travel demand in the city. July, 2004.

Eroshina K, Danishevski K, Wilkinson P, McKee M. Environmental and social factors as determinants of respiratory dysfunction in junior schoolchildren in Moscow. *J Public Health (Oxf)* 26, 197-204, 2004.

ESMAP. Health impacts of outdoor air pollution. Urban Air Pollution. South Asia Urban Air Quality Management Briefing Note No.11, February 2003. Available at <http://www.worldbank.org/sarurbanair>.

ESMAP. Energy Sector Management Assistance Programme. UNDP/World Bank. Urban Air Pollution. South Asia Urban Air Quality Management Briefing Note No.7, July 2002. Available at http://www.worldbank.org/sarurbanair.

Ferraro KF, Thorpe RJ Jr., Wilkinson JA. The life course of severe obesity: does childhood overweight matter? Journal of Gerontology: *Soc Sciences* 58, S110–S119, 2003.

Ferris BG. Epidemiology standardization project. *Am Rev Respir Dis* 118, 1-88, 1978.

Finland M. Pneumococcal infections. In: Evans AS, Feldman HA, eds. Bacterial infections in humans. Epidemiology and control. New York: Plenum, 1982.

Fletcher C, Peto R. The natural history of chronic airflow obstruction. *Br Med J* 1, 1645-1648, 1977.

Forastiere F, Galassi C, Biggeri A, Richiardi L, Baussano I, Simoni M, Viegi G; Gruppo Collaborativo SIDRIA-2. The proportion of respiratory disorders in childhood attributable to preventable and not preventable risk factors. *Epidemiol Prev* 29 (Suppl 2), 67-69, 2005.

Foulke FG, Reeb KG, Graham AV, Zyzanski SJ. Family function, respiratory illness and otitis media in urban black infants. *Fam Med* 20, 128-132, 1988.

Fox JP, Hall CE, Cooney MK, Luce RE, Kronmal RA. The Seattle virus watch II. Objectives, study population and its observation, data processing and summary of illnesses. *Am J Epidemiol* 96, 270-285, 1972.

Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. The relation of overweight to cardiovascular risk factors among children and adolescents: The Bogalusa Heart Study. *Pediatrics* 103, 1175–1182, 1999.

Friedman GD, Klatsky AL, Siegelaub AB. Lung function and risk of myocardial infarction and sudden cardiac death. *N Eng J Med* 294, 1071-1075, 1976.

Frischer T, Studnicka M, gartner C, Tauber E, Horak F, Veiter A, Spengler J, Kuhr J, Urbanek R. Lung function growth and ambient ozone: a three-year population study in school children. *Am J Respir Crit Care Med* 160, 390-396, 1999.

Fritz GJ, Herbarth O. Pulmonary function and urban air pollution in preschool children. *Int J Hygiene Environ Health* 203, 235-244, 2001.

Frostad A, Soyseth V, Andersen A, Gulsvik A. respiratory symptoms as predictors of all-cause mortality in an urban community: a 30-year follow up. *J Intern Med* 259, 520-529, 2006.

Frye C, Hoelscher B, Cyrys J, Wjst M, Wichmann HE, Heinrich J. Association of lung function with declining ambient air pollution. *Environ Health Perspect* 111, 383-387, 2003.

Frye C, Heinrich J. Trends and predictors of overweight and obesity in East German children. *Int J Obes Relat Metab* 27, 963-969, 2003.

Galizia A, Kinney PL. Long term residence in areas of high Ozone: associations with respiratory health in a nation wide samples of non smoking young adults. *Environ Health Perspect* 107, 675-679, 1999.

Gauderman WJ, McConnell R, Gilliland F, London S, Thomas D, Avol E, Vora H, Berhane K, Rappaport EB, Lurmann F, Margolis HG, Peters J. Association between air pollution and lung function growth in southern California children. *Am J Respir Crit Care Med* 162, 1383-1390, 2000.

Gauderman WJ, McConnell R, Gilliland F. Association between air pollution and lung function growth in southern California children. *Am J Respir Crit Care Med* 162, 1383-1390, 2002 a.

Gauderman WJ, Gilliland F, Vora H, Avol E, Stram D, McConnell R, Thomas D, Lurmann F, Margolis HG, Rappaport EB, Berhane K, Peters JM. Association between air pollution and lung function growth in southern California children: results from a second cohort. *Am J Respir Crit Care Med* 166, 76-84, 2002 b.

Gauderman WJ, Avol E, Gilliland F, Vora H, Thomas D, Berhane K, McConnell R, Kuenzli N, Lurmann F, Rappaport E, Margolis H, Bates D, Peters J. The effect of air pollution on lung development from 10 to 18 years of age. *N Eng J Med* 351, 1057-1067, 2004.

Gavett SH, Haykal-Coates N, Copeland LB, Heinrich J, Gilmour MI. Metal composition of ambient PM 2.5 influences severity of allergic disease in mice. *Environ Health Perspect* 111, 1471-1477, 2003.

Gehring U, Pattenden S, Slachtova H, Antova T, Braun-Fahrlander C, Fabianova E, Fletcher T, Galassi C, Hoek G, Kuzmin SV, Luttmann-Gibson H, Moshammer H, Rudnai P, Zlotkowska R, Heinrich J. Parental education and children's respiratory and allergic symptoms in the Pollution and the Young (PATY) study. *Eur Respir J* 27, 95-107, 2006.

Georgieva T, Michailova A, Panev T, Popov T. Possibilities to control the health risk of petrochemical workers. *Int Arch Occup Environ Health* 75, 1-6, 2002.

Gilliland FD, Berhane K, McConnell R, Gauderman WJ, Vera H, Rappaport EB, Avol E, Peters JM. Maternal smoking during pregnancy, environmental tobacco smoke exposure and childhood lung function. *Thorax* 55, 271-276, 2000.

Gilliland FD, McConnell R, Peters J, Gong H Jr. A theoretical basis for investigating ambient air pollution and children's respiratory health. *Environ Health Perspect* 107, 403-407, 1999.

Gilliland HE, Armstrong MA, McMurray TJ. The inflammatory response to pediatric cardiac surgery: correlation of granulocyte adhesion molecule expression with postoperative oxygenation. *Anesth Analg* 89, 1188-1191, 1999.

Glew RH, Brock HS, Vander Voort J, Agaba P, Harkins MS, Vander Jagt DJ. Lung function and nutritional status of semi-nomadic Fulani children and adolescents in Northern Nigeria. *J Trop Pediatr* 50, 20-25, 2004.

Glezen W, Denny FW. Epidemiology of acute lower respiratory disease in children. *N Engl J Med* 288, 498-505, 1973.

Gold DR, Litonjua A, Schwartz J, Lovett E, Larson A, Nearing B, Allen G, Verrier M, Cherry R, Verrier R. Ambient pollution and heart rate variability. *Circulation* 101, 1267-1273, 2000.

Golde DW, Drew WL, Klein HZ, Finley TN, Cline MJ. Occult pulmonary hemorrhage in leukaemia. *Br Med J* 2, 166-168, 1975.

Golshan M, Mohammad –Zadeh Z, Khanlar- Pour A, Iran- Pour R. Prevalence of asthma and related symptoms in junior school children in Isfahan, Iran. *Monaldi Arch Chest Dis* 57, 17 – 22, 2002.

Gordon- Larsen P, Adair LS, Popkin BM. Ethnic difference in physical activity and inactivity patterns and overweight status. *Obes Res* 10, 141-149, 2002

Graham MH. Psycological factors in the epidemiology of acute respiratory infection. MD thesis. Adelaide, South Australia: University of Adelaide, 1987.

Graham MH. The epidemiology of acute respiratory infections in children and adults: A global perspective. *Epidemiol Rev* 12, 149-178, 1990.

Grubb C. Diagnostic Cytophatology: A Text and Colour Atlas. Churchill Livingstone, London, pp. 65-112, 1994.

Guerra S, Sherrill DL, Bobadilla A, Martinez FD, Barbee RA. The relation of body mass index to asthma, chronic bronchitis and emphysema. *Chest* 122, 1256-1263, 2002.

Guo YL, Lin YC, Sung FC, Huang SL, Ko YC, Lai JS, Su HJ, Shaw CK, Lin RS, Dockery DW. Climate, traffic-related air pollutants and asthma prevalence in middle-school children in Taiwan. *Environ Health Perspect* 107, 1001-1006, 1999.

Gupta R. Meta-analysis of prevalence of hypertension in India. *Indian Heart J* 49, 43-48, 1997.

Gwaltney JM Jr, Hendley JO, Simon G, Jordon WS Jr. Rhinovirus infections in an industrial population. I. The occurrence of illness. *N Engl J Med* 275, 1261-1268, 1966.

Gwaltney JM. Epidemiology of the common cold. *Ann N Y Acad Sci* 353, 54-60, 1980.

Gupta P. Nutritional and lung function profile of boy belonging to east Delhi. *J Indian Med Assoc* 95, 176-178, 1997.

Hajat S, Haines A, Goubet SA, Atkinson RW, Anderson HR. Association of air pollution with daily GP consultations for asthma and other lower respiratory condition in London. *Thorax* 54, 597-605, 1999.

Hajat S, Haines A, Atkinson RW, Bremmer SA, Anderson HR, Emberlin J. Association between air pollution and daily consultations with general practitioners for allergic rhinitis in London, United Kingdom. *Am J Epidemiol* 153, 704-714, 2001.

Hajat S, Anderson HR, Atkinson RW, Haines A. Effects of air pollution on general practitioners consultations for upper respiratory diseases in London. *Occu Environ Med* 59, 294-299, 2002.

Halperin JM, Schulz KP. Revisiting the role of prefrontal cortex in the pathophysiology of attention-deficit hyperactivity disorder. *Psychol Bull* 132, 560-581, 2006.

Harris JR, Magnus P, Samuelsen SO, Tamdes K. No evidence for effects of family environment on asthma; a retrospective study of Norwegian twins. *Am J Respir Crit Care Med* 156, 43-49, 1997.

Harrod KS, Jaramilo RJ, Rosenberger CL, Wang SZ, Berger JA, McDonald JD, Reed MD. Increased susceptibility RSV infection by exposure to inhaled diesel engine emissions. *Am J Respir Cell Mol Biol* 28, 451-463, 2003.

Hart H, Bax M, Jenkins S. Health and behaviour in preschool children. *Child Care Health Dev* 10, 1-16, 1984.

He JQ, Ruan J, Connett JE, Anthonisen NR, Pare PD, Sanford AJ. Antioxidant gene polymorphisms and susceptibility to a rapid decline in lung function in smokers. *Am J Respir Crit Care Med* 166, 323-328, 2002

Helleday R, Huberman D, Blomberg A, Stjernberg N, Sanstrom T. Nitrogen dioxide exposure impairs the frequency of the mucocilliary activity in healthy subjects. *Eur Respir J* 8, 1664-1668, 1995.

Henderson RF. Use of bronchoalveolar lavage to detect lung damage. In: *Target organ toxicology-lung*. New York: Raven Press, p. 239, 1988.

Hirsch T, Weiland SK, von Mutius E, Safeca AF, Grafe H, Csaplovics E, Duhme H, Keil U, Leupold W. Inner city air pollution and respiratory health and atopy in children. *Eur Respir J* 14, 669-677, 1999

Hiura TS, Kaszubowski MP, Li N, Nel AE. Chemicals in diesel exhaust particles generate reactive oxygen radicals and induce apoptosis in macrophages. *J Immunol* 163, 5582-5591, 1999.

Hoek G, Brunekreef B. Effects of low level winter air pollution concentrations on respiratory health of Dutch children. *Environ Res* 64, 136-150, 1994.

Hoek G, Brunekreef B, Goldbohm S, Fischer P, van den Brandt PA. Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study. *Lancet* 360, 1203-1209, 2002.

Hogervorst JGF, de Kok TMCM, Briede JJ, Wesseling G, Kleinjans JCS, van Schayck CP. Relationship between radical generation by urban ambient particulate matter and pulmonary function of school children. *J Toxicol Environ Health Part A* 69, 245-262, 2006.

Hole DJ, Watt GC, Davey-Smith G, Hart CL, Gillis CR, Hawthorne VM. Impaired lung function and mortality risk in men and women: findings from the Renfrew and Paisley prospective population study. *BMJ* 313, 711-715, 1996.

Holgate ST. Genetic and environmental interaction in allergy and asthma. *J Allergy Clin Immunol* 104, 1139-1146, 1999.

Holgate ST. The epidemic of allergy and asthma. *Nature* 402, (Suppl. 6760), B2-B4, 1999.

Horak F Jr, Studnicka M, Gartner C, Spengler JD, Tauber E, Urbanek R, Veiter A, Frischer T. Particulate matter and lung function growth in children: a 3-year follow-up study in Austrian school children. *Eur Respir J* 19, 838-845, 2002.

Hwang BF, Jaakkola JJ, Lee YL, Lin YC, Guo YL. Relation between air pollution and allergic rhinitis in Taiwanese school children. *Respir Res* 7, 23, 2006.

Hwang BF, Lee YL, Lin YC, Jaakkola JJ Guo YL. Traffic related air pollution as a determinant of asthma among Taiwanese school children. *Thorax* 60, 467-473, 2006.

Hwang JS, Chan CC. Effects of air pollution on daily clinic visits for lower respiratory tract illness. *Am J Epidemiol* 155, 1-10, 2002.

Holland WW, Reid DD. The urban factor in chronic bronchitis. *Lancet* 1, 445-448, 1965.

Hoppin JA, Umbach DM, London SJ, Alvanja MC, Sandler DP. Diesel exhaust, solvents, and other occupational exposures as risk factors for wheeze among farmers. *Am J Resir Crit Care Med* 169, 1308-1313, 2004.

Horak F Jr, Studnicka M, Gartner C, Neumann M, Tauber E, Urbanek R, Veiter A, Frischer T. The effect of inhalable dust particles (PM10) on lung function and respiratory symptoms of school children in lower Austria. [in German]. *Wein Klin Wochenschr* 112, 126-132, 2000.

Hudson R, Arriola A, Martinez-Gomez M, Distel H. Effect of air pollution on olfactory function in resident of Mexico City. *Chem Senses* 31, 79-85, 2006.

Hwang JS, Chan CC. Effects of air pollution on daily clinic visits for lower respiratory tract illness. *Am J Epidemiol* 155, 1-10, 2002.

IANGV. International Natural gas Vehicle Statistics. August 2001. Available at http://www.iangv.org/html/ngv/stats.html

IARC. International Agency for Research on Cancer. *Diesel and gasoline engine exhausts and some nitroarenes.* Lyon, 1989.

Jacobs MA, Spilken AZ, Norman MM, Anderson LS. Life stress and respiratory illness. *Psychosom Med* 32, 233-242, 1970.

Jakobsson R, Ahlbom A, Bellander T, Lundberg I. Acute myeloid leukemia among petrol station attendants. *Arch Environ Health* 51, 469-471, 1996.

James JW. Longitudinal study of the morbidity of diarrheal and respiratory infections in malnourished children. *Am J Clin Nutr* 25, 690-694, 1972.

Jayatissa R, Ranbanda RM. Prevalence of challenging nutritional problems among adolescents in Sri Lanka. *Food Nutr Bull* 27, 153-160, 2006.

Jedrychowski W, Flak E, Mroz E. The adverse effects of low levels of ambient air pollutants on lung function growth in preadolescent children. *Environ Health Perspect* 107, 669-674, 1999.

Jehn ML, Gittelsohn J, Treuth MS, Caballero B. Prevalence of overweight among Baltimore City schoolchildren and its associations with nutrition and physical activity. *Obesity (Silver Spring)* 14, 989-993, 2006.

Kabra SK, Lodha R, Singhal T. Chronic obstructive pulmonary disease in children. *Indian J Pediatr* 68 (Suppl. 2), S50-S54, 2001.

Kamat SR, Patil JD, Gregart J, Dalal N, Deshpande JM, Hardikar P. Air pollution related respiratory morbidity in central and north-eastern Bombay.

J Assoc Physicians India 40, 588-593, 1992.

Khan A. Air pollution hampers teenagers' lung development. *Thorax* 59, 1045-1045, 2004.

Kiecolt-Glaser JK, Glaser R. Psychological influences on immunity. *Psychosomatics* 27, 621-624, 1986.

Kienast K, Knorst M, Muller-Quernheim J, Ferlinz R. Modulation of IL-1 beta, IL-6, IL-8, TNF-alpha, and TGF- beta secretions by alveolar macrophages under NO2 exposure. *Lung* 174, 57-67, 1996.

Kilburn KH. Visual and neurobehavioral impairment associated with polychlorinated biphenyls. *Neurotoxicity* 21, 489-499, 2000.

Kilburn KH. Effects of diesel exhaust on neurobehavioral and pulmonary function. *Arch Environ Health* 55, 11-17, 2000.

Kim JY, Magari SR, Herrick RF, Smith TJ, Christiani DC. Comparison of fine particle measurements from a direct-reading instrument and a gravimetric sampling method. *J Occup Environ Hyg* 1, 707-715, 2004.

Kinney PL, Ware JH, Spengler JD, Dockery DW, Speizer FE, Ferris BG Jr. Short-term pulmonary function change in association with ozone levels. *Am Rev Respir Dis* 139, 56-61, 1989.

Kinney PL, Nilsen DM, Lippmann M, Brescia M, Gordon T, McGovern T, El-Fawal H, Devlin RB, Rom WN. Biomarkers of lung inflammation in recreational joggers exposed to Ozone. *Am J Respir Crit Care Med* 154, 1430-1435, 1996.

Kinney PL, Chae E. Diminished lung function in young adults is associated with long-term PM10 exposures. In: Proceedings of the 14th Conference of the International Society for Environmental Epidemiology, Vancouver, 11-15 August 2002 (http://www.webstracts.com/ISEA 2002/ catsort/ 10891.pdf, accessed 13 April 2005).

Kjellman B, Gustafson PM. Asthma from childhood to adulthood. *Respir Med* 94, 454 – 465, 2000.

Klassen AF, Miller A, Fine S. Health-related quality of life in children and adolescents who have a diagnosis of attention-deficit/hyperactivity disorder. *Pediatrics* 114, 541-547, 2004.

Klig JE, Shah NB. Office pediatrics: current issues in lower respiratory infections in children. *Curr Opin Pediatr* 17, 111-118, 2005.

Knuiman MW, James AL, Divitini ML, Ryan G, Bartholomew HC, Musk AW. Lung function, respiratory symptoms and mortality: results from the Busselton Health Study. *Ann Epidemiol* 9, 297-306, 1999.

Korte JE, Hertz-Picciotto I, Schulz MR, Ball LM, Duell EJ. The contribution of benzene to smoking-induced leukemia. *Environ Health Perspect* 108, 333-339, 2000.

Kostamo K, Richardson M, Malmberg H, Ylikoski J, Ranta H, Toskala E. Does the triad of fungi, bacteria and exposure to moisture have an impact on chronic hyperplastic sinusitis? *Indoor Air* 15, 112-119, 2005.

Kramer U, Behrendt H, Dolger R, Ranft U, Ring J, Willer H, Schlipkoter HW. Airway diseases and allergies in East and West German children during the first five years after reunification. Time trends and the impact of sulfur dioxide and total suspended particles. *Int J Epidemiol* 28, 865-873, 1999.

Krzyzanowski M, Wysocki M. The relation of thirteen-year mortality to ventilatory impairment and other respiratory symptoms: the Cracow Study. *Int J Epidemiol* 15, 56-64, 1986.

Kunzli N, Lurmann F, Segal M, Ngo L, Balmus J, Tager IB. Association between lifetime ambient ozone exposure and pulmonary function in college freshmen – results of a pilot study. *Environl Res* 72, 8-23, 1997.

Lacar ES, Soto X, Riley WJ. Adolescent obesity in a low-income Mexican American district in South Texas. *Arch Pediatr Adolesc Med* 154, 837-840, 2000.

Lahiri T, Roy S, Basu C, Ganguly S, Ray MR, Lahiri P. Air pollution in Calcutta elicits adverse pulmonary reaction in children. *Ind J Med Res* 112, 21-26, 2000.

Lahiri T, Ray MR, Mukherjee S, Basu C, Lahiri P. Marked increase in sputum alveolar macrophages in residents of Calcutta: Possible exposure effect of severe air pollution. *Curr Sci* 78, 399-404, 2000.

Lambert AL, Dong W, Selgrade MK, Gilmour MI.Enhanced allergic sensitization by residual oil fly ash particles is mediated by soluble metal constituents. *Toxicol Appl Pharmacol* 165, 84-93, 2000.

Lanphear BP, Dietrich K, Auinger P, Cox C. Cognitive deficits associated with blood lead concentrations <10 µg/dL in US children and adolescents. *Pub Health Rep* 115, 521-529, 2000.

Laucht M, Schmidt MH. Maternal smoking during pregnancy: risk factor for ADHD in the offspring? *Z Kinder Jugendpsychiatr Psychother* 32, 177-185, 2004.

Lave LB, Seskin EP. Air pollution and human health. *Science* 169, 723-733, 1970.

Law CM, de Swiet M, Osmond C, Fayers PM, Barker DJ, Cruddas AM, Fall CH. Initiation of hypertension in utero and it amplification throughout life. *BMJ* 306, 24-27, 1993.

Lawther PJ, Waller RE, Henderson M. Air pollution and exacerbations of bronchitis. *Thorax* 25, 525-539, 1970.

Lebowitz MD. The trends in AOD morbidity in the Tucson epidemiological study. *Am Rev Respir Dis* 140 S35-S41, 1981.

Lee BE, Ha EH, Park HS, Kim H, Lee HJ, Lee YK, Lee SJ, Hong YC. Air pollution and respiratory symptoms of school children in a panel study in Seoul. *J Prev Med Pub Health* 38, 465-472, 2005.

Lee CR, Yoo CI, Lee JH, Kim SR, Kim Y. Hematological changes of children exposed to volatile organic compounds containing low levels of benzene. *Sci Tot Environ* 299, 237-245, 2002.

Lee YL, Shaw CK, Su HJ, Lai JS, Ko YC, Huang SL, Sung FC, Guo YL. Climate, traffic-related air pollutants and allergic rhinitis prevalence in middle-school children in Taiwan. *Eur Respir J* 21, 964-970, 2003.

Levy D, Gent M, Newhouse MT. Relationship between acute respiratory illness and air pollution levels in an industrial city. *Am Rev Respir Dis* 116, 167-173, 1977.

Lewne M, Nise G, Lind ML, Gustavsson P. Exposure to particles and nitrogen dioxide among taxi, bus and lorry drivers. *Int Arch Occup Environ Health* 79, 220-226, 2006.

Li N, Sioutas C, Cho A, Schmitz D, Misra C, Sempf J, Wang M, Oberley T, Froines J, Nel A. Ultrafine particulate pollutants induce oxidative stress and mitochondrial damage. *Environ Health Perspect* 111, 455-460, 2003.

Li XY, Gilmour TS, Donaldson K, MacNee W. In vivo and in vitro proinflammatory effects of particulate air pollution (PM10). *Environ Health Perspect* 105 (Suppl 5), 1279-1283, 1997.

Liard R, Neukirch F. Questionnaires: a major instrument for respiratory epidemiology. *Eur Respir Mon* 15, 154-166, 2000.

Linnet KM, Dalsgaard Obel C, Wisborg K, Henriken TB, Rodriguez A, Kotimaa A, Moilanen I, Thomsen PH, Olsen J, Jarvelin MR. Maternal lifestyle factors in pregnancy risk of attention-deficit hyperactivity disorder and associated behavior: review of the current evidence. *Am J Psychiatry* 160, 1028-1040, 2003.

Lippmann M. Health effects of ozone: A critical review. *J Air Pollut Control Assoc* 39, 672–695, 1989.

Lippmann M, Spektor DM. Peak flow rate changes in Ozone exposed children: spirometry vs. mini Wright flow meters. *J Expo Anal Environ Epidemiol* 8, 101-107, 1998.

Lunn JE, Knowelden J, Handyside AJ. Patterns of respiratory illness in Scheffield instant school children. *Br J Prev Soc Med* 21, 7-16, 1967.

Lunn JE, Knowelden J, Roe JW. Patterns of respiratory illness in Sheffield infant school children. *Br J Prev Soc Med* 24, 223-228, 1970.

Mei Z, Grummer-Strawn LM, Pietrobelli A, Goulding A, Goran MI, Dietz WH. Validity of body mass index compared with other body-composition screening indexes for the assessment of body fatness in children and adolescents. *Am J Clin Nutr* 7, 597–985, 2002.

Marano F, Boland S, Bonvallot V, Baulig A, Baeza-Squiban A. Human airway epithelial cells in culture for studying the molecular mechanisms of the inflammatory response triggered by diesel exhaust particles. *Cell Biol Toxicol* 18, 315-320, 2002.

Melia RJW, Florey C duV, Darby SC. Differences in NO2 levels in kitchens with gas or electric cookers. *Atmos Environ* 12, 1379-1381, 1978.

Meltzer EO. Allergic rhinitis: managing the pediatric spectrum. *Allergy Asthma Proc* 27, 2-8, 2006.

Meyer RJ, Haggerty RJ. Streptococcal infections in families: factors altering susceptibility. *Pediatrics* 29, 539-549, 1962.

Mistry R, Wickramaingha N, Ogston S, Singh M, Devasiri V, Mukhopadhyay S. Wheeze and urban variation in South Asia. *Eur J Pediatr* 163,145-147, 2004.

MoEF. Ministry of Environment and Forest, Government of India. White Paper On Pollution In Delhi With An Action Plan. New Delhi, 1997.

Mok JYQ, Simpson H. Outcome for acute bronchitis, bronchiolitis and pneumonia in infancy. *Arch Dis Child* 59, 306-309, 1984.

Moller P, Wallin A. Adduct formation, mutagenesis and nucleotide excision repair of DNA damage produced by reactive oxygen species and lipid peroxidation products. *Mutat Res* 410, 271-290, 1998.

Moshammer H, Hutter HP, Hauck H, Neuberger M. Low levels of air pollution induce changes of lung function in a panel of school children. *Eur Respir J* Feb 2, 2006 [Epub ahead of print].

Mukhopadhyay M, Misra S, Mitra T, Niyogi P. Attention deficit hyperactivity disorder. *Indian J Pediatr* 70, 789-792, 2003.

Must A, Anderson SE. Effects of obesity on morbidity in children and adolescents. *Nutr Clin Care* 6, 4–12, 2003.

Mustaphi P, Dobe M. Positive deviance-the West Bengal experience. *Ind J Public Health* 49, 207-213, 2005.

Mylius EA, Gullvag B. Alveolar macrophage count as an indicator of lung reaction to industrial air pollution. *Acta Cytol* 30, 157-162, 1986.

Namork E, Johansen BV, Lovik M. Detection of allergens adsorbed to ambient air particles collected in four European cities. *Toxicol Lett* 165, 71-78, 2006.

NCHS. National Center for Health Statistics. Body mass index-for-age percentiles. Developed in collaboration with the National Center for Chronic Disease Prevention and Health Promotion. May 30, 2000; modified October 16, 2000. Available at http://www.cdc.gov/growthcharts

Neas LM, Schwartz J. Pulmonary function levels as predictors of mortality in a national sample of US adults. *Am J Epidemiol* 147, 1011-1018, 1998.

Needleman HL, Gunnoe C, Leviton A, Reed R, Peresie H, maher C, Barrett P. Deficits in psychological and classroom performance in children with elevated dentine lead levels. *N Eng J Med* 30, 689-695, 1979.

Nelson HS. Advances in upper respiratory airway diseases and allergen immunotherapy. *J Allergy Clin Immunol* 115, 676-684, 2005.

Nobutomo K. Air pollution and cytological changes in sputum. *Lancet* i: 523-523, 1978.

Oberdorster G. Pulmonary effects of inhaled ultrafine particles. *Int Arch Occu Environ Health* 74, 1-8, 2001.

Okeson CD, Riley MR, Fernandez A, Wendt JO. Impact of the composition of combustion generated fine particles on epithelial cell toxicity: influences of metals on metabolism. *Chemosphere* 51, 1121-1128, 2003.

Oliver C, Lewis PR, Stoward PJ. Histochemical methods for esterases. In Stoward P.J. and Pearse, A.G.E. (eds). Histochemistry, Theoretical and Applied Vol III, Churchill, Livingstone, London, pp. 607-618, 1991.

Oosterlee A, Drijver M, Lebret E, Brunekreef B. Chronic respiratory symptoms in children and adults living along streets with high traffic density. *Occup Environ Med* 53, 241-247, 1996.

Ostro B, Lipsett M, Mann J, Braxton-Owens H, White M. Air pollution and exacerbation of asthma in African-American children in Los Angeles. *Epidemiol* 12, 200-208, 2001.

Osur SL. Viral respiratory infections in association with asthma and sinusitis: a review. *Ann Allergy Asthma Immunol* 89, 553-560, 2002.

Pacheco KA, Tarkowski M, Sterritt C, Negri J, Rosenwasser LJ, Borish L. The influence of diesel exhaust particles on mononuclear phagocytic cell derived cytokines: IL-10, TGF-beta and IL-1 beta. *Clin Exp Immunol* 126, 374-383, 2001.

Pandey MR, Boleji JSM, Smith KR, Wafula EM. Indoor air pollution in developing countries and acute respiratory infection in children. *Lancet* 1, 427-429, 1989.

Pande JN, Bhatta N, Biswas D, Pandey RM, Ahluwalia G, Siddaramaiah NH, Khilnani GC. Outdoor air pollution and emergency room visits at a hospital in Delhi.

Indian J Chest Dis Allied Sci 44,13-9, 2002.

Payling-Wright G, Payling-Wright H. Etiological factors in broncho-pneumonia amongst infants in London. *J Hyg (Camb)* 44, 15-30, 1945.

Pearce N, Pekkanen J, Beasley R. How much asthma is really attributable to atopy? *Thorax* 54, 268-272, 1999.

Peltola VT, Boyd KL, McAuley JL, Rehg JE, McCullers JA. Bacterial sinusitis and otitis media following influenza virus infection in ferrets. *Infec Immun* 74, 2562-2567, 2006.

Perera FP, Tang D, Rauh V, Lester K, Tsai WY, Tu YH, Weiss L, Hoepner L, King J, Del Priore G, Lederman SA. Relationships among polycyclic aromatic hydrocarbon-DNA adducts, proximity to the World Trade Center, and effects on fetal growth. *Environ Health Perspect* 113, 1062-1067, 2005.

Perera FP, Rauh V, Tsai WY, Kinney P, Camann D, Barr D, Bernert T, Garfinkel R, Tu YH, Diaz D, Dietrich J, Whyatt RM. Effects of transplacental exposure to environmental pollutants on birth outcomes in a multiethnic population. *Environ Health Perspect* 111, 201-205, 2003.

Perez-Padilla R, Rojas R, Torres V, Borja-Aburto V, Olaiz G. Obesity among children residing in Mexico City and its impact on lung function: a comparison with Mexican-Americans. *Arch Med Res* 37, 165-171, 2006.

Peters A, Wichmann HE, Tuch T, Heinrich J, Heyder J. Respiratory effects are associated with the number of ultrafine particles. *Am J Respir Crit Care Med* 155, 1376-1383, 1997.

Peters A, Dockery DW, Heinrich J, Wichmann HE. Short term effects of particulate air pollution on respiratory morbidity in asthmatic children. *Eur Respir J* 10, 872 – 879, 1997.

Pierse N, Rushton L, Harris RS, Kuchni CE, Silverman M, Grigg J. Locally generated particulate pollution and respiratory symptoms in young children. *Thorax* 61, 216-220, 2006.

Pilotto LS, Douglas RM, Attewall R, Wilson SR. Respiratory effects associated with indoor nitrogen dioxide exposure in children. *Int J Epidemiol* 26, 788-796, 1997.

Pio A, Leowski J, Ten Dam HG. The magnitude of the problem of acute respiratory infections. In: Douglas RM, Kerby-Eaton E, eds. Acute respiratory infections: proceedings of an international workshop. Adelaide, South Australia: University of Adelaide, 3-16, 1985.

Ponikau JU, Sherris DA, Kephart GM, Adolphson C, Kita H. The role of ubiquitous airborne fungi in chronic rhinosinusitis. *Curr Allergy Asthma Rep* 5, 472-476, 2005.

Pope CA III. Respiratory disease associated with community air pollution and a steel mill, Utah valley. *Am J Public Health* 79, 623-628, 1989.

Pope CA III, Dockery DW, Spengler JD, Raizenne ME. Respiratory health and PM10 pollution. A daily time series analysis. *Am Rev Respir Dis* 144, 668-674, 1991.

Pope CA III, Verrier RL, Lovett EG, Larson AC, Raizenne ME, Kanner RE, Schwartz J, Villegas GM, Gold DR, Dockery DW. Heart rate variability associated with particulate air pollution. *Am Heart J* 138, 890-899, 1999.

Pope CA III, Hill RW, Villegas GM. Particulate air pollution and daily mortality on Utah's Wasatch Front. *Environ Health Perspect* 107, 567-573, 1999.

Pope CA III. Epidemiology of fine particulate air pollution and human health: biological mechanisms and who's at risk? *Environ Health Perspect* 108, 713-72, 2000.

Pope CA III. Air pollution and health- good news and bad news. (Editorial). *N Eng J Med* 351, 1132-1134, 2004 b.

Pope CA III, Burnett RT, Thurston GD. Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. *Circulation* 109, 71-77, 2004a.

Pope CA III, Burnett RT, Thurston GD, Thun MJ, Calle EE, Krewski D, Godleski JJ. Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease.

Circulation 109, 71-7, 2004.

Pope CA III, Hansen ML, Long RW, Nielson KR, Eatough NL, Wilson WE, Eatough DJ. Ambient particulate air pollution, heart rate variability, and blood markers of inflammation in a panel of elderly subjects. *Environ Health Perspect* 112, 339-345, 2004.

Pope CA III, Barnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD. Lung cancer, cardiopulmonary mortality, and long term exposure to fine particulate air pollution. *JAMA* 287, 1132-1141, 2002.

Porterfield SP. Vulnerability of the developing brain to thyroid abnormalities: environmental insults to the thyroid system. *Environ Health Perspect* 102, 125-130, 1994.

Prpic-Majic D, Bobicc J, Simic D, House DE, Otto DA, Jurasovicc J, Pizent A. lead absorption and psychological function in Zagreb (Croatia) school children. *Neurotoxicol Teratol* 22, 347-356, 2000.

Raaschou-Nielsen O. Air pollution and childhood cancer. In: *Effects of air pollution on children's health and development : A review of the evidence*. World Health Organization Special Programme on Health and Environment . European Centre for Environment and Health. Bonn, pp. 138-161, 2005.

Rahman A, Maqbool E, Zuberi HS. Lead-associated deficits in stature, mental ability and behaviour in Karachi. *Ann Tropical Med* 22, 301-311, 2002.

Raizenne M, Neas LM, Damokosh AI, Dockery DW, spengler JD, Koutrakis P, Ware JH, Speizer FE. Health effects of acid aerosols on North American children: pulmonary function. *Environ Health Perspect* 104, 506-514, 1996.

Rao NSN. Elements of health statistics. Tara Book Agency: Varanasi, p.87-101, 1989.

Ratageri VH, Kabra SK, Dwivedi SN, Seth V. Factors associated with severe asthma. *Indian Pediatr* 37, 1072-1082, 2000.

Redline S, Weiss ST. Genetic and perinatal risk factors for the development of chronic obstructive pulmonary disease. In: Hensley MJ, Saunders NA. Eds. *Clinical epidemiology of chronic obstructive pulmonary disease*. New York, NY, Marcel Dekker, pp. 139-168, 1989.

Rinne ST, Rodas EJ, Bender BS, Rinnie ML, Simpson JM, Galer-unti R, Glickman LT. Relationship of pulmonary function among women and children to indoor air pollution from biomass use in rural Ecuador. *Respir Med* Nov 27, 2005.

Ritz B, Yu F. The effect of ambient carbon monoxide on low birth weight among children born in southern California between 1989 and 1993. *Environ Health Perspect* 107, 17-25, 1999.

Roberts ES, Richards JH, Jaskot R, Dreher KL. Oxidative stress mediates air pollution particle-induced acute lung injury and molecular pathology. *Inhal Toxicol* 15, 1327-1346, 2003.

Rodriguez BL, Masaki K, Burchfiel C, Curb JD, Fong KO, Chyou PH, Marcus EB. Pulmonary function decline and 17-year total mortality: the Honolulu Heart Program. *Am J Epidemiol* 140, 398-408, 1994.

Romieu I. Nutrition and lung disease. *Int J Tuberc Lung Dis* 362, 362-374, 2005.

Romieu I, Sienra-Monge JJ, Ramirez-Aguilar M, Tellez-Rojo MM, Moreno-Macias H, Reyes-Ruiz NI, del Rio-Navarro BE, Ruiz-Navarro MX, Hatch G, Slade R, Hernandez-Avila M. Antioxidant supplementation and lung functions among children with asthma exposed to high levels of air pollutants. *Am. J. Respir Crit care Med* 155, 463-471, 2002.

Romieu I, Trenga C. Diet and obstructive lung diseases. *Epidemiologic Reviews* 23, 268-287, 2001.

Roy S, Ray MR, Basu C, Lahiri P, Lahiri T. Abundance of siderophages in sputum: indication of an adverse lung reaction to air pollution. *Acta Cytol* 45, 958-964, 2001.

Ryan G, Knuiman MW, Divitini ML, James A, Musk AW, Bartholomew HC. Decline in lung function and mortality: the Busselton Health Study. *J Epidemiol Com Health* 53, 230-234, 1999.

Saarinen UM. Prolonged breast-feeding as a prophylaxis for recurrent otitis media. *Acta Paediatr Scand* 71, 567-571, 1982.

Salam MT, Millstein J, Li YF, Lurmann FW, Margolis HG, Gilliland FD. Birth outcomes and prenatal exposure to ozone, carbon monoxide and particulate matter: results from the Children's Health Study. *Environ Health Perspect* 113, 1638-1644, 2005.

Salvi S, Blomberg A, Rudell B, Kelly F, Sandstrom, Holgate ST, Frew A. Acute inflammatory responses in the airways and peripheral blood after short-term exposure to diesel exhaust in healthy human volunteers. *Am J Respir Crit Care Med* 159, 702-709, 1999.

Samet JM, Marbury MC, Spangler JD. Health effects and sources of indoor air pollution. Part I. *Am Rev Respir Dis* 136, 1486-1508, 1987.

Sandstrom T, Andersson MC, Kolomodin-Hedman B, Stjernberg N, Angstorm T. Bronchoalveolar mastocytosis and lymphocytosis after nitrogen dioxide exposure in man: a time-kinetic study. *Eur Respir J* 3, 138-143, 1990.

Sandstrom T, Helleday R, Bjermer L, Stjernberg N. Effects of repeated exposure to 4 ppm nitrogen dioxide on bronchoalveolar lymphocyte subsets and macrophages in healthy men. *Eur Respir J* 5, 1092-1096, 1992.

Sanford AJ, Joos L, Pare PD. Genetic risk factors for chronic obstructive pulmonary disease. *Curr Opin Pulmon Med* 8, 87-94, 2002.

Schenker MB, Samet JM, Speizer FE. Risk factors for childhood respiratory disease. The effect of host factors and home environmental exposures. *Am Rev Respir Dis* 128, 1038-1043, 1983.

Schneider H, Eisenberg D. Who receives a diagnosis of attention-deficit/hyperactivity disorder in the United States elementary school population? *Pediatrics* 117, 601-609, 2006.

Schroeder EB, Welch VL, Couper D, Nieto FJ, Liao D, Rosamond WD, Heiss G. Lung function and incident coronary heart disease: the atherosclerosis risk in communities study. *Am J Epidemiol* 158, 1171-1181, 2003.

Schunemann HJ, Dorn J, Grant BJ, Winkelstein W Jr, Trevisan M. Pulmonary function is a long term predictor of mortality in the general population: a 29-year follow up of the Buffalo Health Study. *Chest* 118, 656-664, 2000.

Schunemann HJ, McCann S, Grant BJ, Trevisan M, Muti P, Freudenheim JL. Lung function in relation to intake of carotenoids and other antioxidant vitamins in a population-based study. *Am J Epidemiol* 155, 463-471, 2002.

Schwartz J, Katz S, Fegley R, Tockman M. Sex and race differences in the development of lung function. *Am Rev Respir Dis* 138, 1415-1421, 1988.

Schwartz J, Dockery DW, Wypii D. Acute effects of air pollution on respiratory symptom reporting in children. (Abstract). *Am Rev Respir Dis* 139, A27, 1989.

Schwartz J. Lung function and chronic exposure to air pollution: a cross-sectional analysis of NHANES II. *Environ Res* 50, 309-321, 1989.

Schwartz J, Spix C, Wichmann HE, Malin E. Air pollution and acute respiratory illness in five German communities. *Environ Res* 56, 1-14, 1991.

Schwartz J, Neas LM. Fine particles are more strongly associated than coarse particles with acute respiratory health effects in school children. *Epidemiol* 11, 6-10, 2000.

Schwartz J, Ballester F, Saez M, Perez-Hoyos S, Bellido J, Cambra K, Arribas F, Canada A, Perez-Boillos MJ, Sunyer J. The concentration-response relation between air pollution and daily deaths. *Environ Health Perspect* 109, 1001-1006, 2001.

Schwela D. Air pollution and health in urban areas. *Rev Environ Health* 15, 13-42, 2000.

Seaton A, MacNee W, Donaldson K, Godden D. Particulate air pollution and acute health effects. *Lancet* 354, 176-178, 1995.

Seaton A, Crompton G. Asthma: clinical features. In: Seaton D, Leitch AG, eds. *Crofton and Douglas's Respiratory Diseases*, 5th ed, Blackwell, Oxford, pp. 77-151, 2000.

Salameh PR, Baldi I, Brochard P, Raherison C, Abi Saleh B, Salamon R. Respiratory symptoms in children and exposure to pesticides. *Eur Respir J* 22, 507-512, 2003.

Samet J. Air pollution and epidemiology: "Déjà vu all over again?" *Epidemiol* 13, 118-119, 2002.

Samet JM, Graves LM, Quay J, Dailey LA, Devlin RB, Ghio AJ, Wu W, Bromberg PA, Reed W. Activation of MAPKs in human bronchial epithelial cells exposed to metals. *Am J Physiol* 275 (3 Pt 1), L551-L558, 1998.

Samet JM, Zeger SL, Dominici F, Curriero F, Coursac I, Dockery DW, Schwartz J, Zanobetti A. The National Morbidity, Mortality and Air Pollution study. Part II: Morbidity and mortality from air pollution in the United States. *Res Rep Health Eff Inst* 94, 5-70, 2000.

Savitz DA, Feingold L. Association of childhood cancer with residential traffic density. *Scand J Work Environ Health* 15, 360-363, 1989.

Schnass L, Rothenberg IJ, Flores MF, Martinez S, Hernandez C, Osorio E, Velaseo SR, Perroni E. Reduced intellectual development in children with prenatal lead exposure. *Environ Health Perspect* 114, 791-797, 2006.

Schunemann HJ, Mc Cann S, Grant BJ, Trevisan M, Muti P, Freudenheim JL. Lung function in relation to intake of carotenoids and other antioxidant vitamins in a population-based study. *Am J Epidemiol* 155, 463-471, 2002.

Schwartz J. Lung function and chronic exposure to air pollution: a cross sectional analysis of NHANES II. *Environ Res* 50, 309-321, 1989.

Schwartz J, Dockery DW, Wypii D. Acute effects of air pollution on respiratory symptom reporting in children. *Am Rev Respir Dis* 139, A27, 1989.

Schwartz JD Analysis of spirometric data from a national sample of healthy 6-to 24-year-olds (NHANES II). *Am Rev Respir Dis* 138, 1405-1414, 1988.

Schwartz J, Dockery DW. Increased mortality in Philadelphia associated with daily air pollution concentrations. *Am Rev Respir Dis* 145, 600-604, 1992.

Sidana A, Bhatia MS, Chaudhary S. Prevalence of and pattern of psychiatric morbidity in children. *Indian J Med Sci* 52, 556-558, 1998.

Sinaiko AR. Hypertension in children. (Review). *N Eng J Med* 335, 1968-1973, 1996.

Singh V, Khandelwal R, Gupta AB. Effect of air pollution on peak expiratory flow rate variability. *J Asthma* 40, 81-86, 2003.

Singh V. The burden of pneumonia in children: an Asian perspective. *Pediatr Respir Rev* 6, 88-93, 2005.

Srivastava GP. Socio-economic status scale (Urban) Agra: National Psychological Corporation, 1978.

Srivastava A, Joseph AE, More A, Patil S. Emissions of VOCs at urban petrol retail distribution centers in India (Delhi and Mumbai). *Environ Monit Assess* 109, 227-242, 2005.

Suk WA, Ruchirawat KM, Balakrishnan K, Berger M, Carpenter D, Damstra T, de Garbino JP, Koh D, Landrigan PJ, Makalinano I, Sly PD, Xu Y, Zheng BS. Environmental threats to children's health in Southeast Asia and the Western Pacific. *Environ Health Perspect* 111, 13401347,2003.

Sydbom A, Blomberg A, Parnia S, Stenfors N, Sandstorm T, Dahlen SE. Health effects of diesel exhaust emissions. *Eur Respir J* 17, 733-746, 2001.

Tager IB, Ngo L, Hanrahan JP. Maternal smoking during pregnancy: Effects on lung function during the first 18 months of life. *Am J Respir Crit Care Med* 152, 977-983, 1995.

Tager IB, Segal MR, Speizer FE, Weiss ST. The natural history of forced expiratory volumes. Effect of cigarette smoking and respiratory symptoms. *Am Rev Respir Dis* 138, 837-849, 1988.

Takano H, Yanagisawa R, Ichinose T, Sadakane K, Yoshino S, Yoshikawa T, Morita M. Diesel exhaust particles enhance lung injury related to bacterial endotoxin through expression of proinflammatory cytokines, chemokines, and intercellular adhesion molecule-1. *Am J Respir Crit Care Med* 165, 1329-1335, 2002.

Tee ES. Obesity in Asia: prevalence and issues in assessment methodologies. *Asia Pac J Clin Nutr* 11 (Suppl. 8), S694-S701, 2002.

Thurlbeck WM. Postnatal growth and development of the lung. *Am Rev Respir Dis* 111, 803-844, 1975.

Tiwari SC, Kumar A, Kumar A. Development and standardization of a scale to measure socio-economic status in urban and rural communities in India. *Ind J Med Res* 122, 309-314, 2005.

Toyama T. Air pollution and its effects in Japan. *Arch Environ Health* 8, 153-173, 1964.

Tracey VV, De NC, Harper JR. Obesity and respiratory infection in infants and young children. *BMJ* 1, 16-18, 1971.

Trakultivakorn M. Prevalence of asthma, rhinitis and eczema in Northern Thai children from Chiang Mai. *Asian Pac J Allergy Immunol* 17, 243 –248, 1999.

Tupasi TE, Velmonte MA, Sanvictores ME, Abraham L, De Leon LE, Tan SA, Miguel CA, Saniel MC. Determinants of morbidity and mortality due to acute respiratory infections: implications for intervention. *J Infect Dis* 157, 615-623, 1988.

Ulger Z, Demir E, Tanac R, Goksen D, Gulen F, Darcan S, Can D, Coker M. The effect of childhood obesity on respiratory function tests and airway hyperresponsiveness. *Turk J Pediatr* 48, 43-50, 2006.

Ulrich MM, Alink GM, Kumarathasan P, Vincent R, Boere AJ, Cassee FR. Health effects and time course of particulate matter on the cardiopulmonary system in rats with lung inflammation. *J Toxicol Environ Health A* 65, 1571-1595, 2002.

Update on the Task Force (1987) on High Blood Pressure in Children and Adolescents: a working group from the National High Blood Pressure Education Program. *Pediatrics* 98, 649-658, 1996.

van Eden SF, Hogg JC. Systemic inflammatory response induced by particulate matter air pollution: the importance of bone marrow stimulation. *J Toxicol Environ Health A* 65, 1597-1613, 2002.

van Roosbroeck S, Wichmann J, Janssen NA, Hoek G, van Wijnen JH, Lebret E, Brunekreef B. Long-term personal exposure to traffic-related air pollution among school children, a validation study. *Sci Total Environ* April 28, 2006 (Epub).

van Vliet P, Knape M, de Hartog J, Janssen N, Harssema H, Brunekreef B. Motor vehicles exhaust and chronic respiratory symptoms in children living near freeways. *Environ Res* 74, 122-132, 1997.

van Volkenburgh VA, Frost WH. Acute minor respiratory diseases prevailing in a group of families residing in Baltimore, Maryland, 1928-1930. Prevalence, distribution and clinical description of observed cases. *Am J Hyg* 17, 122-153, 1933.

Vassilev ZP, Robson MG, Klotz JB. Associations of polycyclic organic matter in outdoor air with decreased birth weight: a pilot cross-sectional analysis. *J Toxicol Environ Health A* 64, 595-605, 2001.

Vassilev ZP, Robson MG, Klotz JB. Outdoor exposure to airborne polycyclic organic matter and adverse reproductive outcome: a pilot study. *Am J Int Med* 40, 255-262, 2001.

Victora CG, Smith PG, Barros FC, Vaughan JP, Fuchs SC. Risk factors for deaths due to respiratory infections among Brazilian infants. *Int J Epidemiol* 18, 918-925, 1989.

Wang YC, Chong MY, Chou WJ, Yang JL. Prevalence of attention deficit hyperactivity disorder in primary school children in Taiwan. *J Formos Med Assoc* 92, 133-138, 1993.

Wang CL, Chang HY, Ho CK, Yang CY, Tsai JL, Wu TS, Wu TN. Relationship between blood lead concentrations and learning achievement among primary school children in Taiwan. *Environ Res* 89, 12-18, 2002.

Wang YC, Chong MY, Chou WJ, Yang JL. Prevalence of attention deficit hyperactivity disorder in primary school children in Taiwan. *J Formos Med Assoc*, 92, 133-138, 1993.

Ward DJ, Ayres JG. Particulate air pollution and panel studies in children: a systematic review. *Occu Environ Med* 61(4): e13, Review, 2004.

Ware JH, Dockery DW, Spiro A III, Speizer FE, Ferris BG Jr. Passive smoking, gas cooking and respiratory health of children living in six cities. *Am Rev Respir Dis* 129, 366-374, 1984.

Ware JH, Ferris BG Jr, Dockery DW, Spengler JD, Stram DO, Speizer FE. Effects of ambient sulfur oxides and suspended particles on respiratory health of pre adolescent children. *Am Rev Respir Dis* 133, 834-842, 1986.

Wasserman GA, Liu X, Popovac D, Factor-Litvak P, Kline J, Waternaux C, LoIacono N, Graziano JH. The Yugoslavia Prospective Lead Study: contributions of prenatal and postnatal lead exposure to early intelligence. *Neurotoxicol Teratol* 22, 811-818, 2000.

Weiland SK, Bjorksten B, Brunekreef B, Cookson WO, von Mutius E, Strachan DP. Phase II of the International Study of Asthma and Allergies in Childhood (ISAAC II): rationale and methods. *Eur Respir J* 24, 406-412, 2004.

Wheeler BW, Ben-Sholmo Y. Environmental equity, air quality, socioeconomic status, and respiratory health: a linkage analysis of routine data from Health Survey of England. *Epidemiol Comm Health* 59, 948-954, 2005.

Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. *N Eng J Med* 37, 869–873, 1997.

Winneke G, Walkowiak J, Lilienthal H. PCB-induced neuro developmental toxicity in human infants and its potential mediation by endocrine dysfunction. *Toxicol* Review, 181, 161-165, 2002.

Wjst M, Reitmer P, Dold S, Wulff A, Nicolai T, Loeffelholz-Colberg EF, Von Mutius E. Road traffic and adverse effects on respiratory health in children. *Br Med J* 307, 596-600, 1993.

Wolf C. Urban air pollution and health: an ecological study of chronic rhinosinusitis in Cologne, Germany. *Health Place* 8, 129-139, 2002.

Wong EY, Gohlke J, Griffith WC, Farrow S, Faustman EM. Assessing the health benefits of air pollution reduction for children. *Environ Health Perspect* 112, 226-232, 2004.

Wong TW, Yu TS, Liu HJ, Wong AH. Household gas cooking: a risk factor for respiratory illness in preschool children. *Arch Dis Chil* 89, 631-636, 2004.

Woods HM. The influence of external factors on the mortality from pneumonia in childhood and later adult life. *J Hyg (Camb)* 26, 36-43, 1927.

World Health Organisation. *Principles for evaluating health risks from chemicals during infancy and early childhood: the need for a special approach*, WHO Environmental Health Criteria 59. Geneva: World Health Organisation, 1986.

World Health Organization, Geneva. Integrated management of childhood illness, (documents WHO/CHD 97.3 A-G), 1997.

World Health Organization, The world health report 2000 -health systems: improving performance, 2000.

Young M. The influence of weather conditions on the mortality from bronchitis and pneumonia in children. *J Hyg* 23, 151-175, 1924.

Zeman KL, Bennett WD. Growth of the small airways and alveoli from childhood to the adult lung measured by aerosol-derived airway morphometry. *J Appl Physiol* 100, 965-971, 2006.

Zhang L, Yang W, Hubbard AE, Smith MT. Non random aneuploidy of chromosome 1,5,6,7,8,9,11,12 and 21 induced by the benzene metabolites hydroquinone and benzenetriol. *Environ Mol Mutagen* 45, 388-396, 2005.

Zhu J, Aikawa B, Pigeon R. Measurements of fine particles in diesel emissions using a real-time aerosol monitor. *J Air Waste Manag Assoc* 55, 978-983, 2005.

Zmirou D, Balducci F, Dihenaux J, Piras A, Filippi F, Benoit-Guyod JL. Meta-analysis and dose-response functions of air pollution respiratory effects. *Revue d'Epidemiologie et de Sante Publique* 45, 293-304, 1997

Zmirou D, Gauvin S, Pin I, Momas I, Sahraoui F, just j, Le Moullec Y, Bremont F, Cassadou S, Reungoat P, Albertini M, Lauvergne n, Chiron M, Labbe A, Vesta Investigators. Traffic related air pollution and incidence of childhood asthma: results of the Vesta case-control study. *J Epidemiol Comm Health* 58, 18-23, 2004.

CHAPTER-11.0

GLOSSARY

Abatement	A process to reduce, remove, or discontinue a nuisance disease vector, or pollutant
Acute	Short and severe
Acute illness	A brief but serious episode of illness
ADHD	It is a neurological disorder, usually diagnosed in childhood which manifests itself with symptoms such as hyperactivity, impulsivity or inattention
Adverse reaction	Undesirable or unwanted side effect as a consequence of a preventive, diagnostic, or therapeutic procedure
Aeroallergens	Airborne antigens like pollen, house dust, mite etc. that cause allergic response in sensitive individuals
Aerosol	Atomized particles suspended in the air that are small having diameters ranging from 1/100 μm to 1μm
Air	The gaseous mixture which makes up the atmosphere surrounding the earth. It consists of approximately 78% nitrogen, 20% oxygen, 0.04% carbon dioxide and traces of ozone, neon, helium etc., and a variable amount of water vapour
Air pollution	Detoriation of the quality of air for the presence of impurities
Air quality standards	Maximum allowable concentration of air pollutants. Concentrations that exceed these limits are considered harmful Primary standards are those needed to protect public health. Secondary standards protect against other effects such as damage to materials
Allergen	Substance or agent that causes allergy
Alveoius	The terminal sac like structures of the lung, which provide vast surface area for gaseous exchange. A large number of blood vessels surround them
Allergy	An altered or exaggerated susceptibility to various foreign substances which are harmless to great majority of individuals. Asthma is an allergic condition
Alveolar macrophages	A type of defense cell present inside the lungs. The cells readily engulf inhaled particles and microorganisms
Ambient	Surrounding conditions of the environment, usually referring to air quality and pollutant levels
Ambient level	The level of pollutant in the general ground level atmosphere, i.e. to which people may be exposed

Antibody	Specific proteins produced in the blood as a reaction to foreign substance. Antibodies bind to a specific antigen that elicits its production causing its destruction. Thus antibodies provide protection against infectious disease
Antigen	It is a substance that stimulates an immune response, especially the production of antibody.
Aplastic anemia	A type of anemia caused by the failure of the bone marrow to generate new blood cells of all types.
Asthma	Intermittent narrowing of the airways causing shortness of breath and wheezing. Usually the muscle of the bronchi walls contract and excess mucus collects restricting flow of air
Bacteria	A group of micro-organisms some of which are pathogenic to man
Benign	Innocent. A term used to denote opposite of malignant
B-lymphocyte	A subpopulation of lymphocytes, which produces antibodies
Bilirubin	A pigment derived from the breakdown of hemoglobin from red blood cells
Biomarker	Observable endpoints indicating the process leading to the genesis of a disease.
Biomarker of exposure	Measure of a parent compound or the unique response attributable to that compound in biological samples following exposure
Biomarker of effect	Quantifiable response of an organism that can directly be linked to the exposure
Biomarker of susceptibility of an environmental toxicant	The measure of the capability of an individual to respond to exposure
Bronchiotasis	Abnormal widening of the larger airways in the lung (bronchi) causing persistent cough with large amount of sputum
Bronchi	The two tubes into which trachea divides at its lower end
Bronchitis	Inflammation of the bronchus; may be acute or chronic
Cilia	Fine hair like strands. Cilia line the airways and trap foreign particles as the air pass through them
Carcinogen	A cancer causing substance
Carcinogenesis	The production of cancer or malignant cells

Cancer	Abnormal, unorganized and unregulated growth of cells in an organism
Cardiovascular disease	One or all of the diseases of the heart and blood vessels
Cardiac	Pertaining to the heart
Case-Control Study	A study of persons with a particular disease in comparison to a reference or control group, which is otherwise similar to the study group
Cell	The building blocks of all living organism.
Cellular immune response	Type of immune response characterized by generation of cytotoxic T- cells.
Cholesterol	A fatty material found in tissues and blood
Chromosome	Thread-like bodies found in cell nucleus which carry hereditary factors,the genes. The number being constant in each species e.g. in man 46 in each cell except in mature ovum and sperm where the number is 23
Chronic	Lingering, lasting, opposed to acute
Chronic Disease	An adverse health condition lasting for 3 months or more
Confidence Interval (CI)	The calculated range of numbers in a data set which, with a specified degree of probability (e.g., 95%), includes the true values of variables such as the mean, proportion, or rate of that set of numbers. The upper and the lower boundaries of the confidence interval are called confidence limits.
Confounding Variable (Confounder)	A factor or variable that can potentially cause or prevent the outcome or disease being studied (the dependent variable), and must be taken into account or it will prevent reaching a conclusion regarding the impact of *independent variable*, or hypothesized cause of the outcome disease being investigated
Chronic obstructive pulmonary disease	Progressive damage of the lungs usually leading to shortness of breath and wheezing. (often mean chronic bronchitis and emphysema)
Cost-Benefit Analysis	A system of analysis that attempts to weigh the cost of some policy such as pollution control, directly against the economic gain.
Cost-Effectiveness Analysis	A form of economic evaluation where all costs are expressed in monetary terms, but the consequences are expressed in non-monetary terms, such as life years gained, cases detected, or cases prevented.
Cough	A reflex in response to irritation or infection in the respiratory tract, which helps to clear irritant or blockages from the airways.

Cross-sectional study	A study of the relationship between diseases with other variables of interest in a defined population at a particular point of time. This establishes the disease prevalence and the presence of the characteristic being studied (e.g., smoking or toxic exposure) in the diseased compared to the non diseased person in the group.
Cytology	Microscopic study of the cells
Dependent Variable	An outcome or manifestation we seek to account for by the influence of an independent variable(s) or intermediate factor(s) in a hypothesized relationship being studied
Developed and Developing Countries	Countries are defined as to their level of development by per capita GNP by the World Bank. Those with per capita GNP of $765 or less in the year 1998 are defined as *low income economies* or least developed countries. Those between $765 – 3,035 and $ 3,035 – 9,386 are considered lower and upper income developing countries respectively. Countries with per capita GNP of $ 9,386 or more are defined as developed (*high income economies*)
Disease	Disease is physiological and psychological dysfunction
DNA	Deoxyribonucleic acid. It is present in cell nucleus. Mitochondria also contains some DNA. It carries genetic information from parents to their progeny and therefore accounts for the continuity of species
Dyspnea	Difficult or labored breathing
Elastase	A connective tissue degrading enzyme
Emission standards	Legal limits of the quantities of air pollutants permitted to be emitted from the exhaust of a source
Emphysema	Alveolar distension often accompanying chronic bronchitis. A common problem encountered in smokers
Environment	All factors external to the individual that may affect its health behavior or well-being. Includes physical, biological, social economic and other factors
Enzyme	Protein molecule which acts as a catalyst in the chemical reactions within the body
Eosinophil	A type of white blood cell containing granules staining in acid dyes such as eosin. They form 2-5% of white blood cells in man, but increased manifold in allergy and parasitic infections
Epidemiology	The study of the distribution and determination of health and its disorders
Epithelium	The surface layer of cells below which lies the basement membrane

Erythrocyte	Hemoglobin-containing red cells present in blood
Etiology	The origin or causes of a disease, health condition or risk factors
Exfoliation	The scaling off of tissues in layers
Expiration	The act of breathing out air from the lungs
Exposure	Quantity and duration of contact between a person and a harmful agent. Exposure may be continuous, periodic or episodic
Extrapolation	The prediction of points on a graph outside the range of observation
FEV_1	Forced expiratory volume in one second. The volume of air that can be expired in one second
Fibrosis	The formation of excessive fibrous tissue in a structure
Fungus	Plants including microscopic organisms capable of producing disease in man.
FVC	Forced vital capacity. The volume of air expired forcefully expiration following maximum inspiration
Gene	The basic unit of heredity composed of molecules of deoxyribonucleic acid (DNA) and located on the chromosomes of each cell. Genes govern every single structural and functional characteristic of an individual
Giand	An organ or structure capable of making internal or external secretions
Goblet cells	Secretory cells, shaped like a goblet, found in the mucous membrane
Granulocyte	Any cell containing granules, e.g. neutrophils, eosinophils and basophils
Green house effect	The effect produced by certain gases such as carbon dioxide or water vapor that causes warming of the earth's atmosphere by absorption of infrared radiation.
Health	WHO states "Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity"
Hemoglobin	The respiratory pigment in red blood cells. It is composed of iron containing substance "heme" combined with protein globin.
Hemorrhage	Escape of blood from a vessel.
Hemosiderin	A form of storage iron in tissue.
Hygiene	The science dealing with the maintenance of health.
Hyperactivity	Excessive activity

Hyperplasia	Excessive formation of cells
Hypersensitivity	Type of immune response, which has an adverse impact. Also known as allergy
Immune response	Reaction of the body's defense system to insults by foreign substances Lymphocytes, macrophages, neutrophils, eosinophils and basophils play important role in mediating immune response
Immune system	A network of organs and cells responsible for generating immune response
Immunity	Individual's resistance to infection. Resistance to a disease by the host may be natural, passive or acquired
Infection	Invasion of the body by disease-causing organism, with or without manifestation of the disease.
Inhalation	The breathing in of air or other vapor
Inhalable particles	Particles which may be breathe in. "Inhalability" is defined technically as the orientation – averaged aspiration efficiency for the human head (also termed inspirable)
Inspiration	Drawing of air into the lungs
Inflammation	The reaction of living tissue to injury, infection or irritation, characterized by pain, swelling, redness and heat
Intracellular	Within the cell, opposite to extracellular
Leucocytes	White blood cells engaged in body's defense against infection
Leukemia	Cancer in bone marrow cells
Lipid	Fat
Lungs	The two main organs of respiration which occupy the greater portion of the thoracic cavity
Lung function test	A test used to detect airflow problems in the lungs
Lymphocytes	A kind of white blood cells that protect the body against diseases by direct killing or by producing antibodies
Macrophages	Phagocytic cells widely distributed in the body. Plays important role in body's defense, disposal of debris, and repair of the injured tissues
Metaplasia	A reversible cellular change in which one cell type is replaced by another adult cell type

Mucus	A clear, sticky lubricant secreted by glands in the mucus membranes that line the body cavity
Mutagen	An agent that produces mutation, ie. genetic change
Mutation	A change or alteration in the genes of a living cell. As a result the characters of the cell change
Mortality	The death rate; the ratio of the total number of deaths to the total population
Mucosa	A mucous membrane
Natural killer cells (NK)	Type of leukocytes that preferentially inactivate virus-infected or cancerous cells
Neutrophils	Granular white blood cells with the properties of chemotaxis, adherence to immune complexes and phagocytosis: also produce antimicrobial substances including oxidants and proteases
Particulate matter	Particles of solid or liquid matter in the air, including nontoxic materials (soot, dust, dirt), heavy metals (e.g., lead), and toxic materials (as asbestos, suspended sulfates, nitrates)
Pathogen	An organism, toxin, or other agent capable of causing human, animal, or plant disease
Photochemical smog	Smog caused by the formation of particles due to a chemical reaction driven by sunlight
PM_{10}	Particulate matter less than 10 μ m aerodynamic diameter
Pollutant	Any substance that renders the atmosphere or water foul or noxious or a health hazard.
Pollution	The impairment of the quality of some portion of the environment by the addition of harmful impurities
ppb	Parts per billion, 1 part by volume in 109
ppm	Parts per million, 1 part by volume in 106
Prevalence (Rate)	The total number of all individuals who have a disease including new and previous cases in a given population at a designated time (point prevalence) or time period (period prevalence), expressed usually as a rate per 1000 persons during a year
Relative Risk	The relation of risk of disease or death among the exposed, as compared to that of an otherwise comparable nonexposed population group
Respirable particles	Particles which can penetrate to the unciliated regions of the lung

Risk factors	Those characteristics or behavioral pattern known to increase the risk of disease.
Sampling	A sample is a subset of population selected to be as representative as possible of the total population. A sample may be random or non-random, representative or non-representative. The major categories include: *cluster sample*, a group of persons not individually selected, i.e., all person in city block; *grab sample*, A simple survey among people who happen by or show up at a service offered, such as a street fair, from which no general conclusions can be drawn; *probability (random) sample*, where all individuals have an equal or known chance of being selected, or if stratified, subgroups may be assigned greater weight in the design; *simple random sample*, All person in the group are assigned a number, and the selection of the sample is according to a random numbers table, until the needed sample size is achieved; *stratified random sample*, where the population is divide into subgroups, and each of these is sampled randomly; and *systematic sample*, where the sample is selected on the basis of a predetermined method, such as alphabetic order or birth dates
Secondary particle or aerosol	Particles may be formed when two volatile and non-condensable vapor species react to give rise to a product with a very low vapor pressure. Such a product is described as a secondary particle to distinguish it from those arising from the reaction of liquids or solids
Sinusitis	Inflammation of any of the sinuses leading to headache and tenderness in the face
Spirometer	An instrument used to measure the volume (in litres) that one can inhale or exhale over a period of time .The results can indicate whether the airways are narrowed due to lung disease
Smog	A term often used to describe a mixture of smoke and fog. Also used to describe photochemical air pollution,or smoky fog the word is used loosely to describe visible air pollution
Smog precursor	An air pollutant that can undergo chemical reaction in the presence of sunlight
Smoke	Particulate matter, <15μm diameter, derived from the incomplete combustion fuels. Or an aerosol that is usually produced by combustion or decomposition process
Stress	Response to challenge (“fright- fight- flight”) of changes in the status quo of a individual causing overt or hidden psychological pressure that may manifest themselves in overt psychological symptomatology or in physical illness.
Suppressor T-cells	A subpopulation of T- lymphocytes that suppresses immune response

Susceptible	A person with insufficient resistance or with associated risk factors to a particular pathogenic agent or process, so that there is real danger to this person contracting the specific disease if or when exposed to the agent
Symptom	An organic or physiologic manifestation of a disease of which the patient is usually aware and complains of it
Synergism	A condition in which a whole effect is greater than the sum of its parts
Target cells	Abnormally flat red cells with a central mass of hemoglobin surrounded by a ring of pallor and an outer ring of hemoglobin. They are commonly associated with liver disease, impaired or absent splenic function (hyposplenism) and hemoglobinopathies
T-Lymphocytes	A subpopulation of lymphocytes that matures under the influence of thymus.
Threshold level	The minimal dose of a toxic substance that causes harmful effects.
Total suspended particulate matter volume	A term describing the gravimetrically determined mass loading of air borne particles, most commonly associated with use of the US high air sampler in which particles are collected to filter for weighing.
Toxic substance	Any substance whose physiological action is harmful to health.
Tuberculosis	A bacterial infection *(Mycobacterium tuberculosis*) that most often affects the lungs. Usually transmitted by airborne droplets
Urbanization	A demographic process characterized by movement of people from rural to urban settlements
Ultra fine particles	Particles of less than 100nm diameter
Virus	Essentially a capsule of protein that contains either DNA or RNA as genetic material. There is debate as to if it is a living organism or a chemical entity. However, it has powers of reproducing when it invades a living cell.

ABBREVIATIONS USED

ADHD	Attention-Deficit Hyperactivity Disorder
ALL	Acute lymphoblastic leukemia
AM	Alveolar macrophage
AML	Acute myeloblastic leukemia
ARIs	Acute respiratory infections
ATS	American Thoracic Society
BAL	Broncho-alveolar lavage
B(a)P	Benzo(a)pyrene
BMI	Body mass index
BMRC	British Medical Research Council
CNG	Compressed Natural Gas
COPD	Chronic obstructive pulmonary disease
CO	Carbon monoxide
COx	Oxides of carbon
COPD	Chronic obstructive pulmonary disease
CDC	Centre for Disease Control
CD	Cluster determinant
CPCB	Central Pollution Control Board
95%CI	95% confidence interval
CNCI	Chittaranjan National Cancer Institute
CNS	Central nervous system
CRP	C-reactive protein
CSE	Centre for Science and Environment
CVD	Cardiovascular disease
DSM	Diagnostic and Statistical Manual of conduct disorders
DBP	Diastolic blood pressure
DEP	Diesel Exhaust Particles
ESMAP	Energy Sector Management Assistance Program
et al.	And others
ETS	Environmental tobacco smoke
FEV_1	Forced expiratory volume in one second
$FEF_{25-75\%}$	Forced expiratory flow between 25 and 75 percent of the vital capacity
Fig.	Figure
FVC	Forced vital capacity
GEEs	Generalized estimating equations
GIS	Geographical Information System
IARC	International Agency for Research on Cancer
IAQ	Indoor air quality
IL-8	Interleukin-8
IL-6	Interleukin-6
IUATLD	International Union against Tuberculosis and Lung Disease
LPG	Liquid petroleum gas

LRS	Lower respiratory symptoms
LRTIs	Lower respiratory tract infections
MMEF	Mid maximal expiratory flow
NAAQS	National ambient air quality standards
NEERI	National Environmental Engineering Institute
NHANES	National Health and Nutrition Examination Survey
NHLI	National Heart and Lung institute
NK	Natural killer cells
NOx	Oxides of nitrogen
NSE	Non - specific esterase
O_3	Ozone
OR	Odds ratio
PAH	Polycyclic aromatic hydrocarbon
PCBs	Polychlorinated biphenyls
PEFR	Peak expiratory flow rate
PFT	Pulmonary function test
PM	Particulate matter
PM_{10}	Particulate matter with less than 10 µm diameter
$PM_{2.5}$	Particulate matter with less than 2.5 µm diameter
PM_1	Particulate matter with less than 1 µm diameter
Pap	Papanicolaou
RBC	Red blood corpuscles
ROS	Reactive oxygen species
RSC	Respiratory symptom complex
RSPM	Respiratory suspended particulate matter
RSV	Respiratory syncitial virus
SBP	Systolic blood pressure
SD	Standard deviation of mean
SES	Socio-economic status
SM	Squamous metaplasia
SOx	Oxides of sulfur
SPM	Suspended particulate matter
SPSS	Statistical Package for Social Sciences
TERI	Tata Energy Research Institute
TNF- á	Tumor necrosis factor- alpha
UFP	Ultra fine particle with diameter of less than 0.1µm
UNEP	United Nations Environment Program
URS	Upper respiratory symptom
URTIs	Upper respiratory tract infections
US EPA	United States environment protection agency
VOC	Volatile organic compound
WBC	White blood corpuscles
WBPCB	West Bengal Pollution Control Board
WHO	World health organization

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