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Effect of Air Pollution on Incidence of Asthma: A Case Study in Children

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Abstract

Urban air pollutants cause a wide range of acute and chronic effects in children's respiratory system. Children are more susceptible to environmental pollutants than the adults. Indian capital has the dubious distinction of one of the most polluted cities in the world. Although asthma is genetically controlled, exposure to air pollution exacerbates asthma attacks. The prevalence of asthma in school-going children with respect to ambient air pollution level has been investigated in this study. The prevalence of asthma symptoms was determined through structured questionnaire. Air quality data was obtained from Central Pollution Control Board and was also measured by real-time portable, battery-operated laser photometer. The collected data were processed and analyzed in EPI info 6.0 and SPSS (Statistical Package for Social Sciences) software. Altogether 11,628 children from Delhi and 4536 children from rural areas of Uttaranchal and West Bengal participated in this study. The prevalence of current asthma 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 the control group- 2.5% and 1.1% respectively ($p<0.001$). Asthma was found more prevalent in children from large-sized families. No positive association was found between PM_{10} level and prevalence of physician diagnosed asthma, but the air pollution contributes to asthma aggravation, leading to an increase in symptoms (current asthma).

Keywords: Air pollution, Children, Asthma.

Introduction

Urban air pollutants cause a wide range of acute and chronic effects in children's respiratory system. Children are more susceptible to environmental pollutants than the adults because their immune systems and developing organs are still immature, the special vulnerability of children to air pollution exposure is related to major differences in the physiology of a child and an adult (Gilliland *et al.*, 1999). Children represent the largest subgroup of the population susceptible to the adverse health effects of urban air pollution (Dockery *et al.*, 2005). Irritation or inflammation caused by air pollution is more likely to obstruct their narrower airways. It may also take less exposure to a pollutant to trigger an asthma attack due to the sensitivity of a child's developing respiratory system

Delhi is one of the fastest growing cities in Asia. Air pollution is viewed as a serious problem in Delhi. Indian capital has the dubious distinction of one of the most polluted cities in the world. In 2005, Delhi had an annual average concentration of respirable suspended particulate matter i.e. PM_{10} of 163.4 $\mu g/m^3$, which was 2.7-times more than the Indian standards for residential areas (60$\mu g/m^3$). The major sources of city's air pollution are road traffic (72%), industrial emissions (20%) and emissions from household activities (8%) [MoEF,1997].

Bronchial asthma results from intermittent narrowing of the airways and consequent shortness of breath. Its early symptoms are wheezing, tightening of chest, shortness of breath, and difficulty in exhaling and dry persistent cough. Although asthma is genetically controlled, exposure to air pollution exacerbates asthma attacks (Cakmak *et al.*, 2004).

Excessive airway narrowing is a cardinal feature of bronchial asthma, and is considered to be the most important pathophysiologic determinant in fatal asthma. It is supposed to be associated with mucosal infiltration of inflammatory cells, airway-wall submucosal thickening, and smooth-muscle hypertrophy in both large and small airways, as observed in patients dying from asthma (in't Veen *et al.,* 2000).

A strong association between severe asthma symptoms and breath concentration of benzene has been demonstrated in children (Delfino *et al.*, 2003). High vehicle traffic resulted in asthma, cough and wheeze in children who were additionally exposed to environmental tobacco smoke in Germany (Nicolai *et al.*, 2003). A recently published 10-year study of Southern California communities showed that children living in smoggy areas were three to four times more likely to develop asthma than those living in cleaner areas.

In view of the above, the prevalence of physician diagnosed asthma and current asthma in school-going children with respect to ambient air pollution level has been investigated in this study.

Materials and Methods

Subjects

The study was conducted following stratified random sampling procedure under the general plan of Simple Random Sampling Without Replacement method (Rao, 1989). A total number of 16,164 children aged between 6-17 years participated in this study.

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.

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. 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.

Inclusion and exclusion criteria

Apparently healthy school-age children were included in this study. Children with history of neurological problems like epilepsy, autism and mental retardation were excluded from our study.

Questionnaire survey for asthma symptoms

The prevalence of asthma symptoms was determined through structured 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 Against Tuberculosis and Lung Disease (IUATLD) bronchial symptoms questionnaire (1984) [Burney et al., 1989]. 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 was established from parents' answer to the questions "Has a doctor ever told you that your child had asthma?" (Eisner, 2002).

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).

Air quality data

Data on the concentration of ambient air pollution 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) and sulfur (SOx) and ozone in study areas during and preceding months of this study were obtained from air quality monitoring stations of Central Pollution Control Board (CPCB). Additionally, we have measured real-time particulate pollutant concentration in air by portable, battery-operated laser photometer (DustTrak™ Aerosol monitor, model 8520, TSI Inc., MN, USA).

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.

Results

Demographic characteristics

The characteristics of both groups of children are presented in Table 1. 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 ($p<0.05$).

Air pollution level

The annual average concentrations of PM_{10} was 161.3 ± 4.9 µg/ m^3. In contrast, the concentrations of these pollutants were significantly lower in control areas- 74.6 ± 3.3 µg/m^3. Mean concentrations of sulfur dioxide (SO_2) and nitrogen dioxide (NO_2) in the urban air during this period were 9.6 ± 1.0 and 50.1 ± 7.1 µg/ m^3 respectively, and it was within the permissible limit. In the control areas the concentrations were 5.6± 2.2 and 30.3± 5.2µg/ m^3 respectively (Table 2).

Prevalence of asthma

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 the control group- 2.5% and 1.1% respectively ($p<0.001$ in Chi-square test, Table 3, Figure 1).

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 ($p<0.001$). Similarly, Delhi's boys had greater prevalence of physician-diagnosed asthma than the girls (1.7 vs. 1.5%). Like the urban children, boys in the control group also 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 (Table 3). When compared with the control group, boys from Delhi had higher prevalence of both current asthma (5.5% vs. 2.7%; $p<0.001$) and physician-diagnosed asthma (1.7% vs. 1.1%; $p<0.05$) and the difference was highly significant. Similarly girls from the urban area had higher prevalence of current asthma (2.7% vs.

2.1%; $p<0.05$) and physician diagnosed asthma (1.5% vs. 0.5%; $p<0.01$) when compared with the rural counterpart (Table 3).

Table 1 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
Average family income/month (Rs.)	4700	9800	<0.05

NS, statistically not significant

Table 2 Comparison of air quality of the residential areas of Delhi and the control area

Pollutant (μg/m^3)	Standard for residential areas	Control areas in West Bengal	Delhi
SPM	140	167.6 ± 28.3	341.8 ± 38.3*
RSPM	60	74.6 ± 3.3	161.3±4.9*
NO_2	60	30.3±5.2	50.1± 7.1*
SO_2	60	5.6± 2.2	9.6± 1.0*

*The data (mean ±SD) are annual average values; *, $p<0.05$ compared with control*

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 (Figure 2).

Table 3 Prevalence of bronchial asthma in rural and urban children

Type of asthma	Control (n=4536)	Delhi (n=11628)	p- value
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 percentage

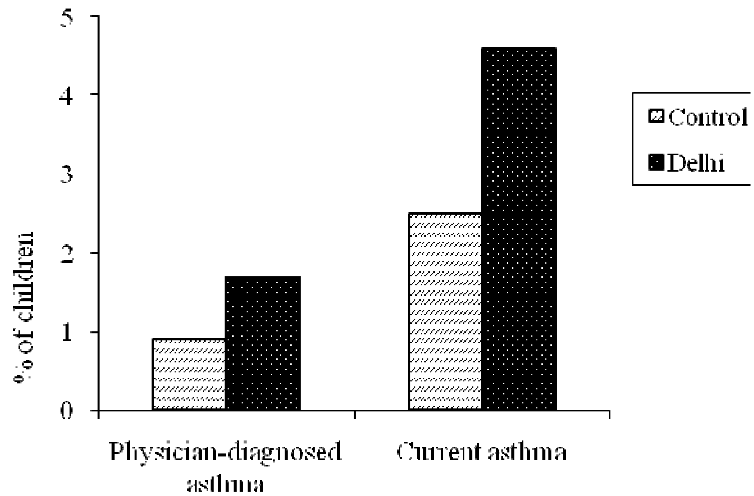

Figure 1 Comparison of physician-diagnosed asthma and current asthma in the two study groups

Figure 2 Prevalence of physician diagnosed asthma in different age group of children

Prevalence of asthma in children from different socio-economic background

Prevalence of asthma in different SES is presented in Table 4. In control group, asthma was slightly more prevalent in children from medium and high SES, especially in case of physician-diagnosed asthma. 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.

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 5). 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 4 Prevalence (%) of current asthma in different socio-economic status

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*

Table 5 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*

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 with the prevalence of asthma par *se* (OR = 1.05, 95% CI 0.87-1.15). 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 in city's children (Table 6).

Table 6 Conditional logistic regression analysis for medically diagnosed asthma

PM_{10} level ($\mu g/m^3$)	Medically- diagnosed asthma
50-75	1
76-100	0.31 (0.09-1.04)
101-125	1.41 (0.88-2.27)
126-150	1.22* (1.02-2.07)
>150	1.55* (1.15-2.56)

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

Discussion

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 lung function parameters like 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, we have recorded current asthma and medically diagnosed asthma 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 reported from some Asian countries including Iran (Golshan *et al.*, 2002) and Thailand (Trakultivakorn, 1999).

A strong association between current asthma in children and parental smoking, and similar illness in father and / or other members of the family was found. It was observed that asthma attacks were most prevalent in children during winter (2.0%)

when the air pollution level was highest. When potential confounders were controlled, logistic regression analysis suggests positive association between particulate pollution and asthma attacks (OR=1.28; 95% CI 1.07-1.42), but not with the prevalence of asthma *par se* (OR = 1.05, 95% CI 0.87-1.15).

Information on the prevalence, magnitude of the problem and risk factors of childhood asthma 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, we did not find any causative role of particulate air pollution with asthma. However, we have recorded a lower prevalence of asthma among the schoolchildren of Delhi compared with earlier report. 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).

Weiland and Forastiere (2005) have 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.

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). 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 *et al.*, 2000).

Besides PM_{10}, a positive and consistent association between asthma outcome and ozone has been reported (Delfino *et al.*, 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. Thus, ozone appears to have some influence on the prevalence of asthma symptom. Delfino *et al.*, (1998) found that asthma symptoms were associated with both PM_{10} and ozone with a greater relative effect from PM_{10}.

Although it has been suggested that IL-10 might be an antiasthmatic cytokine, IL-10 also has some properties that could promote allergic airways inflammation. For example, IL-10 potentiates IgE production by B-cells and is a growth factor for mast cells. Induction of IL-10 by DE could thus contribute to skewing the immune system in the airways mucosa towards enhancement of asthmatic airways inflammation (Stenfors *et al.*, 2004). Thus, exposure to air pollutants exacerbates asthmatic symptoms by recruitment of inflammatory cells and mediators.

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 ≥28 kg/m2) persons (Guerra *et al.,* 2002).

A recent study conducted by Clark et al., (2010) stated that early life exposures to CO, NO, NO_2 PM_{10}, SO_2, black carbon, and industrial point sources were positively associated with asthma, with the strongest associations noted for traffic-related pollutants. Among children with asthma, air pollutants cause increase in the frequency of respiratory symptoms, increase in medication use and transient deficit in lung function. Overall it can be 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. Keeping a check on the pollution load can decrease the asthma like symptoms and give the children a better environment to breathe, something to which they are entitled.

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References

Burney, P.G., Laities, L.A., Perdrizet, S., Huckauf, H., Tattersfield, A.E., Chinn, S., Poison, N., Heeren, A., Britton, J.R., Jones, T. 1989. Validity and repeatability of the IUATLD (1984) Bronchial Symptom Questionnaire: an international comparison. *European Respiratory Journal* 2, 940-945.

Cakmak, A., Ekici, A., Ekici, M., Arslan, M., Iteginli, A., Kurtipek, E., Kara, T. 2004. Respiratory findings in gun factory workers exposed to solvents. *Respiratory Medicine* 98, 52-56.

Chhabra, S.K., Gupta, C.K., Chhabra, P., Rajpal, S. 1999. Risk factors for development of bronchial asthma in children in Delhi. *Annals of Allergy Asthma & Immunology* 83, 385-390.

Clark, N.A., Demers, P.A., Karr, C.J., Koehoorn, M., Lencar, C., Tamburic, L., Brauer, M. 2010. Effect of early life exposure to air pollution on development of childhood asthma. *Environ Health Perspect* 118, 284-90.

Cotes, J.E. 1987. Medical Research Council Questionnaire on Respiratory Symptoms. *Lancet* 31, 1028.

Dockery, D.W., Skerrett, P.J., Walters, D., Gilliland, F. 2005. 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.

Delfino, R.J., Zeiger, R.S., Seltzer, J.M., Street, D.H. 1998. Symptoms in pediatric asthmatics and air pollution: differences in effects by symptom severity, anti-inflammatory medication use and particulate averaging time. *Environmental Health Perspective* 106, 751-761.

Delfino, R.J., Zeiger, R.S., Seltzer, J.M., Street, D.H., McLaren, C.E. 2002. Association of asthma symptoms with peak particulate air pollution and effect modification by anti inflammatory medication use. *Environmental Health Perspective* 110A, 607-617.

Delfino, R.J., Gong, H. Jr., Linn, W.S., Pellizzari, E.D., Hu, Y. 2003. Asthma symptoms in Hispanic children and daily ambient exposures to toxic and criteria air pollutants. *Environmental Health Perspective* 111, 647-656.

Donaldson, K., Gilmour, M.I., MacNee, W. 2000. Asthma and PM10. *Respiratory Research* 1, 12-15.

Eisner, M.D. 2002. Environmental tobacco smoke exposure and pulmonary function among adults in NHANES III: Impact of the general population and adults with current asthma. *Environmental Health Perspective* 110, 765-770.

Ferris, B.G. Epidemiology standardization project. 1978. *American Review of Respiratory Disease* 118, 1-88.

Gehring, U., Pattenden, S., Slachtova, H., Antova, T., Braun-Fahrlander, C., Fabianova, E., et al. 2006. Parental education and children's respiratory and allergic symptoms in the Pollution and the Young (PATY) study. *European Respiratory Journal* 27, 95-107.

Gilliland, F.D., McConnell, R., Peters, J., Gong, H. Jr. 1999. A theoretical basis for investigating ambient air pollution and children's respiratory health. *Environmental Health Perspective* 107, 403-407.

Golshan, M., Mohammad –Zadeh, Z., Khanlar- Pour, A., Iran- Pour, R. 2002. Prevalence of asthma and related symptoms in junior school children in Isfahan, Iran. *Monaldi Archive for Chest Disease* 57, 17 – 22.

Guerra, S., Sherrill, D.L., Bobadilla, A., Martinez, F.D., Barbee, R.A. 2002. The relation of body mass index to asthma, chronic bronchitis and emphysema. *Chest* 122, 1256-1263.

Harris, J.R., Magnus, P., Samuelsen, S.O., Tamdes, K. 1997. No evidence for effects of family environment on asthma; a retrospective study of Norwegian twins. *American Journal of Respiratory and Critical Care Medicine* 156, 43-49.

Holgate, S.T. 1999. Genetic and environmental interaction in allergy and asthma. *Journal of Allergy and Clinical Immunology* 104, 1139-1146.

Hwang, B.F., Lee, Y.L., Lin, Y.C., Jaakkola, J.J., Guo, Y.L. 2005. Traffic related air pollution as a determinant of asthma among Taiwanese school children. *Thorax* 60, 467-473.

Kjellman, B., Gustafson, P.M. 2000. Asthma from childhood to adulthood. *Respiratory Medicine* 94, 454 – 465.

Kostamo, K., Richardson, M., Malmberg, H., Ylikoski, J., Ranta, H., Toskala, E. 2005. Does the triad of fungi, bacteria and exposure to moisture have an impact on chronic hyperplastic sinusitis? *Indoor Air* 15, 112-119.

Mistry, R., Wickramaingha, N., Ogston, S., Singh, M., Devasiri, V., Mukhopadhyay, S. 2004. Wheeze and urban variation in South Asia. *European Journal of Pediatrics* 163,145-147.

MoEF. 1997. Ministry of Environment and Forest, Government of India. White Paper On Pollution In Delhi With An Action Plan. New Delhi.

Nicolai, T., Carr, D., Weiland, S.K., Duhma, H., Ehrestein, O.V., Wagner, C., Mutlus, E.V. 2003. Urban traffic and pollutant exposure related to respiratory outcomes and atopy in a large sample of children. *European Respiratory Journal* 21, 956-963.

Pearce, N., Pekkanen, J., Beasley, R. 1999. How much asthma is really attributable to atopy? *Thorax* 54, 268-272.

Rao, N.S.N. 1989. Elements of health statistics. Tara Book Agency: Varanasi, p.87-101.

Ratageri, V.H., Kabra, S.K., Dwivedi, S.N., Seth, V. 2000. Factors associated with severe asthma. *Indian Pediatrics* 37, 1072-1082.

Salameh, P.R., Baldi, I., Brochard, P., Raherison, C., Abi Saleh, B., Salamon, R. 2003. Respiratory symptoms in children and exposure to pesticides. *European Respiratory Journal* 22, 507-512.

Srivastava, G.P. 1978. Socio-economic status scale (Urban) Agra: National Psychological Corporation.

Stenfors, N., Nordenhall, C., Salvi, S.S., Mudway, I., Soderberg, M., Blomberg, A., Helleday, R., Levin, J.O., Holgate, S.T., Kelly, F.J., Frew, A.J., Sandstrom, T. 2004. Different airway inflammatory responses in asthmatic and healthy humans exposed to diesel. *European Respiratory Journal* 23, 82-86.

Tiwari, S.C., Kumar, A., Kumar, A. 2005. Development and standardization of a scale to measure socio-economic status in urban and rural communities in India. *Indian Journalo of Medical Research* 122, 309-314.

Trakultivakorn, M. 1999. Prevalence of asthma, rhinitis and eczema in Northern Thai children from Chiang Mai. *Asian Pacific Journal of Allergy & Immunology* 17, 243 –248.

in't Veen, J.C., Beekman, A.J., Bel, E.H., Sterk, P.J. 2000. Recurrent exacerbations in severe asthma are associated with enhanced airway closure during stable episodes. *American Journal of Respiratory and Criicalt Care Medicine* 161, 1902-1906.

Weiland, S., Forastiere, F. 2005. Effects of air pollution on children's health and development: A review of evidence. World Health Organization.