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## Indoor air pollution and asthma in children at Delhi, India

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### Abstract

**Introduction:** Several studies in developed countries have shown association between indoor air pollution and asthma in children. The present research was undertaken to study this association at Delhi, India.

**Material and methods:** This study took place at Delhi, capital of India. Eight locations based on the source of pollution such as industrial, residential and villages were included. Recording of the demographic profile and clinical examination of each child was conducted at their residence. Indoor SO<sub>2</sub>, NO<sub>2</sub> and SPM (suspended particulate matter) levels were measured by using Handy Air Sampler (Low Volume Sampler).

**Results:** A total of 3104 children were examined of which 60.3% were male and 39.7% were female. 32.4% children were exposed to environmental tobacco smoke. 31.5 % children's families were using biomass fuels for cooking. History of respiratory symptoms included cough (43.9%), phlegm production (21.9%), shortness of breath (19.3%) and wheezing (14.0%). 7.9% children were diagnosed as having asthma, which was highest in industrial areas (11.8%), followed by residential (7.5%) and village areas (3.9%). The mean indoor SO<sub>2</sub>, NO<sub>2</sub> and SPM levels were 4.28 ± 4.61 mg/m<sup>3</sup>, 26.70 ± 17.72 mg/m<sup>3</sup> and 722.0 ± 457.6 mg/m<sup>3</sup> respectively. Indoor SPM was the highest in industrial area followed by residential area and urban village area. Indoor SPM level was significantly ( $p < 0.001$ ) higher in the asthmatic children's houses.

**Conclusion:** This study suggests that industry plays an important role in increasing the concentration of indoor suspended particulate matter and occurrence of asthma in children in developing countries like India.

**Key words:** Indoor air pollution, SO<sub>2</sub>, NO<sub>2</sub>, SPM, asthmatic children, wheezing

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### Introduction

Urban air pollution primarily due to suspended particulate matter (SPM), nitrogen dioxide (NO<sub>2</sub>) and sulfur dioxide (SO<sub>2</sub>) is an environmental concern of many cities throughout the world. It is responsible for causing serious respiratory health problems like rhinitis, asthma, decreased resistance to respiratory infections, chronic obstructive pulmonary disease (COPD), chronic cough and phlegm production which lead to premature death in the exposed population [1]. Delhi, India's third largest city and its capital, is

also the third most polluted city in the country [2]. The main source of suspended particulate in Delhi are burning of fossil fuels, power stations, vehicular transport, industries, domestic coal and open biomass burning. Delhi's annual average concentration [3] of PM<sub>10</sub> (particulate matter with an aerodynamic diameter less than 10 mm) is the highest among major Asian cities, and was between 3 and 4 times the Indian Standard in 2001–2004.

Indoor air quality (IAQ) has gained great attention in the recent years, mainly due to the large amount of time we spend indoors in modern times. Indoor air pollution refers to chemical,

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biological and physical contamination of indoor air. It may result in adverse health effects [4]. In developing countries, the main source of indoor air pollution is biomass smoke which contains suspended particulate matter (SPM), nitrogen dioxide (NO<sub>2</sub>), sulphur dioxide (SO<sub>2</sub>), etc. The United States National Research Council (NRC) reports [5] that people spend more than 80% of their time indoors. Hence, they are exposed to pollutants generated within the indoor environment, as well as those from the outdoors, which may lead to increased exposure relative to that outdoors [6]. Indoor pollutants depend on both indoor and outdoor sources and removal processes, such as air exchange or chemical reactions [6]. A large number of indoor pollutants sources have been identified which include tobacco smoking, cooking with kerosene oil and wood burning [5, 6]. Combustion process is the main indoor source of smaller particles and gases, with the vast majority of them in the sub micrometer range, containing a host of organic as well as inorganic materials.

Prevalence of asthma has increased during the last decades in the countries worldwide. Vehicle exhausts have been implicated for an increased prevalence of wheeze, rhinitis, asthma and other respiratory symptoms in children [7]. Few studies [8, 9] have reported air pollution as a causative factor for asthma. In a 6-yr follow-up study [8] among Japanese children a significant association was found between the annual average concentration of nitrogen dioxide (NO<sub>2</sub>) and the incidence of asthma.

There are several studies in developed countries showing the association between indoor air pollution and asthma in children. There is lack of data of indoor air pollutant level capital city of India. To the best of our knowledge there is no study from India which correlates the relationship between indoor air pollutants (SO<sub>2</sub>, NO<sub>2</sub> and SPM) and asthma in children, hence this study was planned.

### Material and methods

This study was undertaken at Delhi, capital of India during 2004–2009 after ethical clearance from the Institutional Ethics Committee. According to Central Pollution Control Board (CPCB) [10], India's premier pollution monitoring authority, the study areas were divided in eight locations namely Ashok vihar (residential area), Janakpuri (residential area), Nizamuddin (residential area), Siri Fort (residential area), Shahdara (industrial area), Shahzada Bag (industrial area), Dallupura (Village) and Jagatpur (Village). Central

Pollution Control Board has outdoor pollution monitoring stations which measure daily pollutants levels in each of these study areas except villages. The 1 km area around the monitoring station of CPCB was taken for study in each area. Three colonies, one each representing the lower (family with income less than 3000 rupees per month), middle (family with 3000–5000 rupees monthly income) and upper (family with income more than 10,000 rupees monthly income) socioeconomic segments was randomly selected for the survey. In villages, there was no class wise distribution. Hundred houses with children aged 7–15 years from each socioeconomic class were selected for survey and health checkup. Indoor SO<sub>2</sub>, NO<sub>2</sub> and SPM levels were monitored in 25% houses from each area.

A questionnaire was developed on the basis of ATS (American Thoracic Society) [11], BMRC (British Medical Research Council) [12] and ISAAC (The International Study of Asthma and Allergies in Childhood) [13] questionnaires to detect the presence of symptoms suggestive of asthma. The questionnaire was also converted into Hindi. The questionnaire included built-in demographic details like age and sex, food habits, smoking status of child, smoking habits in the family, indoor structure of home, fuel used for cooking, idea about indoor air pollution, major chronic chest symptoms (cough, phlegm, shortness of breath, wheezing, chest illness). House visits were done by the survey team and the questionnaire was administered at the house itself. Examination of the child, their pulmonary function test or PEFR, was conducted. The diagnosis of asthma was made by the physician examining the children, based on the guidelines of ATS [11].

Spirometry test of children were done by the use of an electronic portable Spirometer. Maximal Expiratory Flow Volume (MEFV) curves were obtained as per American Thoracic Society (ATS) 1995 recommendations [14]. In the children who could not perform spirometry, Peak Expiratory Flow Rate (PEFR) was obtained with a Wright's Peak Flow Meter. The highest of the three recordings was noted.

Indoor SO<sub>2</sub>, NO<sub>2</sub> and SPM pollutants were monitored by the methodology adopted as in earlier studies [15, 16]. Indoor SO<sub>2</sub>, NO<sub>2</sub> and SPM levels were measured by using the Handy Air Sampler or Low Volume Sampler (APM 810) with a flow rate of 1 LPM (liter per minute) with 6–8 hours of sampling period. Handy Air Sampler for indoor samples was positioned in the center or corner of the room, with the inlet roughly 1 m above the ground level, corresponding with

the breathing height of the children. The indoor sulfur dioxide and nitrogen dioxide concentration were measured by West and Gaeke Modified Method [15] and Hochheiser Modified Method [16] respectively.

The statistical analysis was performed with SPSS statistical software. The groups were compared for all variables using the Student t-test to compare equality for means and the chi square test to compare category value. The indoor SO<sub>2</sub>, NO<sub>2</sub> and SPM levels were compared in the three areas (industrial, residential and village) using analysis of variance (ANOVA) followed by Post Hoc Bonferroni multiple comparison test. The differences were considered to be statistically significant at the  $p < 0.05$  (two tailed test) level. Results are presented as percentage and mean  $\pm$  SD.

## Results

There were 6613 houses which were surveyed and they had a total of 3104 children. Some of the houses had more than one child. Consent for study was given by 2513 houses having a total of 3104 children (60.3% males and 39.7% females). The details of the findings of the survey are shown in Table 1 and Figure 1.

Over all a total of 7.9% ( $n = 244$ ) children were diagnosed as having asthma. Diagnosis of asthma varied in different areas but was the highest in Shahdara (14.2%) (Fig. 1). Asthma in children was significantly more frequent in industrial areas than residential and village areas (Table 2). The number of children with asthma was significantly higher in upper socioeconomic class compared to others (Table 1) with  $p$  value  $< 0.001$ . The various characteristics of children with and without asthma were compared and are shown in Table 3.

Indoor SO<sub>2</sub>, NO<sub>2</sub> and SPM level were measured in 819 houses. The mean level of indoor SO<sub>2</sub>, NO<sub>2</sub> and SPM was  $4.28 \pm 4.61$  mg/m<sup>3</sup> (0.00 to 41.93 mg/m<sup>3</sup>),  $26.70 \pm 17.72$  mg/m<sup>3</sup> (0.00 to 141.13 mg/m<sup>3</sup>) and  $722.0 \pm 457.6$  mg/m<sup>3</sup> (80 to 2420 mg/m<sup>3</sup>) respectively. Concentration of indoor air pollutants varied in each area (Fig. 1). Indoor SPM and NO<sub>2</sub> levels were significantly higher in industrial areas followed by residential and village areas (Table 4). The factors affecting the levels of indoor air pollutants are shown in Table 5. Use of biomass fuel and occupancy per room of more than 4 was significantly associated with increased SO<sub>2</sub> levels. SPM levels were significantly higher with presence of smoker in family and increased occupancy ( $> 4/\text{room}$ ).

Mean indoor SPM levels were significantly higher in the houses of asthmatic children in all areas (Fig. 2) and the difference was statistically significant. The mean indoor NO<sub>2</sub> levels were higher in houses of asthmatic children in all areas (Fig. 3) but the difference was not statistically significant. The SO<sub>2</sub> levels were higher in houses of asthmatic children in industrial and village areas but the difference did not achieve statistical significance (Fig. 4).

## Discussion

The major sources of air pollution in any city like Delhi are industrial emissions, residential heating and cooking, vehicular traffic and natural sources, i.e., dust, wind. Suspended particulate matter, sulfur dioxide and nitrogen dioxide are the three major air pollutants in Delhi [17]. Indoor coal combustion is the major source of indoor particulate matter. The suspended particle concentration levels found in the kitchens are very high. Indoor sources of NO<sub>2</sub> include cigarette smoke, gas and oil heaters and cookers which often result in high indoor concentrations [18].

According to WHO air quality guidelines (global update 2005) [19], the recommended permissible limit for SO<sub>2</sub> is 20  $\mu\text{g}/\text{m}^3$  (24hr mean), for NO<sub>2</sub> is 40  $\mu\text{g}/\text{m}^3$  (annual mean) and for SPM is 20  $\mu\text{g}/\text{m}^3$  (24hr mean). Morand et al, [20] studied the long-term exposure of air pollutants in France and found that mean levels of SO<sub>2</sub>, NO<sub>2</sub> and PM<sub>10</sub> were 9.6 mg/m<sup>3</sup>, 40.6 mg/m<sup>3</sup> and 23.8 mg/m<sup>3</sup> respectively. In London [21] the SO<sub>2</sub> and PM<sub>10</sub> concentrations were  $21.2 \pm 7.8$  mg/m<sup>3</sup> and  $28.5 \pm 13.7$  mg/m<sup>3</sup> respectively. In India, in Garhwal [22] the mean level of indoor total suspended particulate (TSP) during cooking by wood and shrubs were found to be 4500  $\mu\text{g}/\text{m}^3$ . In another place in India i.e. Pune [23], the 12–24 hours mean level of indoor PM<sub>10</sub> during cooking by wood was 2000  $\mu\text{g}/\text{m}^3$ . In Tamil Nadu (India) [24] the mean level of indoor TSP during cooking by biomass was 500–2000  $\mu\text{g}/\text{m}^3$ . In the present study, the indoor SO<sub>2</sub>, NO<sub>2</sub> and SPM were found to be  $4.28 \pm 4.61$  mg/m<sup>3</sup>,  $26.70 \pm 17.72$  mg/m<sup>3</sup> and  $722.0 \pm 457.6$  mg/m<sup>3</sup> respectively. Indoor SO<sub>2</sub> concentration is low in comparison to other countries which may be explained probably by low overall outdoor SO<sub>2</sub> due to introduction of compressed natural gas (CNG) fuel in vehicles since 2001. The high levels of indoor NO<sub>2</sub> and SPM are consistent with the above studies.

The prevalence of asthma has increased worldwide during the past two or three decades

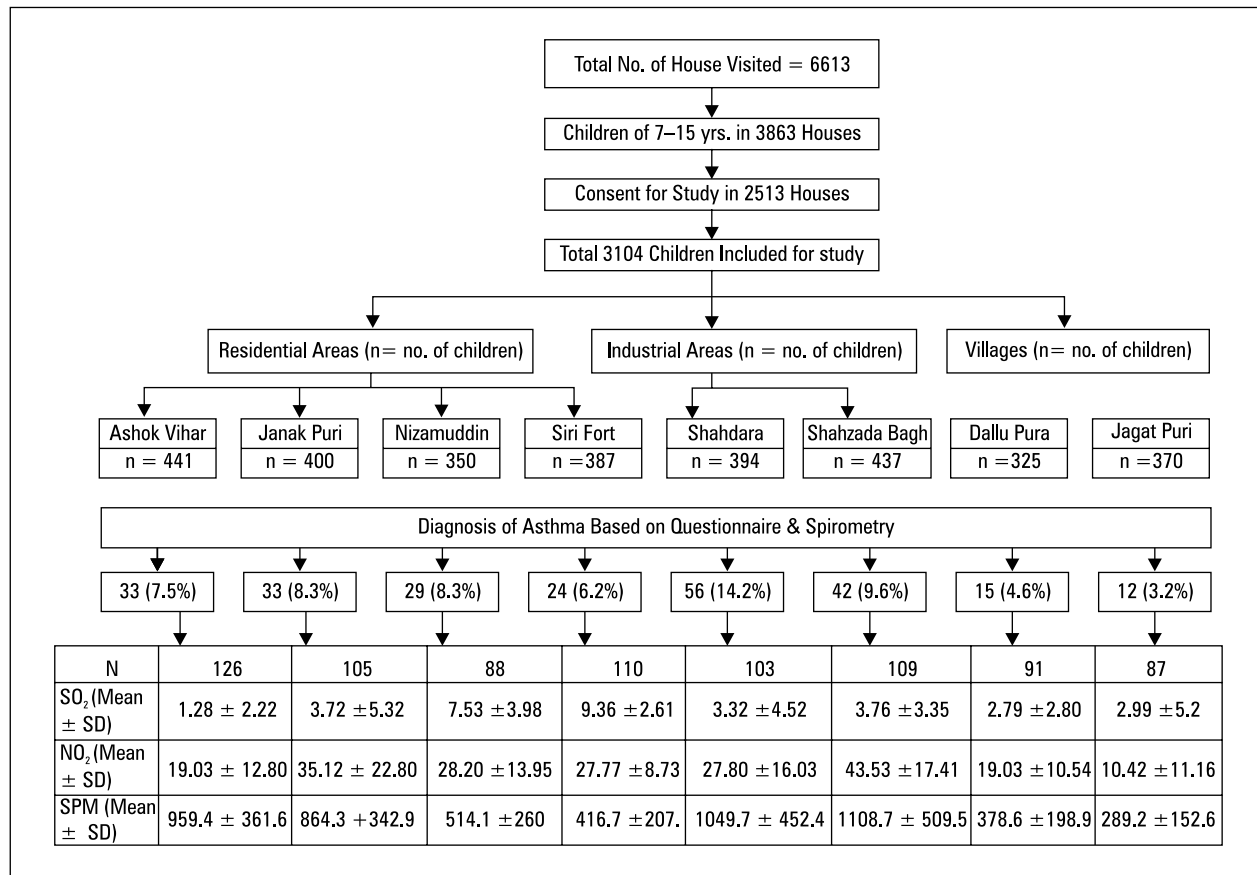
**Table 1. General profile of children**

Profile of Child	Socioeconomic Status of Children				Total
	Lower	Middle	Upper	Villages	
Children studied	801 (25.8%)	821 (26.4%)	787 (25.4%)	695 (22.4%)	3104
Number of children diagnosed with asthma	73 (9.11%)	65 (7.91%)	79 (10.03%)	27 (3.88%)	244 (7.9%)
Male	63.5%	56.0%	58.4%	63.6%	60.3%
Female	36.5%	44.0%	41.6%	36.4%	39.7%
Vegetarian	81.1%	56.4%	40.9%	53.7%	58.2%
Non-Vegetarian	18.9%	43.6%	59.1%	46.3%	41.8%
Students	88%	98.9%	99.4%	98.5%	96.0%
Go to school by bus	35.1%	40.1%	59.1%	18.4%	39.0%
Go to school on foot	64.9%	59.9%	40.9%	81.6%	61.0%
History of smoking	1.1%	0.1%	0	0	0.3%
Children exposed to Environmental Tobacco Smoke (ETS)	50.3%	24.6%	20.7%	39.7%	32.4%
≤ 4 person occupancy per Room	42.2%	97.7%	98.2%	73.7%	78.1%
> 4 person occupancy per Room	57.8%	2.3%	1.8%	26.3%	21.9%
LP Gas used for cooking	22.3%	99.5%	99.9%	49.6%	68.5%
Biomass fuel used for cooking	77.7%	0.5%	0.1%	50.4	31.5%
Idea (knowledge) about indoor air pollution	11.5%	45.4%	71.3%	12.9%	36.0%
History of cough	54.2%	48.8%	47.5%	22.3%	43.9%
History of phlegm production	28.3%	25.1%	24.4%	7.9%	21.9%
History of shortness of breath	27.7%	20.2%	19.2%	8.5%	19.3%
History of wheezing	19.6%	14.6%	14.9%	5.9%	14.0%
Family history of chest diseases	4.7%	10.4%	15.9%	2.2%	8.5%
Airway obstruction	7.4%	7.2%	8.0%	6.3%	7.2%

especially in children and young adults. According to a study [25] conducted in Britain, in England and Scotland, the prevalence of current asthma in children increased from around 3% in 1982 to 6% in 1985, and nearly reached 9% in 1988. In Wales [26], current asthma increased in children from 4% in 1973 to 9% in 1988. In a study [27] in Chandigarh, India, the prevalence of asthma in 9 to 20-years-olds schoolchildren was 2.3%. In India, Mishra [28] also studied the effect of indoor air pollution from biomass combustion on prevalence of asthma in the elderly and found the prevalence of asthma in more than 60-years-olds was around 8–10%. In our study the prevalence of asthma was 7.9% (3.2% to 14.2% in different areas). It was highest in industrial areas (11.8%) followed by residential areas (7.4%) and village areas (3.9%) and the difference was

statistically significant. The area wise prevalence of asthma was the lowest in Jagatpur & Dallupura village with significantly low pollution levels. Jagatpur village is situated near the bank of river Yamuna, agricultural fields and green biodiversity park of Delhi.

There is a substantial epidemiological evidence indicating a link between air pollution and asthma morbidity including deterioration in lung functions, increased number of emergency department visits and hospital admissions [29]. A cross-sectional epidemiologic study [26] done in six French cities found the association between long-term exposure to air pollution and asthma. Asthma was found to be positively related to an increase in the exposure to SO<sub>2</sub> (9.6 mg/m<sup>3</sup>) and PM<sub>10</sub> (23.8 mg/m<sup>3</sup>) but there was no consistent positive association between NO<sub>2</sub> (40.6 mg/m<sup>3</sup>)



**Figure 1.** Flow chart depicting study design and levels of SO<sub>2</sub>, NO<sub>2</sub> and SPM in different study areas

**Table 2.** Asthma in children at industrial, residential and village areas

Area of Monitoring	No. of children studied	No. of children diagnosed with asthma	%age of children with Asthma	Comparison of no. of asthmatic children in different areas p-value
Industrial	831	98	11.8%	
Residential	1578	119	7.5%	Industrial vs Residential < 0.001
Villages	695	27	3.9%	Industrial vs Villages < 0.0001
Total	3104	244	7.9%	Residential vs Villages < 0.01

**Table 3.** Comparison of various characteristics in children with asthma and without asthma

	Children diagnosed with Asthma	Children without asthma	p-value
Male: Female	153: 91	1718: 1142	NS
Vegetarian: Non-vegetarian	86: 158	1209: 1650	NS
Smoking: No Smoker in family	89: 155	1015: 1845	NS
Fuel for cooking LPG: Biomass fuel	195: 45	2340: 520	NS
Kitchen with exhaust present: not present	144: 100	1467: 1393	p < 0.03
History of recurrent rhinitis present: absent	195: 49	640: 2220	p < 0.0001
History of recurrent upper respiratory tract infection present: absent	176: 68	531: 2329	p < 0.0001
Family history of asthma present: absent	48: 196	215: 2645	p < 0.0001

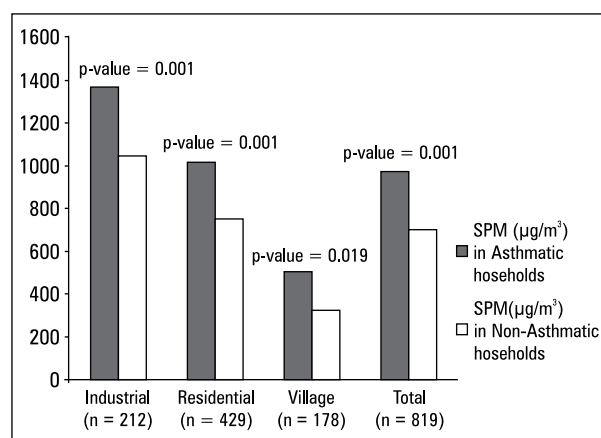
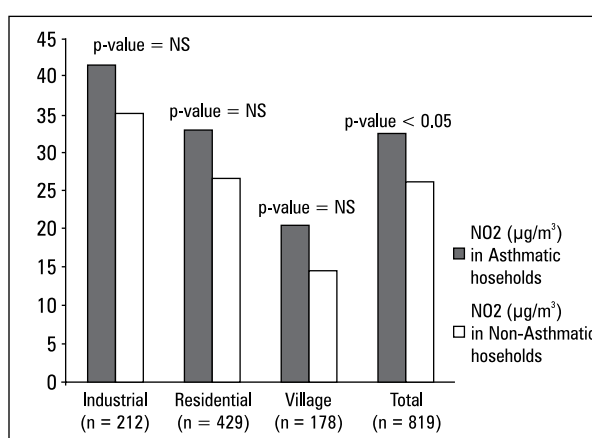
**Table 4. Comparison of levels of SO<sub>2</sub>, NO<sub>2</sub> and SPM (suspended particulate matter) in different areas**

Type of Area	SO <sub>2</sub> (μg/m <sup>3</sup> ) Mean ± SD	NO <sub>2</sub> (μg/m <sup>3</sup> ) Mean ± SD	SPM (μg/m <sup>3</sup> ) Mean ± SD
Industrial (n = 212)	3.54 ± 3.95 <sup>a</sup>	35.88 ± 18.47 <sup>a</sup>	1080 ± 482.36 <sup>a</sup>
Residential (n = 429)	5.22 ± 4.88 <sup>b</sup>	27.09 ± 16.38 <sup>b</sup>	705.6 ± 381.61 <sup>b</sup>
Villages (n = 178)	2.88 ± 4.13 <sup>a</sup>	14.82 ± 11.64 <sup>c</sup>	334.9 ± 182.87 <sup>c</sup>
F-ratio	20.885	83.295	187.649
P-value	p < 0.0001	p < 0.0001	p < 0.0001

N.B. — variation in superscript indicates significance of difference

**Table 5. Indoor air pollutants and factors influencing them**

Factor studied	Status of factor	Mean (± SD) SO <sub>2</sub> levels (μg/m <sup>3</sup> )	Mean (± SD) NO <sub>2</sub> levels (μg/m <sup>3</sup> )	Mean (± SD) SPM levels (mg/m <sup>3</sup> )
Smoker present in family	No	4.46 ± 4.43	29.71 ± 20.75	660 ± 420
	Yes	5.08 ± 7.49	32.64 ± 29.14	780 ± 470
	p-value	NS	NS	< 0.001
Occupancy per room	≤ 4	4.24 ± 4.81	28.91 ± 20.21	680 ± 430
	> 4	6.06 ± 8.10	37.93 ± 34.36	830 ± 430
	p-value	< 0.0005	< 0.0002	< 0.0002
Biomass fuel used for cooking	No	4.24 ± 4.28	30.26 ± 18.60	710 ± 430
	Yes	5.52 ± 8.12	31.80 ± 33.94	690 ± 460
	p-value	< 0.020	NS	NS

**Figure 2.** Comparison of mean SPM levels in households having asthmatic children versus households having non-asthmatic children in different areas**Figure 3.** Comparison of mean NO<sub>2</sub> levels in households having asthmatic children versus households having non-asthmatic children in different areas

and asthma. Modig et al. [30] studied the association between the exposure to air pollutants and increased risk of asthma in adults and found that the association between asthma and measured NO<sub>2</sub> was weak and not significant. However, in a 6-yr follow-up study [8] among Japanese children a significant association was found between

the annual average concentration of nitrogen dioxide (NO<sub>2</sub>) and the incidence of asthma.

A study [31] in Hong Kong, China was conducted to find out association of air pollution and asthma admission among children and it concluded that the ambient levels of PM<sub>10</sub> and NO<sub>2</sub> but not SO<sub>2</sub>, were associated with childhood

asthma hospital admissions. In a longitudinal study of 150 preschool children with asthma (Baltimore Indoor Environment Study of Asthma in Kids [BIESAK] Study), the impact of indoor fine (PM<sub>2.5</sub>) and coarse PM (PM<sub>2.5-10</sub>) on asthma was investigated [32]. The study found that indoor coarse PM concentrations were associated with substantial increases in asthma symptoms and the fine PM were also positively associated with increased respiratory symptoms and rescue medication use. These studies are consistent with our study in which the diagnosed asthma in children was associated with the highest levels of indoor suspended particulate matter (SPM) ( $p = 0.001$ ) and NO<sub>2</sub> ( $p = 0.036$ ). Indoor SPM was significantly higher in the houses of asthmatic

children of industrial ( $p = 0.001$ ), residential ( $p = 0.001$ ) and village ( $p = 0.019$ ) areas.

We also did the statistical analysis of the data using logistic regression analysis with variable selection technique and found the drivers responsible in our study cohort for occurrence of asthma in children. These have been depicted in Table 6. Noticeably, the number of years of breast feeding was the most important driver which had a protective influence on the occurrence of asthma in children.

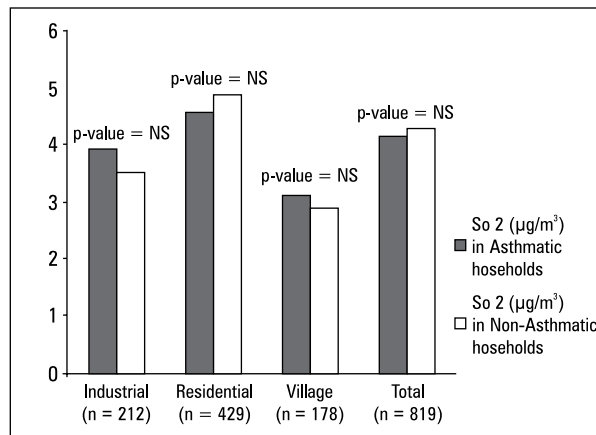
Inability to obtain 24 hour mean values of indoor air pollutants remained a limitation of our study.

## Conclusion

The present research was carried out to study the relationship between indoor air pollutants level and asthma in children. Both indoor SPM levels and occurrence of asthma in children were found to be higher in industrial areas compared to residential and urban village areas. Further, the houses with asthmatic children in all these areas had still higher levels of indoor SPM as compared to houses without asthmatic children and the difference was statistically significant. Hence, this study suggests that industry plays an important role in increasing the concentration of indoor suspended particulate matter, and also increased occurrence of asthma in children in developing countries like India.

## Conflict of interest

The authors declare no conflict of interest.



**Figure 4.** Comparison of mean SO<sub>2</sub> levels in households having asthmatic children versus households having non-asthmatic children in different areas

**Table 6. Factors affecting the occurrence of childhood asthma\***

Variable	Driver rank	Importance	Standardized estimate	Impact
Years of breast-feeding	1	52%	-1.5679	-
Number of sisters	2	9%	-0.2739	-
Industrial area	3	9%	0.2546	+
Air quality in the locality	4	6%	0.1776	+
Number of brothers	5	6%	-0.1667	-
Socioeconomic status	6	5%	0.1511	+
Child suffering from any disease	7	5%	0.1437	+
Family history	8	3%	0.0907	+
Food habit — vegetarian	9	3%	-0.0902	-
Animals & pets	10	3%	0.0756	+

\*Method used: Logistic regression with variable selection technique; Significant level  $\leq 0.10$  (indicating about 90% probability of the impact of the driver on the occurrence of asthma)

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