Environmental health survey in asbestos cement sheets manufacturing industry

Abstract
About 673 small-scale asbestos mining and milling facilities and 33 large-scale asbestos manufacturing plants, (17 asbestos-cement product manufacturing plants and 16 other than asbestos-cement product plants) are situated in India. The present study reveals the exposure of commercial asbestos (chrysotile) in the occupational as well as ambient air environment of the asbestos-cement (AC) sheets industry using membrane filter method of Bureau of Indian Standards (BIS). The fibre concentrations in 15 samples collected in the occupational environment at ingredient feeding site, sheet-producing site, fibre godown were 0.079, 0.057 and 0.078 f/cc, respectively and in five samples from surrounding ambient air at factory gate resulted fibre concentration of 0.071 f/cc. All the samples have shown fibre concentration lower than the threshold limit values (TLVs) prescribed by BIS. Morphological analysis of samples, further under phase contrast and polarized microscopy indicates the presence of chrysotile asbestos, which acts as carcinogen as well as co-carcinogen. A clinical examination of exposed subjects reveals that there was no case of clubbing, crepitation, rhonchi and dyspnea on exertion; however, obstruction and restriction were 10.9 per cent and 25 per cent in exposed subjects, respectively while in control there were 12 per cent and 28 per cent, respectively. The study revealed that chrysotile asbestos is emitted in the occupational as well as ambient environment that may cause adverse health impact.

Key words: Asbestos-cement sheets, chrysotile, pulmonary function test, respiratory morbidity

INTRODUCTION
Asbestos is a fibrous mineral occurring in natural deposits. Asbestos fibres are divided into two classes, serpentine and amphibole, on the basis of their crystal structure.[1] Chrysotile is a fibrous hydrated magnesium silicate mineral [Mg₆Si₄(OH)₁₀], which is used, in about 3,000 commercial products.[5] In reference of the situation persisted few decades earlier; presently the exposure to asbestos fibre is restricted in developed and industrialized countries and pressure has started mounting in the developing countries.[3] The delay between exposure and manifestation of diseases is however, still escalating in both developed and undeveloped countries. Chrysotile, the commercial variety of asbestos is known to cause mesothelioma.[6] Although, chrysotile is the most common variety of asbestos, it accounts for over 95 per cent of the world production and is exploited in more than 40 countries. AC industry is the largest user of chrysotile asbestos through out the world and AC products are made stronger by the addition of approximately 5% to 10% of chrysotile asbestos during mixing of cement[7] because asbestos provides the desired strength to the products. The most important products based on chrysotile fibres are AC sheets and AC pipes as mentioned earlier however, while other products of major importance are friction materials, insulation boards, millboard and brake - shoe etc.

Asbestos-cement makes up 85 per cent of all commercial applications[8] and these products are also being used as low-cost building materials.[6] Currently, manufacturing of chrysotile products is undertaken in more than 100 countries and production is about 27 to 30 million tons annually. India consumes about one lakh tons of chrysotile every year, mostly imported from Canada, Brazil, Kazakhstan, Russia and South Africa.[9] AC products are manufactured mostly in wet process.

Although, air pollution levels of asbestos were reported to be elevated in the areas surrounded by asbestos industries.[3] The emission of asbestos may be negligible to significant depending on its rate of emission and the fibre control measures and technology.[4] Other little possible emission sources during the processing of these products may be feeding of asbestos fibres into mixing, blending
the mixture, cutting and finishing of end products. Cement particles, asbestos fibres and agglomerates of particles and fibres are therefore, released in the surrounding and may be dispersed in air and water in large amounts.\(^9\)

Dave and Beckett, 2005 reported in the past in India there have been nearly 673 small - scale asbestos mining and milling facilities and 33 large - scale asbestos manufacturing plants, (17 AC product manufacturing plants and 16 other than AC product plants). In India, about two to three million active workers are suffering from exposure to asbestos and other dusts or fibres.\(^{10}\) It is well reported that occupational exposure of asbestos may cause simple inflammatory reactions, asbestosis, to bronchogenic carcinoma.\(^{11}\)

An environmental health survey was conducted in and around the surrounding area of asbestos-cement sheets manufacturing industry to observe the occupational asbestos exposure burden. The monitoring and analysis of the asbestos concentration and its type around the asbestos industries address many questions regarding source, distribution and transport. Assessment of all these parameters in any occupational environment where asbestos is present is thus relevant.\(^{12}\) The information obtained on the levels of asbestos concentration and its type in the occupational and ambient environment can be used to assess the asbestos fibre exposure and its burden in the vicinity of the industry.

**MATERIALS AND METHODS**

**Study design**

Asbestos burden in and around the industry was assessed in AC sheets manufacturing industry, India. As per the information provided by the industry staff, industry is a large - scale with production capacity of 36,000 metric tons per annum of asbestos cement sheets and asbestos - based moulded goods. The ingredients used are cement, fly - ash and chrysotile asbestos (imported from Russia) in quantity (Metric tons) 20,000, 12,000 and 4,000 per annum respectively. In this study a total of 71 subjects in asbestos exposed and 69 control groups were appraised.

**Manufacturing process**

- The products are processed in wet mode technically known as "Hatschek Process".
- Pressure packed impermeable polythene bags of chrysotile are opened by semi - automatic machine through mechanical process and milled in Hydro Disintegrator (mill), transferred to a mixing tank via close system. The binding material (cement and fly ash) mixed with water to make slurry, which is fed to the Cylinder Vat (a tank with a sieve cylinder covered by mesh cloth help to sieves the slurry) through the Homogeniser Feeding Cone. Cylinder rotates leaving a thin film of stock deposited at its surface and film was transferred on to endless felt, which remains in contact with the top cover of the sieve cylinder.
- Excess water was being removed from the felt by means of vacuum boxes placed under the felt as it travels towards sheet formation drum in continuous operation until the sheet prepared to build up the desired thickness. The sheets are then knifed along a groove in the sheet formation drum roll and peeled to a moving rubber conveyor belt, which collect the sheet clear from the machine.
- In the sheet corrugation and demoulding section, the wet plain sheets are corrugated by means of template. The corrugated wet sheets stacked on a trolley and allowed for initial maturity of 15-18 hours. After that sheets demoulded i.e., stripped off from the templates. Finally at Curing Section, these sheets are stacked vertically and water poured on them here, sheets covers 25-28 days to develop optimum strength before being dispatched.
- Negative pressure in all these process equipments is maintained by induced draft fan. Discharge of the fan were connected at air pollution control device i.e., counter current scrubber such that if the particulate travels along with air (sucked for maintaining negative pressure) finally trapped by automatized water spray in the scrubber device.

**Fibre monitoring, sampling devices and methodology**

Asbestos sampling was done according to the standard procedure of Bureau of Indian Standard - 11450. Air monitoring for asbestos was done by using a 25 mm diameter, easter cellulose filter (0.8-1.2 µm pore size) at constant flow rate per liter per min. Sampler Model XX5700000 and Low Volume Vacuum/ Pressure Pump Model XX 5600002 attached with filter holder (cowl) MAWP 025AC (Millipore Corporation, USA) were used for the collection of air samples. In both the subjects, filter holders were kept in downward position during sampling to avoid the contamination of heavy particles on filters.\(^{13}\)

**Sample transportation, preparation and analysis**

The collected samples were stored in upward position with sealed boxes and transferred to the laboratory for further analysis as per acetone triacitin technique.\(^{14}\) The mounted samples were analyzed for the presence of asbestos fibre at a magnification of 400x by using phase contrast and polarized light microscope. Asbestos fibres (>5 µm length, < 3 µm in diameter and length to diameter ratio 3:1) were considered for counting and scanned under the phase contrast microscope (LABROX, Germany).\(^{14,15}\)

**Clinical examination**

Subjects working in asbestos industry (exposed) and subjects not exposed (control) to such environment have been appraised with the scope of the study and consent was received from each of them. The care was taken during the selection of subjects from asbestos exposed environment and subjects not exposed...
to such environment (matching controls) are of same socio-economic status. Each subject was given to answer a complete set of questionnaire based on the modified British Medical Research Council[20] to assess an accurate medical history, habits, past and present occupation, duration of exposure along with medical history (previous and present) and respiratory history. A history of cough, sputum production, wheezing and chest pain, occupational history, domestic exposure, smoking, alcohol consumption and nutritional habits were also recorded. Each subject was thoroughly examined by a medical staff having graduation in medical science and special emphasis was given on respiratory system.

**Pulmonary function test**

Pulmonary function tests were performed by forced spirometry in a total of 64 exposed subjects out of total 71 subjects and 50 control subjects out of total 69 subjects using OHD - KoKo Spirometer, USA, following the guidelines of American Thoracic Society.[17] Spirometry is a medical test that measures volume of the air inhaled or exhaled by the subject at a function of time. The appropriate time for this test is recommended between 10 - 12 noon, was followed for Spirometry. In this test, firstly the vital capacity (VC) was measured followed by Force vital capacity (FVC), both the tests are measured in standing position.[18] These tests were performed at least three times and the best of the three was selected for the data analysis. Age, height and weight of the subjects were recorded to predict the normal values of pulmonary function test.

**Roentgenographic studies**

Chest X-ray (PA View) of 55 subjects was taken on 300 MA machine. A panel of three specialists (a radiologist, a chest physician and a specialist in industrial medicine) studied each X-ray. While studying the chest X-ray, occupational history and clinical findings were also taken into consideration. Diagnosis of each subject was made after complete review of clinical history, occupational history, X-ray findings and clinical examination following the criteria of International Labor Organization[19] especially for the presence of (a) Linear shadows of varying thickness, (b) Pleural thickening, (c) Pleural plaques, (d) Bilateral or unilateral pleural calcification, (e) Honey combing, (f) Reticulonodular pattern and (g) Prominent broncho-vascular marking.

**Sputum analysis**

Sputum samples were collected in clean sterilized bottles from the deep of the throat (preferably early morning sample) of the suspected subjects for AFB analysis by Ziehl-Neelsen staining[20] and asbestos bodies analysis following the standard methodology.[21]

**RESULTS**

The concentrations of asbestos fibres (f/cc) at ingredient feeding site, sheet-producing site, fibre godown site and factory gate were 0.079 ± 0.00, 0.057 ± 0.01, 0.078 ± 0.01 and 0.071 ± 0.01, respectively [Table 1]. All the values were found less than the proposed Threshold Limit Value (0.1 f/cc). Fibres, analyzed by phase contrast and polarized light microscopy, were found to be of chrysotile variety (white asbestos). Personal characteristics of subjects such as age, height, weight, smoking habit, alcohol consumption, food habit, domestic fuel exposure and family type are pooled in the Table 2.

The subjects exposed to asbestos (total years including previous) are sub-grouped depending upon their occupational exposure [Table 3]. Significantly higher number of asbestos-exposed subjects were in the sub-grouped of two to four years and minimum number in eight to 10 years and more than 10 years. The observed morbidity pattern is mentioned in the Table 4. The prevalence of hypertension in exposed subjects was 16.9 per cent while in control 5.79 per cent. Upper

![Table 1: Concentration of asbestos fibre in the occupational and ambient environment](image)

<table>
<thead>
<tr>
<th>Location</th>
<th>Concentration, f/cc*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ingredient feeding site</td>
<td>0.079 ± 0.00</td>
</tr>
<tr>
<td>Sheet producing site</td>
<td>0.057 ± 0.01</td>
</tr>
<tr>
<td>Fibre godown site</td>
<td>0.078 ± 0.01</td>
</tr>
<tr>
<td>Factory gate</td>
<td>0.071 ± 0.01</td>
</tr>
</tbody>
</table>

*Mean ± SD, Figures in parentheses are in percentage

![Table 2: Personal characteristics of asbestos exposed and control subjects](image)

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Exposed (N = 71)</th>
<th>Controls (N = 69)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)*</td>
<td>29.1 ± 7.43</td>
<td>28.1 ± 3.4</td>
</tr>
<tr>
<td>Height (cm)*</td>
<td>162.7 ± 6.6</td>
<td>164.5 ± 4.6</td>
</tr>
<tr>
<td>Weight (kg)*</td>
<td>58.8 ± 9.7</td>
<td>54.6 ± 7.7</td>
</tr>
<tr>
<td>Smokers</td>
<td>7 (9.5)</td>
<td>13 (18.84)</td>
</tr>
<tr>
<td>Non-smokers</td>
<td>64 (90.15)</td>
<td>56 (81.15)</td>
</tr>
<tr>
<td>Tobacco smokers</td>
<td>5 (7.04)</td>
<td>3 (4.34)</td>
</tr>
<tr>
<td>Alcohol users</td>
<td>2 (2.81)</td>
<td>3 (4.34)</td>
</tr>
<tr>
<td>Family type-Nuclear</td>
<td>30 (42.25)</td>
<td>44 (63.76)</td>
</tr>
<tr>
<td>Family type-joint</td>
<td>36 (50.70)</td>
<td>44 (63.76)</td>
</tr>
<tr>
<td>Blood pressure (mm of Hg)</td>
<td>35 (49.30)</td>
<td>25 (36.23)</td>
</tr>
<tr>
<td>Systolic*</td>
<td>117 ± 13.7</td>
<td>116.5 ± 14.2</td>
</tr>
<tr>
<td>&gt; 140</td>
<td>5 (7.04%)</td>
<td>3 (4.34)</td>
</tr>
<tr>
<td>Diastolic*</td>
<td>77.85 ± 9.9</td>
<td>79.9 ± 7.4</td>
</tr>
<tr>
<td>&gt; 90</td>
<td>4 (5.63)</td>
<td>8 (11.59)</td>
</tr>
<tr>
<td>Pulse (b/min.)*</td>
<td>74.3 ± 3.37</td>
<td>75.3 ± 4.2</td>
</tr>
</tbody>
</table>

*X ± Sd, Figures in parentheses are in percentage

![Table 3: Number of subjects (N = 71) according to their occupational exposure](image)

<table>
<thead>
<tr>
<th>Exposure years (yr)</th>
<th>Per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-2</td>
<td>27 (38.02)</td>
</tr>
<tr>
<td>&gt; 2-4</td>
<td>36 (50.70)</td>
</tr>
<tr>
<td>&gt; 4-6</td>
<td>3 (4.22)</td>
</tr>
<tr>
<td>&gt; 6-8</td>
<td>3 (4.22)</td>
</tr>
<tr>
<td>&gt; 8-10</td>
<td>1 (1.40)</td>
</tr>
<tr>
<td>&gt; 10</td>
<td>1 (1.40)</td>
</tr>
</tbody>
</table>
respiratory tract infection (URTI) levels were almost 10% in both the populations. The prevalence of musculo-skeletal morbidity was 7.04 per cent, among them 5.63 per cent due to pain in joints and 1.4 per cent from backache in exposed population. In controls no subject has pain in joints while the prevalence of backache and bodyache were 5.79 per cent and 5.79%, respectively. Hypertension was 16.90% and 5.79% in the exposed and unexposed populations, respectively. Other morbidities listed in the Table 4 were also insignificantly higher in the exposed population.

Lung function impairments were recorded 6.25 % of obstruction and 25% of restriction in asbestos-exposed subjects against 8.62% of obstruction and 31.03% of restriction in control subjects which may be due to higher smoking pattern in control subjects [Figure 1]. Out of total 71 asbestos-exposed subjects 55 cooperated for chest radiography and found them normal whereas no one cooperated for X-ray amongst control. Out of 71 asbestos-exposed subjects only 32 subjects whereas 37 out of 69 control subjects cooperated for sputum analysis for the presence of acid-fast bacilli (AFB) and asbestos body. Notably, all were negative including exposed subjects.

**DISCUSSION**

The asbestos fibre concentration was measured in the ambient environment of the AC industry because asbestos mainly presents risk in airborne condition. It is well reported that exposure to asbestos caused serious health effects on the population. [22] In the exposure to low level of asbestos during improper handling and processing and also may be from transportation and waste disposal. [23] In our study analysis of fibres under phase contrast and polarized light microscopy reported chrysotile asbestos, which acts as carcinogen and co-carcinogen. [3,11]

In India, an estimated one-lakh people are exposed to asbestos at work place. [24] The processing for manufacturing of Asbestos-based products increases the air pollution levels in the areas surrounded by the asbestos-based industries [25,26] and health risk to the workers. Moreover, adverse health effects are interrelated to dust exposure in workplace. [27] The people residing in the vicinity of the asbestos-based industries may be exposed to higher levels of asbestos fibre concentration. [3,11]

The environmental profiles of asbestos was observed ranging from 0.057 to 0.079 f/cc including work zone area. The lowest concentration (0.057 f/cc) was observed at sheet producing site and the evident reason might be a complete wet process. This showed healthy industrial hygiene conditions, as the fibre levels are well within the proposed Indian and International Standards (0.10 f/cc). Although exposure to low level of

![Figure 1: Pulmonary function impairment in exposed and control subjects](image-url)
asbestos for long term period can register bioaccumulation and biopersistance in the biological systems including respiratory.\textsuperscript{[28]}

The increasing risk of developing a variety of lung diseases in asbestos exposed workers is a major health concern.\textsuperscript{[29]} Epidemiological and experimental studies have proven that the presence of predisposing factors such as cigarette smoke and bio-mass fuel exposure play an important role in biopersistence of asbestos fibres in lung which resulted enhanced pulmonary inflammatory reactions that accelerate the asbestos induced disease processes.\textsuperscript{[30,31]} Occupational or environmental exposure of asbestos fibres mainly concerns to lung diseases such as bronchogenic carcinoma.\textsuperscript{[29]}

The relatively higher respiratory problems of 10.14\% were observed in control subjects as compared to asbestos-exposed subjects of 9.85\%, which might be due to their exposure at domestic level in indoor environment. The lung function impairments were relatively higher in controls that might be due to higher smoking habits and wood exposure at domestic level during the cooking in indoor environments. The synergistic interaction of asbestos and unprocessed biomass fuels play an important role in asbestos-related malignancies\textsuperscript{[32]} and in our study as most of the workers were nonsmokers. Clinical studies suggested that the use of cow dung and wood is associated with functional changes of airway function.\textsuperscript{[33]}

Radiological examinations reveal no positive case in asbestos-exposed subjects. Radiological abnormalities have already been well demonstrated in asbestos-exposed subjects.\textsuperscript{[19]} Earlier studies also suggested that the functional abnormalities resulting due to exposure of asbestos precede radiographic changes\textsuperscript{[35]} that appear on the postero-anterior view of chest X-ray film. Reticulo-nodular infiltrates and presence of calcified pleural plaques with increased bronchovascular markings are the suggestive feature of exposure to asbestos.\textsuperscript{[36]}

Sputum was also collected from the subjects to analyze the asbestos bodies and AFB. Asbestos bodies in sputum samples are considered as hallmark of asbestos exposure.\textsuperscript{[37]} No one was reported with positive finding of asbestos bodies and AFB.

CONCLUSION

The industrial hygiene status of AC sheets manufacturing industry was in accordance with the National and International Standards. The conclusion is derived from our observations on the environmental levels of asbestos, appropriate pollution control engineering devices, less exposure period due to new establishment of plant, absence of asbestos bodies in workers sputum and radiological negative chest data. Though, prevalence of smoking in industrial workers was lower than the controls, there should be some mechanism for gradual reduction to zero level as smoking accelerates the disease process induced by asbestos. Perhaps incentive in some form, to nonsmoking workers may be a suitable option in this regard. Occupational exposure to asbestos even of its levels within standard limit does not completely eliminate the risk of asbestos-mediated diseases. Notably, it takes about 15-25 years after exposure for the development of asbestosis and related malignancies. Asbestosis is an irreversible disease, which can further advance even after the cessation of asbestos exposure.

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