

Workplace atmospheric asbestos levels in different plants manufacturing asbestos-cement roofing sheets in India

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ABSTRACT

Environmental monitoring was conducted to determine the workplace atmospheric asbestos levels in three different plants of an asbestos-cement (AC) roofing sheet manufacturing industry located in North India. Air samples were collected for analysis of asbestos fibers at key locations in all the three plants including fiber warehouse and factory main gate in order to assess the industrial hygiene conditions prevailed in work zone of this factory. A total of 24 samples were collected and analyzed by phase-contrast and polarized-light microscopy. Observations on asbestos fibers collected on membrane filters suggest that they are chrysotile asbestos and their average concentrations ranged between 0.036–0.148 fiber per cubic centimeter (f/cc) (mean 0.075 ± 0.034 f/cc). These fiber counts are less than the existing Indian Standard (0.5 f/cc). However, fiber counts in the ingredient mixing locations of all the plants of AC factory are higher than the proposed Indian Standard (0.1 f/cc). Further improvement in the pollution mitigation technology is highly desirable in view of the carcinogenic nature of asbestos that persists for very long time in the biological systems as well as in the environment.

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1. Introduction

Asbestos is a naturally occurring hydrated mineral silicate that crystallizes in fibrous form (Mossman et al., 1990). There are two groups of asbestos: serpentine and amphibole. Serpentine includes only chrysotile while amphibole contains amosite, actinolite, anthophyllite, crocidolite and tremolite (ATSDR, 2001). Both groups have different fibrous structures as serpentine is curly and pliable in morphology (Shukla et al., 2003), characterized by a typical scrolled structure of the layers with (4–6) hardness (Wild, 2006) whereas amphiboles are straight and rod-like. In India, chrysotile asbestos is mostly used in the manufacture of asbestos-cement (AC) sheets and AC pipes followed by jointing, brake linings, brake shoes, and clutch facings, fireproof suits. Asbestos is however used in over 5 000 products worldwide due to its unique properties such as high durability, tensile strength, resistance to chemicals and fire (National Cancer Institute, 2009). Currently, manufacturing of chrysotile-based products is carried out in more than 100 countries and the annual production is about 27 to 30 million tons. In India, about 100 000 tons of chrysotile is consumed every year mostly imported from Canada, Brazil, Kazakhstan, Russia, and South Africa (Allen, 2005). Asbestos fibers remain suspended in the air due to their lightweight and small size. The emission may occur from sources other than AC plant operation such as handling and shifting of the asbestos material, cutting or machining of end products, and disposal of wastes. Asbestos-based

products are, thereby manufactured mostly in wet process in order to reduce the possibility of fiber emissions that may also depend on the fiber control measures and technology (WHO, 1998). Issues related to asbestos induced disorders caught more attention worldwide in last couple of decades. The potential health risk of asbestos is well documented. Due to its toxic and carcinogenic nature, use of asbestos fibers has been restricted in developed countries, while it is increasing in the developing countries. All types of asbestos (serpentine and amphiboles) are established to induce malignancy particularly upon its inhalation. Extensive experimental and epidemiological data available globally clearly suggest that chronic exposure of asbestos may lead lung toxicity mediated through reactive oxygen species, progressive pulmonary fibrosis (asbestosis), pleural diseases (pleural plaques and effusion), mesothelioma and lung cancer (Ahmad et al., 1995; Arif et al., 1996; Mossman et al., 1996; Arif et al., 1997; Kamp and Weitzman, 1997; Magnani et al., 1998; Kamp and Weitzman, 1999). The profiles of asbestos exposure to workers usually varies with the concentration of fibers, duration of the exposure, workers inhalation rate (inhalation rate is higher for workers doing manual labor), meteorological and/or weather conditions. About 33 large-scale asbestos-based manufacturing plants (17 asbestos-cement product manufacturing plants and 16 other types of asbestos product plants) exist in India (Ansari et al., 2007a; Ansari et al., 2007b). Industrialization and urbanization in India has enhanced the demand of asbestos and its products resulting likelihood of

further occupational and environmental pollution. It is predicted that consumption of asbestos will yearly increase at the rate of 5 to 10% in India. Recently we also showed that asbestos emission from stacks of AC sheet manufacturing industry contaminate its surrounding area which can potentially affect the flora and fauna (Musthapa et al., 2003; Trivedi et al., 2004; Trivedi et al., 2007). In the present study, asbestos fiber counts were monitored at key occupational locations in different manufacturing plants of a factory in India, in order to assess the safety of occupational as well as atmospheric environment.

2. Materials and Methods

2.1. Studied factory and its process

This AC roofing sheets manufacturing factory is operating since 1974 in India. The annual production capacity is about 108 000 metric tons of chrysotile AC corrugated sheets and AC molded accessories. The factory is equipped with pollution control devices such as vacuum cleaner, dust collector and automatic bag opening machine in the plant manufacturing AC (asbestos cement) sheets. Automatic bag opening and wet processing are known to reduce rate of emission of asbestos fibers into the work environment. All the plants were well ventilated having exhaust fans. The workers were using nose masks, hand gloves and helmets.

2.2. Manufacturing process

In the manufacturing of AC sheets, asbestos fibers (chrysotile) from impermeable bags are taken out and milled in wet mode in the edge runner mills. Milled asbestos is then fed into a "hydro opener" and then pumped to the mixer vessels. The mill capacity is 0.5 metric tons per hour. The cement is conveyed through bucket elevator to the mixer vessel after the fly ash mixed with water is pumped to the mixer vessel. The slurry consisting of above key raw materials (required quantity/proportion) is fed through an agitator. Care is taken that the mixture is homogeneous in nature and

sieved by the sieve cylinder. As the cylinder rotates, the slurry flows through the screen on synthetic belts leaving an even film of stock deposited on its surface and then to a sheet-forming drum. When the "green" (soft and pliable) AC sheets have attained the required thickness, they are removed from the drums and cut into the required size and corrugated in a continuous process. Green corrugated AC sheets are stacked on bogies and placed in heating chamber (temp. 42–45°C) for 12–14 hours in summer and 22–24 hours in winter. The hardened AC sheets are further cured for about 21–30 days by water sprinkling to provide the optimum strength. A brief scheme of manufacturing process is shown in Figure 1.

2.3. Air sample collection and analysis of asbestos fibers

In order to assess the work place atmospheric asbestos levels of the AC factory, air samples from different plants were collected and analyzed for asbestos. These locations were at the factory gate, chrysotile asbestos warehouse, ingredient mixing, and AC sheet formation areas of three different plants. Air samples were collected in triplicate using a low-volume vacuum pump (Model, XX5600002) attached to a monitor cowl (Model, MAWP025AC, Millipore Corporation, Bedford, MA, USA), for a period of 60 min at a flow rate of 1 L/min. The collected samples were stored in upward position in sealed boxes and brought to the laboratory for further analysis. The membrane filters were made transparent by mounting in immersion oil. The mounted samples were analyzed for the presence of asbestos fibers at a magnification of 400X and asbestos fibers with dimension $>5 \mu\text{m}$ length, $<3 \mu\text{m}$ diameter, and length to diameter ratio 3:1 were considered for counting. A minimum of hundred fields were screened for counting of fibers using phase contrast and polarized light microscope (Laborlux S of M/s Leica, Germany) attached with reticules, micrometers, graticules (Pyser-Sgi Ltd, UK). The concentration of asbestos fibers in air samples is expressed as number of fiber per cubic centimeter of air (f/cc) following the method by Bureau of Indian Standards (BIS) (1986).

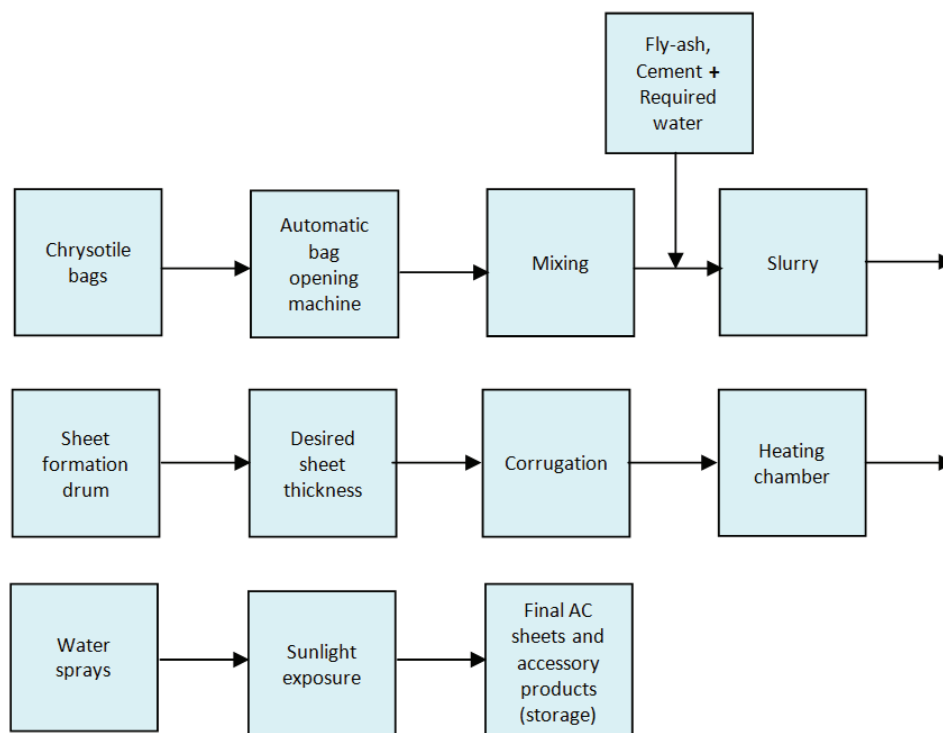


Figure 1. Manufacturing process.

3. Results and Discussion

Asbestos monitoring was done throughout the work environment acknowledging the critical locations in three different plants of the same factory. These locations were at the asbestos fiber warehouse, ingredients mixing, AC sheet production, and the factory main gate. Air concentrations of asbestos fibers at these locations for different plants are shown in Table 1. Asbestos fibers were characterized as chrysotile fibers with the phase-contrast and polarized-light microscopy.

There are only one main gate and one warehouse in the factory that measured asbestos concentrations ranged from 0.039 to 0.052 f/cc (mean 0.046 ± 0.006 , lower and upper 95% CI 0.295 and 0.0618) and 0.036 to 0.051 f/cc (mean 0.042 ± 0.007 f/cc, lower and upper 95% CI 0.0236 and 0.0616), respectively. In plant-I, the fiber concentrations were 0.099 to 0.119 f/cc (mean 0.109 ± 0.010 f/cc, lower and upper 95% CI 0.083 and 0.133) in ingredient mixing and 0.048 to 0.072 f/cc (mean 0.061 ± 0.012 , lower and upper 95% CI 0.304 and 0.090) in AC sheets formation. In plant-II, the fiber concentrations were 0.096 to 0.148 f/cc (mean 0.121 ± 0.026 f/cc, lower and upper 95% CI 0.056 and 0.186) in ingredient mixing and 0.048 to 0.060 f/cc (mean 0.053 ± 0.006 f/cc, lower and upper 95% CI 0.386 and 0.068) in AC sheet formation. In plant-III, the fiber concentrations were 0.085 to 0.129 f/cc (mean 0.112 ± 0.023 f/cc, lower and upper 95% CI 0.535 and 0.163) in ingredient mixing and, 0.042 to 0.082 f/cc (mean 0.059 ± 0.020 f/cc, lower and upper 95% CI 0.008 and 0.110) in AC sheet formation. Among the locations in manufacturing area, the highest concentrations were found at the ingredient mixing sites of all the plants, which might be largely due to partly dry and partly wet process. Locations in AC sheet formations in all the plants showed the lowest concentrations, probably due to completely wet manufacturing process. The fiber concentrations at different locations in all the three plants are less than the present Indian Standard (0.5 f/cc). As compared to asbestos standard (0.1 f/cc), prescribed by many international agencies like ACGIH, NIOSH, OSHA (ACGIH, 1998; OSHA, 1998; NIOSH, 1999). Workplace atmospheric asbestos levels were slightly higher in ingredient mixing locations of all the plants. However, Central Pollution Control Board has requested Ministry of Environment and Forests (Government of India) to collaborate with the Ministry of Labor to reduce the workplace standard for asbestos to 0.1 f/cc. There are

only limited published reports on the fiber counts in AC roofing sheet industries (Ansari et al., 2007c). Recently, workplace atmospheric asbestos levels were reported to be ranging from 0.057 to 0.079 f/cc in an AC factory (Ansari et al., 2007c).

The fibers were characterized and found to be chrysotile fibers by using phase-contrast microscopy following the method by BIS (1986). Asbestos fibers remain suspended in the ambient air for a long time due to their small sizes and lightweight. The presence of fibers in the surrounding environment may be due to emissions of asbestos fibers during improper handling and processing, and also may be from transportation and waste disposal (Magnani et al., 1998).

Due to hazardous nature of asbestos, its emission by asbestos-cement products manufacturing industries particularly in the occupational environment continues to be a serious concern. Asbestos exposure also occurs in the population living near the asbestos sources (Magnani et al., 1998). Asbestos exposure causes various types of lung disorders such as progressive pulmonary fibrosis (asbestosis), pleural diseases (pleural plaques and effusion), mesothelioma and lung cancer (Mossman et al., 1990; Mossman et al., 1996; Kamp and Weitzman, 1997; Hauptman et al., 2002; Marin and Clavera, 2005; Maynard et al., 2006). Notably, adverse health effects are directly correlated with the concentration and the period of asbestos exposure at occupational sites. Occupational exposure through inhalation is the most hazardous to industrial workers (Mossman et al., 1990; Marin and Clavera, 2005). The latency period between the time of initial exposure to asbestos and the development of diseases may be 20–40 years (Vaino and Boffetta, 1994). Airborne asbestos fibers at the occupational environments are considered critical in the assessment of industrial hygiene status of asbestos-based industries (Mossman et al., 1996; Magnani et al., 1998). In India, about 100 000 people are estimated to be exposed to asbestos at different workplaces (Joshi and Gupta, 2003). Considering the commercial scope of AC products for industrial and agricultural growth required in India, the solution lies in safest use of asbestos by ensuring the implementation of regulatory guidelines in the strictest possible manner that is quite achievable through health awareness program for the factory workers as well as the owners.

Table 1. Atmospheric levels of asbestos in workplace environment of asbestos-cement factory

Site No.	Locations	No. of Area Samples	Concentration Range	Occupational Environment		
				Concentration (f/cc) (Mean \pm S.E)	95% CI (Lower)	95% CI (Upper)
1	Industry gate	3	0.039 – 0.052	0.046 ± 0.006	0.295	0.061
2	Asbestos warehouse	3	0.036 – 0.051	0.042 ± 0.007	0.023	0.061
3	Plant I					
	Ingredient mixing	3	0.099 – 0.119	0.109 ± 0.010	0.083	0.133
	Asbestos – cement sheet formation	3	0.048 – 0.072	0.061 ± 0.012	0.304	0.090
4	Plant II					
	Ingredient mixing	3	0.096 – 0.148	0.121 ± 0.026	0.056	0.186
	Asbestos – cement sheet formation	3	0.048 – 0.060	0.053 ± 0.006	0.386	0.068
5	Plant III					
	Ingredient mixing	3	0.085 – 0.129	0.112 ± 0.023	0.535	0.163
	Asbestos – cement sheet formation	3	0.042 – 0.082	0.059 ± 0.020	0.008	0.110

4. Conclusion

Asbestos fibers invariably prevail throughout the occupational environment of the asbestos–cement sheet manufacturing factory. Although asbestos levels were lower than the prescribed Indian standards, the possibility of chronic effects on the exposed population and the environment cannot be ruled out, in view of the persistent nature of asbestos and its chronic mode of malignancy. A continuous monitoring on industrial hygiene and further improvement in pollution mitigation technology are desired in order to achieve the safest working conditions in asbestos-based industries in India. Workers were simultaneously educated about the risk of exposure and preventive measures. Management was also requested to ensure that occupational safety measures are strictly followed in the factory, through good housekeeping to protect fiber diffusion and by supplying personal protection equipments to the workers.

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References

- ACGIH, 1998. Documentation of the threshold limit values and biological exposure indices. 6th Ed. American Conference of Governmental Industrial Hygienists. Cincinnati, OH.
- Ahmad, I., Krishnamurthi, K., Arif, J.M., Ashquin, M., Mahmood, N., Athar, M., Rahman, Q., 1995. Augmentation of chrysotile-induced oxidative stress by BHA in mice lungs. *Food and Chemical Toxicology* 33, 209–215.
- Allen, L.K., 2005. Asbestos and mesothelioma: Worldwide trends. *Lung Cancer* 49 (S1), S3–S8.
- Ansari, F.A., Ashquin, M., Ahmad, I., 2007a. Asbestos fiber counts in work zone area of an asbestos cement factory. *Journal of Scientific and Industrial Research* 66, 935–937.
- Ansari, F.A., Bihari, V., Rastogi, S.K., Ashquin, M., Ahmad, I., 2007b. Environmental health survey in asbestos cement sheets manufacturing industry. *Indian Journal of Occupational and Environmental Medicine* 11, 15–20.
- Ansari, F.A., Ahmad, I., Ashquin, M., Yunus, M., Rahman, Q., 2007c. Monitoring and identification of airborne asbestos in unorganized sectors, India. *Chemosphere* 68, 716–723.
- Arif, J.M., Khan, S.G., Ahmad, I., Joshi, L.D., Rahman, Q., 1997. Effect of kerosene and its soot on the chrysotile-mediated toxicity to the rat alveolar macrophages. *Environmental Research* 72, 151–161.
- Arif, J.M., Ahmad, I., Rahman, Q., 1996. Chrysotile inhibits glutathione-dependent protection against the onset of lipid peroxidation in rat lung microsomes. *Pharmacology and Toxicology* 79, 205–210.
- ATSDR, 2001. Toxicological profile for asbestos. Update (Final Report). Public Health Service, U.S. Department of Health and Human Services Atlanta GA: NTIS Accessories No. PB/2001/109/01, Agency for Toxic Substances and Disease Registry, USA146.
- Bureau of Indian Standards, 1986. Method for determination of airborne asbestos fibres concentration in work environment by light microscopy (Membrane filter method), Indian Standard, New Delhi.
- Hauptman, M., Pohlabeln, H., Lubin, J.H., Jockel, K.H., Ahrens, W., Brucke-Hohfeld, I., Wichmann, H.E., 2002. The exposure time response relationship between occupational asbestos exposure and lung cancer in two German case control-studies. *American Journal of Industrial Medicine* 41, 89–97.
- Joshi, T. K., Gupta, R. K., 2003. Asbestos-related morbidity in India. *International Journal of Occupational and Environmental Health* 9, 249–253.
- Kamp, D.W., Weitzman, S.A., 1999. The molecular basis of asbestos induced lung injury. *Thorax* 54, 638–652.
- Kamp, D.W., Weitzman, S.A., 1997. Asbestosis: Clinical spectrum and pathogenic mechanisms. *Proceeding of the Society for Experimental Biology and Medicine*, 214, 12–26.
- Magnani, C., Mollo, F., Paoletti, L., Bellis, D., Bernardi, P., Betta, P., Botta, M., Falchi, M., Ivaldi, C., Pavesi, M., 1998. Asbestos lung burden and asbestosis after occupational and environmental exposure in asbestos-cement manufacturing area: A necropsy study. *Occupational and Environmental Medicine* 55, 840–846.
- Marin, M.B., Clavera, I., 2005. Asbestosis. *Anales del Sistema Sanitario de Navarra Supplement* 1, 37–44.
- Maynard, A.D., Aitken, R.J., Butz, T., Colvin, V., Donaldson, K., Oberdorster, G., Philbert, M.A., Ryan, J., Seaton, A., Stone, V., Tinkle, S.S., Tran, L., Walker, N.J., Warheit, D.B., 2006. Safe handling of nanotechnology. *Nature* 444, 267–269.
- Mossman, B.T., Kamp, D.W., Weitzman, S.A., 1996. Mechanisms of carcinogenesis and clinical features of asbestos-associated cancers. *Cancer Investigation* 14, 466–480.
- Mossman, B.T., Bignan, S., Corn, M., Seaton, A., Gee, J.B., 1990. Asbestos: scientific developments and implications for public policy. *Science* 247, 294–301.
- Musthapa, M.S., Ahmad, I., Trivedi, A.K., Rahman, Q., 2003. Asbestos contamination in biota and abiota in vicinity of asbestos – Cement factory. *Bulletin of Environmental Contamination and Toxicology* 70, 1170–1177.
- National Cancer Institute, U.S. Institute of Health, 2009. <http://www.cancer.gov/cancertopics/factsheet/Risk/asbestos>. Asbestos Exposure and Cancer Risk.
- NIOSH, 1999. Pocket guide to chemical hazards. Washington D.C.: U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health.
- OSHA, 1998. U.S. Department of Labor. Occupational Safety and Health Administration. Code of Federal Regulations. 29 CFR 1926.1101.
- Ramanathan, A.L., Subramanian, V., 2001. Present status of asbestos mining and related health problems in India - A survey. *Industrial Health* 39, 309–315.
- Shukla, R., Ramos-Nino, M., Mossman, B., 2003. Cell signaling and transcription factor activation by asbestos in lung injury and disease. *The International Journal of Biochemistry and Cell Biology* 35, 1198–1209.
- Trivedi, A.K., Ahmad, I., Musthapa, M.S., Ansari, F.A., 2007. Environmental contamination of chrysotile asbestos and its toxic effects on antioxidative system of Lemna gibba. *Archives of Environmental Contamination and Toxicology* 52, 355–362.
- Trivedi, A.K., Ahmad, I., Musthapa, M.S., Ansari, F.A., Rahman, Q., 2004. Environmental contamination of chrysotile asbestos and its toxic effects on growth and physiological and biochemical parameters of Lemna gibba. *Archives of Environmental Contamination and Toxicology* 47, 281–289.
- Vainio, H., Boffetta, P., 1994. Mechanisms of the combined effect of asbestos and smoking in the etiology of lung cancer. *Scandinavian Journal of Work Environment and Health* 20, 235–242.
- Wild, P., 2006. Lung cancer risk and talc not containing asbestiform fibres: a review of the epidemiological evidence. *Occupational and Environmental Medicine* 63, 4–9.
- World Health Organization, 1998. Chrysotile asbestos: Environmental health criteria. Geneva, Switzerland.