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HUMAN HEALTH RISK ASSESSMENT STUDIES IN ASBESTOS BASED INDUSTRIES IN INDIA



**CENTRAL POLLUTION CONTROL BOARD
(MINISTRY OF ENVIRONMENT AND FORESTS)**

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FOREWORD

The Central Pollution Control Board has published a number of documents under the Programme Objective Series (PROBES), regarding environmental issues and preventive & control measures for pollution. The present document, on the Human Health Risk Assessment Studies in Asbestos based Industries in India, is the latest such document. The Central Pollution Control Board through the Industrial Toxicology Research Centre, Lucknow, undertook the study for this document.

Asbestos is mainly used for manufacturing asbestos-cement sheets, asbestos-cement pipes, brake lining, clutch lining, asbestos yarn & ropes, gaskets & seals etc. Organised asbestos industrial units are mostly using imported chrysotile variety of asbestos. The indigenous asbestos is mostly used by the unorganized sector. This report provides detailed information on human risk of asbestos exposure and its health effects. The study includes asbestos monitoring at work environment, characterization and toxicity of indigenous asbestos, occupational and personal histories of workers, their clinical examinations, lung function tests and chest radiological examinations. It appears from the present investigation that unorganized units have poor industrial hygiene conditions. The report also recommends various preventive measures to reduce the risk of workers exposed to asbestos.

I would like to express our sincere appreciation for the work done by the team of Industrial Toxicology Research Centre, Lucknow. The suggestions made by the Project Advisory Committee members were valuable. The efforts made by my colleagues Sh. P.K. Gupta, Environmental Engineer and Sh. J.S. Kamyotra, Additional Director for coordinating the Study and for finalizing the Report under the guidance of Dr. B. Sengupta, Member Secretary, CPCB, deserve appreciation.

We in CPCB hope that this Study will be useful to the Asbestos manufacturing units, regulatory agencies, research organizations and to all those interested in pollution control.

27th May 2008

(J. M. Mauskar)

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CHAPTER 1.0

INTRODUCTION

1.1 Background

According to Pooley (1972), Piney was the first author to use the word “asbestos” referring to a fibrous mineral of Greek derivation which means “inextinguishable” or “unquenchable”. The word “asbestos” is defined in Webster’s Medical Dictionary as “a mineral that readily separates into long flexible fibres suitable for use as non-combustible, non-conducting, chemically resistant material”.

Asbestos is a naturally occurring hydrated mineral silicate that crystallizes in fibrous form (Mossman et al., 1990b). Mineralogically asbestos can be classified into two major groups; the Serpentine, which includes the most abundant variety of asbestos i.e. Chrysotile and the Amphibole which includes Actinolite, Amosite, Anthophyllite, Crocidolite and Tremolite (Mossman et al., 1996, ATSDR, 2001). Both groups have different physico-chemical nature. Chrysotile is curly and stranded structure whereas amphiboles are straight and rod like structures (ATSDR). Amphiboles are generally more brittle and appear to be dustier and more fibrogenic than chrysotile (Mossman et al., 1990 ; Mossman and Gee, 1989).

Asbestos fibres bear unique properties of a high tensile strength, resistance to heat and many chemicals without having any detectable odor. Mineralogists some times refer that the minerals crystallize into bundles of thousands of flexible fibrils that look like organic fibres. Terms that are sometimes used to describe asbestos or similar minerals include fiber, fibrous, asbestiform and acicular. The term fibrous is used to describe a crystallization habit in which the fibres have a high tensile strength and flexibility than crystals in other parts of the same mineral; asbestiform is generally synonymous with fibrous or sometimes it means “like asbestos”; and acicular” refer to a crystal that has a needle-like form.

Even though the use of asbestos was known to medieval India, it was commercially exploited only since the beginning of this century. Asbestos is attractive in a broad variety of industrial applications because of its resistance to heat and chemicals, high tensile strength, and lower cost compared to man-made minerals. At the peak of its demand, about 3,000 applications or types of products were of asbestos-based (Ramanathan and Subramaniam, 2001). Asbestos is used for the manufacture of a variety of asbestos-based products mainly as asbestos-cement (AC) sheets, AC pipes, brake shoes, brake linings, clothes and ropes. AC industry is by far the largest user of asbestos fibre worldwide accounting for about 85% of all uses. Asbestos is also incorporated into cement construction materials (roofing, shingles, and cement pipes), friction materials (brake linings and clutch pads), jointing and gaskets, asphalt coats and sealants, and other similar products. As a result of these applications, an estimated 20% buildings including hospitals, schools and other

public and private structures contain asbestos containing materials (ACM). Asbestos in building does not spontaneously releases fibres, but physical damage to ACM by decay, renovation or demolition can cause release of airborne fibres.

Asbestos in air at work environment is a major cause of adverse effects on health of industrial workers. Industrialization and modernization with recent developments enhanced the demand and consumption of asbestos thus increasing the risk of exposure to asbestos.

1.2 Classification of Asbestos

1.2.1 Serpentine Group

1.2.1.1 Chrysotile

Chrysotile, the only representative of serpentine, also called as white asbestos accounts for over 90% of the world's production of asbestos. Chrysotile is a sheet silicate, composed of planar-like silica tetrahedral with an overlying layer of brucite. The silica-brucite sheets are slightly warped because of structural mismatch, resulting in the propagation of a rolled scroll that forms a long hollow tube. These tubes form the composite fiber bundle of chrysotile. Some trace oxides are always present as a result of contamination during the formation of the mineral in the host rock. Chrysotile asbestos is composed of soft, silky, long, flexible, pliable, and curly and they tend to form bundles that are often curvilinear with splayed ends. Hydrogen bonding and/or extra fibril solid matter holds such bundles together. The individual fibres take the shape of spirally winded tubes. It is the cylindrical structure of fibre responsible for its fibrous natures. Chrysotile fibres naturally occur in length varying from 1 to 20 mm, with occasional specimens as long as 100 mm. In India, chrysotile fibres occur as thin veins of 10 mm to 100 mm thickness in serpentine rocks (Ramanathan and Subramaniam, 2001). In comparison to amphiboles, chrysotile is less resistant to heat.

1.2.2 Amphibole Group

The amphibole minerals are double chain of silica tetrahedral, cross-linked with bridging cations. The hollow central core typical for chrysotile is lacking. Amphibole fibres are generally more brittle and appear to be dustier and occur as pocket deposit in ultramafic rock.

1.2.2.1 Crocidolite

Typical crocidolite fibres bundles early disperse into fibres that are shorter and thinner than that of other amphibole asbestos fibres. It is also called as 'blue asbestos' because of its colour and possess fair spin ability.

1.2.2.2 Amosite

Amosite varies in color from yellow to gray to black brown. Tensile strength is much less than that of chrysotile or crocidolite, and it has only fair spin ability and poor resistance to heat. It is also called as 'brown asbestos'.

1.2.2.3 Anthophyllite

Anthophyllite fibres are yellowish brown, grayish or white in color with poor spin ability and tensile strength but highly resistant to acids and heat. Anthophyllite asbestos is relatively rare, fibrous, orthorhombic, magnesium, iron amphibole, which occasionally occurs as a contaminant in talc deposits.

1.2.2.4 Tremolite

Tremolite varies in color from gray-white, greenish yellow or bluish which has a slickly luster and generally a harsh texture. These are common as contaminants of other asbestos deposits. Tremolite fibres are quite resistant to heat and acids but have poor flexibility and spin ability.

1.2.2.5 Actinolite

Actinolite fibres have greenish color with a silky luster, a harsh texture and quite hard.

1.3 Chemical Structure

Serpentine

Chrysotile $Mg_3Si_2O_5(OH)_4$

Amphibole

Actinolite $(Ca, Fe)_{2Mg_5Si_8O_{22}}(OH)_2$

Amosite $Fe_2Fe_5Si_8O_{22}(OH)_2$

Anthophyllite $Mg_2Mg_5Si_8O_{22}(OH)_2$

Crocidolite $Na_2Fe^{2+}_3Fe^{3+}_2Si_8O_{22}(OH)_2$

Tremolite $Ca_2Mg_5Si_8O_{22}(OH)_2$

1.4 Asbestos Exposure

Asbestos fibres can enter the air, water and soil from the weathering of natural deposits and the wearing down of manufactured asbestos products. People are most likely to be exposed to asbestos through inhalation of airborne fibres. Asbestos fibres can be broken down in the environment but will remain virtually unchanged over long period. These fibres can come from naturally occurring sources of asbestos i.e., asbestos bearing rocks or from the wearing down or disturbance of manufactured products including insulation, automotive, brakes and clutches, ceiling and floor tiles, dry wall, roofing materials and AC sheets as mentioned above. Asbestos is much more likely to be released to the atmosphere when asbestos deposits are disturbed as in mining operations. Other anthropogenic sources of asbestos emissions besides mining are the crushing, screening, and milling of the ores, the processing of asbestos into asbestos-based products, the use of asbestos-containing materials. The transport and disposal of asbestos containing wastes also add to the exposure of asbestos into the environment.

When mineral fibres are inhaled, many are deposited on the epithelial surface of the respiratory tree. Entry and deposition of the fibres depend upon the type of fibres and more importantly fibre size (length and diameter) which are believed to be important determinants of the health risk posed by asbestos. The number of fibres that are deposited and the location within the airway where deposition occurs is a function of the aerodynamic properties of the fibres. For typical fibres of chrysotile, amosite and crocidolite, about 30-40% of all the fibres in inhaled air are retained with most of these (about 60%) being deposited in the upper air ways (nose, throat, trachea) (Morgan et al., 1977). The fibres in the upper airway consist mainly of relatively thick fibres (greater than 3µm) with thinner fibres being carried deeper into the airways (Timbrell, 1982). Most fibres deposited into the airways are removed from the lung by mucociliary transport or by alveolar macrophages (AM) but a small fraction remains in the lung for long periods (Jones et al., 1988). In addition, some fibres pass from the lung to the pleura, although the precise mechanism of transport is not known (Hillerdal, 1980; Rudd et al., 1980). Those fibres that enter the lymph are presumably able to reach other organs of the body.

Epidemiological surveys and experimental studies established that asbestos is a carcinogen as well as co-carcinogen (Mossman et al, 1996; IARC 1987). Chronic inhalation of airborne pollutants may result in the fibrotic lung disease, and there is evidence that the occurrence of chronically activated alveolar macrophages (AM) linked with the process (LaSalle et al., 1990). Deposition of these pollutants i.e., fibres/particles in the lung is followed by a sequence of events, which starts with change in the free cell population, which includes AM and polynuclear inflammatory cells via their influx (Spurzen et al., 1987).

There have always been debates about the nature of interaction between multiple environmental pollutants in causing diseases to human. One of the most discussed agents is asbestos, a group of fibrous mineral silicates and a well-established carcinogen and co-carcinogen (WHO, 1986). Predisposing factors like food preservatives, exposure to cigarette smoke, kerosene soot and bio-fuels at indoor levels would accelerate the disease processes (Ahmad et al., 1994, Yano et al, 1993, Kamp et al., 1998, Wang et al., 2000). Cigarette smoke alone has been shown to cause lung cancer but the risk of lung cancer increases substantially due to cigarette smoke in conjugation with exposure to asbestos.

The development of the asbestos industries has always been linked with the recognition of health risk involved. Owing to the growing activities in mining, grinding and manufacturing of asbestos-products, the risk of health hazards has also received wide attention world over.

1.5 Asbestos – Mediated Toxicity and Diseases

1.5.1 Inflammation

The sequence of events in the lung, following deposition of fibres includes modulation in the free cell population, primarily characterized by an increase in AM

and polymorpho nuclear inflammatory cells (Brody et al., 1981; Cohen, 1981; Warheit et al., 1984; Spurzen et al., 1987). Further, a change in the composition of the lung lining fluids has also been reported (Last and Reiser, 1984). The inflammatory response to these fibres have been reported to stimulate the release of a variety of inflammatory cell mediators and growth factors which are reported to play an important role in the fibrogenesis of the lung (Cohen, 1981).

1.5.2 Mesothelioma

Mesothelioma was recognized as early as in the late 1700's. Approximately 80% of mesotheliomas occur in men exposed to mineral fibres at workplaces and sometimes in their family members or in persons who lives near asbestos sources. Mesothelioma may develop in pleural and peritoneal cavity of the lung.

1.5.3 Peritoneal Mesothelioma

Peritoneal mesothelioma involves the abdominal cavity, infiltrating the liver and spleen and the bowels pain is the most common presenting complaints. In addition, fluid accumulation in the abdominal cavities (ascots), the abdomen appears enlarged, the patient experience nausea, vomiting, swelling of their feet, fever and difficulty in moving their bowels.

A layer of specialized cells are called mesothelial cells which line the chest cavity and the cavity around the heart. These cells also cover to outer surface of most internal organs. The tissue formed by these cells is called mesothelium. Benign mesothelioma is rare form of peritoneal mesothelioma while malignant mesotheliomas are divided into threes types. About 50-70% of mesothelioma is the epitheliod type. The other two types are the sarcomata types (7-20%) and the mixed or biphasic type (20-35%). Approximately 80% of diffuse malignant mesotheliomas occur in men exposed to mineral fibres in the workplace and sometimes in their family members or in persons who lives near asbestos mines. Diffuse malignant mesothelioma is a fatal tumor arising from mesothelial cells or underlying mesenchymal cells in the pleura, pericardium and peritoneum. The time between diagnosis and initial exposure to mineral fibres commonly exceeds 30 years. Most people with mesothelioma have symptoms for only two to three months before they are diagnosed.

1.5.4 Pleural Plaques

The pleuron is a set of thin membrane (about one cell thick) that lines the chest cavity. Pleura are co-important as provide lubrication, friction free surface to lung for easily expand and contract against. After an exposure of asbestos for minimum 10 years, pleural changes may begin to appear and these changes may be some times called as pleural thickening, pleural calcification and more commonly pleural plaques. Hyaline plaques of the parietal pleura occur in association with exposure to all types of asbestos. The majority occurs after 20 years or more of the exposure.

1.5.5 Asbestosis

This is a typical asbestos-related disease. Asbestos fibres when inhaled and reach in the lung start to damage the lung cells and result asbestosis (formation of scar tissue in the lung), and /or lung cancer. The risk of lung cancer among people exposed to asbestos is increased by 7 times compared with the general population. Asbestosis is an interstitial pulmonary fibrosis, which reduces the lung capacity to deliver the oxygen in proper way to the whole body because the lung tissue loses its ability to function. It is characterized by the airway obstruction and air trapping, reducing vital capacity (Kilburn, 2000). This disease has relatively long latency period of about 40 years.

Clinically, asbestosis is very similar to interstitial pulmonary fibrosis (IPF). Most patients with well-established asbestosis characterized with shortness of breath, dry cough, and physical examination typically reveals dry rales at the base on inspiration. The usual function changes in the fully developed case are a restrictive defect and decreased diffusing capacity (Kilburn, 2000).

1.5.6 Bronchogenic Carcinoma

A number of occupational studies have demonstrated an association between exposure to various types of mineral fibres and bronchogenic carcinoma (McDonald et al., 1987). Bronchogenic carcinoma is tumor, arising in tracheobronchial epithelial or alveolar epithelial cells. The average latency period of the disease i.e. the diagnosis of the disease from the time of first exposure to asbestos ranges from 20 to 30 years. The degree of association varies with the type of mineral fibre, morphology, concentration, exposure regimen, and other predisposing factors like smoking habits or the presence of certain other chemicals, but there is usually a dose- response relation (fiber per cubic centimeter of air times the number of years of exposure). Lung tumor is rare among the mineral fibre workers who do not smoke; although early epidemiological studies indicated that the effect of mineral fibres and smoking combines in a multiple fashion to produce lung cancers (Saracci, 1977).

1.6 Organized and Unorganized Sectors

The labor forces in developing economy consist of two sectors, the unorganized and organized sectors. The unorganized sector covers most of the rural labors and a substantial part of urban labor. It includes activities carried out by small and family enterprises, partly or wholly with family labor, and in which wages paid labor is largely unorganized due to such constrains as the casual and seasonal nature of employment and scattered location of enterprises. This sector is marketed by low income, unstable and irregular employment, and lack of protection either from legislation or trade unions. Apart from those who are poor because they are unemployed, the people from the unorganized sector can be referred to as the "working poor" (Rajhans1993). Unorganized sector can also be defined as the part of economy where earning one's livelihood is precarious. Employment relationship wages and other working conditions are de facto not protected or regulated.

The large manufacturing firms in the organized sector operate in markets where prices are controlled by a few sellers, which are protected from foreign competition by high tariff, and which sell products mainly to middle and upper income groups. Some differences in the unorganized and organized sectors are described below. The unorganized sector consists of a large number of small producers and these producers are operating on narrow margins in highly competitive markets. The products are sold to the lower income groups. Secondly, the organized sector has great access to cheap credit provided by various financial institutions, while the unorganized sector often depends upon moneylenders who charge a high rate of interest. Thirdly, the organized sector uses capital-intensive imported technology, while the unorganized sector uses only labor intensive and indigenous technology. Lastly, the organized sector is protected by various types of labor legislations and is backed often by strong unions. The unorganized sector on the other hand is either not covered by labor legislation at all or is so scattered that the implementation of the legislation is very inadequate and ineffective. There is hardly any union in this sector to act as watchdogs. In the organized sector, it was pointed out that it consists almost wholly of wage and salary earners. The unorganized sector, however, is making up two distinct groups, the wage earners and the self-employed. According to the 1981 census, out of the total labor force of 222.5 million, 125.2 million (56.2%) are self-employed. Out of the remaining wages earners, 22.8 million are in the organized sector and 74.5 million in the unorganized. Thus the number of wage earners in the unorganized sector is almost three and half times of the number in the other sector. This is so simply because 57.1% of the wage earners in the unorganized sector are agricultural labor and the rest are non-agricultural labor.

1.7 Geology, Mining and Processing of Asbestos in Rajasthan

The processing of asbestos bearing rocks obtained from mines involves simple crushing and grinding. For crushing jaw crusher are used. The ore with higher percentage of asbestos contents yields powder, which is fluffy in nature (coarse grains) and light in weight whereas the ore with less asbestos content changes to heavy fine powder. No other operation is involved in processing. In majority of grinding mills (asbestos mills), a pulveriser is used in a close circuit hammer mill consisting of air cyclone.

1.7.1 Salient Features of Beawer Belt

The asbestos mineralization of Ajmer- Beawer belt (about 50 km long & 20 km wide) lies about 40 km west of Ajmer. The deposits are located within central and northern part of the belt. The deposits are located near villages Asan, Naikala, Sendra, Ramgarh, Kotra, Konotia, Manpura, Macarena, Mangliawas and Nad-Arjunpura. The Ajmer-Beawer asbestos belt falls within the met sedimentary rocks of Delhi Super group (Alwar and Ajabgarh Groups) flanked on eastern side by met sedimentary rocks of Aravalli Super group. Unlikely, Jharol belt and Deogarh belt belongs to the districts Udaipur and Rajasmand. Mineralogically, the ore principally consists of anthophyllite-tremolite variety of amphibole group with actinolite. In asbestos belts normally 3 types of ores are observed. One is stick-type almost pure asbestos found as thin vein lets within ultra basics and other one is a rock mass consisting of

radiating anthophyllite-tremolite needles, iron oxides, magnesite, talc and chlorite etc. The recovery of the stick types is about 2% to 5% only wherever it is found while the recovery of the other type is about 70% to 80%. All the deposits in these areas are being processed by open cast method. Because of the soft nature of the country rock as well as of ores bodies, no drilling and blasting is carried out except occasionally in the harder portions of the country rocks (wall rocks). Opencast method is similar to that used for any other minerals. The wall rocks and the ore are frequently broken manually by picks, crowbars, chisels and hammers. The broken material is then taken in pans on head load for transportation to the stack in yards. The gradation of ore is done by hand sorting of the excavated material at the stacking yard. The workable deposits of Beawer area are located at a varying distance of 20 km to 60 km from processing plants/consuming locations like Beawer and Ajmer. The transportation cost thus is moderate.

1.7.2 Salient Features of Deogarh Belt

Amphibole asbestos deposits of Deogarh region occur sporadically in an area extending from east of Charbhujia to northeast of Deogarh for a strike length of about 50 km. The deposits are mainly located from south-west to north-east near the villages Lalji-ka-khera, Roopji-ka-khera, Kalaguman, Tekhi, Kunwathal etc. Mineralogically, the asbestos ore here consists of tremolite and anthophyllite alongwith actinolite with talc, chlorite, magnesite, and iron oxides. Physically, the ore is a white to greenish-white, loose soft fibrous looking mass. Chemically, the presence of the alumina and calcium is more here than Jharol and Beawer belts which is indicative of more of tremolite-actinolite than anthophyllite in the ore. Most of the ore bodies consist of randomly distributed aggregates of asbestos (tremolite-anthophyllite), magnesite, greenish to grayish chlorite and talc, actinolite and iron oxides. Such ore bodies are quite soft and loose in nature due to friable and soft nature of the constituents. Such type of ore is locally known as “jhuri” and forms about 90% of the ore of grade of Deogarh belt. This is typical of this region. Second type of ore which is locally found west of Gomti, i.e., east of Charbhujia, where harder “Compact Type” of ore, with randomly disseminated needles of asbestos occur within the matrix of the gangue miners, which imparts a fibrous look. Recovery of such type of ore in the area is around 15-20%. The third type of ore is the “Stick Type”, bundles of nearly pure asbestos found either at the wall rock contact or in fracture and slip planes of the ore bodies. The production recovery of this type of ore is 2-5%. All the ore deposits in the region are worked by open cast methods only. The method is the same as described in the Beawer belt. Here also because of the soft nature of the country rock as well as that of ore bodies, drilling and blasting is scarcely employed. The most conspicuous features of the Deogarh region are that all the workable deposits fall within a distance of 10-30 km from the processing plants. Thus the transportation cost is the lowest in comparison to Jharol and Beawer regions. The main source of dust generation in plants involves: dislodging/digging of Asbestos Bearing Rock (ABR) with crow bar or pick axe, breaking of ABR by sludge hammer, loading of broken ABR in iron pan, transportation of ABR from pit to the stacking place through carrier by over head on iron pan, breaking ABR to smaller size by hammer, miscellaneous operations like Crane and Wheel loading operations.

CHAPTER 2.0

FIELD STUDIES

2.1 Unorganized asbestos units

The asbestos grinding units at Deogarh selected for in depth studies under unorganized sector are given below:

1. M/s B. K. Grindings Pvt. Ltd.,
2. M/s Kanchan Minerals Pvt. Ltd.,
3. M/s Maharaja Asbestos Grinding Mills Pvt. Ltd.
4. M/s Osawal Minerals Trading Corporation,

The asbestos grinding units at Beawar selected for in depth studies under unorganized sector are given below:

1. M/s Cenera Minerals Pvt. Ltd.,
2. M/s Guru Asbestos Pvt. Ltd.,
3. M/s Gajanand Cement Asbestos Products Pvt. Ltd.,
4. M/s Kamla Grinding Mills Pvt. Ltd.,
5. M/s Super Minerals Pvt. Ltd.,
6. M/s Swastic Udyog Pvt. Ltd.

The details of these asbestos grinding units located at Deogarh and Beawar are given in Table A and B respectively.

Process

Unorganized asbestos milling units grind the raw asbestos collected from near by locally available asbestos sources. Unorganized asbestos-based product manufacturing units usually mix the grinded asbestos with Portland cement and ratio for mixing depends on the quality of the product required in the trade market. After mixing of cement and asbestos powder, water is added to make a paste like slurry as per requirement. The slurry is used for manufacturing a variety of asbestos-based products by the semiautomatic machines or manually. These asbestos-cement (AC) products are kept in sunlight to dry

for 5-10 days then transferred to water tank for 15 to 20 days to become strengthened. Various steps of manufacturing asbestos-based products are sketched in flow chart-1.

2.2 Organized Asbestos Industries

The organized sector industries were mainly involved in the manufacturing of asbestos-based products as AC roofing sheets, ropes, cloth, brake shoes, clutch plates, brake lining etc. These industries were M/s UP Asbestos Pvt. Ltd., Lucknow (UPAL-I); M/s UP Asbestos Pvt. Ltd., Nagpur (UPAL-II); M/s Allied Nippon Pvt. Ltd., M/s Champion Seals Pvt. Ltd., Boisar (A); M/s Mechanical Packing Industries Pvt. Ltd.,(B); M/s Mechanical Packing Industries Pvt. Ltd., Dahisar (C); M/s Hindustan Composite Pvt. Ltd., Aurangabad (D) and M/s Hindustan Composite Pvt. Ltd., Ghatkopar (E).

2.2.1 M/s UP Asbestos Pvt. Ltd.,(I)

M/s UP Asbestos Pvt. Ltd., (UPAL-I) is a medium scale factory, and located in industrial area of Mohanlalganj, Raebareilly Road, Lucknow (U.P.). The factory plant is in operation since 1974 as per record provided by the factory. The total production capacity of industrial products is 108000 metric tonnes per annum. The main products are AC roofing sheets and moulded products. The total workers in the factory including office are 200. The study comprised of 104 workers including staff in this factory. Various steps of manufacturing process are sketched in flow chart-2:

Process

Asbestos fibres from impermeable bags is taken out and milled in the wet mode in edge runner mills. Milled fibre then fed into a "hydro opener" and then pumped to the mixer vessels. The mill capacity is 0.5 metric tonnes per hour. There are two such types of mills. Now the cement is conveyed through bucket elevator to the mixer vessel after the fly ash mixed with water pumped to the mixer vessel.

Table - A: Unorganized Asbestos Units Studied at Deogarh (Rajasthan)

Sl. No.	Unit	Establishment year	No. of workers	Processing	Raw material & source	Products	Production (Tonnes/month)
1.	M/s B. K. Grindings Pvt. Ltd.	1987	06	Grinding	Asbestos (Sambharna ka Mines)	Asbestos powder	50 – 60
2.	M/s Kanchan Minerals Pvt. Ltd.	1984	07	Grinding	Asbestos (Javed Mines, Rajnagar)	Asbestos powder	60
3.	M/s Maharaja Asbestos Grinding Mills Pvt. Ltd.	1982	05	Grinding	Asbestos & stone (near by hill area)	Asbestos & stone powder	40 – 50
4.	M/s Osawal Minerals Trading Corporation	1981	07	Grinding	Asbestos & stone (Hill Area)	Stone & asbestos powder	40 – 50

Table - B: Unorganized Asbestos Units Studied at Beawer (Rajasthan)

Sl. No.	Unit	Establishment year	No. of workers	Processing	Raw material & source	Products	Production Per month
1.	M/s. Cenera Minerals Pvt. Ltd. (Beawer)	1970	03	Grinding	Grinded stone & asbestos (near by hill area)	Stone & asbestos Powder	40 –50 tonnes/month
2.	M/s. Guru Asbestos Pvt. Ltd. (Beawer)	1980-81	13	Grinding & Manufacturing	Asbestos (Hill Area) & Cement (Maihir)	Stone & asbestos powder & Cement Pipes, electric heater' plates	40 tonnes/month and 3000 pipes/month
3.	M/s. Gajanand Cement Asbestos Products Pvt. Ltd. (Beawer)	1990	04	Grinding & Manufacturing	Asbestos Powder (Hill Area) & Cement (Beawer)	Asbestos powder & Cement pipes	2000 pipes/month
4.	M/s. Kamla Grinding Mills Pvt Ltd., Beawer	1970	02	Grinding	Asbestos & stone (Near by Hill Area)	Asbestos & stone powder	50-60 tonnes/month
5.	M/s. Swastik Udyog Pvt. Ltd. (Beawer)	1996	05	Grinding	Large Size Stone (near by Hill Area)	Stone & asbestos powder	50 - 60 tonnes /month
6.	M/s. Super Minerals Pvt. Ltd. (Beawer)	1995	03	Grinding	Large small size stone (near by Hill Area)	Stone & asbestos powder	50 - 60 tonnes/month

Sheet Forming Section:

The slurry consisting of above key raw material (in required quantity/proportion) is fed through an agitator but this is necessary to note that mixture should be in homogeneous form and then it is sieved to 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 it is removed from the drums and cut to the required size and corrugated. During this process the machine continues to run and another sheet begins to form on the sheet-forming drum.

Heating Section: Green corrugated AC sheets then stocked on bogies and placed in heating chamber (temp.42-45°C) where they are kept for 12-14 hours in summer and 22-24 hours in winter for maturing.

Maturing Section: The hardened AC sheets are then cured for about 21 days or a month by water sprinkling to attain the optimum strength.

2.2.2 M/s. UP Asbestos Pvt. Ltd., (II)

M/s. UP Asbestos Pvt. Ltd., (UPAL-II) is located in Butibori Industrial Area, Nagpur (Maharashtra). It is a large-scale factory manufacturing asbestos-cement sheets and accessories as moulded goods. Total production is about 36000 metric tones per annum. Ingredients used in the factory are cement, fly-ash and chrysotile asbestos fibre (imported mostly from Russia) in quantities of 20000 MT, 12000 MT and 4000 MT respectively, per annum. They are manufacturing asbestos-cement sheets and their accessories. The total strength of the factory was of 90 persons including 45 staff and 45 workers. The total 71 individuals including staff and workers were entertained for the study. Various steps of manufacturing asbestos-based products are sketched in flow chart-3:

Process

They are processing and manufacturing the products in wet mode, which is technically known as "Hatschek Process". In this process they are opening the pressure packed impermeable polythene bags containing chrysotile by semi-automatic machine through mechanical process and milled under wet conditions by spraying water in the Hydro Disintegrator (fibre mill). Wet and milled fibres shifted to the mixing tank through closed system. Additionally cement, ordinary Portland cement (OPC) is basically a binding material and it encapsulates the asbestos fibres and fly ash. Fly-ash is a by-product as well as solid waste of thermal power plants and considered mainly as an air pollutant in the vicinity of the power plants fed into the raw material mixing tank by means of close type bucket conveyors and elevators in required proportion. In tank, slurry of raw materials is prepared. At Sheet Formation Section, the slurry obtained is taken to the Cylinder Vat through the Homogenizer Feeding Cone. The Cylinder Vat is a tank with a sieve cylinder covered by mesh cloth to help sieves the slurry. As the cylinder rotates, the water gets removed through the screen leaving a thin even film of stock deposited on its surface. The film is transferred on to endless felt, which remains in contact with the top cover of the sieve cylinder. Surplus water is 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 built up to the desired thickness. The sheet will then be knifed along a groove in the sheet formation drum roll and peeled from it to a moving rubber conveyor belt, which collect the sheet clear of 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 for 15 to 18 hours. After that sheets are demoulded, i.e. stripped off from the templates. Finally at curing section, these sheets are water cured means the sheets stacked vertically and water poured on them. This process takes about 25 to 28 days to develop optimum strength before being dispatched. Once asbestos-cement sheets and moulded goods manufactured asbestos fibres and other raw materials get firmly 'locked – in' or 'encapsulated' within the matrix by means of the binder, saturate, coating or bonding agent, such that cement and fibres could not escape into the atmosphere. In Fibre mill, chrysotile is charged mechanically through semiautomatic bag opening device. Semiautomatic bag opening device, shredding machine, fibre mill, bucket elevator etc were operating in closed system and interconnected to each other. Negative pressure in all these process equipments are maintained by induced draft fan. Discharge of the fan were connected with air pollution control device i.e. counter current scrubber such that if any fibre/dust travels along with air (sucked for maintaining negative pressure) finally trapped by automized water spray in the scrubber device.

2.2.3 M/s Allied Nippon Pvt. Ltd.

M/s Allied Nippon Pvt. Ltd., (A joint Venture of Japan Brake Industrial Co. Ltd., Tokyo, Japan) is located in Sahibabad industrial area, Ghaziabad. The factory plant is in operation since 1983. The total production capacity of industrial products is 1200 metric tones per annum. The asbestos-based manufactured products are brake linings, brake shoes, clutch facing and clutch plates etc. The total workers in the factory including officials are 365. The study comprised of 90 workers including staff in this factory. All the processes involved in the manufacturing of brake-shoes, brake lining, clutch plates and clutch facing are described and sketched with process flow chart-4.

Process

Asbestos fibre bundles that are pressure packed are cut down manually into mixing mill. Other ingredients are also poured manually and mixed automatically by electrical and mechanical machines. Now this mixture is transferred to the tin containers covered with wooden plates manually with the help of covered buckets of tin. The required quantity of mixed material is collected from these containers manually to the moulding section by wood plates covered tin trolleys and here required quantity of mixed material is weighed according to the shape and size of the product with the help of manual balance. The weighed material is moulded with the help of automatic hot moulding machine. After hot moulding, the product is sent for drilling then chamfering, cleaning and finally after stamping products are ready for packing. Mixing mill is totally covered by the tin and cloth curtains in a closed channel form and there is arrangement of exhaust fan to minimize the dust concentration, which is attached with the air pollution control system.

2.2.4 M/s Champion Seals Pvt. Ltd., (A)

Champion Seals Pvt. Ltd., Boisar, Tarapur is a medium scale factory situated in MIDC, Industrial Area, Tarapur. They are manufacturing asbestos yarn, at the rate of 20 tonnes per month. There are 115 employees working in the factory. The raw materials used in the manufacturing amounts to raw asbestos fibers 21 tonnes and poly staple fibers 20 tonnes per year.

Process

Asbestos fiber first goes through mixing process with the poly staple fiber and then carded on the carding machine. The carded cakes are then spun on spinning machine to produce asbestos yarn. The asbestos yarn is then plied and from this yarn cloth and ropes are made. For collection of asbestos dust, the factory is using dust collectors with blower machines. Various steps of manufacturing asbestos-based products are sketched in flow chart-5.

2.2.5 M/s. Mechanical Packing Industries Pvt. Ltd., (B)

M/s Mechanical Packing Industries Pvt. Ltd., Boisar, Tarapur was commissioned in 1978. It is a medium scale industry, situated in MIDC industrial area, Tarapur, Boisar. The production capacity of the plant is 500 metric tonnes per annum. There are 31 employees including 4 staff. They are using asbestos raw fibre, staple asbestos fibre and other materials for manufacturing of asbestos yarn and its allied products. Industry requires 120 metric tonnes of asbestos as raw fibre, 140 metric tonnes as staple fibre and 30 metric tonnes of other raw materials per annum for the production of asbestos yarn and its allied products. They are producing asbestos yarn and its allied products amounting 180 metric tonnes per annum.

Process

The packed form of asbestos fiber and viscous fibre are mixed with other ingredients in carding machine. Now they are fed to slubbing machine and again twisted on doubling machine. The yarn is wound on bobbins and is placed in thick polythene bags. They are using water to eliminate the dust generated in slubbing and doubling operations. Various steps of manufacturing asbestos-based products are sketched in flow chart-6.

2.2.6 M/s. Mechanical Packing Industries Pvt. Ltd., (C)

M/s Mechanical Packing Industries Pvt. Ltd., Dahishar (East) was commissioned in 1974 and it is a medium scale factory situated in industrial area at S.V. Road, Dahisar. There are 23 employees including 5 staff members. They are manufacturing 40 metric tonnes per annum of asbestos-based products like industrial packing and seals.

Process

They are using asbestos yarn, Grafseal and PTFE as raw materials totaling 50 metric tonnes per annum. They are using lubricating oil, paraffin oil, wax along with asbestos fibres, cotton fibres, jute fibres etc and processed these for mixing and winding of yarns followed by braiding. The process was wet and exhaust air passed through pollution control equipments subsequently, braiding coiling and bundling were done, finally sent for packing in polythene and corrugated boxes. Various steps of manufacturing asbestos-based products are sketched in flow chart-7.

2.2.7 M/s. Hindustan Composite Pvt. Ltd., (D)

M/s Hindustan Composite Pvt. Ltd., Paithan, Aurangabad was commissioned in 1987. There were a total of 160 employees including 130 workers and 30 staff members in the factory. It is situated in MIDC industrial area. It is a medium scale industry with total production capacity of 1350 metric tonnes per month.

Process

They are using ferrous material, non-ferrous material, rubber, solvents and asbestos amounting 659.50, 110.16, 88.56, 1120.44 and 273.36 metric tonnes per annum, respectively. They are producing brake linings for 2 wheelers, 3 wheelers and LVCs, brake linings for HCVs; roll lining for industrial uses and disk pads for railways and new generation cars. The production capacity is as 105, 800, 220, 840 and 5 metric tonnes per annum of the above-mentioned products respectively. Various steps of manufacturing process are sketched in flow chart-8.

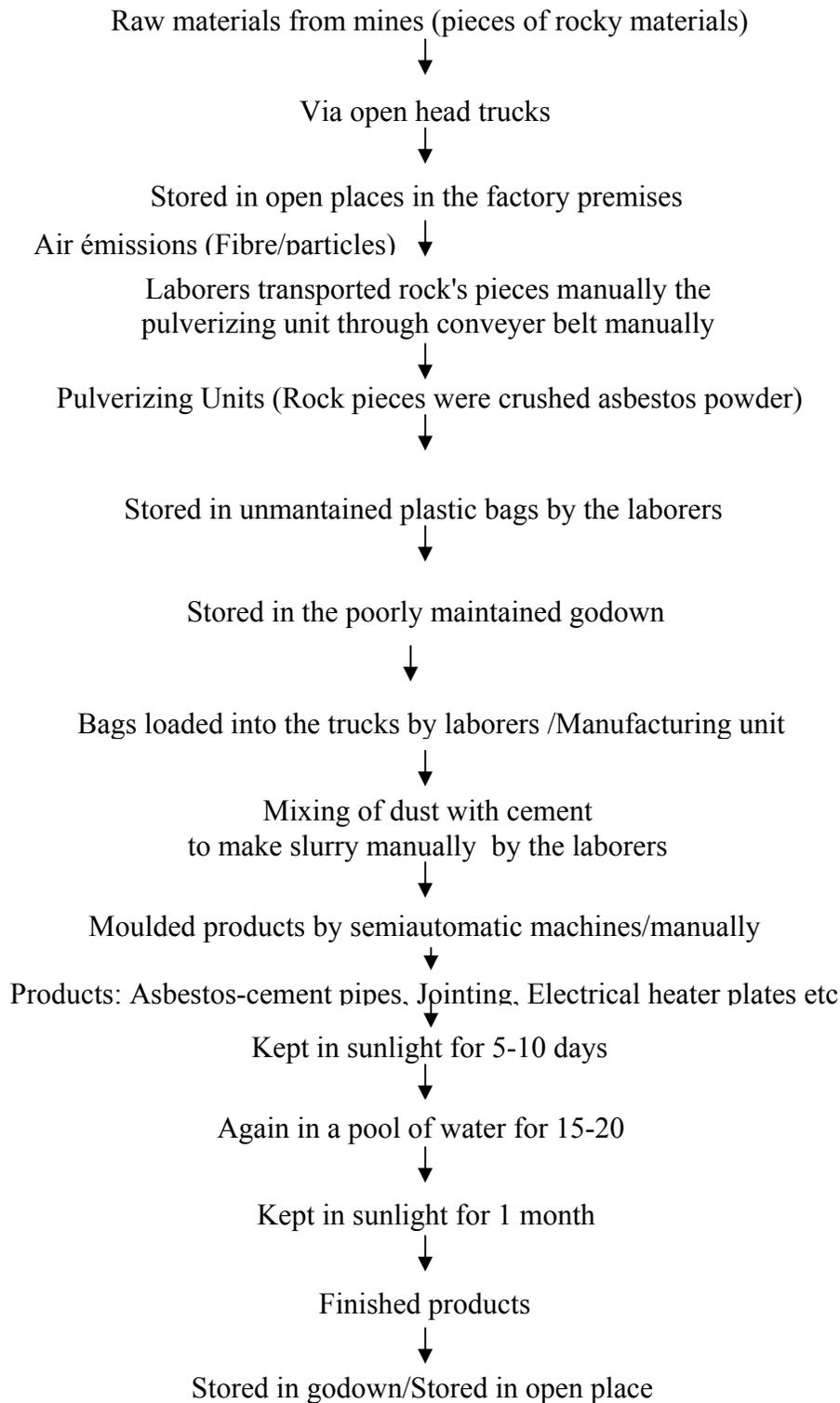
2.2.8 M/s. Hindustan Composite Pvt. Ltd., (E)

M/s Hindustan Composite Pvt. Ltd, Ghatkopar (West), Mumbai was commissioned in the year 1956. It is a medium scale industry. The strength of employees working in the factory is 460, out of which 112 were staff members and 348 workers. This factory produces a variety of products such as textile cloth, ropes (2 tonnes / day), jointing, limpet sheets (9.69 tonnes/day), mill board (1.15 tonnes/day), competes (1.192 tonnes/day) and brake linings (5.73 tonnes/day). The amount of raw materials required daily for the production is asbestos fibers (8.3 tonnes/day), rubber (0.96 tonnes/day), rubber solvents (0.23 tonnes/day), cnsl (2.30 tonnes/day), barites (3.25 tonnes/day), carbon black (0.04 tonnes/day) and sulphur (0.15 tonnes/day).

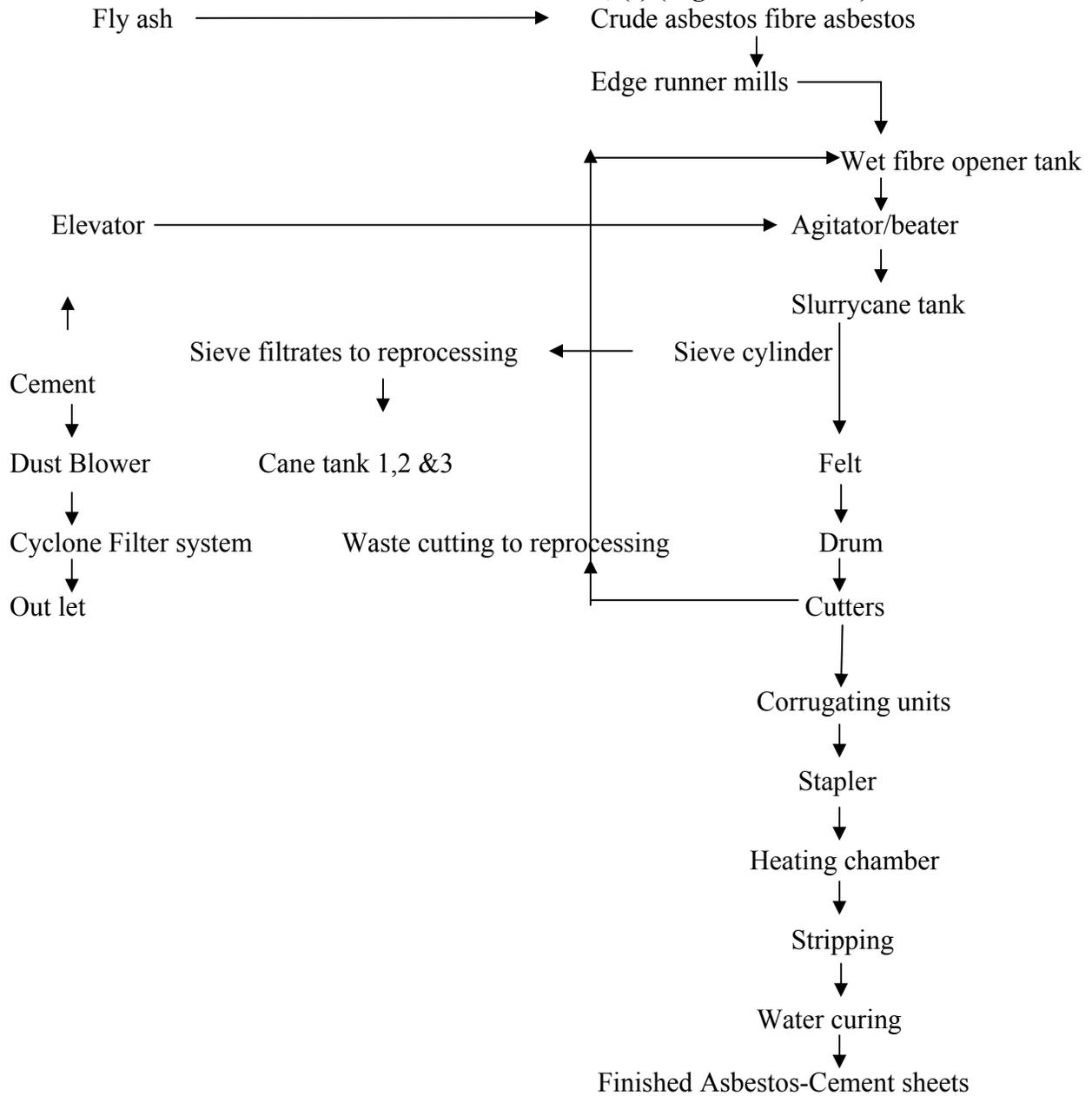
Process

The process for the textile cloth and rope formation is fiberising of asbestos and lap forming which leads to the carding and spinning of the yarn. This yarn is weaved into cloth, plaiting, and rope. The process for the compressed asbestos fiber production is the opening of bags in the material dispensing room, mixing, calendaring, trimming, polishing and stamping of the sheets. Various steps of manufacturing asbestos-based products are sketched in flow chart-9:

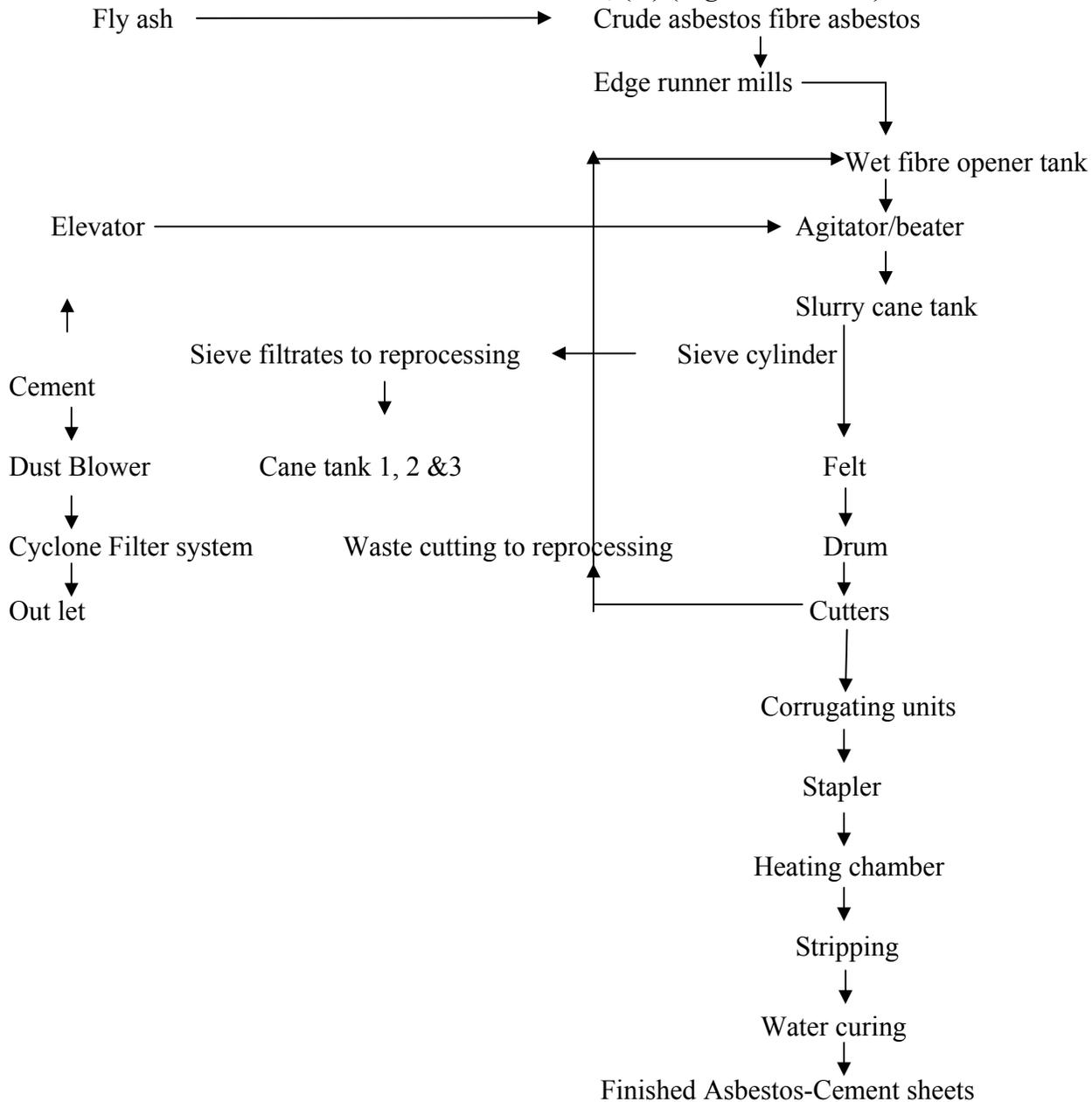
Flow Chart-1: Different steps for the manufacturing of asbestos-based products in unorganized sector



Flow Chart-2: Different steps in manufacturing asbestos-based products by M/s. U.P. Asbestos Pvt. Ltd., (I) (organized sector)



Flow Chart-3: Different steps in manufacturing asbestos-based products by M/s. U.P. Asbestos Pvt. Ltd., (II) (organized sector)



Flow Chart-4: Different steps involved in manufacturing asbestos-based products by M/s. Allied Nippon Pvt. Ltd., (organized sector)

A. Process of brake lining

Pressure packed asbestos bags → Cut and open into mixing unit → Added other ingredients such as black piper, wood powder, resin etc → Mixing → Performing → Curing → Hot Molding → Drilling → Grinding → Chamfering → Cleaning → Stamping → Final Inspection → Packing

B. Process of Clutch Facing

The mixed material sent to the hot molding after curing required grinding, then drilling and finally after final inspection products send for packing.

Mixing → Hot Molding → Curing → Grinding → Drilling → Inspection → Marking → Packing

C. Process of Clutch Plates

Clutch plates are prepared in two parts first metallic and second friction part:

C (i). Metallic part

Die casting (Brought out) → Inspection → Short blasting → Adhesive application

C. (ii). Friction part

Mixing → Rolling → Inspection → Adhesive application → hot pressing → Inspection → Marking → Packing

Flow Chart-5: Different steps involved in manufacturing asbestos- based products by M/s. Champion Seals Pvt. Ltd., (A) (organized sector)

Mixing of asbestos fibers and polystaple fibres



Carding



Spinning

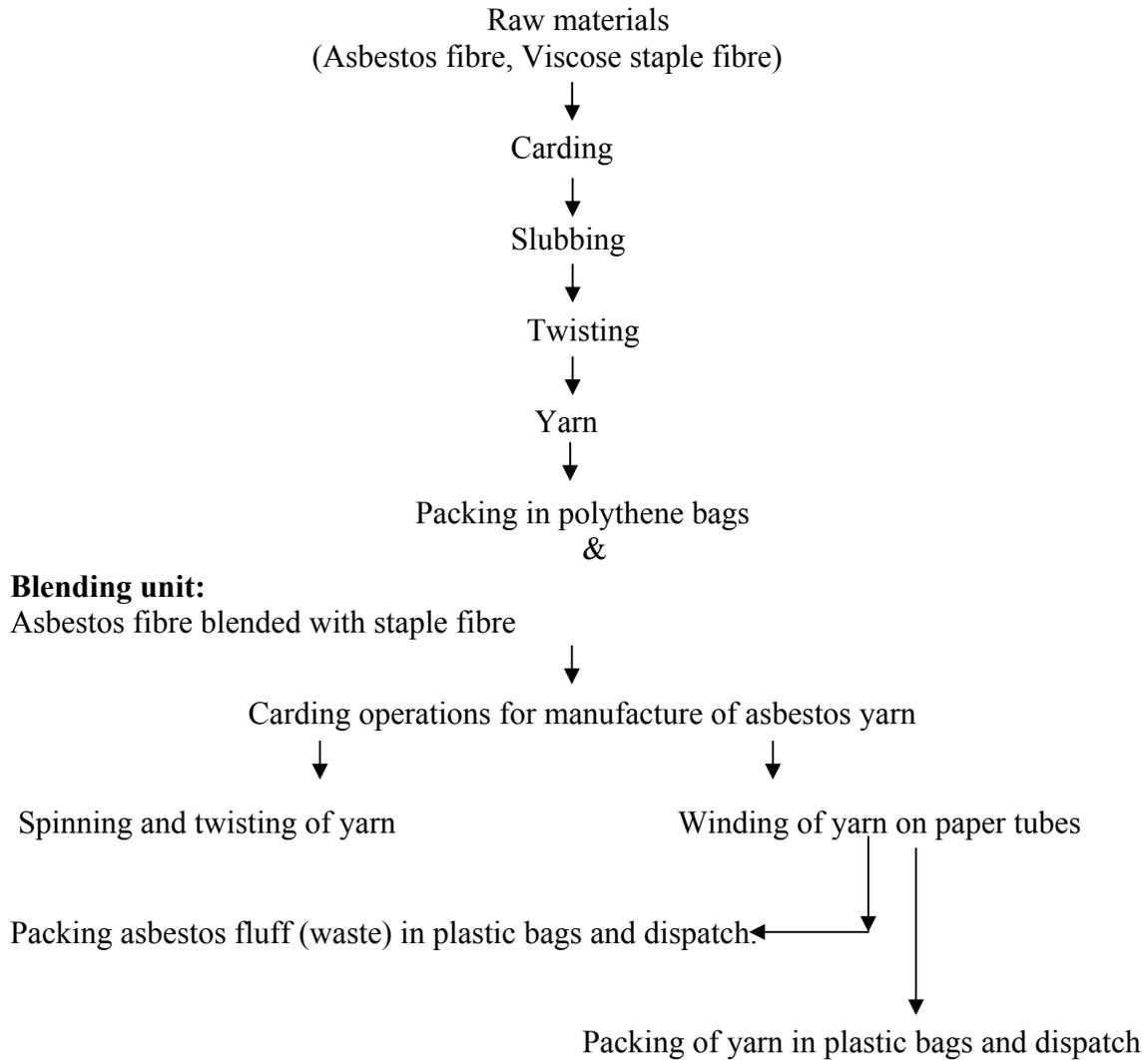


Yarning

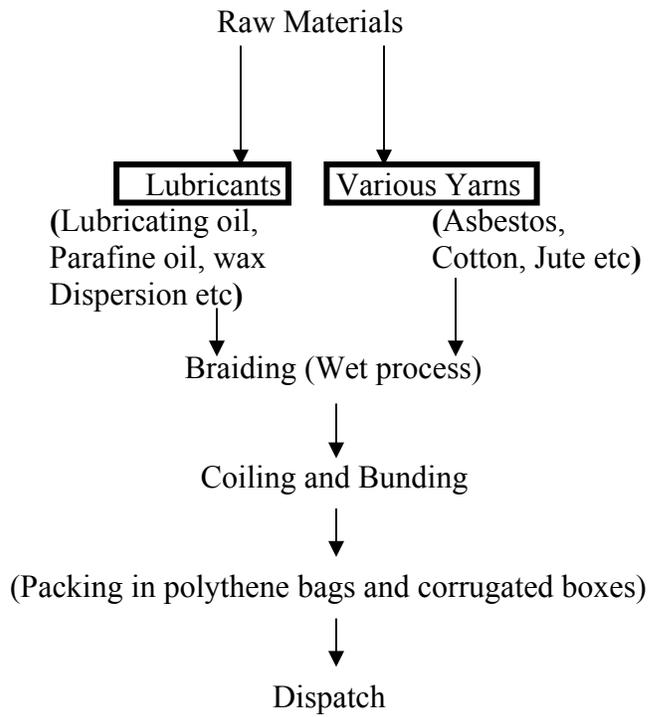


Clothes and Ropes

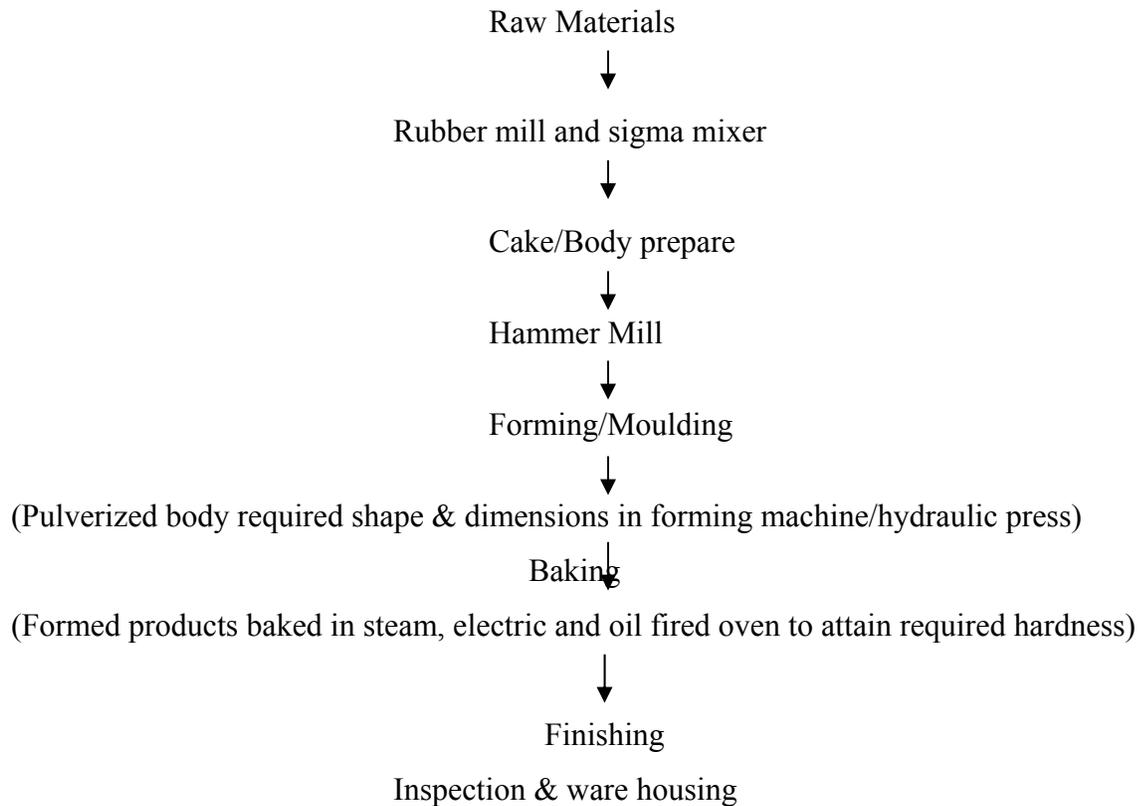
Flow Chart-6: Different steps involved in manufacturing asbestos-based products by M/s. Mechanical Packing Industries Pvt. Ltd., (B)(organized sector)



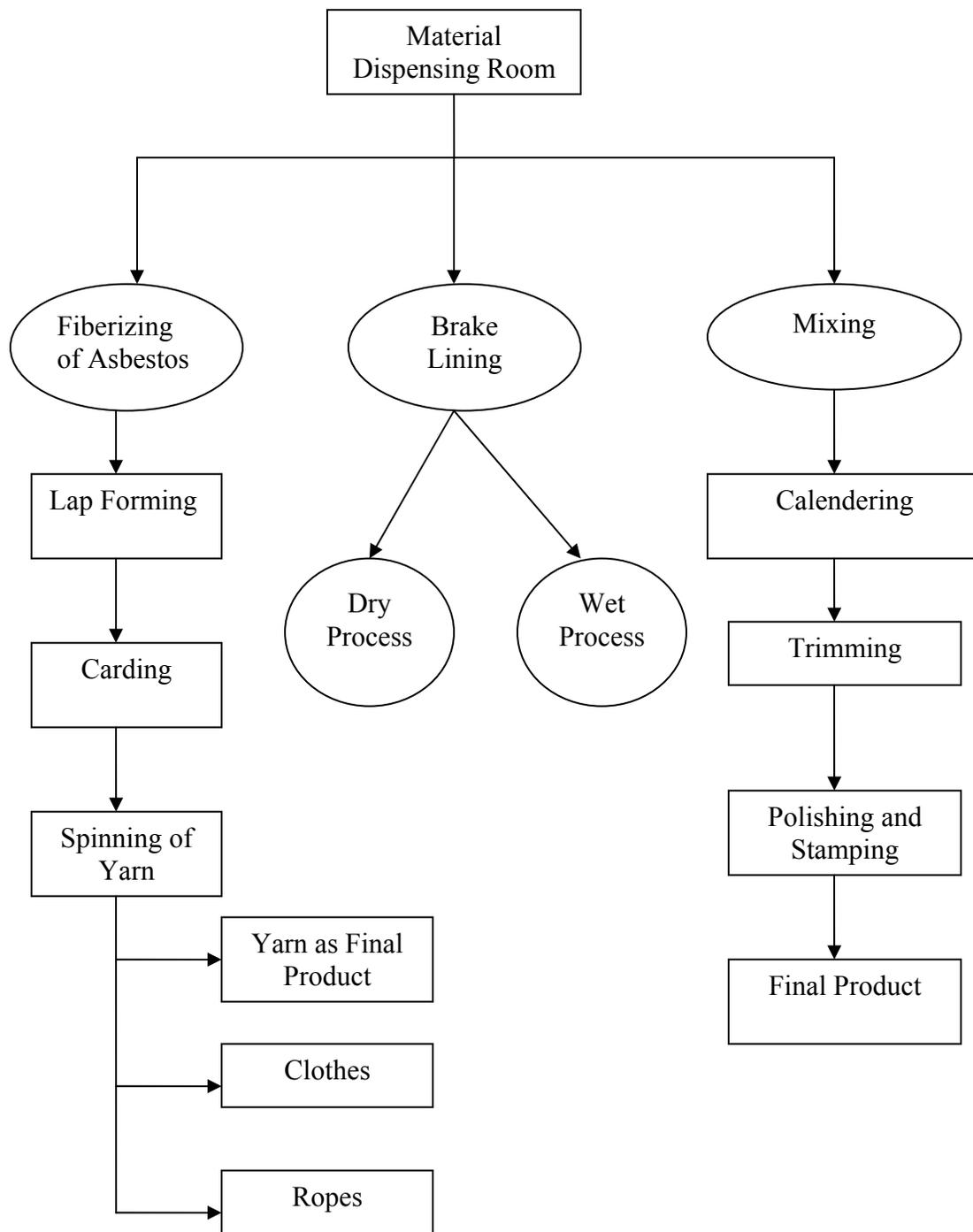
Flow Chart-7: Different steps involved in manufacturing asbestos-based products by M/s . Mechanical Packing Industries Pvt. Ltd., (C) (organized sector)



Flow Chart-8: Different steps involved in manufacturing asbestos-based products by M/s. Hindustan Composite Pvt. Ltd., (D) (organized sector)



Flow Chart-9: Different steps involved in manufacturing asbestos-based products by M/s Hindustan Composite Pvt. Ltd., (E) (organized sector)



CHAPTER 3.0

MATERIALS AND METHODS

3.1 Asbestos Fibre Monitoring, Analysis and Identification

3.1.1 Principle

The collection of environmental samples including air must follow an appropriate sampling procedure. A review of method for sampling of asbestos fibres has been published (IPCS, 1986). The most commonly used analytical method involves phase contrast optical microscopy (PCOM) in the work place and transmission electron microscopy (TEM) in the general environment. The phase contrast optical microscopy (POCM) is universally recommended for asbestos analysis (Eache and Groff, 1997; Dion and Perrault, 1994) including Bureau of Indian Standard. POCM coupled with polarized light is largely used for asbestos analysis in solid samples (USEPA, 1993).

3.1.2 Monitoring of Asbestos Fibre in Air

A general survey of inside and out side factories of the unorganized and organized sectors was conducted to choose the sampling sites. Sampling was carried out at visually selected locations appeared more prone to emission or possibility of release of asbestos fibre. The sample was collected by drawing a measured quantity of air through cellulose ester a membrane filter by a battery operated sampling pump that was fully charged to operate continuously over the chosen sampling time. The exposed filters were placed into plastic petri dishes and transferred carefully to the laboratory.

Two types of samples were taken, one within the workers breathing zone that is 300 mm radius extending in front of the face, and measured from the mid point of a line bisecting the ears called *personal samples*. The samples taken at a fixed location mostly near to the source point called *area or static samples*. Personal sampler model "XX 5700000" and low volume vacuum/pressure pump model "XX5622050" attached with monitor or cowl model "MAWP025AC" of Millipore Corporation, USA were used for the collection of personal and area samples, respectively. The flow rate of pump was adjusted to 1litre per minute. The flow rate checked before and after in each monitoring, those samples showing the difference by >10 percent from the initial flow rate were rejected. In both the samples filter holder (Cowl) always pointed downward position to avoid the deposition of heavy particles. An ester cellulose membrane filter "AAWP02500" having 0.8 μm -1.2 μm pore size and 25 mm diameter was used throughout the sampling for asbestos counts at work environment.

3.1.3 Mounting Procedure

Complete filter was placed on clean microscopic slide, dust side up at room temperature. Electrostatic force keeps the filter usually on the slide. Filter was exposed to acetone fumes and triacetin (Glycerol triacetate, Sigma). In this procedure a small quantity of acetone in round bottom flask (500-1000ml) heated at the boiling point under

water bath, the vapors condensed in a simple condensing column. When the sufficient fumes of acetone become ready passed it throughout on the filter for 3-5 seconds at a distance of 15-25 mm. Put the 1-3 drops of Glycerol Triacetate (Triacetin) on the acetone-cleared filter. Place a coverslip on cleared filter by avoiding the air bubbles. Heated the cleared filter at 50°C for 15 minutes and left it at room temperature for 24 hours under the action of triacetin to clear entire filter. Alternatively, membrane filter could also be made transparent with immersion oil (Leica Microsystems Wetzlar GmbH, Wetzlar). Using a phase contrast microscope with polarized light, Laborlux S (of M/s Leica, Germany) counting was done at magnification 400X-500x.

3.1.4 Counting of asbestos fibre

The counting fields were chosen randomly throughout the filter. Fibres were counted at 400X under phase contrast as well as polarized light microscope using Walton-Beckett graticule. The fibre counting was minimum of 20 microscopic fields if scored 100 fibres or more but continued till microscopic 100 fields maximally. Particular attention was given to the minimum and maximum fibre loading on filter. The considered minimum and maximum fibre loading on filter were 50 fibres per mm² and 650 fibres per mm², respectively. Those filter having average filter loading exceeding 10 fibres per graticule area was rejected. Blank membrane filters, 4 % of the sampled membrane filters, were similarly treated for making slides which were also analysed for the asbestos fibers. The blank filter showing 10% or more of the actual sample fibre count, the samples represented by blank are not considered in fibre count. To calculate the effective filter area coal dust was passed through the blank filter by running the pump. The black area of filter was measured for the calculation of effective filter area. There were certain criteria in fibre counting. Particles with length >5µm, diameter <3µm and length to diameter ratio ≥ 3:1 were treated as fibres. In case of split fibre, diameter was measured across the compact portion of the fibre only. Split fibres were counted as one fibre if its geometric dimensions meet the criteria of fibre. Some times, fibres are in groups and individual fibres were carefully observed and taken into counts. It is also observed that fibres are sometimes attached to particulate matter, which was counted as one fibre if the diameter of the particle is less than 3µm.

3.1.5 Calculation of fibre concentration

$$C = A/a \times N/n \times 1/r \times 1/t$$

Where:

C= concentration in fibres per cubic centimeter rounded to first place of decimal,

N = total no. of fibre counted,

n = number of graticule areas observed,

A= effective filter area in mm²

a= graticule counting area in mm²,

r= flow rate of air through filter in cm³/min., and

t= single sample duration in minutes

3.1.6 Identification of Asbestos Fibres

Membrane filter method is the recommended method for the determination of airborne asbestos fibre concentration. The limitation of this method involving phase contrast light microscopy is the count of particle bearing length and width criteria of fibre as asbestos fibre. Therefore, optical properties of fibre were also studied as shown in the following table, in order to assure at definite counts of asbestos fibres. The technique was largely applied in asbestos characterization in solid samples.

Optical characteristics of various types of asbestos under plane polarized light

Sl. No.	Asbestos Species	Appearance under plane polarized light
1.	Chrysotile	Wavy fibres. Fibre bundles have splayed ends and kinks. Aspect ratio typically >10:1. Colourless, nonpleochronic
2.	Crocidolite	Straight, rigid fibres. Thick fibres and bundles common blue to purple-blue in color. Pleochronic Birefringence is generally masked by blue color.
3.	Amosite	Straight, rigid fibres. Aspect ratio typically >10:1 Colorless or brown, nonpleochronic or weakly so. Opaque inclusion may be present.
4.	Anthophyllite	Straight, single fibres, some larger composite fibres. Cleavage fragment may be present with aspect ratio <10:1. Colorless to light brown.
5.	Tremolite/Actinolite	Tremolite as single or composite fibres, aspect ratio <10:1. Colorless to pale green.

3.1.6.1 X-Energy Diffraction Analysis

Some of the asbestos samples of unorganized asbestos units were also analyzed by X-energy diffraction method (XED) of electron microscopy to confirm the data obtained by optical microscopy. XED is one of the primary techniques used by mineralogists and solid-state chemists to examine the physico-chemical make-up of unknown solids. The unknown solids (indigenous asbestos dust of unorganized sector of Rajasthan) evenly dispersed on 0.2µm polycarbonate filters. The filters were carbon coated and 100 mesh grids were prepared by the direct transfer method. Twenty fibres and/or particles per sample were analyzed consecutively at 16, 000x by X-ED and electron diffraction. Which was done by Prof. Arthur L. Frank (The University of Texas, Department of Cell Biology and Environmental Sciences, USA) and this kind help is gratefully acknowledged.

3.2 Clinical Examination

The subjects from asbestos – based industries (exposed population) and control population having the similar socio-economic status were selected in this study. Each subject was given to answer a complete set of questionnaires based on modified British Medical Research Council (BMRC, 1976) to assess an accurate medical history, habits, past and present occupation, duration of exposure and socioeconomic patterns including respiratory history. Medical history focused on previous and present respiratory illness. A history of cough, sputum production, wheezing, and chest pain was also determined. Information regarding specific occupational history, domestic exposure, smoking, alcohol consumption and nutritional habits was also collected. Each subject was thoroughly examined by a medical specialist for complete clinical examination with special emphasis on respiratory system. All subjects were given detailed information regarding the scope of the study and their consents were obtained.

3.2.1 Pulmonary Function Test

Pulmonary function test of individual subject was performed using Vitalograph Spirometer model Micro Medical Ltd., USA and OHD-KoKo Spirometer, USA, according to the guidelines of American Thoracic Society (1987). Spirometry is a medical test that measures the volume of the air of an individual inhales or exhales at a function of time. Flow or the rate at which the volume is changing at a function of time may also be measured with spirometry. Flow is lower in the early morning hours so best performance is expected between 10 to 12 noon. Spirometry is recommended for patients with respiratory symptoms such as chronic cough, episodic wheezing and exertion dyspnoea in order to detect airways obstruction or restrictions.

After recording the slow vital capacity (VC), a forced expiratory maneuver (FVC) was obtained from each subjects in the standing posture (Wang *et al*, 2001). The forced expiratory maneuver was performed at least three times on each subject and the best of the three attempts was selected for the data analysis. Following the lung function testing, the standing height and body weight were noted to predict the normal values of pulmonary function test using Rastogi's prediction equation (Rastogi *et al.*, 1983).

The following were the Spirometric measures.

1. Vital Capacity or Slow Vital Capacity (VC)
2. Forced Vital Capacity (FVC)
3. Forced Expiratory Volume in one second (FEV₁)
4. FEV₁/FVC%

The severity of the pulmonary function impairment was graded as per Conrad *et al.*, (1983) as follows:

- i) **Mild respiratory impairment**
Observed values of vital capacity and forced expiratory volume in one second ranging between 61-79% of the predicted.
- ii) **Moderate respiratory impairment**
Observed Spirometric values of vital capacity and forced expiratory volume in one second ranging between 40-66% of the predicted.
- iii) **Severe respiratory impairment**
Observed vital capacity and forced expiratory volume less than 40% of the predicted.

3.2.2 Radiological Examinations

For radiological examination of subject, Postero-anterior chest x-ray was taken at the time of survey. Due attention was given during the x-ray examination in such direction that x-ray taken with the film against the front of the patient's chest and x-ray tube, 2 meters behind the patient. X-ray plates were examined systematically by a radiologist on a viewing box according to the guidelines of International Labor Organization, 1980, especially for the presence of:

1. Linear shadows of varying thickness
2. Pleural thickening, pleural plaques, bilateral or unilateral pleural calcification
3. Honey-combing
4. Reticulonodular Pattern
5. Prominent broncho-vascular marking

Chronic bronchitis was diagnosed on the strong basis of clinical history of chronic cough for three consecutive months for two successive years and further deep-rooted radiological evidence of hilar prominence and prominent broncho-vascular markings.

A few High Resolution Computed Tomography (HRCT) was also done on radiologically positive asbestostotic subjects wherever facilities existed for the access.

3.2.3 Sputum Analysis

Acid-fast bacilli

Sputum samples were collected in clean sterilized bottles from the deep of the throat (preferably early morning sample) of the suspected cases. The thick, yellowish, purulent portion of the sputum was transferred on to the slides by using the jagged ends of the broken broom stick (wooden or bamboo) and precautionary used separate stick for each sample. Spread sputum in such a way to cover 2/3 of the central position of the slide, in size of the smear approximately 3x2 cm and taking care that smear is neither thick nor too thin and left the slides to become air dry for 5-10 minutes. Few samples were dissolved in 4% NaOH in 1:1 ratio and kept at room temperature for 5-10 minutes and centrifuged at 1500 rpm for 10 minutes. Supernatant discarded and smear was made from the pellet and dried in the air at room temperature. Further, these slides were stained with 1% carbol fuchsin (boiled) or (heat the slides till the vapors develop) to cover the entire smear area and left for 5 minutes. Rinse the slides with tap water to remove excess carbol fuchsin stain. Tilted the slide to run off excess water and sputum

in this stage appears as reddish in color. 25% sulphuric acid was used for its decolourisation. The reddish color almost completely disappeared from the smears. Tilt the slide to drain off the excess water and to air dry if slide is still reddish, reapply sulphuric acid until the red color disappears from the smear completely. These slides were counter stained with 1% methylene blue as suggested by Ziel-Neelsen staining (MLT, 1985) and let it stand for 30 seconds. Slides were rinsed with tap water, water drained off and allowed to air dry at room temperature. Slides were examined at 40X to check clarity of smear under microscope. Slides were examined under microscope 1000X after placing immersion oil on the stained smear. At least 100 microscopic fields were examined (RNTCP, 1999). Pink in color and rod like structures are suggestive of active tuberculosis lesion.

Examination	Results	Grading	No. of fields to be examined
More than 10 AFB per oil immersion field	+Ve	3±	20
1-10 AFB per oil immersion field	+Ve	2±	50
10-99 AFB per oil immersion field	+Ve	1±	100
1-9 AFB per oil immersion field	Scanty	Record Exact No.	200
No AFB per oil immersion field	-Ve	-	100

Asbestos Bodies Analysis (Papnicolaou Stain) Rapid Stain Method Principle

Ferruginous bodies are typically asbestos fibres that have become coated with an iron-rich material, which is believed to be derived from proteins such as ferritin and hemosiderin (Pooley, 1972). These are known as ferruginous bodies. Literature reveals that these are coated fibre, elongated, golden brown structurally 10-60 µm length and 0.5-2.5 µm wide beaded or pear or round shape in appearance with many segments. These fibres are generally formed on straight fibres and always found to occur by all the commercial type of asbestos but less frequently on chrysotile asbestos fibres. Asbestos bodies have crystalline component which is structurally similar to the extract of ferritin (an inorganic iron containing core, covered by a shell of protein, this protein is iron free and composed of approximately 20-24 peptide chains per ferritin molecule, which forms a hollow sphere with a radius 60-70 Å, the ferritin core may be fericoxyhydroxides (Gross *et al.*, 1999). Fericoxyhydroxide core of ferritin is variable in shape with maximum dimensions of approximate 60Å produced from animal and human organs. The size of the fibre plays no part in deciding which fibre becomes coated or not.

Asbestos Body Staining and Analysis

Asbestos bodies were analyzed following the methodology of Williams *et al.*, (1982). Used the pap stain kit (Bio Lab Diagnostic) for the identification of asbestos bodies in sputum smear.

Papanicolaou Staining

Sputum smear were prepared as described earlier and stained with Papanicolaou stain (Rapid Stain Method) of Bio Lab Diagnostic (Maharashtra). Few samples especially in the poor quality and quantity were dissolved in 4% NaOH in 1:1 ratio and kept at room temperature for 5-10 minutes and further, centrifuged at 1500 rpm for 10 minutes. Supernatant discarded and smear was made from the pellet and dried at room temperature. Sputum samples were fixed in 1:1 ratio ether and alcohol. The slides were stained in Scott's water (Water Buffer) for 2 minutes and excess water was removed by blotting paper and further, slides were stained with nuclear stain (Hematoxylin solution) for 45 seconds and removed again excess stain with blotting paper and again put the slides in Scott's water for 30 seconds and finally slides were transferred into cytoplasmic stain (1:1) 2A (Orange G-6 solution) and 2B (Light Green Eosin solution) for 1 minute. Repeated the Scott's water process and fixed the slides in dehydrant (Propanol) ethyl alcohol for 30 seconds. Transferred the slides in xylene for 1 minute and mounted with DPX (Glass Mounting Medium).

3.2.4 Cytogenetic Analysis

Genetic damages caused by asbestos fibers was analyzed through micronucleus (MN) assay in smokers and non-smoker asbestos-exposed workers of asbestos cement factories, and also from smokers and non-smokers not exposed to asbestos, with matching age, sex and socioeconomic conditions. The details are as followed. Briefly, 1-2 drops (0.06-0.1 ml) capillary blood was collected in a heparinized microcentrifuge tube by finger puncture with a sterilized needle. Methylcellulose solution (0.3%) was added to the whole blood in a V/V ratio of 1:2 to 1:3. Microcentrifuge tubes were set in a water bath at 37°C for about 40 min and then centrifuged at 1000 rpm for 6 min. The supernatant was decanted leaving about 5µl of supernatant and the pellet resuspended in that. The suspended sediment was spread onto a slide. Smears were dried and fixed in 100% methanol for 1 min. The slides were rinsed in buffer, dried and stained with 5% Giemsa. Blind-coded, slides were examined at a magnification of 1000 X and small lymphocytes (1000 no.) are counted per slide.

In vitro analysis of genetic damage was also done by MN assay and sister chromatid exchange (SCE) in human peripheral blood lymphocytes. Asbestos fiber samples were collected from the fields from Beawer and Deogarh, Rajasthan. Fiber size of <30 µm was sieved, sterilized by heating at 100 °C for 2 hours and suspended in PBS (1 µg/ml). For MN analysis, whole blood was incubated in vitro at 37° C, with airflow having 5% CO₂ for 72 hours in complete RPMI 1640 medium. The T-lymphocytes were stimulated by phytohemagglutinin-M. Asbestos fibers were suspended in medium and at the concentration of 5 µg/ ml were added in the culture vials. Cytochalasin-B was added at 44 hours of incubation. After 72 hours of incubation, cells were collected and given hypotonic treatment to fix in Carnoy's fixative. The fixed binucleated cells were spread on the slide and stained with 5% Giemsa. For SCE analysis, whole blood was incubated in a way similar to that of as MN for 72 hours. After 24 hours of incubation, BrdU was added and cultured vials were wrapped with aluminium foil to prevent exposure to light. One hour prior to harvest colchicine treatment was given to arrest the cells in metaphase. After completion of incubation the cells were treated with hypotonic solution, fixed in Carnoy's fixative and were spread on slides. For differential staining, the slides were, incubated with bis benzamide (Hoeschst 33258) in dark. After 10 min, slides were mounted with 2x SSC and exposed to UV radiation for 1-2 hours. Finally slides were stained with Giemsa and studied under light microscope.

CHAPTER 4.0

RESULTS

Many industries belonging to unorganized sector and organized sector were randomly selected for study purposes. Each of them was surveyed to understand the manufacturing processes which helped in understanding the relative degree of exposure risk. In the light of this survey informations were collected with respect to:

1. Industrial processes,
2. Genotoxicity assessment of indigenous variety of asbestos,
3. Monitoring of asbestos,
4. Demographic data of Industrial workers,
5. Clinical examination of industrial workers,
6. Chest radiological examination of industrial workers,
7. Sputum analysis of industrial workers.

Various tables and figures have been prepared from the collected data. The analysis of these tables and figures is given below:

Table-1: Shows data on fiber counts in 4 grinding units at Deogarh, Rajasthan. Asbestos fibers concentration in these grinding units ranged from 4.07-15.6 f/cc. Highest fiber counts, 15.6 f/cc was found in Oswals Minerals Trading Corporation followed by B.K. Grinding Pvt Ltd. (11.8 f/cc), Maharaja Asbestos Grinding Mills Pvt Ltd. (6.07 f/cc) and Kanchan Minerals (4.07 f/cc).

Table-2: Shows data on fiber counts in 6 asbestos grinding units at Beawer, Rajasthan. Asbestos fiber concentrations in these grinding units ranged from 2.0-5.09 f/cc. Highest fiber count, 5.09 f/cc was found in Cenera Minerals Pvt. Ltd. followed by Guru Asbestos Pvt Ltd. (4.67 f/cc), Swastic Udyog Pvt Ltd. (3.34 f/cc), Gajanand Cement Asbestos Products Pvt Ltd. (2.96 f/cc), Kamala Minerals Pvt Ltd. (2.8 f/cc) and Super Minerals Pvt Ltd. (2.0 f/cc).

Table-3: Shows data on the types and contents of asbestos in five samples of visibly asbestos rich pieces, which was done analytical electron microscopically by Prof. Arthur L. Frank (USA). All the samples were highly rich with tremolite asbestos to extent of 100% (Samples 1 and 2), 85% (Sample 3), 30% (Sample 4) and 90% (Sample 5). One of the samples (no.3) also showed 5% anthophyllite.

Table-4: Shows data on fiber counts in UPAL (II), Nagpur, the youngest factory in organized sector surveyed. Asbestos counts ranged from 0.057-0.080 f/cc. Within the work environment, highest concentration was detected at ingredients feeding site (0.08 f/cc) followed by fiber godown (0.078 f/cc), factory gate (0.072 f/cc) and sheet producing (0.057 f/cc).

Table-5: Shows data on asbestos monitoring at different locations within 5 organised industries at Mumbai (Maharashtra), manufacturing different asbestos products. A (Champion Seals Pvt Ltd., Boisar, Mumbai) manufactures asbestos yarn ropes which registered mean fiber concentrations 0.188 f/cc and 0.206 f/cc in its yarning and mixing waste products sections, respectively. B (Mechanical Packing Industries Pvt Ltd., Tarapur – Boisar, Mumbai) manufactures asbestos yarn and its products wherein mean fiber concentrations were 0.146 f/cc, 0.212 f/cc and 0.257 f/cc in its yarning and calendaring and weaving sections, respectively. C (Mechanical Packing Industries Pvt Ltd., Dahisar, Mumbai) showed 0.171 f/cc and 0.215 f/cc concentrations monitored by personal sampler and area sampler, respectively in the yarning section. D (Hindustan Composite Pvt Ltd., Aurangabad, Maharashtra) manufactures asbestos yarn brake linings, disc pads etc. has shown fiber concentrations of 0.321 f/cc and 1.66 f/cc in dust collector and debagging sections, respectively. E (Hindustan Composite Pvt Ltd., Ghatkopar, Mumbai), manufacturing textile cloth ropes, jointings, brake linings, limpet sheet etc., was monitored as 0.189 f/cc, 0.260 f/cc, 0.198 f/cc, 0.311 f/cc, 0.221 f/cc and 0.369 f/cc in yarning, calendaring, weaving, combined dust collector, fiber dust collector and mixing waste products, respectively.

Figure 1 - 4: Show data on fiber counts in 3 different lines of the UPAL, Mohanlal Ganj, Lucknow. Fiber counts in line I (**Figure 1**), line II (**Figure 2**) and line III (**Figure 3**) ranged from 0.086-0.241 f/cc, 0.054 - 0.118 f/cc and 0.072 – 0.245 f/cc, respectively. Asbestos counts in fiber godown, time office and main gate were 0.149 f/cc, 0.072 f/cc and 0.048 f/cc, respectively (**Figure 4**).

Figure 5: Shows levels of fiber concentrations in brake shoe factory, M/S Allied Nippon Pvt Ltd., Ghaziabad at locations in mixing, moulding, finishing, bonding and clutch facing section as 0.112 f/cc, 0.05 f/cc, 0.05 f/cc, 0.082 f/cc and 0.127 f/cc, respectively.

Figure 6: Shows data on fiber counts at three locations in M/S Allied Nippon Pvt Ltd., as 0.038 f/cc, 0.057 f/cc and 1.23 f/cc in fiber godown, time office and main gate, respectively.

Figure 7: Shows the data due to incubation of the human lymphocytes with 10 µg/ml concentration of different dust samples resulting in a significant increase in MN induction as compared to control unexposed cells. The maximum of this effect was observed in the sample 1 (13.33 MN/1000 cells), followed by sample 2 (12.7 MN/1000 cells), sample 5 (12 mn/1000 cells), sample 4 (6 MN/1000 cells), sample 3 (9.6 MN/1000 cells), as compared to control unexposed cells (3.33 MN/1000 cells).

Figure 8: Shows the micronucleus formation (MN) in capillary blood of exposed population and the data have been sub grouped exposure wise as years of exposure <5, <10, <15, <20 and >20. Smoking is known to affect MN, therefore data were also compared between MN of smoking and non-smoking workers in unorganized industrial units. It was interesting to note that MN counts in workers were higher than unexposed subjects and the enhancement in MN count correlated with the period of occupational exposure. It was also noted that MN counts were higher in smokers as compared with non-smokers both in exposed and unexposed populations. Non-smoking workers in comparison with non-smoking unexposed subjects, registered increased MN counts by 1.78, 2.0, 2.1, 2.6 and 3.1 fold in subgroups <5, <10, <15, <20 and >20 years, respectively. Likewise, smoking workers in comparison with smoking unexposed subjects, registered increased MN counts by 1.2, 1.6, 2.0, 3.0, 3.3 fold in subgroups <5, <10, <15, <20 and >20 years, respectively.

Figure 9: Shows induction in sister chromatid exchange (SCE) in human blood lymphocytes caused in vitro by samples (A and B) collected from unorganized industrial units in Rajasthan. Both the samples of tremolite (A and B) induced SCE by 1.5 to 2-fold. Like MN induction, Sample B was more potent than sample A in causing SCE in vitro in human blood lymphocytes.

Figure 10: Shows the formation of MN in the blood lymphocyte from the asbestos-cement factory workers (UPAL, Mohanlal Ganj, Lucknow) along with the control. The maximum no. of MN were observed in the case of exposed smokers (18 ± 0.141 Mn/1000 cells) which was significantly higher than that of exposed non-smokers (14.35 ± 0.494) followed by control smokers (8.5 ± 0.425 MN/1000 cells). The least MN base line frequency was observed in unexposed non smokers (4.7 ± 0.427 MBN/1000 cells).

Figure 11: Shows the formation of MN in the blood lymphocytes from the brake-shoe factory workers (M/S Allied Nippon Pvt Ltd.). MN formation was higher in smokers of both the populations i.e. exposed and the control.

Table-6: Shows demographic data of asbestos – exposed population in three asbestos factories of organized sector. Mean age values were 43 ± 7 , 29.1 ± 7.4 , 33.0 ± 7.0 , 43.0 ± 9.0 in UPAL (I), UPAL (II), ANL and A-E, respectively. Factory wise, heights and body weights were quite matching averaging 164.8 ± 6.3 and 59.5 ± 10.6 , respectively.

Table-7: Shows the personal habits related to the individual life styles in organized asbestos – based factory workers. The over all prevalence include smokers 102 (21.25%) non – smokers 359 (74.79%) and ex-smokers 1 (0.20%), tobacco chewers 18 (3.75%), alcoholic 98 (20.41%), non-alcoholic 382(79.58%), vegetarian 158 (32.91%) non – vegetarian 322 (67.08%). Individual industry as UPAL (I) shows pattern of smoking 39 (37.5%), non – smoking 53 (50.96%) ex-smoking 1 (0.96%), tobacco

chewing 11(10.57%), alcoholic 24 (23.07%), non – alcoholic 80 (76.92%), non vegetarian 52(50%) vegetarian (50%). UPAL (II) shows the pattern as smokers 7(9.85%), non- smoker 59 (83.09%), tobacco chewers 5(7.04%), alcoholic 2(2.81%), non-alcoholic 69 (97.18%), non-vegetarian 48 (67.60%), 23(32.39%). ANL pattern was found as smokers 35 (38.88%), non – smokers 55(61.11%), alcoholic 17 (18.88%), non – vegetarian 41(45.55%) vegetarian 49(54.44%). A-E shows smoking habit 21(9.76%), tobacco chewing 2 (0.93%), alcoholic 55(25.58%), non-alcoholic (160(74.41%), non – vegetarian 181(84.18%), vegetarian 34 (15.81%), prevalence of smoking was almost similar UPAL (I) and ANL. Likewise UPAL (II) and A-E matched together. Alcoholic consumption was maximum in A-E while minimum in UPAL (II). Non – vegetarian were respectively higher in A-E and UPAL (II).

Table-8: Shows the type of domestic energy for cooking, heating and lighting house by industrial workers of organized sector. Maximum numbers of individuals were using LPG amounting 208 (43.33%) and minimum was (wood and animal dung cake) i.e. 2 (0.41%) especially for cooking. Other type of fuel use pattern was recorded as wood 11 (2.29%), wood + kerosene 26 (5.41%), wood + gas 12 (2.5%), kerosene 17 (3.45%), combination of wood + animal dung + coal + crop residues + kerosene + electricity) 204 (42.5%). Individually, UPAL (I) shows fuel type pattern as wood nil, wood + animal dung cake 2 (1.92%), wood + kerosene 2 (1.92%), wood + gas nil, gas 1 (0.96%), kerosene nil, animal dung nil, combination (wood + animal dung + coal + crop residues + kerosene + electricity) 99 (95.19%). Domestic fuel pattern in UPAL (II) shows wood nil, wood + animal dung cake nil, wood + kerosene nil, wood + gas nil, wood + gas nil, gas 71 (100%), kerosene nil, animal dung nil, combination (wood + animal dung + coal + crop residues + kerosene + electricity) nil. ANL shows fuel type patter as wood nil, wood + animal dung nil, wood + kerosene 2 (2.22%), wood + gas nil, gas 58 (64.44%), kerosene 5 (5.55%), animal dung nil, combination wood + animal dung + coal, + crop residues + kerosene + electricity 25 (27.77%). Factory workers of A-E used wood 11(5.11%), wood + animal dung cake nil, wood + kerosene 22 (10.23%), wood + gas 12 (5.58%), gas + electricity 78 (36.27%), kerosene 12 (5.58%) animal dung cake nil, combination of wood + animal dung cakle + coal + crop residues + kerosene + electricity 80 (37.20%).

Figure -12: Shows the prevalence of clinical symptoms and signs of the factory workers of organized sector (N=236). Individually, UPAL (I) shows Cough 27(25.96%), Dyspnoea on exertion 26(25%), Haemoptysis 9 (8.65%), Clubbing 5(4.80%), Crepitations 8 (7.69%), Ronchi 15 (14.42%). In UPAL (II) no one with any clinical symptoms and signs, the reason may be the recent starting of factory. In ANL, Cough 29(32.22%), Dyspnoea on exertion 14(15.15%), Haemoptysis 3 (3.33%) and Clubbing 56 (6.66%), Crepitations 9(10%), Ronchi 9 (10%). A-E shows cough 56(26.04%), Dyspnoea on exertion 65(30.23%), Haemoptysis 7(3.25%) and Clubbing 52(24.18%), Crepitations 52(24.18%), Ronchi nil.

Table-9: Shows the exposure wise distribution of clinical symptoms (59.61%) and signs (26.92%) in factory workers of UPAL I. There were subjects with cough as 27(25.96%), dyspnoea on exertion 26(25%), hemeoptysis 9(8.65%) and Clubbing 5 (4.80%), crepitation 8(7.69%), ronchi 15 (14.42%). Symptoms were found just double than that of signs.

Table 10: Shows the exposure wise distribution of clinical symptoms and signs of the individuals in UPAL (II). There was not a single individual found having any clinical symptoms and signs, reason may be comparatively newer factory.

Table 11: Shows the exposure wise distribution of clinical symptoms and signs in ANL. The total symptoms 46 (51.11%) including cough 29(32.22%), dyspnoea on exertion 14 (15.55%), hemeoptysis 3 (3.33%). The total signs were 24 (26.66%) including clubbing 6(6.66%), crepitation 9 (10%), ronchi 9 (10%). Symptoms were higher as compared to signs.

Table-12: Shows the exposure wise distribution of clinical symptoms and signs in factory workers in A-E. The total clinical symptoms were 128 (59.53%) including cough 56(26.04%), dyspnoea on exertion 65(30.23), haemoptysis 7(3.25%). The total clinical signs were 104 (48.37%) including clubbing as 52(24.18%), crepitations 52(24.18%) and ronchi nil.

Table-13: Shows the lung function status in different factory workers of organized sector. A total of 409 exposed population shows mean of FEV₁ (L) 2.23 ± 0.65 , FVC (L) 2.67 ± 0.77 , FEF (L/s) 81.58 ± 19.33 , PEF (L/min.) 318.82 ± 135.44 , FEV₁/FVC (%) 83.56 ± 22.49 . Individually, 104 workers of UPAL (I) showed as mean FEV₁ (L) 2.00 ± 0.60 , FVC (L) 2.49 ± 0.59 , FEF (L/s) 75.72 ± 22.03 , PER (L/min.) 272.64 ± 123.56 , FEV₁/FVC (%) 80.73 ± 17.90 . ANL showed as mean FEV₁ (L) 2.14 ± 0.68 , FVC (L) 2.48 ± 0.71 , FEF (L/s) 85.30 ± 19.54 , PEF (L/min.) 246.34 ± 126.96 , FEV₁/FVC (%) 86.32 ± 18.02 . Likewise, A-E showed the pattern of LFT parameters FEV₁ (L) 2.37 ± 0.62 , FVC (L) 2.83 ± 0.84 , FEF (L/s) 82.86 ± 17.20 , PEF (L/min.) 371.50 ± 122.03 , FEV₁/FVC (%) 83.82 ± 25.84 .

Figure -13: Shows the lung function impairments as 42 (40.38%) in UPAL (I), 20 (28.16%) in UPAL II, ANL 31 (34.44%) and A-E 100 (46.51%). The overall lung function impairments were in 193 (40.80%). Higher prevalence was found in A-E industries and lower in UPAL (II).

Figure -14: Shows the lung function obstruction and restriction levels in different industries of organized sector. UPAL (I) showed pattern as 24 (23.07%) obstruction [of which mild 12; 50%, moderate 9; 37.5% and severe 3; 12.5%]. Restriction measured 10 (9.61%) of which [mild 5 (50%), moderate 3 (30%), severe 2 (20%)] and mixed type 8

(7.69%) of which [mild 5 (62.5%), moderate 2 (25%), severe 1 (12.5%)]. In UPAL (II) 4 (5.63%) obstruction of which [mild 3 (75%), moderate 1 (25%), severe nil]. Restriction was measured 16 (22.53%) of which [mild 10(62.5%), moderate 6 (37.5%), severe nil but there was no individual in mixed type category]. In ANL 15 (16.66%) obstruction [in which mild 6 (40.0%), moderate 8 (53.33%), severe 1 (6.66%)]. Restriction was measured 12 (13.33%) [in which mild 7 (58.33%), moderate 4 (33.33%), severe 1 (8.33%)] whereas mixed type shows 4 (4.44%) [in which mild 1 (25%), moderate 2 (50%), severe 1 (25%)]. In A-E industries obstruction was in 24 (11.16%) in which [mild 12 (50%), moderate 11(45.83%), severe 1 (4.16%)] and restriction in 76 (35.34%) in which mild 49 (64.47%), moderate 26 (34.66%) severe 1 (1.31%).

Table 14: Shows exposure wise total lung function impairments which were found to be correlated with duration of exposure in UPAL (I). Lung function impairments registered 5 (27.77%) in <5 years of exposure, 5(41.66%) in 5-10 years of exposure and 32 (43.24%) in >10 years of exposure. Obstruction was recorded in < 5 years of exposure as 5 (27.77%) of which [mild 3 (16.66%), moderate 2 (11.11%) and severe was nil]. There was not a single individual in the restriction and mixed type of categories. In 5-10 years of exposure category 3 (25%) was obstruction of mild type. There were 2 (16.66%) mixed and mild type of impairment. In > 10 years of exposure 16(21.62%) was obstruction of which mild 6 (8.10%), moderate 6 (8.10%) and severe 4 (5.40%) while restriction was 10 (13.51%) of which mild 5 (6.75%), moderate 3 (4.05%) and severe 2 (2.70%) and mixed type of impairment shows 6 (8.10%) of which mild 3(4.05%), moderate 2(2.70%) and severe 1(1.35%).

Table-15: Shows exposure wise total lung function impairment which were found to be correlated with duration of exposure in UPAL (II). Total lung function impairment was found in 20 (31.25%) but in <5 years of exposure impairments was recorded as 17(29.82%), obstruction 4 (7.01%) in which mild 3 (5.26%), moderate 1 (1.75%) severe nil. Restrictions was in 13 (22.80%) in which mild 8 (14.03%), moderate 5 (8.77%), severe nil. In 5-10 years of exposure category impairment found as obstruction was nil while restriction was 3(50%) in which as mild 2(33.33%), moderate 1 (6.66%), severe nil. In >10 years of exposure category only one individual was there that was the normal.

Table 16: Shows exposure wise total lung function impairments and type of impairments in ANL which were found to be correlated with duration of exposure. Total impairment was found 31 (34.44%). In <5 years of exposure category impairment was nil. In sub-group 5-10 years of exposure, obstructive impairment was 5(9.25%) in which mild 2(3.70%), moderate 3 (5.55%), severe nil. The restrictive impairment was 5 (9.25%) in which mild 3 (5.55%), moderate 1 (1.85%), severe 1 (1.85%) and mixed type 1 (1.85%) in the severe category. In >10 years of exposure, obstruction was 10(29.41%) in which mild 4 (11.76%), moderate 5 (14.70%), severe 1 (2.94%) and restriction was found 7 (20.58%) in which mild 4 (11.76%), moderate 3 (8.82%) and severe nil and mixed type of impairment was 3 (8.82%) in which 1 (2.49%) mild, 2(5.88%) moderate and severe nil.

Table 17: Shows exposure wise total lung function impairments and type of impairments which were found to be correlated with the duration of asbestos exposure in A-E industries. Total impairments were found in 100 (46.51%). The workers in <5 years of exposure sub-group showed 8 (57.14%) impairment out of which 8 (57.14%) found as restriction in which mild 4 (42.85%), moderate 2(14.285), severe nil. None was found as obstructive case. In 5-10 years of exposure category the impairment was 9 (29.03%) of which 1 (3.25%) as obstruction in moderate category but in restriction category impairment was 8 (25.80%) of which mild 5 (16.12%), moderate 3 (9.67%), severe nil. In >10 years of exposure category impairments was recorded as 83 (48.82%) of which 23 (13.52%) as obstruction in which 12 (7.05% mild, 10(5.88%) moderate and 1 (0.58%) severe. Restrictions was 60 (35.29%) in which 38 (35.52%) mild, 21 (12.35%) moderate and 1 (0.58%) severe.

Figure -15: Shows the radiological impairments in different industries of organized sector. Chest x-ray were performed in 300 subjects and 123 case (41%) found with radiological abnormalities. UPAL (I) showed 53 (59.77%) radiological impairments in which [chronic bronchitis 27 (31.03%), bilateral pulmonary tuberculosis 1 (1.14%), chronic obstructive pulmonary disease 18 (20.68%), chronic bronchitis with healed tuberculosis 5(5.74%), chronic obstructive pulmonary disease with diaphragmatic calcification 1 (1.14%), bilateral right side bronchitis 1 (1.14%)]. In UPAL (II) 55 (100%) x-ray were done and all were negative which might be due to least exposure as the factory is new. ANL showed the radiological impairments 35 (58.33%)of which [18 (30%) shows chronic bronchitis, 1(1.66%) bilateral pulmonary tuberculosis, 1 (1.66%) chronic bronchitis with cardiomagaly, 2(3.33%) healed tuberculosis with COPD, 1 (1.66%) left side pleural thickening, 4 (6.66%) pulmonary tuberculosis, 8 (13.33%) COPD. 35 (35.71%)]. Radiological impairments were recorded in A-E industries of which [1 case of cardiomagaly, 1 of healed tuberculosis, calcification in upper lung region, 22 reticulonodular patterns and 11 COPD].

Table-18: Shows the radiological impairments in factory workers of UPAL (I) and sub-grouped <5 years, 5-10 years and >10 years. Sub-group <5 years of exposure registered 6 (37.5%) including disease pattern 5 (31.25%) chronic bronchitis, 1 (6.25%) COPD. In 5-10 years of exposure category 5 (55.55%) chronic bronchitis, 1 (11.11%) COPD. In sub-group >10 years of exposure category 17 (27.41%) chronic bronchitis, 16 (25%) COPD, 1 (1.61%) COPD with diaphragmatic calcification, 5 (8.6%) COPD with pulmonary tuberculosis, 1 (1.61%) bilateral pulmonary tuberculosis, 1 (1.61%) bilateral right side bronchitis. Overall findings of UPAL (I) reported as 53 (50.96%) radiological impairments of which 27 (31.03%) chronic bronchitis, 18 (20.68%) COPD, 1 (1.14%) COPD with diaphragmatic calcification, 5 (5.74%) COPD with pulmonary tuberculosis, 1(1.14%) bilateral pulmonary tuberculosis, 1 (1.14%) bilateral right side bronchitis.

Table-19: Shows radiological findings of factory workers in UPAL (II) and none showed any kind of radiological abnormalities, which might be due to newer factory.

Table-20: Shows radiological findings of factory workers in ANL and sub-grouped <5 years, 5-10 years and >10 years of exposed groups. There was no radiologically positive findings in sub-group <5 years of exposure. In the 5-10 years of exposure the pattern was found as chronic bronchitis 11(34.37%) pulmonary tuberculosis 2 (6.25%), COPD 1 (3.12%), left side pleural thickening 1 (3.12%). In >10 years of exposure sub-group, radiological findings were recorded as 7 (25.92%) chronic bronchitis, 1 (3.70%) chronic bronchitis with cardiomegaly, 2 (7.40%) pulmonary tuberculosis, 7 (25.92%) COPD, 2 (7.40%) healed pulmonary tuberculosis with COPD and 1 (3.70%) bilateral pulmonary tuberculosis. Overall findings shows 35 (58.33%) radiological impairments in ANL of which 18 (30%) chronic bronchitis, 1 (1.66%) chronic bronchitis with cardiomegaly, 4 (6.66%) pulmonary tuberculosis, COPD 8 (13.33%), 1 (1.66%) left side pleural thickening, 2 (3.33%) healed pulmonary tuberculosis with COPD and 1 (1.66%) bilateral pulmonary tuberculosis.

Table-21: Shows radiological findings in A-E industrial workers and sub-grouped exposure wise. Radiological findings of various kinds were observed in 35 (35.71%). In the <5 years and 5-10 years of exposure sub-groups, no one was found with any type of radiological impairment. However in > 10 years of exposure category; 1 (1.31%) cardiomegaly, 22 (28.94%) RNP, 1 (1.31%) healed tuberculosis, 1 (1.31%) calcification in upper region of lungs, 10 (13.15%) COPD.

Figure -16: Shows the data on asbestos bodies detected in sputum samples collected from industrial workers of organized sector. A total of 244 (50.83%) sputum samples were collected out of which 41 (16.80%) showed positive findings.

Table-22: Shows prevalence of asbestos bodies in different factories sub grouping workers in <5 years, 5-10 years and >10 years. There was no sample of UPAL (I), UPAL (II), ANL showing asbestos bodies in any subgroups of exposure. Only A-E workers registered cases in all subgroups 3(25%), 4% (40%) and 34 (36.17%, in <5 years, 5-10 years, >10 years respectively.

Figure -17: Shows the acid-fast bacilli analysis in sputum samples collected from organized sector industrial workers. A total of 244 (50.83%) sputum samples collected out of which 4 (1.63%) shows positive findings in ANL 1 (2.85%) and 3 (2.58%) A-E industries.

Table-23: Shows the acid-fast bacilli data in sputum samples, exposure wise sub-group. Only in >10 years exposure category 1 (50%) and 3 (3.26%) were found with positive findings in ANL and A-E, respectively. Other subjects of UPAL (I), UPAL (II), <5 years, 5-10 years, >10 years and subgroups <5 years, 5-10 years in A-E sputum samples were negative.

Table-24: This table contains demographic data on the mean age; height and weight in the asbestos exposed and control (unexposed) populations of unorganized sector. Exposed population shows average age, height and weight 31 ± 8 years, 163.5 ± 8.4 centimeters, and 50.5 ± 8.7 kilograms of which males were 31 ± 8 years, 165.5 ± 7.5 centimeters, and 52.4 ± 7.92 kilograms and females 31 ± 8 years, 153.3 ± 4.6 centimeters, and 41.2 ± 6.6 kilograms. In control population average age, height and weight were 33 ± 8 years, 160 ± 8.2 centimeters, and 51.8 ± 10.1 kilograms wherein male were 34 ± 8 years, 168.7 ± 5.5 centimeters, and 58.8 ± 9.2 kilograms whereas females 31 ± 8 years, 156.0 ± 5.5 centimeters, and 47.5 ± 8.1 kilograms respectively.

Table-25: This table shows the prevalence of personal habits related in unorganized sector life style. There were smokers 37 (34.25%), alcoholic 11 (10.18%) and non-vegetarian 60 (55.55%) in exposed population. Control population showed the pattern as smoking 35 (25.92%), alcoholic 10 (7.40%) and non – vegetarian 102 (75.55%).

Figure -18: The fuel type pattern used for cooking and lighting by the population exposed to asbestos (unorganized) which is further described in in text for males and females. Wood was used as 88 (81.48%), males 73 (81.11%) and females 15 (83.33%); Wood \pm Animal dung 8 (7.40%), males 6 (6.66%) and females 2 (11.11%); Wood \pm Kerosene 2 (1.85%), male 2 (2.22%) and female nil; Wood \pm Gas 3(2.77%), male 3 (3.33%) and female nil; Kerosene 3(2.77%), males 2 (2.22%) and female 1(5.55%); Animal dung cake 1(0.92%)male 1(1.11%) and female nil; Combination (Wood \pm Animal dung cake \pm Coal \pm Crop residues kerosene \pm Electricity) 3 (2.77%); male 3 (3.33%) and female nil. Wood exposure was higher in exposed population as compared to control population. Similarly in control population Wood 90 (66.66%), males 36 (70.58%) and females 54 (64.28%); Wood \pm Animal dung 8 (5.92%), males, 5 (9.80%) and females 3 (3.57%); Wood \pm Kerosene 2 (1.48%) in male 1 (1.96%) and female 1 (1.19%); Wood \pm Gas 2 (1.48%), male nil and females 2 (2.38%); Kerosene 1 (0.74%, male nil and female 1 (1.19%); Animal dung cake 6 (11.85%), males 4 (7.84%) and females 12 (2.38%); Combination (Wood \pm Animal dung \pm Coal \pm Crop residues Kerosene \pm Electricity) 16 (11.85%), male 5 (9.80%) and females 11 (13.09%).

Figure -19: The clinical symptoms and signs of subjects of exposed and control populations are shown in this figure. The prevalence of clinical symptoms was higher in exposed population as compared to control population. Likewise, clinical signs were also higher in exposed subjects as compared to control population. There was cough 20 (22.22%), dyspnoea on exertion 20 (22.22%), hemeoptysis 5 (5.55%) and clinical signs as clubbing 25 (27.77%), crepitation 11 (12.22%), ronchi 3 (3.33%). In control population the trend of symptoms and signs was cough 20 (14.81%), dispnoea on exertion 16 (11.85%) m haemoptysis 4(2.96%) and clubbing 16(11.85%), crepitations 14 (10.37%), ronchi 3(2.22%).S

Table-26: Profiles of clinical symptoms and signs are shown exposurewise in asbestos exposed population of unorganized sector. Overall findings suggest that clinical symptoms and signs were found to be higher in >10 years of exposure group as compared to <5 years of exposure. Gender basis pattern in total male and female subpopulation, clinical symptoms were 32 (51.61%) in <5 years of exposure of which Cough 12 (19.35%), Dyspnoea on exertion 16 (25.80%), Haemoptysis 4 (6.45%). Clinical signs total 29 (46.77%) of which individual sign as Clubbing 15 (24.19%), Crepitations 10 (16.12%), Ronchi 4 (6.45%) in < 5 years subgroup. In 5-10 years of exposure total symptoms was as 10 (37.04%) of which Cough 4 (14.81%), Dyspnoea on exertion 6 (22.22%), Haemoptysis nil. Clinical signs total 12(44.44%) of which pattern was as Clubbing 8 (29.62%), Crepitations 4(14.81%), Ronchi 1(3.70%) in 5-10 yrs subgroup. Subgroup > 10 years of exposure total symptoms numbered 16 (84.21%) of which Cough 8(42.10%), Dyspnoea on exertion 7 (36.84%), Haemoptysis 1 (5.26%). Clinical signs total 12 (63.15%) of which Clubbing 8 (42.10%), Crepitations 4 (21.05%), Ronchi nil in >10 years subgroup. Total clinical symptoms in male was found in <5 years of exposure as 23 (46%) of which Cough 9 (18%), Dyspnoea on exertion 10 (20%), Haemoptysis 4 (8%). In males sub group < 5 yrs, clinical signs total 21 (42%) of which pattern was Clubbing 11 (22%), Crepitations 7 (14%), Ronchi 3 (6%). Sub group 5-10 years of exposure total, in male 8 (34.78%) of which Cough 4 (17.39%), Dyspnoea on exertion 4 (17.39%), Haemoptysis nil. Sub – group 5 – 10 years male clinical signs total 10 (43.47%) of which pattern was Clubbing 7 (30.43%), Crepitations 2 (8.69%), Ronchi 1 (4.34%). Sub group > 10 years of exposure total clinical symptoms was in male 14 (82.35%) of which Cough 7 (14.17%), Dyspnoea on exertion 6 (35.29%), Haemoptysis 1 (5.88%). The same sub group > 10 years males shared clinical signs total 9 (52.94%) of which pattern was as Clubbing 7 (41.17%), Crepitations 2 (11.76%), Ronchi nil. Total clinical symptoms in female was found in < 5 years of exposure as 9(75%) of which Cough 3 (25%), Dyspnoea on exertion 6 (50%), Haemoptysis nil and clinical signs total 8 (66.66%) of which pattern was as Clubbing 4 (33.33%), Crepitations 3 (25%), Ronchi 1 (18.33). In female sub group 5-10 years of exposure, clinical symptoms total as 2 (50%) of which Cough nil, Dyspnoea on exertion 2(7.40%), Haemoptysis nil and clinical signs total 3 (75%) of which pattern was Clubbing 1 (4.34%), Crepitations 2 (7.40%), Ronchi nil. In > 10 years of exposure subgroup total symptoms in females patterned as 2 (100%) of which Cough 1 (50%), Dyspnoea on exertion 1 (50%), Haemoptysis nil and clinical signs total 2 (100%) of which pattern was Clubbing nil, Crepitations 1 (50%), Ronchi (50%).

Table-27: Contains data on the lung function impairments in male and female subpopulations working in unorganized sector. In exposed population total profiles as FEV₁(L) 2.14 ± 0.75, FVC (L) 2.46 ± 0.75, FEF (L/s) 87.94 ± 19.21, PEF (L/m) 273.33 ± 121.31, FEV₁/FVC (%) 77.19 ± 25.62 wherein males with 2.25 ± 0.73, 2.56 ± 0.75, 89 ± 16.7, 277.71 ± 126.97 and 40.50 ± 8.92 and females with 1.60 ± 0.58, 1.96 ± 0.56, 82.66 ± 28.67, 251.44 ± 87.26 and 36.69 ± 17.20, respectively. Trends in control population total FEV₁ (L) 2.36 ± 0.88, FVC (L) 2.67 ± 1.03, FEF (L/s) 92.55 ± 15.79, PEF (L/m) 273.79 ± 90.97 FEV₁/FVC (%) 92.58 ± 39.99 while in males 2.81 ± 0.87, 3.34 ± 1.08, 87.49 ± 12.12, 331.13 ± 105.10 and 82.88 ± 11.25 and females 1.60 ± 0.58, 1.96 ± 0.56, 82.66 ± 28.67, 251.44 ± 87.26 and 36.69, respectively.

Figure–20: Shows status of lung function test (LFT) of asbestos exposed (unorganized sector) and control populations. Over all lungs function impairment including obstruction and restrictions were higher in exposed that control population. The overall impairments were 69(63.88%) in which obstruction 16 (14.81%) and restriction 53 (49.07%).

Figure 21: Exposed population was sub grouped exposure wise in <5 years of exposure, 5-10 years of exposure and >10 years of exposure. In <5 years of exposure obstruction was 9 (14.51%) [mild 7(11.29%), moderate 2 (3.22%), severe nil] and restriction was 29(46.77%) [mild 15 (24.19%), moderate12 (19.35%), severe 2(3.22%)]. In 5-10 years of exposure obstruction was 4(14.81%) [mild 1 (3.70%), moderate 2(7.40 %), severe 1 (3.70%)] and restriction 15 (55.55%) [mild 9 (33.33%), moderate 5 (18.51%), severe 1 (3.70%)]. In >10 years of exposure obstruction was 3 (15.78%) [mild nil, moderate 1 (5.26%), severe 2 (10.52%)] and restriction was 9 (47.36%) [mild 7 (36.84%), moderate 2 (10.52%), severe nil].

Figure -22: The total radiological impairments in exposed population of unorganized sector are shown in figure 22 and table 28. There were 28(47.45%) impairments in exposed population which was significantly high than control population.

Table-28: Shows the radiological impairments exposurewise in unorganized sector. In <5 years of exposure category total impairments was as 17(51.51%) of which RNP 6 (18.18%), pulmonary tuberculosis 2 (6.06%), IBVM 9 (27.27%). In 5-10 years of exposure total was 6 (35.29%) of which RNP 1 (5.88%), IBVM 4 (23.52%), Plaque 1 (5.88%). In >10 years of exposure, total impairments was 5 (55.55%), bronchiostasis 2 (22.22%), IBVM 2 (22.225), Calcification in left side diaphragm 1 (11.11%). These data with respect to male and female workers exposurewise are also presented in this table.

Table-29: The HRCT of few cases suspected in the radiological examinations and on the basis of those findings seven cases noticed to be examined for HRCT but at the time of examination only six cooperated. The prevalence was found as 2 (33.33%) in <5 years, 1 (16.66%) in 5-10 years, and 3 (50%) in >10 years of asbestos exposure.

Table-30: Shows asbestos bodies analysis in sputum of asbestos exposed (unorganized sector) and control population. Out of 49 (45.37%) collected samples from exposed population 12(24.48%) was found positive of which 11 (26.82%) from male and 1 (12.5%) from female population. In control population 67 (49.62%) collected samples no one was found with asbestos bodies. The prevalence of asbestos bodies in sputum samples in exposed population was 24.48% compared to none (0.0%) in control population.

Table-31: Asbestos exposed population is subgrouped in <5 years, 5-10 years and > 10 years in unorganized sector. Sputum samples were collected in 49 workers (56.32%), 27 (43.54%) in < 5 years, 11(40.74%) 5-10 years, 11(57.89%) in >10 years. The prevalence of asbestos bodies presence were 7 (25.92%) in <5 years, 1 (9.09%) in 5-10 years and 4 (36.36%) in > 10 years.

Table-32: Shows the acid-fast bacilli analysis in sputum of asbestos exposed population (unorganized) and control population. Samples collected was a total of 49 (45.37%) in asbestos exposed population of which 9 (18.36%) found positive in male only and no one in female. In control population 67 (49.62%) showed positive males 7 (24.13%) and females 5 (13.15%).

Table-33: Data on (unorganized sector) exposurewise prevalence of acid fast bacilli in subgroups <5 years, 5-10% years and >10 years are shown. Acid-fast bacilli in sputum 49 (45.37%) collected samples, 5(17.85%) in <5 years, 3(27.27%) in 5-10 years, 1(10%) in > 10 years was detected.

Table-34: A comparative demographic profiles of workers in unorganized as well as organized sectors have been shown in Table 34. Age and weight were found higher in organized sector than unorganized sector. In the unorganized sector, population average age, height and weight were recorded as 31 ± 8 years, 163.5 ± 8.4 centimeters and weight 50.5 ± 8.7 kilograms. Gender wise distribution with respect to age, height and weight as 31 ± 8 years, 165.5 ± 7.5 centimeters, and 52.4 ± 7.92 kilograms in males and 31 ± 8 years, 153.3 ± 4.6 centimeters, 41.2 ± 6.6 kilograms in females, respectively. The average age, height and weight in organized sector recorded as 39.1 ± 10.3 years, 164.8 ± 6.3 centimeters and 59.5 ± 10.6 kilograms, respectively. Comparison of males in unorganized and organized sectors, average age and weight were found to be higher in organized sector.

Table-35: The personal habits related to the individual life styles prevalence was found as smoking 37(34.25%), alcoholic 11(10.18%), non-vegetarian 60 (55.55%) in unorganized sector while individually male and female showed a pattern as smokers 34 (37.77%), alcoholic 10 (11.11%), non-vegetarian 53 (58.88%) in males and females showed smoking 3 (16.66%), alcoholic 1(5.55%), non-vegetarian 7 (38.88%). In the organized sector, prevalence of smoking was 102(21.25%), ex-smokers 1 (0.20%), tobacco chewers 18 (3.75%), alcoholic 98(20.41%) and non-vegetarian 322(67.08%).

Figure 23: This figure shows the fuel type pattern use during cooking and lighting by the individuals exposed to asbestos in unorganized sector and organized sector. Wood and animal dung cake users were higher in unorganized sector than organized sector while gas users were maximum in organized sector than unorganized sector. In unorganized sector, prevalence of fuel type used was wood 88 (81.48%), male 73

(81.11%) and female 15 (83.33%); wood + animal dung cake 8 (7.40%), male 6 (6.66%) and female 2 (11.11%); wood + kerosene 2 (1.85%) male 2 (2.22%) and female nil; wood + LPG 3 (2.77%), male 3(3.33%) and female nil; Kerosene 3(2.77%), male 2 (2.22%) and female 1 (5.55%); animal dung cake 1(0.92%), male 1 (1.11%) and female nil; Combination (wood + animal dung cake + coal + crop residues + kerosene + electricity) 3 (2.77%), male 3(3.33%) and female nil. In organized sector exposed individuals consuming fuel for cooking and lightning purposes used wood 11 (2.29%), wood + animal dung 2 (0.41%), wood + kerosene 26 (5.41%), wood + gas 12 (2.5%), gas 208 (43.33%), kerosene 17 (3.54%), animal dung nil and mixture of fuels (wood, animal dung, crop residue, electricity) as 204 (42.5%).

Figure -24: This shows profiles of clinical symptoms and signs of exposed individuals in unorganized and organized sectors. Overall symptoms and signs found to be higher in unorganized sector than organized sector. Total clinical symptoms in exposed population in organized sector (males) was found as 45 (50%) of which cough 20 (22.22%), dyspnea on exertion 20 (22.22%), hemeoptysis 5 (5.55%) and total clinical signs were recorded as 39 (43.33%) of which clubbing 25 (27.77%), crepitation 11 (12.22%), ronchi 3 (3.33%). The total symptoms and signs in unorganized sector were found as 58 (53.70%) of which cough 24 (22.22%), dyspnoea on exertion 29(26.85%), haemoptysis 5(4.62%) and clinical signs 53 (49.07%), clubbing 31(28.70%), crepitation 18 (16.66%), ronchi 4 (3.70%).

Figure -25: Shows the exposure wise distribution of clinical symptoms and signs of the workers in unorganized sectors. In <5 years of exposure, clinical symptoms showed as cough 12 (19.35%), dyspnoea on exertion 16 (25.80%), haemoptysis 4 (6.45%) and clinical signs as clubbing 15 (24.19%), crepitations 10 (16.12%), ronchi 4 (6.45%). In 5-10 years of exposure, clinical symptoms recorded as cough 4 (14.81%), dyspnoea on exertion 6 (22.22%), haemoptysis nil and clinical signs as clubbing 8 (29.62%), crepitations 4 (14.81%) and ronchi 1 (3.70%). In >10 years of exposure subgroup, clinical symptoms recorded as cough 8 (42.10%), dyspnoea on exertion 7 (36.84%), haemoptysis 1 (5.26%) and clinical signs as clubbing 8 (42.10%), crepitations 4 (21.05%) and ronchi nil. Over all findings suggest the symptoms found higher in >10 years of exposure and signs mostly equal in unorganized sector. Overall findings also show increase in symptoms and signs with increase in duration of exposure.

Figure-26: Status of clinical symptoms and signs prevalent in organized sector exposurewise are shown in figure-26. Clinical symptoms <5 years of exposure as cough 8 (9.19%), dyspnoea on exertion 8 (9.19%), haemoptysis 2 (2.29%) and clinical signs as clubbing 1 (1.14%), crepitations 5 (5.74%) and ronchi 1 (1.14%). In 5-10 years of exposure clinical symptoms found as cough 27 (23.47%), dyspnoea on exertion 17 (14.78%), haemoptysis 2 (1.73%) and clinical signs as clubbing 9 (7.82%), crepitations 14 (12.17%) and ronchi 7 (6.08%). In subgroup >10 years of exposure, cough 77 (27.69%), dyspnoea on exertion 80 (28.77%), haemoptysis 15 (5.39%) and clinical signs as clubbing 53 (19.06%), crepitations 50 (17.98%) and ronchi 16 (5.75%).

Table-36: Lung function impairments in male and female of asbestos exposed unorganized and organized population are shown in table-36. In unorganized sector population total FEV (L) 2.14 ± 0.75 , FVC (L) 2.46 ± 0.75 , FEF (L/S) 87.94 ± 19.21 , PEF (L/min) 273.33 ± 121.31 , FEV1/FVC (%) 77.19 ± 25.62 while in males these values were 2.25 ± 0.73 , 2.56 ± 0.75 , 89 ± 16.7 , 277.71 ± 126.97 and 85.28 ± 18.30 and in females values were 1.60 ± 0.58 , 1.96 ± 0.56 , 82.66 ± 28.67 , 251.44 ± 87.26 and 81.63 ± 17.20 , respectively. In organized sector, the pattern was found as FEV1 (L) 2.23 ± 0.65 , FVC (L) 2.67 ± 0.77 , FEF (L/s) 81.58 ± 19.33 , PEF (L/min) 318.82 ± 135.44 , FEV1/FVC (%) 83.59 ± 22.49 .

Figure -27: The prevalence of lung function test (LFT) has been shown in figure 26. Over all LFT profiles were generally lower in organized sector than the unorganized sector. The over all LFT impairments and obstructions as well as restriction profiles were higher in unorganized sector than organized sector. The total impairments in unorganized sector was 69 (63.88%) of which 16 (14.81%) obstruction and 53 (49.07%) restriction and mixed type was nil. The organized sector population shows the over all lung function impairments total 193 (40.20%) of which 67 (13.95%) obstruction, 114 (23.75%) restriction, 12 (2.70%) mixed type.

Figure -28: Exposure wise profiles of LFT impairments have been shown in <5 years, 5-10 years and >10 years sub groups in unorganized sector. The exposure wise distribution pattern of lung function impairments in <5 years of exposure, obstruction recorded 9 (14.51%) while restriction found as 29 (46.77%). In 5-10 years of exposure total obstruction shows 4 (14.81%) while restriction found as 15 (55.55%). In >10 years obstruction 3 (15.78%) while restriction found 9 (57.36%).

Figure -29: Shows the exposure wise distribution pattern of lung function impairments in organized sector population. In <5 years of exposure recorded impairment recorded as obstruction 8 (10%) [mild 6 (7.5%), moderate 2 (2.5%) and severe nil] and restriction as 21 (26.25%) [mild 14 (17.5%), moderate 7 (8.75%) and severe nil no one was in mixed type of category]. In 5-10 years of exposure, impairment recorded as obstruction 9 (7.82%) [mild 5 (4.34%), moderate 4 (3.47%) and severe nil] and restriction as 16 (13.91%) [mild 10 (8.69%), moderate 5 (4.34%) and severe 1 (0.86%)]. Mixed type impairments 5-10 years subgroups shows as 3 (2.60%) of which mild 2 (1.73%), moderate nil and severe 1 (0.86%). In > 10 years of exposure, impairment was recorded as obstruction 50 (17.98%) [mild 22 (7.91%), moderate 21 (7.55%) and severe 7 (2.51%)] and restriction 77 (27.69%) [mild 47 (16.90%), moderate 27 (9.71%) and severe 3 (1.07%)]. Mixed type impairments showed 9 (3.23%) in which mild 5 (1.79%), moderate 2 (0.71%) and severe 2 (0.71%).

Figure -30: Radiological impairments in asbestos-exposed unorganized and organized sector populations are shown in figure-30. The maximum impairments were found in unorganized sector than organized sector.

Table-37: Findings on radiological impairments population in asbestos exposed unorganized sector are shown in table 37, in an exposure-dependent manner. In <5 years of exposure subgroup, total impairments was 17 (51.51%) of which pulmonary TB 2 (6.06%), RNP 6 (18.18%), IBVM 9 (27.27%). In 5-10 years of exposure, total impairments was 6 (35.29%) of which RNP 1 (5.88%), IBVM 4 (23.52%), Plaques 1 (5.88%). In >10 years of exposure, total impairments was 5 (55.55%) of which brochiectasis 2 (22.22%), IBVM 2 (22.22%), calcification in left side diaphragm 1 (11.11%).

Table-38: Radiological observations on exposed population in organized sector are sub grouped exposure wise as <5 years, 5-10 years and >10 years (Table-38). In <5 years there were 17 (51.51%) positive findings of which 6 (18.18%) RNP, 2 (6.06%) pulmonary TB and 9 (27.27%) IBVM. In 5-10 years exposure subgroup, there were 6 (35.29%) positive cases of which 1 (5.88%) RNP, 4 (23.52%) IBVM and 1 (5.88%) plaque. In >10 years exposure sub group, there were 5 (55.55%) positive cases of which 2 (22.22%) brochiectasis, 2 (22.22%) IBVM and 1 (11.11%) calcification in left side diaphragm.

Table-39: Data on asbestos bodies have been sub grouped exposure wise in unorganized sector population (table 39). Out of total 49 (56.32%) collected samples, 28 (43.54%) in <5 years, 11 (40.74%) in 5-10 years, and 11 (57.89%) in >10 years. Positive samples were 5 (17.85%), 3 (27.27%) and 1 (10%) in subgroup <5, 5-10 and >10 years respectively.

Table-40: Data on asbestos body in sputum of organized sector have been sub grouped exposurewise (Table 40). A total of 244 (50.83%) collected samples only 41 (16.80%) showed positive findings and categorized as 3 (6.12%), 4 (6.12%) and 34 (21.89%) in <5 years, 5-10 years and >10years of exposure, respectively.

Table-41: Shows data on the acid-fast bacilli in sputum samples in exposurewise subgroups of unorganized sector.

Table-42: Data on acid fast bacilli in sputum of exposed population in organized sector, exposure wise have been shown in table 42. Out of total 244 (50.83%) collected sputum samples, 4 (2.56%) in >10 years found to be positive but no one was positive in <5 years and 5-10 years of exposure category.

CHAPTER 5.0

DISCUSSION

Study on asbestos counts in work environment of asbestos based industries is the most vital approach for the human risk assessment. The project entitled “Human health risk assessment studies in asbestos based industries in India”, therefore special focus was made on the industrial hygiene, however, the approach was extended on other aspects too such as clinical and chest radiological examinations, personal and occupational history of the asbestos-exposed population for epidemiological assessment.

Looking at the scenario of asbestos-exposed industries in India, it is clear to sub-group them into organized and unorganized sectors. Asbestos-based unorganized sector includes, firstly, asbestos rock grinding units (asbestos mills) and secondly, small and household units manufacturing a variety of products using locally milled asbestos and cement. A general survey of unorganized asbestos units made it clear that industrial hygiene conditions are really very poor in asbestos mills, therefore asbestos mills were given study focus in particular. Such asbestos mills are predominantly located in Rajasthan, particularly in Deogarh and Beawer which might be due to their rich natural sources of asbestos (Mansinghka and Ranawat, 1996; Dashawara and Tyagi, 1992).

These asbestos mills were monitored for asbestos fiber counts at their work environment. Appropriate replicates of samples, using personal and area sampler, were collected in order to assess the prevalence of fibers during the work shift. These observations with respect to asbestos concentration and exposure period are very helpful in occupational health risk assessment. Mean asbestos fiber counts in these unorganized asbestos mills were 4.07-15.6 f/cc at Deogarh and 2.0-5.09 f/cc at Beawer. Invariably, the fiber concentrations monitored by area sampler were always higher than that of personal sampler. Briefly, asbestos fibre concentration in the work environment of all the asbestos mills, both at Deogarh and Beawer, exceeded the permissible standard of 1 f/cc. It is beyond doubt that workers in asbestos mills in Rajasthan are at serious risk of asbestos exposure. Studies both light microscopic and electron microscopic showed these asbestos fibers are amphiboles. It is an established fact that amphiboles are much more toxic, asbestotic and carcinogenic than serpentine (Armstrong et al 1998; Barrette, 1994; Luce et al 2000; McDonald et al 1999). These observations further enhanced the serious risk of asbestos exposure in asbestos mills in Rajasthan.

As compared to the unorganized sector, industrial hygiene conditions were satisfactory in organized asbestos industries engaged in the production of roofing sheets, yarn, clothes, ropes, brake linings, brake-shoes etc. These asbestos-based industries of organized sector were found with work place asbestos fibers in the range of 0.057-1.66 f/cc. All the industries registered fiber counts less than 1 f/cc (Indian Standard) except one (Hindustan Composite Pvt Ltd., Aurangabad, Maharashtra, having 1.66 f/cc in its

debugging section). But it is essentially needed that all the organized asbestos industries must comply with the Indian Standard (1 f/cc). It seems to be achievable by proper maintenance and operation of pollution control measures, if needed by replacing these control devices by improved model of pollution control system. By repeated trials, these can be assessed in order to ensure the compliance of Indian standard round the shift and every shift of the industries. Asbestos industries need to show more responsibilities towards the occupational and environmental health. During the survey studies in organized industries, it was found that about 10 % of workers did not put face masks and the rest (about 90% put fresh face masks). Workers should not be permitted to enter the work zone without putting on face masks. There is a need of awareness on this issue which is most important for occupational safety.

From the questionnaire survey, it is evident that quite a good portion of industrial workers are smokers and using biomass fuel for their domestic needs. These workers should be counseled for stopping smoking and using biomass fuel for domestic use. If required, some arrangements should be made towards their availability of cooking gas. These two factors (smoking and exposure to biomass fuel) are known to accelerate the toxic effects due to asbestos exposure (De-Klerk et al 1991; Doll and Peto 1976; Eastman et al 1983; Kampet et al 1998; Selikoff et al 1968).

In India, small-scale as well as large scale asbestos-based industrial units are located in different states. Asbestos grinding is mainly concerned to small-scale units. Most of the industries from organized sector were found to be involved in the manufacturing of asbestos-based products mainly AC sheets and asbestos yarn during the survey. It is estimated that asbestos industry directly gives employment to over 6,000 workers and another 1, 00,000 peoples indirectly depend on both organized and unorganized sectors. About 60% of them are not adequately aware of the consequences of asbestos exposure and its adverse health effects. It is reported that humans may be exposed to asbestos from a variety of sources, occupational settings, ambient environment and consumer products (USEPA,1985). Exposure to asbestos causes various lesions ranging from simple non-malignant inflammatory reactions, pleural thickening and asbestosis to malignant mesothelioma and brochogenic carcinoma (Byers et al., 1984; Craighead et al., 1993; Deklerk 1989a; Earnster et al., 1994; Kelburn 2000; Mossman and Gee 1989; Mossman 1994; Raffin et al., 1993; Mossman et al., 1990a). Present study on organized and unorganized sectors have been grouped in chapters containing individual profile of asbestos exposed population from individual industries of organized sectors, comparative account of asbestos-exposed and control population in organized sectors, comparative account of asbestos-exposed and control population in unorganized sectors, comparative account of asbestos-exposed organized and unorganized sectors population. Findings have shown associations of various demographical characteristics, clinical symptoms, clinical signs, lung function status, radiological lesions and sputum cytology, and asbestos bodies in population occupationally exposed to asbestos and domestically exposed to unprocessed biomass fuels.

Our study findings show variations in the personal habits in all the asbestos exposed individuals that might be due to their life style pattern. Cigarette smoking and asbestos exposure have additive or synergistic interactions in inducing cancer of the lung (Mossman and Gee Yano and Sone, 2000, 1989). The combination of tobacco smoking and asbestos exposure leads to an enormously high incidence of bronchogenic carcinoma (Selikoff and Lee, 1978; Selikoff et al 1980). About 35 % workers were smokers in asbestos-based industries belonging to organized and unorganized sectors. Mostly bronchogenic carcinomas are common among smokers exposed to asbestos (Saracci et al., 1977, Hammond et al., 1979; David et al., 1999). Smoking has been shown to increase the risk of asbestosis too (Schulz. 1994). Asbestos and cigarette smoke both are complex carcinogens affecting more than one stage of carcinogenesis (Vaino and Boffetta, 1994). The information available on the biochemical effects of asbestos with predisposing factors is also known (Ahmad et al 1994; Arif et al 1987, 1993a, b, 1992, 1993). Several epidemiological and experimental studies have proven that the presence of some other predisposing factors like exposure to cigarette smoke, kerosene soot and biofuels smoke at indoor levels would accelerate the asbestos induced disease processes (Arif et al 1987, 1993a, b, 1992, 1993). Some factors such as smoking, occupational exposure to particulate dusts, asbestos fibers suspended in the air, and random gas absorbed in dust particles have been implicated in lung pathology (Rahman 1995; Rahman et al 2000). There is considerable evidence from radiological studies that smoking increases the attack or and, the progression rate for asbestosis (Bamhart et al., 1990). Presence of clinical symptoms and signs were well-documented characteristics of asbestos exposed population . Two factors, magnitude and duration of asbestos exposure play an important role in the asbestos related diseases . Several studies proved that workers with asbestos exposure manifest the disease (asbestosis) and may have finger clubbing and in advanced stages show signs of core pulmonale (Frazer et al., 1990) where as our findings reveal the presence of clubbing suggestive of characteristic as asbestos exposure . Lung function tests were found correlated with the duration of exposure and industrial hygiene conditions and these findings are parallel to that of (Brown K, 1994) reporting usual functional changes in the fully developed case of asbestosis as restrictive defect and decreased diffusing capacity. Presence of pleural plaques was prominent and that is considered marker of asbestos exposure in both occupational and environmental (Albelda et al., 1982). Asbestos bodies were reported as 41% in asbestos exposed population that is the hallmark of asbestos exposure while no one was in the control population , as they were not exposed to asbestos . In case of acid-fast bacilli prevalence was reported as 2.53% in asbestos exposed population may be due to the indoor exposure of biomass smoke.

Several asbestos samples collected in Rajasthan were tested for their genotoxic potential through MN induction and SCE. These indigenous asbestos produced enhanced MN and SCE registering their genotoxic nature. Different genotoxic activity of these samples could be attributed to the asbestos content and different mechanism (s) responsible for these events. Peripheral blood samples were also collected from the asbestos exposed population to access genotoxicity through micronuclei (MN) formation. Micronuclei levels were significantly higher in exposed population in comparison with unexposed population. Interestingly, MN levels correlated with the

duration of occupational exposure as well as smoking habit. Elevation in the MN induction frequency and chromosomal aberration in the asbestos exposed groups clearly consolidated that asbestos exposure may induce undesirable genetic damage in occupational population. The data reveal that exposed population is vulnerable for lung cancer, as MN is a biomarker for cancer. The higher number of induced MN observed in case of exposed smokers which is clearly indicating the synergistic interaction between cigarette smoke and asbestos exposure, which is in agreement to the some studies (Berry et al., 1985; Okayasu et al 1999; Liddell et al., 1984; Selikoff et al., 1980). The elevation of the genetic damage in case of unexposed smokers is also in accordance with the earlier reports (Kamp et al., 1998; Deklerk et al., 1991a; Yano et al., 1993). Higher genetic damage in the case of asbestos exposed smokers groups in comparison to the exposed non smokers group consolidated that smoking may act synergistically with asbestos to enhance the risk of lung cancers in the exposed smokers group. These results are analogous to the lung cancer risk among the insulators where the vast majority of cases occur in those insulators who smoke also.

Types of biomass used were found to be common in both asbestos-exposed and unexposed populations.

Use of unprocessed biomass fuel was higher in control population because of the economy and easy availability to exposed and unexposed populations. Clinical symptoms and signs were higher in exposed populations as compared with reference population which also correlated with the duration of asbestos exposure. Lung function tests (LFT) show higher impairments in exposed as compared to control one while restriction was also more in exposed population as reported by others (Becklake, 1976; Rastogi et al., 1990). Ten previous studies reported higher obstruction in control population than exposed while others suggested a correlation between domestic fuel exposure and acute respiratory infections as well as chronic obstructive lung diseases. Present results show higher obstruction in exposed population than control one that might be due to smoking prevalence. In our study, effect of asbestos on exposed population found to be accelerated in the presence of tobacco smoking (Selikoff et al., 1980; Liddell et al., 1984; Berry et al., 1985). Some studies have also demonstrated that cigarette smoke inhibits asbestos clearance (McFadden et al., 1986, Hobson, 1988) from the lung. Exposure wise LFT as obstruction and restriction did not show much difference in our findings. Radiologically examined population shows higher impairments in exposed than control one increasing with increase in duration of asbestos exposure. Left side pleural thickening was found only in exposed population but none in control population. Wang et al., (2001) also recognized more frequently radiological pleural abnormalities in exposed workers. Asbestos bodies were found in exposed one as earlier mentioned but no case was available in control population. (AFB) was detected in sputum samples of exposed population averaging 1.65% that may be due to exposure to smoke of unprocessed biomass fuels and surrounding environment.

Demographic profile from the exposed and control population for unorganized sector, gender wise, showed much variation. Overall smoking and alcoholic habits and prevalence of wood exposure found higher in exposed population except animal dung which was more prominent in control population that may be due to easy availability and poor economic condition.

Several studies of asbestos workers have shown that cigarette smokers compared to non-smokers have higher risk of developing pulmonary asbestosis, laurel lesions, clinical symptoms and reduced lung function (Richter et al., 1986 and Samet et al., 1979). Clinical symptoms and signs were recorded more in exposed populations practically in subgroup >10 years of exposure as documented by other studies too that magnitude and duration showed correlation with clinical parameters. Other factors namely smoking, kerosene soot may accelerate the disease process in asbestos exposed workers (Arif et al 1997; Lohani et al., 2000). LFT showed higher degree of impairments in exposed population as compared to control in our study. Dave et al., (1996) in exposed population, reported restriction higher than control population. Asbestos is a complete carcinogen in the lung, but a multiplicative effect on lung cancers in human is observed with cigarette smoke and asbestos exposure (Selikoff et al., 1986). Findings from the radiological examination also shows the higher incidence of impairments in exposed population which is in agreement to the study conducted by Dave et al., (1996). On the basis of radiological Findings, few cases were further examined by the high resolution computed tomography (HRCT) confirming asbestosis that shows the alarming situation in the unorganized sector exposing workers to amphiboles especially tremolite . HRCT is an advanced tool for more accurate diagnosis of pneumoconiosis (Suganuma et al., 2001). Variation in counts of asbestos bodies depends on the sample size and duration of exposure. Data on the exposed population of unorganized and organized sectors showed variation unabling to draw a positive inference with respect to degree and duration of exposure of asbestos and its types. Our findings shows the prevalence of smoking habits higher in unorganized sector than organized one although unorganized workers were exposed to amphiboles especially tremolite .Exposure to tremolite has been shown to cause respiratory cancer , where smokers are found to be at nine fold higher risk as compared to exposed non-smokers (Luce et al., 2000). The workers engaged in unorganized units work without using gloves and masks. There are reports that asbestos fibers could penetrate the skin too (Alden and Hpowell.1994). Processing of asbestos grinding, manufacturing of asbestos-based products in unorganized units, their poor life conditions and domestic exposure to smoke of unprocessed biomass fuels reflect their pity health scenario. Asbestos exposure and cigarette smoking acts synergistically to produce dramatic increases in lung cancer compared with those from exposure to either asbestos only (U.S.EPA, 1984). Synergistic interaction between cigarette smoke and asbestos exposure induce genetic damage in asbestos exposed population (Selikoff et al., 1980; Liddel et al., 1984).

Clinical symptoms and signs alongwith improved LFT were higher in unorganized sector than organized sector. Shortness of breathe, accompanied by rales and cough are already documented which in severe cases may prove fatal (ATSDR, 1993). Exposure wise prevalence of symptoms and signs, obstruction and restriction was higher in unorganized sector which might be due to heavy exposure of asbestos. The overall LFT pattern was not similar to organized sector . Radiological lesions were more in unorganized sector while exposure wise trends show variation even in organized sector however, trends generally correlated with duration of exposure. Sputum cytology reveals higher number of asbestos bodies and acid fast bacilli in unorganized sector

indicating very poor industrial hygiene and exposure of biomass fuel during the cooking, heating and lighting. Presence of asbestos bodies (AB) in sputum of individuals with occupational asbestos exposure has been documented and positive sputum samples for AB suggest high lung asbestos burden (Teschler et al., 1996).

Asbestos exposed populations, industry wise, have been fully described, in results, relating to their personal and occupational histories, asbestos bodies counts clinical symptoms and clinical signs, lung function profiles and radiological lesions. Data on sputum asbestos bodies are found suggestion of (a) it is not always a positive indicator of asbestos exposure in industrial works and (b) it does not invariably shows a positive correlation with the duration of asbestos exposure. Presence of asbestos bodies in sputum is certainly an indicator of asbestos exposure but its absence should carefully be assessed therefore for concluding no exposure to asbestos. Clinical symptoms and signs were generally higher in exposed population as compared with unexposed population. Lung function profiles, as compared with unexposed population, recorded in unorganized industrial units as well as organized industries showed impairments (restriction, obstruction) and their correlation with the occupational exposure and ratio duration. Chest x-rays were examined with respect to chronic bronchitis, COPD (chronic obstructive pulmonary disease), COPD with diaphragmatic calcification, COPD with pulmonary TB (tuberculosis), bilateral TB, bilateral right side bronchitis, reticular nodular pattern (RNP), increased broncho-vascular markings, plaque and calcification in left side diaphragm. Radiological lesions in asbestos - exposed population showed their association with asbestos exposure and its duration.

CHAPTER 6.0

RECOMMENDATIONS

In the light of the present study, the following recommendations are made to reduce the human risk of asbestos exposure and its health hazard:

1. All the workers should be provided with medical surveillance by the employer. Medical surveillance programme should consist of the following:

- Pre- employment medical surveillance
- Periodic medical examination
- Medical examination at cessation of employment
- Maintenance of medical records; and
- Health education

Medical examination record should be maintained and stored for a period of 10 years following the termination of employment, or for 40 years after first day of employment, whichever is later.

The Medical Doctor appointed for examining the workers should be trained in Occupational Health. This will facilitate early detection of occupational related diseases in the workers.

In order to comply the recommendations contained in IS:11451-1986, employer should stop existing practice of employment of temporary, contract workers or daily wage workers.

2. The Ministry of Mines had decided to continue the ban on mining of amphibole variety of asbestos. By the time this ban is implemented in totality, following measures are required to be taken by the asbestos milling units of Rajasthan.
 - Preferably complete plant machinery requires to be redesigned with proper engineering controls to reduce asbestos fibre emission.
 - Manual handling of asbestos should be reduced to minimum.
 - Fugitive emissions generated needs to be controlled.
 - Asbestos bearing stones should be stored in a covered shed to avoid fugitive emission.
 - All material transfer point should be connected to dust extraction system.
3. Floor should be cleaned by vaccum cleaner only.
4. Workers likely to get exposed to asbestos should wear protective clothing and respiratory equipment.
5. The asbestos based product manufacturing industries should operate and maintain the air pollution control devices properly so as to comply with the emission standards of 0.2 fibre / cc for asbestos fibre and 2 mg / Nm³ for total dust. The monitoring should be carried out regularly and data should be submitted to State Pollution Control Board and Central Pollution Control Board.
6. The asbestos fibre concentration at work place should not exceed 1 fibre / cc, as per Factories Act, 1948.

7. The asbestos based units should get asbestos fibre monitoring done on regular basis.
8. All the organized and unorganized industries should ensure good house keeping practices to reduce the asbestos exposure.
9. Work place asbestos standards should be brought down from 1 fibre/cc to 0.1 fibre/cc under the Factories Act, 1948 to reduce the risk of asbestos exposure.
10. Workers should be given education about the risk associated with asbestos dust exposure, potential health effects, etc.
11. Display board should be provided showing the hazards associated with asbestos and recommended precautionary measures.

Table-1

Asbestos fiber monitoring in unorganized sector at Deogarh, Rajasthan

Sl. No	Organized asbestos units	Sampling Location	Type of sampling	No. of samples	Fibre Concentration (fiber/cc)	
					Mean (fiber/cc)	Range (fiber/cc)
1.	B. K. Grindings Pvt. Ltd.	Grinding	Personal	6	11.8	6.0-18.2
2.	Kanchan Minerals Pvt. Ltd.	Grinding	Personal Area	3 3	4.07 4.96	3.4-4.3 2.7-7.5
3.	Maharaja Asbestos Grinding Mills Pvt. Ltd.	Grinding	Personal	2	6.07	3.3-6.9
4.	Oswal Minerals Trading Corporation	Grinding	Personal	3	15.6	12.5-17.6

Table-2**Asbestos fiber counts in unorganized asbestos grinding units at Beawer, Rajasthan**

Sl. No	Unit	Place of sampling	Type of sampling	No. of sample	Fibre Concentration (fiber/cc)	
					Mean (f/cc)	Range (f/cc)
1.	Cenera Minerals Pvt. Ltd.	Grinding	Personal	4	5.09	2.09-8.06
2.	Guru Asbestos Pvt. Ltd.	Grinding	Personal	4	4.67	1.48-8.91
3.	Gajanand Cement Asbestos Products Pvt. Ltd.	Grinding	Personal Area	4 2	2.96 4.36	1.57-3.59 4.12-4.51
4.	Kamala Minerals Pvt. Ltd.	Grinding	Personal	4	2.8	2.30-3.55
5.	Super Minerals Pvt. Ltd.	Grinding	Personal Area	2 4	2.0 4.6	1.71-2.29 1.24-3.15
6.	Swastic Udhyog Pvt. Ltd	Grinding	Personal	6	3.34	1.64-4.62

Table 3

Identification of asbestos variety in indigenous asbestos samples in unorganized sector, Rajasthan.

Identification of fibers	
Sample	Composition*
1	100 % Tremolite
2	100 % Tremolite
3	85% Tremolite, 10% Talc and 5% anthophyllite
4	30 % Tremolite, 55 % Talc and 15% MgAlSiFe
5	90 % Tremolite, 10 % Talc

* Composition determined by XED, analytical electron microscope by Prof. Arthur L. Frank, USA.

Table 4
Asbestos fiber count in UPAL(II), Nagpur

S. No.	Site	Asbestos fibre count (Fibre/cc)
1.	Ingredient feeding	0.080
2.	Sheet producing	0.057
3.	Fibre godown	0.078
4.	Factory gate	0.072

Fibre/cc = Fibre/cubic centimeter

Table –5
Asbestos fiber monitoring in organized sector at Mumbai (Maharashtra)

Industry	Name of products	Asbestos Concentration in key industrial sections (fiber/cc)						
		Yarning	Calendaring	Weaving	Combined Dust Collector	Fibre Dust collector	Debagging	Mixing of waste products
A	yarn, ropes	0.188 (0.132-0.214) [4P]	-	-	-	-	-	0.206 (0.199-0.214) [2A]
B	yarn, its products	0.146 (0.089-0.203) [2P]	0.212 [1P] 0.251 (0.094 – 0.490) [2A]	0.257 (0.257-0.257) [2P]	-	-	-	-
C	packing, Seals, yarn	0.171 (0.126-0.221) [3P] 0.215 (0.149 – 0.287) [2A]	-	-	-	-	-	-
								Contd.....

D	yarn, brake lining, Roll linings, Disk pads etc	-	-	-	0.321 (0.180 – 0.280) [2A]	-	1.66 (1.64-1.69) [2P] 1.71 (1.25– 2.18) [2A]	-
E	Textile Cloth ropes, Jointings, Brake linings, Limpet sheets etc	0.189 (0.163-0.206) [3P]	0.260 (0.204-0.298) [3P]	0.198 (0.156-0.230) [5P]	0.311 (0.243 – 0.35) [5A]	0.221 (0.214 – 0.226) [6A]	-	0.369 (0.365-0.373) [2P] 0.345 [1A]

P = Personal Sample, A = Area Sample, - = Industrial section not present / done

A = Champion Seals Pvt. Ltd., Boisar (Mumbai)

B = Mechanical Packing Industries Pvt.Ltd. Tarapur-Boisar (Mumbai)

C = Mechanical Packing Industries Pvt.Ltd. Dahisar (Mumbai)

D = Hindustan Composite Pvt.Ltd. Aurangabad (Mumbai)

E =Hindustan Composite Pvt.Ltd. Ghatkopar (Mumbai)

Table-6
Demographic profiles of asbestos exposed-population

Demographic Parameters	UPAL (I) [104]	UPAL (II) [71]	ANL [90]	A-E [215]	Σ=480
Age (yrs)	43 ± 7	29.1± 7.4	33.0± 7.0	43.0± 9.0	39.1± 10.3
Height (cm)	165.5± 5.7	162.6± 6.5	163.9± 5.7	165.5± 6.7	164.8± 6.3
Weight (kg)	57.8± 10.7	56.8± 9.6	58.5± 8.3	61.6± 11.4	59.5± 10.6

UPAL (I): UP Asbestos Pvt Ltd., Lucknow

UPAL (II): UP Asbestos Pvt Ltd., Nagpur

ANL: Allied Nippon Pvt Ltd.

A-E:

A = Champion Seals Pvt. Ltd., Boisar (Mumbai)

B = Mechanical Packing Industries Pvt.Ltd. Tarapur-Boisar (Mumbai)

C = Mechanical Packing Industries Pvt.Ltd. Dahisar (Mumbai)

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Table-7
Personal habits of asbestos exposed-population

Personal habits/ Life styles	UPAL (I) [104]	UPAL (II) [71]	ANL [90]	A-E [215]	Σ=480
Smoking habits					
Smokers	39(37.5%)	7(9.85%)	35(38.88%)	21(9.76%)	102(21.25%)
Non- smokers	53 (50.96%)	59(83.09%)	55(61.11%)	192(89.30%)	359(74.79%)
Ex- smokers	1(0.96%)	-----	-	-	1(0.20%)
Tobacco chewing	11(10.57%)	5(7.04%)	-	2(0.93%)	18(3.75%)
Alcoholic habits					
Alcoholic	24(23.07%)	2(2.81%)	17(18.88%)	55(25.58%)	98(20.41%)
Non- alcoholic	80(76.92%)	69(97.18%)	73(81.11%)	160(74.41%)	382(79.58%)
Dieting habits					
Vegetarian	52(50%)	23(32.39%)	49(54.44%)	34(15.81%)	158(32.91%)
Non-vegetarian	52(50%)	48(67.60%)	41(45.55%)	181(84.18%)	322(67.08%)

[] = No. of subjects in a particular factory, Σ=Total, - = No subject in this category

UPAL (I): UP Asbestos Pvt Ltd., Lucknow

UPAL (II): UP Asbestos Pvt Ltd., Nagpur

ANL: Allied Nippon Pvt Ltd., Ghaziabad

A-E:

A = Champion Seals Pvt. Ltd., Boisar (Mumbai)

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E =Hindustan Composite Pvt.Ltd. Ghatkopar (Mumbai)

Table-8
Domestic fuel exposure pattern in asbestos exposed population

Domestic fuel type	UPAL (I) [104]	UPAL (II) [71]	ANL [90]	A-E [215]	Σ=480
Wood	-	-	-	11(5.11%)	11(2.29%)
Wood + Animal dung cake	2(1.92%)	-	-	-	2(0.41%)
Wood + Kerosene	2(1.92%)	-	2(2.22%)	22(10.23%)	26(5.41%)
Wood + Gas	-	-	-	12(5.58%)	12(2.5%)
LPG + Electricity	1(0.96%)	71(100%)	58(64.44%)	78(36.27%)	208(43.33%)
Kerosene	-	-	5(5.55%)	12(5.58%)	17(3.45%)
Animal dung cake	-	-	-	-	-
Combination(Wood +Animal dung cake + Coal + Crop residues + Kerosene + Electricity)	99(95.19%)	-	25(27.77%)	80(37.20%)	204(42.5%)

[] = No. of subjects in a particular factory, Σ=Total, - = No subject in this category

UPAL (I): UP Asbestos Pvt Ltd., Lucknow

UPAL (II): UP Asbestos Pvt Ltd., Nagpur

ANL: Allied Nippon Pvt Ltd., Ghaziabad

A-E:

A = Champion Seals Pvt. Ltd., Boisar (Mumbai)

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Table-9
Exposure wise distribution of clinical symptoms and signs of asbestos
exposed population at UPAL (I), Lucknow

EXPOSURE CATEGORY				
Symptoms and Signs	<5 years [18]	5-10 years [12]	>10 years [74]	Σ= [104]
Cough	4(22.22%)	27(16.66%)	21(28.37%)	27(25.96%)
Dyspnoea	3(16.66%)	3(25%)	20(27.02%)	26(25%)
Haemoptysis	2(11.11%)	-	7(9.45%)	9(8.65%)
Total	9(50%)	5(41.66%)	48(64.86%)	62(59.61%)
Clubbing	-	1(8.33%)	4(5.40%)	5(4.80%)
Crepitations	1(5.55%)	1(8.33%)	6(8.10%)	8(7.69%)
Ronchi	1(5.55%)	4(33.33%)	10(13.51%)	15(14.42%)
Total	2(11.11%)	6(50%)	20(27.02%)	28(26.92%)

[] = No. of subjects in a particular factory, Σ=Total, - = No subject in this category

Table-10
Exposure wise distribution of clinical symptoms and signs of asbestos
exposed population at UPAL (II), Nagpur

Symptoms and Signs	<5 years [64]
Cough	-
Dypsnoea on exertion	-
Haemoptysis	-
Total	-
Clubbing	-
Crepitations	-
Ronchi	-
Total	-

[] = No. of subjects in a particular factory, Σ =Total, - = No subject in this category

Table-11
Exposure wise distribution of clinical symptoms and signs of asbestos
exposed population at ANL, Ghaziabad

EXPOSURE CATEGORY				
Symptoms and Signs	<5 years [02]	5-10 years [54]	>10 years [34]	Σ= [90]
Cough	1(50%)	18(33.33%)	10(29.41%)	29(32.22%)
Dyspnoea	-	8(14.81%)	6(17.64%)	14(15.55%)
Haemoptysis	-	2(3.70%)	1(2.94%)	3(3.33%)
Total	1(50%)	28(51.85%)	17(50%)	46(51.11%)
Clubbing	-	2(3.70%)	4(11.76%)	6(6.66%)
Crepitations	-	7(12.96%)	2(5.88%)	9(10%)
Ronchi	-	3(5.55%)	6(17.64%)	9(10%)
Total	-	12(22.22%)	12(35.29%)	24(26.66%)

[] = No. of subjects in a particular factory, Σ=Total, - = No subject in this category

ANL: Allied Nippon Pvt Ltd.

Table-12
Exposure wise distribution of clinical symptoms and signs of asbestos
exposed population at A-E, Maharashtra

EXPOSURE CATEGORY				
Symptoms and Signs	<5 years [14]	5-10 years [31]	>10 years [170]*	Σ= [215]
Cough	3(21.42%)	7(22.58%)	46(27.05%)	56(26.04%)
Dyspnoea	5(35.71%)	6(19.35%)	54(31.76%)	65(30.23%)
Haemoptysis	-	-	7(4.11%)	7(3.25%)
Total	8(57.14%)	13(41.93%)	107(62.94%)	128(59.53%)
Clubbing	1(7.14%)	6(19.35%)	45(26.47%)	52(24.18%)
Crepitations	4(28.572%)	6(19.35%)	42(24.70%)	52(24.18%)
Ronchi	-	-	-	-
Total	5(35.71%)	12(38.70%)	87(51.17%)	104(48.37%)

[] = No. of subjects in a particular factory, Σ=Total, - = No subject in this category, * = few individuals having both clinical symptoms and signs

A-E:

A = Champion Seals Pvt. Ltd., Boisar (Mumbai)

B = Mechanical Packing Industries Pvt.Ltd. Tarapur-Boisar (Mumbai)

C = Mechanical Packing Industries Pvt.Ltd. Dahisar (Mumbai)

D = Hindustan Composite Pvt.Ltd. Aurangabad (Mumbai)

E =Hindustan Composite Pvt.Ltd. Ghatkopar (Mumbai)

Table-13
Lung function tests of factory workers of organized sector

LFT	UPAL (I) [104]	ANL [90]	A-E [215]	$\Sigma=409$
FEV1(L)	2.00+0.60	2.14+0.68	2.37+0.62	2.23+0.65
FVC(L)	2.49+0.59	2.48+0.71	2.83+0.84	2.67+0.77
FEF(L/s)	75.72+22.03	85.30+19.54	82.86+17.20	81.58+19.33
PEFR (L/min.)	272.64+123.56	246.34+126.96	371.50+122.03	318.82+135.44
FEV1/FVC (%)	80.73+17.90	86.32+18.02	83.82+25.84	83.59+22.49s

FEV1= Forced expiratory volume in one second, FVC= forced vital capacity, FEF= Forced expiratory flow, PEFR= Peak expiratory flow rate, FEV1/FVC= Ratio, L= liter, L/s= liter per second, L/min= liter per minute, (%)= percentage, []= no. of subjects in a particular factory, Σ = total.

UPAL (I): UP Asbestos Pvt Ltd., Lucknow

UPAL (II): UP Asbestos Pvt Ltd., Nagpur

ANL: Allied Nippon Pvt Ltd., Ghaziabad

A-E:

A = Champion Seals Pvt. Ltd., Boisar (Mumbai)

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C = Mechanical Packing Industries Pvt.Ltd. Dahisar (Mumbai)

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E =Hindustan Composite Pvt.Ltd. Ghatkopar (Mumbai)

Table-14
Exposure wise pattern of lung function impairments of asbestos exposed population at UPAL (I), Lucknow

LFTests	Exposure category			
	<5 years [18]	5-10 years [12]	>10 years [74]	Σ=104
LFT performed	18(100%)	12(100%)	74(100%)	104(100%)
LFT impairments	5(27.77%)	5(41.66%)	32(43.24%)	42(40.38%)
Normal	13(72.22%)	7(58.33%)	42(56.75%)	62(59.61%)
Obstruction	5(27.77%)	3(25%)	16(21.62%)	24(23.07%)
Mild	3(16.66%)	3(25%)	6(8.10%)	12(11.53%)
Moderate	2(11.11%)	-	6(8.10%)	8(7.69%)
Severe	-	-	4(5.40%)	4(3.84%)
Restriction	-	-	10(13.51%)	10(9.61%)
Mild	-	-	5(6.75%)	5(4.80%)
Moderate	-	-	3(4.05%)	3(2.88%)
Severe	-	-	2(2.70%)	2(1.92%)
Mixed	-	2(16.66%)	6(8.10%)	8(7.69%)
Mild	-	2(16.66%)	3(4.05%)	5(4.80%)
Moderate	-	-	2(2.70%)	2(1.92%)
Severe	-	-	1(1.35%)	1(0.96%)

[] = No. of subjects in a particular factory, Σ=Total, - = No subject in this category, LFT = Lung function tests

Table-15
Exposure wise pattern of lung function impairments of asbestos exposed population in UPAL (II), Nagpur

LF Tests	Exposure category			
	*<5 years [57]	5-10 years [06]	>10 years [01]	Σ=64
LFT performed	57(100%)	6(100%)	1(100%)	64(100%)
LFT impairments	17(29.82%)	3(50%)	-	20(31.25%)
Normal	40(70.17%)	3 (50%)	1(100%)	44(68.75%)
Obstruction	4(7.01%)	-	-	4(6.25%)
Mild	3(5.26%)	-	-	3(4.68%)
Moderate	1(1.75%)	-	-	1(1.56%)
Severe	-	-	-	-
Restriction	13(22.80%)	3(50%)	-	16(25%)
Mild	8(14.03%)	2(33.33%)	-	10(15.62%)
Moderate	5(8.77%)	1(6.66%)	-	6(9.37%)
Severe	-	-	-	-
Mixed	-	-	-	-
Mild	-	-	-	-
Moderate	-	-	-	-
Severe	-	-	-	-

[] = No. of subjects in a particular factory, Σ=Total, - = No subject in this category, LFT = Lung function tests, *7 individuals in <5 years exposure not performed PFT tests

Table-16
Exposure wise pattern of lung function impairments of asbestos exposed population at ANL, Ghaziabad

LF Tests	Exposure category			
	<5 years [02]	5-10 years [54]	>10 years [34]	Σ=90
LFT performed	2(100%)	54(100%)	34(100%)	90(100%)
LFT impairments	-	18(33.33%)	13(38.23%)	31(34.44%)
Normal	2(100%)	36(66.66%)	21(68.76%)	59(65.55%)
Obstruction	-	5(9.25%)	10(29.41%)	15(16.66%)
Mild	-	2(3.70%)	4(11.76%)	6(6.66%)
Moderate	-	3(5.55%)	5(14.70%)	8(8.88%)
Severe	-	-	1(2.94%)	1(1.11%)
Restriction	-	5(9.25%)	7(20.58%)	12(13.33%)
Mild	-	3(5.55%)	4(11.76%)	7(7.77%)
Moderate	-	1(1.85%)	3(8.82%)	4(4.44%)
Severe	-	1(1.85%)	-	1(1.11%)
Mixed	-	1(1.85%)	3(8.82%)	4(4.44%)
Mild	-	-	1(2.94%)	1(2.22%)
Moderate	-	-	2(5.88%)	2(2.22%)
Severe	-	1(1.85%)	-	1(1.11%)

[] = No. of subjects in a particular factory, Σ=Total, - = No subject in this category, LFT = Lung function test

Table-17
Exposure wise pattern of lung function impairments of asbestos exposed population in A-E, Maharashtra

LF Tests	Exposure category			
	<5 years [14]	5-10 years [31]	>10 years [170]	Σ=215
LFT performed	14(100%)	31(100%)	170(100%)	215(100%)
LFT impairments	8(57.14%)	9(29.03%)	83(48.82%)	100(46.51%)
Normal	6(42.85%)	22(70.96%)	87(51.17%)	115(53.48%)
Obstruction	-	1(3.25%)	23(13.52%)	24(12.83%)
Mild	-	-	12(7.05%)	12(6.41%)
Moderate	-	1(3.25%)	10(5.88%)	11(5.88%)
Severe	-	-	1(0.58%)	1(0.54%)
Restriction	8(57.14%)	8(25.80%)	60(35.29%)	76(40.65%)
Mild	4(42.85%)	5(16.12%)	38(35.52%)	49(26.20%)
Moderate	2(14.28%)	3(9.67%)	21(12.35%)	26(13.90%)
Severe	-	-	1(0.58%)	1(0.53%)
Mixed	-	-	-	-
Mild	-	-	-	-
Moderate	-	-	-	-
Severe	-	-	-	-

[] = No. of subjects in a particular factory, Σ=Total, - = No subject in this category, LFT = Lung function tests

A-E:

A = Champion Seals Pvt. Ltd., Boisar (Mumbai)

B = Mechanical Packing Industries Pvt.Ltd. Tarapur-Boisar (Mumbai)

C = Mechanical Packing Industries Pvt.Ltd. Dahisar (Mumbai)

D = Hindustan Composite Pvt.Ltd. Aurangabad (Mumbai)

E =Hindustan Composite Pvt.Ltd. Ghatkopar (Mumbai)

Table-18
**Exposure wise pattern of radiological impairments of asbestos-exposed-
population in UPAL (I), Lucknow**

X-rays	Exposure category			
	<5 years [18]	5-10 years [12]	>10 years [74]	$\Sigma=104$
X-rays done	16(88.88%)	9(75%)	62(83.78%)	87(83.65%)
+ ve finding	6(37.5%)	6(66.66%)	41(66.12%)	53(50.96%)
Normal	10(62.5%)	3(33.33%)	21(33.87%)	34(32.69%)
Chronic bronchitis	5(31.25%)	5(55.55%)	17(27.41%)	27(31.03%)
COPD	1(6.25%)	1(11.11%)	16(25.80%)	18(20.68%)
COPD with diaphragmatic calcification	-	-	1(1.61%)	1(1.14%)
COPD with pulmonary TB	-	-	5(8.06%)	5(5.74%)
Bilateral TB	-	-	1(1.61%)	1(1.14%)
Bilateral right side bronchitis	-	-	1(1.61%)	1(1.14%)

[] = No. of subjects in a particular factory, Σ =Total, - = No subject in this category

Table-19
Exposure wise distribution pattern of radiological findings of asbestos-
exposed-population in UPAL (II), Nagpur

X-rays	Exposure category			
	<5 years [64]	5-10 years [06]	>10 years [01]	Σ [71]
X-rays done	50 (78.12%)	5(83.33%)	-	55(77.46%)
+ ve finding	-	-	-	-
Normal	50 (100%)	5(100%)	-	55(100%)

[] = No. of subjects in a particular factory, Σ=Total, - = No subject in this category

Table-20
Exposure wise distribution pattern of radiological findings of asbestos-exposed-population in ANL, Ghaziabad

X-rays	Exposure category			
	<5 years [02]	5-10 years [54]	>10 years [34]	Σ [90]
X-rays done	1(50%)	32(59.25%)	27(79.41%)	60(66.66%)
+ ve finding	-	15(46.87%)	20(74.07%)	35(58.33%)
Normal	1(100%)	17(53.12%)	7(25.92%)	25(41.66%)
Chronic bronchitis	-	11(34.37%)	7(25.92%)	18(30%)
Chronic bronchitis with cardiomagaly	-	-	1(3.70%)	1(1.66%)
pulmonary TB	-	2(6.25%)	2(7.40%)	4(6.66%)
COPD	-	1(3.12%)	7(25.92%)	8(13.33%)
Left side pleural thickening	-	1(3.12%)	-	1(1.66%)
Healed pulmonary TB with COPD	-	-	2(7.40%)	2(3.33%)
Bilateral PB	-	-	1(3.70%)	1(1.66%)

[] = No. of subjects in a particular factory, Σ=Total, - = No subject in this category

ANL: Allied Nippon Pvt Ltd.

Table-21
Exposure wise distribution pattern of radiological findings of asbestos-
exposed-population in A-E, Maharashtra

X-rays	Exposure category			
	<5 years [14]	5-10 years [31]	>10 years [170]	Σ =215
X-rays done	9(64.28%)	13(41.93%)	76 (44.70%)	98(45.58%)
+ ve finding	-	-	35(46.05%)	35(35.71%)
Normal	9(100%)	13(100%)	41(53.94%)	63(64.28%)
Cardiomagaly	-	-	1(1.31%)	1(1.02%)
RNP	-	-	22(28.94%)	22(24.44%)
Healed TB	-	-	1(1.31%)	1(1.02%)
Calcification in upper region	-	-	1(1.31%)	1(1.02%)
COPD	-	-	10(13.51%)	10(10.20%)

[] = No. of subjects in a particular factory, Σ =Total, - = No subject in this category

A-E:

A = Champion Seals Pvt. Ltd., Boisar (Mumbai)

B = Mechanical Packing Industries Pvt.Ltd. Tarapur-Boisar (Mumbai)

C = Mechanical Packing Industries Pvt.Ltd. Dahisar (Mumbai)

D = Hindustan Composite Pvt.Ltd. Aurangabad (Mumbai)

E =Hindustan Composite Pvt.Ltd. Ghatkopar (Mumbai)

Table-22
Exposure wise distribution pattern of asbestos bodies in sputum of
asbestos-exposed-population in different factories of organized sector

UPAL (I)	Exposure category			
	<5 years [18]	5-10 years [12]	>10 years [74]	Σ [104]
Samples collected	9(50%)	5(41.66%)	47(63.51%)	61(58.65%)
+ ve findings	-	-	-	-
Normal	9(100%)	5(100%)	47(100%)	61(100%)
UPAL (II)	<5 years [64]	5-10 years [06]	>10 years [01]	Σ [71]
Samples collected	29(45.31%)	2(33.33%)	1(100%)	32(45.07%)
+ ve findings	-	-	-	-
Normal	29(100%)	2(100%)	1(100%)	32(100%)
ANL	<5 years [02]	5-10 years [54]	>10 years [34]	Σ [90]
Samples collected	1(50%)	32(59.25%)	2(5.58%)	35(38.88%)
+ ve findings	-	-	-	-
Normal	1(100%)	32(100%)	2(100%)	35(100%)
A-E	<5 years [14]	5-10 years [31]	>10 years [170]	Σ = 215
Samples collected	12(85.71%)	10(32.25%)	94(55.29%)	116(53.95%)
+ ve findings	3(25%)	4(40%)	34(36.17%)	41(35.34%)
Normal	9(75%)	6(60%)	60(63.82%)	75(64.65%)

[] = No. of subjects in a particular factory, Σ=Total, - = No subject in this category

Table-23
Exposure wise distribution pattern of acid-fast bacilli I sputum of asbestos-exposed-population in organized sector

UPAL (I)	Exposure category			
	<5 years [18]	5-10 years [12]	>10 years [74]	Σ [104]
Samples collected	9(50%)	5(41.66%)	47(63.51%)	61(58.65%)
+ ve findings	-	-	-	-
Normal	9(100%)	5(100%)	47(100%)	61(100%)
UPAL (II)	<5 years [64]	5-10 years [06]	>10 years [01]	Σ [71]
Samples collected	29(45.31%)	2(33.33%)	1(100%)	32(45.07%)
+ ve findings	-	-	-	-
Normal	29(100%)	2(100%)	1(100%)	32(45.07%)
ANL	<5 years [02]	5-10 years [54]	>10 years [34]	Σ [90]
Samples collected	1(50%)	32(59.25%)	2(5.88%)	35(38.88%)
+ ve findings	-	-	1(50%)	1(2.85%)
Normal	1(100%)	32(100%)	1(50%)	34(97.14%)
A-E	<5 years [14]	5-10 years [31]	>10 years [170]	Σ [215]
Samples collected	11(78.57%)	13(41.93%)	92(54.11%)	116(53.95%)
+ ve findings	-	-	3(3.26%)	3(2.58%)
Normal	11(100%)	13(100%)	89(96.73%)	113(97.41%)

[] = No. of subjects in a particular factory, Σ=Total, - = No subject in this category

Table-24
Comparative demographic profiles of asbestos exposed and control (unexposed) populations of unorganized sector

Demographic parameters	Asbestos exposed population			Control (unexposed) population		
	Male [90]	Female[18]	Σ = 108	Male [51]	Female [84]	Σ = 135
Age (Yrs.)	31 ± 8	31 ± 8	31 ± 8	34 ± 8	31 ± 8	33 ± 8
Height (Cms.)	165.5 ± 7.5	153.3 ± 4.6	163.5 ± 8.4	168.7 ± 5.5	156.0 ± 5.5	160.8 ± 8.2
Weight (Kg.)	52.4 ± 7.92	41.2 ± 6.6	50.5 ± 8.7	58.8 ± 9.2	47.5 ± 8.1	51.8 ± 10.1

Age, Height, Weight; Mean ± SD, Yrs. = Years, Cms. = Centimeter, kg. = Kilogram, [] = No. of subjects in a particular category, Σ=Total

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Table-25
Personal habits related to the life style of asbestos exposed and control (unexposed) population of unorganized sector

Personal habits/life styles	Asbestos exposed population			Control (unexposed) population		
	Male [90]	Female[18]	Σ = 108	Male [51]	Female [84]	Σ = 135
Smoking habit						
Smokers	34 (37.77%)	3 (16.66%)	37 (34.25%)	23 (45.09%)	12 (14.28%)	35 (25.92%)
Alcoholic habit						
Alcoholic	10 (11.11%)	1 (5.55%)	11 (10.18%)	8 (15.68%)	2 (2.38%)	10 (7.40%)
Dieting habit						
Non - vegetarian	53 (58.88%)	7 (38.88%)	60 (55.55%)	40(78.43%)	62 (73.80%)	102 (75.55%)

[] = No. of subjects in a particular category, Σ=Total, % = Percentage

Table-26
Exposure wise distribution of clinical symptoms and signs in exposed population of unorganized sector

Parameters	< 5 years [62]			5 - 10 years [27]			>10 years [19]		
	M (50) (80.64%)	F (12) (19.35%)	T (62) (100%)	M (23) (85.18%)	F (04) (14.81%)	T (27) (100%)	M (23) (89.47%)	F (04) (10.52%)	T (27) (100%)
Cough	9 (18%)	3 (25%)	12 (19.35%)	4 (17.39%)	-	4 (14.81%)	7 (41.17%)	1 (50%)	8 (42.10%)
Dyspnea on exertion	10 (20%)	6 (50%)	16 (25.80%)	4 (17.39%)	2 (7.40%)	6 (22.22%)	6 (35.29%)	1 (50%)	7 (36.84%)
Haemoptysis	4 (18%)	-	4 (6.45%)	-	-	-	1 (5.88%)	-	1 (5.26%)
Total	23 (46%)	9 (75%)	32 (51.61%)	8 (34.78%)	2 (50%)	10 (37.04%)	14 (82.35%)	2 (100%)	16 (84.21%)
Clubbing	11 (22%)	4 (33%)	15 (24.19%)	7 (30.34%)	1 (4.34%)	8 (29.62%)	7 (41.17%)	-	8 (42.10%)
Crepitations	7 (14%)	3 (25%)	10 (16.12%)	2 (8.69%)	2 (7.40%)	4 (14.81%)	2 (11.76%)	1 (50%)	4 (21.05%)
Ronchi	3 (6%)	1 (18.33%)	4 (6.45%)	1 (4.34%)	-	1 (3.70%)	-	1 (50%)	-
Total	21 (42%)	8 (66.66%)	29 (46.77%)	10 (43.47%)	3 (75%)	12 (44.44%)	9 (52.94%)	2 (100%)	12 (63.15%)

M = Male, F = female, Σ = Total, [] = No. of subjects in a particular exposure category, - = No. of subjects in this category, % = Percentage

Table-27
Lung function profiles in male and female working populations of unorganized sector

Personal habits/life styles	Asbestos exposed population			Control (unexposed) population		
	Male [90]	Female[18]	Σ = 108	Male [51]	Female [84]	Σ = 135
FEV ₁ (L)	2.25 ± 0.73	1.60 ± 0.58	2.14 ± 0.75	2.81 ± 0.87	1.60 ± 0.58	2.36 ± 0.88
FVC (L)	2.56 ± 0.75	1.96 ± 0.56	2.46 ± 0.75	3.34 ± 1.08	1.96 ± 0.56	2.67 ± 1.03
FEF (L/s)	89 ± 1.67	82.66 ± 28.67	87.94 ± 19.21	87.49 ± 12.12	82.66 ± 28.67	92.55 ± 15.79
PEFR (L/m)	277.71 ± 126.97	251.44 ± 87.26	273.33 ± 121.31	331.13 ± 105.10	251.44 ± 87.26	273.79 ± 90.97
FEV ₁ /FVC (%)	40.50 ± 8.92	36.69 ± 17.20	77.19 ± 25.62	82.88 ± 11.25	36.69 ± 17.20	92.58 ± 39.99

FEV₁= Force expiratory volume in one second, FVC = Force vital capacity, FEF = Force expiratory flow, PEFR = Peak expiratory flow rate, FEV₁/FVC% = Ratio, L = Litre, L/s = Litre/ second, L/min. = Litre/minute, % = Percentage, Σ = Total, [] = No. of subjects in a particular category

Table-28
X-rays analysis of < 5, 5-10, and >10 years asbestos exposed unorganized sector population

Parameters	< 5 years [62]			5 - 10 years [27]			>10 years [19]		
	M (50) (80.64%)	F (12) (19.35%)	T (62) (100%)	M (23) (85.18%)	F (04) (14.81%)	T (27) (100%)	M (23) (89.47%)	F (04) (10.52%)	T (27) (100%)
X-rays done	29 (58%)	4 (33.33%)	33 (52.22%)	16 (69.56%)	1 (25%)	17 (62.96%)	8 (47.05%)	1 (50%)	9 (47.36%)
+ ve findings	16 (15.17%)	1 (25%)	17 (51.51%)	6 (37.5%)	-	6 (35.29%)	5 (62.5%)	-	5 (55.55%)
Normal	13 (44.82%)	3 (75%)	16 (48.48%)	10 (62.5%)	1 (100%)	11 (64.70%)	3 (37.5%)	1 (100%)	4 (44.44%)
RNP	5 (17.24%)	1 (25%)	6 (18.18%)	1 (6.25%)	-	1 (5.88%)	-	-	-
Bronchiectasis	-	-	-	-	-	-	2 (25%)	-	2 (22.22%)
Pulmonary TB	2 (6.89%)	-	2 (6.06%)	-	-	-	-	-	-
IBVM	9 (31.03%)	-	9 (27.27%)	4 (25%)	-	4 (23.52%)	2 (25%)	-	2 (22.22%)
Plaque	-	-	-	1 (6.25%)	-	1 (5.88%)	-	-	-
Calcification in leftside diafragm	-	-	-	-	-	-	1 (25%)	-	1 (11.11%)

% = Percentage, T = Total, [] = No. of subjects in a particular exposure category, - = No. of subjects in this category,

Table-29
High resolution computed topography (HRCT) examinations in
asbestos exposed unorganized population

Exposure duration	Percentage of asbestosis (x-rays and HRCT diagnosed)
< 5 years (n=2)	28.6
5 -10 years (n=1)	14.3
11- 20 years (n=3)	42.8
21-30 years (n=1)	NA

NA = Not available for HRCT, n = No. of subjects in a particular category

Table-30
Asbestos bodies analysis in sputum of asbestos exposed unorganized sector population

Sputum analysis	Asbestos exposed population			Control (unexposed) population		
	Male [90]	Female[18]	$\Sigma = 108$	Male [51]	Female [84]	$\Sigma = 135$
Collected samples	41 (45.55%)	8 (44.44%)	49 (45.37%)	29 (56.86%)	38 (45.23%)	67 (49.62%)
Asbestos bodies	11 (26.82%)	1 (12.5%)	12 (24.48%)	-	-	-
Normal	30 (73.17%)	7 (87.5%)	37 (75.51%)	29 (100%)	38 (100%)	67 (100%)

% = Percentage, [] = No. of subjects in a particular category, - = No. of subjects in this category

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Table-31**Exposure wise distribution of asbestos bodies <5, 5-10, and >10 years of asbestos exposed unorganized sector population**

Sputum analysis	< 5 years [62]			5 - 10 years [27]			>10 years [19]		
	M (50) (80.64%)	F (12) (19.35%)	T (62) (100%)	M (23) (85.18%)	F (04) (14.81%)	T (27) (100%)	M (23) (89.47%)	F (04) (10.52%)	T (27) (100%)
Sample collected	23 (46%)	4 (33.33%)	27 (43.54%)	9 (39.13%)	2 (50%)	11 (40.74%)	9 (52.94%)	2 (100%)	11 (57.89%)
+ ve findings	6 (26.08%)	1 (25%)	7 (25.92%)	1 (11.11%)	-	1 (9.09%)	4 (44.44%)	-	4 (36.36%)
Normal	17 (73.91%)	3 (75%)	20 (74.07%)	8 (88.88%)	2 (100%)	10 (90.90%)	5 (55.55%)	2 (100%)	7 (63.63%)

M = Male, F = Female, T = Total, [] = No. of subjects in a particular exposure category, - = No. of subjects in this category, % = Percentage

Table-32
Acid – fast *bacilli* analysis in sputum of asbestos exposed unorganized sector population

Sputum analysis	Asbestos exposed population			Control (unexposed) population		
	Male [90]	Female[18]	Σ = 108	Male [51]	Female [84]	Σ = 135
Collected samples	41 (45.55%)	8 (44.44%)	49 (45.37%)	29 (56.86%)	38 (45.23%)	67 (49.62%)
Acid – fast <i>bacilli</i>	9 (21.95%)	-	9 (18.36%)	7 (24.13%)	5 (13.15%)	12 (17.91%)
Normal	32 (78.04%)	8 (100%)	40 (81.63%)	22 (75.86%)	33 (86.84%)	55 (82.08%)

% = Percentage, [] = No. of subjects in a particular category, - = No. of subjects in this category

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Table-33**Exposure wise distribution of acid – fast bacilli <5, 5-10, and >10 years of asbestos exposed unorganized sector population**

Sputum analysis	< 5 years [62]			5 - 10 years [27]			>10 years [19]		
	M	F	T	M	F	T	M	F	T
	(50)	(12)	(62)	(23)	(04)	(27)	(23)	(04)	(27)
	(80.64%)	(19.35%)	(100%)	(85.18%)	(14.81%)	(100%)	(89.47%)	(10.52%)	(100%)
Sample collected	24	4	28	9	2	11	8	2	10
	(48%)	(33.33%)	(45.16%)	(39.13%)	(50%)	(40.74%)	(47.05%)	(100%)	(52.63%)
+ ve findings	5	-	5	3	-	3	1	-	1
	(20.83%)		(17.85%)	(33.33%)		(27.27%)	(12.5%)		(10%)
Normal	19	4	23	6	2	8	7	2	9
	(79.16%)	(75%)	(82.14%)	(66.66%)	(100%)	(72.72%)	(87.5%)	(100%)	(90%)

M = Male, F = Female, T = Total, [] = No. of subjects in a particular exposure category, - = No. of subjects in this category, % = Percentage

Table-34
Comparative demographical profile of asbestos exposed unorganized sector and organized sector population

Demographic Parameters	Unorganized sector asbestos exposed population			Organized sector asbestos exposed *male population
	Male [90]	Female [18]	$\Sigma = 108$	$\Sigma = 480$
Age (yrs)	31± 8	31 ± 8	31 ± 8	39.1 ± 10.3
Height (cm)	165.5 ± 7.5	153.3 ± 4.6	163.5 ± 8.4	164.8 ± 6.3
Weight (kg)	52.4 ± 7.92	41.2 ± 6.6	50.5 ± 8.7	59.5 ± 10.6

Age, Height, Weight; Mean ± SD, Yrs. = Years, Yrs. = years, Cms. = Centimeter, Kg. = kilograms, Σ = Total, [] = No. of subjects, * No female individual present

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Table-35
Comparative demographical profile of asbestos exposed unorganized sector and organized sector population

Personal habits/Life styles	Unorganized sector asbestos exposed population			Organized sector asbestos exposed *male population
	Male [90]	Female [18]	$\Sigma = 108$	$\Sigma = 480$
Smoking habit				
Smokers	34 (37.77%)	3 (16.66%)	37 (34.25%)	102 (21.25%)
Ex-smokers	-	-	-	1 (1.20%)
Tobacco chewing	-	-	-	18 (3.37%)
Alcoholic habit				
Alcoholic	10 (11.11%)	1 (5.55%)	11 (10.18%)	98 (20.14%)
Dieting habit				
Non-vegetarian	53 (58.88%)	7 (38.88%)	60 (55.55%)	322 (67.08%)

Σ = Total, [] = No. of subjects, * No female individual present, - = No. of subject in this category, % = percentage

Table-36
Lung function tests of asbestos exposed unorganized and organized sector population

LFT	Unorganized sector asbestos exposed population			Organized sector asbestos exposed *male population
	Male [90]	Female [18]	Σ = 108	**Σ = 480
FEV1(L)	2.25 ± 0.73	1.60 ± 0.58	2.14 ± 0.75	2.23 ± 0.65
FVC(L)	2.56 ± 0.75	1.96 ± 0.56	2.46 ± 0.75	2.67 ± 0.77
FEF(L/s)	89 ± 16.7	82.66 ± 28.67	87.94 ± 19.21	81.58 ± 19.33
PEFR (L/min.)	277.71 ± 126.97	251.44 ± 87.26	273.33 ± 121.31	318.82 ± 135.44
FEV1/FVC (%)	85.28 ± 18.30	81.63 ± 17.20	77.19 ± 25.62	83.59 ± 22.49

FEV1= Forced expiratory volume in one second, FVC= forced vital capacity, FEF= Forced expiratory flow, PEFR= Peak expiratory flow rate, FEV1/FVC= Ratio, L= liter, L/s= liter per second, L/min= liter per minute, (%)= percentage,[]= no. of subjects in a particular factory, Σ = total, * = No female individual present, ** = 71 subjects pulmonary function tests showed directly obstruction or restriction instead of these parameters

Table-37
X-rays analysis of < 5, 5-10, and >10 years asbestos exposed unorganized sector population

Parameters	< 5 years [62]			5 - 10 years [27]			>10 years [19]		
	M (50) (80.64%)	F (12) (19.35%)	T (62) (100%)	M (23) (85.18%)	F (04) (14.81%)	T (27) (100%)	M (17) (89.47%)	F (02) (10.52%)	T (19) (100%)
X-rays done	29 (58%)	4 (33.33%)	33 (52.22%)	16 (69.56%)	1 (25%)	17 (62.96%)	8 (47.05%)	1 (50%)	9 (47.36%)
+ ve findings	16 (15.17%)	1 (25%)	17 (51.51%)	6 (37.5%)	-	6 (35.29%)	5 (62.5%)	-	5 (55.55%)
RNP	5 (17.24%)	1 (25%)	6 (18.18%)	1 (6.25%)	-	1 (5.88%)	-	-	-
Bronchiectasis	-	-	-	-	-	-	2 (25%)	-	2 (22.22%)
Pulmonary TB	2 (6.89%)	-	2 (6.06%)	-	-	-	-	-	-
IBVM	9 (31.03%)	-	9 (27.27%)	4 (25%)	-	4 (23.52%)	2 (25%)	-	2 (22.22%)
Plaque	-	-	-	1 (6.25%)	-	1 (5.88%)	-	-	-
Calcification in leftside diaphragm	-	-	-	-	-	-	1 (25%)	-	1 (11.11%)

M = Male, F = female, T = Total, % = Percentage, [] = No. of subjects in a particular exposure category, - = No. of subjects in this category,

Table-38
X-rays analysis of < 5, 5-10, and >10 years asbestos exposed organized sector population

Parameters	< 5 years [62]			5 - 10 years [27]			>10 years [19]		
	M (50) (80.64%)	F (12) (19.35%)	T (62) (100%)	M (23) (85.18%)	F (04) (14.81%)	T (27) (100%)	M (17) (89.47%)	F (02) (10.52%)	T (19) (100%)
X-rays done	29 (58%)	4 (33.33%)	33 (53.22%)	16 (69.56%)	1 (25%)	17 (62.96%)	8 (47.05%)	1 (50%)	9 (47.36%)
+ ve findings	16 (15.17%)	1 (25%)	17 (51.51%)	6 (37.5%)	-	6 (35.29%)	5 (62.5%)	-	5 (55.55%)
RNP	5 (17.24%)	1 (25%)	6 (18.18%)	1 (6.25%)	-	1 (5.88%)	-	-	-
Bronchiectasis	-	-	-	-	-	-	2 (25%)	-	2 (22.22%)
Pulmonary TB	2 (6.89%)	-	2 (6.06%)	-	-	-	-	-	-
IBVM	9 (31.03%)	-	9 (27.27%)	4 (25%)	-	4 (23.52%)	2 (25%)	-	2 (22.22%)
Plaque	-	-	-	1 (6.25%)	-	1 (5.88%)	-	-	-
Calcification in left side diaphragm	-	-	-	-	-	-	1 (25%)	-	1 (11.11%)

M = Male, F = female, T = Total, % = Percentage, [] = No. of subjects in a particular exposure category, - = No. of subjects in this category,

Table-39
Sputum analysis of <5, 5-10, and >10 years of asbestos exposed unorganized sector population

Asbestos body analysis	< 5 years [62]			5 - 10 years [27]			>10 years [19]		
	M (50) (80.64%)	F (12) (19.35%)	T (62) (100%)	M (23) (85.18%)	F (04) (14.81%)	T (27) (100%)	M (17) (89.47%)	F (02) (10.52%)	T (19) (100%)
Sample collected	24 (48%)	4 (33.33%)	28 (45.16%)	9 (39.13%)	2 (50%)	11 (40.74%)	8 (47.05%)	2 (100%)	10 (52.63%)
+ ve findings	5 (20.83%)	-	5 (17.85%)	3 (33.33%)	-	3 (27.27%)	1 (12.5%)	-	1 (10%)
Normal	19 (79.16%)	4 (75%)	23 (82.14%)	6 (66.66%)	2 (100%)	8 (72.72%)	7 (87.5%)	2 (100%)	9 (90%)

M = Male, F = Female, T = Total, [] = No. of subjects in a particular exposure category, - = No. of subjects in this category, % = Percentage

Table-40
Sputum analysis of <5, 5-10 and >10 years asbestos exposed organized sector population

Asbestos body analysis	<5 years [87]	5-10 years [115]	>10 years [278]	Σ [480]
Collected samples	49(56.32%)	69(33.31%)	156(56.11%)	244(50.83%)
Asbestos body	3(6.12%)	4(6.12%)	34(21.89%)	41(16.80%)
Normal	46(93.87%)	35(89.74%)	122(78.20%)	203(83.19%)

M = Male, F = Female, T = Total, [] = No. of subjects in a particular exposure category, - = No. of subjects in this category, % = Percentage

Table-41
Sputum analysis of <5, 5-10, and >10 years of asbestos exposed unorganized sector population

Acid fast bacilli	< 5 years [62]			5 - 10 years [27]			>10 years [19]		
	M (50) (80.64%)	F (12) (19.35%)	T (62) (100%)	M (23) (85.18%)	F (04) (14.81%)	T (27) (100%)	M (17) (89.47%)	F (02) (10.52%)	T (19) (100%)
Sample collected	24 (48%)	4 (33.33%)	28 (45.16%)	9 (39.13%)	2 (50%)	11 (40.74%)	8 (47.05%)	2 (100%)	10 (52.63%)
+ ve findings	5 (20.83%)	-	5 (17.85%)	3 (33.33%)	-	3 (27.27%)	1 (12.5%)	-	1 (10%)
Normal	19 (79.16%)	4 (75%)	23 (82.14%)	6 (66.66%)	2 (100%)	8 (72.72%)	7 (87.5%)	2 (100%)	9 (90%)

M = Male, F = Female, T = Total, [] = No. of subjects in a particular exposure category, - = No. of subjects in this category, % = Percentage

Table-42
Sputum analysis of <5, 5-10, and >10 years of asbestos exposed organized sector population

Acid fast bacilli	<5 years (87)	5-10 years (115)	>10 years (278)	Σ-480
Sample collected	49(56.32%)	39(33.91%)	156(56.11%)	244(50.83%)
+ ve findings	-	-	4(2.81%)	4(1.63%)
Normal	49(100%)	39(100%)	152(97.43%)	240(98.36%)

% = Percentage, [] = No. of subjects in a particular category, - = No. of subjects in this category

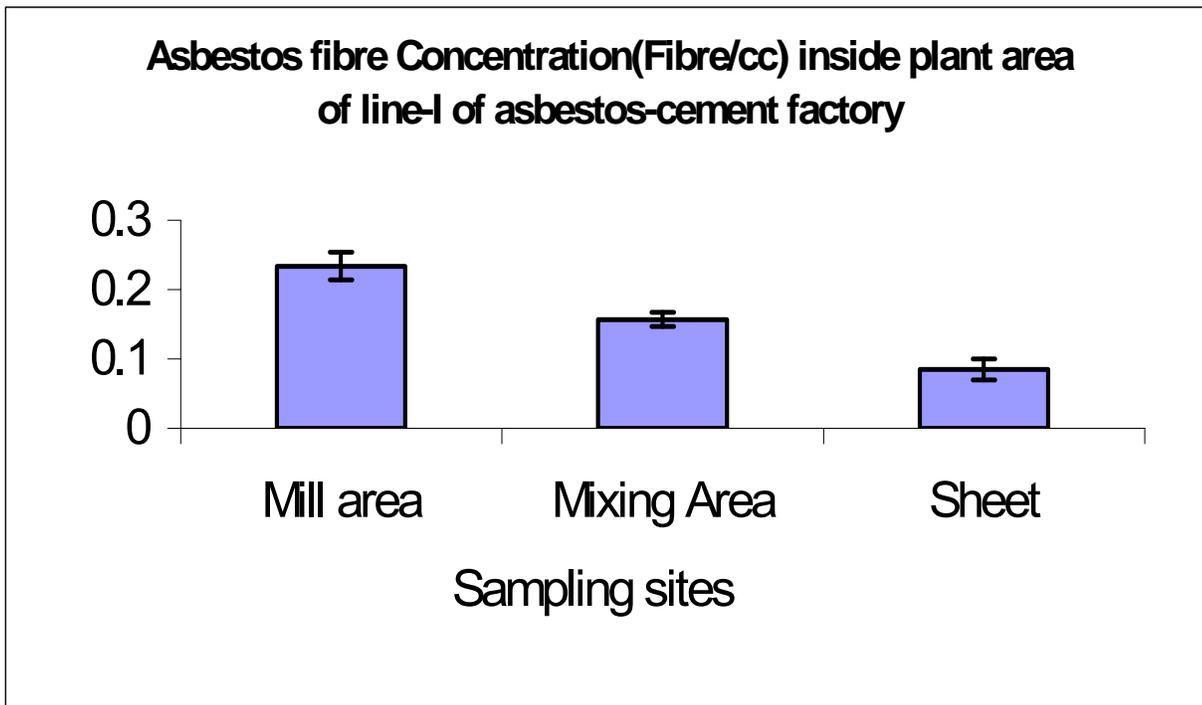


Figure 1: Asbestos fiber count (f/cc) in line-I of UPAL, Mohanlal Ganj, Lucknow.

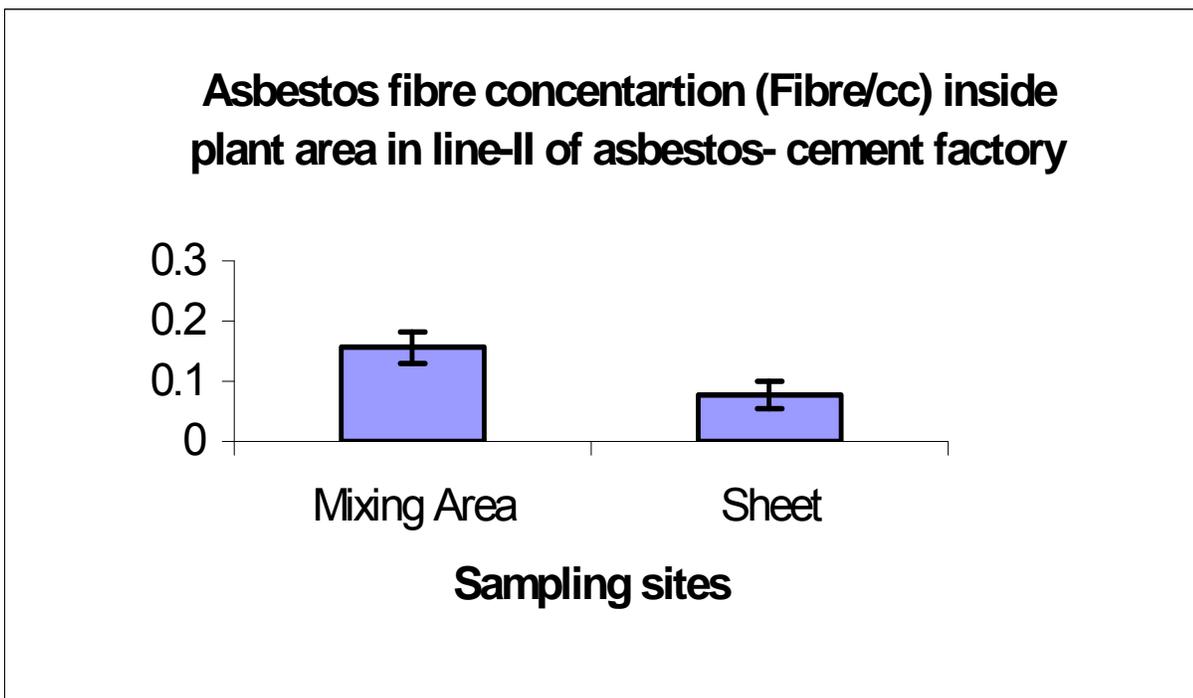


Figure 2: Asbestos fiber count (f/cc) in line-II of UPAL, Mohanlal Ganj, Lucknow.

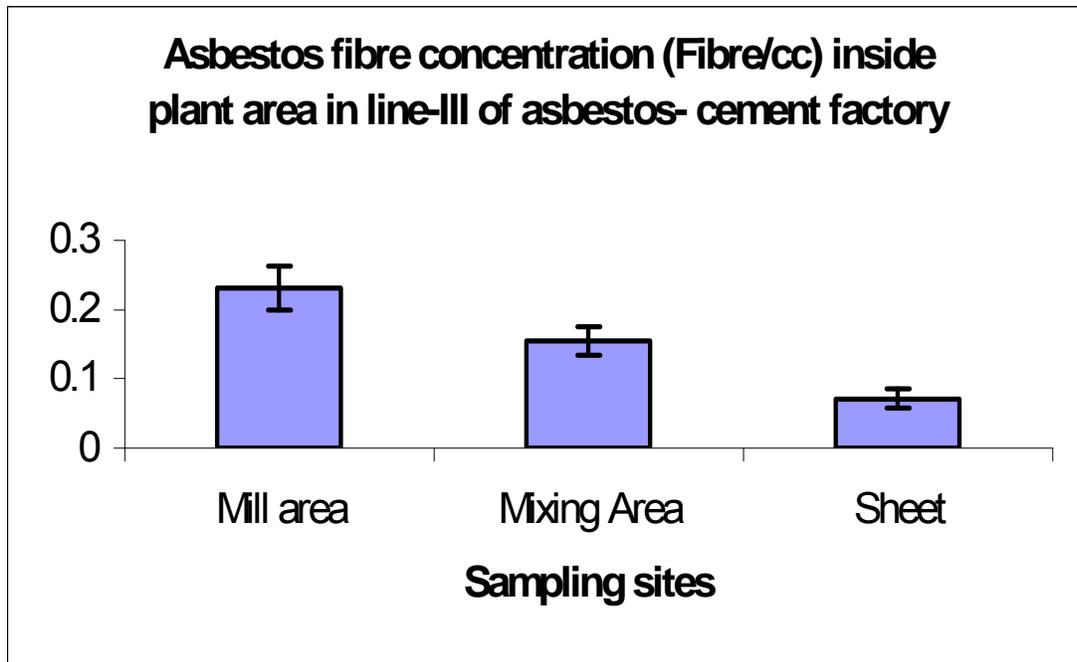


Figure 3: Asbestos fiber count (f/cc) in line-III of UPAL, Mohanlal Ganj, Lucknow.

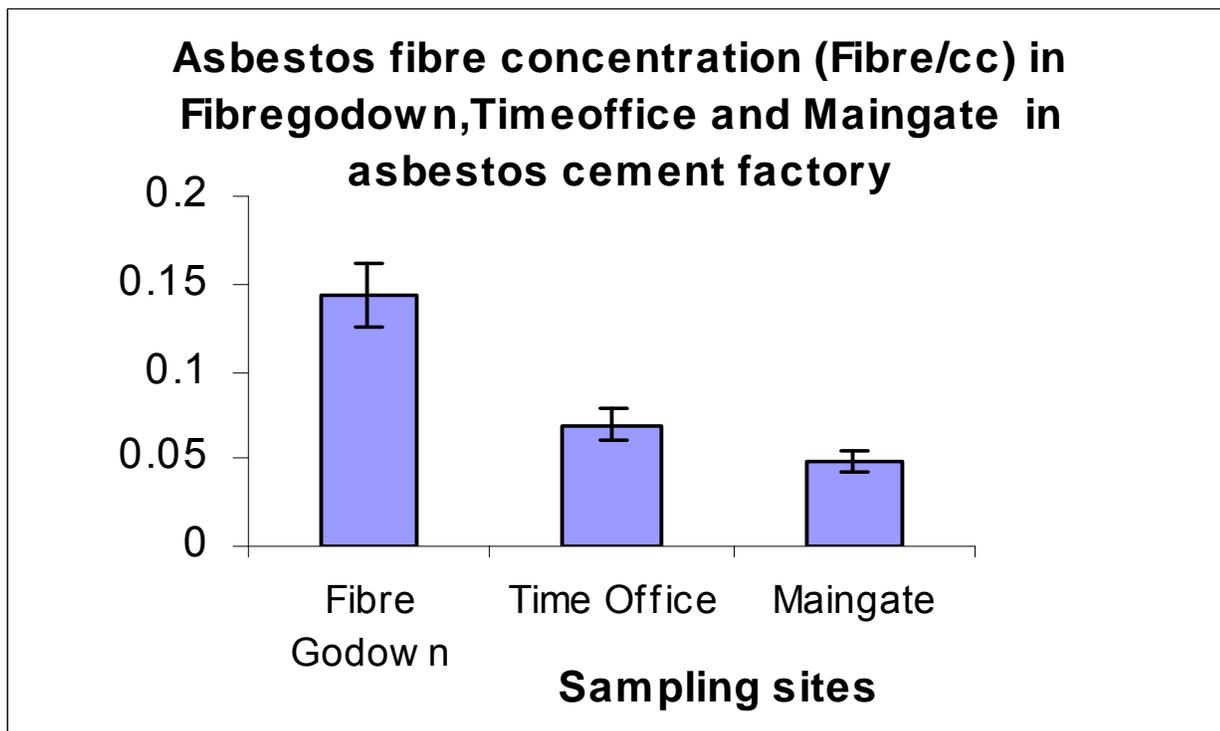


Figure 4: Asbestos fiber count (f/cc) in Fiber godown, Time office and Main gate of UPAL, Mohanlal Ganj, Lucknow.

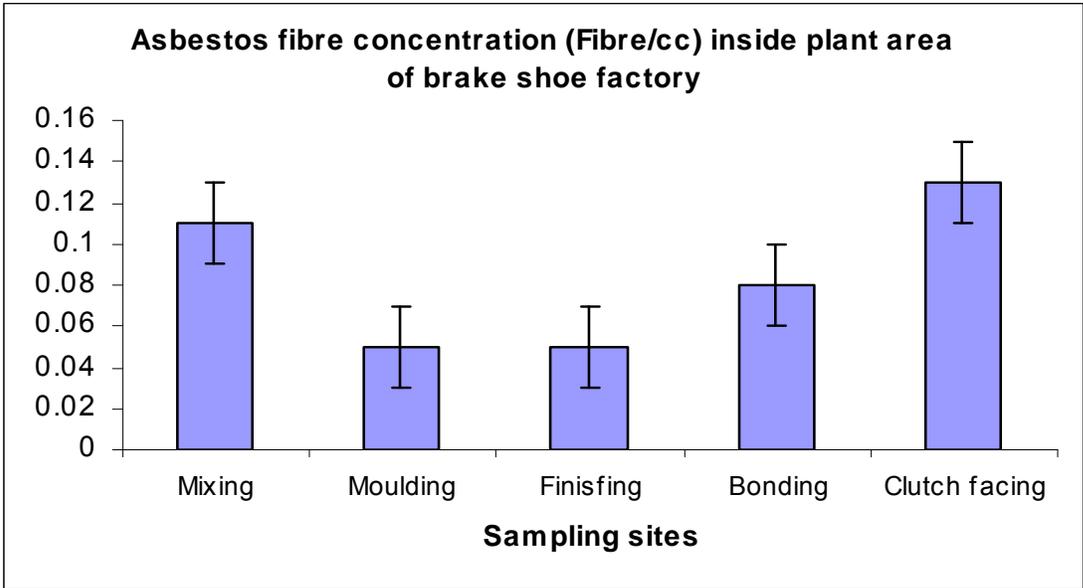


Figure 5: Asbestos fiber concentration (fiber/cc) inside plant area of brake shoe factory, M/s Allied Nippon Pvt. Ltd., Ghaziabad

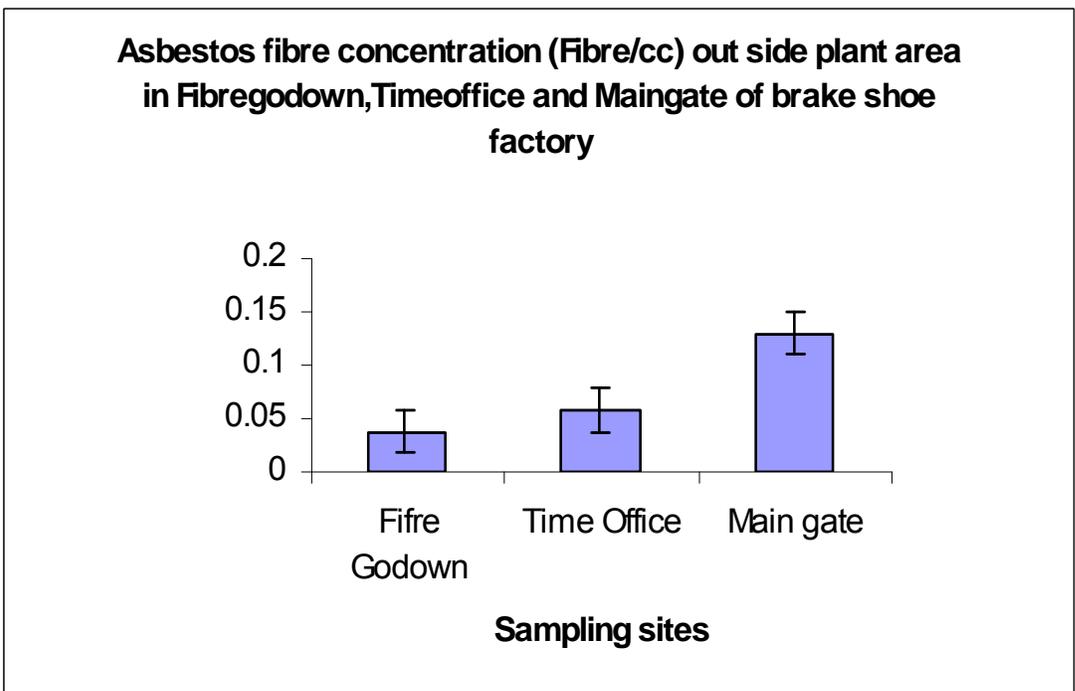


Figure 6: Asbestos fiber concentration (fiber/cc) outside plant area of brake shoe factory, M/s Allied Nippon Pvt. Ltd., Ghaziabad

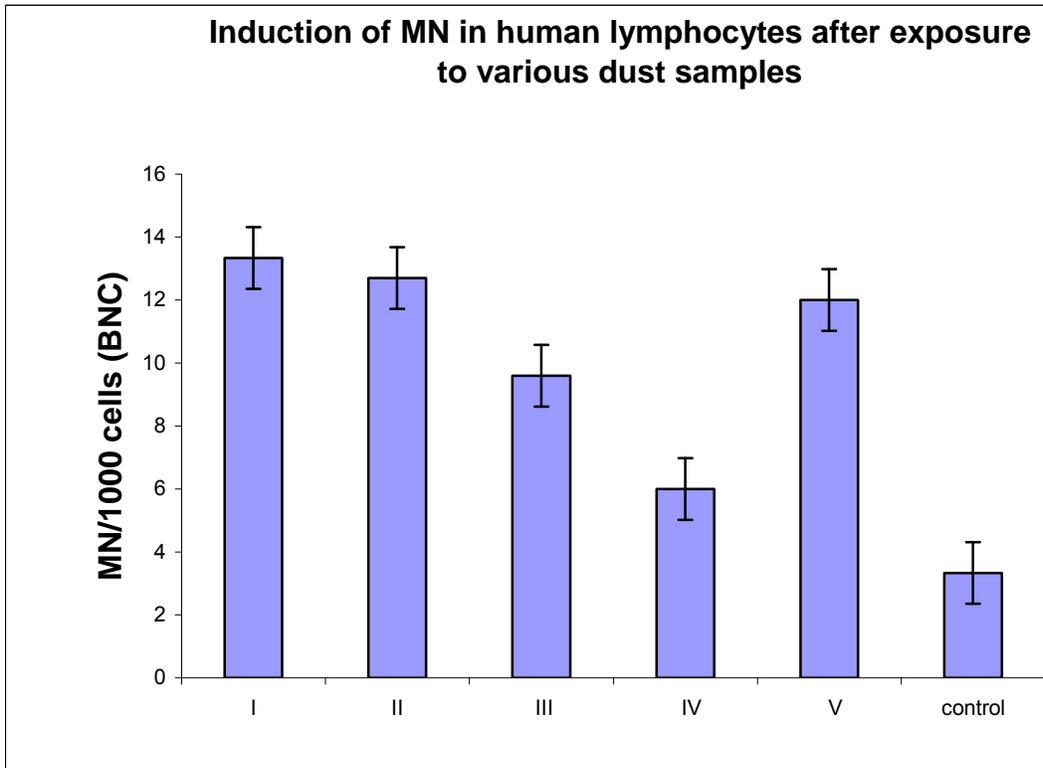


Figure 7: Induction of MN in human lymphocytes after exposure to indigenous asbestos samples

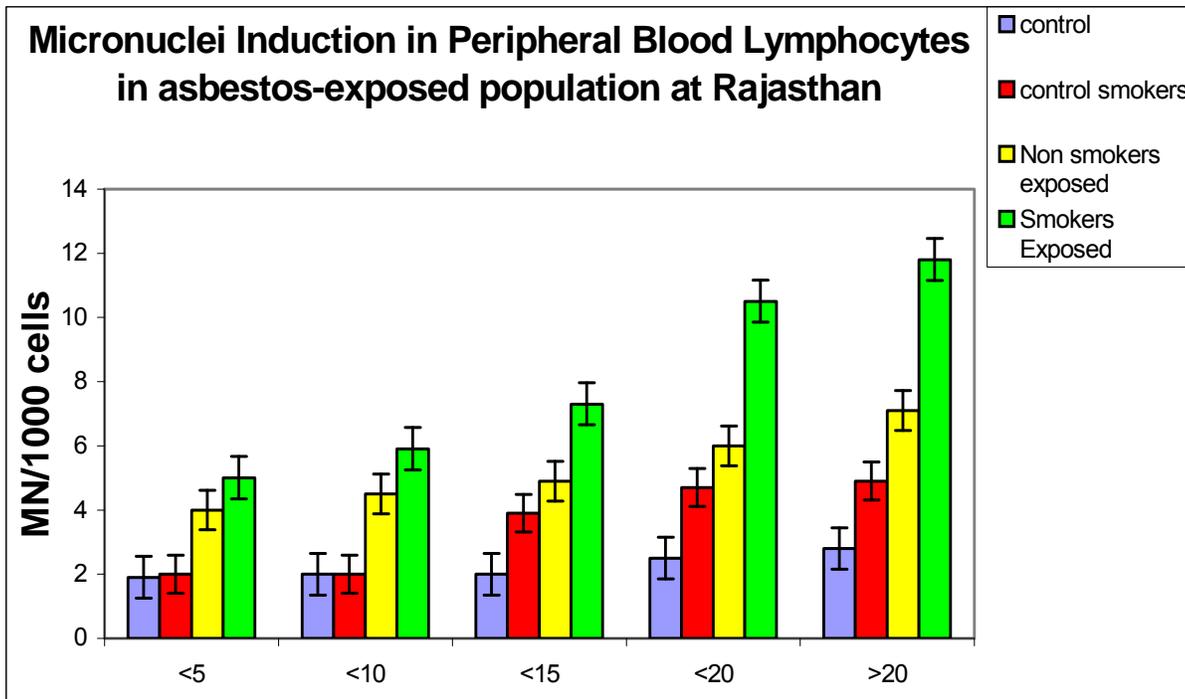


Figure 8: Micronuclei induction in Peripheral Blood Lymphocytes in asbestos-exposed population at Rajasthan

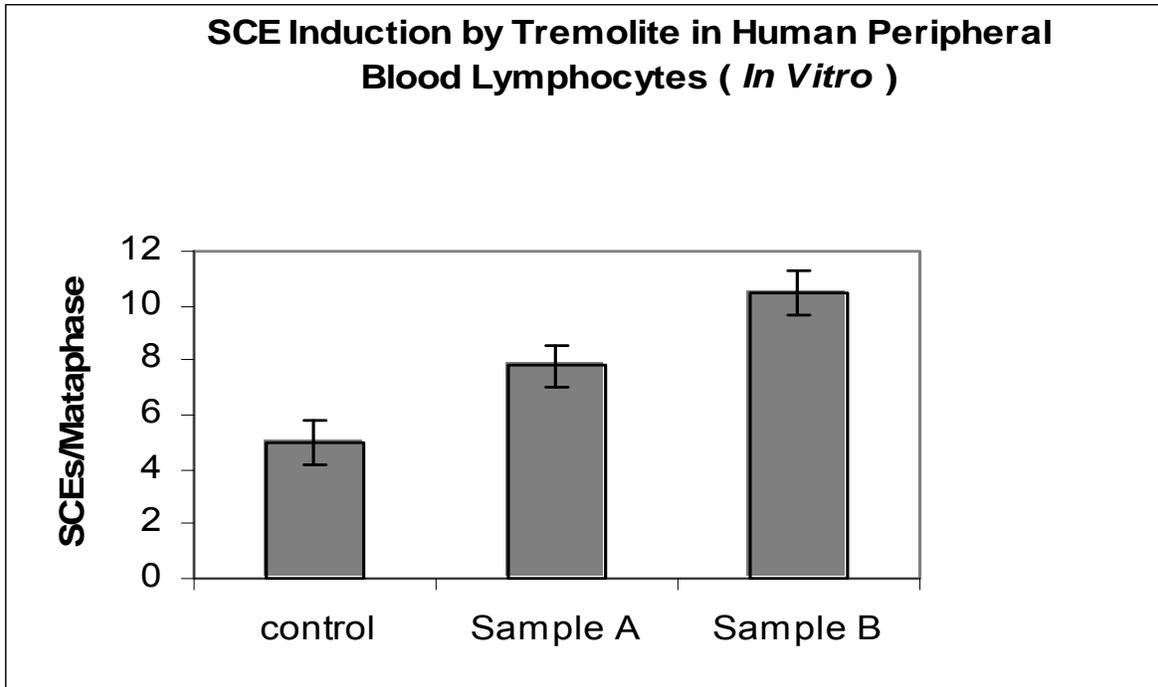


Figure 9: SCE induction by tremolite in human Peripheral Blood Lymphocytes in vitro in Rajasthan

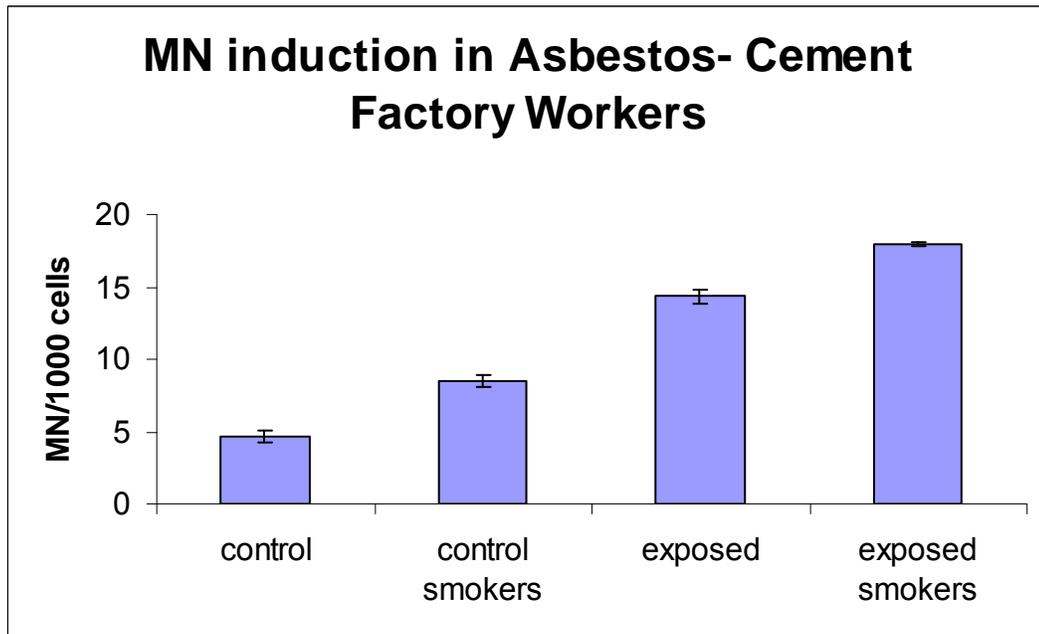


Figure 10: MN induction in asbestos-cement factory workers (UP Asbestos Ltd., Lucknow)

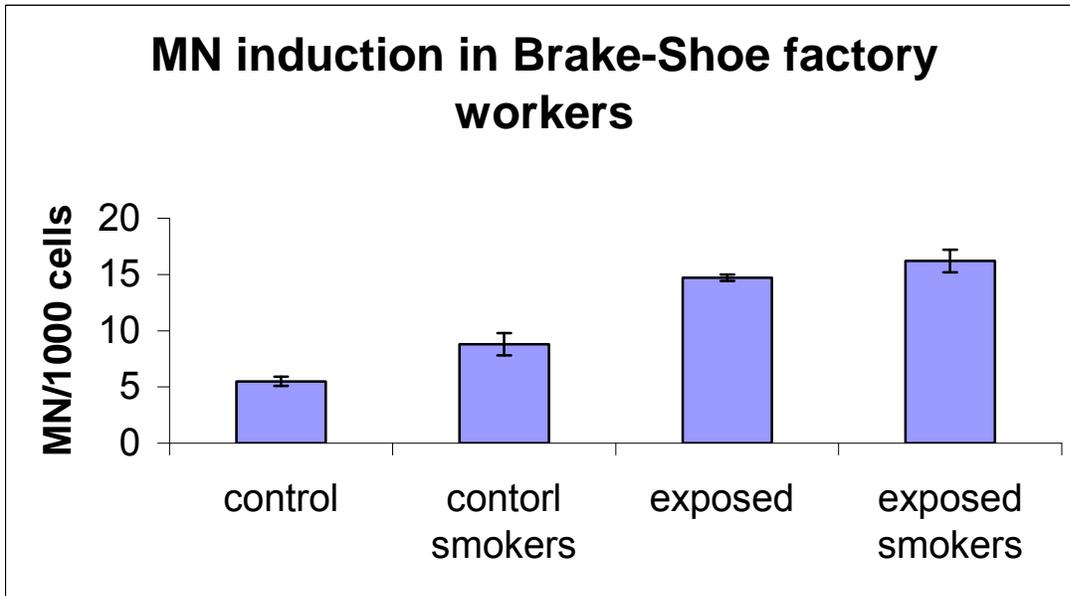


Figure 11: MN induction in brake-shoe factory workers (Allied Nippon Pvt. Ltd., Ghaziabad)

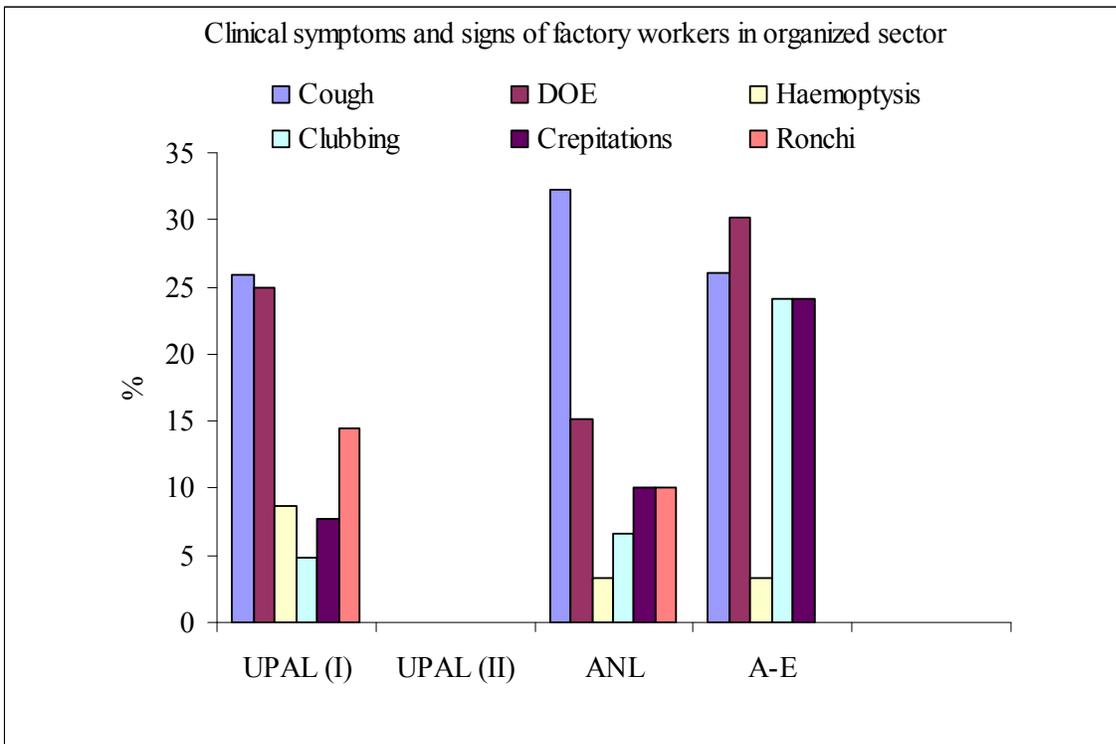


Figure 12: Clinical symptoms and signs of factory workers in organized sector

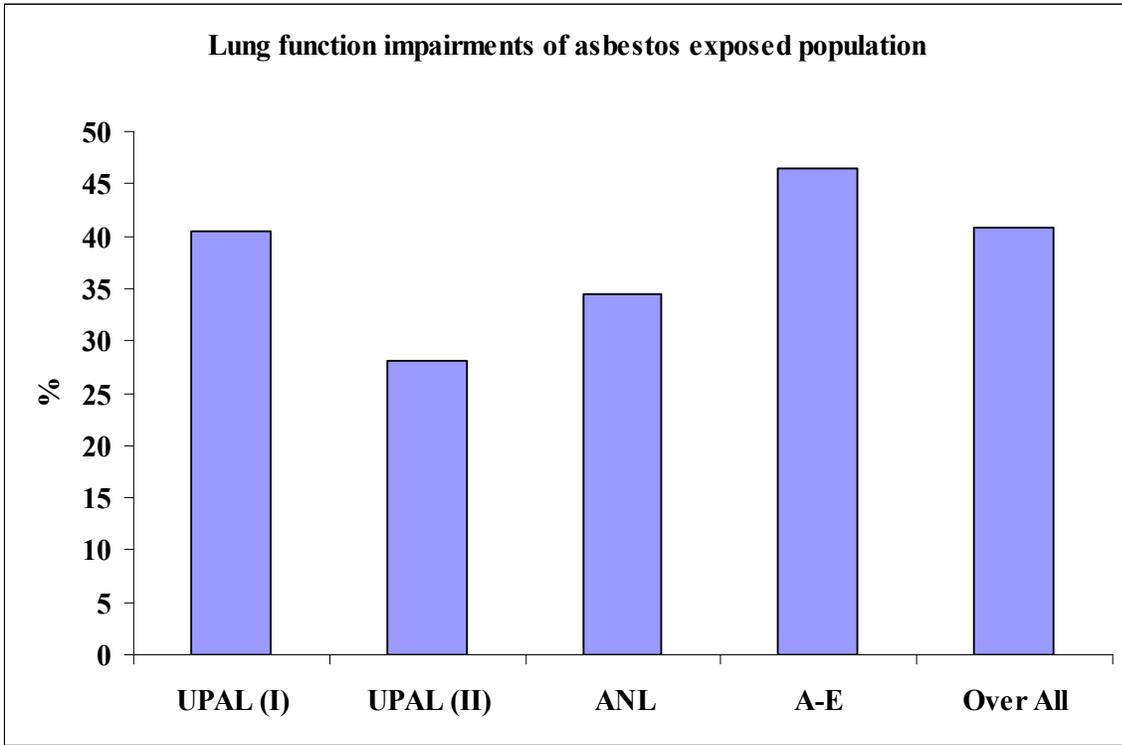


Figure 13: Lung function impairments of asbestos exposed population

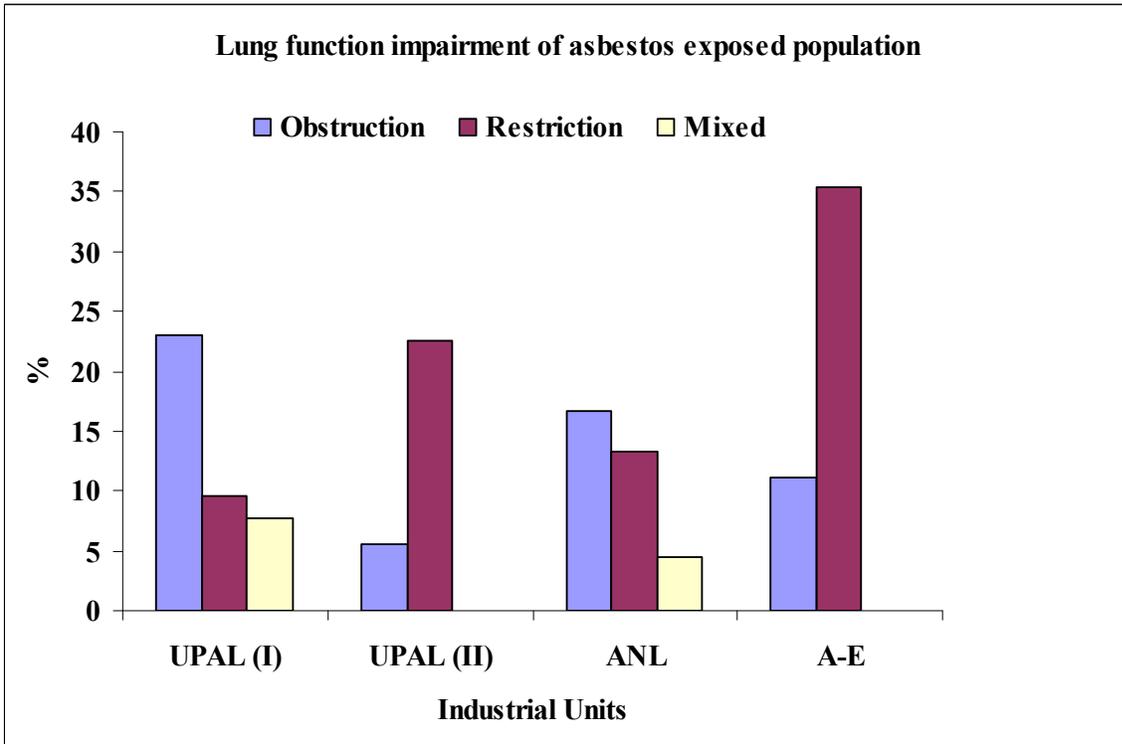


Figure 14: Lung function impairments of asbestos exposed population

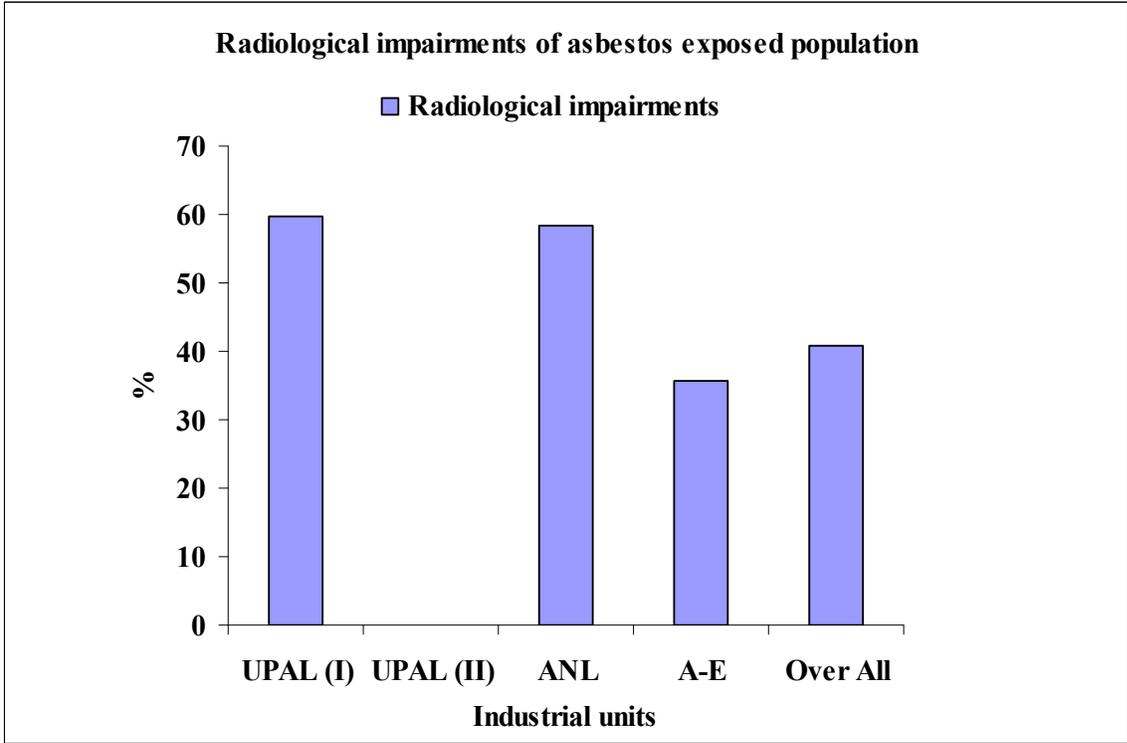


Figure 15: Radiological impairments of asbestos exposed population

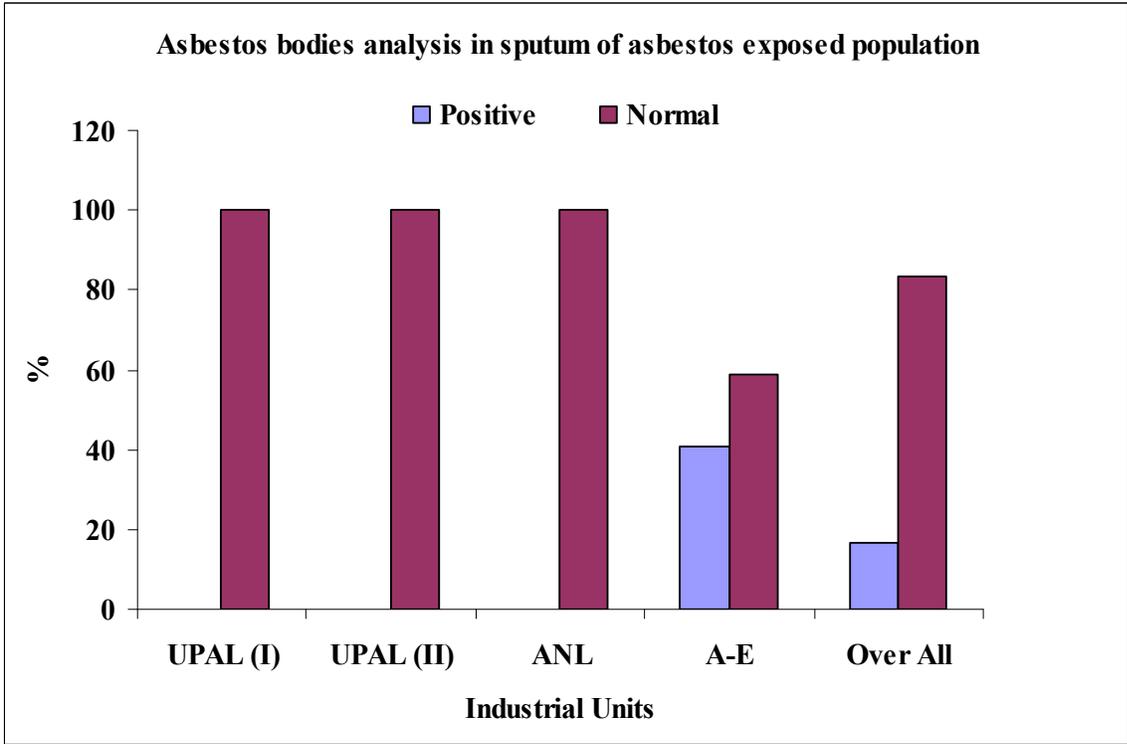


Figure 16: Asbestos bodies analysis in sputum of asbestos exposed population

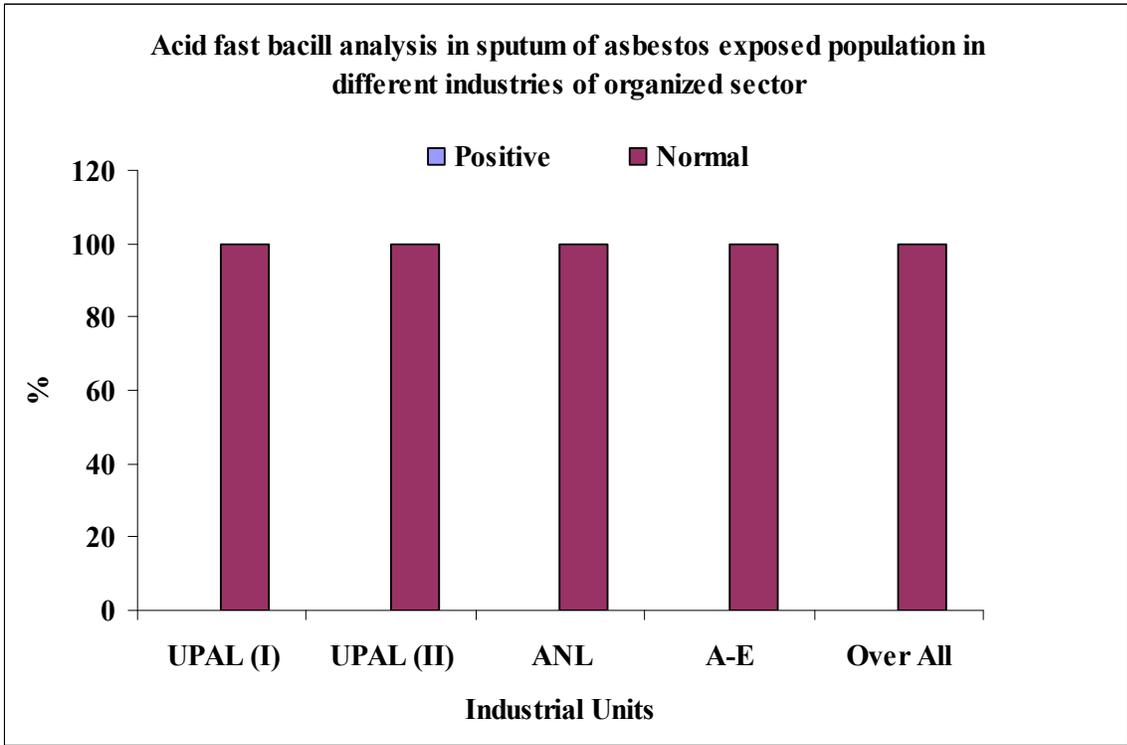


Figure 17: Acid fast bacilli in sputum of asbestos exposed population in different industries of organized sector

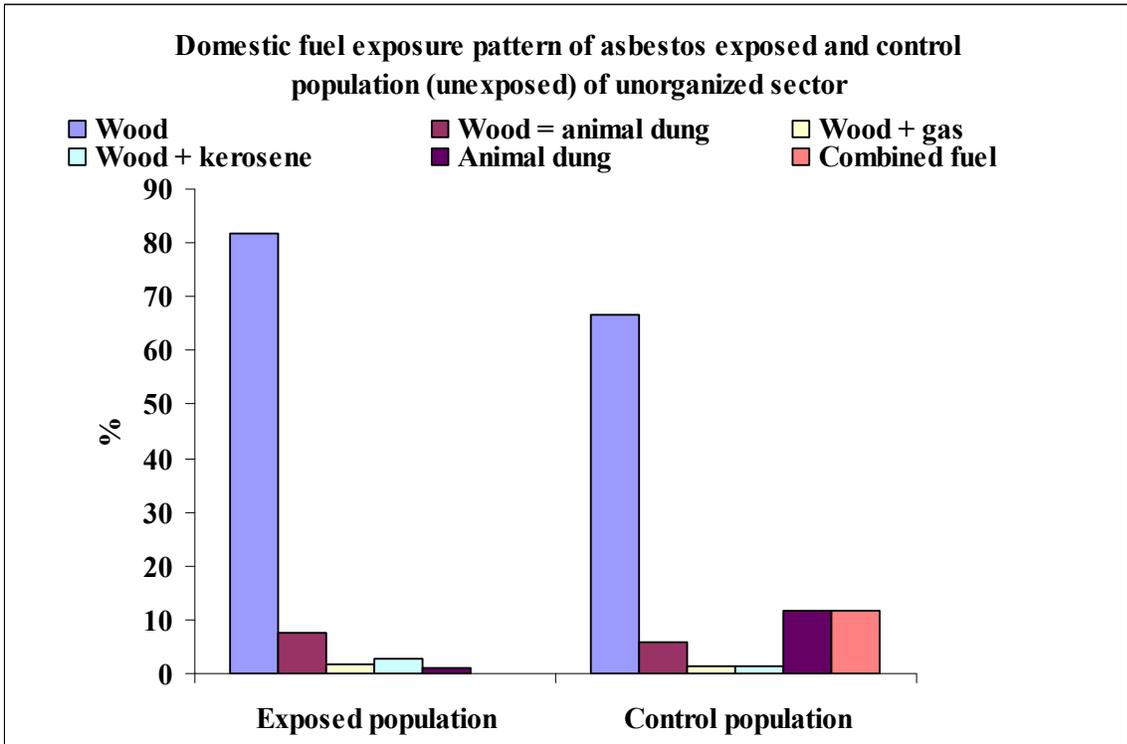


Figure 18: Domestic fuel exposure pattern of asbestos exposed and control population (unexposed) of unorganized sector.

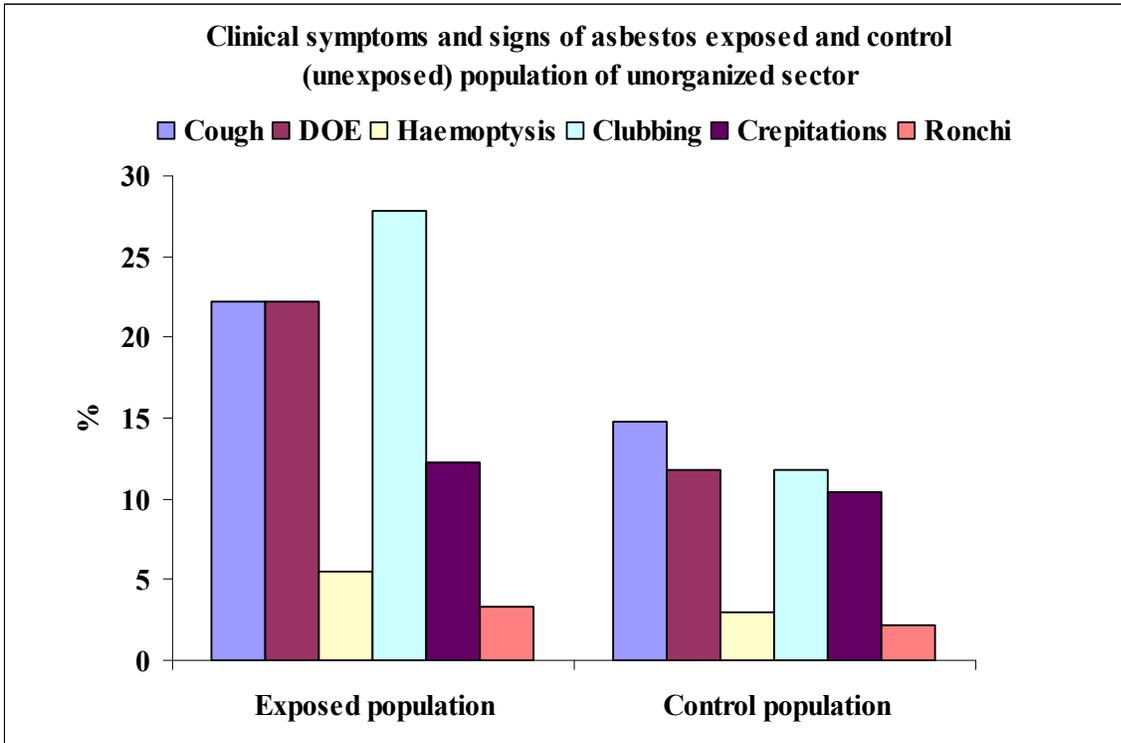


Figure 19: Clinical symptoms and signs of asbestos exposed and control (unexposed) population of unorganized sector.

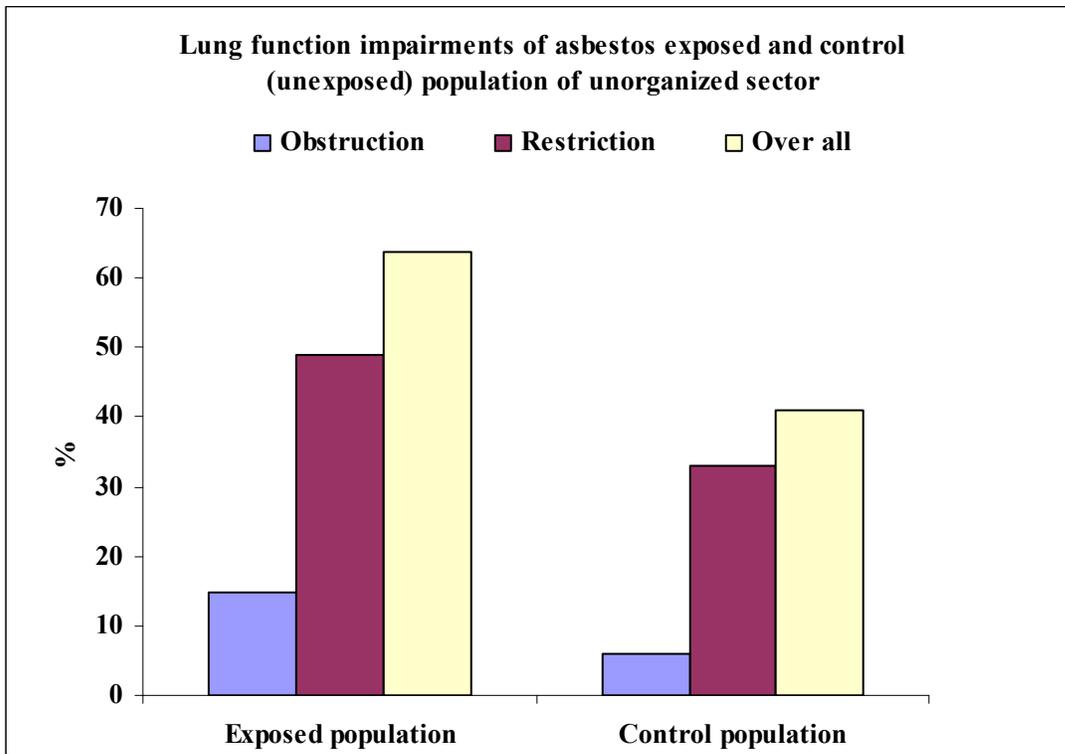


Figure 20: Lung function impairments of asbestos exposed and control (unexposed) population of unorganized sector

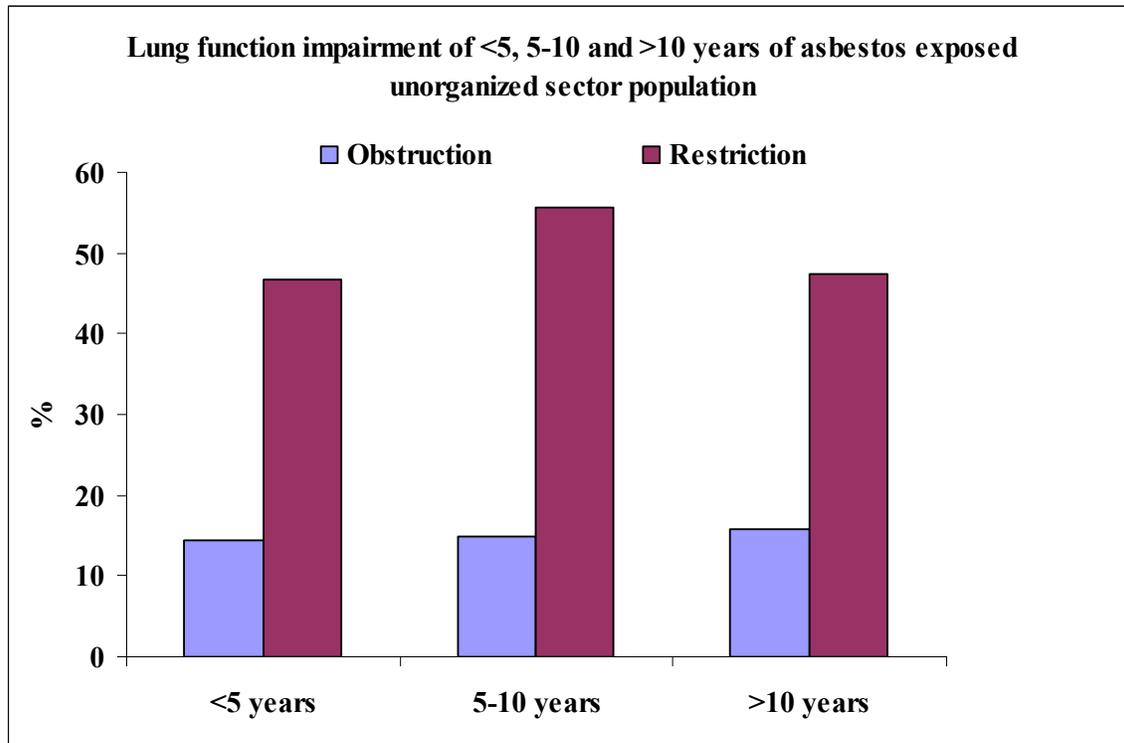


Figure 21: Lung function impairment of <5, 5-10 and >10 years of asbestos exposed unorganized sector population.

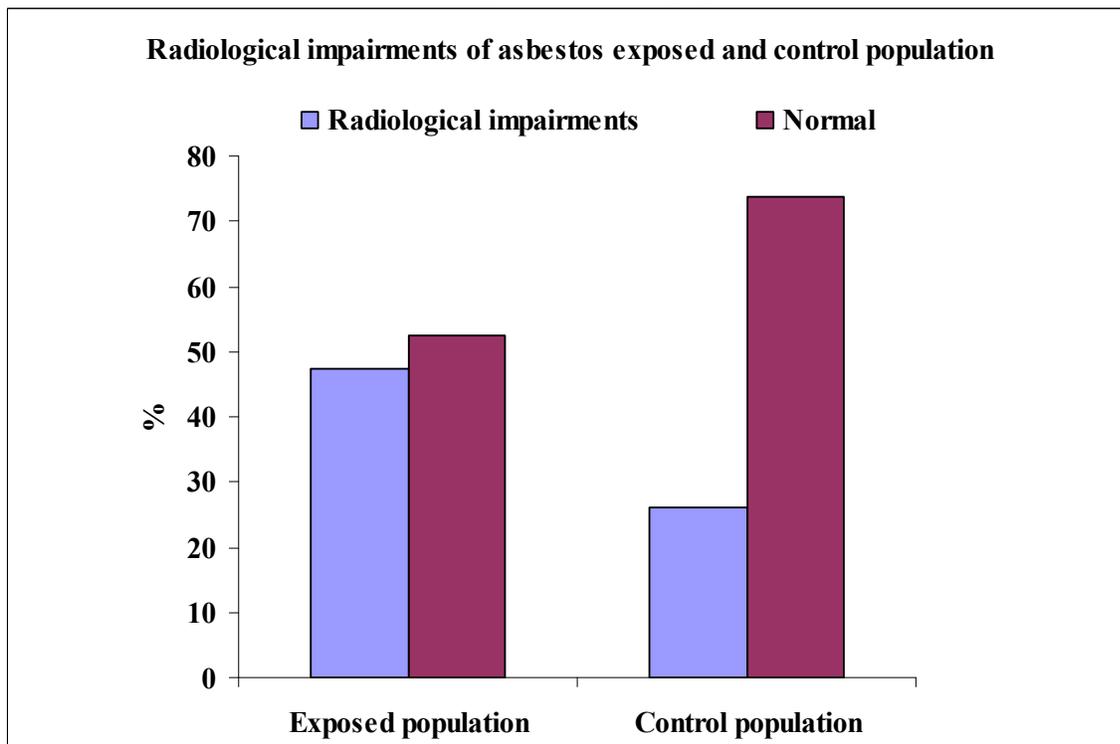


Figure 22: Radiological impairments of asbestos exposed and control population.

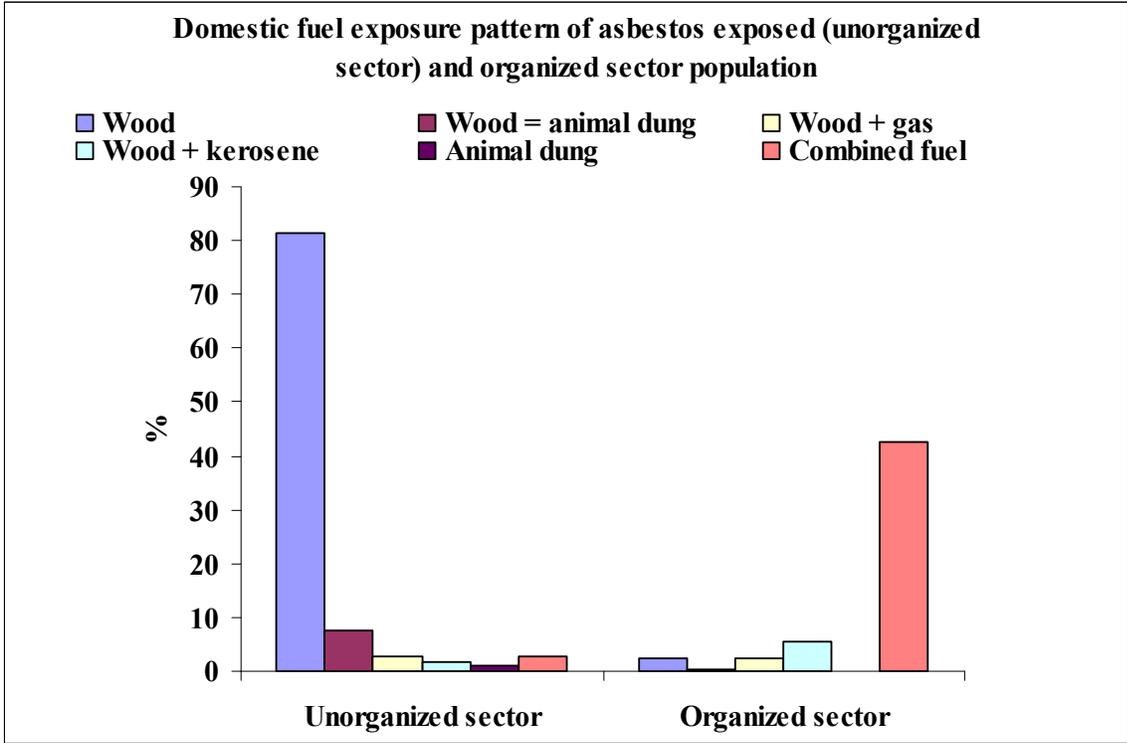


Figure 23: Domestic fuel exposure pattern of asbestos exposed unorganized sector and organized sector population.

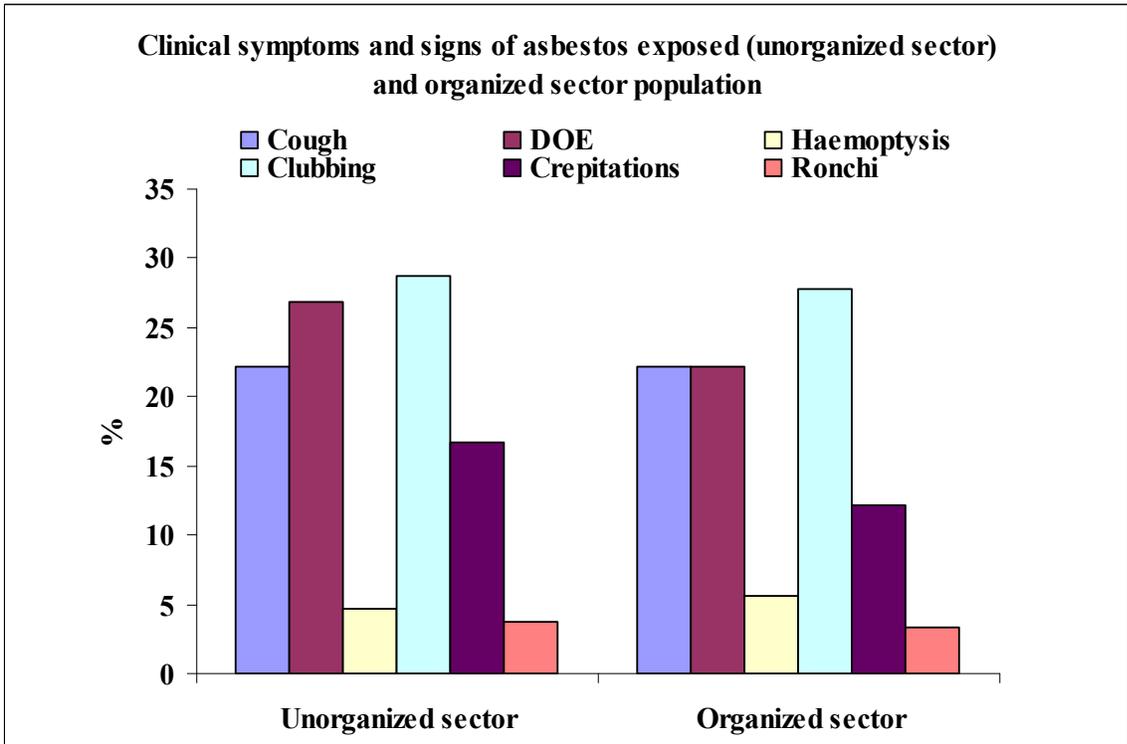


Figure 24: Clinical symptoms and signs of asbestos exposed unorganized sector and organized sector population.

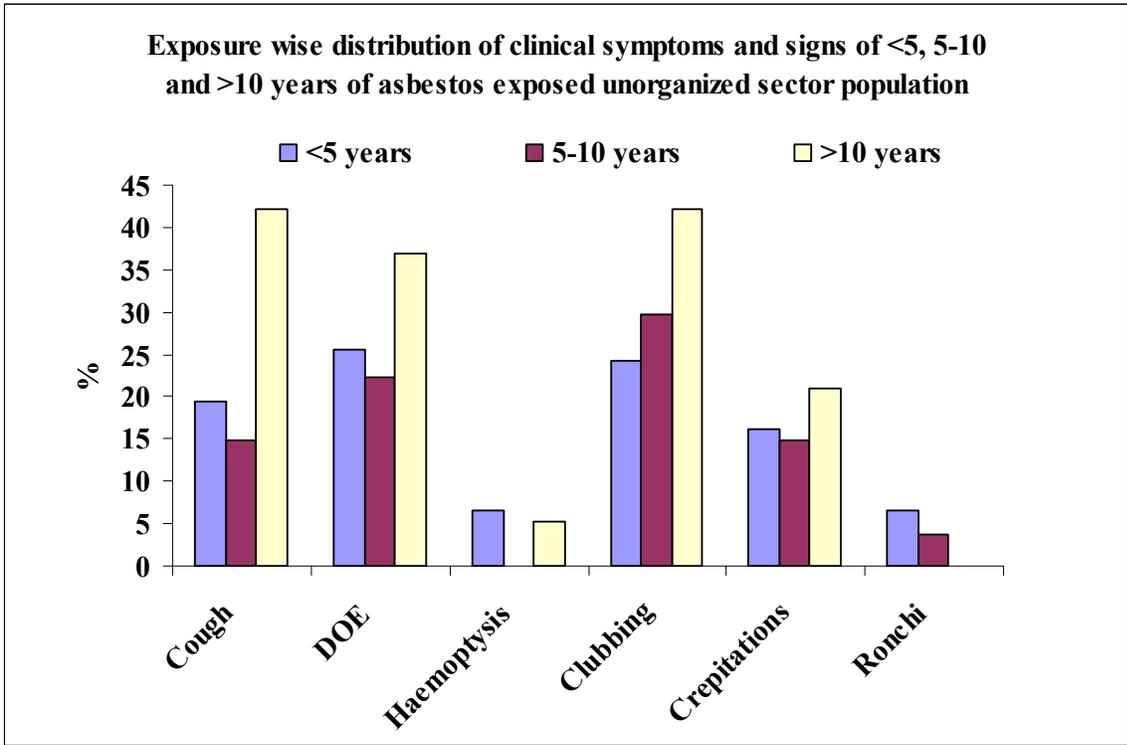


Figure 25: Exposure wise distribution of clinical symptoms and signs of <5, 5-10 and >10 years of asbestos exposed unorganized sector population.

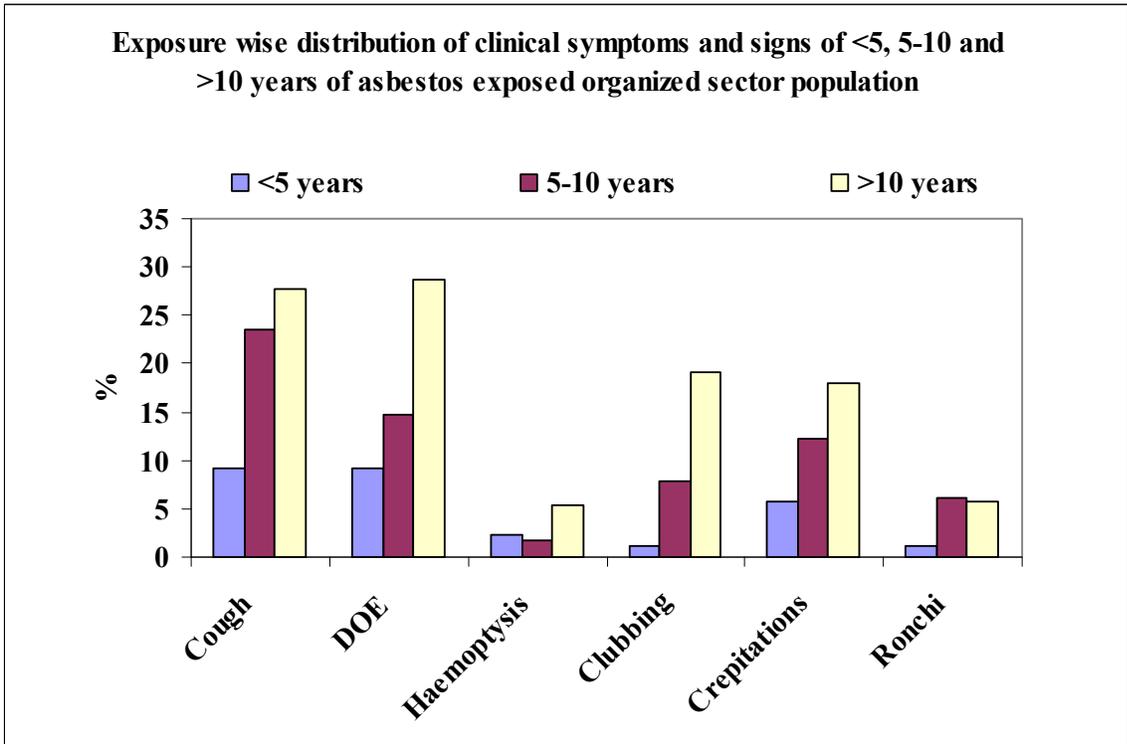


Figure 26: Exposure wise distribution of clinical symptoms and signs of <5, 5-10 and >10 years of asbestos exposed organized sector population.

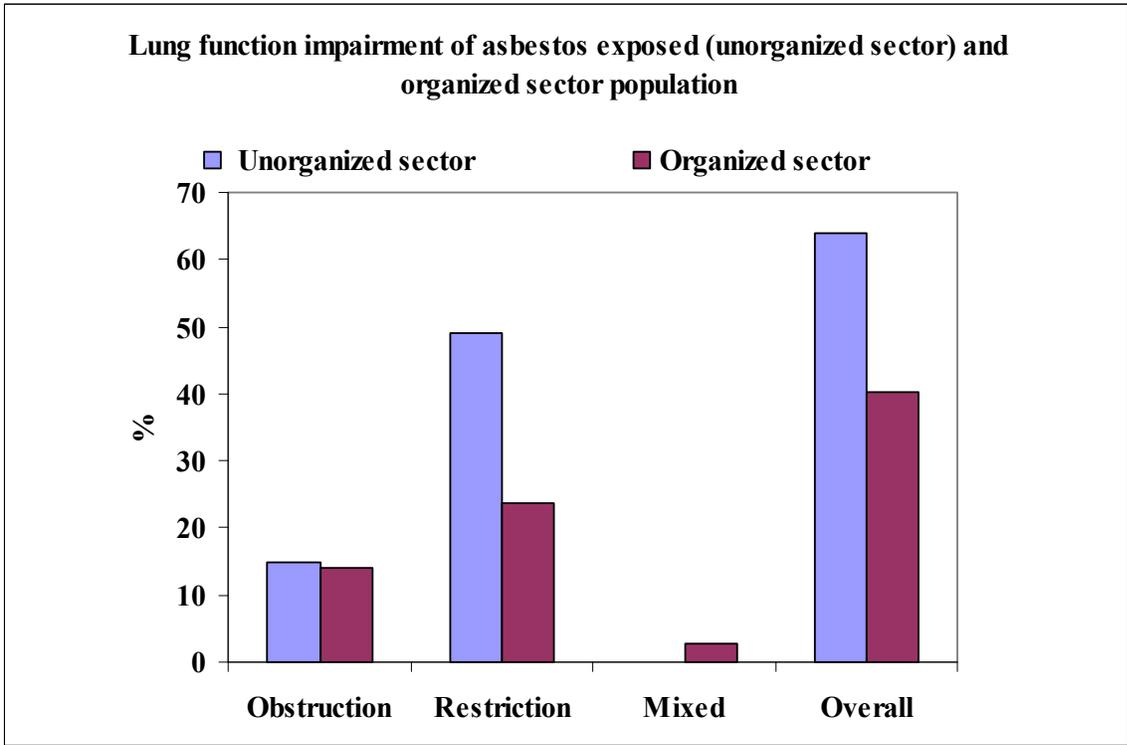


Figure 27: Lung function impairments of asbestos exposed unorganized sector and organized sector population.

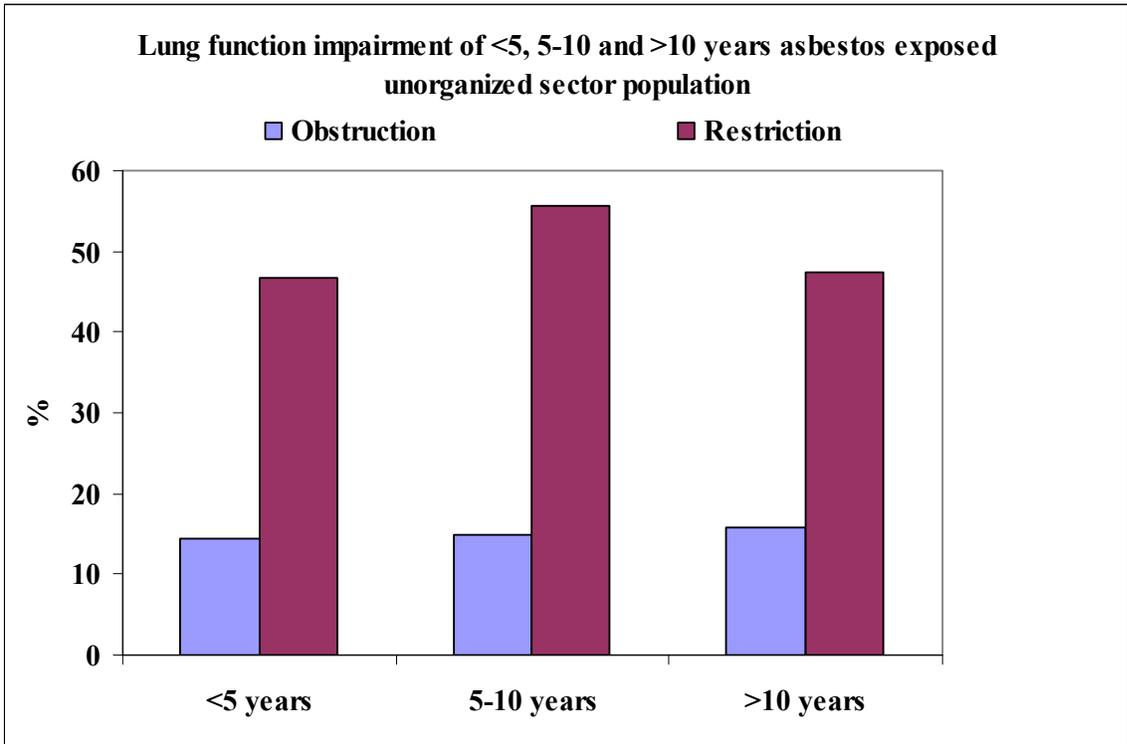


Figure 28: Lung function impairment of <5, 5-10 and >10 years asbestos exposed unorganized sector population.

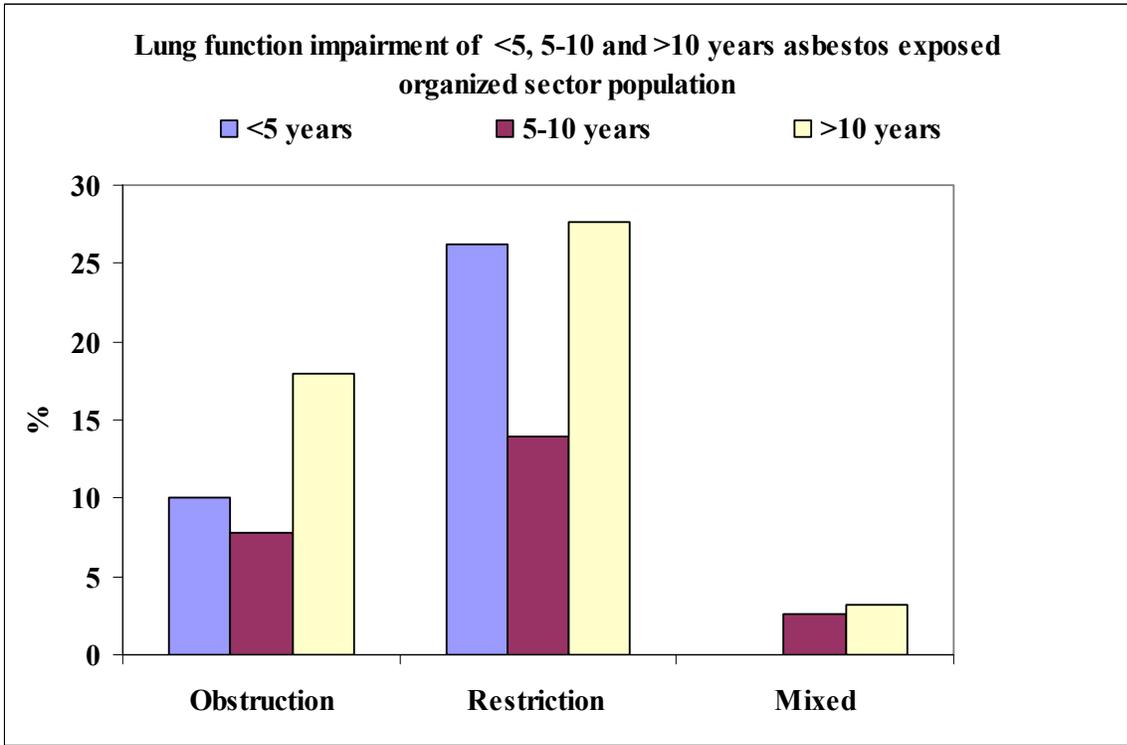


Figure 29: Lung function impairment of <5, 5-10 and >10 years asbestos exposed organized population .

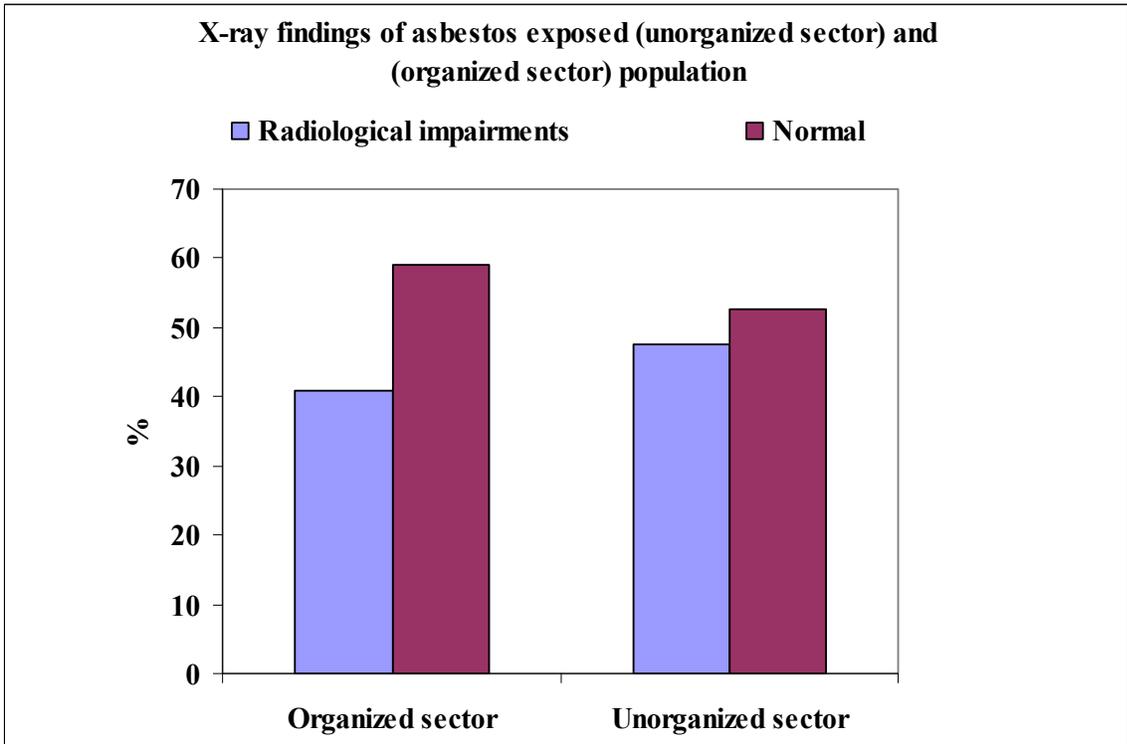


Figure 30: X-ray findings of asbestos exposed unorganized sector and organized sector population.



Picture 1: Showing asbestos-bearing stones spread open in an unorganized asbestos mill.



Picture 2: Showing personal sampler on a woman working in an unorganized asbestos mill. Working woman can be seen without personal protection as well as her hands full of asbestos powder.



Picture 3: Showing unorganized asbestos milling unit. Asbestos powder can be seen on the naked hands of a working woman.



Picture 4: Showing kids playing in an unorganized asbestos milling unit. Hands of the working woman can be seen full of asbestos powder.



Picture 5: Unorganized asbestos unit.



Picture 6: Unorganized manufacturing asbestos unit in Beawer, Rajasthan



Picture 7: Amphibole rich rock collected from ABR Pile in open place of unorganized asbestos mill.

References

1. Agency for Toxic Substances and Disease Registry, ATSDR. Toxicological profile for asbestos. Update (Final Report) (2001). Public Health Service, U.S. Department of Health and Human Services Atlanta, GA: 146 NTIS Accessories No. PB/2001/109/01, USA.
2. Ahmad, I., Krishnamurthy, K., Arif, J.M., Ashquin, M., Mahmood, N., Athar, M., Rahman, Q., (1994). Augmentation of chrysotile-induced oxidative stress by BHA in mice lungs. *Fd. Chem. Toxic.* 33; 209-215.
3. Albelda, S.M., Epstein, D.M., Geftter, W.B., Miller, W.T.Y., (1982). Pleural thickening. Its significance and relationship to asbestos dust exposure. *A rev respir dis.* 126: 621 - 624.
4. American Thoracic Society, (1987). ATS statement; Standardizations of spirometry: Update. *Am. Rev. Respir. Dis.* 136: 1286 - 1296.
5. Arif, J.M., Khan, S.G., Ashquin, M., Rahman, Q., (1993). Modulation of macrophage-mediated cytotoxicity by kerosene soot: Possible role of reactive oxygen species. *Environ. Res.* 61: 232-238.
6. Arif, J.M., Khan, S.G., Joshi, S.D., Ahamad, I., Rahman, Q., (1997). Effect of kerosene and its soot on the chrysotile-mediated toxicity to the rat alveolar macrophages. *Environ. res.* 72: 151 – 161.
7. Arif, J.M., Khan, S.G., Mahmood, M., Aslam, M., Rahman, Q., (1994). Effect of co-exposure of asbestos and kerosene soot on pulmonary drug-metabolizing enzyme system. *Environ. health perspect.* 102 (5): 181 – 183.
8. Arif, J.M., Mahmood, N., Khan, S.G., (1996). Asbestos and lung diseases: A mechanistic approach II. In “Sourcebook of asbestos diseases” Ed. G. A. Peters and B. J. Peters, Vol. 8, Butterworth Legal Pub. USA.
9. Arif, J.M., Sikandar, G.K., Aslam, M., Mahmood, M., Rahman, Q., (1992). Diminution in kerosene-mediated induction of drug metabolizing enzymes by asbestos in rat lungs. *Pharmacol. Toxicol.* 71: 37 – 40.
10. Armstrong, B.K., De-Klerk, N.H., Musk, A.W., and Hobbs, M.S.T., (1988). Mortality in miners and millers of crocidolite in Western Australia. *Br. J. Ind. Med.* 45 (1): 5-13.

11. Barnhart, S.M., Thornquist, G.S., Omenn, G., Goodman, P., Feigl, Rosenstock, L., (1990). The degree of roentgenographic parenchymal opacities. *Am rev respir dis.* 130: 293 – 301.
12. Barrette, J.C., (1994). Cellular and molecular mechanisms of asbestos carcinogenicity: Implications for biopersistence. *Environ. Health. Perspect.* 102, 19-23.
13. Berry, G., New House, M.L., Antonis, P., (1985). Combined effect of asbestos and smoking on mortality from lung cancer and mesothelioma in factory workers. *Br. J. Ind. Med.* Jan. 42(1): 12-8.
14. British Medical Research Council, (1976). British Medical Research Council Standardized questionnaires on respiratory symptoms. *Br. med. J. (Clin Res).* 2: 1965–1968.
15. Brody, A.R., Hill, L.H., Adkins, B., O'Connor, R.W., (1981). Chrysotile asbestos inhalation in rats: Deposition pattern and reaction of alveolar epithelium and pulmonary macrophages. *Am rev. resp. dis.* 123 (6): 670 - 679.
16. Brown, K., (1994). Asbestos-related disorders. In: Parkes W.R., editor. *Occupational lung disorders 3rd*, ed. Oxford. Butterworth – Heinemann. 411–504.
17. Bureau of Indian Standards (BIS): IS-11450-(1986). Method for determination of air borne asbestos fibres by light microscopy.
18. Byers, T.E., Vena, J.E., Rzepka, T.F., (1984). Prediction of lung cancers for upper lobes: An epidemiologic inquiry. *J. Natl Cancer Inst.* 72: 1271-1275.
19. Cohen, R., (1981). Occupational lung disease: pneumoconiosis. *Occup. health nurs.* 29 (4): 10–13.
20. Craighead, J.E., Abraham, J.L., Churg, A., et al., (1993). The pathology of asbestos-associated diseases of the lungs and pleural cavities: Diagnostic criteria and proposed grading schema. *Arch. Pathol. Lab Med.* 106: 540-597.
21. Dashawara, R.S., Tyagi, R.S., (1992). *Mineral Statistics of Rajasthan.* Govt. of Rajasthan. Pub. Udaipur, 36.
22. Dave, S.K., Bhagia, L.J., Mazumdar, P.K., Patel, G.C., Kulkarni, P.K., Kashyap, S.K., (1996). The Correlation of Chest Radiograph and Pulmonary Function Tests in asbestos Mines and Millers. *Indian J chest dis allied sci.* 38: 81 – 89.

23. De-Klerk, N.H., Armstrong, B.K., Musk, A.W., Hobbs, M.S., (1989a). Cancer mortality in relation to measures of occupational exposure to crocidolite at Wittenoom Gorge in Western Australia. *Br. J. Ind. Med.* 46 (8): 529-536.
24. De-Klerk, N.H., Armstrong, B.K., Musk, A.W., Hobbs, M.S., (1989b). Predictions of future cases of asbestos-related disease among former miners and millers of crocidolite in Western Australia. *Med. J. Aust.* 151: 616-620.
25. De-Klerk, N.H., Musk, A.W., Armstrong, B.K., Hobbs, M.S.T., (1991). Smoking, exposure to crocidolite, and the incidence of lung cancer and asbestosis. *Br. J. Ind. Med.* 48: 412-417.
26. Dion, C., and Perrault, G., (1994). Comparison of four methods for determination of asbestos fibre concentrations in work place atmospheres by phase contrast microscopy. *Appl. Occup. Environ. Hyg.* 9: 707-711.
27. Doll, R., Peto, R., (1976). Mortality in relation to smoking: 20 years' Observations on male british doctors. *Br. Med. J.* 2: 1525-1536.
28. Eache, C.A., Groff, J.H., (1997). Proficiency Analytical Testing Program Report, February 28. *Am. J. Ind. Hyg. Assoc.* 58:455-456.
29. Eastman, A., Mossman, B.T., Bresnick, E., (1983). Influence of asbestos on the uptake of benzo (a) pyrene and DNA alkylation in hamster tracheal epithelial cells. *Cancer Res.* 43 (3): 1251-1255.
30. EPA, (1987). Asbestos containing material in schools: final rule and notice.40 CFR 763.121.United States Environmental protection agencies, Washington, DC.
31. Ernster, V.L., Mustacchi, P., Osann, K.E., (1994). Epidemiology of lung cancer. In *textbook of respiratory Medicine.* 2: 1505 –27.
32. Fatima, N., Jain, A.K., Rahman, Q., (1991). Frequency of sister chromatid exchanges and chromosomal aberration in asbestos cement workers. *British Journal of Industrial Medicine:* 48 (2): 103-105.
33. Fenech, M., (1993). The cytokinesis-block micronucleus technique: a detailed description of the method and its application to genotoxicity studies in human populations. *Mutat. Res.* 285 (1): 35-44.

34. Fenech, M., Morley, A.A., (1985). Measurement of micronuclei in lymphocytes .Mutat.Res. 285(1): 35-44.
35. Frazer, R.G., Pare, J.A., Pare, P.D., Frazer, R.S., Genesex, G.P., (1990). diagnosis of diseases of the chest, philadelphia, W.E. Saunders, 3: 2346–2353.
36. Gross, P., De-Treville, R.T.P., Cralley, L.J., Davis, J.M.G., (1999). Pulmonary ferruginous bodies: Development in response to filamentous dusts and a method of isolation and concentration. Asia. path. 85: 539-546.
37. Hammond, E.C., Selikoff, I.J., Seidman, H., (1979). Asbestos exposure, cigarette smoking and death rates. Ann. N. Y. Acad. Sci. 330: 473-490.
38. Hart, G.A., Kathman, L.M., Hesterberg, T.W., (1994). In vitro cytotoxicity of asbestos and man-made vitereouss fibres: role of fibre length, diameter and composition. Carcinogenesis. 15: 971-977.
39. Heddle, J.A., Hite, M., Krikhart, B., Mavounin, K., Macgeror, J.T., Newell, G.W., Salamone, M.F., (1983). The induction of micronuclei as a measure of genotoxicity. Mutat.Res. 123: 61-118.
40. Hilledral, G., (1980). Pleural plaques, occurrence, exposure to asbestos and clinical importance. Acta Univ ersitatis Upsaliensis. 363: 158-178.
41. Hobson, J., Gilks, B., Wright, J., Churg, A., (1988). Direct enhancement by cigarette smoke of asbestos fibre penetration and asbestos induced epithelial proliferation in rat tracheal explants. J Nat Cancer Inst. 80: 518-521.
42. Hunt, J., Pooley, D.F., Richards, J.R., (1981). Biological reactivity of calcium silicate composites: in vitro studies. Environ. Res. 26: 51-68.
43. IARC (International Agency for Registry of Cancer), (1987). Tobacco smoke: IARC monographs on the evaluation of carcinogenic risk of chemicals of humans. Supp. 7: 360-362.
44. IMYB (Indian Mineral Year Book), (1989). IBM, Nagpur: 282-283 and 284 resp.
45. International Labour Office, (1980). Guidelines for the use of ILO International classification of radiographs of pneumoconioses. Geneva: ILO (Occupational Safety and Health series 22 Rev 80).

46. IPCS, (1986). Environmental Health Criteria 53 – Asbestos and other Natural mineral fibres. World Health Organization, Geneva, International Programme on Chemical Safety, 194.
47. Kamp, D.W., Greenberger, M.J., Sbalchierro, J.S., Sbalchierro, J.S., Preusen, S.E., Weitzman, S.A., (1998). Cigarette smoke arguments asbestos – induced alveolar epithelial cell injury: role of free radicals. *Free Rad. Biol. Med.* 25: 728.
48. Kamp, D.W., Weitzman, S.A., (1998). Asbestosis: clinical spectrum and pathogenic mechanisms. *Proc. Soc. Exp. Biol. Med.* 214: 12-26.
49. Kamp, D.W., Weitzman, S.A., (1999). Occasional review: The molecular basis of asbestos induced lung injury. *Thorax.* 54 (2): 638 - 652.
50. Kelburn, K.H., (2000). Prevalance and features of advanced asbestosis (ILO profusion scores above 2/2). International Labour Office. *Arch. Environ. Health.* 55 (2): 104–108.
51. Kilburn, K.H., (2000). Indoor air effects after building renovation and in manufactured homes. *Am. J. Med. Sci.* 320 (4): 249-54.
52. Kulkarni, G.K., (2001). Asbestos-ban or not to ban? *Ind. J.Occup. Environ. Med.* 5: 1.
53. Landrigan, P.J., (1998). Asbestos—still a carcinogen. *N Engl J Med.* 338: 1618–1619.
54. Lasalle, P., Coosset, P., Aerts, C., (1990). Abnormal secretion of interleukin-1 and tumor necrosis factor alpha by alveolar macrophage in coal workers pneumoconiosis; Compassion between simple pneumoconiosis and progressive massive fibrosis. *Exp. Lung. Res.* 16: 73 - 80.
55. Last, J.A., Reiser, K.M., (1984). Collagen biosynthesis. *Environ Health Perspect.* 55: 169 - 177.
56. Liddel, F., Thomas, D., Gibbs, (1984). Fibre exposure and mortality from pneumoconiosis respiratory and abdominal malignancies in chrysotile production in Qubec, 1926-1975 Singapore. *Ann. Acad. Med.* 13: 339–344.
57. Lohani, M., Dopp, E., Weiss, D.G., Schiffmann, D., Rahman, Q., (2000). Kerosene soot genotoxicity: enhanced effect upon co-exposure with chrysotile asbestos in Syrian hamster embryo fibroblasts. *Toxicology Letter.* 114: 111-116.

58. Luce, D., Bugel, I., Goldberg, P., Goldberg, M., Salomon, C., Billon-Galland, M.A., Nicolau, J., Quenel, P., Fevotte, J., Brochard, P., (2000). Environmental exposure to tremolite and respiratory cancer in New Caledonia: a case-control study.
59. Mansingha, B.K., Ranawat, P.S., (1996). Mineral economics and occupational health hazards of the asbestos resources of Rajasthan. *J Geol Soc (India)* 47: 375-382.
60. Mc Donald, A.D., Mc Donald, J.C., Armstrong, B., Cherry, N., Delorme, C.D-Nolin, A., Robert, D., (1987). Occupation and pregnancy outcome. *Br. J. Ind. Med.* 44: 521-6.
61. Mc Fadden, D., Wright, J., Chung, A., (1986). Cigaretta smoke increases the penetration of asbestos fibres into airway walls. *Am J. Pathol* 123: 95-99.
62. McDonald, J.C., McDonald, A.D., Hughes, J.M., (1999). Chrysotile, tremolite and fibrogenicity. *Ann Occup Hyg.* 43 (7): 439-42.
63. Medical Laboratory Manual for Tropical Countries: Microbiology, (1985). Microbiology Monica Chessbrough. Zeil – Neelsen Staining Technique. 32: 34-36.
64. Membrane Filter method for determination of the air borne asbestos fibre concentrations in work environment by light microscopy (Membrane Filter Method) BIS: 11450(1986).
65. Miller, A., (1993). Pulmonary function in asbestosis and asbestos-related pleural disease. *Environ. Res.* 61 (1): 1-18.
66. Morgan, A., Davis, P., Wagner, J.C., Berry, G., Holmes, A., (1977). The biological effects of magnesium leached chrysotile asbestos. *J. Experl. Pathol.* 58: 465.
67. Mossman, B.T., (1994). Carcinogenesis and related cell and tissue responses to asbestos: a review. *Ann Occup Hyg.* 38: 617-624.
68. Mossman, B.T., Bignon, J., Corn, M., (1990). Asbestos: scientific developments and implications for public policy. *Science.* 247: 294-301.
69. Mossman, B.T., Gee, J.B.L., (1989). Asbestos related diseases. *N. Engl J Med.* 320:1724-1730.
70. Mossman, B.T., Kamp, D.W., Weitzman, S.A., (1996). Mechanisms of carcinogenesis and clinical features of asbestos-associated cancers. *Cancer Invest.* 14: 464-478.

71. Mossman, B.T., Kamp, D.W., Weitzman, S.A., (1996). Mechanisms of carcinogenesis and clinical features of asbestos-associated cancers. *Cancer Invest.* 14: 464–78.
72. Mossman, B.T., Marsh, J.P., Sesko, A., Hill, S., Shatos, M.A., Doherty, J., Petruska, J., Adler, K.B., Hemenway, D., Mickey, R., Kogan, E., (1990a). Inhibition of lung injury, inflammation and interstitial pulmonary fibrosis by polyethylene glycol-conjugated catalase in a rapid inhalation model of asbestosis. *Am. Rev. Respir. Dis.* 1141: 1266-1271.
73. Okayasu, R., Takahashi, S., Yamada, S., Hei, T.K., Ullrich, R.L., (1999). Asbestos and DNA double strand breaks. *Cancer res.* 59: 298-300.
74. Oshimura, M., Hesterberg, T.W., Barrett, J.C., (1986). An early nonrandom karyotypic change in immortal syrian hamster cell lines transformed by asbestos: trisomy of chromosome 11 *Cancer cytogenet.* 22: 225-231.
75. Pooley, F.D., (1972). Asbestos bodies, their formation, composition and character. *Environ. Res.* 5: 363-369.
76. Raffn, E., Lynge, E., Korsgaard, B., (1993). Incidence of lung cancer by histological type among asbestos cement workers in Denmark. *Br. J. Indust. Med.* 50: 85-89.
77. Rahman, Q., (1995). Asbestos: An occupational and environmental carcinogen. In: "Mining and Environment" (Eds. B. B. Dhar and D. N. Thakur), Oxford & IBH Publ. Co. Pvt. Ltd., New Delhi: 549 - 564.
78. Rahman, Q., Arif, J.M., Mahmood, N., Khan, S.G., (1993). Asbestos and lung diseases: A mechanistic approach II. In "Sourcebook of asbestos diseases" Ed. G. A. Peters and B. J. Peters, Vol. 8, Butterworth Legal Pub. USA.
79. Rahman, Q., Athar, M., (1994). Asbestos-induced carcinogenesis: An Update. *Advances in Biosciences.* (Review).
80. Rahman, Q., Dopp, E., Lohani, M., Schiffmann, D., (2000). Occupational and environmental factors enhancing the genotoxicity of asbestos. *Inhal. Toxicol.* 2: 157-165.
81. Rahman, Q., Narwood, J., Hatch, G., (1997). Evidence that exposure of particulate air pollutants to human and rat alveolar macrophages lead to different oxidative stress. *Biochem. Biophys. Res. Comm.* 240: 669-672.

82. Rahman, Q., Prasad, R., Das, M., Pandey, U.S., Lohani, M., Ashquin, M., (1998). Follow-up study in asbestos-cement industry with special emphasis on preventive and diagnostic measures. Project report submitted to UP-CST, India.
83. Rajhans, B.N., (1993). The organized and the unorganized sectors contributions and compensations. In: Patel, B.B., Societal security for unorganized labor, Oxford & IBH Publishing, and New Delhi, 68 - 70.
84. Rajhans, G.S., Sullivan, J.L., (1981). Asbestos sampling and analysis. *Ann. A. Science* : 105.
85. Ramanathan, A.L., Subramanian, M., (2001). Present status of asbestos mining and related health problems in India - a survey. *Ind. Health*. 39: 309 - 315.
86. Ramanathans, A.L., and Subramanian V., (2001). Present status of asbestos mining and related health problems in India - A survey *Ind. Health*. 39: 309-315.
87. Rastogi, S.K., Mathur, N., Clark, S.H., (1983). Ventilatory norms in healthy industrial male workers. *Ind. J. Chest. Dis. Allied. Sci.* 25: 186 - 195.
88. Rastogi, S.K., Mehrotra, N.K., Gupta, B.N., Rahman, Q., Kumar, A., Husain, T., (1990). Prevalence of respiratory impairment in asbestos workers, *Indian J. Environ. Protect.* 10: 20–25.
89. Richter, E.D., Tuch, H., Shabbat, Z., Weiler, D., (1986). Smoking, morbidity, and pulmonary function in a group of ex-asbestos workers: a pilot study. *Am J Ind Med.* 10: 515 - 523.
90. Samet, J.M., Epler, G.R., Gensler, E.A., Rosner, B., (1979). Absence of synergism between exposure to asbestos and cigarette smoking in asbestosis. *Am. Rev. Respir. Dis.* 120: 75 - 82.
91. Saracci, R., (1977). Asbestos and lung carcinoma: An analysis of the epidemiological evidence on the asbestos-smoking Interaction. *Int. J. Cancer.* 20: 323-31.
92. Schneider, J., Rodelsperger, K., Bruckel, B., Kayser, K., Voitowitz, H.J., (1998). Environmental exposure to tremolite asbestos : pleural mesothelioma in two Turkish workers in Germany.
93. Schnitzen, R.J., Pundsack, F.L., (1970). Asbestos hemolysis. *Environ. Res.*3: 1-3.

94. Schulz, C.O., (1994). Silicon and silicates, including asbestos. In: Patty's Industrial Hygiene and Toxicology, 4th ed, Volume II, Part E, G.D. Clayton and F.E. Clayton, Eds. New York, John Wiley & Sons. 849-864.
95. Selikoff, I.J., Hammond, E.C., Churg, J., (1968). Asbestos exposure, smoking and neoplasia. JAMA. 204:106-112.
96. Selikoff, I.J., Lee, D.N.K., (1978). "Asbestos and disease" New York: Academic Press.
97. Selikoff, I.J., Sedman, H., Hammond, E., (1980). Mortality effect of cigarette smoking among amosite asbestos factory workers. J. N. C. I. 65: 507-513.
98. Spurzen, J.R., Saltini, C., Rom, W., Winchester, R.J., Crystal, R.G., (1987). Mechanisms of macrophage accumulation in the lungs of asbestos-exposed subjects. Am. Rev. Respir. Dis. Aug. 136: 276-80.
99. Suganuma, N., Kusaka, Y., and Hosoda, Y., (2001). The Japanese classification of computed tomography for pneumoconiosis with standard films: comparison with ILO international classification of radiographs for pneumoconiosis. J Occup Health. 43: 24-31.
100. Timbrell, V., (1982). Deposition and retention of fibres in the human lung. Ann. Occup. Hyg. 26(1-4): 347 - 69.
101. Tokuhata and Lilienfeld, (1963). Familial aggregation of lung cancer in humans. J.Natl.Cancer Inst. 30: 289-312.
102. United States Environmental Protection Agency, (1993). Method for the determination of asbestos in bulk building materials. Pub. No. EPA/600/R-93/116.
103. US–Environmental Protection Agency, (1982). "Interim Method for the Determination of Asbestos in Bulk Insulation Samples, Method 600/M4-82-020.
104. US–Environmental Protection Agency, (1985). Measuring Airborne Asbestos Following an Abatement Action. Pub. No. EPA 600/4-85-049.
105. Vainio, H., Boffetta, P., (1994). Mechanisms of the combined effect of asbestos and smoking in the etiology of lung cancer. Scand J Work Environ Health. 20(4): 235-242.
106. Wang, (2001). Pulmonary function in long–term asbestos. J Occup Environ Med. 43: 23 - 29.

107. Wang, Q., Fan, J., Wang, H., Liu, S., (2000). DNA damage and activation of c-ras in human embryo lung cells exposed to chrysotile and cigarette smoking solution. *J. Environ. Pathol. Toxicol. Oncol.* 19: 13-19.
108. Warheit, D.B., Chang, L.Y., Hill, L.H., Hook, G.E., Grapo, J.D., Brody, A.R., (1984). Pulmonary macrophage accumulation and asbestos-induced lesions at sites of fiber deposition. *Am. Rev. Respir. Dis.* 129: 301-310.
109. Williams, M.G., Dodson, R.F., Corn, C., Hurst, G.A., (1982). A procedure for the isolation of amosite asbestos and ferruginous bodies from lung tissue and sputum. *J. Toxicol. Environ. Health.* 10: 627-38.
110. World Health Organization, (1986). Environmental health criteria 53: Asbestos and other natural minerals fibres, Geneva. World Health Organization.
111. Wotton, I.D.P., (1964). Enzymes in blood. *Microanalysis in medical biochemistry.* Churchill, London.
112. Yano, E., Tanaka, K., Funaki, M., Maeda, K., Matsunaga, C., Yamaoka, K., (1993). Effect of smoking on pleural thickening in asbestos workers. *British J. Ind. Med.* 50: 898-901.
113. Yano, S., Sone, E., (2000). Causative agents for lung carcinogenesis. *Nippon Rinsho.* 58: 1017-1022.