INTERNATIONAL CONFERENCE

on the

BIOLOGICAL EFFECTS OF ASBESTOS

Report on Proceedings

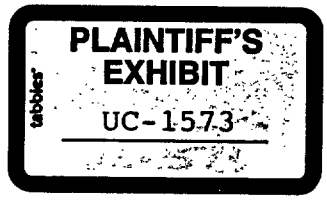
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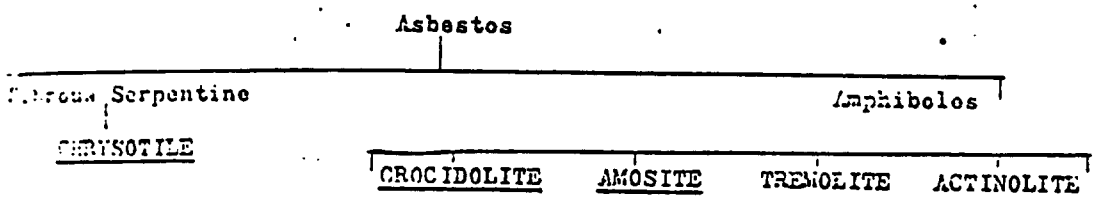
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I. Occurrence and the Major Use of Asbestos



The important members of these groups are:

1) CHRYSOTILE - White asbestos, a hydrated magnesium silicate with the formula ($3MgO\ 2SiO_2\ 2H_2O$). Sp.g. 2.55 with a long fibre of tubular structure.

2) CROCIDOLITE - Blue asbestos ($Na_2O\ 3FeO\ Fe_2O_3\ 8SiO_2\ H_2O$). Sp.g. 3.37 with a high resistance to acids and alkalies.

3) AMOSITE - Whitish-fawn, ferrous magnesium silicate ($1.5MgO\ 5.5FeO\ 8SiO_2\ H_2O$). Sp.g. 3.45

NOTE - the Amphiboles all have 8 silica units with 7 mineral units and 1 water molecule.

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The annual world production of asbestos minerals has risen from 500 tons in 1880 to over 3 million tons in 1933, mainly from Canada (Chrysotile and Crocidolite), South Africa (Amosite), and Russia (all three varieties); those three countries producing 85 per cent of the world production, and eleven countries, including Finland, providing the remaining 15 per cent of the world production.

There are many industrial uses for the manufactured products from asbestos minerals which follow from the chemical inertness and high thermal insulation properties of asbestos fibre.

Because it is important to ascertain whether a clinical case has been exposed during a lifetime to asbestos in any form, it is necessary to know the industrial applications in detail. The following list includes the major uses:-

1. Asbestos Mining.
2. Asbestos Textile Manufacture (woven and non-woven), mattresses, curtains, safety-suits, pipe covers, filter presses etc.
3. Asbestos Cement Products (sheets, partitions etc.)
4. Asphalt and Vinyl asbestos floor coverings.
5. Roof coverings (in spray form).
6. Clutch and Brake Linings in the Automobile Industry.
7. Fillers for paints, caulking compounds, and in plastic resins.

There are now over 3,000 industrial applications recorded.

The hazard from Asbestos Dust arises in Industry in many environments:

(a) Ship breaking and demolition yards.

(b) Ship yard maintenance (dockyards).

(c) Ship building yards.

(d) Sawing, drilling and grinding of asbestos products in brake-lining works.

(e) Sawing, grinding, levelling of asbestos sheets.

(f) Weaving mills, especially in screening and preparation rooms, picking, stacking, carding, spinning, winding and weaving.

(g) Spraying buildings to reduce fire risk.

(h) The bagging of asbestos products - Insulation materials.

(i) Demolition and renewal of pipe lagging in all industrial works.

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II. The Incidence of Asbestosis and the Association of Asbestos and Intra-Thoracic Tumour

At this conference, the need for further epidemiological studies on asbestos in all countries was stressed. There was wide divergence in the few epidemiological studies presented on the incidence of asbestosis, and this was even more apparent in the figures presented from Canada, South Africa, the States and the United Kingdom, on the association of asbestos with intra-thoracic neoplasms. In this respect, the Ministry of Labour, through the Factory Inspectorate[1] appear to be ahead, in that they have been collecting mortality data since 1924. With the size of sample now presented, the figures from England and Wales would appear to be the most reliable of all the countries represented at this conference. In summary these are:

TABLE I

Year	Asbestosis (uncomplicated)	Asbestosis + (Intra-thoracic tumour)
1924-1947	128	22
1924-1955	365	65
1924-1963	584	146

The proportion of asbestosis death certificates which also record a thoracic tumour, has risen in both sexes disproportionately to the number of uncomplicated asbestosis, so that currently over 50 per cent of males dying with asbestosis have also a neoplasm. Even when viewed against the steadily rising incidence of lung cancer in the population as a whole, there seems little doubt that the increase is a real one. Selikoff,[2] in the New York area, and using data from the Asbestos Workers Union (total population 632) for the period 1942-1962, has recorded 45 deaths, 42 of these due to carcinoma of the bronchus and 3 to pleural neoplasms (mesothelioma), giving a death rate 6.8 times higher in asbestos laggers than in the general U.S. white, male population.

In the studies among asbestos workers in this series, it is interesting to note that cancer of the stomach, colon and rectum was three times as frequent as expected, thus focussing attention on the relationship between industrial asbestos exposure and carcinoma of the gastro-intestinal tract.

Whilst x-raying the large New York series, Selikoff[3] noticed a high incidence of pleural calcification amongst asbestos insulation workers, and the suggestion made at this conference is that asbestosis is perhaps the most common cause of pleural calcification in industrial countries at present, especially if bilateral. Of 1,120 pipe laggers examined:

/TABLE II . . . .

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TABLE II

Radiological Examination of Pipe Laggers

Lapsed Time Since Onset of Exposure	Number Examined.	Grade Calcification
40	121	57.9
30 - 39	194	34.5
20 - 29	77	10.4
10 - 19	379	1.1

He concluded that lapsed time, since onset of exposure, is more important than total exposure. The common sites of pleural plaques are diaphragm, costal margins, anterior and posterior-mediastinal, pericardium, inter-lobar fissures and even lung apices. Many plaques were missed in the early days, (e.g. these are obscured by rib shadows). Two modifications of procedure have now brought many more calcifications to light:

(1) Oblique views are always taken (Right and Left)

(2) A higher penetration kilovoltage is used if asbestosis is suspected.

Pleural calcifications appear to be uncommon before 15 years interval or lapsed time following first exposure. An average figure is 20 years.

III, Environmental Asbestos Exposure

This conference high-lighted a maxim well recognised in Industrial Health but insufficiently stressed in medical teaching - the importance of taking a detailed occupational history. Many cases in papers describing pleural calcification and pleural and peritoneal neoplasms could find at first, no association with asbestos. Careful histories, however, revealed chance environmental exposure to asbestos many years previously. Haddow[4] in 1929, reported asbestos bodies in a man not employed in asbestos, but living near an asbestos factory. At this conference Thomson[5] reported the results of 500 autopsies in Cape Town and 500 in Miami, Florida. The results were similar in the two cities. 30 per cent of males and 20 per cent of females showed asbestos bodies. In 6 per cent of the males there was the possibility of occupational origin, but in 80 per cent of the positive cases the presence of asbestos was regarded as the result of contamination of the urban atmosphere. The origin of the asbestos fibres was thought to be brake and clutch lining disintegration, but this has now been disproved and the problem remains unsolved. What is now required is a base line so that future trends may be accurately followed. The extent of the problem may be

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/larger . . . .

larger than we appreciate at present. Among 3,312 individuals x-rayed in an area around an asbestos mine in Finland, Kivilnoto[6] found 499 cases of pleural calcification. In a control area, without an asbestos mine, no cases were found among 7,101 persons x-rayed. In this respect, concern may be felt for certain job categories. Pipe insulation workers undoubtedly share their exposure with other trades - electricians, plumbers, sheet-metal workers, boiler makers, fitters and even foremen. Perhaps [illegible] the supervising engineer or architect should be examined. In Belfast, where there are no asbestos factories, Elmes[7] found 42 cases of pleural mesothelioma in a population of 3/4 million in the period 1949-1963 (14 years). Between 1906 and 1923, 1,000 tons per year of boiler composition (85 per cent asbestos) was imported for shipbuilding purposes and it is thought that the latent period of 40 years has now produced the intra-thoracic tumours in Belfast. A positive history of exposure to asbestos at work was obtained in 32 of the 42 cases of pleural mesothelioma. Newhouse[8] found 83 cases of mesothelioma from hospital records in London, 10 between (1917 - 1950) and 73 between (1950 - 1963). The interval between first exposure and terminal illness ranged from 16 to 55 years with a mean of 37 years. A history of exposure to crocidolite was obtained in 53 per cent of cases and three main types of exposure were recognised:

(1) Asbestos textile works

(2) Laggers or pipe insulators

(3) Exposure to dust brought home by relatives (4).

Of the 36 patients with mesothelioma, with no positive occupational history and no relatives living at home who worked with asbestos, 11 lived within half a mile of an asbestos factory.

Of the 83 cases found in London, 62 had pleural mesothelioma, and in 21 the tumour was peritoneal in origin.

Enticknap and Smither[9] in 5½ years (1958 - 1964) saw 52 cases of asbestosis at necropsy. Nineteen (36 per cent) had lung tumours and fourteen (27 per cent) had abdominal tumours, nine of which appeared to originate in the peritoneum. The lapsed time following exposure varied from 20 to 46 years and exposure time varied from 10 months to 32 years. The interesting feature of the peritoneal tumours associated with industrial exposure to asbestos was that associated lung fibrosis and pulmonary changes are below average in severity (disability 10 - 30 per cent) and in some of those cases there was insufficient lung disability to qualify for asbestosis certification (4 cases). It was also suggested that the incidence of peritoneal tumours might well be very much higher but for:

(a) The shorter life span of the severely affected worker where dust pathology predominates.

(b) The difficult differential diagnosis from other forms of abdominal neoplasm, i.e. Carcinoma of stomach, [illegible] ovaries and many unclassified cases of peritoneal tumour.

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IV. Research Matters Arising from Asbestos Conference

(1) In future work, the exact type of asbestos dust must be identified. There is insufficient data correlating type of dust and subsequent pathology. Animal experiments must be conducted with standard dusts.

(2) The exact significance of particle size is not known. Originally it was thought that only coarse fibres were responsible for lung changes but the conference felt that ultra-microscopic particles will also produce fibrosis (less than 2 microns). The M.A.C. 5 million/m^3 is not now accepted. This conference made it clear that industry should aim at 1 million/m^3 maximum and accept this figure with reservations until our knowledge in this field is extended.

(3) The pathogenesis of asbestos is unknown. The following scheme - asbestos fibre → macrophages → lysis → sclerosis → fibrosis → carcinogenesis is thought to be an immunity reaction associated with gammaglobulin. The reaction is not now regarded as explosive in nature but is a slow, insidious, low grade, progressive fibrosis with early fibrotic changes around the bronchioles. There is thickening of bronchiole walls and of the pleura. In its early phase the lesion is not obstructive. Finally there is the formation of the asbestos body, a ferriton (iron-protein complex) surrounding the fibre and is laid down in concentric rings. Later the asbestos body becomes fragmented. Asbestos bodies are difficult to find, will require polarised light, may not be characteristic of asbestos only, and may contain metals other than iron. Small birefringent bodies seen in lung tissue have always been accepted as asbestos, but caution is now required in interpretation. Electron-microscope studies are now proceeding at Cambridge. The conclusions are (a) that all asbestos bodies so identified may not be due to asbestos, and (b) that there is difficulty finding asbestos particles less than 10 micron without the necessary techniques.

(4) Asbestos fibres may migrate from the lung and have been found in spleen and liver. This migration may explain the presence of gastro-intestinal malignancies, but again, fibres may be swallowed direct (ingestion of bronchial secretions).

(5) There does appear to be a direct correlation between asbestos dust exposure time and tumour formation. There is, however, strong evidence reported by Wagner in a large series of cases of mesothelioma that crocidolite is more dangerous than chrysotile. In North America however, chrysotile has been associated with peritoneal tumours. There have been no cases reported from South Africa of tumour formation where the exposure was to amosite only, but one tumour (peritoneal) has been reported by Selikoff with amosite.

(6) The natural asbestos minerals as mined, always contain mineral oil (up

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[illegible] must be excluded. Work in the chemical field [illegible] proceeding at the Chester Beatty Research Institute (Dr. Harrington). The rising incidence of bronchial carcin[illegible] pleural mesotheliomas, peritoneal tumours and possibly gastro-[illegible]inal malignancy might be explained by the fact that the asbestos worker is now in the so-called "tumour formation period", i.e. 20 - 40 years following the first exposure. If this is the pathology with asbestos fibre, then we shall require to look closely at fibre glass, a later development, now in production 10 - 12 years in Scotland. Personnel exposed to this fibre will not have arrived at the tumour formation period for a further 10 - 12 years in Scotland, but in Germany, where the fibre glass process originated, a production time in the region of 20 years is reported.

(8) There are large differences both in morbidity and mortality of asbestos in the surveys in different parts of the world. These discrepancies may be explained by the different chemical nature of the minerals, but there is at the same time, an urgent need to standardise radiological findings and histology.

(9) Therewere too many small surveys presented at this conference without the required statistical control. An overall epidemiological study is urgently required.

(10) In malignant tissue arising from asbestos, the characteristic lung histology is not easily interpreted. The loose, fibrous tissue is not particularly malignant-looking. The fibrous tissue is infiltrated with clefts lined with capillary and alveolar pseudo-epithelium, the whole picture being difficult to distinguish from adenocarcinoma.

(11) In the experience of the M.R.C. Pneumoconiosis Unit, there is evidence that chest disease in asbestos workers detected by means of x-rays is more serious than pneumoconiosis or silicosis with similar radiological findings.

(12) The average age at death when the asbestos hazard was first recognised (1907) was 33 years. In 1964, the average age at death is 57 years. It must, however, be recognised that heavily exposed people are still dying in 1964 in their 30's and 40's.

(13) There are two groups of asbestos workers in which the risk of asbestos and malignancy is high; (a) pipe laggers and demolition wor[illegible]rs, where the exposure will be constant over many years, and (b) asbestos sprayers where the atmospheric concentration of asbestos is very high.

(14) There is a vast improvement in dust control in <u>large</u> manufacturing works where the asbestos hazards are now recognised and controlled, and where preventive measures will eventually reduce the rising incidence of asbestosis. Small units, however, are still not dust free and are not subject to environmental control. In this respect, the effect of the [illegible]T, combined with long working hours at that

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V. Clinical Criteria - Asbestosis

Throughout the conference there were many references to signs and symptoms of asbestosis. These are:-

1. Vague chest pains - a very early symptom
2. Dyspnoea - may be first clinical symptom
3. Unproductive cough or bronchitis in an otherwise healthy subject, with no previous chest history.
4. Loss of energy and generally off-colour. Early symptom.
5. There is little or no tendency to asthma. Lung function tests are not of great value in the early stages, other than to establish a base line, e.g. pre-employment examinations.
6. Basal râles - early sign.
7. Finger clubbing - noted in about 20 per cent of cases.
8. Pleurisy may be first indication of asbestosis, with or without associated neoplasm.
9. Pneumonia may also be first indication, as in 8.
10. Peritoneal tumours may often come to light
    (a) Vague abdominal pain in any quadrant of abdomen
    (b) Abdominal discomfort. Indigestion is an early symptom and an employee complaining of vague discomfort is seen by the Factory M.O.
    (c) Ascites.

    Patient may report for a slimming diet, as happened in one case.

Radiological Appearances

1. Basal distribution and commonly bilateral is characteristic of asbestosis.
2. The low grade, interstitial fibrosis gives rise to coarse, linear markings, more common in the lower lobes on both sides, and spreading into the middle lobes, described as "honey combing".
3. Pleural thickening (lung, diaphragm and pericardium).
4. The presence of calcified plaques.
5. An uneven right border of the heart - "the shaggy heart" sign - pericardium thickening.

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VI. Names and Units of Colleagues Working in the Asbestos Research Field

1.	Dr. J. C. Gilson	M.R.C. Pneumoconiosis Research Unit, Glamorgan, Wales.
2.	Dr. J. C. Wagner	As above
3.	Dr. W. J. Smither (Member of the Asbestos Research Council)	The Cape Insulation and Asbestos Products Co. Ltd., London. (Medical Officer).
4.	Dr. S. Holmes (Dust sampling and counting techniques)	Turner Asbestos Co. Ltd. Rochdale.
5.	Dr. C. G. Addingley ('Royco' expert)	British Belting and Asbestos Co. Ltd., St. Peters Buildings, York Street, Leeds.
6.	Dr. Ross Hunt (Physiologist and dust expert)	As above
7.	Dr. J. G. M. Davis (Electron-microscope expert physicist)	University of Cambridge, Cambridge.
8.	Dr. J. C. McVittie	Ministry of Pensions and National Insurance, London.
9.	Dr. W. D. Buchanan	Ministry of Labour, London.
10.	Dr. J. S. Harrington	Chester Beatty Research Institute, Royal Cancer Hospital, London.
11.	Dr. H. E. Ayer	Public Health Service, Division of Occupational Health,

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