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ASBESTOS FIBRES IN THE AIR OF TOWNS

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Abstract—Many observers have reported that asbestos bodies are present in the lungs of town dwellers who have had no industrial exposure to asbestos, suggesting that asbestos may be a normal contaminant of the urban atmosphere. The air of several cities—London, Reading, Rochdale, Bochum, Düsseldorf, Prague, Pilsen, Johannesburg and Reykjavik—has been sampled using millipore filters. The samples were transferred to carbon film on a grid and they were examined by electron microscopy. Asbestos fibres were found in samples from every town. They were mostly present as single fibrils but some were in agglomerates that contained many fibres.

ASBESTOS fibres that have been inhaled often become coated in the human lung with a material that is largely ferroprotein, giving characteristic structures called asbestos bodies. Some other fibrous materials may also become coated to give pseudo asbestos bodies. CAUNA, TOTTEN and GROSS (1965) found asbestos bodies in 41 per cent of lung smears taken from 100 autopsies of adults who had had no known connection with the asbestos industry. THOMSON and GRAVES (1966) examined lung smears obtained at 500 autopsies in Cape Town and 500 in Miami, Florida. The results were similar in the two cities, specimens from 30 per cent of the males and 20 per cent of the females showing asbestos bodies. Some 15 per cent of the positive cases had very many asbestos bodies and it was considered that these had been exposed to an industrial risk. In 85 per cent of the positive cases there were few bodies and there were no associated pulmonary changes.

ANDELVEL and THURLBECK (1966) in Montreal found asbestos bodies in 48 of 100 random necropsies. They also found pseudo asbestos bodies but believed they could differentiate between bodies produced from asbestos and those from other sources. ROBERTS (1967) found asbestos bodies in lung smears from 23 of 100 consecutive hospital necropsies in Glasgow, Scotland, near an industrial area where shipbuilding is an important industry. GHEZZI, MOLTENI and PUCCETTI (1967) found asbestos bodies in 51 out of 100 random subjects who died in Milan. WEBSTER (1965) reported asbestos bodies in the lungs of 47 per cent of a group of patients who had had no direct connexion with the asbestos industry. TABERSHAW (1968) found asbestos bodies in 41 per cent of the lung smears he examined, and POLLIACK and SACKS (1968) in 26 per cent of 100 consecutive adult autopsies in Jerusalem.

PENMAN and THOMSON (1970) obtained similar results in Dunedin, a town in New Zealand without heavy industries. They showed that the number of asbestos bodies found in a lung was related to the method of examination; presumably most lungs contain asbestos bodies. An examination (UM, 1971) of histological slides kept in the Central Histological Laboratory of the Archway Hospital, London, suggested that the number of asbestos bodies in lungs is increasing; when 100 lung sections cut from blocks made in each of the years 1936, 1946, 1956 and 1966 were examined, asbestos bodies were found in 0, 3, 14 and 20 per cent respectively.

In France, BIGNON *et al.* (1970) found structures similar to asbestos bodies in tissue from every lung examined. In general, there were more such structures in the lungs of

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persons who had lived in Paris or near to a large factory manufacturing brake linings in which asbestos is used.

The asbestos bodies in the lungs of persons not normally handling asbestos are presumably derived from fibres present in the urban atmosphere but the widespread reports suggest that most atmospheres are contaminated. We obtained samples of dust from the air of several cities by drawing about 50 l. of air through a millipore filter (type VF) by means of an Austin diaphragm pump. The filters were used face downwards to prevent large particles from settling on the membrane. The specimens were transferred to grids by forming a carbon deposit about 200 Å thick over the dust, transferring small pieces (1·5 × 1·5 mm) cut from the filter onto the grids, carbon downwards, then dissolving the filter away with acetone. The dust was left on the carbon membrane on the grid. A careful search of these specimens was made with the electron microscope. Blank millipore filters treated in the same way were examined to ensure that the filters themselves were not contaminated.

The sampling points chosen were well above street level and, so far as could be ascertained, away from any direct source of contamination. In London samples were taken from the top of a five-storey building, in Reading from the top of a two-storey building situated in a park and also inside the building. Sampling positions well above street level were also chosen in Rochdale, England, in Bochum and Düsseldorf in Germany, in Prague and Pilsen in Czechoslovakia, in Johannesburg, South Africa, and in Reykjavik, Iceland. Only Rochdale, which has the largest asbestos textile factory in Europe, can be regarded as having an important asbestos industry.

A careful search of some of the grids revealed no true fibres, but on other grids there were fibres or agglomerates of fibres. It was seldom that more than one or two windows of a grid revealed fibres. Usually the fibres were single, but occasionally large agglomerates were observed. Some appeared to be coated with soot or other material. Fibres were present in the specimens from every sampling point but no fibre was found on any blank filter.

Chrysotile is the most widely used variety of asbestos. The chrysotile crystal has a characteristic appearance in the electron microscope. The lattice is in the form of a tube that may be hollow or filled with amorphous material and the ultimate fibril has a diameter of about 0·03–0·06 μm. Electron diffraction studies made on some fibres having this appearance confirmed that they were in fact chrysotile asbestos. Other fibres were identified as amphibole asbestos. Amphibole asbestos was identified in the air of Bochum and Johannesburg.

Compared with other contaminants the amount of asbestos is very small. A part of a grid as seen in the electron microscope is shown in Fig. 1. Many similar fields contain no fibres. There appears to be no way in which the concentration of fibres can be expressed satisfactorily. Rickards and Badami (1971) found chrysotile asbestos in the air of Rochdale and attempted to estimate the mass concentration by an X-ray technique based on the measurement of the integrated area under the (002) peak. They were able to determine only the upper limit which was 100 μg of chrysotile m^{-3} of air. Gadsden, Parker and Smith (1970) described an infra-red technique for the measurement of the mass concentration of asbestos dust in air but they found the concentration too low for the application of their method even in a car park with a foundation of asbestos waste and a tip used for dumping asbestos waste. They did however, detect fibres on their millipore filters by electron microscopy.

Fig. 2. A single fibre
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FIG. 1. Particulate matter filtered from London air.

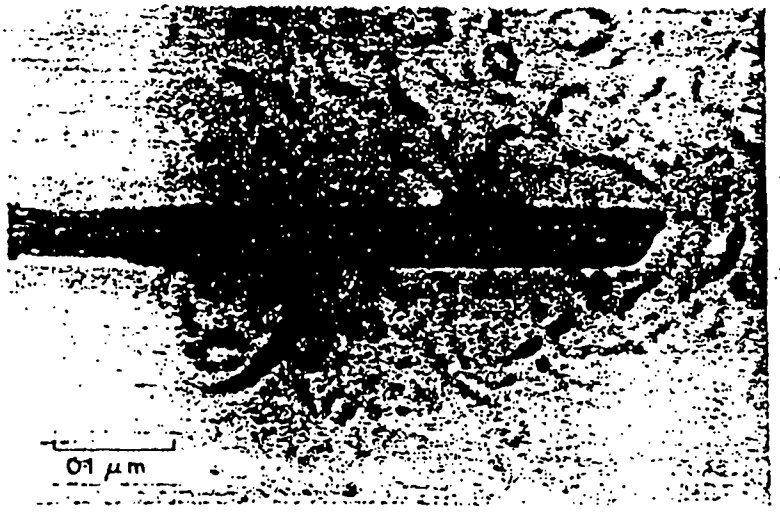

FIG. 2. A single fibre, probably an ultimate fibril of chrysotile, filtered from the air of Düsseldorf. Most fibres appear to be contaminated with soot and tar so that definition is poor, but the central core and fine structure is visible.

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FIG. 3. Single fibrils filtered from London air. The fibril dia. is about 0·04 μm, typical of chrysotile. The fine structure shows changes similar to those observed when chrysotile is heated.

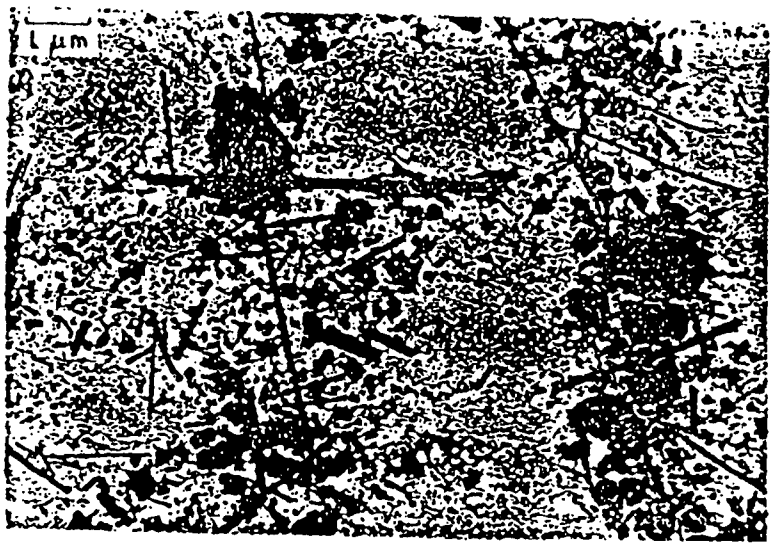

FIG. 4. An agglomerate of fibres collected on a filter at the top of a building in London.

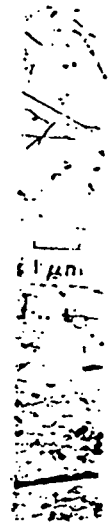

FIG. 5. An

FIG. 5. An agglomerate of fibres collected on a filter at the top of a building in London.

Most of the asbestos found in our millipore filter samples was present as single fibres or as ultimate fibrils (FIGS. 2 and 3). These could be estimated by counting except that some were obliterated by other contaminants. There were, however, some complex agglomerates (FIGS. 4 and 5) and it is impossible to decide in any meaningful way how many particles such masses represent. Such a mass tends to break up when the filter is extracted with acetone and there are far more fibres near an agglomerate than in the rest of the sample. Agglomerates might also break up when meeting surface active substances in the lung.

These observations are offered as an indication that asbestos is a contaminant in the air of cities and as an explanation of the asbestos bodies that have been found in the lungs of persons not industrially exposed to asbestos. The concentration of fibres is small and there is no indication whether the fibres are pathologically significant.

Asbestos fibres are removed from the lung by a mechanism (BOTHAM and HOLT, 1968) that involves the production and eventual fragmentation of asbestos bodies. Evidence from animal experiments suggests that once a fibre is coated it is no longer pathogenic but that the uncoated fibres produce the pathological effects. Since the rate of coating depends on the nature of the fibre, chrysotile (and glass) fibres being coated more quickly than crocidolite and amosite, fibres of the amphiboles are potentially more dangerous. Asbestos fibres in the lung are coated in the cytoplasm of macrophages and giant cells, but if several fibres are retained by the same cell only one is usually coated. Moreover, the number of macrophages in an area of lung is limited. Thus, while low concentrations of isolated fibres are likely to have minimal pathological significance, a high local concentration produced by the break up of an agglomerate of fibres, might represent a potential risk.

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REFERENCES

ANULVEL L. and THURLBECK W. M. (1966) The incidence of asbestos bodies in the lungs at random necropsies in Montreal. *Can. med. Ass. J.* 95, 1179–1182.

BIGNON J., GONI J., BONNAUD G., JAURAND M. C., DUPOUR G. and PINCHON M. C. (1970) Incidence of pulmonary ferruginous bodies in France. *Envir. Res.* 3, 430–442.

BOTHAM S. K. and HOLT P. F. (1968) The mechanism of formation of asbestos bodies. *J. Path. Bact.* 96, 443–453.

CAUNA D., TOTTEN R. S. and GROSS P. (1965) Asbestos bodies in human lungs at autopsy. *J. Am. med. Ass.* 192, 371–373.

GADSDEN J. A., PARKER J. and SMITH W. L. (1970) Determination of chrysotile in airborne asbestos by an infra-red spectrometric technique. *Atmospheric Environment* 4, 667–670.

GHEZZI I., MOLTENI G. and PUCCETTI U. (1967) Asbestos bodies in the lungs of inhabitants of Milan. *Medna Lav.* 58, 223–227.

PEIRMAN H. G. and THOMSON K. J. (1970) Pulmonary asbestos in Dunedin, New Zealand, assessed by two methods. *Pathology* 2, 175–182.

POLLIACK A. and SACKS M. I. (1968) Prevalence of asbestos bodies in basal lung smears. *Israel J Med. Sci.* 4, 223–226.

RICKARDS A. L. and BADAMI D. V. (1971) Chrysotile asbestos in urban air. *Nature* 234, 93–94.

ROBERTS G. H. (1967) Asbestos bodies in lungs at necropsy. *J. clin. Path.* 20, 570–573.

TABERSHAW I. R. (1968) Asbestos as an environmental hazard. *J. occup. Med.* 10, 32–37.

THOMSON J. G. and GRAVES W. M., Jr. (1966) Asbestos as an urban air contaminant. *Arch. Pathlogy*, 81, 458–464.

UM C.-H. (1971) Study of the secular trend in asbestos bodies in lungs in London 1936–66. *Br. med. J.* 2, 248–252.

AUTHOR'S REPLY

PERHAPS the large intradiurnal variation in turbidity I reported (RANDERSON, 1973) has not been normally observed because, in the United States, turbidity observations are frequently collected only two or three times daily (FLOWERS *et al.*, 1969), at local noon and 3 h before and after local noon, weather permitting.

I believe that Dr. Bullrich misinterpreted my second possible explanation for the strong intradiurnal variation in turbidity. The selenium photocell in the sunphotometer is sensitive from 0·3 to 0·68 μ and the wavelength of maximum intensity of solar radiation shifts toward 0·7 μ as the air mass value increases (MOON, 1930). Thereby, the idea was that proportionately more solar radiation might be sensed by the sunphotometer near 0·7 μ rather than at 0·5 μ for $m = 5$. TABLE 1, containing data provided by FLOWERS (1972), supports this contention; however, I would like to point out that Flowers has recently shown that this contribution is quite small and doesn't appear to explain the observed intradiurnal variation in turbidity.

TABLE 1. SOLAR FLUX VS AIR MASS AND WAVELENGTH (ly min^{-1})

	$m = 1$	$m = 5$
I_1(0·44–0·56 μ)	$4{\cdot}04 \times 10^{-3}$	$9{\cdot}15 \times 10^{-3}$
I_2(0·70–0·73 μ)	$7{\cdot}0 \times 10^{-4}$	$3{\cdot}5 \times 10^{-4}$
I_2/I_1	$1{\cdot}7 \times 10^{-4}$	$3{\cdot}8 \times 10^{-4}$

Dr. Bullrich has offered forward scatter as another possible explanation for this intradiurnal variation. To my knowledge, there are no quantitative data available to permit an evaluation of the contribution of forward scatter to local turbidity. A noteworthy study by HULBURT (1941) showed that forward scatter can be greatly increased by the presence of dust and haze particles in the atmosphere. I certainly agree that forward scatter might explain a significant portion of the intradiurnal variation in turbidity I observed, using two Volz sunphotometers. If further study does explain forward scatter as the primary culprit, then it should be accounted for in the observations.

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REFERENCES

FLOWERS E. C. (1972) Personal communication.
FLOWERS E. C., MCCORMICK R. A. and KURFIS K. R. (1969) Atmospheric turbidity over the United States, 1961–1966. *J. Appl. Meteor.* 8, 955–962.
HULBURT E. O. (1941) Optics of atmospheric haze. *J. Opt. Soc. Am.* 31, 467–476.
MOON P. (1940) Proposed standard solar radiation curves for engineering use. *J. Franklin Inst.* 230, 583–617.
RANDERSON D. (1973) A summary of hourly turbidity measurements for Las Vegas, Nevada, during fall months. *Atmospheric Environment* 7, 271–279.

DISCUSSION

ASBESTOS FIBRES IN THE AIR OF TOWNS*

HOLT AND YOUNG are unduly reserved about the potential for quantitating the asbestos content of city air. Other studies have shown that such measurement can provide very useful data concerning asbestos air pollution, even serving as a basis for the implementation of abatement procedures.

* P. F. HOLT and D. K. YOUNG (1973). *Atmospheric Environment* 7, 481–483.

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1. Asbestos bodies are only the tip of the iceberg, representing only the largest fibres (including materials other than asbestos), visible by optical microscopy, but missing fibres below the limit of resolution of the method. Rather, asbestos fibres and fibrils can be directly sought by EM in lungs of city dwellers--and, when they are, found far more frequently than coated bodies (Langer *et al.*, 1971; Pooley *et al.*, 1970). Moreover there need not be the insecurity concerning the nature of the asbestos body core (Langer *et al.*, 1971). As Holt and Young note, by EM chrysotile asbestos has a unique appearance. We know what we are seeing.

2. The authors seem resigned to being unable to satisfactorily express the concentration of asbestos fibres in air. Admittedly, this cannot be done with fine precision, but rather reliable and reproducible estimates can be made. (Duplicate analysis of samples in our laboratory indicate a factor of two or three is possible.) The techniques they quote (X-ray diffraction and infrared spectrometry) are too insensitive for the purposes. On the other hand, mass concentrations can be calculated from number and size of fibrils, and the density of chrysotile (Nicholson *et al.*, 1971). In the United States for example, our early results showed the following concentrations to be found in the air of New York (Selikoff *et al.*, 1972; Selikoff *et al.*, 1968).

Table 1. Chrysotile content of ambient air in N.Y.C. preliminary results

Sample locations	Asbestos air level in 10^{-9} g m^{-3}
Manhattan	25–60
Bronx	25–28
Brooklyn	19–22
Queens	18–29
Staten Island	11–21

Additionally, specific sources of asbestos air pollution were defined from sampling near construction sites.

Table 2. Chrysotile content of N.Y.C. air in vicinity of spray fireproofing with asbestos-containing materials

Site	Asbestos level in 10^{-9} g m^{-3}
1 (downwind from source)	45–180
2 (45° from source)	15–30
3 (upwind from source)	20
4 (downwind from source)	45
5 (upwind from source)	20

Since these data were obtained we have quantitated asbestos levels in over 200 samples from 49 U.S. cities and have found asbestos to be present in all.

3. It is difficult to comment upon the relevance of comparative techniques, in the absence of detailed description. Some reservations might be lifted with alterations in methods. For example, were grids scanned visually or were photographs made and fibrils counted (the latter is much more sensitive. We find, for example, that visual scanning can underestimate concentrations by as much as 50 per cent). What magnifications were used? (Higher magnifications—e.g. scan 20 000–30 000 ×—are much more revealing.)

It is evident that a good deal is now known of asbestos air pollution and consequent, lung contamination by the ubiquitous fibrils. What has not yet been deciphered, as Holt and Young correctly state, is the disease potential of such general community asbestos air pollution. Cancer may occur with occupational asbestos exposure, with that associated with neighborhood pollution

about asbestos works or in households of asbestos workers, but these are presumably heavier exposures than we now observe in the general environment, and the effect of general asbestos air pollution remains to be defined (SELIKOFF *et al.*, 1972; SELIKOFF *et al.*, 1968).

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REFERENCES

LANGER A. M., SELIKOFF I. J. and SASTRE A. (1971) Chrysotile asbestos in the lungs of persons in New York City. *Arch. Environ. Hlth* 22, 348–361.

LANGER A. M., RUBIN I. B. and SELIKOFF I. J. (1972) Chemical characterization of asbestos body cores by electron microprobe analysis. *J. Histochem. Cytochem.* 20, 723–734.

NICHOLSON W. J., ROLE A. N. and FERRAND E. F. (1971) Asbestos air pollution in New York City. In *Proc. Second International Clean Air Congress* (Edited by ENGLUND H. M. and BERRY W. T.) pp. 136–139. Academic Press, New York.

POOLEY F. D., OLDHAM P. D., UM C. and WAGNER J. C. (1970) The detection of asbestos in tissues. In *Pneumoconiosis: Proc. International Conf.* Johannesburg, 1969 (Edited by SHAPIRO H. A.) pp. 108–116. Oxford University Press, Oxford.

SELIKOFF I. J., NICHOLSON W. J. and LANGER A. M. (1972) Asbestos air pollution. *Arch. Environ. Hlth* 25, 1–13.

SELIKOFF I. J. and HAMMOND E. C. (1968) Community effects of non-occupational environmental asbestos exposure. *Am. J. Pub. Hlth* 58, 1658–1666.

AUTHORS' REPLY

QUANTITATIVE results for the concentration of asbestos in New York City are valuable and a reproducibility indicated by a difference of only two or three times is impressive. Values for the 49 other cities will be of great interest.

The difference shown by these figures between concentrations of asbestos fibres near to a point at which asbestos was being sprayed and the concentrations found in the general atmosphere was little more than the difference between duplicate samples estimated in the laboratory, except when the sampling was carried out immediately downwind from source. This proves that there is widespread and rapid dissipation of the dust from the point of origin.

The neighbourhood hazard has again been emphasized in a report given by W. Bouché at the meeting of the *International Societies for Hygiene, Preventive and Social Medicine*, 1972. The report showed that, of the 196 patients with mesothelioma reported in the city of Hamburg, 55 had no known industrial exposure to asbestos but had lived in the neighbourhood of asbestos factories. This indicates the importance of independent surveys of the type carried out by Selikoff and his collaborators.

The object of our investigation was only to determine whether asbestos fibres are present in the atmosphere of towns where there is no asbestos industry. The result was positive in every case.

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DISCUSSION

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THE AUTHORS stress the frequency with which asbestos fibres are found in human lungs. They present

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evidence that it is more common in cities than in rural environments. They do not review the evidence which has been published which suggests that certain forms of asbestos are relatively harmless. They do not inform the reader what they consider to be the source of the asbestos in the urban atmosphere; is it from brake linings or construction materials, for example? I would be grateful if the authors pointed up the nature of the risk to which they allude and stress, if possible, ways of distinguishing between more harmful and less harmful asbestos fibres.

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AUTHORS' REPLY

THE SOURCE of the asbestos fibres in urban air is still a matter of speculation. Some 3 million tons of the material are mined annually and asbestos has very many industrial applications so some general contamination of the air seems certain. Chrysotile is used in brake linings and brake linings are being constantly disintegrated. However, the heat generated when brakes are applied would alter the appearance of the chrysotile fibre in the electron microscope. Some fibrils in our samples appear to have been heated but most of the chrysotile fibrils we observed did not have this appearance and are more likely to have been derived from building material. Photographs by LUMLEY, HARRIES and O'KELLY (1971) illustrate the production of dust from building materials.

The individual fibrils seen in our samples probably have no pathological significance but the appearance of large agglomerates in random samples is less reassuring. They suggest the possible existence of localized sources where the contamination may be appreciable.

Perhaps the most disconcerting aspect of reports on the incidence of asbestosis and related diseases, and particularly mesothelioma, is the frequency with which a "neighbourhood hazard" or "endemic asbestosis" is mentioned, disease occurring in apparently innocent occupations or in members of an asbestos worker's family. Examples that may be quoted are workers near laggers in Glasgow (GOLD and CUTHBERT, 1966), villagers near an asbestos mine in Italy (VIGLIANI, 1969), Finland (KIVILUOTO, 1960) and South Africa (WAGNER, 1959; SELIKOFF, 1963), in carpenters (FLETCHER, 1971) and persons not occupationally exposed to asbestos in London (NEWHOUSE and THOMPSON, 1965) and Hamburg (DALQUEN, DABBERT and HINZ, 1970).

The effects of asbestos may be due, in part, to the morphology of the fibres but, because it is more readily removed from the respiratory part of the lung, chrysotile the commonest form of asbestos is probably the least harmful (BOTHAM and HOLT, 1971a). Submicron glass fibre is also removed at a similar rate to chrysotile (BOTHAM and HOLT, 1971b). Chrysotile can be readily distinguished from other varieties of asbestos with the electron microscope.

Gross fibrosis is probably produced only by very high concentrations of asbestos but the amount needed to produce a carcinoma is not known. STANTON and WRENCH (1972) produced mesothelioma in rats with amosite, crocidolite and chrysotile.

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REFERENCES

BOTHAM S. K. and HOLT P. F. (1971a) The development of glass fibre bodies in the lungs of guinea-pigs. *J. Path.* 103, 149–156.
BOTHAM S. K. and HOLT P. F. (1971b) Development of asbestos bodies on amosite, chrysotile and crocidolite fibres in guinea-pig lungs. *J. Path.* 105, 159–167.
DALQUEN P., DABBERT A. F. and HINZ I. (1970) The epidemiology of pleural mesothelioma. A preliminary report on 119 cases from the Hamburg area. *Germ. Med. Mon.* 15 (2) 89–95.
FLETCHER D. E. (1971) Asbestos-related chest disease in joiners. *Proc. R. Soc. Med.* 64, (8) 837–8.
GOLD C. and CUTHBERT J. (1966) Asbestos—A hazard to the community. *Pub. Health* 80 (6) 261–70.
[illegible] R. (1960) Pleural calcification as a [illegible] sign of non-occupational endemic [illegible]
LUMLEY K. P. S., HARRIES P. G. and O'KELLY F. J. (1971) Buildings insulated with sprayed asbestos: a potential hazard. *Ann. Occup. Hyg.* 14, 255–257.
NEWHOUSE M. L. and THOMPSON H. (1965) Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. *Br. J. Indust. Med.* 22 (4) 261–9.
[illegible] HAMMOND E. C. and ORING J. (1968) Asbestos exposure, smoking and neoplasia. *Am. Med. Ass.* 204 (2) 106–12.