Section of Epidemiology & Preventive Medicine

President J N Morris FRCP

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Michael Williams Lecture

Asbestos Cancers: Past and Future Hazards [*Abridged*]

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Michael Williams was for a while one of my colleagues at the Pneumoconiosis Unit. It is just ten years since he was so tragically killed in a motor accident at the age of 44. He would have wished that a lecture in his memory should include a 'discussion', as he rarely attended any lecture without contributing to the discussion in his characteristically charming and enthusiastic manner.

I remember the joy with which he described to me the facilities ICI had provided for him, and being surprised at their extent as he had only recently joined the Company. I did not realize then that the rapid support he got was due to his quite exceptionally wide understanding of the engineering, chemical, and medical aspects of his work; also, his flair for spotting new risks. No one intent on delaying his proposals for prevention had much chance of outwitting him. He is rightly commemorated; he showed how much can be achieved by an industrial medical officer who is able to learn all about the processes in his factory and reach a stage where he can talk on equal terms with all from the research department to the factory floor. Previous lecturers (Dodds 1964, Bayland 1967; Case 1969, Forrest 1971) have described his character and contributions to knowledge in admirable detail.

When he had established himself as a leading expert on chemical carcinogens he received much help from the International Union Against Cancer (UICC). He visited many countries and by 1961 six out of the nine countries where β-naphthylamine had been made had stopped manufacture or were about to do so.

The part the UICC took in assisting the prevention of chemical cancers through sponsoring Michael Williams's visits does not seem to be well recorded. The same may be true of their assistance in stimulating worldwide interest in the asbestos cancers. I welcome this opportunity to record briefly the help my colleagues and I have received, first from the UICC and later from the International Agency for Research on Cancer (IARC).

In 1963 Dr T F Mancuso, who has had a long interest in occupational cancers, wrote to Dr J C Wagner suggesting that an international effort be made to investigate further the cancers caused by asbestos. With the help of Dr W Gardner, now president of the UICC, and the Anna Fuller Fund, a UICC Working Party on Asbestos Cancers (chairman – Dr H Stewart) was held in New York in 1964. A number of the recommendations at that time (UICC 1965) have now been achieved – for example, the preparation and distribution of standard reference samples of asbestos; the development and testing of a classification of the radiographic appearances of asbestosis; and the setting up nationally and internationally of panels of pathologists expert in pulmonary tumours. Since 1968 support for international investigations in asbestos has been provided by the IARC as part of their general programme on environmental carcinogens. We have received great help from Dr J Higginson (the director) and from Dr P Bogovski. A grant from the IARC through the MRC has led to investigations and research projects on the asbestos problem in eight countries.

The industry has made important contributions through the Asbestosis Research Council in this country and the Quebec Asbestos Mining Association in Canada. The Royal Navy has taken a leading part in reviewing the use of asbestos and

... preceding ... [illegible] ... study of dockyard workers.

Historical

Asbestos has a long history; it has been used for about four thousand years, but up to about a hundred years ago it was regarded more as a treasure than a commercial raw material. In 1725 Benjamin Franklin – inventor of the lightning conductor, bifocal spectacles, and the harmonica – gave the oldest known woven asbestos article – a small purse made of tremolite – to Sir Hans Sloane. Sir Hans, a physician and great collector, sold his treasures to the nation. The purse is still on show with the rest of his collection of minerals in the Natural History Museum.

About a hundred years ago the asbestos industry proper started, following the discovery of large deposits of chrysotile in Canada and almost simultaneously in Russia. Other types – the so-called amphiboles – were discovered principally in South Africa and Finland. It was about fifty years after the start of the industry that lung cancer was first thought to be caused by the dust (Fig 1), about another ten years before this was generally thought probable, and a further ten before it was finally established in the asbestos textile industry (Doll 1955). The first reports of diffuse pleural tumours linked with asbestos appeared in the early 1940s, but there was an interval of about fifteen years before this association was generally thought probable, and another ten before it was widely recognized in all countries. It now looks as though history may be repeating itself. Clinical reports and surveys suggest that other cancers may also be linked with asbestos – gastrointestinal, ovarian, and possibly leukaemias and lymphomas. The excess risk is, however, not yet certain enough to be rated as more than 'probable' and appears lower than for the lung cancers.

Processes within the Industry

There are three broad divisions of the industry where exposure to dust can occur: mining and milling, when the mineral fibres are extracted; manufacture and application, when the fibres are incorporated into a wide range of products; and demolition when the material is removed and scrapped.

Mining and quarrying are usually wet and dust levels are therefore not high. At this stage the fibre is still closely held in the parent rock, so that the proportion of respirable fibre in the dust is small. Dust levels rise during crushing and drying, and in the next processes where the fibre is separated from the rock. The proportion of fibre increases but much of it is still in fairly big bundles. The degree to which the fibre bundles are opened into its constituent fibres before bagging has increased over the years. But the dust control within the mills, which was virtually absent in the 1930s and early 1940s, has gradually and greatly improved by a factor of 100 or more. The bagged fibre with its low humidity is sent to the users in industrialized countries. Significant dust exposures can occur during transport due to the use of jute bags (now replaced by impermeable plastic ones) and inadequate dust control when the bags are opened.

In the manufacturing part of the industry major improvements in dustiness were first made in the textile section, in the 1930s (following the discovery of a major asbestosis hazard), and have

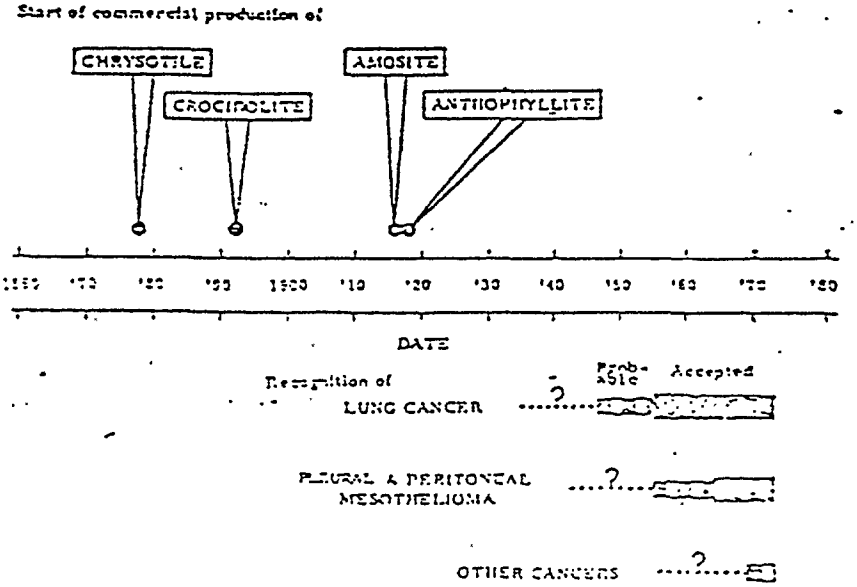

Fig 1 *Chronology of asbestos linked cancers*

Table 1
Factors influencing the incidence of asbestos associated cancers

Dose of asbestos	Total Pattern of exposure
Type of fibre	Chrysotile (C) Amosite (A) Crocidolite (Cr) Anthophyllite (An)
State of fibre	Dispersion Shape—straight or curly Size—diameter, length
Time	Duration of exposure From first exposure
Age	At first exposure
Sex Occupation	
Cofactors	Smoking Trace elements, &c.

continued to be made since. Reduction in dustiness in other parts of the industry, especially in making insulation materials and asbestos cements, came later. Adequate control of the dust in asbestos spraying sections is very recent and is still so difficult that the process has been stopped in some places. There are still very big differences in dust control in different countries.

The last stage of the process, to which until very recently little attention was given, is demolition and disposal of waste. Asbestos is almost indestructible and, therefore, it eventually has to be removed from buildings and ships, and elsewhere. It is at this time often very dry and the proportion of respirable fibre in the dust high, especially during the removal of old insulation. These are, of course, the processes which are most difficult to supervise and control. Levels of dustiness several hundreds times the present standard can easily occur during the stripping of pipe insulation for inspection.

The United Kingdom was the first country to exploit the usefulness of crocidolite and also the first to give it up. There is, however, still much old insulation containing crocidolite in ships and buildings, and elsewhere, and its safe removal is a major task. The Asbestos Research Council has given an important lead by preparing Codes of Practice for all uses of asbestos (Asbestos Information Committee, 10 Wardour Street, London W1V 3HG).

Factors Influencing Incidence of Asbestos Cancers

The more important and specific factors affecting the incidence of asbestos cancers are becoming clear (Table 1). The total dust dose as well as the pattern appears important. A knowledge of the exposure response relationship is needed to agree acceptable standards of dustiness in industry and predict the likely risk, if any, to the general public. The four important common types of fibre—chrysotile, amosite, crocidolite, and anthophyllite—differ in their biological effects. The degree of dispersion, diameter, length, and shape of the fibre markedly influence where the dust is deposited in the lung and its subsequent elimination, and probably its biological effect. The lapse of time between first exposure and the detection of associated cancers is long—rarely less than twenty years for mesotheliomas. This long interval means that the effects now being detected epidemiologically refer to conditions many years ago, and it is difficult to be sure that improvements in working conditions introduced even twenty-five years ago are fully effective. Cofactors, especially cigarette smoking, are certainly important; others, such as heavy metals also in the dust, may be relevant.

Lung Carcinomas

Comparisons of results of recent surveys of asbestos workers: A comparison of the findings in surveys which are technically fairly comparable (Table 2) provides some information about the effect of

Table 2
Prospective surveys of asbestos workers with follow up of 20 or more years from first exposure

		Exposure		Standard mortality ratio (all causes)	Lung cancer deaths (observed: expected)	
Process	Type of fibre	First	Duration (years)			Reference
Mining and milling	C	Before 1940	1/12+	92	1.2:1	McDonald *et al.* 1971
	C	Before 1940	1/12+	120	3:1	Maximally exposed group
	An	1936	10+		3:1	Kiviluoto & Meurman 1970
Manufacturing:						
Insulation	A	Before 1945	1+	155	9:1	Selikoff *et al.* 1973
Textile	C, Cr	Before 1933	20+	232	14:1	Doll 1955
Textile	C, Cr	After 1933	20+	(152)	1.4:1	Knox *et al.* 1968
Textile	C	Before 1938	2+	283	16:1	Mancuso & El-Attar 1967
Textile	C	After 1938	2+	185	3.1:1	Mancuso & El-Attar 1967
Insulation, textile	C, A, Cr	1933—45 (males)	2+	130	4:1	Newhouse 1969
Insulation, textile	C, A, Cr	1938—42 (females)	2+	200	17:1	Newhouse *et al.* 1972
Asbestos:						
Insulation	C, A	Before 1942	20+	139	8:1	Selikoff *et al.* 1979
Insulation	C, A, Cr	Before 1946	20+	140	8:1	Talcott-Braw *et al.* 1979
Insulation	C, A, Cr	Before 1940		262	17:1	Enticr & Simpson 1976

these factors. The populations studied were either from employers' lists or trade union records. Groups in many parts of the industry have now been studied. The exposures have usually been to a mixture of fibres, but some single fibre type exposures have also been reported (Mancuso & El-Attar 1967, McDonald *et al.* 1971, Kiviluoto & Meurman 1970, Selikoff *et al.* 1973). The excess of lung cancer has varied considerably between surveys. The most striking contrast is between the low risk in the survey of 12 000 chrysotile miners and millers in Quebec born between 1891 and 1920, and the high risk in the insulation workers where exposures have been to a mixture of chrysolite and amosite in USA, or to these two and crocidolite in UK. The big difference might at first sight be attributed to the use of crocidolite, but this was rarely or never used in the USA when the groups now being studied for mortality were first exposed more than twenty years ago. A group of insulation workers exposed only to amosite has also been studied (Selikoff *et al.* 1973) and is found to have an excess lung cancer risk comparable to other insulation workers exposed to chrysotile and amosite. Comparison with the low risk in the chrysotile miners and millers in Quebec might suggest that the risk was principally linked with amosite, but this is unlikely because in the early years the insulation workers exposed to chrysotile and amosite in America used mostly chrysotile, and the amosite was not widely used until later. In addition, the lung cancer risk is probably low in the amosite miners and millers in South Africa. There is also evidence of a high risk in the past to textile workers exposed only to chrysotile (Mancuso & El-Attar 1967). Thus, no single type of fibre seems to be especially important in relation to the lung carcinoma risk. It seems likely that the dose of respirable fibre of a particular size, diameter, and perhaps shape are the important factors. Further comparisons between textile workers and miners and millers exposed only to chrysotile may be helpful, especially if use can be made of records of past dust concentrations and the physical characteristics of the dust.

Recent surveys (McDonald *et al.* 1971, Newhouse 1969, Newhouse *et al.* 1972) have produced good evidence that the lung carcinoma risk is dose related (Table 3). Despite the low overall risk in the chrysotile miners and millers, the excess risk was limited to those with the highest past dust exposures. In the insulation workers in UK, with a mixed fibre exposure, there is also evidence of a difference of risk related to probable intensity of past dust exposure. The levels of dust exposure, even in those least exposed in both factory and mine, were probably two or even more orders higher than levels of exposure in the general public from environmental pollution. Thus these surveys, taken with other evidence, suggest that the dose of asbestos dust required to increase the lung cancer risk materially is likely to be well above that which has been encountered up to the present by the general public. It must, however, be recognized that the present high lung cancer risk due to cigarette smoking makes it difficult to detect small excess risks due to occupational or environmental causes.

Relation to length of exposure and to sex: Dr Newhouse's study is the only one where a comparison between a large group of men and women with similar exposures has been possible (Newhouse *et al.* 1972). Table 4 shows that, although the observed/expected ratio of lung cancer is higher in the women, the excess rate per 100 000 subject years of exposure is, if anything, greater in the men. In both sexes it is related to length of exposure. This higher rate in the men may in part be due to the fact that a year of exposure in the men actually entailed a greater dose of dust, or that the interaction between cigarette smoking and asbestos differs in the sexes.

Cofactors: Asbestos is a highly absorptive material. Lung cancers have naturally been blamed on substances absorbed on to the fibre, such as trace elements, cigarette smoke, and hydrocarbons from other sources. Cigarette smoke is certainly important (Selikoff *et al.* 1970), the risk being much greater in smokers than in non-smokers among asbestos workers. Doll (1971) pointed out that the effects of asbestos and cigarette smoke are perhaps multiplicative rather than additive, and this is supported by more recent

Table 3

Dose of dust and excess risk of lung cancer

Process	Fibre	Sex	Ratio observed/expected deaths after Low/medium exposure	Severe exposure
Mining and milling	C, A,	M	1.5:1	6.1:1
Manufacturing and textiles	Cr	F	6.7:1	13.4:1

			Equivalent average death rates related to dust exposure index			
			< 10	200	400	800
Manufacture of C	C	M	8	10.6	15.8	27.7

Table 4

Lung cancer risk in men and women after severe exposure, followed up 20 or more years (manufacturing asbestos insulation and textiles; C, A, Cr)

	Length exposure (years)	Observed/expected ratio	Excess rate per 100 000 subject years
Men	< 2	3.0	120
	> 2	5.6	570
Women	< 2	6.8	70
	> 2	32.6	380

evidence (Berry *et al.* 1972). It will be interesting to see how the pattern of lung cancer mortality in those who stop smoking but retain their burden of asbestos compares with those who stop smoking and have not been asbestos workers.

Mesothelial Tumours

Both pleural and peritoneal tumours are linked with exposure to asbestos, but as these tumours were until recently very rare, and were not on the ICD list until 1968, the past incidence is not available from national records, and special surveys are needed. The panels of pathologists set up following the UICC recommendations mentioned above, have been reviewing cases and developing evidence on the inter- and intra-observer variation. The variation is similar to that reported for other clinical assessments, and disagreements on diagnosis are to be expected. In addition, separation between parenchymal and pleural tumours is not perfect and may be especially difficult in those exposed to asbestos because their tumours may be sited towards the periphery of the lung.

Link with asbestos exposure: Evidence linking mesotheliomas of the pleura and peritoneum with past exposure to asbestos comes from two sources: occupational histories of cases of mesotheliomas in the records of pathologists, and prospective studies of asbestos workers. Studies of both types have now been reported from more than a dozen countries, and confirm the association between mesotheliomas and past exposure to asbestos. But the first approach has always revealed some cases with no association with asbestos. The proportion has been smaller in more recent surveys where the information has been more complete. 'Blind' studies of history of exposure to asbestos in mesotheliomas and case controls have been made in Scotland and in Canada (McEwen *et al.* 1970, McDonald *et al.* 1970) (Figs 2 and 3). In Scotland only 5% of the cases of mesothelioma gave no history of asbestos exposure, and the primary lung cancer controls lie between the mesotheliomas and the cardiovascular controls. In Canada, where the survey was country-wide and covered all cases over an eight-year period, there was a much higher proportion with no likely exposure to asbestos, but there was still a significant excess in the mesothelioma group of definite and probable exposure to asbestos. The cases related to asbestos in Canada occurred more often in the manufacturing than in the mining sections of the industry. This survey indicated an annual incidence of about one per million. The marked difference between findings in Scotland and Canada is not yet explained.

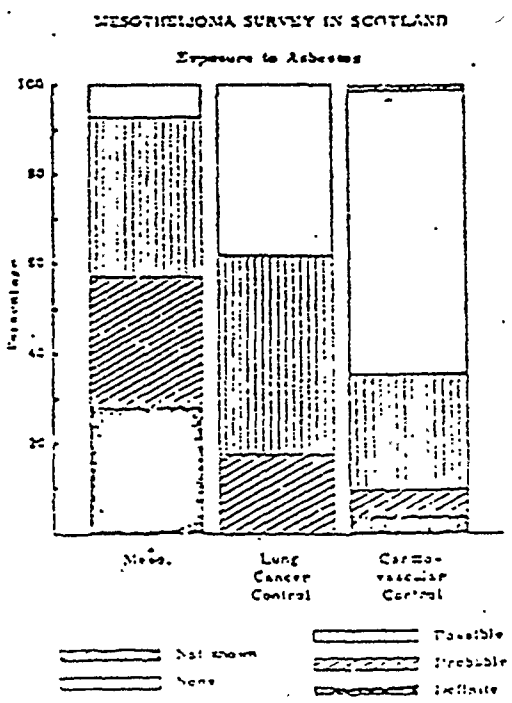

Fig 2

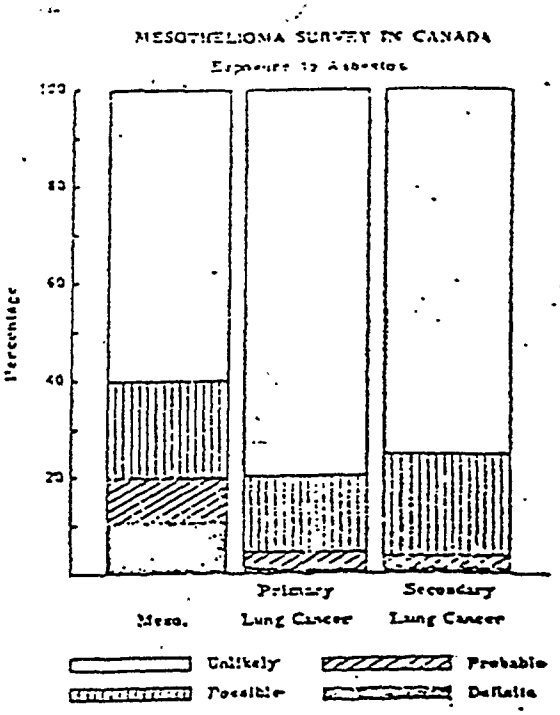

Fig 3

Type of fibre: The reports of mesotheliomas from many countries where the proportion of different types of fibre used has varied widely make it highly improbable that only one type of fibre is always responsible. But evidence of a bigger

variation of mesothelioma risk with type of fibre than is found for lung carcinoma comes from several sources.

In South Africa mesotheliomas were first discovered in relatively large numbers by Dr Wagner in association with the North West Cape crocidolite mines in about 1955 (Wagner *et al.* 1960). In the succeeding fifteen years continuing surveillance of all asbestos producing areas in South Africa has confirmed that excess of mesotheliomas is almost limited to the crocidolite area, with very few reported from amosite mining and none from chrysotile (Wagner *et al.* 1971, Harington *et al.* 1971).

Groups of mesotheliomas have occurred among past workers exposed to crocidolite alone and where the dust concentration has not always been high. For example, Dr J S P Jones (1972, personal communication) discovered nine cases of mesothelioma in a group of workers employed placing crocidolite asbestos filter pads in gas masks during the war. Five of the cases occurred in 200 of the more heavily exposed subgroup, but the process was not excessively dusty. Another example is a small group discovered by Dr H Lewinsohn (1972, personal communication). The men had worked in a factory in the mid 1920s (before asbestosis was discovered!), where possible heavy exposures to crocidolite occurred following its processing in one area of the works. Six cases of mesothelioma have subsequently developed among those working within a few feet of each other. No other cases of mesothelioma have occurred in this factory, where exposures were mainly to chrysotile or to very much lower amounts of crocidolite.

In general more mesotheliomas have occurred after exposure to mixtures of amosite, crocidolite, and chrysotile asbestos than to chrysotile alone (Table 5). Up to the present the proportion of deaths due to mesotheliomas in groups at greatest risk is about 5%. This compares with over 90% developing bladder tumours in groups most exposed to β-naphthylamine (Williams 1962). Only a few cases of mesotheliomas have occurred following exposure to chrysotile alone (Mancuso & El-Attar 1967, McDonald *et al.* 1970, Constantinides 1972, personal communication, Wagner *et al.* 1971), but they have occurred after exposure to pure amosite in the manufacturing of insulation (Selikoff *et al.* 1973). So far anthophyllite seems innocent in this respect, despite intensive search in Finland (Kiviluoto & Meurman 1970).

Dose of dust, duration of exposure, and relation to sex: Information about the dose of dust and the risk of mesothelioma at present is qualitative. There are many reports of cases following short exposures – a few weeks. Cases have followed exposure to dusty clothes in the home. In addition asbestosis is often absent, again indicating a relatively small dose of dust (Wagner *et al.* 1971), but there is evidence of a relation of risk to dose and length of exposure (Newhouse *et al.* 1972) (Table 6).

Cofactors: So far no definite relation between cigarette smoking or other factors and mesotheliomas has yet been revealed.

Rising incidence of mesotheliomas in UK: A Register of Mesotheliomas was started in 1962. Cases from the records of pathologists and the Cancer Registers have been accumulated with new reported cases. Over the years the incidence of confirmed cases has risen as follows: before 1955 total deaths recorded, 17; 1955–59, 6.2 deaths per annum; 1960–64, 17.4 deaths per annum; 1965, 24; 1966, 28; 1967, 43; 1968, 73. When distributed geographically, the new cases nearly all come from places where past occupational exposures are known to have occurred.

In summary, although it is known that a majority of the population carry a small burden

Table 5

Proportion of deaths from lung cancer and mesothelioma related to occupation and type of asbestos

	Fibre	No. of workers	Deaths				
			Total	Lung cancer No.	Lung cancer %	Mesothelioma No.	Mesothelioma %
Mining and milling (Quebec)	C	9981	2413	97	4	3	0.12
Manufacturing insulation (London)	C, A, Cr	4806	436	42	9.7	20	4.6
Applying and manufacturing insulation (New York)	C, A	632	380	72	19.0	22	5.8
Applying and manufacturing insulation (Detroit)	C, A, Cr	165	98	24	24.4	7	7.1

Table 6

Mesothelioma risk per 100,000 person years among workers in asbestos manufacturing industries and textiles (C, A, Cr)

	Years of exposure	Light exposure	Heavy exposure
Men	<2	25	71
	>2	102	156
Women	<2		84
	>2		141

of asbestos fibres in the lung, it seems likely to be too small to produce a detectable effect; this is so even though the amount of asbestos required to produce mesotheliomas of the pleura is considerably less than that which will cause asbestosis or lung cancer.

Pathogenesis

How can this rather confused picture be fitted into a hypothesis of the pathogenesis of the two principal types of cancer? This is not the place to elaborate on the alternatives, but I will outline one hypothesis being developed by my colleague, Dr V Timbrell.

We have to explain: (1) A very low mesothelioma and low lung carcinoma risk in chrysotile mining and milling, despite very heavy dust exposures in the past. (2) The much higher mesothelioma risk in mining and milling crocidolite than amosite. (3) A high mesothelioma and lung carcinoma risk in insulation workers where exposures have been to mixtures of fibre types, but also apparently a high risk in pure amosite exposure in manufacturing insulation. (4) A lung carcinoma risk and asbestosis in anthophyllite mining, but no mesotheliomas. Dr Timbrell's hypothesis can be divided into two parts: first, the influence of diameter, length, and shape of a fibrous particle on where it is deposited in the lung; secondly, the influence of diameter and length on its subsequent movement in the lung and its entrance into a cell without killing that cell.

The first part is based on theory supported by experiments, and it shows that it is diameter and shape (curved or straight), and presence of adherent particles which most affect the site of deposition in the lung. The finer and straighter and cleaner the fibre, the further it will penetrate; length is of secondary importance. From this it follows that: (1) In mining and milling where the fibres are not fully separated and are, therefore, in bundles of large diameter and also have adherent particles, the proportion of dust which

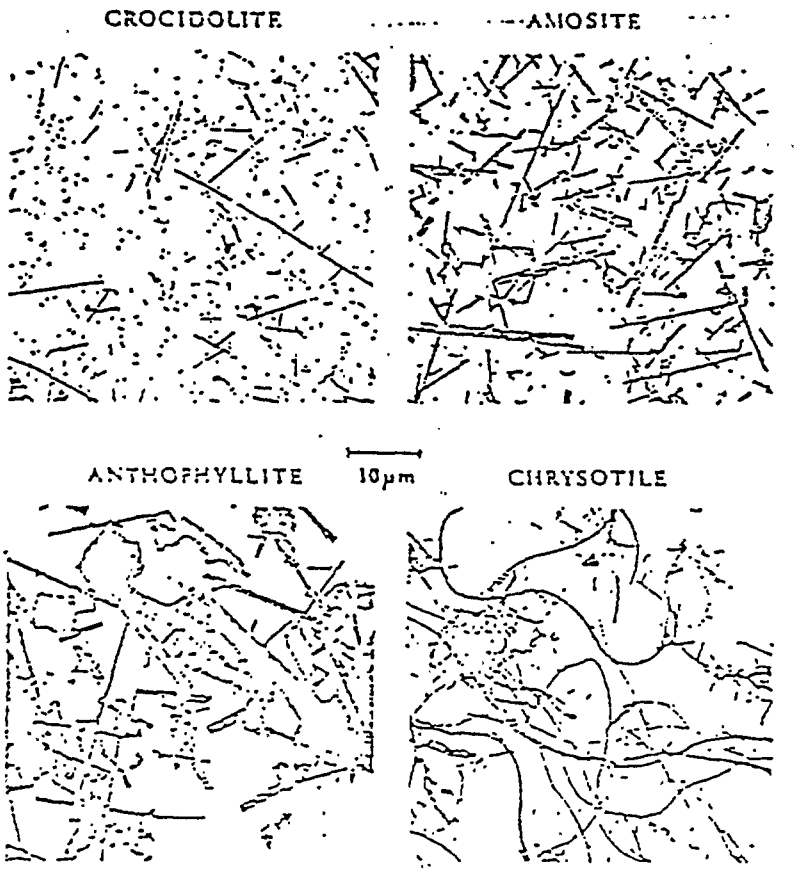

Fig 4 Types of asbestos fibre as seen under electron microscope

Table 7

Asbestos cancer: prevention

(1) Substitution of other insulants: glass fibre, mineral wool, calcium silicate, plastic foams, &c.
(2) Use of the safer types of asbestos (chrysotile and amosite)
(3) Reduction of dust exposure, measurement of dust levels (Codes of Practice, Asbestos Information Committee; Asbestos Regulations 1969)
(4) Periodic examination of workers; biological monitoring (clinical, X-ray, lung function)
(5) Record linkage: process – type of material used; biologically appropriate dust measurements; medical records
(6) Continuing research, including cost/benefit analysis

will get to the pleura is small compared to later stages in manufacture where the fibres are cleaner and more separate and, therefore, smaller in diameter. (2) The fibre diameter and shape will also affect where the dust goes into the lung after deposition.

Fig 4 shows how different the fibres are under the electron microscope. Anthophyllite is much larger in diameter than the rest; therefore, less of it will penetrate deeply into the lung and little to the pleura; in addition, the diameters may be too big to enter cells, except phagocytes, without destroying them. Chrysotile is very fine, but it is also curly, especially when fully opened; much less will, therefore, reach the periphery than would be anticipated from its diameter because interception occurs higher up the respiratory tract; it also happens to be relatively more soluble than the other types; enough may, however, reach the respiratory bronchioles to cause asbestosis. Amosite and crocidolite are very closely related chemically and until recently were thought to be so similar that differences in mesothelioma rate in mining the two types could not be due to differences in the fibre (Wright 1968). It has now been shown that there is an important difference in diameter (Timbrell *et al.* 1971): crocidolite has about one-third the diameter of amosite and, as the falling speed is proportional to diameter squared, the amosite fibres will settle nine times more rapidly than the crocidolite. Thus, there will be less chance of an amosite fibre reaching the periphery of the lung and the pleura. We still have to explain why the crocidolite mined in the Transvaal does not seem to cause mesotheliomas. Surprisingly, it turns out that the crocidolite from this area has the same diameter as the amosite and is not like the fine material from the North West Cape. The crocidolite from Australia (no longer mined) is even smaller in diameter than the fibre from the North West Cape.

This hypothesis goes some way to unifying a confused epidemiological picture. It has the advantage that it may be testable in animals, because rats suffer all the diseases caused in man by asbestos, and it will be possible to measure the amount of dust of different types of asbestos going to the various structures in the lung; also, it has recently been shown that organ cultures of lung and pleura react to the different types of asbestos (Rajan *et al.* 1972). It is, of course, recognized that cigarette smoking is a major factor in the lung carcinomas. Work is in progress to study the combined effect of asbestos and cigarette smoke in animals.

Prevention

What is being done to use this epidemiological knowledge for prevention? Table 7 summarizes steps now being taken in this country. I have already mentioned the extensive review of the uses of asbestos by the Royal Navy, leading to its replacement by other insulants for a large number of purposes. Other large users have done the same.

Imports of crocidolite into the UK have fallen strikingly, compared with an increase with that for the rest of the world. Last year no crocidolite was imported into the UK. Are those countries into which greatly increased amounts are now going taking sufficient precaution in its use?

The UK was the first to introduce new and more exacting environmental standards for asbestos (Department of Employment and Productivity 1969), following the recommendations of the British Occupational Hygiene Society (1968), and also to develop Codes of Practice, worked out by the Asbestosis Research Council in consultation with the Department of Employment; a number of other countries have followed this lead. Following the recommendations of the Advisory Panel of the Senior Medical Inspector, a scheme for the periodic surveillance and long-term follow up of asbestos workers has been put into operation by the Department of Employment in co-operation with industry (Ministry of Labour 1967). This will be gradually extended to cover all groups potentially exposed, as they are identified. The records of this scheme will be linked with the environmental measurements recorded by the employers and the Industrial Hygiene Unit of the Department of Employment.

I would like to stress the last item. Research by Dr E R A Merewether in 1929 revealed the high risk of asbestosis in the textile industry. Alas, no continuing research effort was maintained, despite his proposals at that time (Merewether & Price 1930); had this been done, better controls would almost certainly have been introduced earlier. We shall need to know whether the controls now being introduced are adequate and must also look critically at substitute materials to be sure they are innocuous. If it should turn out that diameter, length, and straightness of a fibre influence its chances of getting into the deeper parts of the lung,

and then into cells capable of becoming malignant, other fine fibres will have to be looked at critically.

I should also like to stress the cost/benefit analysis. Epidemiology is largely directed to assessing ill health. To produce a balanced assessment, we need methods to assess the benefits – for example, the protection afforded by asbestos against fire damage to life and property. The safety of our whole transport system depends heavily on asbestos, due to its essential use at present in brake linings and clutch plates. A start has been made by the Institute of Social and Economic Research, University of York, to assess the kind of information needed to make such a cost/benefit analysis (Akehurst 1973).

I have attempted to bring together in this account some of the pieces of a jigsaw puzzle; a number of the pieces have been shaped by those with whom I have been fortunate to work over the last ten years. They do not yet all fit. But the picture is clearer and important steps towards prevention have already been started, based in part on knowledge acquired as a result of a widely based research effort.

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DISCUSSION

Dr J R Glover (*Cardiff*) asked what was meant by respirable dust in relation to asbestos, i.e. did the definition depend on more than fibre length? He also enquired if there was any use in biologically monitoring asbestos workers; in other words, was there any evidence that a worker who had once contracted a sign or symptom of one of the three fatal lung diseases caused by asbestos ever been helped by taking him away from exposure to asbestos dust?

Dr Gilson, in reply to Dr Glover, said that no single parameter of the dust could be used. It depended on the diameter and the shape – whether straight or curly – but to a less extent on length; density was also relevant.

Dr Gilson said that the use of biological monitoring could not yet be fully assessed. It was not known whether there was a detectable stage at which removal from further dust exposure affected prognosis in relation to asbestosis and asbestos cancers. It would be possible to find this out only by prospective studies of appropriate groups, and this was now being started.