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Volume 2: final report of the advisory committee

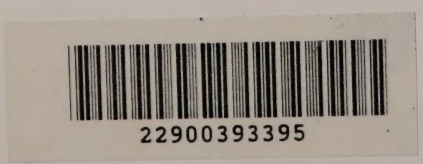
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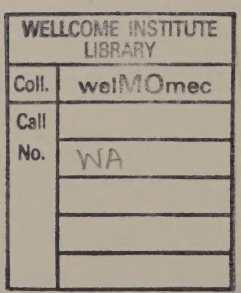
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Preface

This volume contains papers commissioned by the Advisory Committee and used in preparing their final report.

The Ill Effects of Asbestos Upon Health by E D Acheson and M J Gardner is the full report from which the agreed summary at Chapter 3 in Volume 1 is drawn. Material from **Asbestos Control Limits** by J Steel is incorporated more fully into the main text but is particularly relevant to Chapters 3 and 4. The Committee endorses the general conclusions, but responsibility for the content of the papers rests with the authors. The third paper summarises the experience of controlling exposure to asbestos in other countries based on information from the countries concerned, and the fourth gives the results of an exploratory survey into the technical feasibility of substitution for asbestos in particular uses.

The first volume contains the main text of the report, setting out the principal facts and argument on which the conclusions are based. For convenience it also contains a summary of the recommendations from the Advisory Committee's two previous reports **Work on Thermal and Acoustic Insulation and Spray Coating** and **Measurement and Monitoring of Asbestos in Air.** These reports were published separately as consultative documents on 1st June 1978.

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The ill-effects of asbestos on health

E D Acheson and M J Gardner

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1 Introduction

This report originated as a working paper commissioned for the Medical Working Group of the Advisory Committee on Asbestos. Its principal terms of reference were to provide a review of the available scientific evidence on the ill-effects of asbestos in man, a critical commentary on that evidence, and a summary of conclusions in a form suitable for the non-specialist.

The authors were asked to comment on two points in particular. The first was the relationship of fibre type to risk of disease. In other words, are the various types of asbestos fibre met with occupationally and environmentally equally hazardous in terms of asbestosis, lung cancer, mesothelioma or other malignant disease? The second was the relationship of dose to risk in respect of each of the diseases mentioned above.

In order that it may be possible to read the report as a self-contained entity, sufficient background information about the mineralogy of asbestos and the nature of asbestos related disease is given to set the issues in their appropriate context. A brief explanation of some of the more important epidemiological and statistical terms is provided for the non-specialist as Appendix 1.

Work in experimental animals is mentioned where it seems to be relevant to problems in man. However, no treatment of any aspect of the subject other than the questions immediately in view has been possible.

For further information readers are referred to four recent excellent reviews. The *Public Health Risks of Exposure to Asbestos* reviewed for the Commission of the European Communities (1977)[1] by Zielhuis; *Asbestos-Related Diseases of the Lung and Other Organs* by Becklake (1976)[2]; *Asbestos Cancer, Past and Future Hazards*, by Gilson (1973)[3]; and the latest monograph on Asbestos prepared in 1977 by the International Agency for Research on Cancer for the World Health Organisation[4]. In addition the US National Institute for Occupational Safety and Health have recently prepared a re-examination of the problem of asbestos and health. The review of environmental data in this paper is the most detailed which has appeared, and is particularly useful[52].

The evidence available for discussion in the report falls under three headings:

(1) Data published in the scientific literature.

(2) Scientific data made available to the Working Party but as yet unpublished.

(3) Evidence submitted to the Advisory Committee on Asbestos by the industry and others, a selection of which has been published[5].

Data under the second heading have been made available for this report with the permission of the authors.

The third category consists of data on a wide variety of subjects including dust measurements in work places and in the environment, imports of asbestos to the United Kingdom by fibre type and its consumption by principal product groups, an assessment of the risks of damage and abrasion to various asbestos-containing products in buildings, reports of asbestos related disease, and many statements of fact and opinion outside our terms of reference. An important aspect of these extensive data in relation to this report lies in their identification of occupational situations where valuable information has already accumulated and which may be a basis for future studies. A summary of these is shown as Appendix 2.

As the data about asbestos related disease given in the evidence by the industry are not in a form that can readily be related to dose and do not include any systematic attempt to follow workers who have left, these have not been used as a primary source of evidence in the chapters on fibre type and dose-response curves. However, a crude breakdown of the data by duration of use of amphiboles and chrysotile has been made and related to known cases of asbestosis and mesothelioma. It is shown as Appendix 5, and although it has not been used in the arguments, supports the principal conclusion of Chapter 4 of Volume 1.

In a few instances, particularly where the primary source is a US government paper, it has not yet been possible to locate and check data used in this review. The references to such material are marked with an asterisk in the bibliography.

This paper views the problem of the relationship of asbestos to health from one angle only, namely from the point of view of its deleterious effects. No account is taken of its undoubted benefits to health, e.g. in the prevention of death and injury from fire and explosion, and the improvement of working conditions in respect of noise and heat, all of which must be taken into account before rational decisions about public policy can be taken. Nor is any view taken in this report on the possible hazards to health of substitutes.

In the event, although there were differences of opinion about various points of emphasis, the report as a whole was accepted by the Medical Working Group for whom it had been written. As a result of the discussions of the report which took place, various errors were corrected and additional information was made available and incorporated in it.

Although the earlier sections contain expressions of opinion personal to the authors the section entitled 'Summary and Conclusions' represents a concensus of the Medical Working Group and as such is also with certain textual changes incorporated in the Final Report of the Advisory Committee on Asbestos.

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2 Summary and conclusions

1 Asbestos is the generic term for a group of naturally occurring mineral silicate fibres of the serpentine and amphibole series. Although one fibrous serpentine mineral (chrysotile) and five fibrous amphiboles (actinolite, amosite, anthophyllite, crocidolite and tremolite) are generally classified under this term (Fig 1), for practical purposes the types of fibres which are important in the United Kingdom are chrysotile, crocidolite and amosite. On the basis of the data currently available our assumption is that the imported commercial varieties of asbestos mentioned above form the principal source of these minerals with which man comes in contact in the United Kingdom. However, more information about possible contamination of the environment from non-commercial deposits of asbestos and other fibrous minerals in other rocks and ores is required. A study of import trends of commercial asbestos by fibre type (Table 1, Fig 2(a) and (b)) shows that in 1975 chrysotile accounted for about 85% of the total imports of fibres, almost the whole of the remainder being composed of amosite. Expansion in imports of amosite since the second World War has been much greater than of chrysotile so that, both in proportional and absolute terms, the amount of amphibole fibre being imported into the UK is considerably greater than it was in 1945.

2 A summary of the uses of asbestos fibres (Table 2A) shows that asbestos cement products for buildings (32.8%), fillers and reinforcements (22.0%), floor tiles (12.2%) and friction materials (12.1%) consume a large proportion of imported chrysotile but there are many other uses. On the other hand the range of usage of amosite is very restricted, being dominated by the production of fire resistant boards (84.3%) with the construction of pressure piping (7.9%) a poor second. Data about the trends in usage of chrysotile (Table 2B) shows the manufacture of jointings and packings, friction materials, and fillers and reinforcements to have been the main areas of growth since the war. The last sub-head mentioned because of its scale, rate of growth and the variety of products manufactured (it includes felts, millboards, paper, filter pads, underseals, mastics, adhesives and coatings), is an area where much more information about dust levels and conditions of work is required. Although imports of crocidolite ceased in 1970 substantial quantities of this material remain in insulation materials in buildings, ships, locomotives and railway carriages, in cement products including pressure piping and in battery boxes.

BIOPHYSICS OF ASBESTOS

3 The properties of asbestos fibres which determine their commercial and biological interest are their capacity to split longitudinally into finer fibres; their insolubility and resistance to destruction by heat, acids and alkalis; their effectiveness as thermal, electrical and acoustic insulating material; and their ability to be woven. Once embedded in animal tissues some of the fibres remain within the body for extended periods of time. The proportion remaining is higher for amphiboles than for chrysotile.

4 As the likelihood of a fibre to remain airborne or to settle is determined by its diameter, and the number of fibres per unit mass will be greater in the case of finer fibres, it is clear that fibre diameter is the key to the number of fibres inhaled. For a given mass of airborne amphibole dust the number of fibres available for inhalation at a given moment and the length of time the dust cloud remains airborne will be greater for crocidolite than for amosite and greater for amosite than for anthophyllite. For chrysotile, which has a greater tendency to divide into fibrils than the amphiboles, the aerodynamics of the dust will depend to a greater extent on the amount of milling or grinding (Fig 3), so its respirability may be similar to that of the amphiboles in some circumstances.

5 Once inhaled, the depth of penetration of asbestos fibres depends upon the length and diameter of the fibres and upon their straightness. Fibres longer than about 200 μm are impacted in the nose and ejected and swallowed, some of the fibres shorter than about 10 μm are probably incorporated in macrophages and ejected by the cilia of the respiratory passages, while others remain free. The fate of the remaining fibres is thought to depend upon diameter, length, and possibly straightness. Animal experiments show that, for reasons not fully understood, much less chrysotile than amphibole fibre remains in lung tissue after inhalation has ceased.

6 Fibres of a wide range of chemical structures (including substances as diverse as glass and aluminium oxide as well as asbestos) have been shown to produce mesotheliomas after injection into the pleural or peritoneal cavities in animals if the configuration of the fibres falls within a certain range. Experiments showed that for asbestos, glass fibre and aluminium oxide, the degree of carcinogenicity is related to the proportion of fibres with diameters between 0.5 and 2.5 μm and lengths 10-80 μm. In later experiments using glass fibre only, the highest incidence of tumours occurred with fibres up to 1.5 μm in diameter, including very thin fibres of diameter less than 0.25 μm and of length greater than 8 μm. It is important to point out that 0.5 μm represents the approximate limit of light microscopy in respect of fibre diameter.

7 So although the doses administered were relatively massive and the results of intrapleural implantation of fibres in rats cannot be used as a reliable predictor of the risk to man through inhalation, these experiments strongly suggest that the current optical methods of

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measurement underestimate the number of potentially carcinogenic fibres in samples. The development of methods which measure the whole range of respirable fibres is urgently needed.

Asbestos related disease, portals of entry and types of exposure

ASBESTOS RELATED DISEASE

8 *Asbestosis*, which may be defined as fibrosis of the lungs caused by asbestos dusts which may or may not be associated with fibrosis of the parietal (outer) or pulmonary (inner) layer of the pleura, lung cancer (carcinoma of the bronchus), and mesothelioma of the pleura and peritoneum are universally accepted as diseases with a causal relationship to the inhalation of asbestos fibre. Benign pleural effusion (fluid developing in the space between the pleural membranes lining lung and chest wall) may occur acutely in association with other asbestos-related lung diseases or as the only or most prominent disease process. It may be discovered by accident or associated with pain, fever and malaise. Thickening of the pleura may occur, alone or in association with fibrosis of the lung, and may be symptomless or associated with chest discomfort and restriction of breathing. Asbestosis, which by definition is specifically related to asbestos, is difficult to diagnose because its onset is gradual, and because the symptoms and signs may occur in other diseases and are often difficult to detect. Minor radiological changes attributable to asbestos may exist for many years without symptoms or progression. Lung cancer is easier to diagnose than asbestosis but the tumours which occur in relation to asbestos have no special diagnostic features. As tobacco-related lung cancer is common and the effects of tobacco and asbestos are known to interact synergistically in smokers exposed to asbestos, it may be impossible to determine in a particular case whether or to what extent a bronchial tumour is related to asbestos. As far as mesothelioma is concerned, although there are also diagnostic problems, the fact that the disease is rare in the population at large and that asbestos is the only environmental cause of the condition in the United Kingdom so far identified, smoking having no effect, make the attribution of individual cases to asbestos more certain.

9 All the asbestos related diseases mentioned above have in common the existence of a delay or lag-period, usually of many years, between first exposure and onset of symptoms. This has a number of serious implications of which the most important are that the disease may occur many years after the worker has left the relevant job; cases of the disease occurring at present do not relate to current conditions of work; and a period of several decades must elapse before it is possible to give a final answer about the appearance or control of an asbestos related hazard.

10 The evidence for the existence of a risk of cancer of the gastro-intestinal tract associated with exposure to asbestos is discussed (Tables 3(a) and (b)) and it is concluded that, although an excess of gastro-intestinal cancer has not been found in all surveys, substantial excesses are seen in surveyed industrial populations in which workers have been exposed to mixtures of asbestos rich in amphiboles* and in the only population formally studied thought to have been exposed only to amosite. There is insufficient pathological information concerning the accuracy of diagnosis in deaths ascribed to cancer of the gastro-intestinal tract. It is not improbable that some of these deaths are due to misdiagnosed peritoneal mesotheliomas. Finally it is concluded that there is evidence of a risk of cancer of the larynx in association with the inhalation of mixtures of asbestos containing amphiboles, but that this risk is very much smaller in absolute terms than the risk of any of the conditions mentioned above. (Table 3(c)). In one study of the relationship between chrysotile and cancer, no evidence was found of an increased risk of cancer of the larynx.

The relative order of frequency of the types of asbestos-related disease

11 By summing the experience of the various industrial populations which have been followed in different parts of the world (Table 11 and see Appendix 4) it is possible to compare the relative importance of the mortality of the different types of asbestos related disease as they have ocurred in men exposed to the industrial conditions of the past. The excess mortality found in these studies is accounted for in order of importance by lung cancer (associated or not associated with asbestosis), asbestosis, mesothelioma and gastrointestinal cancer. Included in this excess mortality for lung cancer is a substantial element due to the synergistic action of tobacco with asbestos, which cannot therefore properly be attributed to the effect of asbestos alone. These studies indicate the relative importance of lung cancer, asbestosis and mesothelioma in relation to the effects of past asbestos exposure and cigarette smoking patterns. The frequency of these diseases in the future will depend not only on control of exposure to asbestos dust but also upon future trends in cigarette smoking.

12 The study of British death certificates suggests a different picture, namely that asbestos-related mesothelioma is a more frequent cause of death than asbestos-related lung cancer (Table 20). However, death certificates are probably an unreliable indicator. For example, the mention of asbestos on death certificates of people dying from lung cancer who were smokers and who were also exposed to asbestos is almost certainly deficient.

*If in fact the Rochdale industrial population or a substantial part of it was exposed to mixtures of asbestos rich in amphiboles, the absence of an excess of gastro-intestinal cancer provides an exception to this generalisation.

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PORTALS OF ENTRY

13 Although by far the most important portal of entry of asbestos fibres to the body is the respiratory passages, the fibres may also be swallowed (secondarily by swallowing contaminated nasal or bronchial secretions or primarily by the ingestion of contaminated water or food). Accidental parenteral administration of asbestos may also have taken place from the use of syringes, drugs and medicaments containing asbestos.

TYPES OF EXPOSURE

14 A classification of types of exposure to asbestos is given in Table 4. Direct occupational exposure includes exposure during mining and milling, transporting, packing and processing raw fibres, installing, adapting and working on new products, repairing and servicing existing products, demolishing buildings and vehicles containing asbestos products, and dumping asbestos-containing waste. Indirect occupational exposure (to persons working in the vicinity of men handling asbestos), domestic exposure (to domestic contacts, e.g. wives and children of asbestos workers), leisure-time exposure, neighbourhood exposure and general exposure (through ambient air, food or water supplies) also occurs in appropriate circumstances, and a proportion of the fibres present in the environment, particularly in water, may derive from deposits of asbestos in other rocks and ores.

The relationship of medical effects to fibre type

ANIMAL EXPERIMENTS

15 Experimental work on animals shows that inhalation of similar respirable masses of UICC standard samples of chrysotile produces at least as many malignant lung tumours and mesotheliomas as do standard samples of the amphiboles. Furthermore in pleural implantation experiments chrysotile, when the configuration of the fibres is appropriate (i.e. superfine (SFA) Canadian chrysotile), produces as high a yield of mesotheliomas as crocidolite (Tables 7, 8 and 9). However using asbestos (prepared by hammer milling), which probably resembles more closely that used in the past by industry, similar respirable masses of chrysotile and amosite have been found to be less carcinogenic than crocidolite.

LUNG CANCER IN MAN

16 Evidence in man about the relationship of fibre type to lung cancer is inconclusive, but where it exists indicates that exposure to the amphiboles crocidolite or amosite (or to mixtures with chrysotile rich in them) has been more dangerous than to chrysotile or anthophyllite alone (Tables 11 and 21). However, as important gaps in our knowledge of this aspect of the field remain unanswered, the weight which can be attached to this conclusion at present is limited. The high relative risks of lung cancer reported in a single study of workers making insulation material containing amosite even after very short exposure is worrying in view of the increase in utilisation of this material in the United Kingdom since the war (Table 28). There is no reliable information from South African amosite miners to help us on this point. It is concluded that a firm judgment on the question whether amosite has been more dangerous than chrysotile in respect of lung cancer is not possible at present and that further work should be commissioned urgently.

ASBESTOSIS IN MAN

17 Quantitative evidence about impairment of lung function in man in relation to fibre type is limited to one study which suggests that crocidolite may have been more harmful than chrysotile. Information from the same amosite insulation manufacturers as were mentioned in connection with lung cancer, and their families, shows that radiological changes suggestive of asbestosis occured after relatively short exposure. Evidence for a difference in risk of asbestosis with fibre type is at present slight.

MESOTHELIOMA IN MAN

18 As far as mesothelioma is concerned, evidence from miners; from process workers exposed to a single fibre type; from the distribution of neighbourhood and domestic cases; and from the geography of mesothelioma, when combined, presents a powerful case from four different points of view that crocidolite has been more dangerous than chrysotile and anthophyllite. The position of amosite may be intermediate between crocidolite and chrysotile. There is no doubt that some crocidolite was used at Rochdale, but the significance of this as a cause of the mesothelioma cases there is not certain. It can be concluded that exposure to chrysotile alone so far has rarely been shown to cause mesothelioma.

19 The death rate from mesothelioma as certified on death certificates in Britain, although small in absolute terms, has doubled in the decade 1967-76 (Table 20, Fig 17). Although this trend may be affected by a recent tendency to diagnose mesothelioma more readily, the evidence concerning occupational dust exposure conditions in earlier years (to which deaths occurring at present are mainly due) suggests that the number of deaths from mesothelioma may be expected to increase further before the peak is reached.

CANCER OF THE GASTRO-INTESTINAL TRACT IN MAN

20 Significant excesses of cancer ascribed to these sites (see also para 94-97 for reservations about the diagnosis in these cases) have occurred in most populations of industrial workers heavily exposed in the past to mixtures of amphiboles with chrysotile and in one population thought to be exposed exclusively to amosite. The evidence that exposure to chrysotile alone has increased the risk of gastrointestinal tract cancer is less consistent (Table 3a).

CHANGES IN INDUSTRIAL PRACTICE

21 Inhalation experiments in animals show that all fibre types of asbestos have the potential to produce

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similar amounts of the main types of asbestos-related disease if the physical configuration of the fibres in the dust cloud is appropriate (Table 7). Intrapleural injection experiments also show that finely divided chrysotile can be prepared in such a way that it produces a similar number of mesotheliomas to crocidolite (Table 9). It follows that any change in industrial practice in the direction of the production of more finely divided chrysotile fibre is likely to increase the health risk.

Dose-response relationship

22 The incidence and severity of the pathological response to increasing doses of asbestos in man is crucial to framing a rational policy about an acceptable level of exposure. Thus, if there were a level of dose below which no risk of sickness or death were incurred (i.e. a threshold, as defined in para 169) at least such a level would be acceptable. In contrast, if it could be shown that the largest increments in risk per unit of exposure ocurred at the lower end of the range of dose, a policy of very strict control might be indicated. In order to contrast some of the possible responses and to underline their significance hypothetical examples are illustrated in Fig 6.

23 For a number of reasons which are given in detail in Section 6 the available measurements of dose and of response are subject to considerable error and uncertainty. As a result there is plenty of room for differences of opinion about the validity of the conclusions reached in this section. In subsequent paragraphs we set out our conclusions about the dose-response relationship which can be demonstrated from the available data based on the experience of industrial populations. But first we describe one of the most important sources of uncertainty, namely the problems of dust estimation.

PROBLEMS OF DUST ESTIMATION

24 To draw conclusions about dose-response relationships, we must be able to relate the frequency of disease with dust measurements. But methods of measuring dust have differed from time to time and from place to place, and relating measurements arrived at by the different methods is not straightforward. A detailed account of the problem is given in Dr J Steel's paper. Here, we restrict ourselves to summarising the main sources of uncertainty, so as to make it clear why many of our later conclusions must be tentative.

Factors which may vary are:

(a) the sampling instrument used;
(b) the location of the sampling instrument relative to the workman;
(c) the nature of the dust counted (all particles, or fibres only);
(d) the evaluation technique.

25 The standard sampling instrument most commonly used in Britain today is the membrane filter sampler. But before 1964 the commonest instrument was the thermal precipitator, while in the USA and Canada many of the data are derived from midget impingers. Membrane filters are used to count fibres, while midget impingers can only be used to count total particles, and thermal precipitators can be used to count fibres only very inaccurately. There are considerable uncertainties in converting particle counts (particularly from the impinger) to fibre counts, as correlations are poor. Before 1970, membrane filter sampling was typically static, with the instrument in a fixed position in the workplace. Since 1970, personal sampling has become more common. Studies have shown that personal sampling increases the number of fibres counted in a given workplace by a factor which varies widely both within and between manufacturing processes, but which, at dust levels around 2 fibres/ml, is probably between 1 and 2 (Table 37).

26 The results of fibre counts are affected by the method of counting used. The current method of 'eyepiece graticule' counting has been introduced since 1969. When compared with the older 'whole field' counting technique, graticule counting increases fibre counts by a factor which has been estimated as being of the order of 2 - 3 (see Table 37).

27 The combined effect of these last two factors means that an atmospheric chrysotile concentration which would have been evaluated as 2 fibres/ml in 1968 might well today be evaluated as somewhere between 4 and 10 fibres/ml.* So it follows that these changes in sampling and evaluation techniques have brought about a *de facto* tightening of the hygiene standard since 1969.

ASBESTOSIS

28 The signs of developing asbestosis are much less reliable as objective end points than is the occurrence of a malignant tumour. Not only are there major difficulties in the definition of asbestosis of sufficient severity to cause symptoms, but the clinical, radiological and other signs on which the diagnosis is based are non specific and subject to wide variations in interpretation. Furthermore, at present comparatively little is known about the progression of the condition particularly in the stages before symptoms have developed.

29 A study which permits estimates of the occurrence (incidence) of new cases to be related to dose is the second report from Rochdale. Although they are all somewhat irregular in form, the responses of the three parameters studied (certified asbestosis; possible asbestosis; and crepitations) are directly proportional to the cumulative dose (Fig 7). For certified asbestosis, as might be expected, there appears to be a threshold at an estimated cumulative dose somewhere between zero and 100 fibre

*Alternatively a concentration measured today as 2 fibres/ml. might have been measured as between 0.4 and 1 fibre/ml. in 1968.

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years per ml, below which certification has not taken place. However an annual incidence of 0.5% (Standard error (SE) 0.2%) of certified asbestosis (Table 22) has occurred after cumulative doses of less than 100 fibre years per ml, i.e. within a working life at 2 fibres per ml. The annual rates of incidence for 'possible asbestosis' and 'crepitations' are higher at any given dose level and suggests the possible occurrence of asbestos-related disease at cumulative doses of less than 50 fibre years per ml. Restricting consideration to the men first employed after 1950 in whom measurements of dose and response were more accurate does not alter these conclusions significantly. Of two studies from North America, one suggests that impairment of lung function may be detectable at cumulative doses at the lower end of the range (between 10 and 200 million particle years per cubic foot) (Fig 9) while the second suggests a threshold of 200 fibre years per ml below which no impairment of lung function takes place. The only study of mortality from asbestosis which can be related to dose shows an increase of mortality with dose (Fig 18).

30 Any application of the data discussed above to present or future working conditions depends upon the relationship of the measurements of dose made in the past to those on which the current and future standards are enforced, and upon the reliability of the clinical and other parameters of asbestosis. If the data are taken at their face value they indicate the occurrence of serious asbestos related lung disease at an annual incidence rate of at least 0.5% per annum after a lifetime of work within the present hygiene standard. On the other hand, if it is accepted that the doses inhaled by these men in the 1950s and 1960s have been underestimated by a factor of three or more, as compared with current methods, the hygiene standard as currently enforced would have been adequate to control serious disease (Fig 19). The interpretation of this important part of the evidence therefore depends largely on the view taken of the dust measurements used in these studies and upon the accuracy of the diagnosis of certified asbestosis at Rochdale. There is also uncertainty about how far it is reasonable to derive from data about asbestos textile workers conclusions extending to all situations where occupational exposure to asbestos occurs. Additional studies are also being undertaken by the British Occupational Health Society. So far as the committee can tell on the basis of preliminary reports, these studies are unlikely to alter previous findings substantially.

LUNG CANCER

31 For lung cancer the available data in man, all of which are derived from industry, show an increase in risk with increasing dose of dust, and we find no evidence within the range of dust levels studied for a threshold of dose below which there is no evidence of risk. For the Quebec chrysotile miners and millers and for the retired New Jersey asbestos workers that have been studied formal statistical tests show that the data are described by straight lines. (Fig 11 and 12).

32 In a study of amosite factory workers, the occurrence of lung cancer appeared to be sub-linear in relation to the duration of employment (i.e. the rate of increase of risk was steeper at the lower end of the range of lengths of exposure) (Fig 13). Interpretation of this particular study is difficult for a number of reasons including the fact that information was presented only for the duration of employment and not for dust concentrations.

33 No information is available about the dose-response relationship in workers exposed exclusively or almost exclusively to crocidolite.

34 In a discussion of the relationship of the risk of lung cancer asbestos and cigarette smoking it is concluded that the data taken together show that exposure to asbestos dust probably increases the risk of lung cancer in man irrespective of smoking but much more in the presence of smoking than in its absence. The two factors (tobacco and asbestos) act synergistically but the exact relationship is uncertain and may be less than multiplicative. One consequence of this synergism is that any future diminution in the tobacco habit in persons exposed to asbestos will have a greater beneficial effect than in persons not so exposed.

MESOTHELIOMA

35 As far as mesothelioma is concerned the data so far available in man strongly suggest that the risk of this tumour increases with increasing dose. The occurrence of cases due to domestic contact with people occupationally exposed and in the neighbourhood of factories and mines suggests that relatively low doses may be followed by the development of cancer. However, information about the precise relationship between dose and response is lacking at present.

OTHER ASBESTOS RELATED DISEASE

36 As far as cancer of the gastrointestinal tract is concerned (see pp.21-22 for reservations about the nature of these cancers) information about response to dose is limited to two studies reporting small excesses of these cancers, and the relationships are on the whole irregular and weak compared with lung cancer (Figs 15 and 16). It is worth noting, however, that a significant relationship between increasing dose and increasing response in statistical terms is present for cancer of the gastrointestinal tract for maintenance workers (exposed to chrysotile and amphiboles) but not for factory workers engaged in the manufacture of asbestos textiles, building products and friction materials (exposed to chrysotile). Nothing definite can be said about the shape of the dose response curve or the existence or otherwise of a threshold.

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37 For calcified pleural plaques an irregular increase in prevalence with increasing dose is observed in figures from the single study available (Fig 8b) but in Quebec this phenomenon may be due not to asbestos but to a closely related mineral. No information is available on the dose-response relationship in respect of cancer of the larynx.

Extrapolation and the public health

38 The view taken in this section depends upon firstly a number of the conclusions reached in the previous sections (recapitulated in summary form below); secondly upon certain assumptions about extrapolation of data derived from industry to situations where large numbers of persons are exposed to very low doses; thirdly upon the evidence about the size of the population exposed and the severity and duration of the exposure outside the work place.

39 The conclusions already reached relevant to this section are as follows (1) that asbestos fibres, principally comprising chrysotile and amosite, are continuing to accumulate in a wide variety of materials in the UK while, although raw crocidolite imports have ceased, a substantial amount of this material remains; (2) that amphiboles and mixtures of chrysotile rich in amphiboles are certainly more dangerous than chrysotile alone in respect of mesothelioma and perhaps more dangerous in respect of lung cancer; (3) that where quantitative data from industrial experience are available there is generally evidence for the existence of an increasing biological response to increasing dose; within the industry we have found no convincing evidence for the existence of a threshold below which no increment of risk takes place for lung cancer or mesothelioma and where sufficient data are available (as in the case of chrysotile and lung cancer, and mixtures of chrysotile and amphiboles and lung cancer) they are consistent with the linear hypothesis. For the other fibres in relation to lung cancer and for mesothelioma in relation to all three fibre types the precise shape of the dose-response curves is unknown.

40 For gastrointestinal cancer the question of the existence of a threshold is unsettled[4]. The significance of minor clinical and radiological changes is in so much doubt that extrapolation from industrial experience of asbestosis to the general population is not possible. But despite the high proportion of individuals, especially in urban areas, with asbestos fibres in their lungs, there has been no general reporting of asbestosis in the general public. This suggest that there may be a threshold level below which asbestosis is not detectable.

41 In extrapolating from industrial data to the generally lower doses of the non-industrial situation we prefer the linear hypothesis for asbestos-related lung cancer, although it is important to emphasise that there is considerable uncertainty about the form of the relationship, and forms other than a linear relationship cannot be excluded at very low doses.

42 Our reasons for preferring a linear hypothesis are:

(1) it fits the data for occupational exposures;
(2) it is the simplest hypothesis and the one most readily used for extrapolation to the probable effects of low doses;
(3) it is likely to lead to an overestimate rather than an underestimate of risks at very low doses.

CONTAMINATION OF THE AIR, WATER AND FOOD

43 Measurements of small quantities of asbestos in air, water and food are at an early stage, subject to large errors and rarely recorded by a method or in units comparable to the industrial data on which our knowledge of the response of the human body to dose of asbestos currently depends. The uncertainties are very great.

44 With the exception of a study of contamination near dumps of waste containing asbestos, no measurements in air out of doors have shown values within several orders of magnitude of the present standard for chrysotile. In a study of air collected within buildings containing asbestos the peak figure identified was 0.08 fibres per ml (this figure is 4% of the current standard for chrysotile and 40% of the standard for crocidolite based on 8 hour exposure) (Table 33). However most of the buildings (82%) had very low readings, i.e. less than 1% of the present standard for chrysotile. It is concluded that, in view of the presence in certain buildings of asbestos products, some of which contain crocidolite or amosite which may be subject to abrasion or damage during normal use, larger samples of buildings should be studied, particularly where amphiboles have been sprayed or used in insulation board.

45 Studies of lung tissue taken in samples of persons subjected to autopsy have shown that the prevalence of embedded asbestos fibres may be as high as 50%, that the prevalence is increasing and that it varies geographically. The component of this prevalence which is occupational, or non-occupational in origin, is not clear from the published work. Many of the fibres found are extremely short.

46 There is no published information about the presence or otherwise of asbestos fibres in water in the UK. There is evidence from abroad which suggests that fibres may be leached from asbestos-containing pressure piping under certain circumstances. Fibres from asbestos deposits in other rocks and ores and from industrial contamination of water supplies have also been found. It would be advisable to study this phenomenon within the range of acidity of water encountered in the UK. Asbestos fibres associated with the use of filters have been reported in certain alcoholic beverages, soft drinks and certain foodstuffs.

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Air

47 For reasons given on page 44 it is concluded that the presence of chrysotile alone or contaminated with small quantities of amphiboles is unlikely to have produced any material increase in the risk of lung cancer in the general population or any appreciable number of cases of mesothelioma. The same is certainly true for asbestosis. Pleural changes similar to those associated with occupational exposure occur in circumstances where occupationally related exposure is unlikely. The relationship of these to environmental asbestos or other minerals is not proven. Cases of pleural thickening and calcification have been reported in those presumed to have been exposed to asbestos dust non-occupationally in the neighbourhood of mines and factories.

48 As far as the amphiboles crocidolite and amosite and mixtures rich in them are concerned, the position is less certain because of the lack of quantative data about the relationship of response to dose. Such data as exist (Tables 13X, 21 and 24) suggest that the risk of lung cancer associated with amphiboles may be greater than for chrysotile. An excess lung cancer risk cannot be completely excluded at present in those who have been exposed only to amphiboles in buildings. But the number of cases if any is probably very small (see Table 36).

49 While the majority of reported mesotheliomas are attributable to occupational and indirect occupational exposure to amphiboles, some cases have been reported where the exposure has arisen as a result of domestic contact or neighbourhood exposure. Other mesotheliomas cannot be convincingly attributed to asbestos exposure and some of these in other countries appear to have been caused by other fibrous materials. A marked feature of the asbestos-related mesotheliomas has been the geographical clustering of cases in areas where large quantities of amphiboles were handled. While the absolute number of mesotheliomas registered in comparison with other causes of death is low*, and the improvement of industrial practice in the last 20 years, and a number of other favourable points which have been mentioned in Volume 1, all give grounds for optimism, an important area of uncertainty remains.

The risk to children

50 The problem of exposure to asbestos in childhood at home, in schools and in the vicinity of dumps is reviewed on page 46. Although published instances of asbestos related disease due to such exposure attract attention they are rare. However, further surveys of school buildings containing asbestos and of dumps in the vicinity of asbestos factories, on which children might play, are recommended for two reasons. The first is that susceptibility to cancer is known to vary with age, the very young being especially at risk in relation to certain stimuli. The second is that, as children can be expected to live longer than adults, they have more chance of being affected by carcinogens with long latent periods.

Water, food and beverages

51 The scale of consumption of drinking water supplied through asbestos-containing pipes is such that even a very small increase in risk of abdominal tumours, should it be found to exist, might cause an appreciable number of cases in absolute terms. It would also be extremely difficult to detect. There is however no evidence of the existence of a risk in animals associated with the ingestion of asbestos and indeed some evidence that there is no risk. Industrial experience suggests that any risk in man may be limited to persons exposed to high doses, and the quantities of asbestos fibres in water have been found in other countries to be extremely low. It is understood, however, that further studies on the ingestion of asbestos are being carried out. We recommend also that studies of the leaching of asbestos fibres from pressure pipes in the range of acidity encountered in UK public water supplies should be undertaken. Individual items of beverage and food are less widely consumed and more easily controlled. Their study seems less important than is that of water.

Implications for public policy

52 Following the lines laid down in the terms of reference of the report each type of asbestos fibre is dealt with separately in this section. However it is important to repeat here a point made in earlier sections. This is that the conclusions reached about the effects of the different fibre types on man relate to past industrial practices, and that any change in industrial practice in the direction of the production of more finely divided fibre is likely to increase the health risk.

CROCIDOLITE

53 There is strong evidence from a number of sources that inhalation of crocidolite can cause mesothelioma, and that although the risk increases with increasing dose, it is possible for mesothelioma to occur after relatively brief exposure to this amphibole. A substantial proportion of the deaths from mesothelioma which have occurred and are occurring in Britain have probably been due to the inhalation of amiphboles including crocidolite. Although the available evidence is inconclusive it is consistent with the view that crocidolite may also have been more dangerous than chrysotile as far as the causation of lung cancer is concerned. Crocidolite is one of the amphiboles associated with the excesses of cancers of the gastro-intestinal tract (if these

*Deaths in 1975 from all cancers = 139,899 UK
from lung cancer = 37,152
from mesothelioma = 256
Sources: Annual Abstract of Statistics 1976
Mesothelioma Register

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are in reality not misdiagnosed peritoneal mesotheliomas) and may also be concerned in a small number of cases of laryngeal cancer. Evidence from the only study in man in which quantitative comparisons between crocidolite and chrysotile are possible suggests that crocidolite may cause more asbestosis than chrysotile. The animal work on this point is conflicting.

54 We recommend that contact between man and crocidolite should be limited to the minimum practicable and stringent regulations for the protection of workers and of the public should continue to be applied in respect of crocidolite-containing materials still present in buildings, vehicles, ships, dumps and other locations.

AMOSITE

55 The importance of reaching a correct judgment about the amphibole amosite is underlined by the import trends to the UK since World War II, which show a sevenfold increase in tonnage imported compared to a twofold increase in chrysotile. The tonnage of amosite imported in 1975 (19 200 tonnes) was almost three times the largest import figure for crocidolite since the war (6800 tonnes), reported in Table 1.

56 The principal difficulty about the evidence on the effects of amosite upon health is that very few definable groups of men have been exposed to amosite who have not also been exposed to crocidolite, particularly in the United Kingdom. As a cause of mesothelioma, we take the view that the risk from amosite may be intermediate between chrysotile and crocidolite. For reasons argued in detail on page 30 we consider that the majority of the cases of mesothelioma reported in insulators in North America have probably been associated with exposure to mixtures of chrysotile with amosite, not crocidolite. Eleven mesothliomas have been reported among workers in a factory making marine insulation material from amosite. There are also four well attested cases of mesothelioma in domestic contacts of amosite workers. We are inclined to ascribe the lack of an appreciable number of mesotheliomas reported amongst amosite mine workers in South Africa to the lack of follow-up of the workers.

57 The evidence about the capacity of amosite alone to produce lung cancer depends at present upon the experience of the men at a single American factory manufacturing insulation materials. This study shows excess risks of lung cancer even after very short exposures (Table 28; Fig 13) and although measurements of dust levels are not available the most likely interpretation is that the risk was higher than for men exposed to similar doses of chrysotile. Mixtures of amosite with chrysotile in American insulators are associated with very high relative risks of lung cancer, but once again unfortunately dust levels are not available.

58 Excesses of gastrointestinal cancers have occurred in heavily exposed amosite workers and in American insulation workers for whom we believe the principal amphibole inhaled has been amosite.

59 For asbestosis there is no quantitative evidence in man which compares the fibrogenicity of amosite with other types of asbestos, while in animals the evidence about its relative fibrogenicity is conflicting.

60 We conclude that, although strictly quantitative data are lacking, the burden of the evidence suggests strongly that amosite has been more dangerous than chrysotile in respect of mesothelioma and pssibly also in respect of lung cancer. If this conclusion is accepted the question must arise whether the same hygiene standard should be applied to amosite as to chrysotile.

TREMOLITE

61 Although the amphibole tremolite is not used commercially it is known to contaminate certain samples of commercial asbestos and talc. Electron microscopic studies have identified tremolite fibres in the lungs of deceased chrysotile miners. The question has been raised whether tremolite may have biological significance. Animal experiments are being conducted to determine whether tremolite is fibrogenic and/or carcinogenic. Although no human population has been identified which has been exposed industrially to tremolite alone a group of 250 American talc miners who were exposed to tremolite and serpentine have been followed over a period of 25 years. One death from peritoneal mesothelioma, one from pleural fibrosarcoma and an excess of lung cancer deaths were noted. There is sufficient evidence of a possible health risk associated with tremolite to warrant a study of its occurrence and utilisation in the UK.

CHRYSOTILE

62 Chrysotile is the only type of asbestos fibre for which data are available which permit some quantitative estimates to be made of the biological effect of a given dose of fibre. Nevertheless, for reasons already set out in detail, very considerable errors in estimation, both of dose and of the biological effect, may exist and we are dealing with best estimates from uncertain data.

63 The present hygiene standard of 2 fibres per ml was arrived at in 1968 on the basis of a study of asbestos textile workers in employment in 1966 at Rochdale. It was estimated that exposure to an average of 2 fibres per ml 8 hours a day 5 days a week for 50 years would lead to the occurrence of an early but non-specific sign of asbestosis (crepitations) in 1 % of the workers at the end of their working lives. A second study taking account of the men who had left shows that the rate of occurrence of crepitations at the current hygiene standards over 50 years had been under-estimated by a factor of about 15 (see Fig 19). Even if the significance of crepitations as an isolated sign is discounted the second study shows that an annual incidence of certified asbestosis of 0.5 % (SE 0.2 %) would occur after exposure within the present

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standard. However, if one is able to accept, as has been suggested on the basis of parallel measurements, that modern personal sampling techniques such as are used to enforce the standard at present, would have arrived at measurements of dust at Rochdale in the 1950's and 1960's 2 to 5 times those used in the calculation (see pp.34-35), the evidence in favour of certified asbestosis occurring at Rochdale after a lifetime's work at the present standard falls away. The implications of the data from these two studies on public policy with regard to the hygiene standard depend to a large extent therefore on the view taken about the relationship of the dust measurements in them to current measurements.

64 In respect of carcinogenicity we conclude that up to the present time chrysotile has rarely caused mesothelioma or cancer of the larynx. These are favourable points which should be taken into account in framing policy. On the other hand it is essential to restate the proven capacity of chrysotile to produce mesotheliomas in animals if the fibre is of the appropriate configuration, and to emphasise once again that chrysotile is probably becoming increasingly finely divided and respirable.

65 In the case of lung cancer there are two studies from North America and one from Britain which provide data which can be used to help us arrive at a standard. In the North American studies (Quebec miners[129] and Enterline's process workers[128]) the doses of dust are recorded in particles per cubic foot. It follows that if use is to be made of them a transformation must be carried out. This at best introduces additional uncertainties and at worst is indefensible (see p.37). Our approach is to show the effect of three possible conversion factors. For the Rochdale lung cancer study we show the effect of taking the dust levels as measured and of increasing them by factors of 2 and 5 to take account of a range of estimates of the combined effect of modern instruments and personal samplers (see Table 37).

66 The array of figures in Table 35 sets out the excess mortality from lung cancer which would occur to workmen exposed to various chrysotile levels over 50 years from calculations based on the various studies. The Medical Working Group recognise that the decision on what level of excess is acceptable is for others to make. A range of excesses is allowed for from 2% (i.e. that 2% more of the men exposed at the given level would die of lung cancer than if they had not been exposed and die at an earlier age) to 0.1%. As in this table the assumption of a linear relationship between dose and response is made, any other value can be found by simple proportion. The details of the various other assumptions on which the table is based are given in Appendix 6.

67 If we take as an example the column of Table 35 showing the levels at which a 1% excess of lung cancer would occur associated with chrysotile, we find a range of figures from 5 fibres per ml to 0.4 fibres per ml (for 50 years). In other words, taking the various different sets of data and using different assumptions we calculate that any value from 5 fibres per ml to 0.4 fibres per ml might lead to a yield of 1% excess deaths from lung cancer. If one is only prepared to accept an excess yield of deaths half as great (0.5%) the calculated levels are correspondingly lower.

68 The possible risk to the general public also has to be taken into account in making a decision about a hygiene standard. In Table 36, using the same studies as in Table 35, we have calculated the excess number of lung cancer deaths which would occur if a million persons were exposed continuously 8 hours a day 5 days a week for 50 years to the highest recorded concentration of asbestos in the ambient air out of doors (10 nanograms per m^3) (Table 5) or to the median, or highest levels recorded by Byrom and others in their survey of buildings (Table 33). It can be seen that, unless contaminated buildings are very much commoner than seems likely, no appreciable mortality from lung cancer can be associated with any degree of contamination by chrysotile likely to be encountered in the UK in the ambient air or in buildings not under active construction or repair. However we emphasis once more the need for further information about asbestos levels in buildings.

69 The effect of occupational exposure to asbestos on the mortality from lung cancer is therefore the main evidence on which to base a final conclusion about a hygiene standard for chrysotile. In choosing a figure it should be borne in mind that there are a number of identifiable factors in the various estimates which exert opposite effects. The linear hypothesis may over-estimate the risk but the Quebec data (because they are derived from mines and mills) and Enterline's data (because they are restricted to men who survived long enough to reach pensionable age) may under-estimate the average risk to men exposed for the whole of a working life to more finely divided fibres in process work. An assumption implicit in the use of cumulative dose is that the increase in cancer risk of added asbestos dust operates immediately. The assumption also carries with it the implication that the time the fibre remains in the lung is unimportant and that no allowance is made for continuing action of fibres in the body or of reduction of risk with their elimination. In practice it is known that there is a long interval between first exposure and the onset of excess cancer risk. Calculation of the dose of dust from past exposure is difficult, and for Rochdale it has been suggested that past dust levels have been underestimated.

70 A comparison with the health standard for ionising radiation (5 rems per annum) may be helpful. The standard is such that there is likely to be an increase in cancer mortality from 20% to 22% of all deaths if workers are exposed to this level annually for 50 years. However the great majority of workers are only exposed to much lower levels and it is estimated that the overall

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excess mortality from cancer is thus reduced to an average of 0.2%. If this is regarded as an acceptable parallel to the situation with regard to chrysotile, it will be of interest to calculate the level of chrysotile dust exposure associated with a 0.2% overall excess mortality from asbestos-related disease. If we assume that the exposure pattern for asbestos workers is roughly the same as for workers exposed to ionising radiation (that is, that the average exposure will in practice be about one-tenth of the limit set), then we should look for the exposure level associated with a 2% excess mortality among the most highly exposed. Since approximately half the mortality attributable to chrysotile is due to lung cancer and half to asbestosis (see Table 11), this suggests that we should look in the column headed 1% in Table 35, which deals with lung cancer only. The chrysotile levels in this column range from 5 fibres/ml to 0.4 fibres/ml. Bearing in mind the points made in the previous paragraphs a figure towards the lower end of this array might represent a compromise between what are, to be truthful, very considerable uncertainties.

71 In addition to the excess mortality there is also the morbidity from asbestosis to be taken into account. The difficulties of assessing this due to the variation in clinical effects and diagnosis have already been mentioned, but with this proviso, an estimate can be obtained for a given dose in fibre years/ml from Table 22.

3 Classification, distribution, and usage

72 Asbestos is the generic term for a group of naturally occurring mineral silicate fibres of the serpentine and amphibole series. One fibrous serpentine mineral (chrysotile) and five fibrous amphiboles (actinolite, amosite, anthophyllite, crocidolite and tremolite) are generally classified under the generic term. A simple accepted classification is shown in Fig 1. For practical purposes the types of fibre of commercial importance in the United Kingdom are chrysotile, crocidolite and amosite. As none of them is mined in the United Kingdom, British industry depends entirely upon imports from abroad for its supply of these substances. Table 1 shows that, in 1975 (imports of raw crocidolite to the United Kingdom ceased in 1970), chrysotile accounted for about 85% of the total imports of fibre, almost the whole of the remaining 15% being composed of amosite. The Table also shows that, since the Second World War, expansion in imports of amosite (711%) has been much greater than of chrysotile (237%), so that both in proportional and absolute terms the amount of amphibole fibre being imported to the United Kingdom now is considerably greater than it was in 1945.

73 Although the term asbestos is currently restricted to the specific silicates mentioned above, it is important to point out that there are many other natural minerals which may occur in a fibrous crystalline form. In addition to non-asbestos silicates such as clays, micas, pyroxenes and zeolites*, these include certain oxides and hydroxides, carbonates, sulphates, phosphates and borates. These substances do not possess the properties traditionally ascribed to asbestos such as resistance to heat and acids, the capacity to provide good insulation and the ability to be woven. Little is yet known about the distribution and biological effects of these fibres, but animal work suggests that where the configuration of the fibres is appropriate they ought to be considered as possible carcinogens.

74 As asbestos is virtually indestructible at ordinary temperatures and pressures it is reasonable to assume that, even allowing for the re-export of manufactured goods and the export of obsolete ships and locomotives for scrap, some substantial proportion of the cumulative totals shown remain in the United Kingdom, in buildings, pipes, dumps and elsewhere. In Fig 2(a) the cumulative imports of all types of asbestos together (principally chrysotile), and of crocidolite and amosite separately, are shown. Fig 2(b) gives more detailed information about annual and cumulative imports of crocidolite and amosite. The possible importance of tremolite as a

*Zeolite has recently been incriminated as a possible cause of mesothelioma in Turkey.

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biologically active contaminant of chrysotile and talc is mentioned in succeeding sections (para 123 and page 48).

75 Table 2 summarises the range of utilisation of asbestos fibres in the United Kingdom in 1976. For chrysotile, asbestos cement products for buildings consume the largest amount (32.8%) followed by fillers and reinforcements (22.0%), floor tiles (12.2%) and friction materials (12.1%). The production of fire resistant boards dominates the usage of amosite (84.3%) with the construction of pressure piping a poor second (7.9%). The principal uses of crocidolite have been in the insulation of buildings, ships and railway vehicles, often as a sprayed material, and in the production of pressure pipes and other asbestos cement products and in battery boxes.

76 It has been suggested that, as chrysotile and amphibole fibres may occur in association with a wide range of minerals, a range of mining and quarrying operations other than those directed at the extraction of asbestos itself may result in the contamination of the environment with these fibres[4]. An example of the use of crushed serpentine contaminated with chrysotile and tremolite on roads and in building materials has recently been reported from the United States[145]. No information has been found about any similar activities in the United Kingdom, On the basis of the data currently available our assumption is that the imported commercial varieties of asbestos form the principal source of these minerals with which man comes in contact in the United Kingdom. However, more information is required about other possible sources.

BIOPHYSICS OF ASBESTOS

77 Asbestos particles are crystals of consistently fibrous shape. Their commercial and their biological interest depend upon two main features, (1) their capacity to split longitudinally into finer fibres, and (2) their insolubility and resistance to destruction by heat, acids, etc. This means that, once embedded in animal tissues, the usual biological methods of dealing with a foreign material (e.g. solution, enzymatic breakdown, etc.) are relatively ineffective and a proportion of the mineral fibres, which varies with the type of fibre concerned (see below and Ch. 5), remains permanently within the body.

78 Timbrell has made a special study of the properties of asbestos fibres which determine their respirability and their fate when they enter the respiratory passages. Fig 3, taken from one of his articles, shows the characteristic shapes and relative sizes of fibres of crocidolite, amosite, anthophyllite and chrysotile[7,8]. The following are the principal points that have emerged from Timbrell's studies.

AERODYNAMICS OF THE FIBRES

79 The likelihood of a fibre to become airborne and to settle is determined by the reciprocal of the square of its diameter. Thus, for example, a fibre of 0.5μm in diameter is 16 times more likely to remain airborne than a fibre 2.0μm in diameter. As the number of fibres per unit of mass will also be greater in the case of the smaller fibre, it is clear that fibre diameter is the key to the number of fibres available for inhalation in a given environmental situation. For a given mass of amphiboles, therefore, both the number of fibres available for inhalation and the length of time the dust cloud remains airborne will be greater for crocidolite than for amosite, and for amosite than for anthophyllite. For chrysotile, which has a greater tendency to subdivide into fibrils, the aerodynamics of the dust will depend to a greater extent on the amount of milling (Fig 3). Depending upon its preparation the number of respirable fibres per gram may be greater or less than an identical mass of crocidolite[151].

80 Harries and Timbrell have recently pointed out that the presence of particles of other substances attached to the fibres also affects their aerodynamics. 'Clean' fibres tend to remain airborne longer than 'dirty' fibres[8a].

FATE OF FIBRES WITHIN THE AIR PASSAGES

81 In general, where mouth breathing does not occur, fibres longer than 200 μm and wider than 3 μm are impacted in the nose and ejected or transported by cilia into the pharynx and swallowed. Many of the fibres shorter than about 10 μm are incorporated in macrophages, and are rejected by the ciliary mechanism of the bronchial tree. Others are found subsequently lying free in the lung fields. The factors which determine the depth of penetration in the lung of the remaining fibres are fineness (i.e. diameter), length and possibly straightness.

82 In animal experiments carried out by Wagner and his colleagues where rats inhaled the same respirable mass of amphiboles (straight) and chrysotile (wavy) there was an increase in lung dust at autopsy almost proportional to dose for amphiboles, but hardly any chrysotile dust was found in the lungs. Eighteen months after the end of the inhalation period 26% of the amosite and crocidolite and 59% of the anthophyllite were still present in the lungs, but the amount of chrysotile was barely measurable[9] (Fig 4). Davis and his colleagues found similar differences between the retention of chrysotile and the amphiboles in the lung in their experiments[151]. The relative importance of initial failure of penetration and of ejection by cilia on the one hand, and of dissolution of asbestos in biological fluids on the other, to explain the differences in the amounts of the amphiboles and of chrysotile remaining is the subject of controversy, but the former is thought by Timbrell to be more important[7]. It is a curious and as yet unexplained paradox that, in spite of these differences, the pathological effects of chrysotile in these experiments were generally at least as severe as those of the amphiboles, and in the case of Davis's experiments UILC chrysotile, fibre for fibre, produced more fibrosis and malignant thoracic

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tumours than did crocidolite. This is discussed further in the next chapter.

83 Short chrysotile fibres which are relatively straight have been found at autopsy scattered throughout the lung fields. Bundles of longer fibres are often seen apparently impacted at bifurcations of small bronchioles[10].

84 In a series of elegant animal experiments it has been shown that, when mineral fibres of a wide range of chemical structures (e.g. chrysotile, crocidolite, glass, aluminium oxide) are applied directly to the pleura in animals, the degree of carcinogenicity is related to the proportion of the fibres of diameters between 0.5 - 2.5 μm and lengths between 10 - 80 μm (Fig 5)[11,12]. For configurations both larger and smaller than this, carcinogenicity was found to decline. Very short fibrils with diameters below 0.2 μm and lengths of 5 - 10 μm, had negligible carcinogenicity. When the fibrous configuration of the various fibres was destroyed by pulverisation, no mesotheliomas were produced, showing clearly that cancer producing activity, at least as far as mesothelioma is concerned, depends upon shape and size rather than chemical composition. In subsequent work using seven different durable non-asbestos fibrous materials comprising thirty seven different dimensional distributions, Stanton and his colleagues have drawn attention to the mesothelioma inducing capacity of extremely fine fibres with diameters from 0.01 - 0.25 μm. Fibres of lengths $>$ 8 - 64 μm and above in this diameter range were found to be highly carcinogenic and fibres of lengths $>$ 4 - 8 μm mildly so. In a further study limited to various configurations of glass, fibres less than or equal to 1.5 μm in diameter and greater than 8 μm in length yielded the highest probability of the development of a malignant tumour, but thinner (diameter $\leq$ 0.25 μm) and longer (length $>$ 8 μm) fibres were once again found to be highly carcinogenic[12a].

85 While Stanton's fibre specifications quoted above are likely to be relevant to the aetiology of pleural mesothelioma in man, there is no evidence about their applicability or otherwise to asbestos induced cancer of the lung* or to asbestosis. As will be shown in the next chapter these two diseases have been, numerically speaking, at least as important up to the present time as has mesothelioma. Nevertheless as far at any rate as mesothelioma is concerned the evidence suggests that it may be the physical configuration of the fibres rather than their chemical composition which dominate the issue of carcinogenicity.

86 It is important to point out that 0.5 μm represents the approximate limit of resolution of light microscopy in respect of fibre diameter. As Stanton has shown that carcinogencity in respect of mesothelioma extends to fibres with diameters as low as 0.01 - 0.05 μm, it is clear that current optical methods may be underestimating the number of carcinogenic fibres in the environment.

*Although carcinoma of the bronchus is the more accurate term, the more familiar expression 'lung cancer' will be used throughout this report.

4 Asbestos related disease, portals of entry and types of exposure

ASBESTOSIS

87 According to Parkes asbestosis, which was the first disease related to asbestos to be described, may be defined as "fibrosis of the lungs caused by asbestos dusts which may or may not be associated with fibrosis of the parietal or pulmonary pleura"[6]. In cases where there is no concomitant disease of the pleura the findings are usually no different from other conditions inducing diffuse interstitial fibrosis of the lungs and the diagnosis therefore generally depends upon a history of asbestos exposure and exclusion of other causes of fibrosis. Where pleural plaques or calcification accompany the evidence of fibrosis the combined pattern may be almost diagnostic.

88 The following points relevant to the subject of this paper are reported by Parkes: (1) There is a wide range of individual susceptibility, both in terms of the initiation and progress of the disease, which obscures to some extent the relationship between occurrence and dose of fibre inhaled; (2) there is usually a lag period of several years between commencement of exposure and of symptoms and signs; (3) in a proportion of patients, which is not precisely known, asbestosis progresses whether or not the patient leaves the industry; (4) the point at which definite asbestosis is diagnosed is not as clear cut as for example in the case of cancer. This is because most of the signs are non-specific, vary in degree, and are difficult to detect with certainty until well advanced. Awareness of the condition, variation between observers, and medicolegal considerations therefore all influence the point at which the diagnosis is made. These problems of definition are exemplified by the experience of a recent study where it was found that, in three of eight patients certified as having asbestosis, there was no evidence of this condition at autopsy, while, conversely, in 19 deceased workers without certified asbestosis, autopsy proved that it had contributed to death in three[13].

89 According to Harries, in a study of Plymouth dock workers the most sensitive indicator of early asbestosis was the presence of inspiratory crepitations* at the lung bases on auscultation[14]. He found that this sign sometimes occurred in persons who had not yet developed other evidence of the disease, and that its frequency was related to degree of exposure. Radiographic signs involving the lung and pleura and signs of impairment of lung function on physiological testing have also been used as indicators of asbestosis in survey work.

*Crackles audible within the chest through a stethoscope, which are sometimes called râles.

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90 In addition to workers continuously exposed to asbestos in mines and mills and in process work in factories, a variety of workers employed in areas where asbestos is used or who use asbestos as part of their work have been found to develop asbestosis. These include shipboard trades[15], pipe insulators[16], and brake repair and maintenance workers[17]. Radiological findings suggestive of asbestosis have been found in the wives and families of workers employed in a factory making insulation materials[18]. Increased death rates for asbestosis or for respiratory disease (excluding cancer) have been found in all of the principal populations of asbestos workers for whom a follow up in respect of all causes of death has so far been completed. The surveys available are summarised in Table 11.

LUNG CANCER (CARCINOMA OF THE BRONCHUS)

91 Following the classical work of Doll, subsequently confirmed by many others, it is now accepted that persons exposed in industry to asbestos dust experience an increased risk of developing lung cancer[19]. The evidence on the relationship of risk to fibre type and dose will be dealt with in subsequent sections. It has been suggested that asbestos-related lung cancers are more frequently adenocarcinomas and sited in the periphery of the lung than are lung cancers occurring in persons who have not been exposed to asbestos. At least part of the differences shown is probably due to bias associated with the fact that almost all the material in cases of asbestosis comes from autopsies, whereas surgical specimens dominate other series. The association between the occurrence of peripheral adenocarcinoma of the lung with asbestos is unsettled, and, if it exists, it is too weak to be of diagnostic value[21]. Because the tumours have no special microscopal features, and because lung cancer is common in the population at large, individual cases of asbestos related lung cancer are almost impossible to recognise with certainty and are more likely to pass unnoticed than are cases of mesothelioma.

MESOTHELIOMA OF THE PLEURA AND PERITONEUM

92 The classical paper of Wagner, Sleggs and Marchand in 1960, followed by that of Newhouse and Thompson and many others, established the relationship of mesothelioma of the pleura and peritoneum to exposure to asbestos dust[22, 23]. Although these tumours may be confused clinically with other cancers of the thorax and abdomen, their distinct pathology and other biological features, notably the lack of a relationship with tobacco, render it essential that their epidemiological pattern should be considered separately from that of lung cancer. They are invariably fatal. The fact that they occur rarely in the absence of exposure to asbestos dust makes it much easier to distinguish their relationship to it than is the case with lung cancer, which, due to its association with tobacco, is at present the commonest tumour in men in the United Kingdom.

PLEURAL THICKENING AND PLAQUES, AND BENIGN PLEURAL EFFUSION

93 Plaques of hyaline and fibrous tissue involving the parietal pleura, which sometimes calcify, have been reported in miners of anthophyllite, amosite, chrysotile and crocidolite, in workers in a variety of occupations involving asbestos, and in their wives and families [24, 17]. They have also been reported in persons living in the vicinity of asbestos mines and factories and in farmers growing tobacco in soil containing anthophyllite[24, 25, 56, 159]. Recently, the finding of pleural plaques in up to 14% of a Turkish population led to the discovery of previously unrecognised deposits of chrysotile which had been used locally in house paints for many years. They may develop following exposure to talc and mica and possibly other minerals. Benign pleural effusion (fluid developing in the space between the pleural membranes lining lung and chest wall) may occur acutely in association with other asbestos-related lung diseases or as the only or most prominent process. Shipyard workers with pleural plaques have been reported to suffer a higher risk of developing lung cancer than do shipyard workers without plaques[28]. However in a subsequent study of these workers by the same author in which patients with concomitant pulmonary fibrosis were excluded, no excess of lung cancer but an excess of mesothelioma was found[28a].

CANCER OF THE GASTROINTESTINAL TRACT

94 As the association between this group of cancers and asbestos is not universally accepted the evidence will be discussed at greater length than has been the case for the conditions mentioned above. Excesses of deaths from cancer of the gastrointestinal tract have been reported in cohort studies of nine populations within eight surveys in which the mortality of asbestos workers has been measured[124-130]. These are displayed in Table 3a. In one (Quebec) the excess is slight and is limited to one mining area. In another (Enterline's series) a significant excess occurs in only one of the two sub groups studied. In addition, two other surveys, from Rochdale and from Finland*, have reported no excesses of cases of gastrointestinal tract cancer[29, 30]. Of the nine populations showing excesses, in seven the excesses are statistically significant. There is a suggestion of a relationship between the magnitude of the relative risk from gastrointestinal tract cancer and the frequency with which peritoneal mesothelioma is reported; i.e. where the relative risk of gastrointestinal cancer is high, many mesotheliomas are reported and vice versa. This might mean that the two types of cancer both share a similar environmental cause. Alternatively if, as is frequently argued, peritoneal mesothelioma is often misclassified as carcinomatosis peritonei due to bowel cancer, the excess of cases of gastrointestinal cancer might be spurious and in reality represent cases of misdiagnosed peritoneal mesothelioma. In support of this suggestion

*As this is the only survey of anthophyllite workers it is not shown in Table 3a.

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Newhouse noted that histological data from patients dying from alleged gastrointestinal tract cancer was poor[31]. Specimens for examination could only be found in eight of her thirty-one cases and in three of these the cause of death was subsequently revised to peritoneal mesothelioma.

95 No significant excess of cases of cancer of the alimentary tract was found by Lumley in a study of cancer registrations in dockworkers and others in Plymouth[32]. However, as the age structure of the population compared was not known, a relatively insensitive method of analysis had to be employed.

96 More detailed information about the sites of the gastrointestinal cancers from the three American studies of workers manufacturing or using insulation materials is provided in Table 3b[79]. This table shows that separate excesses of deaths from cancer of the stomach and bowel have been reported in two surveys, an excess of carcinoma of the oesophagus in one, and an excess of all three considered together in the third. While only a more detailed and complete examination of autopsy material than is available can exclude the possibility that all of the excess in each of these groups is due to misdiagnosis of peritoneal mesothelioma, such errors would be more likely to be classified under a less specific heading, such as carcinomatosis pertonei. However, it is not clear in the papers summarised in Table 3b under which heading deaths ascribed to this cause have been classified. If they have been classified under "all other cancers" and not grouped with the specified cancers of the stomach and large bowel, the case for real excesses of primary gastrointestinal tract cancers associated with asbestos would be much stronger.

97 In summary, a significant excess of deaths ascribed to cancer of the gastrointestinal tract has been reported from a number of surveys of the mortality of asbestos workers. The clinical and pathological data available about the illnesses from which these men and women suffered is at present sketchy and there remains a possibility at least that they represent undisclosed cases of peritoneal mesothelioma. However, it must be said that the excesses are large to be explained away in this manner, and a real increase in gastrointestinal cancer, possible associated with the use of amphiboles or mixtures rich in them, seems more probable.

CANCER OF THE LARYNX

98 In a carefully executed study, Stell and McGill found that thirty-one of a hundred consecutive patients with cancer of the larynx at Liverpool, as compared with three matched controls, gave a history of exposure to asbestos. The chief types of exposure were unloading asbestos in the docks (9) and in the insulation trades (19)[33]. As might be expected, fewer of the control patients smoked than did the patients with laryngeal cancer, but there were insufficient data to enable a study of possible synergism between the effect of smoking and of exposure to asbestos. A survey of forty-three patients and matched controls designed in a similar way, from Toronto, also gave similar results with a marked and statistically highly significant association between exposure to asbestos and laryngeal cancer. No association was found between laryngeal cancer and exposure to alcohol, X-rays, uranium, chromium, nickel, cobalt or arsenic[34]. Newhouse and Berry, and Selikoff have also reported excesses of cases of laryngeal cancer in asbestos workers[35, 36]. On the other hand McDonald and his colleagues have found no excess mortality from laryngeal cancer in Quebec chrysotile miners and millers[77]. The data are summarised in Table 3c. The wide range of types of exposure to asbestos, in which an excess risk of laryngeal cancer has been found, makes it difficult to incriminate a co-factor, and the weight of the published evidence suggests a causal association at least between the inhalation of mixtures of asbestos containing amphiboles and laryngeal cancer. In absolute terms the risk is much smaller than has existed in relation to any of the other conditions mentioned above, with the exception of cancer of the oral cavity and pharynx.

CANCER OF OTHER ORGANS

99 A single reference has been found to a possible increased risk of cancer of the oral cavity and pharynx in insulators[36]. In a study of 17 800 asbestos insulation workers 17 deaths from these causes were found; 7.41 were expected. This finding requires amplication and confirmation. In her follow-up studies of women exposed to mixtures of chrysotile and amphiboles at the Barking factory, Newhouse has reported small excesses of deaths from carcinoma of the ovary and breast[152]. However, in another survey in which patients with ovarian cancer were compared with controls in respect of various factors, no excess of patients with exposure to asbestos was found.

THE LAG PERIOD BETWEEN FIRST EXPOSURE TO ASBESTOS AND THE DEVELOPMENT OF ASBESTOS RELATED DISEASE

100 All four asbestos related diseases mentioned above have in common the existence of a delay of 'lag-period' between first exposure and the onset of the disease. In the case of asbestosis the interval is extremely variable. In some cases impairment may be detectable within months of first exposure, while in others a relatively short heavy exposure may be followed by an interval of several decades before the first symptom develops. In the case of lung cancer, cases attributable to asbestos begin to appear after ten years, and cases have continued to occur twenty years after first exposure. For mesothelioma the interval varies from ten to sixty years. These delays introduce the following problems:
(1) cases of asbestos related disease may occur many years after the worker has left the relevant job; (2) cases of disease occurring at present relate to conditions of work which are likely to have changed in the intervening period; (3) current trends in occurrence of asbestos related disease can give little indication of recent

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improvements or otherwise (say within the last decade) in working conditions; (4) a period of at least 30 years may have to elapse before it is possible to give a definite answer concerning the introduction or control of an asbestos-related hazard; (5) in persons currently developing disease, data about the relevant dust levels is likely to be poor; (6) there is a serious risk among the uninformed of attributing present disease frequencies to present conditions of work.

THE RELATIVE IMPORTANCE OF THE TYPES OF ASBESTOS RELATED DISEASE

101 By summing the experience of the various industrial populations which have been followed (Table 11) it is possible to compare the importance in respect of mortality of the different types of asbestos related disease mentioned above*. Of the total excess mortality among deaths where the cause was known, approximately 40% has been accounted for by lung cancer (whether or not associated with asbestos), 24% by asbestosis, 13% by mesothelioma, and 8% by gastrointestinal cancer. Included in this excess mortality for lung cancer is a substantial element due to the synergistic action of tobacco with asbestos which cannot therefore properly be attributed to the effects of asbestos alone (see also pp. 39-40). These studies therefore represent the relative ranking of these diseases in terms of mortality as associated with past industrial conditions and cigarette smoking patterns. In addition a substantial proportion of the excess mortality is associated with all other causes of death (15%). This may be due to the selection of inappropriate standard populations for comparison and misclassification of some deaths due to the conditions named above among the remaining causes, or, less likely, to a non-specific deleterious occupational effect. The point is taken up again in Appendix 3.

102 A study of British death certificates suggests a different picture, namely that asbestos-related mesothelioma is a more frequent cause of death than asbestos-related cancer (Table 20)[21]. However, death certificates are in this case almost certainly an unreliable indicator. For example, the mention of asbestos on death certificates of people dying from lung cancer who were smokers and who were also exposed to asbestos is very likely to be deficient.

Portals of entry of asbestos to the body

103 Quantitatively speaking, by far the most important portal of entry of asbestos fibres to the body is through the respiratory passages. The physical characteristics of asbestos fibres which favour penetration of dust to the bronchi, lungs and pleura, have already been described.

*It is important to point out that, if the measure of relative 'importance' took account of years of active life deprived, the order of ranking might differ from the above.

104 Asbestos fibres may also enter the body by ingestion or by parenteral injection. Ingestion may be secondary (i.e. by swallowing nasal and bronchial secretions containing inhaled asbestos fibres) and it is assumed that it is this type of ingestion which accounts for the putative excesses of gastronintestinal cancers in asbestos workers referred to above. Primary ingestion occurs by the consumption of contaminated water, food or beverages. The only estimate of the amount of inhaled material which reaches the alimentary tract comes from an experiment in rats using radioactive crocidolite. Evans and his colleagues found that approximately half of the fibre deposited in the respiratory tissues reached the alimentary tract within one hour[37]. In a study of the problem, the American Water Works Association has estimated that the amount secondarily ingested from contaminated nasal and bronchial secretions over a lifetime of occupational exposure might amount to about 2.4 grams as opposed to 0.07 grams by means of the ingestion of 2 litres of water a day over sixty years[38].

105 Accidental parenteral administration of asbestos may have taken place in connection with the use of asbestos containing syringes or from the use of fluids containing drugs and medicaments filtered through asbestos pads during manufacture. The effects of this, if any, are not known[39].

Types of exposure

106 A useful classification of types of exposure to asbestos has been published in the Zielhuis report and is reproduced here, modified and extended as Table 4[1]. Although the different classes of exposure are not distinct and clear cut but tend to merge into one another, the classification provides a useful overall view of the problem.

107 Direct occupational exposure (Ia) consists of all exposures due to the direct handling of commercial asbestos containing materials in the workplace. It should be noted that this category includes not only jobs where the handling of asbestos is the principal activity, but also those where this is a subsidiary activity which would not be evident in a simple description of the job. It includes the following principal categories of exposure: Mining and milling of asbestos which is not at present carried out in the United Kingdom: Transporting fibre in docks and by road and rail; the packaging of fibre in bags, palletised where possible, and in containers, has done much to reduce exposure to dust under this heading: Processing of raw fibre in the primary manufacture of textiles, insulation materials, asbestos cement and asbestos-plastic products: Installing, adapting and otherwise working on new asbestos products; these include asbestos insulation board; asbestos-plastic products such as brake and clutch linings, gaskets and moulded products; asbestos cement products such as roofing sheets, rainwater goods and pressure pipes; and

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asbestos textiles such as gloves, curtains and cords: Repairing and servicing existing asbestos products; these include not only the materials mentioned above but materials including insulation, sprays, etc. in buildings, locomotives and ships which were installed by methods now outdated and may contain crocidolite: Demolition of buildings and vehicles containing asbestos products; these include power stations, public buildings containing old fashioned central heating plants, factories and furnaces and vehicles: Dumping waste material containing asbestos.

108 Indirect occupational exposure (Ib) includes all persons working in the vicinity of men handling asbestos (e.g. office workers in a textile factory). Agricultural workers (Ic) may be exposed to asbestos by disturbing soils containing asbestos fibres, but there is no evidence that such soils exist in the United Kingdom.

109 Clinically significant exposure to asbestos has occurred to the wives and families of asbestos workers in the home (IIa). Leisure-time activities such as do-it-yourself construction work and other hobbies also constitute a possible cause of exposure (IIb). Exposure to asbestos has taken place to persons other than workers in the neighbourhood of certain asbestos mines and factories (III), and dumps of waste.

110 Due to their essential indestructibility and widespread use asbestos fibres of commercial origin are found in the general atmosphere, and in buildings and water supplies. Their presence in certain beverages and foods is generally due to the use of asbestos containing filters (IV). Asbestos fibres present as natural contaminents of other rocks and ores, or fibres from minerals other than asbestos may contaminate air or water[4, 103, 145]. Tables 5 and 6 show some available data on concentrations of asbestos in air inside and outside buildings and in water[110-111, 4]. It should be noted that the techniques on which these measurements are based are still at an early stage of development and the measurements are subject to substantial errors.

5 The relationship of medical effects to fibre type

111 The evidence about the relationship of asbestos related disease to type of fibre comes from two main sources, experimental work on animals, principally in the rat, and epidemiological surveys and case reports in man. Recently important additional information has become available from the electron microscopal analysis of asbestos in tissues of patients with asbestosis and in mesothelioma patients and controls.

Experimental work in animals

112 Asbestosis, cancers of the bronchial tree of a variety of histological types, and mesothelioma of the pleura have been produced in rats following the inhalation of each of the four principal types of asbestos – amosite, anthophyllite, crocidolite and chrysotile. Data suitable for quantitative comparisons of the relationship of the occurrence of malignant tumours by fibre type are shown in Table 7[9]. They show that all four types of fibre produce both malignant tumours of the lung and mesothelioma, the only exception to the rule being chrysotile from one location (Rhodesia) which did not produce mesotheliomas. In another experiment in rats, dealt with in more detail below, similar masses of the various fibres produced significantly more malignant thoracic tumours in animals exposed to chrysotile (8/40) than in those exposed to amosite (0/43) or crocidolite (0/42)[151]. Apart from amosite, which produced less, all the samples of asbestos fibre tested by Wagner and his colleagues produced similar amounts of asbestosis in the animals, and the degree of fibrosis continued to increase after exposure ceased. The similarity of the pathological responses to the different types of fibre is particularly interesting in view of the finding from the same material and other similar experiments already referred to in a previous section (see Fig 4) that far more of the dust of the amphibole fibres was found in the lungs of the animals than of the chrysotile fibres eighteen months after inhalation had ended[9, 151]. According to Timbrell* the respirable fraction of the UICC standard chrysotile fibres may be greater than for most chrysotiles used in industry, and in this important respect these UICC samples approximate to commercial amphiboles. Reeves' method of using dust clouds produced by hammer-milling may have produced conditions more closely comparable to industrial practice. He found that in rats inhalation of chrysotile and amosite was followed in each case by an incidence of 5% of malignant pulmonary tumours while crocidolite produced a yield of 14%. Although the atmospheric concentrations were the same for each type of asbestos in this experiment the

*V. Timbrell: Personal communication to the authors.

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numbers of optically visible fibres varied considerably, being respectively 54 million/m^3, 864 million/m^3 and 1105 million/m^3 for chrysotile, amosite and crocidolite[40].

113 As is shown, for example, in Reeves' study quoted above, similar respirable masses of asbestos may contain widely differing numbers of optically visible fibres. If it is the number of fibres rather than the mass of material reaching the target organ which is critical in determining the pathological reaction, it is clearly relevant to attempt experiments in which comparisons between chrysotile and the amphiboles are made holding the number of fibres in the aerosol constant. Davis, Beckett, Bolton, Collings and Middleton have shown that when aerosols containing approximately 400 fibres/ml were administered to rats, chrysotile produced substantially more interstitial fibrosis of the lungs than did crocidolite[151]. However, bearing in mind the small number of animals involved, there was no difference in the proportions of malignant thoracic tumours produced by chrysotile (3/42) and crocidolite (1/43). The results of this important study are summarised in Table 7X. Davis and his colleagues conclude that the relative pathogenicity of the different samples of fibres may depend upon the proportion of fibres $> 20\mu$ in length: the differences in the diameter of the fibres in the various samples were relatively slight.

114 In Table 8 the relationship between duration of inhalation of fibre and the incidence of tumours in Wagner's material is shown. The animals exposed to the two samples of chrysotile have been combined, as have those exposed to the various amphiboles. A striking feature is the occurrence of 5 malignant tumours after one day's exposure. UICC standard chrysotile produces more tumours than amphiboles at all durations of exposure[9].

115 Administration of asbestos directly into the tissue of animals is obviously much further removed from experience in man than are the experiments described above, as it eliminates all the factors which determine whether or not the fibres can penetrate under natural conditions to the target organ. However a standard technique has been developed for intrapleural inoculation which, provided this important limitation is borne in mind, makes it possible to compare the capacity of different fibres to produce mesothelioma (see also section of biophysics of asbestos paras 77-86). The figures from a recent paper by Wagner and his colleagues are shown in Table 9. They show that, of the standard UICC samples, there is a gradation in the mesothelioma producing capacity by fibre type in the following sequence: crocidolite, amosite, anthophyllite and chrysotile. However a superfine sample of Canadian chrysotile, fractionated from commercial Grade 7 chrysotile by water sedimentation, with extremely fine straight fibres, was at least as carcinogenic as the crocidolite[41]. Other work in rats and in hamsters is consistent with the view that the configuration of the fibres, in particular the relationship of fibre diameter to length, is decisive in determining the incidence of mesotheliomas in pleural inoculation experiments[12, 42, 43]. This relationship, and in particular the potential hazards associated with the intrapleural inoculation of fine (>2.5 μm) long (10 μm-80 μm) fibres, have already been discussed (para 84). The importance of these very high aspect ratios links in an interesting way with Davis's evidence from inhalation experiments quoted above. Intraperitoneal injection of 2 mg of chrysotile and crocidolite produced respectively 6/37 and 15/39 mesotheliomas in rats[44*]. Intraperitoneal injection of a variety of non-asbestoform fibres produced mesotheliomas but injections of non-fibrous granules did not.

116 The conclusions from animal work are that all of the four main types of asbestos fibre have the capacity to cause pulmonary fibrosis, lung cancer and mesothelioma when inhaled, if the physical configuration of the fibres in the dust cloud are appropriate. There are no consistent differences in the incidence in relation to fibre type in inhalation experiments, but it is clear that under certain conditions chrysotile may produce at least as many tumours as the amphiboles. For reasons not at present understood, much more of the amphibole fibres are retained in the lung than of the chrysotile. When applying these results to the situation in man three points should be borne in mind. The first is that the doses to which the animals have been exposed (measured in hundreds or thousands of fibres per ml) are very much greater than those to which industrial workers are likely to have been exposed in recent years. The second is that finely dispersed chrysotile was unusual in the industrial conditions which produced the tumours occurring in man today. The third is that there is evidence that more finely dispersed fibres are currently being produced and utilised in certain parts of industry (see footnote on p. 34).

117 When asbestos fibres are introduced into the pleura and peritoneum all types of asbestos fibre produce mesotheliomas as do a wide range of other fibres as well. The results suggest that once again fibre configuration is the decisive factor. Thus, although crocidolite produces more tumours than UICC chrysotile, when superfine chrysotile is used it is as carcinogenic as crocidolite. The finding that a wide range of fibrous materials may induce mesotheliomas when introduced into the pleura or peritoneum in animals is highly relevant to the question of the manufacture and use of substitutes for asbestos.

Epidemiological surveys and case reports from man

118 Many asbestos products contain mixtures of more than one type of fibre (Table 10)[1]. When one bears

*The difference in mesothelioma rates, 16% compared to 38%, is statistically significant ($P \simeq 0.05$).

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this point in mind together with the fact that industrial exposure over a lifetime may be relevant to the occurrence of asbestos related disease, and that industrial processes change and are often poorly documented, it is not surprising that populations in which it is reasonably certain that there has been exclusive exposure to one fibre are rare. In essence, the populations available for study are limited to crocidolite miners, amosite miners, chrysotile miners and millers, anthophyllite miners, and a handful of groups involved in specialised manufacturing processes.

ASBESTOSIS

119 Asbestosis, as defined in this paper, has been reported in persons occupationally exposed to each of the main fibre types. Thus it has been reported in chrysotile miners and millers[45], and in workers in a textile producing factory employing principally chrysotile*[13].

120 It has also been reported in crocidolite and amosite miners and in anthophyllite miners[46, 47]. However, we have not been able to find any quantitative data enabling comparisons to be made between the incidence, prevalence or mortality rates of asbestosis by fibre type. Nor can ratios of observed to expected cases be assembled for comparison because the expectation of dying from asbestosis in the general population is zero. The numbers of deaths from asbestosis expressed as a proportion of the total mortality in groups of workers exposed to chrysotile, to amosite, and to mixtures of fibres are summarised in Table 11, but as these populations are almost certainly not comparable in terms of age, dustiness of job and other factors, any comparisons must be interpreted with caution[29, 30, 55, 124-134]. Excluding Enterline's data on pensioners as a special case (see also para 138) there appears to be a discernable gradient with the mortality of chrysotile miners at the lower end and amosite and anthophyllite workers in the intermediate position. Those exposed to mixtures have the highest proportional mortality, but the various populations may not be comparable in terms of the quantity of dust inhaled.

121 Weill and his colleagues have compared the prevalence of various abnormalities of lung function in two groups of men in the asbestos cement industry exposed to similar total quantities of asbestos dust, one of which had been subjected to dust containing considerably more crocidolite (by a factor of 16) and slightly more silica (by a factor of 1.5) than the other[48]. The crocidolite group performed less well in respect of all 14 lung function parameters and the differences were significant in respect of seven parameters. Selikoff has reported positive X-ray findings consistent with asbestosis in members of a group of former insulation plant employees exposed to amosite, some for periods of a few weeks only[49].

122 Asymptomatic radiological changes suggestive of asbestosis have been found in 114 (35%) of a sample of 326 household contacts of workers in the same factory, exposure having taken place from asbestos fibres taken home in the clothing of the workers concerned. Half of these contacts had been exposed to asbestos from the clothes of an asbestos worker for less than one year. A summary of the radiological changes is shown in Table 12[18]. While the illustrations of the relatively small number of cases of advanced disease in these papers are impressive, the significance of minor radiological changes when reported, as is the case here, in the absence of other parameters and of controls, should, as Becklake has pointed out, be accepted with caution[2].

123 The possibility that the amphibole, tremolite, may play a significant role in the pathogenesis of asbestosis has been suggested by Pooley[50]. In an electronmicroscopic analysis of lung tissue derived from post mortem material from twenty previous workers in the Canadian chrysotile industry, in most of whom asbestosis had been diagnosed in life, he found tremolite fibres in eleven, often in large quantities. In the absence of controls, of occupational histories, and of data about the distribution of tremolite as a contaminent in the Quebec mines, it is impossible to interpret this finding. However it raises the question of the possible importance of the contamination of chrysotile with tremolite in the aetiology of asbestosis and points to an urgent need for further study of the biological significance of this substance.

Lung cancer and fibre type

124 The evidence concerning the relationship of asbestos-induced lung cancer to type of fibre is scanty. It comes, principally, from two studies of miners and from an industrial population which is thought to have been exposed exclusively to amosite. Most other populations studied so far have been exposed to mixtures. Table 11 shows for each study the ratio, expressed as a relative risk, of the deaths observed from lung cancer to the deaths expected on the basis of the experience of the local population. For Canadian chrysotile miners the excess risk is 1.2:1, for Finnish anthophyllite miners,

*There appears to be uncertainty and possibly a conflict of evidence about the extent and period of usage of crocidolite at Rochdale which the present authors have been unable to resolve. The first BOHS study of 290 men assumed that exposure had been exclusively or almost exclusively to chrysolite as is borne out by title of the paper. Similar assumptions are made by Peto[61] and by Berry [13]. However, in their evidence to the ACA, Turner and Newall state that 'crocidolite was encountered up to 1969 in the cheese winding and plaiting sections' and 'before 1960 the significance of the difference between chrysotile and crocidolite was not recognised and no special records were kept of the type of fibre to which they were exposed'. A recent communication from Mr H D S Hardie of Turner and Newall states that between 1931 and 1970 not less than 2500 tonnes of crocidolite were used at Rochdale, i.e. an average of about 60 tonnes per annum.

According to a personal communication from Dr J C Gilson, however, about one third of the cards were seen to be employing crocidolite as late as 1963.

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1.7:1. In contrast, for a population of workers employed in the manufacture of insulation materials, thought to be exposed exclusively to amosite, the ratio is 6.3:1. It is unfortunate that no epidemiological study of amosite or crocidolite miners has been done and there is no information available on the relative risk of lung cancer in these groups of workers. Oettlé compared the mortality from various types of cancer experienced by the residents of the district in which crocidolite mining takes place in the Cape Province of South Africa with the mortality in a 'control' district. An excess of lung cancer but not of other cancers was found in both sexes[51].

125 As far as industrial workers exposed to crocidolite only are concerned, no information is yet available about the risk of lung cancer in the British gas mask makers, to whom reference will be made again in the section on mesothelioma. Although the Canadian group of crocidolite gas mask makers is very small and its age structure is unknown (see Table 13X), in terms of proportional mortality it exhibits a higher percentage of deaths from lung cancer (13%) than has been experienced by the Quebec chrysotile miners (6%). This difference does not reach the conventional level of significance ($\chi^2=3.53$, 1 d.f., $P > 0.05$). However, when one bears in mind that about half the gas mask workers were women and therefore probably smoked less than the miners who were almost all men, and their exposure to asbestos was generally over a much shorter period than the miners, it seems possible that crocidolite may prove to have been more dangerous than chrysotile in respect of lung cancer.

126 A study of the other industrial populations for which estimates of the relative risk of lung cancer based on an external standard are available (Table 11) shows that all exhibit relative risks higher than those in chrysotile and anthophyllite miners, the group with what is perhaps the lowest proportional exposure to crocidolite (the Rochdale survey) having the lowest relative risk 2.1:1. The groups of factory workers with mixed exposure to chrysotile, crocidolite and amosite (Enterline's and Newhouse's studies) have relative risks of 2.7:1 and 2.6:1, while the studies of American insulators, who are exposed to mixtures rich in amosite, yield very much higher relative risks; namely 7.3:1 and 4.9:1. It is important to point out, however, that the validity of comparison is limited by other important differences between the studies. Thus Enterline's choice of survivors to pensionable age probably reduces the relative risk in comparison with other studies as does Newhouse's inclusion of men exposed for less than two years. The exclusion of men exposed for less than 10 years from the Rochdale survey and for less than 20 years from one of the studies of American insulators has the opposite effect, inflating the relative risk in comparison with the other studies. It is possible also that in the United States studies based on lists of trades union members obtain more complete information about the fact of death than do studies based on follow-up through the social security system. Unknown differences between the smoking habits of the various groups of men and of the amount of dust inhaled may also exert an influence.

127 Enterline and Henderson have classified the men in their study on the basis of the type of fibre to which they were exposed and have also corrected for differences in the cumulative dust inhaled (Table 21)[135]. These internal comparisons within their study are free of the biases between studies mentioned above. Unfortunately some of the numbers are small and no group exposed to crocidolite alone could be found. Nevertheless it is clear that within this study men exposed to chrysotile only (relative risk 2.4:1) have done better than men exposed to crocidolite in any combination (4.4:1)*. The numbers exposed to amosite are small but suggest an intermediate risk for exposure to this type of fibre. In another study Enterline, Decouflé and Henderson showed that at any given dust level maintenance workers (exposed to amphiboles and chrysotile) had higher risks of lung cancer than production workers (exposed to chrysotile)[128] (see Figs 10 and 11).

Mesothelioma

128 Although isolated case reports had occurred earlier, the paper which demonstrated a relationship between exposure to asbestos in man and mesothelioma was that of Wagner, Sleggs and Marchand[22]. This paper reported thirty-three cases of what was previously thought to be an extremely rare tumour of which thirteen cases occurred in Cape crocidolite miners and millers and fourteen in persons living in the neighbourhood of these mines. All but two of the remaining cases had been exposed to crocidolite, but exposure to other types of asbestos fibre as well could not be excluded. Subsequent to this study cases of asbestos associated mesothelioma of the pleura and peritoneum have been reported from a large number of countries. The most widely accepted view at present is that crocidolite, amosite and chrysotile (but not anthophyllite) cause mesothelioma in man, but that there is a gradient in risk, crocidolite being the most and chrysotile the least dangerous of the three common fibre types in this respect[1, 3]. On the other hand the latest review carried out for the National Institute for Occupational Safety and Health does not distinguish between mesothelioma risks by fibre type, and the latest IARC Monograph on Asbestos is not explicit on this point[52, 4].

129 The evidence concerning the alleged gradient in risk of mesothelioma in man in relation to fibre type comes from the following sources:

(1) the experience of miners and millers concerned with the excavation of asbestos of a single fibre type;

*The standard errors of the relative risks are approximately 0.25 and 0.62 and the difference between the relative risks is statistically significant ($P < 0.01$).

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(2) the experience of process workers said to be exposed to a single fibre type;
(3) the experience of workers exposed to mixes of asbestos relatively rich in amosite and crocidolite;
(4) the pattern of neighbourhood and domestic cases of mesothelioma in relation to fibre type;
(5) the general epidemiological pattern of mesothelioma.

These five points will now be considered in detail.

Miners and millers

130 Within the Republic of South Africa, crocidolite, amosite and chrysotile have all been mined for many years. The crocidolite is of two types, one with extremely fine fibres of aerodynamic diameter 0.09-0.8 μm being mined in the northern part of Cape Province (Cape Blue) while the other with less fine fibres of diameter 0.2-2.5 μm (Transvaal Blue) is mined in the Transvaal[7, 8]. Amosite mines are found close to the Transvaal crocidolite mines and chrysotile mines are found to the south east near the Swaziland border. According to Webster, of 88 cases of mesothelioma reported in miners up to 1973, 83 were men who worked with Cape Blue, 4 with amosite, 1 with Transvaal Blue[53]. None was reported in relation to the mining of chrysotile.

131 In considering the significance of these data the following points have to be borne in mind:

(1) many of the miners were migratory workers;
(2) the area under study extends over thousands of square miles with variable standards of medical care available to mineworkers and ex-mineworkers, and variable degrees of diagnostic awareness of mesothelioma;
(3) no formal follow-up study of any of the populations has been attempted and no computations of incidence are possible from the data available.

132 According to Harington, Gilson and Wagner[54] a thorough attempt was made to find cases of mesothelioma in the Transvaal hospitals in the mining areas in 1959 and 1969. If, as Webster suggests, the population at risk is proportional to the weight of fibre mined the paucity of cases associated with amosite is truly remarkable as up to 1951 more amosite had been mined than crocidolite (Table 13). The lack of cases associated with Transvaal Blue may at least in part be due to the fact that practically none of this fibre was mined before 1944. There is no published account of any special attempt to search for mesothelioma in relation to the South African chrysotile mines, which are near the border and from which the sick may migrate to Swaziland. According to Gilson, a thorough search, though unpublished, has been made for mesothelioma associated with chrysotile mining in Rhodesia without success[3]. While the evidence from South Africa is suggestive of a gradient of risk of mesothelioma by fibre type it cannot on its own be regarded as conclusive in view of the absence of formal epidemiological studies. Fortunately, however, support for a relatively low risk of mesothelioma associated with chrysotile mining can be found in other countries, particularly Canada where massive mining and milling operations have been carried out for many years and an exhaustive search for mesothelioma has been made. According to McDonald, a study of deaths in 11379 chrysotile miners and millers in Canada between 1935 and 1973 has revealed only eleven mesotheliomas, of which two occurred in persons who had worked for a short time with crocidolite gas mask filters[55]. No mesotheliomas were found in a study of the cancer risk of chrysotile miners and millers in the Urals, and only one case has so far been detected in incompletely recorded data from Italy and Cyprus[56]. However, it would be prudent not to give too much weight to the last three studies mentioned.

133 Crocidolite was mined at Wittenoom in Western Australia from 1937 until 1966. In spite of the fact that the labour force is known to have been highly itinerant, 46% being immigrants from European countries other than the British Isles, twenty cases of definite and four of suspected mesothelioma have been identified among a total workforce of 6577 men. As only a quarter of the employees have so far been traced and many are believed to have returned to their countries of origin, the number of cases so far reported underestimates the true position. No peritoneal cases have so far been identified[155].

134 Meurman and his colleagues found no cases of mesothelioma in a careful follow-up study of 1092 miners in the Finnish anthophyllite industry who had worked for at least three months in the mines between 1936 and 1967. As it is possible that only a few of these men were first exposed to anthophyllite before the War, a longer period of follow up is desirable to be certain that no risk of mesothelioma has been incurred[30, 57]. Eighty-five cases of mesothelioma have been recognised in Finland since 1953[154]. The geographical distribution of these patients bears no relationship to the location of the anthophyllite mines.

Industrial workers exposed to single fibre types

135 A study of the population of about 1600 workers, principally women, who assembled gas mask filters using West Australian crocidolite during the Second World War has shown that thirty (1.8%) of them have died of mesothelioma in Nottingham[58]. This figure underestimates the true situation because no attempt has yet been made to trace the histories of workers who have died elsewhere. A similar cohort of 199 persons involved in the manufacture of crocidolite containing gas masks in Canada during the period 1939-42 has been identified. Of these, nine (4.5%) have died of mesothelioma so far,

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of whom two had pleural, three pleural and peritoneal, and four peritoneal tumours[55]. In Table 13X the proportional mortality from malignant disease, including mesothelioma, of the Canadian gas mask workers is contrasted with that of Canadian chrysotile miners. An important aspect of this Table is that both populations worked in the same Province (Quebec) and were subject to similar diagnostic practices and follow-up procedures, thus eliminating two important sources of bias. Although, as the authors point out, it was not possible to adjust the results for differences in age, the excess mortality from mesothelioma is so striking in the gas mask workers (80 fold) that it can be accepted with confidence. It should also be borne in mind that none of the gas mask workers was exposed for more than three years while many of the miners were exposed for much longer periods.

136 Selikoff and Hammond have published the mortality experience of a group of workers who are thought to have been exposed only to amosite. Of 933 men employed in a factory between 1941 and 1945, 881 were traced to death or to the end of 1973. Eleven of the men (2.1 %) are known to have died of mesothelioma[127]. It is generally agreed that these men who worked principally on wartime contracts were exposed to extremely dusty conditions.

137 Industrial cohorts exposed only to chrysotile are rare and possibly non existent. For reasons already mentioned in a footnote on p.26 the extent to which the workforce at Rochdale were exposed to crocidolite is far from clear. Accordingly the question of exposure to specific fibre types of the thirty cases of mesothelioma recorded by Turner and Newall must await electro–microscopic lung tissue analysis[5]. Peto, in a study in progress of 20000 workers at Rochdale who entered after 1933, reports ten cases of mesothelioma – all pleural[61]. Information about the fibre profile of the lung tissue of these men compared with that of controls would also be helpful. If the Rochdale cases of mesothelioma do not turn out to have amphibole fibres in their lung tissues the question will arise whether the specification of chrysotile fibres used in the manufacture of textiles is relevant.

138 Enterline and Henderson studied a cohort of asbestos production and maintenance workers who had survived long enough to retire at 65[135]. In this selected group they found it possible to classify the workers according to type of fibre to which they were exposed, but only one of the men is known to have developed a mesothelioma after retirement, and this exposure history is unknown. The authors mention that they are aware of a large number of mesothelioma deaths in younger men from the same population who were excluded from the study. Clinical details of seventy two of these have been published elsewhere*. According to Borow "chrysotile is the main fibre used although in some of the processes crocidolite is also present", but he gives no data on the histories of exposure of the individual cases[62]. The material from this industrial population, therefore, does not help us assess the risk of mesothelioma associated with exposure to chrysotile*.

139 Based on his experience at Rochdale where no cases of peritoneal mesothelioma have been reported, Peto suggests that crocidolite is responsible for peritoneal mesotheliomas, the risk for pleural mesothelioma being similar for chrysotile and crocidolite[61]. This hypothesis does not fit in with the evidence that peritoneal mesothelioma is so far unknown in West Australian crocidolite miners, and has been reported only once in Nottingham gas mask makers. While admittedly the relative frequency of pleural and peritoneal mesotheliomas in South African crocidolite miners may have been influenced by the practice of limiting autopsy examination in miners to the contents of the thorax, the discovery of hardly any peritoneal cases almost 20 years after the original description of the disease is evidence against Peto's hypothesis. Exposure to mixtures of amosite and chrysotile without crocidolite in American insulators, and manufactures of amosite insulation board, was followed by the occurrence of many cases of peritoneal mesothelioma (Table 11). Diagnostic awareness is probably the factor which contributes most to thereporting of peritoneal mesothelioma.

Exposure to mixtures of fibres

140 Mesotheliomas have been described in men and women exposed to mixtures of chrysotile and amphiboles in shipyards, in the insulation industry and in factories. According to Newhouse and Berry this tumour will eventually account for from 7 to 11 % of all deaths in the group of Barking asbestos factory workers who were exposed to mixtures of chrysotile, amosite and crocidolite[63].

141 Pooley has subjected lung tissue from 120 cases of mesothelioma collected from various countries to electron microscopic analysis by fibre type and has compared the material with that derived from 135 autopsies carried out in persons dying of various other conditions[64]. The material aggregated according to amount of asbestos fibre found and types of fibre is shown in Tables 14 and 15. Table 14 shows a clear relationship between the occurrence of mesothelioma and quantity of asbestos fibre found in the lungs. But a more striking finding is seen in Table 15 which analyses the effect of fibre type on relative risk. Taken at its face

*In a recent letter from Dr M Borow we have been informed that he has now encountered over 120 cases of mesothelioma from this factory.

*Some additional information giving indirect support for the role of crocidolite in the aetiology of these tumours has subsequently become available. The factory opened in 1917, but crocidolite was not used until 1929, since when it has been in continuous use by men making pipes. Almost all the cases have been in men and have occurred since 1960. In the large work force of women employed making textiles from chrysotile only one case has been found[70].

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value it suggests that the presence of amphibole fibres is associated with twice the relative risk of mesothelioma (12) associated with the presence of chrysotile (6). However, the presence of mixtures containing both amphiboles and chrysotile is associated with a relative risk of 61. If the presence of fibres in the lung fields accurately reflects exposure (but see Fig 4) there may be an almost multiplicative relationship between the risks of exposure to mixtures of amphiboles and chrysotile and the risks of exposure to either alone. Clearly there may be alternative explanations of the findings in this Table, and more information is necessary about the occupational histories of the patients and the method of selection of the material. However the magnitude of the relative tisk of mesothelioma in persons carrying mixed burdens of chrysotile and amphiboles in their lungs is so great that it merits further investigation.

142 Both in absolute terms and when expressed as a proportion of the total number of deaths, the largest numbers of cases of mesotheliomas reported in asbestos workers have been found in American and Canadian insulators (see Table 11). It is therefore important to determine if possible to which fibres these men have been exposed and to what extent their experience differs from British insulators who we can be certain were exposed to crocidolite. According to Selikoff's account of the work done by these men, all of whom were exclusively thermal insulators, 'until 1930 chrysotile was almost the only asbestos used, amosite making its appearance later and crocidolite practically not at all'[60]. He bases this view on the following evidence: (1) According to the US Department of Commerce imports of amphiboles from South Africa were low between 1930-39 (a total of 17599 tons). No information is available about imports before 1930, including the period of the first World War. (2) During the period 1920-40, 267000 tons of chrysotile were used in insulation. (3) Crocidolite was never used in insulation materials by the largest producer in the United States. According to Cooper and Miedema 'in the 1930's both chrysotile and amosite were used (for insulation purposes); however amosite became more widely used until by 1950 it was predominant'. After 1960 a trend set in to employ more chrysotile and less amosite[138]. According to Marr, an analysis of insulation materials used in past decades on the west coast of the United States revealed amosite and chrysotile but no crocidolite[140]. In Malloy's textbook *Thermal Insulation* 20 materials containing asbestos are mentioned but in only 3 is amosite specified and in one crocidolite[139]. However this book was published in 1969 and gives no information about past practice.

143 Much increased quantities of amosite and crocidolite were imported to the United States during World War II relative to the 1930's. These fibres were regarded as vital to the war effort and their apportionment was the subject of an agreement between the governments of the UK and USA[142]. In a contemporary account amosite is stated as being preferred for making lightweight insulation around steam machinery and pipes for warships, and a block insulation product containing amosite is also mentioned. Crocidolite is mentioned as superior to amosite for making asbestos cement pressure pipes, chemical filters, acid-resistant packings and gas masks. There is no note of its use in insulation.

144 Amosite and Transvaal crocidolite are mined in the same areas and by the same company. The question may therefore be asked whether an important degree of contamination of amosite with crocidolite took place when both were being mined in large quantities in the early years of the war. However, as the deposits of the two minerals are apparently in proximity in only a few localities this seems unlikely unless deliberate substitution took place at source[143]. Although the evidence is indirect and meagre it suggests that the exposure of US insulation workers to crocidolite, certainly before the War and probably since, has been much smaller than in the UK. On the other hand there seems little doubt about exposure to amosite on a considerable scale before and after the War.

145 On the basis of the assumption that insulators employed in shipyard work in the US will have had more contact with amosite than those employed throughout in the construction industry, Selikoff made comparisons between the two groups[79]. No differences in mortality from mesothelioma were found but some of the groups used in the comparison were small. This may be interpreted either by deducing that chrysotile and amosite used under these two sets of conditions, although in different proportions, were equally dangerous, or that there were no biologically important differences in the amount of amosite to which the two groups of workers have been exposed.

146 On the existing evidence it does not seem possible to attribute the majority of the mesotheliomas in American insulators to crocidolite. Of the three possible explanations, viz: that they are due to exposure to a mixture with a biologically significant proportion of amosite, or to the presence of a cofactor acting in conjunction with asbestos, or to chrysotile alone, we prefer the first, because of the evidence of the occurrence of mesothelioma in men exposed for relatively short periods to the manufacture of amosite containing insulation materials and their domestic contacts[127,118].

Neighbourhood and domestic exposure

147 Neighbourhood cases have been reported in South Africa from the area in which Cape Blue crocidolite is mined, but with the possible exception of one case, have not been reported from the neighbourhood of mines producing other types of fibre. Webster classifies 33 of 232 cases of South African pleural

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mesothelioma as of environmental origin due to living or visiting in the Cape Blue mining area or railhead[65]. One additional case excluded from the main series because of an incomplete occupational history lived near to the Transvaal Blue crocidolite area for a short time. The possibility that there may be biases introduced in relation to the likelihood of diagnosis of mesothelioma in various parts of South Africa has already been mentioned. In contrast, in extensive studies of mesothelioma in Canada, no indication of an association between residence within twenty miles of a chrysotile mine and mesothelioma (only one case and three controls had lived within 20 miles of the mines) was found[66]. Bearing in mind the relative scale of mining in the two countries this is remarkable*. Newhouse and Thompson found eleven patients with mesothelioma and five controls who had lived within half a mile of an asbestos textile factory using a mixture of chrysotile, crocidolite and amosite[23]. Neighbourhood cases of mesothelioma have also been reported from the vicinity of an asbestos factory in Hamburg and near shipyards[67]. In these areas mixtures of chrysotile and amphiboles were used.

148 The first cases of mesothelioma (and indeed any cancer) known to have occurred as a result of exposure to a carcinogen brought to the home by an industrial worker were nine female relatives of asbestos workers described by Newhouse and Thompson[23]. Subsequently similar cases have been reported from a number of countries. In a recent review Anderson collected thirty-seven cases from nine countries and added four more from the 326 traced family contacts of 1664 asbestos workers who had been exposed to amosite[18]. Recently two cases have been reported in relatives of workers in the West Australian crocidolite mine[155]. Table 16 shows that in the majority of cases where data are available, exposure to amphiboles or to mixtures of amphiboles and chrysotile had taken place, and in the insulation industry is once again prominent. However, it has been mentioned that three Canadian patients who had been associated with miners and millers at home had apparently been exposed only to chrysotile, although precise information on this point is not available. It has already been pointed out that, if cases of lung cancer associated with domestic contact to asbestos were to occur, they would be difficult to detect due to the high incidence of this tumour in the general population and the lack of specific diagnostic features.

The general epidemiological pattern of mesothelioma

149 McDonald and Becklake have studied the epidemiology of mesothelioma in Canada and the United States and made some comparisons with figures in Western Europe[66]. Table 17a shows that, for the years 1966-72, the annual incidence of mesothelioma in the United States was 1.2 per million, a figure similar to Canada, excluding Quebec. For Quebec the rate was 2.5 per million, but this was associated with a low rate of acceptance of the diagnoses made in that Province by the panel of referees. When these rates were based on accepted cases the difference between Quebec and the remainder disappeared.

150 In an important study of the geography of mesothelioma within the Province of Quebec during the years 1969-72 Thériault and Gilbert found two cases (1.3 expected) in the chrysotile mining area, forty-two cases (24.4 expected) in Montreal, and a deficiency of cases in the remainder of the Province[67]. The massive chrysotile mining operations were thus not found to be associated with any significant excess of cases of this tumour (Table 17b). This is of particular interest as it is known that visible deposits of dust from dumps and tailings in and around dwellings occurred in at least one of the principal mining towns within living memory[68].

151 Using the mesothelioma incidence rate for Canada as a standard, McDonald and Becklake have calculated rates for countries and cities for which data are available[66]. For countries, the rates show that the incidence is not dissimilar to Canada, but for the cities the incidence is much higher. The remarkable feature of Table 18 is that it shows that almost all the published series of mesotheliomas come from seaports with shipyards. As, apart from Dresden and Manville, New Jersey, large industrial centres with heavy industry other than seaports do not figure in the Table, it seems difficult to attribute this to a bias due to heightened awareness of the risks of asbestos*. It suggests, rather, that there is a special risk associated with work in shipyards, perhaps due to exposure to amphiboles, to work in excessively dusty conditions, or to a co-factor.

152 Greenberg and Lloyd Davies in their study of the geography of 412 registered cases of mesothelioma, demonstrated striking concentrations of cases in areas with naval shipyards (Tyneside, Merseyside, Clydeside and Plymouth) and less striking concentrations in other urban centres with heavy industry[69]. The naval shipyard concentrations may be related to the fact that crocidolite had been specified as a major constituent of marine insulating material for the Royal Navy for a period of at least forty years up to 1965.

153 A number of case control studies have provided information about relative risks of mesothelioma in various occupations. The highest relative risks are generally in insulation workers, workers with furnaces, boilers and pipes, asbestos factory process workers, and shipyard workers. Unfortunately, because of lack of standardisation of definitions and techniques, it is not

*Since the end of World War II the production of chrysolite in Canada has amounted to between 5 and 10 times the production of asbestos (all fibre types) in South Africa.

*An extended version of this Table made available to the authors by J C McDonald since this section was written does not alter the conclusion of a special risk associated with shipyards.

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possible to aggregate the data. Table 19 shows the numbers of cases and controls and relative risks in McDonald and McDonald's extensive survey of the United States and Canada[70]. The high relative risk of insulation workers stands out in contrast to all other classes of workers, even those involved in process work in factories.

154 Although it is not possible to relate them to fibre type, two other features of the epidemiological pattern of mesothelioma may conveniently be considered here: the secular trend and the problem of mesothelioma not known to be associated with asbestos.

THE SECULAR TREND

155 Judging by the numbers of deaths certified as due to mesothelioma, the mortality from this condition has doubled over the decade 1967-76, there being a suggestion of an upward turn in gradient in the last two years, (Table 20). An upward trend has been noted in Holland but there appears to be no consistent upward trend in North America[1·66]. Increasing awareness of the condition probably contributes at least part of the increase, but bearing in mind the long lag period between first exposure and death, the trend of consumption of asbestos demonstrated in Table 1 and Fig 2 and the poor conditions of work existing in many areas in the past, it would be surprising of the number of cases and deaths from this condition did not continue to increase. Newhouse and Berry predict that the peak in the number of mesothelioma deaths in the ex-workers of the Barking factory will occur in the 1980's[63].

156 There are at present too many imponderables to permit any certainty in the prediction of the future trend of incidence of mesothelioma for the population as a whole. If crocidolite is the only fibre significantly involved, the number of cases may follow the shape of annual imports of crocidolite displaced by forty years (shown in Fig 2b). This would indicate a peak in the 1980's and an end about 2010. However this takes no account of further exposures occurring from worked crocidolite, which would defer these dates. The number of deaths in 1970 was 193 (Table 20) Annual imports in the 1950's were three times those in the 1930's. The number of annual deaths in the 1990's on these assumptions might be about three times those of 1970, i.e. about 580 deaths per annum.

157 If amosite is also important the multiplying factor would be similar for 1990, but due to continuing increases after 1950 of imports of amosite, 1990 would not represent the peak for mortality from mesothelioma, and by 2000 the annual number of deaths might have almost doubled from 1990. Against this must be weighed the beneficial effect of the improvements in working conditions and changes in practice, particularly in relation to the use of amphiboles in insulation and textile production. Other important points bearing upon the future trend in mesothelioma include present and future practice in milling chrysotile (pp. 25, 33) and the shape of the dose response curve (page 40).

158 The difficulties inherent in making a complete register of mesothelioma cases are illustrated by a survey of the number of cases with a definite diagnosis made in Southampton during the period 1968-72. Of twenty-two cases, twelve are known to the mesothelioma register, but as six of the remaining ten occurred in 1975 and 1976 it is possible that they would have been the subject of late notification. It is our impression that mesothelioma continues to be confused with other malignant tumours, particularly when it affects the peritoneum, and that the net effect is an underestimate of the number of cases occurring.

MESOTHELIOMA UNRELATED TO ASBESTOS*

159 The proportion of cases of mesothelioma in published series where exposure to asbestos is considered unlikely or impossible varies and depends, amongst other factors, upon the care with which the evidence of exposure is sought for. All observers agree, however, that in some cases no direct or indirect evidence of exposure to asbestos can be elicited by questioning the patient or his family. Using the Mount Sinai questionnaire, McDonald found exposure to asbestos unlikely in about 25% of the male patients in the USA and Holland and 50% of those in Canada. In women, however, exposure to asbestos was unlikely in more than 90% of the cases in all three countries[55]. It has been suggested that the cases of mesothelioma in which there is no history of exposure may represent a response to a general environmental hazard which the patients concerned have incurred without their knowledge, for example in buildings or vehicles containing asbestos. Alternative suggestions are that other carcinogens cause mesothelioma or that our methods to detect occupational exposure are insensitive. The work in animals, which shows that a wide range of fibrous particles other than asbestos may produce mesothelioma if instilled intrapleurally in animals, has already been mentioned on page 20. In considering this problem in man it would be helpful to have a study of the mineral and fibre content of the lung tissue of mesothelioma patients in whom the history of occupational exposure is negative, and in controls.

160 The other side of the question, namely that mesothelioma may incorrectly be attributed to exposure to asbestos, must also be considered. Thus in studies where incidence of exposure to asbestos is sought in mesothelioma cases and controls, a proportion of the controls is almost invariably found to have worked with asbestos. Thus in a study in Tyneside 41% of the matched control patients had probable or definite occupational exposure to asbestos, while in Scotland the proportion was 14%[71, 72]. This indicates the ubiquitous distribution

*A recent report suggests that malignant mesotheliomas occurring in rural Turkey may be due to inhalation of tuf rock containing fibrils of zeolite material.[167]

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of asbestos and makes it likely that a proportion of the cited associations between these tumours and this fibre may be coincidental and not cause and effect. To take the argument a stage further, even when there is a well attested history of exposure, cause and effect cannot be assumed with absolute certainty in an individual case.

Cancer of the gastrointestinal tract

161 In paras 94-97 the evidence for the existence of a definite relationship between gastrointestinal cancer and asbestos was discussed and it was decided that there was at present insufficient evidence to be certain which precise sites, if any, other than peritoneal mesothelioma, were involved. Keeping this reservation in mind it can be seen from Table 3a that there is little, if any, evidence for the existence of an excess risk of "abdominal cancer" in relation to exposure to chrysotile mining or milling or to anthophyllite mining.

162 Table 3a shows that the high rates of abdominal cancer reported occur in insulation workers exposed to mixtures of amosite and chrysotile, factory workers exposed to chrysotile, amosite and crocidolite, and shipyard and maintenance workers also exposed to all three types of fibre. Whatever the exact nature of these cancers may be, there is a suggestion that they are related to amosite or crocidolite or to mixtures of these with chrysotile rather than to chrysotile alone. The Rochdale experience, in which there has been no excess of gastrointestinal cancer, fits in with this view if exposure to crocidolite by the workers followed by Peto has been minimal or non-existent, and the mesotheliomas reported by him are due to chrysotile fibres of the configuration used in the textile industry. On the other hand, if the Rochdale mesotheliomas are due to crocidolite, we are left with no explanation, on the basis of the hypothesis set out above, for the absence of an excess of abdominal cancers there.

Cancer of the larynx

163 There is no clear evidence which relates the excess risk of cancer of the larynx in asbestos workers to fibre type. All the exposures listed in Table 3c were probably to mixtures of fibres. Insulation workers are once again prominent. Chrysotile miners experienced no excess risk[77].

Conclusion

164 Experimental work shows that lung cancer, asbestosis and mesothelioma may be produced in animals following inhalation of the UICC standard samples of the four commonly available commercial types of asbestos, and that some samples of chrysotile are at least as pathogenic as the amphiboles. In one study small numbers of tumours of both types arose after exposure as short as one day. Other work related exclusively to mesothelioma suggests that the pathogenesis of this type of tumour depends on the configuration of the fibre rather than its chemical constitution and that if an asbestos fibre fulfils the appropriate specification of length and diameter it will produce mesotheliomas after intrapleural instillation in experimental animals regardless of fibre type. It has been pointed out that the specifications of the fibres used in these experiments are not necessarily representative of the range of fibres to which man has been exposed up to the present time.

165 Evidence in man about the relationship of the incidence of lung cancer to fibre type is scanty. It is consistent with the view that exposure to the amphiboles, crocidolite and amosite (or mixtures containing them) has been more dangerous than exposure to chrysotile alone or anthophyllite alone. However, as alternative explanations for the findings are possible and important gaps in our knowledge of this aspect of the field remain, the weight given to this conclusion must be qualified by uncertainty. The high relative risks of lung cancer reported in a single study of workers making insulation material containing amosite, even after very short exposure, is worrying in view of the increase in utilisation of this material in the United Kingdom since the war, but it is generally acknowledged that these men worked under extremely dusty conditions. The evidence from amosite miners in South Africa does not help us on this point as there is no data about lung cancer in this group.

166 Evidence about asbestosis in man in relation to fibre type is limited to one study which suggests that crocidolite may have been more fibrogenic than chrysotile. Information from the same amosite insulation manufacturers as were mentioned in connection with lung cancer, and their families, suggests that the fibre used in that factory may have been peculiarly fibrogenic.

167 As far as mesothelioma is concerned, epidemiological evidence from miners, from process workers, and from the distribution of neighbourhood and domestic cases, when combined presents a powerful case from a number of different points of view that crocidolite has been more dangerous than chrysotile and anthophyllite. However none of the fibres except perhaps anthophyllite can be exonerated. The position of amosite is less clear in respect of mesothelioma and may be intermediate. There is a possibility which needs further study that mixtures of chrysotile and amphiboles may be more dangerous in respect of the production of mesothelioma than either alone.

168 In view of the fact that it seems clear from animal work that all fibre types have the potential to produce the three main types of asbestos related disease to a similar extent if the physical conditions of the dust cloud are appropriate, it follows that any change in industrial practice in the direction of the production of more finely dispersed fibre may be significant to health. This work suggests that, should commercial chrysotile be milled in such a way that it approaches the respirability of crocidolite and amosite, particularly if long (10-80μ)

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and extremely thin (< 2.5μ) fibres are produced, the morbidity gradient in favour of chrysotile seen up to the present and discussed in this chapter may disappear in the future*. In considering the possible future significance to health of such a trend it is necessary to balance the very much greater consumption of chrysotile than of the amphiboles (Table 1, Fig 2) against the considerable improvements which have undoubtedly been secured in dust control in many areas in recent years. Changes in the range of usage of asbestos and any effects these would have on the accessibility of the fibre to human contact would also have to be taken into account.

*According to information received by the authors from the Canadian Asbestos Mining Association, which is concerned exclusively with chrysotile, there has been a trend since World War II to produce fibre for most purposes (other than asbestos cement) which has a greater surface area per gram than previously (i.e. the fibres have been 'opened' to a greater extent and tend to have a smaller mean diameter). On the other hand the trend for fibres to be used in the manufacture of asbestos cement has been in the opposite direction, namely to produce fibre with a greater proportion of 'crudie', i.e. unopened bundles of fibres. We are informed that individual companies have data on these extremely important points.

6 Dose response relationships

169 This section discusses the quantitative evidence about the relationship between the dose of asbestos inhaled by members of a population and the frequency of three principal biological responses – asbestosis, lung cancer and mesothelioma. No data have been found which permit any quantitative comment to be made about the relationship between dose and response for cancer of the larynx. That related to cancer of the alimentary tract is limited to two studies (see page 41). The importance of establishing, if possible, the nature of the relationship between dose and biological response is that it may assist in framing policy about an acceptable level of exposure. For example if it could be shown that a level of exposure exists below which no risk of sickness or death is incurred (a so-called threshold) at least such a level would be likely to be acceptable from the medical point of view.

170 Some of the possible relationships between dose and response in connection with the effects of asbestos are illustrated in Fig 6. They are (a) a threshold relationship whereby below a certain limiting value of the dose there is no associated excess sickness or mortality; (b) a (recti) linear relationship whereby the response increases proportionally to the dose; (c) a quadratic relationship whereby the response increases proportionally to the square of the dose; (d) a sublinear relationship where the response increases proportionally to a power of the dose, the power being less than unity; (e) a cumulative normal relationship whereby the response increases as if the distribution of susceptibility levels to asbestos was normally distributed.

171 In order to contrast these responses and to underline their significance a hypothetical example is illustrated in Fig 6. A cumulative dose in fibre-years per cc and a response is indicated on the coordinates of each of the five graphs using identical scales. If we assume that the response is the mortality from (say) lung cancer then, in the hypothetical example, a dose of 50 fibre-years per cc would be associated respectively with the following risks relative to that of an unexposed population of similar age: (a) 1, (b) 3, (c) 1.5, (d) 4, (e) 2.

THE MEASUREMENT OF DOSE

172 Ideally the measurement of the dose that is required is the dose of fibre which reaches the target organ (e.g. bronchus, lung tissue or pleura) and remains there long enough to produce a deleterious biological effect. Some of the facts and uncertainties about the way in which the various types of fibre may be ejected, retained and possibly dissolved or transported elsewhere within the body, have been dealt with in a previous

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section. In man, however, the only information available for studies of the relationship of response to dose consists of estimates of quantity of fibre in the work place available to be inhaled. But methods of measuring dust have differed from time to time and from place to place, and the reconciliation of measurements arrived at by different methods is far from simple. A detailed account of the problem by Steel is published separately[161]. The following is a summary of the main sources of uncertainty:

173 Factors which may vary are (a) the sampling instrument used, (b) the location of the sampling instrument relative to the workman, (c) the nature of the dust counted (all particles or fibres only), (d) the evaluation technique.

174 The standard sampling instrument most commonly used in Britain today is the membrane filter sampler. But before 1964 the commonest instrument was the thermal precipitator while in the USA and Canada many of the data are derived from midget impingers. Membrane filters are used to count fibres while midget impingers can only be used to count total particles, and thermal precipitators can be used to count fibres only very inaccurately. There are considerable uncertainties in converting particle counts (particularly from the impinger) to fibre counts as correlations are poor. Before 1970 membrane filter sampling was typically static, with the instrument in a fixed position in the workplace. Since 1970, personal sampling has become more common. Studies have shown that personal sampling increases the number of fibres counted in a given workplace by a factor which varies widely both within and between manufacturing processes, but which at dust levels around 2 fibres/ml is probably between 1 and 2 (Table 37).

175 The results of the fibre counts are also affected by the method of counting used. The current method of 'eyepiece graticule' counting has been introduced since 1969. When compared with the older 'whole field' counting technique, graticule counting increases fibre counts by a factor which has been estimated as being of the order of 2-2.5 (Table 37).

176 The combined effect of these last two factors means that an atmospheric chrysotile concentration which would have been evaluated as 2 fibres/ml in 1968 might well today be evaluated as somewhere between 4 and 10 fibres/ml. To put it another way, a concentration measured today as 2 fibres/ml might have been measured as between 0.4 and 1 fibre/ml in 1968. In other words these changes in sampling and evaluation techniques have brought about a *de facto* tightening of the hygiene standard of 2 to 5 fold since 1968. Clearly careful account must be taken of this phenomenon when the pathological effects in man of dust levels recorded in the past are interpreted to help frame future policy about a hygiene standard.

177 Two other aspects of the problem of the measurement of dose require to be mentioned. These are: (1) that, unlike biologically active agents which are metabolised swiftly, some proportion of the asbestos fibres taken into the body remain and may be active for prolonged (although unknown) periods; (2) the first manifestation of the response (disease) may appear after exposure has ceased. Account of these points can be taken by weighting the dose by the estimated residence time within the body, making various assumptions about the rate of decay of activity of the fibres in the body, and by making allowances for cases of disease or deaths occurring after workers have left the industry[13]. However, in the succeeding sections of this report, the measurement of dose used will be the cumulative dose as measured by the authors concerned on the basis of the best estimates available to them and expressed either in million particle years per cu. ft. or fibre years per cc*. This technique assumes that the pathological effect of the fibres starts immediately after inhalation and decays instantaneously thereafter.

MEASUREMENT OF RESPONSE

178 In the case of cancer, the measurement of response used throughout the published material is death. The deaths which have occurred over a period of time in a group of men exposed to a given dose of dust are then compared with one of two standards; an internal standard within the population of workers, for example with men involved in a different process in the factory thought to have experienced a lower or higher dose; or an external standard based on the experience of persons of similar age and sex outside the workplace under study who are assumed not to have been exposed. The use of an external standard of comparison is important to assess the working group as a whole against a predominantly non-exposed population and in particular to identify any excess mortality associated with low exposure. Although the approach outlined above is the best available for the purpose, it has many pitfalls, and biases may be introduced which become of particular significance when one tries to reconcile the findings of different studies. A recent review has emphasised the influence of differences in the apparent risk of disease associated with asbestos due to the source of the data (for example, studies commissioned by trades unions consistently show higher relative risks than those commissioned by industry) and length of follow-up[73], trades union based studies usually have the advantage of virtually complete follow up in terms of death (because the payment of death benefits to relatives requires the presentation of a death certificate) but may suffer from uncertainties about the definition of the appropriate population base. Other types of study often have to depend upon less effective meansof determining the fact of death, but may be based on more representative samples of the total workforce. Because of the extended period of latency of asbestos related disease, periods of measurement of risk since first exposure of less than thirty years have given misleading results.

*i.e. 20 fibre years per cc. = 20 fibres per cc. over 1 year, or 1 fibre per cc. over 20 years.

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179 In the case of asbestosis the onset of the disease is gradual and the measurement of the response to a dose of asbestos is complicated by problems of definition and diagnosis which occur in cancer only to a very much lesser extent. Various signs of impairment of the lungs (e.g. crepitations, radiological changes, or impairment of physiological tests of lung function) may appear in advance of a confident diagnosis of asbestosis agreed by a panel of experts, or indeed in advance of symptoms noticeable to the patient. The specificity of these signs as a response to asbestos, and their reversibility or otherwise if exposure ceases, therefore becomes a paramount part of the interpretation of the data.

Asbestosis

180 In 1968 the British Occupational Hygiene Society published the results of a prevalence survey of clinical and radiological chest signs in 290 men who were employed in 1966 at a textile factory using asbestos at Rochdale and had worked there for at least 10 years since 1933[74]. It was decided to accept that the earliest demonstrable effect of asbestos on the lungs was the development of râles (also known as crepitations) at the lung bases which "are considered as the key symptom since every subject exhibiting X-ray changes also exhibited basal râles, whereas some subjects exhibited basal râles without showing X-ray changes'. Sixteen of the men were found to have râles and on the basis of the relationship of the prevalence of râles to estimates of the cumulative dose of the fibre inhaled it was calculated that a cumulative dose of 100 fibre years per cc (e.g. 2 fibres per cc. over a working life of 50 years) would result in 1% of men contracting asbestosis over their working lifetime. As was pointed out in the statistical appendix to the paper this result might be biased by the design of the study which only took account of the manifestations of asbestosis as they occurred in the men still working on 30th June, 1966, and took no account of the men who had worked for 10 years or more but had dropped out of the workforce between 1933 and 1966.

181 A second survey of men employed in the same factory, as yet unpublished but which has been made available, corrects this deficiency and supplies important new data[13]. In the new survey, 285 of the 290 men in the original survey and 107 men who completed ten years' service within the period 30th June, 1966 and 31st December, 1972, have been followed up to the latter date including men who had left the factory after 1966. This makes it possible to study the first occurrence of signs of asbestosis (i.e. the incidence of the signs) according to cumulative dose. In order of increasing severity the measures of incidence of 'asbestosis' by year of first occurrence used were: (1) basal crepitations alone; (2) a combination of findings labelled 'possible asbestosis' on the basis of the findings of the factory doctor reviewed by an independent expert; this combination of findings, although insufficient to satisfy the Pneumoconiosis Medical Panel, generally leads in the factory concerned to a recommendation for the worker to be transferred to a less dusty job at a guaranteed basic wage with an ex gratia payment; (3) certification of disablement due to asbestosis by the Pneumoconiosis Medical Panel. In the presence of an adequate history certification requires two of the following: basal râles, finger clubbing and certain radiological appearances, and alterations in lung function plus evidence of disablement. A variety of radiological and physiological measurements is also available. Another important feature of the second study was that the occupational histories of the men were reviewed by an independent hygienist and a number of the previous estimates of dose revised downwards.

182 The relationship of the occurrence of crepitations, possible asbestosis and certified asbestosis to dose is shown in Table 22 separately for the men first employed before 1951 and after 1950 and combined; and the combined data are plotted in Fig 7*.

183 It can be seen that, even using the strictest and least controversial measure of asbestosis, namely the number of cases certified by the Pneumoconoisis Medical Panel, nine new cases occurred (average annual incidence of 0.5%) in persons with a cumulative dose less than 100 fibre-years per cc. Using more liberal criteria of the occurrence of asbestosis, i.e. by including the category of 'possible asbestosis' a detectable rate of incidence of new cases is found to occur at exposures of less than 50 fibre-years per cc. (average annual incidence 0.5%) while if crepitations are considered the average annual incidence rate for men with a cumulative exposure of less than 50 fibre-years per cc. becomes 1.3%.

184 Crepitations are not a specific finding in asbestosis. However, in a careful study of a population of dockworkers, the prevalance of crepitations was found to be related to degree of exposure, the findings being relatively infrequent in those minimally exposed. Of a sample of fifty-six naval dockyard workers found to have crepitations in 1966, thirty-four (60.7%) had a radiographic abnormality and nine (16.1%) were certified as having asbestosis by 1975 without further exposure to asbestos[75].

185 Fig 7 shows the various responses to dose graphically. Although they all appear irregular, statistical testing suggests that a linear relationship describes the material adequately. For crepitations, extrapolation of a straight line fitted to the data would clearly not pass through the origin, but this is what would be expected since crepitations occur in the general population[14]. Table 22 and Fig 7 show that the inclusion in the second study from Rochdale of the experience of some of the men who had left the industry has radically altered the conclusions which may be drawn from the

*Table 22 and Figure 7 are based on data supplied to the authors by Mr G Berry, which is shown as Appendix 3.

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material*. Under the working conditions which have existed at Rochdale since 1933 cumulative exposures of less than 100 fibre-years per cc. have produced an annual rate of new cases of certified asbestosis of 0.5% (SE 0.2%). Using more sensitive measures of the occurrence of the disease the annual rate of occurrence of 'possible asbestosis' is 1.0% (SE 0.3%) and of crepitations is 2.0% (SE 0.4%) at cumulative exposures of less than 100 fibre years per cc. Thus, annual occurrence rates of crepitations in men who have been exposed to a cumulative dose within the current standard for a working life of 50 years are twice as high as the previously estimated overall life risk. Restricting consideration to the men first employed after 1950 (Table 22 and Appendix 3), in whom measurements both of dose and of response may have been more accurate, does not alter these conclusions significantly, the annual estimates for certified and possible asbestosis becoming respectively 0.4% (SE 0.2%) and 0.7% (SE 0.3%). For crepitations the figures become 1.6% (SE 0.4%). Thus most of the information about the lower doses is dervied from the more accurate data.

186 Two other studies, one from the United States and one from Canada, have related the prevalence of asbestosis to dose of dust. As the authors point out, neither takes account of the sickness experience of men who have left the industry. Judging by British experience both are therefore likely substantially to underestimate the fibrogenic effect of dust. Weill and his colleagues measured the frequency of physiological and radiological changes in a population of asbestos cement workers exposed to a mixture of chrysotile, amosite and crocidolite[48]. They conclude that evidence of diffuse pulmonary fibrosis does not occur below a threshold of 200 fibre years per cc., but that at higher levels of exposure there is an increasing frequency of changes in lung function and of radiographic abnormalities. However, they go on to point out that a study which follows up a group of the population will be necessary to add certainty to their conclusions. The apparent conflict between this finding and that of the second Rochdale study may be due to the lack of information about men who have left the industry in the former, or to differing conditions of work between the textile and cement producing industries or to both factors.

187 The relationship of the frequency of radiological and physiological lung changes to cumulative dose of dust has also been studied in Canadian chrysotile miners[2, 66]. In a study of chest radiographs of employees aged 56-65 a relationship between the frequency of parenchymal abnormalities and dose was found but the association was weak (no correlation coefficient exceeded +0.3) suggesting that other factors were important*. There was no evidence of a threshold below which radiological changes did not occur, the shape of the response was irregular, and differences were observed between the two main mining areas. The relationship between the prevalence of radiological evidence of parenchymal opacities and of pleural thickening and cumulative dose in the two mining areas are shown in Fig 8(a) and (b) respectively.

188 In a study of the prevalence of dyspnoea (shortness of breath) and of impairment of lung function in 1,015 current mine and mill workers, Becklake reports a relationship of all the parameters studied with increasing dose[2]. The data are reproduced in summary form in Table 23a. As Fig 9 shows there is clear evidence of impairment associated with cumulative doses of less than 100 million particle years per cu. ft., and the shape of the curves suggests that increments in dose at the lower end of the range carry a higher increment of impairment than do increments of dose at higher levels. The dose response curve is sublinear (see Fig 5d). Study of smokers and non-smokers from this population separately shows impairment in respect of these parameters in each group at the lower end of the range of dose and a sublinear response[66].

189 The only available mortality data relating to asbestosis which can be related to dose (and which also comes from Quebec) shows a strong relationship between dose and relative risk of death (Table 23b)[77]. Any attempt to relate the Canadian experience to the work at Rochdale is perilous. Gibbs and Lachance have shown that, within the Quebec miners and millers, there is a wide variation in the proportion of the dust which consists of fibres at different sites, so that conversion of the scale of dose from million particle years per cubic foot to fibre years per cc. is likely to be subject to considerable error. The ratios of membrane filter counts (fibres with an aspect ratio greater than 3 to 1) per ml to midget impinger counts (mppcf) in various areas ranged from 1.7 to 21.9[76]. If a rounded median conversion factor of 5 is used we find that most of the increment in respiratory impairment takes place between cumulative doses of 50 and 500 fibre years per cc., i.e. perhaps within the range of dose studied at Rochdale. For what it is worth a conversion factor of 5 corresponds closely to that (6) found for fibres $> 5\ \mu m$ in length in a comparison of the two methods based on a study of dust in four American asbestos textile plants[112]. It should be pointed out that the Canadian study referred to above takes no account of persons who have left the industry and therefore almost certainly underestimates the degree of impairment in the exposed population as a whole. The Canadian evidence is nevertheless consistent with the view that significant functional and radiological impairment

*The magnitude of the selective effect is indicated by the fact that 29 of the 34 men certified as having asbestosis had left or died during the period 1966 to 1972.

*A further study of the relationship of the progression of radiological changes over 20 years in 267 Quebec workers has recently been reported[121]. Once again the correlations between changes and estimated dose of asbestos are weak. As the study appears to exclude men who did not remain or survive to have 5 X-rays over 20 years it seems likely to underestimate the fibrogenic effect of the duct.

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takes place at the lower end of the range of exposure which has occurred within the Quebec chrysotile mining industry.

190 No quantitative data exist concerning the relationship of dose to the frequency of asbestosis for crocidolite or amosite.

191 This section may be summarised by the following statement. Of the studies available only one (the second Rochdale study) is designed in a way which gives a relatively unbiased assessment of the rate of impairment of lung function in a population exposed to asbestos and relates it to dose. Using conservative measures of impairment (i.e. the occurrence of 'possible' asbestosis) this study shows that a significant proportion of men, i.e. 1.0% per annum (SE 0.3%) develop impairment and probable actual incapacity, at cumulative levels of dose between 50 and 100 fibre years per cc. If the occurrence of crepitations alone is regarded as a sign of actual or impending impairment the second study from Rochdale has also identified a hazard in men exposed to a cumulative dose of less than 50 fibres per cc. The data in Canadian chrysotile miners and millers suggest a substantial increment in lung function impairment and of the prevalence of shortness of breath at doses of dust at the lower end of the range (less than 100 million particles per cubic foot) and like Rochdale gives no support for the existence of a threshold within the industrial range of dose. Another study does not agree and suggests the existence of a safe threshold at a cumulative dose of 200 fibre years per cc. The present authors come down in favour of a dose response relationship without a threshold for chrysotile within the range experienced in industry. Data from the only cohort study available are consistent with a linear response. The fibrogenic effect of the fibre as used in the past in a textile factory appears to have been underestimated. However the effects of this are likely to be mitigated at least in part by the fact that (as pointed out in para 174) the present standard is enforced on the basis of measurements with more sensitive instruments and from samples taken in the immediate vicinity of the workers. The validity of extrapolation of the results of the Rochdale surveys to other parts of the industry where other types and mixes of fibre are used is debatable.

Lung cancer

192 The occurrence of lung cancer in relation to estimates of dose of asbestos inhaled by each worker is available in published studies for two industrial populations, the Canadian chrysotile miners and millers already mentioned above and a composite population of retired New York and New Jersey production and maintenance service workers.

193 In Enterline's studies of the New York/ New Jersey workers the standard of comparison used in calculating the relative risk is the mortality experience of the United States white male population and the comparisons are made in terms of standard mortality ratios (SMRs), the baseline being 100[128]. Table 24 shows a progressive increase in SMR with increasing dose, maintenance workers having consistently higher risks than production workers. The difference in the experience of the two groups of workers may be due to an intermittent type of exposure with high peaks among the maintenance workers, or due to a greater exposure to amosite and crocidolite in that group. As previously noted Enterline's results relate to the members of an industrial cohort who survived to become pensionable at age 65. From information published elsewhere it is known that a substantial incidence of asbestosis and mesothelioma existed in the production workers at a younger age. It is likely that asbestos related lung cancer also caused deaths prior to age 65. This being so, Table 24 represents a population of survivors and may underestimate the risk of mortality from lung cancer. There is nevertheless a clearly discernible relationship of increasing risk with increasing dose in each of the two sub groups.

194 According to unpublished submissions to the Committee made by Liddell, mortality data for 10 951 males born between 1891 and 1920 and who had worked for at least one month in the Quebec asbestos industry, is now available up to the end of 1973[77, 129]. The analysis supersedes that previously published. Two standards of comparison have been made. An external comparison based on lung cancer mortality rates for the whole of Quebec Province, the comparisons being made by SMRs (Table 25) and an internal analysis based on a case-control comparison (Table 26). Five controls were chosen for each lung cancer death matched for year of birth and known to have survived at least into the year following the year in which the case had died. Both analytical approaches show a tendency for the mortality from lung cancer to increase with dose.

THE SHAPE OF THE DOSE-RESPONSE RELATIONSHIP

195 Enterline and his colleagues consider that their data are best described by a cumulative normal curve (see Fig 6e)[131, *]. However, inspection of their data replotted to separate production and service workers in Fig 10 suggests a linear response. Liddell finds 'a clear direct relationship which may well be linear between excess lung cancer mortality and total dust exposure'. He has reservations about the linearity of the relationship for the groups of men with the lowest exposures, in Table 26, but as Fig 12 shows, these fall very close together when plotted. An alternative analysis which may be appropr a te for the material in Table 26 is to subdivide the degrees iof freedom for the χ^2 test to test for a linear trend. The test for linearity is found to be highly significant ($\chi^2=37.9$, d.f.$=1$, $P < 0.01$) and the deviations about linearity insignificant ($\chi^2=0.9$, d.f.$=8$, $P > 0.1$).

*Dr P Enterline has subsequently revised his view on this point and now regards the dose response curve as linear (Personal communication to the authors).

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196 The data for Enterline's two groups of workers and the external comparisons for the Quebec miners have been plotted in Fig 10. It is clear that, even if a linear relationship is appropriate within each set of data, the slopes of the lines are quite different. In Fig 11 the same data as in Fig 10 have been shown but with freehand lines through the vertical axis at an SMR of 100 corresponding to zero exposure. In Fig 12 Liddell's line is drawn on plotted material taken from the analysis using controls and is found to provide an excellent description of the data. (See Appendix 6).

SEMIQUANTITATIVE DATA

197 Semiquantitative data bearing upon relationship of the mortality from lung cancer to dose have been published by Newhouse (Table 27)[31, 130]. In general these show a progression upwards in relative risk for men and women according to severity of exposure (low and moderate exposure compared with severe exposure) and according to duration of exposure (less than two years compared with more than two years exposure). A relationship of increasing risk with length of exposure has also been demonstrated for anthophyllite miners. Thus, for Finnish miners exposed for less than ten years, the relative risk for death from lung cancer was 1.3; for those exposed for more than ten years the relative risk was 3.3. In neither of these studies is there data from which it is possible to study the shape of the dose-response curve.

198 In a follow up study of a group of workers at an amosite asbestos factory over a period of thirty years, Seidman and his colleagues found a relationship between length of exposure and relative risk[78]. The cumulative risk after 30 years compared with that of a hypothetical group of men of similar age subjected to New Jersey death rates is shown in Table 28. The effect of using a local standard population based on New Jersey rather than, as previously, one based on the United States, has been to reduce the relative risks reported here as compared with those reported previously[79]. The 'correction' of the death certificates by addition of autopsy data exerts a slight effect in the opposite direction and inflates the relative risks on average by about 17%. Nevertheless, although the factory concerned is known to have had very dusty working conditions, the fact that a detectable excess risk of lung cancer has been found after very short periods of exposure is a matter for concern as also are the very high relative risks for men exposed for one year or longer. The authors are confident that they were successful in identifying men who had been exposed to asbestos prior to employment in this factory (24 men). They have excluded them from the analysis.

199 As this factory was concerned with a single process (the manufacture of insulating materials) and most of the men studied were there only during wartime, it may be reasonable to assume that dose bears a reasonably constant relationship to time of exposure. If this is so a dose response relationship can be plotted (Fig 13). Wherever one places the point for the men exposed for more than two years there is a suggestion of a convex-curved or sublinear relationship (see Fig 5d). To put it in other words, the largest increments in risk of lung cancer occur at the lower exposures.

200 The only information available about the relationship between dose and response in respect of lung cancer for crocidolite comes from the Western Australian miners[155]. In what is acknowledged at present to be a very incomplete follow up, the following relative risks were found for respiratory cancer (excluding mesothelioma) according to duration of employment in the mines. The numbers of observed deaths on which the relative risks are based are given in brackets: 0-2 months, 0.94 (12 deaths); 3-11 months, 1.59 (19 deaths); 12 or more months, 2.24 (24 deaths). As the whereabouts of only 25% of the men have so far been identified and the remainder have been assumed to be living, these figures underestimate the absolute risks.

THE EFFECT OF SMOKING

201 As cigarette smoking was a common habit in all the populations studied and is known to exert a powerful effect on the incidence of lung cancer, it is important to examine the question whether the apparent excess risk of lung cancer in asbestos workers could be due to more smoking taking place in that industry than in the various standard populations taken for comparison. There is at present smoking data available on only some of the industrial cohorts, and such as exist are not suggestive that they smoke generally any more than the standard populations. However all the asbestos workers studied (Table 11) show raised relative risks from lung cancer, and moreover the gradient of risk with increasing dose (Figs 10-13) is most unlikely to be explained by increased smoking in men more heavily exposed to asbestos. As has been shown earlier all types of asbestos fibre are clearly carcinogenic in animals in the absence of the influence of tobacco smoke, and as in the human data an approximately linear gradient of risk with dose exists (Fig 10a).

202 Although a final conclusion of the issues is not possible at the present time because of the small numbers of asbestos workers so far identified who are non-smokers, the balance of the evidence suggests that the risk of lung cancer in asbestos workers who do not smoke is greater than non-smokers who have not been exposed. At Barking[82] an excess of lung cancer was found in women severely exposed to asbestos (2 observed, 0.2 expected). Elmes[162] has suggested that non-smoking insulators have a worse expectation of life than other Northern Ireland males of the same generation, and Wagoner[163] has reported the existence of a higher risk of lung cancer in non-smoking chrysotile workers in Mannheim, Pennsylvania than in non-smokers not exposed to asbestos. The only population in which it is possible to separate the quantitative effects of smoking

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and exposure to dust (Quebec) shows clearly an effect on risk from both asbestos and smoking (Table 29). Using these data it has been tentatively estimated that exposure to 1 fibre/cc. of chrysotile asbestos for fifty years might be equivalent in risk of lung cancer to that from smoking three cigarettes per month[164]. It should be pointed out, however, that the appropriate factor to use to convert particles of dust to fibres is highly speculative (see also para 189 and Appendix 6) and the relationship applies only to chrysotile miners and not necessarily to other workers. On the other hand among non-smoking men exposed to asbestos at Barking no cases of lung cancer have been observed82, and among North American insulators who do not smoke cigarettes a deficit of observed (two) cases to that expected (7.5) has been reported127.

203 A number of workers have suggested that a synergism may exist between the two factors, that is to say the combined risk of the two types of exposure may be greater than the sum of the separate risks of smoking and exposure to asbestos dust[80, 81, 82, 146] Table 30, taken from the experience of New York asbestos insulation workers, illustrates the point. In this population it has been suggested that the effects of the two risks are multiplicative, but at Barking such a relationship is only seen among the female workers in the population from the Cape Asbestos factory. The Quebec data shown in Table 29 exhibit a weak synergistic effect between smoking and dust. Saracci[165] in a recent review paper has concluded that, although the available data do not allow a definite discrimination, the model of multiplicative risks of asbestos and tobacco appears to be more plausible than that of additive risks or one where asbestos has an effect only in the presence of smoking.

Mesothelioma

204 No data are yet available which may be used with confidence in determining the shape of the dose-response relationship for mesothelioma. Such information is only likely to be made available, at least for crocidolite, if an appropriate follow-up study and analysis were completed on the Nottingham gas mask workers and it was possible to reconstruct the conditions of work.

205 The two main studies discussed in the section on lung cancer, where individual dust measurements are available, have reported very few cases of mesothelioma. In Enterline's material only one mesothelioma was reported. In the Quebec data, eleven pleural mesotheliomas have occurred in miners and millers. Table 31X shows an increasing relative risk of mesothelioma with increasing exposure based on an analysis of ten of those 11 cases. Newhouse and Berry found forty-five mesotheliomas in approximately 3 000 men and 7 000 women exposed to a mixture of asbestos fibre (Table 31)[63]. While it is clear that the risk of mesothelioma increases with length and type of exposure and therefore presumably also with dose of fibre, little can be said about the shape of the curve (Fig 14). As in Enterline's material for lung cancer, laggers fare worse than process workers in Newhouse's material. Although in people of both sexes exposed for less than two years the death rate after at least twenty years observation is less than in the other groups, further follow-up will be necessary before much encouragement can be derived from this fact, particularly if lower doses are associated with longer latent periods than are the higher doses. In his evidence to the committee and subsequently published Peto refers to a study of 20 000 workers at Rochdale who started work after 1933[61]. Only one case of mesothelioma has been reported in 15 000 men who worked for less than two years while nine cases have been found in 5 000 men who worked for more than two years, but this study has not yet been completed. According to Peto these data are consistent with a linear dose-response relationship. The existence of well-attested cases associated with domestic contact and in the neighbourhood of mines, factories and docks is a point against the existence of a threshold.

206 In the study of West Australian miners to which reference has already been made the incidence of mesothelioma was found to increase with duration of employment. For those in whom an interval of at least twenty years since first employment had elapsed the incidence rate rose from 4.4 per 1 000 for those employed for less than one year, (based on 6 cases) to 11.4 per 1 000 for those employed between one and two years, and 15.8 per 1 000 for those employed more than two years. One man who worked less than two months in the mine has so far developed a mesothelioma. The shape of the dose response relationship may be linear but the number of cases involved in this study is very small.

207 As far as the relationship between the development of mesothelioma and the inhalation of chrysotile is concerned similar conclusions apply (Table 31). Unfortunately, as no estimates of dust are available from the Australian mines no comparisons can be made between the two curves.

208 In conclusion it may be said that, while precise data concerning the nature of the dose response curve for mesothelioma are lacking, all the available information supports a relationship of increasing risk to increasing does without threshold. The shape of the curve may well be linear.

209 Cigarette smoking does not appear to influence the risk of mesothelioma.

Pleural plaques

210 The only published data which relate asbestos related pleural disease to dose are derived from the Quebec chrysotile industry and are displayed in Fig 8b[2]. A rough increasing prevalence of radiological changes in relation to increasing dose is demonstrated, but the

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differences between the two mining areas are more striking. The high prevalence of pleural changes at very low doses may indicate that these are non specific and that some of them may be brought about by substances other than asbestos, or alternatively may suggest that changes occur at levels of exposure below those experienced in industry.

Cancer of the gastrointestinal tract

211 Unfortunately the only data relating to cancer of the gastrointestinal tract to dose of asbestos dust comes from studies where the excesses demonstrated are small. The material displayed in Fig 15 in relation to Quebec miners and millers comes from the study which follows the cohort to the end of 1969 and which has been superseded[83]. It can be seen that the relationship between cancer of the intestine and rectum and increasing dose of chrysotile is weak and barely perceptible. It is stronger for upper alimentary cancers and for all gastrointestinal cancers combined, but is less striking than for respiratory cancers. According to McDonald and his colleagues the latest information from Quebec (up to the end of 1975) shows a deficiency of gastrointestinal cancers in one mining area (Asbestos) and an excess at the other (Thetford). The excesses are restricted to oesophageal and stomach cancer in men exposed to the highest doses of dust[77].

212 Enterline's data are shown in Fig 16. With the possible exception of the maintenance workers who were exposed to mixtures of chrysotile with amphiboles, the relationships between increasing dose and risk of gastrointestinal cancer are weak and nothing can usefully be said about the shape of the curves. The contrast with the dose-response curve for respiratory cancer is striking.

213 For cancers of the gastrointestinal tract there is insufficient data to determine whether or not a threshold exists. The widespread use of asbestos cement pipes in the distribution of public water supplies means that very large populations may be exposed to the ingestion of low doses of fibres*. It is therefore important to have further data about these dose response relationships and to settle the question of the existence or otherwise of a threshold in relation to abdominal cancers and the ingestion of asbestos.

Cancer of the larynx

214 There is no information on which to base an opinion about the nature of the relationship of the risk of cancer of the larynx to dose.

*According to the Department of the Environment about one third of the pressure piping used in the public water supply in Britain is composed of asbestos cement.

7 Extrapolation and the public health

215 In the previous chapters the evidence has been weighed in respect of the existence and magnitude of the risks of asbestosis and various types of malignant disease in association with occupational, para occupational and neighbourhood exposure to asbestos. Although the evidence is in many of its aspects woefully deficient, two relatively firm conclusions have emerged:

(1) Although animal work shows that all the principal types of asbestos fibre are potentially carcinogenic, there is evidence in man that the use of crocidolite and possibly amosite has in the past been associated with a greater risk of mesothelioma than chrysotile. For lung cancer the information is less complete but is consistent with the view that mixtures of chrysotile rich in the amphiboles, crocidolite and amosite, have been more dangerous than chrysotile or anthophyllite alone. However it was pointed out that, should chrysotile be milled in such a way at present or in the future that its physical characteristics and respirability in industry approach those of the UICC standard samples, this may well remove at least some of the features which have served in the past to render it less dangerous than crocidolite and amosite.

(2) Where quantitative data from industry are available the interpretation which we prefer is that with the possible exception of gastrointestinal cancer there is no safe threshold of dose below which there is no risk of asbestos related illness. This is true for asbestosis and lung cancer (where the data depend heavily on experience with chrysotile) and also for mesothelioma. Quantitative evidence on the shape of the relationship between dose and response suggests that it is linear for lung cancer and asbestosis, at least in respect of chrysotile, within the range of occupational exposure. It is unfortunate that in occupational situations where the highest relative risks have been found (where the amphiboles, crocidolite and amosite, or mixtures rich in them, have been used – see Table 11) little strictly quantitative data about the dose-response relationship are available. Such evidence as exists on the important issue of the dose-response relationship for mesothelioma suggests that it may be linear.

216 If it is accepted that there is no evidence for the existence of a threshold of dose of asbestos below which there is no pathological response, at least as far as respiratory cancer is concerned, the question of general environmental contamination and of its possible effects becomes one which merits serious consideration.

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Environmental contamination

GENERAL ATMOSPHERE

217 This may be measured in two ways. The first, which provides a guide to current conditions, involves the collection of airborne samples of dust in selected areas such as road crossings in cities, buildings containing asbestos insulation, the neighbourhood of areas where asbestos materials are mined or manufactured, and the like. These data so far are not available systematically. The second approach is to measure the quantity of asbestos fibres present in the lungs in routine autopsies of persons not dying of asbestos related disease. Although this method studies the dose retained in the lungs over a lifetime and therefore may give information about past conditions, the selective removal of certain fibres by mechanisms which are currently poorly understood limits its usefulness in this context (see Fig 4).

OUT OF DOORS

218 Tables 5 and 6 summarise data about atmospheric contamination measured in England, France and the United States. As the measurements are expressed in nanograms per cu. metre, it is necessary to attempt a conversion to fibres per cc. if the material is to be seen in perspective against the dose-response material derived from industrial experience and displayed in Figs 7-12, 15, 16 and Tables 22-26. Unfortunately, as the figures summarised in Table 32 show, the number of asbestos fibres over 5 μm in length contained in a nanogram of asbestos material varies widely under different conditions, so that the validity of any single conversion factor for general application is doubtful. However, if we take as an example the conservative relationship of 20 fibres suggested by Bruckman and Robino and Wagg, we find that the current hygiene standard of 2 fibres per cc.* would be equivalent to 10^5 nanograms per metre[3, 84, 85]. Looking again at Table 5, we see that even the highest figures for contamination of the general atmosphere (100 nanograms per metre^3) are three orders of magnitude below the current hygiene standard for chrysotile. Zielhuis, using slightly different assumptions, comes to the same conclusion[1]. Using the extreme average conversion factors in Table 32 we obtain for 100 nanograms per metre^3 respectively 2% (Dement[107]) and 0.025% (Lynch[108]) of the current standard.

219 The potential hazard of discarded asbestos is indicated by Harwood and Blaszak's findings of 60-100 million asbestos fibres/m^3 (i.e. 60-100 fibres per cc.) in air near to waste dumps in the United States[86].

IN BUILDINGS

220 In view of the very widespread use of asbestos in the construction and insulation of buildings, including residences and public buildings such as schools, hospitals and offices, there has been concern about the possible risks associated with inhalation of air in such premises. Nicholson and his colleagues found in nineteen buildings investigated values ranging from 2-200 ng/m^3 but Sebastien and colleagues found levels as high as 800 ng/m^3 [3, 108, 87]. Chrysotile and amphiboles have been identified and the proportion of fibres with diameters > 0.5 μm and with lengths in excess of 5 μm was similar in each case to those in industry, i.e. 1% for chrysotile and 20% for amphiboles[87]. In a survey of buildings containing asbestos materials in the United Kingdom, Byrom and his colleagues found that sixty of seventy-three examined (82%) had concentrations of less than 0.02 fibres per cc. (i.e. $> 1\,000$ ng/m^3)* the peak figure identified being 0.08 fibres per cc. (i.e. 4 000 ng/m^3)[88]. No attempt was made to classify the type of fibre present. This study is of particular importance because the method and scale of measurement used was similar to that used in industry and does not require conversion to a different scale. It can therefore be related with greater confidence to industrial experience of morbidity than can the other data reported above. The peak figure identified is 25 times lower than the current standard for chrysotile and 2.5 times lower than the current standard for crocidolite. The authors were of the opinion that, as many of the measurements were made within two to three years after construction had been completed, they would be unlikely to be sustained and would tend to decline. On the other hand abrasion or damage to certain asbestos containing materials may increase levels (Table 34). Byrom and his colleagues' results are displayed in Tables 33(a) and (b). No relationship is shown between type of building and level of contamination, but the type of material used is seen to be significant. The emissions of asbestos which take place from insulation board and sprayed asbestos are greater than those found in buildings with asbestos cement sheeting and other products. In special circumstances where dry-sprayed asbestos fibre is exposed to the abrasion of air-movements, higher levels have been observed. Young reports an elementary school in the United States where asbestos fell from the ceiling on to the furniture, and personal samplers attached to the children's clothes showed concentrations of up to 3.8 fibres per cc. Harries has described a naval store in which the level of crocidolite exceeded the standard[90].

221 The Royal Institution of Chartered Surveyors, in their evidence to the Advisory Committee on Asbestos, classify materials within buildings on the basis of the putative risks during installation, normal use and removal or demolition[5]. The first and last of these are occupational risks and have been mentioned in Chapter 2. As far as the risks to health associated with normal usage are concerned, these are presumed by the RICS to be closely associated with the liability of the materials to damage, and it is here that any possible

*For fibres other than crocidolite.

*Wagg's conversion factor of 20 fibres to nanograms has been used. The original data were reported in fibres per cc.[85]

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risk to the public may exist. The RICS evidence on risks of damage associated with normal usage in buildings is summarised in Table 34. It can be seen that surveyors consider the liability to damage during normal usage to be high in relation to a wide range of materials, principally those used for fire, heat and acoustic insulation, but that the risk of damage associated with all asbestos cement products, except ducts, is considered to be low. The low probability of damage and therefore release of fibre in respect of asbestos cement materials in buildings is reassuring, particularly in view of the scale of production of these materials. However, the high risk of damage to insulation materials which contain a high proportion of amosite (now) or crocidolite (in the past) gives cause for concern.

222 The published evidence suggests, therefore, that with the possible exception of air in the vicinity of certain uncovered waste dumps, any possible health hazard associated with inhalation of asbestos is more likely to be due to contamination within buildings rather than in the outside air. It would therefore seem prudent to obtain more information about asbestos levels in new and old buildings in relation to type and usage of asbestos containing materials, particularly insulation materials.

PUBLIC SERVICE VEHICLES

223 Crocidolite containing sprayed asbestos provides thermal and acoustic insulation in a substantial proportion of British Rail's passenger carriages. As the ventilating system between the passenger compartments and the external vents on the roofs of the carriages passes between the internal and external shell of the coaches where the crocidolite has been sprayed, and is not sealed off from the crocidolite by pipes or ducts, there is at least a theoretical possibility of contamination of the air in the carriages during motion. The population potentially at risk is large. However in a series of measurements of asbestos dust undertaken by British Rail under operational conditions most of the readings have shown no detectable fibre and none has exceeded 0.05 fibres per cc.[91]. These carriages are being phased out and replaced by rolling stock which do not contain asbestos.

STUDIES OF LUNG TISSUE

224 A number of studies have attempted to measure the quantity of asbestos fibre in lung tissue taken at autopsy. In two in particular there has been an attempt to make comparisons of the asbestos burden in different locations or at different times using comparable techniques. Oldham made a systematic search for asbestos bodies in the lung tissue of samples of persons dying in London in 1936, 1946, 1956 and 1966[92]. The percentage of positive cases rose from 0% to about 20% in each sex during the 30 year period. In a recent electron microscopic study of autopsy material, lung tissue in half of the control subjects exhibited asbestos fibres (see Table 14). Comparison between the prevalence of asbestos bodies in autopsies from different centres in 1966 yielded the following results:— Finland 38%, Glasgow and East London 22-26%, Dresden, Belfast, West London and Liverpool 13-17%, Dorchester and Nottingham 6.9% and Galway 1%. Doniach, Swettenham and Hathorn measured the prevalence of asbestos bodies in a series of necroscopies carried out at the London Hospital in 1965-66[93]. Working from information in the hospital records they identified a relationship between the prevalence of asbestos bodies and manual work and with work in the shipping, electrical and engineering and transport industries. There was also evidence of a relationship with residence in the industrial areas near to the docks, but the occupational detail is insufficient to be sure that this association can be related with confidence to environmental rather than occupational exposure. This is a general objection to this type of study. While it demonstrates that the inhalation of asbestos fibre is widespread and increasing, it does not accurately measure the relative sizes of the occupational and environmental component of the total lung burden.

WATER

225 No information about contamination of water in the United Kingdom has been found. Almost all the information reported has been from Canada and the United States from areas where asbestos is mined or dumped into lakes, and is of little relevance to conditions in this country. In the Netherlands, where contamination from geological sources and mining is not possible, asbestos concentrations in mains and tap water approximating to 10^5 fibres per litre (100 fibres per cc.) have been found[94]. It is thought likely that asbestos cement main water pipes are a source of these fibres. In the United States, the American Waterworks Association Research Foundation undertook a study designed to determine whether asbestos fibres were picked up by water passing through asbestos cement pipes[38]. An increase in the numbers of fibres between well and tap was detected in both systems tested. More data about the leaching of asbestos fibres from water pipes, under various conditions of acidity and age of piping, and with careful attention to the problem of contamination of the samples during experimental processing, is needed.

FOOD AND BEVERAGES

226 Asbestos fibres have been reported in beer in the United Kingdom and North America and in soft drinks and various wines and spirits[4, 95, 96]. Although these beverages have been filtered through filters containing asbestos, the levels of asbestos reported are of the same order as those of the local water supply. Information about the contamination of food with asbestos is scanty. The use of talc in the preparation of foods such as rice and sugar confectionery has been claimed to be a source of ingested asbestos fibres[97]. However, there are no reports of asbestos in food grade talc in the UK.

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The public health risk

227 In considering the public health risk we must take into account the following factors: (1) the increasing scale of distribution and utilisation of chrysotile and amosite in this country (and the continuing presence of crocidolite) (see Fig 2a and b); (2) the very large and continuing improvement in the control of dust in many of the workplaces where asbestos is used. The effect of these two phenomena is probably to decrease the number of heavy, continuous exposures to asbestos and to increase the number of brief or relatively light exposures. Unfortunately it is in the area of response to low doses of asbestos that the current data, depending as it does heavily upon the past industrial experience of miners and textile and insulation workers, is weakest. Bearing this in mind together with all the other inadequacies of the data mentioned in the previous sections, it is clear that a very considerable degree of uncertainty must be attached to any conclusions*. However the scale of use of asbestos and the lag period of up to 30 years which may exist between the adoption of a practice (e.g. the large scale utilisation of asbestos cement in water pipes) and the observation of any effect on health underline the urgency of attempting a judgment on these matters.

CHRYSOTILE AND CHRYSOTILE-RICH MIXTURES

228 In respect of lung cancer a judgment on the possible public health risk can only be obtained by extrapolating from the experience of the Canadian chrysotile miners and Enterline's production workers (Tables 24, 25 and 26). As the dust measurements are unfortunately expressed as millions of particles per cubic foot-years it is necessary to transform the scale to fibre years per cc. if they are to be related to Byrom's figures for buildings. Let us assume that the worst contaminated building in Table 32 would, if the levels were sustained, lead to a cumulative dose of 0.08 fibres per cc. $\times 60$ years $= 5$ fibre years per cc. If we use any conversion factor from particles to fibres exceeding unity (i.e. any figure within the range of means found by Gibbs and Lachance or the figure of 6 suggested by Ayer and Lynch) (see para 189) the estimate of excess risk of lung cancer associated with a cumulative dose of 5 fibre years per cc. would approximate to zero and a very large population would require to be exposed to yield a material number of cases attributable to asbestos.

229 For mesothelioma there are less quantitative data available, but the rarity of this condition in Quebec, both in miners and in their families, and the absence of any correlation there between the occurrence of this tumour and residence near the chrysotile mines are encouraging. The evidence that is available does not suggest that there is likely to be a risk of mesothelioma to the general public associated with chrysotile fibres, at least of the configurations used in the past.

230 For *asbestosis* the situation is similar. When due allowance is made for differences in method of measuring dust between the only available industrial data on early asbestosis (Rochdale) and Byrom's dust measurements from buildings, the latter are found even in the worst case to be more than an order of magnitude smaller than the doses experienced by the former. It therefore seems unlikely that any appreciable number of cases of pulmonary impairment have arisen due to the contamination of buildings such as houses, offices and schools with chrysotile contained in the building materials.

231 Significant prevalences of pleural changes, possibly attributable to asbestos, have been found at the lowest levels of industrial exposure in the Quebec mining area and in the neighbourhood of chrysotile deposits in Turkey. This raises the possibility that some proportion of these were attributable to past atmospheric pollution, but in the absence of measurements of ambient air levels in these towns in the past no extrapolation from these data can be made. The question of the specificity of some of these changes to chrysotile also remains. Four cases of pleural thickening with calcification in persons who never worked in the industry have been attributed by Hourihame and his colleagues and by Mann to pollution of the atmosphere near asbestos factories at Barking and Hebden Bridge respectively[156].

AMPHIBOLES AND MIXTURES RICH IN AMPHIBOLES

232 In view of the very small quantity imported to the UK there is no possibility of a public health risk associated with anthophyllite and this fibre will not be considered further.

LUNG CANCER

233 For crocidolite there is as yet no adequate quantitative information about the dose response curve for lung cancer from which extrapolations can be made. Nothing definite can be said, therefore, about the possible public health risks to persons exposed to this material in buildings and public service vehicles. For mixtures of asbestos rich in crocidolite and also containing amosite, Newhouse's data show no significant excess risk of lung cancer for men who had been exposed to low or moderate concentrations of dust for less than two years and had been followed for over 25 years[31] (see Table 27). For women exposed to low concentrations (e.g. in the clerical department of the same factory) there was an excess of cases but the numbers are very small, and women who had worked in the factory for many years were included. A public health hazard in relation to lung cancer associated with the type of mixtures used in this factory seems unlikely.

*An additional point is that to consider the public health risk one must extrapolate from the experience of persons of working age (16-64) to populations with a different age structure. This may be important if the very young and the aged are more susceptible to carcinogens than those of working age.

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234 The highest risks of lung cancer in relation to work with asbestos have been observed in the insulation trade, and in the production of insulation materials from amosite. Excluding a group selected because of long service in the industry with a particularly high relative risk, the highest recorded risk of lung cancer in insulators is 4.9 and in insulation production workers is 6.3. In this latter group a measurable risk of lung cancer (see Table 28) was reported in men who had worked less than 1 month, and parenchymal and pleural lung changes and mesotheliomas have been found in some of their domestic contacts. If these high risks cannot be explained away as being due to special factors in the working conditions of this particular industry, they may point to a possible area of public risk, especially in connection with the increasing use of amosite as a substitute for crocidolite in insulation materials. Selikoff argues that the presence of very high relative risks of lung cancer in production workers using exclusively amosite is a strong point against conditions of work or cofactors associated with fitting insulation materials being responsible for the high relative risks in the insulation trades[98]. As there is no reliable information about lung cancer in amosite miners, or from any other group of workers employing materials containing amosite exclusively or in high proportions, it would seem sensible to suspend judgment on the existence of a possible public health hazard of lung cancer associated with amosite, and commission further work.

MESOTHELIOMA

235 As has already been mentioned on p.30-31, mesothelioma is known to have occurred as a result of environmental air pollution in the neighbourhood of crocidolite mines and of the Barking factory in which mixtures rich in crocidolite and amosite were used. It seems to be rare and possibly non existent in the neighbourhood of the South African amosite mines, although a question about the adequacy of reporting in this area remains. There seems to be convincing evidence of the occurrence of neighbourhood cases in the vicinity of the shipyards and an asbestos factory in Hamburg[99]. In the excesses of cases reported from other shipyards and from the vicinity of asbestos production plants it is often uncertain what proportion of the cases are of occupational or domestic origin and what proportion due to contamination of the general environment[69,100]. Until much more is known about their distribution and the frequency of asbestos fibres and other minerals in their lungs (relative to controls) no assumptions may be made about the significance of the cases of mesothelioma which are reported without a history of occupational or domestic exposure (usually about one third of the total). In the meantime we have no information on what number, if any, of these cases are due to environmental contamination.

236 The point which has given rise particularly to anxiety in the public mind about mesothelioma has been the brevity and apparent triviality of some of the exposures involved. In the populations of British and Canadian gas mask workers the maximum total period of exposure was limited to a period of 2-3 years during the second World War, and the nature of the activity involved (fitting filter material into containers) suggests that relatively low concentrations of dust by industrial standards may have been present in the air.[55,58] Other cases have been reported amongst persons who had played upon waste dumps of asbestos or who had been associated with asbestos for relatively short periods during recreational activities, but unless careful comparisons are made with controls a chance association with material as ubiquitous as asbestos may mistakenly be interpreted as causal (see also p. 32). In the well controlled Canadian and United States surveys McDonald and McDonald found no evidence of an association between leisure time activities involving asbestos and the occurrence of mesothelioma[101]. Ashcroft, in a study of all diagnosed cases of mesothelioma in Tyneside over a period of twelve years, made a unique attempt to relate the frequency of asbestos bodies in the lungs of the cases to the frequency occurring in a representative sample of autopsies for other conditions[71]. Of thirty one cases of mesothelioma studied, almost all of whom had a history of exposure to asbestos in industry, twenty nine had numerous coated fibres and only two occasional fibres. In contrast he estimated, by extrapolating from his small series of controls, that about 500 000 of the adult population of Tyneside had occasional fibres and only about 50 000 adults had numerous fibres in their lungs. He estimated that the approximate relative rates of mortality from mesothelioma for, respectively, those with many and few fibres in their lungs were 600 and 4 per million. He concluded that there had been little evidence up to that time of an appreciable risk of mesothelioma due to environmental contamination on Tyneside. Another hopeful finding which has already been mentioned is that workers of both sexes exposed for less than two years at the Barking factory have so far (after 20 years) experienced low death rates from mesothelioma (Fig 14). However, if, as has been suggested by Selikoff, latent period is inversely related to dose (i.e. the lower the dose the longer the latent period) it may be too early to base much encouragement on this finding[36].

237 The significance of the well documented cases of mesothelioma due to domestic exposure (almost all were exposed either to crocidolite, to amosite or to mixtures of asbestos containing them) in relation to the presence or otherwise of a more general environmental hazard depends upon the view one takes of the scale of the dose which these exposures represent. Unfortunately practically no information is available on this point. According to the authors of the NIOSH report Selikoff has found "that asbestos concentrations in excess of 100 ng/m^3 may often be present in the homes of asbestos workers with the highest measured concentration being

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5000 ng/m³,"* i.e. in the extreme case a figure approaching the present hygiene standard for crocidolite, but the data on which this statement is based do not appear to have been published[52]. Selikoff, Nicholson and Langer have found amosite fibres in the settled dust and household air of homes occupied by workmen of an amosite factory fifteen years previously[102]. Bearing in mind the virtual indestructibility of asbestos it is possible to envisage circumstances where asbestos fibre could accumulate in the household of an asbestos worker (e.g. lack of changing facilites at work and of vaccuum cleaning and washing facilities in the home) and substantial levels be present in the household air over long periods of time. Even although such conditions are unlikely to recur today it would still be helpful to try to establish, by simulation techniques if necessary, an approximation of the dust levels which were associated with the occurrence of 4 mesotheliomas in a population of 326 domestic contacts of amosite workers (para 148). Unless these are found to have approximated to or exceeded the current hygiene standard, the occurrence of four cases in such a small population is bound to raise the possibility of a more general environmental risk associated, for example, with the use of amosite sprayed asbestos in buildings. There seems little doubt that mesothelioma can occur at dose levels which leave no radiological trace of parenchymal or pleural asbestosis.

238 In conclusion of this section it must be said that it is our opinion that it is on the whole unlikely that there is a material public health risk of mesothelioma in relation to the inhalation of amphiboles and mixtures rich in them. The small absolute number of mesotheliomas registered in comparison with other causes of death, in spite of the widespread use of amphiboles in Britain for at least 50 years, the slow rate of increase of these cases, the improvement of industrial practice in the last 20 years, and a number of other favourable points which have been mentioned above, all give grounds for optimism†. Nevertheless an important area of uncertainty remains. This relates to the significance of small doses, and has its origin in our inability in retrospect to determine with any certainty the size of the doses which led to the occurrence of the attested neighbourhood and domestic cases of mesothelioma in the literature. The only way in which this anxiety can be dispelled finally is by firm information to be made available on the size of such doses and on the characteristics of the dose-response relationship for mesothelioma to the amphiboles.

EXPOSURE TO ASBESTOS IN CHILDHOOD

239 Anxiety about exposure to asbestos in childhood has arisen because it is possible that carcinogenicity may continue as long as the fibre remains in the body, i.e. over a lifetime, and for the obvious reason that as far as mesothelioma is concerned where the average latent period is about 40 years, early exposures are more likely than late to lead to the development of a tumour before death from other causes supervenes. There is also the speculative point that tissues in which natural growth is taking place may be more susceptible to carcinogens than adult tissues. Nothing is known about lung cancer originating from exposure to asbestos in childhood. A small number of the published neighbourhood cases and 10 of the domestic cases of mesothelioma arising in adult life have been ascribed to exposure in childhood at home or in the vicinity of works and dumps, and although uncertainty may be attached to individual attributions, most of these cases, particularly when they have occurred in clusters, can be accepted as valid[18,22]. Although they are very rare, well attested cases of mesothelioma in which the tumour developed in infancy or childhood have been reported. Although the environmental data in these reports is meagre and the relationship of such cases to asbestos is not generally accepted, it is perhaps worth noting the occupations of the fathers of the 9 children[104,105] in whom this information is available in the literature. These were respectively: moulder, plumber, electrician, mechanical engineer, farmer, building constructor, lumber dealer, salesman, and ceramics engineer in a factory utilising chrysotile and amosite. Of these nine cases, it is possible to assume from the nature of the employment parental exposure to asbestos as certain in one and probably in five. There is no evidence up to the present time of cases of mesothelioma having occurred as a result of exposure in schools, although unsatisfactory conditions have been reported in one school in the USA. Bearing in mind the special fire risks of schools and the use of sprayed asbestos including amphiboles to counter these, and the points mentioned above, further surveys of schools are indicated. Exposed dumps of asbestos waste are another possible location for childhood exposure and attempts should be made to locate and cover these.

Beverages and food

240 The only evidence in man with a possible application to this problem is derived from a study of cancer in a population living on the shore of Lake Superior, whose water supply had been contaminated by dumping of asbestiform fibres associated with taconite mining since 1955. No excess of cancer deaths was detected up to the end of 1969 but observation of at least thirty years will be necessary before a final conclusion can be made about this population[103]. The degree of contamination of drinking water due to leaching of asbestos fibres from asbestos cement pipes in the Netherlands is at least two orders of magnitude less than the contamination at Duluth, but information is necessary about this phenomenon in the UK, in particular

*In an interview with E D Acheson, Dr Selikoff mentioned a top level of 1000 ng/m³ based on measurements in homes of men currently employed. Can one assume that conditions in the period 1942-54 were worse?

†The latest available figures available for the United Kingdom are: from all cancers, 139 899; from lung cancer, 37 152 (1975); from mesothelioma (Britain only), 292 (1976).

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concerning the geographical distribution of these pipes, the period they have been in use and the types of fibre involved. In the American Water Works Association's estimate, a lifetime of drinking 2 litres of water a day might give approximately 3% of the dose a man employed in the asbestos industry in the past might have ingested from swallowing his bronchial secretions[38]. Although the basis of both these estimates is dubious, the important point to be made is that, if there is a risk of gastrointestinal cancer associated with asbestos ingestion, and the dose response curve is linear, the occurrence of small quantities of asbestos generally in piped drinking water could produce a significant number of tumours. Because the size of the population potentially at risk would be very great, such a phenomenon would also be extremely difficult to detect from the epidemiological point of view.

241 In animals, although in one experiment no significant number of tumours was induced after feeding large quantities of chrysotile,[144] another experiment involving the feeding of material from filters, which included chrysotile and a number of other substances, produced a significant excess of tumours in a wide range of organs[106]. There is no information yet published about the effects of feeding amphiboles, but ingestion of fibres from bronchial secretions and licking contaminated fur in rats subjected to inhalation of amphiboles was not associated with an excess of alimentary tract cancer[9].

8 Implications for public policy of the ill effects upon health of asbestos

242 Following the lines laid down in the terms of reference of the report each type of asbestos fibre is dealt with separately in this section. However it is important to repeat here a point made in earlier sections. This is that, because of the lag period between first exposure and the diagnosis of asbestos related disease, the conclusions reached about the effects of the different fibre types on man inevitably relate to past industrial practices. Should commercial chrysotile be milled in such a way that its respirability and the configuration of its fibres approach that of the amphiboles, animal work suggests that the morbidity gradient in favour of chrysotile seen up to the present and discussed previously may disappear in the future.

CROCIDOLITE

243 There is powerful evidence from a number of points of view that inhalation of crocidolite can cause mesothelioma, and that although the risk increases with increasing dose it is possible for mesothelioma to occur after relatively brief exposure to this amphibole. A substantial proportion of the deaths from mesothelioma which have occurred and are occurring in Britain have probably been due to the inhalation of amphiboles including crocidolite. Although the available evidence is inconclusive it is consistent with the view that crocidolite may also have been more dangerous than chrysotile as far as the causation of lung cancer is concerned. Crocidolite is one of the amphiboles associated with the excesses of cancers of the gastrointestinal tract reported in various industrial populations and may also be associated with the genesis of a small number of cases of laryngeal cancer. Evidence from the only study in man in which quantitative comparisons between crocidolite and chrysotile are possible suggests that as far as asbestosis is concerned crocidolite may be more fibrogenic than chrysotile. The data from animal work on this point are conflicting.

244 The evidence leads us to recommend that contact between man and crocidolite should be limited to the minimum practicable. The importation of raw crocidolite or articles containing it should be prohibited, and stringent regulations for the protection of workers and of the public should continue to be applied in respect of crocidolite containing materials still present in buildings, vehicles, ships, dumps and other locations.

AMOSITE

245 The importance of reaching a correct judgment about the amphibole amosite is underlined by the import trends to the UK since World War II which show a sevenfold increase in tonnage imported as opposed to

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a twofold increase in chrysotile. To put it another way the tonnage of amosite imported in 1975 (19 200 tonnes) was almost three times the largest import figure for crocidolite since the War (6 800 tonnes), reported in Table 1.

246 The principal difficulty about the evidence on the effects of amosite upon health is that relatively few definable groups of men have been exposed to amosite who have not also been exposed to crocidolite, particularly in the United Kingdom. As far as mesothelioma is concerned we take the view that the position of amosite may be intermediate between chrysotile and crocidolite. For reasons argued in detail in previous sections we consider that the majority of the cases of mesothelioma reported in insulators in North America have probably been associated with exposure to mixtures of chrysotile and amosite, not crocidolite. Eleven industrial cases of mesothelioma have been reported among 933 amosite workers (Table 11), and there are also four well attested cases of mesothelioma in domestic contacts of amosite workers. We are inclined to discount the lack of an appreciable number of mesotheliomas amongst amosite workers in South Africa on the basis of lack of sufficient 'opening' of the fibres as shipped from the mines or lack of follow-up of the workers or both.*

247 The evidence about the capacity of amosite to produce lung cancer depends upon the experience of the men at a single American factory manufacturing insulation materials. This study shows excess risks of lung cancer even after very short exposures (Table 28; Fig 13) and although measurements of dust levels are not available the most likely intrepretation is that the risk was higher than for men exposed to chrysotile. However, as unknown biases may operate in the best conducted epidemiological studies it would be foolish to consider this point proven on the basis of a single study. Mixtures of amosite with chrysotile in American insulators are associated with very high relative risks of lung cancer, but once again unfortunately dust levels are not available. Excesses of gastrointestinal cancers have occurred in heavily exposed amosite workers and in American insulation workers for whom we believe the principal amphibole inhaled has been amosite. For asbestosis there is no quantitative evidence in man which compares the fibrogenicity of amosite with other types of asbestos, while in animals the evidence about its relative fibrogenicity is conflicting.

248 We conclude that, although strictly quantitative data are lacking, the burden of the evidence suggests strongly that amosite has been more dangerous than chrysotile in respect of mesothelioma and possibly also in respect of lung cancer. If this conclusion is accepted the question must arise whether a stricter hygiene standard should be applied to amosite than to chrysotile.

*Five cases of mesothelioma have been reported following exposure to amosite at Penge, Transvaal. Two whites and one black worked in the mine. One case occurred in a person who lived at Penge and another in a person who visited Penge as a child. There was no known exposure to crocidolite or chrysotile in any of these cases. (Webster, I, National Centre for Occupational Health, Johannesburg, Personal communication).

TREMOLITE

249 Although the amphibole tremolite is not used commercially it is known to contaminate certain samples of commercial asbestos and talc. Electron microscopic studies have identified tremolite fibres in the lungs of deceased chrysotile miners. The question has been raised whether tremolite may have biological significance.

250 Although no human population has been identified which has been exposed industrially to tremolite alone a group of 250 American talc miners who were exposed to tremolite and serpentine have been followed over a period of twenty five years. One death from mesothelioma, one from pleural fibrosarcoma, and an excess of lung cancer deaths were noted[160]. On the basis of present knowledge no action is required about tremolite, but it is important that the situation should be kept under review.

CHRYSOTILE

251 Chrysotile is the only type of asbestos fibre for which data are available which permit any quantitative estimates to be made of the biological effect of a given dose of fibre. Nevertheless, for reasons already set out in detail, very considerable errors in estimation, both of the dose of the biological effect, exist and we do not wish to suggest that in the following section we are dealing other than with gross approximations.

252 The present hygiene standard of 2 fibres per c.c. was arrived at on the basis of a study of workers in employment at a particular time at Rochdale. It was estimated that exposure to an average of 2 fibres per c.c. 8 hours a day 5 days a week for 50 years would lead to the occurrence of an early but non specific sign of asbestosis (crepitations) in 1 % of the workers at the end of their working lives. A second study taking account of the men who had left shows that the rate of occurrence of crepitations in the population of exposed men as a whole had been underestimated by a factor of about 15 (see Fig 19). Even if the significance of crepitations as an isolated sign is discounted the second study shows that a material annual incidence of certified asbestosis (0.5 % per annum, SE 0.2 %) would occur after an exposure within the present standard. However, if one is able to accept, as has been suggested on the basis of parallel measurements, that personal sampling with modern techniques of fibre counting such as are used to enforce the standard at present would have arrived at measurements of dust at Rochdale in the 1950's and 1960's some two to five times those used in the calculations, the evidence in favour of asbestosis occurring at Rochdale after a lifetime's work at the present standard falls away. The implications of the data from these two studies on public policy with regard to the health standard depends to a large extent therefore

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on the view taken about the relevance of the dust measurements in them to current measurements.

253 In respect of carcinogenicity we conclude that up to the present time chrysotile has rarely caused mesothelioma and that there is little evidence that it has caused cancer of the gastrointestinal tract or of the larynx. These are favourable points which should be taken into account in framing policy. On the other hand it is essential to restate the proven capacity of chrysotile to produce mesotheliomas in animals if the fibre is of the appropriate configuration, and to emphasise once again that chrysotile, as it is used for many purposes other than for asbestos cement, is probably becoming increasingly finely dispersed and respirable.

254 As far as lung cancer is concerned the matter is far less straightforward. There are two studies from North America and one from Britain which provide data which can be used to help us arrive at a standard. In the North American studies (Quebec miners[129] and Enterline's [128] process workers) the doses of dust are recorded in million particles per cubic foot. It follows that if any use is to be made of them in the present context a transformation must be carried out. As has been pointed out this at best introduces additional uncertainties and at worst is indefensible[76]. Our approach is to show the effect of three alternative conversion factors. For the Rochdale lung cancer study we show the effect of taking the dust levels as measured and of increasing them by factors of 2 and 5 to take account respectively of the effect of modern instruments, and of the combined effect of modern instruments and personal samplers (Table 37).

255 The array of figures in Table 35 sets out the excess mortality from lung cancer which would occur to workmen exposed to various chrysotile levels over 50 years from calculations based on the various studies. As it would be wrong for medical scientists working alone to select any particular yield of lung cancer as appropriate a range of yields is allowed for from 2% (i.e. that 2% more of the men exposed at the given level would die of lung cancer than if they had not been exposed, and die at an earlier age) to 0.1%. As in this table the assumption of a linear relationship between dose and response is made, any other value can be found by simple proportion. The details of the various other assumptions on which the table is based are given in Appendix 6.

256 If we take the column of Table 35 showing the levels at which a 1% excess of lung cancer deaths would occur as an example we find a range of figures for chrysotile from 5 fibres per c.c. to 0.4 fibres per c.c. (for 50 years). In other words, taking the various different sets of data and using different assumptions we calculate that any value from 5 fibres per c.c. to 0.4 fibres per c.c. might lead to a yield of 1% excess deaths from lung cancer. If one is only prepared to accept an excess yield of deaths half as great (0.5%) the calculated levels are correspondingly lower.

257 Another question which has to be taken into account in making a decision about a hygiene standard is the existence of a public health risk. In Table 36, using the same studies as in Table 35, we have calculated the number of deaths which would occur if a million persons were exposed continuously 8 hours a day for 50 years to the highest recorded concentration of asbestos in the ambient air out of doors (10 nanograms per m^3) (Table 5) or to the median, or highest levels recorded by Byrom, Hodgson and Holmes in their survey of buildings (Table 33)[88]. It can be seen that, unless contaminated buildings are very much commoner than seems likely, no appreciable mortality from lung cancer can be associated with any degree of contamination by chrysotile likely to be encountered in the UK in the ambient air or in buildings not under active construction or repair. However we emphasise once more the need for further information about asbestos levels in buildings.

258 It follows from the previous paragraph that we must return to the data in Table 35 on the effect of occupational exposure to asbestos on the mortality from lung cancer to reach a final conclusion about a hygiene standard for chrysotile. In choosing a figure it should be borne in mind that there are a number of identifiable factors in the various estimates which exert opposite effects. The linear hypothesis may overestimate the risk but the Quebec data (because they are derived from mines and mills) and Enterline's data (because they are restricted to men who survived long enough to reach pensionable age) may underestimate the average risk to men exposed for the whole of a working life to more finely dispersed fibre in process work. A further conservative assumption arising from the use of the concept of a cumulative dose in Table 35 is that the biological effect of asbestos is immediate and instantaneous, i.e. the residence time of the fibre in the lungs is ignored and no allowance is made for a continuing action of fibres remaining in the body. For Rochdale the notion that doses of dust have been underestimated has not gone unchallenged[147].

259 It may be helpful to bear in mind that in the case of occupational exposure to ionising radiation a health standard has been set (5 rems per annum) which, if it was reached annually for 50 years, it was calculated would lead to an increase in cancer mortality in the exposed workers from 20% to 22%[150]. If this is regarded as an acceptable parallel to the situation with regard to chrysotile and we allow that approximately half the mortality attributable to chrysotile is due to lung cancer and half to asbestosis (see Table 11) a 2% excess mortality would be incurred in association with the levels recorded in the penultimate column in Table 35. These levels range from 5 fibres per c.c. to 0.4 fibres per c.c. Bearing in mind the points made in the previous paragraphs a figure towards the lower end of this array might represent a compromise between what are, to be truthful, very considerable uncertainties.

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Notes on technical terms

This paper does not pre-suppose any knowledge of epidemiology or statistics, but it was found impossible to avoid technical terms entirely. Readers may find the following explanations useful.

Prevalence and prevalence rates

If the prevalence of asbestosis in a given factory is 5 on 1 January 1978, this means that 5 of the population in that factory had asbestosis on that date. This figure tells us little unless we know the size of the population. Prevalence is therefore usually expressed as a prevalence rate: for example, a 6% prevalence rate for asbestosis in a given factory on 1 January means that on average 6 in every 100 employed in the factory had asbestosis on that date.

Incidence and incidence rates

Incidence, unlike prevalence, measures only the new cases diagnosed or reported in a given period (usually a calendar year). But, again, an incidence figure without any indication of the population involved is not very useful. For this reason, it is again usual to express incidence as a rate. For example, if at a given factory there is an annual incidence rate of 2% for asbestosis during the period under review, this means that on average 2 people were diagnosed as new cases of asbestosis each year for every 100 employed.

Average annual incidence rate

This is the average over a period of years of the annual incidence rates for those years.

Excess mortality

If in a given group of people there is an excess mortality of 2% from cancer, this means that 2% more of these people die of cancer than would do so if their causes of death were exactly the same as those found in some reference group (usually the population as a whole), and these deaths occur earlier than they would otherwise. So, if for example 20% of the reference group die of cancer, the percentage dying of cancer in the group under study will be 22%.

Cumulative dose-fibre years per millilitre and particle years per cubic foot

One hundred fibre years per ml is the cumulative dose that would result from, for example, exposure to a constant dust concentration of 10 fibres per ml over 10 years, or 2 fibres per ml over 50 years, for 8 hours a day and 5 days a week. Similarly 100 million particle years per cubic foot is the cumulative dose that would result from, for example, exposure to a constant dust concentration of 10 million particles per cubic foot over 10 years, or 2 million particles per cubic foot over 50 years, for 8 hours a day and 5 days a week. The abbreviation for 'million particle years per cubic foot' used in the figures is 'mp/cf-y'.

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Appendix 2

(This appendix was prepared by HSE from material in the Selected Written Evidence submitted to the Advisory Committee on Asbestos)

Information on asbestos usage, workforce and asbestos related disease submitted by industry

Company	Division	Asbestos types/period of use				Numbers Employed (Years)	No's of asbestos-related diseases of which firm is aware	
		Chrysotile	Crocidolite	Amosite	Anthophyllite			
CAPE INDUSTRIES LIMITED	Cape Asbestos Barking	1913-1966	1913-1966	1920-1966		10 142 since 1913	369	*workforce as per submission.
	Acre Mill Hebden Bridge	1939-1968	1939-1970	1945-1970		2 199 since 1946	73	*submission gives workforce of 2199 between 1939-1970
	Kentmere Cumbria					150 since 1949	8	*sub. gives workforce of 281 between 1949-1975
	Cape Boards & Panels Ltd Uxbridge	1945-1952		1952-		4 025 since 1950	38	*sub. gives workforce of 5 670 between 1945-1975
	Cape Boards & Panels Ltd Germiston Glasgow			1952-		350 since 1967	1	*sub. workforce 1 463 between 1952-1976
	Cape Insulation Ltd Sterling					800 since 1964	None	*sub. workforce 1 920 between 1954-1976
	Hendham Vale, Manchester (Don International)	1901-Date				10 600 since 1949	3 3	*sub. workforce 14 186 between 1901-1976
	Trist Draper Brislington Bristol	1923-Date	1964-1972 experiments in 1937	1976 experimental use only	1975-Date	1 848 since 1964	6	*sub. workforce 4 418 between 1923-1976
	Cape Universal Claddings Watford	1931-Date	1959-1968	1960-1971 (Intermittently currently stored outside)	None	790 since	18 18	*sub workforce 3 245 between 1932-1975
	Cape Insulation Contracting	1957-	1957-	1957-		2 400 since	55	*sub. workforce 11 260 between 1957-1975
RICHARD KLINGER LTD., SIDCUP KENT	Beater addition Asbestos Sheeting	1969-Date	Never			19 since 1969		*Cape claim there are two groups of workers – at Hendham Vale & Bristol with chrysotile – only exposure
	(Compressed Asbestos Sheeting)	1937-Date	Never			262 since 1968		*Klinger give workforce at present of 400 in their submission
B.B.A.	Mintex Ltd Cleckheaton	1920-Date	Mid 1930's			over 15 000 since 1941	10	
	Scandura Ltd Cleckheaton New Factory W. Auckland	1920-Date 1966-Date	1926-1967			3 500 since 1941 1 300 since 1966	76 None	
TUNNEL BUILDING PROD. LTD.	Grays, Essex	1936-Date	"small amounts of other fibres used for test purposes only"			1 336 since 1959		
BRITISH URALITE LTD	Rochester Kent	1900-Date	1939-1945			records available from 1965		

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Company	Division	Asbestos types/period of use				Numbers Employed (Years)	No's of asbestos-related diseases of which firm is aware	
		Chrysotile	Crocidolite	Amosite	Anthophyllite			
ETERNIT	Meldreth Factory	1929-Date	1958-1965	1958-Date	1958-Date	500 at present		*asbestos related disease described as "within the catchment of the Meldreth Factory" *Eternit letter on single fibre exposure gives total workforce as 350;* of these 33 have been exposed to chrysotile only.
TURNER & NEWALL	Ferodo Ltd Chapel-en-le Frith	1910-Date	1928-1932 1938-1943 (1922-1967) (experiments)			13 500 since 1942	13	*Ferodo Caernavon mentioned in submission. Production started there in 1963.
	TAC Erith	1928-Date	1928-1969 (intermittent use)	?-Date		6 000 since 1920		*T & N say workers exposed to amosite or crocidolite will also have crysotile exposure–the best defined being at Ferodo Caernavon.
	TAC Trafford Park	1914-Date	?-1966	?-Date	?-Date	No figures given		*1 extra mesothelioma victim mentioned. An asbestos sprayer at J. W.
	TAC Widnes & Ditton	1916-Date intermittent use	?-1970	?-Date		10 000 since 1918	46	Roberts before its merger with Ferodo. Exposed to crocidolite and amosite.
	TAC Tamworth	1941-Date	Never	Aug-Nov 1976		1 300 since		
	TAC Rhoose	1935-Date	1933-1935 (prior to TAC takeover)	Aug-Nov 1976		4 500 since 1935		*plus one mesothelioma at Rhoose; the victim worked with AC products from 1932-1935 before TAC took the factory over.
	Engineering Components Slough					1350-2000 since 1932	None	
	Engineering Components Ltd Heckmondwike/ Cleckheaton					300 at present	None	
	Newalls Insulation Co. Ltd	No use of asbestos since 1972. No further specific information				1 100 at present	?	
	TBA Industrial Products Ltd	?	?	?	?	?	235	
	TBA Rochdale Non-Textile Processes	?-Date	?-1969			1550 = since 1971	None	

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Appendix 3

Second Rochdale study of asbestosis: Dr G. Berry's derivation of incidence rates

Crepitations	Pre-1951 group Total	Cases	Incidence % p.a.	Post-1950 group Total	Cases	Incidence p.a.	Both groups Incidence p.a.
Cumulative dose							
< 50	8	2	4.7	83	6	1.1 } 1.6	1.3 } 2.0
50—	27	6	3.6	79	11	2.2 }	2.5 }
100—	39	10	4.2	28	6	3.4	3.9
150—	40	8	3.1				
200—	23	9	7.5				
250+	21	8	6.3				
	158	43		190	23		
Possible asbestosis							
< 50	8	1	2.1	83	2	0.4 } 0.7	0.5 } 1.0
50—	26	5	3.1	77	5	1.0 }	1.5 }
100—	39	6	2.5	30	4	2.0	2.3
150—	43	10	3.8				
200—	24	8	6.2				
250+	22	6	4.4				
	162	36		190	11		
Certification							
< 50	8	0	0	84	1	0.1 } 0.5	0.1 } 0.5
50—	27	2	0.9	81	6	0.9 }	0.9 }
100—	40	3	0.9	32	3	1.1	1.0
150—	46	5	1.4				
200—	29	6	2.7				
250+	29	5	2.4				
	179	21		197	10		

Berry, G (1977) Personal Communication.

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Appendix 4

Calculation of the attributable excess mortality associated with asbestos in eleven industrial cohorts

Table 11 dealt with observed and expected deaths in 11 cohorts from 'asbestos related' disease. This appendix shows mortality from all other causes and gives an assessment of the relative importance of the asbestos related deaths.

If the excess deaths (O - E's) are summed by cause over ten studies (excluding No. 8) the ranking in numerical importance is:

Lung cancer	579
Asbestosis	354
All other causes	257
Mesothelioma	186
G. I. cancer	113

The excess for 'all other causes' may reflect in some cases the use of an inappropriate standard population for calculation of the expected number of deaths – alternatively other asbestos attributable deaths could be included among these 257 fatalities.

The proportion of deaths over and above those expected which may be attributable to asbestos ranges from 3% in Quebec to 116% in the dockyard workers in Belfast. Even correcting for a possible underestimation of expected deaths in this latter population only reduces the attributable excess mortality to 70%, and for insulation workers in US and Canada the proportion is 48-65%.

Extension of Table 11

Cause of death	No. of deaths	Study 1	2	3	4	5	6	7	8	9	10	11
All other causes	O	605	2 666	215	220	376	250	829	207	47	492	197
	E	593	2 559	209	224	293	279	866		31	479	107
	O-E	12	107	6	—4	83	—29	—37		16	13	90
	O/E	1.02	1.04	1.03	0.98	1.28	0.89	0.96		1.53	1.03	1.84
Cause unknown	O	19	69	—	—	—	—	—		—	29	—
Σ (O-E)[a]		66	100	62	86	122	175	435	13	44.2	86	57
A.E.M.		0.10	0.03	0.25	0.32	0.38	0.57	0.45		1.16	0.16	0.42
C.A.E.M.		0.10	0.03	0.24	0.33	0.28	0.65	0.48		0.70	0.16	0.13
Exposure[b]		25 years average	1 month	10 years	10 years	Nil?	20 years		3 months	10 years average	Nil?	2 years

[a]Cancer of lung + G.I. cancer + mesothelioma + asbestosis.

[b]Time from first exposure to entry to study.

A.E.M. = Attributable Excess Mortality = Σ (O-E)[a]/Expected deaths from all causes.

C.A.E.M. = Corrected A.E.M. = $(\Sigma O^a - \lambda . \Sigma E^a)/\lambda . E$ (all causes) where $\lambda = \frac{O}{E}$ for all other causes.

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Appendix 5

Case rates of asbestos related disease by period of chrysotile and amphibole usage calculated from data in Appendix 4

Years of usage		Mesothelioma cases	Asbestosis cases	No. of employees	Rate/1 000	
Chrysotile	Amphibole				Mesothelioma	Asbestosis
< 50	None	0	0	262	0	0
50+	None	0	3	10 600	0	0.3
< 50	< 20	0	0	1 336	0	0
		2	16	790	2.5 } 0.8	20.3 } 6.5
		0	1	500	0	2
50+	< 20	9	37	21 800	0.4	1.7
		4	6	15 000	0.3 } 0.4	0.4 } 1.0
		0	6	1 848	0	3.2
		8	5	13 500	0.6	0.4
< 50	20+	3	70	2 199	1.4 } 0.6	31.8 } 17.2
		1	37	4 025	0.2	9.2
50+	20+	49	320	10 142	4.8 } 4.4	31.6 } 28.2
		11	65	3 500	3.1	18.6

Source: Data in Appendix 2 – information submitted to the ACA by industry.

N.B. The last pair of firms not only have used amphiboles the longest (53 and 41 years) but started earliest (1913 and 1926). The penultimate pair of firms used relatively little chrysotile (29 and 7 years) but did not start to use amphiboles until more recently (1939 and 1952).

Rates of mesothelioma and asbestosis by period of use of chrysotile and amphiboles in firms in operation in 1950 or earlier – based on Appendix 2.

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Assumptions underlying calculations in Tables 35 and 36

Use of dose-response relationships for lung cancer for estimating relative risks associated with various exposures to asbestos

In considering any predictions or extrapolations based on the available data the inadequacies and problems, some already mentioned, should always be borne in mind. As shown in Figure 11, even if a linear relationship between response and dose is a reasonable representation, the slopes vary. These differences may be due, among others, to:

(a) exposures to different fibre types or mixtures
(b) different work conditions
(c) varying accuracies of dust measurements
(d) varying fibre: dust ratios
(e) differences in size distribution of the fibres
(f) different relationships between airborne fibre concentrations and the amount of fibre deposited in the lungs
(g) differing follow-up periods
(h) appropriateness or otherwise of external standard population
(i) differences in background level of lung cancer in standard populations
(j) smoking habits of industrial population relative to standard population
(k) differences in smoking habits between men at varying levels of cumulative dose
(l) use of a retired population compared with a full cohort.

The estimates shown in Tables 35 and 36 are based on the assumption that a linear relationship describes the data adequately as argued in the text (para 195).

Liddell, in a submission to the Advisory Committee, fitted by eye a straight line to the Quebec miners and millers data which is shown in both Figs 11 and 12[157]. Our own eye fits to Enterline's data are drawn on Fig 11. In both studies the duration from first exposure to mortality was long. In the Quebec study only deaths occurring after 20 years from first exposure are included, whereas in Enterline's study of retired workers the average duration of exposure was 25 years. It has generally been observed that at least this period of follow-up is necessary to detect the increased risk of lung cancer from asbestos exposure, and there are suggestions that, after this time the increased relative risk stabilises[29, 78]. This is important to the projections made below, based on the following equations*:

*SMR = Standardised mortality ratio
CD = cumulative dose in million particles per cubic foot-years

(a) Quebec: $\frac{\text{S.M.R.}}{100} = 1 + \frac{3}{1\,600} \times \text{C.D.}$

(b) Enterline (production workers): $\frac{\text{S.M.R.}}{100} = 1 + \frac{6}{1\,600} \times \text{C.D.}$

(c) Enterline (maintenance workers): $\frac{\text{S.M.R.}}{100} = 1 + \frac{20}{1\,600} \times \text{C.D.}$

From the submission to the Advisory Committee by Peto[61], and assuming linearity, it is possible to represent the experience of the Rochdale factory as:

(d) Rochdale: $\frac{\text{S.M.R.}}{100} = 1 + \frac{1}{200} \times \text{C.D.}$

where in this case only the cumulative dose is measured as fibres per cc.-years, and not as million particles per cubic foot-years.

To enable dose levels for (a), (b) or (c) relevant to any given S.M.R. to be calculated in fibres per cc. it is necessary to use some conversion factor from particles to fibres. Although there is considerable disagreement about the feasibility of making such a transformation, we have used three conversion factors:

(i) 5 fibres/cc. per 1 million particles per cubic foot, i.e. 5:1, as
(ii) 2:1, suggested as a minimum by the Beaudry report[155].
(iii) 1:1, apparently not unreasonable at the Rochdale factory[74].

For projections at Rochdale we have incorporated two important features of fibre measurements –

(i) if present instruments and assessment procedures had been employed in the 1960's, they would have yielded higher results by about two to three-fold;
(ii) personal sampling is believed to yield higher results than the original area sampling by a factor of about one to two-fold.

For details of these conversion factors see Table 37.

After the age of 50 years approximately 10% of deaths in men are due to lung cancer, and these represent some 95% of all lung cancer deaths in men. Thus, for example, an S.M.R. of 110 would increase the percentage of deaths that are due to lung cancer from 10% to 11%, i.e. some 1% extra of all deaths* would be due to lung cancer (as 90% of men die over age 50).

Table 35 shows the concentrations of asbestos dust in fibres per cc. which would produce various levels of excess mortality from lung cancer according to the linear models above. The fibre concentrations have been

*This percentage, the extra among all deaths that are due to lung cancer, is shown in the headings of the columns of Table 35.

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calculated by dividing the associated cumulative doses by 50 – i.e. taking a 50 year occupational exposure. The excess mortality can be thought to occur from some 30 years onward after first exposure, i.e. from age about 50 – although in practice the full cumulative dose would not be reached until retirement. So that, for example, the dust levels associated with an excess mortality of 1% from lung cancer range from 0.2 to 5 fibres/cc., being higher for chrysotile than for mixtures (see penultimate column of Table 35). The higher the fibre: particle conversion factor, the higher the associated dust level (this is because the more fibres taken to be in the same amount of dust, the less 'effective' each fibre can be considered). The higher the excess mortality, the higher the associated level.

Table 36 shows, using the same linear models, the number of extra deaths among one million births that might occur due to environmental asbestos dust levels. Figures related to three levels are shown:

(i) the highest urban air levels of asbestos recorded in the UK – 10 nanograms per cubic metre (see Table 5) transformed to 2×10^{-4} fibres per cc. using Wagg's conversion factor shown in Table 32 – which over 50 years would be equivalent to 0.01 fibre years per cc.

(ii) the median asbestos dust concentration found in UK buildings – approximately 0.005 fibres per cc. (see Table 33) – which over 50 years would be equivalent to 0.25 fibre years per cc.

(iii) the highest asbestos dust concentration found in UK buildings – 0.08 fibres per cc. (see Table 33) – which over 50 years would be equivalent to 4 fibre years per cc.

The number of extra deaths at any other cumulative dose, D, can be obtained using the following formula:

$$\text{excess deaths at D} = \frac{D}{0.01} \times \text{excess deaths at 0.01}$$

The excess deaths are based on the linearly estimated relative risk of lung cancer among the approximately 90% of persons who die after the age of 50 years of whom about 6% die of lung cancer, i.e. the figures in Table 36 represent the additional lung cancer deaths over the $10^6 \times 0.9 \times 0.06 = 54\,000$ that can already be expected among one million births.

R. Peto[156] has recently suggested that the relationship between dose and response for cigarette smoking and lung cancer may not be linear, but rather a higher power of the dose than 1. In particular, in an analysis of the data on British doctors, he found that raising the dose to the power 1.5 fitted the 'cleanest' subgroup of smokers. Although Peto's argument was based on dose and not cumulative dose we have considered a power relationship with cumulative dose out of interest. However, inspection of Figs 11 and 12 suggests that this is unlikely.

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Tables

Table 1 Imports of asbestos to the United Kingdom by fibre type. Percentages calculated with 1946 as base.

	Serpentines		Amphiboles								Total Asbestos	
	Chrysotile		Crocidolite		Amosite		Anthophyllite		All			
Year	Metric tonnes	Per cent	Metric tonnes	Per cent	Metric tonnes	Per cent	Metric tonnes	Per cent	Metric tonnes	Per cent	Metric tonnes	Per cent
1946	50 700	100	1 000	100	2 700	100	—	—	3 700	100	54 400	100
1955	123 000	243	6 800	680	12 300	456	—	—	19 100	516	142 100	261
1965	147 000	290	3 400	340	22 600	837	100	—	26 100	705	173 100	318
1975	120 000	237	—	—	19 200	711	200	—	19 400	524	139 400	256

Source: Asbestos Fibres Importers Committee[122].

Table 2a Breakdown of usage of asbestos fibres in the United Kingdom in 1976[123].

	Chrysotile		Amosite		Anthophyllite		Total	
Usage	Metric tonnes	Per cent	Metric tonnes	Per cent	Metric tonnes	Per cent	Metric tonnes	Per cent
Asbestos cement for building	42 400	32.8	500	3.6	—	—	42 900	30.0
Asbestos cement for pipes	7 000	5.4	1 100	7.9	—	—	8 100	5.7
Fire resistant boards	2 700	2.1	11 800	84.3	—	—	14 500	10.1
Other insulation products	200	0.1	200	1.4	—	—	400	0.3
Jointings and packings	10 000	7.8	—	—	—	—	10 000	6.9
Friction materials	15,600	12.1	—	—	50	50	15 650	10.9
Other textile products	6 300	4.9	—	—	—	—	6 300	4.4
Floor tiles	15 800	12.2	—	—	—	—	15 800	11.0
Plastics and battery boxes	800	0.6	400	2.8	—	—	1 200	0.8
Fillers and reinforcements*	28 400	22.0	—	—	50	50	28 450	19.9
Totals	129 200	100.0	14 000	100.0	100	100.0	143 300	100.0

*including felts, millboards, paper, filter pads for beverages, underseals, mastics, adhesives, coatings, etc.

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Table 2b Estimated usage of chrysotile asbestos in the United Kingdom from 1880-1976 (in metric tonnes).

	1880	1890	1900	1910	1920	1930	1940	1950	1960	1970	1973	1976
1 Asbestos cement – building uses	—	—	—	1 000	9 000	12 500	29 000	42 000	58 500	49 000	55 600	42 400
2 Asbestos cement – pressure and other pipes	—	—	—	—	—	300	4 000	12 000	10 000	8 000	7 800	7 000
3 Fire resistant insulation board	—	—	—	—	—	—	—	500	1 000	4 000	3 000	2 700
4 Other insulation products incls. spray	10	500	2 000	2 600	3 400	3 000	5 000	6 000	5 000	1 700	1 300	200
5 Jointings and packings	60	400	1 400	2 000	3 200	3 200	6 200	6 500	7 000	9 000	11 400	10 000
6 Friction materials	—	—	—	100	1 000	1 500	3 000	6 000	10 000	17 000	18 500	15 600
7 Textile products not in (5) & (6)	10	100	600	1 000	1 500	2 000	4 000	6 000	7 000	7 300	6 800	6 300
8 Floor tiles and flooring	—	—	—	50	300	400	1 000	5 500	12 500	19 000	16 200	15 800
9 Moulded plastics & battery boxes	—	—	—	—	—	—	300	2 000	2 000	3 000	2 200	800
10 Fillers & reinforcement (felts, millboard, paper, underseals etc)	—	—	—	50	1 600	2 100	9 500	16 500	18 000	21 000	25 300	28 400
Total	80	1 000	4 000	6 800	20 000	25 000	62 000	103 000	131 000	139 000	148 100	129 200
Approx. chrysotile tonnage imported	80	1 000	4 600	6 800	25 000	22 500	88 000	111 000	142 000	132 500	171 000	130 000

Sources: Board of Trade Census of Production Returns from 1924: Ministry of Works monthly statistics of Building Materials; TBA production statistics from 1933: Submissions to the Monopolies Commission; and Recollection of long-service officials in some of the principal companies.

Table 3a Cancer of the gastro-intestinal tract and peritoneal mesothelioma in 8 surveys (including 2 studying two different sub-populations) in which an excess of the former has been reported.

Reference	Population	Cases Observed (O)	Cases Expected (E)	O/E	O-E	Peri. mes.	Fibre Type†
Selikoff[124]	Insulators	43	13.6	3.15	29.4 (S)*	25	C, A
Elmes & Simpson[125]	Shipyard workers	15	5.2	2.91	9.8 (S)	0	Cr, C, A
Selikoff[127]	Amosite workers	26	12.5	2.07	13.5 (S)	6	A
Enterline[128]	Maintenance workers	22	13.3	1.65	8.7 (S)	0‡	C, A, Cr
Selikoff[127]	Insulators	61	37.8	1.61	23.2 (S)	63	C, A
Newhouse[130]	Insulation manufacturers	31	20.4	1.52	10.6 (S)	15	C, A, Cr
Mancuso[126]	Production workers	16	10.6	1.51	5.4 (NS)	6?	?
McDonald et al[77]	Thetford miners & millers	165	144.8	1.14	20.2 (NS)	0	C
Enterline[128]	Production workers	37	32.5	1.14	4.5 (NS)	0	C
McDonald et al[77]	Asbestos miners & millers	125	150.4	0.83	−25.4 (NS)	0	C
Peto et al[29]	Textile workers	16	15.7	1.03	0.4 (NS)	0	C, ? Cr

†A=Amosite C=Chrysotile Cr=Crocidolite.
‡It is known that many cases of mesothelioma occurred in younger men in this factory.
*(S)=significant excess ($P<0.05$) (NS)=no significant excess ($P>0.05$).

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Table 3b Observed and expected deaths from cancer and from all other causes in insulation workers and amosite insulation material operatives.

Cause of death	New York/New Jersey Insulation workers*		US and Canadian Insulation workers		Amosite insulation material operatives	
	Obs.	Exp.	Obs.	Exp.	Obs.	Exp.
Total cancer: all sites	189	47.2	459	144.1	143	50.2
Lung cancer	84	10.1	213	44.4	73	11.4
Pleural mesothelioma	8	—	26	—	3	—
Peritoneal mesothelioma	24	—	51	—	4	—
Cancer of stomach			16	6.6	11	4.6
Cancer of colon, rectum	41	13.0	26	17.5	15	7.1
Cancer of oesophagus			13	3.2	0	1.2
All other cancers	32	24.1	114	72.3	37	25.9
All other causes	199	232.8	555	661.5	314	249.3

Source: Selikoff et al, 1973[79].

*20 or more years after first exposure to asbestos.

Table 3c Evidence for an association between asbestos inhalation and laryngeal cancer.

Study	Cases	Controls	Relative risk
Stell and McGill[33]	31/100	3/100	14.5
Insulation workers	19		
Dockers	9	3	
Boiler scalers	3		
Shettigara and Morgan[34]	10/43	0/43	—
Insulation workers	4		
Cement workers	3	0	
Sprayer	1		
Others	2		
Newhouse and Berry[35]	2	0.4*	5.4
Selikoff[36]	9	4.5*	2
McDonald et al[77]	16	15*	1.07

*Expected deaths.

Table 4 Types of exposure to asbestos (adapted from the Zielhuis report).[1]

I Occupational exposure
  (a) direct: (1) Mining* & milling and transporting (2) processing fibre (3) installing, adapting and working with new products (4) repairing and servicing existing products (5) demolition (6) dumping
  (b) indirect
  (c) in agriculture*
II Para occupational exposure
  (a) domestic
  (b) hobbies
III Neighbourhood exposure
IV General environmental exposure
  (a) water
  (b) beverages
  (c) food and drugs
  (d) air

*Not in U.K.

Table 5 Composite data concerning asbestos in urban air in the USA and UK.

Sample site	Asbestos concentration nanograms/m³
New York City†	
Manhattan	25-60
Bronx	25-28
Brooklyn	19-22
Queens	18-29
Staten Island	11-21
Philadelphia, Pa.†	45-100
Ridgewood, N.J.†	20
Port Allegheny, Pa.†	10-30
Rochdale (Factory grounds)	1-10*
Rochdale (Town centre)	10*
Lancashire/Yorkshire	1-10*
Industrial site	10*

†Selikoff et al (1972)[110]

*Chrysotile (Rickards, 1973)[111]

Table 6 Examples of exposure levels in air (Reproduced from Table 10 of IARC Monograph No. 14)[4].

Exposure	Asbestos concentration nanograms/m³	
	USA	Paris
Ambient air (urban)	0.1- 100	0.1- 10
Buildings sprayed with asbestos	0.1- 800	0.1- 800
Near asbestos spraying	10 -1 000	
Near asbestos factory	10 -5 000	10 -3 000

Table 7 Numbers and percentages of malignant lung tumours and mesotheliomas found in rats after inhalation of UICC standard samples of asbestos.

Fibre inhaled	Malignant lung tumours	Mesotheliomas of pleura	Total animals at risk
Amosite	11 (7.5)	1 (0.7)	146
Anthophyllite	16 (11.0)	2 (1.4)	145
Crocidolite	16 (11.3)	4 (2.8)	141
Chrysotile (Canadian)	17 (12.4)	4 (2.9)	137
Chrysotile (Rhodesian)	30 (20.8)	0 (0.0)	144
Controls	0 (0.0)	0 (0.0)	126

Source: (Wagner et al, 1974)[9].

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Table 7X The relationship of pathological findings in rats exposed to equal masses (examples 1, 3 & 5) and equal numbers of fibres (samples 2 & 4) in dusts of different types of asbestos, to the characteristics of the aerosols[157].

Sample	1 Chrysotile	2 Chrysotile	3 Crocidolite	4 Crocidolite	5 Amosite
Number of animals*	40	42	43	40	43
Mean mass concentration (mgm/m³)	9.9	2.0	10.0	4.9	10.0
Mean fibre-number conc. fibres of length > 5 μm	1 950	390	860	430	550
Mean fibre-number conc. fibres of length > 20 μm	360	72	34	17	6
% of fibres of length at least 0.2 μm which are greater than 0.5 μm		30		20	24
Interstitial pulmonary fibrosis after 29 months %	9.2	3.9	1.4	0.8	2.6
Number of malignant lung tumours and mesotheliomas	8	3	0	1	0
Number of benign lung tumours	7	6	1	2	2
All the above tumours	15	9	1	3	2

*No lung tumours or mesotheliomas occurred in 20 control animals.

Table 8 Relationship of incidence of lung cancer and mesothelioma tumours to duration of inhalation of asbestos by fibre type (after Wagner et al 1974)[9].
(Figures in brackets are percentages).

Duration of inhalation	Chrysotile			Amphiboles			All types		
	At risk	Lung	Meso	At risk	Lung	Meso	At risk	Lung	Meso
1 day	87	2 (2.3)	0 (0.0)	132	1 (0.8)	2 (1.5)	219	3 (1.4)	2 (0.9)
3 months	70	6 (8.6)	0 (0.0)	110	2 (1.8)	1 (0.9)	180	8 (4.4)	1 (0.6)
6 months	36	4 (11.1)	0 (0.0)	54	3 (5.6)	0 (0.0)	90	7 (7.8)	0 (0.0)
12 months	50	20 (40.0)	3 (6.0)	79	15 (19.0)	3 (3.8)	129	35 (27.1)	6 (4.7)
24 months	38	15 (39.5)	1 (2.6)	57	22 (38.6)	1 (1.8)	95	37 (38.9)	2 (2.1)
Total	281	47 (16.7)	4 (1.4)	432	43 (10.0)	7 (1.6)	713	90 (12.6)	11 (1.5)

Table 9 Percentages of rats developing mesotheliomas after intrapleural administration of various materials. (Wagner et al, 1977, as reported in IARC Monographs Vol. 14, 1977, p.47)[41].

Material administered	% of rats with mesotheliomas
UICC crocidolite	61
UICC amosite	36
UICC anthophyllite	34
UICC chrysotile (Canadian)	30
UICC chrysotile (Rhodesian)	19
Superfine (SFA) chrysotile (Canadian)	66
Fine glass fibre (diameter=0.12 μm)	12
Ceramic fibre (diameter=0.5-1 μm)	10
Glass powder	3
Coarse glass powder (diameter=1.8 μm)	0

Table 10 Asbestos products and their asbestos contents in EEC countries (from the Zielhuis report)[1].

Asbestos product	Asbestos content %	Asbestos† fibre type
Asbestos cement building products	10- 15	C, A, Cr
Asbestos cement pipes	12- 15	C, Cr, A
Fire-resistant insulation boards	25- 40	A, C
Insulation products, including sprays	12-100	A, C, Cr
Jointings and packings	25- 85	C, Cr
Friction materials	30- 70	C
Other textile materials	65-100	C, Cr
Floor tiles and sheets	5- 7.5	C
Moulded plastics and battery boxes	55- 70	C, Cr
Fillers and reinforcements, etc.	25- 98	C, Cr

†C=chrysotile A=amosite Cr=crocidolite

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Table 11 Mortality from selected causes in the principal published industrial cohorts for which data are available.

Cause of death	No. of deaths	Study 1	2	3	4	5	6‡‡	7	8	9	10	11***
All causes	O	754	3 291	317	350	524	451	1 358	248	98	655	281
	E	657	3 015	249	268	319	305	960		38	527	134
	O-E	97	276	68	82	205	146	398		60	128	147
	O/E	1.15	1.09	1.27	1.31	1.64	1.48	1.41		2.61	1.24	2.09
Cancer of lung	O	58	230	51	63	84	89	275	21	26** (28)††	46	30
	E	22	184	24	24	13	12	56	13	1.6**	19	17
	O-E	36	46	27	39	71	77	219	8	24.4** (26.4)††	27	13
	O/E	2.7	1.2	2.1	2.6	6.3	7.3	4.9	1.7	15.9** (17.1)††	2.4	1.8
G.I. cancer	O	53	276	16	31	26*	43*	61	7	15	36	16
	E	42	272	16	20	13*	14*	38	15	5.2	29	11
	O-E	11	4	0	11	13*	29*	23	8†	9.8	7	5
	O/E	1.3	1.0	1.0	1.5	2.1*	3.2*	1.6	0.5	2.9	1.2	1.5
Mesothelioma	O (ple.)	1	8(10)††	10	9	5	10	29	0	1(4)††	0	1
	O (per.)	0(0.1%)	0(1)††(0.2%)	0(3.2%)	15(6.9%)	6(2.1%)	22(7.1%)	63(6.8%)	0(0.0%)	0(3)††(1.0%)	0(0.0%)	6(2.5%)
Asbestosis	O	18(2.4%)	42(1.3%)	25(7.9%)	12‡(3.4%)	27(5.2%)	37(8.2%)	101(7.4%)	13(5.2%)	9(9.2%)	52(7.9%)	31(11.0%)

Source: 1-Enterline (C, A, Cr)[131, 128]; 2-Quebec (C)[77]; 3-Rochdale (C, ? Cr)[29, 132]; 4-Newhouse (C, A, Cr)[130, 149]; 5-Selikoff (A)[127]; 6-Selikoff N. Y. (C, A)[124]; 7-Selikoff US+Canada (C, A)[127]; 8-Finland (An)[30]; 9-Elmes (Cr, C, A)[125]; 10-Wagoner (Cr)[134]; 11-Mancuso (?)[126].

O=Observed E=Expected.

*Stomach+colon+rectum †abdomen ‡based on later data ††only includes men 20+ years in industry **including larynx

††figures in brackets are after revision of death certificate diagnosis

***observed deaths in men with 2+ years employment; expected deaths based on experience of men with less than 2 years employment.

Types of fibre to which the men were exposed are shown in brackets.

N.B. This table is an overall summary of mortality experience in each study and does not for example consider relative dust exposure of the different populations. The problems of comparability are discussed in the text, e.g. p. 22.

Table 12 Radiological abnormalities on chest radiographs among 326 household members of amosite asbestos workers[18].

Radiological abnormality	Number with abnormality
Pleural thickening only	42 (13%)
Pleural calcification only	7 (2%)
Pleural thickening and/or calcification	3 (1%)
Irregular opacities only	35 (11%)
Irregular opacities, pleural thickening and/or calcification	27 (8%)
Any abnormality	114 (35%)

(Figures in brackets are percentages)

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Table 13 Production of asbestos (thousands of tons) in South Africa.
(Quarterly Reports on Minerals, Republic of South Africa). After Harington, Gilson & Wagner, 1971[54].

Period	Amosite	Transvaal Crocidolite	Cape Crocidolite	Chrysotile
1928-35	35	Nil	31	Not available
1936-43	118	13	49	Not available
1944-51	237	48	85	Not available
1952-59	446	126	245	Not available
1960-68	639	83	306	Not available

Table 13X Proportional mortality from malignant disease in a cohort of 11 391 Canadian chrysotile miners and millers and in a cohort of 199 persons who worked on crocidolite gas-mask filters[55].

	Chrysotile miners and millers	Crocidolite gas-mask filter workers
Total cohort	11 379 96% male	199 55% male
Traced	10,259	174 (1976)
Dead	4 247 (since 1935)	56
Malignant disease	885 21%	22 39%
Cancer of the lung or bronchus	242 6%	7 13%
Mesothelioma	11* 0.26%	9** 16%

* 8 certified, one of which was not thought by the pathologist to be a mesothelioma. 3 diagnosed at autopsy.
**4 certified. 2 diagnosed at autopsy. 3 based on pathologists diagnosis.
2 cases were common to both series.

Table 14 Mesothelioma cases and controls by amount and type of fibre.

Amount of fibre	Type of fibre*	Mesothelioma cases	Controls	Relative risk
Nil	—	9	70	1
Low†	C	23	33	5.4
	A	10	21	3.7
	C+A	6	3	15.6
	A+C	9	1	70.0
High†	C	4	2	15.6
	A	27	3	70.0
	C+A	15	2	58.3
	A+C	17	0	—
Total		120	135	

Source: Pooley (1973)[64].
†Low = Pooley's category 5 High = categories 1-4.
*C = Chrysotile A = Amphibole C+A = mixture, chrysotile predominant A+C = mixture, amphiboles predominant.

Table 15 Mesothelioma cases and controls by type of fibre in lung tissue.

Type of fibre*	Mesothelioma cases	Controls	Relative risk
Nil	9	70	1
C	27	35	6.0
A	37	24	12.0
C+A	21	5	32.7 } 60.9
A+C	26	1	202.2 }
Total	120	135	

Source: Pooley (1973)[64].
*C = Chrysotile A = Amphiboles C+A = mixture, chrysotile predominant A+C = mixture, amphiboles predominant.

Table 16 Thirty-five collected cases of mesothelioma associated with domestic exposure to asbestos.

Author	Number	Relationship to contact	Activity of contact	Fibre types*
Newhouse & Thompson[23]	9	3 sisters 5 wives 1 mother		Mixtures†
Rusby[113]	1	Unknown	Navy	Mixtures
Ashcroft & Heppleston[114]	1	Wife	Asbestos wkr.	Unknown
Champion[115]	1	Son	Pipe lagger in Glasgow	Mixtures
Greenberg & Davies[69]	2	Brother Wife	Asbestos factory	Unknown
Lieben & Pistawka[105]	3	Daughter Daughter Mother	Ceramics Insulator Insulator	C, A Mixtures Mixtures
McDonald & McDonald[101]	5	1 son 2 daughters 2 wives	Miner 1 Miner 1 Insulator 2 Insulators	C C Mixtures Mixtures
Rubino et al[116]	3	1 sister 2 wives	Asbestos cement wkr. Asbestos wkrs.	Unknown
Milne[117]	1	Daughter	Asbestos wkr.	
Bittersohl & Ose[118]	1		Unavailable	Unknown
McEwen et al[72]	1			Mixtures
Knappmann[119]	1			Unknown
Anderson[18]	4	2 daughters 1 sister in law 1 son	Insulation mfrs.	Amosite Amosite Amosite
Heller et al[120]	1	Wife	Pipe fitter	Mixtures
Lillington et al[137]	1	Wife	Asbestos wkr.	Unknown
Total	35			

†i.e. Mixtures containing chrysotile and amphiboles *C = Chrysotile A = Amosite.

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Table 17a Malignant mesothelioma, Canada 1966-72, USA 1972, by site and acceptance of diagnosis by panel (from McDonald and Becklake, 1975)[66].

Place	Cases	Pleural %	Diagnosis accepted %	Rates per million Gross	Rates per million Corrected
Canada Quebec	104	75	37	2.5	0.9
Remainder	115	70	61	1.1	0.7
USA	245	73	69	1.2	0.8

Table 17b Geographical distribution of deaths from mesothelioma within Province of Quebec, 1969-72. Populations given in brackets.
(Theriault and Gilbert: unpublished data)[67].

Location	Number of cases Observed	Expected
Mining area (115 270)	2	1.3
Districts adjacent to mining area (419 505)	5	4.8
Rural areas (2 879 705)	12	32.4
Quebec City (423 165)	7	4.7
Montreal (2 187 155)	42	24.4

Table 18 Ratios of observed to expected cases of mesothelioma based on Canadian incidence as a standard (from McDonald and Becklake, 1975)[66].

Countries		Cities			
Canada	1	Nantes	7	Trieste	8
GDR	1	Dresden	17	Haarlem	7
Britain	2	Hamburg	7	Rotterdam	7
Netherlands	2	Wilhelmshaven	22	Walcheren	23
Sweden	1	Clydeside	6	Malmo	7
USA	1	Merseyside	5	Manville (NJ)	27
		Plymouth	14		

Table 19 Distribution of 231 male cases of mesothelioma and 226 controls in Canada and USA according to occupation (after McDonald and McDonald, 1977, Table 2)[70].

Occupation	Cases	Controls	Relative risk
Insulation workers	22	1	43.1
Plumbing and heating	48	17	5.5
Asbestos factory workers	16	6	5.2
Shipyard workers	45	15	5.9
Construction workers	60	46	2.6
Miscellaneous*	45	14	6.3
Occupations not showing an excess	75	151	1.0

*Welding, lathing, sheet-metal, paint, rubber products, dry cleaning and paper work.

Table 20 Death certificates mentioning mesothelioma, etc., Great Britain, 1967-1976.

Year of death	Mesothelioma	Lung cancer with mention of asbestosis or asbestos exposure	All certificates mentioning asbestosis
1967	88	34	60
1968	153	25	81
1969	159	25	77
1970	193	24	80
1971	176	32	78
1972	208	40	103
1973	218	41	102
1974	221	41	120
1975	256	49	163
1976	292	—	—

Source: Health and Safety Executive, 1977.

Table 21 Relative risk of death from respiratory cancer by type of fibre, corrected for differences in cumulative dust exposure (in million particles per cu. ft. years). After Enterline and Henderson (1973)[135].

Type of fibre	Respiratory cancer deaths	Average cumulative dust exposure	Relative risk adjusted for cumulative dust exposure
Amosite only	4	330	3.9
Chrysotile only	39	244	2.4
Amosite and chrysotile	3	266	1.7
Chrysotile and crocidolite	10	209	5.6
Amosite, chrysotile and crocidolite	2	273	2.4
Any amosite	9	286	2.5
Any chrysotile	54	244	2.6
Any crocidolite	12	229	4.4

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Table 22 Occurrence and average annual incidence of newly diagnosed disease related to dust exposure in men in Rochdale from 1966 to last follow-up by time of first employment.

Cumulative Dose	Crepitations				Possible asbestosis			cu	Certified asbestosis			
	Number of men	Cases	%	Annual %	Number of men	Cases	%	Annual %	Number of men	Cases	%	Annual %
(a) Men first employed before 1951												
<50	8	2	25	4.7	8	1	13	2.1	8	0	0	0
50–	27	6	22	3.6	26	5	19	3.1	27	2	7	0.9
100–	39	10	26	4.2	39	6	15	2.5	40	3	8	0.9
150–	40	8	20	3.1	43	10	23	3.8	46	5	11	1.4
200–	23	9	39	7.5	24	8	33	6.2	29	6	21	2.7
250+	21	8	38	6.3	22	6	27	4.4	29	5	17	2.4
Total	158	43	27	4.5	162	36	22	3.7	179	21	12	1.5
(b) Men first employed after 1950												
<50	83	6	7	1.1	83	2	2	0.4	84	1	1	0.1
50–	79	11	14	2.2	77	5	6	1.0	81	6	7	0.9
100–	28	6	21	3.4	30	4	13	2.0	32	3	9	1.1
Total	190	23	12	1.9	190	11	6	0.9	197	10	5	0.5
(c) All men in study												
<50	91	8	9	1.3	91	3	3	0.5	92	1	1	0.1
50–	106	17	16	2.5	103	10	10	1.5	108	8	7	0.9
<100	197	25	13	2.0	194	13	7	1.0	200	9	5	0.5
100–	67	16	24	3.9	69	10	14	2.3	72	6	8	1.0
150–	40	8	20	3.1	43	10	23	3.8	46	5	11	1.4
200–	23	9	39	7.5	24	8	33	6.2	29	6	21	2.7
250+	21	8	38	6.3	22	6	27	4.4	29	5	17	2.4
Total	348	66	19	3.0	352	47	13	2.1	376	31	8	0.9

Based on Berry, G. Personal communication to the authors, shown as Appendix 3.

Table 23a The frequency of various parameters of asbestos related lung and pleural disease in Quebec miners according to cumulative dose.

	Dust index in millions of particles per cubic foot-years					
	<10	10—	100—	200—	400—	800—
Prev. of radiol. ch. in men 56-65 yrs. %						
Small irreg. opac.*						
Thetford	—	7	10	13	21	34
Asbestos	10	6	17	23	15	20
Any pleural changes*						
Thetford	23	20	33	29	34	39
Asbestos	5	15	18	14	15	24
Prevalence %** dyspnoea	7	18	23	26	30	37
Decrease in lung*** function						
VC	0	—4	—9	—11	—14	—15
FEV	0	—4	—7	—10	—13	—14
DL CO at rest	0	—3	—6	— 5	— 9	—11

Source: Becklake (1976) Table 8[2].

* standardised for age and years of employment

** age standardised

***age and height standardised

Table 23b Dose-response relationship between deaths from asbestosis (pneumoconiosis) and dust in Quebec miners and millers. Based on McDonald et al[77].

Cumulative dose (mp/cf-y)	Observed (O)	Expected* (E)	O E	Relative risk
0—	3	1.21	2.5	1
10—	2	0.47	4.3	1.7
30—	2	0.60	3.3	1.3
100—	10	0.51	19.6	7.9
300—	7	0.26	26.9	10.9
600+	20	0.24	83.3	33.6
Total	44	3.29	13.4	—

*External comparison.

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Table 24 Observed and expected deaths and standardised mortality ratios from lung cancer by cumulative asbestos dust exposure in retired asbestos workers.

Asbestos dust exposure	Production workers			Maintenance service workers			Total		
	O	E	SMR	O	E	SMR	O	E	SMR
Under 125	11	7.5	146.7	7	3.2	218.8	18	10.7	168.2
125 – 249	7	3.8	184.2	4	1.1	363.6	11	4.9	224.5
250 – 499	8	3.6	222.2	8	1.8	444.4	16	5.4	296.3
500 – 749	0	1.1	0.0	9	0.7	1285.7	9	1.8	500.0
750+over	3	0.7	428.6	2	0.2	1000.0	5	0.9	555.6
All	29	16.7	173.7	30	7.0	428.6	59	23.7	248.9

Source: Enterline et al (1972)[128].

O = Observed deaths E = Expected deaths SMR = Standardised Mortality Ratio = (O/E) × 100
Asbestos dust exposure measured in million particles per cubic foot-years.

Table 25 Observed and expected deaths and ratios from lung cancer by cumulative asbestos dust exposure in Quebec miners and mill workers.
Based on McDonald et al[77].

Asbestos dust exposure over 20 years since start of service	Lung cancer deaths			
	O	E	O/E × 100	
Under 3	42	43.3	97	} 93
3—	25	25.8	97	
10—	24	28.5	84	
30—	43	36.7	117	
100—	38	31.8	119	
300—	29	16.6	175	
600+over	41	14.6	281	
All	242	197.3	123	

O = Observed deaths E = Expected deaths.
Asbestos dust exposure measured in million particles per cubic foot-years.

Table 26 Cumulative asbestos dust exposure for lung cancer deaths and controls (5 per case) in Quebec miners and mill workers.

Asbestos dust exposure up to 7 years before death of case	Lung cancer deaths (cases)	Controls	Relative risk
Under 6	43	285	1.00
6—	10	62	1.07
10—	24	166	0.96
30—	37	211	1.16
100—	31	168	1.22
300—	27	95	1.88
600—	18	50	2.39
1 000—	10	19	3.49
1 500—	6	8	4.97
2 000+over	9	11	5.42
All	215	1 075	—

Source: Liddell et al[129].

Asbestos dust exposure measured in million particles per cubic foot-years. Relative risks are calculated relative to the lowest exposure group.

Table 27 Observed and expected lung cancer deaths in asbestos factory workers in Barking by duration of employment and severity of exposure to asbestos.

Type of exposure to asbestos	Duration of employment	Length of follow-up in years									
		10–14		15–19		20–24		25+		Total	
		O	E	O	E	O	E	O	E	O	E
Males	Under 2 years	1	1.8	1	1.9	1	1.2	2	1.2	5	6.1
Low to	More than 2 years	3	1.6	2	1.6	2	0.9	1	0.7	8	4.8
moderate	Under 2 years	2	1.5	6	1.8	3	1.3	5	2.0	16	6.6
Severe	More than 2 years	3	2.1	15	2.0	9	1.2	7	1.1	34	6.4
Total		9	7.0	24	7.3	15	4.6	15	5.0	63	23.9

		O	E
Females	Any period	2	0.3
Low	Under 2 years	3	1.0
Severe	More than 2 years	11	0.5
Total		16	1.8

Source: Newhouse (1973)[31,130].

O – Observed deaths E – Expected deaths.

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Table 28 Observed to expected ratios for death from lung cancer 30 years after onset of work in an amosite factory. Men recruited during the years 1941-5.

Length of time worked	Number of men	Observed to expected ratio of deaths after 30 years
<1 month	65	2.5
1 month	101	2.4
2 months	101	3.5
3-5 months	165	1.8
6-11 months	148	3.4
1 year	130	5.1
2+ years	199	7.2

Source: Seidman et al[78].

Table 29 Relationship of dust and smoking habits to the relative risk of lung cancer in Quebec miners and millers.

Smoking	Dust	Observed (O) deaths	Expected (E) deaths	$\frac{O}{E}$	Relative risk
Low	Low	31	71.46	0.43	1.0
Low	High	35	30.38	1.15	2.7
High	Low	67	61.85	1.08	2.5
High	High	58	27.31	2.12	4.9
Total		191	191	1	

Based on Lidell et al[129].

Table 30 The effect of smoking on the risk of death from lung cancer in New York insulation workers.

Type of smoker	Observed*(O) lung cancer deaths	Expected*(E) lung cancer deaths	Number of men (n)	Additive (1) model $E + E \times \frac{n}{A}$	Multiplicative (2) model $E \times \frac{r}{A}$
Non	0	0.05	48	2.8	0.4
Pipe/cigar	0	0.13	39	2.3	1.0
Cigarettes	24	2.98	283	18.9	22.6
Total	24	3.16	370	24	24

Sources: Selikoff et al, 1968, and Doll, 1971[80,81].

*Observed deaths in 52 month period.
†Taking account of age, smoking habits from a) U.S. national rates, b) prospective study of smokers by American Cancer Society: non-occupational expectations.

1. Additive model

$$E_A = \frac{24 - 3.16}{370} = 0.0563 \text{ per man per 52 months}$$

e.g. Never smoked: occupational expectation
$= 0.05 + 0.0563 \times 48 = 2.8$, etc.

2. Multiplicative model

$$r_A = \frac{24}{3.16} = 7.595$$

e.g. Cigarette smokers: occupational expectation
$= 2.98 \times 7.595 = 22.6$ etc.

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Table 31 Mesothelioma deaths in asbestos factory workers in Barking.

Type of exposure to asbestos	Duration of employment	Deaths	Rate per 100 000 subject years*
Males			
low to	Under 2 years	3	31
moderate	More than 2 years	5	83
Severe	Under 2 years	10	77
	More than 2 years	13	195
Total (males)		31	88
Females			
low to	Under 2 years	0	—
moderate	More than 2 years	0	—
Severe	Under 2 years	9	106
	More than 2 years	5	126
Total (females)		14	97

Source: Abstracted from Newhouse and Berry (1976)[63].

*Can be regarded as 'rate per 100 000 per annum'.

Table 31X Cumulative asbestos dust exposure for mesothelioma cases and controls (4 per case) in Quebec miners and mill workers.

Asbestos dust exposure*	Mesothelioma cases	Controls	Relative risk†
Under 6	1	8	1
6—	0	9	
30—	3	5	5.3
100—	2	11	
300+	4	7	9.7
1 000+	0	0	
All	10	40	

Source: McDonald et al[77].

*Asbestos dust exposure measured in million particles per cubic foot-years.
†Relative risks are calculated relative to the lowest exposure group.

Table 32 Conversion of fibres of asbestos > 5 μm to nanograms according to various authors[84,85,107,108,109]. Equivalent to 2 fibres per cc (expressed in nanograms/cu.m) and to 100ng/cu.m. expressed in fibres/cc also shown.

Source	Fibres > 5 μm per ng	Equivalent in ng/cu.m. to 2 fibres/cc	Equivalent in fibres/cc to 100 ng/cu.m.
Bruckman and Rubino (1975)	20	10^5	2×10^{-3} (0.1%)*
Wagg (1977)	20	10^5	2×10^{-3} (0.1%)
Dement et al (1975)	400	5×10^3	4×10^{-2} (2.0%)
Lynch and Ayer (1966)	5	4×10^5	5×10^{-4} (0.025%)
Nicholson (1975)	52	3.8×10^4	5.2×10^{-3} (0.26%)

*Figures in brackets show the equivalent as a per cent of the current hygiene standard of 2 fibres/cc.

Table 33 Asbestos dust concentrations in UK buildings according to (a) materials used, (b) type of building (Byrom and colleagues, 1969)[88].

	Type of material				Total	
Fibres per cm^3	Insulation board	Asbestos cement sheeting	Sprayed asbestos	Other*	No.	%
0–0.005	17	2	7	8	34	46
>0.005–0.01	8	3	5	0	16	22
>0.01–0.02	6	2	2	0	10	14
>0.02–0.03	2	1	3	0	6	8
>0.03–0.04	2	0	0	0	2	3
>0.04–0.05	2	0	0	0	2	3
>0.05–0.08	2	0	1	0	3	4

*compressed flat sheets, partition board and low density panels

Fibres per cm^3	Hospital	Education	Factory or storage	Office	Shop	Place of assembly	Residence	Misc.	Total No.	%
0–0.005	4	15	3	6	1	2	3	0	34	46
0.005–0.01	1	3	2	2	4	2	1	1	16	22
0.01–0.02	1	0	4	0	1	1	2	1	10	14
0.02–0.03	2	0	2	0	0	0	2	0	6	8
0.03–0.04	0	1	0	1	0	0	0	0	2	3
0.04–0.05	0	0	0	1	0	0	1	0	2	3
0.05–0.08	0	0	0	2	0	0	0	1	3	4

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Table 34 Assessment of asbestos materials in buildings as related to susceptibility to damage during normal in situ use. (Evidence of Royal Institution of Chartered Surveyors to ACA)[5].

Degree	Material
Low	Asbestos roofing cement, AC roof decking, cladding, gutters, flue pipes, drainpipes, vinyl asbestos floor tiles, paint.
Moderate	AC ventilation ducts and duct linings, asbestos insulation board partitions and ceilings, firebreaks.
High	Asbestos spray to walls, steel work, pipes, preformed asbestos insulation to pipes, machinery, etc., asbestos insulation board for acoustic purposes, asbestos ducts, asbestos rope and fillers, asbestos fibre in plugs.

Table 35 Dust concentrations in fibres/c.c. that would produce shown excess mortality from lung cancer after 50 years exposure according to linear dose-response models.

Study	Fibre type*	Conversion factor†	Dust levels (fibres/cc) for stated excess mortality from lung cancer 0.1%	0.5%	1%	2%
Quebec	C	5	0.5	3	5	11
		2	0.2	1	2	4
		1	0.1	0.5	1	2
Enterline (production workers)	C	5	0.3	1	3	5
		2	0.1	0.5	1	2
		1	0.1	0.3	0.5	1
Enterline (maintenance workers)	C,A,Cr	5	0.1	0.4	0.8	2
		2	0.04	0.2	0.3	0.6
		1	0.02	0.1	0.2	0.3
		—	0.04	0.2	0.4	0.8
Rochdale	C,?Cr	2	0.08	0.4	0.8	1.6
		5	0.2	1	2	4

* C=Chrysotile A=Amosite Cr=Crocidolite
† For Quebec[129] and Enterline[128] studies figures are ratios of fibres per c.c. to million particles per cubic foot. A multiplication of dust levels by one to two times would further convert to personal as compared to static sampling (see Table 37).
For Rochdale conversion factors are the lowest and highest shown in Table 37 to convert to modern techniques with personal sampling.

Table 36 Number of excess deaths from lung cancer among one million people that might be caused by the highest observed environmental asbestos dust levels in ambient air and the median or highest levels encountered by Byrom in buildings if they were exposed continuously for 50 years.

Study	Fibre† type	Conversion† factor	Number of extra deaths from lung cancer among 10^6 births from stated exposure to asbestos in fibre years per cc. 0.01*	0.25‡	4**
Quebec	C	5	0.2	5	81
		2	0.5	12.5	203
		1	1.0	25	405
Enterline (production workers)	C	5	0.4	10	162
		2	1.0	25	405
		1	2.0	50	810
Enterline (maintenance workers)	C, A, Cr	5	1.4	34	540
		2	3.4	84	1 350
		1	6.8	169	2 700
Rochdale	C, ? Cr	—	2.7	68	1 080
		2	1.4	34	540
		5	0.5	14	216

† See Table 35.
* 10 nanograms per cubic metre for 50 years taken to be equivalent to 0.01 fibres years per cc.
‡ 0.005 fibres per cc. for 50 years is equivalent to 0.25 fibre years per cc.
**0.08 fibres per cc. for 50 years is equivalent to 4 fibre years per cc.

Table 37 Conversion factors for measurements of asbestos fibre concentrations.

Source	Modern counting techniques (M)/ Old counting techniques (O)	Personal sampling (P)/ Static sampling (S)	MP/ OS
Holmes[160]	2:1	1+:1	2+:1
Steel[161]	2.5:1	2:1	5:1

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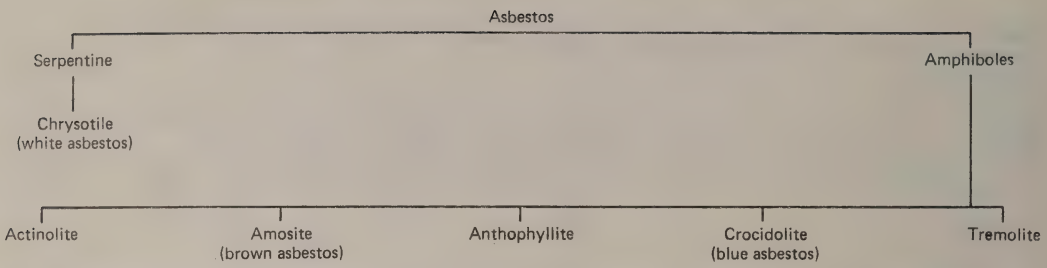


Fig 1 Types of asbestos. IARC Monograph.[4]

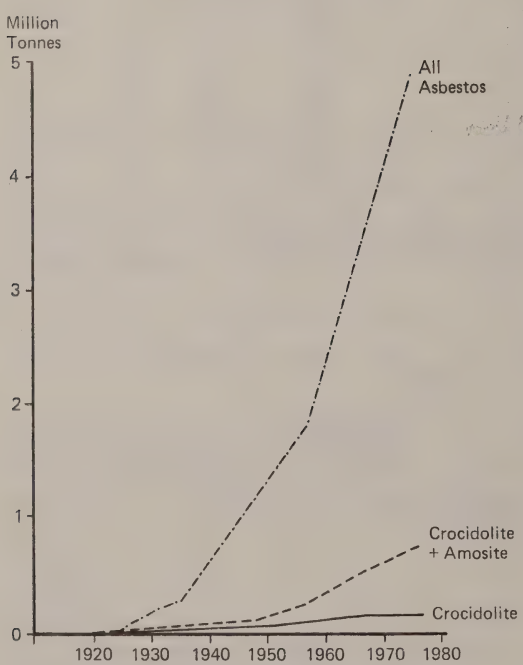


Fig 2(a) Cumulative imports of asbestos to UK by fibre type.[5]

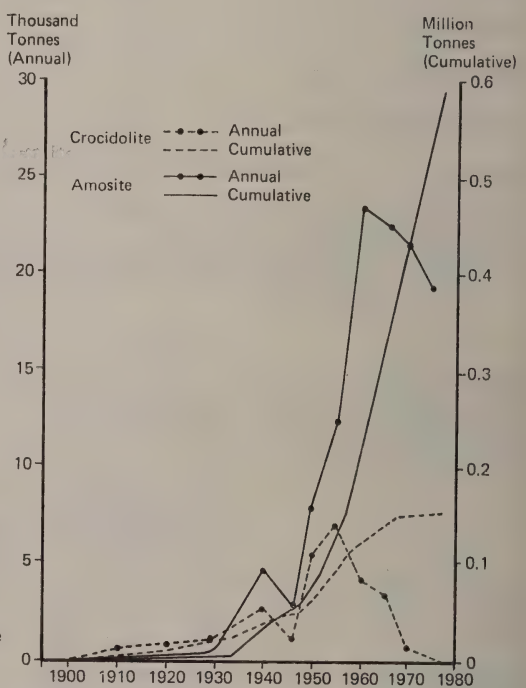


Fig 2(b) Annual and cumulative imports of amosite and crocidolite.[5]

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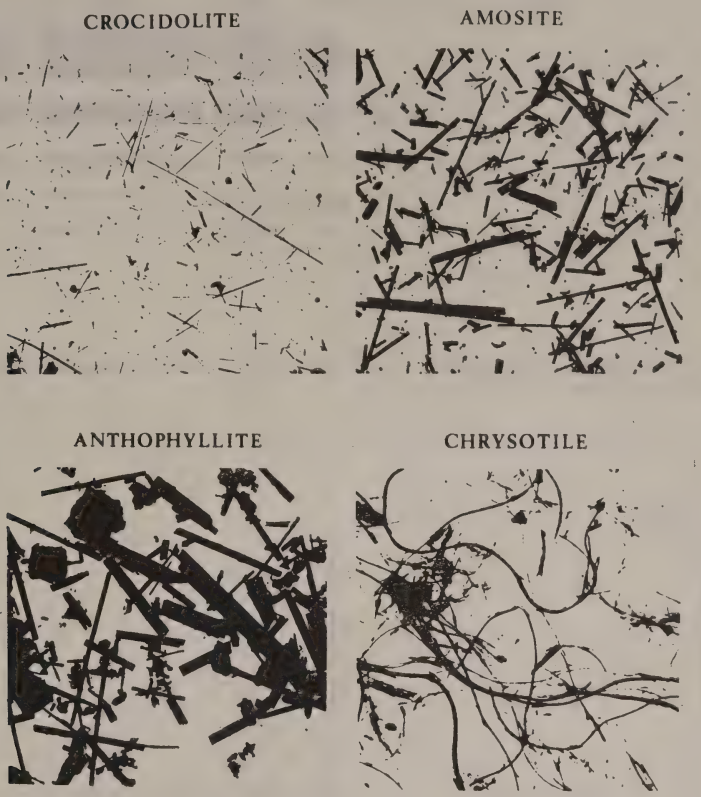


Fig 3 Appearance of asbestos fibres under the electron microscope (10 μm). (Timbrell 1973)[8].

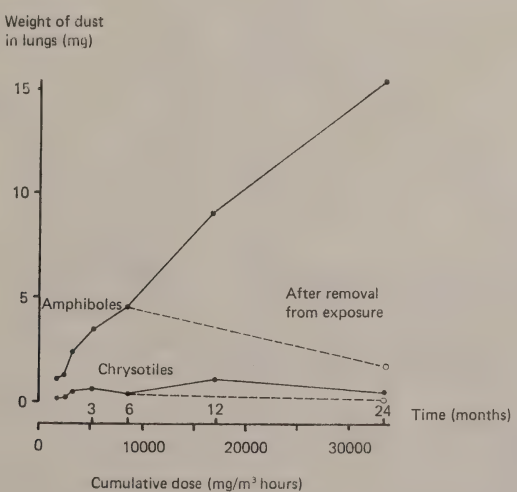


Fig 4 Contrast between the effects of inhalation of amphiboles and chrysotile in rats.
Source: Wagner et al (1974)[9].

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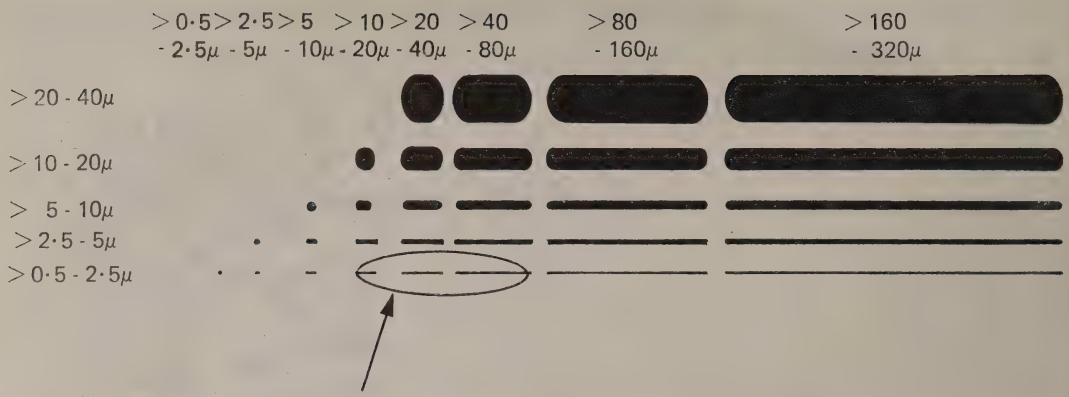


Fig 5 Graphic illustration of mean dimensions of fibres used in Stanton's experiments adapted to show fibres exhibiting carcinogenicity. (Stanton 1973).[12]

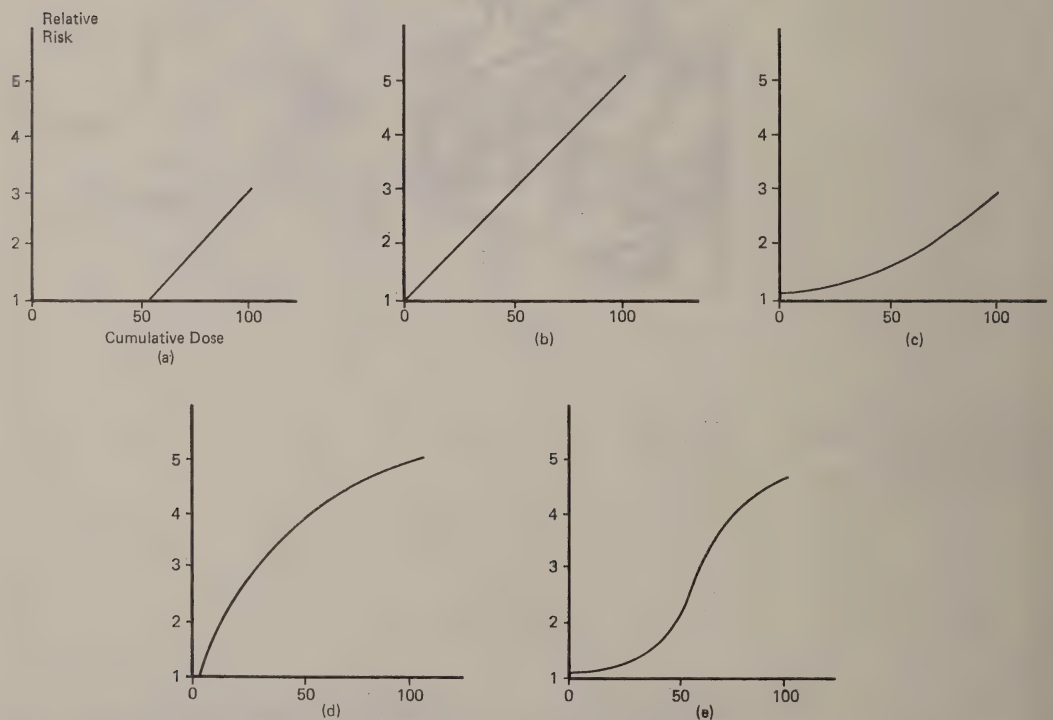
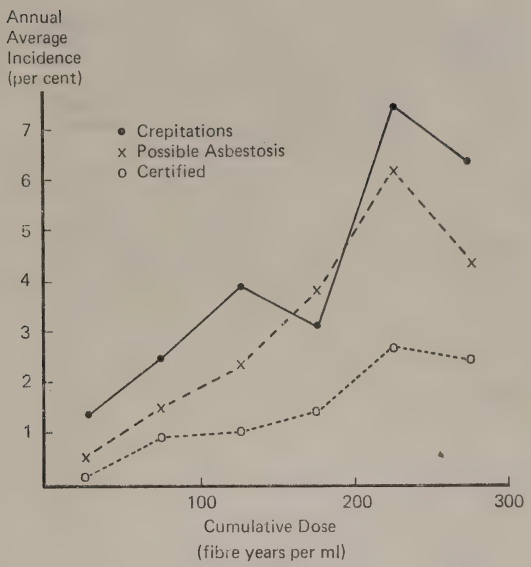


Fig 6 Diagrammatic representations of various different relationships between response and dose. (a) threshold (b) (recti)linear (c) quadratic (d) sublinear (e) cumulative normal.

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Fig 7 Average annual incidence rates (per cent) of "asbestosis" defined in various ways according to cumulative dust exposure.



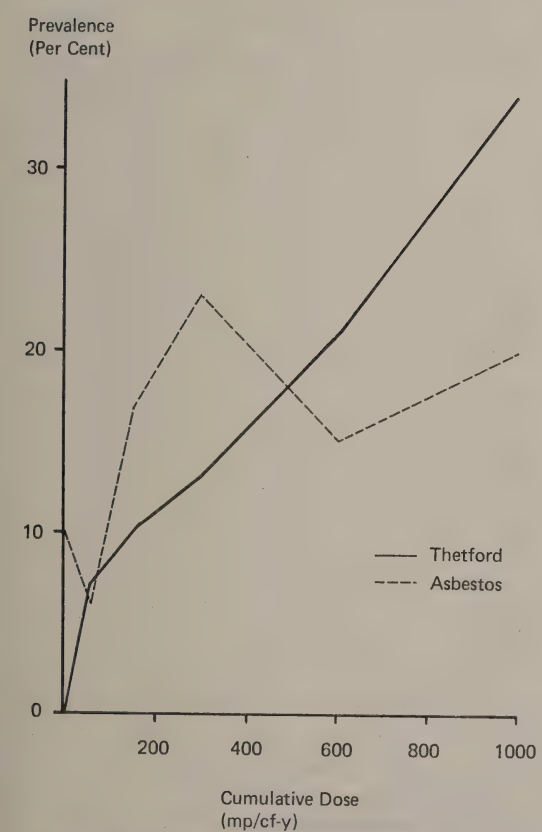


Fig 8(a) The prevalence in Quebec chrysotile miners and millers in the Thetford and Asbestos areas of irregular parenchymal opacities, by cumulative dose of dust.
Source: [2,66].

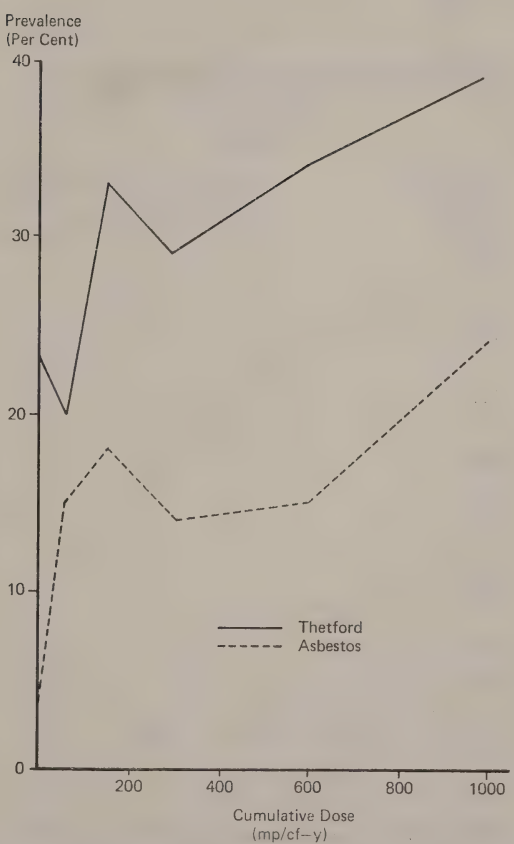


Fig 8(b) The prevalence in Quebec chrysotile miners and millers in the Thetford and Asbestos areas of pleural changes, by cumulative dose of dust.
Source: [2,66].

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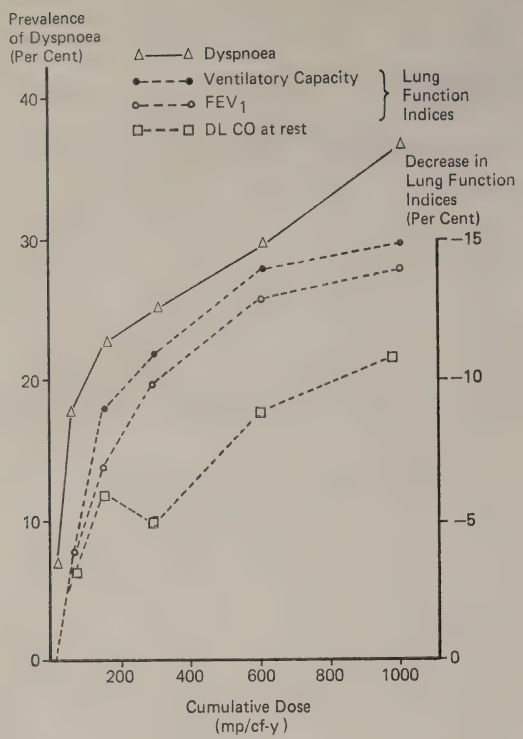


Fig 9 Relationship of various parameters of lung impairment to dose in Quebec miners and millers. *Source:* Becklake 1976.[2]

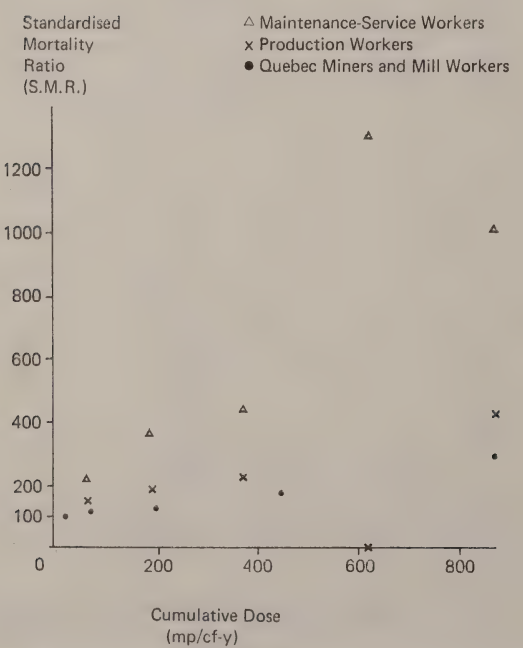


Fig 10 Dose-responses for lung cancer plotted from material in Tables 24 and 25. External comparisons.[128,77]

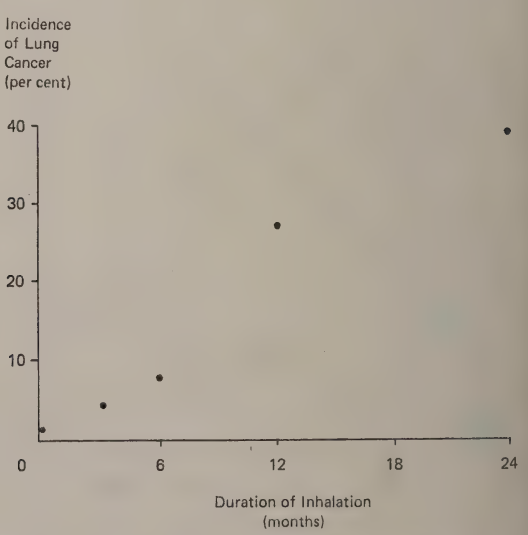


Fig 10(a) Relationship between incidence of lung cancer and duration of inhalation of asbestos in rats. *Source:* Wagner et al.[9]

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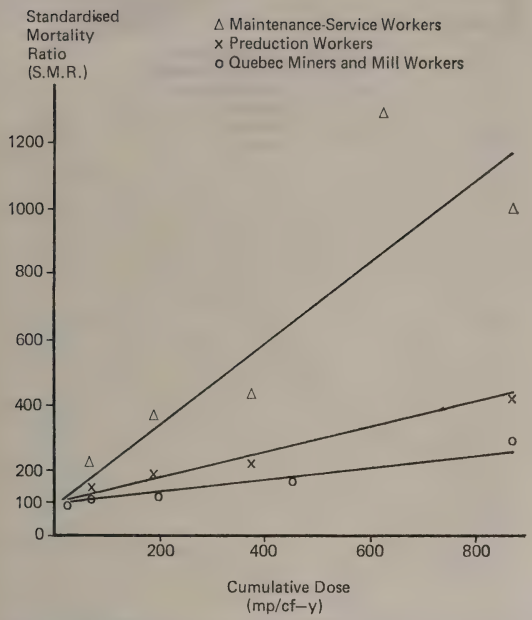


Fig 11 Dose responses for lung cancer as a in Fig 10 but with freehand lines drawn.

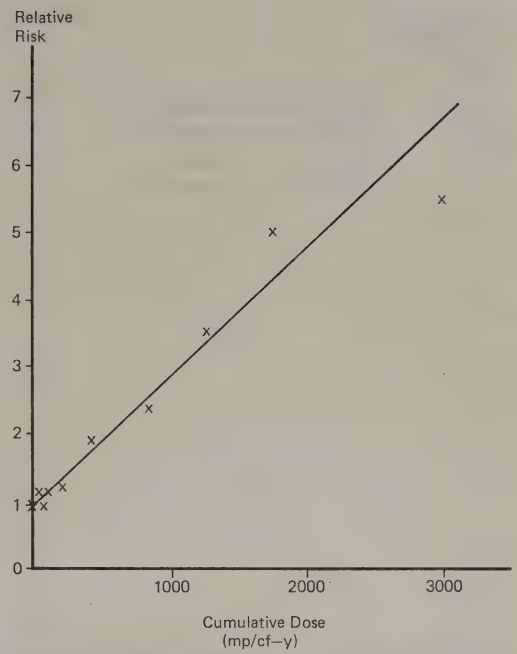


Fig 12 Dose response relationship for lung cancer for Quebec miners and millers. Internal comparison.[129]

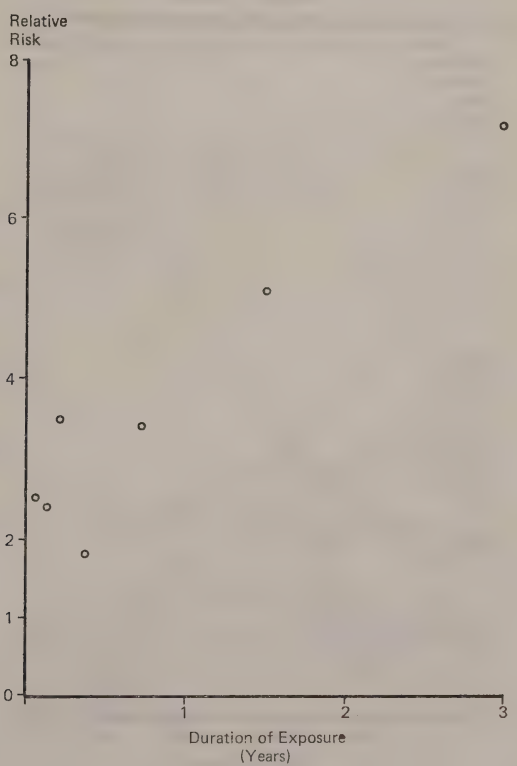


Fig 13 Relative risk of death from lung cancer in a group of amosite insulation workers by duration of exposure. (After Seidman et al).[78]

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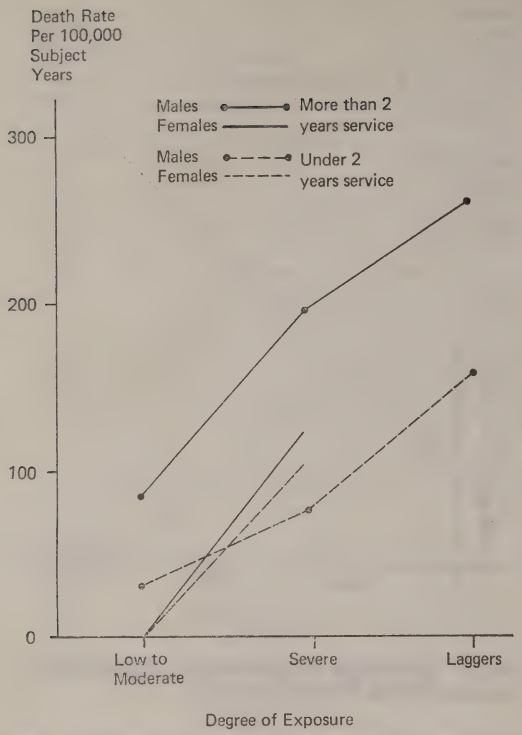


Fig 14 Dose response relationship for mesothelioma.[31,36]

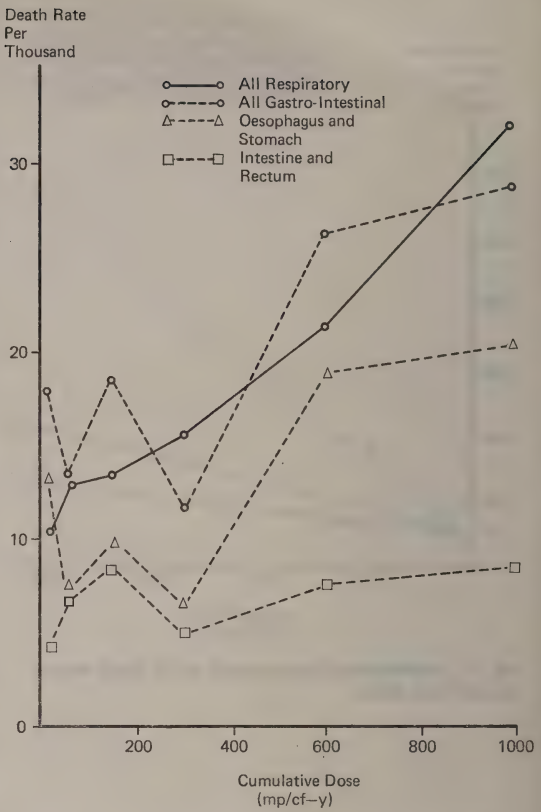


Fig 15 Mortality from G.I. cancer and dust in Quebec miners and millers.
Source: [136].

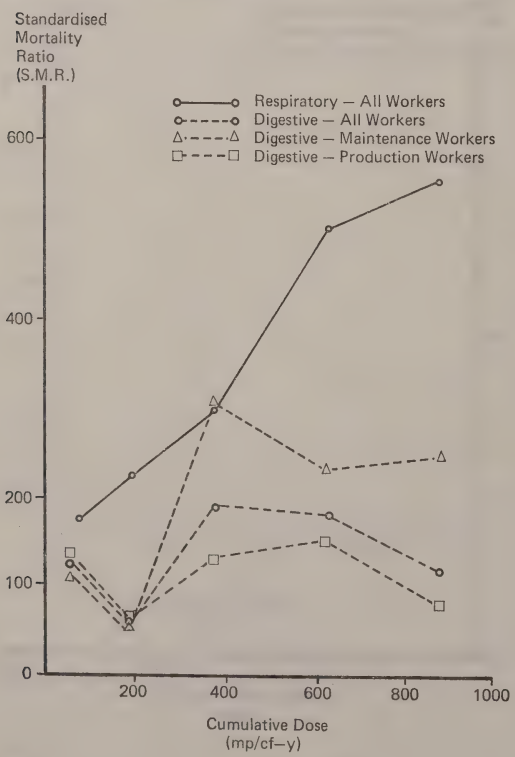


Fig 16 Mortality from G.I. cancer and dust in retired production and maintenance workers.
Source: Enterline 1972.[128]

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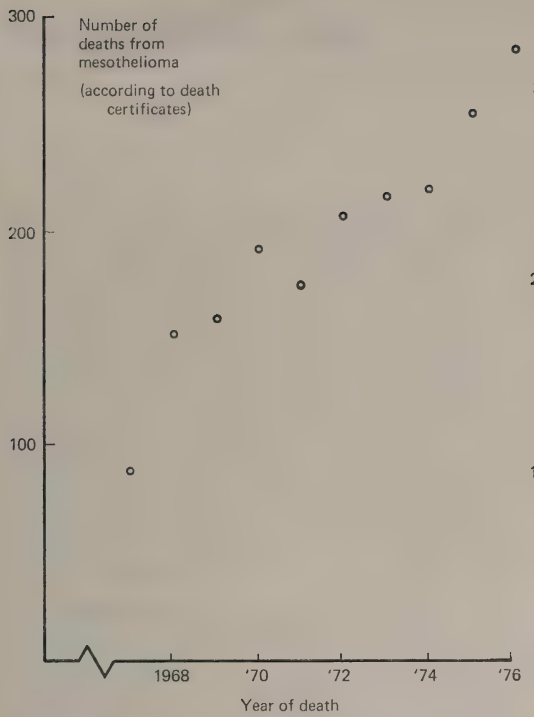


Fig 17 Death certificates mentioning mesothelioma. Great Britain, 1967-1976 by year.
Source: Table 20.

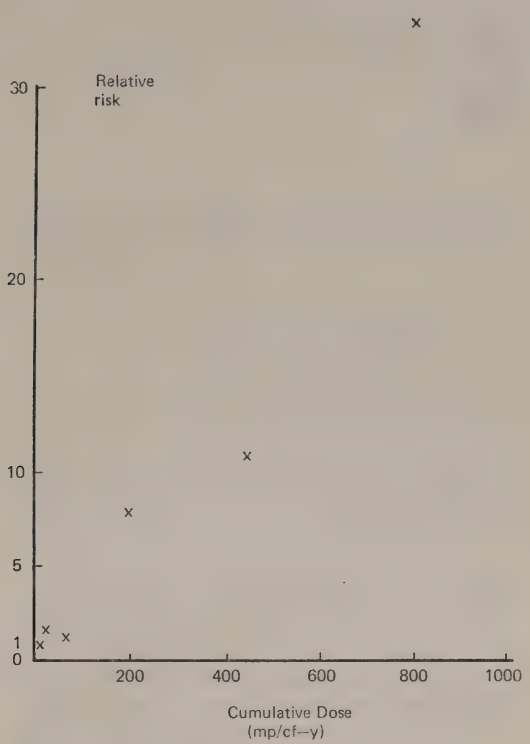


Fig 18 Dose-response relationship between deaths from asbestosis (pneumoconiosis) and dust in Quebec miners and millers.[77]

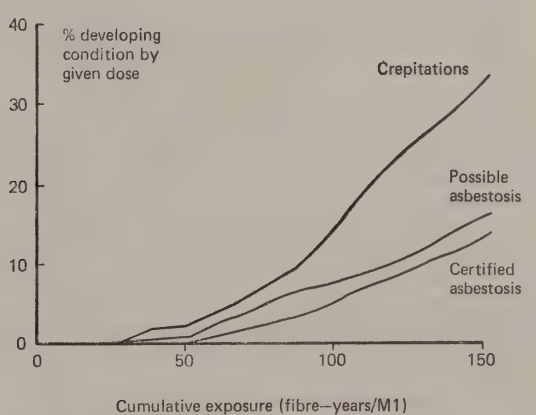
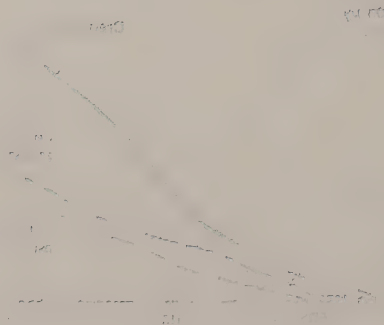


Fig 19 The relationship between the percentage developing crepitations, possible asbestosis and certified asbestosis, and cumulative exposure to asbestos, for men first employed after 1950.
Taken from Berry, G (1977).[13]

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Asbestos control limits

J Steel

2

1 In 1968 the British Occupational Hygiene Society[1] published a hygiene standard for airborne chrysotile dust which was derived from data on two groups of men working in an asbestos textile factory. By relating the earliest demonstrable effects of asbestos on the lung to accumulated exposures to fibres greater than 5 μ in length, the control unit was set at a dose considered to impose a risk of not more than 1 % of developing basal rales. This dose, estimated at 112 fibres/cm^3, implies an atmospheric concentration of about 2 fibres/cm^3 to afford the stated degree of protection for exposure over a working lifetime of 50 years, although it was pointed out that to ensure a 95 % level of confidence that the risk did not exceed 1 %, the concentration would have to be no more than 1 fibre/cm^3.

2 The BOHS limit was quickly adopted by HM Factory Inspectorate (now Health and Safety Executive) as a means of testing compliance with those sections of the Asbestos Regulations 1969 relating to potential asbestos exposure. The requirement and limit for control of chrysotile, amosite and fibrous anthophyllite dusts is related to the airborne concentration of asbestos dust averaged over a 4 hour sampling period with a threshold of action at 2 fibres/cm^3.

3 The current position with regard to chrysotile asbestos in industrialised societies is that most western countries including Canada, Denmark, Finland, France, Italy, Norway, South Africa, Sweden, USA and the UK, apply or are in the process of adopting a 2 fibre/cm^3 limit based on the counting of fibres greater than 5 μ in length by light phase contrast microscopy.

4 In a recent document[3] submitted to the US Occupational Safety and Health Administration, the National Institute for Occupational Safety and Health have recommended that the control limit should be reduced to 0.1 fibres/cm^3 (8 hour average) with peak concentrations not exceeding 0.5 fibres/cm^3 (15 minute average). This is based on the assumption that, when a substance has been shown to be carcinogenic, the exposure limit should be set at the lowest concentration which it is technically feasible to measure.

Sampling techniques and correlativity of data

5 Much of the earlier work in asbestos monitoring was carried out with sampling instruments which yielded results in the form of total particulate concentrations. A limited number of studies comparing old and new techniques of sampling have subsequently been made in an attempt to restate these exposure data in terms of fibre concentrations.

6 Ayer *et al* (1965)[4] carried out a side-by-side comparison of the membrane filter and impinger methods in four US asbestos textile plants. Five main operations were studied (preparation, carding, spinning, twisting and weaving) and comparison of 230 pairs of samples taken by the two methods show that an impinger count of 1 mppcf corresponded to a membrane count of 6 f/cm^3 (fibres > 5 μ per cubic centimetre). It was noted, however, that individual operations may have ratios differing from these values by a factor of 2.

7 Over the period 1971-2, surveys were conducted by Gibbs and LaChance[5] at up to nine selected sites in each of five mines and mills of the Quebec chrysotile asbestos mining and milling industry. A total of 87 pairs of membrane filter and midget impinger samples were collected. The linear correlation coefficient for 56 samples with more than 1 fibre per field was 0.32, and after a logarithmic transformation of both variables, the correlation was increased to 0.45. For 31 samples with less than 1 fibre per field, the correlation was almost zero (—0.03) and the correlation of logarithmically transformed data was 0.25. If membrane and impinger counts were considered by work site, it was clear that the ratios of the two differed from mine to mine, and varied with the stage of production within the same mine or mill. It was concluded that correlation was poor and no single conversion factor was justified.

8 Very recently, Dagbert (1976)[6] has reported on the results of investigations carried out by Gibbs and LaChance in many mines and mills of the Quebec asbestos industry. A study of 623 pairs of side-by-side impinger and membrane counts yielded, on logarithmic transformation of the two parameters, a correlation coefficient of 0.437. When the results for individual mines and different processes are considered, wide variations are observed in the correlation coefficient but overall the relationship between particle and fibre counts is described by the equation mppcf $= 10.97$ (f/cm^3)$^{0.68}$.

9 From the results of the above studies in mines, mills and textile factories, it would appear that in order to predict levels of dustiness in terms of fibres per cubic centimetre from measures of total particles per unit volume, it is first necessary to establish the correlation of the two parameters for each individual site and process considered, and the use of a single conversion factor to transform large masses of impinger data into equivalent fibre counts cannot be justified.

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10 In the United Kingdom, the development of instruments and techniques for the routine monitoring of airborne asbestos is largely based on work carried out by the asbestos textile industry over the last thirty years.

11 During the period 1951-61, the Casella Thermal Precipitator (TP) was used to collect particulate matter, the sampler being placed in fixed predetermined positions in specified working areas, ie static sampling. By 1961 the biological importance of fibres as opposed to particulate matter was recognised and, in order to obtain results in terms of fibre counts, the equipment was changed to a Long Running Thermal Precipitator (LRTP). The latter, while an improvement on the TP method, was not considered to be ideally suitable for the collection of airborne fibres and, after a period of development, was superseded in 1964 by the membrane filter (MF) method of sampling. The static membrane filter technique of sampling continued to be used until 1974, but gradually from 1970 more attention was given to the personal sampling method where the membrane filter sampler was attached to an individual operative rather than being located in a particular work area.

12 Sykes (1977)[7] has recently described studies carried out within the asbestos textile industry to relate data obtained from TP and LRTP sampling to that obtained by modern membrane filter counting. By means of side-by-side comparisons of instruments measuring dust levels generated by present day factory processes, but employing the mounting and counting techniques contemporaneous with each type of instrument, the following expressions have been obtained:
Modern MF fibre count = 0.71 TP particle count +0.6
Modern MF fibre count = 2.155 LRTP fibre count

13 It is pointed out that these relationships, though apparently precise, are derived from sets of data which correlate relatively poorly and, additionally, as the comparisons on which they are based were carried out under modern factory conditions, the application of conversion factors to historical data obtained under different factory conditions requires caution.

14 A limited comparison of static membrane filter samples and personal membrane filter samples has also been attempted. The results, expressed as the ratio of personal fibre count to static fibre count, vary widely, not only between processes but also within the same process as can be seen from the following table.

Process	*Personal fibre count / Static fibre count*
Card Tenters	Range 0.7–7.0
Spinners	0.5–2.2
Fiberising and Spinning	1.7–17.0
Winding	0.5–1.1
Plaiting	0.5–2.2

From this data it is not possible to obtain a generally applicable correlation factor, but it is possible to state that in most situations the personal sample result is higher than the static result. In areas where dust emission levels are high, the ratio is of the order of 10:1, whereas in areas with known good control the ratio is about 1:1.

Sampling method and compliance with the control limit

15 Since the introduction of membrane filter sampling there have been changes in counting techniques which are now recognised to have affected the results of fibre counts. Beckett *et al* (1967)[8] have shown that the current method of eyepiece graticule counting as opposed to whole field counting increases fibre counts by a factor of about 1.5 for amosite and 2.5 for chrysotile.

16 The control limit for chrysotile of 2 fibres/cm^3 was derived from dust data[1] obtained by static membrane filter sampling or from TP data converted to static MF fibre counts. Present day testing for compliance with the control limit is, however, based on personal MF sampling.

17 At the compliance level (2 fibres/cm^3) the ratio of personal fibre count to static fibre count is probably of the order of 2:1. In effect, this means that a 2 fibre/cm^3 count obtained by sampling with static MF and counting by whole field techniques requires to be multiplied by factors of 2 (personal: static ratio) and 2.5 (graticule v. whole field) in order to convert it to the equivalent fibre count which would be obtained by personal MF sampling and modern counting technique. Conversely, if it is demonstrated today by means of personal sampling that a work situation complies with a 2 fibre/cm^3 control limit then it is likely that we are working to a standard at least five times as stringent as that imposed in 1968.

18 In view of the fact that compliance testing will continue to be based on personal sampling, if for no other reason than that it defines the test location, this unacknowledged tightening of the asbestos standard should be taken into account in any proposal to alter the existing control limit.

Conversion of impinger data

19 If use is to be made of impinger sampling data for the purpose of studying the relationship between asbestos dose and biological response, then it is clearly necessary to convert total particulate counts into equivalent counts of biologically important fibres.

20 As far as mining and milling data are concerned, Dagbert's[6] formula may be used to calculate the static sampling count of fibres greater in length than 5μ equivalent to the corresponding static impinger particle count, and the subsequent application of factors to compensate for the differences between static and personal sampling, and whole field and graticule counting, will allow an estimate of exposure to be made

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in terms of the modern membrane filter technique. It must be pointed out, however, that the range of particle concentrations amenable to this conversion is limited by the lack of good information on the ratios of exposure as measured by personal and static sampling. Such experimental data as exists suggests that, at low to moderate dust concentrations, an appropriate factor (personal:static) is of the order of 2 but at high concentrations, estimates of the factor become increasingly speculative and it is therefore suggested that conversion from impinger count to modern membrane filter count should not be attempted where the impinger count exceeds 1 mppcf. In the event, this may not result in the loss of too much impinger data as is indicated by the fact that in Gibbs' extensive survey, the median dust concentration was found to be 0.71 mppcf[5].

Examples of conversions within this range are:

	Conversion factors			
Particle count* mppcf	Particles to fibres 5μ	Static to personal	Field count to graticule count	Impinger count to MF count
0.1	23.0	2	2.5	115.0
0.5	13.7	2	2.5	68.5
0.7	12.4	2	2.5	62.0
1.0	11.0	2	2.5	55.0

*For impinger data collected earlier than 1971, it may be necessary to adjust the particle count by up to 50% to take account of changes in experimental technique[9].

(1) The British Occupational Hygiene Society. Hygiene Standard for Chrysotile Asbestos Dust, Ann. Occup. Hyg. *11*, 47, 1968.

(2) Technical Data Note 13 (Rev). Hygiene Standards for airborne dust concentrations for use with the Asbestos Regulations 1969. HM Factory Inspectorate, Department of Employment, HMSO.

(3) NIOSH. Re-examination and Update of Information on the Health Effects of Occupational Exposure to Asbestos. US Dept of Health, Education and Welfare (December, 1976).

(4) Ayer, H.E. Lynch, J.R. and Fanney, J.H. A Comparison of Impinger and Membrane Filter Techniques for Evaluating Air Samples in Asbestos Plants. Ann N.Y. Acad. Sci. *132*, 274, 1974.

(5) Gibbs, W.G. and LaChance, M. Dust/Fibre Relationships in the Quebec Chrysotile Industry. Arch. Environ. Health, *28*, 69-71, 1974.

(6) Dagbert, M (1976) Etude de Correlation des Mesures d'Empoussierage dans l'Industrie de l'Amiante. Document 5, Beaudry Report (Quebec Committee of Enquiry of Health in the Asbestos Industry).

7) Sykes, R (1977) TBA Industrial Products Ltd. Personal Communication.

(8) Beckett, S.T., Hey, R.K., Hirst, R., Hunt, R.D., Jarvis, J.L. and Rickards, A.L. A Comparison of Airborne Asbestos Fibre Counting with and without an Eyepiece Graticule. Ann. Occup. Hyg., *19*, 69, 1976.

(9) McDonald, J. (1978) Personal communication.

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Asbestos: the situation overseas

Prepared by the HSE from information supplied by the countries concerned

3

1 This paper summarises information made available to the Committee during our investigation on the response to occupational exposure to asbestos in some other major industrialised countries. From this existing data we have attempted to examine legislation in 14 countries: Australia[1], Belgium, Canada, Denmark, The Federal Republic of Germany, France, Italy, Netherlands, Norway, Republic of Ireland, South Africa, Sweden, USA USSR. Where applicable, we have provided a brief comparison with current practice in the UK for ease of reference. More full details for the UK are given in Appendices L and P. Unfortunately, the information available on certain countries (such as Denmark, Belgium and the USSR) is very sketchy, so often only an incomplete picture can be given. We are also very conscious that the information we are able to present quickly becomes outdated as the countries concerned review and amend their controls. We must therefore emphasise that, while believed to be accurate when it was compiled, the information in this Appendix should be checked for currency with representatives of the individual countries.

2 The main aim of legislation in all the countries we have looked at is to reduce the risk to health of persons exposed to asbestos. Most countries have tried therefore to identify operations where the highest concentrations of hazardous asbestos dust will be evolved, considering these to be where the atmospheric suspensions of asbestos fibres are by definition less than 3 μm in diameter and from 10–200 μm in length. In paras 8 and 9 we consider the standards that various countries have imposed to reduce the exposure of persons to concentrations of fibres of this size and in paras 9 to 13 we look at the different systems for monitoring the working atmosphere to see that such standards are achieved. A summary of present standards in the countries is given in Table 1.

3 Some countries require the use of handling of asbestos to be notified to a competent authority. We consider such notifications in paras 14 to 21.

4 In certain countries the danger to health from specific operations has been considered to be sufficiently hazardous for these operations to be banned. We have referred to these bans and other prohibitions on the use of asbestos in paras 22 to 24. We have also covered exemptions from bans in para 25 and Table 2.

5 Some countries have attempted to replace asbestos products with less harmful materials and we discuss these later in paras 26 to 28.

6 Where it is not at present feasible either to ban or provide suitable alternative products for asbestos, most countries have introduced legislation to reduce the generation of and/or exposure of workers to dust by means of enclosure or local exhaust ventilation. We consider these and other control methods later in paras 29 to 52.

7 Finally, in paras 53 to 67 we look at medical surveillance of asbestos workers.

Control limits

8 Table 1 shows the current standard applied in each country at the time of an investigation. From the table the Swedish level of 1 fibre/ml seems to be the most stringent standard currently in force. However, a comparison of standards is not always straightforward, both because different measurement methods and instruments are used, and because ceiling values of "maximum allowable concentrations" vary. For example, both the UK and the USA currently have a basic 2 fibre/ml limit, but it is accompanied here by a limit of 12 fibre/ml when measured over a 10 minute period, and in the USA by an absolute (or instantaneous) limit of 10 fibres/ml.

Monitoring the occupational environment

9 The system most commonly used in the countries we looked at is the membrane filter method (described in our Second Report). The counting of the fibres collected on the filter is done by means of an optical microscope with magnification of 450–500 times (400–450 in USA) using transmitted light and phase contrast techniques. In the Federal German Republic and USSR, however, as their control limits are expressed in terms of mg/m^3 respirable dust is checked for asbestos content, then analysed by weight.

10 One of the problems that has been identified in the countries which use the phase content microscopy is the wide variation in counting techniques that has been noted between individual laboratories. In an experiment covering nine countries conducted by Walton, Atfield and Beckett (2), it was found that counting techniques differences gave ratios of highest to lowest count of 1.7 for amphibole and 2.8 for chrysotile asbestos. We have already drawn attention to this problem in the United Kingdom in our Second Report, and have suggested ways for overcoming it here.

11 In USA, Sweden and France there is a requirement for periodical sampling of the air in the

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breathing zone of the employee (personal) or the background air of the workman (static) or both. Records of the results are required to be kept so that a regular check could be made to ensure that the hygiene standard was being maintained.

12 In the USA an employer is required to carry out both personal and static sampling at least once every six months and more frequently than this if an employee is exposed to concentrations over the hygiene standard. The records are maintained at present for three years, and the employee can have access to these if he wishes. Where exposure greater than the control limit is recorded, the employer has to notify the Assistant Secretary for Labour.

13 In France the workroom air should be sampled at least once a month, or every three months if three preceding samples have shown an average air concentration of less than 1 fibre/ml. The record of monitoring is held for use by the industrial medical officer. Norway require quarterly inspection and records are filed with the district office of industrial inspection. In Sweden there is a requirement for yearly samples, for records to be kept of monitoring, and for details to be submitted regularly by employers to the labour inspectorate.

Notification of use of asbestos

14 Six of the countries we have looked at already require written notice to the appropriate authority that asbestos is being used or will soon be so, though the requirements as to the precise use for the asbestos may not be the same in each country.

15 In the USA the owner/operator has to report his intention to the Environmental Protection Agency 20 days before he starts any demolition operation where asbestos is involved, or before he starts to spray asbestos materials containing more than 1 % asbestos on a dry weight basis.

16 French employers on the other hand have to give written notice to the factory inspector of any work they may undertake which is liable to give off asbestos dust, and to which their employees may be exposed.

17 In the German Federal Republic there is a requirement for employers covered by the Industrial Mutual Accident Insurance (IMAIA) to notify their intention to use asbestos materials to that body.

18 Employers in Sweden are required to consult with the National Board of Occupational Safety and Health for asbestos used in any new process and to let the labour inspectorate know if asbestos dust is unavoidably generated at their workplace.

19 In Australia (1) there are Model Regulations prepared by the National Health and Medical Research Council which are used as a basis for legislation by certain states. These regulations require an occupier to send to the competent authority within 28 days notification of an undertaking to do work involving asbestos and the location where such work is to be done.

20 The Netherlands on the other hand require any employer to notify their labour inspectorate before they start any process where asbestos is used, or where they intend to use a new asbestos product.

21 In the UK there is a requirement under the Asbestos Regulations 1969 for a person undertaking any process involving crocidolite to notify the Factory Inspectorate 28 days before the work starts. In our First Report we recommended that the Health and Safety Executive (HSE) should have the power to require any employer licensed to undertake work involving sprayed coatings or thermal or acoustic insulation, to notify the HSE in advance of jobs for which a licence is required[3].

Restrictions on the use of asbestos

22 Table 2 shows eight countries which at present have imposed restrictions either on particular types of asbestos or on certain of its uses. In the majority of these countries this includes severe restrictions on crocidolite. In Sweden, for instance, the use and spraying of crocidolite is banned since replacement materials are considered to be available; materials "in situ" containing crocidolite must also be replaced by those which are crocidolite free in buildings and equipment and other places where they are known to be, or when they are found during repair or renovation work. In the Netherlands it is the ultimate aim to prohibit crocidolite completely; so far they have forbidden the manufacture, treatment and the stocking of it as well as banning products which contain it.

23 The spraying of other types of asbestos is totally banned in Sweden, the Netherlands and Norway, whereas partial bans are imposed or recommended in France and Denmark. The use of asbestos for certain insulation purposes is prohibited in the Netherlands, Denmark, Norway and Sweden. We have made recommendations in the First Report for a ban on new thermal or acoustic insulation, in the UK except in limited applications for which suitable substitutes are not yet available[3].

24 Other uses for which asbestos has been banned in Sweden are in cushion flooring, the undersides of mats, paint, glue, putty or jointing material and in certain asbestos cement products. In the USA asbestos for patching compounds and artificial fire place logs and ashes has been banned, whereas in Norway there is a ban on the use of asbestos for carpet underlays and floor tiles.

25 Several countries have had to allow certain exemptions from the bans they have imposed. Such exceptions are normally agreed to only where no suitable or acceptable substitute exists for asbestos. For example, in Sweden permission for the continued use of asbestos cement piping has been agreed on condition that wide-

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ranging precautionary measures are taken. The cutting method for such piping has been changed so that average exposure levels have gone down from 5 fibres/ml to 0.1 fibres/ml. At the same time the manufacturers are making efforts to develop replacement materials for asbestos as a reinforcing material in this piping.

Substitutes for asbestos

26 We know of only three countries where significant attempts have been made to replace substances with an asbestos base by substances not containing asbestos. The main problem is identifying a material which will have the characteristics and properties of asbestos, yet not be hazardous to health.

27 In Sweden attempts are being made to replace asbestos cement by glass fibre reinforced cement (with fibres relatively large in diameter and length). In buildings asbestos cement cladding is being substituted by aluminium and steel building sheets, and over 80% of the total market for new materials for the construction and renovation of agricultural buildings consists of profiled metal sheets. Rockwool products are also used as a primary replacement material. In specialised uses, expanded mica can be used for heat insulation and ceramic fibres used where high temperatures occur.

28 In Norway there is hope that as a result of Norwegian-Finnish co-operation in asbestos research, one firm will be able to produce asbestos-free building materials for indoor use by the end of the decade. Moreover, the Norwegians hope to have asbestos-free materials for cladding and roofing for outdoor use before 1981.

Control measures

WETTING

29 Several of the countries studied have issued directions for the wetting of asbestos thermal insulation before dismantling and water spraying in certain other processes for dust abatement: for example, in mines in Canada and South Africa and workplaces in France, Denmark and USA.

VENTILATION

30 Most countries studied have generally encouraged the control of asbestos dust by means of enclosure or by local exhaust ventilation. In the United States, for example, encouragement is given to the use of exhaust ventilation equipment for dust removal since the official view there is that dust masks, although they afford adequate protection, are hampering to the workers and therefore likely to be discarded. Apart from equipment for keeping the workplace air clean, appropriate devices for asbestos dust removal must be fitted on machine tools used to work substances containing asbestos, (such as saws, grinding and drilling machines) and on tables and workbenches where substances likely to emit such dust are unpacked.

31 In the United Kingdom the Asbestos Regulations 1969 lay down requirements for dust concentrations to be kept below prescribed limits, largely by ventilation. The same applies in regulations in force in certain Australian states. Each set of regulations requires the ventilation equipment to be inspected once a week and overhauled thoroughly every 14 months. France also requires periodic checks and maintenance of ventilation equipment.

PROTECTIVE EQUIPMENT AND PROTECTIVE CLOTHING

32 Where it is impracticable to control the dust by enclosure or local exhaust ventilation, most countries require respiratory protective equipment and protective clothing to be provided and used. In Belgium, Denmark, Italy and Norway it is specified that such equipment and clothing must be provided, cleaned and maintained at the employer's expense.

33 In the United States respiratory protective equipment must be worn in all demolition work and asbestos spraying. Such equipment must not be used for more than a specified time and must be cleaned regularly by competent persons.

34 Detailed instructions are given in the United Kingdom and some Australian states that protective equipment must be kept in good condition and be thoroughly cleaned and disinfected before being used by another worker.

LAUNDERING OF PROTECTIVE CLOTHES AND WELFARE FACILITIES

35 Most countries require the protective and other clothing contaminated with asbestos dust to be removed in special changing rooms, often with separate lockers for storage, and to be sent out in sealed containers, either to authorised laundries for washing, or for disposal to prevent re-use. In UK and Australia (1), special arrangements exist for the laundering of clothes. Also in Australia (1), a special meal room has to be provided which is suitably separated from the workplace but conveniently accessible to it.

CLEANING OF PREMISES

36 In most countries this requirement is provided for under general health and safety regulations. In Australia (1) and the United Kingdom, rules specific to asbestos provide that all premises where asbestos is used or worked should be kept free from asbestos dust by the use of approved vacuum cleaners (or other suitable method) so that asbestos dust neither escapes nor is discharged into the air of any workplace. In the USA there are rules prescribing how the premises should be kept and how waste should be removed.

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DESIGNATED AREAS

37 In Australia (1), any workplace area where it is impracticable to provide exhaust ventilation to prevent asbestos dust entering the area in hazardous concentrations, and where respiratory protective equipment is therefore required, is designated a "compulsory respiratory area". Moreover, construction sites where work is performed on asbestos have to be effectively screened and isolated.

38 In the USA cautionary signs have to be posted where the asbestos fibre concentration may be in excess of the exposure limits.

PREFABRICATION OF ASBESTOS PRODUCTS

39 In Denmark there is a requirement for sheets for site work to be pre-drilled and pre-cut. Only small ready-cut mitred sheets can be sold to the general public there.

40 Norway require products to be prefabricated or pre-processed at the point of manufacture where this is practicable; otherwise, local exhaust ventilation has to be provided for tools used on the site.

Employers' and workers' responsibilities

41 In the USA the employer is responsible for installing, operating and maintaining equipment designed to prevent asbestos dust concentrations exceeding a specified level and where necessary he has to supply staff with appropriate protective equipment. He remains responsible even for work given to people outside his direct employ, for example if he gives clothes contaminated with asbestos to a laundry, he is obliged to draw their attention to the risks involved.

42 In Australia (1), the UK and France, employers have, as has already been mentioned, a duty for periodic checks and maintenance of ventilation equipment. In the UK and Australia, employees should be properly instructed in the use of the respiratory protective equipment, and in Australia, employees have a duty to place their protective clothing in special lockers.

43 In the UK, for example, employers have a general duty under the Health and Safety at Work Act to ensure, so far as is reasonably practicable, that substances used at work are safe and without risks to health when properly used. Employees are required to co-operate with their employers to enable this duty to be complied with.

Storage and transport

44 Most countries require asbestos waste to be locked in containers or sealed in impermeable bags. The UK and Australia (1) have rules for storage, warehousing and transport of loose asbestos which must be kept and transported in closed receptacles which in turn must be clearly marked. France also requires packages containing asbestos to be labelled accordingly.

Labelling of products and consumer goods

45 In Sweden, asbestos has been classified by the Product Control Council as a hazardous substance and rules issued in 1977 require suppliers to inform their customers about the asbestos content of various products.

46 Norway requires importers, producers and suppliers of asbestos goods to give clear indication on the products they sell of the health risk from asbestos.

47 In the UK under a voluntary agreement with manufacturers, all asbestos products for use at work manufactured after October 1976 which are liable under any foreseeable circumstances to create asbestos dust, and all asbestos based consumer products, are labelled so that they can be easily identified.

Deposits of asbestos waste

48 Australia (1) requires asbestos waste to be covered by not less than 10 cm of earth. Sweden also requires the burial of such waste.

49 The USA Environment Protection Agency (EPA) administer national regulations for waste disposal sites. They require the sites to be clearly marked with warning signs, for fencing to be provided, and for asbestos to be covered with at least 15 cm of compacted, non-asbestos-containing material every day. Also, every 24 hours the asbestos waste must be covered by a resinous (or petroleum based) dust suppression agent which effectively binds dust and controls wind erosion.

50 In the UK waste deposits are controlled under the Deposits of Poisonous Waste Act 1972, and the Control of Pollution Act 1974 which are both administered by local authorities.

Information and advice

51 In Australia (1), Belgium, the Federal Republic of Germany, the Netherlands and especially the United States and the UK, information on asbestos hazards is disseminated by Government and other interested bodies. The increased risks of smoking and working with asbestos are particularly stressed.

52 France, Sweden and UK have requirements or rules obliging employers to keep their staff fully informed about the precautions to be taken against potential occupational hazards.

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Medical surveillance

EXAMINATIONS

53 In a number of countries national legislation lays down that any worker employed in the extraction of asbestos, the manufacture of products containing asbestos, or the use or application of such products must, if the processes are liable to produce asbestos dust, be given a medical examination before such employment, and then, at appropriate subsequent intervals, supplemented by X-ray and biological examinations if required, so as to keep a check on the worker's health in relation to his level of exposure.

54 Sweden has regulations which require examinations for the prevention of "dust lung" (pneumoconiosis). When employees start work on a process where they may be exposed to dusts (including asbestos), their previous medical history is checked to see if they have had previous exposure to dusts which may have caused "dust lung". In addition, information is collected about their smoking habits and the examination includes a clinical inspection and a chest X-ray. Periodic examinations are required at three yearly intervals though a doctor can ask for more frequent examinations if he feels these are necessary. In June 1976, the Swedish Government made provision for free medical examinations for people who had worked at some time with asbestos and in particular workers who had carried out insulation work in shipyards.

55 Workers in France must have a certificate from a competent works doctor to the effect that there is no special reason why they should not be exposed to asbestos dust. The certificate must contain a record of medical examinations given prior to the new work and is renewable annually. The examinations include a standard radiograph of the lungs and an examination of the lung function. A Government order lays down certain technical instructions that the works doctor responsible for the surveillance should follow.

56 In South Africa all workers in mines and works are medically examined before work. The examinations are administered through the local offices of the medical bureau of industrial diseases. If the worker passes he is given a red card. Subsequent examinations can be required at any time at the discretion of the medical bureau and they must be held at least once a year. If the worker fails an examination, his red card lapses, which means that the area of his subsequent employment is restricted.

57 Canada has pre-employment examinations for underground mineworkers and for all workers in asbestos industries: for example, British Columbia requires a worker to be examined within one month of starting, and then an annual health certificate of fitness being given. In Manitoba, the initial examination is within 60 days of start of work, and annually thereafter. Alberta in contrast has examinations every two years. All examinations in Canada must be supplemented by a chest X-ray.

58 In Australia (1) most states have medical examinations for specified workers. South Australia for example requires any person who is to be employed in any asbestos process to present himself to the medical officer of health for a free examination before or just after he starts the new work. Within 90 days of his start, the employer, at his own expense, must arrange for the employee to undergo an X-ray, and a pulmonary function test and ensure that a statement of the worker's occupation and smoking history is available to the examining authority. The employer must make sure that the worker is examined at least once every three years. When a worker fails to present himself for a medical, he is in breach of the regulation and the employer must not knowingly employ him in any asbestos process. Moreover, no employer should knowingly employ anyone who has been examined and prohibited by the authorised medical officer from working where he may be exposed to asbestos dust.

59 In the Federal Republic of Germany, the IMAIA issued a set of principles in 1971 which called for medical examinations as a preventative measure for asbestos related diseases. Also in 1971, the Federal Ministry of Labour and Social Affairs issued rules governing the medical examination of workers exposed to dust of mineral origin. The employer has to pay for initial and periodic examinations. The latter are required every three years, but the interval can be reduced if the approved medical practitioner considers the worker has been exposed to excessive concentrations of asbestos. The IMAIA also has the authority to exempt workers from examination if the worker holds a certificate from a medical practitioner clearly showing that the worker has no asbestos related disease.

60 In the USA regulations require any worker occupationally exposed to asbestos dust where the exposure exceeds 0.1 fibres/ml to undergo a medical examination within 30 days of being assigned to such work. There is also a requirement for regular annual examinations. Each examination should include at least a medical history, a chest X-ray, and lung function test.

61 In the UK, workers in certain industrial processes where asbestos is worked, are required to have an initial medical examination before the end of the second month after they have started such work. A voluntary survey of all workers subject to the Asbestos Regulations 1969 began in 1970 and is being carried out by the Employment Medical Advisory Service.

62 Of the other countries we considered, the USSR require 6 monthly examinations of workers exposed to hazardous materials, and Norway have pre-employment and three-yearly medical examinations.

63 Australia (1) and USA also require employees to be examined within 30 days of terminating their

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employment whereas in Sweden the medical control continues for some time after the worker's exposure to asbestos dust has ended.

64 Australia (4), France, Denmark and Norway prohibit young persons under 18 years of age from being employed in any industry where they may be exposed to asbestos dust.

65 People who smoke in Norway are not allowed to work in employment where they may be exposed to asbestos dust (this prohibition also covers people with chronic illnesses of the lungs, respiratory system, or heart disease). There is also authority available in Australia (1) to ban or restrict smoking and smokers at work with asbestos and OSHA in the USA have proposed that smoking be banned in designated areas.

Record keeping

66 The Federal Republic of Germany, Australia (1), USA, Norway, France, Sweden and the UK all require some form of medical records to be held for the purpose of deciding compensation claims, for physicians to consult, and to provide statistical information for epidemiological purposes. Australia retains its records for 40, France 30, and USA for 20 years respectively, while Norway requires the records to be held until the government authorises them to be destroyed. In France the records state the type of work performed by the worker, the latest findings of the test, the concentrations of dust to which he is exposed, the length of the exposure and the dates of the various examinations.

Claims for compensation

67 Little is known about claims for compensation by persons suffering from asbestos-related diseases in the countries we considered. In South Africa there is a fund financed by compulsory levies on employers which provides compensation for employees certified as suffering from pneumoconiosis. In the UK any person who is diagnosed as having asbestosis or diffuse mesothelioma of the pleura or of the peritoneum and can substantiate a part exposure to asbestos can make a claim for compensation which, if upheld, entitles him to a disability payment.

(1) Australia is a Federation of states and each state is responsible for implementing its own legislation. Certain states however base their regulations for asbestos on Model Regulations prepared by the National Health and Medical Research Council.

In this paper most of the information is derived from the legislation which is applied in Queensland.

(2) "An international Comparison of Counts of Airborne Asbestos Fibres Sampled on Membrane Filters" by W H Walton, M D Attfield and S T Beckett (Institute of Occupational Medicine, Edinburgh)
"Annals of Occupational Hygiene Vol 19 pp. 215-224 Pergamon Press 1976"

(3) ACA: "Work on Thermal and Acoustic Insulation and Sprayed Coatings", HSC, 1978; pp. 13-15.

(4) Queensland and South Australia.

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Table 1 Present hygiene standards for asbestos in 15 countries

Country	Present hygiene standards for asbestos				
	Type of asbestos	If known TWA in hours*	No. of fibres per ml or equivalent (unless otherwise stated)	Legislation or guidelines	When legislation took effect (if known)
Australia	All		5 million parts ft³	Harmful gases, fumes, mists, smoke and dust regulations	18.4.1977
Belgium	All		2	Law in preparation	
Canada	All	8 hrs	5 Quebec	Each State has set of rules eg. Quebec has laws & regs. covering health & safety in mines and quarries	
		8 hrs	2 Ontario		
Denmark	All		2	Law	1972
France	All Breathing zone Air of workroom	Working day	2 1	Decree	20.10.77
Germany (Federal Republic)	Dust 100% Chrysotile Dust less than 3½% Chrysotile (NB. Precise limit in a given workplace depends on how much Chrysotile there is in the dust when analysed by weight)		0.15 mg/m³ 4 mg/m³	Interlocking requirements from Federal Government, Landergovt. Central Union of Trade Co-operatives	
Ireland (Republic of)	Crocidolite Other		0.2 2	Regulations	1972
Italy	All		5	Guideline only	
Netherlands	Crocidolite Others		Less than 2 2	Decree	1.4.78
Norway	All		5	Regulations	1973
Sweden	All (Crocidolite banned)		1	Regulations	1.7.76
South Africa	All			Occupational diseases in Mines and Works Act	1973
USA	All Ceiling for all employees	8 hrs	2 10	Regulations	1.7.76
USSR	All Dust <10% Asbestos Dust <30% Asbestos		2 mg/m³ 1 mg/m³	Regulations	
United Kingdom	Crocidolite Other	10 mins. 10 mins 4 hrs	0.2 12 2	Regulations supplemented by guidance notes	1970

*TWA = Time weighted average.

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Table 2 Restrictions on the use of asbestos in certain countries

(1) Country	(2) Ban on crocidolite	(3) Ban on the uses etc. of other types of asbestos	(4) Ban on smoking	(5) Exemptions from bans etc.
AUSTRALIA	1 Banned (but see column (5)); 2 The Government inspectorate must give approval before repair dismantling or demolition of work involving crocidolite is undertaken.	All spraying (but see column (5)).	Authority is available to ban or restrict smoking and the employment of smokers.	1 Competent person (eg Government inspector) can approve use of asbestos (including crocidolite) in certain circumstances; 2 The exemption normally lasts for 12 months.
DENMARK	As far as is known no crocidolite is used any more.	1 Thermal insulation and sound proofing. 2 Insulation against dampness and humidity. 3 There are proposals to ban spraying.		Chief Labour Inspector can authorise exceptions for the assembling or dismantling of insulation materials if no risk incurs from the work.
FRANCE		Spraying flock* finishing on walls and other elements of apartment buildings dwellings etc. approved for construction after 1.7.77. *(production of fibrous velvety or fluffy surface).		
NETHERLANDS	From 24.5.78. 1 Manufacture treatment and stocking of crocidolite banned. 2 Products containing it also banned (but see column (5)).	Spraying of asbestos; and asbestos products used for insulation sound proofing conservation and decorative purposes.		1 Water pipes containing crocidolite are exempt until a suitable substitute is found. 2 Factory inspectors can grant exemptions in consultation with Tripartite Advisory Committee.
NORWAY	Banned either in pure form or in combination with other substances from any production process or use of the product where dust may be evolved (but see column (5)).	1 From 1.4.78. Use in carpet underlay and floor tiles. 2 Where it is not practicable or economically feasible to achieve the minimum standard of 2 fibres/ml then the process or machine may have to be stopped. 3 Use of asbestos for certain insulation purposes (eg electrical, thermal or noise) whether done by impression painting or spraying.	1 Smoking prohibited where asbestos used. 2 Smokers not allowed to work in an environment where asbestos fibres evolved.	If it is proven that crocidolite is essential and no acceptable substitute is available then exemption may be granted by the Government.
SWEDEN	1 In 1975 Use and Spraying. 2 Where it exists in buildings or equipment it must be replaced as soon as possible; 3 Where it occurs otherwise it should be replaced when repairs or new construction work done.	Spraying; heat insulation†; cushion flooring backsides of mats; in paint; glue; putty of jointing material; and asbestos cement products‡. † ‡ (see column (5)).	Draft regulations stress the danger of joint exposure to asbestos and tobacco.	1 Asbestos may only be used where no acceptable substitute exists; eg in friction materials, insulation for very high temperature smelters†, reinforcement in heavy dual packaging, in certain personal protective equipment and for special purposes such as chloralkaline electrolysis. 2 Asbestos cement in sewage and water pipes‡.
USA		1 Patching compounds and artificial fireplace logs and ashes containing asbestos. 2 Spraying of asbestos materials containing more than 1% asbestos on a dry-weight basis.	OSHA have proposed that smoking, chewing (non-food products) and drinking are banned in designated areas.	
UNITED KINGDOM	1 Importation of crocidolite ceased in 1970; (but no statutory ban). 2 See column (3) for the recommended ban on spraying and use of asbestos for insulation.	The Advisory Committee on Asbestos have recommended in its first report that the spraying and use of asbestos materials for any new or acoustic insulation should be banned.		ACA in its first report have suggested that limited applications for which suitable substitutes are not yet available could be excepted from the ban recommended at column (3).

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Some products containing asbestos and possible substitutes

4

The Advisory Committee arranged for an exploratory survey to be made with the object of identifying asbestos containing products which because of their probable risk to health should be given priority in efforts to replace the asbestos by a substance believed to be less hazardous.

This paper deals with the technical feasibility of substitution which is only one aspect to be considered when assessing the suitability of possible substitutes for asbestos. Other aspects, such as the possible risks to health from substitute materials, are not covered as these were outside the Committee's terms of reference.

We must also emphasise that these notes are not exhaustive as our information on a number of other products where substitution may also be possible is incomplete. We have recommended (Recommendation 6) that guidance should be provided to employers on substitution. We envisage that there will be additional information on substitution in the light of future research, but believe that this paper provides a useful survey of current technical knowledge.

Asbestos cement ventilation ducts

CHARACTERISTICS

1 Asbestos cement is basically cement reinforced with asbestos fibre (approximately 10% to 15% of the total by weight).

2 Asbestos cement ventilation ducts may be formed by single sheets supported in a frame from purpose made box sections or from asbestos cement flue pipes and fittings.

3 Asbestos cement pipes should conform to the requirements of BS 567: 1973 for light quality pipes (diameters 50–150 mm) or BS 835:1973 for those of heavy quality (diameters 75–600 mm). Thickness varies from 4.5 mm for a pipe of 100 mm internal diameter to 12 mm for a 300 mm ID heavy quality pipe.

4 All products should conform to the strength and performance requirements of their relevant British Standard but care in handling is necessary to avoid breakage or chipping.

5 Some products are available, at extra cost, with the internal surface treated with a sealer which is claimed by the manufacturers to reduce any risk of emission of fibres in use.

USE

6 Asbestos cement ventilation ducts can be used where no fire resistance is required but thermal and acoustic insulation is desired, sometimes more cheaply than other materials. We cannot say how much asbestos cement has been used in the manufacture of ventilation ducts. Such information as we have indicates this material has often been used for extract ducts from laboratories, toilets, incinerators etc, but we have so far found very few examples of its use in air distribution systems. We have been advised that due to lack of demand, the manufacture of preformed box section ducts has now ceased. Where asbestos cement ducts are used in a building they are seldom, if ever, exposed, therefore their poor impact strength and appearance are less important.

EXISTING ALTERNATIVES

7 A wide range of substitute or alternative materials is available for fabrication into ducts. The choice of any particular material will depend on specific performances required by the building designer. Various sheet materials such as plasterboard, metal, plywood, fibreboard, plastic etc. are often used but can be less convenient and cost more. However, for applications such as linings fitted to enclosed ducting, a number of sheet materials have become available that combine the functions of the ducting and of the fire protective enclosure. These recently developed 'asbestos-free' boards are more expensive than the asbestos products they replace; but with full production their costs may become more attractive to the builder.

8 Costs of substitute and alternative materials will depend on the functions required from them (eg fire resistance, sound and thermal insulation etc), but tend to be higher than those of asbestos cement.

NEED FOR FURTHER DEVELOPMENT

9 As suitable substitute and alternative materials are now becoming increasingly available, there would seem to be no urgent need at present to seek additional substitutes.

10 The use of asbestos cement ducts as part of the extract to an air ventilation system is probably a negligible hazard, although alternative material could be used forming the lining to ducts delivering air within the building.

11 All available evidence would appear to indicate that substitute materials will cost more to purchase and fix than the asbestos cement products they replace.

Asbestos insulation board

CHARACTERISTICS

12 Asbestos insulation board has a medium (25–40%) content of Amosite or Amosite and Chrysotile fibre

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which is bonded by a calcium silicate matrix to form a board of medium density (about 700 kg/m^3). Asbestolux and Turnasbestos are proprietary names of some asbestos insulation boards that are manufactured to comply with BS 3536: 1974 for use where thermal insulation and fire protection are of paramount importance. The boards are non-combustible in accordance with BS 476 part 4: 1970 and can maintain integrity for up to 4 hours. They are Class O and Grade A as defined in part E 15(1)(e) of the Building Regulations and the Building Standards (Scotland) Regulations 1971-75 respectively. Their strength properties are good and they have good screw holding power for both face and edge fixings.

13 The boards are very easily worked to ensure close fitting, and will take decorations. Limited quantities have been used in housing but large quantities can be found in some other building types.

USE

14 Asbestos insulation boards have been very widely used to cover a considerable range of applications.

EXISTING ALTERNATIVES

General

15 The manufacturers of Asbestolux, UK Marinite and Turnasbestos have developed asbestos-free insulation boards, such as Supalux, Monolux, and Limpet board, which satisfy the performance requirements of BS 3536. The manufacturers claim that these substitute boards can be used as direct alternatives to their asbestos insulation boards. They cost from about 18 % more than the asbestos boards to produce, and they tend to have a somewhat greater density and therefore a slightly inferior thermal insulation, or a lower strength at the same density. There seem at present to be no other materials that can be considered as satisfactory substitutes in all the present uses of asbestos insulation boards.

Partitions and suspended ceilings

16 Supalux, Monolux and Limpet board can be substituted for asbestos boards in 'deemed-to-satisfy' partition and ceiling constructions with few changes in the method of construction. In addition to the 18 % greater initial cost, the substitute boards entail an unquantifiable extra cost in fixing, and when installation is completed their final cost may be considerably more. No reliable representative data are available as yet on costs. Other possible alternatives are plasterboard, glass reinforced gypsum (GRG) or low density glass reinforced cement (GRC), but in general these would have to be used in greater thickness or with additional insulation to provide the same standard of fire protection. Costs would be correspondingly higher.

Fire protection of building elements

17 Supalux, Monolux and Limpet board can often be substituted in 'deemed-to-satisfy' fire protective casings although some changes in their method of construction may be necessary to attain a four hour fire resistance. The cost of the substitute may, under certain circumstances be as little as 15% more. Other possible alternatives are plasterboard, GRG or low density GRC, but in general these would have to be used in greater thickness or with additional insulation to provide the same standard of fire protection so that a simple comparison of costs is not possible as yet. Both GRG and GRC could be made as shaped casings, to reduce work on site.

Ducting

18 Asbestos insulation boards are used for ventilating ducting mainly when there is a requirement for fire resistance or thermal insulation. Supalux and Limpet board can satisfy those requirements, and can be substituted for the asbestos board normally without any change in the method of construction of the ducting. The cost of substitution should only be 18 % greater.

19 Other materials that are used for this purpose are galvanised sheet steel, spirally wound metal strip, PVC and glass reinforced plastic (GRP); some of these alternatives would need additional insulation to prevent undue heat loss from ducts for heated air. The additional costs of these alternatives will therefore vary with any particular performance requirement, but they may well in any case be considerably higher in cost.

NEED FOR FURTHER DEVELOPMENTS

20 If, in the interests of economy, it is found that asbestos must continue to be used for insulation board, higher costs will undoubtedly be incurred in extra precautions during installations and removal.

21 We wish to acknowledge the assistance of the Trade Union in influencing the development of substitute boards. Notwithstanding the difficulties there are in finding a fully equivalent substitute, we believe that with sufficient effort one could be produced to match the asbestos insulation board in most respects.

Asbestos fibre - millboard and paper

CHARACTERISTICS

22 These materials are used to perform a wide range of tasks, and so the performance requirements are extremely diverse. However, many applications are not subject to specification and, as with so many asbestos-based products, the importance of some characteristics of the materials will only become evident as substitution is attempted.

23 Millboard: There are no specific performance standards for millboard.
Asbestos Paper: BS 3057 defines thickness, area density, electrical resistance tensile and bursting strength, water content and ignition loss of requirements for asbestos paper.

USE

Millboard

24 Millboard finds applications wherever a low cost, relatively soft, low density board material with modest

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mechanical properties, high heat resistance and good thermal or electrical insulation properties are required.

Asbestos Paper

25 Asbestos paper is inherently less dusty than millboard and is largely used as the central layer of a sandwich construction in gaskets or thermal insulation applications. In other applications it is impregnated before use.

EXISTING ALTERNATIVES

Millboard

26 Substitute for millboard based on aluminium silicate fibres are commercially available and are already being used, particularly where high temperature resistance is important.

Asbestos Paper

27 The relatively high cost of substitutes for asbestos paper is limiting their use in more general applications, but they are becoming available for applications at the lower end of the temperature range, where modified cellulose papers are being used. Rag felt (cellulose fibre) and glass tissue are used in place of asbestos fibre in roofing felt but give a lower resistance to fire penetration and flame spread. Aramid papers are being used for some electrical applications. For very high temperatures, ceramic fibres are available at high cost.

NEED FOR FURTHER DEVELOPMENT

28 Manufacturers are developing substitutes at high priority though a single general-purpose replacement will be difficult to produce with properties and cost acceptable to the market. The high priced alternatives presently available are technically unsuitable (eg too abrasive because of high shot content) irrespective of cost for some important applications and these may be designed out rather than substituted. It is not possible at present to show precise costs of substitution.

Asbestos cloth - protective clothing

CHARACTERISTICS

29 Non-inflammability, resistance to molten metal splash, wear resistance, flexibility and seam strength are required together with the minimum weight per unit area which can be achieved consistent with these properties.

30 The asbestos content of abestos cloth is high (between 65% and 100%). Only chrysotile asbestos is used.

USE

Protective clothing.

EXISTING ALTERNATIVES

31 Certain organic fibres, glass and aluminised cloths are available, usually for less severe conditions. Temperature-resistant nylon fibre has been claimed to be suitable in fire-fighting and foundry applications as well as for protective underclothing for racing car drivers. Where high temperature resistance is not so important certain forms of woollen cloth or leather may be used. No specific information is available on costs but nylon fibre is thought to be very expensive.

NEED FOR FURTHER DEVELOPMENT

32 There is a need for continuing work to produce substitute materials. The higher temperature resistant organic fibres such as aramids can cope with the less severe conditions at the expense of higher cost. Aluminised cloths extend the use of organic materials further into the more severe range. The problem is that the main risks are associated with the more arduous applications where substitution is more difficult.

Asbestos rope

CHARACTERISTICS

33 Asbestos rope is a relatively soft 'Conformable' product having sufficient strength for hand-wrapping. Heat resistance and thermal insulation or sealing properties adequate for the particular application are required. Admiralty specification 520 for asbestos fibre rope specifies plait spacing and linear density. Admiralty specification DG Ships 210A for glass fibre rope specifies the diameter of the rope, the length and weight of a coil and the % recovery on compression. The rope must withstand a pull of 30 lb, and no visible fumes may be emitted when the rope is heated to 1000°F. A life of several years is expected from this product.
It accounts for approximately 100 tonnes of abestos fibre each year.

USE

34 This product is used on plant and machinery where heat resistance and insulation are required between components.

EXISTING ALTERNATIVES

35 Dust-suppressed forms of asbestos ropes are readily available, but their hazard is reduced only during installation.

36 About 70% of the UK market for rope lagging has been captured by glass fibre or mineral wool products. Glass fibre rope lagging at prices generally competitive with those for the asbestos product have been on the market for years. For temperatures above the melting point of glass, ropes made from ceramic fibre are available, although at much higher prices.

NEED FOR FURTHER DEVELOPMENT

37 Asbestos rope is recognised as a dusty product used in circumstances where control of dust emission is difficult, and we would like to see the use of substitutes encouraged.

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Flooring tiles - (thermoplastic and PVC (vinyl) asbestos

CHARACTERISTICS

38 Thermoplastic tiles (BS 2592: 1973) and PVC (Vinyl) asbestos tiles (BS 3260: 1969) are made from a blended composition of a thermoplastic binder, asbestos fibre, fillers and pigments. Asbestos fibres contribute to the properties of the dough mix that enable an effective production process, and also largely account for the transverse strength of the finished tile and for other properties. The asbestos content of both types may be up to 22% of the total weight and the grade of chrysotile fibre used has a length of less than 1 mm.

39 The main characteristics of PVC sheet flooring are considered to be:

(i) Ease of application
(ii) Low cost
(iii) Acceptable appearance and range of colours
(iv) Requires less critical design of ground floors
(v) Resistance to spillages

USE

40 The tiles are usually fixed by solvent bitumen adhesives to a suitable sub-floor and are extensively used in dwellings as well as in public buildings such as schools and hospitals. Damaged or work tiles can be relatively easily replaced.

EXISTING ALTERNATIVES

41 There are many alternative floor finishes available although few if any can achieve all the characteristics of thermoplastic tiles and PVC (vinyl) asbestos tiles for a similar cost. Alternatives include linoleum, flexible PVC, cork, rubber.

NEED FOR FURTHER DEVELOPMENT

42 We feel that any hazard associated with thermoplastic and PVC (vinyl) asbestos tiles will be only slight. The asbestos content of the tiles has been reduced by some manufacturers to about 10% and research is in progress to further reduce or entirely eliminate it. Complete elimination of asbestos is believed to be technically feasible although there would be problems to be overcome. The long-term durability of the product is not known and there could be problems in achieving the required dimensional stability.

PVC sheet floor covering and PVC flooring with an asbestos backing

CHARACTERISTICS

43 (a) *PVC sheet floor covering with an asbestos backing*
Some of the coverings have a backing of asbestos paper, and they are produced principally for the home décor market. The backing acts as a carrier sheet during the continuous high speed production process.

(b) *PVC flooring with an asbestos backing*
Some imported PVC flooring has a backing of resin-asbestos composition, under a thick top (wear) layer.

44 The main characteristics are considered to be:

(i) Ease of laying
(ii) Ease of cleaning
(iii) Resistance to kitchen spillage such as fats, oils, alkalis, etc
(iv) Moderate cost
(v) Attractive appearance and colour

USE

45 PVC sheet floor coverings of this type have been introduced into the market in recent years with considerable commercial success. They have been very widely used in existing dwellings, laid mainly in kitchens, bathrooms and hallways over the existing floorings. In some cases the floor covering is fixed with adhesive, but usually it is laid loose, or fixed at the edges only.

EXISTING ALTERNATIVES

46 The nearest alternative would be printed felt base which consists of a bitumen saturated paper felt on which patterns are created with multicoats of paint. Other alternative floor coverings are available (flexible PVC cork, linoleum, carpet, etc) but their characteristics vary widely and none of them can replace fully the technical attributes of this type of material.

NEED FOR FURTHER DEVELOPMENT

47 The characteristics of the flooring listed above are largely those of the PVC sheet and are not always greatly affected by the asbestos backings. Attempts to use a glass or mineral fibre backing instead of asbestos paper have apparently led to difficulties, particularly at high production speeds. Any assessment of the cost of substitution must take account not only of differences in cost of materials but also of the cost of laying and the durability of the various materials.

48 We would recommend that support should be given to the development of substitute materials in this field.

Asbestos fibre: brake pads and linings: asbestos cloth: brake linings

CHARACTERISTICS

49 The modern friction material must exhibit moderate but stable friction, resistance to 'fade' on high duty and repeated applications, low wear rate to ensure long life, compatibility with drum and disc materials to avoid undue scoring, absence of squeal and judder and

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progressive braking response to increasing pedal pressure. It is composed of some 30% to 70% of asbestos fibre bonded in resin.

50 Not only are there national and international specifications, but each vehicle manufacturer and supplier has his own tests and specifications. Specifications cover primary friction levels and wear rate as well as other properties, particularly resistance to fade.

51 These products are expected to last several tens of thousands of miles which could extend over several years. They must give consistent and reliable performance throughout their life.

USE

52 Friction materials are used in almost all situations where friction braking is required (vehicles, trains, machines, etc).

EXISTING ALTERNATIVES

53 A number of alternative materials have been tried or developed for particular friction applications, although none have been found very suitable for ordinary motor vehicles. Examples are:

(a) Slag wool and steel wool, which were used by Germany during World War II when cut off from asbestos supplies, and which may still be used to some extent;

(b) sintered metals, used in brakes and clutches in heavy duty off-road machinery, such as agricultural machinery;

(c) ceramic materials, used in aircraft brakes;

(d) according to one manufacturer, there is quite a large range of alternative reinforcing fillers which can be considered for use in disc brake pads, although for drum brakes and clutch facings the number of possible alternatives is restricted.

A great constraint on the development of alternatives to asbestos brake linings is the close relationship between the present linings and the design of the whole braking mechanism and of the motor system.

NEED FOR FURTHER DEVELOPMENT

54 Any substitute must satisfy rigorous criteria, which have been agreed internationally. The need to develop non-asbestos friction materials has been recognised by the major manufacturers for some years. Research and development is being devoted more and more to this problem. Materials which would 'stop a car' are known to exist, but the provision of safe and reliable alternatives which give the progressive braking characteristics of present products and are capable of integration into present brake systems designed is not an easy process. More resources will be needed to develop alternatives quickly, although £1¼ million (some 50% of the technical effort of the asbestos brake industry) was spent in 1977 on it.

55 Brake failure alone accounts for 2% of the deaths on the roads each year. The research for substitutes must be carried out remembering that this figure should not be allowed to increase.

Asbestos yarn

CHARACTERISTICS

56 The most important characteristics of asbestos yarn are:

(i) Heat fire, chemical and rot resistance;

(ii) Fibre strength;

(iii) High modulus of elasticity, flexibility and toughness;

(iv) Low cost;

(v) Ease of application.

Large quantities of these yarns are processed in factories using plaiting and weaving machinery together with impregnation and coating plant.

57 The asbestos content of asbestos yarn is high (between 65% and 100%). Only chrysotile fires are used.

USE

58 Asbestos yarns have a great variety of uses, e.g. sealing applications in boilers and flues, heating element carries, caulking of cracks in brickwork and wiping of wire when galvanising.

EXISTING ALTERNATIVES

59 Glass yarns have replaced asbestos in some end-uses, probably at similar cost, but their uses are far too diverse for one alternative to be completely satisfactory. In particular, presently available glass yarns lack the heat resistance, and absorptiveness for resins and rubbers which are vital in many applications. Dust-suppressed yarns are becoming available commercially and should allow safer handling in some applications, not least by builders' merchants and shop assistants.

NEED FOR FURTHER DEVELOPMENT

60 We feel that research into substitutes should continue. In the meantime, a real benefit is often derived in normal handling from yarn that has been dust-suppressed or produced by the wet dispersion process and no extra cost involved. A substantial amount of plant for dust suppression and for wet dispersion processing has been installed and we wish to give every encouragement to the change away from textiles which are not dust-suppressed or wet dispersion based.

Asbestos cloth - safety curtains and blankets

CHARACTERISTICS

61 Asbestos cloths need to be flexible, non-flammable and to have low gas permeability. For metal splash protection they must not melt at temperatures up to

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1500°C, must not allow penetration of molten steel droplets and must show some adhesion for the molten metal. The products must meet the surface spread of flame requirements of BS 476 part 7 and for metal splash protection Admiralty specification DG Ships 307.

62 The asbestos content of asbestos cloth is high (between 65% and 100%). Only chrysotile asbestos is used.

USE

63 This material is mainly used for safety curtains, fire blankets, for heat retention during welding and for repairing castings, and for protection against molten metal splashes etc. It is used in some applications as a sound reduction material hung around a noise source.

EXISTING ALTERNATIVES

64 Glass fibre (or a layer of 'saffil' sapphire fibres between two woven glass cloths) may be used for safety curtains and fire blankets. We are unaware of alternatives for products for more severe conditions.

NEED FOR FURTHER DEVELOPMENT

65 These products account for a low percentage of asbestos fibre used in the UK. Attempts are being made to develop substitutes. There is no general alternative development as yet. Glass fibre can be used in low to medium temperature applications but not at high temperatures or where resistance to molten splash is required. For high temperatures, ceramic fibres will have the required heat resistance but they are much more costly as materials and their life in application may be limited by poorer mechanical properties and resistance to wear and vibration, than asbestos.

Asbestos/resin laminates in bearing and chemical plant equipment

CHARACTERISTICS

66 Asbestos/resin laminates are engineering materials and have high strength modulus, interlaminar shear, low friction and wear characteristics, together with low water absorption. BS 2572: 1976 contains specifications for phenolic lanimated sheet covers flatness, machinability, mechanical and electric strength properties and water absorption. The life of these products can vary from one year in a severe chemical plant application to several years in e.g. ships' rudder bearings.
These materials use about 500 tonnes of asbestos fibre each year.

USE

67 These products are used in bearings liable to high temperatures and pressures, chemical attack and water absorption.

EXISTING ALTERNATIVES

68 No adequate substitutes are available at present. Although carbon fibres might be able to meet some of the technical requirements, costs and lack of availability in quantity, severely limit their use in these applications.

NEED FOR FURTHER DEVELOPMENT

69 Work needs to be continued in developing suitable substitute materials.

Paints containing asbestos

CHARACTERISTICS

70 Asbestos fibres are added to paint to give it not only the ability to produce various textured finishes but also strength, ease of application, adhesion and opaqueness. The asbestos content is around 4% by weight.

USE

71 In practice paints containing asbestos fibres are applied to many surfaces particularly plaster/plaster-board surfaces, concrete and sand/cement rendering. We have been unable to obtain information on the full extent of its use, but application would appear to be very wide and in large quantities, particularly in housing.

EXISTING ALTERNATIVES

72 Although some manufacturers are beginning to produce asbestos free materials which they claim have similar properties to paints containing asbestos, current technical opinion is that substitutes still fall short of the desired performance.

73 There are other forms of paint additives and fillers available. Fibrous talc and asbestine may be used, but it should be ensured that there are free from fibrous asbestos materials.

74 Current costs of substitutes are around 20% higher and workability is about 15–20% less.

NEED FOR FURTHER DEVELOPMENT

75 We consider that more work needs to be done to develop suitable substitute materials in this field. With encouragement and sufficient research and development effort it should be possible to develop a cost-effective product that could replace the asbestos content of paints.

Asbestos sliver-cable wrap

CHARACTERISTICS

76 This material is a soft, 'comformable' strand having sufficient tensile strength for machine-wrapping. Heat resistance and thermal and electrical insulation properties adequate for the application are required. A

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life from a few months in a hot, abrasive environment to several years in a less severe application is expected.

77 Each cable manufacturer has his own specification covering linear density, asbestos content, electrical conductivity and packaging.
It accounts for about 100 tonnes of asbestos fibre each year.

USE

78 The main use of this product is a wrapping (as thermal and electrical insulation) around electrical cables, covered with a protective sheathing.

EXISTING ALTERNATIVES

79 We understand that little success has been achieved yet.

NEED FOR FURTHER DEVELOPMENT

80 Cable manufacturers have been seeking alternatives for some years but the small extent of substitution to date reflects the technical difficulties involved. No information is available on the costs of substitution. Effort should be continued in this field.

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Organisations and individuals who have submitted evidence to the Advisory Committee on Asbestos

5

L S Alderman
W L Armour
Asbestos Cement Manufacturers Association
Asbestos Cement Pipe Producers Association
Asbestos Fibre Importers Committee
Asbestos Information Centre
Asbestosis Research Council
Association of County Councils
BBA Group Limited
British Floor Covering Manufacturers Association
British Occupational Hygiene Society
British Railways Board
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Dr G H Jolliffe and Dr E J Shellard
Richard Klinger Limited
Marley Tile Company
Mr Max Madden MP, Chairman, 'Asbestos Action'
Metropolitan Borough of Calderdale
Ministry of Agriculture, Fisheries and Food
(Food Additives and Contaminants Committee)
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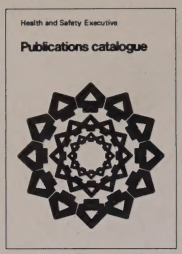
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