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Health Hazards of Asbestos: A Review of Recent Trends

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Health Hazards of Asbestos: A Review of Recent Trends

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Summary

In his 1966 Wyers Memorial Lecture Dr J. C. Gilson emphasized the main areas where further research was needed into the health hazards of asbestos. Since 1966 many of his recommendations have been carried out. This review attempts to examine recent trends in the study of asbestos and its effects on health by comparing them with the points made by Gilson and the action points documented by the Working Group on Asbestos Cancer (UICC, 1965). The relationship of the physical-chemical properties of asbestos fibres to the epidemiology of mesothelioma is discussed. The role of fibre shape and size is also referred to with regard to carcinogenesis. The main facts established by means of epidemiological investigation of exposed workers are examined. The theme throughout is that in the investigation of this problem the major achievements have been accomplished by a multi-disciplinary approach. The important role of the industrial medical officer as an epidemiologist and the practical application of his observations in the future control of the hazard are emphasized.

Introduction

This review of recent trends in the study of the biological effects of asbestos is presented with the object of summarizing events since the publication of J. C. Gilson's Wyers Memorial Lecture (Gilson, 1966). Prior to the lecture appearing in this journal, the account of the conference on the ' Biological Effects of Asbestos ', held in New York in October, 1964, was published (Whipple, 1965). Gilson accurately summarized the main points arising from that conference in the Wyers Memorial Lecture. Following the conference a Working Group on Asbestos Cancer was convened which published its report and recommendations (UICC, 1965). The recommendations were broadly classified under three main headings, namely:

1. Problems requiring epidemiological study,
2. Recommendations relating to pathology and experimental pathology, and
3. Recommendations relating to physics and chemistry.

The subject under review has been discussed at numerous international conferences since 1964, (Vienna, 1966; Tokyo, 1969 and Buenos Aires, 1972), organized by the Permanent Commission and International Association on Occupational Health. The Sub-Committee on Asbestosis of the above Commission met in September, 1970 (Sardinia, 1970). An International Symposium on the biological effects of asbestos was held in Dresden (Hammond and Selikoff, 1972) in 1968 and the subject of asbestos was discussed in depth at the International Conference on Pneumoconiosis in Johannesburg in 1969 (Shapiro, 1970).

In October, 1972 a Meeting of a Working Group to Review the Biological Effects of Asbestos was held in Lyon (WHO, IARC, 1972). In this article the progress reported will be considered to enable a summary of the current state of affairs to be presented.

Physics and Chemistry of Asbestos Fibres

Gilson (1966) pointed out that four main types of fibre are of commercial interest. Speil and Leineweber (1968) have reviewed the role of asbestos minerals in modern technology. Asbestos is a generic term for a variety of hydrated silicate minerals which have one common attribute, namely, the ability to be separated from the parent rock in the form of a fibre. The term ' asbestos ' applies to all minerals which fit the above description and ' asbestiform minerals ' is perhaps a better descriptive term.

There are two main classes of asbestiform minerals, dependent upon their basic crystal structure, namely, serpentine and the amphibole minerals. Chrysotile asbestos is the fibrous form of serpentine and is by far the most importar commercial variety accounting for more tha 95 per cent of the asbestos fibre produced at present. There are five main asbestiform varieties of amphibole, namely, crocidolite, amosite, anthophyllite, tremolite and actinolite.

The commercial uses of asbestos are dependent upon the physical and chemical properties of the various types of fibre. The biological effects of asbestos may therefore also be dependent upon these properties and this may account for notable differences in the incidence of asbestosis, lung cancer and mesothelioma in different industrial and mining situations.

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The 1964 Working Group recommended that standard reference samples of respirable size of amosite, chrysotile, tremolite, anthophyllite and crocidolite be prepared from as pure parent material as possible. Timbrell (1972a) reported that samples of chrysotile and crocidolite had been prepared in Johannesburg from donations by various asbestos producers. The two distributing centres for the 'UICC samples', Johannesburg PRU (Pneumoconiosis Research Unit), and the Medical Research Council PU (Pneumoconiosis Unit), Penarth, have recorded 250 recipients and many more researchers have obtained supplies from colleagues.

The UICC samples have been analysed for their important physical and chemical properties in several countries (Timbrell, 1970). The samples have been checked for homogeneity, contamination and oil content. The chemical composition of the reference samples has been compared with that of commercial fibre of the same type and has been found to be unchanged. The samples have been prepared in such a manner that the fibre length distributions will extend up to 200 μm., being comparable with asbestos fibre lengths detected in lungs. Longer fibres are undesirable because they impede the use of the sample for injection experiments. A special dispenser has been developed to generate dust clouds for inhalation experiments (Timbrell et al., 1968).

Gilson (1966) has stressed the importance of fibre length and aerodynamic equivalent diameter in determining the respirability of airborne dust. Timbrell (1972a) has reviewed the importance of the physical properties of asbestos in the aetiology of asbestos diseases. He states that the ability of long fibres (up to 200 μm.) to reach the alveolar regions, if they are thin enough not to be deposited earlier by sedimentation and impaction, explains satisfactorily the numerous long fibres found in lung sections. He points out that 'the epidemiological, pathological and experimental evidence presents a series of paradoxes which are useful in testing ideas concerning the aetiology'. The marked difference in physical properties between the asbestos minerals from the north-western Cape Province and the Transvaal may be a clue to the difference in the incidence of mesotheliomas in these two mining regions.

Timbrell (1972b) suggests that long fibres are preferentially deposited in the respiratory bronchioles at bifurcations and that this may explain why fibrosis tends to be associated first with respiratory bronchioles and with long fibres. Rats exposed to dust clouds of chrysotile and amphibole UICC samples at equal concentration retain six times more amphibole than chrysotile after six months. Timbrell suggests that this may be partly due to the characteristic 'rectilinear' shape of amphibole fibres compared to the 'curly' morphology of chrysotile fibres, allowing the amphiboles to penetrate to deeper parts of the lung more efficiently than chrysotile fibres. He has demonstrated this by means of studies using lung casts. A reasonable theory has thus been proposed to explain the reason for the development of mesotheliomas. It is based upon the ability of certain types of fibre to penetrate deeper into the lungs than others and to reach the pleural cavity by direct penetration. Timbrell points out that in rats exposed to dust clouds of UICC samples at equal concentrations and killed immediately after a 'short exposure', small numbers of crocidolite fibres have been detected near the visceral pleura, fewer amosite fibres have been found in this region, still fewer anthophyllite fibres and chrysotile fibres have very seldom been found near the pleura. Work is currently in progress using radioactive tracer studies to attempt to confirm this observation. It is possible that short, straight, chrysotile fibres may behave aerodynamically like amphibole fibres.

The significance of Timbrell's theory regarding the role of interception and penetration in the ultimate deposition of asbestos fibres, either in respiratory bronchioles or near the pleural surface, is that it offers a reasonable explanation for the difference in incidence of pleural mesothelioma in crocidolite exposed populations as opposed to chrysotile exposed populations. The difference between the incidence of mesotheliomas in the north-west Cape and the Transvaal (where tumours are very rare), may be related to the fact that differences in particle size in the crocidolite from these regions affect their aerodynamic behaviour and deposition in the lung. It has been shown that Transvaal fibres, both crocidolite and amosite, are on average three times the diameter and three times the length of north-western Cape crocidolite fibres. Hence a sample of north-western Cape asbestos will produce twenty-seven times the number of fibres as the same mass of

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Transvaal asbestos. Transvaal asbestos liberates less respirable fibres, fibres have a higher settling rate and hence shorter time available for inhalation and they have less efficient penetration to the deeper parts of the lung than north-western Cape asbestos. The latter characteristic may explain the difference in incidence of mesothelioma in the two regions.

At the Lyon Conference, the Physics and Chemistry Panel of the Advisory Committee on Asbestos Cancers, in the report which it makes to the Director of the IARC, suggests a number of future studies on the preparation of asbestos samples from different geographical areas to attempt to determine the influence of fibre size and shape on carcinogenicity (WHO, 1973). Information is required to help to identify the biologically important size fraction and to help with the interpretation of epidemiological and pathological studies. Dust counting methods in occupational and environmental studies require to be standardized and data collected over an extended period will be valuable in correlating the health hazard with epidemiological evidence.

Experimental Pathology and Pathology

Gilson (1966) has discussed the importance of the renewed interest in experimental asbestosis. The problems enumerated by him in the Wyers Memorial Lecture may be summarized briefly as follows:

1. Why is inhaled chrysotile so much more rapidly eliminated than the other three dusts?
2. What is the role of particle length in the development of fibrosis and to what extent is this independent of, or dependent upon, mass, surface area and the number of fibres inhaled?
3. What is the role of phagocytosis in the clearance mechanism?
4. How is the asbestos body involved in the pathogenesis of fibrosis?

Not all these questions can be answered as yet, but some plausible theories have emerged to explain some of them. Most of the constituents of asbestos can be made radioactive by neutron irradiation in a nuclear reactor. Morgan and Holmes (1970) have applied radiometric techniques in their investigation of the composition of asbestos and its behaviour in biological systems. In studying lung clearance they have found that their technique may be used satisfactorily in inhalation experiments. Comparatively large amounts of asbestos are removed relatively rapidly from the respiratory tract by ciliary action. This method is being used in an attempt to discover the mode of deposition of fibres in different areas of lung. Langer et al. (1970), using electron microprobe analysis, have studied the inorganic composition of asbestos bodies and one of their conclusions is that biological interactions may tend to affect chrysotile much more than amosite or crocidolite. Asbestos bodies obtained from the ashed lung tissue of an asbestos worker, known to have been exposed to amosite until ten years before his death, were analysed and compared with asbestos bodies from ashed lung tissue of an asbestos worker who was employed in the Canadian chrysotile fields. Their data indicate that amosite has remained chemically unaltered in at least ten years of biological residence, whereas chrysotile bundles have tended to split into sub-microscopic fibrils. The chrysotile fibres break down into units not easily resolved by the probe and they lose magnesium readily. Morgan and Cralley (1972) have recently documented the inorganic mineral content of asbestos and the solubility of the principal types. Chrysotile loses magnesium from the crystal lattice *in vivo* at a finite rate and the effect of this on the cytotoxicity and carcinogenicity of chrysotile requires further investigation. The solubility of the constituents of chrysotile asbestos, as demonstrated by radioactive tracer techniques, associated with the leaching of magnesium, confirm that this asbestos fibre type is chemically unstable in biological environments (Morgan and Holmes, 1970). The marked physical changes which take place *in vivo*, when chrysotile fibres, composed of bundles of fibrils, tend to break open and disperse into ultimate fibrils, may account for its clearance from the lungs. Such effects have not been observed in *in vivo* studies with amphibole asbestos types, e.g. amosite (Langer et al., 1970).

The experimental work briefly reported on thus far, in conjunction with the work on the effect of shape on particle penetration and retention in animal lungs, indicates that, apart from phagocytosis, chrysotile fibres are cleared from lungs because:

1. Interception of long, thin, curly fibres occurs in bronchioles with ciliated epithelium, and these fibres are then partially cleared by the cilial action,
2. Chrysotile is chemically unstable and its constituents dissolve in body fluids, and

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3. It is physically altered in biological residence enabling clearance by (1), (2), or phagocytosis to be more readily achieved.

The role of particle length in the development of fibrosis and the other biological effects of asbestos has not been resolved. This factor must be considered in conjunction with the fibre type and the effects of asbestos on macrophages and other cells.

It is now generally accepted that certain types of asbestos can interact with cell membranes. MacNab and Harrington (1967), Secchi and Rezzonico (1968) and Schnitzer and Pundsack (1970) have demonstrated the marked haemolytic effects of chrysotile and have confirmed the absence of marked haemolytic properties in the amphiboles. Allison (1972) has studied the effects of asbestos particles on macrophages, mesothelial cells and fibroblasts. He has found that, irrespective of the asbestos type, short fibres ($<5\,\mu m$) are readily and completely taken up by phagocytosis whereas long fibres ($>25\,\mu m$) are never completely taken up. Part of the long fibre remains out of the phagocyte. Two or more cells can sometimes be seen attached to a single long fibre and there is a suggestion that cell fusion may eventually occur. Particles of intermediate size (5–25 μm) are sometimes completely ingested and sometimes not. Allison has shown that chrysotile can exert an early cytotoxic effect soon after entering the cell because of interaction with the plasma membrane or a late effect as a result of disruption of the lysosomal membrane. When asbestos particles are added to organ cultures of mesothelium, these cells prove to be highly phagocytic, showing delayed cytotoxicity to high doses of chrysotile. They tolerate small doses of crocidolite well.

The emphasis in experimental pathology has shifted from asbestosis to carcinogenesis. Stanton (1972) has shown that fibres about 20–200 μm in length or 0·5–5 μm in diameter are more carcinogenic than larger or smaller fibres. This applies to both asbestos fibres and to glass fibre particles (the latter having been specially prepared for experimental purposes only), and Stanton believes that carcinogenicity of such particles seems to be related to their shape and size rather than to their physico-chemical properties. Mesotheliomas can be produced experimentally by pleural implants of asbestos, glass fibre and aluminium-oxide fibres of similar size. Maroudas et al. (1973) in an elegant experiment *in vitro*, have shown that the lengths of glass fibres which will induce growth of fibroblasts in culture correlate with the lengths of asbestos and other mineral fibres which will induce mesothelioma in the rat.

Particles smaller than 20 μm. in length do not induce growth *in vitro* or mesothelioma *in vivo*. The experiments of Rajan et al. (1972) and Maroudas et al. (1973) demonstrate the value to be derived from the study of organ culture and cell culture systems in investigating the effect of various fibres, chemicals and carcinogens in a relatively short period.

The significance of labile cations attached to the silicon-oxygen lattice, metallic contaminants, and adsorbed organic compounds (of geological origin or from contamination during commercial handling) is being investigated. Harrington (1962) has demonstrated the presence of oils in asbestos samples, one of which is 3,4-benzpyrene. Harrington (1965) has also described the possible contamination of asbestos fibres by jute oils. Gibbs (1970) has shown that fibre stored in polyethylene bags may become contaminated with 'oils'. Wagner et al. (1970) have shown that the presence or absence of the oils found by Harrington does not appear to alter the incidence of mesotheliomas produced by intrapleural inoculation in rats. Reeves et al. (1971), reviewing progress in experimental asbestos carcinogenesis, point out that the major metallic components present in asbestos are aluminium, magnesium and iron. Trace levels of chromium, nickel and cobalt are regularly found. While iron itself is not regarded as carcinogenic, experimental tumours have been produced by iron-dextran complexes, where the bonding of iron to the polymer may have been similar to that in asbestos (Richmond, 1959 quoted by Reeves et al., 1971). The trace levels of chromium and nickel in asbestos may play a role as environmental carcinogens. Dixon et al. (1970) reported that Fe^{3+} and Cr^{6+} both of which can be found in chrysotile asbestos, inhibit the action of benzpyrene hydroxylase, a detoxifying enzyme that protects the organism from the effects of benzpyrene. The interesting possibilities which this observation opens up with regard to asbestos carcinogenesis and the role of tobacco smoke have not yet been investigated in any depth.

Since Gilson posed the questions regarding the role of asbestos bodies in the pathogenesis of

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fibrosis, a vast bibliography has emerged on this subject. Pooley (1972) has reviewed the formation of asbestos bodies and using the electron microscope has studied the composition and character of such bodies extracted from human lungs. The asbestos bodies studied have come from the lungs of persons exposed to the four types of commercial asbestos fibre. He has found that bodies rarely form on fibres less than 10 μm in length, and practically always on straight fibres. They occur less frequently on chrysotile fibres than the other varieties. Results from all the lung specimens show that bodies always coexist in the material together with uncoated fibres and that uncoated fibres are far more numerous than coated fibres. When amphibole fibre is present there is very little difference in the ratio of coated to uncoated fibres, but in those tissues containing chrysotile the ratio of uncoated to coated fibres is very much larger by comparison. Ashcroft and Heppleston (1973) have attempted to assess asbestos concentrations in lung tissue showing various degrees and forms of fibrosis. In mild and moderate asbestosis they find a progressive increase in concentration of asbestos fibres, both coated and uncoated, with increasing severity of fibrosis, whereas in severe asbestosis no correlation exists between the fibre concentration and the form or the extent of the pathological reaction.

It is obvious that progress has been made in answering some of the questions which Gilson posed, but further study is required. The Advisory Committee on Asbestos Cancers in their report to the Director of the International Agency for Research on Cancer have recommended that work be done on methods for determining the amounts, types and structural features of asbestos in tissue and that the pathological assessment of the severity of asbestosis be standardized. It was felt that lung carcinoma in persons occupationally exposed should be compared with lung carcinoma in non-exposed individuals. The purpose of this study would be to examine the incidence of tumours at various sites of origin in the lung and the cytology of such tumours, and to correlate these with the presence or absence of asbestosis. Smoking habits would be noted in this study to assess the influence of the habit on the incidence of tumour varieties in the two populations. It was suggested that a comprehensive atlas on mesotheliomas should be produced to improve diagnostic consistency.

A significant proposal made by the Advisory Committee is that the possibility of monitoring populations exposed to asbestos by immunological methods should be investigated to ascertain whether it is possible to recognize those who are developing, or will develop, tumours. Turner-Warwick (1972) states that preliminary analysis of her study, undertaken to identify and define some of the immunological host responses in persons exposed to asbestos, supports the suggestion that antinuclear antibody (ANA) acts as an accelerator of fibrosis once it has been initiated by a separate agent.

Epidemiology

This review of the health hazards of asbestos has thus far concentrated upon clarifying current concepts regarding the physical and chemical properties of asbestos and attempting to place these in perspective with regard to the pathological mechanisms involved in the aetiology of asbestosis, lung carcinoma and mesothelioma.

The physician engaged in the practice of occupational medicine is perhaps more conscious of his role as an epidemiologist, in its widest sense, and may wonder at the relegation of this important discipline to the concluding section of this paper. This has been done deliberately, as the epidemiology of asbestos-induced lesions will be all the better understood after examining the aetiological considerations outlined in the preceding sections. The practical lessons to be learned from the epidemiological studies thus far published relate to the monitoring of the industrial environment, the surveillance of exposed individuals and the role of industry in monitoring and controlling the effects of its products on the general environment.

Epidemiological evidence since 1964 has accumulated rapidly from many countries and illustrates the numerous occupations in which asbestos exposure may occur. The presence of asbestos in the urban environment, in filters used for the manufacture of beverages, beers, wines, etc., and its presence in drugs given parenterally, has been documented (Thomson et al., 1963; Cunningham and Pontefract, 1971; Rickards and Badami, 1971; Nicholson et al., 1972; Selikoff et al., 1972b; Rickards, 1973).

Knowledge has increased with regard to the role of different varieties of fibre and is no longer limited to mining populations (Gilson, 1973). A few small groups of factory workers exposed to one type of fibre only have now been identified

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and are being studied (Selikoff et al., 1972a). The British Occupational Hygiene Society published its Hygiene Standards for Chrysotile Asbestos Dust in June, 1968. The data used were scanty, and based on factory experience of continuous exposure during working hours at an asbestos textile factory situated in a conurbation with a high prevalence of chronic bronchitis. This, however, is the only population studied thus far where it has been possible, because of reasonable dust exposure records, to relate clinical findings and X-ray changes to dust counts. When the BOHS Committee on Hygiene Standards' Sub-Committee on chrysotile asbestos reviewed the Standard in 1973, it was recommended that no change be made at the present time but that the Standard be kept under review. The Standard is designed to reduce the risk of contracting asbestosis to 1 per cent of those who have a lifetime's exposure to dust. By asbestosis the Committee meant the earliest demonstrable effects on the lung due to asbestos. The probability that the risk of being affected to this extent will be less than 1 per cent depends upon a cumulative exposure of 100 fibre years per cm^3. If a man works in a dust concentration of 2 fibres per cm^3 for 50 years, he should have less than a 1 per cent risk of developing the earliest demonstrable effects on the lung due to asbestos. The size range of fibres to be counted, based upon pathological observation of fibres retained in the lungs, is expressed in terms of the number of fibres per cm^3 greater than 5 μm in length, as determined with the standard membrane filter method. In industrial practice it is customary to count fibres in the 5–100 μm range.

The standard is derived from the correlation between the development of basal râles and dust exposure, while X-rays were not considered by the Sub-Committee to reflect the earliest effects of asbestos exposure on the lungs. Since the standard was produced an internationally recommended classification of the radiographic appearances of pneumoconiosis, suitable for use in the surveillance of asbestos workers has been developed (International Labour Office, 1972). The early use of this classification indicates that the chest radiograph may well be the most important means for the detection and monitoring of the biological effects of asbestos in the lungs. Systematic studies have allowed wider recognition of the fact that bilateral pleural plaques, pleural thickening and pleural calcification are more often caused by asbestos inhalation than was previously recognized. The use of this classification has shown the importance of separating rounded and irregular opacities, pleural thickening and pleural calcification. Rounded small opacities are more prevalent where exposures have been to other mineral dusts as well as to asbestos, in particular silica. They relate more closely to estimates of past dust exposure intensity. Irregular opacities have not yet been related to the mass of asbestos or other dusts in the lung, and may well be an index of tissue response rather than the amount of dust present. In view of the development of this radiological classification and its apparent effectiveness in demonstrating early changes due to asbestos exposure, the next review of the Standard which is undertaken may have to be based on such changes rather than basal râles.

The evidence for a dose-response relationship for asbestosis appears to have been confirmed in the textile section of the asbestos industry by the decreasing incidence of the disease following the introduction of the Asbestos Industry Regulations (1931), which took effect in 1933 (Smither and Lewinsohn, 1972). In other industries, not covered by the Regulations, the incidence of asbestosis appeared to increase, and case-finding exercises in naval dockyards (Harries, 1968) and elsewhere added to the number. The Asbestos Regulations 1969 were made and took effect in May 1970. These Regulations were designed to apply to all manufacture or uses of asbestos where dangerous concentrations of dust are found. The standards to be applied in the interpretation of these Regulations are to be found in Technical Data Note 13, published by HM Factory Inspectorate in the Department of Employment (1969). Recognizing the apparent difference in biological effects between chrysotile and crocidolite, the Regulations specify certain stringent conditions relating to the use of the latter and impose a tighter standard in TDN 13. It is the hazard of malignant disease which stimulates the continued interest in the biological effects of asbestos. Merewether (1949) first demonstrated a clear-cut association between asbestosis and lung carcinoma. Over 50 per cent of asbestosis sufferers die as a result of lung cancer and in populations where asbestosis still occurs may be the main cause of excess deaths (Buchanan, 1965). It has been shown that where the level of exposure is reduced

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and there is a lowered incidence of asbestosis, the cancer risk is also considerably reduced but not completely eliminated (Knox et al., 1968). The incidence of lung cancers in cigarette smokers exposed to asbestos indicates that smoking may be an important, if not essential, co-factor in this situation (Hammond and Selikoff, 1972).

Malignant mesothelioma of the pleura or peritoneum is perhaps the most important condition requiring detailed epidemiological study. In The Harold Dorn Memorial Lecture (1970) Richard Doll quotes Dorn as follows:

> The absence of reliable statistical data concerning variations in the incidence of cancer partially explains the existence of conflicting and inconclusive theories about the etiology of human cancer, since most of these theories are derived from the analysis of clinical data which fail to satisfy the requirements of statistical adequacy.

The information regarding the incidence of malignant mesothelioma is incomplete, the dust concentrations required to produce it are not known and the diagnostic criteria have not been finalized. It is now known that the fibre type may be important in the aetiology of this tumour but thus far only anthophyllite appears to be innocent. A proportion of mesotheliomas is apparently not related to past exposure to asbestos. In order to determine the exposure risk it is necessary to estimate the dust concentrations and to identify the fibre types to which occupational groups have been or are exposed. Similar data are required to quantify the risk to the general population said to be minimally exposed. The emergence of a possible public health risk is due to the finding of asbestos bodies in random autopsy series, the neighbourhood and domestic exposure cases described and the recent interest in asbestos in the urban environment. As has been commented on in the earlier pages of this review, on epidemiological evidence, crocidolite from the north-west Cape Province of South Africa (and from mines no longer operational in Western Australia) seems to be the most likely fibre to produce mesothelioma. Crocidolite from the Transvaal seems less likely to do so, the risk is also less with amosite and apparently less with chrysotile as well. This difference may be explained by Timbrell's theories already referred to.

Before concluding this section I should like to summarize the main conclusions to be drawn from epidemiological studies to date:

1. Asbestosis appears to be dose-response related and is caused by all fibre types.
2. Lung cancer is responsible for the excess deaths in populations where asbestosis occurs and may be associated with more than 50 per cent of asbestosis cases.
3. Lung cancer incidence decreases with the decline in incidence of asbestosis but has thus far not been entirely eliminated.
4. Cigarette smoking and significant asbestosis exposure have a multiplicative effect in the causation of lung cancer.
5. Mesothelioma appears to occur more often in association with crocidolite from the north-west Cape whereas Transvaal crocidolite is far less dangerous. Only anthophyllite is innocent of causing this malignant tumour thus far.
6. Before denying a public health risk from exposure to asbestos in the urban environment, in food and in fluids and beverages, detailed epidemiological and pathological investigations are required. On the evidence available to date, and in view of the negligible quantities of asbestos detected in the urban air, in foods and in fluids and beverages, the Advisory Committee on Asbestos Cancers of the IARC believes that there is no risk to the general public.

Conclusions

The study of the biological effects of asbestos cannot be conducted unless a multi-disciplinary approach is adopted. The practical application of this approach is to be found in the medical surveillance programme design, the environmental monitoring programme and the detailed scrutiny of morbidity data, mortality records and dust count figures in the asbestos industry.

The future need is for the development of objective methods for the early detection and surveillance of the effects caused by asbestos. The following are listed by the Advisory Committee:

1. Immunological techniques for screening for fibrosis and neoplasia.
2. Functional tests of changes in the peripheral airways.
3. Detection of pleural thickening.
4. Assessment of the specificity of small irregular opacities in the chest radiograph as defined in ILO U/C Classification, 1971.
5. Tests of the usefulness of different techniques

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of chest radiography, including the use of 100 mm. films.

6. Development of statistical procedures for analysis and presentation of serial observations.

The diagnostic criteria for asbestosis have not been greatly altered since the days of Merewether (1930) except for the addition of lung function tests as confirmatory evidence of pulmonary fibrosis. Correlation between the dust content of the factory environment, the dust content in the lungs, radiographic appearances, physical signs and physiological changes is urgently required. The reason for the apparent differences between miners and millers of asbestos and asbestos textile workers might be forthcoming from such detailed studies.

The Department of Employment has commenced a prospective epidemiological survey of asbestos workers which should assist in clarifying the relationship between fibre type, dust concentration, radiological changes and physical signs. The data will be analysed after taking into account individual smoking habits, age, sex and dust exposure besides asbestos in order to determine their combined effects.

The value of all this research to the industrial medical officer is that it enables medical surveillance programmes designed to monitor specific information to be developed, and thereby gives purpose and direction to his efforts on behalf of the exposed worker. There is, as yet, no cure for asbestosis other than that effected by the engineer controlling the emission of dust from industrial processes and reducing it to acceptable safe levels as indicated by epidemiological data obtained from studying those at risk. Asbestos is a useful mineral with unique properties applicable to modern technological developments more than ever before. There are substitutes for some of its conventional uses, but there are many uses of asbestos where no substitute will do the task as effectively or as cheaply. Providing asbestos can be used under controlled conditions there would seem to be no reason for discontinuing its production.

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