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DISEASES ASSOCIATED WITH THE INHALATION OF ASBESTOS DUST

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Examination of publications about asbestosis shows that the disease is caused by breathing asbestos fibres longer than 10—15 μm. Shorter fibres are cleared from the lungs by phagocytes. Soft chrysotile is much more active in the lung in causing asbestosis than any other varieties of asbestos. The relevance of the alleged higher solubility of chrysotile, compared with other varieties of asbestos is uncertain because it is not toxic to phagocytes and the fibres can be extremely fine and difficult to detect, even with a good transmission electron microscope. Solution may result in fragmentation of fibres into short lengths rather than reduction in diameter.

Lung carcinoma is also caused by long soft fibres of chrysotile and is peribronchiolar in origin because the curled, splayed fibres do not work their way through the lungs with the aid of the cyclic respiratory movements. Hard, needle shaped, smooth fibres of the amphiboles travel through the lung tissue and, if long enough to escape phagocytosis, cause carcinoma.

Mesothelioma of the pleura is caused by long inhaled fibres of amphibole which have travelled from the airways to the pleura. It can be caused by direct injection or application to the pleura of animals of chrysotile asbestos fibres of any length but does not result from inhalation because the curled, splayed fibres of soft chrysotile anchor themselves in lung tissue and do not travel to the pleura.

Selective sampling of airborne asbestos dust is necessary to pick out the small proportion of disease-causing fibres from the considerable amount of short fibre dust which is disposed of by phagocytes before it can do any harm.

A selective sampler must sample and retain fibres longer than 10—15 μm, and with diameters in the range 0.1—0.5 μm. It is useless to select on the basis of aerodynamic diameter because such a criterion is insensitive to the length of fibre. Some recent selective sampling systems are described.

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1. INTRODUCTION

There is still some uncertainty about the reasons why breathing asbestos may result in three different diseases, asbestosis, lung carcinoma and mesothelioma. The following review of the last fifty years' experimental work on animals and observations of persons who have breathed asbestos dust, shows that the differentiation of the three diseases can be accounted for, although some of the experiments could have given more help in this direction had they been planned intuitively rather than statistically.

The early realisation of the importance of long fibres failed to prevent the use of gravimetric sampling methods which persisted into the 1970's; they are useless as an index of health risk (1) and have now been replaced by the method of counting fibres on membrane filter samples which is sound but tedious (2).

Asbestos fibres can be too fine for detection by optical microscopy. Use of scanning electron microscopy on membrane filter samples has been described by *Spurny and co-workers* (3) who employed electron microprobe analysis for the identification of the varieties of asbestos and other fibres.

The next development, aimed at speeding up the evaluation of samples, is for selective sampling on a basis of fibre length. When this has been achieved, gravimetric assessment should be possible. Moves in this direction are described in the penultimate section of this paper.

Advances in the understanding of asbestos diseases come mainly from experimental studies of lung residues. Techniques of digestion of tissue for the recovery of inhaled, deposited and retained dust have been discussed by *Weller* (4).

2. ASBESTOS: DISEASES AND HEALTH STANDARDS

Asbestos minerals have been exploited on an industrial scale since 1878. Fibrosis of the lungs caused by inhaling asbestos dust was first recognized in 1900 and named asbestosis about 1927. Association of the airborne dust with lung cancer dates from 1934 and was demonstrated statistically by *Doll* in 1955 (5); mesothelioma was related to asbestos about 1950 (6, 7). In 1930, asbestosis was recognised as a compensatable industrial disease. In 1968 Hygiene Standards for Chrysotile Asbestos Dust were published by the British Occupational Hygiene Society (8); they were based on a study of asbestosis due to industrial exposures and calculated on a 1% chance of a person contracting asbestosis after an accumulated exposure of 100 fibre years per cm^3 of air, the fibres being over 5 μm in length, diameter not being stated. Ignorance of any quantitative relationship between exposure to asbestos dust and risk of cancer of the lung or of mesothelioma made it impossible to specify standards of exposure for these diseases.

In 1970, New Asbestos Regulations were imposed in the U. K., which amounted to fixing the permitted airborne concentration at not more than 2 fibres per cm^3, a fibre being 5 μm or more in length and having a ratio of length to diameter of at least 3 : 1 (9). This concentration was halved in the Final Report of the Advisory Committee on Asbestos (10). Their own 1968 Standards were endorsed by the British Occupational Hygiene Society (BOHS) (11) and a similar Standard for Amosite Asbestos was published at the same time. In 1983 the BOHS studied physiological effects associated with asbestosis in more than 600 persons who had been exposed in factories for over 10 years (12). As a result, for fibres exceeding 5 μm in length, the 1968 airborne level of 100 fibre years/cm^3 was reckoned to produce 17 to 20% recognizable occurrence of adverse effects, rather than the 1% chance of asbestosis given in 1968. For 25 fibre years/cm^3 the chance of adverse effects was now 2% and for 50 fibre years/cm^3, 7%. Directives of the European Communities followed in 1983 and 1984 which were discussed by *Stellingwerf* (13).

3. FIBRE LENGTH AND ASBESTOSIS

The fact that only fibres above a certain length would cause asbestosis was established by L. U. Gardner at Saranac by his classical series of animal experiments with chrysotile and other types of asbestos. He worked through the 1930's but published little; his results were reviewed after his death in 1946 by *Vorwald and co-workers* (14). They concluded that some species of animal, on exposure to asbestos fibres from 20 to 50 μm in length, developed peribronchiolar fibrosis of the lung similar to human asbestosis. Shorter fibres did not provoke this reaction; the upper limit of length was not determined, but would depend on the inhalability of fibres longer than 50 μm. It was also concluded that the formation of asbestos bodies in the lung was due to the coating of fibres by blood and tissue elements, which resulted in loss of ability of the fibres to produce fibrosis.

These conclusions were challenged by *Beattie and Knox* (15) and *Beattie* (16), who examined small parts of the lungs of men who had worked with asbestos; they failed to relate the severity of asbestos lesions to the mineral content of dried lung, probably because they were unable to ascertain the weight of mineral in the fresh whole lung. They proposed a theory of the formation of asbestos bodies on fibres and the subsequent disintegration of the bodies with release of a fibrogenic agent; this theory is unattractive because asbestos bodies are rare in subjects exposed to chrysotile which represented about 92% of the European industrial consumption of asbestos (17) and there is no correlation between the number of asbestos bodies found in sputum and the severity of asbestosis (18); also, asbestos bodies are rarely found in rats with asbestosis which has resulted from inhalation (19).

It is fairly obvious that long fibres are dangerous because phagocytes are unable to transport them (6, 20). Short fibres accumulate in the lymphatic system, where they cause only mild fibrosis (21).

Conning and co-workers (22) show a correlation between the production of asbestosis in rats and the cytotoxicity *in vitro* of the same dust to cultured macrophages from similar rats. The chrysotile asbestos consisted of fibres from 1—3 μm diameter and 0.5—200 μm long; unfortunately the dead cells were not examined so their mortality was not correlated with length of fibre. The toxic action of fibres of asbestos short enough to be engorged by macrophages is very much less than that of quartz particles; chrysotile is the most soluble variety of asbestos (not in alkali).

Evidence of the handling by phagocytes of asbestos of several types, with fibres all shorter than 10 μm, is provided by the inhalation experiments with rats which were carried out by *Morgan and co-workers* (23). Fifteen to twenty percent of the fibres inhaled were deposited in the lower respiratory tract with total depositions of 30% to 75%. The fibre median diameters were from 0.15 μm to 0.46 μm. Of the initial deposits only about 5% remained 120 days after inhalation of the dust, either because they had been removed with the aid of phagocytes or had dissolved.

These observations are paralleled by the intrapleural injection experiments in rats by *Monchaux and co-workers* (24) using fibres of chrysotile and crocidolite, over 90% being below 10 μm in length. Ninety days after injection fibres were recovered from the lung parenchyma and the mediastinal lymph nodes. The chrysotile migrated less readily and produced a greater inflammatory reaction than crocidolite, each of these factors suggesting a connection with the characteristic form of soft chrysotile.

The amount of asbestos in the lungs should correlate with the amount of airborne dust. This is so for amphiboles but less chrysotile has often been found, indicating less deposition or more elimination (25). Chrysotile is the most soluble variety of asbestos and loss in this way has been given as the reason for its short lifetime in much published work. Long fibres, which become coated while in the lung and turn into asbestos bodies, sometimes appear to break into short lengths.

The inability of the lung to free itself of long fibres is commented on by *Timbrell* (26) who compared the distributions by diameter and by length of particles of anthophyllite asbestos, in the air of a mine in Finland, with particles recovered from the lungs of decreased employees of the mine. He believes that his data indicate complete clearance of short fibres with clearance decreasing as length increases, the retention becoming complete at about 14 μm length since the phagocytes cannot handle fibres which are greater than themselves.

There is ample evidence of the ability of occasional fine fibres as long as 100 μm to reach the finest airways of the human lungs (15, 27);

this is because of the tendency of fibres to align themselves along the flow direction, and to stay aligned if they drift to the axis of an airway, coupled with their very small Stokes' number, that is the ratio of the fibre stop-distance to airway diameter. The stop-distance is a measure of the tendency of the mass of a particle to keep it moving in the same direction whilst the fluid drag of the air, which carries it along, tends to hold it in the air stream when this changes direction. Small Stokes' number means that fibres oriented along the flow lines follow their course and do not often make contact with the walls of the airways.

Wagner and co-workers (25) present distributions of the fibre content of lung samples from men who had sought compensation for asbestosis. The median lung loading for chrysotile was only about 5.10^6 fibres/g tissue for all grades of severity of asbestosis. For the amphiboles some 40 times as many fibres were required for grade 3 asbestosis, the most severe. Ten times as many fibres were needed for grade 2 and only 0.2 times for grade 1. These ratios hint at a long term chemical activity in chrysotile.

A comparison of the effects of fibres of glass, chrysotile and of crocidolite asbestos is reported by *Pickrell and co-workers* (28). They were administered to hamsters by intratracheal instillation, doses ranging from 2 to 21 mg, of which 0.01 to 6.2 mg (2×10^5 to over 10^{10} fibres) had aerodynamic diameters below 5 μm. Lengths of the fibres are not given. One of the glass fibre samples (microfibre 2) had a bimodal distribution of diameter, 2/3 of the count had a mode at 0.1 to 0.2 μm and 1/3 at 1 to 1.3 μm; these fibres were of glass without binder. This sample of glass fibre produced more appreciable biological effects than other, coarser samples. The cytotoxicity to pulmonary alveolar macrophages was less than that of crocidolite but more than chrysotile. Like asbestos, instillation of microfibre 2 increased collagen in lung tissue, the maximal response being at 11 months. These observations indicate similar reactions to fine glass fibres and to asbestos. The different chemical natures of these substances suggests that their common reaction is due to their fibrous form.

4. FIBRE LENGTH, LUNG TUMOURS AND MESOTHELIOMA

Wagner and co-workers (19) exposed rats to airborne dusts of UICC standard reference samples of fine varieties of asbestos for periods up to 24 months. The characteristics of the fibres of these dusts have been described by *Timbrell* (29) and *Harris and Timbrell* (30). Mean mass respirable dust concentrations were measured in the exposure chamber by a selective gravimetric sampler which took small account of particles settling at more than 0.08 cm/sec (aerodynamic diameter 5 μm). The respirable dust concentrations ranged from 10—14 mg/m^3. Each kind of asbestos produced asbestosis and lung tumours. The weight of dust

in the lungs of animals breathing the amphiboles (amosite, anthophyllite and crocidolite) increased steadily during exposure up to 24 months. The weight of chrysotile found in the lungs of animals breathing this variety of asbestos dust for 24 months was only about 1/30 of that in the amphibole-breathing animals and did not change much after the first three months. The severity of asbestosis was much the same, irrespective of which dust had been breathed, and increased more or less steadily with the length of exposure. Lung tumours appeared after 10 months, when asbestosis was moderate. Mesotheliomas appeared in animals breathing the amphiboles and Canadian chrysotile but not with Rhodesian chrysotile; their appearance did not correlate with length of exposure.

Mesotheliomas have also been produced by the intrapleural injection into rats of 20 mg of »fine fibred« chrysotile (31). In this paper further inhalation experiments are also described in which aerosols of chrysotile having 500 fibres/cm^3 longer than 5 μm (10.8 mg/m^3 respirable dust) were breathed by rats for up to 12 months. Moderate asbestosis, some lung tumours but no mesotheliomas were recorded.

Bolton and co-workers (32) employed intraperitoneal injection of rats to assess the relative tendencies to produce mesotheliomas of fine chrysotiles and two amosites. The particles were collected by filtration from airborne dusts, 40% being longer than 2—3 μm and 2% longer than 12—20 μm. No distributions of fibre diameters are given but all were less than one third of the length. The chrysotiles were more aggressive than the amosites apart from a sample of chrysotile which had been heated to 850 °C. There was little difference in fibre length between the varieties so these results fail to confirm the association of length with carcinogenicity by *Stanton and Wrench* (33).

Gilson (7) reviewed human exposures resulting in lung carcinoma which indicated that the risk was lowest for chrysotile and was dose-related. He pointed out that asbestos is an absorptive material which may pick up hydrocarbons; smoking increases the risk of lung cancer in asbestos workers (34). This effect, however, is not specific to asbestos; uranium miners who contract cancer due to α-radiation also have an enhanced risk if they smoke. Mesotheliomas were rarely associated with exposure to chrysotile or anthophillite; crocidolite and amosite could cause mesothelioma. There are reports of this disease occurring in persons with no known exposure to asbestos. *Stumphius and Meyer* (35) accept that there is a connection between asbestos and mesothelioma but state that cases have occurred when exposure to asbestos could not be proved.

A survey of lung samples from chrysotile workers (36) showed that the ratio of tremolite to chrysotile in the lungs was much greater than in the inhaled dust, indicating either less deposition or more elimination of chrysotile. It is really essential for the understanding of such

observations that size analyses of fibre lengths should be presented. For example a great deal of short fibre (< 10 μm) chrysotile could have been eliminated from the lungs by phagocytosis or solution leaving significant long fibres in the lungs. It is inadequate to define fibres, as in this paper, by an aspect ratio of 3 : 1 or greater.

In a study of lung tissue from cases of mesothelioma *McDonald and co-workers* (37) also used this aspect ratio as a criterion. In the case of chrysotile workers no association of lung dust with mesothelioma was detected. For subjects with amosite and crocidolite asbestos there were correlations with mesothelioma which can be regarded as well established; however, there are other causes of mesothelioma which have nothing to do with asbestos.

Kannerstein and Churg (38), in a review of mesothelioma in man and experimental animals, emphasize the similar morphology of the disease in each species; they consider it probable that the physical form, but neither the chemical constitution nor the molecular structure of the fibres, is responsible for the tumorigenic effect on the serous membranes. They refer to contradictory findings regarding length of fibres, especially those below 10 μm, but fail to identify an important reason for this. Most animal mesotheliomas of the pleura have been induced by direct application of asbestos to the pleura, a procedure which guarantees the access of fibres of all lengths. When dust is inhaled the fibres can only reach the pleura by their mobility in moving lung tissue and by their escaping phagocytosis; two processes which depend on fibre form and length.

5. FIBRE DIAMETER AND ASPECT RATIO

Asbestos fibres cleave longitudinally down to diameters of 0.01 to 0.02 μm. It has been suggested (6) that 90% of the fibres in human lungs are below 0.2 μm so that they can only be studied by transmission electron microscopy which is a time consuming process.

A study of airborne chrysotile fibres in a textile factory was carried out by *Rood and Streeter* (39), sampling with membrane filters which were directly examined with a transmission electron microscope. Both lengths and diameters of the fibres were distributed near to lognormal with σ_g about 2.4. Lengths ranged from 0.5 to 20 μm (mode 0.74, median 1.6) and diameters 0.02 to 1.0 μm (mode 0.04, median 0.08) The aspect ratios were from 4 to 100 (median 20). They reckoned that 60% of all the fibres would have been invisible to a scanning electron microscope, which would show 70% of the fibres which were longer than 5 μm. A good optical microscope would reveal only 25% of the latter.

Timbrell (26) used scanning electron microscopy to examine samples of airborne dust and of lung dust from workers at an anthophyllite mine

in Finland which were taken before it closed down in 1975. This form of asbestos is an amphibole of low solubility having rigid, straight rod-shaped fibres with clean-cut ends, an ideal type for his magnetic alignment method. Some figures taken from his paper are shown in Table 1. The ranges of length and diameter of fibres recovered from lung tissue are very similar to those of the airborne fibres but the distributions show that the tissue fibres have more long ones and more of larger diameter. This suggests that the normal lung clearance operated better for the shorter fibres than for the longer ones.

Table 1.

Fibres in airborne dust and lung dust from workers at an anthophyllite mine, length and diameter distributions were approximately lognormal (26).

	Fibre length μm			Fibre diameter μm		
	Range	Mode	Median	Range	Mode	Median
Airborne dust	0.4—200	1.4—3.1	3.8—10	0.1—50	0.2—0.4	0.4—0.7
σ_g		2.4—2.9			1.9—2.2	
	Range	Mode	Median	Range	Mode	Median
Lung dust	0.5—100	2.7—7.0	—	0.1—4.4	0.4—0.5	—
σ_g		2.2—2.6			1.5—2.0	

Ayer and Zumwalde (40) give length and diameter distributions of airborne fibres of several varieties of asbestos associated with industrial processes.

It is claimed (31) that a super-fine chrysotile asbestos gives a high incidence of mesothelioma by intrapleural innoculation but no data on diameter and length are given. In the discussion it was stated that this material contained not only extremely fine fibres but also lumps of material compacted by milling; hence mass of injected dust is not of great significance without analysis of shape, length and diameter of fibres. This is another reason for rejecting size selection of inhaled dust by aerodynamic diameter.

Timbrell (26) presents perspective diagrams of bivariate distributions of airborne fibres, tissue fibres and lung retention related to fibre length and diameter. These emphasize the futility of the 3 : 1 aspect ratio (9, 36, 37) suggesting that a figure of 15 to 20 might be more realistic in pinpointing the most dangerous fibres. The difficulties of laboriously preparing quantities of fibres scaled in length and diameter for experimental purposes and of analysing lung residues over realistic ranges of length and diameter have held up progress. The use of selective mass

samplers of »respirable dust« (50% penetration at 5 μm aerodynamic diameter) is unlikely to advance the understanding of the asbestos related diseases.

The handling of bivariate lognormal distributions of diameter and length of fibres has been discussed by *Schneider and Holst* (41). There is some discussion of the possibility of finding fibre length, or length to diameter ratio from a series of measurements of penetration, over a wide range of values, of the same aerosol through a filtration system (42).

Attempts to relate the disease-causing potential in man of inhaled asbestos to the biological activity in animals, elicited by direct application of asbestos fibres to tissue, are misleading because the important features of transfer of airborne fibres to lung tissue and their motion therein are short-circuited. This was emphasised by *Robock and Klosterkötter* (43) who suggest that chrysotile has a greater cytotoxic effect than crocidolite whereas the latter is the greater health hazard because its fibres can travel through lung tissue. For example, *Pott* (44, 45) has presented 3-dimensional diagrams of fibre length, diameter and a carcinogenicity factor which do not give a reliable indication of human risk. The length and diameter, which are important factors in the human diseases because fibre transport through air and tissue is decisive, are doubtfully associated with his carcinogenicity factor which is based mainly on implantation of fibres in animals.

6. WHAT ARE THE FEATURES OF ASBESTOS DISEASES RELEVANT TO SAMPLING PRACTICE?

The following conclusions can be drawn from the experiments and observations which have been selected for mention in Sections 2 to 5 above; they are backed up by a considerable number of publications many of which are referred to in the papers quoted. In a sense, the vast body of research on asbestos diseases is disappointing since the important directions given by early experiments have not been followed up and there is a general confusion as a result. Perhaps this was inevitable because so many factors are involved; however, it would be advantageous for much more attention to be given to the detailed physical nature of the dusts used in animal experiments and of dust recovered from human lungs.

This factor is basic to the design of sampling apparatus which need only record a very small selected fraction of the total airborne dust which is inhaled by persons handling asbestos. The physical nature of the inhaled dust is dependent on the variety of asbestos; the dust deposited in the lungs depends rather broadly upon particle shape, length and diameter; the dust which causes the three diseases is more specifically related to particle shape, length and diameter, probably in a way which varies with the particular disease.

Accordingly, the selectivity of a satisfactory dust sampling system has to be much stricter than the selective sampling systems currently in use for insoluble dust, like quartz and coal, consisting of particles of relatively compact shape which produce illness only if they attain the alveolated region of the human lung. A bonus following the production of selective samplers for asbestos disease-causing fractions of airborne dust would be their adaptation for use as sources of supply of fractionated dust which could be used for animal experiments.

In thinking upon these lines the first consideration is the differences between the varieties of asbestos, physical rather than chemical because specific chemical activity takes second place to the physical factors which govern the transport of fibres to the connective tissues of the lungs, lymphatics and pleura. There are also indications that fibrous form, obviously for completely different reasons, may be a key factor in cytotoxicity, this being supported by recent work on glass fibres.

7. VARIETIES OF ASBESTOS AND FIBROUS HABIT

A good account of the physical nature and properties of the six varieties of asbestos has been given by *Badollet* (46) including photo and electron micrographs. Chrysotile (white) asbestos is a serpentine rock and crocidolite (blue) asbestos, amosite, anthophyllite, tremolite and actinolite are amphiboles. An important point made in this paper is the breakdown of chrysotile into three varieties. Harsh chrysotile has straight, brittle needle-like fibres which are not curved: his Russian asbestos was in this category. Soft chrysotile (Arizona) has thin curved threads, very strong and of a silky nature which, like all asbestos fibres, split longitudinally. Soft chrysotile is the most used in industry. The amphiboles resemble harsh chrysotile. A Canadian sample was classed as semi-harsh.

Hodgson (47) and *Ayer and Zumwalde* (40) give information about the occurrence, production and structure of asbestos varieties but omit to mention the distinct types of chrysotile. This may be important since the experimental production of mesotheliomas has been possible with amphiboles and Canadian chrysotile, but not with Rhodesian (19). The morphological differences are not described in the paper.

In general, soft chrysotile is assumed to have been used in experiments and recovered from human lungs; the possibility that harsh chrysotile, which is close to amphibole morphology, may have gone unnoticed and could account for anomalies is a real one.

No doubt each amphibole variety of asbestos must cover a range of fibrous habit, just like chrysotile. It is not enough to discuss variations of disease parameters solely in terms of variety, unbacked by fibre shape and size measurements. Chemical differentiation follows the latter.

Timbrell (48) has pointed out a considerable difference between African crocidolites. Fibres from North West Cape samples are thinner and shorter than those from the Transvaal; the latter fall under gravity nine times faster than the Cape fibres. This means, amongst other effects, that the heavier Transvaal fibres penetrate to the alveolated region of the lungs less than do the thin, shorter North West Cape fibres. This is associated by Timbrell with the risk of mesothelioma being greater for the thin, shorter fibres. It is doubtful if the attainment of actual alveoli is a prerequisite for the development of mesothelioma since fibres can penetrate the walls of lung airways; the crucial concern is ability to traverse the lung parenchyma, aided by the tissue movements of breathing.

8. MECHANISMS OF THE CAUSATION OF DISEASE

The curved, flexible, splayed fibres of soft chrysotile are up to 40 times more effective in causing asbestosis than the straight, rigid, smooth fibres of the amphiboles (25). A few of the inhaled fibres longer than 10 to 15 μm would pass in and out of the bronchioles (diameter 500 μm) near enough to the surface of these airways to make contact. The straight amphibole fibres would collide with the surface less frequently than chrysotile because they tend to align along the direction of airflow which is of too low a velocity to extend soft chrysotile fibres, these therefore have a better chance than needle-like fibres of striking and penetrating the surface of the airway, of encountering connective tissue near the surface of the airway and causing asbestosis.

The experiments with rats of *Wagner and co-workers* (19) in which both kinds of asbestos were dispersed indicated that asbestosis of much the same severity was caused by chrysotile and amphibole but there was much less chrysotile in the animals' lungs, the quantity not being dose-related. Concentrations were measured by a size selecting gravimetric sampler (50% penetration at 5 μm aerodynamic diameter) and the result demonstrates that this instrument is useless for asbestos. Clearly the chrysotile which caused the disease might collect in the horizontal elutriator and not in the »respirable« dust. Perhaps the chrysotile fibres were more clumped than those of the amphiboles, or perhaps they tangled with the plates and previously deposited fibres.

Lung carcinoma correlates with asbestosis (5). Asbestosis results from fewer fibres of chrysotile than amphibole (25); however (7) lung carcinoma follows chrysotile-induced asbestosis when there are more fibres in the lung than in the case of amphibole-induced asbestosis.

Mesothelioma necessitates the penetration of fibres from the wall of the bronchiole, through the lung parenchyma to the pleura. This is manifestly more probable for the needle-like fibres of amphibole than for the curved, splayed fibres of soft chrysotile which must tend to

anchor themselves in tissue. Cyclic movements of the lung would readily move amphibole fibres but not chrysotile.

These conclusions are summarised in Table 2. Study of the table suggests very strongly that a chemical factor is operative in the fibres which cause lung carcinoma and mesothelioma, possibly the same chain of reaction in each case. The animal experiments of *Pickrell and co-workers* (28) argue against a chemical factor being involved in asbestosis. Merle Stanton, in several publications, argues in favour of the fibrous nature prevailing over biochemistry as a cause of cytoxicity.

9. IDEAS FOR THE SELECTIVE SAMPLING OF AIRBORNE ASBESTOS

A gradual appreciation of the importance of the length of fibres, since the description of Gardner's work during the 1930's by *Vorwald and co-workers* (14), is leading to attempts to estimate long fibres in

Table 2.

*Mechanisms of causation of asbestos disease by fibres longer than 10—15 μm with diameters 0.1—4 μm.**

Disease		Variety of asbestos	
		Soft chrysotile, soft amphiboles	Harsh chrysotile, amphiboles
ASBESTOSIS	Amount in lungs	Small	Large
	Fibre transport by lung movement	No	Yes
	Location of lesion	Peribronchiolar	Distributed
CARCINOMA	Amount in lungs	Large	Small
	Fibre transport by lung movement	No	Yes
	Location of lesion	Epithelial, bronchiolar	Lobe, segments
MESOTELIOMA	Amount in lungs	Any	Small
	Fibre transport by lung movement	No	Yes
	Location of lesion	Disease rare (does not reach the pleura)	Pleura

* This table is confined to asbestos fibres too long to be transported by phagocytes which clean up deposits of shorter fibres. Entry of airbone fibres into alveoli is not essential for the causation of disease.

airborne clouds in which short fibres and compact dust particles considerably preponderate. Up to about 1974 the idea unfortunately persisted that gravimetric sampling through a horizontal elutriator or cyclone, with a 50% cut at 5 μm aerodynamic diameter, would suit asbestos dust as well as coal and quartz for which it was designed. This is not so. Aerodynamic separation of long and short fibres is not satisfactory for the selective sampling of airborne asbestos because it depends mainly on the fibre diameter, d, and relatively slightly on its length (49). This can be seen from Table 3 which indicates the small change in

Table 3.

Stokes' diameter of elongated particles divided by the diameter.

Length diam.	Theory		Experimental (free fall)		
	Prolate spheroids (oriented)				
	Motion ∥ to long axis	Motion ⊥ to long axis	Asbestos Crocidolite	(Stöber) Amosite	Glass (Timbrell)
10	1.94	1.62	1.88	1.65	1.7
20	2.18	1.77	2.12	1.78	1.8
50	2.48	1.96	2.48	1.99	1.9
100	2.68	2.09	2.79	2.15	2.0

Stokes' diameter, d_{St}, which results from a large change in the ratio of length to diameter of fibrous particles. Theoretically, for long cylinders, there is no change at all, the value of d_{St}/d being about 3, depending on the Reynolds number.

Experimental proof of the aerodynamic characterisation of fibres being by diameter rather than length is provided by experiments with a rather complicated Virtual Impactor designed and constructed by *Masuda and co-workers* (50). This divides an aerosol into three fractions according to the aerodynamic diameters of its particles. The results with asbestos clearly demonstrate separation more by fibre diameter than fibre length.

The Stokes' diameter of a fibre (or particle) is equal to the aerodynamic diameter divided by the square root of its density (v. Table 3) which has to be much greater than the density of air. These diameters are related to the rate of fall of the fibre under gravity and therefore depend on its orientation.

In the inertial spectrometer of *Prodi and co-workers* (52) clean air flows round a right angle bend and along a horizontal rectangular duct.

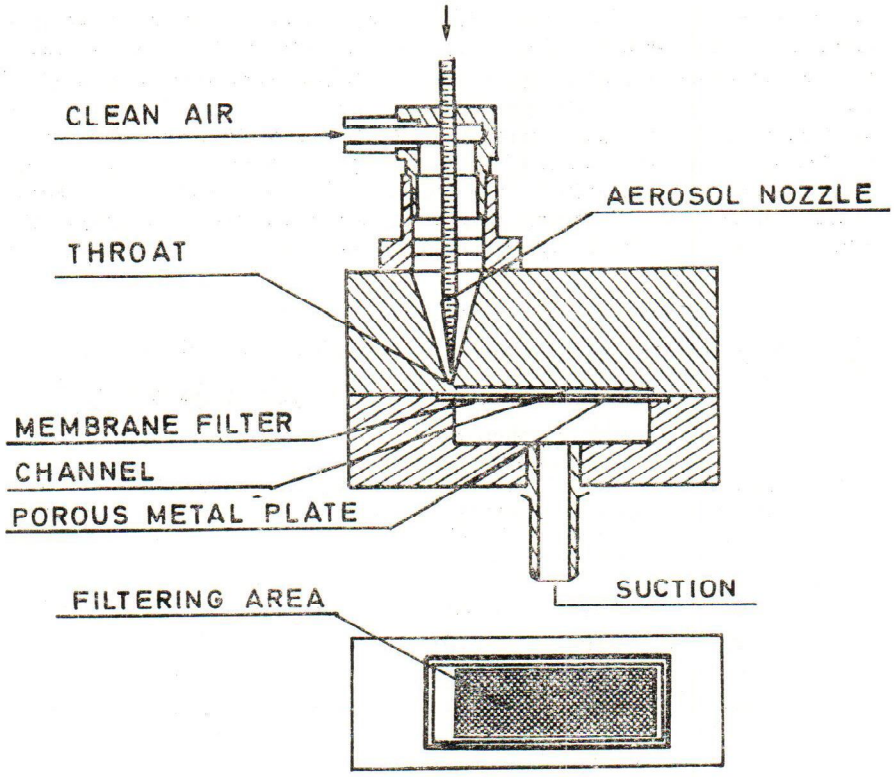

Fig. 1. *Inertial spectrometer of Prodi and co-workers (52). Fibres align parallel to the air flow mainly on account of the longitudinal acceleration of the air passing into the throat.*

Table 4.
Density of asbestos fibres

	Badollet (46)	Hodgson (47)
Chrysotile	2.4 —2.6 g/cm³	2.55
Anthophyllite	2.85—3.1	—
Tremolite	2.9 —3.2	—
Actinolite	3.0 —3.2	—
Amosite	3.1 —3.25	3.45
Crocidolite	3.2 —3.3	2.55

Upstream of the bend a thin stream of aerosol is injected and the particles are separated according to aerodynamic diameter as they round the bend. The separation is magnified after the bend as the aerosol flows along the duct of which the lower boundary consists of a membrane filter; the particles settle on the filter, the large ones not having so far to fall as the small owing to the inertial separation at the bend. When asbestos fibres were sampled with this device they were, as would be expected, graded by diameter in the deposit along the filter. However there was also a very complete alignment of fibres along the flow lines; both the velocity gradient normal to the filter and the acceleration of particles approaching the bend contributed to this (Fig. 1).

A commercially available Aerodynamic Particle sizer (APS 33, TSI Inc) also orients fibres in an accelerating jet of air. All particles lag behind the air, according to the aerodynamic diameter, on account of particle inertia. The instrument times each particle between two laser beams and from its velocity lag calculates the mass/drag ratio. Again it is the aerodynamic diameter which results so that the fibres are classed mainly on diameter. The instrument grew out of the work of *Mazumder and co-workers* (53), whose SPART analyser measured the lag of particles in air oscillating acoustically at about 27 KHz, using laser-Döppler velocimetry. *Wilson and Liu* (54) also used LDV to measure the velocity lag of particles in an accelerating jet, but LDV was abandoned in the commercial instrument in favour of the simple time of transit. *Hiller and co-workers* (55) used the original SPART analyser for measuring deposition in the human respiratory tract simultaneously for particles of two different sizes.

It is theoretically possible to separate fibres by length as was pointed out by *Ogden and Walton* (56). Cylinders fall under gravity, not vertically but at an angle to the vertical which is related to the inclination of the cylinder, which is maintained during falling, and to the length to diameter ratio. Experiments in glycerol showed that the angles agree with values predicted by theory for prolate ellipsoids of revolution. It is doubtful if this could be applied to asbestos fibres because the form is geometrically imperfect and the falling velocities of the fine fibres would be low and there would be diffusion.

Optical differentiation is possible and has been discussed by *Seger* (57). Fibrous particles when illuminated by a narrow beam in a plane of incidence at right angles to the fibre length scatter most of the light in the same plane. In his arrangement a laser beam was used and four detectors looked for scattered light. Presence of a fibre was indicated by a streak of scattered light in the plane of incidence. Compact particles produce equal scattering in all directions at right angles to the incident beam (Fig. 2).

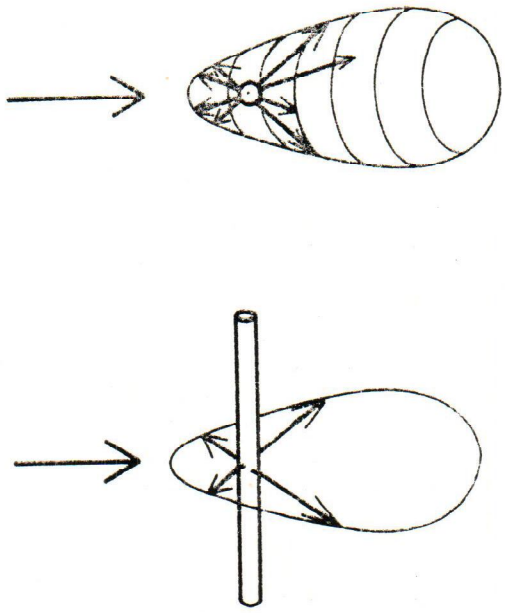

Fig. 2. *Scattering of light by a fibre and by a compact particle (simplified).*

Light scattering used in conjunction with magnetic alignment of fibres differentiates fibres clearly, on the above basis. Magnetic alignment of asbestos fibres has been studied by *Timbrell* (58—60). The fibres can only be aligned if they are suspended in air or liquid but the direction of alignment relative to the field is variable and unpredictable. In special cases this method is useful, but not for selective sampling. It has been applied, for example, in assessing a sample of asbestos on a membrane filter by measuring the light scattered forwards up a microscope tube while rotating the magnetically aligned fibre preparation (Vickers, 1788 rapid fibre counter); there have been difficulties with fibre counts below 100 mm^{-2} (61).

Selection by electrical mobility was first tried by *Zebel and co-workers* (62). The aerosol was fed past a corona discharge producing positive ions which were picked up by particles. The particles then passed through a parallel plate electrical mobility spectrometer in which the

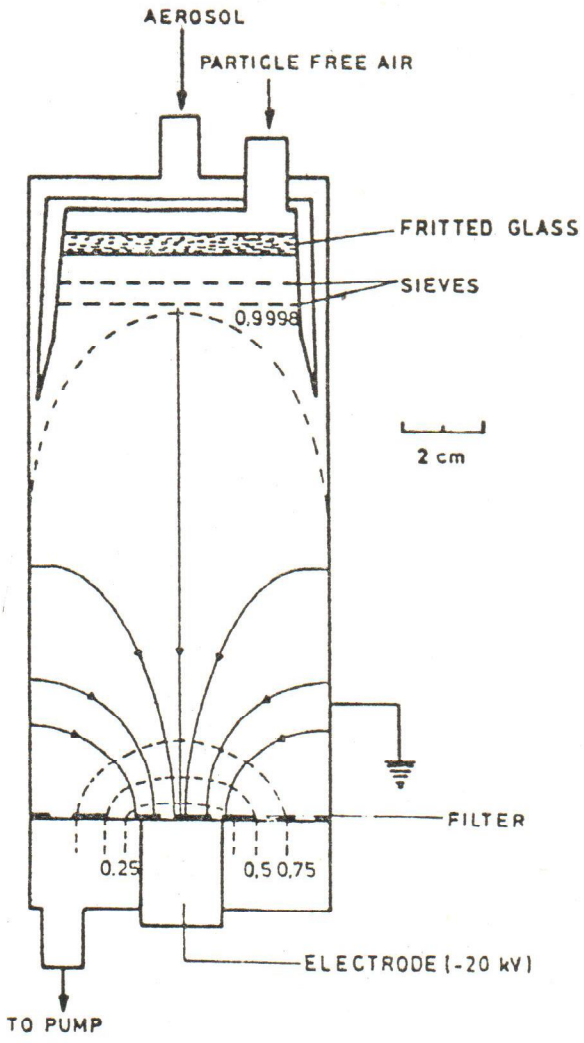

Fig. 3. *System of Zebel and Hochrainer (63) for separating fibres and isometric particles. The electric field (—20 kV) lines of force are marked with arrows. They cross the air flow lines which are vertically downwards and draw the charged particles from the periphery to the centre of the filter. Fibres carry greater charges than compact particles and are found closer to the centre.*

particles deposited on the negative plate. Long fibres collect more ions than short ones while passing the corona discharge, hence they deposit first in the spectrometer with shorter fibres further downstream, followed by particles of compact shape carrying minimal charges.

An improved version of the apparatus was later described by *Zebel and Hochrainer* (63). It was now built on a cylindrical plan with improved charging; the charged aerosol particles were led, in a thin layer, down the inside surface of the cylinder by using a flow of clean air, very carefully introduced to avoid mixing. The cylinder was earthed and a grid, covered with filter paper formed the base of the cylinder which was charged to —20 kV. The inhomogenous field drew all particles towards the centre of the filter where they were spread out radially, the fibres with the highest mobility being nearest to the centre. This apparatus works well but it is doubtful how it would grade soft chrysotile (Fig. 3).

Work on the automation of fibre counting by image analysis has been in progress for several years. The method which has received most development is »Magiscan« in which a television camera records optical microscope fields of a membrane filter sample. The picture is digitised in levels of gray and fed to a microprocessor system programmed to read out fibre length selectively. The current stage of development is described by *Kenny* (64). Use with an electron microscope is projected.

10. AEROSOL GENERATORS FOR FIBROUS PARTICLES

Timbrell, Hyett and Skidmore (65) describe a piston fed disperser for putting up aerosols of the UICC reference samples of asbestos. It will cope with all varieties, subject to small adjustments. Concentrations of about 12 mg/m³ in 12 l/min of air obtained of particles below 5 μm aerodynamic diameter which represents about 80% of the total airborne mass. Fibres up to 50 μm long are emitted, when the charge of asbestos contains lengths up to 200 μm as well as large flocs with which the disperser deals without difficulty; even the entwined, curved fibres of chrysotile are separated. The output of dust is well suited to inhalation experiments.

Hounam (66) describes a small air jet blowing over the top of a tube up which a small plug of asbestos is driven by a screw. It will deal with only a few mg of sample and disperse it over an hour or two. It is a useful means of dispersing neutron activated asbestos.

Although they do not discuss asbestos, a good account of the behaviour of fluidised bed dust generators is given by *Willeke and co-workers* (67). *Guichard* (68) gives detailed designs; also *Carpenter and Yerkes* (69)whose apparatus delivers steadily 125 mg/m³ in 0.28 m³/min of air. *Boucher and Lua* (70) have a system yielding very high concentrations (4 g/m³) which breaks up aggregates. An alternative arrangement to a

fluidised bed, which also breaks up aggregates, has been described by *Seehars and Hochrainer* (71). The behaviour of fluidised beds as a source of glass fibre aerosols has been described by *Carpenter and co-workers* (72).

Spurny, Gentry and Stöber (73) point out that the flow velocities needed to generate an aerosol of fine fibres are too low for a bed to fluidise; hence no aerosol can be generated. In order to overcome this problem they arranged to agitate the vessel containing the bed at frequencies from 15 to 120 Hz with amplitudes of 50 to 1000 μm. This system worked well and samples on nuclepore filters of amosite and chrysotile obtained in this way are illustrated.

Spurny (74) discusses fibre generation and length classification in great detail with 82 references and some fine photographs of fibres.

Pickrell and co-workers (75) constructed a generator for glass fibres. Such fibres which are longer than 20 μm are retained more tenaciously in the lungs than fibres below 5 μm in length, presumably due to »frustrated phagocytosis«. Commercial glass fibre mat was minced, suspended in polyethylene glycol, formed into blocks and frozen. The blocks were then sectioned with a microtome, suspended in water, 100 μm steel pellets added, dried and finally dispersed in fluidised bed generators. Concentrations of aerosol of 40 mg/m^3 resulted of which about 2/3 was in fibres below 5 μm aerodynamic diameter. The diameter of the fibres was about 2 μm and there were some 2000/cm^3 longer than 20 μm.

Friedrichs (1) discusses problems which arise in administering to animals fibres of asbestos, glass and other substances. He gives a nomogram relating diameter, length and density of fibres to the number per mg. Citing Pott's experiments, he relates the probability of tumour formation following intratracheal injection of rats, to the dose; thresholds and maxima are best shown by plotting against the number of fibres administered.

References

1. *Friedrichs, K. H.:* Staub. Reinh. Luft, *38* (1978) 490—493.
2. *Walton, W. H.:* Ann. Occup. Hyg., *25* (1982) 117—247.
3. *Spurny, K., Stöber, W. et al.:* Sci. Total Environ., *11* (1979) 1—40.
4. *Weller, W.:* Staub. Reinh. Luft, *38* (1978) 481—485.
5. *Doll, R.:* Br. J. Ind. Med., *12* (1955) 81—86.
6. *Davies, C. N.:* J. Aerosol Sci., *10* (1978) 477—513. (Ann. Occup. Hyg., *13* (1970) 241—245).
7. *Gilson, J. C.:* Proc. Roy. Soc. Med., *66* (1973) 395—403.
8. *British Occupational Hygiene Society:* Ann. Occup. Hyg., *11* (1968) 47—69.
9. *Luxon, S.:* Ann. Occup. Hyg., *13* (1970) 23—24.
10. *Health and Safety Commission:* Final Report of the Advisory Committee on Asbestos, HMSO, London, 1979.

11. *British Occupational Hygiene Society:* Ann. Occup. Hyg., *16* (1973) 1—5.
12. *British Occupational Hygiene Society:* Ann. Occup. Hyg., *27* (1983) 1—55.
13. *Stellingwerf, J.:* Ann. Occup. Hyg., *28* (1984) 354—356.
14. *Vorwald, A. J., Durkan, T. M., Pratt, P. C.:* Arch. Ind. Hyg. Occup. Med., *3* (1951) 1—43.
15. *Beattie, J., Knox, J. F.:* In »Inhaled Particles and Vapours«, Ed. C. N. Davies, Pergamon Press, Oxford, 1961, pp. 419—433.
16. *Beattie, J.:* In »Inhaled Particles and Vapours«, Ed. C. N. Davies, Pergamon Press, Oxford 1961, p. 434, p. 442.
17. *Zielhuis, R. L.:* Public Health Risks of Exposure to Asbestos, Pergamon Press, Oxford 1973.
18. *Wagner, J. C.:* The Practitioner, *223* (1979) 29.
19. *Wagner, J. C., Berry, G., Skidmore, J. W., Timbrell, V.:* Br. J. Cancer, *29* (1974) 252—269.
20. *Davies, C. N.:* In »Annual Review of Medicine«, Ed. D. A. Rytand and W. Crager, 1957, pp. 323—348.
21. *Beck, E. G., Bruch, J., Friedrichs, K. H., Hilscher, W., Pott, F.:* In »Inhaled Particles III«, Ed. W. H. Walton, Unwins, Old Woking, 1971, pp. 477—487.
22. *Conning, D. M., Hayes, M. J., Styles, J. A., Nicholas, J. A.:* In »Inhaled Particles III«, Ed. W. H. Walton, Unwins, Old Woking, 1971, pp. 499—506.
23. *Morgan, A., Evans, J. C., Holmes, A.:* In »Inhaled Particles IV«, Ed. W. H. Walton, Pergamon Press, Oxford 1977, pp. 259—274.
24. *Monchaux, G., Bignon, J. et al.:* In »Inhaled Particles V«, Ed. W. H. Walton, Pergamon Press, Oxford 1982, pp. 309—318. (Ann. Occup. Hyg., *26*)
25. *Wagner, J. C., Pooley, F. D. et al.:* In »Inhaled Particles V«, Ed. W. H. Walton, Pergamon, Oxford 1982, pp. 423—431. (Ann. Occup. Hyg., *26*)
26. *Timbrell, V.:* In »Inhaled Particles V«, Ed. W. H. Walton, Pergamon, Oxford 1982, pp. 347—369. (Ann. Occup. Hyg., *26*)
27. *Balser, J. L.:* In »Inhaled Particles III«, Ed. W. H. Walton, Unwins, Old Woking, 1971, p. 57.
28. *Pickrell, J. A., Hill, J. O. et al.:* In »Inhalation Toxicity Research Institute, Annual Report, 1978—1979«. National Technical Information Service, US Department of Commerce, Springfield, 1980, pp. 509—513.
29. *Timbrell, V., Hyett, A. W., Skidmore, J. A.:* Ann. Occup. Hyg., *11* (1968) 273—281.
30. *Harris, R. L., Timbrell, V.:* In »Inhaled Particles IV«, Ed. W. H. Walton, Pergamon Press, Oxford 1977, pp. 75—89.
31. *Wagner, J. C., Berry, G. et al.:* In »Inhaled Particles IV«, Ed. W. H. Walton, Pergamon Press, Oxford 1977, pp. 647—654.
32. *Bolton, R. F., Davis, J. M. G., Donaldson, K., Wright, A.:* In »Inhaled Particles V«, Ed. W. H. Walton, Pergamon, Oxford 1982, pp. 569—582. (Ann. Occup. Hyg., *26*)
33. *Stanton, M. F., Wrench, C.:* J. Natl. Cancer Inst., *48* (1972) 797—821.
34. *Doll, R.:* J. Roy. Statistical Soc. A., *134* (1971) 133—166.
35. *Stumphius, J., Meyer, P. B.:* Ann. Occup. Hyg., *11* (1968) 283—293.
36. *Rowlands, N., Gibbs, G. W., McDonald, A. D.:* In »Inhaled Particles V«, Ed. W. H. Walton, Pergamon, Oxford 1982, pp. 411—415. (Ann. Occup. Hyg., *26*)
37. *McDonald, A. D., McDonald, J. C., Pooley, F. D.:* In »Inhaled Particles V«, Ed. W. H. Walton, Pergamon, Oxford 1982, pp. 417—422. (Ann. Occup. Hyg., *26*)
38. *Kannerstein, M., Churg, J.:* Environ. Health Perspect., *34* (1980) 31—36.

39. *Rood, A. P., Streeter, R. R:* Ann. Occup. Hyg., *28* (1984) 333—339.
40. *Ayer, H., Zumwalde, R. D.:* In »Generation of Aerosols«, Ed. K. Willeke, Ann. Arbor, Michigan, 1980, pp. 235—256.
41. *Schneider, T., Holst, E.:* J. Aerosol. Sci., *14* (1983) 1.
42. *Yu, P. Y., Gentry, J. W.:* J. Aerosol. Sci., *13* (1982) 200—205.
43. *Robock, K., Klosterkotter, W.:* In »Inhaled Particles III«, Ed. W. H. Walton, Unwin, Old Woking 1971, pp. 465—475.
44. *Pott, F.:* Staub-Reinh. Luft, *38* (1978) 486—490.
45. *Pott, F.:* In »Umwelthygiene«, Girardet, Essen, *13* (1980) 31—66.
46. *Badollet, M. S.:* Canadian Mining and Mettalurgical Bull, Transactions, *54* (1951) 151—160.
47. *Hodgson, A. A.:* Phil. Trans. Roy. Soc. London, *286* (1977) 611—624.
48. *Timbrell, V.:* In »Assessment of Airborne Particles« Ed. T. Mercer, P. Morrow, W. Stober, Thomas, Springfield, Illinois 1972, pp. 429—445.
49. *Stöber, W., Flaschbart, H., Hochrainer, D.:* Staub. Reinh. Luft, *30* (1970) 277—285.
50. *Masuda, H., Hochrainer, D., Stöber, W.:* J. Aerosol Sci., 10 (1979) 275—287.
51. *Davies, C. N.:* J. Aerosol Sci., *10* (1978) 477—513.
52. *Prodi, V., de Zaiacomo, T., Hochrainer, D., Spurny, K.:* J. Aerosol Sci., *13* (1982) 49—58.
53. *Mazumder, M. K., Ware, R. E., Wilson, J. D., Renninger, R. G., Hiller, R. C., McLeod, P. C., Raible, R. W., Testerman, M. K.:* J. Aerosol Sci., *10* (1979) 561—569.
54. *Wilson, J. C., Liu, B. Y. H.:* J. Aerosol Sci., *11* (1980) 139—150.
55. *Hiller, F. C., Mazumder, M. K., Wilson, J. D., McLeod, P. C., Bone, R. C.:* J. Aerosol Sci., *13* (1982) 337—343.
56. *Ogden, T. L., Walton, W. H.:* Ann. Occup. Hyg., *18* (1975) 157—160.
57. *Seger, G.:* J. Aerosol Sci., *12* (1981) 219—220.
58. *Timbrell, V.:* Microscope, *20* (1972) 365—368.
59. *Timbrell, V.:* Ann. Occup. Hyg., *18* (1975) 299—311.
60. *Timbrell, V.:* Filtration and Separation, *14* (1977) 242.
61. *Verill, K. J.:* Ann. Occup. Hyg., *27* (1983) 111.
62. *Zebel, G., Hochrainer, D., Boose, C.:* J. Aerosol Sci., *8* (1977) 205—213.
63. *Zebel, G., Hochrainer, D.:* J. Aerosol Sci., *10* (1979) 245.
64. *Kenny, L. C.:* Ann. Occup. Hyg., *28* (1984) 401—415.
65. *Timbrell, V., Hyett, A. W., Skidmore, J. A.:* Ann. Occup. Hyg., *11* (1968) 273—281.
66. *Hounam, R. F.:* Ann. Occup. Hyg., *14* (1971) 329—335.
67. *Willeke, K., Lo, C. S. K., Whitby, K. T.:* J. Aerosol. Sci., *5* (1974) 449—555.
68. *Guichard, J. C.:* In »Fine Particles«, Ed. B. Y. H. Liu, Academic Press, New York, 1976, pp. 174—193.
69. *Carpenter, R. L., Pickrell, J. A. et al.:* »Inhalation Toxicity Research Inst. Annual Report 1979—1980.« National Technical Information Service, US Department of Commerce, Springfield, USA, 1980, pp. 501—505.
70. *Boucher, R. F., Lua, A. C.:* J. Aerosol Sci., *13* (1982) 499—511.
71. *Seehars, H. D., Hochrainer, D.:* Staub. Reinh. Luft, *41* (1981) 466—472.
72. *Carpenter, R. L., Yerkes, K. L.:* Inhalation Toxicity Research Inst. Annual Report 1978—1979«. National Technical Information Service, US Department of Commerce, Springfield, USA, 1979, pp. 327—330.
73. *Spurny, K. R., Gentry, J. W., Stöber, W.:* In »Fundamentals of Aerosol Science«. Ed. D. T. Shaw, Wiley, New York, 1978, Chapter 5, pp. 257—324.

74. *Spurny, K. R.:* In »Generation of Aerosols«. Ed. K. Willeke, Ann Arbor, 1980 USA, Chapter 13, pp. 259—298.

75. *Pickrell, J. A., Carpenter, R. L. et al.:* In »Inhalation Toxicity Research Institute, Annual Report, 1978—1979«. National Technical Information Service, US Department of Commerce, Springfield, USA, 1979, pp. 550—554.

Sažetak

BOLESTI POVEZANE S UDISANJEM AZBESTNE PRAŠINE

Proučavanje literature o azbestozi upućuje na to da bolest izaziva udisanje azbestnih vlakana dužih od 10—15 μm. Kraća vlakna uklanjaju se preko fagocita iz pluća. Meki krizotil mnogo je aktivniji u plućima od bilo koje druge vrste azbesta u izazivanju azbestoze. Važnost topljivosti krizotila, navodno veće nego u drugih vrsta azbesta, nije sigurna jer krizotil nije toksičan za fagocite a vlakna mogu biti izuzetno fina i teško ih je otkriti čak i uz pomoć dobrog transmisijskog elektronskog mikroskopa. Ishod može rezultirati usitnjavanjem vlakana po dužini prije negoli smanjenjem njihova promjera.

Rak pluća također izazivaju duga fina vlakna krizotila. Započinje peribronhijalno jer se izvijena, kosa vlakna ne probijaju kroz pluća uz pomoć ritmičkih respiratornih pokreta. Tvrda, igličasta, glatka amfibolna vlakna prolaze kroz plućno tkivo i, ako su dovoljno duga da izbjegnu fagocitozu, uzrokuju karcinom.

Mezoteliom pleure izaziva udisanje dugih amfibolnih vlakana koja su iz dišnih puteva došla u pleuru. Može biti izazvan direktnom injekcijom ili aplikacijom azbestnih krizotilnih vlakana bilo koje dužine u pleuru životinja, ali ne nastaje udisanjem jer se izvijena, kosa vlakna mekog krizotila ukopaju u plućno tkivo i ne putuju do pleure.

Selektivno sakupljanje uzoraka azbestne prašine iz zraka nephodno je da bi se iz znatne količine kratkih vlakana prašine koju uklanjaju fagociti prije nego izazove oštećenja, izdvojio mali dio vlakana odgovornih za izazivanje bolesti.

Selektivni sakupljač uzoraka mora sakupljati i zadržati vlakna duža od 10—15 μm, promjera u rasponu 0,1—0,5 μm. Nema svrhe selekcionirati vlakna na osnovi njihovog aerodinamičkog promjera jer je taj kriterij neosjetljiv na dužinu vlakana. Opisani su neki noviji sistemi sakupljanja i selekcioniranja uzoraka.

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