5. Summary of Data Reported and Evaluation

5.1 Exposure data

Significant commercial production of man-made vitreous fibres began in the early twentieth century. In 2001, it was estimated that over 9 million tonnes of man-made vitreous fibres (MMVFs) were produced annually in over 100 factories around the world. Most of the man-made vitreous fibre produced is used as thermal or acoustical insulation. Usage for this purpose is divided about equally between glass wool (~ 3 million tonnes, used predominantly in North America) and rock (stone) and slag wool (~ 3 million tonnes, used predominantly in Europe and the rest of the world). In recent years, high-alumina, low-silica wools (~ 1 million tonnes) have been increasingly replacing rock (stone) and slag wools in this application. Special-purpose glass fibres are limited-production, small-diameter fibre products that are typically used for purposes other than insulation as in filtration media and batteries. Continuous glass filaments (~ 2 million tonnes) are generally used in the reinforcement of plastics and in textiles. Refractory ceramic fibres, first produced commercially in the 1950s, are widely used (~ 150 thousand tonnes) in high-temperature applications such as furnace insulation. The more recently developed alkaline earth silicate wools (~ 10 thousand tonnes) are replacing refractory ceramic fibres in some applications.

Man-made vitreous fibre products can release airborne respirable fibres during their production, use and removal. In general, as the nominal diameter of man-made vitreous fibre products decreases, both the concentration of respirable fibres and the ratio of respirable to total fibres increase. Although exposure to man-made vitreous fibres during their production, processing and use is thought to have been higher in the past, current average exposure levels are generally less than 0.5 respirable fibre/cm^3 (500 000 respirable fibres/m^3) as an 8-h time-weighted average. Higher levels have been measured in production of special-purpose glass fibres and refractory ceramic fibres, installation of loose-fill insulation without binder, and removal of insulation products.

The concentrations of man-made vitreous fibres measured in outdoor and indoor air in non-occupational settings have been found to be much lower than in occupational settings related to their production, use or removal.

5.2 Human carcinogenicity data

Two large cohort studies and case–control studies nested within these cohorts from the USA and Europe provide most of the epidemiological evidence concerning potential risk for respiratory and other cancers associated with occupational exposure to glass wool, continuous glass filament and rock (stone)/slag wool during manufacture. The United States cohort study included 16 plants, extended the follow-up to 1992 and expanded a previous cohort to include women and non-white workers. This study included information on smoking habits and a new assessment of historical workplace exposure to respirable fibres and several sources of co-exposure including asbestos, formaldehyde and silica. The European cohort extended the follow-up to 1990 in 13 plants.

Glass wool

The findings of the United States cohort study provided no evidence of excess mortality from all causes combined or from all cancers combined, using local rates. A statistically significant 6% excess in respiratory cancer (primarily trachea, bronchus and lung) mortality was observed. When analysis was restricted to long-term workers, the excess was reduced and was no longer statistically significant. Adjustment for smoking based on a random sample of workers suggests that smoking may account for the excesses in respiratory cancer observed in the male glass fibre cohort (glass wool and continuous glass filament combined). The standardized mortality ratios for respiratory cancer were related neither to duration of employment among the total cohort or among long-term workers nor to duration of exposure, cumulative exposure or average intensity of exposure to respirable glass fibre (glass wool and continuous glass filament combined). Analysis by product group showed a statistically significant excess of respiratory cancer for all workers from plants grouped as 'mostly glass wool', but this excess risk for the 'mostly-glass-wool' product group was reduced and no longer statistically significant when the cohort was limited to long-term workers (≥ 5 years of employment). There was no evidence of an excess of mesothelioma or non-respiratory cancers.

The case–control study of respiratory cancer nested within the United States cohort enabled control of plant co-exposure and a more detailed control for confounding by smoking. Duration of exposure, cumulative exposure, average intensity of exposure and the time since first exposure to respirable glass fibre were not associated with an increased risk for respiratory cancer. These results were not altered by using different characterizations of categorized respirable fibre exposure or by alternative models for continuous exposure data.

The European cohort study of glass wool workers demonstrated an increased mortality from lung cancer (trachea, bronchus and lung) but no trend with time since first hire or duration of employment. One death from mesothelioma was observed in this cohort. This study did not estimate fibre exposure, but used surrogate measures such as 'techno-

logical phase at first employment'. No information was available either on co-exposure or on smoking habits.

Continuous glass filament

Two of the plants of the United States cohort study manufactured only continuous glass filament. For all workers and for long-term workers from these two plants, no evidence of excess mortality from respiratory cancer was found when compared with local rates. Adjustment for smoking had little effect on the standardized mortality ratio for respiratory cancer. A nested case–control study that included adjustments for smoking and co-exposure also provided no consistent evidence of excess mortality from respiratory cancer. The exposure–response analyses that combined exposure to continuous glass filament and to glass wool are reported in the section on glass wool.

The European cohort study reported few data to evaluate cancer risks among workers exposed to continuous glass filament. This study provided no convincing evidence of an elevated risk for lung cancer.

Results were also available from two smaller cohort studies in the USA and Canada. The United States cohort study on one continuous glass filament plant, which included a nested case–control study, with information on smoking and co-exposure, provided no consistent evidence of an excess risk for lung cancer. The Canadian cohort study of one continuous glass filament plant did not include an assessment of smoking or co-exposure. This study also provided no consistent evidence of an excess risk for lung cancer.

Rock (stone) and slag wool

The present evaluation relies mainly on cohort and nested case–control studies, in which exposure to rock (stone) wool and exposure to slag wool were not considered separately.

The extended follow-up of the rock (stone)/slag wool cohort from the USA indicated an overall elevated risk of respiratory cancer when either national or local comparison rates were used. However, no association was found with duration of exposure or with time since first exposure. Standardized mortality ratios were no longer elevated when indirect adjustment for smoking was made. The nested case–control study showed no association between respiratory cancer and estimated cumulative exposure to respirable fibres, with or without adjustment for possible confounding by smoking and other sources of occupational exposure. Another nested case–control study partially overlapping with the study in the USA showed no increased risk for respiratory cancer in association with exposure to slag wool.

The extended follow-up of the European cohort study indicated an overall elevated risk for lung cancer when national comparison rates were used. This study showed an increasing risk with years since first exposure. The highest standardized mortality ratio was found among workers with the longest time since first employment

and among those first employed in the 'early technological phase', i.e. before the introduction of oil and binders and use of the batch-processing method. However, in a case–control study that included detailed information on exposure to fibres, individual smoking habits and potential occupational confounders, no increased risk of lung cancer with increasing fibre exposure was reported.

The results from these studies provide no evidence of an increased risk for pleural mesotheliomas or any other tumours.

Refractory ceramic fibres

Preliminary results from a United States epidemiological mortality study of refractory ceramic fibre workers were available. However, the limited epidemiological data do not permit an adequate evaluation of the cancer risk associated with exposure to refractory ceramic fibres.

Man-made vitreous fibres (not otherwise specified)

A number of studies did not separate exposure to glass wool from exposure to rock (stone) and slag wool or other fibre types, or had limited ability to distinguish between these different fibre types. Since much more information was available from epidemiological studies in the fibre production industries, no separate evaluation is made for the studies of mixed exposure. The results of these studies were, however, taken into consideration for the evaluation of the distinct fibre types.

A cohort study of Swedish wooden house industry workers exposed to man-made vitreous fibres demonstrated a decreased risk for lung cancer and no positive trend in standardized mortality ratios for lung cancer with duration of employment. An increased risk for stomach cancer was found, but the risk did not increase with duration of employment.

Two population-based case–control studies in Germany were combined in a pooled analysis that suggested an association between lung cancer and occupational exposure to man-made vitreous fibres. Odds ratios were adjusted for smoking and exposure to asbestos, but exposure to man-made vitreous fibres and asbestos may not have been separated well enough to rule out residual confounding as an explanation of the results. A low response rate in one of the reference groups adds to the uncertainty of the validity of this study.

A population-based case–control study from Canada found no association between lung cancer and occupational exposure to glass wool or rock (stone) and slag wool.

A German case–control study suggested an association between mesothelioma and exposure to man-made vitreous fibres adjusted for asbestos exposure. However, several limitations constrain the interpretation of the reported results, particularly the potential for misclassification of exposure to asbestos and man-made vitreous fibres

and the small number of cases and controls classified as ever having been exposed to man-made vitreous fibres without exposure to asbestos.

An increased risk for laryngeal and hypopharyngeal cancer in association with exposure to man-made vitreous fibres was reported in a case–control study from France, but this was an isolated finding not observed in other studies.

Conclusion

Results from the most recent cohort and nested case–control studies of United States workers exposed to glass wool and continuous glass filament and of European workers exposed to rock (stone) and slag wool have not provided consistent evidence of an association between exposure to fibres and risk for lung cancer or mesothelioma.

These studies, like all epidemiological investigations, have limitations that must be borne in mind when interpreting their results. Although the exposure assessment methods used in these studies are far better than in most epidemiological studies, there is still the potential for exposure misclassification. Notably these studies were not able to examine fully the risks to workers exposed to more durable fibres. Information on smoking and on the other potential confounders that were adjusted for in these studies are also subject to measurement error, which may have influenced the validity of the adjustments made for these factors. Underascertainment and misclassification of mesothelioma may also be a concern in these studies, which primarily relied upon death certificate information. Finally, although these studies are very large by epidemiological standards, their sensitivity may be limited by the fact that fibre exposure levels were low for a large proportion of the study population.

Of some concern are risks for workers in industries that use or remove these products (e.g. construction), who may have experienced higher, but perhaps more intermittent, exposure to man-made vitreous fibres. The data available to evaluate cancer risks from exposure to man-made vitreous fibres in these populations are very limited.

Results on mortality among refractory ceramic fibre workers have also been published since the previous *IARC Monographs* evaluation (1988). However, the epidemiological evidence for refractory ceramic fibres is still extremely limited. Radiographic evidence indicating pleural plaques has been reported for refractory ceramic fibre workers. Although the prognostic significance of pleural plaques is unclear, such plaques are also a common finding among asbestos-exposed workers.

5.3 Animal carcinogenicity data

Continuous glass filament

In experiments in which three types of continuous glass filament of relatively large diameter (> 3 μm) were administered intraperitoneally to rats, no significant increase in tumour response was observed.

Insulation glass wool

Insulation glass wools were tested in well-designed, long-term inhalation studies in rats and hamsters. No significant increase in lung tumours and no mesotheliomas were observed in rats and no lung tumours or mesotheliomas were observed in hamsters exposed to insulation glass wool. Two different asbestos types used as positive controls produced increases in lung tumours and mesotheliomas.

Two insulation glass wools that produced no increase in tumours when administered by inhalation did induce mesotheliomas when injected at high doses (approximately 10^9 fibres) into the peritoneal cavity of rats.

Special-purpose glass fibres

A number of chronic inhalation studies of special-purpose glass fibres have been conducted in rats, hamsters and guinea-pigs. Early inhalation studies demonstrated no significant increases in lung tumours or mesotheliomas. In some of these studies, asbestos did not induce tumours in the controls, which was probably related to use of short fibres in the aerosols. More recent studies of special-purpose glass fibres, using improved methods of fibre preparation and delivery, resulted in significant increases in lung tumours and mesotheliomas in rats (E-glass fibre) and in a single mesothelioma in hamsters (‘475’ fibre).

Many intraperitoneal studies of special-purpose glass fibres have been conducted, most of which have examined the tumorigenic potential of two compositions of special-purpose glass fibres (‘475’ and E-glass fibres) after injection or surgical implantation of fibres at high doses (approximately 10^9 fibres) into the peritoneal cavity of rats. All of these studies reported an increase in peritoneal tumours.

Special-purpose glass fibres were tested by intratracheal instillation in two experiments in rats and two in hamsters. A significant increase in lung tumours was observed in one of the rat studies and increases in lung tumours and mesotheliomas were observed in one of the hamster studies. The other two studies showed no increase in either tumour type.

Rock (stone) wool

In a well-designed, long-term inhalation study in which rats were exposed to rock (stone) wool, no significant increase in lung tumour incidence and no mesotheliomas were observed. Crocidolite asbestos was used as the positive control and led to high lung tumour incidence and one mesothelioma.

After intratracheal instillation of rock (stone) wool in two studies, no significant increase in the incidence of lung tumours or mesotheliomas was found. Tremolite asbestos was used as a positive control and induced lung tumours.

In several studies of intraperitoneal injection of high doses (approximately 10^9 fibres), rock (stone) wool induced a significant increase in mesothelioma incidence.

The more biopersistent rock (stone) wool fibres produced a higher incidence of tumours than fibres with lower biopersistence.

Slag wool

In a well-designed, long-term inhalation study of slag wool in rats, no statistically significant increase in the incidence of lung tumours and no mesotheliomas were observed. Crocidolite asbestos was used as a positive control and led to high lung tumour incidence. In two intraperitoneal studies, a high dose (approximately 10^9 fibres) of slag wool induced a statistically significant increase in the incidence of mesotheliomas.

Refractory ceramic fibres

In a well-designed, long-term inhalation study with refractory ceramic fibres in rats, a statistically significant increase in the incidence of lung tumours and a few mesotheliomas were observed. In a well-designed, long-term inhalation study of refractory ceramic fibres in hamsters, a significant increase in the incidence of mesotheliomas was observed.

After intratracheal instillation, two studies reported no excess in tumour incidence in rats. In three intrapleural studies in rats, no significant increase in tumour incidence was observed. In intraperitoneal studies in rats and hamsters, tumour incidence was related to fibre length and dose.

Newly developed wools

Two newly developed, less biopersistent fibres (an alkaline earth silicate (X-607) wool and a high-alumina, low-silica (HT) wool) have been tested in well-designed, long-term inhalation studies in rats and produced no significant increase in the incidence of lung tumours and no mesotheliomas.

In a study in rats of less biopersistent high-alumina, low-silica (HT) wool administered by intraperitoneal injection at a high dose (approximately 10^9 fibres), no abdominal tumours were observed. Four other less biopersistent fibres (A, C, F and G) have been tested by intraperitoneal injection at a high dose (approximately 10^9 fibres) in rats and produced no significant increase in the incidence of abdominal tumours.

One more biopersistent fibre type (H) was tested by intraperitoneal injection at a high dose (approximately 10^9 fibres) in rats and produced abdominal tumours.

5.4 Other relevant data

Deposition and retention

The deposition of inhaled fibres in the respiratory tract is mainly governed by their aerodynamic behaviour, including deposition by impaction, sedimentation and interception. In addition, deposition by diffusional displacement is induced by Brownian motion. Model calculations show that the respirability of fibres, i.e. their penetration to the alveolar region, differs between rodents and humans. A larger fraction of inhaled long fibres is deposited in the alveolar region of humans than in that of rats.

Chemical composition, fibre size and the deposited dose of fibres in the lung are determinants of their retention kinetics. The main mechanisms of mechanical fibre clearance include mucociliary movement in the nasopharyngeal and tracheobronchial regions and alveolar macrophage phagocytosis in the alveolar region with subsequent removal towards the mucociliary escalator. Macrophage-mediated clearance becomes negligible for long fibres, i.e. fibres with lengths approaching 20 μm and longer, which cannot be completely phagocytosed by alveolar macrophages. Alveolar macrophage-mediated clearance is significantly slower in humans than in rats, with retention half-times of several hundred days in humans and about 70 days in rats. In addition to these mechanisms, chemical dissolution and leaching, as well as breakage, can occur. These processes are important and lead to more rapid elimination of fibres deposited in the respiratory tract, thereby lowering the potential for inducing long-term adverse effects. Because the retention half-time due to mechanical clearance is much longer in humans than in rats, higher fibre solubility reduces persistence more in the human lung than in the rat lung.

Few data are available on retention of man-made vitreous fibres in human lungs. In the one available study, the lung burden of man-made vitreous fibres did not differ between workers in glass, rock (stone) and slag wool production compared between themselves or with controls. The interpretation of this difference is limited by the long delay between the end of exposure and sampling. In other studies, refractory ceramic fibres, some with morphological or chemical alterations, have been recovered from the lungs of both production workers and end-users.

Fibre biopersistence

The biopersistence of fibres deposited in the respiratory tract results from a combination of physiological clearance processes (mechanical translocation/removal) and physico-chemical processes (chemical dissolution and leaching, mechanical breaking). Long and short fibres differ in the way in which their elimination from the respiratory tract is affected by each of these mechanisms. Short fibres are taken up by macrophages and subjected to chemical dissolution/leaching within an acidic milieu while at the same time they are actively removed by these phagocytic cells. In contrast, long fibres which can be incompletely phagocytosed by several macrophages are not efficiently

removed by physical translocation but may be subjected to chemical dissolution/leaching at variable pH. Since long fibres are most potent with respect to carcinogenicity, the focus of an animal biopersistence assay is on long-fibre retention kinetics in the lung. A number of studies in rats have suggested a correlation between the biopersistence of long fibres (> 20 μm) and their pathogenicity with respect to lung fibrosis and thoracic tumours.

In-vitro dissolution

The physico-chemical mechanisms whereby fibres may degrade in the lung have been examined in a variety of cell-free systems. The basic process by which dissolution of man-made vitreous fibres occurs is via attack of water molecules on the surface of fibres leading to dissolution and subsequent disruption of the fibre structure. The dissolution rate of any fibre is determined primarily by its composition. The most informative studies employ flow-through systems using balanced salt solutions at physiological pHs likely to be encountered in the intrapulmonary environment. The results from such studies have shown correlations with rates of removal of long fibres from the lung in short-term biopersistence assays. While considerable variation occurs between laboratories, the rank order of the durability of tested fibres is generally consistent.

The experimental dissolution rates of tested fibres have been reported to span over five orders of magnitude. Such a range may predict that fibres could persist in lung tissue from a few days to several years.

In-vitro studies of man-made vitreous fibres using cell culture techniques allow estimation of dissolution of fibres in the presence of lung cells. These studies provide information on the joint effects of cells and fluid on different types of man-made vitreous fibre that is helpful in comparing the dissolution rates of a given fibre and then assessing the rank order of the relative dissolution of different man-made vitreous fibres. The results of these studies are consistent with those of studies on the solubility of man-made vitreous fibres in cell-free systems.

Toxic effects in humans

With the exception of a single rock (stone)/slag wool plant in the United States cohort study which had a documented history of asbestos use, none of the mortality studies demonstrated a significant risk for non-malignant respiratory diseases. No mortality data were available on workers exposed to refractory ceramic fibres.

No convincing data for an excess of small parenchymal opacities in chest radiographs compatible with pneumoconiosis have been published. No pleural changes related to any glass fibre type or to rock (stone)/slag wool have been observed. However, an excess of pleural changes, particularly pleural plaques, has consistently been demonstrated in the cohorts of workers in the USA and Europe involved in the production of refractory ceramic fibres.

No indications of a significant excess of respiratory symptoms or of a significant decrease in lung function have been reported for glass fibre workers. The results for rock (stone) wool workers are more conflicting due to a possible interaction between fibre exposure and smoking. In contrast, a small exposure-related effect has been observed in the cohorts of workers involved in the production of refractory ceramic fibres in both the USA and Europe.

A few well-designed studies have supported previous findings of mechanical irritative effects on the skin, eyes and upper respiratory tract associated with coarse fibres.

Low levels of exposure were estimated in most production worker cohorts. With similar low cumulative exposure to asbestos, lung fibrosis would not have been detected in epidemiological studies using standard chest radiography. Limited interpretable data are available from end-users (particularly workers involved in the removal or modification of materials containing man-made vitreous fibres).

Toxic effects in experimental systems

The most important end-points that have been associated with exposure to man-made vitreous fibres include chronic persistent inflammation, fibrosis and cell proliferation in the lungs and mesothelial lining. In general, for a range of man-made vitreous fibres, the data support the contention that long, biopersistent fibres cause prolonged inflammation and fibrosis. Although mechanistically they are not conclusively linked, pulmonary and occasionally pleural fibrosis is found with conditions of exposure to man-made vitreous fibres that are carcinogenic in laboratory animals.

Because biopersistence is believed to be an important factor in the toxicity of man-made vitreous fibres, there are limitations inherent in short-term in-vitro assays of fibre toxicity.

Effect on gene expression

Mutation and/or activation of proto-oncogenes, inhibition of tumour suppressor genes and activation of transcription factors controlling the production of inflammatory cytokines and growth factors have been proposed to play a role in asbestos-induced carcinogenesis. The evidence indicates that glass fibres enter cells and cause genetic modification by physically interfering with chromosomal segregation during mitosis. Glass fibres also generate oxidants and/or mobilize intracellular calcium to activate signalling pathways controlling transcription factor activity. This interaction of glass fibres or refractory ceramic fibres with cells has been reported to induce proto-oncogenes, activate transcription factors, increase tumour necrosis factor α production, induce cell transformation and enhance cell growth. The potency of glass fibres is generally lower than that of asbestos on a per unit mass basis.

Several caveats can be raised about these in-vitro studies: (i) these assays are short-term and do not address issues related to fibre dissolution or biopersistence; and (ii) relatively high levels of man-made vitreous fibres on a mass basis have been studied, and the relevance to in-vivo exposure levels is questionable.

Genetic effects

Genotoxic effects of man-made vitreous fibres have been demonstrated in several cultured cell types, including human cells, and in cell-free assays. Many glass wool samples have been found to produce DNA damage, chromosomal aberrations, nuclear abnormalities and cell transformation. The effects were observed to depend on fibre dimensions, with long fibres being more active than shorter fibres. A few rock (stone) wool and slag wool samples have been investigated. Both DNA damage and chromosomal and nuclear aberrations have been observed, as well as mutations in bacterial test systems. The studies on refractory ceramic fibres have so far mostly been limited to the RCF1, 2, 3 and 4 samples and, to a lesser extent, to Japanese standard reference samples. Findings were similar to those with rock (stone) and slag wool.

The occurrence of mutations and some forms of DNA/chromosomal damage may be related to the production of activated oxygen species which have been detected in cell-free systems and in cells exposed to man-made vitreous fibres. Chromosomal and nuclear abnormalities may also be related to the impairment of cell division by the fibres. While reactive oxygen species are produced by either non-fibrous or fibrous particles, cell cycle-associated chromosomal and nuclear abnormalities appear to be a specific response to exposure to fibres. Despite the fact that in-vitro assessment of genetic effects does not address issues related to fibre dissolution or biopersistence, these assays can determine whether a fibre has the potential to be directly genotoxic.

A major gap in the current database is the absence of any studies that correlate genotoxic end-points with the pathogenic effects of man-made vitreous fibres in the same experimental animal system.

Mechanistic considerations

Man-made vitreous fibres deposit in the lungs where they are phagocytosed by macrophages, either completely or incompletely, depending on fibre length. Incomplete phagocytosis is a potent pro-inflammatory stimulus for the release of a cascade of mediators and reactive oxygen and nitrogen species, leading to genotoxicity and proliferation of lung cells. *In vitro*, the direct entry of fibres into cells followed by, or associated with, cell division can produce chromosomal/nuclear abnormalities and genetic changes which may lead to cell transformation and dysregulated proliferation. Animal studies have shown a range of severity of inflammation and fibrosis which has been related to more biopersistent fibres in the lungs. There is a consistent relationship between persistent inflammation, fibrosis and tumour development in animal models.

Refractory ceramic fibres, unlike other man-made vitreous fibres, have the ability to induce pleural plaques in humans. Although pleural plaques in themselves are probably not directly related to cancer development, either in the pleura or the lung, concern over potential carcinogenic effects in the pleura seems valid for refractory ceramic fibres, in view of the ability of asbestos to induce both plaques and pleural cancer.

5.5 Evaluation[1]

There is *inadequate evidence* in humans for the carcinogenicity of glass wool.

There is *inadequate evidence* in humans for the carcinogenicity of continuous glass filament.

There is *inadequate evidence* in humans for the carcinogenicity of rock (stone) wool/slag wool.

There is *inadequate evidence* in humans for the carcinogenicity of refractory ceramic fibres.

There is *sufficient evidence* in experimental animals for the carcinogenicity of special-purpose glass fibres including E-glass and '475' glass fibres.

There is *sufficient evidence* in experimental animals for the carcinogenicity of refractory ceramic fibres.

There is *limited evidence* in experimental animals for the carcinogenicity of insulation glass wool.

There is *limited evidence* in experimental animals for the carcinogenicity of rock (stone) wool.

There is *limited evidence* in experimental animals for the carcinogenicity of slag wool.

There is *limited evidence* in experimental animals for the carcinogenicity of certain newly developed, more biopersistent fibres including fibre H.

There is *inadequate evidence* in experimental animals for the carcinogenicity of continuous glass filament.

There is *inadequate evidence* in experimental animals for the carcinogenicity of certain newly developed, less biopersistent fibres including the alkaline earth silicate (X-607) wool, the high-alumina, low-silica (HT) wool and fibres A, C, F and G.

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[1] Observers/representatives from the industry (B.C. Brown, J.G. Hadley, O. Kamstrup, L.D. Maxim and C.E. Rossiter) were not present during the evaluations.

Overall evaluation

Special-purpose glass fibres such as E-glass and '475' glass fibres are *possibly carcinogenic to humans (Group 2B)*.

Refractory ceramic fibres are *possibly carcinogenic to humans (Group 2B)*.

Insulation glass wool, continuous glass filament, rock (stone) wool and slag wool are *not classifiable as to their carcinogenicity to humans (Group 3)*.

The Working Group elected not to make an overall evaluation of the newly developed fibres designed to be less biopersistent such as the alkaline earth silicate or high-alumina, low-silica wools. This decision was made in part because no human data were available, although such fibres that have been tested appear to have low carcinogenic potential in experimental animals, and because the Working Group had difficulty in categorizing these fibres into meaningful groups based on chemical composition.