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Man-Made Mineral Fibres

1. Chemical and Physical Data

'Man-made mineral fibres' is a generic term that denotes fibrous inorganic substances made primarily from rock, clay, slag or glass. These fibres can be classified into three general groups: glass fibres (comprising glasswool and glass filament), rockwool and slagwool, and ceramic fibres.

'Mineral wool' is a term that has been used to describe rockwool, slagwool and, in some publications, also glasswool. In this monograph, the terms 'rockwool', 'slagwool' and 'glasswool' are used rather than 'mineral wool', whenever possible.

The term 'wool' is used synonymously with fibre when describing vitreous or glassy material that has been attenuated without the use of a nozzle. Fibres that are drawn through nozzles are referred to as filaments or continuous fibres (Loewenstein, 1983; World Health Organization, 1983).

Synonyms and trade names[1]:

Glasswool: JM (Johns Manville) 100; JM 102; JM 104; JM 110

Glass filament: ES 3; ES 5; ES 7

Rockwool: G + H

Slagwool: RH; ZI

Ceramic fibre: Fiberfrax; Fibermax; Fireline Ceramic; Fybex; MAN; Nextel; PKT; Saffil

1.1. Glass fibres

Glass fibre is produced either as glasswool or glass filament. Glasswool is produced by drawing, centrifuging or blowing molten glass and comprises cylindrical fibres of relatively short length (compared to filaments) (Boyd & Thompson, 1980; McCrone, 1980). Glass filaments are continuously drawn or extruded from molten glass. This class of materials includes longer, large-diameter filaments for textile and reinforcing applications as well as fine-diameter filaments (Mohr & Rowe, 1978).

In the production of glass fibres, finely-powdered sand is used as the major source of silica, and kaolin clay and synthetic aluminium oxides are the most common sources of aluminium. Boric oxide is introduced primarily from colemanite (a natural calcium borate), boric acid and boric acid anhydride. Powdered dolomite [$CaMg(CO_3)_2$] or burnt dolomite (MgO.CaO) is used to introduce magnesium oxide (magnesia) and calcium oxide. Uncalcined and calcined limestone are used as magnesia-free sources of calcium oxide. Fluorspar (CaF_2) is used to introduce fluoride. Sodium sulphate is added to the glass mixture as a firing agent and to assist in dissolving residual grains of sand. Iron oxide (Fe_2O_3) may be added to assist the fibre-drawing process (Loewenstein, 1983; Harben & Bates, 1984).

Compositions of some types of glass used in fibre manufacture are shown in Table 1.

'E' glass was first developed for electrical applications, but currently over 99% of all continuous filament produced is of this type. 'E' glass is generally not defined by composition, but rather by its electrical properties, which are related to its low alkali oxide content (less than 1% sodium, potassium or lithium oxide) (Loewenstein, 1983). 'E' glass is insoluble in hydrochloric acid (Miller, 1975).

'C' glass is characteristic of the glass types used to produce glasswool. It is chemically resistant and is also used in composites that come into contact with mineral acids and as a reinforcement material in bituminous roofing sheet (Loewenstein, 1983).

'A' glass, produced from inexpensive glass scrap, is a soda-lime—silica glass. It currently represents an insignificant proportion of world fibre production (Loewenstein, 1983) and is used only in glass fibre insulation (Watts, 1980).

'S' glass is a high-strength glass developed in about 1960 for applications such as rocket motor cases. Produced only in the USA, it is difficult and costly to make and is therefore limited to very sophisticated, high-technology use (Loewenstein, 1983).

'Cemfil' and 'AR' glass are used for cement reinforcement. These fibres, designed to impart strength and support, can reinforce 20—30 times their weight of cement (Loewenstein, 1983). 'Cemfil' and 'AR' glass differ in composition from other glasses by the inclusion of zirconium oxide, which provides the alkali-resistant property necessary for cement reinforcement but also renders the glass more difficult to process (Lee, 1983).

Table 2 presents the composition of some typical commercial glass fibres based on another classification of glass types (low alkali, lime-alumina borosilicate [I]; soda-lime borosilicate [II, III]; soda-lime [IV]; lime-free borosilicate [V]; and high lead silicate [VI]).

Type IV is used very frequently in glass fibres. Table 3 presents some of the characteristics and physical properties of fibrous glass made from these six glass types (National Institute for Occupational Safety and Health, 1977a).

Glass filaments are primarily made from 'E' glass with the following typical composition (% by mass): SiO_2, 53.5–55.5; CaO, 21.0–24.0; $A1_2O_3$, 14.0; B_2O_3, 5.0–8.0; alkaline oxides, 0.5–1.5; CaF_2, 0.0–0.8; MgO, 0–2; minor oxides, <1.0. Glass filaments exhibit the following properties: high tensile strength, dimensional stability, high heat resistance, resistance to chemical attack, high thermal conductivity, low moisture absorption, high dielectric strength and flame resistance. One type of glass filament has a softening point of 849°C and a specific gravity of 2.63 g/cm^3 (PPG Industries, 1984).

The fibre size of bulk fibrous glass is characterized by the nominal diameter (the median distribution of length-weighted diameters in random bulk samples of the product). Table 4 shows a system that is used to designate glass fibre materials by their nominal diameter ranges. Tables 5 gives the nominal fibre diameters

and the types of binders used for a number of commercial products. The most common resins are phenol-formaldehyde and melamine-formaldehyde (Dement, 1975). More recent data suggest that somewhat lower nominal fibre sizes are associated with building insulation; 3.75–7.5 µm for low-density products and up to 15 µm for roof boards (Owens-Corning Fiberglas Corp., 1987).

Individual fibres in a given product have a range of diameters. The range is generally small for continuous filaments and much wider for wool-type fibres.

1.2. Rockwool and slagwool

Rockwool and slagwool are produced by blowing, centrifuging or drawing molten rock or slag.

Rockwool is typically made from igneous rocks such as diabase, basalt and olivine, and carbonate rocks containing 40–60% calcium and magnesium carbonates (Mansmann *et al.*, 1976; Fowler, 1980; World Health Organization, 1983). Rockwool remains in the glassy state because it is cooled so rapidly that it does not recrystallize. It dissolves in dilute hydrochloric acid (Miller, 1975).

Slagwool is made from the fused agglomerate by-products of certain metal smelting processes (Mansmann *et al.*, 1976; Fowler, 1980; World Health Organization, 1983) and its composition is thus a reflection of the range of components in the different slags used in the melt (Stettler *et al.*, 1982). The properties of the fibre vary not only according to the sources of raw material but also from batch to batch. One typical composition (%) is: SiO_2, 41; $A1_2O_3$, 11; CaO, 35; MgO, 6; Fe_2O_3, 5; miscellaneous, 2 (some sulphur is present). Slagwool made from Alabama furnace slags had the following composition (%): SiO_2, 33–36; $A1_2O_3$, 11–14; CaO, 35–42; MgO, 6–13; Fe_2O_3, 0–23; S, trace-1.66. The absence of significant amounts of sodium and boron is typical of slagwool; it is essentially a calcium aluminium silicate with varying amounts of magnesium and iron, and is usually slightly soluble in hydrochloric acid (Miller, 1975).

The composition of some European and US rockwool and slagwool insulation materials is given in Table 6 (Mansmann *et al.*, 1976; Owens-Corning Fiberglas Corp., 1987). The nominal diameters of rockwool and slagwool products are typically 3–8 µm (Cherrie *et al.*, 1986).

1.3. Ceramic fibres

Ceramic fibres comprise a wide range of amorphous or crystalline, synthetic mineral fibres characterized by their refractory properties (i.e., stability at high temperatures). Ceramic fibres are typically made of alumina, silica and other metal oxides or, less commonly, of nonoxide materials, such as silicon carbide (Arledter & Knowles, 1964). Most ceramic fibres are composed of alumina and silica in an approximate 50/50 mixture. Monoxide ceramics, such as alumina and zirconia, are composed of at least 80% of one oxide, by definition; usually, they contain 90% or more of the base oxide, and specialty products may contain virtually 100%. Other ceramic fibres prepared for special applications may incorporate thoria, magnesia, berylia, titania, hafnia, yttria or potassium titanate. Nonoxide specialty ceramic fibres, such as silicon carbide, silicon nitride and boron nitride, have also been produced (Arledter & Knowles, 1964; Miller, 1982; US Environmental Protection Agency, 1986; Anon., 1987a).

Alumina-silica ceramic fibre may be manufactured in two types: ceramic refractory fibre and ceramic textile fibre. The main distinction between the two fibre types is their size. Ceramic textile fibres are typically longer, ranging from about 155 to 250 mm in length, and have diameters that range from 11 to 20 µm. Refractory fibres are smaller and shorter than textile fibres, with average diameters of 2.2–5.0 µm and lengths varying from 40 to 250 mm. Over 90% of ceramic fibres produced in the USA are refractory fibres (US Environmental Protection Agency, 1986).

Table 7 presents typical composition and Table 8 chemical and physical properties of some commercial ceramic fibres (Zircar Products, 1978a,b; Sohio Carborundum Co., 1986; Fireline, undated; 3M Center, undated; Zircar Products, undated).

2. Production, Use, Occurrence and Analysis

2.1. Production and use

(a) Production

(i) Amounts produced

Rock-/slagwool was first produced in Wales in 1840 (Mohr & Rowe, 1978; Fowler, 1980). By 1885, commercial operations had begun in England (Pundsack, 1976), and soon thereafter they began in Germany (Fowler, 1980; World Health Organization, 1983). The first successful commercial rock-/slagwool plant in the USA began operation in 1897 (Fowler, 1980).

Although a few such plants were in operation in the USA and Europe in the early 1900s, it was not until after the First World War that the industry began to develop and grow (Pundsack, 1976; World Health Organization, 1983). By 1928, there were at least eight plants in the USA, and, by 1939, that number had grown to 25 (Pundsack, 1976), a growth attributable primarily to improvements in glass fibre manufacturing technology (Fowler, 1980; Loewenstein, 1983). Glasswool manufacturers were able to open new markets such as textile manufacturing, while rockwool and slagwool manufacturers continued to compete in the thermal insulation market (Mohr & Rowe, 1978; Fowler, 1980). The number of rockwool and slagwool plants in the USA peaked at between 80 and 90 in the 1950s, and then declined as glasswool began to be used in thermal insulation (Pundsack, 1976). By 1985, there were 58 plants in the USA producing glasswool, rockwool, slagwool or ceramic fibres (US Environmental Protection Agency, 1986). According to the European Insulation Manufacturers' Association, there were 37 rock-/slagwool plants (mainly producing rockwool) and 37 glasswool plants in western Europe and Turkey in 1986.

Production of glass filament began in the USA in the 1930s. By 1985, seven companies were manufacturing textile fibres at 14 plants in the USA (US Environmental Protection Agency, 1986).

Estimated world mineral fibre production for 1973 is presented in Table 9 (World Health Organization, 1983). The quantities of glasswool, rockwool and slagwool products manufactured in the USA in 1977 and 1982 are shown in Table 10 (US Department of Commerce, 1985). The production of glass fibre in the USA from 1975 to 1984 is presented in Table 11 (Anon., 1986). In western Europe, production of glasswool, rockwool and slagwool in 1984 amounted to approximately 1550 million kg. Worldwide production of continuous filament in 1984 was estimated at 1384 million kg (Griffiths, 1986).

Although production of ceramic fibres began in the 1940s, their commercial exploitation did not occur until the early 1970s. World-wide production of ceramic fibres in the early-to-mid-1980s was estimated at 70–90 million kg, with US production comprising approximately half of that amount. With the introduction of

new ceramic fibres for new uses, production has increased significantly over the past decade (US Environmental Protection Agency, 1986).

(ii) Methods of production

Mineral fibre products are generally made in a three-step process: (1) fusion of raw materials, (2) fibre formation and (3) the conversion of fibres into the commercial product. Step 1 is the fusion (melting and mixing) of raw materials in a furnace. Raw materials are selected to impart the desired properties to the product. The liquid is drawn from the furnace to produce a preform for remelt at some future date or flows directly to a fibre-production device. Step 2 consists of fibre formation. Fibres are made by directing a jet of hot gas at the liquid stream or by centrifugal attenuation. Fibres drawn (extruded) through nozzles are called filaments. In step 3, fibres are converted into commercial products by chemical treatment and formation of blankets, mats, yarns, cloth, moulded shapes and other product types.

Glasswool

Glass fibre may be produced in two steps (marble melt process) or in a single step (direct melt process). In the marble melt process, a glass-making furnace is linked to a forehearth and to machines for converting the melt into marbles. The furnace fuses the raw material and homogenizes the melt. Homogenization occurs in two zones of the furnace, called the refining zone and the working zone. Within the refining zone, the raw material is liquefied; as the melt passes to the working zone, the temperature decreases and brings the melt to its 'working' or processing viscosity — 500–1000 poises. The melt exits through the supply channel or forehearth to the marble-making machine. The preformed marbles can be stored, distributed and subsequently remelted for formation into fibres. In the direct melt process, the glass-making furnace is linked to the forehearth and to a bushing, from which the glass is directly formed into fibres. Glass-making furnaces are heated by gas, oil or electricity (Mohr & Rowe, 1978; Loewenstein, 1983).

The principal ways in which glasswool is formed are spinning, flame attenuation and the rotary process (Pundsack, 1976; Mohr & Rowe, 1978).

The spinning process was developed in 1955 as an improvement over the steam-blown process. The raw material is cupola-melted, and molten material falls onto a series of rapidly rotating wheels. The wheels induce attenuation as the fibres fall from wheel to wheel. The fibres produced are finer and longer than those that are steam-blown, making them more suitable for use as insulation (Pundsack, 1976; Mohr & Rowe, 1978).

Flame attenuation, developed in the middle-to-late 1940s, is adaptable to either the direct melt or the preform melt process. In this method, primary glass fibres are drawn to approximately 1 mm in diameter, aligned in a uniform array and introduced into a jet flame blast. Fibres with diameters as small as 0.05 μm can be produced by this method (Mohr & Rowe, 1978).

The rotary process is the result of a series of improvements made to bulk fibre production. The resultant fibre is qualitatively equivalent to those produced by the flame attentuation method, but considerably greater output can be achieved. In this process, the molten media fall into a rapidly rotating hollow cylindrical unit with holes in its vertical side walls. Centrifugal force extrudes the glass steam through the holes of the cylinder, where the molten fibres are further attenuated by peripherally located jet flame burners (Pundsack, 1976; Mohr & Rowe, 1978).

Glass filaments

The production of glass filament differs from that of glasswool, rockwool or slagwool. Nozzles are attached to the bushing on the forehearth, and mechanical drawing is used to form the primary package of fibre strand — the cake. A fine mist of water is sprayed onto the strands as the filaments leave the bushing, and a lubricating sizing is applied before the strands are gathered and wound into a cake. Filament attenuation begins when the hot melt is exuded through the nozzle and ends when it enters the water mist. The total distance travelled during the process of drawing and freezing is usually 10–20 mm (Lee, 1983). Limits to the rate of attenuation appear to be based on the specific type of fibre being produced and the size of the nozzle used in the process. The rate of attenuation is usually 3000–4000 m/min (Loewenstein, 1983).

In the manufacture of continuous glass fibre products, the fibres are first combined in the form of chopped strand mats, continuous strand mats, yarns and yarn fabrics, roofing or surface tissue, rovings and roving cloth. The combining of fibre strands to produce these intermediate products requires various types of secondary processing and treatment (Loewenstein, 1983).

Chopped strand mat is a chemically bound fabric consisting of strands 25–50 mm in length. The starting material for chopped strand mats is usually a cake — the primary package of fibre strands. Fibre strands from the cakes are fed into the forming section, where they are chopped and distributed uniformly over the width of the belt. After cutting, the chopped fibres are dropped onto a conveyor for magnetic sifting to remove any pieces of broken blade introduced by the chopping process. A binder is applied, and the chopped strands are passed through an oven, which removes any residual water and cures the binder. Binders that are commonly used are powdered fusible polyesters and polyvinyl acetate emulsions. Once outside of the oven, the product is immediately passed between water-cooled rollers to consolidate the outer layers of the mat (Loewenstein, 1983).

The basic yarn product is formed by twisting a single strand drawn from a cake. Cabled yarns are formed by simultaneously twisting several strands together; the twisting of 40 strands or more constitutes a cord. Twisting is carried out by feeding strands to a bobbin at a controlled rate (Loewenstein, 1983).

Roofing mat or surface tissue is manufactured from chopped fibres. The fibres are suspended in water and passed to a screen, where they are deposited. A conveyor belt carries the fibres to the binder application area and then to an oven for resin curing. The primary binder used in this process is a urea-formaldehyde resin in solution with bitumen (Loewenstein, 1983).

Rovings are manufactured by winding many strands in parallel. Roving cloth is manufactured by the traditional method of cloth making, which involves beaming the yarn for the warp, spooling some of the yarn for the weft, weaving and finishing. The market for such cloth has declined, although it is the highest quality glass-reinforcing material. Once woven, roving cloth requires no additional treatment to give high-quality laminates. It is sometimes used as an alternative to chopped strand mat (Loewenstein, 1983).

Rockwool and slagwool

In the production of rockwool and slagwool, the raw materials are loaded into a cupola, an upright cylindrical furnace, in alternating layers with batches of coke. The coke is burnt, generating temperatures of approximately 1650°C, to melt the raw materials. The molten stream issues from a hole in the bottom of the

cupola and is made into fibres (Pundsack, 1976; Fowler, 1980).

Fibres are formed by directing a jet of hot gas onto the falling molten stream, which breaks it into small globules that then tail out, producing fibres with semispherical heads. The heads detach as the materials cool, producing fibres and shot (cooled heads) (Pundsack, 1976; Fowler, 1980). In the early 1940s, the Powell or dry process was developed in which a group of rotors operating at high centrifugal speeds mechanically attenuate the molten stream (Fowler, 1980). The Downey process, developed at about the same time as the Powell process, combines a spinning rotor with steam attenuation. The molten stream is distributed in a thin pool over the surface of a dish-shaped rotor and flows over its edge, where it is caught up in a high-velocity stream flow surrounding the dish and is fiberized (Pundsack, 1976; Fowler, 1980). The products of the Powell and Downey processes have a relatively high shot content (Pundsack, 1976). Regardless of the manufacturing process, a substantial fraction of the molten raw material becomes shot rather than fibres. Commercial standards for mineral wool insulation specify an upper limit on the shot content of the product, because it is an ineffective insulator (Fowler, 1980).

Raw fibre is sprayed immediately after its formation with a binder and a lubricating oil to reduce breakage and prevent dustiness (Fowler, 1980; World Health Organization, 1983). Binder materials, such as urea-formaldehyde and phenol-formaldehyde resins, are used; other binder solutions are melamine resins, silicone compounds, soluble and emulsified oils, surfactants, extenders and stabilizers. Silicon compounds are used to impart water repellancy to the fibre, and soluble and emulsified oils provide lubrication. Another function of binders is to provide an interface between the vitreous material and added dyes or resins (Loewenstein, 1983; World Health Organization, 1983). The binder content of the finished products depends on the end-use application of the fibres; normally, less than 5% is added to insulation products.

After application of binder, the fibre is conveyed either to temporary bulk storage or directly to a compression bailing machine or bagging station. Further processing may then occur, depending on the intended use of the materials. When the end-use is pouring wool, the loose fibre is passed between counter-rotating toothed drums, forming approximately 2.5-cm wool pellets that can be more easily handled. Should the product require moderate or substantial structural rigidity or stability, a resin may be added immediately after or in place of the oil treatment. Other rockwool products require more complex finishing; for example, residential structural insulation is often covered with a vapour barrier on one side and untreated paper on the other. For industrial insulation, a wire mesh covering is often used (Fowler, 1980).

In 1980, approximately 70% of the rockwool or slagwool sold in the USA was produced from blast furnace slag. Most of the remainder was produced from copper, lead and iron smelter slag. A small amount was produced with natural rock, which is usually added to the slag to impart flexibility to the fibres (Fowler, 1980). In Europe, slags have been used to a lesser extent (Cherrie & Dodgson, 1986).

Ceramic fibres

Ceramic fibres are produced primarily by blowing and spinning; colloidal evaporation, continuous filamentation and whisker-making technologies (vapour-phase deposition) are used to a lesser extent, mainly for special applications (Arledter & Knowles, 1964).

In the steam-blowing system, natural minerals (kaolin clay) or synthetic blends of alumina and silica are fused in an electric furnace, and the melt is drawn off and blown by pressurized steam or other hot gas. The fibres are collected on a screen and may be processed to remove pelletized material or shot (Arledter & Knowles, 1964; Miller, 1982).

As with the spinning processes for glass and rock fibres, those for ceramic fibres produce a high proportion of long, silky fibres and a relatively low proportion of shot, in contrast to the blowing methods. In this method, a stream of molten material is forced onto rapidly rotating discs, which throw off the molten material tangentially, transforming it into a fibrous form (Arledter & Knowles, 1964; Miller, 1982).

Ceramic fibres of alumina, zirconia, silica, mixtures of zirconia and silica, and thoria have been prepared through evaporation of a colloidal suspension (McCreight *et al.*, 1965). An example of this method is the sol process, which is used to produced the silica-stabilized alumina fibre, Saffil® (Miller, 1982).

The rayon spinnerette method has been used to produce gamma-alumina-spinel, alumina, zircon, alumina-silica mixed oxide, zirconia and titania fibres. Named for its similarity to the technique used to produce rayon thread, this wet spinning process involves dissolution of the raw materials in a suitable solvent and subsequent extrusion of the solution into a liquid bath, where a filament is formed by a combination of precipitation, coagulation and regeneration. Subsequent firing at 1550°C yields polycrystalline fibres (McCreight *et al*., 1965; Rebenfeld, 1983).

Other special ceramic fibres, in particular those composed of nonoxide materials, have been produced by a vapour-phase deposition technique in which a volatile compound of the desired coating material is reduced or decomposed on a resistively heated substrate, such as tungsten wire. The feasibility of making composite polycrystalline filaments by this method has been demonstrated with such materials as boron, boron carbide, silicon carbide and titanium boride (McCreight *et al*., 1965); boron and silicon carbide fibres have been produced commercially. Annual US production in 1986 was 15.9–22.7 tonnes boron fibre and 0.9 tonnes silicon carbide filaments. Materials filamentized by this technique display good mechanical properties but in their present state of development are the least economical (Anon., 1987b).

Vapour-phase deposition processes are also used to manufacture another class of fibres, known as ‘whiskers’. Whiskers are monocrystalline ceramic materials with high strength and micron-sized widths or diameters. Whiskers first came under intense study in 1952 after Herring and Galt determined experimentally that the strength of tin whiskers was an order of magnitude greater than that of ordinary tin. The increased strength of whiskers is attributed to their crystalline perfection and their small dimensions, which minimize the occurrence of the defects that are responsible for the low strength of materials in bulk form. High strength, high elastic modulus, low density and a high melting-point make whiskers useful as reinforcing agents for metals, plastics and ceramics (Levitt, 1970; Parratt, 1972).

Whiskers are produced mainly by vapour-phase techniques, characterized by three methods in which the driving force is primary recrystallization or a step-wise decrease in supersaturation. The methods are evaporation-condensation, chemical reduction and vapour-phase reaction (Campbell, 1970). Although the production of whiskers has developed rapidly in recent years, volumes are still small compared with those of other, more conventional products.

(b) Use

(i) Glass fibre, rockwool and slagwool

The overwhelming majority of glasswool, rockwool and slagwool is produced for thermal and acoustical insulation applications in construction and shipbuilding (Mohr & Rowe, 1978; US Department of Commerce, 1985). In 1980, approximately 80% of the glasswool produced for structural insulation was used in houses (US Environmental Protection Agency, 1986). Rockwool and glasswool, in the form of loose-bagged wool, is pneumatically blown or hand-poured into structural spaces, such as between joints and in attics (Mohr & Rowe, 1978; Fowler, 1980). Bulk rockwool and glass-fibre rovings are incorporated into ceiling tile for fire resistance and thermal and sound insulation (Fowler, 1980; Lee, 1983). Batts, blankets and semirigid boards made of glass- or rockwool fibres are commonly used between structural members of residential and commercial buildings (Mohr & Rowe, 1978).

Plumbing and air-handling systems also require insulation. Pipes are insulated against heat flow with prefabricated sleeves made from moulded glass or rockwool fibres impregnated with phenolic resins and may be used either indoors or outdoors. Sleeves may be applied to steam lines, drains or water lines. Sheet-metal ducts and plenums of air-handling systems are often insulated with flexible blankets and semirigid boards usually made of glass fibre. These forms of insulation may be applied internally or externally throughout the air-handling system (Mohr & Rowe, 1978; Fowler, 1980).

Small-diameter glass fibres (0.05–3.8 μm) have been used in air and liquid filtration, and glass-fibre air filters have been used in furnaces and air-conditioning systems. Glass-fibre filters have been used in the manufacture of beverages, pharmaceuticals, paper and other products, such as swimming-pool filters, and for many other applications (Mohr & Rowe, 1978).

Glass fibre used for aerospace engineering is applied in the form of batts, blankets and moulded parts to the inside of the exterior fuselage skin between the ribs. Special high temperature-resistant materials are applied to high-velocity aircraft at the nose, wing and empennage tips. Marine products have glass or rockwool fibres built into structural components for thermal, acoustical and fire protection. Specific areas of use include motor shrouds, cabin walls and around turbines and similar gear (Mohr & Rowe, 1978).

In addition, glass fibres have a number of uses specific to a particular end-product. Most glass fibre is sold as chopped strand mats, continuous strand mats, rovings, woven rovings, chopped fibres, yarns and yarn fabrics, and roofing mat or surfacing tissue (Loewenstein, 1983). These products have over 30 000 documented uses (Dement, 1973), the most common of which are detailed here. Chopped strand mats are used to reinforce thermoplastics in the construction of boat hulls and decks, vehicle bodies, sheeting and chimneys. This type of mat is used when the laminate is made from the open mould process. Continuous strand mats are used in laminate production when press moulding is employed and to improve the appearance and strength of the laminates. Overlay mats are sometimes used as an alternative to continuous strand mats. Rovings have a variety of uses. They may be chopped to produce chopped strands, woven to produce roving cloth, or wound onto a male mould to give rise to convex-shaped composites such as aircraft nosecones (radomes). Rovings can undergo pultrussion, a process for making reinforced plastic parts in continuous lengths and of uniform cross-section, to produce structural shapes such as beams, rails, rods and tubes for use in frames and ladders and for purposes where electrical insulation is required. If rovings are chopped and impregnated with polyester resin and left uncured, the material may be rolled out into sheet moulding or left as bulk material for future moulding applications. Chopped fibres are primarily used in the production of roofing mat, reinforcement of thermoplastics (as chopped strand mats) and as filler for polyurethane in reaction injection moulding. They can also be incorporated into a polyester resin to form a gelatinous pre-mix for future use. Glass-fibre yarns are used in the manufacture of glass cloth and heavy-duty cord for tyre reinforcement. The major uses of glass cloth are for high-quality printed circuit boards, aeroplane structures, and for fireproof textiles, such as draperies and emergency protective clothing. Roofing mat is commonly used to cover concrete or wooden roofs and may be substituted for linoleum floor covering when impregnated with polyvinyl chloride (Loewenstein, 1983).

Continuous filament fibre glass is used as a conductor of light (fibre optics) for communications, light and image transmission and decoration (Mohr & Rowe, 1978).

(ii) Ceramic fibres

Ceramic refractory fibres are also used as insulation materials. Due to their ability to withstand high temperatures, they are used primarily for lining furnaces and kilns. End-products may be in the form of blankets, boards, felts, bulk fibres, vacuum-formed or cast shapes, paper and textile products (Table 12). Their light weight, thermal shock resistance and strength make them useful in a number of industries (Mohr & Rowe, 1978; US Environmental Protection Agency, 1986).

High temperature-resistant ceramic blankets and boards are used in shipbuilding as insulation to prevent the spread of fires and for general heat containment. Blankets, rigid board and semirigid board can be applied to the compartment walls and ceilings of ships for this purpose. Ceramic blankets are used as insulation for catalytic converters in the automobile industry and in aircraft and space vehicle engines. In the metal industry, ceramic blankets are used as insulation on the interior of furnaces. Boards are used in combination with blankets for insulation of furnaces designed to produce temperatures up to approximately 1400°C. Ceramic boards are also used as furnace and kiln back-up insulation, as thermal covering for stationary steam generators, as linings for ladles designed to carry molten metal and as cover insulation for magnesium cells and high-temperature reactors in the chemical process industry (Mohr & Rowe, 1978; Miller, 1982).

Ceramic felts are used in the metal industry for furnace insulation, firewall protection, packing for stress-relieving of welds, insulation for heat-treating ovens and kilns, and coverings for hot ingots during transport. Felts are used as catalytic combustion surfaces in the hot-forming process for production of metals such as beryllium and titanium. They have also been used as gas turbine silencers and mufflers, high-temperature gaskets and seals for expansion joints, and for high-temperature filtration. Some typical applications for bulk ceramic fibres are as filler for expansion joints, as stuffing wool and as construction material for furnaces and ovens. In steel mills, aluminium and brass foundries, and glass manufacturing operations, bulk fibres are used as loose-fill insulation and as a raw material for casting shapes (Mohr & Rowe, 1978; Miller, 1982).

Approximately 20% of the ceramic fibre produced is cast shaped (Miller, 1982). Bulk fibres are mixed in an aqueous suspension with clays, colloidal metal oxide particles and organic binders. The mix is poured into moulds with fine-mesh screen surfaces to produce such shapes as flat discs with flanges, short pipes, tubing, elbow bends, cores and closed-end cylinders. These cast-shaped ceramic end-products are widely used in smelting, casting and foundry operations as riser sleeves, feeder tubes and reusable surface insulation tiles. Such tiles are used to cover 70% of the body of space shuttles and can withstand temperatures as high as 1260°C (Mohr & Rowe, 1978; Miller, 1982).

Ceramic-fibre paper is used in end-products such as gaskets, combustion chamber linings, metal trough backups, hot tops and ingot moulds, and can be used as a parting agent in metal- and ceramic-forming processes. The ceramic paper may be rolled to form laminated tubes and discs or die cut for electronic

components (Mohr & Rowe, 1978; Miller, 1982).

Ceramic textile products, such as yarns and fabrics, are used extensively in such end-products as heat-resistant clothing, flame curtains for furnace openings, thermo-coupling and electrical insulation, gasket and wrapping insulation, coverings for induction-heating furnace coils, cable and wire insulation for braided sleeving, infrared radiation diffusers, insulation for fuel lines and high-pressure portable flange covers. Fibres that are coated with Teflon® are used as sewing threads for manufacturing high-temperature insulation shapes for aircraft and space vehicles. The spaces between the rigid tiles on space shuttles are packed with this fibre in tape form (Miller, 1982).

Nonoxide fibres, such as silicon carbide, boron nitride and silicon nitride, can be dispersed in resins and cast to form special electrical and aircraft parts such as radomes (microwave windows). These fibres are also used as reinforcing inclusions in metals such as aluminium, gold and silver (Miller, 1982).

Applications of ceramic fibres in the automobile industry are being investigated in Japan, western Europe and the USA (van Rhijn, 1984; Walzer, 1984; Anon., 1987c). Ceramic materials may be a substitute for those automobile materials that are insufficiently resistant to heat and corrosion, or in applications where expensive alloys are needed. These areas include prechambers and swirl chambers in indirect-injection diesel engines and the piston crown in direct-injection diesel engines. Examples of engine components that have been made of ceramic metals are combustion chambers, turbine nozzle rings, turbocharger turbine rotors and heat exchangers (Walzer, 1984).

(c) Regulatory status and guidelines

Statements concerning regulations and guidelines are included as indications of potential exposures. The absence of information on regulatory status for a country should not be taken to imply that that country does not have regulations with regard to man-made mineral fibres. In several countries in which specific exposure standards have not been established for man-made mineral fibres, exposure limits for total or respirable inorganic dust are applied.

Czechoslovakia

The average maximum allowable concentration for glass fibre is 8 mg/ m^3 (International Labour Office, 1980).

Federal Republic of Germany

A limit of 6 mg/m^3 is given for fine dust. Man-made mineral fibres less than 1 µm in diameter are listed as compounds that are justifiably suspected of having carcinogenic potential (Deutsche Forschungsgemeinschaft, 1986).

Finland

The 8-h exposure limit for glass- and mineral wool is 10 mg/m^3 (Työsuojeluhallitus, 1981).

France

An 8-h limit value of 10 mg/m^3 is given for mineral wool fibres (Institut National de Recherche et de Sécurité, 1986).

German Democratic Republic

Dust standards in the German Democratic Republic are based on four ranges of free crystalline silica content, assessed in % of weight: over 50% (I); 20–50% (II); 5–20% (III); and under 5% (IV). Exposure limits are specified as MAK_D (average concentration over a workday of 8 h and 45 min) and MAKK (short-term exposures for periods not exceeding 30 min) in particles/cm^3 ($ppcm^3$) (International Labour Office, 1980), as follows:

Group	MAK_D	MAK_K
	$ppcm^3$	$ppcm^3$
I	100	300
II	250	500
III	500	1000
IV	800	1500

Italy

For glass- and mineral wool with a quartz content greater than 1%, an exposure limit (L) is calculated for particles between 0.7 and 5 µm, by the counting method:

$$L = \frac{4500}{q+3} \text{ in ppcm}^3,$$

where q = numerical % of quartz particles as determined by the phase-contrast method, or by the gravimetric method:

$$L = \frac{30}{q+3} \text{ in mg/m}^3,$$

where q = % (by weight) of quartz determined as total dust, and

$$L = \frac{10}{q+3} \text{ in mg/m}^3,$$

where q = % (by weight) of quartz determined as respirable dust.

If the quartz content is less than 1%, the exposure limit (by the counting method) is 1500 ppcm^3; by the gravimetric method, the exposure limit is 10 mg/m^3 for total dust and 3.33 mg/m^3 for respirable dust (International Labour Office, 1980).

Norway

For glass and rock/slag fibres, the total dust exposure limit is 5 mg/m^3 for an 8-h day (Direktoratet for Arbeidstilsynet, 1981).

Poland

A tentative guideline for an exposure limit for glass- and mineral wool has been established at 4 mg/m^3 for an 8-h work shift (International Labour Office, 1980).

Sweden

A limit of 2 fibres/ml for a full working day has been set for glass fibres (synthetic inorganic fibres) (Arbetarskyddsstyrelsen, 1984).

United Kingdom

The long-term exposure limit (8-h time-weighted average) for exposure to man-made mineral fibres is 5 mg/m^3, as measured by gravimetric sampling methods for total dust. A recommended limit of 1 fibre/cm^3 has been agreed for superfine man-made mineral fibres, defined as fibres with a diameter of less than 3 µm and aspect ratios greater than 3:1 (Health and Safety Executive, 1987).

USA

The US Occupational Safety and Health Administration (1986) has established that an employee's exposure to mineral dusts (crystalline quartz) in any 8-h work shift of a 40-h working week should not exceed the 8-h time-weighted average limit calculated by the following formulae:

$$\text{Total dust} = \frac{30 \text{ mg/m}^3}{\%\ SiO_2 + 2}$$

$$\text{Respirable dust} = \frac{10 \text{ mg/m}^3}{\%\ SiO_2 + 2}$$

The American Conference of Governmental Industrial Hygienists (1986) recommends a threshold limit value for mineral wool fibre and fibrous glass dust with less than 1% quartz of 10 mg/m^3 for total dust.

USSR

For glass and mineral fibres, a maximum admissible concentration of 4 mg/ m^3 has been set (International Labour office, 1980).

Yugoslavia

The exposure limits established for mineral wool and glasswool are 4 mg/m^3 for respirable dust and 12 mg/m^3 for total dust (International Labour Office, 1980).

2.2. Occurrence

(a) Occupational exposure

Exposures to man-made mineral fibres are reported as total dust concentrations or respirable fibre concentrations in air. The definitions and methods of measurement of these concentrations are variable (see section 2.3). For respirable fibres, the upper diameter limit is considered to be either 3 µm (Esmen *et al.*, 1978; World Health Organization, 1985) or 3.5 εa (National Institute for Occupational Safety and Health, 1977a).

Strictly, the term 'fibre' should be applied to all particles with a length-to-diameter ratio of ⩾3:1. Aggregates and other morphologically atypical particles that fit these overall dimensions are not considered to be man-made mineral fibres. In most of the tables presented in this section of the monograph, the convention is adopted of reporting only fibres >5 µm in length.

(i) Exposure in production plants

USA

Williams (1970) reviewed industrial hygiene surveys performed by the Pennsylvania Department of Health in the US fibrous glass industry. The earliest survey was reported in 1944, which was of solvents used in yarn production. Dust measurements were apparently first performed in 1951; surveys were performed in 1947, 1951–1954, 1962, 1964 and 1967 to evaluate in particular exposures to phenol, formaldehyde, noise, hydrogen, fluoride, styrene, methyl methacrylate and dust. [The Working Group noted that reporting of dust measurements as millions of particles per cubic foot (mppcf) of air after impinger collection and light microscopic counting precluded later conversion to total dust, respirable dust or fibres/cm^3 as indices of exposure.]

Johnson *et al.* (1969) took measurements in four facilities producing fibrous glass insulation and in one producing fibrous glass textile products. Table 13 gives total dust and respirable dust concentrations in these facilities; in this study, respirable fibres were defined as those having diameters <5 μm. Table 14 displays measured concentrations of respirable fibres. [The Working Group considered that these measurements are probably indicative of exposure of US production workers in the 1960s.] The authors concluded that 'the results in terms of airborne concentrations of glass fibres and total dust would indicate that the workmen's exposure to these materials is negligible', noting that fibre concentrations in the asbestos textile industry were about 20-fold higher. The judgement that exposures were low was again expressed in 1974 in a review of work practices and controls for the US National Institute for Occupational Safety and Health (Schneider & Pifer, 1974).

The largest body of data on exposure of US production workers was collected as part of an epidemiological study of the industry (Esmen *et al.*, 1979a), which encompassed 16 glasswool, glass filament, rockwool and slagwool plants. Table 15 indicates the type of fibre produced, the number of samples collected and the average nominal fibre size in each facility; Table 16 describes the plant operations that were used to classify jobs in this study; Table 17 shows the concentrations of total suspended particulate matter by type of operation performed; and Table 18 is a summary of fibre concentrations in these facilities. There were large differences in the amounts of total suspended particulate matter, expressed as mg/m^3, and of airborne fibres, expressed as fibres/cm^3, in different plants and between areas of the same plant. There was also wide variation in these parameters in the same area of the same plant: during production of fibres of nominal diameter >6 μm, ⩽40% of airborne fibres were respirable; during the production of fibres of nominal diameter <3 μm, 50–90% of airborne fibres were respirable.

The cumulative distribution of measured concentrations of fibres for each of the 16 facilities is shown in Table 19. The distribution of fibre diameters, as determined by transmission electron microscopy, is shown in Figure 1. A relationship was found between measured average exposures and the nominal diameter of fibre manufactured (Fig. 2). The concentrations of fibres <1 εn, determined by transmission electron microscopy, are shown in Table 20. It can be seen from Tables 18 and 20 that, unlike the situation in asbestos production facilities where fibre counts made by electron microscopy are many times higher than those made by optical microscopy, the fibre concentrations determined by optical and electron microscopy of samples collected in man-made mineral fibre facilities are, with few exceptions, roughly comparable.

The data in Tables 17–20 are based on personal samples taken from within the breathing zones of employees, generally over 7–8 h. The limit of detection for the phase-contrast microscopic evaluations was about 0.0012 fibre/ cm^3; that for fibre detection by transmission electron microscopy was 0.0023 fibre/ cm^3, based on an approximately 8-h personal sample collected at a flow rate of 2.0 1/min (Esmen *et al.*, 1979a).

The measurements reported indicate fibre concentrations in the range of 0.1–0.3 fibre/cm^3 >5 μm in length (Esmen *et al.*, 1979a). These results can be compared to earlier measurements of exposure to fibres during the manufacture of glass fibre (Williams, 1970).

These figures and the results in Table 14, obtained by optical (not phase-contrast) microscopy, suggest that concentrations of airborne fibres decreased somewhat during the period 1969–1979. On the basis of airborne concentrations of respirable dust, approximately 80% of the US facilities had <1 mg/m^3 respirable dust and 80% had <5 mg/m^3 total dust. It was demonstrated that airborne fibre concentrations, expressed as fibres/ cm^3, could not be predicted on the basis of total suspended particulate matter concentrations, expressed as mg/m^3 (Corn, 1979; Esmen *et al.*, 1979a).

In general, concentrations in US rockwool and slagwool facilities were higher than those in fibrous glass facilities. In two plants, approximately 50–90% of fibres measured in collected samples of airborne particulate matter were <3 μm in diameter (by phase-contrast microscopy), and approximately 60–90% were longer than 10 μm. Average airborne fibre concentrations varied from 0.01 to 0.43 fibre/cm^3 in one plant producing slagwool and from 0.20 to 1.4 fibres/cm^3 in one producing rockwool; individual plant areas differed widely in airborne fibre concentration. Total airborne particulate matter averaged 0.05–6.88 mg/m^3 in the slagwool plant and 0.5–23.6 mg/m^3 in the rockwool plant. Thus, there were higher levels of total suspended particulate matter in rockwool and slagwool facilities than in glasswool facilities, although there were large differences between plants (Table 21) (Corn *et al.*, 1976).

Fibre concentrations during ceramic-fibre production in the USA were higher than those in glasswool and continuous glass filament facilities, but were comparable with exposures to airborne fibres in rockwool and slagwool facilities. The individual plants displayed wide differences (Table 22), and the correlation between total suspended particulate matter, expressed as mg/ m^3, and total fibre exposures, expressed as fibres/ cm^3, was not good. Approximately 90% of airborne fibres in the three facilities were determined to be respirable, i.e., <3 μm in diameter, and approximately 95% were <50 μm in length.

There were variations in the percentage of respirable fibres and fibre dimensions, depending on the plant and individual plant operations, with a range of respirable fibres of 71–96% (Esmen *et al.*, 1979b).

Europe

In 1977–1980, scientists at the Institute of Occupational Medicine (Edinburgh, UK) studied 13 European plants, of which six produced rockwool, four, glasswool and three, glass filaments (Ottery *et al.*, 1984). The measurements in this study form the basis for the exposure in the study of Saracci *et al.* (1984a,b), discussed in section 3.3. At each factory, the work force was classified into occupational groups on the basis of job and work zone, and a proportion of each group was selected at random for personal sampling. A total of 1078 samples were taken for counting respirable fibres at rockwool and glasswool plants, generally over 7–8 h. The respirable fibre concentrations given in the original reports were too low by a factor of about two, and were thus reassessed (Cherrie *et al.*, 1986). Tables 23 and 24 present the revised concentrations for the glasswool and rockwool plants; only unrevised figures from the study by Ottery *et al.* (1984) are available for the three continuous filament plants (Table 25).

The range of group arithmetic means in the four glasswool plants was 0.01–0.16 fibre/cm^3, but up to 1.0 fibre/cm^3 was found when the manufacture of special fine-fibre ear plugs was included. In the rockwool plants, the combined arithmetic means for occupational groups were 0.01–0.67 fibre/cm^3. Concentrations of respirable fibres in the continuous glass filament plants were very low: occupational group means ranged from 0.001 to 0.023 fibre/cm^3 (Cherrie *et al.*, 1986).

The median fibre lengths were within the range 8–15 μm for the glasswool plants and 10–20 μm for the rockwool plants. Median fibre diameters ranged from 0.7–1 μm for glasswool plants and 1.2–2 μm for rockwool (Cherrie *et al.*, 1986). The size distribution in two Danish rockwool plants is given in Table 26. A linear regression analysis of log diameter *versus* log length for airborne fibres gave a correlation coefficient ranging from 0.48 to 0.67 for rockwool production and use and for glasswool use, implying that the longer the fibres were, the larger (on average) were their diameters (Schneider *et al.*, 1985).

An experimental simulation of a rockwool production process with conditions similar to those operating in the 1940s was carried out at a Danish pilot plant to

determine the effect on airborne fibre concentrations of addition of oil to the rockwools. The time-weighted average concentrations of respirable fibres, as measured by personal sampling, were about 1.5 fibres/cm^3 with oil and about 5 fibres/cm^3 without addition of oil; the concentrations of inspirable dust were about 6 mg/ m^3 and 100 mg/ m^3, respectively. There was no substantial difference in airborne fibre concentration when simulated batch-produced wool and continuously-produced wool were handled (Cherrie *et al.*, 1987).

The National Swedish Board of Occupational Safety and Health (Arbetarskyddsstyrelsen, 1981) took measurements in all the Swedish glass- and rockwool plants. The results, which were not included in the Institute of Occupational Medicine survey, are shown in Table 27.

The sampling strategy was machine- and not person-oriented, and the aim was to sample at least one person exposed to man-made mineral fibres for each job, machine type and production line. [The Working Group considered that the finding of fibre levels higher than any of those found by the Institute of Occupational Medicine may have been due to the sampling strategy.] The parameters of some selected size distributions in the Swedish measurements were comparable with the values in Table 26 (Krantz & Tillman, 1983).

In a survey of glasswool, rock-/slagwool and ceramic fibre plants carried out by the Factory Inspectorate in the UK, separate samples were taken for fibre counting and for gravimetric determination. Overall duration of sampling was chosen to be representative of the process or operation and not 8-h averages; continuous, full-shift processes were usually monitored for at least 4 h. The overall plant means are shown in Table 28. The percentage of fibres ⩽3µm was 60–80% (Head & Wagg, 1980). [The Working Group considered that the finding of both total dust and fibre concentrations several times higher than in the study of the Institute of Occupational Medicine may have been due to differences in sampling strategy.]

In the same survey, breathing zone and static samples taken during the manufacture of woven and nonwoven glass fibre mats contained 0.3–1.7 mg/m^3 total dust (23 samples); the mean concentration of total fibres ranged from 0.007 to 0.15 fibre/cm^3 (36 samples). Individual samples reached 0.65 fibre/cm^3, but only 0.009 fibre/cm^3 were ⩽3 µm in diameter. The overall percentage of fibres ⩽3 µm in diameter was 8% for one and <1% for the other of the two plants under study (Head & Wagg, 1980).

In two French glasswool plants, the concentration of total dust was 1.0–3.4 mg/m^3 and that of fibres <3 µm diameter was 0.05–0.18 fibre/cm^3. The geometric mean fibre lengths were 2.8 and 3.4 µm and the geometric mean diameters, 0.26 and 0.27 µm, in the two plants, respectively. A magnification of at least 5000× was used, and all measurements were made on photomicrographs (Kauffer & Vigneron, 1987). The study of Ottery *et al.* (1984) and the Swedish (Krantz & Tillman, 1983) and Danish (Schneider *et al.*, 1985) studies were based on elemental analyses, and measurements were made directly on the screen using a magnification of 2000–5000×. [The Working Group noted that use of photomicrographs gives improved results but precludes analysis of fibres other than man-made mineral fibres.]

(ii) Exposures to compounds other than man-made mineral fibres in production plants

The technical history of each of the factories in the European study has revealed a variety of other exposures, but, as these were historical, they could not be quantified. Asbestos was used in all factories by a small number of persons for personal protection and thermal insulation. In four factories, asbestos had been used mostly as sticking yarn [estimated exposure, <1 fibre/cm^3] and cloth [estimated exposure, <10 fibres/cm^3]. Furthermore, loose asbestos may have been handled on an experimental basis. In one plant, olivine was used, which is potentially contaminated with natural mineral fibres with a composition similar to tremolite; the probable average exposure in the preproduction area was estimated to have been 0.1 fibre/cm^3 (Cherrie & Dodgson, 1986).

Exposure to polycyclic aromatic hydrocarbons may have occurred close to the cupola furnaces in three rockwool and in one glasswool plants and in one using an electric furnace. The possibility of exposure to arsenic from copper slags is also mentioned (Cherrie & Dodgson, 1986). In one of the plants in the IARC study (Saracci *et al*, 1984a,b), situated in the Federal Republic of Germany, exposures to coal-tar, bitumen, quartz and asbestos have been identified, but not quantified (Grimm, 1983).

Other airborne toxic contaminants have been measured in US man-made mineral fibre plants, including several potentially carcinogenic contaminants, which were measured in areas and in personal breathing zones at selected locations where exposures were likely to occur: asbestos, <0.02–7.5 fibres/cm^3; arsenic, 0.01–0.48 εg/m^3; chromium (insoluble), <0.002–0.036 mg/m^3; benzene-soluble organics, 0.012–0.052 mg/m^3; formaldehyde, 0.03–20 ppm (0.04–24.4 mg/m^3); silica (respirable), 0.004–0.71 mg/m^3; and cristobalite (respirable), 0.1–0.25 mg/m^3 (Manville, CertainTeed and Owens-Corning Fiberglas Companies, 1962–1987). The range of concentrations was similar to that found in one or more of the plants included in the major US epidemiological study (Enterline *et al.*, 1983; Enterline & Marsh, 1984; Enterline *et al.*, 1987). [The Working Group noted that air sampling records for these plants were obtained periodically, rather than systematically, during the years 1962–1987. It is not possible to derive defensible long-term average exposure estimates from these records. The measured personal exposures are cited only to corroborate the presence of other carcinogens in the environment of selected man-made mineral fibre production workers.]

(iii) Exposure during use

The work environment of US insulating workers was described in 1971 when the major concern was asbestos; however, exposure to man-made mineral fibres, in particular fibrous glass, was also addressed (Table 29; Fowler *et al.*, 1971). More recently, measurements have been made of exposures during production of aircraft insulation and installation of duct insulation, acoustical ceilings, attic insulation (blowing fibrous glass and mineral wool), building insulation (blankets and batts) and duct systems (Table 30; Esmen *et al.*, 1982). The results indicate that exposures of users may exceed those of production workers.

In construction work, the time spent in active use of man-made mineral fibres may vary widely. In the USA, the typical work day of an insulation installer included about 4 h of actual installation (Esmen *et al.*, 1982). The exposure pattern of members of the joiners'and carpenters' union in Denmark was determined by means of questionnaires; 60% of members spent 0.5–15% of their working hours per month using man-made mineral fibres (Schneider, 1984).

During blowing of rock-/slagwool, exposure to fibres ⩾1 µm in diameter was 0.035 fibre/cm^3 for a worker in a lorry and 0.55 fibre/cm^3 for a worker who directed the flow of rock-/slagwool into the house. In another study, the levels were 0.9–1.4 fibres/cm^3 for an installer in a house and 0.09–0.24 fibre/cm^3 in the lorry. The time-weighted average concentration for all measurements was 0.6 fibre/ cm^3, as determined by optical microscopy (Zirps *et al.*, 1986). It was stated that a typical work day of an installer included about 4 h exposure to fibres, an estimate also used by Esmen *et al.* (1982).

Large surveys have been made of user industries in the UK and Scandinavia (Schneider, 1979a; Head & Wagg, 1980; Hallin, 1981; Schneider, 1984). The most important is the construction industry, in which a great variety of man-made mineral fibre products are used. [The Working Group noted that full-shift sampling

had not been used in these surveys, but the lengths of time sampled were designed to be representative of the particular product or operation being studied. Since total dust concentrations were measured with various sampling heads of different efficiencies, comparisons of total dust concentrations can be only indicative.] The results from the Swedish and Danish surveys are shown in Tables 31 and 32. The distribution of single results for respirable fibre concentrations had geometric means of 0.22 and 0.14 fibre/cm^3 and geometric standard deviations of 3.3 and 3.8 in the Swedish and Danish surveys, respectively (Schneider, 1984). Very few results exceeded 2 fibres/cm^3. Information on fibre size from the Danish user industry is given in Table 26 (Schneider *et al.*, 1985). The geometric mean respirable fibre concentration was 0.046 fibre/cm^3 in open and ventilated spaces and 0.50 fibre/cm^3 in confined and poorly-ventilated spaces (Schneider, 1984). Handling of man-made mineral fibre batts can redisperse gypsum dust from previous installation of gypsum boards, and high concentrations of respirable gypsum fibres have been found: 30 fibres/cm^3 (as determined by scanning electron microscope; length >5 µm) and 44 mg/m^3 (total dusalculated by the Working Grou-min period (Schneider, 1979a).

In a UK survey of exposures during insulation, application of loose fill appeared to generate the highest respirable fibre concentrations. The survey also included measurements taken during use of ceramic fibres (Table 33) (Head & Wagg, 1980).

Levels of total dust and respirable fibres (<3 µm in diameter) during the use of fine-diameter, special-purpose glass fibres in the UK and the USA are summarized in Table 34. In 1980–1983, the UK Factories Inspectorate (1987) surveyed factories where man-made mineral fibres of <3 µm in nominal diameter were used. The concentrations of total dust and airborne fibres are shown in Table 35.

Data on the nominal diameter of fibres in old glasswool, rockwool and slagwool can give information about the presence of fine fibres in the original bulk material.

In the Federal Republic of Germany, eight samples of old insulating materials (1947–1963) were compared with five samples of materials produced by modern techniques; small pieces (1.44 cm^2, 1–4 mm thick) were investigated under a scanning electron microscope. It was concluded that there were some significant differences between specific products regarding the lowest fibre diameters, but no significant difference between old and new products. The fraction of fibres with diameters <1 µm or <3 µm in the old products was comparable to that in the modern ones. The samples represented a broad range of manufacturing methods (Poeschel *et al.*, 1982).

In Denmark, nine rockwool samples from a single manufacturer covering the years 1953–1980 were tested in a dust box by shaking (5 g) of bulk material. Scanning electron microscopic analysis of the generated airborne dust showed a decreasing trend with time for the relative content of thin fibres (<0.25 µm and 0.5 µm in diameter), in particular for the length-weighted diameters (Schneider & Smith, 1984).

During removal of rockwool insulation laid in a loft in 1951,8-h time-weighted averages in the breathing zone were 9 fibres/ cm^3 (average for three workers) and 33 mg/m^3 total dust. No binder or dust suppressant had been used in 1951. Subsequently, new man-made mineral fibre was applied in the same loft, giving 0.5 fibre/cm^3 and 16 mg/m^3. The diameter distribution of the airborne fibres generated during removal of the old product was comparable to that measured during the use of modern products (Schneider, 1979a). Theoretical calculations indicate that not only the nominal diameter but also the diameter distribution of fibres in the bulk material, as well as the ventilation rate, have an effect on the size distribution and concentration of airborne fibres (Schneider *et al.*, 1983).

Ceramic fibres may transform into cristobalite upon heating (Aldred, 1985; Strübel *et al.*, 1986). Workplace exposure measurements during removal of ceramic fibre insulation from high-temperature applications have shown significant exposures to cristobalite (Gantner, 1986).

In the production of reinforced plastics, dust concentrations were 0.001–0.01 total fibre/cm^3 and up to 0.002 respirable fibre/ cm^3 (three samples) during glass mat preparation and spray lay-up. Trimming operations were more dusty: the total dust concentration reached 62 mg/m^3 in one plant with apparently poor dust control. The mean total fibre concentration in the plant was 0.28 fibre/cm^3 (range, 0.02–1.43), and the respirable fibre concentration was 0–0.08 fibre/cm^3. In another plant with better dust control, total fibre concentrations were only 0.005–0.06 fibre/cm^3, of which about half were respirable (<3 µm in diameter) (Head & Wagg, 1980).

Mineral fibres may also be produced unintentionally. It has been reported that exposure to silicon carbide fibres may occur during the production of silicon carbide. Fibre levels were less than 1 fibre/cm^3, and the highest short-term average concentration was 5 fibres/ cm^3 (as measured by optical microscopy). The geometric mean length was 4.5 µm and the geometric mean diameter, 0.23 µm (as determined by scanning electron microscopy) (Bye *et al.*, 1985).

Table 36 gives an overall summary of estimated fibre concentrations generated during the production and use of man-made mineral fibres, as well as typical levels in nonindustrial environments and outdoor air.

(b) Ambient air

In 36 of 300 ambient air samples taken from sites in California, USA, the arithmetic mean of glass fibres/cm^3 was 0.0026 (range, nondetectable—0.009), as determined by phase-contrast optical microscopy (for fibres with diameters >2.5 µm) and electron microscopy. The geometric mean diameter was 2.2 µm and length, 16 µm (Balzer, 1976). [The Working Group noted that the detection limit of the electron microscopic method was not stated.]

In the Federal Republic of Germany, fibre concentrations in three large cities and in one rural area (Krahm) were monitored in 1981—1982 (Table 37). Samples were analysed under a transmission electron microscope with energy-dispersive X-ray and electron diffraction analysis after ashing to remove organic material; the total fibre count thus represents only inorganic fibres. The fibres identified as glass constituted less than 1% (Krahm) to 5% (Dortmund) of the total concentration of inorganic fibres and represented 3% (Krahm) to 40% (Dortmund) of the asbestos concentration (Höhr, 1985). [The Working Group estimated from the data that about 25% of the glass fibres had diameters exceeding 0.2 µm and lengths exceeding 5 µm and would thus have been counted by optical phase-contrast microscopy.]

The total fibre dust emission to the environment for the whole of the Federal Republic of Germany from the manufacture of man-made mineral fibres has been estimated at 1.8 tonnes per year. The fibres are mostly coarse, and 350 kg of fibres 8—20 µm in length and only 80 kg of fibres <1 µm in diameter are estimated to be emitted per year (Tiesler, 1983).

(c) Other exposures

In the late 1960s, concern was expressed over health problems associated with possible erosion of fibrous glass used to line ventilation and heating ducts. Glass

fibres were found in settled dust on walls and permanent structures in buildings (Cholak & Schafer, 1971); and, in the San Francisco Bay area, CA, USA, the glass fibre concentration in 13 ventilation systems was undetectable—0.002 fibre/cm^3 in 1968—1971, as determined by combined electron and optical microscopy (geometric mean diameter, 1.3 µm; length, 11 µm). In some cases, there was a decrease in fibre concentration after fibre-containing outdoor air had passed through the air transmission system (Balzer *et al.*, 1971; Balzer, 1976).

Medium-efficiency air-cleaning units most often contain glass fibre filters. Laboratory tests showed that fibre entrainment did not depend on operating velocity and that filter damage (tears longer than 8 cm) may increase entrainment. The test implied that, after a short initial surge in concentration, the indoor fibre concentration level becomes indistinguishable from the ambient level (assumed to be 0.00007 fibre/cm^3) within one day (Esmen *et al.*, 1980). Gross fibre contamination of a house with a faulty air-conditioning system was reported (Newball & Brahim, 1976).

Fibre concentrations in air in a hospital building in which air ducts were lined with glass fibres were 0.003—0.020 fibre/ cm^3. No fibre was found in two rooms in a section where ducts were not lined (National Institute for Occupational Safety and Health, 1980).

Extensive measurements of concentrations of man-made mineral fibres in schools and office buildings have been carried out in Denmark. A random sample of mechanically-ventilated schools, most of which had man-made mineral fibre noise baffles or linings in ducts, showed concentrations of undetectable—0.0001 fibre/cm^3 (Table 38). Under special circumstances, such as after water damage or faulty construction, high concentrations were found, e.g., 0.084 fibre/cm^3, in a nursery school in which the ceilings were covered with man-made mineral fibre boards containing water-soluble binder. In general, concentrations were much lower. The institutions were classified into those in which the ceilings were covered with man-made mineral fibre products with resin or water-soluble binder and those which had no readily visible man-made mineral fibre products. The fibres were identified and counted using phase-contrast light microscopy with polarization (Schneider, 1986; Rindel *et al.*, 1987).

Detection of man-made mineral fibres on surfaces can indicate the presence of such fibres in the indoor environment (Schneider, 1986; Rindel *et al.*, 1987). Concentrations in nursery schools on surfaces that were not cleaned regularly ranged typically from 0.3 to 4.5 nonrespirable man-made mineral fibres/cm^2 but reached 760 respirable fibres/cm^2 and 1160 nonrespirable fibres/cm^2; the presence of fibres on fingers was also demonstrated (Schneider, 1986). Fibres have been found in the eyes of office workers (Alsbirk *et al.*, 1983) and of man-made mineral fibre production workers (Schneider & Stockholm, 1981).

Filtering facepiece respirators may have a filter medium containing super-fine man-made mineral fibres. It has been reported that these respirators may release fibres during use (Howie *et al.*, 1986). During laundering, fibrous glass textiles may contaminate other clothing with which they are washed (Lucas, 1976).

2.3. Analysis

Optical microscopy, electron microscopy and gravimetry are the methods most commonly used for measuring man-made mineral fibres in air. Methods of sampling and of optical and scanning electron microscopy have improved with time. The World Health Organization (1985) has proposed reference methods.

Dust samples are collected by drawing a measured quantity of air through a filter. Mass and fibre concentrations have been determined from separate samples (Schneider, 1979a; Head & Wagg, 1980), and from single samples used in turn for weighing and particle counting (Esmen *et al.*, 1979b; Hallin, 1981; Ottery *et al.*, 1984). Usually, separate samples are taken for electron microscopy (Schneider, 1979a; Ottery *et al.*, 1984). Fibre counting requires that the dust be uniformly distributed across the filter, and therefore open-faced filters are used. If the same sample is also used for gravimetric determination, sampling efficiency may not conform to the definition of total dust or inspirability. Settled dusts are sampled from surfaces or from the skin using sticky foils (Cholak & Schafer, 1971; Cuypers *et al.*, 1975; Schneider, 1986). Mucous thread and clumps from the inner corner of the eye can be used to estimate particle deposition in the eyes (Schneider & Stockholm, 1981).

In the USA, the recommended standard procedure is to collect samples for fibre counting on a 0.8-µm pore size and 25- or 37-µm diameter Millipore type AA filter mounted in an open-face cassette (National Institute for Occupational Safety and Health, 1977b, 1980, 1984). The same procedure is required by the US Occupational Safety and Health Administration (1986) in its enforcement of the asbestos standard. The sampler is mounted either in a worker's breathing zone or on individuals in both occupational and nonoccupational environments whose exposure is to be characterized. Air is drawn through the filter with a battery-powered personal sampler pump at a rate of 2 1/min for 30 min. Fibres are then counted and sized by area fields defined by a calibrated graticule using phase-contrast microscopy at 400—450X magnification. The number of fields counted (100) and the uncertainty in fibre count as a function of total fibre count are specified. Results have been reported as total fibres/cm^3 air and as fibres in selected diameter and length classification, with fibres >5 µm in length and <3 µm in diameter as the predominant reporting mode. The latter corresponds to the definition of fibre size in compliance with the Federal work place standard for asbestos. Total or respirable dust concentrations are typically determined with a 37-mm membrane filter at a sampling rate of 21/ min. The filter and sample are desiccated and reweighed, and the airborne particulate, expressed as mg/m^3, is calculated for the sampling rate and the filter weight gain (National Institute for Occupational Safety and Health, 1984).

In the method of the World Health Organization (1985), using optical microscopy, the filter is rendered optically transparent, and the fibres present within randomly selected areas are counted using a phase-contrast microscope with a magnification of 500X. The total number of fibres on the filter is calculated to give the airborne concentration. The microscopic techniques are based on those commonly adopted for asbestos monitoring. A fibre is defined as any particle that has a length >5 µm and a length:diameter ratio >3; a respirable fibre is any such fibre with a diameter <3 µm. Since the visibility of the thinnest fibres is dependent on the optical parameters of the microscope and on the refractive index of the filter medium, these parameters are specified. The performance of the microscopic counting system is tested by using a standard test slide. Criteria have been established to count fibres that are branching or crossing or that are attached to other particles, and the correct criteria must also be used for counting fibres that are not completely within the counting field (World Health Organization, 1985). Some rules may overestimate the prevalence of long fibres. For example, if all fibres located wholly or partly in a field of view are sized, fibre length will be overestimated, and, since length is correlated to diameter, the diameter distribution will also be distorted (Schneider, 1979b).

The diameter distribution of bulk materials is often expressed as accumulated length within each given diameter interval (accumulated lengths or length-weighted diameters), because this distribution is independent of the method of sample preparation. The median is called the nominal diameter of the material. Diameter distributions generated in terms of number frequency *versus* fibre diameter are also used. This procedure results in a smaller nominal diameter (Schneider & Holst, 1983).

In optical microscopy, the limit of visibility is about 0.2 µm. For conventional man-made mineral fibres, this is no great disadvantage, since only a small percentage of fibres with lengths >5 µm are thinner. The median diameter of airborne fibres such as microfibres and other special-purpose fibres, however, can

range from 0.1 to 0.3 µm, and therefore a substantial proportion of these fibres would not be detected using optical microscopy (Rood & Streeter, 1985). Furthermore, some of these fibre types may have a refractive index close to that of the filter medium, further increasing the difficulty in detecting them (UK Factories Inspectorate, 1987). With the method of the World Health Organization (1985), a detection limit of 0.05 fibre/ cm^3 can normally be achieved without difficulty, and lower detection levels may be possible under circumstances in which contamination from other particles is negligible. The risk of obtaining false-positive results can be reduced by adding polarization analysis to phase-contrast microscopy: with this method, detection limits of <0.001 fibre/cm^3 can be obtained (Schneider, 1986).

Scanning electron microscopy is the method of choice for accurate identification of fibre type and for accurate sizing; with a magnification of 5000X, it is possible to detect fibres of about 0.05 µm in diameter. To ensure consistent results, instrument parameters must be specified. A reference method for measuring the size distribution of airborne man-made mineral fibres in work place air, including rules for evaluating fibres that are branching, crossing or attached to other particles, has been published (World Health Organization, 1985). Samples for scanning electron microscopy are taken on filters that have a smooth surface suitable for direct examination (Nuclepore). After sampling, part of the filter is cut out, mounted on a specimen stub and coated, preferably with a thin layer of gold. In practice, the detection limit of scanning electron microscopy is about 0.1 µm (Middleton, 1982).

Transmission electron microscopy may be required to detect very thin fibres (Rood & Streeter, 1985). The elemental composition of individual fibres can be determined with an energy-dispersive X-ray analysis attachment combined with an electron microscope (Middleton, 1982). Fibres are counted and sized either on photomicrographs (World Health Organization, 1985) or directly on the screen.

3. Biological Data Relevant to the Evaluation of Carcinogenic Risk to Humans

3.1. Carcinogenicity studies in animals

Data used in the evaluation are summarized in Table 41, at the end of this section (p. 106).

Glasswool

(a) Inhalation

Rat: Groups of 46 young adult male Sprague-Dawley rats were exposed by inhalation to fibres >5 µm in length, at concentrations of 0.7×10^6/1 [700 fibres/cm^3] ball-milled fibreglass (24.2% fibres with diameter <3 µm) or 3.1×10^6/1 [3100 fibres/cm^3] UICC amosite asbestos (61.9% fibres with diameter >3 µm), for 6 h per day on five days per week for three months and were then observed for 21 months. One group of 46 unexposed animals served as controls. Groups of four to ten animals per exposure group were killed at 20 days, 50 days, 90 days, six months, 12 months and 18 months, and the remainder at 24 months. No pulmonary tumour was observed in animals that were killed or died prior to the end of the study. Nonsignificant [$p > 0.05$] increases in the number of bronchoalveolar tumours were observed in 2/11 (adenomas) fibreglass-treated animals and 3/11 (two adenomas, one carcinoma) amosite-treated animals compared with 0/13 controls killed at the end of the study (Lee *et al.*, 1981). [The Working Group noted the short exposure period and the small number of animals available for evaluation.]

Groups of 24 male and 24 female Wistar IOPS AF/ Han rats, eight to nine weeks old, were exposed by inhalation to dust concentrations of 5 mg/m^3 (respirable particles) French glasswool (42% of fibres <10 µm in length, 69% < 1 µm in diameter), US glasswool (JM 100; 97% respirable fibres <5 µm in length, 43% total fibres <0.1 µm in diameter) or a Canadian chrysotile fibre (6% respirable fibres >5 µm in length) for 5 h per day on five days per week for 12 or 24 months. An unspecified number of rats was killed either immediately after treatment or after different periods of observation (for seven, 12 and 16 months after exposure for animals exposed for 12 months; four months after exposure for those exposed for 24 months). One relatively undifferentiated epidermoid carcinoma of the lung was observed in 1/45 rats treated with French glasswool, and nine pulmonary tumours were seen among 47 rats treated with chrysotile. No tumour was found among 48 rats treated with US glasswool or among 47 control rats not exposed to dusts (Le Bouffant *et al.*, 1984). [The Working Group noted that, because of the lack of survival data, the exact incidences of tumours could not be ascertained.]

Groups of 50 male and 50 female SPF Fischer 344 rats, seven to eight weeks old, were exposed by inhalation to approximately 10 mg/m^3 respirable dust [size unspecified] of US 'microfibre' glasswool (JM 100) or UICC Canadian chrysotile for 7 h per day on five days per week for 12 months and were observed for life. Fifty rats of each sex served as chamber controls. Groups of three to five animals per group were killed at three, 12 and 24 months. Two studies of similar design, A and B, using animals from the same source were conducted at the same time in different laboratories; study B was a part of the study by Wagner *et al.* (1984) which is reviewed in detail below. The authors reported that cumulative exposure to chrysotile was approximately the same in both studies, but cumulative exposure to glasswool was significantly less in study A. No pulmonary neoplasm was observed at three or 12 months. Two of four chrysotile-exposed male rats in study A killed at 24 months had bronchoalveolar tumours (one adenoma, one adenocarcinoma); no tumour was found in animals at 24 months in study B. The incidences of pulmonary tumours (adenomas and adenocarcinomas combined) in the rats in study A observed for life were: chrysotile —males, 9/29; females, 2/27; glasswool — males, 0/28; females, 0/27; control — males, 3/27; females, 0/26. The rates in study B were: chrysotile — males, 7/24; females, 5/24; glasswool — males, 1/24; females, 0/24; control — males, 0/24; females, 0/24 (McConnell *et al.*, 1984). [The Working Group noted that the fibre dimensions used in study A were not reported.]

Groups of 48 SPF Fischer rats [equal numbers of males and females (McConnell *et al.*, 1984); age unspecified] were exposed by inhalation to dust concentrations of approximately 10 mg/m^3 glasswool or chrysotile for 7 h per day on five days per week for 12 months (cumulative exposure, 17 500 mg × h/m^3 for each group). The fibrous dust samples used (and the size distributions of those airborne fibres longer than 5 µm) were: glasswool with resin coating ([source unspecified] 72% fibres <20 µm in length, 52%⩽ 1 µm in diameter), glasswool without resin coating (58% ⩽20 µm in length, 47% ⩽1 µm in diameter), US glasswool (JM 100; 93% ⩽20 µm in length, 97% ⩽1 µm in diameter) and UICC Canadian chrysotile (39% >10 µm in length, 29% >0.5 µm in diameter). Six rats were removed from each group at the end of exposure to study dust retention, and a similar number of animals was sacrificed one year later for the same purpose. The remainder were held until natural death [survival times not reported]. During the period 500–1000 days after the start of exposure, one pulmonary adenocarcinoma occurred in 48 rats exposed to glasswool with resin and one in the 48 rats treated with US glasswool. One benign adenoma was observed in 47 rats exposed to glasswool without resin, and 11 adenocarcinomas and one benign adenoma with some malignant features occurred in 48 rats treated with chrysotile. No lung tumour was observed in a group of 48 untreated controls (Wagner *et al.*, 1984). [The Working Group noted that, because of inadequate data on survival, the exact tumour incidences could not be established.]

Groups of 52—61 female, 100-day-old Osborne-Mendel rats were examined after exposure by inhalation (nose only) to various types of glasswool dusts for 6 h per day on five days per week for two years and were then observed for life. Groups of 59 chamber and 125 room controls were available. The types of glass fibres were classified according to geometric mean diameter, as follows: (1) glasswool with no binder — diameter, 0.4 µm; mass concentration, 2.4 mg/ m^3; 81 % respirable — 3000 fibres/ cm^3 with 530 fibres/ cm^3 > 10 µm in length and ⩽ 1.0 µm in diameter; or 0.24 mg/m^3 (300 fibres/cm^3); (2) loose 'blowing wool' used for building insulation — diameter, 1.2 µm; mass concentration, 4.4 mg/ m^3,30% respirable — 100 fibres/ cm^3 with 30 fibres/ cm^3 > 10 µm in length and ⩽ 1.0 µm in diameter; (3) fibrous glass building insulation with binder — diameter, 1.1 µm; mass concentration, 9.9 mg/ m^3; 13% respirable — 100 fibres/ cm^3 with 25 fibres/ cm^3 >10 µm in length and ⩽1.0 µm in diameter; or 1 mg/m^3 (10 fibres/cm^3); (4) binder-coated building insulation — diameter, 3.0 µm; mass concentration, 7.0 mg/m^3; 19% respirable — 25 fibres/cm^3 with 5 fibres/cm^3 >10 µm in length and ⩽ 1.0 µm in diameter. No respiratory tract tumour was observed in any group. The various forms of fibrous glass did not affect survival and caused little pulmonary cellular change. Of 57 rats exposed to UICC crocidolite asbestos (3000 fibres/cm^3; 5% fibres ⩽ 5 µm in length: mean, 3.1 ± 10.2 µm), three developed one mesothelioma and two, bronchoalveolar tumours (Smith *et al.*, 1987).

Female Wistar rats, 12 weeks old, were exposed in nose-only tubes to fibre aerosols for 5 h, four times a week, for a total exposure period of one year (total exposure, 1000 h). The test group was exposed to US glasswool (JM 104) shortened for 50 min in a knife mill (fibre lengths: 10% <2.0 µm, 50% <4.8 µm, 90% <12.4 µm; fibre diameters: 10% <0.23 µm, 50% <0.42 µm, 90% <0.80 µm; aerosol concentration, 3.0 ± 1.8 mg/m^3, 576 ± 473 fibres/ cm^3; cumulative dose, 3000 mg/ m^3 × h). Two positive-control groups of 50 rats were exposed either to South African crocidolite, containing slightly longer fibres than UICC crocidolite (fibre lengths: 90%>0.72 µm, 50% >1.5 µm, 10%>4.5 µm; fibre diameters: 90% >0.17 µm, 50%>0.27 µm, 10%>0.46 µm; aerosol concentration, 2.2± 1.3 mg/m^3, 2011 ± 835 fibres/cm^3; cumulative dose, 2200 mg/m^3 × h), or to Calidria chrysotile (from California, USA; fibre lengths: 90% >2.0 µm, 50% >6.0 µm, 10% >14.0 µm; fibre diameters: 90%>0.28 µm, 50%>0.67 µm, 10% > 1.6 µm; aerosol concentration, 6.0 ± 5.9 mg/m^3, 241 ± 165 fibres/cm^3; cumulative dose, 6000 mg/m^3 × h). Two negative-control groups were available: 55 rats were exposed to clean air and 50 rats had no treatment. Only 1/107 rats treated with glasswool developed a primary squamous-cell carcinoma of the lung; median lifetime of the group was 110 weeks. In the group treated with crocidolite, 1 / 50 rats developed a lung adenocarcinoma; median lifetime of the group was 111 weeks. No lung tumour was detected in the group treated with chrysotile (median lifetime, 109 weeks), or in either of the two negative-control groups (median lifetimes, 108 weeks). A further group treated with glasswool also inhaled 100 ppm (260 mg/ m^3) sulphur dioxide for 5 h, five times a week for one year; 1/108 rats had a lung adenoma; median lifetime of the group was 106 weeks. In the corresponding control group of 50 rats exposed only to 100 ppm sulphur dioxide, no lung tumour was detected; median lifetime was 99 weeks. According to the authors, the low tumour incidence in the crocidolite-treated group might have been due to the relatively low lung burden of about 1 mg dust, and the absence of tumours after exposure to Calidria chrysotile to the lower persistence of these fibres than of UICC chrysotile samples (Muhle *et al.*, 1987).

Hamster. Groups of 30 or 35 hamsters [sex and age unspecified] were exposed by inhalation to fibres >5 µm in length, at concentrations of 0.7 × 10^6/1 [700 fibres/cm^3] ball-milled fibreglass (24.2% fibres with diameter <3 µm) or 3.1 × 10^6/1[3100 fibres/cm^3] UICC amosite asbestos, for 6 h per day on five days per week for three months and were then observed for 21 months. One group of 30 unexposed animals served as controls. Groups of one to eight animals per exposure group were killed at 50 days, 90 days, six months, 12 months and 18 months, and the remainder at 24 months. No pulmonary tumour was observed in any group (Lee *et al.*, 1981). [The Working Group noted the short exposure period and the small number of animals available for evaluation.]

Groups of 60—70 male, 100-day-old Syrian golden hamsters were examined after exposure by inhalation (nose only) to various types of glasswool dusts for 6 h per day on five days per week for two years and were then observed for life. Groups of 58 chamber and 112 room controls were available. The types of glass fibres were classified according to geometric mean diameter as follows: (1) glasswool with no binder — diameter, 0.4 µm; mass concentration, 2.4 mg/ m^3; 81% respirable — 3000 fibres/cm^3 with 530 fibres/ cm^3 >10 µm in length and ⩽1.0 µm in diameter; or 0.24 mg/m^3 (300 fibres/cm^3); (2) loose 'blowing wool' used for building insulation — diameter, 1.2 µm; mass concentration, 4.4 mg/m^3; 30% respirable — 100 fibres/ cm^3 with 30 fibres/ cm^3 >10 µm in length and ⩽ 1.0 µm in diameter; (3) fibrous glass building insulation with binder — diameter, 1.1 µm; mass concentration, 9.9 mg/ m^3, 13% respirable — 100 fibres/ cm^3 with 25 fibres/ cm^3 > 10 µm in length and ⩽ 1.0 µm in diameter; or 1 mg/m^3 (10 fibres/cm^3); (4) binder-coated building insulation —diameter, 3.0 µm; mass concentration, 7.0 mg/m^3; 19% respirable — 25 fibres/cm^3 with 5 fibres/cm^3 >10 µm in length and <1.0 µm in diameter. A second group of 38 animals was also exposed to the latter fibre because of a high death rate in the first group that was unrelated to fibre exposure. No respiratory-tract tumour was observed in the glass fibre-treated or room-control groups; one of the 58 chamber controls had a bronchoalveolar tumour. The various forms of fibrous glass did not affect survival and caused no pulmonary lesion. Among 58 hamsters exposed to UICC crocidolite asbestos (3000 fibres/cm^3; *5%* fibres ⩽ 5 µm in length; mean, 3.1 ± 10.2 µm), no pulmonary tumour occurred (Smith *et al.*, 1987).

Guinea-pig: Groups of 31 male albino guinea-pigs [age unspecified] were exposed by inhalation to fibres >5 µm in length, at concentrations of 0.7 × 10^6/1 [700 fibres/cm^3] ball-milled fibreglass (24.2% fibres with diameter <3 µm) and 3.1 × 10^6/1 [3100 fibres/cm^3] UICC amosite asbestos, for 6 h per day on five days per week for three months and were then observed for 21 months. One group of 31 unexposed animals served as controls. Groups of one to ten animals per exposure group were killed at 50 days, 90 days, six months, 12 months and 18 months, and the remainder at 24 months. No pulmonary tumour was observed in animals that were killed or died prior to the end of the study. Bronchoalveolar adenomas were observed in 2/7 fibreglass-treated animals, 0/5 asbestos-treated animals and 0/5 controls killed at the end of the study (Lee *et al.*, 1981). [The Working Group noted the short exposure period and the small number of animals available for evaluation.]

Baboon: Two groups of ten male baboons *(Papio ursinus)*, weighing approximately 6—8 kg, were exposed by inhalation to dust clouds of US glasswool (blend of JM 102 and JM 104; concentration of respirable dust, 5.80 mg/ m^3; >60% of fibres <6.3 /µm in length, >70% of fibres <1.0 µm in diameter, 35% were <0.5 µm in diameter) or a UICC crocidolite standard reference sample (concentration of respirable dust, 13.45 mg/m^3; <25% of fibres >3.2 µm in length, <20% of fibres >0.5 µm in diameter) for 7 h per day on five days per week for up to 35 or 40 months, respectively. Lung biopsies were carried out on pairs of animals at eight, 18 and 30 months, respectively, and at six to seven months after termination of exposure. Animals that died spontaneously were also subjected to autopsy. No tumour occurred after exposure to either of the dusts. The authors stated that, in inhalation experiments with asbestos carried out on monkeys and baboons over the preceding 25 years, only one animal exposed to crocidolite for 15 months had developed a mesothelioma five years after start of exposure (Goldstein *et al.*, 1983). [The Working Group noted the very short duration of the study in relation to the life span of these animals and that no untreated control was reported.]

(b) Intratracheal instillation

Rat: Groups of female Wistar rats, 11 weeks of age, received 20 weekly intratracheal instillations of 0.5 mg/dose US glasswool (JM 104; median fibre length, 3.2 μm; median diameter, 0.18 μm) or South African crocidolite (total dose, 10 mg; median fibre length, 2.1 μm; median diameter, 0.2 μm) in 0.3 ml saline or saline alone. Median lifetimes were 107, 126 and 115 weeks for the groups receiving glass fibres, crocidolite and saline only, respectively. A statistically significant increase (5/34 animals) in the incidence of lung tumours was observed with the glass fibres; one tumour was an adenoma, two were adenocarcinomas and two were squamous-cell carcinomas. The mean life span of animals with tumours was 113 weeks; the life span of the first animal with a tumour was 96 weeks. Of 35 rats given crocidolite, 15 developed lung tumours (nine adenocarcinomas, two squamous-cell carcinomas and four mixed tumours; mean life span of tumour-bearing animals, 121 weeks; first tumour after 89 weeks). No such tumour occurred in 40 control animals, or in historical controls of this strain (Pott *et al.*, 1987).

A group of 22 female, 100-day-old Osborne-Mendel rats received five weekly intratracheal instillations of 2 mg glasswool (geometric mean fibre length, 4.7 μm; geometric mean diameter, 0.4 μm; 19% of fibres > 10 μm in length and 0.2—0.6 μm in diameter) in 0.2 ml saline. A group of 25 rats was injected with saline only, and another group of 125 animals was untreated. All animals were observed for life; the median average life span was longer in treated rats (783 days) than in the saline (688 days) or untreated (724 days) controls. No respiratory-tract tumour was observed in any group. Of 25 rats treated similarly with UICC crocidolite (5% fibres ⩽5 μm in length; mean, 3.1 ± 10.2 μm), two developed broncho-alveolar tumours (Smith *et al.*, 1987). [The Working Group noted the relatively small number of animals used and the low tumour response in positive controls, which made interpretation of the study difficult.]

Hamster: Two groups of 136 or 138 male Syrian golden hamsters [age unspecified] were examined after eight weekly intratracheal instillations in 0.15 ml saline of 1 mg of two different glass fibre samples prepared from US glass wool (JM 104) by wet milling in a ball mill for 2 or 4 h, respectively, resulting in different length distributions (2-h sample: length, 50% <7.0 yum; diameter, 50% <0.3 μm; 4-h sample: length, 50% <4.2 μm; diameter, 50% <0.3 μm). Two control groups received eight intratracheal instillations of 1 mg of either UICC crocidolite (length, 50% >2.1 μm; diameter, 50% >0.2 μm)asa positive control, or granular titanium dioxide as a negative control. The incidences of thoracic tumours were: 48/136 2-h glass fibre-treated animals (five lung carcinomas, 37 mesotheliomas, six sarcomas), 38/138 4-h glass fibre-treated animals (six lung carcinomas, 26 mesotheliomas, six sarcomas), 18/142 crocidolite-treated animals (nine lung carcinomas, eight mesotheliomas, one sarcoma) and 2/135 titanium dioxide-treated controls (two sarcomas); lung carcinomas were described as mucoepidermoid carcinomas. The total duration of the experiment was 113 weeks. Nearly all tumour-bearing animals survived up to 18 months after the first instillation, and about 50% lived for longer than two years (Pott *et al.*, 1984a). [The Working Group noted the unusually long life span of the hamsters in this study.]

Six groups of 35 male and 35 female Syrian golden hamsters, 16 weeks of age, received intratracheal instillations in 0.2 ml 0.005% gelatin in saline of 1 mg US glasswool (JM 104; 58% <5μm in length; 88%<1.0μm in diameter), 1 mg glasswool plus 1 mg benzo[*a*]pyrene, 1 mg crocidolite (UICC standard reference sample; 58% >5 μm in length; 63% >0.25 μm in diameter), 1 mg crocidolite plus 1 mg benzo[*a*]pyrene, 1 mg benzo[*a*]pyrene in gelatin solution in saline or vehicle alone, respectively, once every two weeks for 52 weeks. The experiment was terminated at 85 weeks, at which time 53, 43, 43, 50, 48 and 46 animals were still alive in the six groups, respectively. Tumours of the respiratory tract were found only in hamsters treated with benzo[*a*]pyrene: in the 63 animals examined in the group given benzo[*a*]pyrene alone, two carcinomas and one sarcoma were observed plus four papillomas; in 52 hamsters receiving crocidolite plus benzo[*a*]pyrene, two carcinomas and one sarcoma plus one papilloma were observed; and two sarcomas (3%) plus two papillomas were found in 66 animals treated with glasswool plus benzo[*a*]pyrene (Feron *et al.*, 1985). [The Working Group noted the relatively short observation time and the absence of tumours in the positive, crocidolite-treated control group.]

(c) Intrapleural administration

Mouse: Four groups of 25 BALB/c mice [sex and age unspecified] received single intrapleural injections of 10 mg of one of four different samples of borosilicate glass fibres in 0.5 ml distilled water. The injection material was obtained by separating each of two original samples with average diameters of 0.05 μm and 3.5 μm into two samples with lengths of several hundred micrometers and lengths of <20 μm. Animals were killed at intervals of two weeks to 18 months, at which time there were 37 survivors. No pleural tumour was found in any of the treated animals, whereas two mesotheliomas were observed in a total of 150 mice given intrapleural injections of chrysotile or crocidolite [dose not stated] in a parallel experiment. The author concluded that the pleural cavity of mice might be very resistant to tumour production by any type of mineral fibre (Davis, 1976). [The Working Group noted the small number of animals used, the relatively short observation time and the low response in positive controls.]

Rat: Groups of 32-36 SPF Wistar rats (twice as many males as females), 13 weeks of age, received single intrapleural injections in 0.4 ml saline of 20 mg fibreglass (a borosilicate; 30% of fibres 1.5-2.5 μm in diameter; maximum diameter, 7 μm; 60%, >20 μm in length), 20 mg glass powder (a borosilicate; projected area diameter, <8 μm) or 20 mg of one of two different samples of Canadian SFA chrysotile. Animals were held until natural death; average survival times were 774, 751, 568 and 639 days for the groups treated with fibreglass, glass powder and the two chrysotile samples, respectively. No injection-site tumour was observed in the fibreglass-treated group; a single mesothelioma occurred in the glass powder-treated group (after 516 days). Tumour incidences in the two chrysotile groups were 23/36 and 21/32; death of the first animals with tumours occurred after 325 and 382 days (Wagner *et al.*, 1973).

Three groups of 16 male and 16 female Wistar rats, ten weeks of age, received single intrapleural injections of 20 mg of a finer US glasswool (JM 100; 99% of fibres <0.5 μm in diameter; median diameter, 0.12 μm; 2%, >20 μm in length; median length, 1.7 μm) or a coarser US glasswool (JM 110; 17% of fibres <1 μm in diameter; median diameter, 1.8 μm; 10%, >50 μm in length; median length, 22 μm) in 0.4 ml saline or saline alone. Animals were held until natural death; mean survival times were 716, 718 and 697 days, respectively. Between 663 and 744 days after inoculation, 4/32 animals given the finer fibreglass had mesotheliomas. No pleural tumour occurred in animals treated with the coarser fibreglass or in saline controls (Wagner *et al.*, 1976).

Groups of 32—45 male SPF Sprague-Dawley rats, three months old, received single intrapleural injections of 20 mg US glasswool (JM 104; mean length, 5.89 μm; mean diameter, 0.229 μm), 20 mg UICC chrysotile A (mean length, 3.21 μm; mean diameter, 0.063 μm), 20 mg UICC crocidolite (mean length, 3.14 μm; mean diameter, 0.148 μm) in 2 ml saline, or saline alone. Animals were held until natural death; mean survival times for total groups (and for animals with tumours) were 513 (499), 388 (383), 452 (470) and 469 days, respectively. Six thoracic mesotheliomas developed in a total of 45 rats injected with glasswool. The incidences of thoracic tumours in chrysotile- and crocidolite-treated animals were 15/33 (one carcinoma and 14 mesotheliomas) and 21/39 (mesotheliomas), respectively. No such tumour occurred in the 32 control animals (Monchaux *et al.*, 1981).

Groups of 48 SPF Sprague-Dawley rats [sex and age unspecified] received single intrapleural injections of 20 mg fibrous glass dusts or chrysotile in 0.5 ml saline. The dust samples used (and the size distributions of those fibres longer than 1 μm) were: English glasswool with resin coating (70% fibres ⩽5 μm in

length; 85% ⩽1 μm in diameter), English glasswool after removal of resin (57% ⩽5 μm in length; 85% ⩽1 μm in diameter), US glasswool (JM 100; 88% ⩽5 μm in length; 98.5% ⩽1 μm in diameter) and UICC African chrysotile [fibre sizes unspecified]. The animals were kept until natural death [survival times unspecified]. One mesothelioma occurred in the group treated with English glasswool [whether coated or uncoated unspecified], four in the group treated with US glasswool and six in the chrysotile-treated group. No such tumour was observed in a group of 24 saline-treated controls (Wagner *et al.*, 1984).

Groups of 30—130 female Osborne-Mendel rats, 12—20 weeks old, received a single intrathoracic implantation of one of 72 different types of natural and man-made mineral fibres, 19 of which were uncoated or resin-coated fibrous glass. The materials were mixed in 10% gelatin, and 40 mg of each type of glass in 1.5 ml gelatin were smeared on a coarse fibrous glass pledget which was implanted into the left thoracic cavity. The rats were observed for 24 months after treatment and were compared with untreated controls and controls implanted with the pledget alone. The incidences of pleural mesothelioma in animals surviving more than 52 weeks varied from 0/28 to 20/29 depending on fibre size. The most carcinogenic fibres were those <1.5 μm in diameter and >8 μm in length (Table 39). When two of the fibrous glass preparations (diameter, >0.25 μm) were leached to remove all elements except silicon dioxide, they induced incidences of 2/28 and 4/25 pleural mesotheliomas (Stanton *et al.*, 1977, 1981).

(d) Intraperitoneal administration

Rat: Groups of female Wistar rats, eight to 12 weeks of age, received single intraperitoneal injections of 2 or 10 mg or four weekly injections of 25 mg German glasswool (106; 59% fibres <3 μm in length), different doses of UICC chrysotile A or 100 mg of one of seven kinds of granular dust in 2 ml saline. The animals were held until natural death. In the groups given glasswool or chrysotile, dose-dependent incidences of mesotheliomas and sarcomas were observed: 1/34, 4/36 and 23/32 in the groups receiving 2, 10and 100 mg glass fibres, respectively, with corresponding average survival times of 518, 514 and 301 days; incidences ranged from 6/37 (2 mg) to 25/31 (25 mg) in the chrysotile-treated groups, with average survival times of 468—407 days. Of 263 animals treated with granular dusts, three rats developed malignant tumours. No abdominal tumour occurred in 72 saline-treated control animals (Pott *et al.*, 1976).

Groups of female Wistar rats, eight to 12 weeks of age, received single intraperitoneal injections of 2, 10 or 50 mg (the latter given in two doses) of one German glasswool (104; mean fibre length, approximately 10 μm; diameter, approximately 0.2 μm), 20 mg of another German glasswool (112; mean length, approximately 30 *μm;* diameter, approximately 1 μm), 2 mg UICC crocidolite or 50 mg corundum. Average survival times were 673, 611 and 361 days for the groups treated with the finer glasswool (104) and 610 and 682 days for the groups treated with the coarser glasswool (112) or crocidolite. Dose-related increases in the incidences of abdominal tumours (mesotheliomas, sarcomas and, rarely, carcinomas) were observed in the groups treated with the finer glasswool: 20/73 (2 mg), 41/77 (10 mg) and 55/77 (50 mg). The incidences in the groups treated with the coarser glasswool or with crocidolite were 14/37 and 15/39, respectively. Of the 37 rats that received injections of granular corundum, three had tumours in the abdominal cavity; mean survival was 746 days (Pott *et al.*, 1976).

Three groups of 44 female Wistar rats, four weeks old, were examined after intraperitoneal injections of 2 or 10 mg US glasswool (JM 104; milled for 2 h [size not given]) or 2 mg of another US glasswool (JM 100; 50% fibres <2.4 μm in length; 50% <0.33 μm in diameter). Abdominal tumours were observed in 14/44 rats that received 2 mg JM 104 glasswool, in 29/44 rats that received 10 mg JM 104 glasswool and in 2/44 rats that received 2 mg JM 100 glasswool. The first tumour-bearing rat was found 350 days (50 weeks), 252 days (36 weeks) and 664 days (95 weeks) after the start of treatment in the three groups, respectively. In three positive control groups that received intraperitoneal injections of 0.4,2 or 10 mg UICC chrysotile B, tumours developed in 9, 26 and 35 of 44 rats, respectively; the first tumour-bearing rat was found 522 days (75 weeks), 300 days (43 weeks) and 255 days (36 weeks) after start of treatment in the three groups, respectively. A negative control group treated with 2 mg granular corundum dust had a tumour incidence of 1/45; the first tumour-bearing animal was found 297 days (42 weeks) after injection. The tumours observed in both the test and control groups were mesotheliomas or sarcomas. The groups treated with 0.4 mg chrysotile B or with JM 100 glasswool had an infection during the 21st month which might have reduced the tumour incidence. The high tumour incidence in rats treated with JM 104 glasswool was suggested by the authors to be due to the longer fibre length, and the low incidence in rats treated with JM 100 glasswool to the large proportion of shorter fibres (Pott *et al.*, 1984b).

Groups of female Sprague Dawley rats, eight weeks old, received single intraperitoneal injections of 2 mg or 10 mg US glasswool (JM 100; median fibre length, 2.4 μm; median fibre diameter, 0.33 μm) in 2 ml saline. Median survival times were 90 and 79 weeks for the groups receiving 2 mg and 10 mg glasswool, respectively. Sarcomas, mesotheliomas and (rarely) carcinomas occurred in 21/54 low-dose and in 24/53 high-dose animals (first tumour after 53 weeks in each group). Three tumours were found in two groups of 54 rats that received two injections each of either 20 mg Mount St Helen's volcanic ash or 20 ml saline alone (median survival, 93 and 94 weeks; first tumour after 79 and 94 weeks, respectively) (Pott *et al.*, 1987).

Groups of 32 female Wistar rats, five weeks old, received single intraperitoneal injections of 0.5 or 2.0 mg US glasswool (JM 104; median length, 3.2 μm; median diameter, 0.18 *μm)*, 2.0 mg of glasswool treated with 1.4 M hydrochloric acid for 24 h, or 0.5 or 2.0 mg South African crocidolite (median length, 2.1 μm; median diameter, 0.20 μm) in 1 ml saline or saline alone. A group of 32 animals that received three intraperitoneal injections of titanium dioxide (total dose, 10 mg) served as another control. The animals were observed for life; median survival times were 116, 110, 107, 109, 71, 130 and 120 weeks for rats receiving 0.5 mg and 2.0 mg glasswool, acid-treated glasswool, 0.5 and 2.0 mg crocidolite, titanium dioxide and saline only, respectively. The incidences of sarcomas, mesotheliomas and (rarely) carcinomas of the abdominal cavity observed with the glasswool were 5/30 (first tumour after 88 weeks) with 0.5 mg, 8/31 (first tumour after 84 weeks) with 2.0 mg and 16/32 (first tumour after 56 weeks) with acid-treated glasswool. Tumour incidences of 18/ 32 (first tumour after 79 weeks) and 28/32 (first tumour after 52 weeks) occurred in the crocidolite groups, and two tumours (first tumour after 113 weeks) were seen in the saline-control group. No such tumour was found in the group treated with titanium dioxide (Muhle *et al.*, 1987; Pott *et al.*, 1987).

Groups of eight-week-old female Sprague-Dawley rats were injected once with 5 mg of US glasswool (JM 104) cut and ground for 1 h in an agate mill or treated with 1.4 M hydrochloric acid or sodium hydroxide for 2 or 24 h, and administered in 2 ml saline. The loss in weight 2 and 24 h after treatment with acid amounted to 25 and 33%; that after treatment with alkali, 1.7 and 6.8%; and that after treatment with distilled water, 1.7%. A negative control group received 5 mg granular titanium dioxide. The glasswool treated for 2 h with acid induced abdominal tumours (mesotheliomas, sarcomas and, rarely, carcinomas) in 32/54 rats; median survival time of the group was 88 weeks, and average survival time of the tumour-bearing animals was 93 weeks. Glasswool treated for 24 h with acid (fibre length: 50% <5.3 μm; fibre diameter: 50% <0.5 μm) induced tumours in 4/54 rats; median survival time of the group was 99 weeks, and average survival time of the tumour-bearing animals was 111 weeks. Glasswool treated for 2 h with alkali induced tumours in 42/54 rats; median survival time of the group was 71 weeks, and average survival time of the tumour-bearing rats was 69 weeks. Glasswool treated for 24 h with alkali (fibre length: 50% <5.4 μm; fibre diameter: 50% <0.5 μm) induced abdominal tumours in 46/53 rats; median survival time of the group, and average survival time of the tumour-bearing rats was 72 weeks. In the group administered untreated fibres (fibre length: 50%<4.8 μm; fibre diameter: 50%<0.29 μm), 44/54 rats developed abdominal

tumours; median survival time of the group was 64 weeks, and average survival time of the tumour-bearing animals was 67 weeks. In the group treated with titanium dioxide, 2/52 rats were found to have abdominal tumours; median survival time of the group was 99 weeks, and average survival time of the tumour-bearing animals was 97 weeks (Pott *et al.*, 1987).

In another experiment, groups of four-week-old Wistar rats [sex unspecified] received 5 mg of the same glasswool, either untreated or treated for 24 h with acid or alkali, by intraperitoneal injection in 0.8 ml saline. A negative control group received 5 mg granular titanium dioxide. The acid-treated glasswool (fibre length: 50% <5.3 yum; fibre diameter: 50% <0.5 µm*)* induced abdominal tumours (mesotheliomas, sarcomas and, rarely, carcinomas) in 2/45 rats; median survival time of the group was 113 weeks, and average survival time of the tumour-bearing rats was 126 weeks. The alkali-treated glasswool (fibre length: 50% <5.4 µm; fibre diameter: 50% <0.5 µm) led to the formation of tumours in 27/46 rats; median survival time of the group was 58 weeks, and average survival time of the tumour-bearing rats was 64 weeks. Untreated glasswool (fibre length: 50% <4.8 µm; fibre diameter: 50%<0.29 µm) induced abdominal tumours in 20/45 rats; median survival time of the group was 34 weeks, and average survival time of the tumour-bearing rats was 49 weeks. None of 47 rats treated with titanium dioxide developed abdominal tumours; median survival time of the group was 102 weeks (Pott *et al.*, 1987).

A group of 25 female, 100-day-old Osborne-Mendel rats received a single intraperitoneal injection of 25 mg glasswool (geometric mean fibre length, 4.7 µm; geometric mean diameter, 0.4 µm*;* 19% of fibres >10 µm in length and 0.2—0.6 µm in diameter) in 0.5 ml saline. A group of 25 rats was injected with saline only, and another group of 125 was untreated. All animals were observed for life; the median average life span was significantly shorter in treated rats (593 days) than in saline (744 days) or untreated (724 days) controls. Mesotheliomas were found in 8/25 of the glasswool-treated rats and in 20/25 rats injected with 25 mg UICC crocidolite (5% ⩽ 5 µm in length; mean, 3.1 ± 10.2 µm) but in neither control group (Smith *et al.*, 1987).

Hamster. Groups of 40 female Syrian golden hamsters, eight to 12 weeks old, received single intraperitoneal injections of 2 or 10 mg German glasswool (59% of fibres shorter than 3 µm) or UICC chrysotile A in 1 ml saline. Animals were observed for life. No tumour of the abdominal cavity was found (Pott *et al.*, 1976). [The Working Group noted that survival times were not reported and that saline controls were not used.]

Glass filament

Intraperitoneal administration

Rat: Groups of 50 female Wistar rats, 12 weeks of age, received 10 or 40 mg of two German glass filaments — a finer filament (ES 5; median diameter, 5.5 µm; 80% of fibres 4.8—6.3 µm in diameter; median length, 39 µm; 10% of fibres longer than 80 µm) and a coarser one (ES 7; median diameter, 7.4 µm; 80% of fibres, 6.8—8.1 µm in diameter; median length, 46 µm; 10% of fibres longer than 102 µm) — or a granular glass dust [unspecified] by single or double (weekly) intraperitoneal injection in 2 ml saline. Animals were observed for life; median survival times were 111, 107,121 and 119 weeks for the groups given 10 mg finer glass filament, 40 mg finer glass filament, 40 mg coarser glass filament and 40 mg granular glass dust, respectively. Corresponding mean survival times of animals with tumours were 106, 119, 126 and 129 weeks, respectively. No statistically significant increase in the incidences of sarcomas, mesotheliomas or (rarely) carcinomas of the abdominal cavity was observed in the groups treated with finer glass filament (low dose: 2/ 50; death of first animal with tumour after 92 weeks; high dose: 5/46; first tumour after 96 weeks) or with coarser glass filament (1/47; first tumour after 126 weeks), when compared with an incidence of 2/ 45 (first tumour after 121 weeks) in the group treated with granular glass dust (Pott *et al.*, 1987).

Similar groups of female Wistar rats, 12—15 weeks old, received 50 or 250 mg of a very fine German glass filament (ES 3; median diameter, 3.7 µm; 80% of fibres 3.3—4.2 µm in diameter; median fibre length, 16.5 µm; 10% of fibres longer than 50 jum), the finer glass filament (ES 5) described above or granular glass dust by laparatomy in 4 ml saline. Median survival time of the group given 250 mg finer glass filament was 109 weeks; the life span of the other groups was reduced by an infection in month 15: median survival times were 94, 94, 88, 99 and 87 weeks for the groups receiving 50 mg and 250 mg very fine glass filaments, 50 mg and 250 mg granular glass dust and a control group receiving 4 ml saline alone, respectively. Abdominal tumours occurred in 2/28 animals given the finer glass filament (death of first animal with tumour after 76 weeks), in 3 / 48 given the low dose of the very fine filament (first tumour after 71 weeks) and in 4/46 given the high dose of the very fine filament (first tumour after 87 weeks). Similar numbers of abdominal tumours occurred in the control groups: 4/48 with both the low and high doses of granular glass and 2/45 with saline alone; the first tumours were detected after 62, 91 and 95 weeks, respectively (Pott *et al.*, 1987).

[The Working Group noted that the number of fibres injected was much smaller in these studies than in those with glasswool (<0.3 µm diameter) carried out in the same laboratory.]

Rockwool and slagwool

(a) Inhalation

Rat: Groups of 24 male and 24 female Wistar IOPS AF/ Han rats, eight to nine weeks old, were exposed by inhalation to dust concentrations of 5 mg/m^3 (respirable particles) French resin-free rockwool [type of rock unspecified] (40% of fibres < 10 µm in length, 23% <1 yum in diameter) or a Canadian chrysotile fibre (6% respirable fibres >5 µm in length) for 5 h per day on five days per week for 12 or 24 months. An unspecified number of rats was killed either immediately after treatment or after different periods of observation (for seven, 12 and 16 months after exposure for animals exposed for 12 months; four months after exposure for those exposed for 24 months). No pulmonary tumour was observed among 47 rats treated with rockwool or in 47 untreated controls; nine pulmonary tumours occurred among 47 rats treated with chrysotile (Le Bouffant *et al.*, 1984). [The Working Group noted that, because of the lack of survival data, the exact incidences of tumours could not be ascertained.]

Groups of 48 SPF Fischer rats [sex and age unspecified] were exposed by inhalation to dust concentrations of approximately 10 mg/m^3 resin-free rockwool [type of rock unspecified] or UICC Canadian chrysotile for 7 h per day on five days per week for 12 months. The size distribution of those airborne fibres longer than 5 µm was: 71% of rockwool fibres ⩽20 µm in length, 58% ⩽1 µm in diameter; 16% of chrysotile fibres ⩾20 µm in length, 29% ⩾0.5 µm in diameter. Six rats were removed from each group at the end of exposure to study dust retention, and a similar number of animals was sacrificed one year later for the same purpose. The remainder were held until natural death [survival times not reported]. During the period 500—1000 days after the start of exposure, lung adenomas (one with some malignant features) occurred in 2/48 rats in the rockwool-treated group; 11 adenocarcinomas and one adenoma (with some malignant features) occurred in 48 rats treated with chrysotile. No lung tumour was observed in a group of 48 untreated controls (Wagner *et al.*, 1984). [The Working Group noted that, because of inadequate data on survival, the exact tumour incidences could not be established.]

A group of 55 female, 100-day-old Osborne-Mendel rats was exposed by inhalation (nose only) to slagwool dust [type of slag unspecified] (mass concentration, 7.8 mg/m^3; 15.2% respirable — geometric mean diameter, 0.9 µm; geometric mean length, 22 µm; chamber concentration, 200 fibres/cm^3 with 76 fibres >10 µm in length and ⩽1.0 µm in diameter) for 6 h per day on five days per week for two years and then observed for life. Groups of 59 chamber and 125 room controls were available. No respiratory-tract tumour was observed in any group. Average survival in the slagwool-treated group was shorter (677 days) than that of chamber (754 days) and room (724 days) controls. Of 57 rats exposed to UICC crocidolite (3000 fibres/cm^3; 5% fibres ⩾ 5 µm in length; mean, 3.1 ± 10.2 µm), two developed bronchoalveolar tumours and one, a mesothelioma (Smith *et al.*, 1987).

Hamster. A group of 69 male, 100-day-old Syrian golden hamsters was exposed by inhalation (nose only) to slagwool dust [type of slag unspecified] (mass concentration, 7.8 mg/m^3; 15.2% respirable — geometric mean diameter, 0.9 µm; geometric mean length, 22 µm; chamber concentration, 200 fibres/cm^3 with 76 fibres/cm^3 >10 µm in length and ⩽1.0 µm in diameter) for 6 h per day on five days per week for two years and then observed for life. Groups of 58 chamber and 112 room controls were available. No respiratory-tract tumour was observed in the treated animals or in room controls; one of 58 chamber controls had a bronchoalveolar tumour. There was no decrease in life span (about 660 days). Of 58 hamsters exposed to UICC crocidolite asbestos (3000 fibres/cm^3; 5% fibres ⩽5 µm in length: mean, 3.1 ± 10.2 µm), no pulmonary tumour occurred (Smith *et al.*, 1987).

(b) Intrapleural administration

Rat: Groups of 48 SPF Sprague-Dawley rats [sex and age unspecified] received single intrapleural injections of 20 mg fibrous dusts of various wools or chrysotile in 0.5 ml saline. The dust samples used (and the size distributions of those fibres longer than 5 µm) were: Swedish rockwool[type of rock unspecified] with resin coating (70% fibres <5 µm in length; 52%<0.6 µm in diameter), Swedish rockwool after removal of resin (70% <5 µm in length; 58% <0.6 µm in diameter), German slagwool [type of slag unspecified] (67% <5 µm in length; 42% <0.6 µm in diameter), German slagwool after removal of resin (80% <5 µm in length; 62% <0.6 µm in diameter) and UICC African chrysotile [fibre sizes unspecified]. The animals were kept until natural death [survival times unspecified]. Three mesotheliomas occurred in the group treated with rockwool with resin and two in the group treated with rockwool without resin; six mesotheliomas occurred in the chrysotile-treated group. No tumour was observed in the group treated with slagwool or in a group of 24 saline-treated controls (Wagner *et al.*, 1984).

In the experiment by Stanton *et al.* (1977, 1981) (see pp. 93–94), one sample of slagwool (a silica-slag-derived mineral) was implanted in the pleura. A pleural sarcoma developed in 1/25 animals that survived longer than 52 weeks.

(c) Intraperitoneal administration

Rat: Groups of female Wistar rats, 15 weeks old, received 40 mg of two samples of German slagwool [type of slag unspecified] by two weekly intraperitoneal injections in 2 ml saline. The coarser sample (RH) had a median fibre length of 26 µm and a median fibre diameter of 2.6 yum; the finer one (ZI) had a median fibre length of 14 yum and a median fibre diameter of 1.5 µm. The animals were observed for life; median survival times were 111, 107 and 101 weeks for the groups given coarser and finer slagwool and for a control group given saline alone, respectively. Slight increases in the incidences of sarcomas, mesotheliomas and (rarely) carcinomas of the abdominal cavity were observed with the slagwool samples: 6/99 with the coarser sample (first tumour after 88 weeks) and 2/96 with the finer one (first tumour after 67 weeks). No tumour occurred in 48 control animals (Pott *et al.*, 1987). [The Working Group noted that in other studies in this laboratory the historical incidence of abdominal tumours in saline-treated controls ranged from 0 to 6.3%.]

Preliminary results after 28 months of observation were available from another experiment carried out on female Wistar rats, eight weeks of age: groups of about 50 animals received five intraperitoneal injections of a German rockwool (from basalt; total dose, 75 mg; median length, 20 yum; median diameter, 1.8 yum) or 100 mg titanium dioxide in 2 ml saline. Median survival times were 79, 109 and 111 weeks for the rockwool group, the titanium dioxide group and a control group receiving five injections of 2 ml saline alone, respectively. In the group that received the rockwool, tumours of the abdominal cavity developed in 32/53 animals, the first tumour occurring 54 weeks after first injection. Tumour incidences in the control groups were 5/53 with titanium dioxide (life span of first animal with tumour, 38 weeks) and 2/102 with saline (first tumour after 93 weeks). In two positive-control groups, single intraperitoneal injections of 0.25 mg actinolite fibres and of 1 mg chrysotile produced tumours in 20/36 and 31/36 rats, respectively (Pott *et al.*, 1987). [The Working Group noted that most of the diagnoses had not been verified by histo-pathological examination at the time of reporting.]

Groups of female Sprague-Dawley rats, eight weeks old, received intraperitoneal injections of 75 mg Swedish rockwool [type of rock unspecified] (administered in three injections; median fibre length, 23 µm; diameter, 1.9 µm), 10 mg of a fine fraction prepared from the rockwool sample (single injection; median fibre length, 4.1 yum; diameter, 0.64 µm) or 40 mg granular volcanic ash from Mount St Helen's (two injections) in 2 ml saline. Median survivals were 77, 97 and 93 weeks for the animals given the two forms of rockwool and volcanic ash, respectively; the median life span of a control group that received two injections of 2 ml saline was 94 weeks. A high incidence of tumours of the abdominal cavity was observed with 75 mg of the original rockwool sample: 45/63 (life span of first animals with tumour, 39 weeks); a slightly increased tumour incidence occurred with 10 mg of the fine fraction: 6/45 (first tumour after 88 weeks). This compared to a tumour incidence of 3/54 in the volcanic ash group and in the control group (Pott *et al.*, 1987).

Ceramic fibres

(a) Inhalation

Rat: Groups of 45—46 young adult male Sprague-Dawley rats were exposed by inhalation to fibres >5 µm in length, at concentrations of 2.9×10^6/1 [2900 fibres/cm^3] potassium octatitanate (Fybex; 19.1% fibres <3 µm diameter), 2.0×10^6/1 pigmentary potassium titanate (PKT; 45.8% fibres <3 µm diameter) or 3.1×10^6/1 [3100 fibres/cm^3] UICC amosite asbestos for 6 h per day on five days per week for three months, and were then observed for 21 months. One group of 46 unexposed animals served as controls. Groups of four to ten animals per exposure group were killed at 20 days, 50 days, 90 days, six months, 12 months and 18 months, and the remainder at 24 months. No pulmonary tumour was observed in animals that were killed or died prior to the end of the study. Bronchoalveolar tumours were observed in 1 /14 animals treated with potassium octatitanate (one adenoma), 0/19 animals treated with pigmentary potassium titanate, 3/11 animals treated with amosite (two adenomas, one carcinoma) and 0/13 controls killed at the end of the study (Lee *et al.*, 1981). [The Working Group noted the short exposure period and the small number of animals available for evaluation.]

Three groups of about 40 'young' rats [strain, sex and age unspecified] were exposed by inhalation to dust clouds of fibres consisting chemically of >95% alumina with 3-4% silica (Saffil®; median fibre diameter, 3.3 µm), thermally 'aged' fibres (treated at temperatures >1000°C) or UICC chrysotile A for 18

months. The concentration of total dust from the untreated fibres was 20—120 mg/m^3, resulting in a cumulative exposure of approximately 7000 mg × h/ m^3 respirable dust for the untreated and aged forms (respirable fraction, 2.5% on average); cumulative exposure to chrysotile was 13 800 mg × h/m^3 respirable dust. The animals were held to 85% mortality. No pulmonary tumour was found in animals exposed to the ceramic fibres or in 34 undusted controls; 9/39 animals exposed to chrysotile had lung tumours (Pigott & Ishmael, 1982). [The Working Group noted that survival times were not reported and that only a small proportion of the dust cloud was respirable.]

A group of 48 SPF Wistar AF/ Han rats [sex unspecified], 12 weeks of age, was exposed by inhalation to concentrations of 10 mg/m^3 respirable dust from fibrous ceramic aluminium silicate glass ([source unspecified] approximately 90% of fibres <3 µm in length and <0.3 µm in diameter; particles with aspect ratio >3:1) for 7 h per day on five days per week for 12 months (cumulative exposure, 224 days). Four animals were removed from the experiment at 12 months and four at 18 months; seven surviving animals in treated and control groups were sacrificed at the end of the experiment at 32 months; the remainder were allowed to live out their life span. Seven of the 48 treated animals developed malignant pulmonary neoplasms, and one had a benign adenoma. No pulmonary tumour was observed in 39 untreated controls, but two malignant tumours of the peritoneum or digestive system were observed (Davis *et al.*, 1984).

A group of 55 female, 100-day-old Osborne-Mendel rats was exposed by inhalation (nose only) to refractory ceramic fibre dust [source unspecified] at a mass concentration of 10.8 mg/m^3, of which 35% was respirable (geometric mean diameter, 0.9 µm; geometric mean length, 25 µm; chamber concentration, 200 fibres/cm^3 with 88 fibres/cm^3 >10 µm in length and ⩽1.0 µm in diameter) for 6 h per day on five days per week for two years and then observed for life. Groups of 59 chamber and 125 room controls were available. No respiratory-tract tumour was observed in any group. Exposure to refractory ceramic fibres did not affect survival. Of 57 rats exposed to UICC crocidolite (3000 fibres/cm^3; 5%>5 µm in length; mean, 3.1 ± 10.2 µm), three developed one mesothelioma and two broncho-alveolar tumours (Smith *et al.*, 1987).

Hamster: Groups of 34 hamsters [sex and age unspecified] were exposed by inhalation to fibres >5 µm in length, at concentrations of 2.9 × 10^6/1 [2900 fibres/cm^3] potassium octatitanate (Fybex®; 19.1% fibres with diameter <3 µm), 2.0 × 10^6/1 [2000 fibres/cm^3] pigmentary potassium titanate (PKT; 45.8% fibres with diameter <3 µm) or 3.1 × 10^6/1 [3100 fibres/cm^3] UICC amosite asbestos, for 6 h per day on five days per week for three months and were then observed for 21 months. One group of 34 unexposed animals served as controls. Groups of four to 12 animals per exposure group were killed at 50 days, 90 days, six months, 12 months and 18 months, and the remainder at 24 months. One of four animals exposed to potassium octatitanate and killed at 18 months had a pleural mesothelioma. No other pulmonary tumour was observed in any of the groups (Lee *et al.*, 1981). [The Working Group noted the short exposure period and the small number of animals available for evaluation.]

A group of 70 male, 100-day-old Syrian golden hamsters was exposed by inhalation (nose only) to refractory ceramic fibre dust [source unspecified] at a mass concentration of 10.8 mg/m^3, of which 35% was respirable (geometric mean diameter, 0.9 µm; geometric mean length, 25 µm; chamber concentration, 200 fibres/cm^3 with 88 fibres/cm^3 >10 µm in length and ⩽1.0 µm in diameter) for 6 h per day on five days per week for two years and then observed for life. Groups of 58 chamber and 112 room controls were available. One treated hamster developed a spindle-cell mesothelioma on the posterior left lung; one of 58 chamber controls had a bronchoalveolar tumour. There was no decrease in life span. Among 58 hamsters exposed to UICC crocidolite asbestos (3000 fibres/cm^3; 5% ⩾ 5 µm in length; mean, 3.1 ± 10.2 µm), no pulmonary tumour occurred (Smith *et al.*, 1987).

Guinea-pig: Groups of 35 male albino guinea-pigs [age unspecified] were exposed by inhalation to fibres >5 µm in length, at concentrations of 2.9 × 10^6/1 [2900 fibres/cm^3] potassium octatitanate (Fybex®; 19.1% fibres with diameter <3 µm*)*, 2.0 × 10^6/1 [2000 fibres/cm^3] pigmentary potassium titanate (PKT; 45.8% with diameter <3 µm) or 3.1 × 10^6/1 [3100 fibres/cm^3] UICC amosite asbestos, for 6 h per day on five days per week for three months and were then observed for 21 months. One group of 31 unexposed animals served as controls. Groups of one to ten animals per exposure group were killed at 50 days, 90 days, six months, 12 months and 18 months, and the remainder at 24 months. No pulmonary tumour was observed in any of the groups (Lee *et al.*, 1981). [The Working Group noted the short exposure period and the small number of animals available for evaluation.]

(b) Intratracheal instillation

Rat: A group of 22 female, 100-day-old Osborne-Mendel rats received five weekly intratracheal instillations of 2 mg refractory ceramic fibres ([source unspecified] geometric mean fibre length, 25 µm; geometric mean diameter, 0.9 µm; 83% of fibres >10 µm in length and 86% <2.0 µm in diameter) in 0.2 ml saline. A group of 25 rats was injected with saline only, and another group of 125 animals was untreated. All animals were observed for life; the median average life span was approximately the same in treated rats (698 days) and in saline (688 days) and untreated (724 days) controls. No respiratory-tract tumour was observed in any group. Of 25 rats treated similarly with UICC crocidolite (5% fibres ⩽ 5 µm in length; mean, 3.1 ± 10.2 *µm)*, two developed bronchoalveolar tumours (Smith *et al.*, 1987). [The Working Group noted the small number of animals per group and the low tumour response in positive controls, which made interpretation of the study difficult.]

Hamster. A group of 25 male, 100-day-old Syrian golden hamsters received five weekly intratracheal instillations of 2 mg refractory ceramic fibres ([source unspecified] geometric mean fibre length, 25 µm; geometric mean diameter, 0.9 µm; 83% of fibres >10 µm in length and 86% <2.0 µm in diameter) in 0.2 ml saline. A group of 24 hamsters was injected with saline only, and another group of 112 animals was untreated. All animals were observed for life; the median average life span was significantly shorter in the treated hamsters (446 days) than in the saline (567 days) or untreated (563 days) controls. No respiratory-tract tumour was observed in any group. Of 27 hamsters treated similarly with UICC crocidolite (5% fibres ⩽ 5 µm in length; mean, 3.1 ± 10.2 µm), 20 developed bronchoalveolar tumours (13 benign, seven malignant) (Smith *et al.*, 1987).

(c) Intrapleural administration

Rat: Groups of 31-36 SPF Wistar rats (twice as many males as females), 13 weeks of age, received a single intrapleural injection in 0.4 ml saline of 20 mg ceramic aluminium silicate fibres ([source unspecified] 0.5—1 µm in diameter), nonfibrous aluminium oxide (<10 µm projected area diameter) or one of two different samples of Canadian SFA chrysotile. Animals were held until natural death; average survival times were 736, 710, 568 and 639 days for the groups treated with ceramic fibres, aluminium oxide and the two chrysotile samples, respectively. Of the 31 ceramic fibre-treated animals, mesotheliomas developed in three, the first of which died 743 days after injection. One mesothelioma was observed in the aluminium oxide-treated group (after 646 days). Tumour incidences in the chrysotile groups were 23/36 and 21/32; death of the first animals with tumours occurred after 325 and 382 days (Wagner *et al.*, 1973).

Groups of 30—50 female Osborne-Mendel rats, 12—20 weeks old, received a single intrathoracic implantation of one of 13 different types of ceramic fibres [source unspecified]. The materials were mixed in 10% gelatin, and 40 mg of each type of ceramic fibre in 1.5 ml gelatin were smeared on a coarse fibrous

glass pledget which was implanted into the left thoracic cavity. The rats were observed for 24 months after treatment and were compared with untreated controls and controls implanted with the pledget alone. The incidences of pleural sarcomas varied, depending on the number of fibres ⩾ 0.25 µm diameter and >8 µm length (Table 40) (Stanton *et al.*, 1981).

Groups of 24 male and 24 female rats [strain and age unspecified] received single intrapleural injections of 20 mg fibres consisting chemically of >95% alumina with 3-4% silica (Saffil®; median fibre diameter, 3.3 µm), thermally 'aged' fibres (treated at temperatures > 1000°C) or UICC chrysotile A in saline. The animals were held until natural deaths. No mesothelioma occurred in animals treated with either form of ceramic fibre or in 48 saline controls; 7/48 rats treated with chrysotile had mesotheliomas (Pigott & Ishmael, 1982). [The Working Group noted that survival data were not given.]

(d) Intraperitoneal administration

Rat: A group of 32 Wistar AF/ Han rats [age and sex unspecified] received a single intraperitoneal injection of 25 mg fibrous ceramic aluminium silicate glass ([source unspecified] approximately 90% of fibres <3 µm in length and <0.3 µm in diameter) suspended in 2 ml Dulbecco's phosphate buffered saline. Peritoneal tumours developed in three animals (9%), the first tumour occurring approximately 850 days after injection [total length of observation and survival times not reported]. One of the tumours was a typical mesothelioma, and the histology of the others was similar to that of fibrosarcoma. In a group of 39 untreated controls used for a study by inhalation (see pp. 101—102), two malignant tumours (5%) of the peritoneum or digestive system were observed (Davis *et al.*, 1984).

Groups of about 50 female Wistar rats, eight weeks of age, received five intraperitoneal injections of ceramic wool (Fiberfrax®; total dose, 45 mg; median fibre length, 8.3 µm; diameter, 0.91 µm), a US ceramic wool (MAN; total dose, 75 mg; median fibre length, 6.9 (µm; diameter, 1.1 µm) or titanium dioxide (total dose, 100 mg) in 2 ml saline. Preliminary results were reported describing tumour incidences 28 months after first injection. Tumours of the abdominal cavity were found in 32/47 animals (median survival time, 51 weeks; life span of first animal with tumour, 30 weeks) treated with the first ceramic wool and in 12/54 animals (median survival, 91 weeks; first tumour after 60 weeks) treated with the US ceramic wool. Of 53 animals receiving titanium dioxide, five developed tumours (median survival, 109 weeks; first tumour after 38 weeks); and two tumours occurred in a total of 102 rats that received saline alone (median survival, 111 weeks; first tumour after 93 weeks). In two positive-control groups, single intraperitoneal injections of 0.25 mg actinolite fibres and of 1 mg chrysotile produced tumours in 20/36 and 31/36 rats, respectively (Pott *et al.*, 1987). [The Working Group noted that most of the diagnoses had not been verified by histopathological examination at the time of reporting.]

A group of 25 female, 100-day-old Osborne-Mendel rats received a single intraperitoneal injection of 25 mg refractory ceramic fibres ([source unspecified] geometric mean fibre length, 25 µm; geometric mean diameter, 0.9 µm; 83% of fibres >10 µm in length and 86% <2.0 µm in diameter) in 0.5 ml saline. A group of 25 rats was injected with saline only, and another group of 125 was untreated. All animals were observed for life; the median average life span was significantly shorter in treated rats (480 days) than in saline (744 days) or untreated (724 days) controls. Mesotheliomas were found in 19/23 of the refractory ceramic fibre-injected rats; no tumour was observed in either control group (Smith *et al.*, 1987).

Hamster. Groups of 15 and 21 male, 100-day-old Syrian golden hamsters received a single intraperitoneal injection of 25 mg refractory ceramic fibres ([source unspecified] geometric mean fibre length, 25 µm; geometric mean diameter, 0.9 µm; 83% of fibres >10 µm in length and 86% <2.0 µm in diameter) in 0.5 ml saline. A group of 25 hamsters was injected with saline only, and another group of 112 was untreated. All animals were observed until natural death; median average life span was significantly shorter in the two groups of treated hamsters (462 and 489 days) than in saline (560 days) or untreated (503 days) controls. Mesotheliomas were found in 2/15 and 5/21 hamsters treated with ceramic fibre; no tumour was observed in either control group (Smith *et al.*, 1987)

3.2. Other relevant data

(a) Experimental systems

(i) Deposition, retention and clearance

A number of mechanisms result in the deposition of inhaled particles, both fibrous and nonfibrous, in the respiratory tract (Lippmann *et al.*, 1980). Deposition in the nasopharyngeal region occurs mainly by inertial impaction due to the high velocity and abrupt changes in direction of the airstream. Deposition in the tracheobronchial region is determined by inertial impaction and by gravitational settling. A disproportionate amount of deposition in this region of both nonfibrous (Lippmann & Schlesinger, 1984) and fibrous (Morgan *et al.*, 1975) particles occurs at airway bifurcations. On the basis of studies on humans, the estimated deposition of monodisperse particles in the pulmonary region peaks for mouth-breathing subjects at an aerodynamic equivalent diameter of ~3 µm and for nose-breathing subjects at ~ 2.5 µm (Lippmann *et al.*, 1980). [The aerodynamic equivalent diameter of a particle is the diameter of a spherical particle of unit density which has the same falling speed.] Particles of this size are deposited mainly by sedimentation, but, for submicron particles, deposition by diffusion prevails. Other mechanisms are also important for fibrous materials: interception is important when the length of fibres becomes a significant fraction of the airway diameter; however, when fine, straight fibres are inhaled they tend to align themselves along the axes of airways due to the aerodynamic forces acting upon them so that they can penetrate effectively to the pulmonary region (Lippmann *et al.*, 1980). The electrostatic enhancement of lung deposition of fibrous aerosols was reviewed by Vincent (1985), who suggested that it is important for polydisperse fine fibres.

Rats were exposed by nose-only inhalation for 30 min to glass microfibres and to UICC standard reference samples of asbestos, and deposition was measured using a radioactive tracer technique. The amount of fibre respired was calculated from the aerosol concentration, exposure time and minute volume (Hammad *et al.*, 1982). For glass microfibre and anthophyllite, which had activity median aerodynamic diameters of 2.3 and 2.0 µm, respectively, measured with the Cascade Centripeter, ~70% of the respired glass fibre was deposited throughout the respiratory tract, compared with less than half of the chrysotile and of the finer amphibole fibres (activity median aerodynamic diameters, 1.2 — 1.5 µm). [The activity mean aerodynamic diameter of an aerosol is determined from the distribution of radioactivity on the stages of a size-classifying sample previously calibrated with spherical particles of unit density. If the radioactivity is homogeneously distributed within the material, which is likely to be the case in the experiments described above, the activity mean aerodynamic diameter and mass median aerodynamic diameter will be identical.] Deposition in the alveolar region was relatively unaffected by activity median aerodynamic diameter and averaged about 11% (Morgan *et al.*, 1977). In later studies by the same workers, rats were exposed to sized glass fibres with nominal diameters of 1.5 and 3 µm and lengths ranging from 5 to 60 µm. A similar radioactive tracer technique was used. All of the respired longer (⩽ 30 µm), 1.5-µm Diameter fibre was deposited, mainly in the upper respiratory tract; the same applied to thick fibres (diameter, 3 µm) ⩽10 µm in length. Deposition of these materials in the

alveolar region was negligible and, in rats, appeared to peak at an aerodynamic diameter of ~2 μm, which is less than that in humans (Morgan *et al.*, 1980).

Rats were exposed by nose-only inhalation for six days to unsized man-made mineral fibres. The fibres had a count median diameter of ~1 μm and a count median length of ~ 10 μm. They were recovered from lungs using a low-temperature ashing technique, and the fibre content of lung tissue was compared, for different size categories, with the estimated number of fibres respired. The retention of fibres with diameters <0.5 μm reached a peak of 8% at a fibre length of 21 μm; the retention of fibres with diameters >0.5 μm was <1 % for all fibre lengths. A correlation of retention with calculated aerodynamic equivalent diameter confirmed that fibres with an aerodynamic equivalent diameter of >3.5 μm were not found in the lung (i.e., were not respirable) (Hammad *et al.*, 1982). These results, combined with those obtained using sized man-made mineral fibres, indicate that deposition in the alveolar region of rat lung must fall rapidly from a maximum at an aerodynamic equivalent diameter of 2 μm to effectively zero at about 3.5 μm.

Rats were exposed chronically to 'microfibre' glasswool (JM 100) and to thicker glass-and rockwool fibres at a concentration of 10 mg/ m^3 on five days per week for periods of up to one year. The count median diameter of the glass microfibre (<0.5 μm) was less than that of either the rockwool (0.5—1 μm) or of the thicker glasswool (~1 μm). After one year's exposure, the weights in the lung were 4.45 mg microfibre, 0.94 mg thicker glasswool and 3.11 mg rockwool, indicating that the microfibre was more respirable. No fibre longer than 30 μm was found in the lungs, although they were present in the airborne dust cloud (Wagner *et al.*, 1984). In a similar study, Le Bouffant *et al.* (1987) exposed rats to the same microfibre (JM 100) and to aerosols of different samples of thicker glass- and rockwool for periods of up to two years. In this study also, larger quantities of microfibre than of the thicker glasswool or the rockwool were found in the lungs. [The Working Group noted that, in the case of chronic inhalation exposure to fibres, it is difficult to derive accurate data on deposition, as clearance takes place simultaneously.]

After exposure of rats and hamsters by inhalation to monodisperse particles, the deposited material is not distributed evenly between the lung lobes: the apical region of the right lung receives a higher relative concentration, and the diaphragmatic regions receive less (Raabe *et al.*, 1977). Similar observations have been made for glass fibres (Morgan *et al.*, 1980) and for ceramic fibres; the disproportion of fibres between lobes increased with aerodynamic diameter (Rowhani & Hammad, 1984).

The physical clearance of particles deposited in the alveolar region of the lung is thought to be mediated by pulmonary alveolar macrophages (Morgan, A. *et al.*, 1982). These phagocytic cells are found both in the interstitium and free in the alveolar spaces. Count median diameters of rat pulmonary alveolar macrophages range from 11 to 12 μm (Sykes *et al.*, 1983a). Fibres that can be encompassed in their entirety by pulmonary alveolar macrophages can be mobilized and transported to the terminal bronchioles, from where they are cleared from the lung by mucociliary action. Fibres that are too long to be engulfed by a single cell may remain at the site of deposition or penetrate into the interstitium (Morgan, 1979). Similar size considerations apply to the clearance of fibres through lymph nodes associated with the lung (Le Bouffant *et al.*, 1987).

Sized glass fibres (0.5 and 1 mg) were administered to rats by intratracheal instillation. Animals were killed serially, the lungs digested with sodium hypochlorite (Morgan & Holmes, 1984a) and the number of fibres determined by optical microscopy. Of the 5 × 1.5 μm (diameter) fibres and the 10 × 1.5 μm fibres present in the lung immediately after instillation, only 10% and 20%, respectively, remained in the lung after one year. With 30 × 1.5 μm fibres and 60 × 1.5 μm fibres, there was no evidence of clearance over the same period, suggesting that the critical length of fibres for removal from the lung is between 10 and 30 μm (Morgan, A. *et al.*, 1982). In studies with some of the same sized glass fibres, a radioactive tracer method (^{65}Zn; half-life, 245 days) was used to quantify the clearance of 5 × 1.5 μm and 60 × 1.5 μm fibres from the lung of rats. In contrast to the results of the previous study, it was reported that there was relatively rapid clearance of both types of fibre (half-life, one month) and that the clearance curves did not differ significantly (Bernstein *et al.*, 1980). [The Working Group noted that observations that are based on the removal of a radioactive constituent of the fibre from the lung do not enable physical clearance to be distinguished from dissolution and may give misleading results.]

In the same study, only short glass fibres were found in regional lymph nodes 18 months after intratracheal instillation (Bernstein *et al.*, 1984). At various times after exposure of rats by inhalation to glass microfibres (JM 100) and to thicker glass- and rockwool fibres, much greater numbers of the thin microfibres than of either glass- or rockwool were transported to the tracheobronchial lymph nodes. With all of these materials, the fibres in the lymph nodes were shorter than those in the lungs, with very few fibres > 10 μm in length (Le Bouffant *et al.*, 1987).

Following injection of glasswool (JM 104; count median length, 6 μm; count median diameter, 0.23 μm) or asbestos into the pleural cavity of rats, translocation of glass fibre (in terms of number concentration) to the mediastinal lymph nodes was less than that of the asbestiform minerals; however, in mass terms, they were equivalent. Less than 1% of the injected fibres was transported to the lung, but, as ascertained by transmission electron microscopy, the mean length of fibres recovered from lung increased with time. Fibres at concentrations of 10^6— 10^7 fibres/g of tissue were detected in a range of organs, including lung, spleen, kidney, liver and brain; in the intrathoracic lymph nodes, the concentration was ten- to 100-fold higher. These figures suggest that migration occurred *via* the bloodstream (Monchaux *et al.*, 1982).

Solubility in vivo: The solubility of man-made mineral fibres and asbestos fibres, both *in vivo* and *in vitro*, has been reviewed (Morgan & Holmes, 1986).

In a study of lung clearance using sized glass fibres, Morgan, A. *et al.* (1982) noted that short (⩽10 μm) fibres dissolved quite slowly and uniformly in rat lung. Fibres of ⩽30 μm in length dissolved much more rapidly and less uniformly; after 18 months, some had become so thin that they had fragmented, while the diameters of others were relatively unchanged. These observed variations in solubility were attributed to differences in physiological pH; for example, the intracellular pH of pulmonary alveolar macrophages is lower than that of the general lung environment (Laman *et al.*, 1981). Following administration of the same fibres to rats, the fibres in lung sections were characterized using scanning electron microscopy. It was noted that long fibres that had been engulfed by pulmonary alveolar macrophages had dissolved more extensively than those lying free in the alveolar spaces (Bernstein *et al.*, 1984). In both of these investigations, it was noted that the ends of long glass fibres dissolved more rapidly than the middle. In a later, analogous study, dimensional changes of sized rockwool fibres (count median length, 27 μm; count median diameter, 1.1 μm*)* in rat lung were characterized following their administration by intratracheal instillation. After 18 months, there was no change in the median diameter at the middle of the fibres, but it was observed qualitatively that fibres were becoming thinner at their ends. The authors concluded that the rockwool sample tested was much less soluble *in vivo* than the glasswool tested previously (Morgan & Holmes, 1984b).

Rats were exposed by inhalation to 'microfibre' glasswool (JM 100), to a thicker glasswool and to rockwool fibres for one or two years. At the end of the dusting period, gravimetric measurements showed that much greater quantities of the microfibre had been retained; however, after a further 16 months without dusting, the concentration of glass microfibres in the lung had been reduced to an extent similar to that of the thicker glasswool and rockwool fibres, indicating

either a more rapid clearance or more rapid dissolution. Scanning electron microscopy of glass- and rockwool fibres isolated from the lung by low-temperature ashing showed that their surfaces were eroded; examination under the analytical electron microscope revealed that certain constituents of these fibres (mainly sodium and calcium) had been lost (Le Bouffant *et al.*, 1987). Glass microfibres removed from rat lung following chronic exposure and examined by transmission electron microscopy appeared to be more susceptible to surface etching (irregularities in their outlines, loss of electron density, appearance of pits along their edges) than either thicker glasswool or rockwool fibres (Johnson *et al.*, 1984).

The durability of some man-made mineral fibres, including various glasswool, rockwool and ceramic fibres, was studied in rat lung over a period of two years following intratracheal instillation. Both the number of fibres and the size distribution of fibres remaining in the lung were determined by transmission electron microscopy. Count median diameters ranged from 0.1—0.2 µm for glass microfibres to 1.8 µm for rockwool. In all cases, fibres <5 µm in length were removed from the lung more rapidly than longer fibres; however, there was a wide variation in the clearance rates of the latter. Acid-treated JM 104 E glass microfibre was cleared very rapidly, apparently by dissolution; untreated microfibre (JM 104/Tempstran 475) was scarcely cleared at all, but some leaching of sodium and calcium was detected. Of the other fibres, the ceramic fibre had the longest residence time (half-life, 780 days for fibres >5 µm in length), compared with 280 days for rockwool fibres >5 µm in length and for thicker glasswool. The authors concluded that fibres with a high calcium content dissolve most rapidly *in vivo* and that calcium content is a more important determinant of solubility than sodium or potassium content (Bellmann *et al.*, 1987).

Solubility in vitro: A number of studies have been made of the solubility of man-made mineral fibres *in vitro*, using both static and continuous-flow systems. [The Working Group noted that the latter approximates more closely to the situation *in vivo*.] The dissolution of specific constituents has been quantified by analysis of the leachate (Forster, 1984; Klingholz & Steinkopf, 1984).

Man-made mineral fibres were quite stable in water at 37°C, but their solubilities increased in simulated extracellular fluid: with Gamble's solution, fibres dissolved more rapidly in continuous-flow than in static systems; it was reported in one study that slagwool dissolved more rapidly than glasswool, which dissolved more rapidly than rockwool (Klingholz & Steinkopf, 1984). [The Working Group noted that only single samples of each type of fibre were tested.] With glass fibres, the square root of the weight of individual undissolved fibres decreased linearly with leaching time, and glass composition appeared to be a major determinant of the rate of dissolution (Leineweber, 1984), as also appeared to be the case *in vivo* (Bellmann *et al.*, 1987). In a study of dissolution in physiological media, precipitation of alkali earth carbonates occurred at higher than physiological temperatures (60°C). Rates of dissolution of 10 ng/cm^2 per hour or higher were measured at 37°C, indicating that fine fibres (diameter, <1 µm) could dissolve completely after one year in a continuous-flow system (Forster, 1984). The surface layers of leached fibres were converted to colloidal shells (Forster, 1984; Klingholz & Steinkopf, 1984).

The dissolution of silica from a range of industrial man-made mineral fibres (including glasswool, rockwool, slagwool and ceramic fibres) was compared *in vitro* with that of natural amphibole fibres, using a solution with a similar composition to Gamble's. The man-made mineral fibres showed a variety of calculated dissolution velocities, ranging from 0.2 to 3.5 nm/ day. The corresponding value for natural amphibole fibre was <0.01 nm/ day. Dissolution velocities for glass fibres showed a 15-fold variation: the samples of rockwool and slagwool had intermediate solubility among the fibres tested, and the solubility of the ceramic fibres was generally at the lower end of the range (Scholze & Conradt, 1987). Leineweber (1984) also found great variability in the solubility of glass fibres; one ceramic fibre was found to be highly insoluble.

[The Working Group noted that it is important to attempt to predict in-vivo solubility when estimating the possible biological effects of man-made mineral fibres; however, it is difficult to reproduce *in vitro* the varying conditions of pH and concentrations of complexing agents which fibres encounter in the intra- and extracellular environments of the lung. Furthermore, no overall generalization regarding the absolute or relative solubilities of the main families of such fibres can be made on the basis of the results of the studies reported. For example, while most samples of glasswool studied have proved to be relatively soluble and ceramic fibres relatively insoluble, there have been exceptions — at least one sample of glass fibre was extremely durable and one sample of ceramic fibres relatively soluble (Leineweber, 1984).]

(ii) Toxic effects

The toxic effects of man-made mineral fibres *in vivo* and *in vitro* have been reviewed (Hill, 1978; Konzen, 1980; Davis, 1986).

Toxicity in vivo: All 20 hamsters that received an intratracheal instillation of 7 mg of glass microfibre (median diameter, 0.1 µm) died within 30 days; the lungs were haemorrhagic and oedematous. In contrast, only 3/20 animals instilled with a thicker microfibre (median diameter, 0.2 µm) died during this period, and no animal died in groups injected with three types of glass fibre used for insulation purposes (median diameters, 2.3—4.1 µm) (Pickrell *et al.*, 1983). Similar acute deaths were reported in rats following intratracheal instillation of 3—70 mg of very finely-ground (particulate) ceramic fibre (mean size, 0.04 µm), but not following instillation of another preparation of the same material containing coarser dusts (mean size, 0.7—0.8 µm) (Gross *et al.*, 1956). Acute deaths from haemorrhagic peritonitis also occurred in two groups of hamsters (21/36 and 15/36) that received intraperitoneal injections of 25 mg of refractory ceramic fibre (median diameter, 1.8 µm) (Smith *et al.*, 1987).

No toxic effect was found in cats that had inhaled finely-ground rockwool dust (average particle size, 2.2 µm) for two months (total dust levels, 50—900 mg/m^3) (Fairhall *et al.*, 1935).

Decreases in haemoglobin levels and erythrocyte counts, coupled with an increase in reticulocyte count, were reported in rats that had inhaled lead silicate glass fibres at a dose level of 100 mg/m^3 for 4.5 months (Azova *et al.*, 1971).

Rubbing of the shaved skin of guinea-pigs with a tampon of glasswool produced erythema and, rarely, punctiform haemorrhages. Glass fibres were found embedded only in the epithelial layers of the skin (Pellerat & Condert, 1946).

Pulmonary inflammation: Glass fibre (1 mg; nominal diameter, 1.5 µm) was administered to rats by intratracheal instillation. One or seven days after instillation, the number of neutrophil leucocytes in the cell population (obtained by bronchoalveolar lavage) was increased by at least ten-fold over that in controls administered saline. Levels of lactate dehydrogenase in the lavage fluid were raised at seven days, although not at one day, following instillation (Sykes *et al.*, 1983b).

Rats were exposed by inhalation to US glasswool (JM 102; diameter, 0.1 —0.6 µm) for six months; cell populations (obtained by bronchoalveolar lavage) contained 5—10% of lymphocytes and many multinucleate giant cells (10—20% of the cell population). In culture, more macrophages from the treated animals formed erythrocyte aggregating rosettes than did control macrophages (Miller, 1980).

Interaction with cells: Short, thin man-made mineral fibres deposited in lung tissue are rapidly phagocytosed by macrophages (Davis *et al.*, 1970). Long fibres cannot be engulfed completely by single macrophages and so protrude from them (Miller, 1980). Complete engulfment of long fibres is accomplished by the formation of multinucleated giant cells (Schepers, 1955; Sethi *et al.*, 1975; Miller, 1980).

Deposition of various man-made mineral fibres in lung tissues produces ferruginous bodies, most of which appear to form in relation to giant cells (Davis *et al.*, 1970; Botham & Holt, 1971). Factors such as fibre length and thickness which predispose fibres to become coated have been discussed by Morgan and Holmes (1985).

After exposure of guinea-pigs by inhalation to glass fibres, a very thin coating was detected (using Perls' stain) within 48 h on some of the glass fibres inside macrophages. After one month, the typical golden-yellow coating could be seen on fibres by phase-contrast microscopy; it was continuous initially and became segmented with time. By 18 months, the fibres had a beaded appearance and were fragmenting. In hamsters exposed by intratracheal administration to sized glass fibres (3 µm in diameter), partially coated fibres were detected after one month with 60- and 100-µm-long fibres and after two months with 10- and 30-µm — a similar time scale to that observed by Botham and Holt (1971) in guinea-pigs. Fibres <10 µm in length did not become coated (Holmes *et al.*, 1983). The first signs of coating of rockwool fibres in hamsters were detected after about two months; coating occurred only on fibres <2 µm in diameter and was often discontinuous on the longer fibres and did not appear to inhibit their dissolution (Morgan & Holmes, 1984b).

It is likely that all the man-made mineral fibres considered produce ferruginous bodies (Davis *et al.*, 1970).

Alveolar lipoproteinosis: Rats and hamsters exposed for 90 days to 400 mg/ m^3 of mainly short glass fibres (<2 µm) developed areas in the lung where alveoli were filled with granular material (lipoproteinosis), which regressed during a one-year period following termination of dusting. Guinea-pigs treated similarly developed very little alveolar lipoproteinosis (Lee *et al.*, 1979). Rats and hamsters treated with glass fibre developed alveolar lipoproteinosis, but those treated with ceramic fibres (potassium octatitanate, pigmentary potassium titanate) or amosite asbestos did not (Lee & Reinhardt, 1984).

Rats exposed for one year to 10 mg/m^3 respirable ceramic fibre dust (90% fibres with diameter <3 *µm)* developed alveolar lipoproteinosis. While most of the mass of the ceramic fibre dust cloud consisted of relatively thick fibres (diameter, 2—3 µm), many extremely small nonfibrous particles of ceramic material were also present (Davis *et al.*, 1984).

Fibrosis: Rats and guinea-pigs exposed by inhalation to dust from glasswool and then to 'glass cotton' ([fibre length not given] fibre diameter, 3–6 µm) at a dose level of 4 mg/m^3 for two to four years developed no fibrosis; minor areas of focal atelectasis and proliferation of alveolar epithelial cells occurred close to the terminal bronchioles (Schepers, 1955; Schepers & Delahant, 1955).

Rats and hamsters were exposed by inhalation to 100 mg/ m^3 glass fibre (average length, 10 µm; average diameter, 0.5 µm) for 24 months; in most cases only a normal 'dust reaction' was seen in lung tissue; however, a few of the oldest rats showed some foci of 'septal collagenous fibrosis'(Gross *et al.*, 1970a).

Exposure of rats by inhalation to high doses (1200 mg/m^3) of 'microfibre' glasswool (JM 102; all fibres <1 µm in diameter) for eight weeks produced no pulmonary fibrosis during the subsequent four weeks; lung tissue showed only a 'dust reaction' (Hardy, 1979). Exposure of rats, hamsters and guinea-pigs to glass fibre dust (average diameter, 1.2 µm; only 15% with aspect ratio, >3:1; concentration of fibres >5 µm in length, approximately 700/ cm^3) at a dose level of 400 mg/ m^3 for 90 days produced no significant fibrosis during the subsequent two years (Lee *et al.*, 1979).

Rats were treated in two studies with dust clouds of 'microfibre' glasswool (JM 100; mean diameter, 0.2—0.5 µm) and, in one study, with a thicker glasswool (median diameter, 1—2 µm). During the subsequent 24 months, the animals developed a pulmonary reaction described as 'minimal interstitial cellular reaction to the dust with no evidence of fibrosis' (McConnell *et al.*, 1984; Wagner *et al.*, 1984).

Rats and hamsters were treated by nose-only inhalation to dust clouds of four types of glasswool with mean diameters ranging from 0.45—6.1 µm at dose levels of up to 3000 fibres/cm^3 for 24 months. Levels of pulmonary fibrosis were extremely low (Smith *et al.*, 1984, 1987).

'Microfibre' glasswool (JM 100; diameter, <1 µm)and one thicker sample of glasswool (diameter, 1—3 µm) were administered to rats by inhalation at dose levels of 5 mg/ m^3 for up to 24 months. 'Slight septal fibrosis' was reported with all three dusts, which tended to diminish after dusting had stopped (Le Bouffant *et al.*, 1987).

Baboons exposed to 7.5 mg/m^3 glasswool JM 102 and 104 (median diameter, 0.5—1.0 µm) for up to 30 months showed limited pulmonary fibrosis (Goldstein *et al.*, 1983, 1984).

Rockwool (Wagner *et al.*, 1984; Le Bouffant *et al.*, 1987), slagwool and ceramic fibre (Smith *et al.*, 1987) produced similar low levels of pulmonary fibrosis in rats and hamsters exposed by inhalation at doses of 0.5—10 mg/ m^3. In contrast, in another study, rats exposed by inhalation to ceramic fibre dust at a similar dose level (10 mg/ m^3) for one year developed significant levels of pulmonary interstitial fibrosis by the age of 2.5—3 years (Davis *et al.*, 1984).

Fibres of potassium octatitanate (a ceramic fibre, 3—15 µm in length) produced pulmonary fibrosis in guinea-pigs, hamsters and particularly rats following long-term inhalation at dose levels between 3000 and 40 000 fibres/ cm^3 for three months. By two years after exposure, the lesions were well collagenized but were less frequent and less developed than those produced by asbestos (Lee *et al.*, 1981; Lee & Reinhardt, 1984).

One year after intratracheal instillation of 50 mg glasswool dust (length, 20—50 /im) into guinea-pigs, focal areas of pneumonitis were reported but no pulmonary fibrosis (Vorwald *et al.*, 1951). Focal atelectasis but no fibrosis was also reported after administration of two samples of glasswool fibres (diameter, ⩽3 µm) to guinea-pigs by intratracheal instillation of three doses of 25 mg (Schepers & Delahant, 1955). These studies were later expanded to include rats, rabbits and monkeys (Schepers, 1976). Sometimes, a severe tissue reaction occurred in response to the injected dust, but this did not progress to lasting fibrosis.

Intratracheal instillation of 10.5 mg of fine glass fibre (average diameter, 1 µm) in rats and hamsters produced inflammatory lesions that were no longer present one year later (Gross *et al.*, 1970a,b). In contrast, intratracheal instillation of 50 mg of glass fibre dust (diameter, 3 µm; length, 5—8 µm) into rats produced a proliferation of fibroblasts in the pulmonary interstitium and a progressive fibrosis (Wenzel *et al.*, 1969).

Definite areas of pulmonary fibrosis were found in guinea-pigs that received intratracheal instillations (total dose, 12mg) of long glass fibres (50% or 92% longer than 10 µm) but not in those given short fibres (length, <5 µm or 93% < 10 µm; total dose, 25 mg) (Wright & Kuschner, 1977).

After intratracheal instillation of ceramic fibre to rats at a dose of 10.5 mg, dust deposits in the lung tissue were surrounded by inflammatory cells (Gross *et al.*,

1970b).

The effects of binders, coating agents and plastic dust on the pathogenicity of glass fibres has been examined by inhalation and by intratracheal instillation in several species. No effect on the fibrogenicity of the mineral fibres was observed (Schepers *et al.*, 1958; Schepers, 1959, 1961; Gross *et al.*, 1970a). However, glass fibres in combination with styrene were reported to cause more cuboidal metaplasia of the bronchiolar lining cells in mice than styrene alone (Morisset *et al.*, 1979).

Intraperitoneal injection of 10 mg glass fibre (mean diameter, 0.05—0.1 μm or 2.5—4 μm) and three samples of 'man-made insulation fibres' (median diameter, 4—10 μm) into mice caused cellular granulomata, which eventually became collagenized. The degree of cellular response and subsequent fibrosis depended on the fibre length of the dust preparations, finely ground material being much less effective than dust containing long fibres (Davis, 1972).

Toxicity in vitro: Treatment of guinea-pig alveolar and peritoneal macrophages with glass fibre (diameter, 0.25—1 μm; length, 1—20 μm) at a dose of 75 $\mu m/10^6$ cells increased cell membrane permeability, as determined by erythrosin staining of cells and liberation of lactic acid dehydrogenase, but did not affect overall cell metabolism, as measured by lactic acid production (Beck *et al.*, 1972; Beck & Bruch, 1974; Bruch, 1974; Beck, 1976 a, b).

Fine glass fibre preparations (nominal diameters, 0.05—0.1 μm) caused greater release of both lactic dehydrogenase and β-glucuronidase in mouse peritoneal macrophages *in vitro* than thicker samples (1.5—2.5 μm). When respirable fractions of each sample were tested, only that of the thicker glass fibre increased cytotoxicity over that induced by the bulk sample (Brown *et al.*, 1979a; Davies, 1980).

For each of three pairs of long and short glass fibre preparations, long-fibred dust at a dose level of 100 jug/10^6 cells produced more toxicity to rat and guinea-pig alveolar macrophages, as measured by release of lactic dehydrogenase and β-glucuronidase than short-fibred preparations; short fibres showed some toxicity if their diameter was small enough (Tilkes & Beck, 1983a). Long fibres ($\leqslant$ 4 μm) produced a greater release of both prostaglandins and β-glucuronidase in rat alveolar macrophages than did short fibres (<3 μm) (Forget *et al.*, 1986). Long glass fibre increased the membrane permeability of L-cells (fibroblasts), causing release of lactic dehydrogenase. This effect was absent when the fibre was finely ground (Beck *et al.*, 1971). Of four glass fibre preparations, an ultrafine preparation (mean diameter, 0.19 μm) caused a greater reduction in cell numbers and in cellular uptake of ^{3}H-thymidine by phagocytic ascites tumour cells than did three thicker specimens (mean diameters, 0.2—0.43 μm). The toxicity of the fibres increased with increasing length and dose (Tilkes & Beck, 1980, 1983b).

Potassium octatitanate fibres (ceramic fibres) caused marked release of both lactic dehydrogenase and β-glucuronidase in mouse peritoneal macrophages (Chamberlain *et al.*, 1979). The viability of P388D_1 cells (permanent line of macrophage-like cells) up to 48 h appeared to be unaffected by 50 μm ceramic fibre dust/ml solution containing 10^5 cells (Davis *et al.*, 1985; Wright *et al.*, 1986).

Very fine glass fibres (JM 100) were much more active than thicker fibres (JM 110) in reducing the cloning efficiency of Chinese hamster V79/4 cells and in increasing the mean cell diameter in A549 cells (transformed human type II pneumocytes). Glass powder (crushed bulk glass) showed little activity (Chamberlain & Brown, 1978). Respirable fractions of JM 100 fibres had similar activity to manually crushed material; the respirable fraction of JM 110 fibres showed activity approaching that of JM 100 preparations (Brown *et al.*, 1979a). Commercial samples of unspecified glasswool, rockwool and slagwool reduced the cloning efficiency of V79/4 cells but were much less active than crocidolite asbestos. Removal of the resin binder slightly increased their activity, and they increased the diameter of A549 cells under these conditions (Brown *et al.*, 1979b). A sample of potassium octatitanate fibres was very active in both the A549 and V79/4 assays (Chamberlain *et al.*, 1979).

In lung fibroblast cultures, glass fibre induced only a slight increase in collagen production, in contrast to chrysotile asbestos (Richards & Morris, 1973).

Long(unmilled) glass fibres (JM 100; diameter, 0.2 μm; length, 15 μm) were more toxic than short (milled) fibres (diameter, 0.2 μm; length, 2 μm) in a dye exclusion test and in a colony-forming assay with a permanent cell line of rat tracheal epithelial cells (Ririe *et al.*, 1985). Similar cultures of hamster tracheal epithelial cells showed greater production of ornithine decarboxylase when treated with long glass fibres (JM 100) than with glass particles (Marsh *et al.*, 1985).

Neither 'small' nor 'large' glass fibres (JM 100 and JM 110) inhibited blastoid transformation or β_2-microglobulin production in cultures of human peripheral blood lymphocytes. Similarly, natural killer cell activity and antibody-dependent cell-mediated cytotoxicity were unaffected. Chrysotile asbestos proved very active in these test systems (Nakatani, 1983).

(iii) Effects on reproduction and prenatal toxicity

No adequate data were available to the Working Group.

(iv) Genetic and related effects

After treatment of C3H 10T1/2 cells with potassium octatitanate (Fybex®; 0—250 μm/ml), no DNA damage was observed, as measured by sensitivity to S_1 nuclease, whereas crocidolite and erionite gave positive results when tested at a dose of 250 μm/ ml (Poole *et al.*, 1986).

Fine and coarse glass fibres (JM 100 and 110; mean particle lengths, 2.7 μm and 26.0 μm; mean particle diameters, 0.12 μm and 1.9 μm, respectively) did not induce mutation in *Escherichia coli* strains B/r, WP2, WP2 *uvrA* and WP2 *uvrA polA* or in *Salmonella typhimurium* strains TA1535 and TA1538, either in the presence or absence of rat liver microsomal enzymes (S9 mix). Both types of glass fibre were tested over a wide range of concentrations (1—5000 μm/plate) (Chamberlain & Tarmy, 1977).

Glasswool (JM 100 and 110) did not increase sister chromatid exchange in Chinese hamster ovary (CHO-K1) cells *in vitro* at doses of 0.001-0.05 mg/ml or in human fibroblasts or lymphoblastoid cells after treatment of the cells with 0.01 mg/ml (Casey, 1983).

Exposure of CHO-K1 cells to 0.01 mg/ml glass fibres (nominal diameters, 1.5-2.5 μm; >60% longer than 20 μm; Wagner *et al.*, 1973) for 48 h or five days did not increase the frequency of chromosomal aberrations or polyploid cells (Sincock & Seabright, 1975). [The Working Group noted that only one dose level was tested.]

In a preliminary study, an increase in chromosomal aberrations was observed in CHO-Kl cells after treatment with JM 100 glasswool (0.01 mg/ml), but not with the same dose of JM 110 (Sincock, 1977). This finding was confirmed in a later study in which JM 100 (0.01 mg/ml) induced chromosomal breaks and

rearrangements in CHO-K1 cells, while JM 110 glass fibres had no effect; some increase in polyploidy was observed with both fibres (Sincock *et al.*, 1982).

Statistically significant increases in numerical chromosomal changes (aneuploidy and tetraploidy) as well as in the number of binucleated and micronucleated cells were observed after treatment of Syrian hamster embryo cells with JM 100 glasswool (2 µm/ cm^2; diameter, 0.2—0,2 µm). A slight increase, which was not statistically significant, was also noted in the number of chromosomal aberrations. JM 110 glasswool (average diameter, 0.8 µm) was much less potent in inducing cytogenetic damage at the same dose level; a significant increase was observed only in the number of binucleated cells. Milling of JM 100 glasswool abolished its ability to induce cytogenetic effects (Oshimura *et al.*, 1984).

JM 110 glasswool (0.02 mg/ml; nominal diameter, 1.5-2.5 µm) was applied to Chinese hamster V79-4 cells as both total material and respirable fraction. Only the respirable fraction increased chromosomal breaks and fragments significantly (Brown *et al.*, 1979a).

No increase in chromosomal damage or polyploidy was observed in primary human fibroblasts or in human lymphoblastoid cells after exposure of the cells to 0.01 mg/ml JM 100 or JM 110 glasswool (Sincock *et al.*, 1982). [The Working Group noted that only one dose level was tested.] It was reported in an abstract that a slight increase in chromosomal breaks was noted in cultured human primary mesothelial cells after treatment with glass fibres (Linnainmaa *et al.*, 1986).

Both JM 100 and JM 110 glasswool induced a linear, dose-dependent increase in the frequency of transformed colonies of Syrian hamster embryo cells in culture (dose range, 0.1—10 µm/cm^2) after a single treatment of the cells. Thin glass fibres (JM 100; average diameter, 0.1-0.2 µm) were as active as asbestos. When compared on a per-weight basis, thick glass fibres (average diameter, 0.8 µm) were 20-fold less potent than thin fibres (average diameter, 0.13 µm) in inducing cell transformation. When the average length of thin glass fibres was reduced from 9.5 to 1.7 µm by milling, there was a ten-fold decrease in transforming activity; there was no activity when the average fibre length was reduced to 0.95 µm. The cytotoxicity of the glass fibre dusts was found to correlate with their transforming potency (Hesterberg & Barrett, 1984). As reported in an abstract, in similar studies in the same cell systems, JM 100 wool, but not JM 110, increased the frequency of cell transformation (Mikalsen *et al.*, 1987).

Ceramic fibres (potassium octatitanate, Fybex®) at 6.2 and 12.5 µm/ ml caused low levels of transformation of C3H 10T1/2 cells *in vitro* (Poole *et al.*, 1986).

(b) Humans

(i) Deposition, retention and clearance

There are no experimental data on the effects of fibre dimensions on the deposition of man-made mineral fibres in humans. Studies of the falling speeds of fibres indicated that they are probably respirable only if their actual diameter is <3.5 µm (Timbrell, 1965). This hypothesis was confirmed subsequently by an examination of fibres extracted from the lungs of Finnish anthophyllite miners. The mean value for the maximum diameter of fibres from various lung lobes was 3.4 µm (Timbrell, 1982). A rough approximation of the aerodynamic equivalent diameter of a fibre may be obtained by multiplying its *actual* diameter by 3 (Timbrell, 1965). However, the precise relationship depends upon fibre length, shape and density.

A number of studies of asbestos fibres in humans confirm that short fibres are cleared preferentially from the lung (e.g., Morgan & Holmes, 1982). From studies in mine workers, the critical length above which fibres cannot be cleared from the lung has been estimated to be ~ 17 µm (Timbrell, 1982). [The Working Group noted that this value falls within the range of 10-30 µm reported by Morgan, A. *et al.* (1982) on the basis of animal studies with sized glass fibres.]

(ii) Toxic effects

Lung: Epidemiological evidence for pulmonary effects in humans exposed to man-made mineral fibre has been reviewed (Saracci, 1985, 1986). Few consistent pulmonary effects have been noted in populations exposed industrially to glass and other man-made mineral fibres. No abnormality was observed in chest radiographs of 935 employees in a factory manufacturing glass fibre who had been exposed to dust for at least ten years (Wright, 1968). Chest radiographs of 2028 male workers in the glass fibre manufacturing industry, two-thirds of whom had been employed for more than ten years, showed eight cases of 'micronodular' opacities, one of pinpoint nodularity and 17 with questionable 'nodularity' (Nasr *et al.*, 1971). Among 232 glass fibre workers in the Federal Republic of Germany, nine were found to have small rounded opacities on chest radiographs in the category range of 0/1—1/1. In addition, 30 of these workers had irregular opacities of category 0/1—1/1 (Valentin *et al.*, 1977). In a population of 1028 workers from seven glass fibre and mineral wool plants (mean employment period, 18 years), 25 men had a profusion of small rounded opacities of grade 1/0 and six men had grade 1 /1. The occurrence of small rounded opacities was more frequent among smokers, and it was concluded that, although their presence may have been related to glass fibre exposure as well, it was unlikely that they represented pulmonary fibrosis (Weill *et al.*, 1983, 1984). Of a population of 340 workers (275 with over ten years' employment) in a plant manufacturing man-made mineral fibres, 11% showed small rounded opacities of category 1/0 or more. However, no relation was detected between the prevalence of these opacities and the duration and intensity of exposure to fibres (Hill *et al*, 1973, 1984).

Pathological and mineralogical studies of 20 glass fibre workers (employment period, 16-32 years) showed no pulmonary change that could be associated with dust exposure. In addition, the number of mineral fibres/ g of dried lung tissue was not higher than in a control population (Gross *et al.*, 1971).

In contrast, four of seven workers with prolonged exposure to glass fibre during manufacture showed parenchymal involvement of lung tissue, three had evidence of pulmonary fibrosis and one showed both (Tomasini *et al.*, 1986). Chiappino *et al.* (1981) reported that three workers who had been exposed for nine to 17 years to glass fibre during manufacture showed signs of respiratory distress. The only radiographic abnormality was slight pleural thickening in one case. The authors reported that haemorrhagic alveolitis was present, as determined by the presence of siderocytes in the sputum. [The Working Group noted that, while these cells may indicate pulmonary haemorrhage, they are also common following exposure to many dust types, when inhaled particles within macrophages become surrounded by haemosiderin pigment.]

Six workers exposed to glasswool and rockwool for periods of eight to 29 years showed no abnormality in vital capacity, pulmonary compliance or pulmonary diffusion capacity, whereas eight asbestos workers showed a marked restriction in dynamic lung function and reduced diffusing capacity (Bjure *et al.*, 1964). No evidence of small airways dysfunction or resting ventilatory impairment was found in six nonsmoking sheet-metal workers who had been exposed to glass fibre (Sixt *et al.*, 1983). In a group of British workers exposed to glass fibre, forced expiratory volume (FEV_1) and forced vital capacity (FVC) were lower than predicted; however, results in controls from the same town were equally low (Hill *et al.*, 1973, 1984). Twenty-one workers exposed to rockwool for an average of 18 years showed no abnormality in lung function compared to 43 controls during a series of detailed physiological tests (Malmberg *et al.*, 1984). In a group of over 150 workers exposed to rockwool, no difference in FVC, FEV, or maximum expiratory flow rate could be attributed to exposure to man-made mineral

fibres (Skuric & Stahuljak-Beritic, 1984).

Upper respiratory tract: Early reports indicated that heavy exposure to man-made mineral fibres caused irritation and inflammation of the nasopharyngeal region and of the upper respiratory tract (Champeix, 1945; Roche, 1947; Cirla, 1948; Mungo, 1960).

Rhinitis, sinusitis, pharyngitis and laryngitis were all found in a series of 66 cases exposed to fibrous glass reported over periods of only 1.5 years and one year (Milby & Wolf, 1969). Both Müller *et al* (1980) and Maggioni *et al.* (1980) reported that nasopharyngeal irritation was found more frequently in workers exposed to glass fibre than in controls.

While irritation of the nasopharyngeal region obviously predominates, increased frequency of bronchitis was reported among 135 000 construction workers, which appeared to be related directly to their levels of exposure to man-made mineral fibres (Engholm & von Schmalensee, 1982).

Skin: The development of skin irritation following occupational exposure to man-made mineral fibres has been reviewed. Irritation can be mild, disappearing in a few days, or more severe, when it can be follicular or papulopustular in character (Fisher, 1982). The occurrence of this condition was reported by Sulzberger and Baer (1942) and confirmed in numerous subsequent publications (Schwartz & Botvinick, 1943; Champeix, 1945; Pellerat & Condert, 1946; Pellerat, 1947; Cirla, 1948). While skin over large areas may be involved, paronychia and interdigital maceration have been observed (McKenna *et al.*, 1958).

Skin reactions induced by glass fibre are not confined to occupational exposures, but may result from contamination of clothing washed with fabrics manufactured from glass fibre (Peachey, 1967; Fisher & Warkentin, 1969; Lechner & Hartmann, 1979). There is also evidence that fibre contamination of the atmosphere of buildings insulated with glass fibre products can result in skin lesions (Farkas, 1983).

In contrast to pulmonary pathology, it has been demonstrated that thick fibres are the most harmful, very fine fibres causing no skin lesions at all. It has been suggested that fibres <4 µm in diameter do not cause a skin reaction (Heisel & Hunt, 1968; Possick *et al.*, 1970).

Eye: Corneal irritation has also been reported after occupational exposure to man-made mineral fibres (Longley & Jones, 1966).

(iii) Effects on reproduction and prenatal toxicity

No data were available to the Working Group.

(iv) Mutagenicity and chromosomal effects

No data were available to the Working Group.

3.3. Case reports and epidemiological studies of carcinogenicity to humans

Two major studies have comprehensively addressed the cancer experience of workers in glasswool, glass filament, rockwool and slagwool production.

Enterline and others (Enterline *et al.*, 1983; Enterline & Marsh, 1984) reported the findings of a follow-up (through 1977) mortality study of 16 730 white male workers at 17 plants in the USA on which partial reports had previously been issued (Enterline & Marsh, 1979, 1980). The 17 plants included 11 fibrous glass plants: six producing glasswool (dates of starting, 1946-1952), three producing glass filament (dates of starting, 1941—1951) and two producing both (dates of starting, 1938 and 1950) (Enterline *et al.*, 1984), with a total worker population of 14 884 men, and six plants producing rockwool and slagwool (dates of starting, 1929—1948), with a workforce of 1846 persons. The workers had been employed in production or maintenance for one year or more during the years 1945—1963, except for men working in two plants producing small-diameter fibres (<1.5 µm), for which the criterion of inclusion was six months' or more experience. Across these factories, overall mean worker exposures to respirable fibres (length, >5 µm; each worker over his working lifetime in the industry. Most average values were below 0.5 fibres/cm^3 (Esmen *et al.*, 1979a). The most recent follow-up (through 1982) of this cohort has been reported by Enterline *et al.* (1987).

Saracci *et al.* (1984a,b) reported the findings of a mortality and cancer registration follow-up study (through 1977) of 25 146 workers at 13 plants producing man-made mineral fibres in seven European countries (Denmark, Finland, the Federal Republic of Germany, Italy, Norway, Sweden and the UK). Results for some of the national components of this collaborative study have also been published separately (Bertazzi *et al.*, 1984; Claude & Frentzel-Beyme, 1984; Olsen & Jensen, 1984). Of the 13 factories, four produced glasswool (dates of starting, 1933—1943), seven produced rockwool and slagwool (dates of starting, 1937—1950) and two produced glass filament (dates of starting, 1946 and 1961) (Simonato *et al.*, 1986a). Workers ever employed at each plant (with at least one year of employment at one plant in the UK and in all three plants in Sweden) were included in the cohort. At 12 of the 13 factories, environmental surveys were made during the late 1970s(Ottery *et al.*, 1984); average respirable fibre concentrations of 0.02, 0.04 and 0.006 fibre/ cm^3 were found for the main occupational groups in the glasswool, rock-/ slagwool and continuous filament plants, respectively. [The arithmetic mean for combined individual exposures was 0.04 fibre/ cm^3 (excluding secondary process 2 in one plant), and that for the six rockwool plants was 0.1 fibre/cm^3 (0.07 fibre/cm^3 without secondary process 2).]

The results of an extended follow-up of the whole cohort have been reported (Simonato, 1986a,b, 1987), together with detailed presentations of each national component (Andersen & Langmark, 1986; Bertazzi *et al.*, 1986; Claude & Frentzel-Beyme, 1986; Gardner *et al.*, 1986; Olsen *et al.*, 1986; Teppo & Kojonen, 1986; Westerholm & Bolander, 1986). The cohort excluded office workers and production workers in a non-man-made mineral fibre area of one factory that had been included in the original study, reducing the number of workers to 21 967. Mortality and cancer incidence were followed up until 1981—1983 in the various countries, and expected deaths were calculated on the basis of age- and calendar year-specific reference rates. In addition, correction factors for regional or more local lung cancer mortality levels were incorporated (Simonato *et al.*, 1986a). [On the assumption that persons for whom death certificates could not be located had died of causes that were distributed in the same way as those for whom death certificates were located, a correction can be made by dividing the SMR by 0.983.] Exposure assessment was based on a historical environmental investigation carried out by the Institute of Occupational Medicine (Edinburgh, UK) and by the International Agency for Research on Cancer, in which detailed information on production processes, raw materials, dust-suppressing agents, contaminants in the workplace and ventilation systems was collected (Cherrie & Dodgson, 1986). Airborne fibre levels were 'estimated' to be highest when dust suppressants were not used and/ or batch processing was employed (called the 'early technological phase'), and lowest when oil and resin binders were in use with modern mechanized production methods (the 'late technological phase'); the remaining period was termed the 'intermediate technological phase'. This classification could not be applied to the continuous filament factories. In this extension of the study, follow-up was 95% complete (Simonato *et al.*, 1987).

Besides these two major studies, a number of other studies have been reported by investigators who examined the cancer experience of workers in specific types of fibre production.

(a) Glasswool

A number of studies have been reported covering workers involved in the production of glasswool and glass filament in US plants (Enterline & Henderson, 1975; Bayliss *et al.*, 1976a,b; Enterline *et al.*, 1983; Enterline & Marsh, 1984; Morgan *et al.*, 1984; Enterline *et al.*, 1987). The results given in the most recent report (Enterline *et al.*, 1987) largely cover the populations studied in the earlier reports, except for a number of plants described by Morgan *et al.* (1984) and a case-control report of Bayliss *et al.* (1976a,b) which covers lung cancer cases not included by Enterline *et al.* (1987).

Bayliss *et al.* (1976a,b) carried out a 'case-control within a cohort' study of glasswool plant workers to examine whether exposure to small-diameter fibres (1-3 μm) was associated with respiratory disease. Workers who died of respiratory cancer were matched sequentially for date of birth (plus or minus six months), race and sex with an alphabetized list of other workers at the plant. A total of 16 cases and 16 matched controls were studied. Cases and controls were classified according to whether they had worked in at least one of several 'pilot plants' that produced small-diameter fibres: four of the cases and none of the controls had worked in a pilot plant. [The Working Group noted that there has been reconsideration of the criteria for exposure in this study, and the reported findings are thus tentative.]

Morgan *et al.* (1984) reported the mortality experience of 4399 men who had worked for a minimum of ten years in fibrous glass production and who were employed at some time during 1968—1977 at one or more fibrous glass plants owned by a single US company. [One of the major plants investigated in this study was later examined by Enterline *et al.* (1987).] Deaths were followed up for the period 1968—1977. Only men in 'exposed' jobs were included in the study. For respiratory cancer, the standardized mortality ratio (SMR) was 136 (39 observed) in the total cohort and 177(11 observed) in the subcohort of men with 20 years' or more employment and with first exposure dating back 30 years or more. When mortality was examined by job category, the only findings of interest were SMRs for lung cancer of 181 (seven observed) in textile forming and 132 (20 observed) in wool forming and fabrication. [This is a revision of an earlier report (Morgan *et al.*, 1981) in which there were some problems (Morton, 1982; Morgan, R.W. *et al.*, 1982): glasswool production was not separated from glass filament production, and no environmental data were reported.]

Enterline *et al.* (1987) reported a study of eight US plants that produced glasswool (six produced glasswool only and two produced both glasswool and continuous filament). The cohort consisted of 11 380 white male workers with one year or more of experience in production or maintenance during the years 1945—1963, except for men working in two plants that produced small-diameter fibres, for which the criterion was six months' or more experience; 97% were traced, and death certificates were located for 97.5% of those who were believed to have died. In the analyses presented, expected deaths were based on both US and local county, white, male, age- and time-specific mortality rates. The authors concluded that the latter were the more relevant. On the assumption that persons for whom death certificates could not be located had died of causes that were distributed in the same way as those for whom death certificates were located, a correction can be made by dividing the SMR by 0.975. [The Working Group considered that it was, in general, appropriate to make this correction. However, doing so did not alter the category of the *p* value (>0.05 to <0.05, and <0.05 to <0.01) obtained from any test of the statistical significance of a raised SMR.] The SMRs for respiratory cancer for the period 1946—1982 were calculated to be (US) 116 and (local) 109, based on 267 deaths. For workers with fewer than 20 years since first exposure, the SMRs were (US) 95 and (local) 105 (60 observed), and those for workers with 20 years or more since first exposure, (US) 124 ($p < 0.01$ [95% confidence interval, 103—148]) and (local) 111 (207 observed). SMRs for respiratory cancer increased with time since first exposure, but the trend was less steep when local rates were used; there was no relationship with duration of exposure or with a time-weighted measure of exposure expressed as fibres/ cm^3-months. Fibre exposure levels for each of the eight glasswool plants were estimated to range from 0.005 fibre/ cm^3 in one plant to 0.293 fibre/ cm^3 in a plant that produced small-diameter fibres. The highest individual average fibre exposure level estimated for any member of this cohort was 1.5 fibres/cm^3.

A separate analysis was made by Enterline *et al.* (1987) of 7586 workers in four plants where small-diameter fibres (<3 μm in diameter) were produced. For 1015 workers ever exposed during the production of small-diameter fibres, the SMRs for respiratory cancer were (US) 133 and (local) 124 (22 observed), and for those never exposed, (US) 115 and (local) 105 (183 observed). SMRs were higher for workers exposed during the production of small-diameter fibres than for those not exposed in each of the three plants at which deaths were observed. Of the 22 deaths, eight occurred during the period 1946–1977 and 14 during the period 1978–1982. During the period 1978–1982, SMRs were (US) 264 ($p < 0.05$) and (local) 198. SMRs for respiratory cancer increased with time since first exposure to small-diameter fibres. Death certificates for the men indicated two mesotheliomas, one of which occurred in a plant that produced small-diameter fibres. Slides obtained for the other case (exposure unknown) were submitted to the US Mesothelioma Reference Panel of the UICC; no one on the panel considered that mesothelioma was a reasonable diagnosis.

Enterline *et al.* (1987) carried out a 'case-control within a cohort' study of these glasswool workers, adjusting for cigarette smoking. All white men in their cohort study of 11 glasswool and glass filament plants who had died of respiratory cancer between 1950 and 1982 were compared with a 4% stratified (by plant and year of birth), random sample of workers, selected from the cohort of glass workers who had reached the age of 43 prior to 1983. In total, 330 cases and 529 controls were initially selected. Smoking histories for 73% of cases and 73% of controls, and details as to age at which smoking had started and stopped for 64% of cases and 71 % of controls, were obtained by telephone interviews with the worker or a knowledgeable informant. [The Working Group considered that the results of this case-control study may have been affected by differences in the methods of collecting information on smoking, since smoking histories for most cases were obtained from surrogate respondents, whereas those for the majority of the controls were obtained from the respondents themselves, leading to the possibility of bias.] Data were analysed by the method of logistic regression in which age at exit from the study, year of birth, cumulative exposure to respirable glass fibres (expressed as fibres/cm^3-months) and a term reflecting interaction between smoking and exposure to fibrous glass were considered as explanatory variables. Age at exit, year of birth and smoking were statistically significant ($p < 0.05$), but cumulative exposure to glasswool was not. A second analysis was carried out in which smoking was expressed as duration of smoking and time since starting smoking; however, the fit of the model was poor and the results uninformative.

In the European cohort study (Simonato *et al.*, 1987), there was an excess of lung cancer among glasswool workers (93 observed, 73.3 expected; SMR, 127;$p < 0.05$) when compared to national rates; but after local mortality correction factors were applied, the expected number of deaths was increased to 91, giving an SMR of 103. [The Working Group considered that the use of local rates was more appropriate, since one of the glasswool plants, which contributed 76% of the total lung cancer deaths, was located in an area where mortality rates for lung cancer were some 20% higher than the national rates.] For the glasswool workers, there was an increasing trend in SMRs for lung cancer with time since first employment, which was not statistically significant. There was no evidence of a

relationship between lung cancer mortality and duration of employment, nor with technological phase. Similar analyses of lung cancer incidence data in the Nordic countries tend to confirm the mortality patterns, although there were slightly lower ratios of observed to expected numbers of cases. One case of mesothelioma was found, but the specific exposure was not given.

Shannon *et al.* (1987) reported the mortality experience of 2557 men who had worked 90 days or more in a glasswool plant in Canada and who were employed in 1955—1977, following an earlier report (Shannon *et al.*, 1984). They traced 97% of the men, and the cohort was followed for deaths to the end of 1984. The cohort was divided into three groups of workers: plant only, office only and 'mixed exposure'. For the plant-only group, the SMR for lung cancer based on provincial rates was 199 ($p< 0.05$; 19 deaths). In the office-only and mixed-exposure groups together, there were two lung cancer deaths compared to 2.4 expected (SMR, 83). For plant-only workers who had been exposed for five years or more and with ten or more years since first exposure, there were 13 deaths from lung cancer (SMR, 182). The authors examined lung cancer deaths by duration of exposure and time since first exposure and found no increasing relationship. Historical exposure data were not available, but samples taken since 1978 suggested that fibre concentrations were rarely >0.2 fibre/cm^3, mean levels in most areas being <0.1 fibre/ cm^3.

Moulin *et al.* (1986) examined 1374 male workers at a French glass-fibre production factory who were employed at any time during 1975—1984 and who had worked at the factory for at least one year. Occurrence of cancer during these years was ascertained from company records, and the diagnoses were obtained from various medical sources. This study was set up because 'an industrial physician had noticed an excess of cancers in the upper respiratory and alimentary tracts' in the factory. [The Working Group noted that this study could therefore be considered essentially a confirmation of a case report and that the authors did not include oesophagus in the upper alimentary tract.] Expected numbers of cases were calculated using age-specific incidence rates from the combined data of three regional cancer registries covering the period 1975—1981, but which did not include the population of the particular region where the factory was located. To confirm the suitability of the reference incidence rates, the cancer mortality rates in the regions of the three cancer registries were checked against those in the region of the factory. [The Working Group noted that the authors gave no figures from the data used for the check.] For 'upper respiratory and alimentary tract' cancers, 19 cases were observed compared to 8.7 expected (standardized incidence ratio [SIR], 218; 95% confidence interval, 131-341); for lung cancers, five cases were observed compared to 6.8 expected (SIR, 74; 24—172); and, for other cancers, 17 cases (including one case of oesophageal cancer) were reported with 22.1 expected (SIR, 77; 45—124). The expected number of lung cancer cases in workers with 20 years of exposure since first employment was only 1.8 (one observed), indicating the low power of this study. The excess for 'upper respiratory and alimentary tract' cancers was divided among larynx (5 observed, 2.2 expected), pharynx (5 observed, 3.6 expected) and buccal cavity (9 observed, 3.0 expected). The excess was limited to production workers, and among them there was a nonsignificant increasing trend in incidence ratio with increasing duration of employment. These features were not true of lung and other cancers. A survey of cigarette smoking habits among the 1983 workforce indicated slightly lower levels than in France nationally. The authors suspected an etiological role of glass fibre, possibly including fibres both inside and outside the respirable range, because of the sites for which cancer incidence was raised. [The Working Group noted that the paper does not report whether any case of 'upper respiratory and alimentary tract' cancer was later ascertained in addition to the index cases, and that the expected number of lung cancer cases in workers with 20 years of exposure since first employment was very small.]

(b) Glass filament

Enterline *et al.* (1987) reported a cohort study of 3435 white male workers from three US plants that produced continuous glass filament, but not glasswool, who had had one year or more work experience in production or maintenance during the years 1945—1963 and who were followed for deaths to the end of 1982; 97.1% were traced, and death certificates were located for 97% of those who were believed to have died. Expected deaths were based on both US and local age- and time-specific mortality rates. For the period 1946–1982, the SMRs for respiratory cancer were (US) 95 and (local) 92 (64 observed) in the three plants. There was no clear relationship with time since first exposure, nor with duration of exposure nor with a cumulative measure of exposure expressed as fibres/cm^3-months. Estimated mean fibre exposure was low — 0.021, 0.003 and 0.005 fibre/ cm^3 for the three plants, respectively (average, 0.01); the highest individual average fibre exposure level estimated for any member of the cohort was 0.093 fibre/cm^3.

In the European study (Simonato *et al.*, 1987), 15 lung cancer deaths were observed among continuous filament workers, compared to 12.5 expected from national rates (SMR, 120) and 15.4 expected from local figures (SMR, 97) — a change similar to that for glasswool workers, which corresponds generally to higher lung cancer rates among the mainly urban populations in the areas of the factories. There was no trend in SMRs for lung cancer with time since first employment, but the number of expected deaths (2.4 on the basis of local rates) more than 20 years after first exposure was very small. There was no evidence of a relationship between lung cancer mortality or incidence and duration of employment in the glass filament industry. An analysis by technological phase could not be carried out, as the separation into distinct technological phases does not apply to the continuous filament production process.

(c) Rockwool and slagwool

Enterline *et al.* (1987) studied a cohort of 1846 white male workers from six US plants that produced slagwool or rock-/ slagwool. Workers had had one year or more of experience in production or maintenance during the years 1945—1963 and were followed for deaths until 1982; 97% were traced, and death certificates were located for 95% of workers believed to have died. Expected deaths were based on both US and local age- and time-specific mortality rates. For the period 1946–1982, the SMRs for respiratory cancer were (US) 148 and (local) 134 (60 observed; $p < 0.01$ and $p < 0.05$) for the six plants, (US) 156 and (local) 143 (15 observed) for workers with fewer than 20 years since first exposure, and (US) 146 ($p < 0.05$) and (local) 131 (45 observed) for those with 20 years or more since first exposure. The correction factor used for those who had died of unknown causes (see p. 139) was 0.946. SMRs for respiratory cancer were not related to time since first exposure or to duration of exposure. There was a decreasing trend in lung cancer SMR with a time-weighted measure of fibre exposure expressed as fibres/cm^3-months, although this was not statistically significant. SMRs for respiratory cancer were highest for workers first employed most recently. For workers who started during 1950—1959, for example, the SMRs were (US) 216 and (local) 198(19 observed; both $p < 0.01$). One pleural mesothelioma was recorded on the death certificate of a worker with unknown detailed employment history, but the case was not submitted for confirmation to the Mesothelioma Panel. Mean fibre exposure in these plants was estimated to be approximately ten times that in glasswool plants (except for one plant producing small-diameter fibres) and ranged from 0.195 to 0.427 fibre / cm^3. The highest individual average fibre exposure level estimated for any member of this cohort was 1.41 fibres/cm^3.

Enterline *et al.* (1987) carried out 'a case-control within a cohort' study of the rockwool and slagwool workers, adjusting for cigarette smoking. All white men in their cohort study of six slagwool or rock-/slagwool plants who had died of respiratory cancer between 1950 and 1982 were compared with a 4% stratified (by plant and year of birth), random sample of workers, selected from the cohort of workers who had reached the age of 43 prior to 1983. In total, 60 cases and

67 controls were initially selected. Smoking histories for 75% of cases and 73% of controls, and details as to age at which smoking had started and stopped for 63% of cases and 64% of controls, were obtained by telephone interviews with the worker or a knowledgeable informant. Data were analysed by the method of logistic regression in which age at exit from the study, year of birth, cumulative exposure to respirable rock-/slagwool (expressed as fibres/ cm^3-months) and a term reflecting interaction between smoking and exposure to rock-/slagwool were considered as explanatory variables. Only smoking was statistically significant ($p < 0.05$). In a further analysis, in which smoking was expressed as duration of smoking and time since starting smoking, the term representing exposure was positive and statistically significant ($p < 0.01$). Terms relating to smoking were also statistically significant, and the model appeared to be a good fit to the data set ($p = 0.75$). In an attempt to explain the discrepancy between these two analyses and the cohort study, the authors point out that (a) smoking is a powerful variable in this study; (b) smoking multiplies any effect of fibre exposure in the logistic model; and (c) the prevalence of smokers in the cumulative exposure groups varies, ranging from 75% 'ever' smokers in a low category to 54% 'ever' smokers in the highest. Thus, the highest cumulative exposure group in their study consists mainly of US men born in 1900—1909, while the lowest exposure group tends to consist of men born after 1920; and these two cohorts have different smoking patterns. [The Working Group was concerned that the apparent downward trend for lung cancer against cumulative exposure in the cohort analysis had changed to a positive coefficient for cumulative exposure in the case-control study. The results of this case-control study may have been affected by differences in the methods of collecting information on smoking, since smoking histories for most cases were obtained from surrogate respondents, whereas those for the majority of controls were obtained from the respondents themselves. As a result, it is possible that the effects of smoking were not fully controlled, and the variable for time-weighted cumulative exposure to fibres may have been improperly corrected for the effects of smoking. These factors made the results of the analysis difficult to interpret. In view of the major effect of smoking on the incidence of lung cancer, any uncertainty regarding smoking histories makes it impossible to disentagle, with any confidence, by statistical analysis, any effect of estimated amounts of cumulative exposure.]

In the European cohort (Simonato *et al.*, 1987), among rock-/slagwool workers, 81 deaths from lung cancer were observed compared to 65.4 expected from national rates; the expected number remained unchanged after application of local correction factors, giving an SMR of 124 [95% confidence interval, 98—154]. There were increasing trends in the SMRs for lung cancer with time since first employment, which were not statistically significant.

In terms of lung cancer mortality and technological phase, a statistically significant decreasing trend in SMR was observed for rock-/slagwool workers from early to intermediate to late phases, independent of whether the comparison was with national or local corrected rates. The highest SMRs for lung cancer were seen among workers employed during the early phase and followed up for more than 20 years; ten deaths were observed compared to 4.0 expected from national rates [SMR, 250; 120—460] and 3.3 expected from local rates [SMR, 303; 145–557]. The decreasing trend in SMR by technological phase was also observed after follow-up for more than 20 years after first employment, and it reached statistical significance with both national (X^2, 6.5 [one degree of freedom; $p < 0.05$]) and local (X^2, 9.8 [one degree of freedom; $p < 0.01$]) reference rates. Similar analyses of lung cancer incidence in Nordic countries tended to confirm the mortality patterns, although there were slightly lower ratios of observed to expected numbers of cases. There was no evidence of a relationship between lung cancer mortality or incidence and duration of employment in the rock-/slagwool industry (Simonato *et al.*, 1987).

Lung cancer mortality was examined in relation to other workplace conditions on the basis of the historical environmental investigation (Cherrie & Dodgson, 1986). Neither the presence of asbestos in some products, the use of bitumen and pitch as a binder nor exposure to formaldehyde appeared to explain the lung cancer excesses (Simonato *et al.*, 1987). [The Working Group noted that other potential exposures, such as to silica and chromium, were not taken into account.]

The use of slag as raw material is associated with excess mortality from lung cancer in rock-/slagwool production. This finding was, however, difficult to interpret, as the periods during which slag was in use include the entire early technological phase, in which the estimated fibre levels were highest and which contributes most of the lung cancer excess. In later phases, when the estimated fibre levels had been reduced, the use of slag was associated with an SMR of 146 (13 observed, 8.9 expected) (Simonato *et al.*, 1987).

The authors concluded that, since respirable fibres were a significant component of the pollution within the workplace, it is plausible that fibre exposure during the early phase of rock-/slagwool production, alone or in combination with other factors, may have contributed to the observed lung cancer excess. Cigarette smoking was considered unlikely to account for the more than a two-fold excess of lung cancer. There was no evidence of an increase in the incidence of pleural tumours or of nonmalignant respiratory diseases (Simonato *et al.*, 1987).

(d) Cancer at sites other than the lung

In the European study (Simonato *et al.*, 1987), bladder was the only site for which there was a statistically significant increasing trend in cancer mortality with time since first exposure; this trend was limited to rock-/ slagwool workers, but there was no relationship to technological phase, in contrast to lung cancer. [The Working Group noted that this comparison was one of a large number carried out, and the result may be a chance finding.]

There was a small excess of mortality from cancers of the buccal cavity and pharynx (13 observed, 10.6 expected from national rates; SMR, 123) (Simonato *et al.*, 1987), but there was a statistically significant excess in incidence among rock-/slagwool workers (22 observed, 12.2 expected from national rates; SIR, 181; 95% confidence interval, 113—274), as compared, for example, to glasswool workers (4 observed, 4.9 expected; SIR, 83) (Simonato *et al.*, 1986a). The study by Moulin *et al.* (1986) of French glasswool production workers was set up because of the observation by an industrial physician of an excess of cancers of the pharynx, larynx and buccal cavity. [The Working Group commented earlier (p. 142) on the limited nature of this study.]

A small excess of cancer of the larynx (4 observed, 2.1 expected from local rates; SMR, 188) was observed by Bertazzi *et al.* (1984, 1986) in the Italian glasswool/glass filament subcohort of the European study. No parallel finding emerged from the other subcohorts.

An increase in mortality from cancer of the digestive tract was reported in one of the US rock-/slagwool plants (Robinson *et al.*, 1982) in a study of 596 workers. The SMR was 130 (15 observed); SMRs increased with time since first exposure and with duration of exposure. The excess was not related to any particular site in the digestive tract. Claude and Frentzel-Beyme (1986) reported an increase in stomach cancer mortality with time since first exposure, based on small numbers (8 observed, 4.5 expected for ⩾20 years' exposure) in the Federal Republic of Germany subcohort of the European study.

[The Working Group could not regard any of these associations as established due to their relatively weak strength, lack of consistency and to the unaccounted role of exposures such as alcohol and tobacco smoking.]

(e) Overview of results of major epidemiological studies of production workers

Table 42 gives the main findings from the US and European epidemiological studies of glasswool, glass filament and rock-/ slagwool plants, both individually

and in combination, where appropriate. There is a notable similarity between the outcomes of the two large investigations when comparable analyses were made. Findings from the Canadian study of glasswool workers are footnoted.

Table 43 summarizes results from the US study and shows a relationship between estimated fibre concentrations and observed SMRs for the total cohorts and for workers 20 years after first exposure.

(f) Users with mixed exposure

A report by Engholm *et al.* (1987) gave results of an extended follow-up of Swedish construction workers to December 1982 for lung cancer registration and to December 1983 for mortality. Of a total of 135 037 Swedish male construction workers, 135 026 were followed up from 1971—1974, when they were first examined medically. Exposure to man-made mineral fibres (mixed categories), exposure to asbestos, occupation, cigarette smoking habits and other information were determined by questions during the medical examination (Engholm *et al.*, 1984). The numbers of lung cancer cases were 440 observed and 483 expected, giving an SIR of 91 (95% confidence interval, 83-100). A nested case-control study was carried out to examine the relationship between lung cancer and exposures to man-made mineral fibres and to asbestos, classified on the basis of a combination of job category and self-reported information. The authors suggested that these construction workers had had exposure to asbestos because of the occurrence of 23 cases of pleural mesothelioma, with 11 expected; an analysis within the paper suggests that heavy exposure to asbestos was underreported by the workers. Using a revised classification of heavy exposure and adjusting for smoking habits and population density of area of residence in a logistic regression analysis, the authors reported a relative risk of 1.21 (95% confidence interval, 0.60—2.47) for exposure to man-made mineral fibres (adjusted for asbestos exposure) and a relative risk of 2.53 (0.77—8.32) for exposure to asbestos (adjusted for man-made mineral fibre exposure). The authors discuss the difficulty caused in the analysis by the large overlapping of reported exposures to asbestos and man-made mineral fibres.

4. Summary of Data Reported and Evaluation

4.1. Exposure data

More than 5 million tonnes of man-made mineral fibres are produced annually in more than 100 factories located throughout the world. Glass fibre products comprise over 50% of the total. Most glasswool, rockwool and slagwool is used for thermal and acoustical insulation in the construction industry. Glass filaments are used mainly as textiles and as reinforcement materials in plastics. Ceramic fibres are being produced in increasingly large quantities for high-temperature insulation and in specialty products.

Man-made mineral fibre products release airborne respirable fibres during their production and use. In general, as the nominal diameter of man-made mineral fibre products decreases, both the concentration of respirabie fibres and the ratio of respirabie to total fibres increase. Exposure levels in glasswool production have generally been 0.1 respirabie fibre/cm^3 or less; in rockwool and slagwool production, exposures have been somewhat higher. Higher occupational exposures may occur when man-made mineral fibre products are used in confined spaces, such as in the application of loose insulation. Concentrations of man-made mineral fibres have been measured in outdoor air and in nonoccupational settings indoors and found to be much lower than those associated with occupational settings.

4.2. Experimental carcinogenicity data

Glasswool

Several samples of glasswool with different particle size distributions in the respirabie range were tested by inhalation in five experiments in rats, in one experiment in hamsters, and in one limited experiment in baboons. There was no statistically significant increase in the incidence of tumours of the lung or pleura; however, a few respiratory-tract tumours occurred in most experiments in rats. It should be noted that in the intended positive control groups, crocidolite produced no statistically significant increase in lung tumour incidence, while chrysotile usually did.

Glasswool was adequately tested in two experiments in rats and in one experiment in hamsters by intratracheal instillation. Lung tumours were observed in one experiment in rats, and lung tumours and mesotheliomas were observed in the experiment in hamsters, after repeated instillations of samples of glasswool with median fibre diameter less than 0.3 μm. No lung tumour or mesothelioma was induced by glasswool in the other experiment by intratracheal instillation in rats; however, in the positive control group treated with crocidolite, there was a low incidence of lung tumours.

Various samples of glasswool were tested by intrapleural implantation or injection in five studies in rats and in one in mice. Pleural tumours were induced in four of five studies in rats, the incidence varying with the size of the instilled fibres. No pleural tumour was observed in treated mice.

Samples of glasswool were injected into the peritoneal cavity in eight studies in rats and in one in hamsters. Mesotheliomas or sarcomas were induced (the incidence depending on dose and fibre size) in the peritoneal cavity in all studies in rats, but prior ‘leaching’ of the fibres with hydrochloric acid in two studies reduced or eliminated the incidence of these tumours. Treatment of the fibres with sodium hydroxide did not reduce the carcinogenicity. No tumour was induced in hamsters.

Glass filament

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

Rockwool

In two studies in which rats were exposed to rockwool by inhalation, no statistically significant increase in lung tumour incidence was observed in one study and no lung tumour in the other. Chrysotile was used as the positive control in both studies and led to high pulmonary tumour incidence.

Rockwool was tested by intrapleural injection in one experiment in rats, producing a low, statistically nonsignificant increase in the incidence of pleural mesotheliomas. After intraperitoneal injection of two samples of rockwool in two experiments in one laboratory, a high incidence of tumours was observed in the abdominal cavity; however, in one study, the histopathology had not been completed.

Slagwool

Slagwool was tested in one experiment by inhalation in rats and hamsters; no increase in the incidence of respiratory-tract tumours was reported. In the intended positive control groups, crocidolite induced no or few tumours. In two experiments in rats, intrapleural injection of slagwool produced no thoracic tumour in one study and one pleural sarcoma in the other. In one study in rats by intraperitoneal injection, equivocal findings were obtained.

Ceramic fibres

In an experiment in which rats were exposed to ceramic fibres by inhalation, a statistically significant increase in the incidence of benign and malignant tumours of the lung was observed. Two further studies, one in rats and one in hamsters, by inhalation showed no increased tumour incidence in groups exposed to ceramic fibres, whereas, in the intended positive control group, crocidolite produced a few lung tumours in rats but not in hamsters. No pulmonary tumour was found in an experiment in which rats were exposed by inhalation to relatively thick ceramic fibres.

Intratracheal instillation of ceramic fibres did not produce lung tumours in one study in rats and in one study in hamsters, while, in the intended positive control group, crocidolite produced a high percentage of benign and malignant lung tumours in hamsters but only a few in rats.

In one study, intrapleural implantation in rats of several kinds of ceramic fibres produced variable incidences of pleural mesotheliomas or sarcomas. Another study of ceramic fibres injected into the pleural cavity of rats produced equivocal results.

After intraperitoneal injection of ceramic fibres into rats in three experiments, mesotheliomas were found in the abdominal cavity in two studies. Only a few mesotheliomas were found in the abdominal cavity of hamsters after intraperitoneal injection in one experiment; however, the ceramic fibres tested were of relatively large diameter.

In interpreting all these experiments, the Group had in mind considerations outlined in the ‘General Remarks on Man-made Mineral Fibres’, pp. 34–35.

4.3. Human carcinogenicity data

No increase in the occurrence of mesothelioma has been observed in man-made mineral fibre production workers.

Glasswool

The main study of glasswool workers in the USA showed a slightly raised mortality from respiratory cancer compared to local rates. Mortality from respiratory cancer increased with time since first exposure, but was not related to duration of exposure nor to an estimated time-weighted measure of fibre exposure. A subcohort of these workers who were exposed to small-diameter fibres had a higher standardized mortality ratio for respiratory cancer than those not exposed, which increased with time since first exposure. Neither the overall increase nor any of these trends was statistically significant.

In the multinational European study, there was no overall excess mortality from lung cancer compared to regional rates. Mortality from lung cancer showed a statistically nonsignificant increase with time since first exposure but was not related to duration of exposure or to different technological phases reflecting differences in the intensity and quality of exposure.

A study of Canadian glasswool workers showed a substantially raised mortality from lung cancer, which was statistically significant, but this was not related to time since first exposure or to duration of exposure.

Glass filament

Among glass filament workers in the US study, there was no excess of respiratory cancer, and in the European study no excess of lung cancer, and no upward trend with time since first exposure or with duration of exposure in either study. In the US study, there was also no trend with an estimated time-weighted measure of exposure.

Rockwool and slagwool

Effects of exposures in rockwool and slagwool industries could not be distinguished in the studies reported. The two are therefore referred to together as ‘rock-/slagwool’.

The study of rock-/slagwool workers in the USA indicated a statistically significant raised mortality from respiratory cancer compared to local rates. In this cohort, however, there was no relationship with time since first exposure, duration of exposure or an estimated time-weighted measure of fibre exposure.

In the European study, there was an overall, statistically nonsignificant excess of lung cancer among rock-/slagwool workers compared to regional rates, as well as a statistically nonsignificant increasing mortality with time since first exposure. There was no relationship between lung cancer mortality and duration of exposure. The highest and statistically significant lung cancer rates were found after more than 20 years’ follow-up among persons first exposed during the early technological phase (i.e., before the introduction of oil binders and during the use of batch processing methods). Slag was used as a raw material particularly during this phase of the industry. There was a statistically significant decreasing trend in lung cancer mortality with the introduction of oil binders and modern mechanized methods of production. The presence of asbestos, bitumen, pitch and formaldehyde as work place contaminants could not explain the lung cancer excess.

In the US and European studies combined, there was a statistically significant excess of mortality from lung cancer for rock-/slagwool workers.

The raised lung cancer mortality rates were considered unlikely to be the result of confounding due to cigarette smoking, although this was not directly measured in the cohort studies.

4.4. Other relevant data

Many samples of man-made mineral fibres with large fibre diameter have low respirability.

The solubility of man-made mineral fibres *in vitro* and their durability *in vivo* vary with chemical composition. While, in general, glasswool fibres appear to be

relatively nondurable, one sample was shown to be very insoluble *in vitro.* Conversely, while in one study ceramic fibres were very durable, one sample proved to be as soluble as glasswool used for comparison in the same experiment *in vitro.* Insufficient samples of slagwool and rockwool have been tested to allow a prediction of their overall range of solubility in tissues. On the available evidence, no generalization can be made regarding the durability of any single class of man-made mineral fibres.

There is little evidence for acute toxicity after the inhalation of man-made mineral fibres. Glasswool, rockwool and slagwool administered by inhalation produced little pulmonary fibrosis in experimental animals. Glasswool was fibrogenic following intratracheal instillation in some but not all studies. In one study in rats, inhaled ceramic fibres were fibrogenic.

Glasswool induced numerical and structural chromosomal alterations but not sister chromatid exchanges in mammalian cells *in vitro.* It caused morphological transformation in rodent cells *in vitro;* transformation was found to be dependent on fibre length and diameter. Glasswool did not induce mutation in bacteria.

Ceramic fibres caused a weak response in an assay for morphological transformation but did not induce DNA damage in mouse cells *in vitro.*

No adequate data on genetic and related effects of rockwool and slagwool were available.

4.5. Evaluation[1]

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

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

There is *inadequate evidence* for the carcinogenicity of glass filaments and of slagwool in experimental animals.

There is *inadequate evidence* for the carcinogenicity of glasswool and of glass filaments in humans.

There is *limited evidence* for the carcinogenicity of rock-/slagwool in humans.

No data were available on the carcinogenicity of ceramic fibres to humans.

Overall evaluation[1]

Glasswool is *possibly carcinogenic to humans (Group 2B).*

Glass filaments are *not classifiable as to their carcinogenicity to humans (Group 3).*

Rockwool is *possibly carcinogenic to humans (Group 2B).*

Slagwool is *possibly carcinogenic to humans (Group 2B).*

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

Footnotes

1 Only those synonyms and trade names used in this monograph are listed.

1 For definition of the italicized terms, see Preamble, pp. 28–34

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Figures

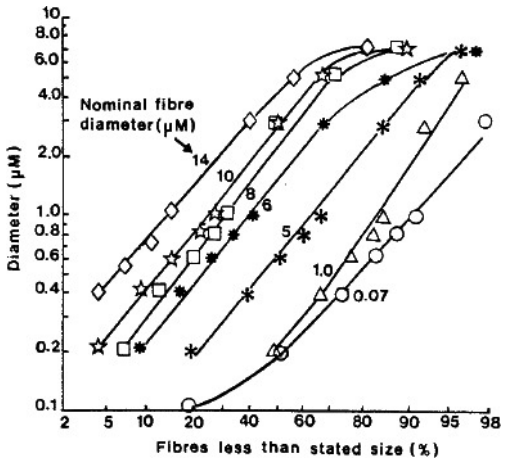

Fig. 1

Distribution of diameters of airborne fibres[a]

[a]From Esmen *et al.* (1979a), expressed as cumulative % of fibres less than stated size measured during production of fibres of different nominal diameter

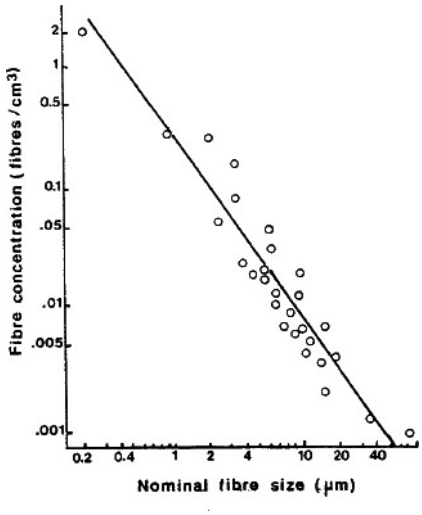

Fig. 2

Relationship between measured average exposures (fibres/m^3 determined by phase-contrast microscopy) and nominal diameter of manufactured fibre[a]

[a]From Esmen *et al.* (1979a); each point represents the average concentration of fibres calculated from all samples collected in a plant production unit or in an entire facility producing the nominal fibre size indicated.

Tables

Table 1 Composition (% by weight) of glasses[a] used in fibre manufacture[b]

Component	Glass type					
	E	**C**	**A**	**S**	**Cemfil**	**AR**
SiO_2	55.2	65	72.0	65.0	71	60.7
Al_2O_3	14.8	4	2.5	25.0	1	–
CaO	18.7	14	9.0	–	–	–
MgO	3.3	3	0.9	10.0	–	–
B_2O_3	7.3	5	0.5	–	–	–
Na_2O	0.3	8.5	12.5	–	11	14.5
K_2O	0.2	–	1.5	–	–	2.0
ZrO_2	–	–	–	–	16	21.5
Li_2O	–	–	–	–	1	1.3
F_2[c]	0.3	–	–	–	–	–
Fe_2O_3	0.3	0.3	0.5	trace	trace	trace

a E, electrical fibre component; C, chemical glass (used in, e.g., surfacing mats for corrosion resistance); A, common soda lime type; S, high-strength, high-modulus (for high-performance structures); AR, alkali resistant (for reinforcement of concrete)

b From Loewenstein (1983)

c Fluorine present in glass presumably as fluorides

Table 2 Composition (% by weight) of some typical commercial glass fibres[a]

Component	Glass type					
	I	**II**	**III**	**IV**	**V**	**VI**
SiO_2	54.5	65.0	59.0	73.0	59.5	34.0
Al_2O_3	14.5	4.0	4.5	2.0	5.0	3.0
CaO	22.0	14.0	16.0	5.5	–	–
MgO	–	3.0	5.5	3.5	–	–
B_2O_3	8.5	5.5	3.5	–	7.0	–
Na_2O	0.5	8.0	11.0	16.0	14.5	0.5
K_2O	–	0.5	0.5	–	–	3.5
ZrO_2	–	–	–	–	4.0	–
TiO_2	–	–	–	–	8.0	–
PbO	–	–	–	–	–	59.0
F[b]	–	–	–	–	2.0	–

a From National Institute for Occupational Safety and Health (1977a)

b Fluorine present in glass presumably as fluorides

Table 3 Characteristics and physical properties of some commercial fibrous glass[a]

Glass type	Form	Fibre diameter range (µm)	Specific gravity (g/cm^3)	Refractive index
I	Textiles, mats	6–9.5	2.596	1.548
II	Mats Textiles	10–15 6–9.5	2.540	1.541
III	Wool (coarse)	7.5–15	2.605	1.549
IV	Packs (coarse)	115–250	2.465	1.512
V	Wool, fine ultrafine	0.75–5 0.25–0.75	2.568	1.537
VI	Textiles	6–9.5	4.3	–

a From National Institute for Occupational Safety and Health (1977a)

Table 4 US glass fibre size designations and associated diameters[a]

Fibre size designation	Nominal diameter (µm)	
	Min	**Max**
AAAAA	0.05	0.20
AAAA	0.20	0.50
AAA	0.50	0.75
AA	0.75	1.50
A	1.50	2.52
B	2.52	3.81
C	3.81	5.08
D	5.08	6.35
E	6.35	7.62
F	7.62	8.89
G	8.89	10.12
H	10.12	11.43
J	11.43	12.70
K	12.70	13.97
L	13.97	15.24
M	15.24	16.51
N	16.51	17.78
P	17.78	19.05
Q	19.05	20.32
R	20.32	21.58
S	21.58	22.86
T	22.86	24.13
U	24.13	25.40

a From Corn (1979)

Table 5 Nominal fibre diameters and binders for commercial fibrous glass products[a]

Product	Nominal fibre diameter (µm)	Type of binder
Wool products		
General thermal insulation	6–15	Resin
Moulded pipe insulation	7–9	Resin
Lightweight aircraft insulation	1.0–1.5	Resin
High-temperature insulation and filter paper	0.05–3.0	Resin
Textile products		
Continuous filament electrical insulation	6–9.5	Coatings
'Silver' type electrical insulation	7–9.5	Lubricant
Plastic reinforcing mat	6–9.5	Resin
Wrap-on pipe insulation	3.5	Resin

a From Dement (1975)

Table 6 Composition (% by weight) of insulation-type rockwool and slagwool fibres[a]

Component	Rockwool[a]			Slagwool[a]		Darkwool[b]		Diabase or basalt fibres[b]		
	1	2	3	1	2	USG Tacoma dark steel slagwool	Rockwool industries	Gullfibre	Fibre in low density tile	German basalt fibres
SiO_2	52.92	47.5	45.54	41.0	40.58	40.97	39.11	42.92	46.94	44.31
Al_2O_3	6.52	13.0	13.38	11.8	12.52	5.09	7.44	12.56	13.40	12.53
MgO	–	–	–	–	–	7.54	8.92	6.79	10.34	10.49
CaO	30.28	16.0	10.80	40.0	37.50	19.69	31.89	29.65	16.85	11.46
FeO	1.01	7.0	5.75	0.9	1.0	21.10	8.97	2.15	6.56	11.07
TiO_2	0.51	1.5	1.99	0.4	0.44	0.28	0.35	2.47	1.83	2.43
MnO	0.06	0.5	0.24	0.6	0.30	0.06	0.47	0.68	0.15	0.20
Na_2O	2.29	2.5	2.52	0.2	1.45	0.71	0.34	1.24	2.34	3.75
K_2O	1.57	1.0	1.36	0.4	0.30	0.73	0.76	0.67	0.87	1.66
SO_3	–	–	–	–	–	3.29	0.46	0.46	0.04	–
P_2O_5	0.15	–	0.06	0.3	0.21	0.29	0.30	0.08	0.27	1.17
Fe_2O_3	1.48	0.5	8.22	–	–	–	–	–	–	–
CaS	–	–	–	–	1.04	–	–	–	–	–
S	–	–	–	0.4	0.46	–	–	–	–	–
F[c]	–	–	–	0.4	–	–	–	–	–	–

a From Mansmann *et al.* (1976)

b From Owens-Corning Fiberglas Corp. (1987)

c Fluorine present in glass presumably as fluorides

Table 7. Radon levels in dwellings in various countries

Component (% by weight)	Fiberfrax® bulk	Fiberfrax® long staple	Fibermax® bulk	Fiberfrax® HSA	Alumina bulk (Saffil®)	Zirconia bulk	Fireline ceramic	Nextel® 312 fibre
Al_2O_3	49.2	44.0	72.0	43.4	95.0	–	95 and 97.25	62.0
SiO_2	50.5	51.0	27.0	53.9	5.0	<0.3		24.0
ZrO_2	–	5.0	–	–	–	92.0	–	–
Fe_2O_3	0.06	–	0.02	0.8	–	–	0.97 and 0.53	–
TiO_2	0.02	–	0.001	1.6	–	–	1.27 and 0.70	–
K_2O	0.03	–	–	0.1	–	–	–	–
Na_2O	0.20	–	0.10	0.1	–	–	0.15 and 0.08	–
CaO	–	–	0.05	–	–	–	0.07 and 0.04	–
MgO	–	–	0.05	–	–	–	Trace	–
Y_2O_3	–	–	–	–	–	8.0	–	–
B_2O_3	–	–	–	–	–	–	0.06 and 0.03	14.0
Leachable chlorides (ppm [mg/kg])	<10	<10	11	<10	–	–	–	–
Organics	–	–	–	–	–	–	2.47 and 1.36	–

From Zircar Products (1978a,b); Sohio Carborundum Co. (1986); Fireline (undated); 3M Center (undated); Zircar Products (undated)

Table 8 Chemical and physical properties of some typical ceramic fibres[a]

Fibre trade name	Description	Melting-point (°C)	Specific gravity (g/cm^3)	Fibre diameter (mean; µm)	Fibre length (mean; mm)	Fibre surface area (m^2/g)
Fiberfrax® bulk[b]	White	1790	2.73	2–3	Up to 102	0.5
Fiberfrax® long staple[b]	White	1790	2.62	5 and 13	Up to 254	NA
Fibermax® bulk[b]	White, mullite polycrystalline	1870	3	2–3.5	NA	7.65
Fiberfrax® HSA[b]	White to light-grey	1790	2.7	1.2	3	2.5
Alumina bulk	White	2040	0.096	3	3	NA
(Saffil®)[c]						
Zirconia bulk[d]	White	2600	0.24–0.64	3–6	1.5	NA
Fireline ceramic[e]	White to cream	1700	NA	NA	NA	NA
Nextel® 312 fibre[f] (filament)	White, smooth, transparent, continuous polycrystalline metal oxide	1700	>2.7	8–12	Continuous	<1

a From Zircar Products (1978a,b); Sohio Carborundum Co. (1986); Fireline (undated); 3M Center (undated); Zircar Products (undated)

b Resistant to attack from most corrosive agents, except hydrofluoric acid, phosphoric acid and strong alkalies; resistant to oxidation and reduction; high temperature stability, low thermal conductivity, low heat storage, thermal shock resistance, light weight, excellent sound absorption

c Corrosion resistant; light weight, low thermal conductivity, low thermal mass, thermal shock resistance, high dimensional stability, high temperature resilience, refractoriness

d Resistant to oxidation and reduction; low thermal conductivity, great refractoriness

e Highly resistant to attack from most corrosive agents, except hydrofluoric acid, phosphoric acid and certain strong alkalies; low thermal conductivity, light weight, thermal shock resistance, moisture resistance

f Corrosion resistant, except for phosphates, alkali metal salts, colloidal silica, colloidal alumina and castable refractory cements and mortars; compatible with silicone, epoxy, and phenolic and polyimide matrix materials; high temperature stability, dimensional stability, low specific heat, thermal shock resistance, low thermal conductivity, high electrical resistance, moisture resistance, abrasion resistance

NA, not available

Table 9 Estimated world production of man-made mineral fibre materials in 1973 (million kg)[a]

Location	Insulation			Textile			Total	
	Quantity	%		Quantity	%		Quantity	%
Europe	1800	48		345	40		2145	47
Western	1200	32		260	30		1460	32
Eastern	600	16		85	10		685	15
North America	1600	43		400	46		2000	43
Japan	200	5		100	12		300	7
Australia	30	1		–	–		–	–
Central/South America	120	3		20	2		140	3
World	3750			865			4585	

a From World Health Organization (1983)

Table 10 Quantities of glasswool, rockwool and slagwool products produced in the USA (million kg)[a]

Product	1977	1982
Mineral wool for thermal and acoustical envelope insulation (for insulating homes and commercial and industrial buildings) made from fibre produced in the same establishment[b]		
Loose and granulated fibre	373.2	327.2
Building batts, blankets and rolls (in thermal resistance values)		
R-19.0 or more	359.9	530.0
R-11.0 to R-18.9	403.9	418.4
R-10.9 or less	NA	52.3
Acoustical, such as wall and ceiling	NA	46.3
Mineral wool for industrial, equipment and appliance insulation made from fibre produced in the same establishment		
Flexible blankets, including fabricated pieces, rolls and batts		
Plain	153.9	173.2
Coated	16.0	21.4
Faced and metal meshed	24.0	
Special purpose insulation pieces such as automobile, appliance and aerospace items and original equipment parts	11.3	11.5
Other blocks and boards	22.0	10.0
Pipe insulation	22.0	26.8
Acoustical, including pads, boards and patches	24.0	NA
Mineral wool for industrial, equipment and appliance insulation made from fibre purchased or transferred from other establishments		
Flexible blankets, including fabricated pieces, rolls and batts Plain	13.9	NA
Coated	0.5	NA
Special purpose insulation pieces such as automobile, appliance and aerospace items and original equipment parts	8.3	NA
Other blocks and boards	24.0	NA
Pipe insulation	8.5	NA

a From US Department of Commerce (1985)

b Based on US dollar value; larger quantities are made into thermal and acoustical insulation at establishments other than those producing the fibre, but production data are not available.

NA, not available

Table 11 Glass fibre production in the USA (million kg)[a]

Year	Quantity
1975	247.88
1976	306.90
1977	357.30
1978	419.04
1979	460.36
1980	393.62
1981	472.61
1982	408.15
1983	530.27
1984	632.88

a From Anon. (1986)

Table 12 Estimated US consumption of alumina-silica ceramic fibres in 1983[a]

Product	Consumption (million kg)	Approximate % of total
Blanket and felt	20.5	50%
Bulk fibre	4.1	10%
Vacuum-formed shapes	5.0	12%
Boards and blocks	3.2	8%
Paper	2.3	6%
Other[b]	5.9	14%

a From US Environmental Protection Agency (1986)

b Includes coatings, sprays, castables, textiles and miscellaneous

Table 13 Dust concentrations (mg/m^3) by plant and operation in fibrous glass production plants in the USA[a]

Operation	Plant no.[b]	Total dust			Respirable dust (<5 µm)	
		Mean	Range		Mean	Range
Batch and marble	1	–	–		–	–
	2	–	–		–	–
	3	1.34	0.18–5.96		0.15	<0.01–0.31
	4	12.29	2.69–21.89		0.55	0.06–1.03
	1–4[c]	6.82	0.18–21.89		0.35	<0.01–1.03
	5	0.12	0.12		0.36	0.19–0.52
Forming	1	0.20	<0.01–0.94		0.03	<0.01–0.24
	2	0.44	0.04–1.70		0.05	<0.01–0.20
	3	0.18	<0.01–0.66		0.07	<0.01–0.45
	4	0.46	0.04–1.74		0.09	<0.01–0.47
	1–4[c]	0.32	<0.01–1.74		0.06	<0.01–0.47
	5	0.06	0.04–0.22		0.05	<0.01–0.36
Spinning and twisting	5	0.11	<0.01–0.40		0.10	<0.01–0.65
Waste recovery	5	0.16	<0.01–0.48		0.12	<0.01–0.73

a From Johnson *et al.* (1969)

b Plants 1–4 are insulation plants; plant 5 is a textile plant.

c Composite results for plants 1–4

Table 14 Fibre concentrations (fibres/cm^3; including fibres <5 µm in diameter) by plant and operation in fibrous glass production plants in the USA[a]

Operation	Plant no.[b]	Total fibres		Fibres longer than 5 µm		Fibres longer than 10 µm	
		Mean	Range	Mean	Range	Mean	Range
Batch and marble	1	–	–	–	–	–	–
	2	3.64	3.64	0.97	0.97	0.54	0.54
	3	0.66	0.41–1.03	0.16	0.10–0.26	0.08	0.02–0.16
	4	0.30	0.08–0.67	0.10	0.02–0.25	0.04	0–0.07
	1–4[c]	1.53	0.08–3.64	0.41	0.02–0.97	0.22	0–0.54
	5	0.09	0.09	0.04	0.04	0	0
Forming	1	–	–	–	–	–	–
	2	0.41	0.04–2.95	0.12	0–0.56	0.08	0–0.35
	3	0.15	0.02–0.45	0.04	0–0.14	0.02	0–0.09
	4	0.19	0.07–0.31	0.07	0.01–0.19	0.03	0–0.06
	1–4[c]	0.25	0.02–2.95	0.08	0–0.56	0.04	0–0.35
	5	0.10	0–0.19	0.02	0–0.04	0.01	0–0.04
Spinning and twisting and waste recovery	5	0.72	0.03–12.67	0.11	0–1.97	0.01	0–0.06

a From Johnson *et al.* (1969)

b Plants 1–4 are insulation plants; plant 5 is a textile plant.

c Composite results for plants 1–4

Table 15 Characteristics of 16 facilities in the USA surveyed by Esmen *et al.* (1979a)

Plant no.	Type of fibre produced	Material	No. of dust samples	Average nominal diameter (µm)
1	Loose and continuous	Glass	97	1–12
2	Loose	Slag	55	6
3	Loose	Glass	70	3–6
4	Loose and mixed	Glass	90	1–6
5	Loose	Slag	60	8
6	Loose, continuous and mixed	Glass	111	5–15
7	Loose	Rock	63	6
8	Loose	Glass	105	7–10
9	Loose	Glass	89	7–10
10	Continuous	Glass	97	6–16
11	Loose	Slag	66	7
12	Loose, continuous and mixed	Glass	225	6–115
13	Loose	Rock and slag	72	7
14	Continuous	Glass	84	6–13
15	Loose	Glass	79	0.05–1.6
16	Loose	Glass	90	6–10

Table 16 Plant operations upon which the grouping of data in Tables 17, 18 and 20 were based[a]

Classification	Description
Forming	All hot-end workers, cupola operators, batch mixers, transfer operators, charging operators
Production	Cold-end workers in direct contact with fibres but not involved in cutting, sawing, sanding or finishing operations; workers such as bailers, stuffing operators, machine tenders
Manufacturing	Workers involved in general manufacturing operations, such as trimming, sawing, cutting, finishing, painting finished boards, moulding-drier ovens, handling boxed and/ or packaged materials
Maintenance	Maintenance workers who repair production machinery and do general work in the production area, including sweeping floors, cleaning dust collecters and machinery, general cleaning within the plant
Quality control	Workers who sample the product and ascertain product quality
Shipping	Transportation of packaged material, fork-truck operators, shipping-yard operators

a From Esmen *et al.* (1979a)

Table 17 Concentrations of total suspended particulate matter (mg/m^3) in 16 facilities in the USA[a]

Plant	Forming		Production		Manufacturing		Maintenance		Quality control		Shipping		Overall	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
1	0.47	0.47	1.04	1.34	0.96	0.96	0.71	0.45	0.21	0.12	0.39	0.09	0.89	1.12
2	1.65	1.17	2.53	2.30	2.28	1.51	2.05	1.32	1.53	0.63	1.34	0.58	1.94	1.68
3	–	–	0.51	0.30	–	–	0.83	0.61	–	–	0.70	0.42	0.65	0.46
4	1.22	0.51	0.77	0.49	1.23	0.95	2.08	4.40	0.52	0.14	1.32	0.96	1.24	2.26
5	0.76	0.25	0.67	1.52	0.29	1.25	0.55	0.32	0.09	_	0.62	0.33	0.60	1.04
6	1.30	0.71	1.77	2.23	0.51	0.39	2.00	2.50	0.49	0.82	0.45	0.19	1.17	1.72
7	2.18	1.62	2.05	0.31	4.31	4.03	6.72	7.84	–	–	1.77	1.02	4.00	4.27
8	–	–	8.48	9.02	1.17	0.55	4.64	8.28	–	–	0.84	0.67	4.73	8.69
9	1.18	0.48	1.90	1.52	1.14	0.53	1.33	0.57	–	–	1.08	0.46	1.33	1.02
10	2.45	0.93	0.75	0.47	0.73	0.33	1.25	1.07	0.32	0.09	0.69	0.15	1.07	0.91
11	2.18	1.64	1.08	1.82	0.87	0.46	1.26	0.49	1.25	–	1.04	0.41	1.37	1.09
12	0.34	0.35	0.20	0.30	0.28	0.26	0.53	0.26	0.53	0.66	0.88	0.08	0.21	0.16
13	4.10	–	1.34	0.46	1.19	1.08	1.80	1.69	–	–	1.31	0.59	1.4	1.08
14	3.00	1.37	0.85	0.59	1.06	0.47	1.57	1.41	–	–	0.91	0.72	1.42	1.21
15	0.30	0.21	0.61	0.51	1.08	0.80	1.09	0.75	1.66	0.73	0.54	0.18	0.75	0.67
16	0.77	0.46	0.82	0.69	0.86	0.52	1.79	1.50	0.44	–	0.76	0.53	1.07	1.02

a From Esmen *et al.* (1979a)

Table 18 Concentrations (fibres/cm^3) of airborne fibres, as determined by optical microscopy, in 16 facilities in the USA[a]

Plant	Forming		Production		Manufacturing		Maintenance		Quality control		Shipping		Overall	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
1	0.002	0.001	0.38	0.32	0.03	0.02	0.02	0.02	0.07	0.10	0.01	0.001	0.01	0.25
2	0.07	0.03	0.17	0.14	0.12	0.11	0.08	0.05	0.19	0.16	0.07	0.06	0.11	0.12
3	–	–	0.02	0.02	–	–	0.07	0.18	–	–	0.005	0.01	0.04	0.10
4	0.01	0.004	0.07	0.12	0.04	0.05	0.03	0.02	0.01	0.01	0.02	0.01	0.04	0.08
5	0.02	0.01	0.03	0.02	0.03	0.02	0.02	0.01	0.03	–	0.03	0.01	0.02	0.02
6	0.05	0.10	0.01	0.01	0.008	0.01	0.01	0.03	0.01	0.02	0.005	0.004	0.01	0.03
7	0.15	0.03	0.24	0.12	0.43	0.32	0.44	0.37	–	–	0.15	0.17	0.34	0.35
8	–	–	0.03	0.02	0.04	0.03	0.01	0.01	–	–	0.01	0.01	0.02	0.02
9	0.02	0.02	0.01	0.01	0.02	0.07	0.01	0.006	–	–	0.004	0.002	0.02	0.01
10	0.001	0.001	0.003	0.004	0.004	0.004	0.002	0.003	0.003	0.003	0.002	0.002	0.002	0.003
11	0.09	0.11	0.05	0.03	0.04	0.03	0.04	0.04	0.08	0.08	0.03	0.02	0.05	0.05
12	0.01	0.01	0.02	0.03	0.01	0.004	0.01	0.02	0.01	0.003	0.007	0.005	0.01	0.02
13	0.58	–	0.08	0.06	0.11	0.17	0.09	0.08	–	–	0.03	0.02	0.10	0.10
14	0.01	0.01	0.04	0.09	0.05	0.05	0.05	0.13	–	–	0.03	0.03	0.04	0.03
15	0.19	0.22	0.92	1.02	1.56	3.79	0.11	0.10	0.89	0.33	0.10	0.09	0.78	2.1
16	0.02	0.01	0.02	0.02	0.05	0.03	0.07	0.23	0.04	–	0.02	0.01	0.04	0.12

a From Esmen *et al.* (1979a)

Table 19 Distribution of measured average employee exposure to fibres, expressed as cumulative percentage of samples less than stated concentrations, in 16 facilities in the USA[a]

Plant	Average concentration (fibres/cm^3)										
	⩽0.005	⩽0.01	⩽0.05	⩽0.1	⩽0.5	⩽1	⩽1.5	⩽2	⩽5	⩽10	⩽20
1	6.1	11.2	72.4	83.7	98.0	98.0	99.0	100			
2	0	0	32.7	63.6	96.4	100					
3	40.8	50.7	87.3	95.8	97.2	100					
4	6.7	27.8	78.9	93.3	100						
5	3.3	13.1	91.8	98.4	100						
6	47.7	76.6	97.3	98.2	100						
7	0	0	3.2	14.3	81.0	92.1	100				
8	17.1	40.0	89.5	99.0	100						
9	16.7	42.2	95.5	98.9	100						
10	82.5	97.9	100								
11	3.9	13.7	66.7	84.3	100						
12	33.2	65.4	98.6	99.1	100						
13	1.3	6.3	60.8	75.9	97.5	100					
14	6.0	27.7	89.2	97.6	98.8	100					
15	0	0	9.3	28.0	69.3	81.3	89.3	93.3	97.3	98.6	100
16	7.8	20.0	87.8	94.4	98.9	100					

a From Corn (1979); samples analysed by phase-contrast microscopy, including fibres ⩽3 µm in diameter

Table 20 Concentration (fibres/cm^3) of fibres <1 µm in diameter in 16 facilities in the USA[a]

Plant[b]	Forming		Production		Manufacturing		Maintenance		Quality control		Shipping		Overall	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
1	0.002	0.001	0.73	2.3	0.01	0.007	0.01	0.03	0.45	0.76	0.002	0.002	0.004	0.17
2	0.04	0.03	0.04	0.02	0.03	0.03	0.04	0.04	0.07	0.08	0.01	0.01	0.04	0.03
3	–	–	0.03	0.03	–	–	0.20	0.71	–	–	0.005	0.003	0.08	0.42
4	0.01	0.005	0.19	0.29	0.12	0.42	0.02	0.02	0.02	0.01	0.009	0.005	0.10	0.28
5	0.01	0.01	0.01	0.007	0.004	0.002	0.01	0.005	0.02	–	0.01	0.002	0.01	0.01
6	0.002	0.001	0.004	0.004	0.004	0.003	0.008	0.03	0.007	0.01	0.004	0.004	0.005	0.02
8	–	–	0.05	0.04	0.02	0.01	0.02	0.02	–	–	0.02	0.01	0.03	0.03
10	0.003	0.002	0.003	0.002	0.004	0.002	0.01	0.01	0.002	0.002	0.002	0.002	0.004	0.007
11	0.12	0.09	0.02	0.02	0.02	0.03	0.01	0.01	0.01	0.01	0.01	0.01	0.03	0.06
12	0.006	0.005	0.004	0.005	0.003	0.003	0.002	0.001	0.005	0.004	0.003	0.001	0.003	0.004
13	0.04	–	0.03	0.03	0.03	0.04	0.02	0.02	–	–	0.01	0.01	0.02	0.04
15	2.0	2.6	6.49	9.37	5.25	14.6	1.3	2.2	12.0	5.83	0.58	0.38	4.4	9.9
16	0.03	0.02	0.04	0.03	0.07	0.04	0.22	0.84	0.03	–	0.01	0.007	0.01	0.04

a From Esmen *et al.* (1979a)

36 Transmission electron microscopic data are not reported for facilities 7, 9 and 14 because the analytical method used was less reliable than that at other plants.

Table 21 Concentrations (fibres/cm^3) of total fibres in one rockwool and one slagwool production plant in the USA[a]

Dust zone	No. of samples	Total fibres (fibres/cm^3)	
		Average	Range
Rockwool			
Warehouse	3	1.4	1.1–1.7
Mixing-Fourdrinier ovens	3	0.14	0.13–0.18
Panel finishing	12	0.40	0.13–1.3
Figre forming	10	0.20	0.07–0.65
Erection and repair	13	0.24	0.04–1.1
Tile finishing	22	0.31	0.10–0.74
All samples	63	0.34	0.04–1.7
Slagwool			
Maintenance	15	0.08	0.01–0.24
Block production	8	0.05	0.02–0.11
Blanket line	5	0.05	0.02–0.09
Boiler room	2	0.05	0.04–0.07
Yard	2	0.09	0.05–0.13
Ceramic block	7	0.42	0.11–0.95
Shipping	3	0.04	0.02–0.06
Main plant	11	0.01	0.006–0.58
Mould formation	19	0.03	0.005–0.08
All samples	72	0.10	0.005–0.95

a From Corn *et al.* (1976); as determined by phase-contrast microscopy

Table 22 Airborne concentrations of total and respirable fibres in three ceramic fibre production plants in the USA[a]

Dust zone	Total respirable fibres[b] (fibres/cm^3)	Total fibres[b] (fibres/cm^3)	Total fibres[c] (fibres/cm^3)	Respirable fraction[d]
Plant A (all)	2.6	3.3	2.6	0.79
Finishing	2.1	2.6	1.9	0.82
CVF[e]	4.2	5.2	4.3	0.80
Lines 1 and 2	0.94	1.1	0.73	0.83
Lines 3 and 4	0.08	0.09	0.04	0.89
OEM[f]	6.9	8.7	7.6	0.79
Maintenance	0.50	0.64	0.52	0.79
GFA[g]	0.53	0.80	0.74	0.66
Shipping	0.27	0.34	0.22	0.78
Quality control	0.11	0.15	0.11	0.71
Plant B (all)	1.4	1.5	0.63	0.92
Textile	0.88	1.1	0.62	0.79
Maintenance	0.95	1.0	0.27	0.96
Furnace	1.5	1.6	0.60	0.96
Process	2.4	2.6	1.1	0.95
Quality control	0.62	0.68	0.33	0.92
Plant C (all)	0.21	0.23	0.05	0.91
Maintenance	0.12	0.12	0.01	0.98
Fiberizing	0.22	0.23	0.04	0.96
Felting	0.02	0.24	0.10	0.82
Pressing	0.23	0.26	0.08	0.89
Finishing	0.26	0.28	0.06	0.93
Fibre cleaning	0.06	0.07	0.01	0.94
Mixing	0.02	0.03	0.01	0.93
Shipping	0.04	0.05	0.03	0.84
Job centre	0.22	0.23	0.04	0.94

a From Esmen *et al.* (1979b)

b As determined by both electron and optical microscopy, including fibres <5 µm in diameter

c As determined by optical microscopy only

d Total respirable concentration/total fibre concentration

e CVF, bulk fibre mixed with colloidal silica and vacuum formed

f OEM, some products from CVF trimmed with hand saws, drilled and packaged

g GFA, blankets from line 2 cut by hand into specific shapes

Table 25 Fibre concentrations (fibres/cm^3)[a] in combined occupational groups at three European continuous glass filament plants (1977–1980)[b]

Combined occupational group	Plant E			Plant J			Plant N		
	No.	Mean	Range	No.	Mean	Range	No.	Mean	Range
Preproduction	12	0.004	0.001–0.015	–			6	0.009	0.005–0.017
Production I	54	0.002	0.001–0.012	19	0.001	0.001–0.003	44	0.007	0.001–0.039
Production II	–			32	0.001	0.001–0.003	22	0.023	0.005–0.112
Maintenance	16	0.005	0.001–0.022	–			15	0.014	0.006–0.023
General	2	0.005	–	11	0.001	0.001–0.003	7	0.012	0.008–0.020
Secondary process 1	70	0.002	0.001–0.016	87	0.002	0.001–0.006	27	0.007	0.005–0.017
Secondary process 2	–			–			6	0.022	0.006–0.056
Research and development	10	0.002	0.001–0.003	–			–		
Plant mean and range (mg/m^3)	145	1.4	0.1–38	132	0.6	0.03–2.7	115	0.9	0.1–2.7

a Including fibres ⩽3 µm in diameter

b From Ottery *et al.* (1984)

Table 26 Fibre distributions of glasswool and rockwool in two Danish rockwool plants[a]

Site	No. of samples	Diameter			Length	
		Geometric mean (µm)	Geometric SD		Geometric mean (µm)	Geometric SD
Rockwool plant A	6	0.95	3.1		13	3.4
Rockwool plant B	38	0.99	3.3		14	3.6
Rockwool, special fibres	6	1.46	2.8		27	3.3
Rockwool, conventional fibres	28	1.73	2.4		22	3.1
Use of rockwool	21	1.20	2.7		22	4.0
Use of glasswool	8	0.75	2.8		16	3.5

a From Schneider *et al.* (1985)

Table 27 Respirable fibre concentrations (fibres/cm^3) in glasswool and rock-wool production plants in Sweden (1978–1981)[a]

Combined occupational group	Three rockwool plants				Two glasswool plants		
	No.	Mean	Range		No.	Mean	Range
Production	90	0.20	0.051–1.9		49	0.22	0.056–0.65
Maintenance	64	0.21	0.031–1.2		89	0.36	0.037–5.3
General	45	0.15	0.031–0.34		34	0.19	0.034–0.53
Secondary process 1	35	0.23	0.058–0.52		59	0.19	0.038–0.73
Secondary process 2	2	0.21	0.15–0.27		5	0.13	0.083–0.16
Cleaning	105	0.32	0.025–2.6		76	0.21	0.026–1.0
Miscellaneous	19	0.20	0.031–0.66		15	0.11	0.014–0.49
Overall mean[b]		0.22				0.20	

a Including fibres ⩽3 µm in diameter; arithmetic means and ranges were computed by the Working Group from data on individual samples taken from the Swedish reports (Arbetarskyddsstyrelsen, 1981).

b Computed by the Working Group as average over occuaptional group mean

Table 23 Fibre concentrations (fibres/cm^3)[a] in combined occupational groups in four European glasswool plants (1977–1980)[b]

Combined occupational group	Plant 7			Plant 2			Plant 6			Plant 10		
	No.	Mean	Range	No.	Mean	Range	No.	Mean	Range	No.	Mean	Range
Preproduction	5	0.01	0.02–0.01	8	0.01	<0.01–0.01	5	0.01	0.01	5	0.01	<0.01–0.03
Production	39	0.05	0.01–0.62	26	0.01	<0.01–0.03	27	0.03	0.01–0.11	61	0.05	<0.01–0.22
Maintenance	20	0.07	0.01–0.06	4	0.03	0.01–0.06	12	0.04	<0.01–0.17	27	0.02	<0.01–0.06
General	15	0.03	0.01–0.06	10	0.02	0.01–0.04	10	0.02	0.01–0.04	12	0.03	<0.01–0.06
Secondary process 1	37	0.04	0.01–0.11	32	0.05	0.01–0.21	26	0.03	<0.01–0.07	36	0.02	<0.01–0.06
Secondary process 2	23	1.00	0.17–4.02	–			2	0.07	0.05–0.09	45	0.16	0.02–1.39
Cleaning	–			–			4	0.01	0.01–0.02	–		
Overall mean[c]		0.2			0.02			0.03			0.05	
Plant mean and range (mg/cm^3)[d]	124	1.3	0.2–21	69	0.6	0.1–2.7	79	1.2	0.1–20	168	1.3	0.15–21

a Including fibres ⩽3 µm in diameter

b From Cherrie *et al.* (1986)

c Computed by the Working Group as average over occupational group mean

d From Ottery *et al.* (1984)

Table 24 Fibre concentrations (fibres/cm^3)[a] in combined occupational groups in six European rockwool plants (1977–1980)[b]

Combined occupational group	Plant 1			Plant 5			Plant 4			Plant 3			Plant 8			Plant 9		
	No.	Mean	Range	No.	Mean	Range	No.	Mean	Range	No.	Mean	Range	No.	Mean	Range	No.	Mean	Range
Preproduction	8	0.08	0.01–0.22	2	0.01	0.01	7	0.03	0.01–0.07	3	0.06	0.03–0.11	1	0.04	0.04	4	0.01	<0.01–0.01
Production	36	0.10	0.02–0.37	22	0.06	0.02–0.14	27	0.06	0.02–0.19	28	0.12	0.03–0.32	19	0.05	0.01–0.13	51	0.05	0.01–0.16
Maintenance	9	0.08	0.05–0.18	12	0.05	0.01–0.14	20	0.05	0.02–0.12	8	0.05	0.03–0.10	9	0.03	0.01–0.07	10	0.04	0.01–0.11
General	16	0.08	0.02–0.37	7	0.04	0.03–0.07	13	0.06	0.02–0.09	8	0.07	0.04–0.14	2	0.04	0.04	23	0.06	0.01–0.36
Secondary process 1	32	0.10	0.03–0.21	16	0.07	0.01–0.15	28	0.08	0.03–0.33	11	0.12	0.06–0.23	24	0.08	0.01–0.20	55	0.06	0.02–0.39
Secondary process 2	11	0.40	0.09–1.40	–			–			3	0.34	0.25–0.41	3	0.25	0.19–0.36	22	0.67	0.06–1.37
Cleaning	–			5	0.09	0.04–0.11	8	0.06	0.02–0.14	4	0.13	0.05–0.29	8	0.09	0.01–0.18	12	0.14	0.02–0.44
Overall mean[c]		0.14			0.05			0.06			0.13			0.08			0.15	
Plant mean and range (mg/m3)[d]	101	2.3	0.3–26	53	1.0	0.2–4.7	86	1.1	0.3–3.5	56	1.6	0.4–4.0	60	1.0	0.06–2.3	164	0.7	0.03–4.0

a Including fibres ⩽3 µm in diameter

b From Cherrie *et al.* (1986)

c Computed by the Working Group as average over occupational group mean

d From Ottery *et al.* (1984)

Table 28 Concentrations of total airborne dust and respirable fibres in insulation wool production plants in the UK (period not stated)[a]

Fibre type	Mean total dust				Mean respirable fibre		
	No. of samples	Concentration (mg/m^3)	Range		No. of samples	Concentration (fibres/ cm^3)[b]	Range
Glass fibre	32	11.1	0.7–78.2		50	0.31	0.02–1.10
Glass fibre	16	4.1	0.5–14.3		35	0.27	0.01–0.79
Glass fibre	30	8.9	0.4–51.3		67	0.12	0.003–0.85
Rock-/slagwool	22	6.5	0.7–16.2		55	0.89	0.03–10.3
Ceramic fibre	16	8.3	0.2–26.3		45	1.27	0.06–6.14
Alumina fibre	15	4.9	0.3–13.4		33	1.09	0.03–5.82

a From Head & Wagg (1980)

b Including fibres ⩽3 µm in diameter

Table 29 Concentrations and dimensions of airborne fibres from various operations using fibrous glass insulation[a]

Operation	Parent material (mean fibre diameter, µm)	Breathing zone air samples	
		Fibres/cm^3	Mean fibre diameter (µm)
Duct wrapping	5.3	1.26	4.7
	6.3	0.90	4.0
	6.4		
	4.0	0.51	3.4
		0.79	3.6
	4.1	1.40	2.6
		1.33	2.3
	7.5	0.80	2.5
	5.8	1.20	6.2
	5.5	2.34	5.0
	7.2	0.53	7.4
Wall and plenum insulation	10.2	3.26	8.4
	8.1	4.18	3.5
	7.6		
	7.8	8.08	3.8
	8.1		
Pipe insulation	8.5	0.93	3.1
		0.48	4.1
	6.7	0.57	3.4
	6.0		
	6.0		
	5.6		
Fan housing insulation	6.9	1.57	3.5

a From Fowler *et al.* (1971)

Table 30 Airborne concentrations of respirable fibres[a] in the final preparation and installation of man-made mineral fibre insulation, as determined by a combination of phase-contrast and electron microscopic techniques[b]

Product and job classification	No. of samples	Fibre concentration (fibres/cm^3)		
		Average	Range	Average respirable fractions[c]
Acoustical ceiling installer	12	0.003	0–0.006	0.55
Duct installation				
Pipe covering	31	0.06	0.007–0.38	0.82
Blanket insulation	8	0.05	0.025–0.14	0.71
Wrap around	11	0.06	0.03–0.15	0.77
Attic insulation				
Fibrous glass				
Roofer	6	0.31	0.07–0.93	0.91
Blower	16	1.8	0.67–4.8	0.44
Feeder	18	0.70	0.06–1.48	0.92
Mineral wool				
Helper	9	0.53	0.04–2.03	0.71
Blower	23	4.2	0.50–14.8	0.48
Feeder	9	1.4	0.26–4.4	0.74
Building insulation installer	31	0.13	0.013–0.41	0.91
Aircraft insulation				
Plant A				
Sewer	16	0.44	0.11–1.05	0.98
Cutter	8	0.25	0.05–0.58	0.98
Cementer	9	0.30	0.18–0.58	0.94
Isolated jobs	7	0.24	0.03–0.31	0.99
Plant B				
Sewer	8	0.18	0.05–0.26	0.96
Cutter	4	1.7	0.18–3.78	0.99
Cementer	1	0.12	–	0.93
Isolated jobs	3	0.05	0.012–0.076	0.94
Fibrous glass duct				
Duct fabricator	4	0.02	0.006–0.05	0.66
Sheet-metal worker	8	0.02	0.005–0.05	0.65
Duct installer	5	0.01	0.006–0.20	0.87

a ⩽3 µm in diameter

b From Esmen *et al.* (1982)

c Arithmetic mean of respirable fibre concentration/total fibre concentration

Table 31 Concentrations of total dust and respirable fibres[a] during insulation in Sweden (1979–1980)[b]

Operation	Total dust (mg/ m^3)		Respirable fibres/cm^3	
	Mean[c]	Range	Mean[c]	Range
Attic insulation, existing buildings	11.6	1.7–21.7	1.11	0.1–1.9
Insulation of new buildings	2.63	0.5–11.1	0.57	0.07–1.8
Technical insulation	3.14	0.4–25	0.37	<0.01–1.39
Acoustical insulation	1.8	1.7–1.9	0.15	0.11–0.18
Spraying	13.5	1.3–43.7	0.51	0.13–1.1
Hanging fabric	4.18	3.6–5.2	0.60	0.30–0.76

a ⩽3 µm in diameter

b From Hallin (1981)

c Calculated by the Working Group

Table 32 Concentrations of total dust and respirable fibres[a] during insulation in Denmark[b]

Operation	Total dust (mg/ m^3)		Respirable fibres/cm^3	
	Mean	Range	Mean	Range
Attic insulation, existing buildings	26.8	1.5–134	0.89	0.04–3.5
Insulation of new buildings	12.6	0.22–44	0.10	0.04–0.17
Technical insulation	7.1	1.8–12.8	0.35	0.03–1.6
Application in industrial products	0.88	0.83–0.91	0.05	0.01–0.11
Hot-house substrate	3.00	0.61–3.9	0.06	0.03–0.09

a <3 µm in diameter

b From Schneider (1984)

Table 33 Concentrations of total dust and respirable fibres[a] in breathing zone and static samples during insulation and during application of ceramic fibres[b]

Product	Total dust (mg/m^3)			Respirable fibres/cm^3		
	No. of samples	Mean	Range	No. of samples	Mean	Range
Construction insulation						
Domestic loft						
Blankets	9	35.6	8.2–90	12	0.70	0.24–1.76
Loose fill	4	30.9	5.0–59.7	6	8.19	0.54–20.9
Fire protection	9	16.6	1.9–51.5	22	0.77	0.16–2.57
Industrial product insulation (one plant)	4	0.8	0.6–1.0	12	0.10	0.02–0.36
Ceramic fibres in manufacture and use of high-temperature insulation and ceramic mouldings	6	1.5	0.7–5.2	11	0.55	0.09–0.87
Alumina fibres in manufacture of stack block insulation and engine silencer insulation	11	10.3	1.5–22.9	30	1.9	0.35–5.64

a ⩽3 µm in diameter

b From Head & Wagg (1980)

Table 34 Concentrations of total dust and respirable fibres[a] during the use of fine-diameter, special-purpose glass fibres

Exposure	Total dust (mg/m^3)				Respirable fibres/cm^3			Reference
	No. of samples	Mean	Range		No. of samples	Mean	Range	
Production of glass fibre		–				10.1	1.6–44.1[b]	Schneider (1984)
paper	28	1.1	0.2–4.3		44	1.54	0.09–18.8[c]	Head & Wagg (1980)
Production of air filters	5	0.4	0.07–1.0		34	0.33	0.02–2.55[c]	Head & Wagg (1980)
Aircraft insulation						4.6	0.4–24.4[b]	Schneider (1984)
Aircraft insulation		0.38	0.04–1.49			0.41	0.012–3.78[d]	Schneider (1984)

a ⩽3 µm in diameter

b Total fibre concentration by optical microscopy; average diameter distribution, no less than 89% of fibres <3.8 µm

c Phase-contrast optical microscopy

d Combined optical and electron microscopy

Table 35 Concentrations of total dust and fibres <3 µm diameter during use of special-purpose fibres[a]

Exposure	Mean concentrations of total dust (mg/m^3)	Total fibres/cm^3 (mean counts)[b]
Manufacture of glass fibre paper	0.47–2.28	2.9–13
Conversion of glass fibre paper	0.17–0.49	0.53–15.1
Manufacture of refractory fibres	0.83–4.0	0.49–9.2
Use of refractory fibres	–	2.7–17.1

a From UK Factories Inspectorate (1987), personal samples

b Determined by transmission electron microscopy

Table 36 Ranges of airborne fibre concentrations in typical exposure situations

Fibre concentration (fibres/cm^3)	Location/use	Reference
Ultralow(<0.0001)[a]	Outdoor: rural area	Höhr(1985)
	Buildings: thermal insulation	
Extremely low (0.0001–0.001)[a]	Outdoor: large cities	Höhr (1985)
	Buildings: ceiling boards	Rindel *et al.* (1987)
	Ventilation systems	Balzer (1976)
Very low (0.001–0.01)		
Glass continuous filament	Production and use	Cherrie *et al.* (1986)
Coarse glass fibre	Production and use	
Ceiling boards	Buildings: some damage, some ventilation ducts	Schneider (1986)
Low (0.01–0.1)		Höhr (1985)
Glasswool	Production and most secondary production	
Rockwool	Production and most secondary production	
Rock-/ slagwool	Production and most secondary production	
Ceiling boards	Buildings: severe damage	Schneider (1976)
Medium (0.1–1.0)		
Fine glass fibre	Production	Höhr (1985)
Rockwool	Some secondary production and user industry	
Ceramic	Primary production and user industry	
Glasswool	User industry	
High (>1.0)		
Very fine glass fibre	Production and use	
Glass-/rockwool, loose	User industry: blowing into attic	
Glass-/rockwool, without dust suppressants	Production and use	
Ceramic	Secondary production and some user industry	

a Estimated from transmission electron microscopic measurements

Table 37 Fibre[a] concentrations in ambient air in the Federal Republic of Germany in 1981–1982[b]

Measuring site	No. of samples	Fibre/cm^3					Size of glass fibres[c] (µm)	
		Total	Chrysotile	Amphibole	Glass		CMD	CML
Duisburg	17	0.041	0.0022	0.0019	0.00050		0.26	2.54
Dortmund	6	0.036	0.0026	0.0019	0.00170		0.25	3.06
Düsseldorf	21	0.027	0.0014	0.0013	0.00040		0.30	3.64
Krahm (rural area)	9	0.012	0.0005	0.0007	0.00004		0.89	2.76

a Not reported in this table are fibres classified as quartz, aluminium, iron, rutile, sulphur or others.

b From Höhr (1985)

c CMD, count median diameter; CML, count median length

Table 38 Mean dust and fibre concentrations in schools, nursery schools and offices in Denmark[a]

Type of institution[b]	No. of institutions	Respirable man-made mineral fibres (fibre/ cm^3)[c]	Nonrespirable man-made mineral fibres (fibre/cm^3)[c]	Other respirable fibres (including organics) (fibre/ cm^3)	Other nonrespirable fibres (including organics) (fibre/cm^3)
A(1984)	10	0.0001	0.000 02	0.18	0.013
B(1984)	6	0.0001	0.000 04	0.15	0.011
C(1984)	8	0.000 04	0–0.000 08	0.17	0.012
D (not stated)		0.000 07	–	0.017	0.0007

a From Schneider (1986); Rindel *et al.* (1987)

b A, ceilings made of man-made mineral fibre with water-soluble binder; B, ceilings made of man-made mineral fibre with resin binder; C, without readily visible man-made mineral fibre products; D, mechanically-ventilated schools

c Several results were below the detection limit of 0.000 04-0.000 08 fibre/ cm^3 and were calculated using statistical procedures

Table 41 Summary table of studies used for evaluation of the carcinogenicity of man-made mineral fibres in experimental animals (the studies of Stanton *et al.* (1977, 1981) are summarized separately in Tables 39 and 40)

Substance	Fibre dimensions: length (L), diameter (D)	Dosing schedule		Length of observation	No. of animals examined	No. of animals with tumours[a]	Histological type[b]	Median or average survival time [weeks]
		Cumulative exposure [mg/m^3 × h]	Duration of exposure					
Inhalation exposure to glasswool and glass fibres								
Inhalation exposure to respirable dust concentrations of 5 mg/m^3 (Wislar IOPS AF/Han rats, equal numbers of females and males 8–9 weeks old) (Le Bouffant et al., 1984)								
Glasswool	L 42%<10 µm d 69%<1 µm	–	5 h/day, 5 days/week, total length of dust-	Up to 28 months (several animals killed	45	1	Ca	–
Glasswool	L 97% <5 µm D 43%<0.1 µm	–	ing: half the animals, 12 months, the other	at 12, 16 and 24 months)	48	0	–	–
Chrysotile (Canadian)	L 6% >5 µm	–	half, 24 months		47	9	Pulmonary tumours	–
Controls	–	–			47	0	–	–
Inhalation exposure to respirable dust concentrations of 10 mg/m^3 (PSF Fischer 344 rats, equal numbers of females and males, 7–8 weeks old) (McConnell et al., 1984)								
Glasswool	Not given	9 035	7 h/day, 5 days/ week,	Lifetime (several	55	0	–	–
UICC Chrysotile (Canadian)		14 559	12 months	animals killed at 3, 12 and 24 months)	56	11	4 A, 7 AdCa	–
Controls	–	–			53	3	1 A, 2 AdCa	–
Inhalation exposure to respirable dust concentrations of 10 mg/m^3 (SPF Fischer rats, equal numbers of females and males) (Wagner et al., 1984)								
Glasswool plus resin	L 72% 5-20 µm d 52%⩽ µm	17 498	7 h/day, 5 days/week, 12 months	Lifetime (some animals killed at 12 and 24	48	1	AdCa	–
Glasswool without resin	L 58% 5-20 µm d 47%⩽ µm	17 458		months)	47	1	A	–
US glasswool	L 93% 5-20 µm d 97%⩽ µm	17 510			48	1	AdCa	–
UICC Chrysotile	L 39%>10 µm	17 499			48	12	1 A, 11 AdCa	–
(Canadian)	D 29% >0.5 µm							
Controls					48	0	–	–
Nose-only inhalation exposure to dust clouds of various glass fibres (female Osborne-Mendel rats, 100 days old) (Smith et al., 1987)								
		Dust cone.						
Glasswool	L g. mean, 4.9 µm d g. mean, 0.4 µm	2.4 mg/m^3 (3000 f/cm^3)	6 h/day, 5 days/week, 2 years	Lifetime	57	0	–	110
Glasswool	L g. mean, 4.9 µm d g. mean, 0.4 µm	0.24 mg/m^3 (300 f/cm^3)			57	0	–	108
Glasswool (blowing wool)	L g. mean, 24 µm d g. mean, 1.2 µm	4.4 mg/m^3 (100 f/ cm^3)			52	0	–	115
Nose-only inhalation (Smith et al., 1987) (contd)								
Glasswool (building insulation)	L g. mean, 20 µm d g. mean, 1.1 µm	9.9 mg/m^3 (100 f/cm^3)	6 h/day, 5 days/week, 2 years	Lifetime	57	0	–	94

Substance	Fibre dimensions: length (L), diameter (D)	Dosing schedule		Length of observation	No. of animals examined	No. of animals with tumours[a]	Histological type[b]	Median or average survival time [weeks]
		Cumulative exposure [mg/m^3 × h]	Duration of exposure					
Glasswool (building insulation)	L g. mean, 20 µm d g. mean, 1.1 µm	1 mg/m^3 (10 f/m^3)			61	0	–	104
Glasswool (binder-coated)	L g. mean, 80 µm d g. mean, 3.0 µm	7.0 mg/m^3 (25 f/cm^3)			58	0	–	100
UICC crocidolite	L 5%>5 µm	3000 f/cm^3			57	3	1 M, 2 BT	109
Chamber controls	–	–			59	0	–	108
Room controls	–	–			125	0	–	103
Nose-only inhalation exposure to glass fibres in concentrations of 3 mg/m^3 (female Wistar rats, 12 weeks old) (Muhle et al., *1987)*								
Glasswool	L 50% <4.8 µm d 50% <0.42 µm	3 000	5 h/day, 4 days/week, 1 year	140 weeks	107	1	ScCa	110
Glasswool with SO_2	L 50% <4.8 µm d 50% <0.42 µm	3 000			108	1	A	106
Crocidolite (S. Africa)	L 50%>1.5 µm d 50% >0.27 µm	2 200			50	1	AdCa	111
Chrysotile (Calidria)	L 50% >6.0 µm d 50% >0.67 µm	6 000			50	0	–	109
SO_2	–	–			50	0	–	99
Clean air	–	–			55	0	–	108
No treatment					50	0	–	108
Nose-only inhalation exposure to dust clouds of various glass fibres (male Syrian golden hamsters, 100 days old) (Smith et al., *1987*)								
		Dust cone.						
Glasswool	L g. mean, 4.9 µm d g. mean, 0.4 µm	2.4 mg/m^3 (3000 f/cm^3)	6 h/day, 5 days/week, 2 years	Lifetime	69	0	–	95
Glasswool	L g. mean, 4.9 µm d g. mean, 0.4 µm	0.24 mg/m^3 (300 g/cm^3)			70	0	–	95
Glasswool (blowing wool)	L g. mean, 24 µm d g. mean, 1.2 µm	4.4 mg/m^3 (100 f/cm^3)			60	0	–	85
Glasswool (building insulation)	L g. mean, 20 µm d g. mean, 1.1 µm	9.9 mg/m^3 (100 f/cm^3)			66	0	–	90
Glasswool (building insulation)	L g. mean, 20 µm d g. mean, 1.1 µm	1 mg/m^3 (10 f/cm^3)			65	0	–	97
Glasswool (binder-coated	L g. mean, 83 µm d g. mean, 3.0 µm	7.0 mg/m^3 (25 g/cm^3)			61	0	–	93(1) 88(2)
UICC crocidolite	L, 7% >5 µm	3000 f/cm^3			58	0	–	78
Chamber controls	–	–			58	1	BT	95
Room controls	–	–			112	0	–	80
Inhalation exposure to respirable dust concentrations of 5.8 mg/m^3 glass fibres or 13.45 mg/m^3 crocidolite (male baboons, 6–8 kg) (Goldstein et al., *1983)*								
Glass fibres	L >60% <6.3 µm d>70%<1.0 µm		7 h/day, 5 days/week, up to 35–40 months	Up to 6–7 months after the end of dusting	10	0	–	Not given
UICC crocidolite	L <25% >3.0 µm d<20%>0.5 µm				10	0	–	Not given

Substance	Fibre dimensions: length (L), diameter (D)	Dosing schedule		Length of observation	No. of animals examined	No. of animals with tumours[a]	Histological type[b]	Median or average survival time [weeks]
		Total dose [mg]	No. of applications					
Intratracheal administration of glasswool and glass fibres								
Intratracheal instillation in 0.3 ml saline (female Wistar rats, 11 weeks old) (Pott et al., *1987)*								
Glasswool	L 50% <3.2 µm D 50%<0.18 µm	10	20	126 weeks	34	5	1 A, 2 AdCa, 2 ScCa, 1 T	107
Crocidolite (S. Africa)	L 50%>2.1 µm d 50% >0.20 µm	10	20	126 weeks	35	15	9 AdCa, 2 ScCa, 4 mixed, **1 T**	126
Saline	–	–	20	124 weeks	40	0	–	115
Intratracheal instillation of glass fibres in 0.2 ml saline (female Osborne-Mendel rats, 100 days old) (Smith et al., *1987)*								
Glasswool	L g. mean, 4.7 µm d g. mean, 0.4 µm	10	5	Lifetime	22	0	–	112
UICC crocidolite	L 5% >5 µm	10	5		25	2	2BT	91
Saline	–	–			25	0	–	98
No treatment	–	–			125	0	–	103
Intratracheal instillation in 0.15 ml saline (male Syrian golden hamsters) (Pott et al., *1984a) [age unspecified]*								
Glasswool 2-h milled	L 50% <7.0 µm d 50% <0.3 µm	8	8	113 weeks	136	48	5 Ca, 37 M, 6S	>104
Glasswool 4-h milled	L 50% <4.2 µm d 50% <0.3 µm	8	8		138	38	6 Ca, 26 M, 6S	>104
UICC crocidolite	L 50%>2.1 µm d 50% >0.2 µm	8	8		142	18	9 Ca, 8 M, 1 S	>104
Titanium dioxide	Granular	8	8		135	2	1 S	>104
Intratracheal instillation in 0.2 ml 0.005% gelatin solution in saline (female and male Syrian golden hamsters, 16 weeks old) (Feron et al., *1985)*								
Benzo[*a*]pyrene (BaP)		26	26	85 weeks	63	7	4 P, 2 Ca, 1 S	No relevant difference in mortality between any of the treatment groups and the control group
Glasswool	L 58% <5 µm D 88%<1 µm	26	(once every 2 weeks for 52 weeks)		64	0		
Glasswool with BaP	L 58% <5 µm D 88% <1 µm	26 + 26			66	4	2P, 2S	
Crocidolite	L 58% >5 µm D 63% >0.25 µm	26			60	0	–	
Crocidolite with BaP	L 58% >5 µm D 63% >0.25 µm	26 + 26			52	4	1 P, 2 Ca, 1 S	
Saline	–	–			59	0	–	
Intrapleural administration of glasswool and glass fibres								
Intrapleural injection in 0.5 ml distilled water (BALB/c mice) (Davis, 1976) [sex, age unspecified]								
Glass fibre (borosilicate)	L several hundred µm	10	1	Up to 18 months	25	0	–	–
Glass fibre (borosilicate)	D average, 0.05 µm L <20 µm D average, 0.05 µm	10	1		25	0	–	–
Glass fibre (borosilicate)	L several hundred µm	10	1		25	0		
Glass fibre	D average, 3.5 µm L <20 µm	10	1		25	0	–	–

Substance	Fibre dimensions: length (L), diameter (D)	Dosing schedule		Length of observation	No. of animals examined	No. of animals with tumours[a]	Histological type[b]	Median or average survival time [weeks]
		Total dose [mg]	No. of applications					
	D average, 3.5 µm							
Asbestos (chrysotile + crocidolite)	Not given	Not given			150	2	2M	
Intrapleural injection in 0.4 ml saline (SPF Wistar rats, twice as many males as females, 13 weeks old) (Wagner et al., 1973)								
Glass fibre (borosilicate)	L 60% >20 µm D 30% 1.5–2.5 µm	20	1	Lifetime	35	0	–	111
SFA chrysotile		20	1		36	23	23 M	81
SFA chrysotile		20	1		32	21	21 M	91
Glass powder	Granular, D < 8 µm	20	1		35	1	1 M	107
Intrapleural injection in 0.4 ml saline (Wistar rats, equal numbers of males and females, 10 weeks old) (Wagner et al., 1976)								
Glasswool	L median, 1.7 µm D median, 0.12 µm	20	1	Lifetime	32	4	4M	102
Glasswool	L median, 22 µm d median, 1.8 µm	20	1		32	0	–	103
Saline			1		32	0		100
Intrapleural injection in 2 ml saline (male SPF Sprague-Dawley rats, 12 weeks old) (Monchaux et al., 1981)								
Glasswool	L mean, 5.89 µm d mean, 0.229 µm	20	1	Lifetime	45	6	6M	73
UICC chrysotile A	L mean, 3.21 µm D mean, 0.063 µm	20	1		33	15	1 Ca, 14 M	55
UICC crocidolite	L mean, 3.14 µm d mean, 0.148 µm	20	1		39	21	21 M	65
Saline	–	–	1		32	0	–	67
Intrapleural injection in 0.5 ml saline (SPF Sprague-Dawley rats) (Wagner et al., 1984) [sex, age unspecified]								
Glasswool with resin	L 70% ⩽5 µm	20	1	Lifetime	48			–
(English)	D 85% ⩽1 µm					1	1 M	
Glasswool without resin (English)	L 57% ⩽5 µm D 85% ⩽1 µm	20	1		48			
US glasswool	L 88% ⩽5 µm D 98.5% ⩽1 µm	20	1		48	4	4M	–
UICC African chrysotile A		20	1		48	6	6M	–
Saline			1		24	0		
Intraperitoneal injection of glasswool in 2 ml saline (female Wistar rats, 8-12 weeks old) (Pott et al., 1976)								
Glasswool (very fine)	L 59% <3 µm	2	1	Lifetime	34	1	1 M	74
	D 50% <0.4 µm	10	1		36	4	2 M, 2 S	73
		100	4 (weekly)		32	23	20 M, 3 S	43
Glasswool (finer)	L 50%<10 µm	2	1		73	20	17 M, 3 S	96
	D 50% <0.2 µm	10	1		77	41	36 M, 4 S, 1 Ca	87
		50	2 (weekly)		77	55	47 M, 8 S	52
Glasswool (coarser)	L 50% <30 µm D 50%<1.0 µm	20	1		37	14	12 M, 1 S, 1 Ca	87
UICC chrysotile A		2	1		37	6	4 M, 2S	67

Substance	Fibre dimensions: length (L), diameter (D)	Dosing schedule		Length of observation	No. of animals examined	No. of animals with tumours[a]	Histological type[b]	Median or average survival time [weeks]
		Total dose [mg]	No. of applications					
		6.25	1		35	27	24 M, 3 S	70
		25	1		31	25	21 M, 3 S, 1 Ca	58
		100	4 (weekly)		33	18	16 M, 2 S	50
UICC crocidolite		2	1		39	15	12 M, 3 S	97
7 kinds of granular dust	–	100	4 (weekly)		263	3	1 M, 1 S, 1 Ca	85
Corundum	–	50	2 (weekly)		37	3	1 M, 2 Ca	107
Saline	–	–	1		72	0	–	85
Intraperitoneal injection of glass fibres in saline (female Wistar rats, 4 weeks old) (Pott et al., *1984b)*								
Glass fibres	L 50% <2.4 µm D 50% <0.33 µm	2	1	Lifetime (some early deaths from infection in month 21)	44	2	2 (M, S)	–
Glass fibres		2	1		44	14	14 (M, S)	–
Glass fibres		10	1		44	29	29 (M, S)	–
Actinolite	L 50% > 1.4 µm D 50%>0.16 µm	2.5	1		45	31	31 (M, S)	–
UICC chrysotile B	L 50% >0.9 µm D 50%>0.11 µm	0.4	1		44	9	9(M, S)	–
Corundum	Granular	2	1		45	1	1 (M, S)	
Intraperitoneal injection of glass fibres in 2 ml saline (female Sprague-Dawley rats, 8 weeks old) (Pott et al., *1987)*								
Glass fibre	L 50% <2.4 µm D 50% <0.33 µm	2	1	134 weeks	54	21	(M, S, Ca)	90
Glass fibre	L 50% <2.4 µm D 50% <0.33 µm	10	1	126 weeks	53	24	(M, S, Ca)	79
Volcanic ash, Mount St Helen's	Granular	40	2	134 weeks	54	3	(M, S, Ca)	93
Saline	–	–	2	134 weeks	54	3	(M, S, Ca)	94
Intraperitoneal injection of glass fibres in 1 ml saline (female Wistar rats, 5 weeks old) (Pott et al., *1987)*								
Glass fibre	L 50% <3.2 µm D 50%<0.18 µm	0.5		142 weeks	30	5	(M, S, Ca)	116
Glass fibre	L 50% <3.2 µm D 50%<0.18 µm	2.0		142 weeks	31	8	(M, S, Ca)	110
Glass fibre 24-h HC1 treated		2.0		141 weeks	32	16	(M, S, Ca)	107
Crocidolite (S. Africa)	L 50%>2.1 µm D 50% >0.20 µm	0.5		141 weeks	32	18	(M, S, Ca)	109
Crocidolite (S. Africa)	L 50%>2.1 µm D 50% >0.20 µm	2.0		103 weeks	32	28	(M, S, Ca)	71
Titanium dioxide	Granular	10	3	142 weeks	32	0	–	130
Saline				142 weeks	32	2	(M, S, Ca)	120
Intraperitoneal injection of glass fibres in 2 ml saline (female Sprague-Dawley rats, 8 weeks old) (Pott et al., *1987)*								
Glass fibre	L 50% <4.8 µm D 50% <0.29 µm	5	1	108 weeks	54	44	(M, S, Ca)	64
Glass fibre 2-h HCl-treated		5	1	133 weeks	54	32	(M, S, Ca)	88
Glass fibre 24-h HCl-treated	L 50% <5.3 µm D 50% <0.5 µm	5	1	142 weeks	54	4	(M, S, Ca)	99

Substance	Fibre dimensions: length (L), diameter (D)	Dosing schedule		Length of observation	No. of animals examined	No. of animals with tumours[a]	Histological type[b]	Median or average survival time [weeks]
		Total dose [mg]	No. of applications					
Glass fibre 2-h NaOH-treated		5	1	115 weeks	54	42	(M, S, Ca)	71
Glass fibre 24-h NaOH-treated	L 50% <5.4 µm D 50% <0.5 µm	5	1	106 weeks	53	46	(M, S, Ca)	72
Titanium dioxide	Granular	5	1	142 weeks	52	2	(M, S, Ca)	99
Intraperitoneal injection of glass fibres in 1 ml saline (female Wistar rats, 4 weeks old) (Pott et al., *1987)*								
Glass fibre	L 50% <4.8 µm d 50% <0.29 µm	5	1	65 weeks	45	20	(M, S, Ca)	34
Glass fibre 24-h HCl-treated	L 50% <5.3 µm d 50% <0.5 µm	5	1	146 weeks	45	2	(M, S, Ca)	113
Glass fibre 24-h NaOH-treated	L 50% <5.4 µm d 50% <0.5 µm	5	1	103 weeks	46	27	(M, S, Ca)	58
Titanium dioxide	Granular	5	1	145 weeks	47	0	–	102
Intraperitoneal injection of glass fibres in 0.5 ml saline (female Osborne-Mendel rats, 100 days old) (Smith et al., *1987)*								
Glass fibres	L g. mean, 4.7 µm D g. mean, 0.4 µm	25	1	Lifetime	25	8	8 M	85
UICC crocidolite	L 5% > 5 µm	25	1		25	20	20 M	82
Saline	–	–			25	0	–	106
No treatment	–	–			125	0	–	103
Intraperitoneal injection of glasswool in 1 ml saline (female Syrian golden hamsters, 8–12 weeks old) (Pott et al., *1976)*								
Glasswool	L 59% <3 µm	2	1	Lifetime	40	0	–	–
		10	1		40	0	–	–
UICC chrysotile A		2	1		40	0	–	–
		10	1		40	0	–	–
Intraperitoneal administration of glass filaments								
Intraperitoneal injection of glass filaments in 2 ml saline (female Wistar rats, 12 weeks old) (Pott et al., *1987)*								
Glass filament (finer)	L 50% <39 µm D 50% <5.5 µm	10	1	165 weeks	50	2	(M, S, Ca)	111
Glass filament (finer)	L 50% <39 µm D 50% <5.5 µm	40	2	165 weeks	46	5	(M, S, Ca)	107
Glass filament (coarser)	L 50% <46 µm D 50% <7.4 µm	40	2	156 weeks	47	1	(M, S, Ca)	121
Glass	Granular	40	2	165 weeks	45	2	(M, S, Ca)	119
Intraperitoneal administration of glass filaments in 4 ml saline by laparotomy (female Wistar rats, 12 weeks old) (Pott et al., *1987)*								
Glass filament (finer)	L 50% <39 µm d 50% <5.5 µm	250	1	144 weeks	28	2	(M, S, Ca)	109
Intraperitoneal administration of glass filaments in 4 ml saline by laparotomy (female Wistar rats, 15 weeks old) (Pott et al. , *1987)*								
Glass filament, ES 3	L 50%<16.5 µm D 50% <3.7 µm	50	1	135 weeks	48	3	(M, S, Ca)	94
Glass filament, ES 3	L 50% < 16.5 µm D 50% <3.7 µm	250	1	139 weeks	46	4	(M, S, Ca)	94
Glass	Granular	50	1	139 weeks	48	4	(M, S, Ca)	88
		250	1	130 weeks	48	4	(M, S, Ca)	99
Saline	–	–	1	139 weeks	45	2	(M, S, Ca)	87

Substance	Fibre dimensions: length (L), diameter (D)	Dosing schedule		Length of observation	No. of animals examined	No. of animals with tumours[a]	Histological type[b]	Median or average survival time [weeks]
		Cumulative exposure [mg/m^3 × h]	Duration of exposure					
Inhalation exposure to rockwool and slagwool								
Inhalation exposure to respirable dust concentrations of 5 mg/m^3 (Wistar rats IOPS Af / Han, equal numbers of males and females, 8–9 weeks old) (Le Bouffant et al., *1984)*								
Rockwool	L 40%<10 µm D 23% <1 µm	Not given	5 h/day, 5 days/ week; total length of dusting: half the animals, 12 months, the other half, 24 months	Up to 28 months (several animals killed at 7, 12, 16 and 24 months)	47	0	–	–
Chrysotile (Canadian)	L *6% >5 µ*m				47	9	Pulmonary tumours	
Controls	–	–			47	0	–	–
Inhalation exposure to respirable dust concentrations of 10 mg/m^3 (SPF Fischer rats) (Wagner et al., *1984) [sex, age unspecified]*								
Rockwool without resin	L 71% 5–20 µm D 58% ⩽1 µm	17 495	7 h/day, 5 days/ week, 12 months	Lifetime (some animals killed at 12 and 24 months)	48	2	2A	–
UICC chrysotile (Canadian)	L 16%>20 µm D 29%>.5 µm	17 499			48	12	1 A, 11 AdCa	–
Controls	–	–			48	0	–	–
Nose-only inhalation exposure to dust clouds of slagwool (female Osborne-Mendel rats, 100 days old) (Smith et al., *1987)*								
		Dust cone.						
Slagwool	L g. mean, 22 µm d g. mean, 0.9 µm	7.8 mg/m^3 (200 f/cm^3)	6 h/day, 5 days/week, 2 years	Lifetime	55	0	–	97
UICC crocidolite	L 5% >5 µm	3000 f/cm^3			57	3	1 M, 2 BT	109
Chamber controls	–	–			59	0	–	108
Room controls	–	–			125	0	–	103
Nose-only inhalation exposure to dust clouds of slagwool (male Syrian golden hamsters, 100 days old) (Smith et al., *1987)*								
		Dust cone.						
Slagwool	L g. mean, 22 µm D g. mean, 0.9 µm	7.8 mg/m^3 (200 f/cm^3)	6 h/day, 5 days/ week, 2 years	Lifetime	69	0	–	95
UICC crocidolite	L 5% >5 µm	3000 f/cm^3			58	0	–	78
Chamber controls	–	–			58	1	1 BT	95
Room controls	–	–			112	0	–	80

Substance	Fibre dimensions: length (L), diameter (D)	Dosing schedule		Length of observation	No. of animals examined	No. of animals with tumours[a]	Histological type[b]	Median or average survival time [weeks]
		Total dose [m]	No. of application					
Intrapleural administration of rockwool and slagwool								
Intrapleural injection of rockwool and slagwool in 0.5 ml saline (SPF Sprague-Dawley rats) (Wagner et al., *1984) [sex, age unspecified]*								

Substance	Fibre dimensions: length (L), diameter (D)	Dosing schedule		Length of observation	No. of animals examined	No. of animals with tumours[a]	Histological type[b]	Median or average survival time [weeks]
		Total dose [m]	No. of application					
Rockwool with resin (Sweden)	L 70% < 5 µm D 52% < 0.6 µm	20	1	Lifetime	48	3	3M	–
Rockwool, resin removed	L 70% < 5 µm D 58% < 0.6 µm	20	1		48	2	2M	–
Slagwool with resin (F.R. Germany)	L 67% < 5 µm D 42% < 0.6 µm	20	1		48	0	–	–
Slagwool, resin removed	L 80% < 5 µm D 62% < 0,6 µm	20	1		48	0	–	–
UICC African chrysotile		20	1		48	6	6M	–
Saline					24	0	–	–
Intraperitoneal administration of rockwool and slagwool								
Intraperitoneal injection of slagwool in 2 ml saline (female Wistar rats, 15 weeks old) (Pott et al., 1987)								
Slagwool, coarser (F.R. Germany)	L 50% < 26 µm D 50% < 2.6 µm	40	2	158 weeks	99	6	(M, S, Ca)	111
Slagwool, finer (F.R. Germany)	L 50%< 14 µm D 50%< 1.5 µm	40	2	155 weeks	96	2	(M, S, Ca)	107
Saline	–	–	2	150 weeks	48	0	–	101
Intraperitoneal injection of various kinds of rockwool in 2 ml saline (female Wistar rats, 8 weeks old) (Pott et al., 1987)								
Rockwool (F.R. Germany)	L 50% < 20 µm D 50% < 1.8 µm	75	5	Preliminary results 28 months after first injection	53	32	(M, S, Ca)[c]	79
Actinolite (F.R. Germany)	L 50% < 1.9 µm D 50% < 0.17 µm	0.25	1		36	20	(M, S, Ca)[c]	90
UICC chrysotile B	L 50% > 0.9 µm D 50% > 0.11 µm	1	1		36	31	(M, S, Ca)[c]	63
Titanium dioxide	Granular	100	5		53	5	(M, S, Ca)[c]	109
Saline	–	–	5		102	2	(M, S, Ca)[c]	111
Intraperitoneal injection of various kinds of rockwool in 2 ml saline (female Sprague-Dawley rats, 8 weeks old) (Pott et al., 1987)								
Rockwool (Sweden)	L 50% < 23.0 µm D 50% < 1.9 µm	75	3	134 weeks	63	45	(M, S, Ca)	77
Rockwool, fine (Sweden)	L 50% < 4.1 µm D 50% < 0.64 µm	10	1	134 weeks	45	6	(M, S, Ca)	97
Volcanic ash, Mount St Helen's	Granular	40	2	134 weeks	54	3	(M, S, Ca)	93
Saline			2	134 weeks	54	3	(M, S, Ca)	94

Substance	Fibre dimensions: length (L), diameter (D)	Dosing schedule		Length of observation	No. of animals examined	No. of animals with tumours[a]	Histological type[b]	Median or average survival time [weeks]
		Cumulative exposure [mg/m^3 × h]	Duration of exposure					
Inhalation exposure to ceramic fibres								
Inhalation exposure to total dust concentration of 20–120 mg/m^3 (rat) (Pigott & Ishmael, 1982) [strain, sex, age unspecified]								
Ceramic fibre 'Aged' fibre	D median, 3.3 um	6 700 resp. f 7 400 resp. f	18 months	Lifetime (to 85% mortality)	32 38	0 0	– –	– –

Substance	Fibre dimensions: length (L), diameter (D)	Dosing schedule		Length of observation	No. of animals examined	No. of animals with tumours[a]	Histological type[b]	Median or average survival time [weeks]
		Cumulative exposure [mg/m^3 × h]	Duration of exposure					
UICC chrysotile A		13 800 resp. f			39	9	5 A, 1 AdCa, 3 ScCa	–
Clean air	–	–			34	0	–	–
Inhalation exposure to respirable dust concentrations of 10 mg/m^3 (AF/Han Wistar rats, 12 weeks old) (Davis, 1984)[sex unspecified]								
Ceramic fibres (aluminium silicate glass)	L ~ 90% <3 µm D ~ 90% <0.3 µm		7 h/day, 5 days/week, 12 months (224 days)	32 months (4 animals, 12 months; 4 animals, 18 months)	48	7	1 A, 3 Ca, 4 malignant unspecified	
Controls	–	–			39	0	–	–
Nose-only inhalation exposure to dust clouds of ceramic fibres (female Osborne-Mendel rats, 100 days old) (Smith et al., *1987)*								
Ceramic fibres	L g. mean, 25 µm D g. mean, 0.9 µm	10.8 mg/m^3 (200 f/cm^3)	6 h/day, 5 days/week, 2 years	Lifetime	55	0	–	100
UICC crocidolite	L 5% ⩾5 µm	3000 f/cm^3			57	3	1 M, 2 BT	109
Chamber controls	–	–			59	0	–	108
Room controls	–	–			125	0	–	103
Nose-only inhalation exposure to dust clouds of ceramic fibres (male Syrian golden hamsters, 100 days old) (Smith et al., *1987)*								
Ceramic fibres	L g. mean, 25 µm D g. mean, 0.9 µm	10.8 mg/m^3 (200 f/cm^3)	6 h/day, 5 days/ week, 2 years	Lifetime	70	1	1 M	96
UICC crocidolite	L 9% ⩾*5 µ*m	3000 f/ cm^3			58	0	–	79
Chamber controls	–	–			58	1	1 BT	95
Room controls	–	–			112	0	–	80

Substance	Fibre dimensions: length (L), diameter (D)	Dosing schedule		Length of observation	No. of animals examined	No. of animals with tumours[a]	Histological type[b]	Median or average survival time [weeks]
		Total dose [mg]	No. of application					
Intratracheal administration of ceramic fibres								
Intratracheal instillation of ceramic fibres in 0.2 ml saline (female Osborne-Mendel rats, 100 days old) (Smith et al., *1987)*								
Ceramic fibres	L g. mean, 25 µm D g. mean, 0.9 µm	10	5 (weekly)	Lifetime	22	0	–	100
UICC crocidolite	L 5% ⩽ 0.5 µm	10	5 (weekly)		25	2	2BT	91
Saline	–	–	5 (weekly)		25	0	–	98
No treatment	–	–			125	0	–	103
Intratracheal instillation of ceramic fibres in 0.2 ml saline (female Syrian golden hamsters, 100 days old) (Smith et al., *1987)*								
Ceramic fibres	L g. mean, 25 µm D g. mean, 0.9 µm	10	5 (weekly)	Lifetime	25	0	–	64
UICC crocidolite	L 5% ⩾ 0.5 µm	10	5 (weekly)		27	20	20 BT(13 benign, 7 malignant)	85
Saline	–	–	5 (weekly)		24	0	–	81
No treatment	–	–			112	0	–	80
Intrapleural administration of ceramic fibres								
Intrapleural injection of ceramic fibres in 0.4 ml saline (SPF Wistar rats, twice as many males as females, 13 weeks old) (Wagner et al., *1973)*								

Substance	Fibre dimensions: length (L), diameter (D)	Dosing schedule		Length of observation	No. of animals examined	No. of animals with tumours[a]	Histological type[b]	Median or average survival time [weeks]
		Total dose [mg]	No. of application					
Ceramic fibres (aluminium silicate)	D 0.5–1 µm	20	1	Lifetime	31	3	3 M	105
SFA chrysotile		20	1		36	23	23 M	81
SFA chrysotile		20	1		32	21	21 M	91
Aluminium oxide	Granular, D <10 µm	20	1		35	1	1 M	101
Intrapleural injection of ceramic fibres in saline (rats, equal numbers of females and males) (Pigott & Ishmael, 1982) [strain, age unspecified]								
Ceramic fibres	D median, 3.3 µm	20	1	Lifetime	48	0	–	–
'Aged' fibres		20	1		48	0	–	–
UICC chrysotile A		20	1		48	7	7M	–
Saline	–	–	1		48	0	–	–
Intraperitoneal administration of ceramic fibres								
Intraperitoneal injection of ceramic fibres in 2 ml saline (AF/Han Wistar rats) (Davis et al., 1984) [sex, age unspecified]								
Ceramic fibres (aluminium silicate glass)	L ~ 90% <3 µm D ~ 90% <0.3 µm	25	1	–	32	3	1 M, 2 FS?	–
Controls	–	–			39	2	2 M (peritoneum or digestive system)	–
Intraperitoneal injection of ceramic fibres in 2 ml saline (female Wistar rats, 8 weeks old) (Pott et al., 1987)								
Ceramic wool	L 50% <8.3 µm D 50%<0.91 µm L 50% <6.9 µm D 50%<1.1 µm	45 75	5 5	Preliminary results 28 months after first injection	47 54	32 12	(M, S, Ca)[c] (M, S, Ca)[c]	51 91
Actinolite (F.R. Germany)	L 50% < 1.9 µm D 50%<0.17 µm	0.25	1		36	20	(M, S, Ca)[c]	90
UICC chrysotile B	L 50% >0.9 µm D 50%>0.11 µm	1	1		36	31	(M, S, Ca)[c]	63
Titanium dioxide	Granular	100	5		53	5	(M, S, Ca)[c]	109
Saline			5		102	2	(M, S, Ca)[c]	111
Intraperitoneal injection of ceramic fibres in 0.5 ml saline (female Osborne-Mendel rats, 100 days old) (Smith et al., 1987)								
Ceramic fibres	L g. mean, 25 µm D g. mean, 0.9 µm	25	1	Lifetime	23	19	19 M	69
Saline	–	–	1		25	0	–	106
No treatment	–	–			125	0	–	103
Intraperitoneal injection of ceramic fibres in 0.5 ml saline (male Syrian golden hamsters, 100 days old) (Smith et al., 1987)								
Ceramic fibres	L g. mean, 25 µm D g. mean, 0.9 µm	25 25	1 1	Lifetime	15 21	2 5	2M 5 M	66 70
Saline	–	–	1		25	0	–	72
No treatment	–	–			112	0	–	72

a Tumours of the lung, pleura, thorax or abdominal cavity

b A, adenoma; AdCa, adenocarcinoma; BT, bronchoalveolar tumour; Ca, relatively undifferentiated epidermoid carcinoma; FS?, somewhat similar to a fibrosarcoma; M, mesothelioma; (M, S), mesothelioma and/ or sarcoma; (M, S, Ca), mesothelioma, sarcoma and/ or carcinoma (rarely) in the abdominal cavity, excluding tumours of the uterus; P, papilloma; ScCa, squamous-cell carcinoma; T, other lung tumours (fibrosarcoma, lymphosarcoma, lung metastases)

c Partly macroscopic diagnosis only

_, not applicable

Table 39 Summary of results of implantation of different forms of fibrous glass in the pleural cavity of rats[a]

Fibre type	Incidence of pleural sarcomas[b]	log fibres/*μg*, ⩽0.25 *μ*m × >8 μm
Glass 1	9/17	5.16
Glass 2	12/31[c]	4.29
Glass 3	20/29	3.59
Glass 4	18/29	4.02
Glass 5	16/25	3.00
Glass 6	7/22	4.01
Glass 7	5/28	2.50
Glass 8	3/26	3.01
Glass 9	2/28	1.84
Glass 10	2/27	–
Glass 12 (coated)	1/25	–
Glass 13	1/27	–
Glass 14 (coated)	1/25	–
Glass 15 (coated)	1/24	1.30
Glass 16	1/29	–
Glass 17	0/28	–
Glass 18 (coated)[d]	0/115	–
Glass 19 (leached)	2/28[c]	–
Glass 20 (leached)	4/25[c]	–
Control (pleural implants described as noncarcinogenic)	17/615 (2.8%)	
Control (untreated)	3/491 (0.6%)	–

a From Stanton *et al.* (1977, 1981)

b Incidence in animals surviving longer than 52 weeks, except where noted (Stanton *et al.*, 1977)

c Survival of animals in which incidence was determined is not specified (Stanton *et al.*, 1981).

d Control in first series of experiments (Stanton *et al.*, 1977)

Table 40 Summary of results of implantation of different ceramic fibres in the pleural cavity of rats[a]

Fibre	Incidence of pleural sarcomas	log fibres/μg, $\leqslant$ 0.25 µm × >8 µm
Potassium titanate 1	21/29	4.94
Potassium titanate 2	20/29	4.70
Silicon carbide	17/26	5.15
Aluminium oxide 1	15/24	3.63
Aluminium oxide 2	8/27	2.95
Aluminium oxide 3	9/27	2.47
Aluminium oxide 4	4/25	2.60
Aluminium oxide 5	4/22	3.73
Aluminium oxide 6	2/28	0.82
Aluminium oxide 7	1/25	–
Aluminium oxide 8	1/28	–
Glass filament >80% aluminium oxide	2/47	–
Glass filament >90% zirconium oxide	1/45	–
Control (pleural implants described as noncarcinogenic)	17/615 (2.8%)	–
Control (untreated)	3/491 (0.6%)	–

a From Stanton *et al.* (1981)

Table 42 Mortality from lung cancer and mesothelioma in the major US and European epidemiological studies of man-made mineral fibre production workers[a]

Feature	Study	Glass filament		Glasswool	Rock-/slagwool
No. of workers in study	US	3435		11 380	1846
Person-years of follow-up			385 924		48 188
Lung cancer deaths		64		267	60
No. of workers in study	European	3566		8286	10 115
Person-years of follow-up		56 332		148 203	160 066
Lung cancer deaths		15		93	81
SMRs from lung cancer compared to local reference rates					
Lung cancer mortality	US	92		109	134 ($p< 0.05$)
	European	97		103	124
	[Combined	93		108	128 ($p< 0.01$)]
Time since first exposure	US	104/53/119/80		92/108/108/114	90/157/127/135
(<10/10-19/20-29/30+years)	European [Combined	176/76/0/0 138/61 /111/79		68/113/100/138 77/110/106/116	104/122/124/185 102/130/125/148]
Duration of exposure (<20/20+ years)	US European	–	106/110	118/60	145/111 143/141
>20 years since first exposure	[Combined		112/100		129/121]
Cumulative exposure In increasing intervals of fibre/ cm^3-months	US cohort	96/51/109/63		120/109/81/108	185/164/119/104
Two models adjusting for smoking	case-control[b]	–		No trend, no trend	No trend, increase ($p<0.01$)
Technological phase	European				
Early/ intermediate/ late		–		92/111/77	257 ($p<0.05$)/141/111
By time since first exposure (early phase only, <10, 10-19, 20-29, 30+ years)		–		108/70/80/121	0/0/317/295 ($p< 0.05$)
Small-diameter fibres	US				
Ever/never exposed		–		124/105	–
By time since first exposure					
(Ever exposed only, <10/10-19/20-29/30+ years)		–		61/128/105/198	–
Mesothelioma		No case		No excess	No excess
Statistically significant results[c]		None		None (but see footnote)	Yes (as shown)
Estimated concentrations of respirable fibres	US	Lower		Intermediate (higher concentration of small-diameter fibres than large)	Higher

a From Enterline *et al.* (1987); Simonato *et al.* (1987). In a much smaller cohort study of glasswool workers in Canada, the overall lung cancer SMR compared to local rates was 199 (19 observed, $p < 0.05$), but there was no increasing relationship with time since first exposure (<10 years, SMR = 241; 10+ years, SMR = 195) nor with duration of exposure (<5 years, SMR = 291; 5+ years, SMR = 174) (Shannon *et al.*, 1987).

b Reservations about the interpretation of this study are expressed in the text.

c These relate to SMRs themselves where only one or two are shown, otherwise the statistical tests used examined for linear trends.

Table 43 Respiratory cancer in man-made mineral fibre production workers in the major US epidemiological study[a]

Fibre type	Estimated average concentration (fibre/cm^3)[b]	SMR[c]	
		Total	20 years' latency
Glass filament	0.01	92 (64)	105 (49)
Glasswool	0.06	109 (267)	111 (207)
Small-diameter	0.1	124(22)	146 (14)
Rock-/slagwool	0.35	134* (60)	131 (45)

a From Enterline *et al.* (1987)

b Fibres <3 µm in diameter and >5 µm in length

c Number of deaths in parentheses; expected deaths based on local mortality rates

* $p < 0.05$

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