WOLLASTONITE

Wollastonite was considered by previous Working Groups in June 1986 (IARC, 1987a) and March 1987 (IARC, 1987b). New data have since become available, and these have been incorporated in the present monograph and taken into consideration in the evaluation.

1. Exposure Data

1.1 Chemical and physical data

1.1.1 *Nomenclature*

Chem. Abstr. Serv. Reg. No.: 13983-17-0

Deleted CAS Reg. Nos: 9056-30-8; 57657-07-5

Chem. Abstr. Name: Wollastonite

Synonyms: Aedelforsite; gillebächite, okenite; rivaite; schalstein; tabular spar; vilnite (Andrews, 1970)

1.1.2 *Structure of typical mineral*

CAS formula: $CaSiO_3$

Wollastonite was named after W.H. Wollaston, an English chemist and mineralogist. Natural wollastonite is an acicular (needle-like) calcium silicate mineral that occurs in triclinic and monoclinic varieties; these varieties are very difficult to distinguish from one another. When triclinic, the unit cell parameters of wollastonite are as follows: a = 0.79, b = 0.73 and c = 0.71 nm; $\alpha = 90°02'$, $\beta = 95°22'$ and $\gamma = 103°26'$ (Deer *et al.*, 1978; Bauer *et al.*, 1994).

Initially, wollastonite was classified as a pyroxene group mineral; however, it has since been shown to have a slightly different chain structure. Wollastonite consists of chains of indefinite length containing three SiO_4 tetrahedra per unit cell. The tetrahedra are joined apex to apex, and one is orientated with an edge parallel to the axis of the chain. These chains are paired; slight offsetting produces the different structural forms of the mineral. Also within the mineral structure are calcium atoms, which occur in octahedral coordination and alternate with layers composed of silica atoms between layers of oxygen atoms (Deer *et al.*, 1978).

1.1.3 *Chemical and physical properties*

From Bauer *et al.* (1994), unless otherwise specified

(*a*) *Description*: Triclinic crystals

(*b*) *Form and habit*: Bladed crystal masses; acicular

(*c*) *Colour*: White when pure; may be grey, pale green, yellowish brown or red with impurities (Bauer *et al.*, 1994; Elevatorski & Roe, 1983; Harben & Bates, 1984; Virta, 1995)

(*d*) *Hardness*: 4.5–5 on Mohs' scale

(*e*) *Density*: 2.87–3.09

(*f*) *Cleavage*: {100} perfect; {001} good; {102} good (Roberts *et al.*, 1974)

(*g*) *Melting-point*: 1540 °C

(*h*) *Inversion temperature*: Pseudowollastonite (high-temperature polymorph) at 1120 °C ± 10 °C

Naturally occurring wollastonite consists almost entirely of α-wollastonite. This low-temperature form can be converted to the metastable β-form, pseudowollastonite, by heating to temperatures of about 1120 °C. Pseudowollastonite, however, occurs rarely in pyrometamorphosed rocks (Deer *et al.*, 1978).

In general, wollastonite is inert chemically; however, it can be decomposed in concentrated hydrochloric acid. Some wollastonite will fluoresce under ultraviolet light, with colours ranging from pink-orange to yellow-orange and, more rarely, bluish green. In addition, wollastonite may show phosphorescence. A 10% wollastonite : water slurry has a naturally high pH of 9.9 (Elevatorski & Roe, 1983; Bauer *et al.*, 1994).

Wollastonite occurs in coarse-bladed masses and rarely shows good crystal form. Because of its unique cleavage properties, wollastonite breaks down during crushing and grinding into lath-like or needle-shaped particles (fibres) of varying acicularity. This particle morphology imparts high strength and is therefore of considerable importance in many applications.

The acicularity of particles is defined by their length : width ratio or length : diameter ratio (aspect ratio). In wollastonite, even the smallest individual particles commonly exhibit an aspect ratio of 7 : 1 or 8 : 1 and have an average diameter of 3.5 μm. Low-aspect ratio products (powder wollastonite, or milled grades) with aspect ratios of 3 : 1 to 5 : 1 are used as general fillers, in ceramics and in metallurgical fluxing. High-aspect ratio products with ratios of 15 : 1 to 20 : 1 are used as functional fillers in the reinforcement of thermoplastic and thermoset polymer compounds and as a replacement for asbestos (Elevatorski & Roe, 1983; Bauer *et al.*, 1994).

1.1.4 *Technical products and impurities*

Wollastonite has a theoretical composition of 48.3% CaO and 51.7% SiO_2, although aluminium, iron, magnesium, manganese, potassium or sodium may partially substitute for calcium (Harben & Bates, 1984; Virta, 1995). The chemical compositions of commercial wollastonite products from several countries are summarized in **Table 1**.

Based on their iron content, two types of synthetic grade wollastonite are produced: extremely low-iron content 'SW' grade (< 0.075% Fe_2O_3) and the low-iron grades 'SM' (< 0.2% Fe_2O_3), 'SE' (< 0.19% Fe_2O_3) and 'SG' (< 0.22% Fe_2O_3) (O'Driscoll, 1990).

Table 1. Chemical composition (%) of commercial wollastonite products from several countries[a]

Component	Finland[b]	USA[b]	India[c]	Kenya[d]	Mexico[d]	China[e]
SiO_2	52	51	49	55	52	46–53
CaO	45	47	48	42	47	43–50
Al_2O_3	0.4	0.3	0.7	0.1	0.5	0.3–0.4
Fe_2O_3	0.2	0.6	0.4	0.07	0.2	0.1–0.2
TiO_2	max. 0.05	0.05	Traces	0.01	0.06	NR
MnO	max. 0.01	0.1	0.1	0.01	0.4	NR
MgO	0.6	0.1	0.06	0.8	0.08	0.2
Na_2O	0.1	NR	0.02	0.04	0.2	NR
K_2O	0.01	NR	0.1	0.04	0.04	NR

max., maximum; NR, not reported

[a] Elements reported as their oxides

[b] From Anon. (1975); Lappeenranta, Finland; Willsboro, NY, USA

[c] From Wolkem Private Ltd (undated); Belkap-ahar, India

[d] From Anon. (1969); Kolkidongai, Kenya; Santa Fe, Mexico

[e] From Roskill Information Services Ltd (1993); Special Grade, Jilin Province, China

Trade names for wollastonite include: Cab-O-Lite; Casiflux; F1; FW50; FW200; FW325; Kemolite; NYAD; Nyad G; NYCOR; Tremin; Vansil; WIC10; WIC40; Wollastocoat; Wollastokup.

1.1.5 *Analysis*

In dust samples, wollastonite can be analysed by phase-contrast optical microscopy (PCOM), X-ray diffractometry and scanning electron microscopy. Identification of wollastonite fibres may be achieved by means of microanalysis and selected area electron diffraction in which the silicon : calcium ratios and structural data are obtained for individual particles (Zumwalde, 1977; Tuomi *et al.*, 1982; Huuskonen *et al.*, 1983a). When characterized on the basis of diagnostic X-ray reflections in powder diffraction patterns, the strongest lines appear at 0.297, 0.352 and 0.383 nm (Roberts *et al.*, 1974). Triclinic and monoclinic wollastonite can be distinguished as lines at 0.405 and 0.437 nm, respectively, adjacent to a common line at 0.383 nm (Deer *et al.*, 1978).

1.2 Production and use

1.2.1 *Production*

Wollastonite was probably first mined in California, United States, in 1933 for mineral wool production. Significant commercial production started in about 1950 at the Willsboro, NY, United States, deposit. Since that time, wollastonite has become widely used, especially in the ceramics industries (Anon, 1969; Power, 1986).

The worldwide production of wollastonite for selected years is presented in **Table 2** for several countries.

Table 2. Wollastonite production by country, 1960–93 (tonnes)[a,b]

Country	1960	1970	1980	1983	1986	1990[c]	1993
China	–	–	–	–	> 13 000	70 000	120 000
Finland	2 300	6 100	8 800	15 400	23 000	40 000	30 000
India	–	600	5 800	16 600	25 000	35 000	62 000
Mexico	4 500	6 600	20 900	10 800	9 000	15 000	36 000
USA	27 000	30 000	76 000	83 000	75 000	110 000	124 000

[a] From Institute of Geological Sciences (1967); Anon. (1975); Institute of Geological Sciences (1978); British Geological Survey (1985); Power (1986); O'Driscoll (1990); Fattah (1994); British Geological Survey (1995)

[b] In addition to the countries listed, Japan, Namibia and Turkey are believed to produce wollastonite (British Geological Survey, 1995). Commercial production has also been reported in Greece, Kenya, New Zealand and South Africa (Andrews, 1970; Power, 1986; Roskill Information Services Ltd, 1993).

[c] Estimates

The original commercially exploited wollastonite deposit in California was worked by open quarrying. An early use of wollastonite was as ornamental slabs or rocks, which simply required collection of the materials close to or on the surface (Andrews, 1970); much of the early Californian and nearly all of the Mexican production were probably carried out in this way. The first mine at Willsboro, NY, which opened in 1943, was also worked on the surface (Anon., 1969). Since 1960, however, at least one of the three deposits in New York has been mined principally underground owing to the presence of a structurally complex wollastonite vein (Anon., 1975). Wollastonite mines in other principal production areas, in Finland, China, India and Mexico, are worked by both opencast and underground mining (Andrews, 1970; Anon., 1975; Fattah, 1994).

The refinement of wollastonite ore into high-grade wollastonite was originally done by manual selection at many mines. This process is now performed by screening and magnetic separators, sometimes in combination with flotation and vacuum filtration (Andrews, 1970). Grinding and milling operations can produce variable mesh powders or aggregates (Power, 1986).

More recently, synthetic wollastonite has been introduced. This product plays a limited role in the wollastonite industry where purity and performance are required. All known production of synthetic wollastonite is as powder grade. Among the countries that produce commercial grades of synthetic wollastonite are Belgium, Brazil and Germany (Fattah, 1994).

1.2.2 *Use*

In descending order of importance, the main markets for wollastonite are as follows: ceramics; plastics and rubber; asbestos substitution; metallurgy; and paints and coatings.

These markets can be divided into two main categories — those for high-aspect ratio wollastonite and those for milled or powder grades. In general, the high-aspect ratio markets rely on the physical acicularity of wollastonite, while the markets for milled wollastonite rely on the mineral's chemical composition (Fattah, 1994).

High-aspect ratio wollastonite, which tends to have an aspect ratio of 10 : 1 to 20 : 1, is used as a reinforcing and functional filler in a variety of applications — especially in plastics and rubber (19–25% of total consumption), as asbestos substitution (20–25%) and, to a lesser extent, in paints and coatings (about 2–5%). In these applications, the wollastonite provides added hardness, flexural strength and impact resistance. In plastics, high-aspect ratio wollastonite can improve the electrical properties and the heat and dimensional stabilities of the finished products.

Milled grades of wollastonite are used as a source of both calcium oxide and silicon dioxide and have unique qualities as fillers. The prime markets in this segment are ceramics (40–45%) and metallurgy (12–15%) (Fattah, 1994).

(*a*) *Ceramics*

In 1993, the ceramics market consumed approximately 150 thousand tonnes of wollastonite, which accounted for approximately 42% of total world production. Within this market, most of the wollastonite was used in wall and floor tile bodies and glazes, while a smaller share was used in sanitary ware, earthenware and specialized applications. Wollastonite is added to these products to help prevent cracking, crazing, breaking and glaze defects (Fattah, 1994).

(*b*) *Asbestos replacement*

Wollastonite has become increasingly popular in the past two decades as a substitute for short-fibre asbestos in fire-resistant wall board and cement products and in certain friction products. Approximately 35–40 thousand tonnes per year of high-aspect ratio wollastonite are consumed in construction and insulation board applications. Wollastonite is used commonly in both indoor and outdoor applications in wall boards, roofing tiles, slates, shaped insulation and sidings, as well as in high-temperature insulation boards for non-refractory applications (Fattah, 1994).

Wollastonite has also been an important additive in friction products such as brake pistons, brake linings and clutches. In North America, asbestos formulations for friction products have been replaced by fibre packages based on high-aspect ratio wollastonite and metallic and organic fibres. Outside the United States, wollastonite is also being used to replace asbestos in gaskets (Fattah, 1994).

(*c*) *Plastics and rubber*

The plastics industry represents the greatest growth market and one of the highest value applications for wollastonite. The popularity of wollastonite as a filler in this industry is due to the reinforcing properties of wollastonite, combined with the following attributes: low water absorbency; low resin demand; thermal stability; thermal conductivity; chemical purity. Although most grades of wollastonite are useful in plastics, the most important are high-aspect ratio, fine and surface-coated wollastonites (Fattah, 1994).

Wollastonite has found applications in both thermosets (in which chemical cross-linking prevents the plastic from softening at high temperatures) and thermoplastics (in which the plastic softens with increasing temperatures). Examples of thermoplastics that use wollastonite include polyamides such as nylon 6, nylon 6/6, polyester, liquid crystal polymers and engineered resins; thermosets that use wollastonite include phenolic moulding compounds, epoxies, polyurethanes, polyurea and some unsaturated polyesters. Typical wollastonite loadings in plastics include: nylon (50%), low-density polyethylene (40%), polypropylene (23–28%) and polystyrene (30%). As is common in other filler markets, wollastonite is used in plastics mainly as a cheaper alternative to other fillers such as short-milled fibreglass, mica and talc (Fattah, 1994).

(*d*) *Metallurgy*

Owing to its low-temperature fluxing properties, wollastonite has found wide acceptance in metallurgical applications, especially in continuous casting. For example, when molten steel from the bottom of a refining ladle is poured into refractory tundishes, wollastonite is added to the melt to maintain the surface in a molten state. This minimizes surface defects of the steel, prevents the oxidation of the metal surface in contact with air, lubricates the wall of the mould and absorbs metallic inclusions. In similar applications, wollastonite is also used to improve the burn characteristics or to inhibit sparking in welding powder formulations. Wollastonite produced for metallurgical uses tends to be of low grade and not extensively processed (Fattah, 1994).

(*e*) *Paints and coatings*

The use of wollastonite in coatings began in the early 1950s when wollastonite of high brightness was introduced to the United States market. At that time, wollastonite was the only acicular extender that was pure white and featured an aspect ratio ranging from 3 : 1 to 20 : 1. The mineral's acicularity proved valuable in the reinforcement of paint films; it improved mechanical strength, durability and weathering, and resulted in better resistance to cracking, checking and other coating ageing defects. Wollastonite is now used as an extender and filler in both oil- and water-based emulsion paints for exterior use and in latex and road-marking paints. Also, because of the brilliant nature of its white colour (when very pure), its low oil absorption, stable high pH (9.9) and good wetting abilities, wollastonite is added to many other types of coatings, where it imparts colour, fluidity and mildew resistance. Paint-grade wollastonite, a fine high-purity grade, has been added at levels of 9–13% wt/wt to many paints in the United States (Andrews, 1970; Anon., 1975; Fattah, 1994).

(*f*) *Other uses*

In the glass and fibreglass industry, small volumes of wollastonite are used mainly as an additive replacing limestone and silica to reduce energy consumption. Wollastonite has also been used in abrasives, in welding electrodes, as a soil conditioner and plant fertilizer, as a filler in paper and as a road material (Andrews, 1970; Anon., 1975; Elevatorski & Roe, 1983).

Wollastonite is finding a new use in synthetic bone implants in which a synthetic β-wollastonite (rather than α-wollastonite, which is normally the form produced synthetically), is used to replace bone loss. It has been used as an effective vertebral prosthesis, and has been found to form strong bonds rapidly with osseous tissue (Fattah, 1994).

The synthetic grades of wollastonite, SW and SM (see Section 1.1.4), are used in ceramic applications; SE and SG grades (which both have sulfur and phosphorus contents $< 0.01\%$) are used in metallurgical applications. SW wollastonite is used extensively as a water-soluble calcium base in white glazes or glaze frits, where coloured metal oxide impurities must be avoided (O'Driscoll, 1990).

1.3 Occurrence and exposure

1.3.1 *Natural occurrence*

Wollastonite occurs most commonly in nature where limestone has reacted at high temperature with igneous rock and created either one of two principal mineral types. In skarn deposits (contact metamorphic genesis), wollastonite is typically of high purity and accounts for most of the world's mined ores. This wollastonite is fine-grained and usually interspersed with other silicates. The other type formed by magmatic process, in which wollastonite occurs in association with carbonatites, is found to a much more limited extent in nature (Andrews, 1970; Kuzvart, 1984; Fattah, 1994).

Ores from the major wollastonite deposits contain 18–97% wollastonite. The associated minerals are most often calcite, quartz, garnet, epidote, apatite, sphene, idocrase and diopside; the approximate mineral compositions of commercial wollastonite deposits from United States, Finland and Kenya are presented in **Table 3**. Indian ores also contain minor amounts of these minerals (Andrews, 1970; Power, 1986; Bauer *et al.*, 1994; Virta, 1995).

Table 3. Mineral composition (%) of some commercial wollastonite deposits

Component	USA[a]	Finland[a,b]	Kenya[a]
Wollastonite	60	90	87
Garnet	30	–	–
Quartz	< 3[c]	2	13
Diopside	10	–	–
Calcite	–	3	–
Other minerals	–	5	–

[a] From Power (1986)
[b] Data for purified commercial product
[c] From Zumwalde (1977)

1.3.2 *Occupational exposure*

Airborne dust and fibre concentrations have been measured at Lappenranta, Finland, and Willsboro, NY, United States (see **Table 4**). These localities represent the two largest wollastonite production sites in the world.

Table 4. Mean concentrations of total dust and fibres in wollastonite mining and milling

Mine site and operations	Total dust (mg/m^3)[a]		Fibres > 5 μm in length (fibres/mL)			
			PCOM[b]		SEM or TEM	
	No. of samples	Mean	No.	Mean	No.	Mean
Lappenranta, Finland, 1981[c]						
Drilling	6	27	–	–	10	4
Crushing	36	33	11	13	20	25
Sorting	16	15	–	–	7	8
Milling	6	22	5	21	4	30
Bagging	2	27	2	19	3	36
Willsboro, USA, 1976–82[d]						
Drilling, loading	2	0.9	3	0.3	3	0.3
Crushing and milling	11	5	1	0.8	1	0.9
Beneficiator and labourer	8	12	1	20	1	11
Packers	26	10	6	32	6	13

PCOM, phase-contrast optical microscopy; SEM, scanning electron microscopy; TEM, transmission electron microscopy

[a] Full-shift sampling

[b] Short-term sampling

[c] From Tuomi *et al.* (1982)

[d] From Hanke & Sepulveda (1983); Hanke *et al.* (1984)

The quarry at Lappenranta, Finland, produced wollastonite as a side-product of limestone mining. Consequently, occupational exposures to wollastonite fibres during the operation stages, from drilling in the opencast mine to fine crushing before froth flotation processing at a separate location, involved concomitant exposures to granular calcite dust. On average, the quarried stone contained about 15% wollastonite and 2–3% quartz; the respirable fraction of dust samples from mining and milling operations had a similar mean composition. In drilling, crushing and sorting, the concentration of total dust ranged from 2 to 99 mg/m^3 and the levels of airborne fibres from 1 to 45 fibres/mL, as measured by PCOM. In the flotation and bagging plant, dust was mainly composed of wollastonite, and workplace concentrations ranged from 15 to 30 mg/m^3 for total dust and from 8 to 37 fibres/mL for fibres, as counted by PCOM. Mean values for samples from breathing zones and stationary samples are shown in **Table 4**; the mean concentration of total dust ranged from 15 to 33 mg/m^3 in various operations. The counting criteria were the same as those most commonly used for asbestos: all fibres > 5 μm in length, < 3 μm in diameter and with an aspect ratio over 3 : 1 were counted. When studied by scanning electron microscopy, the thinnest wollastonite fibres were characteristically 0.2–0.3 μm in diameter. The median fibre lengths and median diameters were 4 μm and 0.8 μm in crushing operations and 2 μm and 0.4 μm in bagging work (Huuskonen *et al.*, 1982; Tuomi *et al.*, 1982; Huuskonen *et al.*, 1983a).

Similar results have been reported from the United States wollastonite production plant in Willsboro, NY (see **Table 4**). In opencast and underground mining, crushing, packing and maintenance, the mean concentration of total dust ranged from 0.9 to 12 mg/m^3. Bulk samples contained less than 2% free silica. In the same operations, airborne fibre counts by PCOM showed a mean of 0.3 fibres/mL in the mine and a range of means of 1–32 fibres/mL (fibres > 5 μm) in the mill. Fibrous particles had a median diameter of 0.22 μm and a median length of 2.5 μm (Zumwalde, 1977; Hanke & Sepulveda, 1983; Hanke *et al.*, 1984).

Where wollastonite has been used in the production of fibre-reinforced cement sheets, airborne respirable fibre (fibre > 5 μm) levels in the range of 0.02–0.2 fibres/mL have been measured during stacking and mixing (SEM analysis) (Danish National Institute of Occupational Health, 1986).

1.3.3 *Non-occupational exposure*

Consumer products, such as tiles, porcelains and cements, generally contain wollastonite that has been subjected to physico-chemical processes that irrevocably alter its original identity and form (Kuzvart, 1984). However, non-occupational exposures may occur from products that contain unaltered wollastonite (such as wallboard or paints). No such exposure data were available to the Working Group.

1.4 Regulations and guidelines

For occupational exposures, wollastonite is regulated by the United States Occupational Safety and Health Administration (1995) with the inert or nuisance dust standard (permissible exposure levels, 15.0 mg/m^3 total dust and 5.0 mg/m^3 respirable fibres). Exposures to crystalline silica, if present, are regulated by the relevant crystalline silica standards (see the monograph on silica in this volume).

In Germany, no MAK (maximal workplace concentration) value has been established for wollastonite (fibrous dust), which is classified as IIB (a substance for which no MAK values can be established at present) (Deutsche Forschungsgemeinschaft, 1996).

In the province of Québec, Canada, a standard of 1 fibre/mL respirable dust time-weighted average (TWA) was established in 1994 (Anon., 1995).

2. Studies of Cancer in Humans

Cohort study

Huuskonen *et al.* (1983b) conducted a cohort study of mortality among all 192 male and 46 female workers who had been on the payroll of a Finnish limestone–wollastonite quarry for at least one year. The study covered the period 1923–80 and expected deaths were calculated from national age- and sex-specific death rates for 1952–72. By the end of 1980, 79 deaths had occurred in the cohort versus 96 expected. Death was due to malignant neoplasms (all sites combined) for 10 men (standardized mortality ratio (SMR), 0.64 [95% confidence interval (CI), 0.31–1.18]) and two women (SMR, 0.67 [95% CI, 0.08–2.41]). Mortality from cancer of the lung and bronchus was the cause of death in four men (SMR, 0.8 [95% CI, 0.22–2.05]) and in no women (0.2 expected). There was a death due to a rare malignant mesenchymal tumour of the retroperitoneum where the pathological re-examination of the tumour could not rule out a primary peritoneal mesothelioma. [The Working Group noted the low statistical power of this study.]

3. Studies of Cancer in Experimental Animals

3.1 Inhalation exposure

Rat: Two groups of 78 male Fischer 344 rats, five to six weeks of age, were exposed to 10 mg/m^3 (360 fibres/mL) commercial wollastonite (NYAD-G from NYCO, Inc., Willsboro, NY, United States) by inhalation. These rats were exposed in inhalation chambers for 6 h per day, on five days per week for either 12 months or for 24 months. Two additional groups of 78 male Fischer 344 rats per group served as controls. One of these groups was an untreated chamber control and the other was a positive control exposed to chrysotile asbestos for 12 months at a concentration of 10 mg/m^3, which corresponded to about 1000 fibres/mL. Scanning electron microscopic characterization of this wollastonite sample revealed a diameter range of 0.1–1.0 μm; 15% of the fibres had a length > 5 μm. [The Working Group noted that the small number of fibres measured (117) was insufficient for a sound characterization of the sample.] The number of fibres with a length ≥ 5 μm, a diameter ≤ 3 μm and aspect ratio ≥ 3 : 1 would be approximately 54 fibres/mL. At three, 12 and 24 months after the start of the experiment, six rats from each exposure group were killed. The remaining rats were held for lifetime observation (until 90% mortality) in each of the groups. Survival of wollastonite- and chrysotile-exposed rats was comparable to that of the controls. Histopathological examination of the lungs of rats held for lifetime showed that wollastonite did not cause an increased tumour rate compared to controls. In the wollastonite-exposed animals, the incidence of interstitial fibrosis was 0/57 in the group treated for 12 months and 1/60 in the group treated for 24 months; the incidence in the chrysotile-exposed group was 50/52. The incidence of bronchoalveolar adenoma or carcinoma (combined) was 1/56 in the chamber control group, 0/57 in the group treated with wollastonite for 12 months, 1/60 in the group treated with wollastonite for 24 months and 20/52 in the chrysotile-exposed group (McConnell *et al.*, 1991). [The Working Group noted the low number of wollastonite fibres in the exposure atmosphere with a fibre length > 5 μm.]

3.2 Intrapleural administration

Rat: Groups of 30–50 female Osborne-Mendel rats, 12–20 weeks of age, received 40 mg/animal wollastonite uniformly dispersed in hardened gelatin directly on the left pleural surface by open thoracotomy (Stanton & Wrench, 1972). It was reported that the four following separate grades of wollastonite with a length > 4 μm and a diameter < 2.5 μm from the same Canadian mine were used [composition and purity of the different grades unspecified]: 'grade 1' wollastonite contained 3.5 × 10^3 fibres/μg; 'grade 2' wollastonite, 2.7 × 10^3 fibres/μg; 'grade 3' wollastonite, 5.0 × 10^3 fibres/μg; and 'grade 4' wollastonite, 0.26 × 10^3 fibres/μg. The corresponding 40-mg doses of these four samples were 140 × 10^6 fibres, 108 × 10^6 fibres, 200 × 10^6 fibres and 10.4 × 10^6 fibres, respectively. [The Working Group noted that the number of fibres was low.] The rats were followed for two years and survivors were then killed. The incidences of pleural sarcomas were as follows: grade 1, 5/20; grade 2, 2/25; grade 3, 3/21; grade 4, 0/24. The corresponding incidence for a positive control group of animals treated with 40 mg UICC crocidolite asbestos was 14/29. In contrast, the incidences of pleural sarcomas was 3/491 from historical controls and 17/615 in a control group receiving pleural implants of 'non-fibrous materials' described by the authors as 'non-carcinogenic' (Stanton *et al.*, 1981). [The Working Group noted the lack of data on the composition and purity of the samples and on the survival of the rats and that none of the grades of wollastonite contained fibres > 8 μm in length and < 0.25 μm in diameter (the hypothetical range for maximal carcinogenesis: Stanton *et al.*, 1981); all the grades contained fibres 4–8 μm in length and < 0.25–0.5 μm in diameter, except grade 4 which contained relatively few fibres of these dimensions.]

3.3 Intraperitoneal administration

Rat: A group of 54 female Wistar rats, eight weeks of age, received five weekly intraperitoneal injections of 20 mg/animal wollastonite in saline. The wollastonite sample was from India. The number of fibres in this sample with a length > 5 μm, diameter < 3 μm and aspect ratio > 5 : 1 was 430 × 10^6; the median fibre length was 8.1 μm and the median fibre diameter was 1.1 μm. After treatment, the median life span of the rats was 107 weeks. No abdominal tumours (mesothelioma or sarcoma; excluding tumours of the uterus) were found in post-mortem examinations of the abdominal cavities of the rats. In contrast, in a positive control group treated with 0.05 mg actinolite, 15/36 abdominal tumours were observed and median survival was 101 weeks. In a negative control group receiving the same number of injections of saline, the incidence of abdominal tumours was 2/102 and median survival was 111 weeks (Pott *et al.*, 1987, 1989).

A group of 50 female Wistar rats, aged 11–12 weeks, was treated with two intraperitoneal injections of a suspension of 30 mg wollastonite in saline (obtained from the company Eternit, Kapelle, Belgium) with a time interval of one week between injections. The median fibre length in this sample was 5.6 μm and the median fibre diameter 0.71 μm. The animals were sacrificed when moribund, and surviving animals were killed 130 weeks after the start of treatment. No abdominal tumours were observed. In a positive control group treated with 3 mg crocidolite, abdominal tumours were observed in

32/50 rats. In a negative (saline) control group, no abdominal tumour was detected (0/50) (Muhle *et al.*, 1991; Rittinghausen *et al.*, 1991, 1992).

4. Other Data Relevant to an Evaluation of Carcinogenicity and its Mechanisms

4.1 Deposition, distribution, persistence and biodegradability

4.1.1 *Humans*

No data were available to the Working Group.

4.1.2 *Experimental systems*

Kinetics

Warheit *et al.* (1988) tested a number of inorganic particles and fibres for complement activation *in vitro* (in serum) and compared these data with results on particle-induced macrophage accumulation *in vivo*. The fibres tested *in vitro* were wollastonite fibres (NYAD-G from NYCO, Inc., Willsboro, NY, United States), UICC chrysotile B from the Jeffrey Mine in Québec, Canada, crocidolite asbestos fibres from a UICC sample, chrysotile treated with ammonium ferrous sulfate, Code 100 fibreglass or carbonyl iron particles. Volcanic ash from Mt St Helens was used as a negative control. Fresh serum was treated with all of the fibre and particle types mentioned above at 25 mg/mL, which was determined to be the optimal particle/sera concentration for complement activation. The in-vivo studies were carried out with male Sprague-Dawley-derived rats (Crl:CD(SD)BR). These animals were exposed by inhalation to aerosols of wollastonite fibres, chrysotile asbestos fibres, crocidolite asbestos fibres, iron-treated chrysotile asbestos fibres, fibreglass, iron particles or Mt St Helens ash particles. The concentrations of these aerosols ranged from 10 to 20 mg/m^3 total mass and exposure durations were 1, 3 or 5 h. The results showed that all of the particulates that activated complement *in vitro* to varying degrees also induced alveolar macrophage accumulation at sites of particle and fibre deposition *in vivo*. In contrast, the negative control, Mt St Helens ash, did not activate complement *in vitro* and did not elicit macrophage accumulation *in vivo*. These results indicate that complement activation by inhaled particles is a mechanism through which pulmonary macrophages accumulate at sites of particle deposition.

Warheit *et al.* (1994) evaluated fibre deposition and clearance patterns to test the biopersistence of an inhaled organic fibre and an inhaled inorganic fibre in the lungs of exposed rats. Male Crl:CD BR rats were exposed for five days to aerosols of *para*-aramid fibrils (900–1344 fibrils/mL; 9–11 mg/m^3) or wollastonite fibres (800 fibres/mL; 115 mg/m^3). The lungs of exposed rats were digested to quantify dose, fibre dimensional changes over time and clearance kinetics. The results showed that inhaled wollastonite fibres were cleared rapidly with a retention half-time of less than one week. Within one month, mean fibre lengths decreased from 11 μm to 6 μm and mean fibre diameters increased from 0.5 μm to 1.0 μm.

Muhle *et al.* (1994) compared the biodurabilities of wollastonite, various glass fibres, rockwool fibres, ceramic fibres and natural mineral fibres in the lungs of rats. Sized fractions were instilled intratracheally into Wistar rats. After serial sacrifices up to 24 months after exposure, the fibres were analysed by scanning electron microscopy following low-temperature ashing of the lungs. The numbers of fibres and diameter and length distributions of fibres were measured at the various sacrifice dates. From these data, analyses could be made of the elimination kinetics of fibres from the lung in relation to fibre length. The half-times of fibre elimination from the lung ranged from about 10 days for wollastonite to more than 300 days for crocidolite.

Bellman and Muhle (1994) tested the in-vivo durability of coated (Wollastocoat) and uncoated wollastonite materials and of xonotlite ($Ca_6Si_6O_{17}(OH)_2$, a synthetic wollastonite). UICC crocidolite fibres, which are known to be of high durability, were used as a positive control. Fibres were instilled intratracheally into female Wistar rats. Rats were sacrificed at two and 14 days after instillation, as well as at one, three and six months after instillation, using low-temperature ashing of the lungs. The fibres were then analysed by scanning electron microscopy to assess the numbers and size distributions of the retained fibres. The elimination kinetics of wollastonite fibres from the lung were found to be relatively fast, with calculated half-times of 15–21 days. The coating of wollastonite in Wollastocoat had no effect on this elimination process. For the thoracic fraction of wollastonite, elimination from the lung was as fast as that for the respirable particulate fraction. The elimination kinetics of xonotlite from the lung were very fast and 85–89% of this material was eliminated by two days after instillation. The total number of crocidolite fibres decreased with a calculated retention half-time of 240 days, but the numbers of fibres > 5 μm in length were unchanged six months after exposure.

4.2 Toxic effects

4.2.1 *Humans*

Huuskonen *et al.* (1983b) made dust measurements in a limestone–wollastonite quarry and flotation plant in Finland. High concentrations of both total dust and respirable fibres were measured in some operational stages. A clinical study of 46 men who had been exposed to wollastonite at the quarry for at least 10 years was also carried out. Radiographs revealed slight lung fibrosis in 14 men and slight bilateral pleural thickening in 13 men. Sputum specimens showed normal cytology and no ferruginous bodies were found. However, flow volume curves and nitrogen single breath tests indicated the possibility of small airways disease in nine of the 46 workers.

Using chest radiography, spirometry and a questionnaire, Hanke *et al.* (1984) conducted medical and environmental surveys in 1976 and 1982 at the single wollastonite mine in the United States. Pneumoconiosis was measured in 3% of workers in 1982; it had already been present in 1976 but without signs of progression. Of the workers examined in 1982, exposure to wollastonite dust was found to affect lung function. A high dust-exposed subgroup of 52 wollastonite workers had a significantly lower FEV_1/FVC (forced expiratory volume in one second/forced vital capacity) and a significantly lower peak flow rate than 86 age-matched control workers. This effect was independent of age, height and smoking habits. These data suggest that long-term cumulative exposure to wollastonite may impair ventilatory capacity, as reflected by deterioration in pulmonary function parameters.

Shasby *et al.* (1979) studied this same cohort of wollastonite workers at the Willsboro, NY, United States, wollastonite deposit in 1976. Workers were studied for pulmonary function by physical examination and questionnaire. Overall, 104 men were included in the analysis, representing 72% of all men with at least one year of exposure since 1952. Analysis of dust collected showed the median fibre diameter to be 0.22 μm and median length to be 2.5 μm. Fibre counts by PCOM showed concentrations of 0.3 fibres/mL in the mine and 23.3 fibres/mL in the mill. The prevalence of symptoms of chronic bronchitis (23%) was higher in the study group than in workers in non-dusty occupations but was not related to years of exposure. Although some evidence was present for increased obstructive lung disease, data are confounded by different age groups. Using chest radiography, diffusing capacity and spirometry, no evidence for restrictive disease was found.

4.2.2 *Experimental systems*

(*a*) *Inhalation studies*

Warheit *et al.* (1991) assessed the pulmonary effects of short-term high-dose inhalation exposures to wollastonite (NYAD-G from NYCO, Inc., Willsboro, NY, United States) at different fibre dimensions (mass median aerodynamic diameters (MMAD), 5.8, 4.3 or 2.6 μm; mean diameters, 0.2–3.0 μm) and fibre concentrations in Crl:CD CR rats. As a positive control, rats were exposed to crocidolite fibres (UICC crocidolite; MMAD, 2.2 μm). Rats were exposed to target concentrations of 40 mg/m^3 (asbestos) or 50 or 100 mg/m^3 (wollastonite; fibre numbers, 123–835 fibres/mL) for 6 h per day for three or five days. Following these exposures, fibre-exposed rats and age-matched sham controls were evaluated at 0, 24 and 48 h, 15 days or one month after exposure. These evaluations involved analysing the enzyme and protein levels in bronchoalveolar lavage (BAL) fluids and the in-vitro phagocytic capacities of alveolar macrophages recovered from fibre-exposed rats. A 6-h inhalation exposure to crocidolite asbestos fibres (41 mg/m^3; 12 800 fibres/mL) produced a transient influx of neutrophils and eosinophils which returned to near normal levels within eight days after exposure. However, BAL fluid lactate dehydrogenase (LDH) and protein values remained elevated ($p < 0.05$) throughout the month after exposure. In contrast, wollastonite exposure produced transient pulmonary inflammatory responses and corresponding increases in lavage fluid parameters only when the MMAD was sufficiently small (i.e. 2.6 μm) and the exposure concentration exceeded 500 fibres/mL. The method of fibre aerosol generation, the fibre aerodynamic size, the aerosol concentration and corresponding fibre number and the exposure duration were all critical factors in producing wollastonite-related acute lung injury. Overall, the severity of duration of the response to wollastonite was less than that observed with crocidolite.

Warheit *et al.* (1984) assessed the effects of wollastonite from the United States (most fibres, 4–9 μm in length) on rat macrophages. Wollastonite was found to decrease significantly both the percentage of activated macrophages and the ability of macrophages to phagocytize carbonyl iron particles. However, these effects were less marked than those associated with crocidolite. In addition, using a chemotaxis bioassay, Warheit *et al.* (1984) found that wollastonite activated rat serum complement.

Male Fischer 344 rats were exposed by inhalation to 10 mg/m^3 wollastonite (NYAD-G from NYCO, Inc., Willsboro, NY, United States) (360 fibres/mL; most fibres < 5 μm) for 6 h per day, five days per week for 12 or 24 months (McConnell *et al.*, 1991). The effects of these exposures were compared to those seen in untreated chamber controls and positive controls. The latter were exposed to chrysotile asbestos (Jeffrey Mine; Canadian chrysotile) at 10 mg/m^3 (estimated to be 1000 fibres/mL) for 12 months. Six rats from each exposure group were killed after three, 12 and 24 months. The remaining rats were allowed to live out their natural life span (until 90% mortality). McConnell *et al.* (1991) found that wollastonite produced only an alveolar macrophage response, which resolved after exposure ceased without evidence of neoplasm induction. Chrysotile administered under similar conditions produced significant fibrosis, hyperplasia and a high incidence of bronchoalveolar carcinomas (see Section 3.1).

(*b*) *Intratracheal instillation*

The following respirable materials were instilled intratracheally into the lungs of male Wistar rats: wollastonite from China, NYAD wollastonite (from NYCO, Inc., Willsboro, NY, United States), crocidolite asbestos, glasswool, polypropylene or polyacrylonitrile. In the samples studied, the numbers of fibres and non-fibrous particles varied, depending on particle size. A gravimetric dose of 25 mg in 1 mL saline was used for each instillation and was estimated to contain a minimum of 3×10^9 particles/sample. Three months after exposure, the animals were killed and the lungs were evaluated for hydroxyproline content, an indicator of fibrosis. Of the rats exposed to the wollastonite from China (geometric mean fibre length and diameter, 11.6 and 1.3 μm), the lung wet weights, lipid content and lung hydroxyproline levels were significantly increased compared with those of unexposed controls and were generally comparable to the effects produced by crocidolite exposure. The NYAD wollastonite (geometric mean fibre length and diameter, 9.2 and 1.2 μm) produced a small increase compared with controls in hydroxyproline levels (Cambelova & Juck, 1994). [The Working Group noted that a single bolus of 25 mg fibrous dust may induce a non-specific granulomatous and fibrotic response. This issue was raised in a published editorial by McConnell (1995)].

(*c*) *Intraperitoneal administration*

Groups of female Sprague-Dawley rats were exposed via intraperitoneal injection to various fibre types. Five rats were injected with 100 mg each of wollastonite from India. After 26–28 months, the omenta of these rats were examined microscopically for mesothelial changes. A low level of mesothelial proliferation was observed in the omenta of these rats; no tumours were observed. In contrast, the injection of doses between 0.01 and 100 mg of dust suspended in saline solution led to a continued proliferation of submesothelial connective tissue cells and focal submesothelial fibrosis (Friemann *et al.*, 1990).

(*d*) *In-vitro studies*

Using a chemiluminescence assay, Klockars *et al.* (1990) studied the capacity of quartz and asbestos fibres to induce the generation of reactive oxygen species (ROS) by human polymorphonuclear leukocytes. Neutrophils were incubated with the following fibre preparations: wollastonite from Finland, wollastonite from the United States, UICC chrysotile A, amosite, crocidolite, anthophyllite from Finland (PT 311) and Fyle quartz particles. The size distributions and numbers of the fibrous samples were similar. On an equal weight basis, the particulates induced chemiluminescence in the following order of magnitude: chrysotile, quartz > amosite, crocidolite > anthophyllite, wollastonite.

Leanderson and Tagesson (1992) investigated the ability of different mineral fibres (wollastonite, rockwool, glasswool, ceramic fibres, UICC chrysotile A, UICC chrysotile B, amosite, crocidolite, anthophyllite and erionite) to stimulate hydrogen peroxide (H_2O_2) and hydroxyl radical ($OH^·$) formation in mixtures containing human polymorphonuclear leukocytes. Fibre numbers or dimensions were not given. All the fibres tested caused considerable H_2O_2 formation, with twice as much H_2O_2 measured from mixtures containing natural fibres (wollastonite, asbestos and erionite) compared to mixtures containing man-made fibres (rockwool, glasswool and ceramic fibres). In addition, the natural fibres such as wollastonite induced the generation of three times more H_2O_2 and $OH^·$ in the presence of externally added iron than synthetic fibres.

Aslam *et al.* (1992) compared the cytotoxic effects of three different samples of wollastonite from India (100 µg in an unspecified volume) with that of chrysotile asbestos using rat hepatocyte cultures. The fibre preparations were not described. End-points were malondialdehyde formation (lipid peroxidation) and intracellular glutathione content. Less lipid peroxidation occurred when hepatocytes were incubated with wollastonite than with chrysotile.

Using human red blood cell suspensions, Alsam *et al.* (1995) compared the toxicity of three commercial samples of wollastonite from India with that of chrysotile. Dust suspensions were added to the cell suspensions to obtain final dust concentrations of 1.0–5.0 mg/mL. Compared with the chrysotile samples, the wollastonite samples caused less haemolysis and less lipid peroxidation in the erythrocytes.

Hedenborg *et al.* (1990) incubated 10×10^6 human polymorphonuclear leukocytes with wollastonite fibres from Finland, UICC chrysotile or UICC crocidolite (final concentrations, 100–800 µg/mL) for 30 min. Overall, the activities of collagenase, cathepsin G, elastase and LDH in the cell-free supernatant were lower after wollastonite exposure than after exposure to the asbestos samples.

Using a chemiluminescent assay, Hedenborg and Klockars (1987) tested the ability of wollastonite to induce the production of ROS in human polymorphonuclear leukocytes. Only slight chemiluminescence was detected after exposure of the cells to wollastonite.

Hahon *et al.* (1980) showed that wollastonite enhanced the induction of interferon by influenza virus in mammalian (LLC-MK2) cell monolayers but the mineral *per se* did not induce interferon. This effect was dose-, particle size- and time-dependent. A ‘synergistic effect’ on viral induction of interferon was noted when cell cultures were interferon-primed and then treated with wollastonite.

Nyberg and Klockars (1990) found that, compared with quartz and chrysotile, wollastonite from Finland was a weak inducer of lucigenin-dependent chemiluminescence by adherent human monocytes.

Using a tracheal organ culture system, Keeling *et al.* (1993) demonstrated that exposure to cigarette smoke increased the uptake of asbestos fibres by tracheal epithelial cells and that this process was mediated by ROS. Further studies, in which tracheal explants prepared from female Sprague-Dawley rats were exposed to cigarette smoke or air and then to several mineral dusts, showed that cigarette smoke did not significantly increase the epithelial uptake of wollastonite.

Skaug *et al.* (1984) assessed the effects on mouse peritoneal macrophage viability and lysosomal enzyme release after the addition of each of the following fibre or particle types: naturally occurring wollastonite from the United States, naturally occurring wollastonite from Finland, synthetic fibrous wollastonite, synthetic fibrous tobermorite, and synthetic non-fibrous tobermorite; DQ 12 quartz was used as a positive control. The two naturally occurring wollastonites were found to induce the selective release of β-glucuronidase. The synthetic fibrous tobermorite was cytotoxic. Skaug and Gylseth (1983) compared the haemolytic activities of these fibre and particle types; UICC chrysotile B was used as a positive control. The haemolytic activities of the three synthetic compounds was found to be higher than those of the naturally occurring wollastonites.

Using A549 cells (human lung type II epithelial cell line) and human bronchial epithelial cells, Rosenthal *et al.* (1994) compared the effects of crocidolite and wollastonite on the production of the chemotactic cytokine interleukin-8 (IL-8) in the absence of endogenous stimuli. Stimulation of epithelial cells by asbestos provoked the induction of IL-8; stimulation by wollastonite did not.

Pailes *et al.* (1984) exposed cultures of rabbit alveolar macrophages to chrysotile, wollastonite or latex beads for three days at concentrations ranging from 50 to 250 μg/mL. Measurements of biochemical indices of cytotoxicity indicated that chrysotile was cytotoxic. In contrast, wollastonite caused no significant effects on rabbit macrophages.

4.3 Reproductive and developmental effects

No data were available to the Working Group.

4.4 Genetic and related effects

4.4.1 *Humans*

No data were available to the Working Group on the genetic effects of wollastonite in exposed humans.

4.4.2 *Experimental systems* (see also **Table 5** and Appendices 1, 2 and 3)

Liu *et al.* (1993) produced morphological transformation of Syrian hamster embryo cells after a single exposure to a sample of wollastonite from China (62% of the fibres

> 5 μm; 64.5% < 1 μm in diameter) of concentration 20 μg/mL. The transformation rate induced by *N*-methyl-*N*-nitro-*N*-nitrosoguanidine was also elevated after several exposures to wollastonite.

Koshi *et al.* (1991) exposed Chinese hamster CHL cells to a wollastonite sample (mostly long and thick fibres) from Québec, Canada, for 48 h. Neither chromosomal aberrations nor polyploidy were induced.

Table 5. Genetic and related effects of wollastonite

Test system	Result[a]		Dose[b] (LED/HID)	Reference
	Without exogenous metabolic system	With exogenous metabolic system		
CIC, Chromosomal aberrations, Chinese hamster CHL cells *in vitro*	–	NT	300	Koshi *et al.* (1991)
AIA, Polyploidy, Chinese hamster CHL cells *in vitro*	–	NT	300	Koshi *et al.* (1991)
TCS, Cell transformation, Syrian hamster embryo cells *in vitro*	+	NT	20	Liu *et al.* (1993)

[a] +, positive; (+), weak positive; –, negative; NT, not tested; ?, inconclusive

[b] LED, lowest effective dose; HID, highest ineffective dose; in-vitro tests, μg/mL; in-vivo tests, mg/kg bw/day

5. Summary of Data Reported and Evaluation

5.1 Exposure data

Wollastonite is a calcium silicate mineral that occurs naturally in deposits in several areas of the world. Wollastonite has been mined in commercial quantities since the 1950s and its production is increasing with its use as a replacement for asbestos. Wollastonite breaks down during processing (crushing and grinding) into fibres of varying aspect ratios. High-aspect ratio wollastonite is used mainly as an asbestos replacement in construction and insulation board and automotive friction products, and in plastics and rubber. Powdered (milled) wollastonite, including small amounts of synthetic wollastonite, is used mainly in ceramics (the major current application of wollastonite) and in metallurgy. Occupational exposure to wollastonite occurs during its mining, milling, production and use.

5.2 Human carcinogenicity data

In the only available small cohort mortality study of workers in a wollastonite quarry, the observed numbers of deaths from all cancers combined and lung cancer were lower than expected.

5.3 Animal carcinogenicity data

Wollastonite was tested for carcinogenicity in an inhalation study in rats. No increase in tumour incidence was observed, but the number of fibres with a length > 5 μm and a diameter < 3 μm was relatively low (about 54 fibres/mL). Therefore, this study has only a limited value for an evaluation of carcinogenicity.

Four grades of wollastonite of different fibre sizes were tested for carcinogenicity in one experiment in rats by intrapleural implantation. There was no information on the purity of the four samples used. A slight increase in the incidence of pleural sarcomas was observed with three grades, all of which contained fibres greater than 4 μm in length and less than 0.5 μm in diameter. Pleural sarcomas were not observed after implantation of the grade that contained relatively few fibres with these dimensions.

In two studies by intraperitoneal injection in rats using two samples of wollastonite (one from India and one of unspecified origin with median fibre lengths of 8.1 μm and 5.6 μm, respectively), no intra-abdominal tumours were found.

5.4 Other relevant data

Evidence from wollastonite miners suggests that occupational exposure can cause impaired respiratory function and pneumoconiosis. However, animal studies have demonstrated that wollastonite fibres have low biopersistence and induce a transient inflammatory response compared to various forms of asbestos. A two-year inhalation study in rats at one dose showed no significant inflammation or fibrosis.

A sample of wollastonite from China produced morphological transformation of Syrian hamster embryo cells. A sample of wollastonite from Québec, Canada, induced polyploidy but not chromosomal aberrations in cultured Chinese hamster lung cells.

5.5 Evaluation[1]

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

There is *inadequate evidence* in experimental animals for the carcinogenicity of wollastonite.

Overall evaluation

Wollastonite *cannot be classified as to its carcinogenicity to humans (Group 3).*

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[1] For definition of the italicized terms, see Preamble, pp. 24–27

6. References

Andrews, R.W. (1970) *Wollastonite*, London, Her Majesty's Stationery Office

Anon. (1969) Wollastonite comes of age, but still a way to go before commercial maturity is reached. *Ind. Miner.*, **April**, 8–15

Anon. (1975) Wollastonite: USA dominates both production and consumption. *Ind. Miner.*, **July**, 15–17, 21–23, 29

Anon. (1995) *Réglementation sur la Qualité du Milieu de Travail* [*Regulations of the Conditions at the Workplace*], 1995, Québec, Canada, Editeur officiel du Québec

Aslam, M., Ashquin, M. & Rahman, Q. (1992) In vitro cytotoxic effects of wollastonites on rat hepatocytes: II. Lipid peroxidation and glutathione depletion. *Bull. environ. Contam. Toxicol.*, **49**, 547–554

Aslam, M., Arif, J. & Rahman, Q. (1995) Red blood cell damage by wollastonite: in vitro study. *J. appl. Toxicol.*, **15**, 27–31

Bauer, R.R., Copeland, J.R. & Santini, K. (1994) Wollastonite. In: Carr, D.D., ed., *Industrial Minerals and Rocks*, 6th Ed., Littleton, CO, Society for Mining, Metallurgy, and Exploration, pp. 1119–1128

Bellmann, B. & Muhle, H. (1994) Investigation of the biodurability of wollastonite and xonotlite. *Environ. Health Perspectives*, **102** (Suppl. 5), 191–195

British Geological Survey (1985) *World Mineral Statistics, 1979–83, Production. Exports: Imports*, London, Her Majesty's Stationery Office, p. 275

British Geological Survey (1995) *World Mineral Statistics, 1990–94, Production. Exports: Imports*, London, Her Majesty's Stationery Office, p. 305

Cambelova, M. & Juck, A. (1994) Fibrogenic effect of wollastonite compared with asbestos dust and dusts containing quartz. *Occup. environ. Med.*, **51**, 343–346

Danish National Institute of Occupational Health (1986) *Arbejdstilsynets Database for Arbejdspatsmålinger [Factory Inspectorate's Data Base for Workplace Measurements]*, Hellerup, Denmark

Deer, W.A., Howie, R.A. & Zussman, J. (1978) Single-chain silicates. In: *Rock Forming Minerals,* 2nd Ed., London, Longman, pp. 547–563

Deutsche Forschungsgemeinschaft (1996) *List of MAK and BAT Values 1996* (Report No. 32), Weinheim, VCH Verlagsgesellschaft mbH, pp. 93, 116

Elevatorski, E.A. & Roe, L.A. (1983) Wollastonite. In: Lefond, S.J., ed., *Industrial Minerals and Rocks*, 5th Ed., Vol. 2, New York, Society of Mining Engineers, pp. 1383–1390

Fattah, H. (1994) Wollastonite: new aspects promise growth. *Ind. Miner.*, **November**, 21–43

Friemann, J., Müller, K.M. & Pott, F. (1990) Mesothelial proliferation due to asbestos and man-made fibers. Experimental studies on rat omentum. *Pathol. Res. Pract.*, **186**, 117–123

Hahon, N., Booth, J.A., Boehlecke, B.A. & Merchant, J.A. (1980) Enhanced viral interferon induction by the mineral wollastonite. *J. Interferon Res.*, **1**, 49–60

Hanke, W.E. & Sepulveda, M.-J. (1983) *Health Hazard Evaluation Determination Report, NYCO, Willsboro, NY* (MHETA 81-112), Cincinnati, OH, United States National Institute for Occupational Safety and Health

Hanke, W., Sepulveda, M.-J., Watson, A. & Jankovic, J. (1984) Respiratory morbidity in wollastonite workers. *Br. J. ind. Med.*, **41**, 474–479

Harben, P.W. & Bates, R.L. (1984) *Geology of the Nonmetallics*, New York, Metal Bulletin Inc., pp. 368–371

Hedenborg, M. & Klockars, M. (1987) Production of reactive oxygen metabolites induced by asbestos fibres in human polymorphonuclear leucocytes. *J. clin. Pathol.*, **40**, 1189–1193

Hedenborg, M., Sorsa, T., Lauhio, A. & Klockars, M. (1990) Asbestos fibers induce release of collagenase by human polymorphonuclear leukocytes. *Immunol. Lett.*, **26**, 25–30

Huuskonen, M.S., Tossavainen, A., Koskinen, H., Zitting, A., Korhonen, O., Nickels, J., Korhonen, K. & Vaaranen, V. (1982) Respiratory morbidity of quarry workers exposed to wollastonite (Abstract). In: *Proceedings of the International Conference on Occupational Lung Diseases, 24–27 March 1982, Chicago, Illinois*, Park Ridge, IL, American College of Chest Physicians

Huuskonen, M.S., Tossavainen, A., Koskinen, H., Zitting, A., Korhonen, O., Nickels, J., Korhonen, K. & Vaaranen, V. (1983a) Wollastonite exposure and lung fibrosis, *Environ. Res.*, **30**, 291–304

Huuskonen, M.S., Järvisalo, J., Koskinen, H., Nickels, J., Räsänen, J. & Asp, S. (1983b) Preliminary results from a cohort of workers exposed to wollastonite in a Finnish limestone quarry. *Scand. J. Work Environ. Health*, **9**, 169–175

IARC (1987a) *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans*, Vol. 42, *Silica and Some Silicates*, Lyon, pp. 39–143

IARC (1987b) *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans*, Supplement 7, *Overall Evaluations of Carcinogenicity: An Updating of* IARC Monographs *Volumes 1 to 42*, Lyon, p. 74

Institute of Geological Sciences (1967) *Statistical Summary of the Mineral Industry, World Production, Exports and Imports, 1960–1965*, London, Her Majesty's Stationery Office, p. 411

Institute of Geological Sciences (1978) *World Mineral Statistics, 1970–74, Production: Exports, Imports*, London, Her Majesty's Stationery Office, p. 306

Keeling, B., Hobson, J. & Churg, A. (1993) Effects of cigarette smoke on epithelial uptake of non-asbestos mineral particles in tracheal organ culture. *Am. J. respir. Cell mol. Biol.*, **9**, 335–340

Klockars, M., Hedenborg, M. & Vanhala, E. (1990) Effect of two particle surface-modifying agents, polyvinylpyridine *N*-oxide and carboxymethylcellulose, on the quartz and asbestos mineral fiber-induced production of reactive oxygen metabolites by human polymorphonuclear leukocytes. *Arch. environ. Health*, **45**, 8–14

Koshi, K., Kohyama, N., Myojo, T. & Fukuda, K. (1991) Cell toxicity, hemolytic action and clastogenic activity of asbestos and its substitutes. *Ind. Health*, **29**, 37–56

Kuzvart, M. (1984) *Industrial Minerals and Rocks* (Developments in Economic Geology 18), Amsterdam, Elsevier, pp. 263–265

Leanderson, P. & Tagesson, C. (1992) Hydrogen peroxide release and hydroxyl radical formation in mixtures containing mineral fibres and human neutrophils. *Br. J. ind. Med.*, **49**, 745–749

Liu, J., Liang, S., Li, S., Xu, D. & Shen, B. (1993) Studies on the transforming effects of wollastonite on SHE cells. *Acta acad. med. sin.*, **15**, 132–137 (in Chinese)

McConnell, E.E. (1995) Fibrogenic effect of wollastonite compared with asbestos dusts and dusts containing quartz — Letter to the Editor. *Occup. environ. Med.*, **52**, 621

McConnell, E.E., Hall, L. & Adkins, B., Jr (1991) Studies on the chronic toxicity (inhalation) of wollastonite in Fischer 344 rats. *Inhal. Toxicol.*, **3**, 323–337

Muhle, H., Bellmann, B. & Pott, F. (1991) Durability of various mineral fibers in rat lungs. In: Brown, R.C., Hoskins, J.A. & Johnson, N.F. eds, *Mechanisms in Fibre Carcinogenesis* (NATO ASI Series A, Life Sciences, Vol. 223), New York, Plenum Press, pp. 181–187

Muhle, H., Bellmann, B. & Pott F. (1994) Comparative investigations of the biodurability of mineral fibers in the rat lung. *Environ. Health Perspectives*, **102** (Suppl. 5), 163–168

Nyberg, P. & Klockars, M. (1990) Measurement of reactive oxygen metabolites produced by human monocyte-derived macrophages exposed to mineral dusts. *Int. J. exp. Pathol.*, **71**, 537–544

O'Driscoll, M. (1990) Wollastonite production: Tempo rises as markets grow. *Ind. Miner.*, **December**, 15–23

Pailes, W.H., Judy, D.J., Resnick, H. & Castranova, V. (1984) Relative effects of asbestos and wollastonite on alveolar macrophages. *J. Toxicol. environ. Health*, **14**, 497–510

Pott, F., Ziem, U., Reiffer, F.-J., Huth, F., Ernst, H. & Mohr, U. (1987) Carcinogenicity studies on fibres, metal compounds and some other dusts in rats. *Exp. Pathol.*, **32**, 129–152

Pott, F., Roller M., Ziem, U., Reiffer, F.-J., Bellmann, B., Rosenbruch, M. & Huth, F. (1989) Carcinogenicity studies on natural and man-made fibres with the intraperitoneal test in rats. In: Bignon, J., Peto, J. & Saracci, R., eds, *Non-occupational Exposure to Mineral Fibres* (IARC Scientific Publications No. 90), Lyon, IARC, pp. 173–179

Power, T. (1986) Wollastonite, performance filler potential. *Ind. Miner.*, **January**, 19–34

Rittinghausen, S., Ernst, H., Muhle, H., Fuhst, R.H. & Mohr, U. (1991) Histopathological analysis of tumour types after intraperitoneal injection of mineral fibres in rats. In: Brown, R.C., Hoskins, J.A. & Johnson, N.F. eds, *Mechanisms in Fibre Carcinogenesis* (NATO ASI Series A, Life Sciences, Vol. 223), New York, Plenum Press, pp. 81–89

Rittinghausen, S., Ernst, H., Muhle, H. & Mohr, U. (1992) Atypical malignant mesotheliomas with osseous cartilaginous differentiation after intraperitoneal injection of various types of mineral fibres in rats. *Exp. Toxicol. Pathol.*, **44**, 55–58

Roberts, W., Rapp, G.R., Jr & Weber, J. (1974) *Encyclopedia of Minerals*, New York, Van Nostrand Reinhold, p. 675

Rosenthal, G.J., Germolec, D.R., Blazka, M.E., Corsini, E., Simeonova, P., Pollock, P., Kong, L.-Y., Kwon, J. & Luster, M.I. (1994) Asbestos stimulates IL-8 production from human lung epithelial cells. *J. Immunol.*, **153**, 3237–3244

Roskill Information Services Ltd (1993) The Economics of Wollastonite 1993, The Minerals Metals & Materials Society (Internet Information)

Shasby, D.M., Petersen, M., Hodous, T., Boehlecke, B. & Merchant, J. (1979) Respiratory morbidity of workers exposed to wollastonite through mining and milling. In: Lemen, R. & Dement, J.M., eds, *Dusts and Disease*, Park Forest South, IL, Pathotox Publishers, pp. 251–256

Skaug, V. & Gylseth, B. (1983) Hemolytic activity of five different calcium silicates. *Environ. Health Perspectives*, **51**, 195–203

Skaug, V., Davies, R. & Gylseth, B. (1984) In vitro macrophage cytotoxicity of five calcium silicates. *Br. J. ind. Med.*, **41**, 116–121

Stanton, M.F. & Wrench, C. (1972) Mechanisms of mesothelioma induction with asbestos and fibrous glass. *J. natl Cancer Inst.*, **48**, 797–821

Stanton, M.F., Layard, M., Tegeris, A., Miller, E., May, M., Morgan, E. & Smith, A. (1981) Relation of particle dimension to carcinogenicity in amphibole asbestoses and other fibrous minerals. *J. natl Cancer Inst.*, **67**, 965–975

Tuomi, T., Salmi, T. & Tossavainen, A. (1982) Evaluation of occupational exposure to wollastonite with scanning electron microscopy. In: SCANDEM-82 *Proceedings, Jyvaskyla, Finland,* Scandinavian Society for Electron Microscopy

United States Occupational Safety and Health Administration (1995) Air contaminants. *US Code fed. Regul.*, **Title 29**, Part 1910.1000, p. 19

Virta, R.L. (1995) *Mineral Industry Surveys: Wollastonite — Annual Review — 1994*, Washington DC, United States Department of the Interior, Bureau of Mines

Warheit, D.B., Hill, L.H. & Brody, A.R. (1984) In vitro effects of crocidolite asbestos and wollastonite on pulmonary macrophages and serum complement. *Scan. Electron Microsc.*, **ii**, 919–926

Warheit, D.B., Overby, L.H., George, G. & Brody, A.R. (1988) Pulmonary macrophages are attracted to inhaled particles through complement activation. *Exp. Lung Res.*, **14**, 51–66

Warheit, D.B., Moore, K.A., Carakostas, M.C. & Hartsky, M.A. (1991) Acute pulmonary effects of inhaled wollastonite fibers are dependent on fiber dimensions and aerosol concentrations. In: Brown, R., Hoskins, J. & Johnson, N., eds, *Mechanisms in Fibre Carcinogenesis* (NATO ASI Series A: Life Sciences, Vol 223), New York, Plenum Press, pp. 143–156

Warheit, D.B., Hartsky, M.A., McHugh, T.A. & Kellar, K.A. (1994) Biopersistence of inhaled organic and inorganic fibers in the lungs of rats. *Environ. Health Perspectives*, **102** (Suppl. 5), 151–157

Wolkem Private Ltd (undated) *Kemolit (Wollastonite). A versatile filler*, Udaipur, Rajasthan, India

Zumwalde, R. (1977) *Industrial Hygiene Study of the Interpace Corporation, Willsboro, NY*, Cincinnati, OH, United States National Institute for Occupational Safety and Health