COMMITTEE OF EXPERTS ON THE TRANSPORT OF DANGEROUS GOODS AND ON THE GLOBALLY HARMONIZED SYSTEM OF CLASSIFICATION AND LABELLING OF CHEMICALS

Sub-Committee of Experts on the Transport of Dangerous Goods

Thirty-third session
Geneva, 30 June-9 July (a.m) 2008
Item 4 of the provisional agenda

LISTING, CLASSIFICATION AND PACKING

New entry for Chrysotile, in Class 9

Addendum to ST/SG/AC.10/C.3/2008/60

Transmitted by the International Dangerous Goods and Containers Association (IDGCA)

Annex 1

DATA SHEET TO BE SUBMITTED TO THE UNITED NATIONS FOR NEW OR AMENDED CLASSIFICATION OF SUBSTANCES

Submitted by **International Dangerous Goods and Containers Association**

Date **04 April 2008**

Supply all relevant information including sources of basic classification data. Data should relate to the product in the form to be transported. State test methods. Answer all questions - if necessary state "not known" or "not applicable" - If data is not available in the form requested, provide what is available with details. Delete inappropriate words.

Section 1. SUBSTANCE IDENTITY

1.1 Chemical name **Chrysotile**

1.2 Chemical formula **$Mg_3Si_2O_5(OH)_4$**

1.3 Other names/synonyms

1.4.1 UN number **3XXX** 1.4.2 CAS number **12001-29-5**

[1] *This and similar references are to chapters and paragraphs in the Model Regulations on the Transport of Dangerous Goods.*

1.5 Proposed classification for the Recommendations

1.5.1 Proper shipping name (3.1.2[1]) **CHRYSOTILE or CHRYSOTILE FIBRE**

1.5.2 class/division .....**9**.....subsidiary risk(s)……...**NO**.....

packing group ....**III**.....

1.5.3 proposed special provisions, if any ..**168**

1.5.4 proposed packing instruction(s). **Packing instructions P002, IBC08**

Special packing provision (PP): Bags 5H2 and 5M1 are permitted when transported in closed cargo transport units or in closed rigid overpacks, or in overpacks in shrink or stretch wrap on pallets, or in flexible IBCs

Special packing provision for IBCs: B3 and B4.

Section 2. PHYSICAL PROPERTIES

2.1 Melting point or range... **1450 - 1500 °C**

2.2 Boiling point or range °C **not applicable**

2.3 Relative density at :

2.3.1 15 °C ..... **2.5-2.65 g/cm^3**

2.3.2 20 °C ..... **2.5-2.65 g/cm^3**

2.3.3 50 °C ..... **2.5-2.65 g/cm^3**

2.4 Vapour pressure at :

2.4.1 50 °C kPa **not applicable**

2.4.2 65 °C kPa **not applicable**

2.5 Viscosity at 20 °C[2] ..... m^2/s

2.6 Solubility in water at 20 °C ...**does not dissolve**..... g/100 ml

2.7 Physical state at 20°C (2.2.1.1[1]) **Solid** solid/liquid/gas[2]

2.8 Appearance at normal transport temperatures, including colour and odour ..

Colour of fibre - white, grew, green or yellow, odourless

2.9 Other relevant physical properties .....

Low electroconductivity ($1 \cdot 10^{-6}$ cm·m^{-1}),

Low heatconductivity (0.134 – 0.177 W·m^{-1} K^{-1})

[1] *This and similar references are to chapters and paragraphs in the Model Regulations on the Transport of Dangerous Goods.*

Section 3. FLAMMABILITY

3.1 Flammable vapour

3.1.1 Flash point (2.3.3[1]) ....**non applicable**..... °C oc/cc

3.1.2 Is combustion sustained? (2.3.1.3[1]) **No**

3.2 Autoignition temperature ....**non applicable**.....°C

3.3 Flammability range (LEL/UEL) .....**non applicable**.....%

3.4 Is the substance a flammable solid? (2.4.2[1]) **No**

3.4.1 If yes, give details .....

Section 4. CHEMICAL PROPERTIES

4.1 Does the substance require inhibition/stabilization or other treatment such as nitrogen blanket to prevent

hazardous reactivity ? **No**

If yes, state:

4.1.1 Inhibitor/stabilizer used . .....

4.1.2 Alternative method .....

4.1.3 Time effective at 55 °C .....

4.1.4 Conditions rendering it ineffective .....

4.2 Is the substance an explosive according to paragraph 2.1.1.1? (2.1[1]) **No**

4.2.1 If yes, give details .....

4.3 Is the substance a desensitized explosive? (2.4.2.4[1]) **No**

4.3.1 If yes, give details .....

4.4 Is the substance a self-reactive substance? (2.4.1[1]) **No**

If yes, state:

4.4.1 Exit box of flow chart .....

What is the self-accelerating decomposition temperature (SADT) for a 50 kg package? **non applicable**

Is the temperature control required? (2.4.2.3.4[1]) **No**

4.4.2 Proposed control temperature for a 50 kg package ..... °C

4.4.3 Proposed emergency temperature for a 50 kg package..... °C

[1] *This and similar references are to chapters and paragraphs in the Model Regulations on the Transport of Dangerous Goods.*

4.5 Is the substance pyrophoric? (2.4.3[1]) **No**

4.5.1 If yes, give details .....

4.6 Is the substance liable to self-heating? (2.4.3[1]) **No**

4.6.1 If yes, give details .....

4.7 Is the substance an organic peroxide (2.5.1[1]) **No**

If yes state:

4.7.1 Exit box of flow chart ... .

What is the self accelerating decomposition temperature (SADT) for a 50 kg package?....°C

Is temperature control required? (2.5.3.4.1[1]) **No**

4.7.2 proposed control temperature for a 50 kg package ..... °C

4.7.3 proposed emergency temperature for a 50 kg package..... °C

4.8 Does the substance in contact with water emit flammable gases? (2.4.4[1]) **No**

4.8.1 If yes, give details .....

…….....

.....

.....

4.9 Does the substance have oxidizing properties (2.5.1[1]) **No**

4.9.1 If yes, give details .....

…….....

.....

.....

4.10 Corrosivity (2.8[1]) to:

4.10.1 mild steel .....0.....mm/year at ..... °C

4.10.2 aluminium.....0…....mm/year at..... °C

4.10.3 other packaging materials (specify)

.....mm/year at..... . °C

.....mm/year at..... °C

4.11 Other relevant chemical properties .....

Poor resistance to acids. Chrysotile quickly dissolved in acids and in strongly caustic solutions at their boiling temperature.

[1] *This and similar references are to chapters and paragraphs in the Model Regulations on the Transport of Dangerous Goods.*

Section 5. HARMFUL BIOLOGICAL EFFECTS

5.1 LD50, oral (2.6.2.1.1[1])... ..**non applicable** mg/kg Animal species .....

5.2 LD50, dermal (2.6.2.1.2[1]) **non applicable** mg/kg Animal species .....

5.3 LC50, inhalation (2.6.2.1.3[1]) **non applicable** mg/litre Exposure time.....hours or ....ml/m^3 Animal species .....

5.4 Saturated vapour concentration at 20 °C (2.6.2.2.4.3[1]) .....**non applicable**.....ml/m^3

5.5 Skin exposure (2.8[1]) results Exposure time ..... hours/minutes

Animal species.....

5.6 Other data. **Not acutely toxic. Chrysotile has low biopersistence, Chrysotile has been shown to be rapidly removed from the lung following inhalation in experimental animals.**

Since the publication of Environmental Health Criteria 53 (IPCS, 1986), there have been only a few studies in which possible harmful effects of the ingestion of chrysotile asbestos have been examined in experimental animals. All these studies gave negative findings. Various experimental samples of chrysotile fibres have been shown in numerous long-term inhalation studies to cause fibrogenic and carcinogenic effects in laboratory rats. Exposure/dose-response relationships for chrysotile-induced pulmonary fibrosis, lung cancer and mesothelioma have not been adequately investigated in long-term animal inhalation studies (IPCS, 1998). Inhalation studies conducted to date, mainly using a single exposure concentration, show fibrogenic and carcinogenic responses at airborne fibre concentrations ranging from 100 to a few thousand fibres/ml. Carcinogenic effects have not been reported in several oral carcinogenicity studies (IPCS, 1998).

5.7 Human experience **Chrysotile can cause asbestosis, lung cancer and mesothelioma in a dose-dependent manner (IPCS, 1998). Asbestotic changes are common following prolonged exposures to 5 to 20 fibres/ml (IPCS, 1998). For chrysotile the overall relative risks for lung cancer are generally not elevated in the studies of workers in asbestos-cement production and in some of the cohorts of asbestos-cement production workers. There is no evidence of increased cancer risk from chrysotile exposure at presently regulated occupational exposure levels (~1 f/ml, 8-hour time-weighted average), as recommended by the Group of Experts convened by the WHO in Oxford (1989).**

"In 20 studies of over 100,000 asbestos workers, the standardized mortality rate ranged from 1.04 for chrysotile workers to 4.97 for amosite workers, with a combined relative risk of 2.00. It is difficult to determine the exposures involved because few of the studies reported measurements, and because it is a problem to convert historical asbestos measurements in millions of dust particles per cubic foot to gravimetric units. Nevertheless, little excess lung cancer is expected from low exposure levels." Concha-Barrientos M, et al. (2004) "Comparative Quantification of Health Risks: Global and Regional Burden of Disease Attributable to Selected Major Risk Factors". in: Ezzati M, Lopez AD, Rodgers A, Murray CJL, eds. Geneva: World Health Organization, chapter 21, pp.1651–1801.

[1] *This and similar references are to chapters and paragraphs in the Model Regulations on the Transport of Dangerous Goods.*

"…the data are consistent with the hypothesis that chrysotile has zero potency toward the induction of mesothelioma." (FINAL DRAFT: TECHNICAL SUPPORT DOCUMENT FOR A PROTOCOL TO ASSESS ASBESTOS-RELATED RISK, EPA, 2003. page 7.50)

For more information on this section please see Supporting paper Annex 2

Section 6. SUPPLEMENTARY INFORMATION

6.1 Recommended emergency action

6.1.1 Fire (include suitable and unsuitable extinguishing agents)……

6.1.2 Spillage. **In proper time to collect and remove chrysotile dust in dense plastics bags avoiding dust generation. The personnel should be equipped by personal respiratory protective masks.**

6.2 Is it proposed to transport the substance in:

6.2.1 Bulk Containers (6.8[1]) **Yes**

6.2.2 Intermediate Bulk Containers (6.5[1])? **Yes**

6.2.3 Portable tanks (6.7[1])? **No**

If yes, give details in Sections 7, 8 and/or 9.

Section 7. BULK CONTAINERS (only complete if yes in 6.2.1)

7.1 Proposed type(s) **BK2**

Section 8. INTERMEDIATE BULK CONTAINERS (IBCs) (only complete if yes in 6.2.2)

8.1 Proposed type(s)... **All types listed in packing instruction IBC08**.....

Section 9. MULTIMODAL TANK TRANSPORT (only complete if yes in 6.2.3)

9.1 Description of proposed tank (including IMO tank type if known).....

9.2 Minimum test pressure .....

9.3 Minimum shell thickness .....

9.4 Details of bottom openings, if any .....

9.5 Pressure relief arrangements .....

9.6 Degree of filling ……..

9.7 Unsuitable construction materials .....

[1] *This and similar references are to chapters and paragraphs in the Model Regulations on the Transport of Dangerous Goods.*

References

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Concha-Barrientos M, et al. (2004) "Comparative Quantification of Health Risks: Global and Regional Burden of Disease Attributable to Selected Major Risk Factors". *in*: Ezzati M, Lopez AD, Rodgers A, Murray CJL, eds. Geneva: World Health Organization, chapter 21, pp.1651–1801**.**
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WHO, IPCS, Environment Health Criteria 203, 1998, 1.4 Uptake, clearance, retention and translocation, para 3. http://www.inchem.org/documents/ehc/ehc/ehc203.htm

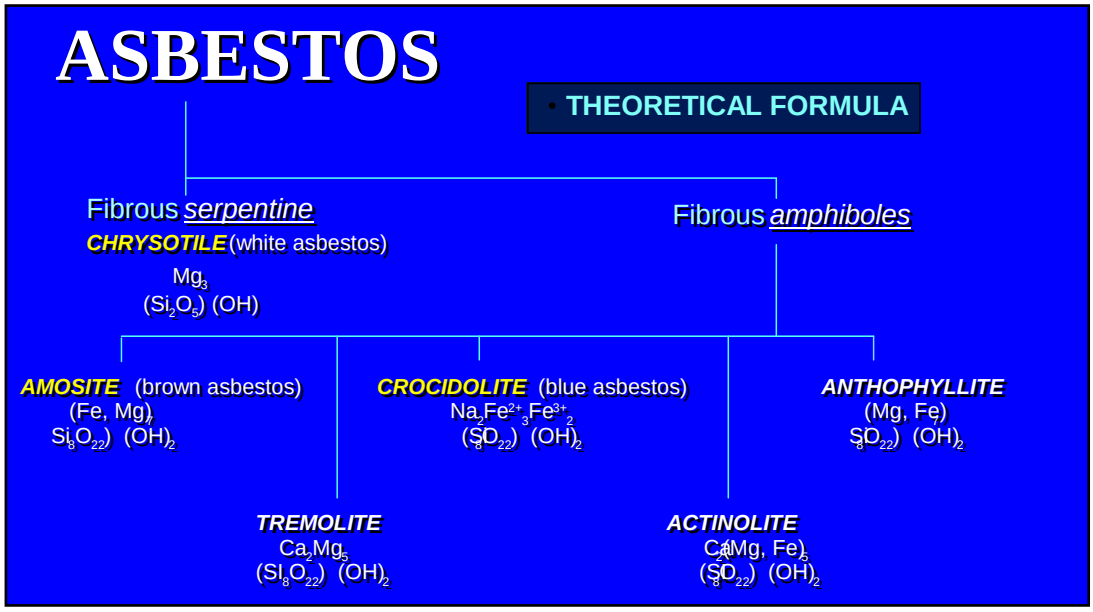
Annex 2

Technical and scientific evidences

1. The Difference between Asbestos Fibre Types

Chrysotile asbestos is often included with other asbestos materials in evaluation and classification. 'Asbestos' is not a mineral in itself. It is a collective term given to a group of minerals whose crystals occur in fibrous forms. The term 'asbestos' was adopted for the purposes of commercial identification alone.

The six minerals commonly referred to as asbestos come from two groups of minerals known as serpentines (chrysotile, white asbestos) and amphiboles (amosite, brown asbestos; crocidolite, blue asbestos; anthophyllite; tremolite; and actinolite). While they are all silicate minerals, the two groups are chemically and mineralogically distinct. In particular, their mineralogical structures are remarkably different and result in a notable difference in the manner they are processed by the lung once inhaled. Today, only one mineral chrysotile, or white asbestos, is currently mined in any quantity. This serpentine mineral has always been the principal asbestos of commerce.



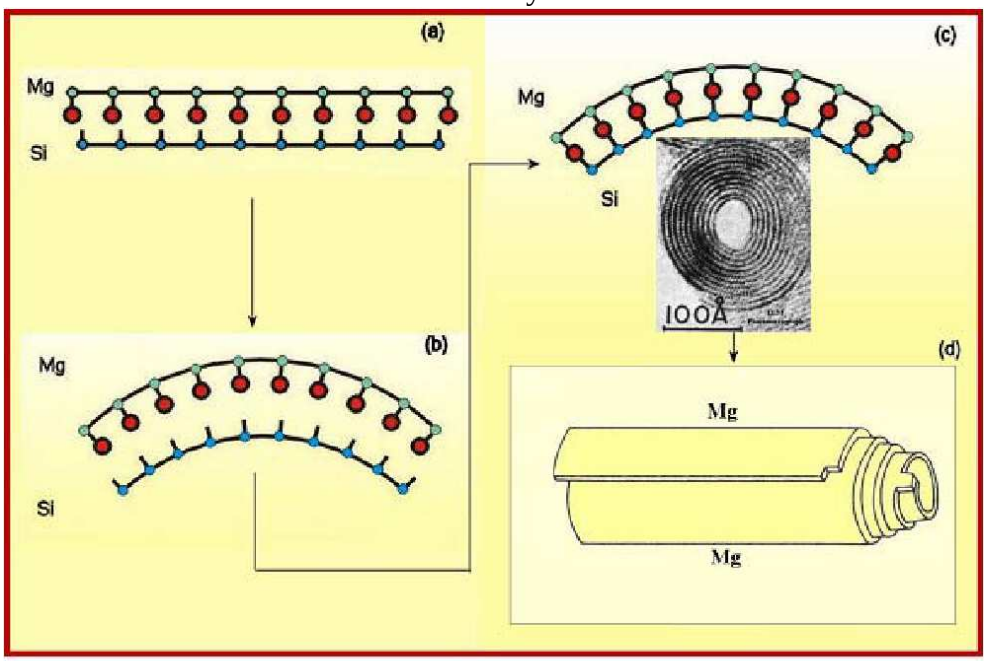
MINERALOGICAL STRUCTURE CHEMISTRY OF CHRYSOTILE AND AMPHIBOLE
Chemically all of the asbestos minerals are silicates but mineralogically and crystallographically the serpentine and amphibole groups are quite different (Deer et al., 1966).

Chrysotile
Chrysotile is a sheet silicate and instead of forming into rods (fibers) as do the amphiboles, the mismatch in spacing between the magnesium ions and the silica ions causes chrysotile to curl into effectively a thin rolled sheet (Figure 1).

The external surface of a chrysotile fibril is the magnesium mineral brucite. Hargreaves and Taylor (1946) reported that if fibrous chrysotile is treated with dilute acid the magnesia can be completely removed. The hydrated silica which remains, though fibrous in form, had completely lost the elasticity characteristic of the original chrysotile and had a structure that was "amorphous" or "glassy" in type. Wypych et al. (2005) recently examined what happens to natural chrysotile fibers when acid-leached under controlled conditions. The authors reported that the leached products consisted of layered hydrated disordered silica with a "distorted" structure resembling the silicate layer existing in the original minerals. Extensive characterization techniques confirmed the removal of the brucite-like sheets, leaving silica with an eminently amorphous structure.

Removal of magnesium from the brucite layer by acid weakens the chrysotile fibrils and eventually destroys their dimensional stability. The sensitivity of chrysotile to acid dissolution is particularly important in the lung where the macrophages in the lung are capable of generating a milieu at a pH of ~4.5. Chrysotile fibers which are cleared from the lung and swallowed would be readily attacked by the hydrochloric acid in the stomach which keeps the lumen of that organ below pH 2.

Figure 1 Structural formation of the sheet silica chrysotile asbestos



(Reproduced from Bernstein et al., 2005b)

Amphiboles

The chemical composition of the amphiboles fibers is more complex and the idealized chemical formulae of the five amphiboles are shown below. Although their structures are the same this variability in composition is a direct consequence of the fact that the silicate framework can accommodate a mixture of many different ions (as determined by the host rock) in the space between the silicate ribbons which form the fibers (Speil & Leineweber, 1969).

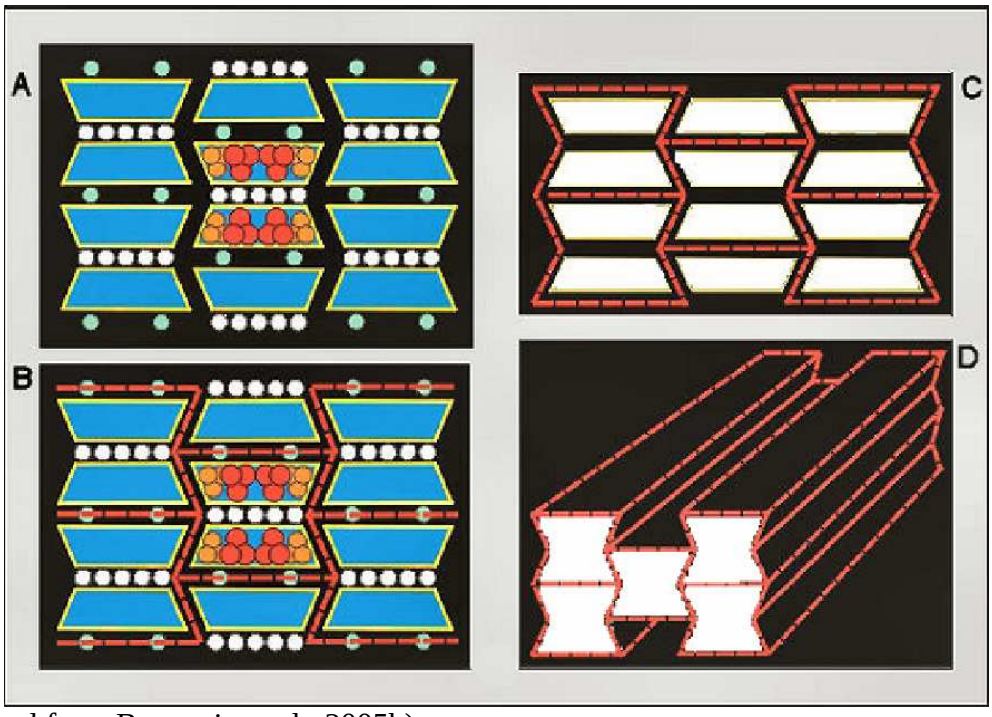
Crocidolite. . . . . (Na2Fe3 2+Fe23+) Si8O22(OH)2
Amosite. . . . . .(Fe2+, Mg)7 Si8O22(OH)2

Tremolite. . . . . Ca2Mg5 Si8O22(OH)2
Anthophyllite. . . . . (Mg, Fe2+)7 Si8O22(OH)2
Actinolite. . . . . .Ca2(Mg, Fe2+)5 Si8O22(OH)2

The external surface of the crystal structures of the amphiboles is quartz-like, and has the chemical resistance of quartz. This structure is illustrated for tremolite in Figure 2 (A-D). Each of the blue boxes in Figure 2A represents a double chain of tetrahedral silicate structures (illustrated in more detail in the middle chains). With tremolite, the white and green circles represent the magnesium and calcium cations that effectively 'glue' one chain to its neighbour. Fewer shared cations bond the chains together along the broad sides of the chains compared to the narrow sides with the result that it is along these broad surfaces weakly bonded surfaces that the mineral will most likely break (Figure 2B, red dashed lines). With tremolite, this weakly bonded region is associated with the Mg cation. Figure 2C simplifies the picture and shows that the doublechain silicates can break into a set of fragments with fibrous shape.

Figure 2D shows the same situation in 3 dimensions. The splits run along the chains and it can be seen how the fiber shape is formed. The chains themselves do not break easily because the bonds between the silica tetrahedra are very strong compared to the cationic bonds 'gluing' one chain to the next.

Figure 2 Structural formation of the double chain silica amphibole asbestos, tremolite



(Reproduced from Bernstein et al., 2005b)

Structural and chemical composition difference of asbestos fibres

There are some features of chemical composition. Although all six types of asbestos are hydrated silicates but they differ appreciably by their metal content and crystal structure.

	Chrysotile	**Crocidolite**	**Amosite**
Fe_2O_3	0-5	13-18	0-5
FeO	0-3	3-21	35-40
MgO	38-42	0-13	5-7

Table 12. Major-oxide composition of commercial chrysotile samples

[In weight percent. Information from Skinner, Ross, and Frondel, 1988, p. 32]

	Canada	**Russia**	**Zimbabwe**	**Swaziland**
SiO_2	38.75	39	39.7	39.93
Al_2O_3	3.09	4.66	3.17	3.92
Fe_2O_3	1.59	0.54	0.27	0.1
FeO	2.03	1.53	0.7	0.45
MnO	0.08	0.11	0.26	0.05
MgO	39.78	38.22	40.3	40.25
CaO	0.89	2.03	1.08	1.02
K_2O	0.18	0.07	0.05	0.09
Na_2O	0.1	0.07	0.04	0.09
H_2O^+	12.22	11.37	12.17	12.36
H_2O^-	0.6	0.77	0.64	0.92
CO_2	0.48	1.83	2.13	1.04
Total	99.79	100.2	100.51	100.22

Table 13. Major-oxide composition of amphibole asbestos

[In weight percent. --, zero. Information from Hodgson, 1979, p. 80-81]

	Amosite	**Actinolite**[1]	**Anthophyllite**	**Crocido-lite**	**Tremolite**
SiO_2	49.7	53.8	57.2	50.9	55.1
Al_2O_3	0.4	1.2	--	Nil	1.14
Fe_2O_3	0.03	1.9	0.13	16.85	0.32
FeO	39.7	25.3	10.12	20.5	2
MnO	0.22	0.4	--	0.05	0.1
MgO	6.44	4.3	29.21	1.06	25.65
CaO	1.04	10.2	1.02	1.45	11.45
K_2O	0.63	0.4	--	0.2	0.29
Na_2O	0.09	0.1	--	6.2	0.14
H_2O^+	1.83	2.6	2.18	2.37	3.52
H_2O^-	0.09	Nil	0.28	0.22	0.16
CO_2	0.09	0.2	--	0.2	0.06
Total	100.26	100.4	100.14	100	99.93

[1]Ferro-actinolite.

Table 11. Properties of asbestos fibers

[Ca, calcium; Fe, iron; Mg, magnesium; Na, sodium; NA, not available. Information from Badollet, 1951]

Property	Actinolite asbestos	Amosite	Anthophyllite asbestos	Chrysotile	Crocidolite	Tremolite asbestos
Structure	Reticulated long prismatic crystals and fibers	Lamellar or coarse to fine fibrous and asbestiform	Lamellar or fibrous asbestiform	Usually highly fibrous fibers, fine and easily separable	Fibrous in ironstones	Long or prismatic and fibrous aggregates
Veining	Slip or mass fiber	Cross fiber	Slip or mass fiber	Cross and slip fibers	Cross fiber	Slip or mass fiber
Essential composition	Ca, Mg, Fe silicate with some water	Fe, Mg silicate with some water	Mg silicate with some iron	Mg silicate with some water	Na, Fe silicate with some water	Ca, Mg silicate with some water
Crystal system	Monoclinic	Monoclinic	Orthorhombic	Monoclinic	Monoclinic	Monoclinic
Color	Greenish	Ash gray or brown	Grayish white, also brown-gray or green	White, gray, green	Lavender blue, metallic blue	Gray-white, greenish, yellowish, bluish
Luster	Silky	Vitreous to pearly	Vitreous to pearly	Silky	Silky to dull	Silky
Hardness	6±	5.5-6.0	5.5-6.0	2.5-4.0	4	5.5
Specific gravity	3.0-3.2	3.1-3.25	2.85-3.1	2.4-2.6	3.2-3.3	2.9-3.2
Optical properties	Biaxial negative extinction inclined	Biaxial positive, extinction parallel	Biaxial positive, extinction parallel	Biaxial positive extinction parallel	Biaxial positive, negative, extinction parallel	Biaxial negative, extinction inclined
Refractive index	1.63± weakly pleochroic	1.64±	1.61±	1.51-1.55	1.7 pleochroic	1.61±
Length	Short to long	2 to 11 inches, varies	Short	Short to long	Short to long	Short to long
Texture	Harsh	Coarse but somewhat pliable	Harsh	Soft to harsh, also silky	Soft to harsh	Generally harsh, sometimes soft
Specific heat, Joules per kilogram per Kelvin	505	449	488	619	468	493
Tensile strength, thousand pascals	6,895 and less	110,316 to 620,528	27,579 and less	551,581 to 689,476	689,476 to 2,068,427	6,895 to 55,158
Temperature at maximum ignition loss	NA	871° to 982° C	982° C	982° C	648° C	982° C
Filtration properties	Medium	Fast	Medium	Slow	Fast	Medium
Electric charge	Negative	Negative	Negative	Positive	Negative	Negative
Fusion point	1,393° C	1,399° C	1,468° C	1,521° C	1,229° C	1,316° C
Spinnability	Poor	Fair	Poor	Very good	Fair	Poor
Resistance to acids and alkalies	Fair	Good	Very good	Poor	Good	Good
Mineral impurities	Lime and iron	Iron	Iron	Iron, chrome, nickel, and lime	Iron	Lime
Flexibility	Poor	Good	Poor	High	Good	Poor
Resistance to heat	NA	Good, brittle at high temperature	Very good	Good, brittle at high temperature	Poor, fuses	Fair to good

"Strong acids aggressively attack chrysotile. Chrysotile also dissolves when exposed to strongly caustic solutions at their boiling temperature (Badollet, 1951). Most amphibole fiber varieties are more acid resistant than those of chrysotile, but they can experience weight losses of 2 to 23

percent through dissolution when exposed to concentrated acids at higher temperatures. Actinolite and amosite exhibit greater weight loss when exposed to acids than the other amphibole asbestos varieties owing to their higher iron contents (Hodgson, 1979, p. 83-85; Virta and Mann, 1994, p. 102)." (***Mineral Commodity Profiles—Asbestos, 2005,*** *U.S. Department of the Interior, U.S. Geological Survey, Robert L. Virta, Circular 1255–KK, page 15)*

"It is generally agreed that the inhalation of long (length generally greater than or equal to 5 micrometers), thin, and durable fibers in high concentrations over a long period of time pose the greatest health risk.
Shorter fibers penetrate deeper into the lung but longer fibers are more difficult to clear (Finkelstein and Dufresne, 1999; Agency for Toxic Substances and Disease Control, 2001, p. 6; Johnson and Mossman, 2001). Fiber solubility is suggested to be the second most critical factor. Chrysotile is more soluble than amphibole asbestos and is removed more rapidly from the lung, reducing its residence time in the lung. Duration of exposure to asbestos is important because long exposure periods increase lung burden; additionally, long and/or high exposure levels counteract the effects of fiber solubility. Some research suggests that iron content may be an important factor in asbestos-induced toxicity (Agency for Toxic Substances and Disease Registry, 2001, p. 51). While still debated, many health scientists believe that there is sufficient evidence to state that the genotoxic and carcinogenic potentials of all asbestos fiber types are not identical; in particular, mesothelial cancer may be most strongly associated with amphibole fibers Gardner and Powell, 1986; Agency for Toxic Substances and Disease Registry, 2001, p. 48 Mineral Commodity Profiles—Asbestos 6; Gibbs, 2001, p. 165; Wilson and Price, 2001; Bernstein, Chevalier, and Smith, 2003, p. 1387; Bernstein, Rogers, and Smith, 2003, p. 1247)".
(***Mineral Commodity Profiles—Asbestos, 2005,*** *U.S. Department of the Interior, U.S. Geological Survey, Robert L. Virta, Circular 1255–KK, page 47)*

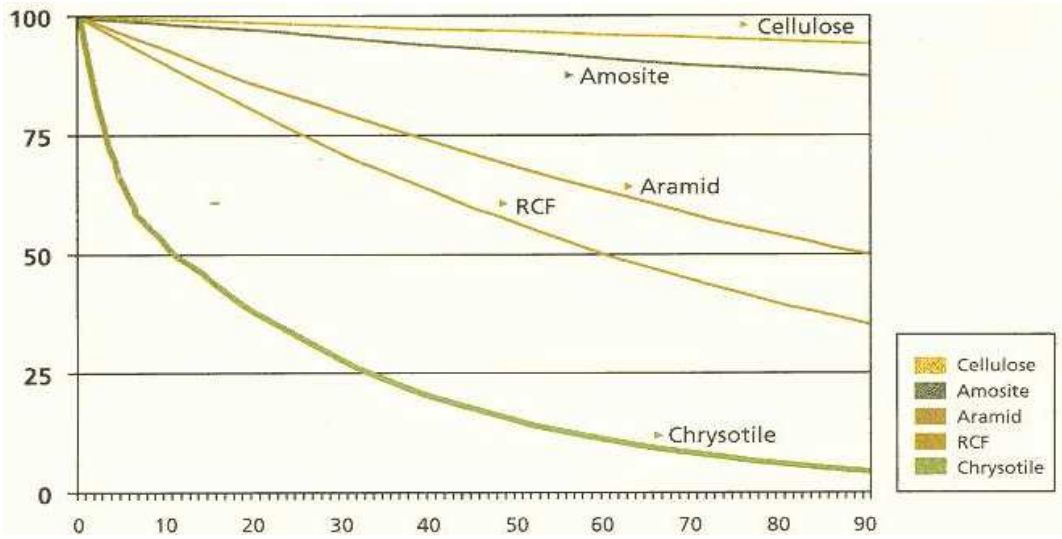
*"A further consensus developed within the scientific community regarding the relative carcinogenicity of the different types of asbestos fibers. There is strong evidence that the genotoxic and carcinogenic potentials of asbestos fibers are not identical; in particular mesothelial cancer is most strongly associated with amphibole fibers (47-51)" ("****Asbestos: Geology, Mineralogy, Mining, and Uses,*** *U.S. DEPARTMENT OF THE INTERIOR, U.S. GEOLOGICAL SURVEY, Robert L. Virta, Open-File Report 02-149", page 14)*

Biopersistence

Recent publications have clearly shown for synthetic mineral fibers the relationship of biopersistence to both chronic inhalation toxicity and chronic intraperitoneal injection tumour response in the rat (Bernstein et al, 2001a & 2001b). In essence, if the longer fibers which the macrophage can not fully engulf dissolve or break rapidly and disappear from the lung, they do not cause a carcinogenic effect. This concept was incorporated, in 1997, into the European Commissions Directive on man made mineral fibers (European Commission, 1997).
Chrysotile has been shown to be rapidly removed from the lung following inhalation in experimental animals (Bernstein et al., 2003a, 2003b, 2004, 2005a, 2005b). In addition, lung analysis of workers exposed to predominately chrysotile show low levels of chrysotile compared to amphiboles (Albin et al., 1994) even when amphibole exposure was only a trace impurity (Rowlands et al., 1982).

As chrysotile is a naturally occurring mined fiber, it is not surprising that there are some slight differences in biopersistence depending on the origin and commercial grade tested. However, across the range of mineral fiber solubilities chrysotile lies towards the soluble end of the scale and ranges from the least biopersistent fiber to a fiber with biopersistence in the range of glass and stone wools. It is less biopersistent than the ceramic fibers tested or the special-purpose glasses (Hesterberg et al., 1998a) and considerably less biopersistent than amphiboles.

"Recent studies on the serpentine asbestos chrysotile have shown that it is not very biopersistent in the lung. Serpentine asbestos is a naturally occurring mined fiber, and there appear to be some differences in biopersistence depending on where it was mined. However, chrysotile lies at the soluble end of this scale and ranges from being the least biopersistent fiber to a fiber with a biopersistence similar to that of glass and stone wools. It is less biopersistent than ceramic and special-purpose glasses and, more than an order of magnitude, less biopersistent than amphiboles.''
(David M. Bernstein, Fiber Toxicology, Chapter 11 "Toxicology of the lung" Published in 2006 by CRC Press Taylor & Francis Group, Page 467, 468)



"The more rapid removal of chrysotile fibres from the human lung is further supported by findings from animal studies showing that chrysotile is more rapidly cleared from the lung than are amphiboles including crocidolite and amosite. (IPCS, EHC 203, 1998, 1.4 Uptake, clearance, retention and translocation, para 3)

"Chrysotile is much more soluble than crocidolite and, consequently, a chrysotile fiber exhibits a much shorter residence time in the body than a comparable-sized crocidolite fiber. Based on in vitro studies, a chrysotile fiber with a diameter of 1 :m, will dissolve in body fluid in approximately 1 year ,whereas a 1-:m crocidolite fiber will take 60 years to dissolve (see Section 6.2.4)''. (FINAL DRAFT: TECHNICAL SUPPORT DOCUMENT FOR A PROTOCOL TO ASSESS ASBESTOS-RELATED RISK, US EPA, 2003, page 7.32) http://www.aeolusinc.com/Protocol_TBD_2003.pdf

"For mineral fibers, the clearance half-time of fibers longer than 20 pm ranges from a few days to less than 100 d. This is illustrated in Table 11.1. Also shown in this table are the results from biopersistence studies performed on chrysotile and amphiboles using the same protocol. For SVFs, the European Commission has established a directive that states that if the inhalation biopersistence clearance half-time of a fiber is less than 10 days, then it is not classified as a carcinogen. Recent studies on the serpentine asbestos chrysotile have shown that it is not very biopersistent in the lung. Serpentine asbestos is a naturally occurring mined fiber, and there appear to be some differences in biopersistence depending on where it was mined. However, chrysotile lies at the soluble end of this scale and ranges from being the least biopersistent fiber to a fiber with a biopersistence similar to that of glass and stone wools. It is less biopersistent than ceramic and special-purpose glasses and more than an order of magnitude less biopersistent than amphiboles.
(David M. Bernstein, Fiber Toxicology, Chapter 11 "Toxicology of the lung" Published in 2006 by CRC Press Taylor & Francis Group, Page 467, 468) Please see table 1.1.1 below

Table 11.1 Clearance Half-Times of a Range of Mineral Fibers as Determined by the Fiber Inhalation Biopersistence Protocol

Fiber	*Type*	*Weighted $T_{1/2}$ Fibers L > 20 μm (days)*	*Reference*
Calidria chrysotile	Serpentine asbestos	0.3	Bernstein et al., 2005
Brazilian chrysotile	Serpentine asbestos	2.3	Bernstein et al., 2004a
Fiber B	B01.9	2.4	Bernstein et al., 1996
Fiber A	Glass wool	3.5	Bernstein et al., 1996
Fiber C	Glass wool	4.1	Bernstein et al., 1996
Fiber G	Stone wool	5.4	Bernstein et al., 1996
MMVF34	HT stone wool	6	Hesterberg et al, 1998b
MMVF22	Slag wool	8	Bernstein et al., 1996b
Fiber F	Stone wool	8.5	Bernstein et al., 1996
MMVF11	Glass wool	9	Bernstein et al., 1996
Fiber J	X607	9.8	Bernstein et al., 1996
MMVF 11	Glass wool	13	Bernstein et al., 1996
Fiber H	Stone wool	13	Bernstein et al., 1996
Canadian chrysotile	Serpentine asbestos	11.4	Bernstein et al., 2004b
MMVF10	Glass wool	39	Bernstein et al., 1996
Fiber L	Stone wool	45	Bernstein et al., 1996
MMVF33	Special-purpose glass	49	Hesterberg et al., 1998b
RCF1a	Refractory ceramic	55	Hesterberg et al., 1998b
MMVF21	Stone wool	67	Hesterberg et al., 1998b
MMVF32	Special-purpose glass	79	Hesterberg et al., 1998b
MMVF21	Stone wool	85	Bernstein et al., 1996
Amosite	Amphibole asbestos	418	Hesterberg et al., 1998
Crocidolite	Amphibole asbestos	536	Bernstein et al., 1996

Mesothelioma

From the 1977 study by Weiss to the most recent investigation by Yarborough (2006), studies have consistently demonstrated **an undetectable risk for mesothelioma in factories when chrysotile only is used.**

"Increasing male mesothelioma incidence for many years was undoubtedly the result of exposure to asbestos. The high mesothelioma risk was prominently influenced by exposure to amphibole asbestos (crocidolite and amosite), which reached its peak usage in the 1960s and declined after that. A differing pattern in some other countries (continuing rise in incidences) may be related to the greater quantity and late ruse of amphibole, particularly crocidolite. The known latency period for the development of this tumour provides biological plausibility for the recent decline in mesothelioma incidence in the USA. This favourable finding is contrary to a widespread fear that asbestos related health effects will show an inevitable increase in coming years, or even decades."(H Weill, J M Hughes and A M Churg, "Changing trends in US mesothelioma incidence", Occup Environ Med 2004;61:438–441. doi: 10.1136/oem.2003.010165, page 1)
"The expert panelists unanimously agreed that the epidemiology literature provides compelling evidence that amphibole fibers have far greater mesothelioma potency than do chrysotile fibers—a finding reported both in the review document (Berman and Crump 2001) and a recent re-analysis of 17 cohort studies (Hodgson and Darnton 2000) that reported at least a 500-fold difference in potency. Two panelists commented further that the epidemiology literature provides no scientific support for chrysotile exposures having a role in causation of mesothelioma—an observation that is generally consistent with the meta-analysis in the proposed protocol, which failed to reject the hypothesis that chrysotile fibers have zero potency for mesothelioma.
(Report on the Peer Consultation Workshop to Discuss a Proposed Protocol to Assess Asbestos-Related Risk, EPA, 2003, page 3-13)

"Amphibole is estimated as being about four times as potent as chrysotile for lung cancer (although the difference is not significant) and about 800 times as potent as chrysotile for mesothelioma (a highly significant difference). Moreover, the data are consistent with the hypothesis that chrysotile has zero potency toward the induction of mesothelioma.(FINAL DRAFT: TECHNICAL SUPPORT DOCUMENT FOR A PROTOCOL TO ASSESS ASBESTOS-RELATED RISK, EPA, 2003. page 7.50)

"*The epidemiological evidence from asbestos workers and well-conducted animal tests shows that while all types of asbestos share the same hazards (e.g. the potential of an early death from lung cancer, asbestosis and mesothelioma) they have varying degrees of risk (the likelihood that death from one of the hazards will occur). The relative risk from the same level of exposure but to different asbestos fibre types is shown in figure 1(as derived by Hodgson and Darnton, 2000). The relative risk from crocidolite asbestos is some 500 times greater than chrysotile asbestos and the relative risk from amosite asbestos is 100 times greater than chrysotile asbestos. This means that the type/s of asbestos in the product are particularly significant when assessing risk".("A COMPARAISON OF THE RISKS FROM DIFFERENT MATERIALS CONTAINING ASBESTOS", HSE, 2006).*

*"Assuming a **mixed** fibre type, the lifetime risk of death from malignant mesothelioma is approximately 100 per 100 000/fibre.year per ml. (This combined estimate is based on best*

estimates of risk of 400 per 100 000/fibre.year per ml for crocidolite, 65 per 100 000/fibre.year per ml for amosite ***and 2 per 100 000/fibre.year per ml for chrysotile,*** *and the changing mixture of amphiboles and chrysotile that has characterized exposure 20 and 50 years ago [Hodgson and Darnton, 2000].)" (Driscoll T et al, "The global burden of disease due to occupational carcinogens", 2005, page 7)* http://www.who.int/quantifying_ehimpacts/global/2carcinogens.pdf

Epidemiology

There are two the most important studies in term of cohort dimension were done by Liddell, McDonald & McDonald in 1997and Hodgson & Darton in 2000. Liddell and McDonald's study has **shown no evidence of increased cancer risk from chrysotile exposure at presently regulated occupational exposure levels** (~1 f/ml, 8-hour time-weighted average), as recommended by the Group of Experts convened by the WHO in Oxford (1989).

Hodgson & Darton's study based on analysis of 17 cohort studies has shown relative risk **differentiated by fibre types** that gave the following results:

For CROCIDOLITE (blue asbestos) 400/100 000/fibre year per ml
For AMOSITE (brown asbestos) 65/100 000/fibre year per ml
For CHRYSOTILE (white asbestos) 2/100 000/fibre year per ml.

Hodgson JT and Darnton A (2000) The Quantitative Risks of Mesothelioma and Lung Cancer in Relation to Asbestos, Ann. Occup. Hyg. 44(8) : 565-601

19 October 2006 IARC published an article in Occupational and Environment Medicine entitled: "Occupational exposure to asbestos and man-made vitreous fibres and risk of lung cancer: a multicenter case-control study in Europe," that was conducted during the period 1998-2002 in seven European countries by Sixteen scientific centers.

Their Conclusion: "*In this large community–based study occupational exposure to asbestos and MMVF does not appear to contribute to the lung cancer burden in men in Central and Eastern Europe. In contrast, in the UK we found an increased risk of lung cancer following exposure to asbestos.* ***Differences in fibre type and circumstances of exposure may explain these differences****."*

Amongst the studies that the multicenter case-control study in Europe refers to we have two interesting.

Mortality of workers at two asbestos-cement plants in Poland
Neonila Szeszenia-Dj\Browska, Department of Occupational and Enrironmental Epidemiology
The Norer Institute or Occupational Medicine, Lodi, Poland
International Journal of Occupational Medicine and Environmental Health, Vol. 13, No 2, 121-130, 2000

"The findings of the studies on cancer risk cited above are, however, contradictory. ln the cohorts of workers from the plants employing chrysotile asbestos no excessive cancer risk was found (6,7,16,22). ln the plants where crocidolite prevailed, an excess was noted in the number

of cases of malignant neoplasms generally and of the following sites: lung, larynx, pleura, mediastinum, colon and rectum, liver, kidney and male genitals (10,11,15,18,19,23). ln view of the fact that no clear evidence of excessive cancer risk among workers of asbestos cement plants has been found, in 1993 the Supreme Court of the United States of America cancelled the ban on the use of asbestos cement products (5).”

Cancer mortality and morbidity among Lituanian asbestos-cement producing workers.
Giedre Smailyte, Msc, Juozas Kurtinaitis, PhD, Aage Andersen
Scand J Work Environ Health 2004:30 (1):64-70

“Methods. The study included 1887 asbestos-cement workers, 1285 men and 602 women, and 37 000 person-years. The two factories were active from 1956 (A) and 1963 (B), and the workers were observed from 1978 to 2000. The analysis was based on a comparison between the observed and expected numbers of cancer and causes of death. The observed numbers of cancer were obtained through linkage with the national cancer registry. The date and causes of death were obtained from two different sources. The expected numbers were calculated on the basis of gender-and age-specific incidence and mortality rates in 5-year periods from the whole country. Standardized incidence ratios (SIR) and standardized mortality ratios (SMR) and 95% confidence intervals (95% CI) were calculated. Duration of employment and time since first exposure were used as indicators of exposure. Results During the follow-up, 1978-2000,473 deaths were observed versus 489 expected. There was no excess risk of deaths from nonmalignant respiratory diseases, except for an elevated risk of mortality in relation to the digestive organs other than cancer, 18 observed versus 12.2 expected (95% CI 0.9-2.3). There was no excess risk for any types of cancer, except for colorectal cancer in men, 17 observed cases (SIR 1.6, 95% CI 1.6-2.6) and one case of mesothelioma in a woman.

Conclusions. This study on asbestos-ex posed workers did not show any excess risk of respiratory cancer or deaths of pneumoconiosis.”

Yarborough (2006) recently reviewed 71 asbestos cohorts and concluded that the evidence does not support the hypothesis that chrysotile, uncontaminated by amphibole fibers, causes mesothelioma.

“Although epidemiological studies have confirmed amphibole asbestos fibres as a cause of mesothelioma, the link with chrysotile remains unsettled. An extensive review of the epidemiological cohort studies was undertaken to evaluate the extent of the evidence related to free chrysotile fibre, with particular attention to confounding by other fibre types, job exposure concentrations, and consistency of findings. The review of 71 asbestos cohorts exposed to free asbestos ***fibres does not support the hypothesis that chrysotile, uncontaminated by amphibolic substances, causes mesothelioma.”*** *Yarborough C M (2006) Chrysotile as a Cause of Mesothelioma: An assessment based on Epidemiology. Critical Reviews in Toxicology 36 : 165-187*

The Concha-Barrientos et al report (2004), published under the aegis of the WHO, acknowledges that there is a difference in risk between chrysotile asbestos and the amphibole varieties. In chapter 21, p.1687, the authors state: *“Currently, about 125 million people in the world are exposed to asbestos at the workplace. According to global estimates at least 90,000 people die*

each year from asbestos-related lung cancer. In 20 studies of over 100,000 asbestos workers, the standardized mortality rate ranged from ***1.04 for chrysotile workers*** *to* ***4.97 for amosite workers****, with a combined relative risk of 2.00. It is difficult to determine the exposures involved because few of the studies reported measurements, and because it is a problem to convert historical asbestos measurements in millions of dust particles per cubic foot to gravimetric units. Nevertheless,* ***little excess lung cancer is expected from low exposure levels."*** *Concha-Barrientos M, et al. (2004) "Comparative Quantification of Health Risks: Global and Regional Burden of Disease Attributable to Selected Major Risk Factors". in: Ezzati M, Lopez AD, Rodgers A, Murray CJL, eds. Geneva: World Health Organization, chapter 21, pp.1651–1801. .*
http://www.who.int/publications/cra/chapters/volume2/1651-1802.pdf

In a position paper of the American Council on Science and Health **"ASBESTOS EXPOSURE: HOW RISKY IS IT**?" it was summarised that:

"Progress on a number of fronts has led to general scientific consensus on the following:
(1) amphibole fibers (which tend to be relatively long and thin) are a more potent risk factor for the development of mesothelioma and, to a lesser degree, lung cancer than are chrysotile fibers (which tend to be relatively short and wide); (2) longer, thin fibers are more pathogenic and there appear to be fiber size thresholds below which asbestos fibers do not pose any threat; and (3) those animal studies in which high exposure concentrations resulted in lung overloading are not considered relevant to humans. Analysis of the epidemiological literature supports some common patterns including: (1) for occupational and industrial exposures, the weight of evidence does not consistently support causal relationships between asbestos exposure and onset of pulmonary disease, some studies showing associative relationships but others showing no relationship between exposure and disease onset; and (2) chrysotile alone, uncontaminated by other fiber types, particularly amphiboles, does not appear to be a risk factor for mesothelioma, as once thought."

Ingestion of asbestos

Extract from Asbestos in Drinking-water Background document for development of WHO Guidelines for Drinking-water Quality. WHO/SDE/WSH/03.04/02

Originally puibished in Guidelines for drinking-water quality, 2nd ed. Vol. 2. Health criteria and other supporting information. World Health Organization, Geneva, 1996.

Conclusions
"Although asbestos is a known human carcinogen by the inhalation route, available epidemiological studies do not support the hypothesis that an increased cancer risk is associated with the ingestion of asbestos in drinking-water. Moreover, in extensive feeding studies in animals, asbestos has not consistently increased the incidence of tumours of the gastrointestinal tract. There is therefore no consistent, convincing evidence that ingested asbestos is hazardous to health, and it is concluded that there is no need to establish a guideline for asbestos in drinking-water."

Extract from Volume 2: Health Criteria and Other Supporting Information. Second Edition from Office of distribution and Sales, WHO. 1211 Geneva, Switzerland, 1996.

"While inhaled asbestos is a known carcinogen, there is no evidence that asbestos has any adverse effect on human health when ingested with drinking-water at current levels. Volume 2 of the Guidelines for Drinking-Water Quality points out that chemical substances can produce very different effects on health depending on the form of exposure. In the case of asbestos, experimental and epidemiological data indicate that there is "no consistent evidence that ingested asbestos is hazardous to health", and it has thus been concluded that there is "no need to establish a health-based guideline value for asbestos in drinking-water".

Extract form Environment Health Criteria 203, International Programme for Chemical Safety. *1.4 Uptake, clearance, retention and translocation, page 15, page 54*

"Available data from studies in humans and animals are insufficient to evaluate the possible uptake, distribution and excretion of chrysotile fibres from ingestion. Available evidence indicates that, if penetration of chrysotile fibres across the gut wall does occur, it is extremely limited."

5.2 Ingestion

"An important question in the evaluation of the possible risks associated with the ingestion of chrysotile asbestos is whether fibres can migrate from the lumen into and through the walls of the gastro-intestinal tract to be distributed within the body and subsequently cleared.

Review of the available data has been published in Environmental Health Criteria 53 (IPCS, 1986). The main conclusions were:

(a) It is not possible to conclude with certainty that chrysotile fibres do not cross the gastrointestinal wall. However, available evidence indicates that, if penetration does occur, it is extremely limited (Cook, 1983).

(b) There is no available information on bioaccumulation/retention of ingested chrysotile fibres. Simulated gastric juice has been shown to alter the physical and chemical properties of chrysotile fibres (Seshan, 1983).

(c) There was no difference in the level of urinary chrysotile between subjects drinking water with high compared to those drinking water with much lower natural chrysotile contamination (Boatman et al., 1983)."

6.2 Effects on laboratory mammals

"The ability of asbestos to cause gastrointestinal cancer following ingestion has been examined in many experimental studies reviewed extensively by Condie (1983) and Toft et al. (1984). Early studies on ingested asbestos were reviewed by IPCS (1986). There was no conclusive evidence of either histopathological or biochemical effects on the gastrointestinal wall, or of carcinogenicity in the animal species studied."

Controversy

As evaluated in the IARC Monographs Volumes 1-83, a list contains all agents, mixtures and exposures circumstances evaluated to date as being in « Group 1 » (carcinogenic to humans) (http://193.51.164.11/monoeval/crthgr01.html).

The International Agency for Research on Cancer (IARC - WHO) has recognized asbestos as a type 1 carcinogen. Because all types of asbestos were used incorrectly in the past, we know that chrysotile and amphiboles have been classified as category 1 carcinogens (proven carcinogenic agents), such as cadmium, chromium, nickel compounds, silica, the sun's rays, vinyl chloride, alcoholic beverages, salted fish, tobacco smoke, saw dust, the manufacture and repair of shoes, the manufacture of furniture and cabinets, iron and steel founderies and the rubber industry. The IARC (WHO) classification identifies a substance's hazard, not the risk.

IARC classification covers only the identification and characterization (hazard) of these agents, mixtures and activities. It does not include the assessment of risk, i.e.: the probability of toxic manifestations under actual conditions of use today. This is an important distinction: « hazard » is not « risk ». The IARC classification is about hazard, not risk. Indeed, characterizing a hazardous substance is not equal to assessing its true risk.

Hazard identification is an essential but insufficient component of risk assessment, which comprises also exposure data over time, and estimation of the likely risk under actual conditions of use. Because of the conceptual confusion and indiscriminate use of the terms « hazard » and « risk », untoward fear of unwelcome end points such as cancer, in many sectors of the general public, is driven by hazard data misrepresented as risk data.

The case of asbestos minerals

It should be realized that the word «asbestos» is a generic, commercial term which encompasses two very different families of fibrous silicates: the serpentine and the amphiboles. With the growing body of recent evidence regarding the distinct «hazard characterization» of chrysotile asbestos vs that of the amphibole varieties of asbestos, the time has come to better differentiate the characteristic hazards associated with the two families of asbestos. While the current IARC classification does not make this distinction for the different varieties of asbestos, the various exercises of « risk assessment » carried over several years of investigation between the two families of asbestos have confirmed that the risk associated with the use of chrysotile asbestos is quite different from that of the amphiboles. In fact, the amphibole asbestos minerals crocidolite and amosite produce perhaps orders of magnitude more disease than does chrysotile asbestos when the fibers are used in the same way. Recent analyses of tremolite exposure in Libby, Montana, suggest that this amphibole asbestos mineral produces 1000 fold more disease than does chrysotile asbestos when the fibers are used in the same way.

Finally, it is now is generally accepted that the much longer residence time (biopersistence) in the lung of inhaled amphibole fibers is one of the key factors for their much higher pathogenicity compared to chrysotile (Wagner & Pooley, 1986; Albin et al, 1994). Recent quantitative reviews that analyzed data from available epidemiological surveys to determine potency of asbestos in relation to fiber types confirmed the difference in risk between chrysotile and the amphiboles (Hodgson & Darnton, 2000; Berman & Crump, 2004). Recently published experimental biopersistence studies (Bernstein et al, 2003; Bernstein et al, 2005) provide strong support for the differences seen epidemiologically between chrysotile and amphibole asbestos.

Additionally, many epidemiological studies (Liddell, McDonald & McDonald, 1997) have shown no evidence of increased cancer risk from chrysotile exposure at presently regulated occupational exposure levels (~1 f/ml, 8-hour time weighted average), as recommended by the Group of Experts convened by the WHO in Oxford (1989).

2. Changes in asbestos regulation

It is important to mention that many countries like Russia, Canada, India, China, Brazil, Thailand and others apply differentiated approach to the regulation of asbestos – controllable use approach to the chrysotile and ban of amphibole asbestos.

But till recently, leading regulatory agencies did not distinguish the differences between amphibole and chrysotile types of asbestos in their policy. But with the evidence of up to date scientific and epidemiological studies these agencies have begun to revise approaches to asbestos policy.

Two leading countries, the United States and the United Kingdom are revising their positions and regulations of asbestos.

In 2001 EPA USA has started to revise their outdated U.S. EPA model for lung cancer and mesothelioma based primarily on a document completed in 1986 (U.S. EPA 1986). The main feature of the last U.S. EPA model for lung cancer and mesothelioma is that this model did not take into account differences in type of asbestos fiber. In other words, according to this model, all fiber types have equal potency of causing cancer.

In 2003 scientists D. Wayne Berman, Aeolus, Inc. and and Kenny S. Crump, Environ Corporation prepared a document by request of U.S. EPA. The final draft of this document is called:

"FINAL DRAFT: TECHNICAL SUPPORT DOCUMENT FOR A PROTOCOL TO ASSESS ASBESTOS-RELATED RISK."[*]

Extracts from that document:

"*The approach currently employed at the U.S. EPA to evaluate asbestos-related risks (IRIS 1988) is based primarily on a document completed in 1986 (U.S. EPA 1986**) and has not been changed substantially in the past 15 years, despite substantial improvements in asbestos measurement techniques and in the understanding of the manner in which asbestos exposure contributes to disease.** Therefore, this document provides an overview and evaluation of **the more recent studies** and presents proposed modifications to the protocol for assessing asbestos-related risks that can be justified based on **the more recent work.**"".*

AND even more important (page 7.50):
*"Results in Table 7-17 also **differentiate between the potency of chrysotile and amphibole** for both lung cancer and mesothelioma. Amphibole is estimated as being about four times as potent as chrysotile for lung cancer (although the difference is not significant) and about 800 times as*

[*] http://www.epa.gov/oswer/riskassessment/asbestos/

potent as chrysotile for mesothelioma (a highly significant difference). ***Moreover, the data are consistent with the hypothesis that chrysotile has zero potency toward the induction of mesothelioma.****"*

Based on FINAL DRAFT: TECHNICAL SUPPORT DOCUMENT FOR A PROTOCOL TO ASSESS ASBESTOS-RELATED RISK U.S. EPA is going to change outdated U.S. EPA model for lung cancer and mesothelioma recognizing huge difference between chrysotile and amphiboles, i.e. U.S. EPA will have two different formulas describing exposure to chrysotile and to amphibole asbestos instead of one.

In June 2006, , Kevin Walkin and Geoff Lloyd under the lead of Giles Denham, a board member, prepared the following document or the HSC, **Health and Safety Commission of the United Kingdom:**

"A COMPARAISON OF THE RISKS FROM DIFFERENT MATERIALS CONTAINING ASBESTOS."[*]

HSC agreed that there should be **a risk-based approach** to the licensing of asbestos, with licensing reserved for high risk products and processes.

Extracts from document:

''There are three main types of asbestos used in commercial products, these are:

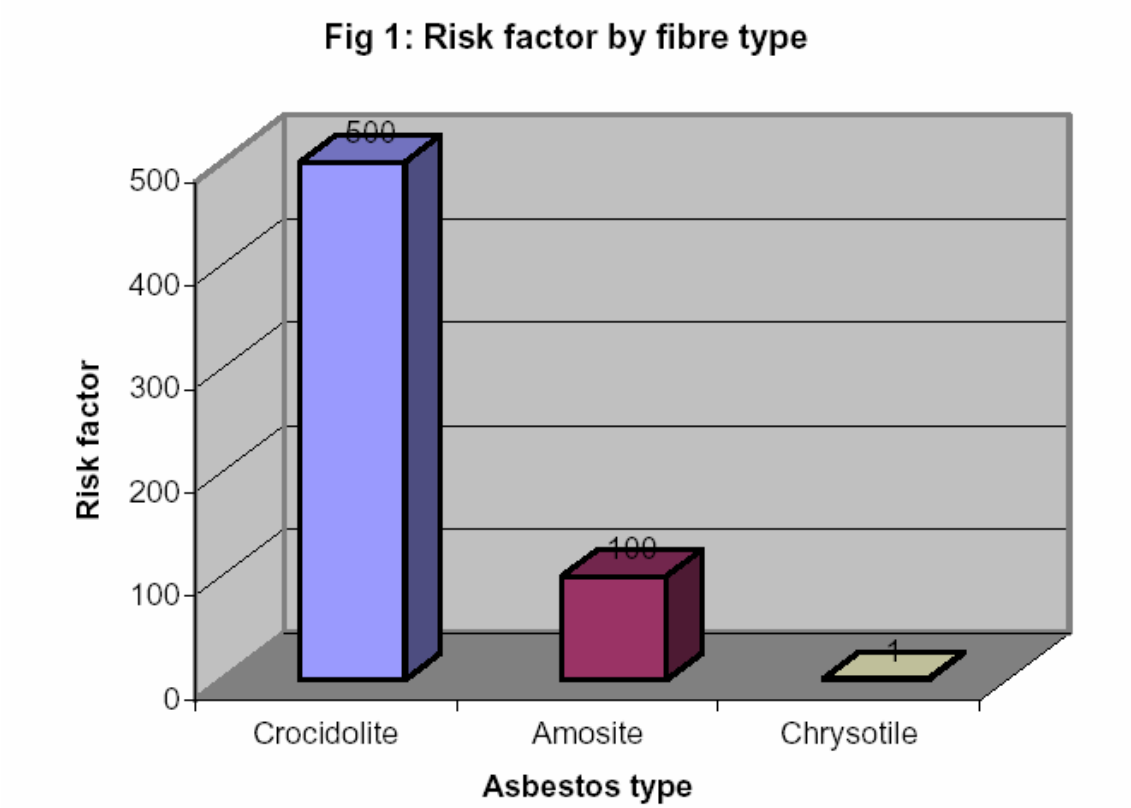
- *Chrysotile (white) asbestos,*
- *Amosite (Brown) asbestos and*
- *Crocidolite (Blue) asbestos*

The epidemiological evidence from asbestos workers and well-conducted animal tests shows that while all types of asbestos share the same hazards (e.g. the potential of an early death from lung cancer, asbestosis and mesothelioma) they have varying degrees of risk (the likelihood that death from one of the hazards will occur). The relative risk from the same level of exposure but to different asbestos fibre types is shown in figure 1(as derived by Hodgson and Darnton, 2000). The relative risk from crocidolite asbestos is some 500 times greater than chrysotile asbestos and the relative risk from amosite asbestos is 100 times greater than chrysotile asbestos. This means that the type/s of asbestos in the product are particularly significant when assessing risk.

If the estimated usage of asbestos in GB from the published RIA (CD 205, see figure 2) is combined with the risk factors for each asbestos type in figure 1, it is possible to obtain an assessment of the relative risk for each asbestos type installed (figure 3). The values in figure 3 have been normalised to the asbestos type with the lowest overall calculated risk (i.e. chrysotile = 1). Therefore it can be seen that amosite represents a risk some 18.5 times greater than chrysotile and crocidolite represents some 9.3 times the risk of chrysotile. Although this is an initial estimate and does not take any account of whether the materials are present in a product type that will be worked on, or the magnitude of the concentration of airborne fibres that would

[*] http://www.hse.gov.uk/aboutus/HSC/meetings/2006/040706/a07.htm

be released, it clearly shows that amosite and crocidolite asbestos need more consideration than chrysotile, if a risk based approach to licensing is to be followed.'



3. International organizations

Recognizing the difference between fibre types the international organizations apply differentiated approach to the regulation of various types of asbestos reserving in its policy strict regulation and ban for amphibole types of asbestos and controllable use approach to the chrysotile asbestos.

Rotterdam Convention

At its tenth session, held in Geneva on 17 to 21 November 2003 the Intergovernmental Negotiating Committee adopted the decision guidance document for crocidolite, actinolite, amosite, anthophyllite and tremolite asbestos with the effect that these chemicals became subject to the interim PIC procedure. http://www.pic.int/home.php?type=b&id=38

All representatives supported the inclusion of the four amphibole forms of asbestos in the interim PIC procedure. A number of representatives, noting that chrysotile was different from the amphibole forms of asbestos, expressed concern about the sufficiency of the scientific evidence of its carcinogenicity.

At its first meeting, held in Geneva 20 to 24 September 2004, the Conference of the Parties agreed to include actinolite, amosite, anthophyllit and tremolite asbestos in Annex III of the Rotterdam Convention, with the effect that these chemicals became subject to the PIC procedure.

Annex III(**)

Chemical *(CAS number(s))*	**Decision Guidance Document** *English French Spanish*		
Asbestos **Crocidolite** *(12001-28-4)* **Actinolite** *(77536-66-4)* **Anthophyllite** *(77536-67-5)* **Amosite** *(12172-73-5)* **Tremolite** *(77536-68-6)*	E	F	S

WHO

In the WHO's World Health Assembly in May 2007, a differential approach in the elimination of asbestos related diseases was presented in the `Workers' health: global plan of action (WHA60.26 Item 10)[*]. This approach states that "Its activities will include global campaigns for elimination of asbestos-related diseases – bearing in mind a differentiated approach to regulating its various forms – in line with relevant international legal instruments and the latest evidence for effective interventions…"

10. WHO will work with Member States to strengthen the capacities of the ministries of health to provide leadership for activities related to workers' health, to formulate and implement policies and action plans, and to stimulate intersectoral collaboration. Its activities will include global campaigns for elimination of asbestos-related diseases – bearing in mind a differentiated approach to regulating its various forms – in line with relevant international legal instruments and the latest evidence for effective interventions, as well as immunization of health-care workers against hepatitis B, and other actions addressing priority work-related health outcomes.

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