A REVIEW OF RECENTLY PUBLISHED EVIDENCE ON HEALTH RISKS ASSOCIATED WITH ASBESTOS FIBRE TYPES

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A REVIEW OF RECENTLY PUBLISHED EVIDENCE ON HEALTH RISKS ASSOCIATED WITH ASBESTOS FIBRE TYPES

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In the area of occupational health, and specifically regarding the use of asbestos, regulatory agencies in all countries have the responsibility to set workplace exposure limits which will reduce the risk to workers to the lowest possible level. That this exercise should be based on the most recent scientific assessment available would seem obvious.

However, some countries, while in the process of formulating so-called "revised" recommended asbestos standards, are still using scientific reviews which are far out of date. This is particularly unfortunate, as much new evidence has accumulated over the last few years, with the resulting frequent publications, not only of scientific papers, but also of editorials and commentaries inspired by the need to revisit the issue of risks related to asbestos. One such recent commentary dates back to July 1997 (Alleman JE and Mossman BT: "Asbestos Revisited"; Scientific American, July 1997; pp. 70-75)

An example of such obsoleteness comes from data published in the early 70's and earlier, generated from experimental designs where *gravimetric* units for the dosage were used, instead of the presently used *fibre number* units. This is now acknowledged as the main reason why the results of the then reported animal experimentation could not account for the differences in pathological potency between asbestos fibre types, as observed in the epidemiological surveys. In 1988, scientists of the US EPA published the results of a study on the comparison of *mass* vs *number of fibers* as the basis for dosage. It was shown that when "number of fibres" was used,

the experimental results became totally consistent with the results of epidemiological surveys (1). Dr. John C. Wagner, himself one of the authors of these earlier reports, had this to say in 1989: *"...we believe therefore that chrysotile is the least harmful form of asbestos in every respect, and that greater emphasis should be placed on the different biological effects of the various amphibole fibres"* (Wagner, J.C. et al. 1989 *in* IARC Sci. Pub. No. 90, p. 448 Lyon)

Another example of confusion in risk perception which invariably leads to bad risk management decision regarding asbestos is the the so-called "hit-and-run" view which alleges that even if chrysotile dissolves and dissappears from the lung faster than the amphiboles, it may still have triggered the mechanism leading to mesothelioma. A German study (Bellman and Muhle, 1995) published by the Schriftenreihe of the Bundesantanstalt fur Abeitsschutz (Federal Office for Worker Protection) indicates that *"biopersistence of inhaled fibrous materials is a critical factor in determining carcinogenic potency"*. This has been confirmed in 1997 by Bernstein in a research report to The Joint Research Center, Environmental institute, European Chemicals Bureau in ISPRA (Italy). The report is entitled: "Correlation Between Short Term Biopersistence and Chronic Toxicity Studies", and was produced in June 1997." This recently published evidence should put to rest the "hit-and-run phenomenon", used by some authors to implicate chrysotile in causing mesothelioma. In other words, the "importance of biopersistence" and the "hit-and-run" view are completely contradictory terms.

It would not be useful to pursue a detailed evaluation of such oudated review documents that unfortunately are still used today by some national regulatory agencies. It is felt that it is better to take stock of the more recently published evidence. This review will therefore concentrate on the more recent scientific publications which have formed the basis of a

wide international scientific consensus. The review will first address the issue of:

1/ the importance of physico-chemical parameters: size and durability;

2/ the pathogenic differences between asbestos fiber types;

3/ the published evidence pointing to a practical threshold level of exposure to chrysotile asbestos below which no adverse health effects are detectable;

This will be followed with a review of evidence on fibre emission resulting from the use of modern, high-density chrysotile composites: friction materials and asbestos-cement.

1/

Physico-chemical parameters pertinent to health effects: the importance of fibre dimensions and durability.

Numerous studies made over several decades, which are still pertinent today, relate to the importance of fibre dimensions (length and diameter) as prerequisites for biological potency, since these two paramenters are related to respirability. It is generally recognized that fibrous structures with a diameter $\leq 3\mu$ and a length $\geq 5\mu$ are those which will penetrate deeply into the respiratory tree. There is almost complete consensus regarding this point, and thus there is no need to review the evidence on this aspect.

However, new evidence published over the last 10 years has come from investigations using modern techniques, in particular from mineral analyses performed on lung tissue, also known as "lung burden" studies. As a result, an additional parameter of fibrous materials is now universally recognized as of paramount

importance for assessing the pathological potential of inhaled particles: durability.

Durability is this characteristic which varies widely amongst different respirable particles, and which is likely related to the different chemical structures and crystalline habits of mineral particles. In turn, durability will determine the extent of a key biological phenomenon: **biopersistence**, which can be described as the length of time for inhaled particles to persist in the lungs and adversely affect surrounding tissue before they are eventually dissolved or otherwise cleared.

Biopersistence studies have been carried out on a number of different respirable particles, and it has become clear that there are vast differences amongst various respirable fibrous materials presently used by industry. In fact, there seems to be a continuum of values for biopersistence of respirable materials, from very short persistence (low durability) to practically indefinite persistence (very high durability).

In 1992, a symposium on the "Biopersistence of Respirable Synthetic Fibres and Minerals" was held in Lyon, under the aegis of the International Agency for Research on Cancer (IARC). For asbestos fibres, it was confirmed repeatedly that **chrysotile** asbestos displays low biopersistence, as opposed to the **amphibole** asbestos fibre types such as crocidolite and amosite, which display exceedingly long biopersistence. In addition, data presented at the symposium indicated that various types of **glass fibres** also have different solubilities and biopersistence characteristics which may vary according to their respective manufacturing processes and chemical compositions. Thus, glass fibres with high aluminum (Al) content were shown to be more durable than those with low Al content. A similar observation was reported for **refractory ceramic fibres** (RCF), i. e.: high Al oxyde content has a negative influence on biosolubility, whereas lower concentrations of alkaline oxydes have the opposite effect. A major study by German scientists of the Fraunhofer Institute in Hannover compared a series of man-made mineral

fibres (MMMF), from glass fibres to RCFs and natural fibres for *in vivo* durability. Half-times for fibre elimination from the lung ranged from 10 to 500 days. A study from the U.S.A. also reported that inhaled RCFs showed no chemical alterations 2 years following end of exposure, whereas glass fibres showed that some components had leached. Another study from the Institute of Occupational Medicine in Edinburgh showed that in experiments using rats, chrysotile and glass fibres were cleared from the lung at approximately the same rate, whereas there was hardly any clearance of crocidolite asbestos.
The general conclusion from this international symposium is that RCFs are certainly not cleared rapidly from the lung, that some MMMFs are cleared more slowly than others, and that the same is true for asbestos, where it appears that amphibole types have clearance half-times in the range of several decades, whereas chrysotile asbestos is cleared within weeks or a few months.

The pathological relevance of this phenomenon is important. In 1986, British scientists J. C. Wagner and F. D. Pooley put it in these terms:

> *"...the importance of selective retention of fibres has been discussed in a recent paper. We are convinced that those diseases associated with exposure to mineral fibres are due to fibres retained in the lungs".* [1]

Indeed, in a more recent study of the retention patterns of fibres in asbestos-cement workers in Sweden, the authors came to the conclusion that:

> *"...adverse effects are associated rather with the fibres that are retained (amphiboles), than with the ones being cleared (largely chrysotile)".* [2]

Thus it has become abundantly clear that biopersistence must now be taken into account when assessing risk associated with

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[1] Wagner JC and Pooley FD (1986) Thorax 41: 161-166
[2] Albin M, Pooley FD, Strömberg U, Attewel R, Mitha R, Johansson L, Welinder H (1994) Occup Environ Med 51: 205-211

the use of respirable materials. In 1995, the Fraunhaufer Institute scientists reiterated their view [3] in these words:

"biopersistence of inhaled fibrous materials is a critical factor in determining carcinogenic potency".

This has been confirmed in 1997 by Bernstein in a research report to the Joint Research Center, Environmental Institute, European Chemicals Bureau [4] . Incidentally, that should put to rest the "hit-and-run phenomenon", used by authors to implicate chrysotile in causing mesothelioma. In other words, "biopersistence" and "hit-and-run" are completely contradictory terms.

CONCLUSION:

Risk assessment and management of respirable fibrous materials must take into account not only the dimensions but also the durability and biopersistence characteristics of all airborne materials used in industry. This should apply not only to the different asbestos fibre types, but to all fibrous materials, whether natural or man-made.

2/

The pathogenic differences between asbestos fibre types.

Review of the evidence published after 1976 points to the definite differences in biological effects and potencies of chrysotile asbestos and amphibole varieties. There are no less than 25 reports from human studies only, and they are presented here under two separate sub-headings:

a/ Morbidity and mortality data in "chrysotile only" users;

b/ Analysis of mineral lung content (human data only).

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[3] Bellman and Muhle (1995) A report presented to The Schriftenreihe (Secretary) of the Bundesanstalt fur Abeitsschutz (Federal Office for Worker Protection

[4] Bernstein D (1997) Correlation between short term biopersistence and chronic toxicity studies. A report to the Joint Research Center, European Chemicals Bureau, ISPRA, Italy

A/ MORTALITY AND MORBIDITY DATA

•**Wagner, J.C., Newhouse, M.L., Corrin, B., Rossiter, C.E.and Griffiths, D.M. (1988).** Correlation between fibre content of the lung and disease in East London asbestos factory workers.
British Journal of Industrial Medicine 45(5):305-308.
"We believe therefore that chrysotile is the least harmful form of asbestos in every respect and that more emphasis should be laid on the different biological effects of amphibole and serpentine asbestos fibre".

•**Kleinerman, J. (1988).** The pathology of asbestos related lung disease.
Proceedings, The Fleischner Society, Eighteenth Annual Symposium on Chest Disease, Montréal, Canada, 16-18 May, pp. 33-46.
"Most asbestos workers who develop mesothelioma are exposed to amphibole asbestos. Few mesotheliomas are found in workers exposed to chrysotile... The tremolite exposure is considered to play a major role in the development of the mesotheliomas in these cases".

•**Dunnigan, J. (1988).** Commentary: Linking chrysotile asbestos with mesothelioma.
American Journal of Industrial Medicine 14:205-209.
Overview of evidence showing unlikeliness of link of mesothelioma with chrysotile exposure. Epidemiological studies from USA (Weiss, McDonald and Fry, Dement), from Britain (Newhouse, Thomas, Acheson) are analysed, and lung burden studies (Pooley, Wagner, Jones, A.D. McDonald) are also pointed to.

•**Hughes, J.M., Weill, H. and Hammad, Y.Y. (1987).** Mortality of workers employed in two asbestos cement manufacturing plants.
British Journal of Industrial Medicine 44(3):161-174.
Mortality of 6,931 employees of two asbestos cement factories was studied. In one of them (plant 2), crocidolite was used along with chrysotile. There were 10 cases of mesothelioma in this study, 8 of whom from the plant 2. The case-control analysis found a significant relation

between risk of mesothelioma and proportion of time spent in the area of making a/c pipes where crocidolite was used.

•**Gardner, M.J. and Powell, C.A. (1986).** Mortality of asbestos cement workers using almost exclusively chrysotile fibre.
Journal of the Society of Occupational Medicine 36(4):124-126.
Three studies are reviewed of asbestos-cement workers using almost exclusively chrysotile in Great Britain and in Sweden. No asbestos-related mortality in meaningful excess of expected was found. The authors state: "This is in contrast with most studies of workers making similar products from mixed fibres containing mainly chrysotile but also amphiboles, crocidolite and amosite".

•**Berry, G. and Newhouse, M.L. (1983).** Mortality of workers manufacturing friction materials using asbestos.
British Journal of Industrial Medicine 40(1):1-7.
Study of 13,400 workers (friction materials) showing no mesothelioma when chrysotile only was used, but 10 mesotheliomas when crocidolite was also used.

•**Thomas, H.F., Benjamin, I.T., Elwood, P.C. and Sweetnam, P.M. (1982).** Further follow-up study of workers from an asbestos cement factory.
British Journal of Industrial Medicine 39(3):273-276.
Study of 1,970 a/c workers, showing no case of mesothelioma over 40-year period when chrysotile only was used, but 2 mesotheliomas when crocidolite was used during a 2-year period.

•**McDonald, A.D. and Fry, J. (1982).** Mesothelioma and fibre type in three american asbestos factories - Preliminary report.
Scandinavian Journal of Work, Environment and Health 8 (Supplement 1):53-58.
Study of yarns, cloth and packings, and also gaskets manufacturing, showing only 1 case of mesothelioma / 2,341 workers when almost exclusively chrysotile was used, and 18 cases / 1,429 workers when mixed fibre types were used.

• **Acheson, E.D., Gardner, M.J., Pippard, E.C. and Grime, L.P. (1982).** Mortality of two groups of women who manufactured gas masks from chrysotile and crocidolite asbestos: a 40-year follow-up.
British Journal of Industrial Medicine 39(4):344-348.
Study of gas mask workers showing no case of mesothelioma when chrysotile only was used, and 5 cases / 757 workers using crocidolite.

•**McDonald, A.D. and McDonald, J.C. (1978).** Mesothelioma after crocidolite exposure during gas mask manufacture.
Environmental Research 17(3):340-346.
Exposure to crocidolite in making war-time military gas-masks in Québec led to accumulation of 9 cases of mesothelioma out of 56 deaths (16%). High amounts of crocidolite (and some chrysotile) were found in their lungs. This compares with incidence of mesothelioma, 0.26% of deaths in the Québec (chrysotile) mines.

•**Weiss, W. (1977).** Mortality of a cohort exposed to chrysotile asbestos.
Journal of Occupational Medicine 19(11):737-740.
Study showing no case of mesothelioma in millboard and paper manufacturing when chrysotile only is used.

B/ ANALYSIS OF MINERAL LUNG CONTENT

•**Wagner, J.C., Newhouse, M.L., Corrin, B., Rossiter, C.E.R. and Griffiths, D.M. (1988).** Correlation between fibre content of the lung and disease in East London asbestos factory workers.
British Journal of Industrial Medicine 45(5):305-308.
The lungs from 36 past workers of an asbestos factory using chrysotile, crocidolite, and amosite were examined. Crocidolite and amosite lung contents were strongly associated with asbestosis, and with mesothelioma, whereas no such correlation was evident with chrysotile and mullite.

•**Wagner, J.C., Moncrieff, C.B., Coles, R., Griffiths, D.M. and Munday, D.E. (1986).** Correlation between fibre content of the lungs and disease in naval dockyard workers.

British Journal of Industrial Medicine 43(6):391-395.
Study showing increasing amounts of amphiboles in lung tissue with increasing severity of asbestosis, but no increase of chrysotile.

•**Churg, A. (1985). Malignant mesothelioma in British Columbia in 1982.**
Cancer 55(3):672-674.
Study showing a 300-fold increase of amphiboles in lung tissue of mesothelioma cases, but no difference with general population with regard to chrysotile lung content.

•**Churg, A. (1988).** Chrysotile, tremolite, and malignant mesothelioma in man.
Chest 93(3):621-628.
Churg maintains that of 53 cases of mesothelioma ever reported as caused by chrysotile, in fact 51 may be attributed to contamination by tremolite, crocidolite and/or amosite.

•**Jones, J.S.P., Roberts, G.H., Pooley, F.D., Clark, N.J., Smith, P.G., Owen, W.G., Wagner, J.C., Berry, G. and Pollock, D.J. (1980).** The pathology and mineral content of lungs in cases of mesothelioma in the United Kingdom in 1976.
In Biological Effects of Mineral Fibres, J.C. Wagner Editor, Vol. 1, International Agency for Research on Cancer, IARC Scientific Publications No. 30, Lyon:187-199.
Study in U.K. showing that patients with mesothelioma have a far greater number of amphiboles in their lungs, but same amount of chrysotile when compared to controls.

•**McDonald, A.D. (1980).** Mineral fibre content of lung in mesothelial tumours: - Preliminary report.
Biological Effects of Mineral Fibres, J.C. Wagner Editor, Vol. 2, International Agency for Research on Cancer, IARC Scientific Publications No. 30, Lyon:681-685.
Same observation as above for patients with mesothelioma in North America.

•**Churg, A. (1982).** Asbestos fibres and pleural plaques in a general autopsy population.
American Journal of Pathology 109(1):88-96.
Study showing that patients with pleural plaques have a 50-fold increase of amphiboles compared to chrysotile.

•**Wagner, J.C., Berry, G. and Pooley, F.D. (1982).** Mesothelioma and asbestos type in asbestos textile workers: a study of lung contents.
British Medical Journal 285:603-606.
In an asbestos textile factory that utilized mainly chrysotile with some crocidolite, less chrysotile and more crocidolite fibre were found in the lungs of 12 persons who had died of mesothelioma than in the lungs of controls without mesothelioma.

•**Wagner, J.C., Pooley, F.D., Berry, G., Seal, R.M.E., Munday, D.E., Morgan, J. and Clark, N.J. (1982).** A pathological and mineralogical study of asbestos-related deaths in the United Kingdom in 1977.
The Annals of Occupational Hygiene, Inhaled Particles V, 26(1-4):423-431.
Study showing a 100 fold increase of amphiboles in lung tissue, but similar amounts of chrysotile in referred pneumoconiosis patients.

•**Gylseth, B., Mowe, G. and Wannag, A. (1983).** Fibre type and concentration in the lungs of workers in an asbestos cement factory.
British Journal of Industrial Medicine 40(4):375-379.
The predominant asbestos type used in a Norwegian asbestos-cement factory (1942-1980) has been chrysotile (91.7%), with small admixture of amosite (3.1%), crocidolite (4.1%) and anthophyllite (1.1%). In the lungs of workers who had died of mesothelioma (4) or of lung cancer (3), the percentage of chrysotile fibres was 0%-9% whereas the corresponding proportion for the amphiboles was 76% and 99%.

•**Rowlands, N., Gibbs, G.W. and McDonald, A.D. (1982).** Asbestos fibres in the lungs of chrysotile miners and millers - A preliminary report.

The Annals of Occupational Hygiene, Inhaled Particles V, 26(1-4):411-415. Lung samples from 47 workers of chrysotile mines in Québec who had died of various causes not related to asbestos were studied. Similar quantities of chrysotile and tremolite were found although tremolite admixture to chrysotile ore is extremely small. It indicates that tremolite persisted in the lungs while chrysotile was dissolved.

•McDonald, A.D., McDonald, J.C. and Pooley, F.D. (1982). Mineral fibre content of lung in mesothelial tumours in North America. The Annals of Occupational Hygiene, Inhaled Particles V, 26(1-4):417-422. 99 case-control pairs of lung tissue specimens were examined from persons who had died of mesothelioma in North America. High content of amosite was found in 26 cases and 8 controls, and high content of crocidolite in 15 cases and 5 controls, while content of chrysotile was equal in cases and controls.

•Gibbs, A.R., Jones, J.S.P., Pooley, F.D., Griffiths, D.M. and Wagner, J.C. (1989). Non-occupational malignant mesotheliomas. *In* Non-Occupational Exposure to Mineral Fibres, Eds. J. Bignon, J. Peto and R. Saracci. WHO/IARC Scientific Publications No. 90, Lyon:219-228. The mineral content of the lungs from 84 cases of malignant pleural mesothelioma was estimated by electron microscopy and energy-dispersive X-ray analysis. These cases were chosen because the history of asbestos exposure was absent, indirect or ill-defined. The chrysotile counts in the lungs from these mesothelioma cases were similar to those in controls an in a previous series of mesotheliomas in which the majority had had direct exposure to asbestos. These findings confirm those of previous studies indicating that amphiboles are more important than chrysotile in the causation of malignant mesothelioma. The results confirm that some mesotheliomas develop in the absence of asbestos exposure. "It is possible that chrysotile might potentiate the effects of amphiboles, but we believe that it has either no potential (or a very low one) for mesothelioma induction on its own".

•Albin A, Pooley FD, Strömberg U, Attewell R, Mitha R and Welinder H (1994)

Retention patterns of asbestos fibres in lung tissue among asbestos cement workers.

A study which showing different kinetics for amphibole and chrysotile fibres in human lung tissue. Amphibole fibre concentrations increase with duration of exposure, whereas chrysotile concentrations do not. The authors indicate that their study supports a former finding of a possible adaptive clearance of chrysotile, and conclude that their findings "support the hypothesis that adverse effects are associated rather with the fibres that are retained (amphiboles), than with the ones being cleared (largely chrysotile)."

3/ Published evidence pointing to a practical threshold level of exposure to chrysotile asbestos below which no adverse health effects are detectable.

A 1996 draft report from a WHO Task Group for Chrysotile Asbestos concludes that "*exposure to chrysotile asbestos poses increased risks for asbestosis, lung cancer and mesothelioma in a dose dependent manner. No threshold has been identified for carcinogenic risks*".

This statement makes sense to those who consider "epidemiology" as the only instrument for assessing risks and for coming to a conclusion regarding the existence or absence of thresholds for toxic substances. This is to be expected from the epidemiological approach for very low levels of exposures to toxic substances. Put simply, the epidemiological approach is just not the most appropriate tool to establish the existence or the absence of thresholds when very low levels of exposure are considered. It is for this reason that it is often said that no threshold has been "identified" for carcinogenic risks. More precisely, it means that no threshold has been identified using the data and the analytical methodology available to epidemiologists. It does not mean that there is no threshold; it simply means that if there is one, it cannot be identified.

For this reason, some epidemiologists feel that more epidemiological data are needed concerning cancer risks for populations exposed to levels below 1 fibre/ml. But the reality is that this is practically an impossible goal, as data from several hundreds of thousands of people would be needed, and several complex confounding factors (ethno-socio-economic) would have to be considered in order to satisfy the requirements of scientifically credible statistical analysis. If however one considers the toxicological evidence, most experimentalists are ready to recognize that indeed, there are thresholds for asbestos-inducible diseases. More prudently perhaps, toxicologists prefer to use terms such as "below detection limits".

That this would be certainly the case for chrysotile asbestos is supported by published evidence from a fairly large number of human studies in various settings and in different countries, showing that at low (~1 f/ml) occupational exposure levels to chrysotile, there is no statistically significant increase of incidence of asbestos-related disease in workers. References to these studies which follow illustrate this point.

HEALTH EXPERIENCE OF WORKERS AT VERY LOW EXPOSURE LEVELS TO CHRYSOTILE ONLY

•**Berry, G. and Newhouse, M.L. (1983).** Mortality of workers manufacturing friction materials using asbestos.
British Journal of Industrial Medicine 40(1):1-7.
A mortality (1942-1980) study carried out in a factory producing friction materials, using almost exclusively chrysotile. Compared with national death rates, there were no detectable excess of deaths due to lung cancer, gastrointestinal cancer, or other cancers. The exposure levels were low, with only 5% of men accumulating 100 fibre-years/ml. The authors state: "The experience at this factory over a 40-year period showed that chrysotile asbestos was processed with no detectable excess mortality".

•**Newhouse, M.L. and Sullivan, K.R. (1989).** A mortality study of workers manufacturing friction materials: 1941-86.
British Journal of Industrial Medicine 46(3):176-179.
The study referred to in 5898 has been extended by seven years. The authors confirm that there was no excess of deaths from lung cancer or other asbestos related tumours, or from chronic respiratory disease. After 1950, hygienic control was progressively improved at this factory, and from 1970, levels of asbestos have not exceeded 0.5-1.0 f/ml. The authors conclude: "It is concluded that with good environmental control, chrysotile asbestos may be used in manufacture without causing excess mortality".

•**Thomas, H.F., Benjamin, I.T., Elwood, P.C. and Sweetnam, P.M. (1982).** Further follow-up study of workers from an asbestos cement factory.
British Journal of Industrial Medicine 39(3):273-276.
In an asbestos-cement factory using chrysotile only, 1,970 workers were traced, and their mortality experience was examined. There was no appreciably raised standardised mortality ratio (SMR) for the causes of death investigated, including all causes, all neoplasms, cancer of the lung and pleura, and cancers of the gastrointestinal tract. The authors indicate: "Thus the general results of this mortality survey suggest that the population of the chrysotile asbestos-cement factory studied are not at any excess risk in terms of total mortality, all cancer mortality, cancers of the lung and bronchus, or gastrointestinal cancers".

•**Weill, H., Hughes, J. and Waggenspack, C. (1979).** Influence of dose and fibre type on respiratory malignancy risk in asbestos cement manufacturing.
American Review of Respiratory Disease 120(2):345-354.
An investigation on 5,645 asbestos-cement manufacturing workers, showing no raised mortality resulting from exposure for 20 years to chrysotile asbestos at exposure levels equal to or less than 100 MPPC.years (corresponding to approximately 15 fibres/ml.years). The authors state: "...However, the demonstration that low cumulative and short-term exposures did not produce a detectable excess risk for respiratory

malignancy may be of assistance in the development of regulatory policy, because a scientifically defensible position based on these data is that there are low degrees of exposure not associated with a demonstrable excess risk".

•**Ohlson, C.-G. and Hogstedt, C. (1985).** Lung cancer among asbestos cement workers. A Swedish cohort study and a review.
British Journal of Industrial Medicine 42(6):397-402.
A cohort study of 1,176 asbestos-cement workers in a Swedish plant using chrysotile asbestos showing no excess related mortality at exposures of about 10-20 fibres/ml.years.

•**Gardner, M.J., Winter, P.D., Pannett, B. and Powell, C.A. (1986).** Follow up study of workersmanufacturing chrysotile asbestos cement products.
British Journal of Industrial Medicine 43:726-732.
A cohort study carried out on 2,167 subjects employed between 1941 and 1983. No excess of lung cancers or other asbestos-related excess death is reported, at mean fibre concentrations below 1 f/ml, although higher levels had probably occurred in certain areas of the asbestos-cement factory.

MOST RECENTLY AVAILABLE EVIDENCE.

McDonald, JC, Liddell, DK, Dufresne, A. and McDonald, AD (1993)

The 1891-1920 birth cohort of Quebec chrysotile miners and millers: mortality 1976-88
Brit. J. Ind. Med. 50: 1073-1081
This study is undoubtedly the largest cohort of asbestos workers ever studied and followed for the longest period is that of the miners and millers of the chrysotile mines in Québec. The cohort, which was established in 1966, comprises some 11,000 workers born between 1891-1920 and has been followed ever since. Optimal use was made of all available dust measurements to evaluate for each cohort member his exposure in terms of duration, intensity and timing. Findings on mortality have been

published on five occasions, and this recent report provides an update of the results of analysis of mortality for the period 1976-1988 inclusive. One of the central findings of this last update is that over several narrow categories of exposure up to 300 mpcf x years, the SMRs for lung cancer fluctuated around unity, with no evidence of trend, and increased steeply above that exposure level.

Still more recently, the same authors further updated their study, this time with 9780 men traced into 1992. Results from exposures below 300 mpcf x years, roughly equivalent to 900 fibres/ml x years - or, say, 45 fibres/ml for 20 years - lead the authors to conclude: *"Thus it is concluded from the point of view of mortality that exposure in this industry to less than 300 mpcf.years has been essentially innocuous"*. The results were published in 1997.[5]

In terms of present day mandated or recommended exposure levels for chrysotile, and whatever hesitations one might have in converting mpcf to f/ml, even by applying a conservative conversion factor of 1 mpcf ~ 3 f/ml, the above mentioned references including these updates provide strong support for the recommendation from the "Group of Experts" convened by the WHO (Oxford, 1989) of a TLV of 1 f/ml for chrysotile asbestos.

RISK ASSOCIATED WITH LOW LEVELS OF ASBESTOS IN GENERAL AMBIENT AIR

Regarding **general population** exposure, repeated studies have consistently failed to find an increased respiratory disease incidence in lifelong residents of Quebec chrysotile mining towns who were never employed in the industry. These populations were exposed to levels less than that of the mining

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[5] **Liddell FDK, McDonald AD and McDonald JC . Ann Occup Hyg 41:13-35 (1997)** The 1891-1920 Birth Cohort of Quebec Chrysotile Miners and Millers: Development from 1904 and Mortality to 1992

workers, but higher than those of general populations elsewhere. References to these studies follow:

•**Churg, A. (1986).** Lung asbestos content in long-term residents of a chrysotile mining town.
American Review of Respiratory Disease, 134(1):125-127.
Study comparing health effects in residents of chrysotile mining towns, where levels are from 200 to 500 higher than in most North American cities, to those seen in urban residents. In spite of higher levels in these mining towns, no evidence of higher asbestos-related diseases were found. The author concludes:
"These observations should provide reassurance that exposure to chrysotile asbestos from urban air or in public buildings will not produce detectable disease". This is in agreement with other reports on residents of chrysotile mining towns in Québec, which have consistently failed to demonstrate excess respiratory disease incidence. These are:

•**McDonald, A.D. and McDonald, J.C. (1980).** Malignant mesothelioma in North America.
Cancer 46(7):1650-1656.

•**Siemiatycki, J. (1982).** Health effects on the general population (mortality in the general population in asbestos mining areas).
Proceedings, World Symposium on Asbestos, Montréal, 25-27 May, pp. 337-348.

•**Pampalon, R., Siemiatycki, J. et Blanchet, M. (1982).** Pollution environnementale par l'amiante et santé publique au Québec.
Union Médicale du Canada 111(5):475-489.

•**McDonald, J.C. (1985).** Health implications of environmental exposure to asbestos.
Environmental Health Perspectives 62:319-328.

•**Camus, M., Siemiatycki, J. and Meek, B. (1998).** Nonoccupational Exposure to Chrysotile Asbestos and the risk of Lung cancer.
New Engl. J. Med. 338(22): 1566-1571

EVIDENCE ON ASBESTOS FIBRE EMISSION RESULTING FROM THE USE OF HIGH-DENSITY CHRYSOTILE COMPOSITES: FRICTION MATERIALS AND ASBESTOS-CEMENT.

ASBESTOS IN FRICTION MATERIALS

The issue of the extent of the contribution of asbestos fibres to the general environment resulting from the use of asbestos in friction materials has also received much attention. Asbestos has been a major constituent of automotive friction materials for more than 70 years, where the presence of mostly chrysotile asbestos (from 25% to 65% by weight) imparts strength, flexibility and heat resistance to brake linings, in addition to friction and wear properties. Comprehensive investigations conducted with the support of the US EPA have shown that on the average, more than 99.7% of the asbestos emitted as a result of wear and abrasion has been converted into other products such as forsterite, a material which has been found non-carcinogenic in animals. Furthermore, it has been determined that such asbestos (less than 1%) as may be present in wear debris consists predominantly of very short (0.3 μ) fibres, which are not considered pathologically important.

Thus, the emission of free fibres resulting from brake lining wear is a negligeable health risk factor of urban air pollution. Indeed, estimates of air concentrations of asbestos resulting from vehicular brakes in large US cities range from 0.051 ng/M^3 (Rochester, NY) to 0.258 ng/M^3 (Los Angeles, CA). If a conversion factor of 30 fibres measured optically per nanogram of asbestos is used, the values for Los Angeles would be 7.74 f/M^3 or 0.000007 f/c. Published evidence pertinent to the above considerations is found in the following references, grouped under two headings:

a: decomposition of asbestos resulting from brake use;

b: asbestos concentrations measured in urban air resulting from vehicular brakes.

a) Decomposition of asbestos resulting from brake use

Lynch, J.R. (1968). Brake lining decomposition products.
Journal of the Air Pollution Control Association 18(12):824-826.
This study by investigators of the US Department of Health, Education and Welfare, Public Health Service (Cincinnati) provides evidence from analysis of dust obtained from inside brake drums removed for brake relining, and also from laboratory experiments devised to permit sampling decomposition products of the lining under operating conditions. In all but a few tests, the automobile drum brake linings showed less than 1% free fibres in the decomposition products, as compared to about 50% in the lining. In those laboratory tests where a significant mass of free fibres was released, the temperature applied was in an extremely high range for the lining in question; had these linings been subjected to similar conditions in a vehicle, the brakes would have failed. The authors conclude: "Only a very small proportion of the asbestos worn from brake linings is released as free fibre; the remainder is converted into some other mineral as a result of the extreme temperatures generated at small spots on the lining surface. Thus, although urban air contains a few free fibres as a result of brake lining wear, they represent a very small proportion of the total asbestos used in manufacture of brakes".

Jacko, M.G., DuCharme, R.T. and Somers, J.H. (1973). Brake and clutch emissions generated during vehicle operation.
Society of Automotive Engineers, Reprint #730548:1813-1831.
In this report by scientists from the Bendix Corporation and the US EPA, the authors state that on the average, more that 99.7% of the asbestos during vehicle operation is trapped or emitted as olivine or forsterite particles.

Le Bouffant, L., Bruyère, S., Daniel, H., Martin, J.-C., Henin, J.P., Tichoux, G. et Nattier, P. (1983). Influence d'un traitement thermique des fibres de chrysotile sur leur comportement dans le poumon.
Pollution Atmosphérique, Janvier-Mars:44-49.

In this study, samples of chrysotile asbestos have been heated to various temperatures, up to 1,300°C. Analyses by electron diffraction show that at 700°C, the chrysotile structure is modified, and x-ray diffraction shows that it is transformed into forsterite. Injection of 20 mg dose of this material into the pleural cavity of rats did not produce a single tumour.

Rohl, A.N., Langer, A.M., Wolff, M.S. and Weisman, I. (1976). Asbestos exposure during brake lining maintenance and repair. Environmental Research 12:110-128.
In this study from the Mount Sinaï School of Medicine, the authors have analyzed the composition of wear debris from brake drum dust of automobiles, and found that in general only 3 to 6% by weight was recognized asbestos (implying that 94 to 97% was some other material). Furthermore, the authors determined that 80% of the small fraction of asbestos found in the wear debris were shorter than 0.37μ in length, which means that perhaps only 1% of the fibres would be longer than 5 μ.

ORCA (1984). Report of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario, pages 571 and 574.
In volume 2 of the Report, the commissioners indicate that according to Sébastien, who has conducted extensive mass measurements, 2 f/cc measured optically are approximately equal to 100,000 nanograms/M^3 based on TEM analysis. This conversion would mean that 1 nanogram of asbestos contains 20 fibres. However they indicate in their Report that they have used a 30 f = 1 ng conversion factor, which is suggested by EPA.

b/ Asbestos concentrations measured in urban air resulting from vehicular brakes.

Anderson, A.E., Gealer, R.L., McCune, R.C. and Sprys, J.W. (1973). Asbestos emissions from brake dynamometer tests. Society of Automotive Engineers, Reprint #730549:1832-1841.
This report by the Scientific Research Staff, Ford Motor Corporation, indicates that asbestos TEM analysis of sampled air during brake-in, normal use and high temperature conditions in dynamometer tests of

production disk pads show that most of the lining asbestos is found to be converted to a nonfibrous material by the high flash temperatures of the braking surface, and that less than 0.02% of the lining wear is released as asbestos fibres. The concentration of asbestos fibres in urban atmosphere, due to brake usage, was conservatively estimated at less than 0.07 nanogram/M^3. Using the conversion factor just mentioned in the reference from ORCA (1 ng = 30 fibres), this value becomes 0,0000021 f/ml.

Versar, Inc.(1987). Revised Draft Report/Nonoccupational Asbestos Exposure.
EPA Contract No. 68-02-4254, Task No. 31, September 25.
In this Report prepared for the US EPA (pages 2-1 to 2-27), the authors estimate that the national ambient asbestos concentration from vehicle brakes is 0.057 nanogram/M^3 (0,0000017 f/ml), with Los Angeles showing the highest estimate at 0.258 ng/M^3 (0.0000077 f/ml).

ASBESTOS CEMENT

It should be mentioned at the ouset that the risk of any health effects from non-friable asbestos in public buildings is regarded by most authors to be non-existent or extremely low and the cost of removal not warranted[6] .
Regarding the contribution to the environment resulting from the use of high-density asbestos-containing construction materials, the following observations are pertinent.

Teichert U.(1986) Immissionen durch Asbestzement-Produkte,Teil 1 Staub Reinhaltung der Luft, Vol. 46, No. 10, pp. 432-434 (1986)
..."The study of immission conducted on coated and uncoated roofing materials revealed low asbestos fibre concentrations, even though severe corrosion was observed on uncoated asbestos cement roofs and a considerable quantity of material containing asbestos could be removed by

[6] **Whysner J, Covello VT, Kuschner M, Rifkind AB, Rozman KK, Trichopoulos, Williams GM (1994)** Asbestos in the air of public buildings: A public health debate? Prev Med 23: 119-125

blowing or suction. The asbestos fibre concentrations that were measured in populated areas are well below the level considered acceptable by the Health Authorities of the Federal Republic of Germany(5), i.e. clearly below 1000 fibres/M^3 (length ≥5 µm)". (1000 fibres/m^3 = 0.001 f/ml)

W. Felbermayer and M.B. Ussar (1980) Research Report: "Airborne Asbestos Fibres Eroded from Asbestos Cement sheets". Summary Institut für Umweltschutz und Emissionsfragen, Leoben, Austria.

..."A comparison of the asbestos fibre concentrations in those areas with and without A/C roofing... lead to the conclusion that there is no statistically significant connection between the use of asbestos cement materials and the asbestos fibre concentrations found in the various measurement areas".

Airborne asbestos fibres (L >5µ; D <3µ) measured in:

-Urban area with heavy traffic:	4.6 f/Litre (0.0046 f/ml)
-Area of naturally-occurring asbestos:	0.2 f/Litre (0.0002 f/ml)
-Urban area with A/C roofing:	<0.1 f/Litre (0.0001 f/ml)
-Urban are without A/C roofing:	<0.1 f/Litre (0.0001 f/ml)

ASBESTOS CEMENT IN SCHOOLS

Concern has been expressed by the public and the news media regarding possible adverse effects on the health of children (in particular), of asbestos fibres released from weathered asbestos cement products in schools and other buildings.
In Australia, a "Working Party on Asbestos Cement Products" was set up by the Western Australia (WA) Advisory Committee on Hazardous Substances. An interim report was to be presented on December 1989 to the Minister of Education on the above matters with reference to asbestos cement in schools. A final report to the WA Advisory Committee on Hazardous Substances was published in August 1990.

Highlights of the report.
The report contains different sections: description of asbestos cement products; production and use; effects on health; surveys of schools and other relevant measurements of asbestos

concentrations. In addition to pertinent recommendations, the report also contains several appendices, including one on "The Effects of Asbestos Cement Products - A Review of the Literature", and one on "Acceptable Air Concentrations of Asbestos Fibres in the General Environment", both prepared by Dr.Nicholas de Klerk of the Medical Research Council Epidemiology Unit, UWA.

The overall impression from the report can probably be best summarized by de Klerk's own conclusion on risk estimates at low air concentrations of asbestos: *"Most of these estimates are on or below the level of what the Royal Society would consider acceptable. They are however above acceptable US levels. The 1986 IPCS report did not even bother to estimate such risks and summarized the risk exposure unrelated to occupation as being undetectably low"*.

Indeed, the Executive Summary indicates:

> "1.7 For school children, risk estimates, extrapolated from occupational situations, indicate that even in "worst case" situations asbestos cement weathering is likely to result in less than one additional death per million persons per year. This is some 100 times less than the normal risks taken by such children in the process of growing up. It may also be compared with a risk of death from all causes for a 40 year old male of 2000 per million persons per year. The level of risk is low enough to be considered to be negligible relative to these other risks in our society".

The Executive Summary also mentions that based on air monitoring results, *"... estimates of the concentration of asbestos fibres in the air around schools with asbestos cement roofs in Western Australia suggest that the concentrations are unlikely to exceed 0.002 fibres per ml and are more likely to be less than 0.0002 fibres per ml. These observations,*

together with what is known from other experience (see appendix 2) would suggest that asbestos cement products in schools present a negligible risk to health".

With regard to the control of asbestos fibres release from in-place asbestos cement products, the report indicates that: "The final results of research undertaken by the WA Advisory Committee on Hazardous Substances indicate negligible risk to health from asbestos cement products. The Committee concludes therefore that it is not necessary on health grounds to require the use of coating agents or other similar containment systems on asbestos cement product".

The Committee expresses concern that some persons may be induced to treat roofs as a result of advertising based on unfounded claims of health risks associated with asbestos cement roofs. The Report[7] mentions: "An asbestos cement roof which has not deteriorated to an extent where physical safety or structural integrity is of concern, should not be replaced. In addition, an asbestos cement roof should not be treated with a coating on the basis of risk to health. Other asbestos cement products are generally less prone to deterioration and do not require attention for health purposes". (Recommendation 2.1).

Asbestos cement pipes

The use of asbestos-cement (A/C) pipes dates back to the early 1920's, and it is estimated that by the end of the 1990's, 3 to 4 million kilometers of pipes will have been laid worldwide to convey potable water. Highly aggressive waters may attack the cement matrix, and consequently lead to the release of fibres into the water circulating through the pipes, and A/C pipes are not recommended for use under such highly corrosive conditions, unless protected with specially designed internal

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[7] Copy of the Report may be obtained from: Chief Scientific Officer (Hygiene), Department of Occupational Health, Safety and Welfare of Western Australia, Westcentre, 1260 Hay Street, P. O. Box 294, West Perth (WA), 600 AUSTRALIA.

linings. The results of most studies published so far indicate that the source waters already contain asbestos fibres (mostly shorter than 1 μ in length) before passing through the A/C pipe systems, often in numbers reaching several millions per liter, and it is generally agreed that A/C pipes do not appreciably raise the asbestos fibre content of water, and that the quantities found are within those which occur naturally.

As to the risk for health resulting from the presence of asbestos in potable water, results of several years of laboratory investigations in animals fed for their entire lifespan very large (several billions of fibres per day) quantities of asbestos incorporated into their diet have consistently failed to indicate any raised incidence of gastrointestinal tumours, or of any other pathological changes in the gastrointestinal tract. Epidemiological studies on human health effects related to asbestos levels in drinking water have failed to indicate any increased risk of alimentary tract tumours following the direct ingestion of asbestos fibres. Published evidence in support of the above three points are found in the following references under the three sub-headings:

a/ presence of asbestos in public drinking water supplies.
b/ ingestion of asbestos: results of animal studies.
c/ ingestion of asbestos: results of epidemiological studies.

a) Presence of asbestos in public drinking water

Hallenbeck, W.H., Chen, E.H., Hesse, C.S., Patel-Mandlik, K. and Wolff, A.H. (1978). Is chrysotile asbestos released from asbestos cement pipe into drinking water.
Journal of American Water Works Association 70(2):97-102.
A study of 15 water supply systems in the State of Illinois (U.S.A.) where some asbestos cement pipes were up to 50 years old, and where the water was non-aggressive to moderately aggressive, showing no

significant differences before and after passing through the asbestos-cement pipe network.

Commins, B.T. (1983). Asbestos fibres in drinking water. Scientific and Technical Report-STR1, Commins Associates, Maidenhead, U.K.:1-73.
This report contains a table (pages 38-44) where the concentrations of asbestos fibres in drinking waters for several locations in Canada, U.S.A., U.K. and Sweden have been tabulated, along with the references to the studies. The table indicates that asbestos fibre concentrations in drinking water range from zero to 1,800 millions per liter.

b) Ingestion of asbestos: results of animal studies.

Truhaut, R. and Chouroulinkov, I. (1989). Effect of long-term ingestion of asbestos fibres in rats.
In Non-Occupational Exposure to Mineral Fibres, Eds. J. Bignon, J. Peto and R. Saracci. WHO/IARC Scientific Publications No. 90, Lyon:127-133.
A study in which rats were fed mixtures of asbestos incorporated in palm oil. The animals were fed daily for 24 months, and surviving animals were kept under observation for a further 6 month-period. The results led the authors to conclude: "In conclusion, the ingestion of chrysotile or of a mixture of chrysotile/crocidolite (75%/25%) at various doses, and even at high ones, did not adversely affect the health of rats and there was no evidence of any increase in tumours of the alimentary tract or of any general increase in tumour frequency".

Bolton, R.E., Davis, J.M.G. and Lamb, D. (1982). The pathological effects of prolonged asbestos ingestion in rats.
Environmental Research 29:134-150.
A study in which the authors, confirming the results of two earlier investigations, find no excess of malignant tumours and no gastrointestinal mucosal abnormalities in laboratory animals after prolonged (up to 25 months) ingestion of asbestos fibres. The authors

state that their work "...suggests that the normal healthy gastrointestine maintains an effective barrier against the potentially damaging effect of ingested asbestos...".

c) Ingestion of asbestos: results of human studies.

Toft, P., Wigle, D., Meranger, J.C. and Mao, Y. (1981). Asbestos and drinking water in Canada.
The Science of the Total Environment 18:77-89.
After reviewing the epidemiological studies in Canadian cities, the conclusion was that these studies provide no consistent, convincing evidence of increased cancer risk attributable to the ingestion of drinking water contaminated by asbestos, even though the observed asbestos concentrations were relatively high in several communities. Worthy of note are the lower mortality rates for all gastrointestinal cancers combined in the Sherbrooke (Québec) area, where there is a high (~150 million fibres per liter) concentration of asbestos fibres in drinking water supplies, when compared with cities with lower concentrations.

Conforti, P.M., Kanarek, M.S., Jackson, L.A., Cooper, R.C. and Murchio, J.C. (1981). Asbestos in drinking water and cancer in the San Francisco Bay area: 1969-1974 incidence.
Journal of Chronic Diseases 34(5):211-224.
The only report among more than half-dozen studies of health and asbestos in drinking water that suggests a relationship with gastrointestinal cancer, and even there, the suggested relationship is weak, because only a fraction of the many analyses performed by Conforti and his co-workers pointed such a relationship, and also because the authors admitted that important confounding factors such as smoking, occupational history and alcohol consumption were not considered in their study.

Meigs, J.W., Walter, S.D., Heston, J.F., Millette, J.R., Craun, G.F., Woodhull, R.S. and Flannery, J.T. (1980).

Asbestos-cement pipe is no danger in Connecticut. The state needn't change its distribution network.
Water and Sewage Works 127(6):66-93.
The principal author of this report, Dr. J. Walter Meigs, Director of the Connecticut Cancer Epidemiology Unit, and Clinical Professor of Epidemiology at the Yale University School of Medicine states: "The lack of evidence for cancer risks from the use of A/C pipe is reassuring. It is consistent with most studies from other areas of the U.S.A. The results provide no evidence for changing current water distribution policies for Connecticut water supplies because of A/C pipe use".

Polissar, L., Severson, R.K., Boatman, E.S. and Thomas, D.B. (1982). Cancer incidence in relation to asbestos in drinking water in the Puget Sound region.
American Journal of Epidemiology 116(2):314-328.
The site of the study was the Puget Sound region of Western Washington, and the state's three largest metropolitan areas (Everett, Seattle and Tacoma) were used for comparison. Everett was the "high exposure municipality", where asbestos levels ranged from 37.2 to 556 million fibres per liter. Seattle and Tacoma had relatively low concentrations, averaging 7.3 million fibres per liter. The three metropolitan areas were subdivided into census tracts grouped by asbestos concentration. Data on cancer incidence were obtained from a surveillance registry; cancer mortality information came from death certificates. Duration of exposure to asbestos in drinking water was estimated and divided into long term (greater than 30 years) versus short term (less than 30 years) groups. Following the analysis of the results the principal investigator, Dr. Lincoln Polissar of the Fred Hutchinson Cancer Research Center, concluded that: "Results of this study and prior studies of cancer in relation to waterborne asbestos are inconsistent, and provide little evidence that asbestos in community water supplies has altered the risk of any cancer".

MacRae, K.D. (1988). Asbestos in drinking water and cancer.
Journal of the Royal College of Physicians of London 22(1):7-10.

In this review article, the author concludes: "it would thus seem highly unlikely that the asbestos-cement pipe distribution system makes any biologically significant contribution to the asbestos content of water passing through it"."...It is highly improbable that asbestos release from asbestos-cement pipes is relevant to the development of cancer".

Millette, J.R., Craun, G.F., Stober, J.A., Kraemer, D.F., Tousignant, H.G., Hildago, E., Duboise, R.L. and Benedict, J. (1983). Epidemiology study of the use of asbestos-cement pipe for the distribution of drinking water in Escambia County, Florida. Environmental Health Perspectives 53:91-98.

Some areas in Florida have been receiving drinking water through asbestos-cement pipes for 30-40 years. The authors mention: "No evidence for an association between the use of AC pipes for carrying drinking water and deaths due to gastrointestinal and related cancers was found in this study".

GENERAL CONCLUSIONS

For all natural and man-made fibrous respirable materials, fibre dimensions (length and diameter) and selective retention times (biopersistence) must be considered in assessing health hazard and risk.

Adverse effects are associated with fibres that are retained in the lung rather than with those which are cleared.

Chrysotile is cleared rapidly from the lung, whereas amphiboles (crocidolite and amosite) are characterized by extremely long biopersistence.

The "hit-and-run" hypothesis is at odds with the evidence from biopersistence studies.

Evidence from morbidity, mortality and lung burden studies support the concept of a much lower pathogenic potential for chrysotile compared to the amphiboles.

These differences should be considered when setting workplace threshold limit values (TLV).

Recent updates of epidemiological studies are consistent with a practical threshold level of exposure below which no adverse effects are detectable.

The health risks associated with chrysotile exposure concern the workplace; risks for the general population, if they exist, are "below detection limits".

With normal use and maintenance, fibre emission from modern, high-density asbestos composites such as friction and asbestos cement materials is minimal, and does not constitute a measurable risk to the general population nor to the environment.

Risks are associated with inhalation, not ingestion. Thus, asbestos cement pipe materials are safe, as epidemiological studies have failed to show demonstrable risks.
