Today’s chrysotile world 2008
Introduction

For many decades throughout the whole world, very few, if any, natural or synthetic products or substances have caused such debate and conflict as “Asbestos”.

Asbestos has been in the grip of a remarkable and very dangerous psychological phenomenon. Repeatedly we have seen the rise of absolute fear centered on perceived danger to human health. People are told and they faithfully believe that millions of people will suffer and die and this threat of a global disaster has been the springboard of the ban asbestos strategists.

Very few products, substances or minerals, natural or man-made, have been studied as closely as asbestos. Recent progress in understanding the mechanisms of action has been impressive, especially in the past two decades, when technology made it possible to understand how breathable fibres can affect the human body, in particular the size of the fibres, their biopersistence in the lungs and the level of exposure (dose). Research has clearly shown and proven that great differences exist between asbestos fibres and, thus, demonstrated that chrysotile fibres are really safer and can be used safely.

Today, distinction has to be made between chrysotile and the amphiboles varieties of asbestos. Numerous and exhaustive studies provide robust data and underscore the major difference in health risks between chrysotile and amphiboles. With the application of control measures, high-density products and the use of uncontaminated chrysotile fibres, there is no significant health risk for workers, the environment or the general public.

Chrysotile is used only in high-density products where fibres are encapsulated in a matrix and 95% of world use is in cement products. Even knowing the difference between chrysotile and amphiboles, some persons still refuse to acknowledge their inherent differences and demand a complete ban on all asbestos fibres, including chrysotile. The proposed replacement or alternative fibres in many cases have not been scientifically proven safer, or as safe, as chrysotile.

Over the years, the combined efforts by governments, industry and labour organizations in several countries have successfully promoted and implemented guidelines for the safe and responsible-use of chrysotile fibres. These efforts must continue!

These documents, which we are pleased to offer you, are not intended to be an exhaustive compendium of the knowledge amassed, as there are thousands of scientific papers written on asbestos and chrysotile. The objective here is to give readers a comprehensive overview of today’s chrysotile world.

TABLE OF CONTENTS

Introduction ..................................................... 1
A Review of recently published Evidence by Jacques Dunnigan ...............2 to 8
Update of the Most Recently Published Evidence...................................09 to 10
General Conclusion ............................................. 11
Appendix ..............................................12 to 24
Presentation of The Asbestos Saga Myths vs Reality.................................25
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 that 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 commentary dates back to July 1997 (Alleman JE and Mossman BT: “Asbestos Revisited”; Scientific American, July 1997; pp. 70-75). More recently, a position paper by the American Council on Science and Health (ACSH) entitled « ASBESTOS EXPOSURE: HOW RISKY IS IT ? (October 2007) updated the published scientific evidence and indicated : « The challenge today is whether regulatory agencies will utilize current scientific knowledge even though it will necessitate a paradigm shift in long-held views on asbestos exposure and its implications for human health. »

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 that invariably leads to bad risk management decisions regarding asbestos is the so-called “hit-and-run” view that 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 outdated 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, and still pertinent today, relate to the importance of fibre dimensions (length and diameter) as prerequisites for biological potency, since these two parameters are related to respirability. It is generally recognized that fibrous structures with a diameter $\leq 3\mu$ and a length $\geq 5\mu$ are particles that will penetrate deeply into the respiratory tree. There is a very large 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 that varies widely amongst different respirable particles. Durability is likely related to the different chemical structures and crystalline habits of mineral particles. Durability will determine the extent of a key biological phenomenon: biopersistence. It can be described as a time period 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, both displaying exceedingly long
biopersistence. In addition, data presented at the symposium indicated that various types of glass fibres also have different solubilities and biopersistence characteristics 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 oxides 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-time » (the typical form of expression for biopersistence) for fibre elimination from the lung ranged from 10 to 500 days. A study from the U.S.A. also reported that inhaled RCFs show 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 the use of respirable materials. In 1995, the Fraunhofer Institute scientists reiterated their view in these words:

“biopersistence of inhaled fibrous materials is a critical factor in determining carcinogenic potency”. ³

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

¹ Wagner JC and Pooley FD (1986) Thorax 41: 161-166
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
CONCLUSIONS
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 (APPENDIX 1):

a. Morbidity and mortality data in “chrysotile only” users;
b. Analysis of mineral lung content (human data only).

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 chryso-
tile 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 substanc-
es. 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 a practically 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 require-
ments 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 diseases in workers. References to these studies illustrate this point (APPENDIX 2).
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 this update 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 workers, but higher than those of general populations elsewhere. References to these studies appear in (APPENDIX 3).

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

**ASBESTOS IN FRICTION MATERIALS**

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 negligible 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/M³ (Rochester, NY) to 0.258 ng/M³ (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/M³ or 0.000007 f/cc. Published evidence pertinent to the above considerations is found in the following references, grouped under two headings (APPENDIX 4):

a. decomposition of asbestos resulting from brake use;

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

It should be mentioned at the outset 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.5

Regarding the contribution to the environment resulting from the use of high-density asbestos-containing construction materials, the evidence which appears in APPENDIX 5 is pertinent.

ASBESTOS CEMENT IN SCHOOLS

Concern has been expressed by the public and in 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 report6 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. The final WA 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”.


6 Copies 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.
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 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 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 (APPENDIX 6):

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.
UPDATE OF THE MOST RECENTLY PUBLISHED EVIDENCE

In this last section, it will be seen that the review of the more recently published scientific evidence (from 1997 to 2006) brings unequivocal support to the vast differences between chrysotile and the amphibole varieties of asbestos. Evidence published during this period includes the following:


**Bernstein D, Rogers R, Smith P (2005).** The Biopersistence of Canadian Chrysotile Asbestos Following Inhalation: Final Results Through 1 Year After Cessation of Exposure. Inhal. Toxicology 17 : 1-14


The four publications mentioned above by Bernstein et al (2003, 2003, 2005 and 2006) relate to the phenomenon of biopersistence of inhaled particles. These animal experiments, performed according to the most stringent protocols recognized by the EU, show that soon after chrysotile fibers are inhaled, they are quickly cleared from the lung, whereas amphiboles, which resist the acidic environment of the pulmonary environment, are not cleared as rapidly and remain in the lung for periods up to a year or more. These animal experiments thus bring robust support to the many epidemiological observations published in the past, as well as the more recent benchmark publication by Hodgson and Darnton (2000), showing that amphiboles are orders of magnitude more potent than chrysotile.

The publication by Paustenbach et al (2004) is a “state-of-the-art” review of the risk associated with the use of asbestos in the manufacture of friction materials and their use in the general automotive service industries. This review, covering studies and observations published over several decades, demonstrates that in general, exposures have been minimal and did not show any demonstrable risk when chrysotile was used, and that the relatively few instances of increased health risks were always associated with the use of amphiboles.

The epidemiological studies by 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). More recently, the multi-centre case-control study in Europe by Carel R et al (2006) has shown that occupational exposure to asbestos does not appear to contribute to the lung cancer burden in men in Central and Eastern Europe while in contrast, the lung cancer risk in the UK is increased following exposure to asbestos. The authors suggest that differences in fibre types and circumstances of exposure may explain their results.

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. »

Finally, following an extensive review, Yarborough (2006) states that: «Although epidemiological studies have confirmed amphibole asbestos fibers 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 fibers, with particular attention to confounding by other fiber types, job exposure concentrations, and consistency of findings. The review of 71 asbestos cohorts exposed to free asbestos fibers does not support the hypothesis that chrysotile, uncontaminated by amphibolic substances, causes mesothelioma. »
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 characterizing health hazard and assessing risk.

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

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 for chrysotile 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 chrysotile composites such as friction and fibro-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, chrysotile-cement pipe materials are safe, as epidemiological studies have failed to show demonstrable risks.
THE PATHOGENIC DIFFERENCES BETWEEN ASBESTOS FIBRE TYPES.

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”.

APPENDIX 1
• 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.


• 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.


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., 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.


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%.


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.


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.


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 and 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 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).”
HEALTH EXPERIENCE OF WORKERS AT VERY LOW EXPOSURE LEVELS TO CHRYSOTILE ONLY

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”.

The study referred to above 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 state: “It is concluded that with good environmental control, chrysotile asbestos may be used in manufacture without causing excess mortality”.

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”.

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”.

APPENDIX 2

• **Gardner, M.J., Winter, P.D., Pannett, B. and Powell, C.A. (1986).** Follow up study of workers manufacturing 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, 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 Liddell FDK, McDonald JC and McDonald A. Ann. Occup. Hyg. 41:13-35 (1997)

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 recent 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.
EXPERIENCE OF THE GENERAL POPULATION

- **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:


ASBESTOS EMISSIONS FROM FRICTION MATERIALS

A. Decomposition of asbestos resulting from brake use

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”.

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.

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.

In this study from the Mount Sinai 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.
In volume 2 of the Report, the commissioners indicate that according to Sébastien, who has conducted extensive mass measurements, 2 \( \text{f/cc} \) measured optically are approximately equal to 100,000 nanograms/\( \text{M}^3 \) 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 \( \text{f} = 1 \text{ ng} \) conversion factor, which is suggested by EPA.

B. Asbestos concentrations measured in urban air resulting from vehicular brakes

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/\( \text{M}^3 \). Using the conversion factor just mentioned in the reference from ORCA (1 ng = 30 fibres), this value becomes 0.0000021 \( \text{f/ml} \).

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/\( \text{M}^3 \) (0.0000017 \( \text{f/ml} \)), with Los Angeles showing the highest estimate at 0.258 ng/\( \text{M}^3 \) (0.0000077 \( \text{f/ml} \)).
EMISSIONS FROM ASBESTOS CEMENT CONSTRUCTION MATERIALS

**Teichert U. (1986).** Immissionen durch Asbestzement-Produkte, Teil 1

"...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 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/M³ (length ≥ 5 µm)." (1000 fibres/M³ = 0.001 f/ml)

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 area without A/C roofing: <0.1 f/Litre (0.0001 f/ml)
ASBESTOS IN WATER

A. Presence of asbestos in public drinking water

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.

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.

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

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”.

APPENDIX 6

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


A Canadian study of water-borne asbestos levels and mortality rates in 71 municipalities across Canada, where the authors conclude that there was no significant relationship between water-borne asbestos levels and gastrointestinal cancer.


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.


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”.


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”.
The problem with myths is that their repetition, over and over again... may lead millions to assume that they represent the reality.

« Plain truths will influence half a score of men in a nation or an age, while mysteries will lead millions by the nose ».  

Henry St-John, Viscount Bolingbroke (1678-1751)  
British Philosopher and Parliamentarian
What is asbestos?

First, a few simple facts
Just like the word « metal » covers a wide variety of substances such as iron, copper, lead, mercury, cadmium, tin etc.

the word « asbestos » is a commercial term referring to no less than six different types, each with different physico-chemical characteristics, and each having a distinct toxicity potential
ASBESTOS

Fibrous Serpentine
CHRYSO TILE
Mg₃(Si₂O₅)(OH)

Fibrous Amphiboles

AMOSITE
(Fe, Mg)₇
(Si₈O₂₂)(OH)₂

TREMOLITE
Ca₂Mg₃
(Si₈O₂₂)(OH)₂

CROCIDOLITE
Na₂Fe²⁺₃, Fe³⁺₂
(Si₈O₂₂)(OH)₂

ACTINOLITE
Ca₂(Mg, Fe)₅
(Si₈O₂₂)(OH)₂

ANTHOPYLLITE
(Mg, Fe)₇
(Si₈O₂₂)(OH)₂

THEORETICAL FORMULA
Asbestos

some features of chemical composition

- All six types are hydrated silicates
- They differ appreciably by their metal content

<table>
<thead>
<tr>
<th></th>
<th>CHrysotile</th>
<th>CROCIDOLITE</th>
<th>AMOSITE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fe₂O₃</td>
<td>0 - 5</td>
<td>13 -18</td>
<td>0 -5</td>
</tr>
<tr>
<td>FeO</td>
<td>0 - 3</td>
<td>3 - 21</td>
<td>35 - 40</td>
</tr>
<tr>
<td>MgO</td>
<td>38 - 42</td>
<td>0 - 13</td>
<td>5 - 7</td>
</tr>
</tbody>
</table>
PRESENT CONSENSUS ON KEY PARAMETERS OF BIOLOGICAL ACTIVITY FOR FIBROUS MATERIALS

**DIMENSIONS**: « RESPIRABLE » \( (L \geq 5 \text{ microns} - D \leq 0.3 \text{ microns}) \)

**DURABILITY**: Related to biopersistence.

**DOSE**: Depending on fiber types, there are low levels of exposure with no measurable effects;
- this may be true for less biopersistent fibers;
- not so for highly biopersistent fibers.
The « 3 Ds » (dimension, durability, dose)

- These three « Ds » are parameters that apply for all respirable fibrous materials.
- What about the asbestos fibers?
- Are there significant differences according to these parameters?
BIOPERSISTENCE

An important phenomenon in the factors affecting the toxicity of inhaled particles.

It can be simply described as the time an inhaled particle remains in the lungs before it is eventually cleared.
“...the importance of selective retention of fibers has been discussed in a recent paper. We are convinced that those diseases associated with exposure to mineral fibers are due to fibers retained in the lungs”.

“The findings thus support the hypothesis that adverse effects are associated rather with the fibers that are retained (amphiboles), than with the ones being cleared (largely chrysotile)”

“Given what we now know, it would be foolhardy, without extraordinary justification, to allow the widespread use of fibers which resemble crocidolite and tremolite physically, and the amphiboles generally in their biological biopersistence”
McDonald JC and McDonald AD (1996) Eur Respir J 9:1932-1942

“Many organic fibers are durable and thus have the potential to persist within the lung and cause disease”
Cullen et al. (2002) Inhalation Toxicology 14 : 685-70

Note how consistent this view is: (1986 to 2002)
PUBLISHED EVIDENCE

LUNG BURDEN STUDIES SHOWING DIFFERENT BIOPERSISTENCE BETWEEN CHrysotile AND AMPHIBOLES
# DIFFERENCES IN ASBESTOS FIBER TYPES

## MINERAL ANALYSIS OF LUNG TISSUE (1)

<table>
<thead>
<tr>
<th>GROUPS STUDIED</th>
<th>OBSERVATIONS</th>
</tr>
</thead>
</table>
| Workers with asbestos-related Diseases reported to the U.K. Pneumoconiosis Panel in 1977 (1) | **Amphiboles**: 100-fold increase in Cases over controls.  
**Chrysotile**: equal amounts in cases and controls |
| Workers with asbestosis in Royal Navy Dockyards for the period 1966-1982 (2)       | **Amphiboles**: amounts increase with severity of asbestosis.  
**Chrysotile**: amounts remain equal.                                      |
| Workers with mesothelioma reported In British Columbia in 1982 (3)                | **Amphiboles**: 300-fold increase compared to general population.  
**Chrysotile**: no difference.                                             |

## DIFFERENCES IN ASBESTOS FIBER TYPES

**MINERAL ANALYSIS OF LUNG TISSUE (2)**

<table>
<thead>
<tr>
<th>GROUPS STUDIED</th>
<th>OBSERVATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with pleural plaques in British Columbia (4)</td>
<td>50-fold increase of amphiboles compared to chrysotile</td>
</tr>
<tr>
<td>Patients with mesothelioma in U.K. in 1976 (5)</td>
<td>Far greater number of amphiboles fibers, but same number of chrysotile fibers in cases compared to controls</td>
</tr>
<tr>
<td>Patients with mesothelioma in North America (6)</td>
<td>Far greater number of amphiboles fibers, but same number of chrysotile fibers in cases compared to controls</td>
</tr>
</tbody>
</table>

Confronting Myths and Reality
“There is no threshold, as for all other varieties of asbestos (amphiboles).”

MYTH # 1
Recently published toxicological data provide strong support for epidemiological observations published over the last 25 years.
# EVIDENCE FOR A NO-EFFECT (THRESHOLD) DOSE FOR CHRYSOTILE ASBESTOS

## Epidemiological Evidence (A)

<table>
<thead>
<tr>
<th>Exposure</th>
<th>Fiber Type Used</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asbestos cement factory (1) (1176 workers / Sweden)</td>
<td>Chrysotile</td>
<td>No asbestos-related excess mortality, at average exposure levels of 1 - 2 f/cc.</td>
</tr>
<tr>
<td>Asbestos cement factory (2) (2167 workers / U.K.)</td>
<td>Chrysotile</td>
<td>No excess of lung cancer or other asbestos-related excess death at level of exposure of 1 f/cc, and possibly higher.</td>
</tr>
<tr>
<td>Asbestos cement factory (3) (U.K. factory: 36-year follow-up)</td>
<td>Chrysotile</td>
<td>No excess death from lung cancer or other asbestos-related tumours, or from chronic respiratory diseases, at levels not exceeding 1 f/cc.</td>
</tr>
</tbody>
</table>

## EVIDENCE FOR A NO-EFFECT (THRESHOLD) DOSE FOR CHRYSOTILE ASBESTOS

### EPIDEMIOLOGICAL EVIDENCE (B)

<table>
<thead>
<tr>
<th>EXPOSURE</th>
<th>OBSERVATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Friction materials factory (U. K.) (1)</td>
<td>Death rates from lung, G. I. tract and all cancers: same as national rates.</td>
</tr>
<tr>
<td>Asbestos cement factory (U. K.) (2)</td>
<td>No raised SMR for lung, pleural, G. I. tract and all cancers.</td>
</tr>
<tr>
<td>Asbestos cement factory (U. S. A.) (3)</td>
<td>No raised morbidity, no raised SMR at ≤100 mpcf (~15 f/cc).</td>
</tr>
<tr>
<td>Residents of chrysotile mining towns in Québec (4)</td>
<td>No higher respiratory disease incidence.</td>
</tr>
<tr>
<td>(Atmospheric concentrations 200-500 higher than in most North American urban areas)</td>
<td></td>
</tr>
</tbody>
</table>

From 1975 to 2002, more than 25 epidemiology studies were conducted examining the risks of asbestos-related diseases in brake mechanics. The mean time-weighted average concentration was about 0.05 fibers per cubic centimeter.

These studies clearly indicated that brake mechanics were not at increased risk of adverse health effects due to exposure to asbestos. Specifically, the studies found no increased risk of mesothelioma or asbestosis in brake mechanics, and no evidence that lung cancer in this occupational group can be attributed to exposure to asbestos during brake repair.

Recommendation No. 2

"For chrysotile asbestos, it is recommended that countries currently having high limits should take urgent steps to lower the occupational exposure limit for an individual worker to 2 fibres/ml (8-hour time weighted average), based on health reasons alone. It is also recommended that countries should move quickly to lower the occupational exposure limit for an individual worker to 1 fibre/ml or below (8-hour time weighted average), if they have not yet already done so."

WHO, Oxford, April 1989
OCCUPATIONAL EXPOSURE LIMIT FOR ASBESTOS

Recommendation No. 3

"For crocidolite and amosite asbestos, on the basis of health, it is recommended that their use should be prohibited as soon as possible. Restricted use in the interim period should be exercised with great care to ensure that exposure is less than permitted for chrysotile."

WHO, Oxford, April 1989
MYTH # 2

There is no threshold for asbestos;

« One fiber kills »
Every Day...

The lungs handle an average of 12 liters of air per minute, or...

\[ 12L \times 60\text{min} \times 24\text{ hrs} = 17,280 \text{ liters/day} \]
General population

Assuming environmental air may contain 0.001 fibers / ml, or...

1 fiber per Liter

It follows that...
every day the lungs handle 17,280 fibers
A concentration of 0.001 f/ml (or 1 f/liter) is considered:

« Acceptable » (ORCA)  🇨🇦

« Not significant » (WHO)  🇨🇭

« Further control not justified » (The Royal Society, London)  🇬🇧

« ...en ce qui concerne la population générale, la situation pour un taux moyen de 1 fibre par litre est parfaitement sûre »

(Académie Nationale de Médecine, France, 1996)  🇫🇷
WORKERS...
For a threshold limit value (TLV) of...

0.1 f/ml*  
i. e.: 100 f/liter
* in the U. S. A.

1 f/ml**  
i. e.: 1,000 f/liter
** WHO « Group of Experts »  
Recommendation, Oxford 1989
IT FOLLOWS THAT EVERY DAY...

For a TLV of 0.1 f/ml* (100 f/liter):

\[ 12 \text{ L/min} \times 60 \text{ min/hr} \times 8 \text{ hrs/day} = 5,760 \text{ liters / work shift} \]

or \[ 100 \text{ f/L} \times 5,760 \text{ liters} = 576,000 \text{ fibers/day} \]

* in the U. S. A.
And it follows also that every day...

For a TLV of 1.0 f/ml* (1000 f/liter):
12 L/min x 60 min/hr x 8 hrs/day =
5,760 liters / work shift

or 1000 f/L x 5,760 liters =
5,760,000 fibers/day

* WHO « Group of Experts »
Recommendation, Oxford 1989
Now you can make up your mind:

Do you think that inhalation of one fiber can kill?
MYTH #3

«For the general environment, there must be a problem with the use of asbestos.»
ASBESTOS FIBERS
CONCENTRATIONS IN URBAN AIR
Possible contribution from A/C roofing materials

GERMANY

« ...the study of emission 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 blowing or suction.

The asbestos fiber concentrations that were measured in populated areas are well below the level considered acceptable by the German Health Authorities of the Federal Republic of Germany, i.e.: clearly below 1000 fibers $\text{m}^3$ (length $\geq$ 5 microns) »

Teihert, U (1986) Staub Reinhaltung der Luft
46: 432 - 434
ASBESTOS FIBERS
CONCENTRATIONS IN URBAN AIR
Possible contribution from A/C roofing materials

AUSTRIA

« ...A comparison of the asbestos fiber 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 concentrations found in various measurements areas. »

Urban areas with A/C roofing ≤ 0,0001 f/ml
Urban areas without A/C roofing ≤ 0,0001 f/ml

Felbermayer, W and Ussar, MB (1980)
Report to the Institut für Umweltschutz und Emissionsfragen
Leoben, Austria.
ASBESTOS FIBERS
CONCENTRATIONS IN URBAN AIR
Possible contribution from A/C roofing materials

AUSTRALIA

Around schools with asbestos cement roofing:

- Never exceeds: 0.002 f/ml
- Mostly: 0.0002 f/ml

Report to the « Working Party on Asbestos Cement Products »
Safety and Welfare of Western Australia (1990)
ASBESTOS AIR CONCENTRATIONS NEAR DISPOSAL SITES

GERMANY

Directly over disposal sites: 0.0005 - 0.003 f/ml

Vicinity of disposal sites: 0.0001 - 0.0009 f/ml

ASBESTOS CONCENTRATIONS IN WATER
Possible contribution from A/C piping materials

**ILLINOIS, USA:** Fifteen public water systems, up to 50 in service, non-aggressive to moderately aggressive source water. Results: no difference before and after passage through A/C pipes. (1)

**U.K.** «A/C pipes do not raise appreciably the asbestos fiber content of water, and the levels found are within the range that occurs naturally». (2)

Modern Usage of Chrysotile Asbestos Cement

Its Impact on the General Environment
Modern Usage
of
Chrysotile Asbestos Cement

Once chrysotile fibers are bound to the cement matrix, emission of free respirable fibers is negligible;

Fiber concentrations around sites are undistinguishable from those found in the general environment;

At these levels, no adverse health effects have ever been reported.
When chrysotile is used in a responsible manner . . .

Compared to products from metallurgy and from petrochemistry. . .

Less energy is used . . .

Compared to products coming from the petrochemical or metallurgical processes, asbestos-cement products consume much less energy; in fact, the largest proportion of energy consumption goes into the production of cement.
When chrysotile is used in a responsible manner . . .

Compared to products from metallurgy
and from petrochemistry. . .

Manufacture is likely less hazardous

*Composition of high density chrysotile-cement products is uniquely simple, and technology is readily available to developing countries, without resorting to the use of more complex ingredients, whose safe handling may present difficulties far greater than what is required for the controlled manufacture of asbestos-cement products.*
Modern Usage

of

Chrysotile Asbestos Cement

Disposal of rejects / demolition is simple

The safe disposal of many modern products has become an environmental and economic nightmare, often requiring specially designed and costly disposal sites and techniques. They must be monitored constantly to prevent leakage of contaminating substances into the environment, and waste management is complex and expensive for such substances. In contrast, asbestos-cement waste disposal is inexpensive, simple, safe, and recognized practices are well known.
All things considered...

Chrysotile-cement is far more environment-friendly than products derived from metallurgy and petrochemistry.
MYTH # 4

Some people argue:

« According to the IARC classification of carcinogens, Asbestos is in « Group 1 ». 

It should therefore be banned »
IARC CLASSIFICATION OF HUMAN CARCINOGENS

SIGNIFICATION and INTERPRETATION
CRITERIA AND EVALUATION FOR CARCINOGENIC HAZARD

A TWO STEP PROCESS:

The quality of evidence is assessed;

Then the hazard evaluation and classification is made.
In the « GROUP 1 » (CARCINOGENIC TO HUMANS),
AMONG THE 105 SUBSTANCES (LAST UPDATE NOVEMBER, 2007)
ARE LISTED THE FOLLOWING, QUOTED EXACTLY AS THEY APPEAR ON
THE IARC WEB SITE: http://monographs.iarc.fr/ENG/Classification/crthgr01.php

Agents and groups of agents :
Asbestos
Benzine
Cadmium
Oestrogen therapy, post-menopause
Oestrogens, both steroidal and non-steroidal
Oral contraceptives, sequential
Silica (crystalline, inhaled in the form of cristobalite)
Vinyl chloride
X-radiation and gamma radiation

(continued on the next slide)
### Mixtures:
- Alcoholic beverages
- Analgesic mixtures containing phenacetin
- Salted fish (Chinese-style)
- Tobacco smoke
- Wood dust

### Exposure circumstances:
- Aluminium production
- Boot and shoe manufacture
- Furniture and cabinet making
- Iron and steel foundry
- Painter (occupational exposure)
- Rubber industry
- Solar irradiation
Question:
Does the presence on the IARC list of « Group 1 » of substances, mixtures and industrial activities imply that these must be banned?

Answer: NO
Because the IARC classification covers only the identification and characterization (hazard) of these substances, mixtures and activities.

It does not include the assessment of risk, i.e.: the probability of toxic manifestation under actual conditions of use.
IMPORTANT DISTINCTION

« HAZARD » is not « RISK »

IARC classification is about hazard, not risk
AN IMPORTANT DISTINCTION

Characterizing a hazardous substance is not equal to assessing the true risk.

HAZARD is an essential, but insufficient component of risk assessment, which also comprises 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 expressions «HAZARD» and «RISK», untoward fear of unwelcome health endpoints such as cancer is driven by «H» data misrepresented as «R» data.

This misperception results in political response to fear, often nurtured by media taste for sensationalism, pushing regulatory action to extremes.
« Regulating trivial risk of exposure to substances erroneously inferred to cause cancer at low doses can (and probably does) harm health by diverting resources from programs that could be more effective in protecting the health of the public. »

Dr. Bruce Ames, (1993)
American Society for Cell Biology
Congressional Biomedical Caucus
Washington, DC
Because the IARC classification refers only to «hazard identification», and does not refer to «risk assessment», because the component of dose under actual conditions is absent,

it is not meant and should not be used as a «risk management» instrument for eventual regulatory action.
On this theme, A recent publication on the difference between «hazard» and «risk» and on the correct signification and interpretation of the IARC classification

Indoor & Built Environment
Bernstein D, Gibbs A, Pooley F, Langer A, Donaldson K, Hoskins J, Dunnigan J

MYTH # 5

Some people claim that « controlled use is not realistic », because vigilance and monitoring are not always adequate, or because workplace safety measures are not always easily available, etc.

If this is really the situation, the controlled-use for the other types of fibers (the substitutes) would also be unrealistic, because the techniques for the controlled-use of chrysotile are exactly the same for all airborne respirable fibers.
THE OPTIONS

NO CONTROL
HAZARD NOT RECOGNIZED
IRRESPONSIBLE APPROACH

All fibrous materials:
- Long, thin, durable
- Natural
- Synthetic
- Mineral
- Organic
- Are biologically active

Workers and general population at risk

CONTROLLED USE
HAZARD RECOGNIZED
RESPONSIBLE APPROACH

Based on scientific evidence; implies:
- regulation, implementation
- dust control; monitoring
- medical surveillance
- education
- training

Allows society to benefit safely from cost-efficient materials.

BAN
HAZARD RECOGNIZED

« LAZY MAN WAY »

Incentive to resort to uncontrolled alternatives

Deprives society of much needed materials.
"Asbestos, unlike any number of other potentially dangerous minerals or chemicals, will never be entirely eliminated from the environment. Therefore, developing improved procedures for managing its proper use, containment, and disposal offer the only realistic prospects for the prevention of asbestos-related injury and disease. In other words, it is better that society use its limited financial resources in learning how to live safely with this valuable material than in attempting to remove it totally from the environment.

Physicians and others in medicine and biology, on the other hand, must continue to drive home to the public the far greater causes of morbidity and mortality, such as smoking, drug and alcohol abuse, improper diet, and inadequate exercise.

Report by the Council on Scientific Affairs of the American Medical Association,
CONCLUSION

« The challenge today is whether regulatory agencies will utilize current scientific knowledge even though it will necessitate a paradigm shift in long-held views on asbestos exposure and its implications for human health »

ASBESTOS EXPOSURE: HOW RISKY IS IT?
A position paper of the American Council on Science and Health
Ruth Kava, Ph.D., R.D. and Eun Hye Choi
October 2007
The Chrysotile Institute
is a private organization established in 1984
by the companies producing chrysotile, unions,
and the Canadian and Quebec governments.

The Institute is dedicated to promoting the safe use
of chrysotile in Canada and throughout the world.