



A REVIEW

OF THE WORLD HEALTH
ORGANIZATION'S PUBLICATION

CHRYSOTILE ASBESTOS

The purpose of the present study is to review the points raised by the WHO's paper and in so doing, draw attention to the many factual errors, policy contradictions, and distortion of facts therein, so that an objective and facts-based understanding of the issues can be established.

EXECUTIVE SUMMARY

In March 2014, the **World Health Organization (WHO)** published an in-house paper titled, “*Chrysotile Asbestos*”. The foreword of this paper was signed by Dr. Maria Neira, Director of Public Health and Environmental and Social Determinants of Health at the WHO.

The purpose of the present study is to review the points raised by the WHO’s paper and in so doing, clarify some of the policy contradictions, factual errors, and factual distortions therein, so that an objective and facts-based understanding of the issues can be established.

FOR THE AVOIDANCE OF DOUBT,
THIS PAPER IS WRITTEN IN A
SPIRIT OF FAIR AND EQUITABLE
INFORMATION PROVISION AND FOR
THE ADVANCEMENT OF INFORMED
DECISION-MAKING AND POLICY
DEVELOPMENT.

Of note, precaution is raised by the WHO at the beginning of their paper wherein it is stated that the materials in the publication do not imply the expression of any opinion on the part of the WHO. This point bears emphasis – as it states that the WHO does not express its own opinion on the matter of chrysotile asbestos. This then raises a central question that informs much of this paper: if the WHO does not support or stand by the views in the paper, whose views are represented? And in that context, what then is the role of the WHO with regard to chrysotile?

The authors of this article argue that the role of the WHO is to promote global public health – as listed in the WHO mandate – by implementing the policies approved by the Member States that together compose the **World Health Assembly (WHA)**. In that context, the WHO should present fair and reliable information, avoid bias - in both science and policy - and eschew any form of unilateralism in interpretation that contravenes the WHA.

A review of the 2014 WHO paper quickly reveals how the paper lacks scientific credibility given that the majority of its affirmations and conclusions are not based on thorough or complete explanations, including no references to recent scientific data. As part of that pattern, the paper does not contain disaggregated information or primary source data, but is nevertheless curiously used to establish and advocate for very specific policy-oriented conclusions - conclusions that the authors argue are applicable to every form of asbestos, which disregards established biochemical and scientific facts that differentiate fiber types.

Reflecting on the positions in the WHO paper, it is evident that no new science or case studies are presented. Rather, the opposite appears to be in effect, wherein well-known studies that could potentially contribute to and/or update certain scientific assumptions have been purposefully overlooked.

Equally noteworthy is the lack of internal WHO/WHA policy consistency with regard to formally adopted positions by the WHA – in particular the 2007 decision which confirmed that in the context of eliminating asbestos related diseases (ARD's), a *differentiated approach* may be taken by Competent Authorities when regulating various forms of asbestos. Rather than reflect WHA policy, the orientation given to this paper seeks to establish a unilateral and interpretive basis to avoid and or ban the use of all types of asbestos fibers, including and especially chrysotile. This form of direct policy advocacy – directed at state-level actors, is a form of interpretive advocacy not authorized by the WHA.

On the subject of the potential dangers of exposure to asbestos, it is clear that many dire predictions trumpeted by the WHO about annual mortality rates are little more than computer models and statistical extrapolations that have never been proven in the real world. That these extrapolations ignore the science behind differentiation and that there appears to have been tremendous “message drift” wherein total cumulative potential deaths were suddenly transformed into *annual* estimates has never been explained.

In the realm of advancement of global public health, it is reasonable to assert that all stakeholders have the right to know the full realm of facts and scientific evidence as they related to every potential health threat. In this context, the WHO has the responsibility to provide to competent authorities such information in an equitable, bias-free and comprehensive manner. Regrettably, the current paper seems to be little more than an attempt to perpetuate and promote the views of vested interests, in particular the well-funded international anti-asbestos lobby – rather than reflecting the objective conclusions of science or the policy guidelines of the WHA.

Given these shortcomings, this paper hopes to fill some of the many gaps that currently exist in the narrative.



INTRODUCTION

In March 2014, the World Health Organization (WHO) published an in-house paper, the foreword of which has been signed by Dr. Maria Neira. Dr. Neira is the Director of Public Health and Environmental and Social Determinants of Health at the WHO. The title of the in-house paper is, “*Chrysotile Asbestos*”.

The purpose of the present study is to review the points raised by the WHO’s paper and in so doing, draw attention to the many factual errors, policy contradictions, and distortion of facts therein, so that an objective and facts-based understanding of the issues can be established. For the avoidance of doubt, this paper is written in a spirit of fair and equitable information provision and for the advancement of informed decision-making and policy development.

Of special significance, precaution is raised by the WHO at the beginning of the paper stating that the material in the publication does not imply the expression of any opinion whatsoever on the part of the WHO. This point bears emphasis – as it states that the WHO does not express its own opinion on the matter of chrysotile asbestos.

Further on the same first page, it is indicated that the mention of companies or manufacturer’s products does not imply that they are endorsed or recommended by the WHO. And in conclusion, that the views expressed herein do not necessarily reflect the views of organizations.

THIS RAISES A CENTRAL QUESTION THAT INFORMS MUCH OF THIS PAPER: IF THE **WHO** DOES NOT SUPPORT OR STAND BY THE VIEWS IN THE PAPER, WHOSE VIEWS ARE ACTUALLY REPRESENTED? AND IN THAT CONTEXT, WHAT IS THE ROLE OF THE **WHO**?

SUMMARY



A review of the content of the 2014 paper presented by the WHO quickly reveals that the paper lacks scientific credibility, and that the affirmations and conclusions presented are not based on thorough or complete explanations or the provision of recent scientific data.

Furthermore, the paper does not contain disaggregated information or primary source data, but curiously is used to establish and advocate for specific policy-oriented conclusions that the authors argue are applicable to every form of asbestos, regardless of established biochemical and scientific differentiation of fiber types.

Also noteworthy is the lack of internal WHO/WHA policy consistency with regard to adopted positions by the World Health Assembly (WHA) – which is the sole authority over the WHO – in particular the 2007 decision which confirmed that a differential approach can be taken by Competent Authorities when regulating various forms of asbestos fiber types. The orientation given to this paper is therefore little more than an attempt to establish a basis to *avoid and or ban* the use of all types of asbestos fibers, including chrysotile – which directly convenes the policy of the WHA.

Reflecting on the positions in the WHO paper, it is apparent that no new science or evidence or studies have been presented. Rather, the opposite appears to be the case, wherein well-known studies that could contravene or call in to question certain scientific assumptions or amalgamations have been purposefully overlooked.

In the realm of advancement of global public health, it is reasonable to assert that all stakeholders have the right to know the full realm of facts and scientific evidence as they related to every potential health threat. In this context, the WHO has the responsibility to provide to competent authorities such information in an equitable, bias-free and comprehensive manner.

POLICY FRAMEWORK

On May 23, 2007, at the World Health Assembly (WHA), the Member States agreed to pursue a Global Action Plan aimed at eliminating asbestos related diseases that stated the following:

“...ITS ACTIVITIES WILL INCLUDE GLOBAL CAMPAIGNS FOR ELIMINATION OF ASBESTOS-RELATED DISEASES BEARING IN MIND A DIFFERENTIATED APPROACH TO THE TWO FORMS OF ASBESTOS – IN LINE WITH INTERNATIONAL LEGAL INSTRUMENTS AND THE LATEST EVIDENCE FOR EFFECTIVE INTERVENTIONS AND ...”

Furthermore, at the same occasion, Assistant General Director for Health and Environment, Mrs. Susan Weber-Mosdorf stated, in response to numerous interventions from representatives of Member States, relating to asbestos and health of workers, that WHO strategies “should be considered by countries ... according to their needs and conditions.”

Because the WHA is the supreme policy making institution in the field of global public health, and until such time as a new framework is created, this is the official policy of and for the administrators and civil servants at the WHO. And in that context, the responsibility of the WHO is not to unilaterally interpret WHA resolutions, but rather to implement them to the best of their ability.

This is precisely why the content presented in the 2014 paper, demonstrates how the WHO authorities are operating in direct contravention to the policy guidelines approved by the WHA regarding a country’s right to adopt a differentiated approach to regulating asbestos in its various forms. Nowhere is that more clear than with regard to chrysotile asbestos fibers. In substance and form, this therefore is an important divergence from the WHA’s official position.

WHO UNILATERALISM

Regrettably, certain senior WHO officials such as but not limited to, the signatories of this paper, have more interest campaigning against chrysotile asbestos than defending and promoting the WHO's officially stated policy.

In so doing, they have chosen to ignore both the numerous recent scientific studies on the responsible use of chrysotile and the relevant and successfully policy choices made by Member States to differentiate and implement safe use programs. The same diversion exists on the WHO website, as it calls for a global asbestos ban in this regard (WHO's facts sheet 2016).

On many occasions, concerns have been brought to the attention of relevant and senior WHO authorities related to statements made by some officials within the organization extolling an extreme negative position on chrysotile. However, this issue has not received an appropriate response and no necessary steps have been taken by WHO authorities to remedy the situation. This is a major concern for numerous countries and their competent authorities that are using chrysotile fibers in a safe and responsible manner today. These countries represent more than 2/3's of humanity – not the minority or fringe that is often intimidated by WHO representatives.

INDUSTRIAL DISEASES



To return to the foreword in the WHO 2014 paper signed by Dr. Neira, it is stated that the use of all forms of asbestos are responsible for asbestos-related diseases, from which at least 107,000 people die each year globally. In this claim, no differentiation made between fiber types is made.

AN EXAMINATION OF CONVENTIONAL BIOCHEMISTRY AND FACT-BASED SCIENCE SHOWS THIS STATEMENT IS GROSSLY MISLEADING, AND REPRESENTS ONLY A SELECTIVE, EVEN CHERRY-PICKED READING OF THE AVAILABLE SCIENTIFIC INFORMATION, IN PARTICULAR THAT REGARDING CHRYSOTILE FIBERS AND DIFFERENTIATION OF FIBERS ET AL.

At this point, it bears repeating that the estimated figured of 107,000 annual global deaths attributed to asbestos and mentioned by the WHO paper, has never been substantiated, observed or recorded as publicly verified fact. It is therefore more accurate to state that this number - including the annual or cumulative historical aspect - is nothing more than a computer modeled extrapolated hypothesis and, when addressed specifically to the chrysotile form of asbestos is neither substantiated nor accurate.

EXTRAPOLATING A HYPOTHESIS

The estimate proposed and repeated by the WHO is based on data collected from a group of select European countries and extrapolated to the rest of the world. This approach does not take into account different fiber types, the divergences in structure and composition of the industry, how products differ in different geographies and markets, various effects of climate on fiber distribution and behavior, and past uncontrolled heavy exposures.

In essence it therefore says the opposite: that how and what and when Europe used various forms of asbestos are the same for everywhere in the world, and everyone, even though it is clear that vast portions of the world may have never used the dangerous amphibole forms of asbestos, or may never have manufactured or installed friable products, or been in environments where airborne fibers behave entirely different. This range of factors is ignored by the hypothesis makers. Instead the WHO tells us that one size fits all, and that the potential mortality extrapolations fit all. One could hardly imagine a less scientific, more biased approach.



WHAT THE SCIENCE SAYS

Because of the long latency period, the diseases appearing today are the results of exposures that were encountered 20 to 40 years ago.

And in fact, the rate of asbestos related diseases has started to decline. This is thanks to direct improvements in working conditions implemented from the 1970's and the prohibitions of amphiboles in the late 1980's. Proper information, good work practices and appropriate control measures – not a blind prohibition – have achieved the objectives of the WHA sanctioned, WHO program on need to adopt measures to eliminate and prevent asbestos-related diseases.

FURTHERMORE, MANY SCIENTIFIC STUDIES PUBLISHED IN THE LAST 25 YEARS HAVE SHOWN THAT THE RATES OF INDUSTRIAL DISEASES OF WORKERS IN THE ASBESTOS-CEMENT INDUSTRY – WHICH ACCOUNTS FOR MORE THAN 90% OF THE USE OF CHRYSOTILE IN THE WORLD TODAY – DO NOT EXCEED THE NATIONAL AVERAGE.

Moreover, the deaths estimate does not take into account the fact that exposure levels have dramatically decreased in the last decades as supported by the latest report published under the aegis of the WHO; (Concha-Barrientos M. et al. (2004) “Comparative Quantification of Health Risks: Global and Regional Burden of Disease Attributable to Selected Major Risk Factors”.

As well, in Ezzati M. Lopez AD, Rodgers A, Murray CJL, eds. Geneva: World Health Organization, chapter 21 pp. 1651-1801), the authors acknowledge that there is a great difference in risk between chrysotile asbestos and the amphibole varieties and that the risk from low exposure levels is undetectable. No real excess in lung cancer is expected from low exposure levels to chrysotile.

So, if exposure to chrysotile does not present a significant health risk, and if low exposure levels to chrysotile do not present excess levels of lung cancer, where do the annual death figures come from?

Indeed, according to a landmark study completed in 2000, Hodgson and Darnton estimated the same risks, differentiated by fiber types.

The results are self-explanatory:

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

It is worth noting and underlining that at present, chrysotile fibers are the sole form of asbestos in use today – and that it is limited to non-friable products.

It is therefore obvious that the WHO have grossly exaggerated the risks associated with exposure to the chrysotile form of asbestos.

In a recent scientific conference held in Birmingham, UK, May 1-4 (2016), some of the world's leading scientists in the field of asbestos research informed participants that chrysotile fiber exposure should not be held or understood to be responsible for historically high mesothelioma rates in U.K. and that although chrysotile was the predominant form of asbestos imported and used in the U.K., it was the amphiboles that were responsible.

This is a fundamental point as it scientifically demonstrates the extreme differences in potential harm to human health from exposure to different fiber types. To put it in layman's terms, even small amounts of exposure to amphiboles can be extremely dangerous whereas the effects of chrysotile, even in large amounts appear negligible. Of note, many scientific studies have also shown this form of lung cancer is principally due to amphiboles exposure.

So why, in the development of extrapolated hypotheses on the annual deaths attributable to asbestos does the WHO continue not to differentiate? Why does the WHO choose to ignore inconvenient science?

It is important to recall that on this specific subject, at the 95th session of the International Labor Organization (ILO), in June 2006, the representative from the United States of America asked the following question:

PREAMBULAR, PARAGRAPH 3

332. "The Government member of the United States asked if the figure of 100,000 deaths a year could be justified.⁽¹⁾

The response to this question to date lacks fundamental explanation, lacks scientific basis and in no way validates this suggested number of deaths. Furthermore, nowhere is it taken into account that there is a difference between the asbestos fiber types (amphiboles & serpentine), yet as shown above and in countless references, this difference exists.⁽²⁾

(1) (<http://www.ilo.org/public/english/standards/relm/ilc95/pdf/drafrep-css.pdf>)

(2) (Hodgson JT, Darnton A. The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. *Ann. Occup. Hyg.* 200, Dec.: 44(8):565-601).

EXTRAPOLATING A HYPOTHESIS EXPLAINED

The exact origin of the 100,000 deaths statement came from an Editorial, published in 2004 by Treasure (Dr. J. Peto, co-author) in the BMJ, where it is stated that in the developed world alone, 100,000 people alive now may or will die from it. The reference to asbestos includes all types of asbestos and that the people living at that time would eventually die. It is not a statement on chrysotile or annual deaths.

For the first time at the “Dresden Declaration of the Projection of Workers Against Asbestos Conference”, a presentation by Mr. J. Takala, a well-known anti-asbestos activist, used statistics from Finland, and mentioned the number of 100,000 deaths per year **worldwide**. So it was a self-proclaimed anti-asbestos activist, Mr. Takala who for the first time, took the idea of aggregated and cumulative potential deaths, and transformed them into annual deaths.

HOWEVER, IN HIS DEFENSE, MR. TAKALA ADDED – THAT IT IS ONLY AN EXTRAPOLATION ON HIS PART. “IN TOTAL, THERE COULD BE SOME 100,000 WORK-RELATED DEATHS CAUSED BY ASBESTOS. THESE FIGURES ARE NOT RECORDED CASES BUT ESTIMATES.”

Since this conference was held, the number of 100,000 deaths/year has been manipulated and repeated in the crusade of anti-asbestos activists around the world, seeking to promote fear and hysteria in support of a global ban of asbestos – including chrysotile. Inexplicably, it has also been used and promoted by the WHO.

ELIMINATION OF ASBESTOS-RELATED DISEASES

Few other natural resources have been the subject of more research than chrysotile asbestos. Nevertheless, in spite of all the scientific data accumulated on the health effects of chrysotile and fiber-type differentiation and, in spite of measures taken by the industry to dramatically improve the workplace including the direct input from workers and various labor organizations, a climate of uncertainty persists among the public. And a climate of fear – often promoted by the range of actors that make up the anti-asbestos lobby who above all want to avoid differentiation.

In truth, the facts-based story is much less interesting or dramatic. Chrysotile is not a devastating threat to the population, to the world, or to workers, and certainly nothing like the stories widely spread and alleged by anti-asbestos activists. The chrysotile world, through the years, has provided answers and argued with logic and common sense in response to many of these accusations. Rational responses and explanations may have been ignored by those who refuse to consider science, but the potential risk that this natural fiber may present has been addressed and is easily manageable by following standard, ILO approved industrial safe use procedures.

In support of this, over the last three decades there has been consistent published evidence that chrysotile can be used safely and under conditions that present no measurable risk to health. Many examples of safe use have been studied, noted, recorded and replicated on the factory, mine, regional and national level.

As stated above, and of relevance for many developing countries, the good news is that the practical implementation of the safe and controlled use of chrysotile remains a relatively simple and straightforward matter, and does not require overly sophisticated equipment.

Numerous scientific studies have been published in recent years that support the assertion that exposure to chrysotile that respects the occupational standard of (1 fiber/cc) is safe; and in particular, that the risk to health at this level of exposure is so low as to not be measurable.

ADDRESSING LEGACY ISSUES

From the early 20th century to the late 1960s, in areas of the world experiencing rapid economic growth, asbestos (different types of fiber) was used in hundreds of thousands of buildings and ships through “spraying”, a process that leaves the asbestos in a friable form that can easily be released into the air.

For at least the last thirty years, there has been a ban on all such processes. Rather, in contemporary applications, chrysotile is always encapsulated in another substance (cement or asphalt, or resin for example) that prevents it from being released into the air. These are known as non-friable products and they are achieved through a wet manufacturing process. Under these circumstances, fibers are encapsulated in the matrix and are not capable of becoming airborne. Nowhere in the world today are friable products made that could lead to fibers becoming airborne.

Also, in the past, research did not differentiate between forms of amphibole asbestos (amosite, crocidolite, tremolite) and chrysotile (serpentine type), whose biochemical molecular structure and risk are different. Today, this difference has been recognized throughout the world. It has been scientifically demonstrated that chrysotile is much less harmful to human health than amphiboles.

A retrospective review of several studies published in scientific journals suggests that there is no increased risk for human health associated with chrysotile at the current standard of (1 fiber/cc). In other words, to our knowledge, no study has successfully measured an increased risk below this standard.

The WHO document therefore does not rely on the most recently published scientific peer-reviewed analysis of evidence.

Among other studies, the following peer-reviewed references are particularly relevant:

Health risk of chrysotile revisited Crit Rev Toxicol, 2013: 43(2): 154-183

David Bernstein, Jacques Dunnigan, Thomas Hesterberg, Robert Brown, Juan Antonio Legaspi Velasco, Raul Barrera, John Hoskins, and Allen Gibbs.

ABSTRACT

THIS REVIEW PROVIDES A BASIS FOR SUBSTANTIATING BOTH KINETICALLY AND PATHOLOGICALLY THE DIFFERENCES BETWEEN CHRYSOTILE AND AMPHIBOLE ASBESTOS ...

And published evidence supporting a “practical threshold” level of exposure to occupational exposure to chrysotile asbestos below which no adverse health effects are observed. **(see Annex A)**

POLICY IMPLICATIONS

It is important to state clearly that all stakeholders fully support responsible approaches to eliminate asbestos-related diseases in the world.

As noted and documented, some activists within the WHO have decided the only possible way to implement a national strategy aimed at eliminating asbestos-related diseases is to advocate and claim that a total ban of all forms of asbestos is the current policy. This approach is both unreasonable and does not reflect the formal guidelines of the WHA as note above. Nor does it acknowledge the evidence from a broad spectrum of recently published scientific studies on chrysotile.

As for any product, substance or activity which may represent a potential health risk, it is logical to put in place programs and enforce legislation to ensure their safe and responsible use.

To ban a substance, product, or natural resource implies that research, evaluation and serious study has taken place; and that prior to making a decision, that the overwhelming body of scientific evidence and policy options concludes there is no other possible choice but a ban. Such a decision is generally taken as a last resort, when other available policy options are ineffective in the fact of a verified and dramatic threat.

Based on that logic, responsible policy formulation is required to take all factors into consideration – in order to be rendered mutually exclusive, collectively exhausted, and objective. In tis context, it is more than reasonable that chrysotile producing and consuming countries should be involved in the development and implementation of a safe action plan with respect to chrysotile. The WHO and the global cadres of anti-asbestos activists are not enough – when it comes to forming a fair, responsible and science-based policy.

Today, millions of workers are involved in international chrysotile industries. Taken together, these countries represent more than two-thirds of the total world population. For all parties of interest to be involved this means including and respecting the views of workers, their organizations, governments and industry.

It is important to accept that only through an inclusive stakeholder process can success will be achieved and a plan created that will he eliminate asbestos-diseases in the world. Why then has there been a refusal from WHO for this open and honest dialog and policy making process? **(see Annex B)**

Office of the
Director General
42-1484
27 AUG 2008

MEMORANDUM

From: Director, PHE To: DGO Date: 27 August 2008

Our ref: Attention: *Agree that was asked to take a no letter from the asbestos industry*

Your ref: Through: ADG/HSE *ADG/HSE*

Originator: A/Coordinator IHE Subject: PUBLIC HEALTH AND ASBESTOS *no issue of relations with WHO should be explored upward*

Recently several letters from asbestos industry interests addressed to DG have asked WHO to change its technical statements on chrysotile asbestos. All these letters use very similar arguments and even wording that seems to be part of a campaign. Furthermore, an NGO sponsored by the asbestos industry tried to establish official relations with WHO, and participants from other NGOs advancing asbestos interests have been attending the 60th and the 61st WHA.

The 58th WHA urged Member States to pay special attention to cancers for which avoidable exposure is a factor, particularly exposure to chemicals at the workplace and the environment (Resolution WHA 58.22). Asbestos is one of the most important occupational carcinogens causing about one third of the deaths from occupational cancer. Furthermore, the 60th WHA has requested the Secretariat to include in its activities "a global campaign for elimination of asbestos-related diseases - bearing in mind a differentiated approach to regulating its various forms - in line with the relevant international legal instruments and the latest evidence for effective interventions..." (Resolution WHA 60.26, annex, para 10). Several technical documents by the Secretariat stated that given the fact that there is no safe threshold for exposure to asbestos, that exposure is difficult to control and that there are safer substitutes, the most effective measure to eliminate asbestos-related diseases is to stop the use of all forms of asbestos. In accordance with the direction given by the Health Assembly, WHO's assistance for elimination of asbestos-related diseases will be particularly targeted at those Member States that still use chrysotile asbestos (see attached WHO documents).

Some organizations claim that Resolution WHA 60.26 has in fact endorsed the so called "safe" or "controlled" use of chrysotile asbestos. There are attempts to undermine the statements in the WHO official documents regarding chrysotile asbestos, based on arguments about the comparative hazard of chrysotile versus the other forms of asbestos, which ignore the inherent hazards of the chrysotile form. Furthermore, some argue that the use of chrysotile asbestos in water pipes helps reaching the MDGs, while WHO has stated that though the presence of asbestos in asbestos-cement water pipes presents no danger to the health of consumers, the fact remains that there is danger during the manufacture of these pipes. Therefore existing pipes with asbestos do not need to be removed, but new water pipes should not contain asbestos (see Press Release WHO/17 from 25.02.1994).

The next Conference of the Parties (COP) of the Rotterdam Convention in October 2008 will again consider the inclusion of chrysotile asbestos under the Convention requirements for information exchange and informed consent of importing countries. In the lead up to COP, industry and other interested parties, such as countries that export or use large amounts of chrysotile may approach WHO including IARC) with respect to its statements on asbestos. Significant producers and users include the Russian Federation, Canada, Zimbabwe, Kazakhstan, Ukraine, Colombia, India, Sri Lanka, and China.

We suggest that no action be taken on letters and communication from the asbestos industry and that measures should be taken to avoid any relations between WHO and organizations or individuals which are related to asbestos interests.

[Signature]
Dr Maria Elena

(see Annex B)

SERIOUS QUESTIONS THAT WHO MUST ANSWER TO-----

THE EMERGENCE OF SUBSTITUTES

Over the last few decades, non-asbestos fibrous materials, both man-made and those extracted from natural deposits, have been proposed and are presently used as substitutes for chrysotile. There are wide variations in competitiveness but on an economic basis, a proper approach must be taken in order to scientifically evaluate that such products are safer in less harmful than chrysotile for human health. In addition, the relative availability, technical performance, ease of handling and mixing, compatibility with other materials in composites, durability, etc. must be fully technically evaluated.

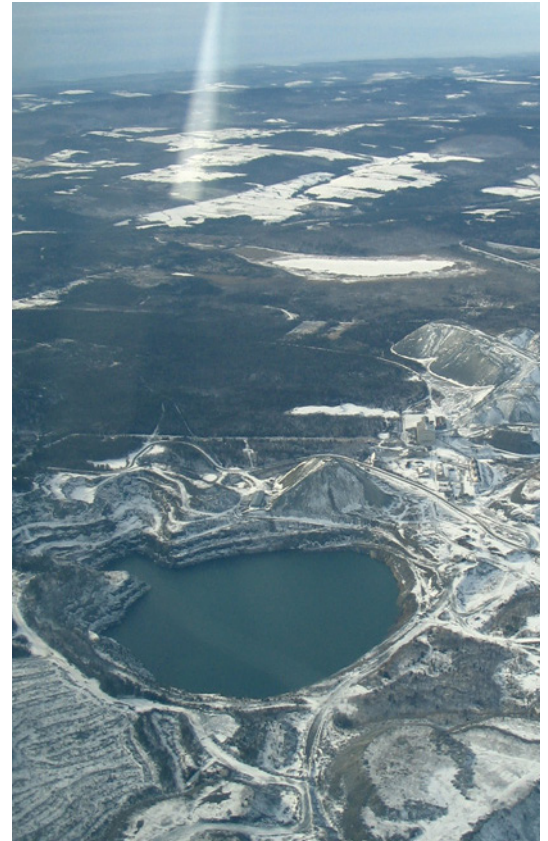
Compared with chrysotile, the evidence of biological activity of non-asbestos fibrous materials has only recently been reported. Except for a very limited number of materials (example: mineral wools), epidemiological scrutiny has yet to be undertaken in order to substantiate possible human health hazards. On the other hand, recently published results from cell, tissue and animal experimentation indicate that most fibrous materials of respirable size display some degree of biological activity. These results suggest that their widespread production and use should be governed by appropriate monitoring and control of dust exposure, especially for materials which are long and thin, and which display long “in vivo” durability (biopersistence). Thus, the safety issues applied for the use of chrysotile should apply to all fibrous substitutes.

THE CONCEPT OF CONTROLLED USE

In the area of occupational health, and specifically with regard to the use of chrysotile, regulatory agencies in all countries have the responsibility to set workplace exposure limits that 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.

Indeed, the latest scientific evidence published strongly supports the following views:

1. Chrysotile is significantly less hazardous than the amphibole forms of asbestos (e.g. crocidolite and amosite);
2. When properly controlled and used, chrysotile in its modern day high-density, non-friable applications do not present risks of any significance to the public and/or worker health.
3. Chrysotile under safe use control is not responsible for mesothelioma



CONTROLLED USE SHOULD APPLY TO ALL FIBROUS SUBSTITUTES

According to standard industrial safety protocols, controlled use applies to the following four areas: monitoring, dust controls, medical surveillance, and training and information.

MONITORING

Monitoring must be carried out by well-trained industrial hygienists, using recognized methods of sampling and counting.

Ideally, monitoring of the workplace should be done by hygienists, and employers and workers should be involved.

Measurements should be done on a regular basis, and the results should be reported to both the employers and the workers. This would ensure that corrective actions are taken when needed.

DUST CONTROL

Adequate and efficient dust controls (ventilation, use of wet methods, etc.) should be in place.

Proper functioning of dust controls should be constantly monitored.

MEDICAL SURVEILLANCE

Medical surveillance (MS) is a necessity. It should be a permanent and well-organized activity that includes a proactive commitment from industry and all respective stakeholders.

TRAINING AND INFORMATION

Every worker should receive adequate training on the safe handling and the best work practices.

All starting materials and finished products must be labelled with adequate warning signs. Information must be in all time a matter of concern.

QUESTIONS AND ANSWERS

In this section of the 2014 WHO report, the WHO wanted to provide the impression to its readers that asbestos fibers (including the chrysotile form) is one of the most dangerous substances known to man.

Like anti-asbestos campaigners and activists, the WHO does not make a case for higher and tighter controls on chrysotile safe use but instead makes a radical policy-jump to argue for excluding chrysotile from the world. It therefore presents a range of unclear questions and biased self-designed answers to justify the story of imminent and grave harm from all forms of asbestos, especially chrysotile – notwithstanding the scientific facts presented herein.

It is precisely the tools of partial information, and misrepresentation, supported by unsubstantiated scientific theories and extrapolated hypothesis that this paper objects to – and the so-called WHO question and answers use to exaggerate the perceived threat and create a monumental scare campaign. This scare campaign is not theoretical: the WHO uses these tactics with the specific intent of influencing – through fear and inference – the decision making and policy formulation process of many countries, who in certain cases are dependent on the WHO for other non-related developmental programs.

Unfortunately, this situation does not allow competent authorities of different countries to comprehensively evaluate the policy choices in front of them – or to fully consider formal WHA guidelines on options for eliminating asbestos related diseases. Absent this objective panorama, on both policy and science, it is challenging for authorities to make important decisions for the future of their respective country's.

INDEED, IT IS DIFFICULT NOT TO CONCLUDE THAT THROUGH THE EFFORTS OF THIS PAPER/ NON PAPER, THE WHO PAPER APPEARS TO BE PROMOTING AND ADVANCING THE INTERESTS OF THE WELL-ORGANIZED AND FUNDED ANTI-ASBESTOS LOBBY. IT IS REGRETTABLE THAT THROUGH THESE ACTIONS, THE WHO DOES NOT SUPPORT OR ADVANCE ITS OWN NEUTRALITY.

Below are some questions and answers that pertaining to chrysotile as produced and commercialized today.

THE USE AND MISUSE OF STATISTICS

Peer-reviewed scientific data, modern safe-use practices, and the science on chrysotile specifically should be of prime interest for any competent authority seeking to promote public health and adopt responsible regulation.

THE THRESHOLD VALUE CONCEPT

Scientific studies refer to an exposure level below which there is no measurable health risk. This is a common scientific norm. Much of the activist/ anti-asbestos lobby refuses to consider this, as if no matter what the level of exposure or fiber type, the risk is the same. This view and position is contrary to widely recognized practices, science and international norms.

As several epidemiological studies show, including those already indicated, workers subject to chrysotile exposure at approximately 1 fiber/cc are not at a measurable risk. By following this standard, chrysotile does not pose an unacceptable risk for health.

Numerous published studies in the last thirty years indicate that the controlled use of chrysotile at ~ 1f/cc, does not increase the risk of excess morbidity and mortality.

RISK MANAGEMENT IN THE WORKPLACE

Risks are present in every working environment (chemical, heavy industry, construction, etc.) and are a feature of our modern world.

In numerous countries, the chrysotile industry together with workers and their unions, have implemented major technical changes for the furtherance of worker and public health. In so doing, they have revolutionized work processes including production and extraction practices to the benefit of all.

It would be both unwise and inaccurate to confound and conflate the unacceptable working conditions of the past, with current standards and practices. Modern technology and improved conditions in workplace have vastly improved the sector and to not recognizing these improvements is an example of obstinacy and bad faith.



ARGUMENTS PRESENTED BY ANTI-ASBESTOS ACTIVISTS AND LOBBY

Since the WHO report gives credence to the views and biases promoted by the anti-asbestos lobby, the following section sheds light and transparency on a series of common – if questionable – questions and answers

CLAIMS OF THE ANTI-ASBESTOS LOBBY

Asbestos is a carcinogen and the only way to protect the health of workers and the population is to ban its use entirely.

IN FACT, THE REALITY IS ...

As the International Labor Organization (ILO) recognized in 1986, and many countries afterwards, regulations on asbestos use must be based on science, not on perceptions or business interests. Some five hundred other products and industrial processes are recognized as carcinogens, but this does not mean that we must prohibit or ban their use.

CLAIMS OF THE ANTI-ASBESTOS LOBBY

Asbestos is widely known, and its effects on health have been documented since the beginning of the 20th century.

IN FACT, THE REALITY IS ...

The effects of various asbestos fibers on health are well known and documented. There is scientific consensus on the fact that fibers in the amphibole group are from 100 to 500 times more harmful to health than chrysotile, particularly for mesothelioma. Chrysotile is not responsible for mesothelioma.

Studies show that:

- a) Asbestos, including both amphiboles and chrysotile, are known carcinogens for human beings and there is no known exposure threshold.
- b) Chrysotile is associated with asbestosis, lung cancer and mesothelioma, based on the level of exposure.
- c) The risk of developing lung cancer or mesothelioma applies to users of products containing asbestos and to the population exposed to it.

The confusion purposely maintained by opponents to the safe use of chrysotile is due to purposeful confusion of the two families of fibers, without differentiating, despite the fact that the type, geological source, use and effects on health are radically different.

Concerning the very existence of a threshold, the scientific community recognizes that this threshold does exist. Cohorts representing tens of thousands of workers exposed only to chrysotile at levels of concentration lower than 1fibre/cm³ have been studied and clearly do not show an in-ordinate increase in disease in relation to the general population.

Industrial diseases related to the use of asbestos are therefore the result of **excessive** and **prolonged** exposure to amphiboles. This is primarily why the ILO indicated that the issue is an issue of **industrial hygiene** not a public health concern.

CLAIMS OF THE ANTI-ASBESTOS LOBBY

The International Agency for Research on Cancer (IARC – WHO) has recognized asbestos as a type 1 carcinogen. Its use must therefore be prohibited.

IN FACT, THE REALITY IS ...

Because all types of asbestos were used incorrectly in the past, chrysotile and amphiboles have been classified as type 1 carcinogens/proven carcinogenic agents), such as cadmium, chromium, nickel compounds, silica, the sun's rays, vinyl chloride, alcoholic beverages, salted fish, tobacco smoke, saw dust, the manufacture and repair of shoes, the manufacture of furniture and cabinets, iron and steel foundries and the rubber industry. The International Agency for Research on Cancer (IARC) classification identifies a substance's **hazard**, not the risk. Consequently, a substance classified in group 1 does not mean its use must be prohibited, only that it should be properly controlled (as chrysotile is used today).

CLAIMS OF THE ANTI-ASBESTOS LOBBY

All types of asbestos are dangerous – this is why distinction between chrysotile and amphiboles are purely semantic.

IN FACT, THE REALITY IS ...

That “chrysotile” asbestos and “amphiboles” are regulated differently is nothing new. This two-pronged approach exists in Convention 162 on the safe use of asbestos issued by the International Labor Organization. Since “asbestos” is a trade name rather than a technical term, it is appropriate that regulation take into account the main differences between fiber types.

Furthermore, many studies and an international consensus proves that chrysotile fiber (white asbestos) is different from other forms. This certainty is the foundation of the ILO convention, as well as of the regulations of most countries in the world. Two significant scientific events recently confirmed this fact: a group of scientists mandated by the EPA unanimously agreed that available studies on epidemiology indicate that the carcinogenic potential of amphibole fibers was one hundred times (100 x) higher than that for chrysotile fibers. Another important study on the biological persistence of chrysotile in the lung has shown, taking into account the scientific literature to date, that the report on this study provides solid new data that clearly confirm the difference between chrysotile and amphiboles.

These fundamental differences are recognized by the group of experts brought together by the World Health Organization, who recommended, based on scientific data, that chrysotile asbestos should be regulated to 1 fiber per cubic centimeter, while amphiboles should be prohibited. Numerous countries have adopted the principle for using chrysotile safely with an allowable exposure level in accordance with this recommendation.

CLAIMS OF THE ANTI-ASBESTOS LOBBY

Controlled use of chrysotile does not take the latency period for diseases associated with asbestos into account, which may take up to 30 years to appear.

IN FACT, THE REALITY IS ...

The laws and regulations adopted by many governments take into account the scientific reality that stipulates that for the general population, the health risk from high-density products with chrysotile content (asbestos cement, brakes, plastics, treated fabrics) are undetectable.

As for workers, laws and regulations require users of chrysotile to implement controls that allow its use while protecting the health and bodily integrity of workers. By introducing a prohibition on amphiboles, the authorities caused on elimination of future cases of mesothelioma, which is imperceptible until after the latency period for those who have been exposed.

CLAIMS OF THE ANTI-ASBESTOS LOBBY

Preventive measures are not sufficient to protect the health of workers. Workers are often not trained to apply these measures or to implement safe methods, In the 1970s, the NIOSH (United States) claimed that only a ban on asbestos could ensure complete protection from the carcinogenic effects of this product.

IN FACT, THE REALITY IS ...

Prevention methods that were suggested in the late 1970s are integrated into the Code of Practice on asbestos by the ILO in 1984. They provided proof of their applicability and effectiveness.

All construction materials contain elements that are likely to be harmful to the health of workers if used incorrectly. One must make sure appropriate equipment is used properly under recommended work methods, regardless of the materials used. This is true for all substances that can be harmful.

The position of the National Institute for Occupational Safety and Health (NIOSH) in the United States has evolved somewhat since the early 1970s when the effects of various types of asbestos on health were not as well documented. During public hearings by the U.S. Congress in July 2001, the directors of the Occupational Safety and Health Administration (OSHA) and NIOS expressed their opposition to banning chrysotile asbestos and stated the current legislation was the most appropriate to protect workers and provide a safe working environment.

CLAIMS OF THE ANTI-ASBESTOS LOBBY

Safe use is a utopian view for the following reasons:

- a) The general population is exposed to a hazard due to products that contain asbestos.
- b) Applying control measures is impossible.

We must follow the example of the United States* and the European Union, which have prohibited asbestos.

IN FACT, THE REALITY IS ...

Products manufactured in the last 20 years or so, encapsulate the fibers in solid materials, such as cement or resin – rendering them non-friable. The conditions described by supporters of a ban have not existed for decades with respect to chrysotile. The conditions they describe as health hazards do however apply to substitute fibers or products and to many other dangerous products that unfortunately remain unregulated and under-studied.

This claim is based on impressions and a false reality that no longer exists. Numerous countries have adopted the principle of controlled use. Use of chrysotile is in practice relatively easy to control given the limited number of sources of supply. Why would this be easier to accomplish with potentially harmful substitute fibers, when they have not always been shown to be safer than chrysotile or too often not subject to regulation to protect the health of workers?

*Contrary to the claims of anti-asbestos advocates, the United States have repeated their confidence in the principle of safe use of chrysotile.

Today, those who handle chrysotile work in an environment where the measured concentration is less than 1 fiber/cm³ have recognized that at this level, the health risk is undetectable.

CLAIMS OF THE ANTI-ASBESTOS LOBBY

The entire world is moving towards a ban. We must follow this trend.

International experts support the ban. As proof, INSERM (France) claims that chrysotile cannot be dissociated as a cause of pleural mesothelioma.

IN FACT, THE REALITY IS ...

Those who oppose the use of chrysotile are focused on selecting and highlighting on that information which matches their views and objectives, but that do not represent the most recent opinions of experts or international organizations. What about experts and evidence that does not object to controlled use of chrysotile and are supporting a safe and responsible approach?

CLAIMS OF THE ANTI-ASBESTOS LOBBY

Asbestos is primarily used in countries that have no regulations about its use, and it is handled by untrained workers who have no access to medical examinations.

IN FACT, THE REALITY IS ...

Many countries ratified Convention 162 on the Safe Use of Asbestos and incorporated its principles into their national law or regulations. Since 1986, chrysotile stakeholders have organized seminars and training workshops in many countries to ensure that users of chrysotile fiber have the necessary expertise and equipment to handle it safely. Producers and users countries are full aware of all aspects regarding the safe use of chrysotile.

CLAIMS OF THE ANTI-ASBESTOS LOBBY

The global trend is clearly leaning in favor of banning all types of asbestos.

IN FACT, THE REALITY IS ...

Speaking of a European campaign as an international trend is a pure exaggeration. The countries of the European Union have adopted the principle of banning chrysotile effective in 2005, and are strongly encouraging other countries to do the same to create an opening for substitute fibers. Outside of Europe, only a few countries are following suit. Curiously, these are countries that export substitute fibers. The real trend is that the majority of countries have adopted the principles of controlled use in their legislation on chrysotile. In addition countries within the European Union such as Germany have repeatedly sought and successfully obtained waivers for the use of chrysotile citing the reality that it is and can be used safely.

CLAIMS OF THE ANTI-ASBESTOS LOBBY

Countries are responsible for taking all necessary measures to protect the health of workers and the population. The prohibition of asbestos is one of these imperative measures.

IN FACT, THE REALITY IS ...

By adopting laws and regulations that support the controlled use of chrysotile, regulatory authorities in the various countries demonstrate their concern for protecting the health and safety of workers, while ensuring that durable, inexpensive and completely safe products are available to consumers.

Moreover, these legislations and regulations are compatible with the principles put forth by the ILO and WHO, as decided by the WHA.

The determination of many governments that have based their decisions on science rather than succumbing to industrial and political pressures must be noted.

Obviously, it will be necessary and urgent to extend the measures adopted for chrysotile to all respirable industrial fibers whose risks (biological persistence) are very often greater than to chrysotile. For all these fibers there must be true concerns about protecting the health of workers.

FURTHER SCIENTIFIC REFERENCES

It may come as a surprise to some readers, but the commercially created terms “asbestos” includes different varieties: specifically chrysotile which forms its own group or family, and a larger group known as amphiboles (amosite, crocidolite, etc). Unfortunately, many lump together all varieties under the word “asbestos”: “asbestos is asbestos, period”.

Science shows that these varieties are different not only regarding their physical structures and chemical compositions, but most importantly in the health risk they present.

A meta-analysis was published by Hodgson JT and Darnton A (2000). *The Quantitative Risks of Mesothelioma and Lung Cancer in Relation to Asbestos*. Ann. Occup. Hyg. 44(8): 565-601. Their conclusions are compelling:

FIBER SPECIFIC RISKS:

	CHRYSOTILE	AMOSITE	CROCIDOLITE
For lung cancer:	1	10	50
For mesothelioma:	1	100	500

Among other studies, a group of scientists produced the following position:

ON SAFETY IN THE USE OF CHRYSOTILE ASBESTOS

It must be recognized that in the past, the uncontrolled use of all commercial types of asbestos has left a sad legacy of disease and death as a result of carelessness in handling these minerals, especially in the workplace and sometimes in the general population.

Yet, over the last 50 years, world production has not declined. The world production in 1960 was around 2M tonnes, and still approximatively to 2M tonnes. However, while world production in the early 1960s included all major forms (chrysotile, amosite and crocidolite), the production

of the amphibole varieties (crocidolite and amosite) has ceased since 1987 and 1992 respectively.

Unfortunately, because of procrastination by certain governments in implementing regulation preventing the use of amphiboles, the remaining amphiboles inventories were allowed to be used in some factories up to the mid-90s. In addition, due to large usage in past years of amphiboles by some countries, a significant background level of amphibole asbestos remains. Due to the characteristic long latency associated with the onset of asbestos-related cancer, especially mesothelioma, a high incidence of this particular cancer of the pleura may be foreseen in those industries for the next two or three decades.

The carcinogenic potency of amphibole asbestos has been established both epidemiologically and toxicologically, leading to it being no longer used in commerce anywhere today. In 1989, a group of international experts convened by the WHO in Oxford (UK) recommended that these asbestos varieties should be prohibited immediately, and that the use of chrysotile should be controlled and regulated at a permissible exposure limit of 1 fiber/ml in the workplace.

Today, the remaining practical concern is whether chrysotile can be produced and used safely, and if indeed this regulation carries a reasonable assurance that workers are adequately protected. Based upon current science, the short answer to this question is that in absence of amphiboles, the use of chrysotile at current permissible exposure limits in the workplace carries no epidemiologically and clinically detectable increase in risk. Indeed, a number of recent scientific studies published in peer-reviewed journals have come to this conclusion (see Annex). From these published studies, it can be seen that safety in the use of chrysotile is not a simple wish, but a reality. The ILO has issued a “Code of Practices” entitled “*Safety in the Use of Asbestos*”, which addresses all pertinent issues regarding the modern and responsible use of asbestos.

Erosion of surface deposits over millennia means that chrysotile is a ubiquitous component of the particulate matter in the air. The WHO (1986) estimates the background exposure to chrysotile as between 0.01 and 0.001 fiber per milliliter of air. The risk to health from this background exposure is, for all practical purposes, non-existent. Industrial and other exposure

at the high end of this range has been labelled “acceptable” by the Ontario Royal Commission on Asbestos (ORCA), “*not significant*” by the WHO, and “... *further control not justified*” by the Royal Society in London (UK).

CONCLUSIONS

The latest scientific evidence published strongly supports the following views:

1. Chrysotile is significantly less hazardous than the amphibole forms of asbestos (e.g. crocidolite and amosite);
2. When properly controlled and used, chrysotile asbestos in its modern day high-density applications does not present risks of any significance to public and/or worker health.

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TECHNICAL SUMMARY OF WHO

The 2014 WHO paper addresses question on production and the use of chrysotile in the world today.

The relevance of global production and usage statistics is questionable at best in the context of strictly defined human health issues. Rather than focus on health, the section is little more than a list of the countries that are using and producing chrysotile – which could be interpreted as a subtle “name and shame” tactic. In this there is nothing new and the readers learn little about health issues related to chrysotile.

It is worth bringing to the attention of readers page 16, the beginning of the second paragraph which states: “Although asbestos has not been banned in the USA...”. Indeed this sentence is not very explicit but the background is highly relevant because it has not been banned.

In the USA, the use of chrysotile has been attacked for many years by anti-asbestos lobbyists and various activists (including within EPA) wherein they exerted great effort and enormous pressure to pass a full and total legislative ban. That effort did not succeed.

The United States Fifth Circuit Court of Appeal was very clear in its ruling when it refused this request based on a meticulous examination of science, facts and the realities associated with risk – not conspiracies and conflated narratives.

It is therefore not accurate to state that countries are going in an “anti-asbestos direction”; just the opposite, countries that

conduct proper and thorough evaluations, absent the politics of fear and exaggeration more often than not come to the conclusion that safe use can and does work. In this respect, the authors of the WHO report are presenting incomplete and misleading information when they indicate that all countries supporting WHO crusade.

Today, a large number of countries use chrysotile fibers and chrysotile containing products and it is their firm intention to continue to do it in a safe and responsible manner. The in fact WHO is fully aware of this fact – notwithstanding their editorial decision to leave out this fact.

This raises an important question regarding why this reality is not mentioned in the 2014 paper? One explanation could be that the WHO is poorly informed – another likely explanation is that the exclusion is a reflection of bad faith and institutional bias. Anti-asbestos lobbies and the lucrative litigation businesses that work in close cooperation with the WHO are fully aware of the current situation in USA and elsewhere, where bans are regularly followed by costly litigation.

One should advise the WHO that this form of “ironic false steps” cannot improve their credibility. Every year, domestic surveys (Virta, among others) are prepared and published and world production tables are well explained. The anti-asbestos lobby’s attempt to advance and coerce the passage of a world-wide chrysotile ban has badly failed – because it is not supported by facts on the ground and the realities of objective science.

On October 18, 1991, the US Court of Appeal for the Fifth Circuit struck down the crusade and EPA against the use of asbestos in the USA. The Court concluded that the EPA failed to master substantial evidence to support this abusive request. The same happened to anti-asbestos activists in the Supreme Court in India. In January this year petitioners to the Supreme Court in India tried to use the same so-called science to ban asbestos. The Judges asked to see the evidence to support their petition but nothing was found. The petitioners were charged with perjury and fined with a short custodial sentence.

It should be a matter of concern to notice the WHO in its in house paper in full transparency did not make a single reference to such important facts. The institutional refusal to explain or cite this open information is very important and raises profound questions about the WHO's objectivity and fairness on the topic. .

The use of the chrysotile form of asbestos in the USA today well understood and is confined to production processes where worker exposure and risk is essentially eliminated and nil.

The current status of asbestos products in the United States following of EPA's asbestos ban rule appears below:

BANNED

- Corrugated paper
- Commercial paper
- Flooring felt
- Rollboard
- Specialty paper
- New use of asbestos

AUTHORIZED

- Corrugated asbestos cement sheet
- Flat asbestos cement sheet
- Vinyl asbestos floor tile
- Asbestos cement piped
- Asbestos cement shingles
- Friction materials
- Brake linings
- Clutch facings
- Disc brake pads
- Asbestos clothing
- Automatic transmission components
- Roofing felt
- Roof coatings
- Non-roof coatings
- Mill board
- Pipeline wrap
- Acetylene cylinder filler
- Asbestos diaphragms
- High-grade electrical paper
- Packings
- Sealant tape
- Brake blocks
- Missile liners
- Arc chutes
- Automatic transmission components
- Roofing felt
- Non-roof coatings
- Battery separators
- Reinforced plastic
- Textile products

In its approach to the subject of world consumption, the WHO is trying hard to denounce countries that are using chrysotile. The WHO indicates (with no scientific solid base) that all forms of asbestos are carcinogenic to humans, and may cause mesothelioma and cancer of the lung, larynx and ovary. Asbestos exposure is also responsible for other diseases, such as asbestosis (fibrosis of the lungs), pleural plaques, thickening and effusions.

Currently, about 125 million people in the world are exposed to asbestos at the workplace. According to the most recent WHO estimates, more than 107,000 people die each year from asbestos-related lung cancer, mesothelioma and asbestosis resulting from exposure at work. One in every three deaths from occupational cancer is estimated to be caused by asbestos. In addition, it is estimated that several thousand deaths annually can be attributed to exposure to asbestos in the home. Such false statements must be declared unacceptable. They categorically do not reflect real or observed facts nor do they reflect the conclusions of recent peer-reviewed scientific evaluation.

WHAT IS THE WHO DOING FOR THE ELIMINATION OF ASBESTOS-RELATED DISEASES?

Other than supporting the vested interests of anti-asbestos lobby, the WHO does very little. They refuse to hear or evaluate any science that disagrees with their position and serially ignore evidence from country-level “safe use” protocols that are accepted and recognized as effective tools to reduce risk to worker health – even though those protocols are in absolute conformity with formal WHA policy and relevant WHO resolutions.

Specifically, the World Health Assembly (WHA) Resolution 58.22 on cancer prevention urges Member States to pay special attention to cancers for which avoidable exposure is a factor, including exposure to chemicals at the workplace. With Resolution 60.26, the WHA requested the WHO to carry out a global campaign for the elimination of asbestos-related diseases... *“bearing in mind a differential approach to regulating its various forms – in line with the relevant international legal instruments and the latest evidence for effective interventions”*.

Eliminating asbestos-related diseases is particularly targeted at countries that still use chrysotile asbestos, in addition to assistance in relation to exposures arising from historical use of all forms of asbestos.

Now is the time for the WHO authorities to listen to other voices and stakeholders and not just the vested interests of the anti-asbestos lobby. This means taking the obligation to be objective seriously and to stop controversial presentations at international seminars including crusade-style interviews posted on You Tube that once again confirm the WHO’s support for questionable lobbies. Too often, data used by the WHO and its activists are misleading, based on unsubstantiated evidence oriented towards promotion of a world ban of chrysotile, and fail to address a safe and responsible approach to protect the health of workers and the general population.

NO BIAS – ONLY SCIENCE

One cannot escape the disturbing reality of these numbers. A ban of chrysotile fibers is not part of the WHO mandate.

Finally, there are also other statistics that need to be carefully evaluated. For instance, in order to support one's particular views, one can quote only parts of the available numbers. An example was used by some ideologues who carefully selected parts of a document prepared for the World Health Organization (WHO Assembly Resolution 58.22 on cancer prevention and control, 2005), citing a WHO publication (Concha-Barrientos et al., 2004), stating that: *“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”*.

Unfortunately, few people would bother to scrutinize the validity and completeness of such numbers. But a careful examination of the Concha-Barrientos report shows that the above statements and statistics are grossly misleading, in that they represent only the selected parts of the report, which suited the intention of some ideologues. Here are the facts and the complete conclusions of the Concha-Barrientos report.

First, the Concha-Barrientos et al. report acknowledges that there is a difference in risk between chrysotile and the amphibole varieties of asbestos. 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”*.

But the authors also add: *“In 20 studies of over 100,000 asbestos workers, the standardized mortality rate ranged from 1.04 for chrysotile workers to 4.9 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”*.

This is a good example of how WHO activists consciously edit and in this case manipulate science in what can only be termed bad faith.

HEALTH EFFECTS

This part of the WHO paper is best understood as a well-articulated attempt to sow confusion and cause unnecessary panic among workers and the general population. Specifically, the conscious decision to ignore bio-chemically proven fiber differentiation – and instead imply that exposure to any form of asbestos airborne fibers is the same as exposure to materials containing chrysotile fiber is false and irresponsible.

Keeping silent on the facts of differentiation that exist between fiber types and the differences that exist in their chemical composition and associative risk levels in respect to public health must be considered a gross error.

Countless studies and reports have presented the same conclusion: that differentiation between serpentine and amphibole fibers means differentiation in health risk. This WHO report (2014) in this regard is misleading and confusing and must be re-written bearing in mind this fundamental scientific concept; to do any less is to prejudice the report from its initiation. The WHO must have the courage to present the real facts.

All chrysotile products manufactured today are high density (non-friable) and in this category of products, where fibers are chemically locked into place, there is no scientific evidence that such these conditions will inevitably cause asbestos-related diseases. Such assurance resides only in the anti-lobby propaganda. Occupational exposure of chrysotile workers today is categorically different from the past and in particular, the description presented by the WHO in its paper. In this

regard, in the chrysotile history, there is the past and there is the present with regard to working conditions. Risk from exposure is dose related. At a level of 1f/cc of chrysotile exposure, that risk is so low that it has become almost technically non-measurable and numerous scientific published studies confirm this fact with peer reviewed data.

WHY IS ASBESTOS A PROBLEM?

In real terms, asbestos is a historical problem and spending vast sums on banning chrysotile will not save more lives today. It will only benefit large groups of vested interest who profit from such the advancement of sham science, fear and wide-spread deception.

A summary list of vested interests is worth considering:

- Asbestos claim's lawyers and their friends.
- Producers of alternative products and fibers who rely on bans to sell their products (as a rule more expensive and less durable).
- Asbestos removal contractors.
- Certain doctors and scientists that accept to be sponsored by anti-asbestos lobbyists and litigation businesses.
- Insurance companies who rely on charging extra high premiums.
- Political parties and others who receive large support from activists and asbestos litigation firms (especially in the USA).

13th INTERNATIONAL CONFERENCE OF THE INTERNATIONAL MESOTHELIOMA INTEREST GROUP

TOWARDS PERSONALIZED CARE

iMig 2016

MAY 1-4, 2016
BIRMINGHAM, UK

As mentioned at the beginning of this document, a conference was recently held in Birmingham, UK.

During the conference, clear statements have been made regarding the relationship between chrysotile and mesothelioma. It has been clearly stated that the mesothelioma observed was a consequence of heavy uncontrolled use of amphibole fibers exposure in the past till 1980.

It has also been indicated that the correlation must be made between mesothelioma and the use of amphiboles and **not chrysotile**. Dr. Peto informed the delegates that the science does not permit to say plainly anything and forever.

Scientists make presumption based on evidence and he added that in this case he was obliged to declare that chrysotile should not be seen as the cause of enhanced mesothelioma rates in the UK. The statement, based on rigorous scientific research and evidence, caused visible frustration from a strong presence of anti asbestos activists and lobbyists.

Many recent scientific publications are of great interest on this matter. However all of them have been ignored or dismissed by the WHO and anti-asbestos activists and the anti-asbestos lobby (updated June 2016) among others.
(see Annex C)

HAZARD IS NOT RISK

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

HAZARD characterization is an essential, but insufficient component of risk assessment, which also comprises exposure data over time and estimation on the likely **RISK under actual conditions of use**.

Because the IARC classification refers only to “**hazard identification**”, and does not refer to “risk assessment”, because the components of dose under actual conditions are absent.

The IARC classification is not meant and should not be used as the only “risk management” instrument for eventual regulatory action.

1. The IARC monograph, on which the WHO is based, has been the subject of misrepresentation of its real meaning: the IARC classification of human carcinogens is about hazard, not actual risk.

ON THE TRUE MEANING OF IARC CLASSIFICATION OF “HUMAN CARCINOGENS”

The present classification of “human carcinogens” by the International Agency for Research on Cancer (IARC) includes some agents, mixtures and activities, divided into five main groups, as shown here.

Group 1	Carcinogenic to humans
Group 2A	Probably carcinogenic
Group 2B	Possibly carcinogenic
Group 3	Not classifiable
Group 4	Probably not carcinogenic

<http://monographs.iarc.fr/ENG/Classification/index.php>

GROUP 1: THE AGENT IS CARCINOGENIC TO HUMANS.

This category is used when there is sufficient evidence of carcinogenicity in humans. Exceptionally, an agent may be placed in this category when evidence of carcinogenicity in humans is less than sufficient but there is sufficient evidence of carcinogenicity in experimental animals and strong evidence in exposed humans that the agent acts through a relevant mechanism of carcinogenicity.

GROUP 2:

This category includes agents for which, at one extreme, the degree of evidence of carcinogenicity in humans is almost sufficient, as well as those for which, at the other extreme, there are no human data but for which there is evidence of carcinogenicity in experimental animals. Agents are assigned to either Group 2A (probably carcinogenic to humans) or Group 2B (possibly carcinogenic to humans) on the basis of epidemiological and experimental evidence of carcinogenicity and mechanistic and other relevant data.

GROUP 3: THE AGENT IS NOT CLASSIFIABLE AS TO ITS CARCINOGENICITY TO HUMANS:

This category is used most commonly for agents for which the evidence of carcinogenicity is inadequate in humans and inadequate or limited in experimental animals.

GROUP 4: THE AGENT IS PROBABLY NOT CARCINOGENIC TO HUMANS.

This category is used for agents for which there is evidence suggesting lack of carcinogenicity in humans and in experimental animals. In some instances, agents but evidence suggesting lack of carcinogenicity in experimental animals, consistently and strongly supported by a broad range of mechanistic and other relevant data may be classified in this group.

THE CASE OF ASBESTOS

Presently, the IARC has classified asbestos (all fiber types, without distinction between chrysotile and the amphiboles) in «GROUP 1» (carcinogenic to human). Currently, some 108 other agents, mixtures and activities are included in this group. No one will permit to itself to propose to stop the use of all these 108 substances for health reason.

In the Preamble* to the IARC Monographs amended January 2006, a cancer “hazard” is an agent that is capable of causing cancer under some circumstances, while a cancer “risk” is an estimate of the carcinogenic effects expected from exposure to a cancer hazard. The Monographs are an exercise in evaluating cancer hazards, despite the historical presence of the word “risks” in the title.

The distinction between hazard and risk is important, and the Monographs identify cancer hazards even when risks are very low at current exposure levels, because new uses or unforeseen exposures could engender risks that are significantly higher.

*<http://monographs.iarc.fr/ENG/Preamble/index.php>

The question then is whether the inclusion of an agent in the Group 1 of the IARC classification implies that it must be banned.

The answer is obviously « NO ». Who would think of banning oestrogen therapy, the contraceptive pill, boot and shoe manufacture and cabinet making, diesel motors, etc. simply because they are in the Group 1 classification of potential carcinogens of the IARC?

As mentioned above, the IARC classification is about hazards, not risk. Risk is the probability that a person will experience an adverse health effect **if exposed to a hazard under actual conditions of exposure**. For example, we know that the sun’s radiations are a hazard, that is, these rays have the potential to cause harm, but the risk will be minimal or non-existent or very high depending on the dose, on the actual conditions of exposure.

The same remark applies to chrysotile asbestos. There are plenty of studies published in peer-reviewed journals showing that at low exposure conditions, chrysotile can be used without demonstrable health effects. **(see Annex D)**

RICH AND POOR COUNTRIES – WHERE THE WHO STANDS ON IT

In today's often-distressed world, up to 1.5 billion humans do not have access to potable water and 2.5 billion have no access to basic hygienic infrastructure. In South-East Asia and in Africa alone, diarrhea is responsible for no less than 8.5% and 7.7% of deaths (UNDP Report 2006). This translates into more than 8 million people who die each year including approximately 2 million children. This is no longer poverty, rather it is profound misery.

In this world where we use thousands of products and substances, some of which can be dangerous to human health or potentially fatal or carcinogenic, instead of demanding a categorical ban, the world has learned to use them by following standardized procedures and measures. Countless such examples exist, including in Europe, where silica is both dangerous and carcinogenic yet used daily and safely.

Today, countries that use chrysotile fiber represent (as previously noted) 2/3's of humanity. Many of these countries are in various stages of development and can be classified as emerging countries, who are making great efforts to provide their populations with a better quality of life. To do so, they need high quality, durable products which are affordable and well adapted to local conditions, which include the imperative of job creation.

Prior to banning products that contain chrysotile, a much more expedient approach is to support the responsible and safe use of chrysotile with an emphasis on fostering good work practices. Chrysotile fiber and chrysotile-containing products are uniquely appropriate to the housing and infrastructure needs of developing countries because of their safety, durability, quality and ease of use.

Collectively, it is important to take stock of the responsibility to ensure that the interests of developing or low income countries are taken into account, before advancing the goals of special interest groups, such as the anti-asbestos lobby. This means respecting the right of all countries and in particular lower income ones to make sovereign and responsible decisions without harassment for or contempt by wealthy nations and activists.

REPLACEMENT PRODUCTS OR FIBERS TO CHRYSOTILE

The safety of replacement fibers and products is critical subject that the WHO has chosen not to address in the 2014 paper. It is mentioned that many national governments, regional bodies and international organizations have identified alternatives and substitutes for the use of asbestos. But where are the serious scientific published studies on this regard?

In 2005, a WHO/IARC workshop highlighted a worrying lack of research and data pertaining to many substitute products and recommended that serious scientific studies should rapidly be done for robust evaluation, before presenting acceptable recommendation regarding their use. What happened to that recommendation and why is the WHO not concerned about the potential and very real health effects of substitute fibers? Why ignore these risks?

International Convention 162 on the Safe Use of Chrysotile is very clear on this matter. When asbestos has to be replaced, it has to be by a substance, a product or fibers that are scientifically proven being safer and less harmful than asbestos. Nevertheless, the WHO keep silence on this matter on its publication.



NEW EUROPEAN UNION DIRECTIVE – A MATTER OF CONCERN

It is understood that the WHO is responsible to guide or identify better work practices and/or implementing worker safety protection measures.

An important amendment has been adopted to Directive 2009/148/EC of the European Parliament and Council, on the Protection of workers from the risks related to asbestos exposure. This is in regard to the omission of Recital (2) from Directive 2003/18/EEC after the codification procedure, which established the obligation of implementing a preventive approach in the use of asbestos substitutes. This new directive came into force in 27 countries of the European Union in January 2010.

It is important to note that before the final amendment of the above-mentioned directive, the European Economic Social Committee give its opinion. The UE institution which gathers the representative of workers and employers of the 28 Member States note that some important part were ignored. They particularly expressed their concerns about the removal of the recital 2 of the directors where it was indicated *“the importance of a preventive approach, with regard to substitute fibers for asbestos”*. This concern was obviated by the Commission that went to approve Directive 2009/148/EC maintaining the removal of recital 2.

It is remarkable that activists and WHO keep silence on it.

In spite of the many interventions before the European Commission, countries are still waiting for a logical answer to such a change. Also despite the objections raised by the workers and employers of 28 countries of the European Union and within European Economic and Social Committee (EESC) regarding this important part has finally disappeared from the legislative text.

Recital (2) from Directive 2003/18/EEC, underscores the importance of a preventive approach to the use of asbestos substitutes. This approach is particularly important that workers who are exposed to substitute fibers and products nowadays mostly in Europe, should be aware that they could pose health problems. This judicious and necessary warning suddenly disappeared from Directive 2009/148/EEC. The WHO is certainly not, or cannot afford to be insensitive, to the potential risks of exposure to substitute products and fibers to which are exposed millions of people worldwide.

It seems reasonable to ask at the same time that all alternative products and fibers carrying a potential health risk should be controlled as strictly as possible. It seems more than logical that any industrial fibers which do have a potential health risk should be subject to the same restrictions and regulations as for chrysotile.

Considering all the efforts deployed in the name of health, and the approach taken by the European Union and WHO activists regarding other potential replacement fibers and products, for example crystalline silica (the EU permits users to conclude a voluntary accord instead of regulating) one must understand that there are two measures: it is evidently incoherent.

The European Commission Directive 1999/77/EEC, dated 26 July 1999, addressed this issue. On many occasions, the fact that replacement fibers and products have not always been adequately evaluated as to their potential dangerousness must be preoccupying. International organizations such as International Agency for Research on Cancer (IARC) and the Scientific Committee on Toxicity, Ecotoxicity and the Environment (SCTEE), have also requested this scientific evaluation.

A genuine comparative risk assessment is necessary and requested. This is a fundamental requirement which will help make clear and honest decisions on the use of chrysotile and replacement fibers or products whose risk must also be well and scientifically documented.



ANOTHER IMPORTANT SUBJECT FORGOTTEN BY THE WHO

1. IS PRODUCED BY LOW ENERGY-CONSUMING TECHNOLOGY

Manufacture on some products involve high energy consumption, which means a drain on finite resources (hydroelectricity, fossil fuels, etc.), some of which are non-renewable.

Compared to products coming from the petrochemical or metallurgical industry, asbestos-cement products consume much less energy, in fact, the largest proportion of energy consumption goes into the production of cement.

2. HAS A LONG USEFUL SERVICE LIFE

Short product life means you have to replace more often, create more waste, and needs more energy consumption, etc.

This resistance of asbestos-cement products to corrosion, to ultra-violet rays, to rot etc. is remarkable and unique. In fact, few other products have such a guaranteed long service life.

3. IS MADE FROM SIMPLE STARTING MATERIALS

Production of final products may involve complex mixtures of synthetic starting materials, which may be harmful by themselves (ex. PVC made from vinyl chloride monomers – a known carcinogen), and present a risk not only for plant workers, but for general population well.

Composition of high density asbestos-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 those required for the controlled manufacture of asbestos-cement products.

4. PRESENTS A RELATIVELY LOW RISK DURING ITS MANUFACTURE

Use of countless products may cause environmental damage to fauna, flora, rivers, lakes, the sea, underground waters may (does) occur, following explosions, radioactive leakage, acid precipitations, etc., as a result of systems malfunction, equipment failure, human error, carelessness or other unforeseen reasons (ex.: Bhopal, Chernobyl, Minamata).

With controlled plant operations, asbestos-cement manufacturing presents a far lesser risk to the environment, compared to many other product manufacturing technologies based on synthetic chemistry or metallurgy.

5. PRESENTS A RELATIVELY LOW RISK WHEN IN USE

Some products may be consumed by fire, releasing large clouds of toxic and/or corrosive gases.

Whereas many combustible construction materials may, in case of fire, release clouds of gases and/or fumes highly toxic to man and to the environment, asbestos-cement products are by definition resistant to heat and fire; in fact, they may actually prevent or minimize the spread of conflagration.

6. PRESENTS A RELATIVELY LOW RISK WHEN STORED OR TRANSPORTED, PRIOR TO OR AFTER USE

Transportation and storage of some raw materials or finished products prior to their use, or when discarded after use (ex.: corrosive liquids, hazardous chemicals, storage of discarded PCBs, spent lead batteries, old tire piles, etc. may pose a hazard to both the environment or the general population.

Transportation and handling of asbestos-cement products does require appropriate care, by efficient and recognized practices are simple and straightforward. The safe transportation and storage of some other products is far more complex, and mishaps can (and do) occur. Compare the risk of environmental damage of a tanker full of crude oil or other petrochemicals to the risk of a cargo of asbestos-cement products.

7. CONSTITUTES A RELATIVELY LOW RISK AT FINAL DISPOSAL SITE

Some products present a high degree of hazard to the environment (soil and/or water contamination) if not securely contained in specially designed and tightly supervised disposal sites.

Safe disposal of many modern products and waste has become an environmental and economic nightmare, often requiring especially designed and costly disposal sites, which must be monitored constantly to prevent leakage of contaminating substances into the environment. Waste management is often so complex and costly that “easier” solutions are often found... Chrysotile-cement waste disposal is inexpensive, simple and recognized practices are well known. They are made of naturally occurring material which returns to the environment after use.

CHRYBOTILE CEMENT VERSUS OTHER BUILDING MATERIALS

Over the past 15 years chrysotile cement products have demonstrated some advantages over other building materials such as asbestos-free fiber-cement products and metal roofing.

Apart from its remarkable properties chrysotile cement has a better price and durability – its service life is 50 to 60 years and over. Besides, the use of local Portland cement helps save currency funds and labor costs. Their production is less energy-consuming. Taking into account all these factors we can conclude that chrysotile cement products have obvious environmental advantages over competitive products.

N	CHARACTERISTICS	ASBESTOS-CEMENT SHEETS	CORRUGATED GALVANIZED IRON SHEETS	ALUMINUM SHEETS
1	Service life	50(min) Stainless	10-15	N/A
2	Maintenance	Not required	Every 3-5 years	Not required
3	Fire danger	Inhibitor	Tend to distort and melt	Tend to distort and melt
4	Heat insulation	Good	Weak	Weak
5	Sound insulation	Good	Weak	Weak
6	Wind and rain-generated noise absorption	Good (reduces the noise)	Weak	Weak
7	Energy consumption required for manufacturing (k.W.h/sq.meter)	1.0	36.6	33.0
8	Potential work pressure	Intensive	Low	Low
9	Aerodynamic resistance after installation	Good	Weak	Weak
10	Weathering	No	Rusting of drilled holes and cracked zinc coating	Oxidation of the surface
11	Bimetallic reaction	No	No	When contacting with concrete and other materials if wet
12	Condensation	Low, having no effect on the sheet	High, leading to corrosion	High, affecting the sheet
13	Protective coating	Not required	Not required	Necessary to prevent a direct contact with cement, lime, iron, copper, etc.
14	Storage	May be kept in the open in working areas	Must be kept indoors to prevent weathering	Must be kept indoors to prevent weathering
15	Efficiency of coverage	Almost 50% higher than that of corrugated galvanized iron sheets and aluminum sheets	The area coverage is only 67% of that with asbestos-cement sheets	The area coverage is only 67% of that with asbestos-cement sheets
16	Cost	Low	High	High

MINERAL COMMODITY PROFILES – ASBESTOS

Energy required by the U.S. asbestos mining industry in 1973 averaged an equivalent to 10.6 million British thermal units (MBtu) per metric ton of cleaned and graded chrysotile product. The survey covered all producers in Arizona, California, North Carolina and Vermont and included estimates of energy content for various fuels used in mining and milling. On a tonnage basis energy used was equivalent to 1,500 kilowatthours per ton (kWh/t) of usable fiber (table 26). Estimated costs for producing asbestos were \$3.5 million or \$25.86 per ton calculated in 1983 dollars. The ease of mining the Coalinga deposit kept the average U.S. energy requirements low (Clifton, 1985). In 1976 energy requirements at a large Canadian mine and mill were higher at 2,725 to 3,110 kWh/t than those of the average U.S. producer requirements (Clifton, 1985: table 27).

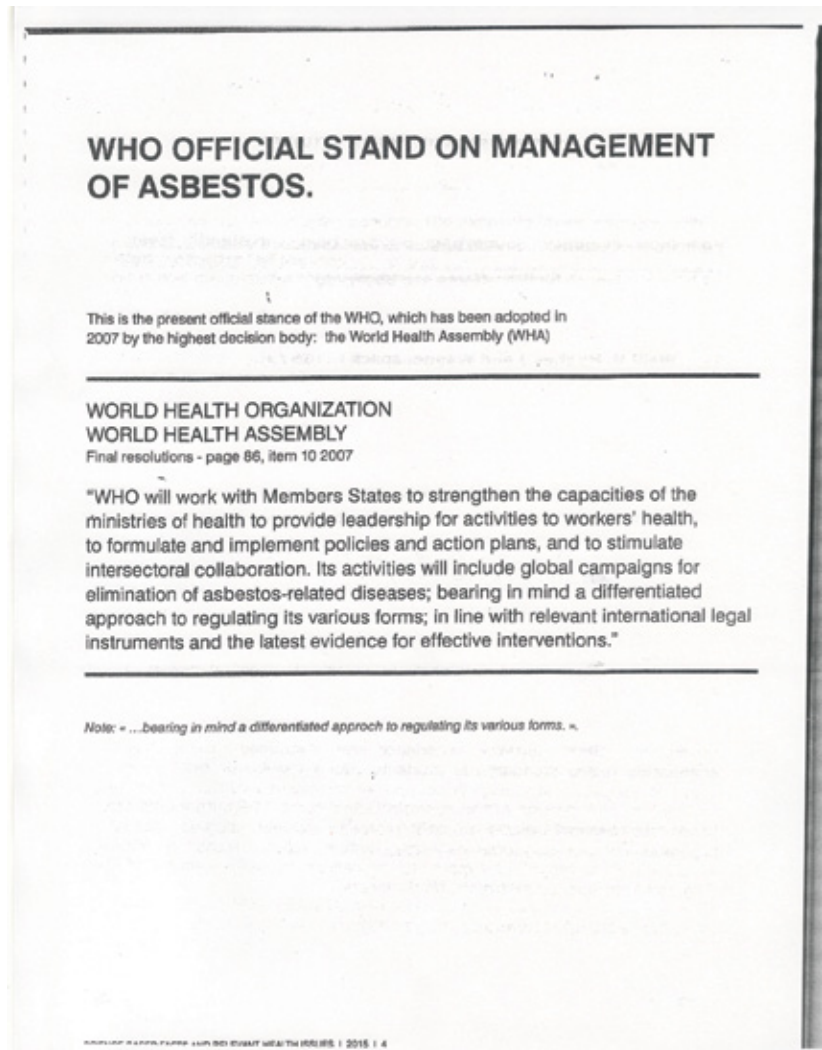
A study of the energy content of three cladding materials was done in the United Kingdom in 1979 for the Asbestos Information Centre. The study started at the mines for the raw materials and ended at the building sites. All relevant and significant energy expenditures and credits were calculated. The study determined that 16.42 kilowatthours (kWh) of energy was required to manufacture a square meter of corrugated asbestos cement sheet, 68.92 kWh was required for a square meter of corrugated aluminum sheeting, and 123.5 kWh was required for a square meter of plastic coated corrugated sheet steel (Schatzberger, 1979).

PARAMETERS	AC TRUSSED ROOFING SYSTEMS	SELF-SUPPORTED ROOFING SYSTEMS
Collateral load on roof	Flexibility in hanging electrical fixtures, ducts, sprinklers from the roof.	Only light weight fixtures can be hung and from predetermined positions only.
Fire protection	Can be provided by column encasement or by intumescent painting of the structure.	Fire protection cannot be easily provided.
Roof geometry	Irregular shaped buildings with high bay & low bay roofs can be easily provided	Providing buildings of irregular shapes & difference in heights is difficult, cumbersome & uneconomical.
Eco friendly	Manufacturing process of steel utilizes high quantum of coal or energy and process leads to slag generation.	Eco friendly process and technology. Uses fly ash (35%) and leads to no pollution. Consumes low energy compared to metal roofing.
Raw materials	100% imported leads to loss in foreign exchange and employment creation.	10% imports.
Supporting R.C. structures	Light R.C. works are required & entire building can be completed in 90 days' time.	Heavy R.C. beams & foundations are required for supporting these roofs, and, require almost 3-4 months for R.C. works itself.
Clear span of building	Clear spans of up to 80 m can be provided.	Maximum clear spans of up to 35 m only can be provided.
Provision of roof accessories	Wide range of roof accessories such as turbo vents, ridge vents, roof monitors, skylights, roof platforms can be provided.	Only limited range of roof accessories such as turbo vents & skylights can be provided.

CONCLUSION

The purpose of this study was to review the points raised by the WHO's 2014 paper titled, "Chrysotile Asbestos" and in so doing, draw attention to the many factual errors, policy contradictions, and distortion of facts therein, so that an objective and facts-based understanding of the issues can be established.

It is hoped that this has been achieved by pointing out the need to establish a fully comprehensive scientific frame of reference for any science-based assertions; for the need to reflect the importance of fiber differentiation in determining results of asbestos exposure and relevant policy implications; for the immediate clarification of contradictions and inconsistencies in certain statistical predictions related to potential deaths attributed to asbestos exposure; and the need to avoid unilateral policy advocacy that ignores the interests of all stakeholders in pursuit of an advocacy agenda of the few.



ANNEX A

Weill H, Hughes J and Waggenspack C. (1979).

Influence of dose and fiber type on respiratory malignancy risk in asbestos-cement manufacturing. *Am Rev Respir Dis.* 120(2): 345-354.

An investigation of 5,45 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 MPPCF. years (corresponding to approximately 15 fibers/ml x 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".

Thomas HF, Benjamin IT, Elwood PC and Sweetnam PM. (1982).

Further follow-up study of workers from an asbestos-cement factory. *Br J Indus Med.* 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 standardized mortality ration (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 is not an excess risk in terms of total mortality, all cancer mortality, cancers of the lung and bronchus, or gastrointestinal cancers".

Berry G and Newhouse ML. (1983).

Mortality of workers manufacturing friction materials using asbestos. *Br J Indus Med.* 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 fiber-ml x years. The authors state: *"The experience of this factory over a 40-year period showed that chrysotile asbestos was processed with no detectable excess mortality"*.

Gardner MJ, Winter PD, Pannett B and Powell CA. (1986).

Follow-up study of workers manufacturing chrysotile asbestos-cement products. *Br J Indus Med.* 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 fiber concentrations below 1f/ml, although higher levels had probably occurred in certain areas of the asbestos-cement factory.

Ohlson CG and Hogstedt C. (1985).
Lung cancer among asbestos-cement workers. A Swedish cohort study and a review. *Br J Indus Med.* 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 fibers/ml.years.

Newhouse ML and Sullivan KR. (1989).
A mortality study of workers manufacturing friction materials: 1941-86. *Br J Indus Med.* 46(3): 176-179.

The study referred to in the preceding slide has been extended by seven years. The authors confirm that there was no excess of deaths from lung cancer or other asbestos related cancers, or from chronic respiratory disease. After 1950, hygienic control was progressively improved at this factory, and from 1970, levels of asbestos have not exceeded 0.5-1.0f/ml. The authors conclude: *“It is concluded that with good environmental control, chrysotile asbestos may be used in manufacture without causing excess mortality”*.

Liddell FDK, McDonald JC and McDonald A. (1997).
A mortality study of workers manufacturing friction materials: 1941-86. *Ann Occup Hyg.* 41: 13-35.

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 Quebec. The cohort, which was established in 1966, comprises some 11,000 workers born between 1891-1920 and has been followed ever since. The authors have updated their study several times, with a total of 9,780 men traced into 1992. Results from exposures below 3000 mpcf x years, roughly equivalent to 900 fibers/ml x years – or, say 45 fibers/ml for 20 years – lead the authors to conclude: *“Thus it is concluded from the point of view of mortality that exposures in this industry to less than 300 mpcf.years has been essentially innocuous”*.

Paustenbach DJ, Finley BL, Lu ET, Brorby GP and Sheehan PJ (2004).
Environmental and occupational health hazards associated with the presence of asbestos in brake linings and pads (1900 to present): A “state-of-the-art review”. *J Toxicol Environ Health. Part B7:* 33-110.

This publication 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, demonstrate 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.

Yarborough CM. (2006).
Chrysotile as a cause of mesothelioma: An assessment based on epidemiology. *Critical Reviews in Toxicology.* 36: 165-187.

This is an extensive review of the epidemiological cohort studies undertaken to evaluate the extent of the evidence related to exposure to free chrysotile fibers, with particular attention to confounding by other fiber types, job exposure concentrations, and consistency of findings. This review of 71 asbestos exposed cohorts to free asbestos fibers does not support the hypothesis that chrysotile, uncontaminated by amphibolic substances, causes mesothelioma.

Carel R. Olsson AC, Zaridze D, Szeszenia-Dabrowska N, Rudnai P, Lowowska J, Fabianova E, Cassidy A, Mates D, Bencko V, Foretova L, Janout V, Fevotte J, Fletcher T, Mannetje A, Brennan P, Boffetta P. (2007).
International Agency for Research on Cancer, Lyon, France. Occupational exposure to asbestos and man-made vitreous fibers and risk of lung cancer: a multicenter case-control study in Europe. *Occup Environ Med.* Aug: 64(8): 502-8
Epub 2006 Oct 19
<http://oem.bmj.com/cgi/content/full/64/8/502>

The multi-center case-control study was carried out in six regions of Eastern and Central Europe and in the U.K. Comparison of odds ratios for asbestos exposure 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 U.K. is increased following exposure to asbestos. The authors conclude: *"In this large community-based study occupational exposure to asbestos. And MMVF does not appear to contribute to the lung cancer burden in men in Central and Eastern Europe. In contrast, in the U.K. the authors found an increased risk of lung cancer following exposure to asbestos. Differences in fiber type and circumstances of exposure may explain these results"*.

Mangold C, Clark K, Madl A and Paustenbach D. (2006).

An exposure study of bystanders and workers during the installation and removal of asbestos gaskets and packing. *J Occup Environ Health*. 3: 87-98.

In response to concerns raised in a report to the US Navy in 1977 on exposure to asbestos associated to gasket work, a series of studies was performed from 1982 to 1991 to evaluate the airborne concentrations of chrysotile asbestos associated with replacing gaskets and packing materials. The results indicated that the 8-hour time-weighted (TWA) average concentrations were between 0.01 to 0.03 fiber/cc.

White N, Nelson G and Murray J. (2008).

South African experience with asbestos related environmental mesothelioma: Is asbestos fiber type important? *Regul Toxicol and Pharmacol* 42: S92-S96.

South Africa, like Australia, represents a very particular situation in the history of the use of asbestos. These countries have historically been the major producers of amphiboles (crocidolite and amosite), and South Africa also produced amosite and chrysotile. In both these countries, the

number of mesothelioma cases has been much higher than anywhere else in the world. The authors have indicated that 23% of cases in South Africa were found in persons never employed in mining, but were found associated with living in neighborhoods close to amphibole mining facilities, thus associated with "environmental" exposure. However, there were no cases of mesothelioma associated with exposure to chrysotile. The authors conclude: *"No cases of mesothelioma were associated with South Africa chrysotile. Consequently, in the vast majority of cases of mesothelioma, environmental exposure to asbestos occurred in the North Cape province, in proximity to mines, mills and dumps where crocidolite was processed. Crocidolite appears more mesotheliomagenic than amosite, and chrysotile has not been implicated in the disease. This is true for both occupationally and environmentally exposed individuals"*.

Sichletidis L, Chloros D, Spyrtatos D, Haidich AD, Fourkiotou I, Kakoura M, Patakas D. (2008).

Mortality from occupational exposure to relatively pure chrysotile: A 39-year study. *Respiration*. 78: 63-68. Published Online: October 9, 2008
<http://content.karger.com/ProdukteDB/produkte>.

An investigation covering a span of almost 40 years on the mortality rate among workers exposed to relatively pure chrysotile in an asbestos-cement factory that opened in 1968 in Greece. The factory used approximately 2,000 tonnes of chrysotile annually until 2005. Fiber concentration was measured regularly, and was always below permissible levels. Date and cause of death were recorded among all active and retired workers. No case of mesothelioma was reported. Overall mortality rate was significantly lower than that of the Greek general population. Conclusions of the authors: *"Occupational exposure to relatively pure chrysotile within permissible levels was not associated with a significant increase to lung cancer or with mesothelioma"*.

ANNEX B

Office of the
Director General
42-1184
27 AUG 2008

MEMORANDUM

From: Director, PHE **To:** DGO **Date:** 27 August 2008
Our ref: **Attention:** *Agree not to allow to take a no letter from the asbestos industry*
Your ref: **Through:** ADG/HSE *ADG/HSE* **Subject:** PUBLIC HEALTH AND ASBESTOS *no issue of relation with asbestos should be explored upward*
Originator: A/Coordinator IHE *[Signature]*

Recently several letters from asbestos industry interests addressed to DG have asked WHO to change its technical statements on chrysotile asbestos. All these letters use very similar arguments and even wording that seems to be part of a campaign. Furthermore, an NGO sponsored by the asbestos industry tried to establish official relations with WHO, and participants from other NGOs advancing asbestos interests have been attending the 60th and the 61st WHA.

The 58th WHA urged Member States to pay special attention to cancers for which avoidable exposure is a factor, particularly exposure to chemicals at the workplace and the environment (Resolution WHA 58.22). Asbestos is one of the most important occupational carcinogens causing about one third of the deaths from occupational cancer. Furthermore, the 60th WHA has requested the Secretariat to include in its activities "a global campaign for elimination of asbestos-related diseases - bearing in mind a differentiated approach to regulating its various forms - in line with the relevant international legal instruments and the latest evidence for effective interventions..." (Resolution WHA 60.26, annex, para 10). Several technical documents by the Secretariat stated that given the fact that there is no safe threshold for exposure to asbestos, that exposure is difficult to control and that there are safer substitutes, the most effective measure to eliminate asbestos-related diseases is to stop the use of all forms of asbestos. In accordance with the direction given by the Health Assembly, WHO's assistance for elimination of asbestos-related diseases will be particularly targeted at those Member States that still use chrysotile asbestos (see attached WHO documents).

Some organizations claim that Resolution WHA 60.26 has in fact endorsed the so called "safe" or "controlled" use of chrysotile asbestos. There are attempts to undermine the statements in the WHO official documents regarding chrysotile asbestos, based on arguments about the comparative hazard of chrysotile versus the other forms of asbestos, which ignore the inherent hazards of the chrysotile form. Furthermore, some argue that the use of chrysotile asbestos in water pipes helps reaching the MDGs, while WHO has stated that though the presence of asbestos in asbestos-cement water pipes presents no danger to the health of consumers, the fact remains that there is danger during the manufacture of these pipes. Therefore existing pipes with asbestos do not need to be removed, but new water pipes should not contain asbestos (see Press Release WHO/17 from 25.02.1994).

The next Conference of the Parties (COP) of the Rotterdam Convention in October 2008 will again consider the inclusion of chrysotile asbestos under the Convention requirements for information exchange and informed consent of importing countries. In the lead up to COP, industry and other interested parties, such as countries that export or use large amounts of chrysotile may approach WHO including IARC) with respect to its statements on asbestos. Significant producers and users include the Russian Federation, Canada, Zimbabwe, Kazakhstan, Ukraine, Colombia, India, Sri Lanka, and China.

We suggest that no action be taken on letters and communication from the asbestos industry and that measures should be taken to avoid any relations between WHO and organizations or individuals which are related to asbestos interests.

[Signature]
Dr Maria Bena

(see Annex B)

Serious questions that WHO must answer to-----

ANNEX C

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Mat 10, 2016. Published by group bmj.com

Pleural mesothelioma and lung cancer risks in relation to occupational history and asbestos lung burden

Claire Gilhan, Christine Rake, Garry Burdett, Andrew G. Nicholson, Leslie Davison, Angelo Franchini, James Carpenter, Johh Hodgson, Andrew Darnton and Julian Peto

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Doi 10.1136/oemed-2015-103074

Updated information and services can be found at:

<http://oem.bmj.com/content/73/5/290>

CONCLUSION

Our results confirm the major contribution of amosite to UK mesothelioma incidence and the substantial contribution of non-occupational asbestos exposure, particularly in women.

Airborne asbestos exposures associated with the installation and removal of roofing products

Lotter, J.T. et al.

J Occup Environ (2016) Vol 13, Issue 8, 121-131
DOI:10.1080/15459624.2016.1183010

The findings indicate that short-term and full-shift exposures from the use of asbestos-containing roofing products were typically well below applicable occupational exposure limits. Additionally, the cumulative exposures associated with roofing work would be well below published chrysotile no-observed-adverse-effect-levels (NOAELs) for asbestos-related diseases.

The WHO document hardly recognizes the vast difference in risk between chrysotile and the amphiboles varieties. The following references from peer-reviewed scientific publications should have also received full consideration by WHO but have not been considered.

DIFFERENCE OF PATHOGENIC POTENTIAL ACCORDING TO FIBER TYPES

a) Evidence from morbidity and mortality studies in persons exposed to chrysotile exclusively

Wagner JC, Newhouse ML, Corrin B, Rossiter CE and Griffiths DM. (1988).

Correlation between fiber 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 last harmful form of asbestos in every respect and that more emphasis should be laid on the different biological effects of amphiboles and serpentine asbestos fiber”.

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 unlikelihood of link of mesothelioma with chrysotile exposure. Epidemiological studies from USA (Weiss, McDonald and Fry, Dement) from Britain (Newhouse, Thomas, Acheson) are analyzed, and lung burden studies (Pooley, Wagner, Jones. A.D. McDonald) are also pointed to.

Hughes JM, Weill H and Hammad YY. (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 MJ and Powell CA. (1986).

Mortality of asbestos cement workers using almost exclusively chrysotile fiber. *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 fibers containing mainly chrysotile but also amphiboles, crocidolite and amosite”.

Berry G and Newhouse ML. (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 HF, Benjamin IT, Elwood PC and Sweetnam PM. (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 AD and Fry J. (1982).

Mesothelioma and fiber 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 fiber types were used.

Acheson ED, Gardner MJ, Pippard EC and Grime LP. (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 AD and McDonald JC. (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 Quebec 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 Quebec (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) Evidence from mineral analysis of lung content

Wagner JC, Newhouse ML, Corrin B, Rossiter CER and Griffiths DM. (1988).

Correlation between fiber 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 JC, Moncrieff CB, Coles R, Griffiths DM and Munday DE. (1986).

Correlation between fiber 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 JSP, Roberts GH Pooley FD, Clark NJ, Smith PG, Owen WG, Wagner JC, Berry G and Pollock DJ. (1980).

The pathology and mineral content of lungs in cases of mesothelioma in the United Kingdom in 1976. *Biological Effects of Mineral Fibers, 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 AD. (1980).

Mineral fiber content of lung in mesothelial tumors. – Preliminary report. *Biological effects of Mineral Fibers*, 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 fibers 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 JC, Berry G and Pooley FD. (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 fiber were found in the lungs of 12 persons who had died of mesothelioma than in the lungs of controls without mesothelioma.

Wagner JC, Pooley FD, Berry G Seal RME, Munday DE, Morgan J and Clark NJ. (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 amount of chrysotile in referred pneumoconiosis patients.

Gylseth B, Mowe G and Wannag A. (1983).

Fiber 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 fibers was 0%-9% whereas the corresponding proportion for the amphiboles was 76% and 99%.

Rowlands N, Gibbs GW and McDonald AD. (1982).

Asbestos fibers 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 Quebec 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 lung while chrysotile was dissolved.

McDonald AD, McDonald JC and Pooley FD (1982).

Mineral fiber content of lung mesothelial tumors 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 AR, Jones JSP, Pooley FD, Griffiths DM and Wagner JC. (1989).

Non-occupational malignant mesotheliomas. *Non-occupational Exposure to Mineral Fibers*, 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 and in a previous series of mesotheliomas in which the majority has had direct exposure to asbestos. These findings confirm those of previous studies indicating the 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 fibers in lung tissue among asbestos cement workers.

A study which showing which showing different kinetics for amphibole and chrysotile fibers in human lung tissue. Amphibole fiber 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 fibers that are retained (amphiboles), than with the ones being cleared (largely chrysotile)".

ANNEX D

IARC 100C ASBESTOS – LIST OF STUDIES THAT WERE NOT INCLUDED IN THE IARC EVALUATION

EPIDEMIOLOGY STUDIES:

Roggli et al. (2002a):

Roggli et al. (2002) examined the association of the development of mesothelioma to contaminating tremolite fibers present in chrysotile dust and talc. The authors examined 312 cases of mesothelioma, for which fiber burden analyses of lung parenchyma had been performed by means of scanning electron microscopy. The amount of tremolite, non-commercial amphiboles, talc and chrysotile was determined. Of the 312 cases, 166 had tremolite with 81 of these above background levels. Fibrous talc was identified in 193 cases with a strong correlation to the tremolite content ($P < 0.0001$). Chrysotile was identified in only 32 cases, but still correlated strongly with the tremolite content ($P < 0.0001$). Non-commercial amphibole fibers (tremolite, actinolite and/or anthophyllite) were the only fiber types found above background in 14 cases. The authors concluded that tremolite in lung tissue samples from mesothelioma victims derived from both talc and chrysotile and that tremolite accounts for a considerable fraction of the excess fiber burden in end-users of asbestos products.

Roggli et al. (2002b):

Butnor et al. (2002) examined the relationship of malignant mesothelioma to occupational exposure to asbestos in 1445 cases (confirmed histologically and immunohistochemically) with known exposure histories. Fiber burden analyses were performed in 268 of the cases. Asbestos body counts were determined by light microscopy, and asbestos fiber content and type were assessed using scanning electron microscopy and energy dispersive X-ray analysis. The predominant manner of asbestos exposure was described by 23 categories with 94% in 19 of the categories which included 6 occupational categories: pipefitter, boilermaker, machinist, electrician, maintenance worker and sheet metal worker, as well as one para-occupational (household contact) exposure category.

The authors concluded that the vast majority of MMs occurring in the USA today can be placed into a limited number of exposure categories which include 12 industries and six occupations with known asbestos exposure. The remainder of the cases was predominately household contacts of asbestos workers, with neighborhood exposures rarely contributing to MM. Commercial amphiboles were found in excess amounts in all 19 categories, non-commercial amphiboles were found in excess amounts in a smaller percentage of cases, and cases in which the level of chrysotile exceeded background were infrequent.

Hodgson et al. (2005):

In a more recent analysis, Hodgson et al. (2005) modelled the expected burden of mesothelioma mortality in Great Britain based upon male mesothelioma deaths from 1968 to 2001 as a function of the rise and fall of asbestos exposure during the 20th century taking account of the difference between fiber types. Two models were fit to the data and the predicted exposure patterns compared with the actual exposure patterns based on imports of amosite and crocidolite. The authors state that chrysotile had zero weight in both (sic) models. Thus, the mesothelioma occurring in Great Britain since 1920 was explained by a combination of amosite and crocidolite reversing the earlier explanation of this as due to chrysotile (Peto et al., 1999). It is noteworthy that Peto who was first author of the 1999 publication is also a co-author of the Hodgson et al. (2005) publication which reverses the conclusion of the 1999 paper. Weill et al. (2004) have recently examined the temporal pattern and change in trend of mesothelioma incidence in the United States since 1973. They concluded that mesothelioma risk was prominently influenced by exposure to amphibole asbestos (crocidolite and amosite) which reached its peak usage in the 1960s and thereafter declined. The known latency period for the development of this tumor provides biological plausibility for the recent decline in mesothelioma incidence in the USA.

Yarborough (2006):

Yarborough (2006) reviewed all available epidemiological studies to determine if chrysotile was a cause of mesothelioma. This review was prompted by the long-standing debate over the potential contribution of chrysotile to mesothelioma risk. Yarborough undertook an extensive review of the epidemiological cohort studies in order 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. A total of 71 asbestos cohorts exposed to free asbestos fibers were reviewed. The authors concluded that the study “does not support the hypothesis that chrysotile, uncontaminated by amphibole substances, cause mesothelioma”.

Carel et al. (2006):

Carel et al. (2006), a study led by the International Agency for Research on Cancer (IARC), examined the risk of lung cancer following occupational exposure to asbestos and man-made vitreous fibers in a multicenter case-control study in Europe. Two regions were studied in this program, six Central and Eastern European countries and the UK, during the period 1998-2002. Comprehensive occupational and socio-demographic information was collected from 2205 newly diagnosed male lung cancer cases and 2305 frequency matched controls. Adjustment was made in the odds ratios (OR: The odds ratio is a relative measure of risk, telling us how much more likely it is that someone who is exposed to the factor under study will develop the outcome as compared to someone who is not exposed; an OR of 1 or less indicates no effect. Even if the OR is greater than 1, if the lower bound of the 95% confidence interval (CI) is 1 or less then the OR is not different statistically from 1). An OR of 1 or less indicates no effect. Even if OR is greater than 1, if the lower bound of the 95% confidence interval (CI) is 1 or less then the OR is not different statistically from 1.) to take into account other relevant occupational exposures and tobacco smoking. The OR for asbestos exposure was 0.92 (95% confidence interval (CI) 0.73-1.15) in Central and Eastern Europe and 1.85 (95%CI 1.07-3.21) in the UK. Similar ORs were found for exposure to amphibole asbestos. The OR for MMVF exposure was 1.23 (95%CI 0.88-1.71) with no evidence of heterogeneity by country. The Central and Eastern European asbestos industry had been reliant upon Russia for supplying asbestos in the 30 to 50 years prior, when exposure would have been important for determining this outcome. Russia, then as now, uses chrysotile asbestos commercially. While not discussed directly in this publication, the differences in the ORs are readily understood by the fact that the UK was the largest importer and users of amphibole per capita in the world. In comparison, in Central and Eastern Europe chrysotile alone was used. The Carl et al. (2006) study clearly demonstrates that when chrysotile alone was used as in Central and Eastern Europe, there is no measurable lung cancer risk.

White et al. (2008):

South Africa, like Austria, represents a very particular situation in the history of asbestos use. Both countries have historically been the major sources of amphiboles (crocidolite and amosite (in South Africa), and have used these varieties of asbestos locally along with chrysotile, which was also mined in both South Africa and Australia.

In both these countries, the number of mesothelioma cases has been much higher than anywhere else in the world. White et al, (2008) have indicated that 23% of cases in South Africa were found in persons never employed in mining. These cases, however, were found associated with living in neighborhoods close to amphibole mining facilities, predominately one area with crocidolite mines, thus associated with environmental exposure.

The authors conclude: “No cases (of mesothelioma) were associated with South African chrysotile. Consequently, in the vast majority of cases of mesothelioma, environmental exposure to asbestos occurred in the Northern Cape province, in proximity to mines, mills and dumps where crocidolite was processed. Crocidolite appears more mesotheliomagenic than amosite, and chrysotile has not been implicated in the disease. This is true for both occupationally and environmentally exposed individuals”.

Pierce et al. (2008):

In an evaluation of reported no-effect chrysotile asbestos exposures for lung cancer and mesothelioma, Pierce et al. (2008) reviewed 368 studies to assess the availability of cumulative exposure information, information on fiber type, and/or evidence of significant exposures to amphiboles. Of these, 350 studies were excluded due to lack of this information. Of the remaining studies, 14 were found to meet the inclusion criteria where lung cancer risk was stratified by cumulative chrysotile exposure, and four such studies were found for mesothelioma.

The authors reported that the majority of the cumulative “no-effects” exposure levels for lung cancer and mesothelioma fell in a

range of approximately 25-1,000 f/cc-yr and 15-500 f/cc-yr, respectively, and a majority of the studies did not report an increased risk at the highest estimated exposure. The authors also discussed potential sources of uncertainty in these values which include errors in the cumulative exposure estimates, conversion of dust counts to fiber data, and use of national age-adjusted mortality rates. Discussed as well were potential biases, which included smoking as being rarely controlled for and that amphibole exposure did in fact occur in a majority of the studies, which would bias many of the reported “no-effect” exposure levels towards lower value.

EPIDEMIOLOGY STUDIES PUBLISHED SINCE THE REVIEW:**Sichletidis et al. (2009):**

Sichletidis et al. (2009) reported on an investigation into the mortality rate among workers exposed to relatively pure chrysotile in an asbestos cement factory in Greece. The asbestos cement plant was opened in 1968 and the investigation covered all 317 workers. The plant uses 2000 tons of chrysotile annually. Regular asbestos fiber measurements were made and the day and cause of death recorded among active and retired workers. Asbestos fiber concentrations were always below permissible levels. Fifty-two workers died during the study. The cause was cancer in 28 subjects, with 16 of those cases diagnosed as lung cancer. No case of mesothelioma was reported. Death was attributed to cardiovascular diseases in 23 subjects and to liver cirrhosis in 1. The overall mortality rate was significantly lower than that of the Greek general population, standardized mortality ratio (SMR) was 0.71 (95% CI 0.53-0.93). Mortality due to cancer was increased (SMR: 1.15, 95% CI 0.77-1.67), mainly due to lung cancer mortality (SMR: 1.71, 95% CI 0.98-2.78), but not significantly. The authors concluded that occupational exposure to relatively pure chrysotile within permissible levels was not associated with a significant increase in lung cancer or with mesothelioma. Decreased overall mortality of workers indicates a healthy worker effect, which – together with the relatively small cohort size – could have prevented the detection of small risks.

Paoletti & Bruni (2009):

Paoletti & Bruni (2009) reported on the size distribution of amphibole fibers from lung and pleural tissue samples of mesothelioma cases due to environmental exposure. This study was initiated in order to evaluate the hypothesis that fibers less than 5µm long could enter the pulmonary pleural barrier and reach the parietal pleura thus inducing mesothelioma. The size of amphibole fibers from healthy lung tissue was compared with those from pleural tissue samples from subjects whose death cause was mesothelioma. The authors, however, did not quantify the tissue burdens of fibers per mg of tissue in the lung or pleura. We note that this hypothesis is flawed in that recent research emphasizes failure of long fibers that reach the pleural space to clear through the parietal pleural stomata, that is the initiating event retaining fiber dose at the parietal mesothelium (discussed later). Four cases of mesothelioma due to environmental exposure were studied with the fibers from pleural tissue characterized by SEM with the chemical composition confirmed by x-ray microanalysis. The authors reported that the average length of fibers from the lung and pleural tissues taken from the same subject did not differ by more than 10 – 12 %. Ninety-five percent of fibers found in the lung tissue had a length greater than 5µm and 98% of the fibers found in the pleural tissues had a length greater than 5µm. Additionally, the authors reported that the average diameter of fibers found in pleural tissue was 70% of the diameter of the fibers found in the lung tissues. The authors concluded that the experimental data obtained in this study confirmed the correlation between malignant mesothelioma and the presence in the lung and pleural tissues of fibers with a length greater, even much greater, than 4 - 5µm, and that the hypothesis that the chief factors inducing mesothelioma are “ultrashort” “ultrathin” fibers appears rather weak.

Schneider et al. (2010):

Schneider et al. (2010) reported on the measurement of asbestos fiber content of the lungs as it was associated with diffuse interstitial (DPF). The asbestos fiber burden was determined in patients with diffuse pulmonary fibrosis who had a history of asbestos exposure in which their biopsies did not meet established criteria for asbestosis. This was compared to the fiber burden and confirmed asbestosis cases. The fiber burden analysis was performed using scanning electron microscopy and energy-dispersive x-ray analysis of lung parenchyma from 86 patients with DPF and 163 patients with asbestosis. The correlation of the number of asbestos fibers found for a quantitative degree of fibrosis was reported. Schneider et al., (2010) reported that the fibrosis scores of the asbestosis cases correlated best with the number of uncoated commercial amphibole fibers.

TOXICOLOGY STUDIES ON CHRYSOTILE AND AMPHIBOLES NOT CONSIDERED BY IARC:**Bernstein DM, Rogers RA, Sepulveda R, Donaldson K, Schuler D, Gaering S, Kunzendorf P, Chevalier J and Holm SE. (2010).**

The pathological response and fate in the lung and pleura of chrysotile in combination with fine particles compared to amosite asbestos following short term inhalation exposure – interim results. *Inhalation Toxicology* 22(11): 937-962.

Bernstein DM, Donaldson K, Decker U, Gaering S, Kunzendorf P, Chevalier J and Holm SE. (2008).

A biopersistence study following exposure to chrysotile asbestos alone or in combination with fine particles. *Inhalation Toxicology* 20: 1009-1028.

Bernstein DM and Hoskins JA. (2006).

The health effects of chrysotile: current perspective based upon recent data. *Regulatory Toxicology and Pharmacology* 45/3 pp. 252-264.

Bernstein DM, Rogers R, Chevalier J and Smith P. (2006).

The toxicological response of Brazilian chrysotile asbestos: A multi-dose sub-chronic 90-day inhalation toxicology study with 92 day recovery to assess cellular and pathological response. *Inhalation Toxicology*, Vol. 18, Issue 5, pp. 313-332.

Bernstein DM, Chevalier J and Smith P. (2005).

Comparison of Calidria chrysotile asbestos to pure tremolite: Final results of the inhalation biopersistence and histopathology following short term exposure. Accepted for Publication in the *Journal Inhalation Toxicology*, *Inhalation Toxicology*, 17(9): 427-449.

Bernstein DM, Rogers R and Smith P. (2004).

The biopersistence of Brazilian chrysotile asbestos following inhalation. *Inhalation Toxicology* 16(9): 745-761.

Bernstein DM, Chevalier J and Smith P. (2003).

Comparison of Calidria chrysotile asbestos to pure tremolite: Inhalation biopersistence and histopathology following short term exposure. *Inhalation Toxicology* 15(14): 101-133.

Bernstein DM, Rogers R and Smith P. (2003).

The biopersistence of Canadian chrysotile asbestos following inhalation. *Inhalation Toxicology* 15(13): 101-128.

FIBER TRANSLOCATION TO THE PLEURAL CAVITY:

Bernstein et al. (2010):

In a recent study by Bernstein et al. (2010), the pathological response and translocation of a commercial chrysotile product similar to that which was used through the mid-1970s in a joint compound intended for sealing the interface between adjacent wall boards was evaluated in comparison to amosite-asbestos. This study was unique in that it presented a combined real-world exposure and was the first study to investigate whether there were differences between chrysotile

and amosite asbestos fibers in time course, size distribution, and pathological response in the pleural cavity. Rats were exposed by inhalation for 5 days (6 h/day) to either sanded joint compound consisting of both chrysotile fibers and sanded joint compound particles (CSP) or amosite-asbestos.

The mean fiber number was 295 fibers/cm³ for chrysotile and 201 fibers/cm³ for amosite. The mean number of WHO fibers (defined > 3µm wide, and with length:width ratios < 3:1; WHO, 1985) in the CSP atmosphere was 1496 fibers/cm³, which was more than 10,000 times the OSHA occupational exposure limit of 0.1 fiber/cm³.

An important part of the Bernstein et al. (2010) study was to design procedures for evaluation of the pleural space while limiting procedural artefacts. These methods included examination of the diaphragm as a parietal pleural tissue and the in situ examination of the lungs and pleural space obtained from freeze-substituted tissue in deeply frozen rats. The diaphragm was chosen as a representative parietal pleural tissue because at necropsy it could be removed within minutes of sacrifice with minimal alteration of the visceral lung surface. The area of the diaphragm chosen for examination included an important lymphatic drainage site (stomata) on the diaphragmatic surface. The use of both confocal microscopy and SEM enabled the identification of fibers as well as examination of the pleural space, in situ, for possible inflammatory response. The examination of the pleural space in situ including the lung, visceral pleura, and parietal pleura in rats deeply frozen immediately after termination provided a non-invasive method for determining fiber location and inflammatory response.

No pathological response was observed at any time point in the CSP-exposure group. The long chrysotile fibers (L > 20 µm) cleared rapidly (T_{1/2} of 4.5 days) and were not observed in the pleural cavity. In contrast, a rapid inflammatory response occurred in the lung following exposure to amosite resulting in Wagner grade 4 interstitial fibrosis within 28 days. Long amosite fibers had a T_{1/2} > 1000 days in the

lung and were observed in the pleural cavity within 7 days post exposure. By 90 days, the long amosite fibers were associated with a marked inflammatory response on the parietal pleura. This study provides support that exposure to chrysotile fibers and joint compound particles following inhalation would not initiate an inflammatory response in the lung, and that the chrysotile fibers present do not migrate to, or cause an inflammatory response in the pleural cavity, the site of mesothelioma formation.

Donaldson et al. (2010):

Donaldson et al. (2010) reviewed the hypothesis regarding the role of long fiber retention in the parietal pleura, inflammation and mesothelioma for the amphibole asbestos amosite, and for carbon nanotubes. This review synthesizes new data with multi-walled carbon nanotubes (CNT) with the hypothesis developed for amphibole asbestos for the behavior of long fibers in the lung and their retention in the parietal pleura leading to the initiation of inflammation and pleural pathology such as mesothelioma. The authors describe evidence that a fraction of all deposited particles reach the pleura and that a mechanism of particle clearance from the pleura exists through stomata in the parietal pleura. They suggest that these stomata are the site of retention of long fibers which cannot negotiate them, leading to inflammation and pleural pathology including mesothelioma. Long fiber retention in the stomata, as a consequence of length-restricted clearance through the normal stomatal clearance system, initiates inflammation and pleural pathology including mesothelioma.

The authors conclude that this general hypothesis on the key role of fiber length-restricted clearance from the pleural space as a mechanism for delivering a high, focuses, effective dose of long fibers to the mesothelial cells around the parietal pleural stomata, has important implications. These lie in future research into the mesothelioma hazard from HARN (High Aspect Ratio Nanoparticles) but also for our current view of the origins of asbestos-initiated pleural mesothelioma and the use of lung parenchymal fiber burden as a correlate of this tumor, which arises in the parietal pleura, not the lung parenchyma or visceral pleura.

REFERENCES:

- Roggli VL, Volimer RT, Butnor KJ, Sporn TA. (2002).** Tremolite and mesothelioma. *Ann Occup Hyg* 46(5):447-53.
- Roggli VL, Sharma A, Butnor KJ, Sporn T, Volimer RT. (2002).** Malignant mesothelioma and occupational exposure to asbestos: a clinicopathological correlation of 1445 cases. *Ultrastruct Pathol* 26(2): 55-65.
- Hodgson JT, McElvenny DM, Darnton AJ, Price MJ, Peto J. (2005)** The expected burden of mesothelioma mortality in Great Britain from 2002 to 2050. *Br J Cancer* 92(3):587-593.
- Yarborough CM. (2006).** Chrysotile as a cause of mesothelioma: An assessment based on epidemiology. *Critical Reviews in Toxicology*. 36: 165-187.
- Carel R, Olsson AC, Zaridze D, Szeszenia-Dabrowska N, Rudnai P, Lissowska J, Fabianova E, Cassidy A, Mates D, Bencko V, Foretova L, Janout V, Fevotte J, Fletcher T, Mannetje A, Brennan P, Boffetta P. (2006).** Occupational exposure to asbestos and man-made vitreous fibers and risk of lung cancer : a multicenter case-control study in Europe. *Occup Environ Med*. 64(8): 502-8.
- White N, Nelson G and Murray J. (2008).** South African experience with asbestos related environmental mesothelioma: Is asbestos fiber type important? *Regul Toxicol and Pharmacol* 52 (1 Suppl): S92-S96.
- Pierce JS, McKinley MA, Paustenbach DJ, Finley BL. (2008).** An evaluation of reported no-effect chrysotile asbestos exposures for lung cancer and mesothelioma. *Crit Rev Toxicol* 38(3): 191-214.
- Sichletidis L, Chloros D, Spyrtos D, Haidich AD, Fourkiotou I, Kakoura M, Patakas D. (2009).** Mortality from occupational exposure to relatively pure chrysotile: A 39-year study. *Respiration*. 78: 63-68.

Paoletti L, Bruni BM.(2009).

Size distribution of amphibole fibers from lung and pleural tissues sampled from mesothelioma cases due to environmental exposure. *Med Lav* 100(1): 11-20.

Schneider F, Sporn TA, Roggli VL. (2010).

Asbestos fiber content of lungs with diffuse interstitial fibrosis: An analytical scanning electron microscopic analysis of 249 cases. *Arch Pathol Lab Med* 134(3): 457-461.

Bernstein DM, Rogers RA, Sepulveda R, Donaldson K, Schuler D, Gaering S, Kunzendorf P, Chevalier J and Holm SE. (2010).

The pathological response and fate in the lung and pleura of chrysotile in combination with fine particles compared to amosite asbestos following short term inhalation exposure – interim results. *Inhalation Toxicology* 22(11): 937-962.

Bernstein DM, Donaldson K, Decker U, Gaering S, Kunzendorf P, Chevalier J and Holm SE. (2008).

A biopersistence study following exposure to chrysotile asbestos alone or in combination with fine particles. *Inhalation Toxicology* 20: 1009-1028.

Bernstein DM and Hoskins JA. (2006).

The health effects of chrysotile: current perspective based upon recent data. *Regulatory Toxicology and Pharmacology* 45/3 pp. 252-264.

Bernstein DM, Rogers R, Chevalier J and Smith P. (2006).

The toxicological response of Brazilian chrysotile asbestos: A multi-dose sub-chronic 90-day inhalation toxicology study with 92 day recovery to assess cellular and pathological response. *Inhalation Toxicology, Vol. 18, Issue 5, pp. 313-332.*

Bernstein DM, Chevalier J and Smith P. (2005).

Comparison of Calidria chrysotile asbestos to pure tremolite: Final results of the inhalation biopersistence and histopathology following short term exposure. Accepted for Publication in the *Journal Inhalation Toxicology, Inhalation Toxicology, 17(9): 427-449.*

Bernstein DM, Rogers R and Smith P. (2004).

The biopersistence of Brazilian chrysotile asbestos following inhalation. *Inhalation Toxicology* 16(9): 745-761.

Bernstein DM, Chevalier J and Smith P. (2003).

Comparison of Calidria chrysotile asbestos to pure tremolite: Inhalation biopersistence and histopathology following short term exposure. *Inhalation Toxicology* 15(14): 101-133.

Bernstein DM, Rogers R and Smith P. (2003).

The biopersistence of Canadian chrysotile asbestos following inhalation. *Inhalation Toxicology* 15(13): 101-128.

Donaldson K, Murphy FA, Duffin R, Poland CA. (2010).

Asbestos, carbon nanotubes and the pleural mesothelium: a review of the hypothesis regarding the role of long fiber retention in the parietal pleura, inflammation and mesothelioma. *Part Fiber Toxicol* 7:5.

ANNEX E

OTHER SCIENTIFIC PUBLISHED STUDIES

REFERENCES

Adamson IY, Bakowska J, Bowden DH. (1993). Mesothelial cell proliferation after installation of long or short asbestos fibers into mouse lung. *Am J Pathol*, 142: 1209-1216.

Adamson IY, Bakowska J, Bowden DH. (1994). Mesothelial cell proliferation: a nonspecific response to lung injury associated with fibrosis. *Am J Respir Cell Mol Biol*, 10: 253-258.

Addison J, Davies LS. (1990). Analysis of amphibole asbestos in chrysotile and other materials. *Ann Occup Hyg*, 34: 159-175.
Aguilar-Madrid G, Robles-Pérez E, Juárez-Pérez Ca, et al. (2010). Case-control study of pleural mesothelioma in workers with social security in Mexico. *Am J Ind Med*, 53: 241-251.

Ashcroft T. (1973). Epidemiological and quantitative relationships between mesothelioma and asbestos on Tyneside. *J Clin Pathol*, 26: 832-840.

Aust AE, Cook PM, Dodson RF. (2011). Morphological and chemical mechanisms of elongated mineral particle toxicities. *J Toxicol Environ Health B Crit Rev*, 14: 40-75.

Ballweg JA, Bray RM. (1989). Smoking and tobacco use by US military personnel. *Mil Med*, 154:165-168.

Balzer JL, Cooper WC. (1968). The work environment of insulating workers. *Am Ind Hyg Assoc J*, 29: 222-227.

Barbieri PG, Mirabelli D, Somigliana A, et al. (2012). Asbestos fiber burden in the lungs of patients with mesothelioma who lived near asbestos-cement factories. *Ann Occup Hyg*, 2012 (Epub ahead of print). doi:10.1093/annhyg/mer126.

Bates TF, Sand LB, Mink JF. (1950). Tubular crystals of chrysotile asbestos. *Science*, 111: 512-513.

Bellmann B, Muhle H, Creutzenberg O, et al. (2003). Calibration study on subchronic inhalation toxicity of man-made vitreous fibers in rats. *Inhal Toxicol*, 15: 1147-1177.

Berman DW, Crump KS. (2003). Draft technical support document for a protocol to assess asbestos-related risk (report). Washington (DC): Office of Solid Waste and Emergency Response US Environmental Protection Agency.

Berman DW, Crump KS. (2008). A meta-analysis of asbestos-related cancer risk that addresses fiber size and mineral type. *Crit Rev Toxicol*, 38: 49-73.

Berman DW, Crump KS, Chatfield EJ, et al. (1995). The sizes, shapes, and mineralogy of asbestos structures that induce lung tumors or mesothelioma in AF/HAN rats following inhalation. *Risk Anal*, 15: 181-195

Bernstein DM. (2007). Synthetic vitreous fibers: a review toxicology, epidemiology and regulations. *Crit Rev Toxicol*, 37:839-886.

Bernstein D, Castranova V, Donaldson K, et al. (2005c). Testing of fibrous particles: short-term assays and strategies. *Inhal Toxicol*, 17: 497-537.

Bernstein DM, Chevalier J, Smith P. (2005b). Comparison of Calidria chrysotile asbestos to pure tremolite: final results of the inhalation biopersistence and histopathology following short term exposure. *Inhal Toxicol*, 17:427-449.

Bernstein DM, Riego-Sintes JM, Ersboell BK, et al. (2001a). Biopersistence of synthetic mineral fibers as a predictor of chronic inhalation toxicity in rats. *Inhal Toxicol*, 13: 823-849.

Bernstein DM, Riego-Sintes JM, Ersboell BK, et al. (2001b). Biopersistence of synthetic mineral fibers as a predictor of chronic intraperitoneal injection tumor response in rats. *Inhal Toxicol*, 13: 851-875.

Bernstein DM, Rogers R, Chevalier J. et al. (2006). The toxicological response of Brazilian chrysotile asbestos: a multidose sub-chronic 90 d inhalation toxicology study with 92 day recovery to assess cellular and pathological response. *Inhal Toxicol*, 18: 313-332.

Bernstein DM, Rogers RA, Sepulveda R. et al. (2010). The pathological response and fate in the lung and pleura of chrysotile in combination with fine particles compared to amosite asbestos following short term inhalation exposure – interim results. *Inhal Toxicol*, 22: 937-962.

Bernstein DM, Rogers RA, Sepulveda R. et al. (2011). Quantification of the pathological response and fate in the lung and pleura of chrysotile in combination with fine particles compared to amosite-asbestos following short-term inhalation exposure. *Inhal Toxicol*, 23: 372-391.

Bernstein DM, Rogers R, Smith P. (2003). The biopersistence of Canadian chrysotile asbestos following inhalation. *Inhal Toxicol*, 15: 101-128.

Bernstein DM, Rogers R, Smith P. (2004). The biopersistence of Brazilian chrysotile asbestos following inhalation. *Inhal Toxicol*, 16: 745-761.

Bernstein DM, Rogers R, Smith P. (2005a). The biopersistence of Canadian chrysotile asbestos following inhalation: final results through 1 year after cessation of exposure. *Inhal Toxicol*, 17: 1-14.

Bolton RE, Vincent JH, Jones AD, et al. (1983). An overload hypothesis for pulmonary clearance of UICC amosite fibers inhaled by rats. *Br J Ind Med*, 40: 264-272.

Bowles O, Barsigian FM. (1954). Asbestos. In: McGann PW, ed. Minerals yearbook 1951, Bureau of Mines. United States Government Printing Office, 167-176. Available from <http://digital.library.wisc.edu/1711.dl/EcoNatRes.MinYB1951..>

Bowles O, Stoddard BH. (1933). Asbestos. In: Kiessling OE, ed. Minerals yearbook 1932-33, United States Government Printing Office, 1933, 745-752. Available from <http://digital.library.wisc.edu/1711.dl/EcoNatRes.MinYB193132>

Bragg GM. (2001). Fiber release during the handling of products containing chrysotile asbestos using modern control technology. In: Nolan RP, Langer AM, Ross M. et al., eds. The health effects of chrysotile asbestos: contribution of science to risk-management decisions. Ottawa, Canada: *Canadian Mineralist, Spec. Publ.* 5: 111-114.

Bray RM, Guess LL, Marsden ME. (1989). Prevalence trends, and correlates of alcohol use, nonmedical drug use, and tobacco use among US military personnel. *Mil Med*, 154: 1-11.

Bray RM, Marsden ME, Peterson MR. (1991). Standardized comparisons of the use of alcohol, drugs, and cigarettes among military personnel and civilians. *Am J Public Health, 81: 865-869.*

Breysse PN, Cherrie JW, Addison J. et al. (1989). Evaluation of airborne asbestos concentration using TEM and SEM during residential water tank removal. *Ann Occup Hyg, 33: 243-256.*

Campbell WJ, Huggins CW, Wylie AG. (1980). Chemical and physical characterization of amosite, chrysotile, crocidolite, and nonfibrous tremolite for oral ingestion studies by the National Institute of Environmental Health Sciences (report of investigation 8452). Avondale (MD). United States Department of the Interior, US Bureau of Mines.

Case BW, Abraham JL. (2009). Heterogeneity of exposure and attribution of mesothelioma: trends and strategies in two American countries. *J Phys: Conf Ser, 151: 012008.*

Case BW, Dufresne A, McDonald AD, et al. (2000). Asbestos fiber type and length in lungs of chrysotile textile and production workers: fibers longer than 18 μm . *Inhal Toxicol, 1: 411-418.*

Case BW, McDonald C. (2008). Chrysotile, tremolite, balangeroite and mesothelioma: similar situations? *Occup Environ Med, 65: 815-819.*

Churg A, Wiggs B, Depaoli L, et al. (1984). Lung asbestos content in chrysotile workers with mesothelioma. *Am Rev Respir Dis, 130: 1042-1045.*

Coin PG, Roggli VL, Brody AR. (1992). Deposition, clearance, and translocation of chrysotile asbestos from peripheral and central regions of the rat lung. *Environ Res, 58:97-116.*

Coleman RG. (1996). New Idria serpentinite: a land management dilemma. *Environ Eng. Geoscience, 2: 9-22.*

Conway TL, Trent LK, Conway SW. (1989). Physical Readiness and lifestyle habit among US navy personnel during 1986, 1987 and 1988. Technical Report No 89-23. San Diego, California: Naval Health Research Center.

Cressey BA, Whittaker EJW. (1993). Five-fold symmetry in chrysotile asbestos revealed by transmission electron microscopy. *Mineral Mag. 57: 729-732.*

Davis JM, Addison J, Bolton RE, et al. (1986). The pathogenicity of lung versus short fiber samples of amosite asbestos administered to rats by inhalation and intraperitoneal injection. *Br J Exp Pathol, 67: 414-430.*

Davis JM, Beckett ST, Bolton RE, et al. (1978). Mass and number of fibers in the pathogenesis of asbestos-related lung disease in rats. *Br J Cancer, 37: 673-688.*

Davis JM, Jones AD. (1988). Comparison of the pathogenicity of long and short fibers of chrysotile asbestos in rats. *Br J Exp Pathol, 69: 717-737.*

Dement JM, Brown DP. (1984). Lung cancer mortality among asbestos textile workers: a review and update. *Ann Occup Hyg, 38: 525-532, 412.*

Dement JM, Harris Jr RL, Symons MJ, et al. (1982). Estimates of dose-response for respiratory cancer among chrysotile asbestos textile workers. *Ann Occup Hyg, 26: 868-887.*

Devesa SS, Grauman DJ, Blot WJ, et al. (1999). Atlas of cancer mortality in the United States 1950-1994 (*NIH Publication No. 99-4564*). Bethesda (MD): National Institutes of Health, National Cancer Institute.

Dreessen WC, Dallavalle JM, Edwards TI, et al. (1938). A study of asbestosis in the asbestos textile industry (*Public Health Bulletin No. 211*). Bethesda (MD): National Institute of Health, 91-125.

Elmes PC, Wade OL. (1965). Relationship between exposure to asbestos and pleural malignancy in Belfast. *Ann NY Acad Sci*, 132: 549-557.

Esmen NA, Corn M. (1998). Airborne fiber concentrations during splitting open and boxing bags of asbestos. *Toxicol Ind Health*, 14: 843-856.

European Commission Joint Research Centre Institute for Health and Consumer Protection, Unit: Toxicology and Chemical Substances, European Chemicals Bureau. (1999). Methods for the determination of the hazardous properties for human health of man-made mineral fibers (MMMF). Bernstein, DM, Riego-Sintes JMR, eds. Vol. EUR 18748 EN, April 93. Available from: <http://ech.ei.jrc.it/DOCUMENTS/Testing-Methods/mmmfweb.pdf>.

Evans BW. (2004). The serpentinite multisystem revisited: chrysotile is metastable. *Int Geol Rev*, 46: 479-506.

Finley BL, Pierce JS, Phelka AD, et al. (2012). Evaluation of tremolite asbestos exposures associated with the use of commercial products. *Crit Rev Toxicol*, 42: 119-146.

Gazzano E, Riganti C, Tomatis M, et al. (2005). Potential toxicity of nonregulated asbestiform minerals: balangeroite from the western Alps. Part 3: depletion of antioxidant defenses. *J Toxicol Environ Health Part A*, 68:41-49.

Gibbs GW. (1994). The assessment of exposure in terms of fibers. *Ann Occup Hyg*, 38: 477-487, 409-410.

Gibbs AR, Pooley F. (2008). Mineral fibers analysis and asbestos related diseases. In: Craighead JE, Gibbs AR, eds. *Asbestos and its diseases*. New York: Oxford University Press, 299-316.

Goodglick LA, Kane AB. (1990). Cytotoxicity of long and short crocidolite asbestos fibers in vitro and in vivo. *Cancer Res*, 50: 5153-5163.

Green FHY, Harley R, Vallyathan V, et al. (1997). Exposure and mineralogic correlates of pulmonary fibrosis in chrysotile asbestos workers. *Occup Environ Med*, 54: 549-559.

Grosso C, Tomatis M, Turcy F, et al. (2005). Potential toxicity of nonregulated asbestiform minerals: balangeroite from the western Alps. Part 1: identification and characterization. *J Toxicol Environ Health, Part A*, 68: 1-19.

Gross P, Cralley LJ, DeTreville RT. (1967). "Asbestos" bodies: their nonspecificity. *Ann Ind Hyg Assoc J*, 28:541-542.

Hain E, Dalquen P, Bohlig H, et al. (1974). Retrospective study of 150 cases of mesothelioma in Hamburg area (author's transl.). *Int Arch Arbeitsmed*, 33: 15-37.

Hammad Y, Simmons W, Abdel-Kader H, et al. (1988). Effect of chemical composition on pulmonary clearance of man-made mineral fibers. *Ann Occup Hyg*, 22: 769-779.

Hargreaves A, Taylor WH. (1946). An X-ray examination of decomposition products of chrysotile asbestos and serpentine. *Mineral Mag* 27: 204-216.

Hein MJ, Stayner LT, Lehman E., et al. (2007). Follow-up study of chrysotile textile workers: cohort mortality and exposure-response. *Occup Environ Med*, 4: 616-625.

Hesterberg TW, Axten C, McConnell EE, et al. (1999). Studies on the inhalation toxicology of two fibreglasses and amosite asbestos in the Syrian golden hamster. Part I. Results of a subchronic study and dose selection for a chronic study. *Inhal Toxicol*, 11: 747-784.

Hesterberg TW, Hart GA, Chevalier J, et al. (1998). The importance of fiber biopersistence and lung dose in determining the chronic inhalation effects of X607, RCF1, and chrysotile asbestos in rats. *Toxicol Appl Pharmacol*, 153: 68-82.

- Hesterberg TW, Miller WC, McConnell EE, et al. (1993).** Chronic inhalation toxicity of size-separated glass fibers in Fischer 344 rats. *Fundam Appl Toxicol* 20: 464-476.
- Hodgson JT, Darnton A. (2000).** The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. *Ann Occup Hyg* 44: 565-601.
- Ilgren EB. (2004).** Coalinga chrysotile a short fiber, amphibole free, chrysotile Part V – lack of amphibole asbestos contamination. *Indoor Built Environ*, 13: 375-382.
- Ilgren EB, Breña MO, Larragoitia JC, et al. (2008a).** A reconnaissance study of a potential emerging Mexican mesothelioma epidemic due to fibrous zeolite exposure. *Indoor Built Environ*, 17: 496-515.
- Ilgren E, Chatfield E. (1997).** Coalinga fiber – a short, amphibole-free chrysotile. Part 1: Evidence for lack of fibrogenic activity. *Indoor Built Environ*, 6: 264-276.
- Ilgren E, Chatfield E. (1998).** Coalinga fiber – a short, amphibole-free chrysotile. Part 2: Evidence for lack of fibrogenic activity. *Indoor Built Environ*, 7: 18-31.
- Ilgren EB, Pooley FD, Larragoitia JC, et al. (2008b).** First confirmed erionite related mesothelioma in North America. *Indoor Built Environ*, 17: 568-568.
- Ilgren E, Ramirez R, Claros E, et al. (2012).** Fiber width as a determinant of mesothelioma induction and threshold Bolivian crocidolite: epidemiological evidence from Bolivia mesothelioma demography and exposure pathways. *Ann Respir Med*. Available from: www.slm-respiratory.com.
- ILO. (1984).** Safety in the use of asbestos: an ILO code of practice. Geneva: International Labor Office.
- Jones RN, Diem JE, Hughes JM, et al. (1989).** Progression of asbestos effects: a prospective longitudinal study of chest radiographs and lung function. *Br J Ind Med*, 46: 97-105.
- Kanarek MS. (2011).** Mesothelioma from chrysotile asbestos: update. *Ann Epidemiol*, 21: 688-697.
- Karbownik J, Clark S. (1997, 2005, 2006, 2007, 2008, 2009, 2012).** Determination of presence of amphibole asbestos fibers in four bulk samples of chrysotile (report to client, project no: 610). Edinburgh, UK: IOM Consulting Limited.
- Kashansky SV, Domnin SG, Kochelayev VA, et al. (2001).** Retrospective view of airborne dust levels in workplace of a chrysotile mine in Ural, Russia. *Ind. Health*, 39: 51-56.
- Kliment CR, Clemens K, Oury TD. (2009).** North American erionite-associated mesothelioma with pleural plaques and pulmonary fibrosis: a case report. *Int J Clin Exp Pathol*, 2: 407-410.
- Kobell F. (1834).** Ueber den schillerrden asbest von Reichenstein in Schlesien. *J Prakt Chemie*, 2: 297-298.
- Kurumatami N, Kumagai S. (2008).** Mapping the risk of mesothelioma due to neighborhood asbestos exposure. *Am J Respir Crit Care Med*, 178: 624-629.
- Larsen G. (1989).** Experimental data on in vitro fiber solubility. *IARC Sci Publ*, 90: 134-139.
- LeBouffant L, Daniel H, Henin JP, et al. (1987).** Experimental study on long-term effects of inhaled MMMF on the lung of rats. *Ann Occup Hyg*, 31: 765-790.
- Legifrance. (1994).** Décret no 94-645 du 26 juillet 1994 modifiant le décret no 88-466 du 28 avril 1988 relatif aux produits contenant de l'amiante, JORF no 173 du 28 juillet 1994, page 10907. Available from: <http://www.legifrance.gouv.fr>.
- Liddell FD, McDonald As, McDonald JC. (1997).** The 1891-1920 birth cohort of Quebec chrysotile miners and millers: development from 1904 and mortality to 1992. *Ann Occup Hyg*, 41: 13-36.

- Liddell FD, McDonald As, McDonald JC. (1998).** Dust exposure and lung cancer in Quebec chrysotile miners and millers. *Ann Occup Hyg*, 42: 7-20.
- Lippmann M. (1990).** Effects of fiber characteristics on lung deposition, retention, and disease. *Environ Health Perspect*, 88: 311-317.
- Mancuso TF. (1983).** Mesothelioma among machinists in railroad and other industries. *Am J Ind Med*, 405: 501-513.
- Mancuso TF. (1988).** Relative risk of mesothelioma among railroad machinists exposed to chrysotile. *Am J Ind Med*, 13: 639-657. Erratum in: (1989). *Am J Ind Med*, 15:125.
- Mast RW, McConnell EE, Anderson R, et al. (1995).** Studies on the chronic toxicity (inhalation) of four types of refractory ceramic fiber in male Fischer 344 rats. *Inhal Toxicol* 7: 425: 467.
- McClellan RO, Miller FJ, Hesterberg TW, et al. (1992).** Approaches to evaluating the toxicity and carcinogenicity of man-made fibers: summary of a workshop held November 11-13, 1991, Durham, North Carolina. *Regul Toxicol Pharmacol*, 16: 321-364.
- McConnell EE, Axten C, Hesterberg TW, et al. (1999).** Studies on the inhalation toxicology of two fibreglasses and amosite asbestos in the Syrian golden hamster. Part II. Results of chronic exposure. *Inhal Toxicol*, 11: 785-835.
- McDonald AD, Case BW, Churg A, et al. (1997).** Mesothelioma in Quebec chrysotile miners and millers: epidemiology and aetiology. *Ann Occup Hyg*, 41: 707-719.
- McDonald AD, Fry JS, Woolley AJ, et al. (1983).** Dust exposure and mortality in an American factory using chrysotile, amosite, and crocidolite in mainly textile manufacture. *Br J Ind Med*, 40:368-374.
- McDonald AD, Fry JS, Woolley AJ, et al. (1984).** Dust exposure and mortality in an American chrysotile asbestos friction products plant. *Br J Ind Med*, 41:151-157.
- McDonald AD, Harper A, McDonald JC, et al. (1970).** Epidemiology of primary malignant mesothelial tumors in Canada. *Cancer*, 26: 914-919.
- McDonald JC, Liddell FD. (1979).** Mortality in Canadian miners and millers exposed to chrysotile. *Ann NY Acad Sci*, 330: 1-9.
- McDonald JC, McDonald AD. (1995).** Chrysotile, tremolite and mesothelioma. *Science*, 267: 775-776.
- McDonald JC, McDonald AD. (1996).** The epidemiology of mesothelioma in historical context. *Eur Respir J*, 9: 1932-1942.
- McEwen J, Finlayson A, Mair A, et al. (1970).** Mesothelioma in Scotland. *Br Med J*, 4: 575-578.
- Morgan A. (1995).** Deposition of inhaled asbestos and man-made mineral fibers in the respiratory tract. *Ann Occup Hyg*, 39: 747-758.
- Morris GF, Notwick AR, et al. (2004).** Development of lung tumors in mutant p53-expressing mice after inhalation exposure to asbestos. *Chest*, 125: 85S-86S.
- Morrow PE. (1988).** Possible mechanisms to explain dust overloading of the lung. *Fundam Appl Toxicol*, 10: 369-384.
- Morrow PE. (1992).** Dust overloading of the lungs: update and appraisal. *Toxicol Appl Pharmacol*, 113: 1-12.
- Muhle H, Bellman B, Heinrich U. (1988).** Overloading of lung clearance during chronic exposure of experimental animals to particles. *Ann Occup Hyg*, 32: 141-147.
- Muhle H, Pott F, Bellmann B, et al. (1987).** Inhalation and injection experiments in rats to test the carcinogenicity of MMMF. *Ann Occup Hyg*, 31: 755-764.
- Musti M, Pollice A, Cavone D, et al. (2009).** The relationship between malignant mesothelioma and an asbestos cement plant environmental risk: a spatial case-control study in the city of Bari (Italy). *Int Arch Occup Environ Health*, 82: 489-497.

- Newhouse ML, Thompson H. (1965).** Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. *Br J Ind Med*, 22: 261-269.
- NIOSH. (2011).** Asbestos fibers and other elongate mineral particles: state of the science and roadmap for research (revised April 2011, publication number 2011-159, current intelligence bulletin 62). Washington (DC): Center for Disease Control and Prevention, National Institute for Occupational Safety and Health.
- Noll W, Kircher H. (1951).** Über die Morphologie von Asbesten und ihren Zusammenhang mit der Kristallstruktur. *Neuws Jb. Mineral., Mh.* 1951: 219-240.
- Oberdörster G. (1995).** Lung particle overload: implications for occupational exposures to particles. *Regul Toxicol Pharmacol*, 21: 123-135. *Regul Toxicol Pharmacol*, 21: 123-135.
- Osmond-McLeod MJ, Poland CA, Murphy F, et al. (2011).** Durability and inflammogenic impact of carbon nanotubes compared with asbestos fibers. *Part Fiber Toxicol*, 8:15.
- Oze C, Solt K. (2010).** Biodurability of chrysotile and tremolite asbestos in simulated lung and gastric fluids. *Am Mineral*, 95: 825-831.
- Pan XL, Day HW, Wang W, et al. (2005).** Residential proximity to naturally occurring asbestos and mesothelioma risk in California. *Am J Respir Crit Care Med*, 172: 1019-1025.
- Pauling L. (1930).** The structure of the chlorites. *Proc Nat Acad Sci USA*, 16: 578-582.
- Perry RH, Chilton CH, eds. (1973).** Chemical engineers' handbook. 5th ed. New York (NY): McGraw-Hill. TIC:242591.
- Pierce JS, McKinley MA, Paustenbach DJ, et al. (2008).** An evaluation of reported no-effect chrysotile asbestos exposures for lung cancer and mesothelioma. *Crit Rev Toxicol*, 38: 191-214.
- Pinkerton KE, Brodt AR, McLaurin DA, et al. (1983).** Characterization of three of chrysotile asbestos after aerosolization. *Environ Res*, 31: 32-53.
- Piolatto G, Negri E, La Vecchia C, et al. (1990).** An update of cancer mortality among chrysotile asbestos miners in Balangero, northern Italy. *Br J Ind Med*, 47: 810-814.
- Pohanish RP, ed. (2008).** Sittig's handbook of toxic and hazardous chemicals and carcinogens. 5th ed. Norwich (NY): William Andrews, 272.
- Poland CA, Duffin R, Kinloch I, et al. (2008).** Carbon nanotubes introduced into the abdominal cavity of mice show asbestos-like pathogenicity in a pilot study. *Nat Nanotechnol*, 3: 423-428.
- Pooley F. (2003).** Personal communication of a report prepared under contract to KCAC of the examination of chrysotile asbestos samples from the asbestos mine and processing plant of KCAC, Inc., 1991. Cited in Bernstein DM, Chevalier J, Smith P. (2005). Comparison of Calidria chrysotile asbestos to pure tremolite: Final results of the inhalation biopersistence and histopathology examination following short-term exposure. *Inhal. Toxicol*, 17: 427-449.
- Pooley FD, Mitha R. (1986).** Determination and interpretation of the levels of chrysotile asbestos in lung tissue. In: Wagner JC, ed. Biological effects of chrysotile. *Accomplishments in Oncology Vol. 1. No. 2, Philadelphia: Lippincoll*, 12-18.
- Roggli VL, Sharma A, Butnor KJ, et al. (2002b).** Malignant mesothelioma and occupational exposure to asbestos: a clinicopathological correlation of 1445 cases. *Ultrastruct Pathol*, 26: 55-65.
- Roggli VL, Vollmer RT, Butnor KJ, et al. (2002a).** Tremolite and mesothelioma. *Ann Occup Hyg*, 46: 447-453.
- Rubino GF, Scansetti G, Donna A, et al. (1972).** Epidemiology of pleural mesothelioma in North-western Italy (Piedmont). *Br J Ind Med*, 29: 436-442.

- Schneider F, Sporn TA, Roggli VL. (2010).** Asbestos fiber content of lungs with diffuse interstitial fibrosis: an analytical scanning electron microscopic analysis of 249 cases. *Arch Pathol Lab Med*, 134: 457-461.
- Sebastien P., McDonald JC, McDonald AD, et al. (1989).** Respiratory cancer in chrysotile textile and mining industries: exposure inferences from lung analysis. *Br J Ind Med*, 46: 180-187.
- Selikoff IJ, Churg J, Hammond EC. (1984).** Landmark article April 6, 1964: Asbestos exposure and neoplasia. By Irving J. Selikoff, Jacob Churg, and E. Cuyler Hammond. *JAMA*, 252: 91-95.
- Seshan K. (1983).** How are the physical and chemical properties of chrysotile asbestos altered by a 10-year residence in water and up to 5 days in simulated stomach acid? *Environ Health Perspect*, 53: 143-148.
- Silvestri S, Magnani C, Calisti R, et al. (2001).** The experience of the Balangero chrysotile asbestos mine in Italy: health effects among workers mining and milling asbestos and the health experience of persons living nearby. In: Nolan RP, Langer AM, Ross M, Wicks FJ, Martin RF, eds. The health effects of chrysotile asbestos: contribution of science to risk-management decisions. Ottawa, Canada: *Canadian Mineralogist, Spec. Publ.* 5: 177-186.
- Skinner HCW, Ross M, Drondel C. (1988).** Asbestos and other fibrous materials – mineralogy, crystal chemistry, and health effects. New York (NY): Oxford University Press, 204.
- Smith AH, Wright CC. (1996).** Chrysotile asbestos is the main cause of pleural mesothelioma. *Am J Ind Med*, 30: 252-266.
- Speil S, Leineweber JP. (1969).** Asbestos minerals in modern technology. *Environ Res*, 2: 166-208.
- Stanton MF. (1973).** Some etiological considerations of fiber carcinogenesis. In: Bogovski P, Gilson JC, Timbrell V, Wagner JC, eds. *Biological effects of asbestos*. Lyon: WHO IARC, 289-294.
- Stanton MF, Layard M, Tegeris A, et al. (1981).** Relation of particle dimension to carcinogenicity in amphibole asbestos and other fibrous minerals. *J Natl Cancer Inst*, 67: 965-975.
- Stanton MF, Wrench C. (1972).** Mechanisms of mesothelioma induction with asbestos & fibrous glass. *J Natl Cancer Inst*, 48: 797-821.
- Stayner L, Kuempel E, Gilbert S, et al. (2008).** An epidemiological study of the role of chrysotile asbestos fiber dimensions in determining respiratory disease risk in exposed workers. *Occup Environ Med*, 65: 613-619.
- Suquet H. (1989).** Effects of dry grinding and leaching on the crystal structure of chrysotile. *Clays Clay Miner*, 37: 439-445.
- Timbrell V, Hyett AW, Skidmore JW. (1968).** A simple dispenser for generating dust clouds from standard reference samples of asbestos. *Ann Occup Hyg*, 11: 273-281.
- Timbrell V, Rendall REG. (1972).** Preparation of the UICC standard reference samples of asbestos. *Powder Technol.* 5: 279-287.
- Titulaer MK, van Miltenburg JC, Jansen JBH, et al. (1993).** Characterization of tubular chrysotile by thermoporometry, nitrogen sorption, drifts, and TEM. *Clays Clay Miner*, 41: 496-513.
- Tossavainen A, Kotilainen M, Takahashi K, et al. (2001).** Amphibole fibers in Chinese chrysotile asbestos. *Ann Occup Hyg*, 45: 145-152.
- Tossavainen A, Kovalevsky E, Vanhala E, et al. (2000).** Pulmonary mineral fibers after occupational and environmental exposure to asbestos in the Russian chrysotile industry. *Am J Ind Med*, 37: 327-333.
- Tossavainen A, Riala R, Kamppi R, et al. (1996).** Dust measurements in the chrysotile mining and milling operations of Uralasbest Company, Asbest, Russia (summary report). Helsinki: Institute of Occupational Health, 220.

- Turci F, Tomatis M, Gazzano E, et al. (2005).** Potential toxicity of nonregulated asbestiform minerals: balangeroite from the western Alps. Part 2: oxidant activity of the fibers. *J Toxicol Environ Health Part A*, 68: 21-39.
- US DHHS. (1989).** Reducing the health consequences of smoking: 25 years of progress. A report of the Surgeon General, 1989 (DHHS Publication No (CDC) 89-8411). Rockville (MD): Public Health Service, Centers for Disease Control, Office on Smoking and Health.
- US EPA. (2001).** US EPA health effects test guidelines OPPTS 870.8355 guideline for combined chronic toxicity/carcinogenicity testing of respirable fibrous particles (EPA 712-C-01-352, July). Washington (DC): US Environmental Protection Agency.
- Van Orden DR, Lee RL, Sanchez MS, et al. (2012).** The size distribution of airborne Bolivian crocidolite fibers (case report). *The Annals of Respiratory Medicine*, Monroeville (PA): RJ Lee Group. Inc. Available from www.slm-respiratory.com (last accessed 26 Jul 2012).
- Veblen DR, Wylie AG. (1993).** Mineralogy of amphiboles and 1:1 layer silicates. In: Guthrie Jr GD, Mossman BT, eds. Health effects of mineral dusts. Washington (DC): Mineralogical Society of America, *Reviews in Mineralogy*, Vol. 28: 61-137.
- Virta RL. (2002).** Asbestos: geology, mineralogy, mining, and uses. Prepared in cooperation with Kirk-Othmer encyclopedia of chemical technology. USGS Open file 02-149. Online Edition. New York (NY): Wiley-Interscience, a division of John Wiley & Sons, Inc.
- Virta RL. (2005).** Mineral commodity profiles – asbestos (US Geological Survey circular 1255-KK). Reston (VA): US Geological Survey.
- Virta RL. (2006).** Worldwide asbestos supply and consumption trends from 1900 through 2003 (circular 1298). Reston (VA): US Geological Survey.
- Virta RL. (2011).** 2010 minerals yearbook – asbestos. US Geological Survey Minerals Yearbook – 2010. Reston (VA): US Geological Survey.
- Von Kobell F. (1834).** Ueber den schillernden asbest von Reichenstein in Schlesien: *Jour Prakt Chemie*, 2: 297-298.
- Vorwald AJ, Durkan TM, Pratt PC. (1951).** Experimental studies of asbestosis, *AMA Arch Ind Hyg Occup Med*, 3: 1-43.
- Wagner JC, Berry G, Skidmore JW, et al. (1974).** The effects of the inhalation of asbestos in rats. *Br J Cancer*, 29: 252-269.
- Wagner JC, Berry G, Skidmore JW, et al. (1980).** The comparative effects of three chrysotiles by injection and inhalation in rats. In: Wagner JC, ed. *Biological Effects of Mineral Fibers*. IARC publication 30. Lyon: International Agency Research on Cancer, 363-373.
- Wagner JC, Sleggs CA, Marchand P. (1960).** Diffuse pleural mesothelioma and asbestos exposure in the North Western Cape Province. *Br J Ind Med*, 17: 260-271.
- Walton WH. (1982).** The nature, hazards, and assessment of occupational exposure to airborne asbestos dust: a review. *Ann Occup Hyg*, 25: 117-247.
- Wang X, Yano E, Qiu H, et al. (2012).** A 37-year observation of mortality in Chinese chrysotile asbestos workers. *Thorax*, 67: 106-110.
- Warren BE, Bragg WL, (1930).** The structure of chrysotile, H₄Mg₃Si₂O₉. *Z Krystallographie*, 76: 201-210.
- Whittaker EJW. (1957).** The structure of chrysotile. V. Diffuse reflexions and fiber texture. *Acta Cryst.* 10: 149-156.
- Whittaker EJW. (1960).** The crystal chemistry of the amphiboles. *Acta Cryst*, 13: 291-298.

Whittaker EJW. (1963). In Research report: Chrysotile Fibers—Filled or Hollow Tubes? Mathematical interpretation may resolve conflicting evidence. *Chem Eng News*, 41: 34-35

WHO. (1985). Reference methods for measuring airborne man-made mineral fibers (MMM): WHO/EURO MMM reference scheme. Copenhagen: WHO.

WHO. (1988). Environmental health criteria 77: man-made mineral fibers. Vol. 77. Geneva: WHO.

Wicks FJ, O'Hanley DS. (1988). Serpentine minerals structures and petrology. In: Bailey S, ed. Hydrous phyllosilicates (exclusive of micas). Washington, DC: Mineralogical Society of America, Reviews in Mineralogy, Vol. 19, 91-167.

Williams-Jones AE, Normand C, Clark JR, et al. (2001). Controls of amphibole formation in chrysotile deposits: evidence from the Jeffrey Mine, Asbestos, Quebec. In: Nolan RP, Langer AM, Ross M, Wicks FJ, Martin RF, eds. The health effects of chrysotile asbestos: contribution of science to risk-management decisions. Ottawa, Canada: *Canadian Mineralogist, Spec. Publ.* 5:89-104.

Work LT. (1962). Size reduction gets a new stature. *Ind Eng Chem*, 54: 52-54.

Wypych F, Adad LB, Mattoso N, et al. (2005). Synthesis and characterization of disordered layered silica obtained by selective leaching of octahedral sheets from chrysotile. *J Colloid Interface Sci*, 283: 107-112.

Yano E, Wang ZM, Wang XR, et al. (2001). Cancer mortality among workers exposed to amphibole-free chrysotile asbestos. *Am J Epidemiol*, 154: 538-543.

Yano E, Wang ZM, Wang XR, et al. (2009). Mesothelioma in a worker who spun chrysotile asbestos at home during childhood. *Am J Ind Med*, 52: 282-287.

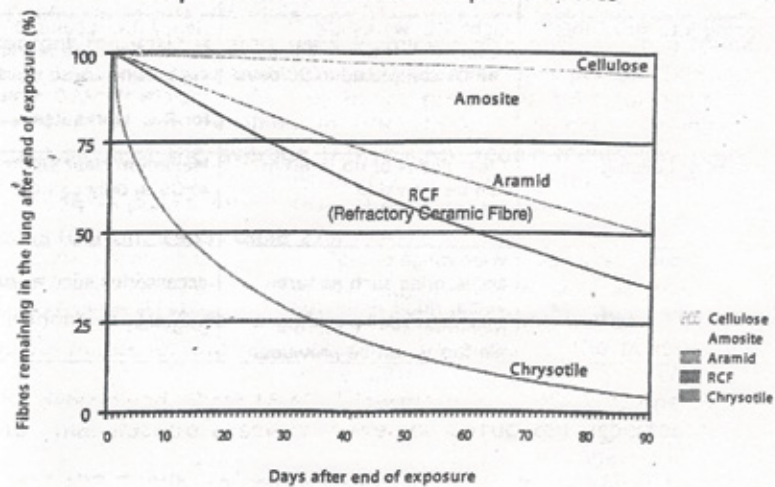
Zamataro RSI, Franzini MJ. (2012). Verificar qualitativamente a tipologia das amostras de amianto extraídas na SAMA, por análise difratométrica de raios X (Relatório No 021E-12). São Paulo – SP, Brazil: Projecontrol Cons. Empresarial e Serviços Ltda.

Zelhuis RL, Versteeg JP, Planteijdt HT. (1975). Pleura mesothelioma and exposure to asbestos. A retrospective case-control study in the Netherlands. *Int Arch Occup Environ Health*, 36: 1-18.

CHRYSTOLE AND AMPHIBOLES: DO NOT MIX UP

Of all the fibres analyzed, chrysotile is the fibre which is most quickly eliminated from the body.

Biopersistence of Several Respirable Fibres



Biopersistence: It is the length of time for inhaled particles to persist in the lungs and adversely affect surrounding tissues before they are eventually cleared.

Biopersistence studies have been carried out on a number of different respirable particles. It has now become clear that there are vast differences among various respirable particles presently used by industry.

There seems to be a continuum of values for biopersistence of mineral particles, from very short persistence (low durability) to practically indefinite persistence (very high durability).



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