SMOKE AND MIRRORS: CHRYSOTILE ASBESTOS IS GOOD FOR YOU – ILLUSION AND CONFUSION BUT NOT FACT

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The illusionist can make objects appear, disappear, or appear to be what they are not by retracting or extending mirrors, disguising the transitions with bursts of confusing blue smoke. Such illusionary tactics are evident in the materials used by the protagonists for the continued use of chrysotile asbestos, their slick glossy color publications depicting tranquil themes, catchy titles surrounded with green leaves, children playing in fields of trees under soaring white birds, etc. The reader is attracted to these colorful and eye catching publications with illusionary titles such as "Why so much emotion?"[1] or "Chrysotile Asbestos Saves Lives"[2] or "Asbestos Fibre Types and Health Risks Are Perceptions Related to FACTS?"[3] Chrysotile asbestos, as portrayed in these texts, is a positive asset to society and its adverse health effects vanishingly insignificant. In fact, they tell us "you can develop a disease working in any industry if you do not take care of your health" and that is why the workers of the "Uralasbest" facilities in Russia "preserved their health by living a healthy life"[4] and "illnesses never affect vigorous, active and cheerful people."[5] Finally, they ask: "Why have billions been spent attacking a minor health risk?"[6]

What are the facts about chrysotile asbestos? Do the statements of chrysotile apologists comport with the science or are they intrinsically biased?

Asbestos has been a commercially viable commodity since the late nineteenth century because of its many useful properties; principally its insulation properties, its weave ability, tensile strength, and suitability for use in binding composites. The main commercially viable types of asbestos are of two varieties: amphiboles and serpentines. The mineralogical makeup of the two is different in both their chemical and morphological states. The amphiboles contain more iron and tend to be solid straight spear-like fibers while the serpentines contain less iron and appear curly, are hollow, and split longitudinally. Because of these differences, the serpentine form (chrysotile) was the most useful and the most exploited type, making up over 95% of all asbestos used, historically.[7]

Some claim this high usage makes chrysotile chiefly responsible for the asbestos epidemic we are now experiencing.[8] Others have suggested that chrysotile can be used safely and even say it is not responsible for the diseases we see today.[9] One group goes as far as to say "Chrysotile Asbestos Saves Lives."[10] Many of the studies supporting these viewpoints are industry-sponsored – where economic interests collide with health facts. This is not new, as early as 1912, the Canadian Department of Labour denied that the health of Quebec's millers and miners was affected by exposure to chrysotile and this attitude continues today, even with evidence to the contrary.[11]

Innovative epidemiology has become a "pseudoscience" as practiced by some industry paid epidemiologists and differs little from the old "smoke and mirrors" trickery.

Pseudoscience and Brake Mechanics

A good example of this "pseudoscience" is the inappropriate use of "meta-analysis," a methodology originally used to assess controlled drug trials.* One such analysis sponsored by three automotive companies[13] concerned the causation of asbestos-related disease among "supposedly" chrysotile asbestos-exposed brake mechanics. In this analysis of mesothelioma, the authors ranked 11 studies into a scoring system. Only four studies fit into the highest ranked tier (I): "studies with the higher (above median) total score were included." Of these four studies, only one had a score meeting over 50% of the scoring criteria. Yet the authors concluded that: "the available epidemiological data show that employment as a motor vehicle mechanic does not increase the risk of developing mesothelioma."[14] This type of flawed reasoning, however, is not unique to this study; many industry-sponsored studies draw negative conclusions on less than adequate data. As Sven Hernberg, internationally known epidemiologist and former editor of the Scandinavian Journal of Work, Environment & Health, states: a truly negative study must (i) be large, (ii) be sensitive, and (iii) have well-documented exposure data.[15] The study by Goodman et al. fails to meet two of these issues: (ii) be sensitive and (iii) have well-documented exposure data.

Deception in India

A "Study of Health hazards/Environmental hazards resulting from use of Chrysotile variety of Asbestos in the country," sponsored by the Ministry of Chemicals and Fertilisers, India, and conducted by the National Institute of Occupational Health of Ahmedabad, India, is a prime example of how to mislead the untrained reader. By design, it lacks sufficient power to determine disease risk. This study which is claimed to be an epidemiological study of all segments of the asbestos industry is a cross-sectional medical study of an active workforce and not a true epidemiological evaluation. The study evaluates the workers by means of a questionnaire to obtain personal characteristics, occupational characteristics, and morbidity details and relies on lung function testing and radiological examinations using the ILO guidelines to determine disease manifestation. The study, described as "multifaceted," is essentially a segmented study with one or two factories (units) representing each industry segment. Included in one segment study (here and below meaning a study at a particular location) is some assessment of non-occupational exposure.

The first segment study evaluated an asbestos cement sheet-manufacturing unit in eastern India. Although 200 workers are included in the study, only 188 actually participated. The authors do not explain the fate of the 12 missing workers. As part of the study, workplace fiber concentrations

** Meta-analyses of observational studies can present inherent biases such as selection bias and other confounding biases. Meta-analysis is a technique, first envisioned for evaluating clinical studies, where combining results based on homogenous data would be less likely to suffer from biases found in observational cohort analysis. If the data relied upon for meta-analysis have flaws, such as confounders or methodological issues, then the outcome of the meta-analysis will also suffer from the impact of such flaws as will the conclusions reached.*

were determined; in these assessments, fibers greater than 5 μm in length and less than 3 μm in width having aspect ratios ≥ 3:1 were counted, using a Walton-Becket graticule at a magnification of 400×. Unfortunately, using this methodology to determine chrysotile content misses many of the chrysotile fibers themselves, thus underestimating the potential work exposures to chrysotile.

The workers examined in the cross-sectional medical study had no reported exposure to asbestos prior to their current employment; thus, the duration of employment represented the maximum time available for development of both progressive and latent asbestos diseases. Since 65% of the workforce had worked in the industry for less than 20 years, the (statistical) power of this study to detect the longer latent asbestos-related diseases such as lung cancer and mesothelioma is quite limited. In addition, by including almost 14% of the workforce with essentially no exposure (stores, laboratory, general pool and other departments) the power of the study is reduced and its ability to detect asbestos-related diseases limited still further. A far greater diminution of the power of the study arises from the choice of only active workers as the study population. Workers not able to work through illness and workers who had quit through ill-health were excluded; this effectively "dilutes" the study population, and thus reduces the significance of any findings connected with disease manifestation. Even with these severe limitations, it is significant that a clear dose-response relationship is evident for both abnormal pulmonary function and restrictive lung disease (the type of lung dysfunction most related to asbestosis): 40% of the long term workers (20+years) had abnormal pulmonary function and 25% had restrictive lung disease. While the authors suggest the restrictive and combined abnormalities were more prevalent in smokers than non-smokers, two issues remain unresolved: firstly, the interaction between smoking and asbestos-related lung disease is not addressed; and secondly, the occurrence of obstructive disease, which is more related to smoking than is restrictive lung disease, remains virtually unchanged as duration of work increases. This would indicate smoking may have played a very small role in the abnormalities observed and that exposure to asbestos was the more likely causative factor. The authors indicate that 107 workers had normal radiographs, 77 had normal features except for prominent bronchovascular markings (which are not explained), and four had radiographs suggestive of interstitial lung fibrosis, which was ruled out after High Resolution Computer Tomography (HRCT) of the thorax. Overall, this study, by design, is very limited in its ability to detect asbestos-related diseases of a non-malignant nature and essentially unable to evaluate the risk of longer-term asbestos-related malignant diseases such as lung cancers, mesotheliomas or gastro-intestinal cancers. In addition, worker exposures would be underestimated.

The second segment study evaluated an asbestos cement sheet-manufacturing unit of western India. This assessment used essentially the same study design as above; however, in this unit, the study population comprised only 60 active workers.

Work durations were much shorter here than in the eastern unit with 62% of the workforce having worked at the factory for 5 years or less, 35% for 6-10 years and only 3% for over 10 years. Since, as for workers in the first segment study, the duration of employment coincides with latency (time since onset of exposure), it would be even less likely here, than for that study, that non-malignant asbestos-related disease would be detected and be virtually impossible to detect any long-term malignant diseases. Since this study used the same type of environmental sampling as the first segment study, it also underestimates the true exposures to chrysotile asbestos. In summary, this second segment study is, by design and composition of the workforce, much less likely to detect any relevant asbestos-related diseases than the first segment study.

The third segment study, "Study of asbestos jointing material-manufacturing unit," examined, in the same fashion as the first two studies, 70 active workers. This study appears also to include workers not exposed in the manufacturing process (15% were described as cleaners; however, it is unclear whether persons employed for cleaning were actually exposed or not). Only about 1/3 of the active workforce had potential exposures dating back more than 10 years, thus severely limiting the possibility of detecting long-term asbestos-related diseases. Using the same sampling techniques as the first two segment studies the likelihood of underestimating the true exposures to chrysotile is great.

The fourth segment study was entitled: "A comparative study of asbestos workers, end-users and community in the vicinity of asbestos factory." Such comparisons are usually of very limited value as they can include members of the workforce under study within the comparison populations, thus resulting in double counting and making any differences between the exposed group of asbestos workers and the community or end-users less distinct. It is also possible that plant emissions drift to the community near the plant and those end-users can also experience exposure from this source in addition to that from asbestos-containing products. In summary, this segment study, by design, is unlikely to detect real differences between the asbestos workers and the two comparison groups.

The next segment study was entitled: "Study of asbestos brake-lining manufacturing unit." The active workforce consisted of 153 workers of which 32.7% had less than 10 years, 65.4% had 11-20 years and 1.9% had greater than 20 years work experience or latency. This study appears to have included 8.5% of the workforce not exposed to asbestos in the production process. Here again, the prevalence of low latencies in this active worker population would make any detection of asbestos-related disease unlikely and the environmental monitoring would likely underestimate exposures to chrysotile asbestos for the reasons given in the analysis of the first segment study.

The last segment study, "Study of asbestos pipe manufacturing unit," assessed 95 active workers. This population appears to include 24% of workers with little or no exposure to the production processes and 96% of the workers had less than 10 years work experience or latency. This study would have extremely limited ability to detect asbestos-related disease because of the short latencies in the active workforce. Once again, airborne asbestos levels would

be underestimated due to the limitations of the sampling methods.

Overall, a reading of this study by the untrained reader would seem to support the safety of using chrysotile asbestos. However, the methods used in the "Study of Health hazards/Environmental hazards resulting from use of the Chrysotile variety of Asbestos in the country," preclude the validity of any such conclusion. In fact, very little light is shed on the safety or otherwise of chrysotile use by this cross-sectional study because it focuses on active workforces. By their very nature such groups of workers are characterized by low latencies – particularly low in some of the workforces studied – so discovery of long-latent asbestos-related diseases is virtually impossible. In light of this fatal flaw and underestimation of exposures due to poor sampling methodology the study is revealed to be pure deception, an illusionist's trick aimed at obscuring the health effects of chrysotile.

Pure Chrysotile or the Old Shell Game

The majority of studies of asbestos exposures relate to mixed fiber types. As expressed by de Klerk and Musk: "arguments that chrysotile in its pure form does not cause mesothelioma and therefore can be safely used for certain products for which other substitutes perform worse are more theoretical than practical: firstly because it is almost never found in its pure form but is contaminated by tremolite (or even 'Balangeroite') and secondly because of its association with lung cancer."[16] Very few studies have considered pure chrysotile fiber exposures, because of the inherent contamination with amphibole asbestos.

However, when researchers report mesothelioma in those relatively few cohorts exposed to pure chrysotile, their findings are readily dismissed by proponents of chrysotile use, who miraculously "discover" contamination of the chrysotile, not identified by the study authors which, it is claimed, accounts for induction of the disease. This technique resembles the three shell game, where tricksters extract money from gullible players by inviting them to guess the location of an object placed beneath one of the shells. After shuffling the shells the trickster has no difficulty in fooling most players into choosing an empty shell. In the chrysotile apologist's version of the game all three "shells" conceal case studies and/or statements supporting chrysotile use: "chrysotile is safe to use"; "pure chrysotile does not cause mesothelioma"; "if mesothelioma has been found from exposure to pure chrysotile then 'obviously' the chrysotile was ***not*** pure." Depending upon the circumstances, those questioning the safety of chrysotile are persuaded to turn over the appropriate shell, since if all three strands of argument were revealed together the contradictions would be evident. This chicanery is designed to disguise the fact that it is the authenticity of studies supporting chrysotile safety that should be scrutinized not the alleged purity of exposure. If certain mixed exposure studies favored by chrysotile apologists are not finding mesotheliomas whereas studies on pure chrysotile are, then the methodologies of the group with negative findings should be regarded as suspect. However, it is expediency rather than truth that drives the continuing multi-national campaign to promote the sale of chrysotile asbestos, and which claims it is safe to use, even with its near universal contamination with amphiboles. The Chrysotile Institute asks "Why so much emotion" and proceeds to tell us that *"Today, if one says that asbestos kills, this person is only confirming his great ignorance of recent scientific studies... or has other motivations to say so."* [17]

Some reports claim that amphiboles are as much as 100 to 500 times more potent in inducing mesothelioma compared to chrysotile, but with the difference less clear for lung cancer.[18] On the other hand, much lower potency ratios have been reported: 2 to 4-fold in one study and 14 to 26-fold in another.[19] It is pertinent to note that none of the reviewed risk analyses concluded that chrysotile does not cause mesothelioma and most did not consider relative risks between the fiber types for induction of asbestosis and other cancers.

The real difference between the fiber types with regard to mesothelioma induction is hard to gauge because few, if any, of the cohorts analyzed were exposed to pure chrysotile or had sufficient latency to manifest this long latent disease; however, considering cohorts exposed to mainly chrysotile there does appear a difference between the amphiboles and chrysotile for the induction of mesothelioma.[20] Proponents of the "amphibole theory" rely heavily on the lower biopersistence of chrysotile compared to amphiboles, as evidenced by their choice of lung burden analysis to determine received asbestos dose and disease causation. When, chrysotile-exposed individuals are examined in this way some time after exposure and their lungs are found to be clear of observable chrysotile fibers the pro-chrysotile view is that they are at no more risk than the background population. Such reasoning misses the fact that the predominant fiber found in the pleural area, where the majority of mesotheliomas occur, is chrysotile.* To use lung burden as a parameter for determining causation of mesothelioma is unscientific. Why should a higher prevalence of chrysotile, approximately 30% greater than amphiboles, being the fiber type proximate to the tumor site be ignored as having a significant role in mesothelioma causation? In addition, results from analyses of cohorts having relatively low mortality due to the young age and short latency of the study population will lead to inappropriate calculations of risk. Selikoff et al. (1973) have shown that the proportion of a cohort dying from mesothelioma can actually change as the cohort ages, with a corresponding change of risk from low to high. In their analysis, they found the proportion of the cohort dying from mesothelioma increased 16-fold as total mortality advanced from 12% to 68%.[25]

Scientific evidence on cohorts where fiber counts have been quantified by using both phase contrast microscopy (PCM) and transmission electron microscopy (TEM) show that both asbestosis and lung cancer occur with frequencies independent of fiber type and that non-regulatory fibers, those less than 5 μm in length, also have a causative role.[26] These findings point again to the flawed logic of using the PCM methodology for cohort exposures or relying on lung burden analysis alone to determine body burden of asbestos exposures. In fact, PCM technology as well as SEM technology will miss chrysotile fibers in the lung because of inadequate resolution.†

** Mesotheliomas develop in the pleura, peritoneum and other mesothelial cells that form a monolayer mesothelium lining the serosal cavities and the organs contained within these cavities.[21] Chrysotile is a cause of cancer in the lung and migrates to the mesothelial linings of the body.[22] Since chrysotile is carcinogenic and is present in high concentrations in the mesothelial linings where the mesothelioma is induced, it is biologically plausible that it causes or contributes to cause mesothelioma. Fiber penetration can rearrange the cytoskeletal apparatus of the cell and this could indicate an interaction between the chrysotile fibers and the normal mitotic process, since giant multinucleated cells are formed. These studies indicate that chrysotile penetrates the cell, enters the nucleus and induces abnormal chromosome formations in dividing cells.[23] Some of these abnormalities include the deletion of the P53 gene.[24]*

†While PCM has been the international regulatory method for analysis, it is not able to detect thin diameter fibers [<0.2µm in diameter].

Threshold, No Threshold or What is This I See Before Me

Multiple governmental scientific agencies concur that there is no exposure threshold for asbestos, including chrysotile;[27] however, proponents for the continued use of chrysotile and those facing litigation stubbornly insist the authorities are wrong about chrysotile. In the most recent attempt to show this, an analysis by Pierce et al. (2008)[28] "funded almost entirely by Chrysler Corporation, Ford Motor Company, and General Motors Corporation" selected four papers with mixed exposures* to address the question: does chrysotile have a NOAEL (no observable adverse exposure level) for mesothelioma.† By using studies with mixed exposures, the most this analysis can show is that low exposures result in low rates of disease; something already established in the epidemiology literature.

The authors state that they reviewed 350 studies and selected cohort studies with the most power – longer follow-up, larger study population – for analysis. In estimating some exposures the authors rely upon a contract report submitted, but not endorsed by the EPA, for "best estimates of the fraction of amphiboles present."[29] This contract study made presumptions based on unsubstantiated scientific data. For example, assuming short fibers were inactive and thus considering only longer fibers; and presuming chrysotile less potent based on studies of cohorts possessing inadequate latency for the full extent of disease manifestation to be observable. This latter point was exactly what Selikoff and colleagues warned about earlier when discussing how mesothelioma risk estimates increase as study cohorts age.[30]

Pierce et al. could find only four studies out of the 350 they reviewed suitable for determining the NOAEL for mesothelioma from chrysotile. The studies selected were Lacquet et al., 1980; McDonald et al., 1984; Albin et al., 1990, and Piolatto et al., 1990.[31] These studies include 15 mesothelioma deaths.

The first study, by Lacquet et al., featured workers from Eternit NV (Belgium), a company that processed about 35,000 tonnes of chrysotile annually along with 3000 tonnes of crocidolite and 1000 tonnes of amosite. The mortality study group consisted of workers who had been employed at the factory for 12 months or more within a 15-year period (1963-1977). No latency analysis is given for the cohort members nor the one mesothelioma victim detected in the mortality study. The cohort did have a high incidence of asbestosis with 29 cases, of which seven died from the disease. Deaths from gastrointestinal cancer, a cause of death found in excess in multiple asbestos cohorts, were also in excess in this cohort, but the authors decided this excess was not asbestos-related due to lack of any relationship to fiber-years; the authors ignored the relationship to latency which they did not disclose. Due to the absence of any discussion of latency by the study authors and Pierce et al., no indication of a possible NOAEL for mesothelioma can be drawn from this study; as has been pointed out earlier, latency is a key factor affecting the (statistical) power of any risk assessment for mesothelioma. The study authors passed the occurrence of one mesothelioma as "almost certainly related to heavy exposure" with no other information given.

The study by McDonald et al., 1984, supported by a grant from the Quebec Asbestos Mining Association is also problematic if it is supposed to provide evidence for a NOAEL. Sixty-four percent of the cohort was still alive and the authors observed no mesotheliomas at the time of the study publication, an observation not unexpected given the earlier analysis by Selikoff on cohort aging.[32] Pierce et al., have slanted their mirrors to deceive the reader by selecting this study, which does not allow sufficient manifestation of latency to evaluate the extent of mesothelioma impact on the population studied.

The Albin et al, 1990 study found 12 mesotheliomas where chrysotile was the main type of asbestos used in conjunction with smaller amounts of both amosite and crocidolite. Pierce et al., have again slanted their mirrors and been exceptionally heavy with the blue smoke. They ruled that none of the observed 12 mesotheliomas was suitable for associating with a NOAEL because the information to do so was "not available," whatever that meant. However, they go ahead and obtain a relative risk (RR) of 1.9 (0.25-55.7) which is insignificant and conclude that 15 fiber/cc-years (f/cc-yrs) with a mean of 3.1 f/cc-yrs and a median of 1.4 f/cc-yrs is the NOAEL.

The last study selected is by Piolatto et al, 1990 and is of a chrysotile mine in Balangero, northern Italy where contamination of 0.2 – 0.5% balangeroite occurs. The authors found two mesotheliomas of which Pierce et al. used one to associate with a NOAEL of greater than 400 f/cc-yrs, the highest NOAEL of the study. It should be noted that only 40% of the studied cohort were dead at the time of publication leaving 60% alive. Thus, the significance of the two mesotheliomas, which Piolatto et al. associated with a moderate excess of mesothelioma, is lost to the reader of the Pierce et al paper, because with 60% of the population under study still alive only those with the highest exposures or longest latency would be expected to have developed mesothelioma by the time of the study. Relying on the smoke and mirrors, Pierce et al. hope that the reader will be of the impression that this same trend will hold true after the next 60% of the cohort die, which the astute reader will recognize as pure speculation.

In attempting to arrive at NOAEL for chrysotile induced mesothelioma, Pierce et al. have used data from studies with multiple deficiencies: inadequate cohort aging and latency, and questionable exposures both with regard to fiber types and levels. Where, the data do not meet the desired pattern the slanted mirrors come into play with the smoke of statistical manipulations hopefully confusing the reader into thinking the authors' arguments have some substance.

**This is because chrysotile only exposures do not generally occur, thus epidemiology studies of "so called" pure chrysotile cohorts are usually found to have questionable exposure to amphibole contamination.*

†Their selection criteria were:
1. Outcomes of interest including lung cancer (variously identified as "lung cancer," "respiratory cancer," "malignant respiratory neoplasms" or "malignant neoplasms of the lung") and/or mesothelioma.
2. The cohort was predominantly exposed to chrysotile asbestos (less than 10% of the potential asbestos exposures involved amphiboles).
3. There were no other known occupational exposures to respiratory carcinogens.
4. Relative risk or relative mortality estimates were provided or could be calculated and stratified by cumulative chrysotile exposure.
5. Cumulative chrysotile exposures were stratified into two or more exposure levels by the authors.

Illusion, Dissolution, and Confusion – the Weapons of those that Claim Chrysotile is Safe

Asbestos industry propagandists[33] rely on older reports by international health agencies to support their arguments; they fail to report that these agencies have changed their positions as newer science developed, the very charge they level at scientists who support a ban for all forms of asbestos. For example, the Chrysotile Institute uses a 1989 World Health Organization report to support a different standard for chrysotile while a later 1998 joint report of the World Health Organization, the International Labour Organization, and the United Nations Environmental Programme states "No threshold has been identified for carcinogenic risks." The most recent statement of the WHO in 2006 is categorical: ". . . there is no evidence for a threshold for the carcinogenic effect of asbestos. . ." and ". . . the most efficient way to eliminate asbestos-related diseases is to stop the use of all types of asbestos; . . ." [34]

While there is a consensus in independently-authored scientific papers that all forms of asbestos cause asbestosis, lung cancer and mesothelioma, the chrysotile lobby continues to disseminate misleading "information" to bolster its assertion that "low risk" chrysotile can be used safely under "controlled conditions" and should not be banned.[35] Supporting this position, pro-chrysotile proponents cite data out of context and without references, something avoided in this paper, which provides the reader with citations for supporting statements. The Chrysotile Institute claims that an international consensus panel and many new studies confirm that chrysotile fiber is definitely less dangerous than other types of asbestos. It described the conclusions of the consensus panel as follows: "A group of scientists mandated by the Environmental Protection Agency (EPA) unanimously agreed that available studies on epidemiology indicate that the carcinogenic potential of amphibole fibres was one hundred times (100×) higher than that for chrysotile fibres." This statement is not true.

First, the EPA did not mandate this group of scientists; this was a contract report where the contractor independently selected the scientists, not the EPA. Second, this was not a consensus report as one can see when reading the independent scientists' comments within the body of the report. Third, this was a report to the EPA that has never been sanctioned by the EPA, nor adopted as official policy; nor has the EPA changed any asbestos policies because of this contract report.[36] As we have seen, this is the same report relied upon to substantiate assumptions made by Pierce et al. in their "no-effect chrysotile paper."

The source of "new scientific data" invoked by the Chrysotile Institute remains a mystery; the "important new study" which it says clearly confirms "the difference, from the epidemiological point of view, between chrysotile and amphiboles" is never identified. Most recently and after publication of the Chrysotile Institute report, a new epidemiological paper, by the U.S. National Institute for Occupational Safety and Health (NIOSH), has shown no difference in potency between chrysotile and amphiboles for inducing asbestosis or lung cancers.[37]

Using current developments to its own ends, the Chrysotile Institute is claiming a major shift in the NIOSH position on the safety of asbestos, citing a statement by the NIOSH Director in Congressional testimony that "the current legislation was the most appropriate to protect workers." The Chrysotile Institute does not realize that legislation is different from regulation and that NIOSH still has the same recommendation as first articulated in 1976, that a ban on asbestos is the only way to eliminate asbestos-related diseases.[38] Furthermore, this position is in fact supported by current legislation: the Occupational Safety and Health Act, 1970.

The chrysotile lobby relies on misinterpretations, false claims and undocumented statements to advance its global propaganda campaign for the continued use of chrysotile asbestos. While its smoke and mirrors strategy or its shell game may be suitable for illusionists and entertainers, the obfuscation of scientific truth resulting from such practices can have grave consequences when evaluating the risk of disease and death for those exposed to the hazards of asbestos. In a profit-driven frenzy, the asbestos alchemists peddle their toxic wares to ill-informed governments and consumers. But blow away their smoke, remove their mirrors, turn over all three shells and the truth emerges for all to see: asbestos is deadly, there is no safe concentration of exposure identified, industry propaganda is unreliable and the continued use of chrysotile is unconscionable.