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Exposing the "Myth" of ABC, "Anything But Chrysotile": A Critique of the Canadian Asbestos Mining Industry and McGill University Chrysotile Studies

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Background *Beginning in the 1930s, the Canadian asbestos industry created and advanced the idea that chrysotile asbestos is safer than asbestos of other fiber types.*
Methods *We critically evaluate published and unpublished studies funded by the Quebec Asbestos Mining Association (QAMA) and performed by researchers at McGill University.*
Results *QAMA-funded researchers put forth several myths purporting that Quebec-mined chrysotile was harmless, and contended that the contamination of chrysotile with oils, tremolite, or crocidolite was the source of occupational health risk. In addition, QAMA-funded researchers manipulated data and used unsound sampling and analysis techniques to back up their contention that chrysotile was "essentially innocuous."*
Conclusions *These studies were used to promote the marketing and sales of asbestos, and have had a substantial effect on policy and occupational health litigation. Asbestos manufacturing companies and the Canadian government continue to use them to promote the use of asbestos in Europe and in developing countries.* Am. J. Ind. Med. 44:540–557, 2003. © *2003 Wiley-Liss, Inc.*

KEY WORDS: asbestos; chrysotile; corruption; crocidolite; QAMA; McGill; tremolite

INTRODUCTION

Chrysotile asbestos was first mined in Canada in the late 1870s. A fierce struggle between the asbestos industry in Canada, England and South Africa and medical researchers began in the early 1930s and has been documented in the professional literature and in the courts and continues to the present [Hardy and Egilman, 1991; Liddell, 1997; Nicholson, 1997; Egilman et al., 1998; Egilman and Reinert, 2000].

QAMA's first efforts to mislead the medical community about the carcinogenic effects of asbestos exposure were published in 1958 [Braun and Truan, 1958]. The individually numbered drafts of the study results circulated to QAMA members reported, "[t]he number of lung cancer deaths combined with asbestosis is larger than would be expected in each cohort and in the combined cohorts. This difference is significant at the 95% level using the chi-square test of significance." At the request of QAMA, the researchers manipulated the denominator and published, "On the basis of what are believed to be complete and reliable data, *it seems fair to conclude that the asbestos miners in the province of Quebec do not have a significantly higher death rate from lung cancer than do comparable segments of the general population*" (emphasis added) [Braun and Truan, 1958].

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In 1964, Irving J. Selikoff, who was concerned with the inadequate response to the public health dangers of asbestos, organized a New York Academy of Sciences (NYAS) conference devoted to understanding the physiological effects of the mineral [Selikoff, 1965]. This conference firmly established the carcinogenicity and other health hazards of exposure to asbestos. The information generated at the conference was widely disseminated in the press, threatening the industry's position domestically and in the global market.

In response, Canadian mining companies, acting through the Quebec Asbestos Mining Association (QAMA), renewed their connection with McGill University [Wright, 1926; Asbestos Textile Institute, 1965; Institute of Occupational and Environmental Health, 1966] to develop contrary scientific evidence, hoping to sow doubt about the toxicity of various asbestos fiber types. As a result of the NYAS conference, the carcinogenicity of asbestos was irrefutable. QAMA knew that existing research revealed the dangers of asbestos, and sought to develop ''counter propaganda'' to the work of Selikoff and others [QAMA, 1967]. Its members looked to the tobacco industry as a model for their own research, noting that that industry ''launched its own program and it now knows where it stands'' [QAMA, 1965]. Accordingly, QAMA developed *Anything But Chrysotile* (ABC) arguments in the hope that it could maintain or expand market share for its form of asbestos and avoid liability. The ABC argument implicates various substances *other* than Canadian chrysotile as the cause for asbestos's toxicity.

QAMA has provided funding to a research unit at McGill University for the past three decades which has promulgated several different ABC theories. Most recently these have been used in an attempt to mislead a variety of international panels on the true risks of exposure to chrysotile asbestos [Castleman, 2001, 2002]. The McGill researchers frequently appear or and sometimes have been hired as ostensibly objective analysts [U.S. Environmental Protection Agency, 2001; Eastern Research Group, Inc, 2003].

Beyond promoting the marketing and sale of asbestos, these studies have had a tremendous effect on litigation from the 1960s onwards. Lawyers for asbestos-manufacturing corporations have used these studies to assert that the causal link between asbestos exposure and cancer was unclear and hypothetical, thus effectively denying injured workers and their dependents compensation for their illness [Georgia-Pacific, 2003].

We analyze and discuss three fallacies that underlie the ABC arguments. The first is that organic and synthetic oil contamination—and not the chrysotile itself—is the cause of lung cancer and primary malignant mesothelial tumors in miners and other people who work with asbestos [Commins and Gibbs, 1969; Gibbs and Hui, 1971]. The second is that crocidolite, allegedly imported from Australia and used at a factory adjacent to one of the mines, caused an increase in mesothelioma in the mine workers [McDonald and McDonald, 1978, 1980]. A third fallacy put forth by the industry is that tremolite was the only culprit, and that current commercial chrysotile is ''innocuous'' because contemporary mining practices either avoid it entirely or remove it during processing [Case, 2001a]. After examining QAMA's ''ABC''construct, we review the methodology that QAMA funded epidemiologists and scientists used to support various industry claims. These include ignoring pertinent dose-response data and mis-estimating dose through use of inadequate and out-dated sampling techniques; and ignoring or misinterpreting worker interview data [McDonald et al., 1970; Gibbs and LaChance, 1972, 1974; Liddell et al., 1984].

INDUSTRY MYTHS

Organic and Synthetic Oil Contamination

In 1965, Harrington and Roe reported that naturally occurring as well as contaminating organic compounds may play a key role in the carcinogenic nature of asbestos [Harrington, 1965; Harrington and Roe, 1965]. From the industry's perspective, this was an ideal way to obfuscate the notion that its product, chrysotile, was deadly. If this ''mystery'' contaminant could be identified and the chrysotile ''cleaned,'' the product and profits could be saved. QAMA supported further studies, both by providing funding and in helping with the collection of samples and other data [Gibbs, 1969; Brodeur, 1974].

Further investigating Harrington and Roe's claims, Graham Gibbs and others at McGill University examined the theory of organic contaminants of chrysotile [Commins and Gibbs, 1969]. Gibbs raised the question of whether the compounds may: act as carcinogens in and of themselves; enhance the carcinogenic activity of other substances, such as trace metals, asbestos itself, or associated oils; inhibit the action of carcinogens present in the fiber; or have no influence on the biological action whatsoever [Gibbs, 1969]. Through their studies, the researchers found that the organic contaminants were implicated in the ''biological action'' of chrysotile asbestos products, suggesting that these contaminants, and not the chrysotile itself, were the cause of cancer in workers [Gibbs and Hui, 1971].

They determined that the long-chained alkanes found in asbestos products resulted from three possible sources: hydrocarbons occurring naturally in the ore body; contamination from the mining and milling process; and contamination from shipping, manufacturing, and utilization processes [Gibbs and Hui, 1971].

Polyethylene bags and asbestos dryers were allegedly the primary culprits in this oil contamination [Gibbs, 1969]. However, Gibbs recognized the lack of mesotheliomas

occurring in animals exposed to organic compounds, and stressed the need for further investigation into the alleged link between these compounds and lung cancer. Emphasis was placed on the importance of assessing the differences in organic content in the types of asbestos to which workers were exposed, but in the end there was no evidence that organic contaminants accounted for increased cancer rates. Wagner and Berry [1969] published a report demonstrating that removal of organic contaminants from asbestos did not decrease, and in fact possibly *increased*, the ability of the fiber to cause mesotheliomas in rats. Therefore, the argument that oil contaminants were the chief cause of the carcinogenic effects of chrysotile exposure was no longer plausible, and had to be rejected in favor of other explanations as to why chrysotile was supposedly safe.

Crocidolite

Studies published in the late 1970s by J.C. McDonald and colleagues offered yet another explanation for the non-carcinogenicity of chrysotile [McDonald and McDonald, 1977, 1978; McDonald, 1978]. They claimed that most of the mesothelioma cases from the mines in Asbestos, Quebec occurred as a result of exposure to *crocidolite* (an amphibole also known as *fibrous riebeckite*), which was allegedly imported from Australia for use in gas mask manufacturing at the factory located ''adjacent to'' the Johns Manville Jeffrey mine during World War II. The McGill researchers postulated that this use of crocidolite in filter pad manufacturing from 1939–1941 accounted for increased mesothelioma and lung cancer rates among miners and millers at the Jeffrey mine, even though none of them had ever worked in the factory [McDonald and McDonald, 1980]. It is interesting to note that there are no citations of interviews with workers or correspondence that could prove imported crocidolite *was* ever used for this purpose at the mine location. The head of shipping and receiving during the relevant time period was also unaware of any crocidolite used in the factory (A.R. Carr, unpublished communication). British regulations mandated the use of either chrysotile or crocidolite for World War II gas mask filters. It is unlikely that QAMA members involved in the production of gas masks would import crocidolite from Australia, past Japanese submarines and battleships, past other filter factories in Ontario that used crocidolite, to fill gas mask filters at a factory adjacent to the largest chrysotile mine in the world. Furthermore, Begin et al. [1992] reported that mesothelioma rates were not as elevated as one would expect if crocidolite was used in gas-mask manufacturing at the Jeffrey mine in the town of Asbestos [Begin et al., 1992]. He revealed his own doubts about the presence of amphiboles at the factory, stating that amphiboles ''*may*'' have been used there and noted that there were no cases of mesothelioma in other departments that were nearby, such as quality control. Begin et al. [1992] and Dufresne et al. [1995] reviewed the detailed job histories of all twenty workers who contracted mesothelioma and confirmed that *none* of them had worked during World War II in the preparation of materials for the fabrication of gas masks as previously noted by McDonald, nor had any of them been exposed to crocidolite in the factory or mill during the 2-year period.

In October 2000, at a presentation to a group of asbestos defense lawyers, Bruce Case reported that the factory was located ''immediately adjacent'' to the mine and mill. He stated, ''not only had some of the men worked there, but most has [*sic*] to pass through the building as it contains common areas'' [Case, 2000]. However, at a deposition a year later, Dr. Case stated that he did not know where the Jeffrey factory was located [Case 2001a]. From 1928 to 1972, the factory at Jeffrey was located across an open pit mine, about a mile from the entrance to the mine and the mill [Tourist Bureau, 2002]. It is hardly believable that miners would ever walk through the old factory as a part of their daily routine; it would include a mile descent, and a 2–3 mile walk. According to F. Spertini, the mine geologist, there were no common areas and two separate roads led from the town to the factory and mine entrances. (F. Spertini, unpublished communication).

Regardless of its source, the QAMA–McGill scientists found crocidolite in the lungs of 71% of miners from Asbestos [Case, 1998]. Initially this finding was only reported for miners at the Jeffrey mine but later the researchers found lower concentrations of crocidolite in 13% of miners from Thetford [Nayebzadeh et al., 2001]. Nayebzadeh and colleagues reasserted that the crocidolite in the lungs of Asbestos miners came from exposure to imported crocidolite used in the gas mask manufacturing. However, they overlooked the fact that if crocidolite was used, it was only during the time from 1939 to 1941, and about half of the workers in their study began working after 1941. They offered no explanation for the crocidolite in the Thetford miners' lungs. The most likely source of this crocidolite was the local mine ore, not imported fibers. Two geological surveys found that mines from both areas contained blue fibrous riebeckite, otherwise known as crocidolite [De, 1961; Hebert, 1980].

How did the McGill researchers miss this information on crocidolite contamination of ore? By 1961, QAMA had been given De's doctoral thesis showing crocidolite in Quebec mines. The McGill research team cited that thesis in 1972 [Gibbs and LaChance, 1972]. However, while noting that De found actinolite in the Canadian mines, they omitted any mention of his *twelve-page discussion* of the crocidolite contamination of the mine ore body. Over two decades later in 1995, Dufresne and others extracted and published considerable information from the De's thesis (See Appendix, Note 1). However, they failed to mention that De had written extensively on the location as well as

performed a mineralogic and chemical analysis of the crocidolite he found in the Eastern Townships asbestos deposits. It is difficult to believe this omission occurred in error, especially since De's study was cited in 1972, 1986, and 2001 in papers that dealt with the issue of crocidolite in the miners' lungs [Gibbs, 1972; Dufresne et al., 1995; Nayebzadeh et al., 2001]. Moreover, in 2001, some of the same authors who had cited De's thesis 6 years earlier actually make a *completely contradictory* claim: ''Jones et al. confirmed amosite and crocidolite are not present in the rocks mined in Asbestos region'' [Nayebzadeh et al., 2001; Williams-Jones et al., 2001]. Moreover, one of the co-authors of both of these papers was aware of the presence of crocidolite in the Asbestos region [Hebert, 1980; C. Normand, unpublished communicaton].

If the QAMA-funded researchers reported on the presence of crocidolite in the ore, it would have cast a pall over any assertion that Canadian asbestos was ''innocuous.'' Even though crocidolite contamination would have been a useful scapegoat to cloak the carcinogenicity of chrysotile asbestos and support the scientific argument that chrysotile did not cause mesothelioma, it would not have served QAMA's need to show that the asbestos they sold was safe. Crocidolite could not be removed from the ore or final product and this fact would have completely undermined the argument that Canadian asbestos was ''innocuous.''

Tremolite Contamination

More recently, the QAMA-funded researchers have shifted the onus of increased mesothelioma risk to contamination of the ore with tremolite. In this instance, they argue that tremolite contamination of chrysotile—and not exposure to chrysotile per se—is the cause of asbestos-related cancers in miners and millers. In 1985, Peto and Doll reviewed this argument and deemed it to be of academic interest only because tremolite was found to contaminate commercial chrysotile fibers, which could be found in the end product [Doll and Peto, 1995]. In response, QAMA needed to give the impression that tremolite-free chrysotile could be safely mined and sold.

Central-peripheral contention

The ''high-low concentration–central-peripheral mine location'' argument was first proposed by J. Corbett McDonald and Allison McDonald in a 1995 Letter to the Editor in *Science*. They proposed that exposure to tremolite, and not chrysotile, was the likely cause of mesothelioma cases present in miners and millers who worked in Thetford mines in Quebec [McDonald and McDonald, 1995]. It was the first in a series of articles in which the McDonalds and other QAMA-funded researchers set forth the argument that there were central (high tremolite) and peripheral (low tremolite) mines in Thetford (See Note 2). They have subsequently presented data showing that all but one of the mesothelioma cases occurred in miners who had worked in ''central'' (high tremolite) mines [Liddell et al., 1997, 1998; McDonald and McDonald, 1997; McDonald et al., 1997, 1999; McDonald, 1998a,b; Vacek, 1998; Nayebzadeh et al., 2001].

McDonald and McDonald [1995] cited a 1989 article by Sebastien and colleagues as the source of data indicating a wide disparity in tremolite contamination levels between central and peripheral mines in the town of Thetford Mines. However, the Sebastien study did not include, categorize, or evaluate the relationship between tremolite lung levels and mine location [Sebastien et al., 1989]. Dr. Sebastien has confirmed that his report did not examine this issue, and he was unaware of any other studies, published or unpublished, that recorded any lung fiber measurements by mine location (P. Sebastien, unpublished communication). McDonald and McDonald failed to specify the exact location or names of the area A and B mines in any published or unpublished study. The authors provided no comparative data on age, work years, date of first exposure, job, underlying disease, or time spent working in other mines [McDonald and McDonald, 1995]. Any of these variables could explain the comparative data, and in their 1997 publication, the McDonalds noted that most of the peripheral mines ''had started so recently that there were inadequate periods of latency'' for mesothelioma to occur in workers at ''peripheral'' mines [McDonald and McDonald, 1997]. As chrysotile is cleared from the lung over time, this fact alone could explain the higher chrysotile/tremolite ratios in workers from ''peripheral'' mines [McDonald, 1994].

If the findings are not confounded by any of these factors, then the accuracy of the dose estimates used in the entire series of epidemiologic studies published on the Canadian miners must be questioned. The study by Sebastien and his colleagues used comparative chrysotile/tremolite ratios between mine workers and textile workers to justify their dose estimates [Sebastien et al., 1989]. If the mine tremolite/chrysotile ratios used for this comparison came from two different mine worker exposure categories, then the analysis is fatally flawed.

In 1997, the McDonalds published a detailed explanation of the ''tremolite hypothesis,'' claiming that they had ''re-analyzed'' the data found in Sebastien et al. [1989]. However, they did not present any data or analysis of lung tremolite levels [McDonald and McDonald, 1997]. The authors presented mesothelioma rates in terms of ''central and peripheral'' locations, but they again did not provide any specific information on the exact mine locations [McDonald and McDonald, 1997]. Gibbs categorized, at most, nine mines at Thetford Mines, and he did not classify them as central and peripheral, but McDonald and his colleagues refer to 15 mines in another paper and 21 mines in still

another [Gibbs, 1979; McDonald and McDonald, 1997; Nayebzadeh et al., 2001]. Of the ten studies that have based their conclusions on the distinction between central and peripheral, the 1995 letter to *Science* is the only publication that provides any foundation for this proposition [McDonald and McDonald, 1995]. Four other papers, all of which are co-authored by the McDonalds, erroneously cited Sebastien et al. [1989], and not the *Science* letter, as the source for the data on different tremolite levels, while five other papers failed to provide any basis for this conclusion [McDonald and McDonald, 1995, 1997; Liddell et al., 1997, 1998; McDonald et al., 1997, 1999; McDonald, 1998a,b; Vacek, 1998; Nayebzadeh et al., 2001].

The assertion that the Thetford mines have varying tremolite contamination levels conflicts with other published data on this topic. McDonald et al. [1997] cited Sebastien et al. [1989] and Gibbs [1979] as support for the central-peripheral theory. However, Gibbs, a McGill colleague, published contrary and confusing information on this inter-mine difference [Gibbs, 1979]. He concluded that differences in tremolite concentration, if they existed, could not explain differing disease rates [Gibbs, 1972, 1979]. He also noted that the mines were all part of the ''same ore body'' and there was no geological evidence showing any difference between the mines in Thetford Mines [Gibbs, 1979].

In pursuit of the missing data, we contacted J.C. McDonald and Janet Hughes (co-author of a post-1995 study). Dr. Hughes did not know which mines were central and which were peripheral (J.M. Hughes, unpublished communication) and McDonald has yet to respond to our inquiries. Case, a co-author of another paper with the McDonalds that relied on the distinction between high and low tremolite mines, also stated that he did not know which mines were central and which were peripheral [Case, 2001b].

It is hoped that it is only the citation that needs to be rectified. However, this incorrect citation, which was overlooked by the reviewers of ten separate articles in five different journals, is further evidence of shortcomings in and the importance of the peer review process [Egilman and Reinert, 2000]. The perpetuation of this error has cast the mantle of sound science upon the tremolite hypothesis. In 1997, McDonald and colleagues contended that, based on the low disease rates in peripheral mines, ''the explanation [for the high rate of mesothelioma] is mineralogical'' [McDonald et al., 1997]. Liddell implied that the medical community had ''generally accepted'' this high-low distinction when he asserted, without citation, that ''. . .contamination of the chrysotile by fibrous tremolite *was known* to be much greater in the central than in the peripheral area,'' going on to conclude that ''. . .it is now clear for all practical purposes that [the excess incidence of mesothelioma] was confined to the central area there (Emphasis added)'' [Liddell et al., 1998]. More recently, the Canadian researchers have used this evidence before the World Trade Organization and in U.S. tort litigation to buttress the proposition that chrysotile is not a cause of mesothelioma [World Trade Organization, 2000]. Medical literature based on the central-peripheral tremolite distinction continues to be used as an argument to promote the sale of Canadian chrysotile in the developing world [Browne, 2000].

Tremolite-free argument

The second theory put forth by the QAMA-funded McGill researchers is that there are mere trace amounts of tremolite in the asbestos mined today because it is avoided during mining or removed during the milling process. Kevin Browne presented these arguments at the International Seminar on Safety in the Use of Chrysotile Asbestos held in Havana, Cuba; the transcript bears the Asbestos Institute logo and is available at http://www.chrysotile.com/en/hltsfty/browne.htm (See Note 3). He states that all Canadian mine-related cases of mesothelioma were related to tremolite contamination. However in 2001, when one of the authors (DE) asked, Browne did not know whether or not Black Lake, the only operating mine at the time, was a central or peripheral mine. After ''checking'' a few days later, he reported that it was a peripheral Thetford mine and therefore had low tremolite contamination (K. Browne, unpublished communication). The Black Lake mine is not even in Thetford; it is 6 miles away in the Town of Black Lake.

The McGill researchers use four pieces of evidence to support the ''Tremolite-Free Argument'' for Canadian chrysotile production. A description of the mining process and the route tremolite travels to get into the final product will highlight each argument used by the researchers, and demonstrate the inherent flaws of each. In reality, the mining and milling process actually *adds* tremolite to chrysotile. The process is described by Spertini (See Note 4).

While it is clear from this process that most of the tremolite is bagged with the short fibers, the Canadian asbestos industry has presented several arguments in the attempt to show that current chrysotile product is tremolite-free. For example, industry advocates note that Frank and colleagues were unable to find tremolite in any UICC-sample chrysotile, which was prepared in the late 1960s [Frank et al., 1998]. However, at that time, it was undisputed that the chrysotile product contained tremolite—as it has been found in the mines, as well as in the lungs of miners and textile workers who used Canadian chrysotile [Dufresne et al., 1995]. The most likely explanation for Frank's tremolite-free samples is that the samples were taken from crude 1 and 2 ore. Historically, the highest-grade chrysotile, crude 1 and 2, did not go through the usual milling process. Miners hand-picked this product from veins of pure chrysotile and it was not milled at the mine (F. Spertini, unpublished communication).

Another myth is that tremolite is removed from the chrysotile in ''processing.'' Bruce Case has claimed that this process takes place at Canada's last and largest operating mine, Black Lake [Case, 2001a]. In fact, Case himself has admitted that he ''do[es] not know how the milling process [works]'' or, indeed, the basis for claiming that tremolite can somehow be separated from the chrysotile fibers:

> I don't know why it is that the miners' lungs, for example, contained so much tremolite whereas the end product users' lungs contained so much less tremolite. Something happens in the processing to remove the tremolite. [W]e can talk about water filtration, we can talk about screening, we can talk about milling but the exact mechanism by which it happens or it occurs I don't know [Case, 2001a].

Contrary to Case's testimony, we cannot ''talk about'' water filtration at Black Lake. That is because the mine is located at the bottom of a waterless lake. It took 4 years to drain the lake and at the time it was viewed as an engineering marvel. There is no water in the milling process, either. No one who has ever visited or reviewed the mine or milling process at Black Lake would ever make this mistake. Case's testimony makes it clear that neither QAMA nor its researchers had sufficient evidence or knowledge to make their claim that tremolite could be removed from the supposed less-dangerous chrysotile.

McGill researchers continue to insist in the literature that there is no tremolite in today's chrysotile. For example, Williams-Jones and colleagues claim that ''Amphibole-free chrysotile can be produced from the Jeffrey mine, and other chrysotile mines, provided that appropriate measures are taken to avoid contamination of the ores'' [Williams-Jones et al., 2001]. While this may be true, any inference that current or past production has utilized these ''appropriate measures'' is incorrect and misleading. Despite the results of this study, the newest shaft at the Jeffrey mine is located in one of the most heavily tremolite-contaminated parts of the mine (C. Normand, unpublished communication).

As discussed earlier, tremolite and other fibers released during the mining process end up in the bags of end product. Therefore, it is likely that current and past shipments from the Jeffrey mine, as well as from all other Quebec mines, were contaminated with tremolite, crocidolite, and amosite [De, 1961; Gibbs, 1972]. This conflicts with information dispensed to the public under The Asbestos Institute logo [Browne, 2000]. The Asbestos Institute does not mention the fact that crocidolite and amosite are also present in the mine ore, and claims that tremolite is removed through various mining and milling processes [De, 1961; Browne, 2000]. This is simply untrue. In fact, other McGill researchers have shown that a substantial quantity of the mined tremolite ends up in the lungs of textile workers [Gibbs, 1972; Sebastien et al., 1989]. Clearly, the idea that current shipments of asbestos are amphibole-free is absolutely false, and one that continues to pose a grave threat to the health of mine workers. It remains to be seen whether the mine operators will implement Williams-Jones and colleagues' suggested survey techniques in the future.

QAMA'S FLAWED METHODOLOGY

The Textile Mystery: Another ABC

The QAMA-funded McGill researchers have claimed that, even if fiber mined from Canada causes mesothelioma and lung cancer, studies show that the dose required to induce these diseases is so high that there is no practical risk to current workers. Yet studies of textile workers exposed to the same fiber have revealed that the ''slope of the exposure response lines for lung cancer in the textile industry was some fifty times steeper than that observed in Quebec chrysotile miners and millers. . .'' [McDonald, 1998b]. They have dubbed this variation the ''textile mystery'' and have failed to provide any kind of explanation for it [McDonald, 1998b]. Ignoring the textile dose response data, the McGill researchers have vociferously opposed the French chrysotile ban before the WTO. In testimony at the WTO hearings, chrysotile asbestos product manufacturers have claimed that their chrysotile products did not in any way contribute to workers' asbestos-caused mesotheliomas and lung cancers (B.I. Castleman, unpublished communication). J.C. McDonald sat with the Canadian legal team, separate from all other experts, and presented part of Canada's argument at the appeal of the WTO ruling on the French ban (B.I. Castleman, unpublished communication).

McDonald summarily dismissed a number of possible explanations for this apparent disparity between mining and textile dose responses, including miscalculations in dose measurement or errors that occurred when the Canadian researchers converted particle to fiber counts. He asserted, ''There is *nothing* to suggest that the estimates of cumulative exposures in the relevant cohorts were seriously in error although questions of peak exposures and fibre size distributions in ambient air have not been examined (Emphasis added)'' [McDonald, 1998b]. However, the McGill researchers evaluated the quality of the dose estimates quite differently when they first reported them [Gibbs and LaChance, 1972, 1974]. While McDonald flatly stated that errors in measurement or actual differences between these populations could not explain more than a tenfold difference in the dose-response slope, Gibbs [1972] and Gibbs and LaChance [1972] reported that dose estimates alone differ by more than one-hundred-fold for the same job, both within the same mine and between mines. Gibbs and LaChance [1974] noted ''If membrane filter and midget impinger counts were

considered by work area, it was clear that the ratios of the two in some mines were of a different order [of magnitude] from those in others where the *same process was employed*. . .(Emphasis added)'' [Gibbs and LaChance, 1974].

Conversion from particle to fiber counts compounded the dose estimate problem: ''Though only 87 pairs of samples were collected in this pilot investigation, these were sufficient to demonstrate that no single conversion factor could be applied to all mines or to all work areas *within a mine* (Emphasis added)'' [Gibbs and LaChance, 1974]. In low fiber specimens, which accounted for nearly one-third of the samples, the QAMA–McGill researchers found that particle counts were *inversely* correlated with fiber counts. That is, the higher the particle counts, the lower the fiber exposure [Gibbs and LaChance, 1974]. They concluded, ''Thus, the conversion of dust-disease relationships for the Quebec mining and milling industry to fiber-disease relationships does not seem possible at the present time'' [Gibbs and LaChance, 1974]. However, later ignoring their own data and recommendations, McDonald et al. [1980b] converted from particle to fiber dose estimates. It appears that incorrect dose estimates and a systematic bias against diagnosing asbestos-related disease in the Canadian asbestos mining region may explain the textile mystery.

Mis-Estimating the Dose: Particles Are Not Fibers

Gibbs and Hui [1971] used available dose measurements from 1949 to 1966, which were measures of ''total'' particles collected by midget impinger. This method cannot distinguish fibers from other dust particles, such as silica and other ''non-toxic'' dusts [Egilman and Reinert, 1996]. Only *fibers*, which may or may not be captured in the total particle measurements, cause disease. In reality, it is difficult to make accurate estimates of the actual exposure of the Canadian miners and millers during that period. However, the estimates that have been made indicate that the miners were, in all likelihood, exposed to fewer fibers than were the South Carolina textile workers—rather than the other way around. The dose estimates from the QAMA–McGill mine studies are wholly inaccurate. Based on the comparative mine/textile risk ratio, it seems clear that they have systematically overestimated the actual exposures.

As early as 1951, QAMA researchers realized that the accurate calculation of dose estimates for Quebec miners was impossible. While the problem of dose conversion exists in all studies that are based on historical particle count data, this problem was exacerbated in the mine studies due to the large number of locations and jobs involved. As Vorwald, a consultant to the QAMA, wrote to Cartier, the director of the QAMA industrial disease clinic in Thetford mines, the data did not exist (See Note 5). Cartier later served as a consultant to the QAMA.

What was clearly ''impossible'' in 1951 became the dose reconstruction of 1971 [Gibbs and Hui, 1971]. Dr. McDonald was aware of this problem by no later than April 23, 1969. During the discussion period following his chairing of a session on asbestos measurement techniques, which was highly critical of the midget impinger method, he asked, ''Can an *inaccurate* instrument like the midget impinger (MI), give an accurate result?'' [Shapiro, 1970]. Despite the MI's drawbacks, the QAMA researchers have continued to use MI data to estimate exposures through the 1990s, a quarter of a century after the mines converted to fiber counting. Dose-response relationships based on a completely inaccurate, but *apparently* large set of exposure data, provide a false sense of statistical security to these results.

Measuring Visible Fibers: The Iceberg Effect

The measurement of fibers by light microscopy and any conversion from particle to fiber count rests on the assumption that the visible fibers measured constitute some fraction of the total number of fibers present in the air. This is because most fibers present in the air are not visible under light microscopy. In addition, by convention, light microscopy does not measure fibers measuring less than 5 microns in length [Sebastien et al., 1989]. Therefore, for QAMA–McGill exposure estimates to be considered valid, two requirements must be met. First, there must have been a consistent proportional relationship between visible fibers and total fibers. This also necessitates a consistent relationship between visible fibers and fibers less than 5 microns in length in various processes (i.e., mining, milling, and maintenance). These relationships needed to be maintained over a 60-year time period during which many processes changed dramatically.

Second, in order to compare exposures between two completely different processes like mining and textiles, the ratio of visible to invisible and uncounted fibers must be similar. There are more invisible fibers per visible fiber in textile manufacturing than in mining. Therefore, each textile fiber counted represents more invisible fibers than each mine fiber counted. An examination of the mining, milling, and textile processes and the history of fiber measurement techniques indicates that neither of these two requirements was ever met in the context of the QAMA–McGill research.

Nicholson [1986] summarized the main technical problems in establishing asbestos exposure-disease relationships:

> Even with the advances in fiber counting techniques, significant errors may be introduced into attempts to formulate general fiber exposure-response relationships. The convention now in use, that only fibers longer than 5 μm be counted, was chosen solely for the convenience of optical

microscopic evaluation (since surveillance agencies are generally limited to such instrumentation). It does not necessarily correspond to any sharp demarcation of effect for asbestosis, lung cancer, or mesothelioma. While it is readily conceded that counting only fibers longer than 5 μm enumerates just a fraction of the total number of fibers present, there is incomplete awareness that the fraction counted is highly variable, depending upon the fiber type, the process or products used, and even the past history of the asbestos material (e.g., old vs. new insulation material), among other factors. For example, the fraction of chrysotile fibers longer than 5 μm in an aerosol can vary by a factor of 10 (from as little as 0.5% of the total number to more than 5%). When amosite aerosols are counted, the fraction longer than 5 μm may be 30%, extending the variability of the fraction counted to two orders of magnitude [Nicholson, 1986].

Fiber length is not the only consideration relevant to fiber counting. Nicholson also notes that as many as half of the fibers may have been missed using optical microscopy that cannot measure fibers of the smallest diameters (See Note 6).

Asbestos fiber-counting is a ''tip of the iceberg'' phenomenon because fibers are counted by light microscopy. Since chrysotile fibers split longitudinally, some of the fibers are too narrow to be seen and are not counted. The first steps of the textile process are specifically designed to split fiber bundles; therefore, textile exposures involve a higher percentage of thin (invisible) fibers than mill or mine exposures [Dement and Harris, 1979]. As a result of increased fiber splitting in the textile process, each fiber counted represents many more uncounted fibers than those in the mining and milling process [Nicholson, 1986; See Note 7].

Mesotheliogenic Potential of Thin Fibers

Fiber width is clinically important to carcinogenic potency. Thinner fibers (generally less than 0.1 μm), which are invisible under light microscopy and therefore uncounted, are far more mesotheliogenic than wider (visible) fibers [Pott et al., 1972; Stanton et al., 1981; Lippmann, 1988]. Lippmann first noted this explanation for the ''textile mystery'' in 1988:

> ''The origin of this lower risk [for miners] is not fully understood, but part of the difference may lie in the different fiber size distributions between the mining and milling of chrysotile and its use in a textile plant or other production facility (See Note 8).''

Critique of McGill Dose Estimates: Sampling Methods

The McGill researchers based their dose estimates on midget impinger measurements taken between 1948 and 1966.[1] However, cohort workers were most heavily exposed before 1946. In fact, the QAMA began the exposure measurement program to help control dust levels. Case [2001a] claimed that the exposure sampling, because it relied on 4,152 individual samples, reflected real exposure levels and was of high quality, stating that ''. . .this is far more data than you're ever going to get in the average epidemiological study, this was a monumental task,'' and ''. . .this environmental data base was of a quality better than most.'' Gibbs asserted, ''Measurements from other sources such as government reports, insurance companies, mining companies, and others were consulted and data gathered when necessary using surveys by the research team. The distribution of measurements was such that it was possible to obtain a reasonable estimate of the concentrations associated with most jobs and work areas on an annual basis'' [Gibbs, 1994].

However, Gibbs and LaChance [1972] admitted the poor quality of sampling from the factory in the town of Asbestos, noting, ''A total of 3,096 dust measurements, made periodically since 1944, was used as a guide to the exposure in the factory. Since they were made by several different persons using various methods, including the Greenberg Smith impinger, midget-impinger, and Owens jet sampler, these measurements were less satisfactory than those for the mills.'' Nonetheless, the QAMA–McGill researchers based several publications on these data [Liddell et al., 1997, 1998; Liddell and McDonald, 1980; McDonald, 1980; McDonald and McDonald, 1980; McDonald et al., 1980b, 1993, 1997, 2001].

In 1984, they asserted, ''We cannot claim precision or certainty for our estimates, only that the available data—more plentiful in this industry than most others—were used to the best of our ability'' [Liddell et al., 1984]. Despite the fact that these researchers recognized that the MI samples were of no practical value, they acknowledged that ''No attempt was made to extend work histories beyond 1966 because exposure levels in the period 1967–75 were much lower than in the past, and exposure in a short period before death could not be expected to contribute to risk'' [Liddell et al., 1984]. However, the QAMA–McGill researchers followed the cohort until 1992, finding that about one-quarter of the cohort had significant post-1966 exposures long before they died [Liddell et al., 1998]. Incredibly, although they had access to the cohort and could have prospectively determined exposure levels from 1966 forward, the QAMA–McGill researchers merely projected the previous 18 years of exposure data measurements forward to 1992 [Liddell et al., 1998].

[1] Some of the studies say the dose estimates began in 1948 and others report 1949.

In addition to attempting to equate the *quantity* of measurements with *quality*, the QAMA–McGill researchers' presentation of the quantity of samples is also misleading. In fact, a very small percentage of samples were taken, given the number of mines and mills, job classifications, variability of exposures in the same process within and between mines, and the 80-year follow-up period. The QAMA mine owners sampled 44 mines. Each mill and mine had at least eight major processes, each of which resulted in variable particle counts and particle/fiber ratios taken over an 18-year period [Gibbs and LaChance, 1972, 1974]. In actuality, the QAMA sampled each of the major mine processes an average of less than once every 3 years for between 5 and 30 min. Furthermore, QAMA *never monitored* two large cohorts of workers, miners, and maintenance workers who comprised 20–30% of the entire study population [Gibbs, 1972].

Gibbs based his dose estimates for these groups on interviews with workers, which depended on remembered visual estimations of dust levels. In his PhD thesis he noted, ''Visibility, which was affected by fog and lighting, as well as dust, probably played a part in the workers' assessment in underground mines and mills and may have led to an *overestimation of dust levels* (Emphasis added)'' [Gibbs, 1972]. It should be noted that lighting and distance variables alone could result in particle estimate differences of one-hundred-fold [Hemeon, 1963]. This problem is compounded by the fact that visibility is a function of total particles and not fiber counts. The researchers stated that their ''historical analysis,'' based on interviews, indicated that the maintenance workers had high exposures compared to the miners [Gibbs and LaChance, 1972]. This may be true, but Gibbs later reported that miners had twice as much pleural disease as millers or rock crushers in the same mines and the maintenance workers who generally worked in the mill or other process buildings [Gibbs, 1979].

In 1971, Gibbs noted that there was ''general. . .agree-agreement'' among the workers questioned on remembered dust conditions [Gibbs and LaChance, 1972]. However, in another paper published 12 years later, Liddell et al. [1984] noted that the occupational histories often conflicted with written records. The QAMA–McGill researchers applied the dose estimates based on worker interviews to particular types of jobs, which included 13,346 different job descriptions [McDonald et al., 1971]. Gibbs ''reduced'' these to 5,783 different jobs in thirteen general exposure categories, and then applied these individual exposure categories to individual work histories based on written records. It should be noted that on the average, each worker had ten different jobs [Gibbs and LaChance, 1972].

Gibbs claimed the QAMA samples were taken to assess both industrial dust control effectiveness and individual exposures. In the same paper, he also noted that the midget impinger is ''a relatively short-term instrument, is difficult to use for personal monitoring and is not specific for fibers'' [Gibbs, 1994]. Gibbs reported that QAMA knew which samples were recorded for control purposes and which were collected to be ''representative'' of actual exposures. However, no breakdown of the relative proportion of samples in each category has ever been provided and there is no indication that any personal sampling was ever done [Gibbs, 1994]. Interestingly, in the same paper, Gibbs criticized Dement's textile exposure estimates for failing to provide ''information on whether or not samples were personal samples'' or any information on ''the distribution of locations at which side by side samples were taken'' [Gibbs, 1994]. Each midget impinger sampling lasted between 5 and 30 min and thus, could not possibly reflect average daily—let alone yearly—exposures. QAMA did not record counts below the supposed ''exposure limit'' and the QAMA–McGill researchers never indicated what the ''exposure limit or limits were during the relevant time period or how they dealt with these 'unrecorded' measurements in their dose estimates'' [Gibbs, 1994].

Missing Data Points

In 1972, Gibbs and Lachance hinted at the inadequacy of the sampling locations when they reported the results of new samples taken ''to obtain information in areas where no dust measurements had previously been made'' during the preceding 60 years [Gibbs and LaChance, 1972]. These areas included most of the job categories involving exposures. While they reported that this data was missing for only three mines, these actually represented ten mines, which had either previously merged or whose exposures the authors felt were comparable. Another five mines were closed at the time of the study and no data of any sort appears to be available for the workers at these mines [Gibbs, 1972]. Gibbs collected some samples for each of the job categories, but he created and used an entirely new method for measurement, one which is not completely described and does not appear to have been validated in any way [Gibbs, 1972]. Gibbs made no attempt to compare these results to the midget impinger total particle counts that were available for the rest of the workers. Although ''median'' values are reported in the published paper, many of these values correspond to only single sample results [Gibbs, 1972].

Gibbs and Lachance [1972] based much of their dose reconstruction on the aforementioned biased results. They reported that maintenance workers had higher exposures than millers, but the range of exposures for maintenance workers was 1.1–61.8 (personal samples). The range for mill workers was 0.3–159 (area samples). Moreover, the exposure measurements varied widely. Gibbs published the data on between-mine variance, but omitted data on the variation of doses at the same mine for the same job. In fact, Gibbs found that exposures at the same mine for the same job had a range of as much as 200% [Gibbs, 1972].

Particle-Fiber Conversion Issues

QAMA has long known both the impossibility of estimating asbestos fiber exposures for every job class, and the irrelevance of particle counts in determining toxicity. In 1953, the QAMA executive board meeting minutes noted, ''The industrial hygiene surveys that have been made in the past, and in which only dust particles were measured, are practically without value'' [Jackson, 1953].

QAMA waited 20 years to change to membrane filter measurement after receiving this information. The McGill researchers were left with only particle counts, but if the particle counts could not be correlated with fiber counts or were inversely related to fiber levels, then the particle counts were useless as indices of exposure to determine asbestos toxicity. Gibbs and Lachance tested this hypothesis by performing 87 matched pairs of tests utilizing light microscopy and membrane filters to count fibers and comparing these with midget impinger particle counts [Gibbs and LaChance, 1974]. They found that, overall, the relationship between particle counts and fiber counts were 13% better than *random* number generation. Incredibly, for low fiber count exposures, the particle counts were *inversely* related to fiber exposures. This inverse relationship occurred in more than one-third of the samples (31/87). Therefore, for at least one-third of the particle counts, it was determined that the higher the count, the lower the workers' exposure to asbestos. Gibbs and Lachance note:

> For thirty-one samples with less than one fiber per field, the linear correlation was very close to zero, −0.03, and the correlation of log rhythmically transformed data was 0.25. However, these correlations suggest that for all mines the regression lines are unsatisfactory for the prediction of fiber counts from impinger counts, as the improvement and prediction for the best correlation, 0.45, is only 13% better than a conversion obtained at random. Thus the conversion of dust disease relationships to fiber disease relationships does not seem possible [Gibbs and LaChance, 1974].

Gibbs and Lachance acknowledge the poor correlation of side-by-side midget impinger samples and recommend

> that safety standards, at least in this industry, should continue to be based on dust counts for which there is considerable epidemiologic support rather on fiber counts, for which there is no direct evidence [Gibbs and LaChance, 1974].

They concluded, ''The conversion of dust disease relationships for the Quebec Mining and Milling Industry to fiber disease relationships *does not seem possible* at the present time.'' Gibbs and Lachance suggest that even though we now know that the fiber and not the particle causes disease, and most of the particles counted are not asbestos, that safety standards should continue to be based on particle counts.

The lack of scientific validity of these dose estimates did not stop the QAMA-funded McGill research team. They selected a single conversion factor for all processes and henceforth all subsequent publications have relied on this single value (although minor adjustments to the value have been made from time to time). McDonald states in a 1973 IARC conference publication that dust-sampling methods, in addition to unreliable particle-fiber conversions, produced data too variable to be considered a reliable basis for estimating exposure [McDonald, 1973].

Attempted Corrections

By 1989, the QAMA-funded McGill researchers realized that they needed to provide better justification for their high dose estimates. Since the chrysotile came from the same mine, the most obvious and simple explanation for this ''mystery'' appeared to them to be the inadequacy or non-comparability of the dose estimates from these two operations. After all, they had already shown that there was, at best, no correlation between particle and fiber counts in the QAMA sampling. At worst, there was an inverse relationship between particle and fiber counts [Gibbs and LaChance, 1974]. On the other hand, the textile particle counts correlated with fiber counts because each textile process produces a narrow range of particle/fiber ratios. The researchers tried to ''fix'' this clearly irreparable problem with the dose estimates by comparing particle counts to retained fiber levels in the lung [Sebastien et al., 1989]. This could only add another level of error since, as they noted, lung fiber counts depend on retention, clearance, and dissolution (See Note 9).

Sebastien reasserted the inadequacy of the original QAMA–McGill exposure data, stating:

> Initially we thought it might be appropriate to use regression analysis to relate exposure, intensity (mpcf) to lung fibre concentrations in the two series and to compare observed values in one with those expected by application of the regression equations from the other. Although the results obtained by this approach were similar to those from the matched pair and stratification analyses, we have not quoted them here because the underlying assumptions as to linearity did not seem justified [Sebastien et al., 1989].

The researchers compared the ratio of chrysotile exposure in millions of particles per cubic foot in miners and

millers, with the exposure data from textile workers in South Carolina with the amount of retained chrysotile and tremolite in the worker's lungs [Sebastien et al., 1989]. However, the QAMA–McGill researchers selected lung cancer cases for 8% of the Charleston workers but 25% of the Quebec miners. This introduced another systematic bias, since it is likely that workers with lung cancer had higher exposures to asbestos. The selected cases from Thetford were not even representative of the Thetford cohort, as Sebastien noted, ''high [exposure] values were over-represented in necropsied cases'' [Sebastien et al., 1989]. Ironically, they concluded that tremolite was not responsible for the ''higher risk of lung cancer in Charleston. In fact, . . . The analyses indicate the reverse.''

The QAMA–McGill researchers did not count fibers less than 5 μm and they only analyzed ''the first five fibers seen.'' Not surprisingly, the mean diameters and fiber lengths were similar. Unfortunately, the comparison of mean ratios did not comport with their theory because the mean ratio of the particle counts between Thetford and Charleston was 11.8 and the mean chrysotile/tremolite lung fiber count ratio was 18. The QAMA–McGill researchers did not report this comparison of means; they simply calculated geometric means to minimize the impact of outlier data points. These outliers, however, comprised precisely the type of information the QAMA–McGill researchers claimed to be evaluating in the first place.[2] Clearly, comparing the averages of ratios to determine if exposure measurements are accurate does not call for an evaluation of geometric means. If any statistical test is appropriate, it is the comparison of arithmetic means. This comparison again showed that the QAMA–McGill exposure measurements were inaccurate and that their research methods were fatally flawed. Of course, knowledge of this shortcoming was already firmly established in 1974 from Gibbs' original analysis [Gibbs and LaChance, 1974].

Worker interviews

Since the QAMA exposure data did not measure exposures for most of the workers in the relevant cohort, the McGill researchers relied on company records to reconstruct exposures. Their reports on this ''check'' of the validity of the work records are wholly contradictory.

As mentioned earlier, Gibbs first reported in 1971, ''Men were asked to relate dust conditions they remembered to those in areas where measurements had been made recently. In general, there was agreement among those we questioned'' [Gibbs and LaChance, 1974]. However, in 1984, Liddell reported:

> Over the years, we had come across several inconsistencies and other evidence of errors in work histories. We took this opportunity to attempt correction where appropriate, but for reasons outlined elsewhere, we were unable to make use of this effort. *Many of the changes in work history, elicited by 'blind' field inquiry and checked against company files, were certainly justified, but we have not made them, and so allowed errors to remain* (Emphasis added) [Liddell et al., 1984].

Even after recognizing their errors, they consistently and continually ignored them. They even attempted to justify their decision not to correct the errors, stating, ''However, this type of error appears to have been distributed unevenly, and so like might not have been compared to like. In case-referent comparisons, minor random unbiased exposure errors are probably less serious than bias; we therefore returned to the situation that existed before the field work was instituted'' [Liddell et al., 1984]. They provided no analysis of the magnitude or randomness of the errors. No effort was made to compare the interviews with the written record to determine whether or not the written records contained a systematic bias.

Conversion factors

First Gibbs and LaChance [1974] and later Liddell et al. [1984, 1998] evaluated the merits of converting particle counts to fiber counts [Gibbs and LaChance, 1974; Liddell et al., 1984, 1998]. In addition to their realization that worker interviews indicated that the original dose estimates were even less accurate than previously assumed, they again recognized that particle/fiber ratios were ''virtually independent of the level of exposure'' [Liddell et al., 1984]. Furthermore, it was clear that if a conversion factor were to be used, it needed to be specific for each job category [Gibbs, 1994]. Disregarding their own findings, they used *a single particle/fiber ratio standard for all years in all job categories*. They based this standard on the worker histories, which they then proceeded to ignore in calculating the actual particle counts because they postulated that the histories would introduce a ''systemic bias'' into their analysis (See Note 10).

Liddell et al. [1998] again recognized the inadequacy of their dose estimates concluding that:

> the classification of jobs by dust category would not be a reliable classification by fibre count [Liddell et al., 1998; See Note 11].

[2] Geometric means are utilized to calculate power function averages. For example, they can be used to determine what an average interest rate would be if $100,000 was invested in the bank in 1990 and had variable interest rates each year of 2, 5, 7, and 10 over the next 4 years.

They also understood why the dose estimates were so inaccurate. Fiber/dust ratios necessarily differed by orders of magnitude for different types of work and for the same work process at different points in time. Liddell et al. [1998] noted that, ''The two important reports by Gibbs and Lachance [1972, 1974] give some indication of the inherent complexity; a simple example is that work on the tailings dump in 1968 was extremely dusty but, as most of the fibre would have been extracted the fibre:dust ratio must have been quite low.'' The exposure data was so inaccurate that, ''taken at face value,'' exposures even appeared protective for the workers. In other words, unmanipulated, the exposure data indicated that chrysotile exposure actually prevented workers from developing pneumoconiosis, lung cancer, or mesothelioma [Liddell et al., 1998]. Their original findings would be plausible if one considered an alternative hypothesis in which workers who had the highest exposures died from non-malignant disease before the latent period for the induction of cancer had been attained. Instead, since they believed an inverse exposure relationship was ludicrous, they manipulated the exposure estimates until the dose-response curve fit their a priori understanding of the proper form for the dose-response relationship. The researchers discarded all of the exposure levels that were inversely related to disease. They described this manipulation in an appendix titled ''Elimination of negative regression coefficients,'' and proceeded to ''revise'' the exposure data to create results that would allow them to argue that chrysotile exposure was ''innocuous'' (See Note 12).

Years of exposure "correction"

In 1980, McDonald commented on the exposure data and noted:

> Relative risks of lung cancer were considered in detail by Liddell et al. and it appeared that there was little to suggest that the way in which dust exposure had been accumulated played any part in determining the risk. . .[McDonald et al., 1980a; See Note 13].

Even after this ''validation'' of the dose estimates, they unequivocally concluded that the dose estimates were worthless when Gibbs wrote, ''thus it is clear that there is no single overall conversion factor that can be applied to the mine and mill data'' [Gibbs, 1994].

Manipulation of the cohort to achieve desired results

In his 1980 paper, McDonald justified the age 45 cut-off, stating that ''by this time most of the men had completed their service'' [McDonald et al., 1980b]. This was simply not true. In 1997, Lidell et al. revealed that over 2,400 men in the 1890–1920 study cohort were still employed in 1967, and the *youngest* of these was 47. Since the age 45 cut-off no longer produced the linear dose-response curve they sought, in 1997 they calculated exposures up to age 55. Rather than using exposure data obtained after 1966, the year the QAMA–McGill studies began, they used the *already-discredited* pre-1966 data to estimate these exposures. They also altered the dose calculation methodology, asserting that ''it did not prove feasible to use the same methods as previously'' [Liddell et al., 1997]. They do not provide any rationale for this change (except perhaps for large computer file size), nor do they provide a comparative analysis of results using the ''old'' and ''new'' dose estimate methods (See Note 14).

Despite these manipulations, the McGill researchers themselves have cast doubt on the reliability of the exposure estimates for pneumoconiosis and mesothelioma, stating:

> ''Pneumoconiosis death rates per 100,000 subject-years were clearly associated with exposure at the two main places of employment, but the exclusions from this table [early deaths: 12 from pneumoconiosis and 1 from mesothelioma] may have distorted these associations, and certainly make comparison between the Asbestos mine and mill and Company 3 particularly difficult. There is little sign of corresponding associations with mesothelioma'' [Liddell et al., 1997].

Misleading Conclusions

In 1998, Liddell delineated the pre-determined conclusion that the QAMA–McGill researchers planned to expound in the last paper of the series, namely, that the health effects of chrysotile were ''essentially innocuous'' (See Note 15).

The QAMA–McGill researchers thus concluded that nearly half (72) of the deaths that they attributed to asbestos exposure were inconsequential because these deaths did not significantly alter overall mortality rates. Since their conclusion is a political one, perhaps a current political analogy will help shed light on this analysis. If one were to apply the same standard to the World Trade Center destruction, one could similarly conclude that this act of terrorism, that has changed the world for years to come, was ''innocuous'' because it did not significantly impact the US SMR for 2001.

Inadequate Case Ascertainment

In addition to the overestimating of dose, systemic underestimates of asbestos-associated diseases may have

also contributed to the apparent ''low risk'' of chrysotile exposure. In 1950, in a meeting with QAMA officials, Dr. Lanza noted that this was a likely explanation: ''It was pointed out that in the Province [Québec] it is the practice not to list cancer as a cause of death even when it is, so that information on this may not be of much help to us'' [Trudeau Institute, 1950]. Metropolitan Life provided group life insurance to the mine workers, and began collecting mortality data and death certificates on the workers in the 1920s. Begin and colleagues provided further support for this diagnostic and/or reporting bias when he documented ''an increasing incidence of cases of malignant mesothelioma in chrysotile miners and millers of the Eastern Townships of Québec, with 49 cases in the last 23 years, and a rate of 2.5 cases per year in the last 10 years in the primary industry, as compared with a rate of 0.3 per year in the years prior to 1969 ''(McDonald et al., 1979 as cited by Begin et al. [1992]).'' A similar eighty-fold undercount of lung cancer case finding can easily account for the fifty-fold ''textile mystery.''

There is clear evidence that this under-count occurred and was, in fact, organized by the main financial sponsor of the studies, QAMA. In 1995, Schepers reported that Ivan Sabourin, head of the Conservative Party of Quebec and legal counsel to QAMA, had systematically removed stored pathological specimens—the removed lungs—of deceased Quebec miners diagnosed with lung cancer and sequestered them at the Trudeau Institute at Saranac Lake New York [Schepers, 1995]. By 1946, at least 17 cancer cases had been removed and are still currently missing from the QAMA–McGill analysis. While unprecedented, this ''organ-snatching'' clearly had a differential impact on the number of cancer cases attributable to the QAMA mine operations and those reported from the control group.

Because the cohort workers were born between 1890 and 1920, some mesothelioma deaths are likely to have occurred before the disease was widely recognized by most physicians in the mid- to late-1960s. Until the eighth revision of the *International Classification of Diseases*, which was adopted in the US in 1968, mesothelioma of the pleura was classified as a benign neoplasm of the respiratory system [U.S. Department of Health, Education and Welfare, 1975]. By this time, much of the cohort was over 65 years of age and had accumulated more than 40 years of latency. It is likely that this diagnostic bias is also a cause of the apparently low mesothelioma rate.

Influence of Political Considerations

Under-reporting by Quebec physicians must also be considered as a likely explanation of the fallacious results. No less an authority than Pierre Elliott Trudeau noted that the QAMA mines have been the absolute center of Canadian politics during this century. In the foreword to *The Asbestos Strike* of 1949, Trudeau called the strike the most important event in Canadian politics in the twentieth century [Trudeau, 1974]. The political importance, power, and influence of QAMA during this century cannot be overestimated. This is particularly true with respect to QAMA's recognition of the potential impact of asbestos-related health problems on profits. At the suggestion of Wade Wright, the medical director of their insurer, Metropolitan Life, the mine owners began undertaking projects to influence the medical literature and individual physicians in Quebec in the 1920s when they took a ''mortgage out on McGill'' [Wright, 1926; See Note 16]. Initially, the QAMA was concerned with the potential financial impact of workers' compensation claims. By the mid-1930s, they had already developed programs to deal with the adverse consequences of the fear of asbestos-related diseases on sales [Lanza, 1937; Lilienfeld, 1991].

CONCLUSION

The Canadian asbestos mining industry has a long history of manipulating scientific data to generate results that support claims that their product is ''innocuous'' [Liddell et al., 1998]. Researchers complicit in this manipulation seem to be motivated by a variety of interests, including a desire to support an important national industry and a pre-existing ideological commitment to support corporate interests over worker or community interests. Conducting industry-friendly research can also anchor an academic career by guaranteeing the steady stream of funding necessary to stay afloat in the ''publish or perish'' environment of the university. Yet, as industry's scientists must know, their research has implications extending far beyond their offices or laboratories.

Today, the impact of QAMA's policies is most dire in the developing world. Almost all of Canada's asbestos is exported to the developing world, and corrupt medical literature continues to be used in arguments to promote the sale of Canadian chrysotile there [Castleman, 2002]. While there are few studies on the extent of asbestos-related disease and death in the developing world the death toll there is likely to be staggering. QAMA–McGill researchers interested in preserving this key market have argued before the World Trade Organization to buttress the proposition that chrysotile is not a cause of mesothelioma and should therefore not be subject to national bans [B.I. Castleman, unpublished communication; Browne, 2000 http://www.chrysotile.com/en/hltsfty/browne.htm; World Trade Organization, 2000].

In the United States, QAMA-supported researchers are currently influencing federal policy on asbestos. A recent report to the Environmental Protection Agency by the Eastern Research Group, Inc. [2003] was based on the QAMA-funded research we have reviewed here. The report was reviewed by Bruce Case and other scientists who have been retained by QAMA member companies [Eastern

Research Group, Inc., 2003]. The legitimacy that has been granted to QAMA's ''anything but chrysotile'' theories is evidence of the success of QAMA's more than three decade campaign of ''propaganda'' [QAMA, 1967]. QAMA has been even more successful than the tobacco industry they emulated. It would be politically impossible for the FDA to depend on the opinions of tobacco industry-funded researchers who stated that tobacco was ''innocuous.'' Yet that is exactly what has happened with EPA's designation of Bruce Case and others as ''experts'' on asbestos risk. QAMA's unsound science does not deserve such credibility. Until their spurious nature of their data and conclusions are exposed, injured workers and bystanders will go uncompensated and chrysotile will produce yet another generation of victims.

APPENDIX: NOTES

1. De's PhD thesis entitled, 'Petrology of dikes emplaced in the ultramafic rocks of South Eastern Quebec' was deposited in 1961 at Princeton University. The objective of the thesis was to study the dike rocks and their relation to the ultramafic rock in the Eastern Townships. The concentration of amphiboles in the rocks are variable and can be substantially high in some dikes of granitic and dioritic composition. For instance, he reported the presence of fibrous actinolite in concentrations as high as 14% in granitic dikes and suggested that the granitic pegmatite from the Jeffrey mines in the town of Asbestos would even contain anthophyllite. Thus as suggested by lung burden analyses and mineralogical data, the concentrations of amphibole fibers (especially tremolite and actinolite) contaminating the chrysotile mineral ore from Asbestos or Thetford-Mines are at about the same level, although this may not be reflected in sporadic air samples. These mineral matters were likely constituents in the final product. There is a need to clarify how high were the concentrations of amphibole fibers in the host dunite rock especially tremolite and actinolite [Dufresne et al., 1995].

2. McDonald and McDonald state, ''The possibility that this distribution [fewer mesothelioma cases in peripheral mines] might be related to the concentration of fibrous tremolite in the two areas was then tested with data on asbestos fiber concentrations in lung tissue from 83 cohort members from Thetford mines who had died from causes other than mesothelioma and had been examined by electron microscopy in 1988. The number of lungs examined was 58 from area A (central-high tremolite mines) and 25 from area B (peripheral-low tremolite mines); the groups were similar in duration of employment (36 and 37 years) and time from termination to death (8 years in both), but estimated accumulative dust exposure was about 30% higher in group B. The geometric mean concentrations of fibers equal to or greater than 5 μm in length per microgram of dried lung were as follows: chrysotile, area A, 7; area B, 13 (not significant); tremolite, area A, 32; area B, 7 ($P = 0.0002$)'' [McDonald and McDonald, 1995].

3. Browne stated, ''The main tremolite-contaminated mines are now closed.'' He also said, ''But in the past, the percentage of tremolite in the fibre could be as high as 1%, whereas the high tremolite-contaminated mines in the central Thetford area have closed, and in any case the high geological research has shown that tremolite is not uniformly mixed with the chrysotile, but occurs in separate seams which can be identified and avoided. And lastly, there is evidence that much of the tremolite is lost in the milling, so that what is delivered to the manufacturer will have an even lower content. So present and future supplies from these sources have and will have minimal tremolite'' [Browne, 2000 http://www.chrysotile.com/en/hltsfty/browne.htm].

4. A. Chrysotile composes about 5% of the ore deposit. It forms in layers or sheets between serpentine rocks. Tremolite and crocidolite are present in the adjacent rock alongside the 100% pure chrysotile vein. The adjacent rock is compressed in rock crushers, a process that is repeated three times. The rock is left to dry for 48 hr after the first crushing. This initial process releases tremolite from the serpentine, and it is mixed in with the chrysotile.

   B. The crushed ore is moved to a conveyer where all fibers on the belt, including the released tremolite and crocidolite, are vacuumed off and carried into the mill As a result, the end product is contaminated with tremolite and crocidolite.

   C. The fiber is transported to the sorting mill. The sorting mill then separates the fiber by size. This is a two-part process. The fiber is shaken and rocked from side to side on a ''sifter,'' which slopes down towards a cyclone vacuum. A five-foot-long by two-inch-wide cyclone at the end of the sifter vacuums the fiber into a tube and it is carried by the force of air to the bagging area. This is how fiber sizing is achieved. The suction is set to pull short fibers, of any chemical composition, off first. The remaining longer fibers are dropped onto another conveyer and the process is repeated until the longest fibers are cyclone-vacuumed off. It is clear that the tremolite is sized along with the chrysotile.

   D. The fiber is blown from the sifting area and transported to the bagging area where it is blown into bags ready for shipment.

5. Vorward wrote, ''Last week, while in Washington, I had the opportunity to discuss our program concerning the epidemiology of pulmonary cancer in subjects exposed to asbestos dust and to present the problem which you posed regarding job classification. I agree with your views. Certainly it is an *impossible* task to tabulate the various jobs on comparable scientific data, since such data does not exist. Therefore the code suggested by both you and Ken [Smith, medical director of Johns-Manville] should be used (Emphasis added)'' [Vorwald, 1951].

6. As Nicholson noted, ''Using electron microscopy, Rendall and Skikne [1980] measured the percentage of fibers with a diameter less than 0.4 μm (the approximate limit of resolution of an optical microscope) in various asbestos dust samples.

In general, they found that more than 50% of the 5 μm or longer fibers are less than 0.4 μm in diameter and, thus, are not visible using a standard phase contrast optical microscope'' [Nicholson, 1986].

7. Nicholson continued, ''Moreover, as with length distribution, diameter distribution varies with activity and fiber types. As a result, the fraction of fibers longer than 5 μm visible by light microscopy varies from about 22% in chrysotile and crocidolite mining and amosite/chrysotile insulation manufacturing to 53% in amosite mining. Intermediate values of 40% are measured in chrysotile brake lining manufacturing and 33% in amosite mill operations. Thus, even perfect measurement of workplace air, with accurate enumeration of fibers according to currently accepted methods, would be expected to lead to different exposure-response relationships for any specific asbestos disease when different work environments are studied'' [Nicholson, 1986].

8. Lippman continued, ''Animal experiments. . .indicate that the fibers most likely to produce cancer are too thin to be observed by a light microscope. In the mine and mill the chrysotile fiber bundles have only been partially broken apart. Many of the fibers are large and easily counted: some of those counted are curly and non-respirable. When shipped to a chrysotile textile mill the fibers are further broken apart during carding. In the high-speed spinning and weaving processes, thin fibers may split off from the threads most of which are not visible in a light microscope. Thus in the air of a textile plant the percentage of thin, uncounted, but highly carcinogenic fibers can be greater than in the mine and mill air and a greater cancer risk observed for the same measured cumulative fiber exposure'' [Lippmann, 1988].

9. Sebastien writes, ''In the absence of an accepted model for lung retention of asbestos fibres comparison between the two groups was restricted to cases having similar time characteristics of exposure (duration and cessation). In these circumstances it was assumed that retention would be proportional to mean intensity of exposure. This assumption, impossible to test without good environmental data, may be questioned, especially for chrysotile'' [Sebastien et al., 1989].

10. Liddell et. al. argue, ''The conversion factor should be amended because many more histories have led to more reliable estimates. We have made four other estimates: the fibre and dust slopes for pneumoconiosis and for lung cancer were in the ratios 3.67 and 3.57 (f/ml)/mpcf; while, based on mean exposures for all subjects, the ratio was 3.46 (f/ml)/mpcf in this report, and was 3.44 in a study of elderly male workers in Thetford Mines. These factors, all based on substantial groups of persons, show little variation. However, the ratios calculated for each of the 2,535 non-zero pairs of exposures in this study ranged between 0.32 and 30 (f/ml)/mpcf, while the correlation of the fibre/dust ratio and its denominator, in the 2,535 sets, was so small that the ratio could be thought of *as virtually independent of the level of exposure.* Nevertheless, any 'average' must depend on each specific group of workers, and on the method of obtaining it. Further, all the above estimates are for workers at Asbestos and Thetford Mines in the period from 1904–66; there is no assurance that they might apply in different circumstances. We would add that there is great assurance that the particle/fiber ratio applied in the circumstance under investigation'' (Emphasis added) [Liddell et al., 1984].

11. The full passage reads, ''Liddell et al. [1984] estimated a factor to convert dust counts to fibre counts as about 3.5 (fibres/ml), mpcf but stated this would be *quite unreliable* except applied to mean dust levels for substantial groups of Quebec asbestos workers. For the many jobs in which the 2217 men included in their study had worked, the fibre:dust ratios had ranged from 0.3 to 30 (fibres:ml):mpcf virtually independently of the dust level; in the current study ratios job by job must have varied similarly so that the classification of jobs by dust category would not be a reliable classification by fibre count (Emphasis added)'' [Liddell et al., 1998].

12. They wrote, ''In all the conditional regression analyses of the full model, i.e., with 13 exposure measures, there was at least one negative regression coefficient, which taken at face value would imply a protective effect of exposure. Years in the highest relevant dust category were pooled with those in the adjacent category and the analysis was repeated. This process was iterated until either all coefficients had become positive, when it was terminated, or until the only negative coefficient was for category 1; in that circumstance, category 1 was eliminated from the model, which was equivalent to setting the coefficient to zero and the odds ratio to unity. . . Admittedly, *there was a degree of arbitrariness in some of the pooling carried out* but every effort was made to retain any 'significant' effects'' (Emphasis added) [Liddell et al., 1998].

13. McDonald et al. continue, ''It therefore seemed appropriate to base a second series of analyses on dust exposure accumulated to a certain age, arbitrarily taken as 45 years, at which age most men had completed their service. After the cohort had been divided by exposure to age 45, two further, but separate, subdivisions were made by mining area (Asbestos and Thetford Mines) and by smoking habit; those whose smoking habit was unknown being added to the largest group—that is, moderate smokers. The study interval started at age 45'' (Emphasis added) [McDonald et al., 1980a].

14. Liddell et al. state, ''As over 2,400 men in the cohort were still employed in 1967, attempts were made to estimate exposures yearly up to 1985, when the last man had retired. It did not prove feasible to use the same methods as previously. Instead, each man was allocated dust levels as follows: for 1967, the same level as in 1966; for each subsequent year, a proportion of that level in accordance with the average trend of fibre concentration for his specific mine or mill. From these levels, we estimated yearly exposures from 1967 to 1985 [McDonald et al., 1993], and extended each man's exposure record by a further 19 years. To give much-needed greater flexibility for the calculation of exposures to the age of 55, for instance, or,

for case-referent analyses, in relation to the age at death of the case the exposure file was re-organized: first, the annual record of exposure, incorporating the adjustment for length of the working week, was changed to the dust level, with an indicator of the work-week adjustment; second, each man's work history was recorded annually from the year in which he started to the year in which he finished, thus reducing the maximum number of years from 82 to 59; and thirdly, the format was changed slightly. With these changes, the complete file was reduced in size by over one-quarter, but remained enormous (5.9 MB)'' [Liddell et al., 1997].

15. The researchers contended, ''For men first employed in Asbestos, mine or factory, they [the SMRs] were very much what might have been expected for a blue collar population without any hazardous exposure. SMRs in the Thetford Mines area were almost 8% higher, but in line with anecdotal evidence concerning socio-economic status. At exposures below 300 (million particles per cubic foot) × years, (mpcf.y), equivalent to roughly 1,000 (fibres/ml) × years—or, say, 10 years in the 1940s at 80 (fibres/ml)—findings were as follows. There were no discernible associations of degree of exposure and SMRs, whether for all causes of death or for all the specific cancer sites examined. The average SMRs were 1.07 (all causes), and 1.16, 0.93, 1.03, and 1.21, respectively, for gastric, other abdominal, laryngeal and lung cancer. Men whose exposures were less than 300 mpcf/y suffered almost one-half of the 146 deaths from pneumoconiosis or mesothelioma; the elimination of these two causes would have reduced these men's SMR (all causes) from 1.07 to approximately 1.06. Thus it is concluded from the viewpoint of mortality that exposure in this industry to less than 300 mpcf/y has been *essentially innocuous*, although there was a small risk or pneumoconiosis or mesothelioma'' (Emphasis added) [Liddell et al., 1997].

16. Wright wrote, ''It is suggested that we approach the Dean of the faculty of Medicine of McGill University with a proposal that upon the establishment of an adequate Department of Industrial Hygiene in the McGill Medical School the Metropolitan enter into an agreement with McGill to secure for the Company certain services and information relating to the health of industrial workers in Canada.

''. . .It would be of great value to the Company to have specific information regarding such matters as:

''1. The distribution of industrial establishments, mines and lumbering operations with data concerning the number of employees in each establishment and district. . .

''. . .To secure such information directly would be difficult and very expensive. If it could be obtained from a department of industrial hygiene in Canada's leading university in return for a moderate annual for the Company would undoubtedly benefit. . .

''. . .*Such a plan involves a definite quid pro quo, payments specifically conditioned upon a commensurate return, the adequacy of such return to be determined by the President, or those to whom he may delegate the decision*. . .

''. . .The Sun Life might well ask—if it secured a mortgage on McGill. . .Technical guidance in regard to matters affecting community or individual health—such as, aid in preparation of publicity, occasional research matters not involving great outlays of money, field investigations as of sanitation, water, or milk supplies, or industrial hazards.

''. . .Observations of our scheme at Harvard leads me to add that Martin should not only be prepared to render certain types of service but certain services, more or less routine perhaps, should be specified. Unless there is a definite, tangible quid pro quo the interest of a financial supporter may well languish after a few years'' (Emphasis added) [Wright, 1926].

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