Asbestos: a status report

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Asbestos is a term given to a group of minerals that crystallize in a fibrous habitat. Four minerals have commercial importance—chrysotile, amosite, crocidolite, and anthophyllite. Of these, chrysotile is the most important, accounting for more than 95% of current world production. The various minerals differ chemically and structurally, but all readily separate into extremely fine fibers, most of which are thinner than 1 μm in diameter. As such, they can easily become airborne during manufacturing processes using asbestos or by abrasion or disturbance of asbestos-containing materials. Their thinness also allows them to be readily inspired and carried into the lower regions of the human lung, where they can become lodged and cause bodily damage.

The modern history of asbestos disease dates from the turn of the century, when two cases of asbestotic lung scarring were briefly described in asbestos textile workers. The pulmonary disease resulting from such scarring was well described in subsequent publications and the term *asbestosis* was applied to it in 1924. An association of bronchogenic carcinoma with asbestos was first suggested in the 1930s. Mesothelioma, a malignancy of the lining of the chest or abdomen, was clearly associated with asbestos in 1960. Asbestos has been declared a proven human carcinogen by the US Environmental Protection Agency, the International Agency for Research on Cancer, and the World Health Organization.

Despite publication over the past four decades of a voluminous literature on the causal association between asbestos and disease in different populations, and despite extensive experimental demonstration of the carcinogenicity of all forms of asbestos in laboratory animals, there currently exists an extensive and often acrimonious debate concerning the carcinogenicity of chrysotile asbestos. Because of the continuing extensive sale and use of asbestos in South and Central America, Asia, and Africa and the continued widespread presence of asbestos materials in buildings in the United States and Europe, it is of vital importance that this debate be brought to a swift conclusion.

This review considers epidemiologic studies of the health effects of asbestos in populations exposed to commercial chrysotile and to mixtures of chrysotile and other forms of asbestos. We show that evidence for the human carcinogenicity of chrysotile asbestos is incontrovertible. We suggest that efforts to describe chrysotile asbestos as "safe" are false, self-serving, and commercially motivated.

THE SPECTRUM OF ASBESTOS DISEASES

A seminal study that established much of our current knowledge of the spectrum of asbestos diseases was conducted by Selikoff and Seidman [1] (Table 1). It followed 17,800 heavily exposed asbestos insulation workers for 20 years and clearly demonstrated the wide range of malignancies caused by asbestos. Of these malignant diseases, bronchogenic carcinoma and mesothelioma were the two most important, accounting for more than 90% of the increased cancer risk in this population. Nearly 24% of the deaths in this group of workers were due to bronchogenic carcinoma and 9% to mesothelioma. Lesser degrees of excess cancer mortality were seen at other sites including the esophagus, stomach, colon and rectum, larynx, pharynx and buccal cavity, gall bladder and bile ducts, and kidney. Overall, among insulators whose asbestos exposure began prior to 1968, 30.9% of deaths were *excess* cancer deaths resulting from one of the above malignancies; additionally another 8.6% of deaths in this population were due to asbestosis.

Although the mortality experience of this unusual group is extraordinary, the insulators were not alone either in their exposure to asbestos or in their risk of asbestos-associated cancer. In 1982 our group made estimates of the expected mortality in the United States from past occupational exposures to asbestos [2]. Considering labor turnover since 1940, it was estimated that approximately 700 asbestos-related deaths occur each year among workers directly installing or removing insulation. Additionally, a much larger population of construction workers was also exposed to asbestos, albeit to lower levels. Because of the much greater number of construction workers, many more asbestos-related deaths are taking place among them. Thus it was estimated that approximately 7000 deaths are occurring each year in current or former shipyard and construction workers and in workers exposed to asbestos through maintaining insulation materials in plants and powerhouses. We estimated that the total kill in the United States from the asbestos epidemic will exceed 200,000 deaths.

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Table 2. Risks of lung cancer and mesothelioma in workers exposed to various asbestos minerals

Asbestos exposure and location	Type of asbestos	Percentage increase in lung cancer for a 1-y exposure to 1 f/cm³ of asbestos*
Textile manufacturing (South Carolina)	100% Chrysotile†	2.6
Textile manufacturing (Rochdale, UK)	98% Chrysotile 2% Crocidolite	1
Textile manufacturing (Pennsylvania)	98% Chrysotile 1% Crocidolite 1% Amosite	1.4
Amosite insulation manufacturing (New Jersey)	100% Amosite	4.3
Insulation application (United States)	60% Chrysotile‡ 40% Amosite‡	0.8
Asbestos products (United States)	80% Chrysotile‡ 15% Amosite‡ 5% Crocidolite‡	0.5
Asbestos cement products manufacturing (Louisiana and Ontario)	89% Chrysotile‡ 10% Crocidolite‡ 1% Amosite‡	1.8
Crocidolite mining	100% Crocidolite	1
Chrysotile mining	100% Chrysotile	0.1

*Details of the calculation of these unit exposure risks are described by the Environmental Protection Agency [3].
†Chrysotile as mined in Canada is contaminated by a small amount of tremolite (generally less than 1%). This contaminant is carried over into fiber used in different industries. The percentages in the column refer to the percentage of commercially sold fiber, including the contamination.
‡The percentages of the various fibers used in the plants studied were not given. The estimates are based on published product compositions.

exposure to 1 fiber/mL. These risks are not statistically different from one another and cannot be attributed to the small use of commercial amphibole asbestos in two of the textile plants. Indeed, the highest lung cancer risk was found in the textile plant that used no commercial amphiboles.

Among the remaining nonmining studies, the percentage increase in lung cancer for each year of exposure to 1 fiber/mL ranged from 0.5% to more than 4%, irrespective of the type of fibers used in the production process. The only exceptions were two studies of friction product manufacturing and one of asbestos cement production. In each of these three studies, severe uncertainties limit the validity of the lower risks reported. All remaining risks, involving substantial amphibole exposure, are similar to those of predominantly chrysotile exposures, within the statistical uncertainties of the data. Even a pure crocidolite exposure in mining demonstrated an increased risk of only 1% for each year of exposure to 1 fiber/mL [4].

Although studies of chrysotile mining and milling demonstrate an excess risk of lung cancer, the risk is more than 10 times lower than that seen in studies of asbestos production workers exposed only to chrysotile or to 97% to 98% chrysotile. The origin of this lower risk in the miners is not fully understood. Part of the difference may reflect the different fiber size distributions between the mining and milling operations and the textile plants and other production facilities. Fibers are presumably clumped together and are larger in mining and milling but more fragmented and smaller in the user industries.

In summary, the available data on workers employed in the production of asbestos products strongly indicate a chrysotile lung cancer risk similar to those seen in workers exposed to amosite and crocidolite asbestos. The best estimate of lifetime lung cancer risk for worker exposure to chrysotile in the using industries (as opposed to the mining and milling industries), is an increase of 1% in risk for each year of exposure to 1 fiber/mL (K_L = 0.01). Higher fiber exposure circumstances or longer periods of exposure would give directly proportional higher risks. In certain textile operations, the unit exposure risk appears to be as much as three times higher than this lower limit. No data are available to indicate the presence of a threshold below which there is no risk from exposure to any asbestos mineral.

MALIGNANT MESOTHELIOMA

The risk of mesothelioma by asbestos fiber type can be analyzed in several ways. First, because the risk of lung cancer is very similar across all exposures to all fiber types, excluding mining and milling of chrysotile, one can use the excess number of lung cancers as a measure of cumulative fiber exposure. With comparable follow-up periods, the ratio of the number of mesotheliomas to excess lung cancer is a

Table 3. Ratio of mesothelioma to adjusted excess lung cancer according to type of asbestos exposure*†

Type of exposure	Studies, *n*	Mesothelioma/excess lung cancer	
		Pleural cases	All cases
Chrysotile	8	0.13	0.14
Predominantly chrysotile	6	0.24	0.48
Amosite	2	0.13	0.22
Predominantly crocidolite	6	0.47	0.61
Anthophyllite	1	0	0
Talc (tremolite)	2	0	0.08
Mixed exposures	16	0.19	0.4

**From* Environmental Protection Agency [3].
†Adjusted to the US male cancer rates in 1970.

Table 4. Risks of mesothelioma in workers exposed to various asbestos minerals*

Asbestos exposure and location	Type of asbestos	Risk coefficient
Textile production (Rochdale, UK)	Chrysotile	1×10^{-8}
Insulation workers (United States)	Chrysotile and amosite	1.5×10^{-8}
Factory workers (Paterson, NJ)	Amosite	3.2×10^{-8}
Miners and millers	Crocidolite	13.4×10^{-8}
Cement workers (Ontario, Canada)	Chrysotile and crocidolite	12×10^{-8}

*The listed risk coefficients correspond to the values K_L in the relation, $I_M = K_L \times f \times [(T-10)^3 - (T-D-10)^3]$, where I_M is the mesothelioma mortality rate, f is the average fiber concentration in f/mL, T is the time since start of exposure, and D is the duration of exposure.

fifth that observed. Data points represent an average of 15 years for the first point and an average over 10 years for the remaining three.

The principal finding is that the time course of mesothelioma risk is totally incompatible with an exposure pattern that begins only in the late 1930s. Indeed, the 95% confidence limits on three of the four data points do not intercept the expected distribution for amosite exposure. Barring unknown exposures to amphiboles prior to 1935, the data present strong evidence that chrysotile is the substantial, indeed the dominant contributor to the mesothelioma risk experienced by this group of insulation workers.

The study by Selikoff *et al.* [5] of the entire union membership of US and Canadian insulators also strongly indicates the existence of a chrysotile mesothelioma risk. Follow-up for this study began in 1967, 30 years after the earliest incorporation of amosite by insulation manufacturers into their products. However, the risk of mesothelioma rises steeply with time from first exposure for at least 50 years. Were only amosite contributing to risk, this pattern would not be expected.

One can also estimate risk of mesothelioma due to chrysotile through direct calculations in mixed-exposure circumstances (Table 4.) These analyses show that risk of mesothelioma/fiber exposure, as measured by K_M, is virtually the same for exposures to 97% chrysotile + 3 % crocidolite, 60 % chrysotile + 40 % amosite, and 100 % amosite. The value of K_M from the study of Canadian cement workers is higher than the chrysotile-amosite exposures, as was a value of K_L in the same group of workers. As noted previously, there may be substantial errors in the exposure estimates of this study. The value for a pure crocidolite exposure, as calculated by de Klerk and Armstrong [6] for the mining population of Australia, is about 10 times greater. As with the values of K_L in Table 1, K_M is not definite because of uncertainties of exposures in the early exposure years of the groups under study and from uncertainties of small numbers. Indeed, from a consideration of the ratios of mesothelioma to excess lung cancer, the mesothelioma potency of crocidolite would appear to be only two to four times greater than chrysotile or amosite mesothelioma potency. Nevertheless, in contrast to the analysis of crocidolite lung cancer risk compared with other fibers, the data do indicate a greater mesothelioma potency for crocidolite.

CONCLUSIONS

The case is strong that chrysotile is a potent causative factor for both lung cancer and malignant mesothelioma among exposed workers. Data to support this case derive from more than 40 studies of different fiber exposure circumstances. Chrysotile is shown to be a powerful carcinogen when the time course of risk is considered in mixed fiber exposures. Chrysotile is shown also to be a potent human carcinogen in direct calculations of risk. All available data suggest that chrysotile dominates the risk in those circumstances in which it is the principal fiber used. The risk of chrysotile in producing mesothelioma is similar to that of amosite on a per fiber exposure basis. Crocidolite would appear to have a potential four to 10 times greater to produce mesothelioma for equal exposure than chrysotile.

Chrysotile is a proven human carcinogen. Assertions that it is safe and claims that it can be used safely in developing nations are contrary to fact and extremely dangerous.

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