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REVIEW ARTICLE

The Hazards of Chrysotile Asbestos: A Critical Review

Philip J. LANDRIGAN[1]*, William J. NICHOLSON[1], Yasunosuke SUZUKI[1] and Joseph LADOU[2]

[1] The Department of Community and Preventive Medicine, The Mount Sinai School of Medicine, New York, N.Y. 10029, USA
[2] University of California at San Francisco, San Francisco, California, USA

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Abstract: Chrysotile, or "white", asbestos is the dominant form of asbestos in international commerce today. It accounts for 99% of current world asbestos production of 2 million tonnes. Chrysotile is an extremely hazardous material. Clinical and epidemiologic studies have established incontrovertibly that chrysotile causes cancer of the lung, malignant mesothelioma of the pleura and peritoneum, cancer of the larynx and certain gastrointestinal cancers. Chrysotile also causes asbestosis, a progressive fibrous disease of the lungs. Risk of these diseases increases with cumulative lifetime exposure to chrysotile and rises also with increasing time interval (latency) since first exposure. Comparative analyses have established that chrysotile is 2 to 4 times less potent than crocidolite asbestos in its ability to cause malignant mesothelioma, but of equal potency of causation of lung cancer. The International Agency for Research on Cancer of the World Health Organization has declared chrysotile asbestos a proven human carcinogen. Sales of chrysotile asbestos have virtually ended in Western Europe and North America, because of widespread recognition of its health hazards. However, asbestos sales remain strong in Japan, across Asia and in developing nations worldwide. The claim has been made that chrysotile asbestos can be used "safely" under "certain conditions" in those nations. That claim is not accurate. The Collegium Ramazzini, an international learned society in environmental and occupational medicine, has called for an immediate worldwide ban on all sales and uses of all forms of asbestos, including chrysotile. The rationale for this ban is threefold: (1) that safer substitute materials are readily available, (2) that "controlled" use of asbestos is not possible, and (3) that the health risks of asbestos are not acceptable in either the industrialized or the newly industrializing nations.

Key words: Chrysotile, Asbestos-related diseases, Epidemiology, Risk evaluation, Human carinogenicity, Malignant mesothelioma, Lung cancer

Introduction

Chrysotile asbestos is an important cause of human illness and death[1]. Clinical and epidemiologic studies have established beyond all reasonable doubt that chrysotile asbestos causes cancer of the lung, malignant mesothelioma of the pleura and peritoneum, cancer of the larynx and certain gastrointestinal cancers[2]. Chrysotile also causes asbestosis, a progressive fibrotic disease of the lungs. The risk of these diseases increases with cumulative exposure to chrysotile and also with time since first exposure. Chrysotile asbestos has been declared a proven human carcinogen by the US Environmental Protection Agency (EPA)[3], by the US Occupational Safety and Health Administration (OSHA)[4],

*To whom correspondence should be addressed.

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and by the International Agency for Research on Cancer of the World Health Organization[5]. The total number of deaths in the United States that will eventually be caused by exposure to asbestos, over 95% of it chrysotile asbestos, is estimated to exceed 400,000[6].

Background

Asbestos is a term given to a group of minerals that crystallize in a fibrous habitat. Four fibrous 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 or by abrasion or disturbance of asbestos-containing materials. Their thinness allows them to be readily inspired and carried into the lower regions of the human lung, where they can become lodged and cause damage.

The modern history of asbestos disease dates from the beginning of the twentieth 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* 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.

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 unnecessary 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. Claims have been made that chrysotile asbestos can be used safely. These claims are not supported by valid data.

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 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 mortality in the United States from the asbestos epidemic will exceed 400,000 deaths. Over 95% of the asbestos responsible for this exposure is chrysotile.

Many of the workers who are now dying of asbestos-related disease were exposed to asbestos in concentrations in excess of 10 fibers/ml of air. The current US standard, promulgated in 1994, is 100 times lower, 0.1 fiber/ml. Additionally, procedural standards now require enclosures and the use of air-supplied respirators in circumstances in which release

Table 1. Observed and expected deaths among 17,800 asbestos insulation workers, United States, 1967-1986*

Underlying cause of death	Observed, (n)	Expected, (n)	Standardized mortality ratio (SMR)
Total deaths, all sites	4951	3453.5	143
Total cancer, all sites	2295	761.4	301
Lung cancer	1168	268.7	435
Pleural mesothelioma	173	0	∞
Peritoneal mesothelioma	285	0	∞
Larynx	18	10.6	170
Oropharynx	48	22	218
Kidney	37	18.9	196
Pancreas	54	39.5	137
Esophagus	30	17.8	168
Stomach	38	29.4	129
Colon and Rectum	121	88.5	137
Gall bladder and bile ducts	14	5.4	261
Noninfectious pulmonary disease	507	144.8	350
Asbestosis	427	0	∞
All other causes	2149	2547.3	84

*From Selikoff and Seidman[4]; with permission. +Based on all available information (autopsy, surgical, clinical, and death certificate).

of asbestos fibers is likely to occur, such as during removal of in-place asbestos. These standards were not in place in years past. If US regulations are followed, asbestos-related disease from future exposures will be sharply reduced. However, workers in other regions of the world, particularly in the developing nations in South America, Asia, and Africa, are not similarly protected. In some of these nations, chrysotile asbestos is not officially recognized as hazardous and controls on its use are often minimal.

Lung cancer

It is widely accepted that asbestos fibers, including chrysotile fibers, increase the existing risk of developing lung cancer in proportion to the cumulative exposure that occurred up to a time 10 years prior to evaluation[3]. The relationship can be expressed formally by the equation $I = I_o (1 + K_L \cdot f \cdot d_{(t-10)})$, where I is the lung cancer incidence or mortality observed in a study population; I_o is the age- and calendar year-specific lung cancer incidence or mortality observed expected in the absence of asbestos exposure (ideally, I and I_o would explicitly consider smoking habits of each study individual); f is the intensity of asbestos exposure to fibers longer than 5 μm; d is the duration of exposure in years up to a period 10 years prior to evaluation; and K_L is a proportionality constant that is a measure of the carcinogenic potency of the asbestos exposure. K_L represents the fractional increase in lung cancer incidence or mortality that occurs from a 1-year exposure to 1 fiber/ml. The relative risk of lung cancer, I/I_o, is independent of age and depends only on exposure characteristics.

Information is available that allows exposure-response relationships between asbestos and lung cancer to be considered in various exposure scenarios. Table 2 summarizes estimates of the percentage increase in lung cancer risk associated with a 1-year exposure to 1 fiber/ml that have been made by individual researchers or by national review groups[3]. There are obviously substantial uncertainties in estimates of K_L. This uncertainty arises first, because only limited data exist on workplace asbestos exposures prior to the mid 1960s. Also when available, the data were usually expressed in particle counts, rather than in fiber counts. The relationship between particle and fiber counts is highly uncertain. When early data were unavailable, estimates of exposure have had to be made, based on later fiber counts and on information on changes in control procedures and exposure conditions over time. These adjustments too, are uncertain. Finally, uncertainties exist in the expected lung cancer risks, which depend on smoking habits and, perhaps, on exposures to lung carcinogens other than asbestos. Despite all these uncertainties, many groups have made estimates

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/ml 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[1]. **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.

of risk parameters. The results (Table 2) indicate remarkable consistency within an industry and even across industries. The risk of lung cancer per cumulative fiber exposure is very similar for all exposure circumstances except chrysotile mining and milling. Risks in the three chrysotile textile manufacturing studies ranged from 1% to 2.6% per 1-year 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[7].

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% pure chrysotile. The origin of this lower risk in the miners and millers 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 lung cancer risk for chrysotile similar to that 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 lung cancer risk for each year of exposure to 1 fiber/ml ($K_L = 0.01$). Higher fiber exposure circumstances or longer periods of

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[1]. **Adjusted to the US male cancer rates in 1970.

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 then a measure of the relative risk of mesothelioma at a given level of fiber exposure. Were mesotheliomas produced only by amphiboles, one would expect large differences in the mesothelioma: lung cancer ratio between populations with pure chrysotile exposure and those with extensive amphibole exposure. Table 3 summarizes this ratio by fiber type usage for more than 40 studies.

The ratio of mesothelioma to lung cancer is seen in this analysis to be the same, within statistical uncertainty, for exposures to 100% chrysotile, 97%+ chrysotile, 100% amosite, and mixtures of chrysotile, amosite, and crocidolite. Only 100% crocidolite exposures appear to have a greater ratio, about two to four times above that of predominantly chrysotile exposures. This relatively small difference in the potential for crocidolite to produce mesotheliomas cannot explain the high mesothelioma risk seen in chrysotile exposures that are accompanied by only a very small exposure to crocidolite. Canadian chrysotile that is amphibole-free

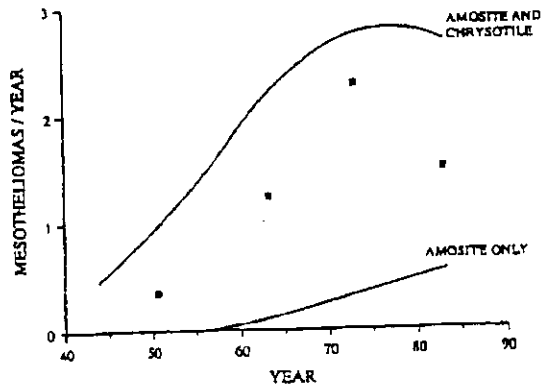

Fig. 1. Estimated and observed cases of mesothelioma/year. 1943–1987 for insulators exposed to amosite alone and amosite/chrysotile. Estimated numbers (smooth curves) are from models of asbestos cancer risk. The solid square represents the number of mesothelioma cases actually observed.

still is asociated with mesothelioma[1]. These data strongly suggest therefore that much of the mesothelioma risk in populations with predominantly chrysotile exposures is due to chrysotile[2].

The mortality risk of mesothelioma from exposure to asbestos can be described by a widely accepted mathematical model (Fig. 1). In this model there is a very low risk of mesothelioma for approximately 20 years from first exposure to asbestos. Thereafter, however, the risk rises very rapidly such that in long-term asbestos-exposed populations mesothelioma may actually become the dominant cancer risk.

Chronologic information is available from two groups of

insulation workers that allows consideration of the contribution of chrysotile to mesothelioma risk. The essence of this analysis is a comparison of the time-course of appearance of mesotheliomas with the time-course of use of (1) chrysotile and (2) other types of asbestos. The analysis takes advantage of the fact that prior to 1935 insulation workers in the United States were exposed only to chrysotile. From 1935 until 1940 they were exposed mainly to chrysotile and only occasionally to amosite. Regular exposure to mixtures of chrysotile and amosite began only after 1940. Because of the strong dependence of mesothelioma risk to time elapsed since exposure, exposures to amosite would not therefore be expected to contribute substantially to a mesothelioma risk in this workforce before the mid-1950s. Observations of mortality from mesothelioma prior to that time would relate directly to chrysotile.

The analysis was undertaken in the 632 members of the New York and New Jersey locals of the Insulator's Union who were members on January 1, 1943[2]. The majority of the group were first employed prior to 1923; for two individuals first employment as an insulator began in 1888. Follow-up of the group has continued from 1943 until the present. Through 1992, all but 40 of the 632 were deceased.

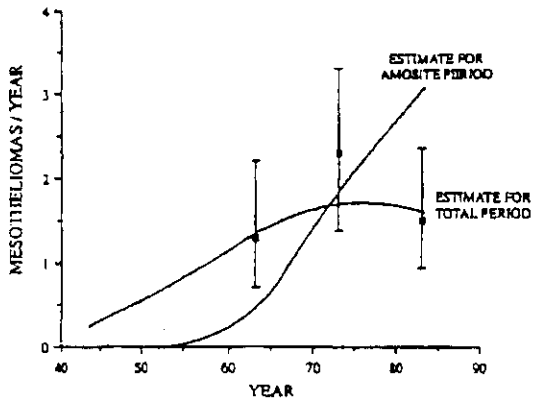

Fig. 2. Estimated and observed cases of mesothelioma/year, 1943–1987. Estimated mesothelioma risk (cases/year) adjusted to yield 56 deaths. The solid squares represent the number of mesothelioma cases actually observed.

The model given above for mesothelioma risk and for estimates of insulation workers' exposure was used to calculate the expected numbers of mesotheliomas for the period from 1943 to 1987 among this cohort. This calculation was done for two periods of time. In one calculation, only exposures subsequent to 1935 were considered ("amosite period") in the other, all asbestos exposures to individuals in the group were considered, from their first exposure to chrysotile asbestos until termination of employment or death ("total period"). The numbers of mesothelioma deaths observed were compared to the number that would be expected if amosite were the sole cause of the disease.

Figure 2 shows the results of these calculations. 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 distribution that would be expected, were amosite exposure responsible for mesothelioma in this population. Barring undiscovered exposures to amphiboles prior to 1935, these data present strong evidence that chrysotile is the substantial, indeed the dominant contributor to the mesothelioma risk experienced by this group of insulation workers[2].

The study by Selikoff *et al.*[10] of the entire union membership of US and Canadian insulators also strongly indicates the existence of a mesothelioma risk associated with exposure to chrysotile. 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 a period of at least 50 years. Were only amosite contributing to risk, this pattern would not be expected. The pattern relates clearly to chrysotile.

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

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}

mesothelioma/fiber exposure, as measured by K_M, is virtually the same for exposures to 98% chrysotile + 2% 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. The value for a pure crocidolite exposure, as calculated by de Klerk and Armstrong[7] 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.

The problem of mesothelioma from exposure to chrysotile continues to the present time. Peto *et al.*[11] predict that deaths from mesothelioma among men in Western Europe will increase from just over 5,000 in 1998 to about 9,000 by the year 2018. In Western Europe alone, past asbestos exposure will cause a quarter of a million deaths from mesothelioma over the next 35 years. The number of lung cancer deaths in Western Europe over the next 35 years[10]. In Sweden, Jarvholm has reported that the number of deaths caused each year by malignant mesothelioma is greater than the number of deaths caused in that country by all workplace injuries.

Current World Uses of Asbestos[13]

Asbestos usage worldwide is extensive, but there have been dramatic changes in the production and use of asbestos in recent years. Table 5 gives representative production data for the past three decades[14]; these data have substantial uncertainties because of limited information on Russian production. The data clearly indicate a rising world production until the late 1970's. After that time a steady decline ensued. Between 1963 and the middle 1970's amphibole mineral constituted from 5% to 7% of the total asbestos production. In subsequent years, the percentage drop in amphibole usage was considerably greater than that for chrysotile. For example, at peak production South Africa mined 269,000 tons of amphibole in 1978, but less than 47,000 in 1991, of which 20,000 was crocidolite. Amosite production ceased in 1992.

Currently over 95% of all asbestos used worldwide is chrysotile, with crocidolite being used only for very specialized purposes. Table 6 shows the 17 top chrysotile consuming nations in 1994[15]. France and other European nations have since banned asbestos from use. The current primary users of chrysotile are countries in Asia and Central/South America. Substantial use also continues in the Middle East and Eastern Europe. While there has been an overall decline in chrysotile asbestos usage, a decline has not occurred in all countries. The decline has been substantial in Western Europe and the United States, but an increase has taken place in Asian nations. Some of these changes with over time can be seen in the data on annual imports and production of asbestos in selected nations (Table 7)[14].

Parallel with the dramatic changes in the global distribution of asbestos, there have occurred changes in its uses.

Table 5. World production of asbestos

Year	Production (tonnes)
1963	2,922,000
1973	4,614,000
1978	5,159,000
1983	4,276,000
1988	4,323,000
1993	2,650,000
1994	2,410,000
1995	2,308,300 (a)
1996	2,140,000 (a)

(a) Chrysotile only.

Table 6. Principal chrysotile consuming countries in 1994

Nation	Annual usage (tonnes)
Commonwealth of Independent States	700,000
China	220,000
Japan	195,000
Brazil	190,000
Thailand	164,000
India	123,000
South Korea	85,000
Iran	65,000
France	44,000
Indonesia	43,000
Mexico	38,000
Columbia	30,000
Spain	29,000
USA	29,000
Turkey	25,000
Malaysia	21,000
South Africa	20,000
Total	2,021,000

Table 7. Annual imports and production of asbestos, in tonnes, in selected countries, by year

Country	1970	1975	1980	1985	1994 (b)
Japan	311,274	261,841	309,305	264,619	195,000
Thailand	21,271	43,024	58,756	75,516	164,000
India	56,008	63,240	99,010	123,000	
South Korea	35,292	56,960	36,787	57,143	85,000
Taiwan	6,589	13,363	31,247	24,519	
Mexico	40,460	60,981		54,871	38,000
France	151,846	138,637	127,123	68,827	44,000
United States	660,000	538,000	162,000	29,000	

(b) Chrysotile only; from reference (15).

Previously, asbestos was used in a wide variety of products. Now, however, asbestos use is largely concentrated in relatively few products, which vary from nation to nation. Worldwide, the dominant use of asbestos is in cement products. For example, in Japan 93% of all asbestos is used in various fire-retardant wallboards and 3.6% in friction products. In developing nations, such as those in Southeast Asia, asbestos-cement pipes are of importance for water systems. South Korea, on the other hand, has extensive asbestos textile and friction product industries and ships the finished products to Japan, West Europe and the United States[16].

There are also differences in national responses to the health hazards of asbestos. Scandinavian and other Western European nations have prohibited all new uses of asbestos. In the United States the permissible exposure level is 0.1 f/ml. In contrast, Japan and South Korea each have a standard of 2 f/ml for chrysotile; amosite and crocidolite use are prohibited in Japan, but lower standards apply in South Korea for amosite (0.5 f/ml) and crocidolite (0.2 f/ml). Generally, developing nations have permissible exposure levels greater than 1 f/ml.

At the international level, the International Labour Organization[17] has published a Recommendation Concerning Safety in the use of Asbestos in which they advocate the banning of new uses of crocidolite and urge controls on the use of other asbestos minerals. They particularly advocate the use of substitute materials and alternative technologies.

With the use of asbestos being predominantly in cement products, a good opportunity for control of workplace and environmental exposures exists. During normal use of such products there is limited release of fibers, and during installation exposures can be well controlled with the use of appropriate dust collectors. However, this may not always be done and workplace monitoring by regulatory agencies is important. Uncontrolled sawing can produce concentrations in excess of ten f/ml.

Conclusion: The Need for an International Ban on Chrysotile Asbestos

Chrysotile is the dominant form of asbestos in use today around the world. Evidence is incontrovertible that chrysotile asbestos is a potent cause of both lung cancer and malignant mesothelioma[1-5]. Data to support this case derive from more than 40 studies of different 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 two to four times greater to produce mesothelioma for equal exposure than chrysotile[3].

The Collegium Ramazzini, an independent international scholarly society in occupational medicine has called for an immediate international ban on the mining and use of chrysotile and all other forms of asbestos. We strongly support that call.

An immediate international ban on the mining and use of chrysotile and all other forms of asbestos is necessary because the risks cannot be controlled by technology or by regulation of work practices. The strictest occupational exposure limits in the world for chrysotile asbestos (0.1 f/ml) are estimated to be associated with lifetime risks of 2.3/1,000 for lung cancer and 0.8/1,000 for asbestosis[18]. These exposure limits

can be technically achieved in the United States and in a few other highly industrialized countries, but the residual risks still are too high to be acceptable. In newly industrializing countries engaged in mining, manufacturing, and construction, asbestos exposures are often much higher, and the potential for epidemics of asbestos disease is greatly increased[18, 19].

Scientists and policy makers in these countries should be wary of claims that asbestos can be used "safely". Those claims are not supported by data. Moreover, even the best workplace controls cannot prevent occupational and environmental exposures to products in use or to waste asbestos. Environmental exposure from the continued use of asbestos is also a serious and continuing problem. A recent study of women residing in communities in Canadian chrysotile mining areas found a seven-fold increase in the mortality rate from pleural cancer[21]. Large quantities of asbestos remain as a legacy of past construction practices in many thousands of schools, homes and commercial buildings in developed countries, and are now accumulating in thousands of communities in developing countries.

An immediate international ban on mining and use of all forms of asbestos is necessary because country-by-country actions have shifted rather than eliminated the health risks of asbestos. The asbestos industry has a powerful influence over public policy in many countries. In the United States, the asbestos industry succeeded in 1991 in overturning the EPA's recommended ban and phase-out of asbestos by a technical ruling in the courts. Canada, Russia, and other asbestos-exporting countries have developed major markets in the newly industrializing nations. Conditions of current asbestos use in developing countries now resemble those that existed in the industrialized countries before the dangers of asbestos were widely recognized.

The grave health hazards of asbestos are entirely preventable. The health risks of asbestos exposure are not acceptable in either industrially developed or newly industrializing nations. Moreover, suitable, safe substitutes for asbestos are available. An immediate worldwide ban on the production and use of asbestos is long overdue, fully justified and absolutely necessary.

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