Review Article

The Carcinogenicity of Chrysotile Asbestos—A Review

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Abstract: The world production of asbestos has been declining dramatically in recent years, particularly in Europe and the United States. However, increases have occurred in Asian nations and chrysotile is the dominant fiber used. Important uses are in cement products, wallboards, friction products and textiles. From studies in the United States and Great Britain, chrysotile has been shown to increase the risk of lung cancer and to produce mesotheliomas in exposed workers.

Key words: Lung cancer, Chrysotile, Mesotheliomas, Exposure, Risk

Introduction

There have been dramatic changes in the production, use, and exposure to asbestos in recent years. Table 1 gives representative production data for the past three decades. The numbers in Table 1 have substantial uncertainties because of limited information on early Russian production, but they clearly indicate a rising world asbestos output until the late 1970’s after which a continuing decline set in. Between 1963 and the middle 1970’s amphibole minerals constituted from 5% to 7% of the above asbestos production. In 1978, when a decline in the total usage of asbestos began, the individual amphibole composition was 3.8% crocidolite, 1.3% amosite and 0.2% anthophyllite. In the 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 tonnes of amphibole in 1978, but less than 47,000 in 1991, of which 30,000 was crocidolite. Amosite production ceased in 1993. Currently, approximately 99% of all new asbestos use involves chrysotile, with crocidolite being used only for very specialized purposes. From the late 1970’s to 1996, total asbestos production declined by more than two-fold, but amphibole usage decreased by greater than a factor of ten.

Because of its dominant presence in new materials, the following discussion will be confined to consideration of trends in chrysotile use, exposure and effects. Table 2 shows the 17 top chrysotile consuming nations in 1994. Europe has since banned asbestos from new uses. Thus, the current primary users of chrysotile are countries in Asia and Central/South America. Substantial use also continues in some Middle Eastern nations.

While there has been an overall decline in asbestos usage, it has not occurred in all countries. The decline has been dramatic in Western Europe and the United States, but a general increase has taken place in Asian nations. Some explicit regional changes with time can be seen in the annual imports and production of asbestos in selected nations, which are shown in Table 3. The countries chosen were major using countries whose production was small in comparison

<table>
<thead>
<tr>
<th>Year</th>
<th>Production (tonnes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1961</td>
<td>2,352,000</td>
</tr>
<tr>
<td>1973</td>
<td>4,814,000</td>
</tr>
<tr>
<td>1974</td>
<td>3,139,000</td>
</tr>
<tr>
<td>1982</td>
<td>4,276,000</td>
</tr>
<tr>
<td>1984</td>
<td>4,320,000</td>
</tr>
<tr>
<td>1993</td>
<td>2,510,000</td>
</tr>
<tr>
<td>1992</td>
<td>2,410,000</td>
</tr>
<tr>
<td>1995</td>
<td>2,308,300</td>
</tr>
<tr>
<td>1996</td>
<td>2,129,000</td>
</tr>
</tbody>
</table>

(1) Estimated only
Table 2. Principal chrysotile consuming countries, 1984

<table>
<thead>
<tr>
<th>Nation</th>
<th>Annual usage (tonnes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Russia</td>
<td>700,000</td>
</tr>
<tr>
<td>China</td>
<td>220,000</td>
</tr>
<tr>
<td>Japan</td>
<td>195,000</td>
</tr>
<tr>
<td>Brazil</td>
<td>180,000</td>
</tr>
<tr>
<td>Thailand</td>
<td>160,000</td>
</tr>
<tr>
<td>India</td>
<td>130,000</td>
</tr>
<tr>
<td>South Korea</td>
<td>85,000</td>
</tr>
<tr>
<td>Iran</td>
<td>65,000</td>
</tr>
<tr>
<td>France</td>
<td>44,000</td>
</tr>
<tr>
<td>Indonesia</td>
<td>43,000</td>
</tr>
<tr>
<td>Mexico</td>
<td>34,000</td>
</tr>
<tr>
<td>Colombia</td>
<td>30,000</td>
</tr>
<tr>
<td>Spain</td>
<td>29,000</td>
</tr>
<tr>
<td>USA</td>
<td>28,000</td>
</tr>
<tr>
<td>Turkey</td>
<td>23,000</td>
</tr>
<tr>
<td>Malaysia</td>
<td>21,000</td>
</tr>
<tr>
<td>South Africa</td>
<td>20,000</td>
</tr>
<tr>
<td><strong>Total above</strong></td>
<td><strong>3,021,000</strong></td>
</tr>
</tbody>
</table>

For expanding water supply and sewage systems. However, in Japan 93% of all asbestos is used in various fire-retardant wallboards and 3.6% in friction products10. South Korea has extensive asbestos textile and friction product industries and ships the finished products to Japan, West Europe and the United States11.

There are also differences in national responses to the health hazards of asbestos. Some 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 and asbestos use has been dramatically curtailed.

In contrast, Japan currently has a 2 f/ml for chrysotile but a recommendation has been made to lower the Permissible Exposure Level to 0.15 f/ml by the Japan Society for Occupational Health. The uses of amosite and crocidolite are prohibited in Japan. South Korea has a 2 f/ml standard for chrysotile but lower standards apply for amosite (0.5 f/ml) and crocidolite (0.2 f/ml). Generally, developing nations have permissible exposure levels greater than 1 f/ml.

With the use of asbestos being predominantly in cement products, a good opportunity for control of workplace and environmental exposures exists. In installation of pipes and boards, exposures during sawing or other abrasive actions can be well controlled with the use of appropriate dust collectors or wetting techniques. However, these precautions may not always be taken and workplace monitoring by regulatory agencies is important. Uncontrolled sawing produces concentrations in the tens of f/ml. During normal use of such asbestos cement products there is limited release of fibers because of the strong binding of the cement. Again, however, abrasion of the cement will lead to fiber release.

One feature of current occupational exposures is that there has been a substantial decrease of the use of asbestos in thermal insulation. Such products are particularly dangerous because asbestos is readily released. Such release during

Table 3. Annual imports and production of asbestos, in tonnes in selected countries, by year

<table>
<thead>
<tr>
<th></th>
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<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Japan</td>
<td>311,274</td>
<td>263,816</td>
<td>309,305</td>
<td>284,617</td>
<td>195,000</td>
</tr>
<tr>
<td>Thailand</td>
<td>21,177</td>
<td>43,024</td>
<td>48,736</td>
<td>75,316</td>
<td>164,000</td>
</tr>
<tr>
<td>India</td>
<td>56,000</td>
<td>62,270</td>
<td>38,875</td>
<td>99,010</td>
<td>53,000</td>
</tr>
<tr>
<td>South Korea</td>
<td>55,292</td>
<td>36,960</td>
<td>37,787</td>
<td>57,143</td>
<td>85,000</td>
</tr>
<tr>
<td>Taiwan</td>
<td>6,590</td>
<td>12,563</td>
<td>31,247</td>
<td>74,519</td>
<td></td>
</tr>
<tr>
<td>Mexico</td>
<td>40,480</td>
<td>65,167</td>
<td>54,671</td>
<td>38,700</td>
<td></td>
</tr>
<tr>
<td>France</td>
<td>121,415</td>
<td>35,872</td>
<td>127,122</td>
<td>62,827</td>
<td>64,000</td>
</tr>
<tr>
<td>United States</td>
<td>440,000</td>
<td>213,000</td>
<td>162,000</td>
<td>213,000</td>
<td></td>
</tr>
</tbody>
</table>

10: Chrysotile only. From reference 12.
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Insulation, repair or removal not only exposes the insulation, but also many other workers in shipyards and construction sites. In the United States, it has been estimated that approximately 70% of current asbestos-related cancers can be attributed to fibers released from thermal insulation materials.

A general feature of current asbestos use is a lesser concern for exposures to chrysotile than to the amphiboles. This is a serious mistake. Available data indicate a similar lung cancer risk per fiber exposure for chrysotile, amosite and crocidolite. There is no question concerning the greater carcinogenicity of crocidolite for mesothelioma, but very strong data from an analysis of the time course of mesothelioma risk among U.S. insulation indicates similar, substantial risks for exposures to amphibole and chrysotile. Let us consider some of that information.

Asbestos-Related Lung Cancer

It is widely accepted that exposure to chrysotile asbestos increases the risk of developing lung cancer in proportion to the cumulative exposure to asbestos up to a time 10 years prior to evaluation. The relationship can be expressed formally by:

\[ I = L (1 + K_i \cdot f_i \cdot d_i) \]

where \( I \) is the lung cancer incidence or mortality in a study population at time of evaluation, \( L \) is the age- and calendar-year-specific lung cancer incidence or mortality expected in the same population in the absence of asbestos exposure (ideally, \( L \) would explicitly consider smoking habits of each study individual); \( f_i \) is the intensity of asbestos fibers longer than 5 \( \mu \)m per ml; \( d_i \) is the duration of exposure in years up to a period 10 years prior to evaluation; and \( K_i \) is a proportionality constant that is a measure of the carcinogenic potency of the asbestos exposure. \( K_i \) represents the fractional increase in lung cancer incidence or mortality that occurs from a 1-year exposure to 1 fiber/ml.

Exposure-response relationships have been developed between asbestos exposure and lung cancer risk in several epidemiological studies. These are summarized in Table 4.

The individual studies in the above table vary substantially in their statistical quality. Some, such as those of textile production or insulation work, involve substantial exposures and large study populations. In such cases, the measures of risk are relatively good. In other, such as those of brake products manufacturing, the exposures were low and large uncertainties exist. In one of the brake studies, a high overall SMR was seen, but there was not a clear dose-response relationship according to exposure, although we are dealing with only six cases for four dose categories. In all studies we must use a relatively limited number of exposure estimates made from particle counts in earlier years.

Considering all studies, except those of mining and milling, the geometric mean value of \( K_i \), the percentage increase in lung cancer for a 1-year exposure to 1 fiber/ml is 1.0. The value for chrysotile mining and milling is approximately two-fold less. Comparing chrysotile mining and milling with chrysotile textile production, the difference is even greater. The complete understanding of this difference is not known at this time. Some of the differences may be the result of numerous fiber bundles being present in the mining and milling environment. These are easily counted, but some of them may not be inspired. In the textile environment, the bundles are opened, producing an environment with a greater percentage of individual carcinogenic fibers, which may not be counted, but which are readily inspired.

| Table 4. Risk of lung cancer in workers exposed to asbestos minerals |
|------------------------|---------------------|---------------------|
| Asbestos exposure       | Study refs          | Type of asbestos     | Percentage increase in lung cancer for 1 year exposure to 1 fiber/ml |
| Circumstances           |                    |                     | 10-200 |
| Textile manufacturing   | 2, 9, 10           | 95% chrys          | 1.0-2.0 |
| Asbestos insulation     | 11                 | Asbestos           | 1.0-2.0 |
| Manufacture             | 12                 | Americium          | 0.8    |
| Asbestos production     | 12, 14, 13         | Chrysotile         | 0.5-6.7 |
| Product production      | 18, 17             | Chrysotile         | 0.01-6.06 |
| Asbestos mining         | 18, 19, 20         | Chrysotile         | 0.01-6.06 |
| Crocidolite mining      | 22                 | Crocidolite        | 1.0    |
Malignant Mesothelioma

The risk of mesothelioma by fiber type can be analyzed in three ways. Firstly, since the lung cancer risk is very similar for all fiber types, excluding mixing 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 cancers is a measure of the relative fiber exposure mesothelioma risk. Secondly, one can use the unique time dependence of mesotheliomas in mixed exposure circumstances to attribute risk to different fiber exposures in different periods of time. Finally, by utilizing a mathematical risk model, in an analogous fashion to what was done for lung cancer to produce Table 4, one can directly calculate a mesothelioma unit fiber exposure risk. We will utilize each of these procedures to assess the mesothelioma risk from different fiber exposures.

Estimates of relative mesothelioma risk by fiber type

The Asbestos Health Assessment Update of the U.S. Environmental Protection Agency used the first method to estimate the relative mesothelioma potency for asbestos fibers. In studies where the mesothelioma risk cannot be estimated directly, it is found that the ratio of the number of mesotheliomas to excess lung cancers is very similar for most studies, within the uncertainties of the estimations. When mesotheliomas produced only by amphiboles, one would have expected large differences in the mesothelioma risk between pure chrysotile studies and those with extensive amphibole use. Table 5, from Nicholson and Raff, summarizes this ratio, by fiber type usage, for the more than 40 studies for which little or no exposure information is available.

In Table 5, we use the excess number of lung cancers as a measure of exposure and compare the ratio of mesotheliomas to excess lung cancer across these studies. In doing so, however, one has to adjust the excess numbers of lung cancer to the same underlying risk of lung cancer. This is necessary because the excess number of lung cancers is proportional to both the cumulative exposure and the expected lung cancer risk. It can be seen that the ratio of mesotheliomas to excess lung cancer is the same for exposures to 100% chrysotile. 97% chrysotile, 100% amosite and mixtures of chrysotile, amosite and crocidolite, within statistical uncertainty. Only 100% crocidolite exposures appear to have a greater ratio, about two to four times that of predominantly chrysotile. This relatively small difference in the potential for crocidolite to produce mesotheliomas compared with other fiber exposures cannot explain the high risk seen in chrysotile exposures accompanied by a very small crocidolite exposure. The data speak strongly that much of the mesothelioma risk in predominantly chrysotile exposures is from the chrysotile.

Analyses utilizing the same course of mesothelioma risk

The mortality risk of mesothelioma from exposure to asbestos can be described by a mathematical model that is widely accepted. It was used for regulatory purposes by the U.S. Consumer Product Safety Commission, the U.S. Environmental Protection Agency, and the U.S. Occupational Safety and Health Administration. It was the model in the recent review of the Health Effects Institutes/Asbestos Research. In this model the risk of mesothelioma, R_m, is given by:

\[ R_m = K_{d} \cdot f \cdot \left( \frac{t-10}{t} \right) \]

for \( t \geq 10 \) (Eq. 2a)

\[ R_m = K_{d} \cdot f \cdot \left( \frac{t}{t-10} \right) \]

for \( 10 < t < 100 \) (Eq. 2b)

\[ R_m = 0 \]

for \( t < 10 \) (Eq. 2c)

Here \( R_m \) is the mesothelioma mortality rate at \( t \) years from

<table>
<thead>
<tr>
<th>Type of exposure</th>
<th>Number of studies</th>
<th>Mesotheliomas</th>
<th>Lung cancer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chrysotile</td>
<td>8</td>
<td>0.13</td>
<td>0.14</td>
</tr>
<tr>
<td>Predominantly chrysotile</td>
<td>6</td>
<td>0.26</td>
<td>0.48</td>
</tr>
<tr>
<td>Asbestos</td>
<td>2</td>
<td>0.13</td>
<td>0.22</td>
</tr>
<tr>
<td>Predominantly crocidolite</td>
<td>6</td>
<td>0.46</td>
<td>0.61</td>
</tr>
<tr>
<td>Amphibole</td>
<td>1</td>
<td>0.80</td>
<td>0.80</td>
</tr>
<tr>
<td>Asbestos</td>
<td>2</td>
<td>0.80</td>
<td>0.80</td>
</tr>
<tr>
<td>Mixed exposures</td>
<td>16</td>
<td>0.19</td>
<td>0.47</td>
</tr>
</tbody>
</table>

*Adjusted to the U.S. male cancer rates in 1970*
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onset of exposure to asbestos for a duration of years at a
concentration (fibers/ml). \( K_a \) is a proportionality constant
related to carcinogenic potency and may depend on fiber
type. For exposures that vary with time, risks for each
separate period are additive. Alternative models have been
used without the lag period of 10 years. These have similar
formulas, but without the factor of 10, and a power of 4 or
higher rather than 3.

Information is available from two groups of workers that
allows consideration of the contribution of chrysotile to
mesothelioma risk by consideration of its time of use
compared to other fibers. United States insulation workers,
while exposed to mixtures of chrysotile and amosite since
1940, also can provide information on health effects related
to pure chrysotile. This arises because their exposure to
asbestos prior to 1937 was to only chrysotile and until 1940
only occasionally to amosite. Because of the strong
dependence of mesothelioma risk with time as indicated by
Eq. 3, exposures to amosite would not be expected to
contribute substantially to a mesothelioma risk until the mid-
1950's. Observations of insulator mortality in the period of
low amphibole mesothelioma risk relate directly to chrysotile
risk. This time course of mesothelioma risk was utilized by
Nicholson and Landrigan21 to compare the expected and
observed mesothelioma rates in the New York and New Jersey
insulation worker cohort established by Selikoff, Churg and
Hammond3. The comparison considered mesothelioma risks
expected from the workers' total exposure to asbestos and
to the exposure from amphiboles, predominantly amosite,
beginning in the late 1930's.

The 632 members of the New York and New Jersey locals
of the insulation union who were members on January 1,
1943 are an ideal group to study the effects of exposure
periods with different asbestos types. The majority of
the group were first employed prior to 1923. Follow-up of
the group extends from 1943 until the present. First
employment as an insulation began in 1933 for two members of the group.
389 were first employed before 1920. Through 1992, all
but 40 of the 632 were deceased. Furthermore, substantial
effort was made to obtain tissue specimens and medical
records to validate the causes of death of cohort members
deceased over the years. Pathological material was reviewed
in the early years of follow-up by Drs. Jacob Churg and
Milton Kanterstein, then members of the U.S. panel on
mesothelioma, and since the 1970's by Dr. Yasuomi Suzuki.
Thus, the analysis does not suffer as much from
the inadequacies of mesothelioma diagnosis that were
common prior to the mid-1960's in the analyses of other
groups for which only death certificate information was
available.

The analysis of Selikoff's original cohort considered only
the 56 identified deaths from mesothelioma, virtually all of
which were confirmed by one of the above pathologists.
The model given above for mesothelioma risk and estimates
of insulation workers' exposure were used to calculate the
expected numbers of mesotheliomas, 1943-1987, among
the cohort of 632 NY-NJ Insulators. This was done for two
periods of time. In one, only exposures subsequent to 1935
were considered, reflecting exposure to amosite; 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. Figure 1 shows
the results of these calculations of expected mesothelioma
cases, by year, compared with the actual numbers of
mesotheliomas observed. Because of uncertainties in the
relative amounts of amosite and chrysotile after 1935, the
expected cases were adjusted to represent the observed 56
total mesotheliomas. The actual estimate for amosite alone
was less than one-fifth that observed. Data points represent
an average of 15 years for the first point and an average
over ten years for the remaining three. As can be seen, the
time course of mesothelioma risk is totally incompatible
with an exposure pattern that begins in the late 1930's.
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

Fig. 1. Estimated and observed cases of mesothelioma/year, 1943-
1987.
Estimated mesothelioma risk curves adjusted to yield 56 deaths. Of
the four displayed points, the first represents 15 years of the follow-up
and the remaining three 10 years each.

In 1935, the data present strong evidence that chrysotile is a substantial, indeed, the dominant contributor to the mesothelioma risk experienced by this group of insulation workers.

The study by Salikoff, Hammond and Seidman with the entire union membership of U.S. and Canadian insulators also strongly reflects a chrysotile mesothelioma risk. Follow-up for the study began in 1967, 30 years after the earliest incorporation of asbestosis by insulation manufacturers into their products. Figure 2 displays the data on mesothelioma risk for the ten-year period, 1967-1976 (the crossed circles). The observed data match a risk estimate made using Eq. 2 and a value of $K_w = 1.5 \times 10^{-4}$ (the heavy line) for the full asbestos exposure period of the insulators, as indicated by the (upper) "years from onset of any asbestos exposure." This match, considering all asbestos exposures, strongly suggests that the pre-1937 exposures of insulators solely to chrysotile contributed substantially to the overall mesothelioma risk.

To fully appreciate the chrysotile effect, consider a hypothetical one-year follow-up during 1967 of these insulators with the assumption that only amosite contributes to their mesothelioma risk, according to Eq. 2. Since we are considering at this time a one-year follow-up, let us also assume an amosite potency that would match estimated risks to the observed insulator mesothelioma risks for the first 30 years from onset of any asbestos exposure (that allows us to use the same graph). Note that these first 30 years correspond to first asbestos exposures in the calendar years, 1937-1966, the years in which amosite was contained in insulation and during which our hypothetical amosite can explain the observed risks. The vertical line identifies the risk for an individual first exposed to amosite in 1937. However, all individuals employed as an insulator before 1937 also had their first exposure to amosite in 1937 and would have the same 30 years from amosite exposure risk, even though their first exposure to chrysotile was 40, 50 or more years previously. The characterization of time from onset of amosite exposure would accord with the "lower" "years from onset of amosite exposure as of 1967." Thus, it would be expected that the risk for this hypothetical one-year follow-up of this cohort would follow the indicated horizontal line.

In actuality, the depicted insulator data are for a 10-year follow-up period and by 1976 some insulators would have had 40 years from onset of amosite exposure. Consideration of the full 10-year follow-up period results in a hypothetical amosite risk curve for the group indicated by the 1967-1976 curve. Considering risk according to time from onset of any asbestos exposure, there is a dramatic difference between an amosite only effect and a combined effect from amosite and chrysotile. Instead of a sharp break of the heavy line at 30 years from onset of exposure, there is an unwavering continuation indicating the substantial contributions of exposures prior to 1937. The data strongly indicate a chrysotile contribution to mesothelioma risk equal to that of amosite.

**Direct calculation of mesothelioma risk**

Finally, one can make an estimate of chrysotile mesothelioma risk from direct calculations of mesothelioma risk in mixed exposure circumstances. Data on duration and intensity of exposure are available in five exposure circumstances that allow one to utilize Eq. 2 and calculate values of $K_w$. The results are shown in Table 4.

These results show that risk of mesothelioma per fiber
exposure, as measured by \( K_w \), is virtually the same for exposure to 97% chrysotile = 3% crocidolite, 60% chrysotile = 40% amphibole, and 100% amphibole. The value of \( K_w \) from the cement workers study of Finkelstein is higher than the chrysotile-amphibole exposures as was a value of \( K_w \) in the same group of workers. As noted previously, there may substantial errors in the exposure estimates of the study. The value for a pure crocidolite exposure, as calculated by de Klerk and Armstrong\(^h, i, j, k, l^1\) for the mixing population of Australia, is about ten times greater. As with the values of \( K_w \) in Table 4, \( K_w \) is uncertain because of uncertainties of exposure in the early exposure years of the groups under study and from uncertainties of small numbers. Indeed, from data to be considered below, the mesothelioma potency of crocidolite would appear to be less than ten times that of other fibers. Nevertheless, in contrast to the analysis of crocidolite lung cancer risk in comparison to other fibers, the data do indicate a clearly greater mesothelioma potency for crocidolite.

However, \( K_w \) is not so much greater for crocidolite that it is likely to be the dominant cause of the mesotheliomas found among the textile workers of the Rochdale, England plant. There, crocidolite was brought into the plant as yarn and the raw fiber was not opened, carded or spun to a significant extent. Overall, from 1932-1968, 2.6% of the asbestos fiber purchased for use at Rochdale was crocidolite\(^i\). Without evidence of overwhelming greater exposures to crocidolite, an estimate from the above data is that crocidolite might account for about 35% of the total mesothelioma risk at Rochdale. If \( K_w(\text{crocidolite}) = 13 \times K_w(\text{Rochdale}) \), for equal percentage use over time and a contribution to air concentrations equal to the percentage use, the \( K_w(\text{chrysotile}) \) can be estimated from the relation:

\[
(13 \times 0.026) = K_w(\text{Rochdale}) + 0.97 \times K_w(\text{chrysotile}) = K_w(\text{Rochdale})
\]

This yields a value for \( K_w(\text{chrysotile}) \) equal to 0.68 \( \times 10^{-4} \) and a contribution to the total mesothelioma risk at Rochdale of 66%. The chrysotile contribution to mesothelioma at Rochdale is substantial.

A direct estimate cannot be made of the exposure specific mesothelioma risk for Quebec chrysotile miners and millers. Among the miners and millers, the ratio of mesotheliomas to lung cancer suggests a lower risk comparable to that seen for lung cancer. In the case of the South Carolina textile workers with only two mesotheliomas, suggesting a low risk, the possibility of misdiagnosis of the disease on certificates of death must be considered.

**Summary of malignant mesothelioma risks**

The case that chrysotile is a potent causative factor in producing mesotheliomas is a strong one. It is shown to be so in a comparison of more than 40 studies of different fiber exposure circumstances. It is shown to be so when the time course of risk is considered in mixed fiber exposures. Finally, it is shown to be so in direct calculations of risk. All available data suggest that it dominates the risk in those circumstances where 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 four to ten times greater potential to produce mesothelioma for equal exposure than chrysotile. However, the crocidolite risk is not so much greater that one can ascribe total causation to a small percentage of crocidolite fibers in a mixed fiber exposure setting.

**References**

2. Asbestos Institute, Montreal, Quebec, Canada: Chrysotile asbestos: an overview.
Mortality Experience in a Historical Cohort of Chrysotile Asbestos Textile Workers

Carlo Mamo¹ and Giuseppe Costa²

¹ Epidemiology Unit, Piedmont Region, Grugliasco, Turin, Italy
² Department of Public Health, Faculty of Medicine, University of Turin, Italy

ABSTRACT:

Introduction and aims
The issue of whether exposure to chrysotile asbestos alone, without contamination from amphibole asbestos, causes lung cancer, mesothelioma and non malignant diseases was investigated in a historical cohort in Grugliasco, Italy, where the largest Italian asbestos textile factory had been in operation in 1900-86.

Methods
The study cohort comprised 1,653 asbestos textile plant workers exposed to chrysotile only. Vital status was ascertained by means of postal follow-up. The cause of death was ascertained through a record linkage with the national mortality registry. Standardized Mortality Ratios (SMR) were computed using the mortality rates of the Turin working population as a reference (in order to reduce the healthy worker effect and the confounding from social class), adjusted for age and birth area. Observation period went from 1/1/1981 to 31/12/1995.

Results
Overall mortality was significantly in excess, in both males (SMR=212; 119 obs.) and females (SMR=265; 84 obs.). Cancer mortality was significantly in excess (SMR=194 males; SMR=261 females). Statistically significant excesses for pleural mesothelioma (SMR=3322 males; SMR=13248 females) and lung cancer (SMR=302 males; SMR=523 females) were observed. Other sites of cancer in excess were: larynx, stomach, pancreas and brain. Mortality excesses for asbestosis (SMR=12797 males; SMR=3124 females), ischemic heart diseases (SMR=139 males; SMR=164 females) and cerebrovascular diseases (SMR=1597 males; SMR=173 females) were estimated. Analysis for length of employment and year of hire evidenced a correlation between mortality rates and length of employment and the latency period for the tumours.

Conclusions
These results confirm that heavy exposure to pure chrysotile asbestos alone, with negligible amphibole contamination, cause lung cancer and malignant mesothelioma in exposed workers. Moreover, the results suggest, in agreement with previous studies, a role of the exposure to asbestos in the etiology of other sites of cancer (particularly larynx and stomach) and of non malignant diseases (ischemic heart diseases and cerebrovascular diseases).