# Asbestos Exposure—Quantitative Assessment of Risk[1-3]



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**SUMMARY** Methods for deriving quantitative estimates of asbestos-associated health risks are reviewed and their numerous assumptions and uncertainties described. These methods involve extrapolation of risks observed at past relatively high asbestos concentration levels down to usually much lower concentration levels of interest today—in some cases, orders of magnitude lower. These models are used to calculate estimates of the potential risk to workers manufacturing asbestos products and to students enrolled in schools containing asbestos products. The potential risk to workers exposed for 40 yr to 0.5 fibers per milliliter (f/ml) of mixed asbestos fiber type (a permissible workplace exposure limit under consideration by the Occupational Safety and Health Administration (OSHA)) are estimated as 82 *lifetime* excess cancers per 10,000 exposed. The risk to students exposed to an average asbestos concentration of 0.001 f/ml of mixed asbestos fiber types for an average enrollment period of 6 school years is estimated as 5 *lifetime* excess cancers per one million exposed. If the school exposure is to chrysotile asbestos only, then the estimated risk is 1.5 lifetime excess cancers per million. Risks from other causes are presented for comparison; e.g., *annual rates (per million)* of 10 deaths from high school football, 14 from bicycling (10–14 yr of age), 5 to 20 for whooping cough vaccination. Decisions concerning asbestos products require participation of all parties involved and should only be made after a scientifically defensible estimate of the associated risk has been obtained. In many cases to date, such decisions have been made without adequate consideration of the level of risk or the cost-effectiveness of attempts to lower the potential risk.

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## Introduction

Knowledge regarding the health effects of asbestos exposure has been emerging since early in the century, but the recent public concerns over the consequences of this naturally-occurring and useful mineral have been unprecedented. Anxiety concerning these exposures escalated when these risks became associated not only with primary industrial contact (mining, milling, manufacturing), but also with less predictable and more numerous occupational exposures, such as among insulators and shipyard workers, some with only indirect (though perhaps heavy) exposures. Currently, there are two areas of major concern: (*1*) whether today's workplace exposure levels, much lower than in the past, have, in fact, been lowered sufficiently; and (*2*) whether there is a potential risk in nonoccupational circumstances, such as from asbestos products inside public buildings.

Although these concerns are widespread, there has generally been little information available to the public, administrators, and concerned professionals regarding how great the potential risks might be in various circumstances. In fact, the dose-relatedness of the risk is often not well understood, and many have concluded that all exposures may be equivalent to those experienced by heavily exposed workers several decades ago, whose subsequent health effects have justifiably received considerable public attention.

Exposure to asbestos results from many sources, both from the natural environment and from commercial asbestos use, and it has been noted that asbestos fibers can be found in the lungs of almost everyone in the population (1). This fact does not indicate that such low exposures as occur in the general population are necessarily without risk, but it does emphasize that there are many sources of asbestos exposure and that any particular exposure source (e.g., products inside buildings), while contributing to the overall exposure of individuals, may well not be the sole or even primary source of exposure. In fact, asbestos exposure levels in the ambient air, especially in urban areas, can be higher than within buildings containing asbestos products, especially if these products are well-maintained.

While there has been debate in many countries about the potential health risks from both occupational and nonoccupational asbestos exposures, much of the recent concern in the U.S. has focused on exposure in schools. The Environmental Protection Agency (EPA) directed all U.S. schools to test for asbestos-containing products, and if asbestos was found, to notify parents and school employees. To date, however, EPA has provided these schools with neither guidance as to what further action should be taken if asbestos materials are found, nor estimates of the magnitude of the potential risk associated with these products. Inevitably, some parents and school administrators have concluded that these materials pose a high cancer risk and have taken action consistent with this conclusion. Others have postponed action. It is likely that in some instances, asbestos products in excellent condition, emitting few or no fibers, have been removed unnecessarily, while in others, materials emitting fibers at an unacceptably high level have not received attention.

This report will derive estimates of the potential risk from asbestos exposure in two situations: the workplace and schools. In deriving these estimates, important factors and issues in asbestos risk estimation will be discussed, as will the assumptions and uncertainties of the methodology. A summary of risk estimates derived by several governmental advisory groups (2–8) will be presented for comparison purposes and to illustrate the important effect of various factors on the resulting risk values. The findings of these advisory groups, although likely to influence governmental decision-making with respect to asbestos and its future use, have, in general, not reached

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a wide scientific audience, nor the concerned public. Moreover, the risk estimates developed by these groups have been based on widely differing assumptions concerning the level and duration of the asbestos exposure, making comparisons difficult. In this report, the estimates of these groups will be adjusted to common exposure assumptions in order to allow direct comparisons; differences in the risk values will emphasize important differences in the methodologies and assumptions of these advisory groups.

## Methods

Most studies of the effects of asbestos exposure have been of workers exposed to asbestos in industry, most employed many years ago. These asbestos concentration levels were considerably higher than in industry today, and several orders of magnitude higher than environmental exposures. Estimates of risk, especially for environmental asbestos exposures, are therefore not based on direct observation of these risks, but instead, on extrapolation of risk observed at higher levels down to these lower levels.

Estimates of potential asbestos risk will be derived for lung cancer and mesothelioma, two causes of death repeatedly shown to be elevated among past asbestos workers. Some studies of asbestos workers have observed an increased risk of cancer of other sites; e.g., larynx, gastrointestinal tract and kidney (9). These findings, however, have not been consistent and there is considerable controversy as to whether these cancers are associated with asbestos exposure. There is general agreement, however, that any potential excess cancer risk at these sites combined would be relatively small—less than 10% of the excess lung cancer risk.

Asbestosis, when it occurs as the result of relatively recent industrial exposure levels, is generally very slow in progressing and does not now often result in serious disability or in increased mortality. There is no evidence of asbestosis occurring as a result of environmental exposures. Risk of this disease, therefore, will not be considered.

## Results

### *Lung Cancer Risk*

The data from several studies suggest that lung cancer risk, as measured by the Standardized Mortality Ratio (SMR, the ratio of the observed number of cases [O] to the number expected [E] in the absence of asbestos exposure, multiplied by 100; i.e., SMR = 100 [O/E]) is linearly related to cumulative asbestos exposure. Because the SMR should be 100 (no excess) when there is no asbestos exposure, this linear model may be written as:

$$SMR = 100 + bx \qquad (Equation\ 1)$$

where b is the slope of the line and x is the cumulative asbestos exposure (usually recorded in fibers per milliliter – years (f/ml – yr). For example, if b = 2 and x = 10 f/ml – yr, then SMR = 120; i.e., the observed number of cases is 120% of that expected.

Because SMR = 100 (O/E), *Equation 1* may be rewritten in terms of the number of excess cases (O – E) occurring:

$$O - E = E\ (b/100)\ x \qquad (Equation\ 2).$$

It is this last equation that will be used to estimate the potential number of excess cases for a specified cumulative exposure x.

Estimates of the slope b may be obtained from epidemiologic studies that have demonstrated a dose-response relationship (by calculating risk for population subgroups defined on the basis of cumulative exposure level). Demonstration of increasing lung cancer risk with increasing estimated exposure is an important requirement for an epidemiologic study because it allows critical examination of its findings. If observed risk in a population shows no sensible pattern with increasing exposure, then the validity of the exposure estimates and/or the risk estimates must be questioned; if risk is substantially elevated at very low exposure levels, then systematic underestimation of exposure or overestimation of risk must be considered.

Errors in risk estimation can occur as a result of many factors, including incomplete trace of the population, inaccurate ascertainment of causes of death, and use of an inappropriate comparison population for the expected mortality rates. Accurately estimating past exposure levels for workers is always difficult because only limited past exposure measurements may be available, and job records may not fully reflect work activities. Because of these potential problems, demonstration of a gradient of risk with estimated exposure is essential because it provides at least partial validation of the exposure estimates.

There have been 7 industrial cohorts for which a reasonable pattern of risk has been observed for categories of increasing cumulative asbestos exposure (10–16). Estimated slopes from the 7 cohorts vary considerably but exhibit a relationship with industrial process: the lowest values are found in studies of miners and friction product manufacturing workers, higher values for asbestos cement and other manufacturing employees, and the highest among textile workers (figure 1). These results indicate that for the same estimated cumulative asbestos exposure, textile workers have the highest potential lung cancer risk, while miners and friction products workers have the lowest. It is believed that, because of the differing state and physical treatment of the asbestos in these different processes, the dust clouds contain asbestos fibers of differing physical dimension, which is related to the level of carcinogenicity. Specifically, exposures in textile manufacturing are more likely to include long, thin fibers than in mining and other manufacturing. Moreover, animal experiments have demonstrated that long, thin fibers are more pathogenic than short, coarse fibers (17, 18).

### *Uncertainties of the Lung Cancer Model*

The linear model for lung cancer risk at low exposure levels is based on several assumptions: that the linearity of risk observed in industrial cohorts applies to all exposure levels (even very low), that the risk for a cumulative exposure is independent of smoking and remains constant after an initial lag or latency period (usually 10 years), and that risk is linear with respect to both components of cumulative exposure, duration and concentration.

It is doubtful if the shape of the dose-response relationship for low amounts of asbestos exposure can ever be determined by direct observation because of the large number of non-asbestos-related lung cancers in the population. However, the linear model is consistent with observations at higher exposure levels and is supported by the mathematical theory of

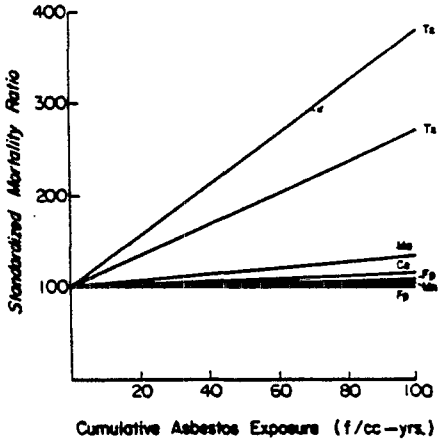


Fig. 1. Estimated lung cancer dose-response relationships from 7 epidemiologic studies (Tx = textiles, Ma = manufacturing, Ce = asbestos cement, Mn = mining, Fp = friction products).

multistage carcinogenicity for low doses of a carcinogen (19). Moreover, there is general agreement that risk is unlikely to increase more rapidly than linearly at low cumulative asbestos levels.

The assumption that asbestos lung cancer risk (as measured by the SMR) is independent of smoking is equivalent to assuming that the effect of asbestos is the same among smokers as among nonsmokers, i.e., that asbestos exposure increases the lung cancer risk among smokers and nonsmokers by the same percentage. Contrary to this assumption, some of the available data suggest, though not conclusively, that the percentage increase among smokers may be somewhat less than among nonsmokers (20, 21). Similarly, there is limited evidence that the assumption of a constant relative risk (observed number/expected number) over time may not be valid: data from North American insulation workers (22) exhibit a decline in the relative risk for lung cancer after 35 years from initial exposure. Both these issues remain controversial and cannot yet be resolved from the available evidence.

Although there are some data (10, 15) to support the assumption that risk is linearly related to both concentration and duration of exposure, the evidence to date is quite limited.

In spite of these uncertainties, the assumptions of the lung cancer model are conservative in the sense of possibly leading to an overestimate of potential risk but, in all likelihood, not underestimating risk. Having accepted the model, however, there remain considerable uncertainties concerning the estimate of the slope b most appropriate for an exposure situation; therefore, a range of slopes is often considered when assessing risk in an exposed population.

### *Mesothelioma Risk*

Mesothelioma, a primary pleural or peritoneal malignancy, is a rare disease, even among asbestos workers (a total of approximately 1,500 cases in the U.S. annually, compared with approximately 130,000 lung cancer cases). Consequently, in comparison with lung cancer, there is much less quantitative information for mesothelioma for establishing a dose-response relationship.

As a result of the very low incidence of this disease in the general population and conclusive evidence of an increased risk among asbestos workers (the only known occupational cause), any case occurring in a person with an industrial asbestos exposure is assumed to be a result of that exposure. However, virtually all studies of mesothelioma cases have included persons for whom no known asbestos exposure could be identified. Moreover, a substantial number of cases have been reported from rural Turkey (23) (attributed to the indigenous occurrence of another fibrous mineral, erionite, a form of the mineral zeolite (24)), and animal studies have demonstrated that fibers other than asbestos can induce mesothelioma in animals (25, 26). For these reasons, it is concluded that there are background cases of mesothelioma in the general population; i.e., cases with no relationship to asbestos exposure.

TABLE 1

MESOTHELIOMA IN POPULATIONS EXPOSED TO DIFFERENT FIBER TYPES

| Fiber Type | Total Deaths Observed | Lung Cancer | | Mesothelioma | |
|---|---|---|---|---|---|
| | | Observed Number | Excess* | Observed Number | As % of Excess Lung Cancer |
| Chrysotile (11, 15, 16, 27–30)† | 5,500 | 407 | 99.0 | 12 | 12.1 |
| Amosite (31, 32) | 861 | 144 | 85.5 | 19 | 22.2 |
| Crocidolite (33–35) | 735 | 80 | 31.5 | 52 | 165.1 |
| Mixed fiber (12, 14, 22, 29, 36–40) | 6,751 | 960 | 485.3 | 320 | 65.9 |

* Studies combined. For each study, excess lung cancers = observed number minus number expected based on comparison population.

† References in parentheses.

Of the several minerals commonly referred to as asbestos (chrysotile and the amphiboles) crocidolite and amosite, have accounted for almost all of the commercially used asbestos. There is strong qualitative evidence of a differential in mesothelioma risk by type of asbestos fiber exposure, with crocidolite, and probably amosite, posing greater risk than chrysotile. (This difference has been recognized by most industrialized nations by setting different control limits for these fibers.) Across-study comparisons of the observed numbers of mesotheliomas cannot be made directly because differences would be partially due to differences in the sizes and exposure levels of the cohorts studied. However, the number of excess lung cancers in a study should also be related to these factors, so this number can be used, in effect, as a measure of these variables for a study. Therefore, by considering the ratio of the number of mesotheliomas to the number of excess lung cancers, an approximate adjustment for exposure level and size differences of the study populations can be made. As shown in table 1, the number of mesotheliomas was 12% of the excess lung cancers among chrysotile-exposed populations, but 165% for crocidolite exposure. Populations exposed to a mixture of fibers exhibited intermediate results (66%). These data suggest that chrysotile exposure poses approximately one fifth the risk of a mixed fiber exposure (12 versus 66%). This pattern was observed for both pleural and peritoneal mesothelioma (see (5) for separate results), although the evidence is more striking for peritoneal tumors.

Similar results have been reported by two case-control studies (41, 42), which found cases to have had greater exposure to crocidolite than noncases, but no differences in chrysotile exposure. Tissue analysis studies (43, 44) have supported these findings, with greater numbers of crocidolite fibers but similar numbers of chrysotile fibers found in the lungs of mesothelioma cases compared with noncases. A recent study (45) of persons exposed to chrysotile asbestos in mining (contaminated with the amphibole tremolite) found, on average, a greater number of chrysotile fibers in the lungs of mesothelioma cases than in matched controls. These findings are consistent with the concept of dose-relatedness of risk with chrysotile exposure or its contaminant, which showed an even larger gradient. It cannot, however, address the question of a difference in risk between chrysotile and crocidolite/amosite exposures because neither crocidolite nor amosite was present.

Concerning the question of whether mesothelioma risk is related to industrial process, results of the three crocidolite exposure studies are suggestive of a difference by process: mesothelioma as a percentage of excess lung cancer risk was 120% among crocidolite miners (33) but 225 and 298% among crocidolite gas

mask manufacturing workers (34, 35). However, other study differences (e.g., length of follow-up) might account for these findings, and similar comparisons of populations exposed to other fiber types are difficult. With only twelve mesothelioma cases among the chrysotile studies (table 1), no meaningful comparison by process can be made for chrysotile; the two amosite populations were both involved in manufacturing; and within the mixed fiber studies, varying fiber type mixtures could account for observed differences in risk. It must therefore be concluded that the data currently available are not adequate to determine if there is a difference in mesothelioma risk by industrial process, after accounting for fiber type.

Because there is no reliable quantitative dose-response information by which mesothelioma risk may be related to asbestos exposure, mesothelioma modeling is based on the observation among asbestos workers that incidence rates increase as a power of time since initial exposure. Peto and associates (46), using data from the study of insulators (22), found that incidence was approximately proportional to the 3.2nd power of time since initial exposure, irrespective of age at initial exposure. Using this observation, together with a mathematical model of carcinogenesis (47) and the assumption of a linear relationship between incidence and exposure concentration, the following model is used for relating the mesothelioma incidence rate I(t) at time t since initial exposure to mean asbestos concentration c and duration d:

$$I(t) = \begin{cases} K\,c\,t^{3.2} & , 0 < t \leq d \\ K\,c(t^{3.2} - (t-d)^{3.2}) & , t > d \end{cases} \quad (\textit{Equation 3}).$$

Thus, during exposure ($0 < t \leq d$), incidence rises as a power of length of exposure; after exposure ceases ($t > d$), incidence rises less steeply.

Using this model and an estimate of the parameter K, ordinary lifetable methods are used to predict the total number of lifetime mesothelioma cases occurring in a cohort exposed to asbestos for d years at a concentration of c f/ml. A slightly different model incorporates a latency period for disease development by replacing elapsed time since first exposure (t) with elapsed time minus 10 yr ($t - 10$); however, there are only minimal differences between the two models in estimated lifelong risk.

As for mesothelioma, incidence rates of many cancers are power functions of time since initial exposure to a carcinogen; e.g., lung cancer and exposure to both smoking and asbestos. Because of this, the question may be raised of why the approaches to modeling lung cancer and mesothelioma risks differ substantially (modeling lung cancer relative risk but mesothelioma incidence). In estimating asbestos-associated lung cancer risk, age-specific background rates (to calculate an expected number E) are available so that relative risk of lung cancer may be considered. However, because of the small number of mesothelioma cases, background rates for mesothelioma are not available and, therefore, relative risk cannot be investigated.

There have been 7 studies (table 2) for which sufficient information is available to estimate the parameter K of the mesothelioma model (*Equation 3*). Most of these 7 studies do not meet minimal criteria concerning availability and validation of exposure information. For example, the study of insulators (22), with by far the largest number of mesothelioma cases, had neither job histories nor exposure measurements available. (See the Consumer Product Safety Commission (CPSC) report (2) for a more detailed review of these studies.) With these limitations in mind, and using the best available estimates of the mean duration and concentration of exposure for each cohort, estimates of K may be derived for each study (table 2).

Observed mesothelioma incidence rates in the 4 cohorts exposed to mixed fibers varied considerably; at 32 yr following initial exposure, rates among insulators (22) and British textile workers (48) were 15 to 20 times the rate in U.S. asbestos cement workers (50). However, according to the model, the differences in exposures (assuming concentrations of 30 versus 11 f/ml and durations 25 versus 4 yr) would result in incidence rates differing by a factor of 9 or more. Thus, if differences in exposures are taken into account, rates in these 3 populations are relatively similar. However, rates in the Canadian asbestos cement workers' study (49) were approximately triple those in the insulators' and textile workers' studies, even though the assumed exposures were lower. These differences in rates are reflected in the estimated values of the parameter K from these studies, with that based on the Canadian study (49) considerably higher than those from the other mixed fiber studies. Some of the differences in incidence rates in these 4 studies may be due to differences in the mixture of fibers, but little quantitative information on this point is available.

The observed and fitted mesothelioma incidence rates for 3 of these studies (figure 2) show that the model provides a reasonable fit to the observed data. (Data for the fourth mixed fiber study (50), omitted from figure 2 because the lower rates require a smaller scale, are also consistent with the model.)

For exposure to mixed asbestos fibers, the best available estimate for the parameter K of the model will be taken to be $0.2(10^{-8})$, approximately the median of the 4 estimates (table 2). To reflect the uncertainties of the value K, values ranging from 0.1 ($10^{-8}$) to 0.4 ($10^{-8}$) will also

TABLE 2

ESTIMATES OF THE MESOTHELIOMA MODEL PARAMETER K FROM 7 EPIDEMIOLOGIC STUDIES

| Industry | Mesothelioma Cases | Mean Duration (yr) | Mean Concentration (f/ml) | K x $10^8$ |
|---|---|---|---|---|
| Mixed Fibers | | | | |
| In (22) | 180 | 25* | 15* | 0.29 |
| | | 25* | 30* | 0.15 |
| Tx (48) | 7 | 25 | 30 | 0.11 |
| Ce (49) | 17 | 10* | 9* | 2.16 |
| Ce (50) | 8 | 4 | 11 | 05 |
| Amosite | | | | |
| Ma (31) | 14 | 1.5 | 35* | 1.1 |
| Chrysotile | | | | |
| Mn (51) | 1 | 31* | 40* | 0.006 |
| Tx (52)† | 1 | 10 | 7.5 | 0.07 |

*Definition of abbreviations:* Mn = mining, Fp = friction products, Ma = manufacturing, Gm = gas mask manufacturing, Ce = cement products, In = insulators, Tx = textile manufacturing, Sh = shipyard.

* Exposure information not available/reported for cohort; estimated from best available source.

† Detailed data presented in reference 53.

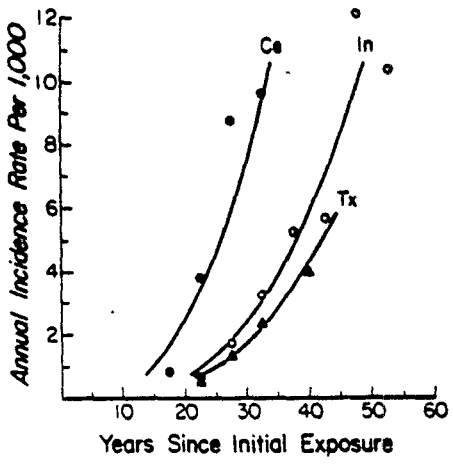


Fig. 2. Observed and fitted annual mesothelioma incidence rates by years since initial exposure in 3 populations with mixed asbestos fiber exposure (Ce = asbestos cement, In = insulation, Tx = textile).

be considered. Data for chrysotile exposures (table 2) suggest a value for K of $0.04(10^{-6})$, or one fifth the value for a mixed fiber exposure. This is consistent with the evidence in table 1 that chrysotile exposure may pose one fifth the risk of a mixed fiber exposure. A reasonable range for the chrysotile parameter will be taken as $0.02\ (10^{-6})$ to $0.08\ (10^{-6})$.

Because rates are assumed to increase with time since initial exposure and to be independent of age, the estimated number of lifetime cases among persons exposed early in life will be greater than for those exposed late in life. Thus, in estimating lifelong risk in an exposed population it is necessary to specify the age at initial exposure for the cohort.

*Uncertainties of the Mesothelioma Model*

The model for estimating the mesothelioma risk associated with asbestos exposure is based on many more assumptions and considerably less quantitative evidence than the model for lung cancer risk. Mesothelioma incidence as a power of time since initial exposure is consistent with other cancers, and the proposed model provides a reasonable fit to the available data sets. There is no quantitative evidence, however, that risk is linearly related to exposure concentration or that exposure duration should be factored into the model as in *Equation 3* (a result of mathematical modeling of multistage carcinogenicity). It should be noted that, in contrast to the model for lung cancer, the mesothelioma model does not assume linearity of risk with exposure duration; i.e., doubling the exposure duration does not necessarily double the risk. In fact, application of the model to a population initially exposed at 25 yr of age finds that doubling exposure duration from 10 to 20 years multiplies predicted risk by 1.4 (not by 2.0). However, for short durations, the model is approximately linear; e.g., doubling exposure from 1 to 2 yr results in approximately a doubling of estimated risk.

As with lung cancer, there is limited evidence that mesothelioma risk after many years following initial exposure rises less steeply than would be estimated by the model. If this evidence is valid, then the model could substantially overestimate lifetime mesothelioma risk.

*Industrial Asbestos Exposure*

While keeping in mind the stated uncertainties of the models, they are useful for obtaining an approximate estimate of the magnitude of asbestos-associated risk.

As an example, an industrial cohort of asbestos cement manufacturing workers who began employment at 25 yr of age will be considered. Of the studies represented in figure 1, the 2 studies with intermediate slopes (asbestos cement and mixed product manufacturing workers) were those concerned with the most similar industrial processes. The slopes for these studies were approximately 0.5, which will be taken as the value of the slope for lung cancer risk for this example. To reflect uncertainties in the estimated slope, slopes ranging from 0.25 to 1.0 will also be considered.

Based on current U.S. rates, the probability of a 25-yr-old man eventually dying of lung cancer is approximately 0.051. Thus, in a cohort of 10,000, approximately 510 lung cancer deaths would be expected (E = 510 in *Equation 2*). If the cohort is exposed to 0.5 f/ml (currently being considered by the U.S. Occupational Safety and Health Administration (OSHA) as the permissible exposure limit) for 20 yr, then cumulative exposure (x) is 10 f/ml – yr. Using *Equation 2* and a slope (b) of 0.5, the lung cancer model estimates a total of 25.5 (or 26) lung cancers attributable to the asbestos exposure (in addition to the 510 background cases). Doubling the exposure duration would double the lung cancer risk to 51 excess cases. The estimated number of lung cancer cases would be the same if the exposure is assumed to be to chrysotile only. See table 3.

For mesothelioma, the model (*Equation 3*) estimates a total of 27 lifetime mesotheliomas (using life table methods and 1980 U.S. mortality rates) if exposure to mixed asbestos fibers lasts for 20 yr; this estimate becomes 31 cases if the exposure continues for 40 yr (table 3). If these exposures are to chrysotile alone, then the estimated mesothelioma risk is reduced by a factor of 5.

Thus, the estimated total number of cancers attributed to 20 yr of exposure at 0.5 f/ml would be 53 cases for a mixed fiber exposure and 31 cases for a chrysotile exposure. Reducing the assumed asbestos exposure concentration level by a factor of 2 would similarly halve the estimated risk.

Such calculations may be performed for other industrial cohorts, with the lung cancer slope chosen appropriately. In the

TABLE 3

LIFETIME RISK ESTIMATES FOR AN INDUSTRIAL COHORT (MALES) OF ASBESTOS CEMENT MANUFACTURING WORKERS EXPOSED FROM 25 YR OF AGE TO AN AVERAGE ASBESTOS CONCENTRATION OF 0.5 F/ML, BY DURATION AND FIBER TYPE

| Duration (yr) | Estimated Attributable Cases per $10^4$ Exposed | | |
|---|---|---|---|
| | Lung Cancer* | Mesothelioma† | Total |
| Mixed Fiber Exposure | | | |
| 20 | 26 (13 to 51)‡ | 27 (13 to 54)§ | 53 (26 to 105) |
| 40 | 51 (26 to 102) | 31 (16 to 62) | 82 (42 to 164) |
| Chrysotile Exposure Only | | | |
| 20 | 26 (13 to 51) | 5 (3 to 11) | 31 (16 to 62) |
| 40 | 51 (26 to 102) | 6 (3 to 12) | 57 (29 to 114) |

* Based on an assumed slope of 0.5 (increase in the lung cancer Standardized Mortality Ratio per unit increase in cumulative asbestos exposure in f/ml – yr).

† Based on an assumed value of 0.2 ($10^{-6}$) for the model parameter K; see text.

‡ Range of estimates, based on a range of slopes 0.25 to 1.0.

§ Range of estimates, based on a range of parameters 0.1 ($10^{-6}$) to 0.4 ($10^{-6}$) for mixed fiber exposures and one fifth these values for chrysotile exposures.

TABLE 4

ESTIMATED NUMBER OF LIFETIME DEATHS ATTRIBUTABLE TO 6 YR OF ASBESTOS EXPOSURE IN SCHOOLS (PER $10^6$ EXPOSED), BY FIBER TYPE AND AVERAGE CONCENTRATION

| | Lung Cancer* | Mesothelioma† | Total |
|---|---|---|---|
| 0.001 f/ml* | | | |
| Mixed fibers | 0.6 | 4.4 | 5.0 |
| | (0.3 to 1.2)‡ | (2.2 to 8.8)§ | (2.5 to 10.0) |
| Chrysotile only | 0.6 | 0.9 | 1.5 |
| | (0.3 to 1.2) | (0.4 to 1.8) | (0.7 to 3.0) |
| 0.003 f/ml* | | | |
| Mixed fibers | 1.9 | 13.2 | 15.1 |
| | (0.9 to 3.7) | (6.6 to 26.4) | (7.5 to 30.1) |
| Chrysotile only | 1.9 | 2.6 | 4.5 |
| | (0.9 to 3.7) | (1.3 to 5.3) | (2.2 to 9.0) |

* Based on an assumed slope of 0.5; see footnote, table 3.
† Based on an assumed value of 0.2 ($10^{-6}$) for the model parameter K; see text.
‡ Range of estimates, based on a range of slopes 0.25 to 1.0.
§ Range of estimates, based on a range of model parameters; see footnote, table 3.

calculations, current lifelong risk of lung cancer was assumed to be valid for the cohort; in fact, there is evidence of a decrease in male lung cancer risk, both in the U.S. (54) and in Great Britain (55), primarily among those in the younger age groups (believed to be a result of the decrease in tobacco use). If this trend continues, then the number of background cases (E of *Equation 2*) should be lower, resulting in a correspondingly lower estimate of the excess cancers attributable to asbestos exposure.

*School Asbestos Exposure*

In its recently released report (6), the Ontario Royal Commission presented a complete review of the data (56–60) concerning asbestos exposure levels inside buildings containing friable asbestos. After reviewing and summarizing these data, the Commission concluded that "the majority of exposures of building occupants in buildings with substantial amounts of friable asbestos would be to fiber levels less than 0.001 f/ml, with a few single readings as high as 0.01 f/ml representing the highest likely exposure" (reference 6, p. 577).

Based on this report, the best estimate of the mean fiber concentration in U.S. schools containing asbestos products will be assumed to be 0.001 f/ml. To take into account the possibility that some students could be exposed to levels as high as 0.01 f/ml occasionally, an upper estimate of the possible mean exposure levels will be taken as 0.003 f/ml (the geometric mean of 0.001 and 0.01 f/ml). For each concentration level, it is assumed that students are exposed to this concentration of asbestos, on average, throughout every school day, for their entire length of enrollment in the school.

Students will be enrolled in schools containing asbestos products for a varying number of years, starting at various ages. On average, they will be assumed to be enrolled for 6 school years, at a mean initial age of 9 yr. Because a student spends approximately 36 wk per yr, 35 h per wk, at school, a school year is equivalent to 0.656 of a work year (48 wk per yr, 40 h per wk). Therefore, the exposure accumulated over a school year at 0.001 f/ml would be equivalent to that accumulated during a work year at 0.000656 f/ml.

Because the exposures of asbestos cement and mixed products manufacturing workers were in large part to dust of building products, these exposures may be assumed to be qualitatively similar to exposures inside buildings. Therefore, a slope of 0.5 will again be used as the best estimate of the slope in the lung cancer model (with a range of 0.25 to 1.0). This may be an overestimate of the slope because the dimensional distribution of the fibers emitted from finished products may be skewed more to shorter, coarser fibers than fibers used in manufacturing these products (including raw fibers).

Among a cohort of one million schoolchildren exposed to 0.001 f/ml of mixed fibers, the models estimate 0.6 lifetime lung cancers and 4.4 lifetime mesotheliomas (table 4), for a total of 5 lifetime *excess* deaths. If exposure is to chrysotile only (the fiber type used in acoustical plaster of ceilings), then the estimated mesothelioma cases would be 0.9 and the total cancers 1.5. If an asbestos concentration of 0.003 f/ml is assumed, then these estimates triple (table 4).

By contrast, in this population of one million students (half male, half female), approximately 32,000 (3.2%) would be expected to die of lung cancer based on recent U.S. rates; i.e., these would *not* be attributable to the school exposure to asbestos (more than 90% due to smoking).

For estimating background mesothelioma cases, Peto and associates (61), using data on Los Angeles residents, found that mesothelioma incidence rates for persons with no known asbestos exposure were approximately proportional to the 3.5 power of age. Checking the validity of this background model by applying it (with a coefficient of $2.3[10^{-12}]$) to the 1980 U.S. population, this model estimates a total of 385 mesothelioma cases during 1980 among persons without known asbestos exposure. This would be an annual rate of approximately 1.7 cases per million, which is in agreement with an estimate of 2.0 made recently by McDonald (62) using a different approach. Applying this model to a birth cohort of one milion, a total of 140 *lifetime* background cases are estimated. The estimated numbers of cases in the asbestos exposed students would be in addition to these background cases.

*Other Known Risks*

For school asbestos exposure, the upper estimate of approximately 15 lifetime excess deaths (mixed fibers at 0.003 f/ml) would constitute an average *annual* rate of approximately 0.25 deaths per million exposed. To place this risk in perspective, some other estimated and observed annual death rates per million in the U.S. are: approximately 1,200 for long-term smoking (63), 15 for bicycling (10 to 14 yr of age (64)), 15 for inhalation/ ingestion of foreign objects (65), 1 for living for 2 months with a cigarette smoker (66), and 10 from playing high school football (1970 to 1980) (67). Although not converted to rates, school bus accidents resulted in approximately 200 deaths in 1975 (68) and whooping cough vaccination, 5 to 20 deaths annually during 1970 to 1980 (69). By contrast, for school asbestos exposure, the upper estimate of an average annual rate of 0.25 deaths per million exposed would be equivalent to 0.75 annual deaths, based on EPA's estimate of 3 million students currently exposed (60).

*Other Estimates of Asbestos-Associated Risk*

Several advisory groups (primarily governmental) have derived quantitative estimates of asbestos-associated risk of lung cancer and mesothelioma under various exposure assumptions (concentration, duration, h/wk). In order to compare these estimates, they have been ad-

justed (where possible) to a cohort of 10,000 workers exposed starting at 20 yr of age, for 20 yr (for 40 h/wk) to 0.5 f/ml of mixed asbestos (table 5). (Where estimates were reported for smokers and nonsmokers separately, they have been combined in table 5 assuming half smokers.)

Because there was fundamental agreement among these reports in the general methodology, there is reasonably good agreement in the risk estimates. Nevertheless, important variability in the estimates remains. These differences are primarily the result of variability in the values assumed for the model parameters. Similarly, each reported range of risks reflects a corresponding range of parameter values. For a particular exposure situation of interest, the range for lung cancer risk can be narrowed considerably based on the appropriate industrial process.

The importance of the model parameters in estimating risk emphasizes the need for critical review of the epidemiologic studies used for estimating these parameters. This is especially true in modeling mesothelioma risk, because the studies with the highest estimated parameter values (31, 49) have potentially important limitations in their exposure estimates (table 2). In the study of Canadian asbestos cement workers (49), which yields the highest estimate for the model parameter K, workers in the highest exposure category were found to have the lowest lung cancer risk, bringing into question the validity of the exposure estimates. The study of amosite workers (31) had no exposure measurements performed at the work site. Because the value of the parameter K estimated from a study is directly related to the assumed exposure for that cohort, errors in exposure estimates would be correspondingly reflected as errors in K.

Differences in estimated background risk for lung cancer also account for some of the variability in the estimates of lung cancer risk in these reports. This is especially clear for the estimates from the CPSC and EPA reports in which the lung cancer risk estimates differ by a factor of 2, even though they used the same range of slopes for the model.

TABLE 5

ASBESTOS RISK ESTIMATES BASED ON VARIOUS REPORTS FOR A COHORT OF 10,000 MALES EXPOSED FOR 20 YR IN THE WORKPLACE TO 0.5 F/ML OF MIXED ASBESTOS FIBERS FROM 20 YR OF AGE

| Report | Lung Cancer | | Mesothelioma* |
|---|---|---|---|
| | Slope b | Excess Deaths | Deaths |
| CPSC (2) | 0.3 to 3.0 | 10 to 101 | 15 to 154 |
| EPA (4) | 0.3 to 3.0 | 21 to 212 | 16 to 159 |
| HSE† (7,8) | 0.04 to 5.3 | 2 to 250 | NA |
| NRC‡ (3) | 2.0 | 120 | 105 |
| OSHA‖ | 1.0 | 69 | 52 |
| This report | 0.05 to 2.0 | 3 to 102 | 38§ (19 to 76) |

*Definition of abbreviations:* CPSC = Consumer Product Safety Commission; EPA = Environmental Protection Agency; HSE = Health and Safety Executive; NRC = National Research Council.

* Because of minor differences in the mesothelioma models, the estimated parameters K of these models are not comparable and therefore are not reported here.

† Estimates based on tables 35 and 36 of 1979 report and table 7 of 1983 report.

‡ Mesothelioma estimate incorrectly calculated in report; recalculated here using NRCs estimate of K.

§ Best estimate and range of estimates; see footnote, table 3.

‖ Part of OSHA's 1983 rule-making procedure for a revised asbestos standard; derived by same contractor as EPA estimates.

## Discussion

Because of a general awareness that asbestos is a human carcinogen, the public has become increasingly concerned about the use of asbestos in our society and is often alarmed to learn of asbestos exposures in some work situations (e.g., brake lining repair) or of the presence of asbestos-containing products in public buildings, especially schools. Generally, however, little information has been available to workers, consumers, parents, or school administrators to assist them in estimating the likely magnitude of the potential risk from other occupational or nonoccupational exposures.

Some governmental agencies concerned with the issues of asbestos use have sought scientific input into asbestos-associated risk estimation as part of their decision-making process. While making valuable contributions, much of this work would likely have been improved by expanded use of the scientific review process. Moreover, these risk estimates have, for the most part, not reached the general public or the medical and health professionals who are often asked for advice in these issues.

Risk estimates such as appear in tables 3 and 4 should provide a basis for rational decision-making regarding asbestos, with input from all concerned parties. Such estimates may be used to identify exposure levels for which the associated risk is deemed "acceptable" by those exposed, and levels deemed unacceptable. In making these judgments, these risks may be placed in context by comparison with other risks in our society.

With respect to asbestos products in the schools, such a rational approach could be used to determine when removal is called for and when proper maintenance of these products is preferable to removal. In fact, some experts have argued that removal of building materials in good condition could pose greater risk than proper maintenance of these products because of the likelihood of increased exposure levels during and following removal.

The process of risk assessment has become increasingly accepted as a necessary base on which to base rational decision-making. However, just as it should not be denigrated as unhelpful because of its inevitable limitations, neither should it be oversold as a panacea. There is evidence that the public now has more limited (and realistic) expectations of science than it once did, while continuing to hold a favorable view of the value of scientific endeavor (70). This situation provides an opportunity for an improved dialogue between scientists and the public on subjects of common concern, and for scientists to play an important role in shaping public policy that is scientifically defensible.

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