

General Description

The term “asbestos” designates a group of naturally occurring fibrous serpentine or amphibole minerals that have extraordinary tensile strength, conduct heat poorly and are relatively resistant to chemical attack. The principal varieties of asbestos are chrysotile, a serpentine mineral, and crocidolite, amosite, anthophyllite, tremolite and actinolite, all of which are amphiboles.^a

Chrysotile fibres consist of aggregates of long, thin, flexible fibrils that resemble scrolls or cylinders of uniform chemical composition. Although chrysotile is a reasonably well defined mineral, the five amphibole asbestiform fibres have such variable chemical compositions and physical properties that positive identification is sometimes troublesome.

The macroscopic asbestos fibres are actually bundles of thinner fibres made up of fibrils which, in the case of chrysotile, have a diameter of 20-25 nm. Each macroscopic fibre is highly anisotropic and tends to decompose into its thinner constituents under industrial handling or from weathering, giving rise to a fibrous, partially respirable aerosol.

Sources

Natural sources are important, because asbestos minerals are widely spread throughout the earth’s crust and are not restricted to the few mineable deposits. In particular, chrysotile is present in most serpentine rock formations. Emissions are due to natural weathering and can be enhanced by man’s activities, such as quarrying or street building. Very little, however, is known about the amounts emitted from natural sources.

Man-made emissions originate from activities in the following categories:

- (a) mining and milling
- (b) manufacture of products
- (c) construction activities
- (d) transport and use of asbestos-containing products
- (e) disposal.

There has been a steep rise in the production and use of asbestos in the last 100 years. Asbestos consumption has levelled off in recent years (1) to about 4 million tonnes (1983) and, because of the relative decline in crocidolite and amosite usage (1) the figure represents essentially chrysotile production (in 1982 only about 5% of the total asbestos produced was in the form of amphibole asbestos (crocidolite and amosite) (2)).

In many industrialized countries most of the asbestos used was in the building sector (70-90% in several western European countries) (2), and the demand for asbestos in these

^a Man-made mineral fibres and other natural mineral fibres such as fibrous zeolites, wollastonite, attapulgite and sepiolite will not be discussed here.

countries had already reached a constant level before the health effects of asbestos were widely debated. Because of its specific technical properties, asbestos has found an extremely large variety of applications (in about 3000 different products). In the future, legislative restrictions and the success in finding substitutes for asbestos in fibre-cement, brake-linings, insulation and many other applications will most probably lead to declining asbestos consumption in the above-mentioned countries.

Asbestos emissions occur during processing. When air filtration is used, the dust emissions from processing can be kept below 100 g per tonne (3).

Rain acidity (from carbon dioxide and air pollutants) is known to corrode asbestos-cement sheets, constituting a further source of emission (4). Brake-linings in cars are another source in the urban environment. Only a few measurements have been made of the contribution made by these sources to fibre emissions in urban air (5,6).

Indoor asbestos fibre concentrations can be considerably higher than outdoor concentrations (7). Indoor asbestos dust originates from insulation material sprayed on steelwork or ceilings (such material may become highly friable after some years), asbestos plasters, low-weight insulation plates, etc. (8,9). Sometimes such materials have been used in direct or close contact with air-conditioning equipment. Even though some of these materials, such as spray asbestos, are no longer used, they are still found in many public buildings. Until the mid-1970s electric storage-heaters and some other electrical household equipment contained asbestos. One of the main forms of use of asbestos is as asbestos-cement; in this case the release of fibres to the general environment is minimized, since the fibres are essentially "locked" in the cement matrix. Asbestos cement products, therefore, do not usually pose problems for indoor air quality.

Factors such as renovation and repair, maintenance, external vibrations and vandalism can considerably increase the emission of asbestos dust from existing indoor sources (10,11). Increased emission is also possible as a result of changes in temperature and reduction of humidity.

Unlike levels of asbestos fibres in ambient air, fibre concentrations in the air of indoor environments can always be related to their source, thus offering the possibility of modifying or removing the source of emission. Because people in temperate climates live indoors most of the time, the most relevant source of inhalation exposure will often be asbestos fibre concentrations in buildings.

Occurrence in air

Asbestos fibres of respirable size form part of a range of fibrous aerosols in the lower atmosphere. Other fibres include man-made mineral fibres, fibrous silica and aluminium oxide, fibrous gypsum and, in some geographical areas, fibrous zeolite, attapulgite, sepiolite and wollastonite (1).

Once emitted into the atmosphere, asbestos fibres may travel considerable distances owing to their aerodynamic properties. Because no chemical breakdown of the fibres occurs, washout by rain or snow is the only cleaning mechanism.

Asbestos fibres normally constitute only a relatively small fraction of the total fibrous aerosol in ambient air (6,12). The biologically more important so-called "critical" fibres are those equal to or longer than 5 µm and having diameters up to 3 µm (1,10,12) with an aspect ratio equal to or greater than 3 : 1.

Although asbestos fibres can be readily detected and monitored in occupational situations by using phase-contrast optical microscopy, their assessment in the environment calls for an integrated method capable of microchemical analysis of single fibres, measurement of fibre length and diameter, and counts of fibre numbers in given air samples. Electron microscopy is the only method which can detect and identify asbestos fibres among the very wide range

of other fibrous and nonfibrous particles, of greatly varying toxic potency, in the ambient air. Used in conjunction with each other, these methods allow the identification of samples. In order positively to identify asbestos fibres in environmental ambient air it is necessary to use selected-area electron diffraction and/or energy-dispersive X-ray diffraction analysers attached to an electron microscope. Such instrumentation is costly and highly-trained personnel are required in order to obtain reliable results. Although integrated electron microscopy methods have been developed during the last 15 years (13), they are still not in widespread use. Therefore, all data measured by these methods are, strictly speaking, representative only for the location tested and the time interval chosen. However, if one compares various sets of such data and restricts quantification to orders of magnitude, the following concentration pattern emerges:^b

Rural areas (remote from asbestos emission sources):

- below 100 F/m³ (6,10)

Urban areas:

- general levels may vary from below 100 to 1000 F/m³ (6,12)

Near various emission sources the following figures have been measured as yearly averages (6,12,14):

- downwind from an asbestos-cement plant at 300 m: 2200 F/m³; at 700m: 800 F/m³; at 1000 m: 600 F/m³ (6);
- at a street crossing with heavy traffic, 900 F/m³ (14);
- on an express-way, up to 3300 F/m³ (9)

Indoor air:

- in buildings without specific asbestos sources, concentrations are generally below 1000 F/m³ (12);
- in buildings with friable asbestos, concentrations vary irregularly; usually less than 1000 F*/m³ are found, but in some cases exposure reaches 10 000 F*/m³ (9), where F* = fibres counted with an optical microscope.

Occupational levels are orders of magnitude higher than those found in the environment, with values from 10⁵ F*/m³ to more than 10⁸ F*/m³ (12,15), but are now being reduced to below 2 × 10⁶ F*/m³ in most countries and to (0.2-0.5) × 10⁶ F*/m³ in some.

^b Note. If not otherwise stated, concentrations in the following text are given as numbers of critical asbestos fibres per m³, i.e. all fibres of length L > 5 µm, diameter D < 3 µm and aspect ratio L : D > 3 : 1 measured by electron microscopy methods. All concentrations are expressed as fibres per m³ air, although concentrations in terms of fibres per litre and fibres per ml are often reported. Some studies have been carried out where results have been expressed as ng or µg per m³ air. Because only results such as fibre number concentrations are considered to be directly relevant to an index of exposure with regard to possible health implications (1,12) and factors in attempts to convert mass to number concentrations are so variable (12), mass concentrations are not considered here.

Routes of Exposure

Air

Various subgroups of the population are exposed to different fibre concentrations for varying lengths of time. It is usually assumed that the risks of any two persons are roughly comparable - other factors being equal - if their accumulated fibre burdens are the same. The fibre burden is the accumulated number of critical fibres (F) = fibre concentration (F/m^3) \times number of years of exposure \times air volume inhaled each year at place of exposure ($m^3/year$). In this context it is important to note that not all fibres inhaled are deposited and available for retention in the body. However, it is assumed that the fraction of fibres exhaled is more or less the same during all inhalation exposures. Assuming a breathing rate of $10 m^3$ for an 8-hour working day and 200 working days per year, a worker inhales $2000 m^3$ per year during working hours, while total inhalation for the general population is $7300 m^3$ per year (10).

Table 1 gives orders of magnitude of lifetime fibre burdens in various population subgroups, thus indicating the relative importance of different types of inhalation exposure (10,16). Values for indoor air exposure have not been included because only a few are available and details are insufficient. Only for the USA has a calculation of indoor concentration been made in terms of average fibre concentration. The median values range from $400 F/m^3$ (10) to $500 F/m^3$ (12). If these estimates were correct, they would result in a lifetime fibre burden of up to $2 \times 10^8 F$. In this case outdoor exposure would be of minor importance to most of the US population.

There can also be more irregular exposure of the general public, with peak concentrations at specific sites. Table 1 shows that such exposure for 1% of a lifetime (0.24 hours per day) could contribute significantly to the total accumulated burden of fibres. Peak exposures in people passing building sites have been reported by several authors. These exposures occurred when asbestos-cement sheets were being cut (16) or spray insulation was being carried out (17); in these cases no dust suppression measures were applied. Peak exposures may also occur in para-occupational situations (e.g. household activities (18)).

Table 1. Lifetime fibre burdens typical for industrial countries

Population ^a (%)	Subgroup	Fibre concentration (F/m^3) ^b	Exposure time (years)	Inhaled volume ($m^3/year$)	Accumulated critical fibres ($F > 5 \mu m$)
70	Urban population (moderate exposure)	30	70	7300	$\sim 1.5 \times 10^7$
25	Rural population	10	70	7300	$10^5 - 10^6$
5	Urban population (high exposure)	200	70	7300	$\sim 10^8$
1-2	General construction workers	$10^3 - 10^5$ ^c	50	2000	$10^8 - 10^{10}$ ^c
0.1	Asbestos workers	$10^5 - 10^6$ ^c	50	2000	$10^{10} - 10^{11}$ ^c
	Irregular exposure (example)	10^4	0.7	7300	5×10^7

^a These percentages reflect an assumed population distribution.

^b $F > 5 \mu m$.

^c Fibre count with optical microscope.

Drinking-water and food

Drinking-water and food may contain asbestos fibres from natural sources (e.g. rock) and man-made sources (e.g. asbestos-cement pipes). The total fibre content (fibres of all lengths) in drinking-water can vary from $10^4 F/litre$ to more than $10^8 F/litre$ (3,4,19,20).

Relative significance of different routes of exposure

Inhalation is by far the most important route of exposure of humans to critical fibres, the amount of uptake by ingestion being questionable and, at the least, significantly lower.

Kinetics

Health effects due to asbestos exposure in the occupational environment have been clearly associated with inhalation. The relevance of the oral intake of asbestos fibres for human health is unclear; in any case, ingestion is far less important than inhalation. For this reason, only the deposition, retention and clearance of fibres from the human lungs are described here.

Deposition

Fibres with a diameter greater than 3 µm are not respirable (10). Symmetrical fibres have a lower probability of deposition in the upper airways than nonsymmetrical fibres such as the “curly” chrysotile. A considerable number of fibres are deposited in the upper ciliated airways, where fibres can be removed by cleaning mechanisms (10). Chrysotile fibres can split into fibrils and undergo partial dissolution within the lungs. The amphiboles do not subdivide into fibrils of smaller diameter or break up by length. They are much less soluble in lung fluids, and they have long residence times in the lungs (10).

Clearance

Several mechanisms are involved in the clearing of fibres from their site of deposition, i.e. mucociliary clearance, translocation of alveolar macrophages containing small fibres, and uptake by epithelial cells lining the airways (17). Overall, the clearing mechanisms are very effective (95-98%), although in the alveolar regions some fibres can remain. It should be noted, however, that one of the most effective clearance mechanisms (mucociliary clearance) is impaired by smoking (1). Deposited fibres less than 5-10 µm long may often be engulfed by a single macrophage and thereafter be translocated; fibres longer than this are more difficult to clear. Long fibres may, however, become chemically dissolved or even mechanically broken down in the lung tissue. Durability seems to be greatest for amphiboles, less for chrysotiles (9,12,21,22); chrysotile can, in fact, split readily into fibrils and undergo partial dissolution in lung fluids (1).

The number of fibres retained in the lungs has been investigated very intensively in post-mortem lung material. All sorts of fibres known to exist in the atmospheric aerosol, including asbestos, have been found. The number of asbestos fibres per cm³ of lung sample is related to past exposure, the ratio of lung burdens for asbestos workers and controls being roughly the same as that of their respective exposures (10,12).

Health Effects

There is an intensive and well documented series of case studies and epidemiological observations which link past occupational exposure to asbestos with asbestosis, lung cancer and mesothelioma (1,3,9,10,12,23). Another series of studies has mainly examined subgroups of the population in particular exposure situations, mostly indoor exposure or para-occupational exposure (see above).

Asbestosis is a slowly developing fibrosis of the lung caused by the inhalation of high concentrations of asbestos dust and/or long exposure (24). Its severity depends both on the

length of time since onset of exposure and on the intensity of the latter (23). Although nonmalignant in itself, advanced asbestosis is often associated with lung cancer, especially among smokers. Hypoxia with cor pulmonale does occur in severe cases, while mild forms may not necessarily be associated with marked disablement (10).

Lung cancer (bronchial carcinoma) is the most frequent kind of cancer in the male population (accounting for about 10% of all male deaths in many industrialized countries) and is clearly related to external factors such as smoking, ionizing radiation (e.g. radon) and occupational exposures to certain substances, including asbestos inhalation, in the latter case even without co-existing asbestosis (9). Many studies have shown that smokers have a higher risk of developing lung cancer than nonsmokers when exposed to asbestos (23).

Mesothelioma is a malignant tumour of the pleura or peritoneum (9,23). It is a rare type of cancer (less than 0.04% of all deaths in the general population of the USA) (10). A higher incidence of mesothelioma has nearly always been related to the inhalation of mineral fibres, and in the majority of cases to occupational asbestos exposure (9). Causing few symptoms initially, mesothelioma is incurable when diagnosed (10).

Effects on experimental animals and *in vitro* test systems

Experimental toxicology has developed models to establish the relative carcinogenic potency of different types and sizes of fibres and to examine possible interactive effects with other airborne pollutants (3). This approach has proved helpful in the interpretation of epidemiological studies. Besides inhalation studies, ingestion and implantation studies have also been carried out; these have additionally been used to explore the association between asbestos and other forms of cancer in man, e.g. cancers of the gastrointestinal tract, which are epidemiologically less well established (9,23).

Species vary in their response to asbestos. Effects in rats seem to resemble those in man more closely than those in other animals (9); if not otherwise stated, the results reported refer to this species.

Inhalation

Lung cancer, mesothelioma and asbestosis have been observed in several studies regarding various asbestos and other fibres (10). In three inhalation experiments with 10 mg chrysotile per m³ over a period of 1 year, at least 20% of exposed rats developed lung tumours (25-27).

Intrapleural and intraperitoneal injection

The direct injection of a specific amount of dust with a given particle-size distribution into the pleura or the peritoneum makes it possible to study carcinogenic potency as a function of fibre type and shape without interference from variable factors such as deposition, clearance and disintegration, which are difficult to control in inhalation experiments.

The injection of asbestos produced mesotheliomas, the resulting incidence being highly fibre-specific and sensitive (3,9). Intraperitoneal injection of 1 mg chrysotile (UICC Canada) or 0.3 mg Actinolite resulted in a tumour rate of about 90% and 80%, respectively (28). Experiments show that fibre length and diameter are the most important factors in causing mesothelioma. As shown by Pott in his graph of the carcinogenicity factor (29), fibres shorter than 5 µm and thicker than 2 µm elicit little response, while those longer than 10 µm and thinner than 0.5 µm yield the highest response. Nonasbestos fibres of the same critical dimensions also caused mesothelioma in animals, while experiments with fibres of noncritical dimensions are negative, irrespective of fibre category (29). This finding is important considering the presence of many other fibre types in ambient air.

Ingestion

In several studies, rats and other laboratory animals were exposed to asbestos in diet or drinking-water. Only a single - usually very high - dose was applied in each investigation. The results are inconclusive, if not negative (1,20).

Effects on humans

Asbestosis

While early stages of asbestosis are still observed at asbestos-cement plants under modern regulations (24), asbestosis has never been observed in relation to nonoccupational asbestos exposure. It is therefore concluded that environmental concentrations of asbestos are not sufficient to induce asbestosis (1,9).

Mesothelioma

Without doubt, exposure to all kinds of asbestos is closely linked to mesothelioma. Several epidemiological studies indicate that amphibole asbestos is more potent in inducing mesothelioma than chrysotile (1), which seems to contradict the findings from intraperitoneal injection experiments (9). The contradiction is, however, to a great extent resolved by the observation that technical processes involving amphiboles produce dust with a higher proportion of long, thin - i.e. more dangerous - fibres (9). Furthermore, amphibole fibres are more resistant in the body and have a lower lung clearance than chrysotile (9).

The time elapsing between first exposure to asbestos and the clinical manifestation of tumours ranges from 20 to 50 years for mesothelioma in the populations of workers studied (23). Dose-response relationships are derived from retrospective epidemiological studies of exposure data relating to situations several decades ago. Clearly these exposure data can only be approximations (9,15). Increased incidence rates seen in nonoccupationally exposed people living in the same household as asbestos workers or in the vicinity of strong asbestos emission sources have been attributed to this exposure (30).

Lung cancer

Clarifying the relationship between asbestos exposure and lung cancer is a much more complicated task. Both the synergism of smoking and asbestos inhalation and the high level of lung cancer in the general population (background exposure) must be taken into account. The synergistic effect can be described by a multiplicative model (31) which is used below in connection with the extrapolation of lung cancer risk. The risk of lung cancer seems to rise consistently from mining and milling operations, through branches such as asbestos-cement production, to asbestos textile and insulation work, according to the increasing portion of more dangerously shaped fibres in these processes (see Table 2).

Other cancers

Recent evaluations (9,10,20,23) of epidemiological occupational studies provide little evidence for the induction of other cancers, including gastrointestinal cancers, although recently it has been suggested that laryngeal cancer may be related to heavy occupational exposure to asbestos (23). Clearly the risk, if any, for the general population from "other" cancers is very small (9,20,45).

Table 2. Increase in the relative risk of lung cancer, as shown by different studies

K _L per 100 F*year/ml	Type of activity	Reference
0.04	mining and milling	(33)
0.045	mining and milling	(34)
0.06	friction material	(35)
0.1	factory processes	(36,37)
(M) 0.4-1.1	factory processes	(38) ^b
(F) 2.7 ^a	factory processes	
0.2	asbestos-cement	(39)
0.07	textiles (before 1951)	(40)
0.8 ^a	textiles (after 1950)	
6(M) 1.6 ^a	textiles	(41)
1.6	textiles	(42) ^c
1.1	insulation products	(43) ^b
1.5	insulation	(44) ^b

^a Fewer than 10 cases of lung cancer expected (i.e. small cohort).

^b Inadequate knowledge of actual fibre concentrations.

^c Same factory as in (41), but larger cohort.

Source: based on a table by Liddell (32).

Evaluation of Human Health Risks

Exposure

Actual indoor and outdoor concentrations in air range from below one hundred to several thousand fibres per m³.

Health risk evaluation

On the basis of the evidence from both experimental and epidemiological studies, it is clear that asbestos inhalation can cause asbestosis, lung cancer and mesothelioma. The evidence that ingested asbestos causes gastrointestinal or other cancers is insufficient. Furthermore, the carcinogenic properties of asbestos are most probably due to its fibre geometry and remarkable integrity; other fibres with the same characteristics may also be carcinogenic.

Current environmental concentrations of asbestos are not considered a hazard with respect to asbestosis. However, a risk of mesothelioma and lung cancer from the current concentrations cannot be excluded.

A WHO Task Group recently expressed reservations about the reliability of risk assessment models applied to asbestos risk. Its members suggested that such models can only be used to obtain a broad approximation of the lung cancer risk of environmental exposures to asbestos and "that any number generated will carry a variation over many orders of magnitude". The same was found to be true for estimates of the risk of mesothelioma. The same document stated: "In the general population the risks of mesothelioma and lung cancer attributable to asbestos cannot be quantified reliably and probably are undetectably low." (1).

The following estimates of risk are based on the relatively large amount of evidence from epidemiological studies concerning occupational exposure. Data from these studies have been

conservatively extrapolated to the much lower concentrations found in the general environment. Although there is evidence that chrysotile is less potent than amphiboles, as a precaution chrysotile has been attributed the same risk in these estimates.

Mesothelioma

A formula by which the excess incidence of mesothelioma can be approximated has been derived by Peto (46). Fibre concentration, duration of exposure and time since first exposure are parameters incorporated in this model, which assumes a linear dose-response relationship. Peto verified this model from data on an urban population exposed for its whole life and on workers exposed for many decades. In both cases, duration of exposure is assumed to be equal or close to time since first exposure. The data show that the incidence of mesothelioma is proportional to the fibre concentration to which the workers were exposed and to time^{3.5} since first exposure for both workers and the general population. Starting from this relationship, one may calculate the risk of lifetime exposure to environmental concentrations from the incidence of mesothelioma in occupational populations exposed to much higher concentrations, but for a shorter time.

Apart from incomplete knowledge about the true workplace exposure, a further complication arises from the fact that workplace concentrations were measured by means of an optical microscope, counting only fibres longer than 5 µm and thicker than, say, 0.5 µm.^c

Several studies have been performed to calculate the risk of mesothelioma resulting from nonoccupational exposure to asbestos. Lifetime exposure to 100 F*/m³ has been estimated by various authors to carry differing degrees of mesothelioma risk (see Table 3).

Table 3. Estimates of mesothelioma risk resulting from lifetime exposure to asbestos

Risk of mesothelioma from 100 F*/m ³	Values in original publication (risk for fibre concentration indicated)	Reference
1.0×10^{-5}	1.0×10^{-4} for 1000 F*/m ³	(45)
$\sim 2.0 \times 10^{-5}$	1.0×10^{-4} for (130-800) F*/m ³	(47)
$\sim 3.9 \times 10^{-5}$	1.56×10^{-4} for 400 F*/m ³	(10,48)
$\sim 2.4 \times 10^{-5}$	2.75×10^{-3} (females) 1.92×10^{-3} (males) } for 0.01 F/ml	(17)

The risk estimates in Table 3 differ by a factor of 4. A “best” estimate may be 2×10^{-5} for 100 F*/m³.

An independent check of this risk estimate can be made by calculating the incidence of mesothelioma in the general population, based on a hypothetical average asbestos exposure 30-40 years ago (49). If the latter had been 200-500 F*/m³ (corresponding to about 400-1000 F/m³ as measured today), the resulting lifetime risk of mesothelioma would be $(4-10) \times 10^{-5}$. With the average United States death rate of 9000×10^{-6} per year, this would give 0.4-0.9 mesothelioma cases each year per million persons from past environmental asbestos exposure. The reported mesothelioma incidence in the USA ranges from 1.4×10^{-6} per year

^c In this chapter all fibre concentrations based on optical microscopy are marked F*/m³ and risk estimates will be based on F*/m³. If concentrations measured by optical microscope are to be compared with environmental fibre concentrations measured by a scanning electron microscope, a conversion factor has to be used: $2 \text{ F}/\text{m}^3 = 1 \text{ F}^*/\text{m}^3$.

to 2.5×10^{-6} per year according to various authors (10, 49). Thus, the calculated risk figures would account for only part of the observed incidence. However, other factors which may account for this discrepancy must be considered.

- Uncertainties in the risk extrapolations result from the lack of reliable exposure data in the cohort studies, errors in the medical reports, and necessary simplifications in the extrapolation model itself (17). Furthermore, the amount of past ambient exposure can only be an educated guess.
- The incidence of nonoccupational mesotheliomas is calculated from the difference between the total of observed cases and the number of those probably related to occupational exposure. Neither of these two figures is exactly known. Moreover, the influence of other environmental factors in the generation of mesothelioma is unknown.

In the light of these uncertainties, the result obtained by using the risk estimate can be considered to be in relatively good agreement with the annual mesothelioma death rate based on national statistical data.

Lung cancer

Unlike mesothelioma, lung cancer is one of the most common forms of cancer. As several exogenous noxious agents can be etiologically responsible for bronchial carcinoma, the extrapolation of risk and comparison between different studies is considerably complicated. In many epidemiological studies, in particular, the crucial effect of smoking has not been properly taken into account. Differentiation of the observed risks according to smoking habits has been carried out, however, in the cohort of North American insulation workers studied by Hammond et al. (31).

This study suggests that the relative risk at a given time is approximately proportional to the cumulative amount of fine asbestos dust received up to this point, for both smokers and nonsmokers. The risks for non-asbestos-exposed nonsmokers and smokers must therefore be multiplied by a factor which increases in proportion to the cumulative exposure.

The dose-response relationship in the case of asbestos-induced lung cancer can be described by the following equation (17).

$$I_L(\text{age, smoking, fibre dose}) = I_L^\circ(\text{age, smoking}) [1 + K_L \times C_f \times d]$$

This equation could also be written as:

$$K_L = [(I_L / I_L^\circ) - 1] / C_f \times d = (\text{relative risk} - 1) / (\text{cumulative exposure})$$

where:

K_L = a proportionality constant, which is a measure of the carcinogenic potency of asbestos

C_f = fibre concentration

d = duration of exposure, in years

I_L = lung cancer incidence, observed or projected, in a population exposed to asbestos concentration C_f during time d

I_L° = lung cancer incidence expected in a group without asbestos exposure but with the same age and smoking habits (this factor includes age dependence).

There are several studies which allow the calculation of K_L . Liddell (32, 50) has done this in an interesting and consistent manner. The results are given in Table 2.

Taking the data in Table 2 as a basis, a reasonable estimate for K_L is 1.0 per 100 F*years/ml. For a given asbestos exposure, the risk for smokers is about 10 times that for nonsmokers (31). In extrapolating from workers to the general public, a factor of 4 for correction of exposure time has to be applied to K_L .

The incidence of lung cancer in the general population exposed to 100 F*/m³ is calculated as follows:

$$I_L = I_L^\circ (1 + 4 \times 0.01 \times 10^{-4} \text{ F*/ml} \times 50 \text{ years})$$

or

$$I_L = I_L^\circ (1 + 2 \times 10^{-4} \text{ F*/ml})$$

The extra risk is $I_L - I_L^\circ$. Values for I_L° are about 0.1 for male workers and 0.01 for male nonsmokers (10).

Lifetime exposure to 100 F*/m³ (lifetime assumed to be 50 years since, in a lifetime of 70 years, the first 20 years without smoking probably do not make a large contribution) is therefore estimated as follows.

Status	Risk of lung cancer per 100 000	Range (using the highest and lowest values of K_L from Table 2)
Smokers	2.0	0.08-3.2
Nonsmokers	0.2	0.008-0.32

This risk estimate can be compared, when adjusted to 100 F*/m³, with estimates for male smokers made by other authors or groups:

Breslow (National Research Council) (48): 7.3×10^{-5} ;

Schneiderman et al. (47): $(14-1.4) \times 10^{-5}$;

US Environmental Protection Agency (17): 2.3×10^{-5} .

A fibre concentration of 100 F*/m³ (about 200 F/m³ as seen by scanning electron microscope) thus gives a total risk of $(2 + 2) \times 10^{-5}$ for smokers or 2.2×10^{-5} for nonsmokers.

Guidelines

Asbestos is a proven human carcinogen (IARC Group 1). No safe level can be proposed for asbestos because a threshold is not known to exist. Exposure therefore should be kept as low as possible.

Several authors and working groups have produced estimates indicating that, with a lifetime exposure to 1000 F/m³ (0.0005 F*/ml or 500 F*/m³, optically measured) in a population of whom 30% are smokers, the excess risk due to lung cancer would be in the order of 10⁻⁶ - 10⁻⁵. For the same lifetime exposure, the mesothelioma risk for the general population would be in the range of 10⁻⁵ - 10⁻⁴. These ranges are proposed with a view to providing adequate health protection, but their validity is difficult to judge. An attempt to calculate a “best” estimate for the lung cancer and mesothelioma risk is described above.

References

1. *Asbestos and other natural mineral fibres*. Geneva, World Health Organization, 1986 (Environmental Health Criteria, No. 53).
2. *Asbestos*. Paris, OECD, 1984, p. 21 (Env/Air 81.18; 2nd Rev.).
3. **Umweltbundesamt** [Federal Office for the Environment]. *Umweltbelastung durch Asbest und andere faserige Feinstäube* [Environmental pollution by asbestos and other fibrous fine dusts]. Berlin (West), Erich Schmidt Verlag, 1980 (Berichte 7/80).
4. **Meyer, E.** Vorkommen von Asbestfasern im Trinkwasser [Occurrence of asbestos fibres in drinking-water]. In: Fischer, M. & Meyer, E., ed. *Zur Beurteilung der Krebsgefahr durch Asbest* [Assessment of the cancer risk of asbestos]. Munich, Medizin Verlag, 1984, pp. 62-78.
5. **Fischer, M. & Meyer, E.** The assessment of the health risk from asbestos fibres by the Federal Health Office of the Federal Republic of Germany. *VDI-Berichte*, 475: 325-330 (1983).
6. **Marfels, H. et al.** Immissionsmessungen von faserigen Stäuben in der Bundesrepublik Deutschland - I. Messungen in der Nähe einer Industriequelle [Measurements of fibrous dusts in ambient air of the Federal Republic of Germany. I. Measurements in the vicinity of an industrial source]. *Staub, Reinhaltung der Luft*, 44: 259-263 (1984).
7. **Lohrer, W.** Asbestbelastete Innenräume - Analyse und Bewertung des Gefahrenpotentials [Asbestos exposed interiors - analysis and evaluation of risk potentials]. *Staub, Reinhaltung der Luft*, 43: 434-438 (1983).
8. **Nicholson, W.J., et al.** *Asbestos contamination of the air in public buildings*. Research Triangle Park, NC, US Environmental Protection Agency, 1975 (Final report, contract 68-02-1346).
9. *Report of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario*. Toronto, Ontario Ministry of the Attorney General, 1984, Vol.1-3.
10. **National Research Council.** *Asbestiform fibers: nonoccupational health risks*. Washington, DC, National Academy Press, 1984.
11. **Sawyer, R.N.** Indoor air pollution: application of hazard criteria. *Annals of the New York Academy of Sciences*, 330: 579-586 (1979).
12. **Commins, B.T.** *The significance of asbestos and other mineral fibres in environmental ambient air*. Maidenhead, Commins Ass., 1985.
13. **Chatfield, E.J.** Measurement and interpretation of asbestos fibre concentrations in ambient air. In: *Proceedings of the 5th Colloquium on Dust Measuring Technique and Strategy, October 1984*. Johannesburg, South African Asbestos Producers Advisory Committee, 1985, pp. 269-296.
14. **Marfels, H. et al.** Immissionsmessungen von faserigen Stäuben in der Bundesrepublik Deutschland - II. Messungen an der Kreuzung einer Großstadt [Measurements of fibrous dusts in ambient air of the Federal Republic of Germany. II. Measurements on a busy crossing of a large town]. *Staub, Reinhaltung der Luft*, 44: 410-414 (1984).
15. **Nicholson, W.J.** Asbestos and inorganic fibres. *Arbete och hälsa*, 17 (1981).

16. **Woitowitz, K.J. & Rodelsperger, K.** Asbestemissionen im Grenzbereich von Arbeitsplatz und Umwelt [Asbestos emissions in the borderline area of workplace and environment]. In: Fischer, M. & Meyer, E., ed. *Zur Beurteilung der Krebsgefahr durch Asbest* [Assessment of the cancer risk of asbestos]. Munich, Medizin Verlag, 1984.
17. *Airborne asbestos health assessment update*. Research Triangle Park, NC, US Environmental Protection Agency, 1985 (EPA-600/8-84-003F).
18. **Lilis, R. & Selikoff, I.J.** Asbestos disease in occupationally exposed workers and household contacts. In: Fischer, M. & Meyer, E., ed. *Zur Beurteilung der Krebsgefahr durch Asbest* [Assessment of the cancer risk of asbestos]. Munich, Medizin Verlag, 1984.
19. **Commins, B.T.** *Asbestos fibres in drinking water*. Maidenhead, Commins Ass., 1983.
20. **Toft, P. et al.** Asbestos in drinking water. *Critical reviews in environmental control*, 14(2): 151-197 (1984).
21. **Friedberg, K.D. & Ulmer, S.** Deposition und Elimination von Staub aus faserförmigem Material in der Lunge von Versuchstieren [Deposition and elimination of fibrous material dust in the lungs of experimental animals]. *VDI Berichte*, 475: 269-274 (1983).
22. **Lippmann, M.** Peer review: inhalation and elimination of MMMF – aids to the understanding of the effects of MMMF. In: *Biological effects of man-made mineral fibres*. Copenhagen, WHO Regional Office for Europe, 1984, Vol. 2, pp. 355-366.
23. **Doll, R. & Peto, J.** *Asbestos: effects on health of exposure to asbestos*. London, H.M. Stationery Office, 1985.
24. **Weicksel, P. & Ulrich, E.** Asbeststaubgefährdung in der modernen asbestverarbeitenden Industrie [The hazard of asbestos dust in the modern asbestos-processing industry]. In: Fischer, M. & Meyer, E., ed. *Zur Beurteilung der Krebsgefahr durch asbest* [Assessment of the cancer risk of asbestos]. Munich, Medizin Verlag, 1984.
25. **Davis, J.M.G. et al.** Mass and number of fibres in the pathogenesis of asbestos-related lung disease in rats. *British journal of cancer*, 37: 673-688 (1978).
26. **Wagner, J.C. et al.** Animal experiments with MMM(V)F fibres - effects of inhalation and intrapleural inoculation in rats. In: *Biological effects of man-made mineral fibres*. Copenhagen, WHO Regional Office for Europe, 1984, Vol. 2, pp. 209-233.
27. **McCohnell, E.E. et al.** A comparable study of the fibrogenic and carcinogenic effects of UICC Canadian chrysotile B asbestos and microfibrils (JM 100). In: *Biological effects of man-made mineral fibres*. Copenhagen, WHO Regional Office for Europe, 1984, Vol. 2, pp. 234-250.
28. **Pott, F. et al.** Carcinogenicity studies on fibres, metal compounds, and some other dusts in rats. *Experimental pathology*, 32: 129-152 (1987).
29. **Pott, F.** Some aspects on the dosimetry of the carcinogen potency of asbestos and other fibrous dusts. *Staub, Reinhaltung der Luft*, 38: 486- 490 (1978).
30. **Ham, E.** Untersuchungen über gesundheitliche Asbestschäden in Hamburg (1969-1979) [Studies of health damage caused by asbestos in Hamburg (1969-1979)]. In: Fischer, M. & Meyer, E., ed. *Zur Beurteilung der Krebsgefahr durch Asbest* [Assessment of the cancer risk of asbestos]. Munich, Medizin Verlag, 1984.
31. **Hammond, E.C. et al.** Asbestos exposure, cigarette smoking and death rates. *Annals of the New York Academy of Sciences*, 330: 473-490 (1979).
32. **Liddell, F.D.K.** Some new and revised risk extrapolations from epidemiological studies on asbestos workers. In: Fischer, M. & Meyer, E., ed. *Zur Beurteilung der Krebsgefahr durch Asbest* [Assessment of the cancer risk of asbestos]. Munich, Medizin Verlag, 1984.
33. **McDonald, J.C. et al.** Dust exposure and mortality in chrysotile mining, 1910-1975. *British journal of industrial medicine*, 37: 11-24 (1980).
34. **Nicholson, W.J. et al.** Long-term mortality experience of chrysotile miners and millers in Thetford Mines, Quebec. *Annals of the New York Academy of Sciences*, 330: 11-21

- (1979).
35. **Berry, G. & Newhouse, M.L.** Mortality of workers manufacturing friction materials using asbestos. *British journal of industrial medicine*, 40: 1-7 (1983).
 36. **Henderson, V.L. & Enterline, P.E.** Asbestos exposure: factors associated with excess cancer and respiratory disease mortality. *Annals of the New York Academy of Sciences*, 330: 117-126 (1979).
 37. **Enterline, P. et al.** Mortality in relation to occupational exposure in the asbestos industry. *Journal of occupational medicine*, 14: 897-903 (1972).
 38. **Newhouse, M.L. & Berry, G.** Patterns of mortality in asbestos factory workers in London. *Annals of the New York Academy of Sciences*, 330: 53-60 (1979).
 39. **Weill, H. et al.** Influence of dose and fiber type on respiratory malignancy risk in asbestos cement manufacturing. *American review of respiratory diseases*, 120: 345-354 (1979).
 40. **Peto, J.** Lung cancer mortality in relation to measured dust levels in an asbestos textile factory. In: Wagner, J.C., ed. *Biological effects of mineral fibres*. Lyon, International Agency for Research on Cancer, 1980 (IARC Scientific Publications, No. 30).
 41. **Dement, J.M. et al.** Estimates of dose—response for respiratory cancer among chrysotile asbestos textile workers. *Annals of occupational hygiene*, 26: 869-887 (1982).
 42. **Fry, J.S. et al.** Respiratory cancer in chrysotile production and textile manufacture. *Scandinavian journal of work, environment & health*, 9: 68-70(1983).
 43. **Seidman, H. et al.** Short-term asbestos work exposure and long-term observation. *Annals of the New York Academy of Sciences*, 330: 61-67 (1979).
 44. **Selikoff, J.J. et al.** Mortality experience of insulation workers in the United States and Canada, 1943-1976. *Annals of the New York Academy of Sciences*, 330: 91-116 (1979).
 45. **Aurand, K. & Kierski, W.-S., ed.** *Gesundheitliche Risiken von Asbest*. Eine Stellungnahme des Bundesgesundheitsamtes Berlin [Health risks of asbestos. A position paper of the Federal Health Office, Berlin]. Berlin (West), Dietrich Reimer Verlag, 1981 (BgA-Berichte, No. 4/81).
 46. **Peto, J.** Dose and time relationships for lung cancer and mesothelioma in relation to smoking and asbestos exposure. In: Fischer, M. & Meyer, E., ed. *Zur Beurteilung der Krebsgefahr durch Asbest* [Assessment of the cancer risk of asbestos]. Munich, Medizin Verlag, 1984.
 47. **Schneiderman, M.S. et al.** *Assessment of risks posed by exposure to low levels of asbestos in the general environment*. Berlin (West), Dietrich Reimer Verlag, 1981 (BgA-Bericht, No. 4/81).
 48. **Breslow, L. et al.** Letter. *Science*, 234: 923 (1986).
 49. **Enterline, P.E.** Cancer produced by nonoccupational asbestos exposure in the United States. *Journal of the Air Pollution Control Association*, 33: 318-322 (1983).
 50. **Liddell, F.D.K. & Hanley, J.A.** Relations between asbestos exposure and lung cancer SMRs in occupational cohort studies. *British journal of industrial medicine*, 42: 389-396 (1985).