

The rapid development of the drywall construction trade in the United States is described. It is estimated that some 75,000 U.S. construction workers are currently employed in the trade. The use of a variety of spackle and taping compounds is shown to be associated with significant asbestos exposure; air samples taken in the breathing zone of drywall tapers during sanding of taping compounds show fiber concentrations exceeding, several times, the maximum level permitted by United States Government regulations. These findings are given together with the result of a clinical field survey of drywall construction workers demonstrating that asbestos disease may be an important health hazard in this trade.

Drywall construction and asbestos exposure

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introduction

The introduction of new industrial processes widens the range of possible occupational health hazards. During the past two decades, drywall construction has achieved an important place in the United States construction industry. In such construction, taping of joints is required, i.e. applying and finishing spackle material at wallboard joints. Spackling and drywall taping compounds consist of extremely fine-grained white powders or pre-mixed pastes. Analyses of samples of spackling, patching and jointing compounds have shown that plaster of Paris is frequently a major constituent but that quartz, talc, micas, clays and ground limestone may occur in many formulations. More significantly perhaps, it has been shown that some compounds contain asbestos minerals, added as reinforcing agents. Either chrysotile or amphibole asbestos, or both, have been found in 13 out of 15 industrial products; analysis of spackle compounds, which are used in home repair work, has demonstrated that they also frequently contain from 5 to 12% by weight of asbestos minerals, as well as quartz.⁽¹⁾

history and terminology

The history of the drywall industry in the United States began at the end of World War II.

Returning servicemen and their families needed living places rapidly and inexpensively. Drywall construction would very soon replace lathing and plastering. While the plasterer had to perform his work alone, the drywall tapers were able to synchronize their work with that of carpenters, electricians, plumbers and other building trades, greatly reducing construction time. Low cost housing went into mass production in 1947-1948; wallboard sections were soon manufactured to fit standard room dimensions, enabling a worker to construct living quarters within a few hours. Drywall construction was also considered superior because of its adaptation to soundproofing and fire codes. The tapers were to be called "drywall workers" as opposed to plasterers or "wet wall workers." Initially part of the International Brotherhood of Painters and Allied Trades local unions, workers engaged in this new trade tended to be separately defined in the 1960's and local union groups of drywall tapers and painters were constituted with currently a total of some 3,500 members. Furthermore, it is estimated that some 75,000 other construction workers were currently employed in this trade in the United States, but are not in defined local unions of tapers. (In addition, carpenters, electricians, and plumbers may work in the vicinity of ongoing drywall construction.)

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United States is described currently employed in this is shown to be associated breathing zone of drywall concentrations exceeding Government regulations. A field survey of drywall may be an important health

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men and their families need a drywall joint compound that is cheap and inexpensive. Drywall joint compound is soon to replace lath and plaster. When the plasterer had to work alone, the drywall tapers were able to do their work with that of the plasterer, plumbers and other tradesmen, greatly reducing construction costs. Housing went into mass production in 1947-1948; wallboard sections were manufactured to fit standard room dimensions, allowing a worker to construct a room within a few hours. Drywall joint compound is also considered superior to plaster for its application to soundproofing and fireproofing. Drywall tapers were to be called "drywall tapers" or "wet wall tapers" by part of the International Union of Painters and Allied Trades local union. They were engaged in this new trade, which was later defined in the 1960's and 1970's by the ups of drywall tapers and the decline of plasterers and substituted with currently a total of 75,000 other construction workers. Furthermore, it is estimated that 75,000 other construction workers are currently employed in this trade in the United States, but are not in defined local unions. (In addition, carpenters, electricians, plumbers may work in the drywall construction.)

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Figure 1 — Pole sanding of drywall taping compounds. Several applications of taping compounds may be required to make a smooth joint between gypsum wall boards, requiring extensive sanding.

work procedures

Work performed by tapers may be associated with significant asbestos exposure. Tapers spend approximately 5-10% of their working time sanding dried spackle and thus may experience intermittent asbestos exposure. Significant exposure may also occur among painters who occasionally apply or sand spackle and taping compounds.

mixing

Prior to applying the spackle material at the wall board joints, the compound is prepared in the following way: Half of a 25 lb. bag of drywall taping compound is poured into a 5 gallon bucket containing about 2 gallons of water. The mixture is stirred until it has obtained a desired degree of consistency, whereupon the remainder of the dry mix is added. The mixture is again stirred. This operation takes approximately 60-90 seconds. Until pre-mix material came into use

in approximately 1975, the mixing was done several times a day.

sanding

After the taping compound has been applied at the wallboard joint and has dried, the surface is sanded. Two different sanding techniques have been used:

pole sanding

This is the most common method at present. The apparatus used consists of a 5-foot pole with a steel plate to which a piece of sandpaper is attached (Figure 1).

hand sanding

This technique is preferred to pole sanding by some tapers, since it allows better visual and manual control. Hand sanding is also required in those situations in which the space relationships prevent the taper from using pole sanding.

analysis of materials

Analysis of taping compounds and spackle materials indicate that a number of biologically active minerals are to be found. The compounds that have been analyzed by x-ray powder diffraction, optical and electron microscopy include both dry and paste types. Of 15 industrial drywall taping and spackling compounds, 13 contained asbestos (Table I). In addition, three compounds contained two different types of asbestos, namely chrysotile and tremolite. Quartz, and other silicates were also present in some other compounds. The results of these analyses showed that most of the taping compounds used in the United States construction industry contained asbestos in the order of 4-5%. One compound contained 10-15% chrysotile and 8-12% tremolite.

personal air sampling during drywall taping

A series of dust counts showed that asbestos fiber concentration may be considerable. Table II shows fiber concentrations experienced during pole and hand sanding of drywall taping compounds. Air samples taken in the breathing zone of the drywall tapers during sanding of wall board taping showed that fiber concentrations often exceeded, by several times, the maximum level permitted by United States government

TABLE I
Mineral Content of Fifteen Industrial Drywall Taping and Spackling Compounds

Mineral Phase	Sample Number														
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Chrysotile	6-8%	5-10%		8-8%	5-10%	5-10%	5-10%	5-10%	5-10%	5-10%		5-8%	10-15%		8-12%
Tremolite	4-7%											4-7%	8-12%	2-3%	
Talc		++	+									++	++	++	+
Quartz	+	++	+	++	++	++	++	++	++	++	++	++	++	++	++
Mica	++	++		+	++	++	++	++	++	++	++	++	++	++	++
Kaolinite		+			+	+	+	+	+	+	+	+	+	+	+
Calcite	+						++			++	++	++	++	++	+
Dolomite	++						++	++	++	++	++	++	++	++	++
Plaster of Paris (bassanite)		++	++	++	++	++	++	++	++	++	++	++	++	++	++

Major constituents are indicated by ++; minor constituents by +; weight percentages of asbestos minerals (chrysotile and tremolite) were determined by step-scanning x-ray diffraction

regulations. Measurements were made utilizing the standard techniques of the National Institute of Occupational Safety and Health (NIOSH) for asbestos sampling and analysis.

During pole sanding, 7 out of 10 samples exceeded the then existing threshold limit value (TLV) of 5 fibers/cm³ longer than 5 μ m. (The TLV has since been reduced to 2 fibers/cm³ longer than 5 μ m). Hand sanding generated fiber concentrations close to or in excess of the TLV. It may be emphasized that the asbestos fiber concentrations generated by sanding were similar to those measured in the work environment of asbestos insulation workers. Mixing of drywall taping compounds, a procedure which is currently not frequently used, generated fiber counts 7-10X the threshold limit values. Moreover, the data in Table II show that, at various distances from the actual taping operations, detectable fiber concentrations are present in adjacent rooms during the various procedures. Other building trades would thus also be exposed. It should be emphasized that the fiber concentrations measured by standard procedures using optical microscopy reflect only a small proportion of the number of fibers actually present since they fail to detail fibers shorter than 5 μ m in length and 0.5 μ m in width. However, electron microscopic analyses of these air samples showed a large number of these smaller fibers too fine to be seen by optical microscopy (Figure 2). The disease-inducing potential of such fibers may be significant.

prevalence of asbestos disease among taping workers

A clinical field survey of 114 taping workers employed in the New York Metropolitan area was conducted. Examination included a careful life-time occupational history with recording of all previous occupations on a specially designed questionnaire. Interview also included past medical history, smoking history and review of chest symptoms, such as chronic bronchitis, dyspnea and cyanosis. Chronic bronchitis was recorded as present if the individual gave a history of phlegm production for more than three months per year during a period of two years or longer. Each individual had a comprehensive physical examination with

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TABLE II
Asbestos Fiber Levels During Use of Taping Compounds
Containing Asbestos Minerals

Operations	Number of samples	Mean fiber concentration (fibers/cm ³)	Range of fiber concentrations (fibers/cm ³)
Pole sanding (3-5 ft.)	10	10.0	1.2-19.3
Background (8 ft.) same room	3	8.6	3.5-19.8
Background (25 ft.) adjacent room	2	4.8	0.7- 8.8
Hand sanding (3-5 ft.)	11	5.3	1.3-16.9
Background (8 ft.) same room	2	2.3	2.1- 2.5
Background (15 ft.) adjacent room	2	4.3	1.5- 7.1
Dry mixing (3-5 ft.)	2	47.2	35.4-59.0
Background (10-20 ft.) same room	3	5.8	0.5-13.1
Background (18-35 ft.) adjacent room	2	2.6	2.1- 3.1
Sweeping floor (10-50 ft.) 15 minutes after sweeping	1	41.4	
35 minutes after sweeping	1	26.4	

special emphasis on examination of the chest. Standard 14" X 17" chest roentgenograms were taken; both PA and oblique projections were taken; interpretation of chest films was done by a panel of 5 experienced readers, and a consensus reading was thereafter obtained.

Readings were classified according to the ILO U/C International Classification of Radiographs of Pneumoconioses, and a reading was considered abnormal if fine or moderately thin irregular opacities were present (s or t $\geq$ 1/0) and/or pleural thickening was present ($\geq$ a-1).

The age distribution of the examined workers is shown in Table III. Only 17 (15%) were less than 30 years of age, while 75 workers (66%) were between 30 and 49 years. Many young workers had been in the taping trade for several years; Table IV illustrates the distribution of duration since onset of taping. The majority of the workers had a long history in the trade; for 67 individuals (59%), 15 years or more had passed since the onset of occupational exposure to taping compounds, while 19 workers (17%) had begun exposure 10 years prior to the examination or later.

TABLE III
Age Distribution of
114 Drywall Tapers

Age	Number	Percent
< 30	17	14.9
30 - 39	31	27.2
40 - 49	44	38.6
50 - 59	15	13.2
60 +	7	6.1
Total	114	100.0

TABLE IV
Duration Since Onset of Exposure
of 114 Drywall Tapers

Years since onset of exposure	Number	Percent
0 - 9	19	16.7
10 - 14	28	24.6
15 - 19	31	27.1
20 - 24	26	22.8
25 -	10	8.8
Total	114	100.0

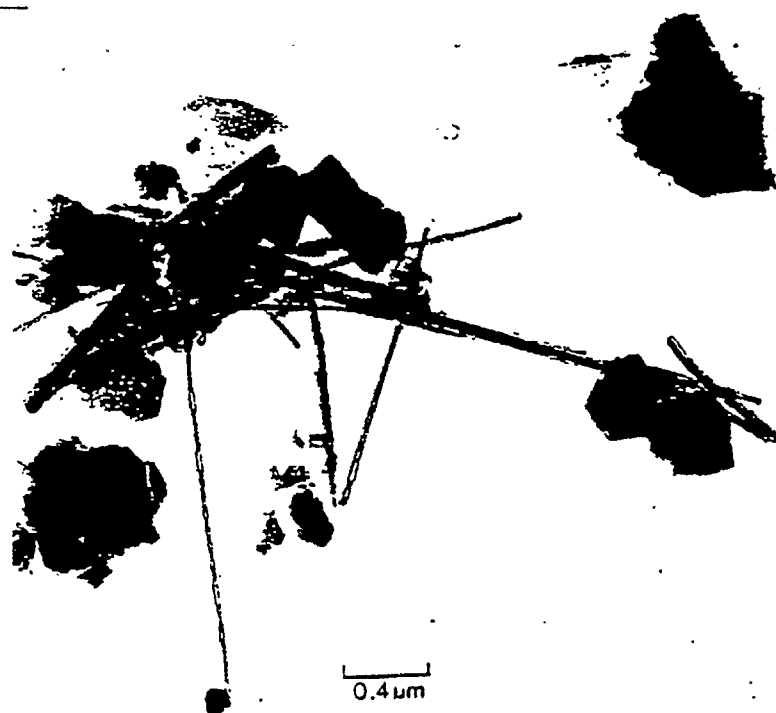


Figure 2 — Electron photomicrograph of air sample taken during sanding of taping compound. Numerous chrysotile fibers are visible, most of which are shorter than 1 micrometer. They would not be visible by optical microscopy. Granular particles are micas, clays and plaster of Paris.

There were 79 smokers (69%), 13 non-smokers (11%) and 22 ex-smokers (19%) among the examined workers. Chronic bronchitis was reported by 25 individuals (22%), all of whom were heavy smokers. 10 workers (9%) gave a history of dyspnea on exertion, 9 of whom were heavy smokers; one was an ex-smoker. Chest examination revealed dry rales in 10 individuals (9%), 7 of whom were smokers and 3 of whom were ex-smokers. 6 of these, however, had a positive chest x-ray for parenchymal disease; none had any visible pleural abnormality.

TABLE V
Roentgenographic Abnormalities (Parenchymal)
Among 110 Drywall Tapers

Years since onset of exposure	Total number examined	Abnormal x-ray ($\geq$ S, 1/0)	
		Number	Percent
0 - 9	19	5	26.3
10 - 19	56	22	39.3
20 +	35	18	51.4
Total	110	45	

Rhonchi were found in 15 workers (13%), 14 of whom were smokers. 5 of these showed parenchymal disease on x-ray, while 2 had pleural thickening.

The roentgenographic abnormalities among the examined workers are shown in Tables V and VI. The prevalence of abnormal chest films increased with longer duration since onset of exposure; among those with 10 years from onset of exposure or less, 26% had positive roentgenographic signs ($\geq$ S, 1/0); this prevalence increased to a high of 51% among

TABLE VI
Pleural X-ray Abnormalities Among
109 Drywall Tapers

Duration (years)	Total number examined	Number of abnormalities	% abnormal of group
0 - 9	34	0	0.0
10 - 19	50	4	8.0
20 +	25	5	20.0
Total	109	9	8.2

workers with 20 years or longer since onset of exposure.

Pleural thickening was found in 9 (8%) of the examine workers, 5 of whom had concomitant parenchymal abnormalities. All individuals with pleural thickening had a history of long duration since onset of taping; the mean duration since onset was 19.1 years (range 13-22), (Table VII).

discussion

The identification of asbestos in drywall spackling and taping compounds and the considerable prevalence of asbestosis among those who use these materials have added a new occupational health perspective to industrial drywall construction, a trade that is of growing importance in the United States and throughout the world. In fact, the prevalence of asbestosis in this population of drywall tapers is similar to that found by others among asbestos insulation workers.⁽²⁾ In these studies, evidence of pulmonary asbestosis was present in 542 of 1,117 examined workers, i.e. in 48.5% (having radiological change as sole criteria); this is to be compared with the present study, which demonstrated a prevalence of 40.9%. It is of considerable importance to emphasize the inadequacy inherent in the standard NIOSH procedure for determining asbestos exposures which uses optical microscopy alone. Electron microscopic examination of air samples shows a large number of fibers too fine to be seen by optical microscopy, but which nevertheless may cause disease. Analysis of the lung of a taping worker who suffered from lung cancer, showed very large numbers of fibers, all too small and fine to be seen with the optical microscope. This has been reported elsewhere.⁽³⁾

conclusion

We have found that taping workers in drywall construction have risk of exposure to asbestos. Our findings suggest that asbestos disease, as a

result, is an important hazard in this trade. It is therefore important and urgent that appropriate measures be taken in order to prevent further asbestos exposure. The availability of asbestos-free taping compounds would be an important step toward this objective. Despite this, however, asbestos-related disease will inevitably be seen in taping workers in the future since asbestos-free materials were not widely introduced into the trade until very recently, and the result of past exposure will be with us for some decades in the future. The question of appropriate long-term surveillance is important and unresolved.

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TABLE VI
Abnormalities Among
Wall Tapers

Number of abnormalities	abnorm of gro
0	0.0
4	8.0
1	14.3
9	83.3

Editorial

Low level exposure to asbestos: is there a cancer risk?

In the early days of asbestos use when its harmful potential was not understood hygiene standards at the workplace were extremely low. Airborne dust concentrations were uncontrolled and fibre levels in excess of 100 per ml were not unusual. This type of exposure led to the discovery that inhalation of asbestos produced pulmonary fibrosis,¹ bronchial carcinoma,² and mesothelioma.³ Efforts to reduce concentrations of asbestos fibre began in the 1930s and, since the 1950s, increasingly stringent limits have been imposed by law. Some countries have banned many uses of asbestos, although it remains almost essential for some special packing and friction products. Various fibre substitutes have been introduced in advanced countries, especially for plastic or cement products, though these are generally less durable and more costly. The latter point is a serious matter in third world countries where asbestos cement products are well-nigh essential for water and drainage pipes and for building construction. In addition, it now seems possible that some manmade mineral fibres could carry similar risks to health.⁴

The use of asbestos today in the developed world may only be undertaken under strict control, in most countries at levels of 1 fibre/ml or less. Exposure levels in buildings and in the general environment of our cities, resulting from the use of asbestos products, are far lower still. The question of whether such low occupational and environmental exposures are free from risk has become a major practical, social, and legal issue.

As it is apparent that controlling dust has largely eliminated the risk of pulmonary asbestosis, the main concern is now over cancer and whether or not there is a safe threshold below which asbestos does not cause tumour production. Evidence on this matter is afforded by both experimental studies in laboratory animals and epidemiological studies in human populations exposed to asbestos. Both approaches have their limitations. Animals may be exposed to measured doses of asbestos but their short life span makes direct extrapolation of the results to man difficult. Epidemiological studies, on the other hand, suffer from great uncertainty about the precise nature and intensity of exposure, especially in the distant past. Whereas the impossibility of proving a complete negative is accepted, both the epidemiological and

experimental findings provide a basis for rational decisions.

Experimental studies have shown that the pathological effects of asbestos depend not only on the mass of dust deposited in the lung tissues but on fibre length and durability. Wagner *et al* reported on rats exposed to the main types of asbestos in which a constant mass of dust was administered for periods of one day to two years.⁵ The degree of diffuse pulmonary fibrosis varied proportionately with duration of exposure. The experience of rats exposed for one day only was similar to that of unexposed control animals. The production of pulmonary tumours was less clearly related to duration but in general more tumours occurred in animals exposed for long periods. Some animals exposed for a single day developed pulmonary tumours but, again, similar numbers were found in control animals. Information is needed on the effect of a range of doses administered over a long period but such studies have not been undertaken. The production of mesotheliomas by intratracheal or intraperitoneal injection of asbestos provides a less expensive method of examining the carcinogenic effects of widely varied doses. Davis reported studies in which reference samples of crocidolite, amosite, and chrysotile had been injected into the peritoneal cavities of rats at doses from 25 mg down to 0.01 mg.⁶ A clear dose response was found with all three types of asbestos fibre, but whereas crocidolite did not produce tumours at the lowest dose level, chrysotile and amosite did. With each fibre type, the relation was linear when plotted on a log scale; however, with all three, both the time to first tumour and the mean induction period increased progressively with reducing dose. Tumours produced by the lowest doses in general occurred during the last few months of the rat's life, suggesting that lower doses still had insufficient time to produce any tumours.

It also appears that not all fibres are equally harmful and evidence is accumulating that very short fibres may be innocuous. Both Stanton and Wrench and Pott and Friedrichs reported that dust samples which produced the most mesotheliomas in rats were those that contained the highest number of long and thin fibres.⁷ This work has been progressively refined, and it is now evident from injection studies that the most dangerous fibres are those with a length $> 8 \mu\text{m}$ and a

diameter of $< 0.25 \mu\text{m}$.⁹ When any fibres longer than $8 \mu\text{m}$ are considered, the lowest injected doses (0.01 mg) which produced any tumours contained about 410 000 fibres of chrysotile and 110 000 fibres of amosite; a dose containing 60 000 fibres of crocidolite produced none. At all dose levels chrysotile was the most carcinogenic dust followed by amosite, with crocidolite the least so.

Long term inhalation studies have recently been conducted using short fibre preparations of amosite and chrysotile.^{10,11} In these experiments the effect of short fibre dust was compared with a normal dust cloud of the same batch of asbestos containing many long fibres. The amosite preparation was extremely short with almost no fibres greater than $5 \mu\text{m}$ in length; the short fibre chrysotile was less satisfactory in that some long fibres remained. Whereas the long fibres of both chrysotile and amosite produced large amounts of pulmonary fibrosis and a considerable number of pulmonary tumours, the short fibre amosite preparation produced neither fibrosis nor tumours. Short fibre chrysotile produced a small amount of fibrosis and some tumours, roughly in proportion to the number of long fibres present.

There remains the question of whether, to cause disease, asbestos fibres need be retained in the lung for long periods; a low retained dose might well produce little disease regardless of the number of fibres inhaled. In this respect chrysotile and the amphibole varieties differ appreciably. After varying inhalation periods at high dose levels, rats have been shown to retain up to ten times more amphibole than chrysotile.¹¹ The recent studies of Davis *et al* have extended these findings to include fibre length.¹⁰ Fewer long than short fibres were present at the end of dusting for both chrysotile and amosite; the elimination of short fibres was faster than that of long but, regardless of size, chrysotile was cleared from lung tissue much faster than amosite. Amphibole fibres are extremely durable even in aggressive chemical environments whereas chrysotile undergoes decomposition readily, even in water. Davis *et al* reported that in rats, 18 months after inhalation of chrysotile at high doses for one year, no fibres remained in pulmonary macrophages or in direct contact with any of the other pulmonary cells.¹² Only a few fibres were detected and these in old acellular fibrous tissue or in thickened basement membranes, chemical backwaters where fibre dissolution is retarded. The rapid removal of chrysotile and the durability of amphibole fibres have also been noted in several studies of human lung tissues.¹³⁻¹⁵

Whereas epidemiological studies in man can attempt to measure risk directly their capacity to do so becomes increasingly unreliable as the level of exposure falls. Partly, this is due to deficiencies in exposure data but mainly it is because no reference

population is ever more than roughly comparable with the exposed study group. Thus in not one of the ten or so eligible cohort studies of lung cancer mortality in asbestos workers does the fitted line for exposure response pass through the origin unless forced to do so.⁶ Even the remarkably good approximation to linearity observed in these data sets is possibly determined more by duration than intensity—these two components of exposure not having yet been separately assessed. Valid though the results of well designed observational surveys are, for these reasons alone any estimate of risk carries a wide margin of possible error. In chrysotile mine and mill workers, for example, no statistically significant increase in lung cancer mortality would have been detected in nearly 2000 men with at least 20 years employment at an average airborne fibre concentration of about 20 fibres/cc, though the linear exposure response hypothesis would imply that there was one.¹⁷ Even in the far more hazardous environment of asbestos textile factories the same would probably be true up to levels of about 2 fibres/cc.¹⁸ Such levels are as high or higher than current occupational limits and orders of magnitude above those causing concern in public buildings. It also appears that in amosite asbestos factory workers the lower the estimated dose, the longer it took for related mortality to become evident and the smaller its magnitude.¹⁹

The epidemiological data on exposure response are much more tenuous for mesothelioma than for lung cancer. Such quantitative information as there is points to a direct relation between the risk of this disease and some combination of duration and intensity of exposure, but strongly modified by fibre type. There is now considerable evidence that pure chrysotile per se seldom causes mesothelioma in man and that most cases are due to amphibole fibres—crocidolite and amosite in particular—but increasing attention is now being directed at fibrous tremolite. Tremolite asbestos has little or no commercial value but commonly contaminates many mineral deposits, some of which, such as chrysotile, talc, and vermiculite, are widely used industrially, and others not. The potential importance of the tremolite factor, noted by Pooley and Rowlands *et al*, was directly shown in vermiculite miners and millers whose only exposure to mineral fibres was from contaminating tremolite.^{11,20} The lung cancer and mesothelioma experience of this cohort was, dose for dose, many times worse than that of Quebec chrysotile miners and millers. The contribution of fibrous tremolite to mortality from these causes had also been suspected in New York State talc miners many years earlier.²² Although the proportion of tremolite in asbestos dust in the Quebec mines and mills is less than 1%, most fibres found at necropsy in the lungs of both mine workers and chrysotile textile

comparable with one of the ten or more mortality in one for exposure less forced to do approximation to is possibly determinancy—these two having yet been the results of well for these reasons wide margin of mill workers, for increase in lung detected in nearly employment at an on of about 20 response hypothesis. Even in the far asbestos textile factories up to levels of high or higher orders of magnitude in public buildings, asbestos factory workers, longer it even and the

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factory workers are of this type, not chrysotile.²² It thus seems probable that even the relatively low risk of mesothelioma in individuals reputedly exposed only to chrysotile may largely result from tremolite contamination.

Further insight into the magnitude of the risk of cancer from low level exposure may be obtained from national trends in mesothelioma incidence and mortality. During the past 20 to 30 years the pattern has been remarkably similar in the United States, Canada, Britain, and probably Scandinavia.²⁴ The data suggest that, until the 1950s, the annual mortality from mesothelioma was similar in men and women, at about 1–2 per million. Since then the trends have separated; mortality in men has risen steeply in parallel with using asbestos some 30 to 40 years earlier whereas in women there has been no significant increase. The female trend is particularly important since it is known that cases have occurred in women who have worked with asbestos or lived in the household of asbestos workers. It would be expected, moreover, that during the years in question increased awareness of what was once considered a rare tumour might lead to improved ascertainment. If, despite this, there has been no apparent increase in female mortality in a combined population of some 300 million subjects observed for at least 15 years the risk attributable to non-occupational exposure must be extremely small.

What conclusions may be drawn from a synthesis of these experimental and epidemiological findings? We cannot say, and it may be impossible to prove or disprove that at very low levels of exposure to asbestos the risk of cancer is zero. None the less, further statistical analyses are needed to test the linearity of risk in relation to intensity of exposure. So far as lung cancer is concerned, clearly any increase in risk at exposure below today's control limits, even in men who smoke more than they now do, would not be detected. The same could be said for mesothelioma in relation to commercial chrysotile but with less confidence for the commercial amphiboles, crocidolite, and amosite. If the tremolite contamination could be removed from chrysotile the safety margin would be wider. Plausible explanations for these assertions have been shown in the laboratory. Chrysotile is poorly retained and rapidly cleared from the respiratory tract; short fibres, which predominate in low level pollution, have little or no carcinogenic potential; with decreasing dose tumour latency may be prolonged beyond the normal life span. General toxicological experience, and the probability of defence mechanisms, would also suggest the existence of an effective threshold, or at least a sigmoid form of exposure-response.

The adverse effects of asbestos have probably been more extensively studied than any other environ-

mental hazard; and manmade mineral fibres may well require the same attention. It seems unlikely that much more scientific information on asbestos can be expected than is now available. Rational decisions will depend on questions of cost, benefit, and the appreciation of risk. It will be for society, not science, to decide what priority should be given to the reduction of unsubstantiated and undetectable risks, even from such dreaded effects as mesothelioma and lung cancer.

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