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ROYAL COMMISSION ON MATTERS OF
HEALTH AND SAFETY ARISING FROM
THE USE OF ASBESTOS IN ONTARIO

SUMMARY OF THE PHASE II HEARINGS ON HEALTH, MONITORING AND REGULATORY ISSUES

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ONTARIO ROYAL COMMISSION ON ASBESTOS

INTRODUCTION TO THE COMMENTS OF
AIA/EA ON HEALTH, MONITORING AND REGULATORY ISSUES

In the course of the proceedings before the Ontario Royal Commission on Asbestos, a substantial segment of the world's experts on the health effects and monitoring of asbestos have appeared. Although there were clearly differences among the witnesses, and although many issues were deemed still unresolved, consensus was reached in important areas that should serve as the basis for determination of a rational societal policy toward asbestos:

-- All human health effects of asbestos are dose-related, and present-day health effects are attributable to high, often uncontrolled, exposures of the past.

-- Asbestos exposures today are orders of magnitude lower than the historical exposures upon which knowledge of ill effects is based.

-- Although the potential health effects of the lower exposure levels found in the workplace today cannot be determined definitively, the best scientific evidence indicates the risks are minimal, if not insignificant.

-- Even conservative overestimates of the health effects of asbestos at low exposure levels based on linear, non-threshold assumptions about the dose-response relationship predict that properly controlled asbestos use today poses de minimis, or well within acceptable level, occupational risks.

-- Often expressed fears about asbestos risks to the general population have been widely exaggerated; no public health risk need exist from the continued proper use of modern encapsulated and locked-in asbestos products.

Based on these principles, a rational societal policy toward asbestos recognizes that the substance continues to serve an important role in many applications where it can be used with minimal or no risk. At the same time, the evidence demonstrates that all uses must be closely scrutinized to minimize unnecessary exposures.

In order to assist the Commission in understanding and synthesizing the substantial record that has been accumulated, we present this Summary. The Summary covers the following questions which are before the Commission:

(1) What is known about the biology of asbestos fibers: their respirability, their deposition and disposition in the body, and the effect on such fibers of the body's clearance and immune mechanisms?

(2) What is known about the pathogenicity of asbestos fibers and the mechanisms by which they cause human disease?

(3) Do asbestos fibers differ, given varying chemical compositions, sizes and dimensions, in their biologic and pathogenic significance?

(4) What diseases have been found to be associated with asbestos exposure and with what dose-response relationships?

(5) To what extent do other factors, particularly smoking, account for disease among asbestos-exposed persons?

(6) What conclusions can reasonably be reached in predicting the risks of asbestos exposure at the lower levels found in the workplace today, and in general population exposure?

Following our discussion of the exposure and health effects evidence, we suggest to the Commission a series of societal measures that appear appropriate to minimize asbestos risks in the future. These measures include:

-- Continuing efforts to employ all reasonable, technologically feasible methods to control asbestos fiber emissions in fixed-site mines and mills and primary and secondary manufacturing facilities and to monitor through use of the membrane filter method such emissions to ensure achievement of acceptably low exposures.

-- Employment of recommended work practices in non-fixed workplaces -- such as construction sites -- in order to eliminate excessive fiber emissions.

-- An educational labeling scheme for asbestos-containing products to ensure their proper handling.

-- Promotion of smoking cessation programs among asbestos-exposed workers.

-- Continuing surveillance and research to answer many of the more particular, but still unresolved, questions concerning asbestos.

Our proposal does not include the government-mandated phase-outs or bans of asbestos use that have been suggested in some quarters. Such regulatory steps cannot be justified given the extensive evidence supporting the feasibility of continued safe use. Appropriate measures are now being used, and further measures could be employed, to control asbestos fiber emissions. It should be the role of Government to ensure that such measures are employed whenever asbestos is used. However, given the minimal risks when such controls are used, the marketplace should be relied upon to decide whether the cost of such practices is justified by the necessity or desirability of asbestos use.

ONTARIO ROYAL COMMISSION ON ASBESTOS

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In an era of increasing public concern about carcinogens, asbestos has often been a centerpiece of public attention. Because the adverse health effects of asbestos generally develop only twenty or more years after initial exposure, the consequences of very high occupational exposures during World War II and before have become apparent only in recent years. The medical consequences of these very high occupational exposures must, however, be distinguished from the related, but distinct, issue of whether the much lower asbestos exposure levels prevalent today pose a significant risk to either workers or the general population. Consideration of this issue requires a detailed examination of the evidence on exposures to and health effects of asbestos.

Asbestos is a naturally occurring mineral found in many parts of the world. Through natural processes, it spreads to ambient air and waters with the result that human exposure is virtually unavoidable. Although very high occupational exposures to asbestos occurred in the past, improved controls in modern manufacturing processes and changes in the products in which asbestos is used have vastly reduced exposure today. Within the past decade, most mines and

manufacturing facilities have achieved continued substantial decreases in exposure levels. As a result, most asbestos workers experience exposures well below existing governmental standards. Almost all products now marketed employ asbestos embedded in a matrix that eliminates or substantially limits fiber release. As a result, little likelihood of harmful exposure exists for product users. General population exposures to asbestos, where they exist at all, are several orders of magnitude lower than today's workplace exposures.

Fiber Type

Since the associations between high asbestos exposures and asbestosis, lung cancer and mesothelioma were first discovered, medical research has concentrated on trying to establish what size and shape and which type asbestos fibers are most dangerous. Animal research has essentially established that fibers longer than eight microns and less than one micron in diameter are the most pathogenic. Distinctions among the major fiber types (chrysotile, amosite and crocidolite) are less clearly established. Apparently diverging medical data complicates determination of relative pathogenicity. Although animal and tissue culture experiments tend to demonstrate more disease-causing potential for chrysotile, the human evidence has usually shown greater disease, particularly mesothelioma, among persons exposed to crocidolite and amosite.

Asbestos Epidemiology

The many epidemiology studies of workers with high asbestos exposures in the past have confirmed associations with asbestosis, lung cancer and mesothelioma and have also strongly suggested that very little if any risk still exists at today's much lower exposures. These studies have also emphasized the extent to which smoking accounts for much of the past disease.

Asbestosis, a fibrotic lung condition that may lead to death in its advanced stages, has occurred almost exclusively among very heavily exposed workers. This chronic disease is closely related to sustained high exposures and at its advanced clinical stages is very unlikely to occur at today's exposure levels -- even among lifetime asbestos workers.

Threshold levels below which asbestosis symptoms have not been found have been established in the asbestos-cement worker studies of Dr. Weill. Even in other studies where no clear thresholds have emerged (the Canadian miners and Rochdale, England textile workers), very little asbestosis would be predicted based on extrapolations from the exposures of those cohorts to workers at today's exposure levels. Continuing surveillance of worker populations in Australia and the United States demonstrates the declining incidence of asbestosis in populations being exposed to lower and lower asbestos concentrations. The consensus opinion of

the experts who appeared before the Commission was that asbestosis is not a significant occupational health concern for the future.

Although greatly increased lung cancer rates have been found among some asbestos workers, the amount of disease is closely related to worker smoking habits, as well as the high doses experienced in the past. Although definitive evidence has not yet been developed of a dose below which no increased risk exists, absence of increased mortality due to respiratory malignancy in several large cohorts for the persons with lower -- although still much higher than modern -- exposures provides strong support for concluding that no substantial hazard exists for modern asbestos workers. The medical evidence strongly suggests that any remaining risk is very small, and, for non-smokers, close to non-existent.

Several large cohorts of asbestos workers have failed to exhibit increased lung cancer risks. The subcohort of the Canadian miners with exposures below approximately 20 fibers/cc for twenty years, the Louisiana asbestos-cement workers with exposures below 100 appcf-years, and the asbestos friction material production workers studied by Newhouse and Berry have not exhibited increased lung cancer risks -- despite their greater than current worker exposures. Although other studied cohorts have exhibited some lung cancer risks at such exposure levels, these three well-documented studies -- each of which specifically focused attention on dose-response

relationships -- suggest strongly that the much lower exposure levels found in industry today are so low that any remaining risks are too small to be detected.

Central to any assessment of the increased lung cancer risk of asbestos is the predominant role of smoking in determining risk. Although there is some evidence that lung cancer risks are increased for non-smokers, almost all lung cancer has occurred among smokers. At today's asbestos exposure levels, reduction of lung cancer incidence is far more dependent on termination of smoking than on elimination or reduction of asbestos exposure.

Mesothelioma is a rare tumor of the pleural and peritoneal tissues. Although often associated with asbestos exposures, mesothelioma also occurs in the absence of asbestos exposure. Like asbestosis and lung cancer, mesothelioma has been shown to be related to dose and to have occurred primarily among heavily exposed persons. Its future occurrence among persons exposed at today's occupational levels is likely to be quite rare.

Unwarranted fears of mesothelioma risks to the general population have been generated by scattered references to individual cases allegedly caused by casual exposures. In fact, all the existing evidence suggests that, on the one hand, those cases discovered among worker families were probably due to quite substantial exposures caused by contact with workclothes and prolonged household reentrainment

of fibers, and, on the other hand, cases where asbestos exposures were in fact casual are most likely attributable to the natural background rate for this disease. At today's exposure levels, the mesothelioma risk predicted, even if based on conservative extrapolation models, would be quite low.

Although some studies have reported associations between asbestos exposure and other forms of cancer, the overall evidence is equivocal. The scientific evidence demonstrates that such cancers normally occur only after very high exposures and account for a minimal amount of asbestos-related disease.

Risk Extrapolation

Because asbestos related diseases typically take more than 20 years to become manifest, no groups of workers exist who have been studied for long enough to provide documented evidence of the risk, if any, at current low exposure levels. However, using risk extrapolation methods, it is possible to set upper limits on the magnitude of such risks.

All human activities, including employment, involve risk. Man accepts risks every day, often without recognizing their magnitude. Many studies have quantified commonly accepted risks in order to place in context the risks posed by use of substances such as asbestos. Once risks are placed in such a context, rational societal decisions can be

made about the efficacy of risk reduction measures. In order to make such assessments, it is necessary to project the asbestos dose-response relationship to the low exposure levels prevalent in current asbestos use.

Dose-response relationships for asbestos-related diseases have been found to be linear at those high doses for which empirical data exist. It has thus often been suggested that a linear relationship will continue to lower doses. Such linear extrapolations, though, have not been empirically demonstrated and most likely overstate risks at low exposure levels. Their use thus sets an upper limit on potential risk.

Central to predicting future risks are determinations of past exposures among the studied cohorts. Concerted efforts to construct individual exposure profiles have been made for asbestos worker cohorts by Drs. McDonald, Weill, Enterline, Newhouse and Berry, Finkelstein, and Dement. It is from their studies that the primary data for extrapolating risks must be sought. Although most of these cohorts' exposures were initially measured in particles, not fibers, adequate work has been done on conversion to provide meaningful guidance for current regulatory purposes.

Also central to predicting current risks is an understanding of current exposure levels. Under the two fibers/cc standard that prevails in most Western nations, average exposure levels well below that standard exist for most

workers. Indeed, other than for the relatively small number of workers in mining and milling and primary and secondary manufacturing, most occupational exposures to asbestos are intermittent, and, when recommended work practices are employed, de minimis. For such workers, average exposures are thus many times lower than the two fiber standard. Among the entire worker population in Canada or the United States, therefore, only a few thousand workers are likely to have lifetime exposures of more than 10 fiber-years; most workers will have lifetime exposures below 1 fiber-year.

Applying extrapolation to the best available data indicates that the current risk to asbestos workers is within the range of risks faced every day by most workers. Once the significant part of that risk due to cigarette smoking is taken into account, the risk is well below most other occupational risks.

The disease risk predicted from the asbestos epidemiologic studies varies to some extent from study to study. Some of the variance can be explained by methodological differences among the studies, but some of the variation may also be due to true differences in disease potential in various asbestos operations. At one end of the spectrum, the risks predicted from the chrysotile mining experience is so low at current exposure levels as to leave little question about conclusions that mining and milling operations are acceptably safe. Some of the primary manufacturing

fibers. However, such discriminations often are unnecessary, and, where needed, can be made by supplementing membrane filter measurements in mixed fiber environments with occasional electron microscope characterization of dust cloud constituents. There is therefore no reasonable basis for change in the currently-employed monitoring system.

Potential alternatives to the membrane filter technique are infeasible for routine monitoring. Electron microscopy may be useful to characterize fiber proportions in mixed fiber environments, but no standardized method for either scanning or transmission electron fiber counts has been developed. Further, electron microscopy measurements cannot be related to membrane filter results or to the existing health evidence; capital costs are very high; insufficient qualified laboratories and technicians exist to meet existing monitoring needs; and, except for scanning electron microscopy at low magnification (offering little advantage over membrane filter measurement), operating costs per sample are very high.

In establishing workplace standards based on membrane filter monitoring, the substantial measurement variability, as well as day-to-day variations in workplace concentrations, must be considered. Given these sources of variability, and the generally recognized 0.1 fiber/cc detection limit for the membrane filter method, the lower bound for possible asbestos standards is in the range of 0.5 to 1 fiber/cc.

With any lower standard, employers and enforcement officials would be unable to determine compliance status with reasonable confidence. Standards in the range of one to two fibers/cc can be implemented using membrane filter monitoring, if employers allow for variability, by maintaining average workplace concentrations well below mandatory standards.

Membrane filter monitoring can be used, along with occupational exposure standards consistent with the method's accuracy, to achieve highly protective workplace conditions. Because employers must measure well below the occupational standard to obtain reasonable assurance of compliance, average workplace concentrations will be maintained well below mandatory standards.

Outside the workplace -- in ambient air and water, or in buildings -- the membrane filter method is inadequate to provide meaningful monitoring results. The extremely low asbestos concentrations found in such settings fall well below the membrane filter detection limit. In addition, membrane filter fiber counts outside the workplace would be meaningless, because the membrane filter method does not distinguish between asbestos fibers and the many other fibrous materials found in the general environment.

Although electron microscope techniques meet some of the technological requirements for environmental monitoring, there is no standardized electron microscopy method and no

studies predict similarly very low risks, but a few studies predict higher risks. Although all such differences cannot be clearly reconciled with the present state of knowledge, even the higher risks predicted by these latter studies fall within the range of normally accepted occupational risks. If that proportion of the risk attributable to lung cancer caused by smoking is clearly delineated, the predicted asbestos risk is toward the low end of accepted occupational risks even when determined based on the studies predicting higher risks.

Environmental Risks

Despite the fears generated by the media, the opinion of most scientists, as well as expert governmental-scientific bodies, has been that no significant risk due to asbestos exists for the general public. Risk extrapolation methods can also be used to establish upper limits on the asbestos risks that theoretically would exist for the general population. These risks are well below a number of commonplace risks faced daily by the general public.

The absence of evidence of a general population risk applies to both airborne and waterborne asbestos. Although serious medical consequences have been associated with heavy exposures through asbestos inhalation, no such body of evidence exists for ingestion. Animal studies of asbestos ingestion, including on-going state-of-the-art bioassays,

have failed to demonstrate any carcinogenic or other health risks. Similarly, the human epidemiologic evidence predominantly fails to find any association between ingestion and cancer.

Monitoring

In addition to the medical evidence, considerable testimony was presented to the Commission on the monitoring of asbestos. This testimony provides further important guidelines for governmental policies on asbestos control.

The membrane filter method is a practical and effective technique for conducting the large volume of routine occupational monitoring required in the asbestos industry. It has been adopted throughout North America and Europe, is the basis for current occupational standards, and can be related to the available epidemiological evidence. The advantages of membrane filter monitoring include its relatively low cost and simplicity, the ready availability of necessary equipment and skilled operators, and the significant progress made in the last decade toward methodology standardization.

The membrane filter method also has several important limitations. The basic method does not distinguish among asbestos fiber types or between asbestos fibers and other fibers; and it provides only an index of fiber concentrations, because it does not count very short or very thin

basis for relating electron microscopy results to the health evidence on asbestos. There is therefore no basis for establishing environmental standards or for comparing results from different laboratories quantitatively.

Nevertheless, electron microscopy monitoring results of air and water -- which are qualitatively useful when compared with similar background measurements -- demonstrate no cause for concern. Both in England and Ontario, investigators have recently determined that fiber concentrations in buildings with asbestos insulation do not exceed background levels found in outdoor measurements -- except where insulation has recently been removed. The present evidence thus shows little need for environmental asbestos control standards.

Policy Implications

The comprehensive medical evidence on asbestos that allows estimation of the extent of risk, if any, posed by current asbestos mining, manufacturing, installation and use, makes it possible to design an appropriate societal policy for the substance. Such a policy recognizes both the value of asbestos as a versatile mineral and the necessity for educational and control measures to minimize human exposures. A variety of government initiatives would be useful to guarantee reasonable control of fiber emissions.

First, in the mining and milling of asbestos, continuation of the extensive control measures that have been adopted in recent years is necessary. The well-documented mining epidemiology evidence demonstrates that such levels provide an acceptably safe workplace.

Second, continuation of the control measures that have succeeded in recent years in vastly reducing exposure levels in the manufacture of asbestos-containing products (including, inter alia, the most significant uses -- asbestos-cement products, friction materials, and flooring) is necessary to maintain a safe workplace. Although variations exist among the risk estimates derived from manufacturing cohorts, the much-reduced exposure levels common today do not pose significant risk of disease.

Third, new inducements or requirements to increase use of recommended work practices in the installation and removal of asbestos-containing products are desirable. Although workers in industries where exposure to asbestos is infrequent and intermittent are unlikely to have cumulative asbestos exposures in the ranges experienced by workers exposed on a daily basis, it is nonetheless advisable to control unnecessary exposures. Most such non-fixed site exposures can be dramatically reduced through proper work practices. Similar cost-effective work practices can and should be employed in fixed site garage work with asbestos friction products.

Fourth, educational labeling of raw asbestos fiber and many asbestos-containing products to describe and encourage the use of safe work practices will greatly reduce the potential for inadvertent and unnecessary fiber emissions.

Fifth, smoking cessation programs can greatly diminish the risk of disease, especially lung cancer, among asbestos workers. Indeed, with asbestos levels much reduced in the workplace today, considerably more will be accomplished by such anti-smoking programs than by further reductions or elimination of asbestos exposure.

Finally, continued medical and technological research is warranted to fill existing gaps in knowledge of the health effects of asbestos and to improve further the capability of minimizing fiber release while still making beneficial use of this versatile mineral.

CHAPTER I

I. THE VERSATILITY AND USEFULNESS OF ASBESTOS AND INDUSTRY'S SUBSTANTIAL PROGRESS TOWARD CONTROLLING HUMAN EXPOSURES.

Prior to exploring the asbestos health effects evidence in depth, it is useful to establish its context by describing both the value of asbestos that has led to its widespread use over the past 100 years, in Canada and throughout the world, and the steps that have been taken to reduce exposures as the adverse health effects of heavy exposures have become known. The following chapter establishes such a context, emphasizing in particular that:

-- Asbestos is a naturally-occurring mineral possessing a variety of unique attributes that make it the most cost-effective material available for many applications.

-- Many of the applications of asbestos that led to considerable human exposures have been terminated in the past ten years.

-- Almost all current applications of asbestos are in products where fibers are locked-in or encapsulated, thus greatly minimizing, if not eliminating, any significant human exposures.

-- Substantial progress has been made in the workplace to reduce asbestos exposures, even within the past ten years, such that the unfortunate high disease rates found among some asbestos workers from previous decades will never occur again.

A. Commercial Uses of Asbestos.

The term "asbestos" refers to a group of naturally-occurring fibrous mineral silicates that possess a crystalline structure. Asbestos is found in seams, generally between 1 and 20 millimeters in width in igneous or metamorphic rocks. Asbestos has been mined extensively in Canada (which presently produces 25% of the world's supply), as well as in Zimbabwe, South Africa, Australia, Italy, Brazil, Columbia, the Soviet Union, Finland, and the United States. Reports of man's earliest use of asbestos date back thousands of years to Stone Age pottery. Modern large-scale mining and production began in the late 19th century.

Numerous definitions of "asbestos" have appeared in the literature over the years reflecting difficulties of mineralogical classification. Although the disputes about nomenclature have significance in some contexts, for the purposes of this document, the relevant forms of asbestos are:

The Serpentine Group:	Chrysotile, or white asbestos
The Amphibole Group:	Crocidolite, or blue asbestos
	Amosite, or brown asbestos

Although there are other asbestos minerals, only these three have ever been used substantially in commercial applications. Approximately 95% of the asbestos used in the U.S. and Canada today is chrysotile; crocidolite is used primarily in manufacture of asbestos-cement pipe; amosite was at one time used, mainly for insulation materials, but is no longer

employed to any significant extent in North America. The table on the next page details some basic characteristics of each fiber type.

VARIETIES OF ASBESTOS: PROPERTIES, SOURCES, AND USAGE[1]

Mineral Type	Serpentine	Amphibole	
Chemistry, approximate	$Mg_3Si_2O_5(OH)_4$	$X_{2-3}Y_5(Si,Al)_8O_{22}(OH)_2$ with X, Y representing different elements	
Fiber type	Chrysotile (White)	Crocidolite (Blue)	Amosite (Brown)
Main elements determining specific composition	Mg	Na, Fe^{2+}, Fe^{3+}	Fe^{2+}, Mg
Physical properties			
Tensile strength, 1,000 psi	350-450	500	175-350
Flexibility	Very good	Good	Poor
Acid resistance	Poor	Good	Good
Texture	Silky to harsh	Harsh	Coarse
Heat resistance	500°C	200°C	200°C
Major sources, present and past	Canada (Quebec, B.C., Yukon, Newfoundland, Ontario) Russia (Urals, Siberia), Zimbabwe, Botswana Swaziland, Australia (NSW), Cyprus, Italy, United States (Vt., Ariz., Calif.)	S. Africa (N.W. Cape, Transvaal), Bolivia, W. Australia	S. Africa (Tvl)
World use, approximate %	95	3.5	2 %
Major commercial uses	Cement products, flooring, friction materials, paper products, textiles	A/C pipe	None

1/ Adapted from E.C.A. Exhibit 2?, Tab 7, Becklake, M. R., "State of the Art: Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," 114 Am. Rev. of Resp. Dis. 187-227, at 191 (1976).

The physical properties of asbestos that make it commercially valuable include its fibrous nature, heat stability, thermal and electrical resistance, flexibility, high tensile strength, and stability in acids or alkalis. Its name reflects one of its principle characteristics -- "asbestos" is derived from the Greek word for "incombustible."

These attributes make asbestos attractive for a wide variety of applications. For example, in the three uses that account for most current consumption, it serves as reinforcement in asbestos-cement (used for pipe, shingles, and flat and corrugated sheets); provides dimensional stability, flexibility and indentation resistance for flooring; and imparts reinforcement and uniform friction and wear characteristics in brake linings, clutch facings and other friction products. Asbestos also is used to impart fire resistance, rot resistance, and dimensional stability for a large variety of asbestos paper applications; to give high resistance to heat, fire, acid and abrasion in asbestos textiles; and to strengthen plastics.[2]

In response to the increasing cost of asbestos and concern over possible health risks, industry has developed

2/ The U.S. Bureau of Mines estimates that of the approximately 401,000 metric tons of asbestos fiber consumed in the United States in 1981, 40% was used in asbestos-cement pipe, 23% in flooring products, 12% in friction products, 7% in roofing products and the remaining 16%, listed in descending order of usage, in packings and gaskets, coatings and compounds, thermal and electric insulation, asbestos-cement sheet, textiles, plastics, paper and other categories. R. A. Clifton, U.S. Bureau of Mines, "Mineral Facts and Problems, 1980."

substitutes for some applications. Asbestos is no longer used, for example, in sprayed-on insulation or joint cements. But, for many current applications, there is no effective alternative. As a 1978 report to the U.S. Consumer Product Safety Commission noted:1/

> Asbestos-containing products which remain in manufacture are increasingly those in which the attributes of asbestos are difficult to duplicate and for which there are no economically feasible substitute products.

For example, there are substitutes for asbestos in some disc brake pads; but, for drum brakes, no other material has yet been found that provides the processing characteristics and the moderate but stable friction characteristics, resistance to fade, low wear rates, and absence of scoring and squeal that asbestos brake pads exhibit. Similarly, despite considerable research to find an alternative for asbestos fibers in flooring, such as vinyl asbestos tile, no feasible alternatives have yet been developed.2/ And, although alternative materials including polyvinyl chloride and ductile iron exist for water and sewer pipes, asbestos-cement continues to provide an economical and effective competitor favored by many jurisdictions.

1/ Kearney, Management Consultants Report to the CPSC, "Review of Asbestos Use in Consumer Products" IV-2 (1978).

2/ Report to the U.S. Environmental Protection Agency by SCA, "Asbestos Substitute Performance Analysis," at 165 (1981).

Some materials can adequately duplicate the properties of asbestos under limited conditions, but are effectively "unavailable" for that use because they cannot be economically adapted to commercial manufacturing processes. For example, fibrous glass, which is a possible substitute for asbestos in several applications, breaks down in alkaline environments such as cement, and is much more abrasive and thus wears out processing equipment much faster. Several other organic fibers nearly equivalent to asbestos in strength and chemical resistance break down under high temperature and cannot be used in existing manufacturing processes.

Where substitutes are available, they most often cost more, sometimes substantially more, than asbestos. Asbestos fiber was chosen for use in many products not only because of its performance, but also for its low cost and availability. Asbestos products compete with alternatives in many markets and the effect on price competition of removing them from the marketplace cannot be ignored. Where asbestos is at present the only competitive product, removal would tend to create a monopoly for the alternative raw material, with concomitant higher prices.

Many asbestos substitutes themselves may pose health risks that must be considered before their use is promoted. Some substitutes are newly developed, and little scientific information has been generated on their safety. Almost no substitutes have been studied as extensively as asbestos.

Virtually all products now contain asbestos in a form that precludes or renders highly unlikely the release of significant numbers of respirable fibers under normal conditions of installation and use. For example, in the construction industry, which consumes more than three-fourths of asbestos products, felts and flooring contain asbestos bound in a matrix from which it is virtually impossible that a consequential amount of fibers would be released under normal or industry-recommended use conditions. Asbestos-cement products, which comprise the single largest category of asbestos use, bind the fibers into the cement so that a de minimis number are released under industry-recommended installation work practices or in normal end use.

In sum, asbestos is a highly versatile mineral with many very valuable economic applications. As a result of health hazards deriving from very high exposures in the past, industry has developed modern applications that use the asbestos in a form that greatly minimizes human exposure Given these control measures, continued calls for bans or phase-outs cannot be justified.

B. Human Exposures to Asbestos Fibers.

Because asbestos is a naturally-occurring mineral, man has been exposed to its fibers for millions of years. However, it is the very high occupational exposures of the mid-twentieth century that have created medical concerns.

The levels of asbestos exposure experienced by workers today are typically hundreds of times lower than those historical occupational exposures. General population exposures are much, much lower -- even at their highest being thousands or tens of thousands times lower -- than even current occupational exposures.

Review of the existing data on asbestos concentrations past and present is important to understanding both the medical evidence and the related regulatory issues. Only by understanding the contrast between the high exposure levels of the past and the very low exposure levels of the present can reasonable decisions be made about the need for future regulation. At the same time, such a review demonstrates the great progress industry has made in affording safer workplaces for its employees.

1. The Ubiquity of Asbestos in the Natural Environment.

As a naturally occurring mineral, asbestos has been present in the atmosphere for millions of years. Asbestos-containing rock formations have been exposed to air, wind and water leading to release of fibers into the ambient

environment. As Dr. Corbett McDonald noted in his Commission testimony:1/

> [C]hrysotile is very common; it's common in water supplies, it's common in rain, it's common in all sorts of things.
>
> So, I suppose the social decisions which will be made will at least take into account background levels ....

In some instances, particularly in areas of human activity, natural asbestos releases have been found to be quite substantial. For instance, dustfall along roads used for recreational vehicles in California have been found to contain 20 milligrams of asbestos per cubic meter; airborne samples on motorcyclists riding through the region have registered as high as 9.3 fibers per cubic centimeter.2/

1/ C. McDonald Tr., Vol. XII at 114-115.

Commission witness Dr. William Nicholson has also noted:

> Construction excavations and road building activities can be potential sources of asbestos in the environment, if they take place in areas of serpentine rock. Since most serpentine rock contains electron microscopic asbestos fibres, the open blasting associated with road building and excavations could release such fibres into the air.
>
> The natural weathering of asbestos-bearing serpentine rock is another potential source of electron microscopic fibre in both the ambient air and in water. Serpentine is a relatively common type of rock in many areas of the world, where it occurs at or near the surface.

B.C.A. Exhibit 10, Tab 2, Nicholson, W. J. and F. L. Pundsack, "Asbestos in the Environment," in Biological Effects of Asbestos 125-30, at [illegible] (IARC 1973).

2/ Cooper, W. C., et al., "Chrysotile Asbestos in a California Recreational Area," 206 Science 685-88 (Nov. 9, 1979).

It is thus not surprising that asbestos has been detected in the general population's lungs. Drs. Churg and Warnock have reported:1/

> [W]e demonstrated that lungs from a series of subjects who had fewer than 100 asbestos bodies per gram of lung, a value that appears to be associated with environmental rather than occupational exposure, nonetheless contained substantial amounts of asbestos.

Much of the detected asbestos was apparently a non-commercial type, indicating a natural source for the fibers:2/

> The bulk of this asbestos (approximately 80% of fibers) was chrysotile; whereas the rest was largely the non-commercial type of amphibole. On the average, commercial forms accounted for only about 6% of the amphibole fibers.

Commission witness Dr. Margaret R. Becklake has noted this prevalence of asbestos in the general population's

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1/ Churg, A. and M. L. Warnock, "Asbestos Fibers in the General Population," 122(5) Am. Rev. Resp. Dis. 669-78, at 676 (1980).

2/ Churg and Warnock continue (at 677):

> Our findings with respect to the type of amphiboles present in these lungs was surprising. Most industrially used amphibole is amosite and crocidolite. However, in these subjects, commercial amphibole constituted less than 1% of the total fibers present and less than 6% of the amphiboles. The amphiboles that were found were generally encountered industrially or environmentally as contaminants of other minerals, for example, talc. We have suggested that cosmetic talc provides the source for the asbestos bodies formed on anthophyllite and tremolite in women. Another possibility in this group of subjects was that the fibers were derived from local rocks either because of natural weathering, or because of digging operations.

lungs and the great likelihood that such exposure is not related to disease:[2/]

> The surprisingly high prevalence of asbestos bodies (as evidence of exposure) in routine autopsies indicates an environmental exposure for the residents of most of the larger cities of the world; however, the amounts in the general atmosphere are small, and these autopsy findings should probably be regarded more as an index of exposure rather than of disease potential. The Advisory Committee report that followed the Lyon Conference concluded that "there is at present no evidence of lung damage by asbestos to the general public," and "the amount of asbestos in the lungs of members of the general public is very small compared to those occupationally exposed."

Asbestos is not unique as a naturally-occurring substance to which man has been exposed for millions of years, but which, at very high doses, can cause detrimental health effects. Lead found in many consumer products and aflatoxin found in many grains and most peanuts are but two of the

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2/ B.C.A. Exhibit 29, Tab 7, Wechsler, H. B., "State of the Art: Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," 114 Am. Rev. Resp. Dis. 187-227, at 193 (1976).

Dr Wechsler has similarly noted in a more recent review article:

> [S]ufficient amounts are inhaled and deposited in the lungs of urban dwellers to result in the almost universal demonstration of asbestos bodies in their lungs at autopsy, a marker of exposure to, and more important, retention of inhaled fibers.

B.C.A. Exhibit 29, Tab 9, Wechsler, H. B., "Environmental Exposure to Asbestos: A Factor in the Rising Rate of Cancer in the Industrialized World?" 76 Chest 345-47, at 345 (1979).

many substances that have been associated with cancer at high doses, to which people are exposed daily throughout the world. As with those substances, asbestos regulation should proceed within a reasonable context that clearly distinguishes between low background levels that are insignificant to human disease potential and high exposures that can and should reasonably be controlled.

2. Heavy Asbestos Exposures in the Workplace in the Past.

Human exposure to asbestos at levels demonstrated by the medical evidence to have adverse health effects is predominantly a historical phenomenon. Appreciation of the vast discrepancy between the exposures that led to disease and current exposures is important to understanding the implications of the historical medical evidence for current concerns about continued asbestos uses.

Determination of past levels of human exposure to asbestos is subject to many uncertainties. Historical data are sparse, and what data do exist are based on measurement techniques that have been altered and refined over the years. Translation of historical results to current measurement scales is often difficult. Even current measurement techniques are limited in their detection abilities and subject to uncertainties, especially at the low levels existent today. Despite these uncertainties, it is apparent that the occupational exposures that caused disease were

several orders of magnitude (hundreds or thousands of times) higher than the exposure levels experienced by workers today and many orders of magnitude higher than levels experienced by the general population.

Much of the medical evidence of the ill effects of asbestos exposure is derived from epidemiology studies of workers first exposed in the 1920's, 1930's, and 1940's. Regular dust monitoring did not begin in many plants until the 1950's, and even 1960's. Some indication of the dustiness of earlier asbestos operations is, however, provided by workers' verbal descriptions.10/

> Before 1930, conditions in the mines, mills, and factories were largely uncontrolled so that some very heavy exposures occurred. Eyewitnesses speak of being unable to see more than a few feet, for instance, because of the amount of dust in the air.

10/ Selikoff, I. J. and D. H. K. Lee, Asbestos and Disease 170 (1978).

Another observer has more graphically described 1920's era conditions:

> Fibre was blended on the floor by hand and most machines were fed manually. Looms were only partially ventilated and a worker who remembers those days has said that asbestos fibre was literally knee-deep under them. The dust and stout fibre that dropped on to the floor under the carding machines had to be cleared by hand; this, and the stripping (or cleaning) of the cards, also by hand, was a very dusty and dirty operation and whilst it was being carried out a man could not be recognized at a distance of six or eight feet. Fibre was screened on the open vibratory screens, the short fibres being allowed to fall on the floor while the "rovers" were bagged by hand.

Mills, D. W., from a paper to British Medical Effects of Asbestos, N.Y. Acad. Sci. (1965).

Even after the British government imposed the first governmental asbestos hygiene regulations in 1931, very high exposures continued. Commission witness Dr. William Nicholson has described these conditions:11/

> [T]he 1931 regulations in Great Britain required that no asbestos dust enter the workroom. This led to the installation of engineering controls in a segment of the asbestos industry. However, the regulations were "riddled with loopholes -- exempting certain processes where dust was 'unavoidable,'" and did not apply to such work as insulation application. Moreover, as enforcement activities, especially during World War II, grew lax, the regulations' description as a "pious aspiration" was quite appropriate.

Dr. Corbett McDonald described to the Commission similar historical conditions in Quebec mills:12/

> [Y]ou couldn't see the workers; ... literally couldn't see them. They were in a cloud that you couldn't measure. ... I'm not speaking of throughout, but I'm speaking particularly of the bagging department, where it was sometimes a habit of even getting into the bag and jumping on it, to pack the stuff in.

In evaluating historic disease prevalence, it should also be noted that in these earlier years the normal work week substantially exceeded 40 hours; and, during World War II, in England particularly, ventilation was sometimes non-existent due to blackout regulations.

11/ Nicholson, W. J., "Regulatory Actions and Experiences in Controlling Exposure to Asbestos in the United States," 330 Ann. N.Y. Acad. Sci. 293-303, at 299 (1979).

12/ C. McDonald Tr., Vol. XIII at 139.

The sparse pre-modern monitoring data available confirm the anecdotal descriptions of high asbestos dust concentrations. Peak exposures equivalent to 1000 fibers/cc were recorded for some occupations. Many workplaces had average dust levels of 20 to 50 fibers/cc or higher. These numbers should be compared to the proposed Ontario standard of 1, 0.5 and 0.2 fibers/cc for chrysotile, amosite and crocidolite, respectively (time-weighted over 40 hours), and the present OSHA limits in the United States of 10 fibers/cc peak (over 15 minutes) and 2 fibers/cc average (time-weighted over 8 hours).

Among the earliest published monitoring data are occupational dust levels obtained by Dreessen and his colleagues at four American asbestos textile plants.[13/] These midget impinger counts were expressed not in fibers/cc -- the current measurement method -- but in millions of particles per cubic foot (mppcf). As discussed in Chapter IV below, no exact correlations exist for translation of such results to fibers/cc; but, in general conversions factors of from 1 to 5 fibers/cc per mppcf have been found in a variety of operations.[14/] Average dust concentrations in the factories

13/ Dreessen, W. C., et al., "A Study of Asbestosis in the Asbestos Textile Industry," 241 Pub. Health Bull. (1938).

14/ One mppcf is equivalent to 35.3 particles per cubic centimeter (as there are 28,317 cc's in a cubic foot). Thus, if the ratio between mppcf and fibers/cc is 1:1, one of 35.3 particles would be a countable fiber.

reviewed by Dreessen were as high as 74 mppcf at some work stations. Particle counts in the same range were recorded in asbestos textile plants in Pennsylvania. The average was 44.26 mppcf in preparation and carding processes with individual counts up to 123.3 mppcf.[15/] Such measurements would translate to peaks in the 100 to 600 fibers/cc range and averages in the 50 to 350 fibers/cc range.

Even with the progressive introduction of dust control measures, fiber preparation in many textile plants remained a dusty occupation, as revealed by membrane filter dust samples collected in 1964 and 1965 in nine American plants.[16/] The mean for all plants was 5.6 fibers/cc, while individual plant averages ranged from 1.7 to 12.1 fibers/cc. In another plant, mean dust counts for carding and weaving operations exceeded 14 fibers/cc as recently as 1969, with peak exposures ranging up to 24.7 fibers/cc.[17/]

In mining and milling, significant dust controls were introduced in the early 1950's. Prior to that time, dust levels in some Canadian mills exceeded 100 mppcf and reached

13/ Fulton, W. B., et al., "Asbestosis," Spec. Bull. 37 4 el. Pa. Dept. Labor Ind. (1934-35).

16/ Lynch, J. R. and H. E. Ayer, "Measurement of Dust Exposures in the Asbestos Textile Industry," 27 Am. Ind. Hyg. Assn. J. 431 (1966).

17/ Lewinsohn, H. C., et al., "Dust Control in a Conventional Asbestos Textile Factory," 330 Ann. N.Y. Acad. Sci. 225 (1979).

200 mppcf in some areas.[18/] As recently as 1973, 80 to 90 per cent of the dust samples taken in several Canadian asbestos mills were in excess of 10 fibers/cc.[19/]

The manufacture, installation and removal of asbestos insulation in the past, also caused high exposures, particularly in shipbuilding operations during the war years. Records of dust sampling during insulation spraying in British navy ship construction from 1940 to 1960 show fiber counts of 177 to 322 fibers/cc.[20/] Measurements in 1970 of fiber levels during spraying of asbestos insulation in a building under construction were lower, but still very high -- 30 to over 100 fibers/cc.[21/]

Removal of insulation aboard ships can be exceptionally dusty due to confined spaces and lack of ventilation.

18/ E.C.A. Exhibit 61, Tab 20, Gibbs, G. W. and H. S. J. De Toit, "Environmental Data in Mining," in Biological Effects of Asbestos (Bogovski, P., ed.) (1973).

19/ Quebec Asbestos Mining Assn., "Submission to the Ontario Ministry of Labour on the Proposed Regulation under the Occupational Health and Safety Act, 1978 Relating to the Designation of Asbestos," App. 1 (Nov. 1980).

20/ Harries, P. G., "Asbestos Dust Concentrations in Ship Repairing: A Practical Approach to Improving Asbestos Hygiene in Naval Dockyards," 14 Ann. Occup. Hyg. 241 (1971); Harries, P. G., "Experience with Asbestos Disease and its Control in Great Britain's Naval Dockyards," 11 Env. Res. 261-67 (1976).

21/ Battee, W. E., et al., "Application of Sprayed Inorganic Fiber Containing Asbestos: Occupational Health Hazards," 33 Am. Ind. Hyg. Assn. J. 179 (1972). See also E.C.A. Exhibit 19, Tab 5, Nicholson, G. J., "Asbestos -- The TLV Approach," 371 Ann. N.Y. Acad. Sci. 152 (1976). From data in Fletcher, W. E., et al., "A Health Survey of Pipe Covering Operations in Constructing Naval Vessels," 28 J. Ind. Hyg. Toxicol. 9 (1946).

Extensive personal membrane filter sampling at the Devonport Naval Shipyard in England in 1967 revealed mean concentrations in excess of 350 fibers/cc during stripping of sprayed crocidolite insulation, with some short term breathing zone samples in the range of 1000 to 2000 fibers/cc.[22/] Contamination of large areas of the ship was evident from 30 fibers cc measurements at hatchways two decks above the actual work area.

Exposures at asbestos insulation factories have been lower than in shipyards but still some two orders of magnitude higher than current exposures. Actual measurements by NIOSH as late as 1972 at an insulation manufacturing plant yielded mean dust concentrations of 14 to 74 fibers/cc with individual samples ranging up to 208 fibers/cc.[23/]

These historical measurements place in perspective the health consequences of past asbestos exposures, especially when contrasted with the dramatically lower exposure levels that have been achieved in recent years.

3. Lowered Asbestos Exposures in the Workplace Today.

Exposure to asbestos in the workplace is currently at levels dramatically lower than in the past. As medical

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22/ Harries (1971), supra note 20, at 243.

23/ NIOSH, "Criteria for a Recommended Standard: Occupational Exposure to Asbestos," at Tables IX, X, DHEW (NIOSH) Pub. No. 72-10267 (1972).

evidence has illuminated the risks of high exposures, numerous steps have been taken to reduce workplace emissions; methods have been developed to produce asbestos-containing products in which the fiber is locked-in or encapsulated to minimize fiber release; and manufacture of some products has been terminated.

The British Advisory Committee Report on Asbestos (the "Simpson Report") addressed a wide variety of British workplace exposures, including those in factories producing asbestos-cement products, millboard and paper, friction materials, textiles, and insulation. Reported exposures for each occupation averaged, at most, .08 fiber/cc. In addition, with the exception of insulation manufacture, at least 95% of worker exposures were less than 2 fibers/cc.[24/]

Weston Environmental Consultants conducted a survey in the mid-1970's of U.S. occupational asbestos exposures and found typical exposures of 2 fibers/cc or less with the exception of the primary textile industry, where the typical exposure reported for most operations was 4 fibers/cc.[25/]

24/ Final Report of the Advisory Committee, Asbestos (hereinafter cited as "Simpson Report"), Vol. I at 17 (1979). Asbestos exposure from insulation ranged from 0.01 to 13.66 *fibers/cc*, and 88% of the *emissions* were below 2 fibers/cc.

25/ Joly, A. R., et al. (Weston), "Technological Feasibility and Economic Impact of OSHA Proposed Revision to the Asbestos Standard (Construction Excluded)," prepared for the Asbestos Information Association/North America, Tables 4-1 to 4-9 (March 1976).

(Footnote 25 continued on next page.)

More detailed data on the progress industry has made in the past decade to control manufacturing exposures can be found in the Johns-Manville submission to the Royal Commission.[26/] For each of the five plants for which data are presented, the same pattern of steadily lower exposures emerges. For example, at the Long Beach, California, *asbestos-cement pipe* plant, measured *fiber* levels dropped from the 1 to 10 fibers/cc range in 1970 to the 0 to 1 fiber/cc range in 1979.

Similar substantial progress toward reducing exposures has occurred in the Quebec chrysotile mines in the past decade. As the data submitted to the Royal Commission by QAMA show,[27/] fiber levels in most areas were in the 2 to 5 and 5 to 10, and even more than 10, *fibers/cc range in 1970*, but have been consistently controlled, to a point where by 1979 more than 90% of measurements were below 2 fibers/cc. On average, exposures have dropped nearly an order of magnitude during the decade.

(Footnote 25 continued from previous page.)

Progress made in the 1970's can be seen through *comparison* with measurement data demonstrating average exposure levels in the late 1960's in many plants in the industry above 5 fibers/cc and in some above 10 and even 20 fibers/cc for some operations as collected by the U.S. National Institute of Occupational Safety and Health for its 1972 criteria document. NIOSH (1972), supra note 23, at Tables I - XXVI.

26/ Brief to the Commission of the Johns-Manville Corp., Appendix A (Jan. 14, 1981).

27/ Submission of the Quebec Asbestos Mining Association (1980), supra note 19, at Appendix I.

In sum, occupational exposures today are typically at least one, and more often two, orders of magnitude lower than the exposures that led to substantial disease in the past.10/ Industry has made immense strides to control fiber emissions.11/

4. Very Low General Population Exposures.

Moving from occupational settings to asbestos exposures experienced by the general population, measurement techniques must be changed because the concentrations are orders of magnitude lower.12/ Much of the environmental monitoring measures asbestos, not in terms of number of fibers, but in terms of their mass, and not in terms of cubic centimeters, but of cubic meters. Difficulties arise in making direct

10/ Further discussion of exposure levels prevalent in installation of asbestos-containing products (e.g., asbestos-cement pipe, brake repair) is included in the discussion of work practice methods for control of asbestos emissions, Chapter VIII, infra.

11/ As Dr. Nicholson has noted:

> The promulgation of the 5 f/ml time-weighted average standard stimulated some of industry to install extensive dust-control equipment. The scheduled reduction in the time-weighted average to 2 f/ml in July of 1976 has stimulated the development of new methods of engineering control and of new asbestos-free materials. Some parts of the industry have responded commendably.

D.C.A. Exhibit 19, Tab 3, Nicholson (1977), supra note 21, at 167 (emphasis supplied).

12. A detailed discussion of environmental monitoring is included in Chapter VI, infra.

comparisons between ambient mass concentrations and occupational fiber concentrations, as no direct correlation exists between the two measurements. Nonetheless, the vast discrepancies in exposure levels can be seen through rough approximations of both types of exposure. As Dr. Henry Anderson noted for the Commission:13/

> [I]n the general community the amount of fibers present are well ... you know, multiple factors below what [existed] ... especially in the past when the mesotheliomas being seen today occurred.

Although each of the figures in the table below is subject to some measurement uncertainty, the data do highlight the enormous differences between occupational and general population exposures:

13/ H. Anderson Tr., Vol. XVI at 24.

ASBESTOS AIRBORNE
CONCENTRATIONS IN VARIOUS ENVIRONMENTS

	Concentration in Nanograms/m³	
Ambient Concentrations		
	Average	**Range**
U.S. Urban Areas:[22/]		
Twenty Cities	18	0.02-200
Manhattan, N.Y.	30	8-65
Pittsburgh, PA	4	2-8
Frankfort, KY	0.09	0.02-0.15
British Urban Areas[23/]		1-100
British Rural Areas[23/]		.1-1
Workplace Concentrations		
The current workplace[23/]		
2 fiber/cc TWA standard	100,000	
10 fiber peak standard	500,000	
Pre-regulation workplaces[24/]	750,000-1,500,000	Up to 25 million

22/ U.S. DHEW, "Asbestos: An Information Resource" Pub. No. 78-1681, at E-1 (citing five published studies) (May 1978).

23/ Asbestosis Research Council, in British Advisory Council Report on Asbestos, Appendix at 14-15 (1979), assuming a conversion factor of 1 fiber/cc = 50,000 ng/m³. See also N.C.A. Exhibit 28, Tab 5, Final Report of the Advisory Committee, Asbestos (the Simpson Report), Vol. II at 66, Tables 5, 6 (1979).

24/ Based on estimates of 15-30 fibers/cc, studies showing peak exposures up to 500 fibers/cc, and the conversion factor explained above.

Various measurements have been made of asbestos emissions from consumer use of, or exposure to, asbestos-containing products. Data exist, for example, on asbestos emissions from automobile brake use, from hair dryers, and in buildings where asbestos insulation was installed. In each case, the data demonstrate exceedingly small exposures.

During the use of drum brakes, disc pads or clutch facings, small quantities of the surface of the friction material are worn away. Although these materials often contain between 30% and 60% asbestos, their wear products are not emitted as respirable asbestos dust. Examination of the residue of wear products in brake drums by the U.S. Public Health Service has confirmed that the asbestos content is usually less than 1%.[25/] An extensive, well-controlled study under actual driving conditions, conducted by the Bendix Corporation for the U.S. Environmental Protection Agency similarly found that less than .01% of the asbestos content of all the wear products over the whole range of U.S. motor vehicles contributes to ambient concentrations by

25/ Lynch, J. R., "Brake Lining Decomposition Products," 18 J. Air Pol. Cont. Assn. 824-26 (1968).

The heat generated in the process of applying the brakes or the clutch (the reason in fact why asbestos is such a vital component) is sufficient to destroy the original structure of the asbestos fiber; the chrysotile structure begins to convert to a non-crystalline amorphous phase. Anderson, et al. (Ford Motor Co.), "Asbestos Emissions from Brake Dynamometer Tests," at 3, Automobile Engineering Meeting, Detroit, MI May 14-18, 1973).

becoming airborne.[16/] A Ford Motor Company study based on dynamometer tests similarly showed that during brake usage less than 0.02% of the lining wear was released in the form of free asbestos. The study estimated that concentrations of asbestos fiber in the urban atmosphere in the U.S. due to brake usage was therefore less than 0.07 nanograms/m³, representing a small fraction of typical ambient levels.[17/]

The potential for asbestos emissions from hair dryers has also been thoroughly investigated. Reports by the National Institute of Occupational Safety and Health and the National Bureau of Standards in the United States and the Canadian Department of National Health and Welfare found only very low levels of asbestos emitted during use of the hair dryers. Most emissions were well within normal ambient levels and similar to those obtained with hair dryers not containing asbestos.[18/]

16/ Jacko, M. G., et al. (Beadix Corp. and EPA), "Brake and Clutch Emission Generated during Vehicle Operation," Automobile Engineering Meeting, Detroit, MI (May 14-18, 1973).

17/ Anderson (1973), supra note 33, at 3-4.

18/ Final Report for Interagency Agreement NIOSH IA-78-29 with CPSC, "Testing of Hair Dryers for Asbestos Emissions" (Sept. 1978) (asbestos emissions ranged from 0 ng/m³ to 1412 ng/m³, with approximately 60% of the emissions being less than 3 ng/m³); "Examination of Hair Dryers from CPSC (87166 and 87167) for Asbestos," memorandum to the CPSC from U.S. Dept. of Commerce, National Bureau of Standards (May 1979). In the NIOSH study two of the thirty pieces of equipment tested emitted fibers in excess of the ambient range, but neither of these were hand-held models (indeed, one was not even a hair dryer); and the fiber counts included large fibers outside the respirable range.

(Footnote 18 continued on next page.)

Although some asbestos insulation materials are friable and have the potential to break-up and release fibers, measurements of asbestos concentrations in buildings have revealed that only a small number of fibers are in fact released during normal use. The Simpson Report, for example, notes the low levels reported in a 1969 study which found that most asbestos dust levels in public buildings were generally the same whether or not the building had been constructed with or without asbestos products.[19/]

The British Committee, nonetheless, called for further monitoring to assure that exposure levels were in fact as low as had been indicated by earlier studies. The new measurements, recently published, confirm the earlier results. Even in buildings, including several schools, where asbestos had been dislodged or otherwise damaged, no measurements recorded concentrations above 10 ng/m³, and in the three

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(Footnote 18 continued from previous page.)

Canadian Dept. of National Health and Welfare, "Preliminary Study on the Possible Release of Asbestos Fibres During the Operation of Hand-Held Hair Dryers" (May 1979) (for all fibers, asbestos emissions ranged from 0 to 0.40 f/cc, averaging 0.09 f/cc; for fibers >5 μm, emissions ranged from .03 to .05 f/cc, averaging .04 f/cc). Emissions from hairdryers not containing asbestos were in the same range. Similar results were obtained in subsequent testing of three aged hair dryers. Memo to Dr. G. Hocking from J. C. Meranger, A/Chief, Monitoring and Criteria Division, Canadian Department of National Health and Welfare, "Possible Release from Aged Hairdryers" (July 17, 1979).

19/ Byrne, J. C., et al., "A Dust Survey Carried Out in Buildings Incorporating Asbestos-Based Materials in their Construction," 12 Ann. Occup. Hyg. 141-45 (1969), cited in the Simpson Report (1979), supra note 25, Vol. I at 39-40.

buildings where measurment techniques were sensitive enough to record levels as low as 1 ng/m³, no measurements that high were made. The authors thus concluded:[40]

> Although the sampling locations were selected to include buildings where airborne asbestos might have been expected because of damaged or previously disturbed asbestos components, etc., all of the samples showed low levels of asbestos contamination. In light of these results no further sampling in buildings will be undertaken unless there are special circumstances giving cause for concern ... .

In an extensive survey of buildings where asbestos-containing spray insulation materials had been applied, Dr. Nicholson also found few cases of higher than ambient concentrations:[41]

> For the large majority of the samples, there was no significant difference between the average concentration of asbestos measured within the buildings and that measured outside at the same site ... . In all circumstances where significant air contamination has arisen, however, directly v sibly damage to the spray material was evident ... . No evidence was developed in this study for asbestos erosion from cementitious spray fireproofing material used in the plenums of building supply systems.

40/ Le Guen, J. M. and G. Burdett, "Asbestos Concentrations in Public Buildings -- A Preliminary Report," 26 Am. Occup. Hyg. 185-89, 189 (1981).

41/ B.C.A. Exhibit 19, Tab 6, Nicholson, W. J., et al., "Asbestos Contamination of the Air in Public Buildings," prepared for the Environmental Protection Agency, at 23, 40 (1975).

Dr. Nicholson has found the same absence of other than ambient level concentrations -- except where the materials have been disturbed -- in his school surveys:[42]

> The majority of the data on asbestos concentrations in schools were obtained in circumstances in which damage had occurred to friable, noncementitious asbestos-containing sprayed material with consequent dislodgement of asbestos fibers ... . Where visible damage or erosion is not evident, asbestos air concentrations are likely to be little different from background ... .

Measurements in public buildings in Ontario have similarly failed to find any significant number of asbestos fibers. As the Ontario Research Foundation reported at the Commission's Second Public Meeting:[43]

> The general experience has been that the total fibres "are consistent with something we would find outside on the street. Air sampling has not generally given us very much of a result." The results have always been low, sometimes bordering on the lower sensitivity of the techniques used. "We usually find some fibres, but then you'll find some fibres wherever you take an air sample."

In sum, the general population is not exposed to significant concentrations of asbestos fibers. Indeed, as the evidence on friction product wear, hair dryers, and buildings described above indicates, it is the very rare environmental

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42/ B.C.A. Exhibit 19, Tab 7, Nicholson, W. J., et al., "Asbestos Contamination in United States Schools from Use of Asbestos Surfacing Materials," 330 Annals N.Y. Acad. Sci. 587-96, at 59--95 (1979).

43/ Minutes of Second Public Meeting, at 7 (Dec. 12, 1980).

setting where use of asbestos-containing products has increased exposures at all over background levels. Although there may exist unusual settings with detectable asbestos concentrations above ambient levels, those few situations need to be considered on a case-by-case basis rather than in the context of any regulatory scheme for general environmental controls.

CHAPTER II

II. THE PHYSIOLOGY OF ASBESTOS-RELATED DISEASE.

Primarily through epidemiologic studies, heavy asbestos exposures have been found to be associated with asbestosis, lung cancer and mesothelioma. Much more equivocal findings have been made of associations between asbestos and other cancers, including gastro-intestinal and laryngeal cancer.

Asbestosis is chronic lung disease characterized by diffuse interstitial fibrosis of the lung. Early indications of asbestosis may include crepitations (crackles within the chest audible through a stethoscope sometimes called rhles), radiographic signs in the lung and pleura, and impairment of lung function as measured in physiological tests.[1/] Pulmonary hypertension is often associated with advanced asbestosis; resultant heart failure can be the cause of death. Deaths due to asbestosis have been reported among heavily exposed occupational cohorts. Asbestosis-related deaths have ranged from 1.3% of observed deaths among Canadian miners to 11% of observed deaths among one U.S. asbestos manufacturing cohort.[2/]

A association between asbestos exposure and lung cancer was first strongly suggested by Sir Richard Doll in his 1955 study of the Rochdale, England, textile asbestos factory.[3/] Subsequent studies of other occupational cohorts heavily exposed to asbestos have confirmed an association with observed vs. expected ratios ranging from 1.2 to 7.3.[4/]

Special attention has been focused on the relationship between asbestos and pleural and peritoneal mesotheliomas, rare cancers originating in the surface linings of the chest or abdominal cavities. Since the initial report in 1960 of mesotheliomas in a crocidolite mining area of South Africa,[5/] the majority of reported cases have been associated with occupational asbestos exposure. Other reported cases have had no asbestos exposures at all.

Before proceeding to examine in detail the epidemiologic studies from which such associations have emerged, it

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1/ In addition to interstitial lung fibrosis, asbestos exposure can also cause scarring of the lining of the lungs, or pleural surfaces. Such pleural plaques or thickening, although an indication of asbestos exposure, are in general not associated with clinical or functional abnormalities. See H. Weill Tr., Vol. IX at 13-14; H. Anderson Tr., Vol. XVI at 14-15; N.C.A. Exhibit 29, Tab 7, Hochiahe, M., "Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," 114 Am. Rev. Resp. Dis. 187-227, at 202 (1976).

2/ See N.C.A. Exhibit 28, Tab 3, Final Report of the Advisory Committee, Asbestos (hereinafter cited as "Simpson Report") Vol. II, Table II at 68 (1979).

3/ Doll, R., "Mortality from Lung Cancer in Asbestos Workers," 12 Br. J. Industr. Med. 81-86 (1955). In his introductory comments, Professor Doll notes (at 81) the substantial conflicting evidence previously collected on the asbestos/lung cancer association.

4/ N.C.A. Exhibit 28, Tab 3, Simpson Report (1979), supra note 2, Vol. II, Table II at 68.

5/ Wagner, J. C., et al., "Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province," 17 Br. J. Industr. Med. 260-71 (1960).

is useful to review the physiology and biology of diseases associated with asbestos exposure. The following summary draws from the Commission's record the evidence presented on:

(1) The physical dimension of asbestos fibers and the effect of differing shapes and sizes on the respirability and deposition of fibers.

(2) The clearance and other defense mechanisms of the body that work against the development of disease from asbestos.

(3) The mechanisms that may account for fibrogenicity and cancer.

(4) The physical and chemical differences among fibers and the possible relationship of such differences to disease-causing potential.

From this evidence, it can be seen that although many unresolved questions remain, considerable data exist to indicate that variations exist among the disease causing potential of various individual asbestos fibers and that there will exist a level of asbestos exposure below which no appreciable disease will be caused.

A. Fiber Physics, Respirability and Deposition.

Asbestos fibers vary significantly in length and diameter. Lengths range from less than 1 micron (µm)[6/] to

6/ A micron, or micrometer, is a millionth of a meter.

more than 200 microns; diameters from less than .1 µm to more than 5 µm. Shapes also vary, with the amphiboles typically straight and needle-like, and chrysotile tending toward curliness. Any given asbestos aerosol may contain a collection of asbestos fibers covering these ranges. These shape and size configurations are important to determination of the ability of fibers:

First, to remain suspended and thus be subject to respiration;

Second, to be breathed and thus begin their passage into the lungs;

Third, to settle out or be implanted at various points in the human respiratory tract; and

Fourth, to be subject to various clearance mechanisms that remove fibers from the lungs.

Finer fibers which have not been mixed or coated with other substances are most likely to remain suspended in the air and thus be inhaled. As the Simpson Report notes:[7/]

> As the likelihood of a fibre to remain airborne or to settle is determined by its diameter, and the number of fibres per unit mass will be greater in the case of finer fibres, it is clear that fiber diameter is the key to the number of fibres inhaled.

7/ B.C.A. Exhibit 18. Tab 5, Simpson Report (1978), supra note 1, Vol. II at 9, 19.

Once inhaled, the fiber faces, as P. Kotin termed it during his Commission testimony, "a very tortuous path"[8] into the lungs:[9]

> [F]ibrous particles enter the lungs via the trachea following inhalation through the nose or mouth and are distributed throughout the tracheobronchial tree, ultimately reaching the alveoli or air sacs.

Dr. V. Timbrell of the British pneumoconiosis unit has most extensively studied fiber deposition in the lungs and bronchial passages.[10] Although it is difficult to define diameter and length of many fibers because of aggregation, particularly of chrysotile, his studies have shown that deposition depends on fiber diameter, fiber length and fiber shape.

Fiber diameter is the basic determinant of the extent to which fibers will flow through the lung's passages. The thicker the fiber, the more likely that it will be filtered out at a higher point in the lungs, with the thickest fibers never getting beyond the filtering mechanisms of the nose.

8/ P. Kotin Tr., Vol. XXIA at 7-8.

9/ B.C.A. Exhibit 30, Tab 3, Kotin, P., "Pathophysiology in Relation to the Chemical and Physical Properties of Fibers," in WHO Pub. No. 506, Proceedings of the Workshop on Asbestos: Definitions and Measurement Methods 133-42, at 134 (1977).

10/ See Timbrell, V. and J. W. Skidmore, "The Effect of Shape on Particle Penetration and Retention in Animal Lungs," in Walton, W. H. (ed.) Inhaled Particles III, Vol. II, at 49 (1971); Timbrell, V., "The Inhalation of Fibrous Dusts," 132 Ann. N.Y. Acad. Sci. 255 (1965); and Timbrell, V., "The Inhalation of Fibres," in Shapiro, H. A. (ed.) Pneumoconiosis: Proceedings of the International Conference Johannesburg at 3 (1969). See also Wagner, J. C., and J. W. Skidmore, "Asbestos Dust Deposition and Retention in Rats," 132 Ann. N.Y. Acad. Sci. 77-86 (1965).

Fiber shape is important because it affects the orientation which the fibers assume within the airways: the straighter, more needle-like amphiboles tend to align themselves with the axis of the airway, and penetrate deeper into the lung, while "curly" chrysotile fibers do not. As Dr. Gibbs noted:[11]

> [T]he curliness of the fiber led to a greater cross-sectional area as the fiber entered, and hence interception at branches and in nasal passages would be much greater. Therefore, chrysotile fibers would not get down deep into the lungs.

Particularly long fibers are also unlikely to get deep into the lungs. The extreme length of these fibers results in increased bronchial deposition by interception, a mechanism whose effect can usually be neglected for particles of more compact shapes. As Commission witness Dr. Margaret Becklake has described the process:[12]

> Inhaled asbestos particles follow the moving airstreams with each inspiration, and, once they make contact with any part of the surface of the airways or airspaces, are not resuspended in the expiratory airstreams.

Dr. Gibbs described such interception:[13]

11/ G. Gibbs Tr., Vol. XXIX at 97.

12/ B.C.A. Exhibit 29, Tab 7, Becklake (1976), supra note 1, at 193.

13/ G. Gibbs Tr., Vol. XXIX at 96.

> [A]s fibers try to pass through airways which are fairly narrow, a fiber which is fairly long and gets out of orientation so it's angled to the wall will be trapped. Also, if there is a change in direction of air flow, as there would be at a branching, there could be momentum of the fiber which would carry the fiber on to the branch.

Although cautioning that "all these dimensions are very close approximates," Dr. Kotin explained the effects determined by Timbrell's work:[14/]

> Fibers greater than 5 μm in diameter are virtually entirely lodged in the nose and do not penetrate the respiratory tract. Fibers greater than 3 μm and less than 5 μm in diameter enter the trachea but do not reach the conducting airways deeply enough to be retained in the lung. Fibers less than 3 μm and more than one micron can penetrate to the smaller branchi. Fibers in the millimicron range in diameter are deposited in the peripheral airways and air spaces through Brownian movement.

Studies of fiber physics both in the air and in the body have thus shown that it is the thinnest fibers that are most likely to penetrate to the lung's air sacs, or alveoli. Fibers with diameters greater than one micron are least

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14/ R.C.A. Exhibit 30, Tab 3, Kotin (1977), supra note 9, at 18.

Dr. Gibbs agreed:

> [Timbrell] was able to show ... that for asbestos fibers, because settling rate depends on density and diameter, for asbestos fibers, fibers greater than around three to three and a half microns would not penetrate directly into the alveolar region.

G. Gibbs Tr., Vol. XXIX at 97.

likely to be suspended in the air and most likely to be filtered out during the tortuous path into the lungs.

B. Clearance and Defense Mechanisms.

The human body has many mechanisms for dealing with undesirable foreign bodies. Two such mechanisms are particularly important to understanding the fate of asbestos fibers. First, the mucociliary system clears many fibers very quickly out of the lungs before they ever reach its outmost parts and have any opportunity to cause harmful effects. Second, the cell's immunity system includes macrophages that are instrumental in detoxifying those fibers that reach the outer portions of the lung.

Dr. Kotin described graphically for the Commission the mucous and ciliated cells of the mucociliary system:[15/]

> One is a cell that has the capability of secreting mucous, synthesizing mucous within the cell, and then excreting the mucous into the lumenal surface of the cell. This mucous is constantly being secreted, it's a mucous that is rich in mucous materials that allow it to diffuse over the lining of the tracheobronchial tree and form a blanket, as it were, a mechanical barrier. This mechanical barrier does not have a static residence, but is propelled by the second of the two types of cells that I was referring to, and these cells are referred to as ciliated cells.
>
> These are cells that have little brush borders. They have little whiplike.

15/ P. Kotin Tr., Vol. XXIA at 19.

> hairlike bra[i]ds which extend from the lumenal surface of the cell into the bore of the tube and they beat constantly, and they beat at a very, very rapid rate. One can visualize the beating with stroboscopic light where you can actually see the cilia beat, and it is a beat that is directed towards the throat. It is the motive force for this *mucous* blanket which we can now refer to as a mucous escalator, and the analogy to an escalator is a very, very precise one.

This escalator acts throughout much of the lung to remove fibers. As Dr. Kotin indicated, it is only in the small, segmental bronchi, that there is a waning and disappearance of the mucociliary apparatus.[16/] Many fibers are thus carried back up out of the lung quite rapidly. Dr. Becklake has described the great efficacy of this clearance system:[17/]

> Clearanc of particles deposited on the mucous blanket is brisk, with half-times of minutes to hours. Mucous and cells from nonciliated airways are cleared to the pharynx; the material trapped in the nose is also cleared to the pharynx.

For these fibers that are not carried out of the lung by the mucociliary escalator, a second human defense mechanism -- the macrophage -- operates to prevent injury. As described by Dr. Morgan:[18/]

16/ Id. at 21.

17/ B.C.A. Exhibit 29, Tab 7, Becklake (1976), supra note 1, at 193-94.

18/ B.C.A. Exhibit 31a, Morgan, A., "Fiber Dimensions: Their significance in the Deposition and Clearance of Inhaled Fibrous Dusts" at 3.

> Short asbestos fibers deposited in the alveolar region of the lung are engulfed by pulmonary macrophages: these are phagocytic cells which can move freely over the alveolar epithelium.

Dr. Kotin described the efficacy of macrophages in handling any foreign bodies:[19/]

> [The macrophage] captures it, incarcerates it within the cell, and then depending upon what the material is, it can do everything from digest it if it's an organic material, dissolve it, or just, if it's incapable of destroying it, just keep it isolated from doing any harm by virtue of its being in this cell. ... [T]he cells can make an effort to ingest the fiber and incorporate it within its substance, within its cytoplasm, after it's ingested, reform its cell membrane so it's again an impact cell, and for all intents and purposes this fiber is out of business ...

The macrophage thus provides an important second line of human defense against inhaled fibers. As Dr. J. M. G. Davis told the Commission:[20/]

> Q. So that if I understand it, while the macrophage has totally engulfed an asbestos fiber it's relatively immobile and presumably not ... .
> A. The fiber is relatively immobile.
> Q. And presumably not harmful ... .
> A. I believe that to be the case. It's very difficult to prove. I believe that to be the case.

And, as Dr. Becklake has noted:[21/]

19/ P. Kotin Tr., Vol. XXIA at 22, 24.

20/ J. Davis Tr., Vol. XXXIV at 12.

21/ B.C.A. Exhibit 29, Tab 7, Becklake (1976), supra note 1, at 193.

> Clearance of inhaled particles by these mechanisms is believed to be more than 98 per cent effective for most deposited particles.

Dr. Morgan has demonstrated the great efficacy of the lung's clearance mechanisms for shorter fibers in an experiment where rats were exposed by inhalation to UICC anthophyllite and then sacrificed so that the number of fibers in the lungs could be counted after 101 days and 2 years. As he reported:[22/]

> [T]here was a progressive decline in the frequency of short (< 5μm) fibres and a corresponding increase in the frequency of all longer categories. This shows that fibres less than 5 μm in length are cleared from the rat lung more efficiently than are longer fibres.

Of utmost importance to the understanding of the disease-causing potential of asbestos is recognition of the important role of cigarette smoking in making both clearance mechanisms much less effective. Dr. Kotin described the effect of smoking -- "the most potent and ubiquitous of all inhalants in its capability to neutralize or destroy the effectiveness of the lung defense mechanisms"[23/] -- on both clearances:[24/]

> It's as though in a department store an escalator were to stop, but people were still to continue coming on

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22/ Morgan, A., "Effect of Length on the Clearance of Fibres from the Lung and on Body Formation," in Biological Effects of Mineral Fibres, 329-35, at 330 (IARC 1980 Lyon).

23/ B.C.A. Exhibit 30, Tab 3, Kotin (1977), supra note 9, at 11.

24/ P. Kotin Tr., Vol. XXIA at 60-61.

> and on and on, and so you ultimately would have rather than one layer, piles and piles of people trying to occupy the limited space.
>
> The result of this, of course, is that it provides one of the necessities that we referred to this morning of increased residence time for whatever biological effect you are going to study for.
>
> So in essence, what [smoking] does is neutralizes more effectively than perhaps any known toxic agent that I am aware of the effectiveness of the mucociliary apparatus.

And macrophage response:[25/]

> [C]igarette smoke, no more or no less than asbestos, is a foreign material, so the same macrophages are mobilized to ingest the chemical components of cigarette smoking. ... [S]ince the supply of macrophages is very, very great, but not inexhaustible, it will ultimately deplete ... not completely, but reduce the number of available macrophages, and then the macrophage distended and laden with cigarette smoke pigment is one that is not available for other things ... .

The human lung thus has two very important defense mechanisms that can remove or detoxify inhaled fibers. Both systems have been found to be quite effective under normal conditions. Smoking, however, greatly reduces the efficacy of both the mucociliary escalator and the macrophage system. It is thus not surprising, as will be discussed in Chapter III below, that in many studies asbestos-related disease has been found to occur predominatly among smokers.

25/ Id. at 13-14.

> [W]hat is rather peculiar there . . . and none of us can really understand it . . . and that is that the conversion is of the same order in that plant as in the mines and mills. We would have expected a difference.

Questions about the conversion factor of three have derived in addition from the somewhat greater factors found by Public Health Service investigators in a 1965 study of textile plants, including the one studied by Dr. Dement in Charleston. These investigators found varying conversion factors emerging from the paired sampling they conducted, between plants and between areas in plants; but, the range found was generally to the higher side of three. Indeed, in carding, the range found was from 3.8 to 11.3.[101]

In light of the surprisingly low conversion factor used by Dr. Dement, questions have arisen about the regression analysis statistical technique he used to determine his result. In light of the extreme variability of both the impinger and membrane filter methods, especially at lower measurements as found in the Charleston plant, there is considerable doubt that the proper statistical technique was used.[102]

101/ Ayer, H. E., et al., "A Comparison of Impinger and Membrane Filter Techniques for Evaluating Air Samples in Asbestos Plants," 132 Ann. N.Y. Acad. Sci. 274-87 (1965).

102/ Dr. Dement testified about the extreme variability of both measurement techniques. J. Dement Tr., Vol. XXXIII at 110.

(Footnote 102 continued on next page.)

Individual exposure values were determined by Dr. Dement by dividing all textile operations into 16 exposure zones, with one of four uniform job categories specified within each zone. Exposure levels were assumed to be related linearly over time to fill in the many gaps where actual measurements did not exist.

The resulting exposure measurements have been questioned both because they are so low and because they do not exhibit the pattern of decreasing exposures over the years that would have been expected. For example, the model results compute no changes in exposure levels in mule spinning, universal winding and draper weaving throughout the 45 years from 1930 to 1975, nor in foster winding from 1938 to 1975, nor in twisting from 1930 to 1975, nor in heavy weaving from 1937 to 1975. Indeed, the model computes higher exposures in carding from 1966 to 1975 than for the preceding 19 years.[103] As Dr. Weill noted:[104]

> [Dr. Dement] showed in Cardiff, a slide in his introduction of a carding machine in this plant of a very responsible

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(Footnote 102 continued from previous page.)

In converting particle counts to fiber measurements, the McGill University team, unlike Dr. Dement, did not use a uniform conversion factor determined by statistical techniques. Rather, they found specific conversion factors for different times and mills and locations within mines and mills, as further discussed in Chapter IV.

103/ R.C.A. Exhibit [illegible] Dement (1968), supra note 85, Table 6 at 10 [illegible] 6.

104/ H. Weill Tr., Vol. IX at 1058-106.

> The findings from the last [Dement] were completely out of line with anything that has gone before; so much so that reasonable scientists must await careful evaluation before allowing this paper to influence judgement based on scores of well-authenticated reports.

During his Commission testimony, Geoffery Berry agreed:[97]

> [T]his is a finding which surprised everybody when it came out, and I think, at the moment, it's not quite clear how it fits in with other findings.

Dr. Dement's preliminary report covers only a cohort of 768 white males who had been employed for six or more months in textile production operations with at least one month employment between 1940 and 1965. They were followed through the end of 1975. Thus excluded are both a large number of shorter term employees and the cohort of black employees. Dr. Acheson noted before the Commission the possible difficulties raised by such exclusions:[98]

> The interesting point is that ... there are two thousand, five hundred men in the plant, and Dement deals with seven hundred and forty-eight. I'm always worried about any cohort study which refers to a selected subsample of a cohort as a whole.

Dement *reports* cumulative exposure *for* individual members of his cohort based on 5,952 environmental samples collected between 1930 and 1975. However, the vast majority of these measurements were made after 1960, whereas the

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97/ G. Berry Tr., Vol. X at 46.

98/ D. Acheson Tr., Vol. XIX at 82.

great bulk of the cohort's exposures were prior to that time. Indeed, for the eighteen-year period between 1937 and 1956, only four measurements exist, all made on one day in 1946.[99]

Because all the pre-1965 measurements were made by impinger particle counts, Dr. Dement converts the readings to *fibers/cc* based on a *uniform conversion factor for all* operations, except preparation, of 3 fibers/cc for each 1 mppcf. For preparation, he employs a uniform conversion factor of 8. These conversion factors have been viewed skeptically by a number of experts. As Dr. Corbett McDonald noted:[100]

99/ See Dr. Dement's Dissertation, "Estimation of Dose and Evaluation of Dose-Response in a Retrospective Cohort Mortality Study of Chrysotile Asbestos Textile Workers," Tables 3.1, 3.2 at 79, 81.

Julian Peto noted the importance of segregating this sparsity of data for the *crucial periods*:

> [The Dement study] suffers from the defect that the other studies suffer from, that in fact most of excess mortality is in people who were first exposed in the thirties or forties, when no reliable measurements were taken, when measurements which can only be converted to fibers on the basis of rather speculative conversion were available.
>
> I mean, it starts off by saying that there were five thousand, nine hundred and fifty-two dust measurements taken, but only a hundred and ninety-three of them were taken before 1965, and that's a period in which the exposure which is accounting for a good deal of the excess mortality occurred.

J. Peto Tr., Vol. XXVA at 142.

100/ C. McDonald Tr., Vol. XIII at 10.

employment.[91] Dr. Finkelstein is expanding the study by reviewing the mortality of shorter term workers.

The mortality cohort includes 339 workers, of whom 166 were asbestos production workers for at least one year, 86 were maintenance workers, and 87 had worked in the plant for nine or more years in rock wool/fiberglass operations and were considered controls.[92] Dr. Finkelstein was not able to trace the vital status of 15 of the men (4.6%). Mortality was compared both between the exposed members of the cohort and the Ontario male population and between exposed and unexposed cohort members.

Of 58 deaths among the production workers, 11 have been from mesotheliomas (6 peritoneal, 4 pleural, and 1 mixed), and 19 from lung cancer.[93] In the interval 20 to 33 years after first exposure, mortality from all causes is doubled, deaths from all malignancies are increased 5 times, and lung cancers are 8 times expected.

Although the mortality study is still at a preliminary stage and the number of deaths quite small, Dr. Finkelstein has attempted to correlate these results with his estimated

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91/ Unlike the morbility study, however, those workers whose time in an asbestos area was less than one year are also reviewed and used as a control population.

92/ An additional eleven men could not be well classified and were excluded from the analysis.

93/ Updated results find 10 mesotheliomas and 20 lung cancers. B.C.A. Exhibit 3b, Tab 3.

exposures. Interpretation of the results is complicated both by the absence of a dose-response relationship for lung cancer deaths (when the workers are divided into three equal sized groups with low, medium and high cumulative exposures, the highest risk is found in the middle group) and by the questionableness of the exposure estimates. As Dr. Finkelstein noted during questioning before the Commission:[94]

> Q. Then your final conclusion, at the bottom of page nineteen, is that because of the large uncertainties of exposure it's impossible to assess the risk with any accuracy?
> A. Yes, I think that's true.
> Q. I gather that's why you give the wide range of the figures both with your calculated exposures and what it would be if the exposures were ten times as great as you calculated?
> A. That's right.

7. The Dement Textile Factory Study.

In conflict with the other studies is the work of Dr. John Dement, formerly of the U.S. National Institute for Occupational Safety and Health (NIOSH), in a study of an asbestos textile plant in Charleston, South Carolina.[95] As one reviewer has noted:[96]

94/ M. Finkelstein Tr., Vol. XXX at 17

95/ B.C.A. Exhibit 3, Dement, J. M., et al., "Estimates of Dose-Response for Respiratory Cancer among Chrysotile Asbestos Textile Workers," presented at the Fifth International Symposium on Inhaled Particles, Cardiff, Wales, Sept. 8-12, 1980).

96/ Liddell, D., "Asbestos and Public Health," In Thorax [illegible], at [illegible] (1981).

The cohort includes 201 men, of whom 157 were production workers and 44 were in maintenance. Most of the production workers had spent their entire career at the plant in asbestos areas:

Production Worker
Time in Asbestos Area

15 or more years	60%
11-14 years	7%
6-10 years	7%
5 or fewer years	6%

Dr. Finkelstein determined that 61 (39%) of the production workers and 9 (20%) of the maintenance workers had been certified as having asbestosis.

To determine dose-response relations among his cohort, Dr. Finkelstein analyzed the data on production worker cumulative exposure through their first 10 working years. He then compared his calculated incidence data to that of Geoffery Berry in the 1979 Rochdale study and found the rates in each generally "in agreement".[88]

88/ Exhibit 36, Tab 4, Finkelstein (1981), supra note 77, at 16-17, 19.

Asbestosis Certification

Fiber-Years Exposure

	0-49	50-99	100-149
	Annual Incidence		
Rochdale	.7%	1.6%	3.4%
Toronto	.4%	1%	2%
	Prevalence		
Rochdale		2.5%	8.5%
Toronto		4%	6%

Dr. Finkelstein further found certification related to cumulative exposure in a lognormal fashion and interpreted these results as suggesting an effective asbestos threshold:[89]

> The response at low levels suggests that for all practical purposes there is probably an effective threshold below which clinical asbestosis will be unlikely to occur, although there could be the occasional very susceptible individual who could develop problems at these low levels.

b. The Mortality Study.

Dr. Finkelstein's companion, preliminary mortality study includes all workers in the morbidity study by being made up of a cohort of all persons who began work at the Toronto plant prior to 1960 with more than nine year's

89/ M. Finkelstein Tr., Vol. XXIV at 41.

> impingers around on little carts and tried to get them close to the employees' noses, so they were close to the workers, but they were different from personal sampling data, it's absolutely impossible for me or for anyone else to assess how ... what each worker was actually exposed to and how to take into account, you know, the little tinkering that was done with the exhaust on any particular machine.

Further unaccounted exposures may be due to long work weeks. As Dr. Finkelstein noted:[95/]

> [A]pparently many of these men did work overtime. The plant worked six or seven days a week, some of them came in during their supposed days off to help in maintenance and whatnot, so that the work week was longer and many of these men did work overtime.

Based on his model, Dr. Finkelstein calculated average exposure levels in the plant experienced by the cohort members as:[96/]

1951	11.3 fibers/cc
1956	7.6 fibers/cc
1966	4.3 fibers/cc
1972	2.1 fibers/cc

Dr. Finkelstein noted that these estimates might be off by a factor of three to five, with all the most probable factors indicating the estimates are too low, and not too high:[97/]

95/ M. Finkelstein Tr., Vol. XXIV at 107.

96/ R.C.A. Exhibit 96, Tab 4, Finkelstein (1981), paper note 77, at 13.

97/ M. Finkelstein Tr., Vol. XXIV at 130.

> I say in my paper the uncertainty of three to five is not unreasonable, based upon this data. ... Certainly work practices in the bad old days were not very good. People were, apparently, jumping in bins, dry sweeping, doing all kinds of not very nice things.
>
> I think it would be a reasonable assumption that exposures were high. On the other hand, there may have been particular employees where I have overestimated their exposures.
>
> The intention in my statement of three to five was that this was the range at which I may have underestimated.

Other observers, including Mr. Peto, have also noted the likelihood these exposure estimates are unduly low:[98/]

> I would suspect that ... the early estimates of dust level in this study are too low, because they have produced estimates which are really quite inconsistent with any other estimates that have previously been produced.

a. <u>The Morbidity Study.</u>

Dr. Finkelstein's morbidity cohort included all persons who began work before 1960 and had worked in the plant for at least 15 years, with at least one year of employment in an asbestos area (pipe or board shops). Results are reported in terms of the number of such workers who have been certified by the Ontario Workmen's Compensation Board as disabled due to asbestos.

98/ J. Peto Tr., Vol. XXVB at 26. Dr. Gibbs similarly expressed great skepticism about the low exposure estimates, noting that it was quite unlikely that any cohort would, as did this cohort, experience 29% certified asbestosis with such low exposures. G. Gibbs Tr., Vol. XXXIII at 66-67.

his estimates to the two major ventilation improvements in the plant in 1962 and 1970, by using the 1969-70 fiber measurements as the baseline values upon which all prior estimates were derived. Dr. Finkelstein estimated exposure levels by assuming:

> (1) Relative exposure among work sites remained constant from 1948 to 1970.

Dr. Finkelstein noted this assumption was supported only "weakly" by the "not very accurate and not very precise" data.[79/]

> (2) Exposures from 1962 to 1969-1970 did not change; and
>
> (3) Exposures from 1955 to 1961 were 1.3 times 1969-70 exposures, and from 1948 to 1954 were twice 1969-70 exposures.

Dr. Finkelstein noted that both exposure multipliers were quite inexact:[80/]

> [T]his is just a guess, analogous to the work that was done at Rochdale where they guessed it was twenty-five percent higher during the mid-forties, and fifty percent higher before. There is some indication from the impinger data that thirty percent might be a reasonable number, but it's pulled out of thin air.
>
> . . .

79/ R.C.A. Exhibit 36, Tab 4, Finkelstein (1981), supra note 77, at 10; H. Finkelstein Tr., Vol. XXIV at 43-44.

80/ H. Finkelstein Tr., Vol. XXIV at 51, 52-53.

> A factor of two, again, is arbitrary, in a sense. ... [I]t's based on the universal opinion of people that things were pretty bad back then, certainly worse than in 1969, probably worse than 1961, and so I chose a factor of two.

He thus cautioned that his estimates are "quite crude" and "have considerable uncertainties associated with them,"[81/] not only because of the absence of strong support for these assumptions, but also because he did not make any adjustments for, inter alia:[82/]

- -- higher fiber counts during the winter;
- -- long work weeks in the plant's early history;
- -- peak exposures during breakdowns;
- -- peak exposures during blowdowns;
- -- changes in personal ventilation.

As Dr. Finkelstein noted in his report, breakdowns could have led to very high peak exposures:[83/]

> Work practices in the early years of operation and equipment breakdowns undoubtedly gave rise to large peak exposures rates which are impossible to accurately assess.

And, area sampling may underestimate many exposures:[84/]

> [T]here are two components to ventilation - there's gross ventilation changes and there's local machine-related and job-related hygiene improvements. Given that this is all area sampling, although the company wheeled their

81/ R.C.A. Exhibit 36, Tab 4, Finkelstein (1981), supra note 77, at 9, 10.

82/ See H. Finkelstein Tr., Vol. XXIV at 126-33.

83/ R.C.A. Exhibit 36, Tab 4, Finkelstein (1981), supra note 77, at 10.

84/ Id. at 10.

Mr. Peto has also noted the extreme fragility of the post-1951 Rochdale results:[75]

> [T]he numbers are extremely small. I think we ought to emphasize how small they are ... . [T]he actual estimated excess changes by a factor of three or four purely as a result of fluctuations, leaving everything else aside in that calculation.

Considerable uncertainty therefore surrounds interpretation of the Rochdale study. Doubts exists as to both the validity and the completeness of the exposure estimates, particularly in connection with the mortality study. In addition, the small size of the mortality cohort leads to considerable uncertainty as to its accuracy.[76]

8. The Toronto Asbestos-Cement Plant Studies.

New to the epidemiologic literature on asbestos are the companion morbidity and mortality studies presented to the Commission by Dr. Murray Finkelstein of the Ontario Ministry

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[75] J. Peto Tr., Vol. XXVA at 125.

[76] As Mr. Peto himself told the Commission, "by any normal criteria these are ... rotten data because of the ambiguities of measurement and so on." J. Peto Tr., Vol. XXVA at 105.

Mr. Peto added that he nonetheless considered the Rochdale results the "best data around if you want to relate lung cancer rates to measured dust levels." Id. That conclusion, however, seems to be based primarily on his peculiar views of the difficulties of converting particle measurements to fiber measurements in every study other than the one at Rochdale, a view not shared by other Commission experts, as discussed in detail in Chapter IV, infra.

of Labour.[77] These studies, one of which -- that on mortality -- is still incomplete, follow cohorts of long-term workers at the Johns-Manville asbestos plant outside Toronto.

The plant studied by Dr. Finkelstein was opened in 1948. Until 1955, its primary asbestos-containing product was asbestos-cement pipe; after that time, it also produced asbestos-cement sheet and beginning in 1960 molded calcium silicate asbestos insulation. Non-asbestos operations were also present at the plant.

Both the morbidity and mortality studies attempt to determine dose-response relationships based on the same exposure information, assumptions and estimates. A mere handful of exposure measurements were made during the first twelve years of the plant's history, during which time much of each cohort's exposure occurred.[78] All of these measurements were impinger particle counts, as were the annual surveys done by the company over the following decade. Quarterly fiber count data performed by the company are available from 1969 to the plant's closing in 1981. Given the paucity of exposure measurements, Dr. Finkelstein keyed

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[77] R.C.A. Exhibit 36: Tab 4, Finkelstein, M., "Asbestosis Among Long-Term Employees of an Ontario Asbestos-Cement Factory," unpublished (1981); Tab 5, Finkelstein, M., "Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory," Draft (1981).

[78] As Dr. Finkelstein noted, measurements were made on only six or seven days during these years. M. Finkelstein Tr., Vol. XXIV at 46-47, 136. These surveys were made in 1949 and 1951 by the Government and 1954 and 1957 by insurance company hygienists.

By having divided the cohort into these groups of successively more recent employees, the Rochdale experience has demonstrated declining disease with improving industrial hygiene conditions. For example, the lung cancer experiences of the three subcohorts with more than twenty years exposure, but varying amounts of such exposure prior to 1933, have been declining:[72]

Exposure Period		Lung Cancer Deaths		
Years of Exposure Before 1933	Total Years of Exposure	Observed	Expected	O/E
≥ 10	30	15	1.49	10.1
< 10	30	10	3.05	3.3
0	30	9	1.16	1.6

The latest mortality report follows the 679 men who entered the factory in 1933 or after and who were exposed for at least ten years through the end of 1970. Excess lung cancer deaths are reported for both the subcohort beginning work between 1933 and 1950 (22 observed vs. 13.85 expected 20 or more years after first exposure) and the 1951 to the present group (8 observed vs. 1.62 expected). Excess asbestosis and mesotheliома deaths are found only in the former subcohort, but the authors caution this may be due to relatively short follow-up. Excess deaths were not found for any other cause, including gastrointestinal cancer.

72/ R.C.A. Exhibit 37, Tab 1, Peto (1977), supra note 62, at 170.

Although increased lung cancer is found even in the most recently exposed subcohort (post-1951 initial exposure), it is quite possible that the excess is attributable to a heavily exposed subgroup. Mr. Peto reported that of the lung cancers in the post-1951 group all were male smokers and "five worked in areas where dust levels were high in 1951, and one may have been exposed to asbestos dust from 1925 to 1930 in a previous occupation."[73] Mr. Peto further testified about the unlikelihood that the apparent increase in the lung cancer relative risk between the pre- and post-1951 subcohorts is meaningful:[74]

> I am inclined to think that our results were inflated by chance. As I said in the paper [Peto (1980)], I thought that this wasn't in fact a sensible estimate, and it was more reasonable to assume that the true relative risk was of the order of two or three.

73/ R.C.A. Exhibit 37, Tab 1, Peto (1977), supra note 62, at 171.

74/ J. Peto Tr., Vol. XXVB at 17, XXVA at 184-185. Mr. Peto had previously written:

> As the majority of pre-1951 employees in this study were still employed in 1951, it seems likely that this apparently marked increase in risk for lung cancer among men with estimated cumulative exposure of about 200-300 fibres/ml-years (the order of magnitude of the average exposures of men first employed in 1951 or later (Table 2)) is therefore probably between 2 and 3. This is in reasonably close agreement with an earlier analysis (Peto, 1978).

R.C.A. Exhibit 37, Tab 7, Peto (1980), supra note 71, at 911.

> with current methods, the hygiene standard as currently enforced would have been adequate to control serious disease.

Although the Simpson Committee Report evidences some hesitation about accepting the new exposure figures, Dr. Acheson, the Report's chief medical consultant, told the Commission such an upward adjustment was warranted:[70/]

> [T]he Simpson Committee, I think, was prepared to accept that there had been an approximately fivefold increase in rigorousness of the standard.

Thus, both the original 1968 BCMS and the more recent 1979 Berry analyses of the Rochdale asbestosis experience have determined that a 2 fibers/cc standard is very protective for prevention of asbestosis. Although the data base shifted with respect to both disease and exposures from one study to the next, both have predicted less than 1% prevalence of asbestosis among lifetime workers at exposure levels equal to existing maximum permissible levels. As average exposures in factories complying with permissible exposure limits will be well below the regulatory standards, an additional margin of safety will exist.

b. The Mortality Study.

The Rochdale cohort has also been reviewed several times for mortality results since the 1955 study by Sir Richard Doll, most recently by Commission witness Julian

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70/ 9 Acheson Tr., Vol. XIX at 55.

Peto.[71/] Detailed individual exposure data, however, have yet to be compiled and published. Instead, only overall averages for the entire cohort exist. Thus, even the type of cumulative exposure information used in the Rochdale morbidity study has not been calculated.

The Rochdale mortality studies have divided workers into several major subcohorts based on whether they had worked more than 10, or more than 20, years, and whether their exposures had begun prior to 1933 when major ventilation systems were introduced, or prior to 1951 when routine dust sampling was begun (but no major changes in dust level occurred). This study, unlike the McDonald, Weill, and Ferodo studies, does not include any short-term (less than 10 year) workers. The total cohort includes 1106 persons in the following categories:

Subcohort		Years of Exposure Total	Years of Exposure Total pre-1933
1	69 Men	20+	10+
2	74 Men	20+	10-
3	263 Men	20+	None
4	679 Men	10-19	None
5	284 Women	10+	None

71/ Doll, R., "Mortality from Lung Cancer in Asbestos Workers," 12 Br. J. Industr. Med. 81-86 (1955). See Knox, J. F. and R. S. Doll, "Cohort Analysis of Changes in Incidence of Bronchial Carcinoma in a Textile Asbestos Factory," 132 Ann. N.Y. Acad. Sci. 526-535 (1965); Knox, J. F., et al., "Mortality from Lung Cancer and Other Causes Among Workers in an Asbestos Textile Factory," 25 Br. J. Industr. Med. 293-303 (1968); and R.C.A. Exhibit 37: Tab 1, Peto (1977), supra note 62; Tab 7, Peto, J., "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," in Biological Effects of Mineral Fibers (IARC Lyon 1980).

things, this decrease in exposure predicted to cause disease reflected a more particularized finding that some of the men had spent some of their time in less dusty jobs than had been assumed in the prior study.[47/] The 1979 study predicted 1% prevalence of crepitations after 43 fiber-years exposure. Possible and certified asbestosis at 1% prevalence levels were estimated at 55 and 72 fiber-years, respectively.

During his testimony before the Commission, Geoffrey Berry noted that since publication of the 1979 update, refinements have been suggested for the exposure data upon which it was based. Recognition that the estimates of fiber concentrations may have been too low by a factor of three to four demonstrates that the earlier 1968 predictions of the exposure levels estimated to lead to a 1% prevalence of asbestosis may have been approximately correct. As Mr. Berry testified:[48/]

> Q. I believe yesterday, with Mr. Laskin, you talked about the difference between the one hundred and twelve fiber years per cubic centimeter calculation, which in 1968 was believed by the BCHS study to equate with one percent prevalence of crepitations, and your recalculation in this study that a

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47/ B.C.A. Exhibit 13, Feb 6, Berry (1979), supra note 41, at 105. The 1979 Berry study also postulates a dose-response model in which earlier exposures are weighted as more significant than more recent exposures. The study concludes that considerably lower standards would have to be set if this weighted fiber exposure calculation were employed, but notes there are insufficient data to substantiate this model.

48/ G. Berry Tr., Vol. XI at 16. See also id. at 13.

> prevalence of one percent might well occur at forty-three fiber years exposure?
>
> A. Yes.
>
> Q. I believe you went through three reasons with Mr. Laskin on why that number has decreased?
>
> A. Yes.
>
> Q. If you were to further refine that estimate, based on the new exposure numbers calculated in the Feto document, historically at Rochdale, what would happen to that number?
>
> A. It would increase by between two and threefold.
>
> Q. In other words, the prediction of at what exposure level one percent crepitations would occur would go up to somewhere between eight[y] and a hundred and thirty fiber years?
>
> A. Yes. Correct.
>
> Q. If there is a further difference between static sampling and personal sampling, that number would go up even higher in terms of a current plant [illegible] with the two fiber standard, [illegible] personal samplers?
>
> A. Yes, that is so.

The Simpson Report similarly noted the importance of the new exposure estimates:[49/]

> [I]f it is accepted that the doses inhaled by these men in the 1950s and 1960s have been under estimated by a factor of three or more, as compared

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49/ British Advisory Committee, Asbestos (hereinafter cited as "Simpson Report"), Vol. I at 11 (1979).

exposures were calculated on a person-by-person basis. The exposure measurements employed were similar to those used in the earlier study, except that concentrations between 1944 and 1953 were assumed to be only 1.25 times the 1951 values. The 1979 report notes that the concentrations particularly for the early years are probably underestimates.[42]

In contrast, person-by-person exposures have never been calculated for the mortality studies. Instead, only rough approximations of average exposures have been made. As Julian Peto, author of the more recent mortality studies, noted for the Commission:[43]

> [T]he things that are said about dust rules there, and measurements, come from the company, and I'm really not competent to comment on them. ... I really don't know about it in detail.

a. The Morbidity Study.

The British Occupational Health Society (BOHS) examined in the late 1960's data on 290 men with more than 10 year exposures after 1933 at the Rochdale plant for indicia of asbestosis, basal rales and x-ray changes.[44] Based on

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42/ B.C.A. Exhibit 13, Tab 8, Berry, G., et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory," 36 Br. J. Industr. Med. 98-112, at 100 (1979). As Mr. Peto also has noted, even in the 1930's, "it is known that dust levels in some areas remained high." B.C.A. Exhibit 37, Tab 1, Peto, J., "A Mortality Study Among Workers in an English Asbestos Factory," 34 Br. J. Industr. Med. 169-73, at 171 (1977).

43/ J. Peto Tr., Vol. XXVA at 83-84.

44/ BOHS, "Hygiene Standards for Chrysotile Asbestos Dust," 11 Ann. Occup. Hyg. 37-69 (1968).

these data and the exposure estimates, BOHS concluded that 1% of the men exposed for 81 fiber-years would develop basal rales and 1% of the men exposed to 31 fiber-years would develop x-ray changes. A two-fiber limit was established in Britain based on this study to prevent more than 1% of the men from developing such indicia of asbestosis.

Based on a number of criticisms, the initial Rochdale asbestosis study was updated in 1979.[45] In a study of 379 men who had worked more than ten years in the factory, asbestosis was diagnosed as certified or "possible" based on the combined judgment of two physicians. This study differed from the previous study by including both active and inactive workers, longer follow-up, use of a lung function test in addition to radiography, and inclusion of an additional 89 workers.[46]

Greater disease was found in the follow-up study; the percentage of workers with crepitations, for example, increased from 6% to 22%. As a result, it was concluded that lower amounts of total fiber exposure than had been estimated in 1968 could cause asbestosis. Among other

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45/ B.C.A. Exhibit 13, Tab 8, Berry (1979), supra note 42. An earlier report of these data appears in B.C.A. Exhibit 13, Tab 7, Berry, G. and M. C. Lewinsohn, "Dose-Response Relationships for Asbestos-Related Diseases: Implications for Hygiene Standards. Part I - Morbidity," 330 Ann. N.Y. Acad. Sci. 185-196 (1979).

46/ Some of the radiological and other signs found among the cohort may have been due to occupational exposure outside the Rochdale plant. Only 40% of the cohort were first employed at the factory before age 30. B.C.A. Exhibit 13, Tab 7, Berry (1979), supra note 45, at 191.

Second, Dr. Enterline notes that most of his cohort was urban and had their deaths been compared to urban, rather than nationwide, lung cancer rates, the SMR for the under 128 mppcf-years exposure group would have been 146 rather than 167.

Finally, Dr. Enterline notes that in a parallel investigation he conducted of retirees in the building trade manufacturing industry, where workers were also exposed to dust, but not asbestos, he had found a lung cancer SMR of 170. Thus, he concluded that although "large increments in respiratory cancer appear at a cumulative asbestos dust exposure of somewhere between 100 and 200 mppcf-years,"[47/] it "cannot definitively be established from these data" whether the increased lung cancer in the lowest subcohort is due to asbestos.[48/]

The Enterline study does not, therefore, as did the McDonald, Weill and Ferodo studies, include a substantial group of workers whose exposures were low enough that no increased risk of disease was found. However, as Dr. Enterline has stressed, the disease found in his lowest exposure subcohort may well not have been due to asbestos exposure. His study is thus not inconsistent with the general picture established in the first three studies that have focused on collection of individual exposure information.

47/ Id. at 166.

48/ Id. at 168.

9. The Rochdale Textile Factory Studies.

The initial attempts to determine dose-response relationships of asbestos-related diseases were based on data from a cohort of workers at a textile factory in Rochdale, England. The health of workers in this plant has been reviewed on several occasions.

Although detailed individual exposure data have been compiled for the Rochdale plant's morbidity cohorts, less refined exposure calculations characterize the mortality studies. In the first 1968 morbidity study, estimates of total exposure were calculated based on the areas of the plant in which men worked and dust levels in such areas as measured by area particle counts between 1952 and 1960 and area membrane fiber counts between 1961 and 1966. Exposure pre-1952 was estimated to be at least 30% higher than what it was in the early 1950's.[49/] In the 1979 morbidity study,

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49/ As Dr. Acheson emphasized fiber/particle conversions were necessary to reach these estimates.

> I think it is important to point out however, that there is a concealed conversion factor which we do not know. . . . [I]n relation to Rochdale as elsewhere, a conversion factor has had to be made by someone at some stage, when they refer to information which was recorded in relation to measurements taken before 1962.
>
> I am not aware that the actual conversion factors used here ever been published, but I may be incorrect.

D. Acheson Tr., Vol. XIX at 57.

his results would not have been changed had he studied only miner retirees:[15/]

> [W]e have thought it useful to take our cohort and to repeat the analysis (and we've recently presented a paper on this), limiting our data to the retirees. And it is again interesting to find that we get precisely the same dose-response relationship as we get on the whole study.

Dr. Crump detailed for the Commission the underlying reason why a retiree study is unlikely to be biased:[16/]

> You see that the relative risk began to rise ... at least ten years past exposure on up to a maximum of thirty years past exposure, and from that point on they began to decrease. This pattern is typical. I think it has an important implication for risk assessment. ... If one estimates risk from this point out, for example using the retirement population, it doesn't necessarily mean that you are underestimating risk because the average relative risk over here is pretty close to what it is over here.
>
> As a matter of fact, the relative risks at the age categories where most deaths occur are ones that need to be estimated most accurately, and that would be the ones that would occur in the low range.

* * *

15/ C. McDonald Tr., Vol. XIII at 56.

16/ E. Crump Tr., Vol. XXVI at 13-11, 41. Indeed, in adjusting for the possible impact of studying retirees, Dr. Nicholson found a 10% correction factor to be all that was required to take into account any deficit caused by missing earlier lung cancer deaths. B.C.A. Exhibit 13, Tab 9, Nicholson, W.J., "Dose-Response Relationships for Asbestos and Inorganic Fibers" 28 (1981).

> If that pattern that Selikoff found holds in this [enterline] study, then there might not be any bias because these are retirees. On average you may still have about the right lifetime risk estimated.

Dr. Enterline's lung cancer results demonstrate a clear dose-response relationship:[17/]

Total Dust Exposure (mppcf-years)(average)	Observed	Expected	SMR
Under 125 (63)	15	9.0	167
125-249 (181)	12	4.8	250
250-499 (351)	17	5.2	327
500-749 (606)	9	1.8	500
750 and over (960)	5	0.9	556

There is an apparent increased lung cancer risk in the lowest subcohort (SMR of 167) -- in contrast to the absence of any such increase in the Weill and McDonald lowest exposure subcohorts, or the overall Ferodo cohort. Dr. Enterline, however, has interpreted the increase as insignificant for several reasons.

First, dividing those least exposed men into further subgroups demonstrates the absence of dose-response relationships at such low exposure levels:[18/]

Total Dust Exposure (mppcf-years)(average)	Observed	Expected	SMR
Under 25 (11)	2	1.9	154
25-62.4 (44)	8	3.1	258
62.5 - 124.9 (89)	5	4.6	108

17/ B.C.A. Exhibit 1, Tab 4, Enterline (1973), supra note 46, at 14.

18/ Id.

In addition to determining exposure levels, Dr. Enterline recorded whether all exposures above 10 ppcf were "intermittent" or "steady." The great predominance of intermittent exposures were among maintenance men. As Dr. Enterline has noted: "[v]ery high levels of brief asbestos dust exposure could be hidden in these data."[51/]

No smoking data are reported for the cohort as a whole, or individual cohort members. Limited smoking data were at one time collected on 373 workers, indicating that smoking was roughly comparable between production and maintenance workers (77.3% and 76.9%, respectively, were smokers).[52/]

In his analyses of both the 1969 and 1973 results, Dr. Enterline found increased overall mortality for the cohort, with almost all the excess accounted for by increased cancer and respiratory disease deaths. Only one mesothelioma was found recorded on the death certificates of these workers by 1969 (three by 1973), but Dr. Enterline noted in his first report that supplementary data might find further mesotheliomas. Subsequent review has discovered such deaths among workers who would not have been included in the study, many of whom because they did not survive to age 63.[53/]

51/ R.C.A. Exhibit 1, Tab 3, Enterline (1972), supra note 46, at 900.

52/ Id. at 899.

53/ Id. at 902; R.C.A. Exhibit 1, Tab 10, Henderson (1979), supra note 12, at 34; see also P. Enterline Tr., Vol. VIII at 114-15.

(Footnote 53 continued on next page.)

Nonetheless, Dr. Enterline believes his lung cancer dose-response data are quite valid:[54/]

> [W]e certainly weren't missing the cancers, though. I mean the cancers are there and there is a good, sharp dose-response relationship. You have to realize that while it's clear what the selection factor is on retirees, that also applies to any other study that is done. If I were to have studied workers, say twenty years or more after first exposure, in other words I leave out all the people who die at less than twenty years, I would have also had a selection factor.

The impact of studying only retirees is quite likely to have been minimal. Dr. Corbett McDonald, for example, noted that

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(Footnote 53 continued from previous page.)

Dr. Enterline's study has been criticized, as he has noted himself, because it looks only at survivors to age 65 who may be "relatively unresponsive to hostile occupational environments." R.C.A. Exhibit 1, Tab 9, Enterline (1973), supra note 46, at 317. But, as Dr. Enterline further noted:

> Any cohort, including newborn babies, is a population of survivors. The objection to starting follow up at age 65 is, then, one of degree, not principle. ... A common fallacy in associating time-related measures of dose with mortality is to allow dose to be inversely correlated with mortality. ... We can correct for this by specifying date as of some age and starting follow-up at that point, as we have done here. As noted above, however, if exposure continues after that age, the initial classification is in error. Short of experimentation with humans, follow-up of retired populations seems to offer the best opportunity for some clear-cut measures of dose-response relationships.

See also P. Enterline Tr., Vol. VIII at 39-41.

54/ P. Enterline Tr., Vol. VIII at 139-40.

retired between 1941 and 1967 and survived to age 65 from plants producing a wide variety of asbestos products. The cohort has been analyzed in terms of type of product produced, cumulative exposure, and fiber type exposure.

The study analyzes the 1,464 men for whom job histories were available. Employment in the asbestos industry ranged from 1 to 51 years and averaged 25 years. Vital status was apparently obtained for all men in the cohort.[47/]

Although Dr. Enterline provided in his published reports few details of his method of determining exposures,[48/] he did detail the methodology for the Commission:[49/]

> My estimates on the Johns-Manville Corporation were made by somebody who I think probably at the time was the most knowledgeable guy in the world on this thing, the industrial hygienist for Johns-Manville. ...

(Footnote 46 continued from previous page.)

Med. 897-903 (1972); Tab 5, Enterline, P. and V. Henderson, "Type of Asbestos and Respiratory Cancer in the Asbestos Industry," 27 Arch. Environ. Health, 312-17 (1973); Tab 6, Enterline, P. et al., "Respiratory Cancer in Relation to Occupational Exposures Among Retired Asbestos Workers," 30 Br. J. Industr. Med. 162-66 (1973); Tab 10, Henderson, V. and P. Enterline, "Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality," 330 Ann. N.Y. Acad. Sci. 117-26 (1979).

47/ As of December 31, 1969 in the earlier reports, there had been 822 deaths, with 802 death certificates located; and as of the end of 1973 in the update for the 1,075 men in the U.S. portion of the cohort, there had been 781 deaths, with 749 death certificates located.

48/ R.C.A. Exhibit 1: Tab 3, Enterline (1973), supra note 46, at 194; Tab 10, Henderson (1979), supra note 46, at 117.

49/ P. Enterline Tr., Vol. VIII at 47.

> We did have about ten years worth of dust counts ... [W]e were estimating dust exposures in 1969, so we had exposures back to 1959, ten years back from then, and then you do know a lot of things about a factory. You know when ventilation equipment was installed, you know the amount of air that it moves, you know the design characteristics, you know the rate at which the machinery operates, you know the rate at which the looms are operating, you know if the looms were running slower, or spinning was running more slowly in 1930 than in 1940 -- this would affect the amount of dust. ...
>
> I think there is an optimum way of accounting for all these things, and I have some faith, I think, in the Johns-Manville estimates that I have. I have ways of checking on these, and I think that in fact they are probably not far off.

Based on this methodology, all jobs were placed in one of six categories, with exposure assumed to be at a midpoint in the category:[50/]

Exposure Categories	Midpoint
0	0.0
Less than 5 mppcf	2.5
5-10 mppcf	7.5
10-30 mppcf	20.0
30-50 mppcf	40.0
50+ mppcf	62.5

By correlating job histories with these job-by-job exposure estimates, cumulative exposures were determined for all cohort members. For the overall cohort, workers averaged 230 mppcf-years of exposure.

50/ R.C.A. Exhibit 1, Tab 3, Enterline (1973), supra note 46, at 193.

Lung Cancer	Follow-Up After 20 Years Exposure	
Males	0 - 10 Years	More than 10 Years
Observed	15	25
Expected	18.8	19.2
Females		
Observed	0	1
Expected	0.6	0.9

Nor were any excesses of gastrointestinal, laryngeal or ovarian cancers, or any other forms of cancer, found in any of the analyses. One worker's death was recorded as due to asbestosis.

These results occurred despite conditions, which although described as much less dusty than other factories over the past 40 years, were clearly heavier than current workplace exposures. Exposure estimates have been calculated for the factory based on both current measurements and reconstructions of historical levels. Sampling was begun in the plant in the late 1960's; prior exposure estimates are based on measurements made by running still-existing old equipment with the assistance of long-time employees to recreate historical conditions. The plant's history is divided into four exposure eras:

(1) Pre-1931 before the British Asbestos Regulations were enacted, and mean concentrations above 20 fibers/cc are estimated throughout the plant;

(2) 1932-1950 after exhaust ventilation was added to much of the machinery, and average exposures are estimated to have varied between 1 and 10 fibers/cc depending on plant area;

(3) 1951-1969 when additional ventilation and housekeeping measures were instituted leading to estimated mean exposures from 1 to 5 fibers/cc; and

(4) The era since the 1971 institution of the 2 fiber occupational standard, during which time average exposures are estimated to have been in the 0.5 to 2 fibers/cc range.

Only within the past decade, therefore, have all average exposures in this plant been below 2 fibers/cc.

Dose-response relationships based on the Ferodo data are to be examined in a subsequent paper. Nonetheless, the absence of increased risks in this substantial cohort -- in a factory with well-controlled, but nonetheless higher than modern, exposure levels -- provides additional support for the proposition that de minimis, if any, risks exist in current asbestos operations. As average exposures in modern plants will be well below regulatory standards, substantial evidence exists that risks will be de minimis.

4. The Enterline Factory Worker Study.

Dr. Philip Enterline and his co-workers have reported on several occasions on a cohort of 1,492 retirees who had been either production or maintenance employees in various U.S. and Canadian asbestos primary production manufacturing plants.[46/] Dr. Enterline's cohort includes all men who

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[46/] R.C.A. Exhibit 11 Tab 3, Enterline, P., et al., "Mortality in Relation to Occupational Exposure in the Asbestos Industry," 16 J. Occup.

(Footnote 46 continued on next page.)

3. The Newhouse and Berry Friction Plant Study.

The most recent study indicating that the reduced exposure levels common in industry today are unlikely to lead to significant disease is that preliminarily described by Geoffery Berry during his Commission testimony. Mr. Berry and his co-investigators, Drs. Muriel Newhouse and J.W. Skidmore, have studied the mortality of all workers employed since 1941 at the Ferodo friction material plant in England.[41/]

Of the more than 13,000 workers at the plant, 99% have been traced through the end of 1979, by which time nearly 3,000 had died. After analysing the data in terms of employment duration at the plant and time since first exposure, the authors report:[42/]

> The main impression is that there is little evidence of any excess mortality either from all causes or from any particular cause and notably lung cancer.

Ten mesothelioma deaths (all pleural, eight male and two female) were found.[43/] However, there was no other

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41/ B.C.A. Exhibit 13, Tab 13, Newhouse, M. L., et al., "A Mortality Study of Workers Manufacturing Friction Materials with Chrysotile Asbestos," BOHS Fifth International Symposium on Inhaled Particles (Cardiff, Wales 1980).

42/ Id. at 10.6.6.

43/ The investigators have separately analysed chrysotile and crocidolite exposures of the mesothelioma victims. An analysis made possible because crocidolite was used only for one product (railway blocks) in

(Footnote 43 continued on next page.)

evidence of excess disease. Excluding the mesotheliomas, the cohort (all of whose members were followed at least ten years beyond first employment) demonstrates a consistent absence of excess lung cancer risk:[44/]

	Lung Cancer Total Male	Length of Employment Less than 1	1-5 years	More than 5
Observed	143	43	24	76
Expected	139.5	41.9	30.1	77.6

	Lung Cancer Total Female	Length of Employment Less than 1	1-5 years	More than 5
Observed	6	5	0	1
Expected	11.3	4.6	2.5	4.2

Nor was a significant increase in lung cancer found even among those workers who were followed for at least ten years after exposure for twenty years:[45/]

(Footnote 43 continued from previous page.)

two limited eras (1929-33 and 1939-44). All but two of the ten mesothelioma cases had crocidolite exposure. Based on a case-control analysis, which classified persons as crocidolite exposed or not, and, in order to control for heavy chrysotile exposure, considered whether each person had a chrysotile exposure over 5 fibers/cc at some time, the authors concluded that crocidolite exposure was predominantly responsible for the mesothelial tumors. Id. at 10.6.7.

44/ Id. at 10.6.3.

45/ Id. at 10.6.5. The 25 vs. 19.2 difference for 20 year employees followed 10 or more years beyond exposure is not statistically significant. G. Berry Tr., Vol. X at 41-42.

more than 100 appcf-years. In contrast to the morbidity cohort, Dr. Weill notes "regrettably" the lack of smoking information for his mortality cohort.

The investigators found no increase in overall expected deaths (based on both national and Louisiana rates) at any exposure level. Nor was there any finding of increased gastro-intestinal cancer deaths, nor deaths for any cause other than respiratory malignancy.

The study did find statistically significant increased respiratory system cancer in the two highest exposure categories (SMR of 290 for between 101 and 200 appcf-years and SMR of 224 for over 200 appcf-years).[17/] Two pleural mesotheliomas were diagnosed in the study population, although not included in the cohort as both deaths occurred fewer than 20 years after initial exposure.[18/]

Dr. Weill also presented his results by use of a case-control method with four controls selected from the cohort for each lung cancer case. This analysis found significantly increased lung cancer risks, but again only in the two highest exposure groups. Enhanced risk of respiratory

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17/ Dr. Weill notes the close correlation in this regard between his results and those at the Rochdale textile plant, described below. For all persons in the Weill cohort followed for at least 20 years with more than 10 years employment, the respiratory cancer SMR is 213; in the Rochdale cohort, as reported by Julian Peto in 1978, the workers employed and followed for the same periods had an SMR of 217. B.C.A. Exhibit Tab F, Weill (1979), supra note 3, at 15).

18/ Dr. Weill notes the possibility mesothelioma has been under diagnosed in his cohort. Id. at 349.

malignancy was found particularly among those workers exposed to crocidolite as well as chrysotile, and among workers exposed intermittently in maintenance jobs. Dr. Weill and his co-authors thus conclude:[19/]

> In this study, whereas excess risk of respiratory cancer was detected in workers with moderate and heavy exposure, the categories of lowest exposure to asbestos dust did not exhibit excess risk of respiratory malignancy. Such findings are not necessarily incompatible with a linear response curve at low doses because of the relative insensitivity of currently used epidemiologic methods in detecting slight increases in risk when compared to background. They do indicate, however, that any excess risks at low degrees of exposure are small.

In both the morbidity and mortality studies, the Louisiana asbestos-cement workers have not evidenced asbestos-related disease in the lowest exposure subcohort. As Dr. Weill thus noted for the Commission,[20/]

> [T]he risk at levels below what we have measured, or estimated, to be a hundred million particles per cubic foot years [is] ... a risk we can't detect.

The Louisiana asbestos-cement plant results thus agree with those found in the Canadian miner studies and demonstrate that the strong likelihood of no, or de minimis, risk at levels much above those in existence today is not limited solely to chrysotile mines.

19/ B.C.A. Exhibit [illegible], Tab [illegible], Weill (1979), supra note 3, at 153 (emphasis supplied).

20/ H. Weill Tr., [illegible] IX at 7.

The study reviews the mortality of all persons employed for at least one month before January 1, 1970.

The originally identified cohort included 7,629 workers, but 312 women were excluded from analysis. In order to maximize the likelihood of detecting carcinogenic effects, results are reported for the 5,645 men followed at least 20 years after initial exposure. Mean follow-up of the analyzed cohort was 28 years. The cohort includes both long and short term employees; 60% were employed for less than one year, 24% for one to ten years, and 16% for more than 10 years. A total of 601 persons were identified as deceased; death certificates were located for 91% of them.

The somewhat lower trace rate in the Weill mortality cohort (vital status was not obtained for 25% of the men because of confidentiality limitations on availability of information from the U.S. Social Security Administration), as compared to other asbestos studies, has led some commentators[33/] to question the study's results, particularly with respect to the findings of no increased lung cancer risk in

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(Footnote 34 continued from previous page.)

Hughes, J., et al., "Mortality Study of Workers Engaged in Manufacture of Asbestos Cement Products," from Proceedings of the National Meeting of the American Lung Ass'n/American Thoracic Society (1978); and Tab 11, Hughes, J., and H. Weill, "Lung Cancer Risk Associated with Manufacture of Asbestos-Cement Products," in Biological Effects of Mineral Fibres 627-35 (IARC Lyon 1980).

33/ See, e.g., N.C.A. Exhibit 26, Tab 11, OSHA-NIOSH Asbestos Working Group Report, at 21 (1980).

the less exposed subcohort. As Dr. Weill explained to the Commission, however, although the trace rate is a legitimate cause for concern, it is unlikely to have had a significant impact on his results:[34/]

> [W]e also had a better trace, a much higher trace, among the high-exposure groups than the low-exposure groups, which might lead to the question ... are we underestimating mortality in the low-exposure groups because our trace rate was less satisfactory than it was in the higher?
>
> [T]here isn't any reason, to a degree or otherwise, to suspect that people who have been lost to follow up are any more likely to die, say of asbestos-related disease, than they are anything else. There is just no reason that I know of, we thought very hard and we had a lot of help in thinking about this, people who believed in the credibility of this and others who perhaps had some doubt, but no one has come up with any reason why there ought to be a relationship between trace and cause of mortality.

Any doubts concerning proper interpretation of the Weill mortality study because of the trace rate will shortly be resolved, as the Tulane investigators are currently updating the study and achieving much higher trace rates.

Following the same procedures as were used for the morbidity study, subjects were classified into total cumulative dust categories from less than 10 mppcf-years through

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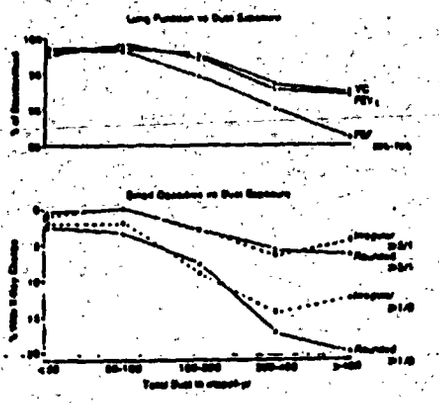
34/ H. Weill Tr., Vol. IX at 30.

As Geoffery Berry noted during his testimony, the persons lost to trace may be either fitter or less fit than the persons traced and accounted-for in any cohort study, thus making it possible for results to be biased either to the high, or low side. G. Berry Tr., Vol. XI at 72-73.

The results showed a dose-response relationship for both radiologic and lung function changes:[20/]

> The prevalence of both small rounded and small irregular lung opacities has been correlated with increasing levels of total dust exposure, findings similar to those reported by the Quebec group. ... Confidence in these dust assessments has been strengthened by the results of this investigation of the effect of exposure on pulmonary function. Progressive reduction in a number of lung function measurements is clearly associated with increasing level of total dust exposure in this population.

The investigators also found, as shown in the graph below, a clear exposure category (under 100 mppcf-years) below which such changes were not detected:



20/ B.C.A. Exhibit 7, Tab 4, Weill (1975), supra note 26, at 95.

They thus wrote:[21/]

> [T]he data from this study demonstrate not only a dose-response relationship but suggest that a threshold dust level exists in this industry below which diffuse pulmonary fibrosis does not occur.

Dr. Weill estimated that the no-effect level he determined of 100 mppcf-years would, based on a conservative particle/fiber conversion factor, be equivalent to 200 fiber-years, or an exposure to 5 fibers/cc for 40 years.[22/] He noted, however, that diffuse lung changes can occur after exposure has ended and therefore continuing follow-up of the workers beyond active employment would be necessary to confirm absence of radiographic or functional abnormalities from the accumulated exposure.[23/]

b. The Mortality Study.

Dr. Weill and his colleagues have recently published mortality results for workers from the same two plants.[24/]

21/ B.C.A. Exhibit 7, Tab 4, Weill (1975), supra note 26, at 97.

22/ During his testimony Dr. Weill noted that more refined subsequent particle/fiber conversions would now lead him to lower these numbers slightly. H. Weill Tr., Vol. IX at 52.

23/ B.C.A. Exhibit 7, Tab 4, Weill (1975), supra note 26, at 97.

24/ B.C.A. Exhibit 7, Tab 7, Weill, H. et al., "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos-Cement Manufacturing," 120 Am. Rev. Resp. Dis. 345-54 (1979). Other reports of the mortality experience of this cohort are found in Tab 6, Abstract.

(Footnote 24 continued on next page)

study of lung function consequences and radiographic opacities in 859 workers from two Louisiana asbestos-cement plants.[26/] The predominant fiber used in these two plants was chrysotile. Crocidolite was used in the pipe department of the one plant (Plant B) which also produced roofing, shingles and flooring; the other plant (Plant A), which produced only shingles, used amosite and crocidolite only infrequently. Silica was present in both plants.

The Weill morbidity cohort consisted of 91% of all workers who were in the two plants as of November 3, 1969. During half-day examinations, each worker was X-rayed,[27/] a brief physical examination was performed, and a variety of lung function tests performed. Individual smoking data were also obtained at the time the cohort members were examined.

26/ B.C.A. Exhibit 7, Tab 4, Weill, H., et al., "Lung Function Consequences of Dust Exposure in Asbestos Cement Manufacturing Plants," 30 Arch. Environ. Health 88-97 (1975). See also the earlier reports of this study in Tab 2, Enterline, P.E., and H. Weill, "Asbestosis in Asbestos Cement Workers," in Proceedings of International Conference on the Biologic Effects of Asbestos 178-83 (IARC 1974); and Tab 3, Weill, H., et al., "Radiographic and Physiologic Patterns Among Workers Engaged in Manufacture of Asbestos Cement Products," 15 J.O.M. 248-51 (1973).

In a later review of these data, Dr. Weill focuses on X-ray and lung function comparisons among long-term employees with and without substantial crocidolite exposures and concludes that both symptoms were more prevalent in crocidolite-exposed workers than in workers with equivalent chrysotile exposures. B.C.A. Exhibit 7, Tab 5, Weill, H., et al., "Differences in Lung Effects Resulting from Chrysotile and Crocidolite Exposure," in Walter, W. H., editor, Proceedings of Fourth International Symposium on Inhaled Particles and Vapors 789-98 (1977).

27/ Following a recommended, but not always followed, procedure (see C. McDonald Tr., Vol. XIII at 86; E. Holstein Tr., Vol. XIII at 31), films of unexposed controls were randomly distributed among the collected X-rays prior to their reading. B.C.A. Exhibit 7, Tab 4, Weill (1975), supra note 26, at 89.

Cumulative dust exposure for the workers -- whose average time in the plant was 17 years, with a range from one month to 45 years -- were calculated based on dust samples collected by midget impingers between 1952 and 1969. Earlier dust measurements were estimated after interviews with long-term employees. Based on detailed occupational histories and ten mutually exclusive dust categories at Plant A and 57 such categories at Plant B, total dust exposures for each individual were calculated by the Tulane investigators and expressed in lifetime mppcf-years.[28/] In an assumption that conceivably underestimated early exposures, the investigators assumed pre-1950 exposures to have been equivalent to those first measured in the plants. Dust composition in terms of chrysotile, crocidolite and silica was also assessed.

Dr. Weill described for the Commission the determination of individual exposures for each cohort member:[29/]

> [W]e have had to reconstruct individual exposures based on total particulates, similar to what has been necessary in Quebec mining and milling operations, and similar to the exposure reconstructions that you heard about, I assume, on Thursday of last week when Phil Enterline appeared before you.

28/ The industrial hygiene team and methods used for Plant B were the same as those used to estimate exposures for this plant in the Enterline study, below, as workers from this plant were also included in that study.

29/ H. Weill Tr., Vol. IX at 18.

I'm quite convinced now, that it's fairly evident to me that safety, at

(Footnote 24 continued from previous page.)

> Additionally, there may be something different in our group in terms of the age distribution as well, although with his I was using the twenty-plus year group, as well as in our case we had a twenty-plus year group. But his twenty-plus year group was a different one in that their exposure was to asbestos in different calendar periods of time as well.
>
> * * *
>
> McDonald indicated in his papers, that individuals employed at Asbestos, Quebec, had a proportionally lower mortality and disease experience than individuals employed at Thetford, and this is within similar dust categories, and he, I don't think, had an explanation of that. Our data were at Thetford Mines, the higher of the two, the area which had the higher mortality experience.

W. Nicholson Tr., Vol. XV at 59-60, Vol. XXV at 73-74.

Cumulative exposures for a group of Italian chrysotile miners were also calculated by Rubino, G. F., et al., "Mortality of Chrysotile Asbestos Workers at the Balangero Mine, Northern Italy," 36 Br. J. Industr. Med. 187-94 (1979).

This study, however, adds little to the McDonald miner dose-response information discussed in the text, because of its small size and limited data on individual worker cumulative exposures. In addition, as Dr. McDonald has noted the Italian findings are "very similar to our own." E.C.A. Exhibit 16, Tab 16, McDonald (1980), supra note 19, at 22.

Among the 932 workers studied, who had worked at the Italian mine for at least 30 days between 1930 and 1965, 332 had died, giving an SMR for all causes of 139. Significant increases over expected were found for laryngeal cancer, non-malignant respiratory diseases, tuberculosis, cardiovascular diseases, cirrhosis and accidents, while lung cancer (including one pleural mesothelioma) was not greater than expected (SMR of 106).

Although based on small numbers with only 11 total lung cancer and pleural mesothelioma deaths, the relative death rates for those men with more than 100 fiber-years exposures was 2.3 times the rate for those with less than 100 fiber-years exposure, indicating a dose-response relationship. All lung and laryngeal cancer victims had been smokers.

23/ C. McDonald Tr., Vol. XII at 137, Vol. XIII at 34.

> least to the level that is acceptable to workers -- and, having talked to them, I believe this is true -- is achievable in the mining industry, almost readily achievable, if not achieved; I've very little doubt about that.
>
> * * *
>
> [I] do believe that on the data that we have available by any likely level of acceptability, it should be possible in asbestos mining and milling to get an acceptable level. I really have no serious doubt about that.

In sum, the study by the McGill University team of the Quebec chrysotile miners and millers provides one of the most comprehensive and precise data bases available for determining asbestos dose-response relationships. This well documented, carefully analyzed study provides significant support for the proposition that the low levels of asbestos prevalent in the workplace today do not pose significant risk.

2. The Weill Louisiana Asbestos-Cement workers Studies.

As in the McDonald miner studies, the studies of both morbidity and mortality conducted by Dr. Hans Weill and his colleagues at Tulane University have focused, since their initiation, on determination of dose-response relationships.

a. The Morbidity Study.

Documentation of a likely threshold exposure below which asbestosis will not occur can be found in Dr. Weill's

> lung cancer risk equivalent to heavy smoking whereas, at more recent concentrations of around 1 mppcf [i.e., up to 5 fibers/cc], the order of risk may now approximate to less than 1 cigarette a day.

As Dr. Weill has similarly commented on the McDonald study:[23/]

> Of particular interest to a number of you, I am sure, is their finding that when they convert total dust counts to fibre levels and use fibre level measurements that have been obtained in recent years, they find no significant excess for lung cancer risk in those exposed over 20 years who have had average exposures of 20 fibres per ml.

The Canadian investigators stress the limited ability of even their large study to detect increased risk at levels which would today be considered unacceptable (about 20 fibers/cc). They thus note the need for dose-response extrapolation models to translate these findings to current occupational levels.[24/] Nonetheless, Dr. McDonald concludes

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23/ AIA/BA Industry-Government Conference Transcript 9, at 7 (Sept. 17, 1980).

24/ R.C.A. Exhibit 18, Tab 18, McDonald (1980), supra note 10, at 23.

A subcohort of the McDonald miners has also been studied by the Mount Sinai group. R.C.A. Exhibit 19, Tab 8, Nicholson, W. J., et al., "Long-Term Mortality Experience of Chrysotile Miners and Millers in Thetford Mines, Quebec," 330 Ann. N.Y. Acad. Sci. 11-21 (1979).

This cohort was made up of all men employed in 1961 at four companies in Thetford who had at least 20 years seniority (most of whom were thus born before 1920 and thus also included in the McDonald study). Id. at 11.

(Footnote 24 continued on next page.)

from his extensive, well-documented and analyzed study that an acceptably safe working environment has been achieved in chrysotile mining:[25/]

(Footnote 24 continued from previous page.)

In this study, exposure estimates were based on 97 air samples (all but five personal) made in 1973-75. Most of the workforce was employed in mills, where average readings of 9 to 35 fibers/cc (varying among the five mills) were found. The authors report that earlier year exposures would have been higher. Id. at 13-14.

With 178 deaths among the 544 person cohort, a slight excess in overall deaths was found (SMR of 111), with significantly increased deaths found for lung cancer (28 observed vs. 11.1 expected) and 26 asbestosis deaths. One mesothelioma death was discovered. There was no excess of gastrointestinal cancer. Id. at 16.

Dr. Nicholson has noted the similarity between his and the McDonald study results:

> A 20% excess in death was noted in the highest dust category [in the McDonald study] with a three to fivefold increase found in bronchogenic carcinoma. These data on the heaviest exposed group, comprising 5% of their cohort, are very similar to the experience of our entire group.

Id. at 20. Dr. McDonald agrees:

> [T]he findings in this study were essentially the same as our own, in mortality. I think they were entirely compatible with the mortality experience of the entire cohort.

McDonald Tr., Vol. XII at 71.

Dr Nicholson did, however, note a few differences during his Commission testimony:

> [W]e were using all medically available information to characterize causes of death, and this had us attribute more deaths due to asbestosis than would have been attributed by McDonald, who used death certificates only. ...:

(Footnote 24 continued on next page.
and footnote 25 on next page.)

The stability of the McDonald data base has been demonstrated by the very similar results that have been achieved over the years as the data base has grown (total deaths having increased from 2413 in 1966 to 6547 in 1978) and new analytic methods have been employed. For example, almost the same dose-response relationship has been found for respiratory cancer at each of the four periods between 1966 and 1978 when the data have been analyzed:[19/]

Cumulative Exposure Category (appf-years)	Relative Risk Range for the Four Analyses
Under 10	1.0 to 1.0
10-99	1.1 to 1.3
100-199	1.3 to 1.5
200-399	1.6 to 1.9
400-799	1.8 to 2.1
Over 800	1.9 to 3.2

As these data show, the study has consistently found a linear relationship for heavy asbestos exposures between indices of exposure (based on dust concentration multiplied

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19/ R.C.A. Exhibit 18, Tab 17, McDonald (1979), supra note 13, at .

Dr. McDonald also noted the small effect random error could have had on his findings:

> [F]rom a methodological point of view we have looked to get some sort of idea of what that kind of random error might do to our exposure response [I]t would be of the order of about sixteen percent on the data we have.

C. McDonald Tr., Vol. XII at 16.

by length of service) and lung cancer. Similar results have been found for pneumoconiosis and total deaths.[20/]

Examination of the data for the 1,904 men in the cohort employed for at least 20 years in the low and medium dust exposure groups, which averaged 6.6 appcf per year, or perhaps 20 fibers/cc, showed no excess deaths, with a Standardized Mortality Ratio of 96.[21/] The increases in number of deaths for lung cancer, esophagus and stomach cancer, and other cancers over expected deaths in this subcohort were so low as not to be statistically significant. These data led the investigators to conclude that any risk at such levels is quite small:[22/]

> [O]ne [may] speculate that the dust concentrations in the Quebec mills in the early 1930's, before effective dust suppression was introduced, carried a

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20/ R.C.A. Exhibit 18, Tab 18, McDonald (1980), supra note 10, at Table 8, p. 19.

A common methodology in equating health risks to asbestos exposure has been to calculate exposures on the basis of both concentrations at any given time and duration of exposure. Such cumulative exposures have been expressed in either appcf-years or fiber-years. One appcf-year represents exposure to an average work-day concentration of one appcf for one year; two appcf-years could be accumulated by two years exposure at 1 appcf, or one year's exposure at 2 appcf's, etc.

Such a procedure equates all asbestos exposures regardless of when they occurred or how high they were. Various reviewers have suggested that earlier exposures, or higher exposures, should be given more weight in calculating relevant lifetime exposures.

21/ Id. at 23. Conversion of the McDonald particle measurements to fiber counts is discussed seperately in Chapter IV, infra.

22/ Id. at 24.

> out the distribution of these counts, their location, the year, and as it were, without reference to the jobs, built up his best available picture of what the dust levels were in different parts of the ... particularly the mills of Quebec ... over the years in question.
>
> * * *
>
> [F]rom that he was able to make an estimate of the pattern of levels. He also interpreted these in the light of all information available to him, such as the information given by workers who were there at the time. For areas that perhaps there was no measurement, he might have well asked somebody to compare one particular part of the mill with another at that time. ... [A]ll these four thousand measurements were page on which the hat was hung, and building up ... then having done that, we then took the jobs, he took the jobs, and said right, this man was working at such-and-such a job on such-and-such a floor of such-and-such a mill from year to year, and thereby allocated his estimate of what the prevailing dust concentration was.

(Footnote 15 continued from previous page.)

After noting the many possible deficiencies in the cumulative exposure measurements used in the McDonald studies, Dr. Becklake similarly concluded:

> You could make the same comments in relation to all of them. But in all these instances, a strong relationship to exposure was shown.
>
> Poor as it is, this dust index, this cumulative index appears to be something that reflects whatever it is at risk in the working environment that produces a bad outcome.

M. Becklake Tr., Vol. XX at 84.

Dr. Gibbs concisely summarized the reasons why the Canadian mines exposure data was superior to the data in most epidemiology studies.[16]

> I would think it's probably far better than most epidemiological studies in the sense that even though we don't have measurements every day and every year and at all locations, we do have essentially systematic measurements made by the same individual over a long period of time.
>
> In most epidemiological studies, many epidemiological studies, we have to search around a lot to find the data, and very often the measurements have been made by a variety of different technicians on different occasions, and the interpretation of that information to calculate your exposure is somewhat limited.

In addition, individual smoking habits were determined through questionnaires administered to 99.6% of the cohort members alive in 1970 and relatives or friends of 93% of the cohort members who died after 1950. These smoking data have been cross-referenced to the individualized exposure data.[17]

Dr. McDonald and his investigators have analyzed the data by several different methods over the years. They have carefully considered a number of different statistical analyses and have presented and sought advice on these techniques from the British Royal Statistical Society.[18]

16/ G. Gibbs Tr., Vol. XXIX at 58.

17/ B.C.A. Exhibit 18, Tab 18, McDonald (1980), supra note 10, at 13.

18/ B.C.A. Exhibit 18, Tab 13, Liddell, F. D. K., et al., "Methods of Cohort Analysis: Appraisal by Application to Asbestos Mining," [illegible]; J. Statist. Soc. A. 469-91 (1977)

for each job at each mine, exposures prior to 1949.[14]

Dr. McDonald described the procedure to the Commission:[15]

14/ See R.C.A. Exhibit 41, Tab 3, Gibbs, G. W. and H. Lachance, "Dust Exposure in the Chrysotile Asbestos Mines and Mills of Quebec," 24 Arch. Environ. Health 189-97 (1972). As Dr. Gibbs told the Commission:

> As far as the pre-1949 period, we sat down with management and with employees and we sat down with a tape recorder and we discussed what was involved in different operations in the pre-1930 period. ...
>
> So we utilized the concentrations back in the early 1950's, in general, to extrapolate back, and modified somewhat by the information we had available from the employee and management groups about what conditions were.

G. Gibbs Tr., Vol. XXIX at 31.

Dr. Gibbs explained that pre-1940 data were available, but that their imprecision led the investigators not to rely on such measurements:

> Insurance companies, Travelers Insurance, various other companies, had made measurements in some of the mines and those data were made available to us.
>
> We chose to look at what they looked like, but . . . to try not to confuse ourselves by mixing in these other sources of information because we had no way of knowing exactly how well they were done or who did them, or when they were done, what conditions were, whether . . . somebody wanted to demonstrate the worst condition or the best condition.
>
> Whereas with the LaChance data we did, we had the man himself there to tell us what his notes meant.

G. Gibbs Tr., Vol. XXIX at 34.

Some of the measurements were noted by Mr. Lachance as peak exposures during breakdowns and other unusual situations. These measurements were not used in the calculation of cumulative exposures. G. Gibbs Tr., Vol. XXXIII at 78-80.

(Footnote 15 on next page.)

> Dr. Gibbs, who was responsible for this work, spent a lot of time in plotting

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15/ C. McDonald Tr., Vol. XII at 10-11. See also Dr. Gibbs' description of the great care that went into exposure determination. G. Gibbs Tr., Vol. XXIX at 29-30.

During the course of his examination, Dr. McDonald was extensively questioned about possible deficiencies in the exposure data (e.g., the lack of actual measurements prior to 1949 and the use of areas rather than personal samples). After explaining how the investigators took each such problem into account in the best way possible, Dr. McDonald noted that the exposure estimates were "an honest and a skilled attempt to estimate prevailing conditions." McDonald Tr., Vol. XIII at 6. He then volunteered:

> If you think the estimate is too high or too low, then one would want to know on what basis you think that, and I could discuss it.

Id. at 8. With respect to some of the criticisms of the exposure data does the record indicate that the estimates made by the McDonald team were "too high or too low."

Dr. McDonald continued:

> I can assure you that I doubt whether ... anybody in this room is as conscious of the problems of estimates as we are, but one of the interesting things is that with this enormous problem of estimate, and variation, that exposure-response lies for lung cancer is one of the most precise estimates of exposure response in cancer published in any field today.

Id. at 14.

He then concluded:

> This is one of the few studies in the world which has any data upon exposure, so that, you know, we have to say right, are we going to just reject it or are we going to say it is the best estimate that exists at this time?

Id. at 8.

(Footnote 15 continued on next page.)

update, the study is based on a cohort of 11,379 persons born between 1891 and 1920 and followed through 1975 who were employed for one month or more in the chrysotile mines of Asbestos and Thetford, Quebec, or (for a small number of persons) at an asbestos manufacturing plant in the same area. Of the total cohort (10,939 men and 440 women), more than 90% have been traced. Almost all of those workers lost to view had short employment in the mines prior to 1935. There have been 4,547 (4,463 men and 84 women) deaths, 44.3% of the persons traced.[11/]

Beyond reviewing death certificates to determine causes of death, Dr. McDonald and his colleagues made special efforts, by seeking autopsy and other medical records, to determine whether this data base included all deaths due to either lung cancer or mesothelioma.[12/]

For each person in this study, considerable effort has gone into determining asbestos exposure. A total of 5,783

11/ B.C.A. Exhibit 18, Tab 18, McDonald (1980), supra note 10, at 12-14.

During his testimony, Dr. McDonald noted that the nearly total trace rate for workers since 1950 and for workers with more than five years employment was the true test of the quality of his studies in this respect. Most of the dose-response analyses were conducted on such workers. C. McDonald Tr., Vol. XIII at 37-38.

12/ C. McDonald Tr., Vol. XII at 76-80, 94-96.

Although the investigators did not make the same concerted efforts to determine whether asbestosis-caused deaths were missed, Dr. McDonald indicated he believed it unlikely that a significant number of such deaths would have been missed by reliance on death certificates. Id. at 94-96, Vol. XIII at 68-71.

jobs have been identified in detailed work histories, and for each job, for each applicable year, an average dust concentration has been estimated in millions of particles per cubic feet (mppcf). Measurements from more than 4,000 midget impinger dust counts in annual surveys at each mine between 1949 and 1966 conducted by Maurice Lachance were used for these calculations.[13/] Lachance and Dr. Gibbs have done extensive interviewing of long-service employees to apply these data to each job and year, as well as to estimate.

13/ As Dr. Gibbs told the Commission:

> [W]e were very lucky in that regard, because Maurice LaChance, who was the engineer for the Quebec asbestos mining industry, had been with them since about 1949, and had been responsible for making all these measurements up to 1966. So we had the opportunity to work with that engineer on going back through his data and determining what his notations in his working books were in terms of whether this was an unusual situation or a usual situation, and so on.
>
> Very rarely do you have that opportunity, to be able to complete data where you have the person who actually made the measurements, that he would be there to tell us, oh, this was essentially for control purposes, we were looking to see whether there was a leak here, and so on.

G. Gibbs Tr., Vol. XXIX at 23-25.

(3) To what extent has consideration been given by the investigators to the adequacy of death certificates as a measure of actual cause of death?

Mortality studies traditionally rely on death certificate information to determine cause of death. Such information generally must be used in order to have a data base comparable to general population mortality rates which are also based on death certificate information. Errors may exist in such determinations of cause of death, leading investigators to collect more precise information. Deaths from asbestosis and mesothelioma, in particular, may require further perusal, and, thus, the extent to which failure to have conducted such reviews may impact on any given report important to its evaluation.

* * *

With those principles in mind, and with the fundamental question to be asked of the medical evidence being whether any significant risk exists from continued use of asbestos at the low exposure levels prevalent today, the following section describes the epidemiology studies which were of most significance in the record established before the Commission.

B. The Important Epidemiology Studies.

1. The McDonald Canadian Chrysotile Miner and Miller Studies.

Since 1966 the research team at McGill University, headed by Dr. Corbett McDonald, and also including Drs. Alison D. McDonald, Margaret Becklake and Graham Gibbs, has been compiling data on a very large cohort of Quebec chrysotile miners and millers. This study has focused -- from its initiation -- on quantitative determination of individual exposures. In terms of size, completeness of tracing of the cohort, precision of individual exposure numbers, and the care with which the data have been analyzed and reanalyzed, it represents the most comprehensive data base available for determining asbestos dose-response relationships.[9/]

Dr. McDonald and his co-authors have described the results of the study several times over the years, as they have updated and refined it.[10/] As of the most recent

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9/ See C. McDonald Tr., Vol. XIII at 33-53 for a detailed discussion of these aspects of the study.

10/ R.C.A. Exhibit 18: Tab 4, McDonald, J. C., et al., "Mortality in the Chrysotile Asbestos Mines and Mills of Quebec," 22 Arch. Environ. Health 677-86 (1971); Tab 11, McDonald, J. C., et al., "The Health of Chrysotile Asbestos Mine and Mill Workers of Quebec," 28 Arch. Environ. Health 61-68 (1974); Tab 17, McDonald J. C. and F. D. K. Liddell, "Mortality in Canadian Miners and Millers Exposed to Chrysotile," 330 Ann. N.Y. Acad. Sci. 1-9 (1979); Tab 18, McDonald, J. C., et al., "Dust Exposure and Mortality in Chrysotile Mining, 1910-75," 37 Br. J. Industr. Med. 11-24 (1980); Tab 21, McDonald, J. C. et al., "Chrysotile Fibre Concentration and Lung Cancer Mortality: A Preliminary Report," in J. C. Wagner, ed., Biological Effects of Mineral Fibres Vol. 2, 511-1 (Lyon I.A.C. 1980).

would not be the matches. Rather, the matches would be an indication of smoking, the true association with the observed disease. Similarly, it is quite important to investigate whether biologic data is consistent with the epidemiologic association.

A separate set of general principles exists for evaluating the quality of an epidemiologic study. These factors, referred to by a number of witnesses during the hearings, include:[7/]

7/ Additional factors more specifically related to the adequacy of studies for purposes of determining quantitative dose-response relationships will be discussed at the beginning of Chapter IV, infra.

Not included among the factors to be considered here is the so-called "healthy worker" effect. Asbestos epidemiology studies that have failed to demonstrate excess risks among exposed workers have often been criticized on the basis that workers are healthier than the general populations (used as a control group) and thus their expected mortality should be less. This effect, however, is unlikely to be significant to the studies or diseases discussed in this Chapter.

The unlikelihood that the healthy worker effect would have a significant impact on any of the major asbestos epidemiology studies, where workers have been followed for twenty or more years since first exposure, was noted by several witnesses, including Dr. Corbett McDonald:

> I think studies of healthy worker effect have shown that that sort of difference is only for perhaps the first ten years of employment, but that, by the end of around ten years of employment, the healthy worker effect almost goes. . . . [T]here's relatively little evidence of the health[y] worker *effect is relation to cancer at all*, which, when you come to think of it, is not surprising, because, after all, what causes cancer is not something which is going to influence whether you're employed or not.

C. McDonald Tr., Vol. XII at 116

(Footnote 7 continued on next page.)

(1) What is the size of the studied cohort?

One means of seeking to avoid the many uncertainties in epidemiology studies is to observe a cohort large enough that random variation is unlikely to be significant. In general, the larger a study, the more comprehensive its coverage of a sample of exposed persons, and the more confidence that can be placed in its results. In addition, the larger a study, the greater is the ability to review the data to assess any possibility of bias.

(2) To what extent have the investigators been able to trace all members of the cohort?

Closely tied to size and scope, is the extent to which health effects have been able to be determined for all of the study population. When serious gaps exist in the extent of such determination, some uncertainty may remain as to whether those persons for whom information does exist are representative of the whole population.[8/]

(Footnote 7 continued from previous page.)

And Geoffery Berry:

> If one follows these people up over a period of time, the SMR's approach a hundred from below ... and the healthy-worker effect had largely disappeared by about fifteen years after the worker was employed.

G. Berry Tr., Vol. XI at 69 See also M. Weill Tr., Vol. IX at 69.

8/ Limited tracing of cohort members in mortality studies need not, however, be a serious problem if the investigators carefully analyze and correct for any inability to determine all health outcomes. See further discussion of this issue with respect to the McDonald, Weill and Dement studies, infra

Review of the asbestos epidemiology studies thus best proceeds within a framework of accepted interpretive principles. Dr. David L. Sackett of McMaster University provided the Commission with one such set of criteria. Among the criteria particularly important for interpreting the asbestos epidemiology studies are the questions:[1]

(1) Is the association strong?

The basic data in epidemiology studies are comparisons of the number of observed and expected health outcomes. When the number of observed outcomes far exceeds what was expected, the association is strong; when differences are smaller, less weight should be given to any apparent association. In light of the many biases that may occur in such observational research, caution must be exercised not to put undue weight on other than strong associations.

(2) Is the association consistent from study to study?

In recognition of the limitations of epidemiologic studies, their practitioners often stress the need for replication of results. Only when an association has been demonstrated in different settings by different investigators

[1] Sackett, D. L. "A Review of Diagnostic Tests for Causation," presented at the Royal Commission's Second Public Meeting, App. B (Dec. 12, 1980).

can it be said to be strongly supported. As Richard Lemen told the Commission:[1]

> [T]he real value of epidemiology does not lie in one individual study. If you have one study that has an overwhelming excess, you may be able to put a lot of reliability upon the finding. But ... the real value, we see in epidemiology, is the consistency in findings.

(3) Is there a dose-response relationship?

When varying dose information is collected, considerable insight into the strength of apparent associations can be gained by determining whether a dose-response relationship exists. As it is a general axiom of biology that the greater the dose, the greater the response, studies which demonstrate not only a response, but also a response that increases with exposure, provide greater support for believing an apparent association to be real.

(4) Does the association make sense?

Finally, any apparent association must be considered within the content of all other knowledge of the chemical and of the health effect. One classic example of the need for such analysis is the apparent association that would be found in a study of persons who carry matches. They would be found to have increased lung cancer risks, but the reason

[1] R. Lemen Tr., Vol. XVII at 84.

with exposures to 100% asbestos became clear by the 1950's, focus shifted to determining whether all types of asbestos caused these same adverse health effects and whether such effects occurred among users of asbestos-containing products

Only in a limited number of the more recent studies has considerable attention been paid to determining systematically and comprehensively a quantitative dose-response relationship. Numerous difficulties exist in obtaining accurate exposure information for historical cohorts. Many studies thus provide no numerical information on asbestos worker exposures.[1/] Additional difficulties exist in translating historical measurements to units comparable to today's workplace measurements. Nonetheless, it is from the handful of studies where documented quantitative exposure information exists that data must be sought to determine whether current levels of exposure pose an unacceptable risk.[2/] Review of these studies is summarized here in two steps. First, in this chapter, we review each of the significant epidemiology

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1/ See, e.g., Selikoff, I. J., et al., "Asbestos Exposure and Neoplasia," 188 JAMA 22-26 (1964); Selikoff, I. J., et al., "Asbestos Exposure, Smoking, and Neoplasia," 204 JAMA 106-12 (1968); Newhouse, L. O., et al., "Mortality and Morbidity Among the Working Population of Anthophyllite Asbestos Miners in Finland," 31 Br. J. Industr. Med. 109-12 (1974); Weiss, W., "Mortality of a Cohort Exposed to Chrysotile Asbestos," 19 J. Occup. Med. 737-40 (1977).

2/ Only limited dose-response information on asbestos is available from animal experiments, but dose related relationships have been shown for both fibrosis and cancer by, inter alia, Wagner, J.C., et al., "The Effects of the Inhalation of Asbestos in Rats," 29 Br. J. Cancer 252-69 (1974).

studies, its major findings, and its qualitative value. Second, in the following chapter, we will return to these studies from a more quantitative viewpoint to assess their usefulness for extrapolating dose-response relationships to the low exposure levels prevalent today.

A. Principles of Interpretation.

As observations of actual human experience, rather than controlled laboratory experiments, epidemiology studies must be interpreted with great caution. In the laboratory, many conditions are controlled. For example, exposure to the substance of interest can, in most cases, be the only variable between treated and control populations. Further, the extent of treatment can be carefully measured.

Controlled experimentation is often not available for determining directly the effect of toxic substances on man. Guidance is thus sought from epidemiology studies where health outcomes of exposed humans are compared to health outcomes in populations assumed not to be exposed. In such studies, however, information collection on both the extent of exposure and health outcome poses many difficulties not found in a laboratory. As a result, epidemiologists traditionally stress the need for close scrutiny of methodology and careful consideration of numerous interpretation issues. Most importantly, it is necessary to determine whether consistency of results occurs among various studies.

CHAPTER III

III. ASBESTOS EPIDEMIOLOGY.

Despite extensive use of asbestos throughout the twentieth century, the major epidemiologic findings of adverse medical effects did not become apparent until the past 20 years. In large part, delayed discovery of medical effects is due to the long periods that occur between asbestos exposure and recognition of increased disease. Lung cancer rates greater than expected usually do not become apparent until at least 10, and more often 15 or 20 years after first exposure.1/ Even longer periods have often been found to exist between exposure and development of mesotheliomas, with incidence of disease generally not occurring until more than 20 years after initial exposure and peaking only after 35 years.2/

Epidemiology studies were first conducted with the aim of determining whether asbestos workers were more likely to die at earlier ages than the unexposed general population and whether any particular diseases accounted for excess mortality. After the relationships between asbestos exposure and asbestosis and lung cancer among manufacturing workers

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1/ See, e.g., Seidman, H., et al., "Short-Term Asbestos Work Exposure and Long-Term Observation," 330 Ann. N.Y. Acad. Sci. 61-89, at 75-76 (1979).

2/ See, e.g., Selikoff, I. J., et al., "Mortality Experience of Insulation Workers in the United States and Canada, 1943-1976," 330 Ann. N.Y. Acad. Sci. 91-116, at 107 (1979).

in the animal experiments often was quite different than that usually found in commercial conditions.

Several possible explanations for the varying animal, in vitro and human data have been suggested: (1) differences between rat and human reactions to asbestos; (2) a requirement for asbestos to be in a species for a large fraction of the animal's lifetime to induce tumors, which would occur for chrysotile in rats but would be less likely in humans, as chrysotile is less chemically stable than amphiboles and more likely to dissolve in lung fluids; (3) higher dust exposures to crocidolite in the human populations studied in part due to the greater ability of crocidolite to produce dust clouds; or (4) differences in fiber size and shape between human-encountered fibers and those tested in the laboratory.

In light of the apparently conflicting evidence on the relative biological effects of asbestos fiber types, Dr. Davis has concluded:[60]

> From the point of view of protection of the workforce, reliable epidemiological evidence must take priority over that obtained from animal experiments. There is very strong evidence that exposure to crocidolite in the past has had a far greater potential to cause mesothelioma than has exposure to chrysotile or amosite and crocidolite should be used by industry with considerable caution if at all.

60/ E.C.A. Exhibit 47, Tab 13, Davis (1980), supra note 34, at 19.

In sum, the animal and in vitro evidence does not implicate the amphiboles as being more pathogenic than chrysotile. Nonetheless, as will be discussed in Chapter III, apparently worse health experiences, especially with respect to mesothelioma, for persons exposed to amphiboles, have led some to advocate their more stringent control.

in descending order by crocidolite and amosite.[12/] Chrysotile was also found more fibrogenic and more carcinogenic in a rat inhalation study by Davis where special efforts were made to characterize and hold constant the number of fibers in the dust cloud.[13/]

Mesotheliomas have been reported by Wagner from intrapleural injections of amosite, chrysotile and crocidolite, with the highest tumor results for chrysotile, followed by crocidolite and then amosite.[14/] Wagner once again found greater prevalence of mesotheliomas with chrysotile as compared to crocidolite in rats in a later injection study at five different doses for each fiber-type.[15/] A third Wagner report of an animal injection study using UICC reference samples of asbestos fiber types, however, found increasing mesotheliomas occurrences most frequently for crocidolite, and then amosite, with least frequent occurrences for chrysotile.[16/] Stanton and Wrench found no significant differences

12/ Wagner, J. C., et al., "The Effects of Inhalation of Asbestos in Rats," 29 Br. J. Cancer 252-69 (1974).

13/ Davis, J. M. G., et al., "Mass and Number of Fibres in the Pathogenesis of Asbestos-Related Lung Disease in Rats," 37 Br. J. Cancer 673-88 (1978).

14/ Wagner, J. C., and G. Berry, "Mesotheliomas in Rats Following Inoculation with Asbestos," 23 Br. J. Cancer 567-81 (1969).

15/ Wagner, J. C., et al., "Mesotheliomas in Rats Following the Intrapleural Inoculation of Asbestos," in Shapiro, ed., Pneumoconiosis 216-19 (1970).

16. Wagner, J. C., et al., "Mesotheliomas in Rats after Inoculation with Asbestos and Other Materials," 28 Br. J. of Cancer 173-85 (1973).

between the three fiber types in their animal injection experiments.[17/]

In vitro studies of the effects of different asbestos fibers on cells in culture have also suggested that chrysotile is more cytotoxic and haemolytic than amphiboles in almost all systems examined.[18/]

After reviewing these animal data, the Simpson Report concluded:[19/]

> The conclusions from animal work are that all of the four main types of asbestos fibre have the capacity to cause pulmonary fibrosis, lung cancer and mesothelioma when inhaled, if the physical configuration of the fibres in the dust cloud are appropriate. There are no consistent differences in the incidence in relation to fibre type in inhalation experiments, but it is clear that under certain conditions chrysotile may produce at least as many tumours as the amphiboles.

The British reviewers caution, however, that the doses in these animal experiments were in the hundreds or thousands of fibers/cc, much higher than in current human conditions, thus perhaps limiting any ability to extrapolate to humans. Further, they note that the finely dispersed chrysotile used

17/ Stanton M. F., and C. Wrench, "Mechanisms of Mesothelioma Induction with Asbestos and Fibrous Glass," 48 J. NCI 797-821 (1972).

18/ See E.C.A. Exhibit 47, Tab 13, Davis, J. M. G. "Evidence for Variations in the Pathogenic Effects of the Different Forms of Commercially used Asbestos," Institute of Occupational Medicine, at 13-17 (Edinburgh, Dec. 1980).

19/ E.C.A. Exhibit 28, Tab 5, Simpson Report (1979), *supra* note 1, Vol. II at 25.

Adding additional support to Dr. Dunnigan's hypothesis that alterations in chrysotile fiber chemistry may be relatively important are the data previously published by the Selikoff Mount Sinai group that indicate ball-milled asbestos is chemically different from commercially milled asbestos, perhaps accounting for the different toxicologic results with such fibers.[49/]

In sum, as Dr. Dunnigan emphasized during his Commission testimony, chemical changes to asbestos fibers may alter their pathogenic significance. Such chemical changes occur in some asbestos processing operations and thus may be reflected in significantly reduced health consequences for persons exposed to the processed, as opposed to native, fibers. Other chemical changes could possibly be made to raw fiber during its processing in order to render it less pathogenic in use.

E. Fiber Types.

Despite concerted investigations, a clear picture has not emerged as to whether adverse health effects of asbestos vary among fiber types. Although generally worse health evidence for the amphiboles as compared to chrysotile emerges somewhat consistently from the human epidemiologic evidence,

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49/ N.C.A. Exhibit 40, Tab II, Langer, A. M., et al., "Variation of Properties of Chrysotile Asbestos Subjected to Milling," 5 J. Toxicol & Environ. Health 173-199, at 190 (1979).

especially for mesothelioma,[50/] the animal and in vitro evidence fails to demonstrate the same differences in risk potential.

The effects of asbestos have been tested in animals through both inhalation and injection experiments. In animal inhalation studies, asbestosis, lung cancer and mesotheliomas have been produced in rats with each of the three principle asbestos fiber types.

Reeves has treated five different species with the three fiber types, and produced pulmonary fibrosis in all species.[51/] He reported that amosite and crocidolite qualitatively produced more fibrosis than chrysotile, but noted that the similar masses of respirable fibers contained widely differing numbers of optically visible fibers, with the number of chrysotile fibers considerably lower than amosite and crocidolite.

A large rat inhalation study by Wagner, using UICC samples of the three fiber types at a target mean concentration of 10 mg/m³ doses for varying time periods, found statistically significant differences in fibrosis among the fiber types, with chrysotile being most fibrogenic, followed

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50/ The human evidence is discussed at in Chapter III below in the context of the overall epidemiology.

51/ Reeves, A. L., et al., "Inhalation Carcinogenesis from Various Forms of Asbestos," 8 Env. Res. 178-202 (1974).

automobile brake linings whose chrysotile content had been transformed through use. When both materials were injected into the pleural cavities of mice, the former produced fibrosis, while the latter did not. As Dr. Davis has written:[46]

> The finding that automobile brake lining dust has a very low fibrogenicity is encouraging. [E]lectron microscope studies of lesions produced in response to brake lining dust have shown that this dust contains extremely little recognizable chrysotile and what there is is mostly embedded in the plastic bonding material. No computation of urban atmospheric pollution by chrysotile from this source can therefore be made by calculating brake drum wear.

Dr. Weill has also noted that the 1979 IARC Lyon Conference participants were similarly concerned that a distinction be drawn between pure and coated fibers:[47]

> Asbestos exposure in the asbestos-cement industry was considered by some at the conference to differ from other types of asbestos exposure, due perhaps in part to altered surface properties, or possibly because asbestos fibers may be coated with small calcium-containing particles. It was suggested that results of studies of 'pure' asbestos exposure should not automatically be applied to exposures in asbestos-cement manufacture.

Dr. Dunnigan further noted that his recent experimental work had confirmed that chelating agents that alter fiber chemistry can inhibit toxic haemolytic effects of chrysotile.[48]

> While our data do not rule out the possibility of the so-called "fibre-effect", it appears that the surface chemical characteristics of chrysotile may be just as important to account for the toxic effects reported here. . . . [W]e wish to propose the hypothesis that hydroxyl groups are more likely to be responsible for the cellular damage. All our modifications of the chrysotile sample were aimed at either depleting the surface hydroxyl ions by heat treatment, or at chemically grafting other chemical groups onto the magnesium, but presumably, none of these treatments led to the leaching of Mg.

46/ Davis, J. M. G., and B. V. Cosimo, "Experimental Studies on the Effects of Heated Chrysotile Asbestos and Automobile Brake Lining Dust Injected into the Body Cavities of Mice," 19 Experimental and Molecular Pathology 339-53, at 351-52 (1973); see also, L. Le Bouffant, et al., "Influence of Thermal Treatment on the Carcinogenic Properties of Chrysotile Fibers," paper presented at the XIth Int'l Cong. on Occupational Health (Cairo, Egypt, Sept. 25 - Oct. 1, 1981).

47/ See also B.C.A. Exhibit 7, Tab 12, Weill, H., "Asbestos -- A Summing Up," in Biological Effects of Mineral Fibres: Proceedings of a Symposium, 867-73, at 868 (IARC Lyon 1980).

48/ B.C.A. Exhibit 40, Tab 9, Dunnigan, J., et al., "Cytotoxic and Haemolytic Effect of Native and Chemically Modified Chrysotile," Fourth International Conference on Asbestos 747-771, at 766 (Torino 1985).

Dr. Nicholson noted similar effects during his testimony:

> [W]hen you look at some experiments where you seriously alter the surface structure of the materials, biological effects can be changed, so particular hemolysis experiments do appear to depend upon the surface integrity of a fiber. Thus, some of the experimental work where severe physical alteration of fibers and reduced toxicity of smaller fibers that would be produced by such vigorous action might be explained in terms of alteration of surface properties, and the greater ease with which the fibers can be dissolved and altered by body fluids or otherwise.

W. Nicholson Tr., Vol. XIV at 87.

In sum, understanding of the mechanisms by which asbestos leads to asbestosis, lung cancer and mesothelioma is limited. However, the data do help explain why long, thin fibers are most likely to be associated with disease and the important role of smoking in asbestos-related lung cancers.

D. The Role of Fiber Chemistry.

Although various investigators have considered the possibility that the differing chemistry among the various asbestos fibers may be significant in explaining their biological significance, most such efforts have not been successful.[41/] However, Dr. Jacque Dunnigan of the Sherbrooke Research Center presented data concerning chemical changes in asbestos fibers that may be extremely important in future examination of asbestos risks.

Dr. Dunnigan stressed particularly the evidence suggesting that asbestos fibers become chemically altered during processing and that those alterations may lead the fibers to be of lesser toxicologic significance than the native fibers to which most studied workers have been exposed. Two examples he cited were the encapsulation of chrysotile fibers in resin as occurs in certain asbestos applications.[42/]

41/ See, e.g., G. Gibbs Tr., Vol. XXIX at 7-20, referring to his work exploring trace metals and organic compounds found in asbestos.

42/ R.C.A. Exhibit 60, Tab 3, Fowlring, G. M., et al., "Detection of Chrysotile Asbestos in Airborne Dust from Thermosetting Resin Products," 3 J. Test. & Eval. 482-90 (1975).

and the calcium coating that chemically changes chrysotile fibers during asbestos-cement production.[43/]

Dr. Dunnigan noted the importance of such findings of altered chemically fibers in light of the animal inhalation studies that have shown remarkably different results for pure chrysotile and asbestos-cement dusts.[44/] Although the explanation for the obviously lesser pathogenicity of the asbestos-cement particles is not proven, the lesser adsorption capabilities of the coated fibers were noted by Dr. Dunnigan as one possible explanation. By being less able to adsorb other chemicals, the coated fibers may not adsorb other carcinogens and thus may be considerably less likely to lead to cancer.[45/]

A significant differential in pathogenicity has also been demonstrated between pure chrysotile and dust from

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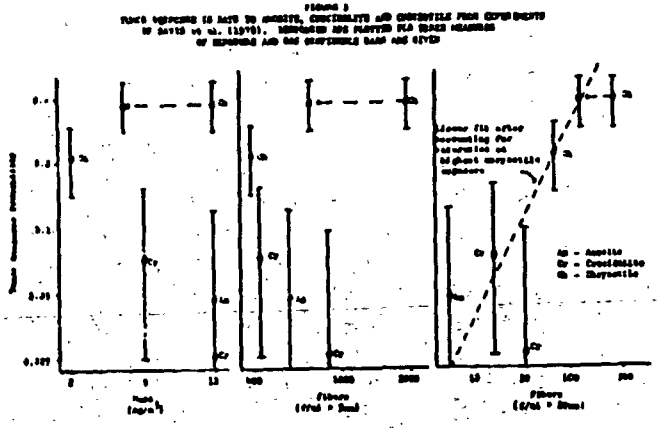
43/ R.C.A. Exhibit 60, Tab 4, Beruttere, A., et al., "Characterization and Properties of Asbestos-Cement Dust," Fourth International Conference on Asbestos (Torino 1980).

44/ R.C.A. Exhibit 60, Tab 5, Wehner, A. P., et al., "Asbestos Cement Dust Inhalation by Hamsters," 17 Environ. Res. 367-89, at 386-88 (1978). Wehner reports:

> [T]he fibrotic response was only slight at the selected AC [asbestos-cement dust] aerosol concentrations of 1 and 10 µg/liter whose asbestos concentrations of 0.1 and 1 µg/liter, respectively, correspond to 1 and 10 times the TRK of the German Research Association. ... By comparison, a 100% incidence of severe asbestosis and a high incidence in adenomas was observed in hamsters that were exposed to pure chrysotile (Wehner et al., 1974 and 1975).

45/ J. Dunnigan Tr., Vol. XXVIII at 44-50.

in his Commission testimony through use of the following graph, neither mass of asbestos nor the number of fibers longer than 5 μm correlated with tumor results.[39] The number of fibers longer than 20μm in each test group, however, correlated quite closely with tumor production, indicating that it was only such long fibers that were associated with lung cancer.



[39] E.C.A. Exhibit 38, Tab 3, Figure 3; E. Crump Tr., Vol. XXVI at 66-66.

Considerable support thus emerges from both animal studies and theoretical explanations of disease mechanisms that it is only the longer asbestos fibers that are significant to disease causation.

The importance of smoking to lung cancer mechanisms was also detailed by Dr. Kotin. While noting that it is possible for lung cancer to occur with high doses of asbestos in non-smokers, Dr. Kotin explained why such disease occurs very infrequently, even among heavily exposed workers of the past:[40]

> I think we have to acknowledge that asbestos, both on the basis of clinical studies and experimental models, is capable of inducing a neoplasm on its own. It is a carcinogen.
>
> Now, in the lung peculiarly you have a situation where the dose that would induce a neoplasm of the lung is one that, in the absence of cigarette smoking, has done this exceedingly rarely. ... [I]f you are going to suggest that asbestos is responsible for a bronchogenic carcinoma, [it] has to have the scarring, the asbestosis, beforehand, which provides the suitable setting for the action of the carcinogen. ... [Y]ou would have to say that the rare cancer of the lung in a non-smoking asbestos worker could indeed be related to the asbestos, but at least to satisfy, I think acceptable criteria of diagnosis, ..., you would have to require the presence of asbestosis as the antecedent.
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> * * *

[40] P. Kotin Tr., Vol. XXIA at 76-77.

Page II-14 & 15 missing.

> Mesothelioma is probably best regarded as a solid-state cancer due to translocation of fibers by essentially unknown mechanisms to the subpleural areas of the lung, the parietal pleura, and to the peritoneal cavity.

Evidence that long thin fibers are also most significant in causing mesothelioma also comes from more general work in solid state carcinogenicity. As Dr. Kotin explained:[17]

> [L]et me use the example of another solid state material, some tin or gold or silver ... in a series of different dimensions from rock candy size to granular sugar size to powder sugar size to triple X powdered sugar size, and kept everything constant in terms of the amount you put into the animal, where you put it into the animal -- the same species, as you got to smaller and smaller dimensions, you would obliterate carcinogenicity.
>
> So that a gram or an ounce or a ton of granular gold or granular lead will produce cancers under the skin or the brain, whereas the same ounce or pound or ton in super powder sugar equivalent will produce no tumors.

The association with long fibers also seems to hold for lung cancer. A demonstration of the great likelihood that it is only long fibers that should be associated with lung cancer comes from the rat inhalation studies. In his 1976 study at the British pneumoconiosis unit, Dr. Davis administered all three fiber types to the animals after carefully characterizing fiber dimensions.[18] As Dr. Crump demonstrated

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17/ P. Kotin Tr., Vol. XXIA at 57-58.

18/ N.C.A. Exhibit 67, Tab 38, Davis, J. M. G., et al., "Mass and Number of Fibers in the Pathogenesis of Asbestos-Related Lung Disease in Rats," 37 Br. J. Cancer 673-88 (1978).

C. Disease Mechanisms.

Clear pictures of the mechanisms by which asbestos fibers cause or contribute to lung cancer and mesothelioma have not emerged from the animal and tissue studies. A number of consistent theories, however, do appear to explain the association with fibrosis.

The association seems best explained by the activity of long fibers. As expressed by Dr. Morgan:[24/]

> There is an accumulating body of evidence which shows that the fibrogenic activity of asbestos is associated with a long rather than short fiber (Vorwald et al. 1951; Gilschar, 1972; Wright and Kuschner, 1977). This could be due, either to the leakage of tissue damaging enzymes from alveolar macrophages involved in the incomplete phagocytosis of long fibers, or to damage to the alveolar epithelial cells.

Dr. Kotin has explained how the genesis of fibrosis is likely to stem from the inability of the macrophages to enclose long fibers totally:[25/]

24/ B.C.A. Exhibit 31a, Morgan A., "Fiber Dimensions: Their Significance in the Deposition and Clearance of Inhaled Fibrous Dusts" at 8.

25/ B.C.A. Exhibit 30, Tab 8, Kotin, P., "Asbestos and Carcinogenesis," Presentation to the Given Institute of Pathobiology, at 4 (1979).

As Dr. Kotin explained during his testimony:

> [A]n incompletely phagocytized, or swallowed, cell, will allow leakage from the cell of those enzymes ... they dissolve proteins, are virtually incapable of distinguishing between host protein or alien protein.

P. Kotin Tr., Vol. XXIA at 28-29.

> [W]e've now got what I think is quite good evidence that the very, very finest ones are the most dangerous ... . [W]e certainly agree that long, very thin fibers are the most dangerous ones.

As did Dr. Kotin:[26/]

> Clearly, the work of Wagner, of Merle Stanton and his associates, of Pott and his associate Friedrichs in Germany, William Smith at [Fairleigh] Dickinson University, and John Davies at Edinburgh, have a consistency in terms of data relative to length and diameter as determinants of potency ... . [I]t's awfully difficult to quote Dr. Stanton, but I think Merle ... and most scientists who have talked to him would agree ... that he tended to lend very little credence to the sporadic tumor that he saw in animals that had not short fibers, but shorter than his eight micron fibers in length, and his one micron or one and a half micron in diameter.

Thus, concluded Dr. Kotin, one can conservatively estimate that "a fiber shorter than five micrometers in length and thicker than two and a half micrometers proved to be nonpathogenic."[27/]

The mechanism by which asbestos fibers lead to mesotheliomas is much less well understood than the fibrosis mechanism. But, as Dr. Kotin has explained, the mechanism is likely to be one of solid state carcinogenesis:[28/]

26/ P. Kotin Tr., Vol. XXIA at 51.

27/ Id. at 53.

28/ B.C.A. Exhibit 30, Tab 8, Kotin (1979), supra note 27, at 3.

> [T]here seems to be no reason to modify the conclusion expressed at the Lyon conference that as far as mesothelial tumors are concerned, "there is no evidence of a risk to the general public at present."

Dr. Davis agreed:[218]/

> [T]here is really nothing, I would say, to contradict the idea that you need pretty moderate doses of asbestos to get mesothelioma

4 Fiber Type Differences.

Additional confirmation of the unlikelihood that future asbestos exposures will be responsible for mesothelioma arises from the predominant evidence that this disease is primarily caused by amphibole exposures. Many of the Commission's witnesses agreed with the Simpson Report conclusion:[219]/

Footnote 217 continues from previous page.,

> mesotheliomas, as, you know, an anecdotal case does not establish that there is a high risk, whereas in the high exposure categories here, you know, we've got six cases out of sixty-two men. That's a risk, whereas in the general public, if Joe Public with no known asbestos exposure or with a neighborhood exposure develops a mesothelioma, and he has had four hundred thousand neighbors, then his risk is one in four hundred thousand compared to six in sixty-two, and that's obviously different.

M. Finkelstein Tr., Vol. XXXIV at 91-92.

219/ J. Davis Tr., Vol. XXXIV at 96.

218/ R.C.A. Exhibit 28, Tab 5, Simpson Report (1979), supra note 116, Vol. II at 2[illegible]. See, e.g., C. McDonald Tr., Vol. XII at 58; W. Weill Tr., Vol. IX at [illegible]; J. McDonald Tr., Vol. XXXI at 43.

> The most widely accepted view at present is that crocidolite, amosite and chrysotile (but not anthophyllite) cause mesothelioma in man, but that there is a gradient in risk, crocidolite being the most and chrysotile the least dangerous of the three common fibre types in this respect.

Despite the conflicting animal evidence, as described in Chapter II above, the evidence that amphiboles are primarily, if not almost entirely, responsible for observed mesotheliomas in man, comes from a variety of sources:

(1) Mining Cohorts. The initial suspicion of association between mesothelioma and asbestos came from the 1960 report of Wagner of 33 pleural mesotheliomas in a crocidolite mining area in northern Cape Province, South Africa.[220]/ Another report in 1973 from South Africa recorded mesotheliomas in 158 persons (all but two of whom had definite asbestos exposure) with the only exposures that could be attributed to a single type of fiber being 84 cases who had been exposed to crocidolite and 4 to amosite.[221]/ Twenty

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220/ Wagner, J. C., et al., "Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province," 17 Br. J. Industr. Med. 260-71 (1960).

221/ Webster, I., "Malignancy in Relation to Crocidolite and Amosite," in B.govska, et al., Biological Effects of Asbestos 135-38 (1973).

Often noted as unexplained is the paucity of mesotheliomas in the Transvaal South African crocidolite mining area -- 83 of Webster's 84 crocidolite-related mesotheliomas were from the Cape area. Dr. Acheson, however, preferred as explanation to the Commissioner

(Footnote 221 continued on next page.)

> lived within 20 miles of a chrysotile mine in California. The places of residence of women were examined for the period 20 through 40 years before death. Of 96 case-control pairs in Canada, 19 subjects and 21 controls had lived only in rural areas; of 50 case-control pairs in the U.S.A., 5 subjects and 10 controls had done so. In Canada, 67 subjects and 66 controls, and in the U.S.A., 18 subjects and 13 controls, had lived in urban areas only.

Both the anecdotal reports and the McDonald case-control study demonstrate that family members who have close (normally laundry room) association with heavily exposed workers are at increased risks due to their apparently heavy exposures. But no evidence indicates that truly casually exposed persons are at increased risk.

The literature includes references to persons with allegedly casual exposures who have had mesotheliomas, but the likelihood that their disease was due to such exposures is slim. As the Simpson Report noted:[215]

> [T]he ubiquitous distribution of asbestos ... makes it likely that a proportion of the cited associations between these tumours and this fibre may be coincidental and not cause and effect.

Were such general environmental exposures responsible for mesotheliomas, such an association would have been expected to have been shown in the McDonald case-control study for

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215/ B.C.A. Exhibit 28, Tab 5, Simpson Report (1979), supra note 114, Vol. II at 32-33.

urban or mining area dwellers. It was not. As Mr. Pate emphasized:[216]

> To assume on the basis of anecdotes that people who drilled a hole in an asbestos roof when they were twenty, who get mesotheliomas fifty years later due to that exposure, I think it's completely unreasonable. I think it's very unlikely that the effect of brief exposure is more than proportional, and the majority of cases seem to be occurring in people who have had fairly substantial exposure.

In sum, the oft-expressed claim that mesotheliomas can occur after but a day's exposure should be recognized as a fallacy. The overwhelming scientific evidence clearly demonstrates the normal dose-response relationship between asbestos and mesotheliomas and the consequent great unlikelihood that minimal exposures will lead to disease. As Dr. Becklake has concluded:[217]

216/ J. Pate Tr., Vol. XXVA at 131-32.

217/ B.C.A. Exhibit 28, Tab 8, Becklake, M., "Environmental Exposure to Asbestos: A Factor in the Rising Rate of Cancer in the Industrialized World?" 76 Chest 245-247, at 246 (1979). Dr. Finkelstein agreed:

> One often hears quoted or sees in print the statement that . . . someone with one day's exposure has developed mesothelioma, [that] there is no dose response and this is hazardous at all levels.
>
> My feeling is that you may certainly develop a mesothelioma with a short exposure, but the risk is substantially less than the risk at higher exposures ... You know, the case who has developed mesothelioma after a week of exposure has had four hundred thousand, perhaps other people, who have also been exposed for a week and have not developed

(Footnote 217 continued on next page.)

A. Yes.

Q. So that there would be the dust as well from the foundation, the very foundation on which the house was located?

A. Oh, yes. The general environmental exposure was quite high even much more recently than that.

Q. So that with respect to those people mentioned in table five here, the exposure that they would have had as a result of the contact with the workers could you now guess at the number of fibers? ...

A. [W]hat I could say is that concentrations of fiber in the homes of asbestos workers can be very high, because all the soft materials accumulate fibers and then as you shake them or whatever, it just puts out a dust which could be quite high. I mean it could be ten fibers, it could be really quite high.

The evidence that domestic exposures may have been extremely high is further supported by the McDonalds' case-control study, where they found evidence "that exposure in the home of children and wives to dust from the clothing of asbestos workers was related to mesothelioma but no association with either lesser degrees of occupational exposure or living near asbestos mines and mills."[212]/

The theory that mesothelioma can be caused by short casual exposures is further repudiated by the consistent findings that rates have not been increasing among women

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212/ N.I.O.A. Exhibit 18, Tab 8, McDonald (1973), supra note 183, at 362.

despite widespread use of asbestos over the past 50 years. As Dr. McDonald noted:[213]/

> [T]he contribution of occupational exposure is the main factor affecting the increased rate in males, and the absence in females implies that the non-occupational exposure is probably not having any changing effect.
>
> There may have been some low level [mesotheliomas] before, but the constancy *of this suggests that non-occupational* exposure is not having any important effect -- in North America.

The McDonald case-control study further repudiates theories about environmentally caused mesotheliomas:[214]/

> Excluding those with occupational or domestic exposure, no subject, but 2 controls, had lived within 20 miles of a Canadian chrysotile mine. In the U.S.A., 1 subject and 2 controls had

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213/ McDonald Tr., Vol. XIII at 74.

214/ McDonald, A. D. and J. C. McDonald, "Malignant Mesothelioma in North America," 44 Cancer 1450-56, at 1454 (1980).

The mesothelioma case series from Plymouth England (Sheers and Cole (1960), supra note 198) exemplifies this point. Six of the 108 cases did not have definite occupational exposure to asbestos. Five of these six had worked on the docks or served in the navy. The authors report substantial fiber counts in the lungs of two of these men, attributing the fibers to the general environment of the dockyards. They note that these five mesotheliomas thus reflect "a degree of risk proportional to exposure." The authors further note that the one mesothelioma death totally unrelated to asbestos was likely to have been a spontaneous tumor, as would have been expected among the 13,000 men from whom this series was collected, and thus conclude:

> [N]o evidence has been found to suggest any increase in the risk of mesothelioma in the unexposed population of Plymouth outside the dockyard.

Id. at 1451.

four years and his wife brushed him down."[206] As Dr. Becklake told the Commission:[207]

> Dr. Newhouse, a colleague of mine in London, measured fiber counts in relation to shaking dust out of work-clothes and found them to be extremely high.

Dr. Davis also described these simulations of past exposures:[208]

> I know some experiments have been done on this and it has been shown that exposing overalls to asbestos clouds of the sort that might have occurred a few years ago, for some days, and then brushing them in open and closed rooms, I think the figure is people have got clouds of over two hundred fibers per ml.

In a later article, Dr. Newhouse reported on a peritoneal mesothelioma patient who reported having played with handfuls of asbestos on a factory waste ground as a small boy 40 years

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206/ Newhouse and Thompson (1965), supra note 203, at 364.

The Simpson Report made similar observations:

> Bearing in mind the virtual indestructibility of asbestos it is possible to envisage circumstances where asbestos fibre could accumulate in the household of an asbestos worker (e.g. lack of changing facilities at work and of vacuum cleaning and washing facilities in the home) and substantial levels be present in the household air over long periods of time.

B.C.A. Exhibit 28, Tab 8, Simpson Report (1979), supra note 104, V. I, at 46.

207/ A. Becklake Tr., Vol XX at 17.

208/ J. Davis Tr., Vol XXXIV at 95.

earlier.[209] In another series of reported female "household contact" mesothelioma patients, all ten had husbands or fathers for whom they "routinely hand-laundered" clothing.[210]

Dr. McDonald described the very dusty conditions that might account for the three non-occupational mesotheliomas identified in Quebec mining areas:[211]

> Q. [T]he conditions in the mines were such that the workers came home with their clothes actually full of fibers?
>
> A. Oh, yes.
>
> Q. That would happen every day?
>
> A. Oh, yes.
>
> Q. In fact, weren't some of the homes even built on asbestos tailings?

209/ Newhouse, M. L., "Asbestos in the Work Place and the Community," Ann. Occup. Hyg., 87-132, at 131 (1973).

In addition, Whitwell notes that the Newhouse and Thompson information base may have been limited, thus leading the investigators to assume incorrectly an absence of direct occupational exposure:

> [I]t is highly probable that asbestos was also shipped into London docks in similar containers for transfer to asbestos factories, and that damaged sacks were repaired locally in sack-repair factories, which were numerous in London. The patients, or more often their relatives, who were questioned by Newhouse and Thompson, were asked about employment in asbestos factories, not sack-repair factories, so many of the cases described as home-environment mesotheliomas may, in fact, have been sack repairers.

Whitwell (1977), supra note 200, at 386.

210/ Vianna and Polan (1978), supra note 203, at 1062.

211/ C. McDonald Tr., Vol. XIII at 91-92.

asbestos exposure may lead to mesothelioma.[203] The Simpson Report, for example, notes:[204]

> There is strong evidence from a number of sources that inhalation of crocidolite can cause mesothelioma, and that although the risk increases with increasing dose it is possible for mesothelioma to occur after relatively brief exposure to this amphibole ... .
>
> While the majority of reported mesotheliomas are attributable to occupational and indirect occupational exposure to amphiboles, some cases have been reported where the exposure has arisen as a result of domestic contact or neighbourhood exposure.

Considerable evidence exists, however, that the few reported mesotheliomas attributed to "domestic contact" or "neighborhood" proximity to asbestos followed quite intense exposures. As Dr. Acheson noted to the Commission:[205]

> Such cases, together with the cases in persons who have only worked for a few months making gas masks, have led to considerable public anxiety. This anxiety may be greater than is necessary, because it may well be that the doses have been quite substantial.

203/ N.C.A. Exhibit 23, Tab 2, Anderson, H. A., et al., "Household-Contact Asbestos Neoplastic Risk," 271 Ann. N.Y. Acad. Sci. 311-323 (1976); Greenberg, M. and T. A. Lloyd Davies, "Mesothelioma Register 1967-68," 31 Br. J. Industr. Med. 91-104 (1974); Newhouse, M. L. and H. Thompson, "Mesothelioma of Pleura and Peritoneum Following Exposure to Asbestos in the London Area," 22 Br. J. Industr. Med. 261-69 (1965); Wignall, B. K. and A. H. Fenn, "Non-Occupational Exposure to Asbestos and Malignant Mesothelioma in Females," The Lancet 1061-63 (May 20, 1978).

204/ Simpson Report (1979), supra note 69, Vol. I at 60.

205/ E. Acheson Tr., Vol. XIX at 16.

(Footnote 205 continued on next page.)

The great majority of the non-occupational mesotheliomas among asbestos worker family members are closely related to the washing of very dusty clothing. Newhouse and Thompson report the "most usual history" of the nine relatives of asbestos workers with mesothelioma "was that of the wife who washed her husband's dungarees or work clothes." They report one relative who said that "the husband, a docker, came home 'white with asbestos' every evening for three or

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(Footnote 205 continued from previous page.)

The original report implicating asbestos with mesotheliomas included cases among residents of South African crocidolite areas. Wagner, J. C., et al., "Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province," 17 Br. J. Industr. Med. 260-71 (1960). There, too, as Dr. Davis told the Commission, exposures were likely to have been very heavy:

> [T]hey used to surface roads with crocidolite waste. We has certainly got one beautiful picture of a land rover or a jeep being driven along one of these roads, trailing a blue cloud of dust for a hundred yards behind it.
>
> Now, some of the roads in residential areas were surfaced with this material. It was good, readily-available material. Vehicles driving along these roads, I'm sure, produced dust clouds probably as high as anybody in the mines were getting, maybe as high as anybody in factories were getting.
>
> So I think perhaps we have exaggerated or used this data to exaggerate the idea of really miniscule doses.

J. Davis Tr., Vol. XXXIV at 93.

patients' lungs and the presence of asbestos-induced meso-

theliomas:[200]

> Ninety-five per cent of the patients with asbestos-induced mesotheliomas had over 50,000 asbestos fibres per gram of dried lung in the base of a lower lobe, whereas only 15% of the control series had as much asbestos. It is true that in many cases the asbestos exposure of mesothelioma patients had been of short duration, sometimes only three months, but from the amount of asbestos fibres found in these patients' lungs the exposure must have been quite intense. (The two women who had worked in a gas mask factory for only one-half year had fiber counts of 1.6 and 17 million.)

200/ B.C.A. Exhibit 33, Whitwell, F., et al., "Relationship between Occupations and Asbestos-Fibre Content of the Lungs in Patients with Pleural Mesothelioma, Lung Cancer, and other Diseases," 32 Thorax 377-86, at 383 (1977).

The Simpson Report described another tissue study with similar results:

> Anscroft, in a study of all diagnosed cases of mesothelioma in Tyneside over a period of twelve years, made a unique attempt to relate the frequency of asbestos bodies in the lungs of the cases to the frequency occurring in a representative sample of autopsies for other conditions. Of thirty-one cases of mesothelioma studied, almost all of whom had a history of exposure to asbestos in industry, twenty nine had numerous coated fibres and only two occasional fibres. In contrast he estimated, by extrapolating from his small series of controls, that about 40,000 of the adult population of Tyneside had occasional fibres and only about 30,000 adults had numerous fibres in their lungs. He estimated that the approximate relative rates of mortality from mesothelioma for, respectively, those with many and few fibres in their lungs were 600 and 5 per million. He concluded that there had been little evidence up to that time of an appreciable risk of mesothelioma due to environmental contamination in Tyneside.

B.C.A. Exhibit 28, Tab 9, Simpson Report (1979), supra note 116, Vol. II at -3.

> The risk of asbestos-induced mesotheliomas to the general public, such as those in the control series, is probably confined to the top 15% referred to above, which include no women and only men working in jobs with a definite occupational hazard from inhaled asbestos.

Despite this evidence of a dose-response relationship, some reviewers have been cautious. The Simpson Report, for example, notes:[201]

> No data are yet available which may be used with confidence in determining the shape of the dose-response relationship for mesothelioma. Such information is only likely to be made available, at least for crocidolite, if an appropriate follow-up study and analysis were completed on the Nottingham gas mask workers and it was possible to reconstruct the conditions of work.

As Dr. Nicholson told the Commission, however, there is no reason to doubt that mesothelioma is a dose-related phenomenon:[202]

> Those data that do exist are completely consistent with a linear dose-response relationship for mesothelioma ... and logic would have such a relationship ...

d. Non-Occupational Exposure.

Much of the reluctance to find a clear dose-response relationship derives from studies that suggest non-occupational

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201/ B.C.A. Exhibit 28, Tab 9, Simpson Report (1979), supra note 116, Vol. II at -1.

202/ W. Nicholson Tr. ... XXV at 00.

Length of Exposure	% with Mesothelioma
10-20 months	3.8%
20-30 months	4.0%
30 plus months	9.8%

Mr. Berry also emphasized the value of these data to the Commission:[196]

> [T]here is a similar sort of result given in our paper on the gas mask workers ... [O]ut of three hundred women with less than five months exposure, there had been no mesotheliomas. But in women with more than five months exposure, there had been sixteen out of four hundred.

Although based only on a small number of cases (eleven), the Toronto asbestos-cement plant mesothelioma experience demonstrated a similar dose-response relationship. Dr. Finkelstein found such a relationship in both his case-control and cohort analyses of the data.[197] Dr. McDonald has also reported what appears to be a "clear exposure trend" for the

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(Footnote 195 continued from previous page.)

> [D]uration of exposure is not the crucial factor in determining the amount of dust that is retained in the lungs, or in determining the liability to mesothelioma. It is more possible that the physical properties of the fibres and the type of dust clouds to which individuals are exposed are of greater relevance.

196/ J. Berry Tr., Vol. X at 18.

197/ B.C.A. Exhibit 36, Tab 7, Figure 1 - "Standardized Mesothelioma Mortality Ratios" and "Case-Control Analysis Preliminary Results," from the Finkelstein mortality study (1981).

11 mesothelioma deaths in his large mining cohort.[198] Finally, mesothelioma dose-response relationships have been demonstrated in animal studies.[199]

Body burden studies also emphasize that mesothelioma operates in accordance with dose-response principles. Dr. F. Whitwell, for example, found "a definite dose relationship between the numberr of asbestos fibres seen in the

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198/ B.C.A. Exhibit 18, Tab 18, McDonald (1980), supra note 10, at 21; see also C. McDonald Tr., Vol. XII at 102.

Dose-response relationships also emerge from the recent study of 108 mesothelioma deaths in Plymouth England, where 96 of the deaths were among workers at the Royal Naval Shipyard. Mesothelioma incidence rates in this series were correlated with both duration of employment in exposed occupations and the relative dustiness of various shipyard jobs (determined both qualitatively and on the basis of prevalence of parenchymal and pleural changes). Rheers, G. and R. Colen, "Mesothelioma Risks in a Naval Dockyard," 35 Arch. Env. Health 376-82, at Tables 7 and 8 and Figures 1 (1980).

A slightly different interpretation of the dose-response relationship is presented in Browne, K. et al., "Intensity of Exposure and Asbestos-Related Mesothelioma," paper presented at the XXth Int'l Cong. on Occupational Health (Cairo, Egypt Sept. 25-Oct. 1, 1981). Drs. Browne and Newhouse conclude on the basis of a review of 102 mesotheliomas from a factory that produced textiles, insulation and friction products, all of which cases were exposed to crocidolite, that:

> [A]n intense exposure, even if for a period as brief as a few weeks, is required for the development of an asbestos-related mesothelioma. Low dose cumulative exposure over many years does not appear to carry a similar hazard.

199/ See, e.g., B.C.A. Exhibit 16, Tab 2, Wagner, J. C., et al., "Mesotheliomata in Rats After Inoculation with Asbestos and Other Materials," 28 Br. J. Cag. 173-85 (1973).

less than two years. Despite these only rough approximations, a dose-response relationship was found:[193]

	Number	Rate/100,000 Subject Years
Women with severe exposure of more than 2 years	7	360
Men with severe exposure of more than 2 years	19	243
Women with severe exposure of less than 2 years	13	136
Men with severe exposure of of less than 2 years	16	104
Men with low-moderate exposure of more than 2 years	7	93
Women with low-moderate exposure of any duration	1	48
Men with low-moderate exposure of less than 2 years	4	33

Geoffrey Berry has emphasized the strong evidence of dose-response relationships in these data:[194]

193/ B.C.A. Exhibit 13, Tab 8, Newhouse, M. L. and G. Berry, "Patterns of Mortality in Asbestos Factory Workers in London," 330 Ann. N.Y. Acad. Sci. 53-60, Table 3 at 57 (1979).

194/ Berry, G., "Is There a Dose-Effect Relation for Cancers Related to Occupational Exposure to Asbestos Fibres? Is a Zero-Effect Threshold Value Meaningful?" in Proceedings of the Colloquium on Asbestos and Human Carcinogenesis 43-46, at 44 (1977).

Geoffery Berry again explained the importance of these data to the Commission:

> Mesotheliomas are known to occur after incidental exposure, and this has led to several people putting forward the opinion that there was no dose-response relationship.

(Footnote 194 continued on next page.)

> It is often said that there is no dose-response relationship for mesotheliomas, but this slide shows quite clearly that there is. That is, of course, not to deny that mesotheliomas can and do occur after a very short or slight exposure. The relatively few cases which have been found with only slight exposure have occurred in probably millions at risk compared with more cases with heavy exposure in fewer at risk.

Geoffrey Berry also collaborated with J.S.P. Jones, who found similar dose-response relationships among Nottingham, England, World War II gas mask assemblers. Among these women, exposed only to crocidolite, the following statistically significant relationship between duration of employment and mesothelioma was found:[195]

(Footnote 194 continued from previous page.)

> The problem with that argument was that the cases had been observed, but nobody knew how many people were at risk. In other words, we didn't know the denominator necessary to calculate an incidence rate.
>
> But in this study where we know the number of cases and we know how many people were at risk for how long, we can work out the rate ... and again there is a dose-response relationship. Less than two years given fewer than more than two years, and slight/moderate gives fewer than severe.

G. Berry Tr., Vol. X at 17.

195/ B.C.A. Exhibit 13, Tab 13, Jones, J. S. P., et al., "The Consequences of Exposure to Asbestos Dust in a Wartime Gas-Mask Factory," in Biological Effects of Mineral Fibres 637-53, at 646 (Lyon [IARC 1980).

Jones failed to find a correlation between length of exposure and lung fiber counts, leading him to comment (at 650):

(Footnote 195 continued on next page.)

> Most all mesotheliomas, in my opinion, are asbestos-related. ... [T]here's very good evidence that mesotheliomas antedate the commercial use of asbestos; that, almost for sure, there is a low base-line occurrence of mesotheliomas due to causes unknown in the general population, and probably one which affects men and women equally.

Among the non-asbestos causes of mesothelioma may be other fibrous minerals. A population with an increased prevalence of mesotheliomas has been identified in the zeolite-bearing area of Turkey.[187] Earlier reports suggested a connection between mesotheliomas and sugar cane farming in India.[188] and excess respiratory system cancer and mesotheliomas in the wetlands of southern Louisiana.[189] Mesotheliomas has also been reported among children in cases where the latency period associated with asbestos-induced disease could not have occurred,[190] and in cases that have been related to radiation exposure.[191]

187/ Baris, Y. I., et al., "Environmental Mesotheliomas in Turkey," 210 Ann. N.Y. Acad. Sci. 423-32 (1973).

188/ Das, P. S., et al., "Mesotheliomas in an Agricultural Community of India: A Clinicopathological Study," 46 Pathology, New Zealand Surgery 218-26 (1976).

189/ Woods, A. J., "Lung Cancer: Etiological Clues from Sugar Cane Farming and Mesotheliomas?" Presentation to Fourth Annual Meeting, Am. Soc. Preventive Oncology, Chicago (March ?, 1980).

190/ Grundy, G. W. and R. W. Miller, "Malignant Mesotheliomas in Childhood: Report of 13 Cases," 30 Cancer 1216-18 (1972).

191/ Babcock, T. L., et al., "Radiation Induced Peritoneal Mesotheliomas," 3 J. Surg. Oncol. 369-375 (1971).

Julian Peto stressed before the Commission his findings in a study of all mesotheliomas in Los Angeles County, California, that many cases should be considered spontaneous, not related to any exposure:[192]

> [J]ust as lung cancer is a disease that people get spontaneously, I would guess that mesothelioma is a disease that people get spontaneously. But I mean, it is difficult to be sure.
>
> But as I say, the fact that the age distribution of unexposed cases is so different, and the fact that the incidence is more or less the same in men as in women, suggests that it's simply a spontaneous cancer.

Because mesotheliomas are rare, attempts have been made in the past to associate all cases with asbestos exposure, however casual. It is now clear that such efforts were ill-conceived. Although heavy asbestos doses may be associated with the disease, all such tumors are not asbestos-related.

c. Dose-Response Relationships.

Dose-response relationships for asbestos and mesothelioma have been found in a number of studies. The first such data were from the Newhouse and Berry report on the Cape Asbestos East London factory -- where about 7% of the deaths have been from mesothelioma. They broke down the cohort into exposure categories of severe or low-moderate and more or

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192/ J. Peto Tr., Vol. XXVA at 132.

b. Other Studies.

Mesotheliomas was at one time assumed to be caused entirely by exposure to asbestos. Virtually all recent studies have, however, verified the occurrence of this cancer in individuals with no asbestos exposure history.

In a comprehensive review of all mesotheliomas reported in the medical literature and North American surveys other than the occupational cohort studies, Drs. Corbett and Alison McDonald found no definite or probable asbestos exposure in 38% (323 out of 1,851) of the cases with a history reported.[184] As the McDonalds noted, such tumors were reported in the medical literature many years prior to wide-scale use of asbestos: "The disease may be increasing but is not new. [...]" Dr. Corbett McDonald stressed the importance of non-asbestos mesotheliomas to the Commission:[185]

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[184] U.S. Exhibit 18, Tab 13, McDonald, J. C. and A. D. McDonald, "Epidemiology of Mesothelioma from Estimated Incidence," 6 Prev. Med [illegible].

[185] Tr. at 437.

Indeed, the McDonalds had previously concluded:

> Our findings suggest that asbestos exposure is associated with a relatively small proportion of the tumours that Canadian pathologists call mesotheliomas, and there is no reason to think that they differ in their diagnostic criteria from pathologists elsewhere.

U.S. Exhibit 18, Tab 9, McDonald (1973), supra note 183, at 361.

[186] C. McDonald Tr., Vol. XII at 1934-35.

CONTINUED

3. Mesothelioma: A Rare Disease Not Solely Related to Asbestos.

Although the epidemiologic findings on mesothelioma are more limited in quantity and quality than the lung cancer and asbestosis data, sufficient information does exist to place the mesothelioma risk in context. That information includes findings that:

(1) A sizeable minority of diagnosed mesotheliomas have been found in persons without any ascertainable exposure to asbestos, confirming that the disease occurs due to other stimuli;

(2) Like other cancers, mesothelioma exhibits a dose-response relationship with asbestos exposure; the highest incidence has occurred among persons with the highest doses;

(3) Although there have been anecdotal reports of short, low asbestos exposures causing mesothelioma, the significance of such reports to determining risk is subject to considerable uncertainty in light of the evidence of a dose-response relationship, the likelihood that many of the exposures characterized as minimal were in fact rather severe, and the potential for mesothelioma to develop without asbestos exposure;

(4) Incidence of mesothelioma in man has almost always occurred in the presence of exposure to amphiboles (crocidolite or amosite), and only rarely where exposure was limited to chrysotile. Despite animal evidence indicating the ability of chrysotile to cause mesothelioma, the human evidence provides some assurance that this disease is limited to heavy amphibole exposures.

a. Diagnostic Difficulties.

Consideration of mesothelioma begins with a recognition that the likelihood of incorrect diagnosis is great. Diagnosis review by panels of experts is recognized throughout

the world as indispensable. The European Economic Community and several nations have established mesothelioma panels to address this need.

As Dr. Weill noted, mesothelioma panels have often not confirmed "as many as fifty percent of the suspected cases submitted to them." Although diagnostic difficulties could lead to both under- and over-counting, he continued, the error among persons known to have been exposed to asbestos is most likely to be one of over-counting.[101] Even among experts, there is, at best, often agreement on the diagnosis in only 80 percent of reviewed cases. Thus the number of mesotheliomas, and whether incidence is increasing or decreasing, is still subject to considerable uncertainty.[102]

The possibility that mesothelioma is over-diagnosed particularly often in areas of asbestos employment is suggested by the experience of the Canadian Mesothelioma Panel. Of 194 cases from throughout Canada reviewed by the Panel between 1960 and 1970, only 55% were accepted. The acceptance rate was lowest (42%) for those tumors reported from the country's primary asbestos mining province, Quebec.[103]

101/ B. Weill Tr., Vol. IX at 86.

102/ Brief to the Commission of the Johns-Manville Corp., at 9 (Feb. 1981).

103/ R.C.A. Exhibit 18, Tab 8, McDonald, A.D. and J.C. McDonald, "Epidemiologic Surveillance of Mesothelioma in Canada," 109 C.M.A.J. 339-42, at 340 (1973).

fiber types,[177] some evidence indicates that lung cancer is related differentially to particular fiber types. The crude risk figures, as described in the Simpson Report, would tend to indicate that chrysotile is the least risky fiber:[178]

> [A]ll exhibit relative risks [among industrial populations that are] higher than those in chrysotile [RR=1.2:1] and anthophyllite [1.7:1] miners, the group with what is perhaps the lowest proportional exposure to crocidolite (the Rochdale survey) having the lowest relative risk 2.1:1. The groups of factory workers with mixed exposure to chrysotile, crocidolite and amosite (Enterline's and Newhouse's study) have relative risks of 2.7:1 and 2.6:1, while the studies of American insulators, who are exposed to mixtures rich in amosite, yield very much higher relative risks; namely, 7.3:1 and 4.9:1.

The Report, however, cautions:

> It is important to point out, however, that the validity of comparison is limited by other important differences between the studies.

Dr. Enterline has reported statistically significant higher relative risks for lung cancer among maintenance workers who would have been exposed to amphiboles and crocidolite as compared to production workers with similar cumulative dust exposures exposed only to chrysotile. He attributes

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177/ Chrysotile -- McDonald (1980); crocidolite -- McDonald & McDonald (1976) and Jones, et al. (1980); amosite -- Seiboss, et al. (1979).

178/ B.C.A. Exhibit 28, Tab 3, Simpson Report (1979), cited note 114, Vol. II at 27.

these differences to fiber type distinctions.[179] Similar conclusions were reached by Dr. Weill in his study of asbestos-cement plant workers:[180]

> The pattern that emerges from the SMR suggests that the addition of crocidolite to chrysotile enhances the risk for respiratory malignancy, particularly for those workers exposed intermittently in maintenance jobs. The exposure history of this latter group is characterized by exposure to high concentrations of dust for short periods of time.
>
> * * *
>
> In sum, strong evidence exists that at prevailing exposure levels, the lung cancer risk is too small ever to be detected. Some confidence can be acheived in reaching the conclusion that the risk has been adequately reduced in cohorts where fibrosis is not occurring, as it is likely that asbestos doses that are not causing fibrosis will also not be causing lung cancer. Even if there is some small lung cancer risk that persists at today's low exposure levels, there is no doubt that reduction of worker smoking would be orders of magnitude more effective in preventing disease than further reductions in exposure levels.

179/ B.C.A. Exhibit 1, Tab 3, Enterline, P., et al., "Mortality in Relation to Occupational Exposure to the Asbestos Industry," 14 J. Occup. Med. 897-903 (1972); and Tab 5, Enterline, P. and V. Henderson, "Type of Asbestos and Respiratory Cancer in the Asbestos Industry," 27 Arch. Env. Health 312-17 (1973).

180/ B.C.A. Exhibit 7, Tab 7, Weill, H., et al., "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," 120 Am. Rev. Resp. Dis. 343-54, at 352 (1979).

> all asbestos situations. ... [T]here have been cases where higher factors have occurred and obviously there have been cases where lower factors have occurred, say threefold, and so five is just the figure for their particular plant or set of insulation workers. The same as the eleven is the figure for an average smoker. People who smoke more would have a bigger factor. People who smoke just a little one much less.
>
> Q. If we were looking at the number that perhaps came out of your recent study, the friction material plant, instead of five we would be talking about something much lower, I assume?
>
> A. Indeed, yes. We would be talking about near enough to one, because there was no excess in that plant.

It is thus dramatically clear that by far the most effective means of reducing asbestos worker lung cancer is through programs to terminate smoking. As Mr. Peto noted:[174/]

> [V]irtually all lung cancers among asbestos workers who are cigarette smokers, as they are in the general population. ... [I]t's normal for something of the order of one in a hundred lung cancers, either in asbestos workers or in the general population, to be nonsmokers.

Dr. Anderson reviewed for the Commission the dramatic effects of smoking cessation:[175/]

> [W]hat everybody wants to know is if I stop smoking, will it do me any good. ... [I]n the general population. ... [a]t

174/ M. Anderson Tr., Vol. XVI at 53-55.

175/ Id. at 53-54.

> ten years, having stopped, their rate is roughly down at what the nonsmokers is. ... [L]ooking at the [asbestos-exposed] insulators only ... if you stop smoking the risk decreases to one-half to one-third of those who continue to smoke.

As did Dr. Nicholson:[176/]

> [I]f you look at the mortality rate for lung cancer in individuals who stop smoking cigarettes, one finds that it can fall dramatically over ten years to perhaps a quarter or even less than that which it would have been had one continued whatever the cigarette habit would have been previously. ... In other words, after five years of cessation of cigarette smoking, the risk of death from lung cancer decreased to perhaps one-half or one-third what it would have been had one continued to smoke cigarettes. So there is considerable benefit that can be occasioned by cessation of that habit, and certainly even more benefit by never beginning it.

In sum, if any lung cancer risk at all exists at today's low exposure levels, it will be among smokers. For such persons, the risk of lung cancer due to smoking will be many times larger than the risk due to asbestos exposure. It is therefore clear that the most effective means of reducing such disease risk will be smoking cessation programs.

d. Fiber Type.

Although increased lung cancer risks have been found for workers exposed predominantly to each of the three major

176/ W. Nicholson Tr., Vol. XIV at 53-54.

of 8.33 is found, but the figure is not statistically significant.[169]

The Selikoff and Hammond study has made possible the determination that the effects of smoking and asbestos on lung cancer are synergistic, with the combined exposures being riskier than mere addition of the two exposures.[170] This multiplicative effect was noted by a number of witnesses, each of whom relied on the 450 lung cancer deaths in the Hammond and Selikoff data.[171]

169/ Hammond (1979), supra note 149.

In light of recent findings of increased lung cancer among non-smoking wives of smokers (E.C.A. Exhibit 25, Hammond, E.C. and I.J. Selikoff, "Passive Smoking and Lung Cancer with Comments on Two New Papers," 24 Env. Res. 444-52 (1981)), the possibility that these small absolute increases in lung cancers may have been due to the effects of passive smoking in the workplace cannot be discounted.

170/ Dr. McDonald interprets his data as showing an effect between additive and multiplicative:

> The other set of data, based on our most recent findings for Quebec chrysotile miners and millers (J.C. McDonald et al., 1980), appear more than additive but less than multiplicative.

E.C.A. Exhibit 18, Tab 23, McDonald, J. C., "Asbestos-Related Disease: An Epidemiological Review," in Biological Effects of Mineral Fibres 587-601, at 598 (IARC Lyon 1980).

But Dr. Crump found otherwise. E. Crump Tr., Vol. XXVI at 46-48. And, Saracci, reviewing several epidemiology studies of the interaction of asbestos and smoking, concludes that the combined effect is most plausibly multiplicative or synergistic rather than simply additive, although the data are not all consistent. Saracci, R. "Asbestos and Lung Cancer: An Analysis of the Epidemiological Evidence on the Asbestos-Smoking Interaction," 20 Int. J. Cancer 323-31 (1977).

171/ Hammond (1979), supra note 149, at 486-87.

Lung Cancer Relative Risks Among Insulators

	Asbestos exposure	
	Never	Ever
Never smoked	1	5.17
Ever smoked	10.85	53.24

Dr. Selikoff has expressed the same results separately by noting how many lung cancer deaths would have occurred without smoking or asbestos:[172]

Calculated number if there had been no cigarette smoking	44
Calculated number if there had been no asbestos exposure	94
Calculated number if there had been neither cigarette smoking nor asbestos exposure	9

In other words, even with the intense asbestos exposures of this insulator cohort, were it not for their smoking habits, there would have been, not 430, but only 44 lung cancer deaths.[173] And, of course, in cohorts with less exposure than that of the insulators, the risk of lung cancer would be many times less. As Geoffery Berry commented:

> [G]ive [the relative risk found for non-smokers by Selikoff and Hammond] isn't a universal figure that applies to

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172/ E.C.A. Exhibit 30, Tab 11, Selikoff, I. J., "Two Comments on Smoking and the Workplace," 71 A.J.P.H. 92 (1981).

173/ G. Berry Tr., Vol. XI at 21

asbestos workers.[162] A follow-up report continued to reveal no additional lung cancer deaths among non-smokers.[163] An early study of Quebec asbestos mine workers similarly disclosed no lung cancers among 1265 non-smokers despite their generally longer exposure and greater age as compared to the smokers in the cohort.[164]

The only study to correlate smoking habit with cumulative dust exposure was also unable to detect any increased lung cancer risk for non-smoking asbestos workers with less than 300 mppcf-years exposure.[165] Of 262 lung cancer deaths among Dr. McDonald's Canadian miners, only 19 were among non-smokers and there was no excess disease in those subcohorts exposed to less than 300 mppcf-years. Because the reference population was uncorrected for smoking habit, the SMR's for non-smokers are probably understated, but the expected number of deaths would have to be reduced very substantially to raise the SMR's above unity, in the least exposed groups.

162/ Selikoff (1968), supra note 3.

163/ Selikoff, I. J. and E. C. Hammond "Multiple Risk Factors in Environmental Cancer," Environ. Cancer 467-83 (1975).

164/ Braun, H. C. and T. D. Truan, "An Epidemiological Study of Lung Cancer in Asbestos Miners," 17 AMA Archives of Industr. Health 634-53 (1958).

165/ A.C.A. Exhibit 18, Tab 18, McDonald (1980), supra note 10, Table 9, at 19. Dr. McDonald does not clearly state whether his "non-smoking" category included ex-smokers. If it did, the results are even clearer as to the absence of significant risk for non-smokers.

Other studies with information on smoking habits have too few non-smokers to yield significant results. Among Berry and Newhouse's East London asbestos factory workers,[166] only 2 of 67 lung cancer deaths observed were non-smokers -- both heavily exposed females. A prospective follow-up cohort recorded 3 non-smokers among 23 lung cancer deaths -- an insufficient number upon which to base any conclusions.[167] Neurman has reported on the lung cancer experience of a cohort of 1045 asbestos miners and millers in Finland.[168] Of 23 lung cancer deaths after 1967, only one was a non-smoker.

Only when very large cohorts were examined was any apparent association between asbestos and lung cancer found among non-smokers. Even then, numbers were quite small. A larger study by Drs. Selikoff and Hammond of approximately 12,000 insulation workers in the U.S. and Canada produced a subcohort of 891 non-smokers followed at least 20 years since first exposure. Of 94 deaths in this group, 5 were from lung cancer. Compared with the 0.7 lung cancers expected in such a non-smoking cohort of blue collar workers, an SMR

166/ Berry, G., et al., "Combined Effect of Asbestos Exposure and Smoking on Mortality from Lung Cancer in Factory Workers," The Lancet 476-79, at 477 (1972).

167/ Newhouse, M. L. and G. Berry, "Patterns of Mortality in Asbestos Factory Workers in London," 330 Ann. N.Y. Acad. Sci. 53-60, at 59 (1979).

168/ Neurman, L. B., et al., "Combined Effect of Asbestos Exposure and Tobacco Smoking on Finnish Anthophyllite Miners and Millers," 330 Ann. N.Y. Acad. Sci. 491-95, at 494 (1979).

> But I would simply put in the kind of theoretical rider that it still seems possible to me that there might be the odd case of lung cancer occurring, that you'd perhaps never be able to identify, which probably was, to a degree, attributable to the work in which there was no important amount of fibrosis.

Dr Kotin also concluded from the physiologic mechanism evidence that asbestos would not cause lung cancer without the heavy doses required to cause fibrosis.[139] A number of the Commission's witnesses thus agreed with Dr. Weill that asbestos exposures so low that they do not lead to asbestosis are also unlikely to lead to detectable excess lung cancer. As the progress toward eliminating fibrosis among asbestos workers is already being tracked in a number of factories, strong support exists for believing that these cohorts will also not experience excess lung cancer.

c. The Role of Smoking

The evidence clearly shows that even at the high asbestos levels of the past, lung cancer would have been much less prevalent had workers not been smokers. For the future, considerably more progress can be achieved through smoking prevention programs than through efforts to lower asbestos exposure levels even further. As Dr. Irving J. Selikoff has remarked:[140]

139/ K. Kotin Tr., Vol. XXIA at 76-77.

140/ Selikoff, I. J. and D. H. K. Lee, Asbestos and Disease 336 (1978) (emphasis supplied).

> [T]he development of pulmonary carcinoma in persons exposed to asbestos is very closely tied to their indulgence in cigarette smoking; in the absence of smoking, there may be an increase in the prevalence, but it is not significant ... .
>
> It would seem that control of cigarette smoking would have a much greater impact on the incidence of bronchogenic cancer in asbestos workers than further reduction of dust levels ... .

Dr. Enterline agreed in his Commission appearance:[141]

> [T]he advantages to be gained by cutting down on smoking ... are obviously tremendous. Not only in terms of lung cancer, but in terms of heart disease and lots of other kind[s] of cancer that seem to be related to smoking.

The cohorts where smoking specific information exist demonstrate strongly that increases in lung cancer risk have either not been found for non-smokers or are based on quite small numbers. The tenuousness of the association between lung cancer and asbestos exposure, in the absence of smoking, is demonstrated by a review of the few studies with smoking data on individual cohort members. For many years, the epidemiologic evidence provided no data demonstrating any association among non-smokers; all asbestos worker/lung cancer victims were smokers. For example, in his early study of 370 insulation workers in New York and New Jersey, Dr. Selikoff observed no lung cancer deaths among non-smokers, whereas significant increases were found among smoking

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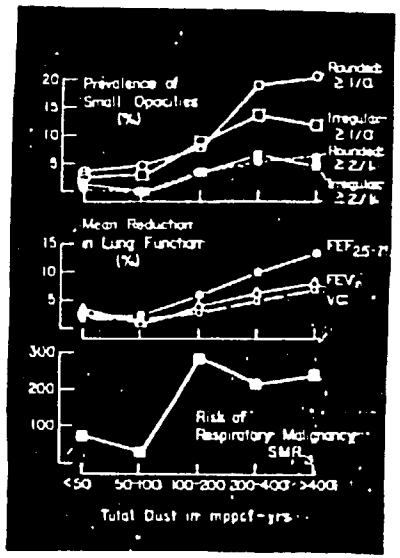
141/ P. Enterline Tr., Vol. VIII at 86.



In light of these data, Dr. Weill told the Commission:[111]

> I think there is awfully good evidence that in fact if we ever get down to a level of exposure that after a suitable period of followup, of working lifetime at least, there was no evidence of asbestosis, no evidence radiographic or any other way of asbestosis, it seems to

[111] H. Weill Tr., Vol. IX at 35. Dr. Weill had previously made the same point in his comments summing up the 1979 Lyon Conference. R.C.A. Exhibit 7, Tab 12, Weill, H., "Asbestos--A Summing Up," in Biological Effects of Mineral Fibers 867-873, at 868-69 (IARC Lyon 1980).

> me that at least in this manufacturing industry one would probably not find excess, detectable excess risk for lung cancer. ... I have heard and perhaps you have heard that standards in the past have been set to prevent asbestosis, with the implication that even if you prevent asbestosis you almost certainly won't prevent the excess risk for lung cancer. I am not sure that's correct. As a matter of fact I suggest that it probably is not.

Dr. Weill noted that the results of the McDonald miner studies supported his thesis:[116]

> [The Canadian miner studies] demonstrated some excess mortality for nonmalignant respiratory disease, and yet no excess in mortality for malignant respiratory disease. Which again would suggest that the nonmalignant disease was occurring at lower levels of exposure than the malignant disease, assuming that followup times and so forth were similar.

Geoffery Berry generally agreed with Dr. Weill:[117]

> [O]n the whole, I would agree with that, because asbestosis ..., is detected in quite a high proportion of the work force, more than developed lung cancer, so that I think it is a reasonable supposition to think that, if one reduced dust levels so that asbestosis didn't occur at all, then one would certainly have mainly controlled lung cancer, but I wouldn't say one had controlled it completely.

As did Dr. Corbett McDonald:[118]

> [I]f you could get the asbestosis down, the lung cancer would no longer be detectable; and, therefore, for practical purposes, I think this is probably true.

[116] H. Weill Tr., Vol. IX at 132.

[117] J. Berry Tr., Vol. XI at 83-84.

[118] C. McDonald Tr., Vol. XII at 41.

3. Lung Cancer: A Problem Related to Smoking.

Although the consensus of Commission witnesses on the risk of lung cancer was not as clear as their consensus with respect to asbestosis, considerable support was presented for concluding that the lung cancer risk is de minimis at current exposure levels. For non-smokers, the risk of lung cancer due to asbestos exposure at current occupational levels, if it exists at all, is well below any occupational risk deserving of societal concern.

a. Studies Not Finding Risk.

The absence of any findings of increased lung cancer risk for workers with less than 300 appcf-years exposure in the McDonald studies, with less than 100 appcf-years exposure in the Weill studies, and the entire cohort in the Ferodo study, all indicate the unlikelihood of significant lung cancer developing at current exposure levels. Although the Enterline study finds increased lung cancer even among workers exposed to less than 125 appcf-years, the numbers are small, and as noted above, interpreted by Dr. Enterline not to be meaningful. As Dr. Davis told the Commission:[134]

> [I]t may be as far as bronchial carcinoma is concerned that we are dealing with very heavy dust doses, and it may be

134. J. Davis Tr., Vol. XXXII at 80.

> that in the good, modern factory somebody starting work now just will not be at risk from bronchial carcinoma at the sort of levels we are talking about.

The possibility that some risk, although non-detectable, may still exist for today's workers will be covered in greater detail in the Chapter IV discussion of risk extrapolation.

b. Less than Effective Doses.

An additional perspective on the question of whether any lung cancer risk remains for today's workers was provided by the observation of Dr. Weill that the epidemiology results indicate that doses which do not cause fibrosis also will not cause lung cancer. Dr. Weill, for example, showed the clear correlation between the levels at which neither fibrosis nor lung function reductions increased lung cancer deaths were detected in his asbestos-cement worker studies:

Geoffery Berry has also analyzed the impact of smoking at Rochdale. For men first employed after 1950, with at least 10 years employment by 1966, very few symptoms were found -- other than in smokers:[131]

Cigarettes per day	Number of Men	Irregularities	Asbestosis Possible	Asbestosis Certified	Small Opacities
Non-smokers	42	0	0	0	5
Smokers					
1- 4	13	0	0	0	0
5-14	48	23	13	10	23
15-24	55	15	5	5	15
25-	13	23	0	0	23
Ex-smokers	24	17	13	8	17

Based on these data, and similar data from the Royal Naval dockyard workers, Berry concluded that "those who smoke have a higher risk of developing asbestosis during life than those who do not smoke," and the asbestosis risk among non-smokers with less than 100 fiber-years exposure is negligible.[132]

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[131] B.C.A. Exhibit 15, Rossiter, C. E. and G. Berry, "The Interaction of Asbestos Exposure and Smoking on Respiratory Health," 4 Bull. Europ. Physiopath. 197-204, at 203 (1978).

[132] Id. at 203. See also Berry, G., et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory," 36 Br. J. Industr. Med. 98-112, at 104 (1979).

(Footnote 132 continued on next page.)

Asbestosis thus is very unlikely to be a significant health effect of future asbestos use. As long as current low exposure levels are maintained, no significant disease will occur.[133]

(Footnote 132 continued from previous page.)

Two small studies of asbestos factory workers also indicate that radiologic lung abnormalities are more prevalent among smoking workers than non-smokers. Weiss, W. and P. A. Theodos, "Pleuropulmonary Disease Among Asbestos Workers in Relation to Smoking and Type of Exposure," 20 J. Occup. Med. 341-45 (1978); and Weiss, W., "Cigarette Smoking, Asbestos, and Pulmonary Fibrosis," 118 Am. Rev. Resp. Dis. 225-29 (1978).

Dr. Weill's cohort exhibited lower pulmonary function values for smokers than for non-smokers at each cumulative exposure level (although there was no apparent synergistic effect between smoking and asbestos). B.C.A. Exhibit 7, Tab 4, Weill (1975), supra note 26, at 96.

[133] Although asbestosis has been reported in persons occupationally exposed to each of the main fiber types, including chrysotile miners and millers and workers in a textile factory employing principally chrysotile, some evidence suggests that asbestosis may occur most frequently among workers exposed to a mixture of fibers. Dr. Weill, for example, found effects on lung function parameters among workers exposed to crocidolite and chrysotile greater than for workers exposed to similar quantities of primarily chrysotile. B.C.A. Exhibit 7, Tab 4, Weill (1975), supra note 26, at 94-97.

Greater mortality from mixed fiber exposure can also be seen in a comparison of the percent of observed deaths due to asbestosis among a number of studied cohorts. The Simpson Report notes:

> These exposed to mixtures have the highest proportional mortality, but the various populations may not be comparable in terms of the quantity of dust inhaled.

B.C.A. Exhibit 28, Tab 5, Simpson Report (1979), supra note 116, Vol. II at 20, referring to Table II, at 68.

Third, evidence of the impact of declining dust levels in decreasing asbestosis is seen in the medical surveillance records of an Australian asbestos manufacturer. James Hardie began medical surveillance of its employees in the late 1960's and found that 1.4 men in every 100 were diagnosed with varying degrees of asbestosis. More recently, only four new cases out of a workforce of 4,000, or 0.1%, have been identified each year.[145/] Dr. Anderson noted during his Commission testimony that the same pattern of "prevalence of fibrosis ... going down" was occurring in Dr. Selikoff's insulator cohort.[146/]

Fourth, even extrapolations from the findings of asbestosis at very high exposure levels in those cohorts not exhibiting thresholds do not predict significant disease. The extrapolations from the Rochdale data indicate only a very small percentage (probably less than 1%) of workers exposed throughout a 30-year career to 2 fibers/cc will develop asbestosis.[147/] Similarly, in the Canadian miner

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145/ Based on these data, Dr. E. Langley, Medical Officer of the Dust Diseases Board, New South Wales, has stated that precautions taken by the asbestos industry to protect its workers had greatly reduced their exposure to asbestos dust, concluding, "I doubt if there will be many more cases, if any, from asbestos factories in Sydney." Brief to the Commission of the Johns-Manville Corp. 3 (Jan. 1981).

146/ M. Anderson Tr., Vol. XVI at 80.

147/ The Rochdale calculations are based on workers exposed at the 2 fiber level for 30 years. As noted in Chapter IV below, the average worker under the current standard would in fact be exposed for a considerably shorter period at considerably less than 2 fibers. No workers would average 2 fibers/cc exposures. Thus, the Rochdale predictions overestimate future asbestosis prevalence, even if their pessimistic dose-response assumptions are correct.

studies, the authors review of the morbidity results led them to conclude that the 1% risk of significant disease would be reached only by workers exposed to more than 100 mppcf-years:[148/]

> Considering all facets of disease--death, roentgenographic changes, pulmonary function changes, and respiratory symptoms--the 1% risk is reached by men in our third dust exposure category (100 to 200 mppcf-yr).

Finally, whatever slim likelihood may persist that asbestosis could continue to be seen at today's exposure levels will be closely related to worker smoking habits. Several studies have investigated the relationship between asbestosis and smoking. The most recent report on Dr. Selikoff's insulator cohort finds an asbestosis death rate among heavy smokers 2.8 times the rate among non-smokers,[149/] leading co-author Dr. E. Cuyler Hammond to comment:[150/]

> It is clear that cigarette smoking greatly increases the risk of an asbestos worker dying from asbestosis or asbestosis combined with pulmonary fibrosis and emphysema resulting from cigarette smoking.

148/ E.C.A. Exhibit 18, Tab 11, McDonald (1974), supra note 18, at 67.

149/ Hammond, E. C., et al., "Asbestos Exposure, Cigarette Smoking and Death Rates," 330 Ann. N. Y. Acad. Sci. 473-90, at 486 (1979).

Dr. Nicholson noted the interaction between smoking and asbestosis in his review of the asbestos epidemiology. E.C.A. Exhibit 19, Tab 9, Nicholson (1981), supra note 16, at 22-23.

150/ Hammond (1979), supra note 149, at 484-85.

of asbestosis severe enough to be implicated in life-shortening disease have occurred only among the much more limited number of workers who had heavy occupational exposures.

Second, a significant demonstration of substantial exposures that did not lead to asbestosis deaths exists in Dr. Weill's New Orleans asbestos-cement worker cohort. As discussed above, no incidence of asbestosis, let alone indication of deaths due to asbestosis, was found in workers with exposures of up to 100 mppcf-years. Although some indication of asbestosis was found in the McDonald and Rochdale studies at such exposure levels, the amount of such disease was minimal. Of 92 men at Rochdale with less than 30 mppcf-years exposure, only one had verified asbestosis, and only three had possible asbestosis.[143] In the Canadian miner study, 36 of the 42 pneumoconiosis deaths were from

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(Footnote 142 continued from previous page.)

Although questions might be raised about whether the United States in fact records all asbestosis deaths, as Dr. Enterline testified, any such effect would quite likely be minimal:

> [W]e have a long history of reporting of asbestosis cases, and it's a well-recognized cause of death in the national vital statistics system in the United States, and certainly in the system in Canada.
>
> So, we know how much asbestosis there is, we know how many people die of it, and that should tell us something about the size of the population that's producing these asbestosis deaths.

P. Enterline Tr., Vol. VIII at 9.

143/ R.C.A. Exhibit 13, Tab 8, Berry (1979), supra note 61.

workers with more than 20 years employment in the mines, and the excess in deaths in the lowest exposure subcohort was based on a small number (five) of deaths.[144]

144/ R.C.A. Exhibit 18, Tab 18, McDonald (1986), supra note 10, at Tables 6, 8, pp. 17, 19. Given the small numbers, minor errors in classification of either disease or exposure could account for the apparent increase.

The absence of a demonstrated threshold in the Canadian and Rochdale data led the Simpson Report to conclude cautiously:

> The data in Canadian chrysotile miners and millers suggest a substantial increment in lung function impairment and of the prevalence of shortness of breath at doses of dust at the lower end of the range (less than 100 million particles per cubic foot) and like Rochdale gives no support for the existence of a threshold within the industrial range of dose. Another study [Weill, et al.] does not agree and suggests the existence of a safe threshold at a cumulative dose of 200 fibre years per cc. The present authors come down in favour of a dose response relationship without a threshold for chrysotile within the range experienced in industry.

R.C.A. Exhibit 28, Tab 5, Simpson Report, supra note 116, Vol. II at 38.

But, Dr. McDonald testified to his belief that an asbestosis threshold probably exists:

> [Y]ou've got to have fibrosis beyond a certain level before it could conceivably do anything to you, because the reserve power of the lung is fairly substantial, and it seems to me you've got to make some significant reduction in your lung capacity before it could really kill or lead to increased probability of death.
>
> Having said that, we still find a [illegible] relationship.

C. McDonald Tr., Vol. XII at 34.

significant future occupational health risk exists.[138] As the Simpson Report noted:[139]

> '[D]espite the high proportion of individuals, especially in urban areas, with asbestos fibres in their lungs, there has been no general reporting of asbestosis in the general public. This suggest[s] that there may be a threshold level below which asbestosis is not detectable.

138/ Radiological changes have been reported among wives and family members of asbestos and insulation production workers in the study by Dr. Anderson described above. However, as the Simpson Report notes, such radiological changes do not equate to asbestosis:

> While the illustrations of the relatively small number of cases of advanced disease in these papers are impressive, the significance of minor radiological changes when reported, as is the case here, in the absence of other parameters and of controls, should, as Bechlake has pointed out, be accepted with caution.

R.C.A. Exhibit 28, Tab 5, Simpson Report (1979), supra note 116, Vol. II at 26.

139/ R.C.A. Exhibit 28, Tab 5, Simpson Report (1979), supra note 116, Vol. II at 26.

Dr. Fischetosis similarly noted:

> I think that asbestosis is not a public health problem. ... I believe that asbestosis is almost exclusively limited to occupationally-exposed populations. That's clinical asbestosis.
>
> It wouldn't surprise me if you removed the lungs of everyone in this room and looked quite carefully, you might find some asbestos fibers and you might find a little bit of scarring, but this will have no effect on your health or your life expectancy.

M. Fischetosis Tr., Vol. XXIV at 71.

In his Commission testimony Dr. Nicholson agreed:[140]

> There really is like a threshold for mortality from asbestosis.

As did Dr. Acheson:[141]

> [I]t is known that a very large proportion of the urban population of industrialized Europe and North America have substantial numbers of asbestos fibers in their lungs, they do not at autopsy have evidence of asbestosis.
>
> So it may very well be that there is a threshold in respect of asbestosis.

The great unlikelihood of asbestosis deaths in the future is evidenced in a number of data.

First, as Dr. Enterline testified, very few asbestosis deaths have occurred despite the millions of workers exposed. In the United States since 1950, fewer than 60 asbestosis deaths each year have been reported, with an upward trend through 1972 and a leveling off since.[142] Clinical cases

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140/ N. Nicholson Tr., Vol. XIV at 66.

141/ D. Acheson Tr., Vol. XIX at 15. See also R. Lemen Tr., Vol XVII at 17.

142/ R.C.A. Exhibit 2, Enterline, P.E., "Proportion of Cancer Due to Exposure to Asbestos," Table 1 (1981).

Dr. Enterline had previously noted the inclusion of almost all U.S. asbestosis deaths among these workers in primary asbestos manufacturing operations. His 1967 study of 21,755 men at asbestos product plants included 30 asbestosis deaths during the 13-year period 1950-1963, accounting for more than one-fourth of all 160 U.S. asbestosis deaths in these years. Enterline, P. E., and M. A. Kendrick, "Asbestos-Dust Exposures at Various Levels and Mortality," 15 Arch. Environ. Health 181-86, at 184 (1967).

(Footnote 142 continued on next page.)

overriding finding relevant to all asbestos-related disease is the clear evidence of a dose-response relationship. As Dr. Becklake noted in 1976:[137]

> Despite the shortcomings of the methods for estimating dose, a relationship of estimated dose to response has, nevertheless, been a consistent finding. This consistency has been true with exposures encountered in mining, manufacturing, and secondary usage, including removal of old asbestos insulation. Furthermore, it applied to all of the responses examined, i.e., lung fibrosis, as reflected by symptoms, lung function and radiographic changes, and lung cancer, as reflected in mortality statistics.

As attention has focused even more clearly on determining dose-response relationships in the subsequent five years, that observation has been many times reaffirmed. There is no doubt that the unfortunate health results of the past, when exposures were high and often uncontrolled, will not be repeated with future controlled use of asbestos. The crucial question is thus not whether the adverse effects of asbestos have been reduced in the past thirty years -- the answer to that question has already been clearly answered "yes." Rather, the crucial question is whether the medical evidence demonstrates that the risks have been reduced to an extent that continued use of asbestos is justified. As the following

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137/ B.C.A. Exhibit 29, Tab F, Becklake, M., "State of the Art: Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," 114 Am. Rev. Resp. Dis. 187-227, at 197 (1976).

synthesis describes, the answer to that question is also yes.

1. Asbestosis: A Problem of the Past.

Although a significant cause of disability and death in heavily exposed cohorts in the past, asbestosis will rarely, if ever, occur in the future. Significant support for the conclusion that asbestosis is no longer an occupational health problem at current exposure levels was forthcoming in the testimony and studies of the epidemiologists who appeared before the Commission.

The consensus opinion of the Commission's expert witnesses was that a threshold level of exposure exists below which asbestosis is unlikely to occur and that current exposure levels are below that threshold. Evidence of the absence of asbestosis in subcohorts of workers exposed at lower -- although much higher than current -- exposures, demonstrates the unlikelihood that clinical asbestosis will be a problem in the future. Even the predictions of asbestosis based on conservative risk extrapolation from those studies which did not exhibit thresholds make it clear no

Further confirmation of the absence of serious disease among this group comes from the observation of Dr. Anderson and his co-authors that the "household contacts considered themselves to be in good health" and "the clinical examinations generally confirmed this."[131/]

During his testimony before the Commission, Dr. Anderson described the "active contamination" that characterized the homes of these amosite workers. As he explained, asbestos was brought into the homes "probably on workclothes, on shoes, in the automobiles and workclothes that these people may have worn."[132/] Wives, among whom the greatest percentage of abnormalities were found, were especially severely exposed when "they would first shake the clothes out to get the majority of the dust off, then depending on the earlier years, many of them wouldn't have electric washing machines and so it would be a hand washing machine, hand wringer, and then external drying outside."[133/] Dr. Nicholson, who worked with Dr. Anderson at Mount Sinai, testified that he was unable to quantitate these family member exposures, but characterized them as "fairly severe circumstances of severe environmental contamination."[134/] As was demonstrated by

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131/ E.C.A. Exhibit 13, Tab 7, Anderson (1979), supra note 128, at 305.

132/ H. Anderson Tr., Vol. XVI at 104.

133/ Id. at 104.

134/ V. Nicholson Tr., Vol. XVI at 33. In more recent measurements in the homes of Newfoundland miners, Dr. Nicholson has measured levels from 30 to 9000 s/cm³. Id. at 128, 137.

the settled amosite dust in rafters found by Dr. Nicholson thirty years later, Dr. Anderson noted, "it is a possibility that everytime you vacuumed or dusted, you would reentrain the material."[135/] Thus, he concluded, this "unusual situation" would be "difficult to extrapolate" to any other setting.[136/]

> It is not our intention to use this [study] to extrapolate to all family members of asbestos workers or factories. This is a unique experience in this particular plant.

C. Synthesis of the Asbestos Epidemiology.

Throughout the Commission's hearings, considerable attention was paid to reconciling apparent differences and inconsistencies among the epidemiology studies. As these discussions disclosed, many of the apparent differences can be explained through close examination of the factual setting of each cohort and the varying methodologies and analysis techniques used by the investigators. The following section seeks to synthesize the epidemiologic findings to determine the health parameters relevant to development of appropriate asbestos regulation.

Before proceeding to review separately the accumulated evidence on asbestosis, lung cancer and mesothelioma, one

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135/ H. Anderson Tr., Vol. XVI at 105.

136/ Id. at 63, 64.

duration of employment, greatly limits its usefulness for determining dose-response relationships. As Geoffery Berry commented to the Commission:[126/]

> [T]he main thing that Dr. Selikoff has shown is a number of work situations where there has been increased mortality, often very much increased. He has shown the smoking/asbestos interaction that we've been talking about earlier, but he has given very little data on actual dose in fiber terms. Therefore, it's difficult to use it in any quantitative sense for deciding on what dust levels might be proven to aim for.

10. The Anderson Family Member Study.

A companion study to the amosite insulation plant in Paterson, New Jersey, also by the Selikoff Mount Sinai group, reviews the x-ray findings among family members who lived with the 1,664 workers at the plant. Dr. Anderson and his colleagues have yet to complete their mortality study,[127/] but have published results of clinical examinations of 679 persons who lived with workers during their employment (between 1941 and 1954) at the plant and who were willing to

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126/ G. Berry Tr., Vol. XI at 50.

127/ Dr. Anderson testified that four mesotheliomas have been identified among the 3106 identified household contacts. He explained that an additional mesothelioma "both mentioned in B.C.A. Exhibit 23, Tab 7, Anderson (1979), infra note 128, at 387, should not be included, as this person also had occupational exposure. H. Anderson Tr., Vol. XVI at 33, 67.

visit the clinic for testing.[128/] During the Commission hearing Dr. Anderson presented data on an additional 97 persons who have subsequently been examined.

The family members were all examined at least twenty years after their initial exposure had begun. For two-thirds more than thirty years had elapsed. Pleural changes were the predominant finding of the study.[129/] Dr. Anderson noted for the Commission the minimal parenchymal abnormalities he found:[130/]

> [T]here were very few of the family members that had parenchymal abnormalities or these irregular opacities in an advanced or a high-grade. The two over two would be considered by most individuals to be moderate parenchymal asbestosis, where the one over zero is usually considered a borderline. As you can see, . . . zero one, one over zero group, accounts for the majority of individuals who were in the comparison group that had anything but an absolutely perfectly normal film.

128/ B.C.A. Exhibit 23: Tab 2, Anderson, H. A., et al., "Household-Contact Asbestos Neoplastic Risk," 271 Ann. N.Y. Acad. Sci. 311-23 (1976) (covering 326 family members); Tab 7, Anderson, H. A., et al., "Asbestosis Among Household Contacts of Asbestos Factory Workers," 330 Ann. N.Y. Acad. Sci. 387-99 (1979) (covering 679 family members); Tab 8, Anderson, H. A. and I. J. Selikoff, "Asbestos-Associated Radiographic Changes Among Household Contacts of Amosite Asbestos Workers," 253-73 in Prager, L., Ed., Induced Disease: Drug, Irradiation, Herpetics (1979) (covering 685 family members); Tab 9, Anderson, H. A., et al., "Household Exposure to Asbestos and Risk of Subsequent Disease," [illegible] in Lemen and Dement, Eds., Dusts and Disease (1979) (covering 679 family members).

129/ B.C.A. Exhibit 23, Tab 7, Anderson (1979), supra note [illegible].

130/ H. Anderson Tr., Vol. XVI at 39.

traced through 1962.[119] These workers, plus an additional 690 men in the same locals who joined the union between 1943 and 1962 were subsequently followed through the end of 1968 (by which time 404 had died).[120] The most recent report covers all 17,800 members of the union throughout the U.S. and Canada as of January 1, 1967 (including the survivors as of that date from the original 1964 cohort). These insulators have been followed through the end of 1976.[121]

From the union records upon which the cohort was compiled, Dr. Selikoff has been able to determine for each man his birth date, the date he joined the union, and employment status of January 1, 1967. Additional information on smoking habits was compiled through use of a questionnaire. The information on this cohort does not, unlike the other cohorts described above, include either the duration of employment or duration of exposure. As Dr. Nicholson, who has worked for many years with Dr. Selikoff, noted:[122]

> We have no data on the effects of differing exposures.

Dr. Selikoff has described the work of these insulators:[123]

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[119] Selikoff (1964), supra note 3.

[120] Seliköff, I. J., et al., "Mortality Experience of Asbestos Insulation Workers 1943-1968," (1976).

[121] Selikoff (1979), supra note 2.

[122] W. Nicholson Tr., p. 6, at 29.

[123] Selikoff (1979), supra note 2, at [illegible]

> Members of this union are insulation workers, primarily employed in the building trades doing construction insulation work but also employed as insulation workers in refineries, industrial plants, shipyards and powerhouse construction and repair. Much of their work is in the open but sometimes, as in shipyards, in rather tight quarters. The men generally work in all parts of the trade, with few specialists at one or another part of it. Conditions at work vary from job to job and from company to company, with fewer than 10% of the men remaining with one company during their working lifetimes.

Based on the 2271 deaths as of year-end 1976, the study found increased mortality for all causes (RR of 1.37), all cancers (RR of 3.11-BE, 2.86-DC),[124] and lung cancer (RR of 4.69-BE, 4.06-DC), and modest increases in gastrointestinal cancers. Based on best evidence, there had been 175 mesotheliomas deaths (63 pleural and 112 peritoneal).[125]

The large size of this cohort, the extent of smoking information that has been collected on its members as discussed further below, and the careful attention that has been paid to determination of cause of death have made it a unique study for examining the interaction of asbestos and smoking and for exploring the possibility of additional asbestos-related diseases. On the other hand, the absence of any exposure information, even information on their

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[124] Results are reported for cause of death both as recorded on death certificates (DC) and from additional best evidence data (BE).

[125] Id., Table 12 at 103.

exposures.[114] Dr. Anderson, who has studied family members of this plant's employees, noted that during the war this was a "very dusty, dirty factory."[115]

In light of the absence of any exposure data for this cohort, the Simpson Report concluded:[116]

> Interpretation of this particular study is difficult for a number of reasons including the fact that information was presented only for the duration of employment and not for dust concentrations.

Even without exposure numbers, however, it is apparent that an unusually high incidence of disease was found. Possible explanations for the unusual experience of this cohort beyond the likelihood of unusually intense wartime exposures include unusual effects of amosite, the only fiber used, and unusual adverse impacts of short-term, high intensity peak

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114/ NIOSH, "Criteria for a Recommended Standard: Occupational Exposure to Asbestos," Tables VIII: and XIX (1972).

115/ H. Anderson Tr., Vol. XVI at 24. Dr Anderson further described conditions by reference to a story well-known among the plant's workers:

> [O]ne of their basic anecdotal stories was that this was a mixed population of blacks and whites, that you could tell who was who when you went in in the morning, but when you came out at night, everybody looked the same.
>
> Q. White, that is?
>
> A. Wh . Unlike the coal mines where it would be the other way around.

116/ Id. at 104.

116a/ B.C.A. Exhibit 28, Tab 3, British Advisory Committee, Asbestos (hereinafter cited as "Simpson Report") Vol. II at 13 (1979).

exposures. As an amosite factory, it is quite likely that an unusually high percentage of the fibers may have been longer than 5 µm. As Dr. Nicholson has noted, 29% of fibers in amosite blanket fabrication were of such length, as compared to much lower percentages for mixing cement (1.2%) and removal of pipe covering (5.9%).[117]

Although the Seidman study is limited in its applicability to determining dose-response relationships by the unavailability of monitored exposures, it has provided a unique data base for determining average latency periods because it is predominantly a cohort of short-term exposures.[118]

9. The Selikoff Insulation Worker Studies.

Since the early 1960's, Dr. Irving J. Selikoff of Mount Sinai Hospital, has been following asbestos insulators, identified by being members of the AFL-CIO International Association of Heat and Frost Insulators and Asbestos Workers. Dr. Selikoff's cohort has grown considerably over the years. His initial 1964 report was based on 632 New York and New Jersey workers who entered the trade before 1943 and were

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117/ B.C.A. Exhibit 19, Tab 8, Nicholson, W.J., "Case Study 1: Asbestos-The TLV Approach," 271 Ann. N.Y. Acad. Sci. 132-49, at 138 (1977). See also B.C.A. Exhibit 19, Tab 9, Nicholson (1981), supra note 56, at 7, noting that in amosite-aerosols 30% of the fibers are often longer than 5 µm.

118/ Dr. Nicholson discussed for the Commission the study's unique value for this purpose. N. Nicholson Tr., Vol. XIX at 33-36.

A number of questions thus arise about the unusually high results found in Dr. Dement's study of a textile plant. Although no one factor appears significant enough in itself to explain why these results fall outside the experience found in other studies where the investigators have attempted to quantify exposures, a combination of factors does appear important. With respect to choice of a particle/fiber conversion factor, interepretation of the exposure measurements available, choice of a control population, and exclusion of the black male subcohort, each assumption made by Dr. Dement served to exaggerate the risk.

6. The Paterson Amosite Insulation Factory Study.

Apparently falling outside of the experiences recorded in the other cohort epidemiology studies are the results of a study conducted by the Mount Sinai Hospital investigators of World War II workers in a Paterson, New Jersey factory.[110/]

The team led by Herbert Seidman studied 933 men first employed between June 1941 and December 1945 in the production of amosite asbestos insulation for U.S. Navy ships. From this group, 113 men were excluded from the analysis.[111/] Duration of employment in this plant among the remaining 820 men broke down in the following manner:

110/ Seidman (1979), supra note 1.

111/ These excluded were 20 with prior asbestos experience, 14 with asbestos experience within 5 years of leaving the plant, 41 who died within 5 years of leaving the plant, and 38 lost to follow-up.

Less than 1 month	61
One month	90
Two months	83
Three-five months	148
Six-eleven months	125
One year or more	313

Of the 820 men, 328 had died by the end of 1977, and 89 lung cancers were observed according to death certificate information, 93 based on best evidence, whereas 33.1 were expected based on New Jersey rates.[112/]

Four mesotheliomas were reported on death certificates in contrast to 14 (7 pleural and 7 peritoneal) which were identified by autopsy and other tissue diagnoses. Thirty deaths were attributed to asbestosis based on best evidence. All but one of the 44 asbestosis and mesothelioma deaths were among six month plus workers. 31 were among two year plus workers. Lung cancer excesses were found for men with as short as one month or less exposure.

Monitored exposure measurements in this factory do not exist, but there are various indications that exposures were quite high. The authors report that measured exposures in a similar plant in 1971, where dust extraction methods were "likely to have been better" showed counts averaging 23 fibers/cc.[113/] Review of the NIOSH data from this similar plant indicates the likelihood of far greater than 23 fibers/cc

112/ Id. at Table 7A, p. 73.

113/ Id. at 62.

Nonetheless, based on his exposure estimates and on the data for white males who worked in the plant for more than six months, Dr. Dement concludes that elevated risks are found for all causes of death and lung cancer even among the 65 deceased workers exposed to less than a 40 fiber-years cumulative dose. The finding, however, is based on a small number (eight) of lung cancer deaths and is subject to considerable question given any problems with the assumed exposure data.

Significant questions about Dr. Dement's observations are also raised by the fact that the lung cancer rate in the county where the plant is located is 75% higher than the U.S. rate for white males. The lung cancers reported among the lowest exposure subcohort are not in excess when compared to local rates. Dr. Enterline emphasized this problem:[108]/

> I personally think that the rate should be compared with local expectations, not national or regional expectations, and that the factors that may perhaps influence the high rate of lung cancer in Charleston are not related to the presence of this particular plant.

As did Dr. Acheson:[109]/

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[108]/ P. Enterline Tr., Vol. VIII at 37.

[109]/ D. Acheson Tr., Vol. XIX at 45-46.

Dr. Dement reported during his testimony that he was able to determine that none of the non-lung cancer victims on whom prior employment data were collected in the 1960's by the Public Health Service had

(Footnote 109 continued on next page.)

If he took the local counties as his standard, most of the excess SMR would disappear.

What are the possibilities? One is that there is a factor operating in South Carolina which we don't understand, in those counties, which is a cause of lung cancer, which is totally unknown, and these men in this factory have been exposed to it - something to do with the environment of those counties. If that is the case, more than the fair share of the effect is being attributed to asbestos, because he is not using the local male population as his standard.

Another possibility, as has been pointed out, is that in these counties during the war there was a big shipbuilding industry. The excess lung cancer rate in those counties, which is seventy-five percent above the national average for the U.S., is due to work in the shipyards, and due to exposure to asbestos in the shipyards, and probably due to a mixture of fiber types.

If that's the case, one wonders how many of the men in the cohort were also ex-shipyard workers. Twenty thousand men, I understand from the paper, in those counties are known to have worked in the shipyards. It seems very unlikely to me that some proportion of the seven hundred and fifty in Dr. Dement's cohort who, from the material in this study, were old enough to work in the war - indeed he refers to deaths from 1940 onwards - didn't also work in the shipyards.

(Footnote 109 continued from previous page.)

worked in the shipyards. He could not, however, report what percentage of the total cohort on whom such data existed had worked in the shipyards. Dr. Mustard asked for such data, and Dr. Dement promised to supply it to the Commission. J. Dement Tr., Vol. XXXIII at 138-39. In addition, even if a significant number of textile workers did not work in the shipyards, Dr. Acheson's first possibility (an unknown factor causing lung cancer in the area) would remain.

> company, in the 1930's, and it was a very impressive slide. It showed things were very, very dusty and you couldn't see things very well. And then showed a chart, and it's in his paper, that he attributed ... he assessed that that average exposure at that time in that job was five fibers per ml ... or six perhaps. I don't want to be unfair.
>
> Well, that's a little hard to believe. It's possible, I suppose, but a little hard to believe.

Dr. Alison McDonald, who has studied the same textile plant, agreed:[105]

> The levels, just to us are extremely perplexing because we have infinitely higher apparent measurements in the mines and in the mills than they have found in the factory, and this is puzzling because in looking at the old pictures of the process in the factory ... the room is covered in a cloud and people reported you couldn't see from one end to the other, and yet here we have what is apparently, compared with the milling of chrysotile, a set of very low measurements.

Dr. Dement's exposure results appear particularly out of line when compared with the exposures determined in an extensive 200-sample monitoring program at the plant in 1971. Those results are consistently higher. For example, this U.S. Public Health Service survey at this plant found a 7.1 fibers/cc average for carding, whereas Dr. Dement's model predicts lower levels for all years in the plant back though 1935. Similarly, the 1971 exposure level for mule

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105/ A. D. McDonald Tr., Vol. XXXI at 23.

spinning was found to average 14.1 fibers/cc, while Dr. Dement's model posits a 4.6 fibers/cc value back through 1930.[106]

Dr. Dement does not present any smoking results for the cohort. He reports only that for the less than half of the workers on whom they have smoking data, no apparent discrepancy between their habits and national averages exists.

For the 191 deaths that have occurred in the cohort, increases over expected are found for all deaths, malignant neoplasms, and non-malignant respiratory diseases. No significant excess was found for neoplasms of the digestive system. Only one mesotheliома was found.

Not included in Dr. Dement's Cardiff presentation are the strikingly different disease results among the black male workers in the same plant. Although tracing has not been as complete for these workers as for the white males, Dr. Dement has found 114 deaths among the cohort and has determined cause of death for all but 28. For these workers, there is no overall increased death rate, nor any increased deaths due to all cancers, nor cancer of the respiratory tract.[107]

106/ Division of Field Studies and Clinical Investigation, United States Public Health Service Survey of Asbestos Plant #311, at 1, 2 (Jan. 1971); see also NIOSH, "Criteria for a Recommended Standard: Occupational Exposure to Asbestos," Table XII-1 Plant K (1972).

107/ Dement Dissertation, supra note 99, at 169-73.

that the best correlations existed for the highest measurements. As Dr. Crump explained to the Commission, it is exactly those higher measurements that are the most important to risk assessment because it is at those higher levels that most of the cohort's exposure occurred:[18]

> [T]he data which are most pertinent ... are the data where the dust counts and the particle counts were higher. So these are the data which would probably be most valuable in estimating those old exposures.
>
> These are also the ones where one gets correlations in the neighborhood of point seven five, point seven six.

In analyzing the converted exposure data, it is important to keep in mind that the apparently wide range of conversion factors among individual paired samples (as was exemplified by the shot-gun graph of the original Gibbs-Lachance data,

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18/ E. Crump Tr., Vol. XXVI at 18-19.

Conversely, low measurements correlate poorly. Dr. Lippmann noted the measurement imprecision and variability that greatly confuse conversion of low exposures:

> [W]hen you get the very low concentrations with the midget impinger, ... your number of particles actually observed, on which you base your midget impinger count, are very, very small. So there's a big error in there.
>
> You also have a problem on the scanning fiber side for the low concentrations, ... in the low end of the scale, the number of fibers that you observe per field will be below those where statistics have told us that we will have ... unless we make very large numbers ...

G. Gibbs Tr., Vol. XXIX at 41.

which was referred to during the hearings on several occasions)[19] does not by itself imply that meaningful correlations cannot be derived. As Dr. Dement testified, were the paired samples from his Charleston textile plant graphed, they would probably demonstrate the same, apparently random, shot-gun pattern. But, in fact, as he testified, statistical analyses of the data led him to conclude that a meaningful conversion factor could be determined.[20]

It is also important that the individual paired sample data displayed on the Gibbs-Lachance graph not be confused with the comparisons of annual fiber and particle average from the Rochdale data. Although use of conversion factors to determine pre-1969 exposures at Rochdale is clearly necessary, the details of such conversions have not been published. The only data that exist are the one table that shows annual averages in 1969 and 1961.[21] Although the range of individual conversion factors shown in that table (from 20 to 60) is not as wide as that found in the Gibbs-Lachance paired sample data, the comparison is not meaningful.

19/ R.C.A. Exhibit 61, Tab 4, Gibbs (1974), supra note 15, at 20.

20/ J. Dement Tr., Vol. XXXII at 113-11 . It is important, however, to keep in mind that Dr. Dement's conversion was done on the basis of an overall index, rather than the site by site conversions determined by Dr. McDonald, as described in note 32, supra.

21/ R.C.A. Exhibit 37, Tab 1, Peto, J., et al., "A Mortality Study Among Workers in an English Asbestos Factory," 14 Br. J. Industr. Med. 169-73, at 172 (1977), reprinted from BOHS, "Hygiene Standards for Chrysotile Asbestos Dust," 11 Ann. Occup. Hyg. 47-49, 57 (1968).

> Between 1969 and 1976, there were 10,203 particle counts and, between 1969 and 1976, 11,819 fibre counts. Unfortunately with little overlap. Each mining company was divided into main areas and subareas, and fibre: dust ratios (ranging from 0.3 to 30) were calculated for each one. Particular attention was given to side-by-side measurements, where recorded, and to characteristic changes in patterns of ratios as dust concentrations have declined over the past 25 years. We cannot claim precision or certainty for our estimates, only that the available data - more plentiful in this industry than most others - were used to the full.

Based upon these conversions, Dr. McDonald and his colleagues have once again calculated the dose-response relationship in their study. As Dr. Gibbs noted:[32]

> [T]he potential existed for totally changing the response relationship.

But, in fact, the result was quite similar to what had been found previously based on particle counts, providing strong

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(Footnote 31 continued from previous page.)

> Now, this doesn't mean that there wasn't a lot of variation with a particular area in terms of the ratios of membrane filter to midget impinger, but they were quite a bit less than we saw overall. ...
>
> So we took all the data that existed in the industry to arrive at an estimate for particular work areas what the ratio would be. ...
>
> These figures were then used from each of these locations. We then linked them with each job and the McDonald analyses, the case control studies that be described, the fiber assessments were then done using these figures. So these figures were derived by location, not a single conversion factor.

G. Gibbs Tr., Vol. XXIX at 44-45.

32/ G. Gibbs Tr., Vol. XXIX at 45.

support for a conclusion that the converted fiber counts are a meaningful expression of the relevant doses.

Dr. Acheson expressed his belief that these Canadian conversions were useful for risk assessment.[34]

> I don't think it's right to say that Quebec [conversion] is necessarily worse than the others. I think they are all difficult and you have to make the decision whether to use them or try and arrive at the standard on purely pragmatic grounds without taking into account any attempt to use the dose-response relationships.

Dr. McDonald's finding that meaningful dose-response relationships could be found -- with particle measurements converted to fibers -- was not unexpected in light of the surprisingly strong correlation between particle and fiber counts that were demonstrated in the paired samples collected in the mines and mills. Although an initial look at this data by Dr. Gibbs and Maurice Lachance had led them to be somewhat pessimistic about converting,[35] additional analysis of the more than 600 paired samples by M. Dagbert showed surprisingly meaningful correlations.[36] As in the initial review of the data by Gibbs and Lachance,[37] Dagbert found

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34/ D. Acheson Tr., Vol. XIX at 101.

35/ See B.C.A. Exhibit 41, Tab 4, Gibbs, G. and M. Lachance, "Dust-Fiber Relationships in the Quebec Chrysotile Industry," 31 Arch. Env. Health 69-71 (1976).

36/ See M. Crump Tr., Vol. CCCI at 13-19.

37/ B.C.A. Exhibit 41, Tab 4, Gibbs, G. and M. Lachance, "Dust-Fiber Relationships in the Quebec Chrysotile Industry," 31 Arch. Env. Health 69-71 (1976).

First, only the study containing quantitative exposure information does not rely on particle-to-fiber conversions, that of a Ferodo plant by Newhouse and Berry (where old exposure levels were determined by simulation of historical conditions that were monitored by modern methods). Although some witnesses referred to various studies as if they did not rely on such conversions for their exposure estimates,[12] in fact such conversions were necessary.

Second, although there is no doubt that considerable variation has been found among conversion factors in different settings, that variation -- which is totally as would be expected -- does not of and by itself demonstrate that meaningful information cannot be derived through systematic attempts to convert particle counts to fiber measurements.

The meaningfulness of a carefully derived index was exemplified in a number of ways, perhaps most significantly,

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12. Some witnesses referred, for example, to the Rochdale data as being based on fiber counts, because the published studies in the last 15 years express results in such terms. See, e.g., N. Finkelstein Tr., Vol. XXX at 45-47. But, as Dr. Gibbs noted:

> [T]he counts made [at Rochdale] prior to the early sixties were essentially in particle counts, and a conversion was made from particle counts into fiber counts in the 1960's, based on some parallel measurements that were available.
>
> So even in the data that we have considered to be fibrous information, first the membrane filter technique was not used to measure the concentration, and secondly, some sort of conversion was necessary in order to assess what the exposure would have been in fiber terms.

J. Gibbs Tr., Vol. XXIX at 39.

in the work on the chrysotile miners. Although previously having argued against attempts to convert his exposure determinations to fibers,[13] Dr. McDonald has recently published the preliminary results of such a conversion project. As he describes it:[14]

13/ See, e.g., R.C.A. Exhibit 18, Tab 11, McDonald, J. C., et al., "The Health of Chrysotile Asbestos Mine and Mill Workers of Quebec," in Ann. Environ. Health 61-68, at 68 (1974).

14/ R.C.A. Exhibit 18, Tab 31, McDonald, J. C., et al., "Chrysotile Fibre Concentration and Lung Cancer Mortality: A Preliminary Report," in Biological Effects of Mineral Fibres 811-17, at 813 (Lyon, IARC 1980).

The results were:

> The mean accumulated dust exposure for the 264 cases and for 488 controls so far completed was 248.8 mppcf/yr., and the mean accumulated fibre exposure 905.0 fibres/ml/yr. Thus, the average conversion factor was 3.64.

Id. at 814.

Dr. Gibbs described the methodology in more detail:

> What we did do was to take subsequently all the measurements which the industry had ever made, prior to 1977 or up [to] the end of 1977, both with the midget impinger and with the membrane filter, plus the McGill data, the figures we had, and we looked to see what the mean concentrations were, or median concentrations within each work area ... by midget impinger and by membrane filter ... for the areas where we had measurements by both techniques.
>
> Then we looked at what the side-by-side measurements in these areas - if they existed - gave us, and we used these ratios, the median concentration, for example, in the bagging area by midget impinger to the median concentration in the bagging area by membrane filter, to convert the concentration in particle term in that area.

[Footnote 12 continued on next page.]

contribution of peak bursts of exposure. In making predictions for future groups in settings where control measures are in force, it should be the historical data from those cohorts with long term, low dose, without peak burst exposures, that should provide the most meaningful guidance.

5) The age distribution of the cohort.

A consideration parallel to that of whether the pattern of exposure of the cohort is similar to the pattern of the group for which predictions are to be made is whether the age distribution of the two groups is similar. To the extent disease incidence may depend on age, such factors affect the assessment's results.

6) The size of the cohort.

Large cohorts are less likely to be subject to random variation problems than smaller cohorts. More importantly, with a large cohort it may be possible to select subcohorts in order to achieve much better matches between the exposure patterns, age distributions and smoking habits of the studied workers and the population for which risks are being assessed. It is thus possible for a large cohort study that in general ranks low on the first five criteria to include subcohorts that rank high and are appropriate for risk assessment purposes.

In sum, determination of the data upon which risk assessments should be made involves both taking advantage of all relevant evidence, and, at the same time, discriminating among the evidence in order to attempt to have the best quality data derived from settings as much like those for which predictions are being made as possible. Although the process necessarily involves some uncertainty in viewing the data critically, it is possible to assess asbestos risks meaningfully in order to place them in context with other occupational risks.

3. Choice of Conversion Factors.

As noted previously, much of the historical exposure information is expressed in particle counts. All data, for example, from the chrysotile mines and mills prior to 1969, from the Louisiana asbestos-cement plants prior to 1973 from the Toronto asbestos-cement plant prior to 1970, from the Charleston textile plant prior to 1965, and from the Rochdale textile plant prior to 1961 were recorded in terms of million particles per cubic foot (mppcf). Such particle counts need to be converted to fibers if they are to be used to determine quantitative risks for current settings monitored by the membrane filter method. Considerable discussion before the Commission focused on the advisability and usability of such conversions. Several important facts emerged, each of which argues in favor of reliance on such conversions.

have found more disease with the same cumulative exposures among maintenance men than production workers. Both investigators have concluded that the intermittent and peak nature of maintenance workers' exposure probably account for their greater disease. Dr. Enterline found increased excess lung cancer risks among maintenance workers as compared to production workers at each level of estimated cumulative exposures:[28]

(Footnote 27 continued from previous page.)

to be exposed more to crocidolite than chrysotile, a possible independent reason for the greater disease. In addition, the numbers from which the conclusions are drawn are not large.

Although testifying that his data did not address this issue (C. McDonald Tr., Vol XII at 29), Dr. McDonald noted:

> [I]t is reasonable to think that there are body defense mechanisms, and therefore one can imagine a situation in which these are overwhelmed, as it were, and therefore it's hard to believe that a peak exposure would be less troublesome than the same amount spread over time. It might be much more.

C. McDonald Tr., Vol. XIII at 62.

28/ B.C.A. Exhibit 1, Tab 3, Enterline, P., et al. "Mortality in Relation to Occupational Exposure in the Asbestos Industry," in J. Occup. Med. 897-903, at 900 (1972).

Dr. Enterline testified.

> [T]he whole notion of time weighted averages doesn't fit that situation very well, you know, but we did ... the best estimate we could make suggested that these high intermittent doses apparently were having a disproportionately large effect on the incidence of lung cancer.

P. Enterline Tr., Vol. VIII at 41.

Cumulative Exposures (mppcf-years)	Production Workers	Maintenance-Service Workers
Under 125	144.7	318.8
125 - 249	184.2	363.6
250 - 499	222.2	446.4
500 - 749	060.0	1385.7
750 and over	428.6	1000.0

Dr. Enterline testified that his belief "high intermittent doses probably have disproportionately large effects on disease" was also "about the only way that I can explain the very high cancer incidence that you observe in insulation workers."[29]

The timing of exposure can thus be important to risk assessment both in terms of the need to match exposure profiles (high dose for a few years vs. low dose for many years) and the need to consider the possible inordinate

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29/ P. Enterline Tr., Vol. VIII at 42.

Dr. Nicholson noted the prevalence of such peak exposures for insulators:

> [T]hey were engaged in ... mixing cement, applying the wet material, applying block around pipes or on turbines and boilers, or whatever.
>
> The dust concentrations in individual activities were highly variable. If you measured for the two or three minutes that a person mixed asbestos cement, it could have a ... one could measure a concentration of as much as a hundred fibers per milliliter, but then for the next hour when he was applying that wet cement there would be minimal dust in the air, and the average over that period of time was relatively low.

N. Nicholson Tr., Vol. XIV at 42.

> [O]ne should try to match the exposure pattern in your study population as closely as possible to the exposure pattern in the population for which you want to estimate risk. ... Some studies, I think which have very short exposures are inappropriate for estimating risks from long-term exposures.

A similar concern is raised by attempts to extrapolate from the experiences of workers whose exposures came in very high bursts for limited periods in a given work day. The insulators studied by Dr. Selikoff, for example, are known to have received much of their asbestos exposures in short periods when concentrations peaked at 50, 100 or more fibers cc. Some factory workers may also have experienced such peak bursts of exposure during maintenance operations, breakdowns, or clean-up operations.

Such peak exposures were not typically included in the exposure estimates for the various cohorts, primarily because there is no data base upon which to determine how frequent and how high such exposures were. Their exclusion from the exposure estimates is doubly significant if such peaks, or bursts, of exposure are inordinately significant to disease.

Inordinate significance of peak exposures is likely from a biological viewpoint. Asbestos clearance mechanisms are much more likely to be overwhelmed by such bursts of

exposure than by steady, lower exposures. A number of witnesses noted this likelihood.[26]

Although the evidence is not without its interpretative difficulties,[27] both Dr. Enterline's and Dr. Weill's studies

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[26] Dr. Nicholson:

> The possibility is that intense exposures may overwhelm clearance mechanisms and thus you have more fibers retained in such circumstances.

N. Nicholson Tr., Vol. XIV at 69.

Dr. Bechtle:

> I think that it is likely and possible, both on the basis of scattered evidence and on the basis of what is known about lung clearance mechanisms, that large doses might indeed permit greater temporary retention.

H. Bechtle Tr., Vol. XX at 47.

Mr. Pete:

> Another possibility, which is what I was talking about there, is that in fact transient high exposures are in fact a major source of risk, and this hasn't been examined in proper detail. It could be very easily. It's simply a matter of doing a lot of detailed measurements, both personal and static measurements, and seeing whether the areas under the large peak actually account for a substantial fraction of the total inhaled asbestos. This is something which I think hasn't been very well studied, and that would certainly be one explanation for this failure.

J. Pete Tr., Vol. XXVII at 33.

[27] See P. Enterline Tr., Vol. VIII at 41; H. Weill Tr., Vol. IX at 70-71.

Interpretation of both investigators' conclusions is made difficult because maintenance workers, at least in the Weill cohort, also tended

[Footnote 27 continued on next page.]

duration of exposure and average concentration play a part, and I think you can see quite clearly that both do ... .

As both Dr. Enterline and Dr. Weill have demonstrated from the exposure and mortality information from their cohorts, "it is not sufficient to equate total exposure with either duration or average concentration which constitutes an important component of risk, and which exhibits J-curves with no apparent excess hazard."[31]

Given the central importance of quantitative exposure information, several of the asbestos epidemiology studies that are important for other purposes are not useful for risk assessment. Among such studies are the insulator studies of Dr. Selikoff and the amosite insulation factory studied by Dr. Seidman. In neither case is there any quantitative data on either individual or even average, exposures.[32]

3) The degree of control for smoking.

Given the significant impact of smoking on the risk of asbestos-related disease as discussed in Chapter III, the

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31. R.C.A. Exhibit 7, Pub 7, Weill (1979), supra note 4, at 331.

32. Also of limited value for quantitative risk assessment are a number of other studies not extensively discussed elsewhere in this summary, such as R.C.A. Exhibit 11, Pub 4, Newhouse, [illegible] and J. Berry, 'Patterns of Mortality in Amosite Factory Workers in Carteret,' [illegible] R.C.A. Exhibit 17, Pub 4, Nicholson, [illegible] 'Long-Term Mortality Experience of Chrysotile Miners and Millers in Thetford Mines, Quebec,' [illegible]

availability of smoking data on a cohort's members provides essential information to predicting risks. With information on which members of the cohort were and were not smokers, it is possible to make the necessary adjustments to determine what percentage of the health outcomes should be attributed to asbestos exposures as opposed to smoking.

4) The exposure pattern of the cohort.

Predictions of risk are further complicated by absence of knowledge about whether the time pattern by which exposure occurred is significant to disease outcomes. It is convenient to talk of cumulative lifetime exposures. Such numbers, however, would obscure any possibility that the risk from short-term high exposures (such as 50 fibers/cc for two years) may differ from the risk of longer-term lower exposures (such as 5 fibers/cc for ten years), despite the fact both patterns yield the same cumulative lifetime exposure (100 fiber-years).

Concern about the possible impact of the pattern of exposure is raised particularly when attempts are made to predict risks for future long-term, low level exposure workers from those epidemiology studies of workers with quite short-term intense exposures, such as the Paterson amosite insulation workers. As Dr. Crump noted:[33]

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33. K. Crump Tr., Vol. XXVI at 24-25.

Thus, he continued, strong consideration should be given in predicting risks for any given group, to seeking studies of persons who had been exposed in similar settings to similar asbestos.

At the same time, Dr. Acheson cautioned against over-reliance on studies from only one industry sector:[10]

> Q. But one would not, therefore, use a mining population study to determine the risk for a textile mill, for instance?
>
> A. Well, the question is, how else are you going to do it? The great strengths of the Quebec data are that you have far more cases - two hundred and fifty deaths from lung cancer - than in any other study, and also you have a number of points at low levels.
>
> If you look at the dose-response relationship, there are quite a lot of points near the origin, which when you are dealing with current practice where inevitably the levels are much less dusty than they were fifteen, twenty years ago, this is particularly helpful.
>
> In contrast, the number of lung cancer cases used both by Rochdale and Dement is very small. In Dement's cases here we have twenty-six cases of lung cancer, and in the Peto case, it was, I think, about the same or possibly a smaller number.

Risk assessments for particular occupational environments would thus optimally be based on experience of workers in similar environments. But, as Dr. Acheson emphasized, limited data from particular environments necessitates that

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[10] D. Acheson Tr., Vol. XIX at 102-103.

reference also be made to data from similar, although not identical, environments.

3) The quality and quantity of the exposure data.

As important as the identity of the asbestos exposures being considered is the quality and quantity of the exposure data. As Dr. Crump noted, for those studies where exposure information does not exist, quantitative risk assessment is next to impossible. Indeed, as he concluded, to attempt to do so would be "only slightly more appropriate" than attempting to quantify risks when exposures -- but not health effects -- are known.[11]

The importance of information on intensity of exposure, and not just duration of employment, was emphasized by several witnesses. As Dr. Weill noted, his studies have found both duration and intensity to be associated with risk parameter.[12]

> We and some others have always felt that simply time of exposure is not enough, because people are exposed at different levels. Some jobs, as I've showed you in our industry, are relatively low-level exposure, and some are very high level exposure.
>
> What we did in the mortality study then, was to see whether or not both

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[11] K. Crump Tr., Vol. XXVI at 33-34.

[12] H. Weill Tr., Vol. VI at 33.

Dr. Crump provided the Commission with criteria for evaluating the suitability of an epidemiological study for risk assessment. These criteria are listed and discussed roughly according to relative importance with identity of asbestos material and quality and quantity of exposure data being of greatest importance.[17/]

1) The identity of asbestos material to which the cohort was exposed.

Almost without exception the medical witnesses emphasized the varying risks found in different settings -- from chrysotile mining to asbestos-cement production to asbestos textile production. Dr. McDonald summarized the variance:[18/]

17/ Factors which relate to the strength of an epidemiological study in general rather than more specifically to risk assessment -- such as the definition of the cohort and the completeness of follow-up and vital status ascertainment -- were discussed in Chapter III, supra.

18/ C. McDonald Tr., Vol. XIII at 58. Dr. Doment agreed:

> The steepness of dose-response data is different, very much different from the McDonald studies, not so different ... not that much difference between us and Enterline as the McDonald

> I think a couple of points . first of all textile operations are not the same as brake lining operations, they aren't the same as mining and milling operations

> We, in some other work, have attempted to look at the airborne fiber size, dimensional characteristics of textile versus other operations using chrysotile.

> I think two things - first we find that in textiles and the nature of the operation, -- fine you need them, you find a prevalence of smaller, thinner fibers. From the mills, [illegible]

(Footnote 18 continued on next page.

> Both Rochdale, on the one hand, and even more definitely, Dement, on the other ... are very hard to reconcile with those other -- the mining, milling, and A/C pipe and friction.

> And, again, we don't have to be surprised, because we're dealing with a number of processes which essentially consist of mixing asbestos; not tearing it apart. And, therefore, it doesn't seem too surprising that the sort of environmental exposures might be similar in their results.

> It doesn't, again, seem surprising that, if you go into textiles, where you have the carding, which is one of the most traumatic kinds of process, that the fibre will be different at the end of it.

Dr. Crump emphasized the importance of these observations to risk assessment:[19/]

> [I]n reviewing the literature there is evidence that _____ is some differential risk from different types of fibers, and even from different processes that use the same fiber type, and whether this is due to different physical dimensions of fibers or chemical dimensions, for whatever reason, in the epidemiological studies there does appear to be a differential in risk from various materials.

(Footnote 18 continued from previous page.)

> these appear to be the most important in terms, at least, of cancer success, from the models that have been used, implantation models.

> So I think it's quite likely that we will see differences by industrial sectors.

J. Doment Tr., Vol. XXXII at 36-37.

19/ K. Crump Tr., Vol. XXVI at 33.

> Our reasons for preferring a linear hypothesis are:
>
> (1) it fits the data for occupational exposures;
>
> (2) it is the simplest hypothesis and the one most readily used for extrapolation to the probable effects of low doses;
>
> (3) it is likely to lead to an overestimate rather than an underestimate of risks at very low doses.

In relying on such a model, the Simpson Report's authors were thus quite explicit in noting that it will "likely lead to an overestimate" of risk. Dr. Crump also emphasized to the Commission the likelihood of over-estimation:[15]

> [A] linear relationship is not apt to underestimate risks appreciably. It may be about right in some cases. It may overestimate risk considerably in some cases.

Dr. Crump noted that he was not aware of "any plausible models of carcinogenesis which predict a superlinear type effect," although a number of models would predict a curvilinear or threshold effect, thus making a linear assessment an upper bound estimate.[16]

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[15] E. Crump Tr., Vol. XXVI at 33; see id. at 105.

[16] Id. at 105.

There is a theoretical argument that suggests cancer incidence should very approximately linearly with dose for low doses, particularly when there is an appreciable background of carcinogenesis in unexposed populations. See E.C.A. Exhibit 38, Tab 1, Crump, K., et al., "Fundamental

(Footnote 16 continued on next page.)

Most risk assessments of low level asbestos cancer risks thus are premised on linear non-threshold models. Such models establish upper limits on the possible risk from such exposures. When such conservative models are used, extra protection is afforded the exposed population.

1. Choice of Data.

Numerous epidemiology studies could be used to extrapolate to low asbestos exposure dose-response relationships. As discussed in detail in Chapter III, significant qualitative differences emerge among the studies, particularly with respect to the amount and accuracy of exposure information.

(Footnote 16 continued from previous page.)

Carcinogenic Processes and Their Implications For Low Dose Risk Assessment," 16 Cancer Res. 2073-79 (1976). But, as Dr. Crump himself noted in his Commission testimony:

> The argument for linearity ... would pertain more particularly to lung cancer in smokers than it would to mesothelioma or certainly I don't think it's very appropriate for asbestosis, or even lung cancer in nonsmokers ...

E. Crump Tr., Vol. XXVI at 103.

A linear non-threshold model will clearly overestimate any [illegible] risk. As Julian Peto has noted:

> There are no grounds for assuming linear dose-response for such a generalized progressive disease, and although a qualitative dose-response has been demonstrated at very high exposure levels, there may well be a safe or virtually safe threshold.

E.C.A. Exhibit 37, Tab 3, Peto, J., "The Hygiene Standard for Chrysotile Asbestos," The Lancet 484-89, at 488 (1)[illegible] [illegible] letter.

> [E]stimation of the shape of the dose-response curve in that very low part of the curve ... is very difficult ... . [J]ust because that relationship [at high exposures] is relatively linear, and many people have shown that, the Quebec study, Enterline, and so forth, that doesn't necessarily mean as this cumulative normal curve shows, that is relatively linear here, it doesn't necessarily mean that it is linear to intercept.

Choosing a model to extrapolate risks at lower levels is necessitated by the unlikelihood that epidemiology will ever be able to determine definitively what the dose-response relationship is at the low levels prevalent today. Dr. Nicholson has described the difficulties in determining a threshold exposure below which asbestos-related disease will not occur:[12/]

> The task confronting one who would define a level below which no carcinogenic risk exists for human populations is virtually an impossible one. ... [T]hree unfulfilled requirements confront the investigator attempting to establish a "no effect level" for asbestos:
>
> 1) a sufficiently large population for observation;
>
> 2) knowledge of the asbestos concentrations to which the population was exposed; and
>
> 3) an observation time sufficient for the effects of asbestos to be manifest.

As a number of witnesses noted, it will be many years before a sufficiently large population of workers at today's exposure

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12/ R.C.A. Exhibit 19, Tab 5, Nicholson, W. J., "Case Study 1: Asbestos--The PLV Approach," 271 Ann. N.Y. Acad. Sci. 132-49, at 133 (1976).

levels is followed for a sufficient time to determine whether they are suffering from asbestos-related disease. Given the low rates of disease predicted for such workers -- even if linear non-threshold models are assumed -- a very large group of workers would need to be followed to assess such an experience definitively.

As Geoffery Berry has noted, even if the existence of a threshold were beyond biological dispute, it would be quite unlikely that epidemiologic evidence would ever be able to establish the threshold dose level. Because of random variation, even if there were no cases among 1,000 studied individuals that would only mean there was reasonable certainty that the risk was less than 3 in 1,000.[13/]

Given the near impossibility of ever being able to document convincingly the existence of a threshold and determining exactly what that exposure level is, many observers have advocated use of a linear non-threshold model for predicting asbestos cancer risks. The Simpson Report, for one, made such a choice, explaining:[14/]

> [W]e prefer the linear hypothesis for asbestos-related lung cancer, although it is important to emphasize that there is considerable uncertainty about the form of the relationship, and forms other than a linear relationship cannot be excluded at very low doses.

13/ R.C.A. Exhibit 19, Tab 4, Berry, G., "Hygiene Standards -- Theory and Application," in Biological Effects of Asbestos 145-149, at 145 (IARC Lyon 1973).

14/ R.C.A. Exhibit 19, Tab 5, British Advisory Committee, Asbestos [hereinafter cited as "Simpson Report"] Vol. II at 14 (1979).

Although the asbestos epidemiology studies are consistent with a linear relationship at all exposure levels, the same data are also consistent with curvilinear and threshold models that would predict far smaller risks.[9] Predictions that the high dose effects will correlate linearly with risks at lower exposure levels must thus be recognized as assumptions, rather than verified conclusions. As Dr. Sackett warned the Commission at its Second Public Meeting, "beware extrapolations beyond actual observations, for they represent acts of faith rather than acts of observation."[10]

Dr. Weill demonstrated the possibility of several models being correct for predicting low exposure risks in the data from his Louisiana asbestos-cement studies. As the

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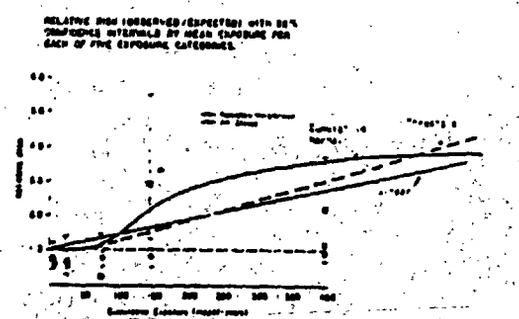
9 Many of the authors of the asbestos epidemiology studies have confirmed this fact. See J. McDonald Tr., Vol. XIII at 43; H. Weill Tr., Vol. IX at 76-77. Dr. Weill has written:

> [A linear non-threshold] model assumes that some excess risk occurs at all degrees of exposure. It must be emphasized that these estimates are based on extrapolation from high-dose effects. It has recently been pointed out that there is no theoretical reason to accept necessarily the "linear at low dose" assumption and that there is experimental evidence to support a contrary hypothesis, i.e., that a low dose may exist that does not result in a carcinogenic outcome, presumably because of some ability of the host to deactivate the process, the carcinogenic effect being detected when such deactivation capability has been saturated.

B.C.A. Exhibit 7, Tab 7, Weill, H., et al., "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," 120 Am. Rev. Resp. Dis. 1-9-34, at 352-53 (1979).

10/ Sackett, D. L., "A Review of Diagnostic Tests for Lung Cancer," Commission's Second Public Meeting, App. B at 8 (Dec. 12, 1980).

following chart demonstrates, the dose-response data from his cohort are not inconsistent with a linear, non-threshold dose-response relationship, but are also not inconsistent with a clear threshold model that would predict no disease at exposures substantially above those prevalent, even in primary manufacturing, today:



As Dr. Weill explained:[11]

11/ H. Weill Tr., Vol. IX at 76-77.

Dr. Nicholson, although arguing for linear extrapolation to occupational exposures, noted the absence of data to justify such a model for even lower environmental exposures:

> Identification of the appropriate extrapolation model is of importance for environmental exposures to asbestos, but has limited relevance for current occupational circumstances.

B.C.A. Exhibit 19, Tab 9, Nicholson, W., "Dose-Response Relationships for Asbestos and Inorganic Fibers," 11 (1981).

by automobile or smoking -- are voluntarily assumed by millions of persons, many of them with full knowledge of the dangers involved. Even though many persons do not consciously assess all risks prior to initiating activities, or, in some cases, recognize the extent of risk, their willingness to conduct such activities establishes a context within which to assess preventable risks. Clearly, for life to continue, some risks can reasonably be tolerated, or are not large enough to justify exorbitant expenditures to achieve further risk reduction. Accordingly, government would not be making wise decisions for its constituents were it to attempt to eliminate risks that citizens would consider worth taking.

B. Risk Extrapolation Principles and Data Selection.

Before using quantitative risk assessment methods with the asbestos data, several methodological questions first have to be addressed:

-- What model should be used to extrapolate from known risks at high exposure levels to much lower exposures prevalent today?

-- What epidemiologic data is best suited for such risk extrapolation?

-- What types of exposures are likely to occur in the future under various regulatory regimes?

The following section summarizes the extensive information on these issues presented to the Commission.

1. Choice of Model.

Any efforts to extrapolate the high dose epidemiology results to predict the consequences of today's low exposures must rely on some model to determine the shape of the dose-response relationship. As illustrated in the many epidemiologic studies discussed in the previous Chapter, the high exposure dose-response relationships for asbestosis, lung cancer and mesothelioma have generally been linear. However, for lower dose subcohorts (where they have been able to be identified), there has often been no apparent response. The overall dose-response relationship could thus take one of several forms:

-- It could be linear throughout, with an absence of response only when exposure is zero (linear non-threshold);

-- It could be linear at high exposure levels, but decrease in severity as exposures near zero (curvilinear);

-- Although linear at high exposure levels, an exposure level could exist below which no response occurs.

As discussed in detail in Chapter III, substantial support emerges from several of the larger and best documented epidemiology studies for the thesis that a dose exists below which asbestosis and lung cancer will not occur. On the other hand, as noted by several witnesses, even those data cannot rule out the possibility of some small risk at lower exposure levels.

Dr. Crump stressed to the Commission the value of expressing risk in such terms:*

> Most people, I would guess, are more concerned about when they die than the specific cause of death, and most people would prefer death to a number of causes at age seventy as opposed to death from any cause an adolescence.
>
> I think the time at which a person dies is an important concept which needs to be taken into account in a risk assessment. One way to do that is to use a measure that incorporates length of life, such as loss-of-life expectancy. This measure incorporates both the risk of acquiring the disease, plus the loss of life resulting from the disease.

It has been argued that average loss of life expectancy is an inadequate measure of risk because it does not reflect the loss of life of the unfortunate person who actually contracts a disease. This objection is usually couched in terms such as "a six-month loss of life expectancy does not mean much to the unlucky individual who contracts the disease at an early age and loses twenty years of life." However, all reasonable measures of risk must reflect both the severity

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*. E. Crump Tr., Vol. XXVI at 7. Dr. Crump also noted the flexibility of this means of expressing risk to deal with exposures associated with multiple diseases:

> [A]nother advantage ... is that you can apply this one single measure ... to exposures which might cause increases in a number of different diseases. You can compute loss of life expectancy from all diseases combined, and as an overall measure, a single measure, of human risk from a particular exposure.

Id.

of the disease and the chance of acquiring it. Loss of life expectancy does both.

Dr. Crump presented to the Commission data demonstrating average loss of life expectancy from a number of well-known occupational and environmental risks:[1]

LOSS OF LIFE EXPECTANCY FROM VARIOUS CAUSES

Cause	Loss of Life Expectancy in Days
Occupational accidents	
Trade	30
Manufacturing	43
Service	47
Government	55
Transportation and public utilities	164
Agriculture	277
Construction	302
Mining, quarrying	328
Commuting to work by automobile	78
Smoking	
(1) Cancer of lung, esophagus, and other respiratory sites; chronic bronchitis and emphysema; and pulmonary heart disease	690
(2A) All causes in (1) plus ischaemic heart disease	1395
(2B) All causes in (1) and (2A) plus various other causes probably attributable to smoking	1618

As the table above emphasizes, risk is a common, everyday fact of life. Indeed, some of the largest risks -- commuting

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1. E.P.A. Exhibit 16, Tab J, Table 13: occupational accident data for 1969 and the 1971 commuting figure based upon 1976 U.S. traffic safety statistics; smoking risks based upon Hill and Doll (1976).

CURRENT OCCUPATIONAL RISKS

	Annual Risk	Lifetime Risk
Mining and Quarrying (accident only)	1 in 1,400	1 in 28
Coal mining - accident (average 1970-1973)	1 in 400	1 in 10
- Black lung disease (1976)	1 in 120	1 in 3
Agriculture - total	1 in 1,700	1 in 34
- tractor driver (1 driver/tractor)	1 in 9,000	1 in 140
Trade	1 in 17,000	1 in 340
Manufacturing	1 in 12,000	1 in 240
Service	1 in 11,000	1 in 220
Government	1 in 8,300	1 in 166
Railroad worker (1976) (all accidents excluding grade crossing)	1 in 400	1 in 16
Fire fighter (1971-1972 average)	1 in 1,200	1 in 24
Construction	1 in 1,700	1 in 34
Police Officer (killed in the line of duty)	1 in 3,300	1 in 66

Comparable occupational fatality rates were provided by Robert Wilson of Ontario Hydro.[1] Mr. Wilson noted that the average fatal accident rate throughout Ontario industry is 4 to 5 per 100 million manhours, that the fatality rate for electrical linemen in the mid-1970's was 11 to 12 per 100 million manhours, and that Ontario Hydro had established a

1 R. Wilson Tr., Vol. XLA at 12, 44, 46-47.

goal of reducing such fatalities to 6 per to 100 million manhours. Given that the average worker employee works 80,000 hours in his working life (2,000 hours/year times 40 years), these Ontario figures translate to a range of 3 to 10 per thousand (or 1 in 333 to 1 in 100) fatalities per working lifetime, a range consistent with that shown in the table above.

Even the calculations of additional risks of death in the tables above, however, provide only limited information. Everyone dies eventually, and an increase in the probability of death from one disease therefore also means a decrease in the risk of death from other causes. The calculations above give no weight to time of death. As Dr. Corbett McDonald noted in his Commission testimony:[2]

> One of the marked limitations of all the analysis we've been doing is that it gives equal importance to a death at ninety or at forty or at twenty. And, I think, in social terms, that is a pity.

One means of taking into account this missing factor is to express additional risk in terms of loss of life expectancy.

2 C. McDonald Tr., Vol. XIII at 125. Dr. Finkelstein agreed:

> [J]ust by saying there is an extra two percent of people who die, you ignore when they die, which I think is socially important.

M. Finkelstein Tr., Vol. XXX at 53.

variety of everyday activities. Such activities include recreational pursuits and many commonplace accidents. For example, the following everyday risks of death are faced annually by most Americans:[2]

	Annual Risk
Motor Vehicles	1 in [illegible]
Pedestrians	1 in [illegible]
Home Accidents	1 in [illegible]
Falls	1 in [illegible]
Fires	1 in [illegible]
Lightning	1 in [illegible]

Over an average 70-year lifetime, each American faces [illegible] times the risks indicated above, e.g., the lifetime risk of death due to home accidents is 1 in [illegible], that due to lightning is 1 in 20,000.

Annual risks of deaths due to cancer have also been estimated for a number of common activities.[3]

	Annual Risk
Cosmic Ray Risks	
One transcontinental flight/year	1 in [illegible]
Living in Denver compared to N.Y.	1 in [illegible]

2/ See Crouch and Wilson, "Estimates of Risk," [illegible] J. of Bus. [illegible] of Dr. Columbia 299 (1979-1980); R. Wilson, "Analyzing the [illegible] of Life," Technology Rev. [illegible] (Feb. 1979).

3/ Crouch and Wilson (1979-1980), supra note 2.

	Annual Risk
Other Radiation Risks	
Average U.S. Diagnostic Medical X-rays	1 in 100,000
Natural background at sea level	1 in 50,000
[illegible]	
[illegible] peanut butter [illegible]	1 in 25,000
[illegible] New Orleans drinking water	1 in 400,000
Smoking	
Smoker (cancer only)	1 in 500
Person in room with smoker	1 in 100,000

Again, lifetime risks will be greater.

Even greater annual risks, on the order of one in ten thousand, i.e., lifetime risks of approximately one in 200 over a 50-year working life, are commonly accepted by workers in many occupations.[4]

4/ Based on Crouch and Wilson (1979-1980), supra note [illegible].

Prior to exploring the quantification of asbestos risks, it is useful to consider first, a framework within which such risks should be placed; second, principles for evaluating epidemiologic evidence for purposes of risk assessment; and, third, several issues peculiar to asbestos risk assessment, including conversion of historical particle counts to currently employed fiber measurements.

A. Measures of Risk.

Epidemiology studies typically express risk in terms of ratios of risks in a cohort of exposed workers compared to an unexposed population, taking into account factors such as age, sex and race. Relative risks are often expressed as Standardized Mortality Ratios (SMR's), which are relative risks of death multiplied by 100.

The relative risk of any single disease is not particularly meaningful as any relative risk greater than one of a common disease is much more significant than a similar relative risk of a rare disease. Relative risks of a specific disease are therefore often re-expressed in terms of additional risk of death by taking into account the death rates attributable to the disease at issue in the unexposed population. It is then possible to speak in terms of the risk, either annually or during a lifetime, that a person will die prematurely due to some exposure. For example, if the relative risk of lung cancer given a certain exposure is 2

(i.e., an SMR of 200) and 10% of the background population die from lung cancer in a given year, then 20% of the exposed population (i.e., twice the expected rate, or two out of 10 persons) would die from lung cancer in that year. The additional risk of death for any person with the given exposure would thus be 10%.

Once risks are placed in such terms, it is possible to put them into a meaningful context. By comparing the risks calculated for toxic chemical exposures to other common risks, meaningful decisions about the necessity or desirability of controlling such exposures can be made. As Dr. David Sackett told the Commission:1/

> There is abundant everyday evidence that "acceptable thresholds" exist for all sorts of environmental hazards: highway speed limits are a case in point. Thus, the issue becomes one of reflecting society's judgements of the utility of the social risks (e.g., health) and social benefits (e.g., economic) of various points along the dose-response relationship.

Society normally accepts annual mortality risks on the order of one in a million (equivalent over a typical 70 year life to a lifetime mortality risk of seven in a hundred thousand), and frequently accepts higher levels of risk in a

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1/ Sackett, D. L., "A Review of Diagnostic Tests for Isbestos," Commission's Second Public Meeting, App. B at 8 (Dec. 12, 1980)

CHAPTER IV

IV. OCCUPATIONAL RISK ASSESSMENT.

In an era of increasing concern about toxic substances -- in the workplace, in the food supply, in the general environment -- it is obvious that it is not possible, nor even desirable, to eliminate all human exposures. Attention must be focused on how society's resources should be allocated to cope efficiently with the most significant risks. Thus, it is necessary to attempt to place risks in some quantifiable context.

If risk quantification is not employed, societal resources will inevitably be misallocated with inordinate sums being spent to eliminate de minimis risks, while significant risks are ignored. Considerable scientific efforts have thus been devoted to quantitative risk assessment, and asbestos has been among the most studied substances. Despite the uncertainties in the asbestos evidence, the fact remains that it is a substance for which a very substantial data base exists.

Quantitative risk assessment typically involves extrapolation of experimental or observational results at high exposures to predict effects of lower exposures. For many chemicals, high dose results can be derived only from animal experiments, creating the need for additional interpolation from animal to man. For asbestos, the most meaningful predictions come from epidemiology studies.

> I think that should be done before anyone assumes that asbestos always ... high doses of asbestos are always associated with an excess of cancer of the alimentary tract.

As Dr. Weill concluded, after referring to the recent negative findings his own, the Dement, and the Ferodo studies, "I think more and more there is becoming some very substantial doubt" about any association between asbestos and gastrointestinal cancers.[230/]

b. Laryngeal Cancer.

Because upper respiratory tract tumors are relatively rare and often cureable, attention was not focused on the possibility of associations with asbestos until the 1973 case-control study of Stell and McGill reported an association for laryngeal cancer.[231/] Subsequent studies have uniformly failed to find an association as dramatic as in this initial report, and have cast doubt as to the existence of any true relationship at all. As Dr. Corbett McDonald has noted:[232/]

> A case-control study in Liverpool by Stell & McGill (1973) showed a very high relative risk (14.5), but the inquiries were not made blind, and it is difficult to accept that only 3% of control subjects in that city had been exposed occupationally to asbestos. In a smaller case-

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230/ H. Weill Tr., Vol. IX at 145.

231/ Stell, P. M. and T. McGill, "Asbestos and Laryngeal Cancer," The Lancet 416-17 (1973).

232/ R.C.A. Exhibit 18, Tab 22, McDonald (1980), supra note [illegible], at 14.

> control study in Toronto by Shettigara & Morgan (1975), also not made blind, an association was found with both asbestos exposure and smoking.

Two further morbidity case-control studies of laryngeal cancer have been conducted, and although one[233/] finds an association, the other does not.[234/] Mortality data from the two large worker cohorts are also in conflict, with the Selikoff insulators reported to have 9 (or best estimate 11) observed vs. 4.7 expected laryngeal cancer deaths, while the McDonald miners experienced no increase,[235/] leading Dr. McDonald to note:[236/]

> Our findings on laryngeal cancer are clear cut, although others' have not been so. There has been no excess in this industry, and there is no quantitative association with exposure; on the other hand, we find a direct relation with cigarette smoking.

In sum, the chance that any significant laryngeal cancer could occur at today's low exposure levels is minimal, if not non-existent.

233/ Morgan, R. W. and P. T. Shettigara, "Occupational Asbestos Exposure, Smoking, and Laryngeal Carcinoma," Ann. N.Y. Acad. Sci. [illegible].

234/ Newhouse, M. L., et al., "Study of Carcinoma of the Larynx," in Biological Effects of Mineral Fibres 987-95 (Lyon IARC 1980).

235/ See Selikoff (1979), supra note 2, at 111; and R.C.A. Exhibit 18, Tab 17, McDonald (1980), supra note 16, [illegible], at 17.

236/ R.C.A. Exhibit 18, Tab 18, McDonald (1980), supra note 10, at 23.

and no increased gastrointestinal cancer has been found -- despite heavy exposures -- among Finnish and Canadian miners, British and American asbestos textile workers, British friction product workers and American asbestos-cement factory workers.[243/]

The studies of Canadian miners by McDonald, et al.[244] threw additional light on the purported association with gastrointestinal cancer. Based on the mortality pattern of some 10,000 chrysotile miners born between 1891 and 1920 and followed through December 1969, for abdominal cancers (which included no peritoneal mesotheliomas), there appears here to be evidence of a no-effect level:

Age-Corrected Death Rates Per 1,000 Men
Born 1891-1920: Deaths to December 1969

	Past Exposure Level, mpcf.yrs.					
Cause	<10	10	100	200	<60	300+
Abdominal Cancers	18.8	13.6	18.7	11.6	26.3	28.7

243/ Newhouse, M. L., et al., "Mortality and Morbidity Among the Working Population of Amphibolite Asbestos Miners in Finland," 31 Br. J. Industr. Med. 133-42 (1974); B.C.A. Exhibit 18, Tab 18, McDonald (1980), supra note 10; B.C.A. Exhibit 19, Tab B, Nicholson (1979), supra note 24; Weill, H., et al., "Influence of Dose and Fiber Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing," 120 Am. Rev. Resp. Dis. 343-54 (1979); Doncet, J. M., et al., "Estimates of Dose-Response for Respiratory Cancer Among Chrysotile Asbestos Workers," presented at the Fifth International Symposium on Inhaled Particles (Cardiff, Sept. 8-12, 1980); Peto, J., et al., "A Mortality Study Among Workers in an English Asbestos Factory," 34 Br. J. Industr. Med. 249-59 (1977); B.C.A. Exhibit 13, Tab 13, Newhouse and Berry (1980), supra note 41.

244/ McDonald, J. C., et al., "The Health of Chrysotile Asbestos Mine and Mill Workers of Quebec," 38 Arch. Environ. Health 81-94 (1983).

The same pattern of excessive disease only in the most exposed subcohorts emerges in the 1980 McDonald follow-up, which reports results separately for esophagus and stomach, colon and rectum, and other abdominal cancers.[247/] Thus, Dr. Corbett McDonald told the Commission:[248/]

> The exposure-response relationship doesn't lie on a straight line. It is true, as you said, that the highest risk is in the highest dose group, but there is a disturbing lack of straightness about the line. . . . [I]t's an irregular relationship. So I think an irregularity of that kind is the kind of thing that at once makes you think that the pattern of causation is not simple. It suggests that perhaps there are some other important factors involved.

In addition, the failure in many of the studies to discriminate between cancers of the stomach, colon and rectum makes it difficult to analyze the data in a meaningful manner, as each of the three sites is known to have different etiologies. Dr. Acheson noted this problem:[249]

> I think that before one attributes an additional anatomical site of cancer to an industrial origin, it is highly desirable that some sample of the cancer should be examined by a pathologist, particularly when there is the possibility of confusion with peritoneal mesothelioma.

247/ B.C.A. Exhibit 18, Tab 18, McDonald (1980), supra note 10, at Table 10, p. 20.

248/ C. McDonald Tr., Vol. XIII at 109.

249/ D. Acheson Tr., Vol. XIX at 70-71.

None of the men who have never smoked regularly died of cancer of the esophagus, larynx, pharynx or buccal cavity. The expected number of deaths from these cancers was 1.5 for men who never smoked regularly. This suggests that in the absence of exposure to tobacco, exposure to asbestos dust may have little or no influence on death rates from such cancers.

a. Gastrointestinal Cancer

Although the Simpson Report concluded evidence existed of associations between inhaled asbestos and gastrointestinal cancer,[240] other commentators, including the distinguished British medical journal, The Lancet, have been less convinced:[241]

> The [Simpson] report gives cautious acceptance to the idea that alimentary-tract cancers may sometimes be caused by asbestos in conditions of heavy industrial exposure, but it points out that the evidence on this matter is not unanimous. Since the report was prepared the results of three further surveys (of crocidolite miners, crocidolite gas-mask workers, and asbestos cement workers) have been made known, none of which shows an excess of alimentary-tract cancers among the workers concerned. These findings

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240. Simpson Report (1979), supra note [illegible], Vol. I at [illegible].

241. "Asbestos and Health," The Lancet [illegible]. Hobbs, M. S. T., et al., "Monitoring of Cancer Risk in [illegible] late Miners in Western Australia," Jones, J. S. P., et al., "The [illegible] of Personnel Who were Exposed to Asbestos Dust in a [illegible] Factory;" and Hughes, J. and [illegible], "Lung Cancer Risk [illegible] with Manufacture of Asbestos Cement Products," papers submitted to the [illegible] ence on Biological Effects of Mineral Fibres [illegible] Sept. [illegible] 1979.

> raise the possibility that a factor additional to asbestos must be present in the industrial environment for alimentary-tract cancers to appear in excess among exposed workers.

Dr. Acheson, author of the Simpson Report's medical section, himself noted the evidence has become weaker since the Report was published:[242]

> Since the publication of our report, I feel personally that the evidence which we expressed reservations about at the time relating certain alimentary tract cancers to asbestos is, if anything, a little weaker.
>
> There have been a number of studies published in which there has been no excess of alimentary tract cancers noted. For example, the studies of the Nottingham gas mask workers, the friction material workers of the Ferrodo factory in England, and also Dr. Dement's study. In none of these has there been a significant excess of alimentary tract cancers.

While there have been reports of an excess of gastrointestinal cancer in asbestos workers, no consistent pattern emerges.[243] Only small not statistically significant numerical excesses have been reported in several studies.[244]

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242. J. Acheson Tr., Vol. XIX at 6.

243. See A.I.A. Exhibit 28, Tab 1, Simpson Report, supra note 116, Vol. II, Table 1a, at 45.

244. Enterline, P. E. and A. A. El-Attar, "Mortality Pattern in a Cohort of Asbestos Workers," [illegible] J. Occup. Med. [illegible] (1967); Enterline, P., et al., "Mortality in Relation to Occupational Exposure in the Asbestos Industry," 14 J. Occup. Med. 897-903 (1972); Lumley, K. P. S., "A Proportional Study of Cancer Registrations of Dockyard Workers," 33 Br. J. Industr. Med. 108-14 (1976).

In light of this human evidence from varied settings, the Simpson Report concluded that crocidolite, and to a lesser extent amosite, have been considerably more responsible for mesotheliomas than chrysotile.[117] Dr. Acheson put its conclusion in perspective for the Commission:[118]

> I am not suggesting this is a difference in kind, even in man, but a difference in degree ... but a difference which has been, up to the present, at least one order of magnitude, at least a tenfold difference. Dr. Alison McDonald suggests a thirtyfold difference in incidence - I wouldn't quarrel with that.
>
> . . .

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[117] B.C.A. Exhibit 28, Tab 5, Simpson Report (1979), supra note 116, Vol. II at 33.

Other reviewers have been even more adamant in their views:

> Speaking on behalf of Europe, I would like to make it categorically clear that we believe that, to man, up until today, all mesotheliomas caused by commercial asbestos have been due to crocidolite. This is an emphatic statement.

Wagner, J. C., "The Complexities in the Evaluation of Epidemiologic Data of Fiber-Exposed Populations," in R. Lemen and J. M. Dement, eds., Dusts and Disease 37-48, at 37 (1979).

Of the Commission witnesses, only Julian Peto argued strongly against the fiber-type distinction being evident in the epidemiology results. Even Dr. Peto, however, noted that the evidence on peritoneal mesotheliomas indicted only the amphiboles:

> I don't think anyone who has been principally exposed to chrysotile has ever had a peritoneal mesothelioma, as far as I know. Peritoneal mesotheliomas seems not to occur in chrysotile workers.

J. Peto Tr., Vol. XXVA at 14.

[118] D. Acheson Tr., Vol. XIX at 13-11.

Mesothelioma, therefore, is clearly associated with asbestos exposures, but this rare cancer is predominantly associated with heavy exposures to amphiboles. Those cohorts where significant disease have occurred have been characterized by heavy exposures, whether they be in an industrial setting or in the homes of workers. Today, with amphibole use being greatly limited, and overall exposure levels much reduced, asbestos-related mesotheliomas will be quite rare.

4. Other Cancers: A De Minimis Problem.

Suggestions have also been made, based on the cohort epidemiology results, that asbestos exposure may be associated with gastrointestinal and laryngeal cancer. These associations are considerably weaker than the associations discussed above. Indeed, many have questioned whether any association exists at all (other than perhaps at very high exposure levels) in light of the failure to find positive associations in many studies. At a minimum, great doubt exists as to whether such cancer will occur at today's low exposures.

As with lung cancer, the effects of cigarette smoking for such cancers are substantial. As Dr. Hammond has noted:[119]

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[119] Hammond (1979), supra note [illegible], at [illegible]

Africa in the Cape Province area where Cape Blue crocidolite is mined and very high exposures at one time existed. Such cases have not been reported in the neighborhood of mines producing other types of fiber. No indication of mesotheliomas associated with residence was found within 20 miles of a chrysotile mine in Canada.[230/] Similarly, the only instances of mesotheliomas found in the neighborhood of factories have been those reported in the vicinity of factories and shipyards in England and in Hamburg, Germany, where chrysotile and amphiboles were both used.[231/]

(4) Shipyards. The incidence of mesothelioma has been most dramatically found to be more than expected around seaport shipyards in an extensive study by McDonald and Sechlake.[232/] The same concentration of mesotheliomas in urban centers with naval shipyard concentrations has been found by Greenberg and Lloyd Davies.[233/] As noted by the Simpson Report, these data suggest "that there is a special risk associated with work in shipyards, perhaps due to exposure to amphiboles, to work in excessively dusty conditions, or to a co-factor."[234/]

230/ McDonald, J. C., and N. R. Sechlake, "Asbestos-Related Disease in Canada," paper presented in Berlin (1973).

231/ Newhouse and Thompson (1965), supra note 203; R.C.A. Exhibit 28, Tab 9, Simpson Report (1979), supra note 116, Vol. II at 31.

232/ McDonald and Sechlake (1973), supra note 230.

233/ Greenberg and Lloyd-Davies (1974), supra note 203.

234/ R.C.A. Exhibit 28, Tab 9, Simpson Report (1979); supra note 116, Vol. II at 31.

(5) Tissue Fiber Concentrations. Studies of fiber concentrations in deceased mesothelioma victims have also supported the proposition that the amphiboles are more hazardous. *Jones et al. found more crocidolite and amosite* in the lungs of mesothelioma victims than in lungs of several control groups, but found no statistical difference between chrysotile concentrations in lungs of cases and controls.[235/] The McDonalds and F.D. Pooley found the same relationships -- equivalent numbers of chrysotile fibers in cases and controls, but more crocidolite and amosite fibers in cases -- in tissue samples from North American lungs.[236/]

235/ R.C.A. Exhibit 13, Tab 8, Jones, J. S. P., et al., "The Pathology and Mineral Content of Lungs in Cases of Mesothelioma in the United Kingdom (1970)," in Biological Effects of Mineral Fibres 187-97 (IARC Lyon 1980).

236/ R.C.A. Exhibit 18, Tab 24, McDonald, A. D., et al., "Mineral Fibre Content of Lung in Mesothelial Tumours in North America," presented at BOHS 5th International Symposium on Inhaled Particles (Cardiff 1976).

Dr. Acheson added that dissolution of chrysotile could not account for these differences:

> [I]n that case you would expect to find less chrysotile in people with mesotheliomas, than controls. You find neither less nor more, you find the same, whereas with the amphiboles, you find more. It seems to me that in logic the most likely explanation is that the amphiboles are responsible for the mesotheliomas.
>
> If the dissolution of chrysotile was relevant, I believe there would be less in the cases in the controls, and this has never been found.

[illegible] Tr., Vol. XIX at 119-20

mesotheliomas cases have been also found among Australian crocidolite miners.[222/]

On the other hand, of 11,379 chrysotile miners and millers studied by McDonald in Canada, only 11 mesotheliomas have been found, two of them occurring in persons who also worked with crocidolite gas mask filters.[223/] No mesotheliomas have been reported in chrysotile miners and millers in the Soviet Union or Rhodesia; only one case has been detected in mining data from Italy and Cyprus.[224/]

(2) Factory Cohorts. Very high mesothelioma rates have been found among crocidolite factory workers, including gas mask filter assemblers during World War II in both

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(Footnote 221 continued from previous page.)

> The Transvaal crocidolite mines were used only to any extent during the Second World War, when crocidolite was required in vast quantities for marine insulation. I don't place a great deal of reliance on the failure to find many cases of mesothelioma in relation to the Transvaal crocidolite mines. I suspect it's a local matter, because they have been found in Western Australia, which is the other place where crocidolite has been mined.

D. Acheson Tr., Vol. XIX at 34.

222/ R.C.A. Exhibit 28, Tab 3, Simpson Report (1979), supra note 116, Vol. II at 28.

223/ R.C.A. Exhibit 18, Tab 18, McDonald (1980), supra note 10, at 21; R.C.A. Exhibit 28, Tab 3, Simpson Report (1979), supra note 116, Vol II at 28.

224/ R.C.A. Exhibit 28, Tab 3, Simpson Report (1979), supra note 116, Vol. II at 28.

Britain[225/] and Canada,[226/] and a group of workers exposed only to amosite during World War II.[227/] High mesothelioma rates have also been found among workers with mixed fiber type exposure, including workers at the East London Barking asbestos factory,[228/] and insulation workers in the United States and Canada.[229/]

(3) Neighborhood Cases. As noted previously, cases of mesotheliomas have been reported in neighborhoods in South

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225/ R.C.A. Exhibit 13, Tab 13, Jones (1980), supra note 195. See also Jones, J. S. P., et al., "Factory Populations Exposed to Crocidolite Asbestos -- A Continuing Survey," 32 Environ 117-20 (1976).

226/ R.C.A. Exhibit 18, Tab 14, McDonald, A. D. and J. C. McDonald, "Mesotheliomas After Crocidolite Exposure during Gas Mask Manufacture," 17 Env. Res. 340-44 (1978). Of 199 employees in the three Canadian World War II gas mask manufacturing plants, 174 were traced to 1975; 56 were found to have died; and, of these, nine (16%) had died of mesotheliomas (three pleural and six peritoneal). Another seven deaths were due to respiratory cancer.

227/ Selikoff, I. J., et al., "Carcinogenicity of Amosite Asbestos," 23 Arch. Env. Health 107-16 (1971). Findings of mesotheliomas among workers in an insulation factory using predominantly amosite have also been attributed to the amphibole exposure in a recently published study by Dr. Acheson. Acheson, E. D., et al., "Mesothelioma in a Factory Using Amosite and Chrysotile Asbestos," The Lancet 1303-1305, 1305 (Dec. 19/26, 1981). An accompanying editorial agrees that "in man exposure to amosite has been associated with a higher risk of mesothelioma than to chrysotile." "Amosite Asbestos and Mesothelioma," The Lancet 1397-99, 1398 (Dec. 19/26, 1981).

228/ Newhouse, M. L. and G. Berry, "Predictions of Mortality from Mesothelial Tumours in Asbestos Factory Workers," 33 Br. J. Industr. Med. 147-51 (1976).

229/ Selikoff and Hammond (1979), supra note 2, Table 3 at 94. Dispute exists about the fiber type exposures of these insulation workers, but considerable evidence exists that they were exposed to amosite as well as chrysotile, and may also have had crocidolite exposure. See R.C.A. Exhibit 28, Tab 3, Simpson Report (1979), supra note 116, Vol. II at 29-30.

The Rochdale data are for annual averages, not individual samples. As Dr. Crump noted:[42]

> I would venture a guess that if these [Canadian] data contained averages in the same way that those data [Rochdale] are, you would see probably at least as good a relationship for these data as you would for those data.

Although he was the author of the recent Rochdale mortality studies, Mr. Pete was unable to shed further light on the issue of the validity of their conversions. As he told the Commission, he has never analyzed the measurements from that factory:[43]

> I was given the data in the form of final estimates. I haven't had access to the results of the parallel measurements or the detailed measurements in different areas during the 1951 to 1961 period. So I really can't comment on that.

42/ R. Crump Jr., Vol. XXVI at 20.

43/ J. Pete Tr., Vol. XXVB at 65.

Despite not having seen the British data, Mr. Pete was quite willing to claim the Rochdale conversions were of more value than those made for the Quebec mines. J. Pete Tr., Vol. XXVA at 84, 97-99; Vol. XXVB at 60-69.

Dr. Finkelstein, however, noted:

> I haven't seen the British factory data. Based on what I've seen of the data from this plant, I am rather skeptical about the accuracy with which, you know, the British hygienists were able to make their ratios.

M. Finkelstein Tr., Vol. XXIV at 52.

Dr. Weill and his colleague Dr. Yehia Hammad have examined the particle-fiber conversion issue in the asbestos-cement plants studied in the Tulane University team's morbidity and mortality studies.[44] From 102 paired particle and fiber measurements made in various areas of the plants correlation coefficients ranging from 0.18 to 0.91 were found. Significantly, the poorer correlations were found in those areas with the lowest counts, where, as the authors note, both measurement methods were close to their limit of sensitivity. In those areas with the highest measurements (which were nonetheless quite low by historical standards) meaningful correlations and similar dose-response relationships were found:[45]

> [I]f average concentrations of particle counts or fiber counts obtained by area samples are used to rank the exposure of workers into broad categories as shown in Table I, we find that we can reach almost the same conclusions by either method.

The Weill study thus provides additional evidence that meaningful conversions can be made from historical particle counts to modern fiber counting methods.[46]

44/ A.C.A. Hammad, Y., Tse T., Hammad, Y. Y., et al., "Evaluation of Dust Exposure in Asbestos Cement Manufacturing Operations," [illegible] Vol. [illegible] (1979). See also H. Weill Tr., Vol. IV at [illegible].

45/ Id. at [illegible].

46/ The Hammad-Weill data provides particle-fiber correlations of [illegible] as consistent as those found at the Rochdale textile plant. [illegible] it exercise fiber concentrations to particle concentrations [illegible] five areas measured varies only from [illegible] from determined in similar comparisons at Rochdale [illegible]

The work that has been done to correlate the historic particle counts to fibers thus has validity and meaning for risk assessment. Particularly for those historic measurements at high levels that are most important [illegible] developing dose-response relationships, the correlations are quite significant. As Dr. Crump noted in conclusion:[1]

> [T]here is a significant amount of information in the fiber concentrations based on particle counts, information which is valuable and certainly should be used.

C. Determination of Current Exposures

Equally as important in determining risks workers face today is assessment of current exposures. Determination of a reasonable regulatory framework can proceed only in the context of what likely exposure levels will be achieved and how many persons will be affected. Such an assessment requires consideration of:

1. The method by which exposures will be measured;
2. The likely exposure levels that will be achieved in meeting a given standard; and
3. The average working lifetime of workers.

Description of these factors is important to determining the actual risks that might occur with a given standard.

[illegible] E. Crump Tr., Vol. XXVI at 1".

1. Exposure Measurement.

Changes have occurred over the years in workplace fiber count measurements. Two changes, in particular, have resulted in a significant de facto tightening of the workplace standard in the United Kingdom. First, fibers are now counted with use of a graticule eyepiece rather than whole field viewing; and, second, personal, rather than area, monitoring is now employed. Both changes, each of which leads to higher counts, were discovered and relied upon by the Simpson Report in its determination of appropriate workplace standards:[2]

> Studies have shown that personal sampling increases the number of fibres counted in a given workplace by a factor which varies widely both within and between manufacturing processes, but which, at dust levels around 2 fibres/ml is probably between 1 and 2 ... The results of fibre counts are [also] affected by the method of counting used. The current method of "eyepiece graticule" counting has been introduced since 1969. When compared with the older "whole field" counting technique, graticule counting increases fibre counts by a factor which has been estimated as being of the order of 2-3 ...

As the Simpson Report noted, workplace concentrations today based on personal monitoring counted with use of a graticule

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[2] R.C.A. Exhibit 28, Tab 5, Simpson Report (1977), quoted note 1, Vol. II at 12. The Simpson Report findings are not surprising in light of the common-sense reality that high membrane filter readings are often undercounted.

eyepiece will register up to five times higher than those same concentrations would have registered 20 years ago:[47]

> The combined effect of these last two factors means that an atmospheric chrysotile concentration which would have been evaluated as 2 fibres/ml in 1968 might well today be evaluated as somewhere between 4 and 10 fibres/ml. So it follows that these changes in sampling and evaluation techniques have brought about a *de facto* tightening of the hygiene standard since 1969.

In general, the shift from whole field to graticule counting techniques has less significance in interpreting North American data where the graticule reading method has always been generally employed. The significance of this change in interpreting the British data from Rochdale, however, is large, as noted in Chapter III.

The shift from area to personal sampling is significant to interpreting data from both sides of the Atlantic. Only in the most recent years have data in most plants been compiled on the basis of personal sampling; much of the historical data reflect area samples, which as the Simpson Report concluded, are likely to underestimate actual worker doses by a factor up to two. As Dr. Gibbs noted:[48]

47/ Id.

48/ G. Gibbs Tr., Vol. XXIX at 75. Drs. Chase and Rhodes agreed that a factor of two approximately expressed the underestimation of personal exposures by area sampling. H. Rhodes and G. Chase Tr., Vol. XXVII at 118-120.

> All of those factors add up to the fact that what we are counting today has increased our concentration, observed concentration, over what it was then. One can argue about the order of magnitude of it. Possibly Steel's, I've heard figures like Steel's discussed in other settings, and maybe one is talking about something like four to one, maybe overall three, four to one change.

Geoffrey Berry has noted a further effect of reliance on area samples that "take no account of individual work-styles" -- the bias toward low level risks when they are used for risk extrapolation at lower dust levels:[51]

> The reason for this bias is that some men with disease would be recorded as having a lower exposure than they had received. Of course, the opposite would also occur, but the errors do not produce a flatter dose-response curve, which would intersect the true dose-response curve near to the mean exposure. Thus, the health risk is exaggerated at low levels.

It is thus important to take into account the area or personal nature of any exposure data in any of the studies, as well as the microscope counting method, before proceeding to extrapolate risks on the basis of such data.

2. Expected Exposures.

Under any given standard, a company seeking to stay in compliance will have to achieve average plant levels well

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51/ R.C.A. Exhibit 13, Tab 7, Berry, G. and M. Lewinsohn, "Dose-Response Relationships for Asbestos-Related Disease," 330 Ann. N.Y. Acad. Sci. 185-94, at 191, 192 (1979).

below the standard. As a result, most workers will have average exposures considerably lower than the standard. This natural outcome of standard setting will occur for a variety of reasons.

First, the difficulty of achieving any given exposure level will vary from worksite to worksite. Given that a plant will be required to be in compliance at all sites, those steps necessary to bring the worst site in compliance will, inevitably, reduce exposures at other sites to even lower levels.

Second, occupational monitoring of asbestos at current levels is subject to considerable uncertainty and unreliability. As discussed in more detail in Chapter VI below, this measurement uncertainty requires a plant to maintain average exposures well below any given standard in order to be able to guarantee consistent measurements below, and thus legal compliance with, the standard.

Recognizing these factors, the Simpson Report assumed that the average worker would experience exposures one-tenth of any given standard.[1] As Dr Acheson told the Commission:[2]

> [I]f you set a standard ... and let's take the present standard so as not to be at all contentious ... the present

[1] E.P.A. Exhibit 28, Tab 9, Simpson Report (1979), supra note 14, Vol. I at 44, Vol. II at 18.

[2] 3 Acheson Tr., Vol. XIX at 20.

> standard in England of two fibers. ... [M]any fewer than all the people in the factory at which that standard is enforced will be exposed to two fibers. Perhaps a tenth of them.

The relevant question in standard setting is not therefore what the risk will be at, for example, a two fiber exposure level, but, rather, what will be the risk at the considerably lower average exposure level that will be the natural consequence of a two fiber standard.

Determination of cumulative exposures for workers at non-fixed worksites (as in the construction industry) or even fixed worksites where asbestos exposure will be only intermittent (as in garages where brake mechanics will be working with asbestos friction material) is more complex. In such situations, the primary exposures to asbestos will be intermittent, occurring during cutting operations, removal of asbestos from brake linings, or other activities that occur infrequently but if not performed in accordance with good work practices can lead to short bursts of exposure.

Considerable data exists on what level of cumulative exposure might be expected among asbestos-cement pipe installers and serves as a good example of what cumulative exposures could be expected in these worksites where exposure is infrequent. The asbestos-cement data show that the average worker will be involved in operations that emit

asbestos for less than 10 hours per year,[54] and that peak concentrations during those periods for most operations (with the prominent exceptions of use of a Doty Machine or abrasive disc saws will be less than 1 fiber/cc.[55] When

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54/ The Research Triangle Institute (RTI) has estimated that one cutting or machining operation is required for each 1,300 feet of installed asbestos pipe. Research Triangle Institute, Asbestos Dust: Technological Feasibility Assessment and Economic Impact Analysis of the Proposed Federal Occupational Standard, III-13 (September 1978). Dividing the annual production of asbestos cement pipe in the U.S. by this figure yields an estimate of 69,230 cutting or machining operations annually.

A typical work crew for installation of asbestos cement pipe is three to four people, and a crew can install an average of 222.5 feet of pipe per day. Id. If one assumes that 225 days per year of labor are expended installing asbestos cement pipe, approximately 6,130 man-years are devoted to this installation.

The estimated 69,230 cutting or machining operations take an average of 15 minutes each to perform. Thus, about 17,307 hours will be spent annually on cutting and machining operations that have the potential to produce exposure to asbestos fibers. If an average of 3.41 workers are exposed to these operations, the result will be 59,016 man-hours of exposure (0.53% of total work hours). This is a liberal estimate because not all workers will actually perform field operations.

If one makes the extreme assumption that a discrete group of workers installs asbestos cement pipe and no other type of pipe, these workers perform 9.3 hours per year of operations that may result in asbestos exposure (1800 hours x 0.53%).

If one adopts the more realistic assumption that the workers who install asbestos cement pipe also install other types of pipe, and vice versa, the frequency of an individual worker's exposure to asbestos fibers is much lower. Under this scenario, the typical worker will have 1.38 hours per year in which he may be exposed to asbestos.

55/ A study by Equitable Environmental Health, Inc. (EEH) measured peak exposures for various operations during installation of asbestos-cement pipe. Equitable Environmental Health, Inc., "Dust Exposures During the Cutting and Machining of Asbestos/Cement Pipe: Additional Studies," (December 15, 1977).

Most cutting, machining and tapping operations produced very low fiber concentrations. Only cutting with an abrasive disc saw and frequent machining with a Doty tool were found to result in peak concentrations in excess of 1.0 f/cc.

these time and exposure level data are combined, it can be determined that time-weighted average exposures will be less than 0.1 fiber/cc for all workers except those rare (and perhaps non-existent) workers who spend their entire time doing nothing but installing asbestos-cement pipe using nothing but an abrasive disc saw for every operation in the installation process.[56]

Thus, asbestos-cement pipe installation workers would have average exposures in almost all circumstances (and all circumstances if good work practices -- including not using an abrasive disc saw -- were employed) below 0.1 fibers/cc. Thus, even if a worker spent a 40-year career installing such pipe, his cumulative exposure would be well below 4 fiber-years.

3. The Exposed Population.

Central to the estimation of risk posed by future asbestos use is characterization of the population likely to

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56/ Based on the RTI and EEH data, supra notes 54 and 55, and even using assumptions that overstate the exposure level attributable to a specific operation, only cutting with an abrasive disc saw produces concentrations in excess of 0.1 f/cc as a time weighted average exposure. And, even a worker who installed only asbestos cement pipe and who used an abrasive disc saw for every operation would have a maximum time weighted average exposure of only 0.34 f/cc.

If it is assumed that half the field operations performed are machining rather than cutting operations, a worker who cuts pipe only with an abrasive disc saw would receive a time weighted exposure of less than 0.2 f/cc. Furthermore, if these workers also install other types of pipe, even exclusive use of the abrasive disc saw for cutting operations will not produce time weighted average exposure levels in excess of 0.1 f/cc.

to exposed. Most asbestos risk assessments to date have referred to the 50-year worker, exposed throughout his working life. It is clear, however, that this hypothetical worker does not represent the typical worker and that any risk calculation for such a worker would grossly exaggerate likely health consequences.

First, harking back, perhaps, to the 1960 BCMS asbestosis study, it has become common practice in asbestos risk assessments to talk of the risks of 50 years of exposure. Such assessments have no basis in fact. Rare will be the worker who is employed in an asbestos environment for anything like 50 years. As Julian Peto noted to the Commission, 25 year working lives would be much more realistic.[57/]

Second, only a small number of workers will be in settings with prolonged exposures to asbestos. Indeed, the portion of the workforce likely to experience other than intermittent exposures is confined to miners and millers and primary and secondary manufacturing workers. Both sectors employ few individuals.

No mines and mills currently operate in Ontario; in the United States, the three operating mines employ fewer than 600 workers;[58/] current employment in the Canadian chrysotile

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57/ J. Peto Tr., Vol. XXVII, at 21

58/ See United States Department of Health, Education and Welfare, "Asbestos: An Information Resource," 41 (May, 1979) (hereinafter cited as "DHEW").

mines is approximately 2,300.[59/] The number of workers manufacturing asbestos products is also small. Estimates of the size of this workforce in the United States have consistently put it around 35,000.[60/] In Ontario, the number of workers exposed in such factories is somewhat below 1,000.[61/]

Thus, the workforce that even theoretically could have the type of steady exposures to asbestos that might lead to accumulation of a fiber-year exposure per year under existing conditions is quite small. In fact, most such workers would average well below 1 fiber/cc average exposures under current 2 fibers/cc standards.[62/] It would thus be unlikely even for any significant portion of this small group of workers to work long enough at high enough exposures to have lifetime exposures greater than 10 fiber-years.

A much larger number of workers is potentially exposed to asbestos intermittently. That population, which would

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59/ Brief to the Commission of the Quebec Asbestos Mining Association 3 (Jan. 14, 1981).

60/ OHEW, supra note 58, at 41.

61/ The Ministry of Labour noted in its brief to the Commission that a total of 14,000 workers in the province are under medical surveillance, but only a small proportion work at the then-operating 15 asbestos operations. Ministry of Labour Brief, at 7-8 (Feb. 1981). Within the last two years, however, the number of workers under medical surveillance has dropped to 8,000 and the number of employers has dropped from 191 to 174. P. Palmour Tr., Vol. XLIIB at 6, 7.

As determined by Dr. Bragg, "The Technical Feasibility and Cost of Controlling Workplace Exposure to Asbestos Fibres," at 5-4 (Ont. Commission Study, April 1982), only a small percentage of these workers, at 14 companies are in asbestos manufacturing facilities.

62/ See Chapter VI, infra.

include construction workers and garage mechanics, has been variously estimated in the United States to be two to three million.[42/] In Ontario, the Ministry of Labour has estimated there are 230,000 construction workers.[43/] Although the proportion of these workers who could occasionally be exposed to asbestos is not known, it is unlikely to be large.

For these workers, average exposures are well below those in the primary manufacturing setting when approved work practices are employed. Rare would be the worker in such settings with average exposures over 0.1 fiber/cc.[44/] Thus, although this portion of the workforce might be large, its lifetime exposures will almost all be below 4 fiber-years.

Estimating risks for future use of asbestos thus involves two basic populations: a very small workforce whose lifetime exposures could range up to 10 fiber-years; and a larger workforce whose lifetime exposures will be below 4 fiber-years.

D Predicted Risks.

As detailed above, it becomes clear that definitive determination of the risk posed by continued asbestos use cannot be made. The substantial evidence that thresholds may exist at levels above current exposures argues strongly

42/ OHEW, supra note 58, at 47-48.

43/ Brief to the Commission of the Ministry of Labour, supra n. 41, at 8.

44/ See discussion at pp. IV-42 to IV-43.

for the existence of no risk. Nonetheless, risk assessment techniques may be used to put upper limits on potential risk. A number of such risk assessments were presented during the Commission's hearings. Each was based on linear, non-threshold models of the dose-response relationship, and thus each conservatively overestimated potential risk.

As the risk assessments presented by Dr. Nicholson and Dr. Acheson demonstrated, differing assessments of the magnitude of risk arise based on different assumptions and use of different epidemiology studies. The Simpson Report lung cancer risk assessment based on three studies and several assumptions for each study has a 30 to 35-fold range from its high to its low estimate of lung cancer risk.[45/] Dr. Nicholson's calculations from nine different studies shows a 1000-fold range in estimated risk.[46/]

It is clear, however, that little reliance should be placed on some of the studies and assumptions used in these assessments. Dr. Nicholson's review of the data, in particular, includes calculations based on several studies that are all ill-suited for predicting the risks of future asbestos use. First, he includes Dr. Selikoff's insulator study, the Paterson amosite insulation factory study, the early Newhouse

45/ E.C.A. Exhibit 28, Tab 5, Simpson Report (1979), supra note 14, Vol. II at 75, Tables 35, 36.

46/ E.C.A. Exhibit 19, Tab 9, Nicholson, W., "Dose-Response Relationships for Asbestos and Inorganic Fibers," Table 1- (1981).

and Berry Cape Asbestos manufacturing plant study, and his own chrysotile mining study, although there is no quantitative data on the intensity of individual exposures of any of these groups (indeed, for the insulators, knowledge of duration of exposure does not exist). Second, some of these studies are also of limited usefulness because the type of exposure experienced varies significantly from that of current workers. The insulators, for example, were exposed to a variety of types of asbestos (including amphiboles) intermittently with high peak exposures. The amosite insulation workers were exposed exclusively to the one form of asbestos not used in commerce today at all, also for short periods of intense exposures.

Dr. Crump also presented to the Commission an assessment of lifetime occupational risks for workers in primary asbestos industries. Dr. Crump was able to perform his most precise assessment on the basis of the McDonald miner studies, as those studies have been published in the most detail with the most pertinent information.[68]

Dr. Crump first assessed the lung cancer risk for a worker who was exposed for at least twenty years in a setting that was meeting a 2 fibers/cc standard. Because of the

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68/ Dr. Crump did note, however, some additional data in this cohort that could probably be determined from the existing unpublished data base that would be useful for refining the assessment, e.g., precise exposure data for each subcohort in the study. E. Crump Tr., Vol. XXVI at 45.

vast discrepancy in risk for smokers and non-smokers, and the existence of data to make that distinction in the McDonald study, Dr. Crump determined the risk for each group separately. In order to make the assessment, he made the following assumptions:[69]

-- The worker would have at least 35 years of exposure from age 20 to 45.[70]

-- The lifetime particle count measurements should be converted to fibers by multiplying by three.[71]

-- Average exposure levels would be half the standard, i.e., 1 fiber/cc.

-- Compliance with the standard would be monitored by personal sampling, which would average twice what the historical mining area samples measured.

Based on these assumptions, Dr. Crump calculated the following risks from such lifetime exposure:[72]

69/ Id. at 49-50.

70/ Dr. Crump used this basis for exposure because the most relevant information from the McDonald data base for risk assessment -- interpretation of cumulative exposures and health outcomes with stratification of the cohort based on smoking habits -- has been presented by Dr. McDonald in terms of exposure through age 45. To the extent workers were exposed to even more asbestos beyond age 45, the risk assessment [illegible] data base overestimates risk per unit of exposure. [illegible]

71/ As Dr. Crump explained, this conversion [illegible] average conversion calculated by Dr. McDonald [illegible] conversions for each job and area in his [illegible] Exhibit 18, Feb 24, McDonald (1980), supra note 2, at [illegible] Crump Tr., Vol. XXVI at 49.

72/ E. Crump Tr., Vol. XXVI at 51-1, R [illegible]

LOSS OF LIFE EXPECTANCY BECAUSE OF LUNG CANCER FROM LIFETIME OCCUPATIONAL EXPOSURE UNDER A 2 f/cc STANDARD BASED UPON DATA OF McDONALD (1980)

	Loss of Life Expectancy (Days)	
Non-smokers	Using Data From	
	British Doctors	U.S. Veterans
Best Estimate	0.16	0.30
95% Upper Limit	0.25	0.31
95% Lower Limit	0.07	0.09
Smokers		
	British Doctors (avg. 18.1 cigs./day)	U.S. Veterans (21-39 cigs. /day)
Best Estimate	1.9	2.3
95% Upper Limit	3.0	3.5
95% Lower Limit	0.83	1.3

The computed lung cancer risks are obviously very small. For non-smokers, average loss of life expectancy would be well below one day, and for smokers the risk would still be but a few days. Comparing these risks to the loss of life expectancy of 690 days among smokers for respiratory disease alone, Dr. Crump noted:[1]

> [W]e see that the smokers' risk of lung cancer is affected only very slightly by their asbestos exposure in this situation.

[redacted]

1/ K. Crump Tr., Vol. XXVI at 5-.

By also determining the risks of other diseases found in excess among the miners, Dr. Crump proceeded to calculate the entire mortality risk -- including an unrealistically high estimate for asbestosis based on linear extrapolation -- that are suggested by the McDonald data base for lifetime workers under a two fiber standard:[2]

LOSS OF LIFE EXPECTANCY FROM VARIOUS ASBESTOS-RELATED DISEASES CAUSED BY LIFETIME EXPOSURE UNDER A 2 f/cc STANDARD BASED UPON DATA FROM McDONALD (1980)

	Loss of Life Expectancy (Days)	
	Non-smokers	Smokers
Lung Cancer	0.16	1.9
Cancer of Stomach or Esophagus	0.30	0.30
Pneumoconiosis	0.43	0.26
Combined Lung Cancer, Cancer of Stomach or Esophagus, and Pneumoconiosis	0.89	2.4

Extrapolation from the results of the McDonald study thus would predict risks under a 2 fiber standard that are clearly

[redacted]

2/ Id. at 54-55; E.C.A. Exhibit 38, Tab 3, Table 1.

is similar. The risks from all causes[73] for smokers is less than two and one-half days loss of life expectancy and for non-smokers is less than one day. Indeed, the asbestos risk is but a small percentage of the accident risk in the safest of occupations (with occupational accidents in trade professions posing 30 day life expectancy loss risks) and a completely insignificant part of the risk of accidental death in riskier occupations (such as mining where the occupational accident risk is a loss of life expectancy of 128 days). As Dr. Crump noted:[74]

> [F]or occupational accidents, these estimates, as you can see, range from thirty days to three hundred and twenty-eight days. It's interesting that the highest risk comes from mining and quarrying. I'm not sure if these can be applied to the Quebec miners or not, but if they can, it's obvious ... that their risk from asbestos exposure, given that these estimates are correct, would be a very minor portion of their total occupational risk.

Moreover, the risks predicted from the McDonald data are likely to exaggerate true risk, as they:

-- Are premised on a conservative, linear non-threshold dose-response model.

-- Assume no threshold for asbestosis-related deaths.

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[73] Dr. Crump was unable to include the mesothelioma risk because the relevant exposure data has not been published, but testified that this additional risk would be approximately another one-half day for both smokers and non-smokers. H. Crump Tr., Vol. XXVI at 56.

[74] Id. at 52-53.

-- Assume average exposures under a two fiber standard of 1 fiber/cc, when in fact average exposures will likely be only one-half that high, if not one-tenth.

Although the published data are much less comprehensive, Dr. Crump also was able to calculate rough approximations of the lung cancer risk predicted by the two studies of asbestos textile plant workers. These predictions again are likely to overestimate risk for the same reasons as the extrapolations from the McDonald data did. Accepting at face value the gross average exposures for workers at Rochdale and Charleston, Dr. Crump calculated the following lung cancer risks from the studies of Mr. Peto and Dr. Dement:[77]

LOSS OF LIFE EXPECTANCY FROM LUNG CANCER
SUBJECT TO A 2 f/cc STANDARD

	Loss of Life Expectancy (Days)	
	Non-Smokers	Smokers
Estimated from Peto (1980)	3.5	41
Estimated from Dement (1980)	26	290

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[77] Id. at IV-19; R C A. Exhibit 38, Tab J, Table 14.

As discussed in Chapter III, considerable doubt exists as to the accuracy of the exposure data in, and some uncertainty arises given the small size of, these two studies of textile plants. In addition, considerably less data on the dose response curves are available than from the McDonald study; and, the Commission's experts expressed many doubts about the unexpectedly high results of Dr. Dement's study. The risk assessments from those data are thus useful only as preliminary indicators of where they fit into the overall picture.

There does, however, appear to be a substantial discrepancy between predicted risks in the chrysotile mines and textile plants.[78/] Nonetheless, even these much higher risks predicted in the textile plants, when compared to a number of other risks, do not fall out of the range of common acceptance. As Dr. Crump explained:[79/]

> [G]iven using the Dement study the loss of life expectancy looks like it's going to be not a very sizeable fraction of the loss already incurred from the smoking without the asbestos exposure, although certainly a much larger fraction that it was using the McDonald data. ...

78/ The range between predicted lung cancer risks from the three -- McDonald, Peto and Dement -- studies as calculated by Dr. Crump and Dr. Nicholson are quite similar. For smokers, Dr. Crump finds the Peto study predicts risks about 20 times what the McDonald study predicts, and the Dement study predicts risks about 1-5 times what the McDonald study predicts. Dr. Nicholson's risk estimates for lung cancer risk in the Peto study predicts 13 times, and the Dement study 11 times, as much risk as the McDonald study.

79/ W. Crump Tr., Vol. XXVI at 31.

> [T]hese numbers are still the same orders of magnitude as the numbers -- loss of life expectancy from occupational accidents.

As Dr. Crump indicated, even when the exaggerated risks from the Dement study are compared with the table of life expectancy losses from other causes at page IV-9 above, it can be seen that lifetime exposure work in a textile plant is less dangerous for non-smokers than the risks of accidental death in most occupational settings. Even for smokers, such work is less dangerous than some occupations. And, even the exaggerated lung cancer risk from the Dement study for smokers, is less than the risk of cigarette smoking.

In light of such risk assessments, it is not surprising that the British Simpson Report was able to conclude that a one fiber/cc standard would acceptably protect workers. Appearing before the Commission, Dr. Acheson reaffirmed his belief in the reasonableness of such a standard:[80/]

80/ D. Acheson Tr., Vol. XIX at 43.

Dr. Acheson noted that several studies that include quantitative exposure information have become available since the British Committee computed its risk assessment. Noting that the Dement study would predict risks at the high end of the Simpson Report's risk assessment range and the Rhode Island plants study would predict risks at the low end, Dr. Acheson confirmed his continued willingness to rely on the Simpson Report's risk assessment:

> So you've got, in fact, as it were ... if they were added to this table, one would be at the bottom and one would be at the top.

Id. at 44.

I have heard nothing which makes me change my view that the one fiber standard is a reasonable standard for chrysotile, and a standard which the industry can adhere to without difficulty.

As discussed in Chapter III, considerable support emerges from some of the best documented epidemiology studies, including those of Drs. McDonald, Weill, Newhouse and Berry, and Enterline, that a threshold exists below which detectable excess deaths will not occur among asbestos workers. In each of these studies, that level of asbestos is well above current exposure levels. Nonetheless, given the uncertainty of health consequences at such low levels and the absence to date of documented studies of workers exposed for long periods at such levels, there is a role for risk assessment techniques to put upper bounds on what potential risks may be posed. As the discussion in this Chapter demonstrates, such upper bounds are not outside the range of generally accepted occupational risks. The risks predicted from the McDonald study, for example, are well below what is commonly accepted in even the safest of occupations. Although larger risks are predicted from some of the studies, most prominently those in asbestos textile plants, even these risks are not out of the range of generally accepted occupational risks.

CHAPTER V

V. THE ABSENCE OF GENERAL POPULATION RISKS.

Throughout the Commission's hearings, considerable debate occurred about determination of occupational asbestos risks. As the preceding chapter notes, some differences of opinion existed in exactly how much risk exists at today's low occupational exposures. Moving from the occupational setting to general environmental exposures, however, clear consensus emerged from the Commission witnesses as to the absence of any risk worthy of governmental attention. This conclusion was reached both generally in terms of ambient air and water exposures and more particularly with respect to potential exposures in buildings constructed with asbestos-containing materials.

A. Ambient Exposures.

No dispute arose during the summer over the proposition that ambient exposures to asbestos do not pose any public health hazard. As Dr. Corbett McDonald expressed it:[1]

> I suppose it then might be that, as a result of going by train, there is one more lung cancer a year in Britain than there would be otherwise, you know, or perhaps more than that; I've no idea.
>
> It's this kind of thing. ... [I]t seems to me so totally unbalanced to

[1] J. McDonald Tr., Vol. XII at 115-16.

> consider that sort of risk in relation to what is probably an infinitely bigger risk from sitting next to somebody who smoked a cigarette -- and I don't mean smoking it yourself.

Dr. Acheson noted explicitly the absence of general population risk for any of the asbestos-related diseases, including mesothelioma:[1]

> Mesothelioma is, in any event, a rare disease if one, like the country as a whole -- either the United States or Britain ... and the proportion of the cases that can be attributed to domestic or neighborhood exposure is very small.
>
> So that for various reasons, in our report we did not feel that there was or was likely to be a major public health risk outside industry in relation to mesothelioma;

asbestosis:

> Nor, for the reason I have mentioned, in relation to asbestosis;

and lung cancer:

> For lung cancer, the problem is more difficult because it's a common disease and you can't easily pick up asbestos-attributable cases. However, by extrapolation from the quantitative data, using the linear model ... and to cut a long story short, our view is that in general the dangers of asbestos in the open air are minimal, even in our great cities, the danger in buildings, with one or two important provisos, are quite small.

[1] J. Acheson Tr., Vol. XII at [illegible]

The conclusions drawn by the Commission's expert witnesses should come as no surprise. Numerous official scientific bodies over the years have reviewed the evidence on asbestos to determine whether any risk was posed to the general public. Their consistent finding has been that the low levels likely to be experienced by the general population pose no significant risk.

In 1971, the National Academy of Sciences in the United States prepared a report collecting all available information on asbestos as an air pollutant. In its Conclusion and Recommendations, the NAS panel notes:[1]

> [T]here are levels of inhaled asbestos without detectable risk. It is not known what range of respirable airborne asbestos fibers will ultimately be found to have no measurable effects on health. At present, there is no evidence that the small numbers of fibers found in most samples of the general environment affect health or longevity.

The National Academy of Sciences Safe Drinking Water Committee similarly concluded in 1977:[2]

> The available data with respect to asbestos orally ingested through drinking water do not suggest an immediate hazard to public health.

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1 National Academy of Sciences, Asbestos: The Need for and Feasibility of Air Pollution Controls [illegible] (1971) (emphasis supplied).

2 NAS Safe Drinking Water Committee, Drinking Water and Health: Full Report: at [illegible] (1977). See also NAS Safe Drinking Water Committee, Summary Report: Drinking Water and Health (1977).

The International Agency for Research on Cancer, after surveying and evaluating all the available published literature on asbestos, also concluded:[3]

> At the present time, there is no evidence that exposure of the general population to past levels of asbestos dust in the ambient air or in beverages, drinking-water, food or pharmaceutical preparations increased the risk of cancer.

The IARC did not retreat from this basic conclusion in a subsequent review published in 1977.[4]

In 1977, after a thorough review of the available data, a working group of experts reported to the Commission of the European Communities:[5]

> [T]here is no established evidence that oral ambient exposure through air, water, drugs, beverages, food, as prevalent in Western European countries at this moment carries ... a definite (health) risk; however there exist too many uncertainties to deny such a risk, though if the risk was substantial, it is likely it would have been detected by now.

3/ IARC, Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man, Vol. 1, p. 35 (1973).

4/ IARC, Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man, Vol. 14 (1977).

5/ Commission of the European Communities, Public Health Risks of Exposure to Asbestos [illegible] (1977) (emphasis in original).

Technological Assessment by the distinguished British scientists Sir Richard Doll and Richard Peto reaches the same conclusion:9/

> The amount [of asbestos] commonly present in town air is, however, three or four orders of magnitude less than the most stringent regulations currently permit[ted] at work, and the risk to the general public from this source is unlikely to increase appreciably the total number of cancers attributable to other forms of air pollution.

In short, the uniform opinion of these respected authorities, each of which has investigated the issue in depth, is that there is an absence of evidence indicating exposure to asbestos from the ambient environment poses any public health problem.

Among the evidence supporting these conclusions are several studies of general population mortality in United States counties where natural asbestos concentrations are found and among residents of an area near New Jersey surrounding an asbestos factory.

No adverse health effects were found among Patterson, New Jersey, residents living near the amosite asbestos factory studied by Siedman and Anderson. Based on dust counts made years after the factory had closed, the authors found that:10/

9/ Doll, R. and R. Peto, "The Causes of Cancer: Quantitative Estimates of Avoidable Risks of Cancer in the United States Today," 66 J. NCI 1191-308, 1268 (1981).

10/ Seemoos, E. C. et al., "Mortality Experience of Residents in the Neighborhood of an Asbestos Factory," 330 Acs. N.Y. Acad. Sci. 417-32, at 417 (1979).

Finally, the United Kingdom's Simpson Report states:1/

> [T]here is no quantitative evidence of a risk to the general public from exposure to asbestos dust.
>
> * * *
>
> [I]t is concluded that the presence of chrysotile alone or contaminated with small quantities of amphiboles is unlikely to have produced any material increase in the risk of lung cancer in the general population or any appreciable number of cases of mesothelioma. The same is certainly true of asbestosis.
>
> * * *
>
> An excess lung cancer risk cannot be completely excluded at present in those who have been exposed only to amphiboles in buildings. But the number of cases, if any, is probably very small ... .

A recent comprehensive study of all environmental causes of cancer written for the U.S. Congress' Office of

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1/ U.K. Exhibit 28, Tab 5, British Advisory Committee, Asbestos (Simpson Report), Vol. I at 89, Vol. II at 13 (1979).

The Report continues:

> In conclusion of this section it must be said that it is our opinion that it is on the whole unlikely that there is a material public health risk of mesothelioma in relation to the inhalation of amphiboles and mixtures rich in them. The small absolute number of mesotheliomas registered in comparison with other causes of death, in spite of the widespread use of amphiboles in Britain for at least 50 years, the slow rate of increase of these cases, the improvement of industrial practice in the last 20 years, and a number of other favourable points which have been mentioned above, all give grounds for optimism.

1a. Vol. II at 4.

> it is safe to assume that people living in the neighborhood of the factory were exposed to asbestos dust, obviously an extremely light exposure as compared with the exposure of men working in the factory.

From this light nonoccupational exposure -- which was still likely to be much greater than the average consumer's exposure to asbestos, given the description of this factory made to the Commission by Drs. Anderson and Nicholson[11] -- the authors found no significant adverse health effects. The mortality statistics for the exposed community (Riverside) as compared with the control community (Ottawa) were virtually identical. The reported deaths from lung cancer and colon-rectum cancer were actually higher in Ottawa than in Riverside.

Although limited in its detection powers, the only study of general population risks caused by ambient asbestos exposures also fails to find any association between asbestos exposure and cancer mortality. A 1976 study by T. R. Fears of the National Cancer Institute compared cancer mortality rates in U.S. counties with natural asbestos deposits to rates in similar counties with such naturally-occurring asbestos. Fears reports:[12]

> This study provides no evidence that naturally occurring asbestos deposits provide a great risk to the general population of counties with asbestos deposits.

11/ See pp. III-70 to III-71, supra.

12/ Fears, T.R., "Cancer Mortality and Asbestos Deposits," 104 Am. J. Epidem. 323, 326 (1976).

Studies of the asbestos body burden of the lungs of lung cancer cases and of controls also lend support to the view that asbestos occurring in the general atmosphere plays no causal role in lung cancer. Two studies have found no significant difference between the asbestos body burden of the lungs of those with lung cancer as compared with the lungs of controls.[13] As described by Dr. Becklake:[14]

> Using a case-control approach, these workers recorded the number of asbestos bodies in lung samples obtained at autopsy or at thoracic surgery in patients who died with one of these diagnoses (lung or gastro-intestinal cancer) and in equal numbers of matched controls who died from other causes. While the number of asbestos bodies so recorded was related to sex (higher in men) and

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13/ B.C.A. Exhibit 33, Whitwell, F., et al., "Relationship Between Occupations and Asbestos-Fibre Content of the Lungs in Patients with Pleural Mesothelioma, Lung Cancer, and Other Diseases," 32 Thorax 377-86 (1977); Denisch, I., et al. "Prevalence of Asbestos Bodies in a Necropsy Series in East London: Association with Disease, Occupation and Domiciliary Address," 32 Br. J. Industr. Med. 16-30 (1975).

Warnock, M. L. and A. N. Churg ("Association of Asbestos and Bronchogenic Carcinoma in a Population With Low Asbestos Exposure," 35 Cancer 1238-42 (1975)) reported a contrary finding, but their cases and controls were poorly matched for sex since 77% of the cases, but only 49% of the controls were male, and because asbestos bodies are more commonly found in the lungs of men than in those of women (B.C.A. Exhibit 29, Tab 7, Becklake, M. R., "State of the Art: Asbestos-Related Diseases of the Lung and Other Organs: Their Epidemiology and Implications for Clinical Practice," 114 Am. Rev. Resp. Dis. 187-227, at 200 (1976)). More careful matching has since reversed this finding: See Churg, A. N., and M. L. Warnock, "Numbers of Asbestos Bodies in Urban Patients with Lung Cancer and Gastrointestinal Cancer and in Matched Controls," 76 Chest 163-69 (1979).

14/ B.C.A. Exhibit 29, Tab 9, Becklake, M., "Environmental Exposure to Asbestos: A Factor in the Rising Rate of Cancer in the Industrialized World?" 76 Chest 245-247 (1979).

> occupation (higher in those engaged in occupations where exposure is known to occur), it was not related to the presence of cancer in either of the sites examined.
>
> Evidently, these findings offer no evidence to support the indictment of environmental (i.e., occupational) asbestos exposure as a factor contributing to the occurrence rates of either of these cancers in urban dwellers. This is reassuring for us all.

There thus seems little doubt that no public health problem is posed by the diminishingly small asbestos concentrations in the ambient air. As a natural mineral found throughout the world, asbestos is among the substances to which man has been exposed for millions of years without adverse effect. No reason exists for public health officials to become concerned with general exposures at this time.

Similarly, substantial scientific data demonstrate the absence of any significant risks from asbestos in drinking water. As some of the quotations from official reviewing bodies above noted, no risks from asbestos in drinking water have ever been found to be of public health significance. The Commission's witnesses agreed. As Dr. Acheson noted about the conclusions of the British Advisory Committee:[15/]

> [T]he Committee as a whole tended to discount a risk relating to drinking water.

Dr. Corbett McDonald further noted that any possibility that asbestos from asbestos-cement water pipes would find its

15/ D. Acheson Tr., Vol. XIX at 93.

way into the drinking supply was of no public health concern:[16/]

> All that we're talking about is just superficial, minor hazards compared with the problems of a world which doesn't have water supplies; water supplies which they will not have if they don't have asbestos pipe.

No doubt because of the general consensus that asbestos in water poses no significant risks, little discussion of this issue occurred during the Commission's hearings. Nonetheless, a plethora of studies relevant to this issue have been reported. They indicate, first, that animals exposed to high doses of asbestos in their diets do not develop cancer.[17/] As Dr. Davis told the Commission:[18/]

> The simple result is ... that our rats can eat vast amounts of asbestos, for their full lifespan, and we find no gastrointestinal tumors.

16/ C. McDonald Tr., Vol. XIII at 119.

17/ See, e.g., Bonser, G. M., and D. B. Clayson, "Feeding of Dice Asbestos to Rats," in 45th Annual Reports of the British Empire Cancer Campaign, at 242 (1967); Gross, P., et al., "Ingested Mineral Fibers: Do They Penetrate Tissue or Cause Cancer?" 29 Arch. Environ. Health 341-47 (1974); Hilding, A. C., et al., "Biological Effects of Ingested Amosite Asbestos, Taconite Tailings, Diatomaceous Earth and Lake Superior Water in Rats," 36 Arch. Environ. Health 298-303 (1981); McConnell, E., et al., "NIEHS Feeding Study - Wanster Data (In Review)" (1981); Saffiotti, U. Z., et al., "Tests for Carcinogenicity of Asbestos," 132 Ann. N.Y. Acad. Sci. 436-48 (1965); Smith, W. E., et al., "Health of Experimental Animals Drinking Water With and Without Amosite Asbestos and Other Mineral Particles," 3 J. Environ. Path. Toxicol. 227-303 (1980); Smith, W. E., "Asbestos, Talc and Nitrites in Relation to Gastric Cancer" 37 Am. Indus. Hygiene Ass'n. J. 227-228 (1973).

18/ J. Davis Tr., Vol. XXXIV at 98.

Second, epidemiology studies have been unable to detect any increased cancer risk among persons with higher asbestos levels in their water than control populations with lower asbestos levels.19/

In sum, both animal and human evidence and the considered opinion of scientific reviewing bodies confirm the absence of any risk from asbestos in water worthy of regulatory action.

E. Exposures in School Buildings.

Particular concern was expressed during the hearings about whether any risk calling for societal action existed in schools. Based on understandings that school exposures were generally below .01 fiber/cc, a number of witnesses

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19/ See, e.g., Levy, B. S., et al., "Investigating Possible Effects of Asbestos in Water Surveillance of Gastrointestinal Cancer Incidence in Duluth, Minnesota," 103 Am. J. Epidem. 362-68 (1976); Hasson, T. J., et al., "Asbestos-Like Fibres in Duluth Water Supply," 228 JAMA 1019-20 (1974); Sigurdson, E. E., et al., "Cancer Morbidity Investigations: Lessons from the Duluth Study of Possible Effects of Asbestos in Drinking Water," 35 Environ. Res. 50-61 (1984); Harrington, J. M., et al., "An Investigation of the Use of Asbestos Cement Pipe for Public Water Supply and the Incidence of Gastrointestinal Cancer in Connecticut, 1935-1973," 107 Am. J. Epidem. 96-103 (1978); Meigs, J. W., et al., "Asbestos-Cement Pipe and Cancer in Connecticut, 1955-1974," 42 J. Environ. Health 187-191 (1980); Wigle, D. T., "Cancer Mortality in Relation to Asbestos in Municipal Water Supplies," 32 Arch. Environ. Health 185-90 (1977); Toft, P., et al., "Asbestos and Drinking Water in Canada," 18 Science of Tot. Environ. 77-89 (1981); Severson, R., "A Study of the Effects of Asbestos in Drinking Water and Cancer Incidence in the Puget Sound Region," M. S. Thesis, Univ. of Wash., Seattle, Wash. (1979); Polissar, "Asbestos in Drinking Water and Cancer Incidence in the Puget Sound Area," Amer. J. Epidem. (In Press) (1982).

(Footnote 19 continued on next page.)

expressed their belief that such school exposures posed no risk worthy of action. As Dr. Acheson responded:20/

> Q. ... I gather it was the view of your Committee that buildings with levels of exposure like the other eighteen [buildings] ... meaning exposure levels below point zero one fibers per centimeter were probably not posing any significant risk to their occupants?
>
> A. Certainly they would not be, as long as the level stayed like that.

Julian Peto agreed:21/

> [T]he asbestos levels in the schools are usually a hundred, and ... no, I think probably always a hundred, and usually very much more than a thousand times lower than one fiber per ML, so the lifelong risk would be at least a hundred, and probably more than a thousand times lower than that, which gives you a risk which is likely to be less than one in ten to the five [one in a hundred

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(Footnote 19 continued from previous page.)

The only asbestos-in-water epidemiology study ever to find a positive association, Kanarek, M. S., et al., "Asbestos in Drinking Water and Cancer Incidence in the San Francisco Bay Area," 112 Am. J. Epidem. 54-72 (1980) and Conforti, P. M., et al., "Asbestos in Drinking Water and Cancer in the San Francisco Bay Area: 1969-1974," 34 J. Chron. Dis. 211-24 (1981), has not generally been accepted as probative of any risk from asbestos in water. Among the major problems with the study are its use of an ecologic design (i.e., attempting to correlate asbestos levels with census tracts with disease), the possibility that many factors may have confounded the results, inconsistencies in the reported data, and the absence of positive findings in any of the other epidemiology studies -- some of which were more sensitive than this study. See K. S. Crump, "Review of a Study of Asbestos in Drinking Water and Cancer in the San Francisco Bay Area," Report to the A/C Pipe Producers Association (1982).

20/ D. Acheson Tr., Vol. XIX at 94.

21/ J. Peto Tr., Vol. XXVA at 47, Vol. XXVB at 119.

thousand), which seems to me negligible. I wouldn't have thought that that was worth spending as much money as you have put in.

* * *

Q. Can you calculate what the average shortening of life would be for a one in a hundred thousand risk?

A. Yes. It's a thousandth of a month. It's about an hour, isn't it?

Indeed, Dr. Finkelstein of the Ontario Ministry of Labor urged upon the Commission the view that the emotional harm caused by public discussion of possible asbestos risks to school children was greater than the risk posed by the asbestos:[12]

> My feeling is that the risk to people working in schools, exposed to whatever ambient levels of asbestos there are in the schools, is probably inconsequential. ... I think this is possibly one instance where people have been worried ... the harm done by the psychological trauma has possibly been greater than the benefit derived in cleaning up the asbestos in the schools.

In most schools containing asbestos, Dr. Chatfield testified he has been unable to measure exposures above ambient levels.[13] There is thus substantial doubt that any risk at all is posed by the asbestos in Ontario schools.

12/ M. Finkelstein Tr., Vol. XXX at 19-23.

13/ E. Chatfield Tr., Vol. XVIII at 40. The recent British monitoring study in buildings, as discussed at pp. I-27 to I-28, also found no detectable asbestos, even with sensitive electron microscopy.

Nonetheless, it is obviously possible to extend risk extrapolations of the type discussed in Chapter IV from the exposure levels experienced in occupational settings to the much lower exposure levels that might exist in schools in order to obtain an upper limit on potential risk. In so doing, one moves from the extrapolation of risks at historical exposures averaging 10 to 20 fibers/cc to current workplace exposures between .1 and 2 fibers/cc, an extrapolation which as discussed in Chapter IV is itself subject to uncertainty, to an extrapolation over a much greater range. Little data exist on asbestos levels in schools, but it seems clear that except in a few extreme situations where badly damaged friable asbestos has been allowed to persist average exposures will be less than 0.0002 fiber/cc, and in fact are likely to be much less.[14]

Based on the same model and data as used for its occupational risk assessment, the Simpson Report calculated the lung cancer risk from 50 years of continuous exposure both

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14/ At the extremely low levels prevalent in schools, the membrane filter method is unuseable; see Chapter VI below. Most measurement has thus been done by electron microscopy with results expressed in nanograms/cubic meter. Among the data collected from buildings by such methods are those of Sebastien in Paris, where median exposure levels were typically around 10 nanograms. Sebastien, P., et al., "Levels of Asbestos Air Pollution in Some Environmental Situations," 330 Ann. N.Y. Acad. Sci. 401-415 (1979). Several conversion factors have been proposed between fibers/cc and nanograms/cubic meter. See N.C.A. Exhibit 28, Tab 5, Simpson Report (1979), supra note 8, Vol. II at 74, Table 32. Using the middle range of these conversions, 10 nanograms is equivalent to .0002 to .0005 fiber/cc.

at a likely exposure level of 17 nanograms.m^3, or approximately 0.0002 fiber/cc, and in the unlikely extreme situation where average exposures were 250 nanograms/m^3, or approximately 0.005 fiber/cc. Those results, which in themselves predict very small risk, need to be reduced by a factor of at least 20 to predict risks to school children, as they assume continuous exposure for 50 years. Reduction by a factor of 20 makes the calculation roughly comparable to the risk from spending one-fourth of one's life for 10 years in a school building. With that adjustment, the risks predicted for school residents are clearly de minimis. For such a school child with 10 nanograms/m^3 average exposures, the predicted risk would range from 1 in 3 million to 1 in 100 million. For the extreme school where average exposures were 250 nanograms/m^3, the risk would range from 1 in 100,000 to 1 in 4 million.

From these data the Simpson Report concluded:[13/]

13/ S.C.A. Exhibit 38, Tab 8, Simpson Report (1979), supra note 8, Vol. II at 17. The Committee also concluded the chrysotile mesothelioma and asbestosis risks were insignificant.

> The evidence that is available does not suggest that there is likely to be a risk of mesothelioma to the general public associated with chrysotile fibres, at least of the configurations used in the past . . . . It therefore seems unlikely that any appreciable number of cases of pulmonary impairment have arisen due to the contamination of buildings such as houses, offices and schools with chrysotile contained in the building materials.

> It can be seen that, unless contaminated buildings are very much commoner than seems likely, an appreciable mortality from lung cancer can be associated with any degree of contamination by chrysotile likely to be encountered in the UK in the ambient air or in buildings not under active construction or *repair*.

Although using a different data base and model, the risk extrapolation presented to the Commission by Julian Peto also predicts de minimis risks for school residents.[36/]

36/ Id. at 44. See R.C.A. Exhibit 37, Tab 10, Peto, J., "An Alternative Approach to the Risk Assessment of Asbestos in Schools: Report to the U.S. E.P.A.," (April 6, 1981). Mr. Peto's risk extrapolations are from the health experience of Dr. Selikoff's insulators, for whom he assumes average cumulative exposures of 600 fibers/cc-years. Id. at 6.

Mr. Peto's mesothelioma extrapolation model is based on the assumption that incidence will increase as a *factor of time since first exposure* cubed. Mr. Peto recognized in his Commission testimony that the Selikoff insulator data from which his model is derived indicate incidence rates may peak at about 45 years after exposure and then decline. R.C.A. Exhibit 37, Tab 9, Peto, J., et al., "Mesothelioma Incidence Among Asbestos Workers," Table 1 (unpublished). His model assumes continued incidence increases. Dr. Crump emphasized this failure of the Peto model to track the empirical data:

> That down turn, I think, could be a real down turn.
>
> It seems to me that it's somewhat unlikely that given you have an exposure, no matter what happens after that the risk keeps continuing on going up at this very rapid rate, even after exposure stopped.
>
> There are no data that I know of that give us any information on what happens more than thirty years past termination of exposure for mesothelioma, and I think it's conceivable that the risk could turn around and go down. They certainly do for lung cancer fifteen years or more after exposure is stopped. They do turn around and go back the other way.

K. Crump Tr., Vol. XXVI at 71-72.

(Footnote 36 continued on next page.)

Mr. Peto calculates the risk of both mesotheliomas and lung cancer to children exposed in schools for six years. His lung cancer estimates assume the children to be cigarette smokers. The results are presented in terms of the risk of exposure to concentrations of 1 fiber/cc; adjustment of those results to determine the risk at 0.0002 fiber/cc and 0.005 fiber/cc, the concentrations assessed by the Simpson Report, yield predicted risks for both diseases together of 1 in 1 million and 3 in 100,000, respectively. Peto points out that contrary to what might have been expected, more than 70% of the predicted mesothelioma deaths and more than 80% of the predicted lung cancer deaths would occur after age 60.[37/]

(Footnote 36 continued from previous page.)

Dr. Crump also noted that models other than the one Mr. Peto used (assuming the rate increases at a cubic power) would fit the data equally well:

> I don't think there is any basis for determining which of these techniques is more valid than any other at this point. I think they all have about the same basis as far as curve fitting and biological rationale.

K. Crump Tr., Vol. XXVI at 75. In particular, Dr. Crump explained to the Commission that a model where incidence increases only at a squared power was equally plausible. Id. at 73-75.

More a mesothelioma prediction model based on less than a cubic power increase and a peaking of incidence rates after forty years, the estimates of Mr. Peto would be substantially reduced.

37/ R.C.A. Exhibit 37, Tab 10, Peto (1981), supra note 18, at 13, 15.

In sum, the Commission's experts were in agreement with the consensus opinion of past governmental advisory committees that have all concluded no general environmental risk exists from continued asbestos use. In the one setting -- schools -- that has drawn the most attention, the experts agree no significant risk exists. Even when conservative risk extrapolation principles are applied, the predicted risk to school residents is de minimis. It is clear that the task for government regulators in assessing future asbestos use should be focused on the occupational setting, not the general environment.

CHAPTER VI

VI. MONITORING WORKPLACE AND ENVIRONMENTAL ASBESTOS CONCENTRATIONS

Although most of the Commission's Phase II witnesses discussed medical issues, the Commission also heard from several of the world's foremost experts on monitoring asbestos. The monitoring evidence has important implications for the development of practical societal control measures for asbestos. In particular, the monitoring testimony established that:

- -- The membrane filter method is the best available technique for routine occupational monitoring; no feasible alternative method currently exists.
- -- The relatively high variability of the membrane filter method and day-to-day variability in actual airborne concentrations impose a practical lower limit for setting occupational standards, in the range of 0.5 to 1 fiber/cc. Thus, although very low workplace standards for asbestos are infeasible, standards in the range of one to two fibers/cc could be implemented if employers allow for variability by maintaining workplace concentrations well below mandatory standards.
- -- Occupational standards which take measurement and inter-day variability into account will achieve very protective workplace conditions, because employers must routinely achieve average exposure levels far below mandatory standards.
- -- Quantitative standards for asbestos outside the workplace are unnecessary and could not be implemented without development of a standardized technique for environmental monitoring.

A. Workplace Monitoring.

The membrane filter method is a simple and relatively inexpensive tool for measuring workplace asbestos fiber

concentrations, and is used for occupational monitoring throughout North America and Europe. Presently, there is no feasible alternative for routine fiber counting in the workplace. Despite some limitations, including the need to account for the substantial variability of membrane filter measurements, this method can be used to achieve highly protective workplace conditions.

1. Use of the Membrane Filter Method for Routine Occupational Monitoring.

Developed during the 1960's in the United Kingdom, the membrane filter method is now used throughout North America and Europe for monitoring workplace asbestos concentrations.[1] Most occupational exposure standards for asbestos are premised on membrane filter measurements,[2] and available epidemiological investigations are based on exposure estimates which can be related to the membrane filter method.[3]

Because it is generally agreed that fiber counts, and not measurements of mass or particles, are critical to

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1. R.C.A. Exhibit 39, Tab 11, G. Chase & M. Rhodes, "Measurement of Asbestos Levels in the Workplace" at 1-2, 6 (August 14, 1981, hereinafter cited as "Chase & Rhodes Statement"); see M. Rhodes Tr., Vol XXVII at 9 (the "membrane filter method in one form or another is used largely throughout the world, in the measurement of occupational asbestos exposure.").

2/ Chase & Rhodes Statement at 8; M. Rhodes Tr., Vol. XXVII at 9.

3/ M. Rhodes Tr., Vol. XXVII at 10; see Chase & Rhodes Statement at 12.

assessing the risk of asbestos exposure,[6] the membrane filter method represents a significant advance over earlier asbestos monitoring techniques. As Dr. William Nicholson told the Commission, fiber counting is a "significant step forward," and "certainly is a better method for assessment than the old method of particle counting which assessed an environment in terms of the total dust concentration present."[7]

The membrane filter method also has significant practical advantages. It is relatively simple,[8] and the necessary equipment is widely available. Operators with the necessary technical skills have been available to meet monitoring needs and can be trained in a reasonable time.[9] As a result, capital and operating costs are reasonable. The most expensive part of the equipment, the phase contrast microscope, costs about $5,000.[10] The cost of a laboratory fiber count for one filter, a process which takes about ten

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6/ B.C.A. Exhibit 39, Feb 19, Chatfield, E. J., "Airborne Asbestos Fibres: A Summary of Some Measurement Problems," 1 (July 9, 1981) (hereinafter cited as "Chatfield Statement"); E. Chatfield Tr., Vol. XVIII at [illegible].

7/ W. Nicholson Tr., Vol. XV at 33.

8/ M. Rhodes Tr., Vol. XXVII at 10; E. Chatfield Tr., Vol. XVIII at 118; W. Nicholson Tr., Vol XV at 33.

9/ Chase & Rhodes Statement at 7-8.

10/ Id. at 7.

to thirty minutes,[9] typically ranges from $20 to $40.[10] If outside personnel must be hired for sample collection, the cost may be $500 per day, although sample collection often is performed in-house.[11]

Through widespread use, a large number of laboratories have developed expertise in the membrane filter method. Although further work is needed, the method has been improved substantially, and there has been significant progress toward standardization.[12] Thus, the membrane filter method has the advantages of an established monitoring system available for current use on a broad scale.

The membrane filter method has some important limitations, which include the substantial variability of monitoring results, the method's inability to distinguish fiber types, and the limited resolution of the phase-contrast microscope. Although these limitations must be considered in establishing standards or industrial hygiene programs, none preclude the effective use of membrane filter monitoring in the asbestos industry.

Despite progress in standardization, membrane filter measurements are subject to substantial variability which

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9/ M. Rhodes Tr., Vol. XXVII at 12, 23-24.

10/ Id. at 24 ("around twenty or thirty dollars"); Chase & Rhodes Statement at 8 ("in the range of $30 to $40").

11/ M. Rhodes Tr., Vol. XXVII at 24.

12/ Chase & Rhodes Statement at 8.

increases at low fiber concentrations. This variability precludes compliance determinations at very low levels and requires employers to maintain average workplace asbestos levels well below mandatory standards. The variability of the membrane filter method, and its significance in establishing occupational standards, are addressed in detail below.

The membrane filter method does not distinguish among asbestos fiber types, nor permit ready discrimination between asbestos fibers and other types of fibers.[11] All particles which meet the specified criteria for length and aspect ratio may be counted as "asbestos fibers," although a variety of methods may be used to provide judgmental adjustments to fiber counts of mixed dusts.[12]

The inability to distinguish fiber types does not, however, seriously affect the utility of membrane filter monitoring in the asbestos industry.[13] As Dr. Nicholson testified, there often is no need for fiber type discrimination:[14]

11/ Id. at 8; M. Trudeau Tr., Vol. XXII at 57-58.

12/ Chase & Rhodes Statement at 8.

13/ By contrast, membrane filter monitoring may be inappropriate in "peripheral industries" such as the vermiculite, talc, or gypsum industries, which generate large numbers of non-asbestos fibers. Because the membrane filter method does not distinguish among fiber types, samples in these industries may show positive fiber counts even when no asbestos is present. E. Chatfield Tr., Vol. XVIII at 33-35.

14/ W. Nicholson Tr., Vol. XV at 33; see M. Trudeau Tr., Vol. XXII at 33.

> If you were going into an asbestos factory, the chances are that virtually all the fibers that you would be counting would indeed be asbestos, and thus unless there [are] very complicated work situations there is not a need to discriminate between fibers of one type or another.

Moreover, where there is mixed exposure to different types of asbestos or to other fibrous materials, electron microscope techniques may be used selectively and occasionally to characterize the identity and proportions of dust cloud constituents.[17] Appropriate corrections then can be applied to membrane filter measurements,[18] or used to weight measurements from an occupational environment containing two or more types of asbestos for which there are different numerical standards.[19] Mr. Trudeau testified that such a weighting approach is used in monitoring workplace dust clouds for silica dust and could be used to weight membrane filter measurements from dust clouds with more than one asbestos fiber type.[20] Even where the mix of fiber types might vary for different processes within a

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17/ M. Rhodes Tr., Vol. XXVII at 110-11, 115-16; Chase & Rhodes Statement at 10. See also G. Rajhans Tr., Vol. XXIIA at 44-45 (suggesting that both analyses of bulk samples, and electron microscope analyses of air samples, could be used to determine the distribution of fiber types in mixed fiber environments).

18/ M. Rhodes Tr., Vol. XXVII at 110-11

19/ G. Chase Tr., Vol. XXVII at 129-32

20/ M. Trudeau Tr., Vol. XXII at 60.

plant, the weighting could be performed on a process-by-process basis.[11/] Thus, it would be feasible to implement different numerical standards for different fiber types without requiring the application of the most stringent standard to the entire plant.

It is generally recognized that the membrane filter method provides an index of fiber concentrations rather than a complete count of all fibers.[12/] The method specifies that only fibers longer than 5 microns, with an aspect ratio of 3 to 1 or greater, should be counted.[13/] In addition, very thin fibers cannot be counted due to the limit of resolution for phase-contrast microscopy, which is about 0.2 microns under optimum conditions, and about 0.4 to 0.5 microns under routine use.[14/]

The 5 micron length limitation was chosen to facilitate counting because of the convenience and the ease of visibility of fibers longer than that length. As Dr. Nicholson explained:[15/]

11/ Id. at 118-19.

12/ Chase & Rhodes Statement at 9; Chatfield Statement at 4; M. Trudeau Tr., Vol. XXII at 7.

13/ See, e.g., R.C.A. Exhibit 39, Tab 5, Asbestos International Association Dust Measurement Advisory Panel, "Reference Method for the Determination of Airborne Asbestos Fibre Concentrations at Workplaces by Light Microscopy (Membrane Filter Method)" § 5.3.5 (1979).

14/ R. Chatfield Tr., Vol. XVIII at 11; Chatfield Statement at 4; Chase & Rhodes Statement at 8-9.

15/ W. Nicholson Tr., Vol. XIV at 47.

> It was a very good choice because most fibers five microns and longer would be at least a half a micron in diameter, and thus visible, and it was a convenient length for observation.

Without more definitive data on the biological activity of short and thin fibers, it would be unwise to require changes in the established monitoring method. As Dr. Chatfield testified, "[t]he medical community has so far been unable to define completely the range of fibre sizes which should be included in measurements of airborne (or waterborne) asbestos fibre concentrations."[16/]

Nevertheless, there are some indications that the membrane filter method provides an index of exposure reasonably correlated with the health evidence. For example, Dr. Gibbs testified that:[17/]

> As it so happens, it looks like the membrane filter counts do relate to health effects in the Quebec mining industry, as measured by mortality. So, yes, one might not have chosen a bad index in the sense that it looks like its beginning to relate to health effects.

Similarly, Dr. Dement observed that:[18/]

> The problem with the phase contrast method really is it counts only a portion of airborne dust. In defense of it though, I think if we believe the bioassay data and we are in fact counting a good

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16/ Chatfield Statement at 3.

17/ G. Gibbs Tr., Vol. XXXIII at 16-17.

18/ J. Dement Tr., Vol. XXXII at 33.

> portion of those fibers that are longer than five microns, then perhaps it is a reasonable index of exposure.

In addition, as discussed in Chapter II, there is strong evidence that fibers shorter than 5 microns pose little or no risk.[9/]

Because any change in the method which results in higher or lower fiber counts would affect a de facto revision of occupational standards, there is a significant practical impediment to altering the measurement technique. As Dr. Chatfield observed:[10/]

> If the fibre count value obtained by an improved procedure is higher than that obtained by the earlier method, this is seen by industry as a "back door" reduction in the legislated maximum airborne concentrations. If the value is lower, labour perceives it as a relaxation of existing standards.

Moreover, because membrane filter measurements are an index "unrelated to any standard samples," Dr. Chatfield concluded that "new methods cannot easily or convincingly be related to the techniques in use today."[11/] Thus, changes in the method or equipment to count more short or thin fibers would implement a change of unknown magnitude in existing standards.

9/ See Chapter II supra, pp. II-3 to II-23.

10/ Chatfield Statement at 3.

11/ Id. at 3-4.

Despite the recognized limitations of membrane filter monitoring, it is generally agreed to be the best available method to conduct the large volume of routine monitoring required in the asbestos industry.[12/] Dr. Nicholson testified at length on the advantages and utility of membrane filter monitoring:[13/]

> [A]t this point [the membrane filter method] appears to be the way one will go in assessing the workplace, because it's simple ... it bears upon previous data that we have. What we are looking for is methods to achieve a reduction in workplace air concentrations, and is variable as it is and with all the problems that it has, I'm unaware of a better method for rapid and economical assessment of the workplace.

Mr. Trudeau acknowledged the limits of the membrane filter technique, but testified that "all methods show an index of the concentration, and if a choice has to be made between all these indexes, the best choice is the membrane filter method."[14/] Dr. Chatfield similarly described the membrane filter method as "a very simple test," and observed:[15/]

12/ The number of workplace samples required in the industry is "virtually staggering." M. Rhodes Tr., Vol XXVII at 10. At one mine and mill, more than 1,500 samples are analyzed every year. Thus, "asbestos monitoring imposes an appreciable burden, even though a fairly large number of laboratories ... are available to do the work." Chase & Rhodes Statement at 5.

13/ N. Nicholson Tr., Vol. XV at 33.

14/ M. Trudeau Tr., Vol. XXII at 126.

15/ E. Chatfield Tr., Vol. XVIII at 113, 113.

> I personally would rather see a lot of measurements by a simple technique, because of this fundamental variability problem, than I would see a single measurement made at one work station being used to define everything, of a much more sophisticated nature.

Dr. Chatfield concluded that "as a dust control measure, I think it is a perfectly reasonable way of approaching the problem."[16] although its use "should be restricted to the well-characterized situations which we find in the asbestos industry."[17]

Drs. Chase and Rhodes, who have studied the membrane filter method extensively, also concluded that "the membrane filter method, despite some limitations, is the only suitable method now available for routine workplace monitoring of asbestos on the large scale that is required to support a numerical occupational asbestos standard."[18] Dr. Rhodes succinctly summarized their thinking:[19]

> Now I don't think there are any illusions that the membrane filter method is the greatest ... but it does have some very strong reasons for use, and basically, it's very simple, relative to some of the alternative methods.

16/ Id. at 119.

17/ Id. at 36.

18/ Chase & Rhodes Statement at 12.

19/ M. Rhodes Tr., Vol. XXVII at 9-10.

> It can be set up and operated at a reasonable cost, and it's suitable for routine measurements, where you have large numbers of samples ... .
>
> And the epidemiology studies that have come out in the last five to ten years, are also generally keyed to exposures as measured by the membrane filter method. It is pretty well in place, widely used, and related [to] epidemiology.
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> * * *
>
> So even with its limitations, I don't know of any better way to monitor workplace exposure, than the membrane filter method. I think it's important that we recognize its limitations, but I would be very concerned if we suddenly switched to some alternative method.

In sum, the clear consensus view of the experts who appeared before the Commission is that the membrane filter method should continue to be used for routine occupational monitoring and should remain the basis for asbestos workplace standards.

2. Absence of a Feasible Alternative for Routine Workplace Monitoring.

Potential alternatives to the membrane filter method are not currently feasible for routine monitoring. Although useful for characterizing mixed fiber dust clouds, electron microscope techniques have not been standardized, are not available for large-scale use, involve high capital and operating costs, and cannot be related to existing epidemiology and health standards. Other potential monitoring techniques are largely experimental.

a. Scanning Electron Microscopy.

Scanning electron microscope (SEM) or transmission electron microscope (TEM) techniques, in conjunction with other methods such as energy dispersive x-ray analysis, may be used to distinguish asbestos fiber types and to discriminate between asbestos fibers and other fibrous materials.[10/] As described above, these methods may be used selectively to characterize mixed fiber environments as a supplement to routine membrane filter monitoring.[11/]

Although SEM has been suggested as a potential alternative to membrane filter monitoring, this technique is almost universally regarded as unsuitable for routine workplace monitoring.

There are at present no standardized procedures for SEM sample collection, sample preparation, or analysis. Its use would therefore compound variability as compared with the present optical technique.[12/]

Filter selection and preparation present a particular problem in SEM analysis. The membrane filter cannot be used for SEM without a method for destroying the filter's sponge structure. While this can be done, "[t]he problem is not to shrink it, not to change its dimensions, or not to have the

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10/ Chase & Rhodes Statement at 10.

11/ See pp. VI-6 to VI-7, supra.

12/ Chase & Rhodes Statement at 11.

fibres disappear down into the goo."[13/] The alternative is the nucleopore filter, which provides no assurance of uniform collection,[14/] and must be hand-carried to the laboratory:[15/]

> There is a problem with using nucle[o]pore filters for ambient air samples. They do have to be handcarried. Otherwise material is going to be moved on the surface, or lost. That has been one of the major criticisms I have made of the U.S. EPA method for ambient atmospheres ... .

Nor is there a standardized procedure for sample preparation; as Dr. Rhodes noted, "[e]very laboratory has their own favourite technique."[16/]

SEM also poses very high capital and operating costs. In contrast to the relatively inexpensive phase-contrast microscope (about $5,000), a scanning electron microscope costs from $70,000 to $80,000. SEM also presents a much bigger maintenance problem, with annual costs as high as $3,000 to $4,000.[17/]

Operating costs for SEM analysis, which vary with the sophistication of the technique, are also significant. SEM

13/ H. Rhodes Tr., Vol. XXVII at 110, 112.

14/ Id. at 110.

15/ E. Chatfield Tr., Vol. XVIII at 126; see H. Rhodes Tr., Vol. XXVII at 110.

16/ H. Rhodes Tr., Vol. XXVII at 113.

17/ E. Chatfield Tr., Vol. XVIII at 121; H. Rhodes Tr., Vol. XXVII at 110, 113; Chase & Rhodes Statement at 10.

counting is "a lot slower" than optical counting, adding to the cost.[10/] Although Dr. Chatfield stated that a simple EM analysis, with no fiber identification, "would not be significantly higher" than with a phase-contrast microscope, he testified that an analysis of an environmental sample using identification procedures, involving three to four hours of microscope time and one or two hours of preparation, would cost approximately $450.[11/] An analysis involving chemical identification and "some indications of ... crystallography" would cost close to $2,000, and precise crystallography may require "two hours or so on a single fiber," followed by "a number of computer analyses."[12/] Thus, SEM techniques which allow fiber identification are far more time-consuming and expensive than a typical membrane filter sample, which can be analyzed in a half-hour or less for $20 to $40.

The SEM requires a relatively skilled operator, with a higher degree of technical skill and training than that required for phase-contrast microscopy. Because of the need for highly skilled operators, and the high cost of SEM equipment, the number of laboratories with the capability for SEM fiber counting is "quite limited."[13/] Thus, unlike

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10/ E. Rhodes Tr., Vol. XXVII at 113.

11/ E. Chatfield Tr., Vol. XVIII at 97-98.

12/ Id. at 111.

13/ Chase & Rhodes Statement at 10; E. Rhodes Tr., Vol. XXVII at 110.

the established membrane filter method, there is no current capability to conduct SEM monitoring on a broad scale.

Ironically, the higher magnification available with SEM, which permits the identification of some short or thin fibers not detectable with phase-contrast microscopy, cannot be used without creating additional problems. As Dr. Rhodes explained, at a magnification equivalent to that of the phase-contrast microscope (about 500 x), the SEM gives better resolution than the optical method, but "you see pretty much the same thing."[13/] However, "[i]f you start going to higher [magnification], two thousand, five thousand, you begin to get into the problem of your field area get[ting] smaller and smaller and smaller, and you're counting a much smaller number of fibres."[13/] At higher magnifications "you have a tendency to miss the big fibres."[14/]

Moreover, although SEM fiber counts at higher magnifications can include substantial numbers of very short and very thin fibers not seen by optical microscopy, there is no known relationship between SEM counts and optical measurements made with the membrane filter method. Even if other roadblocks were overcome, adoption of SEM monitoring would render the current numerical standards "utterly unsuitable."

13/ E. Rhodes Tr., Vol. XXVII at 114; see Id. at 113, 114-15.

13/ Id. at 110; see Id. at 111; Chase & Rhodes Statement at 11.

14/ E. Rhodes Tr., Vol. XXVII at 113.

yet there is "no way to relate an SEM-based standard to the available epidemiological data which are based on optical methods or approximations to optical counts."[15/] Thus, there is no basis for establishing a standard predicated on SEM monitoring.

For all these reasons, Drs. Chase and Rhodes concluded:[16/]

> SEM is presently infeasible for use as a routine monitoring method, and the development of a feasible SEM technique for this purpose would require extended research and great expense.

Similarly, the Ministry of Labour is examining electron microscope measurement, but has concluded that at present, electron microscopy is not a feasible method for routine workplace monitoring.[17/] Undoubtedly, SEM will find selective use for characterizing mixed fiber environments, but it should not be regarded as a suitable method for routine occupational monitoring.

b. Transmission Electron Microscopy.

TEM, like SEM, may be useful for selective characterization of dust clouds, but there has been no serious suggestions to use TEM for routine workplace monitoring.

15/ Chase & Rhodes Statement at 11-12.

16/ Id. at 12.

17/ G. Rajhans Tr., Vol. XLIIA at 63.

Mr. Trudeau testified in detail regarding the disadvantages of TEM for routine analysis:[18/]

> [I]f you ask a transmission electron microscopist to set up an analysis for one single sample, he is most probably going to charge you close to six hundred dollars for every sample he is going to process because the sampling is tedious. The preparation of the sample is very long, and the analysis is even longer and more tedious.
>
> Also, he has to pay for the capitalization of his instrument, which costs a lot of money.

According to Mr. Trudeau, the preparation and analysis of a TEM sample may require several days to complete,[19/] making TEM measurement far more time consuming than the relatively simple membrane filter method.

In addition, because fibers may be broken up or lost during TEM sample preparation, TEM measurements may not accurately reflect the size and number of fibers in the sampled air. As Mr. Trudeau observed:[20/]

18/ M. Trudeau Tr., Vol. XXII at 33.

19/ Mr. Trudeau described one TEM method as requiring "three or four days" for sample preparation, "if you work fast." Id. at 35. After preparation, a direct analysis on the TEM would take five to seven hours, but this approach is unsuitable due to eye fatigue from looking at the fluorescent screen. As Mr. Trudeau noted:

> Even if the fibrils can be seen, your eyes get tired, they get red, you don't see anything, you see particles where there aren't any, you don't see any where there are many, things like that.

Id. at 35. Accordingly, the analyst uses "up to sixty or a hundred" micrographs, the examination of which "may take another day or two." Id. at 36.

20/ Id. at 42.

> With the electron microscopy, sure we see all the fibers, but the preparation is so aggressive that we may ... we may not, but we may ... break the fibers into fibrils, so what we measure on the electron ... when we use electron microscopy, because of the preparation, is again an index.

Similarly, Dr. Rhodes testified that with TEM, "you get into the condensation, washing, and all that sort of thing, that there is some question whether you're gonna lose some, or I don't know how much it would change it, but the losses are a question."[41]

Thus, TEM is far too time-consuming and expensive to serve as a routine occupational monitoring tool, and TEM sample preparation may alter asbestos fibers present in the original sample.

c. Automatic Counting and Magnetic Alignment Techniques.

Other potential alternatives to membrane filter monitoring, such as automatic counting devices and magnetic alignment techniques, are largely experimental. None of these methods are presently available for routine occupational monitoring.

One automatic counting method, developed in England and known as "Magiscan," uses an image analysis device for fiber

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41/ R. Rhodes Tr., Vol. XXVII at 112-113.

counting with phase-contrast optical microscopy. As Dr. Chatfield testified, "[i]t's just an automatic method of looking at the same image."[42]

The Health and Safety Executive in London is using a Magiscan unit, although it is impractical for British workplace samples all to be "fed into this central machine."[43] Dr. Chatfield testified:[44]

> The basic problem with that unit is its expense. We are talking here of <u>something like a quarter of a million pounds</u> having been invested in [installing the Health and Safety Executive's unit]. That would mean, basically, that a sophisticated instrument is going to be located in one center, and then samples sent in to it, which is perhaps not as satisfactory as we would like it.

Thus, the capital cost of the Magiscan far exceeds capital costs for sophisticated electron microscope equipment.

In addition, automatic counting techniques require some technical break-throughs before they could be used for monitoring purposes.[45] There have been calibration difficulties with Magiscan.[46] Moreover, because the Magiscan uses a

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42/ R. Chatfield Tr., Vol. XVIII at 71, 85.

43/ Id. at 87.

44/ Id. at 79 (emphasis supplied); see M. Trudeau Tr., Vol. XXII at 51 ("Magiscan is a very costly instrument ... .").

45/ O. Chase Tr., Vol. XXVII at 50; see M. Trudeau Tr., Vol. XXII at 52, 53.

46/ R. Chatfield Tr., Vol. XVIII at 71.

single focal position, and "doesn't do any racking up and down," the question whether "the machine actually [counts] the same features that a human being does" has not been satisfactorily resolved.[67]

Another potential automatic counting technique, which would use an image analysis device with scanning electron microscopy, is still in the experimental stage.[68] Like the Magiscan, this method poses high capital costs. As Dr. Chatfield noted, "we are talking maybe a hundred, a hundred and twenty thousand dollars for that approach, even assuming it's fully developed."[69] Because it is based on SEM analysis, this method is also subject to all of the deficiencies of SEM monitoring described above.

Finally, Dr. Chatfield described on-going investigations of measurement techniques based on magnetic alignment of fibers.[70] Unlike other potential alternatives to membrane filter-monitoring, the magnetic alignment technique is not a fiber-counting technique. This method has the potential to provide information on mean fiber length and mean fiber diameter, but not an actual fiber count. Dr. Chatfield testified that "we are still working on this technique to

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67/ Id. at 86.

68/ Id. at 72, 87.

69/ Id. at 79. The cost would include about $80,000 for the SEM, and about $-0,000 for the computer. Id.

70/ Id. at 73-83.

try to find out how to take these signals apart to get the data we need," and thus cautioned that "this is one of these things which is still under development so we have to go carefully here ... ."[71]

In short, automatic counting methods and magnetic alignment techniques are in various stages of development. None of these techniques are sufficiently advanced to merit current consideration for adoption in occupational monitoring programs.

3. Substantial Variability of the Membrane Filter Method.

It has been widely recognized for many years that membrane filter measurements are subject to substantial variability. Such measurement variability, as well as day-to-day variability in asbestos concentrations, have important implications for the establishment of occupational standards.

a. [illegible]ment Variability.

The variability of membrane filter measurements has long been recognized based on experience in the U.S. NIOSH

71/ Id. at 82, 80.

Proficiency Analytical Testing program[73] and a number of limited slide exchange studies.[73] For example, Dr. Chatfield described one test showing that the high and low fiber counts by different operators on the same filter may vary by a factor of six.[74]

Although the slide exchange studies showed a high degree of variability, as Drs. Chase and Rhodes testified, such studies had been confined to assessing variability in the sample evaluation step, and often included only limited numbers of laboratories and small numbers of filters. Accordingly, the Air Monitoring Committee of the Asbestos Information Association/North America conducted a large scale round-robin study, designed to provide the most complete assessment of the method's variability which could practically be obtained.

This round-robin measured all sources of variability in the sample evaluation step, including random error and systematic or persistent error. The round-robin also

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[73] Chase & Rhodes Statement at 12-13 (citing R.C.A. Exhibit 39, Tab 6, Memorandum from Jeremiah D. Lynch, Director, NIOSH Division of Training, "A Systematic Approach to the Standardization of Asbestos Counting" (October 4, 1972)).

[73] Chase & Rhodes Statement at 13; see R. Rhodes Tr., Vol. XXVII at 13.

[74] Chatfield Statement at 3; R. Chatfield Tr., Vol. XVIII at 18.

provided the first quantitative assessment of random error in the sample collection step.[75]

> Thus, the overall variability measured in the round-robin includes all sources of error in the membrane filter method except systematic or persistent error in sample collection, which could not practically be measured.[76]

Forty-six laboratories participated in the round-robin study, from a total of about 200 laboratories in the United States actively engaged in occupational monitoring with the membrane filter technique. Twenty-seven of the participating laboratories also were active participants in NIOSH's voluntary quality control Proficiency Analytical Testing (PAT) program. The study results therefore are reasonably representative of the best monitoring practices currently achieved on a broad scale.[77]

Not surprisingly, since it provided a more complete measurement of variability than previous studies, the round-robin demonstrated "that the total variability for the membrane filter method is higher than previously recognized."[78] In addition, the round-robin confirmed earlier

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[75] Chase & Rhodes Statement at 13; R. Rhodes Tr., Vol. XXVII at 15-16. For a description of the sample collection and sample evaluation steps in membrane filter monitoring, see Chase & Rhodes Statement at 7.

[76] Chase & Rhodes Statement at 14.

[77] Id. at 14; R. Rhodes Tr., Vol. XXVII at 12-13.

[78] Chase & Rhodes Statement at 15-16; see id. at 13; G. Chase Tr., Vol. XXVII at 35.

observations that the relative variability increases at lower filter loadings.[79/]

Estimated coefficients of variation (CV) and 95% confidence limits for eight-hour time-weighted average (TWA) measurements of 0.1, 0.5, and 1.0 fiber/cc, based on the round-robin data and a series of idealized filter loadings (Table 1 on next page), demonstrate the substantial variability of membrane filter monitoring. For example, the 95% confidence limits from Table 1 for a measured TWA value of 0.5 fiber/cc, based on one eight-hour sample, show that the "true" workplace concentration could be as low as 0.13 fiber/cc or as high as 1.37 fibers/cc. Indeed, even using the smallest CV shown in Table 1 for a measured value of 0.5 fiber/cc (based on eight one-hour samples), the 95% confidence limits range from about one-half to more than twice the measured value.[80/] The round-robin results indicate that the variability of the membrane filter method is quite large relative to other analytical procedures for industrial hygiene and regulatory compliance measurement.

Current efforts to improve the membrane filter technique include the reference method developed by the Asbestos

[redacted]

79/ Chase & Rhodes Statement at 16.

80/ Chase & Rhodes Statement at 16 and Table 1; see generally G. Chase Tr., Vol. XXVII at 41-44.

ESTIMATED COEFFICIENTS OF VARIATION AND CONFIDENCE LIMITS[81/]

Samples Collected (8-Hour Shift)	Coefficient of Variation of TWA	95% Confidence Limits
TWA OF 0.1 FIBERS/CC		
1	0.68	0.024–0.28
2	0.56	0.031–0.25
8	0.44	0.046–0.21
TWA OF 0.5 FIBERS/CC		
1	0.66	0.13–1.37
2	0.49	0.18–1.14
8	0.35	0.24–0.93
TWA OF 1.0 FIBERS/CC		
1	0.64	0.26–2.74
2	0.50	0.36–2.27
8	0.33	0.50–1.80

81/ From Table 8.1, Rhodes, R. et al., "A Study of the Empirical Precision of Airborne Asbestos Concentration Measurements in the Workplace by the Membrane Filter Method," (Draft, July 1, 1981); see Chase & Rhodes Statement, Table 1.

[redacted]

International Association's Dust Measurement Advisory Panel.[82] The AIA reference method has been well-received internationally as a standardized approach to monitoring,[83] and was described by Drs. Trudeau and Chatfield as the best technique for membrane filter monitoring.[84] Continuing international discussions are addressing the manner in which certain fiber configurations should be counted.[85]

Nevertheless, it would be unrealistic to expect a dramatic improvement in membrane filter variability. For example, although it has been suggested that variability could be decreased by counting more fibers, Dr. Chase testified that "[t]he available data suggest that it would not have a dramatic impact at all on the total uncertainty ... ."[86] Similarly, it has been suggested that pump flow rates be increased to produce higher filter loadings at low airborne concentrations, but flow rates are limited by problems in designing a pump small enough for personal sampling which

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82/ R.C.A. Exhibit 39, Tab 5, Asbestos International Association Dust Measurement Advisory Panel, "Reference Method for the Determination of Airborne Asbestos Fibre Concentrations at Workplaces by Light Microscopy (Membrane Filter Method)" (1979).

83/ H. Rhodes Tr., Vol. XXVII at 8.

84/ H. Trudeau Tr., Vol. XXII at 30-31; E. Chatfield Tr., Vol. XVIII at 30-31.

85/ H. Rhodes Tr., Vol. XXVII at 8.

86/ G. Chase Tr., Vol. XXVII at 49.

will run for eight hours at a constant rate, as well as by the relationship between flow rate and face velocity.[87]

Theoretically, variability could be reduced by replacing the one day (eight hour) TWA standard with a standard based on the average of several daily samples, such as a weekly average, but there are practical limitations to such extensive monitoring.[88] In the same way, the round-robin data suggest that multiple samples during an eight-hour shift should produce lower variability than a single full-shift sample. However, this hypothesis was not specifically tested in the round-robin, and multiple sampling may be infeasible at low fiber concentrations, which would yield very low filter loadings indistinguishable from background levels for short sampling times.[89]

Thus, membrane filter measurements are subject to a relatively high degree of variability. Although progress can be expected, particularly with respect to the uniform treatment of fiber configurations, there is no obvious practical way to achieve a substantial reduction in variability.

87/ H. Rhodes & G. Chase Tr., Vol. XXVII at 105-06.

88/ Id. at 106-105.

89/ Chase & Rhodes Statement at 17-18; G. Chase Tr., Vol. XXVII at 108-109.

b. Implications for Establishing Occupational Standards.

Occupational standards for asbestos must take both the variability of the membrane filter method and day-to-day variations in actual asbestos concentrations into account. Due to these two sources of variability, extremely low occupational standards for asbestos would be infeasible and unenforceable.

Workplace standards cannot be implemented effectively unless employers and enforcement officers can reliably measure compliance. Due to measurement variability, confidence limits -- generally at the 95% level of confidence -- are used to compare measured values with standards.[10/] As Dr. Chatfield explained, the application of such confidence limits differs for the employer who must assure that his workplace is in compliance and the officer who is charged with enforcing the standard:[11/]

> If I am in the situation of the government where, before I take anyone to court I want to be sure, absolutely sure, as sure as I can be that they are out of compliance, then I would try to make sure that my lower confidence limit exceeded two.
>
> And if I'm on the other side, I try to make sure that the upper confidence limit is below two.

10/ E. Chatfield Tr., Vol. XVIII at 103-104; G. Chase Tr., Vol. XXVII at 28-32.

11/ E. Chatfield Tr., Vol. XVIII at 104 (emphasis supplied). See Chase & Rhodes Statement at 19.

Thus, to account for measurement variability, the employer must obtain monitoring results sufficiently below the standard to provide an upper confidence limit at or below the standard.

The variability in actual workplace concentrations, as well as measurement variability, must be considered in evaluating measured values for workplace contaminants. There is general agreement that true airborne workplace concentrations vary from day-to-day and have an approximate lognormal distribution.[12/] Although the average concentration can often be reduced, inter-day variability in actual concentrations cannot be controlled.[13/] Thus, as Dr. Chase testified:[14/]

> [W]e've got uncertainty not only in the measurement we take on a given day, but we've got the uncertainty of -- was that day a typical day, or was that day a non-typical day? And I've put typical in quotes, because what we've got is a spectrum of actual [daily] exposures for that worker.

Work by the U.S. NIOSH shows that, due to measurement variability and inter-day variability in actual concentrations, monitoring measurements well below the standard for a workplace containment are required to determine with reasonable confidence that very few employee exposures will exceed

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12/ Chase & Rhodes Statement at 19; G. Chase Tr., Vol. XXVII at 54-56; E. Chatfield Tr., Vol. XVIII at 104-05.

13/ Chase & Rhodes Statement at 19; G. Chase, Tr. Vol. XXVII at 58-59.

14/ G. Chase Tr., Vol. XXVII at 56.

the standard. The NIOSH calculations -- based on a relatively low degree of inter-day variability and a measurement technique with an assumed CV of only 0.10 -- show that "a measurement of one-half the workplace standard is necessary to provide 95% confidence that no more than 5% of an employee's workdays will exceed the standard."[95/] NIOSH recognized that higher inter-day variability in actual concentrations, or higher measurement variability, would require even lower measurements to achieve the same degree of confidence regarding exposure levels.[96/]

In the asbestos industry, the relatively high variability of the membrane filter method requires that employers obtain monitoring results far below the workplace standard in order to obtain reasonable assurance of compliance.[97/] As Drs. Rhodes and Chase testified:[98/]

> If the standard is set at an extremely low level, without a sufficient lower range of values which can be measured reliably, employers will be unable to ascertain compliance status with a reasonable degree of confidence.

Drs. Chase and Rhodes have estimated the range for the lowest occupational asbestos standard that would theoretically just permit an employer to meet the NIOSH recommendation

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95/ Chase & Rhodes Statement at 30 (emphasis in original).

96/ Id. at 30.

97/ Chase & Rhodes Statement at 31; G. Chase Tr., Vol. XXVII at 59.

98/ Chase & Rhodes Statement at 31.

of 95% confidence that no more than 5% of employee exposures will exceed the standard.[99/] This range of values, as shown in Table 2 on the next page, provides the "absolute lower bound, that would even begin to permit" the specified degree of confidence.[100/]

The lower bound values presented in Table 2 are based on a range of CV's for the membrane filter method from the round-robin data,[101/] and a range of geometric standard deviations for inter-day variability in actual concentrations based on NIOSH data for particulates.[102/] These lower bounds represent the minimum standards which would permit the specified degree of confidence from monitoring results, without requiring measurement below 0.1 fiber/cc;[103/] the

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99/ Id. at 31 and Table 1.

100/ G. Chase Tr., Vol. XXVII at 62.

101/ Chase & Rhodes Statement at 32; G. Chase Tr., Vol. XXVII at 61-62. The CV's range from 0.4, corresponding to the lowest estimated CV at a measured value of 0.1 fiber/cc, to 0.7, corresponding to the highest estimated CV at a measured value of 0.1 fiber/cc. Chase & Rhodes Statement at 32.

102/ Chase & Rhodes Statement at 32; G. Chase Tr., Vol. XXVII at 61-62. The geometric standard deviations used in the calculations range from 1.3 to 1.8, bracketing the median of about 1.65 found by NIOSH for particulate samples such as asbestos. Id.; see E.C.A. Exhibit 39, Tab 8, Leidel, N. A., et al., "Exposure Measurement Action Level and Occupational Environmental Variability" at 11 (1975).

103/ Chase & Rhodes Statement at 32; G. Chase Tr., Vol. XXVII at 60, 61, 64.

TABLE 2

LOWER BOUNDS FOR WORKPLACE STANDARDS IN FIBERS/CC TWA[104/]

CV	GSD 1.5	1.6	1.7	1.8
.4	.49	.59	.70	.84
.5	.54	.65	.77	.91
.6	.60	.72	.85	.99
.7	.67	.79	.93	1.09

104/ From Chase & Rhodes Statement, Table 2.

generally accepted detection limit for the membrane filter method.[105/]

The calculations presented in Table 2 show that the lowest asbestos standard which permits the possibility of achieving the desired degree of confidence, without requiring measurement below 0.1 fiber/cc, falls in the range of 1.5 to 1.7 fiber cc.[106/] As Drs. Chase and Rhodes concluded, it appears unlikely that employers could reasonably assure compliance with a standard of 0.5 fiber/cc TWA, the proposed Ontario standard for amosite asbestos:[107/]

> Using the median variability in true daily exposures found by NIOSH for particulates such as asbestos dust, and the lowest CV estimated in Table 1 for a measured value of 0.1 fiber/cc, an

105/ Chatfield Statement at 12; E. Chatfield Tr., Vol. XVIII at 38; Chase & Rhodes Statement at 22; G. Chase Tr., Vol. XXVII at 60; M. Trudeau Tr., Vol. XXII at 94 (July 24, 1981). Although Mr. Rajhans suggested that the 0.1 fiber/cc detection limit could be reduced by using a longer sampling time or by counting more fields on the filter (S. Rajhans Tr., Vol. XLIIA at 57), other witnesses uniformly disagreed. For example, Dr. Chatfield testified that the 0.1 fiber/cc detection limit represents the "best value" approach with a rigorous fiber count and a suitably loaded filter, and cannot be reduced by filtering a larger volume of air. Chatfield Statement at 17; E. Chatfield Tr., Vol. XVIII at 40. Similarly, Mr. Trudeau observed that the 0.1 fiber/cc detection limit could not be reduced by using longer sampling times because the accretion of non-asbestos material on the filter would make the filter "unreadable." M. Trudeau Tr., Vol. CII at 94. And Dr. Chase testified that even if a larger part of the filter were counted, the problem with the detection limit would remain due to other sources of error. G. Chase Tr., Vol. XXVII at 97-100.

106/ Chase & Rhodes Statement at 22.

107/ Id. at 21.

> employer would have to obtain a monitoring measurement below 0.1 fiber/cc to provide reasonable confidence that no more than 5% of employee exposures will exceed a 0.5 fiber/cc standard. Since the membrane filter method cannot reliably measure concentrations below 0.1 fiber/cc, no employer could state with reasonable confidence that his workplace complies with such a standard, under the stated conditions.

The minimum range presented by Drs. Chase and Rhodes was intended "to illustrate the difficulties which would result from setting very low asbestos standards, and should not be regarded as a desirable range for standard setting."[108/] The calculations do not take into account additional variability from systematic or persistent error in sample collection, which was not measured in the round-robin,[109/] although such error may be relatively small[110/] and might increase the lower bounds only slightly.[111/] In addition, the technological feasibility of reducing asbestos concentrations to extremely low levels was not considered.[112/]

Nevertheless, the Chase and Rhodes analysis "suggests that the adoption of very low workplace standards for asbestos

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108/ Id. at 22.

109/ Id. at 22-23.

110/ H. Rhodes Tr., Vol. XXVII at 16.

111/ G. Chase Tr., Vol. XXVII at 70-71.

112/ Chase & Rhodes Statement at 23.

is infeasible."[113/] Similarly, it suggests that standards in the range of one to two fibers/cc could be implemented if employers maintain workplace concentrations well below the standard.[114/]

Although Dr. Chatfield did not present an equally detailed analysis of minimum standards, his views are in close accord with those of Drs. Chase and Rhodes. Based on a detection limit of 0.1 fiber/cc, Dr. Chatfield testified that "it's really questionable whether point one and point two can be shown to be statistically different using the current methods,"[115/] and agreed that the optical method is not sensitive enough for meaningful control at 0.2 fiber/cc.[116/] As Dr. Chatfield concluded,[117/]

> [t]hat has some consequences for enforcement of a point two fiber per milliliter standard for crocidolite. One is not able to reliably say that the maximum is different from your detection level.

Dr. Chatfield testified that a standard of one fiber/cc would permit meaningful measurement.[118/]

113/ Id. at 23.

114/ Id. at 23.

115/ C. Chatfield Tr., Vol. XVIII at 28; see id. at 106.

116/ Id. at 32.

117/ Id. at 28.

118/ Id. at 32-33, 106-07.

Due to the variability in membrane filter monitoring and variations in actual asbestos concentrations, very low workplace standards -- such as the 0.2 and 0.5 fiber/cc proposals for crocidolite and amosite -- could not be implemented.

c. Achievement of Very Low Workplace Concentrations.

Standards which account for variability in measurement and in actual concentrations by providing an adequate lower range for employer monitoring will assure highly protective conditions in the workplace. In order to assure compliance with reasonable confidence, employers must routinely achieve average workplace levels far below mandatory standards.[119]

Drs. Chase and Rhodes found that employers would need to reduce average TWA concentrations to a small fraction of a 1 fiber/cc standard in order to assure compliance. Their estimates of minimum standards, discussed above, show that reliable measurements of roughly 0.2 fiber/cc would be required to provide reasonable confidence of compliance with a 1 fiber/cc standard.[120] Thus they concluded that:[121]

> [E]ven if workplace standards are not reduced below 1 fiber/cc TWA, average workplace concentrations will be maintained at extremely low levels.

[redacted]

119/ Chase & Rhodes Statement at 21; G. Chase Tr., Vol. XXVII at 61-63.

120/ Chase & Rhodes Statement at 24.

121/ Id. at 24.

Data from the Simpson Report provide a practical illustration of the relationship between mandatory standards and workplace levels. Even under the prevailing two fiber standard, median measurements in manufacturing industries in the United Kingdom, for the period from November 1972 to February 1976, were below 0.5 fiber/cc. There was a high degree of variability in the measured values for each of the five asbestos industries evaluated.[122]

Accordingly, workplace standards should be set with the practical expectation that employers will maintain average exposure levels far below the mandatory standards. Standards in the range of one to two fibers/cc, which can be implemented with available monitoring technology, will result in highly protective occupational conditions.

B. Environmental Monitoring.

There is currently no standardized technique suitable for monitoring asbestos concentrations outside the workplace. It is generally agreed that the membrane filter method is unsuitable for monitoring at the extremely low concentrations found outside the workplace. Although electron microscope techniques provide some useful information, there is presently no standardized technique which can be related to a numerical standard.

122/ Id. at 23-24; R.C.A. Exhibit 28, Tab 5, Simpson Report, Vol. I at 17 (1979); G. Chase Tr., Vol. XXVII at 72.

[redacted]

Dr. Chatfield's studies of environmental and building samples in Ontario suggest that asbestos concentrations are not elevated above normal background levels. Thus, there may be no need for numerical standards outside the workplace. Moreover, without a standardized quantitative method for monitoring, such numerical standards could not be adopted.

1. Absence of an Appropriate Standardized Method.

Although very useful for occupational monitoring in the asbestos industry, the membrane filter method is inadequate for monitoring outside the workplace. Airborne asbestos concentrations in non-occupational settings are much lower than the 0.1 fiber/cc detection limit for the membrane filter method. Moreover, as Dr. Chatfield testified:[121]

> [T]he detection limit of 0.1 fibre/ml for the optical method cannot be improved merely by filtration of a larger air volume. If the fibre count is performed rigorously and the filter is suitably loaded, the detection limit approaches the best value of about 0.1 fibre/ml and cannot be reduced further.

In addition, airborne asbestos fibers outside the workplace usually have diameters below the limits of resolution for optical microscopy.[122] "They are usually one, two

[redacted]

[121] Chatfield Statement at 17; see E. Chatfield Tr., Vol. XVIII at 49.

[122] Chatfield Statement at 17, 18; E. Chatfield Tr., Vol. XVIII at 49, 51.

three fibrils of diameter ... no very large bundles. The only thing you ever see by optical microscopy is a bundle of fibers."[123]

The membrane filter technique is also inappropriate outside the workplace because it cannot be used to distinguish between asbestos fibers and other fibrous material. As Dr. Nicholson testified:[126]

> [M]ost of the fibers that you count at the levels that you are measuring asbestos in the air are organic fibers and fibers of other types. Without a great deal of trouble, using distortion staining and petrographic microscopic techniques, you cannot determine with phase contrast microscopy whether those are asbestos or not, and thus a simple counting of fibers in environmental circumstances has no relationship to the amount of asbestos that's there or the number of fibers of asbestos that is there.

Similarly, Dr. Chatfield presented specific examples of air samples from buildings, which would give high fiber counts but are known to contain few or no asbestos fibers.[127]

For these reasons, Dr. Chatfield concluded that membrane filter results should not be accepted for samples from the ambient air or from buildings where asbestos is not being manipulated.[128] Dr. Nicholson also testified that

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[123] E. Chatfield Tr., Vol. XVIII at 51.

[126] V. Nicholson Tr., Vol. 13 at 34 (emphasis supplied).

[127] E. Chatfield Tr., Vol. XVIII at 49-50.

[128] Id. at 50; Chatfield Statement at 17.

"you cannot use phase contrast microscopy" for environmental measurement.[129/]

In contrast to the membrane filter method, electron microscope (EM) techniques provide higher resolution and lower detection limits, as well as the capability to distinguish asbestos fibers from other fibers. However, they are presently unsuitable for quantitative assessments in connection with a numerical standard, as there are no standardized procedures for SEM analysis,[130/] and no evidence that standardized procedures have been developed for TEM analysis. As Mr. Trudeau testified, a numerical standard is dependent on the measurement method. "It's the most important thing because without a method, a number doesn't mean anything ...."[131/] Thus, there can be no meaningful SEM or TEM-based standard until a standardized EM method is developed, and even when that occurs, other roadblocks to routine use would exist.

EM measurements cannot be related to membrane filter measurements or to the existing health evidence. Drs. Chase and Rhodes found:[132/]

> The SEM would count large numbers of very short and very thin fibers that cannot be seen by current optical methods and that were not included in the

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129/ W. Nicholson Tr., Vol. XV at 34.

130/ See pp. VI-13 to VI-17, supra.

131/ M. Trudeau Tr., Vol. XXII at 47.

132/ Chase & Rhodes Statement at 11-12.

> exposure estimates for available epidemiologic studies on the health effects of asbestos exposure. ... There is no known relationship ... between SEM counts and optical measurements, and no way to relate an SEM-based standard to the available epidemiological data which are based on optical methods or approximations to optical counts.

Similarly, Mr. Trudeau described TEM counts as "orders of magnitude higher" than membrane filter counts,[133/] and testified that a comparison of TEM data with a membrane-filter based standard would be "like comparing apples with oranges."[134/] Because membrane filter measurements are an index "unrelated to any standard samples," Dr. Chatfield concluded that "new methods cannot easily or convincingly be related to the techniques in use today."[135/] Thus, there is no present basis for establishing SEM or TEM-related standards.

Various technical problems must also be resolved before environmental standards based on EM techniques become feasible. For example, in addition to problems with filter selection and preparation for SEM analysis[136/] and the danger that fibers will be broken up or lost during TEM sample preparation,[137/] the preparation of ambient air

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133/ M. Trudeau Tr., Vol. XXII at 49.

134/ Id. at 48.

135/ Chatfield Statement at 3-4.

136/ See pp. VI-13 to VI-14, supra.

137/ See pp. VI-18 to VI-19, supra.

samples for EM analysis is particularly difficult. As Dr. Nicholson testified:[138]

> [C]hrysotile is a very friable fiber, and in order to identify the fibers in air samples collected in the ambient air you have to disperse the sample because there is so much other garbage present to which the fibers adhere, that obscures their viewing in any direct transfer process.
>
> In the dispersal of that material, either with ashing or ultrasonic techniques, you break apart the fibers, and so counting the fibers can in fact overestimate their number by virtue of that number being increased through the processing.
>
> So a fiber count of the ambient air level, even if you could make a conversion between electron microscopic fiber counts and optical fiber counts, would be a difficult and misleading ... could provide misleading information.

Dr. Nicholson and other researchers have used mass measurements rather than fiber counts to quantify asbestos concentrations in the ambient air outside the workplace.[139]

Detection limits for EM analysis are much lower than the 0.1 fiber/cc limit for the membrane filter method, but may be inadequate for urban air samples. Although Dr. Chatfield found "no particular difficulty" in meeting a detection limit of one fiber/litre in a clean atmosphere,[140] he

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[138]/ V. Nicholson Tr., Vol. XV at 34-35.

[139]/ Id. at 35.

[140]/ E. Chatfield Tr., Vol. XVIII at 37-38.

testified that "in the urban atmosphere if I was to take a sample outside in the city of Toronto, we would have difficulties in some cases in meeting detection levels of the Ontario guidelines -- forty fibers per litre longer than five micrometers," due to suspended particulate matter.[141] Dr. Chatfield is investigating possible analytical procedures which would meet a detection level of one fiber/litre in urban atmospheres.[142]

Thus, although EM methods meet some of the requirements for environmental monitoring, they are presently inadequate to support a numerical standard for fiber concentrations outside the workplace.

2. Asbestos Concentrations in Ontario.

Asbestos is a ubiquitous material, occurring naturally in air and water. As Dr. Chatfield testified, "[t]here is absolutely no doubt that one does find a natural background."[143]

Dr. Chatfield's studies indicate that asbestos concentrations in Ontario are equivalent to background levels. As described above, EM techniques presently cannot provide quantitative data for comparison with a numerical standard.

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[141]/ E. Chatfield Tr., Vol. XVIII at 38. "[O]ne may have as many as several milligrams of total solids on the filter, amongst which you are trying to find a very few asbestos fibers." Id.

[142]/ E. Chatfield Tr., Vol. XVIII at 38.

[143]/ Id. at 40.

but the results obtained by a single investigator using his own technique may be used to evaluate the relative concentrations at two or more sampling sites.

For example, in order to determine whether asbestos building insulation adds to the natural background, resulting in higher fiber concentrations, Dr. Chatfield has made measurements inside and outside buildings. "[V]ery largely, the buildings have not in fact given any fibers above what we would expect to find outside."[144/] Dr. Chatfield has found some elevated levels in buildings following removal of insulation, which he presumes resulted from inadequate clean-up after the removal operation, although none of these concentrations exceeded the Ontario guidelines for environmental concentrations.[145/] These findings, showing that building levels are equivalent to background levels except following removal of asbestos materials, raise questions about the wisdom of removing insulation absent direct evidence that the insulation is contributing to higher airborne fiber concentrations.

Dr. Chatfield has also studied drinking water supplies at eighteen locations in Ontario, representing fifty-six

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144/ Id. at 40.

145/ Id. at 95. A recent study of buildings containing asbestos materials in Great Britain also failed to show airborne asbestos concentrations above the level of detection, using several measurement techniques. Le Guen and Burdett, 1981; see discussion at pp. I-27 to I-28, supra.

percent of the provincial population.[146/] He reported that in the majority of these locations, the concentrations "were essentially zero," close to the detection level.[147/] The two exceptions were Thunder Bay and Hearst, but at Thunder Bay the results were not statistically different from zero.[148/] Moreover, with respect to both Thunder Bay and Hearst, no remedial action was taken or recommended. As Dr. Chatfield testified, "let's put it this way ... I would drink the water."[149/]

The Chatfield studies, while perhaps not definitive, suggest little need for environmental and building standards in Ontario. Such standards should not be adopted absent development of a standardized quantitative method for monitoring.

146/ E. Chatfield Tr., Vol. XVIII at 90.

147/ Id.

148/ Id. at 90-92.

149/ Id. at 92.

CHAPTER VII

VII. APPROPRIATE CONTROL MEASURES FOR FUTURE ASBESTOS USE.

The evidence that the harmful effects of asbestos are dose related, the great progress that has been made to reduce asbestos exposures, and the predictions from the medical evidence that de minimis, if any, risk exists at the low levels that have been achieved in most asbestos applications today, establish the major guideposts for developing a societal policy for future asbestos use. Dr. Henry Anderson, formerly of the Mount Sinai research team and now a public health official in Wisconsin, perhaps most succinctly summed up the conclusion of the medical witnesses:1/

> I think asbestos can be handled safely. It's a dangerous substance, yes, but it can be handled safely ...
>
> I'm not saying there is a safe threshold. But I think if you are ... there may well be a relative risk, if we talk in those terms, that will get down so low that any excess one would see would be moderate, that you'll have a very small work force who will be adequately protected. ... I think you can handle the material and I think there are ... this would be for the engineers to tell you ... situations where it is wholly appropriate.

At the same time, the sometimes conflicting evidence and uncertainties point to the need for careful surveillance of

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1/ H Anderson Tr., Vol. XVI at 131, 132.

asbestos use in order to avoid unnecessary risk. The following discussion of a societal program to control asbestos emissions incorporates such cautious guidance.1/

In formulating a regulatory scheme to ensure safe handling of asbestos, cautionary advice presented by a number of the medical witnesses is worth heeding. They stressed the importance of formulating workable, enforceable regulations as opposed to ideal standards that will not in fact be achieved. As Dr. Muir commented at the Commission's Second Public Meeting:2/

> If I were in the asbestos industry, I would rather have 95% confidence that no worker is going to be exposed to greater than a certain fibre level, rather than

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1/ No discussion is included of substitutes for asbestos. As noted in Chapter I, almost all current uses employ asbestos embedded in a matrix that greatly minimizes fiber release. Production facilities have vastly improved exposure levels. As discussed in Chapters III and IV, the occupational risks are de minimis when emissions are properly controlled. Thus, the need for substitution should be primarily an economic question of whether, when such protective measures are used, asbestos will still be a competitive material. If it is, then no substitutes should be mandated as they cannot be justified from a health standpoint.

Moreover, as Dr. Corbett McDonald noted, great caution should be exercised from a health standpoint in mandating asbestos substitutes:

> By all means, I'm sure industry will try to ... develop safer substitutes. I just hope that when they develop these *safer* substitutes, they will take into account that nobody knew the hazards of asbestos until they'd been using it for about seventy years. So we have to be a wee bit cautious.

C. McDonald Tr., Vol. XIII at 119.

2/ Minutes of Second Public Meeting, at 19 (Dec. 12 1980).

> 50% confidence in relation to some more rigid level. I do not think we can avoid this decision that, in getting standards, one has to take account of how readily and how easily they can be measured, and what is the likelihood that they will in fact be observed ... It seems to me that defining standards which are subsequently ignored is likely to be an exercise in futility for everybody.

Dr. Anderson expressed similar views:3/

> [W]hen you are sitting in an office or room such as this it is fairly easy to say well, suppress the dust. But when you get out onto the mill or factory floor, that becomes a very difficult task at times, and we do need to keep in mind that the standard isn't what protects the health, but the actual level that's maintained at the exposure site.
>
> So the lowest possible standard will do nothing to protect the health of the worker if in fact it is not enforced, or we are unable to attain it.

As emphasized by Drs. Muir and Anderson, the task confronting this Commission and other governmental bodies investigating asbestos is to determine what reasonable and workable control measures exist to assure acceptably safe asbestos working conditions.

A. Asbestos Safety in the Mines and Mills.

Little doubt exists as to the acceptably safe levels now existing in the chrysotile mines and mills of Canada.

3/ R. Anderson Tr., Vol XVI at 6.

No dispute exists over the fact that the McDonald study is the preeminent study for determining whether any risk exists in such settings, nor that even conservative linear extrapolation from that study predicts risks well below what workers and the public would consider acceptable. For the future, therefore, what is important will be continued dust control and surveillance to maintain such acceptable working conditions.

The Quebec Asbestos Mining Association detailed for the Commission the steps its members have taken to achieve such safe conditions.[1]

> During the period 1970-1980 inclusive, more than one hundred million dollars have been invested in environmental control within the primary Quebec asbestos industry. To date all equipment has been enclosed and operated under vacuum and the percentage of exhaust air used exclusively for dust control has been raised to more than 40% of the total . . . . In modern plants, bagging, sewing, sealing, palletizing, storing, regrading, shipping and dry rock storage operations have been fully mechanized and automated and all employees have been trained in proper handling of asbestos. High capacity water tanks have been added to control dust on mine roads and platforms; in heavy equipment air conditioning units have been installed in the cabins; bag filter units have been added to cyclone collectors at all remote ore and waste mechanical transportation and transfer points; high capacity central vacuum systems have

[1] Brief to the Commission of the Quebec Asbestos Mining Association (QAMA), at 35-36 (Jan. 1981).

> replaced brooms for cleaning floors and equipment; jute bags have been eliminated and replaced by dust proof paper and plastic bags; standard clean maintenance procedures have been developed for mill equipment.

To ensure the effective operation of this equipment, extensive and careful steps are taken to monitor workplace conditions:[1]

> Weekly, semi-annual personnel-dynamic and spot surveys are carried out on a regular basis using both fibre counts and gravimetric measurements. High volume air sampling is also read daily at stations located around the periphery of the property. Union and company technicians work in parallel during all these surveys and the results are passed on to both the Department of Natural Resources and the President of the union. These data are also reported to all the employees through the monthly union letter to membership.

As a result of this expensive and extensive dust control program, the Quebec mines and mills have substantially reduced asbestos exposure in the past decade and are now able to comply with the 2 fibers/cc standard established after Quebec's Beaudry Commission inquiry. In light of the medical evidence that establishes the acceptability of such working conditions, it is clear that such a standard is adequate, as well as workable.

Although there are no working asbestos mines in Ontario presently, asbestos deposits do exist and the possibility of

[1] QAMA Brief, supra note 1, at 36.

mining exists. It would therefore be useful for this Royal Commission to recommend a standard for the mines and mills in Ontario equivalent to that existing in Quebec. Such a standard has been shown to be practical, achievable and protective of worker health.

B. Asbestos Safety in Manufacturing.

As in the mines and mills, tremendous progress has been made in the asbestos manufacturing industry to reduce asbestos exposures. As detailed in Chapter I, the historical high exposures responsible for the disease occurring today are -- by orders of magnitude -- a thing of the past. Even in the past decade continuing progress has been achieved as industry has met the prevailing 2 fibers/cc standard throughout the Western world. Current average exposures in most manufacturing facilities thus are often below .5 fiber/cc.

Dr. Finkelstein, who was familiar with the asbestos-cement operations at the Toronto Johns-Manville plant, described the great progress made in factory dust control:[7/]

> Johns-Manville, before they shut down, set an excellent example for the rest of the asbestos industry in Ontario.
>
> They were routinely bettering half a fiber, both government and company hygienists were routinely measuring exposures of point one to point three fibers.

7/ M. Finkelstein Tr., Vol. XXIV at 144.

> So I think this is an example which stands out in Ontario and which I think other industries and factories should attempt to emulate.

Dr. Bragg's extensive research report for the Commission agreed:[8/]

> There has been an enormous improvement in the reduction of industrial fibre level concentrations over the last five to ten years. The fundamental reason for this has been a considerable increase in the expertise of those attempting the controls. Although there have been few or no technological breakthroughs in this field, many of the best practices have been adapted from other fields where the toxicity level of the material being handled is much higher than that of asbestos.
>
> * * *
>
> Best available technology in asbestos dust control is now approaching that for beryllium which is the most toxic solid in industrial production.

As noted in Chapters IV and V, efforts to assure compliance with a 2 fibers standard will inevitably, and have in fact, led to average workplace exposures one-fourth or less than the standard. Thus, in many manufacturing facilities today, as was true in the Toronto asbestos-cement plant, average exposure levels are already well below Ontario's proposed chrysotile standard of 1 fiber/cc. As Dr. Bragg found:[9/]

8/ Bragg, Gordon M., "The Technological Feasibility and Cost of Controlling Workplace Exposure to Asbestos Fibres," 101, 102 (Royal Commission Study, April 1982).

9/ Id. at 51.

> [M]any of the processes which we observed in industry that produced very low fibre levels -- around 0.2 to 0.1 f/cc -- were achieved by companies attempting to meet the present fibre standards or in some cases having processes which are only generating low fibre levels.

At the same time, current exposures in most manufacturing plants are so low that the limits of monitoring precision and validity are being approached. As discussed in Chapter VI, the lowest level that the membrane filter method -- the only technique now available for routine occupational monitoring -- can reasonably distinguish from background is .1 fiber/cc. With this limitation, and the substantial variability of the method, especially at low concentrations, it is not possible to establish reasonably verifiable standards much below the *proposed* 1 *fiber/cc* standard.

Similarly, industry has been pushed to the limits of technological feasibility in achieving compliance with a 2 fibers/cc standard in many manufacturing operations. As Dr. Bragg concluded:[10/]

> We have found some secondary industries capable of producing fibre levels consistently at or near this [0.5 f/cc] level; however, this capability has only been achieved in the last year or two and, typically, *for processes* with low emissions originally. On the other hand, a number of specific processes are not capable of achieving fibre levels below 2 f/cc with engineering controls.

10/ Id. at 100.

Achievement of lower levels would be prohibitively expensive in most Ontario manufacturing facilities, according to Dr. Bragg:[11/]

> Control of dust emissions to the 0.1 f/cc level, where possible, would require extensive redesign and rebuilding of control systems. For some industries cases purchase of new production equipment would be necessary, making this level of control significantly more expensive than the higher levels.

Mr. Rajhans agreed that achievment of TWA fiber levels below 0.5 fiber/cc could be achieved only through extremely expensive technology:[12/]

> [L]ocal ventilation and careful housekeeping practices, which I have indicated had resulted in reducing the exposures in various plants down to about ... one fiber per milliliter ... have reached the plateau and anything below one needs different control measures and whatever.
>
> * * *
>
> [T]he technologies ... concerned ... are available. But the cost will be, perhaps, prohibitive, as well as the installation and any other method that can be used will take much longer time to start anything of that kind ... .

It is thus apparent that manufacturing plant exposures have been greatly reduced to levels that today are quite close to the limits of what any reasonable government regulation could require. As discussed in Chapters III and IV,

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11/ Id. at 101.

12/ S. Rajhans Tr., Vol. XIIIA at 65.

the medical evidence indicates that such exposure levels will be acceptably safe for the small number of workers engaged in such asbestos manufacturing operations.

When dealing with manufacturing safety, an additional issue is raised as to whether differential standards for the various fiber types are desirable. At present, this issue is of little significance in Ontario where most manufacturing plants are in the friction products industry and thus use exclusively chrysotile. However, most North American asbestos-cement pipe manufacturing employs crocidolite fiber. The possibility that such pipe production might be reinitiated in Ontario is deserving of consideration.

New British regulations by the Factory Inspectorate in 1970 introduced a hygiene standard for crocidolite ten times lower than that for other forms of asbestos. Other countries including Sweden and the province of Ontario, in its 1980 proposal, have singled out crocidolite and amosite as requiring especially careful handling. U.S. government officials, however, have consistently declined to draw fiber type distinctions. For example, a recent NIOSH-OSHA Report concluded:[13/]

> On the basis of available information, the committee concludes that there is no scientific basis for differentiating between asbestos fiber types for regulatory purposes. Accordingly, the committee recommends that a single occupational health standard be established and applied to all asbestos fiber types.

13/ R.C.A. Exhibit 26, Tab 11, NIOSH-OSHA Asbestos Work Group - April 1980, "Workplace Exposure To Asbestos - Review and Recommendations." DHEW (NIOSH) Publication No. 81-103, at 2 (November 1980).

As discussed in Chapters II and III above, the medical witnesses before the Commission have devoted considerable attention to the relative pathogenicity of different fiber types. Although recognizing that much of the animal and *in vitro* data concluded otherwise, and noting that the epidemiology results are limited in their interpretability because of the absence of well-documented dosage data, the prevailing view of the witnesses was that crocidolite and amosite pose higher risks, particularly of mesothelioma, than does chrysotile. Indeed, even Julian Peto, the Commission witness who argued most strongly against a mesothelioma fiber type distinction, concluded by noting the pragmatic considerations that would lead to tighter standards for crocidolite:[14/]

> [T]here may be no difference fiber to fiber in the risk associated with crocidolite, but it may well be ... and I think, in fact I would expect that for given industrial processes with the same control procedures the risk associated with crocidolite is likely to be considerably higher than it is for chrysotile.

Thus, in accord with the conclusions of the United Kingdom's Simpson Report, the Commission's medical experts all would argue in favor of tighter control standards for amphiboles.

14/ J. Peto Tr., Vol. XXVB at 31.

On the other hand, even if these fiber type differences are recognized from a medical standpoint, they may be of limited significance to attainment and monitoring of permissible workplace exposures. Amosite is rarely used today, and crocidolite is used only in limited applications where, once the raw fibers are combined, it is typically a minor percentage of the total fiber mix. Any airborne crocidolite concentration would thus usually represent but a portion of the total asbestos aerosol. Were an asbestos concentration of 2 fibers/cc measured without distinctions being made about fiber type, for example, the great likelihood would be that the crocidolite concentration in this sample would be much smaller. Recent data from an asbestos-cement plant in Italy confirm this relationship.[13/] In many circumstances, therefore, a workplace meeting the current standard (e.g., 2 fibers/cc) for all asbestos would at the same time be meeting a much tighter standard (e.g., .2 fiber/cc) for crocidolite.

As discussed in Chapter VI above, although the membrane filter method cannot be used to distinguish among fiber types, occasional electron microscopy characterization of

13/ Cocriani, G., et al., "Routine Evaluation of Fibre Concentration in Asbestos Cement Working Environments: Proposal of an Alternative Limit," extract from the Third National Congress of the Italian Industrial Hygienists Association (Genoa, November 26-28, 1980). Measurements of asbestos concentrations in this Italian asbestos-cement factory revealed an average ratio of chrysotile:crocidolite airborne asbestos concentrations of 83:20, which closely paralleled the product fiber mix.

dust cloud constituents could be used to define the fiber type mix in a given occupational setting. As long as fiber mix or major production changes were not instituted, it could safely be assumed that the dust cloud mixture would remain fairly constant, and routine membrane filter monitoring could thus be used to assure compliance with a differential standard.[14/]

In sum, major advances have been made in achieving very low exposures in manufacturing facilities. Under the prevailing two fiber standard, most plants have achieved average exposure levels below .5 fiber/cc. Further significant reduction in such exposures, however, cannot be mandated given the limitations of monitoring capabilities and of reasonable technological feasibility and is not compelled by adverse medical evidence.

C. Work Practices for Non-Fixed Site Workplaces.

Outside of the mines and mills and asbestos manufacturing plants, the nature of asbestos exposures and of control measures to eliminate unnecessary exposures are quite different. In settings such as construction, pipe installation,

14/ Mr. Rajhans of the Ontario Ministry of Labor recognized the feasibility of using product mix bulk samples and occasional electronic microscopy airborne samples to characterize the TWA when more than one asbestos fiber is being used. G. Rajhans Tr., Vol. XIIIA at 40-52. However, he indicated that the Ministry is planning to interpret its new asbestos regulation to require that the lower standard be met whenever mixed fiber types are in use. Id. at 52-56.

brake repair, and building renovation and removal, workers may intermittently be exposed to high concentrations of asbestos if proper work practices are not employed. If, on the other hand, recommended work practices are employed, only de minimis exposures will occur.

The ability of proper work practices to control intermittent peak exposures and thus eliminate any asbestos exposure problem is exemplified well by the data from garages where brake replacements occur. Although peak, short-term exposures as high as 87 fibers/cc have been measured during air hose blowing of debris from brakes that are having their lining replaced, background levels even when such non-approved methods are used are orders of magnitude lower.[17/] It is thus unlikely that mechanics have ever been exposed at TWA levels higher than present occupational standards. If, however, approved work practices, some of them as simple as using a wet rag instead of air hoses for removing dust, are employed, even peak exposures are well below 2 fibers/cc. The result is de minimis overall exposures. As Dr. Nicholson concluded in his paper reviewing data from garages both employing and not employing approved work practices:[18/]

17/ See, e.g., Knight, H. L. and O. E. Nichish, "Investigation into Alternative Forms of Control for Dust Generated During the Cleaning of Brake Assemblies and Drums," 13 Ann. Occup. Hyg. 37-39 (1970).

18/ Nicholson, W. J., "Investigation of Health Hazards in Brake Lining Repair and Maintenance Workers Occupationally Exposed to Asbestos: Task I, Description of Brake Materials, Work Practices and Exposure Levels During Brake Maintenance," Draft for Comment, at 1- (Jan. 1, 1979).

> The existing data indicate that the majority of garage employees are not likely to have been exposed to long-term (over months or years) asbestos concentrations in excess of the current standard of 2 f/ml. The data also show that virtually all asbestos dust is released during easily controlled work activities and that with proper practices TWA asbestos concentrations can be reduced to about 0.1 f/ml or less.

The same pattern of rare, intermittent peak exposures that can be well-controlled by use of recommended work practices exists in asbestos-cement pipe installation as detailed in Chapter IV.

As the brake mechanic and pipe installation scenarios portray, significant features distinguish such exposures from the fixed site workplace for which health and safety regulations are ordinarily designed. These settings are characterized by high employee turnover due to a transient and temporary workforce, predominance of small firms with a high failure rate due to wide seasonal variation in demand, and multiple, discrete tasks with no regularity of worker movement among them and inconstant potential exposure to asbestos. These same features are generally characteristic of all asbestos use in the construction industry, including installation of asbestos-cement sheet, replacement of asbestos floorings, and demolition activities. As Dr. Nicholson noted, elimination of intermittent peak exposures in demolition will "largely control" any asbestos problem:[19/]

19/ Nicholson Tr., Vol. IV at 90.

> But the exposures in maintenance and repair, and in construction work where asbestos is already in place, are largely going to be those of the peaks. If you can eliminate those peaks and deal with those, and focus on how to minimize that peak exposure, you can largely control the problem.

In all these types of activities, the most effective control techniques will be those that reduce substantially the few peak exposures.

In these settings, many of the provisions appropriate for fixed site manufacturing workplaces are impractical. For example, monitoring at construction sites or garages is virtually meaningless, yet very expensive. It is meaningless because there is no consistency to the circumstances of exposure among worker tasks and because sample monitoring has shown exposures to be very low except when a few non-recommended work practices are employed, and expensive because essentially constant monitoring might be required to get a representative sample.

AIA/HA has recently studied these problems in the United States and developed, in conjunction with the Association of Asbestos Cement Pipe Producers, an alternative Recommended Standard for Occupational Asbestos Exposure in Construction and Other Non-Fixed Work Operations.[19/] Briefly, the model standard relies on product categorization and

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19/ AIA/HA's extensive documentation of its proposal was submitted with its Initial Brief to the Commission (1981).

certified work practices rather than engineering controls and monitoring to control asbestos exposures. For those products that testing revealed do not release more than the existing permissible exposure limit (PEL) under any foreseeable conditions of installation or use, no exposure controls would be required. A government would not itself have to determine what individual products meet these criteria as "Category A" products, but could specify laboratories qualified to do the testing or set standards for the acceptance of manufacturer tests.

For a second category of products, "Category B," for which testing revealed potential releases in excess of the PEL under some conditions, but for which exposures could be below that level through the use of proper work practices, the model standard would require that the materials be handled only in accord with approved work practices. The certification of proper work practices could also be done through independent laboratories.

For the third category of asbestos-containing products, "Category C," which covers all materials not meeting the criteria of A or B, the general asbestos control regulations would apply. There is no requirement for medical surveillance where only Category A and B products are in use, since, by definition, exposures are below the control limit.

The Model Standard provides significant advantages over the normal fixed-site regulation both for enforcement and

compliance purposes. At worksites where only Category A and B products are in use, inspectors could ascertain through visual inspection alone whether an employer was in compliance by simply checking that certified work practices were being followed and no Category C products were present. This would avoid the necessity of monitoring, waiting for analysis of the samples, and then returning to the worksite to charge any violations, by which time the nature of the work being performed might have changed completely.

For similar reasons, employers would have an incentive to select and use Category A and B products. Ensuring their own compliance with the regulation would thereby be facilitated, and they would not have to undertake costly medical surveillance measures since, by definition, exposures would be below the control limit. In the face of demand from construction contractors, manufacturers would have a substantial incentive to develop and market products that meet the criteria of Categories A and B.

The Model Standard, which could be easily adapted for Ontario, would introduce free market inducements in aid of the government/industry cooperation that will be necessary to make any asbestos exposure control succeed.

During his Commission testimony, Dr. Nicholson supported adoption of such work practices:[21]

21/ W. Nicholson Tr., Vol. XIV at 81.

> [O]ne need adopt largely procedural methods to devise ways that people required to undertake maintenance activities or removal activities, or otherwise in the course of their work deal with the asbestos that's there, do so in a safe way, both for themselves and for anybody else that might be involved. This largely is a matter of having proper clothing, proper respiratory protection and proper activities identified and utilized.

Dr. Nicholson concurred with the basic rationale for use of mandated work practices, namely that they will reduce exposures to very low levels and are much more expeditiously monitored than dust concentrations:[22]

> The specification of work practices and engineering controls offers an essential supplement to a numerical TLV if the latter is used at all. Application of many economically and technically feasible procedures can reduce exposure to levels much below existing numerical values. These should be mandated. Moreover, such procedures can be specified with joint government-union-management cooperation and can be monitored much more readily than can dust concentrations.

Richard Lemen of the U.S. NIOSH also agreed with the AIA/MA proposal:[23]

> Many of our health problems have been eliminated purely by a simple equipment change, and outside the asbestos industry I think that the garbage industry, the refuse collectors-excess cardiovascular

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22/ R.C.A. Exhibit 18, Tab 5, Nicholson, W., "Case Study I: Asbestos--The TLV Approach," 271 Ann. N. Y. Acad. Sci. 152-68, at 167 (1976).

23/ R. Lemen Tr., Vol. XVII at 120.

> disease has been somewhat affected by just changing the exhaust system upwards instead of back towards the worker.
>
> So I think that these are constructive and I would hope that more of these would be forthcoming from trade associations and industries and the labour movement.

There seems little doubt that asbestos can safely be handled in construction, garage repair work, and demolition activities if recommended work practices are employed. Even peak exposures will be well below current occupational standards in such settings, and thus time weighted average exposures will be de minimis. Indeed, it is possible that the elimination of the type of peak exposures that can occur when recommended work practices are not employed will be of inordinate medical significance, as Julian Peto has noted: 21/

> If certain work practices constitute the major source of risk and actual exposures are considerably higher than static sampling suggests, dose-response analyses based on static measurements may be misleading. Avoidance of such practices, which may, by accident or design, not occur during personal sampling, may be more useful than the imposition of very stringent hygiene standards without adequate personal monitoring.

Appropriate societal regulation of asbestos in the occupational settings characterized by intermittent exposure thus should focus on requiring recommended work practices.

21/ E.C.A. Exhibit 37, Tab F, Peto, J., "Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory," in Biological Effects of Mineral Fibres 829-36, at 834 (Lyon, IARC 1980).

As contemplated by the AIA/NA proposal, products can be characterized into groupings that indicate when, and what, recommended work practices must be employed. Such regulations can be easily enforced at minimal costs with great efficacy. It is quite unlikely that any substantial disease will occur due to asbestos exposures among workers with such infrequent, intermittent exposures even when non-recommended work practices are employed. Nonetheless, the minimal difficulty of assuring that exposures are clearly de minimis should lead this Commission to recommend strongly the employment of a scheme such as that AIA/NA has proposed.

D. Labeling To Promote Safe Handling.

Closely related to a work practices scheme to ensure safe occupational conditions for asbestos is a program for educational labeling of asbestos and asbestos-containing products. Most companies currently do substantial labeling of their asbestos products, but to the extent gaps remain in voluntary efforts, there is a role for governmental assistance. Such a labeling program is, for example, currently under consideration by the U.S. Environmental Protection Agency. 22/

Labeling can serve a vital role in guaranteeing that appropriate practices are used when working with asbestos.

22/ E.P.A., "Commercial and Industrial Use of Asbestos Fibers," 44 Fed. Reg. 71:37-38 (Dec. 17, 1979).

It is obviously desirable, for example, that garage mechanics be warned not to use air hoses to blow out break debris, that asbestos-cement installers be cautioned against use of abrasive disc saws, and that installers of flooring be warned that any old flooring they remove may contain asbestos and thus should not be sanded. Many manufacturers currently label their products with such warnings. Such warnings reach not only persons occupationally involved with such products, but the home handyman who may do his own brake or flooring work.[21]

Labeling may also be useful in buildings where asbestos materials have been installed in the past as, for example, insulation. Although the in-place insulation poses no risk as long as it is not dislodged, the chance that maintenance personnel may not be acquainted with the need for caution in working around the material argues in favor of labeling in order to prevent undue activity in such areas. Dr. Anderson, for example, noted the potentially very significant role of labeling as part of a school surveillance program:[22]

> [W]hen you have a small group of people who are going to be having intimate contact with a given area, you can educate them and alert them to the problem.

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[21] Any labeling program should recognize, as does the AIA/MA work practices scheme described above, that some products, because they will not release significant fibers under any foreseeable conditions of use, need not be labeled.

[22] M. Anderson Tr., [illegible], XVI at [illegible].

> What we do, or part of the recommendations, is to label that.
>
> In other words, they are to take a little tag that says "this material contains asbestos, handle with care", wrap it around the pipe, twist the lock, and somebody comes by, because there is a leak, later on. Hopefully they will see it and if they are properly educated will say, hey, I better find out what to do here.

Such inexpensive labeling could accomplish much in preventing any possibility of airborne asbestos, and, indeed, might well be much more effective than a costly removal program that would risk considerable dispersal of fibers.

In sum, educational labeling offers great opportunities for prevention of asbestos exposures at minimal costs and should be a vital part of any societal program for asbestos control.

t. Smoking Cessation Programs.

The substantial interaction between asbestos and smoking in causing lung cancer provides another substantial opportunity for protection of worker health. As the discussion in Chapter III above emphasizes, excess lung cancer will be exceedingly low, if existent at all, among asbestos workers who do not smoke. Moreover, as the data compiled on the U.S. and Canadian insulation workers has shown, lung cancer risk diminishes substantially once smoking has ceased. Therefore, efforts to employ non-smokers or to encourage cessation of smoking among current workers will pay gigantic

dividends in eliminating any vestigial lung cancer risks among persons exposed to asbestos.

Smoking cessation programs call for close co-operation among workers and management. But, just as employers cannot guarantee workplaces free from accidents unless workers co-operate by following precautionary measures against such occurrences, co-operation in eliminating smoking is an integral part of occupational safety. As Dr. Kotin has written:[28]

> Workplace health and safety is a joint effort, and unless there is a full partnership between labor and management the mutual goal of a healthy workplace will be difficult to achieve. Smoking cessation programs do not "shift the burden" to the worker; rather, they are part of the cooperative and collaborative effort necessary in occupational environmental control.

28/ S.C.A. Exhibit 30, Tab 10, Kotin, P. and L. A. Gael, "Smoking in the Workplace: A Hazard Ignored," 70 A.J.P.H. 575-76 (1980) (emphasis in original).

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