No. 116 Original

RECEIVED
HAND DELIVERED
APR 12 1990
OFFICE OF THE CLERK
SUPREME COURT, U.S.

In The
SUPREME COURT OF THE UNITED STATES
October Term, 1989

ALABAMA, ARIZONA, ARKANSAS, CALIFORNIA, CONNECTICUT, DELAWARE, FLORIDA, ILLINOIS, INDIANA, IOWA, LOUISIANA, MAINE, MISSOURI, MONTANA, NEBRASKA, NEW HAMPSHIRE, NEW YORK, NORTH CAROLINA, NORTH DAKOTA, OHIO, OKLAHOMA, RHODE ISLAND, SOUTH DAKOTA, TENNESSEE, TEXAS, UTAH, VERMONT, WASHINGTON, AND WYOMING,

Plaintiffs,

–against–

W.R. GRACE & COMPANY, NATIONAL GYPSUM COMPANY, UNITED STATES GYPSUM COMPANY, USG CORPORATION, AMERICAN BILTRITE, INC., ARMSTRONG WORLD INDUSTRIES, INC., AZROCK INDUSTRIES, INC., BASIC INCORPORATED, CAREY–CANADA, INC., THE CELOTEX CORPORATION, CERTAINTEED CORPORATION, CROWN CORK & SEAL COMPANY, INC., EAGLE-PICHER INDUSTRIES, INC., FIBREBOARD CORPORATION, THE FLINTKOTE COMPANY, GAF CORPORATION, GEORGIA-PACIFIC CORPORATION, H.K. PORTER COMPANY, INC., KEENE CORPORATION, KENTILE FLOORS, INC., OWENS-CORNING FIBERGLAS CORPORATION, OWENS-ILLINOIS, INC., PFIZER, INC., RAYMARK INDUSTRIES, INC., SPRAYED INSULATION, INC., AND TURNER & NEWALL PLC,

Defendants.

ON MOTION FOR LEAVE TO FILE COMPLAINT

LODGING SUBMITTED BY DEFENDANT GAF CORPORATION

Sidney S. Rosdeitcher
(Counsel of Record)
Robert N. Kravitz
Paul, Weiss, Rifkind, Wharton & Garrison
1285 Avenue of the Americas
New York, New York 10019
(212) 373–3000

RECEIVED
HAND DELIVERED
APR 12 1990
OFFICE OF THE CLERK
SUPREME COURT, U.S.

Randall L. Solomon
Baker & Hostetler
3200 National City Center
Cleveland, Ohio 44114
(216) 621–0200

Patrick J. Lamb
James C. Murray, Jr.
Katten Muchin & Zavis
525 West Monroe Street
Suite 1600
Chicago, Illinois 60606
(312) 902–5200

Attorneys for GAF Corporation

TABLE OF CONTENTS

1. Harvard University Energy and Environmental Policy Center, *Symposium on Health Aspects of Exposure to Asbestos in Buildings* (Dec. 1989).

2. Mossman, Bignon, Corn, Seaton & Gee, "Asbestos: Scientific Developments and Implications for Public Policy," 247 *Science* 294 (1990).

PROCEEDINGS

SYMPOSIUM ON HEALTH ASPECTS OF EXPOSURE TO ASBESTOS IN BUILDINGS

December 14-16, 1988

Harvard University
Energy and Environmental Policy Center

Publication Date
December 1989

PROCEEDINGS

SYMPOSIUM ON HEALTH ASPECTS OF EXPOSURE TO ASBESTOS IN BUILDINGS

December 14-16, 1988

Harvard University
Energy and Environmental Policy Center
65 Winthrop Street
Cambridge, MA 02138

The views expressed in this document are the authors' and do not necessarily reflect those of the Energy and Environmental Policy Center or Harvard University. Reproduction of this paper is not permitted without permission of the Energy and Environmental Policy Center. For additional copies of this document contact Harvard University, Energy and Environmental Policy Center, 65 Winthrop Street, Cambridge, MA 02138.

FOREWORD

Too often in this litigious society, dispassionate and reasoned evaluations are greeted with skepticism. Those who agree with the results will praise the evaluation and those who disagree will labor to discredit it. Even those interests who support such findings will be tempted to extend or misuse them to suit their own agenda.

Few issues are as contentious as that of asbestos in buildings. Therefore it was not surprising that the "Summary of Symposium on Health Aspects of Exposure to Asbestos in Buildings" stimulated responses which at times extended way beyond the findings contained in our original report.

This publication is a compendium of the actual papers produced by the scientists and experts whose work formed the foundation on which our conclusions in the symposium summary were based. We thank these individuals for taking the time to make a thoughtful contribution to the debate on the health aspects of asbestos fibers in buildings.

The symposium was sponsored by the Safe Buildings Alliance, the National Association of Realtors®, the Institute of Real Estate Management Foundation, and the Urban Land Institute. This consortium allowed the Energy and Environmental Policy Center of Harvard University's John F. Kennedy School of Government a free hand in structuring the content of the symposium and proceedings. We thank them for their support and encourage them to continue this process by supporting future research in appropriate fora.

This symposium was about asbestos in buildings. This is an important point because there are those who would wish to extend the findings to occupational settings in which asbestos material is being handled. And there are also those who would like to use the symposium conclusions in reference to EPA's recent planned phase-out of most asbestos containing products. Both of these applications were beyond the scope of this symposium. The symposium focused on human exposures to asbestos fibers in office buildings, schools, and other public access structures. Management of asbestos must consider occupations that come in close physical contact with asbestos containing materials. These groups must be educated and protected.

Occupants in the vast majority of buildings containing undamaged asbestos material also must recognize that the nature of their risk is substantially less than many voluntary and involuntary risks commonly accepted in our modern society. Further, it must be recognized that removal may expose workers to unacceptably high levels of asbestos fibers while not appreciably lowering the risk to occupants, and therefore a range of options for asbestos management should be considered. It is within this context that the Harvard Symposium sought to delineate a rational public health policy.

John D. Spengler
Halûk Özkaynak
John F. McCarthy
Henry Lee

December 1989

TABLE OF CONTENTS

Summary of Symposium on Health Aspects of Exposure to Asbestos in Buildings by John D. Spengler, Halûk Özkaynak, John F. McCarthy, and Henry Lee 1

Session I - Scope of the Problem

Issues Related to the Potential Health Hazard of Asbestos Containimg Materials in Buildings by Morton Corn 31

Epidemiological Observations on Mesothelioma and Their Implications for Non-occupational Exposure to Asbestos by Douglas Liddell 47

Economic Issues in Asbestos Control in Buildings by Donald N. Dewees 71

Session II - Health Studies

Fiber Type and Mesothelioma Risk by Arthur M. Langer and Robert P. Nolan ... 91

Factors Influencing Individual Response to Asbestos: Effects of Smoking and Asbestos on the Development of Lung Cancer by Brooke T. Mossman and Rhonda Gilbert 141

Asbestos Material Inventory, Control Concepts, and Risk Communication by Robert N. Sawyer 155

Session III - Physical Assessment Methods

How Do Current Methods of Measuring Asbestos Exposure Address Risk to the General Population? by Edward T. Peters 171

Assessing Asbestos Exposure Potential in Buildings by Bertram Price 197

Air Sampling and Asbestos-Containing Materials in Buildings by Peter S.J. Lees . 221

Airborne Asbestos Fiber Levels in Buildings and Their Impact on Risk Management by Garry J. Burdett, James J. Smith, and Chris D. Papanicolopoulos 235

Session IV - Risk Assessment

The Derivation and Use of Asbestos Risk Estimates by Janet M. Hughes 267

Fibre Carcinogenesis and Environmental Hazards by Julian Peto 279

Session V - Defining New Directions Group Discussion and Summaries

SUMMARY OF SYMPOSIUM ON HEALTH ASPECTS OF EXPOSURE TO ASBESTOS IN BUILDINGS

JOHN D. SPENGLER[1], HALÛK ÖZKAYNAK[2], JOHN F. McCARTHY[3], HENRY LEE[4]

Symposium held on December 14-16, 1988 at Harvard University, Energy and Environmental Policy Center, John F. Kennedy School of Government, Cambridge, MA

EXECUTIVE SUMMARY

The Energy and Environmental Policy Center of Harvard University's John F. Kennedy School of Government held a three day symposium on the Health Aspects of Asbestos in Buildings on December 14 - 16, 1988. The purpose of the symposium was to assess the health risk that buildings with asbestos containing materials (ACM) present to occupants, facility maintenance personnel, and workers engaged in asbestos removal activities. Internationally recognized experts addressed a series of questions, including: What is the extent of the health risk posed by asbestos in buildings? Who in the population is being exposed? How do we measure the level of exposure and how do we communicate the extent of the associated risk?

New interpretations of published findings on exposures in buildings and of occupational cohort studies were presented at the Harvard symposium. The toxicity of fiber type as well as factors influencing the response of individuals exposed to those fibers was discussed. Mesothelioma and lung cancer risks were projected through extrapolations from epidemiologic studies of workplace environments. The principal findings from the symposium are summarized below.

---

1 Professor of Environmental Health, Harvard School of Public Health, 665 Huntington Avenue, Boston, MA 02115.

2 Research Fellow and Lecturer, Energy and Environmental Policy Center, Harvard University, 65 Winthrop Street, Cambridge, MA 02138.

3 President, Environmental Health and Engineering Inc., 7 Wells Avenue, Newton, MA 02159.

4 Executive Director, Energy and Environmental Policy Center, Harvard University, 79 John F. Kennedy Street, Cambridge, MA 02138.

1. Asbestos is not a single mineral. It is a term used to refer to a number of fibrous inorganic minerals that share specific properties. The most common of these is chrysotile - representing over 95% of all asbestos used in buildings. The second major group is amphiboles - including amosite and crocidolite. The differences in fiber type and dimension can be of significance in determining health risks. For example, the amphibole fibers pose a much greater risk of mesothelioma than chrysotile fibers. Respirable fibers longer than ~5 μm are thought to be of much greater risk than short fibers. Measurements made by EPA indicate that exposures to fibers 5 μm and longer inside buildings are much lower than average airborne concentrations of all fibers.

2. Occupant risk should be determined by exposures to airborne fibers. At the present time there is no single optimum method to characterize potential exposure to airborne fibers from asbestos containing materials in buildings. However, the consensus view was that air sampling, using the direct preparation TEM methodology, is the methodology of choice for current exposure assessment. The available asbestos air monitoring data base for buildings in the U.S. indicates extremely low average concentrations of airborne asbestos under normal building use conditions.

3. The extent of risks associated with exposures to asbestos in contaminated buildings differ for various groups who reside or work in these buildings. Custodial personnel, maintenance and construction workers may be exposed to elevated levels of asbestos for brief periods of time. In contrast, however, most office workers, teachers, students, and other building occupants typically do not come in close contact with asbestos containing materials.

4. There are considerable differences in the value of many of the earlier studies on asbestos health effects. A major, nearly universal deficiency was the differences in measurement techniques as well as uncertainties relating to the amount of asbestos fibers in the workplace environment. For example, a main data source, particle concentration measurements (pre-1960), did not count fibers separately, let alone differentiate them by size, length, or type. Consequently, most studies used to quantify risk were unable to quantify differences in response associated with fiber type and size. In some studies, impurities or the presence of other substances may have affected the risk estimates.

5. Process factors and environmental effects have, potentially, significant impact upon risk. Occupational cohort studies show that relative risk of lung cancer from exposures to amosite or mixed (amphibole and chrysotile) fibers is typically higher than risk associated with processes in which chrysotile is bonded with other materials, as in production of cement or friction products. However, studies of lung cancer among chrysotile textile workers show increases in relative risk higher than those encountered using mixed fiber types (insulation material or cement products).

6. Mesothelioma has been modeled as a risk independent of age. Risk increases in proportion to the product of exposure and as a power of time (typically around 3). For calculating mesothelioma risk, fiber type is believed to be essentially important. The majority of asbestos-related mesotheliomas can be attributed to amphiboles (primarily crocidolite), a much less commonly used variety of asbestos in the U.S. The mesothelioma risk from exposure to chrysotile asbestos, the type of asbestos most commonly found in U.S. schools and buildings, is believed to be considerably lower. Present U.S. regulation does not consider variations in the biological potency of different fiber categories.

7. Lung cancer and mesothelioma risk models have been developed for asbestos exposure that are considered to be conservative (i.e., tend to overpredict rather than underpredict the risks). Recent data indicate that the average concentration of asbestos in schools and other buildings that have asbestos containing materials is generally well below the 0.001 (mixed) fibers/ml used in risk calculations performed for school children. Using these conservative risk models and exposures higher than typically measured, the projected lifetime risk from exposure to mixed asbestos fibers is one death among a cohort of 100,000 children.[1] These risk projections do not differ greatly among different investigators.

---

[1] Assuming 10 year exposure to the mixed asbestos fibers from building insulation material at concentrations of 0.001 (regulatory) fibers/ml, where regulatory fibers are defined to be at least 5 μm in length, with a length-to-diameter ratio of 3 to 1 or greater.

8. The risk of 1 in 100,000 is far less than most other commonly experienced environmental health risks, such as those attributable to environmental tobacco smoke and radon. Even though the risk posed by in-place asbestos is very small, both in absolute and relative terms, the public perception of these risks are high, which often leads to the decision to simply remove all of the asbestos containing materials from buildings that have ACM.

9. Removal itself is not without risk. Removal and disposal operations expose a large fraction of removers to high concentrations of airborne asbestos, and also may potentially increase rather than decrease indoor asbestos concentrations and the associated health risks. Thus, there exists a question whether removing asbestos containing materials now, as opposed to at the time of demolition, will reduce, or even increase, health risks.

10. In many cases, spending money on asbestos removal will likely decrease funding which might be available to support other public and educational measures which could be far more effective in reducing environmental health risks.

11. For buildings with ACM, air sampling and risk calculations should be performed as a component of selection of the most effective management strategy from the point of view of health protection and achieving a net risk reduction. This evaluation should include the risks to both groups: occupants of these buildings; and custodial and maintenance personnel and workers involved in the removal of the asbestos. The potential for exposure to asbestos fibers is much greater for the latter group than the former. Therefore, personal monitoring studies should be initiated to evaluate worker exposure and worker protection procedures under various conditions of asbestos management and abatement.

1. INTRODUCTION

The symposium organized on December 14 - 16, 1988 by the Energy and Environmental Policy Center of Harvard University and jointly co-sponsored by the Center, National Association of Realtors®, the Institute of Real Estate Management Foundation, the Urban Land Institute and the Safe Buildings Alliance was a constructive examination of the scientific knowledge fundamental to environmental asbestos risk management. The symposium identified inconsistencies between current and emerging information on risk of asbestos use in buildings and previous regulatory decisions. This summary provides an overview of the problem and reviews the major findings presented at the symposium. The titles of the papers presented are listed in the Appendix; they will be published in the proceedings of this meeting. Also included in the Appendix is a list of symposium participants.

This summary begins with a section reviewing the nature of the asbestos problem, followed by a synopsis of the various methods available for assessing concentrations and exposures. The third section of the report summarizes the present state of knowledge regarding the human health risks of exposures to asbestos. The fourth section presents a summary of key conclusions. Finally, the references cited in the text are contained at the end of this report.

1.1 The Nature of the Problem

Given the well established and highly publicized association between exposures to asbestos and the diseases of asbestosis, lung cancer or mesothelioma in occupationally exposed populations, "fiber phobia" among the general public is not unreasonable. Knowledge that asbestos related diseases have been documented in populations not working directly with asbestos material but living in adjacent communities or in the household of an asbestos worker, contributes to the fear that asbestos fiber exposure at any level is associated with an unacceptable risk.

Asbestos containing materials (ACM) are present in many homes, offices and school buildings, causing anxiety and concern for the well-being of the occupants. Homeowners, school board members, corporate counselors and CEO's are not expected to differentiate between serpentine and amphibole fibers, or between differential risk for smokers and non-smokers. They are bombarded daily with news of dangers from a myriad of environmental and safety hazards. Radon gas in buildings, tobacco smoke, pesticides in food, and volatile organic compounds in air and water, as well as asbestos fibers are all concerns that must be dealt with by anyone who owns or manages a building. What experience would lead them to appreciate the potential new risk, such as high exposures to asbestos fibers by removal workers? It is not surprising that public and private managers reach the simplest

and liability reducing decision - simply remove the asbestos containing material from their buildings. The correctness of their decisions appears to be bolstered by the Congressional action against asbestos in schools, by courts requiring manufacturers, insurers, installers to pay the cost of removal and by the promotional activities of companies involved with asbestos removal.

Surveys by EPA estimate that 20% of our nation's buildings (733,000 buildings, excluding schools and residential buildings with less than ten units) contain some friable asbestos materials (EPA 1988a). Separate surveys for the City of New York estimate that 67% of the buildings (153,000, excluding schools and one- or two-family homes), contain asbestos (NYC DEP 1988). The estimated cost of removing the asbestos from all buildings is enormous. For example, according to EPA's report to Congress (EPA 1988a), it will cost $51 billion to remove asbestos from all public and commercial buildings.

To address the efficacy of removing asbestos from buildings, we need to reassess whether we are effectively measuring the extent and magnitude of the hazard and whether the risks are large enough for concern - risks not only for the occupants of these buildings, but also for those workers involved in removal of asbestos. Ultimately, we must be confident that removing asbestos fibers either now or prior to demolition will result in a reduction of health risk. In light of scientific evidence published since the National Research Council's 1984 report on asbestos, the experience with current removal practices and the low level of demonstrated asbestos fiber exposures in buildings, and new information on the potential hazards of man-made mineral fibers used as substitute insulation, there is a reasonable conclusion that removal of asbestos may actually increase health risk.

Given this new information, it is appropriate to reappraise the credibility of the scientific data base and assertions made with respect to the health risk of asbestos in buildings. The 100th Congress of the United States was in accord with this appeal when it authorized $2,000,000 to be matched with private contributions for jointly funded asbestos research. Excerpting from the appropriations language....

> "This research is intended to determine actual airborne exposure levels prevalent in buildings, to characterize peak exposure episodes and their significance, and to evaluate the effectiveness of asbestos management and abatement strategies in a scientifically meaningful manner. The conferees believe it is important that the sponsorship of and participation in this effort encompass the full range of private interests, including current and former product manufacturers, realtors, developers, building owners and managers, mortgage bankers, the insurance industry, labor organizations and environmental groups."

1.2 Background on Asbestos and its Use

Asbestos, because of its high tensile strength and thermal properties, has been used in a variety of building materials, including thermal and acoustical insulation, fire protection, and the reinforcement of building products, since the beginning of this century. Asbestos has been used in paints, wall and ceiling plaster as well as in ceiling acoustical and vinyl floor tiles. Until banned during the 1970s, sprayed-on asbestos materials were used to coat pipes, boilers and steel structural beams, structural concrete and the underside of concrete and steel pay decks. Table 1 summarizes some of the important uses of asbestos. Figure 1 indicates the substantial decrease in the U.S. consumption of asbestos since 1973 - a decrease which coincides with EPA's banning certain applications.

It is important to remember that asbestos is not a single mineral. Asbestos is a term used to refer to a number of fibrous inorganic minerals that share specific properties of asbestiform habit, flexibility, high tensile strength, and stability in hostile chemical environments. The serpentine mineral chrysotile is the most commonly used asbestos, representing over 95% of all asbestos used in buildings. Most of the chrysotile asbestos used in the U.S. is imported from Canada. The second major group of asbestos minerals are amphiboles, including amosite and crocidolite. South Africa is a major supplier of these fibers. Used in a number of commercial applications, they have been historically important in terms of worker exposure and disease.

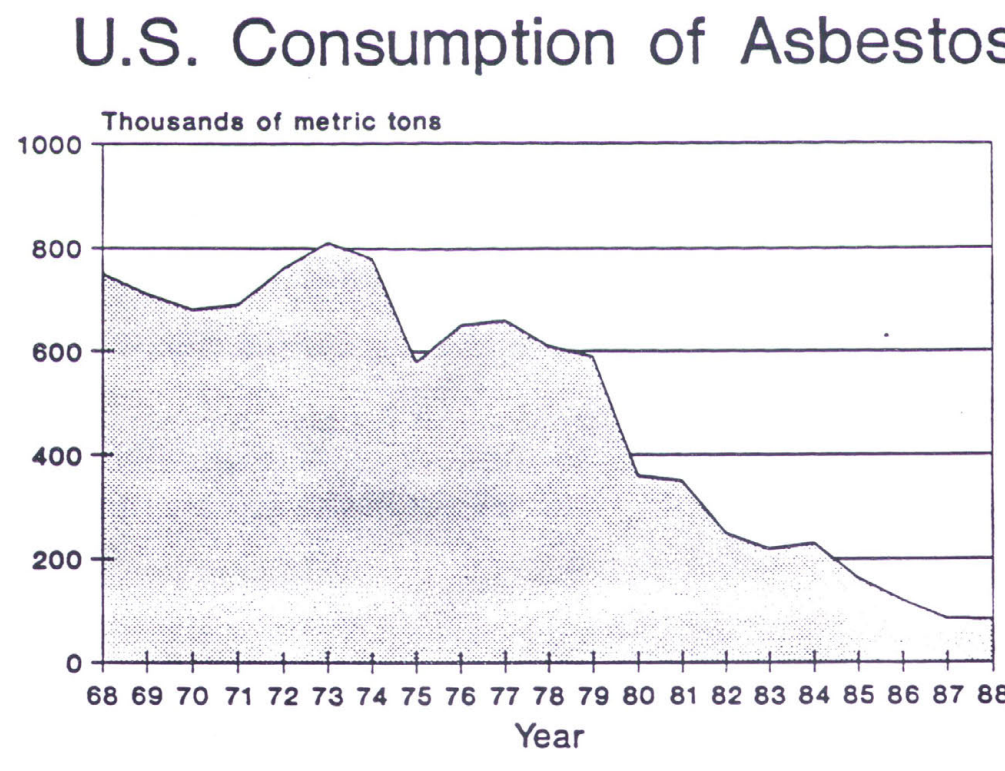
Table 1

SUMMARY OF ASBESTOS-CONTAINING BUILDING PRODUCTS

Product	Dates Used	Average % asbestos
Floor tile and sheet	1950 - present	20
Asphaltic coatings and sealants	1900 - present	10
Cement pipe and sheet	1930 - present	30
Roofing felt	1910 - present	15
Corrugated paper pipe wrap	1910 - present	80
Sprayed insulation	1935 - 1978	50
Trowelled insulation	1935 - 1978	70
Preformed pipe wrap	1926 - 1975	50
Insulation board	unknown - present	30
Boiler insulation	1890 - 1978	10
Other uses	1900 - present	<50

Source: Progressive Builder, September 1986.

Figure 1



Adapted from: Chemical & Engineering News, March 4, 1985

In the mining, milling, fabrication and installation of asbestos, fiber bundles may be manipulated so that individual bundles break open, releasing fibrous particles that are inhalable. However, once asbestos is in place, or in the matrix of a product, fiber bundles are not disrupted without application of external mechanical force, i.e. they do not degenerate spontaneously. This last point is important in evaluating the public health risk to occupants in a building containing asbestos materials. The magnitude, and even existence, of risk will depend on the activities of the occupants. Office workers or school children would have less exposure to fibers under passive circumstances of occupancy than a maintenance worker installing cables in an area where contact with an asbestos-containing material is likely. The population at greatest risk are removal workers who must deliberately disturb the asbestos.

The distinction of different fiber types is also important in evaluating health effects data. Similar to other mineral deposits, asbestos ores may contain a variety of impurities, some of which are source specific. Further, depending on product formulation, different asbestos fiber types might be mixed. There have been many occupational mortality studies of asbestos workers indicating differential response by fiber type. Occupational groups studied include production workers (miners and millers), textile mill workers, insulation installers, cement product workers, structural material fabricators, gas mask fabricators, and car mechanics. These studies have been reviewed in several documents including Omenn et al., 1986; USEPA 1984; CPSC 1983; Ontario Royal Commission 1984; British Health and Safety Commission 1985; NRC 1984 and others. It is concluded that based on these earlier worker studies amphibole fibers (amosite and crocidolite) seem to pose a much greater risk of mesothelioma than chrysotile fibers alone (Langer and Nolan 1988; Liddell 1988).

2. METHODS FOR ASSESSING CONCENTRATIONS AND EXPOSURES

To estimate exposures to asbestos containing materials in buildings, it is necessary to have adequate methods to accurately characterize the airborne fiber concentrations. This characterization ideally should include fiber dimension (length and diameter), concentrations, and fiber type.

EPA regulatory programs rely on visual inspection methods to determine response to ACM, eg. if one sees damaged asbestos materials, one should consider remedial action. There is, however, a lack of correlation between visual assessment methods and fiber concentrations obtained from air samples in spaces with damaged ACM. Participants in the symposium felt that visual inspection does not quantify exposure and is, therefore, not reliable for risk assessment. Since the results of air sampling are directly utilized in risk assessment, future risk assessments should rely more on quantitative air sampling techniques

than the subjective (non-quantitative) method of visual inspection. Discussion of various sampling techniques and their current limitations is summarized below.

2.1 Physical Methods of Measurement and Analysis

At the present time there are two analytical methods typically employed to determine the concentration of airborne asbestos fibers: phase-contrast microscopy (PCM) and transmission electron microscopy (TEM). They vary dramatically in their analytical sensitivity, technical sophistication, and cost.

The method that has been most commonly employed is phase-contrast microscopy (PCM). This method counts fibers longer than 5 μm. According to the National Institute for Occupational Safety and Health (NIOSH), fiber resolution possible with PCM is 0.25 μm diameter under the most ideal conditions. Standard phase-contrast microscopes have a functional resolution limit at about 0.5 μm. Therefore, particles with a smaller diameter will not be visualized, and not included in the fiber counts developed using PCM. This method is unable to differentiate between different types of fibers, including non-asbestos fibers, such as glass fiber, mineral wool and cellulose. Therefore, it lacks both adequate sensitivity and specificity.

With the TEM, the commonly achieved point-to-point resolution is orders of magnitude smaller than obtainable with PCM. Therefore, all fibers may be visualized. TEM can utilize an energy dispersive x-ray spectrometry and selected area electron diffraction to identify the fiber types present. EPA currently requires the use of transmission electron microscopy (TEM) to determine airborne fiber concentrations for area clearance following asbestos material removal.

There are currently two protocols used to prepare an air sample for TEM analysis: the direct method and the indirect method. Each has its own preparation procedure, either of which can result in variability (Chopra 1978). The direct analysis method retains particles on a filter and preserves the pattern of deposition for visualization and characterization. This, of course, should best represent the particles in the airborne state, and should best characterize potential biological effect. However, there are several limitations of this methodology. For instance, if filter loadings are high, it may be difficult or even impossible to visualize, identify and count individual fibers. Also, entire filter surfaces are never completely scanned. Rather, a series of grid pore openings or "fields" are examined. Fiber concentrations are based on statistical analysis of a small portion of the total filter. Unfortunately, non-uniform deposition of fibers on the TEM sample is typical in the direct methodology.

The indirect preparation method suspends the collected particulate matter in liquid and after agitation, refilters it. This filtration should result in a more uniform deposition

of particulates and can be diluted to provide a suitable particle distribution, i.e., no particle overlap. On the other hand, it also has been shown to break up fiber bundles thereby modifying both size and fiber number. This greatly improves counting capability with titration, enhances the statistical reliability, and facilitates identification of individual fibers, but also changes the population size distribution and can increase fiber counts. Thus, with this technique probable biological relevance is greatly reduced.

Because of the introduction of size-number artifacts when using the indirect method, an earlier practice (1960s to early 1970s) was to grind the sample to break down chrysotile fibers into individual fibrils, all assumed to have a uniform diameter. By measuring total fiber length and applying a density factor, fiber mass per volume of air rather than fibers per volume of air was reported. However, there is no systematic or universal transformation to equate mass to fiber concentrations. To date, there is no method or procedure that will allow the results obtained by PCM, TEM direct, or TEM indirect to be compared on a uniform basis.

The ability to accurately quantify the low fiber concentrations in buildings is critical to the assessment of risk. It is clear that occupational studies using PCM methods found fiber concentrations several orders of magnitude higher than currently measured in schools and other buildings. PCM methodology has been practical for occupational settings but is less useful for assessing low level and nonhomogeneous fiber concentrations in buildings. PCM cannot differentiate among fiber types which is important to identifying risks from asbestos fiber exposures in buildings where asbestiform, natural, and synthetic fibers can coexist. For the purposes of exposure and risk assessment, TEM method, incorporating analytical capabilities, should be used as the primary method for air sampling in buildings.

2.2 Building Assessment Methods

The Symposium focused on several issues regarding building assessment methods. Discussions on the 1984 EPA Report entitled "Asbestos in Buildings: A National Survey of Asbestos-Containing Friable Materials" centered on the reanalysis of data regarding the type and condition of asbestos found in the buildings sampled. Further discussions involved EPA's 1988 air monitoring study "Assessing Asbestos Exposure in Public Buildings" which reviewed the relationship between buildings that have asbestos-containing materials in them and the concentration of airborne asbestos fibers found there.

Fundamentally, risk to human health cannot be attributed to just the presence of asbestos-containing material in a building. For a risk to exist, respirable asbestos fibers must be present in the airborne state and be inhaled by building occupants. There are two major methods that are employed in attempting to assess the asbestos exposure potential to building occupants: (1) the simple identification of ACM in buildings by visual

inspection to evaluate the condition of ACM and to assess its potential for disturbance; and (2) air monitoring techniques. The relative merits of these two exposure assessment techniques is currently being debated. However, the Symposium participants agreed on several points that are elaborated below.

Visual inspections evaluate asbestos containing materials by classifying the types found, note their location, accessibility, amount, condition, percentage of asbestos and attempt to determine the potential level of disturbance. Bulk samples of suspect material are collected and analyzed in the laboratory by polarized light microscopy (PLM) to determine if the materials actually contain asbestos. The results from these visual inspections are then incorporated in a summary index which is used as an indicator of exposure potential. Recent studies report the use of visual inspection. These studies include the U.S. Environmental Protection Agency's 1988 report to Congress on ACM in public buildings, and the City of New York's Department of Environmental Protection's study of in-place asbestos (NYC DEP 1988).

Participants felt that visual inspection methods are an efficient way to inventory ACM in buildings. However, several limitations of the ratings used for survey data were discussed. Concern was expressed for the ability of inspectors to consistently and accurately assess the condition of ACM. It is not clear that the potential disturbance of ACM can be consistently determined.

Perhaps more important with regard to assessing exposure potential was the discussion of the relationship between asbestos survey findings and airborne asbestos levels in buildings. The 1988 EPA report (EPA 1988b) stated that the differences among airborne asbestos levels in buildings with ACM deemed to be in "good" or "bad" condition, buildings containing no ACM, and outside air, were not significant. These data, which are summarized in Table 2, support the position held by most of the attendees that the use of these "ratings" to assess the risk to occupant health are a poor surrogate for actual air monitoring.

Measurement of airborne asbestos levels have also been criticized for their inability to reliably represent past or future contamination levels, and that an air sample could be a "snapshot in time" or may not be taken in an area where contamination exists.

The use of various "passive" sampling methods was also discussed. These methods rely on collection and analysis of settled dust from horizontal surfaces as an indicator of previous releases. Limitations of these methods have to do with unknown collection/transfer efficiencies as well as the uncertainty in knowing how long the dust has been allowed to accumulate. These methods are generally only used to obtain relative differences between areas in a building and not useful for quantitative risk assessment.

Therefore, at the present time there is no single optimum method to characterize potential exposure to airborne fibers from ACM in buildings. Projections of future

(potential) exposures should include judicious interpretation of many sources of data. However, the consensus view was that air sampling in buildings should be used to enhance exposure assessment and risk estimation.

Table 2

COMPARISON OF OUTDOOR AND INDOOR ASBESTOS CONCENTRATIONS (structures/ml)

		Inside Buildings		
	Outdoor	No ACM	"Good" ACM	"Bad" ACM
MEAN	0.00039	0.00099	0.00059	0.00073
MEDIAN	<0.00001	0.00010	0.00040	0.00058
N	48 (sites)	6 (bldgs)	6 (bldgs)	37 (bldgs)

Note: Concentrations are building averages - 7 samples per building

Source: EPA 1988b

2.3 Measurement and Exposure Issues in Buildings

The asbestos air monitoring data base for buildings, which has grown significantly during the past two years, indicates extremely low average levels of airborne asbestos under normal building use conditions. Early TEM measurements of airborne asbestos levels (EPA, 1975; Nicholson, 1978; EPA, 1980; EPA, 1983), which were large enough in EPA's judgement to justify initiation of an asbestos regulatory program, were obtained using the indirect filter preparation method. These measurements, therefore, were reported in mass units (nanograms per cubic meter of air). Exposure measured in mass units, however, does not reflect health risk as directly as exposure measured in fibers per milliliter of air

(fibers/ml). More recent data, obtained using the direct preparation TEM methodology and generating data on fibers/ml, indicate lower average exposure levels than were indicated by the earlier indirect methodology studies.

EPA's recent report to Congress concerning asbestos in public and commercial buildings (EPA, 1988a) provides a summary of TEM data utilizing the direct filter preparation method for 94 non-school buildings and 41 school buildings. The average airborne asbestos concentration in non-school buildings was reported to be 0.006 fibers/ml. The average for the studied school buildings was reported to be 0.03 fibers/ml. Since these data were compiled from a number of different air monitoring studies with different degrees of documentation, the averages should be interpreted as representing concentrations for all asbestos structures visible with TEM (i.e., fibers both longer and shorter than 5 microns and other asbestos fibers that may not be considered respirable).

In a different study, detailed documentation, however, is available for the 43 non-school buildings with ACM which were studied by EPA and 73 schools which were studied by asbestos product manufacturers where asbestos removal was imminent (EPA, 1988b; Price, 1988). In the EPA study, the average airborne asbestos concentration when all structures are counted was 0.00070 structures/ml. Of the 387 samples collected in this study, including 48 outdoor samples and samples in 6 buildings with no friable ACM, 83 percent yielded no measurable asbestos fiber counts. Restricting the asbestos count to only those fibers 5 microns in length or longer resulted in an average concentration level approximately 8 times lower, 0.00008 fibers/ml. In the 73 school buildings, the average airborne concentration counting all asbestos structures was 0.01773 structures/ml. Restricted to fibers 5 microns or longer, the average concentration was 0.00022 fibers/ml. The average of measurements taken outdoors in these studies is comparable to the measured indoor level. In general, these results demonstrate that airborne asbestos levels inside buildings are low and suggest that in many buildings the difference between prevalent indoor and outdoor asbestos fiber concentrations is not significant from a public health perspective.

As stated above, occupant risk should be determined by exposures to airborne fibers rather than the presence of asbestos-containing materials in the building. At present there is insufficient information to correlate the relationship between potential exposure and actual indoor concentrations of airborne asbestos fibers. This is due, in part, to the fact that only a few studies have been conducted to examine this relationship. Also, the very nature of periodic or episodic fiber releases makes it difficult to detect a relationship in short-term surveillance or cross-sectional monitoring studies. As a result, the distribution of concentrations both within and across buildings has been not fully characterized. This is primarily a concern for those occupationally exposed, i.e., those who disturb the ACM.

By definition, personal exposures are determined by the fiber concentrations in the immediate vicinity of the breathing zone. Fixed location monitoring within a structure only provides an estimate of what exposures might be for occupants of that building. For example, the actual exposure in the breathing zone of a facility maintenance employee replacing ceiling panels might be quite different than the concentrations monitored at some other fixed location. When considering exposure, it must be recognized that occupants will have different potentials for both short-term and long-term exposures. It is important to differentiate these exposure grouping for both control strategies and risk assessment.

The occupants of buildings can generally be categorized in two groups in respect to potential exposure to asbestos containing materials. The first, and much more numerous group includes occupants who do not come in close contact with asbestos-containing materials. Most office workers, teachers, and students would be in this first category. The second, and smaller, group includes those whose work potentially places them in close physical contact with asbestos material. Several job categories would be included in this group, such as custodial, maintenance, and construction personnel, telephone and computer installers, and ventilation system workers. This distinction is quite important to exposure risk assessment. The lower fiber counts reported in building surveys to date are a better indication of potential exposure for the first, larger occupant group. These lower fiber counts indicate for this group that risk is low and that the frequency of episodic releases are also low.

Similar conclusions cannot be inferred for the occupations that come in close physical contact with ACM. Exposures encountered by these occupational groups can be characterized, but with difficulty (Sawyer 1977). The exposure data of interest is really the cumulative exposure to asbestos fibers over a time period of months to years. In order to understand the factors that contribute to exposure, it may be important to examine short-term episodic conditions for worker populations. Information on the short-term exposure profiles can be used to develop worker protection procedures and to formulate specific removal or containment programs. The only way to adequately address this issue is to conduct personal monitoring during tasks representative of work procedures involving a variety of conditions and materials (Sawyer 1979). Workers' exposures must be monitored while they engage in the routine and unusual activities that have the potential to generate airborne asbestos fibers.

Personal monitoring of asbestos fiber exposures for members of the group that do not come in direct physical contact with ACM is also needed. This effort would demonstrate the relationship between fixed location area monitoring and personal exposures. Issues that need to be resolved concern the potential that asbestos removal might actually increase exposure to this group of building occupants. For example, the currently required (AHERA) monitoring for reoccupancy may not represent fiber

concentrations that would result from resuspension of settled fibers. Aggressive sampling in post-abatement areas evaluated by the EPA has shown fiber levels to increase 3 to 6 times over preabatement levels (Karaffa et al., 1987). Burdett et al., (1988, 1989) found fiber levels increasing 10 to 100 times after incomplete dry removal. Further, the comparison to ambient fiber concentrations as the criteria for reoccupancy does not address the change in exposures to occupants before and after removal. It is the net change in estimated lifetime exposures, before and after asbestos removal, that will determine the effectiveness of abatement strategies.

3. ASSESSING HEALTH RISKS

Essentially, all of our current information on health hazards associated with the inhalation of asbestos fibers comes from studies of diseases among workers exposed to asbestos in various occupational settings. Both occupational cohort studies and animal experiments indicate that three forms of disease can be associated with inhalation of asbestos fibers: (1) asbestosis-fibrosis or scarring of the lung, (2) lung cancer, and (3) mesothelioma - a malignancy of the pleura and peritoneum, linings of the lung and abdomen.

Deaths from asbestosis are observed only among individuals that have been occupationally exposed to high levels of asbestos (Nicholson 1986). Present levels of asbestos in workplaces utilizing current control procedures are not expected to produce any measurable impairment (Berry et al., 1980). Further, if asbestosis occurs, due to recent and much lower levels of asbestos exposure in workplaces, it has a slow progression and rarely results in serious disability or increased mortality (Jones et al., 1989). Consequently, there does not seem to be any evidence that asbestosis is a concern as a result of environmental exposures.

The papers presented at the Harvard Symposium by Drs. D. Liddell, J. Hughes and J. Peto summarized the occupational data base and risk models used in projecting lung cancer and mesothelioma risks for environmental exposures to asbestos. Uncertainties surrounding the exposure variables and the estimated dose-response functions, used in the development of risk models for asbestos, were also addressed by these presenters. Using conservative (pessimistic) assumptions, mesothelioma and lung cancer risk projections from exposures to indoor asbestos for school age children and the general population was generally agreed by all of the symposium participants to be quite small. For example, associated lifetime risk of death is estimated to be around 1 in 100,000 for 10 years of occupancy in contaminated buildings with 0.001 regulatory fibers/ml of mixed fiber types. Researchers attending the meeting attempted to put these uncertainties and risks in perspective by comparison with model predictions of risks to school children and building

occupants from other environmental hazards. This comparison of asbestos risk to other life risks was considered essential to provide understanding of the significance of this level of exposure. The principal findings from the symposium regarding these risk issues are summarized below. We also note that many of the issues discussed in this section were previously addressed and summarized in Hughes and Weill (1986).

3.1 Sources of Exposure Data and Uncertainties in the Dose-Response Models

The epidemiologic studies of asbestos health effects are based on occupational exposures to asbestos which occurred many years ago. These studies cannot be considered without recognizing the fact that these occupational exposures were much greater than those found inside buildings today, and were measured using techniques less appropriate to risk estimation. Based on a method developed for measuring exposure to respirable silica dust, particle measurements, instead of fiber counts, were made. For subsequent studies, the particle concentrations were converted to fiber concentrations, usually on the basis of relatively few comparative measurements in occupational settings. Unfortunately, there are many inconsistencies in this process. According to Peto (1989), no single or consistent relationship exists between particle counts taken in the past with different instruments, or in conversion of particles to fiber concentration. For many occupational cohorts there were also very few exposure data in areas of heavy contamination (Peto 1989).

Animal experiments and physiologic evaluations suggest that asbestos fibers of different length and diameter will have different potencies. According to Lippmann (1988), mesothelioma is most closely associated with fibers longer than ~5 μm and thinner than ~0.1 μm, and that lung cancer is most closely associated with fibers longer than ~10 μm (the approximate diameter of an alveolar macrophage) and thicker than ~0.15 μm. However, these experimental observations have not been fully corroborated with effects observed with humans.

There is general agreement that crocidolite and amosite asbestos pose much greater risk of mesothelioma than chrysotile (Liddell 1988; Hughes and Weill 1986; Langer and Nolan 1988). Most commercial asbestos (i.e. over 95%) used in the United States has been Canadian chrysotile. Therefore, the application of mixed fiber risk models may be conservative. This is an important point for public policy, because present U.S. regulations do not consider variations in the biological potency of different fiber categories (Corn 1988).

Establishing a relationship between estimated dose and observed effects, such as lung cancer or mesothelioma, requires formulation and application of suitable statistical models. According to Peto (1988), the majority of the dose-response relationships reported for lung cancer and mesothelioma have been based on two assumptions: (1) the increase

in relative risk for lung cancer is proportional to cumulative asbestos exposure and constant over time, and the effects of asbestos and cigarette smoking are multiplicative; (2) the increase in mesothelioma incidence caused by asbestos exposure is approximately proportional to the product of the mean asbestos concentration to the 3rd power of time since it occurred, independent of age or smoking history at initial exposure (Peto et al., 1982).

The assumptions of the asbestos lung cancer model, that the risk is linearly related to both concentration and duration of exposure, and relative risk (observed/expected) is constant over time are based on analyses of data with certain limitations (Hughes and Weill 1986). Many experts believe that the utilized risk models are conservative, i.e. most likely to lead to overprediction of risks rather than an underprediction.

The mesothelioma risk model is based on more assumptions than the lung cancer model and relies on fewer quantitative data (Hughes and Weill 1986). Although the model fits the available data sets over a certain time period following first exposure, there is a possibility that the mesothelioma risks after many years (50+) does not rise as steeply as predicted by the model (Hughes and Weill 1986). If this is in fact the case, the current models could considerably overestimate lifetime mesothelioma risks.

In summary, it is difficult to establish the reliability of these risk models in predicting incidence of lung cancer or mesothelioma from environmental exposures to asbestos. The main reason for this difficulty is the frequently encountered "low dose extrapolation" problem, i.e. when models that are developed using high industrial asbestos exposures experienced in the past are used to predict population risks at the current low levels (cf. Zeise et al., 1987). In spite of these difficulties the existing asbestos risk models seem to produce risk projections consistent with the observed background mesothelioma rates of about 2 per million persons (McDonald 1985).

It is important to note that even with the statistical uncertainties in the estimated cancer or mesothelioma risks, asbestos risk estimates reported by various researchers are in close agreement. The summary of these risk estimates presented in Hughes and Weill (1986) suggests that the six commonly reported lung cancer and mesothelioma risk estimates, are typically within a factor of 3 of the mean estimate (i.e. three times higher or lower than the average of all the risk estimates). This finding indicates that different investigators have adopted similar methods but may have used rather different model parameters or data sets to predict asbestos health risks.

3.2 Projected Asbestos Risks and Their Significance

Using the risk models developed it is possible to estimate the magnitude of cancer and mesothelioma risks that may be posed by asbestos in contaminated buildings or schools. Recent data, mostly collected and reported by EPA contractors, suggest that the average level of asbestos in schools and other buildings that have asbestos containing materials, is generally well below the 0.001 fibers/ml (Corn 1988; Price 1988; EPA 1988b) used in the risk calculations performed for school children (Peto 1988 and Hughes 1988). Using these models, the prediction of total additional lifetime risk of death from asbestos related lung cancer or mesothelioma is 1 in 100,000 or less. It is important to note that these are individual probabilities of death and not actual deaths. Furthermore, these deaths, if they ever occur, will not occur until much later in life. Although these predictions cannot be tested directly, it is shown that these estimates are again consistent with the fairly stable background risk of mesothelioma for population groups with no history of asbestos exposure.

These risks may be put in perspective by comparing the projected asbestos risks to other known risks. First of all, a risk of 10 lifetime excess deaths among one million school age children exposed to 0.001 (mixed) fibers/ml for 10 years is a small fraction of expected deaths from non-asbestos related causes, in this same cohort of children. According to Hughes and Weill (1986), using recent U.S. data, out of this population of 1 million students, approximately 32,000 would be expected to die from lung cancer (90% of those deaths would result from active smoking). As shown in Table 3, the lifetime risk of 1 premature death in 100,000 associated with asbestos exposures is also small compared to other environmental risks. For example, lifetime risk of premature death due to indoor environmental tobacco smoke (i.e. living with a smoker) or living in a house that has radon is two to four hundred times greater than the projected risks of dying from exposures to asbestos at concentrations stated above. For most urban areas, such as Los Angeles, and industrialized communities in New Jersey and West Virginia, lifetime cancer risk from ambient air toxics are at least ten times greater than the estimated 1 in 100,000 asbestos risks (Sullivan et al., 1989).

Table 3

PUBLISHED ESTIMATES OF RISK FROM VARIOUS CAUSES (MAINLY U.S. DATA)*

Cause	Voluntary (V) or Involuntary (I)	Lifetime Risk of Premature Death (per 100,000)
Smoking (all causes)	V	21,900
Smoking (cancer only)	V	8,800
Motor Vehicle	I	1,600
Frequent Airline Passenger	V/I	730
Coal Mining Accidents	I/V	441
Indoor Radon	V/I	400
Motor Vehicle - Pedestrian	I	290
Environmental Tobacco Smoke/Living with a Smoker	I/V	200
Diagnostic X-rays	I	75
Cycling Deaths	I/V	75
Consuming Miami or New Orleans Drinking Water	I	7
Lightning	I	3
Hurricanes	I	3
Asbestos in School Buildings	I	1

* Sources of Risk Estimates: Commins (1985), Weill and Hughes (1986), Wilson and Crouch (1982)

As discussed previously in Section 2.3, the extent of risks associated with exposures to asbestos in contaminated buildings differ for various groups who reside or work in these buildings. Custodial personnel, maintenance and construction workers may be exposed to elevated levels of asbestos for brief periods of time under episodic conditions. However, it is often difficult to estimate or measure the magnitude of these brief but potentially high levels of asbestos exposures of these individuals. In contrast, most office workers, teachers, and other building occupants typically do not come in close contact with asbestos containing material. The low airborne concentrations of asbestos commonly found in buildings with ACM are often indicative or representative of the typical exposures to asbestos by the

occupants of these buildings. Keeping these distinctions of potential exposures in mind, government and individuals managing these risks have to decide when to best remove friable asbestos in buildings, and under what controls.

Removal itself is not without risk. Studies of asbestos fiber levels in the United Kingdom (Burdett et al., 1989) have shown that most containment methods used during asbestos removal are not efficient enough to prevent leakage. These studies have also shown that post-removal residual asbestos can continue to pose risks to the building occupants, depending on the amount of remaining asbestos, the time of re-occupation, the ventilation, and the degree of disturbance (Burdett et al., 1989). Moreover, the environment surrounding a building going through asbestos removal can also become contaminated due to spillage of bulk material. Recent data reported in the United States also indicate that removal of asbestos containing building materials frequently creates higher concentrations of indoor asbestos levels during and after removal, than before (Burdett et al., 1989; Corn 1988; Hughes and Weill 1986; Burdett et al., 1988; and Sawyer 1988).

In terms of an effective risk management strategy it is clear that asbestos removal actions should be evaluated seriously, considering the fact that likely risk from exposure to asbestos present in most buildings is very small, both in absolute and relative terms. Case-specific measurements and risk calculations should ideally be used to guide the decisions on whether and when to remove asbestos materials.

4. CONCLUSIONS

The Harvard Symposium considered new evidence on a wide scope of subjects. This evidence included the appraisal of information on exposures both in buildings as well as occupational cohort studies. The toxicity of fiber type as well as factors influencing individual response were discussed. Risks to building occupants were projected for mesothelioma and lung cancer based on extrapolations from epidemiologic studies.

Using the conservative lung cancer and mesothelioma risk models, a lifetime risk of one death among a cohort of 100,000 children, exposed to 0.001 (mixed) fibers/ml for 10 years, was projected. However, adults who occupy buildings with ACM and are exposed to similar concentrations of asbestos will be at a much lower risk because of age dependence of the risk models used. Comparison of the projected asbestos risks to other commonly experienced environmental health risks, such as environmental tobacco smoke and radon, revealed that most of these risks are far greater than those posed by exposures to asbestos in buildings. In some cases, asbestos removal and disposal operations are shown to increase indoor asbestos concentrations, and thus the associated health risks, as compared to concentrations prior to removal. Issues relevant to assessing the risk of asbestos containing materials in buildings are:

- types of asbestos found in these settings
- actual fiber counts in schools and other buildings
- risk associated with these exposures
- significance relative to other life risks
- more cost-effective risk mitigation strategies
- when to optimally remove asbestos
- potential health risk due to removal
- risks of substitute man-made mineral fibers

Factors were also identified that could influence the direction of asbestos control policies in the U.S. The EPA promulgation of regulations and guidance makes it extremely difficult to modify established positions in light of more recent evidence. Consequently, EPA may find it difficult to incorporate newer concepts in the formulation of future asbestos policies. Another factor is the presence of an organized asbestos removal industry with a collective self-interest to promote additional removal.

At this time EPA should incorporate recent findings of low asbestos exposure and associated minimal health risk in buildings with ACM in the formulation of new asbestos policies. The U.S. must also formulate an asbestos risk management policy consistent with the latest risk assessment evidence. Otherwise there will be a mismatch between the risk of individual environmental contaminants and the allocation of resources to reduce them. In many cases, spending money on asbestos removal will likely decrease funding which might be available to support other public health and educational measures which could be far more effective in reducing environmental health risks.

ACKNOWLEDGEMENTS

We are grateful to co-sponsors of the symposium (the National Association of Realtors®, the Institute of Real Estate Management Foundation, Urban Land Institute and the Safe Buildings Alliance) and the many scientists and experts who facilitated a highly successful meeting on this important topic of asbestos health effects. We are especially grateful to the participants of the meeting for their review comments and technical feedback on the draft summary reports. In particular, we acknowledge the contributions of Drs. Robert Sawyer (Entek, Inc.), Douglas Liddell (McGill University), Peter Lees (Johns Hopkins University), Arthur Langer (Brooklyn College of the City University of New York), Bertram Price (Price Associates), Janet Hughes (Tulane University), Edward T. Peters (Arthur D. Little, Inc.), Donald N. Dewees (University of Toronto), Morton Corn (Johns Hopkins University), and Brooke Mossman (University of Vermont) to portions of the summary report. However, the authors of this report share the full responsibility for the content of this summary and the views represented in the report. Finally, we would like to express our appreciation to Stephanie Alpert for her support in the coordination of the conference, and to Janet Garrison for her diligent efforts during the organization of the meeting and the production of the symposium summary.

REFERENCES

Berry, G. et al., "Asbestosis: A Study of Dose-Response Relationships in an Asbestos Textile Factory", British Journal of Industrial Medicine, 36:98-112, 1980.

British Health and Safety Commission. Health and Safety Executive, Asbestos: "Effects on Health of Exposure to Asbestos", Health and Safety Commission, HMSO, London. Guidance note EH35.HMSO, 1985.

Burdett, G.J., S.A.M.T Jaffery, and A.P. Rood, "Airborne Asbestos Fiber Levels in Buildings. A Summary of UK Measurements", Non-occupational Exposure to Mineral Fibres, edited by J. Bignon, J. Peto and R. Saracci, International Agency for Research on Cancer Scientific Publication No. 90, 1989.

Burdett, G.J., J.J. Smith, V.H. Ainslie, and C.D. Papanicolopoulos, "Airborne Asbestos Fiber Levels in Buildings and Their Impact on Risk Management", Paper presented at the Symposium on Health Effects of Exposure to Asbestos in Buildings, Harvard University, December 14-16, 1988.

Chemical & Engineering News. Zurer, P.S., "Asbestos: The Fiber That's Panicking America", Chemical & Engineering News, Vol. 63, No. 9, pp. 28-41, March 4, 1985.

Chopra, K.S., "Interlaboratory Measurements of Amphibole and Chrysotile Fiber Concentration in Water", Journal of Testing and Evaluation, Vol. 6, No. 4, pp. 241-247, July 1978.

CPSC. Report to the U.S. Consumer Product Safety Commission by the Chronic Hazard Advisory Panel on Asbestos, United States Consumer Product Safety Commission Directorate for Health Sciences, Washington, D.C., 1983.

Commins, B.T., The Significance of Asbestos and Other Mineral Fibers in the Environment. Commins Associates, Pippins, Altwood Close, Maidenhead, Berks Sl6 4PP, England, 1985.

Corn, M. "Issues Related to the Potential Health Hazard of Asbestos Containing Materials in Buildings", Paper presented at the Symposium on Health Effects of Exposure to Asbestos in Buildings, Harvard University, December 14-16, 1988.

EPA. Asbestos Contamination of the Air in Public Buildings, United States Environmental Protection Agency, Research Triangle Park, North Carolina, EPA-450/3-76-004, October 1975.

EPA. Measurement of Asbestos Air Pollution Inside Buildings Sprayed with Asbestos, United States Environmental Protection Agency, Washington, D.C., EPA-560/13-80-026, August 1980.

EPA. Airborne Asbestos Levels in Schools, United States Environmental Protection Agency, Washington, D.C., EPA-560/5-81-006, July 1983.

EPA. Asbestos in buildings: A National Survey of Asbestos-containing Friable Materials, United States Environmental Protection Agency, Washington, D.C., Office of Toxic Substances, EPA 560/5-84-006, 1984.

EPA. Study of Asbestos-Containing Materials in Public Buildings: A Report to Congress, United States Environmental Protection Agency, Washington, D.C., February 1988a.

EPA. Assessing Asbestos Exposure in Public Buildings, United States Environmental Protection Agency, Washington, D.C., EPA 560/5-88-002, May 1988b.

Hughes, J.M., "The Derivation and Use of Asbestos Risk Estimates", Paper presented at the Symposium on Health Effects of Exposure to Asbestos in Buildings, Harvard University, December 14-16, 1988.

Hughes, J.M. and H. Weill, "Asbestos Exposure - Quantitative Assessment of Risk", American Review of Respiratory Diseases, 1986.

Jones, R.N., J.E. Diem, J.M. Hughes, Y.Y. Hammad, H.W. Glindmeyer and H. Weill, "Progression of Asbestos Effects: A Prospective Longitudinal Study of Chest Radiographs and Lung Function", British Journal of Industrial Medicine, 46:97-105, 1989.

Karaffa, Mark A., C.E. Miller, R.A. Amick, J. Chesson, and C. Jackson, "Preliminary Evaluation of Potential Asbestos Exposure During Carpet Vacuuming in Buildings Containing Friable Asbestos Material", U.S. Environmental Protection Agency, Unpublished Draft Report, Cincinnati, Ohio. November 1987.

Langer, A.M. and R.P. Nolan, "Fiber Type and Mesothelioma Risk", Paper presented at the Symposium on Health Effects of Exposure to Asbestos in Buildings, Harvard University, December 14-16, 1988.

Liddell, D., "Epidemiological Observations on Mesothelioma and Their Implications for Non-Occupational Exposure to Asbestos", Paper presented at the Symposium on Health Effects of Exposure to Asbestos in Buildings, Harvard University, December 14-16, 1988.

Lippmann, M., "Review: Asbestos Exposure Indices", Environmental Research, 46:86-106, 1988.

McDonald, J.C., "Health Implications of Environmental Exposure to Asbestos", Environ. Health Perspective, 62:319-328, 1985.

National Research Council, Committee on Non-occupational Health Risks of Asbestiform Fibers. Asbestiform Fibers: Non-occupational Health Risks, National Academy Press, Washington, DC, 1984.

Nicholson, W.J., A.N. Rohl, R.N. Sawyer, E.J. Swoszowski, and J.D. Todaro, "Control of Sprayed Asbestos Surfaces in School Buildings: A Feasibility Study", Report to the National Institute of Environmental Health Sciences, June 1978.

Nicholson, W.J., "Airborne Asbestos Health Assessment Update", United States Environmental Protection Agency Office of Health and Environmental Assessment, Washington, D.C. EPA/600/8-84/003F, 1986.

NYC DEP. Final Report on the Assessment of the Public's Risk of Exposure to In-Place Asbestos. City of New York Department of Environmental Protection, New York, New York, 1988.

Omenn, G.S., J. Merchant, E. Boatman, J.M. Dement, M. Kuschner, W. Nicholson, J. Peto, and L. Rosenstock, "Contribution of Environmental Fibers to Respiratory Cancer", Environmental Health Perspective, 70:51-56, 1986.

Ontario Royal Commission. Report of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario: v. 1-3. Toronto, Ont., Canada: Ontario Ministry of the Attorney General, 1984.

Peto, J., "Fibre Carcinogenesis and Environmental Hazards", Paper presented at the Symposium on Health Effects of Exposure to Asbestos in Buildings, Harvard University, December 14-16, 1988. (Reprinted with permission from Non-occupational Exposure to Mineral Fibres, edited by J. Bignon, J. Peto and R. Saracci, International Agency for Research on Cancer Scientific Publication No. 90, 1989).

Peto, J., "Fibre Carcinogenesis and Environmental Hazards", Non-occupational Exposure to Mineral Fibres, edited by J. Bignon, J. Peto and R. Saracci, International Agency for Research on Cancer Scientific Publication No. 90, 1989.

Peto, J., H. Seidman and I.J. Selikoff, "Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment", Br. J. Cancer, 45: 124-135, 1982.

Price, B., "Assessing Asbestos Exposure Potential in Buildings", Paper presented at the Symposium on Health Effects of Exposure to Asbestos in Buildings, Harvard University, December 14-16, 1988.

Progressive Builder. Oppenheim-McMullen, J. and B. Turner, "Facing the Asbestos Menace", Progressive Builder, pp. 21-22, September 1986.

Sawyer, R., "Asbestos Exposure in a Yale Building: Analysis and Resolution", Envir. Res., 13:1146-168, 1977.

Sawyer, R., "Indoor Asbestos Pollution: Application of Hazard Criteria", Ann. N.Y. Acad. Sci., 330:579-586, 1979.

Sawyer, R., "Asbestos Material Inventory, Control Concepts, and Risk Communication", Paper presented at the Symposium on Health Effects of Exposure to Asbestos in Buildings", Harvard University, December 14-16, 1988.

Sullivan, N., H. Özkaynak, J.H. Ware, M.A. Cohen, P.B. Ryan, and J.D. Spengler, "Report on Ambient Exposures to Volatile Organic Compounds in the Kanawha Valley", Report prepared for the National Institute for Chemical Studies and the United States Environmental Protection Agency, January 9, 1989.

Weill, H. and J.M. Hughes, "Asbestos as a Public Health Risk: Disease and Policy", Annual Review of Public Health, 7:171-92, 1986.

Wilson, R. and E. Crouch, Risk/Benefit Analysis. Ballinger Publishing Company, Cambridge, Massachusetts, 1982.

Zeise, L., R. Wilson, and E.A.C. Crouch, "Dose-response Relationships for Carcinogens: A Review", Environ. Health Perspective, 72:259-308, 1987.

APPENDIX
LIST OF SPEAKERS AND PAPERS PRESENTED

Session I - Scope of the Problem

Morton Corn — Johns Hopkins University, Baltimore, Maryland

Issues Related to the Potential Health Hazard of Asbestos Containing Materials in Buildings

Douglas Liddell — McGill University, Montreal, Quebec, Canada

Epidemiological Observations on Mesothelioma and Their Implications for Non-Occupational Exposure to Asbestos

Donald N. Dewess — University of Toronto, Toronto, Ontario, Canada

Economic Issues in Asbestos Control in Buildings

Session II - Health Studies

Arthur M. Langer and Robert P. Nolan — Brooklyn College, City University of New York

Fiber Type and Mesothelioma Risk

Brooke T. Mossman and Rhonda Gilbert — University of Vermont, Burlington, Vermont

Factors Influencing Individual Response to Asbestos: Effects of Smoking and Asbestos on the Development of Lung Cancer

Robert Sawyer — Entek, Inc., Troy, New York

Asbesotos Material Inventory, Control Concepts, and Risk Communication

Session III - Physical Assessment Methods

Edward Peters — Arthur D. Little, Inc., Cambridge, Massachusetts

How Do Current Methods of Measuring Asbestos Exposure Address Risk to the General Population?

Bertram Price — Price Associates, Inc., Washington, D.C.

Assessing Asbestos Exposure Potential in Buildings

Peter S. J. Lees — Johns Hopkins University, Baltimore, Maryland

Air Sampling and Asbestos-Containing Materials in Buildings

Garry J. Burdett, James J. Smith, and Chris D. Papanicolopoulos — Georgia Institute of Technology, Atlanta, Georgia

Airborne Asbestos Fiber Levels in Buildings and Their Impact on Risk Management

Session IV - Risk Assessment

Janet M. Hughes — Tulane University, New Orleans, Louisiana

The Derivation and Use of Asbestos Risk Estimates

Julian Peto — Institute of Cancer Research, Oxford, England

Fibre Carcinogenesis and Environmental Hazards

PARTICIPANT LIST

Charles Achilles
Institute of Real Estate Management Foundation

Tom Black
Urban Land Institute

Jean Chesson
Chesson Consulting

Mort Corn
Johns Hopkins University

Donald Dewees
University of Toronto, Canada

Sandra Eberle
Consumer Product Safety Commission

Nurtan Esmen
University of Pittsburgh

J. Bernard Gee
Yale University School of Medicine

Michael Gough
Center for Risk Management

Timothy Hardy
Kirkland and Ellis

Dick Hopper
U.S.G. Corporation

Sarah Hospedor
National Institute of Realtors\

Janet M. Hughes
Tulane University

Richard Innes
Legislative Assistant to
Senator John Chaffee

Patrick Kinney
Health Effects Institute

Arthur Langer
Brooklyn College of the City University of New York

Henry Lee
Harvard University

Si Duk Lee
Harvard University
U.S. Environmental Protection Agency

Peter Lees
Johns Hopkins University

Sue Lin Lewis
Harvard University

Douglas Liddell
McGill University, Canada

John F. McCarthy
Environmental Health and Engineering, Inc.

Karen Milne
U.S. Environmental Protection Agency

Kenneth Millian
W.R. Grace and Co.

Brooke Mossman
University of Vermont

Halûk Özkaynak
Harvard University

PARTICIPANT LIST

Edward Peters
Arthur D. Little, Inc.

Julian Peto
Institute of Cancer Research, UK

Charles Powers
Health Effects Institute

Bertram Price
Price Associates

Robert Sawyer
Entek, Inc.

James Smith
Georgia Institute of Technology

John D. Spengler
Harvard School of Public Health

Edlu Thom
W.R. Grace and Co.

Hans Weill
Tulane University

Dietrich Weyel
University of Pittsburgh

John Welch
Safe Buildings Alliance

Richard Wilson
Harvard University

Mark Wine
Safe Buildings Alliance

ISSUES RELATED TO THE POTENTIAL HEALTH HAZARD OF ASBESTOS CONTAINING MATERIALS IN BUILDINGS

MORTON CORN

Department of Environmental Health Sciences, School of Hygiene and Public Health, Johns Hopkins University, 15 North Wolfe Street, Baltimore, Maryland 21205

The potential hazard associated with Asbestos Containing Materials (ACM) in schools and buildings is reviewed from the perspective of the industrial hygiene paradigm Anticipation, Recognition, Evaluation and Control. The historical high exposure occupational experience engendered anticipation and recognition of asbestos as a potential hazard. Evaluation should be accomplished by air sampling to establish airborne concentrations associated with the inhalation risk. The technique of risk assessment is then used with measured exposure concentrations to estimate the lifetime risk of carcinogenic effects, based on a linear, no threshold model of high dose to low dose extrapolation. The latter approach is routinely invoked by the Cancer Assessment Group of the U.S. Environmental Agency, as well as by other U.S. regulatory agencies and the International Agency for Research on Cancer. Primary control options are Operations and Maintenance (O&M) programs and removal. O&M is judged to be the preferable option from the points of view of the health risk to removers and occupants and cost. Finally, the magnitude of resources being allocated to a low level health risk is highlighted and attributed to inadequate public understanding of the nature of the occupant risk to ACM in buildings.

INTRODUCTION

The purpose of this presentation is to offer a framework for considering some of the major technical and policy issues relevant to the current concern with asbestos in buildings, and primarily with the concern for the exposure, often referred to as non-occupational exposure of building occupants. The issue has received national attention; there is a Federal regulation administered by the Environmental Protection Agency for

addressing the problem in the nation's schools (1). Also, there are at least two draft congressional bills to extend Federal regulation to commercial and public buildings. This paper uses as its framework the paradigm of the Industrial Hygienist, namely, "recognition, evaluation and control". Superimposed on that proven paradigm is the word "anticipation". By systematically approaching our concern for asbestos with this model as a guide, it is possible to place in perspective some of the past and current ingredients of public policy in this area, and to hopefully resolve some of the current concerns with this potential hazard.

I have been critical of U.S. public policy regarding asbestos containing materials in buildings (2). On the other hand, (by developing this policy) we have learned a great deal in the past ten years about asbestos in buildings and the risk to occupants. It is inappropriate to fault yesterday's policies on the basis of today's facts; that would be a classic example of enlightened hindsight. However, we should be compelled to learn from the past. In this constructive sense it is appropriate to recognize poorly based policy decisions that still greatly influence current regulatory procedures, decisions that were faulty at the points in time at which they were made.

They violated certain hard-won understandings and procedures of the occupational health and environmental professions. With this caveat let us proceed.

1. ANTICIPATION

The public concern with asbestos in buildings could be anticipated and indeed, some far-sighted individuals did anticipate it. In late 1977, I was asked by members of the Real Estate and Construction Division of the IBM Corporation to assist with their current efforts to determine which of the buildings they leased, rented or owned contained asbestos fireproofing, acoustic ceiling materials or pipe lagging. They anticipated occupants of those buildings would be concerned about asbestos because they would have little appreciation for the dose-response characteristics of inhaled toxic materials. Concern would emanate from an emotional response to the word "asbestos". IBM wanted to address this concern in a candid, explicit and forthright manner. Subsequently, in 1986 the IBM Management Program was publicly cited as a model for operations and maintenance programs by the Environmental Protection Agency. The Company determined which buildings contained asbestos after resolving problems with analysis of bulk samples of Asbestos Containing Materials (ACM) from the structures. They informed occupants of the presence of ACM and placed the inhalation risk in perspective with the best risk assessment tools available at the time. They also authored an Operations and Maintenance Manual for corporate-wide use. IBM educated its building administrative managers on the O&M program, which was incorporated into their on-going building maintenance procedures.

The anticipation of concern for ACM presumably extended to the Environmental Protection Agency, which issued the first guideline document addressing this subject in 1979 (3).

2. RECOGNITION

Recognition of an occupational health or environmental potential hazard is a qualitative process. It usually relies on the senses; measurement techniques are infrequently invoked. The determination of whether material in a building is or is not asbestos using a microscopic analysis of a sample of the material, which is a measurement technique, is here used as part of the evaluation step. Recognition of the potential hazards of ACM also implies that one understands the routes of entry by which a toxic material can enter the body. Material can enter the body through the skin, by inhalation via the oronasal route, and by ingestion. In the case of ACM, recognition should lead to an immediate focus on inhalation of airborne asbestos fibers as the major route of entry to the bodies of building occupants who are at potential risk.

Asbestos has inflicted major damage on those occupationally exposed in the first seventy years of this century. Numerous textbooks and journal articles relate this tale of havoc. U.S. courts are deeply enmeshed in sorting out questions of retribution for personal damages. It is not my purpose to defend asbestos. It is my purpose to delineate, as accurately as possible, the risk from asbestos in a different exposure mode than that of the past. The past was concerned with workers in production situations or insulators installing ACM. Exposure concentrations of fibers in the air were orders of magnitude greater than those to which occupants of buildings containing ACM are exposed. This contrast necessitates that environmental specialists recognize that response to a toxic material is a function of the amount of material to which the target organ in the human body is exposed (4). Because we cannot measure the concentration of asbestos to which the lungs are exposed, we use a surrogate for that concentration, namely the concentration of asbestos in air breathed by the exposed individual. The recognition that the airborne concentration of asbestos fibers is the most significant parameter to understand in order to estimate the risk to those non-occupationally exposed is a fundamental ingredient of the public policy development for ACM in buildings. It is a point that did not escape policy planners at the Environmental Protection Agency in the formulation of policy for asbestos in schools, but it was relegated to a minor role in the development of policy.

This failure to accept airborne concentration as an important parameter for assessing inhalation risk to building occupants led to the invocation of an unproven algorithm for evaluating the potential for future damage to health by ACM in school buildings. The EPA algorithm has been repeatedly demonstrated to not correlate with measured concentrations

of asbestos fibers in air and to be an internally inconsistent tool for evaluation of risk due to ACM (5). The first report of problems with the algorithm was presented to EPA by its own contractor in 1981 (6). The agency has not yet publicly recognized the indefensibility of the algorithm as an ACM assessment tool. Algorithm parameters such as proximity of ACM to an air duct and percentage asbestos content of ACM continue to be used in self proclaimed assessment schemes and variations on the EPA algorithm by assessors of ACM risk in buildings. The momentum build up by this error in ACM assessment will not be recognized by the public until the agency publicly disclaims the algorithm.

Another ingredient of recognition that has still not entered into this dialogue for policy formulation and one which is demanding of attention, is that our previous experience with asbestos in the occupational environment indicates that there is a difference in the biological response of those exposed to different types of asbestiform fibers, in particular chrysotile versus the amphiboles, when the health outcome is mesothelioma (7,8). Because an estimated 97% of the asbestos fiber in United States ACM has been Canadian chrysotile, the differential biologic potency of asbestos fiber types is a crucial point for public policy. The National Academy of Sciences report (9) and the various Environmental Protection Agency guidance documents (3,10,11), which briefly allude to what at the times of publication of these documents was considered a "controversy", quickly relegate this issue to a backseat in the formulation of public policy. The more recent scientific literature continues to support the differential fiber type potency re: mesothelioma (12). It is a major point. To my knowledge, the United States is the only nation regulating asbestos that does not recognize the differential biological potential of these categories of fibers. Indeed, if chrysotile is significantly less potent with regard to one manifestation of asbestos disease i.e. mesothelioma, then the public health significance of non-occupational exposure to asbestos is enormously reduced below the presently perceived risk level, as established by risk assessment methodologies.

Another point of recognition in the approach to characterization of the potential problem of ACM in buildings is the recognition of certain proven evaluative tools to deal with analogous potential concerns. Here I refer to the utilization of air sampling as a science and art to evaluate potential inhalation hazards. This science and art has been developed over more than 75 years of research and professional practice. The latest techniques are available in professional compendiums and are widely utilized by professionals. Air sampling is an everyday tool of the regulatory agencies concerned with enforcement of standards for the quality of air, be they in the workplace where OSHA enforces approximately 800 standards for airborne toxic materials, or in the outdoor environment where the EPA measures ambient air quality with the goal of ensuring adherence to ambient air quality standards. New local initiatives on air toxic programs, such as that in Maryland, also rely on air sampling as the tool to ensure compliance with

standards adopted to meet long-term air quality goals. Air sampling, while not a precise tool, is a highly effective tool in the hands of practitioners who have mastered the strategies of sampling and the analytical procedures for the samples collected. A major shortcoming of public policy for asbestos-containing materials in buildings of the United States has been the rejection and consistent denigration of the air sampling tool by the Environmental Protection Agency. The Agency has used rhetoric to characterize air sampling as "a snapshot in time". Also, it is stated that air sampling only characterizes present exposure; it cannot predict the future. Finally, air sampling is condemned for being too expensive. If one is concerned with an inhalation hazard, one must recognize at the outset that a measure of the surrogate for the exposure to the target organ, i.e. the respiratory tract, is the concentration in the breathing zone of those exposed and this concentration must be measured. Rejection of this first principal, a principal hard won over 75 years of scientific and professional practice development, is an error of public policy which will, at the very least, lead to inefficient approaches to control of the potential problem. The inconsistency of using air sampling for post-asbestos removal clearance and not for pre-removal evaluation is an aspect of AHERA regulations that is best characterized as "puzzling".

3. EVALUATION

Evaluation of a potential hazard involves measurement. It is with respect to measurement that evaluation differs from recognition. Because asbestos-containing materials in buildings are associated with a potential inhalation hazard, measurement involves air sampling. The use of an unproven algorithm was not a measurement tool; it was a qualitative tool, one that failed in practice. Air sampling results are needed to perform a risk assessment to estimate the adverse health effects which would be experienced by individuals exposed to the measured airborne asbestos concentrations over extended periods of time. The Cancer Assessment Group of EPA has played a major role in establishing as agency policy the use of the linear, no threshold model for estimation of carcinogenic effects at low levels of exposure of chemicals associated with such effects at higher exposures. It was therefore surprising to read the following statement by the agency in its 1988 Report to the Congress on asbestos in public buildings: "No safe threshold has been established for asbestos. Effects at low levels of non-occupational exposure have been estimated by extrapolation from higher levels, although the validity of this approach has not been empirically demonstrated" (13).

The limitations of risk assessment as a policy making tool were delineated in the National Academy of Sciences Report "Risk Assessment in the Federal Government: Managing the Process" (14). The committee authoring this report urged the adoption of risk assessment by regulatory agencies and this later came to pass by Executive Order.

EPA utilizes risk assessment in all its Heath Assessment Documents for carcinogens and RA is part of all agency considerations. The negative connotations of the above statement in the 1988 Report to Congress on asbestos is unusual and even bizarre considering the extensive use of this methodology by the agency.

In order to insert an exposure concentration in the risk assessment model there must be consensus on fiber type of interest and fiber size. The former topic was discussed above. There is increasing evidence that fibers with lengths greater than 8 μm and diameters less than 0.25 μm are the fibers of biological significance (15). Lippman suggests a diameter 0.15 μm or less for fibers of carcinogenic significance. The exposure level used by EPA in its risk assessments is 0.01 f/cc asbestos-in-air (13). This concentration includes fibers of all sizes, including those less than 5 μm in length, which are thought to not be biologically active based on animal studies (15).

Concentrations of Airborne Asbestos in Buildings

Despite official EPA rejection of air sampling for evaluation of risk, air sampling has been pursued in other countries and it has been pursued in the United States, to a large extent by defendants in litigation. Studies have also been sponsored by the Environmental Protection Agency to obtain baseline data on concentration of asbestos fibers in the air of public buildings (17).

What have we learned from extensive 8-hour air sampling inside and outside of buildings during this past six to eight years? The first samples utilized by EPA for estimates of school children exposed were those from Houston schools. Houston school samples were analyzed by the indirect method of transmission electron microscopy (TEM). It should be noted that all concentrations in buildings fall below the level of sensitivity of phase contrast microscopy (PCM) namely, about 0.01 fiber per cc. The fibers counted in the light microscope must be larger than approximately 0.5 μm in diameter and length because this is the limit of resolution of the PCM optics. We have learned that there are very few fibers of this size in the air of buildings.

In all of the summaries that I present here, I refer to occupant exposure, not to maintenance/employee exposure. The latter is an occupational concern, and there is little question that maintenance personnel who go above the ceiling may do work which by direct contact of their own body or tools with the matrix of ACM causes release of fibers and elevated personal exposures. We do not know the percentage of time maintenance personnel in different buildings receive these exposures, nor do we have a full appreciation of the magnitude of these exposures. We do know that this a situation that the recently revised OSHA Asbestos Standard regulates. Maintenance personnel are entitled to the full

protection of that standard, which has a 0.2 f/cc 8-hour Time Weighted Average permissible exposure limit.

The limitations of PCM necessitates the use of TEM to analyze samples collected on filter papers for purposes of evaluating the exposure concentration. The results of the indirect and direct methods of TEM sample preparation methodology suggests that the indirect leads to elevated estimates of exposure. Thus, the early Houston school results, which triggered the 1979 guidance document (3) were high estimates. These air samples, which were stored, were recently reanalyzed using a direct TEM methodology. The results were pooled with samples from other schools; 473 samples were analyzed, 209 were from Texas (18). Table 1 shows the concentrations of fibers per cc greater than 5 μm length and all fibers. It can be seen that concentrations of fibers >5 μm length are less than heretofore reported. A value of 0.0002 $\pm$ 0.0005 (mean $\pm$ standard deviation) would be appropriate for risk estimation of occupants prior to removal, based on these data.

EPA data from 49 GSA buildings (Table 2) do not differ from Table 1 results with respect to mean concentrations of fibers greater than 5 μm length in air (17).

Risk Assessment

The final step in evaluation is the estimate of the impact of exposure. The asbestos risk assessment approach for non-occupational exposure to asbestos has utilized the dose-response curves of some or all to the studies of occupational exposure (9,19). The procedure has been explicitly delineated by OSHA in its proposal for and promulgation of the modified asbestos standard in 1986 (20). The adaptation of these procedures to schools has been performed by Hughes and Weill (19). Essentially the same procedures were used by the National Academy of Sciences Committee, but different exposure times were assessed. I have quoted the Hughes and Weill results in the past and do so once again (Table 3) as the most reasonable risk estimate for schools to date.

The same procedures can be extended to buildings, with appropriate assumptions for concentration and duration of exposure.

Comparison of these risks with other risks in life were neatly displayed by Commins (Table 4) (21).

The risk from non-occupational exposure to asbestos is clearly at the low end of the risk spectrum.

4. CONTROL

The approach to intervention for asbestos in non-occupational settings should consider the severity of the hazard for which the intervention is designed and the probability of successfully alleviating the hazardous condition. The options for intervention in non-occupational exposure to asbestos include, 1) leaving the asbestos-containing material in place and establishing an operations and maintenance program, 2) encapsulating the material, 3) removing the material. The encapsulation option, which appeared attractive in the early stages of intervention for asbestos in buildings, is now recognized as equivalent to leaving the material in place. Those who encapsulate have merely spent the funds and not gained the benefits. The encapsulant is easily penetrated during renovation. There is no need to "hold fibers in the matrix", as was originally believed. The real alternate options are, 1) operations and maintenance with opportunistic removal during occasions of major renovation when the building has been "opened up", and removal is relatively easy, and 2) removal.

Equating the presence of asbestos with an asbestos inhalation hazard has forced owners of real estate to removal because buyers are genuinely concerned about the asbestos inhalation risk; the public believes that a health risk of concern exists in any building with ACM. Once again, intelligent governmental policy should have preceded the requirements for school administrators with a very broad-based educational program, thus precluding the seeding and subsequent growth of these incorrect perceptions. That is all in the past, and we are now faced with reversing perceptions and opinions that have been formed. A well-written book, now in press, by Michael J. Bennett entitled, "Asbestos Hysteria" describes the growth of public perception of the ACM risk in buildings.

Removal creates dust. Removal must be performed with extraordinary procedures for negative pressure, containment, assurance of the integrity of the containment area, and extensive personal protective equipment for removal personnel. There are good removal firms and there have been exemplary removals. However, the majority of removals I have experienced have been poorly performed, necessitating multiple cleanups after the job was purported to be completed.

The Environmental Protection Agency requires air sampling for certification of removal completion. Curiously, it utilizes the PCM and a fiber concentration that exceeds the concentration of asbestos fibers present in the building before removal. This entire policy is difficult to comprehend. The agency rationale for the air sampling method selected and the lack of specificity of the analytical procedure is non-existent. If removal is performed, air sampling with evaluation by transmission electron microscopy, a technique with the requisite sensitivity that can identify specific fibers, is required.

Early in the concern for asbestos in buildings, it was believed that buildings vibrated, that episodic releases of asbestos, related to abusive or accidental events intruding on the

ACM matrix, created higher levels of asbestos in building air. These concerns have not materialized. All the air sampling data to date indicates that if such releases do occur, the concentrations created in near proximity to the release are quickly damped and of little significance to exposure of building occupants. They may be of significance to maintenance personnel and those creating the disturbance. Interestingly, even asbestos material in poor condition is not associated with elevated concentrations of asbestos fibers in the air of buildings (13).

A substantial asbestos removal industry has developed, reputedly an industry grossing in excess of one billion dollars in 1988. Asbestos removal firms have been on the "buy" list of Wall Street stockbrokers. Approximately 50 thousand persons are employed in the industry, which is projected to grow during the 1990's if current policies persist. Expansion of AHERA to commercial and public buildings will bring enormous growth. Many individuals in public health practice, including myself, believe the next epidemic of asbestos disease in the United States will be experienced by those employed as asbestos removers. Horror stories of the removal process and exposure of removers abound.

All of the above considerations indicate that a careful scientific evaluation of the EPA AHERA policy and its extension to public and commercial buildings is overdue. The enormous national resources we are bringing to bear on ACM in buildings is out of synchronization with the potential risk to health posed to occupants of these buildings. It is too much money chasing too little risk. Our resources can be put to better use to reduce more significant environmental risks to health. Hopefully, this workshop will perform this evaluation and concur in this conclusion.

REFERENCES

1. U.S. Congress. Asbestos Hazard Emergency Response Act ("AHERA"). Pub. L. No 99-519, 100 Stat. 2970 (1986). Section Two of AHERA added a new Title II, 88201-214 to the Toxic Substances Control Act ("TSCA"), codified at 15 U.S.C. 882641-54 (Supp. IV, 1986).

2. Corn, M.: Asbestos and Disease; An Industrial Hygienist's Perspective. Am. Ind. Hyg. Assoc. J. 47 (9), 515-523 (1986).

3. Asbestos-Containing Materials in School Buildings: A Guidance Document. Part 1: U.S. Environmental Protection Agency. Office of Toxic Substances. Washington, D.C. March, 1979.

4. Ottoboni, A.: The Dose Makes The Poison. Vincente Books, Berkeley, CA. 1984.

5. Findley, M.E., V.E. Rose, G.E. Cutter and R.A. Windsor: Assessment of the EPA Asbestos Hazard Algorithm. Am. J. Pub. Hlth. 73: 1179-1181 (1983).

6. Logue, E.E. and T.D. Hartwell: Draft Report on Characteristics of the Asbestos Exposure Algorithm: Emperical Distribution, Correlations and Measurement Validity. Research Triangle, N.C.: Research Triangle Institute. Jan. 1981.

7. McDonald, A.D. and J.C. McDonald: Malignant Mesotheliomas in North America. Cancer 46, 1650-1656 (1980).

8. Newhouse, M.L., G. Berry, and J.W. Skidmore: A Mortality Study of Workers Manufacturing Friction Materials with Chrysotile Asbestos. Ann. Occup. Hyg. 26, 899-909 (1982).

9. National Research Council, Committee on Non-occupational Health Risks: Asbestiform Fibers - Nonoccupational Health Risks. Washington, D.C. National Academy Press. 1984.

10. Guidance for Controlling Friable Asbestos-Containing Materials in Buildings. U.S. Environmental Protection Agency. Office of Pesticides and Toxic Substances. Washington, D.C. EPA Report No. 560/5-83-002. March, 1983.

11. Guidance for Controlling Friable Asbestos-Containing Materials in Buildings. U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances. Washington, D.C. EPA Report No. 560/5-85-002. June, 1985.

12. Churg, A.: Chrysotile, Tremolite and Malignant Mesothelioma in Man. Chest 93, 621-268 (1988).

13. EPA Study of Asbestos-Containing Materials In Public Buildings: A Report to Congress. U.S. E.P.A. Washington, D.C. Feb. 1988.

14. National Academy of Sciences: Risk Assessment in the Federal Government: Managing the Process. Washington, D.C. NAS Press, 1983.

15. Davis, J.M.G. and J.C. McDonald: Low-level Exposure to Asbestos: Is There a Cancer Risk? Brit. J. Ind. Med. 505-508 (1988).

16. Lippman, M.: Asbestos Exposure Indices. Environ. Res. 46 81-106 (1988).

17. Assessing Asbestos Exposure In Public Buildings. Draft Report for the Exposure Evaluation Division, Office of Toxic Substances, Office of Pesticides and Toxic Substances, U.S. EPA Wash., D.C. (Nov. 1987). Prepared by Batelle Columbus Division, Price Associates, Alliance Technologies Corp, Energy Technology Consultants and Midwest Res. Inst. EPA Contract No. 68-02-4294.

18. Corn, M., K.E. Crump, D. McFee and R. Lee: Airborne Concentrations of Asbestos in Schools. Manuscript in preparation.

19. Hughes, J.M. and H. Weill: Asbestos Exposure - Quantitation of Risk. Am. Rev. Resp. Dis. 133, 5-13 (1986).

20. U.S. DOL. OSHA. Occupational Exposure to Asbestos. 29 CFR 1926. 58.

21. Commins, B.T.: The Significance of Asbestos and Other Mineral Fibers in the Environment. Commins Associates, Pippins, Altwood Close, Maidenhead, Berks SL6 4PP. England, 1985. p. 67.

Table 1
Distribution of Mean School Indoor Concentrations (18)
(Number of Schools = 71)

Concentration Measure	f/cc>5μm	Optical[a] f/cc	EPA[b] f/cc	Chr.[c] f/cc	Amp.[d] f/cc	Ind.[e] ng/m3	Total f/cc
Ave.	0.00024	0.00017	0.014	0.017	0.0015	43.0	0.019
Std. Dev.	0.00053	0.00046	0.018	0.024	0.0023	120.0	0.024
Percentiles:							
5.0%	0.	0.	0.00056	0.	0.	0.044	0.00094
10.0%	0.	0.	0.00094	0.	0.	0.29	0.0019
20.0%	0.	0.	0.0017	0.00079	0.	0.52	0.0025
25.0%	0.	0.	0.0023	0.00099	0.	0.64	0.0030
30.0%	0.	0.	0.0036	0.0022	0.	0.86	0.0056
40.0%	0.	0.	0.0056	0.0066	0.00040	1.5	0.0072
50.0%	0.	0.	0.0083	0.0083	0.00065	2.9	0.0098
60.0%	0	0.	0.010	0.014	0.0011	3.8	0.015
70.0%	0.	0.	0.013	0.018	0.0015	9.5	0.019
75.0%	0.	0.	0.016	0.023	0.0019	19.0	0.024
80.0%	0.00045	0.	0.017	0.026	0.0022	25.0	0.026
85.0%	0.00067	0.00042	0.030	0.039	0.0030	36.0	0.042
90.0%	0.00083	0.00067	0.041	0.049	0.0043	110.0	0.049
95.0%	0.0014	0.0014	0.048	0.065	0.0062	300.0	0.065
97.5%	0.0023	0.0019	0.073	0.12	0.010	480.0	0.12
Maxima:	0.0023	0.0023	0.093	0.12	0.012	740.0	0.12

NOTE: The method used to compute percentiles is definition 5 in the SAS Procedures Guide (1985b).

[a]Fibers at least 5 μm long with a diameter of least 0.2 μm (fibers projected to be counted by PCM).

[b]Fibers having length at least 0.5 μm and at least five times width (used by EPA in defining when schools are sufficiently free of asbestos following abatement).

[c]Fibers identified as chrysotile.

[d]Fibers identified as amphiboles.

[e]Fiber mass calculated from indirect TEM methods.

Table 2

Summary Statistics for Average Airborne Asbestos Fiber Concentrations (f/cc) (17)

	Outdoor	Category 1	ACM Category 2	ACM Category 3
Median	0.00000	0.00010	0.00040	0.00058
Mean	0.00039	0.00099	0.00059	0.00073
Sample Size	48 (sites)	6 (buildings)	6 (buildings)	37 (buildings)
Standard Deviation	0.00096	0.00198	0.00052	0.00072

Notes: (1) The data points used in the calculation of each statistic are the average concentration within a building (for indoor samples) or the concentration outside each building (for outdoor samples).
(2) Category 1 - No ACM
Category 2 - ACM primarily in good condition
Category 3 - At least one area of significantly damaged ACM
(3) 387 air samples
48 outdoor air samples
(4) No asbestos fibers detected in 83% of the 387 samples

TABLE 3

Estimated Number of Lifetime Deaths Attributable to Six Years of Asbestos Exposure in Schools (Per 10^6 Exposed) by Fiber Type and Average Concentration (19)*

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

*Refer to original article for model assumptions re: calculation of table entries.

Table 4
Lifetime Risk Values for Selected Situations (21)

Selected risk situations, mainly U.S. data	Lifetime risk per 100,000
Extra High Risk	
Smoking (all causes of death)	21,900
Smoking (cancer only)	8,800
High Risk	
Motor vehicle, U.S.A., 1975 (deaths)	1,600
Elevated Risk	
Frequent airline passenger (deaths)	730
Cirrhosis of liver, moderate drinker (deaths)	290
Motor accidents, pedestrians, U.S.A., 1975 (deaths)	290
Skiing, 40 hours per year (deaths)	220
Moderate Risk	
Light drinker, one beer per day (cancer)	150
Drowning deaths, all recreational causes	140
Air pollution, U.S.A., Benzo(a)pyrene (cancer)	110
Natural background radiation, sea level (cancer)	110
Frequent airline passenger, cosmic rays (cancer)	110
Low Risk	
Home accidents, U.S.A., 1975 (deaths)	88
Cycling (deaths)	75
Person sharing room with smoker (cancer)	75
Diagnostic X-rays, U.S.A. (cancer)	75
(Risk level where few would commit their own resources to reduce risk; Royal Society, London, 1983)	70
Very Low Risk	
Person living in brick building, additional natural radiation (cancer)	35
Vaccination for small pox, per occasion (death)	22
One transcontinental air flight per year (death)	22
Saccharin, average U.S.A. consumption (cancer)	15
Consuming Miami or New Orleans drinking water (cancer)	7
(Risk level where very few would consider action necessary, unless clear causal links with consumer products, Royal Society, London, 1983)	7
Extremely Low 'Rare-Event' Risk	
One transcontinental air flight per year, natural radiation (cancer)	4
Lightning (deaths)	3
Hurricane (deaths)	3
Charcoal broiled steak, one per week (cancer)	3
ENVIRONMENTAL ASBESTOS RISK*, 1985, (cancer) ('around one per 100,000 or lower'; this Report)	1
('Acceptable' risk: World Health Organization for drinking water, 1984, (259) (cancer)	1
(Further control not justified, Royal Society, London, 1983)	0.7

*Excludes possible effects of smoking

EPIDEMIOLOGICAL OBSERVATIONS ON MESOTHELIOMA* AND THEIR IMPLICATIONS FOR NON-OCCUPATIONAL EXPOSURE TO ASBESTOS

DOUGLAS LIDDELL

Department of Epidemiology and Biostatistics, McGill University, 1020 Pine Avenue West, Montreal, Quebec, Canada H3A 1A2

The causes of mesothelioma can be stated as follows:- primarily, the long fibres of amphibole asbestos (inhaled during several months of exposure, usually to high concentrations); secondarily, chrysotile (from several years of exposure to fibre probably contaminated with amphibole); thirdly, where there are deposits, erionite fibres; and some unknown agent(s).

In over 30 occupational cohort studies, the numbers of deaths from mesothelioma, expressed as percentages of total mortality, depended on type of fibre and work: 8.3% for amphibole + chrysotile; 7.7% for insulation work; 6.6% for crocidolite; 2.9% for shipyard work; 2.9% for amosite; 1.6% for mainly chrysotile textiles; 0.6% for chrysotile with a small proportion of amphibole; and 0.2% for chrysotile alone.

The importance of a substantial period of exposure or of high concentrations has been shown in amosite factory workers in Paterson NJ, in female gas mask workers in Nottingham (UK), and in miners and millers of crocidolite (Western Australia and South Africa), of amosite and of amphibole mixtures (South Africa), and of chrysotile (Quebec), and in textile workers in Rochdale (UK).

From lung tissue analyses, it has been concluded that amphibole fibres could explain most mesothelioma cases in Canada, and other inorganic fibres, including chrysotile, very few. Relative risk was very strongly related to the concentration in the lung of long amphibole fibres, no additional information being provided by shorter fibres, nor by chrysotile fibres.

Mesothelioma had been recognized in Europe before asbestos was produced commercially (starting in 1878 in Quebec). Trends have been examined in mortality and in incidence: among females, annual rates remained almost unchanged for mortality in Canada (1965-83), and, for incidence of both pleural and peritoneal mesotheliomas, in the US (1973-84); among males, rates at least doubled over these periods, the increase in the US being confined to pleural tumors. Mesotheliomas from the mid-1960's until recently which were attributable to asbestos would have been due to exposures 30-40 years previously, a period in which consumption increased rapidly. Thus, the rates for males can be explained by occupational exposure, while those for females (together with other evidence) suggest that the unknown "background" agent(s) did not change.

Almost all commercial asbestos has always been chrysotile (95% in the US in 1974). Among the seven cases of mesothelioma at Thetford Mines, found to 1975 in the 1891-1920 cohort of chrysotile miners and millers, the lowest accumulated exposure was 59 (fibres/ml) × (years), i.e. 8.8 years at an average intensity of 6.7 (f/ml). There was one case with only 7 months employment - but at an average intensity of 138 (f/ml).

It is difficult to envisage a realistic scenario in which mesothelioma could be attributed to non-occupational exposure to asbestos.

INTRODUCTION

In his Lecture as the 1985 Charles S. Mott Laureate, Wagner (1986) described work continuing from 1952 to enquire if all types of asbestos caused the same diseases, with particular emphasis on diffuse pleural and peritoneal mesotheliomas. His conclusions included the following: "It is the fibre that is retained in the lung tissue that is responsible for [mesothelioma]" and "... evidence points to exposure to the amphiboles, or material containing amphibole, as being more hazardous than exposure to uncontaminated chrysotile fibres".

Wagner's Lecture was a review of all evidence, but the present paper is confined to epidemiological observations. What is generally taken as the starting point of the epidemiology of mesotheliomas was the report by Wagner et al. (1960) of 33 cases of malignant pleural mesothelioma in the neighborhood of the crocidolite mines in the North-West Cape Province of South Africa. Because mesothelioma is so rare in the general population, it might have been expected that the natural line of further enquiry would have

been what used to be called "retrospective", but there have been few case/referent studies. In all of them, even in what is arguably the best (McDonald & McDonald, 1980), it is difficult to be sure about any subject's exposure to asbestos in terms of type of fibre, duration and, especially, intensity of airborne concentrations. Certain of these doubts have been reduced in at least some of the studies of over 40 cohorts of asbestos workers.

The current review will concentrate on the male occupational cohorts, but mention will be made of other relevant epidemiological findings. Much of the material presented has been extracted from an important chapter by McDonald and McDonald (1986) with some re-arrangement and up-dating as necessary. That chapter listed 132 references, which are not repeated here except where it is appropriate to cite specific reports.

New material is included from an ongoing mortality study of white male workers in amphibole mines South Africa. A total of 7,318 men were followed from first employment until the end of 1980, by which time there had been 1,225 deaths, while 6,093 were known to be alive in 1981; less than 3% had been lost to view. The concern here is with the 30 deaths which, on best available information, were due to mesothelioma. Three cohorts were defined: of 1,804 men exposed only in crocidolite mining (among whom 20 had died from mesothelioma); of 1,406 men exposed only in amosite mining (4 mesotheliomas); and of 4,108 men exposed to amphiboles in combinations or with other minerals (6 mesotheliomas). (Miners exposed only in chrysotile mining had been excluded from the study.) This study will not be reported elsewhere for some months, and we are indebted to Dr. G. K. Sluis-Cremer for permission to present preliminary findings here.

FIBRE TYPE

The question Wagner (1986) was examining is of great importance, because approximately 95% of asbestos utilization in the US has always been of one particular type, namely chrysotile; the remainder has been made up mainly of amosite and crocidolite, both of the amphibole group. Yet, despite the preponderance of chrysotile, the majority of asbestos-related mesotheliomas can be attributed to the much less common amphiboles, crocidolite being apparently the most toxic.

PROPORTIONAL MORTALITY

It is often possible to get some clues as to aetiology by consideration of the simple statistic: proportional mortality rate. In the present context, this rate is the number of deaths due to mesothelioma expressed as a percentage of the total number of deaths. McDonald and McDonald (1986) incorporated a table (2-3) giving the material for 29 male asbestos-exposed cohorts, reporting results - as far as possible - for 20 or more years since

first employment. In Table l, this material has been up-dated from more recent reports on some cohorts, and with our own findings from South Africa. The original classification of the cohorts by fibre type and process has had to be varied slightly, and the entries have been arranged in ascending order of proportional mortality.

It is well known, as pointed out by McDonald and McDonald (1986), that these rates "are subject to differing age composition and length of follow-up". However, the final column of Table 1 gives a "homogeneity χ^2 statistic", which shows that within most of the classes proportional mortality rates were remarkably similar.

The 30-fold difference in proportional mortality rates in cohorts exposed to just one of the three main types of asbestos fibre (0.2% for chrysotile alone, 2.9% for amosite alone, 6.6% for crocidolite alone) appears unlikely to be accounted for by variations in epidemiologic method, particularly in the light of the consistency of the rates within classes. Thus, within chrysotile alone the highest rate was 0.3%, and within amosite alone the rates varied between 1.5% and 4.6%; within crocidolite alone the rates ranged between 4.9% and 11.9%.

In 11 female cohorts, the proportional mortality rates showed a similar pattern: 0.2% for three cohorts exposed to chrysotile only (1 mesothelioma among 414 deaths); 3.6% for six cohorts exposed to mixtures (38/1049); and 10.6% for two cohorts exposed to amphiboles only (19/179).

MESOTHELIOMA RELATED TO LUNG CANCER EXCESS

It is sometimes stated that measurements of exposure are so inaccurate that a more reliable estimate of dose can be obtained from the extent to which lung cancer mortality is inflated. Not only is this thesis quite untenable - as shown in particular by Sébastien et al. (1989) - but there is marked circularity in the ensuing argument. However, for completeness, Table 2 presents again the material from Table 1, but replaces total mortality with "lung cancer excess". This excess is the difference between the numbers of lung cancer deaths observed and those expected had the cohort suffered the mortality of the chosen reference population. It is again possible to examine the consistency of the M:LCE ratio, that is (Deaths from mesothelioma)/(Lung cancer excess), within the nine classes containing more than one study; it is obvious that the within class consistency so common in Table 1 is replaced by gross inconsistency within at least four of the classes.

Thus, the differences among classes are less emphatic than from proportional mortality rates. Nevertheless, for the five cohorts exposed to chrysotile alone the M:LCE ratio averaged only 0.11 (the highest ratio being 0.22) and for the three exposed to crocidolite alone the ratio was 13 times higher on average (the lowest being 1.25). It

remains clear that the differences among classes transcend even the much larger within-class inconsistencies.

In the female cohorts, the M:LCE ratios were: chrysotile 0.2; mixtures 0.8; and amphiboles 2.3. Although the pattern is similar, the ratios were considerably higher than for men, probably a reflection of major differences in smoking habits affecting the lung cancer excesses.

INTERVAL BETWEEN FIRST EXPOSURE TO ASBESTOS AND DEATH DUE TO MESOTHELIOMA

From the start of epidemiological investigation of mesothelioma (Wagner et al., 1960), it has been clear that it usually takes many years after exposure to asbestos before the tumor is manifest. Thus, Selikoff et al. (1979) reported 175 mesotheliomas among 2,271 deaths, 1967-76, in 17,800 asbestos insulation workers in the United States and Canada; 63 of the mesothelioma cases had died more than four decades after starting insulation work. In 1983, Browne reported that in asbestos-related cases the onset of illness was usually 35 to 40 years after first exposure, rarely less than 20 years, and perhaps never less than 15 years.

Seidman et al. (1986) reported a second follow-up of short-term employees at an amosite factory; all 17 mesotheliomas were discovered at least 20 years after starting work (Table 3). Particularly important are the mesotheliomas among female workers exposed to crocidolite in a Nottingham (UK) gas-mask factory. The latest information on the intervals between first exposure and death due to mesotheliomas is in Table 4, taken from a diagram in Jones (1987). In the two cases marked (a) the interval must have been at least 18 years and could have been up to five years longer. The three latest cases occurred in the year before publication of this diagram (Jones, 1987).

In two of the three other cohorts discussed by Peto et al. (1982), Newhouse and Berry (1976) had reported 45 mesotheliomas in factory workers with mixed fibre exposure, and Hobbs et al. (1980) 26 in crocidolite miners and millers; none of those 71 cases were within 10 years of first exposure. In the last cohort, of mainly chrysotile textile workers, there had been seven deaths from mesothelioma, none before 20 years had elapsed since starting work (Peto, 1980).

Hughes et al. (1987) recorded ten mesotheliomas in a study of men in two asbestos cement plants. The shortest intervals from initial employment in the plants to death were 14.6 years and 18.8 years; the other eight intervals ranged from 28.8 years to 54.0 years.

Table 5 is new material from our study in South Africa. The shortest interval, 7 years, occurred in a man who had always lived in Kuruman (N.W. Cape) and died at the

age of 30. It is likely that his mesothelioma was due to non-occupational environmental exposure to crocidolite.

MODELLING RISK

In an important paper, Peto et al. (1982) examined how mesothelioma death rates in asbestos workers varied with time from first exposure and age at first exposure, stating that the latter had little or no influence. In Table II of their paper, they reported an extended follow-up of the 17,800 asbestos insulation workers whom Selikoff et al. (1979) had followed from 1967 through 1976; the new total of deaths from mesothelioma was 242. These are cross-tabulated, together with man-years of observation, by year of first exposure (in 11 five-year groups, the most recent being 1962-66) and by years since first exposure (in 9 five-year groups, from 10-14 to 50-79, thus excluding 6 deaths beyond the age of 80). Because this cohort study had a "cross-sectional start" (Liddell, 1988b), the entries in Table II (Peto et al. 1982) lie down a diagonal band, 20-years wide both vertically and horizontally. These authors analyzed the material for those who started insulation work in the years 1922 through 1946; as all 17,800 subjects were alive when observation started in 1967, any deaths - whether from mesothelioma or not - between the work-start years (1922-1946) and the end of 1966 were excluded. For 1942-46 recruits, observation could not start until at least 20 years after first exposure; for 1922-26 recruits, no observations were possible within 40 years of first exposure. The number of mesotheliomas included in the analysis was 180, of which 65 were pleural and 115 peritoneal.

A regression analysis of log(annual mesothelioma mortality) against log(years since first exposure) gave the slope of the fitted line as 3.2, and the fitted equation was:

$$\text{annual incidence} = \underline{b} \times (\text{years since first employment})^{3.2}$$

$$\text{where } \underline{b} \times 10^8 = 4.37$$

Peto et al. (1982) also showed, in their Table III, that the model fitted well four other data sets, provided a specific value of $\underline{b}$ was obtained for each. A summary of this material, extended and updated, is given in Table 6. In the 1982 table, values of $\underline{b} \times 10^8$ varied only between 2.94 and 5.15; this consistency is remarkable as each value of $\underline{b}$ is a reflection of the average exposure (some combination of duration and intensity - and not unaffected by fibre type), and it would have been anticipated that such averages might have

been quite disparate in the five cohorts. In the extended Table 6, the values of b varied much more widely, which is more in line with anticipation.

EXPOSURE-RESPONSE RELATIONSHIPS

An important aspect of epidemiology is the demonstration of exposure-response relationships. Unfortunately, reliable estimates of the intensity of exposure to respirable airborne contaminates are rare, but it is usually possible to use duration of exposure as a surrogate. Evidence of this nature comes from Paterson NJ, Nottingham, Western Australia, South Africa and Rochdale.

Table 7 has been extracted from several pages of Seidman et al. (1979). No case occurred in men exposed for less than seven months; however, for exposures of 10-12, 13-24, and 24+ months' duration, the numbers of cases per hundred men exposed varied little (between 3.2% and 3.8%).

Jones et al. (1980) gave a table showing the number of women exposed only to crocidolite by length of exposure: none of the 16 mesotheliomas occurred in women exposed for less than five months. Hobbs et al. (1980) gave the distribution of 26 cases of mesothelioma, in miners and millers of crocidolite in Western Australia, by length of exposure in three categories: less than 3 months (0 cases in 47,292 man-years); 3-11 months (10 cases in 38,862 man-years, or 26 per 100,000); 12 or more months (16 cases in 27,825 man-years, or 58 per 100,000). Follow-up to the end of 1980 has been reported (Armstrong et al., 1988), but the only information on exposure-response is in a diagram from which it is impossible to get reliable quantification. Hughes et al. (1987) provided a list of their 10 cases, giving duration of employment: at one asbestos cement plant, the two mesotheliomas arose in men employed for three months and ten months; at the other plant, one mesothelioma occurred in a man employed for five months, the other seven in men employed for at least 13 years and up to 42 years.

Table 8 classifies cases and referents in the current South African cohort studies by length of exposure and two types of exposure: all the exposures of less than a year occurred in mining of amosite only (3 cases) or of both amosite and crocidolite (1 case). For each case, referents were chosen as men who lived to a greater age than the case and who were first exposed to asbestos earlier in life than the case. Table 8 thus provides powerful evidence that the risk of mesothelioma in amphibole mining is negligible without a substantial period of exposure.

In the crocidolite mines, this period was a least a year (in fact 14 months); with longer periods, however, there was little variation in the mesothelioma risk. For other cases, risk increased with duration of exposure, a test for trend (in an unmatched analysis) yielding a χ^2 statistic, with 1 df, of 6.64.

An extension of material given by Peto et al. (1985) is in Table 9. As Doll and Peto (1985) pointed out, the risk of mesothelioma for men with less than ten years scheduled service (from the "All periods" line of Table 9, O = 1; E = 7.42; O/E = 0.13) was very much less than for men with longer service (O/E = 3.50), although the risk did not appear to depend on length of service beyond ten years.

Table 10 presents a previously unpublished case-referent analysis of the seven mesothelioma cases, by 1975, at Thetford Mines, five referents for each case having been selected as described for other causes of death by McDonald et al. (1980) and Liddell et al. (1984). (Corresponding analysis was not carried out on the three cases at Asbestos for reasons described in the references above.) Nowadays, we would have selected referents rather differently (see Liddell, 1988a); however, there was only one case with less than eight years net duration of employment; this man suffered only seven months exposure, but at the extremely high average intensity of exposure (see Liddell et al., 1984) of 138 f/ml.

LUNG TISSUE ANALYSES

In the last few years, there have been many studies, in several different countries, of fibre concentrations in lung tissue of mesothelioma cases and of referents. Wagner et al. (1988) concluded inter alia that crocidolite and amosite were strongly associated with mesothelioma, whereas no such correlation was evident with chrysotile. They recognized that one possible explanation was the weaker pulmonary penetration of chrysotile fibres and their physicochemical dissolution in the lung, so that the damage might have been wrought by chrysotile fibres that had subsequently disappeared. These authors considered a more likely explanation was that chrysotile plays only a minor part in the causation of asbestos-related diseases, especially mesothelioma, where they found less chrysotile than in the referents.

Even more recently, McDonald et al. (1989) have reported a larger study of 78 cases from autopsy of mesothelioma in Canada, 1980 through 1984, and from matched referents. Their main conclusion was that amphibole fibres could explain most mesothelioma cases in Canada, and other inorganic fibres, including chrysotile, very few. Relative risk was very strongly related to the concentration in the lung of long amphibole fibres, no additional information being provided by shorter fibres, nor by chrysotile fibres.

In weighing the evidence from the lung tissue studies, it is important to appreciate that much commercial chrysotile is contaminated with a very small admixture (probably much less than 1%) of tremolite, another amphibole. An important commentary has been published by Dunnigan (1988) which evoked five Letters to the Editor: all this material should be assimilated but cannot, of course, be included here.

POPULATION STUDIES

Jones (1987) states that it is probable the first published report of diffuse malignant mesothelioma of the pleura was in 1870, eight years before the start of the asbestos industry, and it is almost universally accepted that there is some unknown non-fibrous cause of mesothelioma.

Connelly et al. (1987) showed the "time-trends in age-adjusted mesothelioma incidence rates by primary site and sex among whites: SEER date, 1973-84". Over more than a decade there were no discernible trends except for a steady increase, more than doubling in 12 years, in the rates for pleural tumors in males. This rate of increase is quite consistent with the increase in asbestos utilization in the US in the relevant decade between the two World Wars (McDonald, 1985). In Canada, the numbers of fatal cases of malignant mesothelioma reported by pathologists from 1961-83 increased about two-fold in males after 1968, when a survey described by McDonald and McDonald (1986) began. However, the numbers of such cases in females had not been increasing.

In Great Britain, mesothelioma death rates (crude) trebled for men between 1968-71 and 1980-83, and doubled for women. Jones et al. (1988) state that the "trends in the use of asbestos and in age- and sex-specific death rates suggest that the annual number of mesothelioma deaths will continue to rise, possibly until the end of the century. This increase will be concentrated among the men, as the main asbestos exposure of women occurred during the war and the annual death rates due to this exposure may already have peaked".

The simple explanation of the increasing rates for females in Great Britain, and the lack of such increases in North America, suggest that most of the mesothelioma risk among American females is due to one or more unknown factors which are changing little; see also McDonald et al. (1989). This view is supported by the consideration that, if there were going to be an increase in mesothelioma rates in US females due to non-occupational asbestos exposure, it should be evident by now.

RISK FACTORS OTHER THAN ASBESTOS

That there is at least one other risk factor is known from the reports from Turkey of excessive numbers of deaths attributed to mesothelioma in two small villages; the evidence suggests strongly that heavy environmental exposure, from birth, to fine needlelike fibres of erionite was primarily responsible. Other etiologic agents are discussed by McDonald and McDonald (1986); they felt it unlikely that tobacco, man-made mineral fibre, or certain other materials were responsible for mesothelioma, but described evidence suggesting that certain organic fibres might have caused mesotheliomas in sugar cane

workers. Other reports have appeared but are not considered relevant in the present context.

CONCLUSIONS

Of the 483 cases of mesothelioma in the 33 male asbestos-exposed cohorts (Tables 1 and 2), more than half were included in the analysis by Peto et al. (1982), and that was dominated by the 180 mesotheliomas among North American insulation workers. There can be no doubt that the exposures suffered by these victims two to four decades earlier had been extremely severe. Mesothelioma will undoubtedly continue to arise, for a few decades (with a peak around the turn of the century), due to occupational exposure from the time of World War II to the mid-1960's, before industrial hygiene was dramatically improved.

Occupational exposures today are less severe by several orders of magnitude. Non-occupational exposure, where it exists, is less severe again, also by several orders of magnitude. Although there is little evidence on exposure-response relationships in which each individual's intensity of exposure has been estimated - see Peto et al. (1985), Armstrong et al. (1988), Table 10 - it all points clearly to the risk of mesothelioma being directly related to accumulated exposure. This means, of course, that non-occupational exposure, known to be at very low levels, carries minuscule risk.

Further, it must be borne in mind that non-occupational exposure will generally be to chrysotile, and only very slightly to the amphiboles which have been responsible for so much of the mesothelioma reported in occupational cohorts. Thus, the risk is almost certainly lower by at least one order of magnitude than any estimate based on exposure to mixtures of chrysotile and amphiboles.

Even where there is non-occupational exposure to amphiboles, it would seem reasonable from the evidence presented in Tables 7 and 8 and in the text, to believe that such exposure would have to last for several months before any risk of mesothelioma would occur. As the majority of the non-occupational to exposure is to chrysotile, it would have to be maintained for several years (see Tables 9 and 10) before a risk occurred. (In this regard, it should be borne in mind that Tables 9 and 10 relate to "commercial chrysotile", i.e. not pure chrysotile but with its usual tremolite contamination.)

Finally, it seems clear from much evidence that the fibres that are pathogenic for mesothelioma are very fine but long (greater than, say 8 μm). As the fibres in the non-occupational environment are generally much shorter, they are virtually innocuous.

In summary, it is difficult to see how any mesothelioma could be attributed to non-occupational exposure to asbestos in the United States today.

REFERENCES

Armstrong, B.K., DeKlerk, N.H., Musk, A.W. and Hobbs, M.S.T. (1988). Mortality in miners and millers of crocidolite in Western Australia. Brit. J. Ind. Med. 45, 5-13.

Browne, K. (1983). The epidemiology of mesothelioma. J. Soc. Occup. Med. 33, 190-194.

Connelly, R.R., Spirtas, R., Myers, M.R., Percy, C.L. and Fraumeni, J.F., Jr. (1987). Demographic patterns for mesothelioma in the United States. J. Nat. Cancer Inst. 78, 1053-1060.

Doll, R. and Peto, J. (1985). "Effects on Health of Exposure to Asbestos". Health and Safety Commission, London.

Dunnigan, J. (1988). Linking chrysotile asbestos with mesothelioma. Amer. J. Ind. Med. 14, 205-209 (with five Letters to the Editor on pp. 235-249).

Hobbs, M.S.T., Woodward, S., Murphy, B., Musk A.W. and Elder, J.E. (1980). The incidence of pneumoconiosis, mesothelioma and other respiratory cancer in men engaged in mining and milling crocidolite in Western Australia. In "Biological Effects of Mineral Fibres" (J.C. Wagner, Ed.), pp. 615-625. International Agency for Research on Cancer, Lyon.

Hughes, J.M., Weill, H. and Hammad, Y.Y. (1987). Mortality of workers employed in two asbestos cement manufacturing plants. Brit. J. Ind. Med. 44, 161-174.

Jones, J.S.P. (Ed.) (1987). "Pathology of the Mesothelium". Springer-Verlag, London/Berlin/Heidelberg/New York/ Toronto/Paris/Tokyo.

Jones, J.S.P., Smith, P.G., Pooley, F.D., Berry, G. et al. (1980). The consequences of exposure to asbestos dust in a war-time gas-mask factory. In "Biological Effects of Mineral Fibres" (J.C. Wagner, Ed.), pp. 637-653. International Agency for Research on Cancer, Lyon.

Jones, R.D., Smith, D.M. and Thomas, P.G. (1988). Mesothelioma in Great Britain in 1968-1983. Scand. J. Work Environ. Health 14; 145-152.

Liddell, F.D.K. (1988a). Maintaining progress in the analysis of cohort studies. In "Progress in Occupational Epidemiology" (C. Hogstedt and C. Reuterwall, Eds.), pp. 71-74. Excerpta Medica, Amsterdam/New York/Oxford.

Liddell, F.D.K. (1988b). The development of cohort studies: a review. J. Clin. Epidemiol. 41, 1217-1237.

Liddell, F.D.K., Thomas D.C., Gibbs, G.W. and McDonald, J.C. (1984). Fibre exposure and mortality from pneumoconiosis, respiratory and abdominal malignancies in chrysotile production in Quebec, 1926-75. Ann. Acad. Med. Singapore. 13, no. 2 (suppl.), 340-344.

Mancuso, T.F. (1988). Relative risk of mesothelioma among railroad machinists exposed to chrysotile. Amer. J. Ind. Med. 13, 639-657.

McDonald, A.D. and McDonald, J.C. (1980). Malignant mesothelioma in North America. Cancer 46, 1650-1656.

McDonald, A.D. and McDonald, J.C. (1986). Epidemiology of malignant mesothelioma. In "Asbestos-related Malignancy" (K. Antman and J. Aisner, Eds.). Grune and Stratton, Orlando, Florida.

McDonald, J.C. (1985). Health implications of environmental exposure to asbestos. Environ. Health Perspectives 62, 319-328.

McDonald, J.C. (1989). Relative risk of mesothelioma among railroad machinists exposed to chrysotile. Amer. J. Ind. Med. 14 (in press).

McDonald, J.C., Armstrong, B., Case, B., Doell, D., et al. (1989). Mesothelioma and asbestos fibre type: evidence from lung tissue analyses. Cancer 63, (in press).

McDonald, J.C., Liddell, F.D.K., Gibbs, G.W., Eyssen, G.E. and McDonald, A.D. (1980). Dust exposure and mortality in chrysotile mining, 1910-75. Brit. J. Ind. Med. 37, 11-24.

McDonald, J.C., Sébastien, P., McDonald, A.D. and Case, B.W. (1989). Epidemiological observations on mesothelioma and their implications for non-occupational exposure. In "Non-occupational Exposure to Mineral Fibres" (J. Bignon, J. Peto and R. Saracci, Eds.), pp. 420-427. IARC Scientific Publications no. 90. International Agency for Research on Cancer, Lyon.

Newhouse, M.L. and Berry, G. (1976). Predictions of mortality from mesothelial tumors in asbestos factory workers. Brit. J. Ind. Med. 33, 147-151.

Peto, J. (1980). The incidence of pleural mesothelioma in chrysotile asbestos textile workers. In "Biological Effects of Mineral Fibres" (J.C. Wagner, Ed.), pp. 703-711. International Agency for Research on Cancer, Lyon.

Peto, J. (1983). Dose and time relationships for asbestos-related cancers, and risk assessment. VDI-Berichte Nr. 475, 309-312.

Peto, J., Doll, R., Hermon, C., Binns, W., et al. (1985). Relationship of mortality to measures of environmental asbestos pollution in an asbestos textile factory. Ann. Occup. Hyg. 29, 305-355.

Peto, J., Seidman, H. and Selikoff, I.J. (1982). Mesothelioma mortality in asbestos workers: implications for models of carcinogenesis and risk assessment. Brit. J. Cancer 45, 124-135.

Sébastien, P., McDonald, J.C., McDonald, A.D., Case, B., and Harley, R. (1989). Respiratory cancer in chrysotile textile and mining industries: exposure inferences from lung analysis. Brit. J. Ind. Med. 46 (in press).

Seidman, H., Selikoff, I.J. and Gelb, S.K. (1986). Mortality experience of amosite asbestos factory workers: dose-response relationships 5 to 40 years after onset of short term work exposure. Amer J. Ind. Med. 10, 479-514.

Seidman, H., Selikoff, I.J. and Hammond, E.C. (1979). Short term asbestos work exposure and long-term observation. Ann. New York Acad. Sci. 330, 61-89.

Selikoff, I.J., Hammond, E.C. and Seidman, H. (1979). Mortality experience of insulation workers in the United States and Canada, 1943-1976. Ann. New York Acad. Sci. 330, 91-116.

Wagner, J.C. (1986). Mesothelioma and mineral fibres. In "Accomplishments in Cancer Research 1985" (J.G. Fortner and J.E. Rhoads, Eds.). Lippincott, Philadelphia.

Wagner, J.C., Newhouse, M.L., Corrin, B., Rossiter, C.E. and Griffiths, D.M. (1988). Correlation between fibre content of the lung and disease in east London asbestos factory workers. Brit. J. Ind. Med 45, 305-308.

Wagner, J.C., Sleggs, C.A. and Marchand, P. (1960). Diffuse pleural mesotheliomas and asbestos exposure in the Northwestern Cape Province. Brit. J. Ind. Med. 17, 260-271.

Table 1: PROPORTIONAL MORTALITY FROM MESOTHELIOMA IN 33 MALE ASBESTOS-EXPOSED COHORTS

Fibre type (and process)	Total deaths	Deaths from mesothelioma	Proportional mortality (%)	"Homogeneity χ^2 statistic" (df)
Anthophyllite	248	0	0	---
Chrysotile	5,062	11	0.2	3.8 (4)
Chrysotile with small proportion of amphibole - manufacture	3,925	25	0.6	2.1 (5)
- textiles	1,622	26	1.6	0.0 (1)
Talc	165	3	1.8	0.2 (1)
Tremolite	165	4	2.4	---
Amosite	871	25	2.9	6.9 (2)*
Mixed - shipyard work	1,077	31	2.9	1.0 (1)
Mixed amphibole - mining	63	3	4.8	---
Crocidolite	653	43	6.6	11.9 (2)*
Mixed - insulation work	3,055	235	7.7	4.5 (4)
Chrysotile with large proportion of amphibole	926	77	8.3	8.1 (1)*
	17,832	483	2.7	---

* Marked heterogeneity

NB: Mancuso (1988) has been excluded, partly because the data are not in suitable form, but mainly because they are not readily classifiable for the reasons cited by McDonald (1989).

Table 2: LUNG CANCER EXCESS AND MORTALITY FROM MESOTHELIOMA IN 33 MALE ASBESTOS-EXPOSED COHORTS

Fibre type (and process)	Lung cancer excess	Deaths from mesothelioma	Ratio M:LCE (a)	"Homogeneity χ^2 statistic" (df)
Anthophyllite	8.4	0	0	---
Chrysotile	99.2	11	0.11	8.9 (4)
Chrysotile with small proportion of amphibole - manufacture	88.9	25	0.28	55.1 (5)*
- textiles	30.9	26	0.84	73.2 (1)*
Talc	11.5	3	0.26	0.3 (1)
Tremolite	13.6	4	0.29	---
Amosite	145.8	25	0.17	51.3 (2)*
Mixed - shipyard work	21.8	31	1.42	10.5 (1)*
Mixed amphibole - mining	3.8	3	0.79	---
Crocidolite	29.6	43	1.45	2.1 (2)
Mixed - insulation work	454.6	235	0.52	2.1 (4)
Chrysotile with large proportion of amphibole	115.5	77	0.67	0.9 (1)
	1,023.6	483	0.47	---

(a) Ratio: (Deaths from mesothelioma)/(Lung cancer excess)

* Marked heterogeneity

Table 3: INTERVAL BETWEEN FIRST EXPOSURE AT AN AMOSITE FACTORY IN PATERSON NJ AND DEATH DUE TO MESOTHELIOMA

Interval (years)	Man-years of observation	Mesotheliomas	
		Pleural	Peritoneal
<5	---		
5-19	10,778	0	0
20-24	2,656	1	1
25-29	2,094	3	2
30-34	1,576	4	4
35-39	1,086	0	2
	18,190	8	9

Table 4: INTERVAL BETWEEN ASBESTOS EXPOSURE* IN A NOTTINGHAM GAS MASK FACTORY AND DEATH DUE TO MESOTHELIOMA

Interval (years)	Mesotheliomas	
	Pleural	Peritoneal
<15	0	0
15-19	0	2 (a)
20-24	4	0
25-29	14	3
30-34	8	1
35-39	7	3
40-44	3 (b)	0
	36	9

* Exposure was confined to the years 1940-45, and has been assumed completed before 1945.

(a) Both in 1963

(b) All three in 1986

Table 5: INTERVAL BETWEEN EXPOSURE OF WHITE MALE SOUTH AFRICAN AMPHIBOLE MINERS AND MILLERS AND DEATH DUE TO MESOTHELIOMA

Interval (years)	Pleural tumors: crocidolite	Peritoneal tumors	All other mesotheliomas
<10	2 (a)	0	0
10-14	4	0	0
15-19	3	0	5
20-24	5	2	1
24-29	3	3	1
30+	0	1	0
	17	6	7

(a) Intervals of 7.0 and 8.7 years; see text.

Table 6: NUMBERS OF MESOTHELIOMAS AND MAN-YEARS OF OBSERVATION IN VARIOUS STUDIES, WITH VALUES OF THE STUDY CONSTANT b.

Study (with fibre type)	Man-years	Number of mesotheliomas	Value of $b \times 10^8$
Factory workers (mixed)			
Newhouse & Berry (1976)	49,662	45	4.95
North American insulation workers (mixed)			
(a) Selikoff et al. (1979)	166,853	175	3.47
(b) Peto et al. (1982)			
Year of first exposure:			
pre 1921	2,470	13	1.77
1922 - 46	48,812	180	4.37
1947 - 66	131,418	43	2.00
pre 1966	182,700	236	3.37
U.S. Factory workers (amosite)			
(a)* Peto et al. (1982)	12,927	14	4.91
(b) Seidman et al. (1986)	18,190	17	4.12
Australian mines (crocidolite)			
Hobbs et al. (1980)	115,396	26	5.05
Peto et al. (1982)	56,212	26	5.15
U.K. textile factory (mainly chrysotile)			
(a) Peto (1980)	8,093	7	2.94
(b) Peto et al. (1985)	69,861	10	0.80
South African Miners (amphiboles)			
Crocidolite only	44,893	20	10.51
Amosite only	51,052	4	1.30
Mixed	12,346	6	8.17

* From extension of Seidman et al. (1979)

Table 7: MESOTHELIOMAS IN SHORT-TERM EMPLOYEES AT AN AMOSITE FACTORY IN PATERSON NJ

Length of exposure (mo)	Number of men	Cases
<1	61	0
1-2	172	0
3-5	149	0
6-8	99	2 (2.0%)
9-11	26	1 (3.8%)
12-23	125	4 (3.2%)
24 +	188	7 (3.7%)
	820	14 (1.7%)

Table 8: MESOTHELIOMA IN WHITE MALE SOUTH AFRICAN AMPHIBOLE MINERS AND MILLERS

Length of exposure (mo)	Crocidolite only		Other exposures	
	Cases	Referents	Cases	Referents
<3	0	7	0	5
3-11	0	16	4	6
12-47	5	16	1	8
48-95	1	8	1	8
96-191	6	15	0	6
192+	8	17	4	0
	20	79	10	33

Table 9: MESOTHELIOMA MORTALITY [OBSERVED (O) AND EXPECTED[+] (E) DEATHS] AMONG MEN FIRST EMPLOYED IN 1933 OR LATER IN A CHRYSOTILE TEXTILE FACTORY IN ROCHDALE.

Years since first employment	Duration of scheduled service (years)													
	Less than 1		1-4		5-9		10-19		20-29		30 or more		Total	
	O	E	O	E	O	E	O	E	O	E	O	E	O	E
0 -19	* 0	0.35	0	0.06	0	0.11	0	0.06	-	-	-	-	0	0.58
20-24	0	0.79	0	0.15	0	0.24	0	0.24	1	0.14	-	-	1	1.56
25-29	0	1.12	0	0.20	0	0.36	0	0.28	1	0.30	-	-	1	2.26
30-34	0	1.12	0	0.23	0	0.39	3	0.26	2	0.33	0	0.05	5	2.38
35-39	0	0.73	0	0.21	0	0.33	0	0.18	1	0.22	0	0.09	1	1.76
40 or more	0	0.52	1	0.19	0	0.32	1	0.13	0	0.16	0	0.13	2	1.46
All periods	0	4.63	1	1.04	0	1.75	4	1.15	5	1.15	0	0.27	10	10

[+] (E) "Expected" deaths are calculated as:-

$$(7.97)/(10^9) \times (\text{years since first employment})^{3.2} \times (\text{man-years})$$

* Doll and Peto (1985) state that one case in a man who was exposed for 4 months and died from "endothelioma of the pleura" 4 years later in 1950 was excluded from the risk calculation, as the tumor seemed unlikely to have been caused by exposure at Rochdale (see original report).

Table 10: MESOTHELIOMA IN MALE CHRYSOTILE MINERS AND MILLERS AT THETFORD MINES, QUEBEC

Accumulated exposure*	Cases+	Referents
<3	0	5
-10	0	4
-30	0	5
-100	3	6
-300	1	9
-600	2	3
600+	1	3
	7	35

* Expressed as (mpcf) × y, i.e., million particles per cubic feet times years.

\+ For the individual cases (A-G), the (net) duration of employment, the estimated average fibre exposure (f/ml) and the fibre exposure accumulated to nine years before the death of the case were:

Case	A	B	C	D	E	F	G
Duration of employment (mo)	7	105	194	206	374	432	443
Intensity of exposure (f/ml)	138	7	6	47	44	11	13
Cumulative exposure {(f/ml) × years}	80	59	99	802	1377	401	485

ECONOMIC ISSUES IN ASBESTOS CONTROL IN BUILDINGS •

DONALD N. DEWEES

Professor of Economics, Professor of Law, University of Toronto

Asbestos control programs compete for scarce funds with other programs that may improve public health. This means that we must balance the risk reductions of asbestos control with the costs of that control, choosing asbestos control policies such that the benefits are significant and the costs are not excessive. Exposures of building occupants are generally extremely low when the material is in good condition and is not being disturbed. Exposures of building workers are generally low except when their work disturbs friable asbestos materials. This means that generally removal at once does not substantially reduce exposures. Thus public healath promotion only requires the protection of building workers when their activities may disturb friable asbestos-containing materials. Furthermore, it is often far more costly to remove asbestos at once than to postpone removal until major building renovation will disturb the asbestos, or until the building is to be demolished. It is often most cost-effective to remove asbestos when major building renovation would disturb the asbestos, although even this is an extremely costly public health measure. Thus public policy should not compel immediate removal of most asbestos, nor even universal building inspection.

I. Introduction: Why Consider Economic Issues?

It is sometimes argued that economics has no place in the discussion of public health issues such as the control of asbestos in buildings. I believe that view is fundamentally flawed, because, whether it is discussed or not, it is inevitable that economics will influence actual policy-making. The United States does not have unlimited funds to spend on asbestos in buildings, nor on public health generally. People who are involved in public health do not have sufficient resources to pursue projects that would improve the health of the people of this country. As long as we have limited resources, then expenditures for asbestos abatement divert some resources from other worthwhile projects. Thus it is

essential to analyze the costs and benefits of asbestos control programs to assist in assigning an appropriate priority to that activity among the range of public health activities. I will discuss the benefits and the costs of asbestos control and present a framework within which one can evaluate the merits of asbestos work.

II. What Are the Benefits of Asbestos Control?

The benefits of asbestos control are reductions in the possible risk of mesothelioma and lung cancer, two usually fatal cancers. While there is uncertainty in the projection of risks from these two diseases, and the risk at very low exposures may in fact be zero, I will assume, as do most analysts, that the risks are proportional to the exposure to asbestos.[1] Thus estimating the benefits of asbestos control requires estimating reductions in exposure to airborne asbestos.

Consider first the occupants of asbestos-containing buildings. In 1984, the Ontario Royal Commission on Asbestos, which I served for four years as Diretor of Research, concluded that it is unusual for occupants to be exposed to more than 0.001 fibers per cubic centimeter (f/cc) of airborne asbestos[2] unless the material is damaged or being disturbed. This exposure is only modestly above outdoor levels. The Commission has been criticized for underestimating asbestos exposures, but subsequent data suggest that if there was any error, it was in over-estimating those exposures. Table 1 presents the results of some recent studies of occupant exposures in buildings with friable asbestos-containing material. The Constant (1983) study reported relatively high airborne asbestos exposures averaging 179 ng/m^3 in asbestos-containing rooms[3], and these exposures were reflected in the EPA's 1983 guidance document, the Blue Book. Subsequent data, however, have indicated that actual exposures are much lower, and in 1985, the Purple Book (EPA, 1985a) revised downward by a factor of 10 the estimate of exposures in schools. The Doll and Peto (1985), and the Burdette and Jaffrey (1986) studies in the UK concluded that average occupant exposures are well below 0.001 f/cc. Then Crump and Farrar (1987) analyzed exposure data from 61 schools that were involved in litigation over asbestos insulation, and found average exposures in the range of 0.00012 to 0.00015 f/cc, which cast further doubt on the Constant results, since many of Crump's buildings were located in the same state as those of the Constant study. 1988 brought four more studies reporting very low exposures

---

[1] See Dewees (1986, Chapter 3) for a discussion of models for predicting risks from asbestos exposure in buildings. See also EPA (1985b), Hughes and Weill (1986), and other papers in this volume.

[2] Fibers longer than 5 micrometers and with a length to width ratio of at least 3:1.

[3] This may be converted, approximately, to 0.002 f/cc.

in asbestos-containing buildings. Corn (1988, Table 1) reported that a re-analysis of the Constant data, combined with other recent schools data yield an average exposure of 0.00017 f/cc. The EPA (1988b) study of GSA buildings shows higher exposures in buildings without asbestos-containing friable material (ACFM) than in those with ACFM, an implausible result that reinforces the idea that the presence of ACFM does not significantly raise airborne asbestos fiber levels. Unfortunately, the EPA/GSA study reports a count of "structures", that is, pieces of asbestos longer than 0.5 micrometers, which are far more numerous than the traditional count of fibers longer than 5 micrometers, so the actual level cannot be compared to previous studies. Price (1988) analyzes the concentration of airborne asbestos fibers longer than 5 micrometers in these same GSA buildings, and finds average exposures in buildings with ACFM of 0.00008 f/cc. Price also reports on an update of the analysis of the school litigation data, arriving at an average exposure of 0.00022 f/cc. I conclude from all of these data that the best estimate of actual average occupant exposure in buildings containing friable asbestos material is no more than 0.0005 f/cc.

Asbestos removal does not eliminate exposure, since there is usually asbestos in the outdoor air, and since airborne asbestos is generally found in the indoor air of buildings after asbestos is removed. Some studies have found that indoor asbestos concentrations actually increase after removal, often for a considerable period of time. I will assume that removal will reduce average occupant exposures from 0.0005 f/cc to 0.00025 f/cc, recognizing that in some cases the reduction will be greater, and in others there will be no reduction, or an increase.

At these low levels, the risk of asbestos disease is extremely low. ORCA estimated that exposure for 10 years to 0.001 f/cc, which is twice the exposure that I assume, might cause a risk as great as 16 per million exposed persons. Other health models, including the EPA's model, predict lower risks, and the Hughes and Weill (1986) model predicts risks as low as 1/10th that of the ORCA model from the same exposure. These are extremely low risks compared to other risks faced by the general public. See Table 2. The ORCA risk estimate of 16 per million is 1/50 as great as the risk of a traffic fatality while commuting five miles each way to the building for 10 years. It is 1/60 as great as the risk of cancer from exposure for 7 hours per week to second hand cigarette smoke. Other risk comparisons in Table 2 emphasize how low is this risk. ORCA concluded that the risk is insignificant, yet at 0.0005 f/cc the risk would be lower still by 50%. Still, one could calculate benefits from reducing this exposure, and thus the risk, even if it is low.

While the exposure of building occupants to asbestos is generally insignificant, the exposure of building workers, including custodial, maintenance, and renovation workers who disturb friable asbestos may not be insignificant. These workers may experience elevated asbestos exposures depending upon the type of asbestos and the extent of disturbance. Studies have shown such workers experiencing as much as 0.1 to 0.2 f/cc on

average with peaks that are much higher. See Table 3. Wet removal of asbestos may cause exposures averaging 0.5 f/cc, with much greater exposures if the removal is dry. OSHA presented data in 1986 indicating relatively low worker exposures of 0.006 f/cc to 0.075 f/cc contingent on the adoption of an exposure limit of 0.2 f/cc and thus on the use of respirators where high exposures might occur. See Table 3. While these data do not provide an ideal basis for estimating the exposure of workers who spend some proportion of their time in activities that may disturb asbestos, I will assume that the average exposure of such workers in buidings containing friable asbestos material is 0.015 f/cc. Because these exposures have the potential to exceed the occupational exposure limit, it is important to adopt control programs to reduce these worker exposures.

These exposures and the estimated risks suggest that public policy should focus on protecting building workers, not occupants. If the workers are protected, the exposures of occupants will also be controlled. This conclusion was drawn by ORCA, and has been confirmed by the experience of the subsequent four years.

III. How Should We Choose Among Policies For Asbestos in Buildings?

How can we evaluate from an economic point of view, alternative programmes for dealing with asbestos in buildings? A common tool for economists to use in evaluating public policies generally is cost-benefit analysis. In cost-benefit analysis, you estimate the costs of that policy each year over a period of time, estimate the benefits of the policy, put a dollar value on those benefits, calculate the stream of those benefits over a number of years, and calculate the discounted net present value. The larger the net present value, the more attractive is the policy. If the net present value is negative, the policy is unattractive. This analysis is relatively easy to perform in the case of public projects where the benefits are in monetary terms. It is far more difficult in the case at hand because of the difficulty in assigning a dollar value to human life. Some people will argue that it is immoral even to talk about valuing life, while others argue that even if it is not immoral it is difficult to attach a value in which we would have much confidence. The controversy associated with valuing risks to human life may be avoided by backing off one step from cost-benefit analysis to what is called cost-effectiveness analysis. Cost-effectiveness analysis involves calculating the cost of the policy and calculating the physical effect but not translating the physical effect into dollar terms. In the case of asbestos in buildings, the physical effect is the reduction in exposure, which translates through the health model into a reduction in occupant and worker risks. The result of cost-effectiveness analysis is a cost per life saved or per life-year saved as a result of the policy.

Cost-effectiveness analysis allows one to compare several alternative asbestos-control policies in terms of their relative cost-effectiveness. Policies with a high cost per life saved

are not as desirable as those with a low cost per life saved. One can also compare asbestos control policies with other public health policies such as highway safety, smoking reduction campaigns, alcohol control campaigns, environmental protection programs, occupational health programs, or any one of a range of programs promoting public health, and ask what is being spent for reductions in mortality risks in these other programs. One can then compare the results among such programes to get a sense of where the return to expenditures are greatest.

Even if one does cost-effectiveness analysis instead of cost-benefit analysis there is still one important unresolved issue: discounting. How should one allow for the delay between the expenditure on a program now and the reduced risk of mortality far in the future? When benefits are valued in dollar terms, future costs and benefits should be discounted to the present, although the choice of a rate of discount has spawned a rich but inconclusive literature. However if we do not value lives in dollars, should we discount the lives themselves? Not discounting lives means that spending one million dollars now to save a life 30 years from now represents a cost of one million dollars per life saved. But 30 years from now the one million dollars, if invested wisely, would be worth several million dollars, implying that the true cost is several million dollars per life. This suggests that even lives should be discounted. I have trouble escaping that argument but on this issue, economists are seriously divided. In this paper, I will present the results both with and without discounting. To discount or not is important, since discounting for 30 years at a 5% rate reduces the benefits by 77 percent.

IV. Costs of Asbestos Control

Current regulations in both Ontario and the United States require that friable asbestos material be removed from a building before that building may be demolished. The question, then, is not <u>whether</u> the asbestos must be removed, but <u>when</u>. The three principal alternative times for removal are: at once, at the time of a major renovation of the building, or at demolition of the building. Several costs may be involved in each of these choices. Table 4 presents some examples of some data for these costs. Actual costs vary enormously from one city to another and even from one building to another, depending upon the condition of the material, the design and condition of the building, the precautions to be taken, and the state of the market for asbestos control work. If removal takes place at once, then there are costs for removing the asbestos itself, for disposing of it appropriately, and for installing a replacement insulating material. These costs are identified in the top line in Table 4. I have heard of one recent removal project that cost $50 per square foot, so anything is possible, but the costs in the table are at least within the range of costs that may be experienced in the United States today. In addition,

removal now requires moving the ocupants from the building, placing them in alternative space, and moving them back when the work is completed. Estimates of these moving and space costs are included in the table. Note that these costs are not trivial compared to the costs of the asbestos work itself. If the occupants move permanently to alternative space then the moving and space costs are reduced, but other costs may arise in trying to fill the space.

Suppose that the removal is postponed until a major renovation of the building. At this time, the occupants would move anyway, so there are no moving and space costs. The cost of removal is reduced from that experienced with removal now, since less care needs to be taken to protect the finish of the building, as this is presumably being replaced as part of the renovation. An important new category of cost is incurred, however: operations and maintenance (O&M) costs. Once friable asbestos material is discovered in a building, a program should be established to monitor the condition of the material, repair it as necessary, and to ensure that appropriate precautions are taken if work will disturb the material. Such programs are called operations and maintenance programs. Their content varies enormously depending on the building, the material, and the degree of concern of the owner, occupants, and workers. In a building where the material is in good condition and rarely disturbed, it may only be necessary to instruct custodial and maintenance workers and to provide them with protective equipment for occasional use. In a building where the material is already damaged, some clean-up and repair may be required. If the material is often disturbed by maintenance workers, then it may be necessary for them regularly to employ a set of procedures for protecting themselves and the building occupants from asbestos exposures. Requirements by OSHA for monitoring of worker exposures and maintenance of worker exposure records and health records may add considerable costs. The O&M cost data in Table 4 are derived from the EPA's economic analysis of costs of regulation presented in the EPA's 1988 report to Congress (EPA, 1988a, Appendix 5.) Dividing EPA's O&M costs for their typical federal office building and their typical commercial office building by the floor area of each yielded costs of $0.30 and $0.63 per square foot. On the assumption that these costs arose from procedures that EPA approved, and are therefore extremely cautious, I have used $0.15 and $0.45 to represent a more reasonable range of costs.[4]

These costs are presented with some reservations because actual costs are highly variable and thus difficult to represent with a single set of numbers. While I believe that these costs are reasonable, it is clear that costs may fall outside the range represented here. For this reason, the calculations that follow should be regarded as examples only. The

---

[4] Croke et al. (1988, Table 2) assume alternative O&M costs of $0.15 and $0.40 based on the same EPA data in their analysis of the impact of asbestos control costs on building values.

decision regarding the asbestos material in a particular building should be made with regard to the costs and exposures anticipated for that particular building.

Some general comments on costs are also in order. The cost of removal may change over time in response to changing regulations or changes in general practices for removal work. If procedures become far more careful, this should raise costs. If we develop more efficient and effective means of removal that protect workers and the building, then costs may fall. While falling costs in nominal terms is unlikely, it is possible that "real" costs, that is, costs after adjusting for inflation, may decline over time. This is, in fact, what we would expect with increasing experience in what is still an infant industry. For this analysis, I have assumed that costs for a given type of control are constant in real terms. This means that all costs rise at the same rate as inflation. All calculations are in constant dollars.

V. Case Study of The Timing of Removal

The policy question addressed in this analysis is the economic impact of choosing whether to remove asbestos now, at renovation, or at demolition. The question is addressed by evaluating four scenarios for each case study. In the BASE scenario, the asbestos remains in the building for its projected life. The asbestos is left in the building during removal, a practice illegal in both Ontario and the United States, and demolition workers are exposed to 1.0 f/cc. The health model predicts health risks for the demolition workers, and for occupants and workers exposed during the building's life..

In the DEMO scenario, the asbestos is removed just prior to demolition of the building. Building workers and occupants are exposed for the life of the building. Removal workers are exposed at the time of removal, but there is no exposure for demolition workers. While the asbestos remains in the building, there is an ongoing cost for an O&M program to minimize the risk of exposure for building workers and occupants. There is also the cost of removal at the time of demolition.

In the RENO scenario, the asbestos is removed prior to renovation of the building, which occurs 10 years hence. Building workers and occupants are exposed to asbestos in the air until the renovation, and removal workers are exposed at removal. There is an ongoing cost for an O&M program until removal, and there is the cost of removal itself.

In the NOW scenario, the asbestos is removed at once. There is no exposure for building workers or occupants, but there is exposure for the removal workers. There is no O&M cost, but there is an immediate cost for removal.

The present value of the costs associated with the asbestos control are calculated for each scenario, using a discount rate of 4.6 percent. Both a high and a low cost estimate are used. The expected risk of mortality is calculated by the health model for each year from now until all persons exposed in the building have died of one cause or another. The

undiscounted mortality risk is simply the sum of the risks from all years. The discounted mortality risk is the sum of the risk for each year, discounted at 4.6 percent from the projected date of mortality to the present. Thus each scenario has associated with it an overall risk and an overall cost.

The cost-effectiveness of a scenario is computed as the difference in the cost of that scenario and the next most costly scenario divided by the difference in the predicted mortality. DEMO is compared to BASE, RENO is compared to DEMO, and NOW is compared to RENO. Cost-effectiveness is expressed as the present value of the cost per life-year saved. This cost-effectiveness result is presented both using discounted risks, and using undiscounted risks. The costs are always discounted.

In an earlier publication (Dewees, 1986), I analyzed three case studies to give empirical content to the cost-effectiveness framework. There the hypothetical nature of the cases was emphasized, along with the uncertainties in the data. While data are still not ideal, it is possible to perform analyses today that are perhaps somewhat more representative than before.

The two case study buildings analyzed here are based on the EPA's Report to Congress (1988a). EPA has surveyed asbestos-containing buildings in the United States, and has compiled from that survey a description of three types of "model" buildings: federal, residential, and commercial. I have used some characteristics of the federal and the commercial building as the basis for the two cases. See Table 5. EPA has data on the average area of asbestos-containing sprayed surface insulation and of asbestos-containing pipe and boiler insulation for each building. To simplify the analysis, I have combined these into a single category of insulation, essentially treating it as if there were only surface insulation. The time until renovation is arbitrarily chosen as 10 years. EPA assumed that each building would have 30 occupants. This seems extremely low, so I used the more common assumption of one occupant per 250 square feet, yielding 168 occupants for the federal building, and 96 for the commercial building. In the absence of any information about the number of custodial and maintenance workers, I have assumed that each building has two full time workers. This is based on casual observation of staffing levels at the University of Toronto, but probably underestimates the custodial and maintenance staff in a commercial building. Table 5 also summarizes the assumed exposure of the individuals associated with the building, based on the earlier discussion of worker exposures.

Table 6 presents the results of the simulation analysis. The total risk arising from the BASE scenario for the federal building is 0.0377 life-years without discounting, or 0.0040 life-years with discounting. The discounted risk is between one-tenth and one-fifth as large as the undiscounted risk, in both cases, over all scenarios. The undiscounted risk represents an expected loss of about 14 days of life, not on average, but in total for all occupants and workers associated with the building over its 30 year life. This is an

extremely small risk, considering that it includes workers as well as occupants, but it is not surprising given the very low risks that all models predict for building occupants and workers at low levels of exposure. Removal at demolition reduces the risk to 0.0276, at renovation to 0.0112, and removal now reduces it to 0.0030. In both buildings, with and without discounting, the risk is reduced with each advance of the time of removal. The risk is insignificant in each case.

Looking at the cost figures, the present value of the BASE scenario is of course zero, since there are no expenditures on asbestos control. Removal at demolition (DEMO) imposes costs with a present value between $93,000 and $196,000 in the federal building. These costs include the cost of O&M until removal, as well as the cost of removal itself. The present value of the RENO scenario is $140,000 to $270,000 for the federal building, a considerable increase over DEMO. While there is a saving in O&M cost, removal occurs sooner, which increases its discounted cost. Finally, the NOW scenario is most costly, at $296,000 to $502,000. Here there is no O&M cost, but there is the high cost of displacing the building occupants, and the removal costs occur at once. The commercial building repeats the pattern of costs increasing as the time of removal is brought forward.

The incremental cost-effectiveness calculations are presented both with and without discounting of the risks. In the federal building, with discounted risks and the low cost scenario, DEMO costs $182 milllion per life-year saved, RENO costs $32 million per life-year, and NOW costs $110 million per life-year saved. The most cost-effective of these alternatives is removal at renovation, RENO. In fact, RENO is most cost-effective assuming either low or high costs, with or without discounting of risks, and for both buildings. Still, in comparison with other public health programs, $32 million per life-year is a terrible investment. It is often assumed that occupational and environmental programs are available that will cost at most a few million dollars per life, yet $32 million per life-year implies a cost of over $300 million per life. Removal now costs about $100 million per life-year, or over $1 billion per life saved, a truly fantastic cost. If these cases are even remotely realistic, they emphasize the waste of public funds implicit in crash programs of asbestos removal.

If we focus instead on the second set of cost-effectiveness data, in which the costs are discounted and the risks are not, the performance is much better, but still costly. RENO is again the most cost-effective, but $2.8 million per life-year is still almost $30 million per life. This is still a very expensive means of improving public health.

Interestingly, while the case study buildings are entirely different from those examined in the 1986 study, and a number of changes have been made in the cost and exposure assumptions, the results obtained here are generally consistent with the earlier results. Costs are high. Effects are small. Waiting is better than immediate removal.

The rankings for both cost and cost-effectiveness depend upon the cost of O&M prior to removal of the asbestos. One can imagine a very aggressive O&M program involving extensive initial cleaning of the building, a thorough professional inspection of all ACM every 6 months, regular scrubbing of the building, extensive precautions for every operation that brought workers in close proximity to any ACM, extensive record-keeping and worker health programs. In such a case, O&M costs would be high and the savings from delay would be small or zero. If O&M costs are low, as assumed here, the savings from delay may be 50 to 75% or more of the costs of immediate removal.

VI. Private Decisions and the Fear of Litigation

While there is no Federal requirement that asbestos be removed from commercial buildings, the real estate industry reports that there is considerable and growing removal activity in the United States. In many cases, owners are removing ACM against the advice of consultants, who advise that the material is in good condition and may be safely maintained by a modest O&M program. Several forces seem to be driving the current removal activity. First, in some buildings the poor condition of the asbestos or the frequency with which it is seriously disturbed cause removal to be the most economical solution. Second, when a major renovation occurs, it will often be less expensive to remove the asbestos than to use the appropriate precautions while leaving the asbestos in place, because of the disturbance to the material that the renovation will cause. Third, in cities where there is a high vacancy rate, tenants may demand asbestos-free space as the price of signing a lease.

Finally, and perhaps most important, both tenants and owners have expressed fear of future liability arising out of their involvement with a building that contains asbestos. Even if one believes that risks to occupants are insignificant, and that workers may be protected by reasonable precautions, many people believe that there is still a serious risk of future tort liability, perhaps involving punitive damages. Even a small chance of a multi-million lawsuit may persuade an owner to spend a million dollars to remove that risk of liability. Such fears are reasonable only if one believes that the U.S. courts will award damages that exceed the true risks associated with asbestos in buildings. I have calculated that if owners were liable for $1 million for any asbestos-related fatality in their buildings, the levels of cost and risk in these case studies would not cause owners to remove the asbestos to reduce the risk. (Dewees, 1986, p. 86.) Suppose, however, that courts began to award million dollar damages to any lung cancer victim who occupied an asbestos-containing building. While at an exposure of 0.001 f/cc the risk of an asbestos-related fatality is no more than 16 per million, the risk of a smoking-related lung cancer is on the order of 75,000 per million in a population including 50 percent smokers. The owner's

liability would be 5000 times as greate as the actual risk caused by the asbestos, if courts began to give awards to all lung cancer victims. The costs of immediate removal would be easily justified to avoid risks of liability of this magnitude. It is a sad commentary on the perception of the tort system in the United States today, and perhaps on the actual state of that system, that building owners may expend millions of dollars in the belief that courts could commit errors as large as a factor of five thousand in assigning probabilities. If owners and prospective buyers knew they would only be liable for excess cancers attributable to building-related asbestos exposure, and not for punitive damages when reasonable care had been take, the pace of removal would almost certainly slacken from its present stampede.

VII. Conclusions

The available exposure data confirm that asbestos control in buildings should be driven by protection of building workers, not of the occupants, whose risk, if any, is insignificant. Furthermore, immediate removal is often not needed to provide reasonable protection to workers and therefore to occupants. The analysis of a set of cases suggests that considerable cost reductions can be achieved by not removing asbestos at once, but by postponing removal until major renovation or demolition of the building. A cost-effectiveness analysis suggests that removal at renovation may be more cost-effective than removal at once or at demolition. Still, even removal at the optimal time results in costs per life-year saved that may be far in excess of corresponding costs for many other public health programs. Nonetheless, fear of lawsuits may lead to earlier removal.

Acknowledgements

The research reported on in this paper was funded in part by a grant from the Social Sciences and Humanities Research Council of Canada.

References

Burdette, G. J., and Jaffrey, S. A. M. T. (1986). "Airborne Asbestos Concentrations in Buildings." Ann. Occup. Hyg. 30, 185-196.

Constant, Paul C. and coauthors. (1983). Airborne Asbestos Levels in Schools, EPA Doc. No. 560/5-83-003. U.S. Environmental Protection Agency, Office of Toxic Substances, Washington, D.C.

Corn, Morton. "Issues Related to the Potential Health Hazard of Asbestos Containing Materials in Buildings". (1988). Paper presented to the Symposium on Health Aspects of Exposure to Asbestos in Buildings, Harvard University, Cambridge, Mass. Dec. 14-16.

Croke, Kevin; Mensah, Edward, Fabian, Robert, and Tolley, George. (1988). "Asbestos Removal and Treatment: Impacts on Housing and Urban Neighborhoods". Midwest Asbestos Information Center and University of Illinois at Chicago, Working Paper MAIC/UIC/02/88, Chicago.

Crump, Kenny, and Farrar, David. (1987). "Statistical Analysis of Airborne Asbestos Fiber Data Collected from Schools and Analyzed by Transmission Electron Microscope". Tab 2 in "Comments of National Gypsum Company" submitted to the US EPA in re Proposed Rule on Asbestos-Containing Materials in Schools, OPTS 62048. June 29, 1987.

Dewees, Donald N. (1986) Controlling Asbestos in Buildings. Resources for the Future, Washington, D.C.

Doll, Richard, and Julian Peto. (1985). Asbestos-Effects on Health of Exposure to Asbestos. H.M.S.O., London.

EPA. U. S. Environmental Protection Agency. Office of Toxic Substances. (1983). Guidance for Controlling Asbestos-Containing Materials in Buildings: 1983 Edition. US EPA. EPA. Doc. No. EPA-560/5-83-002. Washington, D.C.

----------. (1985a) Guidance for Controlling Asbestos-Containing Materials in Buildings: 1985 Edition. US EPA. Washington D.C. Doc. No. EPA 560/5-85-024.

----------. (1985b). Airborne Asbestos Health Assessment Update. US EPA. Doc. no. EPA-600/8-84-003F. US EPA, Research Triangle Park.

----------. (1988a). EPA Study of Asbestos-Containing Materials in Buildings: A Report to Congress. February, 1988. US EPA, Washington, D.C.

----------. (1988b). Asbestos-In-Buildings Technical Bulletin. Topic: Assessing Airborne Asbestos Levels in Buildings. US EPA. May, 1988, Washington, D.C.

----------, U.S. Department of health and Human Services. 1986. A Citizen's Guide to Radon. US EPA. Washington, D.C.

Hughes, Janet M. and Weill, Hans. (1986). "Asbestos Exposure-Quantitative Assessment of Risk". Am. Rev. of Respir. Dis. 133, 5-13.

ORCA. Ontario Royal Commission on Asbestos. (1984). Report of the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario. Queen's Printer, Toronto.

OSHA. U.S. Department of Labor. Occupational Safety and Health Administration. (1986) "29 CFR Parts 1910 and 1926; Occupational Exposure to Asbestos, Tremolite, Anathophyllite, and Actinolite; Final Rules". 51 Federal Register No. 119; Friday, June 20, 1986, pp. 22611-22790. OSHA, Washington, D.C.

Paik, Nam Won, Walcott, Richard J., and Brogan, Patricia A. (1983). "Worker Exposure to Asbestos During Removal of Sprayed Material and Renovation Activity in Buildings Containing Sprayed Material". Am. Ind. Hyg. Assoc. J. 44. June, 1983, 428-432.

Pinchin, Donald J. (1982). Asbestos in Buildings. Royal Commission on Asbestos Study Series, no. 8. Royal Commission on Asbestos, Toronto.

Price, Bertram. (1988). "Assessing Asbestos Exposure Potential in Buildings". Paper presented to the Symposium on Health Aspects of Exposure to Asbestos in Buildings, Harvard University, Cambridge, Mass. Dec. 14-16, 1988.

Wilson, Richard. (1979). "Analyzing the Daily Risks of Life." Technology Review. 41-46.

Table 1

Mean Asbestos Exposure of Building Occupants
(f/cc longer than 5 microns, unless noted)

Source, Year	Buildings, Units	Outdoors	Indoors	
			Non ACM	ACM
ORCA (1984, p. 577)	General (f/cc)	<<0.001		<0.001
Constant (1983, p. xviii)	Schools (ng/m^3)	6	53	179
EPA (1983, p. 1-5)	Schools (ng/m^3)	1-5		80-800
EPA (1985, p. 1-4)	Schools (ng/m^3)	0.1-1		1-80
Doll and Peto (1985, p. 47)	UK, General (f/cc)			<0.0005
Burdette and Jaffrey (1986)	UK, 39 Buildings (f/cc)			<<0.001
Crump and Farrar (1987, p. 8)	61 Schools (f/cc) (274 samples)			0.00012-0.00015
Corn (1988, Table 1)	71 Schools (f/cc)			0.00017
EPA (1988b, p. 4)	49 GSA Bldgs (s/cc)	0.00039	0.00099	0.00059
Price (1988)	49 GSA Bldgs (f/cc)	0.0001	0	0.00008
Price (1988)	73 schools (f/cc)	0		0.00022

Abbreviations used:

ACM	Buildings with asbestos-containing materials.
Non-ACM	Buildings without known asbestos-containing materials.
f/cc	Fibers per cubic centimeter of air.
ng/m^3	Nanograms per cubic centimeter of air.
s/cc	Structures per cubic centimeter of air, where structures are asbestos fibers, bundles, and clusters longer than 0.5 micrometers.

Table 2

Risk of Fatality from Various Activities
Lifetime Risk of Premature Death Per Million Exposed Persons

Activity	Lifetime Risk
Driving 10 miles/day for 10 years[a]	1125
Leukemia mortality from benzene exposure from self-serve gasoline stations 10 minutes/week, age 20-65[b]	4
Lung cancer from exposure to second hand cigarette smoke 7 hours/week[c]	900
Cancer from radiation from living in a brick house for 10 years[d]	50
Exposure to 0.001 f/cc of asbestos in a building for 10 years[a]	15.6
Lung cancer from living 70 years in a house with radon gas at EPA action level[e]	13,000-50,000
Lung cancer from average outdoor exposure to radon gas[e]	1,000-3,000

---

[a] ORCA (1984, P. 585)

[b] Derived from "The Report of the Advisory Council on the Designation of Benzene in Ontario", 1984.

[c] Doll and Peto (1985, p. 47).

[d] Wilson (1979, p. 182).

[e] EPA (1986).

Table 3

Asbestos Exposure of Building Workers
(f/cc)

Measured Fiber Levels Activity	Number of observations	Geometric mean (f/cc)	Maximum (f/cc)
Renovation[a]			
Carpenters	105	0.13	>2
Electricians	35	0.13	>2
Sheet-metal workers	37	0.19	>2
Painters	7	0.08	0.3
Removal of sprayed insulation			
Wet removal[b]	15	0.5	2-10
Wet removal[c]	58	0.35[d]	10.9
Dry removal[b]	79	16.4	>100

OSHA Estimated Exposures[e]	Exposure Limit 2.0 f/cc	Exposure Limit 0.2 f/cc
Asbestos removal	0.14	0.021
Demolition (asbestos removal)	0.061	0.001
Routine Building Maintenance		
Replace ceiling tiles	0.45	0.045
Repair (adjust ventilation, lighting and other work above drop ceiling)	0.31	0.006
Repair plumbing/boiler	0.18	0.018
Repair roofing	0.12	0.012
Repair drywall	0.75	0.075
Repair flooring	0.020	0.020

[a] Paik, Walcott and Brogan (1983), p. 431, table V.

[b] Paik, Walcott and Brogan (1983), p. 431, table VI.

[c] Pinchin (1982), p. 7.17.

[d] The figure presented here is estimated from Pinchin's data on arithmetic means, and some description of the distribution of individual readings.

[e] OSHA (1986, Table 23, pp. 226-65). Estimated exposures before and after the 1986 reduction in the exposure limit from 2.0 to 0.2 f/cc.

Table 4

Cost of Asbestos Control
(Dollars per square foot)

Cost Category	Low	High
Removal, disposal, replacement[a]	5.31	11.40
Removal, disposal[a]	4.14	9.64
Moving[a,b]	.63	1.25
Alternate space[a]	2.09	4.16
Operations and Maintenance[c]	.15	.45

[a] From Dewees (1986, p. 55), increased by 25% for inflation since 1984.

[b] Add 1 week lost wages per occupant for lost productivity associated with two moves.

[c] Derived from EPA (1988a), Appendix 5.

Table 5

Characteristics of Case Study Buildings

	Federal	Commercial
Floor Area (sq. ft.)	42,000	24,000
Asbestos Area (sq. ft.)	15,300	5,300
Building life (years)	30	30
Time until renovation (years)	10	10
Number of occupants	168	96
Maintenance/custodial staff	2	2
First year repair and O&M cost	$37,400	$18,300

Assumed asbestos exposure.

Occupants (Reduction if removal)	0.00025 f/cc
Maintenance, custodial workers	0.015 f/cc
Removal workers	0.10 f/cc
Demolition workers (if asbestos remains at demolition)	1.0 f/cc

Derived in part from EPA (1988a, Appendix 5).

Table 6

Costs and Cost-Effectiveness of Policies

CASE	Scenario BASE	DEMO	RENO	NOW
1. EPA Federal Building				
Undiscounted risk (L-Y)*	.0377	.0276	.0112	.0030
Discounted risk (L-Y)	.0040	.0034	.0020	.00056
Cost (Present value $000)				
Low	0	93	140	296
High	0	196	270	502
Incremental Cost-effectiveness				
($ millions per life-year)	DEMO-BASE	RENO-DEMO	NOW-RENO	
Risks Discounted				
Low cost	182	32	110	
High cost	383	51	164	
Risks Not Discounted				
Low cost	9.3	2.8	19	
High cost	19.	4.5	28	

	BASE	DEMO	RENO	NOW
2. EPA Commercial Building				
Undiscounted Risk (L-Y)	.0230	.0195	.0072	.0010
Discounted Risk (L-Y)	.0027	.0025	.0013	.0002
Cost (present value $000)				
Low	0	38	60	151
High	0	73	112	248
Incremental Cost-effectiveness				
($ millions per life-year)	DEMO-BASE	RENO-DEMO	NOW-RENO	
Risks Discounted				
Low Cost	212	19	80	
High Cost	413	33	119	
Risks Not Discounted				
Low Cost	11	1.8	15	
High Cost	21	3.1	22	

* L-Y = life-years

FIBER TYPE AND MESOTHELIOMA RISK

ARTHUR M. LANGER and ROBERT P. NOLAN

Environmental Sciences Laboratory, An Affiliate of the Institute of Applied Sciences, Brooklyn College of the City University of New York, Brooklyn, New York 11210

The principal conclusion of this paper is that the quantitative risk of mesothelioma currently used by the U.S. Environmental Protection Agency, calculated for passive occupants of buildings in which asbestos-containing materials are present, is too high by at least one, and possibly two, orders of magnitude. Two facts are at the center of this thesis; one is that the different asbestos fiber types produce mesothelioma to different degrees; the other is that the currently used risk value for mesothelioma is based on the more carcinogenic of these fiber types.

We present data in support of the following: for the first group of facts commercial amphibole asbestos minerals, amosite and crocidolite, have produced the majority of malignant mesotheliomas in occupationally exposed workers; peritoneal mesothelioma associated with asbestos exposure occurs only after exposure to the amphibole asbestos varieties with sufficient intensity to produce asbestosis in the same individual; the absolute carcinogenicity of the amphibole asbestos minerals is greater than for chrysotile, and the higher absolute numbers of mesotheliomas after exposure to the former group is related to the properties of the fibers and not just relative dustiness of products; for the second group of facts the bulk of the cohort data derived from US workers are based on amphibole exposures; the Cape Province environmental mesothelioma experience was associated with exposure to crocidolite. Exposures were probably both episodic and intense; environmental bystander and household mesothelioma associated with shipyard sources of fiber have likely resulted from exposure to crocidolite in Europe and mixed fibers in the United States; the bulk of US buildings contain chrysotile-based products and passive occupants are exposed to almost ambient levels of chrysotile.

On the basis of these data sets, we conclude that quantitative mesothelioma risk for passive building occupants should be recalculated on the basis of fiber type, i.e., with data available for chrysotile-exposed workers.

INTRODUCTION

The Popular Concern About Asbestos

We define passive occupants of buildings as those persons not engaged in maintenance or similar work-related activities, but who rather use or occupy the interior spaces of buildings for other purposes. Asbestos-containing materials may be present in these structures and under some circumstances passive occupants may inhale some amount of fiber. There are concerns that these persons incur some important risk of developing malignant mesothelioma as a result of this exposure. The basis for this concern rests upon data sets, qualitative observations, and unsupported deductions there from. Briefly, they are as follows:

- Almost all cohorts of asbestos-exposed workers have shown some mesothelioma mortality to greater or lesser degree;

- All major commercial varieties of asbestos fiber marketed in the United States, i.e., chrysotile, amosite, crocidolite, are considered potential agents in the etiology of malignant mesothelioma, based upon both human and experimental animal data;

- Mesothelioma is known to occur in working populations which are not "asbestos workers" per se, e.g., workers in shipyards engaged in trades which share the same workplace environment with workers who are in the asbestos trades. It is thought that the bystander exposure to fiber in these instances is less than in the primary occupational setting, although the precise nature of these exposures (both in their magnitude [number of fibers per milliliter of air, or f/ml] or fiber type, length, width, and aspect ratio of fiber), is generally unknown;

- Some mesothelioma is known to occur in individuals (especially offspring and siblings) who live in the households of asbestos-exposed workers. Here the vector is thought to be primarily dusty clothes brought home from the

workplace by the worker, and some people consider that the exposure (dose) is perhaps even less intense than in the bystander situation;

- Reports have entered the literature which state that persons who lived within some short distance of the center of an asbestos-related work activity (factory, plant, mine, mill, shipyard, etc.), but had no direct participation in its activities, nor lived with a worker, developed mesothelioma "in the non-occupational setting". In this instance, both vector and dose are generally unknown as well;

- Pleural mesothelioma is known to occur in asbestos-exposed persons who have minimal-to-no parenchymal scarring and few-to-no light-visible asbestos bodies in their pulmonary tissues. Negative chest radiographs are found among these persons as well. This, in concert with "environmental" and "bystander" mesothelioma, carries the impression that very low exposures, possibly even ambient exposure to asbestos, represent a sufficient dose to produce mesothelioma. At the very least, the amount of fiber required to induce pleural mesothelioma is significantly less than is required to induce, chest x-ray evidence of asbestosis;

- Measurement of fiber in building air indicates levels of exposure commonly on the order of 0.001-0.0001 fiber/ml, a value near those observed out of doors and remote from any obvious asbestos sources. Although these values are obtained by transmission electron microscopy, they are frequently compared directly against fiber values obtained by phase contrast microscopy in the workplace environment. A linear extrapolation is made along the derived dose-response curve to yield health risk estimates for the non-occupationally exposed. This linear extrapolation, without a biological threshold, is thought a valid model for estimation of dose and attendant mesothelioma risk for passive building occupants;

- Mesothelioma risk is time-dependent in that risk increases with time from onset of exposure by some exponential power, k, variably estimated to be between 3 and 4. This has been demonstrated only for times greater than 15 years from first exposure. Observed mesothelioma mortality rates occurring in less than 15 years from initial exposure fall below predicted values calculated by the formula (Peto et al, 1982);

- Because of the time-dependence factor in the calculated mesothelioma risk, derived from a general carcinogenesis model, bt^k, it is shown that, all other factors equal, younger persons incur much greater risk of developing mesothelioma by virtue of their longer life expectancies. The inescapable conclusion here is that children who may be exposed today to fiber as passive occupants of school buildings are at unusually greater risk according to the mathematical formulation of the risk model;

- Few occupational cohorts have ever shown a zero effect after some defined exposure to fiber. It is generally held that every unit of exposure is associated with a unit of increased risk, that this relationship is linear, and that there is no threshold below which there is no disease. The role of other agents, e.g., cigarette smoking, on fiber elimination is never factored into these models;

- The assertion has been made that all asbestos fiber types have equal biological potency in the human, and that all fibers are capable of inducing malignant mesothelioma to the same degree. Therefore, the ability of chrysotile to induce mesothelioma is the same as for crocidolite fiber, which originated from the Kuruman Hills of South Africa. It follows from this assertion that passive building occupants -- who are most often exposed to chrysotile -- are at similar risk of developing mesothelioma, as, for example, persons who lived in the Kuruman region of South Africa and had the same magnitude of exposure;

- If the above is correct, it follows that data obtained from any cohort of asbestos-exposed workers could be used to provide the basis for time dependence, dose response, and components of the model which is used in the quantitative mesothelioma risk calculation;

- Because of the assumption that all fibers have the same carcinogenic potency, and as chrysotile asbestos has been consumed in the United States in a tonnage ratio of 19:1 to the amphibole fibers, then the bulk of the asbestos-related human diseases we observe are chrysotile-exposure related. Buildings with chrysotile-containing products are as dangerous as those with crocidolite-containing products, all other factors being equal.

Based on these data and extrapolations, passive building occupants who inhaled minute quantities of chrysotile asbestos are, therefore, claimed to be at some elevated and measurable risk of developing malignant mesothelioma which is socially unacceptable.

The Focus of This Paper

The focus of this communication will be an evaluation of existing data concerning fiber type and mesothelioma. We intend to show that:

- The assumptions in the introduction are contrary to the data and will show that the commercially used amphibole asbestos varieties amosite and crocidolite account for the majority of mesotheliomas observed among the workers where asbestos fiber is the implicated etiological agent;

- Most mesotheliomas found in "chrysotile-only" exposed cohorts are actually amphibole-related in etiology, based on the direct evidence of tissue burden studies and some related geological and clinical data;

- US workers are primarily amphibole-exposed, and their health experience is likewise amphibole-related as well. Data obtained from these cohorts may be used to calculate amphibole asbestos or mixed fibrous dust mesothelioma risk;

- Careful examination of studies which focus on both bystander and environmental mesothelioma suggests that the assertions made regarding low-level or "ambient" exposure may be seriously inaccurate;

- The asbestos fiber to which passive building occupants are exposed is primarily chrysotile;

- Based on all the available cohort data relating to fiber type, the calculation of risk of mesothelioma to passive building occupants is high by at least one, and possibly by two orders of magnitude.

OCCUPATIONAL EXPOSURE TO THE DIFFERENT ASBESTOS FIBER TYPES AND MESOTHELIOMA MORTALITY

The Cohorts

One of the building stones upon which the foundation of the conventional risk assessment rests is the assertion that all of the major asbestos fiber types, i.e., chrysotile, amosite, and crocidolite, are equally capable of producing mesothelioma in exposed workers. This assertion was examined by review of cohort data produced in cohort studies around the world.

Data extracted from Doll and Peto's 1985 report to the British Health and Safety Executive on the then status of asbestos health effects were used in this evaluation. Data for selected chrysotile cohorts are given in Table 1; data for selected amphibole cohorts are given in Table 2. The total deaths shown for each cohort is due to malignancy only. The absolute number of mesotheliomas which occurred in each cohort, and their proportion expressed as percentage of malignant deaths, are shown. These data are pooled and an expression of the proportion of the malignant death due to mesothelioma is given for each group.

Pooled data show mesothelioma mortality among those exposed to chrysotile is about 2.21% of the total malignancy deaths, as compared to 11.36% for those exposed to the amphibole asbestos (Tables 1 and 2; corresponding ranges are also given). The corresponding standard errors of the percentages were calculated as 0.34% for the chrysotile data and 0.71% for the amphibole and mixed fiber data. Tests of the difference between these percentages, for those populations, show them to be significant. If the true difference between these numbers was zero, the chance occurrence of obtaining these statistics is less than one in 100.

Many of the important drawbacks associated with pooled data are recognized, e.g., diagnosis of both pleural and peritoneal mesothelioma may not be equally good in all countries; cumulative dust exposures, latency (lapsed periods), and the nature of the age distributions within the cohorts may not be similar, etc. Although pooled data necessarily incorporate these shortcomings, the number of separate studies and deaths are so large, we consider the data biases reduced in strength.

The total number of malignancy deaths for the chrysotile cohorts is 1,761 and for the amphibole- and mixed-fiber exposures (amphibole and chrysotile) 2,007 (Tables 1 and 2). If the mesothelioma deaths in the amphibole cohorts are normalized downward to match the total malignancy deaths in the chrysotile base, 1,761/2,007 x 228, 200 mesotheliomas would have occurred. This normalized mortality still represents a considerable excess absolute number of mesothelioma deaths in the amphibole-exposed group, corresponding to greater than five-fold more when compared with the chrysotile

group (200:39, or 5.13:1). Doll and Peto (1985) estimated a similar 5-fold differential in mesothelioma risk for the fiber type.

The Chrysotile Mesotheliomas

The greatest proportion of chrysotile-associated mesotheliomas occur in two cohorts: the Rochdale textile workers in the Midlands of England, reported on most recently by Peto et al (1985), and a British friction product manufacturing plant (Ferodo), reported on by Berry and Newhouse (1983). These two facilities account for 25/39, or slightly more than 64%, of the total chrysotile-associated mesotheliomas reported (Table 1). However, study of the parenchymal lung tissues obtained from these persons for mineral fiber, by analytical electron microscopy, has shown that all 17 of the workers who died of mesothelioma at Rochdale have elevated concentrations of crocidolite (± amosite) present in their pulmonary tissues. These concentrations are far greater than amounts found in the tissues of their workmates who died of other causes. Crocidolite is the suspect agent in these cases. This has also been found for the Ferodo mesothelioma deaths as well (Pooley, 1988, personal communication).

If we consider that these mesothelioma deaths are crocidolite-related, then reclassification of these 25 cases would permit their removal from the chrysotile pooled data. Recalculation of the proportion of mesothelioma deaths in the corrected pooled data yields: (39-25)/(1,761-25) = 14/1,736 = 0.81%. This corrected mortality figure produces more than a 16-fold difference in the proportion of mesothelioma deaths associated with amphibole exposure as compared to "pure" chrysotile exposure (228:14 or 16.29:1). Normalized data reduces the proportion slightly (200:14 or 14.29:1). It may be argued that the data may be further manipulated to yield even higher differentials (228+25:14 = 253:14 = 18:1).

The crucial point here is that the chrysotile content of the lungs in the Rochdale and Ferodo mesothelioma cases were high, but crocidolite, in concentrations greater than trace, were found only in those workers dying with mesothelioma. The mesothelioma disease process, at least for the Rochdale and Ferodo cohorts, appears to require the presence of crocidolite.

Crocidolite Exposure at Rochdale?

Peto et al (1985) appended a description of the fibers used in the various buildings at the Rochdale facility. Although Canadian and Rhodesian chrysotile fibers were the predominant fibers used (over one million tons) between 1932 and 1969, "small amounts of crocidolite were used", averaging about 279 tons per year. During this time, Turner

Brothers directly purchased 3,702 tons of crocidolite and obtained, from secondary sources, an additional 6,620 tons of crocidolite. In total, over the 37 years, 10,322 tons of crocidolite fiber passed through the Rochdale works, representing 2.6% of the total asbestos used in plant.

To appreciate the amount of crocidolite which was processed here, and its potential to produce disease, one must compare these figures with the amounts of crocidolite found in the lungs of persons who die with mesothelioma: $\sim 43 \times 10^6$ fibers/gram of dried lung tissue, as compared to $\sim 3 \times 10^6$ fibers/gram in controls (Pooley and Mitha, 1987a). Re-calculation of fiber number to mass yields ~ 15 μg/gram in dried lung tissue, as compared to ~ 1 μg/gram in controls (Pooley and Mitha, 1987a). The conversion of 10 grams of wet lung tissue to 1 gram of dried lung tissue brings the crocidolite levels, in persons dying with mesothelioma, to ~ 1.5 μg/gram of wet lung tissue. A lifetime dose of tens of milligrams of crocidolite dust may be sufficient to produce mesothelioma in humans. Hence, we conclude that there were ample amounts of crocidolite used at the Rochdale facility to provide sufficient exposure to produce the mesotheliomas observed. This is further supported by the observation that the mesotheliomas occured only in workers who carded, spun, and worked with crocidolite.

The above exercise has not been carried out for the Ferodo facility as yet, although it is known that "small amounts" of crocidolite were used there for a brief time period as well (Berry, 1987).

The Canadian Experience

There is further amplification and clarification required for the Canadian mesothelioma data as well. These mesotheliomas, reported by McDonald et al (1980), account for about 25% of the total number of mesotheliomas found in all the chrysotile cohorts. Although these miners and millers were working chrysotile ore, some may have been exposed to asbestiform tremolite, found as a gangue mineral in association with some horizons (Churg, 1987). Some of the pits in the Thetford region of Canada contain much more tremolite fiber than is found elsewhere in the Eastern Townships (Sebastien, personal communication, 1989).

McDonald and Liddell (1979) showed that of the 243 excess deaths (4,350 OBS/4,107 EXP), among chrysotile miners and millers, which occurred in the eastern townships between the years 1926 and 1945, 34 occurred in the Asbestos area (the Jeffrey Mine and mill), whereas 209 occurred in the Thetford group of pits some 40-50 Km to the east. Both areas contributed about equal numbers of deaths to their study. The lung cancer SMRs calculated for each of these areas was 1.02, with two pleural mesotheliomas, for Asbestos, Quebec, and 1.10, with ten pleural mesotheliomas, for Thetford, Quebec.

Thetford was noted 25 years ago for its unusual prevalence of both pleural plaques and mesotheliomas (Cartier, 1964). At a meeting in Europe in 1964, Cartier, then Director of the Thetford Clinic, reported he had seen 3 mesotheliomas in the miners and millers of the Thetford area the previous year. No incidence data were available to him in that only some 16% of the mining population came to autopsy. However, in the discussion of his presentation which followed, it was asked why he observed pleural plaques and mesothelioma at Thetford, whereas in Asbestos, Quebec, there were no similar findings. After all, it was said, both are chrysotile mining areas. Were the minerals different? Cartier responded that he believed there was good correlation between intensity and duration of chrysotile exposure and x-ray evidence of asbestosis. Therefore, his conclusion was that the agent for asbestosis was clearly chrysotile. However, plaques occurred in persons with no correlation with chrysotile exposure history, nor with x-ray evidence of parenchymal scarring (asbestosis). His conclusion was that he didn't think chrysotile itself was the agent responsible for the plaque development, nor did he think it was the agent for the malignant tumors. Although tremolite was not specifically alluded to then as a possible agent of disease, the pattern of disease observed supported the thesis that an occult agent was present.

The importance of tremolite-chrysotile exposure has been raised in a number of papers in a recent symposium on the biological effects of chrysotile: Langer and Nolan (1987); Pooley and Mitha (1987a); Sebastien et al (1987); Churg (1987); Green et al (1987); Wagner et al (1987). Tremolite fiber, as a contaminant of chrysotile ore, emerges as an important suspect agent in the etiology of chrysotile-associated asbestos diseases, especially pleural disease.

These observations are stressed because the mesotheliomas observed in the miners and millers of Quebec may be associated with exposure, in specific areas, to the presence of fibrous tremolite and not to chrysotile. The assertion we make is that if the tremolite contaminant is the agent of pleural disease then it follows that very much higher concentrations of chrysotile dust are required to deliver a biologically important tremolite dose to exposed persons.

Lung Cancer:Mesothelioma Ratio: Does Dose Play a Role?

It has been suggested that perhaps the nature of the chrysotile fiber extracted from ore, and within fabricated products, is such that it universally produces less dust than comparable manipulation of amphibole fiber ore and products. As supporting evidence for this hypothesis, although differences are noted in absolute numbers of mesotheliomas, their relative proportion to another malignancy, i.e., mesothelioma to lung cancer, is about the

same. Therefore, one may explain the observed pattern to dose differential rather than to differences in fiber carcinogenicity. The pooled data ratios are given in Table 3.

The pooled chrysotile cohort data show 660 lung cancer deaths occurred, with an accompanying SMR of 1.25, yielding 132 excess lung cancers in the pooled population. The ratio of excess lung cancers to total mesothelioma (based on uncorrected data) is 132:39, or 3.38:1. For the amphibole cohorts, the total for lung cancer deaths is 866, with an accompanying SMR of 2.86, yielding 563 excess lung cancer deaths in this pooled population. The ratio of excess lung cancers to total mesothelioma is 563:228, or 2.47:1.

Although the absolute number of malignancies is considerably higher for the amphiboles, the ratios are similar for both groups. However, when modifying the raw data, to reflect lung burden studies, the lung cancer:mesothelioma ratios change dramatically:

Chrysotile: 132:39-25 = 132:14 = 9.43:1

(neglecting tremolite exposure in Canadian chrysotile workers)

Amphibole: 563:228 = 2.47:1[1]

Neglecting the tremolite confounder in Canada, amphibole exposure clearly produces more mesothelioma than is associated with chrysotile exposure alone. Both the absolute number of mesotheliomas and lung cancers are strikingly different when comparing the asbestos-exposed cohorts. The dose differential may be true, but because few data support its validity, fiber type must be considered the singularly important factor in producing the differences.

Other Cancers

We note that the relationship of excess lung cancer with excess gastrointestinal cancer observed by Doll and Peto (1985) holds for these pooled data: about four to five excess lung cancers for one excess gastrointestinal cancer (gastrointestinal excess is ~14-17% excess lung). Although the absolute numbers of excess tumors in the amphibole cohorts are greater than in the chrysotile cohorts (Table 3), the strength of the association is still weak (SMRs < 1.5).

---

[1] If the 25 crocidolite-associated mesotheliomas in the "chrysotile-exposed" cohorts are put in the amphibole group, then the ratio becomes 563:228 + 25 = 563:253 = 2.23:1. This further widens the differential.

Pleural and Peritoneal Mesotheliomas

The Mount Sinai cohorts (Tables 2 and 4) show that greater numbers of peritoneal mesotheliomas have occurred as compared to pleural mesotheliomas (noted by Peto et al, 1982). Mesothelioma mortality data, associated with inhalation of asbestos fiber in the work place, includes both pleural and peritoneal sites. However, peritoneal mesothelioma almost universally occurs in workers exposed to concentrations of amphibole-containing dusts sufficiently intense to have produced radiological evidence of asbestosis as well (see Fig. 1; Table 4). There is only one case (a suspected case) of peritoneal mesothelioma in a chrysotile-exposed worker reported in the world literature (see Dement et al, 1983). Chrysotile's limited stability in an acid environment, and highly sorptive surface character, both mitigate against biological activity in the GI tract and gut.

These unusual ratios of pleural:peritoneal mesothelioma in specific cohorts were discussed in Peto et al (1982). The investigators suggested that both fiber type and dimension played a role in the appearance of the peritoneal form of the disease. Here we stress that exposures sufficient to produce asbestosis may also be an important prognosticator for risk. It should be noted that asbestotics have been shown to produce numbers of uncoated fibers in their sputum. Swallowing long, acid-stable amphibole fiber would appear to be a plausible mechanism for getting agents to the target tissues.

We conclude, based on evaluation of the world literature, that peritoneal mesothelioma risk following chrysotile exposure in buildings appears to be small to nil: fiber type, dimension and dose, all major risk factors for its appearance, are either not present or greatly reduced.

Berry's Review of Cohort Data

Berry (1987) compared data obtained on cohorts of workers exposed to chrysotile alone[2] and cohorts exposed to either amphiboles alone or mixtures of fibers (amphibole plus chrysotile). Of 5,348 deaths observed for workers exposed to chrysotile alone, 14 mesotheliomas were found, yielding a mortality of 0.26%. This compares with values of 5.12% for crocidolite alone (49/957) and 5.27% (228/4323) for amphibole-chrysotile exposure. These latter two account for 277/5280, or a 5.25% mesothelioma mortality. Berry's comparisons, also based on pooled proportional mortality data, suggest a more than 20-fold greater mesothelioma mortality associated with amphibole exposure.

---

[2] Meaning exposure to the amphibole minerals were not previously known.

Comparison of Cohort Experiences as Determined by Similar Study Design

The argument has been raised that study designs are so significantly different that some epidemiological internal error, both large and consistent, drives the observed mesothelioma mortality differential between chrysotile- and amphibole-exposed workers. Two significant factors often cited are latency and accurate ascertainment of cause of death. Table 4 shows data obtained from four studies, all of which were carried out by the Mount Sinai group. All four studies are based on the experience of workers who had more than 20 years of time elapse between onset of exposure to asbestos and their deaths; had their death certificate cause of death verified or changed based on review of pathological materials. Their data are given in Table 4.

Several features are noteworthy. Although the absolute number of deaths due both to total malignancy and mesothelioma are low in the Thetford chrysotile cohort, if the total malignancy deaths, and proportion of mesothelioma deaths, are upwardly normalized in proportion with the insulator (New York-New Jersey) experience, then nine mesothelioma deaths should have occurred in the cohort. The ratio of excess lung cancer to mesothelioma reflects this marked disparity (compare 1.78 for the insulators to 17.00 for the Thetford workers; Table 4). The Sinai chrysotile cohort shows fewer absolute numbers of mesotheliomas, and the lowest proportional mortality from mesothelioma, as compared to the other cohorts. Nicholson and colleagues (1979) suggested these differences were brought about by dimensional characteristics of the fiber.

There appears to be a relationship between the proportion of mesothelioma deaths which are peritoneal and the percentage of deaths in the cohort due to asbestosis. This is true for the amphibole cohorts (1, 2, 3 in Table 4) as well as for the Thetford cohort, which did not manifest the peritoneal form (Fig. 1).

Given the similarity of study design, the amphibole- and mixed fiber-exposed cohorts experienced far greater mesothelioma mortality as compared to the chrysotile cohort. These data conform to the world pattern produced by pooling mortality studies of cohorts elsewhere.

General Conclusions

The proportion of deaths due to mesothelioma is greater in the cohorts of workers exposed to amphibole asbestos and amphibole-chrysotile mixtures of asbestos in the workplace as compared to the cohorts of workers exposed to chrysotile alone.

Almost two-thirds of the mesotheliomas found among the "chrysotile" cohorts occur in the Rochdale and Ferodo populations. Tissue burden studies show these workers were also exposed to amphiboles, especially to crocidolite. The pooled mesothelioma mortality

for chrysotile is further reduced by recalculation of the data to reflect the tissue burden findings.

The mesotheliomas reported in the Canadian chrysotile industry are concentrated in the Thetford area of Quebec. It has been suggested that some pits and mines in this area are contaminated with asbestiform tremolite, and that this fiber is the agent for mesothelioma induction in the workers there. If amphibole fiber contaminant in chrysotile ore is considered the agent of mesothelioma induction, then mesothelioma risk is reduced some 50-fold for pure chrysotile fiber exposure.

The ratio of excess lung cancer deaths to mesothelioma changes dramatically when the individual amphibole-exposed cases are removed from the chrysotile pooled data. The notion that the greater absolute numbers of tumors in the amphibole cohorts is due to difference in dust exposure alone is rejected. These differences may be explained on the basis of absolute carcinogenicity of the two materials. Greater lung cancer and gastrointestinal cancer mortality in the amphibole cohorts support this conclusion.

Studies of cohorts of workers exposed to different fiber types, of identical design, confirm the pooled data conclusions. Amphibole- and mixed-fiber exposures produce more mesothelioma than chrysotile alone. The risk to chrysotile-exposed persons is over-estimated by 20- to 50-fold when calculations based on amphibole- and mixed fiber-exposed populations are used.

ASBESTOS FIBERS IN THE LUNGS OF PERSONS EXPOSED TO ASBESTOS IN THE UNITED STATES

Another portion of the foundation upon which the fiber-type component of risk assessment rests is that of fiber consumption and disease prevalence. It has been thought that because chrysotile asbestos is the major fiber consumed industrially in the United States, that consumption data may be used as an index of worker exposure. If fibers are all the same in their ability to cause cancer, this leads to the following deductions:

> Chrysotile is the most prevalent fiber used industrially, accounting for 95% or more of the asbestos consumption; it must therefore account for 95% of the malignant asbestos-induced diseases observed.

This deduction has led to further extrapolations such as: the insulation worker data can be used for mesothelioma risk modeling because these workers were exposed primarily to chrysotile, with minor amounts of amphiboles; crocidolite exposure in the United States

has been minimal and insulation workers have never been exposed to crocidolite (Selikoff et al, 1970; Nicholson et al, 1979).

A tissue burden study of US workers was undertaken to determine the validity of these assumptions (Langer and Nolan, 1989).

Selection of Cases

The 54 cases evaluated in this study came to our laboratory as referrals. Verification of tumor type (especially mesothelioma) and tissue burden study (fiber type and amount) were the two most frequent inquiries. Occupational asbestos exposure (and domestic contact in one case) was established for all the cases studied. The primary causes of death among the 54 cases show a strong selection bias: mesothelioma, 42.6%; lung cancer, 37.0%; asbestosis, 16.7%; "other causes", 3.7%.

Occupational Categories

The 54 cases were grouped into three major occupational categories on the following basis:

1) Insulation worker: if ever in the insulation trade; no history of shipyard work; with or without experience in other dusty trades (e.g., construction trades). N=16.

2) Shipyard worker: if ever in shipyard environment regardless of trade (e.g., electrician, machinist, insulator, etc.). N=15.

3) Other trades: never in insulation work, never in shipyard environment. Includes brake repair, plasterer, railroad worker, rubber worker, construction trades, etc., and one household exposure. N=23.

The rationale for these categories, and their selection criteria, is based on the perception that insulation work involves exposure to mixed fiber types (at least some amosite and primarily chrysotile); that shipyard work involves exposure to amosite and may carry the greatest potential for crocidolite fiber exposure;[3] "other trades" include workplaces where exposure to primarily chrysotile-containing products was thought to occur.

---

[3] Crocidolite resists corrosion in salt water better than any other fiber type.

Materials Studied

Over 80% of the cases had formalin-fixed bulk pulmonary tissues available for study. The remaining specimens were available from histological blocks. The recovery of the inorganic dust was carried out on tissues ranging in mass from 20 to 5000 mg by the alkali digestion method (Langer and McCaughey, 1982).

Analytical Protocol

The analytical protocol for tissue study is given in Langer and Nolan (1989). Results are expressed as fibers per gram (f/gm) of dry lung tissue. Only data for fibers greater than 1 μm in length are shown (Tables 5, 6, 7).

Findings

The three major fibers used in commerce in the United States, amosite, chrysotile and crocidolite, are found in the pulmonary tissues of US asbestos-exposed workers (Table 5). Amosite appears to be the most prevalent fiber type in the lungs of American workers, occurring in 74% of the tissues. Almost 25% of all workers have been exposed to crocidolite. Tremolite, actinolite and anthophyllite are also frequently found in pulmonary tissues. Amphibole fibers have been observed in 42 of 54 (~78%) of pulmonary specimens examined.

Amosite and chrysotile are observed to occur as the sole commercial fiber with equal frequency. Crocidolite only rarely occurs as the sole fiber type in the lung (Table 5). Tremolite, actinolite and anthophyllite fibers were always present with one of the commercial fiber types. Anthophyllite and actinolite tend to occur with amosite exposure and tremolite with chrysotile exposure.

The lungs of insulation workers contain statistically greater concentrations of total fiber as compared with those of shipyard workers (Table 6). Although amosite is used in shipyard settings, the lungs of insulation workers, without known shipyard work history, contain greater concentrations. Crocidolite was found in 19% of the lungs of insulation workers. Chrysotile fiber concentrations are high in all trades. Crocidolite burden in the various occupational groups show little difference between insulation workers and other trades, but shipyard workers' lungs contained the highest prevalence and concentrations of this fiber. If insulation work in shipyards is neglected, insulation workers' lungs contain, statistically, the lowest concentrations of crocidolite.

Insulators who have ever worked in shipyards may be at some unusually high risk of asbestos-induced malignancies, especially mesothelioma. We note that Selikoff et al (1971)

queried 337 insulation workers, members of Locals 12 and 32 in New York and New Jersey, concerning their shipyard work experience. The question asked was "Did you ever work in a shipyard?". Their responses were: "Yes, but less than two years" (N=118, 35.0%); "Yes, more than two years" (N-121, 35.9%). Only 98 (29.1%) said they never worked in a shipyard. The mesothelioma mortality experience of the two locals is worse (higher percentage of mesothelioma among deaths due to malignancy; smaller lung cancer to mesothelioma ratio) than observed for the union's international membership (Table 4). We assume that members of Locals 12 and 32 had greater opportunity for shipyard work than their brothers in e.g., Kansas or Iowa. We would therefore conclude that their opportunity for crocidolite exposure was similarly greater and that their mesothelioma mortality differential may be explained by fiber type exposure.

The lungs of insulation workers always contain amosite and may also contain chrysotile and/or crocidolite. No other occupational category shows a major commercial fiber as universally present. We note that the prevalence of crocidolite fiber is greatest in shipyard workers, this type of fiber being found in some 40% of these workers (Table 7).

From these observations one may conclude that U.S. insulation workers and shipyard workers, are crocidolite and amosite exposed, and that fiber consumption data are poor indices of exposure, at least for these occupations.

Fiber Type and Risk Assessment

Although 95% of the fiber used in the United States historically has been chrysotile, the two other commercially used fibers, amosite and crocidolite, are commonly found in the lungs of the persons we studied. Chrysotile fiber was anticipated, but amosite occurrence is very much greater, by a factor of more than 25-fold, than would be expected from its US consumption data.

Only about 2% of the fiber used during this same period was crocidolite, yet almost 25% of the workers studied had crocidolite in their pulmonary tissues. It has been found in 40% of the shipyard workers examined. This is nearly 20-fold greater than could be expected from its commercial consumption in the United States. Shipyard work is known to provide significant opportunities for crocidolite exposure, and therefore an appreciable mesothelioma risk for any trade is associated with work in and around shipyard environments.

The same general statements may hold for insulation workers whose lungs contain amosite. Amosite, which accounted for only 3% of fiber used in commerce during this period, is universally present (100%) in the lungs of insulation workers. This is more than

30-fold greater than could be expected from the fiber consumption data in the United States. Amosite was very commonly used in insulating products.

In some instances, however, fiber burden is consistent with patterns anticipated for product composition and use. We note that chrysotile occurrence alone, as the sole major fiber in pulmonary tissues, tends to be associated with specific trades (e.g., brake repair).

Patterns of fiber burden in tissues need not reflect fiber consumption in that much chrysotile may be used in products which are neither dusty nor produce significant amounts of dust when installed, used, or removed (e.g., vinyl-asbestos floor tiles). Ninety-five percent of fiber consumption need not produce 95% of the dust exposure in the workplace. On the other hand, small percentages of amphibole-containing products installed and manipulated in poorly ventilated or uncontrolled, dusty environments, e.g., aboard ships during repair, especially involving ripouts, may be a source of a disproportionately large amount of dust and subsequent exposure to the worker. Most asbestos-associated risk may arise as the result of the convergence of these factors.

The insulation worker experience is associated most commonly with amphibole exposure, and is not chrysotile-dominated, as has been implied in the past.

One of the most important conclusions to be drawn from the tissue burden study is that the use of specific cohort data, e.g., the insulation worker studies, to calculate risk for mesothelioma in the general population may lead to a gross inaccuracy. The insulation trade cohorts are largely amphibole-exposed or, at least, mixed asbestos dust-exposed. The argument that chrysotile "disappears" from the lung is in part true, but it overstates the process, as the fiber is found with very great frequency in the pulmonary tissues of workers. However, as in the European studies, only when specific amphiboles are observed in association with chrysotile do mesotheliomas appear.

Given the astonishing prevalence of crocidolite in the lungs of persons who worked in US shipyards, one is forced to paraphrase a frequently heard dictum regarding mesothelioma in bystander workers: "The fiber had no respect for the jurisdictional boundaries of the trades." Irrespective of whether or not insulation workers ever handled crocidolite-containing thermal insulation products, if any of their work history included time in a shipyard setting, then potential exposure to crocidolite fiber was great. We note for the record that insulation materials obtained in buildings outside of shipyards in New York City, analyzed by polarized light microscopy, showed the presence of all three major commercial fibers: amosite, chrysotile, and crocidolite (see Table 12). Crocidolite was found in about 1.4% of all the thermal insulation products examined (Table 12).

We conclude that asbestos exposure to many categories of workers in the United States included amphiboles. Consumption data are poor indices of asbestos exposure, and cannot be used to predict disease prevalence, nor patterns, especially for mesothelioma.

MESOTHELIOMA IN THE NON-OCCUPATIONAL SETTING

Some portion of risk assessment is built on that which focuses on reports, incorporating anecdotal observations, which indicate that mesothelioma occurs in the non-occupational setting. These include reports of mesothelioma in bystander occupations, in household members of asbestos workers, and in people who live in the environment of an asbestos mine, mill, factory, or facility which used asbestos-containing products (e.g., the vicinity of a shipyard). These reports often carry the presumption of low-to-ambient exposure to asbestos. Frequently cited studies are given in Table 8.

There are several features which require attention. The fiber type most cited in these exposures is crocidolite; asbestiform amphiboles are virtually always implicated as a component of exposure (Table 9).

As an example of a study always cited, but frequently mischaracterized in terms of exposure, is that of Wagner, Sleggs, and Marchand (1960). This single, most important, report gave rise to concern that "environmental exposure" to asbestos was associated with mesothelioma. The report showed that about half of the pleural mesotheliomas observed in the Kuruman area of the Cape Province occurred in persons who "merely lived" in the vicinity of the mine and mill. For those who died with pleural mesothelioma, there was no correlation between severity of asbestosis and the occurrence of mesothelioma (some had little or no evidence of parenchymal scarring nor the presence of asbestos bodies in their tissues). Mesothelioma victims often showed long latency periods ($t \geq 40$ years), and the ages of the dead were such that, for many, the exposure had obviously begun in childhood. These observations reinforced the impression that low-level-to-ambient exposure to asbestos had produced these pleural malignancies. The question is: "How accurate is this assertion?"

The area in which the 33 original mesotheliomas were found is located in the northwestern region of the Cape Province, centered around the town of Kuruman (Fig. 1). At the time, it represented the most important of the three crocidolite fiber-producing areas in the world. The area is located on the southern edge of the Kalahari Desert, Bechuanaland, and therefore experienced long dry seasons (Fig. 1A). The crocidolite occurs as ore in a banded ironstone (Fig. 1B). The local landscape is almost barren, with the only vegetation being desert scrub (Fig. 1C). The mine dumps had no topsoil or vegetation covering them (Fig. 1C). Local children played upon these hilly dumps (Wagner et al, 1960). When one of us (AML) visited the area in 1969, the local mine and access, secondary, roads were unpaved and surfaced with mine tailings (Figs. 1C, 1D). Almost everywhere fibers of crocidolite were visible and could be recovered from the road aggregate surface by simple handpicking (Fig. 1E). AML was informed at that time (1969) that the primary area roads, then blacktopped, had just been paved.

Wagner stated at scientific meetings (1969; 1977) that: the crocidolite process at the Kuruman mill was so dusty that "one couldn't see from one side of the mill to the other"; "the only means of ventilating and cooling the mill in the summer were the windows, so that they remained open virtually all the time"; "the airborne dust concentrations in the environment of the mill was so great that the roofs of nearby native shanties occasionally caved in from the weight of the settled dust".

In addition to the wide use of tailings on roads, and the intense point source of fiber from a mill, local settlers were known to have "hand-cobbed" high-grade fiber from small hillside outcroppings of crocidolite ore and sold wagon loads to the local mill.

The Cape environmental mesotheliomas resulted from unusual factors, with geographic, climatic, economic, and social circumstances rendering dust exposures both frequent and intense. Persons walking along a tailings-covered Kuruman road in, e.g., 1940, with vehicles raising dust as they passed, were likely subjected to dust clouds containing crocidolite fiber.[4] Children playing on the local dumps were similarly exposed. These exposures do not even remotely relate to the present exposures to passive occupants of buildings containing asbestos products in the US today. The impression that ambient levels of fiber exposure known in non-mining areas today may account for a similar number of tumors, appears to be inaccurate.

Fiber Type in South Africa and Mesothelioma

In addition to this unusual dust exposure, there is an equally extraordinary distribution of mesotheliomas in South Africa. Wagner (1964) reported on 120 histologically confirmed cases of mesothelioma in South Africa. Of these cases, more than 100 occurred in persons who either worked in or lived in the vicinity of the crocidolite mines of the Cape Province; of 10 "industrial" cases, 9 had crocidolite exposure; 1 case had occurred in association with chrysotile mining and milling in Havelock, Swaziland; no cases had been reported in the Transvaal amosite field (locations shown in Fig. 1A).

In a follow-up report, Webster (1978) disclosed that 712 mesothelioma cases were reviewed by, and known to, the South African Tumor Reference Panel: of the 127 cases associated with exposure in mining and milling of asbestos, 120 were exposed to Cape crocidolite, 2 to Transvaal crocidolite, 4 to amosite (2 of 4 were also exposed to Transvaal crocidolite) and 1 to Swaziland chrysotile; of the almost 100 "environmental" mesotheliomas, 93 were located within the Cape crocidolite field, 2 were exposed to

---

[4] Although not measured at that time, similar circumstances in Rockville, Maryland, in the United States, with road tailings made of serpentine rock, indicate that fiber is released during vehicular traffic (Rohl et al. 1977).

Transvaal crocidolite, and 1 was a possible exposure to Transvaal amosite. None were known to have occurred in the chrysotile mining area.

The Importance of Shipyards

Many bystander mesotheliomas have occurred in the shipyard setting (Table 8). Studies of environmental mesothelioma (occurring in non-workers) have similarly been linked with shipyard environments (Table 9).

These data in Table 10 represent 53 short-term air samples from on-site air monitoring in a west coast US Naval shipyard, utilizing air pumps pulling at approximately 2 liters of air/minute through 37 mm AA Millipore™ membrane filters for time periods of 10-30 minutes (Area, Ship #1) and for 5-35 minutes (Ship #2). These values are given for short-term excursion comparisons only and have not been recalculated to compare with eight-hour time-weighted averages. We note that the intense exposures were obtained as "snap" samples, with time periods of 5-10 minutes, so that the corresponding eight-hour TWA values are considerably lower.

Several points are worthy of note. The measurable fiber levels found in shipyard environment ranged almost over three orders of magnitude (Table 10). Rip outs aboard ship yielded the highest of these values, and one filter was too loaded to count, the latter feature noted by Harries (1971) in his Devonport study. Harries found ripouts generate intense aerosols, with resulting measurements yielding values up to 800 f/ml. Bystander exposures were recorded as high as 50 f/ml during these operations. The fiber type in these instances was crocidolite.

There are other useful data uncovered in the west coast study. Based on only two air samples, the fiber levels found in the workers' changing rooms were found to average over 5 f/ml. These values may be used to support the hypothesis that household mesothelioma may be associated with measurable levels of fiber exposure rather than the perceived "ambient" levels. Here, frequency and intensity of excursions may be important variables of dose.

The impression that non-occupational mesothelioma occurs after low, invisible, or even ambient exposure, appears not to be the universal case. Even household mesothelioma, with dusty clothing as the vector, may have been associated with exposures greater than 5 f/ml.

There are no specific data to refute the concept that ambient-level exposures to fiber produces mesothelioma. The above are included to question the assertion that the most described "environmental" and "bystander" mesothelioma reports are invariably associated with low-level, possibly ambient, exposures. They are not.

STUDIES WHICH PROVIDE DOSE-RESPONSE AND TIME DATA FOR MESOTHELIOMA RISK CALCULATION

One of the most important, and least easily justified, assumptions of risk assessment is that of extrapolation along a dose-response curve to arrive at a dose value for non-occupationally exposed persons which places them at risk.

The five principal studies which have been used by the Environmental Protection Agency as a basis for the dose-response and time factors in their quantitative mesothelioma risk calculation are given in Table 11. These studies are only a small portion of those which provide fiber measurement in the workplace. They were selected because the respective cohorts yield mesotheliomas in large numbers, which permits calculation of statistically reliable trends. Note that three of the studies involve exposure to crocidolite, one to amosite, and one to mixed fiber types. None involve exposure to chrysotile alone.

Other studies in the US Environmental Protection Agency's (1980) report provide less quantitative data in these areas, but further amplification of the latency factor: Berry and Newhouse (1983); Peto (1980). Both these studies involve exposure to chrysotile, although both the Ferodo and Rochdale mesotheliomas are now considered to be crocidolite-induced.

These data show that the most important elements of the mesothelioma risk model are derived from cohorts in which workers were amphibole-exposed (predominantly to crocidolite). They may be used to estimate risk to workers in appropriate workplaces, handling amphibole- containing products. These studies overstate risk to persons exposed to chrysotile in buildings.

WHAT ARE THE FIBER TYPES PASSIVE BUILDING OCCUPANTS ARE EXPOSED TO?

Fiber type is not considered as an important component of the EPA risk assessment model. Yet, to estimate the mesothelioma risk for passive occupants of buildings, it is crucial to know such factors as how many buildings have asbestos in them, in what form, what fiber type, etc. Such a study was undertaken in New York City in 1984.

In the summer of 1984, New York City's Department of Personnel undertook a preliminary Asbestos Survey, designed to determine the nature and scope of asbestos and asbestos-containing materials present in private and public buildings.[5] The mineralogical

---

[5] The selection of buildings and sampling strategy are contained in the report.

characterization of thermal system insulation and surface[6] materials was undertaken, utilizing polarized light microscopy and, where appropriate, continuous scan x-ray diffraction. The findings are summarized in Tables 12 and 13.

Thermal System Insulation: About one-third of thermal system insulation is non-asbestos-containing (Table 12). The remaining two-thirds material consist of chrysotile alone, mixed fiber and amphibole fiber only. Almost 26% of the total thermal system insulation contains one or more amphiboles. Crocidolite-containing insulation was found in 11 of 1,264 total specimens examined (~0.87%) or 11 of 798 asbestos-containing specimens examined (~1.38%). See details in Table 12.

Surface Materials: Most decorative/acoustic surface materials found in New York City buildings do not contain asbestos fiber of any variety (Table 13). Of the approximately 9.6% of the materials which do contain asbestos, about three-quarters are chrysotile-containing and the remainder amphibole-containing. Twenty of 1,199 surfacing materials contained amphiboles and two of these, crocidolite. It is crucial to note that all the 19 amosite only materials were ceiling tiles.

Although, overall, the number of asbestos-containing surface materials was small, chrysotile-containing formulations form the preponderant material. Most of New York City passive building occupants are exposed to chrysotile (if any asbestos at all). We conclude that the assessment of risk to passive building occupants has never been carried out for the appropriate fiber type.

DISCUSSION AND CONCLUSIONS

Based on a comparison of mortality data obtained on cohorts of workers exposed to specific asbestos fiber types, exposure to the amphibole asbestos varieties is associated with greater mesothelioma mortality as compared to exposure to chrysotile only. This differential does not reflect tremolite-induced mesotheliomas among chrysotile-exposed workers in the Canadian chrysotile industry. Comparing studies of identical design shows that the mesothelioma differential is large and real. The Mount Sinai studies underscore this remarkably different mortality experience.

Tissue burden studies of lungs obtained from exposed workers in Great Britain have enabled investigators to identify the agents implicated in the etiology of mesothelioma. In some cases, the fiber type found has been unanticipated. Here the presence of crocidolite, in the presumably chrysotile-only exposed, emerges as an important etiological agent.

Tissue burden studies of US workers support observational data sets obtained elsewhere that the amphibole asbestos content of their lungs is greater than previously

---

[6] Surface materials included acoustic plasters, fireproofing, soundproofing, ceiling tiles.

assumed. Fiber consumption data cannot be used as indices of exposure. US insulation workers are primarily amosite-exposed; crocidolite exposure has occurred among these workers as well.

Environmental and bystander studies suggest that the amphibole asbestos minerals are the etiological agents for mesothelioma. Some of these studies also suggest that exposure may have been both episodic and intense. Data obtained in the shipyard setting also suggest that both bystander, and by extrapolation, household members may involve exposures which were both high and episodic. Not all mesothelioma studies have been evaluated, but the above studies are included for illustrative purposes only. They merely point out that misconceptions abound in the field.

Persons who passively occupy buildings with asbestos-containing surface materials are generally exposed to chrysotile. However, maintenance personnel in these settings may be exposed to insulation materials which contain amphiboles to large degree. Risk models for each group should reflect appropriate exposures and fiber type. They should produce very different quantitative assessments for mesothelioma risk.

With asbestos risk assessment, several crucial parameters must be well known in order to develop and formulate a reliable and plausible model: age distribution of the exposed population, exposure characteristics, latency, and response. For extrapolation to the non-occupational situation, these parameters must be known over a wide range of values.

The biological activity of asbestos is thought to be related to many factors, among these fiber type, fiber character (length/diameter). Fiber type remains to be separately considered in terms of risk assessment in the US.

Although not included in this text, we have previously suggested that ripouts of old insulation may involve exposure to chrysotile fiber which has been thermally modified. Asbestos removal of "thermally-aged" insulation may result in a more dusty exposure and is, in general, an overall more dangerous material than was originally applied (Langer and Nolan, 1987). Ripout environments for insulation workers stand out in importance.

For each of the major asbestos diseases which may cause death in exposed workers, i.e., pleural mesothelioma, peritoneal mesothelioma, lung cancer, asbestosis, and cancers of other sites, each is likely associated with its own constellation of features. Each is more likely to occur with exposure to a specific fiber with its own set of physical properties, e.g., dimension, etc. The corollary is therefore true: no one set of factors, nor one dose, nor one fiber, nor one set of physical properties, can produce all of the diverse biological outcomes.

It seems plausible to us that insulation workers, by virtue of working with, and experiencing exposure to, all major commercial fiber types, to fiber of all dimensions including those which are very long and thin ($> 20\ \mu m$, $d < 0.2\ \mu m$), to extraordinarily high doses for short periods of time during ripout and replacement in confined spaces, and

prolonged exposures producing high time-weighted averages, would exhibit all the asbestos diseases to a significant degree. The crucial point is, even if data were available on exposure, do their biological outcomes have relevance in other settings? Any more so than the mortality experienced by textile workers can be used to predict mortality outcome for workers in a friction product manufacturing facility? Or can mortality experience for workers in secondary or tertiary industries be used to predict mortality experience for insulation workers? We think not.

Exposure to chrysotile dust in the building setting is generally, according to all available data, non-episodic and of barely measurable level (even by the TEM method). Asbestosis, a disease which requires high levels of exposure to manifest, will not likely develop in passive building occupants; if the building occupants are non-smokers, lung cancer will similarly revert to the anticipated background levels found in non-smokers. Pleural mesothelioma risk is certainly lower, probably by at least one, and possibly two, orders of magnitude, than previously calculated. Peritoneal mesothelioma is unlikely to ever occur. Following this vein, some of the studies used for mesothelioma risk calculation are those with high mortality due to the peritoneal form. Even the amphibole-exposed cohorts must be downwardly adjusted to reflect the pleural form only. The use of data obtained on, e.g., insulation workers, may be appropriate for building maintenance personnel only.

The basic foundation of quantitative risk assessment used by the United States EPA requires modification before future abatement activities are considered for public and private buildings.

ACKNOWLEDGEMENTS

The authors wish to thank Professor Richard Wilson, Department of Physics, Harvard University for his excellent critique and comments.

REFERENCES

Acheson, E.D., Gardner, M.J., Pippard, E.C., and Grime, L.P. (1982). Mortality of two groups of women who manufactured gas-masks from chrysotile and crocidolite asbestos. A 40-year follow-up. Br. J. Ind. Med. 39, 344-348.

Acheson, E.D., Gardner, M.J., Winter, P.D., and Bennett, C.(1984). Cancer in a factory using amosite asbestos. J. Epidemiol. 13, 3-10.

Anderson, H.A., Lilis, R., Daum, S.M., Fischbein, S.A., and Selikoff, I.J.(1976). Household-contact asbestos neoplastic risk. Ann. NY Acad. Sci. 271, 311-323.

Berry, G.(1987). Chrysotile and mesothelioma. Accompl. Oncol. 1, 123-132.

Berry, J., and Newhouse, M.(1983). Mortality of workers manufacturing friction materials using asbestos. Brit. J. Ind. Med. 40, 1-7.

Bianchi, C., Brollo, A., and Ramini, L.(1988). Malignant pleural mesothelioma in Monfalcone, Italy. In "Non-Occupational Exposure to Mineral Fibers", International Agency for Research on Cancer, World Health Organization (J. Bignon et al, Eds.). Sci. Publ. 90. Pp. 330-335.

Bohlig, H., Dabbert, A.F., Dalquen, P., Hain, E., and Hinz, I.(1970). Epidemiology of malignant mesothelioma in Hamburg. A preliminary report. Environ. Res. 3, 365-372.

Bohlig, H., and Hain, E.(1973). Cancer in relation to environmental exposure. In "Biological Effects of Asbestos". IARC Sp. Publ. 8 (P. Bogovski, et al., Eds.), Lyon, France. Pp. 217-221.

Cartier, P.(1964). Dust exposure as an etiological factor of asbestosis and pleural plaques. In "International Congress on Asbestosis," May 29-30, Caen, France. Pp. 77-82.

Churg, A.(1987). Pathologic reactions to chrysotile and their mineralogic correlates. Accompl. Oncol. 1, 54-58.

Clemmesen, J., and Hjalgrin-Jensen, S.(1981). Cancer incidence among 5686 asbestos cement workers followed from 1943 through 1976. Ecotoxicol. Environ. Safety 5, 15-23.

Dement, J.M., Harris, R.L., Jr., Symans, M.J., and Shy, C.M.(1983). Exposures and mortality among chrysotile asbestos workers. Part II: Mortality. Am. J. Ind. Med. 4, 421-433.

Doll, R., and Peto, J.(1985). Asbestos: Effects on health of exposure to asbestos. In "Health and Safety Executive", HMSO, London.

Finkelstein, M.M.(1983). Mortality among long-term employees of an Ontario asbestos-cement factory. Brit. J. Ind. Med. 40, 138-144.

Green, F.H.Y., Harley, R., Vallyathan, V., Dement, J., Pooley, F.D., and Althouse, R.(1987). Pulmonary fibrosis and asbestos exposure in chrysotile asbestos textile workers. Accompl. Oncol. 1, 59-68.

Harries, P.G.(1968). Asbestosis hazards in naval dockyards. Ann. Occup. Hyg. 11, 135-145.

Harries, P.G.(1971). Asbestos dust concentrations in ship repairing: A practical approach to improving asbestos hygiene in naval dockyards. Ann. Occup. Hyg. 14, 241-254.

Hobbs, M.S.T., Woodward, S.D., Murphy, B., Musk, A.W., and Elder, J.E.(1980). The incidence of pneumoconiosis, mesothelioma and other respiratory cancer in men engaged in mining and milling crocidolite in Western Australia. In "Biological Effects of Mineral Fibers", World Health Organization, International Agency for Research on Cancer (J.C. Wagner and W. Davis, Eds.). Lyon, France. Pp. 615-625.

Jones, J.S.P., Smith, P.G., Pooley, F.D., Berry, G., Sawle, G.W., Madeley, R.J., Wignell, B.K., and Aggarwal, A.(1989). The consequences of exposure to asbestos dust in a wartime gas-mask factory. In "Biological Effects of Mineral Fibers", World Health Organization, International Agency for Research on Cancer (J.C. Wagner and W. Davis, Eds.). Lyon, France. Vol. 2. Pp. 637-653.

Langer, A.M., Mackler, A.D., and Pooley, F.D.(1974). Electron microscopical investigation of asbestos. Environ. Health Perspect. 9, 63-80.

Langer, A.M., and McCaughey, W.E.T.(1982). Mesothelioma in a brake repair worker. The Lancet, 1101- 1103.

Langer, A.M., and Nolan, R.P.(1987). The properties of chrysotile asbestos as determinants of biological activity. Accompl. Oncol. 1, 30-51.

Langer, A.M., and Nolan, R.P.(1989). Fiber type and burden in parenchymal tissues of workers occupationally exposed to asbestos in the United States. In "Non-Occupational Exposure to Mineral Fibers", International Agency for Research on Cancer, World Health Organization (J. Bignon et al, Eds.). Sci. Publ. 90. Pp. 330-335.

McDonald, A.D., Fry, J.S., Woolley, G.J., and McDonald, J.C.(1983a). Dust exposure and mortality in an American chrysotile textile plant. Brit. J. Ind. Med. 40, 361-367.

McDonald, A.D., Fry, J.S., Woolley, G.J., and McDonald, J.C.(1983b). Dust exposure and mortality in an American factory using chrysotile, amosite, and crocidolite in mainly textile manufacture. Brit. J. Ind. Med. 40, 368-374.

McDonald, A.D., Fry, J.S., Woolley, G.J., and McDonald, J.C.(1984). Dust exposure and mortality in an American chrysotile asbestos friction products plant. Brit. J. Ind. Med. 41, 151-157.

McDonald, J.C., and Liddell, F.D.K.(1979). Mortality in Canadian miners and millers exposed to chrysotile. Ann. NY Acad. Sci. 330, 1-10.

McDonald, J.C., Liddell, F.D.K., Gibbs, G.W., and Eyssen, G.E.(1980). Dust exposure and mortality in chrysotile mining. Brit. J. Ind. Med. 37, 11- 24.

Mancuso, T.F., and Coulter, E.J.(1963). Methodology in industrial health studies. Arch. Environ. Health 6, 210-226.

Newhouse, M.L., and Berry, G.(1979). Patterns of mortality in asbestos factory workers in London. Ann. NY Acad. Sci. 330, 53-60.

Newhouse, M.L., and Thompson, H.(1965). Mesothelioma of the pleura and peritoneum following exposure to asbestos in the London area. Brit. J. Ind. Med. 22, 261-269.

Nicholson, W.J., Selikoff, I.J., Seidman, H., Lilis, R., and Formby, P.(1979). Long-term mortality experience of chrysotile miners and millers in Thetford Mines, Quebec. Ann. NY Acad. Sci. 330, 11-22.

Peto, J.(1980). Lung cancer mortality in relation to measured dust levels in an asbestos textile factory. In "Biological Effects of Mineral Fibers" (J.C. Wagner and W. Davis, Eds.). Lyon, France: World Health Organization, Internation Agency for Research on Cancer. Vol. 2. Pp. 829-836.

Peto, J., Seidman, H., and Selikoff, I.J.(1982). Mesothelioma mortality in asbestos workers: Implications for models of carcinogenesis and risk assessment. Brit. J. Cancer 45, 124-135.

Peto, J., Doll, R., Goffe, T., and Clayton, R.(1985). Mortality of workers in a Rochdale asbestos textile factory. Ann. Occup. Med.

Policard, A.(1964). On the role of mixed dusts in lung diseases due to dust. International Congress on Asbestosis, May 29-30, Caen, France. Pp. 281-290.

Pooley, F.D., and Mitha, R.(1987a). Fiber types, concentrations and characteristics found in the lung tissues of chrysotile-exposed cases and controls. Accompl. Oncol. 1, 1-11.

Pooley, F.D., and Mitha, R.(1987b). Determiniation and interpretation of the levels of chrysotile asbestos in lung tissue. Accompl. Oncol. 1, 12-18.

Rohl, A.N., Langer, A.M., Selikoff, I.J. (1977). Environmental asbestos pollution related to use of quarried serpentine rock. Science, 196, 1319-1322.

Rossiter, C.E., and Coles, R.M.(1980). HM dockyard Devonport: 1947 mortality study. In "Biological Effects of Asbestos", IARC Sci. Publ. 30 (J.C. Wagner, et al., Eds.), Lyon, France. Vol. 2, pp. 713-721.

Rubino, G.F., Piolatto, G., Newhouse, M.L., Scansetti, G., Aresini, G.A., and Murray, R.(1979). Mortality of chrysotile asbestos workers at the Balangero mine, northern Italy. Brit. J. Ind. Med. 36, 187-194.

Sebastien, P., Begin, R., Case, B.W., and McDonald, J.C. (1987). Inhalation of chrysotile dust. Accompl. Oncol. 1, 19-29.

Selikoff, I.J.(1984). Statement on the proposed asbestos standard. Presented before OSHA-US-DOL, July 2, 1984. Tables 1, 9, 11, and 13. On cohorts of Locals 12 and 32, Asbestos Insulation Workers (NY- NJ); National Survey Asbestos Insulation Workers; Amosite Asbestos Factory Workers, NJ.

Selikoff, I.J., Churg, J., and Hammond, E.C.(1964). Asbestos exposure and neoplasia. JAMA 188, 22-36.

Selikoff, I.J., Hammond, E.C., Churg, J. (1970). Mortality experience of asbestos insulation workers. In: Pneumoconiosis. Proc. Intl. Conf. Johannesburgh, 1969. H. Shapiro, Ed., 97-103. Oxford Univ. Press, Capetown.

Selikoff, I.J., and Hammond, E.D.(1975). Multiple risk factors in environmental cancer. In "Persons at High Risk of Cancer" (J.F. Fraumeni, Ed.), Academic Press, New York. Pp. 467-483.

Selikoff, I.J., Hammond, E.C., and Seidman, H.(1979). Mortality experience of insulation workers in the United States and Canada: 1943-1976. Ann. NY Acad. Sci. 330, 91-116.

Stossel, H.G., Dalquen, P., and Carstens, U.(1972). Zur Frage des Pleuramesothelioma bei Werftarbeitern. Forsch. auf dem Geb. der Roentgenstra. 116, 41-45.

Stumphius, J.(1968). Asbestos and cancer. Abbottempo 3, 26-29.

Stumphius, J.(1971). Epidemiology of mesothelioma on Walcheren Island. Brit. J. Ind. Med. 28, 59-66.

Tabershaw, I.R., Cooper, W.C., and Balzer, J.L.(1970). A labor-management occupational health service in a construction industry. Arch. Environ. Health, 21, 784-788.

Tayot, J., Desbordes, J., Ernoult, J.L., and Potoine, B.(1966). Mesotheliome pleural et asbestose. J. Francais de Medecine et chirurgie Thoraciques 7, 757-774.

Thomas, H.F., Benjamin, I.T., Elwood, P.C., and Sweetnam, P.M.(1982). Further follow-up study of workers from an asbestos cement factory. Brit. J. Ind. Med. 39, 273-276.

Timbrell, V., Griffiths, D.M., and Pooley, F.D.(1971). Possible biological importance of fibre diameters of South African amphiboles. Nature (London) 232, 55-56.

United States Environmental Protection Agency (1986). Airborne asbestos health assessment update. EPA/600/8.84/003F, June, Res. Triang Park, North Carolina.

Wagner, J.C.(1964). Asbestos dust exposure and malignancy. In "International Congress on Asbestosis," May 29-30, Caen, France. Pp. 77-82.

Wagner, J.C.(1968). Asbestos and cancer. Abbottempo 3, 26-29.

Wagner, J.C., Sleggs, C.A., and Marchand, P.(1960). Diffuse pleural mesotheliomas and asbestos exposure in the northwestern Cape Province. Brit. J. Ind. Med. 12, 260-271.

Wagner, J.C., Gilson, J.C., Berry, G., and Timbrell, V.(1971). Epidemiology of asbestos cancers. Brit. Med. Bull. 27, 71-76.

Wagner, J.C., Griffiths, D.M., and Munday, D.E.(1987). Recent investigations in animals and humans. Accompl. Oncol. 1, 111-120.

Webster, I.(1978). Discussion. In "Proceedings of Asbestos Symposium", National Institute for Metallurgy (W.H. Glen, Ed.), Johannesburg, South Africa, October, 1977. P. 79.

Weiss, W.(1977). Mortality of a cohort exposed to chrysotile asbestos. J. Occup. Med. 19, 737-740.

Table 1: Proportion of Deaths Due to Mesothelioma in Chrysotile Cohorts.

Study	Exposure	Deaths[a]	No. Meso[b]	% Meso[c]	SEP[d]
Rubino et al (1979)	Miners and millers of chrysotile; Balangero, Italy.	38	1	2.6	±2.61
McDonald et al (1980)	Miners and millers of chrysotile; Eastern Township of Quebec.	760	10	1.3	±0.41
Thomas et al (1982)	Factory - asbestos cement; possibly amphiboles used.	74	2	2.7	±1.91
McDonald et al (1983a) (1983b)	US Textile plant. US Friction product manufacturer.	120	1	0.8	±0.91
McDonald et al (1984)	US Friction product manufacturer.	213	0	0.0	±0.00
Peto et al (1985)	UK Textile plant; Rochdale.	225	17[e]	7.6	±1.84
Berry & Newhouse (1983)	UK Friction product manufacturer; Ferodo.	331	8[f]	2.4	±0.85
	Cohorts N=8 Total	1,761	39	2.21	±0.35

Table 1: Continued

These data were extracted from Doll and Peto (1985), their report to the Health and Safety Executive, London.

[a] These are total deaths due to malignancy experienced by the cohort.

[b] Number of mesothelioma deaths reported in the cohort.

[c] Percentage of mesothelioma deaths (N-mesothelioma/total deaths due to malignancy).

[d] Standard error of the percentage.

[e] Of 17 mesotheliomas at Rochdale, study of pulmonary tissues has shown that crocidolite and amosite are present in all cases (Pooley, personal communication, 1988).

[f] As for Rochdale, study of tissues has shown that crocidolite and amosite are present in the tissues examined (Pooley, personal communication, 1988). N now equals 13 (Newhouse and Sullivan, Brit J Ind Med, 46:176-179, 1989). Crocidolite exposure established in 11 of 13 cases; diagnosis uncertain in 1 of 13 cases; occupational history uncertain in 1 of 13 cases.

Total deaths due to malignancy include mesothelioma, lung cancer, gastrointestinal cancers, and cancers of other sites.

Note: No confirmed cases of peritoneal mesothelioma have been reported.

Range of mesothelioma mortality: 0.00-7.6%. $\bar{x}$ = 2.21% ± 0.35% (SEP).

Table 2: Proportion of Deaths Due to Mesothelioma in Amphibole and Mixed-Dust Cohorts.

Study	Exposure	Deaths[a]	No. Meso	% Meso	SEP
Acheson et al (1984)	Factory making amosite insulation products.	109	5	4.6	±2.09
Clemmesen & Hjalgrim-Jensen (1981)	Asbestos cement plant. Mixed fiber types.	167	3	1.8	±1.03
Selikoff & Hammond (1975)	Factory making amosite insulation products.	163	11	6.7	±2.02
Selikoff et al (1964)	Insulation workers, Locals 12 & 32, NY-NJ; mixed fibers.	95	7	7.4	±2.79
Selikoff et al (1979)	Insulation workers, National survey; mixed fibers.	846	102[b]	12.1	±1.20
Newhouse & Berry (1979)	Factory making asbestos products; amphibole fibers.	227	46	20.3	±2.99
Rossiter & Coles (1980)	Shipyard workers; mixed	270	31	11.5	±2.06

Table 2: Continued

Study	Exposure	Deaths[a]	No. Meso	% Meso	SEP
Mancuso & Coulter (1963) Weiss (1977) Berry & Newhouse (1979) Finkelstein (1983)	Mixed cohorts; mixed fiber exposures, cement, friction product manufacturing with amphibole, etc.	130	23	17.69	±3.69
	Cohorts N=11 Totals	2,007	228	11.36	±0.75

These data extracted from Doll and Peto (1985).

[a] As for chrysotile cohort data, these total deaths are due to malignancy, number of mesothelioma deaths reported, percentage of mesothelioma deaths.

[b] Mesothelioma deaths in Selikoff cohorts include peritoneal mesotheliomas. Note that about two-thirds of insulation worker mesotheliomas are peritoneal.

Range of mesothelioma mortality: 1.8-20.3%. $\bar{x}$ = 11.36% ± 0.75% (SEP).

Table 3: Pooled Mortality Due to Malignancy for All Asbestos Cohorts.

Fiber Exposure	# of Cohorts	Number of Malignant Deaths by Category (Pooled SMR)						
		Lung Cancer N	Lung Cancer SMR	GI Cancer N	GI Cancer SMR	Cancer OS* N	Mesothelioma N	Totals
Chrysotile	N= 8	660	1.25	476	1.05	586	39	1,761
Amphibole or Mixed	N=11	866	2.86	318	1.34	595	228	2,007
Totals	N=19	1,526		794		1,181	267	3,768

Excess gastrointestinal cancers about 14-17% of excess lung cancers (see Doll & Peto, 1985): Chrysotile 23:132 = 17.4%; Amphibole or mixed 81:563 = 14.4%.

Chrysotile mesothelioma mortality is dominated by both Rochdale and Ferodo data. These have resulted from exposure to crocidolite used in these plants: 17/39 = 43.6% of mesotheliomas for Rochdale; 8/39 = 20.5% of the mesotheliomas for Ferodo.

Note the amphibole pooled mortality data show an almost seven-fold greater excess of lung cancer, and gastrointestinal cancers, as compared to the pooled chrysotile data.

* Malignancies arising at sites and in organs other than those of the lung, gastrointestinal tract and mesothelial surfaces.

Table 4: Comparison of Mesothelioma Mortality in Four Mount Sinai Cohorts.

Study	Total Deaths in Cohort	Total Deaths Due to Malignancy in Cohort	Excess Lung Cancer Deaths	Meso Deaths	% Meso/ Malign Deaths	Excess Lung Cancer: Meso Ratio
Selikoff (1984)	532	238	89	50[a]	21.0	1.78:1
Insulators, NY-NJ		(44.7%)	(32.4%)			
Selikoff (1984)	2,271	995	380	175[b]	17.6	2.17:1
Insulators, US & Canada		(43.8%)	(38.2%)			
Selikoff (1984)	594	195	81	16[c]	8.2	5.06:1
Amosite Factory		(32.8%)	(41.5%)			
Nicholson et al (1979)	130	44	17	1[d]	2.3	17:1
Chrysotile, Thetford		(33.8%)	(38.6%)			

Table 4: Continued

---

[a] 50 mesotheliomas include 36 (72%) peritoneal mesotheliomas. Note that this cohort has 8.6% of its deaths due to asbestosis.

[b] 175 mesotheliomas include 112 (64%) peritoneal mesotheliomas. Note that this cohort has 7.4% of its deaths due to asbestosis.

[c] 16 mesotheliomas include 8 (50%) peritoneal mesotheliomas. Note that this cohort has 5.1% of its deaths due to asbestosis.

[d] One pleural mesothelioma only. Note that this cohort has 1.5% of its deaths due to asbestosis.

Table 5: Concentrations[a] of Asbestos Fibers in Lung Tissues of US Persons (54 Cases).

Fiber Type	No. Cases in Which Fiber Observed N (%)	No. Cases in Which Fiber Observed as Lone Fiber[b] N (%)	Fiber Concentrations[c] Avg	SD	Range
Amosite	40 (74.1)	14 (25.9)	27.2	64.9	0.2-350.0
Chrysotile	33 (61.1)	12 (22.2)	281.8	746.5	0.1-4420.0
Crocidolite	13 (24.1)	2 (3.7)	2.1	7.5	0.1-50.0
Tremolite-actinolite	13 (24.1)	0 (0.0)	3.0	10.9	0.1-60.0
Anthophyllite	16 (29.6)	0 (0.0)	1.6	7.3	0.1-50.0

[a] Per gram of dried lung tissue.

[b] Those cases among the 54 studied in which the fiber type was the only major commercial fiber found in tissues.

[c] Values for average concentration, standard deviation (SD), and range are expressed in millions of fibers per gram of dried lung tissue (for fibers > 1 μm only).

Table 6: Concentrations of Major Commercial Types of Asbestos Fibers in Lung Tissue, By Occupational Category.

		Concentration[a]		
Occupational Category	No.	Amosite	Chrysotile	Crocidolite
Insulation Workers	16	47.5±78.2	299.3±1101.1	0.5±1.7
Shipyard Workers	15	11.4±19.9	91.4±177.4	5.1±13.4
Other Trades	23	25.4±75.6	394.7±699.2	3.1±3.4

[a] Millions of fibers per gram of dried lung tissue. Fibers greater than 1 μm in length.

Table 7: Asbestos Fiber Type by Prevalence Within Occupational Categories.

Occupational Category	No.	Prevalence		
		Amosite	Chrysotile	Crocidolite
Insulation Workers	16	16/16=100%	8/16=50%	3/16=19%
Shipyard Workers	15	10/15= 67%	10/15=67%	6/15=40%
Other Trades	23	14/23= 61%	15/23=65%	4/23=17%
Total	54	40/54= 74%	33/54=61%	13/54=24%

Table 8: Mesothelioma in the Non-Occupational Setting.[a]

Study	Setting	Fiber Type(s)	Comment
Wagner et al (1960)	Kuruman, NW Cape Province, RSA.	Crocidolite	About half of 33 mesotheliomas occur in NOE.[b]
Newhouse & Thompson (1965)	East-end London, Barking plant.	Crocidolite, amosite	Large % of 76 mesotheliomas occur in NOE, DE.[c]
Harries (1968)	Devonport, Dockyard.	Crocidolite.[d] Probably other fiber types.	All but two mesotheliomas are non-occupational "bystanders".
Bohlig & Hain (1973)	Shipyards of Hamburg; Plant in Hamburg-Bergedorf.	Crocidolite. Probably other fiber types.	All mesotheliomas are non-occupational.
Anderson et al (1976)	Paterson, NJ. Household members of plant workers.	Amosite.	Five mesotheliomas. Domiciliary.

Table 8: Continued

Study	Setting	Fiber Type(s)	Comment
Bianchi et al (1988)	Monfalcone, Italy. Shipyard town.	Probably amphiboles; crocidolite and other fiber types.	Five of forty-two mesotheliomas are domiciliary.

[a] Non-occupational setting includes bystander and occult occupational (as in shipyard studies), individuals who lived in the area of an asbestos workplace, and domiciliary exposure.

[b] NOE is non-occupational exposure.

[c] DE is domiciliary exposure.

[d] Wagner (1968) stated that the fiber used throughout the European shipyards was crocidolite; other fiber types were used but to a lesser extent.

Table 9: Non-Occupational Mesotheliomas Near Shipyards.

Tayot et al (1966)	France
Bohlig et al (1970)	Germany
Stossel et al (1972)	Germany
Stumphius (1968; 1971)	Netherlands
Wagner et al (1971)	Great Britain
Tabershaw et al (1970)	United States

Sole cause thought to be dust from shipyards.

Predominant fiber type used in Europe (Great Britian, France, Italy, Germany, Netherlands), is crocidolite (see discussion in Wagner, 1968). Predominant fiber type in the United States is amosite with associated chrysotile and crocidolite (Langer and Nolan, 1989).

Table 10: Environmental Data Obtained in a West Coast US Naval Ship yard - 1975-1976.*

Area or Material	Activity	N	X̄f/ml	±0-±5	Range
Area #1					
Asbestos Paper	Cutting, ripping.	5	1.3	0.6	(0.4-1.8)
Gaskets	Punch, stamp.	3	3.6	1.0	(3.0-4.7)
Change Room	Changing clothes.	2	5.3	0.8	(4.7-5.8)
General Areas (non- or low-dusty)	"General" operations.	18	3.9	2.8	(0.2-9.7)
General Areas (dusty)	Includes ripouts.	5**	56.5	41.7	(13.4-102.6)
General Areas	Distance from worksite.	2	2.0	--	(----------)
Area: Shipboard #1					
Shipboard #1		N = 35	11.0	7.8	(0.2-102.6)
Shipboard #2					
All activities	Low dust,	15	3.5	2.8	(0.3-7.6)
	high dust.	3	68.1	65.0	(13.4-140.0)

Table 10: Continued

Area or Material	Activity	N	X̄ f/ml	±0-±5	Range
Shipboard #2		N = 18	14.3	35.2	(0.3-140.0)

*The data were obtained from Mr. Charles Ay, Long Beach, CA.

**One operation, not included, "filter too heavy to count". We conclude, because filter has been included with 140f/ml, that one measurement exceeded 140f/ml. Two filters of air sampled distant from worksites with low dust showed 0.0 fibers/ml. These are not included.

Table 11: Major Studies Which Provide Dose Response and Time Relationships for Calculation of Mesothelioma Risk.

Study	Exposure
Hobbs et al (1980)	Crocidolite, Wittenoom Gorge, NW Australia.
Jones et al (1980)*	Crocidolite; packers of military gasmasks in Nottingham, UK.
Finkelstein (1983)	Asbestos cement plant, Ontario. Mixed fibers.
Seidman (1984)	Amosite. Insulation for US Navy, WW II, Paterson, NJ.
Selikoff et al (1979)	US Insulation workers. Mixed fibers, including crocidolite.

* The Jones cohort, exposed to crocidolite, shows about a 15% mesothelioma mortality. In counterpoint to this, the Blackburn gasmask workers, also studied by Jones, exposed to chrysotile in the packing of civilian gasmasks, have yet to show a single mesothelioma. A similar differential in mesothelioma mortality was noted by Acheson et al (1982) in their 40-year follow-up study of gasmask workers exposed to the different fiber types.

Table 12: Mineralogical Makeup of Thermal Insulation Found in New York City Buildings.

		With Asbestos			
	Without Asbestos	Chrysotile Only	Mixed Fiber[b]	Amphibole Only[c]	Total
N	466[a]	470	277	51	1264
(%)	(36.87)	(37.18)	(21.91)	(4.04)	(100.0)

[a] These made with glasses and wools; cellulose and borate formulation; other materials.

[b] Mixed Fiber: 269 = amosite + chrysotile
5 = amosite + chrysotile + crocidolite
2 = chrysotile + crocidolite
1 = amosite + crocidolite

[c] Amphibole Only: 48 = amosite
3 = crocidolite

Table 13: Mineralogical Makeup of Surface Materials Found in New York City Buildings.

		With Asbestos			
	Without Asbestos	Chrysotile Only	Mixed Fiber[b]	Amphibole Only[c]	Total
N	1084[a]	84	11	20	1199
(%)	(90.41)	(7.01)	(0.91)	(1.67)	(100.0)

[a] These surfaces were made of glasses/wools, cement, cellulose, vermiculite, plaster; alone or in combination.

[b] Mixed Fiber: 10 - amosite + chrysotile

1 - amosite + crocidolite

[c] Amphibole Only: 19 - amosite (virtually all amosite is in acoustic ceiling tile)

1 - crocidolite

FIGURE LEGENDS

Figure 1: Relationship between percent of deaths in cohort due to asbestosis, and proportion of mesothelioma deaths due to the peritoneal form.

Figure 2: Crocidolite in the Kuruman environment.

A) A schematic map of South Africa showing the approximate location of the crocidolite outcropping in the northwestern Cape Province, cr; arrow pointing toward town of Kuruman. Other asbestos areas are shown: am denotes amosite in the Transvaal and ch denotes chrysotile areas in Swaziland. The Transvaal area contains crocidolite as well.

B) A crocidolite seam (~1.5cm wide, marked with f as fibrous ore) in a banded ironstone. Origin: Cape Province, South Africa. The reddish-brown bands are the iron oxide minerals.

C) A view of the Kuruman hills from the adit of a crocidolite mine outside of Kuruman: Area marked d is a local tailings dump (note the blue color); m is the mill. Note the mill and mine roads are blue, r. The foreground, the edge of the dump, contains waste host rock (mostly banded ironstone without ore). The local vegetation is noticeably sparse. The dump has no vegetation cover over the trailings.

D) A road leading to the dump. Note blue color and footmark impressions in loose gravel and dust. Arrow r points to a small road cut which displays the local reddish color of the soil and banded ironstone, and the blue top of the road itself. The road surface is made of tailings from the crocidolite operation rather than the banded ironstone.

E) Loose tailings and gravel (left hand, unmarked) and crocidolite fiber clump (white arrow) picked from the road surface leading to the dump. The fiber clump (white arrow) is approximately 1.5cm in length.

Figure 1

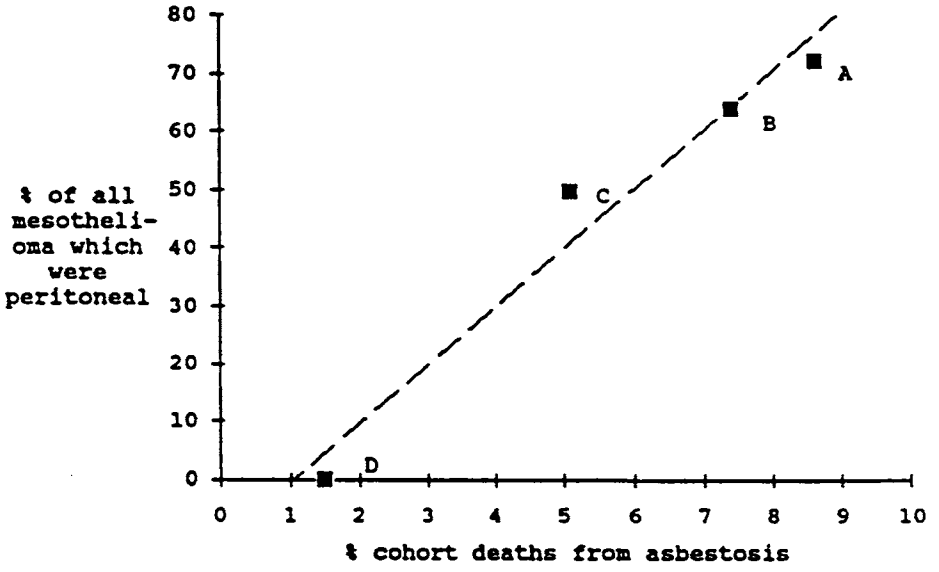
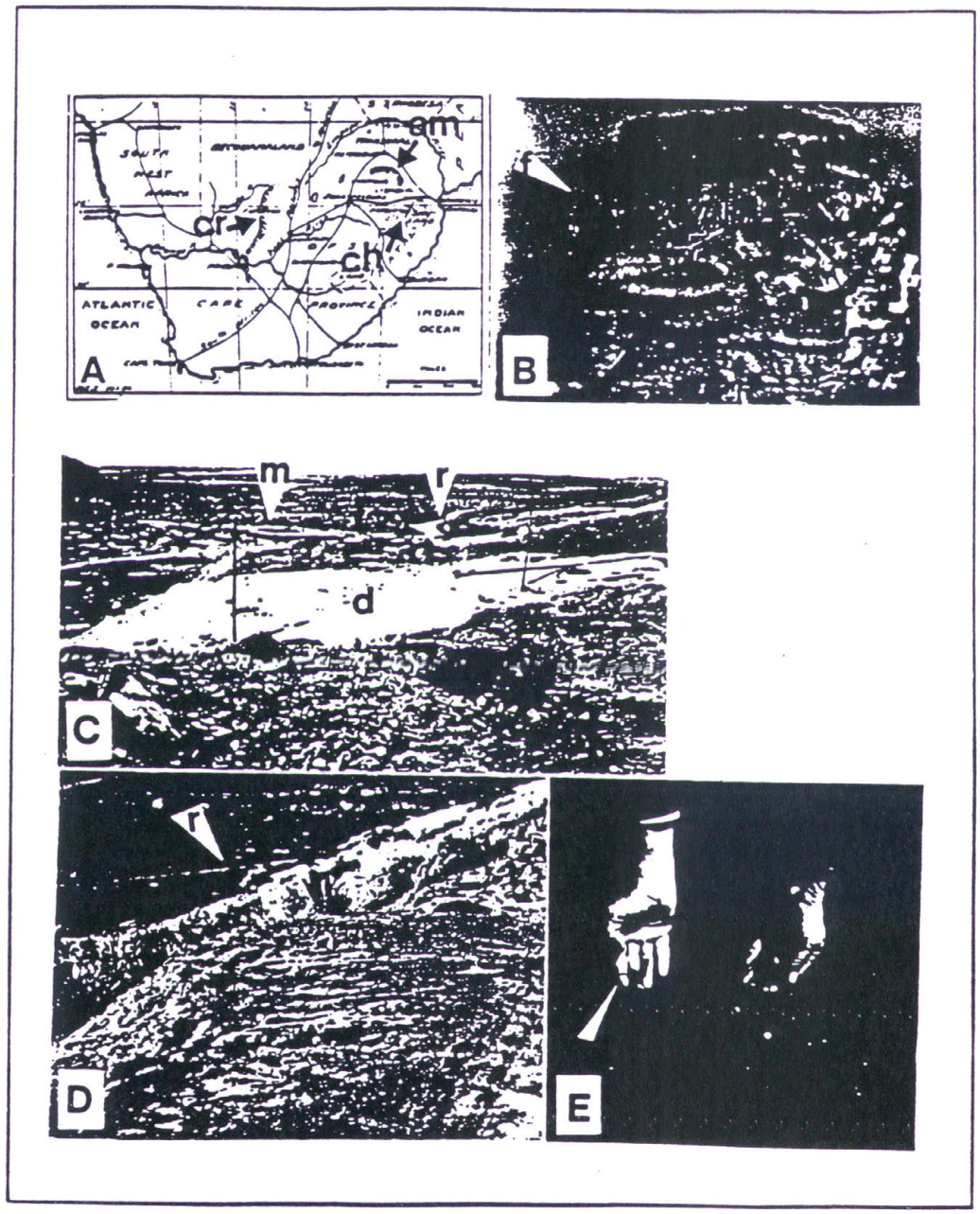


Figure 1. Relationship between percent deaths in cohort due to asbestosis and proportion of peritoneal mesothelioma deaths among total mesothelioma deaths.
Mt. Sinai cohorts:
(A) Insulators - NY, NJ
(B) Insulators - National Survey
(C) UNARCO - amosite
(D) Thetford - chrysotile

Figure 2



FACTORS INFLUENCING INDIVIDUAL RESPONSE TO ASBESTOS: EFFECTS OF SMOKING AND ASBESTOS ON THE DEVELOPMENT OF LUNG CANCER

BROOKE T. MOSSMAN and RHONDA GILBERT

Department of Pathology, University of Vermont College of Medicine, Burlington, VT 05405

Smoking and exposure to other pollutants in the workplace are important cofactors in the development of bronchogenic carcinoma in asbestos workers. Because most asbestos workers have been smokers, the differential effects of asbestos and cigarette smoke in the tumorigenic process have been difficult to address in epidemiologic studies, but experimental work suggests several mechanisms of cocarcinogenesis. For example, smoking inhibits the clearance of asbestos fibers from rodent lungs. Secondly, polycyclic aromatic hydrocarbons (PAHs) in cigarette smoke bind to both crocidolite and chrysotile asbestos as well as other nonasbestos fibers and particles. Thus, PAH-coated particulates increase uptake of these chemical carcinogens by tracheobronchial epithelial cells. Lastly, asbestos acts as a classical promoter in carcinogenesis by inducing biochemical and proliferative alterations in epithelial cells of the respiratory tract whereas PAHs and other genotoxic chemicals in cigarette smoke appear to act as initiators.

INTRODUCTION

Occupational exposure to asbestos is associated with an increased risk of diffuse malignant mesothelioma (DM), a tumor arising in the pleura and peritoneum, and lung cancers, neoplasms most commonly developing from epithelial cells lining either the airways (i.e. bronchogenic carcinomas) or the airspaces of the lung (i.e. peripheral carcinomas). Whereas DMs are an extremely rare tumor (approximately 1500 are diagnosed yearly in the U.S.), lung cancers are more prevalent in that approximately 130,000 occur per year, predominately in smokers in the general population (Hughes and Weill, 1986). Previous analysis of asbestos-exposed occupational cohorts indicated a multiplicative interaction

between smoking and exposure to asbestos in the development of this lesion (Saracci, 1987). However, a more recent examination shows that these interactions may be additive or less than additive in certain workers (Saracci, 1987). Regardless, the paucity of tumors in nonsmoking asbestos workers (Saracci, 1987) and data showing that asbestos is neither mutagenic nor genotoxic in conventional assays (Shelby, 1987) argue against the concept that asbestos is an initiator in the induction of lung tumors. Thus, research in this laboratory has focused on elucidating mechanisms of tumor promotion by asbestos (reviewed in Mossman et al., 1985). Polycyclic aromatic hydrocarbons (PAHs) bind to crocidolite and chrysotile asbestos, a process which appears to facilitate uptake of chemicals by tracheobronchial epithelial cells, the cells which give rise to lung tumors (Eastman et al., 1983). Moreover, the amount of PAH retained in the DNA fraction of the cells is increased when PAHs are introduced on the surfaces of fibers as opposed to when they are solubilized in medium. These results suggest that asbestos fibers (defined as a $\geq$ 3:1 length to diameter ratio) act as vehicles for delivery of carcinogenic PAH to epithelial cells of the respiratory tract. Whether or not other fibers (defined as a $\geq$ 3:1 length to diameter ratio) and particles have this capacity is unclear. In work here, we examined several fibers (Chrysotile A, attapulgite, Code 100 fiberglass) and particles (antigorite, kaolin) comparatively for their ability to adsorb the radioactively-tagged PAH, benzo(a)pyrene [B(a)P] after vapor-coating, a process simulating coating of particles with chemicals during smoking. In an attempt to determine the importance of geometry in transfer of PAH to cells, we then measured uptake of ^{14}C-B(a)P by hamster tracheal epithelial (HTE) cells after addition to cultures of equal surface areas of Chrysotile A and antigorite, its nonfibrous, chemically identical analog.

MATERIALS AND METHODS

Particulates. The types, sources and surface areas of fibers (Chrysotile A, attapulgite, Code 100 fiberglass) and particles (antigorite, kaolin) used here are presented in Table 1. The chemical composition and size dimensions of fibers and particles have been reported previously (Mossman and Craighead, 1982; Woodworth et al., 1983). The surface areas of particulates were determined by nitrogen adsorption (Micromeritics Instrument Co., Norcross, GA).

Vapor-coating of particulates. Dr. Michael Placke (Battelle-Columbus Laboratories, OH) assisted in these experiments. In brief, 7,10 ^{14}C-B(a)P (Amersham Chemical Co., Arlington Heights, IL, Specific Activity = 30-50 mCi/mmol) was combined in a 1:10 ratio with cold B(a)P (Aldrich Chemical Company, Milwaukee, WI) and coated on sterilized glass vials by

evaporation under nitrogen gas. Particulates were added at equal surface areas, and vials were heated by thermocoupling from 27°C to 100°C for 2 hrs. At this time, vials were allowed to cool slowly and then stored at 4°C in the dark to prevent degradation of B(a)P. To determine the relative amounts of ^{14}C-B(a)P adsorbed to equal surface areas of particulates, dusts were resuspended at equal surface areas in serumless Ham's F12 Medium (GIBCO, Grand Island, NY) and aliquots (N = 8 to 12 per group) counted in Ecolume (ICN Chemicals, Irvine, CA) in a scintillation counter.

Cells. The HTE cell line used in studies here has been described previously (Mossman et al., 1980). Cells were grown and plated for assays in 60 mm dishes containing Ham's F12 medium and 10% fetal calf serum (GIBCO, Grand Island, NY) with 50 μ/ml gentamycin. At confluency, serum-containing medium was removed and replaced with serumless medium prior to the addition of ^{14}C-B(a)P-coated Chrysotile A or antigorite, the nonfibrous, chemically identical analog of chrysotile. After addition of Chrysotile A or antigorite at equal surface areas (0.65 and 1.30 μg Chrysotile A/cm^2 dish and .506 and 1.01 μg antigorite/cm^2 dish) to serumless medium, medium was removed at 1,2,4 and 8 hours after introduction of materials. After rinsing of dishes twice with serumless medium to remove non-cell associated ^{14}C-B(a)P, cells were scraped from dishes (N = 4 to 6 per group per time point) using a rubber policeman, pelleted at 1,000 x g, and cell pellets dissolved in Ecolume for scintillation spectrometry. Each experiment was repeated in duplicate or triplicate. The Student's t-test adjusting for multiple comparisons between groups was used to determine statistical significance.

RESULTS

Figure 1 shows the amount, expressed as cpm ^{14}C-B(a)P, associated with equal surface areas of fibers at 1 and 2 X concentrations of each fiber type. At low concentrations (clear bars) less ^{14}C-B(a)P was associated with antigorite in comparison to Chrysotile A, but no statistical differences were observed between Chrysotile A and other groups at either lower or 2-fold higher (dotted bars) concentrations. These data indicate that adsorption of ^{14}C-B(a)P to respective fibers and particles is a function of surface area rather than the geometry or chemical composition of dusts.

^{14}C-B(a)P-coated Chrysotile A and antigorite at equal surface areas (as depicted in Figure 1) then were added at two concentrations to plates of HTE cells to determine whether differences in uptake of ^{14}C-B(a)P by cells were observed at periods from 1 to 8 hours after introduction of dusts. Results illustrated in Figure 2 show that Chrysotile A (open squares) at both higher and lower concentrations significantly increased ($p < .05$) uptake of ^{14}C-B(a)P by HTE cells at 1 hour. Differences at other time points were not

statistically significant. These experiments indicate that fibrous geometry facilitates initial uptake of PAHs by cells but has no unique effects on metabolism or removal of PAHs from cells.

DISCUSSION

The cocarcinogenic effects of asbestos and smoking on the development of bronchogenic carcinomas have been documented in various occupational cohorts (Saracci 1977, 1987). However, study of the mechanisms of cocarcinogenesis has been difficult in inhalation models of disease as exposure of rodents to asbestos and cigarette smoke in combination results in the rapid development of asbestosis which often becomes lethal before lung tumors are observed (Wehner et al., 1975). When PAHs such as B(a)P are administered by intratracheal instillation to rodents, tumors develop with use of B(a)P, but not with use of asbestos alone. Moreover, use of B(a)P-coated asbestos or B(a)P in combination with asbestos results in increased numbers of tumors when compared to numbers developing after exposure to B(a)P alone (Miller et al., 1965; Pylev and Shabad, 1973; Smith et al., 1968). These observations suggest that asbestos facilitates delivery and uptake of B(a)P by tracheobronchial epithelial cells, the progenitor cells of bronchogenic carcinomas. This hypothesis has been confirmed in HTE cells exposed in vitro to either crocidolite or chrysotile asbestos coated with ^{14}C-B(a)P (Eastman et al., 1983). In these previously published studies, however, ^{14}C-B(a)P was solubilized in acetone and mixed with equal mass concentrations of fibers before evaporation of acetone from mixtures. Here, we sought a more authentic route (i.e. vapor-coating of fibers) resembling what might occur during smoking. Moreover, we examined equal surface areas of chemically dissimilar fibers and particles after coating with ^{14}C-B(a)P to determine whether asbestos fibers (i.e. Chrysotile A) were more apt to bind carcinogenic PAHs than nonasbestos fibers and particles. Results show that Chrysotile A has no more affinity for PAHs than other fibers and particles when compared at equal surface areas. Although antigorite bound less ^{14}C-B(a)P than Chrysotile A at the lower concentration tested, this observation was not confirmed at the higher concentration. At 1 hour after introduction of ^{14}C-B(a)P-coated antigorite into HTE cell cultures, less uptake of ^{14}C-B(a)P was observed in comparison to that seen with equal surface areas of Chrysotile A. On the one hand, this phenomenon might reflect the slight differences observed in amounts of ^{14}C-B(a)P bound initially to Chrysotile A vs. antigorite as seen in Figure 1. Alternatively, Lakowicz and Bevan (1980) demonstrated in rat liver microsomes that asbestos, in comparison to a number of chemically different nonfibrous minerals, facilitated immediate uptake of B(a)P by membranes as determined by fluorescence spectroscopy. Our results show that the amounts of ^{14}C-B(a)P in HTE cells after 1 hour of exposure to carcinogen-coated dusts is

comparable when either Chrysotile A or its chemically identical, nonfibrous analog, antigorite, is used as a vehicle for introduction of B(a)P. A good correlation between the surface areas of mineral fibers and the binding of the chemical carcinogens, nitrosonornicotine and N-acetyl-2-aminofluorene also has been noted by Harvey and colleagues (1984). However, equal surface areas of glass wool, glass micro fibers and amphibole types of asbestos appeared to bind less B(a)P than chrysotile or attapulgite in their studies (note: statistical analyses of data were not provided). Figure 3 shows other plausible mechanisms of asbestos-associated cocarcinogenesis in smokers. On the one hand, asbestos and other particulates encountered in cigarette smoke or in the workplace may adsorb lipophilic PAHs which may be solubilized in lung surfactant. After release of PAH from particulates into the bronchial lining layer (BLL), adsorption of PAH by epithelial cell membranes then may occur. Gerde and Scholander (1987) suggest that after adsorption of phospholipids, fibers, in comparison to particles, are more apt to act as short-circuits for transfer of PAHs across the aqueous phase of the BLL. Indeed, fibers or components of cigarette combustion may cause ciliostasis and the induction of squamous metaplasia, phenomena which may impair their clearance from the lung (Woodworth et al., 1983). In support of this concept, studies by McFadden and colleagues (1986) show that exposure to cigarette smoke causes increased retention of asbestos in rodent lungs. Human (Hesterberg et al., 1988) and hamster (Mossman et al., 1983) tracheobronchial epithelial cells do not exhibit significant increases in aneuploidy nor breakage of DNA after exposure to either chrysotile or crocidolite asbestos. Therefore, it is highly unlikely that asbestos is an initiator in the development of bronchogenic carcinoma. However, its ability to cause alterations in proliferation and normal differentiation of cells suggests its importance in the process of tumor promotion in lung. In this regard, asbestos causes inflammatory changes in tracheal grafts (Topping et al., 1980) and generation of active oxygen species such as superoxide (0_2) from alveolar macrophages (Hansen and Mossman, 1987) in a manner similar to classical phorbol tumor promoters in the mouse skin model of carcinogenesis. Asbestos also appears to affect metabolism of PAH to forms which can interact with the DNA of cells. For example, crocidolite asbestos catalyzes the oxidation of 6-hydroxybenzo(a)pyrene, a metabolic intermediate of B(a)P to the highly reactive 6-oxobenzo(a)pyrene radical which may be important in carcinogenicity and toxicity (Graceffa and Weitzman, 1987). Exposure of HTE cells to crocidolite in combination with B(a)P also appears to increase the activity of aryl hydrocarbon hydroxylase (AHH) enzymes (Mossman and Craighead, 1981).

CONCLUSIONS

In conclusion, this paper summarizes several mechanisms of cocarcinogenesis involving smoking and asbestos which may be important in explaining the increased incidence of lung cancers in asbestos workers (Saracci 1977, 1987). Whether any asbestos type alone can cause lung cancer in nonsmokers is uncertain since lung cancers are rarely found in nonsmoking asbestos workers. To suggest that asbestos causes lung cancers in smokers or nonsmokers in the general population would be misleading in view of the fact that airborne concentrations of asbestos reported in schools and buildings are several thousand-fold lower than estimates in past workplace situations (these proceedings).

ACKNOWLEDGEMENTS

This work was supported by grants from the National Cancer Institute, National Heart, Blood and Lung Institute and National Institute of Environmental Health Sciences. Virginia Kelleher and Judith Kessler provided valuable technical assistance.

REFERENCES

Eastman, A., Mossman, B.T., and Bresnick, E. (1983). Influence of asbestos on the uptake of benzo(a)pyrene and DNA alkylation in hamster tracheal epithelial cells. Cancer Res. 43, 1251-1256.

Gerde, P., and Scholander, P. (1987). A hypothesis concerning asbestos carcinogenicity: the migration of lipophilic carcinogens in adsorbed lipid bilayers. Ann. Occup. Hyg. 31, 395-400.

Graceffa, P., and Weitzman, S.G. (1987). Asbestos catalyzes the formation of the 6-oxobenzo(a)pyrene radical from 6-hydroxybenzo(a)pyrene. Arch. Biochem. Biophys. 257, 481-484.

Hansen, K., and Mossman, B.T. (1987). Generation of superoxide (O_2^-) from alveolar macrophages exposed to asbestiform and nonfibrous particles. Cancer Res. 47, 1681-1686.

Harvey, G., Page, M., and Dumas, L. (1984). Binding of environmental carcinogens to asbestos and mineral fibres. Brit. J. Indust. Med. 41, 396-400.

Hesterberg, T.W., Maness, S.C., Kodama, Y., and Boreiko, C.J. (1988). Effects of asbestos on normal human bronchial epithelial cells in culture. Proceedings: N.A.T.O. ARW on "Effects of Mineral Dusts on Cells", p. 9 (abstract), Sherbrooke, Quebec.

Hughes, J.M., and Weill, H. (1986). Asbestos exposure - quantitative assessment of risk. Am. Rev. Resp. Dis. 133, 5-13.

Lakowicz, J.R., and Bevan, D.R. (1980). Benzo(a)pyrene uptake into rat liver microsomes: effects of adsorption of benzo(a)pyrene to asbestos and non-fibrous mineral particulates. Chem. Biol. Inter. 29, 129-138.

McFadden D., Wright J.L., Wiggs, B. ,and Churg, A. (1986). Smoking inhibits asbestos clearance. Am. Rev. Resp. Dis. 133, 372-374.

Miller, L., Smith, W.C., and Berliner, S.W. (1965). Tests for effect of asbestos on benzo(a)pyrene carcinogenesis in the respiratory tract. Ann. N.Y. Acad. Sci. 132, 489-500.

Mossman, B.T., Cameron, G.S., and Yotti, L. (1985). Cocarcinogenic and tumor promoting properties of asbestos and other minerals in tracheobronchial epithelium. In "Cancer: A Comprehensive Survey: Cancer of the Respiratory Tract, Predisposing Factors" (M.H. Mass, D.G. Kaufman, J.M. Siegfried, V.E. Steele, S. Nesnow, Eds.). pp. 1446-1445, Raven Press, New York.

Mossman, B.T., and Craighead, J.E. (1981). Mechanisms of asbestos carcinogenesis. Environ. Res. 25, 269-280.

Mossman, B.T., and Craighead, J.E. (1982). Comparative cocarcinogenic effects of kaolin, hematite, carbon and crocidolite asbestos in implanted tracheal organ cultures. In "Inhaled Particles", pp. 572-578, Pergamon Press, London.

Mossman, B.T., Eastman, A., Landesman, J.M. and Bresnick, E. (1983). Effects of crocidolite and chrysotile asbestos on cellular uptake, metabolism and DNA after exposure of hamster tracheal epithelial cells to benzo(a)pyrene. Environ. Health Perspect. 51, 331-335.

Mossman, B.T., Ezerman, E.B., Adler, K.B., and Craighead, J.E. (1980). Isolation and spontaneous transformation of hamster tracheal epithelial cells. Cancer Res. 40, 4403-4407.

Pylev, L.N., and Shabad, L.M. (1978). Some results of experimental studies in asbestos carcinogenesis. In "Biological Effects of Asbestos" (P. Bogovski, J.C. Gilson and V. Timbrell, Eds.). I.A.R.C. Publ. #8, pp. 99-105, International Agency for Research on Cancer, Lyon.

Saracci, R. (1977). Asbestos and lung cancer: an analysis of the epidemiological evidence on the asbestos-smoking interaction. Int. J. Cancer 20, 323-331.

Shelby, M.D. (1988). The genetic toxicity of human carcinogens and its implications. Mutation Res. 204, 3-15.

Smith, W.E., Miller, L., and Churg, J. (1968). Respiratory tumors in hamsters after intratracheal benzo(a)pyrene with and without asbestos. Proc. Am. Assoc. Cancer Res. 9, 65 (abstract).

Topping, D.C., Nettesheim, P., and Martin, D. (1980). Toxic and tumorigenic effects of asbestos and tracheal mucosa. J. Environ. Path. Toxicol. 3, 261-275.

Woodworth, C.D., Mossman, B.T., and Craighead, J.E. (1983). Induction of squamous metaplasia in organ cultures of hamster trachea by naturally occurring and synthetic fibers. Cancer Res., 43, 4906-4912.

Table 1. Types of fibers and particles examined after vapor coating.

PARTICULATES	SOURCE	SURFACE AREA[a] (m^2/gm)
CHRYSOTILE A	UICC (International Union Against Cancer) Reference Sample	19.8
ATTAPULGITE[b]	Clay Mineral Society, Columbia, MO	254
CODE 100 FIBERGLASS	Manville Corp. (Denver, CO)	4.0
ANTIGORITE	Wards Scientific Est., Rochester, NY	26.2
KAOLIN	Georgia Kaolin Co., Elizabeth NJ	9.4

---

a Determined by Micromeritics Instrument Co. (Norcross, GA) using nitrogen adsorption.

b The correct mineralogic term for attapulgite is palygorskite.

FIGURE LEGENDS

1. Amounts of ^{14}C-B(a)P (cpm) adsorbed to equal surface areas of fibers (Chrysotile A, attapulgite, fiberglass) and particles (antigorite, kaolin). Each dust was evaluated at two concentrations. Clear bars represent low vs. 2-fold higher (stippled bars) concentrations. * = significantly less ($p < .05$) in comparison to equal surface area of Chrysotile A (Student's t-test).

2. Amounts of ^{14}C-B(a)P (cpm) in HTE cells at various periods after introduction of Chrysotile A (-□- = 1.30 $\mu g/cm^2$ dish; –□– 0.65 $\mu g/cm^2$ dish) or antigorite (c -•- = 1.01 $\mu g/cm^2$ dish; –•– = .506 $\mu g/cm^2$ dish) at equal surface areas. * = significantly greater ($p < .05$) in comparison to equal surface areas of Chrysotile A (Student's t-test).

3. Schematic diagram depicting possible mechanisms of asbestos-induced cocarcinogenesis in smokers. (from Mossman, B.T. and Craighead, J.E., (1987). Mechanisms of asbestos-associated bronchogenic carcinoma, In: "Asbestos-Related Malignancy", (K. Antman and J. Aisner, Eds.), pp. 137-150, Grune & Stratton, Inc., Orlando.

Figure 1

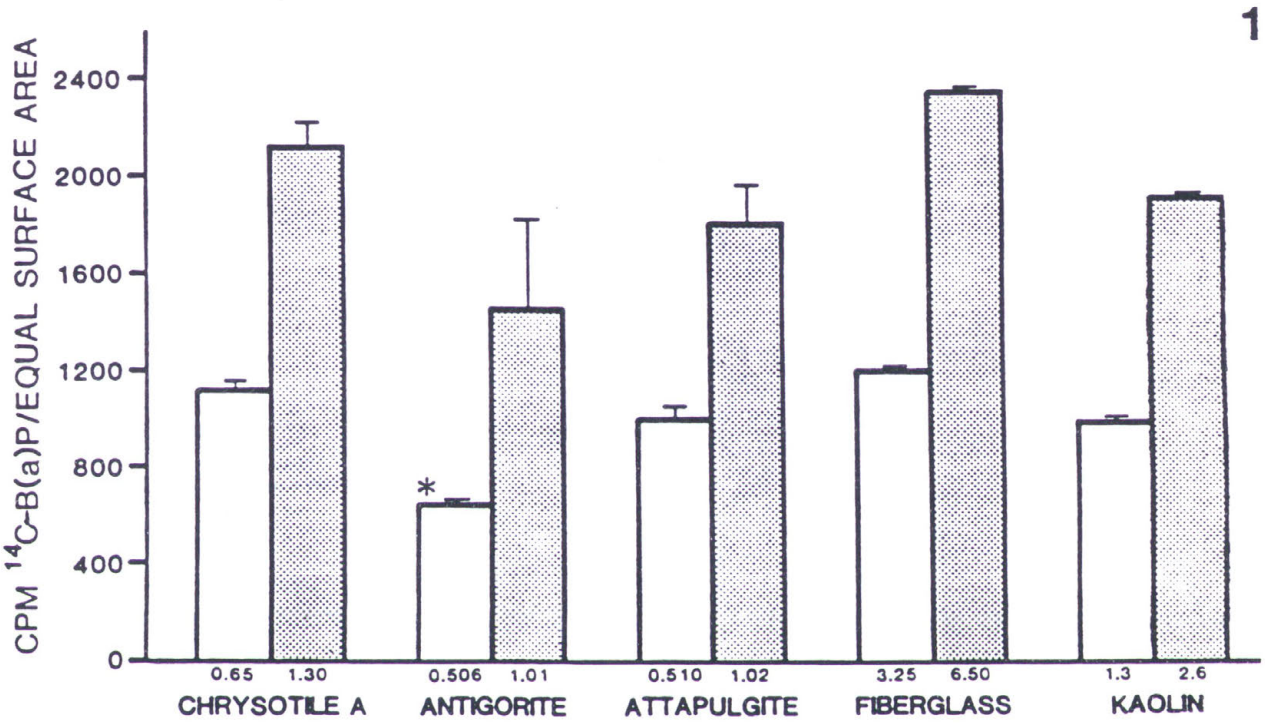

1
CPM ^{14}C-B(a)P/EQUAL SURFACE AREA
2400
2000
1600
1200
800
400
0
*
0.65
1.30
CHRYSOTILE A
0.506
1.01
ANTIGORITE
0.510
1.02
ATTAPULGITE
3.25
6.50
FIBERGLASS
1.3
2.6
KAOLIN

Figure 2

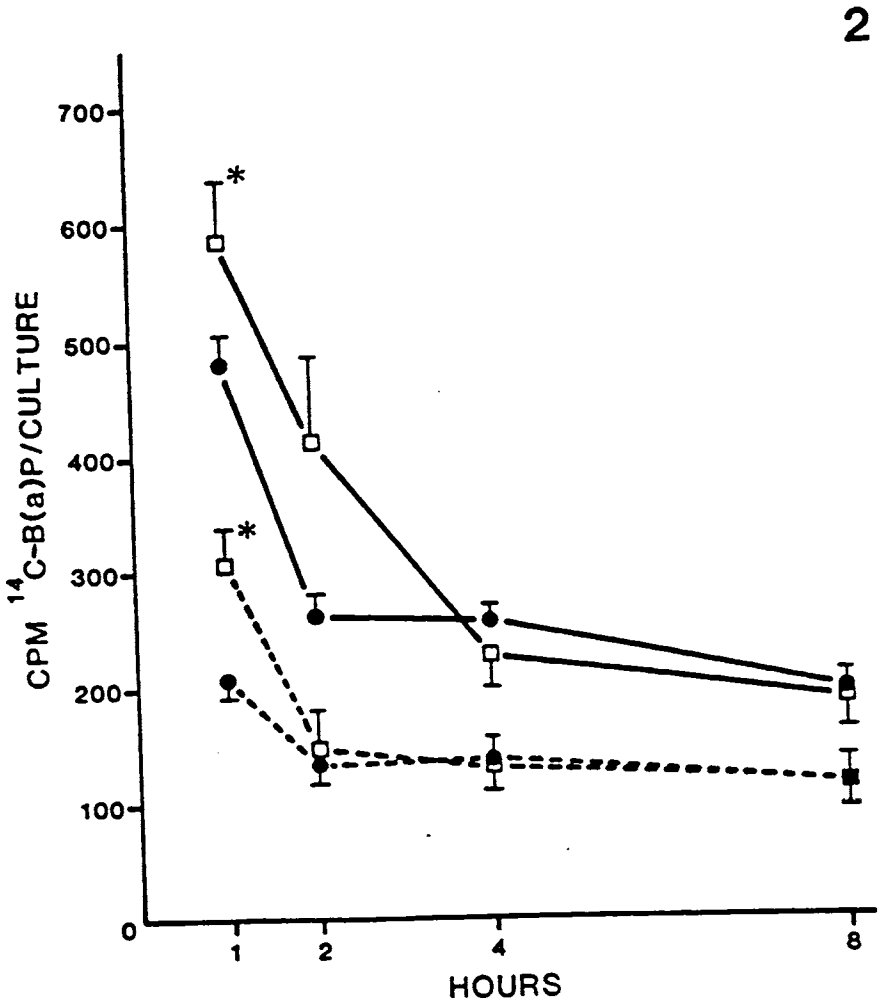
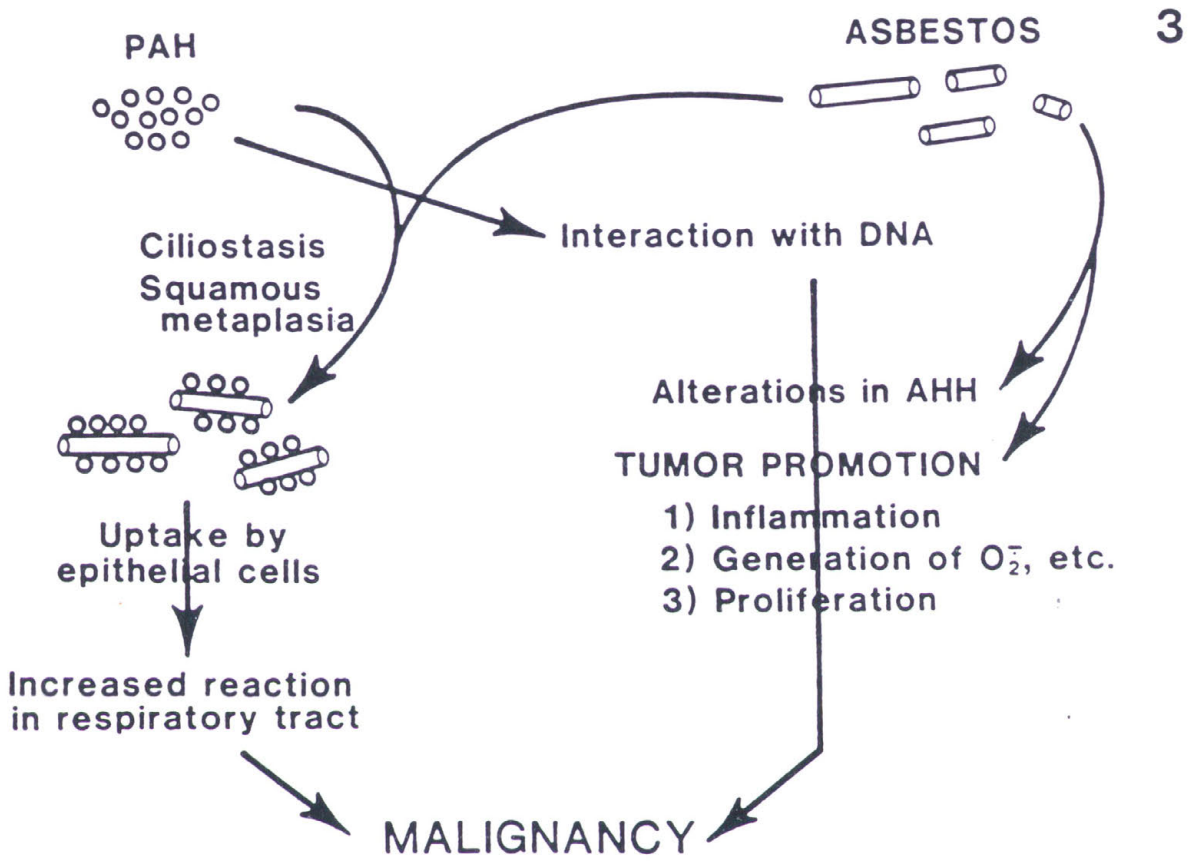


Figure 3


3
PAH
ASBESTOS
Ciliostasis
Squamous
metaplasia
Interaction with DNA
Alterations in AHH
TUMOR PROMOTION
1) Inflammation
2) Generation of O_2^-, etc.
3) Proliferation
Uptake by
epithelial cells
Increased reaction
in respiratory tract
MALIGNANCY

ASBESTOS MATERIAL INVENTORY, CONTROL CONCEPTS, AND RISK COMMUNICATION

ROBERT N. SAWYER

Guilford, Connecticut

BACKGROUND

Materials in buildings that can contain asbestos include structural fireproofing, acoustical finishes, thermal insulation on heating systems, sheet products, tiles and felts, and debris or dust (EPA 1988, NY City 1988). Any of these materials will release component particulates, including asbestos fibers, into building environments when sufficiently disrupted or disturbed.

THE BASIC CONCERN

Exposure to airborne asbestos fibers is known to cause pleural scarring; the fibrotic lung disease asbestosis; an increased risk of lung cancer (predominantly in cigarette users); and mesotheliomas of the chest and abdominal cavity. In studies of exposed populations asbestos related effects generally appear in proportion to integrated dose and follow a delay or latency period, typically of decades, following the initiation of exposure. The incidence of asbestosis and malignancy has been extreme in those workers who processed, fabricated, or applied asbestos products (Cooke 1927, Merewether 1930, Doll 1955, Wagner 1960, Selikoff 1980). Attributable effects have also been found in some cases with neighborhood and domiciliary exposure (Newhouse 1965, Anderson 1976).

In 1972 attention began to focus on risks potentially associated with applications of asbestos in structures with documentation of exposures of workers and bystanders during spraying of asbestos bearing fireproofing (Reitze 1972). In 1973, the Environmental Protection Agency initiated regulation of application, removal, and disposal of friable asbestos materials in buildings (EPA 1973). Studies of possible risk from disturbance of asbestos materials already existing within buildings were contemporary to these actions (Lumley 1971, Sawyer 1975, Nicholson 1976). Schools, with a readily defined population with extended life expectancy (i.e., latency accommodation) became sites of major interest. Some school building studies, using indirect sample preparation and analysis by transmission electron microscopy (TEM), suggested an association between the presence of friable

asbestos materials and increased levels of airborne asbestos, and excess risk was attributed to these levels (Nicholson 1978, Constant 1983).

RESPONSE OF THE ENVIRONMENTAL PROTECTION AGENCY

In 1979, following a citizens petition by the Environmental Defense Fund (EDF), the Environmental Protection Agency released the first of a series of guidance documents (the Orange Book), intended to encourage voluntary identification and control of asbestos in schools (EPA 1979). This technical assistance program, in the estimation of EPA, found only limited success. EPA then moved to regulate asbestos in school buildings, and did so in 1982 (EPA 1982). The regulation required inspections for friable asbestos materials and notification of each parent, teacher, and school employee. The EPA, in thousands of sites throughout the nation, was attempting to regulate a nearly ubiquitous microparticulate that possessed both chronic and malignant disease potential, and a widely perceived sinister reputation (Brodeur 1973). In an effort to follow prudent health policy, EPA selectively utilized risk models considered conservative, or pessimistic (Walsh 1978, EPA 1982, Langer 1984). The technical support document for the 1982 asbestos in schools rule, the rule itself, and the complementary guidance document (Blue Book) translated this theoretical risk model into an operative guidance of no safe limit for asbestos exposure (EPA 1983). The psychologic impact of this concept was amplified by the absence of relative risk information for a rough approximation of significance; other life risk data was not provided for comparison. Communication of risk information is always difficult, and particularly when risk assessment has been performed in a vacuum (Mason 1986, Fisher 1989). Without benchmarks, it is understandable that to many people the mere presence of asbestos meant serious risk (Muir 1985).

In effect, the 1982 EPA inspection and notification rule placed the difficult task of risk assessment and the equally complex risk management process upon the thousands of school boards (EPA 1982, Ruckelshaus 1983). To the parents and school boards the epidemiologic data were essentially unintelligible, but the message was clear: asbestos presence was cancer risk. In hindsight it is clear that few, if any, anticipated the chaos of overreaction and control efforts that now seems an obvious consequence (Smith 1982). Further, problems encountered could not be readily perceived by EPA since no provision had been created for central compilation of the assessment and control actions of the individual school administrations. School control activity, and associated troubles, only began to be officially recognized during the 1984 hearings on a Service Employees International Union petition for extension of asbestos regulation into other buildings (SEIU 1983, EPA 1984). The significant difficulties arising from the 1982 EPA schools program led Congress to create the 1986 Asbestos Hazard Emergency Response Act (AHERA),

tasking EPA with regulatory and guidance reforms on a specific time schedule. In late 1987, EPA responded to AHERA with comprehensive regulations for asbestos in school buildings (EPA 1987a).

Our national response to the presence of asbestos in schools certainly surpasses that of other nations (Ontario 1984). It has also been termed excessive, alarmist, and responsible for creating the pressures that have led to fear and overreaction in schools and other sectors (Bennett 1985, Zurer 1985, Lee 1878, Whitaker 1989). Although the main protagonist in this situation is considered the Environmental Protection Agency (GAO), other driving forces can also be identified. Personal injury settlements (Borel 1973), the financial reorganization of a major corporation (Manville), highly effective media attention (Brodeur 1973, Bergman 1980), and a knowledgeable and successful plaintiff bar (Lee 1989) have sensitized the nation to asbestos hazard. Widespread awareness of asbestos presence and possible exposures, and a popular conception of definite cancer risk under any condition have provided significant reference for both the media and advocacy groups.

CONCERNS OF ECONOMIC SIGNIFICANCE

This asbestos anxiety has been particularly apparent in those who work or live in buildings that contain these minerals. Building owners and operators face major concerns associated with asbestos material presence in a structure. They must confront the requirements of regulatory compliance, increased costs of operations and maintenance, possible claim assertions of asbestos related disease, and decreases in real estate valuation. The awareness and concern of asbestos associated risk has continued to generate intense political and regulatory activity at federal, state, and local levels. Compliance with complex, frequently ambiguous regulation has become difficult, if not baffling (EPA 1973; GAO 1985; EPA 1987a; CA 1986; NJ 1986; NY State 1987; NY City 1985, 1989). In some structures with highly friable and frequently disturbed material previously uncomplicated maintenance, custodial, and renovation tasks have become burdensome (Morse 1988). Asbestos personal injury litigation on behalf of employees exposed to asbestos in shipyards and a number of other industries has already led to massive awards and defense costs. The same causes of action are available against building owners and other employers. These impose potential liability upon parties controlling environments in which hazardous conditions are present (Lee 1989). Judgment was made in favor of a building occupant suffering from mesothelioma for personal injury reputedly resulting from exposure to asbestos in an office environment (Layne). That the presence of asbestos in a building can depress market value and utilization is well documented (Berg 1988, Richman 1988, Guenther 1986, Hinds 1986). Many large lenders and investors have become increasingly reluctant to be involved with

buildings that contain asbestos. These current pressures in the real estate marketplace, combined with the presence of a well developed asbestos abatement industry, has rendered risk assessment essentially obsolete in most removal decisions. This effect is so great that often the type, quality, or quantity of the asbestos materials seem nearly irrelevant. Increasingly, major corporations are making the decision to remove asbestos materials on the basis of market factors alone (Morse 1988). Despite the reassuring evidence that airborne asbestos fibers are not entering occupied spaces in office towers and large residential buildings (Burdett 1988, 1989), the real estate market demonstrates significant concern over asbestos presence, and the removal of asbestos containing materials from buildings continues.

A debate is currently in progress regarding creation of requirements similar to AHERA for public and commercial buildings. A February, 1988, EPA report to Congress recommends temporizing the application of rules to other buildings, but some form of federal regulatory action appears inevitable (EPA 1988).

INCREASED RISK ASSOCIATED WITH REMOVAL

The current vogue of removal is increasing rather than reducing risk (Sawyer 1984). Inadequately controlled removals can create extreme conditions of contamination that can introduce asbestos fibers into the building environment (Morse 1988, Sawyer 1977, 1979). Opinions of EPA inspectors on competence of control on abatement worksites are not reassuring. Some data suggest that airborne fiber levels in structures actually increase for periods of many days following large asbestos removal projects (Cain 1987). Further, the transported bagged waste, compacted at disposal sites, is considered by experienced observers to represent not only a current and significant source of released fibers but also a major future problem (Copeland 1987).

Personnel directly involved in asbestos material removal are at particular risk. Excessive worker exposure under compromised worksite conditions seems extremely likely. Removal workers, members of various trades who immediately access the work site (typically untrained and unprotected), disposal site workers, and other abatement personnel repeatedly face potential exposure in project after project. Such cumulative exposure can exceed, by orders of magnitude, that of building and service personnel.

CONCEPTS OF INVENTORY, CONTROL, AND RISK COMMUNICATION

Owners and operators of asbestos bearing buildings are now grappling with unanticipated and difficult issues that are often complex. Further, evaluations of the usually costly alternative responses are typically complicated by haste and misinformation. Such

decisions can be more rationally constructed if control requirements can be anticipated and defined. The six areas that should be addressed are survey and inventory, material management, removal, waste disposal, technical and professional support, and risk communication.

1. SURVEY AND INVENTORY: Comprehensive inventory information with careful survey, sampling, and documentation is the foundation of a competent control program and defines the scope of control. Inventory dimensions include material identification, asbestos content and fiber type, system location or utilization, accessibility, condition, and quantity. Key plans and computer assisted inventory can enhance interpretation and retrieval of complicated data. The inventory information must be clearly understandable and readily available to the owner, maintenance and custodial personnel, regulators, and asbestos control contractors. When quantity, accessibility, and condition of each asbestos containing material have been simultaneously considered appropriate control strategy can be developed. Such methodologies should maximize architectural and engineering parameters to develop a clear picture of management requirements and priorities (Sawyer and Morse 1986). Survey and inventory can include air sampling. Air sampling has been used with effectiveness to describe the steady state conditions within structures (Corn 1986, EPA 1988).

2. MATERIAL MANAGEMENT: Asbestos management has been appropriately described as the husbandry of asbestos fibers from discovery to disposition (Bomier 1987). Asbestos material management involves the prevention of unnecessary material disturbance, the control of necessary work, controlled response to unanticipated events, and scheduling the eventual removal and appropriate disposal of asbestos materials. This typically requires separate procedures for building operations, contractor work, and major abatement projects. Special operating and maintenance procedures, from trivial to complex, are required as long as asbestos remains in a structure. Air sampling can describe levels encountered within categories of materials and disturbance activities to provide guidance for control and personnel protection, documentation for regulatory compliance, and risk communication (Sawyer 1977, 1979).

3. REMOVAL: The mere presence of asbestos containing material does not mean that removal is immediately required, and, in most structures, asbestos materials need not be removed until renovation or demolition (EPA 1985). However, the anticipation of the eventual removal of asbestos materials from a building should

be an integral part of a control program. The major decision on asbestos material removal is not whether to remove, but when to remove. EPA currently mandates control of removal and disposal of friable asbestos materials in conjunction with demolition or major renovation in buildings and, in schools, removal in instances of severe damage and easy accessibility (EPA 1973, EPA 1987a). Building owners may face their greatest risks from conditions created during a major removal project. The three essentials of effective control during removal are clearly defined project specifications or work procedures, the selection of a competent contractor, and skilled monitoring and documentation of the removal process. Generic reference specifications and procedures are available through the National Institute of Building Sciences (NIBS).

4. WASTE DISPOSAL: Management procedures and removals generate waste. The transport and disposal of asbestos waste is regulated at the federal, state and local levels, and sometimes without consistency. EPA requires burial in a regulated landfill (EPA 1973). The current interpretation of regulation is that responsibility for the material will remain with the generator of such waste, the building owner. This potentially eternal liability can be eliminated. The thermal conversion of asbestos to glass has proven feasible, and has been designated by the EPA as an alternative to burial (EPA 1987b). This method will be economically comparable to burial in urban areas with limited local disposal site capacity (Roberts 1988).

5. TECHNICAL AND PROFESSIONAL SUPPORT: The fifth area essential to control is that of technical and professional program support functions. These include the selection, as needed, of competent providers for sampling and analysis of materials and air, consultation for specific tasks, medical services, legal counsel, communications, and personnel relations. The scope of these services will vary with specific site inventory and management requirements.

6. RISK COMMUNICATION: When available and organized, inventory data and control policy should be communicated to management, regulators, tenants, building personnel, and building users as appropriate to their needs. Communication is best when responsible and rapid. The critical effect of competent communication cannot be overestimated and significant lessons have been learned in risk communication. Such unfamiliar information must be accompanied by comparisons to more familiar life risks to begin the process of recognition, understanding, and rational reaction (Fisher 1989).

DISCUSSION

1. Better appreciation of both the deficiencies and strengths of earlier studies, recent information on risk and exposure, can now more clearly define objectives, and limitations of asbestos control effort (McDonald 1985; Omenn 1986; Weill 1986; EPA 1988; Hughes 1986, 1988; Langer 1987; Wagner 1988, HEI 1988, EEPC 1989). This current information on the reassessment of risk information should be continuously integrated into the policy and administration of existing EPA, state, and local regulations, and become the basis for future regulation and guidance.

2. There appears to be an imbalance between actual structural asbestos health risk and the national control response. This imbalance is severe and diverts resources into inefficient control and abatement activities devoted to one agent, asbestos. The cause of this situation has been multifactorial in nature. However, risk communication is considered a uniquely significant factor in the public and political response.

3. The current disparity between risk and reaction will not be easily resolved. The momentum of EPA regulation and guidance, attitudes of the financial community, the stance of involved politicians, and an organized removal industry will not be susceptible to rapid redirection. The liability associated with the presence of asbestos will be reduced only with the recognition of this current and competent scientific information by the courts, by the financial community, and the public.

4. Comprehensive material surveys and inventories based primarily upon architectural and engineering factors can enhance the objectivity of assessment in structures, the design of effective control action, and the appropriate scheduling of material removal. Unfortunately, current abatement by removal is frequently a reaction to unclear inventory information, confusion over risk factors, and marketplace pressures. Such actions are in all probability increasing risk, principally in personnel involved with the removal and disposal of asbestos materials.

5. Asbestos removal, the activity generating the greatest risk, should become the focus of governmental control and regulatory effort. The abatement industry deserves the greatest attention in operational studies, assembly of epidemiologic and exposure information, and control. If removals continue at the present rate, then the only course is to render them safe by all appropriate means.

6. The evolution of air sampling methodologies utilizing the transmission electron microscope has greatly enhanced the effective evaluation of airborne asbestos levels. However, fiber aerodynamic behavior, sampling and analytical variability, and cost will remain major considerations in air sampling utilization. The value and effectiveness of such measurements can vary significantly according to application.

   a. Initial assessment and initial steady state structural use evaluation: Air monitoring has only limited value as a general assessment tool. For nearly any individual site, an air study capable of producing competent data would of necessity be both complex and costly. Further, applicable generic data are becoming available. Using direct transmission electron microscopy (TEM), the building environment under conditions of steady state use appears to resemble background or community asbestos levels (Burdett 1988, 1989; EPA 1988). The foundation of assessment will remain the evaluation of basic physical structural parameters. Air monitoring results could augment, but should not supercede, this information. Air samples collected in conjunction with assessment can be beneficial. However, the use of air data should be limited to comparison to published building air studies, control program design, and risk communication.

   b. Monitoring of activities for control, maintenance, renovation, or removal: Air sampling data, both optical and TEM, will provide site data for monitoring control procedures, personnel protection selection, and regulatory compliance. Data of this type are limited but can be obtained by monitoring personnel during tasks representative of typical work procedures (Morse 1988, Sawyer 1979, Karaffa 1987). Such studies would provide data to increase perception and understanding of true risk to operating personnel.

7. Effective risk communication should assume a prominent position not only in governmental regulation and guidance, but also in any management and control effort. This would include current concepts of absolute risk, relative risk information, and comparison to other life risks. As example, smoking risks in relation to, and compared to, asbestos exposure are not adequately addressed in current regulation of asbestos in schools (EPA 1987a). If a major goal of EPA regulation is the reduction of asbestos related cancer then smoking avoidance, reduction, and cessation programs should be integrated with other control activities.

8. The thermal conversion of asbestos waste to glass should be adopted as the regulated mode of disposal. The true significance of this technology of asbestos conversion is the complete elimination of subsequent liabilities by the documented conversion of an asbestos waste to a nontoxic, nonregulated material. This is clearly the best available disposal technology. This conversion furnace process could be commercially available by 1990 (Roberts 1988).

REFERENCES

AHERA U.S. Congress. Public Law 99-519. Asbestos Hazard Emergency Response Act of 1986. Title II. October 22, 1986.

Anderson, H.A., Lillis, R., Daum, S. et al. Household contact neoplastic risk. Ann. N.Y. Acad. Sci. 271:311-323. 1976.

Bennett, M. Series: Asbestos Hysteria. Detroit News. Detroit, MI. 1985.

Berg, E.N. Asbestos Buildings Go Begging. Now harder to sell, lease or finance; State laws cited. The New York Times. April 28, 1988.

Bergman, Jules. A Dusty Road to Death. American Broadcasting Company. 1980.

Bomier, B. Institute for Environmental Assessment, Anoka, MN. Personal communication. 1987.

Borel v. Fibreboard Paper Products Corp. 493 Federal Reporter, 2nd, 1076-1109. (5th U.S. Circuit Court, 1973).

Brodeur, P. Annals of Industry: Casualties of the workplace. New Yorker Magazine. NY, NY. October 30 to November 23, 1973.

Burdett, G.J., Smith, J.J., Ainslie, V.H and Papanicolopoulos, C.D. (1988) Airborne Asbestos Fiber Levels in Buildings and Their Impact on Risk Management. Paper presented at the Symposium on Health Effects of Exposure to Asbestos in Buildings, Harvard University. December 14-16, 1988.

Burdett, G.J., Jaffery, S.A.M.T. and Rood, A.P. (1989) Airborne Asbestos Fiber Levels in Buildings. A Summary of UK Measurements. Non-occupational Exposure to Mineral Fibers, edited by J. Bignon, J. Peto and R. Saracci, International Agency for Research on Cancer Scientific Publication No. 90. 1989.

Cain, W.C., Powers, T.J. and Wilmoth, R.C. Results of Air Sampling from Selected Asbestos Abatement Projects. U.S. Environmental Protection Agency. Office of Research and Development, Water Engineering Research Laboratory. Presented at National Asbestos Council Third Annual Fall Conference. Oakland, CA. September 22, 1987.

California, State of. Safe Drinking Water and Toxic Enforcement Act of 1986. (Proposition 65). 1986.

Constant Jr., P.C., Bergman, F.J., Atkinson, G.R. Airborne Asbestos Levels in Schools. Final Report EPA 68-01-5915. Midwest Research Institute. 1983

Cooke, W.E. Pulmonary Asbestosis. Brit. Med. J. 2:1024-1025. 1927.

Copeland. U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. Research Triangle Park. Personal communication. 1987.

Corn, M. Asbestos and Disease: An Industrial Hygienist's Perspective. Am. Ind. Hyg. Assoc. J. 47:515-523. September, 1986.

Doll, R. Mortality from lung cancer in asbestos workers. Br. J. Ind. Med. 12:81-6. 1955.

EEPC Energy and Environmental Policy Center, Harvard University. Symposium on Health Aspects of Exposure to Asbestos in Buildings. Cambridge, MA. June 28, 1989.

EDF Environmental Defense Fund, Inc. Petition to the Environmental Protection Agency to Control Asbestos Emissions from Spray-on Materials Which Have Been Applied in Public School Buildings for Insulation, Fireproofing, Decorative or Other Purposes. December 21, 1978.

EPA (1973) U.S. Environmental Protection Agency. 40 CFR 61. National Emission Standards for Hazardous Air Pollutants: Asbestos. Federal Register, Vol 38, No. 66. 1973.

EPA (1979) U.S. Environmental Protection Agency. Asbestos Containing Materials in School Buildings: A Guidance Document. Parts 1 and 2. Office Toxic Substances C00090. March 1979.

EPA (1982) U.S. Environmental Protection Agency. Friable Asbestos Containing Materials in Schools: Identification and Notification. 40 CFR Part 763. Vol. 47, No 103, FR 23360. May 27, 1982.

EPA (1983) U.S. Environmental Protection Agency. Guidance for Controlling Friable Asbestos Containing Materials in Buildings. EPA Report 560/5-83-002. March, 1983.

EPA (1984) U.S. Environmental Protection Agency. 40 CFR Part 763 Asbestos; Response to Citizens' Petition. Environmental Protection Agency. Proposed Rules. Vol 49, No. 46. March 7, 1984.

EPA (1985) U.S. Environmental Protection Agency. Guidance for Controlling Asbestos-Containing Materials in Buildings: 1985 Edition. Office of Toxic Substances, Washington, D.C. EPA 560/5-85-024. June, 1985.

EPA (1987a) U.S. Environmental Protection Agency. 40 CFR 763, Part III. Asbestos-Containing Materials in Schools; Final Rule. Fed. Reg. 52:210. October 30, 1987.

EPA (1987b) U.S. Environmental Protection Agency. Office of Air Quality Planning and Standards. Research Triangle Park, N.C. Emission Standards and Engineering Division. Letter to Dr. David Roberts, dated October 19, 1987.

EPA (1988) U.S. Environmental Protection Agency. EPA Study of Asbestos Containing Materials in Public Buildings. A Report to Congress. EPA. Washington, D.C. February, 1988.

Fisher, A., McClelland, G.H. and Schulze, W.D. Communicating risk under Title III of SARA: Strategies for explaining very small risks in a community context. JAPCA. 39:3 271-276. March 1989.

GAO U.S. General Accounting Office. School District Officials Face Problems in Dealing With Asbestos in Their Schools. Report to the Chairman, Subcommittee on HUD-Independent Agencies, Committee on Appropriations House of Representatives. GAO/RCED-85-91. March 19, 1985.

Guenther, R. Asbestos fireproofing hinders sales of some office buildings. The Wall Street Journal. July 16, 1986.

HEI Health Effects Institute. Symposium on Asbestos Health Risks in Structures. October, 1988.

Hinds, M. Asbestos risks afflict office and housing markets. The New York Times. August 17, 1986.

Hughes, J.M., Hammad, Y.Y. and Weill, H. (1986) Mesothelioma risk in relation to duration and type of asbestos fiber exposure. Tulane University. Abstract presented at American Thoracic Society Meeting. Kansas City. May, 1986.

Hughes, J.M. and Weill, H. (1988) Pulmonary fibrosis as a determinant of asbestos-induced lung cancer in a population of asbestos cement workers. Proceedings of VIIth International Pneumoconioses Conference. Pittsburgh, PA. August, 1988.

Karaffa, M.A., Miller, C.E., Amick, R.A., Chesson, J. and Jackson, C. Preliminary Evaluation of Potential Asbestos Exposure During Carpet Vacuuming in Buildings Containing Friable Asbestos Material. U.S. Environmental Protection Agency. Unpublished Draft Report, Cincinnati, Ohio. November, 1987.

Langer, A.M. (1984) Testimony before the Environmental Protection Agency Hearings. Boston, MA. July 6, 1984.

Langer, A.M., Nolan, R.P., Constantopoulos, S.H. and Moutsopoulos, H.M. (1987) Association of Metsovo lung and pleural mesothelioma with exposure to tremolite-containing whitewash. The Lancet. 965967. April 25, 1987.

Layne, Geraldine v. GAF Corporation. Cleveland, Ohio. Cuyahoga County Common Pleas Court, Case 74194. Layne, Geraldine v. U.S. Minerals.

Lee, J. Bechtel Corporation, San Francisco, CA. Personal communication. January 1989.

Lumley, K.P.S., Harries, P.G. and O'Kelly, F.J. Buildings insulated with sprayed asbestos: A potential hazard. Occup. Hyg. Vol. 14:255-257. 1971.

Manville Corporation, et al. First Amended Disclosure Statement, Second Amended and Restated Plan of Reorganization and Related Documents. U.S. Bankruptcy Court Southern District of New York. August 22, 1986.

Mason, J.D., Ogden, H.G., Berreth, D.A. and Martin, L.Y. Interpreting risks to the public. Am. J. Prev. Med. Vol 2:3 133-139. 1986.

McDonald, J.C. Health implications of environmental exposure to asbestos. Environ. Health Perspectives. 62:319-328. 1985.

Merewether, E.R.A. The occurrence of pulmonary fibrosis and other pulmonary affections in asbestos workers. J. Indus. Hygiene. 12:198-22, 239-257. 1930.

Morse, R.G. Entek Environmental & Technical Services, Inc. Rensselaer Technology Park, Troy, NY. Personal communication. December, 1988.

Muir, D. Proceedings: American College of Chest Physicians meeting. Montreal P.Q. Canada. November, 1985.

New Jersey, State of. Asbestos Hazard Abatement Subcode. Subchapter 8. New Jersey Department of Community Affairs, Division of Housing & Development, Bureau of Construction Code Enforcement. CN 805. Amended May 5, 1986.

New York City Council. (1985) Local Law 76/85. New York City. 1985.

New York City DEP. (1988) Final Report on the Assessment of the Public Risk of Exposure to In-Place Asbestos. City of New York Department of Environmental Protection, NY. 1988.

New York City Council. (1989) Introduction Local Law 2611. New York City. 1989.

New York State Department of Environmental Conservation (DEC). Regulations regarding waste collector registration. Title 6, Part 364 of the New York State Official Compilation of Codes, Rules and Regulations -6NYCRR36.

New York State Department of Labor. Part 56 of Title 12 of the Official Compilation of Codes, Rules and Regulations of the State of New York. Industrial Code Rule No. 56, July 8, 1987.

New York State Department of Health. Asbestos Safety Program Requirements. Title 10, Chapter II: Administrative Rules and Regulations, Subchapter H, Environmental Health, Part 73. Proposed. March 18, 1987.

Newhouse, M. and Thompson, H. Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. Brit. J. Ind. Med. 22:261-269. 1965.

NIBS National Institute of Building Sciences. Model Guide Specifications for Asbestos Abatement in Buildings. Washington, D.C. October 15, 1985.

Nicholson, W.J., Rohl, A.N. and Weisman, I. (1976) Asbestos contamination of building air supply system. The Institute of Electrical and Electronic Engineers, Inc. Ann. No. 75CH1004-1-29-6. 1976.

Nicholson, W.J., Rohl, A.N., Sawyer, R.N., Swoszowski, E.J. and Todaro, J.D. (1978) Control of Sprayed Asbestos Surfaces in School Buildings: A Feasibility Study. Report to the National Institute of Environmental Health Sciences, June 15, 1978.

Ohlson, C.G. and Hogstedt, C. Lung cancer among asbestos cement workers. A Swedish cohort study and a review. Br. J. Ind. Med. 42:395-402. 1985.

Omenn, G.S., Merchant, J., Boatman, E., Dement, J.M., Kuschner, M., Nicholson, W., Peto, J. and Rosenstock, L. Contribution of Environmental Fibers to Respiratory Cancer. Environmental Health Perspective. 70:51-56. 1986.

Ontario Ministry of the Attorney General. Report of The Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario. J. Stefan Dupre, Chmn. Ontario Ministry of Government Services, Publication Services Branch, 880 Bay Street, Toronto, Ontario, Canada. 1984.

Reitze, W.B., Nicholson, W.J., Holaday, D.A. and Selikoff, I.J. Application of sprayed inorganic fiber containing asbestos. Occupational health hazards. Amer. Ind. Hyg. Assn. 178-191. 1972.

Richman, L.S. Why throw money at asbestos? Fortune. pp 155-170. June 6, 1988.

Roberts, David. Vitrifix of North America. 131 Duke St., Alexandria, VA 22314. Personal communication. May, 1988.

Ruckelshaus, W.D. Communications Strategy for Asbestos in Schools Program. Memorandum to Regional Administrators. September 28, 1983.

Sawyer, R. (1975) Yale Art and Architecture building contamination: Past, present and future. Proceed International Conference of Environmental Sensing and Assessment. September 1975.

Sawyer, R. (1977) Asbestos Exposure in a Yale Building: Analysis and Resolution. Envir. Res 13:1 146-168. 1977.

Sawyer R. (1979) Indoor asbestos pollution: Application of hazard criteria. Ann. N.Y. Acad. Sci. Vol. 330, 579:586, 1979.

Sawyer, R.N. (1984) Testimony before the Environmental Protection Agency SEIU Hearings. Boston, MA. July 6, 1984.

Sawyer, R.N. and Morse, R.G. (1986) An inventory process for determining asbestos control needs and costs. Architecture. December, 1986.

SEIU Service Employees International Union AFL-CIO, CLC. Petition for asbestos in the schools rulemaking. Petition to U.S. Environmental Protection Agency dated November 16, 1983.

Selikoff, I.J., Hammond, E.C. and Seidman, H. Latency of asbestos disease among insulation workers in the United States and Canada. Cancer. 46:2736-40. 1980.

Smith, A.R. Executive Director, Division of School Buildings, City of New York; Report to House Commerce, Transportation and Tourism Subcommittee on Asbestos and the Health Hazards Associated with Exposure, Newark, New Jersey, January 19, 1982.

Wagner, J.C., Sleggs, C.A., Marchand, P. (1960) Diffuse pleural mesothelioma and asbestos exposure in North Western Cape Province. Br. J. Ind. Med. 17:260-71. 1960.

Wagner, J.C, Newhouse, M.L., Corrin, B., Rossiter, C.E.R. and Griffiths, D.M. (1988) Correlation between fibre content of the lung and disease in east London asbestos factory workers. Br. J. Indus. Med. 48:305-308. 1988.

Walsh, J. U.S. Environmental Protection Agency and toxic substances law: dealing with uncertainty. Science 202:598-602. November, 1978.

Weill, H. and Hughes, J.M. Asbestos as a public health risk: disease and policy. Annual Review of Public Health. 7:171-192. 1986.

Whitaker, L. Monster in the Closet. Time. February 6, 1989. page 53.

Zurer, P.A. Asbestos: The fiber that's panicking America. Chemical & Engineering News. 63:9 28-41. March 4, 1985.

HOW DO CURRENT METHODS OF MEASURING ASBESTOS EXPOSURE ADDRESS RISK TO THE GENERAL POPULATION?

EDWARD T. PETERS

Arthur D. Little, Inc., Cambridge, MA 02140

The risk of exposure to asbestos for the general population manifests itself in public settings, such as schools, municipal buildings and commercial and private structures. An attempt to quantify the level of risk is impacted by the ability to sample airspaces in such buildings with appropriate precision, accuracy and sensitivity. Physical measurements of indoor airborne asbestos levels tend to be variable and perhaps inaccurate, resulting in a poor data base from which clinicians can assess risk.

Three key areas are addressed in this paper:

- Case histories of round robin sampling, and how improvement were implemented.
- Experience with indoor-outdoor sampling for asbestos, including methodology.
- Development of a physically and statistically suitable protocol for asbestos measurement at low concentrations.

The Reserve Mining Company case in 1973 included sample analysis by participating plaintiff and defense consultants for both water and air samples. The results indicated orders of magnitude differences by competent investigators, indicating a major shortcoming of state-of-the-art methodology. The systematic improvement to within a range of few times by means of an ASTM working group will be presented.

Concerns about asbestos emissions from home appliances (i.e., hairdryers, washing machines, stoves, etc.,) lead to an evaluation of residential air quality, both in homes known to have asbestos-containing appliances and otherwise. The unpublished results of this work will be given.

Numerous methods for sample presentation and analysis are being utilized. Comments will be made on the importance of establishing an analysis protocol that satisfies scientific scrutiny and peer review. Concluding remarks will address the current methods for establishing laboratory certification for asbestos analysis and views on the effectiveness of the programs.

INTRODUCTION

The link between occupational exposure to asbestos disease, including asbestosis and various forms of cancer, has been well established. The degree of risk for a given occupational setting is generally based upon elevated mortality as compared to a matched but non-exposed cohort. These types of data bases have been used to develop exposure response models for particular diseases that relate fiber exposure times duration (fiber-years) to relative risk (the ratio of occupational mortality to expected mortality). These models for asbestos disease are complicated by several factors:

- Latency - asbestos diseases are usually not observed for 20-30 years after initial exposure. The cumulative fiber-year dosage is very difficult to determine accurately.

- Asbestos concentration - up until the mid-to-late-1960's, asbestos concentration was measured by a particle count procedure. Subsequently, measurements have been made by a fiber number method. The two measurement methods do not correlate, causing a further uncertainty in historic occupational exposure levels.

- Asbestos type - the models do not reflect any difference in asbestos type as is recognized by all other asbestos-regulating countries.

- Occupation - an order of magnitude (or greater) difference can be shown for the potential for asbestos-related disease among various occupations. In one study, for example, the risk of mesothelioma was 46 times greater for insulation workers than for garage workers or building maintenance men (McDonald and McDonald, 1980).

As a result of these complicating factors, estimates of risk can vary several orders of magnitude for an apparent equivalence of asbestos exposure.

The problem is further complicated when attempts are made to estimate the risk to health of the general population by environmental exposure to asbestos as may occur

in buildings. In this case, it is first necessary to evaluate the very low levels of exposure and to then infer the health response, either by significant extrapolation of the already uncertain occupational data or by an epidemiological study of "exposed" and "non-exposed" populations. The difficulties encountered by either approach are vast.

The U.S. Environmental Protection Agency, Office of Health and Environmental Assessment commissioned a study in the early 1980's entitled Asbestos Health Assessment Update (Nicholson, 1986). The abstract from that document is (in part) as follows:

> "Recent data from population studies strengthened the association of asbestos with disease .... calculations of asbestos risk values are uncertain and based on subjective estimates because of the following limitations in data:
>
> - Extrapolation from high occupational levels to much lower ambient levels
> - Uncertainty from mass to fiber conversion
> - Statistical uncertainties
> - Various biases and confounding aspects of medical data
> - Non-representative exposure estimates".

The Symposium from which these Proceedings was prepared attempted to address these issues with respect to asbestos in buildings. In addition to health effects, various institutional issues (moral, legal, financial and legislative) were discussed. The following paper addresses the reliability in estimating asbestos concentrations, especially at low levels. Case histories are given to illustrate the lack of agreement between laboratories and approaches toward improvement. In addition, examples are given of indoor-outdoor studies of asbestos at very low concentrations which illustrate the difficulty in distinguishing low level sources (brake repair stations, sprayed on asbestos insulation in buildings) from the general ambient background to which everyone has exposure. Finally, some comments are provided with respect to preferred sampling and analysis protocols.

INTERLABORATORY ANALYSIS: CASE HISTORIES

Case 1 - Reserve Mining Company - Water

In 1973, as part of Federal proceedings against Reserve Mining Co., a taconite ore processing plant disposing of tailing wastes in Lake Superior, a series of 20 water samples were collected by a Friend of the Court and distributed to all analysis groups to measure asbestos concentrations[1] "by their usual methodology". Subsequently, after results were presented, it was revealed that the samples were taken at five locations with four replicates (Figure 1). The experimental results are summarized in Table 1 and Figure 2. The difference in concentration for high and low estimates is a factor of 20 to 50 times, indicating the wide difference of result by laboratories presumably practiced in the art.

Case 2 - Reserve Mining Company - Air

At a later time in the court proceedings, a Friend of the Court collected a total of twelve air filtration samples from the roof tops of Silver Bay Schools. Pie-shaped pieces of these filters were distributed to the principal expert witnesses for analysis "in accordance with their standard method". The results are presented in Figure 3 and Table 2. The first three laboratories tabulated utilized the indirect method of transmission electron microscopy (TEM), in which the filter is ashed at low temperature to remove organic matter, resuspended in a liquid and agitated, then refiltered before preparing the TEM sample. The fourth and fifth laboratories utilized the direct TEM method and the final laboratory utilized a hybrid method in which the sample was carbon coated, floated on a solvent to dissolve the filter material and then picked up on a microscope grid.

The results provided in Table 2 indicate a difference in estimated fiber concentration in relation to sample preparation as follows:

Direct	Hybrid	Indirect
1-3	40	80-100

Users of the indirect method claim the direct sample preparation method is subject to fiber loss during TEM sample preparation and fiber obscuration by other particulate matter. Those preferring the direct method are concerned about sample alteration during the sample ashing, resuspension and agitation steps of the indirect method.

As a result of these differences, an experimental test program was developed to compare the direct and indirect (reconstitution) methods, including method of ashing,

---

[1] These water samples contained enlongated cleavage fragments which, at the time of analysis, were likened to amosite asbestos.

agitation and filter type for the latter. The experimental plan is given in Figure 4. All of the starting samples were taken from MDH sample No. 9063 (see Table 2). The results are shown in Figure 5, where the internal represents the 95% confidence level. All of the replicates show close agreement. However, the apparent amphibole fiber concentrations by the indirect method are 15-20 times higher than by the direct method when using an ultrasonic bath for agitation and 30-40 times higher for agitation with an ultrasonic horn.

To determine the discrepancy in fiber concentration, median fiber dimensions for each sample were determined from which an average fiber mass was calculated (Table 3). The fiber dimensions and mass observed by the direct method are consistently larger than those observed by any form of the indirect method. The conclusion is that the agitation step utilized for the indirect preparation results in the fracturing of large fibers into many smaller fibers, thereby significantly increasing the apparent fiber concentration.

Case 3 - ASTM Task Group on TEM Sample Preparation for Asbestos

An ASTM Task Group (Chopra, 1978) was formed in 1975 to develop a consensus for TEM sample preparation methodology promising the best opportunity for inter-laboratory agreement. A series of samples were provided to the participants who periodically met and discussed results. Several sets of experiments are summarized in Figure 6.

Notable improvement is observed for replicate analysis of the same starting sample which is attributed to more common sample preparation and analysis practice for the second set in each group on the basis of discussion after the first set. In particular, Set 2 shows a much narrower distribution than Set 1, although both exhibit similar median fiber concentrations. Set 6 shows a similar improvement in distribution compared to Set 5. Sets 3 and 4 are comparable except that Set 3 represents a water sample and Set 4, a prepared filter sample. The median Jaffe wick concentration is only slightly (but not significantly) higher for Set 4. A much larger scatter is found for preparing samples with a condensation washer, and the Task Group recommended that this procedure not be used. The conclusion of this study is that laboratories that practice a common sample preparation procedure will exhibit a maximum difference in result of 4-5 times compared to 40-50 times difference when using their own individual procedures.

MEASUREMENT OF ASBESTOS CONCENTRATIONS AT LOW LEVELS

Case 4 - Indoor-Outdoor Study

In the late 1970's, it became evident that many retail hair dryers utilized asbestos paper liners for electrical insulation, posing a question of asbestos fiber release into the home during use. Some Consumers Product Safety Commission staff suggested that use of an asbestos insulator-containing hair dryer would release a small number of asbestos fibers during each use which would build up to dangerous (even up to occupational) levels after a period of time.

To gain insight on this potential problem, Arthur D. Little (ADL) conducted a comprehensive indoor-outdoor air sampling program designed to measure asbestos fibers at very low concentrations. For this purpose, five homes of ADL staff in a Boston suburban setting were selected for testing. An initial walk-through survey identified possible sources of asbestos in some home including furnace insulation, an asbestos insulation-containing hair dryer and a recent asbestostile removal project. Other homes appeared to be asbestos free. The sampling protocol, given in Table 4, required long collection times to provide a large enough air sample to reliably measure very low concentrations of asbestos fibers. Because of substantial collection of organic matter, primarily soot, the samples had to be subjected to low temperature ashing and then reconstituted for analysis by the indirect method. Given the concerns described in Case 2 above, attempts were made to minimize fiber fracture. However, the results were intended to be comparative of indoor vs outdoor levels, and all samples were treated equally.

The sample results are shown in Figure 7. The results indicate a total asbestos fiber burden about an order of magnitude higher indoors (kitchen and bedroom areas gave similar concentrations regardless of apparent sources of asbestos) than the outdoor samples. However, the mass burden was about the same due to the presence of larger fibers outdoors. All samples had fiber contents of one to two orders of magnitude greater than the blanks, which were taken as the minimum detectible limit.

It is concluded that asbestos is present everywhere at very low concentrations. Furthermore, there was no correlation between homes known to have asbestos containing materials and measured fiber concentrations.

Case 5 - Potential for Inhaled Asbestos:
Garage Mechanics Compared to the General Population

In 1984, ADL was asked to evaluate the risk to garage mechanics from the inhalation of asbestos fibers in brake dust during brake servicing. An evaluation of existing data for prudent brake repair methods (i.e., vacuuming, wet rage, etc) indicated an estimated regulatory asbestos fiber concentration of 0.03 fibers/cm^3. Case 4 above indicates indoor concentrations may be higher. On the basis of a typical garage worker spending 25 percent of his time repairing brakes, this represents 10 hours per week at 0.03 f/cm3 and the remaining 158 hours at 0.004 f/cm^3, equivalent to a total average exposure of 0.0053 f/cm^3. On a statistical basis, asbestos fiber exposure to the garage mechanic and to the general population are the same within a very narrow confidence interval, i.e. there is no increase in risk for the garage mechanic compared to the general population.[2]

SAMPLING AND ANALYSIS PROTOCOLS

Sample collection procedures are quite standard; they involve the collection of a known volume of air (or water) through an efficiently collecting filter of known area. Analysis involves the observation and count of fibers on a portion of the filter from which number concentration (fibers/cm^3) can be computed. For cases where a mass concentration is required, i.e., to evaluate the level of asbestos contamination in a mineral product, it is necessary to measure fiber dimensions (length and width and an assumed thickness) and apply a density factor. The objective of all analyses is to obtain a valid result: one that is accurate (reflects the true value), precise (is reproducible) and sensitive (within detectible limits).

Several reference methods are provisional or promulgated, based upon optical and transmission electron microscopy (TEM); other microscopy methods are useful for special purposes. These are summarized in Table 5. Although the TEM methods have greater validity, they are about ten times more expensive than the optical method.

Some additional issues that affect analysis validity are not addressed by the regulatory methods. They are described below.

Sample Uniformity

All areas of examination should provide equivalent or similar fiber counts. For example, if 100 fields are examined optically and counts are all in the range of 5 to 15, the sample is apparently uniform. If, however, 98 of the pores have counts in the range

---

[2] In a related experiment, equivalent amounts of urban particulate matter showed greater cytotoxicity by in vitro tests than did brake lining dust, using UICC chrysotile as a positive control.

5 to 15, but one is 60 and the last is 150, a non-uniformity exists which braces the calculated average fiber count. Various statistical methods can be invoked to test for acceptable uniformity. One most useful for fiber counts is the Poisson statistic. If not satisfied, the sample should be reprepared.

Confidence Interval

Fiber concentrations represent the average of all observations. Statistical methods are available to indicate the range of values that could be expected by replicate analyses. Again, the Poisson statistic is most useful for describing the range (i.e., confidence interval) around the average of observations. As may be expected, a large number of observations result in a narrower range of confidence interval or uncertainty. This is illustrated in Figure 8, which shows the 95% confidence interval range (i.e., 95 of 100 tests fall in range) as a function of the number of independent observations. Note the diminishing returns as number of observations increases.

Data Display

For estimating effects on health, it is necessary to know fiber size ranges. This is conveniently performed graphically by a length-width-frequency distribution in accordance with Figure 9. In this way, fiber amounts in terms of length, width or aspect ratio can readily be assessed. An example of fiber sizes in a test cloud of 7R chrysotile used to valuate penetration through protective clothing is shown in Figure 10.

CONCLUSIONS

Sampling and analysis of asbestos fibers for estimating concentration is imprecise.

- Wide differences are obtained for the same sample by qualified laboratories.
- Inappropriate methods of sample preparation can fracture fibers into smaller ones increasing the apparent concentration by more than an order of magnitude.
- However, interlaboratory tests indicate that agreement within a factor of 4 to 5 times can be achieved when using the same procedure. Sampling and analysis of asbestos fibers at low, environmental concentrations is difficult.
- Asbestos fibers are ubiquitous

- The uncertainty in sampling and analysis makes measurement of low concentrations of asbestos fibers, as may be present in asbestos insulated buildings, indistinguishable from the general background concentrations.

Regulatory methods are based upon measuring average asbestos fiber concentrations. In addition, these methods should include:

- A measurement of sample uniformity

- The specification of confidence interval associated with measured concentrations.

- A standard method of presenting fiber dimensions and frequency to assist in evaluating the influence of asbestos fiber concentration on health.

Although progress in analytical consistency can be demonstrated for laboratories with experience, as shown by the ASTM Task Group, the proliferating of testing requirements provides an opportunity for new analysts and businesses that can't help but contribute to measurement imprecision until they too are experienced.

REFERENCES

Chopra, K.S. (1978). Interlaboratory Measurements of Amphibole and Chrysotile Fiber Concentration in Water Jnl of Testing and Measurement 6 241-247.

Cook, P.M. and Marklund, D.R. (1982). Sample Preparation for Quantitative Electron Microscope Electron Microscope Analysis of Asbestos Fiber Concentrations in Air. In Asbestos Standards: Materials and Analytical Methods (J. Small and E. Steel, Eds), 53-67, NBS Special Publication 619, National Bureau of Standards, Washington, D.C.

McDonald, A.D. and McDonald, J.C. (1980). Malignant Mesothelioma in North America Cancer 46, 1650-1656.

Peters, E.T. and Doerfler, T.E. (1978). Amphibole Mineral Fiber Analysis by Electron Microscopy: Comparison of Sample Preparation Procedure. In Electron Microscopy and X-ray Applications to Environmental and Occupational Health Analysis (P.A. Russell and A.E. Hutchings, Eds.) pp. 189-1203, Ann Arbor Science Publishers, Inc., Ann Arbor.

LIST OF FIGURES

Figure No.	Title
1	Reserve Mining Co. vs U.S.: Water Sampling Sites
2	Reserve Mining Co. vs U.S.: Water Sample Results
3	Reserve Mining Co. vs U.S.: Air Sample Results
4	Experimental Plan
5	Concentration Estimates from Replicate Samples
6	Summary of Results for Interlaboratory Test Results: ASTM Task Group
7	Results of Indoor-Outdoor Asbestos Fiber Concentration Study
8	Range of 95% Confidence Interval as Function of Observations
9	Fiber Characteristics by Length-Width-Frequency Histogram
10	Example of Fiber Dimension-Frequency Histogram for 7R Chrysotile Dust Cloud

LIST OF TABLES

Table No.	Title
1	Reserve Mining Co. vs U.S.: Water Sample Summary Results
2	Reserve Mining Co. vs U.S.: Minnesota Department of Health Air Sample Results
3	Fiber Median Values
4	Protocol for Indoor-Outdoor Air Sampling
5	Measurement of Number Concentration

Table 1
Reserve Mining Co. vs US.: Water Sample Summary Results

Site	Fiber Concentration (Million Fibers/Liter)			
	Low	High	Median	Mean
1	1.3	22.8	5.9	9.0
2	1.0	46.5	11.4	15.4
3	0	69.8	9.0	18.4
4	5.0	252.5	17.4	63.3
5	0	1.7	0.1	0.5

Table 2
Reserve Mining Co. vs. U.S.:
Minnesota Department of Health Air Sample Results
(Peters and Doerfler, 1978; Cook and Marklunc, 1982)

Site	Sample No.	Ambphibole Fiber Concentration (MFL)					
		A	B	C	D	E	F
1	7144A	335	262	390	5.9	5.5	99
1	7144B	164	235	177	2.7	5.4	110
1	7144C	323	178	174	3.0	6.6	91
2	9040	384	513	450	3.9	12.8	100
2	9041	502	448	351	2.5	6.1	160
2	9042	583	516	569	0.8	6.2	291
3	9061	53	33	67	1.0	1.6	74
3	9062	358	71	112	5.8	12.4	215
3	9063	240	76	120	0.6	3.8	20
4	4221	252	158	138	4.4	10.4	50
4	4222	100	99	96	1.4	8.0	70
4	4223	394	230	221	3.2	20.6	84

A Nicholson, Mt. Sinai; Indirect (LTA/Rubout)

B Cook, EPA/DWQL; Indirect (LTA/C-Coat/Nuc/Jaffe)

C Bishop, MDH; Indirect (LTA/Drop)

D Harwood, ITTRI; Direct (Condensation Washer)

E Peters, ADL; Direct (Jaffe)

F Champness, University of Manchester; Hybred (C-Coat, Dissolve)

Table 3
Fiber Median Values

Method*	Length (μm)	Width (μm)	Mass (pg)
		Amphibole	
A	2.16	0.36	0.64
B	1.80	0.18	0.16
C	1.33	0.22	0.20
D	1.44	0.22	0.17
E	0.90	0.11	0.03
F	1.44	0.18	0.23
		Total Fibers	
A	1.44	0.20	0.18
B	0.72	0.07	0.01
C	0.63	0.06	0.01
D	0.86	0.11	0.03
E	0.72	0.07	0.01
F	0.94	0.10	0.04

* A description of sample preparation procedures for Methods A-F are given in Figure 4.

Table 4
Protocol for Indoor-Outdoor Air Sampling for Low Concentration of Asbestos

- 5 hours in suburban community (away from major highway)
- 2 indoor, 1 outdoor, 1 blank filter samples
- 96 hour samples at 40 l/min
- 230 M^3 volume samples on 4" diameter Millipore filters
- Low temperature ash plus reconstitution, i.e. indirect method
- Analysis by TEM protocol
  - 20 pore openings examined
  - Image (straight, parallel, $R \geq 3.0$) for fiber identification
  - Selected area electron diffraction to confirm as asbestos

Table 5
Measurement of Number Concentration:

$$\text{Concentration} = \frac{\text{number of fibers x total area analyzed}}{\text{area of sample}}$$

- Optical (400X)
  - Phase contrast (NIOSH 7400)
  - Up to 100 fields x 0.00785 = 0.785 mm^2
  - All fibers are assumed to be asbestos
- TEM (10,000X)
  - 47 mm dia. filter (960 mm^2 area)
  - Pore scan for fiber image (3 x 1, 5 x 1 for AHERA, etc.)
  - EM pore size = 90 μm square (A_p = 0.0081 mm^2)
  - 20 pores (0.162 mm^2)
- Other
  - Polarizing light microscopy
    - Useful for asbestos identification in bulk samples by dispersion staining
  - Scanning Electron Microscopy (SEM)
    - Useful for screening samples
    - Wide range of magnification (i.e., 20-20,000X)

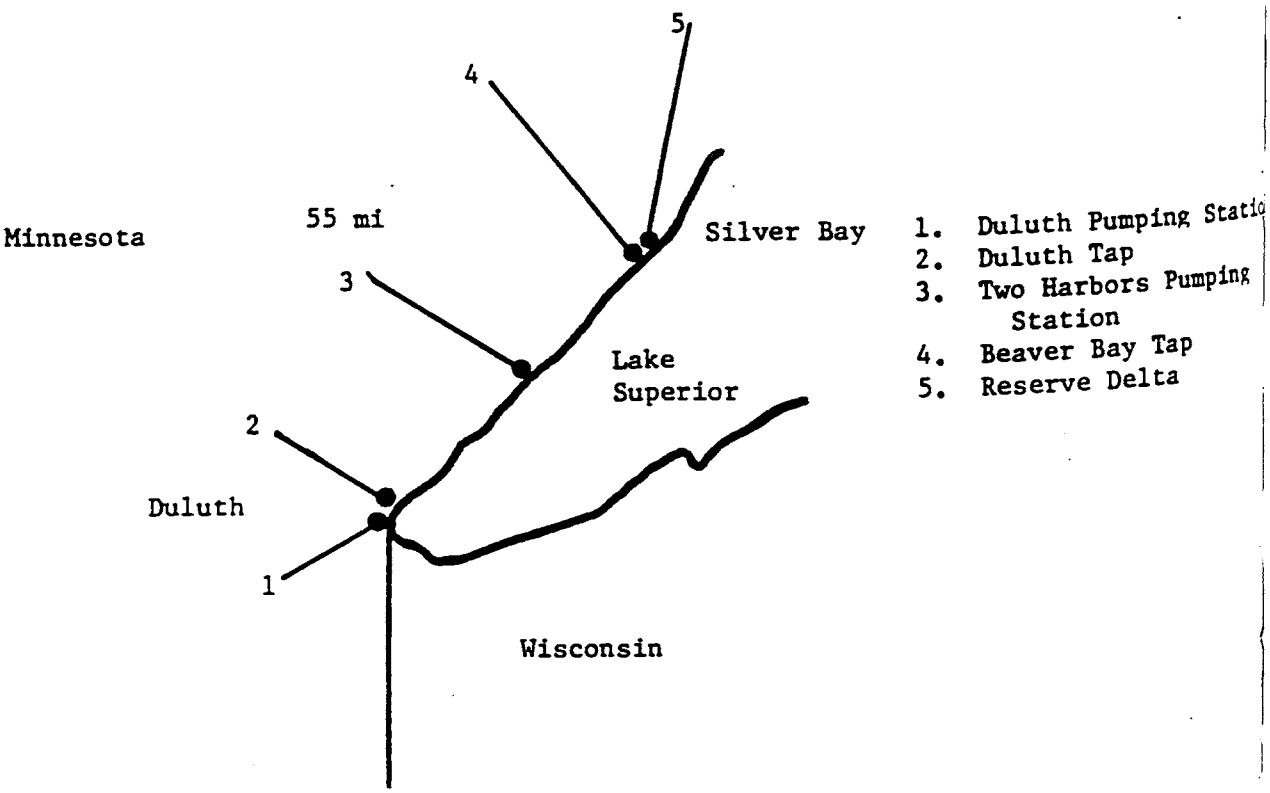


Figure 1. Reserve Mining Co. vs U.S.: Water Sampling Sites

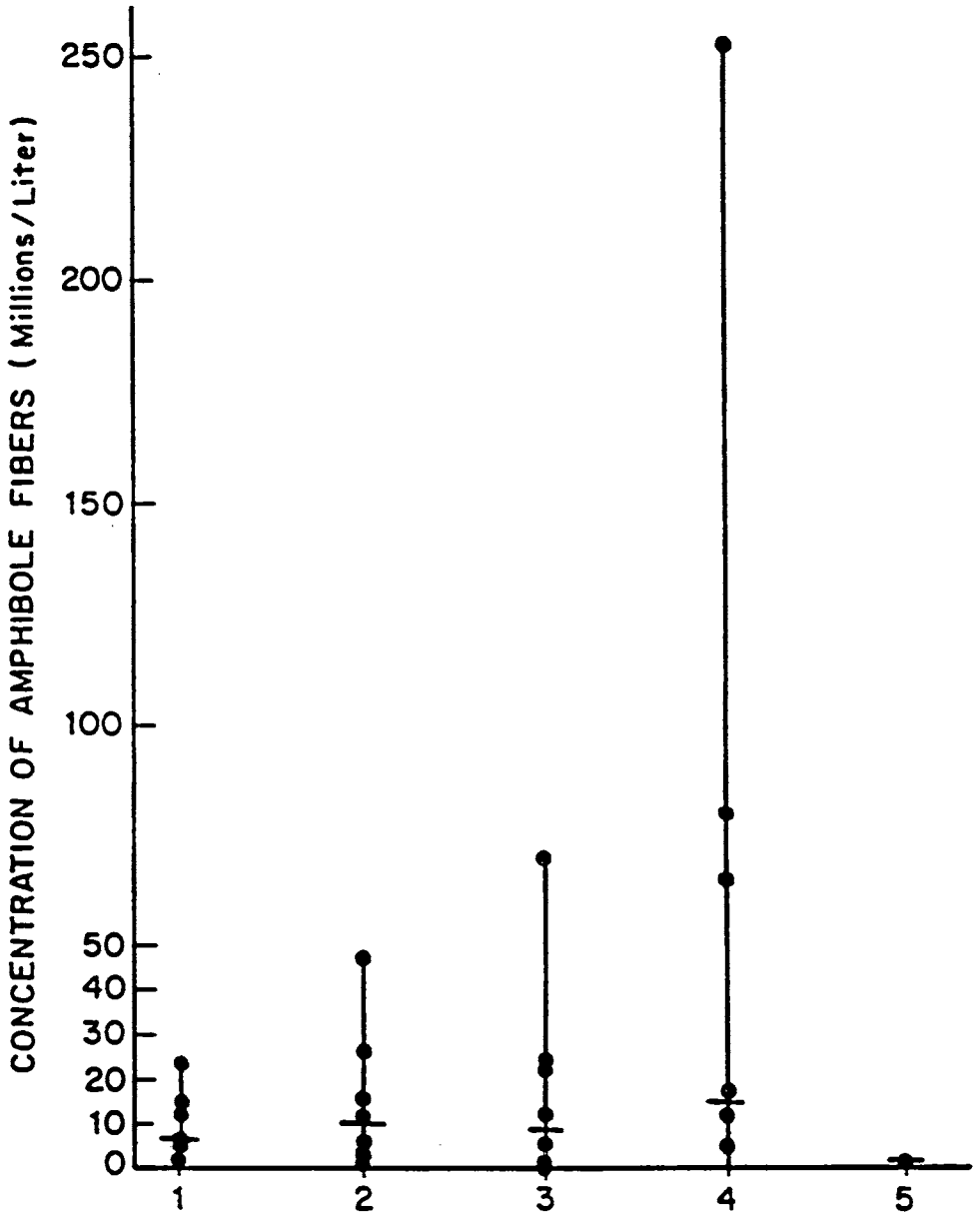


Figure 2. Reserve Mining Co. vs U.S.:
Water Sample Results at Five Sites
(Bar indicates median value)

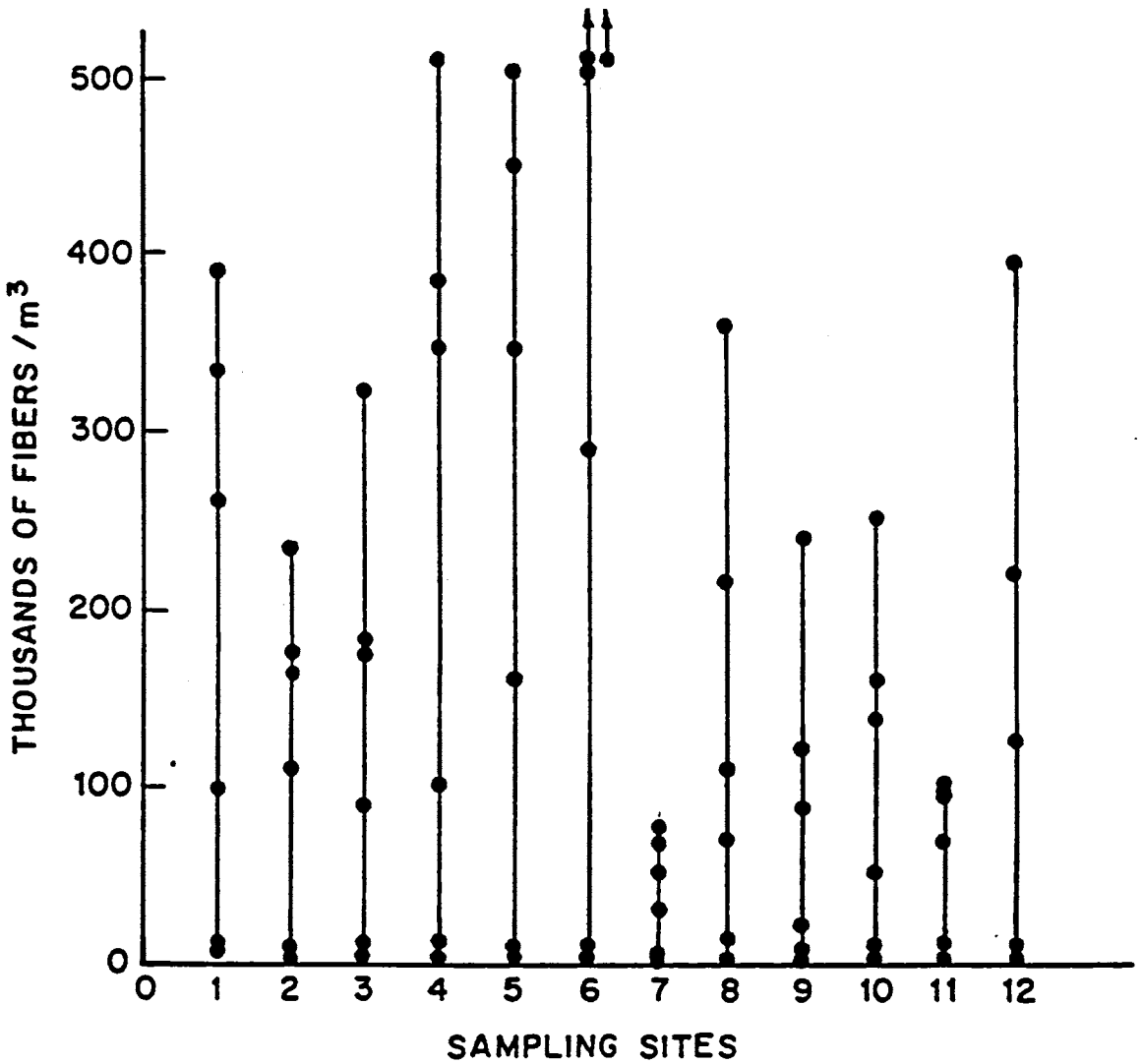


Figure 3. Reserve Mining Co. vs U.S.:
Air Sample Results

- Reproducibility: Direct - 4 replicates (Samples 1 - 4); all other conditions - 2 replicates
- Direct vs Reconstitution: Samples 1 - 4 (Direct) vs 5-14 (Reconstitution)
- Ashing Procedure: Samples 5-6 (Acid Digestion) vs 7-8 (Plasma Ash)
- Ultrasonic Agitation: Samples 7-8 (Ultrasonic Bath) vs Samples 9-10 (Ultrasonic Horn–low setting) vs Samples 11-12 (Ultrasonic Horn–high setting)
- Filter: Samples 11-12 (Nuclepore) vs Samples 13-14 (Millipore)

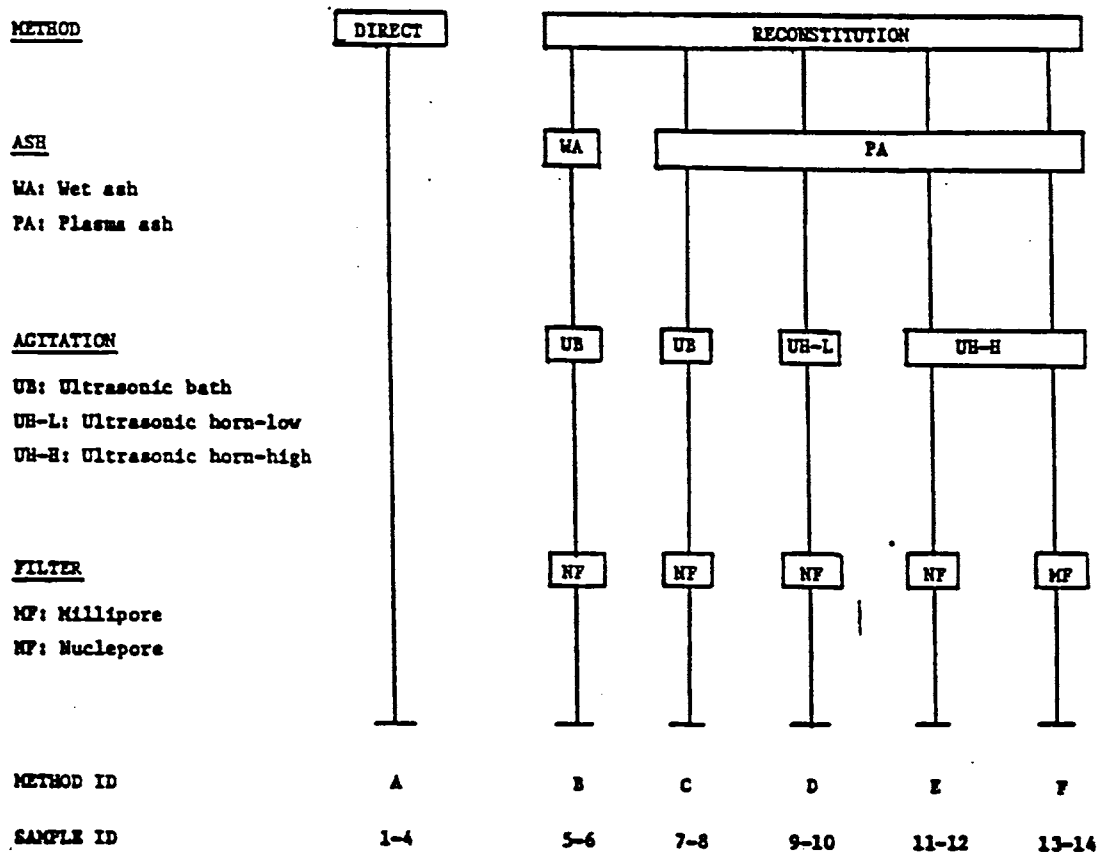


Figure 4. Experimental plan

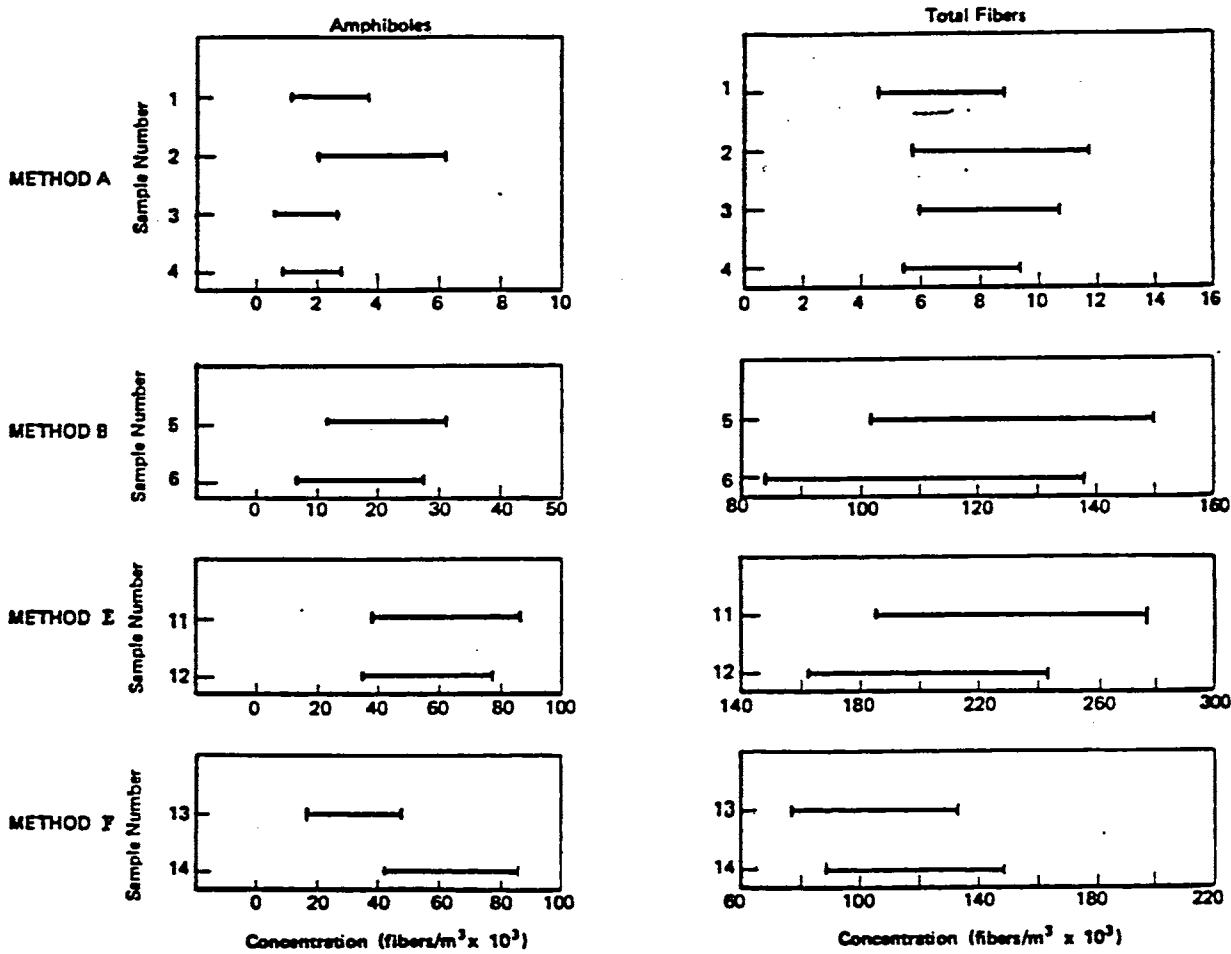
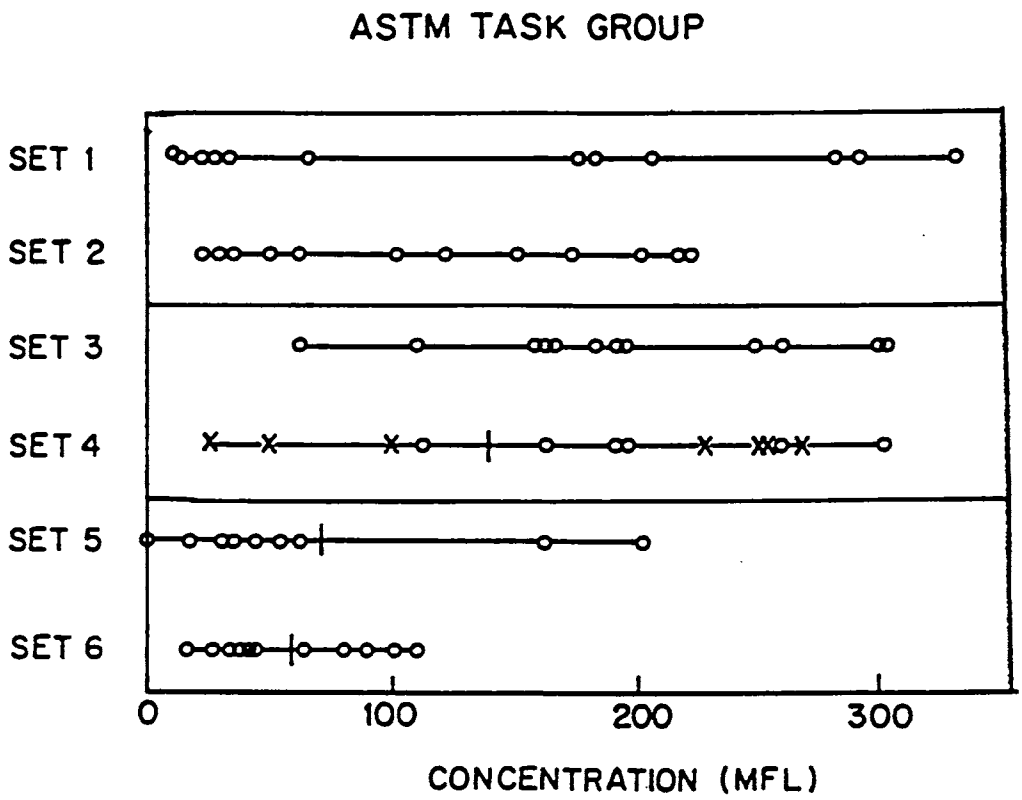
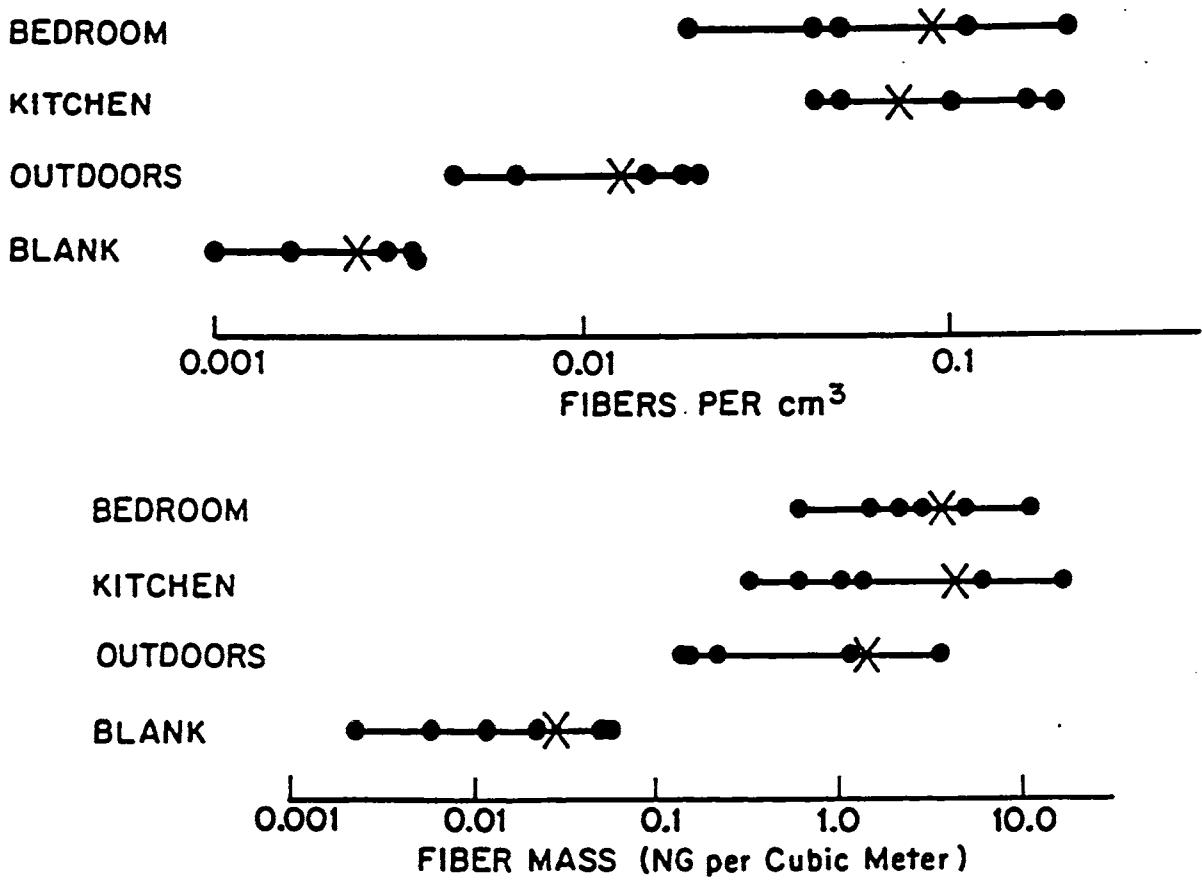


Figure 5. Concentration estimates from replicate samples



Set 1 - Best method of lab (Duluth tap water)
Set 2 - Best method of lab (Duluth tap water)
Set 3 - Jaffe wick of C-coated Nuclepore (DTW)
Set 4 - Duluth Tap Water
o - Jaffe wick (Nuclepore)
x - condensation washer (Millipore)
Set 5 - Jaffe wick of C-coated Nuclepore (DTW)
- spiked chrysotile content
Set 6 - Jaffe wick of C-coated Nuclepore (DTW)
- spiked chrysotile content

Figure 6. Summary of Results for Interlaboratory Test Results: ASTM Task Group



Note: There were too few regulation fibers to develop reliable concentration data. Values were approximately two orders of magnitude lower (0.0001 - 0.001 f/cm^3)

Figure 7. Results of Indoor-Outdoor Asbestos Fiber Concentration Study

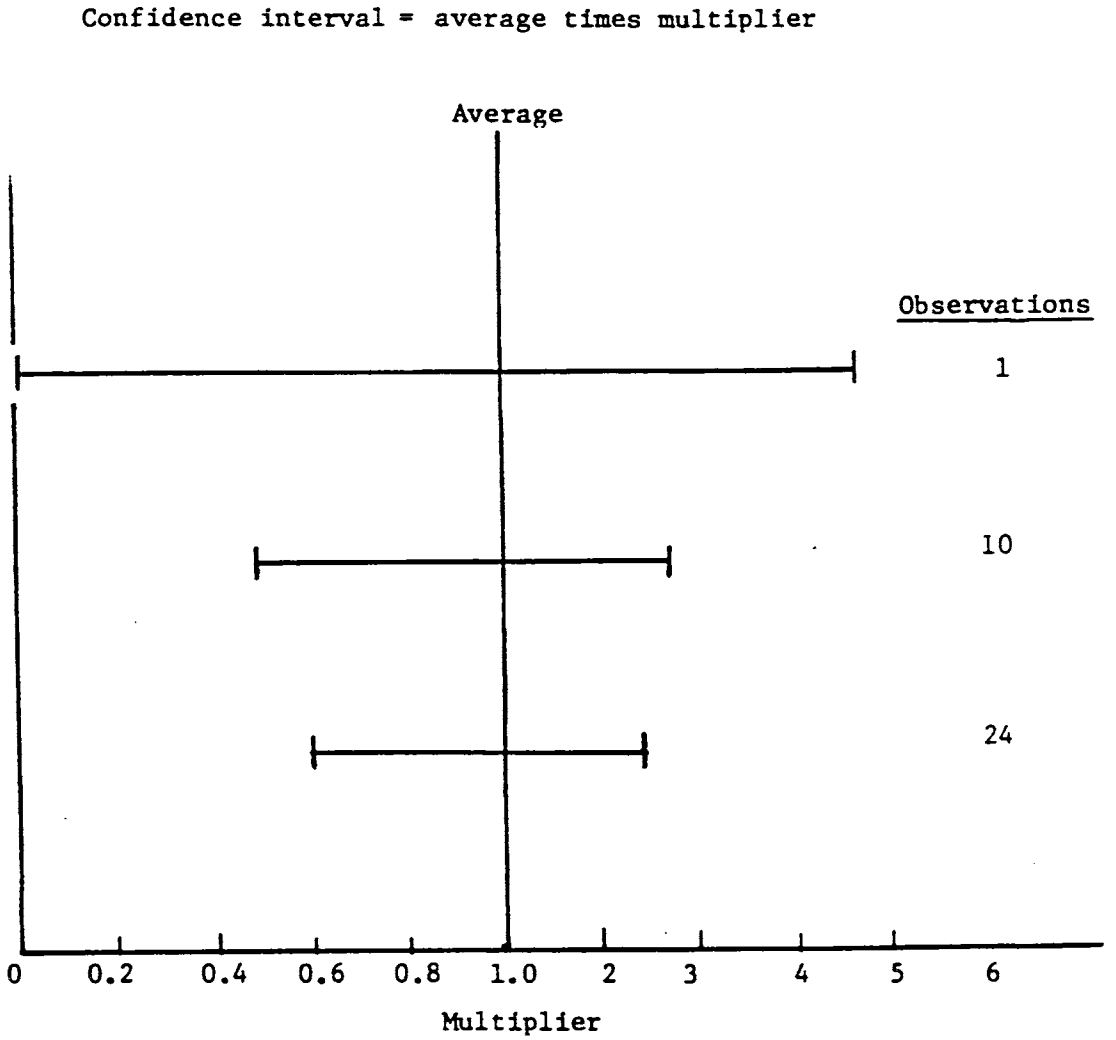


Figure 8. Range of 95% Confidence Interval as Function of Observations

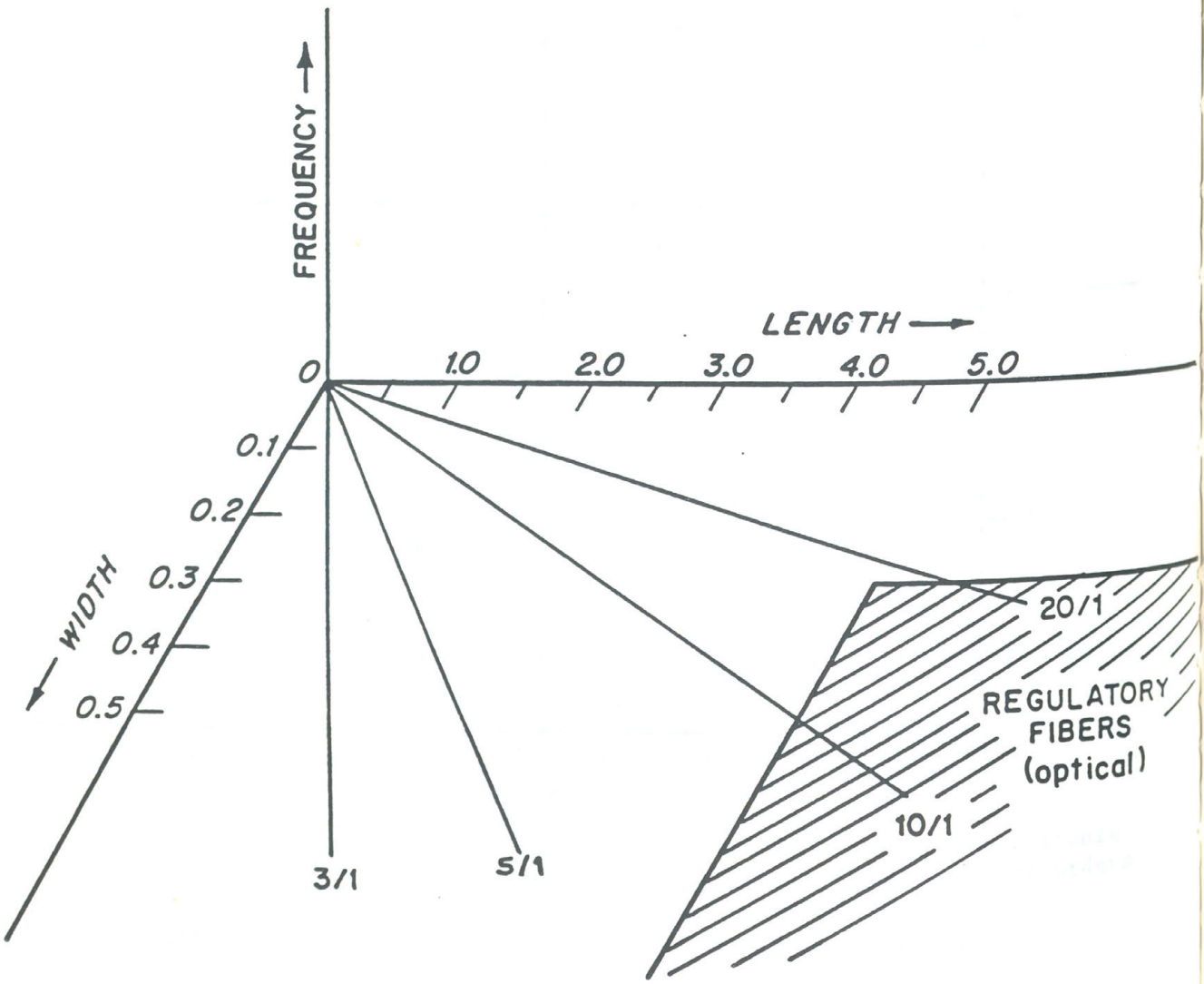


Figure 9. Fiber Characteristics by Length-Width-Frequency Histogram

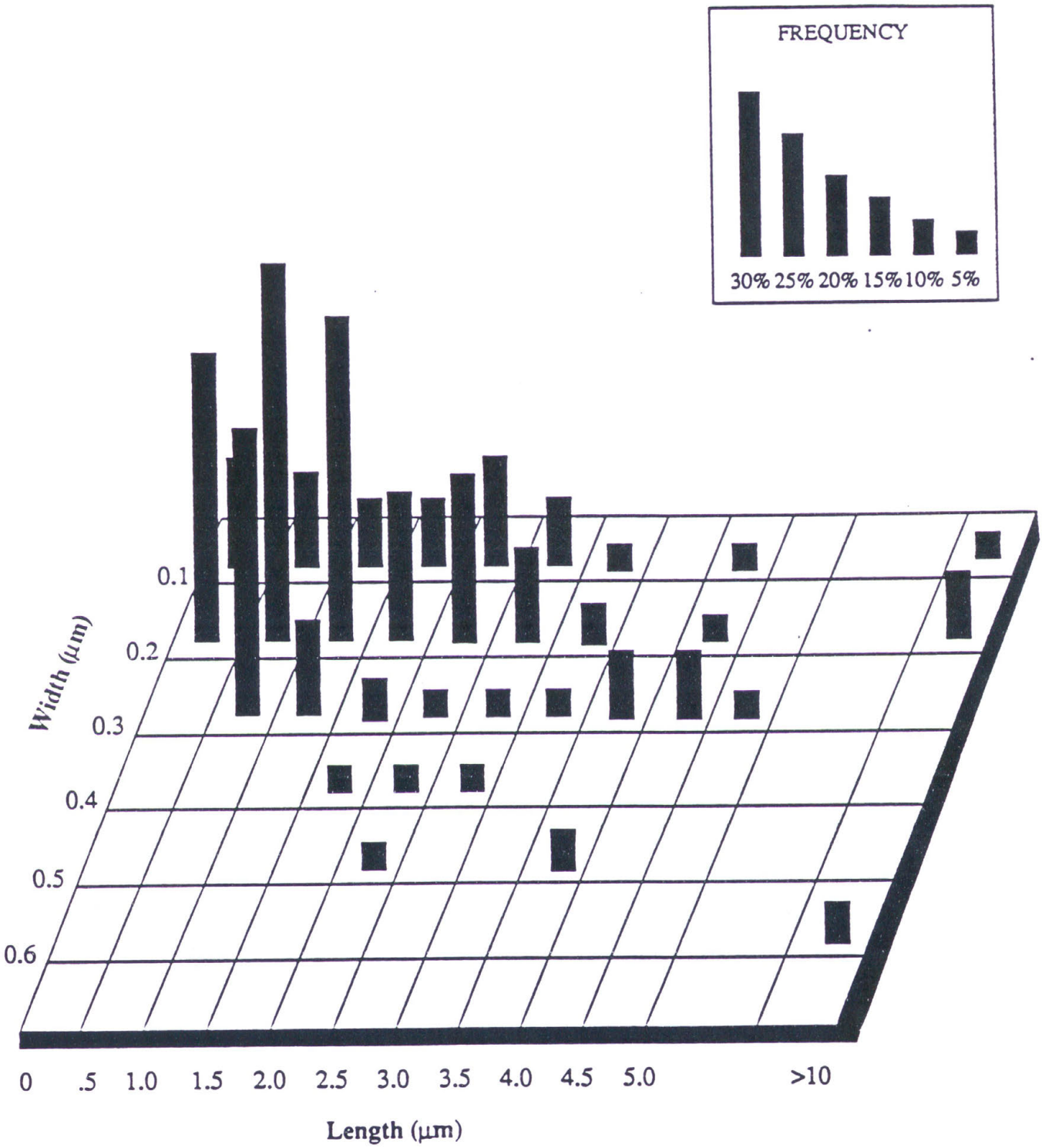


Figure 10
Example of Fiber Dimension-Frequency Histogram for 7R Chrysotile Dust Cloud

ASSESSING ASBESTOS EXPOSURE POTENTIAL IN BUILDINGS

BERTRAM PRICE

Price Associates, Inc., 1825 K Street, NW, Suite 501, Washington, DC 20006

Asbestos-containing building material (ACBM) in schools and other public and commercial buildings has been identified as a source of exposure to airborne asbestos fibers for occupants of these buildings. The presence alone of ACBM does not mean that the health of building occupants is endangered. Exposure and the associated risk to human health depend on the likelihood that asbestos fibers are released from ACBM, become airborne, and are inhaled. Two general approaches for assessing exposure potential are currently used: (i) visual inspection; and (ii) air monitoring. Applications of both approaches have been controversial, however each is useful in the proper setting. Each approach has been used in recent studies concerned with exposure potential. The visual inspection approach was used by the U.S. Environmental Protection Agency (EPA) to develop information for the February, 1988 report to Congress on ACBM in public buildings and by the City of New York in a study of "in-place" asbestos. Air monitoring also was used by EPA for their report to Congress and jointly by three building products manufacturers to estimate airborne asbestos levels in schools where ACBM was scheduled for removal. Study objectives and research methods associated with these applications are presented. Results of the studies are discussed and implications for regulatory policy are noted.

INTRODUCTION

Two methods are used to assess asbestos exposure potential in buildings: (i) visual inspection to evaluate the condition of asbestos containing building material (ACBM) and to assess the potential for disturbance of ACBM that may cause asbestos fibers to be released; and (ii) air monitoring (i.e., air sampling followed by laboratory analysis). Visual inspection means evaluating building materials suspected of containing asbestos by noting their location, amount (e.g., square or lineal feet), function, condition, accessibility, and

other characteristics that affect the likelihood of fiber release. Bulk samples of the material are extracted for laboratory analysis to determine which materials, in fact, contain asbestos. The data obtained through a visual inspection are summarized to produce one or more indices which are used as indicators of exposure potential. Air monitoring measures airborne asbestos fiber concentrations in building air directly. These concentrations are interpreted as exposure levels for individuals occupying the building.

Federal, state, and local governments collect data on exposure potential to aid in the development and analysis of alternative regulatory proposals. Building owners also collect exposure potential data in individual buildings for use in selecting among asbestos management options. Both visual inspection and air monitoring may have roles in each of these situations. The focus of this paper, however, is on regulatory applications.

If exposure potential is assessed for regulatory purposes, a sample of buildings that represents a larger target population of buildings is studied. The visual inspection approach provides an estimated inventory for the target population of the number of buildings with ACBM by type of building and type of material, amounts of ACBM, degree of damage, and other characteristics that determine the extent of potential ACBM problems. Air monitoring data collected in studies to develop regulatory programs provide direct estimates of average exposure levels. High airborne asbestos levels might be used to justify regulations requiring aggressive control measures. Uniformly low levels might be used to support proposals for a limited regulatory program. It is unlikely, however, that low measured concentrations would constitute sufficient support to forgo all regulatory activities. If ACBM is found in a large proportion of buildings, the potential for disturbance, absent any training or special work practices, resulting in significant fiber release cannot be ignored.

The remainder of this paper consists of 2 sections. The following section contains a description of three recent studies of exposure potential using visual inspection, air monitoring, or both methods. These studies are: (i) EPA's analysis, based on both visual inspection and air monitoring of public and commercial buildings with ACBM, conducted for a Report to Congress on regulatory options; (ii) a New York City Department of Environmental Protection (DEP) study using the visual inspection approach to determine what, if any, regulations concerning management of in-place ACBM (i.e., ACBM not involved in renovation and demolition activities) should be developed; and (iii) air monitoring in schools, conducted by defendants in property damage litigation. In each application, objectives, approach, results, and subsequent actions are discussed. The final section of the paper is a brief summary of the findings and includes conclusions.

RECENT APPLICATIONS OF ASSESSMENT METHODS

EPA's Report to Congress

Section 213 of the Asbestos Hazard Emergency Response Act of 1986 (AHERA) required EPA to prepare a report to Congress which was to:

- assess the extent to which asbestos-containing materials are present in public and commercial buildings;
- assess the condition of asbestos-containing materials in commercial buildings and the likelihood that persons occupying such buildings including service and maintenance personnel, are, or may be exposed to asbestos fibers;
- consider and report on whether public and commercial buildings should be subject to the same inspection and response action requirements that apply to school buildings;
- assess whether existing federal regulations adequately protect the general public, particularly abatement personnel, from exposure to asbestos during renovation and demolition of such buildings; and
- include recommendations that explicitly address whether there is a need to establish standards for, and regulate asbestos exposure in, public and commercial buildings.

EPA initiated a multi-part study to meet these requirements.

Objectives.

Study objectives were developed from the first two items in Section 213 of AHERA, which are listed above. Item one indicates that an inventory of ACBM in public and commercial buildings is required (e.g., number of buildings, types and locations of material, and amounts). Item two indicates the need to characterize the condition of ACBM and to evaluate the likelihood of exposure to airborne asbestos fibers by occupants of buildings with ACBM. The results of the study were intended to answer, in part, the last three items of AHERA Section 213.

Approach.

The inventory and condition information were obtained from data collected previously in a statistical survey of the U.S. building population (EPA, 1984). During 1983 and 1984 EPA conducted a national survey of buildings, which had the following stated objectives: (i) to estimate the number of public and commercial buildings which have asbestos-containing friable materials; (ii) to estimate the square feet of the material in such

buildings; and (iii) to estimate the percent of asbestos in the material. Separate estimates were developed for sprayed or trowelled-on surfacing materials, thermal system insulation (TSI), and ceiling tile. A visual inspection scheme was employed to identify friable material and assess its condition.

The survey was designed to produce results representing the U.S. building population. Buildings were selected in a three stage, stratified, clustered statistical sampling design from ten geographic regions. Strata of Federal government, residential, and private nonresidential buildings were established in each region and individual buildings were drawn using a probability proportional to size (building square feet) selection scheme. Residential buildings with fewer than 10 dwelling units were not included in the survey.

The sample design specified 224 buildings: 25 percent Federal government buildings; 25 percent residential buildings; and 50 percent private nonresidential buildings. The size of the survey was based on a compromise between cost and statistical estimation error. An estimation error of ±200,000 buildings (with 95% confidence) was specified as acceptable for estimating the total number of buildings with asbestos-containing sprayed or trowelled-on friable material. The completed survey consisted of 231 buildings: 66 in the Federal government category; 55 in the residential category; and 110 in the private nonresidential category.

To address item 2 in Section 213 of AHERA, the likelihood of exposure of building occupants to airborne asbestos, EPA: (i) used data on the type, location, and condition of ACBM collected in the 1983-1984 building survey; and (ii) conducted an air monitoring study (EPA, 1988b). The design for the air monitoring study specified three types of buildings: Category (1) - buildings without friable ACBM; Category (2) - buildings with most ACBM in good condition allowing for a few areas of moderate damage; and Category (3) - buildings with at least one area of significantly damaged ACBM or many areas of moderately damaged ACBM. The study was conducted in buildings owned by the Federal government and managed by the General Services Administration (GSA) because these buildings posed no barriers to immediate access and the locations, types, and condition of ACBM in these buildings had been determined previously by GSA.

Five cities in different geographic regions of the U.S. were selected for the study. The plan specified 2 buildings in Category 1, 2 buildings in Category 2, and 6 buildings in Category 3 to be selected from each region for air monitoring. Buildings in each region were assigned to categories based on prior GSA evaluations. Buildings were selected for the study at random from each category and were re-inspected to verify the GSA evaluation. The total sample of buildings was intended to consist of 10 buildings in Category 1, 10 buildings in Category 2, and 30 buildings in Category 3. Based on this design, the likelihood of correctly detecting a fivefold or greater difference between airborne asbestos levels in Category 3 and either Category 1 or 2, using standard statistical

methods to test for this difference, would be at least 0.90 (i.e., the statistical power of the test was at least 0.90).

Fewer than anticipated Category 1 and 2 buildings owned by the Federal government were found in five regions. The resulting sample consisted of 6 buildings in Category 1, 6 buildings in Category 2, and 37 buildings in Category 3.

The air monitoring plan specified one outdoor and seven indoor samples for each building. Approximately half of the indoor samples in each building were collected near the ACBM that exhibited the greatest damage. The remaining samples were collected in public areas of the building. Each sample was taken over a two day period, eight hours per day during periods of normal building activity. The samples were analyzed using TEM and the direct filter preparation method. All asbestos structures were counted in the estimate of airborne asbestos concentration.

Results.

The inventory and assessment results from the survey of 231 buildings are summarized in Tables 1 and 2. The results indicate that in 1984 733,000 buildings (20 percent of the target population of 3.6 million U.S. buildings) had some type friable ACBM. In total, 5 percent of the buildings had friable surfacing ACBM and 16 percent had TSI containing asbestos. Approximately 14 percent of the buildings had ACBM classified as "damaged" and 9 percent had "significantly damaged" ACBM (Table 2).

Results on condition were not reported for surfacing ACBM and TSI ACBM separately. In fact, assessing ACBM for condition was not a primary objective of the 1984 EPA survey. The procedures used in that study for assessing condition were not precisely defined in advance. The results, therefore, are best interpreted qualitatively as an indication that some damaged ACBM can be found in these types of buildings.

Results from the air monitoring study, which are summarized as asbestos structures per cubic centimeter of air (s/cc), indicate average levels below 0.001 in each of the three building categories and outdoors (Table 3). Although statistical tests applied to the data suggest statistically significant higher levels in Category 3 (i.e., buildings with some significantly damaged ACBM) than in Category 1 or outdoors, all measured levels are low and the differences among categories are unlikely to be significant from a health risk perspective.

Actions.

The results summarized above were presented in EPA's Report to Congress (EPA, 1988a). The report recommended no new regulatory initiatives. Lee Thomas, EPA Administrator at that time, transmitted his recommendations in a letter to Congress included in the report. The recommendations were:

- enhance the nation's technical capability for managing asbestos;
- focus attention on thermal system insulation asbestos;
- improve integration of asbestos-related programs to reduce imminent hazards; and
- objectively assess the effectiveness of the AHERA school rule and other current activities.

These recommendations are influenced only to a limited extent by the data collected in the two studies described above. There is no reference to the air monitoring findings, and the national survey of 231 buildings is used only to suggest that buildings do in fact have ACBM and that more buildings have TSI, which is often damaged, than have surfacing ACBM. Additional research was recommended to evaluate the effectiveness of current activities of the States and private sector. Thomas warned that steps be avoided that would undermine the school asbestos program. In particular, he cautioned that no new regulations should be considered that would divert the limited supply of professional asbestos specialists from the school program.

New York City Study of In-Place Asbestos

The City of New York Local Law No. 76, adopted in December, 1985, was designed to protect public health by minimizing exposure to airborne asbestos in public and private buildings in New York City. The law has two general requirements: first, that regulations be promulgated by the New York City DEP to address worker exposure during renovation, alteration, and demolition involving ACBM; and second, that DEP submit a report to City Council "...identifying and evaluating the potential risks, if any, which may be posed to workers and the general public from asbestos which is not disturbed by renovation, alteration or demolition work and recommending action deemed appropriate to ameliorate such risks as may be identified...." (The City of New York Local Law No.76, Section 9). This second requirement led to a study of exposure potential using the visual inspection method. DEP's study was a survey of buildings in New York City, which involved inspection, bulk sampling, and visual assessment of ACBM.

Objectives.

The study objectives were:

- estimate the amount and distribution of in-place asbestos present in various types of buildings in New York City;
- determine the potential for exposure of occupants and workers to in-place asbestos in each building category;
- identify building categories and types of areas in buildings that warrant a management program to reduce or control potential for exposure; and
- identify and evaluate asbestos management activities available to the City and develop regulatory options based on these suggested management activities. (DEP,1988).

Approach.

A statistical survey of buildings in New York City was conducted. The plan specified a sample of 900 buildings equally divided among 18 building categories representing the approximately 800,000 buildings in the City. (During the study, the number of building categories was reduced to 16; the number of building inspections completed was 886.) In each building, data were recorded on: the type, location, and amount of material; its condition and potential for disturbance; and the number of people in proximity to the ACBM. Bulk samples were extracted and analyzed to test for the presence of asbestos. These data were collected for surfacing material and TSI separately. Information also was obtained for ceiling tile, floor tile, and other materials, typically not friable, that may contain asbestos.

In each building, data were collected by functional area. "A functional area is a clearly-delineated space containing well-defined activities and people who participate in those activities." (DEP, 1988). All data were summarized and reported by type of functional area. Four major functional area types were defined - mechanical areas, common areas, living/working areas, and special use areas. (These were further subdivided into 26 minor functional area types to allow a more detailed evaluation of the data if needed.)

Each type of ACBM in each functional area was assigned an exposure assessment score that represented the potential for exposure. The assessment scheme is shown in Table 4. (For definitions of good, fair, poor, high, moderate, low, etc., appearing in the table, refer to DEP's report.) The inspectors conducting the inspections were trained as a group and a subset of buildings were re-inspected to verify the inspection results.

Although the scoring scheme incorporates a high degree of detail, the interpretation of these data was based primarily on the condition rating of the ACBM.

Results.

Approximately two thirds of all buildings surveyed in New York City have ACBM. The estimated total amount of ACBM in New York City buildings is 323 million square feet. The majority of this material, 84 percent, is TSI. Fifteen percent (48.6 million sq. ft.) is surfacing material and 85 percent of the surfacing material containing asbestos is in tall office buildings. Table 5 shows the number of buildings by types of material in New York City compared to national results obtained by EPA. (One- and two- family dwellings in New York City are not included in the counts.) A significantly greater proportion of buildings in New York City have TSI ACBM than nationally.

Table 6 summarizes the condition of the ACBM in New York City buildings by type of material. TSI, both in terms of amount and condition (i.e., 19 percent in poor condition), stands out as the ACBM to receive regulatory management attention. The majority of these materials are, to some extent, isolated with 51 percent of the TSI in mechanical areas and 58 percent of the TSI in poor condition also in mechanical areas.

A number of other summary statistics were reported. In particular, a more detailed analysis of building categories, functional areas, and material types by exposure assessment score was developed. The numbers and types of people usually visiting each type of functional area also were estimated. Although these detailed results may be used in the future to refine regulatory recommendations, they had a minimal role in determining the selected course of action described in the DEP report.

Action.

Chapter 4, Asbestos Management Programs, of the DEP report identifies five major components of a program to limit the likelihood that asbestos fibers could be released from ACBM and become airborne: (i) inspection; (ii) worker education and training; (iii) operations and maintenance; (iv) remedial action; and (v) development of management plans. The report states:

> The survey results in Chapter 3 suggest regulatory alternatives and priorities for implementing management programs. The data suggest, first, that one- and two- family dwellings should be considered separately from the other building categories. These buildings account for approximately 70 percent of all buildings in New York City and are typically owner-occupied. Furthermore, all of the ACBM found was TSI, predominately, pipe wrap and elbows. It was found in small amounts [in each dwelling] and approximately

> 94 percent of it was in the basement. Considering the five basic program elements discussed earlier, only inspection and remedial actions apply to one- and two- family dwellings. Instead of formal service worker training and an operations and maintenance program, homeowner awareness and education is appropriate.
>
> The data suggest that the other categories of buildings should not be treated differently from each other because there were broad similarities in the amount, nature and condition of asbestos. The basic program elements discussed earlier, currently being implemented by many building owners and recommended by EPA are appropriate for buildings other than one- and two- family dwellings. (DEP, 1988, p. 4-2).

An index was developed to rank the 15 building categories (excluding one- and two- family dwellings) for regulatory compliance priority. The index is the product of: the percent of buildings with ACBM in the category; the percent of ACBM in poor or fair condition; the average amount of ACBM per building; and the average number of person-area-visits by service workers and resident/workers per building to areas containing poor or fair ACBM. The index was used to separate the building categories into the three groups (Table 7). Compliance with the regulatory program would be required for the first group starting in 1990; for the second group in 1993; and for the third group in 1996.

For one- and two- family dwellings, educational and guidance materials concerning health risk and abatement would be provided starting in 1990. In addition, the City would require inspection and disclosure of inspection results at the time of property transfer. This requirement would begin in 1993.

DEP followed the report with a proposal for legislation to be enacted by City Council, which would provide authority for DEP to promulgate the necessary regulations. As of March, 1989, this legislation had not been enacted.

Air Monitoring by Three Building Products Manufacturers

An air monitoring data base is being developed jointly by three building products manufacturers (BPM's) who are defendants in property damage lawsuits involving asbestos. Air monitoring is conducted in schools where asbestos removal is imminent and the school district has filed a suit against the manufacturer to recover removal and related costs.

To date 288 samples collected in 73 schools have been analyzed and summarized. These data have been introduced through depositions by technical experts and have been the basis of analyses leading to submissions to EPA regarding the AHERA school regulation (Crump, 1987a; 1987b).

Objectives.

Airborne asbestos concentration estimates developed from BPM air monitoring data have two purposes: (i) to establish baseline levels in specific buildings prior to asbestos removal; and (ii) to characterize airborne asbestos levels inside school buildings in general. In the first case, the results are used to weigh the merits of alternative asbestos management approaches for a specific building. In the second case, the results are intended for use in suggesting and evaluating public policy and regulatory alternatives for managing asbestos in buildings through regulation.

Approach.

The buildings selected for sampling are determined by the schedule for asbestos removal established by the plaintiffs. The samples may be stored for analysis at a later date that is determined by the litigation schedule.

The sampling protocol follows the NIOSH Method 7400 requirements with 2000 liters of air as the target sampling volume. Sampling is intended to cover the majority of one or two school days during regular activities. (Note that 2000 liters collected at the recommended flow rate of 2 liters per minute requires 16+ hours of sampling - i.e., two full work days.) At least three sampling locations, identified with high, moderate, and low activity levels, are selected in each building. Multiple samples are prescribed for each of the locations in large buildings. One or more of the samples are taken in areas where the products alleged to contain asbestos are located and, whenever possible, in areas where no ACBM products are located. Between two and five outdoor samples are collected at each building. TEM analysis is used with the direct filter preparation method to count asbestos fibers. Results are reported for: fibers longer than five microns; AHERA structures (i.e., structures defined in the AHERA school regulation); chrysotile only; amphibole only; and all structures.

Results.

The BPM TEM air monitoring results are summarized in Table 8. The concentration indices reflecting fiber types generally regarded to be of greatest health concern (i.e., fibers longer than 5 microns and amphibole fibers) have low average fiber counts. For fibers longer than five microns, the indoor average is 0.00022 f/cc; for amphibole fibers, the indoor average is 0.00073. The indoor averages in both cases are greater than the corresponding outdoor averages and the differences are judged to be statistically significant based on the Mantel-Haenszel test. The indoor and outdoor fiber levels, however, are low. Consequently, neither the absolute nor the differences are likely to be significant from a health risk perspective.

SUMMARY AND CONCLUSIONS

Visual Inspection

The visual inspection methods employed in the EPA and DEP studies discussed above each had two distinct components. One component dealt simply with the inventory of ACBM. The statistical survey approach provides an efficient way to estimate inventory (i.e., numbers and types of buildings, and locations and amounts of ACBM). The proportion of buildings with ACBM and the average amounts of ACBM in the buildings were sufficient both in the EPA and DEP studies to support recommendations for a general program to inspect buildings to identify ACBM. Additional information about locations and amounts could be used to formulate effective work practices and worker training programs. Survey results also indicate the types of situations where the amounts of asbestos are sufficient to warrant aggressive regulatory activity and, in that respect, aid in setting priorities.

The second component, assessing exposure potential, is a more complicated undertaking. In general, the development of surrogate measures for exposure has not been successful. In 1980, a numerical scoring system (referred to as the algorithm) was proposed by the U.S. Department of Education and EPA (45 FR 61950 September 17, 1980). Although the subjectivity inherent to that approach was acknowledged at the time, the description of the method nevertheless implied an objective numerical rule for determining if and which type of corrective action was appropriate. EPA subsequently shifted to a less formal assessment system reflected in the Purple Book (EPA, 1985). The value, however, of factors other than condition of the material being assessed has not been clearly established. When considering potential exposure of typical building occupants, only the presence of asbestos and its condition can be readily determined and are relevant.

The New York City study further demonstrates this point. The inventory data focused the regulators' attention on TSI in mechanical areas and indicated that surfacing ACBM was, primarily, in office towers. The data on condition of the ACBM, again, pointed the regulator to TSI (19 percent of the TSI and 1 percent of the surfacing material was in poor condition). Although the building survey inspectors collected detailed information on factors leading to an evaluation of the potential for disturbance, this information was not used explicitly in formulating the recommended regulatory approach. The index used for setting priorities (i.e, determining which group of building categories should be regulated first, second, and third) was based, primarily, on inventory and condition.

Assessment indices that rely on visual inspection factors other than inventory and condition to estimate exposure potential are likely to produce scores that are different for different inspectors. The best indicator of potential unplanned disturbance of ACBM (i.e.,

disturbance not associated with repair or renovation, which is planned and can be properly controlled) is its current condition. If ACBM is present and in acceptable condition, the only way to determine if exposure is occurring is to monitor the air for asbestos fibers.

Air Monitoring

The asbestos air monitoring data base for buildings, which has grown significantly during the past two years, indicates low average levels of airborne asbestos under normal building use conditions. Early TEM measurements of airborne asbestos levels (EPA, 1975; Nicholson, 1978; EPA, 1980; EPA, 1983), which were large enough in EPA's judgement to justify initiation of an asbestos regulatory program, were obtained using the indirect filter preparation method. Measurements by indirect TEM may overestimate the number of respirable asbestos fibers. These measurements, therefore, were reported in mass units (nanograms per cubic meter of air). Exposure measured in mass units, however, does not reflect heath risk as directly as exposure measured in fibers per cubic centimeter of air (f/cc). More recent data, obtained using TEM and the direct filter preparation method, are reported as f/cc and indicate lower average exposure levels than were implied by the earlier measurements obtained with the indirect method.

EPA's recent report to Congress concerning asbestos in public and commercial buildings (EPA, 1988a) provides a summary of direct TEM data for 94 non-school buildings and 41 school buildings. The average airborne asbestos concentration in non-school buildings is 0.006 f/cc. The average for the school buildings is 0.03 f/cc. Since these data were compiled from a number of different air monitoring studies with different degrees of documentation, the averages should be interpreted as representing concentrations for all asbestos structures visible with TEM (i.e, fibers both longer and shorter than 5 microns and other asbestos structures that may not be considered respirable).

Detailed documentation, however, is available for the 43 non-school buildings with ACBM studied by EPA and 73 schools where asbestos removal was imminent studied by building products manufacturers (EPA, 1988b; Crump, 1987a). In the EPA study, the average airborne asbestos concentration in buildings with ACBM when counting all structures was 0.00070 s/cc (Table 9). Of the 387 samples collected in this study, including 48 outdoor samples and samples in 6 buildings with no friable ACBM, 83 percent yielded a zero asbestos fiber count. Restricting the asbestos count to only those fibers 5 microns in length or longer (i.e., the fibers more likely to be associated with asbestos-related disease) resulted in an average concentration level approximately 8 times lower, 0.00008 f/cc (Table 9). In the 73 school buildings studied by the building products manufacturers, the average airborne concentration counting all asbestos structures was 0.01773 s/cc. Restricted to fibers 5 microns or longer, the average concentration was 0.00022.

The average of measurements taken outdoors in these studies is comparable to the measured indoor levels. In general, these results demonstrate that prevalent airborne asbestos levels inside buildings with ACBM are low and suggest that in most buildings the difference between prevalent indoor and outdoor asbestos fiber levels is not significant from a health risk perspective.

REFERENCES

Crump, K. S., and Farrar, D. (1987a). Statistical Analysis of Airborne Asbestos Fiber Data Collected from Schools and Analyzed by Transmission Electron Microscope. In Comments of National Gypsum Company submitted to EPA regarding Proposed Rule on Asbestos-Containing Materials in Schools. June 29, 1987.

Crump, K. S. (1987b). Comments on Issues Related to the Percentage of EPA Criterion Fibers That Are Longer Than 5 Microns and on the Potential Effects Upon Average Fiber Concentrations of High Transient Levels. In Supplemental Comments of National Gypsum Company submitted to EPA regarding Proposed Rule on Asbestos-Containing Materials in Schools. October 9, 1987.

Crump, K. S., and Farrar, D. B. (1989). Statistical Analysis of Data on Airborne Asbestos Levels Collected in an EPA Survey of Public Buildings, Regulatory Toxicology and Pharmacology, 10:51-62.

DEP, (1988). Final Report on the Assessment of the Public's Risk of Exposure to In-Place Asbestos. City of New York Department of Environmental Protection, New York, New York.

EPA, (1975). Asbestos Contamination of the Air in Public Buildings. EPA-450/3-76-004. USEPA, Research Triangle Park, North Carolina.

EPA, (1980). Measurement of Asbestos Air Pollution Inside Buildings Sprayed With Asbestos. EPA-560/13-80-026. USEPA, Washington, D.C.

EPA, (1983). Airborne Asbestos Levels in Schools. EPA-560/5-81-006. USEPA, Washington, D.C.

EPA, (1984). Asbestos In Buildings: A National Survey Of Asbestos-Containing Friable Materials. EPA 560/5-84-006. USEPA, Washington, D.C.

EPA, (1985). Guidance For Controlling Asbestos-Containing Materials In Buildings. EPA 560/5-85-024. USEPA, Washington, D.C.

EPA, (1988a). EPA Study of Asbestos-Containing Materials in Public Buildings: A Report to Congress. USEPA, Washington D.C.

EPA, (1988b). Assessing Asbestos Exposure in Public Buildings. EPA 560/5-88-002. USEPA, Washington, D.C.

Nicholson, W. J., et al. (1978). Control of Sprayed Asbestos Surfaces in School Buildings: A Feasibility Study. Report to the National Institute of Environmental Health Sciences.

Table 1. Estimated number (in thousands) and percent of buildings with asbestos-containing friable building materials by type of material and type of building - U.S. building population.

Type	Total (000)	Type of Asbestos-Containing Friable Material			
		Surfacing	Ceiling tile	TSI	Any material
Federal	35	5 16%	1 2%	9 25%	14 39%
Residential	350	64 18%	2 1%	155 44%	208 59%
Private Nonresidential	3,221	122 4%	0 <1%	400 12%	511 16%
All	3,606	192 5%	2 <1%	563 16%	733 20%

Source: Tables 1 and 2 in EPA, 1988a

Table 2. Condition of friable ACBM in U.S. buildings (percent) by building type.

Building Type	Number (000)	Condition of Friable ACBM		
		Any	Damaged	Significantly Damaged
Federal	35	39%	14%	1%
Residential	350	59%	23%	2%
Commercial Nonresidential	3,221	16%	13%	10%
All	3,606	20%	14%	9%

Source: Table 4 in EPA, 1988a.

Table 3. Summary of measured airborne asbestos levels based on building averages -7 samples per building. (Concentrations recorded as structures per cubic centimeter of air - s/cc).

Parameter	Outdoor	No ACBM	Bldgs. with ACBM	
		Category 1	Category 2	Category 3
Median	<0.00001	0.00010	0.00040	0.00058
Mean	0.00039	0.00099	0.00059	0.00073
Std. Dev.	0.00096	0.00198	0.00052	0.00072
Number	48 (sites)	6 (bldgs)	6 (bldgs)	37 (bldgs)

Notes: Category 1 buildings had no friable ACBM.

One filter in a Category 1 building produced a measurement of 0.033 s/cc which adds approximately 0.0007 to the mean. If this filter were omitted from the calculations, the mean would be 0.00020.

Source: EPA, 1988b.

Table 4. Exposure Assessment Scoring Scheme

Score	Friable	Condition	Summary of Disturbance Potential	ACBM Disturbance Varaibles		
				Contact	Vibration	Air Erosion
10	yes	poor	any	any	any	any
9	yes	fair	high	high	any	any
9	yes	fair	high	any	high	any
9	yes	fair	high	any	any	high
8	yes	fair	moderate	mod	mod/low	mod/low
8	yes	fair	moderate	mod/low	mod	mod/low
8	yes	fair	moderate	mod/low	mod/low	mod
7	yes	fair	low	low	low	low
6	yes	good	high	high	any	any
6	yes	good	high	any	high	any
6	yes	good	high	any	any	high
5	yes	good	moderate	mod	mod/low	mod/low
5	yes	good	moderate	mod/low	mod	mod/low
5	yes	good	moderate	mod/low	mod/low	mod
4	yes	good	low	low	low	low
3	no	good	high	high	any	any
3	no	good	high	any	high	any
3	no	good	high	any	any	high
2	no	good	moderate	mod	mod/low	mod/low
2	no	good	moderate	mod/low	mod	mod/low
2	no	good	moderate	mod/low	mod/low	mod
1	no	good	low	low	low	low
0	material does not contain asbestos					

Source: DEP, 1988.

Table 5. Projected numbers (in thousands) and percents of buildings containing ACBM by type, nationally and in New York City.

	Building Population			
	U.S.		NYC	
Type ACM	Number (000)	Pct. (%)	Number (000)	Pct. (%)
Any	733	20	153	67
TSI	563	16	153	67
Surfacing	192	5	3	1
Ceiling Tile	2	<1	5	2
Total Estimated No. of Bldgs.	3,606,000		228,000	

Notes: NYC building count does not include schools covered under AHERA or one- and two- family dwellings.

U.S. building count does not include schools covered under AHERA or residential buildings with less than 10 units.

Source: EPA, 1988a; DEP, 1988.

Table 6. Projected percentages of ACBM by type in Good, Fair, and Poor condition in New York City buildings. (Percents based on sq. ft.)

Type of ACBM	Condition			Total Square Feet (millions)
	Poor	Fair	Good	
TSI	19%	68%	13%	272
Surfacing	1%	70%	29%	49
Ceiling	<1%	47%	53%	3

Source: DEP, 1988

Table 7. Index for establishing asbestos management priorities for New York City building categories.

Building Category	Percent of Buildings with ACBM (%)	Percent of ACBM in Poor/Fair Condition (%)	Average Sq. Ft. of ACBM per Bldg. (sq. ft.)	Average Number of Person-Area-Visits in Vicinity of ACBM in Fair or Poor Condition (1) number	Index (2)
First Priority					
Tall Office Buildings	84	76	64,341	1,880	77,314
Hospitals	72	69	6,929	793	2,733
Elevator Apartments	72	91	4,831	682	2,159
Hotels	78	81	3,801	528	1,268
Educational Structures	83	79	3,233	468	992
Second Priority					
Short Office Buildings	64	41	2,109	1,408	779
Government/Trans.	43	77	8,281	156	428
Theatres	57	70	4,437	96	170
Warehouses/Lofts	40	69	2,392	171	112
Factories	61	84	1,759	102	91
Third Priority					
Churches/Synagogues	71	59	919	45	17
Outdoor Recreation	64	77	968	21	10
Walk-up Apartments	74	94	457	24	8
Stores	62	84	363	6	1
Garages/Gas Stations	17	70	419	12	1

Notes: (1) Total person-area-visits by service workers and resident/workers.
(2) Product of the values in columns 1 through 4, divided by 1,000.

Source: DEP, 1988.

Table 8. Concentrations indoors (static and personal) vs. outdoors measured by building products manufacturers in 73 schools.

Description of Sample	No. Samples	Index	Maximum	Mean	Std. Dev.
Outdoor	69	(f≥5 microns)/cc	0.0000	0.0000	0.0000
		AHERA (s/cc)	0.0240	0.0016	0.0037
		Chrys (f/cc)	0.0240	0.0019	0.0038
		Amph (f/cc)	0.0039	0.0002	0.0008
		All (s/cc)	0.0240	0.0021	0.0038
Indoor	288	(f≥5 microns)/cc	0.0120	0.0002	0.0010
		AHERA (s/cc)	0.2900	0.0120	0.0300
		Chrys (f/cc)	0.4300	0.0170	0.0400
		Amph (f/cc)	0.0160	0.0007	0.0019
		All (s/cc)	0.4300	0.0170	0.0400
Overall	357	(f≥5 microns)/cc	0.0120	0.0002	0.0009
		AHERA (s/cc)	0.2900	0.0098	0.0270
		Chrys (f/cc)	0.4300	0.0140	0.0370
		Amph (f/cc)	0.0160	0.0006	0.0017
		All (s/cc)	0.4300	0.0140	0.0370

P - values from Mantel-Haenszel test stratified by school (one-sided tests).

(f≥5 microns)/cc	p = 0.041
AHERA (s/cc)	p = 0.0024
Chrys (f/cc)	p = 0.0019
Amph (f/cc)	p = 0.013
All (s/cc)	p = 0.0015

Source: Update from Crump, 1987a.

Table 9. Average Airborne Asbestos Fiber Levels: Schools Measured by Building Products Manufacturers and Public Buildings Measured by EPA

Fiber Index	Data Source	Indoor Measurements "No ACBM"	"ACBM"	"Sig Dam"	Outdoor
(f≥5 microns)/cc	BPM	-	0.00022	-	0
	EPA	0	0.00008	0.00008	0.00010
AHERA (s/cc)	BPM	-	0.01200	-	0.00160
	EPA	0.00073	0.00054	0.00058	0.00031
Chrys (f/cc)	BPM	-	0.01700	-	0.00190
	EPA	0.00083	0.00055	0.00060	0.00028
Amph (f/cc)	BPM	-	0.00073	-	0.00019
	EPA	0.00016	0.00013	0.00012	0.00010
All (s/cc)	BPM	-	0.01773	-	0.00210
	EPA	0.00099	0.00070	0.00073	0.00039

Notes: BPM - Data collected by building products manufacturers in 73 schools.
EPA - Data collected by EPA in 49 public buildings. "No ACBM" means no friable ACBM. One filter in the "No ACBM" category produced a measurement of 0.033 f/cc which adds approximately 0.0007 to the average for the index "All." If this filter were omitted from the calculations, the average would be 0.00020.

Source: Based on Crump, 1987a; 1989.

AIR SAMPLING AND ASBESTOS-CONTAINING MATERIALS IN BUILDINGS

PETER S.J. LEES

Assistant Professor, Johns Hopkins University, School of Hygiene and Public Health, Department of Environmental Health Sciences, Division of Environmental Health Engineering, 615 N. Wolfe Street, Baltimore, MD 21205

Introduction

The legitimate uses of air sampling for the evaluation of buildings with asbestos-containing materials (ACM) has become a source of disagreement and contention within the asbestos abatement and management community. This controversy, often voiced against a background of litigation, is usually marked by technical arguments about the adequacy of the analytical techniques or simplified arguments about the perceived inadequacies of the sampling process. Many of these arguments reveal a basic lack of conceptual understanding of air sampling and the application of the information collected. In addition, pervasive legal and regulatory pressures have prevented an objective examination of its usefulness.

The purpose of this paper is to examine air sampling from a conceptual basis, to evaluate its uses and value in the building evaluation process, and to make judgments on its strengths and weaknesses. Also, gaps in the understanding of air sampling are identified, and areas for future research which could contribute significantly to the understanding of risk associated with the use of ACM in buildings are proposed.

There are many potential uses of air sampling in a building environment, as enumerated in Table 1. The use of air sampling during and after abatement, as an emergency response measure, and even as a part of operations and maintenance programs is well established (1). Its use to define occupational exposures of persons working with ACM in buildings is also well established (2). These uses of air sampling, therefore, are not discussed in this paper. The major controversy about the efficacy of air sampling is related to its use as a decision-making tool in hazard or exposure evaluation and response. Two different purposes for air sampling may be identified in this regard: public policy making (what actions should be taken with regard to asbestos in buildings to safeguard public health?) and decision-making on individual building basis (what should be done with the asbestos-containing acoustical ceiling on the third floor?). This paper will focus on the

latter purpose, the use of air sampling as a decision-making tool for determining appropriate action in the assessment of an individual building.

Different purposes for air sampling have different associated sampling strategies. Air sampling data used for public policy making are necessarily very broad; sampling is designed to determine if elevated airborne asbestos concentrations are associated with the presence of ACM in buildings and thus, if a generic public health problem exists. This type of sampling, which has been the focus of most EPA-sponsored and other published research usually includes a range of buildings, with sampling focused on buildings with ACM in poor condition. Virtually all published reports which include a significant number of air samples are of this type, i.e. air samples from many buildings, but few in each building, designed to establish the generic relationship between airborne fiber concentrations and ACM in buildings. Air sampling data used for decision-making in individual buildings, on the other hand, are much more narrowly focused. This type of air sampling is designed to determine if a health hazard (exposure) exists at a given location (regardless of the generalized relationship drawn from air sampling conducted to determine if a public health problem exists) and eventually to define the variables or conditions which will alter current exposure. This type of air sampling requires collection of a significant number of samples in the building; there are virtually no published studies of this type. Because of biases introduced by these different sample collection strategies, the air sampling data collected for one purpose may have limited use for a different purpose.

There are two main schools of thought concerning the usefulness air sampling as a decision-making tool in assessing buildings with ACM. The U.S. Environmental Protection Agency (EPA) has historically discounted air sampling as a means of evaluating ACM and for determining an appropriate response action (3,4,5). Many of the arguments used against air sampling evolved from the initial widespread inappropriate use of occupational exposure assessment tools (low volume samples analyzed by phase contrast microscopy) and OSHA exposure standards in a building environment. More substantial questions about air sampling raised by EPA include the representativeness of air samples and the inability of air samples to detect transient "peak" exposures. Others (6,7,8) contend that air sampling can be a valuable tool to aid in the determination of the level of health risk in a building and, as such, is a basis for action. They argue that since the magnitude of the health risk related to ACM in buildings is a function of exposure, better definition of exposure and those factors which influence it will greatly add to the understanding and estimates of risk and will provide a rational basis for institution of control measures.

Historical Background

EPA's publicly stated position on air sampling has evolved little over the last ten years. This position has been defined in a series of guidance documents commonly referred to as the Orange Book (3), the Blue Book (4), and the Purple Book (5).

In 1978 the earliest EPA guidance document (3) stated that "[a]ir sampling is inappropriate to estimate asbestos contamination and exposure. In the school environment, it is virtually impossible to establish exposure potential using standard air sampling techniques. Air sample results from monitoring in school buildings can be misleading if they are compared to the Federal workplace air concentrations levels established by OSHA." In addition, the limitations of phase contrast microscopy (PCM), which was commonly employed at that time for the analysis of air samples collected in a building environment, were described.

Five years later EPA (4) stated that "[a] positive finding (of ACM) indicates the need for a thorough assessment of the potential for exposure to airborne asbestos, followed by the consideration of appropriate corrective measures. ... Another proposed approach to assessing the need for corrective action is to measure asbestos fibers in the air. At best, this approach provides information only on current asbestos contamination and no information about the potential for fiber release and future air levels. Moreover, the use of air monitoring as an assessment tool involves substantial technical and economic problems which limit its use for determining current levels of contamination." The technical problems associated with the use of PCM are recapitulated and the technical and financial problems associated with the use of transmission electron microscopy (TEM), which had come into use since the previous guidance document, were also described. EPA also stated that even after these technical problems are resolved, "the difficulty of sampling in occupied buildings and the number of samples needed to detect peak as well as prevalent airborne levels may preclude the use of air monitoring for routine assessment."

Two years later in the final guidance document to released date (5), EPA stated that "[a]nother way to assess asbestos fiber release is to measure asbestos fibers in the air. This approach is appealing because it quantitatively measures airborne asbestos contamination. However, it measures only current conditions and provides no information about fiber release potential and future air levels. Moreover, implementing an effective air monitoring program to measure current levels of airborne asbestos is difficult and can be expensive." The shortcomings of PCM and the expense of TEM are again discussed. EPA also stated that "... obtaining enough samples to estimate prevalent airborne levels is difficult in occupied buildings" and concluded that "[t]hese limitations, combined with the inability of air monitoring to provide information on future conditions, restrict its usefulness for assessment. EPA, therefore, does not recommend is as a primary assessment tool at this time."

Others (6,7,8) contend that air sampling can be a valuable tool to aid in the determination of the level of exposure/health risk to occupants of a building and, as such, is at least a partial basis for decision-making. It is their position that quantitative air sampling data should be used in conjunction with subjective factors including the results of visual inspection, judgments (e.g., product failure), intimate understanding of how the building is operated (HVAC systems, renovation schedule, etc.), and future building uses to help decide what actions are appropriate to minimize health risks to building occupants. This approach to decision-making is derived from the orthodox approach to virtually all environmental contaminants, i.e., the need for action is based upon quantitative measures of a contaminant in the environment. Samples are collected from the environmental media (air, water, or soil), analyzed for the contaminant in question, and decisions made by comparing sample results to regulations, guidelines, or other estimates of what is acceptable. This model has long been used in virtually every instance involving occupational, para-occupational, and environmental exposures and forms the basis of virtually all current EPA and OSHA regulations. To deny the efficacy of air sampling for asbestos in buildings is to deny the basis of efforts to reduce environmental and occupational exposures in this century.

Adherents of air sampling are also struck by apparent contradictions in EPA policies and regulations. For instance, is it not a contradiction that EPA requires air sampling and has set standards for post-abatement clearance, yet claims that air sampling and standards are inappropriate to determine when abatement is required? What is different about exposure in these instances and how can some airborne asbestos concentration be acceptable after abatement but not before? Is it not a contradiction that EPA states that it is not possible to characterize a building based on a few samples, and then claims to accomplish just that in their own air sampling studies (e.g., 9,10)? Is it not a contradiction that EPA claims air sampling is not useful because it does not predict future concentrations and then invalidates its own algorithm (designed to predict future problems) because it does not correlate with current air sampling results (10)?

In the absence of a generally agreed upon acceptable asbestos fiber exposure standard for building occupants, building evaluation schemes utilizing results of air sampling employ a "relative risk" approach in which the airborne asbestos concentration measured in the building is compared to the simultaneously measured ambient fiber concentration. If the fiber concentration in the building is less than or approximately equal to the ambient concentration, action is not indicated and additional sampling is not required to make a decision. (Additional sampling is required in the future, however, to assure that this condition has been maintained.) If the fiber concentration in the building is found to be only a few times the ambient fiber concentration, there is not a clear basis for action and more frequent follow-up (sampling) is required to make a final determination. If the fiber

concentration in the building is many times the ambient fiber concentration, action is indicated and further sampling is not required. This method of assessing air sampling results is based upon the medical model for clinical testing and test interpretation: if results are normal, testing need only repeated at some future time as part of a routine check-up; if results are borderline or slightly elevated, additional testing is indicated; if results are clearly abnormal, action is indicated. This model has been criticized because it implies less than very precise interpretation of air sample results. In fact, any air sample results need only be interpreted in terms of the action that will result, i.e., leave the ACM undisturbed, conduct more sampling, or undertake abatement activities. Given the great variability in asbestos fiber counting (11), placement of the decimal place to define the concentration may be sufficient evaluation of air sampling in most occupational and para-occupational environments. In addition, regardless of the fiber concentration, air sampling results can be used as a quantitative measure to prioritize actions given limited funds.

The most obvious difference between the two conflicting points of view on the efficacy of air sampling is that each perceives a different role or purpose for air sampling. EPA faults air sampling largely on its inability to define current fiber concentrations or predict future fiber concentrations; air sampling proponents praise it for its ability to quantify current risk levels and rely on future sampling to quantify future levels. The basis of these different points of view appears to have evolved from different implicit assumptions about the variability of airborne fiber concentrations in buildings. It follows from their arguments that EPA assumes great variability and rapid change in fiber concentrations while the proponents of air sampling assume low variability and very slow change in fiber concentrations in buildings. Both sides are forced to rely on data collected for other purposes which are, therefore, less than adequate to substantiate their points. Since the two sides don't agree on the purpose of air sampling and make different assumptions about the basic underlying character of airborne fiber concentrations in buildings, there is little wonder they don't agree on its efficacy.

Sampling Issues

There are five basic arguments, summarized in Table 2, that have been used to discredit the efficacy of air sampling as a decision-making tool for individual buildings. Of these arguments, three are now mostly historic artifacts, but two are still debated. The historic arguments, those for which a consensus now appears to exist, are reviewed below.

The question of the appropriate microscopic method, PCM or TEM, is now historic. There is general recognition and agreement that PCM is inadequate to resolve the exceedingly thin asbestos fibers which may be present in a building atmosphere. Although

some technical problems and controversy are still associated with the use of TEM (direct vs. indirect preparation techniques; apparent undercounting of large fibers), it has found wide use. With wider use, availability has increased greatly and there has been a concomitant reduction in cost. With increased knowledge and recognition of the relationship between fiber size and disease (12), TEM-generated fiber size distributions can only become increasingly important.

At one time the argument was made that air sampling surveys were difficult to conduct in occupied buildings (4). The rationale for this statement has never been explained. Air sampling surveys, however, are now routinely conducted in all types of buildings and are considered more valid when the building is occupied.

Finally, the argument that the OSHA standard for asbestos exposure is not relevant for building occupants (3) is now well accepted. The logic that a standard can be developed for one type of asbestos exposure/population and not another, however, remains unclear.

The two major areas of controversy which remain are addressed below.

The "Snapshot" Issue

EPA has long maintained that air sampling is not a valid method of exposure evaluation in a building environment because the concentration measured represents only a "snapshot" in time, that is, it only represents the fiber concentration at the time and place the sample was collected. As such, it is argued, air sampling does not adequately define current exposure of building occupants. By extension, the argument is made that air sampling can not be used to predict future exposures. The over-simplification of this argument reveals a basic conceptual misunderstanding of what air sampling represents. A conceptual examination of air sampling requires that an air sample be thought of not in terms of a volume of air drawn through a piece of filter paper, but rather as a "sample" in the statistical sense. "A sample consists of a small collection from some larger aggregate about which we wish information. The sample is examined and the facts about it learned. Based on these facts, the problem is to make correct inferences about the aggregate or population. It is the sample that we observe, but it is the population which we seek to know" (13). Placed in the context of ACM in buildings, an air sample is, strictly speaking, a measure of the fiber concentration at a particular time and space. The more important and more appropriate question, completely missing from EPA's over-simplified argument, becomes how representative is this sample of the aggregate population or how does this sample in time and space represent all samples in time and space? This question must be answered in order to generalize the air sampling findings and to use air sampling as a predictive tool.

From the purely statistical perspective, the representativeness of the sample mean (which is used to estimate the population mean) is a function of the variability of the aggregate population and the number of samples collected to estimate it. While one can control the number of samples collected, time and cost considerations dictate that the number be minimized. Thus, an understanding of the variability of the population becomes essential.

Before turning to the question of the variability of airborne asbestos concentrations as it relates to ACM in buildings, it is instructive to examine air sampling and contaminant concentration variability in other contexts. OSHA utilizes air sampling data to determine compliance with prescribed permissible air concentrations for several hundred chemicals in the workplace. Despite the millions of air samples collected in the workplace, few published studies adequately describe the variability of contaminant concentration for a given location or a given job. This is because valid measures of population variability require random or unbiased sampling; most air samples in the workplace are collected for compliance purposes in which the highest exposed individuals are sampled. This means that only part of population is represented by the sample, i.e., this sampling strategy is a biased estimator of the population mean and population variability. This is an excellent example of why it is difficult to use information collected by one sampling strategy for one purpose (compliance) for another purpose (definition of population mean and variability) which would require a different sampling strategy.

There are, however, limited data which can be used to define expected concentration variability in several environments. NIOSH (14) in a survey of (presumably) unbiased occupational exposure data which incorporate both spatial and temporal variability in an indoor workplace indicate mean sample geometric standard deviations (GSD's) to range typically from 1.5 to 2.5; this is an indication of moderate variability. The EPA continuous ambient air quality monitoring data represent another unbiased source of concentration variability data which can be utilized to isolate temporal variability (the monitoring is conducted at fixed sites). Air quality data for carbon monoxide, for instance, display GSD's on the order of 2.0 to 2.5 (15).

How much variability is expected for measurements of asbestos concentrations in buildings? Subdividing this question into spatial and temporal variability, let us first examine spatial variability. Again, despite the millions of air samples collected in buildings, most have been collected to answer other questions and are, therefore, of little use to describe fiber concentrations in an "normal" (i.e., no abatement activity) building. In the few published series of air sampling data from "normal" buildings (e.g., 9,16,17), data are presented in summarized form representing results from multiple buildings and are generally not identified sufficiently to permit assessment of variability within a given

building. In the few cases in which the individual sample results are reported, there is an insufficient number of samples from any one building to adequately assess spatial variability.

In the absence of a major body of substantiating data, indirect inference represents the only other source of information on spatial variability of airborne asbestos concentrations in a building. Comparison of the possible sources of variability of contaminant concentration in the general building environment with the sources of variability in the occupational environment will at least provide a relative ranking of their magnitude. In the occupational environment, multiple discrete point sources of nearly continuous contaminant release, worker mobility (for personal samples), and the use of local exhaust ventilation tend to maximize spatial concentration variability. On the other hand, factors influencing asbestos concentrations in a building environment tend to minimize the variability: a generally widespread source (if general ACM degradation and fallout is a reality), intermittent small point releases from work activities, and a general ventilation system which tends to mix the air throughout the building (or at least within same ventilation system) to produce a more homogeneous fiber concentration. Therefore, it is expected that spatial concentration variability within an individual building would be very low, certainly less than that in the occupational environment. The implication is that all samples collected within the area served by the same ventilation system to measure general building occupant asbestos exposures will be approximately equal. This means that relatively few samples are required to obtain an acceptable estimate of the fiber concentration and occupant exposures. Under these conditions, air sampling is a very powerful assessment tool.

The usefulness of air samples to predict future air concentrations is largely a function of the expected temporal variability. With sufficient knowledge of the temporal variability of airborne asbestos concentrations in a building, air sampling could be used as predictor of future concentrations. Indeed, EPA uses air quality data (GSD's of 2.0 to 2.5) to predict future concentration averages, maximums, ranges and frequency with which air quality standards will be exceeded. If temporal variability is moderately low (i.e., there is little change in concentration over time), then past measures are excellent predictors of future measures, barring a planned or unplanned catastrophic disturbance. Once again, airborne asbestos data from buildings have not been collected under an appropriate sampling regime which would answer this time series question. The only evidence available to answer this question again stems from indirect inference: the author has observed that airborne fiber concentrations appear to be more closely correlated with the state of building repair than the age of the building (one measure of temporal change). If as assumed by air sampling proponents, airborne fiber concentrations vary little with time, then buildings are well suited to the general industrial hygiene air sampling strategy; characterize the environment,

periodically resample to assure that unexpected changes have not occurred (frequency depends on rate of change), and sample after major changes such as roof leaks.

The "Peaks" Issue

The other major argument advanced against the use of air sampling in buildings is that it does not reflect the "peaks" or transient excursions in concentrations associated with maintenance or accidental release of fibers to the air. In this regard, it is essential to stress that the objective of the purpose of the air sampling discussed is to assess exposures of general building occupants. It is not the purpose of this type of air sampling to assess the occupational exposures of, for instance, maintenance persons working with ACM. A different type of air sampling would be appropriate for this group.

Examination of the "peaks" issue requires assessment of several component considerations. The first of these considerations is the rate of dilution or dispersion of fibers in the building air. Again, there are few data to directly answer this question. In one relevant published study, frequent intentional striking of ACM in a small room with no ventilation was found to raise the airborne fiber concentration only slightly (18). There are no published data, however, to quantify how rapidly a short term or point release of fibers disperses (in time or distance) to a concentration that is indistinguishable from the background concentration. The use of direct reading real time instruments to measure the dispersion of a gas in the atmosphere may be a suitable surrogate measure for fiber dispersion. Measurements with such instruments suggest that dispersion is very rapid. Dispersion and dilution are enhanced in buildings by the nature of most ventilation systems in which the air is continually mixed, at least within the confines of the individual ventilation system, to create a more-or-less homogeneous contaminant concentration. This admittedly limited evidence seems to indicate that, unless a person is immediately adjacent to a source of substantial fiber release, there will be no substantial excursion in exposure concentration, i.e., the general building occupant does not experience peaks in exposure.

A stronger argument against importance of peaks comes from a consideration of exposure and biological response to asbestos fibers. The argument has been advanced that exposure monitoring, and sample averaging time in particular, should be related to the biological response of the contaminant in question (19,20,21). For substances such as asbestos fibers, toxicologic modeling suggests that it is "unnecessary to investigate brief transient exposures to toxic agents with elimination half times of more than about two hours" (22). In fact, "[a]nalyses also indicate that day-to-day variation in exposure is unlikely to influence health risk for chemicals with elimination half times of more than forty hours" (22). In other words, even if occasional transient peaks are a reality in the general building environment, they do not have any biological relevance. Thus, the methods of air sample

collection currently utilized in building monitoring are entirely appropriate given the health risk models for asbestos exposure.

Conclusions

Analysis of the historical background relating to the question of the efficacy of air sampling as a decision-making tool in the assessment of buildings with ACM reveals that the proponents and opponents of this technique perceive different purposes and make different underlying assumptions about the nature of exposure. Opponents of air sampling for this purpose raise questions concerning the representativeness of air samples for definition of current and future exposures and the ability of air samples to detect transient "peak" excursions in exposure. Unfortunately, few data have been collected under a sampling regime which permits a definitive resolution of these questions. Analysis of available data and evaluation of various indirect sources of information suggest that airborne asbestos fiber concentrations in a given building are fairly homogeneous and that they change only slowly with time. As to the question of peaks, physical phenomena suggest that they don't even exist for the general building occupant, and secondly, evidence suggests that such short term exposures are biologically irrelevant, i.e., some longer term average is a more appropriate input to health risk models.

References

1. U.S. Environmental Protection Agency: 40 CFR Part 763 - Asbestos Hazard Emergency Response Act Regulations. (1987)

2. Department of Labor, Occupational Safety and Health Administration: 26 CFR 1926.58. (1986)

3. Sawyer, RN and CM Spooner: Asbestos-Containing Materials in School Buildings: A Guidance Document, Parts 1 and 2. U.S. Environmental Protection Agency Report #EPA 450/2-78-014. (1978)

4. Keyes, DL and BP Price: Guidance for Controlling Asbestos-Containing Materials in Buildings. U.S. Environmental Protection Agency Report #EPA 560/5-83-002. (1983)

5. Keyes, DL, BP Price and J Chesson: Guidance for Controlling Asbestos- Containing Materials in Buildings. U.S. Environmental Protection Agency Report #EPA 560/5-85-024. (1985)

6. Paull, JM, M Corn, PSJ Lees, PN Breysse: Non-Occupational Exposure to Asbestos in Buildings: A Practical Risk Management Program. Am Ind Hyg Assoc J 47(8):497-504. (1986)

7. Corn, M: Asbestos and Disease: An Industrial Hygienists Perspective. Am Ind Hyg Assoc J 47(9):515-523. (1986)

8. Gough, M: Uncle Sam Flunks Asbestos Control in Schools. Iss Sci Technol 4(3):81-85. (1988)

9. Sebastien, P, MA Billon-Galland, G Dufour and J Bignon: Measurement of Asbestos Air Pollution Inside Buildings Sprayed with Asbestos. U.S. Environmental Protection Agency Report #EPA 560/13-80-026. (1980)

10. Constant, PC Jr, FJ Bergman, GR Atkinson, DR Rose, DL Watts, EE Logue, TD Hartwell, BP Price and JS Ogden: Airborne Asbestos Levels in Schools. U.S. Environmental Protection Agency Report #560/5-83-003. (1983)

11. Schlecht, PC and SA Shulman: Performance of Asbestos Fiber Counting Laboratories in the NIOSH Proficiency Analytical Testing (PAT) Program. Am Ind Hyg Assoc J 47(5):259-269. (1986)

12. Lippmann, M: Asbestos Exposure Indices. Environ Research 46:86-106. (1988)

13. Snedecor, GW and WG Cochran: Statistical Methods, 6th ed. The Iowa State University Press, Ames, IA. (1967)

14. Leidel, NA, KA Busch and WE Crouse: Exposure Measurement, Action Level and Environmental Variability. NIOSH Technical Information, HEW Pub. No. (NIOSH) 76-131. (1975)

15. Ott, WR, DT Mage and VW Randecker: Testing the Validity of the Lognormal Probability Model: Computer Analysis of Carbon Monoxide Data from U.S. Cities. U.S. Environmental Protection Agency, Office of Monitoring and Technical Support Report #EPA-600/4-79-040. (1979)

16. Burdett, GJ and SAMT Jaffrey: Airborne Asbestos Concentrations in Buildings. Ann Occup Hyg 30(2):185-199. (1986)

17. Crump, KS and DB Farrar: Statistical Analysis of Data on Airborne Asbestos Levels Collected in an EPA Survey of Public Buildings. Reg Tox Pharm 10:51-62. (1989)

18. Burdett, GJ: The Measurement of Airborne Asbestos Releases from Damaged Amosite Insulation Subjected to Physical Attrition. In Proceedings of Workshop on Asbestos Fibre Measurements in Building Atmospheres, held at the Ontario Research Foundation, Mississauga, Ontario, Canada. (1985)

19. Rappaport, SM: Physiological Damping of Exposure Variability During Brief Periods. Ann Occup Hyg 32(1):21-33. (1988)

20. Roach, SA: A More Rational Basis for Air-Sampling Programs. Am Ind Hyg Assoc J 27(1):1-12. (1966)

21. Roach, SA: A Most Rational Basis for Air-Sampling Programmes. Ann Occup Hyg 20:55-84. (1977)

22. Rappaport, SM: Biological Considerations for Designing Sampling Strategies. Paper Presented at the ACGIH Symposium "Advances in Air Sampling" held at the Asilomar Conference Center, Pacific Grove, CA. (February 16-18, 1987)

23. Rappaport, SM, RC Spear and S Selvin: The Influence of Exposure Variability on Dose-Response Relationships. Ann Occup Hyg 32:529-537, Supplement 1. (1988)

Table 1
Possible Uses of Air Sampling in a Building with ACM

Use	Purpose
Building evaluation	decision making for public health policy
Building evaluation	decision making for action in building
Part of O&M program	OSHA compliance
Part of O&M program	program quality assurance
Part of O&M program	early detection of problem areas
After abatement	clearance/re-occupancy
During abatement	OSHA compliance
During abatement	quality assurance to detect fiber escape
Emergency response	OSHA compliance
Emergency response	decision making for action in building

Table 2
Summary of Arguments Used Against Air Sampling

1. measures of current exposure only/can not predict future exposure
2. many samples needed to detect peaks (or can't be detected)
3. PCM not appropriate/TEM expensive
4. surveys difficult in occupied buildings
5. OSHA standards not appropriate

AIRBORNE ASBESTOS FIBER LEVELS IN BUILDING AND THEIR IMPACT ON RISK MANAGEMENT

GARRY J. BURDETT, JAMES J. SMITH, AND CHRIS D. PAPANICOLOPOULOS

Microscopy Research Laboratory, Georgia Institute of Technology, Georgia Tech Research Institute, O'Keefe Building, 151 Sixth and Fowler St., Atlanta, Georgia 30332

Once the hazard has been identified and the limitations of the dose-response model delineated, the level of exposure becomes the primary focus of any risk assessment and the subsequent risk management decisions. However, regulatory agencies are placed somewhat in a dilemma by the use of a non-threshold linear dose- response model to extrapolate risk from the high levels of occupational exposure to the general environment. In practical terms, the general population gives a large multiplier (X 10^8) with potentially constant exposure, therefore a correspondingly very low exposure level must be present to give a minimal risk. The level required is usually well below the sensitivity of a single analysis. The problem is further compounded by the relative inaccuracy of the epidemiology, the non-uniform release of asbestos into the air and a reluctance to set a limit of acceptable safety for environmental carcinogens.

As the potential asbestos exposure of people in buildings has been accepted as being of sufficient concern to require regulatory action (e.g. Asbestos Hazard Emergency Response Act, Oct. 30, 1987), each course of action recommended (e.g. manage, remove, encapsulate etc.) should be evaluated using the same assessment procedure that was originally used to determine the regulatory action. Therefore risk management options should be based on their proven ability to reduce the environmental levels of asbestos.

Unfortunately due to the low airborne levels of asbestos found in buildings under normal occupation and the limit of sensitivity of the analytical methods, this approach has not often been pursued. Instead risk management has continued to rely on taking measures to reduce the potential for airborne release. Often this has been seen as a program of removal of all asbestos materials; however, this is expensive and may not be entirely achieved. Other

less expensive management options exist, but can they achieve the same level of safety for the building occupants as removal? What is the risk without any management procedures? - as is the case for the majority of buildings with asbestos containing materials?

To answer these questions a review of fiber concentrations from a variety of activities in buildings has been attempted. If the duration of the exposure is estimated, the risk assessor may then be able to make some qualified estimates of the efficacy of risk management options. Given the potential scale of the problem of asbestos in buildings and the limited information available, it must be a source of concern that in the U.S. detailed plans of the Operations and Maintenance programs for schools with asbestos containing materials should have already been completed. The authors are actively seeking to improve knowledge in this important area and consider it to be a primary activity impacting on the risk to maintenance/cleaning staff and potentially to all people in the building.

1. INTRODUCTION

The primary aim is to assess the risk to humans from the presence of asbestos containing material (ACM) in buildings. Risk assessment is a process of hazard identification, dose- response assessment and exposure assessment. The major hazards of asbestos have been well-documented; asbestosis, lung cancer and mesothelioma, and a great deal of research has gone into evaluating their dose-response relationships. The existing epidemiological studies and their updates have been reviewed and re-reviewed by various governmental committees: the US Environmental Protection Agency USEPA (1980), 1986a), the U.K. Health and Safety Commission (1979, 1985), the US Department of Labor USDOL (1983), the National Research Council NRC (1984) and the Royal Commission Ontario (1984). Although the accuracy and applicability of the non-threshold, dose-response models adopted by the above reports can be questioned, the only part of the risk assessment model that requires input, is the exposure assessment.

Ideally, the exposure assessment should be carried out using the same standardized methods and units as were used to derive the dose-response relationship. Unfortunately this is not the case with asbestos, and like all particle characterization, the result obtained is highly dependent on sampling conditions and the methods of sampling and analysis used. One further complication is that the biological mechanisms of asbestos related cancer are still not understood and the properties of asbestos associated with the disease have yet to be unambiguously defined. This has led to some disagreement as to what portion of the

asbestos material should be measured (this is usually dependent on the analytical method). It is as well to remember, that because the mechanism is still not adequately described, any exposure assessment is merely an "index of exposure" and it is the degree of correlation between the index and the disease that is really at issue.

The use of non-threshold linear models for estimating asbestos risk has a profound effect on regulatory bodies involved in environmental risk management. It firstly means that their is "no safe level of exposure" which can be recommended and therefore there is a reluctance to set an environmental limit. Without a limit a whole range of environmental requirements are used, e.g. "no visible emissions (USEPA, 1984)," "the airborne asbestos concentration inside the abatement area is no higher than concentrations at locations outside the abatement area and," "below the quantifiable limit of detection "(USEPA, 1987)". This lack of determination to assess and control the environmental impact of asbestos arises partly from the exposed population being very large (e.g. 10^8)- therefore even a low level of exposure may multiply up as a significant number of deaths; and partly from an unwillingness for the state to be seen placing a value on an acceptable risk to human life. The lack of an environmental standard means that the risk assessment attempted in this paper cannot be seen a substitute for risk management, where other monetary, convenience and public relation factors are taken into account.

The exposure assessment will be directed to help answer the following questions. How widespread is the use of asbestos in buildings? Which types of ACM are mostly likely to give releases? What levels of disturbance are necessary to generate airborne asbestos emissions? What are the measured levels in buildings? What is the effect of O&M programs on asbestos levels in building, and what is the effect of removal on asbestos in buildings? The measured levels will then be compared with published estimates of risk and cost.

2. Units of Measurement

A major problem with attempting to relate exposure to risk is the unit of measurement and whether it is suitable for interpreting low asbestos exposure. Recent risk assessments of non-occupational exposure have relied on extrapolation from a relatively small number of occupational studies with a unit of measurement of fibers/cubic centimeter (f/cc) x estimated duration of exposure (in years or lifetime). The f/cc originates from optical phase contrast microscopy (PCM) counts at 400x to 500x magnification of airborne particles sampled onto a mixed esters of cellulose filter. A particle which was visible and met the size criteria of >5 μm long, <3 μm wide and with an aspect ratio >3:1 being denoted as a fiber. These fibers are often termed "PCM equivalent" or regulatory" fibers and are assumed to be asbestos for the purpose of controlling occupational exposure and

disease. This definition has been used for 30 years (Walton, 1982), but due to many improvements in counting methodologies and equipment it cannot be regarded as a stable index of exposure and variations of up to an order of magnitude may be present between different laboratories at different times.

The index was originally defined to regulate the fibrogenic effects of asbestos "asbestosis" and its relationship to the incidence of lung cancer and mesothelioma is arbitrary. However, data from animal experiments (e.g. Stanton et al., 1977; Pott, 1978 and Davis et al., 1986) suggests it also has relevance to the carcinogenic activity. It should also be noted that the occupational risk estimates were based on earlier data of all airborne particles, measured by an impinger-microscope method, for which no reliable conversion exists. Hopefully in the next few years ongoing epidemiological surveys will produce the first well-documented and reliable dose-response relationships from occupational exposures. For low exposure and non-occupational risk assessments the median result of the more acceptable occupational studies is linearly extrapolated for lung cancer estimates while mesothelioma is extrapolated on the basis of concentration and the time since first exposure with time raised to a power of 3 - 5 (USEPA, 1986a). This implies environmental measurements must be made using the same "regulatory" fibers or a further unknown is introduced. Until recently many of the non-occupational measurements and the early risk estimates (e.g. USEPA, 1980; NRC, 1984; USDOL, 1983) were made on mass concentration measurements of all airborne asbestos material. A conversion factor of approximately 30 $\mu g/m^3$ as equivalent to 1 f/cc was used, although the size distributions will vary between different types of asbestos and their source.

PCM fiber counting alone cannot be used for non-occupational assessment as it cannot discriminate between asbestos and non- asbestos fibers. TEM is the recommended method for analysis as it can measure the full size range of the asbestos fibers present and give an unambiguous identification based on their crystal structure and chemistry. TEM results are normally expressed as asbestos fibers >5 μm long and all asbestos fibers/structures. The unit of >5 μm long fibers is not the same as "regulatory" fibers; as fibers below about 0.25 μm width will not be visible by PCM. At the present time all TEM fibers >5 μm long are commonly reported and the "regulatory fiber" count must be calculated from the width dimensions recorded. SEM is also used for asbestos fiber counting (VDI, 1985) but is limited in its ability to identify asbestos and cannot be routinely used to count the smaller asbestos fibers . However, SEM can give measurements of >5 μm long fibers with a tentative separation into the asbestos and non-asbestos components (Breysse et al., 1989).

For simplicity in this report all measurements by PCM, or SEM and TEM fibers >5 μm long, and 0.25 to 3 μm wide with aspect ratios >3:1 are referred to as "regulatory fibers" and expressed in units of f/cc. SEM and TEM measurements of asbestos fibers >5 μm long are described as such and are given in units of f/cc. TEM measurements of all

sizes of asbestos fibers and/or structures are expressed in units of f/l. This latter unit is still not completely defined, as some methodologies (e.g. USEPA, 1987) do not count fibers <0.5 μm long; also there is no widespread agreement about the evaluation of clusters, bundles and matrix structures.

Two other units of measurement can be used to avoid the need to express all measurements in terms of "regulatory" fibers. If measurements inside a building can be shown to be statistically no higher than measurements outside the building, the risk is reduced to the general background level. This method has been adopted by the USEPA for TEM measurements. An alternative method is to report results as below the limit of quantification (LOQ) of the analytical method. In fiber counting methods the LOQ represents a practical limit for the time spent on the analysis of an individual sample, as only 0.2% or less of the exposed filter area is usually examined in a PCM or a TEM analysis.

In the foreseeable future, comparisons in terms of "regulatory" fibers will continue to be used as the main index of risk and the measurements collected should be capable of being expressed in this unit while also allowing for a description of the total respirable asbestos fiber content.

3. Occurrence of ACM in Buildings

Various guides for carrying out inspections have been produced by Government Agencies (e.g.USEPA, 1985a and DOE, 1986) and in a large building an inspection can represent a substantial and costly undertaking. At the present time, only the U.S. has mandated that buildings should be inspected and this was restricted to schools (USEPA, 1987) although a further extension to public buildings is under consideration (USEPA, 1988a). Many school systems, public and private building owners have carried out inspections on varying scales to respond to employee or liability concerns, but few of these are widely available.

Attempts to relate building survey information directly to the airborne exposure levels inside the building have not produced any meaningful correlation for mass (USEPA, 1983) or fiber concentration (USEPA, 1988b) measurements. However, ranking systems based on friability of the material, the extent of its use, accessibility and condition (e.g signs of previous damage) are almost always applied for risk management purposes. In general only friable asbestos surface coatings which have been damaged and are likely to be further damaged are recommended for immediate removal action. Other categories are included in the Operations and Maintenance (O&M) program and may not be removed until the economic lifetime of the building has been reached.

The impact of this advise can be appreciated when looking at the USEPA surveys for friable ACM in public buildings and schools . The results for public buildings have recently been re-examined (USEPA, 1988a) and the findings are summarized in Tables 1 and 2. The figures projected for 3.6 million public buildings was based on a sample of 231 buildings in 5 cities and excluded schools, military, state and local government buildings and residences fewer than 10 units. Some 20% (733,000) public buildings were expected to contain friable ACM, compared to 35% (35,000) school buildings. A recent study by the Philadelphia Dept. of Health (1988) of 839 city owned or occupied buildings found that some 47% contained friable ACM. This result if typical suggests that about one third of public buildings as a whole contain some friable ACM. The figure increases to about one half when other ACM is considered (e.g. floor tile). It is not known whether these figures are typical of buildings in other cities and countries.

4. Release of Asbestos Fibers From ACM

No risk to human health can be inferred just from the presence of ACM in a building. For a risk to be present the asbestos must become separated from the matrix of the ACM and made airborne. Only asbestos material which is of a respirable size can be considered a risk. The ability of ACM to release respirable asbestos and the mechanisms involved have not been widely researched, but the importance of this factor has been acknowledged as most building surveys attempt to classify the type, area, condition and the percentage of asbestos. The data that does exist is based either on PCM fiber counts of occupational type releases (HSE, 1985), or on a reproducible method of mechanical simulation to give a "releasability index". One EPA survey (Falgout, 1985) correlated PCM and TEM measurements of asbestos structures released per gram of material disrupted (by sawing, grinding or drilling) in a small glove box facility. An important conclusion of the study was that <1% of the chrysotile fibers released corresponded to the "regulatory" fiber definition and only a millboard sample gave any PCM fiber counts above the quantification limit. The relative release obtained with the different products and release methods are reproduced in Table 3 and the type of asbestos structures present are summarized in Table 4. Other workers (Jaffery, 1988) have found measurable PCM and TEM (>5 μm long) fiber levels when various abrasion methods (chiselling, sawing, sanding etc.) were applied to chrysotile, amosite and crocidolite containing ACM's. Values of a few fibers/cc by both PCM and TEM were common for short duration activities.

Due to the great variety of ACM's and possible disturbance activities, it is not realistic to conduct widescale laboratory assessments of releasability. However, the results available show that the fibers released from non-friable ACM's are related to the intensity

of the abrasion, e.g. minor surface disturbance < drilling < sawing < sanding or grinding and the area or volume disturbed. The use of power tools for these activities greatly increased the rate of fiber emission - often to the point where over-sampling is difficult to avoid, even for short term samples of 10 minutes.

The above results were obtained using non-friable ACM's with asbestos content in the 10-50% range. As many regulatory agencies define ACM, as material which contain >1% asbestos by weight, it is interesting to consider what fiber emissions are possible from low asbestos content materials. Recent experiments (Burdett, and Scott, 1988) carried out under laboratory conditions in a 9 m^3 static dust chamber (no significant air exchange during sampling period) with decorative plaster containing approximately 3% chrysotile asbestos, gave average PCM levels of 0.13 f/cc and average TEM levels of >5 μm long fibers of 0.77 f/cc, during 30 minutes of vigerous scraping (0.77 f/cc by TEM) and 0.3 and 1.8 f/cc by PCM and TEM respectively for 10 minutes of hand sanding of the surface. These results confirmed previous measurements in a domestic house when a similar ACM was scraped from a wall. Therefore even with low asbestos content materials, fiber emissions close to the permitted occupational levels may be generated during periods of substantial maintenance or renovation activity. This has also found to be the case with encapsulated asbestos materials such as vinyl floor tile.

5. Measurements in Building Under Normal Occupational

Early TEM measurements of asbestos levels in buildings and outside air used indirect sample preparation methods which tended to de-aggregate the fiber bundles, clusters and matrices. This meant that the fiber counts obtained did not have any relationship with the airborne situation except in terms of the asbestos fiber mass concentration. Mass concentration are more time consuming to make, as each fiber must be sized and the individual fiber mass calculated. As a single large fiber may contain a mass equal to 10^6 smaller fibers, asbestos mass analysis has poor precision. There is also the possibility of including non-respirable asbestos, which makes mass results difficult to interpret for the purpose of risk assessment.

Asbestos mass concentration results in outside air and in buildings containing mainly friable or cementitious sprayed asbestos were reviewed and used as the basis of the US National Research Council's estimate of Non-Occupational Health Risks (NRC, 1984). The results are reproduced in Table 5, and summarized measurements from France and the USA and showed evidence of elevated asbestos levels inside buildings with friable asbestos. One particular survey (USEPA, 1983) which monitored the air in 25 schools at 48 asbestos-containing sites over five days of normal usage gave statistically significant differences between the average levels at asbestos containing sites (179 ng/m^3), indoor

controls (53 ng/m^3) and outdoor controls (6 ng/^{3}). However, further analysis has shown that some of the material measured was derived from non-respirable paint flakes and clusters not included in the original analysis; also some deliberate contamination of the unsupervised samples was attempted by the students (Lee, 1987). Evidence for elevated levels in U.S. schools was also found in another study (Chadwick et al., 1985), but the method used is difficult to quantify in terms of mass. TEM measurements of ACM in public buildings have also been reported by Bruckman (1975). Two TEM studies of UK buildings (Leguen and Burdett, 1981; Burdett and Jaffery, 1986) with friable and non-friable ACM found no evidence for elevated levels in buildings except where the asbestos had been damaged.

TEM analysis based on direct preparation methods has and gained some acceptance (e.g. HSE, 1985; Royal Commission Ontario, 1984 and USEPA, 1986) as the most useful way of gathering data for non-occupational risk estimates. The studies published to date using TEM analysis and direct preparation methods have reported low levels of >5 μm long TEM fibers for buildings containing friable (Royal Commission Ontario, 1984; Burdett and Jaffery; 1986; Lee, 1987; USEPA, 1988b) and non-friable ACM (Burdett and Jaffery, 1986; Gazzsard Crockford, 1987; CPSC, 1989) (Table 6). This data base will continue to grow as more measurements are carried out in buildings as assurance samples for O&M programs. SEM measurements of >5 μm long fibers are also being carried out in several countries and this data also shows that low fiber concentrations are generally present (Altree-Williams and Preston, 1985) except when asbestos is being damaged (Marfels et al., 1985). TEM results reported for all asbestos fibers and/or structures have also given low levels in buildings during normal occupation (USEPA, 1988b; Burdett and Jaffery, 1986; CPCS, 1989).

6. Other Measures of Building Exposures During Normal Occupancy

The measurement of airborne asbestos levels during normal occupancy is often criticized for its inability to be related to past or future exposures and their risk. Also samples may not be taken in the area where the release occurs. This approach effectively demands the monitoring of all areas at all times. This is not practical with conventional air filtration sampling methods - a passive sampler is required.

Horizontal surfaces act as passive samplers and settled dust has long been used as an indicator of previous release. Surfaces which are protected from direct fall-out accumulation will provide the best estimate of past airborne levels. Other rarely cleaned inaccessible sites (e.g. behind cabinets and furniture) may also be useful for estimating the period of time the deposit represents (time since the furniture arrived?). The dust retained in carpets is also widely used as a measure of previous asbestos contamination.

Sampling the settled dust is a problem. A number of methods have been described based on either the adhesive capability of the sampling matrix or relying on disturbance of the surface to facilitate the transfer. Table 7 summarize some of the methods which have been applied in buildings with ACM's. The transfer efficiencies will vary with the type of surface sampled and with technique. In most cases, only relative efficiencies between methods are compared and early work in the nuclear industry (Royster and Fish, 1987) remains the only published quantitative work. Results from settled dust deposits are also difficult to interpret as the time of collection may not be known and can usually only provide a relative measure between different areas in a building. These problems may be overcome if media suitable for direct TEM analysis is placed in the building.

With a known time of exposure and a result in terms of asbestos fibers/m^2, simple assumptions can then be made to estimate the likely inhaled exposure to a person in the room. This approach is necessary to evaluate high figures produced from settled dust analyses, where a level of 10^5 fibers/m^2 of surface area is considered to be a background level (Wilmoth et al., 1988).

An alternative approach is to resuspend dust. Personal air samples collected on maintenance personnel sweeping or vacuuming floors give a good estimate of the worst case routine exposure from ACM's in a building. Aggressive sampling techniques as applied after an abatement exercise, are not recommended if there is any possibility of large scale release from friable surface, but can be used to good effect to resuspend settled dust under controlled conditions (e.g. a temporary enclosure is constructed). Aggressive sampling in post-abatement (wet removal) areas evaluated by the USEPA (Karaffa et al, 1987) have given increases of 3 to 6 times. In lightly contaminated areas in the U.K. (after incomplete dry removal) increases of 10 to 100 times were reported (Burdett et al., 1988).

7. Releases from Asbestos Removal

Asbestos removal operations can involve the generation of high levels of airborne fibers. Levels of regulatory fibers of 100 f/cc can be generated during dry removal of friable ACM's (HSE, 1985; Burdett et al, 1988) and represent a risk both to the workers and the building occupants. Workers rely on respiratory protection and decontamination techniques for protection, while the building occupants rely on the performance of the enclosure and the work practices of the contractors. All of these areas have potentially serious flaws and the process of asbestos removal must be conducted a way to give the minimum potential for release of airborne fibers both during and after the removal.

Airborne levels can be greatly reduced by the use of wetting agents on the ACM and/or the use of HEPA vacuum equipment to remove the ACM as directly as possible from the surface (USEPA, 1979). The enclosure must be viewed as a secondary

containment and negative pressure HEPA filtration will help reduce both the airborne concentration inside the enclosure, while preventing escapes outside the enclosure.

Currently regulatory efforts are focused at the end of the removal operation, to decide whether it is safe to remove the enclosure. No evidence for the efficacy of the removal, in terms of monitoring inside and outside the enclosure during removal is required. If an escape event takes place - which cause asbestos fibers to become airborne outside the enclosure this will remain unrecorded and leave a background pollution in the rest of the building. This oversight becomes more serious when regulations permit comparisons of levels inside and outside the enclosure to demonstrate the enclosure is no more polluted than the rest of the building, instead of using the outside air as the reference (USEPA, 1987). Even then, areas downwind of the asbestos disposal containers or negative pressure filtration outlets, may show indications of higher airborne releases (Burdett et al, 1988).

A number of studies of asbestos removal have been carried out and each study has to be judged on its relative results as the conditions, sampling procedures and measurements vary considerably. Again only TEM measurements are considered but it should be remembered that many thousands of asbestos removal operations are passing a 0.01 f/cc clearance by PCM measurements every year.

TEM mass measurements of removal operations have been reported by several authors. Sebastian et. al. (1980) found increased levels in two buildings where dry removal followed by wet cleaning was used, but a reduction in a building where enclosure (plaster on a metal lathe) or an encapsulant were used. In a USEPA study of four schools (USEPA, 1985), levels before and after the removal were not significantly different (<6 ng/m^3) but escapes from the containment gave levels as high as 140 ng/m^3 outside the enclosures during removal. Study in four US schools (by Bozzelli and Russell 1982) found a significant reduction one week after removal (56-90%) compared to pre-removal levels of 5-40 ng/m^3. However, the presence of large clumps in the before samples suggest samples may have been interfered with.

TEM measurements of >5 μm long asbestos fibers - before, during and after removal at several sites have been reported in a U.K. study (Burdett et al., 1988). Typical results are summarized in figs. 1-4 and show that increased concentrations of asbestos were present for a considerable period after the removal. These results are similar to trends reported in a study of 8 Canadian removal sites (Pinchin, 1982). TEM measurements of all asbestos fibers/structures have reported increased levels after removal at sites in the UK (Burdett et al., 1988), Sweden (Walvsewski and Paulsson, 1988) and the USA (USEPA, 1988c and 1988d). The increases above background (e.g. X385) found in research measurements in the USEPA (1988b) study does not match the experiences of routine measurements.

Recent work in the U.S. (Powers, 1988) monitored post-abatement clearance levels at removal sites in 79 New Jersey schools. Of the 598 samples analyzed by TEM for all asbestos fibers, 573 samples gave < 10 f/l and 234 samples were below the detection limit. Although not based exactly on the AHERA - EPA measurement procedure (USEPA, 1987) the use of aggressive sampling suggests that many of the sites would have passed AHERA clearance. These latter results represent a dramatic increase in the efficiency of removal and post-abatement clean-up and the corresponding advances in removal technology and practice compared with a decade ago (Nicholson et al, 1979).

The differences in the magnitude of releases between studies can partly be explained by that all the U.S. studies were carried out with the amended water method, which produces lower airborne concentrations in the enclosure - while studies in other countries monitored used dry removal in the enclosures. However, at present all detailed studies with measurements before, during and after removal have reported escapes from the enclosures. This undoubtedly contributed to the subsequent pollution levels in the buildings. The test for effective removal from buildings is aggressive sampling with TEM analysis, after the abatement contractor has left the site, but before the building/area is re-occupied.

The risk to workers must continue to be emphasized as the performance of respirators in less than ideal conditions is questionable (Ortiz et al., 1988) and the fit of the respirator can drastically reduce the protection factor.

8. Levels During Operations and Maintenance (O&M) Activities

The release of asbestos fibers from maintenance activity, while the least well-researched category of measurement, is the most important for risk assessment and risk management procedures. Two extreme scenarios exist - a contractor comes on site and unknowingly starts extensive maintenance on friable ACM surfaces - or a controlled minor removal, enclosure or encapsulation is carried out on the ACM prior to the maintenance work. For risk assessment purposes both must be evaluated, but with successful risk management, only the latter should occur.

Maintenance activities can in the worst case give levels approaching dry removal concentrations (100 f/cc) and isolated incidents must occur in buildings where no survey of ACM has been carried out. Maintenance releases have previously been regarded as occupational exposures and most of the published information is based on PCM counts of "regulatory fibers". Early experiences reported by Sawyer and Spooner (USEPA, 1979) regularly found airborne levels of several f/cc for maintenance and custodial activities in buildings with damaged friable sprayed ACM's. Paik et. al. (1983) in a review of removal and maintenance activities with sprayed ACM found geometric mean concentrations of 0.18

f/cc for sheet metal workers, 0.13 f/cc for carpenters and electricians and 0.03 f/cc for painters - thus reflecting their closeness and contact with the sprayed ACM. A small scale maintenance activity involving removal of domestic water tank insulation monitored by both TEM and SEM (Breysse et al., 1989) found mean levels of regulatory sized fibers of 0.12 f/cc, in the loft space where the work was carried out, with levels in an adjacent bedroom remaining below the quantification limit. Simulated maintenance activities monitored by TEM in a building with sprayed amosite (fig. 2) gave small but significant releases in the near vicinity (Burdett,et al., 1988).

The efficiency of O&M procedures designed to reduce asbestos exposures in building has had little study. Early work by Johnson and Clapp (1980) to test the performance of HEPA vacuum cleaners showed that measurable emissions were present in the exhaust air. A recent study by Karrafa et al., (1987) of the performance of HEPA and conventional vacuuming of carpets in two buildings containing sprayed ACM found TEM asbestos levels below the detection limit. Subsequent analysis of the carpet showed a low level of asbestos dust contamination. However, substantial increases in the levels of PCM fibers (non-asbestos) were produced by conventional vacuum cleaning.

Olcrest (1988) has recently studied the performance of HEPA vacuuming and steam cleaning on nylon carpet using neutron activated chrysotile asbestos. Removal efficiencies of 51% and 46% were obtained for HEPA vacuum and steam cleaning respectively, and a combined efficiency of 74% when both techniques were used. In studies of other surface types a wet sponge often gave the most effective removal, unless the surface was water absorbent. Flat, sealed surfaces present little problem to clean.

The use of surface cleaning as an O&M procedure to reduce airborne levels due to re-entrainment has a long history of use and is an integral part of any abatement exercise, especially when enclosures have been constructed. After any asbestos release the visible loose material should be efficiently removed from the surface (e.g. wet wiping). However, little attention has been given to the effect of regular cleaning of building surfaces as part of an O&M program. The general levels in buildings suggest reintrainment of settled dust is not a problem during normal activity but sweeping, dusting etc., have the same potential to give increased level as aggressive sampling. Therefore the use of aggressive sampling to measure maximum levels inside buildings has a logical basis, but can cause severe problems about whether the building can be re-occupied if a low result is not achieved. A recent study involving aggressive sampling in an office building found increased levels (Marshall, 1988) by TEM (up to 30 structures/l) which resulted in several floors being evacuated. Cleaning of surface to achieve a level below 10 structures/l during aggressive sampling was used for the arbitrary criteria for re-occupation. This type of procedure is not recommended by regulatory agencies at present, but may have to be reconsidered in terms of an effective O&M program.

The extensive use of vinyl tile in buildings and the constant wear to which it is subject make it a possible source of emission during maintenance and custodial duties. Sawyer (USEPA, 1979) reported levels of 1 f/cc during surface sanding. Increased TEM airborne levels under normal conditions have been reported in France (by Sebastien et al., 1982 and Dufour, 1985), but as these were mass measurements, it is not clear whether the asbestos remained bonded in the vinyl or was of a respirable size. No TEM data is published, from which to assess whether vinyl tile presents a non-occupational risk in normal building use. A number of methods (e.g. amended water, dry ice and heat) have been used to remove vinyl tiles during renovation with minimal emission of asbestos.

The use of encapsulants has been studied by the USEPA (1986) who found significant releases during application (up to 140 ng/m^3) but obtained low levels after application. Similarly enclosures may give rise to asbestos releases during installation. Failure to control and contain releases during encapsulation and enclosure give rise to measurable levels of asbestos pollution in the building after the work has been carried out.

At the present time their is a great need for more occupational and environmental measurements of maintenance and routine cleaning activities in buildings with ACM's.

9. Risk

The exposure assessments in the preceding section are far from complete but the main points to summarize are:

a) U.S. data suggests that about half of schools and public buildings contain some ACM. The majority of the material will be floor tile or limited to thermal insulation in boiler rooms.

b) Laboratory studies of the release of airborne asbestos fibers from ACM show that the type or abrasion of cutting and the surface area or volume involved, are important determinants of the number of fibers released. Only a small percentage of the fibers meet the "regulatory fiber" definition.

c) TEM measurements in buildings using direct-preparation methods have consistently given very low levels of airborne asbestos fibers >5 μm long during normal occupancy (<0.001 f/cc). The exception is where damage to friable ACM is visible and the room surfaces are being disturbed. Schools appear to have the highest incidence of these conditions, and up to 10% of public and commercial buildings have damaged ACM. TEM mass measurements have shown the same trend with

increased levels reported in schools with friable and damaged sprayed asbestos coatings.

d) Poorly executed or unplanned maintenance activities on or near ACM's in buildings, may release concentrations of fibers of up to a few f/cc.

e) Post-abatement, clean-up inside the enclosures can successfully achieve TEM clearance inside the enclosure at a 10 f/l level with aggressive sampling.

f) In depth studies before, during and after removal in a building have frequently shown evidence for increased asbestos levels in the building environment after removal is completed.

g) Airborne asbestos levels inside the enclosures during removal should be kept as low as practical with the available technology.

The exposure assessments can be readily translated into a risk assessment by allotting a duration of exposure and applying any one of the dose-response relationships determined in the references beginning this paper. Recently Doll (1988) estimated that with building exposures not exceeding an average of 0.0005 f/ml, the lifetime risk for 10 years exposure in school or 20 years exposure in adult life, is about 1 in 100,000. This level of risk is around the mean of previous risk estimates for non- occupational exposures in buildings. For an individual, 1 in 100,000 represents a low to minimal risk. For regulatory agencies this corresponds to 10-100 deaths per year, which again is relatively low compared to other sources. However, only relative risk estimates/exposure assessments are necessary to answer the questions of what course of action is necessary to reduce risk?

Results from TEM mass measurements and converted to f/cc in the National Research Council, 1984 study gave outside background levels of 0.00007 f/cc with urban levels up to 10 times higher. Measurements inside buildings/rooms without asbestos gave median levels equivalent to 0.00054 f/cc and when ACM was present levels of 0.0006 f/cc. These figures were weighted to produce an environmental exposure of 0.0004 f/cc..

A recent study by the author at busy urban road junction, found an average level >5 μm long asbestos fibers by TEM of 0.0005 f/cc. Similar TEM measurement using a direct preparation method have established a background level of $\leq$ 0.0005 f/ml in buildings with ACM. This level is only slightly increased when friable ACM is present and in the majority of buildings is not statistically different from the measured outside background. Therefore at the present time there appears to be no published body of

evidence using the direct TEM preparation methodology to support a widespread asbestos removal program to reduce the risk from background exposure levels in buildings.

This evidence does not mean that no action is required but merely gives time for regulatory agencies to fully assess the risks involved from living with ACM's. This is best evaluated in terms of the most likely response actions; monitor the situation, develop management plan, implement an O&M program or remove, encapsulate or enclose the ACM. In several countries "do nothing" is more accurately termed as "deferred action" as there are regulations requiring asbestos removal before demolition.

The assumption that no release occurs, is not realistic and a management plan or an O&M program is necessary to reduce the risk of release (USEPA, 1986c, USEPA, 1989). To help evaluate whether this is likely to be effective, the exposure from a single release event can be analyzed. An example used by Burdett (1986) calculated a child's, exposure if their entire schooling was spent in a building with ACM, at an average exposure level of 0.0005 f/cc. A cumulative exposure of 7.5 fiber hours /cc was obtained, which represents a one hour exposure at 7.5 f/cc or 10 hours exposure at 0.75 f/cc - both levels of exposure are possible if unsupervised or extensive maintenance/removal activities are carried out while the building is still occupied. Therefore a single major maintenance activity has a potential to double the lifetime risk. This is a compelling argument for the identification and management of ACM's in schools and public buildings. The implementation of an O&M program and/or asbestos removal to reduce levels would appear to be prudent where damage has occurred and their is a high probability of further damage or disturbance.

Studies of removal and encapsulation of friable ACM have shown that environmental releases are likely to occur and levels inside the building may remain raised during the following refurbishment and re-occupation of the building. Therefore an increase in lifetime risk equivalent to several maintenance exposures may be produced if the building remains occupied during removal. The possibility of a much higher level of exposure due to incompetence or accident cannot be overlooked. A poorly conducted removal or maintenance activity in a school will have a much greater impact on the risk of mesothelioma, due to a 3rd to 4th power dependence on the age since first exposure.

The occurrence of poor removal activities is hard to estimate, as representatives of regulatory agencies have a general reluctance to prosecute and a flexible approach to improve matters is adopted. In the U.K. only a small number of some 2,000 licenced contractors have had their licenses revoked or cases brought to court. In the U.S. the latest figures available (USEPA, 1988a) for asbestos abatement work showed that the EPA found 2,179 violations in 15,000 inspections under the NESHAP regulations (14%), while OSHA recorded 856 citations in 64,000 inspections under workplace or construction regulations (1.3%). These figures do not reflect the day to day experience of Hygienists

and the USEPA is currently undergoing a major revision of its enforcement strategy. However, even a conservative estimate of 15% violations represents a significant risk due to poor removal performance. As measurements are rarely taken outside the enclosures during removal - no published knowledge of the performance of removal activities exist, other than these reviewed here. The increased use of technology to reduce airborne levels inside the enclosure during removal would appear to be essential.

At present, there appears to be little to choose in terms of reducing non-occupational risk, between management, O&M programs and removal. The fourth option of a deferred action is the most frequent response at present and provided no damage is present and maintenance is not required, it involves little risk to the building occupants. However at some point before demolition, removal will be necessary and the timing of removal, costs and risk, must all be balanced by the risk manager. The cost of the above responses has been calculated by the USEPA (1988a) for the estimated 3.6 million U.S. buildings with friable ACM. These were:

Control Program	Cost (billion $)
No Action	0
Increased Information	0.074
Inspection for ACM	2.0
Inspection and Management Plan + TEM Monitoring of Airborne Asbestos	3.0
Levels in Building (one time only)	10.0
Mandatory Inspection and O&M Program	31.0
AHERA Regulation in Schools Applied to all Public Buildings	51.0

It should remembered the study for which these costs were produced has most, likely under estimated the number of buildings with friable ACM. A reduction in environmental risk is not guaranteed by any of the actions, and if the potential exposure of removal and O&M workers is taken into consideration, the net effect is at best likely to be a high cost per life saved.

REFERENCES

Altree-Williams, S. and Preston, J.S., (1985). Asbestos and other fiber levels in buildings"., Ann. Occup. Hyg., 29, 357- 362.

Bozzelli, J.W. and Russel, J.F. (1982). "Airborne asbestos levels in several school before and after bulk asbestos removal." Intern. J. Environmental Studies. 20, 27-30.

Breysse, P.N., Cherrie, J.C., Addison, J.A. and Dodgson, J. (1989) "Evaluation of airborne asbestos concentrations using TEM and SEM during residential water tank removal" Ann. Occup. Hyg. (in press).

Bruckman, L., and Rubino, R.A. (1975). "Rational Behind a proposed asbestos air quality standard"., J. Air. Poll. Control Assoc., 25, 1207-1212.

Burdett, G.J. (1986). "Measured asbestos concentrations in buildings" in: Proc. of a NSCA Workshop on Asbestos Policies for the Future Pt. II. held at Newcastle, 26-27th March 1986. National Society for Clean Air, Brighton, England.

Burdett, G.J. and Jaffery, S.A.M.T. (1986). "Asbestos concentrations in public buildings"., Ann Occup. Hyg., 30, 2, 185-199.

Burdett, G.J., and Scott, R. (1988) "Fiber release from chrysotile containing decorative plasters". OMHL Internal Report IR/L/DI/8803, U.K. Health and Safety Executive, Cricklewood, London

Burdett, G.J., Jaffery, S.A.M.T. and Rood, A.P., (1988). "Airborne Fiber levels in buildings. A summary of UK measurements" in WHO/IARC Symposium on Mineral Fibers in the Non- Occupational Environment. Lyon, France, 8-10th September 1987. (in Press).

Chadwick, D.A., Buchan, R.M., and Bealieu, H.J., (1985). "Airborne Asbestos in Colorado Public Schools" Enviro. Res., 36, 1-13.

CPSC, (1989). Consumer Product Safety Commission. (in press)

Davis. J.M.G., Addison, J., Bolton, R.E., Donaldson, K., Jones, A.D. and Smith, T., (1986)." The pathogenicity of long versus short fiber samples of amosite asbestos administered to rats by inhalation and intraperitoneal injection. Br. J. Exp. Path., 67, 415-430.

DOE (1986). U.K. Department of the Environment, "Asbestos Materials in Buildings". HMSO. London, England

Doll, R. (1988). "Summary report on mineral fibers in the non-occupational environment": in WHO/IARC Symposium on Mineral Fibers in the Non-Occupational Environment. Lyon, France, 8- 10th September 1987. (in Press).

Dufour, G., (1985). "Indoor asbestos pollution from floor tiles". In Proc. Workshop on the Monitoring and Evaluation of Airborne Asbestos Levels following an Abatement Program held at the National Bureau of Standards, Gaithersburg, Maryland, 12-13 March, 1984. U.S. EPA/NBS Research Triangle Park, N. Carolina, U.S.A.

Falgout, D.A., (1985). Environmental Release of Asbestos from Commercial Product Shaping. US Environmental Protection Agency. EPA/000/52-85/044 Aug. 1985.

Gazzi, D. and Crockford, G.W. (1987). "Indoor asbestos levels on a housing estate (Determined by TEM)," Ann. Occup. Hyg., 31, 4A, 429-439.

Guth, J.H., (1988), "A guide to surface sampling methodology." Paper presented at the Surface Dust Sampling Session of the National Asbestos Council Conference, Boston, MA. September 19- 21. 1988.

HSC (1979), Health and Safety Commission, Asbestos: Final Report of the Advisory Committee on Asbestos, Vols 1 and 2. HMSO, London, U.K.

HSC (1985). Health and Safety Executive, Asbestos: "Effects on Health of Exposure to Asbestos". Health and Safety Commission, HMSO. London. Guidance note EH35.HMSO. London.

Jaffery, S.A.M.T., (1988). "Releasibility of Asbestos Fibers", OMHL Internal Report (U.K. Health and Safety Executive), IR/L/DI/88/12. Cricklewood, London.

Johnston, W.L. and D.E. Clapp, (1980). "An Evaluation of Vacuum Equipment for Collection of Asbestos Waste", NIOSH Technical Report, DHHS No. 80-137, Cincinnati, Ohio. September 1980.

Karaffa, M.A., Amick, R.S., Crane, A.C. and Powers, T.J. (1986). "Methods of Evaluating Asbestos Abatement Technology" US Environmental Protection Agency. EPA/600/S2-86/053.

Karaffa, Mark A., C.E. Miller, R.A. Amick, J. Chesson, and C. Jackson, (1987). "Preliminary evaluation of potential asbestos exposure during carpet vacuuming in buildings containing friable asbestos material", U.S. Environmental Protection Agency, Unpublished Draft Report, Cincinnati, Ohio. November 1987.

Lee, R.J., (1987). "The Constant study; revisited: A comparison of the airborne asbestos fiber concentrations in schools as determined by direct and indirect sample preparation techniques". Monroeville, PA: Energy Technology Consultants.

Le Guen, J.M.M. and Burdett, G.J., (1981). "Asbestos concentrations in public buildings-a preliminary report"., Ann Occup. Hyg., 24, 185-189.

Marfels, H., Spurny, K. Boose, Ch., Schormann, J., Opiela, H., Althaus, W. and Weiss, G., (1984). "Asbestfasermessungen in rundsporthallen, schwimmhallen und schultzentren in der Bundesrepublik Deutschland". Staub 44, 512-514.

Marshall, M. (1988). "Aggressive sampling and surface dust sampling in an Asbestos Contaminated Building." Presented at the National Asbestos Council Conference Boston, MA. September, 19-21, 1988.

Nicholson, W.J., Rohl, A.N. and Ferrand, E.F. (1971) "Asbestos Air Pollution in New York City. in: Proc. of Second Clean Air Congress. Acad. Press. New York 136-139.

Nicholson, W.J., Rohl, A.N., Weisman, I. and Selikof, I.J., (1980). "Environmental asbestos concentrations in the United States". In Proc. Biological Effects of Mineral Fibers, Vol. 2, pp. 823-827. IARC pub. 30. International Agency for Research on Cancer. Lyon, France.

Nicholson, W.J., Swoszowski, E.J. and Rohl, A.J., (1979). "Asbestos contamination in US schools from use of asbestos surfacing materials." Annals. New York. Acad. Sci., 330. 587- 596.

NRC, (1984). National Research Council. Asbestiform fibers: non-occupational health risks. Washington, DC: National Academy of Sciences.

Olcrest, R.B., (1988). "A Radiological technique to quantitate asbestos decontamination" Paper presented at the Surface Dust Sampling Session of the National Asbestos Council Conference, Boston, MA. September 19-21. 1988.

Ortiz, L.W., Soderholm, S.C. and Valdez, F.O. 1988. "Penetration of respirator filters by an asbestos aerosol." Am. Ind. Hyg. Assoc. J., 49, 9, 451-460.

Paik, N.W.; Walcott, R.J. and Brogan, P.A., (1983). "Worker exposure to asbestos during removal of sprayed material and renovation activity in buildings containing sprayed material". Am. Ind. Hyg. Assoc. J., 44, 428-432.

Philedelphia Dept. of Health. (1988). "Survey of Asbestos in State Buildings."

Pinchin, D.J., (1982). "Asbestos in buildings". Study prepared for the Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario. Ontario Research Foundation. Mississauga, Canada.

Pott, F. (1978). "Some aspects on the dosimetry of the carcinogenic potency of asbestos and other fibrous dusts." Staub 38,

Powers, T.L. 1988. "Achieving a TEM clearance Criterion at asbestos abatement sites in New Jersey". Paper Presented at National Asbestos Council Conference, Boston, MA. Sept 19-21.

Royal Commission Ontario, (1984). Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario. Vol. 2,

Royster, W. and R. Fish, (1967). "Techniques for assessing removable surface contamination," Proceedings of a symposium on Surface Contamination held at Gatlinburg, Tennessee in June, 1964, Edited by B.R. Fish, Pergammon Press Ltd., New York, New York. 1967.

Sebastien, P., Billon-Galland, M.A., Dufour, G. and Bignon, J., (1980). Measurement of Asbestos Air Pollution Inside Buildings Sprayed With Asbestos. U.S. Environmental Protection Agency. EPA-560-13-80-026.

Sebastien, P., Bignon J. and Martin, M., (1982) "Indoor airborne asbestos pollution from the ceiling and the floor". Science. 214, 1410-1413.

Stanton, M.F., Layard, M., Tegeris, A., Miller, E., May, M. and Kent, E. 1977, "Carcinogenicity of fibrous glass: Pleural response in the rat in relation to fiber dimension." J. Natl. Cancer Inst., 58, 587-603.

USDOL, 1983. Quantitative risk assessment for asbestos- related cancers. Occupational Safety and Health Administration. U.S. Department of Labor, Washington D.C.

USEPA, (1976). U.S. Governmental Protection Agency. Asbestos Contamination of Air in Public Buildings. EPA-650/3/76-004.

USEPA, (1979). US Environmental Protection Agency. Asbestos Containing Materials in School Buildings: A Guidance Document Pt. II. EPA-450/2-78-014.

USEPA, (1980). U.S. Environmental Protection Agency. Support document: Asbestos-containing materials in schools -- health effects and magnitude of exposure. Washington, DC: Office of Pesticides and Toxic Substances, USEPA. EPA-560/12-80- 003.

USEPA, 1983. Environmental Protection Agency, 1983. Airborne Asbestos Levels in Schools, (EPA 560/5-83-003) Office of Toxic Substances, Washington, D.C.

USEPA, (1984). US Environmental Protection Agency National Emission Standards for Hazardous Air Pollutants (NESHAPS) Asbestos Regulation (40 CFR 61, Subpart M). Federal Register 49 FR 13661 April 5th 1984.

USEPA, (1985a). U.S. Environmental Protection Agency. Guidance for Controlling Asbestos Containing Materials in Buildings. EPA 560/5-85-024.

USEPA, (1985b). U.S. Environmental Protection Agency. Evaluation of asbestos abatement techniques phase I: removal. Office of Toxic Substances, Washington, DC., EPA 560/5-85-019.

USEPA, (1986a). U.S. Environmental Protection Agency. Airborne asbestos health assessment update. Washington, DC: Office of Health and Environmental Assessment, USEPA. EPA.600/8- 84/003F.

USEPA, (1986b). US Environmental Protection Agency. Evaluation of asbestos abatement techniques phase II: encapsulation with latex paint. Washington, DC: Office of Toxic Substances, EPA 560/5-86-016.

USEPA, (1986c) US Environmental Protection Agency Guidance for Assessing and Managing Exposure to Asbestos in Buildings. Draft report EPA Office of Toxic Substances, Washington D.C., Contract No. 68-02-4243.

USEPA, (1987). US Environmental Protection Agency. Asbestos- Containing Materials in Schools: Final Rule and Notice, (40 CFR 763) Federal Register, Vol 52, No. 210 41826-41903 Friday Oct. 30th.

USEPA, (1988a). U.S. Environmental Protection Agency. Study of Asbestos Containing Materials in public building - A report to Congress. February 1988.

USEPA, (1988b). US Environmental Protection Agency, Assessing Asbestos Exposure in Public Buildings: EPA 560/5-88- 002, May 1988.

USEPA, (1988c). U.S. Environmental Protection Agency. "Assessment of asbestos removal carried out using EPA purple book guidance". EPA Contract No. 68-03-4006.

USEPA, (1988d). U.S. Environmental Protection Agency. "Evaluation of asbestos levels in two schools before and after asbestos removal". EPA contract No. 68-03-4006.

USEPA, (1989). U.S. Environmental Protection Agency. Guide for Developing and Implementing Effective Operations and Maintenance Programs for Asbestos Containing Materials. (Draft report, in review).

Verein Deutscher Ingenieure, 1985. VDI Richtinie 3492: Messen Anorganischer, Faserformiger partikeln in der aubenluft: rasterelektronemikroskopisches verfahren. VDI-Verlag, Dusseldorf, F.R.G.

Walton, W. H., (1982). "The Nature, Hazards and Assessment of Occupational Exposure to Airborne Asbestos Dust: A review.," Ann. Occup. Hyg., 25, 2, 157.

Walvszewski, J. and Paulsson, B., (1988). "Airborne asbestos in conjunction with the removal of installed asbestos material from an office building:" A TEM study report by the Swedish National Institute of Occupational Health. Solna, Sweden.

Wilmoth, R.C., Powers, T.J. and Millette, J.R. (1988). "Observations on studies useful to asbestos O&M activities". Paper presented at the 5th Annual Asbestos Abatement Conference of the National Asbestos Council, Atlanta, Georgia February 23rd, 1988.

TABLE 1: Location and Condition of Friable ACM in U.S. Public and Commercial Buildings (Total population 3.6 million,) Sample population 231)

Location and Type of ACM	Condition of Friable ACM in terms of % of total population and (Number of buildings)		
	Present	Some Slight Damage	Some Significant Damage
All Areas (friable)	20 (733,000)	14 (501,000)	9 (317,000)
Fan and Boiler rooms (friable)	13 (462,000)	10 (360,000)	8 (282,000)
Public Areas (friable)	13 (454,000)	8 (272,000)	2 (85,000)
Sprayed or Trowelled Coatings (friable)	5	2	0
Pipe or Boiler insulation (friable)	16	13	9
Floor Tile (non-friable)	42 (1,526,000)	--	--

* Types of friable ACM included in survey are defined in Table 2.

TABLE 2: Estimated percent of buildings with asbestos-containing friable materials by type of material and type of building (95 percent confidence limits in parentheses)

Type of building	Type of asbestos-containing friable material			
	Sprayed-or Trowelled-on	Ceiling Tile	Pipe/Boiler Insultation	Any Material
Federal government	16% (<33%)	2.0 (0.3-3.6%)	25% (8-41%)	39% (28-48%)
Residential (10 + rental units)	18% (10-27%)	0.5% (<1.7%)	44% (26-62%)	59% (45-74%)
Private nonresidential	4% (<9%)	0	12% (2-22%)	16% (9-23%)
All buildings combined	5% (0.5-10%)	0.1% (<0.2%)	16% (7-25%)	20% (14-27%)

TABLE 3: Asbestos Fiber Release Potential Index

Material Operation	Number of Tests	Asbestos Fibers per cc per gm Asbestos Milled		
		Average (f/cc/gm)	Standard Deviation	Relative Standard Deviation (%)
AC sheet Cut-off wheel	9	1,838.3	338.6	18.4
Millboard Saw	8	646.6	435.2	67.3
Brakes Grind	8	465.1	217.3	46.7
AC sheet Saw	12	305.4	363.7	119.1
AC sheet Drill	8	282.9	222.6	78.7
Millboard Drill	8	105.2	32.0	30.5

TABLE 4: Distribution of Asbestos Structure Types Generated During Asbestos Release Experiments (TEM Analysis)

Material Operation	Percent Asbestos Fibers	Asbestos Structure Distribution			
		Percent Asbestos Bundles	Percent Asbestos Clusters	Percent Asbestos Matrices	Percent Asbestos
AC sheet Cut-off wheel	52.8	6.7	4.7	5.4	69.7
Millboard Saw	64.6	4.7	10.2	3.4	83.0
Brakes Grind	45.0	10.6	13.9	2.8	72.4
AC sheet Saw	49.3	7.5	4.8	13.6	75.2
AC sheet Drill	61.6	3.4	4.3	8.4	77.7
Millboard Drill	68.5	0.0	1.1	4.5	74.2

TABLE 5: Summary of Environmental "Regulatory Fiber" Concentrations

Sample Sets	No. of Samples	Measured Concentration (ng/m³) Median	Measured Concentration (ng/m³) 90th Percentile	Equivalent Concentration (fibers/cm³)[b] Median	Equivalent Concentration (fibers/cm³)[b] 90th Percentile	Reference
1. Paris air	161	0.7	3.2	0.00002	0.00011	Sebastien et a (1980)
2. Paris (outdoor control)	19	0.7	5.2	0.00002	0.00017	Sebastien et a (1980)
3. Outdoor control samples, for U.S schools	31	0.9	9.8	0.00002	0.00033	USEPA (1983)
4. Air of 48 U.S. cities	187	1.6	6.8	0.00005	0.00023	Nicholson, (19
5. Air of five U.S. cities	127	2.3	7.8	0.00008	0.00026	USEPA (1974)
6. Air of five U.S. cities (outdoor control sample)	34	6.7	31.9	0.00022	0.00106	USEPA (1976)
7. New York City air	22	13.7	42.9	0.00046	0.00143	Nicholson e (1971)
8. Air 0.5 mile (0.8 km) from asbestos spraying	17	22.5	82.6	0.00075	0.00275	Nicholson e (1971)
9. Air in U.S. school rooms without asbestos	31	16.3	72.7	0.00054	0.00243	USEP/ (1983)
10. Air in Paris buildings with asbestos surfaces	135	1.8	32.2	0.00006	0.00107	Sebastien e (1980
11. Air in U.S. buildings with cementitious asbestos	28	7.9	19.1	0.00026	0.00064	USEP/ (1976
12. Air in U.S. buildings with	54	19.2	96.2	0.00064	0.00321	USEP (197
13. Air in U.S. school rooms with asbestos surfaces	54	62.5	550	0.00208	0.01833	USEP (198
14. Air in U.S. schools with damaged asbestos surfacing materials	27	121.5	465	0.00405	0.01550	Nicholson (197

[a]Adapted from Nicholson, 1983
[b]Based on conversion factor of 30 ug/m³ = 1 fiber/cm³.

As Summarized for the National Research Council 1984

Table 6: Summary of Measurement by Direct TEM Analysis

Site Description	Fiber Concentration	Reference*
Results for Asbestos Fibers >5 um long in Building under Normal Occupation		
19 Buildings containing friable sprayed asbestos	All <0.001	Royal Commission Ontario (1984)
11 Buildings containing friable spray/trowelled asbestos	All <0.001 (except 1 site with damage)	Burdett and Jaffery (1986)
24 Buildings with ACM in heaters	All <0.001	" "
1 Building with low asbestos contents decorative plaster	All <0.001	" "
4 Buildings without asbestos	All <0.001	" "
25 Buildings with amosite containing board	24 at $\leq$ 0.002 (LOQ) (average 0.0003)	Gazzi and Crockford (1987)
5 Buildings with no ACM	All <0.002 (average 0.0001)	" "
45 Buildings with ACM	All <0.001	CPSC (1989)
Samples from 43 Federal buildings with ACM	15 Fibers found in 300 air samples	Lee (1987) USEPA (1988b)
Results for Asbestos Structures (all sizes) f/l		
Federal buildings 6 buildings with undamaged ACM	mean 0.59	USEPA (1988b)
37 buildings with damaged ACM	mean 0.73	
6 buildings no ACM	mean 0.99	

Table 7: Surface Sampling Methods Which Have Been Applied in Buildings with ACM

Method	Performance Comments	Reference
Direct Particle Collection (e.g. Picking or scraping particles) from the surface.	Standard procedure when gross visible contamination is found.	Standard method
Indirect Particle Collection (Surface removed)	Here the surface itself is removed for more vigerous extraction of dust by ultrasonic treatment (normally restricted to carpets and fabrics)	Royster and Fish (1987), Wilmoth et al., (1988), Guth (1988) Olcrest (1988), Karaffa et al. (1987)
Microvacuuming (open head filter)	High flow velocities are required to re-entrain surface dusts-less efficient on rough/carpet surfaces. Standard filter cassettes are often used.	Royster and Fish (1987), Wilmoth et al., (1988), Guth (1988) Olcrest (1988), Karaffa et al. (1987)
SMAIR Sampler	A more specialized filter cassette which has high velocity jets, but does not cover the whole pick-up area and has low efficiency.	Royster and Fish (1967) Wilmoth et al. (1988)
Adhesive tape	Good collection efficiency for smooth surfaces but very limited for uneven surfaces.	Royster and Fish (1967) Guth (1988), Olcrest (1988)
Filter wipe samples (dry and wet)	Generally only applicable to very smooth surfaces - uneven deposit collected.	Guth (1988) Olcrest (1988)
Wet Sponge wiping	Apparently more efficient than filters but difficult to recover sample	Guth (1988) Olcrest (1988)
Adhesive film	New technology-a surface film spray-on polymer which is pulled off when dry.	Guth (1988) Olcrest (1988)

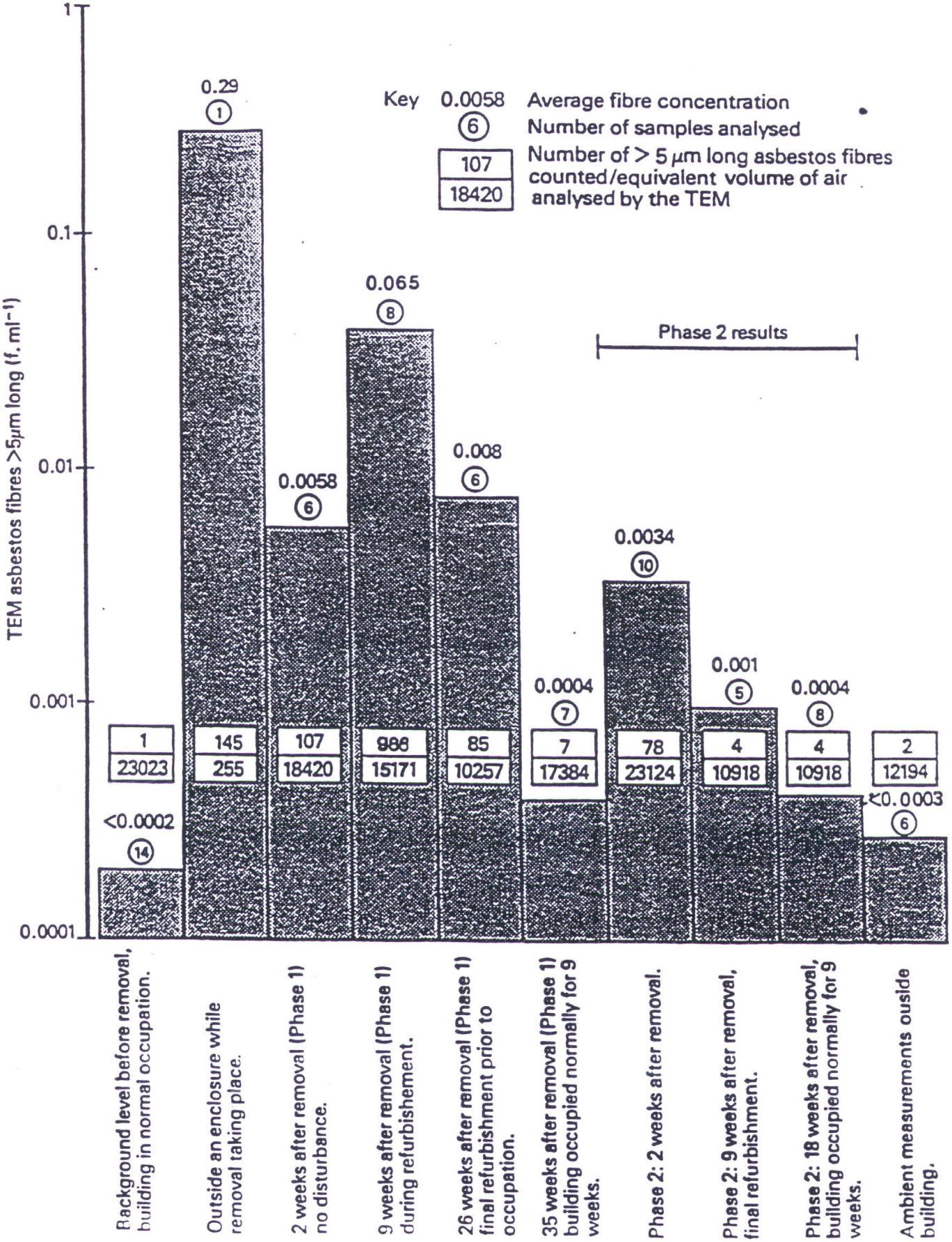


Fig.1 – Average TEM concentrations of asbestos fibres >5μm long: Before, during and after two phases of an asbestos removal programme from a six-storey teaching block.

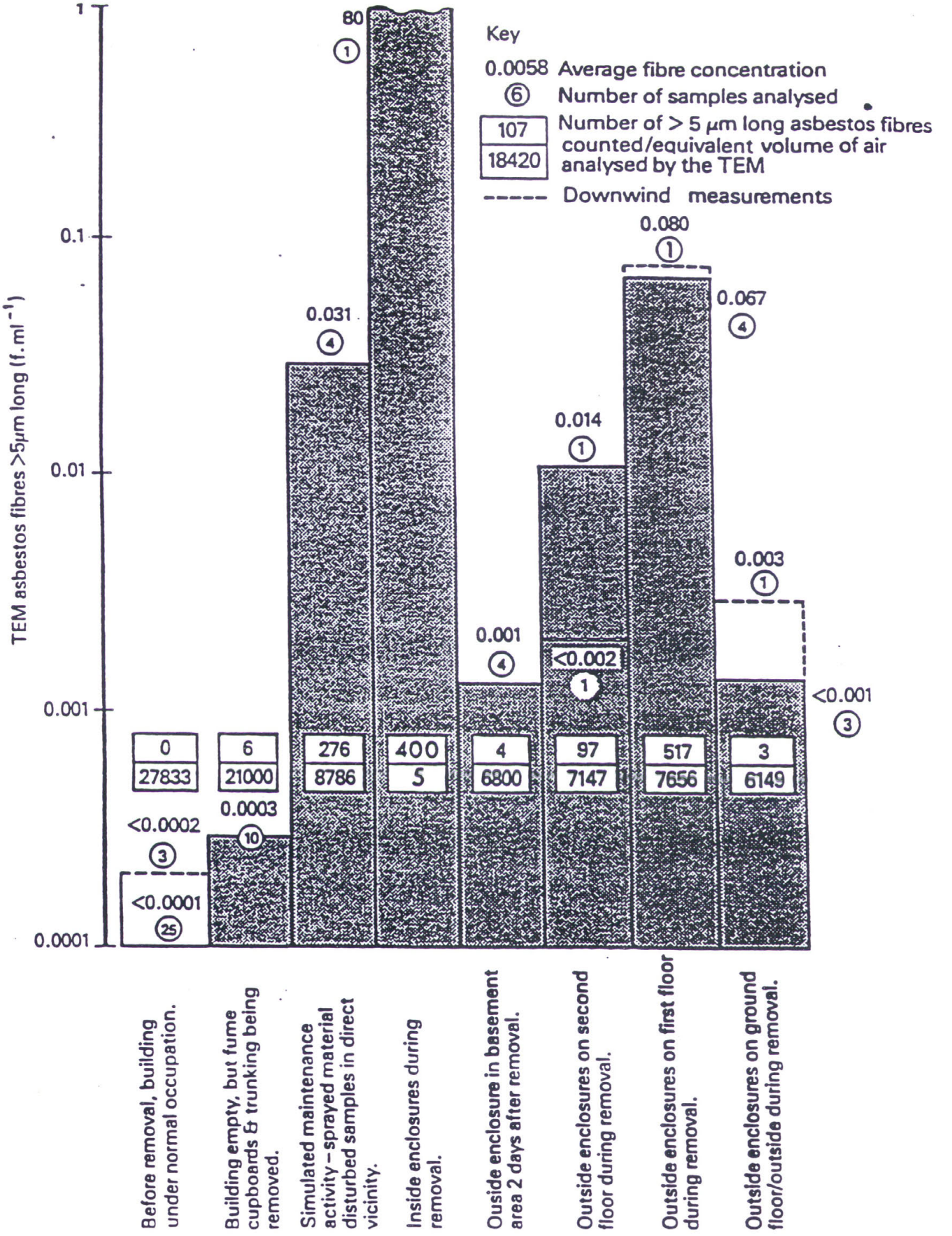


Fig. 2 – Average TEM concentrations of asbestos fibres >5μm long:
During disturbance and removal of asbestos from a four storey building.

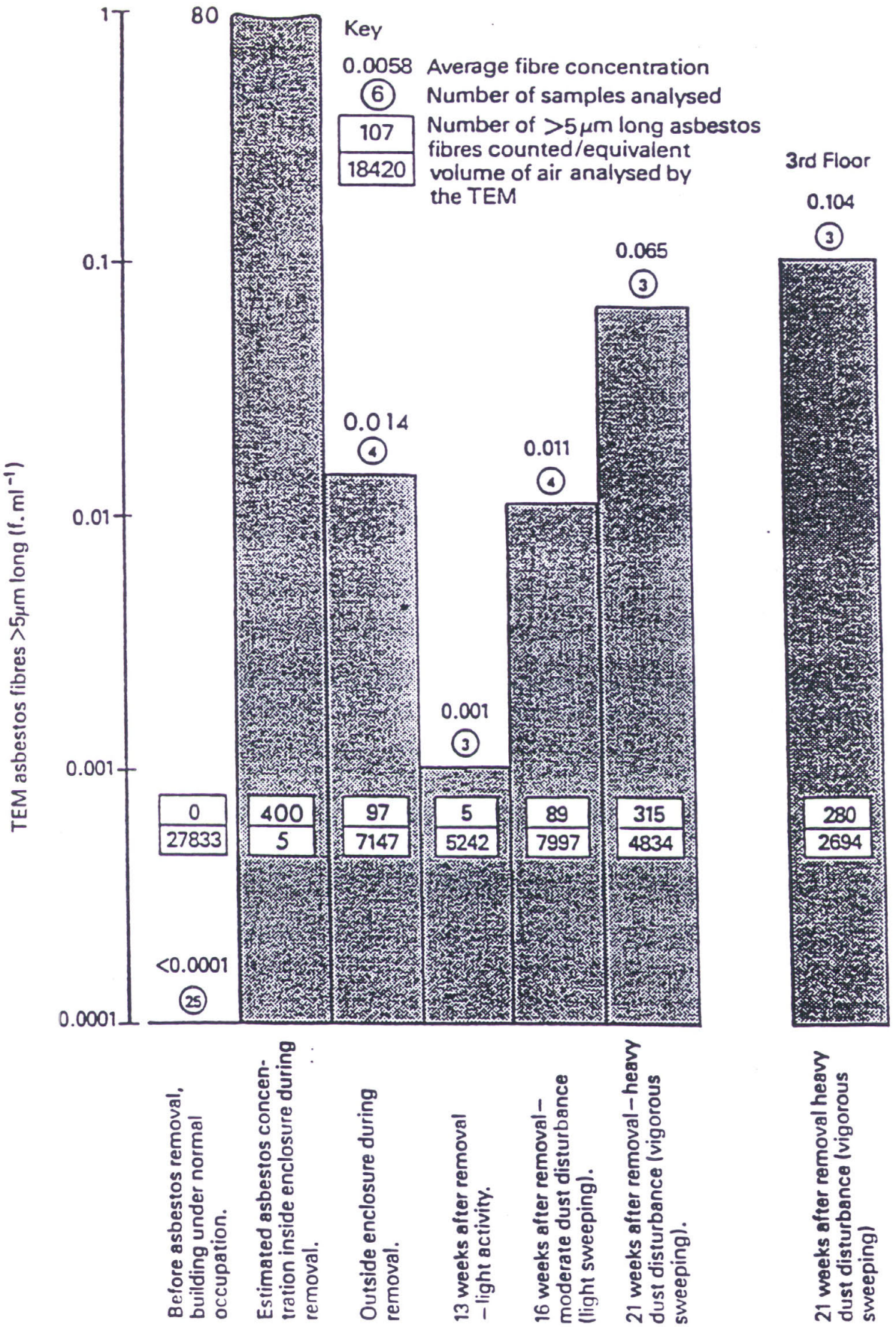


Fig.3 – Average TEM concentrations of asbestos fibres >5μm long: 2nd and 3rd Floor, Before during and after asbestos removal.

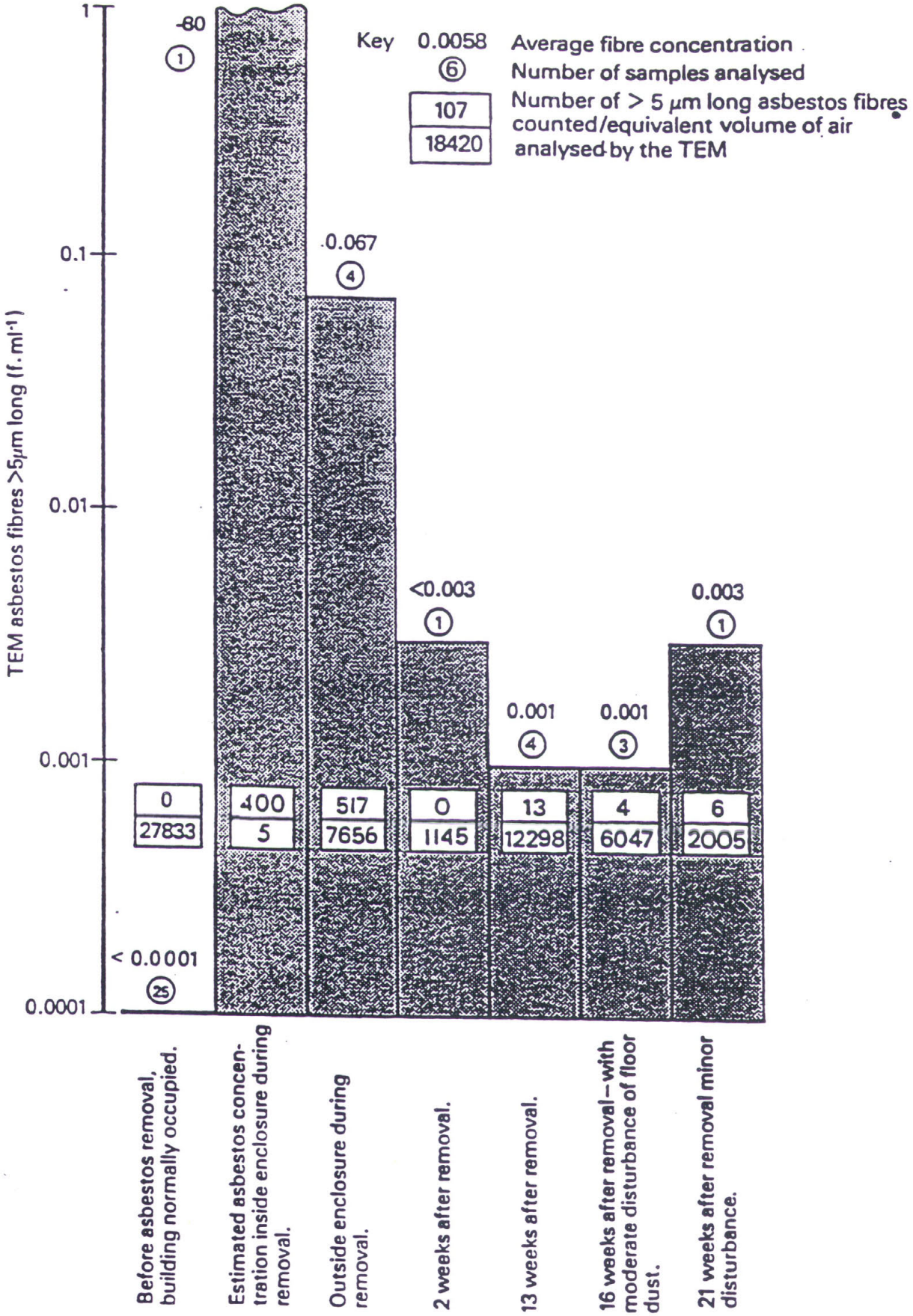


Fig. 4 – Average TEM concentrations of asbestos fibres >5μm long: 1st Floor. Before, during and after asbestos removal.

THE DERIVATION AND USE OF ASBESTOS RISK ESTIMATES

JANET M. HUGHES

Tulane University Medical Center, New Orleans, La. 70112

This review assesses the magnitude of the potential carcinogenic risk from exposure to environmental sources of asbestos exposure such as asbestos-containing building materials. Results of two approaches to assessing this risk are compared and shown to be consistent; these are: 1) the use of mathematical models for extrapolating risks observed at high industrial exposure levels down to current environmental levels, and 2) the examination of direct evidence of risk from environmental exposures. The potential risk from prevalent building exposure levels, assuming such a risk exists, is so small that it cannot be detected and is lower than many common risks in our society.

INTRODUCTION

In attempting to deal rationally with the possibility of a health risk to occupants of buildings with asbestos - containing materials, the first step is to determine the likely magnitude of the potential risk, using the best available evidence to date. Once the level of risk has been estimated, society is able to judge the gravity of the situation, place the risk in perspective by comparison with well-known hazards, and make informed decisions regarding prudent action.

This brief review of the development and use of asbestos risk estimates will describe the methodologies and the assumptions underlying them, and illustrate their derivation under assumed conditions of exposure for building occupants.

The potential risks to be considered are those of lung cancer and mesothelioma. Although lung fibrosis (asbestosis) was a serious risk for past workers occupationally exposed to asbestos, there is general agreement that asbestosis is not a concern for environmental exposures. Similarly, cancers other than lung cancer and mesothelioma are usually not considered because of the very small number of cases compared with lung

cancer, and because, for most cancer sites, there is not convincing evidence of an association with asbestos.

In assessing risk of lung cancer and mesothelioma from environmental asbestos exposure, there are two potential approaches: 1) use mathematical models to extrapolate results at high occupational levels down to exposure levels in buildings, and 2) rely on direct evidence of risk associated with low, non-occupational exposures. Both approaches will be considered here.

MODELLING LUNG CANCER RISK

Although numerous studies have demonstrated an increased risk of lung cancer among past asbestos workers, only a few have demonstrated a dose-response relationship between observed risk and estimated cumulative exposure (based on individual job records and exposure measurements by job area and time). In these, the lung cancer risk is expressed as the ratio of the observed number of lung cancers (O) to the number expected (E) based on general population rates. In the absence of asbestos exposure, this ratio should be approximately one (or 100 if the ratio is multiplied by 100 to obtain the Standardized Mortality Ratio (SMR)).

An approximately linear relationship has been observed between risk and cumulative exposure, although there has been considerable variability in the slope of the lines. Slopes for a mining (McDonald et al, 1980)) and two friction product manufacturing cohorts (Berry and Newhouse, 1983; McDonald et al, 1984) have been very shallow or flat, but studies of three textile (McDonald et al, 1983a; McDonald et al, 1983b; Peto et al, 1985) and an asbestos cement manufacturing cohort (Hughes et al, 1987) have observed slopes of approximately .01, resulting in a dose-response equation of: $O/E = 1.0 + .01\ (x)$, where x is cumulative asbestos exposure in fibers per milliliter - years (f/ml-yr). This equation may be rewritten as: $O = E + E\ (.01)\ x$.

Making the assumption that this equation is valid for all levels of cumulative exposure (x), the equation states that the number of lung cancers projected to be observed (O) in a population with exposure x is the sum of the number expected without asbestos exposure (E) plus an additional amount (E (.01) x) attributable to the exposure.

The background number expected (E) is usually calculated from national or regional mortality rates. For example, by 1982 U.S. rates, the lifetime risk of a man dying of lung cancer is approximately .07; thus, of 100,000 males, approximately 7,000 lung cancer deaths would be expected over their lifetime (E=7,000).

If these 100,000 men work for 20 years in a building with average asbestos concentration levels of .001 f/ml, then their cumulative exposure (x) is .02 f/ml-yr

(.001(20)), resulting in an estimated 1.4 excess lung cancers [7,000 (.01) (.02)] over the lifetime of the group; these 1.4 cases would be in addition to the 7,000 background cases.

The estimated excess (Ebx) depends on the background rate (E). Use of current lung cancer mortality rates implicitly assumes smoking rates remain similar to rates among U.S. men in recent decades. If overall U.S. lung cancer rates decline (as has occurred among younger men as a result of the recent substantial decrease in the percentage of men smoking), then the estimated excess would also decline.

Linear modelling of lung cancer risk has several strengths: it is consistent with data from occupational cohorts; it provides a reasonable approximation to other models for low exposure levels; and, compared to most other models, it is conservative in the sense of possibly overestimating the potential risk from low exposures since it assumes that any amount of exposure, no matter how small, will result in elevated lung cancer risk.

However, a major drawback of the modeling is that extrapolation is being made far beyond the range of available data: past occupational exposures, which are the basis for the model, were often in the range of 100 f/ml-yrs (e.g., 10 f/ml for 10 years) or higher, whereas risk estimates such as above are for levels orders of magnitude smaller (e.g.,.02 f/ml-yr).

Additionally, there are several epidemiologic studies supporting the view (Newhouse et al, 1985; Kipen et al, 1987; Hughes and Weill, 1989) held by a number of researchers (Parkes, 1982; Kuschner, 1987) that asbestos-induced lung cancer is a complication of asbestosis. This evidence contradicts the assumption of a lung cancer risk from environmental asbestos exposures since there is general agreement that asbestosis does not result from such exposures.

MODELLING MESOTHELIOMA RISK

Since mesothelioma is a relatively rare disease, reliable background rates for calculating expected background numbers (E) are not available; therefore, in reporting mesothelioma risk in studies of past workers, it has not been possible to calculate the ratio of observed to expected numbers, as has been done for lung cancer.

Instead, mesothelioma modeling is based on the observation from several studies that incidence rates increase as a power of time since exposure began, a finding consistent with observations for other cancers (e.g., lung cancer in relation to cigarette smoking). This observation, along with the assumption that incidence is proportional to exposure concentration and the incorporation of exposure duration in the model based on multi-stage carcinogenicity (Whittemore), has resulted in the model:

$$I(t) = \begin{cases} K\,c\,t^{3.2}, & 0 < t < d \\ K\,c\,[\,t^{3.2} - (t-d)^{3.2}\,], & t > d, \end{cases}$$

where c is concentration, d duration and t time since exposure began. By this model, during exposure ($t < d$) incidence rises as a power of length of exposure; after exposure ceases ($t > d$), incidence rises less steeply.

The model has been fit to data from seven epidemiologic studies (Hughes and Weill, 1986); as examples, the observed and fitted mesothelioma rates for three studies of mixed fiber exposed populations are shown in Figure 1. Data for a fourth mixed fiber study (Hughes et al, 1987) are also consistent with the model.

Slightly different formulations of the model have been used in estimating mesothelioma risk (Peto et al, 1982; Peto et al, 1985; Consumer Product Safety Commission, 1983), but there are only minimal differences in estimated lifelong risk.

To use the above model, it is first necessary to fit it to the available data sets in order to estimate the multiplicative parameter K (similar to estimating the slope of the line for the lung cancer model). A review of the data (Hughes and Weill, 1986) has found considerable variability in the estimates of K from these studies, but that, for mixed asbestos fiber exposures, the median value of $.2(10^{-8})$ provides a reasonable estimate.

To use the model to estimate potential mesothelioma risk for a population with an assumed concentration and duration of asbestos exposure, lifetable methods are used, with the mesothelioma incidence during each year of follow-up calculated from the above model.

For example, with the same exposure assumptions used previously (100,000 men employed for 20 years in a building with a mixed asbestos fiber exposure concentration of .001 f/ml), assuming exposure begins at age 20, the model estimates .9 mesothelioma cases by age 80 (.5 by age 70).

Since incidence increases with time since exposure started, if the same exposure began at birth, then the estimate would be higher: 2.6 cases per 100,000 by age 80 (1.8 by age 70).

Although the model provides a reasonable fit to the available data, there are also major uncertainties. It has been suggested (Peto et al, 1985) that the model may overestimate the risk from short-term exposures (less than 10 years) and underestimate risk from longer exposures. Additionally, as with lung cancer modeling, extrapolation is being made to exposures several orders of magnitude below which there are available data.

Extrapolation is also being made for years of follow-up: estimates are calculated up to 80 years from the start of exposure, whereas the maximum follow-up time for the industrial cohorts is approximately 50 years. When using the model to estimate cases over an 80 year period, approximately 80% of the projected cases occur after 50 years from start of exposure. The appropriateness of this extrapolation has been questioned (Crump, 1984) because, where data are available, incidence rates more than 40 years from start of exposure appear to be lower than the fitted model. This suggests that incidence may not continue to increase as steeply as assumed.

Many, though not all, of the asbestos products in buildings consist only of chrysotile asbestos. There is extensive evidence that chrysotile exposure is considerably less hazardous with respect to mesothelioma risk than exposure to the amphiboles (McDonald and McDonald, 1988). One reviewer has suggested that chrysotile may be at least two orders of magnitude less dangerous than amosite, and even less dangerous when compared with crocidolite (Churg, 1988). Moreover, based on recent lung burden studies, in the relatively few cases among workers with chrysotile exposure only, it is possible that the causative agent is the contaminant tremolite, an amphibole (Churg, 1988). The lower mesothelioma risk of chrysotile compared with the amphiboles has been recognized by most industrialized countries (with the notable exception of the United States), who have instituted differing regulations for the various types of fibers.

Since the parameter of the mesothelioma model was estimated based on industrial cohorts regularly using amphiboles (in addition to chrysotile), the model overestimates risk due to chrysotile exposure alone. However, it is difficult to reliably determine how to quantitatively adjust the estimate when considering chrysotile exposure only. A comparison of cohort studies by type of fiber exposure suggests a factor of five (Hughes and Weill, 1986), which will be used in this report.

PLACING THE RISK ESTIMATES IN PERSPECTIVE

For the example considered above (100,000 men exposed 20 years to .001 f/ml mixed asbestos starting at age 20), the models estimated 1.4 lung cancer and 0.9 mesothelioma deaths by age 80, for a total of 2.3 deaths attributable to the exposure. If the same exposure were to chrysotile only, then using the adjustment factor of five, there would be an estimated .2 mesothelioma deaths, for a total of 1.6 deaths.

Assuming the average expected lifetime is approximately 74 years, the cohort would have, on average, 54 years of life remaining, so the 2.3 deaths attributable to a mixed asbestos exposure would be an average annual rate of .04 deaths per 100,000 (2.3/54), or 0.4 per million (for chrysotile, 0.3 per million).

Risk estimates for other conditions of building exposures appear in Table 1. In deriving the lung cancer risk assuming a population consisting of half males and females, the background lifetime lung cancer risk was assumed to be .05.

Expressing the risk as an average annual rate enables comparisons with well-known risks which are often reported on an annual basis; such comparisons are useful for making judgments concerning the gravity of the potential risk and the wise allocation of resources. For example, an annual risk of 0.4 per million is far less than the annual risk from home accidents [88 per million], diagnostic x-rays [75 per million], lightening [3 per million] (Commins, 1985) and indoor radon exposures [50 per million] (Nero, 1988).

DIRECT EVIDENCE

Studies which have attempted to directly assess the risk from environmental asbestos exposures have been of two types: 1) community-based, in which populations living around an asbestos facility have been studied for evidence of increased cancer risk, and 2) case-based, in which non-occupationally exposed mesothelioma cases have been investigated for evidence of clustering around an asbestos industry.

Three community studies found no evidence of increased cancer risk (Hammond et al, 1979; Siemiatycki, 1982; Neuberger et al, 1984). Residents of at least one of these communities (Siemiatycki, 1982) were exposed for a lifetime to several hundred times the ambient chrysotile levels of most North American cities.

Two studies of mesothelioma cases found no evidence of a clustering of non-occupationally related cases around asbestos facilities (McDonald and McDonald, 1980; Teta et al, 1983), but other studies found a clustering of such cases in communities around a South African crocidolite mine (Wagner et al, 1960) and factories using crocidolite (Newhouse and Thompson, 1965; Bohlig et al, 1970). However, the relevance of these positive findings to current environmental exposures is doubtful because of the unquantified, but likely much higher, exposure levels and the current limited crocidolite use.

Another approach to obtaining direct evidence of risk attributable to environmental asbestos exposure has been the examination of trends in national mesothelioma incidence rates. Because of the markedly increased use of asbestos since the early 1950's, if exposure to asbestos from building products and in the general environment constituted a substantial hazard, then, after allowing an appropriate lag time of 20 years or so for cancer to develop, a similar increase in mesothelioma rates would be expected, beginning approximately in 1970. In fact, rates among women have been shown (Mowe, 1982; Spirtas et al, 1986; Jones et al, 1988) to exhibit no detectable increase, with rates for U.S. women approximately 2.8 per million. As noted by McDonald (1985), some women could be expected to have had occupational asbestos exposure, so that these findings demonstrate that any mesothelioma

risk attributable to non-occupationally related asbestos exposures, if it exists, must be extremely small.

Mesothelioma risk estimates based on modeling are consistent with the lack of a detectable increase in women over time. If U.S. women are assumed to have been exposed to an average concentration of .0002 f/ml mixed asbestos (chrysotile and amphiboles) continuously (24 hours/day, every day) for the last 40 years (likely an overestimate since prevalent exposures in the approximately 20% of public buildings containing asbestos is around .001 f/ml), then the estimated asbestos-attributable incidence rate would be approximately .2 cases per million annually. Such an increase, if it occurred, would be too small to be detectable from the U.S. background rate of approximately 2.8 per million. (If such an exposure continued for 60 years, the model estimates an asbestos-attributable annual incidence of approximately one case per million.)

SUMMARY

In summarizing the estimates of risk from mathematical modeling, it is important to emphasize that these models assume that any amount of asbestos exposure, no matter how small, results in an increased cancer risk, with risk proportional to the amount of exposure. Having made the assumption that a risk exists, the models then attempt to quantify its amount.

In fact, community-based studies and the examination of trends in mesothelioma rates have detected no evidence of increased risk attributable to environmental asbestos exposures. These findings are consistent with the quantitative estimates, indicating that the amount of risk, if it exists, is so small that it cannot be detected.

REFERENCES

Bohlig H, Dabbert AF, Dalquen P, et al. (1970) Epidemiology of malignant mesothelioma in Hamburg. A preliminary report. Environ Res 3:365-72.

Berry G, Newhouse ML. (1983) Mortality of workers manufacturing friction materials using asbestos. Br J Ind Med 40:1-7.

Churg A. (1988) Chrysotile, tremolite, and malignant mesothelioma in man. Chest 93:621.

Commins BT. (1985) "The significance of asbestos and other mineral fibres in environmental ambient air". Commins Associates, Berkshire, England :

Consumer Product Safety Commission. (1983) Report of the Chronic Hazard Advisory Panel on Asbestos. Washington, D.C.

Crump KS. (1984) Testimony to the U.S. Department of Labor concerning a proposed revision of the asbestos standard. Washington, D.C.

Hammond EC, Garfinkel L, Selikoff IJ, Nicholson WJ. (1979) Mortality experience of residents in the neighborhood of an asbestos factory. Ann NY Acad Sci 330:417-422.

Hughes JM, Weill H. (1986) Asbestos exposure - quantitative assessment of risk. Am Rev Respir Dis 133:5-13.

Hughes JM, Weill H, Hammad YY. (1987) Mortality of workers employed in two asbestos cement manufacturing plants. Br J Ind Med 44:161-174.

Hughes JM, Weill H. (1989) Pulmonary fibrosis as a determinant of asbestos-induced lung cancer in a population of asbestos cement workers. In: Proceedings of VIIth International Pneumoconioses Conference, Pittsburgh, August, 1988. (In press)

Jones RD, Smith DM, Thomas PG. (1988) Mesothelioma in Great Britain in 1968-1983. Scandinavian Journal of Work, Environment and Health 14:145-152.

Kipen HM, Lilis R, Suzuki Y, Valciukas JA, Selikoff IJ. (1987) Pulmonary fibrosis in asbestos insulation workers with lung cancer: a radiological and histopathological evaluation. Br J Ind Med 44:96-100.

Kuschner M. (1987) The effects of MMMF on animal systems: some reflections on their pathogenesis. Ann Occup Hyg 31:791-7.

McDonald AD and McDonald JC. (1980) Malignant Mesothelioma in North America. Cancer 46(7):1650-56.

McDonald AD, Fry JS, Woolley AJ, McDonald JC. (1983a) Dust exposure and mortality in an American chrysotile textile plant. Br J Ind Med 40:361-367.

McDonald AD, Fry JS, Woolley AJ, McDonald JC. (1983b) Dust exposure and mortality in an American factory using chrysotile, amosite, and crocidolite in mainly textile manufacture. Br J Ind Med 40:368-374.

McDonald AD, Fry JS, Woolley AJ, McDonald JC. (1984) Dust exposure and mortality in an American chrysotile asbestos friction products plant. Br J Ind Med 41:151-157.

McDonald AD, McDonald JC. (1988) Epidemiology of malignant mesothelioma. In "Asbestos-Related Malignancy" (K. Antman and J. Aisner, Eds) Grune & Stratton, New York.

McDonald JC, Liddell FDK, Gibbs GW, Eyssen GE, McDonald AD. (1980) Dust exposure and mortality in chrysotile mining, 1910-1975. Br J Ind Med 37:11-24.

McDonald JC. · (1985) Health implications of environmental exposure to asbestos. Environmental Health Perspectives 62:319-328.

Mowe G. (1982) Time trend in the incidence of malignant mesothelioma in Norway (1970-1979). In "Proceedings of the International Symposium on the Prevention of Occupational Cancer, Helsinki, April 21-24, 1981". 46:213-219. International Labor Office, Geneva.

Nero AV. Controlling indoor air pollution. (1988) Scientific American 258 (5):42-48.

Neuberger M, Kundi M, Friedl HP. (1984) Environmental asbestos exposure and cancer mortality. Arch Environ Health 36:261-265.

Newhouse ML, Thompson H. (1965) Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. Br J Ind Med 1965; 22:261-269.

Newhouse ML, Berry G, Wagner JC. (1985) Mortality of factory workers in East London, 1933-1980. Br J Ind Med 42:4-11.

Parkes WR. "Occupational Lung Disorders" 2nd Edition. (1982) Butterworth & Co., Ltd., London.

Peto J, Seidman H, Selikoff IJ. (1982) Mesothelioma mortality in asbestos workers: implications for models of carcinogenesis and risk assessment. Br J Cancer 45:124-135.

Peto J, Doll R, Hermon C, Binns W, Clayton R, Goffe T. (1985) Relationship of mortality to measures of environmental asbestos pollution in an asbestos textile factory. Ann Occup Hyg 29:305-355.

Siemiatycki J. (1982) Mortality in the general population in asbestos mining areas. World Symposium on Asbestos, Montreal, Canada.

Spirtas R, Beebe GW, Connelly RR, Wright WE. (1986) Recent trends in mesothelioma incidence in the United States. Am J Ind Med 9:397-407.

Teta MJ, Lewinsohn HC, Meigs, JW, Vidone RA, et al. (1983) Mesothelioma in connecticut, 1955-1977. J Occup Med 25:749-56.

Wagner JC, Sleggs CA, Marchand P. (1960) Diffuse pleural mesothelioma and asbestos exposure in the north western Cape province. Br J Ind Med 17:260-271.

TABLE 1

ESTIMATES OF ASBESTOS-ATTRIBUTABLE RISK FOR POPULATIONS EXPOSED TO .001 F/ML ASBESTOS FIBERS, UNDER VARIOUS EXPOSURE ASSUMPTIONS

POPULATION	INITIAL AGE	HRS/WK	WKS/YR	DURATION (YRS)	ESTIMATED AVERAGE ANNUAL RISK[a] MIXED[b]	CHRYSOTILE[c]
WORKERS	20	40	50	20	.36	.22
WORKERS	30	40	50	20	.33	.25
SCHOOL CHILDREN	10	35	36	5	.09	.04

a AMONG ONE MILLION EXPOSED, CONSISTING OF HALF MALE, HALF FEMALE

b MIXED CHRYSOTILE AND AMPHIBOLES

c MESOTHELIOMA RISK FROM CHRYSOTILE ASSUMED TO BE ONE-FIFTH THAT FROM MIXED FIBERS

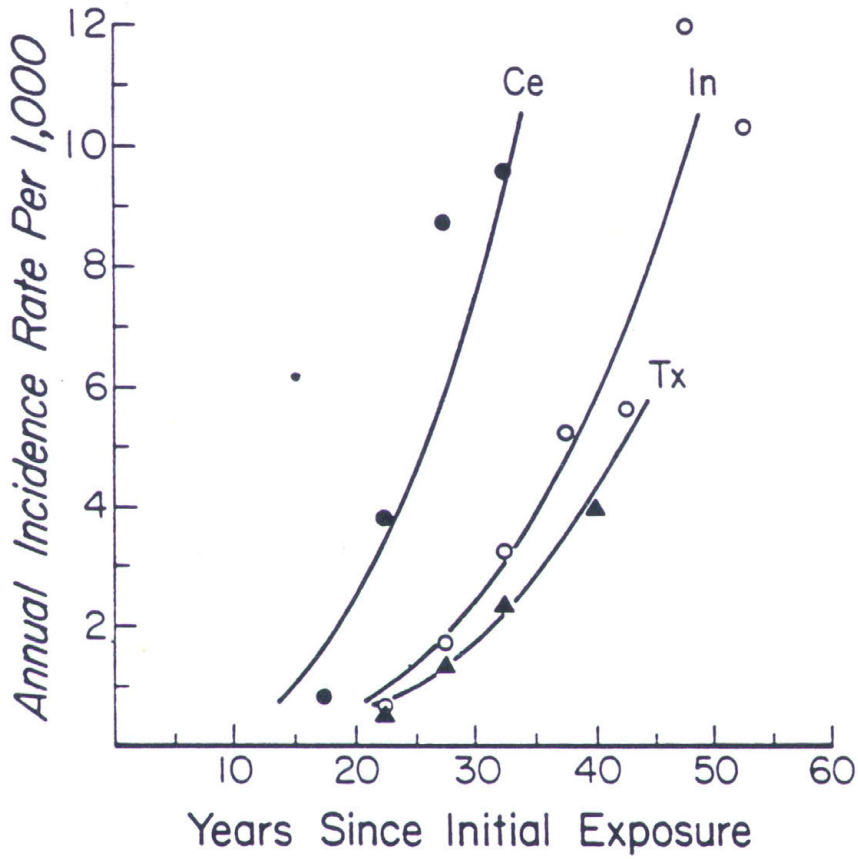


Figure 1

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

FIBRE CARCINOGENESIS AND ENVIRONMENTAL HAZARDS[1]

JULIAN PETO

Section of Epidemiology, Institute of Cancer Research, Sutton, Surrey, UK

The main aim of this paper is to re-examine the epidemiological assumptions and exposure measurements that underlie asbestos risk assessment. Extrapolation from observations on asbestos workers to predict the cancer risk caused by exposure in other industrial or non-occupational situations involves three steps: measuring dose, measuring the resulting excess cancer risk, and establishing a formula for the relationship between exposure and risk. There is little difficulty in measuring the risk, provided that the excess is substantial and suitable comparison rates are available for the local population; but measuring exposure and choosing an appropriate dose-response model are very much more difficult.

Three other aspects of fibre carcinogenesis are also briefly discussed here: (1) the widespread belief that one thin fibre is more dangerous than one thick fibre appears to be the opposite of the truth; (2) the suggestive epidemiological evidence that some synthetic fibres may be more potent bronchial carcinogens than chrysotile asbestos at the same fibre level is therefore not surprising; and (3) the evidence that asbestos can cause gastrointestinal cancer is much weaker than is generally supposed.

Much of the material referred to is described in more detail in a review prepared for the UK Health & Safety Commission (Doll & Peto, 1985). A formal description of the models for lung cancer and mesothelioma incidence can be found in Peto *et al.* (1985).

---

[1] Reprinted with permission from: Non-occupational Exposures to Mineral Fibres, edited by J. Bignon, J. Peto, and R. Saracci (IARC Scientific Publications No. 90), International Agency for Research on Cancer, 1989.

DOSE MEASUREMENT

Prolonged heavy exposure has in the past caused relative risks for lung cancer of between about 2 and 10 in mining, manufacture and use of the major types of asbestos. The only sectors of the industry in which such high risks have not been observed are those in which asbestos, particularly chrysotile, is bonded into other material, as in the manufacture of chrysotile asbestos-cement and friction products. Estimates of the dust levels that caused these high risks have varied widely, however, resulting in much wider variation in risk estimates than in observed cancer rates. This is illustrated by the comparison of the chrysotile textile workers and chrysotile miners and millers studied by McDonald and his colleagues (McDonald *et al.*, 1980, 1983). The most heavily exposed workers in these cohorts suffered relative risks for lung cancer of about 10 for textiles and about 3 for mining. Historical exposure estimates indicated that the highest exposures were more than ten times greater in the mines than in the textile factory, however, resulting in a 50-fold difference in risk estimates (Figure 1).

Historical exposure estimates are thus crucial to asbestos risk assessment. There are only three cohort studies of asbestos workers in which substantial excess cancer risks have been observed, and extensive particle counts taken in the early 1950s or before, together with the basis for their conversion to fibre counts, have been described in reasonable detail. These are the 2 studies described above, and a cohort of English chrysotile textile workers. The dose-response estimates derived from these three studies are often cited, but few reviewers have examined the reliability of the dust measurements on which they are based, perhaps because the original data have not been readily accessible. The exposure data on the English textile factory were not adequately described until 1985 (Peto *et al.*, 1985), although they had been used in several earlier dose-response analyses (e.g., Peto, 1978), and detailed exposure data on the Quebec miners and millers (McDonald *et al.*, 1980) and the South Carolina textile workers studied independently by Dement *et al.* (1982) and McDonald *et al.* (1983) were published separately (Dagbert, 1976; Dement, 1980) and only briefly summarized in the main reports. There are 4 major limitations of historical exposure measurements, each of which casts serious doubt on the validity of any quantitative risk assessment for asbestos:

1. No simple or consistent relationship exists between particle counts taken in the past with different instruments, or between particles and fibres. Comparisons of measurements with different instruments have revealed inconsistencies in the ratio of particle to fibre counts between instruments, between personal and static samples, between different sectors of the industry, and even between different areas within a single factory. In view of these inconsistencies, the ratio of particle to fibre counts observed in parallel

studies provides a very unreliable basis for converting earlier (and often much higher) particle counts to fibre counts.

2. In each instance (English and South Carolina textile factories, and Quebec chrysotile miners), the dose-response relationship deduced was strongly influenced by the high mortality experienced by workers who were heavily exposed in periods and areas where little or no routine dust measurement was done. These exposures could only be guessed.

3. The conclusions drawn have emerged from the handful of workplaces in which extensive historical sampling was carried out. No exposure data are available from which useful risk estimates can be calculated for any type of crocidolite or amosite exposure or for many sectors of the chrysotile industry.

4. Even modern fibre counts by electron microscopy are unlikely to provide a reliable measure of carcinogenic effect. Carcinogenicity and transforming potency per fibre vary with fibre dimension and fibre type in animal studies and *in vitro*, but these relationships are still poorly understood. Furthermore, effects in man may be quite different from those in animals, and there are no useful data on the effects of fibre type or dimensions on human carcinogenicity.

After reviewing the evidence underlying these assessments, we concluded that:

"When so much work has been done in collecting and analyzing measures of ambient pollution, we hesitate to suggest that the results are insufficiently reliable to justify making any quantitative extrapolation from past experience to the effects of current exposures. Nevertheless, this may, in fact, be the case and we may have to be satisfied with qualitative conclusions based on knowledge of the direction in which progress has been made and epidemiological observations of the effects of qualitatively different types of exposure." (Doll & Peto, 1985)

DOSE-RESPONSE RELATIONSHIPS FOR LUNG CANCER AND MESOTHELIOMA

The majority of published dose-specific estimates of the cancer risk caused by asbestos exposure have been based on the following statistical models:

1. The increase in relative risk for lung cancer is proportional to cumulative asbestos exposure, and the effects of asbestos and cigarette smoking multiply each other.

2. The increase in absolute mesothelioma incidence caused by each brief period of asbestos exposure is proportional to the amount of that exposure (exposure level X duration) and to a power of the time since it occurred, independent of age or smoking history. The power of time appears to be about 3 (Peto el al., 1982).

The qualitative predictions of these models for a mixed population including smokers and non-smokers are illustrated in Table 1. The lung cancer risk would be approximately 50% greater in smokers, and only about 10% of these rates in non-smokers, among whom the mesothelioma risk exceeds the lung cancer risk. In relation to environmental exposure, an important prediction is that a higher risk for mesothelioma, but not lung cancer, is caused by early exposure.

The widespread adoption of these or similar models as a basis for asbestos risk assessment has created the false impression that they are reliably established. They were originally proposed on the grounds that they were qualitatively consistent with available data and corresponded to plausible models of carcinogenesis, but both observation and common sense suggest that they are at best useful approximations, and cannot provide reliable predictions far beyond the observed range of dose, age or duration of exposure.

Predictions of the lung cancer model

The model for lung cancer implies that the excess relative risk is: (a) proportional to average duration of exposure for a given level of exposure (fibre/ml); and (b) proportional to average level of exposure at a given duration of exposure. The first of these predictions may be approximately true, although anomalously high risks have sometimes been observed in short-service workers. The second prediction has rarely been examined. Various studies have shown a roughly linear relationship between relative risk and cumulative dose, which increases with duration of exposure and (during employment) with time since exposure. Such a relationship does not, however, necessarily imply that exposures were consistently estimated. Indeed, a cohort in which all exposure levels were

equal, or even one in which different exposure levels were assigned to individual workers at random, would exhibit an apparently consistent increase in risk with increasing cumulative dose by virtue of the correlation between risk and duration of exposure.

The model also implies that the relative risk remains constant after exposure has ceased, but there is evidence from several cohorts that the relative risk eventually falls (Doll & Peto, 1985; Walker, 1984). This eventual decline appears to be more marked for chrysotile than for the amphiboles.

Predictions of the mesothelioma model

The model for mesothelioma was proposed to provide a simple explanation of the observation that mesothelioma incidence continues to increase as the third or fourth power of time since first exposure to asbestos even after exposure has ceased, and is independent of age at exposure (Peto *et al.*, 1982). In spite of its widespread use in risk assessment, however, it has only once been formally fitted in a cohort with individual exposure histories (Peto *et al.*, 1985). This analysis was based on only 10 cases, but it suggested that the risk is lower for shorter exposures and higher for longer exposures than the model predicts.

Relationship to conventional models of carcinogenesis

In terms of conventional notions of multi-stage carcinogenesis, the lung cancer model implies that the rate of the final transition in a multi-stage process is increased in proportion to cumulative dose. Asbestos is thus assumed to act exclusively as a 'promoter' affecting only a late stage in bronchial carcinogenesis. In contrast, the mesothelioma model can be correct only if asbestos acts primarily as an initiator of mesothelial carcinogenesis. There is mounting evidence that many, and perhaps all, 'initiating' carcinogens also act at a later stage in carcinogenesis. A more plausible model may therefore be that the strongest effect happens to be at an early stage for mesothelioma and at a late stage for lung cancer, but that there are both early and late stage effects at both sites. For such a two-stage effect, increases in both exposure level and duration of exposure should cause disproportionate increases in incidence. This cannot be tested adequately, as no accurate data exist on the separate effects of uniform exposure for different durations or equal durations at different measured exposures. The evidence that prolonged exposure causes a disproportionate increase in mesothelioma risk lends some support to a two-stage model, at least for mesothelioma. Cancer incidence increases as a power of duration for various animal carcinogens, and for lung cancer in cigarette smokers.

A further difficulty in formulating any simple model of asbestos carcinogenesis is that inhaled or ingested fibres remain in the body for many years, although they may be chemically and physically altered. They are also progressively dispersed, inactivated or

removed by migration, dissolution, encapsulation or phagocytosis, and the extent of these processes varies with asbestos type and fibre size. The relationship between inhaled and effective dose is therefore likely to be intermediate between that of a chemical carcinogen which is rapidly eliminated and the persistent chronic effect of an increasing cumulative dose. The suggestion that the relative risk for lung cancer eventually falls, and that the reduction is greater for chrysotile than for the amphiboles, is consistent with a late-stage effect modified by progressive elimination of residual fibres. Both human and animal studies suggest that chrysotile is eliminated or inactivated more rapidly than the amphiboles.

In the absence of better evidence, such armchair speculations are of little practical value in formulating quantitative risk assessment models. They may, however, indicate directions for further analysis of existing data. In particular, more detailed analysis of the relationship between mesothelioma risk and duration of exposure in cohorts such as the Quebec chrysotile miners and millers and the Australian crocidolite miners would be informative (Hobbs *et al.*, 1980). Better data on changes over time in lung cancer rates, subdividing observations beyond 20 years after first exposure into 5- or 10-year intervals, could also be obtained from a number of studies.

IMPLICATIONS FOR INDUSTRIAL CONTROL

Many of the uncertainties in these specific models for lung cancer and mesothelioma induction are relatively unimportant in relation to industrial control. Most cohort studies span a wide range of ages at first exposure and durations of exposure, and include workers who have been followed for many years. The only assumption that need be made in relation to these temporal variables is therefore that the patterns observed in the past will persist in the future, and these models provide a convenient device for formalizing this assumption.

The important assumption is that of a linear dose-response relationship, which means that if the exposure level is halved in a particular sector of the industry, the resulting risk will also be halved. Past exposure measurements are so unreliable that this is difficult to test, but most human and animal data on other carcinogens suggest that reductions in exposure level usually produce reductions in risk that are at least proportional, and sometimes greater.

The serious limitation of this common-sense approach to industrial regulation is the absence of any satisfactory measure of exposure level. There may be an approximately linear relationship between average particle and fibre counts within particular sectors of the industry, although this has certainly not been demonstrated; and it may be reasonable to assume that the highest observed dose-specific risk, that based on studies of chrysotile

textile workers, will tend to overestimate the risk caused by other uses of chrysotile. No such assumption can be justified for crocidolite or amosite, however, for which there are no cohorts with measured historical exposures from which dose-specific risks can be calculated. The evidence that the amphiboles, and particularly crocidolite, are more dangerous than chrysotile under similar working conditions may reflect higher fibre levels, a higher risk per fibre, or both.

ENVIRONMENTAL EXPOSURE

The reliability of these models in predicting the effect of environmental exposure to asbestos is very much more dubious, for two reasons. First, the assumption of linear dose-response must be assumed to hold over more than four orders of magnitude of exposure level, from industrial studies of workers exposed at levels of 10 fibre/ ml or more to environmental situations in which average asbestos levels rarely exceed 0.001 fibre/ ml. This difficulty is compounded by differences in the distribution of fibre sizes encountered in environmental and occupational settings. Second, the effects of childhood exposure cannot be predicted. The models described above imply a roughly fourfold increase in risk for mesothelioma, but not for lung cancer, when exposure begins soon after birth rather than at age 20, reflecting the cubic residence time assumption (Table 1). Such an age-related effect would be expected for any carcinogen which initiates the induction of a multi-stage carcinogenic process; but this prediction takes no account of the possibility that children are particularly susceptible to carcinogenesis by virtue of factors such as stem cell expansion during growth and development. The risks caused by exposure in childhood may therefore be substantially greater than those predicted for both mesothelioma and lung cancer.

EXPERIMENTAL EVIDENCE ON THE EFFECTS OF FIBRE LENGTH AND DIAMETER

The evidence that different types of fibre differ in solubility, biological activity and transforming or carcinogenic potential has been discussed in other contributions to this volume. Unfortunately, these experimental differences are unlikely to correlate consistently with the carcinogenic effects of different fibre types in man. Human carcinogenesis occurs over decades rather than days or months, and differences *in vivo* in migration, encapsulation and dissolution over such long periods are difficult to study and impossible to translate into predictions of risk. It may nonetheless be possible to establish a simple relationship between fibre dimension and transforming potency *in vitro* or carcinogenic potency *in vivo* following pleural implantation. Different fibres of similar shape and size may have different

potencies, but it is reasonable to assume, at least provisionally, that the risk will vary with length and diameter in much the same way for all fibre types. The dependence of biological activity on length and diameter is still not well established, but the following hypothesis, which was proposed by Stanton *et al.* (1977), seems consistent with the available data:

(1) Fibres less than 8 μm in length may have little or no transforming or carcinogenic activity, perhaps because they are engulfed by phagocytes following pleural implantation (Stanton *et al.*, 1977). Mesothelioma rates following pleural implantation in rats suggest a threshold between about 4 μm and 8 μm (Stanton el al., 1977, 1981), and inhalation studies in rats (Davis *et al.*, 1986) suggest that fibres shorter than 5 μm rarely or never cause pulmonary tumours or fibrosis.

(2) Above this length, the carcinogenic and/ or transforming potency per fibre (not per unit mass of fibres) increases both with increasing length (see e.g., Stanton *et al.*, 1981) and with increasing diameter (Hesterberg & Barrett, 1984). This last point is not generally appreciated, as the effects of different fibres are usually compared at similar total masses rather than at similar fibre concentrations. Thus, for example, Hesterberg & Barrett (1984) observed that the ratio of the transforming potencies of thin versus thick glass fibres was 20:1 for equal masses, but 1:40 (800 times less) for equal numbers of fibres.

Stanton *et al.* (1977) suggested that the carcinogenic potency of fibres may be proportional to the total surface area that escapes phagocytosis, so that the risk per fibre is very low for fibres less than about 8 μm long, but for long fibres the effect per fibre is proportional to the product of length and diameter. This simple hypothesis has yet to be tested adequately, but it seems at least qualitatively consistent with experiment. In particular, there is no evidence either of an upper limit for length or a lower limit for diameter beyond which such a relationship ceases to hold. This is exactly the opposite of the almost universal belief that a thin fibre is more dangerous than a thick one. The basis of this belief seems to be simply that thin fibres are more dangerous than thick ones per unit mass, and most experiments have been conducted with equal masses of fibres. A moment's reflection reveals the fallacy of assuming the same relationship with diameter for equal fibre concentrations.

CARCINOGENICITY OF MAN-MADE MINERAL FIBRES

Analysis of effects per fibre rather than per unit mass is also relevant to the comparison of asbestos and man-made mineral fibres (MMMF) in the induction of pulmonary tumours by inhalation. The results shown in Table 2, for example, indicate that a similar mass of chrysotile produced a greater tumour incidence than MMMF but that the risk per fibre may have been similar for chrysotile and glass wool, and possibly highest for rock wool, although the numbers are small (Wagner *et al.*, 1984). All groups were exposed to approximately 10 mg/m^3, but the fibre count was more than 10 times higher for chrysotile than for rock wool or glass wool. A separate experiment, again at 10 mg/m^3, in which chrysotile, crocidolite and amosite were compared gave similar results to those shown in the table for chrysotile (8 carcinomas and 7 adenomas in 40 rats), but only 1 mesothelioma and 3 adenomas in 37 rats inhaling crocidolite or amosite (Davis *et al.*, 1978). These differences disappeared when the results were expressed as risks per fibre/ml over 20 μm in length, however, as the fibre count was much higher for chrysotile than for the amphiboles. The low incidence of tumours produced by inhalation for both MMMF and amphiboles may thus be entirely due to the difficulty in achieving high concentrations of long respirable fibres rather than to a lower risk per fibre.

Most MMMFs are thicker than airborne asbestos fibres, and the belief that thick fibres are less carcinogenic than thin ones has made the observation of excess lung cancer among certain MMMF workers exposed to quite low fibre levels seem anomalous (Doll, 1987). If thick fibres are indeed more dangerous than thin ones, however, this anomaly disappears. There is thus a striking and disturbing similarity between the experimental and epidemiological results for MMMF and amphiboles. Both appear less dangerous than chrysotile in experimental carcinogenesis, although these differences may be accounted for by differences in fibre counts; and both are suspected to be more liable than chrysotile to cause lung cancer in humans at the same fibre level, although epidemiological data on their dose-specific effects are still inadequate. This is rather a superficial analogy, as the amphiboles are less liable to dissolution in the lung than chrysotile, while the converse is probably true of many MMMFs. Certain MMMFs may, however, be more carcinogenic than chrysotile in the bronchus, possibly because they are longer and thicker. There is no epidemiological evidence that inhaled MMMF causes an appreciable risk of mesothelioma. Only very fine inhaled asbestos fibres are likely to reach the pleura (Harrington, 1981), and most MMMF may be too thick to migrate extensively.

INGESTION OF ASBESTOS AND GASTROINTESTINAL CANCER

The possibility that asbestos may cause gastrointestinal cancers was first suggested by Selikoff *et al.* (1964), who observed a highly significant excess of stomach and colorectal cancer in a heavily exposed cohort of asbestos workers. Smaller but still statistically significant excesses have been observed in a few subsequent studies, and it has become widely accepted that the effect is real, and is caused by ingestion of asbestos. Some doubt has remained, however, as asbestos has consistently failed to cause any cancer in animal feeding experiments and the excesses observed in humans have been small, with relative risks rarely exceeding 2.

An alternative explanation is that the excess is due largely, or even entirely, to occasional misdiagnosis of mesothelioma and lung cancer, both of which were common in the cohorts showing marked excesses of gastrointestinal cancer (Doll & Peto, 1985). Plots of the relative risks for gastrointestinal cancer (Figure 2a) and all other cancers except lung (Figure 2b) against the relative risk for lung cancer in various studies show marked and similar correlations, the only exception being the hypothesis-generating study (Selikoff *et al.*, 1964), in which there was a large excess of gastrointestinal cancer and no excess at other non-respiratory sites. Subsequent studies thus suggest that the relative risks for cancers of virtually all sites except lung cancer and mesothelioma are increased by more or less the same amount, and that the excess is proportional to, although much less than, the excess of lung cancer. The evidence that gastrointestinal cancers are caused by asbestos is thus neither stronger nor weaker than the evidence that almost all forms of cancer are. The conclusion, albeit tentative, that any excess of gastrointestinal cancer is due largely, and perhaps entirely, to misdiagnosis seems preferable to the only reasonable alternative, that asbestos causes cancer of almost every site. This is an important environmental issue, as many water supplies are contaminated with asbestos released from asbestos-cement pipes or from natural sources, and it has been suggested that this might cause an unacceptable risk of gastrointestinal cancer in the general population supplied with water from these sources.

DISCUSSION

This review has concentrated on the uncertainties inherent in asbestos risk assessment. Certain widespread beliefs, notably that thin fibres are more dangerous than thick ones, and that gastrointestinal cancers are more likely to be caused by asbestos than other non-respiratory cancers, are unsupported or even contradicted by the evidence. The dose-response models generally adopted for lung cancer and mesothelioma are only weakly supported, and certain observations suggest that they are at best only useful approximations. Their predictions should certainly be critically examined in the available cohorts. The

virtual absence of historical exposure measurements for many sectors of the industry is generally recognized; but the weaknesses and internal inconsistencies of the exposure data on the few workplaces in which measurements were taken in the 1940s and 1950s are less well known, if only because most of the data have not appeared in widely accessible publications. Reviews of the evidence have often presented the results of the few studies for which measured exposures are available together with those from the larger number for which exposure levels were guessed on the basis of a handful of historical (or even recent) measurements, conveying the spurious impression of a substantial and varied set of dose-response estimates spanning a range that is likely to include the 'true' value.

In spite of these uncertainties, one practical question has to be addressed. Society must decide when, if ever, the risk from environmental exposure to asbestos or other fibres is high enough to justify the cost, hazard and inconvenience of removing its source. The conventional answer is that the risk assessment calculations shown in Table 1 predict a negligible hazard. Average fibre counts in contaminated buildings, at least in the UK, are usually less than 0.001 fibre/ ml, and the corresponding predicted lifelong risk is of the order of 1 in 100,000 for 10 years' occupancy. The number of people exposed at this sort of level is not known, but some idea of what a risk of 1 in 100,000 means may be conveyed by assuming that a risk of this magnitude is suffered by 1 in 5 of the population. This would imply about one death per year in the UK, or 5 per year in the USA, and corresponds to an average loss of expected life of about 15 minutes. This prediction cannot be tested directly. As McDonald has observed (McDonald *et al.*, 1989), however, data from various countries suggest that the incidence of mesothelioma in those who have no history of any asbestos exposure and do not have unusually high fibre concentrations in their lungs is of the order of 1 per million per year, corresponding to a lifelong risk of about 1 in 10,000. This rate has not increased much, and perhaps not at all, over the last 10 or 20 years, a period in which the incidence due to occupational exposure has risen sharply. The average lifelong risk to the population due to environmental exposure is thus unlikely to be much more than 1 in 10,000, and is probably substantially less.

We thus have an inevitably unreliable risk assessment for chrysotile that suggests a negligible effect of contaminated buildings and a higher, but still very low, upper limit for the overall mesothelioma incidence due to environmental exposure. On this evidence the possibility remains that certain asbestos fibres, and particularly the amphiboles, cause substantially higher individual risks at fibre concentrations of the order of 0.001 fibre/ ml. There is no direct evidence on the dose-specific effects of the amphiboles, but the very high mesothelioma rates that have occurred among Turkish villagers apparently exposed to average erionite levels of the order of 0.01 fibre/ ml (Simonato *et al.*, 1989) suggest that erionite is over 1000 times more potent than chrysotile asbestos. Further studies of these Turkish populations, and particularly case-control studies of fibre concentrations in lung

tissue, are needed to exclude the possibility that heavy occasional exposures rather than ambient levels caused these mesotheliomas; but these results, together with evidence that erionite is an extraordinarily potent carcinogen in animals (Wagner *et al.* 1985), suggest that certain fibre types are so dangerous that no measurable exposure can be regarded as safe. Better understanding of the reasons underlying this remarkable potency is needed to ensure that the dimensions, durability and surface properties of newly introduced synthetic fibres do not produce similar effects.

The pooled observations on MMMF workers suggest an upper limit for the dose-specific effect of certain traditional MMMFs on lung cancer incidence that may well be higher than that for chrysotile; but they also show that most parts of the industry have never suffered the high lung cancer risks that were commonplace in asbestos workers, and mesothelioma has rarely or never been caused (Doll, 1987). MMMFs do not in most applications produce high fibre concentrations, and sensible working practices, combined with a regulated maximum concentration similar to that for chrysotile, should provide acceptable control in the industry. (The maximum chrysotile concentration in the UK is 0.5 fibre/ ml. Since the introduction of this limit, average concentrations rarely exceed 0.25 fibre/ ml and in many sectors are much less.) There is, however, an urgent need for much larger inhalation experiments of the type shown in Table 2. The numbers studied by Wagner *et al.* (1984) were so small that the tumour rates did not differ significantly between the MMMF-exposed groups and the controls; but the results, expressed as risks per unit dose, suggest that some MMMFs may have been at least as carcinogenic as chrysotile asbestos.

In relation to environmental control, my personal view is that the low levels of chrysotile commonly found in most buildings are unlikely to produce lifelong risks much exceeding 1 in 100,000, but that amphiboles and newly introduced synthetic fibres must be regarded more cautiously until the mechanisms of fibre carcinogenicity are better understood. Environmental levels of traditional MMMFs are also usually very low. As for asbestos, however, remedial action may be indicated for friable MMMF on accessible surfaces, which can give rise to relatively high fibre releases (Gaudichet *et al.*, 1989). Those who demand the removal and substitution of all asbestos, irrespective of fibre type or level of contamination, should note that removal can actually increase cumulative doses to both workers and occupants (Burdett *et al.*, 1989), and that substitutes for asbestos may be less innocuous than has generally been assumed. The campaign to eliminate all asbestos on the grounds that 'one fibre can kill', besides being a cost-benefit absurdity, may thus actually increase the risk.

REFERENCES

Burdett, G.J., Jaffrey, S.A.M.T. & Rood, A.P. (1989) Airborne asbestos fibre levels in buildings: A summary of UK measurements. In Non-occupational Exposures to Mineral Fibres, edited by J. Bignon, J. Peto, and R. Saracci (IARC Scientific Publications No. 90), International Agency for Research on Cancer, Lyon, France, 277-290

Dagbert, M. (1976) Etudes de Correlation de Mesures d'Empoussigrage dans l'Industrie de l'Amiante. Document 5 (Beaudry Report), Montreal, Quebec, Comite d'Etude sur la Salubritt dans l'Industrie de l'Amiante

Davis, J.M.G., Beckett, S.T., Bolton, R.E., Collings, P. & Middleton, A.P, (1978) Mass and number of fibres in the pathogenesis of asbestos-related lung disease in rats. Br J. Cancer, 37, 673-688

Davis, J.M.G., Addison, J., Bolton, R.E., Donaldson, K., Jones, A.D. & Smith, T. (1986) The pathogenicity of long versus short fibre samples of amosite asbestos administered to rats by inhalation and intraperitoneal injection. Br. J. Exp. Path., 67, 415-430

Dement, J.M. (1980) Estimation of dose and evaluation of dose-response in a retrospective cohort mortality study of chrysotile asbestos textile workers. (Doctoral dissertation). University of North Carolina

Dement, J.M., Harris, R.L., Symons, M.J. & Shy, C. (1982) Estimates of dose-response for respiratory cancer among chrysotile asbestos workers. Ann. Occup. Hyg., 26, 869-887

Doll, R. (1987) Symposium on Man-made Mineral Fibres, Copenhagen, October 1986. Overview and conclusions. Ann. Occup. Hyg., 31, 805-820

Doll, R. & Peto, J. (1985) Effects on Health of Exposure to Asbestos. A report to the Health and Safety Commission. London, Her Majesty's Stationery Office

Harington, J.S. (1981) Fiber carcinogenesis: epidemiologic observations and the Stanton hypothesis. J. Nail Cancer Inst., 67, 977-989

Hesterberg, T.W. & Barrett, J.C. (1984) Dependence of asbestos and mineral dust-induced transformation of mammalian cells in culture on fiber dimension. Cancer Res., 44, 2170-2180

Hobbs, M.S.T., Woodward, S.D., Murphy, B., Musk, A.W. & Elder, J.E. (1980) The incidence of pneumoconiosis, mesothelioma and other respiratory cancer in men engaged in mining and milling crocidolite in Western Australia. In: Wagner, J.C., ed., Biological Effects of Mineral Fibres, Vol. 2 (IARC Scientific Publications No. 30), Lyon, International Agency for Research on Cancer, pp. 615-625

Gaudichet, A., Petit, G., Billon-Galland, M.A., & Dufour, G. (1989) Levels of atmospheric pollution by man-made mineral fibres in buildings, In Non-occupational Exposures to Mineral Fibres, edited by J. Bignon, J. Peto, and R. Saracci (IARC Scientific Publications No. 90), International Agency for Research on Cancer, Lyon, France, 291-298

McDonald, A.D., Fry, J.S., Woolley, A.J. & McDonald, J.C. (1983) Dust exposure and mortality in an American chrysotile textile plant. Br. J. Ind Med, 40, 361-367

McDonald, J.C., Liddell, F.D.K., Gibbs, G.W., Eyssen, G.E. & McDonald, A.D. (1980) Dust exposure and mortality in chrysotile mining, 1910-75. Br. J. Ind. Med., 37, 11-24

McDonald, J.C., Sebastien, P., McDonald, A.D., & Case, B. (1989) Epidemiological observations on mesothelioma and their implications for non-occupational exposure, In Non-occupational Exposures to Mineral Fibres, edited by J. Bignon, J. Peto, and R. Saracci (IARC Scientific Publications No. 90), International Agency for Research on Cancer, Lyon, France, 420-427

Peto, J. (1978) The hygiene standard for chrysotile asbestos. Lancer, i, 484-489

Peto, J., Seidman, H. & Selikoff, I.J. (1982) Mesothelioma mortality in asbestos workers: implications for models of carcinogenesis and risk assessment. Br. J. Cancer, 45, 124-135

Peto, J., Doll, R., Hermon, C., Binns, W., Clayton, R. & Goffe, T. (1985) Relationship of mortality to measures of environmental asbestos pollution in an asbestos textile factory. Ann. Occup. Hyg., 29, 305-355

Selikoff, I.J., Churg, J. & Hammond, E.C. (1964) Asbestos exposure and neoplasia. J. Am. Med. Ass., 188,22-26

Simonato, L., Barls, R., Saracci, R. Skidmore, J., & Winkelmann, R. (1989) Relation of environmental exposure to erionite fibres to risk of respiratory cancer, In Non-occupational Exposures to Mineral Fibres, edited by J. Bignon, J. Peto, and R. Saracci (IARC Scientific Publications No. 90), International Agency for Research on Cancer, Lyon, France, 398-405

Stanton, M. F., Layard, M., Tegeris, A., Miller, E., May, M. & Kent, E. (1977) Carcinogenicity of fibrous glass: pleural response in the rat in relation to fiber dimension. J. Natl Cancer Inst., 58, 587-603

Stanton, M. F., Layard, M., Tegeris, A., Miller, E., May, M., Morgan, E. & Smith, A. (1981) Relation of particle dimension to carcinogenicity in amphibole asbestos and other fibrous minerals. J. Natl Cancer Inst., 67, 965-975

Wagner, J. C., Berry, G. B., Hill, R. J., Munday, D. E. & Skidmore, J. W. (1984) Animal experiments with MMM(V)F - effects of inhalation and intrapleural inoculation in rats. In: Biological Effects of Man-made Fibres, Vol. 2, Copenhagen, World Health Organization, Regional Office for Europe, pp. 209-233

Wagner, J. C., Skidmore, J. W., Hill, R.C. & Griffiths, D. M. (1985) Erionite exposure and mesothelioma in rats. Br. J. Cancer, 51, 727-730

Walker, A. M. (1984) Declining relative risks for lung cancer after cessation of asbestos exposure. J. Occup. Med., 26, 422-425

Table 1. Predicted excess numbers of deaths before age 80 per 1000 men due to lung cancer or mesothelioma caused by asbestos exposure during working hours at 1.0 fibre/ml

Age at first exposure (years)		Excess deaths per 1000[a]			
	Years exposure:	5	10	20	40
0	Lung cancer	3.7	7.3	14.4	28.4
	Mesothelioma	7.5	13.1	20.1	24.7
20	Lung cancer	3.7	7.4	14.7	27.4
	Mesothelioma	2.1	3.5	5.0	5.6
40	Lung cancer	3.7	7.3	13.2	16.8
	Mesothelioma	0.3	0.5	0.6	0.7

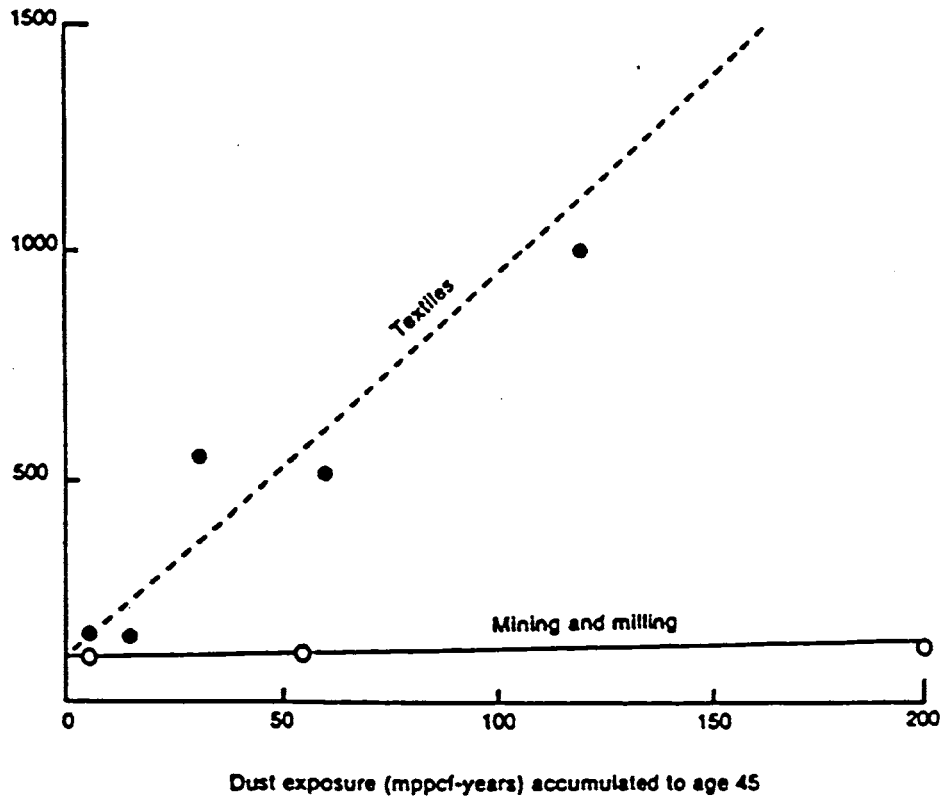
[a] Calculated by extrapolation from observations on English chrysotile textile workers (Peto *et al.*, 1985).

Table 2. Lung tumour induction in rats by chronic inhalation

Material (No. of rats)	Adeno-carcinoma	All neoplasms	Dose (fibre/ ml hours) at 12 months	Tumours per 100 fibre/ ml hours
UICC chrysotile (48)	11	12	656	1.8
Glass microfibre (48)	1	1	223	0.4
Rock wool (48)	0	2	39	5.1
Glass wool with resin (48)	1	1	55	1.8
Glass wool resin-free (47)	0	1	41	2.4
Controls (48)	0	0	-	-

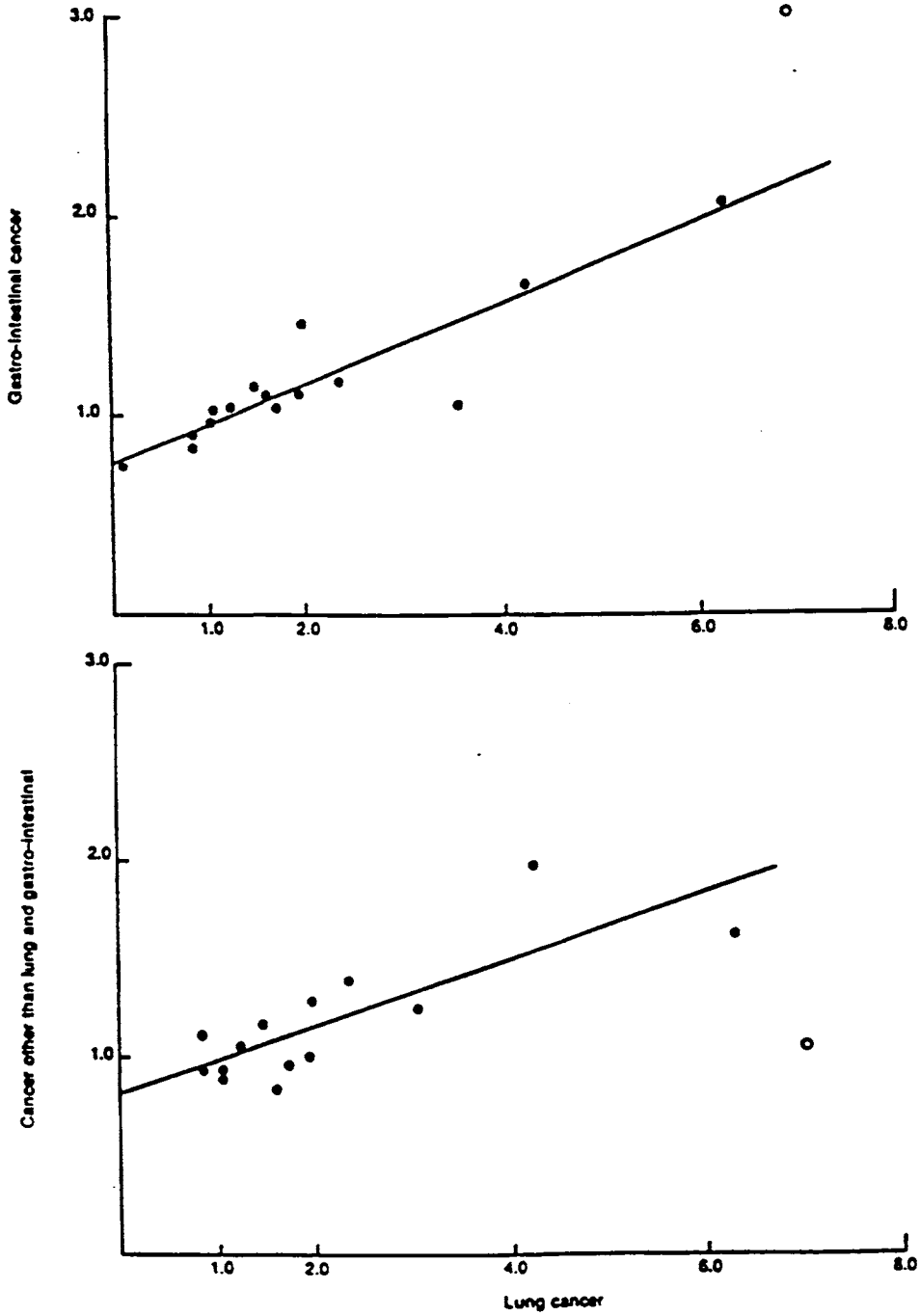
Source: Wagner *et al.* (1984).

Figure 1. Standardized mortality rates for respiratory cancer in relation to dust exposure accumulated to age 45 in chrysotile mining and milling, and in chrysotile textile manufacture.



Source: McDonald *et al.* (1983).

Figure 2. Relative risks for (a) gastrointestinal cancers and (b) cancers other than lung and gastrointestinal plotted against the corresponding relative risks for lung cancer in various cohorts of male asbestos workers.

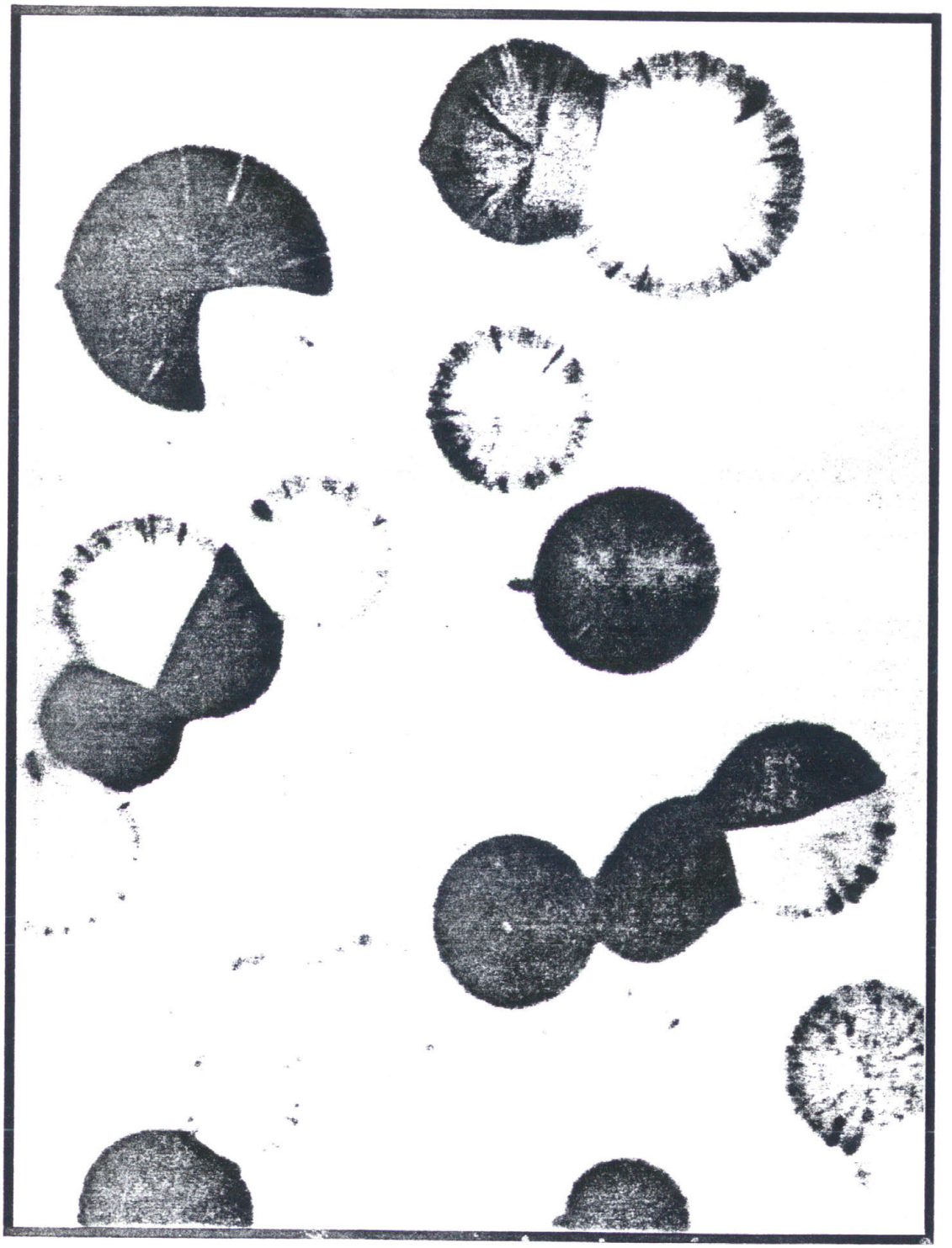


Open circles denote the results reported by Selikoff *et al.* (1964).
Source: Doll and Peto (1985).

American Association for the Advancement of Science

SCIENCE

19 JANUARY 1990 $3.50
VOL. 247 ■ PAGES 257–372



Asbestos: Scientific Developments and Implications for Public Policy

B. T. MOSSMAN, J. BIGNON, M. CORN, A. SEATON, J. B. L. GEE

Asbestos is a commercial term for a group of fibrous minerals often associated with the development of pulmonary interstitial fibrosis (asbestosis), lung cancer, and malignant mesothelioma in occupationally exposed individuals. The pathogenicity of different forms of asbestos varies—long, thin amphibole fibers are most pathogenic, particularly in the induction of mesothelioma. Available data do not support the concept that low-level exposure to asbestos is a health hazard in buildings and schools. The concentration of asbestos fibers in air, type of asbestos, and size of fibers must be considered in evaluation of potential health risks.

ASBESTOS ENGENDERS BOTH FEAR AND PANIC IN U.S. society. Observation that asbestos-containing materials (ACM) have been used in schools, buildings, and hospitals, and the Asbestos Hazard Emergency Response Act (AHERA), a mandate from the Environmental Protection Agency (EPA) that requires inspection of the nation's public and private schools for asbestos, have resulted in the explosive growth of asbestos identification and removal companies. By EPA estimates, extension of EPA requirements to approximately 733,000 public and commercial buildings containing asbestos will cost $53 billion, discounted at 10% over 30 years (*1*). Because of uncertainties regarding the amount of asbestos and its condition in these buildings, estimates for removal of asbestos are as high as $100 to $150 billion (*2*).

Asbestos was shown to cause asbestosis at the turn of the century. Its association with the causation of lung and pleural tumors in asbestos miners and workers was demonstrated in the 1950s and 1960s, respectively (*3*). An important issue is whether these diseases are also hazards to the general population exposed to airborne levels of asbestos in schools and other buildings. Does available evidence support the concept that asbestos causes disease in the nonoccupational environment? What are the mechanisms of asbestos-induced fibrogenesis and carcinogenesis? Most importantly, have recent data been adequately considered in formulating policies in the United States for regulation and banning of asbestos? In this article, we summarize recent developments and discuss their implications for public policy.

B. T. Mossman is a member of the Department of Pathology of the University of Vermont, College of Medicine, Burlington, VT 05405. J. Bignon is director of the Unite INSERM de Recherche sur la Biopathologie et la Toxicologie Pulmonaire et Renale, Creteil, France. M. Corn is director of the Division of Environmental Health Engineering, School of Hygiene and Public Health, Johns Hopkins University, Baltimore, MD 21205. A. Seaton is director of the Institute of Occupational Medicine, Edinburgh, Scotland EH8 9SU. J. B. L. Gee is a director of the Winchester Chest Clinic and member of the Department of Internal Medicine, Yale University School of Medicine, New Haven, CT 06510.

Physico-Chemical Characteristics of Asbestos

"Asbestos" is a broad commercial term for a group of naturally occurring hydrated silicates that crystallize in a fibrous habit. The legal definition of a fiber as promulgated by the EPA and other U.S. regulatory agencies is one that possesses a $\geq 3:1$ aspect ratio. However, this definition has been criticized by mineralogists (*4*).

Asbestos fibers in ores are not respirable until released and made airborne during mining and processing. The family of asbestos minerals can be subdivided into serpentine and amphibole fibers (Fig. 1). Chrysotile, which accounts for over 90% of the world's production of asbestos, is the most common fibrous serpentine, whereas the amphiboles, a chemically diverse group of less industrially important minerals, include the fibrous minerals and crocidolite, amosite, anthophyllite asbestos, actinolite asbestos, and tremolite asbestos. Tremolite, actinolite and anthophyllite, which occur in both fibrous and nonfibrous forms, have been only rarely mined for use as commercial asbestos. Both the fibrous and nonfibrous forms of these amphibole minerals are sometimes found as contaminants of commercial deposits of chrysotile, talc, vermiculite, and other minerals (*4*). The nonfibrous forms of crocidolite and amosite are referred to as riebeckite and grunerite, respectively.

The various types of asbestos fibers differ in their chemical composition, morphology, and durability. Therefore, the biologic effects should be considered individually for each fiber type. Identification of specific types of asbestos in air samples requires sophisticated technology such as transmission electron microscopy, x-ray diffraction, or energy dispersive x-ray spectroscopy. The rod-like amphiboles appear to penetrate the peripheral lung more readily

Table 1. Summary statistics for average airborne fiber concentrations in U.S. schools (*88*) and buildings (*89*). The data used in the calculation of each statistic are the average concentrations (expressed as number of fibers greater than 5 μm in length per cubic centimeter of air) in a building (for indoor samples) or the concentration outside each building [for outdoor samples (*89*)]. By visual inspection, category 1 buildings contained no asbestos-containing material (ACM), category 2 buildings contained ACM in primarily good condition, and buildings in category 3 showed at least one area of significantly damaged ACM. In the study on public buildings, 387 indoor and 48 outdoor air samples were evaluated. No asbestos fibers were detected in 83% of the 387 samples. The sample size is given in parentheses below each heading.

Statistic	Schools (71)	Outdoor air (48)	Public buildings		
			Category 1 (6)	Category 2 (6)	Category 3 (37)
Median		0.00000	0.00010	0.00040	0.00058
Mean	0.00024*	0.00039	0.00099	0.00059	0.00073
SD	0.00053	0.00096	0.00198	0.00052	0.00072

*80th percentile = 0.00045; 90th percentile = 0.00083.

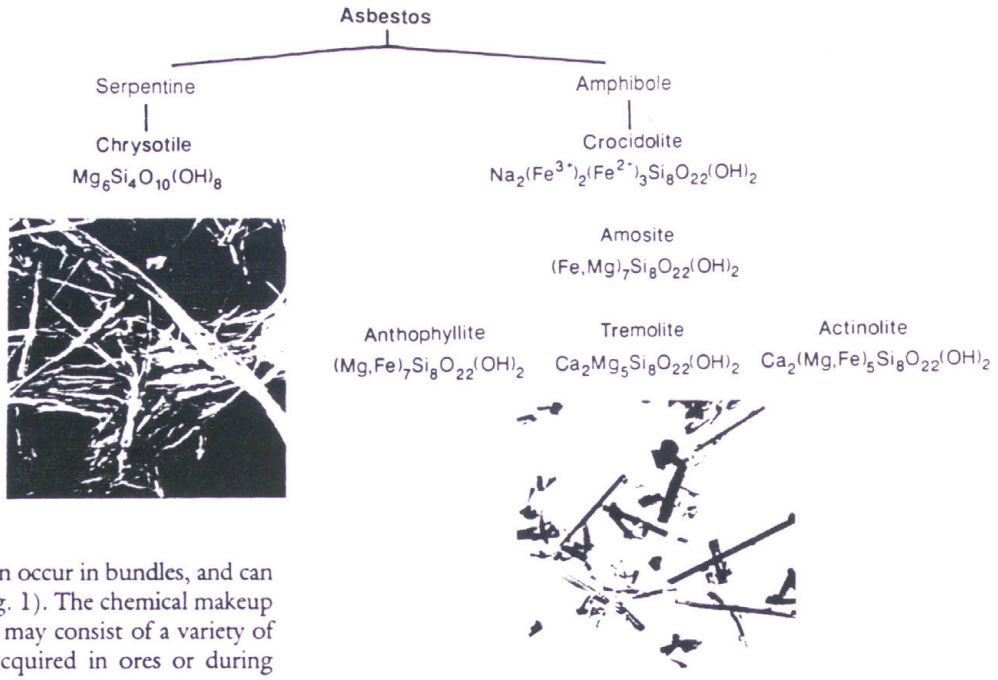


Fig. 1. Classification and morphology of asbestos fibers. The inserted photographs are scanning electron micrographs of Union Internationale contre le Cancer reference samples of chrysotile A (**left**) and crocidolite asbestos (**right**) (field of view is ~12 μm in both photographs). The amphiboles are depicted in order of their relative industrial importance.

than chrysotile fibers, which are curly, can occur in bundles, and can be intercepted at airway bifurcations (Fig. 1). The chemical makeup of each fiber type is complex, and fibers may consist of a variety of trace metals and organic compounds acquired in ores or during processing.

Asbestos is attractive to industry because of its resistance to heat and chemicals, high tensile strength, and lower cost compared to man-made materials. Although use of spray-on asbestos as a fireproofing material or insulation has been banned in this country, as well as in several European countries, asbestos is incorporated currently into cement construction materials (roofing, shingles, and cement pipes), friction materials (brake linings and clutch pads), jointing and gaskets, asphalt coats and sealants, and other similar products. As a result of these applications, an estimated 20% of buildings including hospitals, schools, and other public and private structures contain ACM (*1*). Asbestos in buildings does not spontaneously shed fibers, but physical damage to ACM by decay, renovation, or demolition can cause release of airborne fibers (*5*).

Diseases Associated with Occupational Exposure to Asbestos

Occupational exposure to asbestos can cause four types of disorders: asbestosis; lung cancer; mesotheliomas of the pleura, pericardium, and peritoneum; and benign changes in the pleura (*3*). Asbestosis, a pulmonary interstitial fibrosis with excessive deposition of collagen, caused progressive lung stiffening, impaired gas exchange, disability, and death in many workers exposed before the enforcement of occupational standards. Lung cancers, that is, tumors arising in tracheobronchial epithelial or alveolar epithelial cells, have occurred in asbestos workers in most cases 20 or more years after their first exposure to asbestos. In general, lung cancers have been found in asbestos workers who are smokers and only rarely in nonsmokers (*6*). A number of epidemiologic studies have indicated that the relation between the development of lung cancers and cumulative exposure to asbestos is approximately linear, but wide variations in slope of the line occur apparently related to fiber type and industrial usage (*7*). Death rates from lung cancers in asbestos workers, as measured by standard mortality ratios (SMRs), the observed mortality of a cohort divided by the mortality of a control population, are lowest in chrysotile miners and workers manufacturing friction materials. In contrast, lung cancer deaths are higher in those mining and working with amphibole asbestos. Textile workers in a South Carolina plant in which chrysotile was used exhibited a striking increase in lung cancers with duration of exposure when compared to Canadian chrysotile miners and millers. Solvents and oils in textile production might act as cocarcinogens in the development of these lung tumors (*7*).

Diffuse malignant mesothelioma is a fatal tumor arising from mesothelial cells or underlying mesenchymal cells in the pleura, pericardium, and peritoneum (*8*). The time between diagnosis and initial occupational exposure to asbestos commonly exceeds 30 years. Smoking evidently does not enhance risk of mesothelioma in asbestos workers (*3*). Although mesotheliomas are extremely rare malignancies, that is, only 1648 were recorded from 1973 to 1984 in one survey covering approximately 10% of the U.S. population (*9*), they may account for as much as 18% of the proportional mortality in crocidolite workers (*10*). Mesotheliomas also have been observed after household exposure of family members of asbestos workers and in individuals living in close proximity to asbestos mines (*11*). Although mesothelioma has been considered by some as a disease pathognomonic of exposure to asbestos, approximately 20 to 30% of mesotheliomas occur in the general population in adults not exposed occupationally to asbestos (*12*). Mesotheliomas are rarely found in children.

Diagnosis of mesotheliomas is a challenge as the tumor may resemble metastases of other tumor types occurring in the pleura or peritoneum and assume a wide variety of microscopic appearances. Thus, death certificates may either underestimate (because these tumors are attributed to cancers of the gastrointestinal tract and other organs) (*13*) or overestimate the incidence of mesotheliomas. In France, mesotheliomas are overestimated by a factor of 3 on death certificates in comparison to the mesothelioma registry (*14*).

A number of benign pleural changes that rarely cause functional impairment have been observed in asbestos workers. These include pleural effusions, pleural fibrosis, pleural plaques, that is, accumulations of acellular collagen on the diaphragm and chest wall, and pseudotumors or infoldings of the lung often associated with plaques. These pleural changes may reflect exposure to asbestos but have no demonstrated relation to the development of mesothelioma.

Tumors of the gastrointestinal tract, larynx, and other organs including the kidney, ovary, pancreas, pericardium, eye, and lymphatic system, have been reported in some cohorts of asbestos

workers (*13, 15*). In general, the enhanced SMRs for these tumors are not statistically distinguishable from normal SMRs and have not been confirmed in most cohorts. Both laryngeal and gastrointestinal tumors have other etiologies such as smoking, alcohol, diet, and intestinal polyposis that confound the interpretation of epidemiologic data.

The Amphibole Hypothesis

The association of mesothelioma with asbestos exposure was first described in 1960 in the northwest Cape area of South Africa where long, thin crocidolite fibers were mined (*16*). Since then, an increased incidence of mesothelioma has been reported in a number of occupational settings including factories that presumably used only chrysotile. Within the past decade, sophisticated technology has allowed examination of the types of fibers in the lung tissue of these workers. Results revealed that many chrysotile-exposed workers showed an appreciable lung burden of amphibole fibers, which were used for brief periods in the workplace (*17*). The persistence of amphiboles in human lungs may be attributed to their increased ability to penetrate the peripheral lung, lack of clearance, or durability. In contrast, chrysotile has been found post-mortem in smaller amounts than expected in the lungs of asbestos workers (*18*). It disappears with time most likely because magnesium and silica are leached from the fibers (*19*). Recently, the lung content of asbestos and nonasbestos fibers has been compared in diagnosed cases of mesothelioma, lung cancer, and cardiovascular disease (controls) from the western coast of France, a region containing shipyards (*20*). The number of amphibole fibers (crocidolite and amosite) was significantly higher in lungs from mesothelioma patients, whereas numbers of chrysotile and nonasbestos fibers were similar in all groups. These data suggest that the lung burden of chrysotile and nonasbestos fibers bears no relation to the occurrence of these cancers.

Several recent studies indicate that the risk of pleural mesothelioma is lower where chrysotile is used without admixture or contamination by amphiboles (*21*). For example, a gradation of death rates from mesothelioma has been observed in both male and female asbestos-exposed cohorts. Mesothelioma has been responsible for approximately 6 to 8% of the proportional mortality in men working with mixtures containing crocidolite or amphibole (crocidolite or amosite) in comparison to less than 1% of the proportional mortality in men working with chrysotile (*10*). In female cohorts, the proportional mortality from mesothelioma was highest for amphibole exposure (10.6%) and lowest for chrysotile exposure (0.2%). Thus, these data suggest that amphiboles are the major cause of mesotheliomas in asbestos workers.

Chrysotile miners and millers in Quebec who were supposedly exposed only to chrysotile have developed few mesotheliomas (*22*). However, recent fiber analyses on the lungs of both these workers and chrysotile factory workers showed the presence of tremolite (*23*). This amphibole in the fibrous form has been implicated as the causative agent of mesotheliomas and lung cancers in miners exposed to vermiculite heavily contaminated with tremolite (*24*). Although tremolite composes less than 1% of the asbestos dust in the Quebec mines and mills, the relative ratio of tremolite to chrysotile fibers in the lungs of Canadian miners and millers is related directly to their risk of developing mesothelioma (*25*).

For the reasons above, the few mesotheliomas observed in Canadian chrysotile workers appear to be attributable to fibrous tremolite, an observation compatible with other evidence that amphiboles are the most pathogenic asbestiform minerals. Likewise, recent data on London asbestos factory workers show that the severity of asbestosis and carcinoma of the lung (as well as mesothelioma) correlates with the lung burden of crocidolite and amosite asbestos and that the proportions of chrysotile and nonasbestos fibers are decreased in comparison to matched control patients (*26*). A British cohort exposed since 1970 to chrysotile at airborne levels not exceeding 0.5 to 1.0 fiber per cubic centimeter in the manufacture of friction materials showed no excess of deaths from lung cancer, other asbestos-related tumors, or chronic respiratory disease (*27*). These and other data (*7, 17, 21, 28*) suggest that amphiboles are more potent than chrysotile in the induction of fibrotic lung disease and associated lung cancers.

Experimental Models of Asbestos-Induced Lung Disease

Several studies have shown that mesotheliomas are induced in a dosage-dependent fashion after intrapleural and intraperitoneal injection of asbestos and other asbestos-like fibers into rodents (*29*). Chrysotile was as carcinogenic as the amphiboles by these routes of administration. However, differences have been observed between the carcinogenicity of fibrous and nonfibrous materials. For example, in one study, fibrous tremolite was carcinogenic after intrapleural injection, whereas nonfibrous tremolite was noncarcinogenic at identical concentrations (*30*). Although the natural route of exposure to fibers by inhalation was circumvented in these experiments, they were useful in indicating that fibers longer than 8 μm and less than 0.25 μm in diameter have the most marked carcinogenic potential, that is, the "Stanton hypothesis." These data have been supported by the results of inhalation studies in rats in which short (≤5 μm in length) and long fiber preparations of amosite and chrysotile asbestos have been compared (*31*). In contrast to the batches of amosite and chrysotile asbestos containing many long fibers, short fibers of amosite produced neither asbestosis nor pulmonary tumors. Short chrysotile produced a small amount of asbestosis and malignancies, but these were attributed to contamination of the short chrysotile preparation by longer fibers. Fewer long than short fibers of both types were present in the lungs of all rats at the termination of exposure, but, regardless of size, fewer chrysotile fibers remained in the lung. These results support the observations that chrysotile fibers, in comparison to amphibole fibers, are cleared more rapidly from human lungs (*17*). This phenomenon and limited alveolar penetration of curly chrysotile bundles (rather than their inherent absence of carcinogenicity) may account for the apparent lack of association of chrysotile fibers with the development of mesothelioma in human cohorts.

The exorbitant costs of inhalation experiments with animals preclude long-term studies to determine the carcinogenic potential of asbestos at low-level exposures. The development of malignancies in rodents approaches the 2- to 3-year life-span of these animals (*32*), a period of too brief to reflect the consequences of the long-term solubility of chrysotile in the human lung.

Mechanisms of Asbestos-Induced Inflammation and Fibrogenesis

Both epidemiologic and experimental data support the concept of a threshold for chrysotile-induced pulmonary fibrosis. In a sheep model of asbestosis, inflammation and histopathologic evidence of disease were not observed after less than 100 mg of chrysotile were injected into the trachea of the sheep (*33*). After brief, intense inhalation of chrysotile, the sheep accumulated alveolar macrophages (AMs) at areas of deposition of fibers (*4*). These cell types are

viewed as "effector" cells of disease as they produce a mixture of fibroblast growth factors, chemotactic factors, and fibronectin. Prostaglandins, plasminogen activator, a heat-stable factor similar to platelet-derived growth factor (PDGF), lysosomal enzymes, and active oxygen metabolites, one or more of which may cause proliferation or functional impairment of neighboring epithelial cells and fibroblasts in the lung, were released after exposure of AMs to asbestos in vitro (*35*). These substances might mediate both acute and chronic inflammatory reactions in man and animals after inhalation of asbestos. In support of this hypothesis, AM-derived growth factor (AMDGF), PDGF, superoxide (O_2^-), and H_2O_2 were spontaneously released from AMs recovered by bronchoalveolar lavage from patients with asbestosis (*36*). Similarly, AMs lavaged from both mice and sheep that had an earlier intratracheal injection of chrysotile released enhanced amounts of a growth factor that stimulated proliferation of a human embryonic lung cell line (WI-38) (*37*).

In one study, AMs from both normal individuals and patients with idiopathic pulmonary fibrosis expressed a 4.2-kilobase messenger RNA complementary to c-*sis*, a proto-oncogene coding for the B chain of PDGF (*38*). The amounts were approximately fourfold higher from AMs of patients with pulmonary fibrosis. Because PDGF is mitogenic to mesenchymal cells, which possess functional PDGF receptors, elevated levels of PDGF in lung tissue or fluids could induce lung fibroblasts to divide or to produce exorbitant amounts of collagen, the hallmark of the fibrotic lesion. Quiescent human mesothelial cells also undergo DNA synthesis after exposure to PDGF and a broad spectrum of other growth factors (*39*).

Within the past few years, several laboratories have focused on active oxygen species (AOS) as causative agents of both asbestosis and asbestos-related malignancies. Increased amounts of superoxide (O_2^-) have been produced after rodent AMs were exposed in vitro to long asbestos fibers, whereas generation was minimal after shorter fibers and nonfibrous particles were introduced (*40*). Smaller fibers and particles are incorporated into phagolysosomes by AMs, whereas longer fibers are incompletely phagocytosed, a process liberating more AOS.

The observation that exogenous administration of scavengers of AOS prevents asbestos-induced cell death to cultures of tracheal epithelial cells and lung fibroblasts (*41*) suggests that AOS are intimately related to asbestos toxicity even in the absence of AMs. Fibers may induce generation of AOS after phagocytosis or by extracellular mechanisms. For example, recent studies with asbestos in cell-free systems have demonstrated by electron spin resonance that chrysotile, crocidolite, and amosite generate AOS in the presence of H_2O_2 or physiological saline (*42*). Under these circumstances, Fe^{2+} on the surface of the fiber appears to drive a modified Haber-Weiss (Fenton) reaction that results in production of the toxic hydroxyl radical (OH^-) from H_2O_2 and O_2^-. These reactions result in lipid peroxidation, which is prevented by incubation of asbestos with the iron chelator, desferroxamine (*43*).

At high concentrations, AOS are cytotoxic to cells of the respiratory tract, but at low concentrations they induce functional changes in rodent lung fibroblasts that may be critical to the pathogenesis of asbestos-induced fibrotic lung disease. For example, after addition of xanthine and xanthine oxidase (a chemical generating system producing O_2^-), rat lung fibroblasts in vitro produced increased amounts of cell-associated collagen in a pattern similar to that observed after their exposure to crocidolite asbestos (*40, 44*). In an inhalation model of rapid-onset asbestosis, osmotic pumps containing polyethylene glycol (PEG)–conjugated catalase, the enzyme scavenging H_2O_2, were implanted subcutaneously into rats before they were exposed to crocidolite for 20 days (*45*). This procedure boosted levels of catalase in the sera and lungs of these animals and ameliorated both the inflammation and the severity and extent of fibrotic lesions that normally develop after inhalation of asbestos. This study was the first successful experimental approach to the prevention of asbestos-associated lung disease. Moreover, the results support the concept of a cause and effect relation between AOS and the development of asbestosis.

Mechanisms of Asbestos-Induced Carcinogenesis

Carcinogenesis is a multistage process that classically has been described in two stages (*46*). The "initiation" stage corresponds to a heritable genetic change (point mutation) induced in a cell by a carcinogenic substance. It is followed by the "promotion" stage, a series of events in which the initiated cell undergoes proliferative and genotypic changes conferring the malignant phenotype. During the past few years, the identification of a number of proto-oncogenes has resulted in a new understanding of the successive genetic events involved in the process of malignant transformation. Increased expression of these genes may cause the production of growth factors or growth-factor receptors. Loss of other genes (anti-oncogenes) also appears to contribute to the carcinogenic process. These findings indicate that the distinction between genetic and epigenetic events in carcinogenesis is not simple, especially because chromosomal rearrangements or deletions associated with point mutation and activation or loss of genes can happen at any stage in the process of cell transformation.

Whether the multistage model is directly applicable to asbestos-associated carcinogenesis is unclear. Unlike most carcinogens, asbestos does not cause base substitution and frameshift mutations in bacterial-mutation assays (*47*). Of the 23 agents designated as group 1 human carcinogens by the International Agency for Research on Cancer (IARC), only asbestos and conjugated estrogens were nongenotoxic as defined by both the Ames test and rodent bone-marrow assays for detection of chromosomal aberrations or micronucleated erythrocytes (*48*). Although asbestos was weakly mutagenic in Chinese hamster lung cells (*49*), it was not mutagenic in liver epithelial cells or in Syrian hamster embryo (SHE) fibroblasts (*50*). Asbestos did not cause morphologic transformation of C3H 10T1/2 cells (*51*), but transformed both BALB/c#3T3 and SHE fibroblasts (*52*). Glass fibers and nonfibrous silica (albeit at much higher concentrations) also were active in the SHE bioassay. In this system, longer, thinner fibers were more potent in the induction of transformation and chromosomal anomalies, an observation consistent with the increased malignant potential of these fibers in comparison to shorter fibers or particles after their administration intrapleurally, intraperitoneally, or by inhalation to rodents (*29, 30*).

In these and other in vitro studies, the biologic effects of fiber types have been assessed comparatively on a mass (milligrams of fibers per dish) rather than a numerical (numbers of fibers of a given size per dish) basis. Cytotoxicity and cytogenetic effects of chrysotile, crocidolite, and erionite (an aluminosilicate fiber) recently were compared in Chinese hamster lung fibroblasts (V79 cells) (*53*). Numbers of chrysotile fibers required to produce cytotoxic or cytogenetic changes were several orders of magnitudes higher in comparison to erionite, the most potent fiber, or crocidolite, a fiber of intermediate potency. These results are consistent with the higher tumorigenic potential of erionite in rodent inhalation experiments (*54*).

In some studies, asbestos appears to augment the mutagenic and carcinogenic effects of chemical carcinogens and radiation. For example, both crocidolite and chrysotile increased the frequency of mutation and transformation in rodent epithelial cells and fibroblasts exposed to benzo[*a*]pyrene (BaP) (*50*) and radiation or radon

alpha particles (*51*). However, synergistic effects of asbestos and BaP were not observed in two studies with SHE and rat mesothelial cells, respectively (*52, 55*).

The particulate nature of asbestos and its capacity to bind nucleic acids has prompted transfection studies in which asbestos was used as a vehicle for introducing DNA or RNA into a number of cell lines (*56*). Under these circumstances, asbestos was intermediate in rank in comparison to a number of other insoluble facilitators including calcium phosphate, talc, and kaolin, none of which have been associated with the induction of cancer.

After addition to human or rat mesothelial cells, both chrysotile (in rats) (*57*) and amosite (in humans) (*58*) have caused aneuploidy and altered growth characteristics after repeated passaging. Injection of rat mesothelial cells into nude mice after a single exposure to chrysotile did not cause tumors in animals, but multiple exposures (36 times) to chrysotile and repeated passaging resulted in tumorigenic cell populations (*57*). In contrast, human mesothelial cells displaying chromosomal abnormalities and growth alterations after duplicate exposures to cytotoxic concentrations of amosite were not tumorigenic in nude mice (*58*). Asbestos promoted the proliferation of mesothelial cells both in organ cultures of human mesothelium exposed to asbestos in vitro and in mice given intraperitoneal injections of asbestos (*59*).

Asbestos fibers come in contact with the chromosomes of rat mesothelial cells (*60*) and the mitotic apparatus of V79 (*53*) and SHE (*61*) cells in vitro. These interactions might induce chromosomal misaggregation or abnormalities. Several investigators have examined chromosomal aberrations in human mesotheliomas, but changes appeared inconsistent from tumor to tumor. The most common abnormalities involved inversions, translocations, and deletions of chromosomes 1, 3, 7, 9, 17, and 22 (*62*). Constitutively enhanced expression of the PDGF-B gene, the proto-oncogene c-*sis*, was observed in human mesothelioma cell lines when compared to normal human mesothelial cells (*63*).

In comparison to human mesothelial cells, human bronchial epithelial cells in vitro are relatively resistant to asbestos. In one study, concentrations of chrysotile, crocidolite, or amosite asbestos approximately ten times as high as that required for mesothelial cells were required to achieve a comparable increase in toxicity (as measured by a 50% decrease in colony-forming efficiency of human bronchial epithelial cells) (*64*). In another study, aneuploidy was not increased significantly over a range of concentrations of either crocidolite or chrysotile asbestos (*65*). This latter observation and the demonstration that insertion of asbestos into rat tracheal grafts can cause the development of carcinomas following insertion of subcarcinogenic amounts of the polycyclic aromatic hydrocarbon, dimethylbenzo[*a*]anthracene (*66*), suggest that asbestos is a promoter in the development of lung cancers. In support of this concept, both crocidolite and chrysotile asbestos induced a number of biochemical and proliferative alterations in both rodent and human tracheal epithelial cell and organ cultures that were similar to those observed in mouse skin that had been treated with the tumor promoter 12-O-tetradecanoylphorbol-13-acetate (TPA) (*67*). The repertoire of these asbestos-associated proliferative changes, which were masked in tracheal epithelial cells in a high-serum containing medium or by addition of transforming growth factor–type B (TGF-β_1) (*68*), included enhanced incorporation of ^{3}H-thymidine, increases in colony-forming efficiency, and the development of squamous metaplasia, that is, conversion of differentiated mucociliary cells to keratinizing cells resembling epidermis. Induction of ornithine decarboxylase (ODC), a rate-limiting enzyme in the biosynthesis of polyamines that is increased in mouse skin after exposure to TPA but not after addition of nontumor promoting phorbol derivatives, also occurred in a dosage-dependent fashion in tracheal epithelial cells exposed to long, thin, asbestos and glass fibers. In contrast, nonfibrous particles and shorter fibers did not increase ODC activity at similar concentrations (*69*).

Until quite recently, it was unclear how asbestos triggered proliferation in tracheal epithelial cells. However, several pieces of data suggest that mechanisms of cell signaling by asbestos are similar to those observed with TPA, a soluble tumor promoter that binds directly to protein kinase C (PKC), a calcium- and phospholipid-dependent enzyme that activates a limb of the phosphoinositide signal-transduction pathway (*70*). Mitogenic concentrations of crocidolite asbestos caused increased accumulation of diacylglycerol in tracheal epithelial cells (*71*) and subsequent activation of PKC (*72*), presumably by activation of membrane phospholipases. The increased production of inositol tris- and tetrakisphosphates appeared responsible for the generation of diacylglycerol, which preceded increased cell division. Abrogation of crocidolite-induced ODC activity in tracheal epithelial cells by inhibitors of PKC and calcium channel antagonists (*69*) suggests that PKC is related causally to asbestos-associated cell proliferation.

Mechanisms other than tumor promotion by asbestos also may explain interactions between smoking and asbestos; these mechanisms could be important in the development of lung cancers in asbestos workers (*7, 73*). For example, smoking impaired clearance of amosite asbestos from rodent lungs and increased retention of fibers in airway epithelial cells (*74*). Both cigarette smoke and asbestos induced AOS in a synergistic fashion in vitro and damaged isolated bacteriophage DNA (*75*). AOS liberated from asbestos fibers also catalyzed the oxidation of 6-hydroxybenzo[*a*]pyrene to a more mutagenic and carcinogenic radical (*76*). Because crocidolite and chrysotile asbestos adsorbed BaP and acted as vehicles to increase both uptake of these lipophilic carcinogens and formation of DNA adducts in tracheal epithelial cells (*77*), fibers might facilitate the initiation of lung tumors by BaP.

Public Policy

The available experimental and epidemiological data indicate that both fiber type and size are important determinants of the pathogenicity of asbestos. Although asbestos has caused disease in the workplace (*78*) and such occurrence has resulted in calls for regulations to protect workers (*79*), recent epidemiologic data are concordant with the suggestion that exposure to chrysotile at current occupational standards does not increase the risk of asbestos-associated diseases (*17, 21, 27, 28*). Unlike most other countries, particularly in the European community, which have more stringent requirements for regulation and importation of amphiboles, federal policy in the United States does not differentiate between different types of asbestos.

Does airborne asbestos present a risk to the health of individuals in schools and other buildings? The available data do not indicate that asbestos-associated malignancies or functional impairment will occur as a result of exposure to most airborne concentrations of asbestos in buildings. First and foremost, the levels of airborne asbestos in buildings, even with damaged ACM, are magnitudes lower than concentrations in the unregulated workplace in the past and approximately 1/100 of the permissible exposure of 0.2 fibers per cubic centimeter of air in the U.S. workplace (*80*). Before the enforcement of occupational standards, workplace concentrations of 100 or more fibers per cubic centimeter of air were not uncommon (*81*). In contrast, surveys of asbestos in schools and public buildings show that the mean airborne concentrations are several thousand–fold lower (Table 1). With few exceptions, the type of asbestos fiber found predominantly in buildings is chrysotile. Accumulating evi-

dence indicates that this asbestos type is probably not associated with the occurrence of mesotheliomas at low levels of exposure. For example, recent analyses on the fiber concentrations in lungs of asbestos workers showed that chrysotile workers with mesothelioma had 400 times the median lung fiber burden in comparison to workers exposed to amphiboles. Data indicate that mesotheliomas in chrysotile workers appeared at lung burdens comparable to that required for the development of asbestosis, a disease associated with occupational exposure to asbestos in the past unregulated workplace (*82*).

Transmission electron microscopy of air samples is essential for the identification and quantitation of finer asbestos fibers. In the United States and United Kingdom, the direct transmission electron microscopy method is advocated to determine airborne asbestos fiber concentrations in buildings. In France, the indirect transmission electron microscopy technique is used, and concentrations are expressed on a mass (milligram) basis. The limit for detection of fibers by phase-contrast microscopy is approximately 0.01 fibers per cubic centimeter of air, a concentration higher than that reported in most schools and buildings (Table 1). Moreover, phase-contrast microscopy cannot be used to identify types of fibers (asbestos or nonasbestos) or to detect fibers less than 0.5 μm in diameter, twice the diameter of fibers associated with the greatest biological activity and induction of tumors in rodents (diameters ≤0.25 μm, that is, Stanton fibers) (*29, 30*). Such long, thin asbestos fibers are rarely found in air samples of buildings (*81, 83*). As shown in Table 1, fiber concentrations from recent studies in buildings are comparable to levels in outdoor air, a point surely relevant to assessing the health risks of asbestos in buildings. Airborne concentrations of asbestos in buildings reported in the 1970s were somewhat higher, presumably because of earlier, less sophisticated sampling and analytical techniques.

Recent epidemiologic studies of deaths from mesothelioma in the general population also suggest that risk from asbestos in buildings is miniscule (*9, 10, 84*). In comparison to lung cancers (an average of 130,000 cases per year in the United States, largely attributed to smoking), an estimated 1,500 cases of mesothelioma per year occur in the U.S. population (*85*). The data on death rates from pleural or peritoneal mesotheliomas over the past 10 to 20 years indicate that mesotheliomas are increasing in males over 65 years of age who have a past occupational history of exposure to asbestos (*84*). By contrast, death rates from mesothelioma in females of all ages have declined slightly or remained constant. These results support the concept that asbestos in buildings is not an important risk factor, as one would expect increased mesotheliomas in both males and females in this case. A recent French study did not show increased risks of asbestos-associated malignancies, pleural plaques, or functional impairment of the lung (effects clearly present in asbestos workers) in persons exposed for 10 years to airborne asbestos in buildings (*86*). Although this survey is still in progress, no mesotheliomas have been observed to date among approximately 15,000 permanent occupants.

Although the validity of extrapolating from high to low dose levels has never been confirmed empirically in the evaluation of asbestos, calculated lifetime risks from mesotheliomas and lung cancers attributable to asbestos in schools and other buildings have appeared in recent years (*85, 87*). The linear dose-response equations in these models have been used with the assumption that there is no threshold for disease, a hypothesis which is open to question. Moreover, the range of estimated risks varies from study to study. With the exception of one analysis (*85*), differences between the pathogenic potential of chrysotiles and amphiboles have not been considered in these assessments, and the importance of fiber size has been ignored. Regardless, examination of combined data from published risk estimates shows that risks of asbestos-related total deaths (both lung cancers and mesotheliomas) due to exposure in schools are magnitudes lower than commonplace risks in modern-day society (Table 2).

Table 2. Estimates of risk from asbestos exposure in schools in comparison to other risks in U.S. society. Data from six published risk estimates (*87*) in which total deaths (lung cancer and mesotheliomas) attributable to asbestos exposure over a lifetime were estimated per 1 million students exposed to 0.00024 fibers per cubic centimeter air (the mean airborne concentration in schools, Table 1) for five school years, beginning at age 10. Estimates indicate that the annual rate is 0.005 to 0.093 deaths per million students for an average life expectancy of 75 years. Modified with permission from Weill and Hughes (*90*).

Cause	Annual rate (deaths per million)
Asbestos exposure in schools	0.005 to 0.093
Whooping cough vaccination (1970 to 1980)	1 to 6
Aircraft accidents (1979)	6
High school football (1970 to 1980)	10
Drowning (ages 5 to 14)	27
Motor vehicle accident, pedestrian (ages 5 to 14)	32
Home accidents (ages 1 to 14)	60
Long-term smoking	1200

The AHERA ruling of 1986 brought asbestos to the attention of the U.S. public and instilled fears in parents that their children would contract asbestos-related malignancies because of high levels of airborne asbestos fibers in schools. Panic has been fueled by unsupported concepts such as the "one fiber theory," which maintains that one fiber of inhaled asbestos will cause cancer. As a result of public pressure, asbestos often is removed haphazardly from schools and public buildings even though most damaged ACM is in boiler rooms and other areas which are inaccessible to students or residents (*1*). The removal of previously undamaged or encapsulated asbestos can lead to increases in airborne concentrations of fibers in buildings, sometimes for months afterwards (*83*), and can result in problems with safe removal and disposal. Asbestos abatement also has led to the exposure of a large new cohort of relatively young asbestos removal workers. While these people should be protected by careful regulation of the circumstances of removal, they are often exposed under suboptimal working conditions.

As a result of the AHERA ruling, public and private schools are required to inspect for asbestos and inform parents if ACM are present. Although the law does not require or set standards for the removal of asbestos, schools, often with little expert advice, must submit a management plan detailing how they will deal with damaged asbestos and can be fined a maximum of $5000 per day for lack of compliance to deadlines. The EPA has recommended bulk sampling of ACM to determine the presence of asbestos and visual inspection to determine the course of action, rather than measurement of airborne levels of fibers—data that are far more important in determining the need, if any, for removal of ACM.

The available data and comparative risk assessments (Table 2) indicate that chrysotile asbestos, the type of fiber found predominantly in U.S. schools and buildings, is not a health risk in the nonoccupational environment. Clearly, the asbestos panic in the U.S. must be curtailed, especially because unwarranted and poorly controlled asbestos abatement results in unnecessary risks to young removal workers who may develop asbestos-related cancers in later decades. The extensive removal of asbestos has occurred less frequently in Europe.

Prevention (especially in adolescents) of tobacco smoking, the principal cause of lung cancer in the general population, is both a more promising and rational approach to eliminating lung tumors

than asbestos abatement. Even acknowledging that brief, intense exposures to asbestos might occur in custodians and service workers in buildings with severely damaged ACM, worker education and building maintenance will prove far more effective in risk prevention for these workers.

REFERENCES AND NOTES

1. Report to the Congress, *Study of Asbestos-Containing Materials in Public Buildings* (U.S. Environmental Protection Agency, Washington, DC, February 1988), p. 5.
2. M. Corn, paper presented at the 22nd International Congress on Occupational Health, Sidney, Australia, September, 1986.
3. B. T. Mossman and J. B. L. Gee, *N. Engl. J. Med.* **320**, 1721 (1989).
4. H. C. W. Skinner, M. Ross, C. Frondel, Eds., *Asbestos and Other Fibrous Materials* (Oxford Univ. Press, New York, 1988).
5. M. Corn, *Am. Ind. Hyg. Assoc. J.* **47**, 515 (1986).
6. R. Saracci, *Epid. Rev.* **9**, 175 (1987).
7. J. C. McDonald and A. D. McDonald, in *Asbestos-Related Malignancy*, K. H. Antman and J. Aisner, Eds. (Grune and Stratton, Orlando, 1987), pp. 57–79.
8. J. Chretien, J. Bignon, A. Hirsch, Eds., *The Pleura in Health and Disease* (Dekker, New York, 1985).
9. R. R. Connelly, R. Spirtas, M. H. Meyers, C. L. Percy, J. F. Fraumeni, *J. Natl. Cancer Inst.* **79**, 31 (1987).
10. A. D. McDonald and J. C. McDonald, in *Asbestos-Related Malignancy*, K. H. Antman and J. Aisner, Eds. (Grune and Stratton, Orlando, 1987), pp. 31–55.
11. H. A. Anderson, R. Lilis, S. M. Daum, A. S. Fishbein, I. J. Selikoff, *Ann. N.Y. Acad. Sci.* **271**, 311 (1976).
12. A. Hirsh *et al.*, *Am. J. Ind. Med.* **3**, 413 (1982).
13. R. Doll and J. Peto, in *Asbestos-Related Malignancy*, K. H. Antman and J. Aisner, Eds. (Grune and Stratton, Orlando, 1987), pp. 81–96.
14. J. Bignon, P. Sebastien, L. DiMenza, H. Payan, *Ann. N.Y. Acad. Sci.* **330**, 455 (1979).
15. D. A. Edelman, *Br. J. Ind. Med.* **45**, 75 (1988); C. K. Chan and J. B. L. Gee, *J. Occup. Med.* **30**, 23 (1988).
16. J. C. Wagner, C. A. Sleggs, P. Marchand, *Br. J. Ind. Med.* **17**, 260 (1960); G. K. Sluis-Cremer, *Ann. N.Y. Acad. Sci.* **132**, 215 (1965).
17. J. C. Wagner, G. Berry, F. D. Pooley, *Br. Med. J.* **285**, 603 (1982); M. J. Gardner, P. D. Winter, B. Pannett, C. A. Powell, *Br. J. Ind. Med.* **43**, 726 (1986); A. Churg, *Chest* **93**, 621 (1988); A. M. Langer and R. P. Nolan, in *Non-Occupational Exposure to Mineral Fibres*, J. Bignon, J. Peto, R. Saracci, Eds. (International Agency for Research on Cancer, Lyon, 1989), pp. 330–335.
18. J. C. Wagner *et al.*, *Ann. Occup. Hyg.* **26**, 423 (1982).
19. M. C. Jaurand, J. Bignon, P. Sebastien, J. Goni, *Environ. Res.* **14**, 245 (1977); A. Morgan and A. Holmes, *ibid.* **39**, 475 (1986).
20. A. Gaudichet *et al.*, *ibid.* **32** (suppl.), 213 (1988).
21. J. Dement, R. L. Harris, M. J. Symons, C. M. Shy, *Am. J. Ind. Med.* **4**, 421 (1983); M. Finkelstein, *Am. Rev. Respir. Dis.* **129**, 754 (1984); J. M. Hughes *et al.*, *Br. J. Ind. Med.* **66**, 161 (1987); A. D. McDonald, J. S. Fry, A. J. Woolley, J. C. McDonald, *ibid.* **40**, 368 (1983); *ibid.*, p. 361; C. G. Ohlson and C. Hagstedt, *ibid.* **42**, 397 (1985).
22. A. Churg, *Chest* **93**, 621 (1988).
23. P. Sebastien, J. C. McDonald, A. D. McDonald, B. Case, R. Hartley, *Br. J. Ind. Med.* **46**, 180 (1989).
24. J. C. McDonald *et al.*, *ibid.* **43**, 436 (1986).
25. A. Churg, B. Wiggs, L. Depaoli, B. Kampe, B. Stevens, *Am. Rev. Respir. Dis.* **130**, 1042 (1984).
26. J. C. Wagner, M. L. Newhouse, B. Corrin, C. E. R. Rossiter, D. M. Griffiths, *Br. J. Ind. Med.* **45**, 305 (1988).
27. M. L. Newhouse and S. R. Sullivan, *ibid.* **46**, 176 (1989).
28. G. Berry and M. L. Newhouse, *ibid.* **40**, 1 (1983); H. F. Thomas, I. I. Benjamin, P. C. Elwood, P. M. Sweetnam, *Br. J. Ind. Med.* **39**, 273 (1982).
29. J. M. G. Davis, in *Proceedings of 5th International Colloquium on Dust Measuring Technique and Strategy* (Asbestos International Association, Johannesburg, 1985), pp. 25–35; F. Pott and K. H. Friedrichs, *Naturwissenschaften* **59**, 318 (1972); M. F. Stanton and C. Wrench, *Br. J. Cancer* **48**, 797 (1972); M. C. Jaurand, J. Fleury, G. Monchaux, M. Nebut, J. Bignon, *J. Natl. Cancer Inst.* **79**, 797 (1987).
30. W. E. Smith, D. D. Hubert, H. J. Sobel, E. Marquet, in *Dusts and Disease*, R. Lemen and J. M. Dement, Eds. (Pathotox, Park Forest, IL, 1979), pp. 335–339.
31. J. M. G. Davis *et al.*, *Br. J. Exp. Pathol.* **67**, 415 (1986); J. M. G. Davis in *Non-Occupational Exposure to Mineral Fibers*, J. Bignon, J. Peto, R. Saracci, Eds. (International Agency for Research on Cancer, Lyon, 1989), pp. 33–45.
32. J. C. Wagner, G. Berry, J. W. Skidmore, V. Timbrell, *Br. J. Cancer* **29**, 252 (1974).
33. R. Begin, S. Masse, M. Rola-Pleszczynski, M. Boctor, G. Drapeau, in *Asbestos Toxicity*, G. L. Fisher and M. A. Gallo, Eds. (Dekker, New York, 1987), pp. 87–107.
34. A. R. Brody, L. M. Hill, B. Adkins, R. W. O'Connor, *Am. Rev. Respir Dis.* **123**, 670 (1981).
35. R. T. Mossman and R. Begin, Eds., *Effects of Mineral Dusts on Cells* (North Atlantic Treaty Organization, Advanced Science Institute Series H, vol. 30) (Springer-Verlag, Berlin, 1989).
36. W. N. Rom, P. B. Bitterman, S. I. Rennard, A. Cantin, R. G. Crystal, *Am. Rev. Respir. Dis.* **136**, 1424 (1987).
37. I. Lemaire, H. Beudoin, S. Masse, S. Grondin, *Am. J. Pathol.* **122**, 205 (1986).
38. J. J. Mornex *et al.*, *J. Clin. Invest* **78**, 61 (1986).
39. E. W. Gabrielson *et al.*, *FASEB J.* **2**, 2717 (1988); E. M. Laveck, A. N. A. Somers, L. L. Moore, B. I. Gerwin, J. F. Lechner, *In Vitro Cell Dev. Biol.* **24**, 1077 (1988).
40. K. Hansen and B. T. Mossman, *Cancer Res.* **47**, 1681 (1987); B. T. Mossman, K. Hansen, J. P. Marsh, M. E. Brew, J. Petruska, in *Non-Occupational Exposure to Mineral Fibres*, J. Bignon, J. Peto, R. Saracci, Eds. (International Agency for Research on Cancer, Lyon, 1989), pp. 81–92.
41. B. T. Mossman, J. P. Marsh, M. A. Shatos, *Lab. Invest.* **54**, 204 (1986); M. A. Shatos, J. P. Marsh, B. T. Mossman, *Environ. Res.* **44**, 103 (1987).
42. S. A. Weitzman and P. Graceffa, *Arch. Biochem. Biophys.* **228**, 373 (1984); R. Zalma, L. Bonneau, M. C. Jaurand, J. Guignard, H. Pezerat, *Can. J. Chem.* **65**, 2338 (1987).
43. S. A. Weitzman and A. B. Weitberg, *Biochem. J.* **225**, 259 (1985).
44. B. T. Mossman *et al.*, *Chest* **89**, 160 (1986).
45. B. T. Mossman *et al.*, *J. Free Rad. Biol. Med.* **2**, 335 (1986); B. T. Mossman *et al.*, *Am. Rev. Respir. Dis.*, in press.
46. I. Berenblum, *Cancer Res.* **1**, 44 (1941).
47. M. Chamberlain and E. M. Tarmy, *Mutat. Res.* **43**, 159 (1977); W. G. Light and E. T. Wei, in *The In Vitro Effects of Mineral Dust*, R. C. Brown, I. P. Gormley, M. Chamberlain, R. Davies, Eds. (Academic Press, London, 1980), pp. 139–145.
48. M. D. Shelby, *Mutat. Res.* **204**, 3 (1988).
49. S. L. Huang, *ibid.* **68**, 265 (1979).
50. B. Reiss, C. Tong, S. Telany, G. M. Williams, *Environ. Res.* **31**, 100 (1983); M. Oshimura, T. W. Hesterberg, T. Tsutsui, J. C. Barrett, *Cancer Res.* **44**, 5017 (1984); J. A. DiPaolo, A. J. DeMarinis, J. Doniger, *Pharmacology* **27**, 65 (1983).
51. R. C. Brown, A. Poole, G. T. A. Fleming, *Cancer Lett.* **18**, 221 (1988); T. K. Hei, C. R. Geard, R. S. Osmak, M. Travisano, *Br. J. Cancer* **52**, 591 (1985); T. K. Hei, in *Effects of Mineral Dusts on Cells*, B. T. Mossman and R. Begin, Eds. (North Atlantic Treaty Organization, Advanced Science Institute Series H, vol. 30) (Springer-Verlag, Berlin, 1989).
52. T. W. Hesterberg and J. C. Barrett, *Cancer Res.* **44**, 2170 (1984); Y. P. Lu, C. Lasne, R. Lowy, I. Chouroulinkov, *Mutagenesis* **3**, 355 (1988); S. O. Mikalsen, E. Rivedal, T. Sanner, *Carcinogenesis* **9**, 891 (1988).
53. L. D. Palekar, B. M. Most, D. L. Coffin, *Environ. Res.* **46**, 142 (1988); L. D. Palekar, J. F. Eyre, B. M. Most, D. L. Coffin, *Carcinogenesis* **8**, 553 (1987).
54. J. C. Wagner *et al.*, *Br. J. Cancer* **51**, 727 (1985).
55. M. J. Paterour, J. Bignon, M. C. Jaurand, *Carcinogenesis* **6**, 523 (1985).
56. J. D. Appel, T. M. Fasy, D. S. Kohtz, J. D. Kohtz, E. M. Johnson, *Proc. Natl. Acad. Sci. U.S.A.* **85**, 7670 (1988); G. R. Dubes and L. R. Mack, *In Vitro Cell. Dev. Biol.* **24**, 175 (1988).
57. M. C. Jaurand, L. Kheuang, L. Magne, J. Bignon, *Mutat. Res.* **169**, 141 (1986); St. Etienne *et al.*, in *Effects of Mineral Dusts on Cells*, B. T. Mossman and R. Begin, Eds. (North Atlantic Treaty Organization, Advanced Science Institute Series) (Springer-Verlag, Berlin, in press).
58. J. F. Lechner *et al.*, *Proc. Natl. Acad. Sci. U.S.A.* **82**, 3884 (1985).
59. K. T. Rajan, J. C. Wagner, P. H. Evans, *Nature* **238**, 346 (1973); P. A. Moalli, J. L. McDonald, L. A. Goodglick, A. B. Kane, *Am. J. Pathol.* **128**, 426 (1987).
60. N. S. Wang *et al.*, *Am. J. Pathol.* **126**, 343 (1987).
61. T. W. Hesterberg and J. C. Barrett, *Carcinogenesis* **6**, 473 (1985).
62. N. C. Popescu, A. P. Chahinian, J. A. DiPaolo, *Cancer Res.* **48**, 142 (1988); Z. Gibas *et al.*, *Cancer Genet. Cytogenet.* **20**, 190 (1986); M. Tiainen, L. Tammilehto, K. Mattson, S. Knuutila, *ibid.* **33**, 251 (1988).
63. B. Gerwin *et al.*, *Cancer Res.* **47**, 6180 (1987); M. A. Versnel, A. Hugemeijer, M. J. Bouts, T. H. van der Kwast, H. C. Haagsteden, *Oncogene* **2**, 601 (1988).
64. A. Haugen *et al.*, *Int. J. Cancer* **30**, 265 (1982).
65. Y. Kodama, C. J. Boreiko, S. C. Maness, T. W. Hesterberg, in preparation.
66. D. C. Topping and P. Nettesheim, *J. Natl. Cancer Inst.* **65**, 627 (1980).
67. B. T. Mossman, G. S. Cameron, L. Yotti, in *Cancer: A Comprehensive Survey: Cancer of the Respiratory Tract, Predisposing Factors*, M. J. Moss, D. G. Kaufman, J. M. Siegfried, V. E. Steele, S. Nesnow, Eds. (Raven, New York, 1985), pp. 217–230.
68. A. M. Sesko and B. T. Mossman, *Cancer Res.* **49**, 2743 (1989).
69. J. P. Marsh and B. T. Mossman, *ibid.* **48**, 709 (1988).
70. Y. Nishizuka, *Science* **233**, 305 (1986).
71. A. M. Sesko, M. Cabot, B. T. Mossman, in preparation.
72. M. Pederiset, J. P. Marsh, B. T. Mossman, in preparation.
73. E. C. Hammond, I. J. Selikoff, H. Seidman, *Ann. N.Y. Acad. Sci.* **330**, 473 (1979); J. C. McDonald, *Chest* **78** (suppl.), 374 (1980); G. Berry, M. L. Newhouse, P. Antonis, *Br. J. Ind. Med.* **42**, 12 (1985).
74. D. McFadden, J. L. Wright, B. Wiggs, A. Churg, *Am. Rev. Respir. Dis.* **133**, 372 (1986); D. McFadden, J. L. Wright, B. Wiggs, A. Churg, *Am. J. Pathol.* **123**, 95 (1986).
75. J. H. Jackson *et al.*, *J. Clin. Invest.* **80**, 1090 (1987).
76. P. Graceffa and S. A. Weitzman, *Arch. Biochem. Biophys.* **257**, 481 (1987).
77. A. Eastman, B. T. Mossman, E. Bresnick, *Cancer Res.* **43**, 1251 (1983).
78. I. Selikoff, E. C. Hammond, H. Seidman, *Cancer* **46**, 2736 (1980).
79. J. B. L. Gee and A. Bouhuys, *N. Engl. J. Med.* **285**, 1317 (1971).
80. In 1971, the Occupational Safety and Health Administration (OSHA) passed legislation restricting airborne asbestos in the workplace to five fibers greater than 5 μm in length per cubic centimeter air over an 8-hour time-weighted average. In 1986, this standard was reduced to 0.2 fibers per cubic centimeter air.
81. J. M. G. Davis and J. C. McDonald, *Br. J. Ind. Med.* **45**, 505 (1988).
82. A. Churg and J. L. Wright, in *Non-Occupational Exposure to Mineral Fibres*, J. Bignon, J. Peto, R. Saracci, Eds. (International Agency for Research on Cancer, Lyon, 1989), pp. 314–318.
83. G. J. Burdett and S. A. M. T. Jaffrey, *Ann. Occup. Hyg.* **30**, 185 (1986); M. Corn, in *Proceedings of the Symposium on Health Aspects of Exposure to Asbestos in Buildings*, J. D. Spengler, H. Ozkaynak, J. F. McCarthy, H. Lee, Eds. (Harvard Univ. Press, Cambridge, MA, in press); D. G. Massey and G. Fournier-Massey, *Hawaii Med. J.* **46**, 153 (1987); R. N. Sawyer, A. N. Rohl, A. M. Langer, *Environ. Res.* **36**, 46

(1985).
84. V. Henderson and P. Enterline, *J. Natl. Cancer Inst.* **79**, 31 (1987).
85. J. R. Hughes and H. Weill, *Am. Rev. Respir. Dis.* **133**, 5 (1986).
86. S. Cordier *et al.*, *Arch. Environ. Health* **42**, 303 (1987).
87. National Research Council, Committee on Nonoccupational Health Risks of Asbestiform Fibers, *Asbestiform Fibers: Nonoccupational Health Risks* (National Academy Press, Washington, DC, 1984); Report to the U.S. Consumer Product Safety Commission by the Chronic Hazard Advisory Panel on Asbestos (Consumer Product Safety Commission, Washington, DC, 1983); *Airborne Asbestos Health Assessment Update* (Environmental Protection Agency, Washington, DC, 1986); E. D. Acheson and M. J. Gardner, *Asbestos: The Control Limit for Asbestos* (Her Majesty's Stationery Office, London, 1983); Report on matters of health and safety arising from the use of asbestos in Ontario (Ontario Royal Commission, Ontario Ministry of the Attorney General, Toronto, 1984); R. Doll and J. Peto, *Asbestos: Effects on Health of Exposure to Asbestos* (Her Majesty's Stationery Office, London, 1985).
88. M. Corn, K. E. Crump, D. McFee, R. Lee, in preparation.
89. Battelle Columbus Division, Price Associates, Alliance Technologies Corporation, Energy Technology Consultants and Midwest Research Institute, *Assessing Asbestos in Public Buildings* (*EPA Contr. No. 68-02-4294*, draft report for the Exposure Evaluation Division, 1989).
90. H. Weill and J. M. Hughes, *Am. Rev. Public Health* **7**, 171 (1986).
91. We thank L. Sabens, V. Kelleher, and M. Bergeron. Research in the laboratory of B.T.M. has been supported by grants from the National Cancer Institute (R01 CA33501), National Institute of Environmental Health Sciences (R01 ES03878), National Heart, Lung, and Blood Institute (R01 HL39469 and SCOR grant 14212), and American Cancer Society (BC 415). We also thank H. C. W. Skinner for her helpful comments.

Priming and Human Memory Systems

ENDEL TULVING AND DANIEL L. SCHACTER

Priming is a nonconscious form of human memory, which is concerned with perceptual identification of words and objects and which has only recently been recognized as separate from other forms of memory or memory systems. It is currently under intense experimental scrutiny. Evidence is converging for the proposition that priming is an expression of a perceptual representation system that operates at a pre-semantic level; it emerges early in development, and access to it lacks the kind of flexibility characteristic of other cognitive memory systems. Conceptual priming, however, seems to be based on the operations of semantic memory.

MEMORY WAS TRADITIONALLY THOUGHT TO BE A UNITARY faculty of the mind. Recently, however, many researchers have adopted the hypothesis that memory consists of a number of systems and subsystems with different operating characteristics. The problem of what these systems and their properties are, and how they are related to one another, now occupies the center stage in research on memory.

One broad, as yet tentative, organizational scheme distinguishes procedural, semantic, and episodic memory (*1*). Procedural memory underlies changes in skillful performance and appropriate responding to stimuli; semantic memory has to do with acquisition and use of factual knowledge in the broadest sense; and episodic memory enables people to remember personally experienced events. The domain of procedural memory is behavior, whereas that of semantic and episodic memory is cognition or thought. Cognitive memory systems have the capability of modeling the external world—that is, of storing representations of objects, events, and relations among them—whereas procedural memory does not have this capability.

Evidence is accumulating about yet another category of learning and memory, one that is not procedural, semantic, or episodic. It has come to be known as priming (*2*). Its function is to improve identification of perceptual objects. Priming is a type of implicit memory; it does not involve explicit or conscious recollection of any previous experiences. It has affinities to both procedural and semantic memory. Priming resembles procedural memory in that it enhances perceptual skills. It also resembles semantic memory in that it involves cognitive representations of the world and expresses itself in cognition rather than behavior.

The prototypical priming experiment consists of two stages. In the first (study) stage, the subject is presented with a stimulus object (target). Target stimuli may comprise words, line drawings of objects, drawings of faces, and the like. In the second (test) stage, which may follow the first after an interval that can vary from seconds to months, the subject is given reduced perceptual information about the object and asked to name or categorize it. Reduced cues may consist of initial letters or graphemic fragments of words, partially obliterated words or figures, originally presented faces in a more highly schematized form, or tachistoscopic presentation of stimuli. Priming is said to have been demonstrated if the probability of the identification of the previously encountered targets is increased, or the latency of the identification response is reduced, in comparison with similar measures for nonstudied control items. The difference between performance on the target items and the nonstudied items provides a measure of the magnitude of the priming effect.

Although priming and other kinds of implicit memory have been reported from time to time, systematic attempts to explore it began about 10 years ago (*3*). One of the triggers for the study of priming turned out to be experiments by Warrington and Weiskrantz (*4*) showing that densely amnesic patients, who were severely impaired in their ability to remember recently seen information, exhibited near-normal learning when they were tested by methods that tapped what we now know is priming. A second stimulus for the study of priming lay in research concerned with the nature of and access to lexical representations (*5*). A third source of influence was the growing interest in the classification of memory into distinctive categories such as episodic and semantic memory (*6*) and procedural and declarative memory (*7*).

E. Tulving is a university professor at the University of Toronto, Toronto, Canada, M5S 1A1. D. L. Schacter is a professor of psychology at the University of Arizona, Tucson, AZ, 85721.