POOR LEGIBILITY

ONE OR MORE PAGES IN THIS DOCUMENT ARE DIFFICULT TO READ
DUE TO THE QUALITY OF THE ORIGINAL

5519

RJ LeeGroup, Inc.
350 Hochberg Road, Monroeville, PA 15146
Tel: (724) 325-1776 | Fax: (724) 733-1799

July 7, 2006

Jerelean Johnson
United States Environmental Protection Agency
Superfund Division (SFD-9-1)
75 Hawthorne Street
San Francisco, CA 94105

Daniel A. Meer, Chief
United States Environmental Protection Agency
Response, Planning and Assessment Branch
Superfund Division
75 Hawthorne Street
San Francisco, CA 94105

RE: Natural Occurring Asbestos – El Dorado Hills, California

In October 2004, the U.S. Environmental Protection Agency (EPA) Region 9 conducted a series of tests in and around El Dorado Hills (EDH), California, to assess the potential exposure of residents to naturally occurring asbestos (NOA). EPA released a report of its results to the general public in May 2005 [El Dorado Hills Naturally Occurring Asbestos Multimedia Exposure Assessment El Dorado Hills, California: Preliminary Assessment and Site Inspection Report – Interim Final]. At the request of the National Stone, Sand & Gravel Association (NSSGA), RJ Lee Group, Inc. (RJLG) conducted a review of EPA's May 2005 report and underlying data and issued a report (dated November 2005) entitled "Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project". EPA Region 9 issued a letter (Meer) dated March 9, 2006 to RJLG and NSSGA requesting the submission of supporting documentation to RJLG's November 2005 report. On April 20, 2006, EPA Region 9 issued a report entitled "Response to the November 2005 National Stone, Sand & Gravel Association Report Prepared by the R.J. Lee Group Inc [sic] 'Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project'" (Region 9 April 20 Response). In addition to the sequence of reports listed above, Mr. Gregory Meeker, USGS and a consultant to the EPA, in an undated letter, prepared a "Response to Questions Submitted by Dr. Vicki Barber, Superintendent of Schools, El Dorado County, California regarding Asbestiform Amphiboles" (Meeker Response). Dr. Barber's questions were submitted to Dr. Robert Virta, USGS, in an email dated February 1, 2006.

Upon review of the Region 9 April 20 Response, RJLG concluded there are a number of important differences of opinion as well as factual misstatements in the Region 9 Response that RJLG must address to ensure an accurate public record. To avoid unnecessary reiteration and to ensure that EPA's statements are kept in context, RJLG's responses are provided in the form of annotated comments within the Region 9 April 20 Response (Exhibit A). Similarly, RJLG has provided comments to the Meeker Response (Exhibit B). RJLG's response to EPA's March 9

SFUND RECORDS CTR
2147462

Signed for By Brenda Talverdn
3:40 PM
July 10, 2006

RECEIVED
JUL 1 0 2006
OFFICE OF THE
EXECUTIVE SECRETARIAT

letter is included in Exhibit C.

The major issues are summarized below.

There are serious data quality problems with the data from EPA's contract laboratories (i.e., Lab/Cor and Asbestos TEM Laboratory) that require a complete review and revision prior to using the data for a risk evaluation. Although EPA has continued to insist that they have followed carefully prescribed procedures for assuring data quality so that there could be no problem, RJLG's findings, also recently confirmed in two independent analyses conducted by Dr. D. Wayne Berman (on quality issues) and Mr. John Addison (on mineralogy and quality issues), indicate that the EPA's analytical results display serious data quality problems. Problems with the data include (1) the findings of the Asbestos TEM Laboratory of actinolite asbestos in the soil samples cannot be confirmed; (2) Lab/Cor failed to properly follow the ISO 10312 analytical method; (3) replicate/duplicate analyses demonstrate a lack of consistency in fiber measurements; and (4) selected area electron diffraction pattern (SAED) analysis and mineral identification by energy dispersive X-ray analysis (EDXA) show serious inaccuracies including measurement errors and systematic bias.

Irrespective of one's views regarding data interpretation or the general issues regarding asbestos and health effects in El Dorado Hills, any data used to evaluate such issues must be such that it can be reproduced in basic quality control tests (i.e., there must be reproducible counts when counting the same particle at the same location on the filter). Absent reproducibility, the data cannot be considered reliable, and it is impossible to develop or implement responsible and scientifically valid policies to address asbestos exposures or risks.

EPA Region 9 expanded the definition of "asbestos" to include all elongated mineral particles as asbestos. Region 9's analytical approach in the El Dorado Hills Study was based on counting any amphibole and serpentine particles as asbestos during TEM analyses that are longer than 5 μm and that have an aspect ratio ≥ 3:1 without consideration of whether the particle was actually asbestos or not. Lab/Cor expanded the mineralogical definition of asbestos by including particles that, at a minimum, do not have parallel sides. This modification of the analytical method, in particular the ISO 10312 procedure, is inconsistent with the geological/mineralogical definition of asbestos, and in the NOA context, results in significantly inaccurate estimates of asbestos exposure. Thus, the ISO 10312 method is an improper methodology for estimating health risks from NOA. The consequence of EPA Region 9's broadening of the definition of asbestos to include rock fragments of amphiboles and serpentine is that over 30% of the continental United States will be subject to a designation of being "asbestos" contaminated.

Based on more than thirty years of experience in developing methods for the analysis of asbestos, as well as a thorough knowledge of the most relevant and up-to-date mineralogical and epidemiological literature, RJLG has concluded that the geological and mineralogical

distinctions between asbestos and nonasbestos amphiboles and serpentines are critically important in defining and assessing exposures to asbestos, particularly exposures to NOA, which is found in an unprocessed natural state. Basing NOA exposure estimates on a tally of all elongated particles that are at least 5 μm long, whether or not the particles are asbestos, results in invalid estimates of NOA exposure that do nothing to improve the protection of public health.

Tremolite/actinolite asbestos show parallel extinction, not oblique extinction, in the polarized light microscope. Region 9 continues to state that actinolite asbestos was found in virtually all soil samples. Region 9 and Mr. Meeker compare the oblique extinction angles of the tremolite/actinolite found in the NIST Standard Reference Material (SRM) 1867a with the oblique extinction angles reported by Asbestos TEM Laboratory for particles in the El Dorado Hills soil. Region 9 discounts the RJLG argument that oblique extinction angles are inconsistent with the properties of asbestos, and relies on Mr. Meeker's interpretation of the NIST SRM 1867a to suggest that the NIST SRM 1867a is a "gold standard" for the extinction angles of tremolite and actinolite asbestos. RJLG does not believe the NIST SRM 1867a should be viewed as certifying the extinction angles of asbestos. The optical properties listed in the NIST Certificate of Analysis[1] were measured on *"larger, single crystal fibers"*, not asbestos fibers (page 3 of the NIST certificate). The NIST certificate notes that only *"some portion of the standard is asbestiform"* and also states that *"the unique morphology of asbestos may alter the properties of tremolite, actinolite, and anthophyllite asbestos from those reported for the materials contained in this SRM, as described in Reference 4."* Therefore it is incorrect to suggest that asbestos fibers can exhibit oblique extinction just because some of the particles that are present in the NIST SRM 1867a exhibit oblique extinction.

Asbestiform tremolite collected from Harvard Way near the EPA test site and from other California locations have parallel extinction. RJLG has evaluated microscopically asbestiform particles, as described in SRM 1867a, from a naturally occurring asbestos vein at Harvard Way, some two hundred yards from one of the playgrounds tested in the El Dorado Hills Study. Nineteen out of twenty asbestiform fiber bundles, meeting three or more of the criteria for the asbestiform habit, had parallel extinction. Similar results were obtained for three other samples of naturally occurring tremolite asbestos from California, including SMR 1867a. The vast majority of particles meeting two or more of the criteria that are characteristic of asbestiform minerals had parallel extinction. It is extremely uncommon to observe a population of asbestos fibers without observing parallel extinction. Addison indicates that asbestiform particles in the UK standards have parallel extinction[2]. In contrast, both Asbestos TEM Laboratory and RJLG

[1] National Institute of Standards and Technology, 2003. Certificate of Analysis; Standard Reference Material® 1867a, Uncommon Commercial Asbestos. Available: https://srmors.nist.gov/certificates/1867a.pdf?CFID=4653224&CFTOKEN=3b1dee8a6980cca6-F6F7B807-B6AE-6927-254940B41F0F309C&jsessionid=b4302e2788e7DBFE$A. Accessed May, 2006

[2] Addison (2006). "Comments on the Report Dated November 2005, by the RJ Lee Group of the 'Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project' as presented by the EPA in the document 'El Dorado Hills, Naturally Occurring Asbestos Multimedia Exposure Assessment Preliminary Assessment and Site Inspection Report Interim Final", March 23, 2006.

reported that the amphiboles in El Dorado Hills soil have inclined extinction, and **neither laboratory found particles with the asbestiform morphological characteristics** as described in the NIST SRM 1867a. Thus, RJLG found no evidence to support Mr. Meeker's argument that naturally occurring asbestiform fibers may have different extinction angles or morphologies than asbestiform fibers in commercial asbestos.

Tremolite/actinolite mineral particles do not form as asbestos fibers when their crystals contain more than a very small amount of aluminum. Region 9 disagrees with RJLG's assertion that the aluminum content of the particles in the El Dorado Hills Study is too high to permit them to be asbestiform. Mr. Meeker does not disagree with RJLG assertions concerning the aluminum content, but suggests that there may be exceptions. RJLG evaluated the aluminum content of asbestiform particles from the asbestos sample collected at Harvard Way, and the two other amphibole asbestos samples from California. None had aluminum in excess of one percent. In contrast, 75 percent of the particles in the El Dorado Hills soil analyzed by RJLG, and the particles analyzed by Lab/Cor from the El Dorado air samples, had aluminum content in excess of one percent. Thus, it is unlikely that the particles exhibiting the elevated aluminum content reported by EPA belong to the asbestiform population that is common to the El Dorado Hills area.

The Lab/Cor amphibole particles had aspect ratios consistent with nonasbestos particles, not asbestos fibers. Neither Region 9 nor Mr. Meeker evaluated RJLG's finding that the length/width distribution and mean aspect ratio of the particles in the El Dorado Hills Study were not that of an asbestiform population. Region 9 simply discounted this conclusion. RJLG also found that Lab/Cor did not adhere to the ISO 10312 method requirement that particles must have parallel or substantially parallel sides to be included in the fiber count.

The vast majority of asbestos fibers and nonasbestos particles from the El Dorado Hills location are readily classified as either asbestos or nonasbestos. Region 9 suggested that it is 'virtually impossible' to distinguish the particles in the El Dorado Hills Study from asbestiform particles based on Mr. Meeker and other literature references. As a general matter, federal agencies, including the Agency for Toxic Substances and Disease Registry (ATSDR) and the Occupational Safety and Health Administration (OSHA), have long recognized that nonasbestos particles can be distinguished from asbestos fibers. Nevertheless, to evaluate EPA's assertions, RJLG recorded TEM and SEM images of asbestiform particles from RJLG's Harvard Way asbestiform sample and other asbestiform samples from California, as well as nearly two hundred elongate particles having an aspect ratio greater than 3:1 in soil samples from the El Dorado Hills Study. These photographs, attached to RJLG's Response to EPA's March 9 request (Exhibit C), demonstrate that while there may be circumstances where the ability to differentiate between asbestiform and nonasbestos particles may be difficult, the distinction is readily apparent in the vast majority of amphibole particles from the El Dorado Hills soil samples.

A national review of naturally occurring asbestos policy is essential. Region 9 conducted the October 2004 tests to evaluate possible exposure to asbestos that may occur due to the presence of asbestos in soils. There are documented data quality flaws in EPA's El Dorado Hills Study which Region 9 neither acknowledges nor refutes. EPA Region 9, Mr. Meeker, and their contract laboratories have a very different understanding of key issues and methods than does RJLG and numerous other scientists. The issues, highlighted by the El Dorado Hills Study, have a national impact and underscore the need for substantive clarifications of NOA policy at the national level. A target outcome of this review should include improved clarity in the definitions, methods, and risk analysis procedures used to evaluate the potential health effects of NOA. The National Voluntary Laboratory Accreditation Program (NVLAP) should be charged with conducting additional training of laboratory personnel, whose primary activity to date has been post-abatement analysis of commercial asbestos fibers and who are unfamiliar with the distinction between asbestiform and nonasbestos amphiboles and serpentines, before qualifying them to analyze samples from mixed mineral environments.

The Exhibits attached provide further clarification, detail and support with regard to RJLG's fundamental analytical differences with Region 9 as well as the data quality flaws in the El Dorado Hills Study, the inaccuracies in Region 9's April 20 Response (Exhibit A), the Meeker Response (Exhibit B) and the supporting documentation of RJLG's initial evaluation of EPA's El Dorado Hills Study (Exhibit C).

Respectfully submitted,

Richard J. Lee, Ph.D.
President

copy: W. Ford, NSSGA
Wayne Nastri, Regional Administrator, USEPA Region 9
Dr. Gerald Hiatt, Senior Regional Toxicologist, USEPA Region 9

Enclosures: (listed on next page)

Enclosures:

Exhibit A: Response of RJ Lee Group to the EPA Region 9 "Response (dated April 20, 2006) to the November 2005 National Stone, Sand & Gravel Association Report Prepared by the R.J. Lee Group, Inc [sic] 'Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project'".

Exhibit B: Response of RJ Lee Group to Mr. Meeker's Letter (undated) to Dr. Vicki Barber, El Dorado Hills School District.

Exhibit C: Response of RJ Lee Group to EPA Region 9 (Meer) March 9 2006 Letter

NATIONAL STONE, SAND & GRAVEL ASSOCIATION

Natural building blocks for quality of life

Position on Naturally Occurring Asbestos

Who Are We?

The National Stone, Sand & Gravel Association (NSSGA) represents the construction aggregates industry. Our member companies produce more than 92 percent of the crushed stone and 75 percent of the sand and gravel used annually in the United States. More than three billion tons of aggregates were produced in 2005 at a value of $17.4 billion, contributing $38 billion to the GDP of the United States. Every $1 million in aggregate sales creates 19.5 jobs, and every dollar of industry output returns $1.58 to the economy. Seventy percent of our nation's counties include an aggregates operation, and virtually every congressional district is home to a crushed stone, sand or gravel operation.

Why We're Here

- In 1986 the Occupational Safety and Health Administration promulgated a regulation that regulated common mineral rock fragments as asbestos. In 1992, after six years of comprehensive review, OSHA issued a final regulation, based on risk, which removed common rock fragments from its asbestos standard.
- Since 2003, NSSGA has re-briefed U.S. EPA in five different line offices on the importance of accurate mineral definitions. Briefing Headquarters decision-makers is the next step in the process.
- In the interim, EPA Region 9's work on naturally occurring asbestos in El Dorado County, California concerns us because it does not distinguish between common mineral rock fragments and asbestos. This omission creates the potential to once again overextend the regulatory process to common mineral rock fragments.
- The approach used by Region 9 in El Dorado County is inconsistent with Region 2's investigation of the Southdown Quarry in New Jersey. Region 9's approach will result in defining harmless rocks as asbestos, creating far-reaching consequences for other EPA regions and the regulated communities (i.e. construction, land developers, housing, school districts, and mining).
- Region 9's expansion of the regulatory definition of asbestos impacts over 30 percent of the United States land area, making this a national, not a regional, issue.

NSSGA's Position On Asbestos

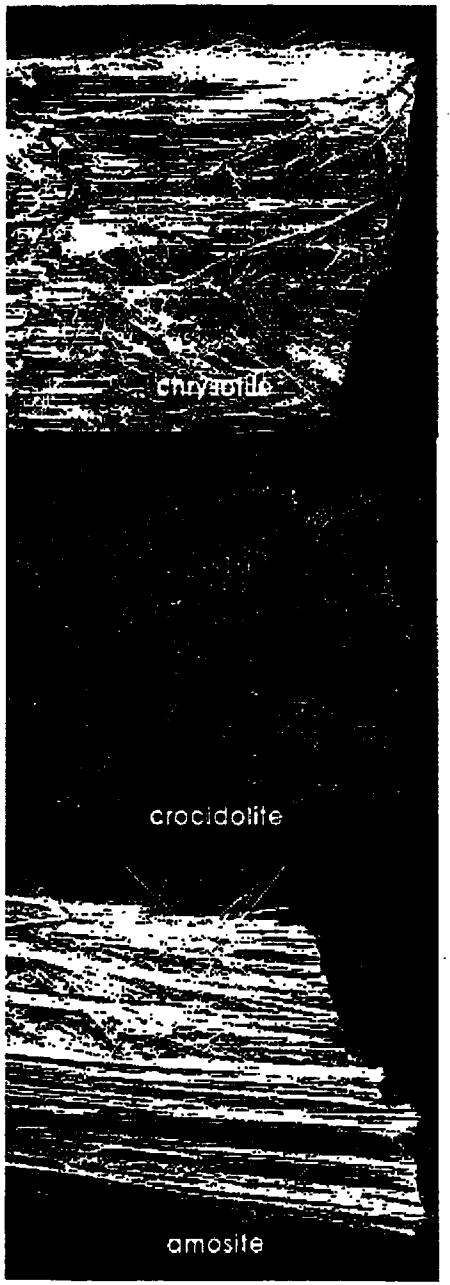
- NSSGA supports strict regulation of harmful exposure to asbestos in commercial products as well as in the natural environment. Regulation and legislation addressing asbestos must have definitions, analytical methods and risk assessment procedures that are based on sound science.
- Definitions and methods must be sufficiently accurate and precise to differentiate regulated asbestos fibers from common rock fragments. Mineral fragments such as these have never been found to cause health effects like those associated with asbestos as evidenced by the fact that asbestos-related diseases have never been associated with the aggregates industry and there are no scientific studies in the literature reporting such health effects from these rock fragments. There are many published studies that show cleavage fragments do not cause asbestos-like diseases.
- In fact, EPA Region 9 has not shown any asbestos-like disease in El Dorado County.

NSSGA Requests That The U.S. Environmental Protection Agency Headquarters...

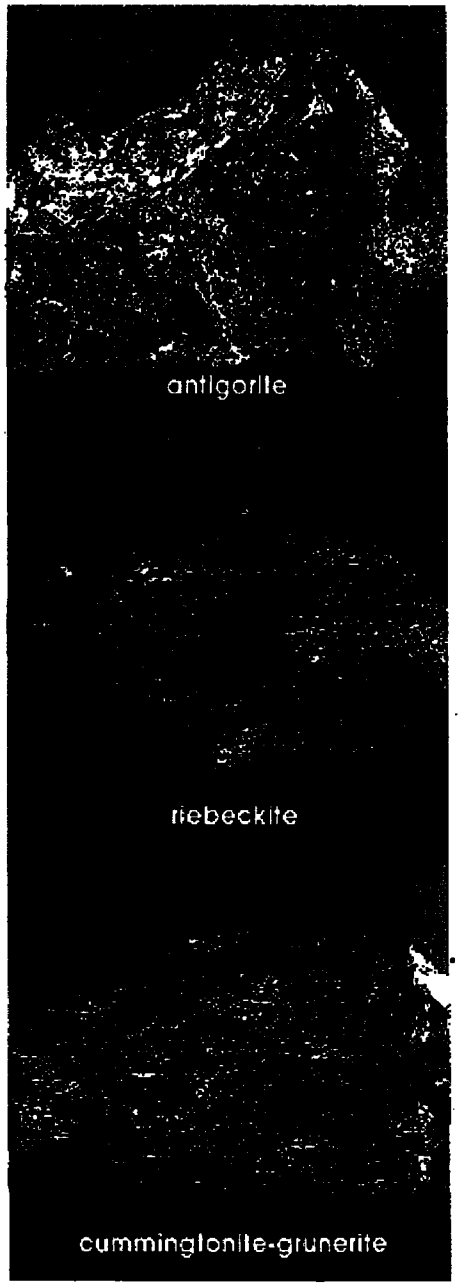
- Expeditiously initiate with an independent, neutral, qualified scientific body, preferably the Health Effects Institute (HEI), a review and study of the definition of the risk fibers in naturally occurring asbestos (both the definition and the method of measurement) and the development of a standard protocol for risk assessment and management when such materials exist in the natural environment. EPA has a 25-year history with HEI. HEI has addressed asbestos in the past. This is needed for both future regulatory and local risk assessment applications.
- Resolve the current inconsistency in assessing naturally occurring asbestos risk differently from region to region (as in the differences between Region 2 and Region 9).
- Hold in abeyance any regional office risk assessment involving naturally occurring asbestos until the conclusion of the Agency's review process.

1605 KING STREET ■ ALEXANDRIA, VA 22314
703 525 8788 ■ 800 342 1415 ■ FAX 703 525 7782
WWW.NSSGA.ORG

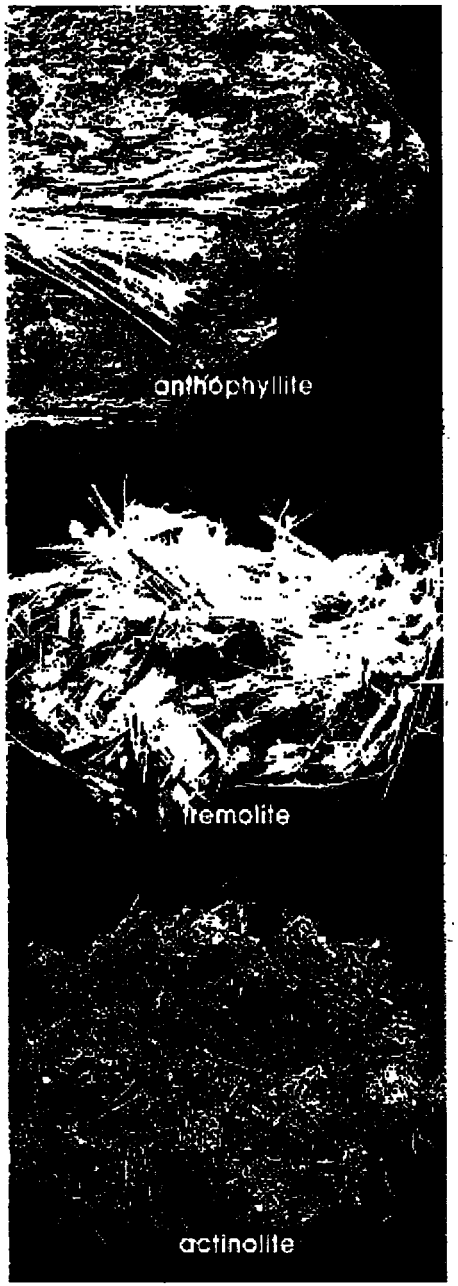
Asbestiform



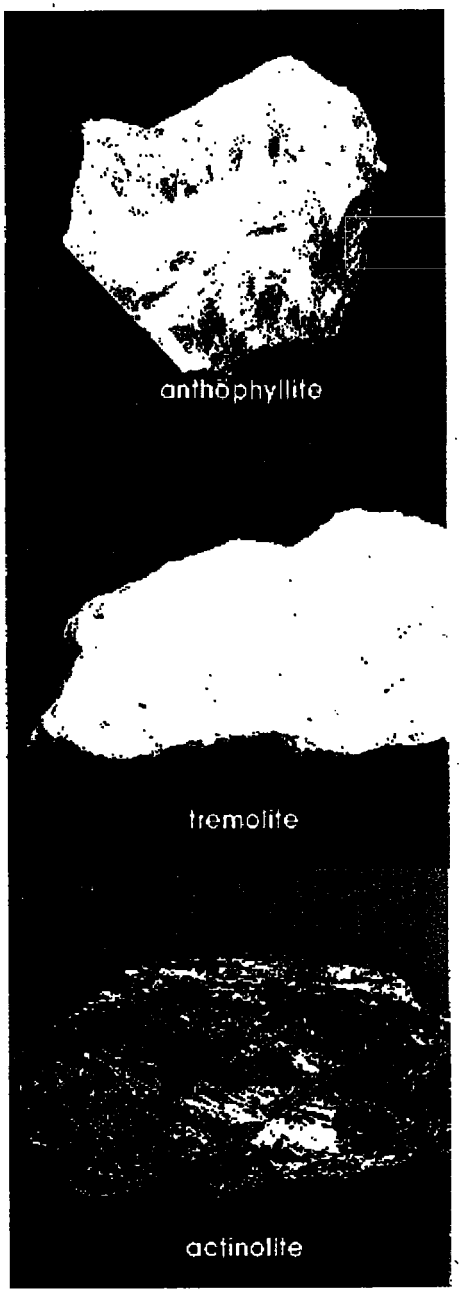
Rocks

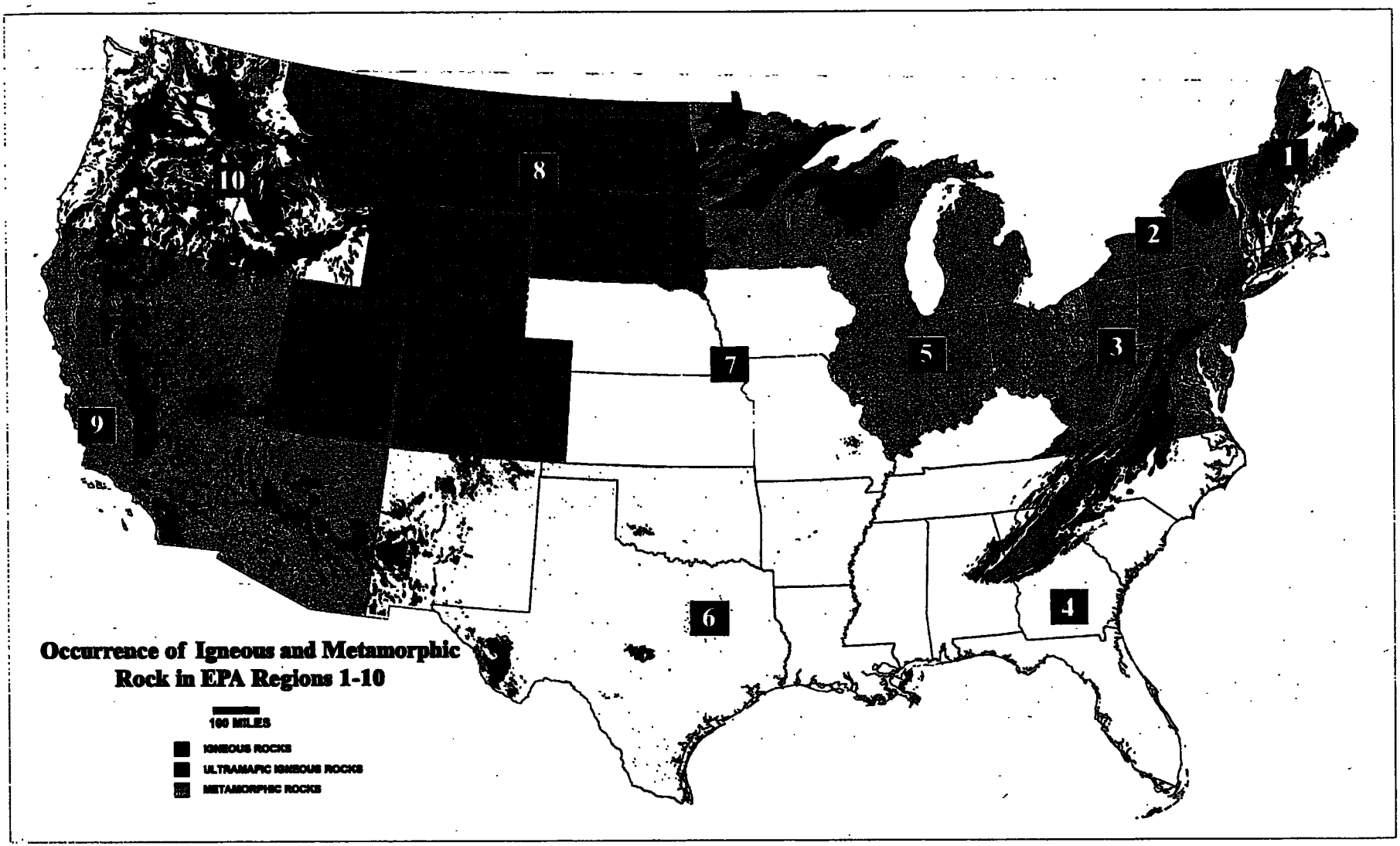


Asbestiform



Rocks





Occurrence of Igneous and Metamorphic Rock in EPA Regions 1-10

NATIONAL STONE, SAND & GRAVEL ASSOCIATION

Natural building blocks for quality of life

July 7, 2006

Mr. Daniel A. Meer, Chief
United States Environmental Protection Agency
Response, Planning and Assessment Branch
Superfund Division

Ms. Jere Johnson
United States Environmental Protection Agency
Superfund Division (SFD-9-1)
75 Hawthorne Street
San Francisco, CA 94105

RE: Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project

This letter serves as the National Stone, Sand & Gravel Association's (NSSGA) response to your request for detailed information dated March 9, 2006. Since receipt of that request, the NSSGA has received a copy of the Environmental Protection Agency (EPA) Region 9's April 20, 2006, official response to the RJ Lee Group Report. Because both documents are related to the same subject, it has required more time than initially anticipated to compile a thorough response. NSSGA has requested that one of its consultants, RJ Lee Group Inc., respond to EPA's technical questions in a separate submittal covering the assertions and issues raised in the two documents. For purposes of completeness, the NSSGA response for the EPA Headquarters distribution list includes the RJ Lee Group, Inc. submittal.

NSSGA's Experience with Environmental Implications of Mining Aggregates

The NSSGA is the world's largest mining association by product volume, according to the U.S. Geological Survey. Its member companies employ 117,000 men and women who produce 92 percent of the crushed stone and 75 percent of the sand and gravel (construction aggregates) used annually in the United States. Sales of natural aggregates generate nearly $38 billion annually for the U.S. economy. During 2005, a total of about 3.2 billion tons of crushed tone, sand and gravel, valued at $17.4 billion, were produced and sold in the U.S.

1605 KING STREET ■ ALEXANDRIA, VA 22314
703 525 8788 ■ 800 342 1415 ■ FAX 703 525 7782
WWW.NSSGA.ORG

Aggregates are used in nearly all residential, commercial and industrial building construction and in most public works projects, such as roads, highways, bridges, railroad beds, dams, airports, water and sewage treatment plants, and tunnels. While the American public pays little attention to these raw natural materials, they go into the manufacture of asphalt, concrete, glass, paper, paint, pharmaceuticals, cosmetics, chewing gum, household cleansers and many consumer goods.

Aggregates also have a number of significant environmental protection applications, including erosion control and slope protection with dams, along roadways and bridges, along shorelines, navigation channels, rivers, stream banks, construction site exits and runoff control and wetland and stream restoration; for filtration in sewage treatment, wastewater control, septic tank leaching fields and infiltration for aquifer replenishment; for flue gas de-sulfurization for acid neutralization in streams, lakes and on agricultural land; for reclamation of mine sites as backfill and land cover; in landfills and waste disposal operations as leachate and gas collection layers, covers and protection and for leachate pH adjustment; in concrete and asphalt materials used in construction for public works infrastructure, to mention just a few.

NSSGA's *Environmental Guiding Principles,* adopted as Association policy by its Board of Directors on January 20, 1991, state in part:

> "The National Stone, Sand & Gravel Association.... believes that environmental laws and regulations should be based on sound scientific, engineering and medical principles. To this end, NSSGA will work with lawmakers and regulators and make available the expertise of its members, staff and research facilities to help in shaping the nation's environmental policies."

NSSGA has a long history of working cooperatively with government agencies, including EPA, in making the results of its research and expertise available to decision makers and regulators at all levels of government. An example is the 15-year, ongoing cooperative program with EPA, and relevant state agencies, to measure and evaluate particulate emissions to the ambient air from aggregates operations. The data from the emissions testing program have resulted in accurate particulate emission factors, which are now included in EPA's AP-42 technical reference publication used by EPA and state air regulatory agencies.

NSSGA Requests National EPA Headquarters to Resolve the NOA Issue

It is apparent from Region 9's official April 20th response to RJ Lee Group's *Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project* (RJ Lee Group Report) that there are serious differences of opinion between NSSGA's many consulting scientists and EPA Region 9 regarding the health effects of cleavage fragments, the optical, mineralogical and chemical properties of asbestos versus cleavage fragments,

and Region 9's proposed expansion of the definition of asbestos to encompass common rock-forming minerals that are present in over thirty percent of the nation's continental land surface.

As documented in this response, the character of fibers described as asbestos fibers in the RJ Lee Report is consistent with well-established mineralogical research concerning the correct identification of asbestos. It is also consistent with the most current health risk assessment research concerning generic or general fiber dimension, including the Berman Crump Protocol[1] and the findings of the referenced May 2003 Peer Consultation Workshop[2] on that protocol. In contrast, the character of particles counted as asbestos fibers in the EPA El Dorado Study is inconsistent with current scientific knowledge and the Berman Crump Protocol and even the current EPA IRIS[3] risk assessments. The ultimate consequence of not correcting this discrepancy will be the issuance of an incorrect and scientifically inaccurate risk assessment which will mistakenly alarm the public and adversely impact local government bodies and economies.

After a comprehensive review by multiple, world-recognized asbestos experts, including experts in human epidemiology[4], animal[5] and cellular toxicology[6] (attached), the mineralogy and geology of asbestos[7,8], the optical and electron microscopic analysis of asbestos and cleavage fragments[9,10] and the risk assessment methods and approaches involved with asbestos[11,12], the NSSGA is convinced that there is an urgent need for an unbiased, comprehensive, independent review of the science addressing the definitions and measurement of NOA so the nation can proceed with an accurate standardized risk assessment method to ensure that public health is protected and unwarranted economic chaos is avoided. The successful model for such a review can be taken from the work of previous asbestos-related working groups enlisted by the Health Effects Institute (HEI)[13], which has a 25 year history of working with EPA on a variety of issues affecting public health, and the National Academy of Sciences (NAS)[14]. The NSSGA urges the EPA national headquarters to expeditiously commission an independent review with either the HEI or the NAS.

This national review is crucial since the approach currently being pursued by Region 9 is precedent-setting by its advocacy of a grossly over-inclusive definition of asbestos which incorporates certain common rock-forming, non-asbestiform minerals that meet simple and arbitrary analytical dimensional counting criteria adopted in 1958 by the asbestos textile industry[15]. This use of an arbitrary dimensional counting criteria to apply a risk factor derived from environments that are essentially free of rock fragments to an environment in which rock fragments constitute the vast majority (if not all) of the particles being characterized in the analysis has serious unintended consequences and is not protective of public health. In addition, Region 9's proposed approach is non-peer reviewed and is derived from data with serious quality assurance and control issues[16]. The expansion of the asbestos definition, without the presentation of any

scientific justification, to common rock-forming minerals will impact ***billions of tons*** stone that comprise the current and future infrastructure of the country. To emphasize Region 9's departure from precedent, the United States government currently does not regulate cleavage fragments as if they were asbestos in any context.

This response addresses some relevant background issues and then sets forth the scientific basis for the conclusions reached in the RJ Lee Group analysis as well as highlighting the relevant risk issues as analyzed by Dr. D. Wayne Berman, with supporting documentation separately attached.

The NSSGA has a Consistent and Long Record for Advocating Sound Science in Asbestos Regulation

The NSSGA has intensively studied the NOA issue since the Occupational Safety and Health Administration (OSHA) promulgated its 1986 asbestos standard[17] that included non-asbestiform actinolite, tremolite and anthophyllite minerals. These non-asbestiform minerals were removed from the asbestos standard after a six-year administrative stay during which the cleavage fragment health issue was studied in depth and OSHA concluded that they did not warrant regulation as if they were asbestos[18]. The science on which that decision was based has become even more supportive over the past 20 years [1,2,4,5,6,19,20,21,22].

NSSGA believes that in order to protect public and worker health, there should be strict regulation of harmful exposure to asbestos, whether it occurs from handling commercially produced asbestos products or occurs in the natural environment. In testimony before the U.S. Mine Safety and Health Administration (MSHA) in 2002[23], the NSSGA put forward its position that ***all*** asbestiform amphiboles (including winchite and richerite, as well as others) and wooly erionite, an asbestiform zeolite, be regulated as strictly as asbestos at the lower permissible exposure limit used by OSHA.

In order to achieve this goal, NSSGA believes it is very important to public health and proper risk assessment to establish an accurate mineralogical and regulatory definition of the term, "asbestiform." As shown in the enclosed document, *The Asbestiform and Prismatic Mineral Growth Habit and Their Relationship to Cancer Studies – A Pictorial Presentation*, the NSSGA has relied upon consensus definitions from world-recognized experts in asbestos mineralogy[24] as well as EPA's own definition[25] of the asbestiform mineral growth habit which is as follows:

> *Asbestiform mineral fiber populations generally have the following characteristics when viewed by light microscopy:*
>
> 1. *Mean aspect ratios ranging from 20:1 to 100:1 or higher for fibers longer than 5 micrometers,*
> 2. *Very thin fibrils, usually less than 0.5 micrometers in width,*
> 3. *Parallel fibers occurring in bundles, and*

4. *One or more of the following:*
    a. *Fiber bundles displaying splayed ends*
    b. *Matted masses of individual fibers,*
    c. *Fibers showing curvature*

Exposure to asbestos fibers with the above properties was the type that the workers experienced in the epidemiological studies used by EPA's Integrated Risk Information System (IRIS) and OSHA's asbestos risk assessments. Mixed dust environments, which have substantial concentrations of cleavage fragments, were explicitly excluded from EPA's and OSHA's risk assessment derivations. This omission is extremely informative because studies containing substantial cleavage fragment exposure (e.g. Quebec chrysotile miners) uniformly show a significantly lower dose-response than what is seen in studies of commercial asbestos[26]. This is because these cleavage fragments (in the case of chrysotile miners - antigorite and lizardite, which have not been shown to cause asbestos-like disease) were mistakenly counted as asbestos, thereby inflating the dose relative to the response[27].

NSSGA's position is based upon numerous published epidemiological and health studies of human, animal and cellular toxicity that, as recently as last year, have been reviewed by international experts in their respective fields that show that cleavage fragments do not pose asbestos-like risks[4,5,6]. The NSSGA also bases its position on OSHA's six-year review of its 1986 asbestos standard on whether to include rock fragments of the asbestos minerals in the standard[18], the late 1980's U.S. Consumer Product Safety Commission (CPSC) decision regarding tremolite cleavage fragments in play sand[28] and the more recent decision concerning "asbestos" in crayons[29], the data and epidemiological studies underlying OSHA's quantitative risk assessment[18] and EPA's IRIS update[3]; and the latest science regarding fibers that pose the most risk, the Berman Crump Protocol[1]. This massive library of science, representing years of research and conclusions, clearly indicates that exposure to common rock fragment counterparts of the more rare asbestiform varieties of the same minerals do ***not*** cause asbestos-related diseases.

Asbestos Measurement in Occupational and Ambient Environments

To fully respond to EPA Region 9's request of NSSGA, it is important to make a few background points relevant to the analytical methodologies at issue for estimating exposure to asbestos that has become dispersed in an outdoor, non-occupational setting. This type of exposure has come to be called naturally occurring asbestos or "NOA", even though, of course, all asbestos is "naturally occurring." The NOA designation points to the fact that the exposures at issue are due to geological occurrences of asbestos that may have become dispersed generally in the ambient, outdoor environment as the result of incidental disturbances, such as construction activities, rather than due to indoor, occupational exposures that occur as the result of intentional handling of commercial asbestos products.

As long recognized by OSHA and other regulatory agencies, including EPA, regulation of any asbestos risks must be based on sound techniques for identifying and estimating the presence and toxicological significance of asbestos fibers. This issue takes on special importance in the context of NOA since typically these exposures occur in the context of dispersed air and water contaminations that are difficult to identify, measure or estimate the health effects. These analytical challenges, however, do not legitimize basing NOA regulatory policy on conventional occupational exposure approaches that are scientifically invalid in the context of NOA.

The methodology used by Region 9's contracting laboratory, Lab/Cor, is based on a particle counting convention that was developed decades ago in the context of estimating exposure to asbestos fibers in occupational settings where asbestos was in commercial use. In such settings, high concentrations of asbestos fibers were known to exist and workers were exposed to them for relatively long periods of time. Because scientists used microscopy to estimate the exposure levels, they developed an estimating methodology based on counting as "asbestos" any particle they could see that was longer than 5 microns and had a ratio of length to width of 3:1 or greater[15].

The use of this approach to estimate NOA exposures is scientifically invalid because it allows simple counting criteria for known asbestos environments to be used to "define" asbestos in unknown asbestos environments containing mixed dust. Mineralogists do not, and have never defined asbestos fibers according to simple shape characteristics such as "longer than 5 microns with a minimum aspect ratio of 3:1." This description in fact could apply to many different non-asbestos minerals that do not have other physical, chemical or toxicological properties of asbestos. Thus, as detailed at much greater length below, mineralogists have evolved a number of techniques to identify asbestos particles in airborne dust and other environmental samples of unknown origin based upon an array of appropriate physical, chemical and optical properties as well as population-based generalizations about fiber length, diameter and aspect ratio[7,8,9,10,30,31,32].

These identification criteria developed by mineralogists differ significantly from the "longer than 5 um, minimum 3:1 aspect ratio" occupational exposure counting convention and, used cumulatively and with insight by skilled mineralogists, result in much more accurate asbestos identification and quantification. Indeed, RJ Lee's analysis applied many of these mineralogical criteria to conclude that Lab/Cor's inappropriate use of the conventional counting methodology to identify the mineral composition of samples collected from the ambient environment of El Dorado Hills results in highly inflated estimates of asbestos fibers in that environment. As the RJ Lee analysis shows, affirmed by three peer reviews and two subsequent reviews by Dr. Catherine Skinner and Mr. John Addison, both noted mineralogists, Lab/Cor

inaccurately identified a large proportion of elongated prismatic or non-asbestiform particles as harmful asbestos fibers.

In short, the counting criteria used by Region 9 cannot distinguish asbestos from non-asbestos and therefore provides no basis for estimating exposures to the physical, chemical or toxicological properties of asbestos that pose human health risks. An agency seeking to regulate asbestos exposure must use an appropriate and scientifically valid methodology for identifying the exposure levels and related risks. In the case of NOA, it is invalid to use an arbitrary analytical construct (i.e., a particle counting criteria) to define a mineral-based health risk. Any analytical protocol for NOA must be based on the best mineralogical science available for identifying and estimating the amount of asbestos in the ambient environment. There is no role for overly broad, simplistic fiber counting criteria in asbestos identification or risk estimation.

The actual history behind the adoption of the 3:1 aspect ratio is also informational for understanding the deficiencies inherent in EPA's current protocol as it pertains to mixed dust environments. In the second half of the twentieth century, UK and US scientists seeking to help employers estimate the occupational exposure of their workers to asbestos in factories involved in commercial usage of asbestos developed a "counting" methodology that consisted of collecting air samples on a filter and then, using a phase-contrast light microscope, counting as asbestos any mineral particles that had a length of 5 um or longer and had an aspect ratio (i.e., ratio of length to diameter) of at least 3:1.

The 3:1 aspect ratio was not based on any scientifically valid definition of asbestos characteristics or the toxicological significance of such characteristics, but reflected a need to improve consistency in exposure measurements by analysts. Since fiber counting analysis was performed using a phase-contrast light microscope at a magnification of 400-450x which made the minimum identifiable width 0.2-0.25 um, and since asbestos fibers may be as small as 0.02 um in diameter, there was recognition that the measurement was only serving as an ***index of exposure*** versus an absolute quantification of asbestos fibers. The researchers found that the 5 um, 3:1 counting convention resulted in the most consistent counting outcomes[15].

Since this convention was used with respect to counting particles known to have originated in a setting where asbestos was commercially in use and where the primary elongated particles present could reasonably be assumed to be asbestos, this convention, which does not require the analyst to identify asbestos, made sense in that setting. In the 1970s, the approach was adopted by the federal governments in both countries for the limited purpose of estimating asbestos exposures in occupational settings, where typically asbestos fibers were known to exist in significant quantities. Phase contrast microscopy has continued to be the method of choice for the

measurement of occupational exposure to asbestos because it is inexpensive and can be performed quickly on-site.

However, it is widely recognized that this traditional counting approach is wholly inadequate to identify asbestos fibers in other environments where elongated particles may or may not be asbestos. The main disadvantage of the approach is that it does not positively identify asbestos fibers, which requires polarized light and/or electron microscopy coupled with the analyst's knowledge and practical experience utilizing these tools to identify minerals. In addition, because the PCM method is unable to see fibers that are less than about 0.2 um in diameter, in some cases substantially more asbestos fibers may be present than are actually counted by the method. This is also true with the PCME methods since, even though the methods use the electron microscope, they also use the simplistic PCM counting criteria.

The conventional counting method is particularly inadequate to identify asbestos fibers in the context of NOA where particles are collected from the ambient, natural environment, such as in El Dorado Hills. There are simply too many non-asbestiform amphibole particles that fit the 5 um, 3:1 definition in such an environment. In these circumstances, the failure to accurately identify and estimate the presence of asbestos fibers, and thus to accurately estimate potential human exposure, can have disastrous consequences. Overestimations can result in unwarranted public panic and costly remedial actions, as well as associated adverse consequences for local economic activities. More importantly, underestimations can result in failures to protect public health adequately. Both errors adversely impact our understanding of asbestos and general fiber risk and therefore do not serve the best interest of anyone.

Thus, the conventional method for counting asbestos fibers, while perhaps adequate in the occupational setting for which it was devised, is severely inadequate for use in evaluating risks from NOA (or indeed, risks from asbestos exposures in other non-occupational environments, such as on city streets from tires, for example).

There is not a divergence of opinion within the scientific community about the appropriate methods for identifying asbestos. The fact is that current analytical methods were designed to measure commercial asbestos in relatively simple matrices not in ambient mixed dust environments. Blindly using existing methods without applying what is known about asbestos mineralogy and morphology is inadequate to assess risk, and in some cases, inflates risk. NSSGA's only goal is to work with Region 9/EPA to produce an accurate protocol for identifying and assessing NOA risks.

Dr. Berman's Evaluation Confirms Inadequacy of Region 9's Approach in El Dorado Hills

The NSSGA, shares EPA's deep concern about the health effects of naturally occurring asbestos (NOA). The NSSGA wants to insure that the American public receives the best

available, most accurate and scientifically sound information on which to base responsible public health and policy decisions. In continuing pursuit of that objective, the NSSGA commissioned Dr. D. Wayne Berman, one of the nation's premier asbestos risk assessors, especially as it pertains to mixed dusts in natural environments, to examine the May 2005 EPA Region 9 El Dorado Hills Study and to address the health risk issues.

Based on his extensive experience in assessing asbestos risks in both commercial asbestos and NOA environments, Dr. Berman is eminently qualified to review the EPA El Dorado Hills Study. Dr. Berman's relevant expertise in this area includes design and/or management and oversight of the original EPA risk assessments of the Atlas Mine Site, Coalinga Mill Site, Clear Creek Management Area, Diamond XX Site, and the Southdown Quarry. He also contributed to the EPA work at Libby, MT. Dr. Berman has also conducted assessments of asbestos-related risks at the Johns-Manville Waukegan Facility, the Klamath Falls - North Ridge Estates Site, the 3rd Street Light Rail Project Site for the City of San Francisco, and other sites around the country. Dr. Berman has also served as the sole non-government member of the National Asbestos Task Force for the EPA, where he developed a set of mutually consistent methods for determining asbestos concentrations in environmental media that are unique because results can be related to risk. These methods have been published by EPA as interim EPA Superfund Methods. Most importantly, Dr. Berman pioneered an approach for the evaluation of asbestos-related risks that was reviewed favorably in 2003 by eleven nationally recognized asbestos experts. Dr. Berman's report[33] on El Dorado County is provided as an attachment to this letter.

One of the main findings of the Berman report is "that the [EPA-proposed] approach [for assessing risk in El Dorado County] may not be as well established by precedent as the approaches that the Agency commonly employs for other hazardous materials." Thus, in order for EPA to effectively protect public health, NSSGA believes it is imperative that EPA conduct a thorough review of its asbestos risk assessment approach because, if the EPA approach is applied uniformly to mixed dust environments, ***it will be less protective of public health*** than other approaches already available (e.g. the Berman Crump Protocol). The Berman Crump Protocol accounts adequately for the differences between cleavage fragments and asbestos due primarily to the size range of the risk fibers as explained in detail in the attached report by Dr. Berman.

Some of the key findings from Dr. Berman's report are:

The EPA approach at El Dorado ***does not*** satisfy two fundamental criteria that are essential for assuring that risk assessments are reliable, namely:

- exposure concentrations determined in terms of the PCMe metric used by EPA in El Dorado County are ***not*** directly comparable to the PCM-based exposures evaluated in the epidemiology studies used to derive the risk factors in IRIS (Current).

- The PCMe exposure metric has been shown ***not*** to remain reasonably proportional to risk across different exposure environments, especially mixed dust environments like El Dorado Hills.

Dr. Berman points out that EPA is citing IRIS as the comparison basis for exposures in El Dorado Hills yet the minimum width of fibers defined for PCMe structures in IRIS is twice the minimum width of fibers defined in the ISO analytical method employed in the study. IRIS further indicates that the correlation between PCM and TEM fiber counts is "highly uncertain." This inconsistency with IRIS makes the approach proposed by Region 9 even more problematic with respect to El Dorado Hills.

The NSSGA has obtained and photographed bulk asbestos samples that are examples of the source of exposures in the various epidemiological studies used in the EPA IRIS risk assessment. These photographs, first shown optically and then by digital SEM, are attached. Figure 1a is an optical image of chrysotile asbestos grade 3, which was typical for use in asbestos textile manufacturing, and Figure 1b is the same material shown under SEM. Figure 2a is an optical image of chrysotile asbestos grade 4, which was typical for asbestos insulation and asbestos cement pipe manufacturing. Figure 2b is the same material shown under SEM. Figure 3a is an optical image of chrysotile asbestos grade 7 which was typical for asbestos used in friction parts and asbestos containing gaskets and is noticeably less "pure" than grades 3 and 4. Figure 3b is the same material shown under SEM. Figure 4a is an optical image of raw Quebec chrysotile asbestos ore, which clearly shows significant quantities of rock fragments mixed with the asbestos fibers. The epidemiological studies whose workers were exposed to this mixed dust environment were excluded from IRIS and OSHA's risk assessment calculations. Figure 4b is the same material shown under SEM. Finally Figure 5a is an optical image of the El Dorado soil obtained from the EPA split samples. Figure 5b is the same material shown under SEM. It is readily apparent that the soil sample is in no way comparable to the IRIS excluded chrysotile ore sample (Figure 4), and it is even much less comparable to the samples that represent the source material for the studies included in EPA's IRIS risk assessment (Figures 1 - 3). EPA Region 9's proposal to apply the IRIS risk assessment factors to the El Dorado Hills data is clearly not appropriate based simply on an examination of the photographs, however, the multitude of other factors pointing to the absence of asbestos (i.e. aluminum content, extinction angle, dimensional characteristics and asbestiform morphological properties) make this even more unreasonable.

Dr. Berman also demonstrates that PCM/PCMe ratios used in risk assessment over the past 20 years have not been uniform and that it does not appear that these approaches have been subjected to formal peer-review at EPA. Studies of the PCM/PCMe exposure metric's ability to predict risk have been formally tested and have been shown to provide a ***statistically significant lack of fit*** among relatively "pure" asbestos exposures. It is highly likely that the fit would be even worse with a mixed dust environment like El Dorado Hills.

One of the most important observations discussed in the attached report by Dr. Berman is that prior to the current EPA study in El Dorado Hills, the Berman Crump Protocol consistently provided higher estimates of risk at sites where amphibole asbestos was a contributor to exposure than either IRIS or other traditional EPA approaches. When applied to the EPA study of El Dorado Hills, however, this appears not to be the case. This strongly suggests something is radically different about the locations tested in El Dorado Hills when compared to every known asbestos environment that has been previously modeled using the Berman Crump approach.

The NSSGA, and an extremely knowledgeable group of mineralogists with decades of experience in asbestos mineralogy (Dr. Malcom Ross, Dr. Ann Wylie, Dr. Catherine Skinner, Dr. Art Langer and John Addison - CVs attached), are of the opinion that the lack of consistency with the historical trends in the relative risk estimated using various approaches is because there was essentially no asbestos in the samples collected in the EPA study of El Dorado Hills[34,35,36,37,38].

Five Asbestos Mineralogists with Decades of Experience Confirm the RJ Lee Group Conclusions

Asbestos is known to be present in the El Dorado Hills area. In fact, RJ Lee Group found amphibole asbestos some 200 yards from the EPA test site. The issue here is that there was little to no asbestos present in the samples collected by EPA in their study.

EPA's Selected Method

Lab/Cor indicated that it used ISO 10312[39] when analyzing samples collected in El Dorado Hills. This method is based on the traditional counting approach, counting fibers longer than 5 um, but uses a longer aspect ratio (5:1 instead of 3:1). However, as documented in the RJ Lee Report, Lab/Cor used the conventional 3:1 ratio to count asbestos fibers for the El Dorado Hills analysis, even though it did not report this modification on its laboratory reports.

Use of historical counting convention, to *identify* asbestos when examining mineral particles of unknown origin, is a wildly inexact science. Lab/Cor's substitution of the 3:1 ratio rather than the 5:1 ratio mandated in the ISO standard is particularly disturbing in this case, since ISO 10312, as the standard states itself, cannot distinguish between asbestos fibers and non-asbestos particles at a 5:1 aspect ratio, let alone an even

less discriminating 3:1 aspect ratio. Thus, using ISO 10312, combined with an over-inclusive aspect ratio, necessarily results as the RJ Lee Group analysis demonstrates, in inflated reported asbestos concentrations.

Aluminum Content of Particles are Not Characteristic of Asbestos

The RJ Lee Group looked at the electron dispersive x-ray analyses (EDXAs) that Lab/Cor had performed on a representative sampling of amphibole (actinolite) particles, and found that the detailed mineralogical analyses showed that 63% of the reported amphibole actinolite particles Lab/Cor identified as asbestos fibers contain too much aluminum to be asbestos (i.e., they contained more than 1.5 percent aluminum). It is well-established in the scientific literature that particles classified as amphibole asbestos contain only trace quantities of aluminum since the amount of aluminum in the mineral's formation stage influences whether an amphibole mineral develops the asbestiform growth habit[9,40,41]. Because a fiber is composed of highly aligned chemical units, there is no room to accommodate larger atoms such as aluminum, and the presence of too much aluminum will result in structural changes that cause prismatic crystals (i.e., the non-asbestiform habit) rather than the characteristic bundle of fibrils (asbestiform growth habit) necessary for the minerals that are regulated and defined as asbestos to develop.

Three mineral scientists, who have decades of experience in asbestos mineralogy, peer-reviewed the RJ Lee Group analysis and all noted that the reported aluminum levels of the particles in the El Dorado Hills study were inconsistent with Lab/Cor's identifying the particles as asbestos. Dr. Ann G. Wylie, a nationally recognized mineralogical expert, stated that the amount of aluminum present in many of the elongated particles identified as asbestos is too high for these particles to be asbestos. Dr. Wylie, along with Jennifer Verkouteren (a mineral scientist at the National Institute of Standards and Technology), is the author of research published in the American Mineralogist in 2000, which completely analyzed 103 members of the amphibole series and found that the total amount of aluminum in atoms per formula unit for samples of asbestos was less than 0.3 .

Similarly, Dr. Arthur Langer, a world-recognized asbestos mineralogist with extensive publications on NOA issues, and Dr. Malcolm Ross, retired USGS mineralogist specializing in asbestos, both stated that that the high aluminum content of the 63% of particles Lab/Cor identified as "actinolites" should be classified, using established mineralogical nomenclature, as common hornblende cleavage fragments because they had greater than 0.5 aluminum pfu.

The RJ Lee Group also analyzed the aluminum content of splits of 23 soil samples collected from areas where EPA's activity-based sampling had indicated elevated asbestos fiber concentrations. They found that the amphibole minerals present in the soil samples, just as in the samples taken from air filters, contained elevated levels of

aluminum consistent with hornblende and non-asbestiform actinolite rather than asbestos.

Dr. Catherine Skinner, a Yale mineralogist with extensive experience in asbestos, and Mr. John Addison, noted asbestos mineralogist in England, both reviewed the RJ Lee Group conclusions and generally confirmed them as well.

Extinction Angles of Particles are Not Characteristic of Asbestos
The RJ Lee Group analysis also found that the reported extinction angles for the soil samples EPA's contract laboratory identified as asbestos are inconsistent with asbestos fibers. TEM Asbestos Laboratories, also an EPA subcontractor, had analyzed the soil samples using polarized light microscopy (PLM) and concluded that the amphibole content was consistent with actinolite asbestos. However, the subcontractor also reported that the amphibole actinolite particles reported as asbestos in the soil samples had a reported extinction angle of 12 degrees. Noting that extinction angles above 10 are an intrinsic property of non-asbestiform cleavage fragments, whereas amphibole asbestos displays an extinction angle that is normally less than 10 degrees, the RJ Lee Group concluded that the amphibole particles reported in the soil samples cannot be asbestos. This conclusion was also consistent with lack of any asbestiform mineral habit morphology (fibrillar bundling) in the soil samples.

Dr. Wylie's peer-review confirmed this conclusion, stating that "The conclusion of the report that an extinction angle of 14 degrees is too large for asbestos is consistent with the findings Jennifer Verkouteren and I published in American Mineralogists in 2002, in an article entitled "Anomalous optical properties of fibrous tremolite, actinolite, and ferro-actinolite. ... When oblique extinction is observed in asbestos fibers in the tremolite-actinolite-ferroactinolite series, it is less than 10 degrees." Dr. Ross concurred with this finding as well, stating "Asbestos fibers grow as bundles of fibers oriented randomly about the common crystallographic c-axis. As such, the bundles behave optically as an orthorhombic mineral with a c-axis extinction angle of zero degrees."

Particle Dimensions are Not Characteristic of Asbestos
Criteria for identifying asbestos fibers in airborne dust and other non-occupational environmental samples must recognize not only appropriate chemistry and optical properties but also the fact that aspect ratios of asbestos fibers are generally extremely large: generally from 20:1 or higher for fibers greater than 5 um in length. Indeed, EPA's "Method for the Determination of Asbestos in Bulk Building Materials," EPA Report NO. EPA/600/R-93/116 (NTIS/PB93-218576), July 1993 (updates and replaces interim version in 40 CFR 763, Subpart F, App A), defines the term asbestiform in part by stating that the asbestiform habit is generally recognized by *"mean aspect [length to width] ratios ranging from 20:1 to 100:1 or higher for fibers longer than 5 micrometers ... very thin fibrils, usually less than 0.5 micrometers in width."*

On the other hand, the far more common non-asbestiform crystal growth habit has distinctly different morphological characteristics[30]. In hand held specimens as well as microscopically, they appear as single crystal particles and, unlike asbestos fibers, particle width is dependent on length (i.e., the longest cleavage fragments are also the widest). Therefore they have a size range (length and width) that is distinctly different than asbestos fibers, there is no evidence of multiple crystals forming a single fiber bundle growing in parallel alignment (splayed ends, separation of the fiber), and they are brittle and therefore show little if any curvature as they break along cleavage planes rather than bend. Though these characteristics may be difficult to distinguish in the case of single, very small particle, they are unmistakable when observed on a population basis.

Understanding the nature of cleavage fragments is central to understanding any health risks posed by NOA. It is clear that amphibole minerals far more commonly exist in the non-asbestiform habit than in the asbestiform habit, and that they produce common, garden variety cleavage fragments when broken down. Further, mineral science tells us that once asbestos is formed in its unique crystal growth habit, it cannot form cleavage fragments. Conversely, cleavage fragments cannot be made into asbestos by any form of manipulation including weathering. Cleavage fragments are regarded by mineral scientists as distinctly different from asbestos fibers due to the marked difference in crystal growth of the two habits (fibrillar structure, strength or flexibility, particle dimensions, how they further break down, etc.). The key distinctions cannot be altered. Even though some cleavage fragments can be found with aspect ratios greater than 3:1, cleavage fragments are not asbestos at any size.

The distinction between asbestos fibers and cleavage fragments has been incorporated in most asbestos regulatory policy as well. Since at least 1984, as stated in a report on non-occupational exposure to asbestiform minerals commissioned by the National Research Council[14], cleavage fragments have been categorized as distinctive from asbestos fibers: *"CLEAVAGE refers to the preferential breakage of crystals along certain planes of structural weakness. ... A mineral with two distinct cleavage planes will preferentially fracture along these planes and will produce ACICULAR fragments. Minerals with one cleavage plane produce PLATY fragments and those with three or more cleavage planes yield POLYHEDRAL fragments.... Cleavage cannot produce the high strength and flexibility of asbestiform fibers."* This critical and clear distinction has been extensively explained, graphically shown and photographed in the scientific literature.

Dr. Wylie and Dr. Langer in their review of the RJ Lee Report again confirm that the dimensions of the elongated particles identified as asbestos fibers by Lab/Cor are not consistent with the conclusions of years of mineralogical research on the dimensions of asbestos particles. "Asbestos populations have distinct and unambiguous dimensional characteristics that are readily distinguished from populations of cleavage fragments," Dr. Wylie states. Both Wylie and Langer point out that Lab/Cor reported that 96% of

the El Dorado Hills Study particles that were longer than 5 um have widths greater than 0.5 um and that these dimensions are not consistent with a population of asbestos fibers. Particle size (especially when applied on a population basis) is an effective way to help distinguish asbestos from non-asbestiform, but it is not the only indicator. Other asbestos fiber properties also influenced by the asbestiform growth habit such as fiber extinction angle, fiber defect characteristics, evidence of fiber bundling and curvature can also be used to help make the distinction.

EPA's Mineralogical Expert's Use of Mineralogical Anomalies and Exceptions as General Rules for Asbestos Identification

Mr. Gregory Meeker, a geologist and electron probe analyst at USGS, serves as EPA's asbestos mineralogy expert. Mr. Meeker's use of mineralogical curiosities and exceptions to common asbestos mineralogy in his attempt to characterize the findings in the El Dorado Study as being consistent with asbestos mineralogy is more comprehensively addressed by RJ Lee Group's submission to EPA's original request and its response to the EPA April 20th formal critique of the RJ Lee Group Report, however, the NSSGA believes it is very important to highlight an example of this point.

Mr. Meeker[42] has characterized the NIST tremolite/actinolite standard 1867a as the "gold standard" by which asbestos can have more aluminum than what is cited in the literature or an inclined extinction angle in polarized light microscopy - just like cleavage fragments of these minerals. The NIST standard is in fact a mixture of asbestos and cleavage fragments of the same mineral as is clearly stated on the NIST certificate[43] that accompanies each and every vial of the standard. Meeker cites the information pertaining to the cleavage fragment portion of the NIST standard as if it was characteristic of the asbestiform portion of the standard. The aluminum and extinction angle parameters are but two facets in the analysis of asbestos along with parallel sides, the presence of bundles, very thin fibers, very high aspect ratios, splayed ends and curved fibers to name several. The particles measured and analyzed in the EPA El Dorado study lacked ***all*** of these characteristics of asbestos along with having too much aluminum and particles with an inclined extinction angle making the "asbestos" found in the samples the most unique asbestos thus far encountered in mineralogical science since they have no classically recognized asbestos properties.

EPA Region 9's Inclusion of Cleavage Fragments in its Asbestos Definition is Unprecedented for a US Enforcement Agency

EPA Region 9 has also maintained that, for purposes of risk assessment, there should be no distinction between cleavage fragments and asbestos fibers. However, the differences between cleavage fragments and asbestos are widely recognized by various agencies of the U.S. government and the intention to regulate rock fragments as asbestos has been consistently rejected as noted below:

- *"OSHA also reviewed available relevant evidence concerning the health effects of non-asbestiform tremolite, anthophyllite, and actinolite and examined the feasibility of various regulatory options. Based on the entire rulemaking record before it, OSHA made a determination that substantial evidence is lacking to conclude that non-asbestiform varieties of asbestos minerals present the same type of magnitude of health effect as asbestos."* **(OSHA, "Final Rule: Occupational Exposure to Asbestos, Tremolite, Anthophyllite, and Actinolite," 57 Fed. Reg. at 24310.)**

- *"In the asbestiform habit, mineral crystals grow forming long, thread-like fibers. When pressure is applied to an asbestos fiber, it bends much like a wire, rather than breaks. ... In the non-asbestiform habit, mineral crystals do not grow in long thin fibers. They grow in a more massive habit. For example, a long thin crystal may not be polyfilamentous nor possess high tensile strength and flexibility, but may break rather than bend. When pressure is applied, the non-asbestiform crystals fracture easily into prismatic particles, which are called cleavage fragments because they result from the particle's breaking or cleavage, rather than the crystal's formation or growth. ...Cleavage fragments are not asbestiform and do not fall within our definition of asbestos."* **(Mine Safety and Health Administration (MSHA), "Asbestos Exposure Limit" Proposed Rule, Notice of Public Hearing, 70 Fed. Reg. 43590, 43593, July 29, 2005.)**

- *"The optimal exposure index that best reconciles the published literature assigns equal potency to fibers longer than 10 μm and thinner than 0.4 μm and assigns no potency to fibers of other dimensions."* **(EPA, Final Draft: Technical Support Document for a Protocol to Assess Asbestos-Related Risk. (Berman and Crump Protocol) Prepared for Office of Solid Waste and Emergency Response, September 4, 2001 updated in 2003; Executive Summary, p. 1.4)**

- *"As your letter indicates, the AHERA definition of asbestos is 'the asbestiform varieties of: chrysotile (serpentine); crocidolite (riebeckite); amosite (cummingtonite-grunerite); anthophyllite; tremolite; and actinolite.' This is also the definition used in EPA's National Emission Standards for Hazardous Pollutants (NESHAP) Asbestos Regulations. EPA does not regulate the non-asbestiform varieties of these materials."* **(Letter from Diane Sheridan, Chief of Abatement Programs Section, EPA, to John Kelse, R.T. Vanderbilt Company, Inc., August 28, 1992.)**

- *"This examination [of play sand sample] found fragments of non-asbestiform tremolite, but did not find tremolite asbestos. Some of the non-asbestiform tremolite cleavage fragments may appear, under some microscopy techniques, to fit a definition of "fiber" used by the Occupational Safety and Health Administration. The staff has conducted a review of the available information on*

non-asbestiform tremolite and concludes that there are no definitive animal or human studies demonstrating that non-asbestiform tremolite presents a health hazard." **(U.S. Consumer Product Safety Commission, Briefing Package of the CPSC Office of the Secretary on a Petition to Ban Play Sand with Non-Asbestiform Tremolite, October 26, 1988, Executive Summary paragraphs 3 & 4.)**

In summary, please see the detailed response of the R. J. Lee Group for the technical information requested in your letter of March 9th and comments included in your April 20 report as well as the Dr. Wayne Berman report on the health concerns associated with the EPA approach to El Dorado Hills.

NSSGA believes there is an urgent need for an unbiased, independent and comprehensive review of the science around the definitions and measurement of NOA. NSSGA looks forward to working cooperatively and productively with EPA towards achieving this goal and believes that debating this complicated science in the media is unproductive and a disservice to the public. With this review, the various stakeholders, including other federal and state agencies, can proceed with an accurate standardized risk assessment method to ensure that public health is protected. The successful model for such a review can be taken from the work of previous asbestos-related working groups enlisted by the Health Effects Institute or the National Academy of Sciences and we urge EPA headquarters to commission an independent review with one of these two organizations as expeditiously as possible.

Respectfully Submitted,

William C. Ford
Senior Vice President

cc: Mr. Michael Cook
Dr. George Gray
Dr. Gerald Hiatt
Dr. Stephen Johnson
Mr. Wayne Nastri

REFERENCES

1. D. W. Berman and K.S. Crump (2003). "Technical Support Document for a Protocol to Assess Asbestos-Related Risk," EPA, U.S. Environmental Protection Agency, Revision of original from September 4, 2001, Peer-reviewed consultation held in San Francisco on February 25-26, 2003.

2. USEPA (U.S. Environmental Protection Agency) (2003). "Report on the Peer Consultation Workshop to Discuss a Proposed Protocol to Assess Asbestos-Related Risk, Final Report". Office of Solid Waste and Emergency Response, Washington D.C.

3. USEPA (U.S. Environmental Protection Agency) (1986) Airborne Asbestos Health Assessment Update, Report 600/8-84-003F

4. J. F. Gamble and G. W. Gibbs (2005). "An Evaluation of the Risks of Lung Cancer and Mesothelioma from Exposure to Amphibole Cleavage Fragments", Peer Reviewed, Revised and Accepted for Publication in Proceedings of the International Symposium on the Health Hazard Evaluation of Fibrous Particles Associated with Taconite and the Adjacent Duluth Complex, St. Paul, Minnesota, March 30-April 1, 2003.

5. J. Addison and E. E. McConnell (2005). "A Review of Carcinogenicity Studies of Asbestos and Non-Asbestos Tremolite and Other Amphiboles", Peer Reviewed, Revised and Accepted for Publication in Proceedings of the International Symposium on the Health Hazard Evaluation of Fibrous Particles Associated with Taconite and the Adjacent Duluth Complex, St. Paul, Minnesota, March 30-April 1, 2003.

6. B. T. Mossmann (2005). "Assessment of the Pathogenic Potential of Asbestiform vs. Nonasbestiform Particulates (Cleavage Fragments) in *In Vitro* (Cell or Organ Culture) Models and Bioassays", Peer Reviewed, Revised and Accepted for Publication in Proceedings of the International Symposium on the Health Hazard Evaluation of Fibrous Particles Associated with Taconite and the Adjacent Duluth Complex, St. Paul, Minnesota, March 30-April 1, 2003.

7. A. G Wylie (1989). "Mineralogical Definitions for Asbestos Fibers and Cleavage Fragments". Report of the Committee on Geology and Public Policy GPP012. Geology Society of America, p. 2-4.

8. A. M. Langer, R. P. Nolan, J. Addison (1991). "Distinguishing Between Amphibole Asbestos Fibers and Elongate Cleavage Fragments of Their Non-

Asbestos Analogues". Mechanisms in Fibre Carcinogenisis, R. C. Brown et al., editors, Plenum Press, New York. P 253 -267.

9. J. R. Verkouteren and A. G. Wylie (2002). "Anomalous Optical Properties of Fibrous Tremolite, Actinolite and Ferro-actinolite". American Mineralogist, 87, p 1090-1095.

10. R. J. Lee and R. M. Fisher (1979). "Identification of Fibrous and Nonfibrous Amphiboles in the Electron Microscope". Health Hazards of Asbestos Exposure, Annals of the New York Academy of Science, 330, p. 645-660.

11. D. W. Berman, K. S. Crump, E. J. Chatfield, J. M. G. Davis, and A. Jones (1995). "The Sizes, Shapes, and Mineralogy of Asbestos Structures that Induce Lung Tumors or Mesothelioma in AF/HAN rats Following Inhalation". Risk Analysis, 15:2, p 181-195.

12. D. W. Berman and K. S. Crump (1999). "Methodology for Conducting Risk Assessments at Asbestos Superfund Sites, Part 1: Protocol". Prepared for Kent Kitchingman, U.S. Environmental Protection Agency (EPA) EPA Contract No. 68-W9-0059, Work Assignment No. 59-06-D800, Final.

13. HEI-AR (1991). "Asbestos in Public and Commercial Buildings: A Literature Review and Synthesis of Current Knowledge".

14. NRC (1984). "Asbestiform Fibers-Nonoccupational Health Risks". Washington D.C., National Academy Press.

15. W. H. Walton (1982). "The Nature, Hazards and Assessment of Occupational Exposure to Airborne Asbestos Dust: A Review". Annals of Occupational Hygiene, 25, p 117-247.

16. R. J. Lee Group Inc. (2005). "Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project" Prepared for the National Stone, Sand and Gravel Association, Project LSH306975.

17. Occupational Safety and Health Administration (1986): "Final Rule Exposure to Asbestos, Tremolite, Anthophyllite and Actinolite for Construction and General Industry". Federal Register v. 51. no. 119, June 20, 1986.

18. Occupational Safety and Health Administration (1992). "Occupational Exposure to Asbestos, Tremolite, Anthophyllite and Actinolite: 29 CFR Parts 1910 and 1926 (Docket No. H-033-d)". Federal Register, v. 57, no. 110, June 8, 1992.

19. Y. Honda, C. Beall, E. Delzell, K. Oestenstad, I. Brill, and R. Matthews (2002). "Mortality Among Workers at a Talc Mining and Milling Facility". American Journal of Occupational Hygiene, 46, p. 575-585.

20. A. G. Wylie, K. F. Bailey, J. W. Kelse, R. J. Lee (1993). "The Importance of Width in Asbestos Fiber Carcinogenicity and Its Implications for Public Policy". American Industrial Hygiene Association Journal, 54(5) p. 239-252.

21. E. B. Illren (2004). "The Biology of Cleavage Fragments: A Brief Synthesis and Analysis of Current Knowledge". Indoor Building Environment, 13, p. 343-356.

22. ATSDR (2002). "Expert Panel on Health Effects of Asbestos and Synthetic Vitreous Fibers (SVF): The Influence of Fiber Length; Premeeting Comments". October 29-30, 2002, Agency for Toxic Substances and Disease Registry, New York, NY.

23. NSSGA (2002). National Stone, Sand and Gravel Association Panel Testimony before the Mine Safety and Health Administration (MSHA) on its Advanced Notice of Proposed Rulemaking for Asbestos, Charlottesville, VA, June 20, 2002.

24. K. F. Bailey, J. Kelse, A. G. Wylie, R. J. Lee (2002). "The Asbestiform and Prismatic Mineral Growth Habit and their Relationship to Cancer Studies". p. 64. Submitted to MSHA's Asbestos Docket, June, 2002.

25. R. L. Perkins and B. W. Harvey (1993). "Method for the Determination of Asbestos in Bulk Building Materials". U.S. Environmental Protection Agency, EPA/600/R-93/116, July 1993.

26. E. D. Kuempel, L. T. Stayner, J. D. Dement, S. J. Gilbert and M. J. Hein (2006). "Fiber Size-Specific Exposure Estimates and Updated Mortality Analysis of Chrysotile Asbestos Textile Workers". Presented at the Society of Toxicology Meeting, March 6, 2006.

27. A. G. Wylie and K. F. Bailey (1992). "The Mineralogy and Size of Airborne Chrysotile and Rock Fragments: Ramifications of Using the NIOSH 7400 Method". American Industrial Hygiene Association Journal 53 (7), p. 442-447.

28. U. S. Consumer Product Safety Commission (1988). "Briefing Package of the CPSC Office of the Secretary on a Petition to Ban Play Sand with Non-Asbestiform Tremolite". October 26, 1988,

29. U. S. Consumer Product Safety Commission (2000). " CPSC Staff Report on Asbestos Fibers in Children's Crayons". August 2000.

30. W. J. Campbell, R. L. Blake, L. L. Brown, E. E. Cather, J. J. Sjoberg (1977). "Selected Silicate Minerals and Their Asbestiform Varieties – Mineralogical Definitions and Identification – Characterization". U. S. Bureau of Mines, U. S. Department of Interior, Information Circular 8751, p. 1-55.

31. A. G. Wylie, R. J. Virta, and E. Russek (1985). "Characterizing and Discriminating Airborne Amphibole Cleavage Fragments and Amosite Fibers – Implications for the NIOSH Method". American Industrial Hygiene Association Journal 46(4), p. 197-201.

32. A. G. Wylie (1988). "Discriminating Amphibole Cleavage Fragments from Asbestos: Rationale and Methodology". Exposure Assessment and Control, Asbestos /Other Fibrous Material, p. 1065-1069.

33. D. W. Berman (2006). "Evaluation of the Approach recently Proposed for Assessing Asbestos-Related Risk in El Dorado County, California. Report to National Stone, Sand and Gravel Association.

34. A. G. Wylie (2005). Letter Review of the RJ Lee Inc. Report: "Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project". Prepared for the National Stone, Sand and Gravel Association, Project LSH306975.

35. A. M. Langer (2005). Letter Review of the RJ Lee Inc. Report: "Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project". Prepared for the National Stone, Sand and Gravel Association, Project LSH306975.

36. M. Ross (2005). Letter Review of the RJ Lee Inc. Report: "Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project". Prepared for the National Stone, Sand and Gravel Association, Project LSH306975.

37. J. Addison (2006). Letter Review of the RJ Lee Inc. Report: "Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project". Prepared for the National Stone, Sand and Gravel Association, Project LSH306975. March 23, 2006.

38. C. Skinner (2006). Letter Review to Laura Gill, Chief Administrative Officer, El Dorado County, California Regarding the EPA Report and the R. J. Lee Group Report. February 24, 2006.

39. International Organization for Standardization (1995). "Ambient Air – Determination of Asbestos Fibers – Direct-Transfer Transmission Electron Microscopy Method". ISO 10312.

40. M. Dorling and J. Zussman (1987). "Characteristics of Asbestiform and Non-asbestiform Calcic Amphiboles". Lithos, 20, p. 469-489.

41. E.S. Dana (1920). "System of Mineralogy of James Dwight Dana 1837 -1898: Descriptive Mineralogy". 6th Edition, John Wiley and Sons, New York, p.1134.

42. G. Meeker (2006). "Letter to Dr. Vicki Barber, in Response to February 1, 2006 email to Robert Virta, USGS mineralogist, Regarding Questions of NOA Properties as Discussed in the RJ Lee Group Report.

43. National Institute of Standards and Technology (NIST). Certificate of Analysis for SRM 1867 and 1867a.

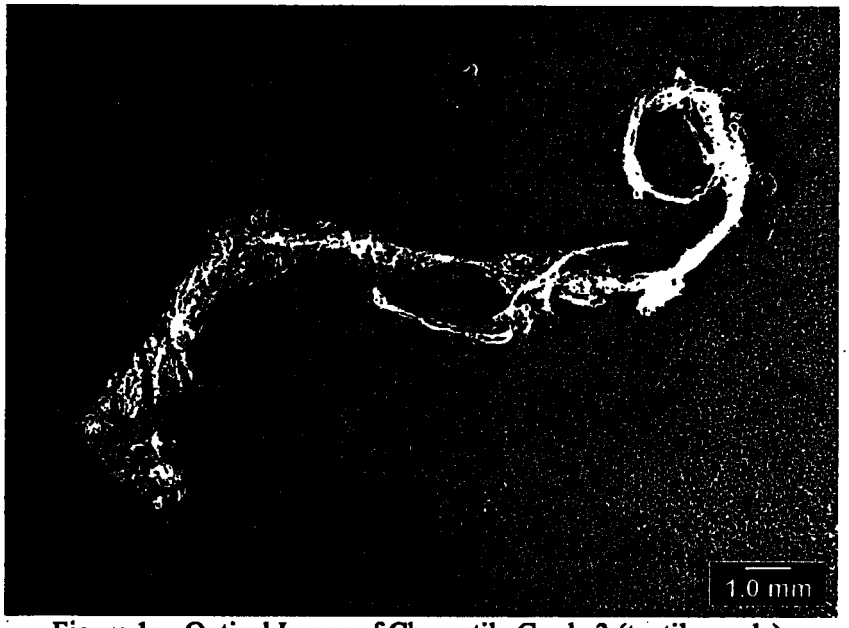


Figure 1a. Optical Image of Chrysotile Grade 3 (textile grade).

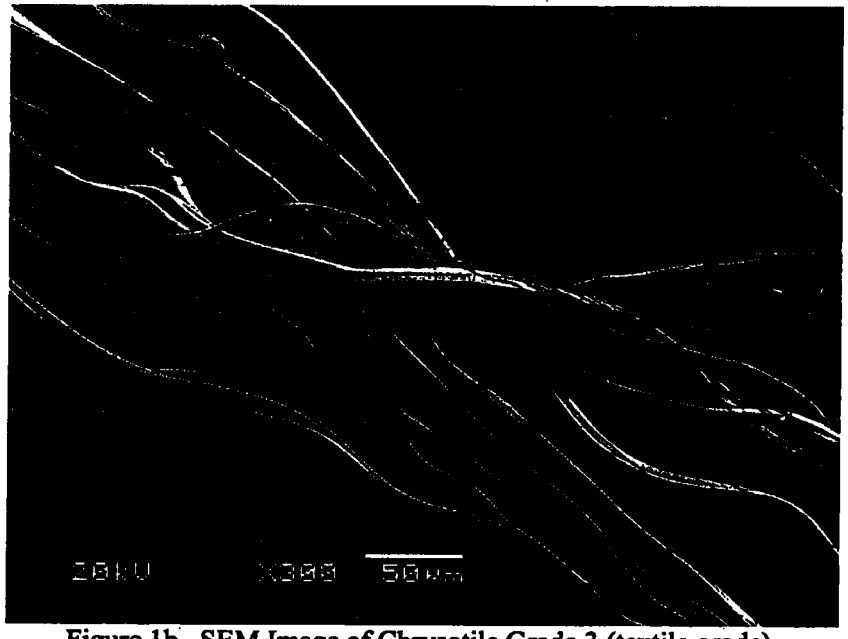


Figure 1b. SEM Image of Chrysotile Grade 3 (textile grade).

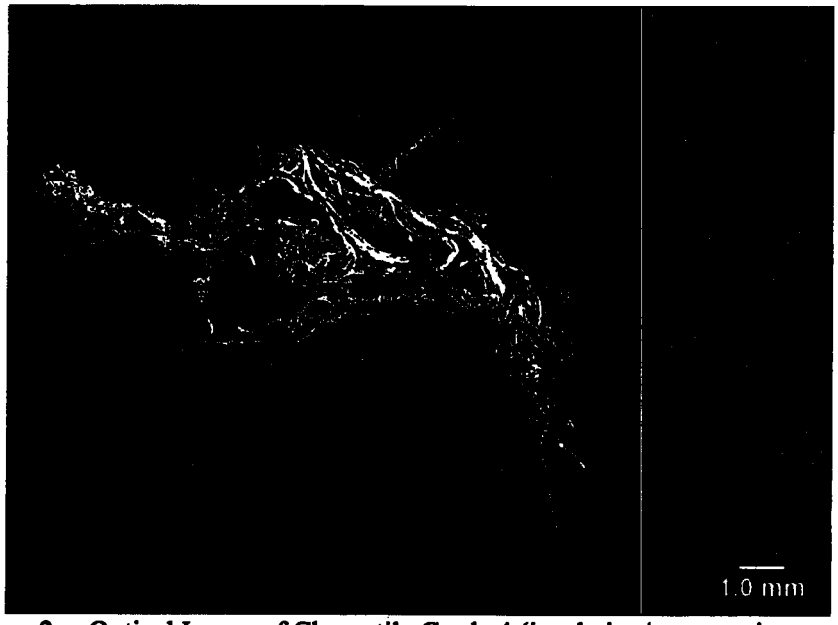


Figure 2a. Optical Image of Chrysotile Grade 4 (insulation/cement pipe grade).

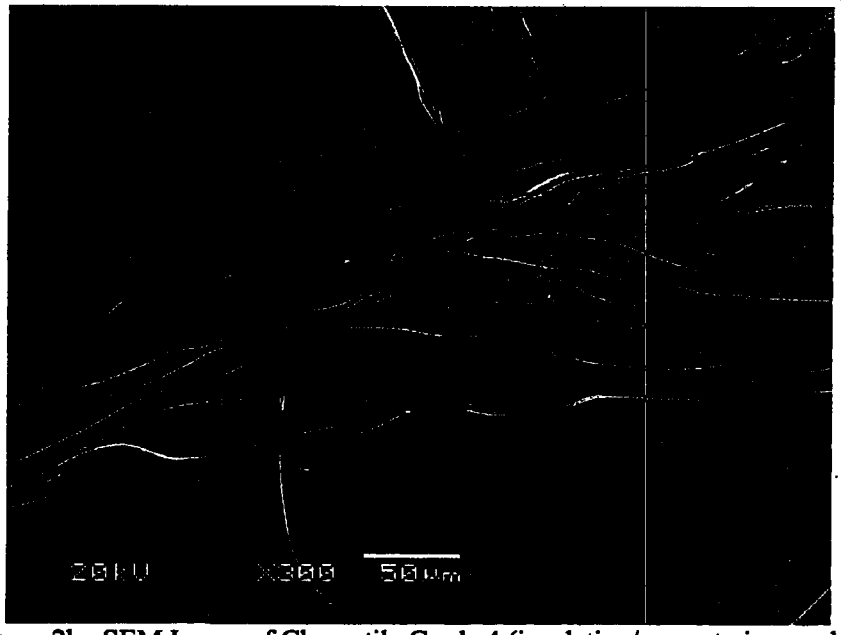


Figure 2b. SEM Image of Chrysotile Grade 4 (insulation/cement pipe grade).

Figure 3a. Optical Image of Chrysotile Grade 7 (friction parts grade).

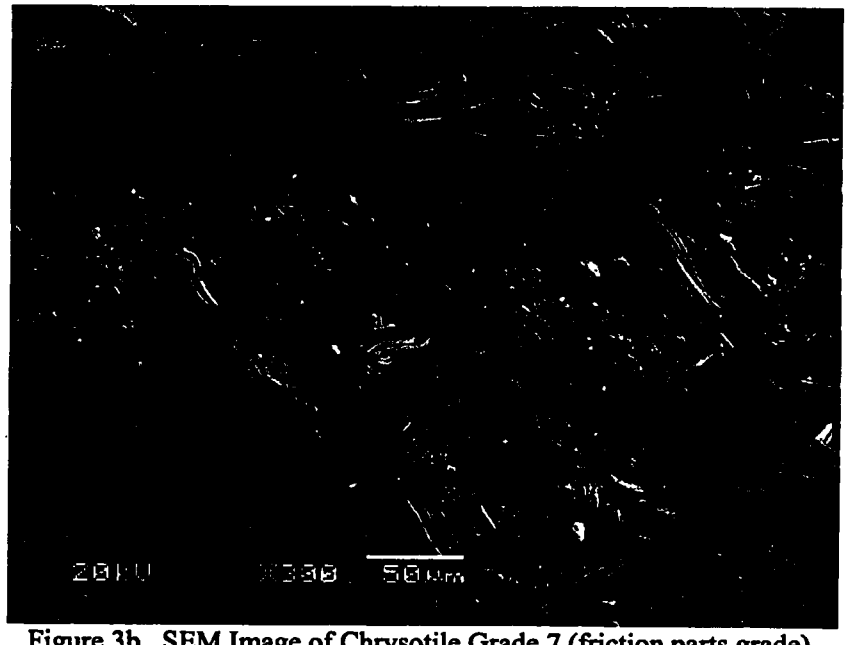


Figure 3b. SEM Image of Chrysotile Grade 7 (friction parts grade).

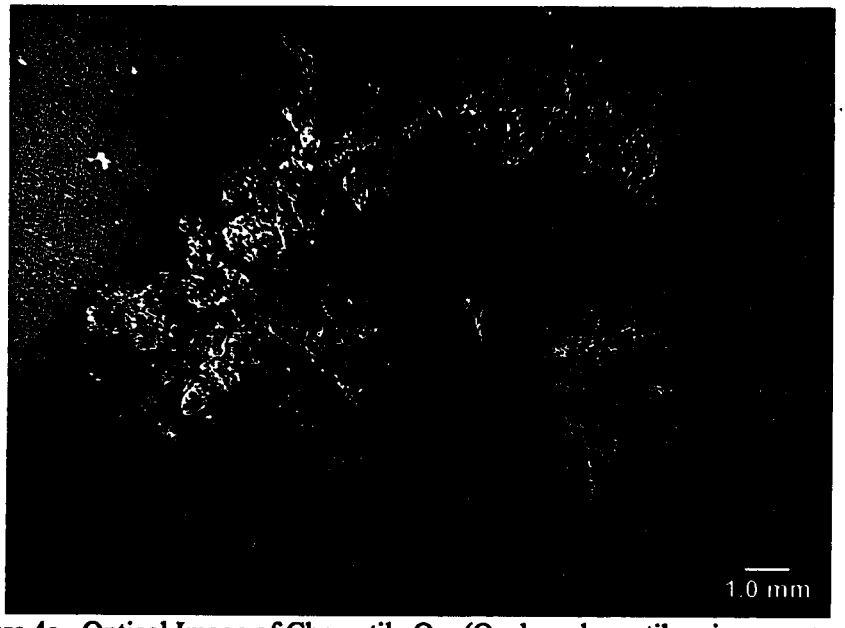


Figure 4a. Optical Image of Chrysotile Ore (Quebec chrysotile miner exposure).

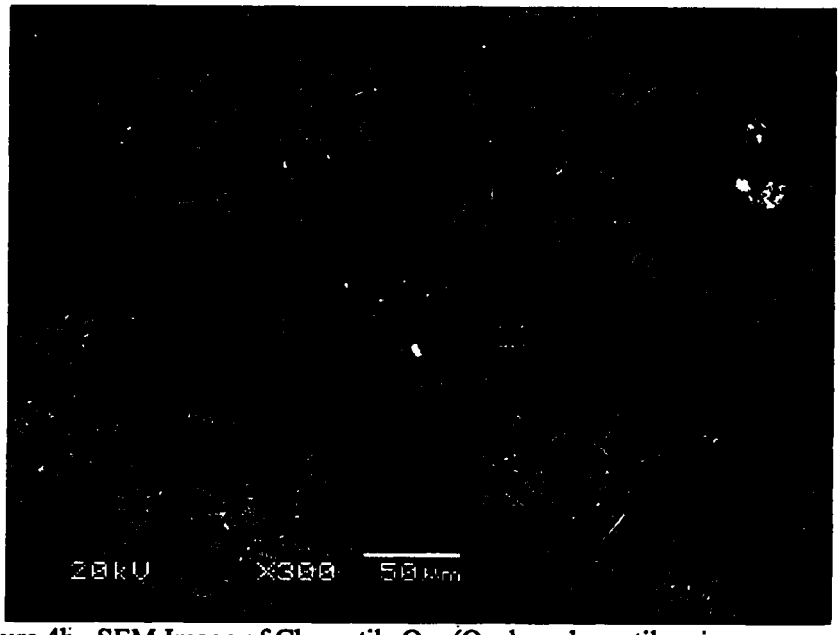


Figure 4b. SEM Image of Chrysotile Ore (Quebec chrysotile miner exposure).

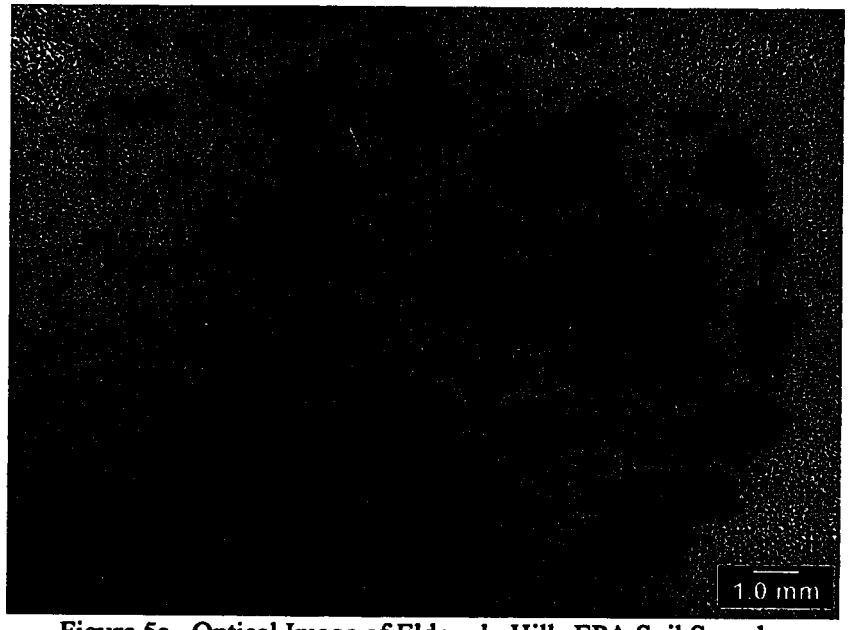


Figure 5a. Optical Image of Eldorado Hills EPA Soil Sample.

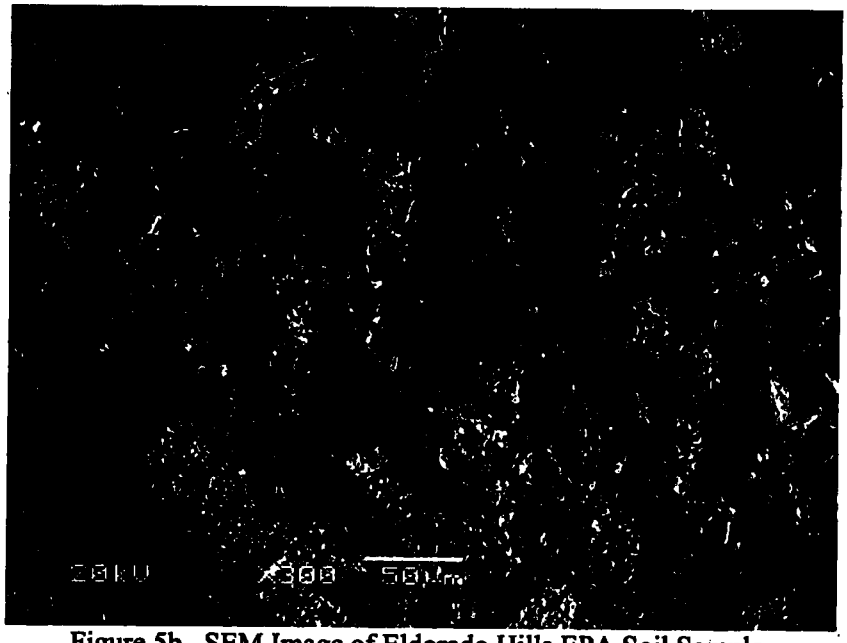


Figure 5b. SEM Image of Eldorado Hills EPA Soil Sample.

An Evaluation of the Risks of Lung Cancer and Mesothelioma from Exposure to Amphibole Cleavage Fragments

John F. Gamble, Ph. D.

International Environmental Research Foundation

Post Office Box 3459

Grand Central Station

New York, New York 10163-3459

And

Graham W. Gibbs, Ph.D.

Safety Health Environment International Consultants Corp.

38 Athabasca Avenue, Suite 101,

Devon, Alberta, Canada

Peer Reviewed, Revised and Accepted for Publication in Proceedings of the *International Symposium on the Health Hazard Evaluation of Fibrous Particles Associated with Taconite and the Adjacent Duluth Complex*
St Paul, Minnesota
March 30 – April 1, 2003

E-Mail: jfgamble@ierfinc.org

ABSTRACT

Amphiboles are hydrated mineral silicates five of which occur in asbestiform habits as asbestos [grunerite (amosite) asbestos, riebeckite (crocidolite) asbestos, anthophyllite asbestos, tremolite asbestos and actinolite asbestos] and non-asbestiform habits (grunerite, riebeckite, anthophyllite, tremolite and actinolite). The asbestiform varieties are characterized by long, thin fibers while non-asbestiform varieties such as cleavage fragments form short fibers with larger widths. The US regulatory method for counting asbestos fibers (aspect ratio ≥3:1, length ≥ 5 μm) does not distinguish between asbestos and cleavage fragments. The method biases toward increased counts of non-asbestiform cleavage fragments compared to long, thin asbestos fibers. One consequence of this regulatory approach is that workers can be erroneously classified as exposed to concentrations of asbestos (asbestiform amphiboles) above the U.S. 0.1f/mL exposure standard when in fact they are not exposed to asbestos at all but non-asbestiform amphibole cleavage fragments. Another consequence is that the known carcinogenic effects of asbestos may be falsely attributed to non-asbestiform amphibole cleavage fragments of the same mineral. The purpose of this review is to assess whether amphibole cleavage fragments pose the same risk of lung cancer and mesothelioma characteristic of amphibole asbestos fibers.

We identified three groups of workers exposed to non-asbestiform amphiboles: two groups exposed to grunerite (Homestake gold miners and taconite miners) and one group exposed to industrial talc containing non-asbestiform tremolite and anthophyllite in St Lawrence County, NY. In addition to assessing strength of association and exposure-response trends in the non-asbestiform amphibole cohorts, comparisons were also made with cohorts exposed to the asbestiform counterpart (positive control) and cohorts exposed to the mineral (e.g. talc) that does not contain amphiboles (negative controls).

The cohorts exposed to non-asbestiform amphiboles had no excesses of lung cancer or mesothelioma. Similar results were observed in the negative control groups, in stark contrast to the excess risks of asbestos-related disease found in the asbestos cohorts. The only possible exception is the two-fold increased risk of lung cancer where exposure was to industrial talc containing cleavage fragments of tremolite and anthophyllite. However, this risk is not considered attributable to the talc or amphibole cleavage fragments for several reasons. A similar increased risk of lung cancer was found in Vermont talc workers, studied in the same time period. Their exposure was to relatively pure talc. There was no relationship between lung cancer mortality and exposure measured as mg/m^3-years and years worked. A case-control study reported that all the lung cancer cases were smokers (or former smokers) and attributed the excess to smoking. There were two mesothelioma cases among the NY State talc workers exposed to cleavage fragments of tremolite and anthophyllite, but talc is not a plausible cause because of too short latency and potential for previous asbestos exposure. The positive controls of tremolite asbestos and anthophyllite asbestos exposed workers showed excess risks of both lung cancer and mesothelioma and positive exposure-response trends. St Lawrence, NY talc does not produce mesotheliomas in animals while amphibole asbestos does. In sum, the weight of evidence fully supports a conclusion that non-asbestiform amphiboles do not increase the risk of lung cancer or mesothelioma.

INTRODUCTION

Asbestos is a generic term applied to a group of hydrated fibrous mineral silicates. Their asbestiform habit permits them to be easily separated into long, thin, flexible, strong fibers and ultimately fibrils (single fibers). Included are the asbestiform serpentine (chrysotile) and the asbestiform amphiboles, riebeckite (crocidolite) asbestos, anthophyllite asbestos, grunerite (amosite) asbestos, tremolite asbestos and actinolite asbestos. These minerals also crystallize with non-asbestiform habits, their counterparts being lizardite or antigorite (chrysotile), riebeckite, anthophyllite, grunerite, tremolite and actinolite respectively. Crystal habit is a description of the shapes in which a certain mineral is likely to occur, both in nature and when grown synthetically. Tremolite is a mineral in the tremolite-ferro-actinolite series that has fewer than 0.5 atoms of iron, and more than 4.5 atoms of magnesium per formula unit; actinolite has between 0.5 and 2.5 atoms of iron, and 2.5 atoms of magnesium per formula unit; ferro-actinolite has more than 2.5 atoms of iron per formula unit with the balance being magnesium.

By the early 1970's, airborne concentrations of asbestos fiber were being measured using "the membrane filter phase contrast method (PCM)". In many countries, including the U.S.A., this method was adopted for the regulatory control of asbestos. Fundamental to the method was the definition of a fiber as an elongated particle having a length: breadth ratio (aspect ratio) of at least 3:1 and a minimum length of 5 micrometers (μm). Such a definition does not allow the microcopist to distinguish between asbestos fibers and non-asbestos amphibole particles. Consequently, in work environments where there exist many elongated particles meeting the PCM fiber definition, they are counted as if they are "asbestos" even if they are neither asbestos minerals nor even amphiboles. This results in concern by workers and health professionals about health risks and potential economic impacts for companies mining ore deposits where amphibole minerals are present. This is because the amphiboles have cleavage planes such that when they are crushed they produce elongated prismatic particles called cleavage fragments.

All amphiboles that were once exploited commercially as asbestos have non-asbestiform counterparts. Hence, workers in industries where amphibole cleavage fragments are present, but not asbestos, are often erroneously reported as being exposed to asbestos based on current regulatory counting strategies and protocols. On the other hand, the evidence concerning the health consequences of exposure to cleavage fragments has never been widely understood. Industries involving exposure to cleavage fragments should not be exempt from similar controls to the asbestos industries, if elongated particles meeting the PCM definition of fibers pose qualitatively and quantitatively the same levels of health risk as their asbestiform counterparts. However, if cleavage fragments pose no or a lesser risk than the asbestos minerals, they should be regulated accordingly.

The purpose of this paper is to compare, as far as possible, the cancer risks (lung cancer and mesothelioma) for workers exposed to airborne amphibole cleavage fragments with

those associated with exposure to amphibole analogues that formed asbestos fibers. Pneumoconiosis risk will not be compared because some of the minerals associated with the amphibole cleavage fragments are recognized in their own right as causing lung fibrosis (e.g.: talc and crystalline silica). However, pneumoconiosis is sometimes used to assess whether exposure is high enough and latency long enough to detect carcinogenic risk and to evaluate the exposure response.

METHODS

The extent to which the carcinogenic risks of exposure to cleavage fragments differ from those associated with exposure to asbestos was examined in several ways.

The potential of particles to cause health effects depends on the characteristics of the particles (e.g.: size, shape, respirability, solubility, toxicity, carcinogenic potential), the level and duration of exposure as well as host and other factors. It is important to determine whether amphibole cleavage fragments differ sufficiently from asbestos fibers for them to pose different levels of health risk than their asbestos counterparts. To do this requires examination of the characteristics of the particle such as dimensions, shape and density that influence fiber respirability, and fiber dimensions and biopersistence that influence carcinogenicity.

Mesothelioma and lung cancer are the health endpoints examined for comparison of the relative effects of non-asbestiform and asbestiform amphiboles. Mesothelioma is considered the more important indicator because it is both more specific and perhaps more sensitive than lung cancer. Mesothelioma is a rare cancer that acts as a marker or "signal" tumor, which is primarily associated with exposure to amphibole asbestos and has occurred in some situations after what appears to be exposure at quite low concentrations. Lung cancer is more subject to being caused by confounding exposures such as smoking, which is the primary cause of lung cancer. Thus while lung cancer might be caused by asbestos, it is an effect that is not specific to asbestos exposure.

If smoking prevalence is not known, the effects of dust exposure and smoking in the occurrence of lung cancer cannot readily be distinguished. Mesothelioma is a more sensitive and specific indicator of amphibole asbestos exposure than lung cancer in that pleural mesothelioma may occur following what are ostensibly brief exposures (Roggli 1990) and up to 80% of the cases in males may be associated with asbestos exposure (Price and Ware 2004). The exposure-response curve is thought to be non-linear for both mesothelioma and lung cancer. While the shapes of relationships are still subject to debate, pleural mesothelioma has been reported to increase less than linearly with cumulative dose. For peritoneal mesothelioma the risk is thought to be proportional to the square of cumulative exposure while for lung cancer the exposure-response lies between linear and square of cumulative exposure (Hodgson and Darnton, 2000). As some mesothelioma have been reported to occur after relatively low and perhaps brief exposures one might anticipate that if amphibole cleavage fragments act like asbestos in causing mesothelioma there might be some cases even if cleavage fragment exposures were low. For mesothelioma to be attributed to amphibole cleavage fragments the time since first exposure must be more than about 20 years and there should be no previous exposure to asbestos or other confounding etiological factors.

The mortality from lung cancer and mesothelioma are compared to that expected in age- and sex-adjusted external populations. The comparison measure is the standardised

observed / expected mortality ratio or Standardized Mortality Ratio (SMR). When the incidence of lung cancer and mesothelioma are compared to that expected in age- and sex-adjusted external populations, the comparison measure is the standardised observed/expected cancer incidence ratio or Standardized Incidence Ratio (SIR). External comparisons for assessing lung cancer risk have inherent limitations such as differences in smoking and lifestyle between the study population and the external referent population. It is generally not feasible to adjust for these differences. An SMR less than 1.5 or a statistically nonsignificant SMR is suggestive, but not conclusive, of no association. A deficit in the lung cancer SMR could be due to exposure levels below a no-effect threshold, or a few highly exposed workers diluted by many workers with low exposure, or negative confounding due to a low prevalence of smoking. A nonsignificant SMR might be due to the small size of the study population and the low power of the study to detect significant differences. Similarly, a positive finding of lung cancer could be due to differences in smoking prevalence between the study and reference populations rather than exposure to non-asbestiform amphiboles.

For mesothelioma, external comparisons using an SMR are often not possible because the expected number of cases is not known or not estimated. Therefore an internal proportional mortality ratio (PMR) is used to estimate risk of mesothelioma. PMR's have their limitations which must be taken into account when using them. For example, as a PMR can increase with length of follow-up of a cohort, attention must be given to the comparability of the follow-up period. Age differences in populations being compared are important as age determines the nature of diseases from which people die as well as the frequency of death. The ratio with total deaths to some extent adjusts for both differences in follow-up and age. Era of death may be important because of diagnostic trends. Never-the-less, comparison of PMRs between non-asbestiform amphibole-exposed and asbestos-exposed populations is a useful way to examine the question of whether non-asbestiform amphiboles cause cancer at the same rates as asbestiform amphiboles.

The actual measured risks of lung cancer and mesothelioma in persons exposed to amphibole cleavage fragments is compared to workers exposed to asbestiform amphiboles as follows:

- The lung cancer and mesothelioma experience of workers exposed to amphibole cleavage fragments is compared with the experience of workers exposed to their asbestiform equivalents. There are three main ore bodies containing non-asbestiform amphiboles where epidemiological studies have been conducted. These are a gold mine in South Dakota (grunerite-cummingtonite exposure), taconite mines in Minnesota (grunerite and other non-asbestiform amphiboles) and a talc mine in St Lawrence County, New York State (transition minerals, non-asbestiform anthophyllite and tremolite). Their experience was compared to that of workers exposed to asbestiform amphiboles. These "positive controls" were in amosite asbestos mines, mills and manufacturing facilities, anthophyllite asbestos mines and vermiculite

mines (exposed to winchite asbestos also known as soda tremolite asbestos). In this report, winchite asbestos from the vermiculite mine in Montana, will be referred to as "tremolite asbestos" as this has been the terminology used in the medical literature.

- The mortality from lung cancer is examined in relation to estimated levels of exposure to "fibers" for workers exposed to asbestos and workers exposed to amphibole cleavage fragments. The existence of a positive gradient of increasing risk with increasing exposure after taking account of potential confounders would be good evidence that the cleavage fragments were posing an increased risk of lung cancer. A negative gradient would be strong evidence against a causal association. The presence or absence of an exposure-response gradient is among the strongest evidence for or against a lung cancer association with cleavage fragment exposure because smoking is the major cause of lung cancer and rarely, if ever, can external comparisons be fully adjusted for smoking.
- The lung cancer and mesothelioma experience of workers exposed to dusts from an ore-body containing amphibole cleavage fragments is compared with that of workers exposed to dusts from a similar ore-body which does not contain asbestos or amphibole cleavage fragments. This is called a negative control. If the experience of the amphibole cleavage fragment exposed workers were worse than that of the negative control (non-cleavage fragment exposed workers), this would be suggestive of an increased risk due to the presence of asbestos cleavage fragments.
- In order to investigate this, the mortality for St Lawrence County talc miners is compared to that of talc miners where it is claimed amphiboles are not present. Also, the mortality of iron ore miners exposed to taconite rocks containing non-asbestiform grunerite and actinolite is compared to that of miners exposed to iron ore (hematite) which does not contain amphiboles.
- The biological plausibility of a difference in the potential of amphibole cleavage fragments to cause cancer compared to amphibole asbestos fibers was assessed by review of the results of toxicological studies involving asbestos and amphibole cleavage fragments. There is a clear pattern of an increased incidence of mesothelioma in animals exposed to amphibole asbestos. Observing a similar pattern for animals exposed to non-asbestiform amphiboles would be evidence supporting the hypothesis that non-asbestiform amphiboles pose a carcinogenic hazard similar to asbestos. The lack of an increased incidence of mesothelioma would be strong evidence against the hypothesis.

THE AMPHIBOLES

The crystallographic structure of amphiboles consists of double chains of silica tetrahedra. Their general chemistry incorporates $(Si, Al)_8 O_{22}(OH)_2$. The amphibole group of minerals is made up of a number of mineral series. These series result from the substitution of different elements in the structure. For example tremolite and actinolite are part of a homologous series of minerals – tremolite-actinolite-ferro-actinolite with chemistry $Ca_2 (Mg\ Fe)_5 Si_8 O_{22} (OH)_2$. Actinolite is $Ca_2 (Mg_{4.5} Fe_{0.5}) Si_8 O_{22} (OH)_2$ - $Ca_2 (Mg_{2.5}Fe_{2.5}) Si_8 O_{22} (OH)_2$. Ferro-actinolite is $Ca_2 (Mg_{2.5} Fe_{2.5}) Si_8 O_{22} (OH)_2$ - $Ca_2Fe_5 Si_8 O_{22} (OH)_2$. Actinolite with less than $Fe_{0.5}$ would be tremolite.

In practice, these minerals can have a fairly wide range of composition within the broad range of substitutions possible. The mineral names are defined where the ranges of the substituted elements fall within certain arbitrary boundaries.

Grunerite is a member of the mineral series cummingtonite-grunerite with chemistry $(Mg, Fe)_7 Si_8O_{22} (OH)_2$. As noted above, the asbestiform variety of grunerite is "amosite". As with the tremolite-ferro-actinolite series, the minerals in this series may display a range of compositions.

Anthophyllite occurs as asbestos and in a non-fibrous form and is an end member of the anthophyllite-ferro-anthophyllite series, which is chemically $(Mg\ Fe^{2+})_7 Si_8 O_{22} (OH)_2$. Anthophyllite is the name reserved for the orthorhombic Mg Fe amphibole where the ratio of Mg/ (Mg+Fe) is greater than 0.5; a lower amount of magnesium in the same type of amphibole requires the name ferro-anthophyllite.

Non-asbestiform riebeckite and crocidolite asbestos have the same chemistry which is $Na_2 Fe_3^{2+} Fe_2^{3+} Si_8 O_{22} (OH)_2$. Amphiboles exhibit prismatic cleavage, a property of nearly all samples of the amphiboles regardless of habit. There are two cleavage directions, both parallel to the length of the double-silicate chains. Cleavage across the crystal is usually poor so that the fracture of amphiboles produces long rods or prisms and repeated cleavage produces thinner rods with a rhombic outline consisting of bundles of I beams (i.e.: structural units of the amphibole) (Skinner et al. 1988). The presence of twinning or chain width errors may results in an additional direction of weakness parallel to the length, enhancing the aspect ratio of cleavage fragments (Langer et al. 1991).

PROPERTIES OF ASBESTIFORM AND NON-ASBESTIFORM AMPHIBOLES

While the chemical compositions of the asbestiform and non-asbestiform amphibole minerals are identical, the characteristics resulting from their differences in crystal habit are significant. The properties of the amphibole asbestos minerals include fibrous habit with parallel fibers occurring in bundles, fiber bundles with split or splayed ends, fibers showing curvature and fibers with high tensile strength. The high tensile strength and axial nature of asbestos means the diameters of asbestos fibrils are largely unaffected by milling. On the other hand, the low tensile strength of non-asbestiform amphiboles means that milling can reduce both particle length and width. The asbestos fibers have good heat insulation qualities, low electrical conductivity, fire resistance, and suitability for weaving. All asbestos minerals separate readily into long flexible fibrils with diameters less than about 0.5 μm and with aspect ratios (length: width ratios) ranging to well over 10,000 (Ross 1978).

In the hand specimen (that is a sample of the rock as it occurs in nature), the appearance of the non-asbestos minerals is distinctly different from that of the asbestos minerals. This difference persists when viewed by optical and electron microscopy where the non-asbestiform minerals appear as blocks, chunks or slightly elongated particles in contrast to the very evident fibrous nature of asbestos. The non-asbestiform counterparts tend not to grow with parallel alignment. The crystals normally fracture when crushed forming cleavage fragments, some of which may appear as acicular or needle-like crystals because of the way in which amphibole minerals cleave. These cleavage fragments have diameters which on average, are much larger than those of asbestos fibers of the same length. Some asbestiform tremolite fibers with the majority of fiber diameters exceeding 0.25 μm, tested by intra-peritoneal injection in rats were found to be highly carcinogenic (Davis et al. 1991 Lee 1990). However, almost 70% of the fibers had aspect ratios greater than 10:1, 42% greater than 15:1 and 25% had aspect ratios more than 20:1. This contrasts with the observations that only about 6% of the aspect ratios of cleavage fragments exceed 15:1. The diameters of cleavage fragments appear to be rarely less than 0.25 μm (Table 1).

Fiber Diameters: The aerodynamic behavior of fibers is determined mainly by their diameter (Timbrell 1982). The majority of airborne asbestos fibers have diameters less than 0.25 μm making virtually all airborne fibers, respirable. In contrast, only very small percentages of non-asbestiform cleavage fragments have diameters less than 0.25μm (Table 1).

For the same length distribution, counting fibers by PCM will, based on fiber diameter differences, lead to higher counts of non-asbestiform cleavage fragments than asbestos fibers, because of their visibility by PCM. On the other hand, assuming the same density for fibers as for cleavage fragments, the respirability (i.e.: ability of particles to enter the alveolar regions of the lung) of the cleavage fragments will be less that that of asbestos

fibers because of their larger diameters. Thus, the PCM method as presently formulated is more stringent for cleavage fragments than for asbestos fibers.

Fiber width is an important parameter determining the potential for causing both lung cancer and mesothelioma. The characteristics of non-asbestiform fiber populations are contrary to the hypothesis of carcinogenicity, while the abundance of thin asbestos fibers supports the hypothesis (Wylie et al. 1993). The evidence from experimental animal studies indicate fibers > 1 μm show no dose-response relationship with tumor incidence (<30% of population of non-asbestiform fibers ≥5 μm long are < 1 μm wide). For fibers < 1 μm (and > 5 μm long) there is an S-shaped dose-response curve with a threshold and then rapid increase in tumor incidence as the number of thin fibers increases. In populations of asbestos fibers >90% are < 1 μm wide and ≥ 5 μm long. Fiber width is also a major factor determining access to the lung. Even long, thin fibers (such as 200 μm long or more) are respirable and are found in lung tissue, while respirability decreases as width increases. Wide diameter cleavage fragments are more likely to be deposited in the upper airways and never gain access to the lower lung to cause disease. The potential for asbestos fiber bundles to disaggregate into increased numbers of even thinner fibers *in vivo* is one of their hazardous features and is not a characteristic of non-asbestiform minerals.

While it has been argued that a major determinant of carcinogenic potential is decreasing fiber width (Wylie et al. 1993), the precise role of the single parameter, diameter in carcinogenesis is still not clear (Addison and McConnell this volume).

Fiber Length: While the majority of asbestos fibers are in fact short (less than 5μm) there are airborne amphibole fibers which exceed 100 μm in length. Complete particle size data (length vs. diameter) on distributions of airborne cleavage fragments and asbestos fibers are extremely limited in number, making it difficult to compare length distribution differences. What data are available indicate that asbestos fibers are longer. For example, Dement et al. (1976) observed that the median length of "fibers" in the airborne dust in the South Dakota Homestake Gold mine was 1.10 μm as seen using scanning electron microscopy. This is less than the median length of airborne grunerite (amosite) asbestos fibers in South Africa mines and mills which were 1.83 μm and 2.53 μm respectively (Gibbs and Hwang 1980) and of grunerite (amosite) asbestos from a pipe insulation operation, 4.9 μm (Dement et al. 1976).

There is other evidence for a clear mineralogical difference between grunerite (amosite) asbestos and grunerite cleavage fragments. Virta et al. (1983) examined airborne particles of grunerite from the Homestake gold mine in South Dakota, particles of cummingtonite, hornblende and actinolite from the Peter Mitchell iron ore pit in Minnesota and particles of grunerite asbestos samples from a shipyard and an electric company. Hornblende is an amphibole that is similar to the tremolite-ferro-actinolite series but with aluminum substituted for some of the iron-magnesium as well as for some of the silicon in order to maintain the stoichiometric balance. There were two distinct particle size distributions. The non-asbestiform grunerite distributions from the mining sites were short, wide fibers

(average length to width equal to 4.6 μm x 1.1 μm and 5.5 μm x 1.2 μm). The amosite fibers from the industrial sites were longer and narrower (average length to width equal to 8.2 μm x 0.4 μm and 15.6 μm x 0.5 μm respectively). Although the populations of grunerite cleavage fragment and grunerite asbestos are distinct, at the submicroscopic level it may be very difficult to be certain about the specific identity of an individual particle and may be extremely difficult, if not impossible to distinguish asbestos and non-asbestiform particles among the small number of fibers where the two fiber population overlap, especially when the source of the fiber is unknown (Langer et al. 1979).

The New York State talc deposit has been extensively studied for its mineralogy and presence of fibers and cleavage fragments. Commercially important deposits of zinc, lead, talc and wollastonite are found in the Grenville Series of sedimentary rock in St Lawrence County of NY. Three zinc mines and eleven talc mines have been worked in the area between Balmat Corners and Edwards, NY, which are about 8 miles apart. All of these holdings contain some non-asbestiform tremolite, encountered as either a gangue mineral or component of the recovered ore. Anthophyllite and transitional metals have also been identified in variable amounts both between and within mines. We will refer to the NY state talc as St Lawrence County talc.

Campbell et al. (1979) note that 5-10% of the earth's crust is amphiboles and therefore many mining industries have amphibole fragments in the gangue mineral tailings. There are at least 3 habits of non-asbestiform tremolite, none of which have the long, thin fibers characteristic of tremolite asbestos as shown in Table 2.

Long narrow fibers have been shown experimentally to be best capable of inducing mesothelioma when placed directly onto the pleura in experimental animals (Stanton et al. 1981). As there are likely to be fewer long fibers and fewer narrow diameter "fibers" in the case of exposure to amphibole cleavage fragments, compared to asbestos, it would be anticipated that cleavage fragments would pose lower carcinogenic risk.

Aspect Ratios: Asbestos fibers have thin diameters and do not readily break transversely. As a result, length/width ratios can be quite high. All "fibers" will by definition have aspect ratios >3:1. Around 30% of asbestos fibers will have aspect ratios >10:1 and nearly 20% greater than 20:1. There were very few cleavage fragments with aspect ratios greater than 10:1 The common blocky variety of non-asbestiform tremolite had less than 2% in the >10:1 class. The acicular and fibrous habits had more particles in the range between 10:1 and 20:1 category than did the blocky variety, but none of the non-asbestiform varieties had more than 0.5% particles in the range between 20:1 and 50:1 and none had any particles >50:1 Nearly 90% of the blocky and acicular habits did not meet the regulatory definition of a fiber. If only fibers that meet regulatory dimensions are counted, 1/100 of non-asbestiform particles have aspect ratios >20:1 while about 35/100 asbestiform tremolite particles have >20:1 aspect ratios (Table 2). A composite aspect ratio distribution reported in the Pictorial Atlas of Mineral Fibers (This Monograph) showed that for non-asbestiform particles with an aspect ratio of 3:1 or greater and length greater

than 5um, 6% on average exceed an aspect ratio of 15:1 and for asbestiform particles, 80% on average exceed an aspect ratio of 15:1. The 3:1 aspect ratio is used principally to eliminate particulates and fiber clumps and improve the precision and accuracy of fiber counts. It is not a defining characteristic of asbestos fibers (Langer et al. 1991).

Wylie et al. (1993) points out that aspect ratio is not a useful parameter for sizing as it is dimensionless, provides no information on width, shows no association with risk of disease, and therefore is of little use in the discussion of risk or exposure.

Biopersistence: As far as we were able to ascertain, there have been no systematic studies of the biopersistence of cleavage fragments. It is known that for long amphibole asbestos fibers, the half-life is extremely long (Berry 1999). However, short fibers (i.e.: less than 20 μm in length) can be removed from the lung by macrophage action (Allison 1973; Bernstein et al. 1994). For later phases of lung clearance, particle solubility is a key factor. In the absence of data, there is no basis for concluding that cleavage fragments will be removed any faster than asbestos fibers during that phase. However, because of their shorter lengths, cleavage fragments are much more likely to be removed more rapidly than amphibole asbestos fibers during the early lung clearance phase. This will reduce their potential for carcinogenic action.

Ilgren (2004) notes dissimilarities that make cleavage fragments much less biopersistent than amphibole asbestos fibers. Surfaces of cleavage fragments have a high density of surface defects, which are preferred sites for dissolution from intracellular acidic enzymes of phagocytic cells that have engulfed them. Amphibole asbestos fibers are smooth and defect free and highly acid resistant. Cleavage fragments are weak, brittle and inflexible because of there weak surface structure, which is further weakened by chemical dissolution. The tensile strength of amphibole asbestos fibers is 20-115 times greater than the non-asbestiform amphibole variety. This difference becomes greater as width decreases and biological relevance more pronounced. When long, thin biologically relevant cleavage fragments are deposited in the lung alveoli and engulfed by macrophages, the fragment begins to dissolve on all surfaces. They are already weak and inflexible and become thinner and weaker (greater surface area; more surface defects) with increasing susceptibility to chemical dissolution and breakage. The defect-free surface of the amphibole asbestos fiber is better able to resist acid attack. Many of the asbestos fibers are too long to be completely engulfed. Attempts at engulfment produce protein deposits that form an "asbestos body" and eventual death of the cell. In short, biopersistence is a characteristic of carcinogenesis. It is reasonable to conclude that cleavage fragments are likely to be far less bio-persistent than asbestos fibers.

Nolan et al (1991) compared activity of tremolite cleavage fragments with that of samples of tremolite-actinolite asbestos. For the same surface area, tremolite cleavage fragments had lower ability to alter the permeability of red blood cells than amosite and approximately the same membranolytic activity as anthophyllite and crocidolite. The surface charge of non-asbestos tremolite was about 70% less than asbestos analogues.

Schiller et al (1980) reported that asbestos fibers and cleavage fragments of the same dimensions had the same net negative surface charge. Short fibers and cleavage fragments have a smaller net charge than highly elongated particles.

COMPARISON OF THE RISK OF HEALTH EFFECTS IN PERSONS EXPOSED TO ASBESTIFORM AND NON-ASBESTIFORM GRUNERITE

Grunerite Occurrence: Grunerite is the mineralogically correct name for amphiboles of the cummingtonite-grunerite series in which iron is at the 50% point in the 100 times Fe / (Fe+Mg)) ratio. Amosite (from the "Asbestos Mines of South Africa") is the commercial asbestiform product that was used in insulation and building materials. Grunerite asbestos is no longer mined.

The non-asbestiform variety of cummingtonite-grunerite (C-G) has no commercial use *per se* other than a aggregate but occurs in nature in conjunction with other asbestiform and non-asbestiform amphiboles and other minerals in ore deposits mined for other purposes. In the USA, ore containing C-G has been mined in at least 2 locations. One location is the Homestake gold mine in Lead, SD, where gold had been extracted since 1876. The other location is Mesabi Range where taconite has been mined since the 1950's and shipped to Silver Bay, Minnesota for extraction of iron. Because of its relationship to grunerite (amosite) asbestos, studies were initiated to determine if these minerals had similar pathogenicity. There have been four cohort studies of Homestake gold miners (Gilliam et al. 1976, McDonald et al. 1978, Brown et al. 1986, Steenland and Brown 1995) and two studies of taconite containing amphiboles; one of the Reserve iron deposit (Higgins et al. 1983) and the other of the Erie-Minntac mine (Cooper et al. 1988, 1992) (Table 3).

Taconite iron ore contains actinolite and cummingtonite-grunerite (probably predominantly grunerite). In 1973, elongated grunerite particles, said to be similar to grunerite (amosite) asbestos, were found in the Duluth, Minnesota water supply. The source was mine tailings from the process plant at Silver Bay, Minnesota (MN) serving the Peter Mitchell Pit. In a suit against the Reserve Mining Company, the US Environmental Protection Agency (EPA) claimed that some of the particles were asbestos. This finding initiated a series of studies to determine if there were effects on the Duluth residents (Cook et al. 1974, Masson et al. 1974, Levy et al. 1976, Sigurdson et al. 1981). These studies of human health are not considered further because they are ecological studies without identification of individual exposures or responses, because the route of exposure is via ingestion and because experimental studies and the epidemiological studies described below have provided no evidence in support of any gastrointestinal cancer risk from ingestion. The other health studies are of taconite miners and millers (Clark et al, 1980, Higgins et al. 1983, Cooper et al. 1988, 1992).

A reasonably valid comparison can be made between the health risks of workers exposed to amosite asbestos in mining and manufacture and the health risks of workers involved in the extraction of minerals from ore bodies containing non-asbestiform grunerite.

GRUNERITE (AMOSITE) ASBESTOS

Amosite is the trade name given to a mineral that was previously mined in Penge region in the Transvaal of South Africa. The mineralogical name is grunerite asbestos. In the bulk specimen the fibers can be several inches long. The color, ranging grey to brown depends on whether the fiber was mined from a weathered or un-weathered zone. The size distribution of the airborne fibers in the mine and mill have been reported by Gibbs and Hwang (1980). In mining and milling 12.6% and 6.6% respectively of airborne fibers exceeded 5 μm in length when all particles with length to breadth ratios greater than 3: 1 were counted using transmission electron microscopy combined with light optical microscopy. The median lengths for mining and milling were 1.83 μm and 2.53 μm respectively. The median diameters were 0.20 to 0.26 μm depending on the process and there were no airborne fibers with diameters exceeding 3 μm.

Grunerite (Amosite) Asbestos Exposed Cohort Studies

The studies of cohorts of amosite-exposed workers include miners and millers in South Africa (Sluis-Cremer et al. 1992) and workers engaged in amosite insulation manufacture (Acheson et al. 1984, Seidman et al.1979, 1986, Levin et al. 1998). Cohorts where the exposure also included riebeckite (crocidolite) asbestos and/or chrysotile have been excluded from consideration as the ratios of the risks of mesothelioma associated with these various asbestos fiber-types have been reported to be in the ratio of 500:100:1 for riebeckite (crocidolite) asbestos, grunerite (amosite) asbestos and chrysotile respectively (Hodgson & Darnton 2000). For lung cancer the differences are not as great or as clear-cut. Crocidolite and amosite pose similar exposure-specific risks for lung cancer (about 5% excess per f/mL-yr), while the risk from chrysotile is estimated as 0.1-0.5% of the risk of crocidolite and amosite. Thus the risk differentials between the amphibole asbestos (crocidolite, amosite) and chrysotile for lung cancer are about 10-50:1 (Hodgson & Darnton 2000). It should be noted that the chrysotile in these risk estimates included sources where the chrysotile contained traces of tremolite, the form of which was not investigated or reported.

Only one of the cohorts with pure grunerite (amosite) asbestos exposure was examined for a quantitative exposure-response relationship (Seidman et al. 1986). There was a clear increase in the risk of lung cancer with increasing exposure expressed in fibers/mL-years.

NON-ASBESTIFORM GRUNERITE COHORTS

Several groups of workers from Homestake gold mine and the Minnesota taconite deposits have been exposed to cleavage fragments of grunerite and studied to assess possible "asbestos-related" diseases (Table 3). The non-asbestiform amphiboles present in these mines generally crystallize in a prismatic habit with well-developed cleavage so breaks occur both perpendicular and parallel to particle length.

Taconite miners: There are several studies of workers who were exposed to cummingtonite-grunerite particles from the above deposits. These include the Reserve taconite miners (Higgins et al. 1983) and the Erie-Minntac taconite miners (Cooper et al. 1988, 1992). Another group of Iron ore (hematite) miners in Minnesota is included for comparison as a negative "control" since the hematite ore does not contain amphiboles (Lawler et al. 1985).

Taconite is an iron-bearing rock that by 1978 was supplying nearly 90% of the iron ore used in the US iron and steel industry. More than 60% of this came from the Mesabi Range that is 110 miles long and 1-3 miles wide extending east to west from Babbitt, Minnesota to Grand Rapids, Michigan. Iron ore has been mined along the Mesabi Range since about 1892 (Langer et al. 1979). Taconite contains 20-50% quartz and 10-36% magnetite with smaller amounts of hematite, carbonates, greenalite, chamosite, minnesotaite, stilpnomelane and amphiboles which are non-asbestiform minerals in the cummingtonite-grunerite series, actinolite and hornblende, (Nolan et al. 1999).

Taconite from the eastern end of the Mesabi Range contains non-asbestiform cummingtonite-grunerite (most probably grunerite) and actinolite with most elongated particles having aspect ratios greater than 3:1 and length less than 10 μm and are mostly acicular cleavage fragments. Respirable dust concentrations in the Reserve mining company ranged from about 0.02 mg/m^3 to 2.75 mg/m^3 at a crusher. The modal range in most jobs was 0.2-0.6 mg/m^3, with occasional concentrations of 1-2 mg/m^3 but mostly below 1 mg/m^3. Fiber concentrations were generally < 0.5 fibers/mL. Area samples suggest no change in concentrations between 1952-1976 and exposure estimates were based on samples collected in the period 1975-8 (Higgins et al. 1983).

In the Reserve mining cohort (Higgins et al. 1983) there were no exposure-response relationships between lung cancer and cumulative exposure to silica dust or taconite (measured as mg/m^3-years) and no excess lung cancer based on the SMR. There were no cases of mesothelioma. Higgins et al. (1983) concluded that the lack of any increased risk of cancer is not surprising given the low silica and fiber exposure plus movement of miners to lower exposed jobs with increased seniority. The average and maximum latencies of lung cancer were 15 and 25 years. At high exposure levels the latency for pneumoconiosis has been as short as about 5 years or even less. As dust levels have declined latency is more in the range of 13-20 years. The cohort was also relatively young with 5% overall mortality and the number of cases was small with 15 lung cancer cases (17.9 expected), 8 with >15 years since hire (7.9 expected). Exposure-response functions were estimated using cumulative total dust exposure and cumulative silica dust exposure in mg/m^3-years as the exposure metrics. The relationship with total dust exposure, which is of interest from the standpoint of cleavage fragments, was not monotonic and the SMRs were at or below 1.0 in the three highest exposure categories. Higgins et al. (1983) concluded there was no suggestion of an association with lung cancer.

In the Eastern Mesabi district, west of the Reserve Mine are the Erie and Minntac operations. The Minntac ore has had a different metamorphic history and contains the lowest percentage of amphiboles. The Erie ore is a blend of the high and low amphibole ores with more amphiboles than Minntac but less than Reserve. Nolan et al. (1999) reported 28-40% quartz in dust from the Erie mine and 20% quartz from the Minntac mine. Concentrations of fibrous particulates were nearly always <2 fibers/mL. These particulates were >5 μm in length and included elongated cleavage fragments.

The Erie-Minntac cohort of taconite miners (Cooper et al. 1992) showed "no evidence to support any association between low-level exposure to non-asbestiform amphibole particles or quartz" and lung cancer. The Erie-Minntac cohort is older and larger than the Reserve cohort with 31% mortality and a minimum time since hire of 30 years. There were deficits in lung cancer SMRs for miners ever working in high or medium dust areas and no trend with years worked. There was no analysis by cumulative exposure.

There was one case of mesothelioma that had been reported in the initial study (Cooper et al. 1988). In this case, exposure to taconite began 11 years before death. Previous employment included work in the railroad industry as a locomotive fireman and engineer. Nolan et al. (1999) suggest it is unlikely that the mesothelioma is related to taconite because mesothelioma generally occurs after at least 25 years although latencies as short as about 18 years have been reported among insulation workers where asbestos exposure can be quite high. The more likely cause is from the railroad employment where there are opportunities for exposure to commercial amphibole asbestos from thermal lagging used on steam locomotives. Also, the time since hire in the railroad jobs is more consistent with the long latency characteristic of mesothelioma.

Although deposits of grunerite asbestos large enough for commercial exploitation are very rare, small deposits are occasionally found as a gangue mineral in a limited area of a mine that is otherwise asbestos-free. Nolan et al. (1999) described the occurrence of such a localized seam of grunerite asbestos in a small portion of an iron ore mine otherwise free of asbestos. Samples from the seam revealed three kinds of morphological types or habits. One kind was the asbestiform habit with fibers occurring as parallel fibrils and forming polyfilamentous bundles. There were two non-asbestiform habits, namely splintery fibers and massive anhedral nodules, which when crushed may form elongated cleavage fragments that morphologically resemble some asbestiform fibers. To evaluate potential asbestos exposure, 179 personal air samples were collected for all relevant jobs associated with work on this localized seam. The mean concentration of fibers ≥5 μm in length and aspect ratio ≥3:1 was 0.05 f/mL and the highest was 0.39 f/mL. All sample results were below the Mine Safety and Health Administration (MSHA) standard of 2 f/mL but 13% were above the Occupational Safety and Health Administration (OSHA) standard of 0.1 f/mL.

Nolan et al. (1999) estimated the potential lifetime risk of lung cancer and mesothelioma based on a worst case scenario. Lifetime lung cancer risks of 0.1 and 0.6 /100,000 for nonsmokers and smokers respectively were estimated using the EPA risk model and assuming a linear exposure-response relationship, age of 45 years at beginning of exposure and continuous exposure for 22 days to 0.05 asbestos fibers/mL. This was considered approximately equivalent to smoking 2 or 12 cigarettes over a lifetime.

Nolan et al. (1999) also estimated risk based on grunerite asbestos fiber content in the lungs of mesothelioma cases from a British grunerite (amosite) asbestos factory (Gibbs et al. 1994). Nolan et al. (1999) estimated it would take 75-265 years of daily 8-hour shifts to inhale the number of fibers found in the lungs of the mesothelioma cases, assuming no clearance. Fiber concentrations were about 45% higher in the lung cancer cases, suggesting about 100-380 years to reach similar fiber content in iron ore miner lungs.

Nolan et al. (1999) suggested concentrations were a minimum of 30 fibers/mL in the Paterson, NJ grunerite (amosite) asbestos factory (Seidman et al. 1986). No mesothelioma cases had less than 6 months employment and 20 years latency. Assuming breathing 0.05 fibers/mL from the gangue rock in the iron ore mine, Nolan et al. (1999) estimated it would take about 300 years to achieve the minimum exposures estimated for the mesothelioma cases in the Seidman et al. (1986) cohort.

Hematite Miners as Negative Control: Hematite from the Mesabi Range in Minnesota is a mixture of about 83% hematite (Fe_2O_3) and limonite ($HFeO_2$). The hematite deposit differs from taconite deposits in that there is the absence of all amphiboles. Some silica (about 8%) is present plus possibly low levels of radon.

Lung cancer mortality was not associated with years worked. Mesothelioma was not mentioned. Lawler et al. (1985) considered that the lack of an excess risk of respiratory disease was possibly due to strict prohibition of smoking while underground, apparent absence of significant radon daughter exposure and/or the aggressive silicosis control program. No estimates of dust exposure are available.

Gold Miners: There are several studies of miners at the Homestake gold mine in South Dakota (Gilliam et al. 1976, McDonald et al. 1978, Brown et al. 1986, Steenland & Brown 1995).

Ore containing cummingtonite-grunerite has been mined to extract gold in Lead, South Dakota, since 1876. An analysis of airborne "fibers" using electron diffraction and x-ray spectrometry was reported to show that it contained "80-90% amphiboles" with the amphiboles being "60-70% fibrous grunerite", "1-2% fibrous cummingtonite" and "10-15% fibrous hornblende" (Gilliam et al. 1976). The free silica content of the respirable airborne dust was reported to be 13.1%. Low concentrations of arsenopyrite were also reported. The NIOSH researchers identified the fibrous grunerite as grunerite (amosite) asbestos. Closer examination of the fiber population statistics suggests strongly that the

fibrous grunerite particles are non-asbestos amphibole cleavage fragments as noted in the section on fiber length.

Measurements of airborne concentrations of "fibers" in the mine in 1974 showed concentrations to be about 0.25 f/mL greater than 5 μm with the highest concentration being 2.8 f/mL based on 200 samples (Gilliam et al. 1976). The mean total fiber concentration in the mine as determined by electron microscopy was 4.82 (±0.68) f/mL with the concentration of fibers greater than 5 μm being 0.36 (±0.08) f/mL. Approximately 94% of fibers were less than 5 μm in length, the mean fiber diameter was 0.13 μm and the mean "fiber" length was 1.1 μm. The US Bureau of Mines in 1960 reported average airborne dust concentrations of 1.7 million particles per cubic foot (mppcf) (Gilliam et al. 1976). This suggests a ratio of f/mL to mppcf of about 0.25/1.7 = 0.146 f/mL per 1 mppcf.

Exposure-response relationships were developed by several of these researchers. Only the results of the latest follow-up by Steenland and Brown (1995) will be considered. However, the exposure-response developed by McDonald et al. (1978) based on semi-quantitative exposure estimates is of interest because this cohort of 1,321 men with 21 or more years of service clearly had adequate latency to observe the occurrence of mesothelioma or increase in lung cancer. There were 17 deaths from respiratory cancer but no convincing evidence of an excess of respiratory cancer or grunerite related mesothelioma. This contrasts with the results of the earlier study by Gilliam et al. 1976, which involved 440 men who had worked more than 5 years underground. They reported 10 deaths from neoplasms of the respiratory system with 2.7 deaths expected. Conclusions from the study by Gilliam et al. (1976) are weakened by the fact that the study population is small, the SMR for men with latency less than 20 years (5.4) was greater than that for men with latency greater than 20 years (3.2) (McDonald et al. 1978), and the results are contradictory to later follow-up studies of the entire cohort (Brown et al. 1986, Steenland and Brown 1995). While the reason for the high overall SMRs is not clear, selection bias is possible as the cohort was comprised of volunteers participating in a 1960 silica X-ray survey. The participation rate of workers from the mine was not reported.

The Homestake study comprises the largest and oldest cohort of workers exposed to non-asbestiform amphiboles with 47% mortality. In the Steenland and Brown (1995) study, there was a 2.6-fold excess of silicosis and a 3.5-fold excess of respiratory TB that were significantly associated with cumulative exposure and SMRs were significantly elevated in the highest exposure category for both dust-related diseases. Lung cancer was not associated with cumulative exposure in the SMR exposure-response analysis and there was a negative trend in the nested lung cancer case-control portion of this study, i.e., as exposure increased there was a trend for lung cancer risk to decrease. There were no mesothelioma deaths.

The mesothelioma and lung cancer experience of the grunerite (amosite) asbestos and non-fibrous amphibole workers will be compared separately below.

COMPARISON OF MESOTHELIOMA EXPERIENCE

One method of assessing whether non-asbestiform grunerite acts similarly to grunerite (amosite) asbestos is to compare the proportional mortality from mesothelioma in to grunerite (amosite) asbestos exposed workers and in non-asbestiform grunerite exposed workers. Mesothelioma is a cancer which can clearly be caused by amosite without known confounders such as smoking, although there are a small number of other potential causes (Pelnar 1988, Price and Ware 2004). Hodgson and Darnton (2000) argue that there is unlikely to be a threshold for asbestos-related mesothelioma, but that the exposure–response function may be non-linear. As previously discussed about 80% of mesotheliomas are asbestos related, mesothelioma is a more specific indicator of amphibole asbestos exposure and also more sensitive as there may be an excess mesothelioma risk in the absence of an excess lung cancer risk (Hodgson and Darnton 2000).

The measure of mesothelioma mortality used in this study is the percent of total mortality (labelled PMR in this context). To assume a work-related mesothelioma in the non-asbestiform grunerite cohorts there should be no previous asbestos exposure, no exposure to other potential etiological factors such as erionite or therapeutic radiation and the time of death should probably be 20 or more years since hire since exposure, or 15 or more years since hire if exposure was intense. Lanphear and Buncher (1992) estimated that for 1,105 mesothelioma cases meeting strict histological and exposure criteria, 99% had a latent period (time since first exposure) of 15 years or more and 96% of 20 years or more. The median latent period was 32 years with a range of 13 to 70 years. The probability was 0% for <10 years and 0.45% for 10-14 years.

Although there were only 19% of persons dead in the grunerite (amosite) asbestos cohorts combined, there was an overall proportional mortality from mesothelioma of 1.2%. In contrast, 23% of persons were dead in the non-asbestiform cohorts combined and no mesothelioma linked to the exposures in the non-asbestiform cohorts (or 0.03% if the non-exposure related deaths are counted). It is well recognized that the proportion of mesothelioma increases with long follow-up as mesothelioma increases as a cubed function of the time since first exposure and so would increase as the percentage of deaths increase. Certainly on present evidence there is no increased risk of mesothelioma in non-asbestiform amphibole exposed workers at the levels of exposure encountered in these industries (Tables 3 and 4, Figure 1).

In view of the fact that there was no detected increase in mesothelioma, one would not anticipate an increased risk of lung cancer due to exposure to fibrous dust, as usually in amphibole-exposed workers the exposure necessary to produce an increased risk of lung cancer is much greater that that required to increase mesothelioma risk.

COMPARISON OF LUNG CANCER EXPERIENCE

There are statistically significant excesses of respiratory cancer in all the grunerite (amosite) asbestos industries (except mining). In contrast, it is very clear that, with the exception of the first small study of Homestake gold miners (Gilliam et al. 1976), there is no increased risk of lung cancer in the non-asbestiform amphibole exposed industries. The results from the study by Gilliam have not been reproduced in subsequent studies with complete ascertainment of the cohort and longer follow-up (Steenland and Brown 1995, McDonald et al. 1978). In the taconite-exposed miners there were some statistically significant deficits of respiratory cancer. This is in spite of the fact that workers in those industries are exposed to significant crystalline silica in addition to non-asbestiform grunerite (if silica increases lung cancer risk).

Another way to examine this question is to compare the exposure-response relationships for the various studies. In Table 5 the exposure-response relationships for the studies by Seidman et al. (1986) and by Steenland and Brown (1995) are compared. While both have limitations in their exposure estimates, there is clearly no increasing trend of lung cancer with increasing exposure to non-asbestiform grunerite (and other non-asbestiform amphiboles). The exponential increase in pneumoconiosis (silicosis) with increasing exposure suggests exposure produced fibrotic but not carcinogenic effects (ratio lung cancer/ silicosis mortality = 1.25) In contrast there is a steep and statistically significant slope for the lung cancer mortality in the grunerite (amosite) asbestos insulation manufacturing plant (lung cancer/asbestosis mortality ratio = 6.8) (Figure 2).

Acheson et al. (1984) reported concentrations of 30 fibers/mL in the late 1960s in the factory using grunerite (amosite) asbestos. Exposures were probably much dustier before 1964 with improved conditions after 1964. However, Acheson et al. (1984) did not attempt to assess exposure-response trends.

It seems clear that exposure to non-asbestiform grunerite cleavage fragments and/or "fibers" at cumulative exposures below about 30 f/mL-years has not resulted in an increased lung cancer risk for workers. The risk for workers exposed to grunerite (amosite) asbestos was increased at cumulative exposures <6 f/mL-years.

OVERALL CONCLUSION CONCERNING ASBESTIFORM AND NON-ASBESTIFORM GRUNERITE

It is evident that the "fibers" to which the non-asbestiform amphibole workers were exposed were considerably shorter (and wider) than those to grunerite (amosite) asbestos workers were exposed. While both studies of grunerite (amosite) asbestos and non-asbestiform grunerite (plus other non-asbestiform amphiboles) may have limitations as far as estimates of fiber exposure are concerned, the results indicate very large differences in the mortality from mesothelioma and from lung cancer from both external and internal comparisons. It seems unlikely that errors in the exposure estimates are responsible for

these very large differences as the grunerite (amosite) asbestos factory shows a definite increase in risk of lung cancer with increasing exposure while there is no statistically valid increase in trend with non-asbestiform grunerite. The results are consistent with cleavage fragments having no (or negligible or very low) apparent carcinogenic hazard for mesothelioma and lung cancer in contrast to the obvious carcinogenic hazard shown by their asbestiform counterparts.

The Evidence from Studies of Talc and Vermiculite Exposed Workers

The Mineral Talc: The term talc is used in two ways. First, it is a term applied to a commercial or industrial product that contains finely divided mineral or rock powder that usually, but not always contains the mineral talc as its main component. Second, it can refer to the mineral talc which is a phyllosilicate mineral with the chemical formula $Mg_6 Si_8 O_{20} (OH)_4$. Since talc is a metamorphic mineral it is often associated with other minerals and is rarely found in its pure form. Co-exposures are specific to each site. Tremolitic talc is a commercial product that contains a high proportion of the amphibole tremolite in addition to the mineral talc; it also can contain other minerals including anthophyllite, a transitional talc/anthophyllite mineral as well as antigorite, lizardite and quartz. Cosmetic and pharmaceutical talcs have strictly controlled mineral contents; industrial talcs may contain other minerals.

Structurally, talc occurs in sheets that can be separated by slight pressure, so that when milled, talc can form cleavage fragments or elongated talc platelets (Wild et al.2002).

THE NEW YORK AND NORWEGIAN TALC DEPOSITS

There are at least two talc deposits containing non-asbestiform tremolite and anthophyllite which have been studied, one in New York State and one in Norway (Table 6). The best known and best characterised is the industrial talc in New York. There has been considerable discussion in the literature concerning whether the tremolite and anthophyllite present in this talc is asbestiform or non-asbestiform. However, the evidence is supportive of non-asbestiform amphiboles (Skinner et al. 1988). Norwegian talc contains tremolite and anthophyllite said to be in trace amounts. However, the mineralogy of this talc is less studied and the cohort of exposed miners/millers is much smaller.

The health experience (mesothelioma and lung cancer mortality) of these two cohorts of talc workers exposed to non-asbestiform amphiboles will be compared to 1) anthophyllite asbestos miners, 2) to workers exposed to vermiculite contaminated with tremolite asbestos; and 3) to workers exposed to talc that is not contaminated with amphiboles from Vermont, Italy, France and Austria.

New York Talc: The St Lawrence County, New York talc deposit has been extensively studied for its mineralogy and presence of fibers and cleavage fragments. The mineralogy is complex and there has been a long and ongoing debate about the amphiboles present in the Gouverneur, NY talc, which is the only mine currently operating in the region. Dement and Zumwalde (1980) concluded that bulk Gouverneur talc samples contained both amphiboles (4.5-15% anthophyllite and 37-59% tremolite) and serpentines (10-15%

lizardite and antigorite) and less than 2.6% free silica as determined by X-ray diffraction and petrographic microscope analysis. It appears that the mineral identified as anthophyllite by Dement and Zumwalde (1980), is, at least in part, a mixed phase mineral with talc evolving from the anthophyllite (Kelse and Thompson 1989). The talc also contains talc fibers. Dement and Zumwalde (1980) considered the airborne dust 'fibers' greater than 5 μm long to contain upward of 70% amphibole asbestos. Based on electron microscopy, Dement and Zumwalde reported that: "In the mine 38% of all fibers were anthophyllite. 19% were tremolite and 39% were unidentified". In the mill 45 per cent of all fibers were anthophyllite, 12 per cent were tremolite and 38 per cent were unidentified. Three percent of the fibers in the mine and 2 percent in the mill reportedly gave chrysotile electron diffraction patterns. According to Thompson (1984) and Harvey (1979) all the amphibole minerals are cleavage fragments and in the non-asbestiform habit and it has now been shown that once the talc fibers are recognized, the talc does not contain asbestiform tremolite or asbestiform anthophyllite (Kelse and Thompson 1989, Dunn Geoscience Corp 1985, Langer and Nolan 1989, Virta 1985, Crane DT 1986, Wylie 1987, Wylie 1993, Nolan et al. 1991).

A survey of the many mortality studies of workers exposed to St Lawrence County, NY talc is summarised in Appendix 1. Most of these have been variations of the original NIOSH cohort study (Brown et al. 1979, 1980). We will focus on the nested case-control study, which addressed three of the hypotheses raised about reasons for the increased lung cancer, namely smoking, other work exposures, and short-term workers (Gamble 1993). Honda et al. (2002) added 6 more years update and estimated quantitative cumulative exposure to talc dust to address the question of exposure-response (Oestenstad et al. 2002).

Gamble (1993) conducted a case-control study nested in the Brown et al. (1990) cohort of NY talc workers. There were 22 cases and 66 controls matched on date of birth and date of hire. All cases were either smokers (91%) or ex-smokers compared to 27% nonsmokers, 73% smokers or exsmokers among controls. Negative trends were consistently observed by years worked after controlling for smoking, 20 or more years latency, and exclusion of short-term workers. Lifetime work histories suggested no apparent association with non-talc exposures or non-Gouverneur talc exposures. The author concluded that "after adjustment for...smoking and the postulated role of very high exposures of short-term workers, the risk ratio for lung cancer decreases with increasing tenure." The time occurrence of lung cancer was consistent with a smoking etiology, and was not consistent with a mineral dust relationship.

Honda et al. (2002) assessed cancer and non-cancer mortality among white male Gouverneur talc workers. The cohort analyzed for cancer endpoints consisted of 809 workers employed 1947-1989 and alive in 1950. The cohort analyzed for non-cancer endpoints consisted of 782 men employed during 1960-1989. The important additions in this study were 6 more years of follow-up (through 1989) and internal exposure-response analyses with cumulative exposure to talc dust as the exposure variable. Smoking status was not taken into account. The internal comparisons by cumulative exposure (mg/m^3-yrs)

showed a significant monotonic decrease in lung cancer risk with increasing exposure. The RR was 0.5 (0.2-1.3) in the highest exposure category. Mortality from 'other NMRD' and pulmonary fibrosis showed monotonic increases in risk as exposure increased with 2-fold and 12-fold increased risks in the highest exposure categories. (Figure 3)

Honda et al. (2002) concluded that talc dust was unlikely to have a carcinogenic potency similar to asbestos for several reasons. First, there were negative exposure-response trends. Second, although lung cancer mortality was increased nearly 4-fold among miners (SMR of 3.94; 95% CI 2.33-6.22, 18 observed (obs)) it was not excessive among millers (SMR of 1.28; 95% CI 0.51-2.63; 7 obs) although exposure was similar in both groups (medians of 739 mg/m^3-years and 683 mg/m^3-years respectively). Third, the cumulative exposure was low for lung cancer cases compared to that of other workers. For example, if median cumulative exposure is set at 1.0 for lung cancer decedents, the relative median cumulative exposure is 1.1 for ischemic heart disease, 1.5 for all decedents, 3.5 for NMRD as underlying or contributory cause of death, and 10.8 for pulmonary fibrosis.

Honda et al. (2002) conclude that the lung cancer excess is unlikely to be due to talc dust *per se*. The reasons for the excess are unclear. Possible explanations for the excess include confounding by smoking or other risk factors or an unidentified constituent in the ore or mine environment that is poorly correlated with talc dust.

Norwegian Talc: Norwegian talc contains trace amounts of quartz, tremolite and anthophyllite; the main minerals are talc and magnesite. Small amounts of magnetite, chromite, chlorite, and antigorite are in the ore, while the surrounding rock contains small amounts of serpentine, mica, feldspar, calcite, and non-asbestiform amphiboles (hornblende, tremolite). Personal air samples were collected 1982-4. Exposures were somewhat higher in the mine with a range for total dust of 0.94-97.4 mg/m^3 and peaks at drilling of 319 mg/m^3. The range in the mill was 1.4-54.1 mg/m^3 with peaks in the storehouse of 109 mg/m^3. Fibers of tremolite, anthophyllite and talc with aspect ratios >3:1 by optical microscopy ranged from 0.2-0.9 f/mL (Wergeland et al. 1990).

The Norwegian male talc cohort consisted of 94 miners employed at least 1 year in talc-exposed jobs 1944-1972 and 295 millers employed at least 2-years 1935-1972 (Wergeland et al.1990). In contrast to NY talc workers, this is a generally healthy work population with a significant deficit in all-cause mortality (SMR of 0.75; 0.62-0.89), which was below expected in both mine and mill. There were only 6 incident cases of lung cancer and 6.49 expected for an SIR of 0.92. There was a small positive trend with years worked because there were zero cases in the low tenure group but no significant excess (SIR) in the 2 groups with longer tenure. There were two lung cancer cases among miners (1.27 expected) and there were more expected (5.22) than observed (4) in the mill. There was no excess of NMRD cases (3 cases of pneumonia), but numbers were too small to make any conclusions. There were no cases of mesothelioma.

It is unclear why the mortality and incidence of cancer are so far below expected. There is no excess NMRD mortality and no cases of pneumoconiosis as a cause of death despite the apparently very high dust exposures. There were 3 cases of pneumoconiosis as a contributing cause of death: 2 cases with silicosis, 1 case with talcosis. In 1981, smoking histories were obtained from 63 of 94 miners. A reduced prevalence of smoking is an unlikely cause of the reduced mortality as only 8% were nonsmokers. In view of the small size of this cohort, interpretation is difficult.

NON-ASBESTIFORM AMPHIBOLES IN SOUTH CAROLINA VERMICULITE

There are several small vermiculite pits in South Carolina containing nearly 50% tremolite/actinolite but is believed to be virtually free of fibrous tremolite (McDonald et al. 1988). Mining and the first part of the milling process are carried out wet. Four types of elongated fibers were identified in air samples using analytical transmission EM and energy dispersive X-ray spectroscopy (EDSX): tremolite-actinolite (48%), vermiculite fragments (8%), talc/anthophyllite (5%), iron rich fibers (23%) and the rest unidentified. Mean fiber size was 1.1 μm diameter and 12.7 μm long. Mean fiber length seems to be quite large for the airborne fibrous dust cloud to be totally cleavage fragments. The mean exposure was 0.75 f/mL-yrs. Nolan et al. (1991) found tremolite cleavage fragments (some of which were >10:1 aspect ratio), but found no asbestos.

The mortality study comprises a small cohort of 194 men with 6 months or more tenure before 1971 and a minimum latency of 15 years. There were 51 total deaths and an all-cause mortality of 1.17 (0.87-1.51). There were 4 deaths from lung cancer and 3 from NMRD with SMRs of 1.21 and 1.22 respectively. There were no cases of mesothelioma and no deaths from pneumoconiosis. There was a negative exposure-response trend between cumulative fiber exposure and lung cancer (Figure 4). Three of the 4 cases were in the lowest exposure category of <1 f/mL-yr (SMR = 1.71) and the 4th case was in the medium exposure category of 1-10 f/mL-yr (SMR = 0.73). Given the low fiber exposures (mean 0.75 f/mL-yrs) and the small sample size the authors concluded there was inadequate power to detect an adverse effect in this population (McDonald et al. 1988).

The health experience of workers at this mine would be of considerable interest for comparison with the miners in Montana where exposures involve asbestiform "tremolite" and other fibers. Exposure levels were so much higher in Montana and the study population is so small and exposures so low in South Carolina that comparisons are difficult. In the longer term, the population is too small for confident conclusions concerning lack of risk. On the other hand, the exposure-response trends (Figure 4) are suggestive that if tremolite asbestos were present instead of cleavage fragments there would likely have been an increase in lung cancer in the highest exposure category (and the work environment would have been more dusty with higher exposures).

Although the actual percentage of "non-asbestiform" anthophyllite in the airborne dust is not clear in these studies, we will assume that the airborne dust contains a proportion of non-asbestiform anthophyllite and non-asbestiform tremolite. In view of this, comparison of the risk of mesothelioma and lung cancer in the NY and Norwegian talc mining industry will be compared with other talc studies (negative control) and with asbestos-exposed workers in anthophyllite mining and workers exposed to vermiculite contaminated with tremolite asbestos(positive comparison). South Carolina vermiculite will be compared with Libby, Montana vermiculite.

OTHER TALC DEPOSITS

There are several mortality studies of talc where amphibole minerals are reported to be absent and the talc is relatively "pure" talc. These include studies of workers in the Vermont talc mines (Selevan et al. 1979), Italian talc mines (Coggiola et al. 2003), French and Austrian talc mines (Wild et al. 2002) (Table 6). According to Wild et al. (2002) "no asbestos contamination has ever been clearly documented in the talc deposits, at least not in the European sites."

LUNG CANCER IN NEW YORK AND VERMONT TALC MINERS AND MILLERS

In contrast to the high levels of amphibole cleavage fragments in New York's St Lawrence County talcs, geological studies conducted since the early1900's have shown no "asbestos" and little quartz in Vermont talc deposits (Boundy et al. 1979). Analyses of bulk samples collected in 1975/1976 from mines and mills of the three major Vermont talc companies showed talc and magnesite as major components (20-100%) and chlorite and/or dolomite as minor constituents (5-20%). There were trace amounts (<5%) of dolomite, calcite, quartz, biotite, ankerite, chromite, phlogopite and oligoclase and no asbestos.

Sampling surveys conducted in summer/winter of 1975/1976 at the 3 talc mines/mills resulted in respirable geometric mean concentrations in the mines ranging from 0.5-5.1 mg/m^3 (median = 0.9) and in the mills from 0.5-2.9 mg/m^3 (median = 1.0). Two methods were used to count "fibers" with aspect ratios ≥3:1 and a "maximum width and minimum length" of 5 μm. Counts using phase contrast microscopy at a magnification of 437x ranged from 0-60 fibers/mL (median = 4.1). Parallel fibers counted by SEM at a magnification of 5000x ranged from 0-0.8 fibers/mL (median = 0). Cumulative exposures were not estimated, but past exposure levels commonly exceeded the MSHA and OSHA standards of 20 mppcf (Selevan et al.1979).

The Vermont talc study provides the best comparison with the New York talc because the original studies were conducted during the same time period using similar methods and

some or the same investigators, and the mines were in adjacent US States (although different ore bodies).

The cohort comprised 392 men who had had a chest radiograph administered by the Vermont Health Department since 1937 and had been employed for more than 1 year in the Vermont talc industry between January 1, 1940 and December 31, 1969. Workers were followed through December 31, 1975. As the inclusion of workers in the cohort required a radiographic examination, it was thought that long-term workers were more likely to have participated than short-term workers. In the 1960's the Health Department reported that 70% of those missing from their radiographic surveys had less than 1-year employment. While the overall effect is not known, the original authors concluded that selection bias could not explain the observed excess mortality.

There were a total of 90 deaths with an overall SMR of 1.16. There was a six-fold excess mortality (11 obs, 1.79 exp) from NMRD (excluding influenza and pneumonia). The largest excess was among millers (7 obs, SMR=7.87), but mortality was also increased among miners (2 obs, SMR = 3.6). Radiographic evidence of pneumoconiosis (80% > category 2/1) taken as part of the annual radiographic surveillance program of active workers, suggested to the authors that Vermont talc exposure was the causal agent. There was a non-significant 1.63-fold overall excess of lung cancer, which was significant among the miners (5 obs, SMR = 4.35) but not millers (2 obs, SMR = 1.02). There were no cases of mesothelioma (Selevan et al. 1979).

The most similar cohorts are Brown et al. (1979, 1980) and Lamm et al. (1988) (Table 6). Lamm et al. (1988) considered workers with >1 year tenure, which can be compared to Vermont. Brown et al. 1979, 1980 included all workers irrespective of tenure.

Risks of lung cancer were similar in Vermont and the NY talc workers with 1 or more year employment (1.63 versus 1.93 respectively) but elevated to 2.7 when all workers are included. The SMR for lung cancer among NY talc workers with less than 1 year tenure was 3.17 (6 obs) (Lamm et al. 1988). This supports the conclusion of Lamm et al. (1988) that the risk of lung cancer in NY talc workers is concentrated in short-term workers and is most likely due to risks acquired elsewhere.

Risks of NMRD were increased 3-fold in all 3 cohorts. Risk of pneumoconiosis appeared to be higher in Vermont as non-infectious respiratory disease mortality (possible surrogate for pneumoconiosis) was increased 6-fold compared to about 4-fold for both studies of NY talc workers.

In the Vermont mills the mortality from NMRD was twice that in the mines. However, the risk of lung cancer was 4-times greater in the mine than mill. Exposures in both mine and mill in Vermont were above the then standard of 20 mppcf, but cumulative exposures were thought to be higher in the mill than the mine because mine operations were more sporadic. Selevan et al. (1979) concluded that for NMRD, "additional etiologic agent(s)

either alone or in combination with talc dust affect mine workers" because exposures were higher in the mill than in the mines yet mortality was higher in the mines. If this same reasoning is used for lung cancer, one would also conclude that other etiological agents were involved since SMRs for lung cancer were near the null among millers in both Vermont (Selevan et al. 1979) and the updated NY talc cohort (Honda et al. 2002) (Table 6).

A clear limitation of the Vermont study is the small number of deaths; there were only 6 lung cancer deaths and 11 deaths from NMRD. Nevertheless, the increased risk of lung cancer in talc miners in Vermont where there is no evidence of exposure to asbestos or amphibole cleavage fragments is consistent with a conclusion that amphibole cleavage fragments are not responsible for the increased risk of lung cancer in the New York Talc miners. On the other hand the increased risk of Non-Malignant Respiratory Disease (Pneumoconiosis) appears to be related to both Vermont and NY talc dust exposure. Further follow-up and quantitative exposure-response analysis of the NY talc cohort tested these hypotheses and found that cumulative exposure to talc dust showed a strong association with pulmonary fibrosis mortality, a moderate association with other NMRD and no association with lung cancer (Honda et al. 2002, Oestenstad et al. 2002).

It is informative to think about the history of these two cohorts of similar size and similar risks and hopefully learn some useful lessons. There has been no further follow-up of the Vermont talc cohort. The NY cohort has been re-analyzed several times both with and without further follow-up (Stille and Tabershaw 1982, Lamm et al. 1988, Brown et al. 1990, Gamble 1993, Oestenstad et al. 2002, Honda et al. 2002). From the earlier studies has come the common (and current) perception that talc in the Gouverneur Talc District contains asbestos and that "exposures to asbestiform tremolite and anthophyllite stand out as the prime suspected etiologic factors associated with the observed increase in bronchogenic cancer" (Brown et al. 1980). We offer two possible reasons for this incorrect perception.

First is the difference between including and not including short-term employees. The evidence that lung cancer risk was concentrated in short-term workers appears to have been outweighed by the known risks associated with asbestos and the presumption that NY talc workers were exposed to talc containing asbestos. The excess lung cancer among Vermont talc miners appears to have been discounted due to "talc free both of asbestiform minerals and significant quantities of free silica" and the potential for additional etiologic agents either alone or in combination with talc dust (e.g., radon).

Second, the most important limitation is with regard to the asbestos standard for regulating asbestos minerals. The OSHA-NIOSH definition of asbestos is inadequate for identifying and regulating non-asbestiform amphiboles. The crushing of rock containing non-asbestiform amphiboles (and other minerals) produces cleavage fragments that conform to the OSHA-NIOSH definition of asbestos (e.g., $\geq$3:1 aspect ratio, $\geq$5 μm length) but are not asbestos fibers.

Using this definition has produced errors regarding asbestos content of the ores that are the subject of this review, i.e., taconite tailings dumped into Lake Superior (see other presentations in this volume), asbestos exposure of Homestake gold miners (Gilliam et al. 1976) as well as talc. Other examples of the potential misuse of the federal fiber definition for asbestos include allegations of asbestos in play sand (Langer et al. 1991) and in crayons. The Agency for Toxic Substances and Disease Registry (ATSDR) in their Public Health Statement for Asbestos suggest that talc may contain asbestos. The Australian Government National Occupational and Health Commission say that industrial talc generally contains "asbestos fibers, notably tremolite." By this standard one might include all the negative control talc cohorts as positive controls of workers exposed to asbestiform amphiboles. More examples are readily available on the internet. While amphiboles are sometimes present in some talc, asbestiform amphiboles occur very rarely as a geological curiosity and not as far as we are aware using a mineralogical definition in any commercial or industrial talc.

The reasons for the increased risks of lung cancer in the New York and Vermont mining areas still remain speculative. Exposure to radon may be one reason as levels were apparently elevated in the Vermont Mines. The possibility that miners worked in areas of high asbestiform tremolite in the past cannot be totally excluded on present evidence as in one closed mine in Vermont "cobblestones of serpentine rock which were "highly tremolitic" have been reported, although workers in the Vermont cohort were considered unlikely to have had such exposure (Selevan et al. 1979). Whether this was asbestiform tremolite is not described although this appears to be inferred.

ITALIAN TALC

Italian talc is very pure and is used in the pharmaceutical and cosmetic industries. Miners and millers in this industry were studied for mortality (Rubino et al. 1976 1979, Coggiola et al. 2003). Miners were analyzed separately from millers because of silica exposure in the mine. The silica content of airborne dust in the mines was as high as 18% in drilling operations from footwall contact rocks, rock type inclusions, and carbonate, calcite and magnesite inclusions. The quartz content of the rock strata was inconsistent, ranging from 10-45%. Other minerals in the inclusions included muscovite, chlorite, garnet, and some carbonate material. A small amount of (non-asbestiform?) tremolite was detected in the inclusions but not in the talc samples. Talc samples were commonly contaminated with chlorite. From 1920-1950 there was dry drilling and no forced ventilation so exposures were over 10 times the TLV (which appears to have been about 25 mppcf at that time) in the mines and a little over the TLV in the mills. Wet drilling and forced ventilation were introduced in about 1950 and dust concentrations dropped precipitously to about 1 mppcf and well below the TLV. Concentrations in the mills were reduced slightly and slowly and after about 1960 were higher than in the mines (Rubino et al. 1976).

Coggiola et al. (2003) updated the earlier talc studies by Rubino et al. (1976, 1979). The updated cohort comprised 1,795 men with at least 1 year of employment 1946-1995 and national rates were used for comparisons. There were 880 observed deaths with an overall SMR of 1.20 (1.12-1.28). There were slight deficits in observed lung cancer and total cancer and there were no mesotheliomas.

The SMR for lung cancer was 1.07 (0.73-1.50) for miners, while there was a deficit of lung cancer with an SMR of 0.69 (0.34-1.23) in millers. There was a 2-fold excess of NMRD due mainly to silicosis with the excess occurring among miners with a significant SMR of 3.05 (2.50-3.70) compared to 1.04 (0.65-1.57) among millers. Exposure-response was examined using duration of exposure. This showed that for miners the only lung cancer excess was in the <10-year exposure group while for NMRD the exposure-response trends were flat with all categories of duration of exposure showing about a 2-fold excess mortality.

The authors concluded there was no association between lung cancer or mesothelioma and exposure to talc containing no asbestos fibers. But there was an association in miners between NMRD (primarily silicosis) and talc containing quartz.

FRENCH AND AUSTRIAN TALCS

Wild et al. (2002) conducted cohort studies of talc workers in France and Austria with nested case-control studies of lung cancer and NMRD. The French ore was a talc chlorite mixture with quartz contamination ranging from undetectable to less than 3%. In Austria, three mines were studied. At one site the ore was a talc-chlorite mixture with 0.5-4% quartz. Rock containing about 25% gneiss was not milled. A talc-dolomite mixture of 25% medium talc and <1% quartz in the final product was the product at the second mine. The ore at the third site did not contain talc but was mixture of approximately equal proportions of quartz, chlorite and mica. Workers were stratified into semi-quantitative exposure categories. The non-exposed group consisted of office workers not exposed to talc and personal dust samples averaged 0.2 mg/m^3. The low exposure group was for workers with no direct contact to talc, such as maintenance workers, and concentrations were less than 5 mg/m^3. The medium exposure category included workers exposed to concentrations between 5-30 mg/m^3 for dustier areas such as bagging or milling and onsite maintenance. Quartz exposures occurred mostly in underground mining, tunneling and barrage building and milling products at site D. The highest exposure category was reserved for past production jobs (all before 1980) where concentrations were >30 mg/m^3. Some samples produced concentrations >50 mg/m^3 and higher. Three samples taken on workers wearing personal protective equipment were 73, 82 and 159 mg/m^3. To calculate cumulative exposures, values of 2.5, 10 and 40 mg/m^3 were assigned to the low, medium and high exposure jobs.

The French cohort consisted of 1,070 men with more than one year tenure between 1945 and 1995, with vital status follow-up through 1996. The Austrian cohort consisted of 542

men with >1-year tenure between 1972 through 1995 and vital status follow-up during this same period. Three controls per each case of NMRD and lung cancer from both the French and Austrian cohorts were matched on age and calendar year of employment.

Overall mortality was below expected. There were 294 deaths in the French cohort in the period 1968-1996 for an SMR of 0.93 (0.82-1.04). The Austrian cohort was smaller with 67 deaths and an SMR of 0.75 (0.58-0.95). In the French cohort SMRs were only slightly elevated for NMRD and lung cancer (1.06 and 1.23 respectively) but were increased over five-fold (SMR 5.56 CI 1.12-16.2) for the 3 cases with pneumoconiosis. There were zero mesotheliomas.

The case-control studies combined the French and Austrian cohorts. There were 40 combined deaths from NMRD: 10 from pneumoconiosis (including silicotuberculosis), 10 from chronic obstructive pulmonary disease (COPD, restricted to chronic bronchitis and airway obstruction), and 20 deaths from pneumonia and other diseases. When analyzed by exposure categories, the exposure-response trend for NMRD was not monotonic, with no apparent increased mortality below 400 mg/m^3-yrs and 2-fold and 2.5-fold increased risks in the 2 highest exposure categories respectively. When analyzed by conditional logistic regression there was a significant exposure-response trend with an 8% increased risk per 100 mg/m^3-yrs exposure. The slope was even higher for pneumoconiosis, 1.17 for pneumoconiosis versus 1.08 for NMRD. The slope was only 1.02 for COPD. Adjustments for covariates in the regression analyses had little effect on these trends. Smoking prevalences were similar between cases and controls with about 40% nonsmokers. (Figure 5)

There were 30 combined lung cancer cases. There was a negative exposure-response trend with odds ratios of 0.6 and 0.73 in the two highest exposure categories. The trend was unchanged when adjustments were made for smoking, quartz, working underground or when lagging the exposure estimates. Also, there were no trends when analyzed by maximum dose, latency, or duration of exposure (data not shown). About 40% of the controls were nonsmokers compared to about 8% (1/19) among cases although smoking classification was unknown on about half of the cases.

Wild et al. (2002) concluded that the small excess of lung cancer was not due to talc, despite follow-up of over 50 years, high exposures and mean duration of exposure >20 years.

The pattern of mortality of workers exposed to cleavage fragments in the New York talc mines and mills (Figure 3) is very similar to that of workers in the French and Austrian mines and mills where there was no exposure to cleavage fragments (Figure 5). A limitation in these comparisons is the very large differences in cumulative exposures. If they are comparable, the dust to which the New York miners and millers are exposed is considerably more potent than that in the French and Austrian mines and mills from the

standpoint of increasing lung fibrosis/pneumoconiosis. On the other hand, this "apparently highly potent pneumoconiosis producing dust" does not increase lung cancer risk.

These studies show that "pure" talc does not increase lung cancer risk. This is consistent with the observations for the New York millers, exposed to talc as there was no excess lung cancer in talc millers.

ASBESTOS-EXPOSED COHORTS FOR COMPARISON WITH TALC WORKERS

There are two ore deposits containing tremolite asbestos or anthophyllite asbestos potentially suitable for comparison with the talc cohorts exposed to non-asbestiform tremolite and asbestos. One site is the vermiculite mine located in Libby, Montana with significant contamination from tremolite asbestos. The other is an anthophyllite asbestos mine in Finland.

LIBBY, MONTANA VERMICULITE MINE CONTAMINATED WITH ASBESTIFORM AMPHIBOLE

Ore fed to the mill in Libby, Montana contains 4-6% asbestiform amphiboles (about half tremolite asbestos and the other half a mixture of winchite and richterite in the tremolitic series, Nolan et al 1991). The health concern is the asbestiform amphibole contamination in these ores and not the vermiculite itself.

The raw ore and vermiculite concentrate from the Libby mine contain both asbestiform and non-asbestiform tremolite-actinolite and non-fibrous anthophyllite. Atkinson et al. (1982) found 21-26% fibrous tremolite-actinolite in the raw ore and 2-6% in the concentrate. Company data taken several years later indicated 3.5-6.4% at the head feed of the mill and 0.4-1% in the concentrate (Amandus et al. 1987a). After removal of coarse rock the ore contained about 20% vermiculite, 21-26% fibrous tremolite-actinolite and the rest augite, biotite, calcite, diopside, hornblende, magnetite, quartz, sphene, and apparently non-fibrous tremolite-actinolite (McDonald et al. 1986).

Eight airborne samples from the mill and screening plant examined by phase contrast light microscopy indicated the asbestiform nature of the particles: 96% had aspect ratios >10, 67% >20 and 16% >50. In addition, 73% of the fibers were longer than 10 μm, 36% >20 μm and 11% >40 μm and width was < 2.5 μm in all instances (Amandus et al. (1987a).

Two independent mortality studies of the Montana vermiculite have been conducted. McDonald et al. (1986a, b) conducted a radiological survey and a cohort and nested case-control study of 406 persons employed for at least a year prior to 1963 with follow-up until 1983. The cohort study was subsequently updated with follow-up to 1999 (McDonald et al. 2002, 2004). We will primarily focus on the up-dated analysis. Exposure was estimated from first exposure (1945) to 1982 when work histories were no longer available. By this date most of the cohort was no longer employed and fiber concentrations were about 0.1 f/mL. The plant closed in 1990. Before wet milling processes were installed, fiber concentrations were very high (estimates of >100 f/mL). A wet mill was installed in 1955 and an entirely wet process replaced both wet and dry mills in 1974 so by 1980 nearly all concentrations were <1 f/ml. Exposure-response was estimated by both categorical and

linear exposure-response (E-R) Poisson regression models and excluding those with <10 years latency. Average and cumulative exposure metrics showed similar relationships with mortality.

The overall all cause SMR was 1.27 (1.13-1.43). SMRs for lung cancer and NMRD were 2.40 (1.74-3.22) and 3.09 (2.30-4.06) respectively; the PMR for mesothelioma was 4.2%. Exposure-response trends were not linear, as risks of lung cancer, NMRD and mesothelioma increased steeply in the second quartile exposure category and showed less steep slopes in the third and fourth exposure quartiles (Figure 4, Table 8).

The other Libby cohort study was by NIOSH and published in 3 sections that included exposure estimates (Amandus et al. 1987a), cohort mortality study (Amandus et al. (1987b) and a cross-sectional radiographic study (Amandus et al. 1987c). Amandus et al. (1987b) also reported positive exposure-response trends for lung cancer with an almost 7-fold increased SMR in the high exposure category with more than 20-years latency. The PMR for mesothelioma was 2.2% considering only those with 20 years or more latency.

These results are a marked contrast to the decreasing trend of lung cancer with increasing exposure seen in the St Lawrence, NY talc workers. There is little doubt that the mesothelioma experience of the Montana work force is considerably worse than that of the talc miners. This is in spite of the fact that the New York talc workers are reported to have been exposed to dusts containing a very high percentage of non-asbestiform amphibole fibers (Kelse and Thompson, 1989).

The amphiboles in St Lawrence, NY talc are non-asbestiform while they are asbestos in the Libby deposit (Kelse and Thompson 1989, Langer and Nolan 1989, Thompson 1984, Dement and Zumwalde 1980).

Risk of pneumoconiosis, lung cancer and mesothelioma clearly increase as cumulative exposure to asbestiform tremolite increases (Figure 4). For the talc workers exposed to non-asbestiform tremolite, the risk of NMRD and pneumoconiosis increase as exposure increases, but the trends are reversed for lung cancer (inverse trend) and for mesothelioma (no cases so there is no trend) (Figure 3).

FINNISH ANTHOPHYLLITE ASBESTOS MINERS/MILLERS

Dement and Zumwalde (1980) mentioned the study of Finnish miners by Meurman et al. 1974 in the belief that both the NY talc and Finnish anthophyllite asbestos cohorts were exposed to asbestiform anthophyllite. They recommended that the risk of mesothelioma should be further studied by further follow-up of the NY talc workers. Both the NY talc (Honda et al. 2002) and anthophyllite asbestos cohorts have had further follow-up so the maximum latency in Finland is now about 40 years (Karjalainen et al. 1994; Meurman et al. 1994), which is about the same as for NY talc workers (Honda et al. 2002).

In the updated Finnish study there was a significant 2.9-fold excess incidence of lung cancer overall with a somewhat higher risk in the heavily exposed males (SIR 3.15) than in moderately exposed (SIR 2.35). There were four mesothelioma cases for a significant 46-fold increased SIR (95% CI = 12.2-115) overall (or a PMR of 0.7%, 4/593). All of the cases were in the heavy exposure group where there was a 67-fold excess (95% CI = 18.3-172) and all four had asbestosis. Asbestosis was mentioned on 20% of all death certificates (Karjalainen et al. 1994, Meurman et al. 1994).

MESOTHELIOMA COMPARISON

In the NY talc cohort, Honda et al. (2002) reported 2 deaths from mesothelioma. One was coded as benign neoplasm of the respiratory system and the other as malignant neoplasm of the lung and bronchus, unspecified. One man worked for 15 years and died 15 years after starting work at the talc facility. He had been a carpenter and millwright for 16 years, 8 years as a lead miner and 5 years as a repairman in a milk plant. The other man worked briefly at the facility as a draftsman during mill construction in 1947-8. He would have had minimal talc exposure. He had been employed on the construction of a previous talc mine, and then installed oil burning heating systems. Honda et al. (2002) concluded it is unlikely that either of these cases occurred as a result of talc exposure in the mine or mill. In essence, there are no mesothelioma cases that are plausibly related to occupational exposure to Gouverneur talc.

Vianna et al. (1981) reported a mesothelioma rate in Jefferson County twice that of New York State based on an incidence study of histologically confirmed mesothelioma cases. A total of six cases, four male and two female cases diagnosed between 1973 and 1978 were reported to have occurred in talc miners. Enterline and Henderson (1987) reported an excess mesothelioma incidence in Jefferson County from 1968 to 1981 with 4 female (0.6 expected) cases and 7 male (1.4 expected) cases for risk ratios of 6.7 and 5.0 respectively. These latter rates were the second and sixth highest in the USA and occur in the county next to the one where the talc mines are located.

Hull et al. (2002) drew attention to these elevated rates, added "five new mesothelioma cases," and concluded that New York talc exposure was associated with an increased risk of mesothelioma. This conclusion is inconsistent with the limited available data as outlined in the following:

- The entire work histories of the "talc miners" with mesothelioma are apparently not known. Exposure to asbestos in other jobs is likely given the diagnosis of asbestosis and the smaller widths of the fibers in lung tissue.

- Hull et al. (2002) attempt to interpret the results of their tissue analyses of only two mesothelioma cases. This sample is too limited to reach any reliable conclusions. Available data do not support a talc etiology.

- Fiber dimensions are consistent with asbestos exposure as the mean fiber widths in the 2 mesothelioma cases examined are less than 0.25 μm, which are the dimensions characteristic of asbestos.

- The source of the fibers in the lungs is unlikely to be NY talc mines. The average width of the fibers in the mesothelioma lungs was 0.15 μm, which is considerably less than the average width of 1.3 μm of anthophyllite and tremolite in milled talc samples (Siegrist and Wylie, 1980). Kelse and Thompson (1989) reported that 0% of the fibers in NY talc samples had widths less than 0.25 μum.

- Asbestos-related employment occurs among residents of both St Lawrence and Jefferson counties. Fitzgerald et al. (1991) reported that 39% of workers with radiographic abnormalities of parenchyma and pleura had been employed for a year or more in asbestos-related industries (e.g., shipyard, construction, pipe and furnace insulation).

- Two of the five cases had worked only four years and two years in occupations likely to be linked to the mining industry. One of these persons died at age 72 and the other at age 53. There was no information concerning their employment during the rest of their lives.

- A non-talc etiology for mesothelioma is plausible. As noted above, females in the talc mining counties have a greater risk of mesothelioma than males (Enterline and Henderson, 1987). On the other hand, the cohort data on talc workers is based on men because less than 5% of those hired in the talc industry were women (Honda et al. 2002, Brown et al. 1990, Lamm et al. 1988).

- In the cohorts, the worker populations and exposures are well defined and no association is observed between talc or non-asbestiform amphibole exposure and mesothelioma in the absence of possible asbestos exposure. The cohort studies provide a more reliable estimate of risk than a small case report with limited information on exposure.

- Hull et al. (2002) indicate the "increased pleural mesothelioma mortality [is] in Jefferson County." Jefferson County stopped producing talc about 100 years ago and all talc over the past century has been mined in St Lawrence County.

- In the Libby cohort there were twelve mesothelioma cases. The PMR was 4.2 %. Exposure to tremolite asbestos in the Libby vermiculite clearly increased the risk of mesothelioma significantly (McDonald et al. 2004). The risk of mesothelioma

among anthophyllite asbestos workers was less than the risk among crocidolite miners but almost as great as among amosite miners (Meurman et al. 1994). These comparisons show a clear excess incidence of mesothelioma for workers exposed to asbestiform tremolite and anthophyllite, but no mesothelioma attributable to exposure to non-asbestiform tremolite/actinolite or anthophyllite. These comparisons are graphically displayed in Figure 6.

LUNG CANCER COMPARISON

There was an overall 2-fold increased rate of lung cancer in the Gouverneur talc miners and millers compared to the surrounding counties in which the mine was located. This excess of lung cancer was not associated with dust exposure but was concentrated in miners with an SMR of 3.94 (CI 3.33-6.22) while millers had only a small increased risk with an SMR of 1.28 (CI 0.51-2.63). In contrast, non-malignant respiratory disease mortality was associated with dust exposure as it was increased in both miners (SMR 2.41, CI 1.16-4.44) and in millers (SMR 2.27 CI 1.13-4.07) to almost the same extent. Smoking was clearly a confounding exposure as 100% of cases were smokers or ex-smokers but only 73% among controls. When exposure-response relationships were examined, the rate ratio for the highest respirable dust exposed workers to the lowest respirable dust exposed workers was 0.5 (0.2-1.3) for lung cancer and 11.8 (3.1-44.9) for pulmonary fibrosis (Figure 3). One would expect that a respirable dust exposure index would reflect the respirable fractions of dust regardless of composition. Thus, the results indicate that the lung cancer excess in this industry is largely due to smoking and unlikely to be the result of exposure to the respirable fraction of dust (which would include talc and cleavage fragments of the various amphibole minerals). However the data suggest that the respirable dust did increase the risk of fibrosis.

In asbestos producing or using industries where midget impinger measurements were used as a basis for exposure estimates (Liddell et al. 1997), the risk of lung cancer increased with increasing levels of exposure. This illustrates the validity of exposure indices based on midget impinger measurements for assessing fiber-related risks, at least when exposures are high. However, in this talc mine, exposure estimates derived from midget impinger measurements (Oestenstad et al. 2002), showed no such relationship. If cleavage fragments were responsible for the lung cancer excess, an exposure-response relationship would have been anticipated.

To date a satisfactory explanation for the observation of an overall excess of lung cancer and for the concentration of the excess in miners rather than millers has not been found for workers exposed to either NY or Vermont talc, although at least part of the excess among NY talc workers is due to smoking (Gamble 1993, Honda et al. 2002). If the airborne dust contained over 70% amphibole asbestos fibers as reported by Dement and Zumwalde (1980), there should an overall increased risk of lung cancer, which there is, but there

should also be a logical increasing risk of lung cancer with increasing dust exposure, with a very high risk of lung cancer in highly exposed workers. This is clearly not the case.

In Finland where the incidence of cancer has been studied in anthophyllite miners, it was found that among heavily exposed male workers, the standardized incidence ratio (SIR) for lung cancer was 5.54 (CI= 3.90-7.63) and among moderately exposed workers it was 1.63 (0.20-5.89). The heavily exposed were those who worked in the mine or mill and the moderately exposed included all other personnel (Meurman et al. 1994). This exposure-response pattern is quite the opposite of that in the New York talc mines and mills.

There were consistent positive exposure-response trends for lung cancer risk as occurred with the increased asbestiform amphibole exposure in the Libby cohort. The slope of the exposure-response curve was steeper for lung cancer than for pneumoconiosis and for mesothelioma (Figure 4).

The clear exposure-response trends for lung cancer to increase with increasing exposure to asbestiform tremolite and anthophyllite is in marked contrast to the negative exposure-response trend for lung cancer risk to decrease with increasing exposure to non-asbestiform tremolite and anthophyllite present in industrial talc. The pattern of increasing risk of fibrosis is consistent with exposure to mineral dust with or without the presence of tremolite asbestos.

These lung cancer comparisons are graphically displayed in Figure 6.

BIOLOGICAL PLAUSIBILITY

Biological plausibility is not a necessary prerequisite to establishing a causal association, but it is considered "helpful" (Hill, 1965). Experimental evidence is available to consider whether or not cleavage fragments are more or less carcinogenic than asbestos fibers. These issues have been independently evaluated by Addison and McConnell and Mossman, elsewhere in this volume.

Experimental studies have the potential advantage of precisely defining the characteristics of the minerals and amount of exposure. However there are also difficulties that affect the studies and their interpretation. Hence it is important to examine the overall pattern of biological responses to asbestos fibers and cleavage fragments rather than the results of single studies. Feeding studies have been considered elsewhere (Wilson et al. this volume).

Many experiments in animals have been used to assess the potential of fibers to produce mesothelioma-type neoplasms. For example, Stanton et al. (1981) counted as a positive response, pleural sarcomas that resembled the mesenchymal mesothelioma of man. The observed response is a measure of potential hazard rather than risk. Nevertheless such

studies have been helpful in suggesting the morphological characteristics of particles in relation to "mesothelioma" producing potency. "Index particles" have been derived from these experiments. For example, based on the work of Stanton and colleagues the index particle is >8 μm long and <0.25 μm wide and is the best predictor of tumors without regard to the chemical composition of the particle. As far as we were able to ascertain, few if any cleavage fragments have the combination of diameter less than 0.25 μm and length greater than 8 μm. This would suggest that cleavage fragments are not the most potent particles for the production of mesothelioma.

Different exposure techniques have been used, but most have not involved the inhalation route of exposure applicable to humans. Most experiments have involved placing fibers onto the pleural or into the peritoneal cavity or injections intratracheally, routes of exposure which are artificial. The incidence of tumors is therefore higher and the tests are likely to be more sensitive than by inhalation. However, these experiments ignore the factors which limit fiber passage to these sites and also the alterations to the particles during their passage to these sites if they get there at all. Nevertheless, these data are useful in hazard assessment, as the absence of "mesothelioma" occurrence when fibers are placed directly on the pleura or peritoneum in sufficient numbers, is strong evidence that human inhalation exposure is unlikely to be hazardous.

Samples used in experimental studies are not always related to the minerals to which workers are exposed. For example, no experimental studies of the Homestake gold ore were found. On the other hand, there are several studies of tremolitic talc samples from the Gouverneur mine in New York State (talc samples 6 and 7 used by Stanton et al. (1981); FD-14 used by Smith et al. (1979) and FD-275 (non-asbestiform tremolite) used by Smith et al (1979) and by McConnell et al. (1983)) in feeding studies. Wylie et al. (1997) used *in-vitro* cell studies to compare the effects of asbestos fibers to talc fibers and transitional fibers in NY talc.

Figure 7 shows the results of rat injection studies of asbestiform and non-asbestiform varieties of amphiboles, primarily tremolite. These data show a consistent pattern of high incidence of mesothelioma tumors with exposure to tremolite asbestos from South Korea, California, Swansea and Italy (Davis et al. 1985, Wagner et al. 1969 1982, Stanton et al. 1981). The mesothelioma incidence of both controls and samples was around 10%. The two Scottish tremolites studied contained relatively few asbestiform fibers and there was little difference between the control and exposed rats irrespective of whether the tremolite was asbestiform or not. Davis et al. (1991) noted that the intraperitoneal injection test used in their experiments is extremely sensitive so that any dust that produces fewer than 10% tumors is unlikely to show evidence of carcinogenicity by inhalation. Thus the non-asbestiform Scottish tremolite from Shinness was considered to pose no hazard.

The Scottish tremolite from Dornie was considered to be probably harmless as well. The latter sample was described as containing mostly cleavage fragments but also some very long, thin fibers, with a possible small asbestiform subpopulation. These results should be

contrasted with those of asbestiform tremolite from Italy, California, Swansea and South Korea, which showed incidences of 70-100%. The Italian tremolite was described as a needle-like (byssolite) tremolite fiber but later shown to have an asbestiform component. For this fiber, the induction of tumors was much later than for the three asbestos types from California, Swansea and Korea. This is a normal response to a small dose of amphibole asbestos. Incidence was reduced to near zero for samples of non-asbestiform tremolite and talc fibers (Wagner et al. 1982, Stanton et al. 1981). Smith et al. (1979) assessed the incidence of tumors after injection of NY tremolitic talc and tremolite asbestos at two different doses. There were clear exposure-response trends for the asbestiform tremolite but no effect of non-asbestiform tremolite at either 10 or 25 mg exposures (Figure 8).

STATISTICAL ANALYSIS OF POTENCY BY SIZE, SHAPE AND MINERALOGY

Berman et al. (1995) conducted a statistical reanalysis of inhalation studies using data from studies of AF/HAN rats exposed to different types of asbestos to identify the exposure metrics that best predicted the incidence of lung cancer or mesothelioma. New exposure metrics were first generated from samples of the original dust because of limitations in the original characterizations. This analysis provided more detailed information on mineralogy [i.e., chrysotile, grunerite (amosite) asbestos, riebeckite (crocidolite), tremolite asbestos)], type of structure (i.e., fiber, bundle, cluster, matrix), size (length, width) and complexity (i.e., number of identifiable components). In particular, transmission electron microscopy (TEM) was added to the descriptions so that asbestos structures less than 0.2 μm could be detected and identified and use in the statistical analysis of size distributions to evaluate combined effects of length and width.

Implantation and injection studies generally indicate long, thin fibers are most likely to induce mesothelioma. However, Berman et al. (1995) considered inhalation studies more relevant for assessing human risk because lung retention and transport from the lungs are likely to be important variables in potency but are bypassed in the implantation/injection studies. Also the exposure metrics from these studies are unable to satisfactorily predict tumor incidence (for example see Oehlert 1991).

The analysis by Berman et al. (1995) indicated that particles contributing to lung tumor risk are long (>5 μm) thin (<0.4 μm) fibers or bundles with the potency increasing as length increases. For example, thin fibers longer than 40 μm are about 500 times more potent than thin fibers 5-40 μm in length. Long and very thick particles (>5 μm) may pose some risk, but these appear to be complex structures rather than fibers. It is hypothesized that these structures with large widths may break down and release additional long thin fibers or bundles. Short particles less than 5 μm in length do not appear to pose any lung cancer risk in this database. Thus in rats a particle length of 5 μm or less (or as Berman et al. suggest, 5-10 μm or less) appears to have zero potency.

The only other available data set for quantitatively assessing particle size is that of Stanton et al. (1981). The Berman et al. (1995) data set is considered more relevant because

1) It is based on an inhalation rather than implantation route of exposure;

2) It includes a range of representative samples of both asbestos fiber-types and particle sizes;

3) There is a more detailed characterization of long particles and complex structures than any other experimental study; and

4) The statistical analysis is more appropriate.

The analysis by Berman et al. (1995) is more appropriate as logarithms were not used, which avoided the problem of zero exposures in some size ranges and 0 tumors at some exposures. Also, an optimum exposure index was determined that provides a statistically adequate fit to the data. The models used by Stanton et al. (1981) do not fit the data well and therefore do not adequately describe the ranking of particle size potency.

In a statistical reanalysis of the Stanton et al. (1981) data, Oehlert (1991) confirmed the Stanton hypothesis that the primary ability of mineral particles to cause tumors are their dimensional properties, namely index particles that are long and thin (> 8 μm long and <0.25 um wide). Using improved models that fit the data better, Oehlert (1991) reinforced the idea that very long, very thin particles were the best predictors for tumors and that particles with dimensions outside the index class did not contribute to carcinogenicity. This is also in agreement with Berman et al. (1995) that non-index particles have essentially zero potency.

Oehlert (1991) disagreed with the Stanton hypothesis that dimensions alone determine carcinogenic potency. Model fit was significantly improved by assessing each mineral type separately, which indicates mineral type is also important. This disagreement was unfounded, as in fact, Stanton, himself noted that the solubility of the fiber was also important, a parameter that would be incorporated in any analysis by considering fiber type. Dimensions are necessary but are not alone sufficient to classify a substance as capable of inducing tumors. It is now well established that factors such as particle solubility and perhaps surface properties are also important. For example, fibrous talc from the Gouverneur talc deposit in New York is not equivalent (0% tumor probability) to grunerite (amosite) asbestos (93% tumor probability) in tumor producing potential although the dimensions are similar (Stanton et al. 1981).

In sum, the Oehlert (1991) reanalysis of the Stanton et al (1981) data is consistent with Berman et al (1995) that particles of certain dimensions are important predictors of tumor incidence. Long and thin particles are the significant dimensions. Also, the minerals comprising sufficient particles in these size ranges to produce tumors included asbestos

(crocidolite, amosite, and tremolite asbestos) but not the non-asbestiform amphibole mineral (tremolitic talc).

Given the importance of width and length from these experimental data, it is useful to summarize available data on dimensions of amphiboles in the epidemiological studies summarized in previous sections [Table 8].

This analysis indicates the low amounts or absence of long, thin particles in the size ranges that predict lung tumors or mesothelioma in the three ore bodies containing non-asbestiform amphiboles (NY talc, taconite and Homestake). A primary interest in studying these workers is the fact that they were exposed to non-asbestiform amphiboles. Steenland and Brown (1995) expressed the interest as follows: "Non-asbestiform amphibole fibers have not been shown to cause lung cancer, *but are suspect because of their similarity to asbestiform fibers* (emphasis added)." The data in Table 8 and noted above suggest that the similarity is applicable only to chemistry since there is no similarity in the occurrence of index particles. The long thin elongated particles (fibers) capable of inducing tumors are common in asbestiform amphiboles and absent in non-asbestiform amphiboles.

The absence of long thin particles in the size ranges identified by Stanton et al. (1981) and by Berman et al. (1995) as responsible for lung cancer and mesothelioma experimentally from ores containing non-asbestiform amphiboles detracts from the hypothesis that non-asbestiform particles have a carcinogenic potency similar to asbestos fibers. The other parameter which is now recognized as being important is biopersistence. As the cleavage fragments are in general shorter than the asbestos fibers they are likely to be more readily removed by macrophages than the asbestos. On the other hand, the solubility difference between cleavage fragments and fibers is not known, although Ilgren (2004) suggests greater solubility of cleavage fragments. However, it is possible that fibers, because they could split apart, would have greater surface areas and might be more soluble than cleavage fragments of the same dimensions. This would mean that they would have greater lung biopersistence than fibers. On this basis, long cleavage fragments would have the potential to pose a lung cancer/mesothelioma risk if cleavage fragments had the same biological potency as asbestos fibers of the same length.

In fact this is not a real problem because the biopersistence of the amphibole fibers is known to be very high. Even if there were long cleavage fragments, their large diameters would reduce the risk compared to asbestos and their retention would be highly unlikely to render them more hazardous than the asbestos fibers. In this regard, it should be noted that the sample FD14 from the NY deposit did contain elongated particles that ranged up to 50 um in length (Griegner and McCrone 1972) and did not produce mesothelioma.

Conclusions about cleavage fragments from some of the other experiments are somewhat limited because, for example, the sample of Greenland non-asbestiform tremolite studied by Wagner et al. (1982) had no fibers greater than 10 um in length and less than 0.25 um in width. The sample FD 275-1 did not contain any particles longer than 10 um in length and no particles with a width less than 1 um. Stanton (1973) showed that riebeckite

(crocidolite) asbestos, pulverized to the state where 80% of the mass of fibres was in the size range less than 10 μm in length, produced a "negligible incidence" of mesotheliomas in pleural implantation studies.

While it is reassuring that none of the samples of non-asbestiform tremolite have produced elevated rates of mesothelioma in experimental animals, it is unfortunate that systematic studies have not been done to determine whether cleavage fragments of the same lengths as asbestos fibers produce the same risks as doses have generally been measured on a mass basis and not on the basis of number of fibers or cleavage fragments of particular lengths. An obvious problem with cleavage fragment studies is that in order to achieve similar numbers of long thin fibers to the tremolite asbestos in the dose, there would have had to be a very much larger mass of cleavage fragments injected, and that alone would have produced difficulties in animal survival. There do not appear to be cleavage fragment-related increases in lung cancer or mesothelioma risk in the studies. The lack of risk may be related to the fact that workers in those industries are not exposed to high concentrations of long cleavage fragments and the fact that because of their diameters such fragments would carry a much lower carcinogenic potency than their equivalent asbestiform mineral.

Our review of the experimental literature did not reveal any findings which would indicate that cleavage fragments have the same or greater carcinogenic potential than asbestos. In fact, they indicated that amphibole cleavage fragments have a much lower carcinogenic potential than their asbestiform counterparts by many orders of magnitude. In conclusion, there are still many unanswered questions relating to the extent to which the asbestiform habit of a mineral influences its biological behavior relative to that of a cleavage fragment (size for size). But the experimental data do provide strong support for the epidemiological findings that the risks of lung cancer and mesothelioma are considerably less [or absent] for persons exposed to amphibole cleavage fragments when compared to persons exposed to amphibole asbestos fibers.

OTHER AMPHIBOLES AND OTHER MINERALS

A search of the literature for studies containing both health outcomes and descriptions of exposure to cleavage fragments failed to identify additional studies that would be of immediate assistance in examining the health risks associated with cleavage fragments. The review did identify studies such as that in Finland where the percentages of asbestiform tremolite and cleavage fragments and fibrous wollastonite and cleavage fragments of wollastonite were characterised in metamorphic limestone and dolomite mines (Junttila et al. 1996). However, epidemiological studies to relate to the environmental studies do not appear to be available. The exposure to "Federal fibers" in quarrying industries and coal mines with their large workforces would be of interest. There were experimental studies and health evaluations of arfvedsonite asbestos in Russia (Kogan et al. 1970, Pylev and Iankova 1975). There were well described studies of crocidolite-exposed populations, but no health studies of workers exposed to non-asbestiform riebeckite have been identified.

There are potentially other populations of workers exposed to the hundreds of other minerals (e.g., erionite; fluoroedenite), which can occur with a fibrous morphology. There is some information on mesothelioma risks for some of these minerals, but no studies were found of populations exposed to the non-asbestiform fibers of these same minerals.

While the gaps in knowledge concerning the US studies need to be filled, a broader base of information would be helpful. In the absence of well defined occupational groups exposed to well- characterised cleavage fragments with well studied health outcomes, it may be useful to consider non-occupational settings. In some of these areas, there are definite concentrations of pleural calcification and definite areas of elevated rates of malignant mesothelioma. Perhaps mapping the geographical distribution of mesothelioma in various countries such as Southern Europe, New Caledonia and the Mediterranean region might identify clusters of cases which might be investigated for asbestiform amphibole exposure and non-asbestiform amphibole exposure in for example, case-comparison studies.

ACKNOWLEDGEMENTS

We acknowledge with thanks the very helpful comments of Dr. Anne G. Wylie, Mr. John Addison, Dr. EE McConnell, Dr. RP Nolan and Mr. J. Kelse. This work would not have been possible without financial support from the National Stone Sand and Gravel Association, Alexandria, Virginia.

REFERENCES

Acheson ED, Gardner MJ, Winter PD, Bennett C (1984) Cancer in a factory using amosite asbestos, *Int J Epid* 13,3-10.

Addison J, McConnell EE (in press) A Review of Carcinogenicity Studies and Non-Asbestos Tremolite and Other Amphiboles. This Volume.

Addison, John (2004) Personal communication, August 6, 2004.

Allison AC (1973) Experimental methods of cell and tissue culture: effects of asbestos particles on macrophages, mesothelial cells and fibroblasts. *In*: *Biological effects of asbestos. Proceedings of a Working Conference*, Bogovski, P., Gilson, J.C., Timbrell, V., & Wagner, J.C., (Eds.), *Lyons, 2-6 October, 1972,* Lyons, International Agency for Research on Cancer, pp. 89-93.

Amandus HE, Wheeler R, Jankovic J, Tucker J (1987a) The morbidity and mortality of Vermiculite Miners and Millers Exposed to tremolite-actinolite: Part I. Exposure Estimates. *Am J Ind Med* 11, 1-14.

Amandus HE, Wheeler R (1987b) The morbidity and mortality of vermiculite miners and millers exposed to asbestiform tremolite-actinolite. Part II. Mortality. *Am J Ind Med* 11, 15-26.

Amandus HE, Althouse R, Morgan WKC, Sargent EN, Jones R (1987c) The morbidity and mortality of vermiculite miners and millers exposed to asbestiform tremolite-actinolite. Part III. Radiographic findings. *Am J Ind Med* 11, 27-37.

Atkinson GR, Rose D, Thomas K, Jones D, Chatfield EJ, Going JE (1982) "Collection, analysis and characterization of vermiculite samples for fiber content and asbestos contamination." MRI report for the Environmental Protection Agency, Project 4901-A32 under EPA contract 68-01-5915.

Berman DW, Crump KS, Chatfield EF, Davis JMG, Jones AD (1995) The Sizes, Shapes, and Mineralogy of Asbestos Structures that Induce Lung Tumors or Mesothelioma in AF Rats following Inhalation, *Risk Analysis* 15,181-195.

Bernstein DM, Mast R, Anderson R, Hesterberg TW, Musselman R, Kamstrup O and Hadley J (1994). An experimental approach to the evaluation of the biopersistence of respirable synthetic fibers and minerals. *Environ Hlth Persp* 102 (suppl5), 15-18.

Berry G (1999) Models for mesothelioma incidence following exposure to fibers in terms of timing and duration of exposure and the biopersistence of the fibers. *Inhalation Toxicology* 11, 111-130.

Boundy MG, Gold K, Martin KP Jr., Burgess WA, Dement JM (1979), Occupational Exposures to Non-Asbestiform Talc in Vermont, *Dusts and Disease*. R. Lemen and J. M. Dement (Eds), Pathotox Publisher, Inc., Park Forest South, Illinois, pages 365-378.

Brown DP, Sanderson W, Fine LJ (1990) Health Hazard Evaluation Report HETA No. 90-390-2065 and MHETA 86-012-2065, RT Vanderbilt Company, Gouveneur, NY.

Dement JM, Zumwalde RD; Gamble JF, Fellner W, DeMeo MJ; Brown DP, Wagoner JP (February 1980) Occupational exposure to talc containing asbestos, Morbidity, Mortality, and Environmental Studies of Miners and Millers I. Environmental Study, II. Cross Sectional Morbidity Study, III. Retrospective Cohort Study of Mortality, NIOSH Technical Report, DHEW (NIOSH) Publication No. 80-115.

Brown DP, Dement JM, Wagoner JK (1979) Mortality patterns among miners and millers occupationally exposed to asbestiform talc. In: *Dusts and Disease*. Lemen R, Dement JM (Eds) Pathotox Publisher, Park Forest South, Illinois, pages 317-324.

Brown DP, Kaplan SD, Zumwalde RD, Kaplowitz M, Archer VE (1986) Retrospective Cohort Mortality Study of Underground Gold Mine Workers. In: *Silica, Silicosis, and Lung Cancer*. Goldsmith D, Winn D, Shy C (Eds): New York: Praeger, pp 335-349.

Campbell WJ, Steel EB, Virta RL, Eisner MH (1979) Characterization of Cleavage Fragments and Asbestiform Amphibole Particulates, In: *Dusts and Disease*. Lemen R and Dement JM (Eds), Pathotox Publishers, Park Forest South, Illinois, pages 276-285.

Clark TC, Harrington VA, Asta J, Morgan WKC, Sargent EN (1980) Respiratory effects of exposure to dust in taconite mining and processing, *Am Rev Respir Dis* 121,959-966.

Coffin DL, Palekar LD, Cook PM (1982) Tumorigenesis by a ferroactinolite mineral, *Toxicology Letters* 13,143-149.

Coggiola M, Bosio D, Pira E, Piolatto PG, Vecchia CL, Negri E, Michelazzi M, Bacaloni A (2003) An update of a mortality study of talc miners and millers in Italy. *Am J Ind Med* 44, 63-69.

Cook PM, Glass GE, Tucker JH (1974) Asbestiform amphibole minerals: detection and measurement of high concentrations in municipal water supplies, *Science* 185,853-855.

Cooper WC, Wong O, Trent LS, Harris F (1992) An updated study of taconite miners and millers exposed to silica and non-asbestiform amphiboles. *J Occup Med* 34, 1173-1180.

Cooper WC, Wong O, Graebner R (1988) Mortality of workers in two Minnesota taconite mining and milling operations. *J Occup Med* 30,506-11.

Crane DT (1986) Memorandum from OSHA Salt Lake City Analytical Laboratory—Microscopy Branch to Greg Piacitelli, NIOSH, Morgantown, West Virginia, (November 26, 1986).

Davis JMG, Addison J, McIntosh C, Miller M, Niven K (1991) Variations in the carcinogenicity of tremolite dust samples of differing morphology, *Ann NY Acad Sciences* 643, 473-90.

Davis JMG, Addison J, Bolton RE, Donaldson K, Jones AD, Miller BG (1985) Inhalation studies on the effects of tremolite and brucite dust in Rats. *Carcinogenesis* 6, 667-674.

Dement JM, Zumwalde RD, Wallingford KM (1976), Discussion Paper: Asbestos Fiber Exposures in a Hard Rock Gold Mine, *Ann NY Acad Sciences*. 271, 345-352.

Dement JM, Zumwalde RD (1980) Occupational exposure to talc containing asbestos I. Environmental Study, DHEW (NIOSH) Publication No. 80-115, US DHEW/PHS/CDC/NIOSH, February, 1980.

Dunn Geoscience Corp (1985) An evaluation of mineral particles at Gouverneur Talc Company 1975 and 1982: A comparison of mineralogical results between NIOSHS and DGC. Contract analysis and report to the R.T. Vanderbilt Company, Inc (January 4, 1985).

Enterline PE, Henderson VL (1987) Geographic patterns for pleural mesothelioma deaths in the United States, 1968-81, *J Natl Cancer Insti* 79, 31-37.

Fitzgerald EF, Stark AC, Vianna N, Hwang S-A (1991) Exposure to Asbestiform Minerals and Radiographic Chest Abnormalities in a Talc Mining Region of Upstate New York. *Arch Env Hlth* 46,151-154.

Gamble JF (1993) A Nested Case Control Study of Lung Cancer among New York Talc Workers. *International Arch Occup Env Hlth* 64, 449-456.

Gamble JF, Greife A, Hancock J. An Epidemiologic-Industrial Hygiene Study of Talc Workers. *Ann Occup Hyg* 26:841-859 (1982).

Gamble JF, Fellner W, DeMeo MJ (1979a) An epidemiologic study of a group of talc workers. *Am Rev Resp Dis* 119, 741-753.

Gamble J, Fellner W, DiMeo MJ (1979b) Respiratory morbidity among miners and millers of asbestiform talc, In: *Dusts and Disease*. Lemen R, Dement JM, (Eds), Park Forest South, Illinois, Pathotox Publisher, pages 307-316.

Gibbs AR, Gardner MJ, Pooley FD, Griffiths DM, Blight B, Wagner JC (1994) Fiber levels and disease in workers from a factory predominantly using Amosite, *Environ Health Perspect* 102 (Suppl 5), 261-263.

Gibbs GW & Hwang CY (1980) Dimensions of Airborne Asbestos Fibers. In: *Biological effects of mineral fibres*. (Wagner, J.C., (ed.) *Proceedings of a Symposium held at Lyons, 25-27 September, 1979*, Lyons, International Agency for Research on Cancer, IARC Scientific Publications N° 30, Vol. 1, pp. 69-78.

Gilliam JD, Dement JM, Lemen RA, Wagoner JK, Archer VE, Blejer HP (1976) Mortality patterns among hard rock gold miners exposed to an asbestiform mineral. *Ann NY Acad Sci* 271, 336-344.

Griegner G, McCrone WC (1972) McCrone Associates analysis of tremolitic talc FD-14 (April 5, 1972).

Harvey AM (1979) Tremolite I Talc-a clarification in industrial minerals, Worchester Park Survey, England: Metal Bulletin Limited, p. 23-59.

Higgins IT, Glassman JH, Oh MS, Cornell RG. (1983) Mortality of Reserve Mining Company employees in relation to taconite dust exposure. *Am J Epidemiol* 118, 710-719.

Hill AB (1965) The environment and disease: association or causation? *Proc Roy Soc Med* 58,295-300.

Hodgson JT, Darnton A (2000) The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure, *Ann Occup Hyg* 44, 565-601.

Honda Y, Beall C, Delzell E, Oestenstad K, Brill I, Matthews R (2002) Mortality among workers at a talc mining and milling facility. *Ann Occup Hyg* 46 (1), 575-85.

Hull MJ, Abraham JL, Case BW (2002) Mesothelioma among workers in asbestiform fiber-bearing talc mines in New York State, *Ann Occup Hyg* 46 (1) 132-135.

Ilgren EB (2004) The biology of cleavage fragments: A brief synthesis and analysis of current knowledge, *Indoor Built Environ* 13,343-356.

Junttila S, Tossavainen A, Hartikainen T, Harma P, Korhonen K, Suominen V, Pyy L (1996) Airborne mineral fibers and quartz dust in Precambrian metamorphic limestone and dolomite mines in Finland, *Appl Occup Environ Hyg* 11, 1075-1080.

Karjalainen A, Meurman LO, Pukkala E (1994) Four cases of mesothelioma among Finnish anthophyllite miners. *Occup Environ Med* 51, 212-215

Kelse JW, Thompson CS (1989) The regulatory and mineralogical definitions of asbestos and their impact on amphibole dust analysis, *Am Ind Hyg Assoc J* 50, 613-622.

Kleinfeld M, Messite J, Langer AM (1973) A study of workers exposed to asbestiform minerals in commercial talc manufacture. *Environ Res* 6, 132-143.

Kleinfeld M, Messite J, Zaki MH (1974) Mortality Experiences among Talc Workers: A Follow-Up Study. *J Occup Med* 16, 345-349.

Kleinfeld M, Messite J, Kooyman O, Zaki M (1967) Mortality among talc miners and millers in New York State, *Arch Environ Hlth* 14, 663-7.

Kogan FM, Valagov AG, Bunimovich GI (1970) Health evaluation of the production of magnesia-arfvedsonite Gig Tr Prof Zabol. 14(10):45-7. (in Russian)

Lamm SH, Levine MS, Starr JA, Tirey SL (1988) Analysis of excess lung cancer risk in short-term employees. *Am J Epidemiol* 127, 1202-1209.

Langer AM, Maggiore CM, Nicholson WJ, Rohl AN, Rubin IB, Selikoff IJ (1979) The contamination of Lake Superior with amphibole gangue minerals, *Ann NY Acad Sci* 330, 549-572.

Langer AM, Nolan RP (1989) Mineralogical characterization of Vanderbilt Talc Specimens, Contract analysis and report to the RT Vanderbilt Company, Inc.

Langer AM, Nolan RP, Addison J (1991) Distinguishing between amphibole asbestos fibers and elongate cleavage fragments of their non-asbestos analogues In: *Mechanisms in Fibre Carcinogenesis,* Brown RC, Hoskins JA, Johnson NF (Eds), Plenum Press, NY in Cooperation with NATO Scientific Affairs Division, pages 253-267.

Langer AM, Mackler AD, Pooley FD (1974) Electron Microscopical Investigation of Asbestos Fibers. *Environ Health Perspect* 9, 63-80

Lanphear BP and Buncher CR (1992) Latent Period for Malignant Mesothelioma of Occupational Origin. *J Occup Med* 34,718-721,

Lawler AB, Mandel JS, Schuman LM, Lubin JH (1985) A Retrospective Cohort Mortality Study of Iron Ore (hematite) Miners in Minnesota, *J Occup Med* 27, 507-517.

Lee RJ (1990) Letter to National Stone Association on results of width distributions of Addison and Davis tremolite samples, April 16, 1990.

Levin JL, McLarty JW, Hurst GA, Smith AN, Frank AL (1998) Tyler asbestos workers: mortality experience in a cohort exposed to amosite, *Occup Environ Med* 55, 155-160.

Levy RS, Sigurdson E, Mandel J, Laudon E, Pearson J (1976) Investigating Possible Effects of Asbestos in City Water: Surveillance of Gastrointestinal Cancer Incidence in Duluth, Minnesota, *Am J Epidemiol* 103, 362-368.

Liddell FDK, McDonald AD, McDonald JC (1997) The 1891-1920 birth cohort of Québec chrysotile miners and millers: Development from 1904 and mortality to 1992. *Ann Occup Hyg* 41, 13-36.

Masson TJ, McKay FW, Miller RW (1974) Asbestos-like fibers in the Duluth water supply: relation to mortality. *JAMA* 228, 1019-1020.

McConnell E, Rutter HA, Ulland BM, Moore JA (1983) Chronic effects of dietary exposure to amosite asbestos and tremolite in F344 Rats. *Environ Hlth Persp* 53, 27-44.

McDonald JC, Harris J, Armstrong B (2004) Mortality in a cohort of vermiculite miners exposed to fibrous amphiboles in Libby, Montana. *Occup Environ Med* 61, 363-366.

McDonald JC, Harris J, Armstrong B (2002) Cohort mortality study of Vermiculite miners exposed to fibrous tremolite: an update. *Ann Occup Hyg* 46 (Suppl 1) 93-94.

McDonald JC, McDonald AD, Sebastien P, Moy K (1988) Health of vermiculite miners exposed to trace amounts of fibrous tremolite, *Br J Ind Med* 45, 630-634.

McDonald JC, McDonald AD, Armstrong B, Sebastien P (1986a) Cohort study of mortality of vermiculite miners exposed to tremolite, *Br J Ind Med* 43, 434-444.

McDonald JC, Sebastien P, Armstrong B (1986b) Radiological survey of past and present vermiculite miners exposed to tremolite, *Br J Ind Med* 43, 445-449.

McDonald GC, Gibbs GW, Liddell FDK, McDonald AD (1978) Mortality after long exposure to cummingtonite-grunerite, *Am Rev Resp Dis* 118, 271-277.

Meurman LO, Pukkala E, Hakama M (1994) Incidence of cancer among anthophyllite asbestos miners in Finland. *Occup Env Med* 51, 421-425.

Meurman LO, Kiviluoto R, Kakama M (1974) Mortality and morbidity among the working population of anthophyllite asbestos mines in Finland. *Br J Ind Med* 31,105-112.

Meurman LO, Kiviluoto R, Hakama M (1979) Combined effect of asbestos exposure and tobacco smoking on Finnish anthophyllite miners and millers. *Ann NY Academy Sciences* 330, 491-495.

Nolan RP, Langer AM, Oechsle GW, Addison J (1991) Association of Tremolite Habit with Biological Potential: Preliminary Report. In: *Mechanisms in Fibre Carcinogenesis,* Brown RC, Hoskins JA, Johnson NF (Eds), Plenum Press, NY in Cooperation with NATO Scientific Affairs Division, pages 231-251.

Nolan RP, Langer AM, Wilson R (1999) A risk assessment for exposure to grunerite asbestos (amosite) in an iron ore mine, *Proc Nat Acad Sci* 96, 3412-3419.

Oehlert GW (1991) A reanalysis of the Stanton et al. pleural sarcoma data, *Environ Res* 54, 194-205.

Oestenstad K, Honda Y, Delzell E, Brill I (2002) Assessment of historical exposures to talc at a mining and milling facility. *Ann Occup Hyg* 46, 587-596.

Pelnar PV (1988) Further evidence of nonasbestos-related mesothelioma. A review of the Literature. *Scand J Work Environ Hlth* 14:141-144.

Pictorial Atlas of Mineral Fibers: The Importance of using Mineral Growth Habits to Distinguish between the Potency of Asbestiform and Non-asbestiform Fibers in the Etiology of Asbestos-Related Cancer (This Volume).

Price B, Ware A (2004) Mesothelioma Trends in the United States: An Update based on SEER data for 1973 through 2003. *Am J Epidem* 159: 107-112.

Pylev LN, Iankova GD (1975) Carcinogenic activity of magnesia arfvedsonite (group of amphibole asbestos minerals) administered intrapleurally to nonbred rats. *Vopr Onkol.* 21(1), 71-6 (in Russian).

Roggli VL (1990) Human disease consequences of fiber exposures: a review of human lung pathology and fiber burden data. *Environ Hlth Persp* 88, 295-303.

Ross M (1978) The 'asbestos' minerals: definitions, description, modes of formation, physical and chemical properties, and health risk to the mining community, National Bureau of Standards Special Publication 506. Proceedings of the Workshop on Asbestos: Definitions and measurement methods held at NBS, Gaithersburg, MD, July 18-20, 1977, pp. 49-63.

Rubino GF, Scansetti G, Piolatto G, Romano CA (1976) Mortality study of talc miners and millers. *J Occup Med* 18, 187-193.

Rubino GF, Scansetti G, Piolatto G, Gay G (1979) Mortality and morbidity among talc miners and millers in Italy. In: *Dusts and Disease*, Lemen, R and Dement, JM (Eds), Park Forest South, Illinois, Pathotox Publisher, pp 357-363.

Schiller JE, Payne SL, Khalafalla SE (1980) Surface Charge Heterogeneity in Amphibole Cleavage Fragments and Asbestos Fibers. *Science* 209, 1530-1532.

Seidman H, Selikoff IJ, Hammond EC (1979) Short-Term Asbestos Work and Long-Term Observation. *Ann NY Acad Sci* 330, 61-89.

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

Selevan SG, Dement JM, Wagoner JK, Froines JR (1979) Mortality patterns among miners and millers of non-asbestiform talc: Preliminary Report. In: *Dusts and diseases*, Lemen R, Dement JM, (Eds) Park Forest South, IL: Pathotox Publisher, pp. 379-388.

Siegrist HG, Wylie AG (1980) Characterizing and Discriminating the Shape of Asbestos Particles, *Environ Res* 23,348-361.

Sigurdson EE, Levy BS, Mandel J, McHugh R, Michienzi LJ, Jagger H, Pearson J (1981) Cancer morbidity investigations: lessons from the Duluth study of possible effects of asbestos in drinking water. *Environ Res* 25, 50-61.

Skinner HC, Ross M, Fondel (1988) Asbestos and Other Fibrous Materials. Oxford University Press. 1- 204pp.

Sluis-Cremer GK, Liddell FDK, Logan WPD, Bezuidenhout BN (1992) The mortality of amphibole miners in South Africa 1946-1980. Br J Ind Med 49, 566-575.

Smith WE, Hubert DD, Sobel HJ, Marquet E (1979) Biologic tests of tremolite in hamsters. In *Dusts and diseases*, Lemen R, Dement JM, (Eds) Park Forest South, IL: Pathotox Publisher, pp. 335-339.

Stanton MF (1973) Some Etiological considerations in Fibre Carcinogenesis. In: *Biological Effects of Asbestos*. Bogovski P, Gilson JC, Timbrell V, Wagner JC (Eds) IARC Sci Publ No. 8, IARC, Lyon, France, pp. 289-294.

Stanton MF, 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 Nat Cancer Inst* 67, 965-976.

Steenland K, Brown D (1995) Mortality study of gold miners exposed to silica and nonasbestiform amphibole minerals: an update with 14 more years of follow-up. *Am J Ind Med* 27, 217-29.

Stille WT, Tabershaw IR (1982) The Mortality Experience of Upstate New York Talc Workers. *J Occup Med* 24, 480-484.

Thompson, CS (1984) Consequences of using improper definitions for regulated mineral, In: *Definitions for Asbestos and Other Health-Related Silicates* (STP-834). Philadelphia, PA: ASTM, p 182.

Timbrell V (1982) Deposition and retention of fibres in the human lung, *Ann occup Hyg* 26, 347-369.

Vianna NJ, Maslowsky J, Roberts S, Spellman G, Patton RB(1981) Malignant mesothelioma; epidemiologic patterns in New York State. *NY State J Med* 81, 735-738.

Virta RL (1985) The phase relationship of talc and amphiboles in a fibrous talc sample, U.S. Department of the Interior, Bureau of Mines Report of Investigations No. 8923.

Virta RL, Shedd KB, Wylie AG, Snyder JG (1983) Size and shape characteristics of amphibole asbestos (amosite) and amphibole cleavage fragments (actinolite, cummingtonite) collected on occupational air monitoring filters, Chapter 47, In: *Aerosols in the Mining and Industrial Work Environments*, Volume 2 Characterisation, Marple VA, Liu BYH (Eds), Ann Arbor Science Publishers, Ann Arbor, Michigan, pp. 633-643,.

Wagner JC, Chamberlain M, Brown RC, Berry G, Pooley FD, Davies R, Griffiths DM (1982) Biological effects of tremolite, *Br J Cancer* 45, 352-360.

Wagner JC, Berry G (1969) Mesotheliomas in rats following inoculation with asbestos, *Br J Cancer* 23, 567-581.

Wergeland E, Andersen A, Baerheim A (1990) Morbidity and Mortality in Talc-Exposed Workers. *Am J Ind Med* 17, 505-13.

Wild P, Leodolter K, Refregier M, Schnidt H, Zidek T, Haidinger G (2002) A cohort mortality study and nested case-control study of French and Austrian talc workers. *Occup Environ Med* 59, 98-105.

Wilson R, McConnell EE, Ross M, Axten CW, Nolan RP (in press) Risk Assessment due to Environmental Exposures to Fibrous Particulates Associated with Taconite Ore. This Volume.

Wylie AG, Virta RL, Segretti JM (1987) Characterization of mineral population by index particle: implication for the Stanton Hypothesis. *Environ Res* 43, 427-439.

Wylie AG, Skinner H., Marsh J., Snyder H., Garzione C., Hodkinson D., Winters R., and Mossman B. (1997) Mineralogical features associated with cytotoxic and proliferative effects of fibrous talc and asbestos on rodent tracheal epithelial and pleural mesothelial cells. *Toxicol Appl Pharmacology* 147, 143-150.

Wylie AG (1988) Relationship between the Growth Habit of Asbestos and the Dimensions of Asbestos Fibers. *Mining Engineering* 40, 1036-1040.

Wylie AG, Bailey KF, Kelse JW, Lee RJ (1993) The Importance of Width in Asbestos Fiber Carcinogenicity and Its Implications for Public Policy, *Am Ind Hyg Assoc J* 54, 239-252.

TABLE 1

THE DIAMETERS OF ASBESTIFORM AND NON-ASBESTIFORM AMPHIBOLES

"FIBER"	REFERENCE	PERCENT DIAMETER > 0.25 μm
Amosite	Gibbs & Hwang [1980]	28% - 42% (> 0.3μm)
All amphiboles [Homestake Gold mine]	Virta et al [1983].	100%
Taconite – Grunerite & Actinolite [East Mesabi Range]	Wylie [1988]	100%
Asbestiform Tremolite [Swansea]	Lee [1990]	76%
Non-asbestiform tremolite, [Alada Stura, Italy]	Lee [1990]	98%
Non-asbestiform tremolite [Greenland]	Wagner & Berry [1969]	100%
All amphiboles [N.Y. State]	Kelse and Thompson [1989]	100%

TABLE 2

PROPORTION OF TREMOLITE PARTICLES LONGER THAN 10 μm AND NARROWER THAN 3 μm FROM MILLED BLOCKY (PRISMATIC), ACICULAR, FIBROUS, AND TREMOLITE ASBESTOS STRATIFIED BY ASPECT RATIO USING PETROGRAPHIC MICROSCOPY§.

Aspect Ratio	% <3:1 Non-regulatory	% 3:1 to 5:1	% >5:1 to 10:1	% >10:1 to 20:1	% >20:1 to 50:1	% >50:1
Non-Asbestiform Tremolite Particles (cleavage fragments)						
Blocky	87	6.5	5	1	0.5	0
Acicular	87	4	6	3	0.5	0
Fibrous	57	18.5	18.5	5.5	0.5	0
Asbestiform Tremolite						
Asbestos1	48.5	6.5	13	13.5	13.5	5
Asbestos2	53.5	3.5	14.5	12	13	4.5

Non-regulatory designates particles that do not meet the length >5 um, width <3 um, and aspect ratio >3 criteria

§ Modified from Table 2 of Campbell et al. 1979.

TABLE 3

MESOTHELIOMA/LUNG CANCER EXPERIENCE – NON-ASBESTIFORM GRUNERITE* WORKERS AND NEGATIVE NON-AMPHIBOLE CONTROLS

STUDY POPULATION	FOLLOW-UP PERIOD	Cohort N (% dead)	N mesothelioma / N DEATHS (PMR)	Lung cancer: O/E = SMR (95% CI)	
Non-asbestiform Grunerite Cohorts (latest follow-up)					
Homestake Gold Miners (Steenland & Brown 1995)	Follow-up 1977-1990	3328 (46.6%)	0 / 1551= 0 7*#	115/101.8 = 1.13 (0.94-1.36)	
Reserve Taconite Miners (Higgins et al. 1983)	More than 1 year in period 1952-1976	5751 (5.2%)	0 / 298	15 /17.9=0.84(0.47-1.38)	
Erie Mining of taconite (Cooper et al. 1992)	> 3 months <1959, Erie-Minntac mine, 1947-1959	3431 (30.8%)	1# 0 / 1058 = 0	62/92.2 = 0.67(0.52-0.86)	
TOTAL		12510 (23.2%)	0/2907=0	192/211.9 = 0.91	
Negative Comparison: Hematite Iron Ore without amphiboles					
Hematite mining in Minnesota [Lawler et al. 1985].	> 1 year employment before 1966. Follow-up 1937-79.	Ugd 4708 (55%) Surface 5695 (36%)	0 / 2642 = 0 0 / 2057 = 0	117/117.6=1.00(0.83-1.20) 95/108 = 0.88(0.71-1.08)	

* It is recognised that these workers were also exposed to non-asbestiform hornblende and actinolite

\# Exposure began only 11 years before death making it unlikely that this mesothelioma is related to work in the taconite mine. He was previously a locomotive fireman and engineer.

*# There were seven cases [4 cancers of the peritoneum and 3 other respiratory cancers] in categories that might include mesothelioma but no mention of mesothelioma on the death certificate or other evidence to support diagnoses of mesothelioma. No mention of mesothelioma was found in a review of deaths from lung cancer or other non-specified cancer, or "categories which at time include mesothelioma" [Steenland & Brown 1996].

TABLE 4

MESOTHELIOMA /LUNG CANCER EXPERIENCE–GRUNERITE (AMOSITE) ASBESTOS EXPOSED WORKERS

STUDY POPULATION	FOLLOW-UP PERIOD	No. IN COHORT (% mortality)	No. meso / No. DEATHS = PMR	Lung Cancer: Obs/exp = SMR (95% CI)
Amosite mining (Sluis-Cremer et al. 1992)	Miners 1945-1955. Follow-up to 1986	3212 (20.2%)	4 / 648 = 0.6%	26/18.8 = 1.38 (0.97-1.91)
Amosite Insulation manufacturing (Acheson et al. 1984)	1945-78: Follow-up to 1980.	4820 (6.9%)	5/333 =1.5%	61/29.1 = 2.10 (1.62-2.71)
Amosite insulation manufacturing (Seidman et al. 1986; follow-up of Seidman et al. 1979)	1941-1945; more than 5 year latency; follow-up to 1983	820 (72%)	6/593=1.01% (death certificates) 17/593=2.9% (Best evidence)	102 / 20.51 =4.97 (4.08-6.1)
Amosite insulation manufacturing (Levin et al. 1998)	1954-1972, >10 years latency; follow-up to 1994	755 (29.4%)	6/222=2.7%	35/12.6=2.77 (1.93-3.85)
TOTAL		9607 (18.7 %)	21/1796=1.2%	224/81=2.77

TABLE 5

LUNG CANCER SMRs BY CUMULATIVE EXPOSURE EXPRESSED AS Fiber/mL-yrs FOR NON-ASBESTIFORM GRUNERITE [Steenland & Brown 1995] AND ASBESTIFROM GRUNERITE EXPOSURES [Seidman et al. 1986].

Non-Asbestiform Grunerite [Steenland & Brown 1995]								
MPPCF-yrs *	<33.3	33.3 – 133.3	133.3 – 200	>200	-	--	--	--
Fiber/mL-yrs **	< 4.8	4.8 – 19.5	19.5 - 29.2	>29.2				
SMR	1.17	1.01	0.97	1.31				
Asbestiform Grunerite [Seidman et al 1986]								
Fiber/mL-yrs **	<6	6-11.9	12-24.9	25-49.9	50-99.9	100-149.9	150-249.9	250+
SMR	14/5.31 = 2.64	12/2.89 = 4.15	15/3.39 = 4.42	12/2.78 = 4.32	17/2.38 = 7.14	9/1.49 = 6.04	12/1.32 = 9.09	11/.94 = 11.7

* Dust days in table II of the paper by Steenland and Brown 1995 (i.e.: 1 day at 1 mppcf was converted to dust years by dividing by 240 days per year [i.e. 48 weeks x 5 day week).

** MPPCF-yrs converted to f/cc-yrs using a factor of 1mppcf = 0.146 f/mL. The conversion is based on the average concentration of "fibers" greater than 5μm and particles measured by the midget impinger and reported by Gilliam et al. (1976) i.e.: 0.25f/mL divided by 1.7mppcf

TABLE 6
LUNG CANCER AND NONMALIGNANT RESPIRATORY DISEASE (NMRD) MORTALTY (SMR) AMONG TALC WORKERS

AUTHOR	YEARS	Lung Cancer SMR	Lung Cancer Mine SMR	Lung Cancer Mill SMR	NMRD Overall SMR	NMRD Mine SMR	NMRD Mill SMR	Mesothelioma
NY Brown et al(1979)	1947-59 Follow-up 1975 19% mortality	9/3.3=2.73 (1.25-5.18)			8/2.9=2.76 (1.19-5.13)			0
					Other 5/1.3 = 3.85(1.25-8.96)			
NY Lamm et al. (1988)	1947-78 >1-year tenure 14.8% mortality	6/3.1=1.93 (0.71-4.20)			7 / 2.5 = 2.78 (1.11-5.72)			
NY Honda et al.(2002)	>1 day tenure 1948-1989	31/13=2.32 (1.57-3.29)	18/46=3.94 (2.33-6.22)	7/5.5=1.28 (0.51-2.63)	28/13=2.21 (1.47-3.20)	10/4.2=2.41 (1.16-4.44)		0
NY Brown et al. (1990)	1947-1978; follow-up 1983; 23% mortality	17 / 8.2 = 2.07 (1.20-3.31)			17 / 6.8 = 2.50 (1.46-4.01)			0
	≥1 yr tenure	9/4.7 = 1.91 (0.88-3.64)			11/3.8 = 2.89 (1.45-5.18)			
Vermont Selevan et al.(1979)	1940-1975; >1-yr tenure before 1970; 23% mortality	6/3.69=1.63 (0.60-3.54)	5/1.15=4.35 (1.41-10.1)	2/1.96=1.02 (0.12-3.68)	11/3.67=3.0 (1.50-5.36)	2/1.23= 1.63 (0.20-5.87)	7/1.72=4.07	0
					Other=11/1.79= 6.15 (3.07-11)	Other= 2/0.56 = (0.43-2.89)	Other =7/0.89 = 7.87 (3.15-16.2)	
Italy Coggiola et al. (2003)	>1 yr, 1946-1995 49% mortality	44 / 46.9 = 0.94 (0.68-1.26)	33 / 30.9 = 1.07 (0.73-1.50)	11/ 16 = 0.69 (0.34-1.23)	127 / 55.7 = 2.28 (1..9-2.72)	105 / 34.4 = 3.05 (2.5-3.7)	22 / 21.3 = 1..04 (0.65-1.57)	0
France Wild et al. (2002)	1945-1995, >1- yr; 27.5% mortality	21/17=1.23 (0.76-1.89)			26/24.6= 1.06 (0.69-1.55) Pneumoconiosis 3/0.5=5.56 (1.12-16.2)			
Austria Wild et al. (2002)	1972-1996, >1-yr; 12.4% mortality	7/6.6=1.06 (0.43-2.19)			1/3.7=0.27 (0.01-1.52)			0
Norway Wergeland et al (1990)	>1-yr: miners 1944 –1972; 28.7% mortality >2-yrs millers 1935-1972; 30.5% mortality.	SIR: 6/6.49 = 0.92 (0.34-2.01)	SIR: 2 / 1.27 = 1.57 (0.19-5.69)	SIR: 4 / 5.22 = -0.77 (0.21-1.96)	Diseases of Respiratory System SMR: 3/10.9 = 0.28 (0.06-0.80)	SMR: 1/ 2.5 = 0.40 (0.01-2.23)	SMR: 2 /8.5 = 0.24 (0.03-0.85)	0

Table 7
MESOTHELIOMA/LUNG CANCER EXPERIENCE –NON-ASBESTIFORM ANTHOPHYLLITE AND ANTHOPHYLLITE ASBESTOS MINERS AND TREMOLITE ASBESTOS

STUDY POPULATION	FOLLOW-UP PERIOD	N in COHORT (% deaths)	PMR)Mesothelioma / total deaths)	Lung Cancer SMR (95% confidence intervals)
Talc workers, NY State. [Honda et al. 2002]	White men actively employed >1 day between 1948 and 1989 and alive in or after 1950. Follow-up 1950 thru 1989	809 (27%) Mill = 377 Mine = 311	2/209= 0.96%*	31/13=2.32 (1.57-3.29) Mill: 7/5.5=1.28 (0.51-2.63) Mine: 18/4.6=3.94 (2.33-6.22)
Norwegian talc workers (Wergeland et al, 1990)	Miners >1 yr 1944-1972; Millers >2 yrs 1935-1972; Follow-up 1953-1987	Total (M) 389 (30.1%) 94 miners (28.7%) 295 millers (30.5%)	0/117=0% 0/27=0% 0/90=0%	Incidence (SIR): 6/6.49=0.92(0.34-2.01) 2/1.27=1.57 4/5.22=0.77
Finnish anthophyllite. asbestos miners Karjalainen et al. (1994) Meurman et al. (1994)	> 3 mos 1953-1967; Follow-up 1953-1991	999 (59.4%) M = 736 (68.3%) F = 167 (53.9%)	4 / 593 (0.7%) M = 4/503 (0.8%) F=0/90 (0%)	Incidence: SIR M: 76/26.4 = 2.88(2.27-3.6) Heavy Exp: 3.15(2.37-4.09) Mod Exp: 2.35(1.45-3.58)
Vermiculite miners, Libby, MN. [McDonald et al 2004]	> 1-year before 1963, followed to 1999	406 70.2% mortality	12 / 285 = 4.2%	44 / 18.3 = 2.40 (1.74-3.22)
South Carolina Vermiculite McDonald et al (1988)	<6 months 1971-1986, followed to 1986	194 51/194 = 27.8% (>15 yrs latency	0 / 51 = 0%	4/3.31 = 1.21 (0.33-3.09)

* See text. Cases were not considered to have resulted from work at the talc mine. One case had latency of 15 years and one was a draftsman during construction only.

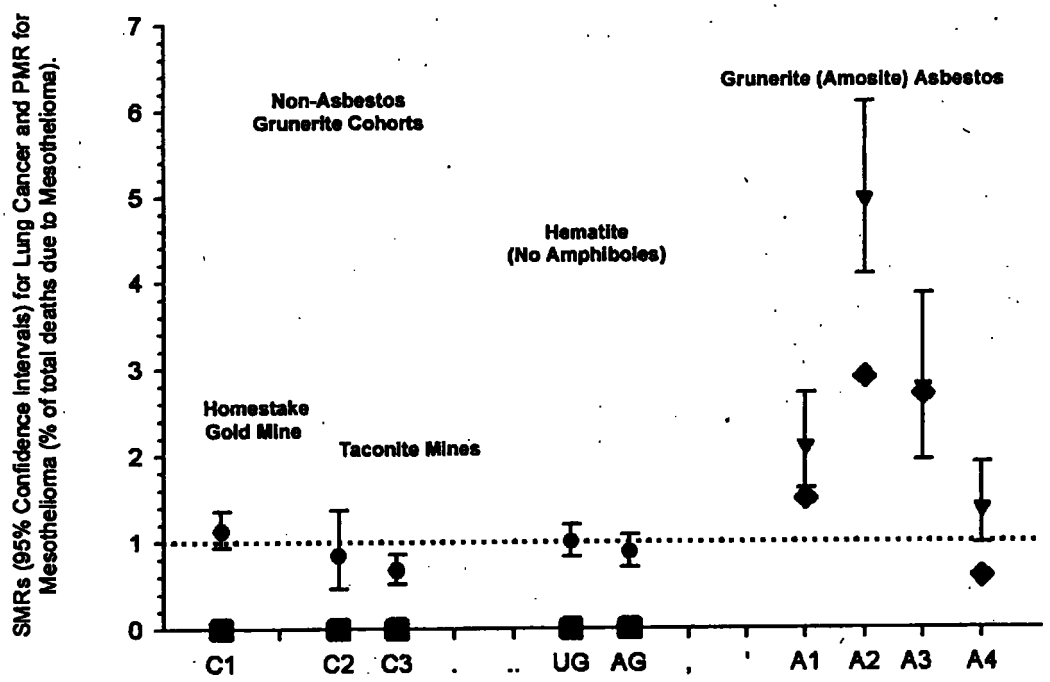
TABLE 8

Dimensions of elongated particles associated with various amphibole exposure industries studied experimentally and/or epidemiologically.

Cohort	Width (μm)	Length(μm)	Reference
Libby vermiculite; tremolite asbestos	46% <0.25	62% >5	Langer et al. (1974)
Homestake gold mine (CG = cummingtonite-grunerite) (TA = tremolite-actinolite) (GM = geometric mean)	69% CG: GM= 0.43 15% TA: GM = 0.27	34% >5 32% >5	Brown et al. (1986)
	0% <0.25 minimum 0.3 mean 1.1	Mean 4.6 Max 17.5	Virta et al. (1983)
Taconite	0% < 0.25 min 0.25 mean 1.2	Mean 5.5 Max 32.4	Wylie (1988)
Vanderbilt tremolitic talc	0% <0.25		Kelse and Thompson (1989)
Experimental Studies			
Korean tremolite asbestos >5 um L	44.7% <0.25	11.8%>5 [1.9]	Addison (2004) Davis et al. (1985)
Californian white tremolite asbestos (Davis and Addison,1981)	50%<0.25	14.9%>5 [3.2]	Addison(2004)
Swansea tremolite asbestos (Davis et al. 1991)	8.2%<0.25	33.6%>5 [1.0]	Addison(2004)
Italian tremolite (Davis et al , 1991)	13.3%<0.25	9.7% >5 [0.27]	Addison (2004)
Greenland tremolite (Wagner, 1982)	0% <0.25	100% <10	Wagner and Berry (1969, 1982).
Dornie,Scotland tremolite Davis(1991)	13.7% <0.25	22.5% >5 [0.1]	Addison (2004)
Shinness tremolite, Davis (1991)	13.8%<0.25	10.6% >5 [0]	Addison (2004)
Ferro-actinolite asbestos	Median: 0.24 Range: 0.03-5.2	Median: 1.50 Range:0.3-52.5	Coffin et al. (1982)
UICC Amosite	Median: 0.22 Range: 0.02-4.1	Median: 1.8 Range:0.15-378	Coffin et al. (1982)

Figures in [] = % >5μm and less than 0.25μm.Addison(2004) provided figures from Davis et al. (1991), calculated from the fiber numbers in the doses used in the experiments by Davis et al.

FIG. 1. Lung cancer and mesothelioma mortality in cohorts of workers exposed to Non-Asbestos Amphiboles (Homestake Gold Mine, Taconite Mines), No Amphibole as a negative control (Hematite Mine) and Grunerite (Amosite) Asbestos as a positive control (insulation factory and amphibole asbestos miners).



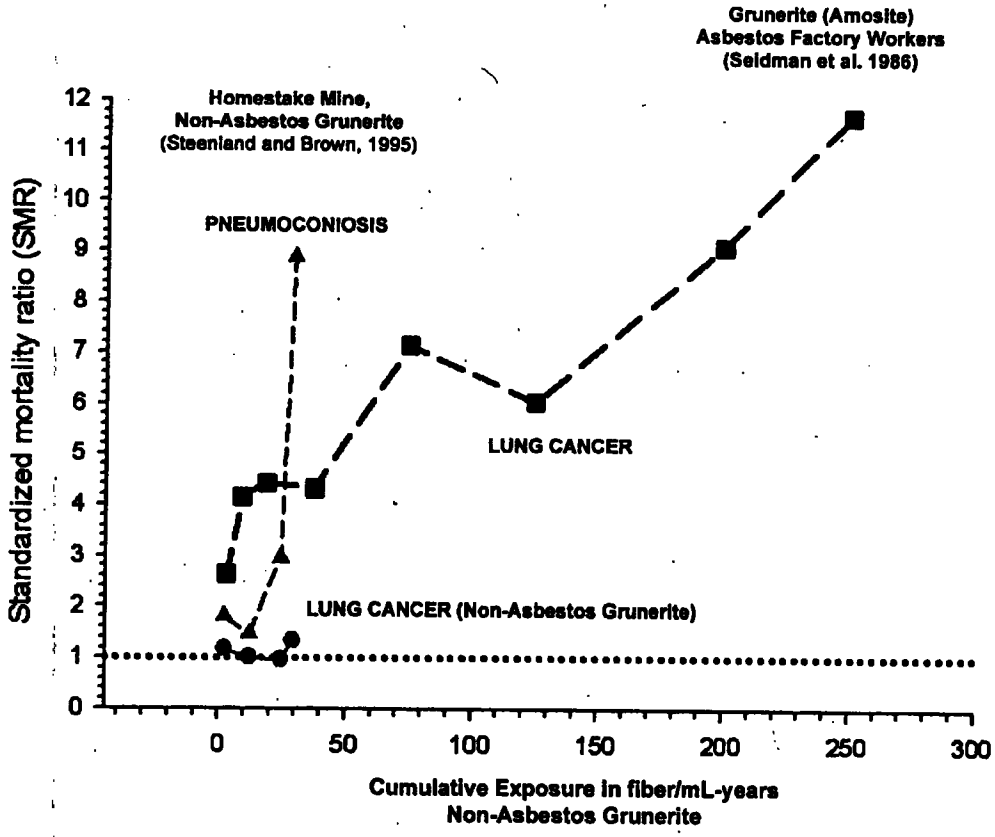
C1 = Steenland and Brown 1996
C2 = Higgins et al. 1983
C3 = Cooper et al. 1992

UG, AG = underground and aboveground Hematite (Lawler et al. 1985)

A1 = Acheson et al. 1984 grunerite (amosite) asbestos insulation factory.
A2 = Seidman et al. 1986 grunerite (amosite) asbestos insulation factory.
A3 = Levin et al. 1998 grunerite (amosite) asbestos insulation factory.
A4 = Sluis-Cremer et al. 1992 amphibole asbestos miners.

● Lung Cancer SMRs
■ % meso (n cases/total death = PMR)
····· No effect level for Lung Cancer (SMR = 1)
◆ % Mesothelioma in the Asbestos Cohorts
▼ Lung Cancer SMRs, Asbestos Cohorts

FIG. 2. Lung cancer SMRs by cumulative exposure (fiber/mL-years) and pneumoconiosis for non-asbestos grunerite (Steenland and Brown 1995) compared to grunerite (amosite) asbestos (Seidman et al. 1986).



Homestake Cohort (Steenland and Brown 1995).
Grunerite (Amosite) Asbestos, Insulation Factory (Seidman et al. 1986)
No increased risk of asbestos-related cancer when the SMR is 1
f/mL-years vs. Homestake pneumoconiosis.

FIG. 3. Exposure-response of lung cancer, other Non-Malignant Respiratory Disease (other NMRD) and lung fibrosis by Cumulative exposure (mg/m^3-years) Honda et al. (2002).

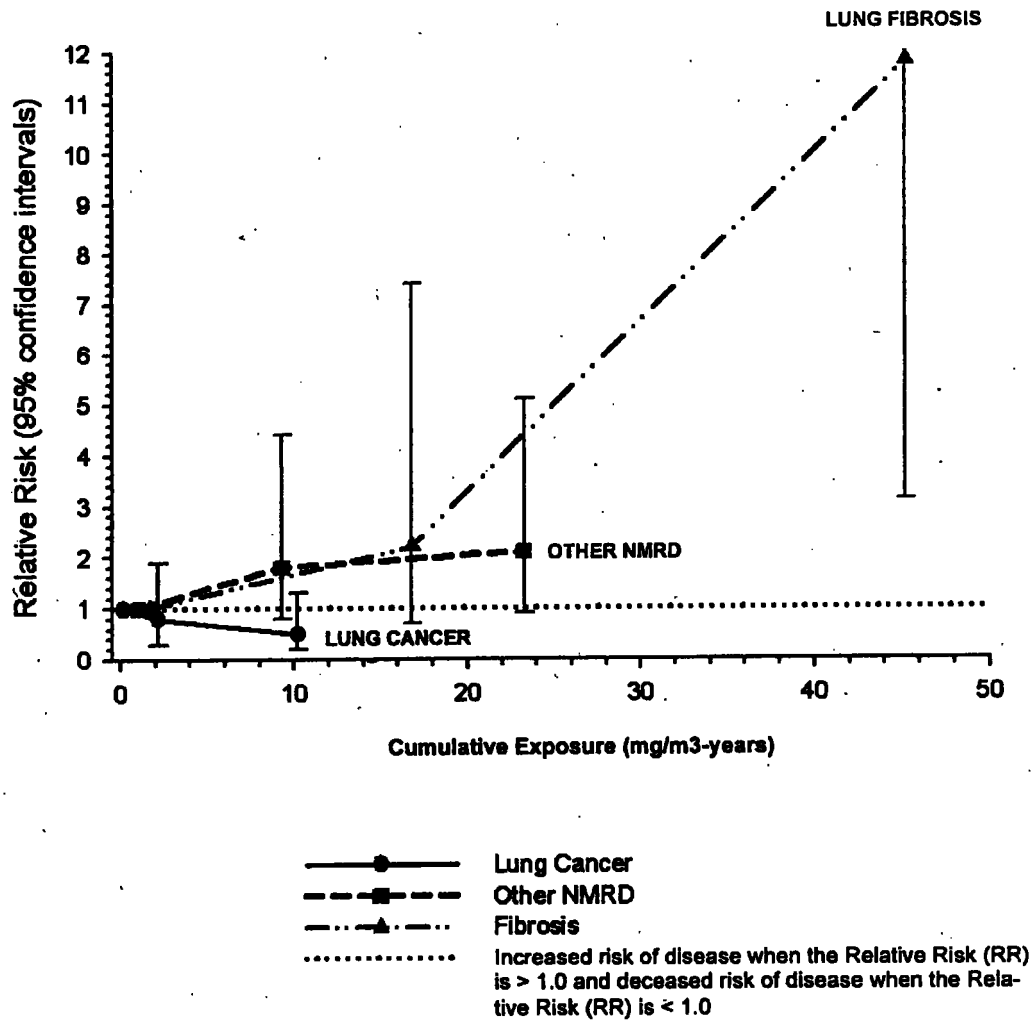
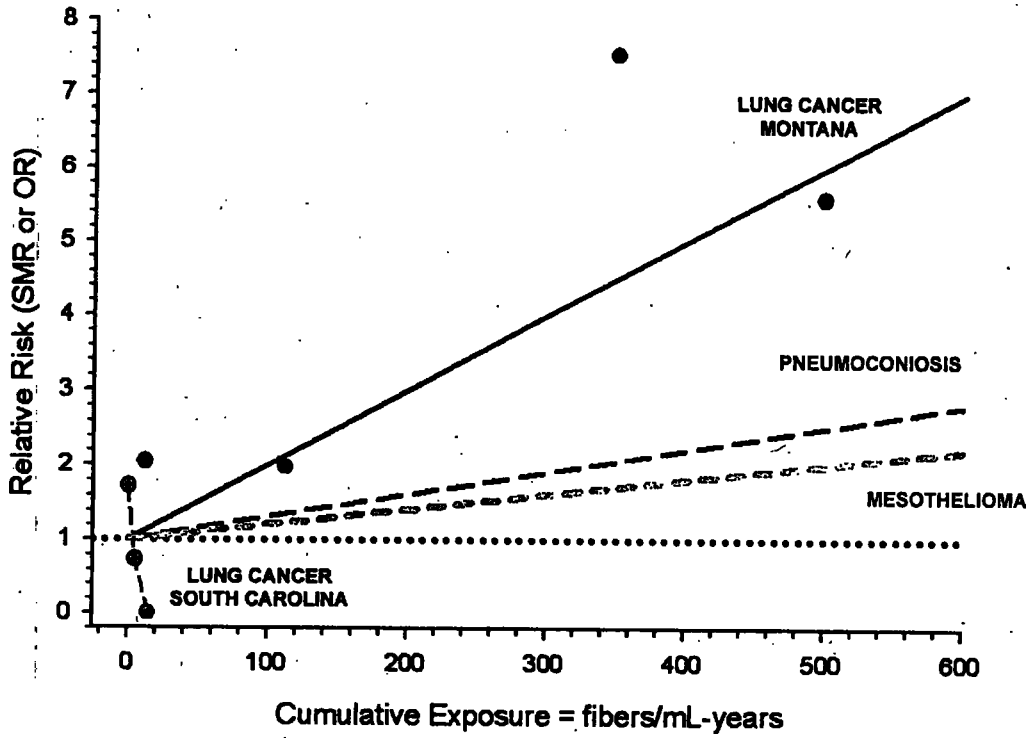


FIG. 4. Exposure-response for lung cancer, mesothelioma and pneumoconiosis among two groups of vermiculite workers one from Libby, Montana (McDonald et al. 1986) exposed to tremolite asbestos and the other from Enoree, South Carolina exposed to non-asbestos tremolite (McDonald et al. 1988). The cumulative exposure is plotted against the Relative Risk (RR) either as the Standard Mortality Ratio (SMR) or the Odds Ratio (OR).



Lung Cancer SMRs, South Carolina

Pneumoconiosis Regression, Montana

Lung Cancer Regression Analysis, Montana

Increased risk of asbestos-related disease above the line SMR or OR > 1.0

Reduced risk < 1.0

Lung Cancer SMR by exposure category in Montana (McDonald et al. 1986)

FIG. 5. Exposure-response trends for lung cancer, non-malignant respiratory disease (NMRD) and pneumoconiosis by cumulative exposure among workers from France, Austria exposed to amphibole-free talc (Wild et al. 2002).

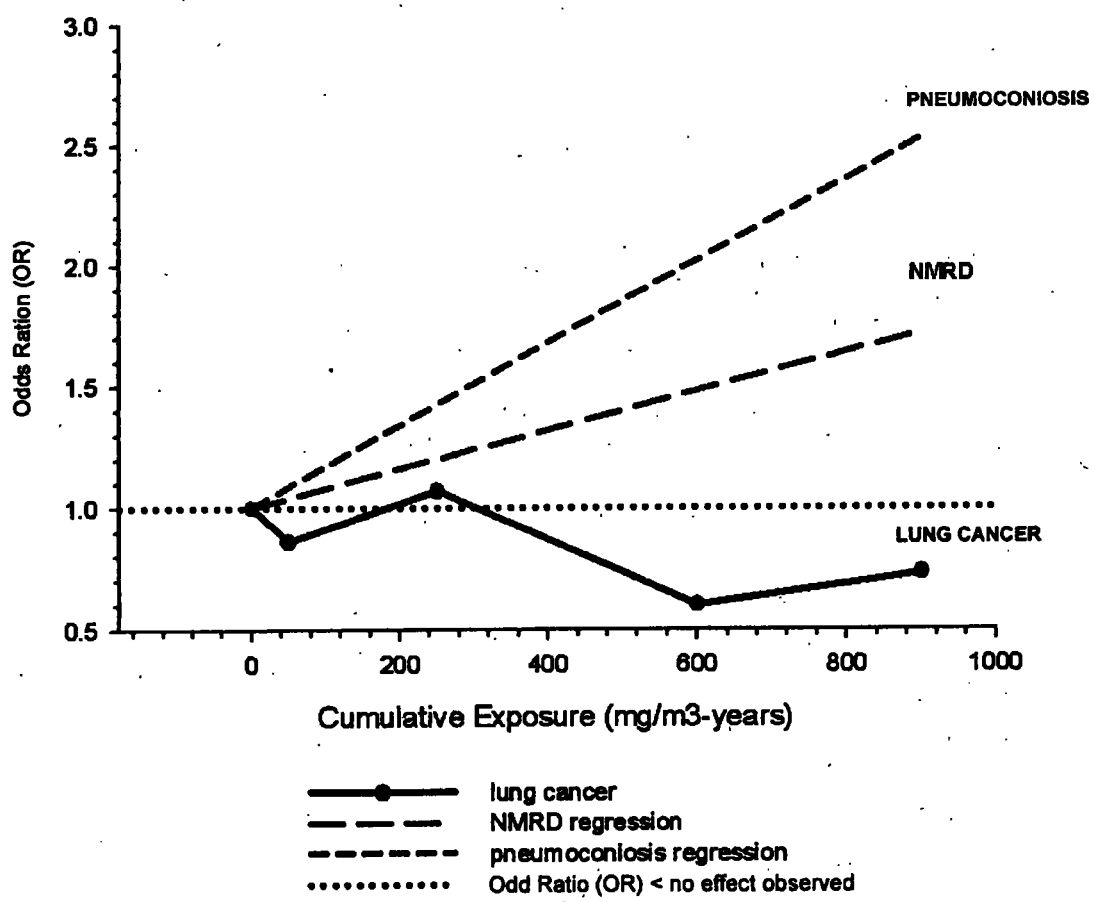


FIG. 6. Lung cancer and mesothelioma mortality in workers from New York State and Norway exposed to talc containing non-asbestos amphiboles and vermiculite from South Carolina also exposed to non-asbestos amphiboles (Honda et al. 2002, Wergeland et al. 1990, McDonald et al. 1988) and workers from Vermont, Italy, France and Austria exposed to amphibole-free talc (Selevan et al. 1979, Coggiola et al. 2003, Wild et al. 2002) compared to workers exposed to Montana vermiculite containing tremolite asbestos (McDonald et al. 1986) and Finnish anthophyllite asbestos miners (Karjalainen et al. 1994, Meurman et al. 1994).

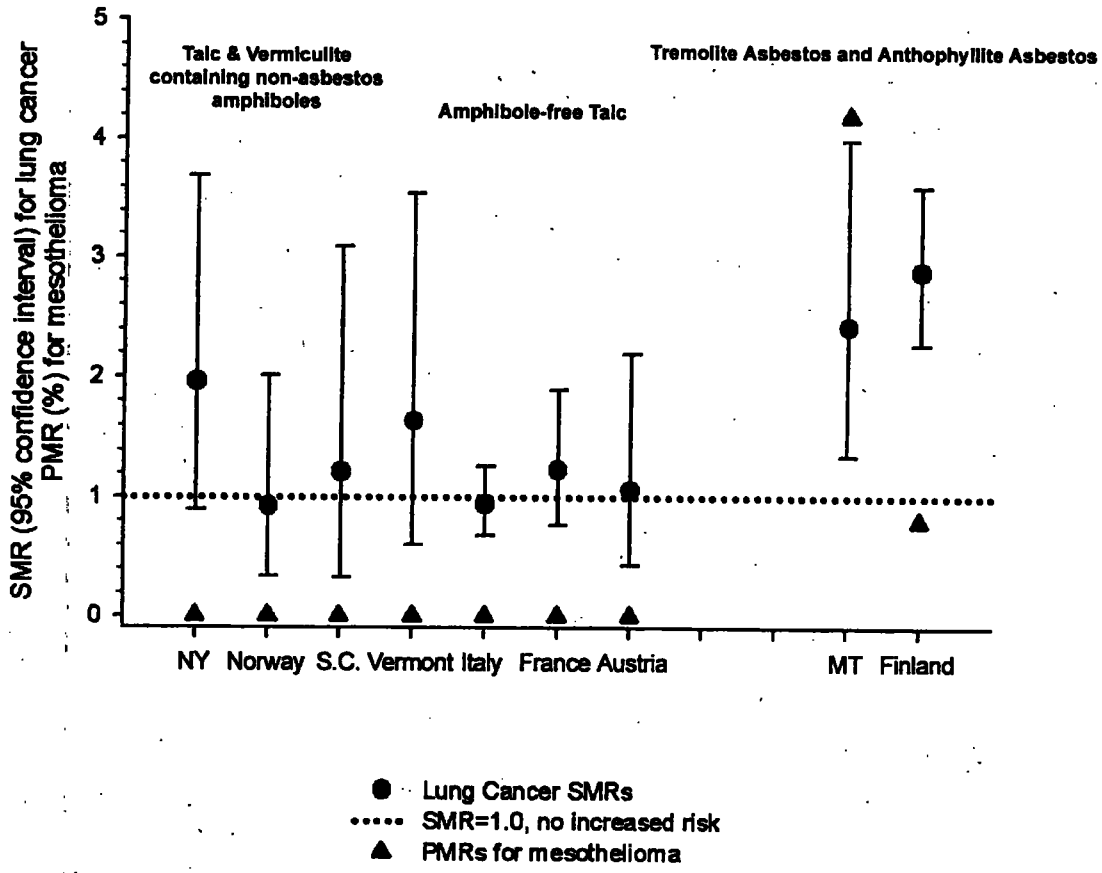
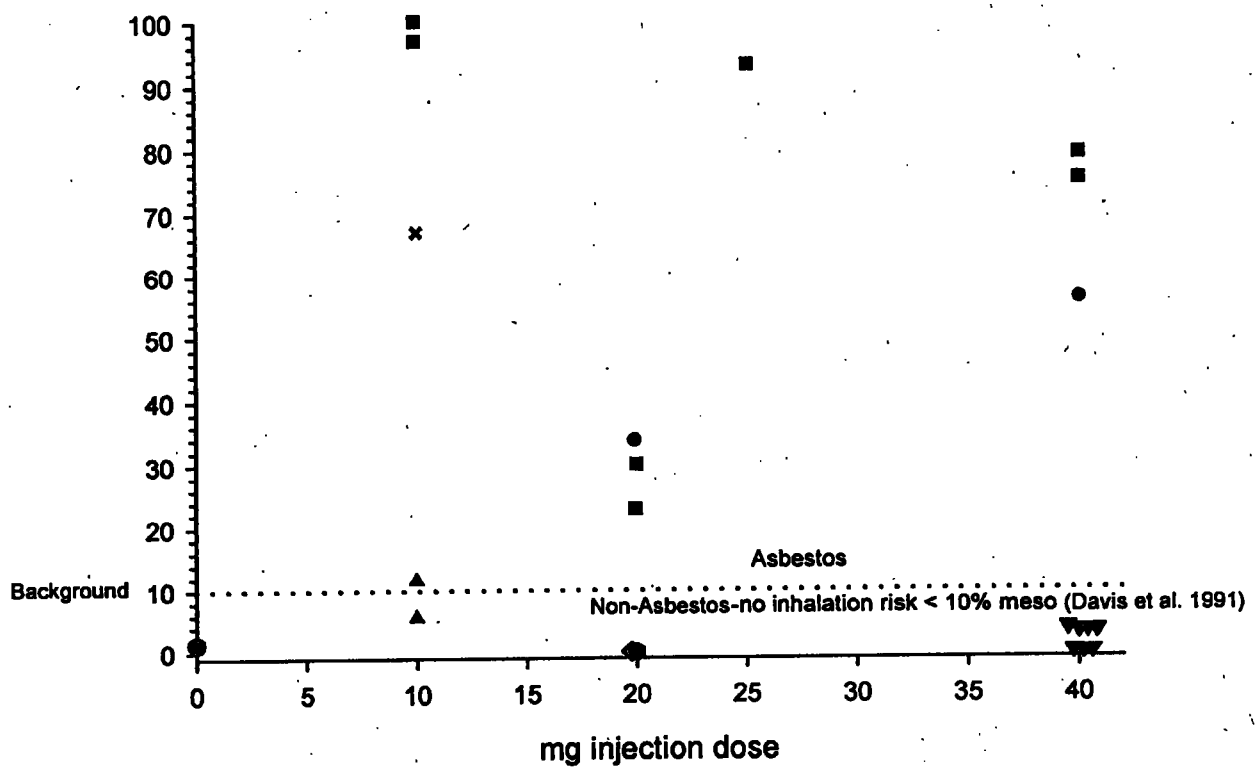


FIG. 7. Experimental animal studies comparing the carcinogenicity of amphibole asbestos and non-asbestos amphiboles in rats using injection or implantation as a route of administration.



■ **The following are all tremolite-actinolite asbestos:**

Unknown Locale, Smith et al 1979 (Trem-Act-1)
California, Stanton et al. 1981(Trem-Act-2)
Minnesota, Coffin et al 1982(Trem-Act-3)
Korea, Wagner at al. 1982 (Trem-Act-4)
Korea, Davis et al. 1985, Davis et al 1991(Trem-Act-4)
California, Davis et al. 1991(Trem-Act-6)
Swansea, Davis et al. 1991(Trem-Act-7)

● **The following are grunerite (amosite) asbestos:**

South Africa, Coffin et al 1982 (Grun-Am-1)
South Africa, Stanton et al. 1981 (Grun-Am-2)

× **The following is a mixture of non-asbestos tremolite and fine asbestiform fibers:**

Italy, Davis et al. 1991 (Trem-Non-Asb & Asbestiform)

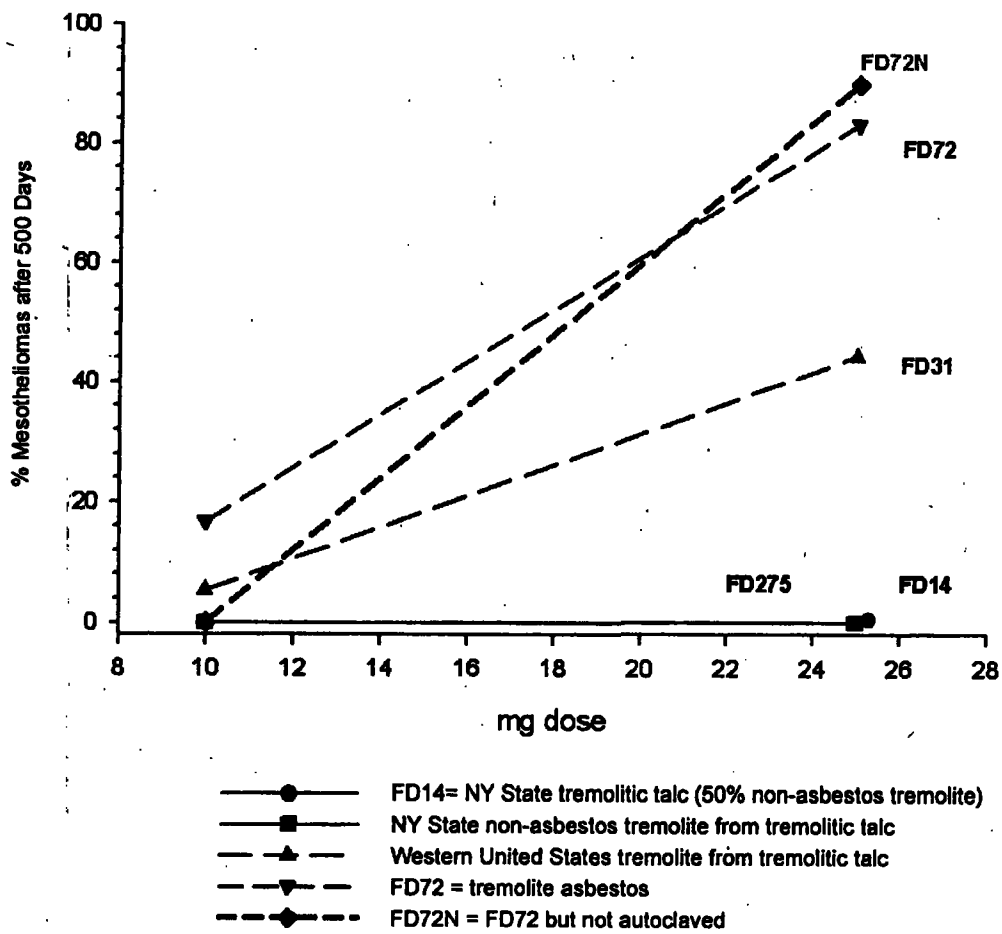
◆ **The following are tremolitic talc with talc fibers:**

Tremolitic Talc, Smith et al 1979 (Trem-Talc-1)
Tremolitic Talc, Stanton et al. 1981 (Trem-Talc-2)

▲ **The following are non-asbestos tremolite-actinolite:**

Tremolite, Smith et al 1979 (Non-Asb-Trem-Act-1)
Greenland, Wagner et al 1982 (Non-Asb-Trem-Act-2)
California, Wagner et al 1982 (Non-Asb-Trem-Act-3)
Dornie, Davis et al. 1991 (Non-Asb-Trem-Act-4)
Shinnes, Davis et al. 1991 (Non-Asb-Tem-Act-5)

FIG. 8. Differences in the percentage of mesotheliomas produced in hamsters after injection at two doses of tremolite asbestos compared to tremolitic talc from NY State with various types of non-asbestos fibers. (Smith et al. 1979)



APPENDIX

There is some overlap between this appendix and the main text in order to maintain the historical development of knowledge concerning the NY Talc deposit.

NEW YORK STATE TALC

Early NY Talc Studies: Kleinfeld et al. (1967) conducted a PMR mortality study among 220 talc miners/millers with 15 or more years of exposure in 1940, with follow-up to 1965. There were 28 deaths (31%) attributed to pneumoconiosis and complications and a PMR of 3.44 for 9 deaths from lung cancer and 1 from fibrosarcoma of the pleura. Kleinfeld et al. (1967) also reported that in a small group of asbestos insulation workers with similar years of exposure, the asbestos workers had about twice the proportion of lung cancer deaths (24% Vs 11%) and the significant excess was in both the 40-59 and 60-79 year age groups. This is "at variance" with the talc workers where the excess was only in the 60-79 year age group (PMR = 4.36) and a deficit (PMR = 0.96) in the 40-59 year age group. Overall, lung cancer mortality among the asbestos insulators was 2.5 times higher than among the talc workers, 8.43 versus 3.44

Kleinfeld et al. (1974) added 4 more years of follow-up (to 1969), 40 more workers in the cohort (for a total of 260), 17 more total deaths (for a total of 108) and 3 more respiratory cancers (for a total of 13). Similar results to the 1967 study were obtained with the only significant excess of respiratory cancers in the 60-79 age range (PMR = 4.61) and not in the 40-59 year age group (PMR = 1.63). The authors thought it was noteworthy that the significant excess respiratory cancer mortality was in the years 1945-1959 (PMR = 3.37) and not in the years 1960-69 (PMR = 1.35) when dust counts were appreciably reduced but fiber counts (fibers/mL >5 um) remained high. Ten of the 13 respiratory cancer deaths occurred in workers exposed 15-24 years (and about the same latency). The authors suggested a more susceptible group develops cancer between 15-24 years leaving a less susceptible group in spite of more years of exposure. The size of the cohort is too small to confirm this hypothesis. There was one case of peritoneal mesothelioma but no information regarding latency or other work exposures.

Exposure was characterized as predominantly talc admixed with silicates such as serpentine, tremolite, carbonates and a small amount of free silica. Exposures were quite high before 1945 when both pneumoconiosis and lung cancer cases began working. Wet drilling began after 1945, which reduced mine levels from 818 to 5 mppcf. Exposures were lower in the mill than the mine prior to 1945, but after 1945 were not reduced as much as in the mine and were now 5 times (or more) higher than in the mine. Workers with lung disease had initial exposures prior to 1945 before wet drilling began and when average dust counts in the mine were 818 (83-2800) mppcf for drilling and 120 (2-475) for mucking. In the mill, averages were 180, 69, 92 and 151 mppcf for crushing, screening, milling and bagging. After 1945 (1946-1965) average dust counts were

reduced to about 5 mppcf in these jobs in the mine and in the mill averages were generally below 50 mppcf.

Kleinfeld et al. (1973) studied 39 workers exposed to commercial talc dust where tremolite and anthophyllite were the major fibrous components. They also examined 16 talc samples from different mining and milling operations as well as finished products from NY State. Analyses included polarized LM, TEM with selected area diffraction, X-ray diffraction and electron microprobe analysis. No data are provided on distribution by fiber sizes. The point is made that there was no correlation between fiber count (fibers > 5 um) and mean dust counts (mppcf). Particles observed included "true talc, talc fibers, serpentine minerals and after fragments, and amphibole fibers and fragments." Fiber counts "may not provide a true picture of exposure to asbestiform minerals because the fiber counts include talc fibers but exclude many small asbestos fibers and 'aggregate fibers' which may contain substantial amounts of asbestiform minerals." The electron micrographs of amphibole fibers present in talc suggested amphibole cleavage fragments.

NY Tremolitic Talc

Brown et al. (1980) reported the dimensions of fibers determined by electron microscopy. Only 3% of tremolite fibers and 8-10% of anthophyllite fibers were longer than 5 μm; median lengths were about 1.5 μm. Median aspect ratios of 7.5 and 9.5 were reported for *all* fiber lengths of tremolite and anthophyllite. Data were not provided on aspect ratios for fibers > 5 um counted using phase contrast microscopy.

There then began a series of mortality studies of workers at the Gouveneur talc mine and mill in NY state (GTC) (Brown et al. 1979 1980 1990, Stille and Tabershaw 1982 Lamm et al.1988, Gamble 1993, Honda et al. 2002, Oestenstad et al. 2002). The extensive literature on GTC talc centers on three major issues that started with the first NIOSH mortality and industrial hygiene study of GTC workers.

Is the reported excess SMR for lung cancer due to the alleged asbestiform amphiboles in the talc or due to confounding? Confounding factors could include other work exposure (primarily in the surrounding mines/mills), from life-style factors such as smoking or short-term employees.

Is the tremolite and anthophyllite content of the talc non-asbestiform cleavage fragments or is the talc contaminated with tremolite asbestos and anthophyllite asbestos?

Is there biological plausibility that the tremolitic talc acts like asbestos producing asbestos-like effects in animal studies?

Epidemiology of Health Effects of GTC Talc: Brown et al. (1979, 1980) studied 398 white males first employed 1947-1959 with vital status determined as of 1975. There was a 2.73-fold excess risk of lung cancer. Risk increased with increasing latency with SMRs of 2.00 and 4.62 at 10-19 and 20-28-yrs latency, which was said to be "consistent with an

occupational etiology." There was no analysis by years worked although 4 / 9 cases had worked less than 1 year. Smoking was considered unlikely to account for all the increased risk by Brown et al. (1979, 1980). Exposures in surrounding mines and mills were higher but all were said to involve exposures to "asbestiform amphiboles." Exposures to "asbestiform tremolite and anthophyllite stand out as the prime etiologic factors associated with the observed increase in bronchogenic cancer."

Stille and Tabershaw (1982) studied 655 white males employed 1948-1977 with vital status determined at the end of 1978. Lung cancer was only significantly elevated among employees with any prior employment history . There was no analysis by years worked and latency was not taken into account.

Because of these conflicting findings, Lamm et al. (1988) reanalyzed these data. They studied 725 male talc workers who had ever worked at Vanderbilt since the plant opened in 1947 through the end of 1977 with follow-up through 1978. Previous employment obtained from job applications were classified as posing a prior risk, no prior risk or unclassifiable (no indication of prior work history) with regard to risk of lung cancer. Among those with more than 1-year employment the SMRs for lung cancer and non-infectious, non-neoplastic respiratory diseases were 1.93 and 3.70 respectively, compared to 3.00 and 0 for those with less than 1-year duration. Adding prior exposure history to the analysis showed that lung cancer risk appeared to be related to prior employment. The SMRs were similar for all job risk categories, although the number of cases was too small to be definitive. Mean latency was 20.8 years (12-25) and all those with less than 20 years latency since being hired at GTC had worked elsewhere. Five of the 12 cases had 3 months or less employment. The authors conclude the increased risk of lung cancer in this cohort of talc workers is concentrated in short-term workers, probably due to prior employment, smoking or other differences in behavioral characteristics.

At the request of RT Vanderbilt and Company, NIOSH conducted a health hazard evaluation (HHE) of the GTC cohort (Brown et al. 1990). Eight years of follow-up (through 1983) and an analysis by latency and tenure were added to the retrospective cohort study. Nearly a third (27%) of the cohort had died, with 161 total deaths and 17 lung cancer deaths with an overall SMR of 2.07. About 50% of the cohort had worked less than 1 year. Among the 13 lung cancer cases with 20 or more years latency, there was a 3.6-fold excess in the 8 cases with less than a year tenure Vs. a nonsignificant SMR of 1.79 among the 5 cases with >1-year tenure. There were also 17 NMRD deaths with an overall SMR of 2.50 (1.46-4.01). Six of the cases had worked for less than 1 year with an SMR of 1.94 (0.72-4.28). There was a 3-fold excess (SMR 2.89; 1.45-5.18) among those with more than 1-year tenure. This pattern for NMRD is "more consistently associated with an occupational exposure at GTC." Principal limitations in this study were small size (especially those with long tenure), inability to precisely characterize past occupational exposures at GTC or elsewhere, and lack of reliable smoking history. The authors concluded it is unlikely these potential confounders alone could account for the observed excess risks.

Gamble (1993) conducted a case control nested in the Brown et al. (1990) cohort. Information was collected on smoking, time exposed to talc plus a risk ranking on non-talc exposure. There were 22 cases and 66 controls matched on date of birth and date of hire. There were zero nonsmokers among the cases (91% smokers, 9% ex-smokers)

compared to 27% nonsmokers, 73% smokers or ex-smokers among controls. Inverse trends were consistently observed by years worked for different subsets of the study population; e.g., all cases and controls, smokers only, those with >20-years latency, total tremolitic talc years. The author concluded that "after adjustment for...smoking and the postulated role of very high exposures of short-term workers, the risk ratio for lung cancer decreases with increasing tenure." The time occurrence of lung cancer was consistent with a smoking etiology, and was not consistent with an occupational relationship.

Finally, Honda et al. (2002) assessed cancer and non-cancer mortality among white male GTC talc workers. The cohort analyzed for cancer mortality consisted of 809 workers employed 1947-1989 and alive in 1950. The cohort analyzed for non-cancer mortality consisted of 782 men employed during 1960-1989. The important additions in this study were 6 more years of follow-up (through 1989) and internal exposure-response analyses with cumulative exposure to talc dust as the exposure variable. Overall mortality continued to remain elevated at 1.31 ((209/160) due largely to 2.32-fold excess from lung cancer (31/13) and 2.21-fold excess in NMRD (28/13). The patterns are consistent with previous results, in particular with the inverse lung cancer trends from the nested case-control study (Gamble, 1993) and the inverse relationships for NMRD and lung cancer reported by Lamm et al. (1988). Honda et al. (2002) reported that among workers with >20-years latency, there was a 3.3-fold excess lung cancer for <5-years tenure and 1. 9-fold excess for >-5 years tenure. For other NMRD (COPD + pneumoconiosis and excluding pneumonia, influenza, asthma, emphysema and bronchitis) the SMRs were 2.71 and 3.02 respectively. The internal comparisons by cumulative exposure (mg/m^3-yrs) and adjusted for age and latency, showed a significant monotonic decrease in lung cancer risk with increasing exposure with a RR of 0.5 (0.2-1.3) in the highest exposure category. Mortality from 'other NMRD' and pulmonary fibrosis showed monotonic increases in risk as exposure increase. Risks were increased 2-fold and 12-fold increased risks in the highest exposure categories (Figure 3).

There were 2 cases of mesothelioma, but because of too short latency in one case and minimal exposure for a short time, Honda et al. (2002) considered it unlikely that exposure to talc ore was the cause.

Because of too short latency, Honda et al. (2002) concluded that the cause of the increased lung cancer mortality in the cohort is unclear, but speculated that it could be due in part to smoking or "other unidentified risk factors." They suggest it is unlikely to be related to talc ore dust per se. Other NMRD (and in particular fibrosis) were considered causally related to talc ore dust, other dusts in other work environments and smoking. This conclusion is supported by the differences in years worked and median cumulative exposures among decedents with these three causes of death and the inverse E-R trend for lung cancer (Table A1).

TABLE A1

Exposure differences between cases of lung cancer, Other NMRD and Fibrosis in NY talc workers (Honda, 2002)

	Lung Cancer	Other NMRD	Fibrosis
Median Yrs worked	1.0	8.3	11.8
Median Cumulative Exposure (mg/m^3-days)	347	1199	3759

These results are not at all consistent with the dust causing fibrosis being responsible for the lung cancer excess.

Summary of Results from studies of NY Talc Workers

The cohorts studied before 1979 by Kleinfeld and colleagues worked in talc mines in St Lawrence County, NY. After 1978 the cohorts were comprised of workers at the Gouverneur mine and mill, some of whom had previous employment in other mines in St Lawrence County, NY (Table A2).

The authors of the two NIOSH studies of GTC talc (Brown et al. 1979, 1980, 1990) concluded that the tremolite and anthophyllite were the most likely etiological agents. This conclusion is based on the following logic.

The excess risk of lung cancer and NMRD were consistent with the findings of Kleinfeld et al. (1967, 1973) among NY talc workers and Meurmann et al (1974, 1979) among anthophyllite asbestos miners.

The etiological agents were considered to be "asbestiform tremolite and anthophyllite," which were said to be in both talc ores at concentrations well above standards. Smoking could not account for the excess lung cancer risk. Short-term workers may have had "very high exposures, especially in the early years of the mining operation," which might account for their excess risk (Brown et al. 1990). There was an increased risk of developing pleural changes (including pleural thickening and pleural calcification), and the prevalence is higher when there is exposure to anthophyllite (Dement et al. 1980).

The lack of an association with years worked could be due to a combination of factors above plus work in other talc operations and/or other work-related exposure to lung carcinogens.

Many of these arguments have been contradicted by further analyses.

Kleinfeld et al. (1967) compared lung cancer risk patterns of talc workers with (apparently) their own data for a similar group of asbestos insulation workers. The asbestos PMRs were 2-3 times higher among the asbestos workers for lung cancer and GI cancers. Kleinfeld et al. commented that a major difference was the increased risk of lung cancer in age groups of 40-59 and 60-79 among asbestos workers, but excesses for talc workers were among only the 60-79 age group. In addition, longevity of talc miners was longer than the national average. Age at death among the talc lung cancer cases was 3-years greater than the average of all deaths and 10-years greater than the U.S. average. The talc lung cancer cases occurred in persons exposed before wet drilling was introduced. Wet drilling reduced mean exposures 164-fold from an average of 818 mppcf to 5. Kleinfeld et al (1967) suggested part of the reason for the earlier deaths of asbestos cases compared to talc cases "may be partly due to the greater carcinogenicity of asbestos dust or to an increased level of exposure to asbestos or both."

There was excess mortality among the NY talc workers, but considerably less than the risk of asbestos workers exposed in the same time period. It is not possible to directly compare risks from the Kleinfeld et al. (1974) cohort with that of the GTC cohort. The Kleinfeld et al. cohort et al is older, had worked decades earlier than the GTC cohort, and consisted of workers with more than 15-years tenure and 40+ years tenure. Vanderbilt workers included many short-term workers with 26-years as the maximum possible years worked and no analysis by years-worked (Brown et al. 1979, 1980). In addition, overall mortality was over twice as great in the Kleinfeld et al cohort, i.e., 42% Vs. 19%. *When stratified by years worked in subsequent follow-ups there were 2 cases with >20-years tenure (SMR = 1.82) and 5 cases with >10-years tenure (SMR = 2.17) (Brown et al, 1990). Gamble (1993) reported risk ratios less than 1.0 for lung cancer cases with >15-years tenure and adjusted for smoking. These data are suggestive of a different mortality pattern of GTC talc workers compared to the Kleinfeld talc cohort.*

Smoking. *Further updates of the GTC cohort revealed that all of the lung cancer cases were or had smoked cigarettes, while only 73% of controls had ever smoked. Also, smoking latencies for GTC cases was consistent with latency from studies of smokers. Talc latencies were too short to attribute lung cancer etiology to talc exposure or work (Gamble 1993). This is particularly true for short-term workers where the risk of lung cancer was highest and talc exposure (or most any work exposure including asbestos) too short to be plausible. Risk among workers with more than 1-year exposure was increased about 2-fold compared to the US population. This degree of increased risk is in large part plausibly attributable to smoking.*

High Exposure of Short-Term Workers. *Gamble (1993) matched on date of hire in the nested case control study of lung cancer. Thus, cases and controls had equivalent opportunities for very high exposures. Six of the lung cancer cases had less than 3-months tenure, several with only a few days, so there were very few opportunities for excessive cumulative exposure. Honda et al (2002) showed that lung cancer cases had lower exposures than other subgroups. For example, median cumulative exposure of lung*

cancer decedents was 347 mg/m^3-days, which was less than all decedents (520), ischaemic heart disease decedents (376), all NMRD decedents (888), other NMRD decedents, pulmonary fibrosis decedents (3,759). Thus there is no evidence to support the speculation that excessively high exposure in short-term workers could explain their increased risk.

Pleural Changes. *Gamble et al. (1979a, b, 1982) showed that the prevalence of pleural changes in GTC talc workers was essentially the same among other workers exposed to talc containing no measurable quantities of amphiboles. Thus it would appear that the pleural thickening observed in NY talc workers and other talc workers is likely due to factors other than exposure to amphiboles.*

Exposure-response (E-R): *The inverse exposure-response trends with duration of exposure were present when adjustments were made for other talc exposures and potential exposure to other work-related carcinogens (Gamble 1993). The inverse E-R trends for lung cancer and cumulative exposure are strong arguments against attributing increased risk of lung cancer to talc exposure. This argument is further strengthened by the very strong exposure-response relationship between fibrosis and cumulative talc exposure as well as the higher exposure of NMRD and fibrosis cases compared to lung cancer cases (Honda et al. 2003).*

TABLE A2

Summary of results for Lung Cancer and Mesothelioma from studies of NY Talc workers. All but two of the studies (Kleinfeld et al. 1967, 1974) were the same cohort of GTC workers.

Reference	Study Characteristics	Lung Cancer	Mesothelioma
Kleinfeld et al. (1967)	220 NY Talc Miners ≥ 15 yrs tenure in 1940; 1965 follow-up, 91 total deaths, PMR	PMR=3.44 (1.65-6.3) (11 deaths)	1 peritoneal mesothelioma (1.1%)
Kleinfeld et al. (1974)	260 NY Talc Workers ≥ 15 yrs in 1940 or between 1940-1969; 108 total deaths, PMR, follow-up of Kleinfeld et al (1967)	PMR resp cancer =3.24 (1.72-5.54) (12 lung cancer, 1 fibrosarcoma of pleura)	1 peritoneal mesothelioma (0.93%)
Brown et al. (1979,, 1980)	398 WM employed GTC 1947-1959, follow-up 1975; 18% < 1month, 24% 1mos-6 mos, 50% < 1 yr; 44% <1950;	9/3.3 = 2.73 (1.25-5.18) (p<0.05); 4 <1-yr tenure	1/74 = 1.4% (16-y talc tenure, 11 yrs construction)
Stille & Tabershaw 1982)	655 WM employed GTC 1948- 1978, vital status 1978;	10/6.4 = 1.57 (10 obs) Prior employment=2.14 (8 obs)) No prior work = 0.76 (2 obs))	
Lamm et al. (1988)	705 men employed GTC 1947-end 1977, vital status 1978	12/5=2.40(1.24-4.19) >1 yr 6/3.1=1.93(0.71-4.20) prior risk = 3.08(6/2) <1 yr 6/1.9=3.160.16-6.88) prior risk=3.33 (3/0.9)	1 electrician 15-yr latency; 20-yrs prior as miner, miller, construction
Brown et al. (1990)	710 WM employed at GTC 1947-1978 with vital status 1983;	17/8.2=2.07(1.20-3.31) >20-yrs latency <1-yr = 3.64(1.54-7.04) 1-9-yrs = 0.83(0.02-4.57) 10-19-yrs = 4.0(0.54-16.1) 20-36-yrs = 1.820.21-6.36)	Not reported,
Gamble (1993)	22 lung cancer cases at GTC 1947-1978 matched 3:1 on data of birth and date of hire.	OR lung cancer Tenure Smokers >20-y latency < 5 yr 1.0 5-15 yrs 0.63 15-36 yrs 0.42	
Honda et al. (2002)	809 WM talc workers employed GTC 1948-89 follow-up Cancer: 1950-1989 Non-cancer mortality = 1960-1989	mg/m3-d RR (n) <95 1.0 (11) <987 0.8 (9) 987 + 0.5(9) Hired : < 1955 SMR 2.86 (0.9-4.1) Hired >1955 SMR: 0. (0.2-2.4)	2 cases not considered causal due to short latency, Case 1 & very low exposure, Case 2 (3.7%)

Pn = pneumoconiosis

DRAFT DRAFT DRAFT

National Stone, Sand and Gravel Association
Steering Committee and Working Group Joint Meeting

Whistler Fairmount Hotel
Whistler, BC Canada

Sunday July 9, 2006
2:45 PM to 4:00 PM

Agenda

1. Welcome, antitrust reminder and self-sntroductions – Don James, Chairman, Steering Committee
2. Briefing – Attorney - Client Privilege - David Landin
3. Current mission and structure

Issues and Projects

4. NSSGA position regarding crystalline silica – David Landin
5. Changing standards: ACGIH, OSHA/MSHA – Gary Walter
6. Tort reform/medical criteria/mixed dust – David Landin, Lori Jarvis
7. Silica Outreach Program – Bill Walton
   a. Customer Communication White Paper (white paper concept) - Bill Walton
   b. Contractor Communication White Paper Need – Bill Walton
   c. Silica tools and materials (what we have, what we need) -- Gary Walter
   d. OHP training program
8. California: DOSH PEL – Gary Walter
9. OEHHA REL , PM_4 emission factors for crystalline silica – John Hayden
10. New Business
    - Public forum questions (Dr. John Richards)
11. Adjourn

Attorney-Client Privilege; Attorney Work Product; Joint Defense Privileged

National Stone, Sand & Gravel Association

Steering Committee
Minerals Definition Task Force
Joint Meeting

Fairmont Chateau Whistler
Whistler, British Columbia, Canada

Sunday, July 9, 2006
1:15 pm – 2:30 pm

Agenda

1. Call to order, welcome and self-introductions – Don James
2. NSSGA's Antitrust and Attorney-Client Privilege Policies
3. Minerals Definition Task Force objectives – Kelly Bailey
4. Review financial statement
5. Briefing / update:
    a. ASTM Quarry Method test protocol
    b. Response to EPA regarding naturally occurring asbestos / El Dorado Hills
        - R.J. Lee Group
        - NSSGA
    c. Meetings with key EPA Headquarters staff
        - Potomac Communications Group
        - Briefing tools
        - Support from other entities
    d. NIOSH activity
    e. Pending publication of three science study papers to update literature
    f. MSHA proposed rulemaking - October 2006
    g. Projects remaining (see summary)
    h. Additional briefings needed
    i. Timeline
    j. Financial implications / budget review
6. Discussion
7. Adjourn

6-26-06

National Stone, Sand and Gravel Association
Steering Committee Meeting

Whistler Fairmount Hotel
Whistler, BC Canada

Sunday July 9, 2006
12:00 Noon – 1:15PM

Agenda

1. Welcome, Antitrust Reminder and Self-Introductions – Don James, Chairman, Steering Committee
2. Briefing and report on attorney client privilege - David Landin, Lori Jarvis
3. Mission, Organization, roles and responsibilities - Don James
4. Financial Report **(ACTION)** – Joy Wilson

Silica Issues

5. Current concerns and response to date – Joy Wilson
   a. OSHA Rulemaking and ACC CSP **(ACTION)** – Joy Wilson
   b. Risk communication tools **(ACTION)** – Gary Walter
   c. Position paper regarding health effects – David Landin
   d. OHP document – Bill Walton
   e. Atlantic Alliance **(ACTION)** – Joy Wilson
   f. California DOSH PEL – Gary Walter
   g. OEHHA REL PM_4 – John Hayden
   h. One page on prevention, State tort reform (medical criteria) – Joy Wilson, Lori Jarvis
   i. 2007 Legal Symposium – David Landin
   j. Silica insurance exclusions, assistance options – David Landin
   k. Options for assisting companies with silica insurance exclusions - David Landin

Minerals Definition Issues

6. Legislation, regulation (MSHA/EPA); Region 9 EPA, research – Bill Ford

Product Information Issues

7. Alkali-silica and alkali-carbonate reactivity issues – Norman Jedmundsen
8. New Issues
   - Question from public forums (Dr. John Richards)
9. Adjourn

November 17, 2005

A Review of Carcinogenicity Studies of Asbestos and Non-Asbestos Tremolite and Other Amphiboles.

John Addison,* and Ernest E. McConnell†

John Addison Consultancy Ltd, Cottingham, Yorkshire, United Kingdom. HU16 4NL,
†ToxPath, Inc., Raleigh, NC.

Short Form of Title: Carcinogenicity Studies of Amphiboles

Peer Reviewed and Accepted for Publication in Proceedings of the *International Symposium on the Health Hazard Evaluation of Fibrous Particles Associated with Taconite and the Adjacent Duluth Complex*

St Paul, Minnesota

March 30 – April 1, 2003

Email: jaddison@jaddison.karoo.co.uk

ABSTRACT

Experimental animal studies comparing asbestos and non-asbestos varieties of tremolite indicate tremolite asbestos is markedly more carcinogenic. By direct analogy, the differences in carcinogenicity between tremolite asbestos and non-asbestos prismatic tremolite should be the same for the other types of amphibole that also crystallize in the asbestos and non-asbestos habits. The earliest of the experiment animal studies, done more than 25 years ago, have design limitations by modern standards including the use of injection or surgical implantation as the route of administration rather than the more relevant route of inhalation. However the differences in the carcinogenicity of amphibole asbestos and non-amphibole asbestos are sufficiently large to be clearly discernable even with the study limitations. Together with later studies on these and related minerals, there is strong evidence of a much lower hazard associated with the shorter, thicker fibers of the non-asbestos amphiboles, than is found for the asbestos analogues of the same mineral. It is possible that the non-asbestos amphiboles are no more hazardous than other silicate minerals widely considered nuisance dusts.

INTRODUCTION

We will define some basic asbestos terminology to clarify the terms used. The glossary in 'The Health Effects of Mineral Dusts' produced by The Mineralogical Society of America (Guthrie & Mossman 1993) has the following definition: "Asbestos is a term applied to asbestiform varieties of serpentine and amphibole, particularly chrysotile, 'crocidolite', 'amosite', asbestiform tremolite, asbestiform actinolite, and asbestiform anthophyllite. The asbestos minerals possess asbestiform characteristics". The Mineral Society's glossary goes on to define asbestiform as: 'an adjective describing inorganic materials that possess the form and appearance of asbestos. When applied to a mineral, the term 'fibrous' is applied when it 'gives the appearance of being

composed of fibers, whether the mineral actually contains separable fibers or not' (Veblen and Wylie 1993). Asbestiform is a subset of fibrous, where asbestiform implies relatively small fiber thickness and large fiber length, flexibility, easy separability, and a parallel arrangement of the fibers in native (unprocessed) samples. Often, asbestos fibers occur in bundles, i.e., they are often polyfilamentous. From the definition it is clear that not all fibers or fibrous minerals are asbestiform and not all fibrous minerals called asbestiform are asbestos.

A convention has developed that a fiber is any particle with an aspect ratio equal to or greater than 3:1. This stems from the fiber definition in the early UK and US fiber counting methods (Asbestosis Research Council 1969, Asbestos Textile Institute 1971, Langer et al. 1991); it could just as easily have been 5:1 or 10:1. In using these methods, the microscopist had to make a decision to count or not count a particle depending on whether the shape and size met certain size criteria. The decision was more easily and consistently made for particles with aspect ratios just higher or lower than 3:1, and much more difficult with the higher aspect ratio thresholds. Similarly, a minimum fiber length of 5 μm was arbitrarily introduced for a fiber to be counted by these methods.

The inclusion of the abundant short fibers (less than 5μm length) in the count would have made it much less consistent or reliable. Since the aim of the fiber counting rules was to differentiate between asbestos and total particles aspect ratio and length cut-off chosen was one that produced consistency and not the ratio or length that might have had greater toxicological significance. By convention then, for a fiber to be counted it has to have an aspect ratio equal to or greater than 3:1 and a length equal to or greater than 5μm (and in some rules a diameter less than 3 μm, e.g. WHO 1985).This counting strategy has nothing to do with a definition of asbestos per se; it is simply helpful to microscopists doing fiber counting method. Since fiber counting analysis is performed using a phase-contrast light microscope at a magnification of 400-450x, the minimum width that can be counted is 0.2-0.25 μm.

Many non-asbestos particles, including non-asbestos amphiboles and other minerals can have aspect ratio greater than 3:1, but that does not make them 'asbestos' even though they are technically fibers. However, it does mean that they would be counted *as if they were an asbestos fiber* when seen in the course of a count of fibers in a membrane filter sample of airborne dust. In addition, asbestos will produce asbestos dust particles that mostly have aspect ratios equal to or greater than 3:1, but it will also produce particles that have lower aspect ratio. That does not mean that these low aspect ratio particles are not asbestos, but simply that they would not be counted as asbestos in the membrane filter method. The same is true for asbestos fibers with lengths shorter than the 5 μm minimum specified in the fiber counting method.

The adoption by some scientists and regulatory agencies of the fiber counting protocol using a 3:1 aspect ratio and a length of 5 μm or greater as being in some way a definition of asbestos has no scientific basis. It has been useful an improved metric when compared to just counting particles for assessing workplace exposure to airborne fiber dust leading to better epidemiological correlations between asbestos exposures with disease.

MINERALOGY

Tremolite is one member of the calcic amphibole group of minerals that all possess similar crystal structures, basic chemical formula, although the various crystal forms have profoundly different physical properties. The group is characterized by a crystal structure described as a double chain of silicon oxide tetrahedra that is common to all members of the group. Within this chain structure are between 7 and 8 metal cations allowing wide range in elemental composition that still maintains the basic crystalline form (Deer et al. 1997). This has produced the large number of named variants or species within the amphibole group (Leake et al. 1997, 2004) Actinolite and ferro-actinolite are part of a solid solution series with tremolite and differ only in the amount of substitution of magnesium by iron.

All of the amphibole minerals, and particularly tremolite, are very resistant to chemical attack by strong acids and bases (Addison & Davies 1990) so that their biopersistence when inhaled would be expected to be very high. In addition to the chemical variability there is further variability in what is known as the crystal habit of the minerals that may arise independent of chemistry (Dorling & Zussman 1987). The habit of a mineral is a description of the way that the crystals are commonly formed, and might otherwise be described as morphology.

The most common crystal habit for any amphibole is that called prismatic; elongate prisms with a lozenge shaped cross section that grade one way into short stocky prisms and in the other way into fine needle-like crystals or ultimately fine hair-like crystals (sometimes known as byssolite). The prismatic habit is the normal form for amphiboles in igneous and metamorphic rocks and is very widespread throughout the continental crust of the planet. Some amphiboles are also found in the habit that is termed asbestiform; this means that they have crystallized as bundles or matted masses of extremely fine fibers. The appearance of these forms usually implies some sort of secondary modification such as shearing and faulting or hydrothermal alteration. These may be found in three types of geological situations; 1) cross-fiber veins where the fibers have filled planar fissures, such as in the riebeckite (crocidolite) asbestos and grunerite (amosite) asbestos mines of South Africa; 2) in shear planes where slip fiber has formed in the plane of movement of a fault or shear plane; or 3) as disseminated fiber formed by hydrothermal alteration, such as in Libby, Montana (Meeker et al. 2003).

The differences in the manner of the formation of asbestos amphiboles, compared to the prismatic and other forms, have led to subtle differences in the details of the crystal structure that, while not sufficient to warrant a different mineral name, nevertheless lead to profound differences in physical properties (Langer et al. 1991). The commercial exploitation of the asbestos amphiboles depended upon these properties, including their capacity to be readily split into long, thin fibers with high tensile strength. These physical differences also lead to

differences in the size distributions of dusts formed when the minerals are crushed, and arguably properties which impact the pathogenic potential of the material, especially their carcinogenic properties when these dusts are inhaled. Cleavage planes are planes of relative weakness along which certain minerals tend to fracture and are determined by the crystal lattice geometry. Mica, for example, is described as having a single perfect cleavage because it splits easily along the silicate sheet structure. Calcite has three perfect cleavages that form perfect rhombohedra when the mineral is crushed. Amphiboles have two sets of cleavage planes at 126° to each other and parallel to the long axis of the crystals (and parallel to the dominant prismatic crystal faces). In addition they also have a cleavage plane on (100).

These are not perfect cleavages; they are not persistent across or along the crystals and tend to be more widely spaced than the separations between the fibers of the asbestos amphiboles. The prismatic amphiboles, including byssolites, have relatively low tensile strength and the thin needle-like crystals fracture easily across the length. They also fracture along cleavage planes that are parallel to the length of the crystals. When prismatic amphiboles are crushed a relatively small proportion of the fragments formed are elongate with faces determined by the cleavages along which the crystal fractures. These elongate particles will often meet the regulatory size criteria for an asbestos fiber within the asbestos permissible exposure limits, but differ from the asbestos fibers in critical ways. The cleavage fragment fibers often show the typical lozenge shape cross section as determined by the cleavage faces, at 126° degrees to each other. The cleavage fragment fibers tend to be thicker than asbestos fibers because of the spacing of the cleavage planes, and for any given length the cleavage fragment fibers are roughly twice as thick as asbestos fibers. Very few, if any, of the cleavage fragment fibers longer than 10μm will have diameters less than 1μm. With cleavage fragment fibers the width distribution is much broader and width increases with length so aspect ratios tend to be lower and of narrower distribution. In overall size distributions the asbestos fibers have a very narrow width distribution and the width

of fibers is largely independent of length. As a result, the aspect ratio of fibers increases with length.

Since the cleavage fragments and asbestiform fibers tend to be morphologically defined by somewhat different crystal surfaces it is tempting to speculate that this may go some way to explaining the apparent differences in toxicological properties as described below.

EXPERIMENTAL ANIMAL STUDIES

Five *in vivo* experimental animal studies provide information on the variation in carcinogenicity of dusts derived from prismatic or non-asbestos tremolite and tremolite asbestos. Davis et al. (1985) remains the only inhalation experiment to be carried out using tremolite asbestos. Previously, Smith et al. (1979) used a variety of tremolite types for intrapleural injection in hamsters; Stanton et al. (1981) used two different tremolites for intrapleural implantations in rats, while Wagner et al. (1982) report on three different tremolites for intrapleural injection in rats. Later, Davis et al. (1991) used six tremolites of different morphology for intraperitoneal injections in rats. If the actinolite and ferro-actinolite amphiboles are included the number of studies increases slightly but is still small. Coffin et al. (1978, 1982, 1983) and Cook et al. (1982), used a fibrous ferro-actinolite in intrapleural injection and intratracheal instillation into rats. Pott et al. (1974, 1989, 1991) reported results from intraperitoneal injection of a granular actinolite and (later) an asbestiform actinolite. A lifetime (including exposure to the dams and gavage during the neonatal period) oral ingestion study (1% in the diet) in rats of 'blocky' tremolite did not to show evidence of carcinogenic activity (NTP 1990, McConnell et al. 1983).

Other studies might also be considered as contributing to the debate about the relative carcinogenicity of amphiboles and their asbestiform varieties. Berman et al. (1995) reviewed the size distributions of all of the asbestos dust exposures used in the Institute of Occupational Medicine inhalation studies over many years, including the Korean asbestos tremolite, and

concluded that, while no univariate measure of exposure could be found to predict lung tumor incidences, the concentration of total structures longer than 20 μm provided the best fit. Furthermore the best estimate for the carcinogenic potency of fibers greater than 0.5 μm in width was zero. The inhalation and intraperitoneal injection experiments of Davies et al. (1986) with long and short fiber amosite, the inhalation studies of various sized chrysotile (Ilgren and Chatfield 1998, McConnell et al. 1984, Wagner et al. 1984), and the cell studies of Donaldson et al. (1989, 1991), Donaldson and Golyasnya (1995), Brown et al. (1986) were aimed at understanding the relative importance of fiber length in carcinogenicity and fibrogenicity. Other mechanistic studies such as those by Kane (1991), and reviews such as those by Oberdorster & Lehnert (1991) and Jaurand (1991), among others also have a bearing on the understanding of the different reactions observed between asbestos particles and other particles with the same mineral chemistry but different morphology.

Inhalation Experiments

Davies et al. (1985) exposed rats (SPF male Wistar, whole body exposure) to a commercially mined tremolite asbestos form South Korea at concentrations of 10 mg/m^3, around 1600 f/mL, (>5 μm) for 12 months. Having produced very high levels of pulmonary fibrosis as well as 16 carcinomas and two mesotheliomas (rarely found in rat inhalation experiments) among the 39 treated animals the tremolite asbestos was described by them as the most dangerous mineral ever studied at the Institute of Occupational Medicine, UK. The Korean tremolite asbestos is the same one used later in the intraperitoneal injection experiments (Davis et al. 1991) for which full size distributions of the respirable dust were given, as shown in Figure 2.

The important feature of the size distribution of the Korean tremolite asbestos is that the vast majority of fibers are less than 0.5 μm in diameter and shorter than 5 μm in length, which is typical of asbestos amphiboles. The geometric mean diameter for Korean tremolite asbestos

fibers longer than 0.4 μm was 0.24 μm (SD 1.6) and the mean length was 1.97 μm (SD 2.11) which are somewhat longer and thicker than airborne fibers in crocidolite mining (GM diameter 0.076 μm, GM length 0.98 μm, Hwang & Gibbs 1981).

The high carcinogenicity of the Korean tremolite asbestos was attributed to the much higher airborne fiber concentration for fibers longer than 5 μm (1600 f/mL) which was almost twice that of the UICC amphiboles at the same 10 mg/m^3 dust mass concentration used grunerite (amosite) asbestos 550f/mL and riebeckite (crocidolite) asbestos 860 f/mL, Davis et al. 1978). This also is a reflection of the finer diameter of the Korean tremolite asbestos.

Injection and Implantation Experiments

Smith et al. (1979) injected a range of tremolites and tremolitic talcs intrapleurally into hamsters (of unspecified type) at doses of 10 mg and 25 mg. The samples were identified as follows:

The animals were allowed to survive up to 600 days after which the final survivors were sacrificed for necropsy. No tumors were found in the final survivors. The samples used by Smith et al. 1979 and described as asbestos or asbestiform produced higher levels of fibrosis and numbers of mesotheliomas in the hamsters than those described as tremolite or tremolitic talc. Most of the tumors were diagnosed as mesotheliomas.

Campbell et al. (1979) examined some of the tremolites used by Smith et al. 1979 and described two of the tremolites (275 and FD72) in more detail. The images of the fibers clearly show FD72 (tumor rate 5/23 and 3/13) to be asbestos and 275 (tumor rate 0/31 and 0/34) to be a prismatic amphibole. This is reflected in the numbers of fibers of length > 10 μm and diameters less than 1μm in the tremolite asbestos, and their absence in the non-asbestos minerals. Similarly, in tremolite FD72 many more of the fibers longer than 5μm had aspect ratios greater than 10:1 than in tremolite 275 (23 – 0, and 19 – 1 using the petrographic microscope and the Scanning Electron Microscope respectively).

Non-asbestos tremolite 14 (FD 14, tumor rate 0/35) was later evaluated by Wylie et al. (1993) and confirmed to be a tremolitic talc with very few tremolite fibers in the size ranges longer than 5 μm and less than 1μm diameter.

This study was criticized for being deficient in a number of ways (Federal Register, 1992). In particular, the fiber size measurements and fiber characterizations were found to be inadequate for the purposes of identification of the materials as tremolite asbestos or prismatic tremolite. The later characterizations by Campbell et al. (1979) and by Wylie et al. (1993) improved on the original ones and the classification of the mineral types appears established. The higher carcinogenicity of those materials described as asbestiform compared to those of tremolitic talc or non-asbestos tremolite is without doubt.

Wagner et al. (1982) used a tremolite from the California talc deposits (A), a prismatic tremolite from Greenland (B) and a tremolite asbestos from Korea (C, probably from the same source as the one in Davies et al. 1985) for a series of intrapleural injection experiments with SPF Sprague-Dawley and Wistar rats and a range of *in vitro* tests. The rats were 8 –10 weeks old when injected and were allowed to live out their lives. Median survival times after injections were 644, 549, and 557 days respectively for samples A, B and C.

The value of the Wagner et al. (1982) injection experiments was impaired by the poor survival rates as a result of infection of the positive control animals injected with riebeckite (crocidolite) asbestos. Nevertheless, the tremolite (C) asbestos was the only one the three tremolites that showed carcinogenic activity producing mesotheliomas in 14 of 47 rats (30%). Neither of the other non-asbestos tremolites produced any tumors in the 31 and 48 rats used. The fiber size data as presented are not amenable to numerical evaluation, but measurements taken from the published diagrams show that in the tremolite (C) asbestos about 25% were longer than 10 μm and less than 0.6 μm in width. The non-asbestos forms had no fibers at all in that size range

(Sample A California, or Sample B, Greenland). Table 2 shows Wagner's figures for the numbers of particles, fibers longer than 1 μm, and fibers longer than 8 μm with widths less than 1.5 μm; the differences are obvious with Tremolite C containing many more long fibers.

The *in vitro* tests used by Wagner et al. (1982), including mouse peritoneal macrophage lactic dehydrogenase (LDH) and B-glucuronidase (BGL) release, cytotoxicity to V79-4 cells and giant cell stimulation with A549 cells confirmed the relative toxicity of the different tremolite morphologies *in vivo*. So, while the study remains limited by the poor survival of the positive controls, it is nevertheless useful in that it reproduces the general findings of Smith et al. (1979).

Stanton et al. (1981) described a series of 70 experiments where a wide range of different fibers were implanted at doses of 40 mg in hardened gelatin on to the left pleural surface of Osborne-Mendel rats by thoracotomy. It should be noted that in contrast to intrapleural or intraperitoneal injection, the use of the "hardened gelatin" exposure technique literally holds the fibers in contact with the target tissue (pleura) and does not allow for potential macrophage phagocytosis and clearance of the particles. Therefore this technique may create the highest effective dose of all of the exposure methods used for assessing the potential carcinogenicity of fibers. Stanton et al. 1981 reported on two tremolite asbestos samples from the same lot, described as "in the optimal range of size for carcinogenesis" and "distinctly smaller in diameter than the tremolite fibers used by Smith et al. (1979)". As they anticipated the two tremolites produced mesotheliomas in 21 and 22 animals out of the 28 used, with a 100% tumor probability. The tremolites contained very high numbers of fibers in the Stanton size range (>8 μm in length and <0.25 μm diameter) with 1.63×10^{8} and 2.76×10^{7} respectively *in each dose* for tremolites 1 and 2. In addition, the talc (No 6), which produced no tumors in the Stanton study, was actually New York State tremolitic talc (Wylie et al. 1993) with 40-50% non-asbestos tremolite and talc fibers, in fact the same material as used by Smith et al. (1979) and identified as FD 14. The general relationship between the probability of developing a tumor in these experiments and the

common logarithm of the number of fibers > 8μm in length and less than 0.25 μm in diameter per microgram of implanted dust was highly significant (Figure 3).

There were however a number of problematical experiments in the Stanton series where tumors developed for test materials with no fibers in the critical size range, and one where no tumors had developed even with large numbers of critical fibers present. Some of these results were attributed to large numbers of fibers with sizes close to the critical range, and others to problems of clumping and fragmentation in the fiber preparations for transmission electron microscopy analysis

Figures 3 and 4 show the general relationships developed, and described by Stanton as highly significant, between the numbers of fibers per microgram in the dose and the probability of tumor development. The statistical relationships between the fiber numbers in the different sets and probabilities of tumor development have not been evaluated but the diagrams show that the correlation for the shorter classes of fiber is much weaker than that for the longer fibers. It is reasonable to suggest that there must be more short fibers per microgram in the short fiber dusts than in the longer fiber dusts so the poorer correlation for short fibers is, if anything, even more indicative of their lack of importance in tumor development.

The size distributions given in Stanton et al. (1981) do not make it easy for full comparison with other size distributions of known asbestos minerals because the size classification was relatively crude and the method of exposure (hardened gelatin) was unique. The two tremolite samples however have sufficient numbers of long fibers with diameters less than 0.5 μm to indicate that their identification as asbestos is reasonable. The size distributions are somewhat unusual for pure asbestos as is seen in Figure 5 which shows Tremolite 2 to have a bimodal distribution which suggests that it is actually a mixture of tremolite asbestos and prismatic

tremolite. Such an occurrence in poor commercial quality tremolite asbestos formations is common.

Wylie et al. (1993) re-examined Tremolite 1 and 2 as well as Talc 6 that were used in the Stanton studies. They state that Tremolites 1 and 2 are the same material, tremolite asbestos from California, with all the characteristics of commercial amphibole asbestos. The two size distributions given by Stanton differ somewhat but they are similar and have the appearance of a mixed asbestos – prismatic fiber assemblage.

In contrast, the size distribution of Stanton's Talc 6 shows the much thinner, shorter distribution (Figure 6) not typical of a prismatic tremolite fiber population even though it consists of 40-50% tremolite. Talc 6 produced no tumors despite containing more fibers in the "Stanton fiber" range than Tremolite 2, and almost as many as Tremolite 1, both of which had a 100% probability of producing tumors.

This talc (6), or tremolitic talc, was reported by Wylie et al. (1993) as being identified in Stanton's laboratory notes as Nytal 300. Pure talc is a specific mineral with a closely defined chemical composition and crystal structure. Commercial producers however often named their products as 'talc' even though they contained less than 50% of the mineral talc.

Davies et al. (1991) used six tremolites of differing morphologies in a series of intraperitoneal fiber in saline injection experiments with male SPF Wistar rats. These were identified as follows:

1. Tremolite asbestos from Jamestown, California, United States;
2. Tremolite asbestos from Korea;
3. Tremolite asbestos from National Coal Board Laboratory, Swansea, Wales, Great Britain;

4. Tremolite, long needle-like crystals from Ala di Stura, N. Italy;
5. Tremolite, short needle-like crystals from Dornie, NW Scotland, Great Britain;
6. Tremolite, prismatic crystals from Shinness, N. Scotland, Great Britain.

The tremolite from Korea was the same material as was used in the earlier tremolite inhalation and injection experiments by Davis et al. (1991). The fiber size distributions were assessed by counting and measuring 300 fibers of all sizes in a known weight of sample deposited on to a polycarbonate filter using Scanning Electron Microscopy. At 10,000 times magnification the effective minimum diameter that is visible is 0.1 μm, so the effective minimum length of a counted fiber was 0.4 μm. This was followed by the counting and measurement of a further 100 fibers longer than 5 μm. The data were combined to calculate the numbers of fibers in a series of length and diameter classes in the 10 mg dose administered to the rats. In addition, the numbers of particles (Aspect ratio less than 3:1) were also counted and estimated for each dose.

The rats were allowed to live out their full life span or until they showed signs of debility or tumor formation. Statistical analysis of the times at which death from mesothelioma occurred was used to calculate survival curves and these were correlated with the fiber doses received by each animal.

Table 3 shows the relative hazard ranking, the numbers of mesotheliomas and the fiber numbers in the doses. The relative hazard was derived from Cox's proportional hazards model (Cox & Oakes 1984) and is a function of the numbers of animals developing mesothelioma and their median survival times. The values given in the table differ from those shown in Davis et al. (1991) only in that the hazard is expressed arithmetically as a multiple of the lowest hazard, and the fiber numbers are expressed as those in the dose.

The main conclusions of the study were: 1) that all of the materials appeared to have some potential to cause mesothelioma by intraperitoneal injection in rats; 2) that fiber numbers alone were not sufficient to explain the differences in response, nor were the fiber numbers in the 'Stanton' fiber class able to fully explain the response; and 3) that the Dornie and Shinness material would be unlikely to pose a risk of mesothelioma to humans from inhalation of the dust. The spontaneous occurrence of peritoneal mesothelioma in male rats of this strain may account for the small numbers of tumors found in the animals injected with the latter two dusts (Pott et al. 1991).

Coffin et al. (1978, 1982, 1983) and Cook et al. (1982) confirmed that ferro-actinolite asbestos has a high potency for generating mesothelioma in rats. In each case the ferro-actinolite asbestos had large numbers of fibers in the 'Stanton' range. The papers by Coffin and his colleagues were based on experiments using intratracheal instillation and intrapleural injection of an actinolite asbestos from the Mesabi Range (USA) iron ores in comparison to UICC amosite. The results were problematical in that the response from the amosite was lower than expected from previous experiments (Stanton et al. 1981). In Coffin et al. (1983) 33.6% of F344 rats injected intrapleurally with 20 mg of UICC amosite developed mesothelioma. The response to the actinolite asbestos was lower than that from the UICC amosite or amosite in general in terms of the mass dose used, but the response relative to the numbers of Stanton fibers was higher. Cook et al. (1982) explained the relatively high response from the ferro-actinolite as resulting from shortening and splitting of the fibers in the lungs and on the pleural surface of the rats.

Pott et al. (1988) reported more than 80% of rats with tumors two years after intraperitoneal injection of 0.3 mg of a German actinolite although the given size distribution of the actinolite is not provided. Pott et al. (1989) then reported 56% of rats with tumors after an injection with 0.25 mg of (presumably) the same German actinolite. The size distribution is not detailed but shows 90% of the fibers as less than 0.2 μm in length and 10% longer than 4.2 μm. In contrast,

when a dose of 4 x 25 mg of 'granular' actinolite was used in similar experiments (Pott et al. 1974) no tumors were found.

Grunerite (Amosite) Asbestos Studies

The inhalation and intraperitoneal injection experiments of Davies et al. (1986) used long and short fiber amosite asbestos. These were produced from the same bulk batch of amosite, the short form by ceramic ball milling and the long by elutriation. Importantly, TEM examination showed no loss of crystallinity in the milled short fiber sample. In the inhalation studies rats were exposed for one year (224 days in 12 months) to 11.9 and 11.6 mg/m^3 of respirable dust for the long and short fiber types respectively. The aerosol contained 2,060 and 70 f/mL for fibers longer than 5 μm, and 1,110 and 12 f/mL for fibers longer than 10 μm. In the injection studies two batches of rats received doses of 10 mg and 25 mg of the respirable dust collected from the inhalation experiment chambers using a vertical elutriator.

The results showed that rats exposed to the long fiber grunerite (amosite) asbestos developed significantly higher levels of pulmonary fibrosis and more lung tumors than rats exposed to the short fiber grunerite (amosite) asbestos. In fact the animals exposed to the short fiber developed no more fibrosis than did the control animals, no pulmonary tumors and only one peritoneal mesothelioma that was considered to be unrelated to the dust exposure as the type had previously been reported in untreated rats. The animals exposed to the short fiber had significantly higher burdens of asbestos in their lungs immediately after the inhalation period, and they remained higher throughout the following six months of clearance. The injection experiments produced mesothelioma in 88% and 95% of rats treated with 10 and 25 mg respectively of the long grunerite (amosite) asbestos, while the short fiber grunerite (amosite) asbestos produced 0% and 4% (1 animal) tumors with the same respective doses (mass) (Table 4). The short fiber grunerite

(amosite) asbestos contained about 0.1% of fibers longer than 10 μm and about 2% longer than 5 μm while the long fiber grunerite (amosite) asbestos contained more than 11% longer than 10 μm and 3% longer than 25 μm. The diameter distributions were very similar with about 50% less than 0.5 μm in width.

These results were taken as an indication that the short fiber grunerite (amosite) asbestos showed a much lower relative pathogenicity than the long fiber grunerite (amosite) asbestos.

In Vitro CELL STUDIES

The cell culture studies of Donaldson et al. (1989, 1991, and 1992) Brown et al. (1986) and Hill et al. (1995) have generally confirmed the impression that fibers shorter than 5 μm, and indeed possibly less than 10 μm, have little pathologic effect other than what might be expected from a general respirable silicate mineral dust. Tumor necrosis factor released from macrophages was shown to be dependent on fiber length as demonstrated by the long and short fiber grunerite (amosite) asbestos (Donaldson et al. 1992). The same minerals showed that release of superoxide anions by macrophages differed significantly (Hill et al. 1995). Since such factors are associated with the development of inflammation, pulmonary fibrosis, and tumor formation, this supports the view that fiber length is an important element in determining the pathogenicity of fibers.

OTHER RELEVANT STUDIES

The studies at IOM (Miller et al. 1999a, b and Searl et al.1999) confirm that biopersistence was a significant factor controlling the pathogenicity in animals of a wide range of different synthetic mineral fibers, but for durable fibers the most important factor was fiber length. The fibers used were: glass microfiber, JM 100/475; MMVF 10, 21, 22 and Refractory ceramic Fibers 1, 2, and 3, from the Thermal Insulation Manufacturers Association repository of size selected fibers; a

silicon carbide whisker fiber and the long fiber grunerite (amosite) asbestos as used by Davis et al. (1986). In the intraperitoneal injection studies the best correlation with capacity to produce mesothelioma was with the *in vivo* biopersistence factor (derived from measurement of fibers before and after intratracheal instillation) and the number of fibers longer than 20μm with diameters less than 0.95 μm. In the inhalation studies with the same suite of fibers the pulmonary tumor production (lung cancer) was best predicted by a function of the dissolution rate (measured in continuous flow through with simulated physiological saline solution) and the numbers of fibers in the length range greater than 20 μm with diameters less than 0.95 μm.

DISCUSSION

The main question that has been asked of these studies is to what extent they support the hypothesis that the carcinogenicity of fibers depends upon morphology. A second question that is being debated to what extent the short mineral fibers contribute to the carcinogenicity in humans. There are limitations to the injection or implantation assessments of carcinogenicity that reduce their ability to predict the outcome of inhalation of the same materials by humans (US EPA 1986). These include the avoidance of normal defence mechanisms of the inhalation process, the unnatural introduction of large doses to sensitive tissue sites, possible clumping of dusts introducing even higher doses at some sites, and the reduction of normal lung clearance mechanisms. However, the net result of these limitations is to over-estimate carcinogenicity by these methods, so that a negative finding is a strong indication that a given mineral dust is unlikely to be carcinogenic when inhaled by humans.

The early studies of Wagner et al. (1982) and Smith et al. (1979) are limited by poor survival and uninformative size distribution measurements. However, both experiments showed no potential for prismatic amphibole fibers to cause tumors by inhalation or by injection. So while limitation do exist, they ought not to be seen as grounds for disregarding the results and general concepts

derived from the experimental animal studies indicating that amphibole asbestos minerals are carcinogenic while the prismatic amphiboles or cleavage fragments are markedly less active.

Some questions have been raised about the interpretation of the Davis et al. (1991) study which we will answer. For example, the authors stated that the response from the Shinness fiber was no more than would be expected from control animals, and that the non-asbestos tremolites were unlikely to pose a specific mesothelioma risk to humans by inhalation. It was suggested that these two tumors, with the non-asbestos Shinness dust, were significant since there were no tumors among animals in many other experiments from the same laboratory (IOM) (Federal Register 1992). The experiments referred to in the Federal Registry were inhalation experiments with other asbestos fibers, and that, other than with the Korean tremolite, these have rarely produced mesotheliomas in rats. The background mesothelioma incidence is higher when the route of administration is by injection. Furthermore, Stanton et al. (1981) implantation studies have shown a percentage of animals with tumors in the range of 0 to 10% may well be within the expected range for a 40 mg dose of mineral particles of any type not introduced by inhalation.

The size distributions of the fiber types show that the tremolite asbestos from different geological locales as exemplified by the Californian (Jamestown) sample, are dominated by very much thinner fibers than the prismatic tremolites, as exemplified by the Shinness sample, which contain almost no fibers longer than 8 μm and less than 1 μm in diameter. While it is true that the response could be explained simply as a dose response to the numbers of Stanton fibers, yet this fails to explain all of the variance in the results between the various growth habits in which tremolite naturally occurs. It is a distinct possibility that, as with Stanton's experiments, the low responses from the non-asbestos Shinness fibers and the Dornie fibers are inert dust responses.

A second criticism in the interpretation of these results stems from the high tumorigenicity of the Italian (Ala di Stura) tremolite (Davis et al. 1992) This was described in the paper as a spicular

(the same as acicular, a sub-type of prismatic) non-asbestos variety of tremolite which would not be expected to produce tumors; so the high tumor rate has been used to suggest that acicular and byssolite amphiboles do indeed have a similar carcinogenicity to the asbestos amphiboles. It has been shown that the Ala di Stura tremolite sample contains a sub-set of asbestiform tremolite fibers that appear as extremely long and fine fibers but which, because of the limitations of fiber sizing methodology, are not fully expressed in the fiber numbers as reported in the study (Figure 1b).

The tumor response from the Ala di Stura tremolite was unusually high compared to the number of Stanton fibers in the sample, but an important factor in the response was the timing of the mesotheliomas in the life spans of the animals. Two thirds of the rats exposed to the Ala di Stura tremolite developed mesothelioma, but very late in life (median survival time was 755 days). In contrast the three asbestos samples had much shorter median survival times ranging from 301 days to 428 days. (The Korean tremolite asbestos had a median survival time of 428 days compared to 325 days in the earlier study with a 25 mg dose). The median survival time for those animals that develop mesothelioma appears to be inversely related to dose, as seen in Davis et al. (1991), so the response from this dust could be simply that which might be expected from a trace asbestos component in a dust injected into animal at high concentrations.

It was also pointed out in the original report that the tremolite asbestos from Swansea had produced a response that was much higher than expected given the number of Stanton fibers in the dose. Both the Swansea tremolite asbestos and the Korean tremolite asbestos produced the maximum response in mortality but the high Hazard Index of the Swansea asbestos, calculated in the statistical analysis, was the result of the much faster tumor induction. It was suggested that this may have been the result of a masking of the response to simple fiber numbers by the overdose of asbestos forms, and that a multi-dose-response experiment might produce a clearer picture of the relative potencies of these types.

The Stanton studies confirmed the high tumorigenicity of tremolite asbestos and identified the Stanton Fiber range, fibers > 8 μm with diameters < 0.25 μm, for which the correlation between fiber numbers and mesothelioma generation was highly significant. Had the size classes and instillation method been different, the 'Stanton Fiber' critical size may well have been different. The authors stated that shorter and thicker size classes also correlated with mesothelioma potency, and that it should not be assumed that they had no potency. However, as can be seen in Figure 7, the numbers of fibers in the different classes are strongly correlated.

So it is to be expected that if the tumorigenicity is correlated strongly with numbers in the long, thin class of fibers it will also correlate with the fiber numbers in the shorter classes. That does not necessarily imply a causal relationship, and these short fibers may indeed have insignificant tumorigenicity. Even particulates that are considered relatively innocuous, e.g. FeO, magnetite can produce tumors by injection techniques if the dose is high enough (Pott et al. 1991).

As can be seen in Figure 8 many of the mineral and glass fibers in the experiments had less than 10 % probability of generating mesothelioma despite having huge numbers of fibers in the administered dose in the size range of 4 –8 μm length with no fibers in the longer classes. It is noteworthy; the fibrous talc minerals (5 and 7) produced no tumors despite having large numbers of short, thin fibers. The halloysites produced only 5 and 4 tumors despite having among the highest numbers of short fibers. Halloysite has the same tubular morphology as chrysotile asbestos despite having a little thicker fundamental diameter (0.07 μm). The attapulgites (palygorskite) produced few (2/29) tumors with similarly high numbers of fibers shorter than 8 μm. However, one long fiber attapulgite has been found by Wagner et al. (1987) to be capable of producing large numbers of mesotheliomas in rats by intraperitoneal injection. Both halloysite and attapulgite have been described as asbestiform but neither fiber-type is asbestos.

The size distributions of the various fibers used by Stanton et al. (1981) are in many cases highly unusual but a detailed discussion of all their full fiber size distributions is beyond the scope of this paper; some contained no long fibers, some contained no short fibers, some contained no fibers thinner than 0.5 μm, and others contained no fibers thicker than 0.5 μm. The tremolites however were unusual in having bimodal distributions consistent with a mixture of tremolite asbestos and prismatic non-asbestos tremolite (Figure 5).

One important factor in the Stanton studies that has implications for many other injection and implantation experiments is the range and distribution of the results found. There are a large number of dusts producing between 0 and 10% mesothelioma in experimental animals even though many of these samples contained more than 100,000 fibers per microgram of implanted dust. In a 40 mg dose implanted there are 40,000 times more fibers present than in a microgram. It is reasonable to conclude that this range of tumor production may be the "normal" background for his mineral dust implantation technique. In addition, Stanton's implantation controls had a 2.8% incidence of pleural sarcomas and all controls had an age-adjusted rate of 7.7±4.2%. Also, Pott et al. (1991) using intraperitoneal injection stated that tumor rates of below 10% in small groups should be regarded as spontaneously occurring or induced non-specifically. The background rate of his non-injected controls is 0%, but up to 10% for saline injection controls, which is highly significant when compared to non-injected animals.

One implication of this observation would be that the testing of materials by the implantation or injection of unrealistically high doses might be useful a screening test for mesothelioma potency in humans by inhalation. In addition, both routes of exposure do not allow for normal physiological removal as would be expected after inhalation (McConnell, 1995). The Stanton method is particularly problematic in this regard because the fibers are 'held in place', i.e. in contact with the mesothelium in the gelatin vehicle. For these reasons the methods is very useful when a negative result is obtained for the assessment of fundamental differences between fiber-

types and concepts of carcinogenic activity. But positive results are of limited use as predictors of the risk to humans from inhalation of more general dusts. Furthermore, the doses to which the animals are exposed are probably many orders of magnitude higher than would be expected from exposure of humans to airborne dust.

CONCLUSIONS

The conclusion that should be drawn from the evaluations of this set of studies is that there is very little evidence of carcinogenicity from exposure of animals to mineral fragments or short fibers formed from normal prismatic amphibole minerals. No positive carcinogenicity has been found with any experiment using non-asbestos amphibole dust (Ilgren 2004). Furthermore, when genuinely short fiber amphibole asbestos has been used in inhalation or injection experiments they have also been shown to have no carcinogenic properties. Evidence from experiments with other mineral fibers suggests those fibers in excess of 20 μm and with diameters less than 1 μm are necessary to cause cancer. This is probably because such long fibers cannot be phagocytized by resident macrophages and therefore, cannot be removed from the lung (Lippmann et al. 2000). This explains the lack of carcinogenicity of cleavage fragment fibers of amphiboles since these rarely if ever contain fibers of these critical dimensions.

ACKNOWLEDGEMENTS

The authors are grateful to the National Stone, Sand and Gravel Association of the United States for financial support in the preparation of this document. The authors would like to thank to Dr. R.P. Nolan for helpful comments during editing.

References

Addison, J., Davies, L.S.T. (1990). Analysis of amphibole asbestos in chrysotile and other minerals. *Ann. Occup. Hyg.* **34**, 159-175.

Asbestos Textile Institute. (1971). Measurement of airborne asbestos fiber by the membrane filter method. Asbestos Textile Institute, Pompton Lakes, N.J.

Asbestosis Research Council (1969). Technical Note No. 1, Measurement of Airborne Asbestos Dust by the Membrane Filter Method, Asbestosis Research Council, Rochdale, England.

Berman, D.W., Crump, K.S., Chatfield, E.J., Davis, J.M.G., Jones, A.D. (1995). The shapes sizes and mineralogy of asbestos structures that induce lung tumors or mesothelioma in AF/HAN rats following inhalation. *Risk Analysis.* **15**, 181-195

Brown, G.M., Cowie, H., Davis, J.M.G., Donaldson, K. (1986). In vitro assays for detecting carcinogenic mineral fibers: a comparison of two assays and the role of fiber size. *Carcinogenesis* **7**, 1971-1974.

Campbell, W.J., Steel, E.B., Virta, .R.L., Eisner, M.H. (1979). Relationship of mineral habit to size characteristics for tremolite cleavage fragments and fibers. U.S. Bureau of Mines Report of Investigation No. 8367.

Coffin, D.L., Palekar, L.D., (1978) EPA Study of biological effects of asbestos-like mineral fibers. In National Bureau of Standards Special Publication 506. Proceedings of the Workshop on Asbestos: Definitions and Measurement Methods held at NBS, Gaithersburg. 163-177.

Coffin, D.L., Palekar, L.D., Cook, P.M. (1982). Tumorigenisis by a ferroactinolite mineral. *Toxicol. Lett.* **13**, 143-150.

Coffin, D.L., Palekar, L.D., Cook, P.M. (1983). Correlation of in vitro and in vivo methods by means of mass dose and fiber distribution for amosite and fibrous ferroactinolite. *Environ. Health Perspect.* **51**, 49-53.

Cook, P.M., Palekar, L.D., Coffin, D.L. (1982). Interpretation of the carcinogenicity of amosite asbestos and ferroactinolite on the basis of retained fiber dose and characteristics in vivo. *Toxicol. Lett.* **13**, 151-158.

Cox, D.R., Oakes, D. (1984). Analysis of Survival Data. Chapman & Hall. London.

Davis, J.M.G., Addison J., McIntosh, C., Miller, B.G., Niven, K. (1991). Variations in the carcinogenicity of tremolite dust samples of differing morphology. *Ann. N. Y. Acad. Sci.* **643**, 473-490

Davis, J.M.G., Addison, J., Bolton, R.E., Donaldson, K., Jones, A.D., 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. Pathol.* **67**, 415-430

Davis, J.M.G., Addison, J., Bolton RE, Donaldson K, Jones AD, Miller BG. (1985). Inhalation studies on the effects of tremolite and brucite dust in rats. *Carcinogenesis* **5**, 667-674.

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.

Deer, W.A., Howie, R.A., Zussman J. (1997) Rock-Forming Minerals, Volume 2B, *Double Chain Silicates.* The Geological Society, London, pp. 764.

Donaldson, K., Brown, G.M., Brown, D.M., Bolton, R.E., Davis, J.M.G. (1989). The inflammation-generating potential of long and short fibre amosite asbcstos samples. *Br. J. Indust. Med.* **46**, 271-276

Donaldson, K., Szymaniec, S. Li, X.Y., Brown, D.M., Brown, G.M. (1991). Inflammation and immunomodulation caused by short and long amosite asbestos samples. In: *Mechanisms in Fiber Carcinogenesis*, (Brown, R.C., Hoskins, J.A., Johnson, N.F., Eds), Plenum Press, New York, pp. 287-307.

Donaldson, K., Li, X.Y., Dogra, S., Miller, B.G., Brown, G.M. (1992). Asbestos-stimulated tumour-necrosis-factor release from alveolar macrophages depends on fiber length and opsonization. *J. Patho.* **168**, 243-248.

Donaldson, K., Golyasnya, N. (1995) Cytogenetic and pathogenic effects of long and short amosite asbestos. *J. Pathol.* **177**, 303-7.

Dorling M, Zussman J. (1987) Characteristics of asbestiform and non-asbestiform amphiboles. *Lithos* **20**, 469-489.

Federal Register (1992) Occupational exposure to asbestos, tremolite, anthophyllite and actinolite; Final Rule. 29 CFR Parts1910 and 1926.

Guthrie, G.D., Mossman, B.T. (1993) Health Effects of Mineral Dusts. Reviews in Mineralogy, Volume 28, Mineralogical Society of America, Washington, D.C., pp 555-576.

Hill, I.M., Beswick, P.H., Donaldson, K. (1995) Differential release of superoxide anions by macrophages treated with long and short-fiber amosite asbestos is a consequence of differential affinity for opsonin. *Occup Environ Med.* **52**, 92-96

Hwang, C.Y., Gibbs, G.W. (1981) The dimensions of airborne asbestos fibres- I. Crocidolite from the Kuruman area, Cape Province, South Africa. *Ann. Occup. Hyg.* **24**, 23-41

Ilgren, E., Chatfield, E. (1998). Coalinga Fiber – A short amphibole-free chrysotile. Part 2: Evidence for lack of tumourigenic activity. *Indoor Built Environ.* **7**, 18-31.

Ilgren EB (2004) The biology of cleavage fragments: A brief synthesis and analysis of current knowledge, *Indoor Built Environ.* 13,343-356.

Jaurand, M-C. (1991) Mechanisms of fibre genotoxicity. In: Mechanisms in Fiber Carcinogenesis, (Brown, R.C., Hoskins, J.A., Johnson, N.F., Eds), Plenum Press, New York, pp. 287-307.

Kane, A.B. (1991) Fiber dimensions and mesothelioma: a reappraisal of the Stanton Hypothesis. In: *Mechanisms in Fiber Carcinogenesis*, (Brown, R.C., Hoskins, J.A., Johnson, N.F., Eds), Plenum Press, New York, pp. 131-141.

Langer AM, Nolan RP, Addison J (1991) Distinguishing between amphibole asbestos fibers and elongate cleavage fragments of their non-asbestos analogues In: *Mechanisms in Fibre Carcinogenesis,* (Brown RC, Hoskins JA, Johnson NF Eds), Plenum Press, New York, pp 253-267.

Leake, B.E., Wooley, A.R., Arps, C.E.S., Birch, W.D., Gilbert, W.D., Grice, J.D., Hawthorne, F.C., Kato, A., Kisch, H.J., Krivovichev, V.G., Linthout, K., Laird, K, Mandarino, J.A., Maresch, W.V., Nickel, E.H., Rock, N.M.S., Schumacher, J.C., Smith, D.C., Stephenson, N.C.N., Ungaretti, L., Whittaker, E.J.W., Youzhi, G. (1997) Nomenclature of amphiboles: Report of the Subcommittee on Amphiboles of the International Mineralogical Association, Commission on New Minerals and Mineral Names. *Min. Mag.* **61**, 295-321.

Leake, B.E., Wooley, A.R., Birch, W.D., Burke, E.A.J., Ferraris, G., Grice, J.D., Hawthorne, F.C., Kisch, H.J., Krivovichev, V.G., Schumacher, J.C., Stephenson, N.C.N., Whittaker, E.J.W., (2004) Nomenclature of amphiboles: additions and revisions to the International Mineralogical Association's amphibole nomenclature. *Min. Mag.* **68 (1)**, 209-215.

Lippmann, M., Chiazze, L., Coultas, D.B., Driscoll, K.E., Kane, A.B., Lockey, J.E., McConnell, E.E., Oberdörster, G., Rhomberg, L.R., Utell, M., and Warheit, D.B. (2000). NRC Report on

the Expert Panel on Health Effects of Asbestos and Synthetic Vitreous Fibers: The Influence of Fiber Length. Board on Environmental Studies and Toxicology, National Research Council, National Academy Press, pp. 1-80.

McConnell. E.E., Rutter, H.A., Ulland, B.M., Moore, J.A. (1983) Chronic effects of dietary exposure to amosite asbestos and tremolite in F344 rats. *Environ. Health Perspect.* **53**, 27-44.

McConnell, E.E., Wagner, J.C., Skidmore, J.W. and Moore, J.A. (1984). A comparative study of the fibrogenic and carcinogenic effects of UICC Canadian chrysotile B asbestos and glass microfiber JM 100. In: Biological Effects of Man-made Mineral Fibers. World Health Organization, pp. 234-252.

McConnell, E.E., (1995). Advantages and limits of in vivo screening tests. *Ann. Occup. Hyg.* **39**, 727-735.

Meeker, G.P., Bern, A.M., Brownfield, I.K., Lowers, H.A., Sutley, S.J., Hoefen, T.M., Vance, J.S. (2003) The composition and morphology of amphiboles from the Rainy Creek Complex, near Libby, Montana. *American Mineralogist* **88**, 1955-1969.

Miller, B.G., Searl, A., Davis, J.M.G, Donaldson, K. Cullen, R.T., Bolton. R.E., Buchanan, D., Soutar, C.A., (1999a). Influence of fibre length, dissolution and biopersistence on the production of mesothelioma in the rat peritoneal cavity. *Ann. Occup. Hyg.* **43**, 155-166.

Miller, B.G., Jones, A.D., Searl, A., Buchanan, D., Cullen, R.T., Soutar, C.A., Davis, J.M.G, Donaldson, K. (1999b). Influence of characteristics of inhaled fibers on development of tumours in the rat lung. *Ann. Occup. Hyg.* **43**, 167-179.

NTP 1990. National Toxicology Program Technical Report on the carcinogenesis bioassay of tremolite in Fischer 344/N rats feed study. NTP Technical Report No. 277, National Institute of Environmental Health Sciences NIH, Research Triangle Park, NC, USA.

Oberdörster, G., Lehnert, B.E. (1991). Toxicological aspects of the pathogenesis of fiber-induced pulmonary effects. Mechanisms in Fiber Carcinogenesis, (In: Brown, R.C., Hoskins, J.A., Johnson, N.F., Eds), Plenum Press, New York, pp. 157-179.

Pott, F., Huth, F., Friedrichs, K.H. (1974). Tumorigenic effects of fibrous dusts in experimental animals. *Env. Health Perspect.* **9**, 313-315.

Pott, F., Roller, M., Ziem,.U., Reiffer, F-J., Bellmann, B., Rosenbruch, M., Huth. F. (1989). Carcinogenicity studies on natural and man-made fibers with the intraperitoneal test in rats. In: *Non-occupational Exposure to Mineral Fibers.* (Bignon, J., Peto, J., Saracci, R. Eds), IARC Scientific Publications No. 90. International Agency for Research on Cancer. Pp.173-179.

Pott, F., Roller, M., Rippe, R.M., Germann, P-G., Bellmann, B. (1991). Tumours by the intraperitoneal and intrapleural routes and their significance for the classification of mineral fibers. In: *Mechanisms in Fiber Carcinogenesis,* (Brown, R.C., Hoskins, J.A., Johnson, N.F., Eds), Plenum Press, New York, pp. 547-565.

Searl, A., Buchanan, D., Cullen, R.T., Jones, A.D., Miller, B.G., Soutar, C.A. (1999). Biopersistence and durability of nine mineral fiber types in rat lungs over 12 months. *Ann. Occup. Hyg.* **43**, 143-153.

Smith, W.E., Hubert, D.D., Sobel, H.J., Marquet, E. (1979). Biologic tests of tremolite in hamsters. In: *Dusts and Disease*, (Dement J.A., and Lemen, R.A., Eds) Pathotox Publishers, Inc., Park Forest South, Illinois, pp. 335-339.

Stanton, M.F., Layard, M., Tegeris, A., Miller, E., May, M., Morgan, E., Smith, A. (1981). Relation of particle dimension to carcinogenicity in amphibole asbestoses and other fibrous minerals. *J. Natl. Cancer Ins.* **67**, 965-975.

USEPA. (1986). Guidelines for carcinogen risk assessment. Federal Register. **51**, 33992-34003.

Veblen, D.R., Wylie, A.G. (1993) Mineralogy of Amphiboles and 1:1 layer Silicates. In: *Health Effects of Mineral Dusts. Reviews in Mineralogy,* (Guthrie, G.D., Mossman, B.T. Eds) Mineralogical Society of America, Volume 28, pp. 61-137.

Wagner, J.C., Griffiths, D.M., Munday, D.E. (1987). Experimental studies with palygorskite dusts. Br. J. Ind. Med. 44,749-63.

Wagner, J.C., Berry, G.B., Hill, R.J., Munday, D.E. and Skidmore, J.W. (1984). Animal experiments with MMMV F - Effects of inhalation and intrapleural inoculation in rats. In: Biological Effects of Man-made Mineral Fibers. World Health Organization, pp. 209-232.

Wagner, J.C., Chamberlain, M., Brown, R.C., Berry, G., Pooley, F.D., Davies, R., Griffiths, D.M. (1982). Biological effects of tremolite. *Br. J. Cancer* 45, 352-360.

WHO. (1985). Reference methods for measuring man-made mineral fibers MMMF. Prepared by WHO/EURO Technical Committee for evaluating MMMF, WHO Regional Office, Copenhagen, Denmark.

Wylie, A.G., Bailey, K.F., Kelse, J.W., Lee, R.J. (1993). The importance of width in asbestos fiber carcinogenicity and its implications for public policy. *J. Am. Ind. Hyg. Assoc.* 54-5, 239-252.

Figure 1. Typical prismatic form of amphibole showing main cleavages and prism faces.

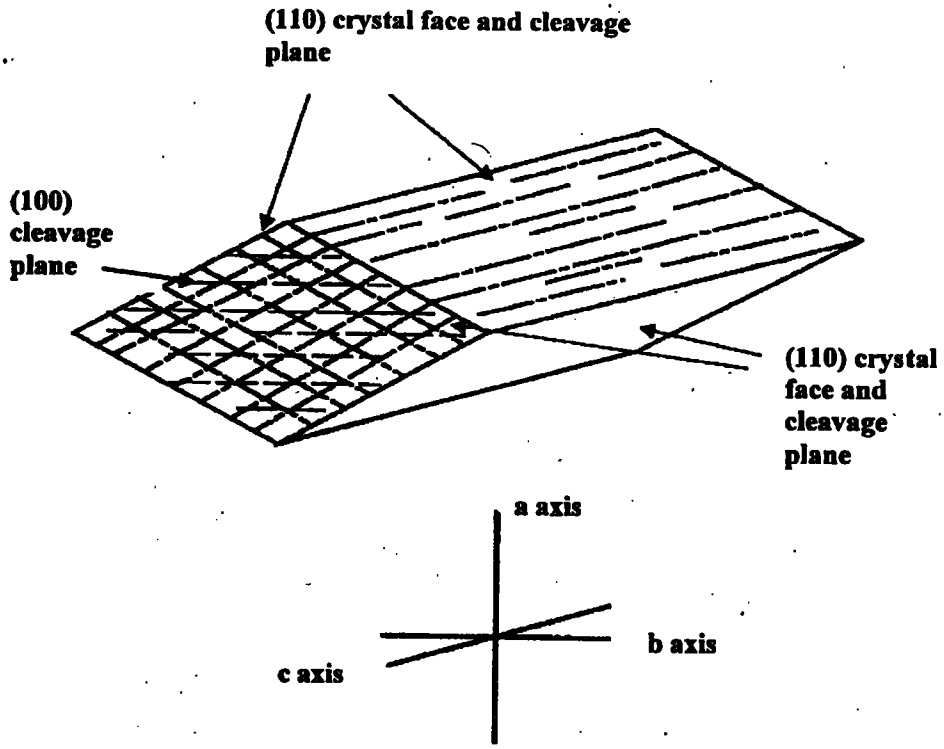


Figure 1b. Scanning Electron Microscope microphotograph of Ala di Stura tremolite showing a large prismatic crystal with cross section with a cross section shape determined by (110) crystal faces; also evident are the traces of the (100) cleavage planes. Thin asbestiform tremolite fibers with diameters finer than 1 micron are also visible on the right hand side of the image.

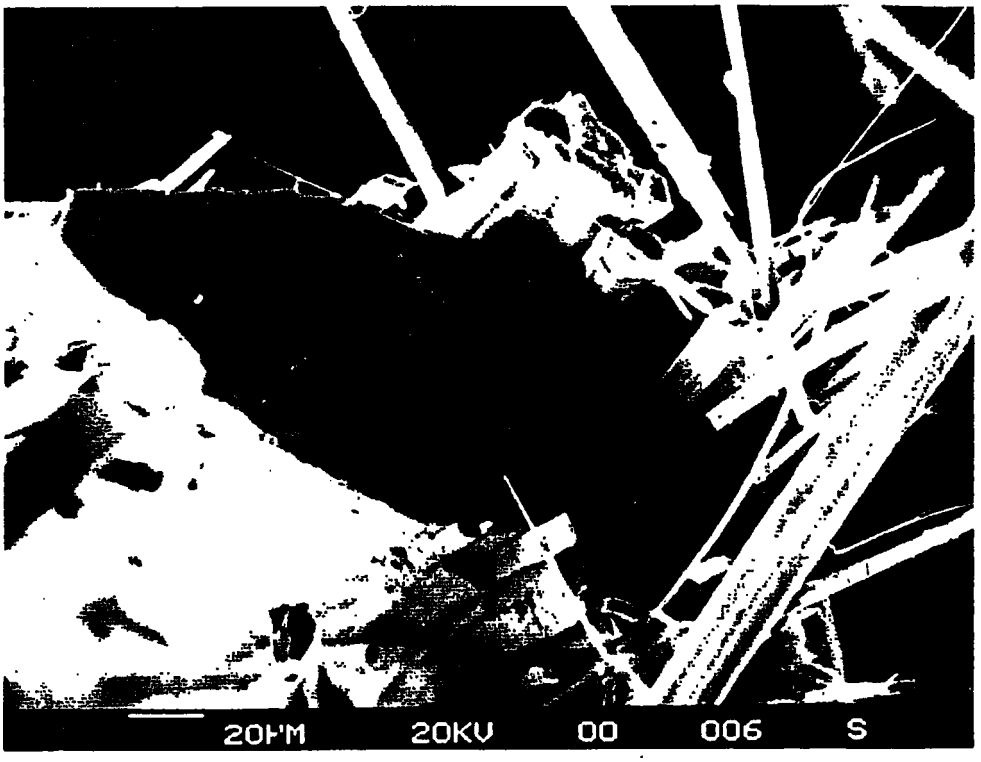


Figure 2. Length and width distribution of fibers (microns) in elutriated respirable dust of the Korean Tremolite Asbestos. This is a typical asbestos fiber size distribution with most fibers less than 5 microns in length and less than 0.5 microns in diameter. There are however some thin fibers with length greater than 10 microns, and some cleavage fragment fibers with diameters greater than 1 micron.

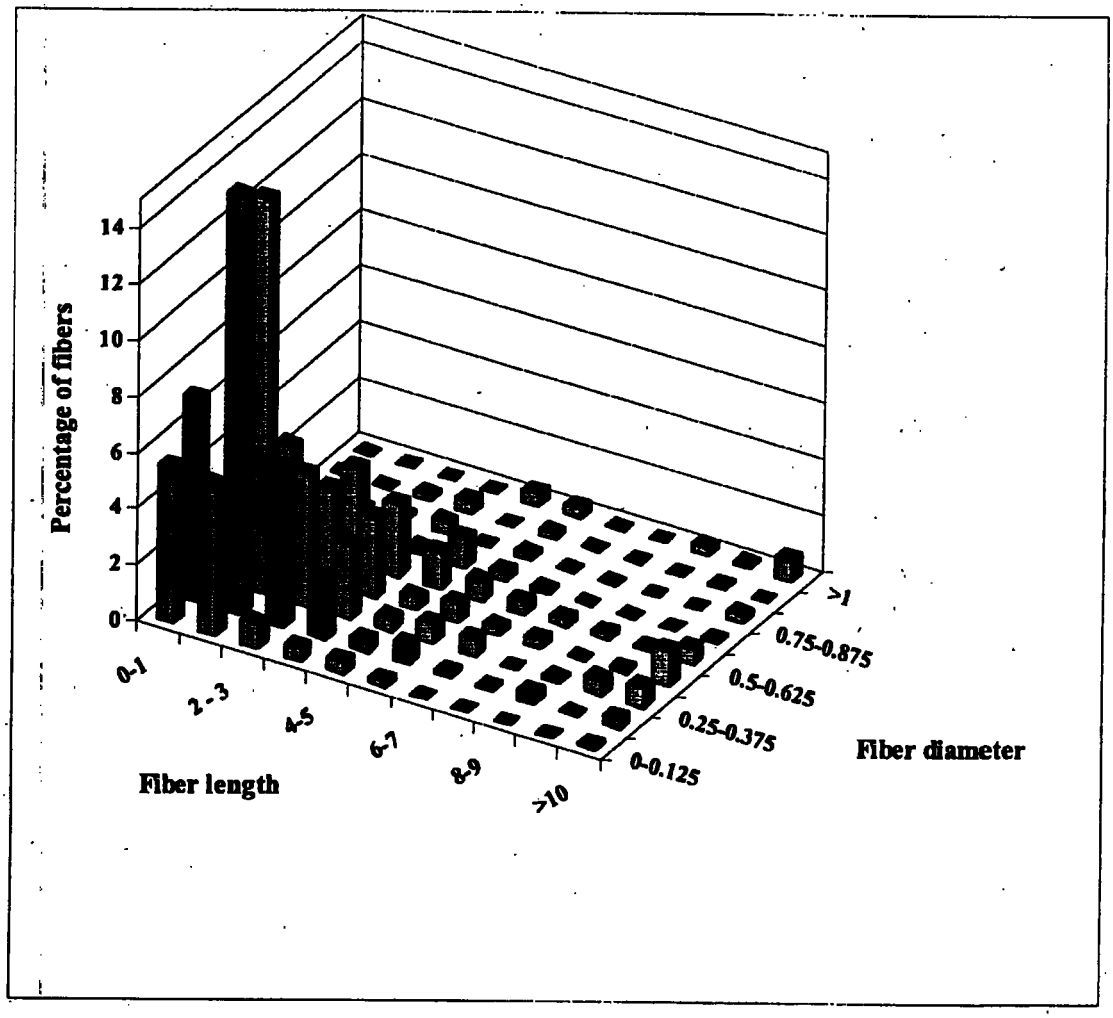
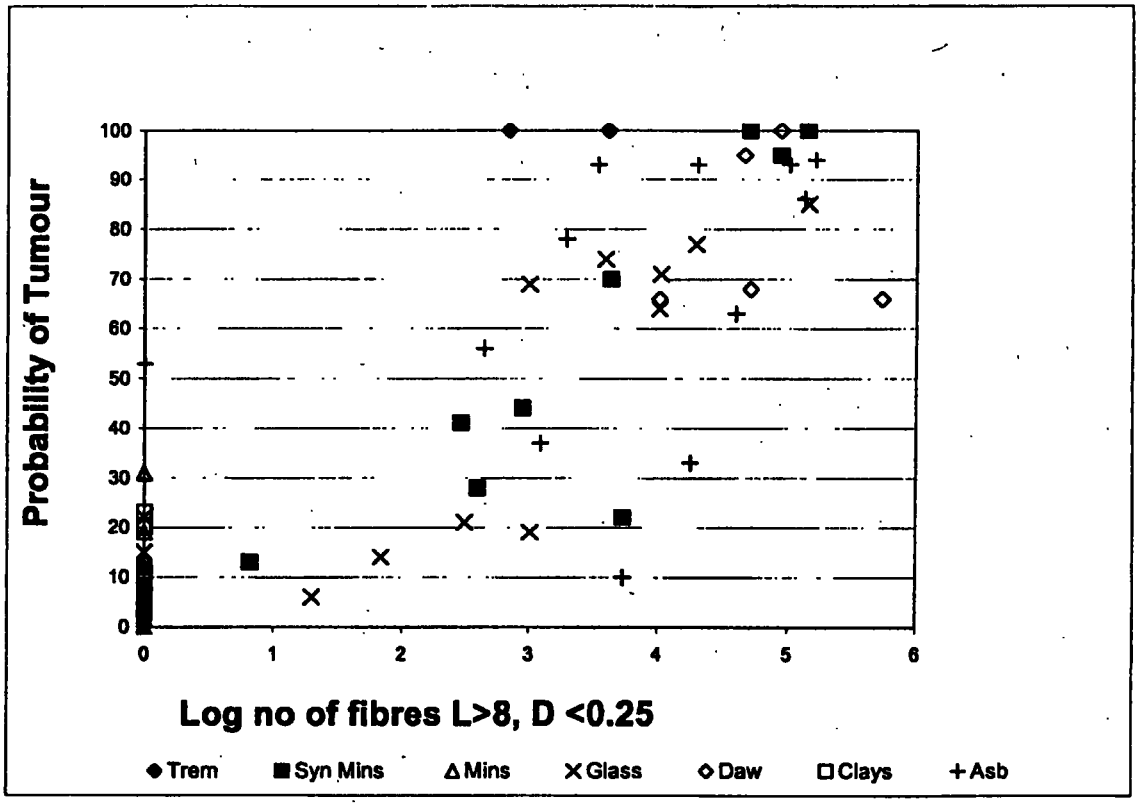


Figure 3. The probability of fibre generating a tumour compared to the log of numbers of fibre per microgram longer than 8 microns with diameter greater than or equal to 0.25 microns. This is the same data as in Stanton et al (1981).



Tremolite: Tremolite
Syn Mins: Synthetic minerals, Silicon carbide, Aluminum oxide, Potassium octatitanate
Mins: Minerals, Wollastonite, Talc
Glass: Borosilicate glass fibers
Daw: Dawsonite (synthetic)
Clays: Attapulgite, Halloysite
Asb: Asbestos (mostly crocidolite)

Figure 4. Probability of fibres generating mesothelioma compared to the numbers of fibres per microgram in dose within the size range of 4 – 8microns long with diameters in the range 0.01 – 1.5 microns. Data from Stanton et al (1981)

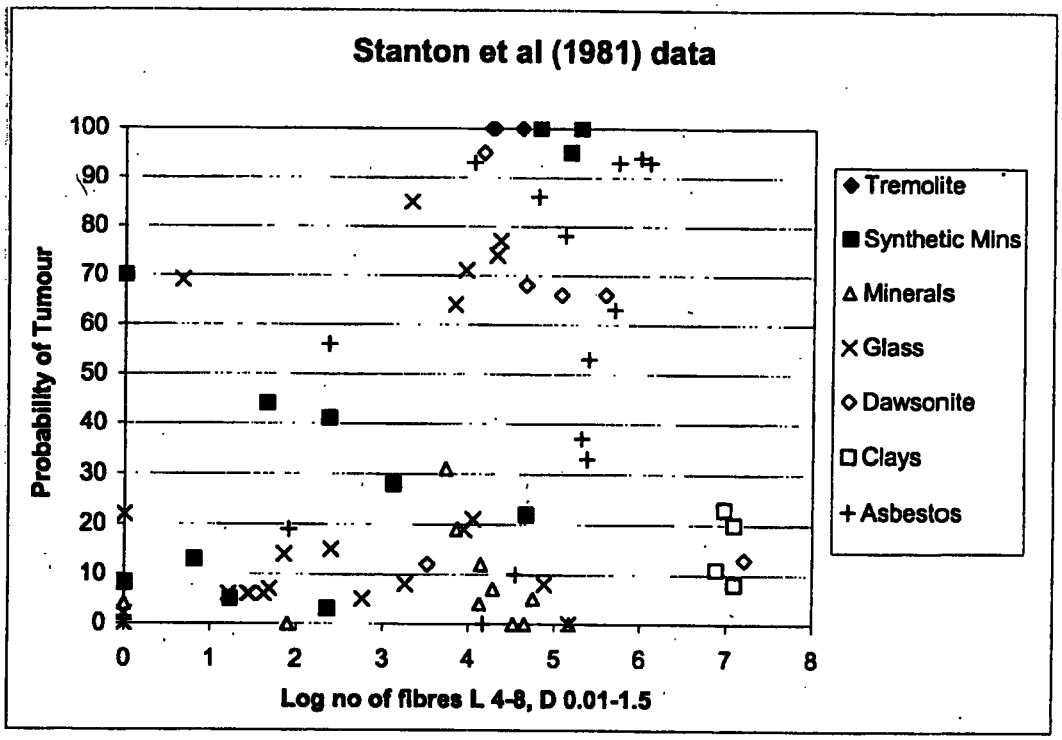


Figure 5. Length and diameter distribution of Tremolite 2 from the experiments of Stanton et al (1981) showing the bimodal distribution of the fibres. Based upon the Stanton data.

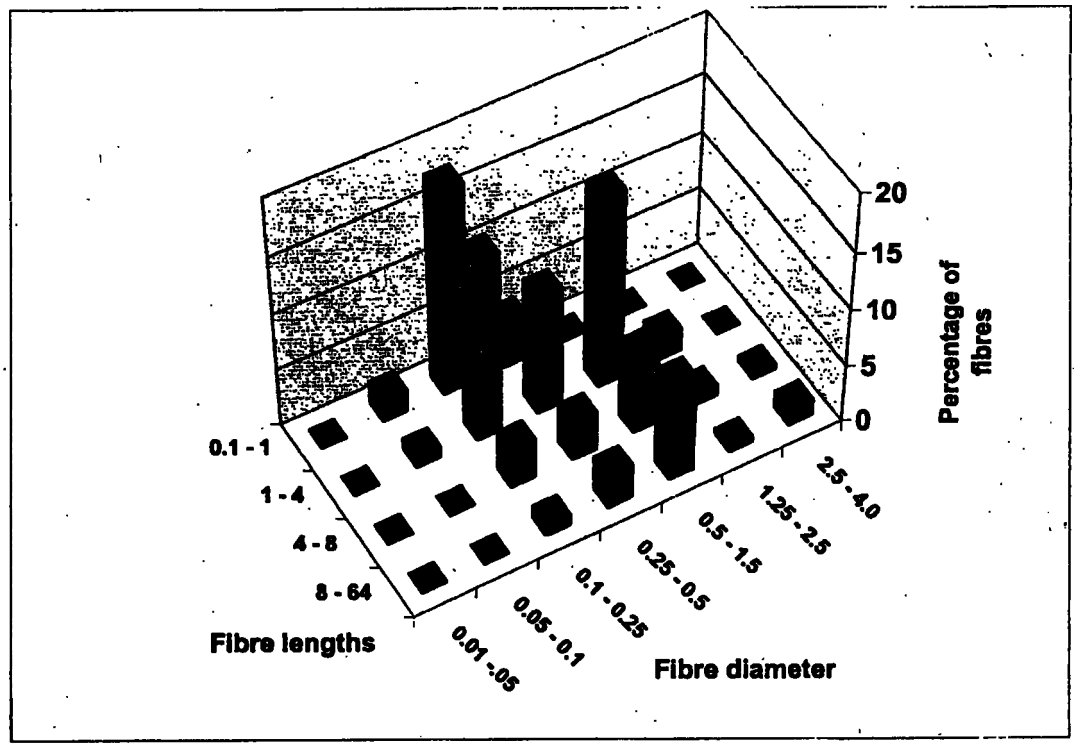


Figure 6. Size distribution of Talc 6 using the size data from Stanton et al (1981)

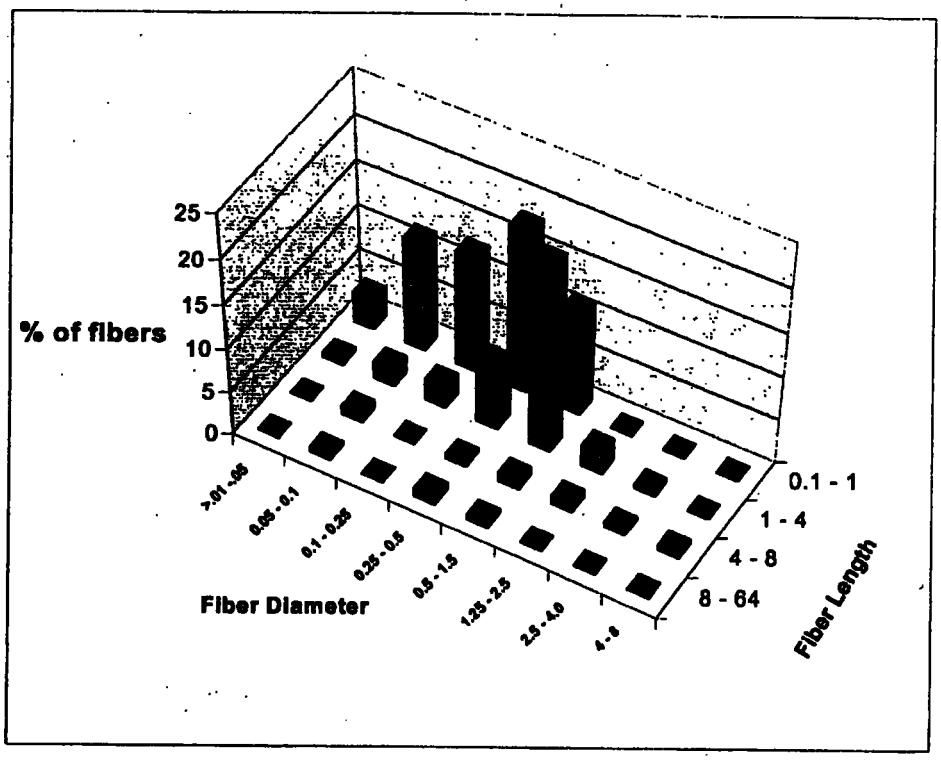


Figure 7. Numbers of 'Stanton Fibers' per microgram compared to the numbers of fibres in the size range 4 –8 microns long and 0.01 – 1.5 microns diameter showing an obvious relationship (Correlation coefficient 0.74). Fibers with no fibers in either class have been omitted. Data from Stanton et al (1981)

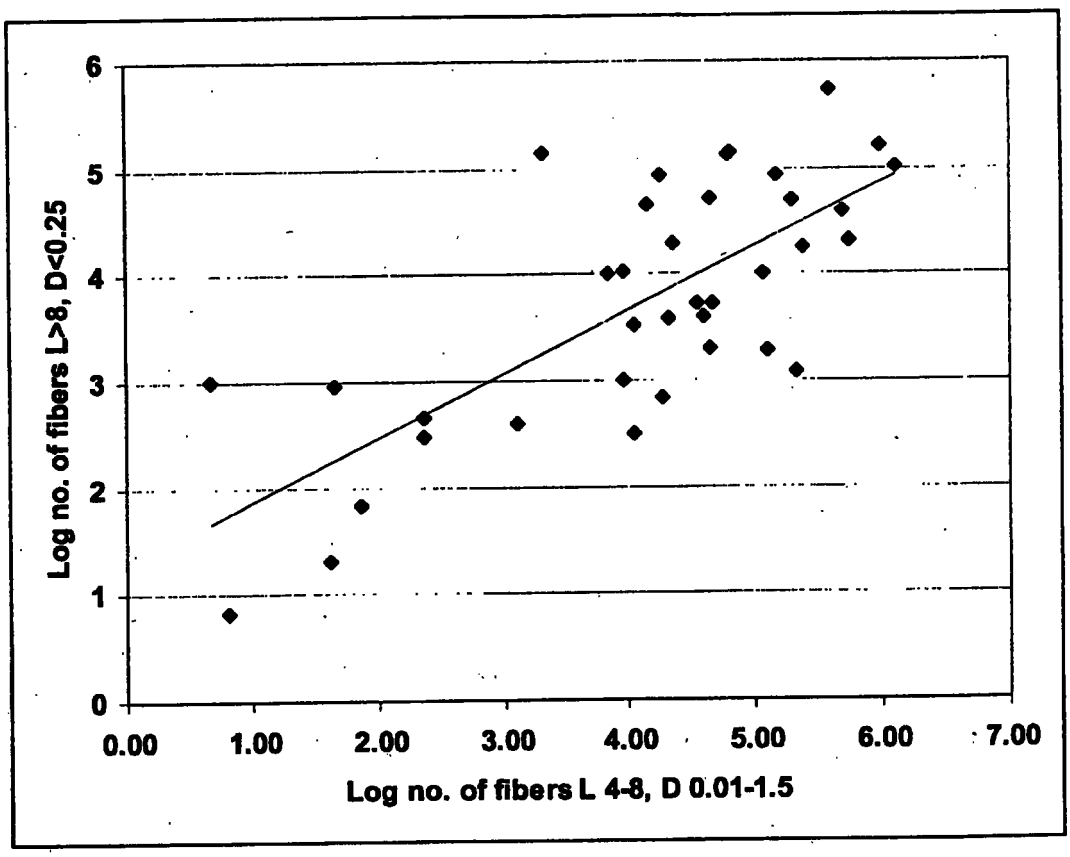


Figure 8. Probability of producing tumor vs number of fibers per microgram in the dose that were longer than 4.0 microns with diameter between 0.1 and 1.5 microns (and no fibers longer than 8 microns). Data from Stanton et al (1981). Very large numbers fibers of clay minerals and dawsonite produced relatively low response.

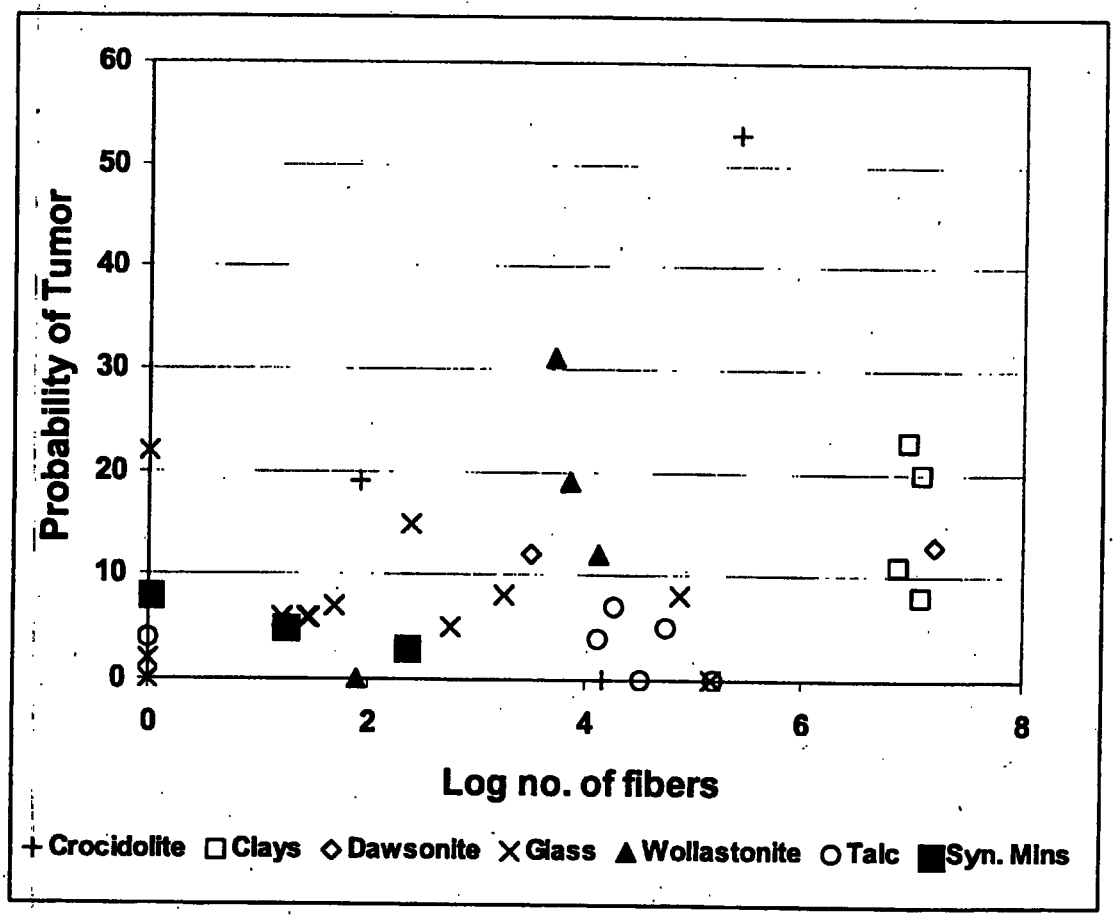


Table 1. Summary of the samples and results of the toxicological testing of Smith et al. (1979)

Sample Number	Descriptor	Tumor Incidence		Composition
		10 mg dose	25 mg dose	
14 (or FD-14)	Tremolitic talc, New York State,	-	0/35	50% non-asbestos tremolite
275	Tremolite selected from NY Tremolitic talc	0/34	0/31	95% non-asbestos tremolite
31	Tremolitic talc, unspecified location in W. USA	1/42	6/30	90% tremolite, possibly asbestiform
72 (or FD72)	Asbestiform tremolite, unspecified location	3/13	5/23	95% tremolite asbestos
72N	Asbestiform Tremolite	6/25	11/26	95% Tremolite Asbestos

Table 2.

Tremolite particles per microgram of injected dose in Wagner et al. (1982).

Sample	Non-fibrous Particles x 10^4	All fibers x 10^4	Fibers > 8 μm long and <1.5 μm wide x 10^3
A	6.9	5.1	1.7
B	20.7	4.8	0
C	3.3	15.5	56.1

Table 3.

Results of the Davis et al. (1991) intraperitoneal injection experiments with tremolites of differing morphologies.

Tremolite Source	N° of Animals	N° of Mesothelioma	Median Survival Time (days)	Relative Hazard	Millions of Fibers in Dose Injected	Millions of fibers, (length >8 µm and diameter <0.25 µm)
California Asbestos	36	36	301	346,939	1,3430	121
Swansea Asbestos	36	35	365	183,673	2,104	8
Korea Asbestos	33	32	428	51,020	7,791	48
Italy Non-Asbestos	36	24	755	1,020	1,293	1
Dornie Non-Asbestos	33	4	§	6.4	899	0
Shinness Non-Asbestos	36	2	§	1	383	0

§ Insufficient animal death for calculation

Table 4.
Results from Davis et al. (1986) inhalation and intraperitoneal injection experiments with long and short fiber grunerite (amosite) asbestos.

Injection Experiments	Long Grunerite (Amosite) Asbestos		Short Grunerite Amosite (Amosite)	
Dose	10 mg	25 mg	10 mg	25 mg
Number (%) of Animals with Mesothelioma	21 (88%)	20 (95%)	0	1(4%)
Mean Tumor Induction Period	535	520	N/A	837
Millions of Fibers in Dose Length ≥5 µm	1731	4327	60.3	150.75
Millions of Fibers in Dose Length ≥10 µm	932	2330	10.34	25.85

Inhalation Experiment	Long Grunerite (Amosite) Asbestos	Short Grunerite Amosite (Amosite)
Number (%) of Animals with Mesothelioma	14(35%)	1(2.4%)
Lung contents (mg) Immediately after dusting (Std Dev)	3.57 mg (1.59)	5.64 mg (0.37)
6 months clearance (Std Dev)	3.08 mg (0.37)	4.47 mg (0.58)

10/31/05

Assessment of the Pathogenic Potential of Asbestiform vs. Nonasbestiform Particulates (Cleavage Fragments) in *In Vitro* (Cell or Organ Culture) Models and Bioassays

Brooke T. Mossman, Ph.D.
Professor of Pathology
University of Vermont College of Medicine,
Burlington, VT 05405

Peer Reviewed and Accepted for Publication in Proceedings of the *International Symposium on the Health Hazard Evaluation of Fibrous Particles Associated with Taconite and the Adjacent Duluth Complex*
St Paul, Minnesota
March 30 – April 1, 2003

ABSTRACT

Asbestos fibers are highly fibrous silicate fibers that are distinguished by having a large aspect (length to diameter) ratio and are crystallized in an asbestiform habit that causes them to separate into very thin fibers or fibrils. These fibers are distinct from nonasbestiform cleavage fragments this may appear as thick, short fibers which break along cleavage planes without the high strength and flexibility of asbestiform fibers. Because cleavage fragments of respirable dimensions have generally proven nonpathogenic in animal studies, little data exists on assessing well-characterized preparations of cleavage fragments in *in vitro* models. The available studies show that cleavage fragments are less bioreactive and cytotoxic than asbestiform fibers.

INTRODUCTION

'Asbestos' is a commercial and regulatory designation for a family of naturally occurring asbestiform fibers. Asbestos fibers are recognized as human carcinogens and also cause pleural and pulmonary fibrosis, i.e., asbestosis in occupationally exposed individuals (Mossman *et al.*, 1990; Mossman & Churg, 1998; Mossman & Gee, 1989). Mineralogical and biological differences exist between various types of asbestos fibers, and much research has focused on the characteristics of fibers that are associated with the causation of lung disease. The different types of asbestos include chrysotile [$Mg_6 Si_4 O_{10} (OH)_8$] , the only asbestos in the serpentine family of minerals, and other types of asbestos classified as amphiboles. These include crocidolite [$(Na_2 (Fe^{3+})_2(Fe^{2+})_3 Si_8 O_{22} (OH)_2$], asbestiform grunerite or amosite [$(Fe,Mg)_7 Si_8 O_{22} (OH)_2$] , anthophyllite [$(Mg,Fe)_7 Si_8 O_{22} (OH)_2$], tremolite [$Ca_2 Mg_5 Si_8 O_{22} (OH)_2$], and actinolite [$(Ca_2 (Mg,Fe)_5 Si_8 O_{22} (OH)_2$]. These formulae are indeed ideal, and natural amphiboles differ to varying degrees from these as the chemical environment, pressure and temperature at the time of formation control the mineral chemistry. Other factors such as shear stresses and directed pressures determine whether or not an amphibole that crystallizes is asbestiform. Although various types of asbestos are different chemically, structurally and biologically, they are common in that they are highly fibrous silicate minerals that are crystallized in an asbestiform habit, causing them to separate into thin fibers or fibrils (Klein, 1993; Veblen & Wylie, 1993). In addition, asbestos fibers are distinguished by having large aspect (length to diameter) ratios, generally from 20:1 or higher for fibers > 5 microns in length. Smaller fibers (<0.5 microns in width) appear by microscopy as very thin fibrils as defined by the American Society of Testing Materials in 1990. In contrast, nonasbestiform cleavage fragments, although sometimes elongated with aspect ratios of >3:1 which can be defined as fibers, have widths much larger than asbestos fibers of the same length. Though the more common nonasbestiform analogs of asbestos share the same, or essentially the same chemical composition, they do not share the same crystal structure (the crystals form or grow differently).

Cleavage fragments of amphiboles lack the tensile strength of asbestos amphiboles and are traditionally regarded by mineral scientists as distinctly different from asbestos fibers, primarily based on their morphology, and lack of strength or flexibility. For example, in the report of the Committee on Nonoccupational Health Risks of Asbestiform Fibers commissioned by the National Research Council (National Research Council, 1984), cleavage fragments were categorized as distinctive from asbestiform fibers, i.e.:

"CLEAVAGE refers to the preferential breakage of crystals along certain planes of structural weakness. Such planes of weakness are called cleavage planes. A mineral with two distinct cleavage planes will preferentially fracture along these planes and will produce ACICULAR fragments. Minerals with one cleavage plane produce PLATY fragments and those with three or more cleavage planes yield POLYHEDRAL fragments..... Cleavage cannot produce the high strength and flexibility of asbestiform fibers" (National Research Council, 1984).

These definitions were also recognized by the members of the panel of the Health Effects Research-Asbestos Research in their report on Asbestos in Public and Commercial Buildings (Health Effects Institute-Asbestos Research, 1991). Because epidemiologic and animal studies have not suggested that nonasbestiform amphiboles or cleavage fragments are pathogenic or biologically active, they have not been used in many *in vitro* models, except as negative or nonpathogenic controls for testing of asbestos fibers. Moreover, the results of numerous epidemiologic, animal, and *in vitro* studies, have led scientists to conclude that short asbestos fibers (< 5 microns in length) are inactive or much less active biologically than long, thin asbestos fibers (ATSDR, 2003; Health Effects Institute-Asbestos Research, 1991). Thus, it is unlikely that cleavage fragments of respirable dimensions (i.e., less than 3 microns in diameter) will be pathogenic or targeted extensively for *in vitro* fiber testing in the future. The results of limited work with these minerals from our laboratory and others are summarized below.

Advantages and Caveats of *In Vitro* Mineral Studies

In vitro studies have been used historically to compare the effects of different types of minerals on cells or organ (explant) cultures (Mossman & Begin, 1989). Regardless of cell type, asbestos fibers, in comparison to a variety of other nonpathogenic, synthetic or naturally occurring fibers (glass, cellulose, etc.) or particles, have been most biologically active in these models. In addition to elucidating the properties of minerals (size, fibrous morphology, surface charge, chemical composition, etc.) that are associated with toxicity (cell injury or death), DNA damage, proliferation and/or alterations in cell function that may be predictive of their pathogenic potential, *in vitro* studies have shed light on the complex features of bioreactive minerals that may be important in reactions with cells and their ability to cause disease. Cell and organ culture models are also much more inexpensive than animal testing. Thus, they have been suggested as screening tools for new synthetic fibers developed for industry.

However, there are also caveats that must be recognized in *in vitro* work with minerals. First, dependent upon the cells used in these models, cell type and species-specific responses may exist. Thus results from lab to lab working with the same mineral might be inconsistent. Although the most appropriate *in vitro* cell types to use in these models are normal cells of respiratory tract origin, i.e., epithelial or mesothelial, these are notoriously difficult to isolate and maintain in a differentiated state for prolonged periods of time. It also should be acknowledged that concentrations of minerals used in short term *in vitro* assays, where weighed amounts of fibers or particles are precipitated on cells, do not mimic normal clearance patterns and long-term dissolution patterns after inhalation into the human lung, factors that are important in dosimetry and disease causation (Mossman *et al.*, 1990). Lastly, different minerals are generally evaluated in *in vitro* studies on an equal weight basis, which might be misleading based on the

facts that different weights of dissimilar fiber types or particles may reflect vastly different total numbers of fibers and surface areas. Regardless of these caveats, however, *in vitro* studies have helped to establish mechanisms of fiber carcinogenesis and differentiated between responses to asbestos fibers and nonasbestiform particles.

Studies Using Tracheal Explants

In comparison to cell cultures, tracheal explant cultures can be maintained for weeks in a differentiated state in which the respiratory epithelium is maintained in a normal, mucociliary phenotype. We have used this model to show that crocidolite and chrysotile fibers (asbestos) and long glass fibers cause squamous metaplasia, a reversible but often premalignant lesion, and increased DNA synthesis, a signature of injury and proliferation of fibers that might be important in tumor promotion and progression and/or repair (Woodworth *et al.* 1983). In contrast, the non-fibrous mineral analogs of these asbestos types, riebeckite (similar in chemistry to crocidolite) and antigorite (similar in chemistry to chrysotile) failed to induce these changes at a range of concentrations and exposure times. Though a number of these riebeckite and antigorite particles were elongated, they were thick, short single crystal cleavage fragments. These studies highlight the importance of fibrous geometry, crystal growth and aspect ratio in bioreactivity.

Studies Using Cell Types of Lung or Pleural Origin:

The antigorite and riebeckite preparations used in the Woodworth et al. 1983 study above were also evaluated in cell cultures of hamster tracheal epithelial cells (HTE) for their ability to induce ornithine decarboxylase (ODC), an enzyme associated with cell proliferation and tumor promotion in mouse skin models of cancer, with asbestos fibers (Marsh & Mossman, 1988). These studies showed that crocidolite and chrysotile (fibers> 10 microns in length) fibers stimulated ODC, but neither of the two nonasbestiform (cleavage fragment) preparations were bioreactive. Subsequent studies revealed that both antigorite and riebeckite were less potent than crocidolite (asbestos) in stimulating survival or proliferation of HTE cells in a colony-forming assay (CFE) in which proliferation was measured directly over a 7 day period in low-serum containing medium (Sesko & Mossman, 1989). Experiments in HTE cells also revealed that antigorite and riebeckite were less cytotoxic than crocidolite or chrysotile to these cells when release of radioactive chromium, a marker of cell damage, was measured (Mossman & Sesko, 1990).

Another exciting development in our laboratory was the observation that crocidolite (asbestos) generated Reactive Oxygen Species (ROS) which have been linked to cell injury, inflammation, mutagenesis, and the development of many cancers, (Shukla *et al.* 2003). In a study in which we isolated alveolar macrophages (AMs) from rodents and measured release of the ROS, superoxide, after addition of crocidolite and riebeckite (nonasbestiform analog of crocidolite) to these cells, as well as nonasbestiform mordenite (note that all particle diameters and/or fiber lengths were measured by scanning electron microscopy), the nonasbestiform particles were taken up, i.e., phagocytized, by cells, but were much less bioreactive than crocidolite at comparable concentrations, only causing release of superoxide at concentrations 5- to 10-fold

higher than asbestos in the rat cells and never causing significantly increased release in the hamster macrophages (Hansen & Mossman 1987). It should be emphasized that lung epithelial cells, mesothelial cells and fibroblasts are target or progenitor cells of lung cancers, mesotheliomas and pulmonary fibrosis, respectively, and that alveolar macrophages are inflammatory cells that first encounter asbestos and may contribute to and/or alternatively, be important in lung defense from pathogenic minerals. This is an important question that has yet to be resolved by scientists. However, alveolar macrophages are studied because these cells accumulate in the lung at sites of deposition of inhaled particles or fibers and responses of alveolar macrophages to dusts are known to produce ROS after phagocytosis of minerals.

In recent years, we have used riebeckite and antigorite preparations as nonasbestiform control minerals to determine whether early response proto-oncogene (*fos/jun* cancer- causing genes) (Janssen *et al.* 1994) or signaling pathways leading to activation of these genes (Janssen *et al.* 1997; Zanella *et al.* 1996; Zanella *et al.* 1999) are selectively induced by asbestiform, cancer-causing fibers (crocidolite and chrysotile asbestos, erionite) in HTE cells, rat lung epithelial cells (RLE) and isolates of normal rat pleural mesothelial cells (RPM). These studies have consistently revealed that these nonasbestiform minerals are inactive, regardless of endpoint. Moreover, they are incapable, in contrast to asbestos fibers, of causing alterations in cell proliferation or death in RPM cells (Goldberg *et al.* 1997).

Comparative studies in HTE and RPM cells with well-characterized mineral samples of crocidolite and chrysotile (asbestos) and 3 mineral samples containing various proportions of fibrous talc have also been useful in illustrating fundamental differences in response to asbestos fibers and fibrous talc preparations based on various dose parameters including equal weight concentrations, equivalent surface areas and numbers of fibers > 5 microns in length (Wylie *et al.* 1997). Using the CFE assay described above to document proliferative potential (increased numbers of colonies as compared to untreated control cells) or cytotoxicity (decreased numbers of colonies as compared to untreated control cells), exposure of RPM cells to both asbestos types, but not fibrous talcs, elicited cytotoxicity in RPM cells that was more striking at higher weight concentrations of asbestos. In contrast, HTE cells proliferated in response to asbestos at nontoxic lower concentrations, but not to fibrous talcs. Since cell responses could not be correlated directly with the presence of mineral fibers > 5 microns in length or aspect ratios, mineral type rather than fiber length *per se* appeared to be a more important determinant of bioreactivity. This study suggests that while fiber morphology is important, it is not the only factor important in biologic responses. This has also been noted by critics of Stanton's famous pleural implantation studies in rats (Oehlert, 1991) (Wylie *et al.* 1987).

Studies Using *In Vitro* Models of Non-Respiratory Cells:

As detailed above, cytotoxicity testing in cells of non-respiratory origin was used decades ago to determine differences in fiber-cell interactions and the ability of asbestos fibers to induce cell death or lysis. Since dead cells can not give rise to cancers, the extrapolation of these results, especially to mechanisms of cancer causation, is questionable. However, studies by Palekar and colleagues (Palekar *et al.* 1979) used sheep red blood cells (RBC) and Chinese Hamster Ovary (CHO) cells to test the hemolytic potential and cytotoxicity of 4 samples of cummingtonite-grunerite including amosite asbestos fibers, and 3 other samples of various crystallization habits,

predominantly asbestiform cummingtonite, acicular cummingtonite ,and acicular grunerite. At the same surface areas of dose, these minerals were found to be hemolytic and cytotoxic in this same order, again showing the increased potency of amphibole asbestiform fibers.

SUMMARY AND CONCLUSIONS

The results summarized above represent a large body of work showing that nonasbestiform minerals are less potent than asbestos fibers in a number of *in vitro* bioassays. In most assays, these cleavage fragments or non-fibrous minerals are virtually inactive. These observations have been incorporated into the conclusions of several panel reports that should be recognized by regulatory agencies. For example, the HEI-Asbestos Research Panel (page 6-75, 1991) concluded:

"Good evidence exists that thick fibers (>2 to 3 microns in diameter) are less harmful than thin fibers".

"Support for the importance of fiber length in the production of biological effects has been obtained from the use of non-fibrous analogues of asbestos and other fibers. In general, these materials produce no detectable biological effects, or do so only at high dose levels"

ACKNOWLEDGEMENTS

Research in Dr. Mossman's laboratory has been supported by federal agencies (EPA, NIOSH, NIEHS, NCI, and NHLBI) for over 20 years. Ms. Laurie Sabens was invaluable in the preparation of this manuscript.

References

ATSDR (2003). Report of the Expert Panel on Human Effects of Asbestos and Synthetic Vitreous Fibers: The Influence of Fiber Length, Agency for Toxic Substances and Disease Registry (ATSDR), Division of Health Assessment and Consultation, Atlanta, GA.

Goldberg J., Zanella C., Janssen Y., Timblin C., Jimenez L., Taatjes D., and Mossman B. (1997). Novel cell imaging approaches show induction of apoptosis and proliferation in mesothelial cells by asbestos. *Am. J. Respir. Cell Mol. Biol.* 17, 265-271.

Hansen K., and Mossman B. (1987). Generation of superoxide (O2-.) from alveolar macrophages exposed to asbestiform and nonfibrous particles. *Cancer Res.* 47, 1681-6.

Health Effects Institute-Asbestos Research (1991). Asbestos in Public and Commercial Buildings: A literature reviewed synthesis of current knowledge. Health Effects Institute, Cambridge, MA.

Janssen Y., Driscoll K., Howard B., Quinlan T., Treadwell M., Barchowsky A., and Mossman B. (1997). Asbestos causes translocation of p65 protein and increases NF-kappa B DNA binding activity in rat lung epithelial and pleural mesothelial cells. *Am. J. Pathol.* 151, 389-401.

Janssen Y., Heintz N., Marsh J., Borm P., and Mossman B. (1994). Induction of c-fos and c-jun proto-oncogenes in target cells of the lung and pleura by carcinogenic fibers. *Am. J. Respir. Cell Mol. Biol.* 11, 522-530.

Klein C. (1993). Rocks, minerals and a dusty world. *In* "Health effects of mineral dusts." (G. D. Guthrie, and B. Mossman, Eds.), pp. 7-59, Washington, DC.

Marsh J. P., and Mossman B. T. (1988). Mechanisms of induction of ornithine decarboxylase activity in tracheal epithelial cells by asbestiform minerals. *Cancer Res* 48, 709-14.

Mossman B., Bignon J., Corn M., Seaton A., and Gee J. (1990). Asbestos: Scientific developments and implications for public policy. *Science* 247, 294-301.

Mossman B., and Churg A. (1998). State-of-the-Art: Mechanisms in the pathogenesis of asbestosis and silicosis. *Am. J. Respir. Crit. Care Med.* 157, 1666-1680.

Mossman B., and Gee JBL. (1989). Asbestos related disease. *N. Engl. J. Med.* 320, 1721-1730.

Mossman B., and Sesko A. (1990). In vitro assays to predict the pathogenicity of mineral fibers. *Toxicology* 60, 53-61.

Mossman B. T., and Begin R. (1989). "Effects of Mineral Dusts on Cells, NATO ASI Series on Cell Biology, Springer-Verlag, Berlin, 1989.," Springer-Verlag, Berlin.

National Research Council (1984). Asbestiform fibers: Nonoccupational health risks, National Academy Press, Washington, D.C.

Oehlert G. W. (1991). A reanalysis of the Stanton et al. pleural sarcoma data. *Environ Res* 54, 194-205.

Palekar L. D., Spooner C. M., and Coffin D. L. (1979). Influence of crystallization habit of minerals on in vitro cytotoxicity. *Ann N Y Acad Sci* 330, 673-686.

Sesko A., and Mossman B. (1989). Sensitivity of hamster tracheal epithelial cells to asbestiform minerals modulated by serum and by transforming growth factor beta 1. *Cancer Res.* 49, 2743-2749.

Shukla A., Gulumian M., Hei T., Kamp D., Rahman Q., and Mossman B. (2003). Multiple roles of oxidants in the pathogenesis of asbestos-induced diseases. *Free Rad. Biol. Med.* 34, 1117-1129.

Veblen D. R., and Wylie A. G. (1993). Mineralogy of amphiboles and 1:1 layer silicates. *In* "Health Effects of Mineral Dusts." (G. D. Guthrie, and B. Mossman, Eds.), pp. 61-137, Washington, DC.

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

Wylie A., Skinner H., Marsh J., Snyder H., Garzione C., Hodkinson D., Winters R., and Mossman B. (1997). Mineralogical features associated with cytotoxic and proliferative effects of fibrous talc and asbestos on rodent tracheal epithelial and pleural mesothelial cells. *Toxicol. Appl. Pharmacol.* 147, 143-150.

Wylie A. G., Virta R. L., and Segreti J. M. (1987). Characterization of mineral population by index particle: implication for the Stanton hypothesis. *Environ Res* 43, 427-39.

Zanella C., Posada J., Tritton T., and Mossman B. (1996). Asbestos causes stimulation of the ERK-1 mitogen-activated protein kinase cascade after phosphorylation of the epidermal growth factor receptor. *Cancer Res.* 56, 5334-5338.

Zanella C., Timblin C., Cummins A., Jung M., Goldberg J., Raabe R., Tritton T., and Mossman B.T. (1999). Asbestos-induced phosphorylation of epidermal growth factor receptor is linked to c-fos expression and apoptosis. *Am. J. Physiol. (Lung Cell Mol Physiol)* 277, L684-L693.

THE ASBESTIFORM AND PRISMATIC MINERAL GROWTH HABIT AND THEIR RELATIONSHIP TO CANCER STUDIES

A PICTORIAL PRESENTATION

March, 2004

The Asbestiform and Prismatic Mineral Growth Habit and Their Relationship to Cancer Studies

Kelly F. Bailey, CIH
Manager, Occupational Health
Vulcan Materials Company
Birmingham, Alabama

John Kelse
Corporate Industrial Hygienist
Manager, Risk Management Dept.
R. T. Vanderbilt Company
Norwalk, Connecticut

Ann G. Wylie, PhD
Asst. President and Chief of Staff
Professor of Geology
University of Maryland
College Park, Maryland

Richard J. Lee, PhD
President
R. J. Lee Group, Inc.
Monroeville, Pennsylvania

The recognition and regulation of asbestiform and prismatic minerals is of critical concern to the entire mining and aggregates industry, to individuals exposed to these materials and to the economic vitality of the United States.

CONTENTS

INTRODUCTION 1
WHY IS THIS DISTINCTION IMPORTANT? 2

REFERENCE EXHIBITS
1. What is Asbestos? 4
2. Macroscopic Raw Ore Comparisons 8
3. Light Microscopic Comparisons 10
4. The Aspect Ratio and Particle Width 12
5. Byssolite 16

EXPOSURE EXHIBITS

ASBESTOS EXPOSURES
A. Libby Montana Vermiculite 18
B. Greek Tremolite 20
C. Korean Tremolite 22
D. Addison/Davis - Tremolite (Jamestown) 24
E. Addison/Davis - Tremolite (Swansea) 26
F. Smith - Tremolite FD-72 28
G. Stanton - Tremolite 1 and 2 30

ASBESTIFORM AND/OR HIGHLY FIBROUS
H. Cook/Coffin - Ferroactinolite 32
I. Smith - Tremolite FD-31 34
J. Addison/Davis - Tremolite (Italy) 36

COMMON PRISMATIC EXPOSURES
K. Homestake Gold Mine 38
L. East Mesabi Range - Taconite 40
M. N.Y. State Tremolitic Talc 42
N. Smith - Tremolite FD-275-1 and McConnell - Tremolite 275 46
O. Wagner - Tremolite (Greenland) 48
P. Addison/Davis - Tremolite (Dornie) 50
Q. Addison/Davis - Tremolite (Shinness) 52
R. Pott - Actinolite 54

SUMMARY 56

CONCLUSION 58

REFERENCES 59

APPENDIX I - Asbestiform Definition Contributors and Supporters 64

APPENDIX II - Analytical Issues 65

JOHN ADDISON - Consultancy 196 New Village Road, Cottingham, Hull, HU16 4NL
Asbestos, Analytical Training, Laboratory Quality Control, Expert Witness, Contaminated Land, Industrial Minerals

JOHN ADDISON, BSc F.Min Soc.

CURRICULUM VITAE
January 12, 2000

John Addison Consultancy Tel. and Facsimile 44 (0) 1482 840476
In Association with D I.Thomas Associates, Consulting Scientists and Environmental Technologists
addiscon@globalnet.co.uk

CURRICULUM VITAE JOHN ADDISON

SUMMARY OF PERSONAL DETAILS

ADDRESS	**196 New Village Road** **COTTINGHAM** **HU16 4NL**	**TELEPHONE / FAX 44 (0)14882 840 476** **e-mail addiscon@globalnet .co.uk**
DATE OF BIRTH	**23/4/46**	**PROFESSION/ MEMBERSHIP OF PROFESSIONAL SOCIETIES**
MARITAL STATUS	**MARRIED, NO CHILDREN**	BSc Bachelor of Science Member of the British Occupational Hygiene Society
NATIONALITY	**BRITISH**	Member of the Mineralogical Society Member of the Glasgow Geological Society

QUALIFICATIONS

1964 to 1969	**BSc in Pure Science with Upper 2nd Class Honours in Geology** **The University of Glasgow, Glasgow.**
1995	**Qualified Quality Assessor for United Kingdom Accreditation Service**

PROFESSIONAL EXPERIENCE

John Addison graduated with an honours degree in geology from the University of Glasgow in 1969. He carried out research in geochemistry there before becoming the Research Assistant in Geology at the Hunterian Museum with responsibility for mineralogy and petrology. He was employed as the mineralogist at the Institute of Occupational Medicine (IOM) in Edinburgh in 1978 and was later made Head of the Mineralogy Section in the Chemistry and Mineralogy Group. While at the IOM he was responsible for the management of a team of nine scientists and technicians working on the identification and quantification of chemicals and minerals in airborne dusts and bulk samples using a wide range of analytical techniques. He was responsible for the acquisition, running and maintenance of the equipment of the section including a Hitachi H7000 Scanning Transmission Electron Microscope with Energy Dispersive X-ray facility, two Scanning Electron Microscopes with EDX (Cambridge Instruments S250 MkII, Hitachi S720), a Philips X-ray Powder Diffractometer, a Philips Fourier Transform Infrared Spectrophotometer, atomic absorption spectrophotometer, and a range of other optical microscopes as well as image processing equipment. His research work involved extensive studies of coal-mine dusts, asbestos minerals, toxicology of asbestos and other minerals, analysis of asbestos in autopsy cases and oil-shale studies. He has successfully led or been involved with a wide range of research projects for national and international clients including the Department of the Environment, the Health and Safety Executive, the EEC Health and Safety in Mines Program and the US Department of Energy.

He set up in partnership as a scientific consultant in asbestos and general mineralogy, occupational hygiene, and health and safety in June 1993 that successfully operated in Edinburgh until June 1995 when by mutual agreement the partnership was terminated. Since then he has operated as an independent scientific consultant based in Edinburgh, and recently moved to Cottingham for personal reasons from where he continues to work.

Upon setting up the partnership he became involved in the issues associated with asbestos in parenteral medicines. Having been brought in as a consultant by F. Hoffmann-La Roche in Basle, he was a co-author of the Clinical Expert Report, a review document on the health effects of injected asbestos submitted to the Bundesgesundheitsamt (BGA) by Roche, and now published in Regulatory Toxicology and Pharmacology. He was also co-author of the Technical

Experts Report on the analytical methods proposed for the determination of asbestos in parenteral medicines also prepared for Roche and submitted to the BGA in 1993. He helped to organise the conference, held by the Verband der Chemischen Industrie in Frankfurt in May 1994, and drafted the technical method for the determination of asbestos in parenteral medicines by Transmission Electron Microscopy that was endorsed by the conference for general use in the pharmaceutical industry.

He is a member of Working Group 2 of the H&SE Committee on Fibre Measurement and made major contributions to the development of the UK Reference Method for the identification of asbestos (MDHS 77), new methods for fibre discrimination in light microscope fibre counting (MDHS87), methods for the quantitative analysis of asbestos in contaminated land (MDHS 90), methods for the survey and sampling of asbestos in buildings (MDHS 100) and the development of the syllabus of a national training course for asbestos surveyors and analysts with WG2. He was active in establishing the pilot scheme for proficiency testing of laboratories involved with asbestos identification in bulk materials and is a member of the WG1, the oversight committee charged with the task of administering the proficiency testing scheme for asbestos and other fibre counting, and bulk asbestos analysis in the United Kingdom. He was directly responsible for establishing the UK Reference Asbestos Minerals that are used now in almost every UK asbestos laboratory and are gaining wide acceptance in Europe.

He is an internationally recognised expert on asbestos and other minerals and his research work and testimony to US Government Federal Hearings was cited in the revision of the US Asbestos Regulations with respect to the non-asbestos varieties of tremolite, actinolite and anthophyllite.

He has acted as a consultant mineralogist in a large number of areas including vermiculite assessment, the importance of the silica minerals in commercial products and has been consulted in a range of legal cases. He has developed and provided training courses in the quantitative analysis of asbestos in mineral raw materials, and asbestos training courses to the syllabus of the British Examination Board in Occupational Hygiene for clients in the UK and Europe. He is currently involved in the development of assessment and remediation of contaminated land, as well as the use of microbiological tracing methods for groundwater contamination sites, with business associates in Edinburgh. He is author of more than 40 scientific publications in books, encyclopedias, scientific journals and technical reports, and has presented scientific papers at many national and international conferences

Industrial and other clients have included:

F. Hoffman La Roche, Basle

Dansk Eternit

The Vermiculite Association

Mandoval UK. Ltd

Cape Industrial Products

Skamol (Denmark)

Casella London

Williams Kastner & Gibbs, Seattle

Environmental Contamination Services

Scottish Courage

Dibb, Lupton & Allsop, Sheffield

McClure Naismith, Glasgow

Yorkshire Electricity

EDUCATION

1958 - 1964 High School of Stirling

SCE O-level : Latin, Arithmetic

SCE Higher : English, French, Mathematics, Science, Physics, Chemistry, Geography, Analysis

1964 - 1976 : University of Glasgow

BSc : Pure Science with Honours Degree in Geology

Subsidiary subjects : Chemistry, Natural Philosophy, Geography, Mathematics

1969 NERC Research Studentship, Dept. of Geology, University of Glasgow.

Research in geochemistry, petrology, mineralogy of basic and intermediate plutonic rocks of Moidart, Inverness-shire; mineralogy and chemistry of associated amphibole minerals.

WORK HISTORY

1972 Research Assistant/Assistant Keeper,

Dept. of Geology, Hunterian Museum, University of Glasgow

Responsibility for display and design of exhibits; curation of rock and mineral collection. Teaching mineralogy and petrology in lectures and laboratories.

1978-1993 Institute of Occupational Medicine Limited

Head of Mineralogy Section; Chemistry and Mineralogy Group Responsibilities for the identification and quantification of minerals in airborne dusts and bulk samples using X-ray Diffractometry (XRD), Infrared Spectrophotometry (IR.), Atomic Absorption Spectrophotometry, Optical Microscopy (OM), Scanning Electron Microscopy (SEM), Transmission Electron Microscopy (TEM), Energy Dispersive X-ray Spectrometry (EDXS).

Managing a section of nine staff involved in analysis of mineral and dust samples for major research projects and on contract to third parties, with general managerial duties, research and budgetary control, research programme development and consultancy to clients.

Research involvement in various fields has included:

Asbestos Studies

Identification and quantification of asbestos in relation to airborne dust exposure and airborne dust samples (OM, SEM, TEM)

Development of methods for identification and quantification of asbestos in bulk samples (OM, SEM) (Dept. of Environment and Health and Safety Executive)

Preparation of the Health and Safety Executive Method for the Determination of Hazardous Substances for Sampling and Identification of Asbestos by Polarised Light Microscopy (MDHS 77)

Establishment of UK National Reference Asbestos types on behalf of Health and Safety Executive

Evaluation of the release of asbestos fibres into air from disturbance of asbestos contaminated soils (Dept. of Environment)

Quantification of asbestos as trace contaminant in soils and loose aggregates (Dept. of Environment & Health and Safety Executive)

Determination of tremolite asbestos content of chrysotile, vermiculite, and other minerals (Asbestos Research Council)

Assessment of the toxicology of asbestos in parenteral medications and establishment of analytical methods for the determination of asbestos in medical preparations.

Quantification of asbestos and other minerals in lung burdens recovered from animals exposed during inhalation studies (IR., XRD, SEM)

Analysis of mineral components in respirable dusts generated from bulk samples from UK coalmines (IR., XRD)

Evaluation (IR., XRD, SEM) of general mineral dusts for experimental use for toxicity and carcinogenicity

Assessment of amphibole minerals (asbestiform and non-asbestiform tremolite) used in carcinogenicity assays.

Coal and Other Minerals

The shape, size and composition of particles in coalmine dusts (SEM, OM). (Coal and Steel Community Health in Mines Programme)

European co-operation in improvements of analysis (IR. and XRD) of minerals in respirable coalmine dusts (Directorate General - Social Affairs, Commission of the European Communities).

Epidemiology of diseases in UK opencast coalmines

Epidemiology of pneumoconiosis in quarrying industries

Epidemiology of diseases in heavy clay (brick etc.) industries

Toxicity Studies

Toxicity of quartz in pure mineral and mixed dust materials

Experiments on the toxicity of dusts in the London Underground System

Asbestos Autopsy

Identification and quantification of asbestos in human lung tissue (SEM, OM, TEM) including asbestos workers and control populations in UK (Asbestos Research Council)

Oil Shale Studies

Analysis (IR. and XRD) of mineral components in oil-shales of Scotland and Colorado (US Dept. of Energy), the geology of UK and US oil shales.

Teaching and instruction Courses

Instruction in asbestos mineralogy and use of optical microscopy and SEM in identification of asbestos in dust and bulk samples.

Development of distance learning programme for Diploma and MSc courses for Occupational Hygienists.

In-house training courses on asbestos identification, etc. for major national companies.

Training courses to BEBOH S603 syllabus for asbestos.

Instruction in mineralogy of asbestos in 'Asbestos and Lung Disease - A short course on the problems of asbestos for physicians'

Committee Memberships, Conferences etc.

Member of Working Group II of H&SE Committee on Fibre Measurement. Responsible for drafting of H&SE methods for asbestos analysis including, MDHS 77, Asbestos in bulk materials; Sampling and identification by polarised light microscopy, new methods for fibre discrimination in light microscope fibre counting (MDHS87), methods for the quantitative analysis of asbestos in contaminated land (MDHS 90), methods for the survey and sampling of asbestos in buildings (MDHS 100).

Member of Working Group 1 responsible for the oversight of the RICE proficiency testing scheme for asbestos fibre counting, the MMMF fibre counting proficiency testing scheme and AIMS the proficiency testing scheme for asbestos and other fibres in bulk materials.

Expert witness and consultant to various companies and individuals in UK and USA with respect to asbestos, asbestos lung burden analysis, asbestos in the general environment; asbestos carcinogenicity etc.

Attended and presented papers at international conferences including British Occupational Hygiene Society Annual Conference and Inhaled Particles V-VII, VII International Conference on Pneumoconiosis, 1988; Asbestos International Association Symposium from 1978, 1986, 1988, 1991 etc.; International Geological Congress, Kyoto , 1992,

Member of the Mineralogical Society, London since 1979.

Member of the British Occupational Hygiene Society since 1983.

Member of the Geological Society of Glasgow since 1967.

Referees:

Prof. Anthony Seaton
Department of Environmental and Occupational Medicine
The University of Aberdeen
Foresterhill
Aberdeen

Prof. Dr. Donald Kaiser
F. Hoffmann-La Roche
Global Biometrics
Grenzacherstrasse
CH-4002 Basel
Switzerland

Mr. R.M. Howie
12 Morningside Road
Edinburgh
EH10 4DB

PUBLICATIONS

Addison J (1997 in press). Asbestos, Analysis of. in Encyclopedia of Environmental Analysis and Remediation, Ed. Robert A. Meyers, John Wiley and Sons, New York.

Addison J. (1995). Asbestos, Methods of determination: in Encyclopaedia of Analytical Science; Academic Press

Addison J. (1995) Vermiculite : a review of the mineralogy and health effects of vermiculite exploitation. Reg. Tox. and Pharm : 21, 397-405.

Addison J. (1994) . Vermiculite is not asbestos: Report for The Vermiculite Association.

Addison J, Burdett, GJ, Chatfield, EJ, Christiansen, C-P, Gotz, J, Kaiser, D, Kurz, G, Langer, AM, Muhle, H, Wachberger, E. (1994). Analytical method for the Determination of Asbestos in Parenteral Medicines. Der Verband der Chemischen Industrie, Frankfurt, In Press.

Addison J, Browne K, Davis JMG, Gruber UF. (1993). Asbestos Fibers in Parenteral Medication. Reg Tox and Pharm : 18, 371-380.

Addison J, Davies LST, McIntosh C, Porteous R. (1992). Bulk Asbestos Reference Minerals for Optical Microscope Identification: Preparation, Evaluation and Validation. Edinburgh: IOM Report TM 90/06.) Final Report to client HSE Contract No R2490/R48.44.

Addison J. (1991). Improvements in the analysis of mineral components of coalmine dusts. Final Report on CEC Contract 7260.03.044.08. Edinburgh: Institute of Occupational Medicine Limited. (IOM Report TM/82/22)

Addison J, Asbestos. (1990). In Curwell S, March C, Venables R, Des Buildings and Health. RIBA Publications Limited, London.

Addison J, Davies LST. (1990). Analysis of amphibole asbestos in chrysotile and other minerals. Annals of Occupational Hygiene, 34: 159-175.

Addison J, Dodgson J. (1990). The influence of shape, size and composition of individual dust particles on the harmfulness of coalmine dusts: Development of Methods of Analysis. VIIth International Pneumoconiosis Conference Proceedings; Pittsburgh 1988, 287-290. US Department of Health and Human Services (NIOSH). Publication No. 90-108 Part 1.

Addison J, Miller BG, Porteous RH, Dodgson J, Robertson A. (1990). Shape, size and composition of coalmine dusts in relation to pneumoconiosis in British coalmines. Edinburgh: Institute of Occupational Medicine Limited. (IOM Report TM90/10).

Addison J, Davies LST, Robertson A, Willey RJ. (1988). The release of dispersed asbestos fibres from soils. Edinburgh: Institute of Occupational Medicine. (IOM Report TM88/14).

Addison J, Bolton RE, Davis JMG, Dodgson J, Gormley IP, Hadden GG, Robertson A. (1982). The relationship between epidemiological data and the toxicity of coalmine dusts. Final report on CEC Contract 7253-32/8/096. Edinburgh: Institute of Occupational Medicine. (IOM Report TM/82/22).

Bolton RE, Addison J, Davis JMG, Donaldson K, Jones AD, Miller BG, Wright A. (1986). Effects of the inhalation of dusts from calcium silicate insulation materials in laboratory rats. Environmental Research. 39: 26-43.

Bolton RE, Vincent JH, Jones AD, Addison J, Beckett ST. (1983). An overload hypothesis for pulmonary clearance of UICC amosite fibres inhaled by rats. British Journal of Industrial Medicine. 40: 264-272.

Breysse PN, Cherrie JW, Addison J, Dodgson J. (1989). Evaluation of airborne asbestos concentrations using TEM and SEM during residential water tank removal. Annals of Occupational Hygiene. 33: 243-256.

Cherrie JW, Addison J, Seaton A. (1991). Asbestos - an introduction. Handbook for asbestos training course. Institute of Occupational Medicine and Portsmouth Polytechnic.

Cherrie JW, Addison J, Dodgson J. (1989). Comparative studies of airborne asbestos in occupational and non-occupational environments using optical and electron microscope techniques. In: Bignon J, Peto J, Saracci R, eds. Non-occupational exposure to mineral fibres. 304-309 Lyon 1989: International Agency for Research on Cancer. (IARC Scientific Publication No. 90).

Cullen RT, Addison J, Brown GM, Cowie HA, Davis JMG, Hagen S, Miller BM, Porteous RH, Slight J, Robertson A, Vallyathan V, Wetherill GZ, Donaldson K. (1995). Experimental studies on dust in the London Underground with special reference to the effects of iron on the toxicity of quartz. Edinburgh, Institute of Occupational Medicine; (IOM Report TM/95/01)

Davies LST, Wetherill GZ, McIntosh C, McGonagle C, Addison J. (1993). Development and Validation of an analytical method to determine the amount of asbestos in soils and loose aggregates. Final Report on HSE Contract No 2491/R42.46 and DOE Contract No PECD 7/10/234. Edinburgh, Institute of Occupational Medicine.

Davis JMG, Addison J, McIntosh C, Miller BG, Niven K. (1991). Variations in the carcinogenicity of tremolite dust samples of differing morphology. Proceedings of the Collegium Ramazzini Symposium, New York, 1990. Annals of the New York Academy of Sciences; 643; 473-490.

Davis JMG, Addison J, Brown GM, Miller BG, Jones AD, McIntosh C, Whittington M. (1991). Further studies on the importance of quartz in the development of coalworkers pneumoconiosis. Final Report on CEC Contract 7284.33.044. Edinburgh: Institute of Occupational Medicine Limited. (IOM Report TM91/05)

Davis JMG, Addison J, Bolton RE, Donaldson K, Jones AD. (1986). Inhalation and injection studies in rats using dust samples from chrysotile asbestos prepared by a wet dispersion process. British Journal of Experimental Pathology. 67: 113-129.

Davis JMG, Addison J, Bolton RE, Donaldson K, Jones AD, Smith T. (1986). The pathogenicity of long versus short fibre samples of amosite asbestos administered to rats by inhalation and intraperitoneal injection. British Journal of Experimental Pathology. 67: 415-430.

Davis JMG, Addison J, Bolton RE, Donaldson K, Jones AD, Miller BG. (1985). Inhalation studies on the effects of tremolite and brucite dust in rats. Carcinogenesis. 5: 667-674.

Davis JMG, Addison J, Bolton RE, Donaldson K, Jones AD, Wright A. (1984). The pathogenic effects of fibrous ceramic aluminium silicate glass administered to rats by inhalation or peritoneal injection. In: World Health Organization: Regional Office for Europe. Biological effects of man-made mineral fibres. Proceedings of a WHO/IARC Conference in association with JEMRB and TIMA, Copenhagen 20-22 April 1982. Vol.2. Copenhagen: WHO, 1984: 303-322.

Davis JMG, Addison J, Bolton RE, Donaldson K, Jones AD, Wright A. (1983). Toxicology of calcium silicate insulating materials. Final report on MOD contract NCS 361/71364. Edinburgh: Institute of Occupational Medicine. (IOM Report TM/83/01).

Davis JMG, Addison J, Bolton RE, Donaldson K, Jones AD, Wright A. (1983). The pathogenic effects of fibrous ceramic aluminium silicate glass on rats. Abstract. In: World Health Organization: Regional Office for Europe. Biological effects of man-made mineral fibres. Report on a WHO/IARC meeting, Copenhagen, 20-22 April 1982. Copenhagen: WHO, 1983: 124. (WHO Euro Reports and Studies 81).

Davis JMG, Addison J, Bruch J, Bruyere S, Daniel H, Degueldre G, Dodgson J, Gade M, Gormley IP, Le Bouffant L, Martin JC, Reisner M, Robock K, Schliephake RW. (1982). Variations in cytotoxicity and mineral content between respirable dusts from the Belgian, British, French and German coalfields. In: Walton WH, ed. Inhaled particles V. Proceedings of an international symposium by the British Occupational Hygiene Society. Cardiff, 8-12 September 1980. Oxford: Pergamon Press, 1982: 541-549. (Annals of Occupational Hygiene; 26).

Donaldson, K, Addison, J, Miller, BG, Cullen, RT, Davis, JMG (1994) Use of short-term inflammatory response in the mouse peritoneal cavity to assess the biological activity of leached vitreous fibers. Environ. Health Perspect. 102 (Suppl 5): 159-162.

Donaldson K, Robertson A, Addison J, Waclawski ER, Soutar CA. (1991). Toxicity of quartz in mixed dusts with special reference to the London Underground. Edinburgh, Institute of Occupational Medicine Ltd. (IOM Report TM/91/03).

Gormley IP, Addison J. (1983). The in vitro cytotoxicity of some standard clay mineral dusts of respirable size. Clay Minerals. 18: 153-163.

Howie RM, Addison J, Cherrie JW, Robertson A, Dodgson J. (1986). Fibre release from filtering facepiece respirators. Letter. Annals of Occupational Hygiene. 30: 131-133.

Jones AD, Vincent JH, McIntosh C, McMillan CH, Addison J. (1989). The effect of fibre durability on the hazard potential of inhaled chrysotile. Exp. Pathol. 37, 1-4.

Jones AD, Vincent JH, McMillan CH, Johnston AM, Addison J, McIntosh C, Whittington MS, Cowie H, Parker I, Donaldson K, Bolton RE. (1988). Animal studies to investigate the deposition and clearance of inhaled mineral dusts. Final report on CEC Contract 7248/33/026. Edinburgh: Institute of Occupational Medicine. (IOM Report TM/88/05).

Langer AM, Nolan RP, Addison J. (1991). Physico-chemical properties of asbestos as determinants of biological potential. In Liddel FDK, Miller K, eds. Mineral Fibres and Health; CRC Press, Boston.

Langer AM, Nolan RP, Addison J. (1991). Distinguishing between amphibole asbestos and elongated cleavage fragments of their non-asbestos analogues. In: "NATO Advanced Research Workshop on Mechanisms in Fibre Carcinogenisis". Johnson NF Eds. Plenum Press, New York 1991.

Le Bouffant L, Addison J, Bolton RE, Bruch J, Bruyet B, Daniel H, Davis JMG, Degueldre G, Demarez J, Dodgson J, Gormley IP, Hadden GG, Kovacs MP, Martin JC, Reisner MTR, Robertson A, Rosmanith J. (1988). Compared in vitro and in vivo toxicity of coalmine dusts. Relationship with mineralogical composition. In: Dodgson J, McCallum RI, Bailey MR, Fisher DR, eds. Inhaled particles VI. Proceedings of an International Symposium and Workshop on Lung Dosimetry organised by the British Occupational Hygiene Society in co-operation with the Commission of the European Communities, Cambridge 2-6 September 1985. Oxford: Pergamon Press, 1988: 611-620. (Annals of Occupational Hygiene; 32 (suppl.1)).

Louw SJ, Addison J, (1985). Studies of the Scottish oil shale industry. Seaton A, ed. Vol.1. History of the industry, working conditions, and mineralogy of Scottish and Green River Formation shales. Final report on US Department of Energy Project DE-AC02-82ER60036. Edinburgh: Institute of Occupational Medicine. (IOM Report TM/85/02).

Nolan, RP, Langer, AM, Addison, J. (1994) Lung content analysis of cases occupationally exposed to chrysotile asbestos. Environ. Health Perspect. 102 (Suppl 5): 245-250.

Nolan RP, Langer AM, Oechsle GW, Addison J, Colflesh DE. (1991). Association of tremolite habit with biological potential: In "NATO Advanced Research Workshop on Mechanisms in Fibre Carcinogenesis". Brown RC, Hoskins JA and Johnson NF Eds. Plenum Press, New York.

Seaton A, Addison J, Davis JMG, Hurley JF, McGovern B, Miller BG. (1987). The toxic effects of silica. A report prepared for the Health and Safety Executive. Edinburgh: Institute of Occupational Medicine. (IOM Report TM/87/13).

Seaton A, Ruckley VA, Addison J, Brown WR. (1986). Silicosis in barium miners. Thorax; 6; 41: 591-595.

Tylee BE, Davies LST, Addison J. (1996) Asbestos Reference Standards - Made Available for Analysts. Ann. occup. Hyg., Vol 40, No 6, pp. 711-714.

Vincent JH, Johnston AM, Jones AD, Bolton RE, Addison J. (1985). Kinetics of deposition and clearance of inhaled mineral dusts during chronic exposure. British Journal of Industrial Medicine; 42: 707-715.

CURRICULUM VITAE
Ann G. Wylie

Notarization. I have read the following and certify that this curriculum vitae is a current and accurate statement of my professional record.

Signature_____ Date_____

1. PERSONAL INFORMATION

Ann Gilbert Wylie
Assistant President and Chief of Staff
Interim Dean of the Graduate School
Office of the President
1101 Main Administration
University of Maryland
College Park, MD 20742
E-mail: awylie@umd.edu
Phone: (301) 405-6848
Fax: (301) 314-9560

Professor of Geology
Department of Geology
University of Maryland
College Park, Maryland 20742
E-mail: awylie@umd.edu
Phone: (301) 405-4079
Fax: (301) 314-9661
Web site: http://www.geol.umd.edu/pages/faculty/WYLIE/wylie.html

Educational: Ph.D. 1972 Columbia University, New York, New York
Major: Economic Geology
Minors: Mineralogy, Mining Engineering, and Petrology

B.A. 1966 Wellesley College, Wellesley, Massachusetts
Major: Geology

Employment:

a. Academic:

2. Research, Scholarly, and Creative Activities

a. Books

i. Chapters or Articles in Books:

Gilbert, J. Ann (1967) "Units, Numbers, Symbols and Constants", Encyclopedia of Atmospheric Sciences and Astrogeology, Rhodes Fairbridge (ed.). Reinholt Publishing Company, p. 1049-1062.

Wylie, A.G. (1981) Numerous Mineral Descriptions in Encyclopedia of Mineralogy, K. Frye (ed.). Reinholt Publishing Company.

Steel, E. and A. Wylie (1981) "Mineralogical Characteristics of Asbestos". In Geology of Asbestos Deposits, P.H. Riordon (ed.). Society of Mining Engineers of AIME, p. 93-100.

Invited
Veblen, D.R. and A.G. Wylie (1993) "Mineralogy of Amphiboles and 1:1 Layer Silicates" in Health Effects of Mineral Dusts, G.D. Guthrie & B.T. Mossman (eds.). Reviews in Mineralogy, v. 28, Min. Soc. Am., p. 61-131.

Invited
Wylie, A.G. (1995) "The Analysis of Industrial Mineral Products for Crystalline Silica by Optical and Electron Microscopy: A Literature Review". In: Review Papers on Analytical Methods, Chemical Manufacturers Association.

Invited
Wylie, A.G. and P.A. Candela (1999) "Metallic Mineral Deposits - Chromite". In Geol. of Pennsylvania, Pennsylvania Geol. Survey and Pittsburgh Geol. Survey, Special Publication 1, p.588-595.

b. Articles in Refereed Journals

Wylie, A.G. and P.J.M. Ypma (1974) "Determination of the Optical Parameters, n and k, of Absorbing Minerals with the Microscope: Isotropic Minerals". Economic Geology 52, p. 1300-1327.

Wylie, A.G. (1979) "Fiber Length and Aspect Ratio of Some Selected Asbestos Samples". Annals of the New York Academy of Science 330, p. 640-643.

Wylie, A.G. (1979) "Optical Properties of the Fibrous Amphiboles". Annals of the New York Academy of Science 330, p. 600-605.

Zoltai, Tibor and A.G. Wylie (1979) "Definitions of Asbestos-related Mineralogical Terminology". Annals of the New York Academy of Science 330, p. 640-643.

Wylie, A.G., Candela, P.A. and Burke, T.M. (1987) "Compositional Zoning in Unusual Zinc-rich Chromite from the Sykesville District, Carroll County, Maryland". American Mineralogist 72, p. 413-423.

Wylie, A.G. (1988) "Relationship between the Growth Habit of Asbestos and the Dimensions of Asbestos Fibers". Mining Engineering, November (1988) p. 1036-1040.

Muller, P.D., Candela, P.A. and Wylie, A.G. (1989) "Liberty Complex: Polygenetic Melange in the Central Maryland Piedmont". GSA Special Paper 228 Melanges and Olistostrome of the U.S. Appalachian, J.W. Horton and N. Rast (eds.), p. 113-135.

Candela, P.A., Wylie, A.G. and Burke, T.M. (1989) Genesis of the Ultramafic Rock-Associated Fe-Cu-Co-Zn-Ni Deposits of the Sykesville District, Maryland Piedmont". Economic Geology 84, p. 663-675.

Wylie, A.G. (1990) "Discriminating Amphibole Cleavage Fragments from Asbestos: Rationale and Methodology". Proceedings of the VIIth International Pneumoconiosis Conference, Pittsburgh, p.1065-1069.

Linder, D.E., Wylie, A.G. and Candela, P.A. (1992) "The Mineralogy and Origin of the State Line Talc Deposit, Pennsylvania". Economic Geology 87, p. 157-165.

Wylie, A.G. and Bailey, K.F. (1992) "The Mineralogy and Size of Airborne Chrysotile and Rock Fragments: Ramifications of Using the NIOSH 7400 Method". American Industrial Hygiene Association Journal 53, p. 442-447.

Wylie, A.G. (1993) "Modeling Asbestos Populations: The Fractal Approach". Canadian Mineralogist 30, p. 437-446.

Wylie, A.G., Bailey, K., Kelse, J. and Lee, R. (1993) "The Importance of Width in Fiber Carcinogenicity and its Implications for Public Policy". American Industrial Hygiene Association Journal 54, p. 239-252.

Verkouteren, J.R., Wylie, A.G., Steel, E.B. and Lim, M.S. (1995) "Analysis of the Tremolite/Actinolite series using high precision refractive index measurements," Proceedings of the 29th Annual Meeting of the MAS, Breckinridge, CO, p.27-28.

Wylie, A.G., Skinner, H.C., Marsh, J., Snyder, H., Garzione, C., Hodkinson, D. and Winters, R. (1997) "Mineralogical Features Associated with Cytotoxic and Proliferative Effects of Fibrous Talc and Asbestos on Rodent Tracheal Epithelial and Pleural Mesothelial cells. Journal of Toxicology and Applied Pharmacology 147, p. 143-150.

Invited
Wylie, A.G. (1988) "The Relationship Between the Growth Habit of Asbestos and the Dimensions of Asbestos Fibers". Society of Mining Engineers Preprint #88-85, p.1-7.

Invited
Wylie, A.G. (1989) "Mineralogical Definitions for Asbestos Fibers and Cleavage Fragments". Report of the Committee on Geology and Public Policy GPP012. Geological Society of America, p. 2-4.

Invited
Wylie, A.G. (1996) "Factors Affecting Risk from Biologically Active Minerals", Proceedings, Mineral Dusts: Their Characterization and Toxicology, Washington, D.C. Society for Mining Metallurgy & Exploration, Littletown, Colorado, Sept. 19-20, 1996, p. 33-46.

Prestegaard, K., Wylie, A.G. and Piccoli, P.M. (1999) Characterization of Grout Samples at Winding Ridge." Power Plant Research Program, Maryland Department of Natural Resources.

Schwartz, C., A.G. Wylie, A. Davis, B. James (2000) "Investigation of the Expansive Behavior of Chromium Tailings: Final Report on Phase II Investigations".

Piccoli, P.M., DeHarde, A., Wylie, A.G., and Prestegaard, K. (2000) "Development of a Grout for the Kempton Mine: Characterization (XRD, Chemical Analyses, and SEM/EPMA Data) of Starting Materials. Power Plant Research Project Report, Maryland Department of Natural Resources.

e. Abstracts and Professional Papers presented

Gilbert, Jean Ann and P.J. Ypma (1969) "The Use of an Electro-Optical Compensator for the Determination of the Optical Properties of Opaque Minerals Under the Microscope", GSA Annual Meeting, Atlantic City, New Jersey.

Siegrist, H.G. and A.G. Wylie (1979) "Characterizing and Discriminating the Shape of Asbestos Particles", GSA Annual Meeting San Diego, California.

Invited
Wylie, A.G. and P. Schweitzer (1980) "The Effects of Grinding on the Shape of Wollastonite Particles". Symposium on Electron Microscopy and X-ray Applications to Environmental and Occupational Health Analysis, Penn State.

Huggins, C., A.G. Wylie and W. Campbell (1980) "Preparation and Selected Properties of Amosite, Chrysotile, Crocidolite and Non-fibrous Tremolite for Use in NIEHS Oral Ingestion Studies". Symposium on Electron Microscopy and X-ray Applications, Penn State.

Invited
Wylie, A.G. (1989) "Distinguishing Tremolite-Asbestos from Tremolite Cleavage Fragments on a Light Optical and Morphological Basis", VII International Pneumoconiosis Conference Proceeding of Workshop: Hazard Recognition of Mineral Dust. Pittsburgh, NIOSH-ILD-BOM-MSHA-OSHA.

Wylie, A.G., Linder, D. and Candela, P. (1990) "Sedimentary Features of Appalachian Serpentinites". Geol. Soc. of Amer. National Meeting, Nov. (1990), p. A230.

Invited
Skinner, C. and Wylie, A. (1990) "Fibrous Tremolites". Bloss Symposium, VPI, Blacksburg, Virginia.

Invited
Wylie, A.G. (1992) The Analysis of Industrial Mineral Products for Crystalline Silica by Optical and Electron Microscopy. The Measurement of Crystalline Silica International Symposium, August (1992).

Wylie, A.G. (1993) The Fractal Distribution of the Mass of Asbestos Fiber and its Application to the Analysis of Industrial Minerals. Geological Society of America Annual Meeting, Boston.

Verkouteren, J.R. and Wylie, A.G. (1994) "Anthophyllite, Tremolite, and Actinolite Asbestos: Reference Materials and Optical Properties" Inter/Micro 94, Chicago.

Verkouteren, J.R., Wylie, A.G., Steel, E.B., Lim, M.S. (1995) "Analysis of the Tremolite-Actinolite Series using High Precision Refractive Index Measurements". Microbeam Analysis.

Invited
Wylie, A.G. (1996) "Factors Affecting Risk from Biologically Active Minerals". Society of Mining, Metallurgy & Exploration Symposium. Mineral Dusts: Their Characterizations and Toxicology.

Invited
Wylie, A.G. (1997) "The Habit of Asbestiform Amphiboles: Implications for the Analysis of Bulk Samples" 1997 Boulder Conference: Advances in Environmental Measurement Method for Asbestos. University of Colorado, Boulder, July 13-17 (1997).

Verkouteren, J.R. and A. G. Wylie (2001) "Microdiffraction Analysis of Fibrous Talc: Asbestos in Crayons". 2001 Denver X-ray Conference, Steamboat Springs, Colorado, USA, August 2, 2001.

Piccoli, P.M., DeHarde, A., Wylie, A.G. (2001) "Recycling coal Combustion Byproducts: a Laboratory Study to Evaluate Grout Formulations for Use in the Kempton Mine Complex, Western Maryland. Geological Society of America, Abstracts with Programs.

Principal Investigator, "Mineralogical Analysis of Road Aggregate Used in Maryland", Maryland Highway Administration, $8,089. November 1974-July 1975.

Co-Principal Investigator, "Optical Microscopic Electron Optical and Microprobe Characterization of Asbestos-Related Materials", U.S. Bureau of Mines, $16,645. December 1975-November 1976.

Principal Investigator, "Serpentines", Faculty Research Board, University of Maryland, $750. Summer 1976.

Principal Investigator, "Optical Microscopic and Electron Optical Characterization of Asbestos-Related Materials", U.S. Bureau of Mines, $11,188. December 1976-November 1977.

Principal Investigator, "Characterization of Asbestos-Related Materials Including Particle Size Distribution, Aspect Ratio and Orientation", U.S. Bureau of Mines, $21,437. December 1977-November 1978.

Principal Investigator, "Mineralogical and Size Distribution Studies of Amphibole Asbestos", U.S. Bureau of Mines, $84,200. April 1979-April 1981.

Principal Investigator, "Dispersion Staining in Optical Mineralogy", Undergraduate Fund for Improvement of Instruction, University of Maryland, $700. 1982.

Principal Investigator, "Quality Control in the Analysis of Asbestos by PLM", $10,000. Sept. 1985-Sept. 1986. Occupational Medical Center.

Principal Investigator "Mineralogy of the Sand Fraction of Aquifer in Northwestern Washington". United States Geological Survey, $2,450. June-October 1986.

Univ. of Maryland General Research Board Semester Research Award, $1,500. 1987.

Mineralogy of Waste Product of Sand and Gravel Processing". Aggregate Industries, $12,000. 1987-1988.

Characterization and Quantification of Fibrous Tremolite in Tremolitic Talc. Southern Talc Company, $17,000. 1989-1990.

Principal Investigator, "Mineralogical Characteristics of Fibrous Talc". R.T. Vanderbilt Company, $23,500. September 1992-December 1997.

Project Director, "Fellowship for the Study of Industrial Talc". R.T. Vanderbilt Company, $33,500. January 1, 1993-December 31, 1997.

Co-Project Director, "Research and Laboratory Testing of Chromium Processing Waste at

3. Teaching, Mentoring, and Advising

a. Courses taught since 1986

Course	Approximate Average Enrollment
Physical Geology	150
Economic Geology	10
Optical Mineralogy	6-10
Ore Microscopy	3
Senior Thesis Research	10
Advanced Topics in Economic Geology	14
Geology of Maryland	6
Geology and Public Policy	15
Environmental Geology	60

b. Advising: Research Direction

i. Undergraduate Thesis (beginning 1980) Major Advisor:

1980 [1]Ed. Jacobsen "Coal Geology of Garrett County, Maryland"

1982 Sharron O'Donnell "Coal Geology of Southwestern Kentucky
Eric Windsor "Shape Characterization of Amphiboles"
Morris Levin "Characterization of Part of the Sykesville Magnetite District by a Magnetometer"
Lyle Griffith "The Use of a Magnetometer in Characterizing the Beasman Prospect, Sykesville, MD."
[2]John Varndell "Heavy Element and Particle Size Relationships in a Sludge Disposal Site, Baltimore, Maryland"
Joe Segretti "Relationship between cytotoxicity and coating of chrysotile fibers"
Mark Beal, A Geologic Evaluation of a Placer Gold Deposit in Southern Fauquier Co., Virginia

1983 Keith Mason "A Preliminary Evaluation of Copper and Cobalt in Conjunction with Iron Mining in the Beasman Prospect of Sykesville, Md."
Michael D. Jones "Chromium in the Soils and Streambeds above the Hunting Hill Serpentinite Body, Montgomery County, Md."
Theresa Baker "Crack Growth in Quartz: The Effects of Chemical Environments"
Mark Hevey "Gas Production and Faulting in Gas Field, Kansas"

---

[1]Winner of the AAPG National Undergraduate Research Award

[2]2nd Place Winner of the AAPG National Undergraduate Research Award

1988 Robert Virta, M.S., "An Evaluation of the Adequacy of Morphological Data for Determining the Carcinogenicity of Minerals"

1990 Dan Linder, M.S., "The Mineralogy and Origin of the State Line Talc Deposit, Lancaster Co., Pennsylvania"

1991 Tim Rose, M.S., "Petrology and Chemical Variation of Peraluminous Granitic Rocks from the Northern Lobe of the Phillips Pluton, Maine"

1996 Jiang Feng, M.S., "Evidence for compositional variation in phyllite from Carroll and Frederick Counties, MD"

1988 William Greenwood, M.S. "Mineralogical Characteristics of Fibrous Talc"

Diane Hanley, M.S., "Overland flow evaluation of lava flow platform"

1999 Mark Watson, M.S., "Effects of intergrowths on the Physical Characteristics of fibrous Anthophyllite"

2001 Amina DeHarde, M.S., "Characterization of Grouts made from Coal Combustion By-Products: Mineralogy and Physical Properties"

2005 Courtney Crummett, M.S. (co-chair) "Examination of the Thermal decomposition of Chrysotile"

iii. Ph.D.

1991 James Crowley, Ph.D., "Geochemical Study of Playa Efflorescent Salt Crusts and Associated Brines by Using Spectral Reflectance, X-ray Diffraction and Brine Chemical Data"

1999 Martitia Tuttle, Ph.D., "Late Holocene Earthquakes and their Implications for Earthquake Potential of the New Madrid Seismic Zone, Central United States"

SEM, Phase Contrast Optical Microscopy and Polarized Light Microscopy. 1985-1990. Author of Polarized Light Microscopy Method (grey sheets).

Expert witness, Occupational Safety and Health Administration hearing on asbestos regulation, 1985, 1990.

Invited participant, Penn. Geol. Survey Conference on Mapping in the Piedmont, 1987.

Expert panel member, EPA, Superfund Bulk Asbestos Method, 1990-1991.

b. Campus

i. Departmental

Committee of the Whole 1972-1981
Department Chairman Search Committee 1980-81
Department Secretary 1982-83
United Way Representative 1982
Development of Upper-level Courses for General Student 1984
Building Committee (1986-1990)
Scheduling Committee (1986-1994)
Undergraduate Affairs Committee (1987-94)
Departmental Field Trip Coordinator (1987-88)
Library Representative (1987-1990)
Acting Chair (1989-1990)
Graduate Committee (member 1990-present; Chair 1990-94)
Ad-hoc Committee for Departmental Policy on Training TAs. 1991 Chair
Ad-hoc Field Trip Committee (1992)
Salary Committee (1993; Chair, 1994)
Ad-hoc Committee on Faculty Workloads (1994-95)
Search Committee Chair - Associate Staff (1994-95)
Search Committee - Faculty (1995-96)

ii. Division of Agriculture and Life Sciences Committees:

Science in Action, 1973, "X-ray Diffraction of Minerals"
Committee of the Faculty, Division Council, Chairman, 1973-74, 1974-75
Division Council, 1973-76, Chairman, 1976-77
Committee for By-laws, Division Council, 1975-76
Executive Committee, Division Council, 1973-76
Search Committee, Director Ag. Exper. Station 1975-76
Opportunities for Women in Engineering and Science Planning Committee, 1975-76
Library Committee, 1975-1979
Ad Hoc Committee to revise Plan of Governance, 1978
Search Committee, Department Chairman (Geology), 1979
Search Committee, Provost of the Division, 1980
Ad Hoc Committee for the review of salaries of faculty women, 1982
Ad Hoc Committee on enrollments, 1982-83

Creative and Performing Arts Board, 1984-1985. Chairman, 1985
CMPS Dean Search Committee, 1987
Athletic Council, 1987-1990, 1996-2000 (Chair, Academic Committee, 1989-1990; Vice Chair of Athletic Council, 1996 - 2000)
Academic Standards Committee, Graduate School, 1987-1990; Chair, 1989-90; 1992-94
Water Resources Research Board, Scientific Review Committee, 1987, 1988, 1989
Packard Fellowship Review committee, 1991, 1995
Learning Assessment Outcomes Committee, Middle States Review, 1991
Graduate Council, 1991-94
President's Medal Committee, 1991, 1992, 1999
Advise 5 Advisor, 1992-1998
Graduate Council Committee on Academic Standards 1990-1994
Graduate Council Committee on Programs, Courses and Curricula, 1993-1996
Search Committee - Associate Provost for Faculty Affairs, 1993-94
Campus Writing Board, 1994-1995
APT Appeals Committee, 1994, 1996
Letters and Science Review Committee, 1994
College of Arts and Humanities Writing Center Review Committee, 1994-95
Search Committee - Director of Libraries, 1995-1996
Provost's Advisory Committee on Admissions and Advising, 1995-1998; Chair, 1996-1998, 2000-2001
Environmental Science and Policy Planning Committee, 1995-1996
Review Committee, CMPS Dean, 1996
Enrollment Management Advisory Committee, 1996-1998
Environmental, Safety and Health Policy Committee, 1996-1999
Steering Committee, Environmental Science and Policy Program, 1997-1999
Screening Committee ORAU Junior Faculty Enhancement Award, 1997, 1998
UMCP representative to Mineral Resources Section, National Association of State Universities and Land Grant Colleges, 1997-present
McNair Program, Mentor to Blessing Asuguo, 1997
Undergraduate Programs Advisory Committee 1998-1999
Banneker-Key Selection Committee 1999
Task Force on grading 1999-2000
Steering Committee NCAA Recertification 1999-2000
Campus Assessment Work Group, Chair, 2000-2001
Middles States Accreditation 5-year Interim report, Steering Committee Chair, 2000-2001
President's Task Force on Student Success, 2000-2004
Chair, Search Committee for the Vice President for Research and Dean of the Graduate School, 2001-2002

c. Community, State, National

Rural Southeast Olney Civic Association, 1977-present.
Wellesley College Alumnae Association, Treasurer of the Class of 1966, 1981-1986.
Co-Chairman, St. John's Episcopal School Building Campaign, 1987-1990.

Ann Wylie is Professor of Geology and Distinguished Scholar Teacher at the University of Maryland. Dr. Wylie graduated with honors from Wellesley College and received her Ph.D. from Columbia University in 1972. She has been on the faculty at the University of Maryland for 33 years and is the author of more than 50 articles on mineralogy, including many of the mineralogical characteristics of asbestos. She has served in a variety of administrative positions. In 2001, she was appointed Assistant President and Chief of Staff of the University of Maryland and in 2004, she was appointed Interim Dean of the Graduate School.

MALCOLM ROSS, Ph.D.

PROFESSIONAL TECHNICAL RECORD (updated to December 7, 2005)

Scientific or technical specialties

a) Mineralogy b) Igneous and metamorphic Petrology c) Minerals and Health d) Health Policy e) Crystal structure analysis.

Other scientific, technical, or special skills

Operates transmission and scanning electron microscopes, electron microprobe, optical microscopes, and all types of X-ray diffraction equipment including single crystal precession and Weissenberg cameras, manual and automated diffractometers, and powder cameras.

Education (including secondary schools)

School	Major and Minor Specializations	Dates Attended	Degree, Year
Calvin Coolidge H.S.	Science	9/44 - 6/47	Grad., 6/47
Utah State University	Zoology, Chemistry	9/47 - 6/51	B.S., 6/51
University of Maryland	Physical Chemistry	9/56 - 6/59	M.S., 6/59
Harvard University	Geology, Chemistry	9/59 - 6/62	Ph.D., 6/62

Specialized training (including postgraduate and Government courses).

1970 Massachusetts Institute of Technology special 2 week summer program for training in the use of the electron microprobe.
1981 Amphiboles and Other Hydrous Pyriboles - Mineralogical Society of America Short Course (Erlanger, Kentucky, 2 days).
1984 Yale University School of Medicine 8 hour course on "Indoor Air Quality".
1984 Micas - Mineralogical Society of America Short Course (Reno Nevada, 2 days).
1986 Scanning electron microscope training (USGS, Reston, 3 days).
1986 Hydrous Phyllosilicates - Mineralogical Society of America Short Course (Denver, Colorado, 2 days).
1989 Advanced Powder Diffraction - Mineralogical Society of America Short Course (St. Louis, Missouri, 2 days).
1990 Geochemistry and Mineralogy of Rare Earth Elements - Mineralogical Society of America (USGS, Reston, 1 day).

Membership in professional societies: Dates and significant offices held.

1) Mineralogical Society of America (1952-present), elected Fellow in 1959, Treasurer 1976-1980, Vice President 1990, President 1991.
2) Geological Society of America (1963-present), elected Fellow in 1979.
3) American Geophysical Union (1971-present).
4) American Association for the Advancement of Science (1965-present), elected Fellow in 1983.
5) Clay Minerals Society (1960-1980, 1988-1994).
6) Mineralogical Association of Canada (1989-present).
7) Geological Society of Washington (1956-present), Chairman of the Finance Committee in 1976.

Scientific and Public Service

Lectureships, symposia, invited conference participation.

1) March, 1965. Invited lecturer on the topic of the crystal chemistry of micas at the USGS Centers in Washington DC, Denver, and Menlo Park.
2) September 7-11, 1969. Invited lecturer "Pyroxene-Amphibole Symposium", V.P.I., Blacksberg, VA. Topic was "Chemical Reactions in Pyroxene Crystals".
3) August 26-September 7, 1972. Invited speaker at the 9th International Congress of Crystallography, Kyoto, Japan - gave two talks on mineralogy and crystal structure of rock-forming silicates
4) November 13-15, 1972. Co-convener of a symposium on "Exsolution and Domain Structure in Minerals, Geological

Society of America meeting, Minneapolis, Minnesota

5) October 29-30, 1981. Invited lecturer on asbestiform silicates for the Mineralogical Society of America Short Course on Amphiboles, Erlanger, Kentucky.
6) May 24-27, 1982. Invited speaker at the "World Symposium on Asbestos", Montreal, Quebec, Canada. Topic was the geological occurrences of asbestos
7) February-March, 1984. Distinguished Regional Lecturer, USGS, Geologic Division, 1983-1984. Gave a talk titled "The Dangers of Asbestos in Our Environment: Separating Fact from Fiction", Reston, VA, February 21, 1984; Denver, CO, March 13, 1984; Menlo Park, CA, March 15, 1984, Flagstaff, AZ, March, 19, 1984.
8) July 13-18, 1986. Plenary speaker, 14th General Meeting, International Mineralogical Association, Stanford, CA, "The Dangers of Asbestos in Our Environment: Fact and Fiction".
9) October 1988. Co-chairman of the Geological Society of America Forum on "Fibrous Minerals, Mining, and Disease", 1988 GSA Meeting, Denver, CO.
10) July 9-19, 1989. Co-convener at the 28th International Geological Congress, Washington, DC of a symposium titled "Alkaline Igneous Rocks and Carbonatites".
11) Feb. 28, 1991. Invited speaker at British Columbia's Mining Work Symposium - "Suspect Minerals and Human Health".
12) Oct. 22, 1992. Gave the Presidential address at the annual meeting of the Mineralogical Society of America, titled "Crystalline solution series and order-disorder within the natrolite mineral group".
13) Jan. 10, 1992. Invited talk at the Geophysical Laboratory, Washington DC, titled: "Crystalline solution series and order- disorder within the natrolite mineral group".
14) May 5, 1992. Invited talk at the 28th Forum on the Geology of Industrial Minerals, titled: "The effect of regulations on asbestos and other designated mineral carcinogens on the vitality of the industrial minerals industries".
15) Sept. 29, 1992. Invited talk at the annual meeting of the American Association of Professional Geologists, titled: "Suspect minerals-an environmental concern?".
16) Nov. 3, 1992. Invited talk at the annual meeting of the Clay Minerals Society, titled: "Suspect minerals and human health".
17) Nov. 17, 1992. Invited talk at the Intertech conference - A Management perspective on Crystalline Silica, titled: "The asbestos fiasco - a model for crystalline silica?".
18) April 16, 1993. Invited lecturer on the health effects of mineral dusts for the Society of Economic Geologists Short Course on Environmental Geochemistry of Mineral Deposits.
19) Oct. 22-24, 1993. Invited lecturer on the health effects of mineral dusts other than asbestos for Mineralogical Society of America Short Course on Minerals and Health, Nantucket, MA.
20) Oct. 7-11, 1996. Invited lecturer for a course titled "Minerals and Health", eight lectures given at the Institute of Mineralogy and Petrology, University of Fribourg, Fribourg, Switzerland.
21) October 16, 1996. Invited lecturer at the Institute of Physics of the Globe at Paris, University of Paris, Paris, France. Title the lecture was " The United States Asbestos Abatement Program.
22) November 7-9, 1998. Co-organizer of National Academy of Sciences Colloquium, "Geology, Mineralogy, and Human Welfare, NAS Beckman Center, Irvine, CA.
23) November 12-22, 2000. Engaged in a Fulbright sponsored mission to study the possible health effects of asbestos fibers originating from the asbestos deposits in the Troodos mountains of Cyprus and presented opinions as to the possible hazards of asbestos in air and water to the residents. Talks were given at the Cyprus Geological Survey and at the Near East University of North Cyprus.

<u>An additional 61 invited talks on the subjects of minerals and health and the geological occurrences the asbestos minerals were given at the following institutions or organizations (by year):</u>

1976 1) Penn State U., special conference on measurement of mineral particles, State College, PA.

1977 1) SME-AIME Meeting, St. Louis, MO.
2) National Institutes of Health symposium on health effects of dust from the Rockville Quarry, Bethesda, MD.
3) National Bureau of Standards symposium on asbestos, Gaithersburg, MD.

1978 1) ASTM symposium on asbestos, Rockville, MD.
2) CON-AGR Convention, Los Vegas, Nev.
3) Washington Crystal Colloquium, Washington, DC.
4) Geological Society of Washington meeting, Washington. DC.

1979 1) Staff at the Dept. of Justice, Washington, DC.
2) Geological Society of Washington meeting, Washington, DC.
3) Staff , Dept. of Pathology, Vermont Medical School, Burlington, Vermont.

4) Special symposium at the 1979 Geological Society of America meeting, San Diego, CA.
5) Special conference at the NIH on minerals and health, Bethesda, MD.
6) Symposium on regulation of fibrous minerals, Clay Minerals Society, Rochester, NY.

1980 1) Dept. of Geology, U. of Maryland, College Park, MD.
2) Officials of Homestake Mining Company, San Francisco, CA.

1981 1) River Road Universal Church, Bethesda, MD.
2) Geophysics seminar, USGS, Reston, VA.
3) Dept. of Geology, Harvard U., Cambridge, MA.

1982 1) Dept. of Geology, Lehigh U., Bethlehem, PA.

1983 1) Philadelphia Geological Society, Bryn Mawr, PA.
2) Dept. of Geology, Indiana U., Bloomington, Ind.
3) School of Public and Environmental Affairs, Indiana U., Bloomington Indiana.

1984 1) Vermont Marble Company, Proctor, VT.
2) To Officials at the U.S. Dept. of the Interior, Washington, DC.
3) Arizona Geological Commission, Tucson, AZ.
4) U.S. Bureau of Mines, Washington, DC.
5) Society of Chemical Engineers, Baltimore, MD.
6) U.S. Environmental Protection Agency, Washington, DC.
7) U.S. National Museum, Washington, DC.

1985 1) 21st Annual Forum on the Geology of Industrial Minerals, Tucson, AZ.
2) Asbestos Information Association meeting, Alexandria, VA.
3) Dept of Geology, U. of New Mexico, Albuquerque, NM.

1986 1) Harvard U., Friends of the Museum meeting, Cambridge, MA.
2) U.S. General Services Administration conference on asbestos in buildings, Denver, CO.

1987 1) USGS-USBM mineral commodity meeting, Reston, VA.
2) Workshop on "Tremolite in Play Sand", U. of Massachusetts, Amherst, MA.
3) All Souls Church, 16th St. NW, Washington, DC.
4) American Institute of Mining and Metallurgical Engineers, Mariposa Section, Phoenix, AZ.

1988 1) Baltimore Cytological Society, Baltimore, MD.
2) Rutgers Geological Museum Open House, New Brunswick, NJ.
3) U.S. General Services Administration conference on Federal building management problems, Colorado Springs, CO.
4) U.S. General Services Administration Real Estate Policy Conference, San Antonio, TX.

1989 1) Dept. Of Geology, U. of Delaware, Newark, Delaware.
2) Society of Mining Engineers conference on "Fibers, Fibers, Fibers", Baltimore, MD.
3) Commencement Address to the geology graduates, Virginia, Polytechnic Institute, Blacksburg, VA.
4) Talk on asbestos and public policy to Officials at V.P.I., Blacksberg, VA.
5) Talk for the Docents, U.S. National Museum, Washington, DC.

1990 1) Geophysical Lab, Washington, DC.
2) Talk to Administrators, State Government of Virginia, Richmond, VA.
3) Talk at the 1990: National Consumer Coalition press conference, National Press Club, Washington, DC.

1991 1) Dept. of Geology, University of Oklahoma.

1992 1) The Cato Institute, Washington, D.C.

1993 1) Dept. of Geology, Lafayette College, Easton, PA (two talks)
2) Dept. of Geology, Yale University, New Haven, CT.

1994 1) Smithsonian Institution, Washington DC.
2) Department of Geology, University of Texas-Permian Basin, at Odessa, Texas.
3) Society of Independent Professional Earth Scientists, Midland, Texas.
4) Department of Geology, Texas A&M University. College Station, Texas.
5) Public Relations Society of America National Environmental Conference, Washington, DC.

1995 1) Geological Society of Washington, Washington DC
2) V.M. Goldschmidt Conference, State College, PA

1996 1) Geology Department, George Washington University, Washington DC
2) Lecture: "The Asbestos and Health Issue, an example of a Costly Conflict Between Science and Public Policy", given before the Department of Geology, California State University, Sacramento, Sacramento, CA, Nov. 12, 1996.

1997 1) U.S. Geological Survey, Reston VA. Lecture titled :But is it True? An examination of the science used to establish asbestos, radon, DDT, acid rain, and global warming risks.
2) Lecture titled "Occurrence, production and uses of asbestos", Workshop on The health effects of chrysotile asbestos, Montreal, Canada, September 15,1997.

1999 1) Geology Department, George Mason University, Fairfax City, VA.. Lecture titled :But is it True? An examination of the science used to establish asbestos, radon, DDT, acid rain, and global warming risks.

2000 1) Geological Survey of Cyprus, Nicosia, Cyprus. Three Lectures: Causes of Cancer, Asbestos Diseases and Asbestos Minerals, and Geological Occurrences of Asbestos.
2) Department of Civil Engineering, Near East University, Nicosia, North Cyprus. Three Lectures: Causes of Cancer, Asbestos Diseases and Asbestos Minerals, and Geological Occurrences of Asbestos.

Committees to render scientific judgment. Include scientific review panels, editorial boards, editorships, with dates. Include the capacity in which you served (chairman, subcommittee chairperson, member, observer, expert consultant, etc.).

1) Reviewer for National Science Foundation proposals. About two proposals per year, 5-10 hours work, average funding request about $75K (1965-present).
2) Member of the Roebling Medal Committee, Mineralogical Society of America, in 1970.
3) Associate Editor, The American Mineralogist, average about 30 manuscripts a year, many hours of work involved in editing (1982-1986).
4) USGS liaison to the National Academy of Sciences panel convened to evaluate the health hazards of the various types of asbestos minerals (1979-1982). In 1978 I contacted the NAS, to suggest that a panel be composed of geoscientists and medical scientists to investigate the problem of minerals and health since it then appeared that the issue would in the future have a very large impact on the U.S. economy. The panel was finally formed in 1979 and I was the USGS representative to the panel and was an advisor to the panel members.
5) Member of the USGS Analytical Instrument Committee, 1987-present. The Committee submitted two major reports to the Chief Geologist, one in 1988 and other in 1990 regarding the status of the USGS instrumentation.
6) On three member Committee to investigate a case of scientific fraud within a government organization and to inspect various files relating to the question of fraud. A report was written and submitted to the Director of the USGS.
7) Co-editor of a special issue of the journal *Lithos* (vol. 26, p.1-188, 1990) titled "Alkaline Igneous Rocks and Carbonatites".
8) Member of the U.S.G.S. Promotion Panel, Branch of Igneous and Geothermal Processes, March 12-16, 1990.
9) Member of the pyroxene nomenclature committee of the International Mineralogical Association, 1982-1988.
10) Chairman of the U.S.G.S. Promotion Panel, Branch of Igneous and Geothermal Processes, April 22-27, 1991.
11) Member of the U.S.G.S. Promotion Panel, Branch of Lithospheric Processes, March 8-10, 1994.

Other committees, special assignments, and administrative duties. Name organization, group, dates, and nature of contribution.

A) Principal Investigator, in the NASA lunar science program (1969-1974).

B) I gave asbestos and health briefings and was consultant to Officials in various government agencies and other organizations, including the National Academy of Sciences, the U.S. Dept. of the Interior, The Institutes of Health, the

Environmental Protection Agency, the U.S. Congress, the National Bureau of Standards, the Bureau of Mines, etc. The subject of the briefings was on the health effects of the various types of asbestos. I also furnished information to hundreds of individuals, law firms, companies, etc. on the general subject of asbestos and asbestos related diseases. one of the main purposes of the "asbestos" briefings" was to demonstrate that it was not necessary to remove most asbestos from schools and other buildings, a concept that was finally supported by and 1990 EPA Advisory to the United States School Systems. The briefings included the following individuals and organizations:

1. (4/9/79) Briefing for lawyers, U.S. Dept. of Justice.
2. (2/24/84) Briefing for Wayne Marchant and Jed Christensen, Deputy Assistant to Assistant Sec. of the Interior.
3. (2/28/84) Briefing for Officials to the U.S. Bureau of Reclamation.
4. (5/7/84) Briefing for Dr. Ralph DeVries, Office of Science and Technology Policy.
5. (5/8/84) Briefing for Officials of the EPA and the Dept. of the Interior.
6. (5/9/84) Briefing for Officials of the General Services Administration.
7. (5/11/84) Briefing for Mr. Martin Smith of the White House staff.
8. (5/11/84) Briefing for Mr. Phil Smith and staff, National Academy of Sciences.
9. (5/22/84) Briefing for the Staff, Senate Committee on Environment and Public Works.
10. (5/25/84) Briefing for Officials of the Office of Management and Budget.
11. (6/5/84) Briefing for Secretary of Interior, William Clark and Assistant Sec. Robert Broadbent.
12. (19/84) Briefing for Under Sec. of Interior, Ann McLaughlin.
13. (7/9/84) Briefing for William Ruckelshaus, Administrator of the Environmental Protection Agency and his senior staff.
14. (7/10/84) Briefing for Dr. Bernardine Buckley and Allan Young, Office of Science and Technology Policy.
15. (7/26/84) Briefing for senior officials from Interior, EPA, GSA, OMB, CPSC, OSHA, NIOSH, HHS, the White House, and the Dept. of State.

Honors, awards, recognition, elected membership.

1) Elected Fellow, Mineralogical Society of America, in 1959.
2) Elected Fellow, Geological Society of America, in 1979.
3) Received on March 19, 1979 from the National Aeronautics and Space Administration, a Special Recognition Certificate for scientific contributions as Principal Investigator in the lunar sample program.
4) Received the U.S. Department of the Interior's Superior Service Award in 1981 for extensive and effective efforts to emphasize the geoscience prospective in public-health issues relating to mineral fibers.
5) Elected Fellow, American Association for the Advancement of Science, in 1983..
6) Received cash award, Feb. 1986, for outstanding performance in 1984-85.
7) Received on September 26, 1986 the U.S. Department of the Interior's Distinguished Service Award in recognition of outstanding contributions to the understanding of the various health effects of the different types of asbestos minerals and application of this knowledge to the national interest.
8) Received on October 30, 1990 from the Mineralogical Society of America its Distinguished Public Service Medal. This award, first given in 1990, is for contributions by the recipient to the understanding of the health effects of the asbestos minerals and in promoting a rational national policy with regard to use and control of mineral fibers in the mines, mills, and non-occupational settings.
9) Received Cash Award on August 26, 1991 for Outstanding Performance, 1990-1991
10) Cited in Who's Who in America, 1991 to present.
11) Cited in Who's Who in Medicine and Healthcare, 1995 to present

Career experience: (1945-1995)

06/45 to 09/45 Rodman for USGS (Topographic Division) surveying party, Fairhaven, Vermont.

06/48 to 09/48 Lab technician, Trace Elements Section, Geologic Div., USGS, Washington, DC.

06/49 to 09/49 Recorder for USGS (Topographic Division) surveying party, Coraopolis, PA.

06/51 to 03/52 Lab technician, Trace Elements Lab, Geologic Division, USGS, Washington, DC.

03/52 to 04/54 Lieutenant, artillery, U.S. Army, Ft. Bliss, Texas.

06/52 to 10/56 Physical Science Aid (chemistry); Trace Elements Lab, Geologic Division, USGS, Washington DC. Operated an electron microscope and electron diffraction unit to study fine-grained minerals.
10/56 to 06/61 Chemist, Crystal Chemistry Project, Geologic Div., USGS, Washington DC. Studied the crystallography of fine-grained minerals by electron diffraction - and particularly the fine-grained vanadium minerals from the Colorado Plateau. Also solved the crystal structure of various uranium-bearing minerals and compounds by single crystal X-ray diffraction techniques.

07/61 to 07/64 Physical chemist, Crystal Chemistry Project, Geologic Div., USGS, Washington DC. Solved the crystal structures of the torbernite group uranium minerals including abernathyite, meta- torbernite, various synthetic analogs of these minerals, and cesium uranyl sulfate by X-ray diffraction methods. Studied the crystal chemistry of the beryllium minerals, the crystallography of paragonite, and the mineralogy of a new uranyl carbonate. Utilized electron diffraction methods to obtain accurate unit cell constants for fine-grained minerals.

07/64 to 06/74 Physical chemist, Geologic Div., USGS. As Project Leader of the Crystal Chemistry Project an extensive program was initiated to very accurately describe the structural nature of important rock-forming silicates. Seven new structure refinements of very high accuracy were made of tremolite, cummingtonite, Mn-cummingtonite, hornblende, richterite, and two gedrites. In addition to these crystal structure studies a detailed examination was made of the phase relationships of the chain silicates including studies of the nature of exsolution in pyroxenes and amphiboles in relation to their chemical compositions, crystal structure, and crystallization history. In 1969 a study of the lunar samples was initiated and the knowledge obtained by the parallel studies of terrestrial silicates was applied to the lunar minerals. An extensive study was started (with H. Takeda and David Wones) on polytypism in micas and a unique method was developed to analyze and describe the mica polytypes. An algorithm was invented to described the crystallography and crystal structure of all possible mica polytype crystal structures. The petrographic relationships of the minerals in the metamorphic rocks of the Gouverneur talc district, NY was completed.

07/74 to 04/77 Physical chemist, Mineral Investigations Project, Geologic Div., USGS, Washington DC. Investigations (with Peter Robinson, Howard Jaffe, and Gordon Nord) were started on metamorphic amphiboles from western Massachusetts. The anthophyllite- gedrite solvus was described for the first time and the solid solution series between these two minerals was delineated for the naturally occurring minerals. The crystal structures of biotites that were reduced and then oxidized (addition and removal of hydrogen) were determined. This research led to a much clearer understanding of the role of hydrogen in the formation of H-bearing rock-forming minerals.

07/77 to 10/82 Physical chemist, Project leader Asbestiform Minerals Project, Geologic Div., USGS, Washington DC. Entered into a long term study of the geological occurrence of the various types of asbestos minerals, their abundances, their use and distribution in the environment, their health effects on man and in experimental animals, the Federal regulatory processes related to asbestos control and mitigation, the nature of the Federal definition of asbestos as it might relate to mining activities other than asbestos mining, and the social and economic consequences of State and Federal actions. In 1978 (reference no. 86 in my bibliography) I was the first to show the very strong correlation between exposure to crocidolite asbestos and a pronounced negative correlation between this disease and exposure to the very common chrysotile asbestos. This led to proposing the "fiber hypothesis" for asbestos-related disease and is the foundation for the conviction that exposure to chrysotile asbestos in schools and other non-occupational settings presents essentially zero health risk. To bring to the attention of the scientific community, the public, and political leaders I have given approximately 66 lectures and briefings (see 12b., 12c.) on the various aspects of the asbestos controversy. In addition to these project activities I serve as the USGS Asbestos Commodity Specialist. Work was continued on extending the "optimal phase boundary" concept to studies of the pyroxenes. Careful measurement of the orientation of pyroxene exsolution lamellae within the host pyroxenes were made thus establishing temperature-composition-orientation relationships for these minerals. These observations present a method of formulating new geothermometers and barometers.

10/82 to present Physical chemist, Geologic Div., USGS, Washington DC. From 1982 to 1986 was a principal investigator, National Acid Precipitation and Assessment Program, also project leader of the Acid Rain Project (1982-1986), the Rock-forming Silicates Project (1982-1989), and the Alkaline Rocks and Carbonatites Project (1983-1992).

The acid rain research involved a cooperative effort with a consortium of about 20 investigators from several different research agencies. My first critical task was to take responsibility for selecting and procuring the various marbles and limestones that would used in our materials effects studies. Visits to the quarry site and cutting mills were made while the stone was being extracted from the quarries and cut into sized slabs and briquettes. Samples were placed at six stations where various atmospheric parameters (such as rainfall amount and chemistry, pH, and temperature) were continually monitored. The relationships between these parameters, the amount of dissolution of the marble and limestone samples, and

the mineralogical changes were established. Characterization of the samples (with Elaine McGee and Daphne Ross) was accomplished by chemical analysis using electron microprobe, light optical, scanning electron microscopy, and X-ray diffraction techniques

The Alkaline Rocks and Carbonatites Project (previously titled the Rock-forming Silicates Project) concentrated its research on the alkaline igneous rocks of the Magnet Cove igneous complex and the adjacent mineralized country rock shales and quartzites. I initially set up the Project's objectives; the immediate applied interest being the mode of origin of the titanium, vanadium, and niobium deposits in the mineralized country rocks. Marta Flohr joined me in this research program and has taken the lead in much of the work. Samples were collected in seven one week field trips. Also, a detailed map was made (in cooperation with the Arkansas Geological Commission) of the most important locality for sampling a large variety of fresh alkaline igneous rocks as well as contact metamorphosed country rocks. A complete geochemical and petrological examination was made of the igneous rocks and included a study of the secondary minerals formed on cooling from the primary melt temperatures. Various metasomatic processes, including metasomatism by later-stage Ca- and Na-rich carbonatitic fluids and low temperature alteration (lateritic processes), also caused replacement of previously formed minerals and released metals that were later deposited in the adjacent country rocks. Some of the important results of this investigation are given in section 15.

In this period I continued serving as the USGS commodity specialist on asbestos and also remained involved part time (30 percent) in various aspects of the asbestos controversy and in particular to how the Federal definition of asbestos is now affecting large segments of the U.S. mining industry not involved in asbestos mining (see section 15).

In 1992 and 1993 I became more and more involved with the issue of minerals and health due to an increased Division interest in environmental hazards and due to the fact that the common mineral quartz is now on the Federal carcinogen list. In 1992, I gave five invited talks on the health effects of mineral dusts and was engaged for several hours per week on the telephone responding to questions concerning this same subject.

In 1993 I spent nearly full time on health issues including preparing two long review papers on the health effects of mineral dusts for Mineralogical Society of America and Society of Economic Geologists short courses. In addition, I prepared three other papers and gave five talks on this general subject and started an investigation of the possible health effects that might be generated by dusts and water from the asbestos Superfund sites within the New Idria serpentinite of California.

Career experience since retirement from the U.S. Geological Survey

January 1995 to December 1999. Scientist Emeritus, U.S. Geological Survey.

January 4, 1993 to 1999. Affiliated with The Science and Environmental Policy Project, 4084 University Drive, Fairfax City, VA 22030. Engaged in studies of public policy issues pertaining to mineral and geochemical effects on human health and global warming issues.

March 1, 1995 to present. Appointed Senior Scientist in the Environmental Effects Laboratory, Institute of Applied Sciences at Brooklyn College of the City University of New York. Research is directed towards analyzing various mine dusts and evaluating their possible effects on human health. Examination was made of possible asbestos occurrence and asbestos health risk in iron ore, vermiculite, and asbestos mines in the United States and Cyprus.

Bibliography

Published Reports:

1. Dwornik, E.J. and Ross, Malcolm, 1955, Application of the electron microscope to mineralogic studies: American Mineralogist, v. 40, p. 261-274.

2. Ross, Malcolm and Christ, C.L., 1958, Mineralogical application of electron diffraction. I. Theory and techniques: American Mineralogist, v. 43, p. 1157-1178.

3. Rose, H.J., Jr., Blade, L.V. and Ross, Malcolm, 1959, Earthy monazite at Magnet Cove, Arkansas: American Mineralogist, v. 43, p. 995-998.

4. Ross, Malcolm, 1959, Mineralogical applications of electron diffraction. II. Studies of some vanadium minerals of the Colorado Plateau: American Mineralogist, v. 44, p. 322-341.

5. Fahey, J.J., Ross, Malcolm and Axelrod, J.M., 1960, Loughlinite, a new hydrous sodium magnesium silicate: American Mineralogist, v. 45, p. 270-281.

6. Ross, Malcolm and Evans, H.T., Jr., 1960, The crystal structure of cesium biuranyl trisulphate, $Cs_2(UO_2)_2(SO_4)_3$: Journal of Inorganic Nuclear Chemistry, v. 15, no. 3/4, p. 338-351.

7. Straczek, J.A., Horan, A., Ross, Malcolm and Warshaw, C.M., 1960, Studies of the manganese oxides. I.V. Todorokite: American Mineralogist, v. 45, p. 1174-1184.

8. Ross, Malcolm, 1962, The crystallography, crystal structure, and crystal chemistry of various minerals and compounds belonging to the torbernite mineral group: Ph.D. Thesis, Harvard University, Cambridge, Massachusetts, 179 p.

9. Ross, Malcolm, 1963, The crystallography of meta-autunite (I): American Mineralogist, v. 48, p. 1389-1393.

10. Ross, Malcolm, 1963, The crystal chemistry of beryllium: Professional Paper 468, 30 pp.

11. Zen, E-an, Ross, Malcolm and Bearth, Peter, 1964, Paragonite from Tasch Valley near Zermatt, Switzerland: American Mineralogist, v. 49, p. 183-190.

12. Ross, Malcolm and Evans, Howard T., Jr., 1964, Studies of the torbernite minerals (I): The crystal structure of abernathyite and the structurally related compounds $NH_4(UO_2AsO_4)\cdot 3H_2O$ and $KH(UO_2AsO_4)_2\cdot 7H_2O$: American Mineralogist, v. 49, p. 1578-1602.

13. Ross, Malcolm, Evans, H.T., Jr. and Appleman, D.E., 1964, Studies of the torbernite minerals (II): Structure of metatorbernite: American Mineralogist, v. 49, p. 1603-1621.

14. Meyrowitz, R., Ross, D.R. and Ross, Malcolm, 1964, A new uranyl tricarbonate $K_2Ca_3(UO_2)_2(CO_3)_6\cdot 9\text{-}10\ H_2O$: U.S. Geological Survey Professional Paper 501-B, p. D64-D69.

15. Ross, Malcolm and Evans, H.T., Jr., 1965, Studies of the torbernite minerals (III): Role of the interlayer oxonium, potassium and ammonium ions, and water molecules: American Mineralogist, v. 50, p. 1-12.

16. Ross, Malcolm, 1965, The torbernite minerals as model compounds for the hydrous layer silicates: Clays and Clay Minerals, v. 13, p. 65-71.

17. Ross, Malcolm, 1965, Precision unit-cell parameter determination of layer silicates by use of electron and X-ray diffraction techniques: Clays and Clay Minerals, v. 13, p. 105-106 (extended abstract).

18. Ross, Malcolm, Takeda, H. and Wones, D.R., 1965, Systematic description and identification of mica polytypes: Science, v. 151, p. 191-193.

19. Skinner, B.J., Jambor, J.L. and Ross, Malcolm, 1967, McKinstryite, a new copper-silver sulfide: Economic Geology, v. 61, p. 1383-1389.

20. Ross, Malcolm, 1968, Fourier transform analysis: (1) X-ray diffraction effects of finite montmorillonite and mica crystals: Zeits. Kristallographie, v. 126, p. 80-97.

21. Ross, Malcolm, Smith, W.L. and Ashton, W.H., 1968, Triclinic talc and associated amphiboles from the Gouverneur Mining District, New York: American Mineralogist, v. 53, p. 751-769.

22. Ross, Malcolm, Papike, J.J. and Weiblen, P.W., 1968, Exsolution in clinoamphiboles: Science, v. 159, p. 1099-1102.

23. Ross, C.S., Bergquist, H., Fahey, J.J. and Ross, Malcolm, 1968, Alunites in the Upper Cretaceous sedimentary rocks, North-Central Texas: Journal of Sedimentary Petrology, v. 38, no. 4, p. 1155-1165.

24. Allen, V.T., Fahey, J.J. and Ross, Malcolm, 1969, Kaolinite and anauxite in the Ione Formation, California: American Mineralogist, v. 54, p. 206-11.

25. Robinson, Peter, Jaffe, Howard, Klein, Cornelis, Jr. and Ross, Malcolm, 1969, Equilibrium coexistence of three amphiboles: Contributions to Mineralogy and Petrology, v. 22, p. 248-258.

26. Ross, Malcolm, Papike, J.J. and Shaw, K. Weir, 1969, Exsolution textures in amphiboles as indicators of subsolidus thermal histories: Mineralogical Society of America Special Paper #2, p. 275-299.

27. Papike, J.J., Ross, Malcolm and Clark, J.R., 1969, Crystal-chemical characterization of clinoamphiboles based on five new structure refinements: Mineralogical Society of America Special Paper #2, p. 117-136.

28. Ross, Malcolm, Bence, A.E., Dwornik, E.J., Clark, J.R. and Papike, J.J., 1970, Lunar clinopyroxenes: Chemical composition, structural state, and texture: Science, v. 167, p. 628-630.

29. Ross, Malcolm, Bence, A.E., Dwornik, E.J., Clark, J.R. and Papike, J.J., 1970, Mineralogy of the lunar clinopyroxenes, augite and pigeonite: Proceedings of the Apollo 11 Lunar Science Conference, Geochimica et Cosmochimica Acta, Supplement 1, v. 1, p. 839-848.

30. Rooney, T.R., Riecher, R.E. and Ross, Malcolm, 1970, Deformation twins in hornblende: Science, v. 169, p. 173-175.

31. Prewitt, C.T., Papike, J.J. and Ross, Malcolm, 1970, Cummingtonite, a reversible, nonquenchable transition from $P2_1/m$ to C2/m symmetry: Earth and Planetary Science Letters, v. 8, p. 448-450.

32. Papike, J.J. and Ross, Malcolm, 1970, Gedrites: Crystal structures and intracrystalline cation distributions: American Mineralogist, v. 55, p. 1945-1972.

33. Clark, J.R., Ross, Malcolm and Appleman, D.E., 1971, Crystal chemistry of lunar pigeonite: American Mineralogist, v. 56, p. 888-908.

34. Robinson, P., Ross, Malcolm and Jaffe, H.W., 1971, Composition of the anthophyllite-gedrite series, gedrite-hornblende comparisons and the anthophyllite-gedrite solvus: American Mineralogist, v. 56, p. 1005-1041.

35. Robinson, P., Jaffe, H.W., Ross, Malcolm and Klein, C., 1971, Orientation of exsolution lamellae in clinopyroxenes and clinoamphiboles: consideration of optimal phase boundaries: American Mineralogist, v. 56, p. 909-939.

36. Dowty, E., Ross, Malcolm and Cuttitta, F., 1972, Fe2+-Mg site distribution in Apollo 12021 clinopyroxenes: Evidence for bias in Mossbauer spectra, and relation of ordering to exsolution: Third Lunar Science Conference Proceeds, MIT Press, v. I, p. 481-492.

37. Ross, Malcolm, 1972, Book Review: The Electron-Optical Investigation of Clays by J.A. Gard, Mineralogical Society, London, 1971: in Economic Geology, v. 67, no. 7, 1972, p. 986-987.

38. Ross, Malcolm, Huebner, J.S. and Dowty, E., 1973, Delineation of the one atmosphere augite-pigeonite miscibility gap for pyroxenes from lunar basalt 12021: American Mineralogist, v. 58, p. 619-635.

39. Virgo, D. and Ross, Malcolm, 1973, Pyroxenes from Mull andesites: Carnegie Institute Yearbook 72, p. 535-540.

40. Jaffe, H.W., Robinson, P., Tracy, R.J. and Ross, Malcolm, 1975, Orientation of pigeonite exsolution lamellae in metamorphic augite: Correlation with composition and calculated optimal phase boundaries: American Mineralogist, v. 60, p. 9-28.

41. Huebner, J.S., Ross, Malcolm and Hickling, N., 1975, Significance of exsolved pyroxenes from lunar breccia 77215: Proceedings of the Sixth Lunar Science Conference, Geochimica Cosmochimica Acta, supplement 6, v. 1, p. 529-546.

42. Takeda, H. and Ross, Malcolm, 1975, Mica polytypism: Dissimilarities in the crystal structures of coexisting 1M and 2M1 biotite: American Mineralogist, v. 60, p. 1030-1040.

43. Ross, Malcolm, 1976, Chemical, structural, and geochemical relationships between pyroxenes, amphiboles, and asbestos: in Electron Microscopy of Microfibers, I.M. Asher and P.O. McGrath, Eds., Proceedings of the first FDA Office of Science Summer Symposium, p. 34-35.

44. Robinson, P., Ross, Malcolm, Nord, G.L., Jr., Smyth, J.R. and Jaffe, H.W., 1977, Exsolution in augite and pigeonite:

fossil indicators of lattice parameters at high temperature and pressure: American Mineralogist, v. 62, p. 857-873.

45. Ross, Malcolm, 1978, The "asbestos" minerals: Definitions, descriptions, modes of formation, physical and chemical properties, and health risk to the mining community: Workshop on Asbestos: Definitions and Measurement Methods, National Bureau of Standards special publication 506, p. 49-63.

46. Ross, Malcolm and Huebner, J.S., 1979, Temperature-composition relationships between naturally occurring augites, pigeonite, and orthopyroxene at one bar pressure: American Mineralogist, v. 64, p. 1133-1155.

47. Ross, Malcolm, 1980, Book Review (Asbestos, Vol. I, Properties, Applications and Hazards by L. Michaels and S.S. Chissisk, John Wiley, N.Y., 553 pp., 1979): in EOS, no. 27, July 1, 1980, p. 516.

48. Ross, Malcolm, 1981, The Geological Occurrences and Health Hazards of Amphibole and Serpentine Asbestos: in Reviews in Mineralogy, Vol. 9A, Amphiboles and Other Hydrous Pyriboles - Mineralogy, D.R. Veblen, Ed., Mineralogical Society of America, Washington, D.C., Nov., 1981, 279-323.

49. Ross, Malcolm, 1981, Book Review (Asbestos: Final Report of the Advisory Committee, Vol. I, pp. 100, Vol. 2: Papers Prepared for the Advisory Committee, pp. 103. Committee Chairman, W. Simpson, Health and Safety Commission of Great Britain. Her Majesty's Stationery Office, 40 High Holborn): in Journal of Applied Cryst., v. 14, p. 77.

50. Ross, Malcolm, 1982, A Survey of Asbestos-Related Disease in Trades and Mining Occupations and in Factory and Mining Communities as a Means of Predicting Health Risks of Non-Occupational Exposure to Fibrous Minerals: U.S. Geological Survey Open-file Report 82-745, 41 pp.

51. Ross, Malcolm, 1982, Asbestos, Health, and Society: Levels of Exposure of the General Population from Natural Sources: in Proc. World Symposium on Asbestos, May 25-27, 1982, Canadian Asbestos Information Centre, Montreal, p. 288-294.

52. Ross, Malcolm and Clifton, R.A., 1983, Arizona Long Fiber, Low Iron Chrysotile Asbestos as a Government Stockpile Source. April 7, 1983: Thru W.P. Pendley, Deputy Assistant Secretary for Energy and Minerals, Dept. Interior--for the Federal Emergency Management Agency.

53. Ross, Malcolm and Knab, L., 1984, Procurement and Chemical Characterization of Limestone and Marble for Materials Effects Testing, Acid Deposition Program: in Materials Effects Test Group (G) and Assessments and Policy Analysis Test Program, Raleigh NC, 14-17 Feb., 1984, p. G29-G36.

54. Ross, Malcolm, 1984, A Survey of Asbestos-Related Disease in Trades and Mining Occupations and in Factory and Mining Communities as a Means of Predicting Health Risks of Non-Occupational Exposure to Fibrous Minerals: in Definitions for Asbestos and Other Health-Related Silicates, American Society for Testing Materials, STP Report No. 834, B. Levadie, Ed., p. 51-104.

55. Ross, Malcolm, Kuntze, R.A. and Clifton, R.A., 1984, A Definition for Asbestos: in Definitions for Asbestos and Other Health-Related Silicates, American Society for Testing Materials, STP Report No. 834, B. Levadie, Ed., p. 139-147.

56. Ross, Malcolm, 1984, A Definition for Talc: in Definitions for Asbestos and Other Health-Related Silicates: in Definitions for Asbestos and Other Health-Related Silicates, American Society for Testing Materials, STP Report No. 834, B. Levadie, Ed, p. 193-197.

57. Ross, Malcolm and Knab, L., 1984, Selection, Procurement, and Description of Salem Limestone Samples Used to Study Effects of Acid Rain: National Bureau of Standards Report NBSIR 84-2905, p. 1-17.

58. Flohr, Marta J.K. and Ross, Malcolm, 1985, Nepheline syenite, quartz syenite, and ijolite from the Diamond Jo Quarry, Magnet Cove, Arkansas: in Alkalic Rocks and Carboniferous Sandstones Quachita Mountains - New Perspectives. A Guidebook for the GSA Regional Meeting, Fayetteville, Arkansas April 16-18, 1985, p. 63-75.

59. Ross, Malcolm, 1985, Description, selection, and procurement of Shelburne Marble samples used to study effects of acid rain: U.S. Geological Survey Open-File Report 85-594, 15 pp.

60. Ross, Malcolm, 1985, Book Review. Micas, Reviews in Mineralogy, v. 13, edited by S.W. Bailey, Mineralogical Society of America, Washington, DC, 1984, 584 pages, paperback: in Clays and Clay Minerals, v. 33, p. 367-368.

61. McGee, Elaine S. and Ross, Malcolm, 1986, Applications of mineralogic and petrographic techniques to material damage: in Materials Effects Task Group VII Peer Review Summaries, 6-11 April 1986, Durham, NC, North Carolina State University Acid Deposition Program, NAPAP, p. 167-174.

62. Ross, Malcolm, 1987, Minerals and health: the asbestos problem: in the Proceedings of the 21st Forum on the Geology of Industrial Minerals, H.W. Peirce, Ed., Special Paper 4, 1987, Arizona Bureau of Geology and Mineral Technology, Tucson, AZ, p. 83-89.

63. Ross, Malcolm, 1988, The occurrence and dangers of amphibole and chrysotile asbestos in our environment: in the Proceedings of the Conference on Asbestos in Play Sand - Tremolite in Play Sand: Public Health Implications, C. E. Gilbert, Ed., NE Regional Environmental Public Health Center, Amherst, MA.

64. Morimoto, N., Ferguson, A.K., Ginzburg, I.V., Ross, Malcolm, Seifert, F.A. and Zussman, J., 1988, Nomenclature of pyroxenes: American Mineralogist, v. 73, p. 1123-1133.

65. Skinner, H.C.W., Ross, Malcolm, and Frondel, C., 1988, Asbestos and Other Fibrous Materials: Oxford University Press, NY, 204 pp.

66. Flohr, Marta J.K. and Ross, Malcolm, 1989, Alkaline igneous rocks of Magnet Cove, Arkansas: Metasomatised ijolite xenoliths from Diamond Jo Quarry, American Mineralogist, v. 74, p. 113-131.

67. Ross, Malcolm, McGee, E.S., and Ross, D.R., 1989, Chemical and mineralogical effects of acid deposition on Shelburne Marble and Salem Limestone test samples placed at four NAPAP weather monitoring sites: American Mineralogist, v. 74, p. 367-383.

68. McGee, E.S. and Ross, Malcolm, 1989, Mineralogical characterization of stone deterioration. in Materials and Cultural Resources Effects, Task Group VII Research Summaries, 22-27 January 1989, Charleston, SC, National Acid Precipitation Assessment Program (NAPAP). Organized by N. Carolina State U. Atmospheric Research Program and Science Policy Associates, Inc., p. 173-184.

69. Skinner, H.C.W. and Ross, Malcolm, 1989, Summary of the Geological Society of America Forum on "Fibrous minerals, mining and disease. The Professional Geologist, v. 26, no. 3, March 1989, p. 3-4.

70. Ross, Malcolm, 1990, Hazards associated with fibrous minerals. U.S. Geological Survey Circular 1033, in Proceedings of the U.S. Geological Survey Workshop on Environmental Geochemistry, Bruce Doe, Ed. p. 175-176.

71. Flohr, Marta J.K. and Ross, Malcolm, 1990, Alkaline igneous rocks of Magnet Cove, Arkansas: Mineralogy and geochemistry of syenites. Lithos, v. 26, p. 67-98.

72. Ross, Malcolm, 1990, Book Review. Classical Marble: Geochemistry, Technology, Trade, N. Herz and Marc Waelkens, Eds., Kluwer Academic Publishers, London, 1988, 482 p.: in American Mineralogist, v. 75. p. 721-722.

73. Langer, A.M., Nolan, R.P., and M. Ross, 1990, Letter to the Editor, Asbestos Policy. Science, v. 249, p. 1485.

74. Ross, Malcolm, 1991, Acceptance of the Distinguished Public Service Medal for 1990: American Mineralogist, v. 76, p 1744-1745.

75. Ross, Malcolm, 1991, Suspect Minerals and human health: A commentary. The Lattice (Mineralogical Society of America Newsletter), v. 7. No. 1, p. 10-11.

76. Ross, Malcolm, 1991, "Carcinogenic" minerals and human health: A commentary. The Cross Section, U.S. Geological Survey Pub., v. 22, no. 3, p. 17-21.

77. Ross, Malcolm, Flohr, Marta J.K., and Ross, Daphne, 1992, Crystalline solution series and order-disorder within the natrolite mineral group: American Mineralogist, v. 77, p. 685-703.

78. Ross, Malcolm, Nolan, R.P., Langer, A.M., and Cooper, W.C., 1993, Health effects of mineral dusts other than asbestos. in Reviews in Mineralogy, v. 28, Health Effects of Mineral Dusts, G.D. Guthrie and B.T. Mossman (eds.), Mineralogical Society of America, Washington, DC. p 361-407.

79. Ross, Malcolm, and Drew, L.W., 1993, Directions (discussion on geology and the environment). Nonrenewable Resources, v. 2, p. 257-261.

80. Ross, Malcolm and Skinner, H.C.W., 1993, Geology and Health. Geotimes, v. 39, p. 10-12.

81. Skinner, H.C.W. and Ross, Malcolm, 1994, Minerals and cancer. Geotimes, v. 39, p. 13-15.

82. Wilson, Richard, Langer, A.M., Nolan, R.P., Gee, J.B., and Ross, Malcolm, 1994, Asbestos in New York City schools, public policy: Is there a scientific basis? Regulatory Toxicology and Pharmacology, v. 20, p. 161-169.

83. Ross, Malcolm, 1995, The effect of regulations on asbestos and other designated mineral carcinogens on the vitality of the industrial minerals industries: Proceedings of the 28th Forum on the Geology of Industrial Minerals, May 3-8, 1992, B.J. Walker, Ed. Circular C-46, West Virginia Geological and Economic Survey, Morgantown, West Virginia, p. 149-151.

84. Takeda, Hiroshi and Ross, Malcolm, 1995, Mica polytypism: Identification and origin. American Mineralogist, v. 80, p. 715-724.

85. Ross, Malcolm, 1995, Environmental pollution. McGraw-Hill Yearbook of Science and Technology-1996, McGraw-Hill, Inc., New York`, p. 102-105.

86. Ross, Malcolm, 1995, The perils of risk-free cancer policy. EPA Watch, v. 4, no. 3, 4-6.

87. Ross, Malcolm, 1995, Failed policy on asbestos abatement. The Washington Times, October 8, p. B3.

88. Ross, Malcolm, 1995, The schoolroom asbestos abatement program: a public policy debacle. Environmental Geology, v. 26, p. 182-188.

89. Ross, Malcolm, 1996, Did risk reduction backfire in space?. The Washington Times, January 28, p. B3.

90. Ross, Malcolm, 1997, Asbestos. McGraw-Hill Encyclopedia of Science and Technology.

91. Coombs, D.S., Alberti, A., Armbruster, T., Artioli, G., Collella, C., Galli, E., Grice, J.D., Liebau, F., Mandarino, J.A., Minato, H., Nickel, E.H., Passaglia, E., Peacor, D.R., Quartieri, S., Rinaldi. R., Ross, Malcolm, Sheppard,, R.A., Tillmanns, E., and Vezzalini, G., 1998, Recommended nomenclature for zeolite minerals: Report of the subcommittee on zeolites of the International Mineralogical Association, Commission on New Minerals and Mineral Names. The Canadian Mineralogist, v. 35, p 1571-1606.

92. Ross, Malcolm, and Post, J.E., 1998, Memorial of Daniel E. Appleman. American Mineralogist, v. 83, p. 1133-1135.

93. Ross, Malcolm, 1999, The health effects of mineral dusts. in Reviews in Economic Geology, v. 6A, The Environmental Geochemistry of Mineral Deposits. Part A: Processes, Techniques, and Health Issues, Society of Economic Geologists, G.S. Plumlee and M.J. Logsdon, Eds., p. 339-356.

94. Ross, Malcolm, 1999, Presentation of the Distinguished Public Service Medal for 1998 to Daniel E. Appleman. American Mineralogist, v. 84, p. 1205-1206.

95. Evans, H.T., Jr., Konnert, Judith A., and Ross, Malcolm, 2000, The crystal structure of tetranatrolite from Mont Saint-Hilaire, Québec, and its chemical and structural relationship to paranatrolite and gonnardite: American Mineralogist, v. 85, p. 1808-1815.

96. Ross, Malcolm, 2000, Howard T. Evans (1919-2000). American Crystallographic Association - Newsletter, Spring 2000, p 20-21.

97. Ross, Malcolm and Nolan, R.P., 2000, Asbestos. McGraw-Hill Encyclopedia of Science and Technology, 9th Edition, New York, NY.

98. Ross, Malcolm, 2000, The Asbestos abatement in United States schools and other buildings. Newsletter of the Mineralogical Association of Canada, No. 63, Dec. 2000, p. 17-18.

99. Nolan, R.P., Langer, A.M., Ross, Malcolm, Wicks, F.J., and Martin, R.F. (Eds.) 2001, Health Effects of Chrysotile Asbestos: Contribution of Science to Risk Management Decisions. The Canadian Mineralogist Special Publication 5, 304 pp.

100. Ross, Malcolm and Virta, R.L., 2001, Occurrence, production and uses of asbestos. in Health Effects of Chrysotile Asbestos: Contribution of Science to Risk Management Decisions, .Nolan, R.P., Langer, A M., Ross, M., Wicks, F.J., and Martin, R.F., Eds. The Canadian Mineralogist Special Publication 5, p. 79-88.

101. Ross, Malcolm and Nolan, Robert, 2003, History of asbestos discovery and use and asbestos-related disease in context with the occurrence of asbestos within ophiolite complexes. in Ophiolite concept and evolution of geologic thought, Dilek, Y. and Newcomb, S., Eds. Geological Society of America Special Publication 373, p. 447-470.

102. Nolan, R.P., Ross, M., Nord, G.L., Axten, C.W., Osleeb, J.P., and Wilson, R., 2003, Aassessment of risk of asbestos-related cancer based on analysis of air and settled dust samples from the 9/11 attack on the World Trade Center Complex. International Conference on "Saftey and health in the production and use of asbestos and other fibrous materials", June 3-7, 2002, Ekaterinburg, Sverdlovsk Region, Russian Federation, in Collected reports and speeches, NPO Asbestos Association, 2003, p. 99-128.

103. Morse, S.A. and Malcolm Ross, 2004, Kiglapait mineralogy IV: The augite series. American Mineralogist, v.89, p. 1380-1395.

104. Nolan, R.P., Ross, Malcolm, Nord, G.L., Axten, C.W., Osleeb, J.P., Domnin, S.G., Price, Bertram, and Wilson, Richard, 2005, Risk assessment for asbestos-related cancer from the 9/11 attack on the World Trade Center. Journal of Occupational and Environmental Medicine, v. 47, no 8, p. 817-825.

105. Nolan, R.P, Ross, Malcolm, Nord, G.L, Raskina, M., Phillips, J.I., Murray, J., and Gibbs, G.W., 2005, Asbestos fiber-type and mesothelioma risk in the Republic of South Africa. Clay Science, International Clay Conference, Tokyo, Japan (in press).

Published abstracts

1. Ross, Malcolm, 1955, Interpretation of transmission electron diffraction spot patterns of certain monoclinic crystals: Journal of Applied Physics, v. 26, p. 1391.

2. Ross, Malcolm and Evans, H.T., Jr., 1959, Crystal structure of abernathyite: Geological Society of America Bulletin 70, p. 1666.

3. Ross, Malcolm and Evans, H.T., Jr., 1959, The crystal structure of CS2(UO2)2(SO4)3: American Crystallographic Association Program, p. 27-28.

4. Ross, Malcolm and Evans, H.T., Jr., 1962, The crystal structure and crystal chemistry of various members of the metatorbernite group: American Mineralogist, v. 47, p. 202.

5. Ross, Malcolm, 1964, The torbernite minerals as model compounds for the layer silicates: First Annual Clay Meeting, Madison, Wisconsin.

6. Ross, Malcolm, 1964, Precision unit-cell parameter determination of layer silicates by use of electron and X-ray diffraction powder techniques: First Annual Clay Meeting, Madison, Wisconsin.

7. Ross, Malcolm and Wones, D.R., 1965, Polytypism in biotites: American Mineralogist, v. 50, p. 291.

8. Ross, Malcolm, Wones, D.R. and Takeda, H., 1966, Systematic description and identification of mica polytypes: American Mineralogist, v. 51, p. 270-271.

9. Ross, Malcolm, 1966, Fourier transform analysis: (1) X-ray diffraction effects by finite montmorillonite and mica crystals: 15th Annual Clay Minerals Conference, October 10-13, 1966, Mellon Institute, Pittsburgh, PA.

10. Ross, Malcolm, 1967, Characterization of crystalline solutions by Fourier transform methods: Program, 48th Annual Meeting, American Geophysical Union, April 17-20, 1967, Washington, DC.

11. Allen, V.T., Fahey, J.J. and Ross, Malcolm, 1968, Kaolinite and "annauxite" in the Ione Formation, California: Geological Society of America abstracts for 1967.

12. Ross, Malcolm, Papike, J.J. and Weiblen, P.W., 1968, Exsolution in clinoamphiboles: Geological Society of America abstracts for 1967.

13. Ross, Malcolm and Papike, J.J., 1968, Exsolution and recrystallization textures in clinoamphiboles as indicators of subsolidus thermal histories: Transaction AGU, v. 49, p. 340.

14. Papike, J.J. and Ross, Malcolm, 1970, Gedrites, crystal structures and intercrystalline cation distributions: American Mineralogist, v. 55, p. 304.

15. Prewitt, C.T., Papike, J.J. and Ross, Malcolm, 1970, Cummingtonite, a reversible, nonquenchable transition from P21/m to C2/m symmetry: American Mineralogist, v. 55, p. 305.

16. Robinson, P., Ross, Malcolm and Jaffe, H., 1970, The composition field of anthophyllite and the anthophyllite miscibility gap: American Mineralogist, v. 55, p. 307.

17. Ross, Malcolm, 1970, Chemical reactions in pyroxene crystals: American Mineralogist, v. 55, p. 310.

18. Clark, J.R., Ross, Malcolm, Appleman, D.E., Papike, J.J. and Bence, A.E., 1970, Fe/Mg distribution in lunar pigeonite: Transactions of the American Geophysical Union, v. 51, p. 436.

19. Takeda, H. and Ross, Malcolm, 1970, Computer method of identifying complex mica polytypes: International Mineralogical Association, IAGOD, 1970 Meetings, Tokyo, Japan, p. 177.

20. Ross, Malcolm, Huebner, J.S. and Dowty, E., 1971, Delineation of an augite-pigeonite solvus at zero pressure by homogenizing single clinopyroxene crystals from lunar rock 12021: 1971 Lunar Science Conference, Houston, Texas, p. 146-147.

21. Ross, Malcolm, Huebner, J.S. and Dowty, E., 1971, Delineation of the one atmosphere augite-pigeonite solvus and orthopyroxene-pigeonite reaction curve, and pigeonite melting relations: Geological Society of America 1971 annual meetings, abstracts with programs, p. 690.

22. Huebner, J.S. and Ross, Malcolm, 1972, Phase relations of lunar and terrestrial pyroxenes at one atm.: Lunar Science - III, revised abstracts of papers presented at the Third Lunar Science Conference, Houston, 10-13, January 1972 (C. Watkins, Ed.), p. 410-412.

23. Steward, D.B., Ross, Malcolm, Morgan, B.A., Appleman, D.E., Huebner, J.S. and Commeau, R.F., 1972, Mineralogy and Petrology of Lunar Anorthosite 15415: Lunar Science - III, revised abstracts of papers presented at the Third Lunar Science Conference, Houston, 10-13, January 1972 (C. Watkins, Ed.), p. 726-728.

24. Takeda, H. and Ross, Malcolm, 1972, Structural studies of coexisting mica polytypes and pyroxene polymorphs. Abstracts for International Union of Crystallography, August 1972 Kyoto Meeting: Acta Crystallographic, v. A28, part 54, p. S-117.

25. Ross, Malcolm, 1972, Polytypism, polymorphs, and subsolidus reactions in rock-forming micas, amphiboles, and pyroxenes. Abstract for International Union of Crystallography, August 1972 Kyoto Meeting: Acta Crystallography, v. A28, part 54, p. S-116.

26. Ross, Malcolm, Robinson, P. and Jaffe, H.W., 1972, Pigeonite lamellae in Bushveld augite sequentially exsolved on phase boundaries at angles controlled by differential thermal contraction: GSA 1972 annual meeting, abstracts with programs, v. 4, no. 7, p. 644-645.

27. Jaffe, H.W., Robinson, P., Tracy, R.J. and Ross, Malcolm, 1972, Correlation of angles of pigeonite exsolution lamellae, composition, and calculated optimal phase boundaries in metamorphic augite: GSA 1972 annual meeting, abstracts with programs, v. 4, no. 7, p. 552.

28 Huebner, J.S., Ross, Malcolm and Turnock, A.C., 1972, Phase diagrams for pyroxene compositions: GSA 1972 annual

meeting, abstracts with programs, v. 4, no. 7, p. 547.

29. Ross, Malcolm, Huebner, J.S. and Hickling, N., 1973, Delineation of the orthopyroxene-pigeonite transition and its bearing on pyroxene phase relations in lunar rocks: Lunar Science IV (Chamberlain and Watkins, Eds.), p. 637-639.

30. Huebner, J.S., Ross, Malcolm, Hickling, N. and Turnock, A.C., 1973, Partial melting of pyroxenes and the origin of Mare basalts: Lunar Science IV (Chamberlain and Watkins, Eds.), p. 397-399.

31. Jaffe, H.W., Robinson, P., Tracy, R.J. and Ross, Malcolm, 1974, Ortho ferrosilite in Adirondack Gneiss: Transactions of American Geophysical Union, v. 55, p. 468.

32. Robinson, P., Jaffe, H.W. and Ross, Malcolm, 1974, Dimensional fit as the control on orientation angles of exsolution lamellae in pyroxenes and amphiboles: International Mineralogical Association, 9th General Meeting, Berlin - Regensburg, 1974, p. 83.

33. Ross, Malcolm, 1974, Geology, asbestos, and health: Environmental Health Perspectives, v. 9, p. 123-124.

34. Takeda, H. and Ross, Malcolm, 1974, Dissimilarities in the crystal structure of 1M and 2M1 biotite polytypes coexisting in a rhyodacite lava flow: Geological Society of America 1974 annual meetings, abstracts with programs, v. 6, no. 7, p. 980-981.

35. Ross, Malcolm and Huebner, J.S., 1975, Estimation of the minimum temperature for coexistence of orthopyroxene, pigeonite, and augite and its application to prediction of temperatures of crystallization of lunar pyroxenes: Lunar Science VI, Sixth Lunar Science Conference, March 1975, p. 689-691.

36. Huebner, J.S., Ross, Malcolm and Hickling, N.L., 1975, Cooling history and significance of exsolved pyroxene in lunar noritic breccia 77215: Lunar Science VI, Sixth Lunar Science Conference, March 1975, p. 408-409.

37. Robinson, P., Ross, Malcolm, Smyth, J.R., Nord, G.L. and Jaffe, H.W., 1975, Clinopyroxene exsolution lamellae: Fossil indicators of lattice parameters at high temperature and pressure: International Conference on Geothermometry and Geobarometry, Pennsylvania State University, October 5-10, 1975, p. 1-4.

38. Ross, Malcolm and Huebner, J.S., 1975, A pyroxene geothermometer based on composition-temperature relationships of naturally occurring orthopyroxene, pigeonite, and augite: International Conference on Geothermometry and Geobarometry, Pennsylvania State University, October 5-10, 1975, p. 1-4.

39. Nord, G.L., Jr., Ross, Malcolm and Huebner, J.S., 1976, Lunar troctolite 76535: Mineralogical Investigations: Proceedings of the Seventh Lunar Science Conference, Part 2, p. 628-630.

40. Nord, G.L., Jr., Huebner, J.S. and Ross, Malcolm, 1977, Structure, composition, and significance of "G-P" zones in 76535: Proceedings of the Eighth Lunar Science Conference, Part II, p. 732-734.

41. Ross, Malcolm, 1977, Minerals commonly occurring in the earth's crust that might be defined as asbestos by various regulatory agencies: Workshop on Asbestos: Definitions and Measurement Methods, July 18-20, 1977, USNBS-USDL.

42. Ross, Malcolm, 1977, Nonasbestiform minerals commonly occurring in mining operations that might be defined as "asbestos" by state and federal regulations: SME-AIME fall meeting, October 19-21, 1977, St. Louis, MO.

43. Ross, Malcolm, 1978, Asbestos health risks to the mining communities of North America: Geological Society of America Abstracts with Programs, v. 10, no. 7, p. 481.

44. Rutstein, M.S. and Ross, Malcolm, 1983, Goodbye "asbestos": Geological Society America Abstracts with Programs, v. 15, no. 3, 1983 NE Section Annual Meeting, p. 139.

45. Ross, Malcolm, 1984, Ultraalkalic arfvedsonite and associated richterite, acmite, and aegerine-augite in quartz syenite, Magnet Cove Alkalic Igneous Complex, Arkansas: Transactions of American Geophysical Union, v. 65, no. 16, p. 293.

46. Flohr, Marta J.K. and Ross, Malcolm, 1985, Pyroxene zonation trends in mafic nepheline syenite and ijolite, Diamond Jo Quarry, Magnet Cove Igneous Alkalic Complex, Arkansas: Geological Society of America Abstracts with Programs, v. 17, no. 7, 1985 Annual Meeting, p. 584.

47. Ross, Malcolm, 1985, Minerals and health: the asbestos problem: Program with Abstracts, 21st Forum on the Geology of Industrial Minerals, April 9-12, 1985, Tucson, AZ, p.17.

48. Ross, Malcolm, 1986, The dangers of asbestos in our environment: fact and fiction: Abstracts with Program (1986), the Fourteenth General Meeting of the International Mineralogical Association, 13-18 July 1986, Stanford University, Stanford, CA, p. 215.

49. Flohr, Marta J.K. and Ross, Malcolm, 1987, Geochemical trends of and relationships between alkaline rocks from the Magnet Cove igneous complex, Arkansas: Geological Society of America Abstracts with Programs, v. 19, no. 7, 1987 Annual Meeting, p. 664.

50. Ross, Malcolm, McGee, E.S. and Ross, D.R., 1988, Chemical and mineralogical effects of acid deposition on limestone and marble samples placed at four NAPAP weather monitoring sites (1988): Program and Abstracts, V. M. Goldschmidt Conference, May 11-13, 1988, Geochemical Society, p. 70.

51. Ross, Malcolm, 1989, Minerals and health: the "asbestos problem": Society of Mining Engineers Symposium, "Fibers, fibers, fibers", April 7, 1989, Baltimore, MD, p. 1-7.

52. Flohr, Marta J.K. and Ross, Malcolm, 1989, Mineralogy and alkali metasomatism of syenites from Magnet Cove alkaline igneous complex, Arkansas, USA: 28th International Geological Congress Abstracts, v. 1, 1989, p. 494.

53. Ross, Malcolm, Flohr, Marta J.K. and Ross, D.R., 1989, Compositional relations between natrolite, gonnardite, and thomsonite - products of nepheline alteration in alkaline rocks of the Magnet Cove igneous complex, Hot Springs, Arkansas. Geological Society of America Abstracts with Programs, v. 21, no. 6, 1989 Annual Meeting, p. A326.

54. McGee, E., Mossotti, V. and Ross, Malcolm, 1990, Gypsum accumulation on carbonate stone. International Conference on Acidic Deposition: Its Nature and Impacts, Glasgow, Sept. 16-21, 1990, Conference Abstracts, p. 482.

55. Ross, Malcolm, Flohr, Marta J.K. and Ross, Daphne R., 1990, Compositional and crystallographic relationships among the natrolite minerals thomsonite, gonnardite, natrolite, tetranatrolite, mesolite, and scolecite: in Abstracts for F.D. Bloss Symposium, Virginia Polytechnic Institute, Blacksburg, VA, July 22-25, 1990, p. A12.

56. Ross, Malcolm, 1991, Crystalline solution series and order-disorder within the natrolite mineral group. Geological Society of America Abstract with Programs, v. 23, No. 5, 1991 Annual Meeting, p. A159.

57. Ross, Malcolm, 1992, The effect of regulations on asbestos and other designated mineral carcinogens on the vitality of the industrial minerals industries: Proceedings of the 28th Forum on the Geology of Industrial Minerals, May 3-8, 1992, West Virginia Geological and Economic Survey, Martinsburg WV, p. 52-54.

58. Ross, Malcolm, 1992, Suspect minerals-an environmental concern?: Symposium and Annual Meeting, Sept. 27-30, 1992, American Institute of Professional Geologists, The Professional Geologist, v. 29, p. 8.

59. Ross, Malcolm, 1992, Suspect minerals and human health: Clay Minerals Society annual meeting, Nov. 1-6, 1992, New Visions, New Perspectives, 84 Annual Meeting Program, p. 150.
Abstracts (continued):

60. Ross, Malcolm, 1993, Mineral dusts, chemicals, chronic disease, and cancer: the dose makes the poison. Geological Society of America Abstracts with Programs, v. 25, no. 6, 1993 Annual Meeting, Boston, MA, p. A354.

61. Ross, Malcolm, 1993, The health effects of mineral dusts. The Environmental Geochemistry of Mineral Deposits Short Course and Seminar, April 13-16, 1993, Denver, CO, in Seminar Abstracts, Society of Economic Geologists, p. 12-13.

62. Takeda, Hiroshi and Ross, Malcolm, 1993, Characteristics of mica polytypes that are commonly observed and possible formation mechanisms (in Japanese). Program and Abstracts for the Crystallographic Society of Japan, November 1993, Tokyo.

63. Ross, Malcolm, 1994, The New Idria serpentinite of California: A toxic rock? Geological Society of America Abstracts with Programs, v.26. No. 7, 1994 Annual Meeting, Seattle, WA, p. A320.

64. Ross, Malcolm, 1995, The schoolroom asbestos abatement program: A public policy debacle, V.M Goldschmidt

Conference Program and Abstracts, p. 85, The geochemical Society, 1995.

65. Ross, Malcolm and Virta, R.L., 1997, Occurrence, production and uses of asbestos. International workshop on the health effects of chrysotile asbestos, September 14-16, 1997, Montreal, Quebec, Canada.

66. Nolan, R.P., Ross, M., Nord, G.L., Axten, C.W., and Wilson, R., 2002, Risk assessment based on an analysis of air and settled dust samples from the collapse of the Twin Towers of the World Trade Center. International Conference "Saftey and health in the production and use of asbestos and other fibrous materials", June 3-7, 2002, Ekaterinburg, Sverdlovsk Region, Russian Federation.

Published Reports by Malcolm Ross and others on asbestos related topics to 12/19/05

1. Ross, Malcolm, 1978, The "asbestos" minerals: Definitions, descriptions, modes of formation, physical and chemical properties, and health risk to the mining community: Workshop on Asbestos: Definitions and Measurement Methods, National Bureau of Standards special publication 506, p. 49-63.

2. Ross, Malcolm, 1980, Book Review (Asbestos, Vol. I, Properties, Applications and Hazards by L. Michaels and S.S. Chissisk, John Wiley, N.Y., 553 pp., 1979): in EOS, no. 27, July 1, 1980, p. 516.

3. Ross, Malcolm, 1981, The Geological Occurrences and Health Hazards of Amphibole and Serpentine Asbestos: in Reviews in Mineralogy, Vol. 9A, Amphiboles and Other Hydrous Pyriboles - Mineralogy, D.R. Veblen, Ed., Mineralogical Society of America, Washington, D.C., Nov., 1981, 279-323.

4. Ross, Malcolm, 1981, Book Review (Asbestos: Final Report of the Advisory Committee, Vol. I, pp. 100, Vol. 2: Papers Prepared for the Advisory Committee, pp. 103. Committee Chairman, W. Simpson, Health and Safety Commission of Great Britain. Her Majesty's Stationery Office, 40 High Holborn): in Journal of Applied Cryst., v. 14, p. 77.

5. Ross, Malcolm, 1982, A Survey of Asbestos-Related Disease in Trades and Mining Occupations and in Factory and Mining Communities as a Means of Predicting Health Risks of Non-Occupational Exposure to Fibrous Minerals: U.S. Geological Survey Open-file Report 82-745, 41 pp.

6. Ross, Malcolm, 1982, Asbestos, Health, and Society: Levels of Exposure of the General Population from Natural Sources: in Proc. World Symposium on Asbestos, May 25-27, 1982, Canadian Asbestos Information Centre, Montreal, p. 288-294.

7. Ross, Malcolm and Clifton, R.A., 1983, Arizona Long Fiber, Low Iron Chrysotile Asbestos as a Government Stockpile Source. April 7, 1983: Thru W.P. Pendley, Deputy Assistant Secretary for Energy and Minerals, Dept. Interior--for the Federal Emergency Management Agency.

8. Ross, Malcolm, 1984, A Survey of Asbestos-Related Disease in Trades and Mining Occupations and in Factory and Mining Communities as a Means of Predicting Health Risks of Non-Occupational Exposure to Fibrous Minerals: in Definitions for Asbestos and Other Health-Related Silicates, American Society for Testing Materials, STP Report No. 834, B. Levadie, Ed., p. 51-104.

9. Ross, Malcolm, Kuntze, R.A. and Clifton, R.A., 1984, A Definition for Asbestos: in Definitions for Asbestos and Other Health-Related Silicates, American Society for Testing Materials, STP Report No. 834, B. Levadie, Ed., p. 139-147.

10. Ross, Malcolm, 1984, A Definition for Talc: in Definitions for Asbestos and Other Health-Related Silicates: in Definitions for Asbestos and Other Health-Related Silicates, American Society for Testing Materials, STP Report No. 834, B. Levadie, Ed, p. 193-197.

11. Ross, Malcolm, 1987, Minerals and health: the asbestos problem: in the Proceedings of the 21st Forum on the Geology of Industrial Minerals, H.W. Peirce, Ed., Special Paper 4, 1987, Arizona Bureau of Geology and Mineral Technology, Tucson, AZ, p. 83-89.

12. Ross, Malcolm, 1988, The occurrence and dangers of amphibole and chrysotile asbestos in our environment: in the Proceedings of the Conference on Asbestos in Play Sand - Tremolite in Play Sand: Public Health Implications, C. E. Gilbert, Ed., NE Regional Environmental Public Health Center, Amherst, MA.

13. Skinner, H.C W., Ross, Malcolm, and Frondel, C., 1988, Asbestos and Other Fibrous Materials: Oxford University Press, NY, 204 pp.

14. Skinner, H C.W. and Ross, Malcolm, 1989, Summary of the Geological Society of America Forum on

"Fibrous minerals, mining and disease. The Professional Geologist, v. 26, no. 3, March 1989, p. 3-4.

15. Ross, Malcolm, 1990, Hazards associated with fibrous minerals. U.S. Geological Survey Circular 1033, in Proceedings of the U.S. Geological Survey Workshop on Environmental Geochemistry, Bruce Doe, Ed. p. 175-176.

16. Langer, A.M., Nolan, R.P., and M. Ross, 1990, Letter to the Editor, Asbestos Policy. Science, v. 249, p. 1485.

17. Ross, Malcolm, 1991, Suspect Minerals and human health: A commentary. The Lattice (Mineralogical Society of America Newsletter), v. 7. No. 1, p. 10-11.

18. Ross, Malcolm, 1991, "Carcinogenic" minerals and human health: A commentary. The Cross Section, U.S. Geological Survey Pub., v. 22, no. 3, p. 17-21.

19. Ross, Malcolm, Nolan, R.P., Langer, A.M., and Cooper, W.C., 1993, Health effects of mineral dusts other than asbestos. in Reviews in Mineralogy, v. 28, Health Effects of Mineral Dusts, G.D. Guthrie and B.T. Mossman (eds.), Mineralogical Society of America, Washington, DC. p. 361-407.

20. Ross, Malcolm and Skinner, H.C.W., 1993, Geology and Health. Geotimes, v. 39, p. 10-12.

21. Skinner, H.C.W. and Ross, Malcolm, 1994, Minerals and cancer. Geotimes, v. 39, p. 13-15.

22. Wilson, Richard, Langer, A.M., Nolan, R.P., Gee, J.B., and Ross, Malcolm, 1994, Asbestos in New York City schools? public policy: Is there a scientific basis? Regulatory Toxicology and Pharmacology, v. 20, p. 161-169.

23. Ross, Malcolm, 1995, The effect of regulations on asbestos and other designated mineral carcinogens on the vitality of the industrial minerals industries: Proceedings of the 28th Forum on the Geology of Industrial Minerals, May 3-8, 1992, B.J. Walker, Ed. Circular C-46, West Virginia Geological and Economic Survey, Morgantown, West Virginia, p. 149-151.

24. Ross, Malcolm, 1995, Failed policy on asbestos abatement. The Washington Times, October 8, p. B3.

25. Ross, Malcolm, 1995, The schoolroom asbestos abatement program: a public policy debacle. Environmental Geology, v. 26, p. 182-188.

26. Ross, Malcolm, 1997, Asbestos. McGraw-Hill Encyclopedia of Science and Technology.

27. Ross, Malcolm, 1999, The health effects of mineral dusts. in Reviews in Economic Geology, v. 6A, The Environmental Geochemistry of Mineral Deposits. Part A: Processes, Techniques, and Health Issues, Society of Economic Geologists, G.S. Plumlee and M.J. Logsdon, Eds., p. 339-356.

28. Ross, Malcolm and Nolan, R.P., 2000, Asbestos. McGraw-Hill Encyclopedia of Science and Technology, 9th Edition, New York, NY.

29. Ross, Malcolm, 2000, The Asbestos abatement in United States schools and other buildings. Newsletter of the Mineralogical Association of Canada, No. 63, Dec. 2000, p. 17-18.

30. Nolan, R.P., Langer, A.M., Ross, Malcolm, Wicks, F.J., and Martin, R.F. (Eds.) 2001, Health Effects of Chrysotile Asbestos: Contribution of Science to Risk Management Decisions. The Canadian Mineralogist Special Publication 5, 304 pp.

31. Ross, Malcolm and Virta, R.L., 2001, Occurrence, production and uses of asbestos. in Health Effects of Chrysotile Asbestos: Contribution of Science to Risk Management Decisions, .Nolan, R.P., Langer, A.M., Ross, M., Wicks, F.J., and Martin, R.F. (Eds.). The Canadian Mineralogist Special Publication 5, p. 79-88.

32. Ross, Malcolm and Nolan, Robert, 2003, History of asbestos discovery and use and asbestos-related disease in context with the occurrence of asbestos within ophiolite complexes. in Ophiolite concept and evolution of geologic thought, Dilek, Y. and Newcomb, S., Eds. Geological Society of America Special Publication 373, p. 447-470.

33. Nolan, R.P., Ross, M., Nord, G.L., Axten, C.W., Osleeb, J.P., and Wilson, R., 2003, Aassessment of risk of asbestos-related cancer based on analysis of air and settled dust samples from the 9/11 attack on the World Trade Center Complex. International Conference on "Saftey and health in the production and use of asbestos and other fibrous materials", June 3-7, 2002, Ekaterinburg, Sverdlovsk Region, Russian Federation, in Collected reports and speeches, NPO Asbestos Association, 2003, p. 99-128.

34. Nolan, R.P., Ross, Malcolm, Nord, G.L., Axten, C.W., Osleeb, J.P., Domnin, S.G., Price, Bertram, and Wilson, Richard, 2005, Risk assessment for asbestos-related cancer from the 9/11 attack on the World Trade Center. Journal of Occupational and Environmental Medicine, v. 47, no 8, p. 817-825.

35. Nolan, R.P, Ross, Malcolm, Nord, G.L, Raskina, M., Phillips, J.I., Murray, J., and Gibbs, G.W., 2005, Asbestos fiber-type and mesothelioma risk in the Republic of South Africa. Clay Science, International Clay Conference, Tokyo, Japan (in press).

REVISED: April, 2005

CURRICULUM VITAE

Arthur M. Langer, Ph.D.

CURRENT POSITIONS:

Professor – Ph.D. Program in Earth and Environmental Sciences, Graduate School and University Center of the City University of New York.

Deputy Executive Officer, Ph.D. Program in Earth and Environmental Sciences, The Graduate School and University Center, New York, NY.

Director - Center for Applied Studies of the Environment, Applied Sciences Coordinating Institute, City University of New York.

Research Associate, Department of Earth and Planetary Sciences, American Museum of Natural History, New York, NY.

ADDRESSES:

University Office: Ph.D. Program in Earth and Environmental Sciences
The Graduate School and University Center
365 Fifth Avenue – Room 5418
New York, NY 10016
Telephone: (212) 817-8123

Home Office: 6 Rochambeau Drive
Hartsdale, NY 10530
(914) 428-3667 Telephone
(914) 946-6524 Facsimile
Electronic Mail: ARTLANGER@AOL.com

ACADEMIC DEGREES:

B.A., Geology, Hunter College-CUNY, New York, NY, 1956.
M.A., Geology (Petrology), Columbia University, New York, NY, 1962.
Ph.D., Geology (Mineralogy), Columbia University, New York, NY, 1965.

PREVIOUS POSITIONS:

Professor and Director Environmental Sciences Laboratory of the Institute of Applied Sciences, Brooklyn College of the City University of New York, Brooklyn, NY 11210.

Associate Professor, Center for Polypeptide and Membrane Research, Mount Sinai School of Medicine, New York, NY 1986-1989.

Associate Professor, Mineralogy, Department of Community Medicine, Mount Sinai School of Medicine, New York, NY, 1968-1986, 1987-1988.

Science Administrator, Environmental Sciences Laboratory, Mount Sinai School of Medicine, New York, NY, 1983-1984.

Associate Director, Environmental Sciences Laboratory, an NIEHS Center, Center for the Study of Biological Effects of Environmental Agents, Mount Sinai School of Medicine, New York, NY, 1969-1986.

Director of Laboratories, Environmental Sciences Laboratory, Mount Sinai School of Medicine, 1976-1986.

Head, Physical Sciences Section, Environmental Sciences Laboratory, Mount Sinai School of Medicine, New York, NY, 1969-1986.

Adjunct Associate Professor, Mineralogy, Graduate Division, City University of New York, NY, 1968-1969.

Assistant Professor, Mineralogy, Department of Community Medicine, Mount Sinai School of Medicine, New York, NY, 1967-1968.

Research Associate, Environmental Medicine, Department of Medicine, Mount Sinai Hospital, New York, NY, 1965-1967.

Geologist, United States Geological Survey, Astrogeology Branch, Flagstaff, AZ. Appointment September, 1965. Leave of Absence.

Lecturer, Geology, City College of City University of New York, NY, 1964-1965.

Research Assistant, Mineralogy, Department of Geology, Columbia University, New York, NY, 1961-1964.

Teaching Assistant, Economic Geology, Department of Geology, Columbia University, New York, NY, 1959-1960.

Teaching Assistant, Department of Geology, Columbia College, New York, NY, 1958-1959.

Field Assistant, Geology, Beartooth Mountains, MT, Columbia University, 6/57-8/57; 6/58-8/58.

Exploration Geologist, Rosario Exploration Chibougamau Mining and Smelting, 6/56-8/56.

Consulting Mineralogist, Columbia University, New York, NY:

-Texas Gulf Sulphur Company, 1965 (with Professor Paul F. Kerr).
-Tidewater Oil Company, 1962 (with Professor Kerr).
-Consulting Mineralogist, Creole Oil Company, 1962 (with Professor Kerr).
-Consulting Mineralogist, American Metals Climax, 1961 (with Professor Kerr).
-TAMS Dam site, East Pakistan, WHO, 1960 (with Professor Rhodes Fairbridge).

FELLOWSHIPS:

Research Mineralogist, Department of Geology, Columbia University, New York, NY. Sponsored by the U.S. Air Force Cambridge Research Laboratories under the direction of Prof. P.F. Kerr, 3/61-9/63.

FELLOWSHIP IN PROFESSIONAL ORGANIZATIONS:

Fellow of the Geological Society of America.
Fellow of the Mineralogical Society of America.
Fellow of the New York Academy of Sciences.
Fellow of the Collegium Ramazzini.

HONORS AND AWARDS:

Biography in: American Men of Science; Who's Who in the East; Who's Who in America (September, 1992).
Fellow, Collegium Ramazzini, 1983.
Letter of Commendation – Mayor of the City New York. August, 1979. Asbestos in School Buildings.
Hunter College Hall of Fame HC-CUNY, June, 1978.
Phi Beta Kappa, Nu Chapter, Hunter College, New York, NY, June, 1977.
Career Scientist Award, National Institute of Environmental Health Sciences, June, 1969-May, 1974.
The John Polachek Foundation Award, For Medical Research – September, 1965-June,1967.
Sigma Xi, Kappa Chapter, Columbia University, New York, NY, September, 1964.
Department Honors, Department of Geology, Hunter College, New York, NY, June, 1956.

LABORATORY ORGANIZED CONFERENCES – ESL CUNY:

An International Workshop on the Health Effects of Chrysotile Asbestos: Contribution Of Science to Risk Management Decisions. Sponsored by the Mineralogical Society of America, Mineralogical Association of Canada. Montreal, Quebec. September 14-16, 1997.

An International Workshop of Simian Virus 40 (SV – 40), Asbestos and Mesothelioma. Sponsored by the Coalition to Support Mesothelioma Research. New York, NY November 9-10,1997.

AMICI CURIAE:

Atlantic Legal Foundation. Amici Curiae in support of Respondents to the Supreme Court of the United States. October Term, 1992. Daubert et.al. Petitioners v. Merrell Dow Pharmarceuticals,Inc., Respondent.
Atlantic Legal Foundation. Amici Curiae in support of Petitioners to the Supreme Court of the United States. October Term, 1996. General Electric Company, Westinghouse. Electric Company and Monsanto Company, Petitioners v. R. Joiner & K. Joiner, Respondents.
Atlantic Legal Foundation. Amici Curiae in Support of Defendants-Appellees to the United States Court of Appeals for the Fifth Circuit. Moore (Plaintiff-Appellant) vs.

Ashland chemical (Defendant-Appellee). Term 1998.
Request for Depublication. Arena v. Asbestos Corporation, Limited, First District Court of Appeal, Division 1, Number A077005. California. Filed May 12, 1998.

GRADUATE THESES:

Geology of the Manhattan Formation. Submitted in partial fulfillment for the degree of Master of Arts, in the Faculty of Pure Science, Columbia University, NY, p. 132, 1962.

Mineralogy and Physical Properties of Mojave Desert Playa Crusts. Submitted in partial fulfillment for the degree of Doctor of Philosophy, in the Faculty of Pure Science, Columbia University, NY, p. 155, 1965.

EDITORIAL BOARD SERVICE:

Assistant Editor, Environmental Research, 1978-1985.
Advisory Editor, Environmental Research, 1985-1987.
Assistant Editor, American Journal of Industrial Medicine, 1980-1985.
Associate Editor, American Journal of Industrial Medicine, 1985-1986.
Editorial Review Board, Journal of Environmental Pathology and Toxicology, 1978-1982.
Editorial Advisory Board, Advances in Modern Environmental Toxicology, 1981-1982.
Editorial Review Board, Journal of Environmental Pathology Toxicology and Oncology, 1983-Present.

REVIEW MANUSCRIPTS (Journals other than those cited above):

Journal of Histochemistry and Cytochemistry.
Pharmacology Reviews.
American Chemical Society Reviews.
Advances in Chemistry
Annals New York Academy Sciences.
Science (AAAS).
SEM-IITRI Symposia. Scanning Microscopy International.
Lung.
American Review of Respiratory Diseases.
American Mineralogist.
Clay Minerals Society (Clays and Clay Minerals).
Canadian J. Fisher Aquatic Sciences.
Journal National Cancer Institute.
American Journal Pathology.
Annual Meeting SEMA.
Chest.
Laboratory Investigations.
Journal of the American Medical Association.
New England Journal of Medicine.
Toxicology In Vitro.
Journal of the American Industrial Hygiene Association.

Annals of Occupational Hygiene.
Pathology Annual.
Environmental Health Perspectives.
Health Effects Institute.
Bulletin of the Geological Society of America.
Applied Occupational and Environmental Hygiene.
Medical Research Engineering.
American College of Chest Physicians.
Heterogeneous Chemistry Reviews.
American Institute for Testing Materials (ASTM)
Canadian Mineralogist
Journal of the American Industrial Hygiene Association
Journal of Indoor and Built Environment.

REVIEW AND CONTRIBUTIONS TO FEDERAL AND INDUSTRY DOCUMENTS (1978-PRESENT):

NIOSH Fibrous Glass Criteria Document, 1978. Reviewer. CDC-NIOSH.
NIOSH Talc Criteria Document, 1979. Reviewer, contributor. CDC-NIOSH.
OSHA Fiber Manuscript (Definitions, Nomenclature, Properties), 1980. Reviewer. US Department of Labor.
ALOSH Mineralogy Manuscripts (Occupational Lung Disease), 1979-1980. Reviewer, contributor. CDC-NIOSH.
Asbestos, Smoking and Disease. The Scientific Evidence. Burns, DM, Ed., Commercial Union Insurance, 1982. Contributor.
Environmental Protection Agency, Office of Pesticides and Toxic Substances-Asbestos in Buildings Guidance Documents; Guidance Documents; Operations and Maintenance Programs. "Orange", "Purple", "Blue" Books. Reviewer, Contributor, Committee member. US Environmental Protection Agency.
Surgeon General's Report: Cancer and Chronic Lung Disease in the Workplace: The Health Consequences of Smoking, 1985. Reviewer, Contributor. Federal. DHEW.

INTERNATIONAL GOVERNMENT, LABOR, COMMITTEES AND CONSULTATIONS:

US-Japan Cooperative Science Program Working Group on Air Pollution and Health. NIEHS-Institute of Health, Japan, 2/69.
Environmental Protection Agency, Superfund Cases, 3/85-Present.
National Institute for Occupational Safety and Health, 6/75-Present.
National Institute for Environmental Health Sciences, 4/75-Present.
Environmental Protection Agency, 6/75-Present.
National Heart, Lung and Blood Institute, 6/75-Present.
National Institutes of Health, Section of Grants, 6/74-Present.
World Health Organization, Geneva, Biomedical Expert, 6/75-Present.
International Agency for Research on Cancer, WHO Development of V.14 Chemical Carcinogenesis Monograph Series, Evaluation of Carcinogenic Risk of Chemicals to Man: Asbestos. Member of working group, Lyon, France, 1976.
International Association of Geochemistry and Cosmochemistry, Working Group on the

Geochemistry of Health and Disease, 1976-1979.

Consultant, South African Ministry of Mines, Asbestos Symposium, Johannesburg, South Africa, 1977.

Consultant, Institute of Public Health, Norway, Microscopy Facility, Oslo, Norway, 1977. Seminars on vitreous fibers as asbestos substitutes.

Consultant, International Metalworkers Federation, Problems Focusing on Asbestos Contamination of Nickel Ores, Geneva, Switzerland, 1980.

Research Consultant, Societe Nationale de l'Amiante, Quebec, Canada, Work on Modified Fiber, 1984.

WHO-International Program on Chemical Safety (IPCS), Environmental Health Criteria Document on Asbestos and Other Natural Mineral Fibers, Section Chairman, Hannover, Federal Republic of Germany, 1986.

Societe Nationale de l'Amiante. Consultant. Modified Fiber and the Environmental Protection Agency Asbestos Ban, Montreal, Canada, 1986.

International Agency for Research on Cancer, WHO Development of V.42, Chemical Carcinogenesis Monograph Series, Evaluation of Carcinogenic Risk to Chemicals in Man: Silica and Some Silicates, Member of Working Group, Lyon, France, 1986.

Consultant, Asbestos Institute, Canada, Organization of Symposium on Biological Effects of Asbestos Substitutes, 1987.

VIIth International Pneumoconiosis Conference. International Organizing Committee; Organizer of Session: Hazard Recognition of Mineral Dust. Pittsburgh, PA, August, 1988. Chaired Two Sessions at Meeting: Mineral Recognition by Membranes and Mineral Toxicity; Mineral Fiber and Diseases of the Pleura.

International Federation of Building and Wood Workers, Conference on Interior Works, Geneva, Switzerland. Presented paper: Hazards in the Painting Trades. Panel on Hazards in the Interior Workplace, May 9-12, 1989.

Hoffman-LaRoche Pharmaceuticals. Development of Microscopy Method for the Analysis of Mineral Fiber in Parenteral Drugs. Edinburgh, April, 1993; Basel, March, 1994; Frankfurt, May, 1994.

Eurometaux - European Association of Metals. Protocol Format and Development of Proposed Meeting on Carcinogenicity of Metals and the Compounds. Brussels, November, 1994.

Eurometaux - European Association of Metals. Protocol Format and Development of Proposed Meeting on Carcinogenicity of Metals and the Compounds. Brussels, September, 1995.

WHO – International Program on Chemical Safety. Chrysotile Asbestos. Environmental Health Criteria Document 203. Geneva. 1998.

NATIONAL COMMITTEES, WORKSHOPS:

Food and Drug Administration, Asbestos, Talc, Asbestos Bodies, and Consumer Talcum, Seminar, Washington, DC, June, 1968.

National Air Pollution Control Administration, Asbestos in Ambient Air. Arlington, VA. June, 1969.

National Institute for Occupational Safety and Health, USPHS, Asbestos Research in the United States, Cincinnati, OH, January, 1970.

Food and Drug Administration, Asbestos in Consumer Talcum, Seminar, Washington, DC, August, 1971.

Environmental Protection Agency, National Air Pollution Control Techniques Advisory Committee, Asbestos Emissions Document, Atlanta, GA, 1971.

NIOSH Task Force on Occupational Respiratory Diseases, 1975.

Environmental Protection Agency, HERL, Ad Hoc Committee for the Fibrous Amphibole Study Protocol, Triangle Park, NC, 1976.

NIEHS-NIOSH-EPA-ERDA. Interagency Retreat and Colloquium. Man-made Vitreous fibers, Asbestos Substitutes and the Energy Crisis. Potential Risks to Health. Pinehurst, NC. 7-9 January, 1976.

NIOSH Criteria Document Recommended Standard For Occupational Exposure to Fibrous Glass, Reviewer. November, 1976.

Environmental Protection Agency, HERL, Biological Effects on Fibrous Inorganic Particles, 1977.

NIH Possible Health Effects of Asbestos in the Local Environment. Bethesda, June 8, 1977.

NHLBI, Evaluation of Existing Inorganic Microparticulate Laboratories: Vermont Lung Group, Tulane Occupational Lung Hazards Group, 1978.

DHHS, Interagency Committee to Coordinate Environmental and Related Problems, Biological Effects of Fibrous Particulates: Serpentine-Containing Host Rocks, Amphibole-Containing Host Rocks, 1980.

Mount St. Helen's Volcanic Ash: Mineral Nature and Biological Activity, Interagency Task Force, Bethesda, MD, 1980.

National Institute of Environmental Health Sciences, Workshop, Pathobiology of Mesothelioma, RTP, NC, January, 1983.

Occupational Safety and Health Administration, Expert to write and review Asbestos Standard-Talc Standard, US Department of Labor, 1983.

National Academy of Sciences, Division of Life Sciences Committee to Evaluate Risk to Low-Level Exposure to Asbestiform Fibers in the Environment, 1982-1984.

Environmental Protection Agency. Prepared for meeting Substitute Fibers. Pre-market Testing of Fibers. USEPA, Washington, DC. 26 June 1984.

Lung Center and NHLB-SCOR, in the Departments of Physiology, Medicine, Pathology, and Engineering in the University of Vermont, Burlington, VT, 1984.

Environmental Protection Agency, Committee Member, Review and Rewrite EPA "Guidance Document for Controlling Friable Asbestos-Containing Minerals in Buildings," 1985

American Society Testing Materials, Committee, D22-Indoor Air Pollution Asbestos: D22.05-Methodology and Measurement of Fibers in Air. Electron Microscopy, 1985.

American Society Testing Materials, Organizing Committee, Silica and Silica-Induced Diseases, International Conference, 1985.

NIOSH, Mine Health Research Advisory Committee Meeting, Tucson, AZ: The Impact of Mineral-Asbestos Definitions on the Mining Industry, 1986.

Environmental Protection Agency. Develop guidance document for identifying asbestos hazards and implementing abatement programs in public buildings, Washington, DC, April, 1986.

NIOSH Review Panel for Project "Evaluation of Mesothelioma Production by Asbestos

Substitutes." Cincinnati, OH, June, 1986.
Environmental Protection Agency, Guidance Document for Assessing and Managing Exposure to Asbestos in Building, Arlington, VA, September, 1986.

Environmental Protection Agency, Operations and Maintenance Programs for Asbestos-Containing Materials in Buildings: A Guide for Building Owners and Managers, Washington, DC, January, 1987.
NIEHS-NIOSH - National Toxicology Program. To formulate study protocol comparing biological activities of asbestiform and non-asbestiform amphibole minerals. NIEHS - Res. Triangle Park, NC, October 11, 1989.
Health Effects Institute. Asbestos Literature Review Panel. April, 1990 - August, 1991.
Health Effects Institute. Reviewer of Research Proposals - RPI-1-91.
US FDA and Intl. Soc. Reg. Tox. Pharmac., NIH Bethesda, Workshop. Talc: Consumer Uses and Health Perspectives. 31 Jan - 1 Feb, 1994.

TESTIMONY BEFORE FEDERAL AGENCIES, FEDERAL COURTS:

Testimony National Toxicology Program Committee Evaluating Talc as a Human Carcinogen. Washington DC, 13-14 December, 2000.
Testimony Occupational Safety and Health Administration, U.S. Department of Labor. Asbestos Standard Revisions. Asbestos Fiber and Cleavage Fragments, April, 1990. Washington, D.C.
Testimony, Consumer Products Safety Commission. Asbestos in Play Sand. Washington, DC, December 7, 1988.
Testimony, Occupational Safety and Health Administration, US Department of Labor, Asbestos Standard, 1984. Modified Asbestos and It's Biological Behavior. Washington, D.C.
Expert Witness, Contamination of Lake Superior. On behalf of the Department of Justice of the United States, 1974. Minneapolis, MN. Mineral Fiber Analysis.
Testimony, Toxic Substances Control Act, 1973. Presented evidence to Sen. Tunney's committee hearing, concerning asbestos in society. Washington, D.C.

REGIONAL CONSULTATIONS:

Health Research Council of New York City, Subcommittee on Asbestos Hazards - Air Pollution Working Corp. Seminar on Asbestos in Construction Products, Rockefeller University, June 9, 1969.
New York City Board of Education: Asbestos in Schools, 1981-1983.
New York City Department of Sanitation; Fire Department: Insulation Products, 1979-1983.
New York State Consumer Affairs and Protection: Construction and Insulation Products, 1980-1983.
City University of New York-Brooklyn College: Asbestos Problems, 1981-1982.
New York City Asbestos Taskforce. Inspector General's Office. Asbestos in schools. Analysis and evaluation of samples removed from school buildings. Fall, 1993.

ACADEMIC SERVICE AND COMMITTEES:

Academic Council, Department Representative, MSSM, 1977-1979.
Elections Committee of the Academic Council, MSSM, 1978-1980.
Medical Center Safety Committee, MSSM, 1979-1988.
Chemical Hazards Committee, MSSM, 1979-1988.
Alternate Medical Safety Officer (in absence of Dr. S. Kochwa), MSSM, 1980-1983.
Space Committee (Department and Institution), MSSM, 1982-1985.
New York Academy of Sciences, Conference Organizing Committee, 1976-1980.
Visiting Professor Program. Mt. Sinai School of Medicine. 1971-1973.
Educational Policy Committee. Mt. Sinai School of Medicine. 1972-1974.
Ad Hoc Reviewer, National Institutes of Health, Minneapolis Medical Center, 1977.
Ad Hoc Reviewer, National Institutes of Health, Harvard Medical Center, 1978.
Workshop Organizer and Chairman, Significance of Aspect Ratio in Asbestos Diseases, New York Academy of Sciences, 1977.
Workshop Organizer, Third International Workshop on In Vitro Testing of Mineral Dusts, 1984.
Curriculum and Examination Committee, Ph.D. Program, Earth and Environmental Sciences, City University of New York, 1987-1990; 1996-present.
Faculty Membership Committee - Chairman - Ph.D. Program, Earth and Environmental Sciences, City University of New York, 1989-1992; 1996-present.
Executive Committee, Ph.D. Program, Earth and Environmental Sciences, City University of New York, 1985-1993; 1995-present.
First Examination Committee, Ph.D. Program, Earth and Environmental Sciences, City University of New York, 1996-present.
Elections Committee, Ph.D. Program, Earth and Environmental Sciences, City University of New York, 1996-present.
Graduate Faculty Council, The Graduate Center of the City University, 2000-2003.

CONSULTATIONS:

Oil, Chemical and Atomic Workers International Union, Safe Handling of Asbestos, Training Film, 1976.
Cyprus Minerals-Nature of US Talc Deposits, 1979.
General Accident Insurance Company: Asbestos Compensation, 1981.
Gulf Minerals: Modified Chrysotile Fiber, 1982.
Societe Nationale de l'Amiante: Phosphorylated Fiber; Asbestos Substitutes, 1984.
W.R. Grace & Company: Asbestos and Indoor Air Pollution, 1984.
Asbestos Institute of Canada, Asbestos and Asbestos Substitutes, 1985-1986.
R.T. Vanderbilt Company, Nature of Tremolite in the Gouverneur Talc Deposit, 1987.
Safe Building Alliance, Problems of Asbestos in Buildings, 1987.
Review applications for funding requests: FCAC, Quebec, Canada, 1985.
United States Consumer Products Safety Commission, 1986-1987.
Society Nationale d'L'Amiante – Canada, 1987.
US National Toxicology Program, 1989.
Battelle Columbus Laboratories, 1987-1989; 1991.
Durabla Gasket Manufacturing Co., 1992.

Hoffmann-LaRoche-Basel, 1993.
Lapinus Fibres-BV-Netherlands, 1993-1994.
Ready Mix Group-Australia, 1994.
Cleveland-Cliffs Group, 1995-1996.
Blown Glass Wool Contractors Assn., 1996.
Novacor Chemicals-Canada, 1996.
Talc Luzenac-Paris, 2000.

PEER-REVIEWED PUBLICATIONS:

1. Kerr PF, Thomas AM, Langer AM: The nature and synthesis of ferrimolybdite. Amer Mineral 48:14-32, 1963.

2. Kerr PF, Langer AM: Mineralogical features of Mojave playa crusts. In: Mineralogy and Hydrology of US Playas. Neal J (ed), US Air Force Cambridge Research Laboratories Environmental Research Paper No. 96, pp. 31-72, 1965.

3. Langer AM, Kerr PF: Experimental variables influencing DTA curves of kaolinite. Dupont Thermogram 3:1-4, 1966.

4. Langer AM: Older Paleozoic metamorphism and pegmatization in Bronx, New York. Ann NY Acad Sci 136:1-32, 1966.

5. Langer AM, Kerr PF: Mojave Desert playa crusts: physical properties and mineral content. J Sed Petrol 36:377-396, 1966.

6. Langer AM, Kerr PF: Evaluation of kaolinite and quartz differential thermal curves with a new high-temperature cell. Am Mineral 52:509-523, 1967.

7. Berkley C, Langer AM, Baden V: Instrumental analysis of inspired pulmonary particulates. Trans NY Acad Sci 30:331-350, 1967.

8. Neal JT, Langer AM, Kerr PF: Giant desiccation polygons of Great Basin playas. Bull Geol Soc Am 79:59-90, 1968.

9. Langer AM, Bowes DR: Polyphase deformation in the Manhattan formation of Manhattan Island, New York City. Memoir 115 Geol Soc Am 358-373, 1969.

10. Langer AM, Selikoff IJ, Sastre A: Chrysotile asbestos in the lungs of persons in New York City. Arch Environ Health 22:348-361, 1971.

11. Langer AM, Mackler AD, Rubin IB, Hammond EC, Selikoff IJ: Inorganic particles in cigars and cigar smoke. Science 174(4009):585-587, 1971.

12. Selikoff IJ, Nicholson WJ, Langer AM: Asbestos air pollution in urban areas. Arch Environ Health 25:1-13, 1972.

13. Liebling R, Langer AM: Optical properties of fibrous brucite from Asbestos, Quebec. Am Mineral 57:857-864, 1972.

14. Langer AM, Rubin IB, Selikoff, IJ: Chemical characterization of asbestos body cores by electron microprobe analysis. J Histochem Cytochem 20 (9):723-734, 1972.

15. Langer AM, Rubin IB, Selikoff IJ, Pooley, FD: Chemical characterization of uncoated asbestos fibers from lungs of asbestos workers by electron microprobe analysis. J Histochem Cytochem 20(9):735-740, 1972.

16. Bowes DR, Langer AM: Petrochemistry of the Manhattan formation, New York City. Krystalinikum 10:39-51, 1974.

17. Langer AM, Ashley R, Baden V, Berkley C, Hammond EC, Mackler AD, Maggiore CJ, Nicholson WJ, Rohl AN, Rubin IB, Sastre A, Selikoff IJ: Identification of asbestos in human tissues. J Occup Med 15:287-295, 1973.

18. Ehrenreich T, Mackler AD, Langer AM, Selikoff IJ: Les fibers d'amiante dans les poumons humains: leur signification medicolegale dans les maladies de l'environment. Arch des Maladies Professionelles 34(4-5):189-204, 1973.

19. Bowes DR, Langer AM: Hornblende schists in the Manhattan formation in the Bronx, New York:Discussion. Bull Geol Soc Am 84:1483-1490, 1973.

20. Ehrenreich T, Mackler AD, Langer AM, Selikoff IJ: Identification and characterization of pulmonary dust burden in pneumoconiosis. Ann Clin Lab Sci 3(2):118-131, 1973.

21. Kleinfeld M, Messite J, Langer AM: A study of workers exposed to asbestiform minerals in commercial talc manufacture. Environ Res 6(2):132-143, 1973.

22. Nicholson WJ, Langer AM, Selikoff IJ: Discussion: asbestos fibers in the air of towns. Atmosph Environ 7:666-668, 1973.

23. Langer AM: Inorganic particles in human tissues and their association with neoplastic disease. Environ Health Perspect 9:229-233, 1974.

24. Langer AM: Approaches and constraints to identification and quantitation of asbestos fibers. Environ Health Perspect 9:133-136, 1974.

25. Langer AM: Research perspectives concerning asbestos minerals and their effects on biological systems. Environ Health Perspect 9:335-338, 1974.

26. Langer AM: The subject of continuous vigilance. Environ Health Perspect 9:53-56, 1974.

27. Rohl AN, Langer AM: Identification of asbestos in talc. Environ Health Perspect 9:95-109, 1974.

28. Langer AM, Mackler AD, Pooley FD: Electron microscopical investigation of asbestos fibers. Environ Health Perspect 9:63-80, 1974.

29. Miller A, Langer AM, Teirstein, AS, Selikoff IJ: "Non-specific" interstitial fibrosis: Association with fibers detected by electron microscopy. N Engl J Med 292:91-93, 1975.

30. Rohl AN, Langer AM, Selikoff IJ, Nicholson WJ: Exposure to asbestos in use of consumer spackling, patching, and taping compounds. Science 189(4204):551-553, 1975.

31. Rohl AN, Langer AM, Klimentidis R, Wolff MS: Asbestos exposure during brake lining maintenance and repair. Environ Res 12:110-128, 1976.

32. Rohl AN, Langer AM, Selikoff IJ, Tordini A, Klimentidis R, Bowes DR, Skinner D: Mineral and chemical characterization of selected consumer talcum products. J Toxicol Environ Sci 2:255-284, 1976.

33. Bowes DR, Langer AM, Rohl AN: Nature and range of mineral dusts in the environment. Phil Trans Roy Soc London 286:593-610, 1977.

34. Rohl AN, Langer AM, Klimentidis R, Wolff MS, Selikoff IJ: Asbestos content of dust encountered during brake maintenance and repair. Proc Roy Soc Med 70:32-39, 1977.

35. Rohl AN, Langer AM, Selikoff IJ: Environmental asbestos pollution related to use of quarried serpentine rock. Science 196:1319-1322, 1977.

36. Langer AM, Wolff MS, Rohl AN, Selikoff IJ: Variation of properties of chrysotile asbestos, subjected to prolonged milling. J Toxicol Environ Sci 4:173-188, 1978.

37. Fischbein A, Langer AM, Suzuki Y, Selikoff IJ: Carcinoma of the lung in a drywall taping worker: report of a case. Toxicol Letters 2:231-236, 1978.

38. Langer AM: Crystal faces and cleavage planes in quartz as templates in biological processes. Quart Rev Biophys 11:543-575, 1978.

39. Rohl AN, Langer AM, Selikoff IJ: Airborne asbestos in the vicinity of a freeway: discussion. Atmosph Environ 12:2030-2031, 1978.

40. Langer AM, Rohl AN, Selikoff IJ: Asbestos on Maryland's roads. The Lancet i:1263-1264, 1978.

41. Fischbein A, Rohl AN, Langer AM, Selikoff IJ: Drywall construction and asbestos exposure. Am Industr Hyg Assoc 40:402-407, 1979.

42. Langer AM, Rohl AN, Selikoff IJ, Harlow G, Prinz M: Asbestos as a co-factor among nickel processing workers. Science 209:420-422, 1980.

43. Nolan RP, Langer AM, Harington JS, Oster G, Selikoff IJ: Quartz hemolysis as related to its surface functionalities. Environ Res 26:503-520, 1981.

44. Rohl AN, Langer AM, Moncure G, Fischbein AS, Selikoff IJ: Endemic pleural disease associated with mixed fibrous dust exposure in Turkey. Science 216(4545):518-520, 1982.

45. Langer AM, McCaughey WET: Mesothelioma in a brake repair worker. The Lancet ii:1101-1103, 1982.

46. Ehrenreich T, Espinoza T, Langer AM, Rohl AN, Daum SM: Algorithm for forensic pathological diagnosis of asbestos-related diseases. Am J Foren Med Pathol 3(4):315-321, 1982.

47. Langer AM, Rohl AN, Fischbein AS, Selikoff IJ: Cancer in nickel processing workers in New Caledonia: discussion. Science 215:425-426, 1982.

48. Yeager H, Russo D, Yanez M, Gerardi D, Nolan RP, Kagan E, Langer AM: Preliminary observations on acute cytotoxicity of short fiber chrysotile asbestos for human alveolar macrophages. Environ Res 30:224-232, 1983.

49. Brody J, Miller A, Langer AM: Pneumoconiosis associated with exposure to glass and abrasive fragments. Am J Industr Med 6:339-346, 1984.

50. Constantopoulos S, Goudevenos J, Saratzis N, Charalampopoulos C, Laparidou S, Langer AM, Selikoff IJ, Moutsopoulos H: Endemic asbestos-like pleural calcifications in the absence of occupational asbestos exposure. Arch Hellenic Med 1(10):165-171, 1984.

51. Sawyer R, Rohl AN, Langer AM: Fiber contamination in buildings resulting from removal of asbestos-containing materials. Environ Res 36(1):46-55, 1985.

52. Constantopoulos SH, Goudevenos JA, Saratzis N, Langer AM, Selikoff IJ, Moutsopoulos HM: Metsovo lung: pleural calcification and restrictive lung function in northwestern Greece. Environmental exposure to mineral fiber as etiology. Environ Res 38:319-331, 1985.

53. Langer AM, Nolan RP: Asbestos in potable water supplies and attributable risk of gastrointestinal cancer. Northeast Environ Sci 5(1/2):41-53, 1986.

54. Langer AM, Nolan RP, Constantopoulos SH, Moutsopoulos HM: Association of Metsovo lung and pleural mesothelioma with exposure to tremolite-containing whitewash. The Lancet i:965-967, 1987.

55. Constantopoulos SH, Langer AM, Saratzis N, Nolan RP: Regional findings in Metsovo lung. The Lancet ii, 452-453, 1987.

56. Nolan RP, Langer AM, Weisman I, Herson R: Surface character and membrolytic properties of the titania polymorphs. Br J Ind Med 44(10):687-698, 1987.

57. Nolan RP, Langer AM, Eskenazi RA, Herson GB, Foster KW: Membranolytic activities of quartz standards. Toxicol In Vitro 1(4):239-245, 1987.

58. Langer AM, Nolan RP, Constantopoulos SH: Endemic pleural calcification and mesothelioma. JAMA 260 (3) 339-340, 1988.

59. Langer AM, Nolan RP: Fiber type and mesothelioma risk. Symposium of Health Aspects of Exposure to Asbestos in Buildings. Energy and Environmental Policy Center, Kennedy School of Government. Harvard University. December, 1989, 91-141.

60. Freed JA, Miller A, Gordon R, Fischbein A, Kleinerman J, Langer AM: Desquamative interstitial pneumonia associated with chrysotile asbestos fibers. Br J Ind Med, 48: 332-337, 1991.

61. Nolan RP, Langer AM, Herson GB: Characterization of palygorskite specimens from different geological locales for health hazard evaluation. Br J Ind Med, 48: 463-475, 1991.

62. Langer AM, Nolan RP: Chrysotile biopersistence in the lungs of persons in the general population and exposed workers. Env. Health Perspect. Supplts., 102, Suppl. 5, 235-239, 1994.

63. Nolan RP, Langer AM, Addison J: Lung content analysis of cases occupationally exposed to chrysotile asbestos. Env. Health Perspect. Supplts., 102, Suppl. 5, 245-250, 1994.

64. Langer AM, Nolan RP: Chrysotile: Its Occurence and Properties as variables controlling Biological Effects. Ann. Occup. Hyg., 38, No. 4, 427-451. 1994.

65. Wilson R, Langer AM, Nolan RP, Gee JBL, Ross M: Asbestos in New York City public school buildings. Public Policy: Is there a scientific basis? Jour. Regulatory Toxicology and Pharmacology, 20, 161-169, 1994.

66. Langer AM, Nolan RP: Physical chemical properties to fibers other than asbestos in global use. J. Occup. Health and Safety. Aust and New Zeal., 12, No. 3, 263-278, 1996.

67. Langer AM, Nolan RP: Asbestos in the lungs of persons exposed in the United States. Monaldi Arch Ches Dis, 53, No.2, 168-180. 1998.

68. Nolan RP, Langer AM, Wilson RP: A risk assessment for exposure to grunerite asbestos (amosite) in an iron ore mine. Proc. Natl. Acad. Scis. 96, 3412-3419, 1999.

69. Wilson R, Langer AM, Nolan RP: A risk assessment for exposure to glass wool. Regulatory Toxicol. and Pharmacol. 30 (2): 96-109, 1999.

70. Nolan RP, Langer AM: Risk assessment of the influence of chrysotile asbestos on human health. In: Reports for the International Scientific-Practical Conference on the Safe Usage of Chrysotile Asbestos. 42-49. Ivano-Frankovsk, Ukraine. 1999.

71. Langer AM, Health experience of some U.S. and Canadian workers exposed to asbestos. Foundation for risk assessment. The Canadian Mineralogist. Special Publication No. 5, The Health Effects of Chrysotile Asbestos. 9-20, 2001.

72. Nolan RP, Langer AM: Concentrations and type of asbestos fiber in air inside buildings. The Canadian Mineralogist. Special Publication No. 5, The Health Effects of Chrysotile Asbestos, 39-51, 2001.

73. Langer AM: Summary of the Symposium. The Canadian Mineralogist. Special Publication No. 5. 291-296, 2001.

74. Langer AM: The carcinogenicity of chrysotile asbestos. Is the evidence sufficient for a global ban? Columns. Asbestos. Raising the Bar in Asbestos Litigation. 2001 4-5,61-68, 2001.

75. Langer AM, Morse RG: The World Trade Center Catastrophe. Was the type of spray fireproofing a factor in the collapse of the Twin Towers? J Indoor and Built Environment 10: 350-360. 2001.

76. Langer AM: Reduction of the biological potential of chrysotile asbestos arising from conditions of service on brake pads. J Regulatory Toxicology and Pharmacology. 38: 71-77. 2003.

77. Langer AM: Control and monitoring of asbestos in the United States. Regulatory overview of asbestos and other fibrous minerals. Manuscript in review. 2005.

78. Langer AM: Risk Analysis for Chrysotile in Friction Products and Cement. Evidence in Support of Continued Use. Manuscript in Review. 2005.

79. Langer AM: The future of terrorism risk insurance. Protecting buildings from terrorist attacks. TRIA Compendium. DRI Chicago. 231-242. 2005.

PUBLICATIONS IN SYMPOSIA PROCEEDINGS:

1. Berkley C, Langer AM, Sastre A, Arneson A: Electron microprobe analysis of asbestos bodies. In: Int'l Konferenz uber die Biologischen Wirkungen des Asbestos,

Dresden, 22-24 April, 1968. Holstein, Anspach M (eds), Deutsches Zentralinstitut fur Arbeitsmedizin, Berlin, DDR, 12-22, 1968.

2. Schwartz J, Langer AM: Technique of removal and analysis of single fibrous particles from human lung tissue. In: Int'l Konferenz uber die Biologischen Wirkungen des Asbestos, Dresden, 22-24 April, 1968. Holstein, Anspach M (eds), Deutsches Zentralinstitut fur Arbeitsmedizin, Berlin, DDR, 8-12, 1968.

3. Bowes DR, Langer AM: Polyphase deformation in the schist of the Bronx, New York City. In: Geology, Queens College, CUNY, Alexandroff E (ed) 1(3):17-32, 1969.

4. Langer AM: Electron microprobe analysis (study of asbestos fibers and bodies from lung tissue). In: Laboratory Diagnosis of Diseases Caused by Toxic Agents. Sunderman FW, Sunderman Jr FW (eds), W.H. Green; St. Louis, MO, Chapt. 14, 126-136, 1970.

5. Langer AM, Rubin IB, Selikoff IJ: Electron microprobe analysis of asbestos bodies. In: Pneumonconiosis, Proc Int'l Conf, Johannesburg. Shaprio HA (ed), Oxford University Press; Capetown, 57-69, 1970.

6. Langer AM, Baden V, Hammond EC, Selikoff IJ: Inorganic fibers, including chrysotile in the lungs at autopsy: preliminary report. In: Inhaled Particles III, Proc Conf Brit Occup Hyg Soc, London. Walton WH (ed), Unwin Bros; Surrey, England, 2:683-694, 1971.

7. Langer AM, Selikoff IJ: Chrysotile asbestos of lungs in residents of New York City. In: 2nd Int'l Clean Air Congress, Washington, DC. Englund HM, Beery, WT (eds), Academic Press; New York, 161-165, 1971.

8. Langer AM, Pooley FD: Identification of single asbestos fibers in human tissues. In: Proc Int'l Agency for Research on Cancer, Biol. Effects of Asbestos, Lyon, 1972, Bogovski P, Gilson JC, Timbrell V, Wagner JC (eds), 119-125, 1973.

9. Langer AM: Aspects of mineralogy of talc. In: Proc Symp on Talc, Washington, DC, May 9, 1973. Goodwin A (ed), USBM IC 8639, US Department of Interior, 82-88, 1974.

10. Pooley FD, Rohl AN, Langer AM, Bowes DE, Skinner DL: Mineralogy and chemistry of British talc and consumer talcum products. In: 4th Int'l Symp Inhaled Particles and Vapors. Walton WH (ed), Pr. 8.6, 1-18, 1975.

11. Langer AM, Rohl AN, Wolff MS, Klimentidis R, Shirey SB: Review of current techniques for the analysis of fibers in talc. In: Electron Microscopy of Microfibers. Asher IM, McGrath PP (eds), US Government Printing Office, 28-33, 1977.

12. Langer AM, Wolff MS: Asbestos carcinogenesis. In: Inorganic and Nutritional Aspects of Cancer. Adv Exper Med Biol Series, Schrauzer GN (ed), Plenum Press;

New York, 29-55, 1977.

13. Nicholson WJ, Langer AM, Selikoff IJ: Epidemiological evidence on asbestos, Part I: Human health effects; Part II: Extrapolation to other inorganic fibers. In: Proc of Workshop on Asbestos, NBS SP 506. Gravatt CC, LaFleur PD, Heinrich KFJ (eds), with discussion. US Government Printing Office, Washington, DC, 71-93, 1978.

14. Langer AM: Relationship between chemical and physical properties of mineral fiber and health effects. Proc of Workshop on Asbestos. NBS SP 506. Gravatt CC, LaFleur PD, Heinrich KFJ (eds). 194-195, 198, 1978.

15. Rohl AN, Langer AM: Fibrous mineral content of consumer talc-containing products. In: Dusts and Diseases. Lemen R, Dement JM (eds), Pathotoxicol Publishers, Inc; Forest Park, IL, 393-403, 1979.

16. Langer AM, Rohl AN, Wolff MS, Selikoff IJ: Asbestos, fibrous minerals, and acicular cleavage fragments: nomenclature and biological properties. In: Dusts and Diseases. Lemen R, Dement JM (eds), Pathotoxicol Publishers, Inc; Forest Park, IL, 1-22, 1979.

17. Langer AM, Maggiore CJ, Nicholson WJ, Rohl AN, Rubin IB, Selikoff IJ: The contamination of Lake Superior with fibrous amphiboles. In: Proc NY Acad Sci Asbestos Conf, New York, NY, June 1978, Ann NY Acad Sci 330:549-572, 1979.

18. Langer AM: Significance of aspect ratio in regulation of asbestos fiber exposure. Ann NY Acad Sci 330:601-604, 1979.

19. Langer AM, Selikoff IJ, Rosenberg C: Defining new asbestos high-risk groups. In: Int'l Conf Critical Current Issues in Environmental Health Hazards, Tel Aviv, Israel, March 4-7, 11, 1979.

20. Langer AM, Sebastien P: Physical-chemical properties of fibers: discussion. In: Biological Effects of Asbestos, IARC, Vol. 1, Wagner JC (ed), WHO, Lyon, France, 143-145, 1980.

21. Suzuki Y, Rohl AN, Langer AM, Selikoff IJ: Mesothelioma following intraperitoneal administration of zeolite. Fed Proc 39 (3): 640, 1980.

22. Langer AM: Natural non-fibrous asbestos substitutes: Talc. In: Proc Nat'l Workshop on Substitutes for Asbestos, Guimond RJ, Rowe J (eds), US Environmental Protection Agency, Office of Pesticides and Toxic Substances, Washington, DC, 563-569, 1980.

23. Langer AM: Mineralogy and physical properties of fibers. Talcs contaminated with other minerals. Discussions. In Dusts and Disease. Lemen R, Dement J (eds). Pathotox Pub Inc., Park Forest, IL. 120; 122-123; 129; 213-214; 265; 341-344; 348; 354; 414-415. 1979.

24. Rohl AN, Ehrenreich T, Langer AM: Characterization and identification of asbestos materials in human tissues. In: Adv Pathol (Anat and Clin), Vol. 2, Anatomic Pathology, Cytopathology, Forensic Pathology and Toxicology, Levy E (ed), Pergamon Press, New York, 523-525, 1982.

25. Langer AM, Weisman I, Adams A: Characterization of crystalline particles in human tissues by analytical electron microscopy. J Rheumatol 8(6):1018-1019, 1982.

26. Nolan RP, Langer AM: Quartz and hemolysis: physico-chemical factors controlling membrane activity. In: 4th Ann RMCOEH Conf Health Issues Related to Mining and Minerals, Wagner W, Merchant J, Rom W (eds), Chapt. 4, Ann Arbor Press, Michigan, 63-81, 1983.

27. Langer AM: Surface characteristics and biological properties of minerals. In: World Symp on Asbestos, May 25-27, 1982, Canadian Asbestos Information Center, Montreal, Quebec, 393-400, 1983.

28. Langer AM, Nolan RP: Minerals, rocks and ore bodies: sources of agents of human disease. In: Proc 2nd Int'l Congr on Applied Mineralogy in the Minerals Industry. Park WC, Hausen DM, Hagni RP (eds),The Metallurgical Society – AIME. 1159-1179, 1985.

29. Harlow GE, Kimball MR, Dowty E, Langer AM: Observations on amosite-grunerite dusts. In: Proc 2nd Int'l Congr on Applied Mineralogy in the Minerals Industry. Park WC, Hausen DM, Hagni RP (eds), The Metallurgical Society – AIME. 1147-1157, 1985.

30. Nolan RP, Langer AM, Foster KW: Particle size and chemically induced variability in the membrolytic activity of quartz: preliminary observations. In: In Vitro Effects of Mineral Dusts, 3rd Int'l Workshop. Beck EG, Bignon J (eds), Springer-Verlag, Berlin, 39-50, 1985.

31. Langer AM, Nolan RP: Physico-chemical properties of minerals relevant to biological activities. State of the Art. In: In Vitro Effects of Mineral Dusts, 3rd Int'l Workshop. Beck EG, Bignon J (eds), Springer-Verlag, Berlin, 9-24, 1985.

32. Langer AM, Nolan RP: Physico-chemical properties of quartz and biological activity. In: Silica, Silicosis, and Cancer. Controversy in Occupational Medicine. Ca Res Monogr 2, Goldsmith DF, Winn DM, Shy CM (eds), Praeger, NY,125-136, 1986.

33. Langer AM, Nolan RP: The properties of chrysotile asbestos as determinants of biological activity. Variations in cohort experience and disease spectra as related to mineral properties. In: General Motors Workshop, Biol Effects Chrysotile Asbestos, Cardiff, 7-9 May, 1986, Wagner JC (ed), Accomplishments in Oncology, Lippincott Press, 1(2): 30-51, 1986.

34. Langer AM: Fibers in friction products. Discussion and Summary of Symposium. In: Proc Fibres in Friction Materials Symposium. The Asbestos Institute, Montreal, Canada, 67-74, October, 1987.

35. Nolan RP, Langer AM: Quantitative aspects of fiber morphology. In: Proc Fibres in Friction Materials Symposium. The Asbestos Institute, Montreal, Canada, 75- 97, October, 1987.

36. Langer AM, Nolan RP: Fiber type and burden found in parenchymal tissues of workers occupationally exposed to asbestos in the United States. In: Mineral Fibers in the Non-Occupational Environment, Spec. Publ. 90. IARC-WHO, Lyon, France, 310-315, 1989.

37. Langer AM, Nolan RP, Bowes DR, Shirey SB: Inorganic particles found in cigarette tobacco, cigarette ash, and cigarette smoke. In: Biological Interaction of Inhaled Mineral Fibers and Cigarette Smoke. Wehner AP, Felton DL, Eds., Battelle Press, Columbus, OH, 421-439, 1989.

38. Nolan RP, Langer AM, Herson GB: Physico-chemical properties and membranolytic activities of the titanium dioxide polymorphs compared to quartz. In: Biological Interaction of Inhaled Mineral Fibers and Cigarette Smoke. Wehner AP, Felton DL, Eds., Battelle Press, Columbus, OH, 391-419, 1989.

39. Langer AM, Nolan RP: Mineral fibers in lung tissues of persons exposed to asbestos in the United States. In: VIIth Int'l Pneumoconiosis Conf, August 23-26, 1988.

40. Nolan RP, Langer AM: Physicochemical characteristics of quartz dust which controls its biological activity. In: VIIth Int'l Pneumoconiosis Conf, August 23-26, 1988.

41. Langer AM, Nolan RP: Distinguishing asbestiform tremolite from non-asbestiform tremolite. In: VIIth Int'l Pneumoconiosis Conf, August 23-26, 1988.

42. Nolan RP, Langer AM, Herson GB: Membranolytic activity of palygorskite and sepiolite. In: IVth Int'l Workshop: Effects of Mineral Dusts on Cells. Mossman BT, Begin RO, Eds., Springer-Verlag, Orford, Quebec, Canada, September, 37-48, 1989.

43. Langer AM: Chairman's Summary and Discussion. In: Biological Interaction of Inhaled Mineral Fibers and Cigarette Smoke. Whener AP, Felton DL (eds). Battelle Press, Columbus, Ohio. 590-593, 1989.

44. Langer AM, Nolan RP, Pooley FD,: Phyllosilicates:associated fibrous minerals. In: Health Related Effects of Phyllosilicates. J. Bignon, Ed., 59-74, Springer-Verlag, N.Y., 1990.

45. Langer AM, Nolan RP, Addison J: Physico-chemical properties of asbestos as

determinants of biological potential. In: Mineral Fibers and Health. Liddell FDS, Miller K, Eds., CRC Press, Ann Arbor, Michigan, 207-224, 1991.

46. Langer AM, Nolan RP, Addison J: Distinguishing between amphibole asbestos fibers and elongate cleavage fragments of their non-asbestos analogues. In: NATO Advanced Research Workshop on Mechanisms in Fibre Carcinogenesis. RC. Brown, J. Hoskins, N. Johnson, Eds., Albuquerque, New Mexico, October 22-25, 1990. 253-267. 1991.

47. Nolan RP, Langer AM, Oechsle GW, Addison J, Colflesh DE: Association of tremolite habit with biological potential. In: NATO Advanced Research Workshop on Mechanisms in Fibre Carcinogenesis. RC. Brown, J. Hoskins, N. Johnson, Eds., Albuquerque, New Mexico, October 22-25, 1990. 231-251. 1991.

48. Langer AM, Nolan RP: Zeolite catalysts. Is there a health risk? The erionite experience and its application to other zeolites. In: Chemicals and the Environment. Symposium on Chemical Specialties USA, Philadelphia. Spring Innovations, Ltd. 91-95, 1992.

49. Nolan RP, Langer AM: Limitations of the Stanton Hypothesis. Chapt. 9. Health Effects of Mineral Dusts, Reviews in Mineralogy, 28, Guthrie GD., Jr. and Mossman BT, Eds, Mineral. Soc. America, Wash., D.C., 309-326, 1993.

50. Ross M, Nolan RP, Langer AM, Cooper WC: Health effects of mineral dusts other than asbestos. Chap. 12. Health Effects of Mineral Dusts, Reviews in Mineralogy, 28, Guthrie GD., Jr. and Mossman BT, Eds, Wash., D.C., 361-407, 1993.

51. Langer AM, Nolan RP: Factors controlling the biological potential of inorganic dusts. Surface chemistry and character. In: Toxic and Carcinogenic Effects of Solid Particles in the Respiratory Tract, Mohr U, Dungworth DL, Mauderly JL, Oberdorster G, Eds., ILSI Monographs, ILSI Press, Wash. D.C., 147-157, 1994.

52. Nolan RP, Langer AM, Murray R: Correlation between exposure levels and dose-response to chrysotile asbestos. In: First All-Russian Practical Scientific Asbestos and Health. Asbest City, Urals Region, Russian Federation. 31-33.

53. Nolan RP, Langer AM: Physico-chemical properties of inorganic particles controlling biological activity. In: Carcinogenicity of Inorganic Substances. Risk From Occupational Exposure, J. H. Duffus, Ed., Roy. Soc. Chemistry. Bookcraft Ltd., G. B., 39-57, 1997.

54. Langer AM, Park MV: Party 1 Report: Physico-chemical characterization of exposures. In: Carcinogenicity of Inorganic Substances. Risks From Occupational Exposure. JH Duffus (ed). Roy Soc Chemistry, Bookcraft Ltd., U.K., 246-249, 1997.

55. Langer AM: The mineralogy of asbestos fibers in relation to causation. In: Asbestos

Medicine. Proceedings of the Conference. Defense Research Institute, Las Vegas, NV. 205-218, 1999.

56. Langer AM: A look at the mineralogy of asbestos. In: Essential Skills in Asbestos Litigation. Andrews Publications. Washington DC., 1-20. Nov 30-Dec 1, 2000.

57. Langer AM: The future of urban architecture. Will skyscrapers continue to be built? John Jay College of Criminal Justice. Homeland Security After 9/11. January 24, 2003. jjay.cuny.edu.

58. Langer AM: Characterization of fibrous minerals. Rapporteur Report. Session II. International Symposium on the Health Hazard Evaluation of Fibrous Particles Associated with Taconite and the Adjacent Duluth Complex. St. Paul, Minnesota. March 30 – April 1, 2003. In review.

59. Langer AM: The EPA, Risk and Fiber Type. In Proceedings of the Conference Defense Research Institute, Miami, FL. November 6-7, 2003.

60. Langer AM: The Mineralogy of the Silica Minerals. Properties as Determinants of Biological Activity. In Proceedings of the Conference Defense Research Institute, Atlanta, GA. June 10-11, 2004.

61. Langer AM: The Silica Minerals. Mineralogy and Properties. Silica Medicine Seminar. Atlanta, GA. June 16-17, 2005. Defense Research Institute, Chicago, IL..

LETTERS TO THE EDITOR:

1. Miller A, Teirstein AS, Langer AM, Selikoff IJ: Submicroscopical asbestos fibers and disease. N Engl J Med 292:1195-1196, 1975.

2. Wolff MS, Langer AM, Shirey SB: Gas chromatographs: health effects. Science 191:13, 1976.

3. Rom WN, Langer AM: Carcinogenicity of fibrous glass. West J Med 126:413, 1977.

4. Rohl AN, Langer AM, Selikoff IJ: Asbestos pollution. Science 197:716-718, 1977.

5. Rohl AN, Langer AM, Selikoff IJ: Chrysotile asbestos, effects of human exposure. Science 198:1202, 1977.

6. Fischbein AS, Rohl AN, Langer AM, Selikoff IJ: Pleura-und-peritoneal mesothelioma. Med Klin 74(19):742, 1979.

7. Fischbein AS, Langer AM, Rohl AN, Selikoff IJ: Asbestos disease in drywall construction workers. Am Industr Hyg Assoc 40(9):829-830, 1980.

8. Fischbein AS, Rohl AN, Langer AM, Selikoff IJ: Past asbestos exposure among

cable splicers. Am J Pub Health 71:1277-1278, 1981.

9. Langer AM, Selikoff IJ: Scientific data on chrysotile. Chem Engineer News 2:59, 1984.

10. Fischbein AS, Langer AM, Rohl AN: Asbestos-associated diseases: lessons from the past for the future. JAMA 254:1309-1310, 1985.

11. Langer AM, Nolan RP: Asbestos in play sand. N Engl J Med 316:882, 1987.

12. Langer AM, Nolan RP, Ross M: Asbestos policy. Science, 249:1485, 1990.

13. Langer AM, Nolan RP: Pleural mesothelioma resulting from exposure to amosite asbestos in a building. Respiratory Medicine 84:509-510, 1990.

14. Langer AM, Nolan RP, Addison J: On talc, tremolite and tergiversation. British Journal Industrial Medicine 48:359-360, 1991.

15. Langer AM, Nolan RP: The amphibole hypothesis: Neither gone nor forgotten. Am. J. Pub. Health, 87 (4) 688-689: 1997.

16. Langer AM, Nolan RP: Asbestos disease in foundrymen. J. Occup. Env. Med., 39, 699-700. 1997.

17. Langer AM, Non-occupational exposure to chrysotile asbestos and the risk of lung cancer. New Engl J Med. 1998, 339, No.14, 1000-1001.

18. Wilson R, Langer AM, Nolan RP: A risk assessment for exposure to glass wool. Regulatory Tox and Pharmacol. 33:103. 2001.

19. Roggli V, Langer AM: Asbestos in brakes: Comments regarding paper by Dr. R. Lemen. Amer J. Indust. Med. In press. 2005.

OTHER PUBLICATIONS:

1. Langer AM, Mackler AD: Mineral particles and human disease. In: Encyclopedia of Geochemistry and Environmental Sciences. Fairbridge RW (ed), Van Nostrand; Reinhold, NY, Vol. IVA, pp. 730-739, 1972.

2. Langer AM, Pooley FD: Mineralogy of asbestos minerals and methods of their characterization in human tissues. In: Asbestos: Volume 14 in the Chemical Carcinogenesis Series, Int'l Agency for Research on Cancer, p. 107, 1977.

3. Langer AM, Holaday D, Nicholson WJ, Rohl AN: Chapter 2. Asbestos Minerals: Nature, Occurrence and Properties, pp. 34-50. Chapter 4. Identification and Quantitation of Asbestos Fibers, pp. 71-100. Chapter 5. Environmental Distribution, pp. 102-134. In: Asbestos and Disease. Selikoff IJ, Lee WHK (eds), Academic

Press; NY, 1978.

4. Langer AM: Mineralogical factors in asbestos health effects. NSB SP506, Workshop on Asbestos: Definitions and Measurement Methods. Gravatt CC, LaFleur PD, Heinrich KFJ (eds), pp. 91-92;198, 1978.

5. Rohl AN, Langer AM, Wylie AG: Mineral characterization of asbestos-containing spray finishes. In: Asbestos-Containing Materials in School Buildings. US Environmental Protection Agency, Document EPA 450-2-78-014, US Government Printing Office; Washington, DC, pp. 59-64, 1979.

6. Langer AM: Electron microscopy and xray applications to environmental and occupational health analysis. Book Review. J Toxicol Environ Health, 1979.

7. Langer AM: Round-table discussion on talc as an asbestos substitute. Chairman's remarks. In: Proc Nat'l Workshop on Substitutes for Asbestos, Arlington, VA, Guimond RJ, Rowe JN, Levin A, Pillsbury H (eds), USEPA Office Toxic Substances, Washington DC, pp. 612-619, July 14-16, 1980.

8. Langer AM: Mineralogy of dust diseases. In: Maxcy-Rosenau Public Health and Preventive Medicine. Last J (ed), Appleton-Century-Crofts Publishers; NY, 11th Edition, pp. 637-641, 1981.

9. Langer AM: Host rocks and gangue materials in relation to pneumoconiosis and cancer. Editorial. Am J Industr Med 2:89-90, 1981.

10. Langer AM, Sebastien P: Physical-chemical properties of fibers. Rappateur Report. In: Biological Effects of Mineral Fibers, Int'l Agency for Research on Cancer, Wagner JC (ed), IARC-WHO, Lyon, France, 1:143-145, 1981.

11. Zoltai T, Langer AM: Chapter 2. Asbestiform fibers: historical background, terminology, and physicochemical properties. In: Asbestiform Fibers - Non-Occupational Health Risks. NAS-NRC, Nat'l Acad Sci, pp. 25-47, 1984.

12. Lynch J, Langer AM: Chapter 4. Measurement of exposure to asbestiform fibers. In: Asbestiform Fibers - Non-Occupational Health Risks. NAS-NRC, Nat'l Acad Sci, pp. 82-96, 1984.

13. Langer AM: Mineral dust and pulmonary lesions: etiological link or epiphenomenon? Editorial. Am J Industr Med 6:169-171, 1984.

14. Langer AM, et al.: Asbestos and other natural mineral fibers. In: Int'l Program on Chemical Safety. Environ Health Criteria 53, Chapters II, III, IV, V, WHO, Geneva, 1986.

15. Langer AM: Mineralogy. Chapter II. Presidential Report on Occupational Chest Diseases, Vol. 1, Characterization and Measurement of the Environment.

Merchant J, Dement J (eds), ALOSH-NIOSH-CDC; Washington, DC, pp. 3-40, 1987.

16. Langer AM: Asbestos. In: Collier's Encyclopedia, 3:4-5, 1987.

17. Langer AM: Chairman's Summary and Discussion. In: Biological Interaction of Inhaled Mineral Fibers and Cigarette Smoke. Wehner AP, Felton DL, Eds., Battelle Press, Columbus, OH, pp. 590-593, 1989.

18. Langer AM: Asbestos. In: Merit Students Encyclopedia, Macmillan Ed Pub, NY, pp. 270-271, 1990.

19. Langer AM: Chrysotile: The mineral and its properties. Summary of Presentation In: Health Risks Associated with Chrysotile Asbestos. Ann. Occup. Hyg., 38, 407. 1994.

20. Nolan RP, Langer AM: Mineralogy. Chapter 12. In: Occupational and Environmental Respiratory Disease, Harber P., Schenker M., Balmes J., Eds., Mosby Press, N.Y., 173-187, 1996.

21. Langer AM: The aluminum reclamation industry work environment: Are unidentified neurotoxins present? Editorial. Arch. Environ. Health, 53, No.5, 311-312. 1998.

PUBLISHED ABSTRACTS:

1. Berkley C, Langer AM, Berkley LE, Anderson CA: Chemical changes in inspired asbestos fibers. Microscopy Symp, McCrone Laboratory, Chicago, IL, August, 1966.

2. Berkley C, Langer AM, Selikoff IJ: Microbeam analysis and identification of the degradation products of inspired pulmonary particulates. Trans NY Acad Sci, May, 1967.

3. Langer AM, Kerr PF: The nature of soft and harsh chrysotile. In: Conference on the Physics and Chemistry of Asbestos Minerals, Oxford, Pr. 2-2, July, 1967.

4. Berkley C, Langer AM, Arneson A: Electron microprobe and electron diffraction analysis of asbestos bodies. In: 2nd Intl. Conf. Biol. Effects Asbestos, p. 7, Dresden, 22-25 April, 1968.

5. Berkley C, Langer AM, Rubin IB: Electron Microprobe analysis of particles in tissues. In: Proc 5th Nat'l Conf Elect Probe Soc Am, New York, Pr. 29A-29B, July, 1970.

6. Selikoff IJ, Nicholson WJ, Langer AM: Asbestos air pollution in urban areas. In: Proc AMA Air Pollution Med Res Conf, New Orleans, October, 1970.

7. Ehrenreich T, Langer AM, Selikoff IJ: Asbestos fibers in human lungs: forensic significance in environmental disease. In: 6th Int'l Mtg Forensic Sci, Edinburgh, Int'l Assn Forensic Sci, p. 86, September, 1972.

8. Teirstein AS, Miller A, Langer AM, Selikoff IJ: Sublight microscopic mineral particles in the etiology of pulmonary fibrosis. In: Proc Am Thorac Soc, New York, pp. 33-34, May 21-21, 1973.

9. Everett GA, Langer AM: Environmental contamination of Lake Superior asbestos from amphiboles in taconite wastes. In: Proc Ann Mtg Geol Soc Am, Miami, p. 728, November 18-20, 1974.

10. Rohl AN, Anderson H, Langer AM: Asbestos exposure during brake lining maintenance and repair. In: Proc Am Ind Hyg Assn, Miami, May 12-16, 1974.

11. Langer AM, Pooley FD, Bowes DR: Environment, disease and minerals. Read before the Geological Society, London, November 20, J Geol Soc, with discussion, 1974.

12. Langer AM, Wolff MS, Rubin IB: Environmental assay: particles in human tissues. In: Proc Int'l Conf Environ Sensing and assessment, Environmental Protection Agency, Pr. 29-34, September 14-19, 1975.

13. Bowes DR, Langer AM, Rohl AN: Nature and range of mineral dusts in the environment. Roy Soc London (Geological), p. 18, April 7, 1976.

14. Langer AM, Suzuki Y, Rohl AN: Identification of Microparticles. In: 3rd Intl. Symp. on Detection and Prevention of Cancer. ACS. New York, p. 91, April 26-May 1, 1976.

15. Fischbein A, Langer AM, Suzuki Y, Selikoff IJ: Carcinoma of the lung in a drywall taping worker: report of a case. In: Prog Abstr 3rd Int'l Symp Detect Prevent Cancer, No. 280, September 7, 1976.

16. Rom WN, Frank A, Suzuki Y, Langer AM, Selikoff IJ: Rapidly progressive interstitial pulmonary fibrosis from short term mixed dust exposure. 42nd An Sci Assem Am Coll Chest Phys, Atlanta, GA, October 21-28, 1976.

17. Fischbein A, Langer AM: Shale oil: A future energy alternative. A review of the mineralogic and carcinogenic properties of shale oil. Tenth Ann Conf Trace Substances in Environ Health, Columbia, MO, June 8-10, 1976.

18. Langer AM: Identification of microparticles in tissues. Workshop 13, 3rd Int'l Symp on Detection and Prevention of Cancer, September 1976.

19. Langer AM, Wolff MS: Asbestos carcinogenesis. In: Inorganic and Nutritional Aspects of Cancer, La Jolla, CA, January 2-5, 1977.

20. Selikoff IJ, Nicholson WJ, Langer AM: Epidemiological evidence on asbestos. Workshop on Asbestos, Definition and Measurement Methods, NBS Workshop, Gaithersburg, MD, July, 1977.

21. Langer AM: Contamination by submicroscopic particles in the biosphere. Int'l Symp Anal Electron Microscopy in Biol and Environ Routine and Research Work, Nat'l Inst Pub Health, Oslo, May 10, 1977.

22. Langer AM: Asbestos minerals and their carcinogenic effect. Int'l Symp Anal Electron in Biol and Environ Routine and Research Work, Nat'l Inst Pub Health, Oslo, May 10, 1977.

23. Langer AM, Oster G: Crystal faces and cleavage planes in quartz as templates in biological processes. Biophys Soc Am Phys Soc Mtg, Washington, DC, March, 1978.

24. Lilis R, Langer AM: Chronic interstitial pulmonary disease with severe respiratory disfunction due to unsuspected occupational beryllium exposure. In: XIXth Int'l Congr Occup Health, Dubrovnik, Yugoslavia, pp. 89-90, September 25-30, 1978.

25. Rohl AN, Langer AM: Asbestos and asbestiform minerals in non-asbestos mines in the United States. In: XIXth Int'l Congr Occup Health, Dubrovnik, Yugoslavia, pp. 121-122, September 25-30, 1978.

26. Russo DA, Kagan E, Langer AM, Yeager H: Human alveolar macrophages: cytotoxicity of naturally-occurring short fiber asbestos. Federation Proceedings, Section Physiology, Federation American Societies of Experimental Biology, 40 (3), 679, 1981.

27. Langer AM: Physical-chemical characteristics of chrysotile and their relationship to biological activity. In: 29th Ann Clay Minerals Conf, Baylor, Waco, TX, p. 58, October 5-9, 1980.

28. Bowes DR, Langer AM: Mineralogy and geochemistry of particulates in cigarette smoke. In: Ann Mtg Geol Soc, London, Environmental Mineralogy, January 11, 1980.

29. Langer AM: Particulate inhalants. In: Occupational Toxicology, College of Medicine and Dentistry, NJ, September 26, 1980.

30. Langer AM: Minerals as toxic agents: parameters and mechanisms. In: Proc 50th Geol Alumni Assn, Brooklyn College, CUNY, May 7-9, 1981.

31. Langer AM, Weisman, I, Adams A, Pooley FD: Characterization of crystalline particles in tissue by analytical electron microscopy. In: Symp on Crystalline Deposits in Tissues, Toronto, August 13-14, 1981.

32. Russo DA, Kagan E, Langer AM, Nolan R, Yeager H: Cytotoxicity of naturally-occurring short fiber asbestos. In: 20th Int'l Congr Occup Health, Cairo, September 25-October 1, 1981.

33. Ehrenreich T, Langer AM, Selikoff IJ: Algorithm of pathologic diagnosis of asbestos-related diseases for forensic purposes. Significance of quantitative and morphological correlates. In: 20th Int'l Congr Occup Health, Cairo, September 25-October 1, 1981.

34. Rohl AN, Langer AM, Suzuki Y, Lilis R, Moncure G, Selikoff IJ: Endemic pleural disease associated with mixed fibrous dust exposure in Turkey. In: 20th Int'l Congr Occup Health, Cairo, September 25-October 1, 1981.

35. Nolan RP, Langer AM: Surface functionalities of quartz and hemolytic activity. In: Proc 2nd Int'l Workshop of the In Vitro Effects of Mineral Dusts, Arkadelphia, AR, April 5-6, 1982.

36. Langer AM, Nolan RP: Membrolytic properties of quartz. In: Proc 2nd Ann Geol Alumni Assn, Brooklyn College, CUNY, p. 4, April 28-30, 1983 (abstract).

37. Nolan RP, Langer AM, Foster KW: Recognition of quartz by erythrocyte membranes. In: 6th Int'l Penumoconiosis Conf, Bochum, FDR, September 20-23, 1983 (Abstract 198).

38. Nolan RP, Langer AM, Foster KW: Physico-chemical factors effecting membrolytic properties of quartz. In: 6th Int'l Pneumoconiosis Conf, Bochum, FDR, September 20-23, 1983 (Abstract 199).

39. Langer AM, Nolan RP: Surface properties of quartz and membrane activity. In: Int'l Symp Silica, Silicosis and Cancer, Chapel Hill, NC, April 3-5, 1984.

40. Langer AM: Asbestos, fibrous minerals, acicular cleavage fragments and the mineral industries. In: Int'l Congr Applied Mineralogy, Los Angeles, February, 1984.

41. Langer AM: Minerals and disease: the association between exposure to minerals and rock dust and the occurrence of human disease. In: Int'l Congr Applied Mineralogy, Los Angeles, February, 1984.

42. Langer AM: Mineral fiber in water? In Medical Geology Conference on Health Threatening Toxins in Water, Brooklyn College, NY, May, 1984.

43. Constantopoulos SH, Langer AM, Saratzis N, Goudevenos JA, Selikoff IJ: Metsovo lung: pleural calcifications and malignant pleural mesothelioma in northwestern Greece. Tremolite asbestos used for whitewashing as etiology. In: 3rd Int'l Conf Environmental Lung Disease, Am Coll Chest Phys, Montreal, Canada, October 15-18, 1986. Chest 91(2):300, 1987.

44. Langer AM, Nolan RP: Fiber type and parenchymal burden found in workers occupationally exposed to asbestos fiber in the United States. Implications for risk assessment in the general population. Mineral Fiber in the Non-Occupational Environment, Lyon, France, October 8-10, 1987.

45. Langer AM, Nolan RP: Mineralogical and biological comparison of asbestiform and non-asbestiform tremolite. AIME Annual Meeting, Phoenix, AZ, January 25-28, 1988.

46. Langer AM, Nolan RP, Bowes DR, Shirey S: Inorganic particles in cigarettes and cigarette smoke. In: Biological Interaction of Inhaled Mineral Fibers and Cigarette Smoke. International Symposium/Workshop, Seattle, WA, April 10-14, 1988.

47. Nolan RP, Langer AM, Herson GB: Physico-chemical properties of the titanium dioxide polymorphs and biological activity. In: Biological Interaction of Inhaled Mineral Fibers and Cigarette Smoke. International Symposium/Workshop, Seattle, WA, April 10-14, 1988.

48. Nolan RP, Langer AM, Herson GB: Membranolytic activity of palygorskite and sepiolite. In: IVth International Conference on the Effects of Mineral Dusts on Cells, p. 15, Oxford, Quebec, Canada, 1988.

49. Langer AM, Nolan RP, Herson GB: Membranolytic activity and physicochemical characterization of phosphorylated Canadian chrysotile. In: IVth International Conference on the Effects of Mineral Dusts on Cells, p. 14, Oxford, Quebec, Canada, 1988.

50. Nolan RP, Langer AM, Herson GB: Physicochemical character-istics of quartz dust which controls its biological activity. In: VIIth International Pneumoconiosis Conference, DHHS (NIOSH) Publication No. 90-108, Part I, 754, 1990.

51. Langer AM, Nolan RP: Fiber type and risk of mesothelioma to building occupants. In: Health Aspects of Exposure to Asbestos in Buildings. Energy and Environmental Policy Center, John F. Kennedy School of Government, Harvard University, Cambridge, MA, December 14-16, 1988.

52. Langer AM, Pooley F D, Nolan RP: Phyllosilicates: Associated fibrous minerals. In: First International Conference on Health Related Effects of Phyllosilicates. NATO Advanced Research Workshop, Paris, France, March 16-18, 1989.

53. Nolan RP, Langer AM, Herson GB: Characterization of palygorskite specimens from different geological locales for health hazard evaluation. In: First International Conference on Health Related Effects of Phyllosilicates. NATO Advanced Research Workshop, Paris, France, March 16-18, 1989.

54. Langer AM, Nolan RP: The importance of mineral subpopulations in biological assays. In: NATO Advanced Research Workshops on Mechanisms in Fibre Carcinogenesis. Albuquerque, New Mexico, October 22-25, 1990. 49-50.

55. Nolan RP, Langer AM, Oechsle GW, Addison J, Colflesh DE: Association of tremolite habit with biological potential. In: NATO Advanced Research Workshops on Mechanisms in Fibre Carcinogenesis. Albuquerque, New Mexico, October 22-25, 1990. 57.

56. Langer AM: Fiber types, fiber sizes, and asbestos diseases. Andrews Communications Seminars. 2nd Asbestos Litigation. The Eye of the Storm. 25-26 February, 1991. Bal Harbour, Florida.

57. Nolan RP, Langer AM, Oechsle GW, Johnson NF: Physicochemical characterization of selected zeolite minerals for health hazard evaluation. Fourth Intl. Conf. Env. Lung Disease. Montreal, Canada, September 25-28, 1991.

58. Nolan RP and Langer AM: Characterization of fibers for Health Hazard evaluation. Critical Research Needs. Workshop on Chemical and Biological Interactions of Glass. Bethesda, MD, 5-6 Mar, 1992.

59. Langer AM and Nolan RP (1992). Comparison of lung tissue mineral fibre retention of exposed workers and the general population. In: Biopersistence of respirable synthetic fibres and minerals. 7-9 Sept, 1992. Lyon. p. 26.

60. Nolan RP, Langer AM, and Addison J (1992). Health hazard evaluation of the lung tremolite fiber content among Canadian chrysotile workers. In: Biopersistence of respirable synthetic fibres and minerals. 7-9 Sept, 1992. Lyon. p. 52.

61. Langer AM, Nolan RP (1993). Factors controlling the biological potential of inorganic dusts: surface character and chemistry. 4th International Inhalation Symposium, Hannover. Toxic and Carcinogenic Effects of Solid Particles in the Respiratory Tract, 1-5 March, 1993, Hannover. Abst. A-10, p. 48.

62. Langer AM, Nolan RP (1993). Physico-chemical characteristics of quartz dust which controls its biological activity. Clay Minerals Society Meeting. San Diego, 25-30 Sept, 1993.

63. Nolan RP, Langer AM (1995). Evaluation of the health hazards of asbestos in a mine. Goldschmidt Symposium, Penn State University, May 24-26, 1995, p. 75.

64. Langer AM, Nolan RP, Herson GB (1995). Chemically modified chrysotile asbestos: Physical-chemical characterization and membranolytic activity of phosphorylated Canadian chrysotile. Goldschmidt Symposium, Penn State University, May 24-26, 1995, p. 63.

65. Langer AM, Nolan RP (1995). Physical chemical properties of fibers other than asbestos used in global commerce. Symposium on the Health Effects of Fibrous Materials used in Industry Excluding Asbestos. Sydney, Australia, October 30-31, 1995, p. 12.

66. Nolan RP, Langer AM, Murray R (1996). The exposure response relationship of chrysotile asbestos. Conference on Asbestos and Health, Asbestos City, Russian Federation. June 4-7, 1996.

67. Nolan RP, Langer AM, Murray R, Grinberg LM, Kogan FM, Tregubov ES, Kuschner M (1997). Pulmonary fibrosis and lung content analysis of Russian chrysotile workers. Preliminary results. 6th International Inhalation Symp., "Relationship Between Respiratory Disease and Exposure to Air Pollution." Hannover, February 24-28, 1997.

68. Langer AM (2003) Control and monitoring of asbestos in the United States. Regulatory overview of asbestos and other fibrous minerals. International Symposium on the Health Hazard Evaluation of Fibrous Particles Associated with Taconite and the adjacent Duluth Complex. St. Paul Minnesota, March 30 – April 1, 2003, p. 45-46.

MAJOR PROJECT REPORTS:

1. Report to the U.S. Consumer Products Safety Commission: Mineralogical Analysis of Two "Play Sands" For Their Asbestos Contents. Langer AM, Nolan RP. 26 November, 1986. 9p., 4 Tables, 18 plates.

2. Chrysotile and chrysophosphate. A comparative study of their physicochemical properties and membrane activities. A report to SNA-Chrysophosphate, Canada. Langer AM, Nolan RP, p. 87, June 1, 1987.

3. Report to the U.S. Consumer Product Safety Commission: Mineral Analysis of a Carbonate Play Sand. Langer AM, Nolan RP. July, 1987. 19 p., 3 Tables.

4. Wollastonite in the pulmonary tissues on animals. A report to Northrop Services Industries, National Toxicology Program. Langer AM, Nolan RP, p. 101, June 15, 1989.

5. Preparation, examination and characterization of mineral standards by analytical electron microscopy. A report to Battelle Columbus Laboratories, Langer AM, Nolan RP, Pooley FD, Gieseke JA, Fisher G, 61 p., December 12, 1989.

6. Comparison and evaluation of the analytical capabilities of three U.S. laboratories for submicroscopic particulate analysis. A report to Battelle Columbus Laboratories, Langer AM, Nolan RP, Gieseke JA, Fisher G. February, 1991.

7. Report on the measurement of fiber release from simulated steam-stressed Durabla gaskets (120E, 160E, 190E for 5 hours) for health hazard evaluation. A report to Durabla, Inc., Langer AM, Nolan RP, 21 p., January 31, 1992.

8. Measurement of fiber release from simulated steam-stressed Durabla gaskets for

health hazard evaluation. A report to Durabla, Inc., Langer AM, Nolan RP, (revision 1/31/92 report) 29 p., July 16, 1992.

9. Report on the measurement of fiber release from simulated steam-stressed Durabla gaskets (120EC, 160EC, 190EC for 5 days) for health hazard evaluation. A report to Durabla, Inc., Langer AM, Nolan RP, 23 p., November 6, 1992.

10. Report on the measurement of fiber release from simulated steam-stressed Durabla gaskets (250EC, 30 days) for health hazard evaluation. A report to Durabla, Inc., Langer AM, Nolan RP, 25 p., December 9, 1992.

11. The Identification and Quantitation of Small Asbestos Fibres in Injectable Medicines. Technical Expert Report Prepared for Hoffman-LaRoche, Basel. 16 p. Addison J, Burdett GJ, Langer AM, Muhle H. 1993.

12. Analysis of rockwool fibers by scanning electron microscopy. A report to Lapinus Fibres, B.V., Netherlands. Nolan RP, Langer AM, 13 p., November 29, 1993.

13. Analysis of rockwool fibers by scanning electron microscopy. A report to Lapinus Fibres, B.V., Netherlands. Nolan RP, Langer AM, 25 p., November 29, 1993.

14. Report on Chrysotile Fibers Released from the Manipulation of Asbestos Packings. Nolan RP, Langer AM. 3p. 2Figs. November 30, 1993.

15. Release of Rockwool Fibers From Compressed Gasket Material. A report to Lapinus Fibres, B.V., Netherlands. Nolan RP, Langer AM, 38 p., Appendix, January 31, 1994.

16. Analysis of Quarry Sample #01, #08 and #18 for asbestos content. The Readymix Group, Australia. Nolan RP, Langer AM, 39 p., March 9, 1994.

17. Report on Air Samples From Pine Lake Elementary School, Bloomfield, Michigan. Asbestospray Corporation. Langer AM, Nolan RP. 2p., 44 p Appendix, 2 Tables. July 14, 1994.

18. Analytical Method for the Determination of asbestos Minerals in Parenteral Medicines. Der Verband der Chemischen Industrie. Addison J, Burdett GS, Chatfield EJ, Christiansen CP, Gotz J, Kaiser D, Kurz G, Langer AM, Muhle H, Wachberger E. 76p. August, 1994.

19. Report of the Examination of Australian Quarry Dust and Mineral Specimens. The Readymix Group, Australia, Nolan RP, Langer AM, Pooley FD, 12 p., 18 figs., October 28, 1994.

20. Health Hazard Evaluation of the CD #1 Pit Empire Mine, Palmer, Michigan. A report to Cleveland-Cliffs Corp., Nolan RP, Langer AM, Wilson R, 90 p., January, 1995, February 17, 1995.

21. Environmental Analysis of Four Tailing Surface Samples for Arsenic and Asbestos. Nolan RP, Langer AM, Pooley FD, 41p., March 20, 1996.

22. A Risk Assessment for Installers of Blown Glass Wool Insulation. Prepared for the Blown Glass Wool Contractors, U.S. Wilson R, Nolan RP, Langer AM, 57 p. Text,16 Tables, Appendix, April 16,1996.

23. Is Respirable Fine Crystalline Silica Present in Polyethylene Dust? A Report to Novacor Chemicals, Canada. 39 p., 3 Tables; 28 Figures, June 15, 1996.

24. Comments on the National Toxicology Program's Draft Report on Carcinogens. Talc Luzenac. Background Document for Talc Asbestiform and Non-Asbestiform. Addison J, Langer AM. November 2000. 15p.

Books Edited

Carcinogenicity of Inorganic Substances. Risks from Occupational Exposure. The Royal Society of Chemistry. Cambridge, England. 286 p. 1997. Co-Editor.

The Health Effects of Chrysotile Asbestos. Contribution of Science to Risk Management Decisions. Mineralogical Society of Canada. Canadian Mineralogist Special Paper No. 5. 398 p. 2001. Co-Editor.

INTERNATIONAL ORGANIZATION DOCUMENTS:

1. Evaluation of Carcinogenic Risk of Asbestos. Volume 14. Asbestos. Series. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. Intl Agency for Res. on Cancer - WHO, Lyon. 106 p. 1977. Co-Author.

2. Asbestiform Fibers - Non-Occupational Health Risks. National Academy of Sciences. National Research Council. National Academy Press, 334 p., 1984. Co-Author.

3. International Programme on Chemical Safety (IPCS). WHO. Environmental Health Criteria 53: Asbestos and Other Natural Mineral Fibres. Hannover, Germany. 194 p., 1986. Co-Author.

4. Silica and Some Silicates. Volume 42. Series IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. Intl Agency for Res. on Cancer - WHO, Lyon. 289 p., 1987. Co-Author.

5. Asbestos in Public and Commercial Buildings: A Literature Review and Synthesis of Current Knowledge. Health Effects Institute, Cambridge, MA and U.S. Environmental Protection Agency. 300 p., 1991. Co-Author.

6. International Programme on Chemical Safety (IPCS), WHO. Workshop on Health Risks Associated With Chrysotile Asbestos. Isle of Jersey. Ann. Occup. Hyg., 38,

397-646. 1995. Co- Author.

7. International Programme on Chemical Safety (IPCS), WHO. Environmental Health Criteria Document For Chrysotile. Geneva, Switzerland - July 1-6, 1996. Co-Author.

MANUSCRIPTS IN PREPARATION:

Langer AM: Distribution of amosite and chrysotile fibers in the lung of an exposed worker: role of fiber size and type.

Langer AM, et al: Amosite fiber size-distribution at a fabrication plant: mesothelioma and fiber dimension.

Langer AM, et al: Asbestos bodies in two periods of time: review, secular trends, New York City, 1915-1970.

POSTGRADUATE EDUCATION COURSES:

Page and Black Postgraduate, Mount Sinai School of Medicine: Asbestosis. June, 1970

NY Academy of Sciences, Seminars for Trade Union Representatives: Occupational Health Hazards. April, 1971

American College of Chest Physicians, Mount Sinai School of Medicine, NY: Interstitial Pneumonias--Acute and Chronic. April, 1971

United States-USSR Onco-Epidemiology Delegation: Workshop on Basic Practical Approaches to Environmental Carcinogenesis, Mount Sinai. February 12, 1975

Seminar Series. Division of Natural Sciences. Agents in the Environment. May, 1974.

Page and Black Postgraduate, Mount Sinai School of Medicine: Environmental Lung Disease. June, 1976

Office of Continuing Education, Baylor Medical College, Houston, TX: Pulmonary Diseases and Carcinoma of the Lungs, Baylor Medical School, Houston, TX. February, 1977

New York Lung Club, Cornell University Medical College: Silicosis revisited. New York City Tunnel and Caisson Workers: 1928-1977. November, 1977

Page and Black Postgraduate, Mount Sinai School of Medicine, New York, NY: Recent Advances in Occupational Medicine. January, 1978

Page and Black Postgraduate, Mount Sinai School of Medicine, New York, NY:

Occupational and Environmental Pulmonary Diseases. March, 1978

Page and Black Postgraduate, Mount Sinai School of Medicine, NY: Health Effects of Asbestos Exposure. Course Director. June, 1978

Page and Black Postgraduate, Mount Sinai School of Medicine, NY: Asbestos Associated Diseases: With Particular Reference to United States Shipyards. November, 1978

Page and Black Postgraduate, Mount Sinai School of Medicine, NY: Scientific Basis for Evaluation of Occupational and Environmental Asbestos Disease. January 1979

Office of Continuing Education, Baylor Medical College, Houston, TX: Asbestos Symposium. Asbestos-Associated Diseases, Houston, TX. October, 1979

Page and Black Postgraduate, Mount Sinai School of Medicine, New York, NY: Asbestos in Shipyards. January, 1980

Page and Black Postgraduate, Mount Sinai School of Medicine, New York, NY: Management and Control of Asbestos in Public Buildings. March, 1980

Page and Black Postgraduate, Mount Sinai School of Medicine, New York, NY: Health Effects of Asbestos in Public Buildings. June, 1980

College of Medicine and Dentistry of New Jersey: Rutgers Medical School, Recent Advances in Occupational Toxicology, Graduate Center, NY. September, 1980

Page and Black Postgraduate, Mount Sinai School of Medicine, New York, NY: Recent Advances in Occupational Medicine. November, 1981

Page and Black Postgraduate, Mount Sinai School of Medicine, New York, NY: Scientific Basis for Evaluation of Asbestos-Associated Diseases. December, 1982

Mount Sinai School of Medicine Electives Program. Department of Community Medicine. 1984-1985. Mineralogy of Biologically Active Inorganic Particles. Laboratory Studies.

Page and Black Postgraduate, Mount Sinai School of Medicine, New York, NY: Asbestos in Schools and Public Buildings, Hasbrook Heights, NJ. March, 1985

Environmental Lung Disease. American College of Chest Physicians. Mineralogy Techniques. Montreal, Canada. September, 1991

Environmental Geology Seminar. Graduate Center - CUNY. September, 1993-February, 1994; 1994-1995; 1995-1996; 1996-1997; 2000-2001; 2003; 2004.

Current course listing in the Graduate School include seminars in Earth and Environmental Sciences; Interdisciplinary Concentration in Health and Society; Public Policy and Urban Studies.

INVITED SEMINARS AND LECTURES:

Lawrence Radiation Laboratory, Livermore California. Crystallography Division. "Analysis of Ultra-fine Mineral Dust". June, 1964.

Union College, Schenectady, NY, Department of Geology and Civil Engineering. "Asbestos and Lung Disease." April, 1967.

Barnard College, NY, Department of Geology. "Asbestos Fiber in the Ambient Air and Disease Potential." December, 1968.

Food and Drug Administration, Washington DC. "Asbestos Bodies in the General Population – Fibers in Talc as Possible Sources of Exposure." May, 1968.

The Rockefeller University, Air Pollution Working Group, Health Research Council. "Analysis of Air Samples for Inorganic Constituents." June 9, 1969.

New York University Medical Center, New York Lung Club. "Sublight Microscopic Mineral Particles in Talc Pneumoconiosis. Physiology and Mineralogical Studies." November, 1970.

Leheigh College, Department of Material Sciences and Metallurgy. "Electron Probe Characterization of Particles in Tissues." April, 1971.

Oberlin College, OH, Department of Geology. "Asbestos Dust in the Workplace and Human Disease." May, 1971.

Department of Housing and Urban Development. Asbestos and Disease. New York. August, 1971.

State University of New York at Buffalo, Department of Biophysical Sciences. "The Asbestos Problem. An Interdisciplinary Approach." April, 1972.

Queens College of the City University of New York, Department of Earth and Environmental Sciences. "Asbestos and Disease. How Wide is the Spectrum?" November, 1972.

Columbia University, NY, Department of Geology and Biological Sciences. "Contamination of the Environment with Mineral Particles." December, 1972.

College of South Hampton, Long Island University, Division of Chemical Sciences and Oceanography. " The Contamination of Lake Superior with Fibrous Silicates." January, 1973.

National Institutes of Health. Biological Effects of Asbestos. "Mechanism of Fiber Action." National Institutes of Health, Bethesda, MD, February 1, 1973.

City College of the City University of New York, Department of Earth and Planetary Sciences. "Mineral Particles and Human Disease." May, 1973.

Queens College of the City University of New York, Department of Health Sciences. "Occupational Hazards as Harbingers of Environmental Hazards." September, 1973.

Rutgers University, Colonial Conference Tri-State Environmentalists. "Recognition of Environmental Hazards: Use of Academic Facilities for Monitoring and Control of Hazards." November, 1973.

Johns Hopkins University, School of Public Health. "Asbestos Minerals and Disease." Seminar. April, 1974.

Rutgers University, Department of Environmental Sciences. "Mineral Particles in the Air." June, 1974.

Mt. Sinai Pathology Seminar Series. Studies of Minerals in Disease. With J. Churg. May, 1974.

Temple University, Department of Geology. "Amphibole Contamination of Lake Superior Water from Taconite Processing." September 20, 1974.

Mt. Sinai Physical Medicine and Rehabilitation. Environment Hazards. January 8, 1975.

National Cancer Institute. Soviet Onco-Epidemiology Delegation. Mineralogy of Asbestos. Mt. Sinai, New York. February 12, 1975.

Rutgers University. Department of Environmental Science. Occupational Health 1975: Status and Prognosis. February 25, 1975.

New York Lung Club. Cornell Medical Center. A Silicosis Revisited. New York City Tunnel and Caisson Workers - 1928-1977. September 25, 1977.

American Museum of Natural History, Department of Mineral Sciences. "Minerals and Disease." September, 1978.

Rutgers University, Department of Chemistry. "Minerals and Their Biological Activity." October, 1978.

University of North Carolina, Department of Medicine. 1) "Analytical Methods of Tissue Analysis, Asbestos Bodies and Asbestos Fibers"; 2) "Inorganic Particles in Cigarettes and Cigarette Smoke." November, 1978.

Lutheran Medical Center. Current Perspectives in Environmental Health Hazards. Pulmonary Dust Burden and Response to Inorganic Particles. February 8, 1979.

University of Maine, Farmington, Department of Geology. 1)"The Asbestos Problem"; 2) "Evolution of an Environmental Scientist, A Personal View." March, 1979.

National Institutes of Health. Forms of Asbestos of Public Health Concern. Scientific Basic For Public Health Control of Environmental Asbestos Hazards. The Physical-Chemical Nature of Asbestos. Factors Relating to Biological Activity. Mazur Auditorium, Bethesda, Maryland. March 29, 1979.

School of Public Health, Columbia University. "Epidemiology of Asbestos Diseases." May 7, 1979.

New York Medical Examiner's Conference. "Analysis of Tissues for Asbestos Fiber and Asbestos Bodies." June, 1979.

Seton Hall University, Department of Chemistry. "Minerals and Disease: Interaction Mechanisms." October 2, 1979.

American Institute of Mining Engineers, Tucson. "The Biological Effects of Inorganic Fibers: The Case Against Asbestos" (invited speaker). October, 1979.

Geological Society of London. "Mineralogy and Geochemistry of Particulates in Cigarette Smoke", with Dr. Bowes. January 11, 1980.

American Chemical Society, Staten Island Section. "Minerals and Diseases: Physical and Chemical Factors." February 19, 1980.

Society of Sigma Xi Lecture. "Man in Conflict with His Physical Environment." Invited Speaker for Initiation Ceremony, Hunter College of the City University of New York, May 13, 1980.

Universities Occupational Safety and Health Educational Resource Center. Particulant Inhalants In: Recent Advances in Occupational Toxicology. NIOSH.
26 September, 1980.

National Institutes of Health, Bethesda, Department of Health and Human Services CCERP-26. "Serpentine-Containing Host Rocks: Amphibole-Containing Host Rocks." November, 1980.

Economic Geology Chapter Queens College of the American Institute of Mining, Metallurgical and Petroleum Engineers. " Conflict Between Man and the Mineral World: Asbestos." March 26, 1981.

NIEHS-EPA-NIOSH Joint Committee at Bethesda. "Mount St. Helen's Volcanic Ash: Mineral Nature and Biological Activity." July 23, 1981.

The Rockefeller University. "Minerals as Toxic Agents: Important Parameters and Mechanisms." Invited speaker in series on Comparative Toxicology, February 12, 1981.

Kettering Laboratory, University of Cincinnati Medical Center. "Silica and Silicosis: Mechanisms." April 8, 1981.

University of California, Irvine, Occupational and Environmental Health Center. "Physicochemical Properties of Inorganic Dusts and Biological Consequences of Exposure." Occupational Medicine Series, December 2, 1981.

Barlow Hospital, Los Angeles, University of Southern California Medical School. "Tissue Analysis for Asbestos, Comparative Techniques." Chest Service, Department of Medicine, December 3, 1981.

Appalachian Laboratory for Occupational Safety and Health NIOSH-CDC, Morgantown, West Virginia. "Surface Properties of Silica and Its Biological Activity." February 24, 1982.

National Institute for Occupational Safety and Health. "Silica and Silicosis." Cincinnati, March 24, 1982.

Society of Sigma Xi Lecture, Queens College of the City University of New York. "The Case Against Chrysotile Asbestos." December 2, 1982.

National Institute of Environmental Health Sciences, Pathobiology of Mesothelioma, Research Triangle Park, NC. "Physicochemical Character of Dust and its Role in Inducing Human Mesothelioma." January, 1983.

Geology Seminar, Long Island University, College at South Hampton. "The Biological Effects of Asbestos." March 18, 1983.

Graduate Seminar, Rutgers University, Department of Geology. "Physical-Chemical Properties of Minerals and Their Biological Activities." April 13, 1983.

Symposium on Fibrous Minerals, American Lung Association Annual Meeting, Kansas City, MO. "Mineralogy of Mineral Fibers." May 8, 1983.

Faculty Research Collegium, Ph.D. Program Earth and Environmental Studies, CUNY, Graduate Center, NY. "Physicochemical Properties of Minerals Controlling Biological Activity." March 6, 1984.

Graduate Seminar, Department of Geology, Princeton University. "Minerals and Disease." Princeton, NJ, March 8, 1984.

Graduate Seminar, Department of Biology, New York University, New York, NY. "Asbestos: Biological Considerations." April 16, 1984.

Medical Geology Conference on Health Threatening Toxins in Water. "Asbestos Fiber in Water." May 3, 1984.

Asbestos Information Association of North America, Annual Meeting, Alexandria, VA. "Asbestos Substitutes. How Safe are They?" September 18, 1985.

American Society for Testing Materials, Annual Meeting, Bal Harbour, FL. "Method for Testing Indoor Air for Asbestos. Analytical Electron Microscopy." November 4, 1985.

University of Southern California Medical School. "Membranolytic Properties of Quartz and Phosphorylated Chrysotile Fiber." January 29, 1985. Main Campus. "Asbestos Fiber in Lung Tissue - Analysis by Analytical Electron Microscopy." January 30, 1986.

Mine Health Research Advisory Committee. CDC-NIOSH. The Mineralogy of the Asbestos Minerals. Tuscon, Arizona. May 29, 1986.
Milton Kannerstein Lecture. "Mineral Dust and Pulmonary Lesions." Annual Meeting of the Klemperer-Otani Society. Mount Sinai, June 5, 1986.

Northeast Regional Environmental Public Health Center, University of Massachusetts. Symposium: Asbestos in Play Sand. "Tremolite Analysis, Morphology, and Health Significance." February 11, 1987.

The Defense Research and Trial Lawyers Association, Reno, NV. "Analytical Protocol for the Study of Sublight Microscopic Particles in Human Tissues"; "Types and Amounts of Asbestos Fibers in the Pulmonary Tissues of Asbestos-Exposed Workers in the United States." October 30, 1987.

The Manville Settlement Trust Asbestos Disease Seminar, Washington, DC. "Asbestos Fibers: The Physico-Chemical Properties and Health Effects." March 16, 1988.

Electron Microscopy Society of America, Milwaukee, Wisconsin. "Electron Diffraction of Mineral Fibers v. Cleavage Fragments." August 9, 1988. Invited Speaker.

Asbestos Fiber Type and Risk of Disease, Conference: New York - New Jersey Environmental Expo: The Source for Environmental Solutions. Seminar 7E, The Management of Asbestos in the 1990's. Thursday, October 18, 1990.

American Industrial Hygiene Association, Georgia Section, Atlanta Georgia. Conference: The Asbestos Controversy. Has the Public Been Properly Informed? "Understanding the Asbestos Risk." November 30, 1990.

Armstrong Defense Group. Memphis, Tennessee. The Asbestos Saga and the Star Wars Trilogy: The Empire Strikes Back! Thursday, April 25, 1991.

Department of Medicine - Pulmonary, New York University Medical Center. Pulmonary Grand Rounds. Particle Characteristics Responsible for Pneumoconiosis and Cancer. Tuesday, September 3, 1991.

Nelson Institute of Environmental Medicine, New York University School of Medicine.

Surface Properties of Minerals as Determinants of Biological Activity. Tuxedo, New York. Wednesday, October 23, 1991.

Defense Research Institute. Current Issues in Asbestos in Buildings Seminar. Report From the Health Effects Institute. Orlando, Florida. Friday, January 24, 1992.

Characteristics of Mineral Fibers - Critical Research Needs. Invited to Workshop on Chemical and Biological Interactions of Glass. Bethesda, Maryland, March 5-6, 1992.

Mealey's National Lead Litigation Conference. Presented: Bioavailability of Lead. April 22-23, 1993. Philadelphia, Pennsylvania.

McGrath Fund Lectures. Current Topics in Environmental Science. Division of Natural Sciences, South Hampton Campus of Long Island University. Update on Asbestos. A Asbestos Risk Assessment. May 3, 1994.

United States Environmental Protection Agency. Environmental Asbestos Workshop: Chrysotile Asbestos. Comparative carcinogenicity with the amphibole asbestos varieties. Monterey, California. October 7, 1994.

Defense Research Institute Symposium. Asbestos Medicine Seminar. Health risks associated with low-level asbestos exposures. (with Dr. B. Gee). New York. October 26-28, 1994.

Environmental Business Association of New York State. Environmental Research in New York State. April 26, 1995.

McGrath Fund Lectures. Current Topics in Environmental Science. Division of Natural Sciences, South Hampton Campus of Long Island University. United Nations Conference on the Environment and Development. May 3, 1995.

Lecture Series Department of Chemical Engineering, City College of the City University of New York. Inorganic Dust in the Environment. Risk Analysis. February 1, 1996.

City University Graduate Center. Asbestos and Radon: Dangers of Diversions? Graduate Center, April 10, 1996.

McGrath Fund Lectures. Current Topics in Environmental Science. Division of Natural Sciences, South Hampton Campus of Long Island University. Man-made Vitreous Fibers as Asbestos Substitute. Are They Safe? April 24, 1996.

McGrath Fund Lectures. Current Topics in Environmental Science. Division of Natural Sciences, South Hampton Campus of Long Island University. Epidemiology and Human Studies of PCBs. Is there a risk? April 29, 1998.

Defense Research Institute. Asbestos Seminar. The Mineralogy of Fibers and Its Relation

to Asbestos Causation. November 11-12, 1999. Las Vegas, Nevada.

McGrath Fund Lectures. Current Topics in Environmental Science. Division of Natural Sciences, South Hampton Campus of Long Island University. "Is South Hampton Surrounded by a Sea of Carcinogens?" April 26, 2000.

Andrews Essential Skills in Asbestos Litigation. A Look at the Mineralogy of Asbestos. November 30, 2000. Washington DC.

John Jay College of Criminal Justice and the United States Federal Emergency Management Plan (FEMA). "The Future of Urban Architecture" January 23-24, 2003.

McGrath Fund Lectures. Current Topics in Environmental Science. Division of Natural Sciences, South Hampton Campus of Long Island University. "Is Global Warming a Factor in the Spread of Infectious Disease?" May 7, 2003.

Defense Research Institute. "The Environmental Protection Agency. Revisiting the Issue of Fiber Type and Properties." November 6-7, 2003. Miami, FL.

American Industrial Hygiene Association. St. Louis Chapter. "The US EPA. Looming Policy Changes." January 23, 2004. St. Louis, MO.

Asbestos Cement Products Producers Association International. "Asbestos-Containing Friction Products. Is There a Risk in Use? Miami, Florida. April 19-20, 2004.

Defense Research Institute. "The Mineralogy of Silica." Atlanta, Georgia. June 10-11, 2004.

Defense Research Institute. "The Mineralogy and Properties of Crystalline Silica." Atlanta Georgia. June 16-17, 2005.

Defense research Institute. "Tremolite. Mineralogy, Habits, and Biological Activity." Miami Florida. November 2-4, 2005.

INVITED INTERNATIONAL MEETINGS AND CONFERENCES:

Physics and Chemistry of Asbestos Minerals. Oxford, July, 1967. Presented: The Nature of Soft and Harsh Chrysotile (with Kerr PF.).

Second International Conference on the Biological Effects of Asbestos. Dresden, DDR, April, 1968 (two papers with Berkley C, Sastre A, Arneson A and Schwartz J).

International Conference on Pneumoconiosis. Johannesburg, April 23-May 2, 1969. Presented: Electron Microprobe Analysis of Asbestos Bodies (with Rubin IB and Selikoff IJ).
Working Group: Asbestos and the Asbestos Diseases. Cardiff, April 6-9, 1970. Presented: Identification of Minerals in Tissue with the Electron Probe.

British Occupational Hygiene Society: Inhaled Particles and Vapors. Imperial College, London, September, 1970. Presented: Inorganic Fibers, Including Chrysotile, in Lungs at Autopsy (with Baden V, Selikoff IJ, and Hammond EC).

Fifth National Conference on Electron Microprobe Analysis. New York, July 21-24, 1970. Invited paper: Electron Microprobe Analysis of Particles in Tissues (with Berkley C and Rubin IB).

Second International Clean Air Congress. Washington, DC, May 1971. Paper with Selikoff IJ.

International Agency for Research on Cancer. WHO, Lyon, September, 1972. Presented: Identification of Single Asbestos Fibers in Human Tissues with Pooley FD.

6th International Meeting of Forensic Sciences. Edinburgh, September, 1972. Presented paper Asbestos Fiber in Human Lungs: Forensic Significance in Environmental Disease (with Ehrenreich T).

Dialogues in Microscopy, 1973, NY. Microscopical Society. Chaired Session: Identification of Asbestos, May, 1973.

International Conference on the Biological Effects of Ingested Asbestos. NIEHS sponsored. Pinehurst, NC, November, 1973.

American Industrial Hygiene Conference. Miami. Presented paper Asbestos exposure During Brake Maintenance and Repair. With Rohl AN, Anderson H, Nicholson WJ. May, 1974

International Conference on Environmental Sensing and Assessment. Las Vegas, NV, September, 1975 (paper with Rubin IB and Wolff M). Chaired 2 Sessions: Fine Particles-7; Fine Particles-2. United States Environmental Protection Agency.

Conference on Health Problems of Energy Technologies. Held by NIEHS, January, 1976. Pinehurst, North Carolina.

Mineralogical Society of London. Centenary of the Mineralogical Society. London, April, 1976. Presented paper Nature and Range of Mineral Dust in the Environment. London.

International Agency for Research on Cancer. The World Health Organization, Lyon. Invited member of working group, December, 1976. Chemical Carcinogenesis Monograph Series. Evaluation of Carcinogenic Risk of Asbestos, Vol. 14.

Third International Symposium on Detection and Prevention of Cancer. International Agency for Research on Cancer. April 26-May 1, 1976. New York. Identification of Microparticles. Workshop Chairman.

Royal Society of Medicine. Meeting Focusing on Occupational Diseases. London, April, 1977. Presented paper Asbestos Content of Dust Encountered During Brake Maintenance and Repair (with Rohl AN, Weisman I, and Klimentidis R).

Invited Consultant to Institute of Public Health. Oslo, Norway, May, 1977. Presented three papers: Contamination of the Biosphere by Submicroscopic Particles; Asbestos Minerals and Their Carcinogenic Effects; Are There Safe Substitutes for Asbestos Materials?

Invited Consultant to the Ministry of Mines. Biological Effects of Asbestos. Johannesburg, October, 1977. Monograph, Ministry of Mines.

Health Hazards of Asbestos Exposure. New York, June 24-27, 1978. Presented paper on Contamination of Lake Superior with Amphibole Gangue Minerals and organized and chaired workshop on Significance of Aspect Ratio in Regulation of Asbestos Fiber Exposure. Co-authored with A Rohl Asbestos Content of Host Rock in Non-Asbestos Mining and Quarrying.

International Conference on Critical Current Issues in Environmental Health Hazards. Tel Aviv, March 4-7, 1979. Presented paper Defining New Asbestos High Risk Groups. Chairman Workshop: Laboratory Approaches in Environmental Medicine.

International Agency for Research on Cancer. WHO, Lyon, October, 1979. International Conference on the Biological Effects of Inorganic Fibers. Invited Rappateur. Physics and Chemistry of Asbestos Minerals.

Expert Consultant, International Metal Workers Federation. Geneva, September, 1980. Focus on problem: Asbestos Fiber Contamination of Metal Ore Deposits.

International Conference on Crystalline Deposits in Human Tissues. August 13-14, 1981. Mount Sinai Hospital, Toronto. Presented paper The Role of Analytical Electron Microscopy in Diagnosis of the Pneumoconiosis (with Weisman I, Adams A, and Pooley F).

Annual Symposium Scanning Electron Microscopy. Anaheim, CA, April, 1982. Presented paper Identification Characterization and Quantitation of Asbestos Fibers in Human Lung Tissue by Analytical Electron Microscopy.

Fourth Annual Rocky Mountain Center for Occupational and Environmental Health Center. Salt Lake City, April, 1982. Invited to present two papers: Fibrous Minerals: Properties and Biological Activity; Relationship Between Surface Properties and Biological Activity in Health Issues Related to Metal and Non-Metallic Mining.

World Symposium on Asbestos. Montreal, May 24-27, 1982. Presented paper Mineral Fibers: Properties Imparting Biological Activity.

6th International Pneumoconiosis Conference. Bochum, Federal Republic of Germany,

September, 1983. Presented two papers: Recognition of Quartz by the Erythrocyte Membrane; Physicochemical Factors Effecting Membrolytic Properties of Quartz (with Nolan RP, Foster KW, and Simenski R). Chaired Session: Etiopathogenesis of Silicosis.

Program of Research on Asbestos. University of Sherbrooke, December 5-6, 1983. Presented lecture Physicochemical Properties of Minerals as Related to Biological Activities.

Workshop on the Pathobiology of Mesothelioma. Etiology and Pathogenesis. Presented Properties of Mineral Fibers and Disease. National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina. June 11, 1983.

2nd International Congress on Applied Mineralogy. February 22-25, 1984. Presented two papers: Asbestos, Fibrous Minerals, Acicular Cleavage Fragments and the Minerals Industries; Minerals and Disease:. The Association Between Exposure to Minerals and Rock Dusts and the Occurrence of Human Disease.

Workshop on the Assessment of Mineral Fibers from Human Lungs. Oxford, England, September 17-19, 1984. Keynote speaker Problems of Tissue Analysis with the Analytical Electron Microscope.

3rd International Workshop on the In Vitro Effects of Mineral Dusts. Schluchsee, Hochschwarzwald, FRG, October, 1984. Physicochemical Properties of Minerals Relevant to Biological Activities. State of the Art, (paper with Nolan RP); Alteration of surface of quartz and altered biological activity. Member International Organizing Committee.

Workshop on Biological Effects of Chrysotile. General Motors Cancer Research Foundation. Cardiff, Wales, May 7-9, 1986. The Mineralogy of Chrysotile Asbestos: Physicochemical Properties as Determinants of Disease Potential (with Nolan RP).

International Programme on Chemical Safety (IPCS). WHO Environmental Health Criteria 53. Asbestos and other Natural Mineral Fibres. Hannover, Germany. Session chairman, co-author. 194 p., 1986.

International Agency for Research on Cancer. The World Health Organization. Invited member of working group. Lyon, France, July, 1986. Chemical Carcinogenesis Monograph Series. Evaluation of Carcinogenic Risk of Fibrous Talc, Wollastonite, Palygorskite, Sepiolite, Erionite and Crystalline Silica. Vols. 34-42.

Symposium: Mineral Fibers in the Non-Occupational Environment. IARC-WHO. Lyon, France, September 8-10, 1987. Summary of five presented papers; Round-table discussant on Environmental Risk. Presented paper: Fiber Type and Parenchymal Burden Found in Workers Occupationally Exposed to Asbestos Fiber in the United States (with Nolan RP).

Symposium: Fibers in Friction Materials. Society Automotive Engineers and the Asbestos Institute. Symposium organizer and Co-Chairman. Fibers and Health Issues. Atlantic City, NJ, October, 1987. Presented summary paper; co-authored Physicochemical Properties of Fibers and Biological Potential (with Nolan RP).

Symposium. 117th Annual Meeting of the Society of Mining Engineers. Tuscon, Arizona. Mineralogical and Biological Comparison of Asbestiform and Non-asbestiform Tremolite (with RP Nolan). January 25-28, 1988.

Symposium: Safe Use of Asbestos Cement. Peruvian Ministry of Health and the Environment. The Asbestos Information Association of South America. Lima, Peru, March, 1988.

International Symposium/Workshop: Biological Interaction of Inhaled Mineral Fibers and Cigarette Smoke. Session Chairman. Presented paper: Inorganic Particles Found in Cigarette Tobacco, Cigarette Ash, and Cigarette Smoke; co-authored Physico-Chemical Properties and Membranolytic Activities of the Titanium Dioxide Polymorphs Compared to Quartz. April 10-14, 1988.

Electron Microscopy Society of America; the Microscopical Society of Canada: Presented paper: Electron Diffraction of Mineral Fibers Vs. Acicular Cleavage Fragments. Milwaukee, WI, August 7-12, 1988.

VIIth International Pneumoconiosis Conference. National Organizing Committee, Workshop Chairman, Scientific Organization Committee. Chair: Hazard Recognition of Mineral Dusts. Presented: Mineral Fibers in the Lung Tissues of Persons Exposed to Asbestos in the United States; Distinguishing Between Tremolite Asbestos and Tremolite Cleavage Fragments; Hazard Recognition of the Silica Polymorphs.
Pathology Discussion Group. Pittsburgh, PA, August, 1988.

Session Chairman: Asbestos, Pleural Pathology, and Lung Fiber Burden. VIIth International Pneumoconiosis Conference, Pittsburgh, PA, August, 1988.

Workshop on Asbestos Research. Health Effects Institute, American Academy of Arts and Sciences, Cambridge, MA, October 31-November 1, 1988.

Symposium on the Health Aspects of Exposures to Asbestos in Public Buildings. Presented: Fiber Type and Risk of Mesothelioma to Building Occupants. John F. Kennedy School of Government, Harvard University, Cambridge, MA, December 14- 16, 1988.

First International Conference on Health Related Effects of Phyllosilicates. International Scientific Organizing Committee. Member International Scientific Committee. Session Chairman: Health Related Effects After Non-Occupational Exposure. Presented: Fibrous Minerals as Natural Contaminants of Phyllosilicates (with Pooley F). Paris, France, March 16-18, 1989.

International Federation of Building and Woodworkers. Health Hazards in Painting and Allied Trades. Presented: Hazards in the Painting Trade. Panelist. Geneva, Switzerland, May 8-12, 1989.

NATO Advanced Research Workshop on Mechanisms in Fibre Carcinogenesis. Presented: The Importance of Mineral Subpopulations in Biological Assays. Alburquerque, NM, October 22-25, 1990. Co-authored paper: Association of tremolite habit with biological potential, co-authored with Nolan RP, Oechsle G, Addison J, Colflesh D.

American Lung Association - American Thoracic Society. 1991 International Conference. Presented: Mechanisms of Asbestos-Induced Pulmonary Disease: Importance of Surface Properties of Asbestos and Non-asbestos minerals in cell interaction. Anaheim, California, May 12-15, 1991.

American College of Chest Physicians. Fourth International Conference - Environmental Lung Disease. Presented: Diagnostic Methods in Occupational Lung Disease: Mineralogic Techniques. Co-Chair session: Diagnostic Methods in Occupational Lung Disease. Montreal, Canada. September 25-28, 1991.

Chemical Industry Institute of Toxicology. Workshop on Approaches to Evaluating the Toxicity and Carcinogenicity of Man-made Fibers. "Characterization of Man-made Vitreous Fibers for Experimentation Purposes." Durham, North Carolina. November 11-13, 1991.

Institute for Glass Science and Engineering. Workshop on Chemical and Biological Interactions of Glass. Discussant on Biologically Important Properties. Bethesda, Maryland. March 5-6, 1992.

International Geological Congress. Environmental Mineralogy in relation to human health and activities. Session I-3-49. Co-chair with N. Kohyama, J. Addison, Kyoto, Japan. 24 August-3 September, 1992. Introduction. Importance of environmental mineralogy. Summing up. 1 September, 1992.

Biopersistence of Respirable Synthetic Fibres and Minerals. Presented: Comparison of lung tissue mineral fibre retention of exposed workers and the general population. With RP Nolan, co-authored: Health hazard evaluation of the lung tremolite fibre content among Canadian chrysotile workers. With RP Nolan and J Addison. Round-Table discussant on: Role of Biopersistence in Pathogenicity. IARC, Lyon. September 7-9, 1992.

Symposium on Chemicals and the Environment. Chemical Specialities, '92. Invited presentation: Zeolite catalysts. Is there a health risk? Philadelphia, 3-4 November, 1992.

International Life Sciences Institute. 4th International Inhalation Symposium, Hannover. Invited Faculty Presentation: Factors Controlling the Biological Potential 6 Inorganic Dusts. Surface Chemistry and Character. With R.P. Nolan.

1-5 March, 1993.

Hoffmann-LaRoche Pharmaceuticals. Analysis of Medicines for Asbestos. Microscopy Protocol. Edinburgh, U.K. Institute of Occupational Medicine. April 25-26, 1993.

Cellular and Molecular Effects of Mineral and Synthetic Dusts and Fibres. NATO Advanced Workshop. Invited paper: Phosphorylated Canadian chrysotile. With RP Nolan and G Herson. Chaired session: Physico-chemical properties of minerals in relation to their biologic effects. Paris, 11-13 October, 1993.

Workshop on the Health Risks Associated with Chrysotile Asbestos. Intl. Commission on Occupational Health - Intl. Program on Chemical Safety (WHO) Presented paper: Chrysotile: The Mineral and Its Properties. Jersey, Channel Islands. 14-17, November, 1993.

Hoffmann-LaRoche Pharmaceuticals. Protocol for Analysis of Injectable Medicines for Asbestos. Workshop. Basel, Switzerland. March 2-4, 1994.

Verband der Chemischen Industries. Protocol for the Analysis of Chemicals for Asbestos. Workshop. Frankfurt, Germany. May 30-31, 1994.

Eurometaux (European Association of Metals) and the European Commission, Directorate General V. Scientific Organizing Committee InH. Seminar on Assessment of Respiratory Carcinogenic Risk from Occupational Exposure to Inorganic Substances. Meeting of the Scientific Committee. Brussels, 17-18 October, 1994.

V.M. Goldschmidt Conference. An Intl. Conference for the Advancement of Geochemistry. The Geochemical Society. Invited presentation: Chemically Modified Chrysotile Asbestos: Physical-chemical Characterization and Membranolytic Activity of Phosphorylated Canadian Chrysotile. With RP Nolan and G Herson. Pennsylvania State University, May 24-26, 1995.

Eurometaux (European Association of Metals) and the European Commission, Directorate General V. Scientific Organizing Committee. In: Seminar on Assessment of Respiratory Carcinogenic Risk from Occupational Exposure to Inorganic Substances. Meeting of the Scientific Committee. Brussels, 5-8 September, 1995.

International Seminar on Assessment of Carcinogenic Risk from Occupational Exposure to Inorganic Substances. Luxembourg, October 17-20, 1995. Scientific Committee; Editorial and Scientific Board; Chairman of Session III: Physico-chemical characterization of exposures; Co-author of presentation; Working Party Chairman.

Symposium on the Health Effects of Fibrous Materials Used in Industry Excluding Asbestos. Sydney, 30-31 October, 1995. Invited Presentation: Physical-Chemical Properties of Fibers Other Than Asbestos Used in Global Commerce. AM Langer, RP Nolan.

International Programme on Chemical Safety (IPCS), WHO. Environmental Health Criteria Document For Chrysotile. Geneva, Switzerland. Rappateur. July 1-6, 1996. Senior author chapters 2, 3, 4, 5 and 6.

International Workshop The Health Effects of Chrysotile Asbestos: The Contribution Science to Risk Management Decisions. Member of Scientific Committee; International Organizing Committee. Co-chairman Session: Mineralogy, Modern Products and Exposures. Presented Health Experience of some U.S. and Canadian workers exposed to amphibole asbestos and mixed fiber types. Co-author paper Asbestos fiber types and their concentrations in the inside air of buildings. Summary of the symposium. Montréal, 14-16 September, 1997

International Workshop on Simian Virus 40 (SV-40), Asbestos and Mesothelioma. Member of the Scientific Organizing Committee. Presented Historical Development of the Association Between Mineral Fiber Exposure and Human Mesothelioma. AM Langer; The Role of Lung Content Analysis in Mesothelioma Etiology. (with RP Nolan). New York 8-9 November, 1997.

International Conference on Urban Terrorism. Homeland Security After 9/11. Terrorism in the 21rst Century. Presented Paper: Future of Urban Architecture-Skyscrapers, Will They Be Continued To Be Built? AM Langer. John Jay College of Criminal Justice, New York. January 23-24, 2003.

International Symposium on the Health Hazard Evaluation of Fibrous Particles Associated with Taconite and the Adjacent Duluth Complex. Chaired Sessions I, VI; Rapporteur Session II. Presented paper: Control and Monitoring of Asbestos in the United States. Regulatory Overview of Asbestos and Other Fibrous Minerals. St. Paul, March 30- April 1, 2003.

Funded Research

Asbestos Disease in New York City Workers. Co-Principal Investigator. Health Research Council, New York City. July 1, 1966 – June 30, 1967. $ 39,703.

Silica and Silicosis in New York City Foundation Workers and Tunnel Drivers. Principal Investigator. Polacheck Foundation. September 1, 1966 – August 31, 1968. $ 10,000.

Microprobe Analysis of Asbestos Bodies and Pseudo-Asbestos Bodies. Co-Principal Investigator. USPHS-National Institutes of Health. June 1, 1967 – May 31, 1969. $ 133,245.

Asbestos Exposure Risk in Defined Human Population. Co-Principal Investigator. National Institutes of Health. May 1, 1968 – April 30, 1973. $ 1,306,281.

Biological Effects of Modified Inorganic Fibrous Particles. Co-Principal Investigator. Johns Manville Corporation. $ 61,930.

Insulation Industry Hygiene Reports. Co-Principal Investigator. Johns Manville Corporation, International Association of Heat and Frost Insulators and Asbestos Workers, National Institutes of Health. September 15, 1968 – September 14, 1973. $ 2,500,000.

Asbestos Air Pollution in New York City. Co-Principal Investigator. Health Research Council and the Department of Air Resources, New York City. December 1, 1968 – November 30, 1970. $ 110,455.

Inorganic Particles – Biological Systems Interactions. Principal Investigator. **Career Development Award**, National Institute of Environmental Health Sciences. June 1. 1969 – May 31, 1974. $ 125,000.

Asbestos Exposure and Cancer in the General Population. Co-Principal Investigator. National Institutes of Health, American Cancer Society. December 1, 1969– November 30, 1971. $ 105,539.

Inorganic Particles in Cigarettes and Cigarette Smoke. Principal Investigator. National Institutes of Health. July 1, 1971 – June 30, 1973. $ 65,730.

Environmental Cancer in US Industries. Co-Principal Investigator. American Cancer Society. September 1, 1971 – August 31, 1974. $ 289,321.

Asbestos Exposure and Cancer in the General Population. Co-Principal Investigator. National Institutes of Health, American Cancer Society. December 1, 1971 – November 30, 1973. $ 254,569.

Asbestos Air Pollution. Extent, Persistence, Effects. Co-Principal Investigator. National Institutes of Health. January 1, 1972 – December 31, 1974. $348,858.

Asbestos in Therapeutic Drugs for Human Use. Co-Principal Investigator. National Institutes of Health. April 1, 1972 – March 31, 1973. $ 49,230.

Effects of Asbestos in Water (Lake Superior). Co-Principal Investigator. US Environmental Protection Agency. June 1, 1973 – October 30, 1973. $ 47,970.

Environmental Agents. Relation to Human Health Effects. Co-Principal Investigator. National Institutes of Health (NIEHS). June 1, 1973 – August 31, 1978. $ 2,823,220.

Asbestos in Brake Drum Dusts. Principal Investigator. Ford Motor Company. July 1, 1973 – June 30, 1975. $ 10,000.

Populations at High Risk for Lung Cancer. Establishment of Cohorts and Development of Serial Surveillance. Co-Principal Investigator. National Institutes of Health, American Cancer Society. August 1, 1973 – July 31, 1975. $ 78,572.

Asbestos Exposure of Workers and Public From Brake Linings. Co-Principal Investigator. Health Research Council, New York City. July 1, 1975 – June 30, 1976. $ 12,980.

Asbestos Exposure with Brake Lining Service and Repair. Co-Principal Investigator. Health Research Council, New York City. July 1, 1976 – June 30, 1977. $ 51,142.

Residency Training in Occupational Medicine. Co-Principal Investigator. National Institute for Occupational safety and Health. July 1, 1976 – June 30, 1978. $ 105,491.

Asbestos in Brake Workers Lungs. Principal Investigator. National Institute for Occupational Safety and Health. September 1, 1977 – August 31, 1979. $ 95,223.

Environmental Health Sciences Center Grant. Co-Principal Investigator. National Institutes of Health (NIEHS). September 1, 1978 – August 31, 1983. $ 4,841,529.

Environmental Dusts and Macrophage Activation. Co-Principal Investigator. National Cancer Institute. March 1, 1979 – February 28, 1982. $ 148,461.

Surface Properties of Chrysotile Asbestos and Its Role in Biological Potential. Principal Investigator. July 1, 1979 – June 30, 1981. $ 20,000.

Environmental Cancer Research Project. Co-Principal Investigator. American Cancer Society. September 1, 1980 – August 31, 1981. $ 284,540.

Saint Regis Health Study. Co-Principal Investigator. Canadian Ministry of Health. July 1, 1981 – June 30, 1982. $ 1,016,581.

Activity of Amosite Asbestos and Grunerite Fragments. Co-Principal Investigator. National Institute of Occupational Safety and Health. September 1, 1982 – August 31, 1984. $ 143,396.

Environmental Health Sciences Center Grant – Supplement. Co-Principal Investigator. Group Leader. National Institutes of Health (NIEHS). July 1, 1984 – June 30, 1986. $ 300,000.

Biological Activity of Modified Chrysotile Asbestos Fiber. Principal Investigator. Societe Nationale de L'Amiante, Canada. September 1, 1984 – December 31, 1986. $ 300,000.

Biological Activity of Asbestos Substitutes. Principal Investigator. Societe Nationale de L'Amiante, Canada. January 1, 1987 – March 31,1987. $ 15,000.

Analysis of Play Sand for Its Asbestos Content. Principal Investigator. US Consumer

Products Safety Commission. November 1, 1987 – February 1, 1988. $ 4,000.

Analysis of Talcs for Its Mineral Character. Principal Investigator. Vanderbilt Minerals Company. April 1, 1988 – December 1, 1988. $ 32,000.

Surface Properties of Asbestos and Substitutes. Principal Investigator. Asbestos Institute of Canada. April 1, 1987 – March 31, 1988. $ 10,000.

Evaluation of Microscopy Laboratories Engaged in Particle Analysis. Co-Principal Investigator. Battelle Laboratories. March 1, 1988 – June 30, 1990. $ 130,500.

Wollastonite in Animal Lungs. Principal Investigator. National Toxicology Laboratory, NIEHS. August 1, 1988 – June 1, 1989. $ 38,000.

Modified Asbestos, Chrysophosphate, Surface Properties. Principal Investigator. Societe Nationale de L'Amiante, Canada. April 1, 1988 – September 1, 1990. $ 50,000.

Fibers in Vermiculite. Principal Investigator. State of Montana. $ 900.

Characterization of Asbestiform and Non-Asbestiform Amphiboles. Principal Investigator. Vanderbilt Minerals Company. June 1, 1989 – May 31, 1994. $ 162,000.

Studies of Asbestos Contamination of Building Air. Principal Investigator. Continental Insurance Company. June 30, 1988 – June 30, 1994. $ 50,000.

Asbestos-Emissions During Gasket Replacement. Co-Principal Investigator. Durabla Company. January 30, 1991 – December 31, 1992. $ 40,000.

Release of Rockwool Fibers From Gaskets. Principal Investigator. Lapinus Fibers, B.V. January 1, 1993 – June 30, 1994. $ 7,000.

Asbestos Release From Plumbing Packing. Co-Principal Investigator. Sexauer Plumbing Supplies. October 1, 1992 – June 30, 1993. $ 10,000.

Asbestos in the Lungs of Exposed Workers. Principal Investigator. W.R.Grace Company. March 1, 1993 – February 28, 1995. $ 90,000.

Applied Technology Equipment Assistance Grant. Co-Principal Investigator. New York State. September 30, 1993 – August 31, 1995. $ 710,000.

Mineral Analysis of Quarry Samples. Co-Principal Investigator. Ready Group of Australia. December 10, 1993 – June 1, 1994. $ 10,000.

Ecaterinburg Working Project. Chrysotile in Russia. Co-Principal Investigator. The

Asbestos Institute, Canada, W.R.Grace Company. June 1, 1994 – May 31, 1995. $ 30,000.

Risk Identification and Management of Fibers in Iron Ore. Co-Principal Investigator. Cleveland Cliffs, Incorporated. April 1, 1994 – September 1, 1996. $ 40,000.

Health Risks to Workers Applying Blown Glass Insulation. Co-Principal Investigator. Glass Insulation association. July 1, 1995 – September 1, 1996. $30,000.

Health Risks to Workers Applying Blown Cellulose Insulation. Co-Principal Investigator. January 15, 1997 – September 1, 1997. $ 10,000.

Workshop on Biological Risks Associated With Chrysotile Asbestos Exposure. Co-Organizer. Health Quebec. June 30, 1997 – December 31, 1997. $ 100,000.

International Workshop on SV-40 Virus and Asbestos. Co-Organizer. Coalition to Support Mesothelioma Research. August 1, 1997 – December 1, 1997. $ 25,000.

Mechanisms and Treatment Strategies of Lung Injuries Following Exposure To Fine Particles of Desert Sand. Co-Principal Investigator. US Army Medical Research Acquisition Activity. In Review. $ 887,316.

DATE: 2006

VITA

H. CATHERINE W. SKINNER

Born: Brooklyn, N.Y., January 25, 1931.
Nationality: U.S. Citizen
Personal: Married 1954, to Brian J. Skinner, three children.
Education: B.A. Mt. Holyoke College, 1952.
M.A. Radcliffe, 1954.
Ph.D. University of Adelaide, South Australia, 1959.

Present Positions:

Lecturer, Dept. of Orthopaedics and Rehabilitation, Yale Medical School and Research Affiliate, and Lecturer, Dept. of Geology and Geophysics, Yale University.

Previous Positions:

Mineralogist, Section of Molecular Structure, Lab. of Molecular Biology, National Inst. of Arthritis and Metabiolic Diseases, N.I.H., 1961-1965.

Research Mineralogist, Section of Crystal Chemistry, Lab. of Histology and Pathology, National Inst. of Dental Research, N.I.H., 1965-1966.

Research Associate, Dept. of Surgery, Yale University, 1967-1972.

Senior Research Assoc., Dept. of Surgery, Yale University, 1972-1975.

Associate Professor of Biochemistry in Surgery, Yale University, 1978-1984.

Lecturer Department of Geology and Geophysics, Yale University, 1967-1969, 1977-1978, 1984-1985, and 1988-1989, 1992- present.

Research Affiliate, Department of Geology and Geophysics, Yale University, 1984-present, and Department of Orthopaedics and Rehabilitation 1984-present.

Lecturer, Department of Biology, Yale University, 1977-1983.

Lecturer, Department of Orthopaedic Surgery and Rehabilitation, Yale University, 1972- present.

Master, Jonathan Edwards College, Yale University, 1977-1983.

President, Connecticut Academy of Arts and Sciences, 1985-1994.

Chairman, Publications Committee, Connecticut Academy of Arts and Sciences 1994-2001.

Visiting Positions:

Alexander Agassiz Visiting Lecturer, Department of Biology, Harvard University, 1976-1977.

Visiting Professor, Section of Ecology and Systematics, Department of Biology, Cornell University, 1980,1981,1983.

Distinguished Visiting Professor, Department of Geology, University of Adelaide, South Australia, 1991.

Allan Cox Visiting Professor, Department of Geology and Environmental Sciences, Stanford University, Stanford, CA. WINTER TERM 2003

Honors and Awards:

President, Connecticut Academy of Arts and Sciences, 1985-1994.
Public Service Award, Mineralogical Society of America, 1991.
Fellow, Geological Society of America, 1989.
Fellow, American Association for the Advancement of Science, 1993.

Professional Recognition:

Mineralogical Society of America: Councilor 1979-1981; Finance Committee 1981-1986, Chairman 1984-1986; Development Committee, 1986-1990, Chairman 1986-1988; Fellowship Committee, 1977; Nominating Committee 1973; Public Service Award Committee 1995-1998.
American Mineralogist, Associate Editor 1977-1983.
American Geological Institute: Publications Committee 1993-1996, 2000-2005
Geological Society of America: Fellow 1989; Committee on Committees, 1991
Geological Society of America Foundation, Trustee 1998-PRESENT National Academy of Sciences/National Research Council, Co-Chairman of Panel of the Geochemistry of Fibrous Materials Related to Health Risks, Subcommittee on Geochemical Environment in Relation to Health and Disease, 1979-1982.
National Institutes of Dental Research, N.I.H. Committee on Research Institutes and Special Programs. 1971-1975. Chairman of Site Visit Team, 1974-1975.
National Academy of Sciences/National Research Council. Chairman, Committee on Research Priorities in Earth Sciences and Public Health 2004- present

Keynote/invited speaker (Selected RECENT LISTINGS)

International Symposium "Setting an Agenda for Health In the Environment, International Council on Scientific Unions- Queens University, Ottawa, Ontario, Canada, held in Jamaica, 11/11-11/14/99

International Symposium "Health and the Geologic Environment" International Union of Geologic Sciences-Geological Survey of Sweden, held in Uppsala, Sweden. 9/1-9/7/2000 (also organizer with others).

European Mineralogical Union Short Course "Environmental Mineralogy" Budapest, 5/15-5/21/00

Workshop on Porphyry Copper Deposits, Mining and Life Cycles US Geological Survey-University of Tucson, held in Tucson, AZ. 5/15-5/17/2001

Pardee Symposium, Medical Geology, Geological Society of America Annual Meetings, 11/05/2001 (organizer with others)

International Symposium " Medical Geology, Nutrition and Cancer" Organized by Ministry of Health of Turkey and Ankara University, Turkey. Held in Ankara, 3/30-4/5, 2002.

Workshop on "Geology and Human Health" NAGT and NSF held at Chico Hot Springs, Montana May 12-15th, 2004

Hallimond Lecturer Mineralogical Society, Great Britain at Annual Meeting of the Society January 5-8th, 2005 Environmental Geology, Geochemistry and Human Health, Bath England.

Geology and Human Health, a Symposium to honor Charles Singler, Youngstown State University, Youngstown, Ohio. May 7-8, 2005.

19th General meeting of the International Mineralogical Association, Kobe, Japan, July 23-28, 2006

VISITING PROFESSORSHIPS: Harvard University, 1976; Cornell University 1981, 1983; Adelaide University, Australia 1990, University of Wyoming, 1995, Stanford University 2003

Yale Committees(Selected):

Yale University Medical School: Funds and Fellowships, International Health, Research Ranks, etc. 1973-1977.

Yale - New Haven Hospital: Dental Advisory Committee, 1973-1980.

Yale University, Committees: Women's Education (Chairman, 1981), Henry Fellowship, Security (Chairman, 1980-1982).

Yale University Press: Publications Committee, 1974-1976.

Yale -New Haven Teachers Institute: Faculty Advisor Committee, 1983-1999.

Professional Societies:

American Association for the Advancement of Science (Fellow, 1993).

American Geologic Institute (Publications Committee)

Connecticut Academy of Arts and Sciences (President 1985-1994, Publications 1994-2001)

Geological Society of America (Fellow, 1989)

Geological Society of America Foundation (1998- present)

Mineralogical Society of America (Fellow 1978, Councilor 1979-1981)

Public Service(Selected):

Connecticut Fund for the Environment, 1983- Education Committee, 1985-1989. Scientific Advisory Council, 1989-1997.

Greater New Haven Chamber of Commerce Board, 1979-1985 Program Chairman: Women, Business and the Future, 1981-1983

Investor's Strategy Institute, New Haven, Founder and President, 1983-1985

Miss Porter's School, Farmington, Ct., Trustee 1985-1990; Search Committee for Head 1991-1992. Education Committee, 1986-1988; Salaries and Benefits, 1988-1990.

YWCA, New Haven 1982-1985 (Treasurer, 1983-1984).

Geological Society of America Foundation, Trustee (1998- present) 68

PUBLICATIONS, 4 BOOKS

PUBLICATIONS

1. Alderman, A.R. and Skinner H.C.W. (1957) Dolomite in the South East of South Australia. Am. J. Sci. 255:561-566.
2. Skinner, H.C.W. (1963) Precipitation of Calcian Dolomites and Magnesian Calcites in the South East of South Australia. Am. J. Sci. 261:449-472.
3. Skinner, H.C.W., Skinner, B.J. and Rubin, M. (1963) Age and Accumulation Rate of recent Dolomite-Bearing Carbonate Sediments in South Australia. Science 139:335-336.
4. Sigler, P.B. and Skinner, H.C.W. (1963) The Reaction of Di-isopropyl fluorophosphate with Crystals of Γ-Chymotrypsin. Biochem. Biophys. Res. Com. 13:226-229.

5. Sigler, P.B., Skinner, H.C.W. Coulter, C., Kallos, J., Braxton, H. and Davis, D.R. (1964) An Isomorphous Heavy Atom Replacement at the Active Site of Γ-Chymotripsin. Proc. Nat. Acad. Sci. 51:1145-1151.
6. Muzii, E.O. and Skinner, H.C.W. (1966) Calcite Deposition During Shell Repair by the Aragonitic Gastropod Murex Fulvescens. Science 151:201-203.
7. Skinner, H.C.W. and Burnham, C.W. (1968) Hydroxyapatite, Annual Report of the Director, Geophysical Laboratory, Carnegie Inst. Washington, D.C. p. 293-294.
8. Clayton, R.N., Skinner H.C.W., Berner, R.A. and Rubin, M. (1968) Isotopic Compositions of South Australian Carbonates. Geochem. et Cosmochem. Acta 32:983-988.
9. Cowgill, U.M., Hutchinson, G.E. and Skinner, H.C.W. (1968) The Elementary Composition of Latimeria Chalumnae Smith. Proc. Nat. Acad. Sci. 60:456-463.
10. Pak, C.Y.C., and Skinner, H.C.W. (1968) Ionic Interaction with Bone Mineral. IV Varying Affinity of Synthetic Calcium Phosphates for Calcium. Biochem. Biophys. Acta 165:274-269.
11. Skinner, H.C.W. (1968) X-ray Diffraction Analysis Techniques to Monitor Composition Fluctuations within the Mineral Group Apatite. Applied Spect. 22:412-414.
12. Skinner, H.C.W. (1970) Transition of Calcium Orthophosphate to Pyrophosphate at Elevated Pressures. Mat. Res. Bull. 5:437-448.
13. Skinner, H.C.W., Kempner, E. and Pak, C.Y.C. (1972) Preparation of the Mineral Phase of Bone using Ethylene Diamine Extraction. Calcified Tiss. Res. 10:257-268.
14. Jokl, P. and Skinner H.C.W. (1973) Fluoride Induced Changes in Ashed Mineral of Growing Sheep. J. Bone and Jt. Surg. 55A:761-770.
15. Skinner, H.C.W. (1973) Studies in the Basic Mineralizing System CaO-P_2O_5-H_2O Calc. Tiss. Res. 14:3-14.
16. Skinner, H.C.W. (1973) Phase Relations in the CaO-P_2O_5-H_2O System from 300-600°C at 2 kb H_2O Pressure. Amer. J. Sci. 273:545-560.
17. Bullock, J., Albert, J. and Skinner, H.C.W. (1974) Calcium Oxalate Retinopathy Associated with Generalized Oxalosis: X-ray Diffraction and EM studies on Crystal Deposits. Invest. Opthamol. 13:256-265.
18. Skinner, H.C.W., Kittleberger, S. and Beebe, R.A. (1975) Thermal Instability in Synthetic Hydroxyapatites. J. Phys. Chem. 79:2017-2019.
19. Skinner, H.C.W. and Pak, C.Y.C. (1975) Ionic Interactions with Bone Mineral VI Crystal Chemical Changes in Synthetic Calcium Phosphates. Bioinorg. Chem 4:323-335.
20. Skinner, H.C.W. and Nalbandian, J. (1975) Tetracycline and Mineralized Tissues: Review and Perspectives. Yale J. Biol. and Med. 48:377-397.
21. Skinner, H.C.W., Osbaldistan, G. and Wilner, A. (1977) Monohydrocalcite in a Guinea Pig Bladder Stone: A Novel Occurrence. Am. Min. 62:273-277.
22. Skinner, H.C.W., Bartz, Z. Ladenbauer-Belis, I., Pooley, A. and Albright, J.A. (1978) Scanning Electron Microscopy of Osteogenisis Imperfecta and Normal Deciduous Enamel J. Dental Res. 57:418-419.
23. Skinner, H.C.W. (1979) Bone: Cellular and Molecular Components. in The Scientific Basis of Orthopaedics. R. Brand and J.A. Albright (Eds.) Appleton-Century-Crofts, New York. p.105-134.
24. Albright, J.A. and Skinner, H.C.W. (1979) Bone Remodeling Dynamics. in The Scientific Basis of Orthopaedics. R. Brand and J.A. Albright (Eds.) Appleton-Century-Crofts, New

York. p.185-229.

25. Meinke, D., Skinner, H.C.W., and Thomson, K.S. (1979) X-ray Diffraction of the Calcified Tissues in Polypterus. Calc. Tiss. Int. 28:27-42.
26. Skinner, H.C.W., Hunt, H.T. and Griswold, J. (1980) Automatic Scanning of Multiple Sample Guinier X-Ray Powder Diffraction Films. J. of Physics E: Scientific Instruments 13:74-79.
27. Skinner, B.J. and Skinner, H.C.W. (1982) Is There a Limit on the Number of Minerals? Min. Rec. Sept-Oct:335-337.
28. Skinner, H.C.W. (1982) Teeth. McGraw Hill Yearbook of Science and Technology p.453-456.
29 Skinner, H.C.W. (1985) Mineralogical Analysis of Fragments (Appendix to The Sargonic Victory Stele from Teloh by B.R. Foster) in IRAQ XLVII:15-30.
30. Ballarino, J., Howland, H.C., Skinner, H.C.W., Brothers, E.B. and W. Bassett (1985) Studies of Otoconia in the Developing Chick by Polarized Light Microscopy. Am. J. Anat. 174:131-144.
31. Skinner, H.C.W. (1987) Bone: Mineral and Mineralization. in The Scientific Basis of Orthopaedics. J.A. Albright and R. Brand (Eds.) Second Edition. Appleton and Lange, Norwalk, CT. p.198-212.
32. Albright, J.A. and Skinner, H.C.W. (1987) Bone: Structural Organization and Remodeling Dynamics. in The Scientific Basis of Orthopaedics. J.A. Albright and R. Brand (Eds.) Second Edition. Appleton and Lange, Norwalk, Ct.p.165-198.
33. Skinner, H.C.W., Malcolm Ross and Clifford Frondel (1988) Asbestos and Other Fibrous Materials: Mineralogy, Crystal Chemistry and Health Effects. Oxford University Press., New York. 204 pp.
34. Skinner, H.C.W. (1989) Low Temperature Carbonate Phosphate Materials or the Carbonate-Apatite Problem. A Review. in Crick, R. (Ed) Origin, Evolution and Modern Aspects of Biomineralization in Plants and Animals. Proceedings of the 5th International Symposium on Biomineralization. Arlington, Texas, May 1986. Plenum Press. New York.p.251-264.
35. Skinner, H.C.W. and Malcolm Ross (1989) Fibrous Minerals, Mining and Disease. Rept # 012. Geology & Public Policy Com., Geological Society of America. 11 pages
36. Broeder, N. and Skinner, H.C.W. (1992) Beads from the 1986 Season. in MacDonald, B. (Ed.) The Southern Ghors and Northeast 'Arabah Archaeological Survey. Sheffield Archaeological Monographs 5, J.R. Collis, Pub., U. of Sheffield, England. 40 p.
37. Skinner, H.C.W. and Robert W. Fitzpatrick (1992) Biomineralization Processes of Iron and Manganese: Modern and Ancient Environments. Supplement #21 CATENA, Germany. 432 p.
38. J.B. Dixon and H.C.W. Skinner (1992) Manganese minerals in surface environments. in Skinner, H.C.W. and R.W. Fitzpatrick Biomineralization Processes of Iron and Manganese: Modern and Ancient Environments. Sup.#21 CATENA, Germany.
39. Skinner, H.C.W. (1993) A review of apatites, iron and manganese minerals and their roles as indicators of biological activity in black shales. Precambrian Research 61:209 229.
40. Ross, Malcolm and H.C.W. Skinner (1994)Geology and Health Geotimes 39(1):10-12.
41 Skinner, H.C.W. and Malcolm Ross (1994) "Minerals and Health" Geotimes 39(1):13-15.
42 Skinner, H.C.W. (1996) "Public Health and Earth Science." Encyclopedia of Earth Sciences, Macmillan, Inc., New York.
43. Skinner, H.C.W. (1996) "Women in Earth Sciences." Encyclopedia of Earth Sciences, Macmillan, Inc.

44. Petruk, William and H.C.W. Skinner (Dec 1996) Characterizing particles in airborne dust by image analysis. JOM (Journal of the Minerals, Metals & Materials Society)58- 61.

45. Pawlikowski, Maceiej, Roman Pfitzner, H. Catherine W. Skinner (1996) Mineral cholesterol concentrations of the aortic aneurysmatic wall. Polish J. Pathology 47:225- 232.

46. Dana's New Mineralogy, 8th Edition (1997) Richard Gaines, Skinner,H.C.W., Eugene Foord,Brian Mason, Abe Rosensweig. John Wiley & Sons, N.Y. 1819 p.

47. Skinner, H.C.W. and Jillian F. Banfield (1997) Microbes All Around. Geotimes 42(8):16-19.

48. Skinner, H.C.W. (1997) Preface to Geomicrobiology: Interactions between Microbes and Minerals. Reviews in Mineralogy. Vol #35 Ed. by Banfield, J.F. and K. Nealson Mineralogical Society of America, Washington, D.C. p.1-4.

49 Wylie, Ann, H. Catherine W. Skinner, Joanne Marsh, Howard Snyder, Carmela Garzione, Damian Hodkinson, Roberta Winters and Brooke T. Mossman (1997)Mineralogical features associated with cytotoxic and proliferative effects of fibrous Talc and Asbestos on Rodent tracheal epithelial and pleural mesothelial Cells. Toxicology and Applied Pharmacology 147:143-150.

50. Skinner, H.C.W (1998) Medical Geology. In *Oxford Companion to the Earth.*

51 Poggi, S.H., Skinner, H.C.W., Ague, J.J. and Carter, D. (1998) Using scanning electron microscopy to study mineral deposits in breast tissue. Amer. Min. 83:1122-1126.

52 Skinner, H.C.W. (1999) Paracelsus Revisited: Geology and Health. Electronically published on Web site of International Geography Commission, UNESCO and published in COGEOENVIRONMENT Newsletter (May, 2000).

53 Seilacher, A., Marshall, C, Skinner, H.C.W. & Takanobu, T. (2000) A fresh look at sideritic "coprolites". Paleobiology 27: 7-13.

54 Skinner, H.C.W. (2000) In praise of phosphates, or why vertebrates chose apatites to mineralize their skeletal elements. Int. Geol. Review 42:232-240.

55. Macintyre, I.G., Bayer, F.M., Logan, A.M.V. & Skinner, H.C.W. (2000) Possible vestige of early phosphatic biomineralization on gorgonian octocorals (Coelenterata Geology 28: 455-458.

56. Skinner, H.C.W. (2000) Minerals and Human Health. In Vaughan, D.J. and Wogelius, R.J.(Editors) European Mineralogical Union Short Notes in Mineralogy. Eotvos University Press, Budapest. Hungary.

57. Poggi, S.H., Bostrom, K.I., Demer, L.L., Skinner, H.C.W. and Koos, B.J. (2001) Placental Calcification: a metastatic process? Placenta 22:591-596.

58. Skinner, H.C.W. (2000) In praise of phosphates, or why vertebrates chose apatite to mineralize their skeletal elements. Int. Geol. Review 42:232-240

59. Finkelman, R.B., Skinner, H.C.W., Plumlee, G.S. and Bunnell, J.E. (2001) Medical Geology p. 20-23 in Geotimes 46 (November Issue).

60. Skinner, H.C.W. and Berger, A.R. (2003) *Geology and Health: Closing the Gap.* Oxford University Press.

61. Skinner, H.C.W. (2003) Medical Geology In McGraw-Hill Yearbook of Science and Technology.

62. Broeder, Nancy H. and Skinner, H.C.W. (2003) Jewelry and Ornaments. Chap. 19 p. 566-598 in *Bab edh-Dhra': Excavations at the Town Site (1975-1981)*

63. Skinner, H.C.W. and Jahren, A.H. (2003) Biomineralization Chapter 4 Vol 8 of *Treatise on*

Geochemistry [Edited by F. Holland and K.K. Turekian] Elsevier, New York.

64. Skinner, H.C.W. (2003) Mineralogy of asbestos minerals. Indoor Built Environment 12(6):385 389

65. Skinner, H.C.W. Nicolescu, S, and Raup, T.D.(2004) A tale of two apatites p. 283-288 in Petrescu, I and Ozunu, A (eds) Environment and Progress Studia Universitatis Babes-Bolyai, Seria Arbientum EFES Vol. 2

66. Skinner, H.C.W. (2005) Mineralogy of Bone. Chapter 28 In *Medical Geology* Olle Selinus, Ed., Academic Press.

67. Skinner, H.C.W. (2005) The Web of Magnesium. International Geology Review 47:1111-1119.

68. Skinner, H.C.W. (2005) Biominerals. Mineralogical magazine 69:621-641.

Aeolus, Inc.
751 Taft St., Albany, California 94706
(510)-524-7855 Fax: (510)-524-7854 bermanw@comcast.net

June 30, 2006

Mr. William C. Ford
Senior Vice President
National Stone, Sand & Gravel Association
1605 King St.
Alexandria, Virginia 22314

RE: Final Report

Dear Mr. Ford:

I am pleased to submit the enclosed report, "Evaluation of the Approach Recently Proposed for Assessing Asbestos-Related Risk in El Dorado County, California." This report was prepared in response to your request that I comment on some of the issues currently being discussed concerning the evaluation of asbestos-related risks in the County. Given the extensive work I have done over the last 20 years in developing mutually consistent methods for measuring asbestos and a companion protocol for assessing asbestos-related risk (primarily for the U.S. Environmental Protection Agency – EPA), I thought my perspective on these issues could prove helpful.

Having worked with EPA for most of my career, I understand that the Agency strives first and foremost to protect public health. I also understand the need to respect precedent, as the regulatory environment needs to remain stable so that the regulated community can anticipate requirements as they go about their activities. Thus, change must be slow and deliberate. Modifications to Agency policy must consequently occur only after substantial and formal review. Therefore, I have endeavored to frame my comments with these constraints in mind. In fact, I have explicitly addressed issues associated with both precedent and the overall protection of public health in the enclosed report.

Interestingly, the concerns over asbestos being aired in El Dorado County are not new. They were raised at least as far back as 1998, when I was personally invited by Mr. Peter M. Rooney, then the head of the California EPA, to serve as a member of the State Asbestos Task Force. At that time (and ever since) I have been recommending that a detailed (and proper) characterization of the nature and distribution of asbestos in County soil and rock be conducted in a manner suitable for supporting risk assessment. Unfortunately, however, this still remains to be completed. Hopefully, the recent attention that is focused on asbestos in the County will serve as the impetus to

complete the required investigations and evaluations so that County residents will finally be provided with the information needed to make informed decisions about asbestos and their lives.

If you have any questions about the enclosed report, please do not hesitate to contact me.

Sincerely,

D. Wayne Berman

D. Wayne Berman, Ph.D.
President

EVALUATION OF THE APPROACH RECENTLY PROPOSED FOR ASSESSING ASBESTOS-RELATED RISK IN EL DORADO COUNTY, CALIFORNIA

D. Wayne Berman, Ph.D.
Aeolus, Inc.
751 Taft St.
Albany, CA 94706

Prepared at the request of:
William C. Ford
Senior Vice President
National Stone, Sand & Gravel Association
1605 King St.
Alexandria, Virginia 22314

June 30, 2006

TABLE OF CONTENTS

Section	Title	Page
1	**Executive Summary**	2
2	**Introduction**	4
3	**Background**	5
3.1	Terminology	5
3.2	The Characteristics of Asbestos Dusts	7
3.3	Asbestos Measurements and their Corresponding Exposure Metrics	9
3.4	Issues Associated with Estimating Risk Attributable to Asbestos Exposure	14
3.5	The Nature of Conditions in El Dorado County	16
4	**Evaluating the Proposed EPA Approach in El Dorado County**	16
4.1	The State of the Science	16
4.1.1	The limitations of the PCMe metric	17
4.1.2	General limitations of the Ladd (2005) study	21
4.1.3	Implications from the literature concerning cleavage fragments	26
4.2	Considering Precedent	28
4.2.1	Approaches used at other government-lead sites	29
4.2.2	A Comparison of the relative degree of review of the proposed approach and the Berman and Crump approach	32
4.3	Considering Health Protectiveness	34
5	**Conclusions**	36
6	**References**	37
	Tables and Figures	44

1 EXECUTIVE SUMMARY

The U.S. Environmental Protection Agency (EPA) recently conducted a multi-media assessment of exposure to naturally occurring asbestos (NOA) in El Dorado County (Ladd 2005). In this study, exposure to asbestos was evaluated by monitoring airborne concentrations obtained both under ambient conditions and while various recreational activities were simulated at locations selected because the soil was believed to contain NOA. An approach was also proposed in this study for assessing the risks associated with the observed exposures.

The merits of the approach proposed by the U.S. Environmental Protection Agency (EPA) for assessing asbestos-related risk in El Dorado County were evaluated. The approach involves assessing asbestos exposures by determining the concentration of airborne structures satisfying a particular set of dimensions defined in what is termed the phase contrast microscopy equivalent (PCMe) metric and combining these with the current EPA-recommended risk factor (IRIS Current) to assess risk.

The evaluation was conducted by considering:

- the current status of science and the limitations of the PCMe metric;
- the historical consistency with which the PCMe metric has been applied;
- the general limitations of the Ladd (2005) study;
- implications from the literature concerning structure sizes and types;
- precedents set by approaches used for assessing risk at other government-lead sites;
- the relative degree of peer and regulatory review for the various steps of the proposed approach and an alternate approach also considered (the approach for assessing asbestos-related risks proposed by Berman and Crump); and
- the degree of overall health protectiveness afforded by the approach proposed for El Dorado County relative to that afforded by the approach proposed by Berman and Crump.

<u>Conclusions</u>

The conclusions from this evaluation are that:

- it appears that the proposed approach satisfies neither of two criteria that are critical for assuring that risk assessments are reliable. First, due to substantial differences in character, exposure concentrations determined in terms of the PCMe metric in El

Dorado County (Ladd 2005) are not directly comparable to the PCM-based exposures evaluated in the epidemiology studies used to derive the risk factor in IRIS (Current). Second, the PCMe exposure metric itself has been shown not to remain reasonably proportional to risk across exposure environments. Given these findings, applying the IRIS risk factor to exposures measured in El Dorado County will not provide reliable estimates of risk;

- the Ladd (2005) study appears to suffer from quality control (QC) problems that will need to be resolved before any attempt is made to interpret the data. Even after the QC issues are resolved, however, it may prove difficult to extrapolate findings that may be gleaned from the study more broadly than to the specific locations at which airborne measurements were collected. This is because no relationship between bulk concentrations and airborne exposure measurements was established in the Ladd study;

- until the quality control issues are resolved and an appropriate statistical analysis of the data is conducted, a proper assessment of risk cannot be completed from the Ladd (2005) data. Thus, it is not possible to tell at this time whether risks estimated using either protocol structures (another exposure metric considered in this report) or PCMe structures will prove to be acceptable for the areas represented by the Ladd study environment. However, assuming that the ratios of concentrations are approximately correct, it appears that the IRIS approach for assessing risk yields a higher risk estimate than the Berman and Crump approach (another approach considered in this report) for the specific locations that were studied;

- as the above observation (should it hold up) is highly unusual, compared to findings based on broad experience at other sites, it reinforces the finding that conditions at these specific locations in El Dorado County are very different from conditions found at most sites where asbestos is a hazard (potentially including other parts of El Dorado County);

- if applied uniformly at sites across the nation (and other parts of El Dorado County), the approach proposed for assessing risk by EPA will be less health protective than if such risks are assessed using the approach proposed by Berman and Crump. This is based on a growing body of experience at multiple, varied sites;

- whatever the relative risks that might be estimated for El Dorado County based, respectively, on the approach proposed by EPA and the approach recommended by Berman and Crump (2001), it appears that the proposed EPA approach is no better supported by precedent; and

- given that (based on discussions with multiple geologists) about 30% of the soil and near-surface rock in the nation may contain amphibole, if the agency intends to apply their asbestos regulations consistently to all areas where amphibole may be present, then it is in everyone's interest to employ an approach that will adequately

distinguish situations that are potentially risky from those that are not. Otherwise, there is a potential either to miss those sites in which true risks exist or, conversely, to *unnecessarily* wreak economic havoc. Neither result is in the public interest, although the first kind of error is clearly the more important to avoid.

2 INTRODUCTION

The U.S. Environmental Protection Agency (EPA) recently conducted a multi-media assessment of exposure to naturally occurring asbestos (NOA) in El Dorado County (Ladd 2005). In this study, exposure to asbestos was evaluated by monitoring airborne concentrations obtained both under ambient conditions and while various recreational activities were simulated at locations selected because the soil was believed to contain NOA. An approach was also proposed in this study for assessing the risks associated with the observed exposures.

The approach that the EPA proposed to assess risk in El Dorado County, if it is to be applied uniformly, may not be generally protective of public health. Given the status of the science, it also appears that the approach may not be as well established by precedent as the approaches that the Agency commonly employs for other hazardous materials.

When evaluating the risks associated with exposure to asbestos, it is important to recognize that the situation with asbestos is particularly complex. Following a brief background discussion highlighting the complexity of the issues involving asbestos sampling, analysis, exposure assessment, and risk assessment as well as conditions in El Dorado County, the remaining sections of this report address:

- scientific considerations concerning the validity and reliability of the proposed approach;
- an overview of relevant precedent;
- implications for health protectiveness;
- conclusions; and
- references.

Note, a sub-section on quality control was also added to highlight what appear to be serious quality control (QC) issues with the data set generated during the recent El Dorado County study (Ladd 2005). When the quality of data can be questioned, it is in everyone's interest to address the problem. Thus, conducting whatever corrective actions might be necessary to examine and address the problems appear to be appropriate.

Importantly, based on the available information, it is possible that the QC problems with the Ladd (2005) study are primarily related to documentation errors. Thus, these problems may be easily correctable. Nevertheless, it is not possible to determine this at this time. Therefore, before anyone should consider the data from this study to be reliable, the QC issues need to be formally addressed. To assist in initiating this effort, a discussion is provided below that is intended to better define the quality control issues that appear to be associated with these data.

3 BACKGROUND

A brief overview of asbestos terminology, the characteristics of asbestos dusts, asbestos measurement methods and their corresponding exposure metrics, and the nature of conditions in El Dorado County is provided in this section.

3.1 Terminology

Asbestos is a term traditionally used to describe a particular fibrous form (asbestiform crystalline habit) of a set of minerals from the serpentine and amphibole mineral groups. The most widely accepted (traditional) definition of asbestos includes the asbestiform habits of six of these minerals (IARC 1977). The most common type of asbestos is chrysotile, which belongs to the serpentine mineral group. Chrysotile is a magnesium silicate. The other five asbestos minerals are all amphiboles (i.e., all partially hydrolyzed, mixed-metal silicates). These are: asbestiform riebeckite (crocidolite), asbestiform grunerite (amosite), anthophyllite asbestos, tremolite asbestos, and actinolite asbestos.

All six of the minerals whose asbestiform varieties are termed asbestos occur most commonly in nonfibrous, massive crystalline habits. While unique names have been assigned to the asbestiform varieties of three of the six minerals (chrysotile and the two amphiboles noted parenthetically above) to distinguish them from their massive forms, such nomenclature has not been developed for anthophyllite, tremolite, or actinolite. Therefore, when discussing these latter three minerals, it is important to specify whether a massive habit of the mineral or the asbestiform habit is intended.

Among the difficulties associated with any discussion of asbestos risk is that the terminology developed for asbestos was designed to address the macroscopic properties of commercially useful materials. However, it is the properties of the microscopic structures that are released from bulk asbestos (when it is disturbed) and their subsequent inhalation that ultimately determine the potential for disease. Thus, the available terminology is limited and can lead to ambiguities if not carefully applied.

Among other things, for example, it has been proposed that the term asbestos be expanded to include the asbestiform habits of a broader range of amphibole minerals (see, for example IRIS Current) and the documents from the Libby, Montana Site (e.g. EPA 2003). This was also recommended by Berman and Crump (2001, 2003). The

reason for this change has been driven by increasing evidence that the asbestiform habits of all amphiboles contribute to the induction of asbestos-related diseases. It should also be noted, however, that the scientific justification for specifically applying the current procedures for assessing asbestos-related risk (e.g. IRIS Current) to these additional minerals has not been formally evaluated or reviewed heretofore.

Another important, but less obvious issue related to the definition of asbestos is the question of the size range of structures that determine biological activity, which is clearly what needs to be regulated. This affects both the measurement of asbestos and the assessment of risk, in addition to the application of regulations. This issue is addressed further in Section 2.3.

To facilitate clarity, definitions for several critical terms used in the remainder of this report are provided below.

Asbestiform means the particular crystalline habit of a mineral that exhibits the common characteristics of asbestos (e.g. highly fibrous, polyfilamentous – existing in bundles, flexible, high tensile strength, and good chemical and thermal resistance). Geologically, the dimensions of fibers formed in this habit are defined by the growth of the crystals (in contrast to cleavage fragments).

Asbestos Minerals means the suite of serpentinite and amphibole minerals currently included in the definition of asbestos when they occur in *any* of their crystalline habits.

Cleavage Fragment means a structure that is formed by physical separation from a larger crystal. Thus, the dimensions of such a structure are defined by the orientation of the weakest cleavage planes in the parent crystal, which is in contrast to the manner in which dimensions are determined for asbestiform structures.

Exposure Metric means the set of sizes, shapes, and morphological types of structures (e.g. fibers, bundles, clusters, or matrices) that are included in the determination of concentration. Sometimes, a particular exposure metric also includes mineralogical constraints (i.e. only structures identified as specific mineralogical types are included). Therefore, exposure metrics for a particular analysis are defined as a function of both the rules of the specific analytical method applied to determine concentration and the limitations of the particular instrumentation employed during the analysis.

Fiber is a relative term that has come to mean any elongated particle that satisfies specific dimensional constraints. The term is relative because the dimensional constraints placed on the definition of the term fiber are specific to the analytical method/exposure metric by which fiber concentrations are determined for a particular application.

Fibril means a single fiber of asbestos (i.e. from an asbestiform population). Single asbestiform fibers cannot be further reduced in width without altering their properties.

Fibrous is a relative term that is used to denote a material composed primarily of fibers. The term is relative because the term for fiber is relative (see above). Note, for example, a dust composed primarily of elongated particles that nevertheless satisfy the dimensional definitions for fibers from a particular application could therefore be defined as fibrous.

Fibrous structure is a collective term used to mean any fiber, bundle, cluster, or matrix. These latter terms for specific types of structures are discussed further in Section 3.2 and concisely defined in ISO (1995).

3.2 The Characteristics of Asbestos Dusts

Structures comprising the dusts from asbestiform minerals come in a variety of shapes and sizes. Not only do single, isolated fibrils vary in length and somewhat in thickness, but such fibrils may be found combined with other fibrils to form bundles (aggregates of closely packed fibrils arranged in parallel), which represent the actual structure of all large "fibers" in an asbestiform population. In turn, fibers may form clusters (aggregates of randomly oriented fibers) or (may be combined with equant particles to form matrices (asbestos fibers embedded in non-asbestos materials). Consequently, asbestiform dusts (even of one mineral variety) are complex mixtures of structures. For precise definitions of the types of fibrous structures typically found in asbestos dusts, see ISO (1995).

In addition to the above, which describes the asbestiform component of a dust, dusts created from asbestos will also contain particles from any material with which the asbestos may be associated. Thus, for example, dusts at mining and milling sites may include particles (including elongated particles that may pass as fibers) that are rock fragments (cleavage fragments) from host minerals. If the mineral being mined is the asbestos itself, likely the host mineral would simply be the massive crystalline habit of the same mineral type as the embedded asbestos.

In environments in which an asbestos dust is derived from an asbestos product (either during manufacture, as a consequence of use, or associated with disposal), the dust may also contain particles (including elongated particles that may pass as fibers) composed of any of the various other component materials of the asbestos product.

Detailed descriptions of the characteristics of dusts typically encountered at environmental and occupational asbestos sites have been reported in the literature and the following summary is based on a previously published review (Berman and Chatfield 1990). Typically, the major components of the dust observed in most environments are non-fibrous, isometric particles. A notable exception to this general observation are the dusts from asbestos textile manufacturing, which is highly fibrous.

The likely reason these dusts are fibrous is that the only major source of dust in such an environment is refined, nearly pure, asbestiform fiber (Walton 1982). However, for asbestos dusts in general, fibrous structures consistently represent only a minor fraction of the total dust. In addition, fibrous structures composed of asbestos minerals typically represent only a subset of the total number of fibrous structures that may be observed in such environments.

The magnitude of the fraction of total dust represented by fibers and the fraction of fibers composed of asbestos minerals vary from site to site. However, the fraction of asbestos in total dusts has been quantified only in a very limited number of occupational and environmental settings (see, for example, Cherrie et al. 1987 or Lynch et al. 1970).

Importantly, as the definition of the term fiber is relative (Section 3.1), the fractional concentration of fibers observed in a particular environment will vary as a function of the analytical methodology employed to determine their concentration. Historically, fibrous structures have been arbitrarily defined as structures exhibiting aspect ratios (the ratio of length to width) greater than 3:1 to distinguish them from isometric particles (Walton 1982). However, alternate definitions for fibers have also been proposed, which are believed to better relate to biological activity (see, for example, Berman et al. 1995 or Wylie et al. 1993).

The gross features of structure size distributions appear to be similar among asbestos dusts characterized to date (Berman and Chatfield 1990). The major asbestos fraction of all such dusts are small fibrous structures less than 5 µm (micrometers) in length. Length distributions generally exhibit a mode (maximum) between 0.8 and 1.5 µm with longer fibers occurring with decreasing frequency. Fibrous structures longer than 5 µm constitute no more than approximately 25% of total asbestos structures in any particular dust and generally constitute less than 10%.

In some environments, the diameters of asbestiform structures (e.g. fibers and bundles) exhibit a narrow distribution that is largely independent of length. In other environments, diameters appear to exhibit a narrow distribution about a mean for each specific length. In the latter case, both the mean and the spread of the diameter distribution increases somewhat as the length of the structures increase. Among asbestiform materials, this increase appears to be due to contributions from bundles. Thus, for example, the increase in diameter with length appears to be more pronounced for chrysotile than for the amphiboles, presumably due to an increase in the fraction of chrysotile bundles contributing to the overall distribution as length increases. This is likely true since a single chrysotile fibril exhibits the thinnest diameter of all asbestiform structures.

Only a few studies have been published that indicate the number of complex structures in asbestos size distributions. The limited data available indicate that complex structures may constitute a substantial fraction (up to one third) of total structures, at least for chrysotile dusts (see, for example, Sebastien et al. 1984). Similar results were

also obtained during a re-analysis of dusts generated from the asbestos samples evaluated in the animal inhalation studies conducted by Davis et al. (Berman et al., in preparation). This is the same re-analysis used to support a study to identify asbestos characteristics that promote biological activity (Berman et al. 1995), which is discussed further in Berman and Crump (2003).

The degree to which fibers are combined within complex structures in a particular dust may also affect the biological activity of the dust (Berman et al. 1995). Therefore, proper characterization of asbestos exposure requires that the relative contributions from each of many components of exposure be simultaneously considered. Factors that need to be addressed include the distribution of structure sizes, shapes, and mineralogy in addition to the absolute concentration of structures. Such considerations are addressed further in Berman and Crump (2003). Thus, unlike the majority of other chemicals frequently monitored at hazardous wastes sites, asbestos exposures cannot be adequately characterized by a single concentration variable.

3.3 Asbestos Measurement Methods and Their Corresponding Exposure Metrics

Exposure to asbestos primarily involves inhalation of asbestos dust and evidence indicates it is primarily the size and shape of the fibrous structures in the dust that determine potency (in addition to their absolute concentrations). As a result, estimates of asbestos exposure concentrations vary radically as a function of both the particular type of instrumentation employed for analysis and the specific method applied during the analysis (see, for example, Berman and Crump 2003). Consequently, the ability to establish the relationship between asbestos exposure and disease has been confounded by use of multiple exposure metrics and by the fact that the relationships between exposure metrics *do not remain proportional to each other* from one environment to the next.

A variety of exposure metrics have been (and are being) used for the determination of asbestos concentrations. Those most important to the discussion in this report include "PCM", "PCMe", and "protocol structures" and each of these are briefly described below. Other potentially relevant exposure metrics are also introduced and briefly described in a table at the end of this section.

PCM is the size range of particles *traditionally* included for the determination of asbestos concentrations when analyzed by phase contrast microscopy (an optical microscopy technique). These are defined as "fibers" longer than 5 µm with an aspect (length-to-width) ratio equal to or greater than 3 and exhibiting largely parallel sides. At the magnification at which this type of asbestos analysis is typically conducted (~400x), PCM fibers are also typically limited to those thicker than approximately 0.25 µm because thinner fibers cannot be seen by the microscopist. Actually, this lower limit on width also varies somewhat as a function of the condition and quality of the microscope,

the visual acuity and training of the analyst, and the type of mineral.[1] Further, because there is no mechanism for distinguishing among mineral types when conducting analysis by PCM, all particles that are observed to satisfy the defined dimensional criteria are counted. Depending on environment, these may include, for example, cellulose and other organic fibers as well as a much broader range of inorganic fibers than have traditionally been included in the definition of asbestos (see last section).

It also needs to be understood that, due to limitations in the resolution of the microscope, the internal details of the structures that are observed by PCM cannot be distinguished. Thus, what may appear to be a simple and solid fiber by PCM may in fact be a complex structure composed of finer components. A fiber visible by PCM may alternately be a component of a larger structure whose other components are too fine to resolve. In fact, it is sometimes due to these differences (as opposed simply to mineralogy) that PCM and PCMe (defined below) concentrations determined for the same sample do not coincide. This complicates the relationship between PCM and PCMe in different environments.

An account of the history of the development of the PCM exposure metric was published by Walton (1982), which traces the origin to its definition back to meetings of a group of asbestos industry personnel in Britain (The Asbestos Research Council) in 1958. Methods suitable for determining concentrations in terms of this metric have been adopted in several countries, including the United States, and the World Health Organization. One version of the method in broad use in the United States is NIOSH Method 7400 (NIOSH 1985, 1994).

PCMe or "phase contrast microscopy *equivalent*" represents a range of particles nominally exhibiting the same range of sizes and shapes as PCM fibers, except that they are adjusted to exclude contributions from any countable particles not composed of the defined set of minerals included in the definition of asbestos[2]. As indicated above, however, mineralogy may not be the only reason for differences in concentrations estimated, respectively, by PCM and PCMe.

Originally, determining a PCMe concentration formally involved use of two, complimentary analytical techniques: phase contrast microscopy (PCM) and transmission electron microscopy (TEM) with the manner in which PCMe

---

1 The ability to observe a structure using a phase contrast microscope is also a function of the contrast between the structure and the base on which it resides. If the contrast is limited, the structure will be invisible. Contrast in turn is a function of the relative refractive index of the structure and the base, which is therefore a function of the mineralogy (chemical composition) of the structure (Kenny et al. 1987).

2 As evidence of their ability to cause asbestos-related diseases has increased, the range of minerals proposed for inclusion in the definition of asbestos has been broadened in recent years from what was originally defined in IARC (1977) and even what is defined in the current version of NIOSH Method 7402 (1994) to include virtually all amphiboles.

concentrations are determined described in NIOSH Method 7402 (NIOSH 1986,1994). By this method, asbestos concentrations are determined by analyzing sample filters using both analytical techniques and the concentration estimated by PCM is then modified by a factor derived by TEM to determine a final (adjusted) asbestos concentration expressed in terms of PCMe.

Over the years, some have adapted Method 7402 by using only the TEM component to determine an absolute concentration for PCMe (rather than using it to determine an adjustment factor for the PCM component). Other modifications to the PCMe metric (such as changes to size restrictions) have also been developed over time. In 1995, for example, ISO Method 10312 (ISO 1995) incorporated a definition for PCMe that includes an upper limit of 3.0 µm on the width of a countable particle[3] and also reduces the minimum width to 0.20 µm (from 0.25 µm)[4]. Other modifications to the definition of PCMe have also been proposed in other documents.

Table 1 presents a summary of definitions for PCMe that are provided in several Federal and California sources. In descending rows, the table provides:

- the (current) year of revision for each reference cited;
- the original year that the reference was published;
- the minimum length of structures included in the definition;
- the minimum width;
- the maximum width;
- the aspect (length-to-width) ratio; and
- relevant comments.

As can be seen in Table 1, the size definitions for PCMe vary across the different documents cited. Of these, for example, ISO 10312 incorporates a maximum width. As indicated by the comments, it is also noteworthy that an entirely different procedure is employed for deriving PCMe estimates when evaluating hazards under either California Proposition 65 (COEHHA 2006) or the California Air Resources Board's background document (CARB 1986) for their Asbestos Air Toxics Control Measure (ATCM). By CARB's rules, PCMe is determined by counting total TEM structures (of "all sizes") and dividing the count by between 100 and 1,000 (depending on whether an estimate in the low or high end of their risk range is desired). In fact, another California document that is labeled as "not to be cited or quoted" suggests an intermediate value of 320. Similarly, ATSDR (2001) defines PCMe concentrations as approximately equivalent to the concentration of total TEM structures (of all sizes longer than 0.5 µm) divided by 60.

---

3 This is also consistent with the definition originally proposed for PCM (see Walton 1982).

4 While this latter change may appear minor, as shown later, even minor changes in the minimum width for PCMe actually represent critical changes because asbestos structures tend to be particularly numerous in this range of widths and they also tend to be particularly potent (see, for example, Berman and Crump 2003).

It is also interesting that the minimum width defined for PCMe structures in EPA's IRIS is twice the minimum width defined by ISO and IRIS further indicates that the correlation between PCM and TEM fiber counts is "highly uncertain." Note that EPA has applied the ISO rules to determine PCMe concentrations in El Dorado County (Ladd 2005), which suggests inconsistency with IRIS (among other things).

Overall, the information presented in Table 1 suggests a procedure that has been subject to some modification over the years (which may appear minor but can be important)[5]. Given these distinctions, it appears that PCMe concentration estimates for asbestos may not have been derived entirely consistently over time by various parties generating such estimates.

In fact, the variability in PCMe definitions and determinations described in Table 1, does not represent the full range of variability in the manner that PCMe has been defined and applied over the last 20 years. In some studies, for example, PCMe has also been informally defined simply as "all TEM fibers longer than 5 µm", with no minimum width defined. Moreover, the concentration of TEM fibers used to estimate PCMe has sometimes been obtained using methods requiring magnifications of 10,000 and greater, which could result either in the counting of substantially greater numbers of structures or somewhat smaller numbers of structures than what can be seen at the PCM magnification of approximately 400. This depends on whether more "solid" structures become visible at the greater magnification or more structures that appear solid at the lower magnification appear to be non-countable complexes of smaller structures at the higher magnification. Thus, it does not appear that determination of PCM/PCMe ratios for use in risk assessment over the last 20 years has been entirely uniform. Nor is it clear whether any of these approaches have been subjected to formal peer-review at EPA. Thus, it does not appear that an established precedent currently exists.

Protocol Structures represent a size range of asbestos structures that is expected to better correspond to those that contribute to the induction of cancer than PCM structures.[6] Implications regarding the relationship between various exposure metrics and disease induction are addressed further below. A detailed presentation of the

---

5 For example, Hwang and Gibbs (1981) suggest that the median fiber diameter for amosite asbestos observed in mining environments lies at approximately 0.35 µm (for fibers longer than 2.5 µm and remains approximately constant for longer fibers). This suggests that the fraction of such fibers that would be alternately included or excluded in an analysis may vary radically as the minimum width to be included changes between 0.2 and 0.4 µm. Thus, the ratio between PCM and PCMe may also vary radically, depending on which cutoff is selected for PCMe.

6 Importantly, the defining dimensions of protocol structures were also somewhat constrained by limitations in the published size distributions available for applying this exposure metric in the meta analysis used to evaluate its utility (Berman and Crump 2001).

rationale for the definition of protocol structures is also available (see Berman and Crump (2001).

Protocol structures are defined as a weighted average of two size ranges of structures, whose concentrations are separately determined and then combined using the following equation:

$$C_{protocol\ structures} = 0.003*C_{size\ A} + 0.997*C_{size\ B} \qquad \text{(Equation 1)}$$

where:

$C_{protocol\ structures}$ is the concentration of protocol structures;

$C_{size\ A}$ is the concentration of structures between 5 and 10 µm in length with widths less than 0.5 µm; and

$C_{size\ B}$ is the concentration of structures longer than 10 µm with widths less than 0.5 µm.

The concentration of protocol structures is typically determined by analyzing a sample by TEM using ISO Method 10312 (ISO 1995) and incorporating a modification to include only structures of the above-indicated sizes in the structure count. Importantly, the rigorous procedures defined in the ISO Method for considering contributions from both simple structures (i.e. fibers and bundles) and complex structures (i.e. clusters and matrices) and their components are incorporated into the determination of the concentration of protocol structures.

Note that including instructions for detailed characterization of complex structures contrasts with the determination of PCMe, which involves only consideration of fibers and bundles. Such lack of detailed instructions for handling the analysis of complex structures represents a further means by which inconsistency may have been introduced into determinations of PCMe.

Other exposure metrics are also considered in this report in a variety of contexts. A summary of the characteristics of all of these exposure metrics is presented in Table 2.

In Table 2, successive rows provide the following information for each exposure metric:

- the structure dimensions defining each exposure metric;
- the associated instrumentation and method required for sampling and analysis;
- the origin of the metric;
- the theoretical basis linking the metric to risk;
- other evidence supporting/refuting the relationship between the metric and risk;
- the original (design) intent of the metric;
- pre-requisites for applying the metric to assess risk; and
- the strength of evidence supporting application of the metric to environments in which asbestos may be naturally occurring.

Note that the last row is provided to indicate the degree to which the metric might be considered to be applicable to assess risks in places such as El Dorado County.

3.4 Issues Associated with Estimating Risk Attributable to Asbestos Exposure

As with any hazardous material, asbestos-related risks are typically estimated by multiplying exposure concentrations determined in a site study (such as the study conducted in El Dorado County) with an exposure/response (risk) factor that is derived from one or more control studies (such as an epidemiology study)[7]. However, asbestos is unlike other hazardous materials because the exposure metrics employed for determining and reporting its concentration are necessarily complex.

For most hazardous materials, concentrations are expressed by a single exposure metric (e.g. mass per unit volume) incorporating a single parameter: mass. In contrast, there are multiple exposure metrics for asbestos and they each necessarily incorporate multiple parameters (i.e. dimensional limitations on a range of structures). Moreover, risk can only be reasonably estimated for asbestos when the particular exposure metric used to estimate concentrations is properly matched to the exposure metric in which the corresponding risk factor is expressed. This is because concentrations estimated in each of the multiple exposure metrics that have been used for asbestos may vary by orders of magnitude for the same sample (see, for example, Berman and Crump 2003).

Choice of the particular exposure metric is also critical to the proper estimation of risk. This is because asbestos exposure metrics do not remain proportional to one another from one environment to the next. Of course, this is simply another way of saying that the size distribution of airborne structures in an asbestos dust do not remain proportional from one environment to the next (Section 3.2).

Importantly, to successfully extrapolate risk from control studies (in which potency is determined) to a site study (in which risk must be ascertained), the metric chosen to characterize exposure must satisfy *both* of two criteria:

(1) asbestos must be measured in a comparable manner in the two environments; *and*

(2) such measurements must remain reasonably proportional to the characteristics of exposure that contribute to risk.

---

7. Actually, the manner in which risk is evaluated for asbestos is somewhat more complicated than for other materials in that the relationship between exposure and risk involves a complex function of time as well as exposure level so that, strictly, risk factors and exposure concentrations may not be simply multiplied together (see, for example, Berman and Crump 2003). However, the details of such complexities are not directly relevant to the issues at hand. Thus, they will not be addressed further in this discussion.

These requirements derive from common sense (as illustrated below) and are universal. Moreover, the importance of satisfying these criteria was clearly demonstrated in a mathematical model developed by Chesson et al. (1990). If they are not satisfied, risks estimated in the traditional manner (described above) are not valid.

Satisfying the above criteria is trivial for most chemical toxins because their effects remain proportional to mass in all environments. Thus, this single exposure metric supports valid risk assessment for these toxins. Not so for asbestos. This is a direct consequence of the nature of asbestos exposure metrics (Section 3.3) and the characteristics of asbestos dusts (Section 3.2).

To illustrate how the first of the above two criteria needs to be addressed for asbestos, consider that one would clearly not apply a risk factor for nickel (derived from dose-response studies in which exposure concentrations are determined explicitly for nickel) to assess the risks from exposure concentrations measured for chromium. That is because the two exposure metrics are not comparable. Similarly, risk factors derived for one particular exposure metric (incorporating a specific size range of asbestos structures) should not be applied to exposure concentrations determined using a different exposure metric (incorporating a different size range of structures).

To illustrate how the second of the above two criteria needs to be addressed for asbestos, consider that measuring the concentrations of nickel in various study environments (each containing dusts of mixed metals) tells one nothing of the relative concentrations of chromium in those environments; there is clearly no reason to expect that the concentrations of nickel and chromium will remain proportional from one environment to the next. Thus, it would be absurd to attempt to assess chromium-related risks based on measurements of nickel. This is true even though the relationship between the risk factors for nickel and chromium is known. It is not the relative potency, but the unknown relationship between exposure concentrations that prevents extrapolation in this case.

Similarly, because different exposure metrics for asbestos do not remain proportional from one environment to the next, unless risk is assessed using an exposure metric that specifically remains proportional to biological activity, one cannot reliably assess risk. This is because, if a particular exposure metric does not remain proportional to biological activity, the relationship between this metric and the truly biologically active fraction of an asbestos dust will vary in an undefined manner between control and study environments. Thus, a risk factor defined for such a metric in a control environment will not relate in the same manner to an exposure concentration determined for that same metric in a study environment. Therefore, it would not be valid to apply such a risk factor to the exposure determined in that study environment.

Given the above, to assess asbestos-related risk, it is therefore critical that exposures determined in terms of a particular exposure metric be combined *only* with a risk factor

that is properly matched to that particular exposure metric *and* the two must be appropriate for the environments in which they are applied.

3.5 The Nature of Conditions in El Dorado County

Conditions in El Dorado County have raised concern for years. It is an established fact that asbestiform amphibole is present in the soil and rocks of El Dorado County. The real question is whether it is ubiquitous or "patchy." Thus, there are areas of El Dorado County where various kinds of activity restrictions are prudent, but there are likely other areas where they may not be required. Thus, a reliable procedure is needed to distinguish among such areas. It is also important to consider the need to be able to distinguish "clean" fill (which might be brought in from elsewhere) from either asbestos-containing fill or local, asbestos-containing soil. In fact, these needs are common to every area of the nation in which the presence of asbestos is a concern.

4 EVALUATING THE PROPOSED EPA APPROACH IN EL DORADO COUNTY

It appears that the EPA is planning to assess risk in El Dorado County primarily by applying the current EPA slope factor for asbestos (IRIS current) to estimates of PCMe exposure derived from the Ladd (2005) study. Assuming that the QC issues that are discussed in 4.1.2 are first resolved, there still appear to be several potential problems with this approach so that the Agency needs to consider:

- the state of the science informing the validity and reliability of the proposed approach, especially as applied in El Dorado County and including considerations concerning QC;
- the degree with which the proposed approach appears to be supported by precedent; and
- the associated implications concerning the general health protectiveness of the proposed approach.

4.1 The State of the Science

Relevant issues that need to be considered to address the potential validity and reliability of the proposed approach for El Dorado County are:

- the limitations of the PCMe metric;
- more general limitations of the Ladd (2005) study; and
- implications from the literature concerning cleavage fragments.

4.1.1 The limitations of the PCMe metric

The limitations of the PCMe exposure metric are reasonably well documented and include:

- that the metric does not appear to satisfy the second of the two criteria identified in Section 3.4 that are required to support reliable risk assessment (i.e. it does not remain reasonably proportional to risk across environments of interest); and

- at least when applied at sites exhibiting the specific characteristics of the areas studied by Ladd (2005), the metric may not satisfy the first of the two criteria articulated in Section 3.4 (i.e. it is not comparable to the concentrations determined in the control studies evaluated to develop the IRIS risk factor).

Regarding the first of the above, evidence that PCMe does not remain adequately proportional to risk across environments comes from a diverse variety of sources. First (and perhaps simplest), one should consider that PCMe is intended to mimic the dimensional range of structures counted by PCM. However, the dimensional range counted by PCM was never designed or intended to reflect the characteristics of asbestos that contribute to disease. Rather it was simply designed as an arbitrary index of exposure.

A history of the development of the PCM exposure metric, at least up to the time of its publication by Walton (1982), clearly indicates that the dimensions chosen for defining PCM (by a British Council in 1958) were *arbitrary* and designed primarily to facilitate analysis. Moreover, while the minimum length may have been selected with some thought for the range of structures believed to contribute to disease (although the primary motivation was to promote analytical reproducibility), the minimum width was entirely arbitrary, as it was an artifact of the choice of magnification and the type of microscope.

Further evidence that PCMe may not adequately track the characteristics of asbestos that contribute to risk also comes from a study of animal inhalation experiments (Berman et al. 1995). In that study, the ability of various exposure metrics to predict risk (including PCM/PCMe) was formally tested. In that study, PCM/PCMe was shown to provide a *statistically significant lack of fit*.

Perhaps the most compelling evidence comes from the meta analysis reported in Berman and Crump (2003). In this study, the range of variation in risk factors reported across available epidemiology studies is compared with exposure expressed, respectively, in terms of PCM (which is considered to be equivalent to PCMe in this case) and expressed in terms of long protocol structures (defined in Section 3.3 above). The results of this comparison are illustrated in Figure 1.

In Figure 1, the ratios of the maximum to the minimum values of the risk factors derived from the set of available epidemiology studies (excluding a single, negative study) are presented. The ratios for lung cancer are presented on the left and mesothelioma on the right. The ratios labeled "PCM" are derived using the PCM exposure metric and preserves the current EPA policy of a common risk factor for chrysotile and the amphiboles. The ratios labeled "protocol" are derived using long protocol structures as the exposure metric and incorporate distinct risk factors for chrysotile and the amphiboles (which is recommended in the Berman and Crump protocol).

As can be seen in Figure 1, when exposure is expressed in terms of PCM/PCMe, risk factors derived from the available epidemiology studies range over almost two orders of magnitude (by a factor of 90) for lung cancer and over more than three orders of magnitude (by a factor of 1100) for mesothelioma. With such variability across the known studies, the confidence that can be placed in extrapolating risk estimates derived from these control studies to new environments is limited.

In contrast, when the risk factors from the same set of studies is adjusted to reflect exposure in terms of the long protocol structures metric, the range of lung cancer factors drops to about 60x (a modest improvement) and the range for mesothelioma factors drops to about 30x (a substantial improvement). Thus, the confidence that risk factors derived in terms of long protocol structures can be extrapolated to new environments is substantially improved. Note, that a more formal statistical analysis (conducted without omitting the one negative study) is also presented in Berman and Crump (2003) and the results are similar.

To address whether the PCMe exposure metric satisfies the first of the two criteria needed to assure reliable risk assessment (Section 3.4), one needs to consider two issues. The first is the relationship between PCMe and the various metrics employed to assess exposure in the original epidemiology studies and the second is the relationship between the characteristics of the dusts studied in those control environments and the character of the dusts observed in El Dorado County (or at least the specific sites in El Dorado County studied by Ladd).

Table 3 presents a comprehensive list of the quantitative epidemiological studies used to support development of the slope factor for asbestos that is currently recommended by EPA (IRIS Current). In Table 3, the eight columns respectively indicate:

- the type of asbestos: chrysotile, amosite, or mixed;
- the type of operation studied;
- the specific cohort studied;
- the potency factor for lung cancer;
- the potency factor for mesothelioma;
- the majority of the types of measurements relied on to estimate exposure;
- the study reference; and
- relevant comments.

As can be seen in the sixth column of Table 3, concentrations were initially determined based on three different methods of measurement, which resulted in three different exposure metrics among these studies. These include:

- MI or midget impinger, which is a device used to determine concentrations of total respirable particles in the air;
- PCM; or
- TP or thermal precipitator, which is another device used to determine concentrations of total respirable particles in the air. Note that MI and TP measurements are not entirely comparable (Walton 1982).

The fourth designation in the sixth column of Table 3, "NS" means non-specific. To derive a dose/response factor from the Selikoff et al. (1979) study, Nicholson simply assumed that exposures to the entire cohort could be considered equal to the average exposure concentration estimated for the entire industry at the time.

As can be seen in this same column of the Table, of the 13 available risk factors for lung cancer that were considered, nine (70%) were derived primarily by measurements other than PCM and thus had to be converted. Moreover, of these, five (60%) used factors to convert the measurements to PCM that were non-study specific.

As indicated in Walton (1982), La Ville de Thetford Mines (1994), and Smith, G.W. (1968), as well as based on general commercial considerations regarding the need for pure product material, the processes that were used to separate and isolate fiber product from the ore in asbestos mills was very efficient. Thus, the fraction of host rock fragment remaining in most commercial asbestos fiber product was extremely small. This is particularly true of the textile grade material, although it is possible that slightly greater amounts of grit and dirt (left over from mining and milling) might remain with the lower grade fiber products (especially the lowest grade fiber primarily used in the manufacture of friction products).

Given the above, the last column of Table 3 indicates the potential for rock fragments (i.e. non-asbestiform cleavage fragments) composed of asbestos minerals to be present in the various control environments studied. As can be seen in the table, the only environment in which a substantial fraction of any such fragments (primarily serpentinite fragments in this case) could potentially be present is in the Quebec mine and mill environment. Yet this environment was in fact excluded from the analysis conducted to derive the recommended EPA slope factor (EPA 1986, IRIS Current).

It should also be noted from the table that most of the control environments (other than for textiles or mining/milling) potentially contain some kind of non-asbestiform fragments, but these are generally expected to be composed of materials not related to the asbestos minerals. In such environments, therefore, the potential relationship

between PCM and PCMe will be very different than what is observed in places where large numbers of amphibole rock fragments exist (such as in El Dorado County). Further evidence for this is provided by Lynch et al. (1970). Also, see Section 3.3.

Given the above and because no study of any amphibole mining or milling operation was available at the time that the analysis was conducted (EPA 1986), there are no control environments among those studied to support development of the current EPA risk factor in which amphibole rock fragments were more than a very minor component of dust exposures. Therefore, given the radically contrasting conditions in the specific locations of El Dorado County studied by Ladd (in which amphibole rock fragments appear to be plentiful), PCMe does not satisfy the first of the criteria listed in Section 3.4 when applied to environments such as that found at these specific sites.

In contrast, the exposure metric recommended by Berman and Crump should be considered applicable to the environment in El Dorado County for two reasons. First, the Quebec mining studies (e.g. Liddell et al. 1997) were not excluded from the analysis used to evaluate the metric (Berman and Crump 2003). Second, and perhaps more importantly, the more recent studies of crocidolite (amphibole asbestos) miners in Wittenoom, Australia (de Klerk et al. 1994) and the Vermiculite miners in Libby (e.g. Amandus and Wheeler 1987) were also included. Note that the vermiculite mined in Libby is contaminated with amphiboles that include both rock fragments and what appears to be particularly hazardous forms of asbestiform amphiboles (most likely due to size).

In fact, there is direct evidence of the kinds of differences in the various environments that are described in the previous paragraphs. It comes from the examination of data from every environment characterized in a set of readily available studies in which PCMe and protocol structures were simultaneously determined (including airborne dusts from asbestos products, dusts at sites in which the source of asbestos is known to be debris from commercial asbestos products, and dusts at sites in which the source of asbestos was a minor, natural contaminant of a matrix composed of a non-asbestos mineral). In virtually all of these environments, protocol structure concentrations were comparable to or greater than that of PCMe concentrations. Among other things, the above confirms that asbestiform structures are almost exclusively thin, as the thinnest structures are included in the protocol structure metric but excluded from the PCMe metric.

In contrast, the data from the Ladd (2005) study show samples in which the concentration of PCMe fibers is *two orders of magnitude* greater than the concentration of protocol structures. Based on the size distributions reported by RJ Lee for the data from Ladd (2005), only 4% of the structures longer than 5 µm are protocol structures while 96% are PCMe (although only 25% of these are respirable). Even if one assumes a greater width cutoff than the respirable limit (such as the 1.5 µm proposed by the peer review committee of the Berman and Crump protocol, ERG 2003), almost 50% of the PCMe fibers would still be excluded. Clearly, something is very different about these

samples relative to samples that have been collected in environments known to be contaminated with asbestos.

4.1.2 General limitations of the Ladd (2005) study

There appear to be two important limitations that need to be addressed before the data from the Ladd (2005) study can be properly interpreted. These are:

- QC-related issues; and
- the extent to which the results of the study can be considered generally applicable to conditions within El Dorado County (i.e. beyond the specific locations studied).

These are each addressed below.

Quality Control Issues. Based on interpretation of the data reported from the analysis of QC samples from the Ladd study, there appear to be potentially serious laboratory quality control issues.

It appears that a number of QC analyses have been performed in which either the same analyst has re-analyzed a sample by examining the same set of grid openings twice (replicate analysis) or two different analysts have independently examined the same set of grid openings from the same sample (duplicate analysis). In several cases, such analyses were also conducted in triplicate for the same sample.

Although the EPA analyses were not conducted in a fashion allowing interpretation using the formal rules of verified counting (see, for example, Turner and Steel 1994; Steel and Small 1985; and Turner and Steel 1991), their results can still be evaluated to test whether the same sets of structures were observed over the same area scanned during each analysis. If one is to have faith that analyses have been properly conducted and documented, it is critical that one be able to show that analysts see the same structures when scanning the same areas of a sample.

Importantly, the QC evaluation discussed here is based simply on an independent interpretation of the results reported in Ladd (2005) for the analyses of QC samples. This is not a case in which an independent microscopist is working to verify specific results. Thus, direct access to the samples is not required. Rather, the role being filled here is simply one of a data analyst evaluating the performance that is to be expected when data become available from multiple analyses of the same set of grid openings on the same sample.

The procedure by which the QC results are evaluated here represents a *less* severe test of the comparability of the analyses than are typically performed for verified counting. Therefore, the degree of agreement one should expect should be at least as good as what is commonly achieved during verified counting. This means that false

positives (i.e. observation of a structure by one analyst that cannot be verified by another) should represent no more than 5% of the total number of structures reported and true positives (i.e. observations of the same structure by each analyst) should represent no less than 85% of the total number of structures reported[8,9]. Yet, across the five sets of replicate or duplicate analyses that were examined, substantially worse agreement was observed.

The evaluation was conducted simply by comparing the number of primary structures that each analyst reported for each specific grid opening. If the numbers disagreed, it would be concluded that there was an error in counts on that grid opening. Since whether one value reported by a particular analyst was higher or lower than the other was not considered in this evaluation, each observed error could be due either to a false positive or a false negative. Thus, this represents the total error that might occur on a particular grid opening and the total error should be less than 20% = (1 - 85%) + 5% where the number of false negatives is assumed to be the total number minus the number of true positives (see Turner and Steel 1994).

Clearly, this is the most general possible comparison, as it entirely ignores comparisons involving specific features of any of the structures (such as type, mineralogy, or dimension). Even multiple count errors were ignored (i.e. errors in counts from particular grid openings that differ by more than one unit were still counted as a single error).

Results for the set of five samples evaluated are presented in Table 4. Note that, when the same grid openings were analyzed by three (rather than two analysts), the error rate for each analyst is reported as the number of grid openings for which a disparate number was recorded against the average of the other two analysts.

---

8 Importantly, comparing results of analyses across the same areas of a scanned surface is qualitatively different than simply comparing structure counts across multiple analyses (or independent preparations) of the same sample when each analyst analyzes unique areas of the scanned surface (i.e. different grid openings). In the latter case, at best, one can expect agreement across analyses to be no better than what is predicted based on Poisson statistics. This is because the distribution of asbestos structures on a filter are random so that the chance of encountering a certain number of structures on any particular area of the filter exhibits a statistical distribution. In contrast, however, if multiple analysts scan the same area of a sample (i.e. the same grid openings), they should observe the same, unique set of structures that were deposited on that particular area. Thus, ideally, their counts and observations should be identical.

9 Based on the performance shown to be achievable for verified counting in general (Steel and Small 1985 and Turner and Steel 1991), the targets defined above appear reasonable for analysts counting structures in support of the Ladd study and this is especially true given the extremely favorable manner in which performance is evaluated (see main body of text).

In Table 4:

- the first column provides the Sample Identification Number;
- the second column indicates the number of analyses conducted on the specific set of grid openings from the indicated sample;
- the third column indicates the total number of grid openings analyzed;
- the fourth column indicates the number of differences in counts observed between the indicated analysis and the other analyses of the sample;
- the fifth column indicates the total error rate; and
- last column indicates whether the counts are consistent (i.e. whether they exceed the total error rate).

As can be seen in Table 4, analyses from four of the five samples that were evaluated are inconsistent. That there are problems with four of these five samples, indicates that further investigation is warranted. Moreover, although the remaining 18 QC analyses conducted on the same grid openings that were reported by Ladd are not further evaluated here, the findings reported by RJ Lee (RJ Lee 2005) concerning these remaining samples suggests that the same kind of QC problems are more prevalent than what has been reported here.

Table 5 is provided both to illustrate how the estimates in counts of differences were derived for Table 4 and to illustrate the strength of the evidence that QC problems may be even worse than what is indicated by the data in Table 4.

Table 5 displays the sets of structures observed over the same set of 15 grid openings during each of three analyses conducted for sample SRA-R05-110604. Note that the data are presented in such a manner so as to line up corresponding structures in the same rows, to the extent possible. When not possible, however, a series of arrows between the columns representing each analysis are also displayed to connect structures in different rows that, however unlikely due to clear differences in character, were assumed to be equivalent. Thus, each analyst was given every possible benefit of the doubt in the evaluation described above.

For each analysis presented in Table 5, the 10 columns respectively present:

- the grid specimen number (typically, analyses are spread across grid openings from each of two grid specimens);
- the sequential number of each grid opening scanned;
- the code identifying the particular grid opening scanned;
- the code representing the manner in which the mineralogy of a particular structure was identified (see ISO 1995);
- the sequential number of each primary (isolated) structure encountered;
- the sequential number of the total number of structures encountered (including structures embedded in larger, complex structures);
- the class (type) of each structure encountered (i.e. fiber, bundle, cluster, matrix, matrix-fiber, etc., see ISO 1995);
- the length of the structure (μm);

- the width of the structure (μm); and
- the aspect ratio of the structure.

To determine the number of primary structures reported on a particular grid opening by a particular analyst (in support of the evaluation reported in Table 4), the number of primary structures (denoted by having a numerical entry in Column 5 of Table 5) for each unique grid address (denoted by the combination of grid specimen in Column 1 and the specific grid opening location in Column 3) were simply counted. These values were then compared across analysts and the total number of grid openings for which a disagreement was observed was summed (with the results presented in Column 5 of Table 4). This sum was then divided by the total number of grid openings included in each analysis to derive the fraction (percentage) of total errors that are reported in Column 6 of Table 4.

Also in Table 5, rows representing missed structures in a particular analysis (false negatives) are highlighted in pink and rows representing an unconfirmed structure (false positives) are highlighted in green. Mismatches between dimensions or structure types are highlighted in blue. Note that, although *none* of this information was used in the evaluation of performance conducted as described above (and reported in Table 4), the degree of color observable in the table suggests substantially greater problems than what is reported in Table 4. For example, as indicated at the bottom of Table 5:

- for the Original Analysis reported on the left, of the 11 structures observed during this analysis:
  - four (Nos. 3, 8, 9, and 11) are unconfirmed during either of the other analyses (rows highlighted in green);
  - two (Nos. 1 and 7) are disputed (identified during only one of the two other analyses);
  - 6 structures identified during the other analyses were entirely missed during this analysis (rows highlighted in pink); and
  - although these structures were nominally matched with other structures, the character and/or dimensions of four structures (Nos. 1, 6, a component of 6, and 7) reported in this analysis do not even reasonably match the character and/or dimensions reported for these structures during the other analyses. These discrepancies are highlighted in blue;

- for QC Analysis No. 1 (in the middle of Table 5), of the 14 structures observed during this analysis:
  - three (Nos. 3, 7, and 9) are unconfirmed during either of the other analyses (rows highlighted in green);
  - seven (Nos. 1, 2, 4, 11, 12, and 13) are disputed (identified during only one of the two other analyses);
  - 1 structures identified during the other analyses were entirely missed during this analysis (rows highlighted in pink); and

- although these structures were nominally matched with other structures, the character and/or dimensions of 12 structures (Nos. 2, 4, a component of 4, 6, 8, 10, a component of 10, 11, a component of 11, 12, 13, and 14) reported in this analysis do not even reasonably match the character and/or dimensions reported for these structures during the other analyses. These discrepancies are highlighted in blue; and

- for QC Analysis No. 2, of the 19 structures observed during this analysis:
  - 12 (Nos. 1, 4, 5, 6, 7, 8, 9, 12, 14, 15, 17, and 18) are unconfirmed during either of the other analyses (rows highlighted in green);
  - three (Nos. 2, 3, and 16) are disputed (identified during only one of the two other analyses);
  - 2 structures identified during the other analyses were entirely missed during this analysis (rows highlighted in pink); and
  - although these structures were nominally matched with other structures, the character and/or dimensions of 12 structures (Nos. 3, a component of 3, 10, 11, 13, a component of 13, a component of 15, 16, a component of 16, a component of 17, a component of 18, and 19) reported in this analysis do not even reasonably match the character and/or dimensions reported for these structures during the other analyses. These discrepancies are highlighted in blue.

The source of the errors indicated in Table 4 is not immediately apparent. However, an evaluation of all of the 57 paired analyses reported in the Ladd (2005) data set show statistical agreement among pairs. This suggests that the errors may be associated with reporting and documentation, rather than the actual performance of the analysts. Nevertheless, these problems are still serious. One cannot consider data reliable until one has confidence not only that analyses are correct, but that the results have been properly documented. Therefore, until these problems are addressed through some appropriate corrective action, one cannot place confidence in the concentrations reported in the Ladd (2005) study. This is simply because there is otherwise no independent means of confirming whether the analysts in fact saw what they reported.

The general applicability of the Ladd study. Exposures linked to a small number of specific areas within El Dorado County were studied by Ladd (2005). These include, for example, specific school yards and a nature trail (among other places). If broader conclusions concerning asbestos exposure in El Dorado County (beyond those linked exclusively to the specific areas studied) are to be derived from this study, however, the degree with which the specific locations studied reflect broader conditions in El Dorado County needs to be characterized.

It is expected that conditions in El Dorado County will vary substantially from one location to the next. This is likely true both in terms of the concentrations of serpentinite and amphibole minerals in local soils and rock as well as the fraction of such minerals

that are truly asbestiform.[10] For example, despite evidence that the fraction of true asbestiform amphibole is small in soils in the areas specifically studied by Ladd (Sections 4.1.1, 4.2.1, and 4.3), it is known that asbestiform amphibole exists in at least some parts of the county (see, for example, Davis et al. 1991).

Given the above, without tying exposure estimates from the Ladd (2005) study to bulk determinations of asbestos in the soil (e.g. through some type of appropriate modeling validated with field confirmation from a robust and properly designed study), any results derived from the Ladd study cannot be extrapolated beyond the bounds of the specific areas within which the study was actually conducted. Moreover, without developing some type of general approach to link airborne measurements to bulk measurements, it will prove impractical to conduct simulations in every area of concern around El Dorado County (let alone the nation) in which the presence of amphibole or serpentinite minerals may suggest concern with regard to the presence of asbestos.

4.1.3 Implications from the literature concerning cleavage fragments

A wealth of studies have been published that potentially provide information distinguishing the relative potencies of amphibole cleavage fragments and true asbestiform structures. These include, for example, the studies cited by Ilgren (2004)[11] and those included in the docket supporting the OSHA final rule (OSHA 1992). However, the interpretation of these studies remains controversial.

It is true that many of these studies suffer from the various kinds of limitations that commonly plague similar studies typically associated with true asbestos, including primarily the inadequate manner in which the relevant exposures have been characterized in many studies. Also, individual studies exist that "appear" to contradict the impressions gleaned from the majority of these studies. However, the apparent contradictions simply suggest a robust database that may actually provide an opportunity to evaluate and identify exposure models capable of reconciling these disparate results (see below). It is expected that a single unified model can ultimately be developed that adequately predicts the risk associated with exposure to elongated particles of serpentine and amphibole, whether asbestiform or not.

In fact, it appears that the protocol developed by Berman and Crump (2003), perhaps with minor modifications, may be close to achieving the goal of reconciling this set of literature studies. However, further study is clearly required to test this possibility.

---

10 This will also radically affect overall size distributions and thus the relationships between various exposure metrics. Thus, exposure and risk estimates will be affected, no matter how one chooses to assess risk.

11 Importantly, it is primarily the citations reported in Ilgren (2004), rather than the specific findings reported by Ilgren that should be the focus here.

Taken as a whole, the evidence from the available literature is strongly suggestive either that cleavage fragments (structure for structure) are less potent than true asbestiform structures or that populations composed primarily of cleavage fragments contain fewer structures within the size range that induces biological activity than populations containing substantial fractions of asbestiform material.

In fact, this general impression is consistent with the findings by OSHA. In their final rule, OSHA (1992) concluded that the evidence from these studies was insufficient to regulate cleavage fragments as asbestos. Nevertheless, controversies persist and these need to be thoroughly explored and reconciled.

In fact, the best interpretation of the literature may be that controversies concerning the distinction between the hazards associated with cleavage fragments and true asbestiform structures are driven primarily by use of an inappropriate metric for characterizing asbestos-related exposures. There is ample evidence that the size range represented by "regulatory fibers" (i.e. those included in the PCM/PCMe metrics) does not adequately reflect the size range of asbestos structures that predict risk (Section 4.1.1).

That the controversies surrounding cleavage fragments are largely a function of size and the associated need to employ an appropriate exposure metric when evaluating asbestos risk is directly supported by the findings of both the American Thoracic Society (ATS 1990) and the expert panel that contributed to the peer consultation workshop on the Berman and Crump protocol (ERG 2003). Both of these groups explicitly question the appropriateness of "regulatory fibers" as an exposure metric for asbestos. Moreover, given such comments, it is clear that neither the ATS nor the expert panel explicitly supports the approach proposed by EPA for assessing risks in El Dorado County.

Many studies (including the extensive work documented by Berman and Crump) point to longer and thinner structures (thinner than PCMe fibers) as the ones that contribute most to disease. Thus, once an appropriate exposure metric (which focuses on these structures) can be fully evaluated and optimized:

(1) the disparate results of the existing epidemiology studies will be fully reconciled by a unified model of exposure and risk; and

(2) the need to distinguish true fibers from cleavage fragments will be unimportant in this model. Thus, the entire controversy surrounding the differences between true fibers and cleavage fragments may simply disappear.

The exposure metric proposed by Berman and Crump (2003), even though not fully optimized (due to the limitations of the data available for supporting such optimization) already provides substantial improvement toward reconciliation of the disparate epidemiology studies (relative to that observed when exposure response factors from

these studies are expressed in terms of the regulatory fiber metric). In fact, the improvement is statistically significant for mesothelioma (Section 4.1.1).

Although it is recognized that the data set recently studied by a team from NIOSH (Kuempel et al. 2006) has limited power to evaluate such questions, the results that they report support the findings of Berman et al. (1995) and the overall direction for optimization proposed by Berman and Crump. This direction is ultimately to consider exposure metrics focusing on even longer structures than currently considered. Kuempel et al. (2006) also proposed better evaluating the cutoff for width, once an adequate data set can be found for supporting such an evaluation. Unfortunately, the available exposure characterizations are insufficient to adequately evaluate the effects of width across the published epidemiology studies (Berman and Crump 2003).

It should also be pointed out that (absent the ability to identify or manufacture study environments in which exposures are known to be pure) the best and most definitive way to resolve the controversies involving cleavage fragments would be by:

(1) reconstructing the characteristics of the historical exposures in the available epidemiology studies conducted in the complete set of environments in which exposure is known to have been almost exclusively composed of pure asbestiform structures (i.e. in the various asbestos product factories studied historically), in environments in which exposures have been demonstrably mixed (i.e. the various mining environments studied historically), and in environments in which exposures appear to have been primarily (but not necessarily exclusively) to non-asbestiform amphiboles; and

(2) conducting a meta analysis over this entire suite of studies incorporating the data derived from (1) that provides an improved characterization of the associated exposures.

If, as expected, the result of such a study would be the identification of a single exposure metric (with multiple risk factors) that would explain the observed variation in dose-response across all three sets of studies, this would provide reasonable confidence that the studies had been adequately reconciled so that risks for all of these types of sites can be adequately predicted by a single model.

4.2 Considering Precedent

To evaluate the degree with which the approach proposed by EPA for evaluating asbestos-related risk in El Dorado County is supported by precedent, it is important to consider:

- the overall consistency of approaches used to evaluate asbestos exposure and risk at government-lead sites; and

- a comparison of the relative degree of review of the proposed approach and the Berman and Crump approach.

4.2.1 Approaches used at other government-lead sites

Table 6 presents information about a set of government-lead studies in which the EPA played a major role. In fact, EPA was the lead agency on all of the projects listed except the Southdown Project for which the lead was shared with the New Jersey Department of Environmental Protection (NJDEP). These studies were selected primarily to indicate the diversity of approaches that EPA has recently taken to assess asbestos-related risk[12].

In Table 6, the studies are presented in chronological order (based on the date of the respective reports from which the information about each project was derived). Successive rows of the upper portion of the table respectively indicate:

- the year that the study was reported;
- the source of asbestos at the studied site (e.g. natural or commercial products);
- the nature of the surrounding matrix in which the asbestos is found;
- the type of asbestos;
- the types of microscopic structures associated with each matrix;
- the specific versions of the definition(s) employed for the PCMe exposure metric;
- the analytical method(s) employed to determine the concentrations of asbestos structures in the samples collected from the site; and
- the approach(es) employed to assess asbestos-related risks.

The middle portion of Table 6 provides information on the relative magnitude of risks estimated using each of the various approaches adopted in each study. This, in turn, provides a general indication of the relative degree of health protectiveness afforded by the various approaches. Rows in this section of the table respectively indicate:

- whether the ratios of risk presented in this section were observed or estimated. Risk ratios were considered to be observed if they were derived directly from risks reported in the study indicated for each of the exposure metrics considered. Risk ratios were considered to be estimated if the relevant risk estimates were not reported directly but the ratios could be extrapolated from information on the distribution of structure sizes observed in the analyses conducted to support each study;

- the ratio of risks estimated by combining PCMe concentrations with the risk factor in IRIS to risks estimated for a selected, baseline case. Because this approach also

---

12 Importantly, while the set of studies presented in Table 6 are neither comprehensive nor statistically representative of the broader range of studies conducted by EPA over the years, their review is nevertheless instructive. Moreover, the findings presented in this section requires neither comprehensiveness nor representativeness for validity.

(approximately) represents the baseline case, all the ratios in this row are reported as one;[13]

- the ratio of risks estimated by combining PCMe (as defined by COEHHA) with the risk factors recommended by COEHHA to risks estimated for the baseline case. The COEHHA definition of PCMe is provided in Table 1 under the heading: "CA Proposition 65." Note that the COEHHA definition of PCMe was only considered in the first study listed in the table (i.e. Diamond XX);

- the ratio of risks estimated using the approach recommended by Berman and Crump (2001) to the risks estimated for the baseline case; and

- based on the ratios presented in the previous rows, whether risks derived using Berman and Crump (2001) or those derived using IRIS would be expected to be larger and thus drive risk management decisions. The procedure providing the greatest estimates of risk would generally be expected to drive these decisions.

It should be noted that the ratios presented in this section of the table for El Dorado County (the last column of Table 6) are all listed in parentheses to highlight the fact that they are especially uncertain due to a need to resolve QC issues associated with the data from this study as well as the need to address other study limitations (Section 4.1.2).

The lower portion of Table 6 provides information on the risk levels equivalent to an AHERA benchmark criterion that was used in some studies to support risk management decisions. Details concerning the manner in which this benchmark was established for the various sites in which it was applied (i.e. Libby and the World Trade Center) are provided in the respective studies cited in the table for those sites.

Depending on the availability of data from a particular study, the level of risk that would be equivalent to the concentration represented by the AHERA benchmark were derived using both the risk approach employing the IRIS risk factor and for the approach

---

13 In fact, the baseline case is intended to be one in which PCMe concentrations with dimensions *matching* those indicated in IRIS would be combined with the IRIS risk factor (see Table 1). In contrast, PCMe concentrations derived in the studies presented in Table 6 actually represent PCMe structures with the dimensions defined either by NIOSH or by ATSDR, which include thinner structures than those included in the IRIS definition (see Table 1). This makes the exposure concentrations slightly larger than what would have been determined in the strict manner defined in IRIS. Thus, the ratios presented in the "IRIS (Current)" row of the table should all be somewhat smaller than one. Unfortunately, however, without access to the raw data from each study (and the time required to conduct the requisite calculations), it is not possible to determine the exact value of this ratio. Thus, they are all presented as "one" in the table, with footnotes indicating the problem.

recommended in Berman and Crump (2001)[14]. For the former, the concentration of PCMe structures equivalent to the AHERA benchmark (given the characteristics of the asbestos structures at each particular site) was first determined from the data and this was then multiplied by the risk factor in IRIS. Similarly for the Berman and Crump approach, the concentration of protocol structures (and the fraction of long protocol structures) equivalent to the concentration represented by the benchmark were first determined from the site data and the protocol structure concentration was then multiplied by a risk factor appropriate for the type and size distribution of asbestos, as described in Berman and Crump (2001). IRIS-based risk estimates and Berman and Crump-based risk estimates are presented, respectively, in the last two rows of Table 6.

A number of findings can be gleaned from the information presented in Table 6. It is apparent, for example, that the EPA has been applying the Berman and Crump protocol (or a forerunner to the protocol) to assess asbestos-related risks at least at some sites as far back as 1994. Interestingly, the Diamond XX study was also the first of several studies of asbestos roads commissioned by the EPA in which highly robust and statistically significant results were obtained (ICF Technology 1994).

It is also interesting to note that, at least at the Southdown site, the EPA supported distinguishing contributions to risk from true asbestiform structures and cleavage fragments. Thus, it appears that this issue has received past attention.

The information presented in the middle portion of Table 6 indicates that, except for the El Dorado County Study, risks estimated using the Berman and Crump protocol are equivalent to or higher than those estimated using IRIS. In fact, for sites in which amphibole asbestos is present, the Berman and Crump protocol provides risk estimates that are substantially higher than those estimated using IRIS. This observation is further supported from observations at virtually all other sites in which both approaches have been applied to assess risk. These include both sites at which asbestos is naturally occurring and sites at which the source of asbestos is debris from asbestos-containing construction materials.

That the above contrasts sharply with what is observed for the El Dorado County Study (i.e. that risks estimated using the Berman and Crump protocol are substantially lower than those estimated using IRIS) reinforces the notion that something may be radically different about the nature of exposures in the *specific* locations in which this study was conducted than for the exposures characterized at most other asbestos sites. This and related considerations are addressed further in Section 4.3, below.

The information provided in the lower portion of Table 6 reinforces the findings obtained from the middle portion. It also suggests that use of the AHERA benchmark to

---

14 Note that in all cases here, estimated risks were derived assuming lifetime-continuous exposure, which may or may not be appropriate for specific situations. Thus, such considerations need to be more carefully explored before drawing definitive conclusions.

delineate potentially hazardous exposures to asbestos may not be particularly health protective. As can be seen in the second to last row of the table, the risk equivalent to the AHERA benchmark (using IRIS) is near or at the upper end of the range of risks potentially considered acceptable by EPA (i.e. $1x10^{-6}$ to $1x10^{-4}$) for both the Libby and the World Trade Center sites. Moreover, based on the characteristics of the exposures at Libby, the risk equivalent to the AHERA benchmark estimated using the Berman and Crump approach is substantially above the range of risks potentially considered acceptable by EPA.

Unfortunately, the available data were not sufficient to estimate a risk equivalent to the AHERA benchmark using the Berman and Crump protocol at the World Trade Center site. If it is true, however, that virtually all of the asbestos observed is chrysotile (and that is not entirely clear), then the Berman and Crump protocol would not necessarily be expected to produce a risk estimate that is substantially higher.

4.2.2 A comparison of the status of review of the proposed approach with the Berman and Crump approach

Table 7 is a side-by-side comparison of the steps required to assess asbestos-related risk using, respectively, the approach proposed by EPA for El Dorado County and the Berman and Crump protocol. It also indicates what appears to be the current (review) status of each of the steps, based on a brief review of relevant documents.

In Table 7, the first column lists the major phases required for assessing risk (from acquisition of data through applying a risk factor to exposures estimated using a particular metric). Obviously, the steps of these phases had to be streamlined for brevity, although an effort was made to capture all steps in which distinctions are potentially important.

The remaining columns of Table 7 respectively indicate:

- the steps employed by EPA to develop the current risk factor for asbestos (IRIS Current) and to apply it using the approach proposed for El Dorado County;
- comments highlighting important considerations for some of these steps;
- the steps employed to develop the risk factors proposed by Berman and Crump (2001, 2003) and to apply it to El Dorado County; and
- comments highlighting important considerations for some of these steps.

As can be seen in Table 7, the Berman and Crump approach has substantially benefitted from the advantage of 14 additional years of research over development of the risk factor currently listed in IRIS. Among other things, this means that control environments potentially relevant to environments in which asbestos is naturally

occurring (and may therefore coexist with substantial contributions from massive forms of the same mineral) were considered.

It is also acknowledged in the table that the current IRIS risk factor enjoys the precedent of having been subjected to the entire, formal EPA review process needed for establishing such values. In contrast, the Berman and Crump protocol has only been subjected to an initial peer-review consultation (by a panel of 11 experts) heretofore. At the same time, even EPA staff acknowledge that the IRIS risk factor is out of date and needs to be revised (Fed Reg 2006).

What may be more important to the issues at hand, however, is the status of the steps listed in Table 7 that are subsequent to the establishment of risk factors. As can be seen in the table, because one is applying "apples directly to apples," and because the exposure metric recommended in the Berman and Crump protocol has already been converted to a TEM-dependent exposure metric (during development of the risk factor itself), no further assumptions are required (or need review) when applying the factor to assess risks at particular sites.

In contrast, as has been shown in previous sections of this report, determination of PCMe-based concentrations may not have been conducted entirely consistently heretofore. Moreover, the manner in which PCMe relates to risk in an environment such as observed in El Dorado County are entirely different than the kinds of environments studied by epidemiologists in the control studies used to derive the current risk factor in IRIS. In addition, it does not appear that either of these critical considerations have been subjected to any kind of formal agency review at this point in time.

The comment from the Peer Review Committee concerning the idea that the minimum diameter of the size range for protocol structures needs to be increased to 1.5 µm also needs to be addressed. It is important to understand that, currently, this is only a recommendation from the group of reviewers. It is *not* a finding from a formal analysis of any kind. This contrasts with the current size range limit, which has been formally evaluated as part of a meta analysis of the human epidemiology studies and extrapolated from a formal analysis of animal inhalation studies. Moreover, it is unlikely that members of the peer-review committee would suggest that such a change should be applied for exposure determination without first defining an appropriately matching risk factor (which would require that a formal meta analysis be completed using appropriate exposure data)[15].

---

[15] Unfortunately, the database of existing size distributions is not sufficiently rich to adequately evaluate the effects of length or width further than what has already been done (Berman and Crump 2003). It is important to remember, for example, that the effects of length and width are confounded so that the unfortunate length truncation of the existing database (i.e. that no information is available for the distribution of lengths beyond 10 µm) prevents more detailed consideration of either width or length using the

In fact, it is not even known whether such a change would result in risk estimates increasing or decreasing in specific environments. This is because the result of the meta analysis (which would need to be conducted to develop properly matched risk factors) would be to spread the "fixed" risk from the mortality observed in the epidemiology studies across a larger number of structures than is the case for the exposure metric currently recommended by Berman and Crump. The relative magnitude of the risk estimated using the new, thicker structures (versus current protocol structures) would then depend on the relative ratios of the two sets of structures in control studies vs. site studies.

To illustrate the above consideration, if the ratio of the new exposure metric (incorporating the thicker structures) to protocol structures is greater in the control environments (studied by epidemiologists) than in environments of interest at specific sites (where risks are assessed), then risks estimated using the new metric will be lower than risks estimated using the current (Berman and Crump) metric. Thus, it is possible that even this approach could potentially be "less health protective," although an appropriate meta analysis might (or might not) show that it is more reliable.

4.3 Considering Health Protectiveness

It is instructive to evaluate the relative degree of health protectiveness potentially afforded by the various approaches for assessing asbestos-related risk that are considered in this report. The information provided in the middle and lower sections of Table 6 can be used for this purpose.

Based on the factors presented in the row of Table 6 labeled: "Berman and Crump (2001)" and confirmed in the row labeled: "Risk Driver," it appears that the Berman and Crump protocol provides a more sensitive measure of asbestos-related risk than the approach using IRIS. Moreover, for sites in which asbestiform amphiboles are the primary contributors to exposure, risks estimated using the Berman and Crump protocol tend to be an order of magnitude or more greater than those estimated using IRIS. Such observations are further confirmed by studies at other sites (including sites at which amphibole asbestos is naturally occurring and sites at which it is derived from manufactured asbestos product debris). At virtually all such sites in which data are available for comparing the two approaches for assessing risk, the Berman and Crump protocol yields risk estimates that are substantially higher than those estimated using IRIS.

---

human epidemiology data.

It should also be noted that the results reported by NIOSH at a recent conference (Kuempel et al. 2006) tend to support the direction of the Berman and Crump work (i.e. toward very long and very thin fibers as the cause of disease), it is also important to recognize that the single environment available to the authors in this analysis is not sufficiently robust to adequately examine these kinds of questions.

The information in Table 6 also highlights the fact that the set of sites exhibiting elevated risks and the rank order of such risks varies as a consequence of the choice of the exposure metric used to assess risk. This helps to inform the question of which approaches, if applied consistently, are likely to best reflect what is known about the incidence of asbestos-related disease.

Use of the Berman and Crump protocol focuses attention on sites where long, thin, asbestiform amphiboles contribute substantially to exposure. These include (for example) sites such as Libby, where asbestos-related diseases have actually been observed among the exposed population.

In contrast, the approach proposed by EPA for use in El Dorado County (i.e. estimating exposure using the PCMe metric and combining such results with the IRIS risk factor) tends to focus attention on sites where local soils and rock contain high concentrations of non-asbestiform amphiboles (or serpentinite). Thus, locations such as the specific areas of El Dorado County studied by Ladd are emphasized. However, given that surface soils and rock over approximately 30% of the nation apparently contain substantial concentrations of non-asbestiform amphiboles with no current evidence of elevated disease in these areas, it is not clear how helpful such emphasis may be.

At the same time, the approach proposed by EPA may "miss" elevated risks at sites in which asbestiform amphiboles are present at low concentrations, but the host rock does not otherwise contain substantial concentrations of other (non-asbestiform) amphiboles. Thus, there may be situations in which "diluted" versions of Libby may be missed by this approach. Given such possibilities, it appears that the proposed EPA approach, if applied consistently, may miss potentially risky situations in various parts of the nation or even other parts of El Dorado County.

It should also be emphasized that, based on the information provided in the last two rows of Table 6, use of the AHERA benchmark as a screen for distinguishing potentially risky situations from those that are relatively safe, may not be as effective as desired (see Section 4.2.1).

One final note is also relevant here. As further work will inevitably be conducted to refine exposure metrics for assessing asbestos-related risk, it is important to debunk one widely held misconception. As it is a requirement of sound science for assessing risk, exposure concentrations estimated using any particular exposure metric should only be combined with risk factors that are properly matched to that particular exposure metric. Assuming this is the case, it is *not* true that an exposure metric resulting in greater numbers of structures being counted to determine concentration will necessarily result in greater estimates of risk than those derived using other exposure metrics.

To derive a risk factor matched to a particular exposure metric, it is first necessary to convert estimates of exposures relevant to the control studies (epidemiology studies) to the particular exposure metric. The manner in which this is accomplished is described

in detail in Berman and Crump (2003). However, the consequence of this step is that the risk factor derived from control studies will decrease as the number of structures included in exposure concentration estimates increase in these studies.

Given the above, whether risk estimated using a particular exposure metric will increase or decrease relative to a baseline case is a function of the ratio of the concentrations estimated for the particular exposure metric at the study site to the concentrations estimated at sites evaluated in the control studies. If more of the particular kinds of structures (defined by the exposure metric) are present in control study exposures than observed at a study site (relative to the baseline case), the risk estimated using the particular exposure metric will be *lower* than the baseline case for the study site in question.

5 CONCLUSIONS

Based on the evaluation presented above, it appears that the approach proposed by EPA to assess risk in El Dorado County satisfies neither of two criteria that are critical for assuring that risk assessments are reliable. First, due to substantial differences in character, exposure concentrations determined in terms of the PCMe metric in El Dorado County (Ladd 2005) are not directly comparable to the PCM-based exposures evaluated in the epidemiology studies used to derive the risk factor in IRIS (Current). Second, the PCMe exposure metric itself has been shown not to remain reasonably proportional to risk across exposure environments.

Given these findings, applying the IRIS risk factor to the exposures measured by Ladd will not provide a reliable estimate of risk. In contrast, use of the protocol structure metric combined with the appropriately matched risk factors recommended by Berman and Crump (2001)[16] can potentially provide a reliable estimate of risk for the locations studied by Ladd, subject to the additional considerations discussed below.

The Ladd (2005) study appears to suffer from quality control (QC) problems that will need to be resolved before any attempt is made to interpret the data. Even after the QC issues are resolved, however, it may prove difficult to extrapolate findings that may be gleaned from the study more broadly than to the specific locations at which airborne measurements were collected. This is because no relationship between bulk concentrations and airborne exposure measurements was established in the Ladd study.

---

16 The analyses conducted to generate the data reported in Ladd (2005) were not explicitly designed to determine concentrations of long structures (longer than 10 μm) with sufficient sensitivity and precision to support risk assessment exclusively using these longer structures. Therefore, if there is ultimately a desire to apply the Berman and Crump protocol to these data, the 2001 version of the protocol should be applied rather than the 2003 version.

Until the quality control issues are resolved and an appropriate statistical analysis of the data is conducted, a proper assessment of risk cannot be completed from the Ladd (2005) data. Thus, it is not possible to tell at this time whether risks estimated using either protocol structures or PCMe structures will prove to be acceptable for the areas represented by the Ladd study environment. However, assuming that the ratios of concentrations are approximately correct, it appears that the IRIS approach for assessing risk yields a higher risk estimate than the Berman and Crump approach for the specific locations that were studied.

As the above observation (should it hold up) is highly unusual, compared to findings based on broad experience at other sites, it reinforces the finding that conditions at these specific locations in El Dorado County are very different from conditions found at most sites where asbestos is a hazard (potentially including other parts of El Dorado County).

If applied uniformly at sites across the nation, the approach proposed for assessing risk in El Dorado County will be less health protective than if such risks are assessed using the approach proposed by Berman and Crump. This is based on a growing body of experience at multiple, varied sites.

Whatever the relative risks that might be estimated for El Dorado County based, respectively, on the approach proposed by EPA and the approach recommended by Berman and Crump (2001), it appears that the proposed EPA approach is no better supported by precedent.

Given that (based on discussions with multiple geologists) about 30% of the soil and near-surface rock in the nation may contain amphibole, if the agency intends to apply their asbestos regulations consistently to all areas where amphibole may be present, then it is in everyone's interest to employ an approach that will adequately distinguish situations that are potentially risky from those that are not. Otherwise, there is a potential either to miss those sites in which true risks exist or, conversely, to *unnecessarily* wreak economic havoc. Neither result is in the public interest, although the first kind of error is clearly the more important to avoid.

6 REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). *Toxicological Profile for Asbestos.* U.S. Department of Health and Human Services. September, 2001.

Amandus, H.E., Wheeler, R., Jankovic, J., Tucker, J., "The Morbidity and Mortality of Vermiculite Miners and Millers Exposed to Tremolite-Actinolite: Part I. Exposure Estimates." American Journal of Industrial Medicine, Vol. 11, pp. 1-14, 1987.

American Thoracic Society (ATS). "Health Effects of Tremolite." *Amer Rev Respr Disease* 142:6:pp 1453-1458. December, 1990.

Berman, D.W. *Analysis and Interpretation of Measurements for the Determination of Asbestos in Core Samples Collected at the Southdown Quarry in Sparta, New Jersey.* Prepared for the U.S. Environmental Protection Agency, Region 2 and the New Jersey Department of Environmental Protection. November 12, 2003.

Berman, D.W. and Crump, K.S. *Final Draft: Technical Support Document for a Protocol to Assess Asbestos-Related Risk.* Prepared for Mark Follensbee, Syracuse Research Corporation, Syracuse, New York and the Office of Solid Waste and Emergency Response, U.S. Environmental Protection Agency, Washington, D.C. 2003. Limited revision draft.

Berman, D.W. and Crump, K.S.; *Technical Support Document for a Protocol to Assess Asbestos-Related Risk.* Prepared for Mark Raney, Volpe Center, U.S. Department of Transportation, 55 Broadway, Kendall Square, Cambridge, MA 02142. 2001. Peer-reviewed draft.

Berman, D.W. "Asbestos Measurement in Soils and Bulk Materials: Sensitivity, Precision, and Interpretation – You *Can* Have It All." in *Advances in Environmental Measurement Methods for Asbestos, ASTM STP 1342,* M.E. Beard, H.L. Rook, Eds., American Society for Testing and Materials. Pp. 70-89. 2000.

Berman, D.W.; Crump, K.S.; Chatfield, E.J.; Davis, J.M.G.; and Jones, A. "The Sizes, Shapes, and Mineralogy of Asbestos Structures that Induce Lung Tumors or Mesothelioma in AF/HAN Rats Following Inhalation." Risk Analysis. 15:2:181-195, 1995.

Berman, D.W. and Chatfield, E.J. "Interim Superfund Method for the Determination of Asbestos in Ambient Environments, Part 2: Technical Background Document." U.S. Environmental Protection Agency publication: 540/2-90/005b, May 1990.

Berry G; Newhouse ML. Mortality of Workers Manufacturing Friction Materials Using Asbestos. *British Journal of Industrial Medicine.* 40:1–7. 1983.

California Air Resources Board. *Staff Report: Initial Statement of Reasons for Proposed Rulemaking. Public Hearing to Consider the Adoption of a Regulatory Amendment Identifying Asbestos as a Toxic Air Contaminant.* February 10, 1986.

California Air Resources Board. *Method 427: Determination of Asbestos Emissions from Stationary Sources.* March 27, 1988.

California Office of Environmental Health Hazard Assessment. *Chemicals Listed Effective February 3, 2006 as Known to the State of California to Cause Cancer.* Required as part of the Safe Drinking Water and Toxic Enforcement Act of 1986 (California Proposition 65). 2006.

Cherrie, J; Addison, J; and Dodgson, J. "Comparative Studies of Airborne Asbestos in Occupational and Non-occupational Environments Using Optical and Electron Microscope Techniques." *IARC Sci Publ* (90). pp 304-9. ISSN 0300-5038. 1989.

Chesson, J; Rench, JD; Schultz, BD; and Milne,KL. "Interpretation of Airborne Asbesos Measurements." *Risk Analysis.* 10(3):437-47. 1990.

Davis, JMG; Addison, J; McIntosh, C; Miller, BG; Niven, K; "Variations in the Carcinogenicity of Tremolite Dust Samples of Differing Morphology." *Annals New York Academy of Sciences.* pp. 473-490. 1991.

de Klerk NH; Musk AW; Armstrong BK; Hobbs MST. Diseases in Miners and Millers of Crocidolite from Wittenoom, Western Australia: A Further Followup to December 1986. *Annals of Occupational Hygiene.* 38(Suppl 1):647-655. 1994.

Dement JM; Harris RL; Symons MJ; Shy CM. Exposures and Mortality Among Chrysotile Workers. Part I: Exposure Estimates. *American Journal of Industrial Medicine.* 4:399–419. 1983a.

Dement JM; Harris RL; Symons MJ; Shy CM. Exposures and Mortality Among Chrysotile Workers. Part II: Mortality. *American Journal of Industrial Medicine.* 4:421–433. 1983b.

EPA (see, U.S. Environmental Protection Agency).

Eastern Research Group, Inc. (ERG). *Report on the Peer Consultation Workshop to Discuss a Proposed Protocol to Assess Asbestos-Related Risk.* Prepared for the Office of Solid Waste and Emergency Response, U.S. Environmental Protection Agency, Washington, D.C. 20460. Final Report, May 30, 2003.

Federal Register 71/36/pp. 9333-9336. February 23, 2006.

Finkelstein MM. Mortality Among Long-Term Employees of an Ontario Asbestos-Cement Factory. *British Journal of Industrial Medicine.* 40:138–144. 1983.

Henderson VL; Enterline PE. Asbestos Exposure: Factors Associated with Excess Cancer and Respiratory Disease Mortality. *Annals New York Academy of Sciences.* 330:117–126. 1979.

ICF Technology, *Evaluation of Risks Posed by Residents of Diamond XX Who Are Exposed to Airborne Asbestos Derived from Serpentine Covered Roadways. Final.* Prepared for: The U.S. Environmental Protection Agency, Region 9. June 9, 1994.

Ilgren, E.B. "The Biology of Cleavage Fragments: A Brief Synthesis and Analysis of Current Knowledge." *Indoor Built Environ* 13:pp 1-14. 2004.

International Agency for Research on Cancer (IARC). Monographs on the Evaluation of Carcinogenic Risks to Man. Volume 14. IARC Scientific Publications. Lyon, France. 1977.

Integrated Risk Information System (IRIS). Toxicological Review of Asbestos. U.S. Environmental Protection Agency. Office of Research and Development, National Center for Environmental Assessment. Washington, D.C. http://www.epa.gov/iris/subst/0371.htm. Current.

International Organization for Standardization. *Ambient Air-Determination of asbestos fibers - Direct-transfer transmission electron microscopy method.* ISO 10312. 1995.

Kenny, L.C.; Rood, A.P.; and Blight, B.J.N. "A Direct Measurement of the Visibility of Amosite Asbestos Fibers by Phase Contrast Optical Microscopy." *Annals of Occupational Hygiene,* 31/pp. 211-214. 1987.

Kuempel, E.D.; Stayner, L.T.; Dement, J.D.; Gilbert, S.J.; and Hein, M.J. "Fiber Size-Specific Exposure Estimates and Updated Mortality Analysis of Chrysotile Asbestos Textile Workers." Presented at the Society of Toxicology Meeting. March 6, 2006.

La Ville de Thetford Mines. *Thetford Mines a ciel ouvert: Histoire d'une ville miniere 1892-1992.* La Ville de Thetford Mines. 144 Notre-Dame Sud, Thetford Mines, Quebec, Canada G6G 5T3. 1994.

Ladd, K (Ecology and Environment, Inc.). *El Dorado Hills Naturally Occurring Asbestos Multimedia Exposure Assessment El Dorado Hills, California: Preliminary Assessment and Site Inspection Report, Interim Final.* Prepared for The U.S. Environmental Protection Agency, Region 9 under Contract No. 68-W-01-012, Work Assign. No. 001275.0440.01. May, 2005.

Liddell FDK; McDonald AD; McDonald JC. The 1891–1920 Birth Cohort of Quebec Chrysotile Miners and Millers: Development From 1904 and Mortality to 1992. *Annals of Occupational Hygiene.* 41:13–36. 1997.

Lynch JR; Ayer HE; Johnson DJ. The Interrelationships of Selected Asbestos Exposure Indices. *American Industrial Hygiene Association Journal.* 31(5):598–604. 1970.

McDonald JC; Gibbs GW; Liddell FDK. Chrysotile Fibre Concentration and Lung Cancer Mortality: A Preliminary Report. In *Biological Effects of Mineral Fibres.* Wagner JC (ed). IARC Scientific Publications. pp. 811–817. 1980a.

McDonald JC; Liddell FDK; Gibbs GW; Eyssen GE; McDonald AD. Dust Exposure and Mortality in Chrysotile Mining, 1910–1975. *British Journal of Industrial Medicine.* 37:11–24. 1980b.

McDonald AD; Fry JS; Wooley AJ; McDonald JC. Dust Exposure and Mortality in an American Chrysotile Textile Plant. *British Journal of Industrial Medicine*. 39:361–367. 1983a.

McDonald AD; Fry JS; Woolley AJ; McDonald JC. Dust Exposure and Mortality in an American Factory Using Chrysotile, Amosite, and Crocidolite in Mainly Textile Manufacture. *British Journal of Industrial Medicine*. 40:368–374. 1983b.

McDonald AD; Fry JS; Woolley AJ; McDonald JC. Dust Exposure and Mortality in an American Chrysotile Asbestos Friction Products Plant. *British Journal of Industrial Medicine*. 41:151–157. 1984.

The National Center for Environmental Assessment (NCEA). *Exposure and Human Health Evaluation of Airborne Pollution from the World Trade Center Disaster.* Office of Research and Development, U.S. Environmental Protection Agency. October, 2002.

Nicholson WJ; Selikoff IJ; Seidman H; Lilis R; Formby P. Long-Term Mortality Experience of Chrysotile Miners and Millers in Thetford Mines, Quebec. *Annals New York Academy of Sciences*. 330:11–21. 1979.

National Institute for Occupational Safety and Health (NIOSH). Method for Determination of Asbestos in Air Using Positive Phase Contrast Microscopy. NIOSH Method 7400. NIOSH, Cincinnati, Ohio, U.S.A. 1985. Current revision: 1994.

National Institute for Occupational Safety and Health (NIOSH). Method for Determination of Asbestos in Air Using Transmission Electron Microscopy. NIOSH Method 7402. NIOSH, Cincinnati, Ohio, U.S.A. 1986. Current Revision: 1994.

The Occupational Safety and Health Administration (OSHA) "Final Rule: Occupational Exposure to Asbestos, Tremolite, Anthophyllite, and Actinolite," *Federal Register* 57:110:pp 24310-24331. 1992.

Peto J. Lung Cancer Mortality in Relation to Measured Dust Levels in an Asbestos Textile Factory. In *Biological Effects of Mineral Fibres*. Wagner JC (ed.). IARC Scientific Publications. pp. 829–836. 1980a.

Peto J. The Incidence of Pleural Mesothelioma in Chrysotile Asbestos Textile Workers. In *Biological Effects of Mineral Fibres*. Wagner JC (ed.). IARC Scientific Publications. pp. 703–711. 1980b.

Peto J; Seidman H; Selikoff IJ. Mesothelioma Mortality in Asbestos Workers: Implications for Models of Carcinogenesis and Risk Assessment. *British Journal of Cancer*. 45:124–135. 1982.

RJ Lee (R.J. Lee Group, Inc.). *Final: Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project*. Prepared for the National Stone, Sand, and Gravel Association, Alexandria, Virginia. Project No. LSH306975. November, 2005.

Sebastien P; Plourde M; Robb R; Ross M. Ambient Air Asbestos Survey in Quebec Mining Towns - Part 1, Methodological Study. Environmental Protection Service, Quebec Region, 3/AP/RQ/1E. pp. 1–41. 1984.

Seidman H; Selikoff IJ; Hammond EC. Short-Term Asbestos Work Exposure and Long-Term Observation. *Annals New York Academy of Sciences*. 330:61–89. 1979.

Seidman H. Short-Term Asbestos Work Exposure and Long-Term Observation -- July 1984 Update. Department of Epidemiology, American Cancer Society. 1984.

Selikoff IJ; Hammond EC; Seidman H. Mortality Experience of Insulation Workers in the United States and Canada 1943–1976. *Annals New York Academy of Sciences*. 330:91–116. 1979.

Smith, G.W. *The Thetford Mines. 1878-1967*. Copyright: George Washington Smith, Bell Asbestos Mines Ltd. 1968.

Steel, E.B., Small, J.A., "Accuracy of Transmission Electron Microscopy for the Analysis of Asbestos in Ambient Environments." Analytical Chemistry, Vol. 57, pp. 209-213, 1985.

Turner, S. And Steel, E.B. *Airborne Asbestos Method: Standard Test Method for Verified Analysis of Asbestos by Transmission Electron Microscopy*. Version 2.0. National Institute of Standards and Technology. U.S. Department of Commerce. 1994.

Turner. S. And Steel, E.B. "Accuracy of Transmission Electron Microscopy Analysis of Asbestos in Filters: Interlaboratory Study." *Analytical Chemistry* 63(9)pp. 868-872. 1991.

U.S. Environmental Protection Agency. *Libby Asbestos Site Residential/Commercial Cleanup Action Level and Clearance Criteria. Technical Memorandum. Draft Final.* U.S. Environmental Protection Agency, Region 8. December 15, 2003.

U.S. Environmental Protection Agency. Asbestos-containing Materials in Schools; Final Rule and Notice. 40 CFR Part 763. Federal Register, Vol 52 (210): 41826-41905. October 30, 1987.

U.S. Environmental Protection Agency. Airborne Asbestos Health Assessment Update. Report 600/8-84-003F, U.S. Environmental Protection Agency. 1986.

Walton WH. The Nature, Hazards, and Assessment of Occupational Exposure to Airborne Asbestos Dust: A Review. *Annals of Occupational Hygiene*. 25:117–247. 1982.

Weill H; Hughes J; Waggenspack C. Influence of Dose and Fibre Type on Respiratory Malignancy Risk in Asbestos Cement Manufacturing. *American Review of Respiratory Disease*. 120:345–354. 1979.

Wylie AG; Bailey KF; Kelse JW; Lee RJ. The Importance of Width in Asbestos Fiber Carcinogenicity and its Implications for Public Policy. *American Industrial Hygiene Association Journal*. 54:239–252. 1993.

TABLES AND FIGURES

TABLE 1:
DEFINITIONS FOR PCM EQUIVALENT FROM VARIOUS SOURCES[a]

Source:	IRIS	NIOSH 7402[b]	ISO 10312	CARB Staff Report	CARB Method 427	CA Proposition 65	ATSDR
Year:							
Referenced	Current	1994	1995	1986	1988	2002	2001
Original	1988	1989	1995	1986	1988	1987	2001
TEM Criteria:							
Min Length (μm)	5	5	5	ND	5	ND	5
Min Width (μm):	0.4	0.25	0.2	ND	0.2 or 0.3	ND	0.3
Max Width (μm):	ND	ND	3	ND	ND	ND	3
AR:	≥3	≥3	≥3	≥3	≥3	≥3	≥3
Comments:	Indicates that correlation between TEM and PCM fiber counts are "very uncertain."	Count those structures that "would have been counted by PCM"		Defined as total TEM structures (no minimum length or width defined) divided by either 100 or 1000.		Defined as total TEM structures (no minimum length or width defined) divided by either 100 or 1000.	Defined as total TEM structures longer than 0.5 μm divided by 60
		Indicates that potential interferences include non-asbestos amphibole particles with AR ≥ 3:1 and some non amphiboles with similar diffraction patterns to amphiboles	Indicates that the method cannot distinguish between the asbestiform varieties of amphibole minerals and their non-asbestos analogs.				

NOTES:

ND means: "not defined in the method"

[a] To the extent possible, the most recent version of each of the above documents are presented, based on the results of a search of the appropriate agency websites. If there are newer versions, they are not easily located.

[b] This method was not designed to provide concentrations of asbestos fibers directly. Rather, it was designed to provide a factor that would be used to "adjust" a concentration measurement derived by PCM.

D. Wayne Berman, Aeolus, Inc.

TABLE 2:
COMPARISON OF STATUS OF VARIOUS EXPOSURE METRICS FOR EVALUATING ASBESTOS RISK

	EXPOSURE METRIC						
	Total Respirable Particles	PCM	PCMe	Total Protocol Structures	Long Protocol Structures	Long Protocol Structures Further Optimized	Long Protocol Structures Extended Across Mouth Breathing
Dimensions	AED < 10 µm	Length > 5 µm Width > ~0 25 µm Aspect Ratio ≥ 3	Length > 5 µm Width > ~0.25 µm Aspect Ratio ≥ 3	Length > 5 µm 0 5 µm > Width	Length > 10 µm 0 4 µm > Width	Dimensional criteria would be optimized based on new meta analysis	Length > 5 µm 1 5 µm > Width
Sampling and Analysis	Midget Impinger with Analysis by Optical Microscopy[a]	Membrane Filter with Analysis by Optical Microscopy[b]	Membrane Filter with Tandem Analysis by both Optical and Transmission Electron Microscope[c]	Membrane Filter with Analysis by Transmission Electron Microscopy[d]	Membrane Filter with Analysis by Transmission Electron Microscopy[d]	Membrane Filter with Analysis by Transmission Electron Microscopy[d]	Membrane Filter with Analysis by Transmission Electron Microscopy[d]
Magnification	~400	~400	500-1,000	10,000	10,000	10,000	10,000
Origin		Asbestos Research Council (ARC)[e]	NIOSH[c]	Berman and Crump[f]	Berman and Crump[g]	Hypothesis proposed by Berman[h]	Peer Review Committee[i]
Year		1958	1989	2001	2003	2001	2003
Theoretical Basis for Linking to Risk	None. In common use for particulate matter at the time[e]	Adhoc.[e] Developed primarily for analytical convenience with general recognition of need to distinguish fibers from particles.[e]	Based informally on presumption that measuring same size range as PCM (but adding mineral confirmation) would allow link to epidemiology study results	Size rationally extrapolated from findings in Berman et al 1995 with a modification required by the published size data available for application to the epidemiology studies[f,g]	Modified from 2001 protocol based on *formal hypothesis test* of effect of length on ability to reduce variability across existing epidemiology studies[g] Width interval reduced to match that indicated in Berman et al. (1995) per recommendation of the peer review committee[g,i]	Proposed for testing based on implications from the literature that even longer structures are the major contributors to risk. Would also have optimized width dimensions based on a new analysis using new data	Proposed for consideration based on general idea that this range of structures includes all structures that potentially contribute to risk. Importantly, this metric may not automatically prove more health protective than others[i]
Other Evidence	Recognized as inadequate for asbestos when extrapolating across environments[a]	(1) Recognized as inadequate for asbestos when extrapolating across environments.[a,i,e] (2) Shown not to adequately predict risk in animal inhalation studies[k]	As PCM has not been shown to reasonably predict risk, utility for extrapolating across exposure environments is questionable	In a formal meta analysis, shown to substantially reduce variability across existing epidemiology studies compared to use of PCMe.[f]	Shown to provide some improvement over 2001 protocol, based on limited hypothesis testing involving effects of length.[g]	Proposed for testing by completing a new meta analysis as soon as data from now cancelled study would have become available.[h]	The peer review committee proposed this metric for consideration as part of further meta-analysis, which is required to define matching dose-response factors for the metric.[i]
Intent	Designed originally for general application to toxins inhaled as particulate matter.[a]	Designed originally for evaluating exposure to commercial asbestos. NOT initially designed for application to other environments.[e]	Based on interferences listed in the method, it appears to have been designed originally for evaluating exposure to commercial asbestos.[c]	Designed originally for general application to asbestos in any environment.[f]	Designed originally for general application to asbestos in any environment.[g]	Proposed for consideration for application to asbestos in any environment.	Proposed for consideration for application to asbestos in any environment
Prerequisites for Implementation for Linking to Risk	No Longer Applied	None for most environments involving exposure to commercial asbestos, although direct link to risk is questionable.[a,b,l] Shown to be NOT applicable in natural environments due to presence of extensive interfering materials.[a,l,m]	NONE for environments involving exposure to commercial asbestos (but subject to some of the same limitations as PCM).[a,b,l] Applicability to natural environments still not demonstrated (and this is the current controversy).	NONE Already shown to provide substantial improvement over PCMe[f]	NONE Already shown to provide some improvement over Total Protocol Structures.[g] *Can be further optimized with data from now-cancelled study*	Need to evaluate in a formal meta-analysis both: (1) to develop appropriately matched dose-response factors and (2) to compare against the performance of Long Protocol Structures.	Need to evaluate in a formal meta-analysis both (1) to develop appropriately matched dose-response factors and (2) to compare against the performance of Long Protocol Structures.
Strength of Evidence for Supporting Extrapolation to Natural Environments	No longer Applied and clearly not applicable	Shown not to be applicable in natural environments.[a,l,m]	Applicability to natural environments still not demonstrated (and this is the current controversy).[n]	Based on a growing track record, expected not to under-estimate asbestos risk relative to PCMe.	Expected not to under-estimate asbestos risk relative to PCMe.	Unknown Will require validation with a meta analysis incorporating appropriately relevant control environments	Unknown. Will require validation with a meta analysis incorporating appropriately relevant control environments[o]

D Wayne Berman, Aeolus, Inc

TABLE 2 (cont.):
COMPARISON OF STATUS OF VARIOUS EXPOSURE METRICS USED FOR EVALUATING ASBESTOS RISKS

Notes: AED means aerodynamic equivalent diameter

[a] TBP

[b] NIOSH Method 7400 (1989). The history of the development of precursor methods predating the NIOSH Method is provided in Walton (1982).

[c] NIOSH Method 7402 (1994).

[d] ISO Method 10312 (1995), with modifications incorporated to focus on the indicated size range of structures. Note that complex structures (bundles, clusters, and matrices) are also incorporated into the counting rules

[e] Walton (1982)

[f] Berman and Crump (2001).

[g] Berman and Crump (2003).

[h] Until February of this year, I was conducting a study to generate improved characterizations of the historical exposures relevant to critical epidemiology studies, which would have been used to support a revised meta analysis The study was terminated.

[i] ERM (2003)

[j] As noted in the table, the potency assigned to structures representing any particular exposure metric needs to be determined by a formal meta analysis. If the concentrations of structures representing a particular exposure metric are more plentiful in the exposure environments of the original epidemiology studies (i e. the control environments) than in the test environments (e.g. El Dorado County), than risks estimated in such environments will be lower than if such risks are estimated using an exposure metric in which such a difference is not as extreme (or the ratios are even reversed).

[k] Berman et al. (1995)

[l] Cherrie et al. (1989).

[m] Berman (no date) Unpublished data from the Oakland Hills Fire Project.

[n] At a minimum, an appropriately matching slope factor should be redeveloped for this exposure metric from a meta analysis that appropriately incorporates considerations of environments in which cleavage fragments predominate such as the Homestake Mine in South Dakota and the Taconite Mines in Minnesota The slope factor currently being employed with this metric was derived from an analysis in which cleavage fragments were at most a minuscule component of the dusts in the environments studied (see text)

[o] The existing database of size distributions is not sufficiently rich to evaluate effects of diameter among the human epidemiology data with adequate statistical power. Among other things, for example, the existing database is truncated for length so that, due to the confounding effects of length and width, hypothesis testing using this truncated data set may not provide reliable determinations beyond what has already been reported by Berman and Crump. The study described in Footnote h was designed to provide the needed, additional data

TABLE 3:
CHARACTER OF EXPOSURES IN ENVIRONMENTS INCLUDED IN THE 1986 HEALTH EFFECTS ASSESSMENT UPDATE REVIEW OF ASBESTOS EPIDEMIOLOGY STUDIES AND REPORTED IN IRIS 1988 AS THE BASIS FOR ESTABLISHING THE CURRENT UNIT RISK FACTOR

Fiber Type	Operation	Cohort	Lung Cancer K_L x 100	Mesothelioma K_M x 100	Exposure Metric[a]	Reference	Comments
							All samples contain asbestiform fiber. The indicated samples contain fiber with:
Chrysotile	Mining and Milling	Quebec mines and mills	*Not Used*[b]		MI	(1)	serpentinite and trace amphibole cleavage fragments[b]
			Not Used[b]		MI	(2)	serpentinite and trace amphibole cleavage fragments[b]
	Friction Products	Connecticut plant	0.01		MI*	(3)	*at most*, small amounts of serpentinite cleavage fragments[c]
	Textiles	South Carolina plant	2.8		PCM	(4)	*at most*, trace serpentinite cleavage fragments
			2.5		PCM	(5)	*at most*, trace serpentinite cleavage fragments
Amosite	Insulation Manufacture	Patterson, NJ factory	4.3		PCM**	(6)	*at most*, trace amphibole cleavage fragments[d]
				1.00E-06		(7)	
Mixed	Friction Products	British factory	0.058		PCM	(8)	*at most*, trace amphibole cleavage fragments[c,d]
	Cement Manufacture	Ontario factory	6.7	1.20E-05	MI*	(9)	*at most*, trace serpentinite and amphibole cleavage fragments[e]
		New Orleans plants	0.53		MI	(10)	*at most*, trace serpentinite and amphibole cleavage fragments[e]
	Factory workers	U.S. retirees	0.49		MI*	(11)	*at most*, trace serpentinite and amphibole cleavage fragments
	Insulation Application	U.S. insulation workers	0.75	1.50E-06	NS	(12), (13)	*at most*, trace serpentinite and amphibole cleavage fragments[d]
	Textiles	Pennsylvania plant	1.4		MI*	(14)	*at most*, trace serpentinite and amphibole cleavage fragments
		Rochedale plant	1.1	3.20E-06	TP*	(15), (16)	*at most*, trace serpentinite and amphibole cleavage fragments

NOTES:

[a] Symbols in this column indicate the primary metric by which exposure was monitored in the indicated study. "MI" means midget impinger with a study specific factor applied to convert to PCM. "MI**" means midget impinger with a non-study specific conversion factor. "PCM" means phase contrast microscopy. "PCM***" means PCM, but with measurements determined at a different plant from the one where mortality was monitored. "TP**" means that the initial measurements were collected by thermal precipitator and a non study specific conversion factor was applied. "NS" means non-specific; exposures were estimated for the Selikoff et al. (1979) simply as the average concentration reportec for the overall insulation industry.

[b] Although these are the only environments in which serpentinite or amphibole cleavage fragments might be present at greater than very small amounts (due to the presence of the parent rock in which the asbestos is embedded), these studies were excluded from the EPA analysis used to derive the EPA recommended unit risk factor for asbestos.

[c] Although cleavage fragments are potentially present (at most in small amounts) in friction product environments (because the lowest grade asbestos fiber used to manufacture these materials may not have been as well purified as higher grade fiber (Walton 1982), these environments also exhibit among the lowest dose-response factors.

[d] In these environments, it is possible that particles composed of organic materials or other non-serpentinite and non-amphibole inorganic materials may be present (which are distinct from serpentinite or amphibole cleavage fragments). However, it is not clear whether any of these materials have been shown to cause cancer in other environments where asbestos was not used. Certainly, up to this point, EPA has not applied the asbestos regulations to environments where particles of these other materials might be present without asbestos also being present.

[e] In these environments, particles composed of the cementitious binders and fillers used in cement manufacture may be present (which are distinct from serpentinite or amphibole cleavage fragments). However, whether any of these materials have been found to be carcinogenic in other environments in the absence of asbestos is not relevant here. Certainly, up to this point, EPA has not applied the asbestos regulations to environments where these types of cementitious binders and fillers are present without asbestos

D. Wayne Berman, Aeolus, Inc.

TABLE 3 (cont.)

CHARACTER OF EXPOSURES IN ENVIRONMENTS INCLUDED IN THE 1986 HEALTH EFFECTS ASSESSMENT UPDATE REVIEW OF ASBESTOS EPIDEMIOLOGY STUDIES AND REPORTED IN IRIS 1988 AS THE BASIS FOR ESTABLISHING THE CURRENT UNIT RISK FACTOR

REFERENCES

(1) McDonald et al., (1980)
(2) Nicholson et al., (1979)
(3) McDonald et al., (1984)
(4) Dement et al., (1983)
(5) McDonald et al., (1983a)
(6) Seidman (1984)
(7) Seidman (1979)
(8) Berry and Newhouse (1983)
(9) Finkelstein (1983)
(10) Weill et al., (1979)
(11) Henderson and Enterline (1979)
(12) Selikoff et al., (1979)
(13) Peto et al. (1982)
(14) McDonald et al., (1983b)
(15) Peto (1980)
(16) Peto et al. (1982)

TABLE 4:
COMPARISON OF REPORTED NUMBERS OF STRUCTURES BY DIFFERENT ANALYSTS IN COMMON GRID OPENINGS

Sample Identification	Number of Analyses	Analysis Identification	Number of Grid Openings Compared	Number of Differences in Counts	Total Error Rate	Consistent?[a]
SR-B5-110604	3	Original	15	6	40%	NO
		QC Analysis 1	15	4	27%	NO
		QC Analysis 2	15	5	33%	NO
SR-B2-100604	2		14	7	50%	NO
NR-02-101104	3	Original	17	2	12%	[illegible]
		QC Analysis 1	17	1	6%	[illegible]
		QC Analysis 2	17	2	12%	[illegible]
NR-B3-101104	2		16	9	56%	NO
SFBC-21-1FD-10064	2		22	8	36%	NO

NOTES:

[a] Analyses were considered consistent if the total error rate was less than 20% (see text)

D. Wayne Berman, Aeolus, Inc.

TABLE 5:
COMPARISON OF REPLICATE EXAMINATIONS OF THE SAME GRID OPENINGS ACROSS GRID SPECIMENS PREPARED FROM SAMPLE SRA-R05-110604

Original Analysis										QA Analysis #1										QA Analysis #2									
Gr	No	Loc.	ID	Prim	Tot	Class	Len	Wid	Asp	Gr	No	Loc.	ID	Prim	Tot	Class	Len	Wid	Asp	Gr	No.	Loc.	ID	Prim	Tot	Class	Len	Wid	Asp
A	1	A2		NSD						A	1	A2		NSD						A	1	A2	AQ	1		MD1-1	20	15	1.3
A	1	A2								A	1	A2								A	1	A2	AQ		1	MF	5.5	0.55	10
										A	2	A20	AQ	1	1	F	12	2	6	A	2	A20	AQ	2	2	F	12	2	6
A	3	B11	AZQ	1	1	B	13	0.7	19	A	3	B11	AQ	2	2	B	12.5	1	12										
A	3	B11								A	3	B11	AQ	3	3	F	13	2.5	5	A	3	B11							
										A	3	B11	AQ	4		MD1-1	12	10	1	A	3	B11							
										A	3	B11	AQ		4	MF	12	1.5	8	A	3	B11							
A	3	B11																		A	3	B11	AQ	3		MD2-2	16	15	1
A	3	B11																		A	3	B11	AQ		3	MF	15	1.2	12
A	3	B11																		A	3	B11	AQ		4	MF	13	2.5	5.2
A	3	B11																		A	3	B11	AQ	4		MD1-1	7.5	6	1.2
A	3	B11																		A	3	B11	AQ		5	MF	6	0.75	8
A	3	B11																		A	3	B11	AQ	5	6	F	1.5	0.25	6
A	3	B11																		A	3	B11	AQ	6		MD1-1	20	12	1.7
A	3	B11																		A	3	B11	AQ		7	MF	12	2.5	4.8
A	3	B11																		A	3	B11	AQ	7		MD1-0	2.5	2	1.2
A	3	B11																		A	3	B11	AQ		8	MF	2.5	0.2	12
A	4	B23	AQ	2	2	F	12	1	12	A	4	B23	AQ	5	5	F	12	0.9	13										
A	4	B23																		A	4	B23	AD	8		MD1-0	10	7.5	1.3
A	4	B23																		A	4	B23	AD		9	MF	5	1	5
A	4	B23																		A	4	B23	AD	9		MD1-1	17	10	1.7
A	4	B23																		A	4	B23	AD		10	MF	12	1	12
A	5	C12	AQ	3	3	F	1.7	0.5	3.4	A	5	C12								A	5	C12							
A	5	C12	AQ	4	4	F	7	0.5	14	A	5	C12	AQ	6	6	F			14	A	5	C12	AQ	10	11	F			20
A	6	C31		NSD						A	6	C31	AQ	7		MD	15	15	1	A	6	C31		NSD					
A	6	C31								A	6	C31	AQ		7	MF	8	0.5	16	A	6	C31							
A	7	D21	AQ	5	5	F	8	1.2	6.7	A	7	D21	AQ	8	8	F			8	A	7	D21	AQ	11	12	F			6.2
B	8	D2		NSD						A	8	D2		NSD						A	8	D2	AQ	12	13	F	30	5	6
B	9	A11	AQ	6		MD21			1.1	B	9	A11								B	9	A11							
B	9	A11	AQ		6	MF			31	B	9	A11								B	9	A11							
B	9	A11	AQ		7	MF	4.8	0.3	16	B	9	A11								B	9	A11							
B	9	A11								B	9	A11	AQ	9	9	F	3	0.3	10	B	9	A11							
B	9	A11								B	9	A11	AQ	10					2	B	9	A11							
B	9	A11								B	9	A11	AQ		10	MF			19	B	9	A11							
B	9	A11								B	9	A11								B	9	A11	AQ	13		MD1-0			1
B	9	A11								B	9	A11								B	9	A11	AQ		14	MF			6.7
B	10	A30		NSD						B	10	A30		NSD						B	10	A30	AQ	14	15	F	2.5	0.25	10
B	11	B23	AQ	7	8				3.8											B	11	B23							
B	11	B23								B	11	B23								B	11	B23	AQ	15		MD1-0	6	3	1.7
B	11	B23								B	11	B23								B	11	B23	AQ		16				3.8
B	12	C1		NSD						B	12	C1		NSD						B	12	C1		NSD					
B	13	C32	AQ	8	9	F	2.5	0.3	8.3	B	13	C32								B	13	C32							
B	13	C32	AQ	9	10	F	4.9	0.7	7	B	13	C32								B	13	C32							
B										B	13	C32	AQ	11					3	B	13	C32							
B										B	13	C32	AQ		11				11	B	13	C32							
B	13	C32								B	13	C32								B	13	C32	AQ	16			7.5	1.5	1
B	13	C32								B	13	C32								B	13	C32	AQ		17				4.5
B	13	C32								B	13	C32								B	13	C32	AQ		18	MF	2.5	0.6	4.2
										B	14	D40	AQ	12	12				13	B	13	C32							
B	14	D40								B	14	D40	AQ	13	13				6	B	13	C32							
B	14	D40								B	14	D40								B	14	D40	AQ	17		MD1-0	5	5	1
B	14	D40								B	14	D40								B	14	D40	AQ		19				13
B	14	D40								B	14	D40								B	14	D40	AQ	18		MD1-1	7.5	7.5	1
B	14	D40								B	14	D40								B	14	D40	AQ		20				5
B	15	D11	AQ	10	11	B	11	1.3	8.5	B	15	D11	AQ	14	14		11	1.3	9	B	15	D11	AQ	19	21		11	1.2	9.2
B	15	D11	AQ	11	12	F	5.3	1	5.3	B	15	D11								B	15	D11							

D. Wayne Berman, Aeolus, Inc.

Key:

- Structure Missed on this G.O.
- Phantom Structure on this G.O.
- Disagreement in Structure Dimensions Between Analysts on this G.O.

Findings:

Original Analysis
of 11 structures observed, 4 are unconfirmed and 2 are disputed. Also, there are 8 missing

QC Analysis #1
of 14 structures observed, 3 are unconfirmed and 7 are disputed. Also there is 1 missing

QC Analysis #2
of 19 structures observed: 12 are unconfirmed and 3 are disputed. Also, 2 are missing.

NOTES:
Comparisons were performed using rules that are most favorable to finding agreement among the analysts.
The colors in the table highlight the descrepencies noted.

TABLE 6:
COMPARISON OF APPROACHES FOR EVALUATING ASBESTOS-RELATED RISKS APPLIED AT SELECTED GOVERNMENT-LEAD SITES

	Diamond XX[a]	World Trade Center[b]	Southdown[c]	Libby[d]	El Dorado[e]
Year of Study	1994	2002	2003	2003	2005
Source of Asbestos	Natural	Construction Products	Natural	Natural	Natural
Surrounding Matrix	Serpentinite road aggregate	Varied construction materials	Marble with massive amphibole	Soil with vermiculite and massive amphibole	Serpentinite soil with massive amphibole
Type of Asbestos	Chrysotile	Primarily Chrysotile	Amphibole Asbestos	Amphibole Asbestos	Chrysotile and Amphibole Asbestos
Type of structures	Chrysotile with serpentinite rock fragments	Pure, milled asbestos with fragments of other construction debris	Mixed massive and asbestiform amphibole with other rock fragments	Mixed massive and asbestiform amphibole with other rock fragments	Mixed massive and asbestiform serpentinite[f] and amphibole with other rock fragments
PCMe Definition[g]	(1) NIOSH; and (2) COEHHA	ATSDR	NIOSH	NIOSH	NIOSH
Analytical Method for PCMe Determination[h]	ISO (1993)	AHERA	ISO 10312	(1) ISO 10312; and (2) AHERA	ISO 10312
Risk Assessment Approach	(1) Combined $PCMe_{NIOSH}$ with IRIS URF (2) Early version of Berman and Crump Protocol	Used standards rather than risk analysis: (1) PCM < 0.1 f/ml for workers (2) $PCMe_{ATSDR}$ < 0.0003 f/ml (converted from 70 s/mm^2) for residents	(1) Combined $PCMe_{NIOSH}$ with IRIS URF (2) Berman and Crump Protocol In both cases, separately evaluated "total structures" and the asbestiform component	Combined $PCMe_{NIOSH}$ with IRIS URF	Not Yet Completed: Requires attention to QC issues
Relative Risk[i] (Relative to IRIS)					
Observed or Estimated[i]	Observed	Estimated	Observed	Estimated	Estimated
IRIS (Current)	1[j]	1[j]	1[j]	1[j]	(1[j])[k]
COEHHA PCMe (1986)	0.3x - 2x	NA[l]	NA[l]	NA[l]	NA[l]
Berman and Crump (2001)	1.2x[m]	DNA[n]	15x - 90x	5.9x - 7.5x	(0.04)[k]
Risk Driver[o]	Berman and Crump Protocol	DNA[n]	Berman and Crump Protocol	Berman and Crump Protocol	IRIS
Risk Equivalent for AHERA Benchmark[p]					
Compared to IRIS	NA[l]	8.E-05	NA[l]	1.E-04	NA[l]
Compared to B and C protocol	NA[l]	DNA[n]	NA[l]	6.E-04	NA[l]

D. Wayne Berman, Aeolus, Inc.

TABLE 6 (cont)
COMPARISON OF APPROACHES FOR EVALUATING ASBESTOSRELATED RISK APPLIED AT SELECTED EPALEAD SITES

NOTES:

[a] ICF Technology 1994

[b] NCEA 2002

[c] Berman, 2003

[d] EPA 2003

[e] Ladd 2005

[f] Asbesiform serpentinite is just a synonym for chrysotile.

[g] The PCMe definitions that are referenced in this table vary by the specific dimensions (primarily the minimum width) of the structures included when counting to determine PCMe concentrations. Thus, "NIOSH" means PCMe as defined in NIOSH 7402 (NIOSH 1994); COEHHA means PCMe as defined in COEHHA 2006; and ATSDR means PCMe as defined in ATSDR 2001. For further information about these various definitions, see Table 1.

[h] The specific analytical methods employed for determination of asbestos concentrations in each cited study (from which PCMe concentrations were estimated) are defined in this row. In this row, ISO (1993) is a draft version of ISO Method 10312 (ISO 1995) and that "AHERA" refers to the analytical method defined in the Asbestos Hazard Emergency Response Act (EPA 1987). Interestingly, for some studies, there is a mismatch in the size range defined for PCMe structures actually recorded in various studies (i.e. NIOSH) and the size range defined in the specific analytical method employed to determine PCMe concentrations (i.e. ISO 10312). For details, see Table 1.

[i] The ratios of the levels of risk estimated using the indicated approach for assessing risk to the risk estimated based on the approach recommended in IIRIS (Current) are provided in this section of the Table. When these ratios are listed as "observed" for a particular study, it means that risk estimates derived in the study itself were directly compared to derive the indicated ratios. When listed as "estimated" it means that the ratios were derived indirectly from information concerning the distribution of asbestos structure sizes reported for the site studied. Note that, when the ratios indicated for a particular approach are greater than one, it means that risks estimated using that approach would be *more* health protective than the approach recommended in IRIS *for the particular environment studied.*

[j] This footnote was added to the specific cases in which there is actually a mis-match in the size range of PCMe structures counted to determine exposure concentrations and the size range indicated in IRIS (Current). For details, see Table 1.

[k] The ratios estimated for the El Dorado County study are shown in parentheses because they are highly uncertain due to a combination of QC questions that remain to be addressed for this study and the fact that the analytical methods employed in the study may not have been optimized to adequately determine protocol structure concentrations.

[l] In this table, "NA" means not applied in the study indicated.

[m] For this one study, an earlier draft of the Berman and Crump protocol was applied, as the study was conducted 7 years prior to completing the 2001 version of the protocol.

[n] In this table, "DNA" means dimensions not analyzed (or, at least, the data are not readily available).. Thus, it was not possible to estimate relative concentrations for the exposure metric indicated.

[o] The approach for risk assessment that produced the greatest risk estimate (between the Berman and Crump protocol and IRIS) is indicated in this row for each of the site studies presented in the table. This is based simply on whether the ratios of relative risks indicated in the previous row are less than or greater than 1.

[p] The level of risk that would be equivalent to the benchmark health criteria employed in each indicated study is presented in this portion of the table. In the row labeled, "compared to IRIS," the level of risk equivalent to the health criterion is determined based on the approach in IRIS. In the row labeled, "compared to B and C protocol," the level of risk equivalent to the health criterion is determined based on the Berman and Crump protocol.

D. Wayne Berman, Aeolus, Inc.

TABLE 7:
COMPARISON OF STEPS USED TO ASSESS RISK BY THE BERMAN AND CRUMP PROTOCOL AND THE CURRENT IRIS APPROACH, RESPECTIVELY, ALONG WITH THEIR RELATIVE REVIEW STATUS

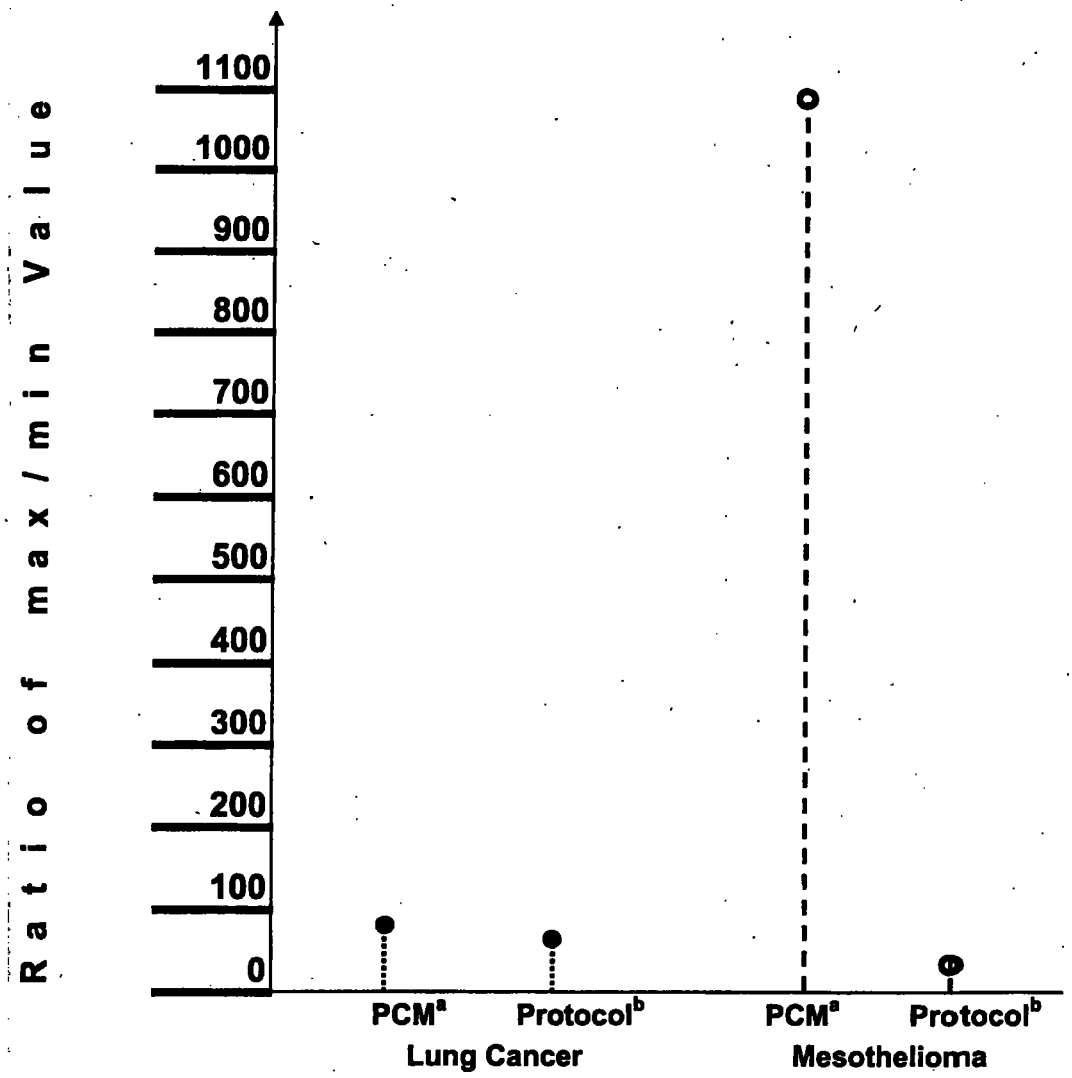
Step in Assessing Risk			
Current IRIS Approach	**Comments**	**Berman and Crump Approach**	**Comments**
Assemble Database of Control Studies			
Used 1986 database of 13 studies, rejected two studies	The studies rejected were the two available mining studies: McDonald et al. (1980) and Nicholson et al. (1979).	Used 2000 database of 19 studies	
Contains no amphibole mining studies		Includes an amphibole mining study	de Klerk et al. (1994)
Contains no amphibole contaminated mining studies		Includes 2 amphibole contaminated mining studies	Liddell et al. (1997) and Amandus and Wheeler (1987).
Derivation of Risk Factors in Control Studies			
1 Mortality Evaluated		1 Mortality Evaluated	
2 Exposure Evaluated		2 Exposure Evaluated	
3 Exposure Converted to PCM		3 Exposure Converted to PCM	
		3a Exposure Converted to Protocol Structures based on published TEM size distributions matched to each respective control study.	
4 Informally "averaged" exposure/response factors generated for PCM metric from existing studies excluding mining studies.		4 Optimized risk factors across all studies by fitting data as part of a meta analysis.	Resulting agreement across control studies is substantially improved over agreement observed using the current EPA approach (see Figure 1)
Status of Review Process for Derivation of Risk Factors			
Completed full, formal EPA review process	EPA has recognized the need to update this document and is in the process of doing so. Also, see comment on Berman and Crump approach to the right.[a]	Completed initial peer-review consultation (by a panel of 11 experts)	Review comments suggesting changes to dimensions for protocol structures are not based on formal analysis and the comment would apply equally to IRIS approach in any case.
Derivation of Exposure Estimates from Site Studies			
Determine PCMe concentrations by direct measurement using TEM		Determine protocol structure concentrations by direct measurement using TEM	
Evaluate Site Specific Risk			
Combine risk factors derived for PCM metric to exposure estimates derived in PCMe metric	Requires consistency in manner that PCMe is determined and equivalence in risk/PCMe relationship across environments. Evidence suggests neither. Process has not been subjected to formal agency review.	Combine risk factors matched to protocol structure metric with exposure estimates derived in matching metric	No assumptions required
Considerations for Application to Amphibole Contaminated Soil and Rock			
Risk factors not derived from potentially relevant control studies	Mining studies were excluded from the analysis used to derive the current IRIS risk factor.	Risk factors derived from potentially relevant control studies	The mining studies are most relevant to environments with naturally occurring asbestos

NOTES:

[a] Federal Register 2006

D. Wayne Berman, Aeolus, Inc.

FIGURE 1:
RELATIVE RANGE OF POTENCY ESTIMATES FOR LUNG CANCER AND MESOTHELIOMA BASED ON EXISTING MODELS



Notes: In all cases, ranges are evaluated using the studies available in 2000 with one negative study excluded.

[a] PCM with common potency for chrysotile and amphibole, as is current EPA policy.

[b] Long Protocol Structures with differing potency for chrysotile and amphibole, as in Berman and Crump (2003).

D. Wayne Berman, Aeolus, Inc.

Response of RJ Lee Group

to the

United States Environmental Protection Agency Region IX Response (dated April 20, 2006) to the November 2005 National Stone, Sand & Gravel Association Report Prepared by the R.J. Lee Group, Inc [sic] "Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project"

Regarding Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project

Exhibit A

Date: July 2006

Prepared by:
RJ Lee*Group*, Inc.
350 Hochberg Rd.
Monroeville, PA 15146
www.rjlg.com

Table of Contents

1.0 EPA Opening Paragraph 2
Overview 2
2.0 Background 13
3.0 Detailed Comments on the R. J. Lee Report 15
3.1 R.J. Lee Finding #1:"Based on Mineralogy, Sixty-Three Percent (63%) of the Amphibole Particles Identified as Asbestos Fibers can not be Asbestos." 15
3.2 "R.J. Lee Finding #2:"The Laboratory Procedures did not Comply With the NVLAP Quality Assurance Standard." The R. J. Lee Report says that the false positive rate in our air samples was 35% when the acceptable limit in the National Voluntary Laboratory Accreditation Program (NVLAP) is 10%." 22
3.3 R. J. Lee Finding #3:"The Soil Samples do not Demonstrate the Presence of Amphibole Asbestiform Minerals." 23
3.4 R.J. Lee Finding #4: "The ISO 10312 Analytical Method can not Distinguish Between Asbestos Fibers and Non-Asbestos Cleavage Fragments." 29
3.5 R.J. Lee Finding #5:"Applying the Latest Science and Definitional Techniques, the El Dorado Hills Study Shows no Significant Exposure to the Type of Amphibole Asbestos Fiber Connected To Health Risk." 33
4.0 R. J. Lee Report Peer Reviews 35
5.0 Conclusions 36
6.0 RJLG Attachments 37

Response of RJ Lee Group

to the
United States Environmental Protection Agency Region IX Response (dated April 20, 2006) to the November 2005 National Stone, Sand & Gravel Association Report Prepared by the R.J. Lee Group, Inc [sic] "Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project"

Regarding
Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project

Exhibit A
Attachment A-3a
Lab/Cor Photographs of Particles

Date: July 2006

Prepared by:
RJ Lee*Group*, Inc.
350 Hochberg Rd.
Monroeville, PA 15146
www.rjlg.com

Response of RJ Lee Group

to the

United States Environmental Protection Agency Region IX
Response (dated April 20, 2006) to the November 2005
National Stone, Sand & Gravel Association Report
Prepared by the R.J. Lee Group, Inc [sic]
"Evaluation of EPA's Analytical Data from the
El Dorado Hills Asbestos Evaluation Project"

Regarding
Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project

Exhibit A
Attachment A-4a
TEM and FESEM Particle Images and Data

Date: July 2006

Prepared by:
RJ Lee*Group*, Inc.
350 Hochberg Rd.
Monroeville, PA 15146
www.rjlg.com

Table of Contents

1.0 Data Description 1
2.0 Analytical Procedure 1

Response of RJ Lee Group

to the

United States Environmental Protection Agency Region IX Response (dated April 20, 2006) to the November 2005 National Stone, Sand & Gravel Association Report Prepared by the R.J. Lee Group, Inc [sic] "Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project"

Regarding Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project

Exhibit A

Attachment A-8a

Particles and their Related Extinction Angles

Date: July 2006

Prepared by:
RJ Lee*Group*, Inc.
350 Hochberg Rd.
Monroeville, PA 15146
www.rjlg.com

Table of Contents

Summary 1
1.0 Background 1
2.0 Extinction Angle Study 2
2.1 Results 2
2.2 Images of Fibers 3
2.2.1 Fibers with 3 or more Criteria of Asbestos 3
2.2.2 Fibers with 2 Criteria of Asbestos 25
2.2.3 Fibers with No Criteria of Asbestos 35

Response of RJ Lee Group to Mr. Meeker's Letter (undated) to Dr. Vicki Barber, El Dorado Hills School District

Regarding Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project

Exhibit B

Date: July 2006

Prepared by:
RJ Lee*Group*, Inc.
350 Hochberg Rd.
Monroeville, PA 15146
www.rjlg.com

Table of Contents

1.0 *"Question 1* 2
2.0 *"Question 2* 6
3.0 *"Question 3* 9
4.0 *"Concluding Remarks* 9
5.0 "References 9

Response of RJ Lee Group to EPA Region 9 (Meer) Letter dated March 9, 2006

Regarding Evaluation of EPA's Analytical Data from the El Dorado Hills Asbestos Evaluation Project

Exhibit C

Date: July 2006

Prepared by:
RJ Lee*Group*, Inc.
350 Hochberg Rd.
Monroeville, PA 15146
www.rjlg.com

Table of Contents

Preface 1

Introduction 2

Executive Overview and Policy Implications 3

Response to EPA "Questions regarding the R.J. Lee Report [sic]" attached to the EPA Region 9 (Meer) Letter dated March 9, 2006 19

1.0 EPA Air Samples 19

1.1 Evaluation of EPA Analyses 19

1.2 Evaluation of EPA Data 19

1.2.1 Count Sheets 20

1.2.2 Diffraction Patterns 20

1.2.3 Spectra 21

1.2.4 Additional Reviews Performed 21

1.2.5 Documentation of Additional Reviews 26

2.0 Soil Samples – Preparation Methodology 27

2.1 Drying Times 28

2.2 Grinding 28

2.3 Sieving 28

2.4 Other 28

3.0 Soil Samples – Analytical Methodology 29

3.1 Sample Analyses 29

3.2 Laboratory Count Sheets 29

3.3 Laboratory Notes and Logbooks 30

3.4 Images 30

3.5 Chains-of-Custody 30

3.6 Sample Tracking 30

3.7 Personal Qualifications 30

4.0 QA/QC Procedures 30

4.1 Certifications 31

4.2 Laboratory Standard Operating Procedures 31
4.3 Laboratory Monitoring 31
5.0 Review Documents 31
5.1 Third Party Audits 31
5.2 Cited Literature 32
5.3 Communications 32
6.0 RJLG QA/QC Procedures 32
7.0 Issues Related to Asbestos Identification 33
7.1 Characteristics of Asbestos Fibers 33
7.2 Iron Valence State 37
7.3 Clay Contamination of Amphibole Particles 37
7.4 Variation of SAED with Particle Size 38
7.5 Different Morphologies of Amphibole Particles 39
7.6 Analysis of Amphibole Minerals 40
7.7 Parallel Extinction of Asbestos Fibers 41
7.8 NIST SRM 1867a 42
7.9 Fiber Terminations 44
8.0 Verified Analyses 45
9.0 Reference Minerals 47
10.0 Optical Properties of Asbestos 48
11.0 Air Sample Analysis Procedures 48
11.1 Laboratory Count Sheets 52
11.2 Laboratory Notes 52
11.3 Logbook Pages 52
11.4 Sketches 52
11.5 Images 52
11.6 Spectra 52
11.7 Diffraction Patterns 52
11.8 Chain of Custody Forms 53

11.9 Sample Tracking Sheets 53
11.10 Quality Control ("QC") Sample Information 53
11.11 Required Calibrations 53
12.0 Bundles 53
13.0 Selected Area Electron Diffraction Patterns 54
14.0 Rock Samples 61
14.1 Documents, Count Sheets, EDS spectra 61
14.2 Images 61
14.3 Chain-of-Custody 61
15.0 Elutriator Tests 61
15.1 Counts Sheets and Laboratory Notes 61
15.2 Chain-of-Custody 61
16.0 Qualifications of Richard J. Lee, Ph.D. 62

Environmental