NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH)

PUBLIC MEETING

ASBESTOS AND OTHER MINERAL FIBERS: A ROADMAP FOR SCIENTIFIC RESEARCH

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P R O C E E D I N G S

MR. HEARL: Good morning. My name is Frank Hearl. I am the Chief of Staff of the National Institute for Occupational Safety and Health, NIOSH. NIOSH is in the U.S. Department of Health and Human Services and is the agency established to help assure safe and healthful working conditions for working men and women by providing research, information, education, and training in the field of occupational safety and health. On behalf of NIOSH and our Director Dr. John Howard, I want to welcome to this public meeting here in Washington, D.C.

We have organized this meeting to obtain your input and comments on the draft document "Asbestos and Other Mineral Fibers: A Roadmap for Scientific Research." As the federal agency responsible for conducting research and making recommendations for the prevention of worker injury and illness, NIOSH is undertaking a 21st century reappraisal of the areas of research needed to pursue on its own and in collaboration

with others. New scientific knowledge will be generated to serve as the basis for evidence-based public-health policies for asbestos and other mineral fibers.

NIOSH invites comments on occupational safety and health issues identified and fiber research strategies suggested in the Roadmap. We seek other views about key issues that need to be identified, additional research that needs to be conducted, and suggest methods to conduct that research. In particular, NIOSH is seeking input from stakeholders concerning study designs, techniques for size-selected fibers, analytical approaches, sources of particular types of fibers suitable for experimental studies, and worker populations suitable for epidemiological studies. We are interested in available and forthcoming research results that can help answer the questions set forth in the Roadmap. Information is also requested on existing workplace exposure data, health effects, and control technologies.

I will chair this meeting, and my

principal job here will be to make sure that everyone has a fair chance to be heard, to assure that NIOSH receives the input it is requesting, and to try to keep us on time. This meeting will be concluded at 4 o'clock today.

I would like to begin by making a few housekeeping announcements. First, in the event of an emergency, it appears that the best exit route would be out the door and to the right and directly out to the street. In the event of an evacuation, please move quickly and safely to the exists and await instructions before returning.

Second, the restroom facilities, I found two sets of restroom facilities. One is if you go out this door and then all the way to the end of the hallway up back into the lobby of the Holiday Inn there is a set of restrooms there. The second set is a little more difficult to find but probably easier to get to, and that is you go out again to the hallway and to the right past the glass wall and then turn right at the first corridor, when you go down to the end there is one

door that has a card key access and the next door has no sign on it whatsoever, but it is right next to a water foundation. If you push that door open, you will find both a men's and women's room. So those are the two sets of restroom facilities.

Third, I would like to ask everyone to please either turn off your cell phones or set them to a nonaudible vibrate mode so as not to disturb others at the meeting. If you could please do that I would thank you for your cooperation. Again, our meeting today is scheduled to run from 9:00 a.m. to 4:00 p.m., and if we have no further speakers or commenter we may close the meeting before 4 o'clock, but in looking at the number of people signed up, I do not think that is going to be our problem.

The meeting is being transcribed and we expect to have transcripts posted to the Internet as soon as they can be made available. Persons wishing to submit written comments for the record may do so by providing a copy of your comments to me today or sending them by mail, email or using

the website that we have posted on the Internet. You can get to it through the main NIOSH website www.cdc.gov/niosh. The docket will be open to receive comments on the Asbestos Roadmap until May 31, 2007.

In accordance with our Federal Register announcement and website announcement, we have a number of individuals who pre-signed up in advance here to make oral presentations. Each of those individuals will have to up to 15 minutes to make an oral presentation. If the presentation ends early, we are going to move immediately to the next presentation so we can try to make available time at the end of the meeting for anyone else who has signed up outside.

We will take a 15-minute break today around 10:30, and we will take a 1-hour and 15-minute break for lunch at 11:45 or thereabouts. And as the meeting goes this morning, I may ask us to shorten that a little bit to make again time available. And we will also take a break around 2:15.

Our last preregistered presentation now is scheduled to end around 3:30 I believe, 3:45. If you did not preregister, you may sign up to speak at the sign-up table outside the meeting room. After the last preregistered presentation is complete, I will divide the remaining time up until 4 o'clock among those who have signed up outside and you will have the chance to speak here. Like I said, after we have no more signed-up people, we may open the mike for walk-up comments until 4 o'clock.

Individuals who are making oral presentations are welcome to use their time to ask clarifying questions of the NIOSH panel members who are the principal authors of the draft, and they are seated up here at the front. Note that both question and the answer, I am going to count that against that individual's time, so I would also ask the panel members to be succinct in their responses.

The NIOSH panel members are Dr. Paul Middendorf, Dr. Robert Castellan, and Mr. Ralph

Zumwalde. I would ask that you do not address questions to the other presenters when they are up here. This is not a scientific symposium, but a public meeting to present information to NIOSH.

As a note for presenters, too, any written statement you provide will be entered into the record so there is no need for you to read your written statement. We hope the information you provide will augment the written statement and have special emphasis on the five points that we identified in the Roadmap, and that would identifying whether the hazard identification and discussion of health effects for asbestos, mineral and mineral fibers is a reasonable reflection of the current understanding of the evidence in the scientific literature. Two, appropriate and relevancy of the discussion of our current understanding of the analytical issues in research for asbestos and mineral fibers. Three, the appropriateness and relevancy of the discussion of the current understanding of epidemiological issues and research needs for understanding health

effects of asbestos. Four, the appropriateness and relevancy of discussion of the discussion of the current understanding of toxicological issues and research needs in our understanding of asbestos. And fifth, the appropriateness and relevancy of the discussion of the path forward that is outlined in the document and whether the ultimate vision is a reasonable outcome for the proposed research strategy for asbestos and mineral fibers.

For those speakers who have signed up for the 15-minute timeframe, I am planning on giving you a few warnings. I am going to ask you to come up and make your presentation here and I will slip this little green card up here at the twelfth minute, I will give you the yellow card up at the thirteenth minute, and the red card at the fourteenth minute, and at the fifteenth minute I will break in and we will introduce the next speaker. So we will try to keep us on time that way.

There are copies of the document out

back at the table. If you would like, you can go out and get those. And if you have not signed in, I would ask you to do so. Are there any procedural questions from the speakers or anyone here before we begin? Given that, I would like at this time to introduce Dr. Paul Middendorf who is going to provide a brief summary of the draft document, and then we will move directly to the agenda speakers. Dr. Middendorf?

DR. MIDDENDORF: Thank you, Frank. Good morning. Over the last 40 years or so there has been considerable public-health interest in asbestos and activity in the development and recommendations and regulations to protect workers. Also during this period, the amount of published research on asbestos is among if not the most for any group of chemicals. Yet despite this interest and activity, there is still considerable disagreement on the interpretation of some of the seminal studies, and substantial uncertainty remains in key areas that prevent a fuller understanding of these important issues that could

lead to a development of more informed recommendations to protect workers. Because of the recent events such as those associated with the Libby, Montana vermiculite mine and in Eldorado Hills, California, there issues have once again been brought to the forefront and additional knowledge is needed to address them.

NIOSH has begun the process of developing this knowledge starting with the development of the document "Asbestos and Other Mineral Fibers: A Roadmap for Scientific Research." The document has been in preparation for well over a year and is the result of input from the NIOSH mineral fibers working group and substantial review from the NIOSH community. Before we get to the comment and discussion part of the meeting, I will provide just a general overview of the draft of the Roadmap.

The Roadmap is intended to describe the current understanding of the science and the uncertainties in that science associated with asbestos and other mineral fibers. It is also

intended to provide some background information on how we came to this current understanding. Going through this process, we identified what we think are the key scientific issues that have implications for the development of recommendations and identified research directions that would address these key issues. Let's start by reviewing some of the background important in developing the Roadmap, looking first at asbestos use in the United States.

Over the last 15 years or so there has been a consistent decline in asbestos mining and use of raw asbestos in the United States. I will point out that the numbers reported here are limited to the six minerals traditionally identified as asbestos. At this time there is no known domestic of raw asbestos, and the amount of raw asbestos imported from other countries is substantially reduced. What we do not know at this time though is how much asbestos has been imported in manufactured products. We also do not know how much asbestos is present in building

stock that will have to be dealt with at some point in the future. Nor can we predict the potential for exposure from construction and other activities in areas where there is naturally occurring asbestos.

We focused on asbestos-related disease. Asbestosis deaths reported on death certificates and available in NIOSH's National Occupational Respiratory Mortality Surveillance System have increased twentyfold from the 1960s to the 1990s. The number of deaths from asbestosis appears to have peaked in recent years and is expected to begin declining at some point in the future because of decreases in exposures.

Data from mesothelioma deaths are available only more recently because a separate code for mesothelioma was not previously available. The trend in mesothelioma deaths appears to still be on the rise which is not entirely unexpected because mesothelioma has a longer latency than asbestosis. Other asbestos-related diseases are not currently tracked, to

trend data are not available for them.

Through this time period of increasing deaths from asbestos exposure, there has been a large amount of activity in developing recommendations and regulations for asbestos. The Bureau of Mines which is the predecessor of MSHA began establishing exposure limits for asbestos in the 1960s. Shortly after OSHA and NIOSH were established in the early-1970s, they began developing specific recommendations and regulations for asbestos and there was a flurry of activity through the mid-1970s. Most of the activity was focused on reducing the exposure limits as more information on the health effects became available and control methods were identified. However, in the 1980s, the character of the discussion began to change. Not only were the discussed on the exposure limits, but they started to include questions about what should be covered. Recently these questions have been brought to the forefront with the events associated with the vermiculite mine in Libby,

Montana, and the debate about the nature of the minerals found in Eldorado Hills.

Early in these discussions NIOSH developed its current definition of asbestos and transmitted in testimony to OSHA in 1990. The definition includes both a policy component and an analytical component. The policy component identifies what is covered, and the analytical component specifies how it will be identified and measured. Ideally, the analytical methods would produce results that are specific for what is covered in the policy. The policy component of NIOSH's current definition states that particles should be counted when they have an aspect ratio of at least 3 to 1 and are longer than 5 micrometers when viewed under phase contrast microscopy. The PCM method is documented as NIOSH Analytical Method 7400 which provides the specifications for equipment and counting procedures to be used for analysis. In some situations such as mixed dust environments it may be necessary to use transmission electron

microscopy as a backup to the PCM method. The TEM method is documented as Method 7402 and includes procedures for converting the TEM results to PCM counts.

The last part of the policy component states that NIOSH includes particles that have the crystal structure and elemental composition of asbestos minerals. To be more specific, that statement is intended to include the minerals commonly referred to as asbestos which includes the serpentine mineral chrysotile, as well as the five amphibole minerals named actinolite asbestos, amosite, anthophyllite asbestos, chrysolite, and tremolite asbestos.

The NIOSH definition also includes cleavage fragments of the nonasbestiform analogues of the asbestos minerals as long as they meet the specified size requirements. The minerals include the sepentines antigorite and lizardite, as well as the amphibole minerals in the cummoningtonite-grunerite series, the tremolite-ferroactinolite series, and the glockothane-redakite series.

These are referred to in the Roadmap as fiber-like cleavage fragments to indicate that they have a length greater than 5 micrometers and an aspect ratio of at least 3 to 1.

NIOSH developed this definition after considering four elements. The first of these elements was the results from animal studies which indicated their carcinogenic potential depends on the particle length, diameter, and biopersistence. The specific mineral identity and origin of the mineral did not seem to be critical factors in the development of cancer and so were not considered.

The second element considered was the result of epidemiological studies. One of the problems with these studies is that the populations studied were exposed to a mixture of asbestosiform and fiber-like cleavage fragments. Other limitations include the small size of the cohort and limited information on confounders which make interpretation of these studies difficult, and determination of whether fiber-like cleavage fragments was not clear.

The third element considered was that asbestiform minerals and their nonasbestiform analogues are also co-located so that predicting the presence of asbestiform minerals within deposits is difficult and could lead to inadvertent contamination and exposure.

The fourth element considered was the limitations of the routine analytical methods used for asbestos. It is well known that neither PCM nor TEM can always distinguish between asbestiform fibers and fiber-like cleavage fragments. So after considering each of these four factors, NIOSH made the determination that despite the limitations of the epidemiological studies, the evidence provided by the other three elements was sufficient to support a prudent public-health position to include the fiver-like cleavage fragments in its definition.

Since then, the decision to include the fiber-like cleavage fragments has been criticized. The critics have argued that the human and animal toxicity studies do not definitively demonstrate

the carcinogenicity of fiber-like cleavage fragments and so they should not be included in an asbestos policy. They also argue that including the fiber-like cleavage fragments does not provide additional protection of worker health, and at the same time increases both the cost of operation and exposure to liability.

The uncertainties in the research results have also led to different federal actions. In 1992 OSHA adopted a different view than NIOSH and removed the nonasbestiform forms of the minerals actinolite, anthophyllite, and tremolite that had been included in the asbestos standard promulgated in 1986. OSHA based its determination on two factors. The first was that the uncertainties in the data combined with other data showing no carcinogenic effect do not allow them to form the needed risk assessments for occupational exposure. The second factor OSHA used to make its decision was that the rule-making record did not indicate that there were exposures to these minerals in the workplaces that OSHA

regulates. More recently in 2005, MSHA has proposed a new rule that is intended to harmonize their rule with OSHA's and would also exclude nonasbestiform anthophyllite, tremolite, and actinolite.

In contrast to MSHA and OSHA, however, when an EPA peer consultation panel was asked in 2003 about how to deal with fiber-like cleavage fragments, they indicated that they knew of little data to address the question, that in the face of having no direct evidence and knowing that dimension and durability are critical factors in pulmonary pathogenesis, their consensus opinion was that it is prudent to assume equivalent potency for cancer in the absence of other information to the contrary.

After considering the information available, it appears that additional knowledge is needed to enable us to update the NIOSH recommendations, and there seem to be three key issues related to the development of a new policy component of the NIOSH definition. The first

issue is whether other minerals should be included. There is substantial information available for investigations at Libby that could be used to support the inclusion of other amphibole minerals such as winchite and richterite in a mineral fibers recommendation. Substantial information is also available for other minerals such as aereonite that indicate that it should be included in the recommendation also. What still needs to be determined is whether there are minerals that should also be included.

The second issue is whether fiber-like cleavage fragments should be included. Various interpretations of the same research results suggest the available information does not provide a clear answer to this and that additional research is needed to provide better insight into the answer to this question.

The third key issue is whether the specified dimensions are the most appropriate. The cutoff at 5 micrometers in length was based on analytical requirements, though we have

information that potency varies with length, and it has not been demonstrated that particles less than 5 micrometers have no effect. Potency also seems to vary with particular diameter, so some additional investigations into the effect of dimensions seem appropriate.

Intertwined with the question of what to cover in a recommendation are the issue of how the minerals covered will be identified and quantified. With NIOSH's current asbestos definition, the analytical issues take on additional importance because the recommended exposure limit is based on limitations of the analytical method rather than being set at a health-protected level. Improvements in the sampling and analytical methods may allow us to develop an REL on health effects.

One of the issues that should be addressed is that the current counting rules do not restrict the counted particles to an aerodynamic diameter that is likely to reach that lungs so that some particles that are not

important in disease production can be counted. Another issue is that the PCM method can resolve particles down to about a quarter of a micrometer, but we know that fibers less than this width are important in the disease process. This would not be such an important issue if the ration of the unresolved particles were consistent between processes and workplaces, but we know that the ratio varies. We also know that PCM does not differentiate between asbestiform particles and fiber-like cleavage fragments.

Although TEM is used as a backup method for PCM, it also has limitations. The electron defraction pattern of asbestiform and nonasbestiform amphiboles are not significantly different and similar patterns can be obtained from each.

The inability to routinely differentiate between asbestiform fibers and fiber-like cleavage fragments has implications for both research and potentially practice. Methods to distinguish these forms will be necessary to clearly

understand whether there are differences in their health effects, and if there are differences, methods must be usable in practice for risk-management purposes as well.

So here we are in 2007 and there are still a number of uncertainties and issues in our understanding of both the health effects and the sampling and analytical methods which need to be addressed to allow us to move forward in developing new recommendations for asbestos and other mineral fibers. NIOSH is proposing that the best way to move forward is to develop a research agenda that will begin to address these key issues, and the intent of this research agenda should be, first, to provide the scientific information needed to craft evidence-based worker protection policies for mineral fibers. Second, that research should address the broad range of mineral fibers to which workers are exposed. And third, to refine our understanding of the characteristics of mineral fibers that are associated with their toxicity.

To achieve these broad goals, NIOSH is suggesting that three strategic goals for research should be pursued. The first is to develop improved sampling and analytical methods; the second is to develop information and knowledge on occupational exposures and health outcomes; and the third is to develop a broader understanding of the important determinants of toxicity. At this point in the process of developing the research agenda, the suggested research is largely directional in nature. We are identifying the types of research that should be undertaken, rather than taking the prescriptive approach and identifying specific research projects. The exceptions to this are where we have ongoing research projects which are described in the Roadmap.

Looking at the first of these strategic goals, the desired outcomes of research to improve sampling and analytical methods, are methods that accurately identify and quantify the particles contained in the policy. It is also important

that the sampling and analytical methods be able to clearly differentiate between particular types to enable both epidemiological and toxicological studies. At this time, the opportunities for addressing the major limitations of PCM seem to be limited. The alternative to PCM would be to rely on TEM which has come advantages but is also substantially more costly and time consuming which may not be acceptable for some work situations. Unfortunately, alternatives to these two methods has not been identified, so we have limited suggested research to improvements in the methods currently used. One of the major implications of either changing or modifying the sampling and analytical methods is that new risk assessments would be required based on exposure assessments using these new methods. With that as background, we identified five research objectives to be pursued.

The first objective is to improve the current PCM method by reducing interoperator and interlaboratory variability. A method under study

uses grids that are embedded on the filter and allow microscopists to consistently return to the same field so that differences between operators and laboratories could be identified and the causes of the differences evaluated. These procedures are currently being evaluated in collaboration with other researchers.

One of the major limitations of the current PCM method is the resolution. Optical microscopes are available that can resolve particles with smaller diameters, but they still may not resolve all of the particles of interest and further investigation of this option is needed.

The third objective for sampling and analytical research would be to develop methods that differentiate between the asbestiform fibers and fiber-like cleavage fragments. NIOSH currently has research underway to evaluate the new ASTM method for asbestos in mining, but additional research ideas for alternative methods that would differentiate between them would also

be valuable.

Because biopersistence is an important factor in the toxicity of mineral fibers, methods that incorporate an assessment of particle durability might prove to the valuable and could also improve the assessment of heterogeneous and unknown mixtures. If these methods are developed, they would need to be integrated with toxicity assessments to ensure that there is a high correlation.

The fifth objective is to address the issue of including for analysis only fibers that can reach the lung. Research is ongoing to identify and validate prefilters that meet the established thoracic size conventions.

The second strategic goal of the research agenda is to develop information and knowledge on occupational exposures and health outcomes. Information is needed to determine the numbers of workers exposed to various minerals as well as the exposure levels. This type of information is needed to identify populations for

health surveillance and possibly epidemiological research. It can also be used to prioritize toxicological and epidemiological research.

The objectives for research to accomplish this goal are threefold. The first objective is the identification of populations exposed to various mineral fibers and the subsequent collection and analysis of available exposure information, as well as the development of new exposure-related information as appropriate. The second objective is to collect and analyze available information on health outcomes and then analysis within the context of the exposures. This may be accomplished through the identification and review of available surveillance systems and registries as well as new systems and registries as appropriate. By combining the exposure and health outcome information we may be able to identify worker populations that can be included in epidemiological studies which could then be used to develop a better understanding of the

association between particle exposures and health effects, as well as the association between particle attributes and health effects.

One of the anticipated limitations of the epidemiological studies is that it will be difficult to identify populations that are exposed to specific minerals and are also exposed to particles in narrow ranges of length and diameter, so it seems that we will need to rely on toxicological studies to systematically study the effects length, diameter, and chemical composition as well as the various morphological characteristics such as asbestiform, acicular, and prismatic.

To accomplish this broad characterization of particle attributes that determine their toxicity, we envision the need for both in vitro and animal studies. The in vitro studies would be used to assess the effects of mineral particles on specific biological processes. At this time, in vitro tests are not available to study all of the biological processes

of interest, so there would be a need to develop and validate some new in vitro tests. Short-term animal tests would be needed to evaluate fiber deposition, translocation, and clearance mechanisms, as well as serve as a reference for development in validation of in vitro methods to assess biopersistence. Long-term animal studies are needed to address the impacts of dimension, morphology, and biopersistence on the chronic disease endpoints such as cancer and nonmalignant respiratory diseases. However, there is an important technological barrier to doing longer-term animal tests. Method to generate large amounts of narrow-size-range particles of naturally occurring minerals have not been identified or developed and so this is a key limiting factor to performing these tests. NIOSH is currently working on a method with a contractor to produce enough suitable material for long-term animal tests and we have had some promising results so far, as well as some disappointments.

That finishes the overview of the

proposed research agenda and NIOSH believes that the directions outlined in the research agenda would provide better information on which to base recommendations to protect workers from the elongated neuroparticles that impact their health.

In addition to what we hope to accomplish with this research agenda for asbestos and other mineral fibers, it is helpful to look at the long term and think about how this research can be used more broadly. When we do that, we suggest that it would be beneficial if we can use this research in combination with research on other elongated particles such as synthetic vitreous fibers and nanofibers to build toward a unified theory of fiber toxicity. As a starting point, the toxicity may be able to be predicted by some combination of chemistry, dimension, and biopersistence, but there may be other factors that are identified in research that should be included too.

If we can develop this unified theory, it could be used to develop evidence-based risk-

management approaches which could be implemented to protect workers from exposure to newly identified or manufactured materials. It would also be advantageous if a combination of in vitro and short-term animal tests could be identified that accurately characterize the toxicity of thoracic-sized fibers so that the resources needed to characterize and confirm their toxicity would be minimized.

Turning our thoughts back to the current proposed research agenda, we believe that the outcomes of this research are reasonably anticipated to produce new knowledge in occupational safety and health and to benefit workers' health which are outcomes directly related to NIOSH's mission. We recognize that achieving the established goals would require a significant investment of resources and that the results will have impact beyond the workplace. We are interested in leveraging our resources by developing partnerships with other federal agencies and other groups to conduct the research

needed as well as to move the research results effectively into recommendations and practice.

That is an overview of NIOSH's understanding of the issues and the directions we think research should take to enable us to develop more-informed recommendations to protect workers. We are interested in comments and input from our stakeholders so we can improve our understanding of the issues and develop a more-refined Roadmap. To that end we have identified five discussion issues about the Roadmap that we have asked for input on.

The first discussion is whether the hazard identification and discussion of health effects for asbestos and other mineral fibers are a reasonable reflection of the current understanding of the evidence in the scientific literature. The second discussion issue is the appropriateness and relevancy of the discussion of the current understanding of the analytical issues and the research needs for analysis for asbestos and asbestos and mineral fibers. The third

discussion issue is the appropriateness and relevancy of the discussion of the current understanding of the epidemiological issues and the research needs for understanding the health effects of asbestos and mineral fibers. The fourth issue is the appropriateness and relevancy of the discussion of the current understanding of the toxicological issues and the research needs for understanding the health effects of asbestos in mineral fibers. The fifth issue is the appropriateness and relevancy of the discussion of the path forward and whether the ultimate vision is of reasonable outcome for the proposed research strategy for asbestos and mineral fibers. Those are the five issues in summary, and with that I will turn it back to Frank.

MR. HEARL: Thank you, Paul. We are now ready to begin with the main agenda that was passed out in the back for the people who had presigned for 15-minute time presentations. The first person on our list is Mr. William C. Ford from the National Stone, Sand, and Gravel

Association. I ask you if you could come on up, Mr. Ford, and make your presentation. I would also ask that as you begin if you could state your name, your affiliation, and the identity of any other party or organization on whose behalf you are presenting.

MR. FORD: Good morning. Mister Chairman, members of the NIOSH Peer Review Panel and the NIOSH Mineral Fibers Work Group, ladies and gentlemen. My name is Bill Ford. I am Senior Vice President of the National Stone, Sand, and Gravel Association located in Alexandria, Virginia.

On behalf of the National Stone, Sand, and Gravel Association, our fellow stakeholders and cosponsors of three presentations which you will see this morning, the American Road and Transportation Builder's Association, the Associated Builders and Contractors, and the U.S. Chamber of Commerce, we are pleased to bring you three presentations that are relevant to the Draft Roadmap for Asbestos Research in response to the

invitation for comment from the National Institutes for Occupational Safety and Health. We appreciate very much the agency's outreach to obtain their views and our views on this very important matter before us today.

At the outset, I want to make a very important fundamental point, and the point is that asbestos is a serious human health hazard and a known human carcinogen. Harmful exposure to it must be strictly controlled. Also at the outset I want to cover some basic mineralogy to set the stage for presentations that you will see later today and provide some context for what you are going to hear. You will hear more about mineralogy from the other presenters today, but we need to lay some basic groundwork at the beginning.

I am going to be talking about two different types of minerals, asbestiform and nonasbestiform minerals. My fellow presenters, this is a very tricky button and it is very sensitive, so be careful as you use it.

This set of 12 pictures shows the difference between the two types. Those minerals in the first and the third column here and here are the six minerals which are known commercially as asbestos. Note that they have a unique physical structure. They are composed of bundles of long, slender fibers. The minerals in the second and the fourth columns are chemically identical minerals to those in the first and third columns, but they are ordinary rock. Why are they different?

As the drawing shows, the asbestiform minerals consist of fibers that grow almost exclusively in one dimension. They are easily bent and they appear as bundles of smaller fibers which are called fibrils. Asbestiform minerals are also long and thin with aspect ratios typically 20 to 1, or 100 to 1 or greater. Most asbestiform fibers are less than a micron on width and nearly all are less than a half-micron in width, and the individual fibers are visible only with the microscope.

Unlike asbestiform minerals, some ordinary rock-forming minerals grow in several directions at once. Under pressure the asbestiform minerals bend, however, the ordinary rock-forming minerals fracture easily into particles called cleavage fragments. Of those, some are needle-shaped and some show stair-step cleavage patterns. Cleavage fragments tend to be shorter and thicker than their asbestiform counterparts, and nearly all have widths that exceed a half-micron and lengths below about 10 microns. The green areas on this map show where igneous and metamophoric rock occur in the United States. These are the types of rock where asbestos may be found, not necessarily found, but it may be found.

The Draft Roadmap for Asbestos Research deals with a very serious and important subject and the risks of not getting it right are high. Failure to accurately define asbestos and disease-causing asbestiform fibers correctly and failure to develop the analytical tools to measure

asbestos and disease-causing minerals in the natural mixed-dust environment risks failure to accurately disease-causing asbestiform minerals and it risks underestimating the adverse health effects from those minerals. Getting it wrong can also cause us to misinform the public and to misdirect and misuse scarce public-health resources on problems that do not exist.

In summary, let me make several key points. Asbestos is a serious human health hazard and a known human carcinogen. Harmful exposure to it must be controlled. The six regulated commercial asbestos minerals can exist in the rare asbestiform variety, or commonly they exist in the ordinary nonasbestiform variety found in many igneous and metamorphic rocks. Studies show that the ordinary nonasbestiform rocks do not cause asbestos-like disease, and this has been studied extensively for over 30 years. Differences between the asbestiform and nonasbestiform mineral varieties are evident in their physical form but not in their chemical composition and the

challenge then becomes to differentiate between the two. This can be done through carefully and clearly drawn definitions and discriminating analytical methods.

Current analytical methods in fiber definitions for asbestos were designed for settings where commercial asbestos was produced and were not based on mineralogical characteristics nor health effects. These test procedures are not useful in the natural mixed-duty environment where asbestos is rarely present because they cannot distinguish between asbestiform and cleavage fragments that are frequently found in the outdoor environment. New test methods to measure the lower concentrations of asbestos that can occur in the natural mixed-dust environment are needed. Pure asbestos analytical standards without cleavage fragment contamination are needed to help laboratories identify and distinguish asbestos from common rock fragments.

In addition, a voluntary laboratory

accreditation program similar to the National Voluntary Laboratory Accreditation Program, the NVLAP program operated by NIST is needed to help assure local testing laboratories produce accurate results. ASTM's new consensus standard which was published last July, and that is D70-200-06 for measuring asbestos in the natural mixed-duty environment is a positive step in the right direction. Regulation and legislation addressing asbestos must have definitions and test methods based on peer-reviewed science and be both accurate and specific enough to measure regulated asbestiform minerals while excluding ordinary prismatic rock-forming minerals.

I am pleased to introduce and present to you now three experts who are going to briefly review their work in this field. Dr. Ernest McConnell has spent a lifetime designing, conducting, and interpreting animal carcinogenesis studies including several involving various types of asbestos and man-made mineral fibers. Dr. Graham Gibbs has over 40 years of experience in

fiber and health field research. And Wayne Berman, a Ph.D. physical chemist, began his career in a group that pioneered procedures for site risk-assessment under the Superfund Program. He has conducted hundreds of risk assessments for government and private clients and since 1985 he has been conducting research to investigate the characteristics of asbestos that predict risks, and he co-authored the Asbestos Risk Assessment Protocol that EPA suggested to a peer-review consultation workshop in 2003.

On behalf of our co-sponsors, thank you in advance to our three presenters that you are going to hear shortly, Dr. McConnell, Dr. Gibbs, and Dr. Berman, and thank you to NIOSH for inviting us to share our comments and research with you. We will provide additional information to the docket and copies of the presenters' papers. Thank you very much.

MR. HEARL: I would like to welcome up our second speaker, Dr. Ernest McConnell. Dr. McConnell? If you could also begin by stating

your name and affiliation.

DR. MCCONNELL: My name is Gene McConnell. I am President of ToxPath Inc., in Raleigh, North Carolina. My expertise as you heard is in the design and conduct and interpretation of rodent animal bioassays particularly the long-term ones that involve production of chronic effects such as in this case pulmonary fibrosis and cancer.

First I would like to state that these comments that I am going to make represent my own personal views and not necessarily those of the sponsor of this. Second, that this presentation is in large part from a paper that John Addison and I gave at the Taconite Conference in Minnesota in 2003.

What I am going to do is try to reiterate, some of you know this, why a fiber can be toxic. Second, animal studies that are pertinent to the subject today particularly cleavage fragments. I am going to stress that because that seems to be the new part as I

understand it of what the Roadmap is about. And finally, I am going to try to put this in the context of the Bradford Hill criteria that have been used in epidemiological studies, but I have found them very useful in studying a problem like this.

What makes a fiber pathogenic? First of all, there is no intrinsic toxic chemical in these fibers. If you would happen to dissolve them in the lung, there is no particular mineral in there that is going to make you sick or anything else. In fact, I have calculated in the past that in these animal studies that if every fiber in the animal dissolved, it would not add more than about 5 percent to the body burden of those minerals that are already in that animal. So you cannot think of this in terms of the toxicity of the material itself, you have to think of it in the physical parameters as we alluded to earlier.

What are those physical parameters? First of all, you have to remember dose, a lot of times we forget this in our studies of minerals,

and that is, if you never get exposed to something, obviously it cannot cause any disease, so dose has to be considered in any study that somebody does. My own personal view is that when you design these studies, at least one of the doses should be relevant to what humans might get exposed to to put it in the context of whether this is a true hazard or not.

The second is dimension. You heard more about that earlier. The only point I want to make here that is specific to the Roadmap and to the cleavage fragments is that if you believe in the dimension issue, then you have to look at the number of those structures that meet that critical size, that is fibers probably longer than 10 micron and less than a half-micron in diameter. If the structure does not meet that criteria, obviously it will behave more like a nuisance dust than an asbestos fiber, so you have to think of that. If you do not create those with cleavage fragments, then it's my view that they will not be very hazardous unless you get the very long ones,

and then you have to think about the number of very long ones that you could get.

Durability is not a very big problem in the minerals we are talking about today because probably except for chrysotile are equadurable and therefore biopersistent. Some people look at these two terms as the same, biopersistance and durability, and they are not, although durability does impact on biopersistence. Durability you might want to think of as how of as how fast the fiber can dissolve in a biological environment like the lung because we know that the figures to cause disease in humans as well as animals have to reside in that lung for quite a long period of time. What argues for that is that the development of the various diseases does not occur immediately after exposure but takes a long time. In other studies where you have used temporal studies where you have only exposed for short periods of time, I am talking about animals now, it is very clear that those do not produce the same amount of disease as the chronic exposure.

So there is a lot of good scientific information that shows that those materials have to reside in the lung for very long periods of time to be effective in causing disease. So you have to consider that.

Finally, one I am becoming more and more intrigued with is the surface activity. I was a little critical of the importance of surface activity initially, but I am a believer now. That is that it really does help me explain why these fibers that reside in the lung a long period of time start causing pathologic changes. I can see no other explanation for it other than they are stimulating something in that lung of a nonchemical nature because of their properties, not their chemical properties, but probably their surface properties simulate the cells to produce the cytokines which can be protective but also can hazardous or pathogenic. I think if I were putting some money into research I would push this a little bit more particularly in trying to see if cleavage fragments are different than fibers. The

information that we have to date, however, suggests they are different and therefore you would a different biological response, but some more work could be done in that area.

I am not going to go over all the animal studies because we do not have time for that. To summarize, in 2003 when we reviewed this, John Addison and myself, we tried to find every paper we could where a mineral in the asbestiform and the nonasbestiform had been given using the same sort of protocol. We found quite a few of those kinds of studies. Without exception, the asbestiform caused lung cancer and mesothelioma in rodents, while the cleavage type of the very same mineral did not with the same exposure. For me, I thought the question was settled at least for these end points that there was a true difference cleavage fragments and asbestiform minerals of the same type.

Similarly, we reviewed the in vitro studies and found the same sorts of things, although the database was not as robust as it was

with the animal studies. It may be more robust today. I have not reviewed the literature in the last 3 years in that area. That is not an area of my expertise. But when we reviewed that at that time, it appeared that there was a difference between cleavage fragments and asbestiform fibers in terms of their activity in cell cultures, and you may hear more about that today.

Let's look at the Bradford Hill criteria. As I mentioned, these are criteria that are used to evaluate epidemiological studies, but some of them, in fact most of them, I think are relevant to viewing any kind of a science problem, and let's go through those.

Strength of association. What that means is it is used in the weight of the evidence approach, is there an association between the material you are interested in and the events you see? It is very clear with the study of asbestiform fibers that these diseases are associated. So we have met that criteria.

Consistency. I think this is

particularly important in animal studies, and that is, does one study mimic another study, mimic another study, and the next study and so forth, or are there a lot of exceptions? If there is a real mix in results, that says that there may or may not be an affect. If the results are consistent from one study to another using different routes of exposure in the case of these minerals we are talking about today, then that increases your view that there is a true effect or a true no effect. In this case, I think it is very clear that with the asbestiform fibers that you do consistently get these same effects, that is, when it is inhaled or instilled in the lung, pneumoconiosis, if you will, or fibrosis, either lung cancer and/or mesothelioma. In contrast, when you use the cleavage fragments in the same studies, you consistently do not get these diseases which for me suggests that there is quite a difference.

Specificity. It is a little bit like the strength of association and is the effect specific to the cause, and it obviously is. The

temporality in the Bradford Hill is probably not applicable to experimental studies because in epidemiological one of the criteria is that you have to have the exposure prior to the disease. In animals we make sure that happens, or we should.

The biological gradient. Again, this is pretty clear with animal studies. What that is is essentially dose response, do you get an increasing effect with increasing dose, and with all of these mineral fibers you do. So it is very clear and I think it meets that criteria.

Plausibility. Plausibility is, does this make sense? That is the way I interpret it. Does it meet the I feel right about this criterion? That is, if you give this mineral, for instance, either a cleavage fragment or -- does it make sense, and these do. You get the same effects in the lung that you would expect to get in an animal and a human, and therefore for me there is strong plausibility.

Coherence is similar to plausibility but

incorporates the temporality into the equation, so does not fit animal experiences as well.

The experiment, was it designed right, was it conducted right, was it interpreted correctly, and I think that these studies, at least the ones I reviewed, while I could have tweaked them and been critical and made them a little better, I think the bottom line is that they are quite adequate to answer the question, the question being, whether cleavage fragments are different.

Analogy. We have that, too, because analogy is if you take one kind of asbestos, compare it to another kind of asbestos and to another kind, do you get the same events, and you do.

In summary, for me the weight of the evidence using the Bradford Hill paradigm strongly suggests that the pathogenic potential of cleavage fragments is clearly less than that of the asbestiform variety of the same mineral. Second, there is no evidence that cleavage fragments are

carcinogenetic in rodents, but there are asbestiform counterparts that clearly are.

With regard to the Roadmap, I would suggest that if you are going to develop some new tests that you look at that ISLI document that EPA sponsored that essentially did very similar kinds of things for manmade mineral fibers and I think it will help you a great deal because we went through a lot of work to prepare that.

Finally, I will submit some suggestions with regard to the Roadmap that you consider at your leisure. I think I am on time.

MR. HEARL: Yes, you are, sir.

DR. MCCONNELL: Thank you.

MR. HEARL: Thank you very much. Our agenda says that we go to break but we are actually a half-hour ahead of schedule. We are trying to make up some extra time so we have time to hear from people who signed up in the back who were not on the preregistered agenda. So we are going to move to the next presentation directly, and that will be Dr. Graham Gibbs from Safety

Health Environment International Consultants. Again I would ask as you begin if you could state your name, your affiliation and identify and parties that you are speaking on behalf of.

DR. GIBBS: I am Dr. Graham Gibbs. I have my own company which is Safety Health Environment International Consultants Corp., and I am also an adjunct professor at the University of Alberta, and this tells you something about my background. I was invited today by the National Stone, Sand, and Gravel Association to provide some comments. I would like to thank you for the opportunity here to do so.

My background, I've spent a fair amount of my life on asbestos and dealing with occupational cancer, occupational disease and in particular in the field of epidemiology and some occupational hygiene.

What I'd like to do is to share with you the results of a report that I prepared with Dr. Gamble. What you're going to hear will be my opinions, but also I'd like to provide a couple of

comments concerning the mesothelioma issue in Minnesota as well. Any additional comments, I understand the association is going to provide some comments to NIOSH on the roadmap, and I will provide them with some information to add into my presentation.

What we did was to look and compare the lung cancer mesothelioma experience of workers exposed to cleavage fragments with the mesothelioma and lung cancer experience of people exposed to asbestos.

To do this, we looked at where have epidemiological studies been done, and they've been done in the gold mine in South Dakota -- this is a Homestake gold mine -- in taconite mines in Minnesota and in a talc mine in St. Lawrence County in New York.

We identified asbestos exposed workers from abestiform amphibole exposed workers for any amocite asbestos mines and in manufacturing facilities. We also identified anthophyllite asbestos mines and mills and asbestiform tremolite

exposed workers in the vermiculite minutes in Montana. In my presentation, I'm going to use the term, tremolite. I think in the roadmap, they've already raised the issue that other minerals might be involved in some of these mining activities.

So let's have a look at the results for grunerite. Here, we have the non-asbestiform grunerite, and we're looking here at standardized mortality ratios. Here are the sources of the data that are provided along the bottom.

So, on the left, we have the experience for lung cancer, looking at the non-asbestiform grunerite, and you can see that basically there's less than one except for a little blip here in the Homestake mine and in terms of mesothelioma, really nothing.

We did include a mine hematite study where there was no amphibole involved at all. We chose this because we were mining iron, and here we have iron-rich rock. Again, the picture was the same.

When we come to the actual production of

asbestos and manufacture asbestos products, you can see that we have increased risk of lung cancer, very clear, quite high SMRs, and mesotheliomas are evident.

When we took a look at the data from Steenland and Brown, we could see for lung cancer that really there is not much of a dose response relationship within the range of exposures that we were able to estimate for these workers. On the other hand, pneumoconiosis was extremely steep. We think this is in part, of course, due to the fact they were exposed also to silicate in the mine. Almost certainly, this is silicate-related pneumoconiosis and not, in our view, at least my view, the non-asbestiform grunerite pneumoconiosis.

On the other hand, for lung cancer in the insulation workers, reported by Seidman, there's a very clear exposure response relationship. So the asbestos shows extremely clear exposure response even down in this region, whereas the non-asbestiform minerals did not.

Let's look at the results now for the tremolite and anthophyllite. If we take a look at the paper by Honda, looking at the exposure response for lung cancer, they found in fact that the risk for increasing exposure in the New York talc miners actually decreased with increasing exposure. That's not what you expect when in fact you have a relationship between an agent's exposure and risk.

If we take a look, on the other hand, at fibrosis which is in that same mining activity, it clearly increases with increasing exposure. So the fibrosis increases, but the carcinogenic risk does not seem to be there.

If we now look at the situation with tremolite which, of course, is one of the minerals, and non-asbestiform tremolite is reported to occur in the talc mine together with non-asbestiform anthophyllite as well. If we look at the Libby situation where workers are exposed to tremolite, there's a very clear exposure response with the tremolite. There is an exposure

response related to pneumoconiosis and some slope associated with the mesothelioma risk in that industry.

Now, if we look again at the question of talc but now in mines where in fact there are no amphiboles present, we took a look at France and Austrian talc workers to look and see, did they show with talc, in the absence of any amphiboles at all, any increase in risk. In fact, the lung cancer risk in these workers clearly is there are no increased risks. On the other hand, they did see an increase in pneumoconiosis which rather suggests that maybe the talc is related to the pneumoconiosis. But certainly the non-abestiform amphiboles are not increasing the risk of lung cancer in the other situation, and the risk is about the same as in the non-amphibole containing mining activities.

If we now take a look at the amphibole fragments from Vermont, the curious thing, which has not been explained and is still one of those questions that probably has to be tackled, is why

in Vermont they show -- sorry, in the New York talc area -- show an increased risk of lung cancer. The lung cancer does not increase with increasing fiber exposure, I mean as defined by NIOSH. It decreases. So why do they have an increased risk of lung cancer?

Now you might ask the same thing for the Vermont activities, where in fact they have a talc which does not contain amphiboles, but in fact they have an elevated risk. For some reason, whether the smoking explains it all, we really don't know.

In terms of talc without amphiboles, this is the situation there. Again, if we look at the anthophyllites from Finland, we look at the tremolite from Libby, the situation is high risk of lung cancer and mesotheliomas.

For those who like to work with numbers, what I've done here is to sort of total the picture, add up the various study numbers to see what the overall SMRs might be. You can see here that for the grunerite, the total population adds

up to over 9,000 people of whom about 20 percent are dead. It's very similar.

Here, we've got 12,000 for the non-asbestiform grunerite. The population, again a slightly higher percentage are dead. So we're not comparing apples and oranges. They're about the same point in time.

But when we look at mesothelioma here, we see 1.2 percent but here, none. Now there were some mesotheliomas mentioned in the reports, but they all gave good reasons why in fact they were not counted or they were excluded for inclusion. The final report, which will be made available to NIOSH, does include the details of these as well.

In terms of SMR, the SMR was almost three for the asbestiform grunerite -- that's the amocite exposed workers -- whereas for the non-asbestiform grunerite, it was less than one. These are quite reasonable numbers, so this is a pattern which seems to be holding on.

Something we attempted to do was to take a look at the exposure response I showed you

earlier on one of the pictures, and the numbers look like this. What we did was to use some data which had been, I think, based on measurements made by NIOSH at one point to convert a million particles to fibrous per cc, and we applied this. We can argue about whether these are the right numbers or not, but this is what was published and what was available.

When we applied that to look at this dose response, we found that the risk for the non-asbestos grunerite did not really have any dose response relationship, but even within that lower range of exposure for the abestiform fiber, clearly an increase in SMRs. Of course, this went out to quite a high risk out at that end, but we had no exposures to the non-asbestiform grunerite at those levels.

Now about Minnesota, recently, we've seen headlines in newspapers concerning an excess mesothelioma in the northern part of Minnesota, something like twice the average level for the rest of the state. I suggested some years ago,

and I think one of the things that really does need to be done is a well conducted epidemiological study of mesothelioma with appropriate controls and I think tissue analyses to find out what are these people actually exposed to and what are the controls.

I suspect that what the state is currently saying, that any mesotheliomas are probably related to other exposures from work involving commercial asbestos fibers, is probably correct. I think that needs to be examined.

Two other thoughts I'd like to throw in: The thoracic fraction, I think we need to be cautious about jumping to whole new methods. We already have problems with conversion in epidemiology. In fact, even though we've known for more than 20 years that if we count fibers, we're counting fewer crocidolite fibers than we are amocite fibers and yet none of the standards are taking that into account. So we're going to develop new methods. Whether or not they're going to be applied becomes an important issue.

I hear a few suggestions perhaps on ways that we might look at in terms of distinguishing cleavage fragments. It seems to me that maybe magnetic alignment of fibers might be something worth looking at to see whether or not cleavage fragments behave the same way as other fibers. Of course, new nanotechnology experience will surface.

One overall general comment and suggestion I'd like to offer as I close is that I think a meeting like this provides such a superficial look at such a complex issue, that I think that really what would be beneficial for NIOSH would be to have a number of workshops or think tanks on very specific topics, where you bring together people who really have spent a lot of their time doing this in the past, so you don't reinvent the wheel.

Secondly, I think it's important to recognize at the same time what has changed is that nowadays the levels of exposures are so low that some of the things we would like to have done

and could have done in the past aren't there, and the technology has also changed which maybe permits us to do some of these things we would loved to have done 20 years ago which now we may be able to.

Thank you very much.

MR. HEARL: Thank you, Dr. Gibbs. Our next speaker, and I think this is going to be the last speaker before we take a short break, will be Dr. Wayne Berman, and he is from Aeolus, Incorporated.

Again, I would ask as you begin if you could state your name and your affiliation and who you're representing here.

DR. BERMAN: Again, I'm Wayne Berman, and I'm President of my own corporation, Aeolus, Inc. I'm representing myself here today, providing my own comments, although I was invited by the National Stone, Sand and Gravel Association to come here.

One of my areas of expertise is in risk assessment and since we're all interested in

protecting public health, we therefore need to evaluate risks and then apply them to areas where we're concerned so that we can predict risk and therefore develop appropriate risk management procedures. I thought I would provide some practical ideas that I got which might suggest some other ways of refocusing some of the research that's being proposed, and I got these ideas from reading through the roadmap.

I'm going to focus just very briefly on some comments on the literature review that is in the roadmap. Then I want to illustrate some potential misconceptions that I hope to make obvious and should be taken into account when designing and focusing the research efforts, and then actually make some recommendations regarding future research.

With regard to the literature review, I would like to suggest that the literature is much richer and broader than certainly the list of citations in the roadmap suggest. One of the things that I plan to do is to provide a list of

citations and a set of written comments with an additional set, well, a much larger set of studies that should be considered.

Just as one example, in the review of the epidemiological literature, the roadmap, from what I could see, basically talks about studies from three environments when there are close to 30 environments that are relevant and should be considered. In fact, some of those other ones, Graham Gibbs has talked about today.

The next thing I want to talk about now is I want to talk about some potential misconceptions. I think it's important to recognize that arbitrarily including a greater range of structure sizes and types and counts to determine exposure concentrations is not automatically health-protective, and I hope to illustrate that shortly.

I also want to suggest that efficient evaluation of the effects of structure, size and type does not necessarily require creation of samples containing pure sizes or types, in other

words, samples that are in narrow ranges of structures, sizes and shapes.

I also want to suggest that animal and cell culture studies are not necessarily more informative than better characterizing the historical human exposures in the existing epidemiology studies. After all, we are interested in disease among humans. If we could better understand the exposures that the various cohorts have already been studied and even some of the newer ones that are being studied, if we can better understand those exposures, we might be able to do much better risk assessment using the human data directly.

Finally, one other misconception I want to touch on is that it's important to consider that to reasonably evaluate the effects of size and type, it's difficult to do this in single exposure environments. You need to have a robust range of environments that have very varying characteristics so that you can get good statistical power for distinguishing among the

effects.

So with regard to counting everything, I put together an illustration here. Very briefly, this is kind of the paradigm for how one does risk assessment. What you do is you do a series of research studies where you track the disease. In the case of humans, you track the disease in a cohort that you follow, and you characterize the exposure. Then by looking at the relationship between the disease that you see and the exposure, you develop a series of slope factors that represent the relationship between exposure and response.

Then what you do is in your study environments, which are the environments which you're worried about, you're concerned about risk, you don't know what the disease is because you want to predict it. But what you do then is you characterize the exposure and you apply the exposure response factors that you derive from your research studies, and you predict risk.

Now what I want to show is, just to

simply this, let's suppose you have a single epidemiology study in which case the amount of disease in that study among those cohorts is fixed. If you then define the exposure in two different ways, one with a larger number of structures included and one with a smaller number of structures, obviously when you then calculate the exposure response factors or the slope factors, what will happen is, if you use the metric where there's a larger number of structures, you're dividing the same amount of disease among a greater number of structures. So what will happen is the slope factor will be much shallower. So you'll be predicting that each individual fiber is much less potent than if you look at a metric in which you include a smaller number of structures.

Now if you then go out and apply those different slope factors to studies where you want to predict disease, what will happen is, depending on the ratio of the various metrics, in some cases, because you're predicting a shallower

slope, you can potentially underestimate the risk in at least some of those environments. That's why I think it's important to understand that arbitrarily counting larger numbers of structures will not automatically be heath-protective.

Really, the best way to be health-protective is to best understand what the actual biologically active set of structures are, to develop the actual slope factor that's corresponding to that and then applying it to the environments that you want to predict risk.

Let me just illustrate in another way more generally how this works, specifically with regard to the phase contrast microscopy metric which is the metric that NIOSH currently uses.

This is just a graph that represents the kinds of structures that might appear in a dust. Along the x-axis are the lengths of the structure; along the y-axis are the widths of the structures. This line here represents a 3:1 aspect ratio which is the minimum length to width aspect ratio that's currently in the definition of a fiber that's used

by NIOSH.

A couple of other lines here that are important: These two blue lines, based on my understanding from the literature and from speaking with geologists, represents the range of structures, ranges of sizes that are typically found among cleavage fragments. In fact, they tend to straddle this line. You get fewer and fewer of them as you go this way. In other words, most of them have very low aspect ratios.

In contrast, this green line here, between this green line here and the x-axis represents where most of the structures occur that are true asbestiform structures. In fact, in this case, most of them hug this x-axis. There are fewer and fewer of them as you head up this way.

What you see in the crosshatch here, this is the set of fibers that are actually counted by the PCM metric. One very important thing is you see that it misses these thinnest and longest asbestiform structures which many in the literature suggest are in fact the most potent.

Also, this red line here represents the limitive respirability, and you can see that the PCM metric in fact includes a lot of structures that are not respirable. This is probably one of the reasons that in the 1995 study of the animal inhalation data, that I collaborated with and published, showed that the PCM metric in fact showed a statistically significant lack of fit to the animal inhalation data. So it was not a good predictor of risk, at least in those data.

So you can choose other metrics, and here's another example of a metric. This actually is a metric that at the moment is proposed in the protocol that I co-authored, which is in the bluish area here. You can see it's long and thin structures that it focuses on, and it captures most of the asbestos structures, captures fewer cleavage fragments, but that's coincidental. We weren't looking to distinguish that.

What we were looking for was to try and improve the ability to predict risk. In fact, in the protocol document that we developed, we did

show that this metric in fact does better predict risk among the existing studies than the PCM metric. When the appropriate data become available, I believe that this metric can even be further optimized, but at the moment it does apparently do a better job at predicting risk certainly than the existing PCM metric.

Now the next thing I want to talk about briefly is I want to try and illustrate why it may not be necessary to spend a lot of time trying to create samples that contain pure sizes and types. It's a lot of math, but let me just point this out.

Let's suppose for the moment this is a very highly stylized and simplified, believe it or not, representation. Let's suppose you have a series of animal studies, and let's suppose you have five of them, for example. In these animal studies, each of the animals are dosed with a different type of material that has a range of sizes and types in it. Let's suppose you break those down into four categories of sizes and

types.

So here you would see the Xs would be the concentration of each of those categories in each of the studies. The A would be the relative potency of those concentrations for each of the studies. The B is the average potency overall. This would be for a linear model, for example. The Q would be a term that represents background incidents of tumors. Then P would be the actual observed tumors.

So what you see is you have five unknowns -- that would be the four As and the B -- and you have five equations. Obviously, if you solve this simultaneously, you get an exact solution where you can determine each of the As and the B, value for B. So without having pure sizes and types, you can solve these equations, and you can get information about what the effects are of these different categories.

If you do manage to produce pure fiber sizes and types, it's the same set of simultaneous equations. All that happens is that you simplify

the math somewhat by removing. Because some of the Xs become zeros in each of the categories, you remove some of the other terms, but it doesn't really simplify. It doesn't really improve your ability to extract the information.

In fact, there are reasons why this may make things more complicated because, first of all, if it turns out you guess wrong and you're producing the wrong range of structures that are important, you have to go and conduct a new study and produce more material to go back and check that. In contrast, if you have the mixed environments, since usually each individual fiber was characterized for its length and width, all you have to do is redo the calculation to change and test different hypotheses about size and shape.

Moreover, by looking at these things, you can't possibly pick up things, potentially. For example, if there are potential interactions between different sizes and types, you can't pick those up in these kinds of studies which you would

in the studies where you have the mixed exposures.

Lastly, the last point is you also can't test for continuous effects. If there's a continuous variation, it's very difficult to try and extract that from these kinds of studies than if you look at the mixed exposures. So really what you want to do is you want to look at a range of very robust studies with very different characteristics. They don't have to be pure.

Since I'm almost out of time, let me just go through this one real quickly. While you can't reasonably evaluate effects of fiber size and type from a single environment because the occurrence of the varying sizes and types categories tend to be highly correlated and also confounded in single environments. That's because most of th4e material is from a single source.

It's also important to recognize that negative environments are equally important to consider as positive environments. They're just as informative.

You can only reasonably evaluate type

and size effects by comparing across environments where you have a very rich and robust variation in the characteristics across environments. It's also important to recognize that you can only extrapolate to environments where the characteristics are similar to the range of those you've studied.

In summary, I suggest some refocusing of research efforts. I do suggest that we emphasize strongly the epidemiological studies and to improve the characterization of the historical exposures in those studies.

I also suggest that because of its versatility, that we use TEM for research while developing less expensive alternatives to support routine analysis under new regulations. By the way, to reduce the cost of TEM, we can consider automating TEM analysis, and I suggest we deemphasize the quest to produce pure samples.

We also need to recognize that the adequacy of the PCM metric and the need to distinguish asbestiform fibers and cleavage

fragments may be confounded.

Thank you.

MR. HEARL: Thank you, Dr. Berman. We have reached the time where we're going to take our first break. I have a couple of questions and a couple of announcements to make before we do leave. First, I want to ask, is Dr. Lee in the room?

Yes, Dr. Lee, we're going to get right to you after the break and before lunch. So I hope you will be ready to do that.

Also, I understand that Mr. Kelly Bailey is not going to be making presentation. He is on the agenda, but he said he would not be making presentation as well as Dr. Castleman indicated by email to us that he would not be making presentation.

Diane Miller in our office, who was taking the registrations for making presentations, advised me yesterday that she had inadvertently missed Gary Fore who is going to be making a presentation right after we come back from the

break. He's with the National Asphalt Pavers Association. It was just an oversight. So he will substitute in as our next speaker and then we'll follow with Dr. Lee and Dr. Strohmier.

At this time, I'd like for us to take a 15-minute break, and we'll start promptly after 15 minutes.

Again, restrooms, if you go out here and all the way down to the left, you'll find a set of restrooms that way. If you go to the right, all the way down and take a right at the end of the hallway, the last door on the right is unmarked, but believe me there are a set of restrooms in there.

Thank you.

(Recess)

MR. FORE: Good morning, Mr. Chairman. I am Vice President of Health and Safety for the National Asphalt Pavement Association.

Today, I am appearing on behalf of our more than 1,100 members. NAPA is an association --

It is estimated that there are at least 300,000 workers employed in the paving operations associated with hot mix asphalt operations, excluding the mineral aggregate supply industry in the U.S.

Our comments today will be brief as they are strategically directed at answering your Questions 2 through 5 regarding the appropriateness and relevance of research needs identified in the roadmap. In doing so, we will also emphasize the importance of the proposed research to our workers in the hot mix asphalt industry and the affiliated mineral aggregates.

First, we applaud NIOSH for your efforts to create a roadmap for scientific research relating to asbestos mineral fiber and other mineral fibers including naturally occurring minerals. Any time there are questions relating to workers' health and safety, it becomes a serious matter and, make no mistake about it, we think it is such.

Your efforts are also important to the

vitality of our industry for the following reasons: Approximately 94 percent of the more 2.3 million miles of paved roads in the U.S. are paved with asphalt. Naturally occurring mineral aggregates make up approximately 95 percent of this hot mix asphalt. High quality mineral aggregates needed for highway and street construction are today in short supply in various regions of the country. The transportation infrastructure in the U.S. depends on the steady supply of these naturally occurring mineral aggregates.

Not surprising, many of our member asphalt companies are general contractors and integrated companies that are engaged in the process of highway and street construction including mineral aggregate production, earth-moving, bridge-building as well as hot mix asphalt operations. Most important, thousands of workers are involved in the hot mix asphalt and affiliated aggregate industries. Worker health and safety are in their minds and plans.

Also, from an environmental perspective, our member companies and their employees are an integral part of the various communities and environments across the U.S.

There is a parallel between the asbestiform mineral situation and the asphalt fume situation involving health uncertainties and unanswered questions. For many years, NAPA and our member companies have worked in partnership with NIOSH, the Labor International Union of North America, the International Union of Operating Engineers, the Federal Highway Administration, OSHA and others in the proactive pursuit of worker health and safety. This partnership stands on a track record of accomplishment and success. Examples include engineering controls on paving machines to minimize worker exposure to fumes, the Alliance for Roadway Work Zone Safety to reduce fatalities and injuries in work zones, and the current silica asphalt milling machine partnership to evaluate and deal with potential exposure surrounding asphalt milling operations.

As I look around the room, I see numerous familiar faces that are the foundation of these highly productive government-industry-labor partnership efforts. We believe this kind of forum involving key stakeholders represents a model for the pursuit of worker health and safety research needs.

We have thoroughly reviewed the NIOSH proposed scientific research roadmap. As you have identified, the roadmap represents a significant, significant research undertaking in terms of scope. Our specific comments are strategic in nature and are consistent with the roadmap. We are quick to add that we will leave the Question 1 discussions relating to hazard identification and current understanding of the science to those more qualified.

The intent of NAPA's comments is to help focus the priorities and the scope of proposed research from the perspective of the hot mix asphalt industry:

Number one, the fibers of concern need

to be defined based upon sound evidence-based and health effects science in relation to the chemical and physical chemistry properties.

Second, there needs to be developed practical, reliable sampling and analytical methods to measure asbestos, that is, the fibers of concern in a mix, naturally occurring mineral environment.

And, third, any legislative or regulatory recommendations developed from such research activities should be based upon an understanding of the specific exposure situations along with a cognizance of the best, most current and evidence-based science available.

Thank you for this opportunity to provide our views to NIOSH on this important research undertaking. We will be pleased to assist as the research further develops. Thank you.

MR. HEARL: Thank you, Gary. The next presentation on our schedule is by Dr. Richard Lee from the RJ Lee Group, and Dr. Lee has indicated

to me that he and Dr. Brian Strohmier actually have kind of a tag team thing going on. So they have each signed up for their 15 minutes, and we will now hear from Dr. Richard Lee.

Dr. Lee, if you would, as with the others, state your name and affiliation and whom you are representing.

DR. LEE: My name is Rich Lee. I'm the R.J. in RJ Lee Group. Dr. Strohmier will talk when he gets back. If not, I'll just keep talking.

First of all, I want to also compliment NIOSH on hosting and defining and putting out in a manner of public debate the issues relating to mineral science.

My comments are going to be primarily driven at the analytical world. I think on the front end, everything you've heard and everything you will hear is a question of do you have reliable data. There's an old adage, garbage in equals garbage out, and I think the analytical method by which you determine, regardless of what

standards you set, is critical.

For those of your that aren't familiar with RJ Lee Group, we've been involved in asbestos for a long time. You'll hear us talk about SEM today, scanning electron microscopy. Just to make sure you understand we're balanced. We have about a dozen TEMs which primarily involve asbestos analysis. So when we start talking about something else, it's because we don't have a particular preference for that methodology.

We have been involved in a lot of method development. In the process of doing that, we've looked at materials from around the world. We're a certified laboratory which means we look at life from the perspective of what is the result, what is the analysis you're doing, and what is the certification you're making.

I think, from a laboratory perspective, regardless of where you go, the current ambiguity between NIOSH, OSHA and EPA, sometimes looking both ways, sometimes towards NIOSH and sometime towards OSHA, raises the largest problem at the

laboratory. The laboratories really should be, for legal purposes, certifying that what they're measuring and reporting is asbestos or such regulated mineral. There's nothing to prevent on a contract basis collecting information about any other species, but when we sign the bottom line for a laboratory director, you're certifying that you measured asbestos. Asbestos is defined in regulation, and the method is simply specifying the size and shape of the asbestos to be counted.

That is a major uncertainty, and it's raising havoc in the laboratory world as more and more labs go from analyzing blanks relating to asbestos clearing samples to analyzing samples out of mixed mineral environments.

In the real world, laboratories count anything and everything as asbestos, and there's no consistency. The reason for that is that current laboratory methods, by and large, are inadequate for mixed mineral evaluations. The current methods, many of which we helped to write, really are drafted to examine the presumption,

like PCM. PCM was originally developed with a presumptive that fibers were predominantly asbestos. Then we brought 7400, 7402 along because we realized as we lowered the concentration, the air related to interferences becomes more significant.

The same kind of issue is true in the TEM world. What people forget is that the commercial asbestos that you see in a building product or in the air related from a disturbance of commercial asbestos has had most of the non-fibrous minerals removed, but it started out as a mixed mineral. It did not occur in an isolated environment.

Those methods, by and large, don't meet the needs of NIOSH, the various stakeholders, in general. I think this review is overdue.

To give you an idea of how old I am, I got involved in asbestos at the Reserve Mining case, and very little has really changed except, as pointed out by Dr. Berman and a couple of other people, technology has changed. Our ability to

measure and characterize minerals is dramatically different than it was 20 years ago. But when you look at these cases that have come up, raised public concern and generated debate, the same questions are being raised today.

What NIOSH does in making their next generation recommendation and in setting the standards, recommending the type of standard is critically important, but it's not going to just affect the environment, the occupational environment. It spills out because those methods picked up and arbitrarily used in the analysis of samples from playgrounds, and so it has significance far beyond the occupational environment.

We really have a need for a coherent policy, and that policy should come from the top level in all the agencies. Currently, there's a huge lack of uniformity. The typical thing that happens is a laboratory will make an analysis, report asbestos. It will get in the newspaper. The next thing you know, somebody has got to go in

there and analyze samples, spend a lot of money. Often, that's me, and I like that. But it generates issues for the producer of minerals, for the school district if that data is not reliable and accurate.

What happens when we use current analytical procedures is we not just use them but relax them when we go into the mixed mineral environment. This was mentioned this morning. About a year or two ago, I forget what, a contract lab for EPA reported elevated presence of asbestos in playgrounds. We conducted a paper review, which subsequently was followed up with actual analytical work from soils and minerals. Based on the mineralogy, we said at least 63 percent of these could not be asbestos, these particles that were reported as asbestos.

About a year later, USGS came along, did another extensive review and found that 40 percent of the particles were not even a regulated mineral but, worse than that, they were a home blend which I don't think anybody really seriously ever put an

idea that it's a potential health hazard. Moreover, the majority of the particles were prismatic, not fibrous.

What was the implication of that? They spent a lot of money in El Dorado, and they still haven't got, there's not a consensus emerged on how this situation will be resolved because of this lack of definition. It doesn't matter what the definition is, but it can't be non-uniform.

There's an even more serious one from my perspective since that's my grandson in the picture, and that was that a few years ago there was asbestos reported in talc. After we analyzed it, after Datachem analyzed it, after the OSHA laboratory analyzed it, and after RTI analyzed it, the consensus that emerged was that there was little, if any, asbestos in the talc. Meanwhile, my grandson is sitting there thinking crayons just aren't what they used to be because the manufacturer had to pull the talc out of the crayon, so we get crappy crayons.

The real significance, what drove that

was this statement right here. The Seattle Post-Intelligencer: Fiber has a length of 22 microns and a width of 3.4 microns. This length to width ratio of 6.4 to 1 means that, according to EPA protocol, it must be counted as asbestos.

The typical asbestos fibril is.1 micron. If that were asbestos, you should be seeing hair sticking out of the top of that fiber. When you don't see it, it's not asbestos. It's that simple with that dimension of a particle.

You notice Dr. Fisher is not taking responsibility when saying this is asbestos. He's saying according to EPA protocol. He's taking no ownership of the science.

When you go back to El Dorado, what both USGS and we found is that most of the particles in the El Dorado soil and in the El Dorado air samples have in fact well developed cleavage faces that simply cannot exist in an asbestos fiber.

When you do your TEM work, this is a scanning electron microscope picture. The difference is TEM will be black and white, dark

and light. The SEM pictures look gray. With SEM, you look at the surface of the particles. But when you combine the SEM and TEM, which Dr. Strohmier will tell you about, you can really come to understand both the crystal structure and morphology in a manner that is unique.

Now, let's turn to the analytical issues. Why is this important? Well, you listened this morning. I think what you hear is that depending on your view of the science, everybody pretty much agrees that the hazardous material, most hazardous material is long, thin fibers. There may be what is at issue is should other things be counted and to what extent and how do you do a risk assessment.

As Dr. Berman pointed out, unless you have a rich data set, and by rich, he means informative. He means that classify things differently. Even if you get it wrong, it will show up in the uncertainty that you've built into the data. But a certain number of asbestos fibers and a certain number of cleavage means you can

obviously distinguish on a particle by particle basis. A certain number, you may not be able to.

What we need to do is design the next generation analytical methods to comprehend the most toxic minerals in the most least expensive manner we can and then take that data, design these methods so we also capture information about other potentially hazardous materials in the most effective manner.

This paper is actually is one of my favorites because it goes back. It's a paper from Littman. It has data in there from Timbril, looks at the comparison, the actual long deposition compared to fiber, really tells us where the most toxic is.

This is another one. This is another one. Okay, we've all discovered this.

So from an analytical position, the issue is not short and/or fat fibers. Depending on what you think, they may be innocuous and may not or very long. What is the real issue, where the debate centers is on intermediate, somewhere

between 5 and 10 microns long and.25 microns and a micron wide. Once they get above a micron, a blind man can tell whether they're cleavage fragment or an asbestos fiber.

What we would propose is that there is a way to optimize the measurement process, and that measurement process can be optimized by using the extension of the ASTM method, 7200 which classifies what I call Categories 2 and 3 fibers into two groups, still preserves your fundamental PCM number and then use SEM first, supplemented by TEM for the long, thin fibers. Analytically, there's a lot of reasons to do that.

The idea, what Brian will tell you is the idea that the SEM is not adequate is simply no longer the case. There are technology changes that have made it very adequate.

Basically, this is the end for me. I need to have a little bit of thought. This, I think, highlights the real issue with fiber counting. When you relax the rule from saying substantially parallel sides to simply a 3:1

aspect ratio, all the methods are written with substantially parallel sides.

In the TEM on the left, you get this particle. It's 3:1.

When you look at it in a scanning microscope, you see it's a sheet silicate. It's not a fiber simply because you're using a projection image.

Brian will pick it up from here, and thank you.

MR. HEARL: Thank you very much. Now, we will have Dr. Brian Strohmier. Dr. Strohmier, I would ask also and I note that this may be repetitive but if you could state your name and organization.

DR. STROHMIER: Yes, hello. Good morning. It's a pleasure to be here. I'm Brian Strohmier. I'm with RJ Lee Group in Monroeville, Pennsylvania.

Sad to say, I have 27 years experience using x-ray beam, ion beam and electron beam techniques to study the surface and microscopic

analysis of various materials. I wish I was younger, but that's the way it is.

I'm going to follow up where Rich started off. He had the dubious task of trying to summarize the last 30 years of debates that have been going on in 15 minutes where all I have to do is talk about some pretty pictures. What I'm going to do is try and give you a feel for some of the really exciting we've been doing with scanning electron microscopy.

The NIOSH white paper, one of the main themes is to develop new and improved techniques, cost effective techniques to take over where PCM may be lacking. They do mention electron microscopy in the white paper, and they also say that electron microscopy may not be cost effective, that it may be too time consuming, that it may have some other shortcomings which I disagree with.

As I go through this today, I'm going to make four main points in this presentation. I'm probably going to make several others, but there

are four main points I want you to carry away today and I will make each of those plain as I go through this.

But he just showed you one fiber here or one particle here that was not a fiber, and what we have done in the last year and a half at RJ Lee Group is we've characterized over 10,000 particles using this SEM technique that we've developed in-house.

Point one that I want to make is that SEM has the visibility and resolution is adequate -- in fact, it's more than adequate -- to see fibers and cleavage fragment. On the left here, we have a TEM image which is a projection of the particles in the field of view, and you see one long obvious fiber here obviously displaying curvature, parallel sides, very long and thin, high aspect ratio. Here's a bundle with straight ends.

Now, here at lower magnification is the SEM image of that same area in the box plus a larger area showing one of the advantages of SEM

is that you can go to a lower magnification and see a larger field of view, get a very good picture of what type of particles are in your sample very quickly. You can see the long stringy fiber is actually much longer than it looks like in the TEM image which is done at higher magnification. We can see the bundle. We can also see prismatic and asbestiform particles.

One of the advantages of the SEM, especially what we're using, is a field emission SEM. High current density in the electron beam, small spot size, gives high contrast, high signal to noise in the image. So it makes things stand out. You get the contrast you need to look at and see things very quickly.

Here's just a lower magnification image. In this case, it was done at 350x, and you can see all these fibers. This is on a TEM sample grid. These are the copper bar grids that TEM would not be able to see through. So TEM would only be able to see in these squares which are about 90 micrometers by 90 micrometers.

You can see by these circles. They don't stand out quite as good in this image, but every one of these elliptical dotted patterns is encircling a cleavage fragment and/or fiber, that you can see is crossing the grid bars. So if you looked at TEM, you would only see a little bit of that fiber right in the corner. This one, you'd see part. These others, you would be missing things in the TEM but pick them up in the SEM.

So this is point two, that the SEM is optimum for long, thin fibers. It's the logical extension of PCM which would miss the very thin fibers and also the logical complement to TEM that would miss the very long fibers that cross grid bars.

Now, we developed this in-house protocol which I'm only going to touch on very briefly. We start with TEM, and here's a TEM image of a particle that is crossing a grid bar which is right here.

So you see this fragment. It probably would not be counted as a fiber because it doesn't

have parallel sides. It looks chunky. It is a more than 3:1 aspect ratio, but it probably would not be counted by someone who was really strictly following the rules. There is a little tiny hint of a fiber there, but it's less than five microns in length.

We use the TEM to look at the chemistry and the electron defraction pattern to get the chemistry and crystallography of the particles.

Now, here is the FESEM image of that same particle, and you can see it actually extends out onto the grid bar quite a ways. So what we do with the FESEM to complement the TEM is we take full fiber images. We also look at each end, and we take images along the surface at a much higher magnification, which I'm not showing here, to look at the surface topography and just make sure that is this a cleavage fragment or is it asbestos.

You can see in this case what would not be called asbestos here actually is asbestiform. This is a sample from Libby, Montana, and this was identified as a winchite particle. You can see

what is a fiber here and a fiber up at the other end that will be totally invisible to TEM, and that's actually pulling out of this chunk.

I'm sorry it's not quite as visible in this as when you see it on the real screen on the SEM, but this is actually a bundle of fibers, and you can start to see a hint of the separation of the fibers right there. I just wanted to make a point that here's a case where someone wouldn't count something that is asbestos or asbestiform and it is.

Here's a case where we have a particle completely traversing the grid bar that would be totally hidden from TEM, and it's almost 200 micrometers in length. This was actually a true fiber. You even can't see it here because it's so thin, but believe me, when you zoom up on it, you can see it. That's another advantage of the SEM, that you can zoom in, zoom around, check all over your sample very quickly. So it is not as time consuming as people might think.

Here's another case that you can see

this one. Here, we have a fiber that is right on the TEM grid bar. This would be the spots that TEM could see. You see some obvious big, chunky cleavage fragments, but here is a long, thin fiber, about 35 micrometers in length, less than.25 microns in width. Here's the actual end over here and the end at this end. So that is less than 150, maybe between 100 and 150 microns in diameter, a true asbestos fiber that would be totally hidden from TEM.

If you think that in a typical mixed mineral environment, the asbestiform content may only be a percent or two of all the other rock fragments and cleavage fragments, and if you're missing one out of a hundred, you're going to underestimate the true risk that might be there by just using TEM.

Now, point three I'll get into on the next one. Well, I'll get into point three on this one.

Point three is that the SEM will give you improved morphological characterization of the

particles compared to TEM. TEM, you're just seeing a projection. As Rich said, you'll see black and gray. You're not going to see any surface features.

You don't see too much on this one, but what I want to point out with this one is here's the TEM image of a single chrysotile fiber, and there's the SEM image. You can see the cross section here because the fiber is actually sticking out of the screen at you, down at a slight angle, but it's sticking out. You can't see that in the TEM. The assumption is that everything is laying flat.

But, in the SEM, what I didn't mention is that we look at the whole fiber, ends, surface, full fiber. We also take stereo projection images which give us the orientation out of the plane of the sample. So we can tell if the fiber is a big, chunky block like the one he showed of the sheet silicate. You couldn't really see it there because that sheet was actually projecting way out from the screen. You would need to be wearing

stereo glasses, and we would have to provide the stereo projection of that image to see that.

I have about 25 booklets that will be out on the table at lunch time with glasses that you can take with you. We'll also put them on our web site. You can email us. Give me your business card, I'll give you mine, and we'll provide free glasses to you to look at the stereo images because they're very impressive. Not only do they give you the orientation, but they also allow you to look at particle association, what's bound to these particles. Now, this one doesn't have anything bound to it, but I just wanted to point that one out.

Now, I'm going to go through the next ones very quickly so I can stay within the time limit.

Here's a TEM image of what would most likely be counted as a fiber under strict counting rules, greater than 3:1 aspect ratio, substantially parallel sides. There's a little bit of an end problem there, but most people

following the counting rules would say fiber.

But when we do the SEM, you see it's a very rough surface. You can't see it on this end shot, but on the high magnification end shot, it's a perfect euhedral single crystal. So this would clearly not be asbestiform but a cleavage fragment.

Same with this one, here's a particle, little blockier, little chunkier. People may not call that a fiber because the sides aren't completely parallel. There's some debris obviously around it.

But what the FESEM shows you immediately is this is clearly not asbestiform. You see the clear cleavage planes and crystal faces on that particle.

Now, this is one you've seen before. Rich showed this one with the crystal faces actually drawn on it. Again, someone probably would not count that as a fiber. The sides aren't completely parallel. There's some chunky pieces missing there.

But, again, it's much easier to see in the SEM. You see it immediately there that that's not asbestiform, but a cleavage fragment.

Now, here's one that's a little trickier. This one, greater than 3:1 aspect ratio in the TEM projection, pretty parallel sides, that would probably be counted as a fiber.

But when you look at it in the SEM, again, it does not have the asbestiform habit. It's not smooth. It has crystal faces. It has chunks broken out of it. The SEM just shows that much quicker.

Same thing with this one, we have a particle here, pretty parallel sides. There's some problems on the end. In the TEM projection, you really can't tell. Someone might call that a fiber, depending how strictly they're following the counting rules or maybe they want to find asbestos, and so they'll say, okay, it's asbestos because the customer wants to find asbestos.

But you look at it in the FESEM, and this is actually a plate-like structure. You

can't see it unless you zoom way up on it which I can't show you unfortunately. The stereo shows very clearly that that's not an asbestiform habit.

Again, this one is a little tricky. It really looks like a fiber in the TEM, greater than 3:1 aspect ratio, parallel sides, has blunt ends. A lot of people would say that is a fiber and they, a lot of times, would be right.

But in the FESEM projection, you can start to see there's growths coming out of here, thin layers of some type of material that we call wings in-house, and it's also got a rough surface and rough edges and doesn't have a true asbestiform habit.

Now, point four, the NIOSH white paper as well as other publications and studies suggest that there's a population of cleavage fragments with fiber-like dimensions. What I want to show with this is on the left, we have a TEM image of a cleavage fragment, about 2.2 microns wide. I'm not sure how long, but it has a greater than 3:1 aspect ratio and parallel sides. People may count

that as a fiber.

Now, here's a TEM image of a chrysotile standard from Canada. You can see it has the true asbestiform habit. It has splaying ends. It's a bundle, very thin, but the important thing is to look back in the background and all these other fibers, much thinner. So this is an asbestiform habit that's cleaving to smaller and smaller fibers.

This is just the SEM projection of the same two samples, the SEM projection.

The point we're trying to make here is if you look at the width here projected over here, how much different a true asbestiform habit is. Much thinner, it's a smoother surface. Here, we have a rough surface. Much greater aspect ratio, as was said earlier today, the 20:1 to 100:1 or higher. Much lower aspect ratio over here.

So our contention is that this concept that there's a significant portion of a cleavage fragment population that has the dimensions of asbestos is a complete myth.

I'll stop there since the red flag was given to me.

MR. HEARL: Thank you very much, Dr. Strohmier.

I think at this point we will move on to our next presentation, and we have enough time, I think, to get that in before the designated time we are going to take our lunch break.

Our next speaker is R.P. Nolan, and so if Dr. Nolan would come forward. He's with the Earth and Environmental Sciences Graduate School, University Center, City University of New York.

If you would just state your name and affiliation.

DR. NOLAN: You just did it, and I'm here to represent myself today.

Could I have the next slide, please? I went through the roadmap, and basically NIOSH focus on expanding the definition of asbestos and other fiber types and cleavage fragments has a long history. This goes back at least to 1970. So this started about the time I was a sophomore

in high school.

Can I have the next slide? My first introduction to it was with the Consumer Products Safety Commission when a claim was made in the New England Journal of Medicine then 2 to 4 percent tremolite asbestos was found in children's play sand. When you looked at that 2 to 4 percent, it was all blocky tremolite and within that, there was about a hundredth of a percent was fibrous but not asbestos.

So CPSC had hearings on this. They said the scientific evidence was insufficient to regulate the cleavage fragments as asbestos. This was about the same time the experimental animal studies that Dr. McConnell discussed this morning were becoming available. The chairman of the CPSC at that time, Clarence Scanlon, said to call cleavage fragments asbestos was like hollering fire in a crowded theater. So the evidence at that time was very clear about this issue.

Now, this went on to a rulemaking which came out in 1992, and NIOSH proposed a policy to

OSHA almost identical to what's found in the roadmap. When I read the roadmap and I attended the hearings that were around 1990, they were basically the same issues that were raised. OSHA decided that the non-asbestos amphibole minerals should not be regulated as asbestos on the evidence that was available at that time.

Can I have the next slide? They said there was no evidentiary basis to support having cleavage fragments having the same morphology as asbestos presented a similar hazard. So, basically, OSHA did not accept the fact that things that had the same shape but really were different materials were the same and should be regulated as asbestos.

The population of cleavage fragments can be distinguished from asbestos. You saw part of the Lee Group discuss that this morning. For most mineral deposits, you can tell the asbestiform and non-asbestiform amphiboles. This was all recognized by OSHA 15 years ago.

Can I have the next slide? OSHA

recommended that non-asbestos fibers should be defined using common mineralogical usage. OSHA does not recognize NIOSH's efforts to define asbestos. This has been a sore subject with me, this kind of policy and analytical technique.

The analytical technique is not a method to define asbestos. The analytical technique was developed to monitor asbestos in the occupational environment. It was never meant in and of itself to define what was asbestos. That had to be done at a different step, either in a geological survey or through some other pathway.

Next, NIOSH's definition of asbestos, it's a regulatory definition with both a policy and an analytical component. NIOSH and other federal agencies have no scientific basis for developing mineral definitions. Federal agencies should not be in the business of defining what minerals are anymore than they should be in the business of defining what tumor types are. It's a different discipline.

They should incorporate mineralogy which

is not in this phrase here, and they should have that as a mineralogical basis for how you define minerals. Mineralogists have a role in that.

OSHA found the following: Mineral fibers should be regulated based on using mineralogical criteria to define and rejecting NIOSH's position that similarity in morphology is acceptable criteria for inclusion in the asbestos standard.

Go to the next one. Now, I just want to hit a series of topics. The health effects in Libby, Montana are asbestos-related. The white paper by NIOSH of the Libby fires identified predominantly as winchite and rectorite as well as tremolite asbestos, quoting Meeker's paper.

Next, our analysis by transmission electron microscopy, individual fibers from the vermiculite mine were in a tremolite acting series and could be regulated as asbestos. About half the fibers were tremolite, and the other half were some kind of sodium potassium whether it's winchite or rectorite, but the morphology is

almost identical. It's just that the chemistries vary between the two populations.

Libby provides no information about cleavage fragments. The health effects with the miners in Libby were all asbestos-related health effects. They don't tell us anything about cleavage fragments.

Now, they also mention fibrous erionite. Fibrous erionite is probably the most potent fiber ever tested experimentally in animals. A.6 million fiber milliliter hour's dose in rats produced 96 percent mesotheliomas -- this is unheard of -- and in the same experiment, no lung cancers.

Crocidolite, which many consider to be the most potent fiber type ever identified, certainly the most mesotheliomagenic of the asbestos fibers, the dose was 10 times higher and it produced no mesotheliomas and 3.6 percent lung cancers.

The two fibers types had a size distribution that was almost identical. So you

have an identical size distribution. You have a 10 times higher dose of crocidolite, and the erionite produces almost 100 percent mesotheliomas. So there's something. We heard Dr. McConnell talk a little about surface properties today. There's something about these surface properties.

In the United States, we know of no human mesothelioma in the U.S. associated to fibrous erionite. Somebody may. I do not. I don't think there have been any fibrous erionite mesotheliomas outside of the central plateau of Turkey, and there's been some discussion lately whether there's some genetic co-factor to these cases.

This is another thing which I'm going to depart a little bit from what my colleague, Dr. Lee, said today. There are asbestiform fibers that have been tested in experimental animals and not shown to be dangerous. One of them is fibrous talc.

Merle Stanton implanted tumors. If you

look, these are 100 percent tumors caused by two tremolite asbestos samples. These are the number of Stanton fibers. This is the talc. It has more Stanton fibers. The talc produced no tumors. So you have two populations of fibers, almost identical number of Stanton fibers. One is 100 percent. One is zero.

The unified fiber theory, I think, doesn't exist. I think that the experimental data that's available today indicates that this is not something that's fruitful to pursue because you can see that it's far too complicated, that there aren't just that simple rubric.

If you look at the data, the subparts of it don't hang together. You get some overviews, but the talc fibers are durable and they produce no tumors in these animals. Both erionite and fibrous talc are thought to be biopersistent. Yet, one is a powerful animal carcinogen and one is not.

Society has to become used to looking at some things that are long and thin and not

immediately think cancer.

Expanding the definition of asbestos, Hodgson and Darton produced asbestos fiber type specific assessments for human mesothelioma 2000. This is a very important paper which I don't think is referenced in the white paper. Chrysotile-amocite-crocidolite increased the carcinogenicity for mesothelioma 1:100:500. So crocidolite is 500 times more dangerous than chrysotile in this model, and when you look at erionite, it has to be at least 10 times more carcinogenic.

Within respirable fiber ranges, you can have this enormous range of mesotheliomagenic potency. These three fiber types do not belong in the same standard. They should have never been in the standard. Now, we want to add more things to the asbestos standard when we have too many things in the asbestos standard to begin with.

Worldwide, amocite and crocidolite asbestos are no longer in commerce. This is not clear in the white paper. Chrysotile asbestos can be used but contrary to NIOSH's opinion about the

low cost, others may be impressed with the low health effects associated with the use of chrysotile.

Can I have the next? Dr. Rubin will talk more about this. Can I have the next slide? Go to Table 1. One of the things that I've found to be very useful over the years -- if you can rank that up a little bit -- is to look at comparative risks. Heavy cigarette smoking, about 9,000 cancers per 100,000. Let's take U.S. motor vehicle accidents, about 1,200; pedestrian deaths, about 100 per 100,000. These are lifetime risks.

Persons living in a brick building, about 70. One continental flight per year, accidents and cosmic rays are about the same. Fifteen is the upper limit that NIOSH or EPA claims to regulate.

Five per 100,000 from a falling meteorite, now no one has been killed by a falling meteorite, but we know that meteorites strike the Earth every so often, and a meteoritic accident could be catastrophic. So, though it's a very low

probability event, if it occurs, it will kill a lot of people so this number is higher than people would anticipate.

This is struck by a falling airplane part. This is smoking three cigarettes.

This is.25 per 100,000 is taking the Hodgson-Darton model. Taking the chrysotile value, assuming that of the 2.5 million people that die in the United States each year, 5 percent of them are exposed 25 years at.1 fiber per millimeter. So they have 2.5 fiber millimeter years exposure to chrysotile. If you plug the numbers in, and they don't smoke, you get 5 deaths in 2.5 million people. It turns out to be about.25 per 100,000.

Now, we also did a risk assessment for an iron ore mine in Michigan several years ago which we published in PNAS in 1999, which doesn't appear in the roadmap, where we actually mine through a seam of grunerite asbestos in an iron ore mine. We did air sampling. We did risk evaluation for the period of time that the people

were going to be exposed, and we found that that risk was about.05 per 100,000.

So every little bit of asbestos in a mine is not necessarily catastrophe, and all of these things can be managed. We have to look at them in slightly different ways.

That's all I have to tell you today. Thank you.

MR. HEARL: Thank you, Dr. Nolan. We are at the time where I'd like to call us off for the lunch break, and I want to make a couple of comments before we do that. I want to thank the presenters for the morning session.

Any comments that anyone has that they would like to put on the official record in addition to what's been heard today or in response to that, you're welcome to do so. We have an open docket. You can send it by mail to NIOSH Mail Stop C-34 at the Robert A. Taft Laboratory, 4676 Columbia Parkway, Cincinnati, Ohio, 45226.

You can send it by email to our docket office or you can use the online web form. All of

the information you can get through the NIOSH main web site at www.cdc.gov/NIOSH.

The docket is open until 5:00 p.m. EDT on May 31st, 2007. Then all the material we have will be posted on the NIOSH web site. We now have a location off the main home page where you can examine dockets. This one, you should identify the material as Docket NIOSH-099. So that's information for you. You can continue to submit information on our document.

We will begin at 1:00 sharp, and we will continue with the next person on the agenda which is Dr. Langer and then followed by Dr. Rubin, Dr. Lemen, Mr. Hartley, Jonathan Ruckdeschel -- I'm sorry, I can't pronounce it well -- Robert Paul and Dr. David Egilman.

If those of you who are speaking after lunch could see Dr. John Pechetino who is right up here in the front, running the computer, you can get your presentations loaded in so we can move swiftly through the afternoon.

After the last of the presentations,

after Dr. Egilman has gone, I'll be pulling the sheet that we have at the back table where you can sign up still now if you'd like to make a presentation, and we'll divide the remaining time among those who want to speak. So that's what we're doing.

Are there any questions at this point concerning the program?

SPEAKER: Are things secure in this room if we decide to leave them here?

MR. HEARL: I can't vouch for the security of the room, a good lawyerly answer, right.

Okay, well, thank you all very much, and we'll see you all back here at 1:00 when we will resume promptly. Thank you.

(Whereupon, at 11:45 a.m., a luncheon recess was taken.)

A F T E R N O O N S E S S I O N

(1:00 p.m.)

MR. HEARL: Good afternoon and welcome back to the second half of the public meetings on comments on the NIOSH draft on Asbestos and Other Mineral Fibers: A Roadmap for Scientific Research.

We're going to continue with the program where we left off. As I said in the opening comments this morning, I'll check the signup sheet in the back, and if we have any new individuals who have signed up, they'll be able to take a period of time to speak. Then if we still have time remaining before 4:00, and actually it's starting to look like we do, then we can take some walk-up comments to the microphone as well.

I was advised that Dr. Langer is not present and will not be making comments this afternoon, and so our first presentation will be by Dr. Emanuel Rubin, M.D. from the Thomas Jefferson Medical College.

As I said before, Dr. Rubin, I would ask

that you state your name, your affiliation and identify any party or organization on whose behalf you are presenting.

DR. RUBIN: I'm Emanuel Rubin. My title today is Gonzalo E. Aponte Distinguished Professor of Pathology, Thomas Jefferson University in Philadelphia.

In terms of where I sort of come from, why I became a pathologist when men first descended from the trees to assume an upright posture. So I've been at this game for quite a while.

Just briefly, though, I've been on quite a few NIH study sections. I've been editor of probably the most important journal of experimental pathology, Laboratory Investigation, as editor in chief for 14 years and have had quite a few grants and still maintain three NIH grants. So I had a lot of experience in evaluating scientific data and papers and grant requests and things like that.

It's in that context that I want to

emphasize that the mineralogy and industrial hygiene and identification of fibers is certainly a legitimate and probably important area of study, but that simply identifying fibers is not really going to do the job. Without good controlled, carefully thought out epidemiology, it simply is not going to give you any reliable information.

I think the best example is smoking. Cancer of the lung, I believe and I think virtually everyone believes that the risk is certainly increased many fold by smoking, and yet in experimental animals it has not been possible at all to produce lung cancer by inhalation of tobacco smoke. This shows the discrepancy between experimental data and epidemiologic data. There are many other examples. But if you try to take the composition of tobacco smoke or the experimental data, you would not be able to predict that it causes cancer in humans, but it does.

Now, one of the things, for instance, that is not entirely detectable by mineralogic

analysis -- I think it's been pointed out previously -- are the surface properties of asbestos fibers. Those properties are really important because it's been shown that if you take asbestos fibers that cause mesothelioma in rats by injection into the pleural cavity, that coating those fibers simply within globulins reverses that and you can no longer produce mesothelioma.

Not only that, in some experiments, they have isolated so-called asbestos bodies from the lungs of people exposed to asbestos. These are bodies. These are asbestos fibers in the body coated with protein and iron complexes. They have injected those into rats and cannot produce mesothelioma, showing that if you change the surface properties of the fiber, probably even monomolecular coating, it will change the ability to cause tumors.

Those things cannot be determined simply by viewing the fiber, and that's why the epidemiologic studies are so important because there are genetic differences between animals and

man, exposure times, routes of administration, et cetera, et cetera. So I urge that no decisions be made on the role of any type of fiber until good epidemiologic studies have been done.

Now, in that context, the data is presented in this roadmap, much of it is based on death certificates. A lot of people don't realize how death certificates are made out. They are often made out by the practicing physician who is often a general practitioner and not acquainted certainly with asbestos-related diseases. They may be made out by an intern who saw the patient once. They may be made out sometimes by a physician who has never seen the patient. Most of these cases are not subjected to autopsies. In academic hospitals to date, only 10 to 12 percent of deaths are reviewed by autopsy, and in community hospitals it's becoming vanishingly rare.

So the same thing goes for many of these cases where the death certificate puts down an asbestos-related disease of any kind. Many of

them don't even have surgical pathology, and if they do, you don't even know whether it's right because you don't know if immunohistochemical analysis has been performed on the sections, so on and so forth. Death certificates, particularly in uncommon diseases, are notoriously unreliable and should not serve as the basis of epidemiologic studies.

Now, I notice, for instance, can we have that first slide? Yes, here is an example from the roadmap, and it's numbers of deaths from asbestosis. You see from 1968 all the way to 2002, there has been a definite increase in asbestosis. In other words, prior to regulation of the workplace, you had low asbestosis and after strict regulations were put in place, it kept increasing.

This would be then based on the idea that it has a very substantial latent period which it doesn't. It's all dose-related and particularly since chrysotile has been used, it would require extreme doses and would certainly

not have a long latent period. Once the asbestos exposure has ceased or chrysotile was substituted, it probably would not increase further. This is highly suspect and may represent differences in publicity about the asbestos and fears and things like that.

There's another figure, Figure No. 4, which is the number of malignant mesothelioma deaths. Now, what it shows is in 1999, it says there the figure shows 3,650 deaths a year from malignant mesothelioma in the United States. Then if you look at the text, it says 2,485, totally different. Then if you look at 2004 on the graph, it's about something over 4,000, but in the text it says 2,657. I mean I think you've got to get this straight. It just doesn't make sense.

In terms of asbestosis from a pathologist's point of view, it is put down on death certificates, again either as the principal cause or contributing cause. These are not even controlled for smoking which is the major cause of acquired respiratory illness. I mean there's much

more COPD, chronic obstructive pulmonary disease, than there is cancer from smoking.

Now, if you don't control for smoking and you don't even know whether there has ever been a pathologic analysis of any particular case, these are meaningless numbers.

Just in finishing, I would urge the panel and those who are interested in this subject to consider very carefully what is the accuracy of the data on which the roadmap relies and to acknowledge that good, sound and accurate epidemiologic data which accounts for confounding problems is the only way to go if you really want to establish any dangers associated with fiber types.

MR. HEARL: Thank you, Dr. Rubin. Our next presenter will be Dr. Richard Lemen. He's a consultant, retired NIOSH deputy director.

As with the others, I'd ask that you begin with your name, affiliation and any other parties or organizations on behalf of whom you are presenting.

DR. LEMEN: Hi, I'm Richard Lemen, and I've been retired several times, so I'm here today as a private citizen. I'm paying my own expenses. However, I do testify in litigation on behalf of plaintiffs in asbestos-related litigation cases.

I'm also the co-science director of an organization called The Asbestos Disease Awareness Organization. It's a voice of victims organization. It's a non-profit organization which I donate my time to. I'm also the retired acting and deputy director of NIOSH.

I'll start my comments. These are not my prepared comments, but I just have to comment on what Dr. Rubin said, that actually regulation does not cause disease, that we didn't have very good statistics before regulation went into effect. But if you look at the graph, it looks like the regulation has caused disease, but that's just my own personal observation. I just don't want you to go away and say regulation is a bad thing.

Also, I'd like to preface my comments by

saying that some of you have talked about how long you've been in this field. Anyhow, many of you talked about how long ago you started working in asbestos. When I walked through the doors of the old Bureau of Occupational Safety and Health in 1970, 37 years ago, I first met Ralph Zumwalde. So if anything I say today you don't like, just blame it on Ralph because he taught me all I know about analytical techniques.

But I'd like to start by saying a little bit of nostalgia, and that is NIOSH put out its first criteria document in 1972. That document called for a fiber concentration, two fibers per cc. This was based on the old Bureau of Occupational Health data out of England, and that was our first criteria document.

NIOSH then put out a revised criteria document in 1976 where we called for a lowering of that standard, using the phased contrast microscope to.1 fiber per cc based upon its analytical resolution and ability to measure in the workplace. At that time, NIOSH was the first

federal agency to say that we should ban the use of asbestos. This has been the policy of the institute since 1976 so far as I know. I don't think it's changed since that point in time.

But I would like to commend NIOSH on this roadmap. I think it's a very good start at addressing issues that need to be addressed in there of occupation-related issues and environmental-related issues to asbestos.

One of the things I would caution all of you, and I'm not trying to put a pitch in for NIOSH, but NIOSH doesn't have a lot of money and if you really want to see this roadmap work, they're going to have to get money. So if any of you have influence on that, I think you're going to have to get the money for NIOSH to carry this roadmap out.

What I'd like to say is that NIOSH has maintained this position as far as it's recommended standard and exposure limit of.1 using the NIOSH phase contrast microscope method, 7400. Unfortunately, that method cannot measure or see

chrysotile under the light microscope when it occurs in the fibril form and thus most of the chrysotile is not counted in an air sample using the NIOSH 7400 count scheme with a diameter resolution of about.25 microns since most individual fibrils of crocidolite and chrysotile are in the range of about.02 to.05 microns in diameter.

OSHA has recognized the disadvantages and advantages of the phase contrast microscope, and in my submission to NIOSH, in my appendices, I have given that information. I will not go into that right now.

NIOSH's new roadmap, I think, represents its continued leadership role in occupational safety and health by addressing asbestos-related issues needing clarification and further elucidating as well as addressing questions that are still unresolved. By so doing, NIOSH is fulfilling what I think is it's Congressionally mandated role under the Occupational Safety and Health Act.

NIOSH should not back away from including all respirable fibers and all respirable asbestiform fibers including cleavage fragments which appear to be in a fibrous habitat and thus fitting the asbestos definition by light microscope that are clearly respirable dust. I have given some information from papers that NIOSH has written, showing these findings about the cleavage fragments.

This should only be changed if there exists irrefutable data, both human as well as animal, showing the safety of any such fibrous material being excluded since the only difference in these entities is from the structure of the same mineral and true asbestiform habitat is the structural morphology with all other characteristics being the same.

NIOSH should develop valid methodology to sample for all size fibers including those less than 5 micron in length, now not addressed by OSHA regulatory standards. Both animal and human data support such an inclusion as can be seen by the

attachments in another appendices I'm giving to NIOSH.

NIOSH should address and refine their current surveillance of fiber-related diseases. For example, it is well known that the National Cancer Institute CRA Database underreports mesothelioma.

NIOSH should continue its respiratory disease surveillance system and should assure that other NIOSH surveillance systems become more comprehensive and inclusive, and analyses should not rely solely on proportionate mortality or morbidity analysis for determining mortality or instance data which many of the NIOSH reports have been doing to this point in time. This is true especially for rare diseases which become underreported, using this methodology, and one example of that is mesothelioma.

NIOSH should also determine how much of the background mesothelioma and other asbestos-related diseases are related to increased consumption of asbestos within any reference

populations used for control comparison and thus adjust expected rates accordingly in order to determine the true risk of asbestos-related diseases. Evidence suggests as consumption of asbestos has gone up, so have background rates of asbestos-related diseases. I've submitted another paper in my attachment discussing that.

NIOSH should review the epidemiology literature on all fibrous materials, not just those related to currently regulated asbestiform fiber types. Such research should address all respirable fiber types and all size parameters including short respirable fibers. Since biopersistence has been used as a surrogate for identifying and looking at lung burden studies as a critical factor in causation, and toxicological studies should evaluate whether external airborne concentrations are actually representative of the fiber concentrations and morphologies once the fibers have been inhaled into the lung.

Data suggests that the breathing zone samples of chrysotile may not represent the actual

fiber burden of chrysotile fibers in the lung as they break apart from fiber bundles and multiple once within the lung while the amphiboles tend to not do that. This is important as it means a higher dose of chrysotile in the lung as well as a higher rate possibly for translocation of chrysotile from the lung. Because dose plays a significant role in the toxicology of chrysotile as compared to amphiboles, such findings would be important in determining the actual role of chrysotile in asbestos-related diseases such as mesothelioma. This translation of chrysotile asbestos may indicate a more specific role for chrysotile in the etiology of mesothelioma.

Mesotheliomas develop in the pleura, peritoneum and other serosol surfaces of the body. It is universally accepted that chrysotile is a cause of cancer in the lung and migrates to and is concentrated in the pleura. Since chrysotile is carcinogenic and is present in high concentrations in the pleura where the mesothelioma is induced, it is biologically plausible that it causes or

contributes to the cause of mesothelioma.

This is also shown in many mechanistic and molecular studies that indicate how chrysotile may cause mesothelioma. Fiber penetration can rearrange cytoskeletal apparatus of the cell and thus could indicate an interaction between the chrysotile fibers and the normal mitotic process since giant, multi-nucleated cells are formed.

These studies indicate that chrysotile penetrates the cell and enters the nucleus and induces abnormal chromosomal formations in the dividing cells. Some of these abnormalities include the deletion of the P53 gene that controls cell growth.

Additional research should include evaluation of the synergistic effects between amphiboles and serpentine fiber exposures since it is highly unlikely that uncontaminated serpentine exposures exist in occupational and environmental settings. To date, such findings have suggested such a synergistic action between the mixed fiber types.

It has been suggested by some that the fibrous tremolite contamination of chrysotile, usually a very small percentage, less than 1 percent, is the cause of mesothelioma among predominantly chrysotile-exposed persons. New evaluation of the South Charleston chrysotile-exposed population of textile workers has confirmed a dose response relationship between asbestosis and lung cancer. This is important as entities suggesting that chrysotile is the safe asbestos base their conclusions only on the outcome of it causing mesothelioma. While it is generally recognized that chrysotile on a dose by dose basis is less potent than the amphiboles in producing mesothelioma, however, this does not appear the case of other asbestos-induced diseases.

Therefore, future NIOSH research should continue to look at other asbestos-induced diseases when determining recommended regulatory actions for the prevention of asbestos-related diseases.

I'll conclude by just giving you some comments about epidemiology. I have six points, very quickly.

When NIOSH does epidemiological studies or contracts out epidemiological studies: One, they should determine the actual exposure to the fibrous material and not allow dilution of any finding because non-exposed individuals or groups were included in the cohort;

Two, allow sufficient size of the study population to assure sufficient power;

Three, conduct sufficient follow-up to assure at least 95 percent of the cohort is followed up and traced and the vital status is taken into consideration;

Four, allow sufficient latency to determine if adverse effects are developing.

Five, identify and account for any possible cofounders or cofactors that may skew or alter the outcome of the study;

And, six, if case control analyses are conducted, make sure that all match controls are

selected so that the confounders or cofactors will not skew the outcome including securing adequate occupation histories to rule out other causative agents or past occupational exposures.

In closing, I'd just like to say that I think NIOSH is on the right track with putting this together. I hope that we can get the funding to NIOSH so that they can carry it out.

Thank you. The rest of my comments are submitted.

MR. HEARL: Thank you, Dr. Lemen. I want to say, since you mentioned a couple of times and I think it's probably worth my while letting everyone else know too, if you have materials, data or other supporting information that you would like to have entered into the record, you can bring those to me here today at the meeting before we close, and we'll be happy to get those on the records. Alternatively, there are the other methods of mailing and emailing it or using the web address to contribute to the docket, and those are all available to you.

Our next presenter today is Mr. Christian Hartley, Esquire from Richardson Patrick Westbrook and Brickman, LLC.

As I've told others, if you could identify your affiliation and support.

MR. HARTLEY: My name is Christian Hartley, and I am a lawyer. I represent plaintiffs, victims in asbestos litigation, but I am here on my own behalf and I am paying my own way. I am not here to represent any of my clients.

I want to talk to you briefly about some of the issues that I think NIOSH needs to consider in looking at this roadmap. I think it's interesting that many people have not really commented on the actual roadmap and kind of came to represent their own scientific issues.

The roadmap brings up several things. One of the things I want to talk about is the importance of fiber dimensions and what I have seen because I see these things misused in litigation all the time.

The NIOSH 7400 method picks a size fiber, and it's based on convenience related to the ability of the microscope to see those asbestos fibers. We've heard about that today.

Short fiber asbestos is one of the issues that's been brought up here, and it's been brought up in the Berman and Crump methodology which I think is substantially flawed.

Short fibers, there's no way to exonerate them from being causative of disease. The evidence is scant if at all. The evidence that's out there from Davis, et al. indicates -- this is animal studies -- that short, fat fibers play a role in disease. If you look at that study, I think you'll see that short fiber chrysotile cause disease in rats, and it's not surprising.

Another study, where one of the speakers who actually did not show up today, Yeager, et al., in which Dr. Langer was a participant at least, indicates that short fiber chrysotile, calidria chrysotile from California, is more

cytotoxic than other types of asbestos. I think that's something that you all need to consider.

The human evidence is also important. Dr. Suzuki's 2002 work is mentioned in the roadmap. The more recent work is also important. It indicates. There's a strong indication in there as I see it that short fiber chrysotile, very short in fact, is the predominant type of asbestos that you see in the tumor, in the target organ, the pleura. I think that's important to consider.

Also, Dr. Dodson has also mentioned. Dr. Dodson did a wonderful review of the evidence on short fibers to show just how scant it is and how it's very difficult to rule out short fibers as a source of disease.

Biopersistence is another issue that's brought up. It's brought up in the Berman and Crump methodology. It's one that's been researched recently. Of late, the research seems to be funded by companies that were involved in litigation. Union Carbide has funded some work by

Bernstein and others that's mentioned. I think it's important to ask yourself why a company, 20 years after it got out of the chrysotile business, is funding research in Europe on this when they sold short fiber chrysotile which they claim is not biopersistent.

Clearly, if you're going to look at biopersistence, let's look at the target organ and whether there's biopersistence in that organ. The issue for mesothelioma is going to be the pleura. There's no data. The only data that's out there is Suzuki, et al. and maybe some others but very little, but it shows that chrysotile is biopersistent in the pleura. I think that's a very important factor.

Some people have advocated the use of scanning electron microscopy and TEM. I think both are important. TEM is going to catch all of the fibers. We know and I know from my own work as a lawyer that experts, microscopists who are looking at tissue with scanning electron microscopy are missing fibers. They're missing

thin fibers of crocidolite. They're missing thin fibers of chrysotile. So lung burden studies, fiber burden analysis with scanning electron microscopy is not telling, and it's important to recognize that.

Obviously, another factor is whether or not chrysotile asbestos is biopersistent, we know it causes these diseases. Maybe biopersistence isn't really that important. The question is we know asbestos of all types causes mesothelioma, we know it causes lung cancer, and we know that, as the roadmap makes clear, there is no safe level that's been identified.

How important is biopersistence? Maybe it's important, but the evidence is not clear.

It's very important to recognize that when we're looking at potency estimates and the like, that the dose data out there that is available is very inaccurate. You're going to hear actually from Dr. Egilman today. I understand he's on the list. Dr. Egilman has pointed out very clearly and very succinctly how

the McGill, the Canadian chrysotile data is very inaccurate. It's based on conversions from an old midget impinger method to the current fibers per cc method, and there is no accurate conversion.

I also would point out that Hodgson and Darton who were mentioned here today, they point out specifically in their own work that their estimates of the potency are based on what they call guesstimations. If you looked at the appendix in that article, it's very clear that that sort of estimate is very inaccurate. It may be good for coming up with a general feeling as to relative potency, but it's garbage in, garbage out. If you don't know what the dose is for one, then you're not going to know what the relative potency is, and you've got to be very careful about that. We talked about that.

The same problem exists with the EPA methodology which apparently has been rejected finally by the EPA, and they're going to move to a new look at that issue. The dose data there is very, very unreliable. It's based on unreliable

dose data from studies. To come up with a relative potency and to come up with a risk estimate based on unreliable data, I say is garbage in, garbage out.

Obviously, with any meta-analysis, the author of the meta-analysis gets to put out the data. They choose what goes in, so they will choose what comes out. I think that we need to be very careful to accept any kind of methodologies where data is not available to the peer reviewers, which was the case in the Berman and Crump methodology. There were some private data that was not permitted to be released.

I think generally we just need to be watchful on that, and I'd ask NIOSH to do that. I know they are. Dr. Lemen has given me a good feeling that we can rely on the good scientists here.

It's very important, and this is another thing that happened with the proposed risk assessment methodology that was offered to EPA. The peer review process was greatly skewed. There

were several people on the peer review panel who did not disclose their industry contacts. This is a very important issue. Several of the people failed to disclose that they were working for current defendants in litigation, people like James Crapo who did not disclose his work for Union Carbide, a company that has a substantial issue in arguing that fiber shorter than 5 microns, or even 10 microns as it was in the Berman and Crump methodology, were not hazardous and had zero risk.

This sort of thing is very important in my opinion, to make sure that the folks who are reviewing this have no ties to an outcome. That was not the case, and it has been pointed out by at least one EPA commentator, Dr. Cate Jenkins, that these issues need to be addressed.

We heard a little bit about some of the data out there, about talc and some of the studies there. I think it's very interesting to hear that there is very little disease in the talc industry. There are some published studies about talc where

there are mesotheliomas. I have litigation involving talc. We are being stonewalled by R.T. Vanderbilt. They have refused to provide us worker data that we believe indicates that there are more mesotheliomas than have been revealed.

The question I guess I would ask and I would ask NIOSH to consider this is why are we being stonewalled on this? Why shouldn't the world know about the worker histories for people who are exposed to things that are being considered in this situation?

I guess I'd also point out that on today's panel there are several folks who have connections to R.T. Vanderbilt. Dr. Langer, for instance, has consulted extensively with R.T. Vanderbilt. Although he is not here today, I am going to submit some of the bills that indicate his connections with R.T. Vanderbilt. I believe several of the other people who have spoken here today have had consulting relationships with R.T. Vanderbilt, and I think that's important as we look at these things.

One of the things that seems very interesting to me is that we have R.T. Vanderbilt, which their data is very important in this because they maintain that they have tremolite cleavage fragments rather than asbestos. Back in 1975, in their own documents, they're telling their customers that they're going to warn about the asbestos hazard with their product.

If we could go to the next slide. In 1977, a competitor and a well known asbestos seller, Johns Manville, actually recognized the fallacy of R.T. Vanderbilt's new argument which is that their asbestos is no longer asbestos.

The scientist, Mr. Lamar at Johns Manville, said, "I object strongly to an earlier statement," and this is in reference to C.S. Thompson's article entitled Asbestos in Your Future.

"I object strongly to an earlier statement on page 3 regarding misinformation supplied by a competitor. Furthermore, in all of Thompson's gobbledygook regarding the mineralogy

of Vanderbilt's talc, at no point does he admit the fact that their talcs contain not only fibrous tremolite but chrysotile and anthophyllite as well. This, we have proved by every available technique. These findings are well documented in numerous R and D reports.

"I'm afraid that Dr. Thompson," and Dr. Thompson is still a representative of R.T. Vanderbilt.

"I'm afraid that Dr. Thompson long ago gave up any professional ethics he might have and is now persisting with a program that is not only technically false but, even more tragic, morally and ethically wrong. He totally ignores the medical consequences of his immorality."

I think it's very important also to consider this study was mentioned, the Honda study. The Honda study, which is purportedly indicating that there is no hazard for mesothelioma with talc, was of course supported by R.T. Vanderbilt. I bring this up because I show you the order from the Kentucky court indicating

that R.T. Vanderbilt has refused to produce its worker records and also my motion in a court in Illinois, trying to compel the same thing because they have refused to produce this information.

I think it's very important for the folks in this room, for NIOSH to get the data before we make any decisions about this. The roadmap is a good idea. They are clearing up areas for proper regulation. But we also need to make sure that all of the data is received, and that includes the secret data that they're not producing.

With that, I think also there's been some reference to the taconite studies today. I think the more recent data has indicated there were a lot more mesotheliomas in the taconite mining groups, and I think that's really important to look at as well.

Thank you. Let me just add that I am going to provide you with some of the documents that I've shown.

MR. HEARL: Thank you, Mr. Hartley. Our

next presenter is Jonathan Ruckdeschel from the Ruckdeschel Law Firm. Hopefully, I didn't get your name too far off.

MR. RUCKDESCHEL: If you have a name like Ruckdeschel, you can't get uptight about it.

MR. HEARL: If you would identify yourself.

MR. RUCKDESCHEL: I will, of course. Good afternoon. My name is John Ruckdeschel. I'm an attorney who, for the last seven years, has had the privilege of representing individuals and families suffering with the difficulties of mesothelioma.

I came today on my own accord and without compensation to raise some concerns that I have regarding not the intention of the roadmap but some of the practical issues that I see becoming difficult as a result of the way that some of the things that are in the roadmap are phrased. Specifically, the roadmap advocates the laudable scientific goal of development and perfection of a grand unification theory of fiber

toxicity. I think that that and the other scientific questions posed in the roadmap are all worthy and laudable goals.

Unfortunately, as written, the roadmap contains various statements that will inevitably and immediately be seized upon by companies that are involved in litigation relating to mesothelioma and other asbestos-related diseases as claims that they have been exonerated by NIOSH. Specifically, the roadmap suggests in at least two places that absent finalizing this grand unification theory, there may not be a sufficient scientific basis to support the current policies that have protected American workers and families for decades.

What I'm referring to here specifically is the statement that: "Achieving the goals will be well worth the investment because the occupation health protection policies that NIOSH recommends for asbestos and other mineral fibers must be based on the results of sound scientific research."

I believe that that is undoubtedly true, and I believe that they are. Current existing policies are based on sound scientific research. That that research may be incomplete is a fact of science. However, that quotation will be seized upon in litigation by the proponents and before regulatory agencies by the proponents of the use of hazardous materials as an exoneration that the current policies are in fact not supported by sound science. I understand that that is not the intention of NIOSH, and I simply appeal today to raise that as a concern.

Similarly, at various points, the roadmap as worded appears to suggest that until all so-called uncertainty gaps, as they're referred to in the roadmap, are resolved, scientific basis for the policies that protect workers and their families may be lacking. Such a model cannot be what is being advocated by NIOSH as it would be shockingly reminiscent of the tobacco industry model of scientific inquiry which would assert that the default position is

unleashing a poison upon the public unless all evidence uniformly supports the conclusion of a danger when in fact sound public policy that NIOSH and other health agencies have followed for decades and should continue to follow is that when the weight of the current scientific evidence demonstrates that there's a hazard to life and health of individuals, action should be taken to protect those individuals.

Again, I do not believe that it is the intention of the panel or of any of the authors of the roadmap to suggest such a model. That being said, my experience in the last seven years in the asbestos litigation has demonstrated to me that, as written, the report will immediately be portrayed in courts and regulatory agencies and industry-sponsored peer-reviewed papers as validating such an approach. Every time the report states that various issues are uncertain without discussing the source, often industry-manufactured dispute, the roadmap will immediately be seized upon by industry-sponsored scientists to

assert that NIOSH supports what I say.

It is critical that NIOSH recognize that the roadmap does not exist within the well meaning walls of the agency. If it did, I would have no concerns. It does not, unfortunately.

Over the past 10 years, there has been an explosion of doubt science based upon the rhetorical model of the tobacco industry. The modus operandi of the doubt scientist is to twist statements of disinterested and well meaning scientists in ordinary course of their thoughtful scientific inquiry into alleged validation of the position of the sponsoring industries.

I want to make clear that in finalizing the roadmap, NIOSH appreciate the larger context in which it exists. In that regard, I have two suggestions. The first is that the roadmap be reviewed to examine the possibility of such manipulation of accurate and well meaning statements that will be misplaced and misportrayed outside of NIOSH.

The second suggestion is that the

roadmap be kept as a draft and an evolving document. As an expressly evolving draft, the roadmap will provide direction while simultaneously recognizing the flexibility recognized in the roadmap in following such a significant scientific inquiry. At the same time, by keeping the roadmap as a draft, it will avoid many of the problems of manipulative mischaracterization that has happened so often in the past by advocates for industries who do not share the benign and laudable goals of this agency.

I want to thank all of the authors of the roadmap for the substantial piece of work that they've done and for giving me the opportunity to come here today.

MR. HEARL: Thank you. Our next present is Robert Paul from Paul Reich and Myers. As with the others, I'd ask if you can identify your affiliations.

MR. PAUL: Well, you might have trouble pronouncing Jon's last name. My problem all my

life has been people asking me, your name is Robert Paul what? That's been my cross to bear.

Anyway, my name is Robert Paul. I'm a plaintiffs' asbestos lawyer. I've been doing asbestos for 27 years. I can't stand up here and not acknowledge Mr. Zumwalde and all the things that he has done. Which one is he? Is that you?

I've read some of the papers you and Dick Lemen did and the things that this agency has done over the years to protect the folks that I represent. It hasn't been praised enough in this meeting, and I want to make sure that that's done. You guys have done a great job.

I also want to echo the comments that Jon made with respect to what are clearly the goals of what this roadmap is about, but I want to talk about some other things that I think are important. My presentation really breaks down into two conversations. One, I want to talk about the bias issues and, secondly, I want to talk about the science issues that are presented by the roadmap.

The first issue on the bias issue, one of the problems that we have as plantiffs' lawyers is defense doctors come in. Of course, we're all familiar with Dr. Powstenback's $250,000 that he told me in Indiana plus he was paid to write the paper for which he has now testified in a range of over $10 million for the car companies in terms of the brakes that Ralph and this agency investigated at Firestone in 1972, following up documentation that had been done at Firestone since 1946 on the dangers of asbestos and the millions of fibers generated by chrysotile but on the disease incidents in that particular plant.

I represent a woman who was diagnosed in February who worked at the Raybestos brake manufacturing plant in Crawfordsville, Indiana, as an inspector. She has peritoneal mesothelioma. If chrysotile doesn't cause mesothelioma, then why does my young lady have mesothelioma? That is really one of the perspectives that a lot of what we've talked about today has really missed, and that's the human context of the folks that are

dying of this disease which this agency has fought so hard to protect against all these years.

One of the things that I think this agency should consider is a requirement that any presenter must present to you before they present any papers of any kind or any statements to you purporting to be science, that the exact biases of that particular scientist be made clear. I can talk about how many times Dr. Gibbs has testified on behalf of asbestos defendants, how many times Dr. Langer has testified on behalf of asbestos defendants, how many times Dr. Rubin has testified on behalf of asbestos defendants and how many times Mr. Lee has testified on behalf of asbestos defendants, and you didn't hear any of that. Now, that's my point.

I think there needs to be a rule that the agency issues. I know issuing rules is hard, but I think there needs to be a bias description and a clear description of how much money each of these scientists have received from asbestos or commercial interests, and you can apply that to

our guys too. That's okay. I don't have any problem with that because the point is especially in these times in this town, the issue of how much money is being spent to effect science is a problem, and people don't always tell you that.

So that's my pitch about one of the things the roadmap ought to require is a bias description. I'm happy to submit. I won't do it today, but I'll submit some proposals on how that might be done.

Let me talk about the science issues a little bit too. The first issue is biopersistence, and I'll try not to cover what Jon and Christian did. But one of the issues with biopersistence is the assumption, which I disagree with in the paper, in the white paper, that merely by measuring how long chrysotile, amocite or crocidolite remains in the lung, that that is somehow the only measure for mesothelioma. Well, that's not true.

Let me give you an example. We all know that doctors diagnose people dying from gunshot

wounds every day and there's no bullet, right. Why isn't the same thing true?

An example of that is a paper that some of you are familiar with, that Dr. Frank did for his Ph.D. dissertation for this agency, where they found immediate effects upon lung tissue of rats, immediately upon exposure to asbestos. Now, why isn't it equally plausible that the fibers break up, the fibers migrate to the lungs, to the pleura, the peritoneum, the pericardium as the white paper points out, and then cause the mesothelioma and are cleared out of the body?

What is conceptually wrong based on the evidence that we have which is primarily Dr. Suzuki's work?

Now, another problem that I have with the white paper is the notion that predictive measures to determine lung cancer are somehow automatically predictive for mesothelioma. I'll leave that for Dr. Rubin. He's much more expert at this than I to explain why I'm wrong about this, which I'm sure he'll be happy to do. But it

seems to me that there is a difference and that most of the papers that we've seen that are discussed in the paper and that the agency talked about when Howard Aro was here in the fifties, talked about even then was the fibers break up; they migrate; they cause disease; they move on.

The discussions also in the paper don't really discuss the more traditional synergistic effect of cigarettes and asbestos on the causation of lung cancer. The problem of the focus on what I call the pure science exposures, that is, the pure exposures, ignore the complicated use of different products that each of our fellows have as well as the whole issue, which the agency is much more familiar with than I, about the contamination of the tremolite, about the contamination of the talc, of these other things. Are we so certain that Suzuki's and Dodson's papers on the short fibers aren't sufficient in and of itself that we don't need any more research on that subject at all?

We need to also look at the foreign

exposures because the foreign exposures are where the new exposures are happening.

I'll comment about Mr. Lee's comment about the crayons because, as some of you may be familiar with, the paper that Ford did when in the 1970s, Ford's scientific director discovered that the Girl Scouts as a project as part of the Girl Scouts for Brownies for use of asbestos. Ford, in 1972, wrote a letter saying, you know, asbestos is dangerous in this context, and the Girl Scouts should take it out of the projects for the Girl Scouts.

So I don't think this issue about crayons is as funny as he seems to think it was. At least Ford thought it was significant 30 years ago.

The issue about what I call the attempt to create doubt, what we really have here is an attempt to create scientific doubt in order to defend cases. If any of us think that that's not true, you're wrong. Obviously, that's my perspective, but I think that on analysis, it does

make sense. There's enormous advantage to creating scientific issues that this agency decided 30 years ago such as asbestos kills, that all fibers cause it, that all types of fibers cause it.

This new cleavage fracture notion that we hear, that's I guess the latest chrysotile defense. We all know there's always been enough papers to talk about chrysotile. So now it's all about cleavage fractures. Now, we talk about cleavage fragments. Now, cleavage fragments don't cause mesothelioma.

I'm going to close with this comment: If chrysotile doesn't cause meso, if short fibers don't cause meso, then why do I have 100 mesothelioma cases and why does my lady in Crawfordsville, Indiana, have peritoneal mesothelioma when all she ever did was work for 13 years as an inspector of brake linings in the Raybestos plant?

MR. HEARL: Thank you, Mr. Paul. Our last signed-up presenter, and then we will take a

break after this presentation, is Dr. David Egilman, M.D./M.P.H. from Brown University.

Dr. Egilman.

MR. BROWN: Thanks. I'm Ed Brown. They didn't pay me to come. Nobody else did, and I also am not getting paid for being here.

On the other hand, I do a lot of work consulting on asbestos issues at the request of injured workers and at the request of a variety of large asbestos and small asbestos companies. So I do both, and I was a consultant to Turner Newell for a while, which some of you may know is the largest asbestos company in the world, having divided the world into a large cartel with Johns Manville who you saw a document from before.

SPEAKER: It's a little hard to hear you.

I'm sorry. That isn't said about me very often. I'll try to fix that.

Most of the things I was going to talk about have already been mentioned, so I'll just try to do it with less technical jargon. Since I

came all the way here and I had these few slides, I want to talk anyhow. So I'll be emphasizing two main scientific points.

The first is that biopersistence is not a relevant factor in analyzing asbestos toxicity, particularly with respect mesothelioma but also with respect to lung cancer. There are a variety of studies that have been done, looking mechanistically at the induction of cancer, most of which have been done by Carl Barrett here at NIH, who doesn't do any or a lot of litigation.

By the way, in terms of bias, I don't really think that money is the sole bias. It may be a potential bias, but there are other biases as well, and historically it's harder to get at those.

So, from my perspective, I would rather see, by the way, for panels like this, a circle and talk and exchange of ideas back and forth around a circle rather than constant presentations so that we can discuss iteratively things. I think that's a better process. You could have

both. That would be my process suggestion, and then we could discuss maybe some data, and we might actually make some progress rather than talking about each other or one another. I know that's not a traditional government construct, but at any rate it is sometimes an occasionally useful construct, at least in academia.

There are two reasons biopersistence is irrelevant, or three. One is that mechanistically it looks like the injuries begin within days, weeks or months of the contact of the substance with a cell. Asbestos has been studied that way. This is an old model. I think it's from a Carl Barrett presentation. It doesn't take long, 15 to 17 changes maybe in the DNA over the course of the time. It's not a one-shot model. It's complicated. Life is complicated. Human beings are complicated. Even rats are complicated, it turns out. So a lot of this stuff is hard to figure out.

The second reason it is probably not, if you believe that crocidolite is more potent than

chrysotile, it turns out that in Finkelstein's study, which is the best one I know about biopersistence, biopersistence turns out to be a function of fiber length, not fiber type. If you're going to say there's a difference in fiber type potency, which in humans there appears to be, although in animals it looks reversed, that is, chrysotile is more potent than the amphiboles, then you have to say there must be something besides biopersistence that's the key issue because that's a function of fiber length.

The third reason it's irrelevant is all the studies on biopersistence deal with biopersistence in the lung, and of course the cancers of major concern here are in the pleura.

You're controlling this? Okay, when I push this, that means you should push yours.

The second point I wanted to make is what you can't see can kill you, and this is the problem with all the dose reconstructions and all the epidemiology, and you've heard it all day long. We have not been measuring the right stuff

for the last 50 years, okay, and it can't be fixed, the study. You can't go back, and it can't be fixed for a couple of reasons.

Nicholson did some papers in the early nineties, and it turns out that the pattern of fiber, there's not a consistent pattern of fibers. By the way, the 5 micron length was made for efficiency reasons. If you talk to Mort Corn, he'll tell you, well, we had to cut it off at five because there were too many under five, and it wasn't practical to count them.

Well, it turns out there's not a uniform distribution of fibers from every mine and every deposit and every product. So if you use a five cut-off for some chrysotile, you may be counting one out of a thousand fibers; for other chrysotile, it may be one out of a hundred; for another chrysotile, it may be one out of a million. So you can't go back and recreate exactly how many fibers people in the mines in Canada, which is the best long-term mortality data.

By the way, just a quick comment, death certificates are pretty standard for epidemiology. In this field, particularly Selikoff's studies, they went back and they actually did best evidence. They went back and looked and tried to correlate the epi data from death certificates with actual path because they could do that in New York and New Jersey. So, in some cases, you can do better than death certificates, but death certificates are pretty standards. If you're going to wait for something better than death certificates, we could have our next meeting around 2050. Hopefully, it will be a small meeting because hopefully nobody will have any mesos generated in the next 50 years, at least in this country.

Here, you have some problems. The PCM detection limit is.25, and only PCM fibers count in 7402. Chrysotile fibers are.02 to.05. So when you count chrysotile, you're actually not counting chrysotile fibers. You're counting bundles, and they bundles break up when they're in the body.

They split longitudinally. A single bundle may be hundreds of fibers, so there's another problem with calculating dose response.

Even if you figure out what the chrysotile doses is in the air, it's different when it gets in the body, and I'm sure it's different in different people with different latencies because you have more or less time to split those fibers longitudinally.

This is not lead or beryllium. It's not a molecule. It's a fiber that splits longitudinally, and you can't use other models for dose response for these, particularly chrysotile fibers but also other fibers which also split. You have to have a different kind of thought process for assessing dose, and it's much more complicated than the usual occupational hazard.

Unfortunately, we're locked into the way we've been doing things, and so when your only tool is a hammer, everything looks like a nail including this bottle. Well, it turns out it's not so easy to open that bottle with a hammer.

Sometimes you can break it. Not this one, it's plastic, but maybe the glass.

This turns out to be true, I think, for crocidolite. I think you heard as well.

You can't say short or long fibers are irrelevant because we haven't been measuring them for the last 40 or 50 years. The thin ones have not been measured. So we don't really know about this. None of the dose reconstructions and none of the meta-analyses can deal with these because the data is not there. It cannot be reconstructed.

I published on this, but I didn't publish my own work. I republished. The Canadians first looked at this in 1974 because they had to convert particles to fibers, and what they found was an inverse correlation between disease and particle counts. There's an explanation for that, at least one explanation. It turns out the higher the exposure in Canada, the less disease there was, and that was true in their epi studies.

Yes, it's true. You've got to look at the appendix, Dr. Berman, published on this.

The reason for that was that the higher exposures were actually in the miners, but most of what they were exposed to was mine dust because they were exploding the stuff. It's only 5 percent chrysotile; the rest was dust. Well, the midget impingers were measuring dust. So the higher dust levels were in the miners. They had less chrysotile exposure. The lower exposures were in the millers, where there were good controls, but a higher percentage was actually asbestos fibers.

So there was an inverse relationship, and they had to manipulate the data in those studies. They did manipulate the data, and they got away with it for a while. In other words, they threw out the inverse data until it became linear. The nonsense data, they just threw out.

This is from an industry-sponsored meeting, Archibald Cox, remember him, from Nixon days when he was the guy they hired to do this.

This is from 1993, and this is from the Health Effects Institute. They started looking at this, and they found that PCM overestimates exposures in buildings. That's because they were representing builder owners and asbestos in place people, but underestimates worker exposures. There is some science to this that's legitimate.

This is some data that you don't get. In other words, this is secret data from Union Carbide. This is from calidria, the calidria mine. What they found out since they were doing a lot of sampling is that the calidria had a lot of ultra fine material, and they figured this out and didn't tell anybody and never told the people that they were monitoring it for, that there were lots more fibers than the standard methods were finding.

But this has been known by some industries for a long time, the problem of thin fibers in the products were producing much higher. This is a 20 to 40-fold difference. That's not trivial.

Okay, so here's the key point. You've got a picture here. You're measuring it. Where that X is, that's where all the data comes from. What's asbestos in the lung? How long does it last in the lung? Unfortunately, the cancer is occurring in the pleura, and there is an inverse relationship, not no relationship, between what you find in the lung and what you find in the pleura.

Oops! What you find in the pleura is almost all chrysotile. There are two studies, Suzuki's, but the best one is Sebastian's done by and funded by the Canadian Asbestos Mining Association, okay, funded by them, and he did pleural evaluations along with Suzuki. There are only two studies like this because this is apparently too hard to do. So when you don't do the right thing, you do the easy thing. We study the lung even though that's not where the cancer occurs.

But these guys actually did the right thing. Let's look and see what's at the scene of

the crime, and it turns out that most of the time, 98 percent of the time, there's chrysotile in the pleura and it's mostly short fiber; 23 percent of the time there's amphiboles found; 21 percent of the time, it's both; 2 percent of the time, it's only amphiboles. Well, that's what you find where the cancer occurs.

I think there's a general consensus that the asbestos has to come in contact with the tissue to cause cancer.

This is my dose response curves that I just did as I was sitting here. There's some assumption here that there is a single dose response relationship. The human data would seem to indicate that that's not true. There are many cases of people who have -- and NIOSH has published this in a Greenberg paper in 1974 on this -- brief exposures to asbestos, and they've gotten meso.

Whereas, we know that the most heavily exposed populations, insulators who, by the way, are also exposed to highly toxic silicates,

tremolite, because it was heated. Those pipe coverings are heated. That's why they were there, and there were a lot of very toxic silicates produced in that. That's the reason, I think, that insulators got far more disease than other populations because of the silicate contaminant in the insulation. But that's for another day.

It looks like there are some people who are very sensitive, but 10 percent, at best, of insulators get meso, and they're really highly exposed. But some people with a day or more, they get meso, of exposure, a couple days. So you've got to think that there's more than one population of people, never mind rats.

MR. HEARL: Thank you, Dr. Egilman. We've reached the end of the pre-

Programmed set of speakers who signed up to speak at the meeting today. I went out at lunch and took a look at the sign-up sheet, and I noticed that the people who had signed up, which I first panicked because there are like 15 names on there, were all names of people who were already

on our program.

I just want to do a quick check before I let everyone off to break and see. Was there anyone who has signed up that hasn't had a chance to speak?

If not, what we'll do is we'll take a break, and then we'll come back, and we'll see if anyone wants to make remarks from the microphone, and we'll finish the meeting off. As I said before, we'll be closing this meeting out at 4:00 for sure, and if we don't have any walk-ups, we'll end it right after we come back.

Just in case some of you do decide to duck out, I want to say, to start with, thanks to Dr. Roger Rosa in the back who helped coordinate pulling this thing together, Dr. Anita Schill who is sitting up here in the front row, Dr. John Pechetino who was helping everyone with their slides, working at the computer, Retina Holmes who isn't here but who made arrangements for the hotel, David Bang and Christina Bowles who worked the registration desk and anyone else whom I may

have forgotten. I also want to thank the presenters.

Like I said, we'll take a 15-minute break now and come back and see if folks want to come to the microphone. We'll be happy to take more input from you.

(Recess)

MR. HEARL: Thank you. If you could take your seats, we'll resume and conclude the meeting today.

We have one other individual. As I looked down the list of sign-ups, there's a D. Grace signed up to make presentation.

MR. GRACE: (off mike)

MR. HEARL: Oh, because you're present, okay. In that case, I think then out of the list of people who signed up to be on-site speakers included everyone who has actually already made an on-site presentation. I think there was a little confusion about that, but that's fine.

If you haven't signed up as attending on either of these sheets, please do so, so that we

can have a record of that, and there are some blank sheets still out by the back table.

We have run through the program. We have a microphone at center floor, and at this point, I would invite if there is anyone who would like to make any further comment, provide us any kind of input here orally, we still do have some time before the meeting concludes. So is there anyone who would like to add any comment?

It is very silent. I think that pretty much says that we may have covered our course. I think what I will do is give you an idea of what we're planning to do from here.

I want to thank everyone who came and made presentations. As I said a couple of times before, I still have a folder here where I will be taking any written inputs that you would like to submit for the record. If you want to give those to me today, that will be fine.

If you want to mail it in to the docket, you can go to the NIOSH web site, and there's an address on an announcement about this meeting.

It's Docket 099. You can submit by email, you can send it mailing it in or you can send it using the web submittal form to do so.

After the docket is closed on May 31st, we will be posting all of the materials that we receive by mail, by email and here at the meeting. We'll be posting a copy of the transcript that was made at the meeting here today. So all that will be available publicly.

We also have a peer review panel that we have selected, and that panel is also listed up on our web site at present. They will be able to review the roadmap document, the comments submitted to the record, comments that have been made at the meeting here today, and they each, individually, will provide NIOSH with their review comments of the document.

After that, we will take this all into consideration and decide what final product might come from what we have as a draft that is on the web right now. In the meantime, the current draft as it exists will remain up on the web site as a

draft document.

I don't know that we have a timeframe following up for after we get the peer review comments back.

If you could come to the microphone and identify yourself.

MR. GLENN: Bob Glenn, Crowell and Maurey.

A procedural question, will the slides be a part of the docket, the presenters' slides?

MR. HEARL: I've asked Dr. Pechetino to contact each of the presenters and request permission to post copies of their slides on the docket. So, to the extent that they give us permission to do so, we will post them in the docket as pdf files.

Yes, could you come to the microphone? Identify yourself.

MS. HUTCHISON: Cherie Hutchison with the Mine Safety and Health Administration.

I wanted your exact web site location because it's difficult to find NIOSH on the web.

MR. HEARL: Our exact location is www.cdc.gov/NIOSH or you can just Google NIOSH, and it's probably the first link that will come up, actually. The docket page and the asbestos roadmap, you'll find those right on the home page, and down the center line is information about this meeting and the asbestos roadmap document.

Any other?

SPEAKER: (off mike)

MR. HEARL: Www.cdc.gov/NIOSH, that's it. Thanks.

I want to check and see if any of the panel members want to make any comments.

Dr. Mittendorf.

DR. MITTENDORF: On behalf of my co-authors and the Mineral Fibers Working Group, we just wanted to thank each of the presenters for taking their time to come and present and share their thoughts and ideas with us. This is clearly an iterative process, and we will certainly be taking into consideration each and every thing that you have provided to us.

If you have any other thoughts or ideas that you would like to share with us, the docket will be open until May 31st, and we encourage you to provide those thoughts and ideas to the docket.

Thank you very much.

MR. HEARL: Ralph, do you have any?

DR. ZUMWALDE: No. That's fine.

MR. HEARL: Okay, great. If there are no other comments from the floor, I'd like to again add my thanks to everybody for coming to the meeting. We really appreciate your taking the time to do this. We tried to do this in as open a process as possible and take maximum input from stakeholders and scientists and others interested in this topic.

Thank you very much, and I hope you have safe travels back home.

(Whereupon, at 2:40 p.m., the PROCEEDINGS were adjourned.)

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