

**EXAMINING THE HUMAN HEALTH EFFECTS OF
ASBESTOS AND THE METHODS OF MITIGATING
SUCH IMPACTS**

HEARING

BEFORE THE

**COMMITTEE ON ENVIRONMENT AND
PUBLIC WORKS**

UNITED STATES SENATE

ONE HUNDRED TENTH CONGRESS

FIRST SESSION

JUNE 12, 2007

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ONE HUNDRED TENTH CONGRESS
FIRST SESSION

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**EXAMINING THE HUMAN HEALTH EFFECTS
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GATING SUCH IMPACTS**

TUESDAY, JUNE 12, 2007

U.S. SENATE,
COMMITTEE ON ENVIRONMENT AND PUBLIC WORKS,
Washington, DC.

The committee met, pursuant to notice, at 10 a.m. in room 406, Dirksen Senate Office Building, the Hon. Barbara Boxer (chairman of the committee) presiding.

Present: Senators Boxer, Inhofe, Carper, Lautenberg, Isakson, Vitter, and Klobuchar.

Also present, Senator Murray.

**STATEMENT OF HON. BARBARA BOXER, U.S. SENATOR FROM
THE STATE OF CALIFORNIA**

Senator BOXER. The committee will come to order. We welcome Senator Patty Murray.

Senator Murray, I want to congratulate you on your leadership on this issue. I also want to say, I know Senator Isakson has been working closely with you and I want to thank him for trying to reach some agreement on your bill. This is an important hearing for millions of Americans who have been exposed to asbestos, for their families and especially for the thousands of American families who have lost family members to asbestos-related lung disease and cancer.

Millions of Americans are still being exposed to asbestos today. If we don't act, countless more people will get sick and die in the future.

Your legislation, Senator, the Ban Asbestos in America Act of 2007, would place the United States clearly on the side of protecting the health of the public from this dangerous substance. It would ban nearly all uses of asbestos in products. I am proud to be an original co-sponsor of this bill, as is Senator Baucus, who you know has had so many issues with asbestos in the town of Libby, MT.

We must take every reasonable step we can to end exposure to asbestos, when we see our fathers, mothers, sisters and brothers dying from asbestos. There is no justification for allowing the number of dead to continue to mount. Just this past year, we lost a Congressman, we lost Eli Segal, who was exposed at a very young age to asbestos. This is a deadly situation.

Asbestos fibers can be 1,200 times smaller than a human hair. These microscopic fibers can stay invisible and suspended in the air for days. People, including children, can breathe these fibers deep into their lungs, where they cause their damage. We see the results of this in communities across our country.

This nationwide, actually worldwide tragedy, has hit my State of California especially hard. According to the National Institute of Occupational Safety and Health, or NIOSH, between 1993 and 2002, more than 1,000 people died from asbestosis caused by exposures at their work. From 1999 to 2002, NIOSH reports 1,001 people died from mesothelioma, a rare cancer and deadly cancer generally caused by asbestos. These figures do not include the deaths from lung cancer and other diseases that asbestos can cause, or the deaths that the Government tracking system may have missed.

The deaths of hard-working people exposed to asbestos at their work only tell part of the story. Workers can take asbestos into their homes on their clothes. After a hard day at work, they go home and hug their children or sit with their families at the dinner table. Their spouses may handle their asbestos-laden clothes. Nobody can see the fibers, but they can still kill.

We have a picture here of Rebecca Martinez. She lived in Baldwin Park, CA. This is a picture of Rebecca Martinez. Margarito Martinez lived in Baldwin Park, CA with his wife of 39 years, Rebecca, pictured here on the right. Margarito worked as a plasterer, and Rebecca would clean his asbestos-covered clothes when he came home, breathing in the dust as she shook them out and did the laundry. They say they were never warned about the dangers of asbestos.

Rebecca was diagnosed with the deadly cancer mesothelioma in 2002. She died 4 months later.

Now we have a picture of Georgina Bryson. She lived in Riverside, CA when she died of mesothelioma. From 1962 until 1980, Georgina lived downwind from two cement companies that used asbestos to manufacture their products. She was also exposed to asbestos when she lived with her dad, who worked with gaskets that contained asbestos. She was only 40 years old when she died from mesothelioma.

I have a picture of a lung damaged by mesothelioma, just one of a number of devastating diseases caused by asbestos.

Despite all of this death, we continue to allow the importation and use of asbestos and products that contain asbestos. What is interesting about it, Senator Murray, you know this as well as I, people think we have already banned asbestos. But we continue importing it.

World production of asbestos actually increased in 2005, from 2.36 million metric tons in 2004, to 2.40 million metric tons in 2005. In the United States, we imported 2,530 metric tons of asbestos, and we imported more than 90,000 metric tons of products that may contain asbestos, products like cement and gaskets, as well as brakes and clutch parts for autos. Even the Environmental Protection Agency acknowledges that people who work on cars should be careful because of the danger of breathing in asbestos.

The good news is that there are safer alternatives to asbestos that are available today. Because of this and the continuing risks

to people's health, many nations have adopted bans on asbestos. Countries that have banned or phased out asbestos, we have a chart, I won't read it, but it is available for everyone to see, how many countries have banned asbestos. Due to the on-going dangers of using asbestos, the WHO reports that more than 40 countries have banned or are phasing out the use of asbestos. I believe the United States should squarely address the problem. That is why, again, I am so proud to be a sponsor of Senator Murray's bill, S. 742.

In scores of nations, products that used to be made with asbestos now are being made without it. I have great faith in American ingenuity, and I strongly believe that these products can be made here from safer materials as well. This hearing's focus is clear. It is on people, and the terrible price they continue to pay because asbestos is being used, despite the availability of safer alternatives. Senator Inhofe.

**STATEMENT OF HON. JAMES M. INHOFE, U.S. SENATOR FROM
THE STATE OF OKLAHOMA**

Senator INHOFE. Thank you, Madam Chairman.

The health effects of exposure to certain kinds of asbestos are well-known and tragic. As you pointed out, I don't know that there is any debate there, in fact, there is no doubt. This is why the United States has essentially eliminated the use of most dangerous forms of asbestos, and our use of other forms is severely limited to those critical areas for which there is no readily available substitute. It is also why bipartisan language to ban asbestos has been included in the bills in the last two Congresses.

It may sound simplistic, but the debate is not over true asbestos minerals and their health effects. That has been extensively studied and we now have an entire legal liability system built around it. But rather, any debate here, if there is one, has to do with the potential effects of other types of minerals. These non-asbestiform minerals have the same chemical makeup as asbestos but have entirely different physical structures, similar to coal and diamonds or water and ice.

However, our primitive analytical techniques used for indoor remediation of commercially produced asbestos falsely identifies these rocks as asbestos. In fact, the U.S. Geological Survey said that, "The counting criteria developed for analysis of asbestos in the workplace or in commercial products may not be appropriate for direct application to what is currently referred to as naturally occurring asbestos." Let me show you what I mean. Put that chart up that has the rocks.

As you can plainly see, dangerous asbestos minerals consist of fibers that are long, skinny, very flexible. That would be columns 1 and columns 3. Research has shown that these fibers are hard for the human lung to eliminate. They essentially get trapped in the lung, sometimes causing disease decades after the initial exposure.

Non-asbestiform minerals, these rocks here, that is columns 2 and 4, break up into particles called cleavage fragments, which are short, fat and bulky. Studies have shown that these cleavage fragments do not pose the same health risks as the fibrous asbestos counterparts.

We do not know if these non-asbestiform minerals have specific health risks, but yet they are regulated currently as airborne particle by the U.S. EPA, OSHA and the Mineral Safety and Health Administration, thereby protecting against occupational exposure. But what we do know is that these cleavage fragments do not cause the same diseases as asbestos. Therefore, they must be treated differently. It should be noted that the National Institute of Occupational Safety and Health has recently begun an effort to collect and analyze available data on asbestos and other materials. Other agencies are working on this, too, including the EPA, OSHA, Mining Safety and Health Administration, Agency for Toxic Substance and Disease Registry, and the U.S. Geological Survey.

The previous bipartisan language to ban asbestos recognizes these fundamental mineralogical and medical differences, and banned the true culprit. Despite the fact that this language was not debated here in the Environment and Public Works Committee, as it should have been, I have never stood in the way of the substance of that language as it represented a carefully constructed agreement providing a process for critical use exemptions and was scientifically sound with respect to the mineralogy of asbestos.

The ban language was supported by the affected industries and negotiated with Senator Murray and her staff and has held intact through two Congresses. Any legislation that comes through this committee in this Congress should do the same thing.

So I guess what we really need to do is recognize that this is a different form and treat it differently, if our investigation warrants it. Thank you, Madam Chairman.

[The prepared statement of Senator Inhofe follows:]

STATEMENT OF HON. JAMES M. INHOFE, U.S. SENATOR FROM THE
STATE OF OKLAHOMA

Thank you Madame Chair for holding this hearing today.

The health effects of exposure to certain kinds of asbestos are well known and tragic. Chest, lung and gastrointestinal cancers are horrible diseases. On that, there is very little debate. This is why the United States has essentially eliminated the use of the most dangerous forms of asbestos and our use of the other forms is severely limited to those critical uses for which there is no readily available substitute. That is also why bipartisan language to ban asbestos has been included in the bills addressing the asbestos liability situation in the last two Congresses.

It may sound simplistic but the debate is not over true asbestos minerals and their health effects. That has been extensively studied and we have an entire legal liability system built around it. But rather, any debate here, if there is one, has to do with the potential health effects of other types of minerals. These non-asbestiform minerals have the same chemical makeup as asbestos but have entirely different physical structures. Similar to coal and diamonds or water and ice.

However, our primitive, analytical techniques used for indoor remediation of commercially produced asbestos falsely identify these rocks as asbestos. In fact, the U.S. Geological Survey said that ". . .the counting criteria developed for analysis of asbestos in the workplace or in commercial products may not be appropriate for direct application to what is currently referred to as naturally occurring asbestos."

Let me show you what I mean. (SEE EXHIBIT). As you can plainly see, dangerous asbestos minerals consist of fibers that are long, skinny, and very flexible. Research has shown these fibers are hard for the human lung to eliminate. They essentially get trapped in the lungs, sometimes causing diseases decades after the initial exposure. Non-asbestiform minerals, these rocks here, break up into particles called cleavage fragments, which are short, fat and bulky. Studies have shown that these cleavage fragments do not pose the same health risk as their fibrous asbestos counterparts.

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the Mining Safety and Health Administration, thereby protecting against occupational exposure. But what we do know is that these cleavage fragments do not cause the same diseases as asbestos and therefore, they must be treated differently. It should be noted that the National Institutes of Occupational Safety and Health has recently begun an effort to collect and analyze available data on asbestos and other minerals. Other agencies are working on this too, including EPA, OSHA, Mining Safety and Health Administration, the Agency for Toxic Substances and Disease Registry, and the U.S. Geological Survey.

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I look forward to hearing from the witnesses today and to further understanding the various minerals and the differences in their health effects.

Senator BOXER. Thank you so much.
Senator Lautenberg, you have 5 minutes.

**STATEMENT OF HON. FRANK R. LAUTENBERG, U.S. SENATOR
FROM THE STATE OF NEW JERSEY**

Senator LAUTENBERG. Thank you very much, Madam Chairman.

I commend you for holding this hearing and Senator Murray in particular for her aggressive action to try to get rid of this threat in our State and our communities.

I have some degree of familiarity with problems with asbestos. When I went to high school in Patterson, New Jersey, a lot of the kids who I went to high school with worked in the asbestos factory, it was call Raybestos Manhattan. A friend mine, who spent 20 years practicing law after having been in high school, was called by a member of a union and asked if he had any x-rays of his chest in recent years. He said no, he hadn't. The fellow from the union suggested that he does that promptly because there have been signs of illness from people in that class group.

Well, the story had a terrible ending, because my friend the lawyer was dead in a year from mesothelioma. When they took an x-ray, they found out that the asbestos had started the process of spoiling his health. The Chairman, Senator Boxer, talked about, showed pictures of a family that got sick from asbestos brought home in clothing. I met a family where the father worked for Johns Manville down in central New Jersey and would bring home his clothes for laundry. He came in with his son, who is about 30 years old, and the man's wife, and all three of them had asbestosis as a result of just cleaning his clothing.

So we know the terrible toll that asbestos takes. With more than 2,000 Americans dying premature and painful deaths from exposure to asbestos. Needless to say, the consequence of this to these families is terrible, terrible, and to the people who were exposed. We have had enormous reluctance by the industry, any of the companies that we have had contact with, have fought fiercely to reduce any legislation that would impact the ability of those who work there to collect damages who worked in the asbestos factories

and would do little if they weren't pushed to deal with the problem forthrightly.

We went through a series here a few years ago where ads were run in the papers, Roll Call and the Washington papers, about stopping any legislation that would enable those who were rendered ill from having compensation. I picked up a piece of material that shows an exchange of letters in 1935, 1935, between Raybestos Manhattan and Johns Manville, alerting the companies, from a lawyer working for Johns Manville, to the concern about asbestos. In 1935, one letter says, "After discussing the hazards of asbestos, as I see it personally, we would be just as well off to say nothing about it. I think the less said about asbestos, the better we are." Once again, October 1, 1935. That is a letter from one president, from the president of Raybestos Manhattan, to a Manville attorney.

So we could continue with the exchange, but all of them suggest that they were fully aware of how dangerous asbestos was, and chose, like the tobacco companies with cigarettes way back in the 1930's, to ignore it and hope that the problem would go away.

So thank you again, Senator Boxer, and you, Senator Murray, for your persistence here. I am glad to be a co-sponsor of your legislation. I hope we can get it through.

[The prepared statement of Senator Lautenberg follows:]

STATEMENT OF HON. FRANK R. LAUTENBERG, U.S. SENATOR FROM THE
STATE OF NEW JERSEY

Madam Chairman, thank you for holding today's hearing on the health effects of asbestos. Let me welcome Senator Murray to the committee and thank her for working to keep Americans safe from asbestos.

Every year, more than two-thousand Americans die premature and painful deaths from exposure to asbestos. Their deaths leave children without parents, and families struggling to make ends meet.

New Jersey has America's sixth-highest number of deaths from asbestos. From asbestos used in ship insulation at shipyards to asbestos used to insulate pipes at refineries and factories, at least two-thousand seven-hundred and seventy-five New Jerseyans died because of asbestos exposure from 1979 to 2001. Just last week, a school in Asbury Park was closed because part of the ceiling fell and asbestos was found. This toxin's presence in offices, schools and homes could pose health risks for years to come—ranging from breathing problems to lung damage and cancer.

One of the leading researchers on the link between asbestos and lung disease was Dr. Irving Selikoff, who lived in New Jersey. Dr. Selikoff did his research on workers across my state, including those in my home town of Paterson. In 1979, Dr. Selikoff showed that one in five asbestos workers developed a fatal lung disease. Senator Murray's bill is a strategy for real action to reduce asbestos in the places we live and work.

The bill will ban the use of asbestos to the maximum extent possible and benefit companies who are producing safer alternatives. It also calls for more research on the health affects of asbestos, as well as the best treatment options for asbestos-related illnesses and better coordination among federal agencies. Congress owes our children and grandchildren action now to protect them from asbestos in the future.

I look forward to hearing the testimony of today's witnesses.

Thank you Madam Chairman.

Senator BOXER. Thank you, Senator.

Senator Isakson.

**STATEMENT OF HON. JOHNNY ISAKSON, U.S. SENATOR FROM
THE STATE OF GEORGIA**

Senator ISAKSON. Thank you, Madam Chairman. It is my privilege to serve as Ranking Member on the Occupational Safety Sub-

committee of the Health Committee, which is chaired by Senator Murray.

Over the course of, in particular the last 6 or 8 months, but over a number of years on Senator Murray's instigation, this issue has been brought forward. I want to commend her for both the intensity of her effort as well as her willingness and the willingness of her staff to work together to find common ground, which I think in large measure is about to take place. There have been a couple of issues in terms of the natural occurrence of asbestos and in terms of a reasonable transition out of asbestos and in terms of the couple of remaining uses that it has in the United States. Our staffs have talked and I have talked and have the greatest of respect for Senator Murray.

So it is my belief that it is very important that this hearing take place today as sort of the foundation, hopefully, for a common sense agreement that reflects the majority of the Congress and the majority of American people and the majority of all those, the absolute majority of all those in health care.

So I just want to commend Senator Murray, thank her for her willingness to work together. I look forward in the next few days ahead to trying to complete those negotiations to have a significant bill for this Senate to deal with very quickly.

[The prepared statement of Senator Isakson follows:]

STATEMENT OF HON. JOHNNY ISAKSON, U.S. SENATOR FROM THE STATE OF GEORGIA

Thank you Madam Chairman. I welcome Sen. Murray before the committee. I am pleased to work with her as her Ranking Member on the Subcommittee on Employment and Workplace Safety which she chairs in the HELP Committee. On March 1 of this year, we held a hearing on just this topic in the subcommittee, and I am pleased to be able to participate again in a hearing on this issue.

Of course, there is no debate that certain forms of asbestos are toxic and deadly.

Over the past 30 years, we have learned the sad truth that exposure to some airborne asbestos fibers pose potentially serious health risks. Continued exposure to airborne asbestos can increase the amount of fibers that remain in the lung. Once embedded in lung tissue, these fibers over time may cause serious lung diseases including asbestosis, lung cancer, or mesothelioma.

As we will hear today, there are several kinds of asbestos. Different forms of asbestos pose different health risks. Any ban passed by Congress must recognize these differences.

The EPA initially proposed a ban of most asbestos-containing products in the late 1970s. At the time, the U.S. consumed over 500,000 tons of asbestos, about 7 percent of which was the very toxic amphibole asbestos.

The rule was then struck down the 5th Circuit, because EPA had "failed to muster substantial evidence" in support of the ban. The Court of Appeals remanded the matter back to EPA, demanding the Agency demonstrate that all asbestos poses an "unreasonable risk" to Americans.

During the 1990s, the worldwide trade of the most hazardous form of asbestos, amphibole asbestos, ceased. Thus, this very toxic form of asbestos is no longer available to the United States. Essentially, there is a de facto ban on amphibole asbestos already in place.

Today, asbestos is still used in the United States, albeit very sparingly. According to the U.S. Geological Survey, the U.S. consumes about 2,000 tons of chrysotile asbestos yearly, down from almost 800,000 tons consumed in mid-1970s. Take note: consumption of asbestos decreased 99.75 percent without government fiat. Amphibole asbestos, the most dangerous kind, is not used. Chrysotile asbestos is used for three purposes only: roof coatings, NASA shuttle motor parts and specialized filters used in the manufacture of chlorine.

Last Congress, I was happy to support Senator Specter and Leahy's "FAIR Act." As part of that important legislation, Senators Specter and Leahy included a workable, reasonable asbestos ban that recognized the important distinctions between various kinds of asbestos.

In closing, there are many different kinds of asbestos. It comes in many different forms. There is room for bipartisan compromise on this issue, as Senators Specter and Leahy have demonstrated. I hope to work with all sides to resolve this issue. I yield my time.

Senator BOXER. Senator, I just want to thank you so much for your positive attitude, and Senator Murray, too. Sometimes an author of the bill will just say, I have done all I can, I don't want to discuss it further. But Senator Murray was very open to your common-sense thoughts on this and I am very hopeful that we will have this agreement, we can have, I am just suggesting my dream ticket, of a Murray-Isakson bill. It would really be wonderful for this committee to take up such a bill.

Senator Vitter.

STATEMENT OF HON. DAVID VITTER, U.S. SENATOR FROM THE STATE OF LOUISIANA

Senator VITTER. Thank you, Madam Chair. Thanks for this hearing and thanks also to Senator Murray for her leadership.

I want to echo what so many folks have said. There is absolute consensus and unanimity about the deadly nature of asbestos. Certainly in most industrial uses in the past, the obvious example of these uses where you had airborne asbestos, which has killed thousands upon thousands of people. I look forward to legislation that builds on that clear consensus.

I hope we focus in large part in this hearing on the more difficult issues, issues like brought up by Senator Inhofe in terms of different types of material, non-asbestos material on which there are different interpretations and rulings, even among Federal agencies.

Second I want to bring up that I hope we can focus on and come to a good resolution on, based on sound science, and that is the use of asbestos in chlor-alkali production. I am very concerned that we might ban this completely, when the science does not justify it, because the chlor-alkali industry relies on technology that safely uses asbestos diaphragms. That is really for two reasons. One is the use of asbestos there is confined in asbestos diaphragms and produced in a continuous wet environment that remains in a closed process, so there is minimal to no release of asbestos and absolutely no worker exposure. So I think again, two things are significant: wet environment and completely closed process.

Again, it is significant that this use in this production is also in accord with OSHA and EPA standards. This was specifically allowed in the final rules on this issue on asbestos from EPA in 1989.

It is important to get this right and base whatever we do on sound science, because of the significant uses of this in this country. There are 16 chlor-alkali plants operating in 9 States that rely on this technology, that is Louisiana, Alabama, Indiana, Kansas, Nevada, New York, Texas, West Virginia and Wisconsin. But it really goes well beyond that in terms of impact, because this provides critical benefits to society and the economy. Today, over 60 percent of U.S. chlorine production uses this technology. About 93 percent of pharmaceuticals sold in the United States rely on chlorine chemistry. So this has a major, major impact on society and the economy.

Now, if this were harming people or potentially killing people, that would be the end of the argument, we should outlaw it. But there is no known case of asbestos-related disease from the chlor-alkali industry using this technology. So I hope in part our discussion can focus on that, so we delve into those details as we finalize a consensus on the issue.

Thank you again, Madam Chair, for the hearing.

Senator BOXER. Thank you.

Senator Murray, we would love to hear from you for 10 minutes.

**STATEMENT OF HON. PATTY MURRAY, U.S. SENATOR FROM
THE STATE OF WASHINGTON**

Senator MURRAY. Thank you so much, Chairman Boxer, for holding this hearing and for your longstanding support of my efforts to ban asbestos in the United States. I want to thank all of our committee members who are co-sponsors, potential co-sponsors. I especially want to recognize Senator Isakson and his staff, who have worked very long and hard with us to reach a consensus, which I hope we can do fairly quickly.

I am very pleased as well to be here this morning with the distinguished group of witnesses that you have assembled for this hearing. I especially want to acknowledge the efforts of three of your witnesses: Dr. Barry Castleman, Dr. Dick Lemen and Linda Reinstein. Without their tireless work, we would not be where we are today, on the verge of finally protecting Americans from deadly asbestos.

You have called this hearing to examine the health effects of asbestos and ways to minimize its harm. I have worked now on this issue for 6 years, and I can tell you, asbestos is deadly. It is devastating to families and communities. Every day that we wait to ban it we are sentencing more Americans to an early and avoidable death. Asbestos exposure, as studies show, kills up to 10,000 Americans each year. I want to take a minute to introduce you to two of them.

This is Fred, his real name is George, but Fred Biekkola. He is from Michigan. Fred served in World War II, and for almost 30 years, he worked for a mining company in Michigan, where he was exposed to asbestos. Fred testified at my very first hearing on asbestos 6 years ago. I will never forget what he told us.

He said, "Senators, please make sure what happened to me won't happen to anyone else. Workers like me are counting on you to protect us. Please don't let us down."

Well, I am said to say that we have let Fred down. We didn't ban asbestos. We didn't warn the public. We didn't invest in research and treatment. Fred died of asbestos and mesothelioma on April 7, 2004.

Sadly, Fred is not the only friend and advocate that I have now lost over the years because Congress has failed to act. This is Brian Harvey. He is a teacher from Marysville, WA. Brian stood by my side when I introduced my very first bill to ban asbestos back in July 2002. Now, most asbestos victims die within a year of being diagnosed.

But amazingly, Brian stood with me and lived for 6 years. He knew he was living on borrowed time. So he told me he was using

his time to help fight for others. He stood by my side again in 2004 at a press conference we held here to try and ban asbestos. Sadly, I lost Brian to this fight as well in July 2005.

Well, Fred and Brian aren't with us any more. But their words and their spirit hang over this hearing. As I said, it is estimated that up to 10,000 Americans die every year from asbestos-related causes. Now, I have been at this for 6 years. This is my third bill, and I know we can't wait another year to fix this problem, because the stakes are just too high. To anyone who says, we don't need this bill, I would pose one question: how many more Americans like Fred, like Brian, like the pictures you showed, Madam Chairwoman, how many more have to die before our Government finally does the right thing and bans asbestos? We have to do the right thing and we need to do it now.

Now, as I look at this issue, four problems stand out. First, asbestos is deadly. It is so deadly that there is no known safe level of exposure. It only takes a tiny bit of fiber to cause disease.

Second, asbestos is everywhere. It is put into consumer and industrial products on purpose every day.

Now, my staff bought these brake pads in an automotive repair shop in my home State of Washington. They contain asbestos. They bought these off the shelf. It says on the sign, warning, contains asbestos. Brake pads like these are in tens of thousands of cars in this country today. Any time one of the cars with brake pads like this goes in for maintenance, a mechanic could unknowingly be exposed to deadly asbestos.

Now, Madam Chairwoman, there are alternatives. These brake pads, which we also bought here, don't contain asbestos, and they work just as good as the ones that do. We should not keep selling asbestos products and putting workers and countless consumers at risk. Madam Chairwoman, there are thousands of other products that contain asbestos today in this country, floor tiles, roofing material, cement pipes and even hair dryers.

Deadly asbestos is still putting construction and maintenance workers at risk. Today in this hearing room we have some of the workers who work in the tunnel of the Capitol Building, right below this room. They know asbestos exists, they have been exposed to it, and that is wrong. For them alone we should be doing a lot more. But the very least we can do is to ban asbestos so other workers are not put at risk as well.

Third, we know asbestos is still legal. Now, many Americans, as you alluded to, assume as I did that asbestos has already been banned in this country. But it is not. In 1989, the EPA did try to ban asbestos. But most of those regulations were overturned by a court in 1991. As a result, while new applications for asbestos were banned, asbestos is still being imported and used in consumer and industrial products that are on our shelves today.

Fourth, research and treatment for asbestos diseases is not very far along. Doctors have been hampered by a lack of funding for research on how asbestos fibers actually cause disease and what treatment strategies work best. Industrial hygienists have been hampered by lack of research on how to best measure asbestos fibers in the air.

I know that the Mesothelioma Applied Research Foundation has privately awarded over \$4 million in grants and their investment in research is helping to motivate brilliant investigators to study mesothelioma. But the foundation seed money is not enough. Federal funding is critical to the research effort if we truly are going to help people. That is why my bill requires collaboration among the 10 research and treatment centers established under the bill, along with the National Cancer Institute, the Department of Veterans Affairs and the National Institute for Occupational Safety and Health.

Because nearly one-third of mesothelioma victims were exposed to asbestos while serving in the U.S. Navy, my bill directs the Pentagon to conduct additional research on asbestos disease, early detection and treatment as well. I am also very encouraged that NIOSH has embarked on an ambitious research road map to better answer current scientific questions about appropriate occupational levels of exposure.

To address the national scourge of asbestos, I have again this year introduced the Ban Asbestos in America Act of 2007, S. 742. My bill basically does three things. First of all, it bans asbestos. It prohibits the importation, manufacture, processing and distribution of products containing asbestos. Unfortunately, some 2,500 metric tons of asbestos was used in the United States in 2005, and imports of products containing asbestos in cement pipe, tiles, brake gaskets and linings continue unabated today.

Second, my bill dramatically expands research and treatment and creates a \$50 million, 10-center Asbestos-Related Disease Research and Treatment Network. It creates a new National Asbestos-Related Disease Registry. And it supports research at the Department of Defense and launches a study to determine the most promising areas for new research.

Third, my bill launches a very important public education campaign to better inform all Americans of the dangers of exposures to asbestos in the workplace and in the environment while also providing helpful steps so all of us can better protect our families.

I know we can and we should be making progress in banning asbestos. As you stated, Madam Chairwoman, more than 40 other industrialized countries have already banned asbestos. Around the world, chlorine producers are phasing out dangerous and inefficient methods in favor of safer and more environmentally responsible technology. We need to help our U.S. companies embrace those new greener approaches today.

I am also very grateful that industry leaders have stepped up to the plate to work with me in achieving a goal that everyone supports: a ban on the production and importation of asbestos in the United States. These corporate leaders also strongly support the need to better educate the public and to provide more for research and treatment dollars to better mitigate the effects of asbestos on workers and their families.

I look forward to working with all of the members of this committee to achieve a bipartisan consensus on banning asbestos in the United States in this Congress. Chairwoman Boxer, I know this hearing will help us go a long way in achieving that goal, and I really want to thank you. We have lost enough people, Fred, Brian

and others, and we have a responsibility to protect tens of thousands of people just like them. So thank you very much for this opportunity to testify and for your hearing on this important legislation. Thank you.

[The prepared statement of Senator Murray follows:]

STATEMENT OF HON. PATTY MURRAY, U.S. SENATOR FROM THE
STATE OF WASHINGTON

Thank you, Chairwoman Boxer, for convening this hearing and for your long-standing support of my efforts to ban asbestos in the United States.

I'm so pleased to be here this morning with the distinguished group of witnesses you have assembled for this hearing.

I especially want to acknowledge the efforts of three of your witnesses, Dr. Barry Castleman, Dr. Dick Lemen and Linda Reinstein. Without their tireless work, we would not be where we are today—on the verge of finally protecting Americans from deadly asbestos.

You've called this hearing to examine the health effects of asbestos and ways to minimize its harm. I've worked on this issue for 6 years, and I can tell you that

- asbestos is deadly,
- it's devastating families and communities,
- and every day that we wait to ban it, we're sentencing more Americans to an early and avoidable death.

Studies show that asbestos exposure kills up to 10,000 Americans each year. I want to introduce you to two of them.

This is George "Fred" Biekkola from Michigan. Fred served in World War II.

For almost 30 years, he worked for a mining company in Michigan, where he was exposed to asbestos. Fred testified at my first hearing on asbestos 6 years ago. I'll never forget what he told us. He said:

"Senators, please make sure that what happened to me won't happen to anyone else. . . . Workers like me are counting on you to protect us. Please don't let us down."

I'm sad to say that we let Fred down. We didn't ban asbestos. We didn't warn the public. And we didn't invest in research and treatment. Fred died of asbestosis and mesothelioma on April 7, 2004.

Sadly, Fred is not the only advocate we've lost over the years because Congress has failed to act.

This is Brian Harvey, a teacher from Marysville, Washington. Brian stood by my side as I introduced my first bill to ban asbestos in July 2002.

Most asbestos victims die within a year of being diagnosed. Amazingly, Brian lived for 6 years. He knew he was living on borrowed time, so he used his time to fight for others. He stood by my side again in 2004 at another press conference, but sadly Brian died in July of 2005.

Fred and Brian are not with us, but their words hang over this hearing.

As I mentioned, it's estimated that each year, up to 10,000 Americans die every year from asbestos-related causes. I've been at this for 6 years, this is my third bill, and I know we cannot wait another year to fix this problem. The stakes are just too high.

To anyone who says, "We don't need this bill," I would just pose one question:

- "How many more Americans have to die before our government finally does the right thing and bans asbestos?"

We have to do the right thing, and we have to do it now. As I look at this issue, four problems stand out.

1. Asbestos is Deadly

First, asbestos is deadly. It's so deadly that there is no known safe level of exposure. It only takes a tiny bit of fiber to cause disease.

2. Asbestos is Widespread

Second, asbestos is everywhere. It's put into consumer and industrial products on purpose every day.

My staff bought these brake pads in an automotive repair store in my home state. They contain asbestos. Brake pads like these are on tens of thousands of cars. Anytime one of those cars goes in for maintenance, a mechanic could be unknowingly exposed to deadly asbestos. Fortunately, there are alternatives.

These brake pads are made without asbestos, and they work just as well. We shouldn't keep selling asbestos products and putting workers and countless consumers at risk. There are thousands of other products that contain asbestos includ-

ing floor tiles, roofing material, cement pipes, and even hair dryers. And deadly asbestos is still putting construction and maintenance workers at risk. Below this hearing room and under the Capitol there are tunnels where we know asbestos exists and workers have been exposed.

3. Asbestos is Still Legal

Third, asbestos is still legal. Many Americans assume—as I did—that asbestos is already banned, but it's not. In 1989, the EPA tried to ban asbestos, but most of those regulations were overturned in court in 1991. As a result, while new applications for asbestos were banned, asbestos is still being imported and used in consumer and industrial products.

4. Strong Need for Research and Treatment

Fourth, research and treatment for asbestos diseases are not very far along. Doctors have been hampered by a lack of funding for research on how asbestos fibers actually cause disease and what treatment strategies work best. Industrial hygienists have been hampered by the lack of research on how to best measure asbestos fibers in the air.

I know that the Mesothelioma Applied Research Foundation (MARF) has privately awarded over \$4 million in grants.

The Foundation's investment in research is helping motivate brilliant investigators to study mesothelioma. But the Foundation's seed money is not enough. Federal funding is critical to the research effort if we are truly going to help people.

My bill also requires collaboration among the 10 research and treatment centers established under the bill along with the National Cancer Institute, the Department of Veterans Affairs and the National Institute for Occupational Safety and Health (NIOSH).

Because nearly one-third of mesothelioma victims were exposed to asbestos while serving in the U.S. Navy, my bill directs the Pentagon to conduct additional research on asbestos disease, early detection and treatment as well.

I am very encouraged that the NIOSH has embarked on an ambitious research roadmap to better answer current scientific questions about appropriate occupational levels of exposure.

THE BAN ASBESTOS IN AMERICA ACT OF 2007

To address the national scourge of asbestos, I've again introduced the Ban Asbestos in America Act of 2007 (S. 742).

My bill does three things:

First, my bill bans asbestos. It prohibits the importation, manufacture, processing and distribution of products containing asbestos. Unfortunately some 2,500 metric tons of asbestos was used in the U.S. in 2005 and imports of products containing asbestos in cement pipe, tiles, brake gaskets and linings continue unabated today.

Second, my bill dramatically expands research and treatment. It creates a \$50 million, 10-center "Asbestos-Related Disease Research and Treatment Network." It creates a new National Asbestos-Related Disease Registry. It supports research at the Department of Defense and launches a study to determine the most promising areas for new research.

Finally, my bill launches a public education campaign to better inform Americans of the dangers of exposures to asbestos in the workplace and in the environment, while also providing helpful steps all of us can take to better protect our families.

OTHER COUNTRIES ARE PROTECTING THEIR CITIZENS

I know we can and should make progress in banning asbestos. More than 40 other industrialized countries have already banned asbestos. Around the world, chlorine producers are phasing out dangerous and inefficient methods in favor of safer and more environmentally responsible technology. We need to help U.S. companies embrace new, greener approaches today.

I am very grateful that industry leaders have stepped up to the plate to work with me in achieving a goal everyone supports—a ban on the production and importation of asbestos in the U.S.

These corporate leaders also strongly support the need to better educate the public and to provide for more research and treatment dollars to better mitigate the effects of asbestos on workers and their families.

I look forward to working with all of the Members of the Environment and Public Works Committee to achieve a bipartisan consensus on banning asbestos in the U.S. in this Congress. Chairwoman Boxer, I know this hearing will go a long way in helping us achieve that goal.

We've lost enough people like Brian and Fred, and we have a responsibility to protect tens of thousands of people just like them.

Thank you again for the opportunity to testify.

Senator BOXER. Senator Murray, thank you once again for your testimony.

I don't have any questions for you, I just want to tell you my intent as Chair, and I think Senator Inhofe knows this, is to really move this bill as quickly as I can, with the great hope that you and Senator Isakson can reach an accord. I think it would be a proud day for this committee, and I think it would be a proud day for the Senate if we finally did something that frankly most Americans think we have already done in the past, and do it in a wise way and make a statement to all those people out there who have lost loved ones and those who fear for the future that we are relevant to their lives. And we are going to do this.

So I don't have any questions. I guess I have one. Are you ready to work with us to get this bill to the floor, for as long as it takes?

Senator MURRAY. I am ready to go. I again want to thank Senator Isakson and his committee staff for working with us on this.

Senator BOXER. Very good.

Senator Isakson, do you have any questions for Senator Murray?

Senator ISAKSON. Just to thank her for her diligence and hard work and courtesy to me and my staff. I think we can put this together quite quickly.

Senator BOXER. Senator Lautenberg, any questions?

Senator LAUTENBERG. Just to commend Senator Murray for, as I said before, her persistence in doing this, and alerting the country to the danger of this product, and to take it away as quickly as we can, so that people aren't exposed to it. My congratulations.

Senator BOXER. Senator Vitter.

Senator VITTER. I would just ask Senator Murray her thoughts on the chlor-alkali issue in particular and where you are perhaps with Johnny and others on discussion of that aspect of the bill.

Senator MURRAY. Senator Vitter, as you know, a number of the chlorine producers are coming up with alternative methods. Part of our bill hopes to help provide support for them to do that.

But I think the important thing in our bill that will help everyone is that not enough research has been done. Senator Inhofe mentioned a number of different fibers that people don't know enough about. We want to make sure that we do the right thing. That is why the research that is part of this bill is absolutely critical.

You mentioned a number of times that it is important to be science-based. If you don't have the science, it is very difficult to make a decision. Meanwhile, people are dying because we are not doing the right thing. So I think that you will be satisfied that Senator Isakson has addressed a number of those issues that you raise concerns about.

Senator VITTER. As that science is being done or whatever you are describing, would use of asbestos in chlor-alkali production under the parameters I was describing, with the process fully enclosed, no humans in contact, would that be allowed or not?

Senator MURRAY. Let me refer to my staff on the latest reiteration of the language that we have been working with. Perhaps we can have my staff work with yours as we are working through the committee process and get you an answer so we are all accurate.

Senator VITTER. OK.

Senator BOXER. Let me say, Senator Vitter, if I might have your attention for a second, Senator Murray, I know Senator Murray's staff has been meeting with Dow and others to see if there is a way to do this right. I wanted to place into the record, without objection, an article, Responding to A Harsh Business Environment: a New Diaphragm for the Chlor-alkali Industry. It talks about a new industry, PPG Industries has responded by developing a new separator for its diaphragm cells operating at its plants at Natrium, West Virginia and Lake Charles, LA. The new separator is asbestos-free, energy efficient and durable. The new separator is named Tephram.

In any case, I am going to put this into the record for you to read.

[The referenced material follows:]

Responding to a Harsh Business Environment: A New Diaphragm for the Chlor-Alkali Industry

by Sajjad Ahmed and Peter Foller

The last several years have been difficult for producers of U.S. chlor-alkali chemicals. Low prices for chlorine and caustic and escalating fuel costs have resulted in several plant closures and have squeezed the profitability of those that have remained. For those plants operating with diaphragm cell technology, which still comprises a major portion of domestic production, conversion to membrane cells is not a viable option. Replacement of diaphragm cells with membrane cells entails abandonment of existing highly efficient caustic evaporator trains, provision for solid salt to replace solution-mined salt, and significant brine treatment upgrades, along with writing off the original investment in diaphragm cells.

In addition to the direct cost issues, there are indirect costs associated with diaphragm cell production of chlorine. These issues center on the environmental health and safety concerns over asbestos, which is currently used in diaphragm cells as a separator to keep products from mixing. Although the chlorine industry has an excellent track record of using asbestos safely, many businesses have been forced into bankruptcy due to high damage awards resulting from the past sale of asbestos-containing products. A great deal of pressure exists to find alternative diaphragm cell separators, if only to assure continuity of supply of raw materials.

PPG Industries has responded to this difficult period by developing a new separator for its diaphragm cells operating at its plants at Natrium, West Virginia, and Lake Charles, Louisiana. The new separator is asbestos-free, energy efficient, and durable. The new separator, which we have named Tephram, is capable of retrofit to existing diaphragm cell facilities with minimal changes to existing equipment and procedures. Full circuit operation has been under-

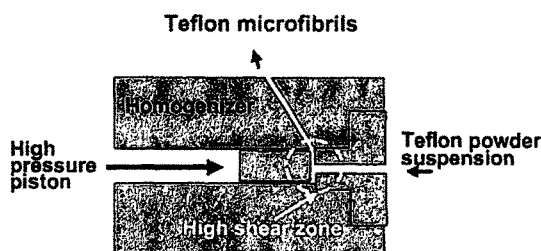


Fig. 1. Manufacture of pTFE Microfibrils.

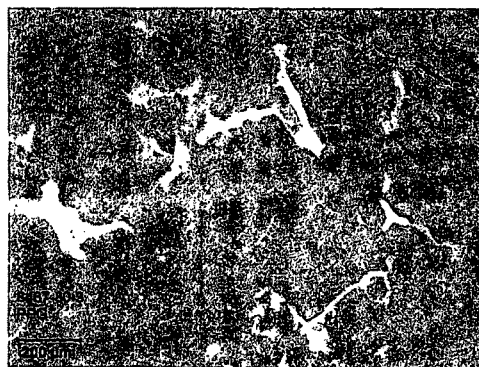


Fig. 2. Typical pTFE Microfibrils.

way at a small circuit at our Natrium location for over 10 years with the Tephram diaphragm. Just this year, PPG completed conversion of its largest diaphragm cell area at Lake Charles from asbestos to Tephram diaphragms. Currently, conversion of the Natrium plant's largest circuit is underway. The

success seen so far indicates that the diaphragm will be adaptable to a variety of cell designs. The cells to which the Tephram diaphragm has been successfully applied at PPG locations include the Columbia N6, the Glanor V-1244, and the Oxytech MDC-55.

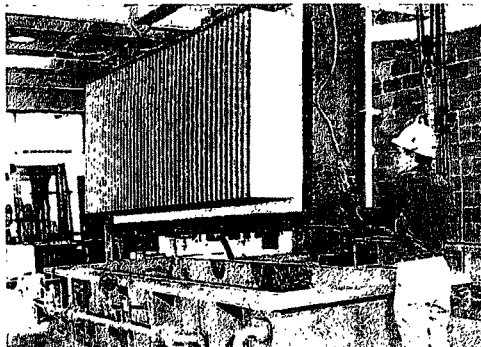


Fig. 3. Tephram Diaphragm on a Glanor V-1244 Electrolyzer Cathode.

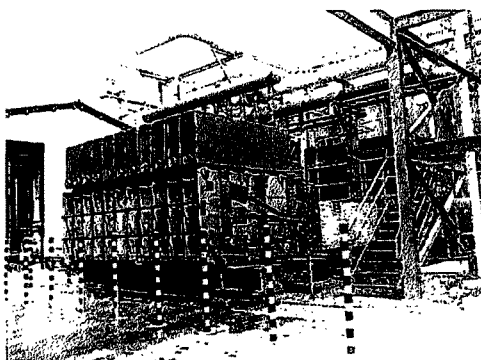


Fig. 4. Assembled Glanor V-1144 Electrolyzer at Lake Charles "Plant C."

The requirements for any new chlor-alkali diaphragm are very demanding. Here is a partial list:

- Resistance to chlorine and caustic
- Separation of chlorine and hydrogen
- Permeability sufficient to match the desired brine feed
- High conductivity
- Resistance to brine upsets
- Rapid recovery from outages
- Minimal impact on established plant procedures and existing capital
- Long life

What is a Tephram Diaphragm?

The Tephram diaphragm is a fluoropolymer-based separator comprised

of pTFE (polytetrafluoroethylene) microfibrils and a secondary, longer, pTFE fiber. The longer fiber, DuPont's Teflon Floc pTFE fiber, offers good reinforcement of the diaphragm but is too coarse to be used as the primary fiber. The microfibrils are prepared by a PPG-patented process and comprise the bulk of the diaphragm material. The microfibrils are made by forcing an aqueous suspension of pTFE powder through an orifice at high pressure. The process is depicted in Fig. 1, and the resulting microfibril product is shown in Fig. 2.

The microfibrils prepared by this process become highly entangled in the deposition process so that there is no need for high temperature sintering to assure diaphragm integrity. The fact

that the diaphragm is an entangled mat instead of sintered is thought to play an important part in avoiding hydrogen transfer from the catholyte to anolyte along Teflon pathways during cell operation, a problem commonly associated with perfluorocarbon based diaphragms. To assure that the diaphragm is wettable, DuPont's Nafion solution is used to coat the pTFE so that the normally hydrophobic fibers are permanently wetted.

The pTFE microfibrils and Teflon Floc fiber components are mixed with other deposition aids in a water-based slurry containing 2-4 wt.% suspended solids and vacuum deposited directly onto the cell cathode. Both woven screen and perforated plate cathodes can be used. The diaphragm mat is then dried at low temperature (< 100 °C) to remove water. The fact that drying steps are done at low temperature puts less stress on cell components. A topcoat is next applied by vacuum depositing inert inorganic fillers into the diaphragm to adjust permeability as desired. The fillers typically used are Attapulgite clay and zirconium oxide. Once the topcoat is applied, the diaphragm is dried and is ready for assembly into a cell.

The steps required for Tephram diaphragm manufacture are:

- High shear manufacture of pTFE microfibrils
- Mixing of aqueous slurry (filter aids, surfactants, viscosity modifier, etc.)
- Vacuum deposition of diaphragm onto cathode and drying
- Topcoat application (zirconium oxide & Attapulgite clay)
- Drying
- Cell assembly

Except for the microfibril preparation and some mixing equipment, the diaphragm manufacture uses the same equipment as asbestos diaphragms. This allows for easy transition from asbestos to Tephram diaphragms. The final Tephram diaphragm is typically about 0.10 inch thick and has an area density of about 0.4 lb/ft². A photo of a completed Tephram diaphragm on one of 12 cathode elements of a bipolar Glanor V-1244 electrolyzer is shown as Fig. 3.

A fully assembled 25 Ton Cl₂/day Glanor V-1244 Electrolyzer ready to move into place is shown as Fig. 4. Sixty four such electrolyzers were converted to Tephram diaphragms in the conversion of Lake Charles' Plant C.

Start-up and Operating Maintenance of Tephram Diaphragm Cells

Tephram diaphragms are started up and operated much like their asbestos diaphragm counterparts. The diaphragm is flushed with water to remove surfactants and to wet the diaphragm. This gives a low start-up voltage and avoids excessive foaming from surfactant residue. Following the water flush, the cell is filled with hot brine and started at full operating load. The cell is monitored closely during the first several hours of operation to judge diaphragm permeability. The Tephram diaphragm is initially more permeable than required for high strength caustic operation. This allows for the addition of materials to the brine feed to adjust the permeability of the diaphragm to match circuit requirements. The higher permeability also gives a built in cushion for the occasional brine upset, which typically adds an additional burden of magnesium and calcium.

Materials used for permeability trimming include primarily Attapulgite clay and magnesium hydroxide. The clay particles act by filtration and they swell within the diaphragm matrix. The magnesium forms a gelatinous hydroxide precipitate upon contact with hydroxide diffusion or leakage from the catholyte. Careful attention to amounts of materials added, flow rates, and pH allow for permeability control with good efficiency. Overdosing with magnesium hydroxide can result in excess plugging and lower efficiency, just as with asbestos. However, the magnesium will generally be dissolved from Tephram diaphragms with continued operation, and the occasional circuit outage, so that the efficiency loss is not irreversible. Uneven magnesium hydroxide precipitation is, nevertheless, the most common cause of longer term decline in efficiency with Tephram diaphragms.

Operating Experience with Tephram Diaphragm Cells

Operating results in the smaller Columbia N6 circuit at Natrium have been described earlier.¹⁻⁴ Typically in this smaller (160 T Cl₂/day) circuit, the power requirements were slightly better or equal to those obtained with asbestos diaphragms. The smaller cir-

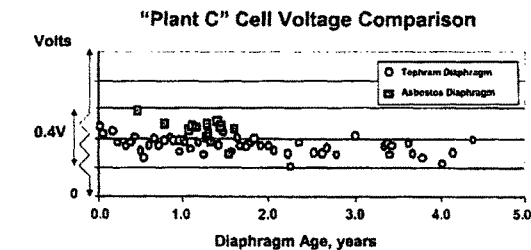


Fig. 5. Glanor V-1244 Cell Voltage Comparison.

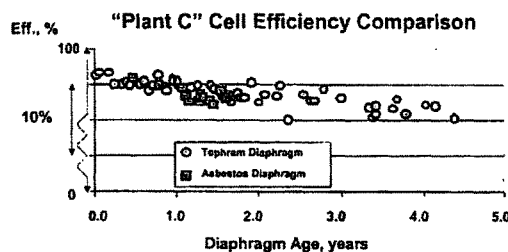


Fig. 6. Glanor V-1244 Cell Efficiency Comparison.

$$Cl_2 \text{ Eff} = \frac{\text{vol\% } Cl_2}{\left(\text{vol\% } Cl_2 + 2 \otimes \text{vol\% corr } O_2 \right) + 6 \otimes \left(\frac{\text{gpl NaClO, catholyte}}{\text{gpl NaOH catholyte}} \right) \otimes \text{vol\% } Cl_2}$$

Eq. 1. The Calculation of Oxy "6" Current Efficiency for Chlorine.

cuit demonstrated the ruggedness of the Tephram diaphragm. Generally speaking, cell renewal replacements were due to cell mechanical problems, such as mat leaks or gasket failures, before the diaphragm failed. Operating lives as long as four years were obtained. The ruggedness of the diaphragm has been further demonstrated by empirical evidence: the circuit was mothballed for several months with no special precautions being taken, such as addition of reducing agents or cathodic protection, and restarted with no unusual problems.

At Lake Charles' 1,800 Ton Cl₂/day Glanor V-1244 Plant C circuit, Tephram diaphragms have demonstrated both a longer life and better power efficiency than asbestos diaphragms. Relative comparisons of voltage and efficiency with asbestos are shown in Figs. 5 and 6. Absolute values for efficiency and

voltage are not shown in order to protect company-confidential information. The data shown in the figures are for a period where both types of diaphragms were still in operation so that a same-time, same-circuit comparison could be made.

The chlorine current efficiency values were calculated using the standard Oxy "6" equation which estimates efficiency from caustic concentration, oxygen in chlorine, and sodium chlorate in cell liquor (see Eq. 1 above).

The results for the Lake Charles V-1244 electrolyzers show that the voltage is slightly better and efficiency generally about the same as for the asbestos cells. This gives a power consumption advantage to the Tephram diaphragm cells. It should be noted that the comparison is conservative with respect to the power consumption advantage. This is due to preferential removal of

Table I. Plant C Operating Data with Tephram Diaphragms.				
V-1244 Electrolyzers	Diaphragm Age, days	NaOH, gpl	H ₂ , vol.%	O ₂ , vol.%
Average.....	764	137	0.12	2.24

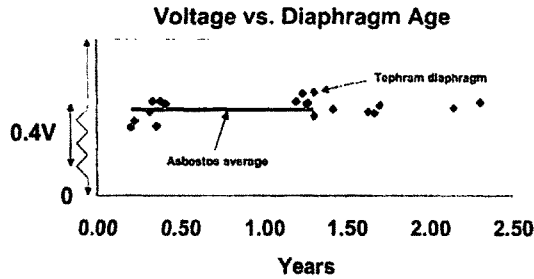


Fig. 7. Sodium MDC-55 Cell Comparison of Voltage.

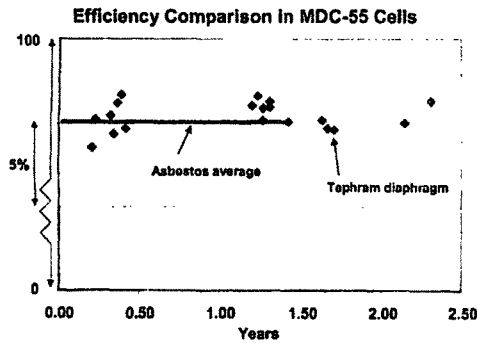


Fig. 8. Comparison of Asbestos and Tephram Diaphragms in Sodium MDC-55 Cells.

Table II. Sodium MDC-55 Cell Operating Data.				
Diaphragm Age, days	NaOH, gpl	H ₂ , vol.%	O ₂ , vol.%	
Tephram Diaphragms.....	398	126	0.10	1.87
Asbestos Diaphragms.....	299	141	0.15	2.15

the worst-performing shorter lived asbestos diaphragm cells while all Tephram diaphragms were maintained in operation as the conversion proceeded. Also noteworthy is the substantially longer life seen with the Tephram diaphragm cells. The asbestos cells have a life shorter than two years due to the degradation of asbestos from load swings and brine upsets. These factors cause asbestos diaphragms to become less permeable to brine flow. The Tephram diaphragms are much more forgiving, because the fluorocarbon matrix is not chemically attacked as is asbestos. With load swings becoming more common as power costs and chlorine demand fluctuate, the operating stability of Tephram diaphragms becomes an important advantage.

The average caustic strength and anodic byproduct performance of the Lake Charles Plant C diaphragms is listed in Table I. The averages are for 64 electrolyzers, each of which contains 12 bipolar cell units. The data were taken shortly after the plant was fully converted from asbestos to Tephram diaphragms in 2003.

Conversion of the MDC-55 cell circuit at Natrium, WV, has been started. The incentive for this location has been the Tephram diaphragm's durability and recovery from load swings and brine upsets. Of the circuit of monopolar MDC-55 cells, at this writing approximately 15% have been converted to Tephram diaphragms. Because there is a large population of asbestos diaphragm cells still in operation, direct side-by-side comparisons are possible. A plot of recent Tephram diaphragm cell voltages is compared with the average voltage for asbestos diaphragms during the same period in Fig. 7.

A similar plot is given in Fig. 8 for Oxy6 chlorine efficiency. The plot compares efficiency for the two types of diaphragms during the same operating period.

With the MDC-55 cells, voltage and efficiency appear to be generally stable over the test period for the Tephram diaphragms. Again, the asbestos timeline does not extend as far as the Tephram diaphragm timeline due to the shorter life of the asbestos diaphragms.

The data in Table II are for a recent measurement of caustic strength and anodic by-product performance. Note that hydrogen in chlorine is not an issue, being generally less for Tephram

diaphragm cells than for the asbestos diaphragm cells.

The power consumption for the cells with the two types of diaphragm are roughly equivalent, because voltages and efficiency values are very close (power is proportional to voltage/efficiency). Even without a clear power savings, operating advantages such as resistance to load swings and brine upsets and longer diaphragm life have led to the decision to fully convert to the Tephram diaphragm in PPG's MDC-55 circuit.

The current status of the conversion of PPG's diaphragm circuits to Tephram diaphragms is shown in Fig. 9. At this time, over half of PPG's diaphragm cell chlorine capacity utilizes the Tephram diaphragm.

A summary comparison of PPG's experience with the Tephram diaphragm is given as Table III. These conclusions were developed through more than a decade of R&D. Table IV, the U.S. patents for the Tephram diaphragm, shows the program's origins in the 1980s.

PPG is well on the way to becoming asbestos-free at its diaphragm plant facilities. This is being achieved under very difficult business conditions for the chlor-alkali industry. The move to the non-asbestos diaphragm is being driven by two factors, power savings and the operating flexibility to tolerate load swings to match production needs. This does not imply that load swings have no impact on the Tephram diaphragm, only that it will not be destroyed, as will asbestos, and

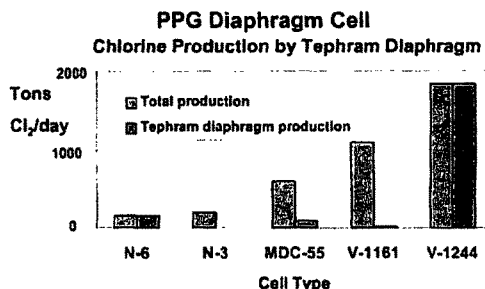


FIG. 9. PPG Tephram Diaphragm Chlorine Production by Cell Type.

Table III. Comparison of Asbestos and Tephram Diaphragms.

Life	Asbestos	Tephram
Life	8-15 months	4 years
Ruggedness	Easily plugged with impurities	Recovers from upsets
Responses to swings	Cycling destroys asbestos may go high level	No long-term effect
Safety	Carcinogen, caustic used in diaphragm preparation	No asbestos, non-hazardous water-base slurry
Materials Cost	Low cost but uncertain supply	Higher than asbestos, secure supply
Electrical Power Use	Lower or equivalent to asbestos

Table IV. Tephram Diaphragm Technology is protected by the following U.S. Patents.

Patent #	Inventor	Title	Year
6,299,939	DuBois, et al.	Method of Preparing a Diaphragm for an Electrolytic Cell	2001
6,296,745	DuBois, et al.	Method of Operating Chlor-alkali Electrolytic Cells	2001
6,059,944	DuBois, et al.	Diaphragm for Electrolytic Cell	2000
5,683,749	DuBois, et al.	Method of Preparing Asbestos-free Chlor-alkali Diaphragm	1997
5,630,930	Maloney	Method of Starting a Chlor-alkali Diaphragm Cell	1997
5,612,089	Dilmore, et al.	Method of Preparing Diaphragm for use in Chlor-alkali Cells	1997
5,567,298	DuBois, et al.	Method of Operating Chlor-alkali Cells	1996
5,192,401	DuBois, et al.	Diaphragm for Use in Chlor-alkali Cells	1993
5,188,712	Dilmore, et al.	Diaphragm for Use in Chlor-alkali Cells	1993
5,030,403	Pickens, et al.	Method of Making Polymeric Fibrils	1991
4,720,334	DuBois, et al.	Diaphragm for Electrolytic Cell	1988
4,680,101	Darlington, et al.	Electrolyte Permeable Diaphragm including a Polymeric Metal Oxide	1987
4,666,573	DuBois, et al.	Synthetic Diaphragm and Process of Use Thereof	1987

that eventual recovery is possible. The longer life of the Tephram diaphragm allows for savings in cell renewal materials and labor, since fewer cell turn-arounds are required. These savings more than offset the higher costs that are unavoidably associated with fluorocarbon materials. □

Acknowledgments

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(ret.); M. Schmidt; and H. Schussler; at Natrium: D. Bush; J. Maxwell; C. Hill; and R. Toumala; at Lake Charles: J. Cimini; T. Jeffery (ret.); S. Richardson; J. Hutchins (ret.); and others.

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Replacement of Asbestos in the Diaphragm Cell Process for Manufacture of Chlorine and Caustic Soda

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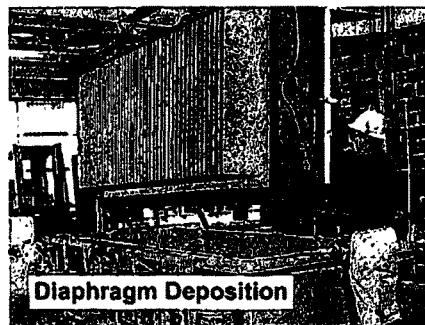
PPG has developed the Tephram[®] non-asbestos diaphragm for use in diaphragm electrolysis cells for the production of chlorine and caustic soda (NaOH). The Tephram diaphragm uses non-hazardous Teflon[®] fluoropolymer materials to replace asbestos. The Tephram diaphragm technology offers advantages in decreasing the complexity in handling raw materials (both asbestos itself and the corrosive chemicals used in asbestos diaphragm deposition) as well as in the disposal of asbestos materials at the end of their useful lives. Tephram diaphragms are easier to handle safely and are more environmentally friendly than asbestos diaphragms, and last much longer than asbestos diaphragms. At PPG's Lake Charles chlor-alkali complex an advantage in energy efficiency has been demonstrated. These advantages of greater durability and energy efficiency combine to reduce expenditure of cell renewal labor and consumption of both materials and energy. The impact of this technology is significant. The Chlor-alkali industry consumes approximately 1% of all the electric power generated in the United States. Domestically, diaphragm electrolysis cells account for about 54 million lbs/day of chlorine and 58 million lbs/day of caustic soda, or roughly 75% of U.S. chlorine and caustic soda production. These versatile basic chemicals are used as building blocks for the production of many products important to the U.S. economy, such as plastics, organic chemicals, aluminum, and titanium metals, coatings, refrigerants, safe drinking water, and pharmaceuticals.

Laboratory development of the Tephram diaphragm has been underway at PPG's Chemicals Technical Center (Monroeville, PA) for over 10 years. The first full conversion of a commercial diaphragm cell circuit to Tephram diaphragms was done at PPG's Nairium, West Virginia Plant in 1992. This 160 TPD diaphragm cell plant has operated exclusively with Tephram diaphragms since its conversion, marking its 10-year anniversary in 2002. Additional improvements to the Tephram diaphragm technology have been implemented regularly and full-scale demonstrations at PPG's Lake Charles, Louisiana Chlor-alkali complex were begun in 1996. At Lake Charles, an energy consumption advantage of about 3% over asbestos has been achieved. The conversion from asbestos to Tephram diaphragms was justified on the basis of reduced energy consumption, with health, safety, and environmental benefits as added bonuses.

Plant trials indicate that life of the diaphragm will be more than doubled with the Tephram diaphragm, with a 4-year average life expected. As of this writing (November, 2002), Tephram diaphragm cells are producing ~1700 ton/day of chlorine at the Lake Charles plant. Completion of this 1,800 ton/day conversion is expected by 2003. Further extension of the Tephram diaphragm to other cell designs is anticipated.

The Tephram diaphragm is mainly made of chemically inert Teflon fluoropolymer microfibers. These microfibers are made by pumping a fluoropolymer dispersion through a small orifice under very high shear conditions. The microfibers, along with wetting agents, thickeners, and other materials, are suspended in a water-based slurry. (A water-based slurry is much easier to handle safely than is the caustic-based slurry required with asbestos diaphragms.) The slurry is placed in a large tank and a diaphragm base mat is filtered onto the cathode structure by vacuum filtration. A photograph of Lake Charles' procedures for this diaphragm-forming step is shown in Figure 1. What is shown is one element of a 12-element PPG V-1244 bipolar electrolyzer. Each 12-element PPG V-1244 bipolar electrolyzer produces 25 T/day of chlorine.

Figure 1. Commercial Deposition of Diaphragm



The porosity of the diaphragm is controlled by the vacuum deposition of a topcoat (onto the base mat). This is deposited from a slurry of fine particulates following procedures similar to the deposition of the base mat. (In operation, porosity is further controlled with additives to the feed brine.) The deposition of Tephram is very similar to the deposition of asbestos, and, therefore, plant operations are minimally impacted by conversion to the new technology.

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Tephram[®] is a Registered Trademark of PPG Industries, Inc.

Senator BOXER. As usual, the entrepreneurship spirit has kicked in here, and there are alternatives coming. So I would love to share this with you.

Senator VITTER. I have looked at many things like that and I would love to read that. I guess the question in my mind is not, is there an alternative. The first question is, is there a safety issue and if there is, we need to do something about it. But if there is not, then we need to think.

Senator BOXER. A safety issue with the alternative?

Senator VITTER. No, with the use of asbestos in chlor-alkali production under an enclosed, wet process.

Senator MURRAY. I believe you have a number of witnesses who will be able to help you answer that question.

Senator VITTER. OK. Because I am not aware of any known cases of asbestos-related disease from that. If there is a safety issue, great.

Then the second question is, certainly there are alternatives. At what cost?

Senator BOXER. Senator Vitter, I think we will explore this in the next panels. My understanding is that there is a danger if bags rip and you have to clean up the asbestos, so it is not as clean as one would think. Certainly this is something that Senator Murray is trying to work on. If we can find alternatives, we ought to encourage alternatives. That is for sure.

OK, why don't we call up our next panel. Senator Murray, we thank you very much. We will let you know how the rest of the hearing went.

David Weissman, M.D., Director, Division of Respiratory Diseases at National Institute for Occupational Safety and Health; Captain Aubrey Keith Miller, M.D., Senior Medical Officer, Toxicologist, U.S. Public Health Service and Environmental Protection Agency; Melanie Marty, Ph.D., Chief Air Toxicology and Epidemiology Branch, California EPA, Office of Environmental Health Hazard Assessment.

So we welcome you to panel one. Your titles are very impressive and we welcome you here. Dr. Weissman, from NIOSH, why don't you begin? We will give you 5 minutes.

STATEMENT OF DAVID N. WEISSMAN, M.D., DIRECTOR, DIVISION OF RESPIRATORY DISEASE STUDIES, NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH, CENTERS FOR DISEASE CONTROL AND PREVENTION, U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES

Dr. WEISSMAN. Thank you. Madam Chair, members of the committee, thank you for giving me the opportunity to testify today. My name is David Weissman, and I direct the Division of Respiratory Disease Studies in the National Institute for Occupational Safety and Health, NIOSH.

For the last 21 years, I have been a pulmonary disease physician, serving in both academic medical centers and in Government. When asked to testify, I couldn't help but think of a colleague who recently died of mesothelioma. He was a very distinguished physician whose only known exposure to asbestos was as a college stu-

dent during a summer job. Forty years later, he developed mesothelioma and died at the age of 62.

In my oral comments today, I will focus on three of the issues addressed in the written testimony. First, I will describe the continued burden of asbestos-related diseases in the United States. Second, I will mention several issues relevant to prevention efforts in the occupational setting. Finally, I will address NIOSH's efforts to identify key research needs and strategies to address them as described in the draft NIOSH Road Map document.

A substantial number of people still die from asbestos-related disease in our country. Asbestosis deaths increased almost 20-fold from the late 1960s to the late 1990s and have plateaued since the year 2000 at about 1,500 per year. By contrast, mesothelioma deaths since 1999 have increased each year, up to 2,657 deaths in 2004, the most recent year for which we have data.

It should be noted that because the latency between exposure and disease onset is so long, current disease, to a large degree, reflects past exposures. Asbestos usage, as we have heard, hasn't been completely eliminated. Although domestic production of asbestos has ceased and importation of raw asbestos fibers has markedly declined, finished asbestos-containing products continue to be imported into the United States.

Asbestos-related diseases can be prevented by eliminating or limiting exposures to asbestos. The OSHA Permissible Exposure Limit, or PEL, for asbestos is 0.1 fibers per cubic centimeter of air. This limit was set in part based on the limit of detection of the exposure assessment method, a light microscopic method called phase contrast microscopy, or PCM. Exposure limits are usually set to reduce risk associated with exposures to a level at or below 1 per 1,000 working lifetimes with exposure every day over the working lifetime. Over such a working lifetime, exposure at the asbestos PEL is estimated to be associated with excess risk of cancer of 3.4 per 1,000, an excess risk of asbestosis of 2.5 per 1,000.

A major recent NIOSH effort has been the development of a draft road map document that details key scientific issues in asbestos and identifies research directions. One key question is which minerals should be treated as asbestos. Most regulatory definitions of asbestos do not explicitly include fibers of minerals such as winchite, richterite and erionite, despite their known similar health effects to asbestos.

In addition, significant controversy exists regarding other types of mineral particles that have the dimensions of fibers. For example, El Dorado, CA, is a site with natural mineral deposits that have been disturbed by construction and crushing of rock. Analyses of air and rock samples have identified structures called acicular actinolite. These particles have a different crystalline structure from that of fibrous actinolite asbestos. Research is needed to be better characterize their toxic potential.

Asbestos minerals have analogs that are crystallized in non-asbestiform or massive forms. A controversial type of mineral particle that we have heard about is the cleavage fragment, which can be generated from massive forms during their handling, crushing or processing, as occurs in mining and construction. Using current analytical methods, these cleavage fragments are often microscopi-

cally indistinguishable from asbestos fibers of asbestos mineral counterparts.

Another key question is whether the specified dimensions of asbestos fibers are appropriate. Currently, a mineral particle is detected by PCM and counted as a fiber if it has a length to width or aspect ratio of 3:1 and a length of at least microns. These counting rules include particles with diameters greater than 3 microns, which are unlikely to reach the airways or gas-exchange regions of the lung.

Also, PCM can't detect particles with diameters less than .25 microns, which although not visible by PCM are capable of causing harm. Finally, although longer fibers have been associated with greater potential for carcinogenicity, studies of fibers deposited in human tissues suggest that fibers less than 5 microns in length may also contribute to human disease, including cancer.

In order to address these questions, NIOSH has put forth a draft document called the Roadmap. It is developing a range of partnerships to address the goals in the Roadmap, including with other Federal agencies, labor, industry, academia and interested parties.

To summarize, asbestos-related diseases continue to be an important problem. Fortunately, much progress has been made. However, there is room to do better and several key issues remain to be fully addressed.

Thank you for the opportunity to testify today. I would be happy to answer any questions.

[The prepared statement of Dr. Weissman follows:]

STATEMENT OF DAVID N. WEISSMAN, M.D., DIRECTOR, DIVISION OF RESPIRATORY DISEASE STUDIES, NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH, CENTERS FOR DISEASE CONTROL AND PREVENTION, U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES

Madam Chair and members of the committee, I am Dr. David Weissman, and I direct the Division of Respiratory Disease Studies in the National Institute for Occupational Safety and Health (NIOSH), a part of the Centers for Disease Control and Prevention (CDC) within the Department of Health and Human Services (HHS). NIOSH is the federal agency responsible for conducting research and making recommendations to identify and prevent work-related illness and injury. I am also a pulmonary diseases physician, and over the last 20 years have seen firsthand the human suffering caused by asbestos. Thank you for the opportunity to provide testimony on the health effects of asbestos and efforts by NIOSH to address this important problem.

My testimony today will address current scientific knowledge about the health risks posed by exposure of workers to airborne asbestos. I will also provide an update on NIOSH's recent activities in this area, including NIOSH efforts to define key areas for research as described in the draft NIOSH document released in February for public comment, *Asbestos and Other Mineral Fibers: A Roadmap for Scientific Research*.

BACKGROUND

Asbestos is a term that is generally used to refer to a group of fibrous silicate minerals with exceptional resistance to degradation by heat, acids, bases, or solvents. The minerals are not combustible and have a high melting point and low thermal and electrical conductivity. Their fibers can be woven or incorporated into other materials. These and other useful properties resulted in their widespread commercial application during much of the 20th century. Unfortunately, widespread use of asbestos was followed by a marked increase in asbestos-related disease.

The definition of asbestos in many Federal regulations is limited to the fibrous forms of six specific commercial types of asbestiform minerals. One is from a class of minerals called serpentines, which have curved fibers: chrysotile. The other five are members of a class of minerals called amphiboles, which have straight fibers:

crocidolite, amosite, tremolite asbestos, actinolite asbestos, and anthophyllite asbestos. The elemental composition of the six asbestos minerals can vary slightly, even within a single fiber, as a result of geological conditions such as pressure, temperature, or proximity of other minerals. Recognizing these variations in elemental composition, the six asbestos minerals can be defined by their "solid-solution" mineral series. For example, the mineral series tremolite-ferroactinolite contains the asbestos mineral actinolite. These mineral series are considered solid-solutions in which cations (i.e., sodium, calcium, magnesium, iron, etc.) are replaced by other cations which can affect the elemental composition of the mineral without significantly altering the structure. As another example, the Libby, Montana vermiculite ore body contains amphibole asbestos fibers of the tremolite-actinolite-richterite-winchite solid solution series. The minerals in the solution series have only minor differences in chemical content and have similar, if not identical, health effects. A third example of a mineral that produces similar diseases as asbestos is erionite, a fibrous mineral that is neither a serpentine nor an amphibole. It belongs to an entirely different class of minerals called zeolites.

ASBESTOS-RELATED DISEASES

Exposure to asbestos significantly increases the risk of developing several types of cancer and non-cancerous diseases. Most asbestos-related diseases, particularly the cancers, have long latency periods often extending 10–40 years from initial exposure to onset of illness. These include:

(1) Asbestosis—a non-cancerous disease characterized by scarring of the air-exchange regions of the lungs. Progressive lung damage can cause progressive shortness of breath and inability to engage in physical activity, as well as other symptoms such as coughing and chest pain;

(2) Lung cancer—for which asbestos is one of the leading causes among non-smokers, and which occurs at dramatically high rates among asbestos-exposed smokers;

(3) Malignant mesothelioma—an almost invariably fatal cancer of the tissue covering the lungs and chest wall (called the pleura) or abdomen (called the peritoneum) for which asbestos and similar fibers are the only known cause; and

(4) Non-malignant pleural disease—asbestos exposure can affect the pleura in several ways. It can cause a painful accumulation of bloody fluid surrounding the lungs. It can cause a circumscribed thickening, fibrosis, and sometimes calcification of pleural tissue—a condition called pleural plaques. Finally, it can cause a more severe condition with more extensive and sometimes constricting scarring of the tissue surrounding the lungs called diffuse pleural thickening.

In addition, asbestos exposure is associated with excess mortality due to cancer of the larynx and cancer of the gastrointestinal tract. The various types of cancers caused by asbestos are often fatal within a few years after initial diagnosis. In contrast, asbestosis deaths typically occur only after many years of suffering from impaired breathing.

The risk of developing adverse health effects from asbestos is related to the amount and duration of exposure to airborne asbestos fibers. Exposure occurs in the occupational setting when microscopic asbestos fibers become airborne during various industrial processes or from handling of asbestos-containing materials. The fibers can then be inhaled and/or swallowed. In the lungs, asbestos fibers can interact with cellular targets such as alveolar macrophages and alveolar epithelial cells, inducing a chain of events leading to scarring and/or cancer in the lungs. Fibers can also translocate through the lungs to the pleura, where they can cause malignant mesothelioma and nonmalignant pleural disease. Key factors associated with the carcinogenic potential of asbestos fibers include: particle length (longer fibers are more toxic than shorter fibers); diameter (fibers ≤ 3 micrometers in diameter are more likely than thicker fibers to be inhaled into the lungs, and fibers < 0.5 micrometers in diameter are more likely to migrate through lung tissue to the pleura); and biopersistence (fibers able to persist in the lung and not be cleared from the lung by physiological lung defense mechanisms are more likely to cause adverse health effects).

Asbestos-related diseases can be prevented by eliminating or limiting exposures to asbestos. The Occupational Safety and Health Administration (OSHA), the Mine Safety and Health Administration (MSHA), and the Environmental Protection Agency (EPA) regulate the six asbestos minerals. The OSHA permissible exposure limit (PEL) for asbestos is 0.1 fibers per cubic centimeter (cc) of air. This limit was set in part based on the limit of detection of the exposure assessment method specified in the standard (phase contrast microscopy (PCM)) and is not completely protective against asbestos-induced disease. Occupational exposure limits are generally set to

reduce risk associated with exposures to a level at or below 1 per 1,000 working lifetimes.

The risk analyses upon which the OSHA PEL and MSHA's proposal to revise its PEL are based were recently detailed by MSHA in its proposed rule. It should be noted that these risk analyses make the maximally protective assumption that exposure would be at the PEL every work day over an entire 45-year working lifetime. Over such a working lifetime, exposure at the OSHA asbestos PEL is estimated to be associated with an excess risk of cancer (lung, mesothelioma, and gastrointestinal) of 3.4 cases per 1,000 exposed individuals and an excess risk of asbestosis of 2.5 cases per 1,000 exposed individuals. In mining, the current MSHA PEL for asbestos is 20-fold higher at two fibers per cc air. Were exposure to the current MSHA PEL to occur every day over a 45-year working lifetime, it would be associated with an excess risk of cancer of 64.1 cases per 1,000 exposed individuals and an excess risk of asbestosis of 49.7 cases per 1,000 exposed individuals. Fortunately, the U.S. mining industry does not currently mine or produce asbestos and asbestos sampling data presented in MSHA's proposed rule showed low exposures for the mining population. MSHA has proposed to reduce its PEL to make it consistent with the OSHA PEL, and NIOSH has provided public comments in support of this proposed rule.

BURDEN OF ASBESTOS-RELATED DISEASES

NIOSH has tracked annual U.S. asbestosis deaths since 1968 and malignant mesothelioma deaths since 1999 using death certificate data in the National Occupational Respiratory Mortality System (NORMS). Data from NORMS show that asbestosis deaths increased almost 20-fold from the late 1960s to the late 1990s and have apparently plateaued only since 2000 at approximately 1,500 per year (Figure 1). By contrast, mesothelioma deaths continue to rise (Table 1). Current asbestos and mesothelioma mortality reflect past exposures because the latency between exposure and disease onset is long, particularly for mesothelioma, and asbestosis is a chronic disease, with affected individuals typically living for many years with the disease before succumbing.

Figure 1. Number of asbestosis deaths, U.S. residents age 15 and over, 1968–2004. Source: National Occupational Respiratory Mortality System (NORMS), found at: <http://webappa.cdc.gov/ords/norms.html>.

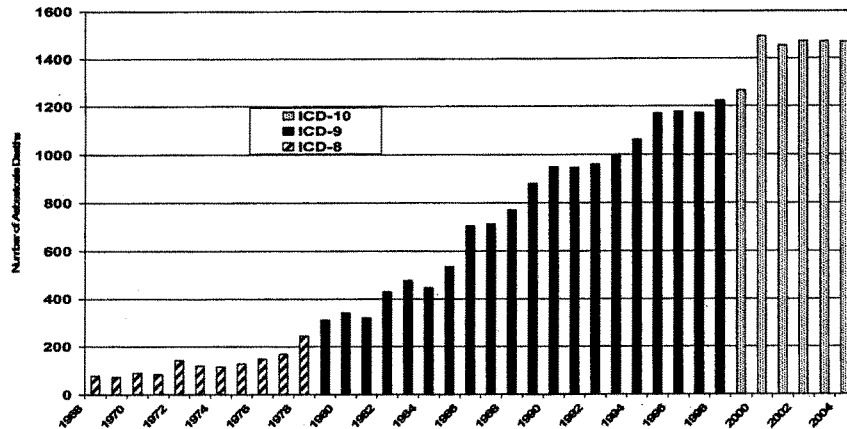


Table 1—Number of mesothelioma deaths, U.S. residents age 15 and over, 1999–2004

Year	Deaths
1999	2,484
2000	2,531
2001	2,509
2002	112,573
2003	112,625
2004	112,657

**Table 1—Number of mesothelioma deaths, U.S. residents age 15 and over, 1999–2004—
Continued**

Year	Deaths
1999–2004 (total)	115,379

Source: NORMS (<http://webappa.cdc.gov/ords/norms.html>)

Over time, the annual number of deaths should decrease substantially as a result of reductions in exposures. However, asbestos usage has not been completely eliminated. Although domestic production of asbestos has ceased and importation of raw asbestos fibers has markedly declined, many finished asbestos-containing products continue to be imported into the United States. These include asbestos-cement sheets, panels, and tiles; corrugated sheets; and automotive friction products. In addition, a reservoir of asbestos-containing materials remains in place in older buildings and machinery. Thus, even with limitations or exclusions from new use, occupational exposures to asbestos will continue, albeit at a far lower level than in the past.

UPDATE ON NIOSH ACTIVITIES RELATED TO ASBESTOS

NIOSH continues to work actively to address issues related to asbestos-induced lung disease. We are continuing to track asbestosis deaths, mesothelioma deaths, and occupational exposures to asbestos and have plans to include updated findings in an upcoming new edition of the recurring NIOSH document, the “Work-Related Lung Disease Surveillance Report.” Updates are also available on the NIOSH Web site.

NIOSH recently reported updated information on the occupational respiratory disease mortality among workers who mined, milled, and processed vermiculite contaminated with asbestiform fibers, including winchite, richterite, and tremolite from the mine near Libby, Montana. These workers had significantly increased rates of death from cancer, including lung cancer and malignant mesothelioma. They also had significantly increased rates of death from nonmalignant respiratory disease, including asbestosis and chronic obstructive pulmonary disease. Exposure-response relationships were demonstrated, with increasing fiber exposure associated with increasing mortality from lung cancer, asbestosis, and noncancerous chronic respiratory disease. This report adds to the growing body of literature documenting the adverse effects of exposure to Libby amphibole fibers.

With regard to Libby, the activities of the Agency for Toxic Substances and Disease Registry (ATSDR), an important partner of NIOSH, should be noted. A medical screening program conducted by ATSDR in Libby revealed an unusually high rate of asbestos-related disease among participants. Although many of these participants were former mine workers, others were their household contacts or community members with possible environmental exposures. Based on these findings, ATSDR established a Tremolite Asbestos Registry, which will complement NIOSH’s work by tracking the health outcomes of exposed individuals over time. To date, ATSDR has enrolled more than 4,000 individuals—comprising 83 percent of former Libby mine workers, their household contacts and a defined set of other local residents—and will administer follow-up interviews and medical screenings on a regular basis. “Take-home” exposures—involving family members of workers who bring asbestos home on their hair, clothing, or shoes—is a well-recognized hazard addressed by NIOSH in a 1995 report to Congress (<http://www.cdc.gov/niosh/contamin.html>), so ATSDR’s inclusion of household contacts in the registry will contribute important information to the body of research. In addition to research, ATSDR will use the registry to provide participants with information about new therapies that may become available in the future. ATSDR is also studying exposures to asbestiform fiber-contaminated vermiculite ore from Libby that was processed at sites in California, Ohio, Minnesota, New Jersey, New York and Wisconsin. ATSDR plans to use the findings of the registry and studies conducted at processing sites to develop a research agenda for Libby amphibole-related research.

NIOSH is doing research to clarify the relationships between fiber dimensions (length and diameter) and the risk for developing lung cancer or asbestosis through follow-up studies of a cohort of chrysotile-exposed South Carolina textile workers. NIOSH originally reported on this cohort in the 1980s. Exposures were originally evaluated by PCM. Since then, archived samples collected by NIOSH have been re-analyzed by transmission electron microscopy (TEM) to better evaluate fiber dimensions, including fibers too small to be seen by PCM. Also, mortality information about the cohort has been updated. Based on these data, fiber size-specific exposure

estimates have been developed for the cohort. Analyses are underway to determine the influence of fiber length and width on lung disease risk. These findings will help to inform approaches to quantitative risk assessment, particularly the potential utility of risk assessment based on fiber size.

NIOSH is also doing research in the area of exposure assessment. A recently published American Society for Testing and Materials (ASTM) International Standard—“Method for Sampling and Counting Airborne Fibers, Including Asbestos Fibers, In Mines and Quarries, by Phase Contrast Microscopy” (D7200-06)—contains a proposed methodology for separating fiber-like particles other than asbestos from probable asbestos fibers. The new ASTM procedure has not yet been validated to confirm that it produces accurate, reproducible results. A current NIOSH study will address this issue by documenting the performance of the ASTM procedure. Another important issue in asbestos exposure assessment is sampling in dusty environments, such as mines. Traditional filter samplers quickly become overloaded with dust, limiting the ability to detect asbestos fibers. One approach to reducing this problem is to use a sampler that only collects particles small enough to reach the airways of the lung when inhaled, and not larger particles that mostly deposit in the mouth, nose, and throat. NIOSH is currently evaluating two such “thoracic” particulate samplers in comparison to the traditional filter sampler in two different mining environments.

NIOSH is pursuing research relevant to the detection of asbestos-related respiratory diseases. Traditionally, film-based chest radiographs have been used in epidemiological studies evaluating workers for pulmonary and pleural disease associated with asbestos exposure. This is because only film-based chest radiographs may be systematically classified for changes of dust-induced lung disease (pneumoconiosis) using the widely accepted International Labour Organization (ILO) classification system. However, in the United States, digital chest radiography has largely replaced film-based radiography. NIOSH has funded research to evaluate the impact of classifying digital, instead of film-based, chest x-rays on the detection and classification of pulmonary and pleural disease. Initial results suggest that the two methods do not differ significantly in detection of interstitial (lung tissue) processes, but do differ in detection of pleural processes, with fewer pleural changes detected in those undergoing digital chest radiography. In follow up to this finding, NIOSH is assisting ATSDR in performing a study to compare detection of pleural changes in those exposed to Libby amphibole by film-based and digital radiography, with findings of computed tomography scans of the chest serving as a “gold standard.”

In 2006, NIOSH published a Recommended Exposure Limit (REL) for another type of inorganic fiber, refractory ceramic fibers (RCF). Although RCF are man-made fibers which differ from asbestos in toxicity, many of the same issues relevant to asbestos such as fiber length, diameter, and biopersistence were considered in developing the NIOSH REL of 0.5 fibers per cc.

ASBESTOS AND OTHER MINERAL FIBERS: A ROADMAP FOR SCIENTIFIC RESEARCH

A major recent NIOSH effort has been the development of a draft “Roadmap” document that details key scientific issues in asbestos and identifies research directions to address these issues. Key issues include the following:

Which minerals should be treated as asbestos?

As already described, most regulatory definitions of asbestos do not explicitly include minerals such as winchite, richterite, and erionite, despite the known similar health effects of their fibers to those of the explicitly listed asbestos minerals. In addition, significant controversy exists regarding other types of mineral particles that have the dimensions of fibers. For example, El Dorado, California, is a site with natural deposits of amphibole that have been disturbed by construction and crushing of rock. Analyses of air and rock samples have identified the presence of actinolite in the form of needle-like crystalline structures called “acicular/prismatic actinolite.” Although many of these amphibole particles meet the dimensional criteria of asbestos fibers, they have a different crystalline structure from fibrous actinolite asbestos. A recent report by investigators from the University of California found that residential proximity to deposits of “naturally occurring asbestos” such as those in the vicinity of El Dorado was associated with increased risk for mesothelioma, implicating these minerals as a possible health hazard. It should be noted that this report did not include actual measurement of fiber exposures associated with residence in these areas.

Asbestos minerals have analogs that are crystallized in non-asbestiform (massive) structures. A controversial type of mineral particle is the “cleavage fragment,” which can be generated from massive forms of these analog minerals during their handling, crushing, or processing, as occurs in mining and construction. Using current analytical methods based on light microscopy, these “cleavage fragments” are often

microscopically indistinguishable from asbestiform fibers of their asbestos mineral counterparts. The toxic potential of these mineral particles, in particular their carcinogenicity, has been an area of great controversy.

Are the specified dimensions of asbestos fibers appropriate?

Currently, a mineral particle is detected by PCM and counted as a fiber if it has a length to width (“aspect”) ratio of 3:1 and length of at least 5 micrometers. These counting rules include particles with diameters greater than 3 microns, which are unlikely to reach the airways or the gas-exchange regions of the lungs when inhaled. Also, PCM cannot detect particles with diameters less than about 0.25 micrometers, which, although not visible by PCM, are capable of causing harm. Finally, although longer fibers have been associated with greater potential for carcinogenicity, studies of fibers deposited in human tissues suggest that fibers less than 5 micrometers in length may also contribute to human disease, including cancer.

The broad goals of the research outlined in the Roadmap are to: (1) provide a scientific framework for evidence-based worker protection recommendations; (2) address the broad range of mineral fibers to which workers are exposed; and (3) refine our understanding of fiber characteristics associated with toxicity. Strategic goals identified by the Roadmap are to: (1) develop improved sampling and analytical methods for mineral fibers; (2) develop information and knowledge on occupational exposures to the range of mineral fiber types and their health outcomes; and (3) develop a broader understanding of the important determinants of fiber toxicity. In particular, it would be useful to develop approaches that would make it possible to predict the ability of various mineral fiber types to cause human disease and apply this information for risk management.

NIOSH has solicited public comment on the draft Roadmap document via docket submissions and a public meeting. The draft document was first made available to the public on February 28, 2007, and public comments were accepted into the docket from the time of posting until May 31, 2007. The public meeting was held on May 4, 2007. Peer reviewers have been selected and are being provided with a copy of the public comments as well as the draft Roadmap document. Revision of the document will take into account both public and peer review comments. The goals expressed in the Roadmap are ambitious. NIOSH plans to develop a range of partnerships to address these goals, including with other Federal agencies, labor, industry, academia, and other interested parties. Although NIOSH will focus on occupational safety and health, we will pursue opportunities to ensure that the results of research arising from the Roadmap can be extended outside of the occupational setting.

CONCLUSION

Despite the ability to prevent asbestos-related diseases by preventing exposure, they continue to be an important problem in the United States. At least in part because of the long lag in time between exposure and mortality, deaths from asbestos-related diseases such as asbestosis and mesothelioma have not yet declined. Furthermore, asbestos exposure continues to occur due to the presence of asbestos in older buildings and continued importation of asbestos-containing products from other parts of the world. Asbestiform erionite, a non-serpentine, non-amphibole mineral fiber that is well-established as having toxicity similar to asbestos, is not included within regulatory definitions that are limited to the six commercial types of asbestos. Controversy surrounds the toxic potential of several other mineral fiber types, in particular acicular/prismatic actinolite identified in El Dorado, California; and “cleavage fragments” of non-asbestiform amphibole minerals encountered especially in mining and construction. NIOSH continues to work actively in this area and has developed a draft Roadmap describing current issues and research strategies to address these issues. Working with a range of partners, our ultimate goal is to develop, disseminate, and facilitate the adoption of evidence-based recommendations to better protect workers from diseases caused by asbestos and other mineral fibers.

Thank you again for the opportunity to testify before you today. I would be happy to answer any questions you may have.

RESPONSES BY DAVID N. WEISSMAN, M.D., TO ADDITIONAL QUESTIONS
FROM SENATOR BOXER

Question 1. Your testimony states, “using current analytical methods . . . cleavage fragments are often microscopically indistinguishable from . . . fibers of their asbestos mineral counterparts.”

Please describe what that tells us about most federal definitions of asbestos, particularly given the need to protect public health from diseases associated with asbestos.

Response. Phase contrast microscopy (PCM) is a light microscopy-based method that is specified by OSHA and MSHA for use in determining the level of exposure to asbestos fibers. There is insufficient data to suggest that this method can accurately or reproducibly distinguish between asbestiform fibers of the six asbestos minerals on one hand; and "cleavage fragments" formed by handling, crushing, or processing of amphibole minerals crystallized in a massive habit on the other hand. Thus, no current practical definition of asbestos could distinguish between asbestiform fibers and cleavage fragments unless it specified the use of other analytical methods, such as electron microscopy, and provided guidance on how the analytical method would be performed and when it would be used.

The public health impact of PCM's inability to distinguish between asbestiform fibers and cleavage fragments is not entirely clear, given the uncertainties about the toxicity of cleavage fragments. Those who believe that cleavage fragments are likely to have similar toxicity as asbestiform fibers would view the inability of PCM to distinguish between them as unimportant. Those who believe that cleavage fragments have less toxicity than asbestiform fibers would take the opposite view. Specifically, they would feel that undercounting of asbestiform fibers in mixed dust would result in underestimation of risk; and over-counting of asbestiform fibers would result in over-estimation of risk. Definitive resolution of these differing viewpoints will require research to better document the ability of cleavage fragments to cause toxicity. In response to this need, CDC has nominated dusts containing a variety of mineral fibers for laboratory toxicology studies by the National Toxicology Program (NTP).

Question 2. U.S. Geological Survey data indicates this country still imports more than 2,500 metric tons of asbestos a year as well as products that contain asbestos.

Can you describe the types of diseases that may be associated with the use of these materials, and whether these diseases may be a concern for people who use these products in or around their homes?

Response. Many finished asbestos-containing products continue to be imported into the U.S. These include products such as: asbestos-cement sheets, panels, and tiles; corrugated sheets; and automotive friction products. Asbestos exposure associated with these products could potentially occur in either work or home settings. Regardless of country of origin, sufficient exposure to asbestos in either setting would be associated with the potential to develop any of the diseases caused by asbestos. A special concern for exposure in the home setting is that children can inhale asbestos, which could potentially remain within their lungs for a lifetime. Another special concern for exposures in the home is that exposures can potentially occur at any time and are not limited to a 40-hour work week. Since asbestos is a carcinogen, even low exposures are of concern for their potential to cause malignancies. An important consideration in older homes is past installation of asbestos-containing products, such as tiles, shingles, or insulation. Libby vermiculite was widely used as loose attic insulation. Installation of newer asbestos-containing materials would also be a concern.

When asbestos-containing products are identified in the home setting, steps must be taken to prevent exposure. The materials can either be removed or "managed in place." Management in place involves prevention of exposure by encapsulation of asbestos-containing materials so they cannot break down and become aerosolized, resulting in exposure of home occupants.

Senator BOXER. Thank you, sir.
Captain Miller, U.S. Public Health Service and EPA.

STATEMENT OF CAPTAIN AUBREY MILLER, M.D., M.P.H., U.S. PUBLIC HEALTH SERVICE, REGION 8, U.S. ENVIRONMENTAL PROTECTION AGENCY

Dr. MILLER. Good morning, Madam Chairman and members of the committee. I am Captain Aubrey Miller, a physician in the U.S. Public Health Service and currently a senior medical officer and toxicologist with the U.S. EPA in Denver. I am board certified in occupational medicine and have cared for patients with asbestos-related disease prior to beginning my Federal career.

Over the last 8 years I have worked for both the Department of Health and Human Services and for EPA in their Denver offices, where my efforts have been largely focused on improving our understanding of the health effects associated with asbestos exposure in Libby, MT. During this time, I have also worked toward strengthening the health care infrastructure of the Libby community and helped to establish a new community health center to provide primary care for those in need.

I have personally come to know the pain, suffering and courage of the good folks in Libby, like Les Skramsted and Mick Mills, who finally succumbed to this terrible disease. Our work is about these individuals and the countless others across America with ongoing exposures or illnesses from asbestos. Thank you for the opportunity to discuss EPA's perspective and progress in understanding the human health effects associated with exposures to asbestos.

Asbestos is a general term for fibrous silicate minerals in the serpentine and amphibole classes, and include chrysotile, amphiboles, amosite, crocidolite, antophyllite, tremolite and actinolite. Asbestos has been classified as a human carcinogen by the EPA and the World Health Organization. Mesothelioma and lung cancers are the malignancies most consistently and strongly associated with such exposures.

The non-cancerous conditions related to asbestos exposure may be more prevalent than cancer and just as debilitating and lethal. The American Thoracic Society defines non-malignant asbestos-related disease to include conditions of interstitial pulmonary fibrosis, or asbestosis, benign pleural effusions, pleural fibrosis, both circumscribed and diffuse, and obstruction of pulmonary airflow.

Asbestos diseases have a latency period ranging from 1 year to several decades, depending upon the health endpoints of concern. Once established, asbestos-related fibrosis can remain static or progress in severity in the absence of continued exposure. But they rarely regress.

As a natural mineral, serpentine and amphibole deposits may be present as natural outcroppings and can be found in native soils in a number of communities in the United States and abroad. As a result, community members can be exposed to asbestos during various activities outdoors or in their homes. Studies of communities with such environmental exposures have found health effects similar to those observed in asbestos-exposed workers. EPA is currently evaluating several sites impacted by such natural outcroppings.

There is a scientific debate concerning the differences in the extent of disease caused by different fiber types and sizes. Some of these differences may be due to the physical and chemical properties of the different fiber types. For example, several studies suggest that amphibole asbestos types may be more harmful than chrysotile, particularly for mesothelioma. Studies also indicate that fiber size dimensions, the length and diameter, are important determinants in the risk for disease.

Asbestos minerals can also occur in a non-fibrous or so-called massive form that can be found geologically in some ore deposits in which fibrous asbestos minerals also occur. Cleavage fragments, small mineral shards that are often microscopically indistinguish-

able from typical asbestos fibers, can be generated from these non-fibrous forms of asbestos minerals during crushing or processing.

Based upon scientific evidence from studies suggesting that the dimension, specifically length and diameter, as well as durability, may be more critical factors in causing disease than chemical or elemental composition, NIOSH and the Centers for Disease Control have recommended that the definition of asbestos encompass cleavage fragments from the non-fibrous forms of these minerals. EPA recognizes there is considerable controversy regarding the toxicity of fiber-like cleavage fragments. Because of this uncertainty, more work needs to be done to understand which of the many forms of asbestos or asbestos-like fibers are associated with adverse health effects. To this end, EPA is engaged in a number of activities to update and improve our understanding of the human health effects associated with asbestos exposure.

EPA is currently developing a set of toxicological and epidemiological research projects to address data gaps and scientific uncertainty regarding the health effects from exposure to the Libby amphibole and other asbestiform fibers. A more detailed description of the Agency's efforts has been included in my written testimony.

In conclusion, EPA will continue its efforts to increase our understanding on the health effects from asbestos and mineral fiber exposure. These efforts by EPA and its partners will provide needed health effects data and help inform Federal, State and local decisionmaking on how best to reduce and mitigate potential exposures.

I will be pleased to answer any questions that the committee may have on these issues. Thank you.

[The prepared statement of Dr. Miller follows:]

STATEMENT OF CAPTAIN AUBREY MILLER, M.D., MPH, U.S. PUBLIC HEALTH SERVICE,
U.S. ENVIRONMENTAL PROTECTION AGENCY

Good morning Madame Chairman and members of the committee. I am Captain Aubrey K. Miller, MD., MPH, a physician in the U.S. Public Health Service and a Senior Medical Officer and Toxicologist for the U.S. Environmental Protection Agency (EPA), Region 8 Office. In addition to my experiences prior to working for the federal government caring for patients suffering from asbestos-related disease as a Board Certified occupational physician, over the last eight years my work has been directly focused on improving our understanding of the health effects associated with asbestos exposure in Libby, Montana. Further, the early activities of my involvement, while employed in the Department of Health and Human Services (DHHS) Region 8 Office, were focused on strengthening the health care infrastructure of the Libby community to better care for those affected by this terrible tragedy. Thank you for the opportunity to discuss EPA's perspective and progress in understanding the current state of the science concerning the human health effects associated with exposure to asbestos.

DEFINITIONS OF ASBESTOS

Asbestos is a general term for fibrous silicate minerals, including minerals in the amphibole and serpentine classes. A 1971 National Academy of Sciences (NAS) report distinguished the general term "asbestos" and commercial varieties as follows:

"Asbestos" is a generic term for a number of hydrated silicates that, when crushed or processed, separate into flexible fibers made up of fibrils. [footnote omitted]. Although there are many asbestos minerals, only six are of commercial importance: Chrysotile, a tubular serpentine mineral, accounts for 95 percent of the world's production; the others, all amphiboles, are amosite, crocidolite, anthophyllite, tremolite, and actinolite. (NAS 1971).

With respect to a definition of asbestos which is most relevant to our current understanding of health effects, the Centers for Disease Control, National Institute for Occupational Safety and Health (NIOSH), in 1990 testimony before the Occupational Safety and Health Administration (OSHA), and reiterated again in 2001, broadened its science-based definition of “asbestos” as a result of concerns about the microscopic identification of the six commercial forms of asbestos minerals. The six minerals can also occur in a non-fibrous (so-called “massive”) form. The non-fibrous mineral forms of the six asbestos minerals can be found geologically in the same ore deposits in which the fibrous asbestos minerals occur or in deposits where other commercially exploited minerals are mined (e.g., industrial grade talc). “Cleavage fragments,” small mineral shards that are often microscopically indistinguishable from typical asbestos fibers, can be generated from the non-fibrous forms of the asbestos minerals during their handling, crushing, or processing, and these “cleavage fragments” are often microscopically indistinguishable from typical asbestos fibers of the (fibrous) minerals.

The elemental composition of the six asbestos minerals can vary slightly as a result of geological conditions such as pressure, temperature, or proximity of other minerals. Recognizing these variations in elemental composition, NIOSH stated that the six asbestos minerals can be defined by their “solid-solution” mineral series. For example, the mineral series tremolite-ferroactinolite contains the asbestos mineral actinolite. These mineral series are considered solid-solutions in which cations (i.e., sodium, calcium, magnesium, iron, etc.) are replaced by other cations which can affect the elemental composition of the mineral without significantly altering the structure.

NIOSH bases this expanded “asbestos” definition—encompassing the entire solid-solution mineral series for each of the six currently regulated asbestos minerals and including cleavage fragments from the non-fibrous forms of these minerals—on scientific evidence from cellular and animal studies suggesting that dimension, specifically length and diameter, as well as durability, may be more critical factors in causing disease than chemical or elemental composition [CDC 2001]. EPA recognizes that there is considerable controversy regarding the toxicity of fiber-like cleavage fragments, and additional research will help to improve understanding of important health determinants.

WHERE ASBESTOS OCCURS NATURALLY

As a natural mineral, serpentine and amphibole deposits may be present as natural outcroppings. The fibers present may exhibit a range of mineral forms and morphologies. There are many communities where these minerals are present in native soils. Community members have been exposed to elevated ambient levels of these materials in outdoor air, to materials brought into the home (e.g., fibrous clays used for interior wall coverings), and during outside activities like farming. Residents in communities exhibit health effects similar to those noted in the occupation cohorts including pleural fibrosis, asbestosis, lung cancer, and mesothelioma. These deposits in some cases include minerals which were commercially mined and milled (chrysotile and crocidolite). In addition, health effects have also been seen in communities that are exposed environmentally to actinolite, tremolite, and erionite. Erionite, which is not asbestos, represents a third class of silicate minerals, zeolites or framework silicates. EPA is currently evaluating sites impacted by natural outcroppings of silicate minerals including actinolite-tremolite, anthopholyte, chrysotile, anthopholyte and erionite.

HEALTH EFFECTS

Asbestos has been classified as “carcinogenic to humans” by EPA (1986) and as a “Class A” carcinogen by the World Health Organization. Although mesothelioma and lung cancer are the malignancies most consistently and strongly associated with such fiber exposures, cancers of the gastrointestinal tract (Jarvholm et al. 1984; Kolonel et al. 1985; Sanden, Naslund, & Jarvholm 1985), larynx (Blot et al. 1980; Burch et al. 1981; von Bittersohl 1977; Rubino et al. 1979), pancreas, (Selikoff and Seidman 1981), and ovary (Acheson et al. 1982; Wignall and Fos 1982) have also been identified. A recent review by the National Academy of Sciences Institute of Medicine concluded there was sufficient evidence to infer a causal association for laryngeal cancer; but, the evidence for pharyngeal, stomach and colorectal cancers is only suggestive, not sufficient (NAS 2006).

The noncancerous conditions related to asbestos exposure may be more prevalent than cancer and just as debilitating and lethal. Exposure to asbestos fibers via inhalation is associated with noncancer diseases to the pleura and lungs. The American Thoracic Society (ATS) recently defined nonmalignant asbestos-related disease to in-

clude the conditions of interstitial pulmonary fibrosis (asbestosis), benign asbestos-related pleural effusions, pleural fibrosis (both circumscribed fibrosis, or plaques, and diffuse fibrosis), and obstruction of pulmonary airflow (ATS Documents 2004). Rounded atelectasis, a benign form of subpleural lung collapse, has also been associated with asbestos exposure (Terra-Filho et al. 2003). Asbestos diseases have latency periods ranging from a year to several decades, depending on the health endpoint of concern. The latency varies for nonmalignant effects, from approximately a year for pleural effusion to several years for asbestosis (Cugell and Kamp 2004). Once established, asbestos-related nonmalignant interstitial and pleural disorders may remain static or progress in severity in the absence of continued exposure, but they rarely regress (Becklake 1994). Asbestos-related pleural effects are often found in individuals without occupational exposures and even asbestosis has been noted in some communities where materials may have been brought into homes (Luce et al. 2000; Luce et al., 2004; Bernardini et al. 2003; Luo et al. 2003; Baumann et al. 2007; Metintas et al. 2003).

There is a scientific debate concerning the differences in the extent of disease caused by different fiber types and sizes. Some of these differences may be due to the physical and chemical properties of the different fiber types. For example, several studies suggest that amphibole asbestos types (tremolite, amosite, and especially crocidolite) may be more harmful than chrysotile, particularly for mesothelioma. Other data indicate that fiber size dimensions (length and diameter) are important factors for cancer-causing potential. Some data indicate that fibers with lengths greater than 5.0 μm are more likely to cause injury than fibers with lengths less than 2.5 μm . (1 μm is about 1/25,000 of an inch). Additional data indicate that short fibers can contribute to injury. This appears to be true for mesothelioma, lung cancer, and asbestosis. However, fibers thicker than 3.0 μm are of lesser concern, because they appear to have less of a chance for penetrating to the lower regions of the lung. (ATSDR Tox Profile for Asbestos (2001), p. 6.)

Because of this uncertainty, more work needs to be done to understand which of the many forms of asbestos or asbestos-like fibers associated with adverse health effects require additional study. To this end, EPA is engaged in an asbestos toxicology research program.

EPA'S HEALTH ASSESSMENT ACTIVITIES FOR ASBESTOS AND SILICATE MINERAL FIBERS

EPA's Integrated Risk Information System (IRIS) database provides health assessments and tools for quantitative risk characterization which represent a consensus agency position. The current asbestos assessment was posted on IRIS in 1988 and provides cancer risk estimates based on a meta-analysis of 14 studies of workers exposed to commercial asbestos (primarily chrysotile, amosite, and crocidolite). The risk estimate represents both lung cancer and mesothelioma risk. At that time, EPA discussed many of the complexities regarding the health effects of asbestos, including: mineral form, fiber dimension, and fiber morphology. However, the exposure data available in the epidemiologic literature did not allow for refinement of the cancer risk estimate based on these factors (EPA 1986).

In 1991, the EPA published a Health Assessment on vermiculite, reviewing the studies available at that time on workers exposed to amphibole asbestos-contaminated vermiculite (Libby, MT and the Enoree region of South Carolina). The document concluded that weight of evidence for asbestos-contaminated vermiculite is sufficient to show a causal relationship for increased lung cancer in miners and millers (EPA 1991).

In preparation to update the asbestos health assessment, EPA held several conferences regarding asbestos toxicity, convening national experts on the mechanisms of fiber toxicity: "Asbestos Health Effects Conference" in 2001 and "Mechanisms of Toxicity Workshop" in 2003. In 2004 EPA initiated a health assessment focused on the noncancer effects of asbestos. In February 2006, EPA announced that it would begin a cancer health assessment for asbestos as well. In expectation of updating the cancer assessment, EPA has coordinated with NIOSH to reanalyze historical worker cohorts with state of the art exposure measurements for a key chrysotile study (Dement et al. 1994). EPA is continuing this collaboration and is working with nationally recognized experts from academia to conduct similar reanalysis, using state-of-the-art exposure measurements for key studies of workers exposed to amosite (Levin et al. 1998; Seidman et al. 1986).

As part of its ongoing activities, EPA is developing a set of research projects to assess the dosimetric and toxicologic effects of amphibole fiber-containing vermiculite ore from Libby, Montana. The objective of these projects is to address data gaps and scientific uncertainty for the quantitative characterization of health risks from exposure to the Libby amphibole and other asbestos-form fibers. The re-

search plan for these projects was initiated from the recommendations of a multi-agency meeting in January 2007 and is now being revised in response to external peer review. Funding has been approved and research is anticipated to commence by July 2007. The research involves the following assessment studies:

- Libby Amphibole RfC Development;
- Libby Amphibole Cancer Assessment;
- Fiber Size Distribution in Libby Vermiculite;
- Dosimetry Model Development and Simulation Studies;
- *In Vitro* Dissolution Assays;
- *In Vitro* Toxicity Endpoints;
- Comparative Toxicology In Mice and Rats;
- Inhalation Toxicology In Rats;
- New Epidemiologic Information From Libby, Montana and other cohorts; and
- Interim Risk Methodology For Quantification Of Cancer Risk From Inhalation Exposure to Asbestos.

EXPOSURE AND EXPOSURE MITIGATION

Over the past several years, EPA conducted research designed to reduce uncertainties in asbestos exposure scenarios. This work was a collaboration among ORD's National Exposure Research Laboratory, National Risk Management Research Laboratory, and National Health and Environmental Effects Research Laboratory. A report addressing the state-of-the-science for various exposure scenarios was completed in 2006. Additionally, a database of exposures, doses, and physical-chemical properties has been developed for more than 40 asbestos fibers. An air sampling study was also completed, as was an analysis of the Comprehensive Soil Method.

Workplace exposure mitigation practices have been in place for decades. To minimize exposure from building demolition, EPA has been working on an alternative to the National Emissions Standards for Hazardous Air Pollutants (NESHAP) method for demolition of buildings containing asbestos. Also, the California Air Resources Board and the Agency for Toxic Substances and Disease Registry (ATSDR) provide advice for limiting exposure to naturally occurring asbestos.

CLOSING

EPA will continue its efforts to increase our understanding on the health effects from asbestos and mineral fiber exposure. These efforts by EPA and those of its Federal, state, and local partners will provide needed health effects data and help inform Federal, state, and local decision making on how best to reduce and mitigate potential exposure. I will be pleased to answer any questions that the committee may have on these issues.

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Senator BOXER. Thank you very much.

Our next and last panelist is Dr. Melanie Marty, chief, Air Toxicology and Epidemiology Branch from the California EPA. We welcome you.

STATEMENT OF MELANIE MARTY, PH.D., CHIEF, AIR TOXICOLOGY AND EPIDEMIOLOGY BRANCH, CALIFORNIA ENVIRONMENTAL PROTECTION AGENCY, OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT

Ms. MARTY. Good morning, Madam Chair and members of the committee. I am a toxicologist with Cal/EPA.

My testimony today focuses on naturally occurring asbestos in California, the assessment of potential health impacts from expo-

sure and ways California is addressing exposure to naturally occurring asbestos.

Asbestos was identified as a toxic air contaminant in 1986 in California, based on the evidence that you just heard, asbestosis, lung cancer and mesothelioma in workers, and on the ubiquitous presence of asbestos in urban air due to its widespread use.

The health effects assessment conducted for the identification of asbestos as a toxic air contaminant was based on studies of workers exposed to asbestos in a number of industrial settings. We evaluated the relationship between the extent of exposure to asbestos and the subsequent development of asbestos-related disease in the workers with a focus on the cancers caused by asbestos in order to assess cancer risk from exposure to asbestos of the general population in urban air.

The workers in the occupational studies we used in our risk assessments were exposed to mixed forms of asbestos from relatively pure chrysotile to predominantly amphibole. Both types of asbestos are found naturally in the Sierra foothills and elsewhere in California and frequently together.

When asbestos fibers become airborne, they can be inhaled deep into the lung. Some are cleared by normal physiological processes, but many fibers remain in the lung tissue essentially forever. Inhaled asbestos fibers can migrate to the lining of the chest wall, the pleura, and also be transported to other organs. There is no question that asbestos is a human carcinogen. You have heard my colleagues mention that as well. It is regulated as such in the United States by OSHA, in California and other countries.

While many researchers consider the amphiboles to be substantially more potent than chrysotile in causing mesothelioma, toxicology studies in animals and human studies show that all forms of asbestos can cause mesothelioma, including chrysotile, and that further, are more or less equally potent in producing lung cancer, which accounts for the majority of the asbestos-induced cancers.

The disease that has been most well investigated in relation to exposures to naturally occurring asbestos is mesothelioma, in part because it is a rare cancer and it is strongly associated with asbestos exposure. There are many studies that describe mesothelioma in people exposed as a result of the presence of asbestos in the soil in their communities in Greece, Turkey, New Caledonia, China and elsewhere. Many but not all of the mesotheliomas in these populations were related to use of the amphibole-containing soils in the community in various ways.

Further, some studies have shown elevated mesothelioma and lung cancers in populations in close proximity to mines or asbestos factories where predominantly chrysotile asbestos-containing products were made. It is difficult to use these studies to develop quantitative estimates of risk that Californians may face from naturally occurring asbestos, but these studies heighten concern about environmental exposure.

The typical approach for assessing risk from environmental exposure is to use a long-term average concentration of the carcinogen in air. That gets difficult in the case in El Dorado County, where you have asbestos in the soil, because the exposures of concern are primarily episodic, short-term exposures to relatively high levels of

asbestos occurring from activities that release soil-borne fibers into the air, for example, driving down a dirt road or playing baseball in asbestos-contaminated soil, making it difficult to actually quantitatively assess risk. However, episodic exposures are important in view of the long time asbestos fibers can remain in the body and the cumulative nature of the injury and risk.

Also, there is general concern about exposing children to any carcinogen. Children breathe more on a body-weight basis than adults, thus experiencing higher exposures in the same setting. Cancer has a long latency between exposure and manifestation of the disease. So when exposure occurs during childhood, the risk from carcinogens, including asbestos, is higher, because there is more time to develop the disease.

Cal/EPA estimated risks from episodic exposures related to serpentine rock used for surfacing unpaved roads. The agency conducted a number of studies, measuring fibers that became airborne after vehicles drove down such roads. Furthermore, EPA Region 9 conducted activity-based sampling and showed elevated levels of airborne fibers released by soil-disturbing activities, including sports and mountain biking, running and so forth.

As a result of such investigations, the California Air Resources Board promulgated two airborne control measures designed to reduce the allowable level of asbestos in aggregate use for surfacing and to reduce dust generation during construction and grading activities. Cal/EPA also has mandates to ensure school sites are free of asbestos. Furthermore, we worked with local air districts and EPA Region 9 to educate citizens on the presence and dangers of asbestos in the soil and how they can reduce their exposures.

In closing, many studies have found mesothelioma, lung cancer and pleural abnormalities in populations exposed to naturally occurring asbestos. The presence of asbestos fibers in soil can pose elevated risks of cancer when the fibers are released into the air from activities that disturb the soil, such as construction activities, driving on unpaved roads and sports. These episodic exposures are important and mitigation measures are necessary to reduce exposure to naturally occurring asbestos.

Thank you.

[The prepared statement of Dr. Marty follows:]

STATEMENT OF MELANIE MARTY, PH.D., CHIEF, AIR TOXICOLOGY AND EPIDEMIOLOGY BRANCH, CALIFORNIA ENVIRONMENTAL PROTECTION AGENCY, OFFICE OF ENVIRONMENTAL HEALTH HAZARD ASSESSMENT

Good morning Senator Boxer and Members of the committee. My name is Melanie Marty. I am a toxicologist in the California Environmental Protection Agency and I direct the Air Toxicology and Epidemiology Branch in the Office of Environmental Health Hazard Assessment, or OEHHA. We are the Cal/EPA department mandated to assess the health risks of exposure to chemicals in our environment. My testimony today focuses on naturally occurring asbestos, or NOA, in California, the assessment of potential health impacts from exposure, and ways California is addressing exposure to NOA.

Asbestos was identified as a Toxic Air Contaminant in 1986 in California, based on the evidence that asbestos causes asbestosis, lung cancer and mesothelioma in workers, and the ubiquitous presence of asbestos in urban air due to its widespread use in brake lining, building materials and so forth.

The health effects assessment conducted for the identification of asbestos as a Toxic Air Contaminant was based on studies of workers exposed to asbestos in a number of industrial settings (such as textile and other products manufacturing). We evaluated the relationship between extent of exposure to asbestos and subse-

quent development of asbestos-related disease in the workers, with a focus on the cancers caused by asbestos, in order to assess cancer risk from exposure to asbestos in ambient air.

The workers in the occupational studies we used in our risk assessments were exposed to mixed forms of asbestos ranging from relatively pure chrysotile to predominantly amphibole. Both types of asbestos are found naturally in the Sierra foothills, frequently together.

When asbestos fibers become airborne, they can be inhaled deep into the lung. While some are cleared by normal physiological processes, many fibers remain in the lung tissue forever. Inhaled asbestos fibers can migrate from the lung to the pleura (the lining of the chest wall), and can be transported to other organs as well.

There is no question that asbestos is a human carcinogen, and it is classified as such by the International Agency for Research on Cancer, and the U.S. EPA. Asbestos is regulated as a human carcinogen by OSHA, as well as by many countries around the globe.

As you have heard from other witnesses, in occupational settings, chrysotile and amphibole asbestos exposure causes lung cancer and mesothelioma, a rare and fatal cancer of the lining of the chest wall and abdomen, and nonmalignant respiratory disease such as asbestosis. While many researchers consider the amphiboles to be substantially more potent than chrysotile in causing mesothelioma, all forms of asbestos can cause mesothelioma and are more or less equipotent in producing lung cancer, which accounts for a majority of asbestos-induced cancers. The disease that has been most well investigated in relation to exposures to naturally occurring asbestos is mesothelioma, in part because it is a rare cancer and strongly associated with asbestos exposure.

Although initial studies focused on workers, there are many studies that describe mesothelioma in people exposed as a result of the presence of asbestos in the soil in their communities in Greece, Turkey, New Caledonia, and China. Many but not all of the mesotheliomas in these populations were related to use of the amphibole-containing soils in the community in various ways. Further, some studies have shown elevated mesothelioma and lung cancers in populations in close proximity to mines or asbestos factories where predominantly chrysotile asbestos-containing products were made. I submitted a short bibliography of key papers (there are many more studies) regarding environmental exposures to asbestos and cancer as well as a copy of some of these papers for your information. While it is difficult to use these studies to develop quantitative estimates of risks that Californians may face from naturally occurring asbestos, these studies heighten concerns about environmental exposures to asbestos.

I'd like to make a few comments on the difficulties of assessing risk from exposure to naturally-occurring asbestos present in the soil. The typical approach for assessing risk from environmental exposure to airborne carcinogens is to use long-term average concentrations of the carcinogen in the air in the calculation.

But in the case of naturally-occurring asbestos in the soil, the exposures of concern are primarily episodic short-term exposures to relatively high levels of asbestos occurring from activities that release soil-borne fibers into the air, for example, while driving down a dirt road, or playing in asbestos-contaminated soil. It is difficult to determine an average air concentration to use in the typical cancer risk assessment calculation. However, episodic exposures to asbestos are important, in view of the long time asbestos fibers can remain in the body and the cumulative nature of the injury and risk.

There is general concern among scientists about exposing children to any carcinogen. Children breathe more on a body weight basis and thus experience higher doses than an adult in the same setting. Cancer has a long latency between exposure and manifestation of the disease; this is particularly true with asbestos-induced mesothelioma where there appears to be a long average latency, on the order of 30 to 40 years in most cases. When exposure occurs during childhood, as opposed to adulthood, the risk from carcinogens including asbestos is higher because there is more time to develop the disease.

Cal/EPA has tried to estimate risk from episodic exposures related to serpentine rock used for surfacing unpaved roads. The Agency conducted studies which measured asbestos fibers in the air after vehicles have driven down such roads. Any way one cuts the data, it is clear that asbestos fiber exposures are elevated, particularly very close to these roads, and the cancer risk is elevated as well. Further, USEPA Region 9 conducted activity-based sampling, measuring the airborne fibers released by soil-disturbing activities including playing baseball, riding a mountain bike or running along an unpaved trail. These measurements clearly indicate that activities that disturb the soil result in locally elevated asbestos fiber concentrations.

I would like to touch briefly on some of the mitigation measures that have been put in place in California. The California ARB promulgated an airborne toxic control measure designed to reduce the allowable level of asbestos in aggregate and other materials used for surfacing unpaved roads. The local Air Pollution Control Districts in areas with asbestos in the soil have adopted measures to reduce dust generation during construction and grading activities. The Dept of Toxic Substances Control as part of its mandate to ensure that school sites are safe to build on, evaluates sites for the presence of asbestos in the soil, and requires mitigation and maintenance of such sites to reduce as much as is practicable the exposure of children attending these schools. In addition, there has been an effort by ARB and the local air districts to educate citizens on the presence of and dangers of asbestos in the soil, and on ways they can reduce their exposures. Information including fact sheets on these activities have been submitted for your review. And finally, we have been actively working with USEPA Region 9 to evaluate exposures and risk and provide information to the public in El Dorado County about asbestos in their soil.

In closing, many studies have found mesothelioma, lung cancer, and pleural abnormalities in populations exposed to naturally occurring asbestos. The presence of asbestos fibers in soil can pose elevated risks of cancer (above background asbestos risks) when the fibers are released into the air from activities that disturb the soils. Construction activities, driving on unpaved roads surfaced with asbestos-containing rock, and other activities that people do (including sports) can elevate the concentration of airborne fibers in the immediate vicinity and expose individuals engaged in those activities to elevated fiber levels. These episodic exposures are important and increase the risk of asbestos-induced cancers to a level that is of regulatory concern. Finally, mitigation measures are necessary to reduce exposures to NOA.

Thank you for the opportunity to testify.

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CALIFORNIA GOVERNMENT WEB SITES RELATED TO NATURALLY OCCURRING ASBESTOS

1. The California EPA, Air Resources Board has a number of fact sheets and posted documents related to the identification of asbestos as a Toxic Air Contaminant and the presence of asbestos in California soils:

<http://www.arb.ca.gov/toxics/asbestos/asblinks.htm>

<http://www.arb.ca.gov/toxics/asbestos/asbestos.htm>

The following provides a link to the 1986 health effects assessment for asbestos as a Toxic Air Contaminant:

<http://www.arb.ca.gov/toxics/id/summary/summary.htm>

2. The California EPA, Office of Environmental Health Hazard Assessment has a fact sheet on asbestos health hazards:

<http://www.consrv.ca.gov/cgs/minerals/hazardous—minerals/asbestos/index.htm>
 3. The California EPA, Department of Toxic Substances control school site assessment program evaluates school sites for presence of naturally occurring asbestos as part of their program to ensure adequate protection of public health at schools. They have several documents regarding activities to reduce exposure located at the following link:

<http://www.dtsc.ca.gov/Schools/index.cfm#Environmental—Advisories—and—Guidance>

4. The California Geological Survey web site contains numerous publications regarding the presence of asbestos in California soils including maps of various areas with known asbestos in the soil.:

<http://www.consrv.ca.gov/cgs/minerals/hazardous—minerals/asbestos/index.htm>

Senator BOXER. Thank you very much.

I want to thank the panel. Let me try to cut to the chase here. I have a few questions for Dr. Weissman.

Dr. Weissman, this country still imports more than 2,500 metric tons of asbestos a year, as well as products that contain asbestos. Can you describe the types of diseases associated with the use of these materials and whether these disease may be a concern for people who use these products in or around their homes?

Dr. WEISSMAN. As we have heard, the types of diseases that are caused by asbestos exposure break into cancerous conditions and non-cancerous conditions. Among the non-cancerous conditions are asbestosis, which is a fibrosing lung disease that causes shortness of breath and impaired respiratory function. Also, asbestos can damage the pleura, which is the tissue lining the surfaces of the lung and chest wall, resulting in fibrosis. It can either by plaques or more extensive fibrosis that can constrict the lungs.

From the side of carcinogenic effects, lung cancer, mesothelioma, there are also associations with cancer of the larynx and cancers of the gastrointestinal tract.

With regard to the impact of continued exposure to asbestos, in protecting workers, there is something called the industrial hygiene hierarchy of controls, which is the approach to reducing exposure to reduce disease. Really the No. 1 best way to reduce disease caused by a hazardous exposure is to eliminate the exposure. The No. 2 thing that we think about is whenever possible substituting for other products that are less hazardous. Then we get into other kinds of controls, like engineering controls and respirators.

Senator BOXER. So limiting exposure would certainly be achieved if we were to, I am not asking your opinion on this, stop the importation so the products wouldn't have it, that would limit the exposure, obviously, to the products that were still on the market. So I ask you specifically if it would impact people around the home, who might be exposed. I am assuming you would say yes. You are not making a distinction between workers and people in their homes. You are saying if you are exposed to it, it could be a problem, is that correct?

Dr. WEISSMAN. That is correct.

Senator BOXER. OK. I just wanted to note, I am taking this from the USGS Minerals Yearbook, the importations include corrugated cement sheet, flat cement panel sheet, cement pipe, tube and pipe fittings, other cement products, yarn and thread, cord and string, woven or knitted fabric, articles for us in civil aircraft gaskets, other building materials, brake lining and pads, mounted brake lin-

ings for tractors. I am going to put this into the record without objection.

[The referenced material follows:]



2005 Minerals Yearbook

ASBESTOS

ASBESTOS

By Robert L. Virta

Domestic survey data and tables were prepared by Subina W. Pandey, statistical assistant, and the world production table was prepared by Regina R. Coleman, international data coordinator.

Asbestos has not been mined in the United States since 2002 and imports, mostly from Canada, satisfied domestic manufacturing needs. U.S. apparent consumption declined to 2,530 metric tons (t) in 2005. World production was 2.40 million metric tons (Mt), an increase from 2.36 Mt in 2004.

Legislation and Government Programs

The Agency for Toxic Substances and Disease Registry (ATSDR) and the National Institute for Occupational Safety and Health (NIOSH) of the U.S. Department of Health and Human Services and the U.S. Environmental Protection Agency (EPA) continued with their exposure and health studies of residents and former vermiculite miners and millers in Libby, MT. The EPA continued its cleanup of asbestos-contaminated properties in Libby and conducted studies of asbestos exposure in El Dorado County, CA. The ATSDR continued to review health statistics for communities near vermiculite exfoliation plants located throughout the United States. The NIOSH continued its study of workers formerly employed at a South Carolina textile plant and to develop a model to better predict risk to humans from fiber exposure for which dose response data are not available (U.S. Environmental Protection Agency, 2005[§]).

The Mine Safety and Health Administration continued to evaluate its proposed reduction of the 8-hour time-weighted average permissible exposure level to 0.1 fiber per cubic centimeter (f/cm³) from 2 f/cm³ for asbestos. A decision was scheduled for July 2006 (U.S. Department of Labor, 2005[§]).

Consumption

U.S. consumption of asbestos was 2,530 t in 2005, a decrease from 3,450 t in 2004 (table 1). Roofing products accounted for 55% of U.S. consumption; coating and compounds, 26%; other uses, 19%; and electrical insulation, less than 1%. Chrysotile was the only type of asbestos used in the United States. About 73% of the chrysotile used in the United States in 2005 was grade 7; the rest was grades 5, 4, and 3, in descending order of percentage (table 2).

Prices

The average free alongside ship (f.a.s.) unit value of asbestos fiber exports and reexports was \$263 per metric ton in 2005, an increase from \$211 per ton in 2004. The average U.S. customs unit value for all grades of imported asbestos increased to \$561 per ton in 2005 from \$234 per

ton in 2004. The average value of imported crude chrysotile increased to \$188 per ton in 2005 from \$46 per ton in 2004 because of unusually low-value transshipments through Germany in 2004. The average unit value for imports of spinning-grade chrysotile from all sources was \$150 per ton in 2005, unchanged from 2004. The unit value of other grades of chrysotile from all sources was \$600 per ton, an increase from \$318 per ton in 2004. This resulted because larger quantities of higher valued chrysotile were imported from Canada and transhipped through South Africa in 2005 than in 2004 (table 6). Average prices for chrysotile imported from Canada, which composed 88% of U.S. asbestos imports, are given in table 3.

Foreign Trade

Imports of asbestos products and products manufactured using asbestos substitutes are reported under the same Harmonized Tariff Schedule of the United States (HTS) codes (U.S. Census Bureau, 2001[§]). With the decline in use of asbestos products in the United States and bans on the manufacture of asbestos products in many other countries, it is likely that products manufactured using asbestos substitutes account for a significant portion of the product imports under some HTS categories. This fact must be taken into consideration when evaluating the trade data that follow.

The f.a.s. value of exported asbestos fibers increased to \$398,000 in 2005 from \$333,000 in 2004. Mexico was the leading importer of asbestos fiber from the United States. Canada was the leading importer of U.S. products manufactured using asbestos or asbestos substitutes, followed by Mexico, Japan, Thailand, Germany, the Republic of Korea, the United Kingdom, Saudi Arabia, Australia, and Brazil. These 10 countries accounted for 79% of the value of manufactured products reexported from the United States in 2005 (table 4).

In 2005, approximately 1,510 t of asbestos was exported (U.S. International Trade Commission, 2005[§]). The exports included asbestos crude, fiber, refuse, sand, and stucco. There has been no U.S. production since 2002, so exports were either from stockpiles or reexports of imported fiber (table 5).

Brake linings, clutch linings, disk pads, mounted brake linings manufactured using asbestos, other mineral substances, or cellulose accounted for 86% of the value of manufactured products that were exported or reexported in 2005 (table 5). Products in these categories composed more than 79% of the value of exports to each of the countries specified in table 4.

In 2005, Canada supplied 88% of the asbestos imported by the United States. Asbestos also was imported from Zimbabwe (table 6). Only chrysotile was imported into the United States in

[§]References that include a section mark (§) are found in the Internet References Cited section.

2005. Based on the import source, asbestos listed under "Other, unspecified asbestos type" in table 6 probably was chrysotile.

The United States also imported \$579 million worth of products with a basis of asbestos, asbestos and magnesium carbonate, cellulose fiber, or other mineral substances (U.S. International Trade Commission, 2005§) (table 7).

World Review

World production of asbestos was estimated to be 2.40 Mt in 2005, an increase from 2.36 Mt in 2004. Russia continued to be the leading producer of asbestos, followed by China, Kazakhstan, Canada, Brazil, and Zimbabwe. These countries accounted for 96% of the world production (table 8).

Outlook

Domestic use of asbestos probably will continue its downward trend in the United States because of liability issues. World production has remained relatively unchanged since 2002 and probably will remain between 2.3 and 2.4 Mt for the next couple of years.

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TABLE 1
SALIENT ASBESTOS STATISTICS¹

	2001	2002	2003	2004	2005
United States:					
Production, sales	metric tons	5,260	1,720	--	--
Exports and reexports:²					
Unmanufactured, value	thousands	\$4,890	\$2,020	\$920	\$333
Asbestos products, value	do.	\$298,000	\$203,000	\$290,000	\$341,000
Imports for consumption, unmanufactured:					
Quantity	metric tons	13,100	6,850	4,650	3,450
Value ³	thousands	\$2,640	\$1,770	\$5,840	\$806
Consumption, apparent ⁴	metric tons	13,100	6,850	4,650	3,450
World, production	do.	2,060,000 ¹	2,320,000 ¹	2,360,000 ¹	2,360,000 ¹
				2,360,000 ¹	2,400,000 ¹

¹Estimated. ²Revised. -- Zero.

³Data are rounded to no more than three significant digits.

⁴Free alongside ship value; includes exports of crudes, fibers, stucco, sand, and refuse. May also include nonasbestos materials.

⁵U.S. customs declared value

⁶Production plus imports minus producer exports of asbestos fiber plus adjustments in Government and industry stocks.

TABLE 2
U.S. ASBESTOS CONSUMPTION BY END USE, GRADE, AND TYPE^{1,2}

(Metric tons)

End use	Chrysotile						Total
	Grade 3	Grade 4	Grade 5	Grade 6	Grade 7	Unspecified grade	
2004	--	29	240	--	1,990	1,190	3,450
2005:							
Coatings and compounds	--	--	293	--	367	--	660
Electrical insulation	11	--	--	--	--	--	11
Roofing products	--	--	--	--	1,380	--	1,380
Other	10	84	--	--	87	298	479
Total	21	84	293	--	1,840	298	2,530

-- Zero.

¹Data are rounded to no more than three significant digits; may not add to totals shown.²Estimated distribution based upon data provided by the Chrysotile Institute, Montreal, Quebec, Canada.

TABLE 3
CUSTOMS UNIT VALUE OF IMPORTED ASBESTOS

(Dollars per metric ton)

	2004	2005
Canada, chrysotile:		
Crude	193	188
Spinning	157	170
Other	213	334

Source: U.S. Census Bureau.

TABLE 4
VALUE OF U.S. EXPORTS AND REEXPORTS OF ASBESTOS FIBERS AND PRODUCTS^{1,2}

(Thousand dollars)

Country	2004			2005		
	Unmanufactured fiber ³	Manufactured products ⁴	Total	Unmanufactured fiber ³	Manufactured products ⁴	Total
Australia	--	3,470	3,470	--	4,060	4,060
Brazil	--	4,760	4,760	--	3,800	3,800
Canada	--	169,000	169,000	--	177,000	177,000
Germany	--	9,180	9,180	--	10,100	10,100
Japan	--	15,100	15,100	--	15,500	15,500
Korea, Republic of	--	11,500	11,500	--	9,450	9,450
Kuwait	--	999	999	--	1,150	1,150
Mexico	317	44,200	44,500	347	46,300	46,600
Saudi Arabia	--	5,040	5,040	--	7,160	7,160
Thailand	--	13,000	13,000	--	12,000	12,000
Turkey	--	129	129	--	140	140
United Kingdom	--	7,360	7,360	--	8,780	8,780
Venezuela	--	1,410	1,410	--	1,590	1,590
Other	16	56,300	56,300	51	76,900	77,000
Total	333	341,000	342,000	398	374,000	375,000

See footnotes at end of table.

TABLE 4—Continued
 VALUE OF U.S. EXPORTS AND REEXPORTS OF ASBESTOS FIBERS AND PRODUCTS^{1,2}

-- Zero.

¹Data are rounded to no more than three significant digits; may not add to totals shown.

²Free alongside ship value.

³Includes exports of crudes, fibers, stucco, sand, and refuse. May also include nonasbestos materials.

⁴Includes products manufactured using asbestos, cellulose fiber, and other asbestos substitutes.

Source: U.S. Census Bureau.

TABLE 5
 U.S. EXPORTS AND REEXPORTS OF ASBESTOS AND ASBESTOS PRODUCTS¹

	2004		2005	
	Quantity (metric tons)	Value ² (thousands)	Quantity (metric tons)	Value ² (thousands)
Unmanufactured, asbestos³	1,380	\$333,000	1,510	\$398,000
Manufactured:				
Brake linings and disk brake pads ⁴	NA	275,000	NA	293,000
Clutch facings and linings ⁵	NA	23,300	NA	28,600
Clothing, cord, fabric, yarn	NA	2,110	NA	--
Gaskets, packing and seals	NA	1,810	NA	1,480
Panel, sheet, tile, tube ⁶	NA	27,700	NA	39,700
Paper and millboard	NA	860	NA	983
Other articles ⁷	NA	10,200	NA	10,700
Total	NA	341,000	NA	374,000

-- Zero. NA Not available.

¹Data are rounded to no more than three significant digits; may not add to totals shown.

²Free alongside ship value.

³Includes crudes, fibers, stucco, sand, and refuse. May also include nonasbestos materials.

⁴Includes asbestos and cellulose fiber brakes and similar materials.

⁵Includes clutches and other friction materials, excluding brakes and brake pads.

⁶Includes asbestos cement and cellulose fiber cement products.

⁷Includes asbestos and cellulose fiber products.

Source: U.S. Census Bureau.

TABLE 6
 U.S. IMPORTS FOR CONSUMPTION OF ASBESTOS FIBERS, BY TYPE AND ORIGIN¹

Type	Canada		South Africa		Other		Total	
	Quantity (metric tons)	Value ² (thousands)	Quantity (metric tons)	Value ² (thousands)	Quantity (metric tons)	Value ² (thousands)	Quantity (metric tons)	Value ² (thousands)
2004:								
Chrysotile:								
Crude	254	\$49	--	--	1,020	\$10	1,270	\$59
Spinning fibers	20	3	--	--	--	--	20	3
All other	1,650	258	122	\$239	45	82	1,820	579
Other, unspecified asbestos type	334	165	--	--	--	--	334	165
Total	2,260	475	122	239	1,060	92	3,450	806

See footnotes at end of table.

TABLE 6—Continued
U.S. IMPORTS FOR CONSUMPTION OF ASBESTOS FIBERS, BY TYPE AND ORIGIN¹

Type	Canada		South Africa		Other		Total	
	Quantity (metric tons)	Value ² (thousands)	Quantity (metric tons)	Value ² (thousands)	Quantity (metric tons)	Value ² (thousands)	Quantity (metric tons)	Value ² (thousands)
2005:								
Chrysotile:								
Crude	288	54	--	--	--	--	288	54
Spinning fibers	20	3	--	--	--	--	20	3
Milled, grade 4	--	--	32 ³	157 ³	266	562	298	719
All other	1,510	366	--	--	--	--	1,510	366
Other, unspecified asbestos type	417	278	--	--	--	--	417	278
Total	2,240	701	32³	157³	266	562	2,530	1,420

-- Zero.

¹Data are rounded to no more than three significant digits; may not add to totals shown.

²U.S. customs declared value.

³These are probably transshipments of chrysotile from Zimbabwe through South Africa.

Source: U.S. Census Bureau.

TABLE 7
U.S. IMPORTS OF PRODUCTS WITH BASIS OF ASBESTOS, CELLULOSE, OR OTHER MINERALS IN 2005

HTS ¹ code	Category	Quantity (metric tons)	Value	Major sources ²	Percentage of category total ³
2524.00.00.00	Asbestos	2,530	\$1,420,000	Canada	88% of weight.
6811.10.00.00	Corrugated cement sheet ⁴	705	421,000	Finland ⁵ , Denmark ⁵	80% of weight.
6811.20.00.00	Flat cement panel, sheet, and tile ⁴	91,400	38,200,000	Mexico, Canada, Chile, Malaysia	94% of weight.
6811.30.00.00	Cement pipe, tube, and pipe fittings ⁴	353	171,000	Mexico	99% of weight.
6811.90.00.00	Other cement products ⁴	3,070	2,300,000	Japan	88% of weight.
6812.50.00.00	Fabricated asbestos fibers; clothing ⁶	9	50,500	Denmark ⁵	82% of value.
6812.60.00.00	Felt, millboard, and paper	NA	9,230	Denmark ⁵ , Japan	100% of value.
6812.70.00.00	Compressed asbestos fiber jointing	NA	694,000	Canada	87% of value.
6812.90.01.01	Other, miscellaneous ⁶	1	5,070	China	100% of value.
6812.90.01.02	Yarn and thread ⁶	99	437,000	Mexico	100% of value.
6812.90.01.03	Cord and string ⁶	1	30,500	Japan, Taiwan, Germany	83% of value.
6812.90.01.04	Woven or knitted fabric ⁶	50	460,000	South Africa	99% of value.
6812.90.01.10	Articles for use in civil aircraft ⁶	NA	16,700	Japan, United Kingdom ⁵ , Germany ⁵ , France	100% of value.
6812.90.01.20	Gaskets, packing, and seals ⁶	154	934,000	India, Japan	60% of value.
6812.90.01.25	Other, building materials ⁶	NA	144,000	Australia	98% of value.
6812.90.01.55	Other, fabricated asbestos fiber ⁶	NA	130,000	France, United Kingdom ⁵	74% of value.
6813.10.00.10	Brake lining and pads, civil aircraft ⁷	NA	3,600,000	France ⁵ , United Kingdom ⁵	95% of value.
6813.10.00.50	Brake lining and pads, other ⁷	NA	110,000,000	Brazil, China, Canada	71% of value.
6813.90.00.10	Other, articles, civil aircraft ⁷	NA	332,000	United Kingdom ⁵	92% of value.
6813.90.00.50	Other, friction materials ⁷	NA	25,700,000	United Kingdom ⁵ , Japan, Mexico	84% of value.
8708.31.00.00	Mounted brake linings for tractors	NA	601,000	Germany ⁵ , Taiwan	52% of weight.
8708.31.50.00	Mounted brake linings, other	NA	395,000,000	Japan ⁵ , Canada	60% of weight.

NA Not available.

¹Harmonized Tariff Schedule of the United States.

²Countries are listed in decreasing order.

³Percentage contribution of total imports by major import sources, by weight or value.

⁴Articles of asbestos-cement, of cellulose fiber-cement or the like.

⁵Source likely a supplier of nonasbestos products only.

⁶Mixtures with basis of asbestos or with a basis of asbestos and magnesium carbonate.

⁷Articles with a basis of asbestos, of other mineral substances, or of cellulose.

Source: U.S. Census Bureau.

TABLE 8
ASBESTOS: WORLD PRODUCTION, BY COUNTRY^{1,2}

(Metric tons)

Country ³	2001	2002	2003	2004	2005 ⁴
Argentina	203	155	166	267 ¹	270
Brazil, fiber	172,695	194,750	194,350	194,800	195,000
Bulgaria ⁴	350	300	300	300	300
Canada	276,790	240,500	200,500	200,000 ⁴	200,000
China ⁴	310,000	562,000 ⁴	500,000 ⁴	510,000 ⁴	520,000
Colombia, crude ore	96,140	62,785	60,000 ⁴	60,000 ⁴	60,000
Egypt ⁴	-- ¹	-- ¹	-- ¹	-- ¹	--
India ⁴	21,000	18,000	19,000	18,000	19,000
Iran ⁴	2,000	1,500	1,470 ^{1,4}	6,000 ^{1,4}	5,000
Japan ⁴	-- ¹	-- ¹	-- ¹	-- ¹	--
Kazakhstan	271,300	291,100	354,500	346,500	355,000
Russia ⁴	750,000	775,000	878,000 ⁴	923,000 ^{1,4}	925,000
Serbia and Montenegro	194	372	111 ¹	110 ¹	100
South Africa, chrysotile	13,393	--	6,218 ¹	-- ⁴	-- ⁴
United States, sold or used by producers	5,260	2,720	--	--	--
Zimbabwe	136,327	168,000 ⁴	147,000	104,000 ¹	122,041 ⁴
Total	2,060,000⁴	2,320,000⁴	2,360,000¹	2,360,000¹	2,400,000

¹Estimated. ⁴Revised. -- Zero.²World totals, U.S. data, and estimated data are rounded to no more than three significant digits; may not add to totals shown.³Marketable fiber production. Table includes data available through April 8, 2006.⁴In addition to the countries listed, Afghanistan, North Korea, Romania, and Slovakia also produce asbestos, but output is not officially reported, and available general information is inadequate for the formulation of reliable estimates of output levels.⁵Reported figure.

Senator BOXER. I guess as I listen to you, and I so appreciate the panel, we had some questions from Senator Vitter and raised by my Ranking Member on a certain type of product that perhaps that is a safe form. I wanted to ask you about chrysotile. What I want to know is whether exposure to this type of asbestos has adverse health impacts.

Dr. WEISSMAN. Yes.

Senator BOXER. What would they be?

Dr. WEISSMAN. Yes, chrysotile asbestos is hazardous and has the same health impacts as other types of asbestos. There is a debate in the literature over the potency, over whether you need the same dose of chrysotile to cause one of the health effects, which is mesothelioma. But it certainly causes all of the same health effects.

Senator BOXER. So you would agree with Dr. Marty, who made that point. And I see that Dr. Miller is shaking his head.

OK. I guess my last question is this. In a lot of these issues, the more vulnerable populations are more at risk. Have you found that in this whole thing of asbestosis? For example, if a worker comes home and hugs a child, and he had asbestos fibers on him, have you had any studies that indicate that the more vulnerable populations would be more apt to get sicker earlier, or have there been no studies of that?

Dr. WEISSMAN. We don't have any information from NIOSH about take-home exposures of families. I would defer to Dr. Miller on that.

Senator BOXER. Dr. Miller or Dr. Marty, either.

Dr. MILLER. With respect to that, our concern is certainly that children, being exposed at an earlier age, at a minimum would have a longer period of life to express disease. Those fibers get into their lungs, they are durable, they are going to stay in their lungs and they will have that.

Another part of that concern is, are children just more susceptible, at the developmental time of their life.

Senator BOXER. That is why I asked the question. I wrote a bill called The Children's Environmental Health Protection Act. It makes the point that when we set standards for anything, when we do laws about anything, we have to focus on the children, because they are developing and it may be more dangerous.

Dr. Marty, do you have any comment on that, exposure to most vulnerable, like our children?

Ms. MARTY. Yes. I think for children, we also have to recognize that they probably have higher exposures in the same setting as an adult, simply because they breathe more on a body weight basis and particle deposition appears to be higher. This would probably be the same for fiber in a child's lung than in an adult's lung.

Senator BOXER. So it is more a proportion of their body, because of their size. I think the point that Dr. Miller makes is important as well, that because they would be exposed at this early age, the disease would take shape at a younger age than an adult.

Ms. MARTY. Yes.

Senator BOXER. So if an adult is exposed at age 30, it may take how many years on average to get mesothelioma?

Ms. MARTY. Thirty, approximately.

Senator BOXER. But if it is a child, they could die at 36 or 40.

Ms. MARTY. And there are definitely case reports in the literature of children being exposed from take-home exposure or from environmental exposure and having mesothelioma at a relatively young age.

Senator BOXER. I think, colleagues, this is a really important point, that when we ban asbestos and we ban materials like asbestos, we are really protecting the children, in addition to everybody else.

Senator Vitter.

Senator VITTER. I just wanted to ask all the panelists their impression or summary of the science on specifically chlor-alkali production.

Dr. WEISSMAN. I don't have any comments on that. I come at it purely from my expertise as a physician. And as a physician, any potential exposure creates the potential for disease. But as to the criticality of use of asbestos in the process and the viability of alternatives, it is outside of my expertise.

Dr. MILLER. I don't have specific knowledge of that production. But concerns would be the production, processing, transportation of these materials and the disposal of it. While it may be controlled in the work environment, and that can be done with a lot of toxic substances we deal with, our concerns with this situation, as with other asbestos products in commerce would be the concerns of how it is handled, what is done with it and who may be exposed outside of those controlled conditions.

Ms. MARTY. In other words, someone mined it, someone milled it, someone packed it in bags before it ever got into the diaphragm.

Senator BOXER. Senator, do you have any further questions?

Senator VITTER. No, I am fine.

Senator BOXER. OK, thank you.

Senator Lautenberg.

Senator LAUTENBERG. Thank you, Madam Chairman.

I may have defined the disease incorrectly in my earlier statement. I used the term asbestosis. I kind of thought that was a coverall for all forms of the illness derived from exposure. But the people who came to see me, the family I described, who, three of them were terminally ill, it was mesothelioma. And a very close personal friend of mine was a physician named Dr. Irving Selikoff. He had a practice in Patterson, NJ, where I earlier described the fact that high school mates of mine worked in an asbestos factory and suffered some terrible results as a consequence of that very short exposure. As I hear you talk, the latency period suggests that there is a time bomb in the body of these folks who have been exposed. And when it is going to go off, we are not sure. But we are sure of one thing, that it is going to explode.

I would ask you this, Dr. Miller. Prevention is the ideal program to avoid this. Is treatment available for mesothelioma or related lung disease?

Dr. MILLER. Just going back to the first element of your question, we certainly see asbestos exposures, low exposures, resulting in disease of great concern. As a matter of fact, we have been working closely with Dr. Selikoff at Mount Sinai on research on this. They had actually done work at Patterson and looked at the households, the home contacts of these workers, and even people that worked

there for just a short time and looked at the household contacts of those workers. They found that there was very high elevation of disease in these folks in their homes, just as a result of the workers' contamination, and bringing it home.

They even looked at some folks that were either born in the house or came into the house subsequent to the workers' stopping, with just that residual contamination. It would suggest that residual contamination of asbestos in their home is producing this, not even having an active worker coming in and out and shaking off their clothes and washing them. So that is of great concern.

Certainly the efforts are to prevent this, to prevent these diseases from occurring. I am not familiar with the current treatments of mesothelioma, and I wouldn't be the best to try and comment on that. I am not sure if my colleagues here could do that.

Senator LAUTENBERG. Do either one of our friends at the table have any comments on treatment possibilities?

Dr. WEISSMAN. The bottom line is that the pulmonary fibrosis caused by asbestos exposure, asbestosis, the treatment is only symptomatic. There is no treatment for the underlying process. That is also the case for diffuse pleural thickening, the pleural fibrosis that constricts the lungs, and of the treatment results for the cancers that are caused by asbestos are dismal.

Senator LAUTENBERG. It is essentially a death sentence if exposed.

Dr. Miller, based on your work at EPA, is there any safe level of exposure to asbestos that would not cause disease to follow?

Dr. MILLER. Thank you for the question, Senator. Asbestos is one of the first diseases I studied on entering occupational health and it is one that I thought we had resolved. A situation like Libby, MT came up and caused me to go back in and try to reevaluate this and look at the evidence that is available. Asbestos was first described to cause disease back in 50 A.D., by Pliny the Elder. So the fact is, we have been struggling with this for a long, long time.

The issue of what is safe, to our understanding there is no safe level that has been identified. The more you are exposed to asbestos, the more it increases your risk for disease. The fact is, we have seen disease, while fairly rare, resulting from people that had relatively inconsequential exposures, very short exposures, children of a parent that worked in an asbestos factory for a short time.

So at this point in time, we do not know of a safe level with respect to asbestos.

Senator LAUTENBERG. Madam Chairman, may I take 1 more minute?

Senator BOXER. Yes.

Senator LAUTENBERG. I would ask this, also. Dr. Miller, last week a New Jersey school was forced to close its doors because asbestos was found in one of the classrooms. Are there Federal resources available to assist State and local school districts in helping to prevent exposure as a result of that condition?

Dr. MILLER. I can't comment entirely. I know that EPA has been involved in a number of situations in providing technical assistance and certainly I personally provide technical assistance to schools, as you have mentioned, with respect to trying to do appropriate

testing and evaluation and assisting with discussions about appropriate remediation.

So as far as active programs from the Federal side, I think it mostly resides in the domain of technical assistance in trying to help folks evaluate these situations and provide technical assistance in that respect.

Senator LAUTENBERG. Thank you. Thank you, Madam Chairman.

Senator BOXER. Thank you. Senator Carper.

Senator CARPER. Thank you. My thanks to our witnesses for coming this morning.

I missed your testimony. I am going to ask you a question, I will ask you all the same question. I apologize for missing your testimony. But I just want you to take maybe a minute apiece and give me what you think should be our takeaway from your testimony. If we remember nothing else of what you said, what might that be?

I understand, while they are thinking about that, I understand, Madam Chair, that Senator Murray is moving forward with the legislation. I think that is good. I understand that they are making at least one modification with respect to production of chlorine and trying to model it after what they are doing in the European Union. I think that makes sense. I understand that there is some issue maybe involving last year's definition involving common rocks to try to make an accommodation there. Going back to last year's definition, I think if those two changes are made, I think we have a bill that is going to roll right out of here and get to the Senate floor and through the Congress.

With that having been said, let me ask of our panelists, any takeaway you would like to share with me? Let's start with Dr. Marty, if we could.

Ms. MARTY. I think the upshot is that all forms of asbestos cause asbestos-related disease, chrysotile, amphiboles, and even things that aren't quite called asbestos, at least yet. Environmental exposures are a concern. We have studies across the world showing an epidemiological way that mesothelioma incidence is elevated in populations that have naturally occurring asbestos in their soil and we must reduce exposures as much as is practicable.

Senator CARPER. All right, thank you.

Captain? I used to be a captain in the Navy.

Dr. MILLER. I think it is important with respect to this issue, a lot of what we have been focusing on has kind of been defined by mineralogists and by techniques that have been available, older techniques that were available at the time we started into this. And what we really want to focus on is capturing those fibers which are causing illness and not being limited by either antiquated methods or older understandings of disease and exposure.

So with that, I really want, I guess, to further the understanding of what are the fibers, what are the minerals that are causing this problem. There are things about the fibers we measure, that we measure a certain sector. As Dr. Weissman mentioned, these phase contrast optical microscopy fibers. But we know that there are fibers outside of that phase contrast optical microscope, looking at things under a microscope versus a big microscope, a TEM microscope, which we use and have been using in our environmental situation in Libby and across the country.

So to use this, we see a lot more. Our understanding is there are a lot of these fibers that have toxic effects. As a matter of fact, the shapes and what the fibers look like, I believe Senator Inhofe provided a diagram of pictures of rocks and fibers. Rocks and fibers, it is not one or the other. They run across a gamut. They have different sizes and shapes, from one extreme of being a willowy looking fiber to another extreme of being kind of a short, stubby, rock-like material.

Senator CARPER. Captain, I asked for a 1-minute takeaway. I want to make sure Dr. Weissman gets to speak. So finish up.

Dr. MILLER. So in between, these fibers all have health effects that we need to be concerned about and captured in whatever efforts we make to control and ban asbestos.

Senator CARPER. Thank you.

Dr. Weissman?

Dr. WEISSMAN. Senator, I have three big points. First, there are still a lot of people getting disease because of past exposures. We need to think about them.

Second, even at our permissible exposure limit for asbestos, there is still an appreciable, detectable risk of developing diseases, including lung cancer. It is a very hazardous thing to be exposed to.

Third, and finally, we need research. We need better exposure assessment methods that include all of the hazards, that count all of the hazardous fibers to which people are exposed, not just those that we can see under a light microscope. And we need to understand the toxicities of all the different fiber types and drive our public policy based on that. So there is still room to do better.

Senator CARPER. Good. Thank you very much for that response. My thanks to all of you and thank you, Madam Chair.

Senator BOXER. Let me thank the panel. For my takeaway, Senator Carper, I took away that there is no safe level of exposure and the kids are the most vulnerable.

In terms of people still getting sick, I thank you, Dr. Weissman, because Patty Murray's bill does get help to those people. I think that is a very important point.

We thank this panel. You have been just terrific, thank you very much.

We invite our last panel—oh, I forgot that we have been joined by the wonderful Senator Klobuchar, who I missed, even though I shook her hand on the way in. I am sorry. Senator, you are welcome to sit over here. The floor is yours, you can use it either for questions or an opening statement. You have 5 minutes.

Senator KLOBUCHAR. I just have a few quick questions. That is of you, Dr. Weissman, first, and about your work with NIOSH. You talked about the need for research. So I was wondering what type of monitoring and tracking system NIOSH has in place now for asbestos-related diseases?

Dr. WEISSMAN. It is not a perfect system by any means. The primary stream of data that we rely upon for surveillance is mortality data, which is based on death certificates. Death certificates are well known to incompletely capture all of the cases of disease. In addition, since 1999, the electronic data bases that are abstracted from death records don't include information about usual occupation and usual illness.

So we can track things that are tightly related to asbestos exposure, like asbestosis and mesothelioma. But things like lung cancer that have a background in the population can be caused by other things than asbestosis. We have some trouble tracking that. But death data is really the main thing that we have to work with.

Senator KLOBUCHAR. So do you think there is potential for under-reporting of asbestos-related illnesses, then?

Dr. WEISSMAN. That is right, because we depend upon the way that people fill out death certificates. It is well known that death certificates under-report.

Senator KLOBUCHAR. And you were talking about how there are more cases being diagnosed from the past, people have gotten this from the past. Is there a large amount of asbestos-related product still in existence in the United States?

Dr. WEISSMAN. Yes. There are reservoirs of asbestos in older buildings where the asbestos is being managed in place but can still be encountered when buildings are demolished or renovated. Then there is still important of asbestos-containing products.

Senator KLOBUCHAR. What kinds of products are those?

Dr. WEISSMAN. Things like automotive friction products, cement products that contain asbestos.

Senator KLOBUCHAR. Does better equipment, I guess I would ask all of you this, like ventilators or some kind of personal dust respirators, does that result in lower illness? Have any of you looked into this?

Dr. WEISSMAN. Respirators are considered in one of the, in what we call the industrial hygiene hierarchy. They are the least preferred method of control, because even if someone wears them, they might not work. Then also, people have to wear them whenever they are exposed, and they might not always know when they are exposed.

So respirators are the least preferred method to protect people.

Senator KLOBUCHAR. Do you want to add anything to that?

Dr. MILLER. I think with respect to protection, NIOSH-approved personal protection equipment and controls are the most appropriate.

Senator KLOBUCHAR. I have heard, I think one of the other witnesses who is going to testify talked about the need to better coordinate with States to improve the surveillance of fiber-related illnesses. In fact, I guess I would ask you as well, Dr. Weissman, about how NIOSH, OSHA, both of them are currently coordinating with State governments to address asbestos-related diseases?

Dr. WEISSMAN. NIOSH has an activity with the States to do State-based surveillance for occupational diseases. So we have a granting program. Not every State has a grant under that program. But it supports State-based surveillance for diseases. Different States have somewhat different portfolios of what they monitor for. But that is the main NIOSH interaction with States in terms of surveillance.

In terms of OSHA, our interaction is largely from hazard surveillance. OSHA, under an agreement with NIOSH, provides us with their compliance data and allows us to track levels of exposure, which appears in our surveillance report that we put out at intervals.

Senator KLOBUCHAR. Thank you very much.

Senator BOXER. Senator Inhofe said he had no questions for this panel. So we are going to thank you again, and I am sorry, Senator Klobuchar. I am so pleased that you are here and to have you on this committee is just so fortunate for America.

Senator KLOBUCHAR. Thank you very much. I am glad you are here, too.

[Laughter.]

Senator BOXER. Now we will call up our third panel, or our second panel but third group of witnesses. Barry Castleman, Sc.D., Environmental Consultant; Ann Wylie, Ph.D., University of Maryland, Department of Geology; David Weill, M.D., director, Lung and Heart-Lung Transplant Program, Stanford School of Medicine; Richard Lemen, Ph.D., M.S.P.H., former director of Division of Standards Development and Technology Transfer at NIOSH, Assistant Surgeon General, U.S. Public Health Service, retired; and Linda Reinstein, executive director and co-founder, of the Asbestos Disease Awareness Organization.

We will start with Dr. Castleman, an environmental consultant. We will ask each of you to speak for 5 minutes. We will put your full statement into the record and then we will start with questions.

Senator INHOFE. Madam Chairman?

Senator BOXER. Yes.

Senator INHOFE. Let me do a U.C., here, first if I could.

Senator BOXER. Of course.

Senator INHOFE. We received just yesterday a letter from the El Dorado County Office of Education regarding this hearing and their experience with non-asbestiform rock that has been mistaken as dangerous asbestiform. I would like to include this in the record.

Senator BOXER. Of course, without objection, it will be done.

[The referenced material follows:]



El Dorado County Office of Education

June 11, 2007

Vicki L. Barber, Ed.D.
Superintendent

Francie Helm
Deputy Superintendent

Cathy Bean
Deputy Superintendent

County Board of Education
Dolores Garcia
John Lane
Matt Boyer
Gene Rasmussen
Heidi Weiland

Senator James Inhofe
453 Russell Senate Office Building
Washington, DC 20510 -3603

Dear Senator Inhofe:

Thank you for requesting comments for the hearings that are being held on the health effects associated with Naturally Occurring Asbestos (NOA). Our public schools have been addressing this issue for many years and we would like to share our experience to help you in your deliberations.

Although we appreciate the assistance we have received from federal regulators to minimize the health effects of NOA at our school sites, we have experienced frustration with the lack of established standards and testing protocols. For example, the EPA and our high school district spent millions of dollars mitigating a school site based upon soil samples collected by the EPA. Split samples of the soil tests indicated conflicting and significantly different results. Over a year later, the EPA declared that soil sampling is a "notoriously imprecise method," and is "really not scientifically credible." When the EPA conducted subsequent tests in the community, an independent review of the tests by the USGS found that most of the particles counted by the EPA were not asbestiform and were not actionable. *Mineralogy and Morphology of Amphiboles Observed in Soils and Rocks in El Dorado County*, pages 41-42.
http://www.edcoe.org/asbestos/documents/USGS_edhreport.pdf

We do not doubt that mitigation of the high school was a prudent course of action. Our school districts share with the EPA a strong commitment to protect the health and safety of the public, including our students, employees and guests. However, we also share a commitment as guardians of public funds to insure that our public funds are spent wisely with maximum benefit to the public. This has proved difficult in El Dorado County because of the lack of science-based regulatory standards applicable to natural environments. We have spent millions of dollars removing and covering up soil that may or may not pose a health risk. This is particularly significant given that the Cancer Surveillance Program reported to Dr. Mistry of ATSDR in an August 24, 2004, letter (attached) that "...there was not a greater than expected number of mesothelioma of the pleura in either the county of El Dorado or in the selected census tracts in El Dorado County, at the 99-percent confidence level, from 1988 through 2001." Although we agree that we should take a precautionary approach, because the issue exists on a national level we also believe

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that it is critical to obtain scientific answers to whether in fact soil with non-asbestiform rock is a threat to public health.

Since this issue has cost our schools millions of dollars, we have advocated a coordinated, accountable, accurate, and scientifically based approach to deal with the control and mitigation of any possible health risks from NOA. Specifically, we question whether the "particle counting" procedure developed in industrial settings is appropriate in the natural environment. Numerous experts have expressed concern that this practice results in the misidentification of minerals and thus the mischaracterization of health risks in real world, mixed dust environments.

Because of the many different opinions in the scientific community on this issue, we have hired numerous experts to advise us. One of these experts is Dr. Wayne Berman, a physical chemist (Caltech) and a noted risk assessor in the field of asbestos. Dr. Berman has addressed the issue as follows:

Over the years, asbestos has been defined in numerous ways for numerous purposes. These include four types:

- *Commercial definitions designed to highlight the properties of asbestos that impart commercial value;*
- *Geologic definitions that distinguish true asbestiform materials from non-asbestiform (cleavage fragments) based on their mechanism of formation;*
- *Regulatory definitions that distinguish materials to be regulated from those that are not; and*
- *Analytical definitions that provide laboratories with the tools required to characterize and count structures to determine their concentration.*

What is important for risk assessment, however, is to characterize and count asbestos based on properties that contribute to biological activity. Unfortunately (at least to date), the definitions developed for the purposes described above have been inconsistent and none have succeeded as a definitive measure that can be used to support risk assessment. This is because the existing definitions do not coincide sufficiently with the characteristics of asbestos that contribute to biological activity. Indeed, most regulatory and analytical definitions typically contain the caveat that they are not "risk-based." Moreover, although much is now known, there are still controversies surrounding at least some of the details concerning the asbestos characteristics that contribute to biological activity. Thus, with all of this in mind, it is important to manage asbestos risks in a manner that is demonstrably health protective while avoiding incorporation of conservative assumptions that are so overwhelmingly broad as to preclude the ability to distinguish potentially risky situations from those that are clearly not.

Dr. Berman's opinion on this issue is further discussed in the enclosed letter.

Many of the issues and recommendations that we believe would help resolve some of the conflicts surrounding NOA were set forth in a September 15, 2003, letter from Wayne Nastri,

Letter to Senator Inhofe
June 11, 2007

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Region IX Administrator to Edwin Lowry, Director, California Department of Toxic Substances Control. In that letter Mr. Nastri makes the following recommendations:

A. Asbestos Sampling and Analytical Protocols. The EPA shall establish consistent asbestos sampling and analytical protocols for risk assessment purposes. The protocol will include reference samples, method standardization and reproducibility testing. Protocols for consistent sample collection and analysis shall be defined that will measure asbestos with appropriate sensitivity to support decisions made in the risk assessment protocols.

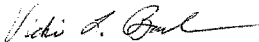
B. Background Asbestos Concentrations in Soil and Air. Provide data and information on the background level of asbestos in the air and soil in different regions of the United States to assist in determining what is considered to be an elevated level of asbestos.

C. Exposure Assessment Tools for Asbestos in Soil. Establish an accepted risk assessment for asbestos, including what mineralogy and fiber size characteristics should be considered for conducting risk assessments, and which fibers are of most concern.

D. Laboratory Certification Program. A protocol shall be established for testing both soil and air samples of asbestos and incorporated into the National Voluntary Laboratory Accreditation Program.

Implementing these recommendations and establishing a protocol to provide measurements using an exposure metric that matches the range of structures that are found to contribute to adverse health effects will help establish risk based standards so that all communities will have clear and unambiguous requirements for mitigating NOA exposure. Thank you for your consideration. Please let us know if we may be of any assistance on this issue.

Sincerely,



Vicki L. Barber, Ed.D., Superintendent
El Dorado County Office of Education

VB:jkm

Cc: Chair Barbara Boxer
Wayne Nastri
Wayne Berman, Ph.D.

Attachments

S:\Naturally Occurring Asbestos\2007\Letter to Senator Inhofe 6-11-07.doc



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bermanw@comcast.net

June 11, 2007

Vicki L. Barber, Ed.D.
Superintendent
El Dorado County Office of Education
6767 Green Valley Rd.
Placerville, CA 95667-8984

RE: Opinion concerning the toxicity of asbestiform and non-asbestiform particles

Dear Vicki:

In response to your request, my opinion concerning the relative hazard of asbestiform and non-asbestiform particles (of similar mineralogy) follows.

The published studies that directly address this issue indicate a substantial difference in the toxicity of these particles. At the same time, my own work indicates that the controversy surrounding this issue is exacerbated by use of analytical methods that do not provide adequate measurements of biologically active particles (whether asbestiform or not). When asbestos concentrations are measured using methods that adequately track biological activity, the need to further distinguish asbestiform from non-asbestiform particles appears to largely disappear (see Berman and Crump 2001, 2003).

More importantly, I have shown that "counting everything" (i.e. arbitrarily including non-asbestiform particles in counts to determine asbestos concentrations) is *not* automatically health protective (see Berman 2007). In fact, in real world situations, the approach Dr. Crump and I have developed for assessing asbestos-related risks is demonstrably more health protective than the approach in which non-asbestiform particles are counted.

For example, when applied at the Libby, Montana site, our approach (Berman and Crump 2001) provides risk estimates that are 7 times what is estimated using the approach in which all non-asbestiform particles are counted (Berman 2007, Table 2). Similarly, at a marble quarry in Sparta, New Jersey, risks estimated using our approach are almost 20 times greater than those estimated using the approach in which non-asbestiform particles are counted and 50 times greater than this latter approach when non-asbestiform particles are excluded (Berman 2003, Tables 8 and 9).

In general, at sites in which chrysotile (one form of asbestos) is the primary risk driver, these various approaches tend to provide similar risk estimates. However, when amphibole asbestos is the risk driver, the Berman and Crump approach (Berman and Crump 2001, 2003) tends to provide substantially higher estimates of risk even when non-asbestiform particles are included in the counts (Berman 2006).

In summation, use of the Berman and Crump protocol for assessing asbestos related risk appears to obviate the need to distinguish between asbestiform and non-asbestiform particles,

while better protecting the public from asbestos-related risks. It should also be pointed out, however, that arbitrarily extending the asbestos regulations to non-asbestiform particles is not science. This is because non-asbestiform particles were not adequately represented in the epidemiology studies used to develop the regulations for asbestos (see Nicholson 1985; Berman and Crump 2001, 2003; Berman 2007, Table 1). Such an arbitrary extension would be equivalent to applying the regulations for chromium to all metals (including such non-toxic metals as iron) simply because they are collectively called "metals". As far as I know, no one advocates such a change.

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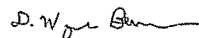
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Nicholson, W.J., *Airborne Asbestos Health Assessment Update*. U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, EPA 600/8-84-003F, 1985.

I hope you find the above helpful. Please call me if you have any questions or comments.

Sincerely,



D. Wayne Berman, Ph.D.
Aeolus, Inc.
751 Taft St.
Albany, CA 94706

Jun 11 07 10:52a William M. Wright

530-876-8426

p.2

Cancer Surveillance Program
 2800 L Street, #440
 Sacramento, CA 95816-5600

August 24, 2004

Kelna Mistry, M.D., F.A.A.P.
 Agency for Toxic Substances and Disease Registry (ATSDR)
 Exposure Investigations and Consultations
 1600 Clifton Road, NE
 Mailstop: E-29
 Atlanta, GA 30333

Dear Dr. Mistry;

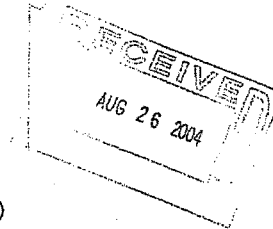
This letter is to inform you of the results of a data analysis conducted at your request to examine the incidence of mesothelioma of the pleura in El Dorado County. The Cancer Surveillance Program, Region 3 of the California Cancer Registry (CCR), collects information about cancer diagnosed among residents of thirteen counties in the Sacramento area. Cases are reported to the registry by hospitals, physicians and other facilities and are estimated at 100% for non-prostate cancer cases diagnosed between 1988 and 2001.

In this analysis we examined the incidence of mesothelioma of the pleura in El Dorado County and selected census tracts in El Dorado County, California. The census tracts selected for analysis were: 306.01 & .02; 307; 308.01-.04; 309.01 & .02; 310-312; 313.01-.03; 314.01-.03; 315.01 & .02. These census tracts cover all of the western slope of El Dorado County and include the towns of Placerville, Diamond Springs, Pollock Pines, Shingle Springs, Cameron Park, El Dorado Hills, Georgetown, Cool and Garden Valley. In addition, these are the same census tracts (updated to the 2000 census) used by Dr. Rosemary Cress in her 1997 analysis of mesothelioma of the pleura in El Dorado County.

The methodology for this analysis has been standardized by the CCR and is the same used throughout the state by CCR regional offices. The analysis consists of a comparison between the number of cancer cases observed in the specified geographic area and the number of cancer cases that would be expected to occur in the surrounding geographic area (the Sacramento Region) during a specified time period. The observed cancer cases are those who reported their residence, at the time of diagnosis, in the specified geographic area. The expected number of cancer cases is estimated by applying incidence rates for the specified cancer in the region for the same time period, specific for sex, race/ethnicity, and the 5-year age group to the corresponding numbers of person-years-at-risk for the observed cancer cases. The current CCR policy is to conduct this statistical comparison of the observed and expected number of cancer cases by estimating a 99-percent confidence interval based on the Poisson distribution, around the observed number of cases, and determining

Region 3 of the California Cancer Registry

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whether the observed number of cases falls within this interval. The results of your requested analysis of mesothelioma of the pleura in El Dorado County are shown in the table below.

Observed vs. Expected Analysis of Cases of Mesothelioma of the Pleura, El Dorado County, 1988-2001					
Geographic Area	Observed cases	Average annual observed cases	99% confidence interval	Expected number of cases	Observed/Expected cases
El Dorado County	30	2.14	0.10 – 9.27	2.30	0.93
Selected census tracts in El Dorado County	19	1.36	.001 – 7.43	1.22	1.11

Our results indicated that there was not a greater than expected number of mesothelioma of the pleura in either the county of El Dorado or in the selected census tracts in El Dorado County, at the 99-percent confidence level, from 1988 through 2001.

We are aware of the great impact that cancer has on families and the community and are pleased to be able to provide information and answer questions about cancer incidence in the Sacramento Region. I hope this information is helpful. Please feel free to call me if you have further questions or have additional information that you would like me to consider.

Sincerely,



Monica Brown, MPH, PhD
Epidemiologist

cc: Dr. Stephen Drogin, Health Officer, El Dorado County Dept. of Health and Human Services
William Wright, Ph.D., Chief, Cancer Surveillance Section, California Dept. of Health Services
Cynthia M. Creech, C.T.R., Director, Cancer Surveillance Program

Senator BOXER. Dr. Castleman, we welcome you. Please proceed.

STATEMENT OF BARRY CASTLEMAN, Sc.D., ENVIRONMENTAL CONSULTANT

Mr. CASTLEMAN. Thank you, Senator Boxer.

I have worked for 35 years with U.S. Government agencies, non-governmental organizations, international agencies on asbestos issues. I also testify as an expert witness about the public health history of asbestos, the subject of my doctoral thesis.

I work with other public health workers all over the world on asbestos, and we all hope to see the United States join about 40 other countries that have banned asbestos.

The World Trade Organization has concluded that controlled use of asbestos products is unrealistic, supporting national asbestos bans. Here, as we have noted, the EPA tried to ban asbestos, but the rules were overturned in a court challenge.

There is broad support for banning asbestos in the United States today. A statement in support of the Ban Asbestos in America Act has been endorsed by 18 groups, including leading American unions, environmental groups and asbestos victims groups, groups including the AFL-CIO, the Service Employees International Union, the Natural Resources Defense Council, the White Lung Association, Asbestos Diseases Awareness Association. There are also a number of groups from around the world that have also joined in this, showing the tremendous impact on the rest of the world of the United States having not up until now banned asbestos. So I offer this for the record, the statement and the groups that have endorsed it.

U.S. consumption of asbestos annually is now what it used to be in a single day in 1973. It is about one 400th of what it was at its peak. There is practically nothing left of the asbestos industry here. The main use appears to be in roofing products and one process for making chlorine. The European ban on asbestos has no exemption for roofing products and they don't seem to have any problem with that. Here too we have plenty of alternative materials.

As for chlorine, it is made by two processes, two old ones and one modern one. One of the old processes is the diaphragm-cell process, in which an asbestos diaphragm has been used. The newer membrane-cell process is the only type used in new plant construction since 1987 around the world, because it is much more energy efficient and it doesn't use mercury or asbestos.

There were questions raised about the exposures that you can get in this industry, Senator. Asbestos exposures arise from transport and storage of sacks of asbestos involving tears in the sacks that must be identified and sealed and the spillage cleaned with special vacuum cleaners; cutting open and emptying sacks of asbestos; transferring sacks into slurry mixing tanks can cause additional exposures; if there is any spillage of the slurry, that has to be cleaned up very carefully or you have the drying of the material and the creation of an airborne asbestos hazard.

Then the diaphragm has to be properly handled and stored again, providing for the possibility of exposure. Then the hydroblasting for removal and replacement of the asbestos is another possible source of area contamination and drying and airborne ex-

posure. Then you have the waste asbestos from all of this that has to be dealt with as well as the personal protective clothing that workers may wear, throw away garments that then are hazardous waste themselves. I have comments on this in my prepared statement, Senator.

The diaphragm-cell chlorine plants can also be operated with non-asbestos diaphragms, as Senator Boxer pointed out there, available from companies including PPG in the United States, which has used it in their own plants. So they can replace the asbestos diaphragms with non-asbestos, or they can convert to the membrane process. In Japan, the chlorine industry is solely membrane cell. In Europe, I think there are only three plants left using asbestos diaphragms.

I would say that there shouldn't be a statutory exemption for the chlorine industry. They should have to justify that based on current technology to the EPA along with any other party that wants to have an exemption to the ban that Senator Murray has put into her bill.

The main problem, as pointed out, is the import of asbestos products. I simply would point out that these products compete against safer products made in the United States. There is just no reason why they should be allowed to be continuing to be imported.

As for contaminant asbestos, there are problems with talc. Four months ago I sent a letter with several other scientists to the Consumer Product Safety Commission. There is a notorious talc in upstate New York that is contaminated with asbestos. People have died with asbestosis, lung cancer and mesothelioma from mining the stuff. Yet the company that makes it persists in selling the stuff as if it doesn't have asbestos, and making mineralogical arguments to that effect. Meanwhile, this product is being used in consumer products used all over the United States.

Senator BOXER. Can you wrap up, Doctor?

Mr. CASTLEMAN. Sure. So I think that it is very important for the Government to deal with the issue of contaminant asbestos and talc, vermiculite and construction stone. We can definitely deal with a ban on commercial forms of asbestos very quickly and I hope you will proceed to do that.

[The prepared statement of Mr. Castleman follows:]

STATEMENT OF BARRY CASTLEMAN, Sc.D., ENVIRONMENTAL CONSULTANT

Members of the committee, thank you for inviting me to testify about public health issues related to asbestos exposure in America today. I have worked on public health issues surrounding asbestos for 35 years, including product bans at the Consumer Product Safety Commission and regulations at EPA, OSHA, and FDA. My bachelor's degree is in chemical engineering, my masters is in environmental engineering, and my doctorate is in public health policy from the Johns Hopkins School of Hygiene and Public Health. I will discuss public health issues related to present asbestos hazards and banning asbestos in the U.S.

BACKGROUND

The public health and corporate history of asbestos were the subject of my doctoral thesis and a 900-page book (asbestos: Medical and Legal Aspects). The book is in its 5th edition and has been cited in judicial opinions up to the Supreme Court. I have testified about this history as an expert witness in courts across this country since 1979.

It is tragic that so much of the public health catastrophe we are seeing now was not only foreseeable but foreseen long ago. The cancer hazard of breathing asbestos

dust was noted in *The New York Times*, *Business Week*, *Scientific American*, and *Newsweek* all before this time in the year 1950. But it would not be until after social developments led Congress to establish the EPA, OSHA, and NIOSH in 1970 that workers and the public were first protected from or even warned about the dangers of most asbestos products. So it was not until 1973 that our use of asbestos peaked, at around 800,000 metric tons. U.S. consumption of asbestos for the year 2006 was down to around 2000 metric tons, approximately the amount we used each day in 1973.

Americans are now dying from asbestos cancers and asbestosis at the rate of 10,000 per year, as a result of past asbestos use. That is more than one death per hour. The medical literature is replete with tragic cases of mesothelioma in people with minimal occupational and environmental exposures to asbestos. The World Trade Organization has rejected the idea that there is really such a thing as "controlled use" of asbestos, citing do-it-yourself home repair as a prominent example of something no government can make safe through regulations. Starting in the early 1980s, Sweden and other countries pressed manufacturers to substitute asbestos in vehicle brakes so they could impose national asbestos bans.

The U.S. EPA tried to phase out the major uses of asbestos in regulations published in 1989, but the rules were overturned in a court challenge. EPA was unable to persuade the Department of Justice to appeal the court's 1991 decision, leaving the matter to Congress to resolve.

U.S. ASBESTOS USE TODAY

The main problem now is imported asbestos products, commercial asbestos product manufacture is almost extinct in the U.S. Because there is practically no restriction on what can be sold with asbestos in the U.S., we continue importing asbestos-containing brake linings, asbestos gaskets, asbestos yarn and thread, etc., despite the fact that these products are no longer made in the U.S. Given the abject lack of OSHA enforcement of asbestos product labeling requirements, there is a real concern that some imported asbestos products are not even labeled with the required health warnings.

The last U.S. asbestos mine closed in 2002. U.S. consumption of commercial asbestos in domestic manufacturing seems to be limited now to roofing felts and related products, and chlorine manufacturing (see below). No asbestos roofing products are needed or allowed in the many countries of Europe where asbestos has been banned for over 10 years (e.g., Sweden, Germany, Italy, France, Denmark, Holland), and alternative non-asbestos roofing products are widely available here. U.S. brake manufacturers no longer use asbestos, and the auto industry has already stopped using asbestos brakes in new vehicles and replacement parts throughout Europe and elsewhere.

ASBESTOS EXPOSURE IN CHLORINE MANUFACTURING

Asbestos has long been used in the diaphragm-cell process for making chlorine. This process and the old mercury-cell process are still operated, although a newer and more environmentally and technically superior membrane-cell process has been the only type built anywhere in the world for the past 20 years. Some diaphragm and mercury cell plants have been converted to membrane cells. Power requirements are substantial for chlorine manufacture, and the membrane cell process requires 15–20 percent less energy than diaphragm cells.

Asbestos exposures in the chlorine industry arise from transport and storage of sacks of asbestos, typically involving tears in the sacks that must be identified and sealed, with spillage cleaned with high-efficiency vacuum filters. Cutting open and emptying sacks of asbestos and transferring asbestos into slurry mixing tanks can cause additional exposures. The empty sacks are an additional exposure source, they must be carefully gathered up, placed in sealed containers, and landfilled at approved sites. Storage and handling of partially used sacks are also sources of exposure. If the slurry is spilled, this has to be meticulously cleaned up right away, because once it dries it becomes a source of airborne asbestos exposure. Handling and storage of prepared or purchased pre-deposited asbestos diaphragms can cause additional exposures. Hydro-blasting for removal/replacement of asbestos diaphragms is another possible source of area contamination, drying, and airborne exposure. The water used for hydro-blasting has to be contained and the asbestos filtered from it. The waste asbestos from this water and the spent diaphragms have to go to a landfill that accepts asbestos.

To some degree, workers can be protected against these asbestos exposures if they wear respirators that will remove some of the asbestos from the air they breathe, and if they wear personal protective clothing such as disposable coveralls. But these

safeguards are partial. The respirators must be fit-tested and properly maintained; and even the protective clothing is a hazardous waste that requires special precautions for disposal. Chlorine Institute pamphlet 137, Guidelines: Asbestos Handling for the Chlor-Alkali Industry, recommends personal protective clothing and respirators only for workers exposed in excess of the permitted limits in the OSHA standard, which is all that is legally required. But OSHA has admitted that compliance with its limits will not fully prevent deaths from asbestos. Dr. Richard Lemen and NIOSH epidemiologists estimate that exposure at OSHA's permissible exposure limit for asbestos will still cause 5 deaths from lung cancer and 2 deaths from asbestosis in every 1000 workers exposed for a working lifetime. (L. Stayner et al., Exposure-Response Analysis of Risk of Respiratory Disease Associated with Occupational Exposure to Chrysotile Asbestos. *Occ. Env. Med.* 54: 646-652, 1997).

While company manuals may state that the workers are supposed to observe various precautions to minimize asbestos exposure, there is virtually no OSHA inspection of these workplaces, and the usual combination of production demands, Gulf coast heat and humidity, and carelessness will assure that things are not always done "by the book" to minimize workers' asbestos exposure.

In the past 15-20 years, non-asbestos diaphragms have become available for relatively simple replacement in asbestos diaphragm cell plants. These are sold by Eltech/DeNora and PPG Industries in the U.S. The non-asbestos diaphragms cost more and last longer than asbestos. Although two-thirds of the chlorine made in the U.S. in 2006 was from diaphragm cells, I don't know how many of these used non-asbestos diaphragms. The technology continues to advance, however, and has had wide acceptance in Europe, where the European Union's temporary exemption allowing asbestos use in chlorine manufacturing comes up for reconsideration next year. I understand that there are only 3 chlorine plants in Europe still using asbestos diaphragms.

PPG Industries has been a leader in the development of non-asbestos "Tephram" diaphragms, and PPG is also a major producer of chlorine in the U.S. I understand that PPG routinely replaces non-asbestos Tephram diaphragms in its asbestos diaphragm-cell units when they are taken down for periodic maintenance. I do not know of any technical reasons why other diaphragm-cell chlorine manufacturers could not do the same thing.

Therefore, if chlorine manufacturers want extra time to convert to non-asbestos technology, perhaps that could be allowed but with the requirement that when the equipment is shut down for maintenance overhauls, the new diaphragms used be non-asbestos. A similar several-year time frame might be allowed for diaphragm-cell units that manufacturers want to convert to membrane cells.

CONTAMINANT-ASBESTOS IN TALC, VERMICULITE, STONE, AND OTHER MINERALS

1. Talc

Aside from commercial asbestos minerals that have been used for the past century in various products, asbestos also occurs as a contaminant in other minerals. This has been long recognized, and at times the occurrence of asbestos fibers in these products has even been noted in advertisements for them. For example, it was repeated that "asbestiform varieties are common" in a 1966 brochure describing NYTAL, the trade name for a talc mined in New York by R. T. Vanderbilt Company.

Health officials had long ago noted that New York talc miners were dying from lung scarring, including asbestos bodies in the scarred lung tissues and pathology "similar to [findings] reported in asbestosis." (FW Porro et al., *Pneumoconiosis in the Talc Industry*. *Am. J. Roent. Radium Therapy* 47: 507-524, 1942. Quote from FW Porro et al., *Pathology of Talc Pneumoconiosis with Report of an Autopsy*. *North. N. Y. Med. J.* 3: 23-25, 1946). New York state labor protection officials noted that other writers had attributed talc lung scarring to the fibrous varieties of talc, and observed that, for New York talc miners, "In general, the clinical, [chest X-ray], and pathological findings were similar to those observed in asbestosis." (M Kleinfeld et al., *Talc Pneumoconiosis*. *Arch. Ind. Health* 12: 66-72, 1955; M Kleinfeld et al., *Talc Pneumoconiosis/A Report of Six Patients with Postmortem Findings*. *Arch. Env. Health* 7: 101-115, 1963) So it should have come as no surprise that these talc miners also had an excessive death rate from cancers of the lung and pleura (M Kleinfeld et al, *Mortality among Talc Miners and Millers in New York State*. *Indust. Hyg. Review* 9: 3-12, 1967).

Starting in 2002, there have been published reports of cases of mesothelioma, considered a signal tumor for asbestos exposure, among New York talc miners. An epidemiology report sponsored by R. T. Vanderbilt Company found 2 cases among the 782 white men who had been employed for at least one day at the New York talc mines between 1948-1989 (Y Honda et al., *Mortality among Workers at a Talc Min-*

ing and Milling Facility. *Ann. Occup. Hyg.* 46: 575–585, 2002) R. T. Vanderbilt Company has stipulated that, subsequent to the period covered in this study, at least 5 more cases of mesothelioma have occurred among its employees (*Hirsch vs. RT Vanderbilt Co. Middlesex Co. NJ Superior Court, Nov. 2, 2006*). It appears that some of these cases were the subject of workers' compensation claims.

Meanwhile, independent pathologists reported finding at least 8 confirmed cases of mesothelioma among New York state talc miners and millers as of 1986, and added 5 new cases (MJ Hull et al., *Mesothelioma among Workers in Asbestiform Fiber-bearing Talc Mines in New York State. Ibid. Suppl. 1, 132–136, 2002*) Commercial amphibole asbestos fibers were virtually absent in the lung tissues of all 10 cases subjected to pathological examination, indicating that other occupational asbestos exposures (e.g., in construction) were not responsible for these mesotheliomas of these workers.

R. T. Vanderbilt denies that there is asbestos in its talc and that its talc causes asbestos diseases. It is interesting to read internal memoranda of the Johns-Manville Corporation, the country's largest asbestos company, shortly after J-M bought a talc mine in the early 1970s. J-M's talc had asbestos in it, and J-M labeled it accordingly, pursuant to the 1972 OSHA asbestos regulations. This upset executives at Vanderbilt, who claimed that J-M placing asbestos warnings on containers of talc was causing a "big stink" and "irreparable damage" to Vanderbilt in 1974. J-M laboratories proceeded to examine the Vanderbilt talc product grades microscopically. Their comments on what they found were expressed in internal memos that only came to light in recent years, after the consummation of the J-M bankruptcy plan in 1988.

The J-M people found plenty of asbestos in the New York talc and used very strong language about Vanderbilt's insistence that there was no asbestos in its talc:

It is apparent that the R. T. Vanderbilt presentations to OSHA, NIOSH, FDA, MESA, etc. are based on something less than the truth. I feel it difficult to believe that they could be so grossly misinformed as to what their materials really are.

(RS Lamar, J-M Internal Correspondence, Oct. 11, 1974)

The R. T. Vanderbilt position with respect to labeling must be deliberately perfidious; they cannot be this misinformed. Slim Thompson, their technical director, has a Ph.D. in mineralogy. At the moment, Vanderbilt is misleading their customers and confusing ours with the decision not to label. Ultimately, the truth will out, and they will be forced to label.

(RS Lamar, "An Assessment of the J-M Position with Talc," J-M Internal Correspondence, Mar. 20, 1975)

The truth still hasn't won out, I am sorry to say. Fully 32 years after this was written by a morally offended official at the largest asbestos corporation in America, R. T. Vanderbilt still sells talc that they say has no asbestos hazards.

As a result, this talc is used commercially in an unknown number of industrial processes and consumer products, endangering thousands, perhaps millions, of unwitting workers, consumers, and children. Along with two other scientists, I filed a complaint with the Consumer Product Safety Commission about Durham's Water Putty, a product sold across the country in Ace Hardware stores, because it exposes users to airborne asbestos arising from the product's ingredient of Vanderbilt talc (Jan. 29, 2007). The Center for Environmental Health, in Oakland, filed a complaint last month with the California Attorney General's Office, asserting that Durham's product has violated state law because it has been sold without cancer warning labeling. The Connecticut Department of Public Health filed another complaint with CPSC about asbestos hazards to school children from Vanderbilt talc in art clay products (Feb. 6, 2007). In 2006, a jury awarded \$3.3 million to the estate of a New Jersey potter, finding that Vanderbilt's talc was a substantial cause of his death. Only Vanderbilt knows what other commercial uses and products expose the American people to this talc.

The people in this country urgently require the government's protection against the sale of such products by manufacturers who prefer to play semantic games over what mineralogists and government regulations call "asbestos", while people continue to be unknowingly exposed to mortal peril. The grossly excessive number of mesotheliomas among the New York talc mine and mill workers is very powerful evidence that this material is lethal and should be regulated as asbestos and banned when asbestos is banned.

2. VERMICULITE

We have seen this suppression of contaminant-asbestos health warnings in other cases. WR Grace sold vermiculite insulation that was contaminated with asbestos until 1990, ultimately placing it in millions of homes in the U.S. and Canada. The

company had been called the Vermiculite and Asbestos Corporation when it opened in 1919, and Montana State Board of Health reports on the high asbestos exposures of the workers were sent to the company in the 1950s and 1960s. WR Grace sold this material without applying OSHA asbestos warning labels first required in 1972. By 1985, a "Personal and Confidential" memo reviewed the serious business problems from Grace continuing to sell vermiculite products that contained asbestos (RC Walsh, Feb. 2, 1985). Noting the difficulty of continuing to obtain insurance, one of the parties to this exchange commented that this "increases attractiveness of setting business up as a subsidiary or some other legal form to distance it from Grace assets." Criminal proceedings are currently pending against Grace executives for selling this product as they did, but it was a public health failure that the government had not taken earlier action to prevent the widespread sale and exposure of millions of people to this deadly product. (A. Schneider, Big Asbestos Prosecution in Jeopardy, U.S. Argues. Seattle Post-Intelligencer, June 5, 2007) <http://seattlepi.nwsource.com:80/local/318479-grace05.html?source=rss>

Vermiculite is still mined in the U.S. by Virginia Vermiculite. Mine Safety and Health Administration (MSHA) officials have raised concern about asbestos exposure of workers at this site and at plants receiving and processing this material. Vermiculite has been widely used in such products as potting soil, insulation, and cat litter.

(A. Schneider, Virginia Miners at Risk from Asbestos. Seattle Post-Intelligencer, Oct. 4, 2000) <http://seattlepi.nwsource.com/uncivilaction/asb04.shtml>

3. STONE

Asbestos can also be present in basic stone used in construction. In 2005, research was published linking residence in areas of California with naturally-occurring asbestos outcrops and increased risk of mesothelioma (Pan et al., Am. J. Resp. Crit. Care Med. Oct. 2005). Dr. Marc Schenker, one author of this study, expressed concern about the health hazard faced by people with environmental exposure in areas where land development was proceeding in El Dorado County, California, and other areas where asbestos minerals are known to be present in the soil in significant amounts. <http://www.medicalnewstoday.com/medicalnews.php?newsid=32149>

IRON ORE

There has been controversy for at least 35 years over asbestos-like material in the host rock of ore mined in the Iron Range of Minnesota. By March 2006, State officials identified 35 deaths from mesothelioma among the miners, in addition to 17 fatal cases previously known to have occurred between 1988–1996. <http://www.startribune.com:80/462/story/1250516.html>

What is needed is a process whereby the EPA does surveillance of possible sources of contaminant-asbestos around the country, starting with Vanderbilt talc and Virginia Vermiculite, using USGS mineral survey maps to help identify hot spots. Then, as operations of concern are discovered, there needs to be a process of investigation, first for the government to realistically sample the products of these operations and do bulk sample analysis. Then, if there is any concern over public and worker exposure, the company should have to disclose its commercial customer list to EPA. EPA could then contact the customers to see how the material is handled, ask what products it is used to make, and assess what asbestos exposures result for workers, consumers, and people living where the stuff is shipped, processed, and put to end use. In annual reports, EPA should disclose what operations it has under investigation, and summarize the state of these investigations, describing the commercial uses of the suspect materials. And of course, the EPA needs the authority to close operations and stop the sale of products that are deemed a threat to public health.

BROAD SUPPORT FOR BANNING ASBESTOS IN THE U.S. AND INTERNATIONAL IMPLICATIONS

A statement in support of the Ban Asbestos in America Act of 2007 has been endorsed by groups that have been active on asbestos issues for many years in the U.S. and around the world. These include trade unions, leading environmental groups, asbestos victims' groups, and medical and public health groups. I ask that this statement and list of supporters be made a part of the record of this hearing.

You can see that there are many groups from other countries that signed the statement in support of banning asbestos in the U.S., countries where asbestos is still used and is the subject of public health struggles. I work with people all over the world on asbestos, and everywhere the local asbestos industry points to the U.S. and says, "But asbestos is not banned in the United States." It would be great value

to public health workers the world over if the U.S. finally banned asbestos. It would significantly assist efforts in Brazil, India, South Africa, Thailand, and many other countries. So, on behalf of the rest of the world and the people in our country, I urge you to ban asbestos in the U.S. now. It is long past time for the U.S. to take a 21st century position on this issue and catch up to Croatia by banning asbestos.

I have not been paid by anyone for my preparation and testimony here today. Nor do I represent anyone but myself, a public health worker. Thank you for inviting me to speak.

RESPONSES BY BARRY CASTLEMAN TO ADDITIONAL QUESTIONS FROM SENATOR BOXER

Question 1. What benefits do businesses get when developing or using alternatives to asbestos?

Response. Businesses benefit by developing safer alternatives to asbestos products in a number of ways. They have improved labor and community relations, compared to firms persisting in asbestos use. They avoid damaging publicity that often comes with resistance of employees, consumers, and plant neighbors to asbestos use. They don't have to spend money complying with EPA and OSHA asbestos standards (e.g., industrial ventilation system fixed and operating costs, periodic employee medical exams and air sampling, 30-year retention of medical records, cancer warning product label requirements, hazardous waste disposal requirements, etc.). They save money on group life, group health, workers' compensation, and product liability insurance. They will be around a lot longer than companies still using asbestos, because asbestos is hazardous, discredited technology losing markets worldwide.

Question 2. Your testimony contained disturbing references to individuals in business that potentially hid information on health threats related to their products that may contain asbestos. In your experience, have other businesses potentially tried to hid information that their products may contain material that could cause diseases associated with asbestos?

Response. There are widespread examples of businesses that hid information that the use of their products could cause asbestos diseases. Many examples are given in my book, *Asbestos: Medical and Legal Aspects* (5th Ed., 2005). Despite published reports between 1932–1964 from around the world that commercial asbestos products were causing death and disease, and despite workers' compensation claims around the country by workers harmed by asbestos products in construction in the 1940s and 1950s, no asbestos products appear to have borne even mild health warning labels before 1964. No manufacturer placed health warnings on brake linings, drywall patching compounds, and many other asbestos products until after they were required to by OSHA in 1972, sometimes many years after (e.g., Ford, Chrysler). OSHA has failed for 35 years to monitor the marketplace to assure that required labels were placed on asbestos products, so that products we import today may well contain asbestos that is not disclosed by foreign manufacturers and distributors. This problem is mainly confined to imports, as there are practically no commercial asbestos products made in the U.S. anymore.

Other businesses selling products with contaminant-asbestos have withheld information from consumers to this day, and the scale of this menace is unknown. I gave examples in my statement of asbestos-contaminated talc from R. T. Vanderbilt and vermiculite from Virginia Vermiculite. These companies deny that there is asbestos in their products, which are sold to commercial customers. At the customers' plants, workers are consequently unaware of the danger they face in handling these materials. The products go out to the public with no labeling warning of the cancer danger that the dust can pose. Workers are also endangered by disturbing, extraction, and construction involving iron ore in Minnesota, where there have been a large number of mesotheliomas among the workers, and extracting stone in El Dorado County, California.

One product containing Vanderbilt talc is Durham's Water Putty, which has been analyzed and shown to contain asbestos; airborne asbestos is released in alarming concentrations when the product is used. With two other scientists, I urged the Consumer Product Safety Commission to get this product off the market on January 29, 2007; we also urged CPSC to investigate all other commercial applications of Vanderbilt talc. No reply has yet come from CPSC, and this appears to be a matter of considerable public health importance.

Question 3. Please submit a copy of the statement of support for Senator Murray's bill that you discussed at the hearing.

Response. [The "Statement in Support of the Ban Asbestos in America Act of 2007" follows.]

Statement of Support for the Ban Asbestos in America Act of 2007

Americans are dying at the rate of one per hour from past use of asbestos and the continuing contamination it leaves behind. National and international authorities agree that even slight exposures to asbestos carry some risk of cancer, and there are many tragic examples of this.

The US is practically the only industrial nation (along with Canada, a major exporter of asbestos) that has not banned asbestos. As a result, even though very little asbestos is used in American industry, asbestos brakes, asbestos-cement panels, and other asbestos products not made here continue to be imported. These imported products endanger workers and the general public, while competing against safer products made in the USA.

In addition, the presence of asbestos as a contaminant of other minerals, vermiculite, talc, and stone, has not received the attention it deserves. This has resulted in environmental and occupational hazards at quarries and mines and the asbestos contamination of consumer products. The government urgently needs to analyze this problem from coast to coast and take appropriate regulatory action to protect public health and the environment.

Over 40 countries have banned asbestos, including all of Europe and countries around the world, the latest to announce bans being Peru and South Korea. The US EPA tried to ban all major uses of asbestos in 1989, but the regulations were overturned in a court challenge. It now falls to the Congress to shut down what little asbestos use remains in the US economy and close the door to deadly asbestos product imports.

We therefore support the enactment of S. 742, the Ban Asbestos in America Act of 2007, sponsored by Senators Patty Murray, Harry Reid, Clinton, Kennedy, Durbin, Feinstein, Baucus, Boxer, Leahy, Casey, Brown, Cardin, Feingold, Harkin, Kerry, Lieberman, and Whitehouse.

Signatory Groups in the United States:

AFL-CIO	(Peg Seminario)
Service Employees International Union	(Bill Borwegen)
Natural Resources Defense Council	(Jennifer Sass)
Asbestos Diseases Awareness Organization	(Linda Reinstein)
White Lung Association	(Jim Fite)
Environmental Working Group	(Jane Houlihan)
Greenpeace	(Rick Hind)
New York Committee for Occupational Safety and Health	(Joel Shufro)
Health Care Without Harm	(Bill Ravanese)
Commonweal	(Charlotte Brody)
Kentucky Environmental Foundation	(Elizabeth Crowe)
Center for Environmental Health	(Caroline Cox)
Center for International Environmental Law	(Glen Wiser)

Environmental Health Fund (Gary Cohen)
 Connecticut Coalition for Environmental Justice (Mark Mitchell)
 Healthy Child Healthy World (Christopher Gavigan)
 Clean New York (Bobbi Chase Wilding)
 Citizens Environmental Coalition (Steve Breyman)
 Center for Science in the Public Interest (Michael Jacobson)
 Making Our Milk Safe (Mary Brune)

Other Groups:

Canadian Auto Workers (Buzz Hargrove)
 Ban Asbestos Network Japan (Sugio Furuya)
 Japan Occupational Safety and Health Resource Center
 Japan Association of Mesothelioma and Asbestos-Related Diseases Victims and
 Their Families
 Ban Asbestos Network of India (Gopal Krishna)
 Occupational and Environmental Health Network of India
 Brazilian Association of Asbestos Exposed People from Rio de Janeiro e Sao Paulo
 (Fernanda Giannasi)
 Asbestos Exposed People from Bahia (Brazil)
 Virtual-Citizen Network for the Ban of Asbestos in Latin America
 Canadian Partnership Against Cancer (Larry Stoffman)
 National Committee on Environmental and Occupational Exposures
 BC and Yukon Building Trades Council
 BC Federation of Labor
 United Food and Commercial Workers British Columbia
 BC Government and Services Employees Union
 United Association of Plumbers and Pipefitters Local 170
 Canadian Office and Professional Employees Union
 Canadian Union of Public Employees BC Region
 Labor Environmental Alliance Society
 Occupational Health Clinic for Ontario Workers (Lyle Hargrove)

RESPONSE BY BARRY CASTLEMAN TO AN ADDITIONAL QUESTION FROM
SENATOR INHOFE

Question. Please describe, to the best of your knowledge, every instance in which you testified or were deposed as an expert witness for any party in asbestos litigation or were officially retained to provide expert advice to any party involved in asbestos litigation. For each instance provide the following:

(a) The name of the case, (b) Court (and whether State or Federal); (c) The name of the party that retained you or for whom you provided a deposition or testimony; (d) The dates on which you were deposed or testified or were otherwise retained; (e) What service you provided (testimony, deposition, etc.); (f) An explanation of the nature of your testimony or deposition; (g) Who paid you, and please provide the invoice (if you have the records).

Response. My records of my work as an expert witness in asbestos litigation are limited, and I am providing what I can. I have kept a running listing of trial and deposition testimony since starting this work in 1979. There is a one-line entry for each testimony, listing the name of plaintiff, whether trial or deposition, location of State or Federal court where the case was filed, and date. (Attachments: "Castleman Testimony 1979-1993"; and for 1993 to date, "trials")

In all cases but one in which I have testified, I was retained by plaintiffs. In that one, I was a witness for the United States of America in the U.S. Court of Claims. There, Johns-Manville Corporation was suing the government for partial reimbursement for damages paid by J-M to workers with asbestos diseases, arising from J-M's sale of asbestos products (without health warning labels) for shipbuilding during World War II. This was in 1987.

Two of the cases I have testified in were property damage cases, the rest were personal injury cases brought by workers, their family members and survivors. The property damage claims were brought by the State of Maryland and by Chase Manhattan Bank. The State of Maryland and Chase were suing asbestos product sellers for the costs of carefully removing and replacing asbestos products in their buildings.

My testimony in asbestos litigation is referred to as "State-of-the-art" testimony. It is about the public health and corporate history of asbestos, the subject of my doctoral thesis at Johns Hopkins. I trace the history of knowledge about the dangers of asbestos, describing the earliest and most significant reports of asbestosis, then various forms of cancer, tracking the development of knowledge as the population-at-risk was gradually recognized to be increasing with the addition of different populations of workers and other individuals over time. I describe what individual corporations and industries did as the problem of asbestos disease arose in different ways for them, based on a historic record replete with documents from institutional, governmental, and corporate archives.

The knowledge available in medical writings, safety publications, government publications, laws, industry trade magazines, major newspapers, encyclopedias, etc. is, on the whole, the standard against which the defendants' conduct is judged. In these cases, the manufacturers are held to the knowledge of experts about the hazards of the products they are selling to the public, and the product seller has the duty to warn about lethal, non-obvious hazards. Similarly, premises owners, such as oil and chemical companies, that bring in contract workers, have a duty to warn and protect these workers against hazards that the premises owners know or should know are there.

Payment for my services in litigation has been by the law firms that have hired my services, the U. S. Treasury, Chase Manhattan Bank, and the State of Maryland. I do not retain invoices after receiving payment of my bills, I am just a single person working as an independent consultant in occupational and environmental health. I try to minimize paperwork burdens for myself, as I have not employed any full-time employees since starting as a consultant in 1975.

TRIALS

Asner, trial, Baltimore s.c., Nov. 17
Asbestos Cases III, deposition, Charleston WV s.c., Dec. 8

1994

Dikun, trial, Ft. Lauderdale s.c., Jan. 24
Adams, trial, Baltimore s.c., Feb. 23, 24, 28
Gordon, deposition, Austin s.c., Feb. 25
Chavers, deposition, Mobile s.c., Mar. 24

Rones, deposition, Washington DC s.c., Apr. 21
 Hannon, trial, Baltimore s.c., Apr. 27
 Purcell, trial, Portland s.c., May 3-4
 Monahan, trial, L.A. s.c., June 16, 17, 20,21
 Norris, trial, Wilmington s.c., June 30
 One Wilshire, deposition, L.A. s.c., Sept. 13
 Chase Manhattan, deposition, New York f.c., Nov. 1; and Feb. 22, Oct. 9, 1995
 Olson, trial, Sydney Australia Dust Diseases Tribunal, Dec. 10-11

1995

Adams, deposition, Baltimore s.c., Jan . 5, 12
 Wiggins, trial, San Francisco s.c., Jan. 10-11
 One Wilshire, trial, Los Angeles s.c., Mar. 20, 22
 White, trial, Austin s.c., April 13
 In Re Asbestos, deposition, Travis Co. TX, May 12
 Bowser, trial, San Francisco s.c., May 26-27
 Adams, trial, Baltimore s.c., June 6-7
 Richmond, trial, San Francisco s.c., June 14
 Boyd, videotaped evidence deposition, Spokane s.c., June 29
 Automobile worker asbestos cases, deposition, Birmingham MI s.c., Sept. 7
 Hicks, deposition, Bloomington IL s.c., Oct. 12
 Zumas, trial, Baltimore s.c., Oct. 25, 30, Nov. 6
 Heisler, deposition, Cincinnati s.c., Oct. 26
 Dye, trial, San Francisco s.c., Oct. 31-Nov. 1
 White, trial, Austin s.c., Nov. 2
 Hicks, trial, Bloomington IL s.c., Nov. 7-8
 Sirbaugh, trial, Martinsburg WV s.c., Nov. 15
 Drake, trial, San Antonio s.c., Dec. 5

1996

Crabtree, trial, Bloomington IL s.c., Jan. 17
 Sloan, deposition, San Francisco s.c., Feb. 13
 Butler, deposition, San Francisco s.c., Feb. 14
 Lee, trial, Brunswick GA s.c., Mar. 14
 Chaney, deposition, San Antonio s.c., Mar. 14
 Greive, deposition, Baltimore s.c., April 2
 Sherer, trial, Bloomington s.c., May 20
 Pusey, trial, Wilmington s.c., May 23
 Anderson, deposition, Little Rock, June 6
 Danilowicz, trial, San Francisco s.c., June 13-14
 Williams, deposition, San Francisco, June 13
 Williams, trial, San Francisco s.c., June 14
 Roa, trial, Portland OR f.c., July 19
 Perepechko, deposition, Chicago s.c., Sept. 4
 White, deposition, El Paso, Sept. 5
 Biebel, trial, Baltimore, Oct. 7-9
 Buyard, trial, Los Angeles, Oct. 31
 Overly, deposition, San Francisco, Nov. 4
 Adams, videotaped deposition, Houston s.c., Nov. 6
 Overly, trial, San Francisco, Nov. 12-13
 Childress, deposition, San Francisco, Nov. 13
 Becknell, trial, Bloomington, Nov. 15, 18
 Childress, trial, San Francisco, Nov. 21, 22, 25
 Ronzini, trial, New York s.c., Dec. 9-10
 Scanlon, deposition, Chicago s.c., Dec. 20

1997

Arthur, trial, Wilmington s.c., Feb. 21, 24
 Ehret, deposition, L.A. s.c., May 9
 Crowe, trial, Cleveland s.c., May 21
 Ehret, trial, Los Angeles, May 28
 Driver, trial, Dallas s.c., June 25
 Derr, deposition, Wilmington, July 8
 Sanchez, trial, SF s.c., July 30-31
 Abshire, deposition, Charleston WV, Sept. 22
 Britton, French, depositions, Bloomington s.c., Oct. 13
 DeBolt, trial, Bloomington s.c., Nov. 13

Pruitt, trial, San Francisco s.c., Nov. 19
 Varga, trial, Fairfield CA s.c., Nov. 20
 Trujillo, deposition, Albuquerque s.c., Dec. 5
 Harpham, deposition, L.A. s.c., Dec. 6
 MONMASS, deposition, Morgantown WV, Dec. 29

1998

Armstrong, deposition, San Francisco s.c., Jan. 2
 Group 119, deposition, San Francisco s.c., Jan. 2
 Valadez, deposition, San Francisco s.c., Jan. 2,5
 Armstrong, trial, San Francisco s.c., Jan. 6
 Group 119, trial, San Francisco s.c., Jan. 7-8
 Burgess, trial, Bloomington, Jan. 26
 Schedel, trial, Bismarck ND, Jan. 28
 Valadez, trial, San Francisco, Feb. 4-5
 Burks, trial, San Francisco, Feb. 5-6
 Lowery, trial, Baltimore, Feb. 10
 Ball, trial, Cleveland s.c., Feb. 25
 Group 129, trial, San Francisco s.c., Mar. 9-11
 Group 131, deposition, San Francisco, Mar. 12
 Silveira, trial, San Francisco, Apr. 23
 Woods, deposition, Chatanooga s.c., Apr. 27
 Cosey, trial, Fayette MS s.c., May 21
 Brady, deposition, Buffalo NY s.c., June 4
 Gramley, trial, Cleveland, July 10
 Frost, deposition, Bloomington IL, July 28
 Cavitt, deposition, Cameron TX, July 30
 Padron, evidence deposition, Cameron TX, Aug. 3
 Charley, deposition, Cedar Rapids Iowa, Nov. 2
 Henderson, deposition, Charlotte NC, Nov. 9
 Corbal, deposition, L.A., Dec. 8
 Briggs, evidence deposition, Beaumont TX, Dec. 10
 1999
 Missik, deposition, Cleveland s.c., Jan. 15
 Lilienthal, deposition, San Francisco s.c., Jan. 16-17, Feb. 15
 Salke, deposition, Bridgeport CT s.c., Feb. 11
 Luevano, deposition, Oakland CA s.c., Feb. 19
 Lilienthal, trial, S.F. s.c., Feb. 22
 Raper, deposition, Dallas s.c., Mar. 11-12
 Brittin, trial, Bloomington s.c., Mar. 15-16
 Sanford, deposition, S.F. s.c., Apr. 2
 Harris, deposition, Waycross GA s.c., Apr. 14
 Zeleny, deposition, Chicago s.c., Apr. 15
 Epperson, trial, Dallas s.c., May 28
 Taylor, deposition, San Francisco s.c., June 1, 1999
 Malang, deposition, San Francisco s.c., June 2
 Townes, trial, Augusta GA s.c., June 16
 Shank, trial, Cleveland s.c., June 28-29
 Sanchez, trial, El Paso s.c., July 29
 Rasmussen, deposition, San Francisco s.c., Aug. 9, 15
 Powell, deposition, San Francisco s.c., Aug. 24
 Miller, deposition, Bloomington s.c., Aug. 30
 Thompson, deposition, Seattle s.c., Aug. 31
 Widing, deposition, San Francisco s.c., Sept. 10
 Ball, trial, Cleveland s.c., Sept. 23
 Albright, deposition, Salisbury NC s.c., Oct. 3
 Thompson, trial, Everett WA s.c., Oct. 12
 Jones, deposition, Cleveland s.c., Oct. 22
 Hoppmann, deposition, San Francisco s.c., Oct. 22
 Chiasson, deposition, Los Angeles s.c., Nov. 2
 Castillo, trial, El Paso s.c., Nov. 4
 Raigoza, deposition, S.F. s.c., Nov. 11
 Haig, deposition, S.F. s.c., Nov. 15
 Hart, trial, Canton NY, Nov. 16
 Hedrick, deposition, Fayette MS, Nov. 22
 Grizzle and McElheney, depositions, S.F. s.c., Nov. 23
 Gotter, deposition, Bloomington, Dec. 17

Cicchillo, trial, Cleveland s.c., Jan. 27
 Hollis, trial, Wilmington s.c., Feb. 1
 Chavers, deposition, S.F. s.c., Feb. 2-3
 Chavers, trial, S.F. s.c., Feb. 3
 Peralta, deposition, El Paso s.c., Mar. 27
 McLeod, deposition, Buffalo NY s.c., Apr. 3
 Hines, deposition, SF s.c., May 1
 Burnside, deposition, WV s.c., May 12
 Ockerman, deposition, Oakland CA s.c., May 21
 Ockerman, trial, Oakland s.c., May 22
 Thornton, deposition, San Francisco s.c., June 9
 Kasun, trial, Milwaukee s.c., June 16 and 19
 Pavolini, deposition, San Francisco s.c., June 21
 Pavolini, trial, San Francisco, June 25-26
 Davis, deposition, San Francisco s.c., June 26
 Perez, trial, San Francisco s.c., July 10-12
 Tolbertson, trial, San Francisco s.c., July 20
 Chiasson, trial, Los Angeles s.c., Aug 1-2
 Chiasson, deposition, L.A. s.c., Aug. 1
 Davis, trial, San Francisco s.c., Aug. 21-22
 Pickle, deposition, San Francisco s.c., Aug. 25
 Atchison, deposition, Oakland CA s.c., Aug. 28
 Kinsman, deposition, San Francisco s.c., Sept. 7
 Bouldin, deposition, Houston s.c., Sept. 8
 Lyons, deposition, Washington D.C., Sept. 11
 Emrick, trial, Portland OR s.c., Oct. 9-11
 Moro, deposition, San Francisco s.c., Nov. 7
 Moore, deposition, Daingerfield TX s.c., Dec. 29

Gault, deposition, San Francisco s.c., Jan. 3
 Overly, trial, San Francisco s.c., Jan. 5
 Vasen, deposition, SF s.c., Jan. 8
 Jestes, deposition, SF s.c., Jan. 12
 Hoskins, deposition, Kansas City MO s.c., Jan. 22
 Jacobs, deposition, Dallas s.c., Jan. 25
 Lambertson, deposition, SF s.c., Feb. 13
 Hoskins, trial, Kansas City MO s.c., Mar. 1
 Jones, trial, New York NY s.c., Mar. 2
 Dunn, trial, Oakland CA s.c., Mar. 5
 Lee, trial, SF s.c., Mar. 6
 Edwards, deposition, SF s.c., Mar. 8 and 12
 Watkins, deposition, SF s.c., Mar. 9
 Highsmith, deposition, Brunswick GA s.c., Mar. 21
 Branscum, deposition, SF s.c., Mar. 27
 Padalecki, deposition, Houston s.c., Apr. 9
 Kingsland, trial, New York s.c., May 21
 Peterman, trial, Portland OR s.c., May 24-25
 Alexander, deposition, SF s.c., May 29
 Chandler, deposition, SF s.c., June 11
 Calhoun, deposition, Bloomington s.c., June 25
 Smith, trial, SF s.c., June 27
 Stanfill, deposition, SF s.c., July 2
 Wass, deposition, Seattle s.c., July 13
 Rasmussen, deposition, SF s.c., July 16
 Thompson, deposition, El Paso s.c., July 20
 Moore, trial, Daingerfield TX s.c., July 23
 Shingle, deposition, SF s.c., July 24
 Miller, deposition, Salisbury NC s.c., July 27
 Carter, deposition, SF s.c., Aug. 3
 Shingle, trial, SF s.c., Aug. 14
 Novo, deposition, Baltimore s.c., Aug. 17
 Hunt, deposition, Bloomington IL s.c., Aug. 21
 Petruzzelli, deposition, New Haven CT s.c., Aug. 24
 Steenberger, deposition, Marshall TX s.c., Aug. 24
 Alber, deposition, Boulder CO s.c., Aug. 27

Amos, deposition, Charleston WV s.c., Aug. 28
 Book, deposition, S.F. s.c., Sept. 6
 Skinner, deposition, Austin s.c., Sept. 7
 Peterson, deposition, SF s.c., Sept. 14
 Kiber, trial, Bloomington IL s.c., Sept. 25
 Kinsman, deposition, Seattle s.c., Sept. 28
 Turley, deposition, S.F. s.c., Sept. 28
 Henderson, trial, Greenville SC s.c., Oct. 10
 Dressler, deposition, SF s.c., Oct. 12
 Jernigan, trial, Wilmington s.c., Oct. 16
 Wilson, trial, Baltimore s.c., Oct. 18
 Gerke, deposition, Oakland s.c., Oct. 19, 24
 Stringfellow, trial, Little Rock s.c., Oct. 23
 Colwell, deposition, Oakland CA s.c., Oct. 23
 Guerra, deposition, Oakland CA s.c., Oct. 26
 Cargile, deposition, Baltimore s.c., Oct. 30
 Kiber, trial, Bloomington s.c., Nov 5
 Elliott, deposition, S.F. s.c., Nov. 6
 Henderson, deposition, Oakland CA s.c., Nov. 6
 Weiner, trial, Bethlehem PA s.c., Nov. 28
 Jordan, deposition, San Francisco s.c., Dec. 11
 Wells, deposition, SF s.c., Dec. 13
 Brown, deposition, SF s.c., Dec. 14

2002

Jacques, deposition, Chicago f.c., Jan. 3
 Campbell, deposition, SF s.c., Jan. 15
 Franklin, deposition, SF s.c., Jan. 15
 Burns, trial, SF s.c., Jan. 22
 Todak, deposition, SF s.c., Feb. 14
 Jones, trial, Atlanta s.c., Feb. 20
 Tolbertson, deposition, SF s.c., Mar. 1
 Meiers, deposition, Cleveland s.c., Mar. 4
 Meiers, trial, Cleveland s.c., Mar. 7
 Todak, trial, SF s.c., Mar. 12-13
 Totman, deposition, Providence s.c., Mar. 21
 Cave, deposition, SF s.c., Mar. 22
 Peterson, deposition, Oakland s.c., Mar. 29
 Matteson, deposition, New York s.c., Apr. 22
 Matteson, trial, New York s.c., Apr. 24
 Brown, deposition, Oakland CA s.c., Apr. 25
 Anderson, deposition, Port Gibson MS s.c., Apr. 26
 Flores, deposition, Corpus Christi TX s.c., May 1
 Farrell, deposition, SF s.c., May 3
 Peterson, trial, Oakland s.c., May 6
 Anderson, deposition, Port Gibson MS s.c., May 10
 Kuhn, deposition, SF s.c., May 17
 Robinson, deposition, SF s.c., May 20
 Trinchese, deposition, SF s.c., June 3, 17
 Rivenbark, trial, Galveston s.c., June 4-5
 Highsmith, trial, Atlanta s.c., June 6
 Bennett, deposition, SF s.c., June 7
 Caruso, trial, Springfield IL s.c., June 11-12
 McCarthy, deposition, Los Angeles s.c., June 19
 Trinchese, trial, SF s.c., June 28
 Roca, deposition, Wilmington DE s.c., July 1-2
 McCarthy, trial, Los Angeles s.c., July 11
 Sledz, deposition, Baltimore s.c., July 29
 Schmidt, deposition, Cleveland s.c., Aug. 5
 Probst, deposition, Cleveland s.c., Aug. 9
 Jensen, deposition, SF s.c., Aug. 16
 Otten, deposition, SF s.c., Aug. 19
 Nelson, deposition, SF s.c., Aug. 21
 Barry, deposition, Galveston TX s.c., Aug. 29
 Hansen, deposition, SF s.c., Aug. 30
 Anderson, trial, Port Gibson MS s.c., Sept. 5
 Frederick, deposition, SF s.c., Sept. 16

Kuhl, deposition, SF s.c., Sept. 17 and July 25, 2003
 Langford, deposition, Center TX s.c., Sept. 26
 Graham, deposition, S.F. s.c., Oct. 2
 Lansford, trial, Center TX s.c., Oct. 9
 Gunderson, deposition, SF s.c., Oct. 11
 Shauan, deposition, Providence RI s.c., Oct. 14
 Campbell, trial, Seattle s.c., Oct. 17
 Vincent, deposition, Wilmington s.c., Oct. 21
 Flood, deposition, Chicago s.c., Nov. 7
 Bottner, deposition, S.F. s.c., Nov. 8
 Couch, deposition, S.F. s.c., Nov. 8
 Consolini, deposition, Providence RI s.c., Nov. 18
 Gunderson, trial, S.F. s.c., Nov. 19
 Scott, deposition, S.F. s.c., Nov. 19
 Wallstrom, deposition, S.F. s.c., Nov. 20
 Wirt, deposition, Dallas s.c., Nov. 25
 Yoakum, deposition, Cameron TX s.c., Nov. 26
 Rhynes, deposition, S.F. s.c., Nov. 27
 Skelton, deposition, S.F. s.c., Dec. 2
 Miller, trial, Austin s.c., Dec. 3-4
 Cash, trial, Wilmington s.c., Dec. 10
 Kruchuk, deposition, SF s.c., Dec. 27

2003

Kubik, deposition, Warren OH s.c., Jan. 3
 Clark, deposition, S.F. s.c., Jan. 13
 Hofstetter, deposition, Alton IL s.c., Jan. 16
 Sargent, deposition, Amarillo s.c., Jan. 17
 Roseman, trial, Indianapolis s.c., Jan. 21-22
 Falcone, trial, New Haven CT s.c., Jan. 30
 Wells, deposition, S.F. s.c., Jan. 31
 Kavanaugh, deposition, West Palm Beach s.c., Feb. 1
 Kubik, trial, Warren OH s.c., Feb. 4
 Davis, trial, Cleveland s.c., Feb. 5
 Richardson, deposition, SF s.c., Feb. 6
 Lundsford, trial, SF s.c., Feb. 14
 Kavanaugh, trial, Palm Beach FL, Feb. 18
 Lee, deposition, SF s.c., Feb. 21
 Niemeier, deposition, SF s.c., March 3
 Mintz, deposition, SF s.c., March 7
 Dexter, deposition, NYC s.c., March 17
 Sparks, trial, Beaumont TX s.c., March 26-27
 Lilly, deposition, Charleston WV s.c., Apr. 4
 Griffith, deposition, SF s.c., Apr. 30
 Curtright, deposition, SF s.c., May 1
 Wajer, deposition, Baltimore s.c., May 9
 Kelley, deposition, SF s.c., May 12
 Green, deposition, SF s.c., May 19
 Marr, deposition, Dallas, May 23
 Brackett, deposition, Orange TX, May 27, 2003
 Lukac, trial, Warren OH, May 28
 Pernowsky, deposition, Cleveland s.c., May 29
 Toma, deposition, SF s.c., June 2
 Gomez, trial, NY s.c., June 3, 9
 Gartner, trial, Minneapolis s.c., June 19-20
 Miller, deposition, Bloomington IL s.c., June 24
 Andrade, deposition, SF s.c., June 24
 Connor, deposition, SF s.c., June 24
 Prasel, deposition, Cameron TX s.c., July 1-2, 21-22
 Marshall, trial, Alameda CA s.c., July 9
 Bangs, deposition, SF s.c., July 14
 Tripp, deposition, SF s.c., July 25
 Robinson, trial, Marietta GA, Aug. 12
 Nolan, deposition, Chicago s.c., Aug. 15
 Keyser, deposition, SF s.c., Aug. 22
 Waishes, deposition, Wilmington s.c., Sept. 8
 Wirts, trial, Baltimore s.c., Sept. 15

Bertucci, deposition, New Orleans, Oct. 3
 Huck, deposition, Oakland s.c., Oct. 6
 Weller, deposition, Cleveland s.c., Oct. 20
 Robinson, deposition, Wilmington s.c., Oct. 21
 Martin, deposition, Houston s.c., Oct. 24
 Davis, deposition, Houston s.c., Oct. 31
 Polito, trial, Rochester NY s.c., Nov. 21
 Anzulis, deposition, Baltimore s.c., Nov. 24
 Mikolich, deposition, SF s.c., Dec. 1
 Chauvin, deposition, New Orleans s.c., Dec. 15
 Lombardo, deposition, San Francisco s.c., Dec. 16
 Baker, deposition, San Francisco s.c., Dec. 16
 Ward, deposition, Belton TX s.c., Dec. 19
 2004
 Jameson, deposition, Seattle, Jan. 6
 Harris, deposition, SF s.c., Jan. 12
 Ross, deposition, SF s.c., Jan. 16
 Smith, deposition, SF s.c., Jan. 16
 Williamson, deposition, Jacksonville s.c., Jan. 19
 Douglas, deposition, Orange TX s.c., Jan. 20
 Jones, deposition, New Orleans s.c., Jan. 26
 Ford, deposition, Wilmington s.c., Jan. 30
 Korenek, deposition, Cameron TX s.c., Feb. 9
 Amento, trial, Philadelphia s.c., Feb. 10
 Munro, deposition, Indianapolis s.c., Feb. 23
 Mason, deposition, Beaumont s.c., Feb. 24
 Logston, deposition, Louisville s.c., Mar. 1
 Stephens, trial, Angleton TX, Mar. 2
 Prather, trial, Dallas, Mar. 3
 Dori, deposition, Sweetwater TX s.c., Mar. 5
 Kubic, deposition, Warren OH s.c., Mar. 8
 Wise, trial, SF s.c., March 11
 Dori, trial, Sweetwater TX s.c., Mar. 19
 Braden, deposition, SF s.c., Mar. 22
 Stover, deposition, SF s.c., Mar. 22
 Donahue, deposition, SF s.c., Mar. 28
 Roberts, deposition, Houston s.c., Apr. 2
 Rhines, deposition, Covington Co. MS, Apr. 12
 Burdo, deposition, Cleveland s.c., May 7
 Whitney, trial, Los Angeles s.c., May 19
 Mills, deposition, Corpus Christi, June 1
 Kolson, trial, Ebensburg PA, June 9
 Compton, deposition, Bloomington, June 10
 Garzee, trial, Peoria, June 11
 Wilson, deposition, SF s.c., June 14
 Coleman, deposition, Cleveland s.c., June 25
 Carter, deposition, Oakland s.c., July 2
 Kell, deposition, SF s.c., July 13
 Odum, deposition, Copiah Co. MS, July 16
 Carter, trial, Oakland s.c., July 20
 Wilson, deposition, SF s.c., July 27
 Hinchman, deposition, Houston s.c., July 28
 Kruger, deposition, SF s.c., Aug. 3
 Pisani, deposition, SF s.c., Aug. 9
 Pretko, deposition, Dallas s.c., Aug. 20
 Kennedy, deposition, Portsmouth OH, Aug. 26
 Ocegueda, deposition, SF s.c., Sept. 7
 Lorenzino, deposition, Oakland s.c., Sept. 24
 Barone, deposition, Warren OH s.c., Sept. 27
 Cameron, deposition, Bloomington IL s.c., Sept. 30
 Tracy, deposition, Oakland s.c., Oct. 1
 Anthony, trial, NY s.c., Oct. 6
 Gadeleta, trial, NY s.c., Oct. 8
 Bearer, deposition, SF s.c., Oct. 11
 Giesick, deposition, SF s.c., Oct. 11
 Bishop, deposition, New Orleans s.c., Oct. 18
 Marco, deposition, St. Louis s.c., Oct. 19
 Cullison, deposition, Austin s.c., Oct. 22

Gendreau, deposition, SF s.c., Nov. 4
 Coen, deposition, Milwaukee s.c., Dec. 20
 Brown, deposition, SF s.c., Dec. 21
 McWard, deposition, Peoria s.c., Dec. 23

2005

Bruner, deposition, SF s.c., Jan. 10
 Hamilton, trial, Cleveland s.c., Jan. 13
 Zavacky, deposition, Cleveland s.c., Jan. 14
 Walsh, deposition, S.F. s.c., Jan. 14
 Walraven, deposition, Boston s.c., Jan. 17
 Hargrave, deposition, Edwardsville IL, Jan. 31, Feb. 28
 Auckland, deposition, Cleveland s.c., Feb. 4
 Poore, deposition, Houston s.c., Feb. 7
 Plathe, trial, St. Paul s.c., Feb. 16
 Bruner, trial, SF s.c., Feb. 23
 Miller, deposition, SF s.c., Mar. 14, 30
 Flax, deposition, Baltimore s.c., Mar. 21
 Hoover, deposition, SF s.c., Apr. 5
 Konecny, deposition, SF s.c., Apr. 8
 Coffey, trial, Buffalo s.c., April 13
 Bouhanna, deposition, Boston s.c., Apr. 15
 Clark, deposition, SF s.c., May 19
 Pendergast, deposition, NY s.c., May 20
 Rizzi, trial, NY s.c., May 26–27, 31
 Goodman, deposition, Tacoma s.c., June 10
 Nisselius, deposition, SF s.c., July 7
 O'Halloran, deposition, SF s.c., July 8
 Hartford, deposition, SF s.c., July 11
 Lightsee, deposition, Brunswick GA s.c., July 15
 Ammons, deposition, Brunswick s.c., July 15
 Dawson, trial, Wilmington s.c., July 14 and 18
 Cotton, deposition, Beaumont s.c., Aug. 5
 Grisez, deposition, SF s.c., Aug. 10
 Lantz, trial, SF s.c., Aug. 12
 Ballenger, deposition, SF s.c., Aug. 15
 Dukes, deposition, Bloomington s.c., Aug. 17
 Schadt, deposition, Edwardsville IL s.c., Sept. 1
 Coca, deposition, SF s.c., Sept. 2
 Orlando, trial, NY s.c., Sept. 8
 Kleineke, deposition, Cleveland s.c., Sept. 9
 Dukes, trial, Bloomington IL s.c., Sept. 26
 Lightsee, trial, Atlanta f.c., Sept. 27
 Barnhill, deposition, SF s.c., Sept. 29
 Hicks, deposition, Newport News s.c., Sept. 30
 Dodson, deposition, Kansas City MO s.c., Oct. 4
 Fletcher, deposition, NY s.c., Oct. 14
 Richardson, deposition, Baltimore s.c., Oct. 28
 Jellum, trial, St. Paul s.c., Nov. 8
 White, trial, Bloomington IL s.c., Nov. 15–16
 Franklin, deposition, Louisville s.c., Nov. 18
 Riggle, deposition, Dallas s.c., Nov. 21
 Adamson, deposition, Atlanta s.c., Nov. 22
 Demster, deposition, SF s.c., Dec. 5
 Cerny, deposition, Cleveland s.c., Dec. 6
 Parsons, deposition, Ft. Lauderdale s.c., Dec. 9
 Saenz, deposition, Cameron TX s.c., Dec. 19
 Jacobelly, deposition, SF s.c., Dec. 22
 Thalman, deposition, Galveston s.c., Dec. 27

2006

Whiting, deposition Cleveland s.c., Jan. 4
 Pisani, deposition, SF s.c., Jan. 5
 Konecny, deposition, SF s.c., Jan. 9
 Potts, deposition, Cleveland s.c., Jan. 13
 Horr, trial, Oakland s.c., Jan. 18
 Robinson, deposition, Angleton TX, Jan. 30

Thalman, trial, Galveston s.c., Feb. 1
 Betti, deposition, SF s.c., Feb. 13
 Smyth, trial, NY s.c., Feb. 16
 Stroker, deposition, Oakland s.c., Feb. 21
 Ryan, trial, Edwardsville IL s.c., Feb. 23–24
 Woolston, deposition, Wilmington s.c., Feb. 27
 Garrison, trial, Cleveland s.c., Mar. 1
 Wallace, trial, Austin s.c., Mar. 2
 Kovacevich, deposition, Houston s.c., Mar. 3
 Troncali, deposition, Galveston s.c., Mar. 10
 Jagid, deposition, New Brunswick NJ s.c., Mar. 13
 Hellen, trial, Angleton TX s.c., Mar. 16
 Brent, deposition, Edwardsville IL s.c., Mar. 22
 Gortney, deposition, Beaumont TX s.c., Mar. 23
 Slanina, deposition, Houston s.c., Mar. 24
 Gregory, deposition, Kansas City MO s.c., Apr. 3
 Miller, deposition, SF s.c., Apr. 6
 Stone, deposition, Great Falls MT s.c., Apr. 10
 Terrance, deposition, Baton Rouge s.c., Apr. 11
 Burgeson, deposition, SF s.c., Apr. 12
 Halsema, deposition, Oakland s.c., Apr. 13
 Campbell, deposition, SF s.c., Apr. 13, 18
 Miller, deposition, SF s.c., Apr. 17
 Spurgeon, deposition, Edwardsville s.c., May 1
 Sells, deposition, Cleveland s.c., May 3
 Faulkoner, deposition, Wagoner OK s.c., May 8
 Robinson, trial, Houston s.c., May 10
 Flexner, trial, NY s.c., May 12
 Fulton, deposition, SF s.c., May 15
 Finnefrock, deposition, Cleveland s.c., May 18
 King, deposition, Angleton TX s.c., May 19
 Dancho, deposition, Chicago s.c., May 22
 Haanstra, deposition, SF s.c., May 26
 Bolen, trial, Garden City NY s.c., May 30
 Giero, deposition, Los Angeles s.c., June 1
 Jones, deposition, SF s.c., June 5
 Gibson, deposition, Beaumont s.c., June 19
 Loboda, deposition, NY s.c., June 26
 Pitts, deposition, Fredericksburg VA s.c., June 28
 Price, deposition, Oakland s.c., July 10, Aug. 12
 Jones, trial, Newport News VA s.c., July 12
 Jones, trial, SF s.c., July 31
 Poindexter, deposition, Angleton TX s.c., Aug. 3
 Christian, deposition, SF s.c., Aug. 10
 Sutterfield, deposition, Houston s.c., Aug. 11
 Hoser, deposition, New Brunswick NJ s.c., Aug. 14
 Hegele, deposition, SF s.c., Aug. 17
 Reese, trial, Bloomington IL s.c., Aug. 28
 Siegwald, deposition, Dallas s.c., Aug. 29
 Ferrera, deposition, Dallas s.c., Aug. 29
 Bergin, deposition, SF s.c., Sept. 7
 Adair, deposition, Orange TX, Sept. 8
 Copenhaver, deposition, Dallas s.c., Sept. 8
 Ard, deposition, Beaumont s.c., Sept. 8
 Price, trial, Oakland s.c., Sept. 11
 Pounds, deposition, SF s.c., Sept. 11
 Homewood, deposition, Houston s.c., Sept. 18
 Voight, deposition, Houston s.c., Sept. 18
 Lindquist, deposition, Providence RI s.c., Sept. 22
 Anderson, deposition, Denver s.c., Sept. 25
 Rodriguez Negrón, deposition, L.A. s.c., Sept. 28
 Sheffield, deposition, Oakland s.c., Oct. 2
 Shreiner, trial, Wilmington s.c., Oct. 3–4
 Colella, trial, New York s.c., Oct. 10, 12, 17
 Luckey, deposition, Houston s.c., Oct. 13
 Hewitt, deposition, SF s.c., Oct. 16
 Whitlock, deposition, SF s.c., Oct. 16
 Boyer, trial, Boston s.c., Oct. 20

Johnson, deposition, Memphis s.c., Oct. 23
 Cable, deposition, Bridgeport CT, Oct. 24
 Hogan, deposition, Oakland s.c., Nov. 6
 Stewart, trial, Wilmington s.c., Nov. 9
 Oney, deposition, Houston s.c., Nov. 13
 Blessing, trial, Bloomington s.c., Nov. 16
 Young, deposition, Seattle s.c., Nov. 20
 Whitlock, trial, SF s.c., Nov. 21
 Duncan, deposition, Dallas s.c., Dec. 12
 Morell, deposition, Edinburg TX s.c., Dec. 19

2007

Anzulis, deposition, Baltimore s.c., Jan. 5
 Dodd, deposition, Edwardsville IL s.c., Jan. 8
 Duncan, deposition, Edwardsville IL s.c., January 16
 Boyle, deposition, SF s.c., Jan. 18
 Link, deposition, Cleveland s.c., Jan. 19
 Knight, deposition, Houston s.c., Jan. 22
 Gomez Gonzales, trial, NY s.c., Jan. 24
 Foster, deposition, Angleton TX s.c., Jan. 26
 Malcolm, deposition, Bloomington IL, Jan. 29
 Lathrop, deposition, SF s.c., Feb. 2
 Drinkwater, deposition, SF s.c., Feb. 2
 Jones, deposition, Boston s.c., Feb. 5
 Pollard, trial, Galveston s.c., Feb. 7
 Metzger, deposition, Wilmington s.c., Feb. 12
 Irvin, trial, Edmonton KY s.c., Feb. 15
 Lee, deposition, Salisbury NC, Feb. 20
 Pinedo, deposition, SF s.c., Feb. 26
 Melon, deposition, Dallas s.c., Mar. 2
 Murray, trial, Oakland s.c., Mar. 5
 Farmer, deposition, SF s.c., Mar. 9
 Rincon, trial, SF s.c., Mar. 16,19
 Ridgley, deposition, Baltimore s.c., Mar. 22
 Graves, deposition, Edwardsville IL, Mar. 23
 Monroe, trial, Edwardsville IL, Mar. 27
 Beckler, deposition, Dallas s.c., Mar. 30
 Bock, deposition, Richmond s.c., Apr. 2
 Melon, trial El Paso s.c., Apr. 5
 Justice, deposition, Wilmington s.c., Apr. 9
 Martin, trial, NY s.c., Apr. 25-26
 Cox, trial, Cleveland s.c., May 4
 Rodamer, deposition, SF s.c., May 7
 Felker, trial, SF s.c., May 8
 Passig, deposition, SF s.c., May 10
 Asworth, deposition, Orange TX s.c., May 14
 Heppe, trial, Bloomington s.c., May 15
 Gilson, deposition, Atlanta s.c., May 18
 Lucadamo, deposition, Providence s.c., May 25
 Stirm, deposition, SF s.c., June 4
 Ormonde, deposition, SF s.c., June 6
 Dachauer, deposition, SF s.c., June 11
 Matel, deposition, SF s.c., June 21
 Cook, deposition, Baltimore s.c., June 22
 Buttitta, deposition, Hackensack NJ, July 2
 Dachauer, trial, SF s.c., July 9-10
 Scott, deposition, SF s.c., July 13
 Gardner, deposition, Houston s.c., July 16
 Eubanks, deposition, SF s.c., July 17, 25
 Venturini, trial, Bloomington s.c., July 18-19
 Lagrone, deposition, Wilmington s.c., July 23
 Lagrone, trial, Wilmington s.c., July 24

Senator BOXER. Thank you, sir. Thank you very much.

Now, Dr. Ann Wylie, University of Maryland Department of Geology.

**STATEMENT OF ANN G. WYLIE, PH.D., PROFESSOR OF
GEOLOGY, UNIVERSITY OF MARYLAND**

Ms. WYLIE. Thank you, Madam Chairman and members of the committee.

I am pleased to be here today to speak to you about definitions, in particular, the definition of asbestos and the definition of asbestos fiber. I have been a professor for 35 years, and I have developed over this time expertise on the properties of minerals that produce human disease when inhaled. I provided a written text from which the following short summary is taken.

The Federal definitions of fiber and asbestos both date back to the early 1970s. Let me first address the definition of fiber.

As defined, a fiber is any particle that fits into a particular size and shape category. The category is large, and it includes a wide range of particle sizes and shapes. Included in this range are rock fragments as well as asbestos fibers.

The size and shape category is not specific for asbestos. These non-asbestos particles that fit this category are very common. They may be found in bedrock in large portions of the United States. Epidemiological studies of miners exposed to these particles have found no excess of asbestos-related diseases.

The first pictures that I have shown over here are particles in both of these that fit the definition of fiber. The one on the left is asbestos; the one on the right is rock fragments. Rock fragments meeting the fiber definition from South Dakota are shown in this photograph. This is the site at Leeds, SD of one of the negative studies for asbestos-related diseases among the miners.

Asbestos is well-studied and well-characterized. A better dimension definition of asbestos fiber would be relatively simple to construct.

I also mention the definition of asbestos. The Federal description of asbestos, in my view, needs to be amended. It needs to explicitly include these asbestos fibers from Libby, MT. These are the mineral winchite, and it is not listed in the Federal regulatory definition of the minerals that make up asbestos. As you can see from this photomicrograph, this is actually an electron micrograph, these are clearly asbestos.

A more comprehensive description of asbestos and an accurate, scientific definition of asbestos fiber will exclude non-asbestos particles. They can be incorporated into regulatory policy without compromising protection against asbestos-related diseases. I would be happy to answer questions.

[The prepared statement of Dr. Wylie follows:]

STATEMENT OF ANN G. WYLIE, PH.D., PROFESSOR OF GEOLOGY AT THE
UNIVERSITY OF MARYLAND

My name is Ann G. Wylie. I hold a baccalaureate degree from Wellesley College and a Ph.D., from Columbia University. I am Professor of Geology at the University of Maryland. I have spent more than 30 years studying asbestos and the minerals that compose it.

I am here today to discuss both the scientific and the federal regulatory definition of asbestos.

REGULATORY HISTORY

In the early 1970s the United States lagged behind the rest of the world in the strict regulation of occupational exposure to airborne asbestos. Regulation of asbes-

tos was one, if not the first, major initiative of both EPA and OSHA when they were formed at this time. Needless to say, these two agencies were in a hurry.

OSHA wrote a definition of asbestos and specified a method for its measurement; both were incorporated into law. Together these comprise the federal regulatory definition of asbestos.

The federal regulatory definition was written without any consultation with the mineral experts at the United States Geological Survey or the U.S. Bureau of Mines, and, consequently, it was not mineralogically correct.¹

OSHA's regulatory definition identified mineral names without specifying the asbestiform character. This is the same as saying that hail and snow are the same thing. Both are ice, but everyone knows that they are not the same and that have different potentials for harm.

The measurement method, called the membrane filter method², compounded the definitional problem. The foundation for the membrane filter method was developed in the 1960s in British factories that utilized asbestos. The particles included in exposure estimates were specified by both a minimum length and a minimum length to width ratio. A length of >5 micrometers was chosen to reflect an acceptable level of reproducibility among analysts.³ A length to width ratio of 3:1 was also specified, but its choice was not explained. Whatever the reason, 3:1 was arbitrary. It is not a scientific definition of a fiber, it does not reflect the length to width ratio of asbestos fibers, and it was not chosen because of any studies linking it to health effects.

Because of the membrane filter method, particles longer than 5 micrometers with a length to width ratio of 3:1 or higher meet what has become known as the Regulatory Fiber Definition (RFD). They are also referred to as "federal fibers."

The effect of these two specifications, a mineralogically incorrect definition of asbestos and the development of an arbitrary Regulatory Fiber Definition (RFD), is that sometime during the 1970s, rock fragments, sometimes called cleavage fragments, became fibers and fragments of six minerals became *de facto* asbestos.

In 1992, OSHA examined this issue in detail. They concluded that there was no scientific evidence that cleavage fragments have the same health potential as asbestos. OSHA removed them from the asbestos standard.⁴ I am not aware of any epidemiological, animal or cellular studies that have been done since the OSHA decision that would change this conclusion.

NIOSH disagreed with OSHA, and up to this time, it has been the practice of NIOSH to assume that the RFD describes the size and shape of fibers that correlate with their potential to cause human disease⁵. The RFD was also recently applied by EPA in the El Dorado Hills, CA, study. It is clear that there is disagreement within the regulatory community of the appropriateness of the RFD in the protection of health.

NIOSH has just opened this question for study.⁶ This year, NIOSH issued a White Paper outlining in detail a research agenda to examine this question and held public hearings on it last month. The adverse health effects of asbestos are widely known and, with the exception of the differences between chrysotile-asbestos and amphibole-asbestos, are not in dispute. What the NIOSH White Paper addresses is the need to examine the health effects of nonasbestos particles that meet the RFD.

While the NIOSH White Paper does not provide evidence that challenges OSHA's 1992 decision, it calls for study of the issue, including, animal inhalation studies, epidemiological studies of miners, and cell culture studies. These are necessary before the health effects of nonasbestos particles that meet the RFD can be understood fully.

Why is this issue still in debate after the 1992 OSHA decision? Partly, I believe, that it comes from (1) lack of knowledge about the nature of asbestos, (2) acceptance of the hypothesis that only the size, shape, and durability of mineral particles affect their carcinogenic potential, and (3) a reluctance to change.

¹ OSHA's list of asbestos is also incomplete. One very public effect of the latter mistake is that most of the asbestos occurring at Libby Montana is not technically covered by asbestos regulations. (Verkouteren and Wylie, 2000)

² Leidel et al., 1979

³ Addingley, C.F., 1966; Lynch et al., 1970

⁴ OSHA, 1992

⁵ NIOSH, 2007

⁶ NIOSH, 2007

THE NATURE OF ASBESTOS

Asbestos is unusual.⁷ It is a mineral habit, like snow and hail are habits of ice. Habit is a form of "growth".

Asbestos grows as bundles of single fibers, (referred to as fibrils), that are easily separated from each other by hand pressure. The geologic environment that enables asbestos to form is limited and involves the presence of warm, water-rich conditions and open underground spaces.

Fibrils have narrow widths and extraordinary tensile strength imparted to them by their strong outer layers. They are difficult to break and their strength makes them flexible and almost impossible to grind. They are able to enter the body because of their narrow widths and they are retained because their lengths (as much as several hundred micrometers) thwart the body's mechanisms to remove them.

Asbestos can form from a number of different minerals. A mineral name implies only a particular atomic arrangement of a fixed set of elements in particular proportions. Mineral names are not synonyms for asbestos, just like ice is not a synonym for snow although snow is made of ice. To specify asbestos, the mineral name is followed by the term asbestos, e.g., tremolite-asbestos. Two forms of asbestos have a specific name, e.g., crocidolite is riebeckite-asbestos, and amosite is cummingtonite-grunerite asbestos.

The dimensions of asbestos fibrils found in occupational air and in the lung of asbestos workers are published in the literature, providing the basis for a dimensional definition of asbestos fibers. Although accurate dimensional definitions of asbestos may have been unnecessary in monitoring asbestos factories, mills and mines where what was in the air was only asbestos, they are essential in a mixed dust environment, essential when dealing with environmental exposures, and essential if asbestos were to be banned in the United States

Published data on the width of asbestos fibers found in bulk samples, on air monitoring filters, and in lung tissue show that asbestos is composed of mineral fibrils that are less than 1 micrometer in width.⁸ Fibrils wider than 1 micrometer are brittle (lack tensile strength) and cannot be used as asbestos.⁹ The widths vary somewhat within and among asbestos deposits, but the range is narrow. The dimensions of the most abundant forms of asbestos are similar: crocidolite fibrils are about 500 to 2000 A in width, amosite and anthophyllite-asbestos are about 2000 to 10,000 A in width, and chrysotile-asbestos is about 200–650 A.¹⁰

Other types of asbestos have equally narrow widths. Actinolite-asbestos has fibril widths of 600–2000 A and tremolite-asbestos fibrils range from about 2000 to 6000 A. At Libby Montana, mean widths are about 5000A and the range is 2000 to about 10,000A.¹¹

Studies of the lung burden of asbestos workers also report very narrow fibers. Martha Warnock measured 3723 fibers from lung tissue from 27 mesothelioma cases and identified them as crocidolite, tremolite-asbestos, anthophyllite-asbestos, actinolite-asbestos, chrysotile-asbestos, amosite, or other by TEM. More than 60 percent of the fibers are either amosite or chrysotile-asbestos. The mean width of the entire population was 2600 A; for amosite it was 2300 A and for chrysotile-asbestos, 600 A. Similar dimensions were observed by Warnock in asbestosis and lung cancer cases.¹²

The width of asbestos fibers is independent of length.¹³ Width is the same no matter how long the fibers because width is an independent characteristic imparted during the "growth" of the fibers.

Berman et al.¹⁴ extensive and careful evaluation of the 13 different rat experiments conclude that the fibers that contribute to tumor risk are <4000A in width or they are bundles and aggregates of such fibers. Stanton and others also find that fibers less than 5000 or less in width are most likely to be carcinogenic. The NIOSH White Paper states: "Fibers and particles with diameters less than 0.5um (5000 A) are more likely to cross membranes and translocate to pleural and peritoneal spaces

⁷ Wylie, 1979, 1993, 1988; Verkouteren and Wylie, 2002

⁸ Wylie et al., 1993

⁹ See Zoltai, 1981, for an excellent discussion.

¹⁰ Polygonal serpentine fibers may have diameters up to 10,000A. Baronnet and Devouard, 2005.

¹¹ Wylie et al., 1993

¹² Warnock, 1989

¹³ Siegrist and Wylie, 1980

¹⁴ 1995

and are more likely to enter the lymphatic and circulatory systems." Thus, not only is the width of asbestos a defining characteristic, it is key to its carcinogenicity.

Cleavage fragments are different. Cleavage fragments, formed by crushing rock, get wider as they get longer and width is therefore dependent on length¹⁵. They do not possess the asbestos characteristic of high tensile strength and their surfaces are different in fundamental ways. While a 40 micrometer asbestos fiber could easily have a width of 0.2 micrometers, such dimensions could never be formed by breakage and no cleavage fragments have such dimensions.

SIZE AND SHAPE HYPOTHESIS

The hypothesis that only dimensions and durability (biopersistence) determine a mineral particles potential to cause mesothelioma, lung cancer, laryngeal cancer, and asbestosis is known as the Stanton Hypothesis. It was based on a large number of experiments in which Stanton and coworkers at the NCI implanted a number of different fibrous materials in rats.¹⁶ They found that the number of long thin fibers highly correlated with the sarcomas that developed after implantation. Other researchers have found similar results¹⁷.

If the Stanton Hypothesis is correct, then any biopersistent particle that has the dimensions of real asbestos should have the same carcinogenic potential as asbestos. In fact, we know that this is often the case for asbestiform fibers. Long thin fibers of erionite, a mineral not regulated as asbestos, are thought to be responsible for a high incidence of mesothelioma among several small villages in Turkey.¹⁸ Furthermore, the long, thin fiber (not specifically regulated as asbestos by the federal government) from Libby, Montana, has been identified as the agent in a number of mesothelioma cases among those occupationally exposed¹⁹.

However, we also know from the experience of miners exposed to other durable long, thin fibers such as fibrous talc²⁰ that all durable long, thin fibers are not the same. Many studies have shown the importance of the surface in the biological activity of mineral fibers.²¹ Understanding the basis of the carcinogenicity of mineral fibers requires further study.

Can the Stanton Hypothesis be used to justify concern for nonasbestos, durable, RFD particles? If the RFD corresponds to a high carcinogenic potential, then many mineral particles would be potential carcinogens. Many common durable minerals break into elongated particles that conform to the RFD even though they are not asbestiform and do not have the dimensions of asbestos fibers. These include pyroxenes, feldspars, zeolites, some sheet silicates, and many other mineral groups. In fact, the Appalachian and Rocky Mountain Chains contain abundant minerals that would form particles meeting the RFD when crushed.

What does the epidemiology tell us? The studies that have examined the epidemiology of workers exposed to dusts that contain nonasbestos amphibole particles that meet the RFD have found no asbestos-related diseases. Amphiboles make up 5 percent of the Earth's crust and, although a large group of minerals of variable chemical composition²², most amphibole fragments exceed 3:1 in length to width ratio if they are longer than 5 micrometers. These studies include miners and millers from a talc mine in New York, gold miners from Lead, South Dakota; vermiculite workers at Enoree, South Carolina; and iron miners from the Minnesota taconite iron district.²³

Asbestos fibers do meet the RFD. They exceed the 3:1 length to width ratio. But because of their narrow widths, they also exceed a 5:1 and a 10:1 and most exceed a 20:1 ratio. Therein lays the problem. While asbestos fibers conform to the RFD, they are not DEFINED by it, and they cannot be separated from other mineral particles by it. While we know that it is very likely that among amphiboles it is the

¹⁵ Siegrist and Wylie, 1980

¹⁶ Stanton et al., 1981

¹⁷ Bertrand, and Pezerat, 1980, Davis et al., 1991, Smith et al., 1979, Pott et al., 1974.

¹⁸ Baris, 1987, Wagner et al., 1985

¹⁹ Amandus et al., 1987; Sullivan, 2007.

²⁰ IARC, in press; Honda et al., 2002; Gamble, 1993; Stille and Tabershaw, 1982

²¹ For example: Chamberlain and Brown, 1978; Feuerbacher et al., 1980; Flowers, 1980; Marchisio and Pernis, 1963; Schlipkoter et al., 1963; Brown et al., 1990; Weitzman and Graceffa, 1984; Weitzman and Weitberg, 1985; Hochella (1993) provides an excellent discussion of the variability of surface chemistry, structure and reactivity of mineral surfaces that may affect biological activity.

²² Leake et al., 1997, 2004

²³ McDonald et al., 1988, McDonald et al., 1978, Brown et al., 1986, Higgins et al., 1983, Cooper et al., 1992, Honda et al., 2002, Gamble, 1993, Steeland and Brown, 1995, Stille and Tabershaw, 1982

size and shape that affects their carcinogenicity, the question is “What size and what shape?”

RELUCTANCE TO CHANGE THE REGULATORY FIBER DEFINITION

Neither OSHA nor MSHA consider cleavage fragments to be asbestos. NIOSH has put the issue up for discussion. It is time for this issue to be resolved.

CONCLUSIONS

I conclude by asking you to support the work that NIOSH has proposed to address unanswered questions about the carcinogenicity of nonasbestos mineral particles. I also ask that the National Institute of Standards and Technology (NIST) be funded to develop new analytical methods for identifying and monitoring asbestos, and that NIEHS fund a comprehensive risk assessment. At the present time, these issues are being decided in the courts, not the appropriate venue for scientific discourse.

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RESPONSE BY ANN G. WYLIE TO AN ADDITIONAL QUESTION FROM SENATOR INHOFE

Question. Are there universally accepted methods by which minerals with asbestiform morphology can be distinguished via testing from chemically similar cleavage fragments?

Response. Asbestos is a commercial term describing a group of highly fibrous silicate minerals composed of very narrow fibrils that easily separate by hand pressures yet possess tensile strength that is higher than the same minerals in a different form. These physical properties are universally understood as the characteristics necessary for the term asbestos to apply. It has been in use for at least 130 years. E. S. Dana, then curator of the Mineral Museum at Yale, and J.D. Dana, Professor of Physics, also at Yale, published a Textbook of Mineralogy in 1877 in which they gave the following definition of asbestos:

“Tremolite, actinolite, and other varieties of amphibole, excepting those containing much alumina, pass into fibrous varieties, the fibres of which are sometimes very long, thin, flexible and easily separable by the fingers and look like flax. These kinds are called asbestos.”

Since this definition was written, it is known that the form of serpentine known as chrysotile can also be asbestos. The properties the Danas describe are the same for both chrysotile-asbestos and amphibole-asbestos and are distinctly different from chemically similar materials that fragment by cleavage.

The identification of a mineral as amphibole or serpentine is readily accomplished by a chemical analysis and an x-ray diffraction pattern, the universally recognized basis for mineral identification.¹ In hand specimens of known amphibole or serpentine, where long thin fibers that look like flax² are visible and hand pressure can be applied to determine if the fibers are flexible and easily separable, the Dana definition of asbestos is universally accepted.

Another definition has been developed over the past 20 years for identification of asbestos in material taken from bulk samples and examined under the optical microscope. To apply this definition, the identification of the mineral as serpentine or amphibole must be known. It has never to my knowledge been criticized and it is widely used; applying the term “universal” however, suggests that it would be accepted by mineralogists worldwide and I cannot say that it has been so widely discussed. It would probably surprise mineralogists not from the United States that a definition is needed at this level since the Danes’ definition is so clear and has served so well for so long. This definition of asbestos at the microscope level for a population of asbestos fibers is as follows:

The following characteristics of a population of asbestos fibers can be observed by light microscopy and enable it to be distinguished from a chemically similar population of cleavage fragments:

¹ There are other recognized methods of identification, but x-ray is most reliable and its results are unequivocal.

² See photographs attached.

(1) Aspect ratios of 20: 1 and greater for particles longer than 5 micrometers are common.

(2) Fibers are composed of very thin fibrils, often less than 0.5 micrometers, which occur in bundles.

(3) Fibers wider than 0.5 micrometers display splayed ends, demonstrating their fibrillar structure.

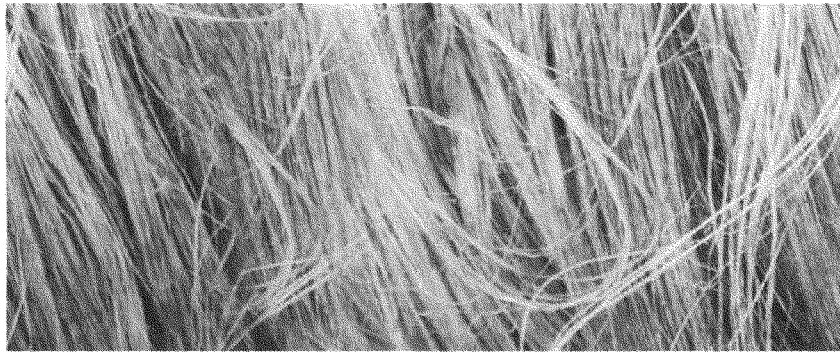
(4) Matted masses of individual fibers may be found in some samples

(5) Long fibers frequently display curvature, a sign of flexibility.

There is no accepted method by which asbestos can be distinguished from cleavage fragments on a particle by particle basis on air monitoring or water filtration filters. The NIOSH fiber definition is not specific for asbestos. However, I believe, and I so testified, that it is possible to develop a method that will enable a sufficiently accurate distinction between asbestos fibers and cleavage fragments found on air and water monitoring filters such that reliable exposure estimates/concentrations of each can be made. The method would be based on the unusual and distinctive dimensional characteristics of asbestos that are already well known and well described in the published literature. The National Institute of Standards and Technology has the experience and knowledge to develop such a method.

Flax (linen fiber). Photo taken from:

www.alibaba.com/catalog/10851478/Flax_Linen_Fibre.html#largepic



Senator BOXER. Thank you very much.
Dr. Weill.

**STATEMENT OF DAVID WEILL, M.D., ASSOCIATE PROFESSOR,
DIVISION OF PULMONARY AND CRITICAL CARE MEDICINE,
STANFORD UNIVERSITY MEDICAL CENTER, STANFORD, CA**

Dr. WEILL. Good morning, Senator Boxer and members of the committee. Thank you for the opportunity to testify.

I will comment today specifically on the differences in toxicity associated with a variety of inhaled fibrous and non-fibrous minerals. These minerals are often grouped under the broad category of asbestos, but there are fundamental differences among these minerals in terms of their potential to cause human disease.

My testimony is from a clinician's point of view, using appropriate support from the scientific background. As was mentioned, I hold several positions at the Stanford University Medical Center, including Director of the Lung and Heart-Lung Transplant Program. I am also a "B Reader," certified by NIOSH as competent to classify chest x-rays for lung conditions such as those caused by exposure to asbestos dust.

I have also had the opportunity to testify before the U.S. Senate Judiciary Committee when it was considering the FAIR Act legisla-

tion in 2005 as well as the Texas State legislature when it was considering legislation addressing the handling of asbestos and silica claims. It is of course a privilege to testify before you here today.

Asbestos exposure, as you have heard today, can lead to both non-malignant and malignant diseases, such as lung cancer and mesothelioma. The asbestos-related diseases, and for that matter, all pneumoconiosis, are dose-dependent, meaning that increased level and total amount of exposure results in increased risk and/or severity of the disease. Conversely, as workplace exposures have been substantially reduced in the last several decades, asbestos-related health effects have also become less prevalent.

While our focus here today is to discuss the differences between asbestiform and non-asbestiform substances, it is important to note that there are important differences, even among various asbestos fiber types, and considerable evidence that different types of asbestos have different potentials to cause disease. While many epidemiologic studies have demonstrated an association between asbestos exposure and mesothelioma, the asbestos-mesothelioma association is particularly strong in occupations that involve heavy amphibole exposure, such as shipyard workers and insulators.

The message of these studies is simple: different asbestos fiber types have different potential to cause disease.

Now, examining the health effects of amphibole minerals more closely. There has been a considerable body of literature about the health effects of cleavage fragments derived from non-fibrous amphibole minerals, specifically whether or not they can cause human disease.

Although I am by no means a mineralogist, I have some understanding about the physical properties of these fibers and cleavage fragments. Most amphibole minerals are non-asbestiform, designated as such because they have different characteristics that make them behave differently. Cleavage fragments result from a physical manipulation of these non-asbestiform particles.

They are sometimes difficult to distinguish from amphibole asbestos fibers using standard counting procedures. Based on the scientific literature in my experience as a clinician, I have three general opinions regarding the health effects of cleavage fragments. No. 1, the different properties of asbestiform amphibole fibers and non-asbestiform cleavage fragments impact human health differently and should not be considered as the same. No. 2, animal data reveal lack of pathogenicity; and No. 3, human epidemiologic studies have established no association between cleavage fragments and human disease.

Others testifying here today will describe in detail the differences in physical properties of asbestos fibers and cleavage fragments. In the interest of time, I will skip any discussion of these physical properties, except to say that the fundamental physical difference between amphibole asbestos fibers and cleavage fragments results in each having very different health effects. That is my first opinion that I want to express.

Now my second opinion, specifically that there are animal studies involving exposure to cleavage fragments, not finding any adverse health effects from these exposures, I have also outlined in my written testimony. I wanted to be able to skip to my third opin-

ion, looking at human epidemiologic studies involving exposure to cleavage fragments, specifically that they have not found any adverse health effects. The occupational settings for these studies include gold, nickel and taconite mines, as well as talc and pottery workers and tunnel diggers. In each of these cohorts, no excess mesothelioma, lung cancer or pneumoconiosis risk could be shown from exposure to cleavage fragments.

Fortunately, with the institution of policies which limit occupational exposure to asbestos, the incidence of asbestos-related lung conditions is decreasing. Further, it is my opinion that not all types of asbestos have the same potential to cause human disease. Even further, cleavage fragments are naturally occurring and rarely meet the regulatory definition of asbestos fiber.

Currently, there is no existing evidence that cleavage fragments are pathogenic for the reasons that I reviewed. The impetus to perform—

Senator BOXER. Doctor, could you just wrap up?

Dr. WEILL. Sure.

Senator BOXER. Thank you.

Dr. WEILL. The impetus to perform epidemiologic studies on substances that may have a human health risk evolve from hypothesis-generating information that suggest there might be a risk. I do not believe such data exists with regard to cleavage fragments.

I feel my opinions today are based on the scientific evidence already available. Thank you for the opportunity to testify, and I hope my perspective is helpful.

[The prepared statement of Dr. Weill follows:]

STATEMENT OF DAVID WEILL, M.D., ASSOCIATE PROFESSOR, DIVISION OF PULMONARY AND CRITICAL CARE MEDICINE, STANFORD UNIVERSITY MEDICAL CENTER, STANFORD, CA

Senator Boxer, Senator Inhofe, and Members of the committee: Thank you for the opportunity to testify before you about the health effects of asbestos. I will comment today specifically on the differences in toxicity associated with a variety of inhaled fibrous and non-fibrous minerals. These minerals are often grouped under the broad category of "asbestos," but there are fundamental differences among these minerals in terms of their potential of each mineral to cause human disease. My testimony is from a clinician's point of view, using appropriate support from the scientific literature.

I'll begin by telling you a bit about my background. I am board certified in Pulmonary and Critical Care Medicine. Currently, I hold several positions at the Stanford University Medical Center, including Associate Professor of Medicine in the Division of Pulmonary and Critical Care Medicine, and I am the Director of the Lung and Heart—Lung Transplant Program.

I am also a "B Reader," which means I have been certified by the National Institute of Occupational Safety and Health ("NIOSH") as competent to classify chest x-rays for lung conditions such as those caused by exposure to asbestos dust. At Stanford, we are referred and treat patients with both common and rare respiratory conditions. Such referrals include patients with both occupational and non-occupational diseases.

I have also had the opportunity to testify before the United States Senate Judiciary Committee when it was considering the FAIR Act in 2005 and the Texas State Legislature regarding legislation addressing the handling of asbestos and silica claims. It is of course a privilege to testify before you here today.

HEALTH EFFECTS OF ASBESTOS

Asbestos exposure can lead to nonmalignant conditions such as asbestosis (a parenchymal fibrotic lung disease) and pleural changes (pleural effusion, pleural thickening, pleural plaques, and rounded atelectasis), as well as malignant conditions such as lung cancer and mesothelioma. The asbestos-related diseases and, for that

matter, all pneumoconiosis, are dose-dependent, meaning that increased level and total amount of exposure results in increased risk and/or severity of the diseases. Conversely, as workplace exposures have been substantially reduced in the last several decades, asbestos-related health effects have become less prevalent.

HEALTH EFFECTS OF DIFFERENT ASBESTOS FIBERS

Asbestos is the commercial designation for 6 fibrous minerals of two broad types: serpentine and amphibole. Chrysotile is the only type of serpentine asbestos, while there are five different amphibole asbestos fibers: crocidolite, amosite, tremolite, actinolite, and anthophyllite. While our focus here today is to discuss the differences between asbestiform and non-asbestiform substances, it is important to note that there are important differences even among various asbestos fiber types and considerable evidence that different types of asbestos have different potentials to cause disease. While many epidemiologic studies have demonstrated an association between asbestos exposure and mesothelioma, the asbestos-mesothelioma association is particularly strong in occupations that involved heavy amphibole asbestos exposure, such as shipyard workers and insulators.

The message of these studies is simple: different asbestos fiber types have different potential to cause disease.

HEALTH EFFECTS OF CLEAVAGE FRAGMENTS

Now, let's examine the health effects of amphibole minerals more closely. There has been a considerable body of literature about the health effects of cleavage fragments derived from non-fibrous amphibole minerals, specifically whether they can cause human disease. Although I am by no means a mineralogist, I have some understanding about the physical and chemical properties of asbestos fibers and cleavage fragments, particularly as they are important to the development of human lung disease.

Most amphibole minerals are "non-asbestiform", designated as such because they have different characteristics that make them behave differently. Cleavage fragments result through the physical manipulation of these non-asbestiform particles and are sometimes difficult to distinguish from amphibole asbestos fibers using standard counting procedures.

Based on the scientific literature and my experience as a clinician, I have three general opinions regarding the health effects of cleavage fragments:

- (1) The different properties of asbestiform amphibole fibers and non-asbestiform cleavage fragments impact human health differently and should not be considered as the same;
- (2) Animal data reveal a lack of pathogenicity;
- (3) Human epidemiological studies have established no association between cleavage fragments and human disease

PHYSICAL PROPERTIES OF AMPHIBOLE ASBESTOS FIBERS AND CLEAVAGE FRAGMENTS

First, a bit about the different properties of asbestos fibers and cleavage fragments. Although the non-asbestiform and asbestos amphiboles are chemically similar, they differ with regards to morphology. Asbestiform amphiboles are made up of fiber bundles that run parallel to each other, which when they split, form single fibrils. Each individual fibril is long, thin, and very flexible. Non-asbestiform amphiboles are not unidirectional fibers but run in two or more different planes, forming a prism. These non-asbestiform structures do not break down into fibers or fibrils but instead into cleavage fragments that are thick and short and therefore not likely to be inhaled into the more distant (or deep) parts of the lung.

If one then compares more closely asbestiform and non-asbestiform amphiboles, they differ with respect to three important characteristics: surface properties, tensile strength, and dissolution.

1. *Surface properties.* The outside surface of amphibole asbestiform fibers is smooth, free of defects, and very strong, largely because there are no crevices or cracks in the fiber surface that can be subject to degradation strategies present after inhalation into the lung. This is not the case in non-asbestiform structures that have mechanical planes that can be exploited and lead to degradation.

2. *Tensile strength.* Amphibole asbestos fibers have inherent flexibility, giving them great tensile strength. Cleavage fragments, however, are inflexible and brittle, making them vulnerable to physical stress.

3. *Dissolution properties.* The human body's natural defenses, particularly macrophages, generate an acidic environment to break down inhaled particles in the lungs. Amphibole asbestos fibers are resistant to acidic dissolution and are said to be biopersistent, meaning they remain in the lungs indefinitely. Cleavage fragments

have surface defects or cracks that make these fragments amenable to acidic dissolution, which enables the body's natural defenses to expel them.

These fundamental physical differences between amphibole asbestos fibers and cleavage fragments result in each category of minerals having different health effects. Cleavage fragments are generally too wide to penetrate into the deep parts of the lung, particularly when longer than 5 microns. If shorter than 5 microns, as is commonly the case, there is a body of literature that suggests that, even if they shared the same properties as those of asbestos fibers, that these smaller particles have no pathologic effect, either in terms of fibrosis or mesothelioma development. In fact, the epidemiology and basic science literature (beginning in 1968) demonstrates that fiber length correlates strongly with development of asbestos-related diseases. This proposition is described as the Stanton hypothesis and assumes that fibers greater than about 8 microns in length and less than a quarter of a micron in diameter are the most potent in producing mesothelioma.

Highlighting this point, the EPA in 2003 reviewed the available literature to devise a protocol to assess asbestos-related risk. The expert panel agreed with the development of a protocol that considered, for purposes of evaluating asbestos-related risk, that fibers less than 0.5 microns in diameter and greater than 5 microns in length were more important in disease development. Fibers with greater diameters were believed to be unlikely to be inhaled to the more distal parts of the lung.

ANIMAL STUDIES INVOLVING EXPOSURE TO CLEAVAGE FRAGMENTS

Let's move on to my second opinion, specifically that animals studies involving exposure to cleavage fragments have not found any adverse health effects from such exposures. It should be noted that there are limitations of the findings of any animal studies of this nature. First, animal studies generally use direct intrapleural or intraperitoneal injection of the substance being studied, bypassing the lung's natural defense mechanisms. And secondly, the amount of a substance administered to the animals (i.e. the dose) is usually massive and well beyond what could be observed in any occupational setting. However, notwithstanding these limitations, there are several animals studies that have been conducted that show no carcinogenic potential for cleavage fragments. This is very different from similarly conducted studies when true amphibole asbestos fibers were instead injected.

HUMAN STUDIES INVOLVING EXPOSURE TO CLEAVAGE FRAGMENTS

Finally, my third opinion is that the body of human epidemiological studies involving exposure to cleavage fragments has not found adverse health effects from exposure to cleavage fragments. The occupational settings for these epidemiological studies included gold, nickel, and taconite miners, as well as talc and pottery workers and tunnel diggers. In each of these cohorts, no excess mesothelioma, lung cancer, or pneumoconiosis risk could be shown from exposure to cleavage fragments.

The largest study of workers exposed to cleavage fragments has been the Homestake gold mining cohort. In this study, there was no excess lung cancer risk identified. In fact, as exposure levels increased, the lung cancer risk tended to decrease, indicating no association of exposure with lung cancer development. Importantly, there were no mesothelioma deaths in this group. A study was also conducted of the Minnesota taconite miners who were exposed to grunerite cleavage fragments and this cohort showed no evidence of an excess of asbestos-attributable diseases. Other studies of cohorts exposed to cleavage fragments have reached similar conclusions. Therefore, the health risks demonstrated to be associated with amphibole asbestos exposure should not be assumed to apply to cleavage fragments.

Fortunately, with the institution of policies which limit occupational exposure to asbestos, the incidence of asbestos related lung conditions is decreasing. Further, it is my opinion that not all types of asbestos have the same potential to cause human disease. Even further, cleavage fragments are naturally occurring and rarely meet the regulatory definition of an asbestos fiber. Therefore they are designated as "non-asbestiform" and have fundamentally different properties than amphibole asbestos. Currently, there is no existing evidence that cleavage fragments of nonasbestiform fibers are pathogenic for the reasons that I reviewed in my testimony, and there is no animal or human data that implicates these fragments as a cause of disease.

The impetus to perform epidemiologic studies on substances that may have a human health risk generally results from hypothesis-generating information to suggest that there might be a health risk. I do not believe such data exists. Further, with the asbestos exposure levels so low currently and the inability to study in isolation the health effects of cleavage fragments, I do not feel that human studies could be conducted which would result in meaningful conclusions. The medical literature is already informative on non-asbestiform fragments, and while it is always impor-

tant to gain new scientific knowledge, I feel my opinions expressed today are based on the sound scientific evidence already available.

I hope that my perspective is helpful to the committee's efforts. Thank you.

RESPONSES FROM DAVID WEILL, M.D., TO ADDITIONAL QUESTIONS FROM
SENATOR INHOFE

Question 1. Can you please clarify your response during the hearing regarding questions posed to you associated with scientific methodology and acceptable risk?

Response. Science, through epidemiologic study, provides society with risk assessments of various elements of our society. These elements are broad and include non-occupational activities, such as driving a car, drinking the water, and flying in an airplane, just to name a few. Epidemiology has also given us information about the risks present in a variety of occupational settings and informs employers, employees, and society in general about the risks that might be present in a particular work environment. Science can quantitatively assess these occupational and non-occupational risks, but it cannot determine what is an acceptable risk. Instead, the determination of acceptable risk is a societal function.

Question 2. Do you believe it is the proper role for scientists and data analysts to make policy, regulatory, and legislative decisions regarding health protections or is it the role of these technical professionals to fairly and without bias evaluate scientific data to inform the public policy debate?

Response. I clearly think it is our job as scientists to provide the scientific data and analysis to policy makers, who then have the responsibility to set policy that reflects societal values and concerns. I do not think it is my place as a physician to make determinations about what risks are acceptable in non-occupational or occupational settings. This should be a function of our policy makers.

Senator BOXER. Thank you, Doctor.

Dr. Lemen.

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U.S. PUBLIC HEALTH SERVICE (RETIRED), REAR ADMIRAL,
U.S. PUBLIC HEALTH SERVICE (RETIRED)**

Mr. LEMEN. I would like to thank you, Chairman Boxer, Ranking Member Inhofe and Senator Lautenberg and the rest of the committee for inviting me here today. My name is Dr. Richard Lemen. I am a former Assistant Surgeon General of the United States, and was former Acting and Deputy Director of the National Institute for Occupational Safety and Health.

As we address asbestos during this hearing over the next 2 to 3 hours, approximately three to four people will die of an asbestos-related death. These diseases could have been prevented. Unfortunately, these diseases represent an under-estimate because there are no nationwide surveillance systems that capture adequately the true nature of asbestos-related diseases.

For example, one of our premier surveillance systems, the Surveillance Epidemiology and End Results data base of the National Cancer Institute has been found to under-report mesothelioma in some areas of the United States by as much as 80 percent. We need to fix this and perfect better systems to capture all asbestos-related diseases, if we are ever to have data to measure the true impact of asbestos and to determine if our public health efforts to prevent asbestos-related diseases are effective.

As we will see in countries that have banned or placed strict regulations on the import and use of asbestos, the trend of asbestos-related diseases is beginning to slow down. However, this is not

true in the United States, according to the National Institute for Occupational Safety and Health, where asbestosis is the only one of the induced lung diseases that continues to increase. This is also true for mesothelioma.

While this country is still experiencing asbestos-induced and disease epidemic that continues to get worse and shifting from occupational to non-occupational victims, proponents of asbestos usage are trying to influence the regulatory agencies with efforts to exclude some forms of asbestos, as well as rewrite the definition of asbestos to exclude exposures to non-asbestos materials that are contaminated with fibrous asbestos. These issues include the relaxation of regulatory standards for the main commercial asbestos fiber type chrysotile.

In doing this, two issues will be accomplished. First, the ability to continue to use chrysotile asbestos in this country and to promote new markets in developing countries not having regulations or adequate knowledge of the hazards of this form of asbestos.

Second, by redefining asbestos and eliminating types of fibrous particles such as cleavage fragments that are contaminants of talc mines and other types of mines such as vermiculite mines, allow these operations to continue exposing their workers and spreading their contamination and deadly products to unsuspecting consumers. Such shennannery must be exposed.

Chrysotile asbestos is dangerous and no exposure threshold has ever been established for its safe use. It causes all asbestiform-related diseases. Regulation of asbestos has been historically dependent upon the definition of asbestos, and as you heard from NIOSH, even at the current standard of .1 fiber per cc, 3.4 per 1,000 workers will die over a working lifetime.

I would like to provide some data which may shed some light on the arguments for a better fiber definition which comes to materials contaminated to fibrous asbestos. For many years, NIOSH has been looking at this issue. NIOSH's Dement and co-workers found from one mine and mill reported by a company to be producing non-asbestiform talc air samples of 5 fibers per cc as time-weighted averages in 6 job categories containing 48 percent mineral talc, tremolite and phosolyte, serpentine, lizardite, antigorite. Thus, the TWA for asbestos was exceeded by both the OSHA and the Federal Mine Safety and Health Administration.

I would like to end this testimony by saying that in some of the testimony that has come before, we have heard that the mines that have some of these fibers, such as the gold mine, have not had related diseases. I would like to correct that, because there is a three-fold excess in the study that I was conducting with my NIOSH colleagues in 1976 for respiratory cancer, and a twofold excess for respiratory disease.

In the study that was done by Dr. McDonald, when you look at the—

Senator BOXER. We need you to wrap up now.

Mr. LEMEN. When you look at the latency period, which is an important, critical factor, and those highest latency periods disease did occur.

Thank you, and I will have the rest of my comments submitted to the committee.

[The prepared statement of Dr. Lemen follows:]

STATEMENT OF RICHARD A. LEMEN, PH.D., M.S.P.H., FORMER DIRECTOR, DIVISION OF STANDARDS DEVELOPMENT AND TECHNOLOGY TRANSFER, ASSISTANT SURGEON GENERAL, U.S. PUBLIC HEALTH SERVICE (RETIRED), REAR ADMIRAL, U.S. PUBLIC HEALTH SERVICE (RETIRED)

I would like to thank Chairman Boxer, Ranking Member Inhofe and the entire EPW Committee for the honor and opportunity to testify today.

My name is Dr. Richard A. Lemen. I am retired from the United States Public Health Service where I was an Assistant Surgeon General of the United States. At the time of my retirement I was also Deputy Director and had been Acting Director of the National Institute for Occupational Safety and Health (NIOSH). I have spent my entire career, since 1970, studying the epidemiology of asbestos-related diseases and have conducted numerous epidemiology studies, written many scientific papers, advised the World Health Organization, various other National governments, and have testified before the United States Congress on several occasions concerning the health risks from exposure to asbestos. I am an adjunct professor of environmental and occupational medicine at Emory University and a consultant in occupational health and epidemiology. I also testify in asbestos-related litigation on behalf of plaintiffs. My CV, which I have supplied the committee, will give you further information concerning my studies on asbestos.

Often asbestos is referred to as the “magic Mineral” having at least 3000 or more uses, such as being woven into cloth, with vegetable fibers; for wrapping the corpses, referred to by Pliny as the funeral dress of kings prior to cremation in order to help collect the ashes; in making clay pots some 4000 years ago; and was even mentioned by Marco Polo, during his travels to the far east, where he found it called “salamander” skin which was mined from the mountains, extracted then crushed, by subjects of the Great Khan, into a fibrous like wool that was then spun and made into cloth of which some were used for table cloths, that when soiled, were thrown into the fire and came out “white as snow” for use again; one was sent to the Pope, in Rome, “in which cloth he keeps the Sudarium of our Lord.” Benjamin Franklin even bought a purse from the “northern part of America” made from woven asbestos.¹

Our modern knowledge of asbestos usage and asbestos-related disease began in the early 1900s, with reports of lung diseases among asbestos workers in the United Kingdom as well as the United States. By 1930, the disease asbestosis was well established as a lung disease contracted from exposures to asbestos. Unfortunately, by the mid-1930s it was suspected that, in addition to asbestosis, cancer may also result from exposure to asbestos. Today we know that various cancers, including lung cancer, gastrointestinal cancers, and mesothelioma are all causally associated from exposure to asbestos. We know that all forms of commercially used asbestos, including chrysotile, as well as the amphiboles cause all of the asbestos-related diseases including asbestosis, lung cancer, mesothelioma and gastrointestinal cancers.²

Asbestosis is a progressive disease which can eventually result in death after much disability and suffering, even after occupational exposures have ceased. Asbestosis does not respond to medical treatment, only palliative care can be given.³

Asbestos-induced cancers are not confined to just the workers exposed at work, but asbestos exposures can be brought home to family members, as a result of contamination of their work clothes, prompting asbestos-induced disease in them as well. Asbestos-related diseases can also occur to residents living near asbestos sources.⁴

In the United States it is estimated that between 189,000 and 231,000 deaths have occurred since 1980 due to workplace exposure to asbestos. Another 270,000 to 330,000 deaths are expected to occur over the next 30 years and for those workers

¹Lemen, RA, 2005. Epidemiology of Asbestos-Related Diseases and the Knowledge that Led to What is Known Today. In: ASBESTOS Risk Assessment, Epidemiology, and Health Effects, Eds. RF Dodson, SP Hammar. CRC Taylor & Francis, 201–308.

²Lemen, RA, 2005. Epidemiology of Asbestos-Related Diseases and the Knowledge that Led to What is Known Today. In: ASBESTOS Risk Assessment, Epidemiology, and Health Effects, Eds. RF Dodson, SP Hammar. CRC Taylor & Francis, 201–308.

³ATSDR, 2001. Agency for Toxic Substances and Disease Registry Questions and Answers Exposure to Asbestos. Department of Health and Human Services, Atlanta, GA, July 26.

⁴NIOSH, 1995. Report to Congress on Workers' Home Contamination Study Conducted Under The Workers' Family Protection Act (29 U.S.C. 671a). U.S. Department of Health and Human Services, Public Health Service, Centers For Disease Control And Prevention, National Institute for Occupational Safety and Health (NIOSH), Cincinnati, OH 45226, September. See sections on Asbestos p. 6–11; 45–46; 55; 62–63; 86–87; tables 2–6 (pp. 145–159).

exposed, over a working lifetime, to the current Occupational Safety and Health administration (OSHA) standard of 0.1 fibers/cc—3.4/1000 workers are estimated to die as a result of asbestos-related diseases.⁵ A more recent study suggested the use of linear extrapolation, as used by OSHA, from high exposure levels may underestimate the risks at low doses (Gustavsson et al., 2002).⁶ Unless asbestos use in the United States is not banned there is no end of its ability to expose workers and consumers to its dangers.

Products containing asbestos can still be found in things found in the home such as lamp sockets, floor tiles, cat box fill, braking mechanism in washing machines and cars, furnaces, and other products. Because these products are not only manufactured by workers, but are also used, maintained, and repaired by workers—they (workers) suffer additional exposure from consumer products as do the consumers using these products.

The most recent Criteria Document from the World Health Organization's (WHO) International Programme for Chemical Safety (IPCS) states in 1998 that no threshold has been identified for carcinogenic risks to chrysotile asbestos.⁷ Chrysotile is the main commercially used asbestos in the World. This 1998 WHO statement is consistent with the WHO's earlier conclusion in 1989 "[T]he human evidence has not demonstrated that there is a threshold exposure level for lung cancer or mesothelioma, below which exposure to asbestos dust would not be free of hazard to health".⁸ The WHO recognizes what NIOSH concluded 31 years ago, in 1976, that ". . . (only a ban can assure protection against carcinogenic effects of asbestos)".⁹ I cannot tell any of you, on this committee, why some will develop asbestosis or other asbestos-related cancers and why others won't. But what I can tell you is that asbestos-induced diseases are preventable. Each and every one!

The first criteria document from the newly formed NIOSH of 1970, was on asbestos, after NIOSH's first Director Dr. Marcus Key had sent a letter to OSHA stating the inadequacy of OSHA's new start-up standard for asbestos, based on the then ACGIH TLV®. NIOSH was the first federal agency to call for a ban on asbestos in its 1976 Revised Criteria Document. NIOSH has maintained this position to the present, while suggesting in the interim that the only reliable and practical analytical method, in 1976, was 0.1 fiber/cc using the NIOSH Phase Contrast Method (PCM) 7400 asbestos analytical method. Unfortunately chrysotile cannot be seen in the light microscope when it occurs in the fibril form and thus most chrysotile is not counted in an air sample using a NIOSH 7400 count scheme-diameter resolution of approximately 0.25 microns where as most individual fibers of crocidolite and chrysotile are 0.02–0.05 microns in diameter. OSHA describes the advantages and disadvantages of the Phase Contrast Microscope (PCM) as can be seen in the footnote.¹⁰

⁵ OSHA, 1986. OSHA, 1986. Final Rule: Asbestos. 51 FR 22612. U.S. Department of Labor. Occupational Safety and Health Administration, Washington, D.C., June 20.

⁶ Gustavsson P, Nyberg F, Pershagen G, Schéele P, Jakobsson R, Plato N, 2002. Low-dose exposure to asbestos and lung cancer: Dose-response relations and interaction with smoking in a population-based case-referent study in Stockholm, Sweden. *Am J Epi*, Vol. 156 (11); 1016.

⁷ IPCS, 1998. Environmental Health Criteria 203: Chrysotile Asbestos, International Program on Chemical Safety, World Health Organization.

⁸ WHO, 1989. Occupational Exposure Limit for Asbestos. WHO/OCH/89.1, Office of Occupational Health, World Health Organization, Geneva.

⁹ NIOSH, 1976. Revised Recommended Asbestos Standard. DHEW (NIOSH) Publication No. 77-169. U.S. Department of Health, Education, and Welfare. Public Health Service. Centers for Disease Control. National Institute for Occupational Safety and Health. December.

¹⁰ Rules and regulations—Dept Labor—OSHA 29 CFR Parts 1910, 1915, 1926—Occupational Exposure to Asbestos—Final rule—Aug. 10, 1994 59FR4096

"1.3 Advantages and Disadvantages

There are four main advantages of PCM over other methods:

(1) The technique is specific for fibers. Phase contrast is a fiber counting technique which excludes non-fibrous particles from the analysis.

(2) The technique is inexpensive and does not require specialized knowledge to carry out the analysis for total fiber counts.

(3) The analysis is quick and can be performed on-site for rapid determination of air concentrations of asbestos fibers.

(4) The technique has continuity with historical epidemiological studies so that estimates of expected disease can be inferred from long-term determination of asbestos exposures.

41066 The main disadvantage of PCM is that it does not positively identify asbestos fibers. Other fibers which are not asbestos may be included in the count unless differential counting is preformed. This requires a great deal of experience to adequately differentiate asbestos from non-asbestos fibers. Positive identification of asbestos must be performed by polarized light or electron microscopy techniques. A further disadvantage of PCM is that the smallest visible fi-

Continued

Any definition of asbestos should include all respirable asbestiform fibrous minerals, including fibrous cleavage fragments which are respirable.¹¹ This should only be changed if there exist irrefutable data, both human and animal, showing the safety of any such fibrous mineral being excluded. Valid methodologies now exist to sample for all size fibers, including those less than 5 μm in length, not currently addressed in regulatory standards. These smaller fibers should be included in any asbestos definition. Both animal and human data support such an inclusion as can be seen by the attached Appendix 1.¹²

Federal and State governments should work together to address, refine, and/or develop surveillance of fiber-related diseases, including those from asbestos. For example it is well known that the National Cancer Institutes Surveillance Epidemiology and End Results (SEER) data base underreports mesothelioma.¹³ NIOSH should be funded to continue its Respiratory Disease Surveillance System and should assure that other NIOSH surveillance systems become more comprehensive and inclusive. None of the systems should rely solely on Proportionate Mortality/Morbidity Analysis for determining mortality or morbidity data, as this type analysis underreports low incidence diseases, albeit important diseases i.e. mesothelioma.

Research should determine how much of background mesothelioma and other asbestos-related diseases are related to the increased consumption of asbestos in any reference populations used for comparison and thus adjust expected rates accordingly in order to determine the true risk of asbestos-related diseases.

Epidemiology literature on all fibrous materials, not just those related to the currently regulated asbestiform fiber types should be reviewed and new research conducted when necessary. Such research should address all respirable fiber types and all size parameters of a respirable nature, including short respirable fibers less than 5 microns in length.

Since biopersistence has been used as a surrogate for exposure and fiber type of exposure through identifying their persistence in the lung as a critical factor in causation, toxicological studies should evaluate whether the external airborne concentrations of fibers are actually representative of the fiber concentrations and morphologies once the fibers have been inhaled into the lung. Data suggest that the correlation of breathing zone samples of chrysotile may not represent the actual fiber concentration of chrysotile fibers once in the lung as they break apart from fiber bundles and multiply within the lung, while the amphiboles do not.¹⁴ This is important not only as it means a higher dose of chrysotile within the lung but a higher number of fibers that can translocate from the lung to other parts of the body, such as the pleura. Because dose plays a significant role in the toxicity 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. Translocation of chrysotile asbestos from the lung indicates a specific role for chrysotile in the etiology of mesothelioma since the chrysotile fibers reach the areas where the tumor develops. Mesotheliomas develop in the pleura, peritoneum and other serosal 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 pleura¹⁵. 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 con-

bers are about 0.2 μm in diameter while the finest asbestos fibers may be as small as 0.2 μm in diameter. For some exposures, substantially more fibers may be present than are actually counted.”

¹¹Dement J M, Zumwalde RD, Gambel JF, Fellner W, DeMeo MJ, Brown DP, Wagoner JK, 1980. Occupational exposure to talc containing asbestos-Morbidity, Mortality, and environmental studies of miners and millers. NIOSH Technical Report-DHEW (NIOSH) Publication No. 80-115, Feb.

¹²See Appendix 1—Short Fibers, Richard A. Lemen, Ph.D.

¹³See Appendix 2—Mesothelioma Surveillance, Richard A. Lemen, Ph.D.

¹⁴Bellman B, Muhle H, Pott F, Konig H, Kloppeel H, Spurny K, 1987. Persistence of man-made fibers (MMF) and asbestos in rat lungs. *Annals of Occup Hyg*, 31: 693-709.

¹⁵Suzuki, Y. & Kohyama, N., 1991. Translocation of Inhaled Asbestos Fibers from the Lung to Other Tissues. *Am J Ind Med*, Vol. 19, p. 701-704; Kohyama, N. & Suzuki, Y., 1991. Analysis of asbestos fibers in lung parenchyma, pleural plaques, and mesothelioma tissues of North American insulation workers. *Ann N Y Acad Sci*, Vol. 643, p. 27-52; Suzuki, Y., Yuen, S., Ashley, R. & Calderaro, A., 1998. Asbestos fibers and human malignant mesothelioma. *Advances in the Prevention of Occupational Respiratory Diseases*, Eds. Chiyotani, K., Hosoda, Y., & Aizawa, Y., Elsevier Science B.V., p.709 and Sebastien, P., Janson, X., Gaudichet, A., Hirsch, A. & Bignon, J., 1980. Asbestos retention in human respiratory tissues: comparative measurements in lung parenchyma and in parietal pleura. *IARC Sci Pub*, Vol. 30, p. 237-246; Dodson RF, Graef R, Shepherd S, O'Sullivan M, Levin J, 2005. Asbestos burden in cases of mesothelioma from individuals from various regions of the United States. *Ultrastruct Pathol*. Sep-Oct;29(5):415-33.

tributes to the cause of mesothelioma. This is also shown by many mechanistic and molecular studies that indicate how chrysotile may cause mesothelioma. Fiber penetration can rearrange the cytoskeletal apparatus of the cell and this could indicate an interaction between the chrysotile fibers and the normal mitotic process, since giant multinucleated cells are formed. These studies indicate that chrysotile penetrates the cell, enters the nucleus and induces abnormal chromosome formations in dividing cells.¹⁶ Some of these abnormalities include the deletion of the P53 gene that controls cell growth.¹⁷

Additional research should include evaluation of the synergistic effects between amphibole 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.¹⁸ It has been suggested by some that the fibrous tremolite contamination of chrysotile, usually less than 1 percent, is the cause of mesothelioma among predominately chrysotile exposed persons.¹⁹ New evaluation of the South Charleston chrysotile exposed population of textile workers has confirmed a dose-response relationship between asbestosis and lung cancer.²⁰ This is important as entities suggesting that chrysotile is the “safe asbestos” are basing their conclusions on only one outcome, that being mesothelioma. While it is generally recognized that chrysotile on a dose-by-dose basis is less potent than the amphiboles in producing mesothelioma; this does not appear the case in its ability for causing other asbestos-induced disease. Therefore, future research should continue to look at all asbestos-induced diseases when determining recommended regulatory actions for the prevention of asbestos-related diseases.

The current OSHA regulations govern exposure to minerals defined in the regulations as asbestos; however, formations that contain tremolite asbestos also have tremolite cleavage fragments. Thus, just because the cleavage fragments are not covered under the current OSHA regulations, as regulated fibers, does not mean that they are biologically inactive. The emphasis of the fiber pathogenicity being related to the fact that any asbestos structure is a fiber is only one explanation of how it causes disease. The fact is that the non-asbestiform cleavage fragment is an analog of the fibrous asbestos structure and is chemically made of the same composition. The complexity of asbestos induced lung disease/injury includes a wide array of issues other than just physical features (Kamp and Wiseman, 1999).²¹

Next I will provide some data which may shed more light on the arguments for including a broader fiber definition when it comes to materials contaminated with asbestos. As former Deputy and Acting Director of NIOSH I know the agency has been dealing with the issue of talc contaminated with fibrous asbestos for many years. Researchers found among miners and millers from two counties in Northern New York eight talc miners identified as having mesothelioma and now Hull, Abraham and Case (2002) have added five new cases.²² Rohl and Langer (1974) have stated “Talc because of its composition, conditions of formation and geological occurrence, is frequently contaminated with asbestos fibers.”²³ “The data, however, support earlier studies that indicate that talc miners and millers experience excess pa-

¹⁶ Malomi, W., Loai, F., Falchi, M., and Donnelly, G., 1990. On the mechanism of cell internalization of chrysotile fibers: An immunocytochemical and ultrastructural study. *Environmental Research*, Vol. 52, No. 2, pages 164–177.

¹⁷ Levesse, Renier, Fleury-Feith, Levy, Moritz, Vivo, Pilatte, Jaurand, 1997. Analysis of Cell Cycle Disruptions in Cultures of Rat Pleural Mesothelial Cells Exposed to Asbestos Fibers. *Am J Respir Cell Mol Biol*, 17: 660–671.

¹⁸ Nicholson WJ, Landrigan PJ, 1994. The carcinogenicity of chrysotile asbestos, In : *The Identification and Control of Environmental and Occupational Diseases : Asbestos and Cancer*. Eds. M Mehlman, A Upton: Princeton Scientific Publishing Co., Inc. Vol XXII; Acheson ED, Gardner MJ, 1979. Mesothelioma and exposure to mixtures of chrysotile and amphibole asbestos.

¹⁹ McDonald J.C., McDonald AD, Chrysotile, Tremolite and Mesothelioma. Letter published in *Science*, 10 Feb 1995, Vol. 267:775

²⁰ Hein MJ, Stayner L, Lehman E, Dement JM, 2007. Follow-up study of chrysotile textile workers : cohort mortality and exposure-response. *Occup Environ Med* (published online 20 Apr. 2007), 031005.

²¹ Kamp DW, Weitzman SA, 1999. The molecular basis of asbestos induced lung injury. *Thorax*.54:638–652

²² Hull MJ, Abraham JL, Case BW, 2002. Mesothelioma among workers in asbestiform fiber-bearing talc mines in New York State *Ann Occ Hyg*, 46, (Supplement 1):132–135

²³ Rohl AN, Langer AM, 1974. Identification and quantitation of asbestos in talc. *Env Health Perspectives*, Dec., 9; 95–109.

renchymal fibrosis and pleural changes. The data also suggest that individuals in the paper industry and construction trades may be at risk."²⁴

Dement et al., in 1980 found from one mine and mill, reported by the company to be producing non-asbestiform talc, air samples of 5 fibers/cc as time weighted averages (TWA) in six job categories containing 48 percent mineral talc, 37–59 percent tremolite, 4.5–15 percent anthophyllite, and 10–15 percent serpentine, lizardite, antigorite. Thus the TWA exposures to asbestiform amphiboles (anthophyllite and tremolite) were found to be in excess of the present U.S. Occupational Safety and Health (OSHA) and Mine Safety and Health Administration (MSHA) occupational exposure standards. They also found that in many mine and mill operations more than 90 percent of the total airborne fibers were less than 5µm in length. They found asbestiform tremolite, anthophyllite and in a couple of samples chrysotile and found they were fibers when using Analytical Transmission Electronic Microscope (ATEM) as well as PCM and not cleavage fragments.²⁵

I recommend that that all fibrous asbestiform minerals and that all other minerals or materials contaminated with fibrous asbestos be treated as hazardous and regulated as asbestos.

Finally when new epidemiology studies are conducted strict criteria must be followed to assure the best quality studies possible. These criteria should include, but not limited to areas such as:

(1) Determine actual exposure to the fibrous material and not allow dilution of any effect finding by including those in the cohort not exposed to the fibrous material;

(2) Allow sufficient size of the study population to assure sufficient power to detect adverse effects if they exist;

(3) Conduct sufficient follow-up to assure that at least 95 percent of the cohort is traced and vital status known and evaluated;

(4) Allow sufficient latency to determine if adverse effects do develop, this is important since known traditional latency periods may be extended due to lower level cumulative exposures experienced today;

(5) Identify and account for any possible confounders that may affect the outcome of the study;

(6) If case-control analyses are conducted make sure that all matched controls are selected so that confounding factors will not skew the outcome, including adequate occupational histories to rule out other causative agents or past occupational exposures; and

(7) Dose-reconstruction should not be allowed unless adequate data points exist, from actual exposure samples taken at multiple points during the entire exposure period, as extrapolation from more recent exposures will often reflect control technologies not in place earlier in the persons exposure history, thus resulting in an under estimate of the individuals true exposure. Dose-reconstruction should never be applied from one work situation to another without adequate working conditions being explained and/or described by the affected worker or from actual witnesses to the workers exposure conditions, including an explanation of both environmental or personal control-technologies applied in the specific workplace(s).

I would hope all who have testified here today have disclosed their own affiliations and potential conflicts of interest. Since my retirement I have testified numerous times for plaintiff's attorneys in asbestos litigation, I am also Co-Science Director to the Asbestos Disease Awareness Organization (ADAO) which has covered some of my expenses to attend this hearing today, and no expenses for my testimony or preparation for it have been covered by plaintiff attorneys or any other entity other than myself.

Last, I would encourage members of this committee to support the Ban Asbestos Act introduced by Sen. Murray to include a ban on all commercial uses and importation of asbestos to or within the United States. I look forward to be of assistance should further questions arise.

²⁴Fitzgerald EF, Stark AD, Vianna N, Hwang S-A, 1991. Exposure to asbestiform minerals and radiographic chest abnormalities in a talc mining region of upstate New York. Archives of Environmental Health. May/June, 46 (3); 151–154.

²⁵Dement J M, Zumwalde RD, Gambel JF, Fellner W, DeMeo MJ, Brown DP, Wagoner JK, 1980. Occupational exposure to talc containing asbestos-Morbidity, Mortality, and environmental studies of miners and millers. NIOSH Technical Report-DHEW (NIOSH) Publication No. 80-115, Feb.

SHORT ASBESTOS FIBERS, RICHARD A. LEMEN, PH.D.

EPA reported that millions of asbestos fibers can be released during brake and clutch servicing and that such asbestos can linger around the garage long after brake jobs are done and can be breathed in by everyone inside the garage which can present a hazard for months or years. Grinding of used brake block linings has been shown to release up to 7 million fibers per cubic meter and beveling new linings up to 72 million fibers and even light grinding of the new linings up to 4.8 fibers.²⁶ It has also been reported that during this decomposition process the majority of fibers that remain are of small diameter as well as below 5 micron in length²⁷ and thus are less harmful.²⁸

Any assumption that short fibers, less than 5 micron in length, are not hazardous cannot be justified based on the available science. Because the analytical method of choice, for regulatory purposes, has been the phase contrast method [PCM] which counts only fibers greater than 5 μm in length, epidemiology studies therefore have been forced to compare doses of exposure within their cohorts only to fibers greater than 5 μm in length. It must be noted that the PCM analytical method was chosen based on its ability to count fibers only and not on a health effect basis.²⁹ While PCM has been the international method for analysis, it should also be noted that it is not able to detect thin diameter fibers [$<0.2\mu\text{m}$ in diameter]. The evidence suggests that PCM may underestimate exposures and the health risks as found in the analysis of brake residue,³⁰ or other such exposures where short fibers may be found and because of this, it has been suggested that transmission electron microscopy [TEM] should be an adjunct to PCM.

Stanton and Wrench (1972)³¹ and Stanton et al. (1981)³² found that the longer, thinner fibers were more carcinogenic, but they could not identify a precise fiber length that did not demonstrate biological activity. It must be kept in mind that Dr. Stanton has never said long fibers are bad and short fibers are good. In fact, he appreciated that a large number of short fibers, individually of low tumorigenic

²⁶USEPA, 1986. Guidance for Preventing Asbestos Disease Among Auto Mechanics. United States Environmental Protection Agency. EPA-560-OPTS-86-002, June.

²⁷Rohl, AN, Langer, AM, Wolff, MS & Weisman, I, 1976. Asbestos exposure during brake lining maintenance and repair. Environ Research, Vol. 12, p. 110; Sheehy, J. W., Cooper, T. C., O'Brien, D. M., McGlothlin, J. D., & Froehlich, P. A., 1989. Control of Asbestos Exposure During Brake Drum Service. National Institute for Occupational Safety and Health, Public Health Service, Centers for Disease Control, U. S. Department of Health and Human Services, August; & Yeung, P, Patience, K, Apthorpe, L, & Willcocks, D, 1999. An Australian study to evaluate worker exposure to chrysotile in the automotive service industry. Appl Occup Environ Hyg, Vol. 14, No. 7, July, p. 448.

²⁸Hatch, D, 1970. Possible alternatives to asbestos as a friction material. Ann Occup Hyg, vol. 13, p. 25.

²⁹"The first decision made concerned that part of the dust spectrum which should be counted and it was agreed that only fibers or fiber bundles having a minimum length of 5 microns and a maximum of 100 microns should be counted, the definition of a fiber being arbitrarily taken as a particle whose length was at least three times its diameter. This decision was taken in the light of evidence to the effect that the particle size distribution or spectrum of an asbestos dust cloud was reasonably constant over a wide range of textile processes, although later work has suggested that this might not be strictly true." This decision represents the conclusions made for use of the Thermal Precipitator Method in collecting asbestos-containing dust and when the Membrane Filter Technique came into use, the basis for the method referred to as the PCM method, it was determined that the 5 micron in length would remain the standard as "The filter on the other hand, having a pore size in the region of 0.45 micron, would appear to be quite adequate for trapping fibers in the length range 5-100 microns." While it was thought the Membrane Filter Technique would be more representative in assessing the "true health hazard to which an operative is subjected" it did not rely upon knowledge that fibers less than 5 micron in length had been shown harmless. Holmes S, 1965. Developments in dust sampling and counting techniques in the asbestos industry. Ann NYA Sciences: 132(1); 288-297.

³⁰Yeung, P, patience, K, Apthorpe, L, & Willcocks, D, 1999. An Australian study to evaluate worker exposure to chrysotile in the automotive service industry. Appl Occup Environ Hyg, Vol. 14, No. 7, July, p. 448.

³¹Stanton, M.F., and Wrench, C., 1972. Mechanisms of mesothelioma induction with asbestos and fibrous glass. J. Natl. Cancer Inst., Vol. 48, p. 797.

³²Stanton, M.F., Laynard, M, Tegeris, A, et al. 1981. Relation of particle dimension to carcinogenicity in amphibole asbestos and other fibrous minerals. JNCI, Vol. 67, No. 5, November, p. 965.

probability, might be more hazardous than fewer long fibers, individually of high probability.³³

Studies have also found that the majority of asbestos fibers in lung and mesothelial tissues were shorter than 5µm in length, thus indicating the ability of the shorter fibers to reach the tumor site, remain there, and therefore their role in the etiology of disease is implicated.³⁴ Research has found in typical occupational environments fibers shorter than 5µm in length outnumber the longer fibers by a factor of 10 or more.³⁵

Shorter fibers must be studied in more depth and they should not be disregarded especially when clearance is retarded.³⁶ That chrysotile fibers tend to spit longitudinally as well as partially dissolve, resulting in shorter fibers within the lung, was reported in a review of several articles.³⁷

Davis et al., 1986, 1988 and the Berman et al., 1995 reanalysis of the Davis data and the McDonald et al., 1989 papers examine both the toxicity or lack thereof for short fibers.³⁸ The Davis papers show that: (1) long fibers produced 6 times more fibrosis and 3 times more tumors than the short fiber preparations after inhalation; (2) injection studies, at the highest dose levels 25 mg, found little difference in the numbers of tumors produced by both long and short-fibre chrysotile, while at lower levels there was a significant difference between the long and short-fibre preparations with the longer fibers being more carcinogenic; (3) the mean tumor induction period was longer for the short-fibre preparation in producing mesotheliomas at both the 25mg and 2.5mg dose level and the authors conclude “. . . would probably have been seen with the 0.25mg dose if the short-fibre chrysotile had produced any mesotheliomas at this level.”; and (4) the authors state that the alteration of the short-fibre chrysotile produced by ball-milling is subject to a level of crystal damage which is sufficient to make results difficult to interpret in relation to hazards resulting from short fibres produced during the manufacture of asbestos products or during the subsequent usage of these materials. Berman et al., 1995, using a risk analysis model of their choice choose to eliminate all fibres less than 5 µm in length as “Structures <5 µm in length do not appear to make any contribution to lung tumor risk.” Such an assumption is unwarranted given the conclusions of the Davis et al. papers along with the other data, discussed in this affidavit, showing toxicity for the short asbestos-fibers.

McDonald et al., 1989 examined 78 cases of mesothelioma from autopsy between 1980 through 1984 with matched referents to evaluate the lung burden of long vs. short fibers, concluded that the role of short-fibers was nil. Looking only at lung burden analysis for chrysotile short-fibers is not the only way nor is it the most appropriate analysis to determine the role of either chrysotile or short-fibers, as they are cleared from the lung rapidly compared to longer non-chrysotile fibers. This same criticism is applicable to the Butnor et al.,³⁹ analysis of 10 cases of mesothelioma among brake exposed workers where analysis was only made of lung tissue.

Butnor et al. also dismiss the ‘hit-and-run’ hypothesis for chrysotile as ‘flimsy’ and having no solid scientific support and cite Hesterberg et al., 1994, 1995, 1996 studies,⁴⁰ of man-made vitreous fibers, as their proof for this contention. While there is

³³ Greenberg, M, 1984. S Fibers. Am J Indust Med, Vol. 5, p. 421–422 & Personal correspondence from Dr. Morris Greenberg, 23 May 2003.

³⁴ Suzuki, Y. & Yuen, SR., 2002. Asbestos fibers contributing to the induction of human malignant mesothelioma. Ann NY Acad Sci, Vol. 982, pp. 160–176 & Dodson, RF, O’Sullivan, MF, Brooks, DR & Bruce, JR, 2001. Asbestos content of omentum and mesentery in nonoccupationally exposed individuals. Tox Indust Health, Vol. 17, p. 138.

³⁵ Dement, JM & Wallingford, KM, 1990. Comparison of phase contrast and electron microscopic methods for evaluation of occupational asbestos exposures. Applied Occ Env Hyg, Vol. 5, p. 242.

³⁶ Oberdorster, G, 2001. Fiber characteristics, environmental and host factors as determinants of asbestos toxicity. 2001 Asbestos Health Effects Conference, May 24–25, Oakland, CA, U. S. Environmental Protection Agency.

³⁷ Dement, JM & Brown, DP, 1993. Cohort mortality and case-control studies of white male chrysotile asbestos textile workers. J Occup Med Toxic, Vol. 2, No. 4, p. 355.

³⁸ Davis JM, Addison J, Bolton RE, et al. 1986. The pathogenicity of long versus short fibre samples of amosite asbestos administered to rats by inhalation and intraperitoneal injection. Br J Exp Pathol 67: 415–430; Davis JM, Jones AD. 1988. Comparisons of the pathogenicity of long and short fibres of chrysotile asbestos in rats. Br J Exp Pathol 69: 717–737; Berman DW, Crump KS, Chatfield EJ et al. 1986. The sizes, shapes, and mineralogy of asbestos structures that induce lung tumors or mesothelioma in AF/HAN rats following inhalation. Risk Analysis 15: 181–195; & McDonald JC, Armstrong B, Case B et al. 1989. Mesothelioma and asbestos fiber type: Evidence from lung tissue analyses. Cancer 63: 1544–1547.

³⁹ Butnor KJ, Sporn TA, Roggli VL. 2003. Exposure to brake dust and malignant mesothelioma: A study of 10 cases with mineral fiber analyses. Ann Occup Hyg 47: 325–330.

⁴⁰ Hesterberg TW, Miiller WC, Mast R, McConnell EE, Bernstein DM & Anderson R. 1994. Relationship between lung biopersistence and biological effects of man-made vitreous fibers after

clear proof of the biopersistence for amphibole asbestos, the lack of such biopersistence of other fibers, as shown in the Hesterberg et al papers, provide support to the contrary, and are an indication that pathogenicity of a fiber is dependent upon more than simply the dose, dimension, and the durability of the fibers found with in the lung. It is also important to note that chrysotile asbestos produced fibrosis, lung tumors and mesothelioma in rats after inhalation studies as shown in the Research and Consulting Company (RCC) studies cited in the Hesterberg et al., 1995 paper.

APPENDIX 2

MESOTHELIOMA SURVEILLANCE, RICHARD A. LEMEN, PH.D.

Two recent papers have concluded the beginning of a decrease in mesothelioma rates in the United States.⁴¹ Their data analyses bring to the fore additional questions about the reliability of surveillance data for mesothelioma based solely on death certificate analysis or mortality data without pathological confirmation of mesothelioma. SEER data, for example, prior to the implementation of the new ICD 10 codes, are inaccurate and underestimate the true incidence of mesothelioma in the U.S.⁴²

The new ICD-10 codes for mesothelioma are C45.0 for pleural and C45.1 for peritoneal.⁴³ Before the new ICD-10 codes went into effect in 1999 the reporting based on incidence data was likely underreported and thus analysis using such data is likely to have underreported the incidence of mesothelioma. In some cases, SEER data reported only 12 percent of the mesothelioma cases were accurately reported and even with the new ICD 10 codes it is estimated that only about 80 percent will be detected through SEER data, indicating that mesothelioma reporting will still be problematic but much less so than in the past.⁴⁴ The new ICD 10 codes have only been in existence for the past 8 years and any trends based on this data are unwarranted at this time and it will be many years until a more accurate picture can be seen as to mesothelioma trends within the U.S. It is important that NIOSH address this underreporting gap.

Since it has been generally reported that the incidence of mesothelioma in women is much less associated with asbestos exposure, Steenland et al.⁴⁵ suggest that if take-home asbestos exposure were considered the attributable risks may rise to around 90 percent. Price and Ware (2004) unjustly suggest that because the female lifetime mesothelioma risk across birth cohorts has remained constant this supports a threshold exposure for mesothelioma, which is yet to be shown and no epidemiological study to date has been able to demonstrate such a threshold. Trends in mesothelioma are on the rise in many countries and a large multicentric study on malignant pleural mesothelioma and non-occupational exposures to asbestos projects that low-doses from the home and general environment may carry a measurable risk of mesothelioma over the next few decades.⁴⁶ The findings of this multicentric study have direct implications to the risk of mesothelioma from exposures to asbestos among end-product user of asbestos-containing products, e.g. brake mechanics, as their exposures have generally been of a lower magnitude than those encountered by the various highly exposed and predominately studied trades including insulators, construction workers, shipyard workers, pipefitters to name a few.

chronic inhalation in rats. *Env Health Perspect* 102(S); 133-137; Hesterberg TW, Miiller WC, Thevenaz P, & Anderson R. 1995. Chronic inhalation studies of man-made vitreous fibres: Characterization of fibres in the exposure aerosol and lungs. *Ann Occup Hyg* 39 (5): 637-653
 Hesterberg TW, Miiller WC, Musselman RP, Kamstrup RD, Hamilton RD & Thevenaz P. 1996. Biopersistence of man-made vitreous fibers and crocidolite asbestos in the rat lung following inhalation. *Fund Appl Toxicol* 29: 267-279.

⁴¹Price B & Ware A, 2004. Mesothelioma trends in the United States: An update based on surveillance, epidemiology, and end results program data for 1973 through 2003 &

⁴²Pinheiro GA, Antao VCS, Bang KM & Attfield MD, 2004. Malignant mesothelioma surveillance: A comparison of ICD 10 mortality data with SEER incidence data in nine areas of the United States. *Int J Occup Environ Health*; 10: 251-255.

⁴³World Health Organization, 1992. ICD-10 International Statistical Classification of Diseases and Related Health Problems Tenth Revision: 1: 201.

⁴⁴Pinheiro GA, Antao VCS, Bang KM & Attfield MD, 2004. Malignant mesothelioma surveillance: A comparison of ICD 10 mortality data with SEER incidence data in nine areas of the United States. *Int J Occup Environ Health*; 10: 251-255.

⁴⁵Steenland K, Burnett C, Lalich N, Ward E & Hurrell J, 2003. Dying for work: The magnitude of U.S. mortality from selected causes of death associated with occupation. 43; 461-482.

⁴⁶Magnani C, Agudo A, Gonzalez CA et al., 2000. Multicentric study on malignant pleural mesothelioma and non-occupational exposure to asbestos. *Br J Cancer*: 83(1); 104-111.

DR. RICHARD A. LEMEN, PH.D., M.P.S.H.

DEPOSITION AND TRIAL
TESTIMONY INFORMATION

1996 - 2007

CONSULTANT IN OCCUPATIONAL HEALTH, EPIDEMIOLOGY, AND PUBLIC HEALTH

1996 - 1999

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/ Trial	Type of Case	Firm
Feb. 23, 1996	Paul Cochran, et al. v. Keene Corporation, et al.	A-931,139-C	128 th Judicial District Court of Orange County, Texas	Trial	Asbestos	Baron & Budd, P.C.
May 22, 1996	William B. Hornberger, et al. v. Watson Brothers Plumbing, et al.	94-9718-G	134 th Judicial District Court of Dallas County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Sept. 6, 1996	Jesse A. Justice, et al. v. Owens-Corning Fiberglass, et al.	95-CI-11364	285 th Judicial District Court of Bexar County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Dec. 2, 1996	S. Martinez, et al. v. Owens-Corning Fiberglass, et al.	96-CI-13337	37 th Judicial District Court of Bexar County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Jan. 31, 1997	Tommy Boyles, et al. v. Pittsburgh Corning Corporation, et al.	94-L-1223	20 th Judicial Circuit of Illinois St. Clair County	Trial	Asbestos	Baron & Budd, P.C.

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Mar. 18, 1997	P. Sanchez, et al. v. Kelly-Moore Paint Company, et al.	96-2807	346 th Judicial District Court of El Paso County, Texas	Trial	Asbestos	Baron & Budd, P.C.
Aug. 7, 1997	Clifford Richter, et al. v. Owens-Corning Fiberglass, et al.	22950	20 th Judicial District Court of Milam County, Texas	Deposition	Asbestos	C. Andrew Waters, Esq.
Aug. 7, 1997	James McNiel, et al. v. Owens-Corning Fiberglass, et al.	23076	20 th Judicial District Court of Milam County, Texas	Deposition	Asbestos	C. Andrew Waters, Esq.
Aug. 7, 1997	Charles Shelton, et ux. v. A. C. and S, Inc., et al. In Re: Monongalia II	90-8-12304	24 th Judicial District Court of Calhoun County, Texas	Deposition	Asbestos	C. Andrew Waters, Esq.
Sept. 3, 1997			Circuit Court of Monongalia County of West Virginia	Deposition	Asbestos	Multiple firms
Dec. 12, 1997	R. Bickham, et al. v. Metropolitan Life Insurance Company, et al.	70,760	22 nd Judicial District of Louisiana, Washington Parish	Deposition	Asbestos	Howard, Laudumiey, Mann & Reed
Jan. 27, 1998	In Re: Monongalia II		Circuit Court of Monongalia County of West Virginia	Trial	Asbestos	Mr. McClain?

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Jan. 29-30, 1998	R. Bickham, et al. v. Metropolitan Life Insurance Company, et al.	70,760	22 nd Judicial District of Louisiana, Washington Parish	Deposition	Asbestos	Howard, Laudumiey, Mann & Reed
Feb. 5, 1998	W. Brown, Jr., et al. v. Borg Warner, et al.	95-1922	County Court at Law #2 of El Paso County, Texas	Trial	Asbestos	Baron & Budd, P.C.
Mar. 3, 1998	W. Becton, Sr., et al. v. Rhone-Poulenc, Inc., et al.	H-96-1904	U.S. District for the Southern District of Texas, Houston Division	Deposition	Asbestos	Baron & Budd, P.C.
Apr. 2, 1998	Frank Cashio, et al. v. Owens-Corning Fiberglass, et al.	48,138	18 th Judicial District Court of Louisiana, Iberville Parish	Trial	Asbestos	Baron & Budd, P.C.
July 1-2, 1998	R. Bickham, et al. v. Metropolitan Life Insurance Company, et al.	70,760	22 nd Judicial District of Louisiana, Washington Parish	Deposition	Asbestos	Howard, Laudumiey, Mann & Reed
Aug. 25, 1998	Bendly, et al. v. Exxon, et al.			Trial	Asbestos	
Feb. 2, 1999	Gilbert Moreno, et al. v. National Gypsum, et al.	94-CI-04248	37 th Judicial District of Bexar County, Texas	Trial	Asbestos	Baron & Budd, P.C.

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/ Trial	Type of Case	Firm
Mar. 11, 1999	Robert Durbin, et al. v. Owens-Corning Fiberglass, et al.	49,399	18 th Judicial District Court of Louisiana, Parish of Iberville	Deposition	Asbestos	Baron & Budd, P.C.
Mar. 18, 1999	Kenneth Raper, et al. v. Owens-Corning Fiberglass, et al.	98-8060-C	County Court at Law #3 of Dallas County, Texas	Trial	Asbestos	Baron & Budd, P.C.
Mar. 31, 1999	Jesse M. Watts, et al. v. Owens-Corning Fiberglass, et al.	49,341	18 th Judicial District Court of State of Louisiana Iberville Parish	Trial	Asbestos	Baron & Budd, P.C.
Apr. 16, 1999	Carlton Rivett, et al. v. Anco Insulation, Inc., et al.	1029788	18 th Judicial District Court of Louisiana West Baron Rouge Parish	Deposition	Asbestos	LeBlanc, Maples & Waddell, LLC
June 30, 1999	F. Goodman, et al. v. Georgia Pacific Corporation, et al.	E-68314	Superior Court of Georgia Fulton County	Trial	Asbestos	Baron & Budd, P.C.
Oct. 21, 1999	Clifford Peck, et al. v. A C and S, Inc., et al.	98-102790	Supreme Court of New York St. Lawrence County	Trial	Asbestos	Baron & Budd, P.C.
Nov. 5, 1999	Alvin Hebert, et al. v. Anco Insulation, et al.	51,810	18 th Judicial District Court of State of Louisiana Iberville Parish	Deposition	Asbestos	LeBlanc, Maples & Waddell, LLC

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/ Trial	Type of Case	Firm
Nov. 9, 1999	R. Castillo, et al. v. Garlock, Inc., et al.	97-4051	County Court at Law #3 El Paso County, Texas	Trial	Asbestos	Baron & Budd, P.C.
Nov. 12, 1999	Alvin Hebert, et al. v. Dow Chemical, et al.	51,810	18 th Judicial District Court of State of Louisiana Ibberville Parish Court of Common Pleas of Pennsylvania Allegheny County	Trial	Asbestos	LeBlanc, Maples & Waddell, LLC
Dec. 1, 1999	V. Eisenreich, et al. v. A-Best Products Company, Inc., et al.	97-16440		Trial	Asbestos	Aaron Deluca

2000

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Jan. 14, 2000	William Stark, et al. v. Alaska Steamship Co., et al.	1:98CV20002	U. S. District Court for the Northern District of Ohio Eastern Division	Deposition	Asbestos	Jacques Admiralty
Jan. 31, 2000	Derry v. CSX Transportation, Inc.	92-2167-CA	Fourth Judicial Circuit of Florida Duval County	Deposition	Asbestos	Lane & Gossett
Feb. 7, 2000	P. Amason, et al. v. Allied Signal, et al.	CV98-04124	Circuit Court of Alabama Jefferson County	Deposition	Asbestos	Casino Vaughan Law Office
Feb. 8, 2000	W. Moore, et al. v. Owens Corning Fiberglass Corp., et al.	29668	27 th Judicial District Court of Texas Angelina County	Deposition	Asbestos	Waters & Kraus, LLP
	Hollandsworth, et al. v. Owens Corning Fiberglass, et al.	D960072-C	260 th Judicial District of Texas Orange County			
	R. Durham, et al. v. Able Supply Company, et al.	98-758-B	124 th Judicial District Court of Texas Gregg County			

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Feb. 9, 2000	A. Mayer, et al. v. A C and S, Inc, et al.	99CV000042	Circuit Court Branch 1 of Wisconsin Ozaukee County	Trial	Asbestos	CasinoVaughan Law Offices
Feb., 18, 2000	Cicchillo, et al. v. Pittsburgh Corning Corp., et al.		Ohio	Trial	Asbestos	Kelly & Ferraro, LLP
Feb. 18, 2000	Salerno, et al. v. Foster Wheeler Corp., et al.		Texas	Trial	Asbestos	Jacques Admiralty
Feb. 22, 2000	Jose Peralta, et al. v. Garlock, et al.	99-3990	County Court at Law #3 El Paso County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Feb. 25, 2000	Earlon Nunez, et al. v. Owens Corning, et al.	98-70432-H	Judicial District Court of Louisiana Vermillion Parish	Trial	Asbestos	Baron & Budd, P.C.

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Mar. 22, 2000	B. Hoffman, et al. v. Georgia Pacific, et al.	98-CV-745-RLV	U.S. District Court for the Northern Division of Georgia, Atlanta Division	Deposition	Asbestos	Baron & Budd, P.C.
	In Re: All Asbestos-Related Personal Injury or Death Cases Filed by Baron & Budd, P.C. in Fulton County, GA	1998CV02684	Superior Court of Fulton County, Georgia			
Mar., 2000	Kasum			Deposition	Asbestos	Casino Vaughan Law Office
Apr. 24, 2000	F. Cavanaugh, et al. v. Owens Corning Corp., et al.	99CL08314	57 th Judicial District of Bexar County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
May, 2000	Watson			Deposition	PCB	Stuart Caldwell
May 22, 2000	B. Hoffman, et al. v. Georgia Pacific, et al.	98-CV-745-RLV	U.S. District Court for the Northern Division of Georgia, Atlanta Division	Deposition	Asbestos	Baron & Budd, P.C.
May 24, 2000	Fulmore, et al. v. CSX Trans., et al.	91V-932	Superior Court of Ware County, Georgia		Asbestos	Roger B. Lane, Esq.

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
May 30, 2000	F. Cavanaugh, et al. v. Owens-Corning, et al.	99CJ08314	57 th Judicial District Court of Bexar County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
June 1, 2000	T. Stenaski, et al. v. Allied Signal, et al.	99-CV-080	Circuit Court of Wisconsin Door County	Deposition	Asbestos	CasinoVaughan Law Offices
June, 2000	Baker			Deposition	TCE	Baron & Budd, P.C.
June 12, 2000	V. Howland, et al. v. Owens-Corning, et al.	40,551	County Court of Smith County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
July, 2000	TCE Ground Water Contamination case			Trial	TCE	Baron & Budd, P.C.
July, 2000	Samuel D. Lambert, et al.			Deposition	Asbestos	Paul, Reich & Myers
July, 2000	R. Bickham, et al. v. Metropolitan Life Insurance Company, et al.	70,760	22 nd Judicial District of Louisiana Washington Parish	Deposition	Asbestos	Howard, Laudumley, Mann, Reed & Hardy
Aug. 1, 2000	S. Blumberg, et al. v.	95-C-559	5 th Judicial District Court of Cass County, Texas	Deposition	Asbestos	Waters & Kraus, LLP
Aug, 2000	Able Supply, et al. J. Blackburn, et al. v. Owens-Corning Fiberglass, et al.	98-03696-F	116 th Judicial District of Dallas County, Texas	Trial	Asbestos	Baron & Budd, P.C.

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Aug. 4, 2000	V. Howland, et al. v. Owens-Corning, et al.	40,551	County Court of Smith County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Aug. 17, 2000	V. Howland, et al. v. Owens Corning, et al.	40551	County Court of Smith County, Texas	Trial	Asbestos	Baron & Budd, P.C.
Aug. 29, 2000	Earl Atchison, et al. v. A C and S, Inc., et al.		Superior Court of California Alameda County	Deposition	Asbestos	Shepherd Hoffman
Aug. 31, 2000	Dews, et al. v. Swan Transportation, et al. Coutson		Texas	Trial	Asbestos	
Sept., 2000				Deposition	Asbestos	Cook, Doyle & Bradshaw
Sept. 26, 2000	H. Plumb, et al. v. A C and S, Inc., et al.	00-106851	New York Supreme Court St. Lawrence County	Trial	Asbestos	Baron & Budd, P.C.
Oct. 5, 2000	W. Roberts, et al. v. Exxon, et al.	45664-A	18 th Judicial District Court of Louisiana Iberville Parish	Trial	Asbestos	Baron & Budd, P.C.
Oct. 12, 2000	Jorden Wyatt, et al. v. Brown & Root, et al.	97-42860	189 th Judicial District Court of Harris County, Texas	Trial	Asbestos	Williams & Bailey

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Oct, 2000	Hamilton v. Anco Insulation, Inc., et al. Satterfield			Deposition	Asbestos	LeBlanc, Maples & Waddell
Nov., 2000	Berry			Deposition	Asbestos	Casino Vaughan Law Office
Nov. 20-21, 2000	Shelton, et al. v. Pittsburgh Corning Corp., et al. Fred Gilcrease, et al. v. A C and S, Inc. et al. Lewis, et al.	00-0016-C 99-C-07037	241 st Judicial District Court in Smith County, Texas 73 rd Judicial District of Bexar County, Texas	Trial Deposition	Railroad Solvent Asbestos	Lane & Gossett Waters & Kraus. LLP
Nov. 22, 2000	A.P. Green Industries, Inc., et al. B. Hoffman, et al.	49D02-9501- MI-001-290	Superior Court, Civil Division of Marion County, Indiana	Deposition	Asbestos	Ness, Motley, Loadholt, Richardson & Poole, P.A.
Dec. 5, 2000	USXX Corporation, et al.	1:98-CV-745- TWT	U.S. District Court of the Northern District of Georgia Atlanta Division	Trial	Asbestos	Baron & Budd, P.C.

2001

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Jan., 2001	Chavez		Philadelphia	Trial	Asbestos	Foster & Sear
Jan., 2001				Trial	Asbestos	Paul, Reich & Myers
Jan. 10, 2001	F. Davis, et al. v. Owens-Corning Fiberglass Corp., et al.	96-3062	County Court at Law #3 El Paso County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Jan. 29, 2001	H. Wells, et al. v. U.S. Gypsum, et al.	A-161,748	58 th Judicial District Court of Jefferson County, Texas	Trial	Asbestos	Baron & Budd, P.C.
Feb., 2001	Flexitallic & USG			Trial	Asbestos	Robins & Cloud
Feb. 1, 2001	H. Clark, et al. v. Union Pacific, et al.	98-3176	310 th Judicial District of Nueces County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Feb. 2, 2001	S. Toney, et al. v. Georgia Pacific Corporation, et al.	1999CV17762	Superior Court of Georgia Fulton County	Deposition	Asbestos	Baron & Budd, P.C.
Feb., 2001				Trial	Asbestos	Silber Peatman

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Feb. 14, 2001	Calderon, et al. v. GAF Corporation, et al.	2000-1703	168 th Judicial District Court of El Paso County, Texas	Trial	Asbestos	Baron & Budd, P.C.
Feb. 19, 2001	F. Davis, et al. v. Owens-Corning Fiberglass, et al.	96 -3062	County Court at Law #3 of El Paso County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Feb., 2001	W. Horton, et al			Deposition	Asbestos	Kaeske & Reeves
Feb., 2001	TCE		Phoenix, Arizona	Trial	TCE	Golberg, Jennings & White
Feb., 2001	Kane			Deposition	Asbestos	Ness, Motley, Loadholt, Richardson & Poole
Mar., 2001	Weaver			Trial	Asbestos	Patten, Wornom, Hatten & Diamonstein, LLC
Mar. 8, 2001	Bufford, et al. v. U.S. Gypsum, et al.	97-2590-E	101 st Judicial District Court of Dallas County, Texas	Trial	Asbestos	Baron & Budd, P.C.

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Mar. 19, 2001	Cogley, et al. v. Foseco, Inc., et al.	CV-318966	Court of Common Pleas of Ohio Cuyahoga County	Deposition	Asbestos	Patten, Wornom, Hatten & Diamonstein, LLC
Mar. 30, 2001	Jones, et al. v. Owens-Corning Fiberglass Corp., et al.		Superior Court of Georgia Fulton County	Deposition	Asbestos	Lane & Gossett, P.C.
Apr. 19, 2001	Francis, Sr., et al. v. Dresser Industries, et al.	99-CV-0739	212 th Judicial District Court of Galveston County, Texas	Deposition	Asbestos	Robins & Cloud
	M. Castro, et ux. v. Dresser Industries, et al.	B-158,036	172 nd Judicial District Court of Galveston County, Texas			
Apr. 24, 2001	V. Brunell, et al. v. A.L. Burbank & Company, Ltd., et al.	97-4198	Civil District Court of Louisiana Orleans Parish	Trial	Asbestos	Martzell & Bickford
June 11, 2001	McKinley, et al. v. Gen. Corp., Inc., et al.		Court of Common Pleas of Ohio Ashtabulas County	Deposition	Asbestos	William B. Baggett, Jr.

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
June 18, 2001	Jerrigan Trial Group: Limited to Harry Bargelski	00C-06-260ASB	Superior Court of Delaware New Castle County	Deposition	Asbestos	Baron & Budd, P.C.
June 19, 2001	J. Derose, et al. v. Tenneco, Inc., et al.	89-643	34 th Judicial District Court of Louisiana St. Bernard Parish	Deposition	Asbestos	LeBlanc & Waddell
July 5, 2001	J. Peralta, et al. v. Owens Corning, et al.	99-3990	County Court at Law #3 of El Paso County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Aug. 14, 2001	Hutchison, et al. v. Able Supply Company, et al.	18,215	32 nd Judicial District Court of Nolan County, Texas	Trial	Asbestos	Waters & Kraus, LLP
Aug. 21, 2001	Hernandez, et al. v. Kelly-Moore Paint Company	2000-3559	County Court at Law #3 of El Paso County, Texas	Trial	Asbestos	Baron & Budd, P.C.
Aug. 31, 2001	Reed, et al. v. Avondale Industries, Inc., et al.	95-17141	Civil District Court of Louisiana Orleans Parish	Deposition	Asbestos	Martzell & Bickford
	J. Bryan, et al. v. Alcoa Steamship, Inc., et al.	98-19651				

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Sept. 14, 2001	Hess, et al. v. Norfolk Southern Railroad Company, et al.	398164	Court of Common Pleas of Ohio Cuyahoga County	Deposition	Asbestos	Kevin McDermott
Sept. 27, 2001	Guillory, et al. v. Abex Corporation, et al.	E-164,041	172 nd Judicial District Court of Jefferson County, Texas	Deposition	Asbestos	Ethan L. Shaw
Oct. 3-4, 2001	J. Bailey, et al. v. Norfolk & Southern Railway Company, et al.	94-03454C	331 st Judicial District Court of Travis County, Texas	Trial	Asbestos	Baron & Budd, P.C.
Oct. 5 & 10, 2001	T. Cichoki, et al. v. A C and S, Inc., et al.	2001-1082	County Court at Law #3 of El Paso County, Texas	Trial	Asbestos	Waters & Kraus, LLP
Oct. 8, 2001	Henderson, et al. v. A C and S, Inc., et al.	98-GS-23-2792	State of South Carolina County of Greenville	Trial	Asbestos	Christopher Mauriello, Esq. Robert Paul, Esq.

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Oct. 12, 2001	Joe Keller, et al. v. Pittsburgh Corning Corp., et al.	99-CV-1224	122 nd Judicial District Court of Galveston County, Texas	Deposition	Asbestos	
Oct. 16, 2001	L. Howard, et al. v. A C and S, Inc., et al.	33,246	62 nd Judicial District Court of Hopkins County, Texas	Deposition	Asbestos	Waters & Kraus, LLP
Oct. 26, 2001	L. Bailey, et al. v. A-Best Products Co., et al.	37258	Court of Common Pleas of Ohio Cuyahoga County	Deposition	Asbestos	Goldberg, Persky, Jennings & White
Nov. 8, 2001	Wakeland, et al. v. Garlock, et al.		Illinois	Trial	Asbestos	
Nov. 19, 2001	Robinson, et al. v. NARCO, et al.	CV939414	Court of Common Pleas of Ohio Cuyahoga County	Trial	Asbestos	Baron & Budd, P.C.
Nov. 21, 2001	Bargelski, et al. v. Garlock, et al.	00C-04-260-ASB	Superior Court of Delaware, New Castle County	Trial	Asbestos	Baron & Budd, P.C.
Nov. 28, 2001	Robinson, et al. v. NARCO, et al.	CV393414	Court of Common Pleas of Ohio Cuyahoga County	Trial	Asbestos	Baron & Budd, P.C.

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Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/ Trial	Type of Case	Firm
Jan. 17, 2002	Longnecker, et al. v. Commonwealth Edison Co., et al.				Asbestos	Casino Vaughan Law Offices
Jan. 31, 2002	Quigley		Ohio		Asbestos	Kelly & Ferraro, LLP
Feb. 1, 2002	In Re: Asbestos Personal Injury Civil Action Cases	01-C-9002	Circuit Court of Kanwha County, West Virginia	Deposition	Asbestos	Ness, Motley, Loadholt, Richardson & Poole P.C.
Feb. 14-15, 2002	J. Pace, et al. v. Synkoloid Company, et al.	94-4749-C	94 th Judicial District Court of Nueces County, Texas	Trial	Asbestos	Baron & Budd, P.C.
Feb. 25-26, 2002	Luce, et al. v. Dupont, et al.	157, 109	172 nd Judicial District Court of Jefferson County, Texas	Deposition	Asbestos	Waters & Kraus, LLP
Mar. 8, 2002	Hill, et al. v. A C and S, et al.	2001-CI-06058	288 th Judicial District Court of Bexar County, Texas	Deposition	Asbestos	Waters & Kraus, LLP

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Mar. 11, 2002	W Roberts, et al. v. A.P. Green, et al.	49D02-9601-MI-0001-687	Superior Court of Marion County, Indiana	Deposition	Asbestos	Ness, Motley, Loadholt, Richardson & Poole
	Mary K. Denny, et al. v. A C and S, et al.	49D02-9601-MI-0001-662				
Mar. 19, 2002	McAnally, et al. v. Pittsburgh Corning, et al.	99-CV-1223	122 nd Judicial District of Galveston County, Texas	Deposition	Asbestos	Robins, Cloud, Greenwood & Lubel, LLP
Mar. 25-26, 2002	R. Maney, et al. v. A C and S, Inc., et al.	AO-1033-C	129 th Judicial District Court of Orange County, Texas	Deposition	Asbestos	Waters & Kraus, LLP
	R. Wheeler, et al. v. A C and S, Inc., et al.	CV33246-A	62 nd Judicial District Court of Hopkins County, Texas			
	E. Royer, et al. v. A C and S, Inc., et al.	14293-RM00	District Court of Brazoria County, Texas			
	W. Gabbert, et al. v. A C and S, Inc., et al.	1010259-C	128 th Judicial District Court of Orange County, Texas			

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
	Douglas Kwasnik, et al. v. A C and S, Inc., et al.	2001-297	County Court at Law #3 in El Paso County, Texas			
	Donald Cipov, et al. v. A C and S, Inc., et al.	141-189827-01	141 st Judicial District of Tarrant County, Texas			
Apr. 23, 2002	Umphries, et al. v. A C and S, Inc., et al.	01-6745-M	298 th Judicial District Court of Dallas County, Texas	Deposition	Asbestos	Waters & Kraus, LLP
	J. Freeman, et al. v. A C and S, Inc., et al.		County Court at Law #2 of Dallas County, Texas			
May 9, 2002	R. Dailey, et al v. GAF Corporation, et al.	CC-00-06741-B	County Court at Law #2 of Dallas County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
May 10, 2002	J. Mansel, et al. v. U.S. Gypsum, et al.	15287	149 th Judicial District Court of Brazoria County, Texas	Deposition	Asbestos	Clyde Joe Beatty, Sr.
May 16, 2002	McAnalley, et al. v. Pittsburgh Corning Corp., et al.	99-CV-1223	122 nd Judicial District of Galveston County, Texas	Deposition	Asbestos	Robins, Cloud, Greenwood & Label, LLP

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
May 23, 2002	J. Freeman, et al. v. A C and S, Inc., et al.	01-00742B	County Court at Law #2 of Dallas County, Texas	Trial	Asbestos	Waters & Kraus, LLP
May 28, 2002	Thomson, et al. v. Able Supply Co., et al. D. Beary, et al. v. Owens-Corning Fiberglass, et al. Lois Salter, et al. v. Armstrong World Industries, Inc., et al. Sharon Sturdevant, et al. v. A C and S, Inc., et al.	2002-184	Court Court at Law of El Paso County, Texas	Deposition	Asbestos	Bradshaw Law
June 20, 2002	Moore v. Brown & Root, Inc., et al.		Texas	Trial	Asbestos	
June 28, 2002	N. McGary, et al. v. Crown, Cork & Seal, et al.		102 nd Judicial District Court of Bowie County, Texas	Deposition	Asbestos	Baron & Budd

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
July 26, 2002	McKenzie, et al. v. A.P. Green, et al.	1999-42808	15 th Judicial District Court of Harris County, Texas	Deposition	Asbestos	Ness, Motley, Loadholt, Richardson & Poole, P.A.
July 30, 2002	Pecukonis, et al. v. A C and S, Inc., et al.	348-187059-01	348 th Judicial District Court of Tarrant County, Texas	Deposition	Asbestos	Waters & Kraus, LLP
	D. Cipov, et al. v. A C and S, Inc., et al.	141-189827-01	141 st Judicial District Court of Tarrant County, Texas			
	Arrowood, et al. v. A C and S, Inc., et al.	A-010037-C	128 th Judicial District Court of Orange County, Texas			
	Short, et al. v. A C and S, Inc., et al.	GNI-02143	53 rd Judicial District Court of Travis County, Texas			
Aug. 5, 2002	C. Duncan, et al. v. Owens-Corning Fiberglass, et al. L. Robin, et al. v. Owens-Corning Fiberglass, et al.	00-04543-F D16170	116 th Judicial District Court of Dallas County, Texas 136 th Judicial District Court of Jefferson County, Texas	Deposition	Asbestos	Foster & Sear

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/ Trial	Type of Case	Firm
Aug. 5, 2002	B. Drake, et al. v. Garlock, Inc., et al.	2001-2537	County Court at Law #6 of El Paso County, Texas	Deposition	Asbestos	Waters & Kraus, LLP
Aug. 6 & 19, 2002	G. Brunner, et al. v. A C and S, Inc., et al.	2001-1880	County Court at Law #3 of El Paso County, Texas			
Aug. 20, 2002	McKenzie, et al. v. A.P. Green, et al.	1999-42808	157 th Judicial District Court of Harris County, Texas	Trial	Asbestos	Ness, Moiley, Loadholt, Richardson & Poole, P.A.
Aug. 20, 2002	R. Chavez, et al. v. A C and S, Inc., et al.		County Court at Law #3 of El Paso County, Texas		Asbestos	Brent Coon & Associates
Aug. 26, 2002	M. Colvin, et al. v. A C and S, Inc., et al.		County Court at Law #1 of Dallas County, Texas		Asbestos	Waters & Kraus, LLP
Aug. 27, 2002	L. Bryant, et al. v. U.S. Gypsum Company, et al.	45 215-A	County Court at Law #2 of Smith County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Aug. 29, 2002	A.J. Miller, et al. v. GAF Corporation, et al.	00-06517-K	192 nd Judicial District Court of Dallas County, Texas	Deposition	Asbestos	Baron & Budd, P.C.

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Aug. 30, 2002	J. Auzenne, et al. v. U. S. Gypsum Company, et al.	CC-01-05948-C	County Court at Law #3 of Dallas County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Aug. 31, 2002	J.W. Byron, et al. v. Falcon MPP, Corp. et al.	2-02-CV-24	U.S. District Court, Eastern District of Texas		Asbestos	Welborn, Houston, Adkison, Mann, Sadler & Hill
Sept. 6, 2002	M.A. Goss, et al. v. Owens Illinois, et al.				Asbestos	Waters & Kraus, LLP
Sept. 6 & 10, 2002	J.Mullen, et al. v. A C and S, Inc., et al.	01-4294	193 rd Judicial District of Dallas County, Texas	Deposition	Asbestos	Waters & Kraus, LLP
Sept. 16, 2002	Brannon, et al. v. Weyerhaeuser Company, et al.	117831	Before the North Carolina Industrial Commission		Asbestos	Wallace & Graham
	Lawrence, et al. v. Owens-Corning Fiberglass Corp., et al.	99-7072-B	117 th Judicial District of Nueces County, Texas		Asbestos	Foster & Sear
Sept. 19, 2002	J. Lausford, et al. v. Able Supply Company, et al.	26658	Judicial Court of Shelby County, Texas		Asbestos	Waters & Kraus, LLP

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Sept. 23, 2002	H. Daniels, et al. v. A C and S, Inc., et al.	B-150, 374	60 th Judicial District Court of Jefferson County, Texas	Deposition	Asbestos	Ness, Motley, Loadholt, Richardson & Poole
	R. Havens, et al. v. A C and S, Inc., et al.	B-150, 374-AJ	60 th Judicial District Court of Jefferson County, Texas			
	In R: All Asbestos Cases R.J. Burts, et al. v. Amoco Chemical Company, et al. Jose Ayala, et al. v. Alcoa, Inc., et al.	CL99-2000-00 99-CV-1214 00-6775-D	Circuit Court of Newport News, VA 212 th Judicial District Court of District Court of 105 th Judicial District Court of Nueces County, Texas	Deposition	Asbestos	Robins, Cloud, Greenwood & Lubel, LLP
Oct. 7, 2002	T.G. Moss, et al. v. GAF Corporation, et al.	00-CV-1111	10 th Judicial District Court of Galveston County, Texas			
	Bobby Fife, et al. v. GAF Corporation, et al.	00-CV-0611	122 nd Judicial District Court of Galveston County, Texas			
	Asbestos trial	01-C-9004	Circuit Court of	Trial	Asbestos	Mr. Hulsey

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
	group		Kanawha County, West Virginia			
Oct. 28, 2002	E. Allcorn, et al. v. Ethyl Corp., et al.	1993-42492	55 th Judicial District Court of Harris County, Texas	Deposition	TCE	Williams & Bailey
Nov., 2002	Bell, et al. v. Celanese, et al.		Salisbury, NC	Trial	Asbestos	Troy Chandler, Esq.
Nov. 7, 2002	Freeman, et al. v. Quigley, et al.		Texas	Trial	Asbestos	
Nov. 7, 2002	Lansford, et al. v. Able Supply Company, et al.				Asbestos	
Nov. 11, 2002	M. Jones, et al. v. A C and S, Inc., et al.	01-10131	116 th Judicial District Court of Dallas County, Texas		Asbestos	Kaeske Law Firm
Nov. 12, 2002	J. Caudillo, et al. v. Able Supply Company, et al.	02-60464-00-04	County Court at Law #4 of Nueces County, Texas		Asbestos	Watts Law Firm
Nov. 21, 2002	VonRaeder, et al. v. Crown, Cork & Seal, et al.	DV02-02436-C	68 th Judicial District Court of Dallas County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Nov. 21, 2002	C. Flowers, et al. v.	01-vs-014834-D	State Court of Fulton County,	Trial	Asbestos	Richardson, Patrick

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
	A C and S, Inc, et al.		State of Georgia			
Nov. 22, 2002	S. Lucero, et al. v. Crown, Cork & Seal, et al.	2001-2577	County Court at Law of El Paso County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Dec. 4, 2002	VonRaeder, et al. v. Crown, Cork & Seal Company, et al.	02-02436-C	68 th Judicial District Court of Dallas County, Texas	Trial	Asbestos	Baron & Budd, P.C.
Dec. 18, 2002	Chambers, et al. v. National Service Industries, et al.	2001-20	Circuit Court of Claborne County, Mississippi		Asbestos	Wells, Moore, Simmons & Hubbard, PLLC
Dec. 19, 2002	T. Harmon, et al. v. Owens Corning Fiberglass, et al.	99-06508-M	298 th Judicial District Court of Dallas County, Texas	Deposition	Asbestos	Baron & Budd, P.C.

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Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Jan. 10, 2003				Deposition	Asbestos	Casino Vaughan Law Office
Jan. 13, 2003	R. Hill, et al. v. A C and S, Inc. et al.	2001-C1-06058	288 th Judicial District Court of Bexar County, Texas		Asbestos	Waters & Kraus, LLP
Jan. 14, 2003	Sidebottom, et al. v. GAF Corp., et al.	C-2036-00-F	332 nd Judicial District Court of Hidalgo County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Jan. 14-15, 2003	F. Sanchez, et al. v. Celanese, et al.	99-6508-M	298 th Judicial District of Dallas County, Texas	Trial	Asbestos	Baron & Budd, P.C.
Jan. 15, 2003				Deposition	Asbestos	Patten, Wormon, Harten & Diamonstein, LLC
Feb. 3, 2003	J.W. Duckett, et al. v. Able Supply Company, et al.	01-CV-280	County Court at Law #1 of Calhoun County, Texas	Deposition	Asbestos	Brent Coon & Associates

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm	
Feb. 9, 2003	H. Bryant, et al. v. Quigley Company, et al.	03CV1877	122 nd Judicial District Court of Galveston County, Texas	Deposition	Asbestos	Baron & Budd, P.C.	
	Hernandez, et al. v. Quigley Company, et al.	26996	149 th Judicial District Court of Brazoria County, Texas				
	C. Brannan, et al. v. Quigley Company, et al.	03-05379-A	14 th Judicial District Court of Dallas County, Texas				
	S. Hubbs, et al. v. Crown, Cork & Seal, et al.	18977*JG02	239 th Judicial District Court of Brazoria County, Texas				
	Perez, et al. v. Quigley Company, et al.	26994	239 th Judicial District Court of Brazoria County, Texas				
	J. Qilantan, et al. v. Quigley Company, et al.	27116	149 th Judicial District Court of Brazoria County, Texas				
	C. Orchard, et al. v. Quigley Company, et al.	02-0094-A	14 th Judicial District Court of Dallas County, Texas				

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Feb. 20, 2003	J.W. Duckett, et al. v. Able Supply Company, et al.	01-CV-280	County Court at Law #1 of Calhoun County, Texas	Trial	Asbestos	Brent Coon & Associates
Feb. 24, 2003	Connie Beal, et al. v. A. P. Green, et al.	2001-449958-CV	Court of Common Pleas of Cuyahoga County, Ohio	Deposition	Asbestos	Ness, Motley, Loadholt, Richardson & Poole
Feb. 27, 2003	John E. Liles, et al. v. Ametek, et al. Kenneth C. Knight, et al. v. Ametek, Inc., et al.	17656*[G01	23 rd Judicial District of Brazoria County, Texas 239 th Judicial District Court of Brazoria County, Texas	Deposition	Asbestos	Kaeske Law Firm
Mar. 6, 2003	D. Driver, et al. v. Able Supply Co., Inc., et al. J. Dixon, et al. v. GAF Corporation, et al.	01-CV-1211 14274	40 th Judicial District Court of Galveston County, Texas 239 th Judicial District Court of Brazoria County, Texas	Deposition	Asbestos	Robins, Cloud, Greenwood & Lubel, LLP
Mar. 6, 2003	Charles F. Dudley, et al. v. AMOCO Chemical Company, et al.	99CV0757	40 th Judicial District Court of Galveston County, Texas	Deposition	Asbestos	Robins, Cloud, Greenwood & Lubel, LLP

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
	Willis Fears, et al. v. GAF Corporation	01-CV-1110	405 th Judicial District Court of Galveston County, Texas			
	J. Frost, et al. v. Able Supply Company, et al.	01-CV-0490	10 th Judicial District Court of Galveston County, Texas			
	A.P. Howard, et al. v. Able Supply Company, et al.	02-CV-0422	10 th Judicial District Court of Galveston County, Texas			
	D. Longoria, et al. v. ALCOA, et al.	99-7095-E	148 th Judicial District Court of Nueces County, Texas			
	T.G. Moss, et al. v. GAF Corporation, et al.	01-CV-1111	10 th Judicial District Court of Galveston County, Texas			
	J.G. Peck, et al. v. GAF Corporation, et al.	01-CV-1099	122 nd Judicial District Court of Galveston County, Texas			
	W. Temple, et al. v. Able Supply Company, et al.	01-CV-0571	122 nd Judicial District Court of Galveston County, Texas			

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
	G. Twist, et al. v. AMOCO Chemical Company, et al. J. Monzingo, et al. v. GAF Corporation, et al.	8111*JG99	239 th Judicial District of Brazoria County, Texas			
	Jose Ayala, et al. v. ALCOA, Inc., et al.	01-CV-1085	122 nd Judicial District Court of Galveston County, Texas			
	Armstrong, et al. v. Able Supply Company, et al.	00-6775-D	105 th Judicial District Court of Nueces County, Texas			
	D. Zumwalt, et al. v. Air Products and Chemicals, et al.	02-CV-0695	122 nd Judicial District Court of Galveston County, Texas			
	S. Mekvin, et al. v. Texaco, Inc., et al.	99-CV-0131	10 th Judicial District Court of Galveston County, Texas			
Mar. 17, 2003		B-150,374-AU	60 th Judicial District court of Jefferson County, Texas		Asbestos	Provost Umphrey Law Firm
Mar. 24, 2003	All Pending Weythacuser cases		Before the North Carolina Industrial Commission		Asbestos	Wallace & Graham

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Mar. 26, 2003	D. Marr, et al. v. Quigley Company, Inc., et al.	19452*BH02	23 rd Judicial District Court of Brazoria County, Texas		Asbestos	Baron & Budd, P.C.
Apr. 3, 2003	Merchant Seaman					Donnie Young, Esq.
Apr. 21, 2003	Mehia, et al. v. Union Carbide, et al.				Asbestos	Troy Chandler
Apr. 24, 2003	S. Melvin, et al. v. Pittsburgh Corning Corp., et al.	B-150,374-BE	60 th Judicial District Court of Jefferson County, Texas	Deposition	Asbestos	Provost Umphrey
Apr. 29, 2003	R. O'Neal, et al. v. Crow, Cork & Seal, et al. J. Vallejo, et al. v. Quigley Company, et al.	01-4447-D 2002-5480	105 th Judicial District Court of Nueces County, Texas 205 th Judicial District Court of El Paso County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
May 2, 2003	S. Beck, et al. v. U.S. Gypsum Company, et al.	2001-2098	County Court At Law #3 El Paso County, Texas	Deposition	Asbestos	Baron & Budd, P.C.

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
May 14, 2003	M. Mitchell, et al. v. Ametek, et al.	02-8988	13 th Judicial District Court of Dallas County, Texas	Deposition	Asbestos	Kaeske Law Firm
May 14, 2003	P. Gustafson, et al. v. Ametek, et al.		19 th Judicial District Court of Dallas County, Texas	Deposition	Asbestos	Kaeske Law Firm
May 22, 2003	E. Alvarado, et al. v. Aventis Corp. Science, et al.				Asbestos	Motley Rice
May 27, 2003	K. Madden, et al.			Deposition	Asbestos	Gary DiMuzio, Esq.
May 29, 2003	Certainteed trial			Trial	Asbestos	Motley Rice
June 12, 2003	Hill			Deposition	Asbestos	Casino Vaughan Law Office
June 25, 2003	W.S. Cowan, et al. v. Crown, Cork & Seal, et al.	A-010526	U.S. District Court for the Northern District of Georgia, Atlanta Division	Deposition	Asbestos	Baron & Budd, P.C.
July 8, 2003	Gallagher, et al. v. Union Carbide Corp., et al.				Asbestos	Richardson, Patrick, Westbrook & Brickman
July 16, 2003	Bennett, et al. v. Sulzer Metco, et al.					Jones, Martin, Parris & Tressener, LLC

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
July 28, 2003	E. Bradley, et al. v. Ford Motor Company, et al.	00C-05-31-ASB	Superior Court of Delaware, New Castle County		Asbestos	Jacobs & Crumplar
July 28, 2003	E. Waishes, et al. v. Ford Motor Company, et al.	02C-06-130-ASB	Superior Court of Delaware, New Castle County		Asbestos	Jacobs & Crumplar
July 29, 2003	J. Gallagher, et al. v. Union Carbide, et al.	GN-202177	200 th Judicial District Court of Travis County, Texas	Deposition	Asbestos	Richardson, Patrick & Brickman, LLC & The Hendler & The Hendler Law Firm
July 30, 2003				Deposition	Asbestos	Motley Rice, LLC
Aug 4, 2003	Douma, et al. v. ABB, et al.				Asbestos	Kaeske Law Firm
Aug 7, 2003	Pippen, et al. v. Quigley, et al.			Trial	Asbestos	Troy Chandler
Aug 8, 2003	Eddie Caffey, et al. v. Foster Wheeler Energy Company, et al.	01-C-753	5 th Judicial District Court of Cass County, Texas		Asbestos	Motley Rice, LLC
Aug 11, 2003	Reta Orem					
Aug 18, 2003	G. Deakins, et al. v.	2002-VS-034346	State Court of Fulton County,		Asbestos	Motley Rice, LLC

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Aug. 18, 2003	A C and S, Inc., et al. v. C. Allen, et al.		Georgia		Asbestos	Motley Rice, LLC
Aug. 25, 2003	A C and S, Inc., et al. Hill, Nolan, Coen, et al. v.				Asbestos	Casino Vaughan
Aug. 29, 2003	J. Campbell, et al. v. Quigley Company, et al.	03-3376-G	347 th Judicial District Court of Nueces County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
	Abel Canales, et al. v. Quigley Company, et al.	03-3381-B	117 th Judicial District Court of Nueces County, Texas			
	G.A. Balli, et al. v. Owens-Corning Fiberglass, et al.	98-03869-G	319 th Judicial District Court of Nueces County, Texas			
	E.Amsworth, et al. v. Crown, Cork & Seal, et al.	02-02429-J	191 st Judicial District Court of Dallas County, Texas			

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Aug. 29, 2003	Laurel, et al. v. Quigley Company, et al.	02-08380-J	191 st Judicial District Court of Dallas County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
	E. Reynolds, et al. v. Exxon Mobil Corporation, et al.	02-08205-C	68 th Judicial District Court of Dallas County, Texas			
	D. Barnhill, et al. v. Quigley Company, et al.	03-04759-E	101 st Judicial District Court of Dallas County, Texas			
	L. Malveaux, et al. v. Quigley Company, et al.	03-05227-C	68 th Judicial District Court of Dallas County, Texas			
Sept. 4, 2003	M. Ramirez, et al. v. Owens-Corning Fiberglass, et al.	97-4054-B	214 th Judicial District Court of Nueces County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Sept. 11, 2003	Jones, O'Sheen, et al. v. USX, et al.			Deposition	Asbestos	Lane & Gossett
Sept. 15, 2003	Jones, O'Sheen, et al. v. USX, et al.			Trial	Asbestos	Lane & Gossett
Sept. 16, 2003	Richardson, et al. v. AMF, Inc., et al.		60 th Judicial District Court of Jefferson County, Texas		Asbestos	Herschel Dodson

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Sept. 16, 2003	Jameson, et al v. Texaco, Inc., et al				Asbestos	Bergman, Senn, Pageler & Frocht
Sept. 22, 2003	S. Yeager, et al. v. Marathon Oil Company, et al	2002-L-17	2 nd Judicial Circuit Court of Crawford County, Illinois		Asbestos	Motley Rice, LLC
Sept. 24, 2003	J. Douglas, et al. v. Henry Vogt Machine Company, et al	02-05005	44 th Judicial District Court of Dallas County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Sept. 25, 2003	Saville, Scafa		Baltimore, Maryland	Trial	Asbestos	Motley Rice, LLC
Oct. 1, 2003	R. Messier, et al. v. Honeywell International, Inc., et al.	2002-61853	215 th Judicial District Court of Harris County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Oct. 6, 2003	Peggy Bell, et al. v. Ametek, et al	22047-JG02	239 th Judicial District Court of Brazoria County, Texas	Deposition	Asbestos	Clark, Depew & Tracey, LLC
Oct. 16, 2003	In Re: Asbestos Personal Injury Litigation	03-C-9600-2	Circuit Court of Kanawha County, West Virginia	Deposition	Asbestos	Motley Rice, LLC

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Oct. 17, 2003	R. Alvarado, et al. v. Certainteed, et al.	2002-7-1494	135 th Judicial District Court of Calhoun County, Texas	Deposition	Asbestos	Richardson, Patrick, Westbrook & Brickman, LLC
Oct. 17, 2003	S. Szabo, et al. v. Anchor Packing Company, et al.	GN-203244	126 th Judicial District Court of Travis County, Texas	Deposition	Asbestos	Richardson, Patrick, Westbrook & Brickman, LLC
Oct. 21-22, 2003	William Lomas			Trial	Asbestos	Brent Coon & Associates
Oct. 27, 2003	Beddingfield, et al. v. Quigley Company, et al.	24525-PS03	239 th Judicial District Court of Brazoria County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
	Jackson, et al. v. Quigley Company, et al.	03CV0736	122 nd Judicial District Court of Galveston County, Texas			
Oct. 28, 2003	Varnadove, et al. v. CSX Trans., et al.			Trial	Asbestos	Roger Lane & Associates
Oct. 29, 2003	D. Bertucci, et al. v. Northrup Grumman Systems, et al.	2002-17551	Civil District Court for the Parish of Orleans, State of Louisiana	Deposition	Asbestos	Martzell & Bickford, LLC

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Nov. 10, 2003	K. Blandford, et al. v. Garlock Sealing Tech., et al.	437948	Court of Common Pleas of the State of Ohio, Cuyahoga County	Trial	Asbestos	Baron & Budd, P.C.
Nov. 14, 2003	Ingraham, et al. v. Anchor Packing Company, et al.				Asbestos	Christian Hartley
Nov. 17, 2003	Bourgeois, et al. v. David Colvin, et al.	98049	16 th Judicial District Court, Division B		Asbestos	
Nov. 18, 2003	Robinson, et al.		Brunswick, Georgia	Trial	Asbestos	Roger Lane
Nov. 20, 2003	v. NVE, Electric Hose & Rubber, et al.				Asbestos	Jacobs & Crumplar
Nov. 25, 2003	Michael Chauvin, et al.			Deposition	Asbestos	Martzell & Bickford
Dec. 4, 2003	Adams, et al. v. A.W. Chesterton, et al.				Asbestos	Motley Rice, LLC
Dec. 5, 2003			Austin, Texas	Trial	Asbestos	Richardson Patrick
Dec. 8, 2003	Manfred Schiller, et al.			Trial	Asbestos	Motley Rice, LLC

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Dec. 12, 2003	H. Janssen, et al. v. A.M.F., Inc., et al.	E-161739	172 nd Judicial District Court of Jefferson County, Texas	Deposition	Asbestos	Baron & Budd, P.C.

2004

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Jan. 7, 2004	H. Morgan, et al. v. Quigley Company, et al.	03-05192-1	162 nd Judicial District Court of Dallas County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Jan. 9, 2004	T. Hickox, et al. v. CSX Trans., Inc., et al.			Deposition	Asbestos	Roger Lane
Jan. 12, 2004	H.B. Beck			Deposition	Asbestos	Foster & Sear
Jan. 16, 2004	G. Russell, et al. v. Anchor Packing Company, et al.	2003-VS-044530, 20051		Deposition	Asbestos	Richardson, Patrick
Jan. 27, 2004	Linstrom, et al. v. A-C Liability Trust, et al.	1:98CV13222	Northern District of Ohio	Deposition	Asbestos	Jacques Admiralty
Jan. 21-22, 2004	Nolan, et al. v. Crane, et al.	01-L-117	Circuit Court of Vermillion County, Illinois	Trial	Asbestos	Casino Vaughan Law Office

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Jan. 29, 2004	Jones, et al. v. Meyer's Auto Parts, Inc., et al.	03-3367	District Court of Louisiana, Orleans Parish	Deposition	Asbestos	Martzell & Bickford
Feb. 4, 2004	Adams, et al. v. A.W. Chesterton, Inc., et al.	2000-32	Little Circuit	Trial	Asbestos	Motley Rice, LLC
Feb. 6, 2004	Chalmers, et al. v. Industrial Insulation, Inc., et al.	01-CV-22464 Div. 2	Circuit Court of Jackson County, Missouri		Asbestos	
Feb. 9, 2004	Bryant, et al. v. Quigley Company, et al.	03CV1877	122 nd Judicial District Court of Galveston County, Texas	Deposition	Asbestos	Baron & Budd
Feb. 9, 2004	Hernandez, et al. v. Quigley Company, et al.	26996	149 th Judicial District Court of Brazoria County, Texas	Deposition	Asbestos	Baron & Budd
Feb. 9, 2004	Brannan, et al. v. Quigley Company, et al.	03-05379-A	14 th Judicial District Court of Dallas County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Feb. 9, 2004	S. Hubbs, et al. v. Crown, Cork & Seal, et al.	18977*JG02	239 th Judicial District Court of Brazoria County, Texas	Deposition	Asbestos	Baron & Budd, P.C.

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Feb. 9, 2004	Perez, et al. v. Quigley Company, et al.	26994	239 th Judicial District Court of Brazoria County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Feb. 9, 2004	J. Qilantan, et al. v. Quigley Company, et al.	27116	149 th Judicial District Court of Brazoria County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Feb. 9, 2004	Orchard, et al. v. Quigley Company, et al.	02-0094-A	14 th Judicial District Court of Dallas County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Feb. 16, 2004	Dahlke, et al. v. Alcate, et al.			Deposition	Asbestos	Kaeske Law Firm
Feb. 16, 2004	Walker, et al. v. Bondex, et al.			Deposition	Asbestos	Kaeske Law Firm
Feb. 16, 2004	Sumbock, et al. v. GM, et al.			Deposition	Asbestos	Kaeske Law Firm
Feb. 16, 2004	Monk, et al. v. Exxon, et al.			Deposition	Asbestos	Kaeske Law Firm
Feb. 17, 2004	H. Daniels, et al. v. Pittsburgh Corning, et al.	B-150, 374	60 th Judicial District Court of Jefferson County, Texas	Deposition	Asbestos	Provost Umphrey

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Feb. 17, 2004	S. Melvin, et al. v. Mobil Oil Refining, et al.	B-150-374-AU	60 th Judicial District Court of Jefferson County, Texas	Deposition	Asbestos	Provost Umphrey
Feb. 18-19, 2004	L. Yunque, et al. v. Crown, Cork & Seal, et al.	2003-573	County Court at Law #3 of El Paso County, Texas	Trial	Asbestos	Baron & Budd, P.C.
Mar. 4, 2004	Benavides, et al. v. International Flavors and Fragrances, et al.	01-CV-68302			Asbestos	Humphrey, Farrington & McClain
Mar. 15, 2004	Asbestos Trial group		Wheeling, WV		Asbestos	Modley Rice, LLC
Mar. 18, 2004	John Crane Depo			Deposition	Asbestos	The Lanier Firm
Apr. 1, 2004	W. Strey, et al. v. Bondex, et al.	2003-CL-08297			Asbestos	Baron & Budd, P.C.
Apr. 4, 2004	Williamson, et al. v. CSX Transportation, Inc., et al.				Asbestos	Roger Lane
Apr. 4, 2004	Ellington, et al. v. Owens-Corning, et al.	30,628-97-10			Asbestos	Foster & Sear

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Apr. 19, 2004	S. Dryden, et al. v. Able Supply Company, et al.	B-171,472	60 th Judicial District Court of Jefferson County, Texas		Asbestos	Herschel Hobson
Apr. 21-22, 26, 2004	L. Altimore, et al. v. Quigley Company, et al.	03-CV-0588	405 th Judicial District Court of Galveston County, Texas	Trial	Asbestos	Heard, Robbins, Cloud, Greenwood, & Lubel, LLC Motley Rice, LLC
Apr. 24, 2004	Personal Injury Litigation Asbestos	03-C-9600			Asbestos	
May, 2004	Altimore, et al. v. Exxon Corp., et al.		Galveston County, Texas	Trial	Asbestos	Troy Chandler, Esq.
May 1, 2004	L. Eaton, et al. v. Allied Signal, et al.				Asbestos	Paul, Reich & Myers
	J. Perazzi, et al. v. Allison Transmissions, et al.				Asbestos	Paul, Reich & Myers
May 26-28, 2004	P. Chapin, et al. v. A & L Parts, Inc., et al.	03-324-775-NP	3 rd Judicial District Court of Wayne County, State of Michigan	Trial	Asbestos	Richardson, Patrick & Brickman, LLP

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
June 4, 2004	T. Langved, et al. v. Quigley Company, et al.	A-031048-C	Judicial District Court of Orange County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
June 7, 2004	Betty Pollman, et al.	01L146			Asbestos	Casino Vaughan
June 12, 2004	Ghelson, et al. v. Owens Illinois, et al.	18977*[G02			Asbestos	Baron & Budd
June 12, 2004	L. Stanford, et al. v. Dow Chemical, et al.	18977*[G02			Asbestos	Baron & Budd
June 14, 2004	S. Hubbs, et al. v. Crown, Cork & Seal, et al.	18977*[G02	239 th Judicial District Court of Brazoria County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
June 15, 2004	E. Marsden, et al. v. Upjohn	A030113C		Deposition	Asbestos	Silber Pearlman
July 19, 2004	Quigley Company, et al. v. Arrington, et al.	CL02-36195T-01	128 th Judicial District Court of Orange County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Aug. 25, 2004	Georgia Pacific Corporation, et al.		Circuit Court of Virginia for City of Newport News	Trial	Asbestos	Patten, Wornom, Harten & Diamonstein, L.C.
Sept. 20, 2004	S. Stanick, et al.	03-3480-F	214 th Judicial	Deposition	Asbestos	Baron & Budd,

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
	v. Quigley Company, et al.		District Court of Nueces County, Texas			P.C.
Oct. 4, 2004	Ferrell Ivie, et al. v. Quigley Company, et al.	2004-25996	11 th Judicial District Court of Harris County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Oct. 12, 2004	S. Tanner, et al. v. Garlock, Inc., et al.	03-06294	191 st Judicial District Court of Dallas County, Texas		Asbestos	Modley Rice, LLC
Oct. 12, 2004	G. Adams, et al. v. A.W. Chesterton Company, et al.	CIV 2000-32			Asbestos	Modley Rice, LLC
Oct. 20, 2004	O. Sather, et al. v. Metropolitan Life Insurance Company, et al.	A 030041-C	128 th Judicial District Court of Orange County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Oct. 25, 2004	E. Bishop, et al. v. Honeywell International, Inc., et al.	2004-1201	Civil District Court of Louisiana, New Orleans Parish		Asbestos	Martzell & Bickford

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Oct. 28, 2004	Dabukoski, et al. v. Garlock, et al.	03CV421	56 th Judicial District Court of Texas		Asbestos	The Lanier Firm
Nov. 1, 2004	Community Exposure of Lisa Clark			Affidavit	Asbestos	The Lanier Firm
Nov. 8, 2004	In Re: Asbestos Litigation	2004-03964	11 th Judicial District Court of Harris County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Nov. 10, 2004	C. Nassif, et al. v. Quigley Company, et al.	200329777	269 th Judicial District Court of Harris County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Nov. 17, 2004	W. Pyatt, et al.				Asbestos	Robert R. Hatten
Nov. 29, 2004	In Re: Texas MDL Asbestos		11 th Judicial District Court of Harris County, Texas	Hearing	Asbestos	Multiple firms
Dec. 16, 2004	D. Root, et al. v. Quigley Company, et al.	A-31045-C	128 th Judicial District Court of Orange County, Texas	Deposition	Asbestos	Baron & Budd, P.C.

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Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Jan. 14, 2005	T. Durham, et al. v. A.W. Chesterton, et al.	032231	Superior Court of Rhode Island, Providence	Deposition	Asbestos	Motley Rice, LLC
Feb. 4, 2005	J. Tedrow, et al. v. Quigley Company, et al.	03-06626-D	95 th Judicial District Court of Dallas County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Feb. 9, 2005	Mary Leech, et al. v. A.W. Chesterton, et al.	CV495702	Court of Common Pleas of Ohio, Cuyahoga County	Deposition	Asbestos	Baron & Budd, P.C.
Feb. 16, 2005	L. Berry, et al. v. Quigley Company, et al.	DV02-10802-A	14 th Judicial District Court of Dallas County, Texas	Deposition	Asbestos	Silber Pearlman
Feb. 18, 2005	D. Fowers, et al. v. 3M Company, et al.	BC318781	Superior Court of California, County of Los Angeles	Deposition	Asbestos	Baron & Budd, P.C.
Feb. 24-28, 2005	T. Bostic, et al. v. Georgia Pacific Corporation, et al.	CC-03-01977-C	County Court at Law #3 of Dallas County, Texas	Trial	Asbestos	Baron & Budd, P.C.

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Mar. 1, 2005	L. Vaughn, et al. v. 3M Company, et al.	BC322230	Superior Court of California, County of Los Angeles	Deposition	Asbestos	Baron & Budd, P.C.
Mar. 9, 2005	L. Vaughn, et al. v. 3M Company, et al.	BC322230	Superior Court of California, County of Los Angeles	Deposition	Asbestos	Baron & Budd, P.C.
Mar. 14, 2005	O. LaClair, et al. v. Quigley Company, et al.	2004-11055	11 th Judicial District of Harris County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Mar. 22-23, 2005	D. Fowers, et al. v. 3M Company, et al.	BC318781	Superior Court of California, County of Los Angeles	Trial	Asbestos	Baron & Budd, P.C.
Apr. 18, 2005	O. Adams, et al. v. Quigley, et al.	2003-29774	269 th Judicial District Court of Harris County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Apr. 20, 2005	A. Watts, et al. v. Georgia Pacific, Inc., et al.	55,941	18 th Judicial District of State of Louisiana, Iberville Parish	Trial	Asbestos	Baron & Budd, P.C.
May 11, 2005	O. Withers, et al. v. GAF Corp., et al.	12902*BH00	23 rd Judicial District Court of Brazoria County, Texas	Deposition	Asbestos	Baron & Budd, P.C.

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
June 1, 2005	Joe James, et al. v. Quigley Company, et al.	03-05383-L	193 rd Judicial District Court of Dallas County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
June 3, 2005	In Re: Asbestos Litigation	2004-03964	11 th Judicial District Court of Harris County, Texas	Deposition	Asbestos	Multiple firms
June 7-8, 2005	In Re: Asbestos Litigation	2004-03963	11 th Judicial District Court of Harris County, Texas	Deposition	Asbestos	Multiple firms
June 14, 2005	Josephat Henry, et al. v. St. Croix Alumina, et al.	1999/0036	District Court of the Virgin Islands, Division of St. Croix	Deposition	Asbestos	Baron & Budd, P.C.
June 23, 2005	J. Brundrett, et al. v. Crown, Cork & Seal, et al.	01-CV-210	County Court at Law of Calhoun County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Aug 11, 2005	M. Evans, et al. v. A.W. Chesterston, et al.	04-L-453	Circuit Court of Madison County, Illinois, 3 rd Judicial Circuit	Deposition	Asbestos	Baron & Budd, P.C.
Aug 15, 2005	Gagneaux, et al. v. Union Pacific Railroad Company	97-3678	Civil District Court of the Parish of Orleans, State of Louisiana	Deposition	Asbestos	LeBlanc & Waddell

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Sept. 1, 2005	Ingraham, et al. v. Gatlock, et al.	GN102642	126 th Judicial District Court of Travis County, Texas	Trial	Asbestos	Richardson, Patrick, Westbrook & Brickman, LLC
Sept. 14, 2005	J. Brundrett, et al. v. Crown, Cork & Seal, et al.	01-CV-210	County Court at Law of Calhoun County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Sept. 28, 2005	J. Ham, et al. v. Quigley Company, et al.	2003-31186	152 nd Judicial District Court of Harris County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Oct. 18-19, 2005	In Re: Asbestos Litigation	77C-ASB-2	Superior Court of State of Delaware, New Castle County	Hearing	Asbestos	Multiple firms
Oct. 24, 2005	S. Stanick, et al. v. Quigley Company, et al.	03-3480-F	214 th Judicial District Court of Nueces County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Oct. 25, 2005	R. Beamon, et al. v. Quigley Company, et al.	04-11678-J	191 st Judicial District Court of Dallas County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Nov. 3, 2005	J. Couturier, et al. v. Georgia Pacific Corp., et al.	524,866	19 th Judicial District Court of Louisiana, Parish of East Baton Rouge	Deposition	Asbestos	LeBlanc & Waddell

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Dec. 16, 2005	In Re: Pflbrico Company	02-B-09952	U.S. Bankruptcy Court of the Northern District of Illinois, Eastern Division	Deposition	Asbestos	Nemeroff Law Firm

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Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Jan. 19, 2006	P. Palmer, et al. v. Georgia Pacific Corporation, et al.	61900	18 th Judicial District of State of Louisiana, Iberville Parish	Deposition	Asbestos	LeBlanc & Waddell
Jan. 31, 2006	Rittenberry, et al. v. Guard-Line, Inc., et al.	03-H-0362-C	23 rd Judicial District Court of Matagorda County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Feb. 16 & 21, 2006	O'Connell, et al. v. Bondex International, Inc., et al.	04-L-0676	Circuit Court of Madison County, Illinois, 3 rd Judicial Circuit	Trial	Asbestos	Baron & Budd, P.C.
Feb. 24, 2006	K. Haynes, et al. v. Georgia Pacific Corp., et al.	052-00845	Missouri Circuit Court, 22 nd Judicial Court (City of St. Louis)	Deposition	Asbestos	Baron & Budd, P.C.
Mar. 8, 2006	J. Lang, et al. v. Eagle, Inc. et al.	05-8042	Civil District Court of Louisiana, Orleans Parish	Deposition	Asbestos	LeBlanc & Waddell

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
May 18 & 22-23, 2006	T. Bostic, et al. v. Georgia Pacific Corporation, et al.	CC-03-01977-C	County Court at Law #3 of Dallas County, Texas	Trial	Asbestos	Baron & Budd, P.C.
June 5, 2006	B. Spillman, et al. v. Anco Insulation, et al.	536,903	19 th Judicial District Court of Louisiana, Parish of East Baton Rouge	Deposition	Asbestos	LeBlanc & Waddell
July 11, 2006	J. Norris, et al. v. Ampco-Pittsburgh, et al.	BC340413	Superior Court of California County of Los Angeles	Deposition	Asbestos	Baron & Budd, P.C.
July 24-25, 2006	McNamara, et al. v. BWIP, et al.	BC342803	Superior Court of California County of Los Angeles	Deposition	Asbestos	Waters & Kraus, LLP
Aug 1, 2006	Fake Trial Group	05C-07-135	Superior Court of Delaware New Castle County	Deposition	Asbestos	Baron & Budd, P.C.
Aug 7, 2006	C. Wells, et al. v. Georgia Pacific Corp., et al.	536,867	Judicial District Court of Louisiana, Parish of East Baton Rouge	Deposition	Asbestos	LeBlanc & Waddell
Aug 21, 2006	J. Norris, et al. v. Ampco-	BC340413	Superior Court of California County of Los	Trial	Asbestos	Baron & Budd, P.C.

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
	Pittsburgh, et al.		Angeles			
Aug. 30, 2006	Campbell, et al. v. A.W. Chesterton, et al.	052-01551	Circuit Court of State of Missouri, 22 nd Judicial Circuit	Deposition	Asbestos	Baron & Budd, P.C.
Oct. 5, 2006	R. Fortini, et al. v. 3M Company, et al.	BC334407	Superior Court of California County of Los Angeles	Deposition	Asbestos	Baron & Budd, P.C.
Oct. 11, 2006	Blandford, et al. v. A.W. Chesterton, et al.	437948	Court of Common Pleas of Ohio Cuyahoga County	Deposition	Asbestos	Baron & Budd, P.C.
Nov. 8, 2006	S. Delisle, et al. v. 3M Company, et al.	BC334402	Superior Court of California County of Los Angeles	Deposition	Asbestos	Baron & Budd, P.C.
Dec. 5-6, 2006	R. Fortini, et al. v. 3M Company, et al.	BC334407	Superior Court of California County of Los Angeles	Trial	Asbestos	Baron & Budd, P.C.
Dec. 13, 2006	Norris, et al. v. Foster Wheeler Corporation, et al.	05CV009475	Circuit Court of the State of Wisconsin, Milwaukee County	Trial	Asbestos	Penn Rakauski
Dec. 18, 2006	D. Jackson, et al. v.	BC332382	Superior Court of California	Deposition	Asbestos	Baron & Budd, P.C.

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Dec. 19 2006	3M Company, et al. v. Ray Rando, et al. Anco Insulations, Inc.	538,254	County of Los Angeles 19 th Judicial District, Parish of East Baton Rouge, State of Louisiana	Deposition	Asbestos	LeBlanc & Waddell, LLP

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Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Jan. 17, 2007	J.H. Martin, et al. v. Quigley Company, et al.	2004-19730	11 th Judicial District Court of Harris County, Texas	Deposition	Asbestos	Baron & Budd, P.C.
Jan. 22, 2007	In Re: Asbestos Litigation Cote Trial Group	Multiple Case Numbers	Superior Court of Delaware New Castle County	Deposition	Asbestos	Baron & Budd, P.C.
Jan. 26, 2007	Chambers, et al. v. Ampco- Pittsburgh Corporation, et al.	BC328067	Superior Court of California County of Los Angeles	Deposition	Asbestos	Baron & Budd, P.C.
Jan. 29, 2007	John Perez, et al. v. 3M Company, et al.	BC354964	Superior Court of California County of Los Angeles	Deposition	Asbestos	Baron & Budd, P.C.
Feb. 2, 2007	S. Oney, et al. v. Garlock Sealing Technologies, et al.	00301TF	Circuit Court of Virginia City of Newport News	Deposition	Asbestos	Patten, Wornom, Hatten & Diamonstein, LC
Feb. 13 & 15-16, 2007	In Re: Asbestos Litigation - Cote Trial Group	Multiple Case Numbers	Superior Court of Delaware New Castle	Trial	Asbestos	Baron & Budd, P.C.

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Feb. 21, 2007	Beauchamp, et al. v. Allis Chalmers Corp., et al.	BC357289	County Superior Court of California County of Los Angeles	Deposition	Asbestos	Baron & Budd, P.C.
Mar. 6, 2007	D. Jackson, et al. v. 3M Company, et al.	BC332382	County Superior Court of California County of Los Angeles	Trial	Asbestos	Baron & Budd, P.C.
Mar. 22, 2007	Beauchamp, et al. v. Allis Chalmers Corp., et al.	BC357289	County Superior Court of California County of Los Angeles	Trial	Asbestos	Baron & Budd, P.C.
Mar. 28, 2007	Sungter			Deposition	Asbestos	Aaron Deluca
Mar. 29, 2007	Piazza, et al.			Deposition	Asbestos	Aaron Deluca
Mar. 30, 2007	Advocate Mines Limited, et al.	450687	County Superior Court of California County of San Francisco	Deposition	Asbestos	Baron & Budd, P.C.
Apr. 3, 2007			Newport News	Trial	Asbestos	Patten, Wormom, Hatten & Diamonstein, LLP
Apr. 6, 2007	W.R. Grace property damage case			Deposition		Motley Rice
Apr. 11, 2007	Piazza, et al. v. Advocate Mines Limited, et al.	450687	County Superior Court of California County of San Francisco	Deposition	Asbestos	Baron & Budd, P.C.

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
Apr. 23, 2007	Mulkarnicek			Deposition	Asbestos	Silber Pearlman
Apr. 24, 2007	Piazza, et al. v. Advocate Mines Limited, et al.	450687	Superior Court of California County of San Francisco	Deposition	Asbestos	Baron & Budd, P.C.
May 9, 2007			Court of Common Pleas of Ohio, Cuyahoga County	Trial	Asbestos	Motley Rice
May 10, 2007	Robert Dudoit, et al.			Deposition	Asbestos	Baron & Budd, P.C.
May 18, 2007	Richard Belt, et al.			Deposition	Asbestos	Simon, Eddins & Greenstone
May 11, 2007	Milton Knutson, et al.			Deposition	Asbestos	Baron & Budd, P.C.
May 16 & 22, 2007	Edwards, et al. v. Quigley Company, et al.	2003-58354	165 th Judicial District Court of Harris County, Texas	Trial	Asbestos	Baron & Budd, P.C.
May 30, 2007	Shake, et al. v. Quigley Company, et al.	2004-21092	11 th Judicial District Court of Harris County, Texas	Deposition	Asbestos	Silber Pearlman
June 14, 2007	Richard Belt, et al.			Trial	Asbestos	Simon, Eddins & Greenstone
July 6, 2007	G. Hoover, et al. v. CSR, America, et al.	572500	Court of Common Pleas of Ohio Cuyahoga County	Deposition	Asbestos	Robert Sweeney Co., LPA
July 10, 2007	R Walton, et al. v.	05-L-94	Circuit Court of Madison County,	Deposition	Asbestos	Motley Rice

Date	Case	Cause/Case No.	Court/Jurisdiction	Deposition/Trial	Type of Case	Firm
July 12, 2007	Norfolk Southern Railway, et al. v. J. Meyers, et al. v. Pittsburgh Corning		Illinois 6 th Judicial District District Court of Jefferson County, Texas	Trial	Asbestos	Provost Umphrey
July 13, 2007	J. Monihan, et al. v. Consolidated Railway, et al.	552314	Court of Common Pleas of Ohio, Cuyahoga County	Deposition	Asbestos	Baron & Budd, P.C.
July 16, 2007	Dachauer, et al. v. Amcord, et al.	06-454837	Superior Court of California Los Angeles	Deposition	Asbestos	Baron & Budd, P.C.
July 19, 2007	Currole, et al. v. The McCarty Corp., et al.	546,905	19 th Judicial District Court of Louisiana East Baton Rouge Parish	Deposition	Asbestos	LeBlanc & Waddell
July 26, 2007	D. Taylor, et al.			Deposition	Asbestos	Bergman & Frocht
July 27, 2007	R. Belt, et al.			Deposition	Asbestos	Simon, Eddins & Greenstone

Senator BOXER. Thank you, and we will have some questions.

Ms. Linda Reinstein. We welcome you and we are so sorry for your loss.

**STATEMENT OF LINDA REINSTEIN, EXECUTIVE DIRECTOR
AND CO-FOUNDER, ASBESTOS DISEASE AWARENESS ORGA-
NIZATION**

Ms. REINSTEIN. Thank you, Madam Chairman Boxer and Ranking Member Inhofe and the entire EPW Committee for the honor and opportunity to testify today.

My name is Linda Reinstein. I am the executive director of the Asbestos Disease Awareness Organization, ADAO, and now a mesothelioma widow. My husband, Alan Reinstein, lost his 3-year battle with mesothelioma, a deadly asbestos cancer, in May 2006. I am neither a lobbyist nor an attorney, just a volunteer.

Today, I somberly represent the victims and their families who have suffered the traumatic effects of asbestos diseases. For many of us, this is an especially difficult week, as Fathers Day is on Sunday.

Hundreds of thousands of asbestos victims around the world pay the ultimate price for asbestos exposure: their lives. These diseases are all preventable. Before I share the faces, it is important to understand the facts. We cannot alter history or bring back the dead, but we can learn from the past to save lives and money.

Most Americans trust that their air, soil and water are safe from toxic contaminants. But as victims, we know the truth. For a century, asbestos exposure has been linked to incurable diseases. Yet we continue to face an enormous man-made public health crisis. Just walk the streets of Libby or New York City, or talk to the U.S. Capitol tunnel workers, here today. They all know too well about the irreversible effects of asbestos poisoning.

The stress and trauma is life-altering for those Americans with known exposure, waiting for time to reveal their medical fate. The IARC declared asbestos as a human carcinogen nearly 30 years ago. The EPA, WHO, IRO agree. There is no safe level of asbestos exposure. The simple truth: asbestos kills.

The penny slide on the easel compares the nearly invisible deadly fibers just under President Lincoln's nose to grains of rice and human hair. These virtually indestructible fibers are 700 times smaller than human hair and can remain suspended in the air from seconds to days. Asbestos is an equal opportunity killer. Its dust doesn't discriminate. Inhaling or swallowing the fiber can cause malignant or non-malignant diseases.

Asbestos diseases are difficult to diagnose and treat. The evolution from exposure to death can take 10 to 50 years. Children are even more susceptible to carcinogens. It is important to focus on all asbestos-caused diseases, not just mesothelioma. The Samia 7-year study on the board shows 65 percent of the victims suffered from asbestos-caused cancers and the remaining 35 percent from asbestosis. Lung cancer and mesothelioma accounted for 25 and 11 percent, respectively, of all asbestos diseases.

Although asbestos safety measures have been in place since the 1970s, exposure continues. The CDC reports an increase in asbes-

tosis deaths from 1968 to 2000. These and other related diseases are not going away, only the victims who die.

Victims suffering from pulmonary diseases and cancer feel like they are breathing through a pinched straw every breath, every minute, every day. When victims' oxygen levels become critically low, they are tethered to supplemental oxygen to prolong life, like my husband. Lack of oxygen can cause death by respiratory failure or cardiac arrest.

To prolong a victim's life, many cancer patients opt for radical treatments, such as having their entire diseased lung and diaphragm surgically removed. We call this death by a thousand cuts. Victims living with these painful, aggressive and hopelessly incurable diseases sometimes commit suicide or ask their spouses to commit mercy killings.

Mesothelioma patients' medical expenses can exceed a million dollars before death. The physical and financial devastation is immeasurable to victims and their families. Each time a patient dies, a shattered family is left behind. The new patient profile is now a 51-year-old woman. Younger victims are dying. There is a 16-year-old girl newly diagnosed in New York. Federal surveillance in the United States under-report.

So what is a human life worth? Certainly banning asbestos and investing in safe alternatives. Without an asbestos ban, death and litigation will continue. To profit over people is unconscionable. It is time to eliminate asbestos exposure and invest in research to improve treatment.

We applaud Senator Patty Murray for the Ban Asbestos Act. An immediate worldwide ban on the production and use of asbestos is long overdue, fully justified and absolutely necessary.

Support for my testimony comes from some of the most well-respected members of the science community and an outpouring from victims around the world. I have included a list of these endorsements in my written testimony. Thank you.

[The prepared statement of Ms. Reinstein follows:]

STATEMENT OF LINDA REINSTEIN, EXECUTIVE DIRECTOR, CO-FOUNDER AND
MESOTHELIOMA WIDOW

I would like to thank Chairman Boxer, Ranking Member Inhofe and the entire EPW Committee for the honor and opportunity to testify today.

My name is Linda Reinstein, Executive Director of the Asbestos Disease Awareness Organization (ADAO) and now a mesothelioma widow. My husband, Alan Reinstein, lost his three year battle with mesothelioma, a deadly asbestos cancer, on May 2006. I am neither a lobbyist nor an attorney, only a volunteer.

Today I somberly represent the victims and their families who have suffered the traumatic effects of asbestos diseases. Hundreds of thousands of asbestos victims around the world paid the ultimate price for asbestos exposure: their lives. These diseases were preventable.

Before I share the faces, it is important to understand the facts. We can not alter history or bring back the dead, but we can learn from the past to save lives and money.

Most Americans trust that their air, soil and water are safe from toxic contaminants—but as victims, we know the truth. For a century, asbestos exposure had been linked to incurable diseases, yet we continue to face an enormous man-made public health crisis. Just walk the streets of Libby or New York City or talk to the U.S. Capitol Tunnel Workers—they also know all too well about the irreversible effects of asbestos poisoning. The stress and trauma is life altering for those Americans with known asbestos exposure waiting for time to reveal their medical fate. The International Agency for Research on Cancer (IARC) declared asbestos as a human carcinogen thirty years ago. The Environmental Protection Agency, (EPA)

World Health Organization (WHO) and the International Labor Organization (ILO) agree—there is no safe level of asbestos exposure. The simple truth is—asbestos kills.

The Penny slide compares the nearly invisible deadly fibers just under President Lincoln's nose to grains of rice and human hair. Once known as the "The Magic Mineral", these virtually indestructible asbestos fibers can be 700 times smaller than human hair and remain suspended in air from seconds to days.

Although asbestos safety measures have been in place since the 1970s, The Center for Disease Control (CDC) reported that deaths from asbestosis, a debilitating lung disease, increased from 77 deaths in 1968 to 1,493 deaths in 2000. These and other asbestos-related diseases are not going away, only the victims who die.

The World Health Organization estimates 125 million workers are exposed to asbestos and 90,000 workers die annually. There is no global data estimating deaths from non-occupational or environmental exposure.

Asbestos is an equal opportunity killer and the dust does not discriminate. Inhaling or swallowing asbestos fibers can cause malignant and nonmalignant diseases.

Asbestos diseases are difficult to diagnose and treat. Evolution of disease, from exposure, screening, surveillance, detection, treatment and death can take 10–50 years. Children are even more susceptible to carcinogens and have a shorter latency period.

It is important to focus on all asbestos-caused diseases, not just mesothelioma. The Sarnia seven year study sited: 65 percent of the victims suffered from asbestos-caused cancers and the remaining 35 percent suffered from asbestosis. Lung cancer and mesothelioma accounted for 25 percent and 11 percent respectively of asbestos diseases.

Asbestosis is the scarring of lung tissue resulting only from the inhalation of asbestos fibers which reduces oxygen transfer to the blood as well as the removal of carbon dioxide. Asbestosis is a painful, progressive and incurable lung disease with no effective treatment.

Victims suffering from pulmonary diseases and cancer feel like they are breathing through a pinched straw, for every breath, every minute, every day. When the victims' oxygen levels become critically low, they are tethered to supplemental oxygen to prolong life. Lack of oxygen causes death by respiratory failure and/or cardiac arrest.

To prolong a victim's life, many cancer patients opt for radical treatments such as having their entire diseased lung and diaphragm surgically removed. We call this, death by 1,000 cuts. Victims living with these painful, aggressive and hopelessly incurable diseases sometimes commit suicide or ask spouses to commit mercy killings.

Mesothelioma patients' medical expenses can exceed \$1 million—until death.

MEDICAL EXPENSES INCLUDE:

- Tri-modal Cancer Treatment, Surgery, Radiation Chemotherapy
- Medication & Oxygen
- Home Health/Hospice
- Psychiatry
- Interstate Travel to Medical Surgery Centers

The physical, financial and physiological devastation are immeasurable to the victims and their families. After the patient dies, financial and psychological problems continue to plague the family. For each life lost, a shattered family is left behind.

Psychological issues are tormenting—as victims hold hands with death.

- Constantly facing death is debilitating for both the patient and family.
- Constantly facing death is debilitating for both the patient and family.
- Caregivers face both mental and physical exhaustion—Alan needed 24-hour care for 12 months.
- Depression is a common factor.

Physical pain and treatments are brutal; the prognosis is grim

- Screening, surveillance, and detection are exhausting and remain a constant reminder of exposure and possible terminal diseases.
- Late stage diagnosis is common, as many victims are asymptomatic.
- Victim has baffling and radical treatment options to navigate.
- Dangerous surgeries and toxic medicine—if the diseases don't end your life, the treatments may.

Financial issues devour assets and threaten financial stability

- Expensive medical treatments. One month of Alan's prescriptions, oxygen and chemotherapy averaged \$104,000.

- Lost jobs or reduced income results when victims are too ill to work.
- Health benefits are also terminated as a result of losing a job.

Constant fear and extreme isolation magnifies these three factors.

In 1990, the average patient was a male, age 70. Our data shows the new patient profile to be a 51 year old woman. Younger victims are dying from diseases. Recently, a 16 year-old girl was diagnosed with mesothelioma. Patients diagnosed with asbestos-caused diseases are completely innocent. They are firefighters and veterans, construction workers and engineers. They are the women who became exposed washing their husbands' work clothes. They are children whose loving hug turned deadly.

Surveillance in the U.S. continues to under report asbestos-related disease. Without disease registries, effective outreach and well implemented surveillance programs; we can not accurately forecast the magnitude of disease.

DEADLY CONTAMINATION CONTINUES TODAY

Asbestos was widely used in the construction and attic insulation in millions of homes in the U.S. and Canada built before 1975. More than 30 million homes, schools and office building are still contaminated with deadly asbestos.

In 2002, the collapse of the World Trade Center towers led to the release of hundreds of tons of asbestos from the towers. An estimated 20,000 responders, workers, volunteers and residents suffer from respiratory diseases. The annual direct treatment costs are \$140 million dollars. We can only extrapolate the cost of human tragedy and treatment expenses from the Hurricane Katrina disaster one of whose consequences is poorly controlled asbestos exposure in the buildings being repaired and demolished. The World Health Organization has started an asbestos action program to help countries all over the world develop national plans, based on the conclusion that "the most efficient way to eliminate asbestos-related diseases is to stop using all types of asbestos."

Asbestos continues to be mined and exported from Canada. The United States and Canada remain the only two industrialized nations that have not yet banned the use of asbestos in common products while more than 40 countries have banned asbestos. Consumers are at risk with imported products contaminated with asbestos such as brakes and asbestos-cement building panels. The asbestos ban will only be as effective as the laws that are enforced. Presently, minimal fines and lack of enforcement make our existing laws weak and deadly asbestos exposure continues.


What is a human life worth? Certainly banning asbestos and investing in safe alternatives. Without an asbestos ban, deaths and litigation will continue. To profit over people is unconscionable. It is time to eliminate asbestos exposure, while simultaneously investing in research for a cure and improved treatments. An immediate worldwide ban on the production and use of asbestos is long overdue, fully justified and absolutely necessary. We applaud Senator Patty Murray for the Ban Asbestos in America Act, and hope that this is only the start of a ban across the globe.

Support for this critical issue comes from some of the most well respected members of the scientific and medical community from around the world. I have included a list of these endorsers in my written testimony.

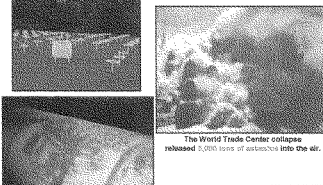
Thank you.

"Asbestos Epidemic in the U.S."


Linda Reinstein
 Asbestos Disease Awareness Organization
 Executive Director and Co-Founder
 Mesothelioma Widow
 Linda@AsbestosDiseaseAwareness.org

Reinstein - Senate EPW Hearing 

From Libby to the U.S. Capitol Tunnels to WTC




The World Trade Center collapses released 2,000 tons of asbestos into the air.

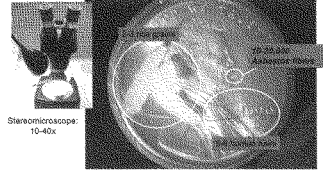
Reinstein - Senate EPW Hearing 

ASBESTOS
"The Magic Mineral"


- Asbestos fibers can be 700 times smaller than human hair.
- Asbestos fibers can remain suspended into air from seconds to days.
- Asbestos fibers are virtually indestructible.

Reinstein - Senate EPW Hearing 

How small is asbestos?




Stereomicroscope: 10-40x

Reinstein - Senate EPW Hearing 

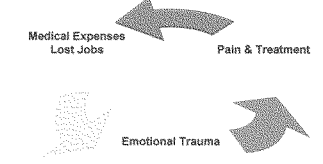
World Health Organization Estimates

125 million workers are exposed to asbestos

90,000 workers die annually

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Devastating Issues
 Physical, Financial and Psychological



Medical Expenses
 Lost Jobs

Pain & Treatment

Emotional Trauma

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Linda Reinstein
ADAO Executive Director and Cofounder

Asbestos Disease Awareness Organization
www.AsbestosDiseaseAwareness.org

Occupational Exposure

- Contaminated Buildings
 - Abatement, Renovation and Repairs
 - U.S. Capitol Tunnel Workers
- Construction
 - Carpenters, Metal workers, Electricians and Plumbers
- Automotive
- Boilermakers
- Department of Defense
- School Teachers

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Non-occupational Exposure

- Consumer Products
- Take-home
- Renovations, Implosions and Demolitions
- Environmental
 - Libby, MT - Mined
 - El Dorado Hills, CA - Naturally Occurring Asbestos
 - W. R. Grace Hotspots - 40 sites around the Nation
 - Ambler, Pennsylvania - Former Factory
- Disasters
 - New York City World Trade Center (9/11)
 - Hurricane Katrina
- **UNKNOWN**

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Inaccurate Data

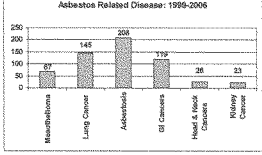
Impossible Forecasting

- Difficulty diagnosing diseases
- Primary focus on occupational exposure
- Surveillance under reports asbestos-related disease
- Miscoding of death certificates

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Sarnia OHCOW Clinic, Canada
Asbestos-related Disease Cases: 1999 - 2006
Cancers accounts for 65% of all diseases

Asbestos Related Disease: 1999-2006





Disease Category	Number of Cases
Mesothelioma	87
Lung Cancer	145
Asbestosis	208
GI Cancers	139
Heart & Blood Vessels	28
Pulmonary Fibrosis	33

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Alan Reinstein - Mesothelioma Victim

Just one year, from May 2005 - May 2006

1938 - 2005

2007 Asbestos Victim Profile

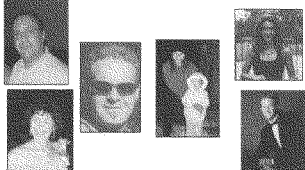
- Median Age 51 at Diagnosis/Death
- ADAO estimates 50% of victims are women
- Increasing number of victims under 40
- Many victims are asymptomatic, until late stages
- 40% of the victims have non-occupational exposure


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Linda Reinstein
ADAO Executive Director and Cofounder

Asbestos Disease Awareness Organization
www.AsbestosDiseaseAwareness.org


The New Younger Victims




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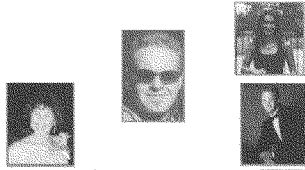
The New Generation


- Paul, age 56, Peritoneal Mesothelioma
- Eva, age 58, Pleural Mesothelioma
- Todd, age 39, Pleural Mesothelioma
- Julie, age 35, Peritoneal Mesothelioma
- Adam, age 33, Pleural Mesothelioma
- Elizabeth, age 23, Pleural Mesothelioma



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Asbestos: Equal Opportunity Killer




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*A face is worth 1000 words
We Will Never Forget
Those We Have Lost*

**For every life lost,
a shatter family is left behind.**

Reinstein - Senate EPW Hearing 

Asbestos Kills

**Prevent exposure
Eliminate diseases**

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Linda Reinstein
ADAO Executive Director and Cofounder

Asbestos Disease Awareness Organization
www.AsbestosDiseaseAwareness.org

In 72 hours, there was support from doctors, scientist, medical institutions and victims around the world.

The signatures below support these four points and wish to have their names entered into the Congressional Record.

1. **Ban Asbestos**
2. **No safe level of asbestos**
3. **Invest in research for a cure and improve treatments**
4. **Education to prevent exposure**

U.S. Capitol Tunnel Shop Workers

John Thayer
Scott Smith
Tom Baker
Martin Blanchet
Ed Hill
Frank Binns
Charlie Morris
Tim Taylor
Christian Raley
Rick Leanord

Jordan Zevon
Los Angeles, CA

International Mesothelioma Interest Group
Dr. Hedy Kindler, President, IMIG

Arthur L. Frank, MD,PhD,
Professor of Public Health and Chair, Department of Environmental and Occupational Health
Drexel School of Public Health
Co-Chair-ADAO Scientific Advisory Board.

Michael R. Harbut, MD, MPH, FCCP
Wayne State University
Chief, Center for Occupational and Environmental Medicine
118 N. Washington
Royal Oak, Michigan 48067-1751

Dr. Hedy Kindler
University Of Chicago
5841 S. Maryland Avenue, Mc 2115
Chicago, Il., 60637-1470

Dr. Robert Taub
Herbert Irving Comprehensive Cancer Center
161 Fort Washington Ave.
Suite 922
New York, NY 10032

Daniel H. Serman, M.D.
Associate Professor of Medicine
Associate Professor of Medicine in Surgery
Director, Interventional Pulmonology Program
Clinical Director, Thoracic Oncology Gene Therapy Program

Pulmonary, Allergy, & Critical Care Division
University of Pennsylvania Medical Center

Brad Black, M.D.
Medical Director
Center for Asbestos Related Disease
214 E. 3rd Street
Libby, Montana

Lincoln County Asbestos Victims Relief Organization
Gayla Benefield
245 Cedar Meadow Road
Libby, Montana

Steven Markowitz MD
Queens College
City University of New York

Dr. Clark Fuller
Cedars Sinai Medical Center
8635 W. Third Street
Suite 975-W
Los Angeles, CA 90048

Laurie Kazan-Allen
International Ban Asbestos Secretariat (IBAS)

Bill Ravanese MA, MPH
Health Care Without Harm
19 Pleasantview Ave.
Longmeadow, MA 01106

Scientific Analytical Institute
Sean Fitzpatrick
302-L Pomona Drive
Greensboro, NC 27407

Paul Zygielbaum
5916 Yerba Buena Rd.
Santa Rosa, CA

Andrew F. Oberta, MPH, CIH
107 Route 620 South
Austin TX 78734

Michael Bowker, Author
Fatal Deception

The Safety and Health Training Center
Ray Turpin, Executive Director
Adjunct Professor, University of S. Florida
OSHA Training Institute Education Center
2495 Main Street, Suite 118,
Buffalo, NY 14214

Rachel Lidov

Co-Coordinator
9/11 ENVIRONMENTAL ACTION
P O Box 250192 Columbia University Station
New York, NY 10025

White Lung Asbestos Information Center-WLAIC
Barbara Zeluck
New York, NY

Asbestos Victims Support Groups' Forum
Windrush Millennium Centre
Tony Whitston
Forum Chair
70 Alexandra Road
Manchester
M16 7WD UK

Helen Clayson
Sheffield, UK

Southeast Asbestos Awareness and Victims Support Group
Pauline Bonney
East Sussex.BN23 8DU. England

Save Spodden Valley campaign
Jason Addy
185 Rooley Moor Rd, Rochdale
United Kingdom

Sheffield And Rotherham Asbestos Group
Paula Walker
Aizlewood's Mill, Nursery Street
Sheffield, South Yorkshire, UK

Derbyshire Asbestos Support Team
Joanne Carlin
70, Saltergate
Chesterfield, S40 1JR

Ban Asbestos Network of India (BANI)
Gopal Krishna
Occupational & Environmental Health Network of India (OEHNI)

ABREA - Brazilian Association of Asbestos Exposed People from Rio de Janeiro e Sao Paulo

ABEA- Asbestos Exposed People from Bahia

Virtual-Citizen Network for the Ban of Asbestos in Latin America

Fernanda Giannasi, Civil and Safety Engineer, Labour Inspector
Labour Department in São Paulo State

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Charles R Archer	P O Box 348	Ragland	AK	35131
Karen Moss	3400 Snow Goose Dr.	Wasilla	AK	99654
Kenny Moss	3400 Snow Goose Dr.	Wasilla	AK	99654
Melissa G. Rose	1551 Oakwood Drive	Brookwood	AL	35444
Betty Kincaid	6 Ridge Drive	Pelham	AL	35124
Ronnie Kincaid	6 Ridge Drive	Pelham	AL	35124
June Jensen	28 Ciclamor Way	Hot Springs Village	AR	71909
Eileen Day	1 Browning Place Seven Hills 2147	Sydney	Australia	
Cindy Bundrock	1339 W. Boston Street	Chandler	AZ	
Lois Schwarting	201 E Ivanhoe	Gilbert	AZ	85295
Marty Schwarting	201 E Ivanhoe	Gilbert	AZ	85295
Lois Schwarting	201 E Ivanhoe	Gilbert	AZ	85295
Greg Lee	40104 W. Marylou Drive	Maricopa	AZ	85239
Lisa Lee	40104 W. Marylou Drive	Maricopa	AZ	85239
Debra J Farnsworth	10907 E. Acoma Drive	Scottsdale	AZ	85255
Rita McGuire	21305 N. 107th Drive	Sun City	AZ	85373
Erica Bender	1164 Euclid Ave	Berkeley	CA	94708
Daniel Horodysky	2213 Acton Street	Berkeley	CA	94702
Tom Condit	2217 1/2 McGee Ave	Berkeley	CA	94703
Natalie Rosenberg	320 Lemon Drive	Camarillo	CA	93010
Laura Earhart	4091 Vincente Ave	Camarillo	CA	93010
Arlene O'Connor	24697 Gleneagles Drive	Corona	CA	92883
Rhio Oconnor	24697 Gleneagles Drive	Corona	CA	92883
Arlene Oconnor	24697 Gleneagles Drive	Corona	CA	92883
Rhio O'Connor	25697 Gleneagles Drive	Corona	CA	92883
Burke McCarthy	912 Powell Court	Costa Mesa	CA	02626
F. Burke McCarthy	912 Powell Court	Costa Mesa	CA	92626
Laurie Pollan	33243 Ocean Ridge	Dana Point	CA	92629
Eric Johnson	5343 Tanaya	Fresno	CA	93722
Susan E. Burke	25761 Le Parc #104	Lake Forest	CA	92630
Bert Mintz	5657 Wilshire Blvd Suite 310	Los Angeles	CA	90036
Hanne Mintz	5657 Wilshire Blvd Suite 310	Los Angeles	CA	90036
Marina Mintz	5657 Wilshire Blvd Suite 310	Los Angeles	CA	90036
Amanda Urnberg	90 F Street	Martinez	CA	94553
Julia McCarthy	2827 Catalpa Street	Newport Beach	CA	92660
Russell Beemer	2827 Catalpa Street	Newport Beach	CA	92660
Joseph Mennealy	11242 Lakeland Road	Norwak	CA	90650
Terri Gutai	438 Vernon Street	Oakland	CA	94601
Tony Vickers	1336 Via Romero	Palos Verdes Estates	CA	90274
Kelly Casserly	480 Portafino Ct., #301	Phillips Ranch	CA	91766
Jessica Like	8500 Pershing Drive #208	Playa del Rey	CA	90293
Jen Tanguileg	71 Viking Drive	Pleasant Hill	CA	94523
Robert Tanguileg	71 Viking Drive	Pleasant Hill	CA	94523
Ellen Tunkelrott	1613 Stanford Ave	Redondo Beach	CA	90278
Vickie Castaldi	234 N. Juanita Ave.	Redondo Beach	CA	90277
Kevin Akin	20212 Harvard Way	Riverside	CA	92507
Trisa Zembron Endicott	1015 Tanzania Drive	Roseville	CA	95611

David Hurlburt	240 Second Street	San Francisco	CA	94044
Michelle Zygielbaum	5926 Yerba Buena Rd	Santa Rosa	CA	95409
Alene Lee	21847 E. Otero Pl	Aurora	CO	80016
Jerry Lee	21847 E. Otero Pl	Aurora	CO	80016
Lori Steevens	2741 14th St Apt B	Boulder	CO	80304
Marty Steevens	2741 14th St Apt B	Boulder	CO	80304
Matelsky	6965 Gaylebyn Lane	Colorado Springs	CO	80919
Sharry Erzinger, DrPH	2518 County Rd 5	Fraser	CO	80442
Abbey Secord	9190 Ironwood Way	Highlands Ranch	CO	80129
Don Secord	9190 Ironwood Way	Highlands Ranch	CO	80129
Steven Goral	1085 West 148th Avenue	Westminister	CO	80020
Holly Hansen-Estick	16F2Scuppo Road	Danbury	CT	06811
David Allgaier	205 Baldwin Ave.	Meriden	CT	06450
Marc Zimmerman	95 Hillspoint Road	Westport	CT	06880
Stacey Zimmerman	95 Hillspoint Road	Westport	CT	06880
Shannon Roche	1307 Wallach Place NW	Washington	DC	20009
Noah Rosenberg	1307 Wallach Place NW	Washington	DC	20009
Eduardo Serrano	1015 NE 120 th Street	Biscayne Park	FL	33161
Linda Calderon	930 SW Mulberry Way	Boca Raton	FL	33486
Gerry Lagreux	930 SW Mulberry Way	Boca Raton	FL	33486
Alex Kaplinski	6797 NE 7th Ave	Boca Raton	FL	33487
Norma Mantz	100 Cane Breakers Drive	Cocoa	FL	32927
Jim Jones	10555 NW 40th st	Coral Sprins	FL	33065
Lauri Jones	10555 NW 40th st	Coral Sprins	FL	33065
Steve Adamchack	2500 SW 81st Ave Apt. 301	Davie	FL	33324
Lucy Howie	2500 SW 81st Ave Apt. 301	Davie	FL	33324
Daniel Villarruel	111 NW 54th Court	Fort Lauderdale	FL	33309
Amy Greene	3260 Riverland Road	Fort Lauderdale	FL	33312
Kim Morgan	525 SW 18th Ave., Unit 25	Fort Lauderdale	FL	33312
Tony Vagnuolo	525 SW 18th Ave., Unit 25	Fort Lauderdale	FL	33312
Christine Shaw-Johnson	5772 NE 17th Avenue	Fort Lauderdale	FL	33334
Troy Johnson	5772 NE 17th Avenue	Fort Lauderdale	FL	33334
Mike Dolfinger	17 th Ave	Fort Lauderdale	FL	33334
Deb Dolfinger	17 th Ave	Fort Lauderdale	FL	33334
Jill Allen	646 Powell Drive	Fort Walton	FL	32547
Danette Lopez-Montney	103 Compass Rose Drive	Groveland	FL	34736
Ron Schmidt	2210 N 51st Ave	Hollywood	FL	333021
Corey Hulbert	2210 N 51st Ave	Hollywood	FL	333021
Donna Prieto	1025 Washington Street	Hollywood	FL	33019
Yesenia Courtney	331 Laurina Street Apt. 548	Jacksonville	FL	32216
Melissa Kral	7423 Leroy Drive	Jacksonville	FL	32244
Michael Kral	7423 Leroy Drive	Jacksonville	FL	32244
Ellen Canfield	15707 Ibis Ridge Drive	Lithia	FL	33547
Pershing W. Canfield Jr	15707 Ibis Ridge Drive	Lithia	FL	33547
Annette Reddy	15731 Citrus Grove Blvd.	Loxahatchee	FL	33470
Patrick Reddy	15731 Citrus Grove Blvd.	Loxahatchee	FL	33470
Elaine Forin	6623 Coral Lake Drive	Margate	FL	33063
Norman Forin	6623 Coral Lake Drive	Margate	FL	33063
Linda Swayze	915 New Hampton Wat	Merritt Island	FL	32953

Leonard Swayze	915 New Hampton Wat	Merritt Island	FL	32953
Jessica Francos	2420 NW 29th Street	Miami	FL	33142
Luis Lopez	4960 SW 95 th Court	Miami	FL	33165
Russell W. Everett	2900 N. Hwy. A1A 9B	N. Hutchinson Island	FL	34949
Alicia Murray-Bodnar	221 18th Street S.E.	Naples	FL	34117
Linda Heuertz	4310 NE 16 Avenue	Oakland Park	FL	33334
Jimmy Turnbull	14 Oack Circle Pass	Ocala	FL	34472
Anita Stewart	14 Oack Circle Pass	Ocala	FL	34472
Jose Albarra	6572 Lk Pembroke PL	Orlando	FL	32829
Jeffrey Schwartz	1075 Beckstrom Drive	Oviedo	FL	32765
Laura Schwartz	1075 Beckstrom Drive	Oviedo	FL	32765
Joe Pastaia	4891 Kylemore Court	Palm Harbor	FL	34685
Barbara Vagenas	305 Carolyn Ave	Panama City Beach	FL	32407
Jim Vagenas	305 Carolyn Ave	Panama City Beach	FL	32407
Rebecca Sera	17136 NW 10th Street	Pembroke Pines	FL	33028
Octavio Sera	17136 NW 10th Street	Pembroke Pines	FL	33028
Larry Stewart	7891 NW 12th Street	Pembroke Pines	FL	33024
Deidre Stewart	7891 NW 12th Street	Pembroke Pines	FL	33024
Meghan Stewart	7891 NW 12th Street	Pembroke Pines	FL	33024
Allison Stewart	7891 NW 12th Street	Pembroke Pines	FL	33024
Aleem Mailk	2101 Scenic Hwy J108	Pensacola	FL	32503
Carlos Gomez	151 NW Willow Grove Ave	Port Saint Licie	FL	34986
Terrance Navin	540 Carillion Parkway 1016	Saint Petersburg	FL	33716
Dorinda Davis	8417 Cypress Lake Circle	Sarasota	FL	32430
Holly Roderman	174 East Riverbend Drive	Sunrise	FL	33326
Brian Roderman	174 East Riverbend Drive	Sunrise	FL	33326
Valerie Schwartz	816 SW 158th Ter	Sunrise	FL	33326
Steven Schwartz	816 SW 158th Ter	Sunrise	FL	33326
Mary Brady	816 SW 158th Ter	Sunrise	FL	33326
Joseph Turnbull	816 SW 158th Ter	Sunrise	FL	33326
Patrick Turnbull	816 SW 158th Ter	Sunrise	FL	33326
Mary Lou Brady	816 SW 158th Ter	Sunrise	FL	33326
Laura Ferency	840 South Wind Circle	Sunrise	FL	33326
Bill Ferency	840 South Wind Circle	Sunrise	FL	33326
Wendy Johnston	1404 Sturbridge Place	Tallahassee	FL	32308
David Johnston	1404 Sturbridge Place	Tallahassee	FL	32308
Martha Soder	2464 Papillion Way	Tallahassee	FL	32309
Catherine Hanks	Green Street	Tallahassee	FL	32303
Mariene Hanna	2826 Frogs Leap Way	Tallahassee	FL	32309
Jean Ainsworth	6389 Mallard Trace	Tallahassee	FL	32312
Paul Rollin	6389 Mallard Trace	Tallahassee	FL	32312
Eleanor Dietrick	640 Muriel Ct	Tallahassee	FL	30303
Jessica Stribling	3704 Carroll Wood Pl Circle 9 305	Tampa	FL	33618
Louise Huntman	1567 Apache Circle	Tavares	FL	32778
Barbara Ford	Ramblers Rest 1300 N Rivers Rd W#17	Venice	FL	34293
Blaine Payne	220 Egret Court	Weston	FL	33327
Carrie Lutenberg	3664 Heron Ridge Lane	Weston	FL	33331
Greg Lutenberg	3664 Heron Ridge Lane	Weston	FL	33331
Janine Tomlinson	7449 SE 182 Blvd	White Springs	FL	32096

Malcolm Tomlinson	7449 SE 182 Blvd	White Springs	FL	32096
Gayle Esdaille	14103 Pipevine Dr	Winter Garden	FL	34787
Robert Turnbull	1424 Las Cruces Drive	Winter Springs	FL	32708
Jan Turnbull	1424 Las Cruces Drive	Winter Springs	FL	32708
Young Phan	9919 Majestic Way	Boynton Beach	FL	33437
Carolyn Mills	2007 A Schley Ave.	Albany	GA	31707
John and Carolyn Mills	2007 A Schley Ave.	Albany	GA	31707
Marsha Thomas	2120 Powers Ferry Road, SE, Ste. 200	Atlanta	GA	30339
Frances Lee		Columbus	GA	31909
Lucy Pfeffa	1229-B Church Street	Decatur	GA	30030
Pamela J. Rodgers	301 Charity Ave A2	Fort Valley	GA	31030
Willie E. Rodgers, Jr	301 Charity Ave A2	Fort Valley	GA	31030
LaWanna Welch	306 Humble Ave. # A-8	Fort Valley	GA	31030
David Anderson	P O Box 701	Lithonia	GA	30058
Linda Anderson	P O Box 701	Lithonia	GA	30058
C.F. Authement	307 Humble Ave	Perry	GA	31069
Doris Authement	307 Humble Ave	Perry	GA	31069
Gail Authement	307 Humble Ave	Perry	GA	31069
Nancy Woods	4079 Foorhill Avenue	St. Ansgar	IA	50472
Roger Woods	4079 Foorhill Avenue	St. Ansgar	IA	50472
Helen May	4872 Quail Drive	Staceyville	IA	50476
Susan May	4872 Quail Drive	Staceyville	IA	50476
Duane May	4872 Quail Drive	Staceyville	IA	50476
Peggy Shaw	1502 Colorado Ave	Boise	ID	83708
Vern Shaw	1502 Colorado Ave	Boise	ID	83708
Cecelia A. Connors	322 East 8th Street	Moscow	ID	83843
Peggy Brown	2102 Woodhavens Drive	Bloomington	IL	61701
Jennifer Prill	3511 Ballyford Drive	Bloomington	IL	61704
Santhos Valloppillil	2555 North Clark St Apt 901	Chicago	IL	60614
Nancy Malcolm	1106 S McLean St	Hudson	IL	61748
Carolyn Zembron	9328 N. Oak Park Ave.	Morton Grove	IL	60053
Andrea Kichinko	542 Wellington Ct	Mundelein	IL	60060
Janet Graeff	437 Illinois Ave Ext.	Murphysboro	IL	62966
Lacey Caraway	7 Bodkin Lane	Murphysboro	IL	62966
Cindy Traska	3601 Greenleaf Court	Spring Grove	IL	60081
Carolyn Bagley	4130 Highbury Drive	Springfield	IL	62711
Joe Bagley	4130 Highbury Drive	Springfield	IL	62711
Richard C Bagley Jr.	4130 Highbury Drive	Springfield	IL	62717
Diane Dooley	119 N. Bergan	W. Peoria	IL	61604
Lupe Martinez	119 N. Bergan	W. Peoria	IL	61604
Pat Bakanec	2710 5th Street	Winthrop Harbor	IL	60096
Dean Boswell	814 Landon Ave	Winthrop Harbor	IL	60096
Teresa M. Miller	307 Victory Hill	Coatesville	IN	46121
Chad Swagart	10580N. Portland Arch Road	Covington	IN	47932
Debra Swagart	10580N. Portland Arch Road	Covington	IN	47932
Glenn Swagart	10580N. Portland Arch Road	Covington	IN	47932
Jackie Swagart	10580N. Portland Arch Road	Covington	IN	47932
Fred Essary	403 Pfaffin Ct	Mt. Vernon	IN	47620
Sue Essary	403 Pfaffin Ct	Mt. Vernon	IN	47620

Nichole Kreuger	306 Indiana Ave #2	Valparaiso	IN	46383
Christopher Kreuger	306 Indiana Ave #2	Valparaiso	IN	46383
Renee Allen	504 Garretson Street	Burlington	KS	66839
Carmen Landreth	1131 E. Sheridan Bridge Lane	Olathe	KS	66062
Colleen Floyd	16016 Grandview Street	Stilwell	KS	66085
Angelia Terry	7440 Vest Talcum Road	Bulan	KY	41722
William B Terry	7440 Vest Talcum Road	Bulan	KY	41722
Billy Sizemore	2019 Hwy 80	Emmalena	KY	41740
Marie Sizemore	2019 Hwy 80	Emmalena	KY	41740
Christopher D. Handshoe	2601 Reed Street	Flatwoods	KY	41139
Shirley Watley	33 Mitchell Lane	Hindman	KY	41822
Salina Gibson	PO Box 49	Hindman,	KY	41822
Vivian Watts	3625 Laurel Fork Road	Hueysville	KY	41640
Ina Owens	11 Sunshine Lane	Leburn	KY	41831
Ford Thomas	1416 Hwy 1087 East	Leburn	KY	41831
Sharon Watts Thomas	1416 Hwy 1087 East	Leburn	KY	41831
Carole Hamilton	3439 Buckhorn Drive #130	Lexington	KY	40515
Yvonne Hall	5365 Stewart Road	Lexington	KY	40516
Paul Hall	5365 Stewart Road	Lexington	KY	40516
Anna Beth Mekus	920 Cherrywood Drive	Lexington	KY	40515
Janet Blaine		Louisville	KY	40207
Bill Francis	8888 Hwy 550 East	Mousie	KY	41839
Michael Heath	105 Brentwood Drive	Winchester	KY	40391
Phil Handshoe	4355 Colby Road	Winchester	KY	40391
Michael Diana	124 Atlantic Ave Apt 4	Revere	MA	02151
Peggy Cone	6810 Maple Leaf Court Apt 201	Baltimore	MD	21209
Onika Herman	1304 Chalmers Road	Silver Spring	MD	20903
Jeff Herman	1304 Chalmers Road	Silver Spring	MD	20903
Herman Hamilton	8893 Water St. Rd	Walkersville	MD	21793
Jode Murray	P O Box 2655	Kennebunkport	ME	04046
Jennifer Cutts	P O Box 1212	York Harbor	ME	03911
Kathy Leslie	P O Box 1411	Ann Arbor	MI	48106
Shirley Lafontaine	12347 N Jennings Rd	Clio	MI	48420
Anthony R. Karabacz	7267 Pheasant Run	Gaylord	MI	49735
Nancy J Karabacz	7267 Pheasant Run	Gaylord	MI	49735
Tina Stewart	18014 Meridian Road	Grosse Ile	MI	48138
Lynn Hahn	20221 Island Estate Drive	Grosse Ile	MI	48138
Terry M. Hahn	20221 Island Estate Drive	Grosse Ile	MI	48138
Heidi Miklos	20450 Canal	Grosse Ile	MI	48138
Christian Hahn	8672 Lake Road	Grosse Ile	MI	48138
Jill Hahn	8672 Lake Road	Grosse Ile	MI	48138
Christy Cobb	8772 Woodside Drive	Grosse Ile	MI	48138
Don Critchfield	9135 Church Rd	Grosse Ile	MI	48138
Phyllis Critchfield	9135 Church Rd	Grosse Ile	MI	48138
Anthony G. Rich	35912 Joy Rd.	Livonia	MI	48150
Harry Turnbull	11796 s. Coleman Road	Maple City	MI	49664
Dennis Felax Sr	2236 Shore Rd	Rogers City	MI	49779
Russ Martin	665 South Lake Street	Rogers City	MI	49779
Wendy Martin	665 South Lake Street	Rogers City	MI	49779

Lisa Moscynski	1133 Cedar	Wyandotte	MI	48192
Paul Moscynski	1133 Cedar	Wyandotte	MI	48192
Zachary Moscynski	1133 Cedar	Wyandotte	MI	48192
Exequiel Bravo	5340 Emerald way	Apple Valley	MN	55124
Lois Bravo	5340 Emerald way	Apple Valley	MN	55124
Jean Johnson	338 N. 5th Street	Bayport	MN	55003
Brian Maxey	6248 Sunrise Terrace No.	Brooklyn Park	MN	55428
Roxy Maxey	6248 Sunrise Terrace No.	Brooklyn Park	MN	55428
Tony Wolynic	8609 West River Road	Brooklyn Park	MN	55444
Katrina Wolynic	8609 West River Road	Brooklyn Park	MN	55444
Delayne Landon	321 Park Street W. #301	Cannon Falls	MN	55009
Larry Pederson	1021 W. River Parkway	Champlin	MN	55316
Helen Pederson	1021 W. River Parkway	Champlin	MN	55316
Bob Sorensen	11216 Xenia Ave N	Champlin	MN	55316
Deb Sorensen	11216 Xenia Ave N	Champlin	MN	55316
Larry Urbanski	4529 Chatham Place	Col. Heights	MN	55421
Deb Urbanski	4529 Chatham Place	Col. Heights	MN	55421
Gardner Carruthers	4937 N.E. 6th Street	Col. Heights	MN	55421
Margaret Carruthers	4937 N.E. 6th Street	Col. Heights	MN	55421
Regina Wolynic	3700 Jackson St	Columbia Heights	MN	55421
Vicky Brusseau	565 51st Ave NE	Columbia Heights	MN	55421
Dan Brusseau	565 51st Ave NE	Columbia Heights	MN	55421
Dustin Wallin	11316 Palm St. N.E.	Coon Rapids	MN	55448
Chelsea Wallin	11316 Palm St. N.E.	Coon Rapids	MN	55448
Megan Casserly	6718 Valley Place	Crystal	MN	55427
Mary Olson	6718 Valley Place	Crystal	MN	55427
Jenny Olson	6718 Valley Place	Crystal	MN	55427
James Olson	6718 Valley Place	Crystal	MN	55427
Tom Lipke	4623 Tamie Ave	Eagan	MN	55123
Patrick Goral	6570 184th Street North	Forest Lake	MN	55025
Sherrie Goral	6570 184th Street North	Forest Lake	MN	55025
Russell May	603 13th Avenue NW	Kasson	MN	55944
Arlys May	603 13th Avenue NW	Kasson	MN	55944
Brad Krusemark	12827 90th place North	Maple Grove	MN	55369
Gina Krusemark	12827 90th place North	Maple Grove	MN	55369
Jan Kaehler	14800 80th Place North	Maple Grove	MN	55311
Darnell Kaehler	14800 80th Place North	Maple Grove	MN	55311
Kari Lindblad	7900 Lawndale Lane	Maple Grove	MN	55311
Julie Anderson	8676 Terrace View lane	Maple Grove	MN	55311
Tony Anderson	8676 Terrace View lane	Maple Grove	MN	55311
Clifford Shaw	13856 9th Ave. S.E.	Milaca	MN	56353
Ruby Shaw	13856 9th Ave. S.E.	Milaca	MN	56353
Virginia Krysa	1721 4th Street NE	Minneapolis	MN	55413
Lorraine Wolynic	1806 3rd Street NE	Minneapolis	MN	55418
Donald Wolynic	1806 3rd Street NE	Minneapolis	MN	55418
John D'Agostino	1918 Walden Place	Minneapolis	MN	55418
Barb D'Agostino	1918 Walden Place	Minneapolis	MN	55418
Patrica Matelsky	2109 St. Anthony Pkwy. NE	Minneapolis	MN	55418
Mike Shaw	2113 - 4th St N.E.	Minneapolis	MN	55418

Coleen Shaw	2113 - 4th St N.E.	Minneapolis	MN	55418
Mordan Shaw	2222 Benjamin St. N.E.	Minneapolis	MN	55418
Stanley Wolynic	2555 Ulysses Street NE	Minneapolis	MN	55418
Chris Wolynic	2555 Ulysses Street NE	Minneapolis	MN	55418
Marc Shaw	3605 Architect St. N.E.	Minneapolis	MN	55418
Jeanna Shaw	3605 Architect St. N.E.	Minneapolis	MN	55418
Steven Goral	333 10th Street NW, No. 212	New Brighton	MN	55112
Wayne Tembreull	2850 Zanzibar lane N.	Plymouth	MN	55447
Kathy Henkel	2850 Zanzibar lane N.	Plymouth	MN	55447
Ben Weigert	2445 Stonecrest Path NW	Prior Lake	MN	55372
Stephanie Wiegert	2445 Stonecrest Path NW	Prior Lake	MN	55372
Heather Stilwell	17576 305th Lane	Shaefer	MN	55074
Joan Gerr	4547 Churchill Street	Shoreview	MN	55126
Al Birr	8116 Fillmore St. N.E.	Spring Lake Park	MN	55432
Sandra Thorson	8116 Fillmore St. N.E.	Spring Lake Park	MN	55432
Shirley Edwards	2601 Kenzie Terrace	St. Anthony	MN	55418
William Edwards	2601 Kenzie Terrace	St. Anthony	MN	55418
Mayard Shaw	2835 Roosevelt St. N.E.	St. Anthony	MN	55418
Elizabeth Shaw	2835 Roosevelt St. N.E.	St. Anthony	MN	55418
Shirley Cashin	3517 Chalmersford Road	St. Anthony	MN	55418
Joan Seabold	360 Lexington Parkway	St. Paul	MN	55105
Mark Meyer	360 Lexington Parkway	St. Paul	MN	55105
Doloros Irwin	4027 Whitebear Parkway	White Bear	MN	55110
Gayle Chandler	9048 Duckwood Trail	Woodbury	MN	55125
Tom Chandler	9048 Duckwood Trail	Woodbury	MN	55125
Nick Frohnauer	5204 Sandstone Dr.	Columbia	MO	65202
Jen Frohnauer	5204 Sandstone Dr.	Columbia	MO	65202
Jackie Landreth	149 Canyon Forest Way	Kimberling City	MO	65686
David Landreth	149 Canyon Forest Way	Kimberling City	MO	65686
Hazel Harrison	454 Farmers Lane	Lebanon	MO	65536
Craig Kozicki	10 Robert Johns Way	St. Charles	MO	63303
Emily Kozicki	10 Robert Johns Way	St. Charles	MO	63303
Shelly Kozicki	10 Robert Johns Way	St. Charles	MO	63303
Yvonne Rousse	10 Robert Johns Way	St. Charles	MO	63303
Brent Skramstad	925 2nd Street North	Havre	MT	59501
Tanis Hernandez	214 E. 3rd Street	Libby	MT	59923
G. Benefield	245 Cedar Meadow Road	Libby	MT	59923
Norita Skramstad	3647 Highway 2 Sourt	Libby	MT	59923
Myra Cole	E. Lincoln Blvd	Libby	MT	59923
Kimberly Rowse	214 East 3rd Street	Libby	MT	59923
Dorothy Ansell	2816 Maple Ave	Burlington	NC	27215
Wanda Jumper	51 Toshas Way	Fletcher	NC	28732
Karen Hammons	17240 Cabarrus Road	Midland	NC	28107
Barbara Bedwell	4012 Mardela Spring Drive	Raleigh	NC	27616
Joan Curry,	9131 Anson Way	Raleigh	NC	27615
Patricia Glatz	204 Woodland Dr	Swansboro	NC	28584
Susan Sehorn	74 Taylor Terrace	Swansboro	NC	28584
Arthur I. Zygielbaum	6602 Pinecrest Dr.	Lincoln	NE	68516
Carole H. Carlo	8 Bunting Drive	Chesterfield	NJ	08515

Donna Carlo	8 Bunting Drive	Chesterfield	NJ	08515
Susan Perez	246 Edgar Place 2D	Elizabeth	NJ	07202
Julie Chen	189 Kemper Court	Hackettstown	NJ	07840
John Bartolomeo	62 Ivy Hill Road	Lakewood	NJ	08701
Gail Mattsson	79 Sunrise Court	Lakewood	NJ	08701
Robert Mattsson	79 Sunrise Court	Lakewood	NJ	08701
David E. Cutts	81 Sunrise Court	Lakewood	NJ	08701
Bonnie Diana	3303 Rio Vista Drive	Mahwah	NJ	07430
Kate Diana	3303 Rio Vista Drive	Mahwah	NJ	07430
Charlene Komar Storey	318 Pershing Ave.	Roselle Park	NJ	07204
Gregory D. Storey	318 Pershing Ave.	Roselle Park	NJ	07204
Elizabeth Toner	102 Rayewood Drive	Wantage	NJ	07461
Lawrence Bamdas	12 Pine Terrace	Wayne	NJ	07470
Olga Diana	1211 Richmond Rd	West Milford	NJ	07480
Margy Urnberg	3760 Meadow Wood Road	Carson City	NV	89703
Paul Urnberg	3760 Meadow Wood Road	Carson City	NV	89703
Stella (Moran) Stephens	4591 Wagon Wheel Rd.	Carson City	NV	89703
Will Smith	4601 Wagon Wheel Road	Carson City	NV	89703
Kimberly Smith	4601 Wagon Wheel Road	Carson City	NV	89703
Sandy May	31-28 47th Street	Astoria	NY	11103
David Merkrebs	9 Lake Drive	Hewlett	NY	11557
Randy Merkrebs	9 Lake Drive	Hewlett	NY	11557
Kristina Graff	34-20 74th Street, Apt 3B	Jackson Heights	NY	11372
Joseph Collins	34-20 74th Street, Apt 3B	Jackson Heights	NY	11372
Linda Turpin	6773 Rapids Road	Lockport	NY	14094
John Wang	1230 York Ave Box 92A	New York	NY	10021
Cory Crayn	170 West 23rd Street #4T	New York	NY	10011
Cecile Miller	184 W 134th Street 3	New York	NY	10030
Lisa Labrado	220 East 57th Street No. 15k	New York	NY	10022
Sheila Zachman	276 West 11th Street, Apt. 1	New York	NY	10014
Trina Semorile	445 West 46 Street, #1E	New York	NY	10036
Alexis Feldman	75 Chambers Street, No. 3	New York	NY	10007
Christopher Leahy	75 Chambers Street, No. 3	New York	NY	10007
Alexis Ufland	9 Barrow Street	New York	NY	10014
David Beanning	99 East 4th street Apt 3C	New York	NY	10003
Mary Kahal	6042 Lockard Ave.	Cincinnati	OH	45230
Lisa H Corsale		Columbus	OH	43085
Garry Handshoe	434 1/2 South Broadway	Greenville	OH	45331
Jayne Hall	173 Beau Chemin	Louisville	OH	44641
S. T. Adkins	53 E. Whitney Ave.	Shelby	OH	44875
Travis Inga	7500 N Oakcliff Drive	Tulsa	OK	74126
Bobbi Inga	7500 N Oakcliff Drive	Tulsa	OK	74126
Robert Waxman	289 York Blvd	Hamilton	Ontario	
Chester Waxman	289 York Blvd	Hamilton	Ontario	
Katie Vaughan	900 State St Box A227	Salem	OR	97301
Teresa Findlay	6262 Hubbard Creek Rd	Umpqua	OR	97430
Daniel McCormick	130 Tennis Ave	Ambler	PA	19002
Sharon McCormick	130 Tennis Ave	Ambler	PA	19002
Jill Adaman	234 S. Bryn Mawr Ave	Bren Mawr	PA	19010

Caesar Augustus	112 Hasian Lane	Coatesville	PA	19320
Jeanne Singer	2202 Rankintown Road	Finleyville	PA	15332
Joel Steinberg	1332 Wright Drive	Huntington Valley	PA	19006
Pat Karpowicz	717 Willow Street	Lansdale	PA	19446
Susan Foehl	79 Line Road	Malvern	PA	19355
June E. Breit, RN, BSN, CCM	8 Pawlings Circle	Phoenixville	PA	19460
Mark Shanaway	10419 Lindberg Ave	Pittsburg	PA	15235
Polly Boore	RR2 Box 601	Tyrone	PA	16686
Joanne Caulfield	1533 Meadowbrook Lane	West Chester	PA	19380
Patrick McBride	20 South New Street	West Chester	PA	19380
Carolyn S McDowell	1146 Brookside Drive	Hanahan	SC	29406
Byron Roach	1211 Hawthorne Road	Hanahan	SC	29406
Karen Roach	1211 Hawthorne Road	Hanahan	SC	29406
Sylvia Chennault	1232 Hawthorne Circle	Hanahan	SC	29406
Jeanette Tarr	2812 Hume Ave	Hanahan	SC	29410
Randolph Wicker	113 Miller Springs Drive	Moore	SC	29369
Mabel Wicker	113 Miller Springs Drive	Moore	SC	29369
Stanley Jones	2561 Fassitt Road Apt C-13	North Charleston	SC	29406
Ellen Walker	4969 Centre Pointe Drive, Suite 200	North Charleston	SC	29418
Pat Peterson	3700 S. Westport Ave #3590	Suix Falls	SD	57106
Russ Peterson	3700 S. Westport Ave #3590	Suix Falls	SD	57106
Melinda L Robinson	200 Oxford Drive	Franklin	TN	37064
Diane K. Walter	221 Springpark Drive	Arlington	TX	76014
Wilbur F. Walter	221 Springpark Drive	Arlington	TX	76014
Jason L. Walter	5804 Falconcrest	Arlington	TX	76017
Martin T. Murray	1500 N Alexander Dr. #1	Baytown	TX	77520
Jonathon Dekle	223 South Rosemont	Dallas	TX	75208
Tara Dekle	223 South Rosemont	Dallas	TX	75208
Susan Canon	2394 CR302	Floresville	TX	78114
Kirby Goldsberry	2629 Timberhaven Drive	Flower Mound	TX	75028
Marcus Lewis	2929 Socrates Drive	Grand Prairie	TX	75052
Tami L. Lewis	2929 Socrates Drive	Grand Prairie	TX	75052
Tom Kendle	2904 Crest Haven Drive	Grapevine	TX	76051
Amy Hall	16415 Crossfield Drive	Houston	TX	77095
Greg Hall	16415 Crossfield Drive	Houston	TX	77095
Laura Martinez	9223 Red Castle Lane	Humble	TX	77396
Bud Vaughn	1415 Bayshore Drive	Kemah	TX	77565
Jill Vaughn	1415 Bayshore Drive	Kemah	TX	77565
Jack Ramsey	5 Bethpage Dr	Laguna Vista	TX	78578
Joan Herum	5 Bethpage Dr	Laguna Vista	TX	78578
Paul Zeugin	2732 Dunbar DR	McKinney	TX	75070
Chad Medlin	4341 Waskom	Plano	TX	75024
Crystal Medlin	4341 Waskom	Plano	TX	75024
Lee Hurtado	162 Waxwood Lane	San Antonio	TX	78216
Chuck Every	1310 Kloecker Rd	Sealy	TX	77474
Karen Every	1310 Kloecker Rd	Sealy	TX	77474
Dirk Cox	1300 Shelby Court	Wylie	TX	75098
Anna Marie Murray	1004 Timber Ridge Court	Chesapeake	VA	23322
Risha Cross	1223 Woods Way	Chesapeake	VA	23323

Christine Smith Oxford	43428 Westchester Square	Leesburg	VA	20176
Edna E. Wright	3671 Bell Street	Norfolk	VA	23513
Ellen Whitehurst	2224 Kendall Street	Virginia Beach	VA	23451
Erin Baker	5825 Ludington Drive	Virginia Beach	VA	23464
Grant Baker	5825 Ludington Drive	Virginia Beach	VA	23464
Mary Charlotte Boyette	27443 Buckhorn Drive	Windsor	VA	23487
Donna Greenup	P O Box 84	Colbert	WA	99005
Pam Johnson	4608-B 164th Street SW	Lynnwood	WA	98087
Roy Bell	8028 186th Ave. SW	Rochester	WA	98579
Virgie Bell	8028 186th Ave. SW	Rochester	WA	98579
Rebecca Jones	4743 - 48th Ave. N.E.	Seattle	WA	98105
Chisholm Jones	4743 - 48th Ave. N.E.	Seattle	WA	98105
Lynn A DeBeal	5023 42nd Ave S	Seattle	WA	98118
Lynda Hanley-Cole	3007 W. 17th Ave	Spokane	WA	99224
Carla Groce		Vancouver	WA	98660
Richard Bruder	1349-A 105th ave	Amery	WI	54001
Judy Bruder	1349-A 105th ave	Amery	WI	54001
Diana Dicosimo	524 Birchwood Ave	Amery	WI	54001
Jerry Johnson	649 70th Ave	Amery	WI	54001
Marcia Johnson	649 70th Ave	Amery	WI	54001
Stacy Dosch	738 85th	Amery	WI	54001
Donovan Dosch	959 Vijobi Trail	Amery	WI	54001
Peggy Dosch	959 Vijobi Trail	Amery	WI	54001
Don Johnson	319 E. River Ave	Barron	WI	54812
Craig Allram	284 6th Avenue	Clayton	WI	54004
Cindy Allram	284 6th Avenue	Clayton	WI	54004
Hillary Arcand	677 1st street	Clayton	WI	54004
Shannon Arcand	677 1st street	Clayton	WI	54004
Trish Monson	277 75th Street	Clear Lake	WI	54005
Andrew Monson	277 75th Street	Clear Lake	WI	54005
Jan Monson	277 75th Street	Clear Lake	WI	54005
Ashley Kehoe	277 75th Street	Clear Lake	WI	54005
Donald Dosch	279 75th street	Clear Lake	WI	54005
Artie Dosch	279 75th street	Clear Lake	WI	54005
Doug Allram	387 60th Avenue	Clear Lake	WI	54005
Louann Allram	387 60th Avenue	Clear Lake	WI	54005
Amy Wienke	764 30th ave	Clear Lake	WI	54005
Ben Wienke	764 30th ave	Clear Lake	WI	54005
Wendy Stoeckler	W5936 Lee Drive	Fort Atkinson	WI	53538
Steve Dosch	13501 N Refuge RD.	Grantsburg	WI	54840
Linda Dosch	13501 N Refuge RD.	Grantsburg	WI	54840
Julie Everson	1203 Red Oak Road	Hudson	WI	54016
John Everson	1203 Red Oak Road	Hudson	WI	54016
Roberta Pabich	N9899 County Road K	Loyal	WI	54446
Jesse Gerhardt	14 Glen Hwy	Madison	WI	53705
Daniel Gerhardt	14 Glen Hwy	Madison	WI	53705
Linda Commer	1700 East Chateau Place	Milwaukee	WI	53217
Jan Spangler	2844 N. Hackett Ave	Milwaukee	WI	53211
Jean Eisenman	2844 N. Hackett Ave	Milwaukee	WI	53211

Noel Spangler	2844 N. Hackett Ave	Milwaukee	WI	53211
Shawn Bird	1008 192nd ave	New Richmond	WI	54017
Shawn Bird	1008 192nd ave	New Richmond	WI	54017
Michelle Holland	538 Park Veidrive	New Richmond	WI	54017
Michelle Holland	538 Park Veidrive	New Richmond	WI	54017
Mike Jackelen	1680 County Rd T	New Richmond	WI	54017
Bonnie Jackelen	1680 County Rd T	New Richmond	WI	54017
Angie Johnson	358 195th Street	Osceloa	WI	54020
Dan Logan	358 195th Street	Osceloa	WI	54020
Rick Allram	299 County road F	Prairie Farm	WI	54762
Darice Allram	299 County road F	Prairie Farm	WI	54762
Kyle Pierce	W7180 Sunset Lane	Spooner	WI	54801
Kari Pierce	W7180 Sunset Lane	Spooner	WI	54801
Justin Allram	205 Jerdee Ave	Star Prairie	WI	54026
Jessica Allram	205 Jerdee Ave	Star Prairie	WI	54026
Jeff Pierce	N7865 Trego Landing Road	Trego	WI	54888
Jerad Pierce	N7865 Trego Landing Road	Trego	WI	54888
Jean Pierce	N7865 Trego Landing Road	Trego	WI	54888
Joy Pierce	N7865 Trego Landing Road	Trego	WI	54888
Janice McNitt	W5543 Cty F	Trego	WI	54888
Janice McNitt	W5543 Cty F	Trego	WI	54888
Tonia Luna	3101 N 103rd st	Wawuatosa	WI	53222
Marcello Luna	3101 N 103rd st	Wawuatosa	WI	53222

RESPONSE FROM LINDA REINSTEIN TO AN ADDITIONAL QUESTION
FROM SENATOR BOXER

Question. You have a number of health professional and other people who have signed on in support of your statement. Can you please describe how widespread this support is?

Response. [The names in support of the statement follow:]

In 72 hours, there was support from doctors, scientist, medical institutions and victims around the world.

The signatures below support these four points and wish to have their names entered into the Congressional Record.

1. **Ban Asbestos**
2. **No safe level of asbestos**
3. **Invest in research for a cure and improve treatments**
4. **Education to prevent exposure**

U.S. Capitol Tunnel Shop Workers

John Thayer
Scott Smith
Tom Baker
Martin Blanchet
Ed Hill
Frank Binns
Charlie Morris
Tim Taylor
Christian Raley
Rick Leanord

Jordan Zevon
Los Angeles, CA

International Mesothelioma Interest Group
Dr. Hedy Kindler, President, IMIG

Arthur L. Frank, MD,PhD,
Professor of Public Health and Chair, Department of Environmental and Occupational Health
Drexel School of Public Health
Co-Chair-ADAO Scientific Advisory Board.

Michael R. Harbut, MD, MPH, FCCP
Wayne State University
Chief, Center for Occupational and Environmental Medicine
118 N. Washington
Royal Oak, Michigan 48067-1751

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5841 S. Maryland Avenue, Mc 2115
Chicago, Il., 60637-1470

Dr. Robert Taub
Herbert Irving Comprehensive Cancer Center
161 Fort Washington Ave.
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New York, NY 10032

Daniel H. Serman, M.D.
Associate Professor of Medicine
Associate Professor of Medicine in Surgery
Director, Interventional Pulmonology Program
Clinical Director, Thoracic Oncology Gene Therapy Program

Pulmonary, Allergy, & Critical Care Division
University of Pennsylvania Medical Center

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Medical Director
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Lincoln County Asbestos Victims Relief Organization
Gayla Benefield
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Laurie Kazan-Allen
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Michael Bowker, Author
Fatal Deception

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Rachel Lidov

Co-Coordinator
9/11 ENVIRONMENTAL ACTION
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Barbara Zeluck
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Asbestos Victims Support Groups' Forum
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Helen Clayson
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Southeast Asbestos Awareness and Victims Support Group
Pauline Bonney
East Sussex.BN23 8DU. England

Save Spodden Valley campaign
Jason Addy
185 Rooley Moor Rd, Rochdale
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Sheffield And Rotherham Asbestos Group
Paula Walker
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Sheffield, South Yorkshire, UK

Derbyshire Asbestos Support Team
Joanne Carlin
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Ban Asbestos Network of India (BANI)
Gopal Krishna
Occupational & Environmental Health Network of India (OEHNI)

ABREA - Brazilian Association of Asbestos Exposed People from Rio de Janeiro e Sao Paulo

ABEA- Asbestos Exposed People from Bahia

Virtual-Citizen Network for the Ban of Asbestos in Latin America

Fernanda Giannasi, Civil and Safety Engineer, Labour Inspector
Labour Department in São Paulo State

Name	Address	City	State	ZIP
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Charles R Archer	P O Box 348	Ragland	AK	35131
Karen Moss	3400 Snow Goose Dr.	Wasilla	AK	99654
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Melissa G. Rose	1551 Oakwood Drive	Brookwood	AL	35444
Betty Kincaid	6 Ridge Drive	Pelham	AL	35124
Ronnie Kincaid	6 Ridge Drive	Pelham	AL	35124
June Jensen	28 Ciclamor Way	Hot Springs Village	AR	71909
Eileen Day	1 Browning Place Seven Hills 2147	Sydney	Australia	
Cindy Bundrock	1339 W. Boston Street	Chandler	AZ	
Lois Schwarting	201 E Ivanhoe	Gilbert	AZ	85295
Marty Schwarting	201 E Ivanhoe	Gilbert	AZ	85295
Lois Schwarting	201 E Ivanhoe	Gilbert	AZ	85295
Greg Lee	40104 W. Marylou Drive	Maricopa	AZ	85239
Lisa Lee	40104 W. Marylou Drive	Maricopa	AZ	85239
Debra J Farnsworth	10907 E. Acoma Drive	Scottsdale	AZ	85255
Rita McGuire	21305 N. 107th Drive	Sun City	AZ	85373
Erica Bender	1164 Euclid Ave	Berkeley	CA	94708
Daniel Horodysky	2213 Acton Street	Berkeley	CA	94702
Tom Condit	2217 1/2 McGee Ave	Berkeley	CA	94703
Natalie Rosenberg	320 Lemon Drive	Camarillo	CA	93010
Laura Earhart	4091 Vincente Ave	Camarillo	CA	93010
Arlene O'Connor	24697 Gleneagles Drive	Corona	CA	92883
Rhio Oconnor	24697 Gleneagles Drive	Corona	CA	92883
Arlene Oconnor	24697 Gleneagles Drive	Corona	CA	92883
Rhio O'Connor	25697 Gleneagles Drive	Corona	CA	92883
Burke McCarthy	912 Powell Court	Costa Mesa	CA	02626
F. Burke McCarthy	912 Powell Court	Costa Mesa	CA	92626
Laurie Pollan	33243 Ocean Ridge	Dana Point	CA	92629
Eric Johnson	5343 Tanaya	Fresno	CA	93722
Susan E. Burke	25761 Le Parc #104	Lake Forest	CA	92630
Bert Mintz	5657 Wilshire Blvd Suite 310	Los Angeles	CA	90036
Hanne Mintz	5657 Wilshire Blvd Suite 310	Los Angeles	CA	90036
Marina Mintz	5657 Wilshire Blvd Suite 310	Los Angeles	CA	90036
Amanda Urnberg	90 F Street	Martinez	CA	94553
Julia McCarthy	2827 Catalpa Street	Newport Beach	CA	92660
Russell Beemer	2827 Catalpa Street	Newport Beach	CA	92660
Joseph Mennealy	11242 Lakeland Road	Norwak	CA	90650
Terri Gutai	438 Vernon Street	Oakland	CA	94601
Tony Vickers	1336 Via Romero	Palos Verdes Estates	CA	90274
Kelly Casserly	480 Portafino Ct., #301	Phillips Ranch	CA	91766
Jessica Like	8500 Pershing Drive #208	Playa del Rey	CA	90293
Jen Tanguileg	71 Viking Drive	Pleasant Hill	CA	94523
Robert Tanguileg	71 Viking Drive	Pleasant Hill	CA	94523
Ellen Tunkelrott	1613 Stanford Ave	Redondo Beach	CA	90278
Vickie Castaldi	234 N. Juanita Ave.	Redondo Beach	CA	90277
Kevin Akin	20212 Harvard Way	Riverside	CA	92507
Trisa Zembron Endicott	1015 Tanzania Drive	Roseville	CA	95611

David Hurlburt	240 Second Street	San Francisco	CA	94044
Michelle Zygielbaum	5926 Yerba Buena Rd	Santa Rosa	CA	95409
Alene Lee	21847 E. Otero Pl	Aurora	CO	80016
Jerry Lee	21847 E. Otero Pl	Aurora	CO	80016
Lori Steevens	2741 14th St Apt B	Boulder	CO	80304
Marty Steevens	2741 14th St Apt B	Boulder	CO	80304
Matelsky	6965 Gaylebyn Lane	Colorado Springs	CO	80919
Sharry Erzinger, DrPH	2518 County Rd 5	Fraser	CO	80442
Abbey Secord	9190 Ironwood Way	Highlands Ranch	CO	80129
Don Secord	9190 Ironwood Way	Highlands Ranch	CO	80129
Steven Goral	1085 West 148th Avenue	Westminister	CO	80020
Holly Hansen-Estick	16F2Scuppo Road	Danbury	CT	06811
David Allgaier	205 Baldwin Ave.	Meriden	CT	06450
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Stacey Zimmerman	95 Hillspoint Road	Westport	CT	06880
Shannon Roche	1307 Wallach Place NW	Washington	DC	20009
Noah Rosenberg	1307 Wallach Place NW	Washington	DC	20009
Eduardo Serrano	1015 NE 120 th Street	Biscayne Park	FL	33161
Linda Calderon	930 SW Mulberry Way	Boca Raton	FL	33486
Gerry Lagreux	930 SW Mulberry Way	Boca Raton	FL	33486
Alex Kaplinski	6797 NE 7th Ave	Boca Raton	FL	33487
Norma Mantz	100 Cane Breakers Drive	Cocoa	FL	32927
Jim Jones	10555 NW 40th st	Coral Sprins	FL	33065
Lauri Jones	10555 NW 40th st	Coral Sprins	FL	33065
Steve Adamchack	2500 SW 81st Ave Apt. 301	Davie	FL	33324
Lucy Howie	2500 SW 81st Ave Apt. 301	Davie	FL	33324
Daniel Villarruel	111 NW 54th Court	Fort Lauderdale	FL	33309
Amy Greene	3260 Riverland Road	Fort Lauderdale	FL	33312
Kim Morgan	525 SW 18th Ave., Unit 25	Fort Lauderdale	FL	33312
Tony Vagnuolo	525 SW 18th Ave., Unit 25	Fort Lauderdale	FL	33312
Christine Shaw-Johnson	5772 NE 17th Avenue	Fort Lauderdale	FL	33334
Troy Johnson	5772 NE 17th Avenue	Fort Lauderdale	FL	33334
Mike Dolfinger	17 th Ave	Fort Lauderdale	FL	33334
Deb Dolfinger	17 th Ave	Fort Lauderdale	FL	33334
Jill Allen	646 Powell Drive	Fort Walton	FL	32547
Danette Lopez-Montney	103 Compass Rose Drive	Groveland	FL	34736
Ron Schmidt	2210 N 51st Ave	Hollywood	FL	333021
Corey Hulbert	2210 N 51st Ave	Hollywood	FL	333021
Donna Prieto	1025 Washington Street	Hollywood	FL	33019
Yesenia Courtney	331 Laurina Street Apt. 548	Jacksonville	FL	32216
Melissa Kral	7423 Leroy Drive	Jacksonville	FL	32244
Michael Kral	7423 Leroy Drive	Jacksonville	FL	32244
Ellen Canfield	15707 Ibis Ridge Drive	Lithia	FL	33547
Pershing W. Canfield Jr	15707 Ibis Ridge Drive	Lithia	FL	33547
Annette Reddy	15731 Citrus Grove Blvd.	Loxahatchee	FL	33470
Patrick Reddy	15731 Citrus Grove Blvd.	Loxahatchee	FL	33470
Elaine Forin	6623 Coral Lake Drive	Margate	FL	33063
Norman Forin	6623 Coral Lake Drive	Margate	FL	33063
Linda Swayze	915 New Hampton Wat	Merritt Island	FL	32953

Leonard Swayze	915 New Hampton Wat	Merritt Island	FL	32953
Jessica Francos	2420 NW 29th Street	Miami	FL	33142
Luis Lopez	4960 SW 95 th Court	Miami	FL	33165
Russell W. Everett	2900 N. Hwy. A1A 9B	N. Hutchinson Island	FL	34949
Alicia Murray-Bodnar	221 18th Street S.E.	Naples	FL	34117
Linda Heuertz	4310 NE 16 Avenue	Oakland Park	FL	33334
Jimmy Turnbull	14 Oack Circle Pass	Ocala	FL	34472
Anita Stewart	14 Oack Circle Pass	Ocala	FL	34472
Jose Albarra	6572 Lk Pembroke PL	Orlando	FL	32829
Jeffrey Schwartz	1075 Beckstrom Drive	Oviedo	FL	32765
Laura Schwartz	1075 Beckstrom Drive	Oviedo	FL	32765
Joe Pastaia	4891 Kylemore Court	Palm Harbor	FL	34685
Barbara Vagenas	305 Carolyn Ave	Panama City Beach	FL	32407
Jim Vagenas	305 Carolyn Ave	Panama City Beach	FL	32407
Rebecca Sera	17136 NW 10th Street	Pembroke Pines	FL	33028
Octavio Sera	17136 NW 10th Street	Pembroke Pines	FL	33028
Larry Stewart	7891 NW 12th Street	Pembroke Pines	FL	33024
Deidre Stewart	7891 NW 12th Street	Pembroke Pines	FL	33024
Meghan Stewart	7891 NW 12th Street	Pembroke Pines	FL	33024
Allison Stewart	7891 NW 12th Street	Pembroke Pines	FL	33024
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Carlos Gomez	151 NW Willow Grove Ave	Port Saint Licie	FL	34986
Terrance Navin	540 Carillion Parkway 1016	Saint Petersburg	FL	33716
Dorinda Davis	8417 Cypress Lake Circle	Sarasota	FL	32430
Holly Roderman	174 East Riverbend Drive	Sunrise	FL	33326
Brian Roderman	174 East Riverbend Drive	Sunrise	FL	33326
Valerie Schwartz	816 SW 158th Ter	Sunrise	FL	33326
Steven Schwartz	816 SW 158th Ter	Sunrise	FL	33326
Mary Brady	816 SW 158th Ter	Sunrise	FL	33326
Joseph Turnbull	816 SW 158th Ter	Sunrise	FL	33326
Patrick Turnbull	816 SW 158th Ter	Sunrise	FL	33326
Mary Lou Brady	816 SW 158th Ter	Sunrise	FL	33326
Laura Ferency	840 South Wind Circle	Sunrise	FL	33326
Bill Ferency	840 South Wind Circle	Sunrise	FL	33326
Wendy Johnston	1404 Sturbridge Place	Tallahassee	FL	32308
David Johnston	1404 Sturbridge Place	Tallahassee	FL	32308
Martha Soder	2464 Papillion Way	Tallahassee	FL	32309
Catherine Hanks	Green Street	Tallahassee	FL	32303
Mariene Hanna	2826 Frogs Leap Way	Tallahassee	FL	32309
Jean Ainsworth	6389 Mallard Trace	Tallahassee	FL	32312
Paul Rollin	6389 Mallard Trace	Tallahassee	FL	32312
Eleanor Dietrick	640 Muriel Ct	Tallahassee	FL	30303
Jessica Stribling	3704 Carroll Wood Pl Circle 9 305	Tampa	FL	33618
Louise Huntman	1567 Apache Circle	Tavares	FL	32778
Barbara Ford	Ramblers Rest 1300 N Rivers Rd W#17	Venice	FL	34293
Blaine Payne	220 Egret Court	Weston	FL	33327
Carrie Lutenberg	3664 Heron Ridge Lane	Weston	FL	33331
Greg Lutenberg	3664 Heron Ridge Lane	Weston	FL	33331
Janine Tomlinson	7449 SE 182 Blvd	White Springs	FL	32096

Malcolm Tomlinson	7449 SE 182 Blvd	White Springs	FL	32096
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Robert Turnbull	1424 Las Cruces Drive	Winter Springs	FL	32708
Jan Turnbull	1424 Las Cruces Drive	Winter Springs	FL	32708
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Carolyn Mills	2007 A Schley Ave.	Albany	GA	31707
John and Carolyn Mills	2007 A Schley Ave.	Albany	GA	31707
Marsha Thomas	2120 Powers Ferry Road, SE, Ste. 200	Atlanta	GA	30339
Frances Lee		Columbus	GA	31909
Lucy Pfeffa	1229-B Church Street	Decatur	GA	30030
Pamela J. Rodgers	301 Charity Ave A2	Fort Valley	GA	31030
Willie E. Rodgers, Jr	301 Charity Ave A2	Fort Valley	GA	31030
LaWanna Welch	306 Humble Ave. # A-8	Fort Valley	GA	31030
David Anderson	P O Box 701	Lithonia	GA	30058
Linda Anderson	P O Box 701	Lithonia	GA	30058
C.F. Authement	307 Humble Ave	Perry	GA	31069
Doris Authement	307 Humble Ave	Perry	GA	31069
Gail Authement	307 Humble Ave	Perry	GA	31069
Nancy Woods	4079 Foorhill Avenue	St. Ansgar	IA	50472
Roger Woods	4079 Foorhill Avenue	St. Ansgar	IA	50472
Helen May	4872 Quail Drive	Staceyville	IA	50476
Susan May	4872 Quail Drive	Staceyville	IA	50476
Duane May	4872 Quail Drive	Staceyville	IA	50476
Peggy Shaw	1502 Colorado Ave	Boise	ID	83708
Vern Shaw	1502 Colorado Ave	Boise	ID	83708
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Peggy Brown	2102 Woodhavens Drive	Bloomington	IL	61701
Jennifer Prill	3511 Ballyford Drive	Bloomington	IL	61704
Santhos Valloppillil	2555 North Clark St Apt 901	Chicago	IL	60614
Nancy Malcolm	1106 S McLean St	Hudson	IL	61748
Carolyn Zembron	9328 N. Oak Park Ave.	Morton Grove	IL	60053
Andrea Kichinko	542 Wellington Ct	Mundelein	IL	60060
Janet Graeff	437 Illinois Ave Ext.	Murphysboro	IL	62966
Lacey Caraway	7 Bodkin Lane	Murphysboro	IL	62966
Cindy Traska	3601 Greenleaf Court	Spring Grove	IL	60081
Carolyn Bagley	4130 Highbury Drive	Springfield	IL	62711
Joe Bagley	4130 Highbury Drive	Springfield	IL	62711
Richard C Bagley Jr.	4130 Highbury Drive	Springfield	IL	62717
Diane Dooley	119 N. Bergan	W. Peoria	IL	61604
Lupe Martinez	119 N. Bergan	W. Peoria	IL	61604
Pat Bakanec	2710 5th Street	Winthrop Harbor	IL	60096
Dean Boswell	814 Landon Ave	Winthrop Harbor	IL	60096
Teresa M. Miller	307 Victory Hill	Coatesville	IN	46121
Chad Swagart	10580N. Portland Arch Road	Covington	IN	47932
Debra Swagart	10580N. Portland Arch Road	Covington	IN	47932
Glenn Swagart	10580N. Portland Arch Road	Covington	IN	47932
Jackie Swagart	10580N. Portland Arch Road	Covington	IN	47932
Fred Essary	403 Pfaffin Ct	Mt. Vernon	IN	47620
Sue Essary	403 Pfaffin Ct	Mt. Vernon	IN	47620

Nichole Kreuger	306 Indiana Ave #2	Valparaiso	IN	46383
Christopher Kreuger	306 Indiana Ave #2	Valparaiso	IN	46383
Renee Allen	504 Garrettson Street	Burlington	KS	66839
Carmen Landreth	1131 E. Sheridan Bridge Lane	Olathe	KS	66062
Colleen Floyd	16016 Grandview Street	Stilwell	KS	66085
Angelia Terry	7440 Vest Talcum Road	Bulan	KY	41722
William B Terry	7440 Vest Talcum Road	Bulan	KY	41722
Billy Sizemore	2019 Hwy 80	Emmalena	KY	41740
Marie Sizemore	2019 Hwy 80	Emmalena	KY	41740
Christopher D. Handshoe	2601 Reed Street	Flatwoods	KY	41139
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Salina Gibson	PO Box 49	Hindman,	KY	41822
Vivian Watts	3625 Laurel Fork Road	Hueysville	KY	41640
Ina Owens	11 Sunshine Lane	Leburn	KY	41831
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Sharon Watts Thomas	1416 Hwy 1087 East	Leburn	KY	41831
Carole Hamilton	3439 Buckhorn Drive #130	Lexington	KY	40515
Yvonne Hall	5365 Stewart Road	Lexington	KY	40516
Paul Hall	5365 Stewart Road	Lexington	KY	40516
Anna Beth Mekus	920 Cherrywood Drive	Lexington	KY	40515
Janet Blaine		Louisville	KY	40207
Bill Francis	8888 Hwy 550 East	Mousie	KY	41839
Michael Heath	105 Brentwood Drive	Winchester	KY	40391
Phil Handshoe	4355 Colby Road	Winchester	KY	40391
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Peggy Cone	6810 Maple Leaf Court Apt 201	Baltimore	MD	21209
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Jeff Herman	1304 Chalmers Road	Silver Spring	MD	20903
Herman Hamilton	8893 Water St. Rd	Walkersville	MD	21793
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Jennifer Cutts	P O Box 1212	York Harbor	ME	03911
Kathy Leslie	P O Box 1411	Ann Arbor	MI	48106
Shirley Lafontaine	12347 N Jennings Rd	Clio	MI	48420
Anthony R. Karabacz	7267 Pheasant Run	Gaylord	MI	49735
Nancy J Karabacz	7267 Pheasant Run	Gaylord	MI	49735
Tina Stewart	18014 Meridian Road	Grosse Ile	MI	48138
Lynn Hahn	20221 Island Estate Drive	Grosse Ile	MI	48138
Terry M. Hahn	20221 Island Estate Drive	Grosse Ile	MI	48138
Heidi Miklos	20450 Canal	Grosse Ile	MI	48138
Christian Hahn	8672 Lake Road	Grosse Ile	MI	48138
Jill Hahn	8672 Lake Road	Grosse Ile	MI	48138
Christy Cobb	8772 Woodside Drive	Grosse Ile	MI	48138
Don Critchfield	9135 Church Rd	Grosse Ile	MI	48138
Phyllis Critchfield	9135 Church Rd	Grosse Ile	MI	48138
Anthony G. Rich	35912 Joy Rd.	Livonia	MI	48150
Harry Turnbull	11796 s. Coleman Road	Maple City	MI	49664
Dennis Felax Sr	2236 Shore Rd	Rogers City	MI	49779
Russ Martin	665 South Lake Street	Rogers City	MI	49779
Wendy Martin	665 South Lake Street	Rogers City	MI	49779

Lisa Moscynski	1133 Cedar	Wyandotte	MI	48192
Paul Moscynski	1133 Cedar	Wyandotte	MI	48192
Zachary Moscynski	1133 Cedar	Wyandotte	MI	48192
Exequiel Bravo	5340 Emerald way	Apple Valley	MN	55124
Lois Bravo	5340 Emerald way	Apple Valley	MN	55124
Jean Johnson	338 N. 5th Street	Bayport	MN	55003
Brian Maxey	6248 Sunrise Terrace No.	Brooklyn Park	MN	55428
Roxy Maxey	6248 Sunrise Terrace No.	Brooklyn Park	MN	55428
Tony Wolynic	8609 West River Road	Brooklyn Park	MN	55444
Katrina Wolynic	8609 West River Road	Brooklyn Park	MN	55444
Delayne Landon	321 Park Street W. #301	Cannon Falls	MN	55009
Larry Pederson	1021 W. River Parkway	Champlin	MN	55316
Helen Pederson	1021 W. River Parkway	Champlin	MN	55316
Bob Sorensen	11216 Xenia Ave N	Champlin	MN	55316
Deb Sorensen	11216 Xenia Ave N	Champlin	MN	55316
Larry Urbanski	4529 Chatham Place	Col. Heights	MN	55421
Deb Urbanski	4529 Chatham Place	Col. Heights	MN	55421
Gardner Carruthers	4937 N.E. 6th Street	Col. Heights	MN	55421
Margaret Carruthers	4937 N.E. 6th Street	Col. Heights	MN	55421
Regina Wolynic	3700 Jackson St	Columbia Heights	MN	55421
Vicky Brusseau	565 51st Ave NE	Columbia Heights	MN	55421
Dan Brusseau	565 51st Ave NE	Columbia Heights	MN	55421
Dustin Wallin	11316 Palm St. N.E.	Coon Rapids	MN	55448
Chelsea Wallin	11316 Palm St. N.E.	Coon Rapids	MN	55448
Megan Casserly	6718 Valley Place	Crystal	MN	55427
Mary Olson	6718 Valley Place	Crystal	MN	55427
Jenny Olson	6718 Valley Place	Crystal	MN	55427
James Olson	6718 Valley Place	Crystal	MN	55427
Tom Lipke	4623 Tamie Ave	Eagan	MN	55123
Patrick Goral	6570 184th Street North	Forest Lake	MN	55025
Sherrie Goral	6570 184th Street North	Forest Lake	MN	55025
Russell May	603 13th Avenue NW	Kasson	MN	55944
Arlys May	603 13th Avenue NW	Kasson	MN	55944
Brad Krusemark	12827 90th place North	Maple Grove	MN	55369
Gina Krusemark	12827 90th place North	Maple Grove	MN	55369
Jan Kaehler	14800 80th Place North	Maple Grove	MN	55311
Darnell Kaehler	14800 80th Place North	Maple Grove	MN	55311
Kari Lindblad	7900 Lawndale Lane	Maple Grove	MN	55311
Julie Anderson	8676 Terrace View lane	Maple Grove	MN	55311
Tony Anderson	8676 Terrace View lane	Maple Grove	MN	55311
Clifford Shaw	13856 9th Ave. S.E.	Milaca	MN	56353
Ruby Shaw	13856 9th Ave. S.E.	Milaca	MN	56353
Virginia Krysa	1721 4th Street NE	Minneapolis	MN	55413
Lorraine Wolynic	1806 3rd Street NE	Minneapolis	MN	55418
Donald Wolynic	1806 3rd Street NE	Minneapolis	MN	55418
John D'Agostino	1918 Walden Place	Minneapolis	MN	55418
Barb D'Agostino	1918 Walden Place	Minneapolis	MN	55418
Patrica Matelsky	2109 St. Anthony Pkwy. NE	Minneapolis	MN	55418
Mike Shaw	2113 - 4th St N.E.	Minneapolis	MN	55418

Coleen Shaw	2113 - 4th St N.E.	Minneapolis	MN	55418
Mordan Shaw	2222 Benjamin St. N.E.	Minneapolis	MN	55418
Stanley Wolynic	2555 Ulysses Street NE	Minneapolis	MN	55418
Chris Wolynic	2555 Ulysses Street NE	Minneapolis	MN	55418
Marc Shaw	3605 Architect St. N.E.	Minneapolis	MN	55418
Jeanna Shaw	3605 Architect St. N.E.	Minneapolis	MN	55418
Steven Goral	333 10th Street NW, No. 212	New Brighton	MN	55112
Wayne Tembreull	2850 Zanzibar lane N.	Plymouth	MN	55447
Kathy Henkel	2850 Zanzibar lane N.	Plymouth	MN	55447
Ben Weigert	2445 Stonecrest Path NW	Prior Lake	MN	55372
Stephanie Wiegert	2445 Stonecrest Path NW	Prior Lake	MN	55372
Heather Stilwell	17576 305th Lane	Shaefer	MN	55074
Joan Gerr	4547 Churchill Street	Shoreview	MN	55126
Al Birr	8116 Fillmore St. N.E.	Spring Lake Park	MN	55432
Sandra Thorson	8116 Fillmore St. N.E.	Spring Lake Park	MN	55432
Shirley Edwards	2601 Kenzie Terrace	St. Anthony	MN	55418
William Edwards	2601 Kenzie Terrace	St. Anthony	MN	55418
Mayard Shaw	2835 Roosevelt St. N.E.	St. Anthony	MN	55418
Elizabeth Shaw	2835 Roosevelt St. N.E.	St. Anthony	MN	55418
Shirley Cashin	3517 Chalmersford Road	St. Anthony	MN	55418
Joan Seabold	360 Lexington Parkway	St. Paul	MN	55105
Mark Meyer	360 Lexington Parkway	St. Paul	MN	55105
Doloros Irwin	4027 Whitebear Parkway	White Bear	MN	55110
Gayle Chandler	9048 Duckwood Trail	Woodbury	MN	55125
Tom Chandler	9048 Duckwood Trail	Woodbury	MN	55125
Nick Frohnauer	5204 Sandstone Dr.	Columbia	MO	65202
Jen Frohnauer	5204 Sandstone Dr.	Columbia	MO	65202
Jackie Landreth	149 Canyon Forest Way	Kimberling City	MO	65686
David Landreth	149 Canyon Forest Way	Kimberling City	MO	65686
Hazel Harrison	454 Farmers Lane	Lebanon	MO	65536
Craig Kozicki	10 Robert Johns Way	St. Charles	MO	63303
Emily Kozicki	10 Robert Johns Way	St. Charles	MO	63303
Shelly Kozicki	10 Robert Johns Way	St. Charles	MO	63303
Yvonne Rousse	10 Robert Johns Way	St. Charles	MO	63303
Brent Skramstad	925 2nd Street North	Havre	MT	59501
Tanis Hernandez	214 E. 3rd Street	Libby	MT	59923
G. Benefield	245 Cedar Meadow Road	Libby	MT	59923
Norita Skramstad	3647 Highway 2 Sourt	Libby	MT	59923
Myra Cole	E. Lincoln Blvd	Libby	MT	59923
Kimberly Rowse	214 East 3rd Street	Libby	MT	59923
Dorothy Ansell	2816 Maple Ave	Burlington	NC	27215
Wanda Jumper	51 Toshas Way	Fletcher	NC	28732
Karen Hammons	17240 Cabarrus Road	Midland	NC	28107
Barbara Bedwell	4012 Mardela Spring Drive	Raleigh	NC	27616
Joan Curry,	9131 Anson Way	Raleigh	NC	27615
Patricia Glatz	204 Woodland Dr	Swansboro	NC	28584
Susan Sehorn	74 Taylor Terrace	Swansboro	NC	28584
Arthur I. Zygielbaum	6602 Pinecrest Dr.	Lincoln	NE	68516
Carole H. Carlo	8 Bunting Drive	Chesterfield	NJ	08515

Donna Carlo	8 Bunting Drive	Chesterfield	NJ	08515
Susan Perez	246 Edgar Place 2D	Elizabeth	NJ	07202
Julie Chen	189 Kemper Court	Hackettstown	NJ	07840
John Bartolomeo	62 Ivy Hill Road	Lakewood	NJ	08701
Gail Mattsson	79 Sunrise Court	Lakewood	NJ	08701
Robert Mattsson	79 Sunrise Court	Lakewood	NJ	08701
David E. Cutts	81 Sunrise Court	Lakewood	NJ	08701
Bonnie Diana	3303 Rio Vista Drive	Mahwah	NJ	07430
Kate Diana	3303 Rio Vista Drive	Mahwah	NJ	07430
Charlene Komar Storey	318 Pershing Ave.	Roselle Park	NJ	07204
Gregory D. Storey	318 Pershing Ave.	Roselle Park	NJ	07204
Elizabeth Toner	102 Rayewood Drive	Wantage	NJ	07461
Lawrence Bamdas	12 Pine Terrace	Wayne	NJ	07470
Olga Diana	1211 Richmond Rd	West Milford	NJ	07480
Margy Urnberg	3760 Meadow Wood Road	Carson City	NV	89703
Paul Urnberg	3760 Meadow Wood Road	Carson City	NV	89703
Stella (Moran) Stephens	4591 Wagon Wheel Rd.	Carson City	NV	89703
Will Smith	4601 Wagon Wheel Road	Carson City	NV	89703
Kimberly Smith	4601 Wagon Wheel Road	Carson City	NV	89703
Sandy May	31-28 47th Street	Astoria	NY	11103
David Merkrebs	9 Lake Drive	Hewlett	NY	11557
Randy Merkrebs	9 Lake Drive	Hewlett	NY	11557
Kristina Graff	34-20 74th Street, Apt 3B	Jackson Heights	NY	11372
Joseph Collins	34-20 74th Street, Apt 3B	Jackson Heights	NY	11372
Linda Turpin	6773 Rapids Road	Lockport	NY	14094
John Wang	1230 York Ave Box 92A	New York	NY	10021
Cory Crayn	170 West 23rd Street #4T	New York	NY	10011
Cecile Miller	184 W 134th Street 3	New York	NY	10030
Lisa Labrado	220 East 57th Street No. 15k	New York	NY	10022
Sheila Zachman	276 West 11th Street, Apt. 1	New York	NY	10014
Trina Semorile	445 West 46 Street, #1E	New York	NY	10036
Alexis Feldman	75 Chambers Street, No. 3	New York	NY	10007
Christopher Leahy	75 Chambers Street, No. 3	New York	NY	10007
Alexis Ufland	9 Barrow Street	New York	NY	10014
David Beanning	99 East 4th street Apt 3C	New York	NY	10003
Mary Kahal	6042 Lockard Ave.	Cincinnati	OH	45230
Lisa H Corsale		Columbus	OH	43085
Garry Handshoe	434 1/2 South Broadway	Greenville	OH	45331
Jayne Hall	173 Beau Chemin	Louisville	OH	44641
S. T. Adkins	53 E. Whitney Ave.	Shelby	OH	44875
Travis Inga	7500 N Oakcliff Drive	Tulsa	OK	74126
Bobbi Inga	7500 N Oakcliff Drive	Tulsa	OK	74126
Robert Waxman	289 York Blvd	Hamilton	Ontario	
Chester Waxman	289 York Blvd	Hamilton	Ontario	
Katie Vaughan	900 State St Box A227	Salem	OR	97301
Teresa Findlay	6262 Hubbard Creek Rd	Umpqua	OR	97430
Daniel McCormick	130 Tennis Ave	Ambler	PA	19002
Sharon McCormick	130 Tennis Ave	Ambler	PA	19002
Jill Adaman	234 S. Bryn Mawr Ave	Bren Mawr	PA	19010

Caesar Augustus	112 Hasian Lane	Coatesville	PA	19320
Jeanne Singer	2202 Rankintown Road	Finleyville	PA	15332
Joel Steinberg	1332 Wright Drive	Huntington Valley	PA	19006
Pat Karpowicz	717 Willow Street	Lansdale	PA	19446
Susan Foehl	79 Line Road	Malvern	PA	19355
June E. Breit, RN, BSN, CCM	8 Pawlings Circle	Phoenixville	PA	19460
Mark Shanaway	10419 Lindberg Ave	Pittsburg	PA	15235
Polly Boore	RR2 Box 601	Tyrone	PA	16686
Joanne Caulfield	1533 Meadowbrook Lane	West Chester	PA	19380
Patrick McBride	20 South New Street	West Chester	PA	19380
Carolyn S McDowell	1146 Brookside Drive	Hanahan	SC	29406
Byron Roach	1211 Hawthorne Road	Hanahan	SC	29406
Karen Roach	1211 Hawthorne Road	Hanahan	SC	29406
Sylvia Chennault	1232 Hawthorne Circle	Hanahan	SC	29406
Jeanette Tarr	2812 Hume Ave	Hanahan	SC	29410
Randolph Wicker	113 Miller Springs Drive	Moore	SC	29369
Mabel Wicker	113 Miller Springs Drive	Moore	SC	29369
Stanley Jones	2561 Fassitt Road Apt C-13	North Charleston	SC	29406
Ellen Walker	4969 Centre Pointe Drive, Suite 200	North Charleston	SC	29418
Pat Peterson	3700 S. Westport Ave #3590	Suix Falls	SD	57106
Russ Peterson	3700 S. Westport Ave #3590	Suix Falls	SD	57106
Melinda L Robinson	200 Oxford Drive	Franklin	TN	37064
Diane K. Walter	221 Springpark Drive	Arlington	TX	76014
Wilbur F. Walter	221 Springpark Drive	Arlington	TX	76014
Jason L. Walter	5804 Falconcrest	Arlington	TX	76017
Martin T. Murray	1500 N Alexander Dr. #1	Baytown	TX	77520
Jonathon Dekle	223 South Rosemont	Dallas	TX	75208
Tara Dekle	223 South Rosemont	Dallas	TX	75208
Susan Canon	2394 CR302	Floresville	TX	78114
Kirby Goldsberry	2629 Timberhaven Drive	Flower Mound	TX	75028
Marcus Lewis	2929 Socrates Drive	Grand Prairie	TX	75052
Tami L. Lewis	2929 Socrates Drive	Grand Prairie	TX	75052
Tom Kendle	2904 Crest Haven Drive	Grapevine	TX	76051
Amy Hall	16415 Crossfield Drive	Houston	TX	77095
Greg Hall	16415 Crossfield Drive	Houston	TX	77095
Laura Martinez	9223 Red Castle Lane	Humble	TX	77396
Bud Vaughn	1415 Bayshore Drive	Kemah	TX	77565
Jill Vaughn	1415 Bayshore Drive	Kemah	TX	77565
Jack Ramsey	5 Bethpage Dr	Laguna Vista	TX	78578
Joan Herum	5 Bethpage Dr	Laguna Vista	TX	78578
Paul Zeugin	2732 Dunbar DR	McKinney	TX	75070
Chad Medlin	4341 Waskom	Plano	TX	75024
Crystal Medlin	4341 Waskom	Plano	TX	75024
Lee Hurtado	162 Waxwood Lane	San Antonio	TX	78216
Chuck Every	1310 Kloecker Rd	Sealy	TX	77474
Karen Every	1310 Kloecker Rd	Sealy	TX	77474
Dirk Cox	1300 Shelby Court	Wylie	TX	75098
Anna Marie Murray	1004 Timber Ridge Court	Chesapeake	VA	23322
Risha Cross	1223 Woods Way	Chesapeake	VA	23323

Christine Smith Oxford	43428 Westchester Square	Leesburg	VA	20176
Edna E. Wright	3671 Bell Street	Norfolk	VA	23513
Ellen Whitehurst	2224 Kendall Street	Virginia Beach	VA	23451
Erin Baker	5825 Ludington Drive	Virginia Beach	VA	23464
Grant Baker	5825 Ludington Drive	Virginia Beach	VA	23464
Mary Charlotte Boyette	27443 Buckhorn Drive	Windsor	VA	23487
Donna Greenup	P O Box 84	Colbert	WA	99005
Pam Johnson	4608-B 164th Street SW	Lynnwood	WA	98087
Roy Bell	8028 186th Ave. SW	Rochester	WA	98579
Virgie Bell	8028 186th Ave. SW	Rochester	WA	98579
Rebecca Jones	4743 - 48th Ave. N.E.	Seattle	WA	98105
Chisholm Jones	4743 - 48th Ave. N.E.	Seattle	WA	98105
Lynn A DeBeal	5023 42nd Ave S	Seattle	WA	98118
Lynda Hanley-Cole	3007 W. 17th Ave	Spokane	WA	99224
Carla Groce		Vancouver	WA	98660
Richard Bruder	1349-A 105th ave	Amery	WI	54001
Judy Bruder	1349-A 105th ave	Amery	WI	54001
Diana Dicosimo	524 Birchwood Ave	Amery	WI	54001
Jerry Johnson	649 70th Ave	Amery	WI	54001
Marcia Johnson	649 70th Ave	Amery	WI	54001
Stacy Dosch	738 85th	Amery	WI	54001
Donovan Dosch	959 Vijobi Trail	Amery	WI	54001
Peggy Dosch	959 Vijobi Trail	Amery	WI	54001
Don Johnson	319 E. River Ave	Barron	WI	54812
Craig Allram	284 6th Avenue	Clayton	WI	54004
Cindy Allram	284 6th Avenue	Clayton	WI	54004
Hillary Arcand	677 1st street	Clayton	WI	54004
Shannon Arcand	677 1st street	Clayton	WI	54004
Trish Monson	277 75th Street	Clear Lake	WI	54005
Andrew Monson	277 75th Street	Clear Lake	WI	54005
Jan Monson	277 75th Street	Clear Lake	WI	54005
Ashley Kehoe	277 75th Street	Clear Lake	WI	54005
Donald Dosch	279 75th street	Clear Lake	WI	54005
Artie Dosch	279 75th street	Clear Lake	WI	54005
Doug Allram	387 60th Avenue	Clear Lake	WI	54005
Louann Allram	387 60th Avenue	Clear Lake	WI	54005
Amy Wienke	764 30th ave	Clear Lake	WI	54005
Ben Wienke	764 30th ave	Clear Lake	WI	54005
Wendy Stoeckler	W5936 Lee Drive	Fort Atkinson	WI	53538
Steve Dosch	13501 N Refuge RD.	Grantsburg	WI	54840
Linda Dosch	13501 N Refuge RD.	Grantsburg	WI	54840
Julie Everson	1203 Red Oak Road	Hudson	WI	54016
John Everson	1203 Red Oak Road	Hudson	WI	54016
Roberta Pabich	N9899 County Road K	Loyal	WI	54446
Jesse Gerhardt	14 Glen Hwy	Madison	WI	53705
Daniel Gerhardt	14 Glen Hwy	Madison	WI	53705
Linda Commer	1700 East Chateau Place	Milwaukee	WI	53217
Jan Spangler	2844 N. Hackett Ave	Milwaukee	WI	53211
Jean Eisenman	2844 N. Hackett Ave	Milwaukee	WI	53211

Noel Spangler	2844 N. Hackett Ave	Milwaukee	WI	53211
Shawn Bird	1008 192nd ave	New Richmond	WI	54017
Shawn Bird	1008 192nd ave	New Richmond	WI	54017
Michelle Holland	538 Park Veidrive	New Richmond	WI	54017
Michelle Holland	538 Park Veidrive	New Richmond	WI	54017
Mike Jackelen	1680 County Rd T	New Richmond	WI	54017
Bonnie Jackelen	1680 County Rd T	New Richmond	WI	54017
Angie Johnson	358 195th Street	Osceloa	WI	54020
Dan Logan	358 195th Street	Osceloa	WI	54020
Rick Allram	299 County road F	Prairie Farm	WI	54762
Darice Allram	299 County road F	Prairie Farm	WI	54762
Kyle Pierce	W7180 Sunset Lane	Spooner	WI	54801
Kari Pierce	W7180 Sunset Lane	Spooner	WI	54801
Justin Allram	205 Jerdee Ave	Star Prairie	WI	54026
Jessica Allram	205 Jerdee Ave	Star Prairie	WI	54026
Jeff Pierce	N7865 Trego Landing Road	Trego	WI	54888
Jerad Pierce	N7865 Trego Landing Road	Trego	WI	54888
Jean Pierce	N7865 Trego Landing Road	Trego	WI	54888
Joy Pierce	N7865 Trego Landing Road	Trego	WI	54888
Janice McNitt	W5543 Cty F	Trego	WI	54888
Janice McNitt	W5543 Cty F	Trego	WI	54888
Tonia Luna	3101 N 103rd st	Wawuatosa	WI	53222
Marcello Luna	3101 N 103rd st	Wawuatosa	WI	53222

Senator BOXER. Thank you for your powerful testimony.

Senator Inhofe has asked to be recognized first, since he needs to go to a very important meeting. So Senator, the floor is yours.

Senator INHOFE. I appreciate it, Senator Boxer. I will just take a couple of minutes, I won't take the whole time.

I have a few problems with this. It is always difficult when you have a panel of scientists and you are expected to make determinations by listening to two opposing views. One thing that I noticed, Dr. Lemen, in your written testimony, you talk about, you do testify occasionally in asbestos-related litigation on behalf of the plaintiffs.

One of the problems I have is that when you get into something like this, like we have gone through with asbestos, there are big winners, and the big winners are the trial lawyers. In asbestos claims, so far it has now exceeded \$70 billion claims, and there is a remaining liability of somewhere between \$145 billion and \$200 billion. More than 70 bankruptcies have taken place, and most of the current defenders are users and not manufacturers of asbestos. That was a Rand report.

So 60 cents out of every dollar goes to the lawyers. This bothers me.

Second, and let me start with you, Dr. Wylie, if we were to count these non-asbestiform minerals as asbestos in the regulatory definition, change your definition, include them all, what would that mean in terms of the land area of the world? Put up that one chart that shows the United States. This would be, as I understand it, just the United States part, but go ahead. Do you have a percentage that you could use?

Ms. WYLIE. What is shown there in green roughly outlines the areas in the United States where amphiboles are naturally occurring. And amphiboles make up about 5 percent of the earth's crust overall. So these are extraordinarily common rock-forming minerals. These minerals, when crushed, do form elongated particles.

Senator INHOFE. I see. In his testimony, Dr. Lemen stated, I am going to read this and then I am going to ask both of you to respond to it, "Any definition of asbestos should include all respiratory asbestiform fibrous materials, including fibrous cleavage fragments that are respirable." Now, I will start with you, Dr. Wylie, to respond to that just real briefly. Because I want to get it in the record in terms of his exact quote.

Ms. WYLIE. I believe that that quote suggests that cleavage fragments are asbestiform fibers, that that is not true.

Senator INHOFE. OK. Dr. Weill, would you respond to the same quote there?

Dr. WEILL. Yes, I also agree that cleavage fragments have not been shown to be pathogenic to humans.

Senator INHOFE. As the only practicing lung physician here today, could you briefly discuss the differences in how asbestiform minerals and non-asbestiform minerals and cleavage fragments affect the human body?

Dr. WEILL. Yes, I think the large majority of cleavage fragments aren't even respirable, because of their width. They are not able to make it into the distant parts of the lung, where they do most of their damage. The physical properties of these fragments are dif-

ferent from asbestos fibers. There have been animal studies that have shown that they are not pathogenic whereas asbestos fibers clearly are in animal studies.

Also, there have been several studies, human epidemiologic studies, of thousands of workers exposed to these fragments demonstrating no disease.

Senator INHOFE. I think that is extremely significant. What I would like to ask you to do for the record is to elaborate on that, showing the studies by name, where they were conducted, who was involved, so that we will have that in the record, not today but for the written record, if you would be good enough to do that.

Dr. Wylie, you made a statement concerning the mining, that it has not been such a case found after a period of time, those who work in the mines. I think that was refuted by Dr. Lemen. Would you like to have a chance to refute the refutation?

Ms. WYLIE. I am not a medical scientist. But as I read the studies, I find no excess of asbestos-related diseases. There are in some of these studies some excesses in lung cancer. But there are other compounding variables, such as the smoking history of the workers, radon daughters that can reasonably account for these excesses in lung cancer.

I know of no cases of mesothelioma associated with exposure to cleavage fragments.

Senator INHOFE. Thank you very much. Thank you, Madam Chairman.

Senator BOXER. Yes, Senator, thank you very much.

I am going to ask Dr. Lemen and any others, Dr. Castleman, to respond to this. I just want to say for the record that Senator Isakson and Senator Murray are working on some of these definitions, too. But if you would like to respond, Dr. Lemen, to the other two who challenged your point.

Mr. LEMEN. I agree with Dr. Weill, yes, with Dr. Weill, that some of the cleavage fragments will not get into the lung. It is not those that we are concerned about. What we are concerned about are the respirable ones.

Senator BOXER. Right.

Mr. LEMEN. And the respirable ones can get into the lung. They do have the same mineralogical characteristics as asbestos. We are concerned about what gets into the lung that can cause disease.

As far as the gold mine that was talked about, as I said in my brief comments, when you look at the latency, you do find, in two different studies, both the study that NIOSH conducted and the study that the McDonalds conducted, that after a long latency in the higher exposed groups, you do see an excess of respiratory cancer as well as respiratory disease.

So finally, the animal studies that have been conducted are basically negative. However, there are some cellular studies that have shown cellular reaction with these types of small, short cleavage-type fibers.

Senator BOXER. You are saying that if those fibers get loose, that is a problem?

Mr. LEMEN. That is right.

Senator BOXER. So I don't know that there is any disagreement whatsoever here. I think that is a phony kind of distinction without

a difference. If they break off, and they are inhalable, I am sure both Dr. Weill and Dr. Wylie would agree, if they are inhaled, they are a danger, is that correct?

Dr. WEILL. Inhaled and reach the distant parts of the lung?

Senator BOXER. Yes.

Dr. WEILL. No, they are—

Senator BOXER. They are not a danger?

Dr. WEILL. No, the chemical properties may be similar between asbestos fibers and cleavage fragments. But their physical morphology is different and the body can handle them differently—

Senator BOXER. This is important, because NIOSH disagrees with you, sir.

Dr. WEILL. I understand that.

Senator BOXER. NIOSH believes that durable inhalable fibers with characteristics similar to asbestos should be considered potentially harmful. Exposure to these fibers should be avoided if possible or otherwise minimized through standard industrial hygiene practices.

I am going to move on. I wanted to ask the Capitol workers here, who we know are exposed to asbestos, if they would stand up, just to be recognized by the audience, if they would stand up. The reason I want to ask you to stand is because I want you to know that all of us are very determined to make sure that your problem, (a) has been stopped, in other words, there is no more exposure; and (b) if there was exposure, which you I think were informed there was, we are going to stand with you on this. I just want to thank you very much for coming.

I want to get to a couple of other things, and you can start my clock now at 5 minutes and I will just come back to it.

Dr. Weill, describe the health effects of asbestos. Have you ever treated or personally evaluated a patient who had asbestos-related disease?

Dr. WEILL. Yes. The health effects of asbestos, as I mentioned in my testimony, can include both malignant and non-malignant diseases. I have—

Senator BOXER. How many patients have you personally evaluated and treated?

Dr. WEILL. Somewhere in the neighborhood of 50 to 100, I would say, with true asbestos-related disease.

Senator BOXER. OK. Did you co-author a commentary on the American Thoracic Society's statement on the diagnosis and initial management of non-malignant disease related to asbestos?

Dr. WEILL. Yes, I did.

Senator BOXER. Did the Society point out that your commentary cited a 1993 study by William Weiss to make a point which the study specifically stated is not the question considered in this review?

Dr. WEILL. I am not certain I understand your question.

Senator BOXER. OK, well, this is—are you aware that the Society pointed out in response to your story that your commentary cited in a 1993 study by William Weiss, which the study specifically stated is not the question considered in this review, did you hear from the Society on this point?

Dr. WEILL. Yes, I did.

Senator BOXER. Thank you.

Have you worked for businesses that make money selling products that may have caused lung disease?

Dr. WEILL. I have been retained by lawyers who represent these companies.

Senator BOXER. Well, thank you for your honesty in answering this question, because I believe it is important that this information be so stated in the record.

Now, Dr. Wylie, are you a doctor, are you a geologist or do you treat patients?

Ms. WYLIE. I am a geologist.

Senator BOXER. OK. Then do you agree with the statement by the U.S. Geological Survey, "It is absolutely not the role of the analytical or mineralogical communities to make health-based decisions or to make independent analytical assessments that directly or indirectly influence health-based outcomes"? Do you agree with that statement?

Ms. WYLIE. I am not sure that I do. I think that is the role of mineralogists to make clear the nature of the materials to which people are exposed. And in that regard, it is an independent analytical assessment that might indirectly influence the outcomes of some studies. But it is only our job to tell about the materials, what they are like—

Senator BOXER. Well, that is not what you did. I find aspects of your testimony troubling, including your statement, not only is the width of asbestos a defining characteristic, it is the key to its carcinogenicity. That you are stepping into another field that your own profession says you should avoid.

So I am rather shocked by your statement. I have another question. Have you worked for business that makes money selling products that may have caused disease associated with asbestos?

Ms. WYLIE. No.

Senator BOXER. Well, I have a number of receipts that show you have worked as a paid defense witness for business in asbestos litigation. I ask unanimous consent that these documents be placed into the record.

[The referenced documents are retained in the committee's file.]

Senator BOXER. Why didn't you answer my question honestly?

Ms. WYLIE. I did. I have never worked for an asbestos manufacturer.

Senator BOXER. I didn't say that.

Ms. WYLIE. Or an asbestos fabricator.

Senator BOXER. I didn't ask you that. I said, have you worked as a paid defense witness for a business in asbestos litigation?

Ms. WYLIE. I have testified on about three occasions for, on the nature of materials involved—

Senator BOXER. Who paid you?

Ms. WYLIE. R.T. Vanderbilt, three times or thereabouts.

Senator BOXER. So your original answer was incorrect?

Ms. WYLIE. I misunderstood—

Senator BOXER. Well, let me be clear. I think it is very important that we be totally honest before this committee.

Ms. WYLIE. I agree.

Senator BOXER. Senator Lautenberg.

Senator LAUTENBERG. I am a little bit astonished to say the least at what we hear from two of our witnesses, Dr. Weill and Dr. Wylie, in terms of the contradictory nature of your views and those for instance, Dr. Lemen's presentation. The 1998 WHO statement, consistent with their early conclusions, 1989, human evidence has not demonstrated there is any threshold exposure level for lung cancer or mesothelioma below which exposure to asbestos dust would not be free of hazard to health. Do you disagree with that conclusion, Dr. Weill?

Dr. WEILL. I think I would just state it differently. I think it is very difficult scientifically to render something "safe." I think all we can do is estimate the risk as best we can and try to determine, and this is more of a public policy question, how much risk is tolerable. I think it is very difficult to say something is safe, whether it be air travel, water, asbestos fibers—

Senator LAUTENBERG. Those comparisons are not valid, air travel, that—you are not risking exposure when you get in an airplane that is commonly thought to be a dangerous exercise.

Dr. WEILL. No, but I think my point really was, and maybe it wasn't a perfect analogy, was that all science can do really is estimate risk. It can't render something safe or unsafe. Because the circumstances that somebody is exposed to something differs, what they are exposed to differs. I think that is why we have to really rely on the scientific evidence to assign risk to these different exposures.

Senator LAUTENBERG. Is mesothelioma directly connected with asbestos exposure or are there other exposures?

Dr. WEILL. There are other causes of mesothelioma that are very uncommon.

Senator LAUTENBERG. Dr. Lemen, is mesothelioma typically a result of exposure to asbestos, the larger share, let's say, of cases that we see?

Mr. LEMEN. Yes. And there are some other causes, they are very small, related to asbestos. But in man, about 80 percent of the mesotheliomas have been related to exposure to asbestos. That is somewhat less in women, because we just don't have good surveillance data on women. But when I was at NIOSH, we put out a paper and we titled mesothelioma as a signal tumor. That is, once you see the disease, look closely to see if there is any asbestos exposure. Because in almost all cases, there is some exposure to asbestos.

Senator LAUTENBERG. So Dr. Weill, I get the suggestion from you that we are just alarmists with our concerns about this, and that it is not, the threats are not really what we think we are talking about here?

Dr. WEILL. No, Senator, I am sorry if I left that impression. I don't think you are alarmist at all. I think, though, what is important, particularly as it relates to asbestos, given the long history of looking at this disease, both scientifically and in the public policy arena, is that we rely on the science. We keep coming back to risk assessment and not just tend to lump everything together without regard to the scientific evidence that is available.

Senator LAUTENBERG. I ask each of you again, Dr. Wylie and Dr. Weill, and the Chairwoman asked you about whether or not you

have testified on behalf of companies, helping to protect companies that are facing lawsuits. You both agreed that you have testified in those cases.

Now, who brings these suits? Are these people who are sick, people who are at risk from exposure to asbestos?

Dr. WEILL. As you can imagine, in litigation, some are sick and some are not.

Senator LAUTENBERG. But have you, are you familiar with the condition of your physician and the condition of the people who are bringing this suit who were trying to prove that they were sick? Were you invited to examine these people?

Dr. WEILL. In some instances?

Senator LAUTENBERG. And you found that, you testified that they weren't really sick?

Dr. WEILL. Sometimes yes, sometimes no.

Senator LAUTENBERG. Even though other physicians said they were sick?

Dr. WEILL. There has been disagreement.

Senator LAUTENBERG. Dr. Lemen, where do we get differences like this? How is that possible? Are you so blindsided that you think these things really relate to exposure to asbestos cleavage in particular, et cetera?

Mr. LEMEN. I believe, and I have been in this field for a long time, that when you have a respirable fiber, and if you look at the issues about fiber size, length and diameter, we see that these types of fibers get into the lung, are capable of causing damage. So I pointed out a particular facility, R.T. Vanderbilt facility, where our agency went into in the 1970s, where the company claimed that they were having a non-asbestiform talc. But when we went in, we actually found two types of asbestos fibers in that talc, and they were above the OSHA and MSHA risk.

So it lies a lot in the definition. As I say in my extended testimony, a lot of this depends upon getting a good definition. I would agree with all the panelists here that we need to get a good definition and come to some conclusion amongst ourselves of what that definition is. But as a health scientist, I am concerned when fibers get into the lung and stay in the lung and have the characteristics of asbestos fibers that they can cause damage. And that is where I am coming from and have been from that point of view for the whole time that I was with the Federal Government, 26 years. I still believe that.

I would like to say one thing about the lawsuit. I think that there is a lot of concern about frivolous lawsuits. But there are a lot of real lawsuits. It was the real lawsuits that brought the attention of the asbestos issue, your friend, Dr. Selikoff, that I had the privilege of working with for many years, brought this to the attention when the Occupational Safety and Health Act passed in 1970, and asbestos was a major issue because the companies were not doing their part to prevent these diseases. I would end with that.

Senator LAUTENBERG. I thank you very much, Madam Chairman, for having this hearing. I would ask that the record be kept open so that we have a chance to review in a little more detail the differences that we see, the testimony differences. I am of the view that with the exposure from my high school days and friends that

I know and people who worked in New Jersey had a lot of work on Johns Manville in the Raybestos Manhattan, the shipyards and the whole thing.

So thank you to all of the witnesses. Madam Chairman, that concludes my questions. Thank you for permitting me the extra time.

Senator BOXER. Always happy to. And I think that gets really back to the heart of the matter I was trying to get at. I have a couple more points.

I think the point that Senator Inhofe, whom I have great respect for, my Ranking Member, and my good friend, when he says that this is all about the trial lawyers, I don't know what he is thinking. We want to ban asbestos. That would put the trial lawyers out of business at the end of the day. So let's get it straight. We are going to ban it, at least in this committee, and we are going to get the ball rolling. That is going to put the trial lawyers out of business eventually. So that is point No. 1.

Dr. WEILL. You said it is an issue of how much risk is tolerable, which is something I hear a lot from people who always defend the folks who are pushing poison on the public, if you will. So how much risk is tolerable to you? Are you married with a family?

Dr. WEILL. Yes.

Senator BOXER. Is it tolerable for your child to get mesothelioma? Would that be tolerable for you, sir?

Dr. WEILL. Of course not. And—

Senator BOXER. Would it be tolerable if you knew the company knew they could use an alternative but yet you came home and you had asbestos on your clothes, like one of these guys might have done, and your child got close to you and breathed it in, is that tolerable? Would that be tolerable to you, sir?

Dr. WEILL. I don't understand the specific—

Senator BOXER. Would it be tolerable to you if you worked in a place where you were exposed to asbestos, the kind you admit is dangerous, and your child breathed it in, and pretty soon she or he had some kind of asbestos-related disease and could die from it, would that be tolerable to you?

Dr. WEILL. No. If there is amphibole asbestos in a dose that is important, that would not be tolerable.

Senator BOXER. So you would support banning this product, I assume?

Dr. WEILL. By banning it, we would have to define what we are talking about in terms of the—

Senator BOXER. Banning a product that was dangerous, you wouldn't have objection to that?

Dr. WEILL. Products that are dangerous shouldn't—

Senator BOXER. You would support that?

Dr. WEILL. Absolutely.

Senator BOXER. Because I don't know what—that sort of contradicts what you said before, how much risk is tolerable, which indicates to me that some of the risk is tolerable. So I am trying to ask you, how many people a year could die from mesothelioma, and it would be tolerable to you, sir?

Dr. WEILL. I answered the question, though, to look at a certain type of asbestos in a certain dose. That is—

Senator BOXER. Well, I am asking you a different question. I am asking you, how many people dead every year from mesothelioma would you consider tolerable?

Dr. WEILL. I would hope that none would die from mesothelioma. It is a personal tragedy for the families—

Senator BOXER. So no deaths are tolerable?

Dr. WEILL. That is right.

Senator BOXER. So that contradicts what you said, how much risk is tolerable.

Dr. WEILL. No, we are not saying that deaths are tolerable. We are saying that risk assessments are a sign in the scientific literature for a variety of things.

Senator BOXER. Sir, what if the risk is 1 per 300, 1 per 1,000, 1 per 500,000? What is tolerable? You started this. You said before it is a question of how much risk is tolerable.

Dr. WEILL. And I—

Senator BOXER. You know what risk benefit means. Some people die. What is tolerable?

Dr. WEILL. I think that is a public policy question, not a question—

Senator BOXER. Oh, OK, so you are ducking it. So you don't sit here and tell me, it is a question of how much risk is tolerable and then refuse to answer it, because that is wrong. That is just saying, I can testify in front of any court and then I can say, well, my Senator, Barbara Boxer, I am your Senator, oh, God, I think I lost a vote here—

[Laughter.]

Dr. WEILL. I have a very open mind about that, Senator.

Senator BOXER. About voting for me?

Dr. WEILL. Sure.

Senator BOXER. OK. It seems to me, if you are going to say, it is a question of how much risk is tolerable, you have to then be prepared to answer the question. Because let me tell you what is intolerable, I agree with what you said, any death, whether it is your kid, my kid or any kid or any worker or anybody. And here we have Linda Reinstein sitting here, having lived through this experience with her husband. And here we have, you know, deaths going down? Doesn't look that way, sir. But you can take a look at it. It looks like we have lost 10,000, and this is under-reported, from NIOSH, they admit it is under-reported, 10,000 since 1999, 10,000 dead. Close your eyes and think about 10,000 families.

In my case, I raised my kids in Marin County. And a lot of those towns just had 11,000 people. So just think about what that means.

So I agree with what you said, it is intolerable to lose anyone. I don't agree with saying, well, it's a public policy decision as to how much risk. I think everyone is responsible, if you are in this game and you are in the game, you have money in the game, you have to be prepared to tell me how much risk is tolerable.

Was it worth Ms. Reinstein losing her husband? And maybe she will want to talk about—do you have a daughter? Do you want to talk about what it is like, to tell people who go and testify on behalf of the industry, please tell us what it is like.

Ms. REINSTEIN. Are you asking me to tell you?

Senator BOXER. Yes, I am asking you to please tell us what it was like for your child.

Ms. REINSTEIN. I think it is really important for the record and I thank you for asking. Because there are hundreds of thousands of women just like myself, we go from wife to widow. Our children are raised by single parents. Emily walked Alan's oxygen around the house. He struggled for a year with chemotherapy. I stayed home lovingly to walk my husband to the bathroom, he was too weak. He was a brilliant businessman, a mountain climber and a marathoner who died a shell of a man who weighed 135 pounds. Emily stood there over her father's body as he gasped for his last breath. He got to her bat mitzvah, he died 5 months later.

None of these deaths are tolerable. And the victims and the families want a ban and education. It is heartbreaking, Senator Boxer. It really is painful.

Senator BOXER. Let me just thank you very much for that.

I know I sounded harsh. And I feel concerned that people have talked themselves into a position where they are part of the problem and don't see it. It is not right. I don't think geologists should talk about what causes cancer. I don't think the USGS said they should and I don't think they should appear before here and do it and I don't think they should deny they got paid until they are reminded. Call me old-fashioned, I don't think it is ethical. I don't.

I will just say this. The facts are in. We are going to have a bill. It is going to ban asbestos. We are going to do that. We are not going to allow this moment, this opportunity, to pass us by. Because if we do, we are part of the problem, Senator Lautenberg and I. We don't want to be part of the problem. I can't speak for other Senators, except I know Senator Isakson wants to be part of the solution.

So let me just say to all the panelists, whatever side you are on, that I appreciate the fact that you came here. I know it isn't easy. There are some withering questions sometimes. I know it is emotional. I saw people in the audience with tears, and I know that Linda is fighting them back at the moment.

But just think about what it will mean, the memory of your husband, when we get this signed, thanks to Patty Murray's bill, thanks to Senator Isakson for working with her. And thanks to the happenstance that I am holding the gavel. This is good. These are good things. And we can spare other people what you went through. And I will give it all I have.

So I want to thank everybody on all the panels. Again, the Capitol workers who are here as a reminder that this is hitting right close to our home right here, to our family right here at the Capitol.

We stand adjourned, and hopefully we will be marking this bill, Senator Lautenberg, in the near future. We stand adjourned.

[Whereupon, at 12:18 p.m., the committee was adjourned.]

[Additional statement submitted for the record follows.]

STATEMENT OF HON. MAX BAUCUS, U.S. SENATOR FROM THE STATE OF MONTANA

After the field hearing this committee held in Libby, Montana, this last April, an outraged constituent approached me to talk about asbestos. He didn't want to talk about WR Grace's disgraceful history of poisoning the town of Libby. Nor did he want to discuss the ongoing EPA cleanup in Libby. What outraged him, and rightly

so, was that given all the dangers of asbestos, the heartache it has causes thousands of families throughout the country, and the over 200 confirmed asbestos disease related deaths in Libby alone, the United States continues to use over 2000 metric tons of asbestos every year. We here on the committee ought to share that constituent's outrage.

Madame Chairman, I want to thank you for calling this hearing. It is beyond comprehension that after years of studies and thousands of deaths we are still fighting to ban this deadly substance. It is an affront to all those who have suffered throughout the country to not learn the lessons from places like Libby.

Libby epitomizes what happens to a town devastated by the health effects of asbestos. As I mentioned previously, there have been over 200 confirmed deaths due to asbestos exposure in Libby. And it is not only the former employees of WR Grace that have been victims. For years miners came home with their clothes covered in the deadly fibers. The WR Grace mill spewed 5,000 pounds of asbestos into the air every day. The entire community was exposed. This resulted in "take home" and environmental exposure on a frightening scale. According to an Agency for Toxic Substances and Disease Registry, asbestosis mortality in Libby is 60 times higher than in the rest of the U.S.

We must learn from this tragedy and prevent asbestos exposure. We must prevent more asbestos exposure. We must do two things. First, we need to continue to fund research on asbestos and non-asbestiform structures as well as minerals such as erionite, richterite, and winchite. With a better understanding of the toxicity of these materials, we will be better able to protect public health.

Secondly, we must pass Senator Murray's "Ban Asbestos in America Act." This bill would put an end to this dangerous product that has been used for far too long. Senator Murray has been a champion of this issue, and I'm proud to have joined her as an original cosponsor of the "Ban Asbestos in America Act." This is an important piece of legislation, and I look forward to working with her to bring an end to asbestos use in America.

We must learn from history. In Libby and across the country too many lives have been devastated by asbestos related diseases to continue asbestos use in this country. It is an outrage, an affront to the victims of asbestos related disease, and we ought to put an end to it.

