Testimony of

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February 2, 2005

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On Asbestos Related Diseases and Other Dust Diseases

Before the Senate Judiciary Committee

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Chairman Specter, Senator Leahy and members of the committee, I want to thank you for the opportunity to appear before the committee to testify on asbestos-related diseases and other dust diseases. I had the honor of testifying before this committee in June 2003 on the medical and diagnostic criteria for asbestos related diseases, and appreciate the chance to again assist in the development of legislation to establish a trust fund to compensate workers with asbestos-related diseases.

I am a physician with board certification in both Occupational and Environmental Medicine and Internal Medicine. I received my medical degree from the State University of New York at Stony Brook, and have held faculty positions at the Schools of Medicine at Albert Einstein, Yale and George Washington Universities. I have extensive experience in diagnosis and treatment of asbestos-related diseases. I have been in occupational medicine practice for over 20 years, and a substantial part of my practice has always been devoted to examination of workers exposed to asbestos.

In addition, I have many years of experience in medical surveillance programs for asbestos. Since 1987 I have been the medical advisor to the Sheet Metal Occupational Health Institute Trust, a joint labor-management organization within the sheet metal industry established to provide medical examinations for sheet metal workers exposed to asbestos and other respiratory hazards. To date, SMOHIT has provided medical examinations to over 30,000 sheet metal workers, and is now the largest epidemiological database of asbestos-exposed workers in the country. I also developed similar medical screening programs for the Laborers National Health and Safety Fund and other construction trades, in conjunction with the Occupational Health Foundation. I currently serve as medical director for a Department of Energy-funded medical screening program to provide medical examinations for former construction workers at a number of former atomic weapons production facilities. In each of these programs I have designed programs to detect asbestos-related disease, and designed algorithms for the examining physicians to use in interpretation of the results. I have been active in efforts to improve validity and reliability of x-ray reading to detect asbestos related disease in the United States; this work included publication of a paper on variability between readers' classification of x-rays using the International Labor Organization Guide to Classification of Pneumoconiosis, based on an analysis of results from these screening programs.

I currently am medical director at The Center to Protect Workers Rights, a research institute devoted to improving health and safety in the construction industry. Attached as Exhibit 1 is a copy of my current curriculum vitae, which sets forth my education, training, professional affiliations, research activities and publications.

We are here today to discuss a trust fund for compensation of asbestos-related disease, and the best way to ensure that those who have asbestos-related disease are compensated fairly by that fund. The bill to establish the trust fund sets for specific criteria to confirm that the disease present is related to asbestos, and to measure the impairment due

to asbestosis in Levels I-V. The bill does not set forth diagnostic criteria for other occupational lung diseases, nor does it need to, for the criteria in the bill ensure that diseases compensated are caused by asbestos.

I understand that some have the concern that cases of asbestosis will be filed as injury attributed to other dusts such as silica either in addition to this fund, or instead of this fund. I don't think this is a problem. Asbestosis and silicosis cause a different pattern of lung injury, and can be distinguished with occupational history, pulmonary function testing, and x-ray, so a case of asbestosis can't be turned into a case of silicosis or another dust disease. Let me explain in more detail.

Differentiation of Asbestosis from Other Dust Diseases of the Lung

The hearing today addresses the differentiation of asbestosis from silicosis or other occupational lung diseases, the "mixed dust" issue, and the need for additional language in the legislation for these questions.

The term "mixed dust" has been used broadly, and in my view inappropriately. To understand the issues being raised, it is important to be clear what we are talking about.

First, there are a whole range of diseases caused by exposure to dusts. A subset of the lung diseases from dusts is called pneumoconioses; one of these pneumoconioses is asbestosis. The pneumoconioses are distinct diseases caused by exposure to different substances. Asbestosis, the focus of the legislation, is a scarring of the supporting structures of the lung, called interstitial fibrosis. Silicosis is due to the formation of nodules in the lung, on pathology quite distinct from the scarring seen in asbestosis. Coal workers' pneumoconiosis is caused by the lung's reaction to coal dust often in combination with silica, and appears as bronchitis, emphysema, and nodular lung scarring.

The textbook definition of mixed dust pneumoconiosis is lung disease caused by simultaneous exposure to crystalline silica and other dusts such as iron oxides, coal, and graphite. Asbestos exposure is not a contributor to this mixed dust pneumoconiosis.

In addition, there are many other minerals, fibers and dust agents such as beryllium, cobalt, cotton dust, and wood dust, that cause occupational lung diseases but are not included in the pneumoconioses. Each disease has its own characteristic appearance and diagnostic criteria. These agents cause different kinds of lung disease, and result is different patterns of lung injury, such as obstruction, restriction, asthma, and bronchitis. They are clearly distinct from asbestosis.

It is important to point out that as a result of exposure to multiple hazards, which is not uncommon for workers; it is possible to have two diseases, such as asbestosis plus COPD from dust exposure. The question has been raised is it possible to differentiate these diseases. The answer is yes. Asbestosis, silicosis and coal workers' pneumoconiosis (CWP) are different diseases, separable from each other based on occupational history, chest x-ray, and pulmonary function testing.

? Asbestosis primarily causes loss of lung volume, and had a characteristic pattern on chest x-ray.
? Classic silicosis and CWP, in contrast, cause primarily obstructive lung disease with decreased air flow and an increase in lung volumes, and a different characteristic pattern on chest x-ray.
? The occupational histories of workers with these diseases are clearly different.

To understand how these diseases can be distinguished, one must understand how the chest x-rays are viewed and how pulmonary function testing measures lung disease.

Brief Overview of Pulmonary Function Testing

Spirometry measures lung volume and air flow with equipment that is readily available in many physicians' offices. Spirometry is reliable and reproducible when performed according to the specifications set by the American Thoracic Society (ATS). The primary measures produced by spirometry are the forced vital capacity (FVC), the forced expiratory volume in one second (FEV1) and the ratio of the two (FEV1/FVC). FVC is a measure of lung volume. The

FEV1/FVC ratio measures how quickly that lung volume is expelled from the lung, and so measures airflow. A reduction in FVC with a normal FEV1/FVC ratio is due to loss of lung volume (restriction), while a reduction in FEV1 with a reduced FEV1/FVC is likely due to air flow obstruction.

Total lung capacity (TLC) is a more extensive test than spirometry; it also measures lung function. Determination of lung volumes can be done by the gas dilution method or by body plethysmography; both are standard measures and also are reliable and reproducible. When the disease in the lung causes restriction, the TLC is decreased. If the lung disease is one of obstruction, TLC may increase.

Overview of the International Labor Organization Classification of the Radiographic Appearance of Pneumoconioses

The International Labor Organization provides a system of grading chest x-rays for dust diseases of the lung (pneumoconiosis) that is accepted around the world. The most recent version is the 2003 Classification of the Radiographic Appearance of Pneumoconioses. It provides a standard notation, so that if one reader calls a film a "1/1" another reader will know to what the first reader is referring.

The classification uses a 12-point scale to define the degree, or severity, of increased lung markings. This scale runs from 0/- to 3/+; a "0" film is normal and a "3" film has the most severe scarring. Each reading on the scale is characterized by a number between 0 and 3, and a second number, separated from the first by "/". The first number, preceding the "/", is the final score assigned to that film by the reader. The second number, following the "/", is a qualifier. The numbers 0, 1, 2, and 3 are the main categories. An x-ray read as a category 1 film might be described as 1/0, 1/1, or 1/ 2. When the reader uses the descriptor "1/1", he is rating the film as a 1, and only considered it as a 1 film. If he uses "1/0", he is saying he rated the film as a "1", but considered calling it a "0" film before deciding it was category 1. Finally, when the reader uses "1/2", he is saying he is rating the film as a "1", but did consider calling it a "2" film. Any category "1" film is abnormal; therefore a 1/0 film in an asbestos-exposed worker is consistent with asbestosis.

The classification also uses a series of letters to denote the type and size of the scarring seen on a chest x-ray. P,Q and R means that rounded opacities are present, with P representing diameters up to 1.5 mm, Q from 1.5 to 3 mm, and R diameters between 3 and about 10 mm. (Opacities over 10 mm are describes as large opacities in a different part of the reading form). Small irregular opacities in the same size ranges are classified as S, T and U.

Differentiating Asbestosis and Other Dust Diseases Using the ILO Classification

The diagnosis of silicosis is based on an appropriate history of exposure to silica, characteristic chest x-ray findings, and the absence of other diseases that mimic silicosis. On chest x-ray, simple silicosis is characterized by rounded opacities less than 1 cm in diameter in the upper lobes of the lung (P,Q or R on ILO scale), often accompanied by characteristic calcium deposits in lymph nodes. Silicosis can progress to massive fibrosis, in which the nodules of simple silicosis appear to aggregate into masses in the lung; this is a very distinct finding of silicosis.

In contrast, on chest x-ray classic asbestosis is characterized by small irregular opacities less than 3 mm in diameter in the lower lobes of the lung (S, T on the ILO system), often accompanied by pleural scarring. Asbestosis does not cause calcification in lymph nodes nor massive fibrosis.

Determining That Impairment on PFTs Is Caused By Asbestos

Asbestosis primarily causes a reduction in lung volume, leading to a reduction in FVC and total lung capacity on pulmonary testing (restrictive disease). Asbestosis also causes reduction in diffusion capacity, and this test is included for classification for Level V in the current legislation.

Silicosis and CWP cause a reduction in air flow out of the lung (obstructive disease), and cause an increase in lung volume.

Since the general pattern of injury is different, we can establish medical criteria that largely differentiate asbestos-related diseases from other lung diseases. The ratio referred to as the FEV1/FVC ratio serves as a measure of the amount of obstructive lung disease present, and is an objective test that has been incorporated into the medical criteria for the trust fund in Levels II-V.

The differentiation of these diseases of course also depends on the occupational history, and the fund as proposed incorporates exposure to asbestos as one of the criteria for compensation. This is important, for there can be components of obstruction in asbestosis as well as components of restriction in silicosis. It is the sum total of the occupational history, PFT and chest x-ray that allow differentiation of silicosis and asbestosis, or identification of workers who have a component of injury from both.

The Medical Criteria in the Bill (Section 121) Have Been Developed to Identify and Compensate Asbestos-Related Diseases

As I stated earlier, I had the honor of working with this committee in 2003 on the development of the medical criteria in the bill. It is important to understand that the medical criteria were designed to identify and compensate individuals for asbestos-related diseases, not other dust diseases. Indeed there was great debate about what the specific criteria should be, with industry greatly concerned that the criteria not be set to result in compensation for diseases that were not related to asbestos. The x-ray requirements were designed to identify asbestosis and pleural disease caused by asbestos. And, similarly the pulmonary function test criteria were set to evaluate loss of lung function as a result of restrictive diseases. Level II of the bill does recognize that some workers with asbestosis may also have an obstructive disease caused by smoking or some other exposure. But the compensation provided to these workers - $35,000 - is only for their asbestos-related disease and is much lower than the awards for Levels III - V.

Comments on Proposed Language in the January 19, 2005 draft on Treatment of Non-Asbestos Claims

The January 19 Discussion Draft includes provisions for the treatment of Non-Asbestos claims. The provisions set conditions that must be met for any personal injury claim attributable to airborne dust, fibre, or minerals may proceed in the tort system. I am not here today to testify on the legal aspects of current tort litigation related to such exposures. However, as a medical matter, I do believe that the proposed language is seriously flawed; and that if adopted would bar workers with serious dust related diseases from seeking redress for their injuries.

Section 403(b)(1)(A)(i) and (ii) of the draft requires that to proceed with a civil suit for a disease attributable to a airborne dust, fibre or mineral that a claimant must prove that their functional impairment was not caused by exposure to asbestos. This is an impossible requirement to meet.

As a physician, I cannot swear that exposure to asbestos made no contribution to a patient's lung disease. I can make an affirmative statement, that a particular exposure was a substantial cause, even a predominate cause of a disease. But it is not possible to make the opposite statement, that an exposure had no effect. It may be the case that all exposures to asbestos elicit an inflammatory response in the lung, and lead to some scarring. It would be impossible to prove asbestos had absolutely no role, even when a physician does not diagnose asbestosis.

The medical criteria for the fund do require a physician statement that asbestos exposure is a contributing factor in causing the pulmonary condition in question and excluding other more likely causes of that pulmonary condition. This is in keeping with medical practice. Medicine focuses on diagnosis, using diagnostic criteria to reach the most probable diagnosis. The focus of medicine is to pinpoint a disease so that the right treatment can be used. It is irrelevant to medicine if there is a small contribution from a secondary exposure, for that will not change the diagnosis or the treatment.

The scope of diseases and exposures covered in the definition used in 403(b)(1)(A), "personal injury claim attributable to exposure to airborne dust, fibre, or minerals"--is very broad, and would apply to hundreds of exposures and diseases.

The term mineral encompasses over 500 substances, and includes all metals and metal compounds. Attached as exhibit 4 is a list of minerals and a list of occupational diseases recognized to be caused by minerals As you can see, this is a very extensive list, and includes silicosis, coal workers' pneumoconiosis, byssinosis from cotton dust, asthma from wood dust, nickel, flour and other organic dusts, chronic beryllium disease, hard metals disease and many others.

Under section 403, any individual suffering from one of these diseases would be barred from filing a personal injury claim unless they could prove that their impairment was not caused by exposure to asbestos, which as stated above is an impossible requirement.

Moreover, as the legislation is currently written, any person with a personal injury claim for any of these diseases, or due to any of these exposures, would have to submit the evidence required in 403(b)(2)(A), even though these diseases have nothing to do with asbestos exposure. The language could cover personal injury claims for medical malpractice, for some minerals are therapeutic drugs with significant toxicity.

The number of workers with silicosis or other occupational lung diseases other than asbestosis that also had asbestos exposure, and so might be impacted by 403(b)(2)(A), is very large.

Analysis of fiber burden in the lung in the 1980s found that asbestos could be found in the lungs of almost everyone in the American population, showing that essentially everyone had had exposure to asbestos. , . Since workers filing a personal injury claim attributable to exposure to airborne dust, fibre or minerals would have been alive in the 1980s they can be presumed to have been exposed to asbestos; the language in 403(b)(2)(A) would require submission of an x-ray and B reader report in thousands of cases, even for cases where the type of disease at issue would not need an x-ray or be medically subject to a B reader review.

The Effect of Section 403 is to Bar Thousands of Workers with Serious Diseases from Seeking Redress for their Injuries

As set forth above, Section 403 is extremely broad in its scope covering hundreds of toxic agents and dozens of different diseases. As crafted, it imposes impossible burdens on claimants, and would bar personal injury claims for many occupational diseases.

It is important to point out that most of these claimants would not have a right to compensation under the Asbestos Trust Fund, since most do not have an asbestos-related disease. Those claimants who have some asbestos disease, but predominantly another disease (e.g. silicosis) would also be greatly disadvantaged, since under the bill's medical criteria, the maximum compensation for a claimant with two such diseases would be $35,000 for disease Level II, since the bill's medical criteria for the higher levels and awards are set to identify and compensate only asbestos-related diseases.

Asbestos-Related Disease and Silicosis - Are These Diseases Still Occurring?

Although my testimony addresses issues specific to compensation of asbestos-related diseases, it is important to understand the context in which those diseases occur. Decades of uncontrolled use of asbestos, even after its hazards were known, have resulted in an occupational disease crisis both in the United States and throughout the world of monumental scope. In this country, from 1940 to 1979, 27.5 million workers were occupationally exposed to asbestos in shipyards, manufacturing operations, construction work and a wide range of other industries and occupations; 18.8 million of these had high levels of exposure. As a result hundreds of thousands of workers and their family members suffered from or died of asbestos-related cancers and lung disease, and more than a million more cases of malignant and non-malignant disease are expected. In the year 2003 alone, almost 10,000 people in the United States were expected to die from asbestos-related diseases. Because of the long lag between exposure to asbestos and the development of an asbestos related cancer or another asbestos disease, the asbestos disease epidemic is only now reaching a peak.

Groups known to be at highest risk at the time of the Nicholson report were insulators, shipyard workers (many who worked during World War II) and workers engaged in the manufacture of asbestos products. Other high- risk industries and occupations included other construction trades, railroad engine repair, utility services, stationary engineers, chemical plant and refinery maintenance, automobile maintenance and marine engine room personnel. Exposures for some of these workers regularly exceeded 20 - 40 f/cc, levels that are 200 - 400 times the current OSHA standard of 0.1 f/cc, with exposures of several months resulting in an increased risk of mesothelioma and lung cancer. The 1982 Nicholson analysis projected that the occupational exposures that occurred between 1940 and 1979 would result in 8,200 - 9,700 asbestos related cancer deaths annually, peaking in 2000, and then declining but remaining substantial for another 3 decades. Overall, the Nicholson study projected that nearly 500,000 workers would die from asbestos related cancers between 1967 and 2030. It is important to point out that these projections did not include mortality or morbidity from non-malignant asbestos diseases, which have or will affect an even greater number of workers.

Due to the long delay between exposure to asbestos and the onset of most asbestos related diseases (this latency can be over 40 years), many of the cases of disease today are occurring among workers who were first exposed in the 1940's, 1950's and 1960's, before asbestos was regulated and controlled. Information on the current impact of asbestos-related disease is presented in the recently issued National Center for Health Statistics report, Health, United States, 2004 (Table 48 - Exhibit 2). It shows that reported deaths from asbestosis have increased from 1,259 in 1999 to 1,467 in 2002, and reported deaths from mesothelioma have increased from 2,485 to 2,573 during the same time period. In addition a report from NIOSH in 2002, the Work Related Lung Disease Surveillance Report (WoRLD 2002). Exhibit 3) shows the dramatic increase in hospitalizations with a diagnosis of asbestosis. Hospitalizations of persons with a diagnosis of asbestosis have increased from 300 in 1970 to 20,000 in 2000 (see table 11-1). These data show that Americans are still sick from and dying of asbestos-related diseases.

The same data sources tell us that Americans are also hospitalized and dying from silicosis, coal workers pneumoconiosis, and other occupational dust diseases of the lung, and also show that hazardous exposures to silica and other dusts are still occurring. In 2000, there were 146 deaths and an estimated 1000 people were treated in a hospital with a diagnosis of silicosis, and for coal workers pneumoconiosis there were 850 deaths and 10,000 hospitalizations.

Exposures to silica are not yet well controlled. Exhibit 3 includes data from the WoRLD report showing that exposures to silica in construction exceeded the OSHA permissible exposure limit in 30% of the samples taken by OSHA in 1999, and exceeded a more protective NIOSH recommended exposure limit in 43% of samples. A similar pattern is seen in other industries with known silica exposures (see exhibit 2 table 6-15b, 6-16). It should be pointed out that both NIOSH and OSHA have recognized that silica exposure still poses a serious threat to workers and both agencies have had special programs directed towards reducing exposures and disease. OSHA has recognized that the current permissible exposure limit for silica is inadequate to protect workers and is in the process of setting a new standard that will reduce the legal exposure limit for silica. But unfortunately even with these current actions, we will see cases of silicosis for decades to come.

Exhibit 2
National Center for Health Statistics report, Health, United States, 2004 (Table 48)
http://www.cdc.gov/nchs/data/hus/hus04trend.pdf#tables

Exhibit 3
WoRLD 2002: preface, asbestosis, mesothelioma sections

http://www.cdc.gov/niosh/docs/2003-111/pdfs/2003-111a.pdf
http://www.cdc.gov/niosh/docs/2003-111/pdfs/2003-111b.pdf
http://www.cdc.gov/niosh/docs/2003-111/pdfs/2003-111h.pdf
http://www.cdc.gov/niosh/docs/2003-111/pdfs/2003-111g.pdf - Table 6-15b, Table 6-16

Exhibit 4

Diseases caused by exposure to airborne dust, fibre, or minerals:

Substances covered by the terms "dust" and "mineral":

(a) "mineral" includes hundreds of compounds, including all metals and metal alloys. As noted below, many diverse diseases are caused by metals and metallic compounds.

? Elements Class: The Metals and their alloys and the Nonmetals.
? Sulfides Class: The Sulfides, the Selenides, the Tellurides, the Arsenides, the Antimonides, the Bismuthinides and the Sulfosalts.
? Halides Class: The Fluorides, the Chlorides and the Iodides.
? Oxides Class: The Oxides and the Hydroxides.
? Carbonates Class: The Carbonates, the Nitrates and the Borates.
? Sulfates Class: The Sulfates, the Sulfites, the Chromates, the Molybdates, the Selenates, the Selenites, the Tellurates, the Tellurites and the Tungstates (or the Wolframates).
? Phosphates Class: The Phosphates, the Arsenates, the Vanadates and the Antimonates.
? Silicates Class: The Silicates (the largest class).
? The Organics Class: The "Minerals" composed of organic chemicals
? The Mineraloids: The "Minerals" that lack crystal structure
?
(b) "dust" is a non-specific term, and would include metal fumes, organic agents carried into the lung on dust, and radioactive particles carried into the lung on dust

Lung diseases caused by dust, fibers and minerals (not intended to be exhaustive):

? Asbestosis
? Silicosis
? Coal workers' pneumoconiosis (black lung)
? Byssinosis (cotton dust disease)
? Asthma (wood dust, nickel, flour, other organic agents where exposure is to a dust)
? COPD (after acute lung injury, chronic dust exposures, metal dust/fume exposure)
? Lung Cancer (arsenic, nickel, beryllium, chromium, radon)
? Hypersensitivity pneumonitis (wood dust, cork dust, other dust containing fungi)
? Acute inhalation injury from metals: lithium, chlorines, fluorides, antimony, cadmium, cobalt, manganese, mercury, zinc
? Bronchiolitis obliterans from inhalation of minerals: chlorine, sulfur dioxide, lithium, others
? Organic toxic dust syndrome, from grain dust
? Chronic beryllium disease
? Hard Metal Disease (Cobalt-related interstitial lung disease)
? Interstitial fibrosis from other specific elements: aluminum and aluminum silicates, antimony, barium, graphite, iron, kaolin, talc, tin, others

Occupational disease that are not lung diseases but are caused by exposure to minerals, fibre or dust (not intended to be exhaustive)

? Rheumatoid arthritis from silica
? Systemic sclerosis from silica
? Osseous fluorosis from fluoride
? Coronary artery disease from carbon disulfide, arsenic
? Cardiac heart failure from cobalt
? Hypertension from lead

? Anemia from lead
? Renal disease and renal failure from antimony, arsenic, barium, cadmium, chromium, copper, iron, lead, lithium, mercury, and other metals
? Liver disease from a range of metals
? Developmental toxicity from lithium, lead, mercury
? Infertility and spontaneous abortion from lead
? Cortical blindness from mercury
? Peripheral neuropathy from arsenic, mercury, lead
? Central neurological injury from metals - Mad-hatter disease, manganism
? Allergic skin disease from chromium, nickel, cobalt.
? Esophageal, gastric and colorectal cancer from silica, metals
? Liver cancer from arsenic, other metals
? Skin cancer from arsenic