Expert Report
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Qualifications: 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. Details of my education and training are set for in my curriculum vitae, attached as exhibit 1.

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. Because of my expertise in medical programs for asbestos-exposed workers, I participated in a working group with representatives from labor, industry, and insurance companies to develop medical criteria for Senate Bill 1125, a bill to establish a national trust fund for compensation of asbestos related disease in the United States.

Attached as Exhibit 1 is a true and correct copy of my current curriculum vitae, which sets forth my education, training, professional affiliations, research activities and publications.

Data considered: I reviewed the medical criteria for compensation of asbestos-related diseases set forth in the draft Owens Corning Personal Injury Settlement Trust Distribution Procedures (TDP). I reviewed a report written by Dr. Gary K Friedman on his review of x-ray and pulmonary function classification among Owens Corning Nonmalignant Claims Submissions 1994-1999, reviewed the settlement agreement with Owens Corning and Fibreboard dated 9/8/00. In reaching the opinions set forth in this report, I am relying upon my background, training, and experience; the literature cited below; and generally available medical knowledge about asbestos-related diseases.

Compensation: My fee schedule is attached as exhibit 2.

Prior testimony: A listing of all cases in which I have testified as an expert at trial or at deposition is attached as exhibit 3.

THE LEGACY OF ASBESTOS

Although this report 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, and will be with us for decades to come.

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.

Many of these workers were in the group sometimes referred to as the "first wave" of asbestos exposed workers – those directly involved in the manufacture or installation of asbestos insulation or products before there were any control measures or standards in place. 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. Nor do these projections reflect the full risk of disease among populations who were exposed in the 1950's and 1960's, but didn't have sufficient latency for asbestos related diseases to be manifested at the time the Nicholson study was conducted. This includes many of the building trades and construction workers who not only installed asbestos products, but also were exposed during removal, demolition, and renovation. This group is often referred to as the "second wave" of asbestos exposed workers, who account for much of the disease that is being manifested today. Similarly, the Nicholson study did not address the risk of exposures that occurred after 1979. While, OSHA and EPA regulations reduced asbestos exposures in the

1970's, strict regulation of asbestos did not occur until 1986. Even today, some workers are exposed to levels of asbestos that place them at increased risk of disease.

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. Nicholson's work provides a good foundation for estimating the future cases of asbestos disease, and has been utilized in many of the models to develop future asbestos disease claims projections, including claims projections made by the ARPC for the Manville Trust.

It is important to recognize that there is a good deal of uncertainty associated with these projections, reflected in the wide range of future disease projected by Manville and others (ranging from a low of 750,000 future claims to a high of 2.6 million future claims). There are a number of factors responsible for this uncertainty. As noted above, the Nicholson study and model projected cancer mortality related to asbestos. There have been no similar studies or estimates made for the non-malignant asbestos related diseases, such as asbestosis. All of the estimates in the projections for future disease and future claims for non-malignant disease have been based upon ratios of non-malignant disease to lung cancer cases or claims, not independent estimates of non-malignant disease. Epidemiological evidence shows that hundreds of thousands of workers have developed and will develop non-malignant disease. The claims information from the Manville Trust shows the majority of claims from 1995 – 2002 were for non-malignant diseases. While we know that certain groups of workers are at increased risk, and that these diseases will decrease as a result of reduced exposures, the extent and magnitude of non-malignant asbestos disease is not as well defined as the malignant diseases.

A recent Federal report from the National Institute for Occupational Safety and Health (NIOSH) shows that non-malignant lung disease from asbestos is still causing a significant number of deaths in this country. Exhibit 4 is taken from this report, and shows an increase in the number of deaths due to asbestosis from 1968 to 1999. Although some of the increase in deaths from asbestosis is likely due to increasing recognition of asbestos as a cause of serious lung disease and therefore lung disease deaths, the number of deaths in 1999 is a significant finding in its own right.

OVERVIEW OF ASBESTOS RELATED DISEASE

There are several medical diseases that occur as a result of asbestos exposure. The ones of greatest concern and importance are pleural plaques and thickening; asbestosis; lung cancer; colon, laryngeal, pharyngeal cancer; and mesothelioma. For many workers, these diseases are disabling or fatal.

Brief Overview of Lung Function

The lung's primary function is to transfer oxygen from the air into the blood stream, and transfer carbon dioxide from the blood stream into the air. To accomplish this, the lung must deliver oxygen to air sacs deep in the lung (alveoli). The alveoli are located into close proximity to small blood vessels (capillaries) so that the gases can cross from lung to blood and vice versa by diffusion across a very thin membrane. Any process that thickens the membrane between the alveolus and the capillary will reduce oxygen transfer into the blood stream.

The chest around the lungs acts like a bellows. When the chest expands, the lungs are stretched; pressure inside the chest drops relative to the atmosphere outside the chest, and air is pulled in through the nose and mouth into the lungs. When the chest relaxes the lung springs back to its resting shape, expelling air out of the lungs. Any disease process in the lung that makes the lung stiffer will decrease chest expansion and so limit how much air can be inhaled with each breath, as well as increase the energy needed to breathe. Scarring of the lining of the lung in any way that interferes with chest wall motion will have the same effect.

The lung has mechanisms to defend against foreign substances, from bacteria to asbestos. Those defense mechanisms are an integral part of the disease process after asbestos exposure. Inhaled air is delivered to the alveoli along a duct system of bronchi, and the bronchial tubes are lined with mucus to trap particles before those particles penetrate into the lung. If foreign substances do reach the alveoli, scavenger cells called macrophages attach them. Macrophages engulf asbestos fibers and try to destroy them. In many cases, the fiber survives, the macrophage dies, and oxygen radicals and inflammatory substances are released into the lung. The end result is scar formation in the lung from the release of substances that promote activity of fibroblasts, the cells that lay down scar after injury.

The lung also gets rid of foreign substances through the lymphatic drainage system. Asbestos fibers are carried through the lymph system to the pleural space, and can become trapped there. Once located in the pleura space, these fibers can induce scar formation.

Pulmonary function testing

The American Medical Association has developed guidelines for the evaluation of impairment from many diseases including lung disease. The AMA Guide states that each worker should undergo spirometry and DLCO as part of the evaluation of impairment, and exercise testing can add additional information if needed. Lung function can be measured accurately and reliably with 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, 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. The advantage of measuring lung volume with the TLC is this method is less dependent on the effort of the patient, and TLC measures different compartments of lung volume. The disadvantage to using TLC as part of the testing required for a compensation trust is that determination of TLC is considerably more expensive, and less available, than spirometry.

Diffusing capacity (DLCO) is a measure of gas exchange from the lung to the blood stream. While spirometry and total lung capacity measure lung volume and air flow, DLCO measures a different component of lung function. The ATS also sets standards for diffusion capacity, which ensure uniformity and reproducibility among laboratories. (The recent ATS statement on the *Diagnosis and Initial Management of Nonmalignant Diseases Related to Asbestos* points out that TLC has been shown to be insensitive to impairment in asbestosis, and points out the importance of DLCO in evaluation.)

Non-Malignant Asbestos-Related Diseases:

Pleural Plaques and Thickening

The pleura is a thin lining that surrounds the lung. There are two pleural layers, one on the chest wall and one on the lung, with a negative pressure relative to the atmosphere and a small amount of fluid between. The pleura allow the lung to expand easily inside the chest wall.

As noted above, asbestos fibers that are breathed into the lung are transported to the outside of the lung into the pleural space, and cause a scar to form in the pleural lining. When these scars reach a certain size they are visible on chest x-ray. A majority of persons with heavy exposure to asbestos develop some kind of pleural scarring. Pleural scars are described as pleural plaques, pleural thickening, diffuse pleural thickening, pleural fibrosis, and pleural asbestosis. There is no universal agreement on the meaning of each of these terms. The 2003 revision to the ILO classification will help with these definitions, by defining pleural thickening as diffuse "only in the presence of and in continuity with an obliterated costophrenic angle."

The ATS statement cited above reviews studies of large groups of asbestos-exposed persons and concludes these studies have found a significant reduction in lung volume attributable to pleural plaque; this lung function loss averages about 5% of FVC. It is possible that this loss of lung function is due to asbestosis that is not visible on chest x-ray, or due to a change in elasticity of the chest wall. Most pleural scars alone do not cause enough loss of lung function to cause a disability, but even a small loss may be significant if combined with other impairments. However, some types of pleural scarring do cause more significant loss of lung function in their own right. Workers with diffuse pleural thickening have a loss of lung function that is higher by a factor of

two or more than that seen with circumscribed plaque. Scars that involve the costophrenic angle (the angle between the base of the lung and the diaphragm) can cause loss of lung function, as can extensive pleural scarring on both sides of the lung.

Parenchymal Asbestosis (Pulmonary Asbestosis)

Asbestosis occurs when asbestos exposure causes scar formation in the substance of the lung itself. These scars can interfere with lung function, for they block the transport of oxygen from the air in the lungs into the blood vessels that travel through the lungs. Oxygen can only cross the membranes of the lung if they are thin; asbestosis causes them to thicken. Asbestosis also makes the lungs stiffer, which results in a decrease in lung volume and an increase in the energy needed for chest expansion. As a general rule the greater the exposure to asbestos the more likely the disease is to be present and the more severe the scarring; there is a dose-response relationship between exposure and disease. However, some people seem to form scars more readily than others, and so we see a range in the severity of disease after similar levels of exposure to asbestos.

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.

Classification of pleural scarring uses a separate scale, with specific notations made for location and the specific type, length, and width of the scarring.

Even though the ILO system was designed to standardize reading x-rays for asbestosis and other dust diseases of the lung, studies using the classification in asbestos exposed workers have found readers often disagree about classification of the same x-rays. Using the classification is somewhat of an art. The "best" readers agree 80% of the time with each other; 20% of the time they assign a different score to the same x-ray. If the scarring is extensive, a difference of one

grade on the scale is not important. But if the x-ray shows less extensive scarring, a difference of one grade can be the difference between making diagnosis of asbestosis or deciding asbestosis is not present. For this reason experts agree that the x-ray alone should not be used to make a diagnosis of asbestosis; the examining physician should use the occupational and medical history, results of pulmonary function testing, and other medical data to reach a diagnosis. Experts agree that asbestosis can be present in the lung even though the x-ray is normal using the ILO classification system. A recent document from the ATS, *Diagnosis and Initial Management of Nonmalignant Disease Related to Asbestos,* affirms the use of high resolution CT scanning for diagnosis of asbestosis when the chest x-ray is normal.

As described in numerous peer-reviewed publications and in the ATS report cited above, high-resolution computed tomography (HRCT) is now widely accepted as a diagnostic tool for asbestosis and asbestos-related pleural scarring. HRCT is an excellent technique for diagnosis of asbestosis and asbestos-related plaque. Recent studies show that readers using a scoring index for HRCT were more accurate and reliable in the diagnosis of asbestosis than when using plain chest x-rays. This study concluded that "the examined HRCT scoring method proved to be a simple, reliable, and reproducible method for classifying lung fibrosis and diagnosing asbestosis also in large populations with occupational disease, and it would be possible to use it as a part of an international classification". Expert consensus supports this conclusion.

Disease from asbestos is also detectable on pulmonary function testing, and PFTs are used to quantity the level of lung impairment due to asbestosis. Asbestosis makes the lung stiffer and smaller, so the volume of air in the lungs is decreased. Oxygen transport as measured by the diffusion capacity is also decreased. Abnormalities are measured using spirometry, lung volumes, and gas exchange testing. Asbestosis can affect each of these tests without necessarily showing an abnormality in the other two. Spirometry and total lung capacity both measure lung volume, but one may be abnormal while the second remains normal. The diffusion capacity measures a decrease in oxygen exchange in the lung, and so is measuring a different function of the lung than lung volumes. Asbestosis can just as easily be manifest with a decreased lung volume or a decrease in gas exchange; neither is a better, more sensitive or more accurate test, and both types of tests must be used in any set of diagnostic criteria. The diffusion capacity has been shown to correlate with the severity of fibrosis found on pathologic examination of the lung, and a reduction in diffusion capacity can precede x-ray changes and changes in total lung capacity. The changes in pulmonary function at times can be subtle, and test results should be interpreted by someone with experience in asbestos-related diseases. Pulmonary exercise testing can be used to clarify subtle abnormalities.

Once asbestosis develops it is irreversible. The disease can get worse even after exposure stops. Factors that are associated with worsening scarring include the severity of disease (the more the scarring, the more likely it is to get worse), and the amount and intensity of exposure to asbestos. Because of the damage to the lungs a person with asbestosis is at increased risk of lung infections and so should get regular medical care and influenza vaccines.

Determination of Impairment

Lung function can be measured accurately and reliably with pulmonary function testing. The American Medical Association has developed guidelines for the evaluation of impairment from many diseases including lung disease. The AMA Guidelines have been incorporated into compensation systems in states, and are widely used by physicians. The diagnosis of asbestosis depends in part on characteristic findings on pathology, chest x-ray or CT scan, but impairment must be measured with pulmonary function testing.

The AMA Guide states that each worker should undergo spirometry and DLCO as part of the evaluation of lung impairment, and exercise testing can add additional information if needed. Using a combination of forced vital capacity (FVC), forced expiratory volume in one second (FEV1), DLCO, and oxygen consumption on exercise testing (VO_2max) when needed, the patient is placed into one of four levels. The first level under the AMA has no impairment, the second level is between the lower limits of normal lung function and 60% of normal; the third level is less than 60% and more than 50% of normal lung function; the most severe level is a loss of more than 50% of lung function. The illustration in the AMA Guide for the second level is a good example to use here:

> *Fifty four-year-old retired power plant mechanic with a history of asbestos exposure from age 18-37. He is short of breath when walking on level ground with other people his own age. His chest x-ray shows asbestos-related pleural changes, but no parenchymal asbestosis. His FVC is 64% of predicted, his FEV1/FVC ratio is 81%, and his DLCO is 78%.*

This man has asbestos related disease. He has lung impairment significant enough to make him short of breath with normal walking; in my experience, this degree of impairment would make him unable to continue in a physically demanding job. As noted in the example, this man is retired at age 54. He would fall into AMA level II.

At the highest level using the AMA Guides, where the worker has lost more than 50% of lung function, the worker would be unable to perform activities of daily living, such as getting dressed, taking a shower, cooking dinner, or any minimal work around the house.

A worker can have demonstrable impairment but still have test results that are in the normal population range. Comparing an individual's results on spirometry, lung volumes and diffusion to the normal range for the population is how we generally determine impairment. In some cases we know the person's pre-disease lung function, and so can compare current testing to his own normal tests from the past. This comparison allows much better precision in estimating impairment. The AMA Guides explicitly state that "in individuals where the pre-injury or pre-disease values differ from the population listed values, the examiner may depart from the population listed normal values for determining an impairment rating..."

Determining That Impairment on PFTs Is Caused By Asbestos

Asbestos scarring of the lung 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, as discussed above. Smoking causes a reduction in air flow out of the lung (obstructive disease), and causes 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 smoking-related 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 can be incorporated into compensation criteria. Many workers have asbestosis and concomitant obstructive lung disease; compensation criteria can be set to allow compensation of the asbestos-related disease.

The differentiation of asbestosis from smoking-related lung disease is somewhat complicated by the fact that asbestos exposure causes a small amount of obstructive lung disease in addition to the restrictive disease that is well recognized. The ATS document *Diagnosis and Initial Management of Nonmalignant Diseases Related to Asbestos* concludes that the magnitude of the asbestos effect on airway function is relatively small, but can contribute to overall lung impairment if superimposed on, and therefore added to, another lung disease. When we want to differentiate asbestosis from obstructive lung disease caused by smoking, we must allow some degree of obstruction to be present in asbestosis to account for the known effect of asbestos on airway function.

Lung Cancer and Respiratory Cancers

All major types of lung cancer are caused by asbestos. Lung cancer is incurable in 90% of cases at the time of diagnosis, and those diagnosed with lung cancer usually die within a year. Numerous studies show that there is a dose-response relationship between exposure to asbestos and the risk of lung cancer, with increasing exposure leading to increasing risk of disease. Workers with asbestosis have a higher risk of lung cancer than asbestos exposed workers without asbestosis, but in this case the asbestosis may simply be a surrogate measure of exposure; significant asbestos exposure is required to cause asbestosis. Asbestosis is not a necessary intermediary for development of asbestos related lung cancer. Workers with pleural plaque do not appear to be at higher risk for lung cancer than workers with similar exposure who did not develop plaque. Pleural plaque is a convenient marker of prior exposure to asbestos, and so has been used as a surrogate for significant occupational exposure in bankruptcy settlement agreements, but the risk of lung cancer is not restricted to workers with pleural plaques.

The Helsinki Criteria were developed in 1997 by an international group of experts, as a set of state of the art criteria for attribution of disease to asbestos exposure. These criteria establish an exposure level of 25 fiber-years, or the equivalent exposure using an occupational history, as a level of exposure that significantly increases the risk of lung cancer. Several European countries have established this or a similar level of exposure as the criterion to be used for compensation of a lung cancer in an asbestos exposed worker.

Smoking and asbestos act in concert to cause lung cancer, each multiplying the risk conferred by the other. In a large study of asbestos insulation workers in North America, non-smoking asbestos workers were five times more likely to die from lung cancer, smokers not exposed to asbestos were approximately 10 times more likely to die from lung cancer, and asbestos workers who smoked were more than fifty times more likely to die from lung cancer. Asbestos workers who stopped smoking demonstrated a sharp decrease in lung cancer mortality.

Although smoking does increase the risk of lung cancer, this effect does not detract from the risk of lung cancer attributable to asbestos exposures. The risk of lung cancer after exposure to asbestos is related to the amount of asbestos inhaled.

The risk of cancer of the pharynx and larynx is also increased by asbestos exposure. Smoking also contributes to the development of these diseases, and the risk from asbestos is thought to multiply the risk from smoking as it does for lung cancer.

Colon Cancer and Gastrointestinal Cancer

There is a higher incidence of cancers of the gastrointestinal tract among asbestos workers than among the general population. In people exposed to asbestos for more than 20 years, the rate of colon cancer is increased by a factor of 2. It is important for all asbestos-exposed workers to have regular check-ups with their doctors, to look for early signs of colon cancer.

Mesothelioma

Mesothelioma is a rare cancer of the pleura, the lining of the lung, and the peritoneum, the lining of the abdomen, that occurs in persons exposed to asbestos. Mesothelioma can result from a limited exposure to asbestos, such as working in a shipyard for a few months or living with a worker exposed to asbestos at work. All types of asbestos fibers cause mesothelioma. Virtually all of mesotheliomas in this country are caused by past exposure to asbestos. This cancer is almost impossible to treat and is usually fatal within 18 months of diagnosis. (There are some promising new developments, from effective surgery in selective cases to chemotherapy for others, but the prognosis overall is still poor).

Opinions

(1) It is my opinion that asbestosis is not required in order to attribute a lung cancer to asbestos exposure

Some authors express the opinion that clinically diagnosed asbestosis must be present in order to attribute a lung cancer to asbestos exposure. The available body of medical evidence does not support this opinion. To understand the relationships between asbestos exposure, asbestosis and lung cancer, several important characteristics of radiological detectable asbestosis must be described. One, the likelihood of developing asbestosis increases with the amount of asbestos dust inhaled. Second, workers who smoke have a higher likelihood of developing asbestosis, because of reduced clearance of asbestos from the lung (smoking damages the lungs' defense mechanisms). Third, asbestosis is a disease that generally takes 15 or more years to develop. All three factors also describe the likelihood of developing an asbestos-related cancer: increasing risk with increasing dose, higher risk in a smoker, and a substantial latency between onset of exposure and onset of disease. Therefore it is clear that workers with asbestosis will have a higher risk of lung cancer than workers without asbestosis, on average.

The question at issue is not whether workers with asbestosis have a higher risk of lung cancer than workers without asbestosis, but whether workers without asbestosis have a risk of lung cancer increased above that of the general population. Many well-conducted epidemiological studies support a direct relationship between asbestos exposure and risk of lung cancer, and show an elevated risk of lung cancer in asbestos-exposed workers in general. An international group developed the Helsinki criteria for attribution of lung cancer in asbestos exposed workers, and concluded that an exposure of 25 fiber-years, in the absence of any other disease, doubles the risk for lung cancer. The numerous studies that support my opinion are included in the references attached.

(2) It is my opinion that a 1/0 classification of an x-ray, using the International Labor Organization Classification for Pneumoconiosis, is sufficient for the diagnosis of asbestosis when used as defined in the TDP.

Asbestosis is often present on histological examination of the lung when it is absent on chest x-ray in the same person; a good estimate is that 20% of asbestosis is missed on the chest x-ray among groups of workers with significant exposure to asbestos. Asbestosis is often present on HRCT even when the chest x-ray is normal. Huuskonen calculated that an x-ray read as 1/0 or higher had only a 51% sensitivity for diagnosis of asbestosis, using the HRCT as the gold standard. Kipen reported that 18% of insulators who had asbestosis found on pathological examination of the lung had a normal chest x-ray. If we were to require a 1/0 film in all cases of asbestosis, these workers would be excluded. Pathological examination is not required in the absence of x-ray abnormalities; a combination of CT scan and exercise testing can reasonably approximate the specificity as tissue examination. Based on these two other ways of determining that asbestosis is present, by pathology or HRCT, it is clear that even a 1/0 film fails to detect 20-50% of asbestosis. Requiring a 1/1 film would exclude even more workers with asbestosis.

The ATS document *Diagnosis and Initial Management of Nonmalignant Diseases Related to Asbestos* states in the section on clinical diagnosis of asbestosis that "A profusion of irregular opacities at the level of 1/0 is used as the boundary between normal and abnormal in the evaluation of the film..."

(3) Functional impairment need not be present to diagnose a disease.

A lung disease can exist without impairing lung function tests. Steadman's medical dictionary defines disease: "A disease entity, characterized usually by at least two of these criteria: a recognized etiological agent (or agents); an identifiable group or signs and symptoms; consistent anatomical alterations." There are numerous diseases that can be diagnosed at an early stage, before they cause organ system damage and loss of function: diabetes and hypertension are two clear examples. For lung disease specifically, a cancer of the lung, or a mesothelioma, can be present without any symptoms or loss of lung function; no one would postulate that cancer is not a disease. The ATS statement on *Diagnosis and Initial Management of Nonmalignant Disease Related to Asbestos* affirms that functional impairment is not required for the diagnosis of nonmalignant asbestos-related disease.

(5) A worker does not have to have an FVC or TLC lower than 80% of predicted in order to have impairment.

Asbestos-related diseases clearly can cause impairment of function even if the FVC or TLC is above 80% of predicted (or above the lower limits of normal using the 95% confidence interval). Many workers will have a significant loss of lung function with an FVC over 80% of predicted. The line between "normal" and "abnormal" is set using 80% of predicted, or the lower limits of normal using a regression equation (for most people, these values are the same or very close to each other). Either "cut-off" is the level at which we can be 95% confident that a particular test is truly different from the average for the general population without lung disease. The normal is defined using the average FVC for a group of individuals without known lung disease. Differences between healthy people are to some part due to age and height, but even when those factors are taken into account there can be a wide range of FVC in the healthy population. The cut-off between normal and abnormal is set so that 95% of healthy people are above that line.

In any one individual, a change of 15% or more in FVC represents a true decline in lung function. Because the range of "normal" lung function is from 120% to 80% of predicted, it is easy to see how a person who starts work with a high normal value for FVC can lose more than 15% of his lung function and still be defined as "normal" because his tests are greater than 80% of predicted. When repeated tests on an individual are available we can detect a loss of lung function without using averages, but for most people no such database exists.

Another way to look at the same question is to ask if we can we detect loss of lung function using more sensitive tests than the FVC, since it is clear that some individuals can have loss of

lung function and have a "normal" FVC. The American Thoracic Society, in a recent publication on cardiopulmonary exercise testing (CPET), said "Increasing awareness of the inadequacy of resting cardiopulmonary measurements and tests in adequately predicting function impairment (work capacity) and exercise limitation in patients with respiratory disease has focused attention on the expanded role of CPET in evaluation of impairment and disability. CPET compliments other clinical and diagnostic modalities and by directly quantitating work capacity improves the diagnostic accuracy of impairment/disability evaluation." The American Medical Association Guide to the Evaluation of Impairment recognizes the value of CPWT as well, by including VO2 max as one of the criteria on which to evaluate impairment.

The AMA Guides also rely on the diffusion capacity (DLCO) as a valid measure of impairment; DLCO a test that measures oxygen transfer from the lungs to the blood stream. The DLCO is often abnormal in persons with asbestosis even with a normal FVC or TLC. This again demonstrates that other tests are more sensitive to loss of lung function than the measurements of lung volume.

(6) A physician can diagnose asbestosis from medical records, without a hands-on examination of the patient.

The elements used for the diagnosis of asbestosis generally include the occupational history, the medical history, a physical examination, radiology (conventional chest x-ray or CT scan), and pulmonary function testing. As stated above, The American Thoracic Society, in a 1986 statement on the diagnosis of asbestosis, states that the only two requirements for a diagnosis of asbestosis are that there be sufficient exposure to asbestos and sufficient latency. The findings on the other components are then added into the assessment. All these elements can be presented in a medical file, and be sufficient for review and diagnosis without an examination of the patient. More specifically, the physical examination is the least important of these elements of diagnosis. Characteristic findings of asbestosis on physical exam are rales in the lung and clubbing of the fingertips. Clubbing is seen with severe lung impairment and so is often absent in well defined cases of asbestosis. Rales are helpful but not necessary for the diagnosis of asbestosis.

(7) Conclusions of Dr. Gary K Friedman on x-ray classification among Owens Corning Nonmalignant Claims Submissions 1994-1999 are not supported by his analysis and the methods used.

I reviewed the above report and its appendices. The primary objective of Dr. Friedman's review of a set of claims to the National Settlement Program was to generate data for the debtor's valuation expert, Dr. Thomas Vasquez. Dr. Vasquez was expected to use the results of this report to estimate the proportion of claims to the NSP that would be considered impaired using the NSP criteria. Dr Friedman presented data that show between 13% and 26% of the 1360 claims reviewed were either impaired or might be impaired if additional pulmonary function testing was obtained to meet the specific criteria of the NSP. The range is large due to

variability in the PFT data; not all cases could be conclusively assigned to either "impaired" or "unimpaired" categories.

In the report, Dr. Friedman reaches several conclusions that cannot be supported by the data he reviewed. Let me address two specifically.

(a) He notes that 5 "B" readers accounted for 80% of the cases reviewed, and concludes that *"The spectrum of asbestos related non-malignant disease reported by these five "B" readers was inconsistent with that anticipated from the peer review literature authored by other plaintiff experts."* Dr. Friedman is looking at a group of x-rays submitted as part of a claim for compensation, and comparing the findings on those x-rays to studies done on entire populations of workers. He does not know the entire population of asbestos-exposed workers from which these cases came, he does not know how many workers were screened out of that population, nor does he know how many of those who were screened submitted a claim. One cannot compare rates of disease among claimants to rates of disease among an exposed population. There are many variables that affect whether an exposed worker submits a claim, making a group of claimants very different from the larger group from which it comes. For example Dr. Friedman finds that the x-rays reviewed for his analysis had a lower proportion of pleural disease than expected based on population-based studies. However, if a settlement agreement will only compensate a subset of all pleural disease cases, then the claims filed would be expected to have a lower proportion of pleural disease.

(b) Dr. Friedman concludes that *There was a high degree of inter-reader variability among these five "B" readers"* and that *"There was a high degree of inter-reader variability when these five "B" readers were compared to over 40 other "B" readers or other physicians submitting plaintiff reports in this study."* Inter-reader variability can only be assessed if two or more readers classify the **same** x-ray. The classifications submitted by the "B" readers in this analysis were all on **different** x-rays, with one B reading per film. One cannot reach any conclusions about variability between readers. Differences from one reader to another reader, when the readers are looking at different workers, could easily be explained by differences in the actual disease in the workers examined.

I may be asked to review expert witness reports submitted by other parties in this case, and I know I will not be able to see those reports until after October 15th. I reserve the right to modify this report and provide opinions on those expert witness reports after my review.

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