

Testimony of

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Asbestos: Mixed Dust and FELA Issues

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Senator Specter, Senator Leahy, and Members of the Judiciary Committee: Thank you for the opportunity to testify before the Committee about medical issues concerning silicosis and asbestosis. My name is Dr. David Weill, and I will focus my remarks today on two subjects. First, I will compare silicosis and asbestosis from a medical point of view. As I think you will see, silicosis and asbestosis are different diseases; they are not easily confused in practice; and it is very rare for one person to have both diseases. Second, I will comment on the medical claims that have been made in the recent wave of silica lawsuits, many of which involve diagnoses of both silicosis and asbestosis. In many cases, these two distinct diagnoses are based on the same clinical information - that is, the same worker is diagnosed with asbestosis at one point in time and silicosis in another, and the two diagnoses are then used in separate lawsuits.

Before turning to the substance of my testimony, I should say a few words about my background. I am board certified in Internal Medicine and Pulmonary Medicine. I received my bachelor's degree from Tulane University and my medical degree from the Tulane University School of Medicine. My training includes a residency at the University of Texas Southwestern Medical Center and two Fellowships at the University of Colorado Health Sciences Center (first in the Division of Pulmonary and Critical Care Medicine and subsequently in the Lung Transplant Program). From 1996 to 1999, I served as the Director of Pulmonary Rehabilitation and as Medical Director of the Lung Transplant Program at Medical City Hospital in Dallas, Texas. From 1999 to 2002, I was Associate Professor of Medicine, Division of Pulmonary and Critical Care Medicine, and Medical Director of the Lung Transplant Program at the University of Alabama at Birmingham.

Currently, I hold several positions at the University of Colorado Health Sciences Center in Denver, Colorado. I am Associate Professor of Medicine, Division of Pulmonary and Critical Care Medicine, Associate Director, Lung Transplant Program and Attending Physician, Surgical Intensive Care Unit. Since 1999, I have also been Associate Editor for the Journal of Heart and Lung Transplantation.

At the University of Colorado respiratory center, we are referred and treat patients with both common and rare respiratory conditions that are amenable to either novel medical or surgical therapy. Such referrals include people with cases of asbestosis or silicosis, and in my pulmonary practice I have diagnosed and treated patients with both diseases. 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 x-rays for lung conditions (pneumoconiosis) such as those caused by exposure to silica and asbestos dust.

In addition to seeing silicosis and asbestosis patients in my clinical practice, I have in the last five years been involved in reviewing asbestosis and silicosis claims made in lawsuits. I have reviewed hundreds of patient histories and chest radiographs during this time. A few years ago the number of silica claims that I saw was comparatively small. That number has increased dramatically since 2002, however, and the trend line is still going up.

In the Spring of 2004, I had the privilege of serving as visiting professor at the National Institute of Occupational Medicine and Poison Control in Beijing, China. One purpose of my tenure in China was to determine the feasibility of a collaborative study involving silica and asbestos exposed workers in the Chinese workforce. During my time in China I saw hundreds of cases of asbestosis and silicosis, many involving very serious and advanced stages of disease. The Chinese experience is sobering, and far different from what I have seen in the U.S., where genuine cases of these diseases are quite rare.

I. Silica and Asbestos from a Medical Perspective

Let me turn now to my first subject - a comparison of the diseases caused by exposure to asbestos and silica, respectively. I will focus on two non-malignant respiratory diseases - asbestosis and silicosis -- because that is where the confusion normally occurs. For most other diseases caused by either asbestos or silica inhalation, there is no conceivable overlap. Asbestos exposure can cause mesothelioma, for example, but silica exposure does not. Conversely, some observers have associated silica exposure with autoimmune disorders and tuberculosis, but those diseases are not associated with asbestos exposure. The only category where overlap is possible is lung cancer, which some believe can be caused both by asbestos and silica exposure in certain situations.

A. Silicosis and Asbestosis

1. Silicosis

Silica (silicon dioxide) is the most common mineral in the earth's crust and comprises 20% of the crustal weight of the earth. It is found in sand, gravel and all soils and is the principal constituent of 95 percent of the earth's rocks.

Silicosis can result when sufficient amounts of respirable silica are inhaled and become deposited in the lungs, overwhelming the body's natural defense mechanisms. Whether disease develops depends on the intensity and duration of exposure, and, likely, on individual susceptibility.

The principal disease associated with silica exposure is silicosis. Silicosis can occur in three different forms: chronic silicosis (subdivided into simple or complicated forms of the disease), accelerated silicosis, and acute silicosis.

Chronic simple silicosis is the most common form of the disease in the United States today. Chronic silicosis, in its simple form, typically requires more than twenty years of moderate exposure. It is characterized by rounded nodules, like tiny marbles, principally in the upper lobes of the lungs. In its lower grade forms, simple silicosis does not generally result in respiratory impairment. It may progress, or become more serious over time, especially in workers who continue to be exposed to silica. However, progression tends to be slow and depends on several factors.

In the complicated form of chronic silicosis, the small rounded nodules found in simple silicosis form conglomerate lesions, whose largest diameter exceeds 1 centimeter. Most patients with complicated silicosis have respiratory impairment with abnormal pulmonary function test findings typically indicating lung "restriction" or a reduction in the lungs' capacity for inhaled air. Accompanying airway obstruction is not uncommon. Rare today, accelerated silicosis results from higher exposure to silica, usually over a period of five to ten years. This form of the disease may progress whether or not continued workplace exposure occurs. Chest x-rays can show either a pattern of small rounded nodules alone or in conjunction with larger conglomerate opacities.

Acute silicosis is a rapidly progressive, fatal disease. It occurs after massive exposures which can be as short as several months to a few years. In acute silicosis, the spaces in the lung where oxygen exchange takes place (the "alveoli") become filled with fluid and cells. In contrast to chronic and accelerated silicosis, acute silicosis is not characterized by small rounded nodules.

Accelerated and acute silicosis are rarely found in developed countries today, although there have been case reports many years ago of acute silicosis among sandblasters and drill workers drilling through silica-containing rock. In developing countries the picture is very different, and during my visit to China a year ago I saw a number of cases of accelerated and acute silicosis.

2. Asbestosis

Unlike silicosis, which is characterized by the presence of small nodules in the lungs, asbestosis involves fibrosis in the parenchymal tissue of the lungs in the area where oxygen exchange takes place. Asbestosis can result in both a "restrictive" pattern of disease - effectively a reduction in the volume of the lungs - and interference with the gas exchange process. From a pathologic, radiographic, and clinical perspective, asbestosis and silicosis are very distinct diseases.

B. Diagnosis

In general, the diagnosis of both silicosis and asbestosis focus on three clinical criteria: (1) an occupational exposure history, including sufficient latency, to cause disease; (2) the presence of characteristic chest x-ray abnormalities; and (3) the exclusion of other pulmonary diseases that can mimic either disease radiographically. While, of course, it is impossible to diagnose either disease on the basis of chest x-rays alone, there are characteristic differences in the way these diseases appear on x-ray films. Outside the litigation setting, confusion between silicosis and asbestosis does not occur.

In evaluating pneumoconioses, chest x-rays are normally interpreted using the International Labor Office ("ILO") radiograph classification system. The purpose of the ILO system was to standardize the interpretation of chest x-rays using descriptions of the size, shape, and degree of involvement (i.e., the profusion) of radiographic abnormalities. The system is used to describe shape (regular or irregular) and size (regular: p, q, r and irregular: s, t, u) characteristics of radiographic abnormalities. The extent of radiographic abnormalities (profusion) is numbered from normal (or 0) to increasingly abnormal (1, 2, and 3). The ILO classification scheme also addresses which of the six lung zones are involved (upper, middle, and lower in either the right or left lung). Also, particularly important when distinguishing between asbestosis and silicosis, the presence and type of pleural abnormalities are noted. In

completing the ILO form, the reader is directed to include all the abnormalities that exist.

Chronic silicosis (simple) is characterized by tiny round nodules, primarily in the upper lobes of both lungs. On an x-ray, these round nodules show up as small, rounded opacities, which would be rated on the ILO form as p, q, or r. In contrast, asbestosis is characterized by linear parenchymal fibrosis, which shows up on an x-ray as small irregular opacities (s, t, or u), primarily in the lower lobes of both lungs.

Chronic silicosis (complicated) is even harder to confuse with asbestosis on chest x-rays than is the simple form of the disease. In complicated silicosis, the tiny round nodules found in simple silicosis join together, and the opacities that show up on the x-ray film are large, measuring greater than 1 centimeter (as opposed to 1 to 3 millimeter nodules in simple silicosis). No form of asbestosis shows large opacities on chest x-rays.

Although acute silicosis is now almost never seen in the United States, it would also be hard to confuse with asbestosis on chest x-rays. Acute silicosis, which involves fluid and cells in the air sacs of the lung, has a completely different appearance on x-rays, causing consolidation in the lung which would appear radiographically as uniformly "white".

In summary: When confronted with the presence of diffuse abnormalities on chest x-rays, pulmonary doctors attempt to categorize the abnormalities into broad radiographic patterns. Asbestosis involves reticular, or "linear" abnormalities; silicosis is characterized classically by nodular (rounded) abnormalities. Not only are these appearances different in individual cases, these two broad radiographic patterns point one toward entirely different diagnostic categories. Diseases other than asbestosis fall into the reticular group. Examples included idiopathic pulmonary fibrosis, radiation pneumonitis, chronic hypersensitivity pneumonitis, and chemotherapy-induced lung disease. Diseases other than silicosis fall into the nodular group, including sarcoidosis, berylliosis, coal workers' pneumoconiosis, and metastatic cancer. Distinguishing among diseases that fall into the same radiographic categories requires the clinician to consider other factors, most notably a careful history and pulmonary function test. There should not, however, be confusion between diseases that fall into different categories, such as asbestosis and silicosis.

Although asbestosis and silicosis are different diseases that look different on x-ray films, it is theoretically possible for one person to have both diseases. A person could be exposed to both silica and asbestos in sufficient quantities to cause either disease, but it would be extremely unusual for one person in a working lifetime to have sufficient exposure to both types of dust to cause both diseases. In my clinical experience in the United States, I have never seen a case like this and colleagues who saw patients in periods where exposure levels were much higher have difficulty recalling an individual worker who had both asbestosis and silicosis. Even in China, where I saw workers with jobs involving high exposure to asbestos and silica (such as sandblasting off asbestos insulation), I did not see anyone or review chest radiographs of anyone who had both silicosis and asbestosis.

II. Recent Silica Litigation

I was first asked to review the medical evidence in support of silica claims about five years ago. In the beginning, there were relatively few claims, but they have accelerated since then. All in all, I have reviewed 300-400 case files, more than half over the last two years. I have reviewed numerous diagnoses in the ongoing multidistrict litigation ("MDL") concerning silicosis liability in the United States District Court for the Southern District of Texas. Almost invariably these cases have involved alleged, low profusion category simple chronic silicosis in which one would not expect significant respiratory impairment caused by the presence of silicosis.

From a medical standpoint, it is puzzling to see so many ostensible silicosis cases in such a short period of time. In my clinical practice and those of colleagues in the occupational medicine field, it is unusual to see new silicosis cases, at least in the United States, largely because of the workplace regulations that have been put in place by OSHA. The situation in China, and the rest of the developing world, is very different.

Although statistical evidence is incomplete and imperfect from a methodological point of view, few would question the proposition that industrial dust control mechanisms have made silicosis much less common today than it was a generation ago. This conclusion is supported by reviews of death certificates undertaken by NIOSH. Death certificate data has limitations because (among other things) there is no rigorous control over the filling out of death certificates and because the frequency with which a given cause of death is reported can vary over time due to factors such as the prominence of certain diseases in doctors' minds and patient family's ability to recall accurately the involved diagnoses. Further, invalid diagnoses may have been assigned to patients who did not in fact have a reported disease and only carry a certain diagnosis because rigorous diagnostic criteria had not been previously applied. Nevertheless, even with methodological reservations, the overall picture with silica is very clear. As NIOSH reports, "Over the past several decades, silicosis mortality has declined, from well over 1,000 deaths annually in the late 1960s to fewer than 200 per year in the late 1990s." In fact, from 1968 to 1992 "mortality associated with silicosis . . . declined more than that associated with other types of pneumoconiosis."

Silicosis and, for that matter, all pneumoconioses 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, silica - related health effects have become less prevalent. The declining incidence of silicosis should be associated with fewer and fewer silica lawsuits, but in my experience exactly the opposite is taking place. Silica lawsuits are sharply increasing even though from a medical perspective silicosis is declining.

I have several observations about the silica litigation that may help explain this discrepancy.

First, nearly all of the litigation diagnoses do not come from treating physicians but from screening companies that sell their diagnostic services to plaintiffs' law firms. The screening companies employ a small group of physicians who have been certified by NIOSH as B Readers and who make diagnoses of silicosis or asbestosis or both. As I shall explain, whether a screening company diagnoses silicosis or asbestosis appears to depend on litigation rather than medical factors.

Second, among the three or four hundred silicosis claims I have reviewed, only two involved actual silicosis. One of those cases involved significant breathing impairment, and the other did not. The remaining cases were in my view clearly not silicosis at all.

Third, many of the silicosis plaintiffs have also been diagnosed by plaintiff experts, at one time or another, with asbestosis. In my experience, in the vast majority of these cases of dual diagnoses, the plaintiff has been x-rayed twice. The first x-ray was typically taken as part of an asbestos screening conducted several years ago, before the recent increase in silicosis claims, and resulted in the conclusion that the plaintiff had abnormalities consistent with asbestosis. Subsequently, the screening companies and lawyers have asked these plaintiffs to return for a second x-ray, and a new, silicosis diagnosis is based on the second film (which in all instances was very similar to the first x-ray). Almost never does the second "diagnostic report" mention the first "diagnostic report".

For example, one screening firm is responsible for many of the silicosis claims in the ongoing Texas MDL. To generate these claims, this firm simply screened for silicosis many of the same people it had previously screened for asbestosis years earlier. But the lapse between the first and second x-rays is not always a matter of years. I encountered one case where a different screening firm diagnosed a plaintiff in February and then again in March. The former screening generated an asbestosis "diagnosis" while the latter generated a silicosis "diagnosis." Silicosis was not mentioned in the first report, and asbestosis wasn't mentioned in the second one. A treating physician, of course, would have noted all potential abnormalities on the first report. This is one important way in which medical reports in clinical practice are different from the litigation-oriented reports that I have reviewed.

In the remaining cases involving dual diagnoses of silicosis and asbestosis, the claimant was x-rayed only once and has received two different and divergent diagnoses (asbestosis and silicosis) based on the same x-ray film. I understand that this practice enables plaintiffs to pursue separate asbestosis and silicosis claims, seeking compensation for each. There is no satisfactory medical explanation for it. Even in the rare case in which a person might have both asbestosis and silicosis, a doctor performing an examination for medical reasons would indicate both diseases on the same form.

By contrast, in the two cases of actual silicosis I have encountered in litigation, each patient had been x-rayed once and diagnosed once. As these cases demonstrate, there do exist real cases of silicosis. But the majority of silicosis claims I have seen are not valid; they are simply recycled or duplicated asbestos claims.

III. Conclusion

The current rise in silicosis claims cannot be explained by medical factors. In a disease like silicosis that is dose-dependent, a reduction in exposure levels would be expected to result in a decrease in disease incidence, and this is exactly what we have seen in the clinical setting. In the litigation arena, the vast majority of the alleged silicotics that I have reviewed do not involve real silicosis at all, which is not surprising because silicosis is a relatively rare disease today. Unfortunately, many of the silicosis claims are derived from the same workers who originally filed asbestos claims. However, based on characteristic chest x-ray findings and other clinical factors, it should not be difficult for a doctor to distinguish between these two conditions - genuine confusion in a purely medical setting would be rare.