Testimony of

Dr. Paul Epstein

February 2, 2005

STATEMENT OF PAUL E. EPSTEIN, M.D., FACP

Thank you for inviting me to testify before your committee today. My name is Paul Epstein and I am a pulmonary physician and a Clinical Professor of Medicine at the University of Pennsylvania. I have spent a large portion of my career studying occupational lung diseases and taking care of people who have been exposed to a variety of toxic materials at work.

I would like to describe a little about the diagnosis of dust-related diseases of the lung. When an individual inhales certain types of potentially toxic dust, the lung may react by developing scar tissue. This combination of the presence of dust in the lung and the development of scar tissue is known by the medical name, pneumoconiosis.

There are several different types of pneumoconiosis. The most common types are asbestosis and silicosis. Both asbestosis and silicosis are caused by long-term inhalation and retention of each particular kind of dust in the lungs. Although each of these diseases requires a substantial amount of dust retention, a longer and more consistent daily exposure to silica dust is required to produce silicosis than the amount of asbestos needed to produce asbestosis. Lung diseases like asbestosis and silicosis are both characterized by scar tissue formation and take a long time to develop after the initial exposure. The time lapse between exposure and the onset of lung disease related to that exposure is called the latency period and for both asbestos and silica exposure, the latency period is at least 20 years. There is an individual susceptibility to the scar-producing effects of both asbestos and silica so that if two individuals work side-by-side, one may develop the disease while the other may not.

While both diseases share common factors, such as dust inhalation, scar tissue formation and a long latency period, each of them have very different clinical appearances and can be recognized easily by their relatively distinct patterns of abnormality on chest x-rays. For instance, asbestosis produces a linear, streaky, or feathery pattern on the chest x-ray, predominantly in the lower portions of both lungs. This pattern of asbestos-related scar tissue formation is almost always accompanied by patches of thickening of the membrane that covers the outer surface of the lung. These thickened patches are known as pleural plaques or pleural thickening. Frequently, the pleural plaques caused by asbestos exposure contain calcium that can be seen on the chest x-ray.

Silicosis has quite a different appearance on the chest x-ray. In this disease, the deposits of scar tissue occur as distinct, rounded nodules, similar to the appearance of buckshot and they are seen predominantly at the top of the lungs. The rounded nodules of silicosis are not accompanied by pleural plaques or pleural thickening. In other words, the x-ray appearances of these two dust-related diseases are vastly different.

Abnormalities on breathing tests are also somewhat different in people who have asbestosis as compared to those who have silicosis. In asbestosis, the characteristic changes cause a restriction in the amount of air that can fit inside the lungs and there is a decrease in the efficiency of lung tissue in taking up oxygen. These changes occur relatively early in the evolution of asbestosis, even when chest x-ray abnormalities are mild. On the other hand, people with silicosis often have no abnormalities on their breathing tests until the rounded nodules proliferate in great numbers and become larger in size. At that point, the volume of air in the lungs may decrease and there may be a decrease in the person's ability to exhale air rapidly from the lungs.

When people have both diseases, (that is, both asbestosis and silicosis) the characteristic clinical and x-ray manifestations are each discernible as separate features and the diagnosis of dual disease processes can be made with relative ease.

Over the course of the past 30 years I have personally examined approximately 17,000 individuals who have been occupationally exposed to asbestos. These workers have held many different jobs, including those of shipyard workers, oil refinery employees, construction workers, steel mill employees, chemical workers, insulators, electricians, painters, and riggers, to name a few. Additionally, I have evaluated many workers who were occupationally exposed primarily to silica, including coal miners, sandblasters, stone-quarry workers, glass makers, and refractory brick manufacturers. A large number of these workers were exposed to both silica and asbestos.

While it is theoretically possible to have combined disease consisting of asbestosis and silicosis, it has been my clinical experience that the overwhelming majority of patients I have seen with asbestos-related diseases have had no evidence of silicosis. In fact, I can recall no more than a dozen or so individuals who have had combined asbestosis and silicosis and these were people who had substantial occupational exposure to silica, often in jobs that were separate from their subsequent jobs that involved exposure to asbestos. For this reason, it is my professional opinion that the dual occurrence of asbestosis and silicosis is a clinical rarity.