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PLAINTIFF'S EXHIBIT
TPI-63

September 27, 1989

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P.O. Box 1210
Tyler, TX 75710-1210

Re: Anderson, et al vs. Pennsylvania Glass Sand Corporation, et al
Cause No. 87-1403-A

Dear Mr. Bailey:

In preparation for this preliminary report, the undersigned has reviewed your letter of September 14, 1989, and accompanying documents which include the following:

1. Depositions of Matthew S. Moore, a ceramic engineer employed by Kilgore Ceramics. The first of these depositions was taken on March 18, 1986. The second deposition is dated November 16, 1988.

2. A three-part deposition taken of Johnnie E. Chanler on November 3, 1988, November 7, 1988, and November 8, 1988.

3. A deposition of Vernon Edney, taken April 17, 1986.

4. The deposition of Ernest Johnson, taken April 17, 1986.

5. The deposition of Noal Musslewhite, taken January 23, 1986.

6. The deposition of Jonnie Thompson, taken August 2, 1984.

In addition to the above-described documents, a series of OSHA inspection reports from the years 1977 and 1978, relative to violations involving the Tyler Pipe Company, were provided for review.

BACKGROUND

According to information contained in the above-described documents, each of the plaintiffs was significantly exposed to silica-containing dust and talc in the performance of their jobs with Kilgore Ceramics and Tyler Pipe Company. We find that Jonnie Chanler was employed by Kilgore Ceramics for 25 consecutive

years. During that employment, he worked as a mold hustler, a kiln fireman, and a caster. Vernon Edney was first employed as a night clean-up man, later moved to the shipping department, and finally worked the balance of his career in three separate areas: dusting molds, making slit, and trimming as a caster. Ernest Johnson was employed by Kilgore Ceramics for 16 years. He describes his work as operating a mud bucket, mold hustling, and in the spray department, specifically performing a function called "blowing out." Mr. Musslewhite started to work with Kilgore Ceramics in 1950, working in the slip room for 5-6 years, and then as a caster for the balance of his career with the company. He reports his first injuries in 1984. Jonnie Johnson worked for the Tyler Pipe Company for 24 years, performing various jobs during that employment.

Each of the above plaintiffs has been diagnosed as having occupational lung disease, pneumoconioses, and silicosis.

DISCUSSION OF PNEUMOCONIOSES-SILICOSIS

Silicosis is described as a parenchymal lung disease produced by the inhalation of respirable particles of crystal and silica and a tissue reaction to the retained silica dust. Some common forms of silica include quartz, cristobalite, and tridymite. Amorphous forms of silica include diathomite. Less fibrogenic silicates that produce different pneumoconioses include silicates such as asbestos, kaolin, and talc. Theories of the pathogenesis included antigenic theory, phospholipid fibrogenic theory, the extended solubility theory, and the solubility theory.

In a development of silicosis and silica-related pneumoconioses, the particle size of the silica is important. Sizes between 0.5 micrometers and 5.0 micrometers are the most liable to cause silicosis. Larger particles are removed by the upper air passageways. In addition to particle size, the concentration of dust particles in the air breathed determines whether silicosis will occur. Threshold values for these concentrations have been developed. The NIOSH Pocket Guide to Chemical Hazards, published in September of 1985, indicates two listings for silica, one form is amorphous silica, the second is crystalline silica. The document provides both a NIOSH recommendation and an ACGIH (American Conference of Government Industrial Hygienists) standard. 50 micrograms per cubic meter of free respirable silica is the standard established by ACGIH.

In OSHA inspection information provided on the Tyler Pipe Company, this value has been exceeded in several workplace situations.

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In Mr. Moore's deposition concerning work conditions at the Kilgore Ceramics Plant, he indicates that the silica standards have been exceeded in certain work areas in that process.

In a book entitled, "Occupational Diseases: A Guide to Their Recognition," revised in June of 1977, and published by the U. S. Department of Health, Education and Welfare jointly for the Public Health Service Center for Disease Control, and National Institute for Occupational Safety and Health, we find a section entitled, "Pneumoconioses," This term is defined as literally meaning "dust in the lungs," with dust being further described as an aerosol composed of solid, inanimate, particles. These particles, when inhaled, are carried into the lungs by the air during the inspiratory phase of respiration. The upper respiratory tract is anatomically designed to trap some of the larger particles so that only materials with diameter 5 microns or less are considered capable of conduction through the upper respiratory system, subsequently gaining admission to the lung. In an effort to classify the different forms of pneumoconioses, Nagelschmidt has proposed four types of reactions which provide some method of distinguishing the various forms of this pulmonary disorder.

Nagelschmidt describes a hyaline-nodular fibrosis as being classical for silicosis. Other types of reactions include a simple pneumoconioses of coal minors, mixed dust pneumoconiosis and diffuse interstitial fibrosis.

Classic lesion of silicosis is the silicotic nodule. Early in the course of silicosis, these nodules appear adjacent to, or on the walls of, the respiratory bronchioles. The nodule is thought to result from the death of macrophages and the subsequent fibrosing is the product of enzymes released by these macrophages. Macrophages, which ingest silica particles, subsequently die, releasing a potent intracellular enzyme which promotes further fibrosis, accounting for the progressive nature of this disease. The upper lobes of the lung and hyalar lymph nodes are usually more severely affected than the lung base. In the simple form of silicosis, these nodules remain isolated. As the disease progresses, the nodules crowd closer together until they appear to form a continuous mass of fibrous tissue. In the complicated form of silicosis, the immunological protective mechanism of the lung is adversely affected, creating an environment of susceptibility to bacterial and viral complications. Silicosis is often associated with pulmonary hypertension and cor pulmonale, most likely as a consequence of damage to the walls of the blood vessels as a result of mechanical obstruction.

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In a book entitled, "Medical Toxicology, Diagnosis and Treatment of Human Poisoning," by Ellenhorn and Barceloux, we find a section entitled, "Occupational Lung Disease." In a subsection entitled, "Pneumoconiosis," the authors indicate that this type of pulmonary disease results from the deposition of dust particles in the lung. The authors indicate that the deposition of the particles depends on the physical forces that move the particles and the airway anatomy. Particles with diameters of 5 to 30 micrometers are generally deposited in the nasopharyngeal region by impaction. Particles with diameters of .5 to 5 micrometers provide the greatest danger in that they reach the alveoli and the terminal bronchioles. A simple pneumoconiosis is then defined as a diffuse nodular fibrosis which develops from heavy exposure to certain dusts, including coal workers' pneumoconiosis (CWP) and silicosis. The lesion is described as a focal nodule with a specific terms called the "coal macule" in CWP and silicotic nodule in silicosis.

The authors indicate that the interstitial pulmonary fibrosis of silicosis may continue to progress despite cessation of exposure. Silicosis increases the susceptibility to microbacterium, tuberculosis, atypical bacteria and fungi, by suppressing immune reactions. In the complicated phase of pneumoconiosis, there is progressive massive fibrosis involving the coalescence of large nodular lesions, usually in the upper lobes of the lung and usually associated with parenchymal destruction. This chapter contains a diagram which indicates the various lung volume definitions referred to in the pulmonary testing. A table of the severity of pulmonary function abnormalities is also provided and is consistent with Dr. Kagal's testimony concerning the forced vital capacity's relationship to obstructive changes in the lung and the expiratory volume changes consistent with restrictive changes in the lung. In a table which describes common occupational lung diseases (partial list), free silica is listed first, producing silicosis due to exposure in hard rock or metal mining, foundry work, sand blasting, pottery industry, slate industry.

In a document issued by the National Safety Council, identified as I-531-REV80, the subject of dust, fumes and mists in industry is considered. In a section titled, "Pneumoconiosis," the term is once again defined as "Dusty lung." In the subsection entitled, "Silicosis," the authors indicate that this is the most important lung disease caused by the inhalation of mineral dust. Exposure to crystalline-free silica dust may occur in such industries as foundries, glass manufacturing, granite cutting, mining, and tunneling in quartz rock. Silicosis has been described by

other names in the past, such as miners' asthma, grinders' consumption, and stone masons' disease. In each case, the dust from crystalline-free silica is the cause of the pulmonary disorder. Silicosis is defined as a disease due to breathing air containing silica, characterized anatomically by generalized fibrotic changes and the development of a miliary nodulation in both lungs, and clinically by the shortness of breath, decreased chest expansion, lessened capacity for work, absence of fever, increased susceptibility to tuberculosis, and by characteristic x-ray findings.

Factors which influence the development of the disease silicosis include the following: the amount and kind of dust inhaled, the percentage of free silica contained in the dust, the form of the silica, the size of the particles inhaled, the duration of the exposure, the power of resistance of the individual concerned, and the presence or absence of a complicating process such as infections. It is generally accepted that the silicotic fibrosis is not caused by the hardness or sharpness of the particles, but by a combination of the slight solubility of the material with a physiochemical effect, and an immunological effect.

The National Safety Council document describes three stages of silicosis. The first stage of the disease produces no disability. The individual affected is allowed to work as usual and is unaware that anything is wrong. At this point, the disease would be revealed only by an opaque nodular shadow in the chest x-ray and would have to be associated with known exposure to crystalline-free silica. In the second stage, respiration may be affected in some persons, demonstrating labored breathing on heavy exercise and a dry cough. The third stage may develop even though the individual has been removed from exposure to silica dust. The progress of the disease may be slower without continued dust exposure. Breathing becomes severely labored. The worker is determined to be susceptible to respiratory diseases at this point. Chest x-rays may show an enlarged heart as a result of the body's attempt to overcome the resistance of restricted blood vessels in the lungs. Pulmonary tuberculosis is a frequent complication and occasionally results in death. Silicosis seldom develops in less than five years and, in many cases, takes 20 years or longer to become disabling.

In describing the action of the silica on the lungs, the authors indicate that the silica dust is deposited and accumulates, and a fibrous tissue develops and grows around the particles. This fibrous tissue is tough, like scar tissue, and is not as elastic as the normal lung tissue. The passage of oxygen and carbon

dioxide is, therefore, restricted, which ultimately reduces the available function volume of the lung. The presence of fibrous tissue will slow down or even prevent the diffusion of oxygen from the lung to the blood in the capillaries. The effect, here, is to tend to limit the rate at which oxygen is supplied to the body tissues. Emphysema is the most obvious symptom.

In a series of books dealing with the "Physiological Principles in Medicine," published by University Park Press, we find one entitled, "Respiratory Disorders," by Cameron & Bateman. In a chapter entitled, "Diffuse Interstitial Fibrosis," the authors describe the pulmonary capillary bed as being distorted by interstitial fibrosis and in turn producing a decrease in pulmonary capillary volume. The reduced efficiency of gas exchange is further compromised by thickening of the alveolar membrane. The lungs, at this point, are described as small, stiff, and inefficient. The authors give as a clinical example of this process silicosis and indicate that the silica produces a low-grade inflammatory process which leads to extensive fibrosis.

In "The Textbook of Pulmonary Diseases," Second Edition, by G.L. Baum, we find a chapter entitled, "Silicosis, Asbestosis, and Talc Pneumoconiosis." The authors define silicosis as a disease of the lung caused by inhalation of free, or uncombined, silicon dioxide, characterized by discrete or conglomerate nodular masses of fibrous tissue in the lung and associated lymph nodes. The authors refer to the lesion of simple silicosis as a hyalinized nodule. In a subsection entitled, "Clinical Signs and Symptoms," the authors indicate that there may be no clinical signs or symptoms in the early stages of silicosis, and it has been established that it might take 20-30 years for the disease to develop to a clinically obvious state.

As the disease is developing, respiratory symptoms may be related to respiratory infections, or the effects of tobacco smoke. The chronic bronchitis, cough and sputum production associated with cigarette smoking may not at first be related to silica exposure. With regard to pulmonary function, the authors indicate that, at the uncomplicated, simple stage of silicosis, there may be no objective evidence of any abnormal function. If the subject is a heavy smoker, some evidence of airway obstruction may be evident.

When conglomeration of the nodules occurs, the functional picture is usually that of severe airway obstruction. Tests of maximal voluntary air flow will be impaired to a variable degree and specific airway resistance is increased. The ratio of residual volume to total lung capacity is usually considerably increased.

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At rest, the arterial blood gases are usually minimally effected. In a severe obstructive disease process, arterial PO_2 may be below normal levels. As the disease progresses from the simple to the complicated form, conglomerate lesions develop producing the complication of obstructive airway diseases which may be related to the extent of the patient's smoking. Tuberculosis, as a complication, becomes a statistically significant consideration.

The authors indicate that one complication which the patient with silicosis is usually spared, is that of primary bronchiogenic carcinoma, unless he is a heavy cigarette smoker, in which case the risk imposed by smoking history increases the potential for development of cancer. The authors go on to indicate that, in patients with severe obstructive lung disease complicating their silicosis, they may succumb to numerous and often fatal complications associated with the obstructive disease, including pulmonary thromboembolism, respiratory insufficiency, and failure, recurrent pneumonitis, pneumothorax, and cor pulmonale with right ventricular hypertrophy and cardiac failure.

In a book entitled, "Pulmonary Diseases and Disorders," by Alfred Fishman, the chapter entitled, "Disorders of Pulmonary Interstitium," includes a subsection dealing with silicosis. The author, once again, indicates that silicosis is a pulmonary fibrosing process produced by respirable dust containing crystalline-free silica. The author indicates that exposure produces disseminated discrete hyalinized nodules within the lung, coalescence of small foci into enlarged fibrotic masses is common. Silicosis is described as a cellular reaction to inhalation and alveolar deposition of free silica particles ranging from 1 to 3 micrometers in diameter. The foundry industry is included with industrial activities associated with silicosis. The authors go on to consider various complications of silicosis including susceptibility to infection, and various immunological abnormalities. Classification of the nodules is also described within the classification of the ILO U/C for grading of co-workers' pneumoconiosis.

In a publication from the New York State Department of Health, entitled, "Occupational Medicine Current Concepts," Volume 2, #4, December, 1979, we find an article entitled, "Non-Asbestos Occupational Pulmonary Disease," by Stuart A. Levy, M.D. Silicosis is considered first among the non-asbestos occupational pulmonary diseases. The author first discusses the difficulty encountered in establishing a satisfactory threshold limit value for silica. The author indicates that short exposures to high concentrations

of silica is not equivalent to longer, less intense, exposures, although the total exposure may be identical. The author further points out that the concomitant exposure to other dusts may alter the response of the lung to silica. He cites specifically the apparent protective effect of iron hydroxide, blocking significant fibrotic reaction to silica in iron miners.

The author indicates an increasing body of evidence that the fibrotic reaction to the presence of silica in a lung is the result of an immune reaction. He points to the fact that antinuclear antibodies are found in approximately 1/2 of the silicotic sandblasters. There is evidence of higher-than-expected incidences of connective tissue diseases, including systemic lupus erythematosis, glomerular nephritis, and sclera derma, in workers with silicosis. The fibrotic process may be rapidly accelerated by the influence of rheumatoid factor or superimposed tuberculosis.

The author speculates that silica particles in the lung may point out self-perpetuating fibrosis in the absence of further exposure. The particles are initially ingested by macrophages which eventually disrupt, liberating the silica and a substance which initiates the immune fibrotic reaction. The silica released can be ingested by another macrophage, repeating this same reaction. Silica has been shown to impair the ability of macrophages to inhibit the growth of tubercle bacilli, which would in part explain the higher-than-expected incidence of tuberculosis in silicotic workers.

Dr. Levy refers us to a publication in the Journal of Occupational Medicine, in 1979, which provides preliminary evidence that silicosis may predispose to the development of lung cancer.

A computer-based search of the literature, cross-indexing the term "silicosis" with the term "all sand" produced the following citations:

1. A paper entitled, "Pulmonary Silicosis and Disseminated Lupus Erythematosis," appeared in a foreign-language journal, Poumon. Coeur. 1983; 39(4): 205-207. The abstract of that paper describes a 59-year-old sand blaster with histologically-proven silicosis, complicated by systemic lupus erythematosis. The abstract contains a notation that the connection between silicosis and SLE lies in changes in humoral immunity.

2. A second article entitled, "Does Occupational Exposure to Silica Cause Lung Cancer?", by Goldsmith and Co-authors, is a

review article which appeared in the American Journal of Industrial Medicine, 1982; 3(4): 423-40. The author indicates that, although silica is generally not considered to be a carcinogen, occupational exposure to crystalline silica has been associated with excessive rates of lung cancer mortality. Iron and steel foundry workers are specifically cited within the occupational groups considered by the authors. The abstract refers to a Swedish Pneumoconiosis Register, and an Ontario Ministry of Labor document, both of which indicate silicosis statistically related to significant increases in risk of lung cancer mortality. Various mechanisms of carcinogenic activity are considered.

3. In the Journal of the National Cancer Institute; 77(4); 1986, 883-890, is an article entitled, "Silica Dust and Lung Cancer: Results from the Nordic Occupational Mortality and Cancer Incidence Registers." The authors indicate that although autopsy studies have failed to establish a relationship between silicosis and lung cancer, that recent epidemiological studies have indicated a positive association and an excess lung cancer risk has been observed in some occupational groups with exposure to silica dust.

4. Landrigan and Co-authors describe, "Silicosis in a Gray Iron Foundry. The Persistence of an Ancient Disease," in the Scandinavian Journal of Work, Environment. Health: Volume 12, Issue 1, 1986, 36-9. The article deals with the presence of silicosis in foundry workers.

5. An article entitled, "Experimental Silicosis II. High Temperature Effects on Fibrogenic Action of Quartz and Quartz Sands Used in the Ceramic Industry," by Goscicki and Co-authors, published in Med. Tr.; Vol. 32, Issue 2, 1981, 109-118. The authors attempt to study various quartz samples after heating for 72 hours at 1200 degrees. The authors found no visible relationship between the fibrogenic properties of dust and temperature-induced changes in their chemistry and phase composition.

TALCOSIS

Talc is a form of magnesium silicate which may be either relatively pure or may be mixed with tremolite or with dolomite. Talc is generally a term applied to carbonate mixtures that have the same general feel and property. An aluminum silicate, known as pyrophylite, is frequently mixed with a high percentage of quartz. The free silica generally found in pyrophylite can cause silicosis. Talcosis is usually associated with the tremolite-type talc. This disease produces changes in the lungs and symp-

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toms similar to those of asbestosis.

CONCLUSION

As a Board Certified Toxicologist, and a Professor of Pharmaceutical Sciences at St. John's University, the undersigned would conclude that each of the plaintiffs cited earlier in this report have described work conditions which are consistent with those which would produce exposure to significant amounts of airborne silica-containing materials. These silicate products were inhaled over extended work histories, ranging from 16 to 34 years. Even when individuals were not employed in an area of the factory that was recognized as a high-dust area, the factory environment produced significant silica exposures. The dust in the environment was clearly visible, and ventilation systems, described in the various depositions, appeared to be inadequate for alleviating this condition.

Very truly yours,

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