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June 28, 1999

Mr. Mike Hutchins
Hawkins and Parnell, LLP
4000 Suntrust Plaza
303 Peachtree Street
Atlanta, GA 30308-3243

Re: Warren v. Swan Transportation (Tyler Pipe)

Dear Mr. Hutchins:

You have asked me to provide opinions concerning the plaintiff's (Mr. Odis Warren) asbestos exposure while an employee of a pipe and pipefittings foundry—Tyler Pipe--located in Tyler, Texas. Further, you have requested that I opine on the state of knowledge regarding asbestos that the company could have had during the period of Mr. Warren's employment—1962 to 1975.

I base my opinions on the following:

- Two visits and tours to the foundry (February 11 and March 10, 1999)
- Detailed interviews of company representatives (Mr. Olin. Jackson, Mr. Charles Kuenemann, et al.)
- Depositions given by Mr. Olin Jackson and Mr. Paul Lowry dated February 15, 1991.
- A summary of an OSHA industrial hygiene inspection dated May 4, 1979
- The industrial hygiene literature in the public domain and unpublished articles
- The asbestos literature in the public domain and unpublished articles.
- My 24 years as a practicing industrial hygienist

Exposure

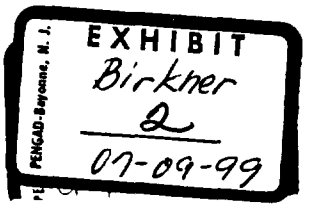
A detailed tour of the foundry with Mr. Olin Jackson (a former foundry manager) led me to identify four uses of asbestos during the period of Mr. Warren's employment. They are:

- Asbestos cloth used to cover hot air ductwork block insulation of the cupola (metal melter).

- Asbestos cloth covering block insulation of the core oven fireboxes.

- Asbestos cloth (called sheeting by the foundry) used for a period of time to cover blowboards on the core machines in the shell core area.

- Gloves, coats and jackets used sporadically at the cupola.



I did not observe old pipe insulation anywhere in the foundry, nor did I observe any other obvious potential asbestos applications in the foundry. The limited use of asbestos-containing materials ceased at the foundry in 1980.

The foundry advised me that Mr. Warren worked as a shell core machine operator and had used but did not construct the blowboards. Thus Mr. Warren's exposure can be estimated as follows.

> Marr[i], in 1964 reported *concentrations* between 0.3 and 1.8 million particles per cubic foot of air (mppcf) for installation of 80 to 95% chrysotile asbestos cloth in shipboard operations. Such operations use large volumes of cloth, requiring significant cutting and manipulation, in a poorly vented spaces. Applying the correlation of 6 fibers/mppcf reported by Ayer, et al[ii], this equates to fiber *concentrations* of 1.8 to 10.8 f/cc. Blowboard construction on the other hand is a low volume task in a well ventilated (by fans and convection) work environment. Airborne *concentrations* are conservatively estimated to be one order of magnitude below those reported by Marr or 0.18 to 1.08f/cc in the breathing zone of the core machine setup operator.
>
> If, conservatively, an average of 33 blowboards are constructed during each eight-hour shift for 22 operating core machines, taking 5 minutes/blowboard or a total of 165 minutes/shift, the time-weighted average core machine setup operator exposure is estimated to be between 0.062 to 0.37f/cc.
>
> Blowboards in use in the core machines do not appear to have added to employee work room concentrations. Thus core-machine operators would have had exposures significantly below (estimated to be approximately half an order of magnitude less) the core machine setup operator or a time-weighted-averages of between 0.012 and 0.074f/cc.

Shell core operator exposures were well below even today's regulatory level (0.1f/cc) and all previous applicable exposure limits. Thus, Mr. Warren, having no other asbestos exposure at Tyler Pipe, was not exposed to asbestos dust levels during his employment at Tyler Pipe, which are known to cause disease. A 1979 OSHA industrial hygiene evaluation conducted in the North Plant Core Room Maintenance Shop supports this opinion. Pathology should further underpin this opinion.

State of the Art

The 1960s were a period of scientific turmoil with respect to the development of asbestos knowledge in the medical community. Asbestos-related pneumoconiosis while first seen in 1900 was not formally recognized until Cook's work[iii] in the mid-1920s. Similarly, suspected asbestos-related lung cancer was first discussed in medical case reports in the mid-1930s, but took nearly 40 more years to become scientifically accepted, publicly recognized and regulated.

Knowledge grew slowly over that period through case reports, autopsy series and a few epidemiologic studies. Confounding the development of scientific and public health knowledge in this area was the long latency of the asbestos-related diseases, the rapid growth in cigarette smoking and related lung cancer, and the public health issues

associated with pneumoconiosis. For example, a physician reviewing the topic of pneumoconiosis in Index Medicus (where asbestos related articles were indexed), between 1925 to 1956 would have barely noticed the 25 articles on asbestos-related lung cancer mixed in with thousands of articles on pneumoconiosis. Further, Enterline[iv] reports that between 1935 and 1965 there were only 104 significant asbestos-related lung cancer *writings* (articles, case reports, book chapters, literature reviews, etc.), and 61 *writings* on asbestos-related mesothelioma (mostly after 1960). Some articles supported the asbestos disease relationships and others refuted it. In 1974, Selikoff[v] commenting on the *scientific research* of the same period notes:

> "...from the early 30's to the early 60's, there was virtually a total absence of scientific research into the problem. Therefore when, in 1969, 70 and 71, decisions were to be made, we didn't have a great deal on which to rely."

After Selikoff's historic conference addressing the biologic effects of asbestos in 1964, a number of residual questions remained. They were discussed and placed on the research agenda of the International Union Against Cancer[vi]. Three of the key issues were:

- *That the importance of fiber type on the risk of developing asbestosis, carcinoma of the lung and mesothelial and other tumors be investigated.*

- *That the relationship of dust dosage (including concentration and duration of exposure), and the composition and physical state of the dust to the incidence of asbestosis, carcinoma of the lung, mesothelial and other tumors be studied.*

- *That further investigations be made of past and all future cases of diffuse mesothelial tumors of the pleura and peritoneum to establish any association with asbestos and other factors.*

A consensus view on these questions was not achieved until 1972 when the International Agency on Research on Cancer (the International Union Against Cancer merged its work with IARC) issued its report on asbestos and cancer[vii]. Indeed research and the debate on some of these issues continues today. Another focus of research and debate over the last 25 years is what types of workers were at risk of disease and what were their exposures--research and debate continues here too.

Exposure Limits and Regulations

The first regulations aimed at reducing exposure in asbestos textile manufacturing were promulgated in 1933 in Great Britain, while no exposure limits were stated, dust controls were specified. In 1946, the American Conference of Governmental Industrial Hygienists, based on available literature, adopted a Maximum Allowable Concentration of 5 million particles per cubic foot of air (mppcf). In 1958, the Federal Government, through the Walsh Healy Act, and a number of states, adopted the 5mppcf level which by then was designated a Threshold Limit Value (TLV). This limit was accepted as the

"safe" limit[viii] by the medical and industrial hygiene community until the early 1970s. In 1968 Balzer and Cooper[ix] suggested that this number may be two high, based on an industrial hygiene-medical study of insulation workers. In 1969, Walsh Healy reduced the exposure limit to 2mppcf or 12fibers/cc (5f/cc). In 1971, OSHA issued an Emergency Temporary Standard reducing worker exposure to 5fiber/cc, and in July 1972 they issued a permanent standard of 5f/cc until 1976, when the limit would automatically be reduced to 2f/cc. Taking this action, OSHA stated:

> *...it appears that levels of exposure which may be safe with regard to asbestosis are not safe with regard to mesothelioma; because the statute requires the protection of every employee, even of ones who may have regular exposure to asbestos during a working life which may reach, or even exceed 40 years...the conflict is resolved in favor of the health of employees. As of July 1, 1976, TWA concentrations of asbestos fibers longer than 5 micrometers will not be allowed to exceed two fibers/cc with a ceiling value of 10 fibers/cc.*

Since 1976, OSHA has reduced its exposure limit to 0.1 f/cc, based on an ever-growing body of science addressing asbestos.

Opinions

1. To a reasonable degree of scientific certainty, while an employee of Tyler Pipe, Mr. Warren was not exposed to levels of asbestos that exceeded the exposure standards that existed between 1962 and 1975. Further, Mr. Warren was not exposed in excess of the rigorous standards that exist today, and thus he was not exposed to asbestos levels known to cause asbestosis or lung cancer.

2. Prior to the promulgation of the OSHA Emergency Temporary Standard in December 1971, and the subsequent permanent standard in July 1972, Tyler Pipe would have had no obvious reason to know, understand, or seek out medical or industrial hygiene information on asbestos exposure or exposure controls.

At trial I will testify to these and other issues which have been previously disclosed to plaintiff's counsel.

Sincerely yours,

Lawrence R. Birkner, CIH

LRB:hol
Cc: Mr. Joe Harrison, Esq.—Guardere & Wynne, LLP

References

[i] Marr, WT, Asbestos Exposure During Naval Vessel Overhaul, AIHA Journal 25:264-268 (1964)
[ii] Ayer, HE, Lynch, JR, and Fanney, JH. The Comparison of Impinger and Membrane Filter Techniques for Evaluating Air Samples in Asbestos Plants. Ann. NYAS, 132:274-287
[iii] Cooke, WE. Pulmonary Asbestosis. Brit Med. J. 2:1024-1025
[iv] Enterline, PE. Changing Attitudes and Opinions Regarding Asbestos and Cancer, 1934-1965. Am J. Ind Med. 20(50):685-700 (1991)
[v] Selikoff, I.J. Asbestos Criteria Document Highlights, ASSE Journal, March 1974.
[vi] Report and Recommendations of the Working Group on Asbestos and Cancer. International Union Against Cancer. Ann. NYAS 132:706-721
[vii] Report of the Advisory Committee on Asbestos Cancers to the Director of the International Agency for Research on Cancer. October 1972.
[viii] Isselbacher KJ, Klaus, H and Hardy, HL Asbestosis and Bronchogenic Carcinoma, Am. J. of Med. November 1953 p721-732
[ix] Balzer, JL and Cooper, WC. The Work Environment of Insulating Workers. AIHAJ 29:222-227 (1968)

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APPENDIX B

SURVEY METHODS/PROCEDURES

Monitoring, Airborne Contaminants

Personal breathing zone air samples were conducted to evaluate actual worker exposure levels, as follows:

Airborne Contaminant	Collection Medium	Sampling Equipment
Redacted		
Silica	PVC Filters/ Cyclones	Gilian HFS-113
Redacted		

Equipment Calibration

All air pumps were calibrated before and after the survey by means of the soap bubble/air displacement method. No significant deviations were noted.

Sample Analysis

Analytical sample evaluations were performed by the Ctek Industrial Hygiene Laboratory located in Dallas, TX. This laboratory is accredited by the American Industrial Hygiene Association (AIHA). National Institute for Occupational Safety and Health (NIOSH) and/or other appropriate standard analytical methods were used as noted below:

Analysis For:	Method
Redacted	

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Silica	7500

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APPENDIX C
EXPOSURE STANDARDS/GUIDELINES

1. OSHA

AIR CONTAMINANTS:

OCCUPATIONAL SAFETY & HEALTH ADMINISTRATION (OSHA - "AIR CONTAMINANTS 29 CFR 1910.1000")

This general standard provides a listing of numerous potential air contaminants and the permissible exposure limit (PEL) for each material listed. According to this standard, these PEL's may not be exceeded for any 8 hour work shift of a forty hour work week, on the basis of an 8 hour time weighted average (TWA) exposure determination. Time weighted averages permit excursions above the PEL provided that they are compensated for by equivalent excursions below the PEL during the work day.

Some substances, however, have supplemental limits designated as **Short Term Exposure Limits (STEL)** and **Ceiling Limits**, to control these excursions above the 8-hour TWA. The STEL is the 15 minute TWA which shall not be exceeded at any time during the work shift. On the other hand, ceiling "C" limits must not be exceeded for any time. The "**skin**" notation designates substances which may be absorbed directly through unbroken skin and/or the mucous membranes and eyes, thus contributing to the overall exposure potential.

PEL's may be expressed in either parts of the specific substance per million parts of air (ppm) or milligrams per cubic meter of air (mg/m^3). Appropriate excerpts from Table Z-1 of Standard 1910.1000 are shown below:

TABLE Z-1

Chemical	PEL
Silica, Quartz	0.1 mg/m3

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2. ACGIH - American Conference of Governmental Industrial Hygienists

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH)

In it's publication: "TLV's- Threshold Limit Values For Chemical Substances In The Work Environment Adopted By ACGIH With Intended Changes For 1991-1992", ACGIH has established recommended exposure guidelines for a wide variety of potential air contaminants. As defined in the preface of the listing, "Threshold limit values refer to airborne concentrations of substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed day after day without adverse effect." Appropriate selections from this listing are shown below:

Substance	TLV-TWA
Silica, Quartz	0.1 mg/m3

"TLV-TWA" - (Threshold Limit Value - Time Weighted Average)

The time weighted average concentration for a normal 8 hour work day and a 40 hour work week, to which nearly all workers may be repeatedly exposed day after day, without anticipated adverse effects.

mg/m^3 - Milligrams of contaminant per cubic meter of air.

A1 - Chemical listed as a "Confirmed Human Carcinogen".

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APPENDIX D

HEALTH EFFECTS

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Silica, Crystalline

Crystalline silica or quartz dust causes silicosis, a form of disabling, progressive, and some times fatal pulmonary fibrosis characterized by the presence of typical nodulation in the lungs. The clinical signs and symptoms of silicosis tend to be progressive with continued exposure to quantities of dust containing free silica, with advancing age, and with continued smoking habits. Symptoms may also be exacerbated by pulmonary infections and cardiac decompensation. Symptoms include cough, dyspnea, wheezing, and repeated nonspecific chest illnesses. Impairment of pulmonary function may be progressive. In individual cases there may be little or no decrement when simple discrete nodular silicosis is present, but when nodulations become larger or when conglomeration occurs, recognizable cardiopulmonary impairment tends to occur. Progression of symptoms usually continues after dust exposure ceases. While there may be a factor of individual susceptibility to a given exposure to silica dust, the risk of onset and the rate of progression of the pulmonary lesion is clearly related to the character of the exposure (dust concentration and duration). The disease tends to occur after an exposure measured in years rather than months. Occasionally, exposures to very high concentrations occur in short periods of time in occupations such as sandblasters and tunnel workers; in these cases of acute or rapidly-developing silicosis there may be severe respiratory symptoms resulting in death. It is generally accepted that silicosis predisposes to active tuberculosis, and that the combined disease tends to be more rapidly progressive than uncomplicated silicosis.

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APPENDIX E

GRINDING TABLE LAYOUT