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PLAINTIFF'S EXHIBIT
USX-1066

November 9, 1998

Eric L. Horne
Eckert, Seamans, Cherin & Mellott
600 Grant Street
44th Floor
Pittsburgh, Pennsylvania 15219-2788

Re: Estate of Eisenreich v. CBS f/k/a Westinghouse Electric Corporation, United States Steel Corporation, et al.
GD No. 97-16440; Allegheny County

Dear Mr. Horne:

At your request, I have prepared this report concerning my anticipated "State of the Art" testimony in this case. This is a complicated subject. Included are those articles which I usually discuss on direct examination; I try to keep this presentation to less than two hours. I am certainly aware of other articles, and they may be mentioned during my trial testimony, particularly in response to cross examination.

In addition to discussing State of the Art, I usually describe the role of case reports, retrospective studies, and prospective studies in the development of the scientific and medical literature. Case reports cannot be used to describe risk, since there is no estimate of the expected number of cases. Lacking risk data, case reports cannot be used to determine causation. Retrospective studies are often plagued by bias and confounding, since the experimenters were not present when the injurious exposures occurred. Prospective studies provide the most robust data; such studies did not become available for asbestos until after Dr. Selikoff began publishing

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in the 1960's.

I also review the epidemiologic method of proving causation, often referred to as the Bradford-Hill criteria. These criteria include the strength, consistency, specificity, biological plausibility, dose-response, temporal relationship, and statistical significance of the association. Because of the need for replication (i.e. consistency) of data, and the long latency for developing asbestos-related diseases, it took decades for the health effects of asbestos to be truly appreciated.

Plaintiffs' counsel are very familiar with my testimony on these subjects. I have provided direct testimony, and been cross examined in a number of trials and depositions. I anticipate that my testimony in this case will be very similar to that given previously, and there should be no surprises.

For the sake of clarity and brevity, I have organized this report in decades:

PRIOR TO 1930

I usually begin my testimony by discussing Cooke's article published in 1927.[1] This described two cases of asbestosis. Following this article, Seiler published what is described as the first "pure" cases of asbestosis, where tuberculosis was not an issue.[2] As a result of these publications, the British government requested a survey of the textile industry, which was performed by Dr. Merewether, and reported in 1930.

1930 TO 1939

Dr. Merewether performed a survey of 374 textile workers in England. His results were reported both in England[3] and in the United States.[4] These articles described various aspects of the textile industry. Dr. Merewether concluded that asbestosis could be prevented if the dust levels could be reduced to the level pertaining to spinners. Those levels would be approximately 17 to 170 million particles per cubic foot (mppcf). Following Merewether's report, the British government passed the Asbestos Industry Regulations to reduce the level of exposure to asbestos dust. In 1933 and 1934, Merewether reviewed events to date, and concluded that keeping exposures below the dust level pertaining

to spinners would prevent the development of asbestosis.[5]

In 1935, Lanza surveyed five textile plants in the United States, and described findings in 126 people.[6] In the same time frame, other articles reviewed asbestos fabricating methods, and the effects of various methods of dust control.[7,8]

In 1938, Dreessen published a survey of four asbestos textile plants in the United States, and concluded that new cases of asbestosis would not occur if asbestos dust exposures were kept below 5 mppcf.[9] In a subsequent article, Dreessen again predicted that new cases of asbestosis would not appear if asbestos dust concentrations were kept below 5 mppcf.[10]

To summarize, by the end of the 1930's it was recognized that asbestosis was a pneumoconiosis distinct from silicosis or coal workers' pneumoconiosis, and that it was not a complication of tuberculosis. Most data came from individuals engaged in the primary production of asbestos products, such as the textile industry. It was generally believed that the disease could be prevented if exposure to asbestos dust was controlled. The 5 mppcf level recommended by Dreessen became widely accepted as a safe exposure limit, and was adopted as such by many States. The 5 mppcf level remained in force until 1968, when data indicated that new cases of asbestosis were occurring despite adherence to this standard.

1940 TO 1949

Prior to 1940, the medical and scientific literature had focussed on workers engaged in the primary production of asbestos products. In 1941, Brown reported on a survey of the New York Navy Yard pipe insulating shop and commented on findings in two other yards; no cases of asbestosis were found.[11] This was the first survey of end-users of insulation products.

In 1946, Fleischer reported a survey of 1074 pipe insulators working in four shipyards.[12] Total dust and asbestos dust concentrations during various operations were described; with one exception, the asbestos counts were below 5 mppcf. There were only 3 cases of asbestosis in this population, and it was concluded that pipe covering was not a dangerous occupation. The authors felt that the low prevalence of asbestosis was not

surprising, considering the nature of shipyard pipe covering work.

In 1946, the American Conference of Governmental Hygienists (ACGIH) adopted 5 mppcf of asbestos dust as the Threshold Limit Value (TLV). As stated above, the 5 mppcf standard was widely accepted in the United States, and remained in place until 1968, when a new TLV of 2 mppcf (equivalent to 12 fibers/cc) was recommended.

In 1949, an editorial was published in the Journal of the American Medical Association.[13] This described an increased frequency of lung cancer in people with asbestosis. The conclusion was that asbestosis was associated with an increased risk of lung cancer. Of course, it was generally believed that asbestosis could be prevented by reducing exposure to asbestos dust. Therefore, the risk of lung cancer could be eliminated by the same methods.

To summarize, by the end of the 1940's the published literature concerning end-users of asbestos indicated minimal or no risk, probably because exposure to asbestos dust was intermittent and relatively low compared to primary producers. The issue of lung cancer had been raised, but this was in the context of asbestosis, which was believed preventable by limiting asbestos dust exposure.

1950 TO 1959

In 1952, Smith described a visit to England, where he met with Drs. Gloyne, Merewether and Wyers. The consensus of opinion was that the risk of lung cancer due to asbestosis no longer existed under current working conditions.[14]

In 1953, Isselbacher and Hardy reported a case of asbestosis and bronchogenic cancer, and reviewed the existing literature.[15] They indicated that 5 mppcf of asbestos dust was accepted as a safe working concentration, and recognized the work of Doll and others implicating smoking as a cause of lung cancer.

In 1955, Richard Doll published the first epidemiological study indicating an increased risk of lung cancer in people with asbestosis.[16] He studied workers employed at a textile factory

in England and concluded that there was approximately a 10-fold risk of lung cancer compared to the general population. All of the cases included in his risk assessment had asbestosis, and all had been employed prior to 1923, at least 9 years prior to passage of the Asbestos Industry Regulations. Doll believed that the risk had become progressively less as duration of employment under the old dusty conditions had decreased.

In 1958, Braun and Truan published a study of chrysotile miners.[17] They concluded that lung cancer was not increased among the miners, or in areas contiguous to mining operations.

To summarize, by the end of the 1950's there was some epidemiological evidence that lung cancer risk was increased in people with asbestosis, though negative studies also existed.

1960 TO 1969

In 1960, Wagner published a description of 33 cases of mesothelioma associated with crocidolite exposure in South Africa.[18] He noted that pathological evidence for associating mesothelioma with asbestos exposure was not conclusive, since asbestos was found in only 8 of the 33 cases.

In 1962, the Public Health Service reviewed the amounts and uses of asbestos imported into the United States, and concluded that there were many unanswered questions concerning health effects.[19] One of the questions was whether malignancies were an occupational risk among asbestos workers.

In 1964, Selikoff reported an increased risk of lung cancer and mesothelioma in a group of 632 insulation workers.[20] In the same year, the Public Health Service again concluded that it was still unknown whether malignancies were an occupational risk among asbestos workers.[21] The Public Health Service was aware of Dr. Selikoff's findings.

In October 1964, a major conference on the health effects of asbestos was held in New York. The proceedings of the conference were published in the Annals of the New York Academy of Sciences in December 1965. Among the articles published was a paper by Selikoff describing the occurrence of asbestosis among insulators.[22] Selikoff pointed out that the only previous large

scale survey of asbestos insulation workers (Fleischer 1946) had concluded that pipe covering was a relatively safe occupation. However, Fleischer's study had included few workers employed for more than 10 years. Selikoff indicated that counts for asbestos fibers were generally less than 5 mppcf during insulation activities.

In 1965, Selikoff published a paper concerning mesothelioma.[23] He noted that crocidolite asbestos (implicated as a cause of mesothelioma by Wagner in 1960) had been imported to the United States. He questioned whether mesothelioma was due solely to crocidolite, or whether other types of asbestos could cause this tumor. The paper reported pathology and epidemiologic data indicating that mesothelioma was a problem in the United states, but available information could not determine whether crocidolite was responsible. That question was not answered until 1972, when Selikoff described 4 cases of mesothelioma in which he believed crocidolite had been excluded, and only amosite exposure had occurred.[24]

In 1968, Balzer and Cooper published a paper which questioned the safety of the 5 mppcf standard.[25] In the same year, the ACGIH recommended lowering the standard to 2 mppcf, which they equated to 12 fibers/cc. This began a series of reductions in the allowable concentration of asbestos (see below).

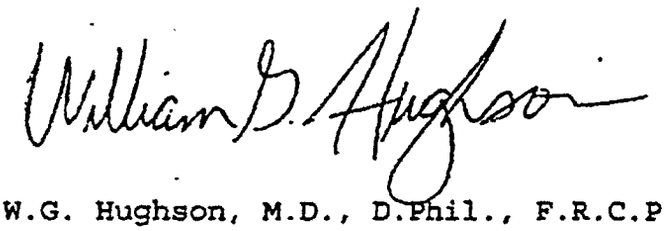
1970 AND THEREAFTER

Legislation creating the Occupational Safety and Health Administration (OSHA) was passed in 1970. In 1972, OSHA promulgated its first Permissible Exposure Limit (PEL) for asbestos; the limit was set at 5 fibers/cc as a time-weighted average (TWA). Following this, there was a succession of new PEL's in 1976, 1986, and 1994. The current PEL is 0.1 fiber/cc.

Generally speaking, I stop my discussion of State of the Art after the 1972 Selikoff article.[24] In the years since then, there have been literally thousands of articles describing asbestos exposure under different circumstances, and various actual or anticipated health consequences.

In addition to discussing State of the Art, I will describe the fact that the risk of mesothelioma rises as latency increases. As described by Peto, the risk of mesothelioma increases as time passes, where time is raised to the power of 3-4 (i.e. t^{3-4}).[26] Thus, earlier exposures are more important than later exposures in causing mesothelioma.[27]

Sincerely,



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