PLAINTIFF'S EXHIBIT

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Mr. Ollie Harton,
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Re: Asbestos containing electrical wire
Date: 09/30/96

Dear Mr. Harton.

I have reviewed a series of industrial hygiene studies regarding asbestos containing electrical wire. From my perspective as an occupational pulmonary medicine specialist, I am pleased to submit the following opinions.

1. Maxim Engineers, 1990

On 12/03/90 Maxim Engineers, Inc. a Dallas, Texas engineering consultation firm, released a report titled **Industrial Hygiene Exposure Dose Simulation Study, An Evaluation of Airborne Asbestos Concentrations Associated With Asbestos-Containing Electrical Wire Stripping Operations, Project No. D963.** The Maxim Engineers report was prepared under the supervision of Kyle Dotson, a certified industrial hygienist, for Ericcson, Inc. The stated purpose of the study "was to evaluate the airborne concentration of asbestos fibers to which an electrician employee might be exposed while stripping asbestos containing insulation off of electrical wire." The test chamber that was constructed for this study and the experimental technique were described in detail in the body of the report. The Maxim Engineers report provided three important findings:

1. The observed 8 hour time weighted average (TWA) worker exposure, based on breathing zone sampling, was 0.012 fiber/cc by phase contrast microscopy (PCM NIOSH 7400 method).

2. The general workroom (test chamber 10 x 10 x 8 feet) airborne asbestos fiber concentration was 0.005 fiber/cc.

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3. The "area percent" of 4 different wires tested ranged from 25-40% chrysotile asbestos. There was no detection of crocidolite or amosite.

The Maxim Engineering study concluded :

> The sample results indicate that airborne concentrations of asbestos fibers in the electricians' breathing zone were well below workplace employee exposure standards established for asbestos by the Occupational Safety and Health Administration (OSHA) and the American Conference of Governmental Industrial Hygienists (ACGIH). Additionally, the sample results of the general workroom air indicate airborne concentrations of asbestos fibers were less than the current post-abatement final clearance "clean air" standard established for public and private schools by the Environmental Protection Agency (EPA).

2. Penn Environmental Health Company, 1991

On 03/04/91 the Penn Environmental Health company of Pittsburgh released a report titled **The Measurement of Asbestos Fibers Released From Electric Wires Coated With Asbestos-Containing Insulation.** This report was conducted for Mr. Theodore Goldberg of the legal firm Goldberg, Henderson & Goldberg of Pittsburgh. The stated purpose of the study was "to determine whether asbestos fibers are released upon working with the wires." Four samples of coated electric wire were studied. A somewhat different experimental approach was used, in comparison to the testing method of Maxim Engineers. For the Penn Environmental study, wire stripping was performed inside a test chamber 30" x 15" x 15". Samples of the wire were subjected to bending, twisting, cutting and stripping. The Penn Environmental Health report provided two important findings:

1. The airborne asbestos fiber concentration was (in two tests) 0.0026 fiber/cc and 0.0073 fiber/cc. An 8 hour TWA was not calculated.

2. Chrysotile was the only fiber type detected.

3. Vittorio K. Argento, Ph.D., Environmental Engineering Services, Inc., 1992

On 01-10-92 Vittorio K. Argento, Ph.D., P.E. (Environmental Engineering Services, Inc.) of Duncanville, Texas released a report titled Asbestos Fiber Release Study Navy Cable at the request of Ericsson Radio Systems, Inc. The stated purpose of the report "was to evaluate the airborne concentration of asbestos fibers that would exist in the breathing zone of an electrician employee due to cutting, stripping and manipulating asbestos insulated Navy cable." Dr. Argento presented a detailed description of "two inch diameter navy cable" and two different kinds of "one inch diameter navy cable." These wires contained 20% chrysotile asbestos in a waxy matrix. The study technique was very similar to the Maxim Engineers study described above and was conducted inside a 10 x 10 x 8 ft test chamber. This report by Dr. Argento revealed:

1. The observed worker exposure, based on breathing zone sampling, was 0.034 structures (transmission electron microscopy) /cc equal to or greater than 5 micron in length for the one inch wire.

2. The observed worker exposure, based on breathing zone sampling, was 0.01 structures (transmission electron microscopy) /cc equal to or greater than 5 micron in length for the two inch wire.

3. The percent (?weight) of the different wires tested was 20% chrysotile asbestos. There was no detection of crocidolite or amosite. The chrysotile asbestos was impregnated in a waxy resin.

The above described study prepared by Vittorio K. Argento, Ph.D concluded:

> The results of the airborne asbestos fiber counting, using the most current analytical techniques and sampling methodology, show that airborne concentrations of asbestos fibers in the electrician's breathing zone were well below the allowable worker exposure standards established for asbestos by the Occupational Safety and Health Administration (OSHA). The airborne fiber concentrations were even further below the maximum worker exposure levels recommended by the American Conference of Governmental Industrial Hygienists (ACGIH).

4. MVA, Inc., 1992

On 12/06/92 MVA, Inc. of Norcross, Georgia released a report titled **Report of Results: MVA0410, Studies to Determine Asbestos Fiber Release During Cutting and Stripping of Wire Cables** at the request of Richard F. Scruggs, P.A. of Pascagoula, Mississippi. The stated purpose of the study was to "determine under controlled conditions, the levels of asbestos, if any, which might be released into the air when asbestos-containing wire cable is cut and stripped and what might be re-suspended into the air when sweeping the dust released during the cuttings." Testing methods similar to the techniques described in the above studies were utilized within test chambers 4 x 8 x 5 ft. Four different wire cables produced by General Cable, Phelps Dodge, U.S. Steel Corporation and General Cable Corporation were studied. Again, similar to the methods of the previously described studies, the test electricians were fitted with double personal air sampling devices. Area air samples were also taken after "aggressive disturbance" by area sweeping.

The MVA report did not give time weighted averages. A range of PCM and TEM results was given but the report noted that "The limits of detection vary with the volume of air collected. The detection limits for the cutting and stripping experiments were less than 0.1 f/cc." The MVA report found that:

1. "PCM values of airborne fibers greater than 5 µm were not significantly over the detection limits during cutting and stripping of wire cables."

2. The levels detected (by PCM) during sweeping were limited by small collected volumes. In an experiments in which 25 liters were collected the observed level was <.1 f/cc and in an experiment in which 30 liters were collected the level was <.08 f/cc.

3. Chrysotile was the only fiber type detected.

The MVA study concluded:

> PCM values of airborne fibers greater than 5 µm were not significantly over the detection limits during cutting and stripping of wire cables. Similar

results were found for asbestos fibers >5 µm in the TEM analyses, however, concentrations of asbestos fibers of all sizes in the air were increased over background air levels during the cutting and stripping of the wire cables containing asbestos.

5. M. Douglas Mueller, BCM, 1993

On 02/05/93 a report was submitted by M. Douglas Mueller, a certified industrial hygienist and EPA certified building inspector to Gallagher & Kennedy, P.A. of Phoenix, Arizona. The report was titled **Test Chamber Studies on Shipboard Electrical Cable Handling Operations, BCM Project No. 05-6684-0101.** The stated purpose of the study was "to ascertain if airborne fibers or asbestos structures were being generated during normal marine electrician tasks that would be adverse to the health of the marine electricians performing those activities." Five different shipboard electrical cables were studied. The cables had been in service on U.S. naval ships for many years. It was possible to determine that these cables had been manufactured by Anaconda Wire and Cable Company, Phelps Dodge Copper Products Corporation, Collyer Insulated Wire Company, General Cable Corporation and General Electric Company.

The studies were conducted inside test chambers 10 x 10 x 10 ft in dimension. A marine electrician performed the study tasks including cutting, stripping, manipulating and cleaning. As in the previously described studies, breathing zone air samples were obtained during wire manipulation and stripping work performed by the test electrician. In addition, work area samples were also obtained. The results are given in table 3 of this study. The results were not averaged and no attempt was made to give a TWA. Results were stated for both PCM and TEM. The study revealed:

1. The observed worker exposure, based on breathing zone sampling, ranged from <0.01 f/cc or structures (transmission electron microscopy) /cc to 0.045 f/cc or structures (transmission electron microscopy) /cc with most of the values in the undetectable or very low range.

2. The observed work area exposure was very low, ranging from <0.003 to 0.02 with most of the values in the undetectable or extremely low range.

3. By TEM, all identified structures were chrysotile.

The Mueller study concluded:

> At no time did the airborne fiber or airborne asbestos structure levels approach or exceed the prescribed OSHA Action Level (AL) of 0.1 f/cc or the Permissible Exposure Level (PEL) of 0.2 f/cc. Thus, it can be concluded that, based on the studies conducted to date and assuming the cables tested to be representative of shipboard electrical cables that there would be no exceedances of the OSHA AL or PEL during normal marine electrician tasks associated with shipboard electrical cable.

6. Clayton Environmental Consultants, Inc., 1994

On 01/31/94 a report was submitted by Clayton Environmental Consultants, Inc. of Edison, New Jersey to the legal firm of Lowenstein, Sandler, Kohl, Fisher & Boylan. The report was titled Industrial Hygiene Assessment Limited to the Evaluation of Asbestos Fibers Released During Wire Stripping of Rockbestos Cable. The Clayton report duplicated "the workplace exposure of the licensed electrician to airborne fibers, including asbestos" by stripping and cutting 3 different kinds of wire over 2-hour periods within test chambers. Clayton analyzed the cables for their content and collected personal air samples from the electrician cutting the cable. Personal and area sample analysis was performed using PCM and TEM. Bulk wire analysis was performed using polarized light microscopy (PLM).

Consistent with the above described earlier studies, the Clayton study found:

1. Personal and area air samples analyzed by PCM were at or below 0.01 f/cc.

2. Personal and area air samples analyzed by TEM were at or below 0.068 structures/cc.

3. The insulation (not including embedded wire) of two of the kinds of wire

contained 20% chrysotile asbestos and the third wire contained 50% chrysotile asbestos. There was no amosite or crocidolite detected.

The Clayton report concluded

> The results of the testing show that the electrician's exposure to airborne asbestos fiber concentrations was well below the current OSHA standards."

Summary of engineering reports

Six separate studies of asbestos containing electrical wire were conducted by industrial hygiene and engineering consultants between 1990 and 1994. The methodology of each of the six studies was presented in detail and was very similar. Within specially constructed test chambers, experienced electricians performed tasks identical to the kind of work done in the past in the construction of ships and buildings. Wire stripping and similar activities were conducted continuously during sampling periods, producing a "worst case scenario" measurement, rather than an 8 hour TWA. Phase contrast microscopy and transmission electron microscopy were used to analyze personal air zone, work area and bulk wire samples. Although conducted by different consultants, with analysis performed in different laboratories in different cities and using wire produced by different manufacturers, the results of these studies were remarkably consistent: activities such as stripping, bending, breaking and cutting asbestos insulated electrical wire produces minimal airborne asbestos dust with worst case (not TWA) exposures an order of magnitude less than the 1986 OSHA[1] peak exposure limit (PEL) of 0.2 f/cc. The results were also lower than the more recent 1994 OSHA[2] PEL of 0.1 f/cc. All of the asbestos insulated wire was found to contain chrysotile with no detection of any amphibole fibers.

In summary, working with asbestos insulated electrical wire results in very low dose chrysotile exposure. In the rest of this report I consider the implications of such exposure.

Low dose asbestos exposure and the risk of asbestosis

In general, the risk of pneumoconiosis increases with increasing intensity and duration of exposure to dust. Total or cumulative exposure is the product of exposure multiplied by

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duration of exposure and is typically represented in "fiber/ml-years". For example, a man who has worked in an environment where the average dust count was 2 fibers/ml for 10 years has a total exposure = 2 x 10 or 20 fiber/ml-years.

Most individuals with some asbestos exposure do not develop asbestosis.[3] Residents of chrysotile asbestos mining towns had an increased lung burden of asbestos fibers but no increased incidence of asbestosis.[4]

In the past, asbestos insulators and other workers with hands-on asbestos exposure were heavily exposed, often to levels in excess of 10 f/ml. Asbestosis was common in such workers; nevertheless, only a few studies attempted to establish a dose-relationship in asbestos workers because of the difficulty encountered in retrospectively establishing reliable airborne occupational asbestos levels.

In 1975 Weill et al[5] reported on a large scale study of 859 workers employed in two New Orleans asbestos cement factories. Weill suggested that diffuse asbestosis would not likely occur in workers who had a cumulative work exposure of less than 200 f/ml-years.

In 1979 Berry et al[6] reported on a study of 197 asbestos workers employed after 1950 for an average of 16 years. Berry estimated that the average annual exposure was 5 f/ml-yr and the cumulative exposure was 84 f/ml-yrs. The overall prevalence of "possible asbestosis" (as determined by two physicians) was 6.6% but the prevalence of the disease fell to zero below a cumulative lifetime work exposure of 10 f/ml-yrs.

In 1982 Finkelstein[7] reported on his study of 201 workers at an amosite asbestos-cement factory who were first exposed to asbestos dust prior to 1960 and who had been employed for at least 15 years. Using "limited" air sampling data, Finkelstein calculated an 18 year fiber exposure and the cumulative probability of being certified by the Ontario Workers' Compensation Board as having asbestosis. Finkelstein reported that among heavily exposed workers with an estimated cumulative occupational exposure of 200-249 f/ml-years the probability of asbestosis was 90%, in contrast to workers with a 50 f/ml-yr history for whom the probability was 10% and workers with a low exposure of 10 f/ml-yrs who had an asbestosis probability of 1% of less.

In 1983 the British Occupational Hygiene Society[8] (BOHS) updated Berry's 1979 report

with a study on 295 men who were employed after 1950 and who had a total of at least 10 years of asbestos exposure by 1976. The BOHS committee established 7 abnormal radiological and clinical criteria for the diagnosis of asbestosis and reported that the probability of at least one of these criteria occurring was 7% in workers who had a cumulative asbestos exposure of 50 f/ml-yrs and was less than 2% in workers who had a lifetime exposure of only 25 f/ml-yrs.

In 1984 the Ontario Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario[9] (Canada) summarized the evidence pertaining to a dose-response relationship for the development of asbestosis by stating:

> . . . our assessment of the evidence finds us in agreement with Dr. Finkelstein's comment [in testimony to the Royal Commission on 07/28/81] that there is probably an effective threshold below which clinical asbestosis may be unlikely to occur, although there may be occasional cases developing in very susceptible individuals. At low levels of occupational exposure to asbestos the fibrotic process in the lungs, if indeed it can be initiated, will not likely progress to the point of clinical manifestation or even the mildest discomfort. On the basis of the available data, our best judgement as to the lifetime occupational exposure to asbestos at which the fibrotic process cannot advance is in the range of 25 f/cc-yrs and below.

In 1985 Doll and Peto[10] accepted the same cumulative dose threshold in an important report prepared for the British government. Further support of the often-quoted Ontario Royal Commission report was published in 1990 by Huang[11] who studied the dose-response relationship for asbestos exposure in a chrysotile product factory for which exposure data had been available for many years. In that report, the threshold for 1% prevalence of asbestosis at the factory was 22 fiber/ml years.

In 1991 Browne[12] theorized that fibrogenesis only predominates when the fiber burden is "excessive." A threshold effect was therefore "implied."

> The effects of macrophage mediators on the surrounding tissues depend on the quantity released. Macrophages are modulating agents capable of

> producing fibrinolysis and fibrogenesis, and fibrogenesis will only predominate when the fiber burden is excessive. A threshold effect is therefore implied in this fibrinolysis-fibrogenesis balance...

Quinlan[13] also demonstrated dose-related cellular responses. Fibrogenic reactions were observed only for high concentrations of asbestos:

> Differential cell counts revealed a dose-related infiltration of neutrophils that preceded elevations in gene expression. Neutrophil infiltration into lung and focal lesions of fibrosis as well as increased levels of hydroxyproline were observed only at high concentrations of asbestos. These results indicate that high airborne concentrations of asbestos cause molecular changes in lung that may be related to antioxidant defense and the triggering of cell proliferation, a feature of asbestosis and lung cancer.

Asbestosis as a biological threshold for an increased risk of lung cancer

In 1947 the British Annual Report of the Chief Inspector of Factories noted that "Thirty one cases of carcinoma of lungs and pleura were found at autopsy of 235 cases of Asbestosis.[14] Commenting on this report, a 1949 editorial in the Journal of the American Medical Association[15] observed that ". . . the available evidence shows that the occurrence of cancer of the lung is related to pulmonary asbestosis and is not merely a possible sequela of exposure to asbestos dust . . . ."

In 1955 Doll[16] reported that of 113 men who worked for at least 20 years with asbestos, 11 developed lung cancer compared to an expected incidence of 0.8. Doll observed:

> All the cases of lung cancer were confirmed histologically and all were associated with the presence of asbestosis.

The landmark 1979 study by Hammond, Selikoff and Seidman[17] attempted to define the relative risks of lung cancer in a group of 17,800 asbestos insulators. Hammond et al

quoted a lung cancer incidence of 11.3/100,000/year for non-smokers and 122.6/100,000/year for smokers who had not worked with asbestos. Of the 17,800 insulators studied, 891 claimed to have been nonsmokers and 4 died of lung cancer, resulting in a corrected incidence of 58.4/100,000/year. The rest of the group studied by Hammond et al were considered cigarette smokers and they were found to have an incidence of lung cancer of 601.6/100,000/year.

Unfortunately, the 1979 Hammond report did not identify the sub-cohort that had asbestosis. Subsequent analysis of lung cancer cases derived from the original 17,800 cases published by Kipen et al (including Selikoff)[18] revealed that all of the patients who died of lung cancer also had pathological evidence of asbestosis.

In 1980 Liddell and McDonald[19] reported a study of Quebec chrysotile asbestos miners. Liddell and McDonald identified 118 lung cancer deaths which represented an excess of 52 deaths over predicted. Of 49 men whose last chest x-ray was rated as normal by the ILO system, the SMR for lung cancer was not elevated at 1.08; however, of the remaining miners whose x-rays were abnormal (and who presumably had asbestosis) the SMR for lung cancer was 3.5. Liddell and McDonald commented:

> . . . most, but not necessarily all, cases of lung cancer attributable to chrysotile exposure in mining and milling probably have small parenchymal opacities before death.

In 1981 Berry[20] reported on a study of asbestos workers who were certified by British Pneumoconiosis Medical Panels as having asbestosis. Berry observed that the standardized mortality ratio (SMR) for lung cancer increased from 9.1 for men classified as 10% disabled to 25 for men with a 50% disability. These findings suggest an increasing risk of lung cancer with increasing degrees of asbestosis.

In 1989 Sluis-Cremer and Bezuidenhout[21] reported an autopsy study of 399 former amphibole asbestos miners in South Africa. Thirty-five cases of lung cancer were identified on autopsy. Of the 399 patients, 302 were found to have no pathological evidence of asbestosis, 69 had "slight" asbestosis and 28 had moderate to severe asbestosis. In the group of 302 without asbestosis, the authors calculated that 12.4 lung cancers were expected (based on smoking habits) but only 11 were observed for an SMR of 887. Of the 69 patients with slight asbestosis, 3.6 lung cancers were expected but 15

were observed resulting in an SMR of 4.16. Among the 28 patients who were demonstrated to have moderate or severe asbestosis on autopsy, 1.6 lung cancers were expected but 9 were observed for an SMR of 5.63. The authors concluded:

> The SPMR for both the 35 cases proved by necropsy and the 25 cases certified as bronchial cancer on the death certificate show no excess bronchial cancer in the group without asbestosis whereas there is an excess in those with asbestosis. The excess increases with the severity of the asbestosis found at necropsy.
>
> In conclusion this study suggests that asbestos caused bronchial cancer is almost always associated with some degree of histologically demonstrable asbestosis.

Sluis-Cremer and Bezuidenhout also observed that:

> In the absence of asbestosis at necropsy a bronchial cancer in a man exposed to asbestos is unlikely to be due to asbestos.

In 1991 Hughes and Weill[23] described a study of all 839 workers employed in two New Orleans asbestos cement factories in 1969. By 1983 154 of the 839 men had died and death certificates of 153 of these men were available. X-rays on all of the men were interpreted by three prominent radiologists using the ILO system. The authors observed:

> Among workers with no x-ray film abnormalities in 1969, the lung cancer risk was not raised (five mesotheliomas, however, were found). For the long term workers in this group, there were six lung cancers compared with 5.8 expected. By contrast, among those with small opacities, profusion ≥ 1/0, there were around seven excess malignancies, all of them due to lung cancer; the lung cancer risk was significantly raised (9 v 2.1; $p<0.001$) and significantly different from the risk in long term workers without abnormalities ($p<0.01$, likelihood ratio test).

Hughes and Weill commented that:

> The current study is the latest in an emerging body of evidence supporting the view that asbestos is a lung carcinogen because of its ability to cause lung fibrosis. . . .
>
> Because detectable asbestosis is not likely to result from current occupational and general environmental exposures, the prevention of the effect of exposure on lung fibrosis is likely also to prevent the excess risk of lung cancer. . . . Finally, these data may provide further evidence to support the common practice of attributing lung cancer to exposure to asbestos only *if* asbestosis is also present; otherwise, these tumors are, in most part, due to cigarette smoking.

Almost all cases of lung cancer, including those which occur in asbestos workers, are related to cigarette smoking. Of the 35 lung cancer cases described in 1989 by Sluis-Cremer and Bezuidenhout, 33 were current or ex-smokers. Of the 29 cases of lung cancer described in 1991 by Hughes and Weil, all were smokers. A 1991 study published by the Edinburgh Lung Cancer Group[23] reviewed 3,070 patients who developed lung cancer from 1981-1985 drawn from a population center of 950,000. Of the 3,070 lung cancer patients, only 74 (2%) were lifelong non-smokers. Stopping smoking at any age significantly reduces the subsequent risk of lung cancer, but does not reduce that risk to the level experienced by never-smokers.[24] The persisting risk is most likely significantly greater for former heavy smokers than for former moderate or light smokers.

To summarize the above: Asbestos workers who smoke cigarettes and have asbestosis are at significantly greater risk of developing lung cancer than other smokers who do not have asbestosis. In contrast, asbestos workers who do not have asbestosis experience the same risk of lung cancer as non-asbestos workers who smoke (or smoked) the same amount. In a 1991 editorial in the British Journal of Industrial Medicine, Kevin Browne[25] presents the possible explanations for the difference in lung cancer risk experienced by workers who have or do not have asbestosis. Browne reviewed the Cairns hypothesis of mutagenesis, originally presented in the journal Nature in 1975[26] and the evidence that asbestos,

> . . . is not a mutagen, and attempts to establish a mechanism whereby it can directly produce malignant transformation in bronchial epithelium or mesothelium have either failed completely or have required unrealistic in vitro conditions.

Browne observed that:

> A consensus exists that asbestosis (diffuse interstitial pulmonary fibrosis, DIPF) results from the effect of mediators released by lung macrophages after incomplete phagocytosis. . . . The effects of macrophage mediators on the surrounding tissues depends on the quantity released. Macrophages are modulating agents capable of producing fibrinolysis and fibrogenesis, and fibrogenesis will only predominate when the fiber burden is excessive. . . .
>
> Powerful evidence that no additional carcinogenic action specific to asbestos is needed comes from studies of DIPF from other causes. In cryptogenic fibrosing alveolitis the histological picture may be indistinguishable from asbestosis apart from the absence of excess asbestos bodies or fibers, and patients suffering from this disease, or from DIPF secondary to other systemic diseases, show a greatly increased incidence of lung cancer. . .
>
> . . . . If the carcinogens in cigarette smoke have caused DNA damage to large numbers of lung epithelial cells, chronic proliferation of some of these cells will more readily result in the expression of malignancy than from both mechanisms independently. Asbestos is then acting as a classical promoter on initiated cells. . . .

In summary, asbestos workers who smoke and who develop asbestosis are at increased risk for the development of lung cancer because: (a) cigarette smoke is a primary carcinogen or "inducer" which stimulates mutations (i.e. malignant transformation of DNA) in bronchial epithelial cells; (b) chronic proliferation of pulmonary cells associated with pulmonary fibrosis increases the likelihood that mutated cells will produce a malignant tumor; and (c) pulmonary fibrosis disrupts the lung's natural protective mechanisms which normally limit the proliferation of mutated cells.

Pulmonary parenchymal asbestos fiber burden and risk of lung cancer

Greenberg and Roggli[27] have expressed agreement with the above described relationship between asbestosis and lung cancer but suggested that the simple presence of an elevated tissue asbestos burden (without associated asbestosis) might also be enough to increase the risk of lung cancer:

> . . . the weight of the evidence at this time seems to indicate that, in an asbestos worker with carcinoma of the lung who also smokes cigarettes, asbestosis must be present clinically or histologically (*or there should at least be a tissue asbestos content within the range of values observed in patients with asbestosis*) [emphasis added] in order to assign a substantial contributing role to asbestos in the causation of the lung cancer.

Roggli, Pratt and Brody[28] reviewed 5 reports of tissue asbestos content in asbestosis. In all 5 reports the median uncoated fiber count exceeded (usually by a considerable amount) 1×10^6 fibers per gram of dry lung tissue. Roggli et al suggested that 1×10^6 fibers is a threshold tissue burden for the development of asbestosis. Roggli further reported that the mean uncoated fiber count in 76 patients who had pathological evidence of asbestosis was 3.3×10^6 fibers per gram of dry lung tissue. Roggli et al also reported on a study of 143 patients who had been exposed to asbestos and who developed lung cancer. All but 6 were cigarette smokers or ex-smokers. Among the 48 patients who had pulmonary parenchymal asbestosis the mean asbestos fiber count was 3.07×10^6 fibers per gram of dry lung tissue.

Roggli[29] reported that of 76 patients who had asbestosis, the mean asbestos body count was 378,000 per gram dry lung. Roggli, Greenberg and Pratt[30] reported that of 48 patients who had asbestosis and lung cancer, the mean asbestos body count was 334,000 bodies per gram dry lung.

Asbestos fiber type and mesothelioma

Wagner was the first to report in 1960[31] of the association between asbestos exposure and the development of mesothelioma. Wagner suggested that it was only "Cape Western

Blue" or crocidolite asbestos that was the cause of mesothelioma. In 1965 Selikoff[32] noted:

> This remarkable concentration of cases in one area of South Africa was explained by the hypothesis that mesothelioma was the result of exposure to one special type of asbestos (crocidolite) . . . .

Despite this finding, Selikoff suggested that ". . . mesothelioma may not necessarily be a problem of only one kind of asbestos (crocidolite) . . . ." but in 1965 Sluis-Cremer[33] reported on a methodical search for mesothelioma cases outside of the South African North West Cape Province crocidolite mining area and concluded that there were no cases from the amosite mines of the Transvaal Province. It was not until 1972 that Selikoff, Hammond and Churg[34] demonstrated that amosite was a cause of mesothelioma. In 1980 Selikoff, Seidman and Hammond[35] reported on a further study of amosite factory workers. Selikoff et al noted that:

> In 1941, with the encouragement of the U.S. Navy, a factory was established in Paterson, New Jersey, to manufacture asbestos products for the armed forces for industrial uses. We have ascertained that amosite asbestos was used almost exclusively (with very small amounts of chrysotile asbestos also being used).

Amosite was specified by the U.S. and other navies for the construction of war ships. Stivers and Coles (HM Naval Base, Devonport, England)[36] noted "Two major developments since 1944:

> (1) the very extensive use of crocidolite for environmental insulation and fire protection from 1944 to 1963, and (2) the large increase in the amount of amosite used for machinery insulation between 1950 and 1963.

An earlier report by Harries[37] described the same kind of amosite use in British shipyards.

In 1982 McDonald[38] reported on a study of asbestos textile workers exposed to chrysotile, amosite and crocidolite. The authors noted that:

> The much greater risk of mesothelioma from exposure to processes in which even quite small quantities of amphiboles were used was also confirmed.

In 1984 McDonald[39] reported that long term follow up of a large cohort of men who were exposed only to chrysotile did not reveal any cases of mesothelioma "consistent with a much lower risk of this tumor with chrysotile than with amphiboles."

In 1989 Newhouse and Sullivan[40] described a long term follow up of men who had worked in a factory that produced friction materials (principally brakes). The factory used chrysotile asbestos except for certain specific contracts. All of the mesothelioma cases were attributed to crocidolite exposure.

In 1988 Warnock[41] reported on the lung tissue fiber burdens of workers in a shipyard in the Solano County area of California who developed mesothelioma. The median fiber burden for all types of amphiboles was 2.7 million f/g dry lung. Warnock noted that amosite was the most prevalent fiber type.

In 1992 an important study was published by Begin[42] who identified a total of 49 cases of mesothelioma in former Quebec chrysotile asbestos mine and mill workers. Begin noted:

> The present study documents an increasing incidence of cases of malignant mesothelioma in chrysotile miners and millers of the Eastern Townships of Quebec, with 49 cases in the last 23 years, and a rate of 2.5 cases per year in the last 10 years in the primary industry. . . .

In their report, Begin et al also noted that:

> In the past, these cancer cases among Quebec chrysotile miners and millers were considered to be likely attributable to amphibole contamination of the worksite and/or of the mineral ore. . . Nonetheless, cases of mesothelioma in Quebec miners have been documented where chrysotile fibers were the only fibers in lung tissue.

Begin noted evidence for a significant difference in the tremolite contamination of chrysotile ore mined in the two main Quebec asbestos centers, the towns of Asbestos and

Thetford Mines, but the incidence of mesothelioma was proportional to the work force and not related to residence in one of the two areas.

> Our data suggest that some of the cases of malignant mesothelioma in Quebec chrysotile miners and millers may not be necessarily attributable to amphibole and could be chrysotile-induced.

In 1993 Churg et al[9] reported on the fiber burdens in the lungs of 94 long term chrysotile asbestos miners from Thetford Mines, Quebec. In the patients who had mesothelioma, the chrysotile fiber burdens were very large, with a mean of 34 million f/g dry lung. The tremolite burdens in the mesothelioma cases were even greater, 180 million f/g dry lung. Churg et al concluded:

> . . . In this population of heavily exposed chrysotile miners and millers, the presence of airways fibrosis and asbestosis and, probably, mesothelioma reflects high tremolite burden. Whether chrysotile fibers themselves play a role in disease induction remains uncertain.

In a subsequent report, Churg and Vedal[44] reported on a study of 144 shipyard workers and insulators from Oregon Washington state and British Columbia. Churg and Vedal stated:

> Our results show clearly that, despite known historic exposure to amosite and chrysotile, amosite is by far the predominant residual fiber, and there are correlations between amosite measures and disease. Chrysotile was present inconstantly and in relatively small amounts, and no correlations were found between chrysotile measures and disease.

Churg and Vedal also commented on the issue of tremolite contamination of chrysotile:

> We have proposed elsewhere that tremolite, which is a natural minor constituent of chrysotile ore, might serve as a substitute marked for chrysotile, and that tremolite rather than chrysotile may actually be the agent responsible for "chrysotile-induced" mesothelioma . . . .

Becklake[46] provided an in-depth editorial comment of Churg and Vedal's study. Professor Becklake noted that:

> . . . the major contribution of the two studies of Churg and associates reviewed here is in adding further weight to the already substantial body of evidence concerning differential disease-generating potential for different asbestos fiber types. Although the facts of differential clearance of chrysotile are acknowledged in both papers, we believe the authors are right to conclude that "there are major differences in the relationship of fiber concentration and disease for chrysotile and tremolite compared with amosite or crocidolite."

Although the medical literature published since 1980 reflects significant controversy, (not presented here, but see Roggli[48] and the multiple responses in the literature such as Hughes and Weill[47]) at this time there is strong scientific support for the following conclusions about mesothelioma:

1. Without an amphibole contaminant, chrysotile is unlikely to be a cause of malignant mesothelioma.

2. In terms of mesothelioma inducing potency, the different forms of asbestos can be rated as: crocidolite ◇ amosite ◇ tremolite ◇ ?chrysotile

3. In terms of frequency, amosite represents the cause of most mesothelioma cases in the United States. Amosite exposure was typical of World War II era shipyard work.

4. Where chrysotile has been associated with mesothelioma, the majority of cases have probably been caused by tremolite contamination of chrysotile ore.

5. Since tremolite is a minor contaminant of chrysotile ore, very large chrysotile exposures are necessary before mesothelioma can occur. As noted above, in 1993 Churg et al reported that the mean chrysotile fiber burdens in asbestos miners who developed mesothelioma were enormous - 34 million f/g dry lung and that the tremolite burdens were even greater.

Conclusions from the medical literature

The medical literature presented above provides strong, consistent, rational evidence for the following conclusions:

1. There is a threshold of exposure below which diffuse interstitial pulmonary asbestosis does not occur. That threshold is in the range of 25-50 f/ml-years. Compliance with the peak exposure limit (PEL) of 0.2 f/cc 8 hour time weighted average set by the United States Occupational Safety and Health Administration in 1986 makes it impossible to accumulate a lifetime occupational exposure >25 f/ml-yrs even after 40 years of employment.

2. The risk of bronchogenic carcinoma is increased in asbestos workers who smoke cigarettes and have pulmonary parenchymal asbestosis. The risk of lung cancer in asbestos workers who do not have asbestosis is related to amount of cigarette smoking and is unrelated to asbestos exposure. Thus, the development of pulmonary parenchymal asbestosis is a necessary, biological threshold below which an increase in the risk of lung cancer is not observed.

3. Evidence for a lifetime threshold of asbestos exposure below which there is no increased risk of mesothelioma was not presented in this report. The risk of mesothelioma is much more closely related to elapsed time since first exposure than to dose[48] but the literature does suggest a threshold of exposure below which the risk of mesothelioma is extremely low or non-existent.[49 50 51] Asbestos fiber type is of greater significance than threshold fiber burden. The weight of the medical evidence strongly indicates that chrysotile exposure alone does not increase the risk of mesothelioma.

The "one fiber" theory vs. biological plausibility

Some medical experts have suggested that <u>any</u> exposure to asbestos fibers contributes to the development of asbestos-related diseases. Such experts argue that for an asbestos insulator who has accumulated, for example, a moderate fibrogenic asbestos burden of

100 f/ml-yrs, exposure to even one asbestos fiber contributed in a meaningful way to his eventual development of asbestosis and, possibly, lung cancer. According to this view, exposure to the minute quantities of asbestos released during the manipulation of asbestos insulated electrical wiring would be significant. However, the medical literature presented above reveals that such "one fiber" theories are scientifically incorrect. A considerable asbestos fiber burden must be accumulated before there is any risk of asbestosis.

The average amount of asbestos in the lungs of patients who have asbestosis is in excess of 3,000,000 fibers per gram of dry lung. Thus, patients who have asbestosis have accumulated hundreds of millions of asbestos fibers during their occupational lifetime. Exposure to chrysotile asbestos within the limits (PELs) established by OSHA in 1994 (or 1986) cannot result in a sufficient pulmonary asbestos tissue burden to result in asbestosis. Work with asbestos insulated electrical cable can, in "worst case" scenarios, result in asbestos exposure 10 to 20 times lower than the OSHA peak exposure limits.

In conclusion, it is my opinion to a reasonable degree of medical certainty, that work with asbestos insulated electrical cable cannot result in dangerous asbestos fiber exposure. Electricians working with such cable cannot, in a lifetime, accumulate a significant pulmonary asbestos burden. If an individual worker who has other occupational asbestos exposure develops asbestosis, the exposure to asbestos fibers from insulated cable does not represent a biologically significant contribution to his disease. Because asbestosis cannot result from the level of asbestos exposure associated with asbestos-insulated cable, such exposure cannot result in lung cancer. Exposure to chrysotile asbestos from asbestos-insulated cable cannot result in mesothelioma.

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