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95

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Mr. Jamie B. Mc Stecher
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Dear Mr. Stecher:

Per our discussions, I have reviewed as much of the asbestos-related medical literature as I could find in an attempt to find answers and documentation thereof to the questions you posed early in September.

I. Is there a safe level of asbestos exposure in relation to asbestos-related disease?

Genercily speaking, it is my opinion that a threshold does exist for all substances, even the most potent of carcinogens. Most testing of asbestos-related cancer effects looks at the mechanism or at crude epidemiologic indicators and does document effects at high dosage levels. I could find no studies of asbestos-related health effects testing for no effect dose levels in humans. Even very strong carcinogens such as methylcholanthrene, a polycyclic aromatic hydrocarbon, have been shown in animal studies to have levels below which no carcinogenic effect occurs. [Hayes '75] This and several other substances tested in animals show S-shaped dose response curves which is also consistent with the known toxicology of chemicals which have toxic effects other than carcinogenesis [e.g. lead, cyanide, etc.]

One reason for the lack of studies testing the no effect level for low incidence cancers such as asbestos related pulmonary cancer or mesothelioma is as follows. If a cancer occurs at the rate of 1 per 10,000 or 1 per one million as a result of an asbestos exposure, in order to test

047

Mr. Jamie B.W. Stecher
Page two

October 13, 1980

for the level below which no cancer would occur. [with 99% certainty], extremely large groups of animals would be required at various dose levels. That is, if a cancer has an incidence of 1 in 10,000 due to asbestos exposure, it would require $500 \times 10,000$ or 5 million animals in each dosage group in order to determine the no effect level with 99% certainty. [Hayes '75]

Because such testing is impractical and prohibitively expensive, many toxicologists have simply assumed that the dose response-threshold relationship is linear rather than S-shaped. In order to predict low level dose response effects, such toxicologists draw a straight line based on findings at very high dose levels administered to relatively small groups of animals [5 to 200 average] such that a very high response rate will occur. In extrapolating linear relationships to low dose levels, such toxicologists assume that identical circumstances and identical biologic mechanisms are operative at extremely low doses as are operable at high dose levels. [Hodge '79] In other words, it is assumed that the organism's ability to defend itself against pathological effects of a foreign substance do not change regardless of dose level; simply fewer cells and/or individuals develop pathological effects. Contrarily, we do know that toxic substances for which we do have low dose-response data do have an S-shaped dose response curve with a definite safe level. [see Figure 1]. For such substances the body's normal metabolic and other defense mechanisms are capable of handling small quantities of toxins. Pathologic effects occur only when the body's defense mechanisms are overwhelmed quantitatively. Such common toxins as carbon monoxide, cyanide and lead have demonstrated this principal repeatedly. Cyanide, for example, begins to exert pathological effects only when the quantity absorbed into a human system is greater than that quantity which the cyanide metabolic pathway is able to handle; an even more common example is our experience as individuals with alcoholic beverages. Although the quantity varies somewhat, all healthy human beings can tolerate low to moderate levels of alcohol intake without feeling or developing any toxicologic effect. The same is true of silica [sand].

042

CRMC-M&S-002977

Mr. Jamie B.W. Stecher
Page three

October 13, 1980

Complicating the issue of determination of a safe level for asbestos exposure are several problems. Most measurements of suspected effects of asbestos rely on very crude indicators, such as group mortality by cause. Also, diagnosis of mesothelioma is not uniform. Various studies of agreement between pathologists on mesothelioma diagnoses have shown as much as 30% disagreement according to tissue diagnosis. [McCaughey, '73] Also, in measuring morbidity and/or mortality related to asbestos exposure, there is no way at present to separate out effects due to other types of exposures. Investigations relating excess morbidity and/or mortality to asbestos exposure are relying on the assumption that there was no other common carcinogenic factor involved. Although animal studies can certainly be conducted under controlled circumstances to eliminate other carcinogenic agents, human epidemiologic studies have no such assurance, especially in the involved industries where other carcinogens such as arsenic, hexavalent chromium, nickel, beryllium, and cadmium are known to occur at relatively high levels. Similarly, many human studies do not even attempt to account for the obvious confounding factor of cigarette smoking, which is presently felt to account for far and away the greatest proportion of human cancer deaths.

Because we are dealing with a substance, the toxicologic effects of which may not be completely known, when a certain pathologic outcome is found to be in excess in a group exposed to asbestos, it has been generally entirely attributed to asbestos. Many such associations may eventually be shown to be indirect, that is associated with both asbestos and the pathologic outcome, but in fact caused by a third factor common to both.

Measurements of airborne levels of asbestos which occurred proximal to individuals who later developed pathologic effects attributed to asbestos exposure are poorly, if at all, documented. The airborne exposure levels which are recorded are consistently well above the levels which are now accepted as legally safe, which are in turn 1000 to 100 times higher than the levels associated with Garlock product usage. Investigation of relationships between airborne levels of asbestos fibers and pathological outcomes have often been separated

Mr. Jamie B.W. Stecher
Page four

October 13, 1980

into large increments usually above approximately 10 to 20 fibers per cc [McDonald '80, Enterline '73, Weill '79]. The few reports which do attempt to correlate exposure levels with pathologic outcomes range from levels of 5 to higher than 100 fibers per cubic centimeter before 1960. [Gibbs '73, McDonald '80, Fleisher '46, Nicholson '79]. After 1960 the few reported levels in the literature ranged between 2 and approximately 9 fibers per cc averaging approximately 3 to 6 fibers per cc. [Marr '64, Balzar '68, and Nicholson '79]. Additionally, fiber type was frequently not specified, mixed, or unknown. However, most of the asbestos related disease accounted for in the present literature would probably have been related to exposures occurring prior to 1960.

In support of a dose response, threshold relationship for asbestos in cancer incidence, the epidemiologic investigations show a clear trend of increasing incidence of pulmonary cancer in trades and groups with the highest expected and demonstrated levels of asbestos exposure [asbestos miners, millers, asbestos production workers, and insulators]. [Selikoff '73, Newhouse '73, Wagner '60, Rubino '72, Elmes '71, Enterline '73, McDonald '74, Cochrane '78 and Hasan '78].

II. Is there a safe or threshold level for asbestos exposure in relation to malignant mesothelioma [MM] occurrence?

I do believe there is a safe level of asbestos exposure in relation to mesothelioma induction, but there is no good data to assess numerically what that level is. There is significant data suggesting what the level is not. There is a large amount of data suggesting a strong association between mesothelioma and relatively high levels or levels above where I would expect that threshold to occur. Human epidemiologic studies indicate mesothelioma related lifelong asbestos exposures ranging upwards from 5 fibers per cubic centimeter to as high as 100 fibers per cc [Nicholson '79, McDonald '80, Berry '79, Selikoff '76]. Such studies also indicate a trend of increasing mesothelioma incidence associated with increasing duration as well as increasing intensity of exposure [Newhouse '79, IARC '79]. Because MM has such a long latency period (average 30 years) and because asbestos exposures which occurred 30 to 50 years ago were not generally or precisely recorded, there is essentially no precise data correlating airborne dust levels and MM incidence.

050

Mr. Jamie B.W. Stecher
Page five

October 13, 1980

The literature which is available on mesothelioma attempts to measure dose in terms of length of employment or years since first employment [Selikoff '79, Seidman '79]. Nevertheless, this data does suggest a threshold relationship, since for example Seidman's study showed no mesothelioma incidence in males with less than 6 months employ in an amosite asbestos factory. Many other studies combine all pulmonary cancers, including bronchogenic carcinoma and mesothelioma in one category, so it is impossible to interpret such data in terms of mesothelioma incidence alone. Less direct indicators of exposures such as duration of employment and type of work are available and indicate that occupational MM cases have occurred in persons with fiber exposures of high intensity occurring over long periods [McDonald '77]. Estimates of proportionate mortality also show greatest percent of deaths due to MM in certain occupational groups were those with the greatest asbestos exposure [McDonald '77].

The few long term studies of highly exposed groups such as insulators [Selikoff '79] asbestos factory workers [Newhouse '79] show similar findings. There have been no comparable studies of MM incidence in groups with exposures at very low levels consistent with those levels generated in the use of Garlock products. Further complicating the threshold issue is a suspected difference in the carcinogenicity of different fiber types. In decreasing order, carcinogenic potency [the lesser amount of asbestos fiber causing the same effect is considered the most potent] may be crocidolite, chrysotile, amosite and anthopholyte as shown in some human and animal studies [Wagner '77, Neuman '74, IARC '77]. Although the order of potency may vary according to different studies, it is generally accepted that carcinogenic potency does vary with fiber type. Unfortunately, many epidemiologic human studies of MM have either mixed or unknown fiber type exposures. Recent studies have shown differences in carcinogenic potential according to fiber length; the longer thinner fibers being more potent carcinogens [Davis '79, Stanton '73]. In support of this hypothesis, Stanton found no significant difference in carcinogenic potency if different fiber types administered to animals were all reduced to the same size. Such findings suggest that not fiber type of asbestos but rather fiber length is the critical factor in tumorigenesis.

051

CRMC-M&S-002980

Mr. Jamie B.W. Stechor
Page six

October 13, 1980

If so, the length of the fiber generated by the particular industrial process may be more critical than the actual duration, intensity or total dust exposure.

A strong association has been shown between high asbestos exposures and MM, mostly on the basis of occupational histories which show employment in or around a very dusty asbestos trade. Most mesothelioma autopsy series show 50-75% of the cases with a likelihood by history of high asbestos exposure long prior to their tumor development. Because this association repeatedly appears globally in various settings and because it has been duplicated in animal studies, it is likely that a causal relationship exists. However, the assumption that if asbestos causes cancer in a relatively large number of subjects exposed at a high fiber level, it must have the same effect at a very low fiber level in a smaller number of subjects, is not supported by known toxicologic principals, laboratory investigations, or human experience. Cases have been reported in which exposure to asbestos was by supposition or history, apparently lower than in highly exposed occupational groups. Such cases have occurred in children or spouses of asbestos workers or in occupations having casual asbestos exposure or in neighborhood contacts [Anderson '79, Viano '78, Mostert '79]. Yet the environmental sampling data which exists for home and asbestos factory or mine neighborhood exposures shows levels 10 to 1000 times the levels in ambient urban air [IARC 77]. Also the likelihood of a short but intense exposure during clothes washing at home is high considering that in those years dusty clothing was worn and washed at home. Most of these studies either indicate amosite, mixed or unknown fiber types.

Estimates of asbestos incidence during and prior to the present MM epidemic ranged from .07% of autopsies between 1910 and 1943 to .24% of autopsies between 1950 and 1970 in general populations [McDonald '77]. This shows a substantial increase in MM occurrence but does lend support to the observation that approximately 1/3 of the MM cases may have no relation to asbestos exposure since they would have occurred

052

CRMC-M&S-002981

Mr. Jamie B.W. Stecher
Page seven

October 13, 1980

during periods when asbestos was not used heavily. This observation has been borne out in most recent autopsy series where history of asbestos exposure ranges from 40-87% of MM cases [McDonald '77, Elmes '67, Viano '78, Hasan '77, Borow '73, Oles '71]. The International Agency for Research on Cancer has suggested that approximately 85% of MM cases are asbestos related.

What many MM investigations reported in the literature have done is to search for any remote indication of past asbestos exposure regardless of other types of possible tumorigenic exposures and, if any such asbestos exposures exist, ascribe the causation of that case to asbestos exposure. Similarly, some MM reports in which no obvious asbestos exposure has been found may be implied to be due to exposure to ambient asbestos without any other basis in fact. In other words, because MM occurs due to high exposure levels, it has been assumed with very weak supporting evidence that it similarly occurs due to low levels of airborne fiber exposure. No control studies of low doses in either humans or animals have been done.

Animal investigations of MM resulting from inhalation as well as intrapleural administration of asbestos fibers clearly support a dose response, threshold relationship with increasing rate of MM occurrence in association with increasing levels as well as duration of asbestos administrations. Wagner's rat studies [1974] of asbestos inhalation exposure did however have 2 cases of MM, at the lowest dose level, one each related to crocidolite and amosite exposure respectively. However, the dose administered over a 7 hour period for one time only was 12 mg per cubic meter. Although this dose is not directly convertible to human dose measurements in fibers per cc, I suspect it is roughly a thousand to a million times the dose found daily in heavy human asbestos exposures [based on a comparison with data from Nicholson and Sebastian, p 34 IARC]. It is not unlikely that even one such massive dose could overwhelm the carcinogen defense mechanisms of rats. Other animal studies involving oral inhalation and intrapleural administration of various asbestos types have involved doses far in excess of what would be expected to occur over the lifetime of a worker exposed exclusively to Garlock

053

CRMC-M&S-002982

Mr. Jamie B.W. Stucher
Page eight

October 13, 1980

products. Although it is strictly conjectural to compare doses in milligrams per cubic meter to fibers per cc, based on what little comparison data is available I suspect that the doses given in most of these animal experiments range from a thousand to a million or more times the expected dose one would encounter in a lifetime of exposure to Carlock products.

The existence of a threshold is further supported by Whitwell's studies of fiber content in lungs from MM patients. 99% of MM cases with a significant asbestos exposure history had greater than 20,000 asbestos fibers per 5 grams of dried lung tissue. 95% of the MM cases had greater than 50,000 fibers per 5 grams of dried lung tissue, which indicates high absorption levels and high occupational exposure levels. The women in this study who had MM, who had a history of short duration exposures, had greater than 250,000 fibers per 5 grams of dried lung tissue suggesting short but very intense exposures. Whitwell also found non-malignant mesothelioma control patients from urban areas with high expected ambient asbestos exposure levels who did not have high lung fiber counts. Whitwell interprets this data as firm support of a dose response, threshold relationship between asbestos and MM. Ashcroft's findings are similar and add support to the hypothesis that a threshold exists and it may be related not necessarily to length of exposure but to the total lifelong cumulative dose.

A possible explanation for MM occurrence in individuals with minimal asbestos exposure [assuming a threshold exists] is related to the animal and human studies showing MM to be due to exposure to other non-asbestos form materials [as well as asbestos] many of which have similar fiber characteristics [manganese dust, silica, polyurethane, diatomaceous earth, beryllium, fiberglass, and avian leukosis virus [Stanton '73, Borows '73, Wagner '62, Hueper '64, Chabot '70, and Webster '65]. Baris ['79] investigation of a MM epidemic in Turkish villagers having negligible asbestos exposure shows an association between MM and zeolite fibers which he suggests may be the cause of some other non-asbestos related MM. Obviously, many of the above mentioned substances are encountered in the industrial and urban environment and could be the cause of MM in

Mr. Jamie B.W. Stecher
Page nine

October 13, 1980

persons with minimal or negligible asbestos exposure history. I doubt if many of the investigators inquired as deeply into possible history of exposure to these agents as they did into asbestos exposure.

In summary, there is good evidence supporting existence of a threshold for MM due to asbestos exposure in addition to evidence which could explain apparent but non-causal relationships between MM and low asbestos exposures. More importantly, there is no data on MM incidence in human or animal populations exposed to the levels encountered by workers whose asbestos exposures would be limited to use of Garlock products.

III. What is the likelihood that MM occurred in workers whose only asbestos exposure would result from use of Garlock products?

Because no data exists for exposures of .006 to .05 fibers per cc, [the levels encountered during Garlock product usage] estimates of risk are purely speculative. Crump states that the effect on carcinogenesis will be proportional to the amount of the carcinogen added if the subject resides in an environment with multiple carcinogenic processes. At very low doses and in the presence of other possible carcinogens also commonly encountered in shipyard and other asbestos related industries [zeolite, manganese, fiberglass] the role of low levels of asbestos in MM occurrence could possibly be completely overshadowed by these other mesothelioma producing agents.

Peto ['79] reasons that if an exposure is reduced by a given proportion, the outcome [MM] will be reduced similarly. If we assume an average proportionate mortality [1% of all deaths] due to mesothelioma of 7% in insulation workers exposed to approximately 15 fibers per cc, on a time weighted average, an estimate of proportionate mortality at .03 fibers per cc [average Garlock product usage level] should according to Peto's model equal .03 divided by 15 x 7% which equal .014%. This is certainly less than the .03% proportionate mortality found in the United States prior to heavy asbestos use [McDonald '77].

Mr. Jamie B.W. Stecher
Page ten

October 13, 1980

These crude calculations show that even if a linear dose response relationship exists, it includes a MM asbestos exposure threshold above the levels encountered by individuals whose only asbestos exposure is use of Garlock products. It therefore suggests that exposures at this level place one at no greater risk for MM than exposures to pre 1940 United States urban air.

Consistent with this hypothesis is Schirripa's [1980] study of ambient asbestos levels in approximately 40 general use office and school buildings containing asbestos products. His findings showed that 62% of these buildings had a time-weighted average asbestos fiber level of .01 to .15 fibers per cc. Therefore, individuals working or who have attended school in such office or school buildings having asbestos products would be at the same risk of asbestos related disease as Garlock product users. In fact, approximately 25% of the cumulative life long asbestos exposure of persons whose only asbestos exposure would be through the use of Garlock products and attending school would have occurred during the 12 years of attending primary and secondary schools.

IV. Is there a safe level for lung cancer [exclusive of mesothelioma] and what is the likelihood of pulmonary cancer due to exposure at garlock product user levels?

Although I believe a threshold for asbestos related pulmonary carcinoma exists, as in the case of MM, data has not been produced to determine where that level lies. Existing data is based upon the asbestos exposure levels indicated in the previous section [malignant mesothelioma] which range from approximately 100 to 10,000 times that encountered with exposure to garlock products. Existing data does indicate a strong association, probably causal, between high level asbestos exposure and bronchogenic carcinoma. Becklake [76] summarizes these studies which indicate relative risks for lung cancer in association with asbestos exposure ranging from 1.5 to 17.6 with a mean of approximately 5.0 [relative risk is determined by dividing observed cases by expected and has decreasing significance as it approaches 1.0].

Mr. Jamie B.W. Stecher
Page Eleven

October 13, 1980

Complicating this issue is the finding that smoking cigarettes and asbestos exposure act together to increase one's risk of pulmonary cancer in either an additive [Enterline '79] or multiplicative fashion, such that the risk of pulmonary carcinoma in the presence of both smoking cigarettes and asbestos exposure may or may not exceed the sum of their individual risks [Sellikof '79, Wagner '80, Martischniig '77]. Smoking can affect the findings of studies which do not adjust their analyses for smoking habits of the cases, if the cases actually smoke more than the control or comparison group. Such was the case with Muerstein [73] study which found a higher rate of smoking in asbestos exposed workers than in non-asbestos exposed controls. Many studies have not considered this factor.

As with MM, few long term studies in either animals or humans have been performed which attempt to correlate exposure levels with lung cancer. Also in some of the human studies it appears that MM are included in the lung cancer statistics, the effect of which would be to artificially amplify the relationship between asbestos and lung cancer [McDonald 1979, Henderson 1979]. Nevertheless, McDonald's data supports a linear dose response relationship and threshold level for asbestos and pulmonary cancer at approximately 90 fiber years of cumulative exposure [fiber years are determined by multiplying the mean fiber concentration per cubic centimeter times the total number of years of exposure at that level]. Garlock product users would be estimated to have from .3 to 2.5 fiber years with an expected mean of 1.5 fiber years total cumulative asbestos dust exposure, if their asbestos exposure was due solely to the asbestos generated from the use of Garlock products and assuming a 50 year work life with an average of 240 work days per year. Similarly, Henderson and Enterline [79] suggest a good correlation between cumulative exposure and risk of pulmonary cancer mortality. However, the lowest exposure level which they had correlated between pulmonary carcinoma mortality and asbestos exposure was 62 million particles per cubic foot/years [approximately 120-180 fiber/years] which, like McDonald's data, is approximately 100 times that of expected cumulative Garlock product user exposure. In extrapolating to lower [as yet unstudied] exposure levels, Henderson and Enterline developed the following

057

Mr. Jamie B.W. Stecher
Page twelve

October 13, 1980

formula for standardized mortality ratios (1.0 is the "normal" baseline SMR); 100 plus .658 times the cumulative exposure in million particles per cubic foot. For Garlock product users this would be approximately 100.658, a negligible change. Similarly McDonald's formula for relative risk of pulmonary carcinoma due to asbestos exposure is 1.0 plus .0014 times the cumulative dust exposure. For Garlock product users this would be approximately 1.006 and indicates that out of every 10,000 cases of lung cancer occurring in Garlock product users, 6 would be due to asbestos exposure. Using 1978 statistics, this would result in a total of approximately 55 deaths or an annual rate of approximately 1 per 4 million persons exposed at those levels. Considering that far less than 4 million are exposed to Garlock products, this risk is extremely low. These formulas also are based on the assumption that no safe threshold exists. Therefore, even without a threshold the risk of lung cancer at Garlock levels appears very low. If we accept Peto's hypothetical model which indicates a 1.8% rate or proportionate mortality of pulmonary carcinoma with 50 years of exposure at approximately 2 fibers per cc, Garlock exposures of .03 fibers per cc would correspondently yield .00027% or 2.7 asbestos cancer related deaths per 10,000 deaths or approximately 1 to 2 per million exposed per year. As low as these estimates are, they probably over estimate the impact of asbestos alone because smoking was not taken into account in either study. Unless otherwise stated, it is impossible to know whether exposed persons smoke more, less or the same as unexposed persons.

Several studies have demonstrated [Schneiderman '79, Enterline '76, Newhouse and Berry '79] that pulmonary carcinoma is related to intensity and duration of dust exposure as well as length of time from first exposure [latency]. If in fact there is no safe level but instead very low level exposures result in extremely long latency periods which would well exceed the lifetime of the individual exposed, such a risk due to low exposures would then be virtually zero. Schirripa's analysis of exposure in school, office and general purpose buildings supports this concept of extremely low risk of carcinoma during present expected life times due to the very low [comparable to Garlock] exposures presently encountered in office, school and other general purpose buildings.

058

CRMC-M&S-002987

Mr. Jamie B.W. Stecher
Page thirteen

October 13, 1980

Martischnis ['79] supports a dose response, threshold relationship between asbestos and pulmonary carcinoma in his study of asbestos bodies found in autopsies of pulmonary carcinoma cases. He observed a statistically significant relationship between asbestos bodies and known history of asbestos exposure. He also found a higher rate of anaplastic carcinoma in patients with asbestos bodies than in patients without, suggesting a specific tumor cell type relationship to asbestos exposure. Weiss ['77] cohort of workers in a chrysotile manufacturing plant had lower exposures than most other studies and found pulmonary carcinoma standard mortality ratios below 1.0 [at 0.93].

Whitwell ['77] supports a threshold level for pulmonary carcinoma for cigarette smokers based on his autopsy findings of no difference in asbestos fiber counts in the lungs of patients with pulmonary carcinoma regardless of whether they lived in an urban or rural environment prior to their death. Goff ['72] identified asbestos related disease in an individual whose exposure was in the manufacture of asbestos cigarette filters. [I am wondering if the increase in pulmonary carcinoma in smokers could be related to asbestos in the cigarette filters rather than other intrinsic synergistic biological interactions?]

Animal data, although showing a clear dose response relationship, has not really tested for threshold levels. Leong ['78], Lynch ['57], Gross ['74], Reeves ['74, '76], Wagner ['74], and Noordman have conducted inhalation animal studies using exposure levels 100 to 1000 times in excess of Garlock product users' exposure, and these exposures were administered within a very short period in comparison to the working lifetime of a Garlock product user. In mice and rats pulmonary tumors have occurred with exposures as short as 1 day (Wagner '74). Wagner's one day dose, however, was approximately equal to the same amount a Garlock product user would encounter over a 10 year period. Reeves ['74] study of rabbits, guinea pigs and gerbils found no pulmonary tumors. Goldstein ['78] found cancer in one out of 12 baboons who were subjected to approximately 166 fibers per cc for 266 days [approximately 100 times the total lifetime dose of Garlock product users].

056

CRMC-M&S-002988

Mr. Jamie B.W. Stecher
Page fourteen

October 13, 1980

In summary, the animal and human data is strong evidence for a causal dose response relationship between asbestos and pulmonary carcinoma, but it has not yet been tested for carcinoma outcomes at sufficiently low levels to determine whether or not a threshold exists. Furthermore, even if no safe thresholds exist, the risk for pulmonary carcinoma due to exposures consistent with Garlock product use appears extremely low. Cigarette smoking confounds and increases the risk of asbestos related pulmonary carcinoma and appears to have a multiplicative relationship.

V. Is there a threshold level for asbestosis and what is the risk of asbestosis at the levels found in Garlock product users?

As with asbestos induced pulmonary carcinoma, airborne asbestos exposure levels which have been associated with asbestosis [diffuse interstitial fibrosis] have been in the order of 100 to 10,000 times that expected for Garlock product users, whether expressed as cumulative lifelong exposures or average daily exposures. Asbestosis incidence rises with both intensity and duration of asbestos exposure [Enterline '79, IARC '77, Sellikof '78]. Although a few family contacts have developed, x-ray or clinical signs of asbestosis or other possible causes thereof were not addressed and no exposure data was available. Neighborhood proximity to asbestos plants may have caused additional and measured asbestos exposure in these patients. In attempts to define a threshold Berry noted no significant x-ray findings in asbestos exposed persons with less than 25 fiber/years of exposure [Garlock product users have approximately 1.5 fiber/years exposure]. Berry ['79] estimates however that to assure a 1% prevalence of asbestosis, exposures should be in the range of approximately 1 fiber per cc or 55 fiber/years, but this projection is based on cases occurring as the result of much higher exposures. Berry states that it is not possible to assess effects of relatively low doses [2 fibers/cc] with today's data. Berry also found normal lung function tests in individuals whose cumulative dust exposure was approximately 35 fiber/years [20 times that of Garlock product users]. Merewether and Price [1930] report of examinations of textile workers

050

CRMC-M&S-002989

Mr. Jamie B.W. Stecher
Page fifteen

October 13, 1980

formed the basis for the exposure levels during the 50's and 60's. Using duration of employment as an exposure indicator, they found no evidence of disease in persons with less than 5 years employment. Additionally, early evidence of the relationship between high asbestos exposure and asbestosis developed due to the appearance of this disease almost exclusively in only the heavily exposed workers [Smithers '75].

The lowest cumulative doses related to asbestosis mortality were in the range of 100 or more fiber/years exposure and they did show a moderate increase in mortality. However, McDonald [1979] found no cases of asbestosis in individuals exposed to approximately 10 fibers per cc for 20 or less years, although one asbestosis mortality did occur in the short duration, high intensity category. This supports the concept of a cumulative dose response mechanism. McDonald further implies that at cumulative exposures of less than approximately 100 fiber/years (or 2 fibers/cc) there is no increase in risk. Murphy's data ['71] shows evidence of asbestosis only above 60 million particles per cubic foot/years (approximately 120 to 200 fiber/years). Animal studies have supported a dose response relationship but have not adequately tested for a threshold level. Wagner's ['74] rat inhalation studies shows minimal evidence of asbestosis in rats subjected to 12 mg per cubic meter of asbestos for up to 6 months. The mean cumulative dose of asbestos to which these animals were exposed was 8400 mg per cubic meter which is roughly 5 to 10 times that expected over the working lifetime of a Carlock product user.

Although the data for a dose response relationship is strong, data establishing a safe level has not been developed. However, there does appear to be a consensus in the medical literature that low exposure levels do not result in the risk of asbestosis occurrence and do not merit the concern that is found in relation to similar issues surrounding pulmonary and mesothelial malignancy [Sellikof '78]. It is the recommendation of the National Institute of Occupational Safety and Health ['78] that a time weighted average of .1 fiber per cc would protect workers from the occurrence of asbestosis.

061

CRMC-M&S-002990

Mr. Jamie B.W. Stecher
Page sixteen

October 13, 1980

As of July 5, 1980, the State of California Department of Occupational Safety and Health [Cal OSHA] established 0.1 fibers/cc as its action level [General Industry Safety Orders section 5208(J)(1) p. 442.2.1]. In the medical literature I reviewed I could find no evidence in support of a lower standard. Based on the meager data above, I suspect that the probability of asbestosis occurring due to exposure to Garlock products only would be in the order of zero to 1 per 100,000.

VI. When should Garlock have suspected a hazard due to the asbestos exposure generated during the use of its products?

Because the California Occupational Safety and Health Administration standard continues at 2.0 fibers per cc, because most other industrialized nations have a recommended asbestos exposure standard between 5.0 and .1 fibers per cc, because there is no good data showing toxic effects at levels lower than 2.0 fiber per cc and because Garlock product user exposures are and previously have been well below that, I do not feel that Garlock should have ever suspected a hazard and I don't believe a hazard exists for persons whose only occupational exposure to asbestos occurs as a result of the use of Garlock products. Even if it were ever shown to exist that a pathological effect could occur at this low level of exposure, the Garlock exposure levels are comparable in many instances to those found in non-occupational environments, and it would be very difficult to determine the relative contributions of the occupational vs. the non-occupational exposures. Because of the existence of natural asbestos deposits and man made non-occupational exposures [general purpose buildings], asbestos exposure occurring as a result of Garlock product usage barely exceeds that which could be encountered involuntarily in the non-occupational environment.

Please be advised that the preceding statements, analyses and conclusions are based on the assumption that the environmental measurements of asbestos generated by use of Garlock products are accurate and valid.

062

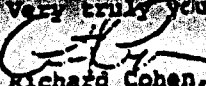
CRMC-M&S-002991

Mr. Jamie B.W. Stecher
Page seventeen

October 13, 1980

Attached is a list of the references cited and I have additional references and articles in my office obtained during the process of gathering this data. I will be glad to forward to you all or any part of the references I have obtained in preparing this report. Unfortunately, because some of the references are in obscure or foreign journals, I have not been able to review all of the articles I have requested, but will continue to attempt to get these articles. This has been a laborious yet most enjoyable experience and I am greatly looking forward to your assessment of this report. If you have any further questions, comments, or if I can be of any further service, please do not hesitate to call. I look forward to hearing from you.

Very truly yours,


Richard Cohen, M.D., M.P.H., F.A.C.P.M.

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063

CRMC-M&S-002992