PLAINTIFF'S EXHIBIT

UC-3832

United States Department of the Interior

GEOLOGICAL SURVEY
RESTON, VA. 22092

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In Reply Refer To
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February 28, 1985

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R. C. LEWINSOHN, M.D.

Dr. Bernard D. Goldstein, M.D.
Assistant Administrator for Research
and Development
United States Environmental Protection
Agency
401 M St., S.W., Washington, DC 20460

Dear Dr. Goldstein:

This letter is in reply to your request for my comments on the paper by Dr. Churg and coworkers titled "Lung asbestos content in chrysotile workers with mesothelioma." I am quite familiar with Dr. Churg's previous work in the area of fiber counting and identification in lung tissue. I have found his work to be well done, very useful, and in good agreement with data published by other scientists. His present paper published in the American Review of Respiratory Disease (Vol. 130, p. 1042-1045, 1984) is a significant contribution, the major points being: (1) the ratio of amphibole to chrysotile fibers detected in the lung tissue of mesothelioma victims is approximately eight to one, and (2) the concentration ratio of tremolite in the lungs of the mesothelioma cases compared to controls is 9.3, while the ratio of chrysotile is only 2.8. The authors state on page 1045, "Our observations in chrysotile mine industry workers thus raise the possibility that the amphibole component of the chrysotile ore is important in the genesis of mesothelioma in this group."

It would appear from the work of Churg, et al. and other similar reports, that the human lung has the ability to concentrate amphibole over chrysotile even though the workers have been exposed to rock dust containing much more chrysotile than amphibole. A similar observation is very strikingly made by Gylseth et al. (Brit. J. Ind. Med., Vol. 40, p. 375-379, 1983). Their analysis of fiber type in the lung tissue of eight workers from a Norwegian asbestos cement plant who died either of pleural mesothelioma or lung cancer showed that the percentage of amphibole asbestos in the tissue varied between 76% and 99% whereas the percentage of chrysotile varied between 0 and 9%. Yet the use of asbestos in the cement plant during the period 1942 to 1980 was 91.7% chrysotile and 8.3% amphibole asbestos (4.1% crocidolite, 3.1% amosite, 1.1% anthophyllite)! This extraordinary reversal of fiber ratios between exposure and lung burden supports an idea alluded to in previous medical publications -- that chrysotile fibers are somehow cleared from the lung, perhaps by slow dissolution (we know that chrysotile is more soluble than amphibole asbestos). Other clearance mechanisms may also preferentially remove chrysotile.

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Concerning asbestos exposure in the non-occupational setting, it is of interest to note that measurement of fibers in the lung tissue of 20 urban dwellers who had no history of occupational exposure to asbestos showed that 84% of the fibers counted were chrysotile, 16% amphibole. Of the amphibole, 95% was of the non-commercial type (Churg and Warnock, Am. Rev. Resp. Disease, Vol. 122, p. 669-678, 1980).

I do not believe that the paper by Churg et al. should be treated as an epidemiological study but rather as a census of fiber type within the lung tissue of mesothelioma victims. Nor do I believe that this paper can be interpreted as disagreeing with the conclusion of Dr. J. C. McDonald and coworkers -- the conclusion that chrysotile has a low potential to cause mesothelioma. I might cite Dr. Churg's previous work in this regard: (1) "These observations suggest that most mesotheliomas are associated with increased numbers of commercial amphiboles and not with chrysotile asbestos" (Churg, Hum. Pathol., Vol. 13, p. 381, 1982), and (2) "Mesothelioma is rare in chrysotile miners" (Churg and Wiggs, Am. J. Pathol., Vol. 115, p. 441, 1984).

Perhaps I misunderstood your impression of Dr. Churg's paper. In my opinion it does not say that chrysotile asbestos causes mesothelioma in Quebec asbestos miners; it does not pertain in any way to the total incidence or incidence rates of this disease in those exposed to chrysotile dust. Mesothelioma is a rare disease among the workers and residents of the Quebec asbestos mining towns as documented by J. C. and A. D. McDonald and coworkers, G. P. Theriault, L. Grand-Bois, R. Pampalon, P. Toft and coworkers, and J. Siemiatycki.

A striking example of low mesothelioma mortality in chrysotile factory workers is given by McDonald and Fry (Scand. J. Work Envir. Health, Vol. 18, Suppl. 1, p. 53-58, 1982). Two factories, one in South Carolina and one in Connecticut, used predominantly chrysotile asbestos. Among the 2,341 deaths in these two chrysotile factories there was one mesothelioma death (recall that McDonald et al. report 11 cases of mesothelioma in a series of 4,547 deaths in the Quebec chrysotile miners and millers). Contrast this to a third factory in Pennsylvania which used amosite and crocidolite asbestos in addition to chrysotile asbestos. Among the 1,429 deaths in workers at this factory using mixed amphibole-chrysotile asbestos there were 18 mesothelioma cases. Or, consider 67 mesothelioma cases among 745 deaths in a London factory cohort exposed to amosite, crocidolite, as well as chrysotile (Newhouse and Berry, Annals NY Acad. Sc., Vol. 330, p. 53-60, 1979). McDonald (Annals Acad. Med., Singapore, Vol. 13, No. 2, Suppl., p. 345-352, 1984) summarizes mesothelioma incidence in 30 male cohorts as follows:

Type of Exposure (cohorts)		No. of Deaths	No. of Mesotheliomas	Rate/1000
Chrysotile	(8)	6,539	15	2.3
Amosite	(2)	959	19	20.0
Anthophyllite	(1)	248	0	-
Crocidolite	(2)	279	33	118.3
Tremolite	(2)	165	3	18.2
Mixtures	(15)	10,728	369	34.4
Total	(30)	18,918	439	23.2

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The possible health effects of exposure to rock dust containing one or more of the many amphibole minerals is an important issue. Amphiboles are contained within the gangue (waste rock) of many hard-rock mines; gold, vermiculite, talc, iron ore, crushed stone and aggregate, copper, etc. Certainly control of most dust, regardless of the mineral content, is necessary for past heavy exposure to dusts containing crystalline silica (SiO_2), slate, coal, talc, and radio-active minerals have caused significant disease. Unusually tight control of amphibole mineral particles such as proposed by NIOSH in 1976 for asbestos fibers (0.1 fibers per cm^3), however, would stop major mining in the United States.

Several epidemiological studies have been performed on hard-rock miners who were exposed to non-commercial amphibole dusts. One example is a cohort of gold miners at the Homestake Gold Mine, Lead, S. D. who were exposed to rock dust containing very significant amounts of amphibole belonging to the cummingtonite-grunerite series. The 861 observed deaths included 43 lung cancer deaths (43 expected) and no mesothelioma deaths. Non-malignant respiratory disease due to quartz dust, however, was excessive. This cohort thus showed no evidence of mortality that could be related to amphibole dust (Brown et al., symposium, April 5, 1984, unpublished preprint). Studies of Reserve taconite (iron ore) miners who were exposed to cummingtonite amphibole contained in the mine dusts also show no amphibole-related disease (Ross, ASTM STP 834, p. 75, ref. 43, 1984). It should be noted that no mesothelioma deaths have been reported in Finnish anthophyllite asbestos miners and millers (Ross, ASTM STP 834, p. 74, Ref. 8, 1984).

In addition to the very common exposure of man to naturally occuring fibrous minerals, he is also exposed to many different types of synthetically made fibers. More and more of these substances are being developed each year in the greatly expanding fiber and composite industries. Our knowledge of the health effects of most of these fibers is minuscule or non-existent. The health effects of those exposed to man made vitreous fibers, however, have been studied intensively and statistically significant studies are just now being reported. These glassy fibers, usually referred to as man-made mineral fibers (MMMF), include slag wool, rock wool, glass wool, fiber glass, and continuous filament products. Two separate reports, one of 25,146 workers at 13 plants in Europe (Saracci et al., Brit. J. Ind. Med., Vol. 41, p. 425-436, 1984), and one of 16,730 workers at 17 plants in the United States (Enterline and Marsh, WHO-EURO, Biol. Effects Man-Made Mineral Fibers, Proc., in press) have just been published or are about to be published. The table below gives a summary of the lung cancer mortality for male workers in the European and United States MMMF industries who lived at least 30 years since first exposure.

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[redacted]

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TABLE

LUNG CANCER MORTALITY, MALES, AT LEAST 30 YEARS OBSERVATION SINCE FIRST EMPLOYMENT IN MMMF INDUSTRY

	LUNG CANCER		
EUROPE (13 FACTORIES)	(Observed)	(Expected)	(Excess)
Rock wool	11	5.7	93%
Glass wool	4	2.6	54%
Continuous filament	2	0.6	233%
Subtotal	17	8.9	91%
UNITED STATES (17 FACTORIES)	(Observed)	(Expected)	(Excess)
Glass wool	47	36.0	31%
Slag wool	45	28.1	60%
Rock wool	14	8.1	73%
Subtotal	106	72.2	47%
TOTALS:	123	81.1	52%

Thus, a statistically significant excess of lung cancer (52%) is seen in these workers who lived at least 30 years since first exposure. Exposure was generally less than 1 fiber per cm^3 and most commonly in the range of 0.01 to 0.1 fibers per cm^3. Recall that Quebec chrysotile asbestos miners and millers with at least 20 years service in the industry and exposed to between 10 and 21 fibers per cm^3 showed an excess of lung cancer of 12%. (Ross, ASTM STP 834, p. 93, 1984). If one had a vendetta against fiber glass it could be said from these data that, fiber for fiber, man-made minerals products may be more dangerous than chrysotile asbestos. There is ample epidemiological evidence available to show that under controls now in place at modern well run plants, except possibly asbestos textile factories, risk due to chrysotile will be difficult, perhaps impossible to detect. The very low risk of non-occupational exposure to chrysotile asbestos has been well documented by various studies of the mortality of the women of the Quebec chrysolite asbestos mining towns, women who did not work in the asbestos industry but who were exposed to very high levels of ambient chrysotile dusts from the local mines and mills throughout their lives, 24 hours a day (Ross, ASTM STP 834, p. 82-86, Ref. 70, 71, 72, 73, 81, 86, 1984; see also J. Siemiatycki, Proc. World Symp. Asbestos, Montreal, May 25-27, p. 337-348, 1982).

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Many types of fibers produce tumors in animals subjected to suitable types of experiments. All forms of commercial asbestos (except perhaps for actinolite asbestos) and various forms of man-made mineral fibers have been shown to cause cancer in man, usually after long-term exposure. In view of these facts what are we to say about control of fibrous materials in our environment? Should not all fibers falling within a critical size range be regulated? If so, to what levels? If all asbestos is considered equally dangerous, the prevailing opinion in the United States, should all other fibers also be considered as equally dangerous? If all fiber in the critical size range is banned, such as presently being attempted for major uses of asbestos or regulated to 0.1 fibers per cm^3 (NIOSH proposal), what industries will be affected? Is it proper to assume that a substitute for asbestos is safe when there are no scientific data on human exposure to support such an assumption? Recall that a great variety of fibers, when implanted into the pleura of rats, cause tumors. Is it in the public interest to ban the use of materials which have had significant benefits, such as fire-proofing, insulation, and materials strengthening, when proposed substitutes have not been proven to furnish similar benefits or economy?

The prevailing cancer dogma in the United States espouses the "no threshold" theory for cancer genesis. It is said repeatedly by influential health specialists that since no one knows the minimum amount of a carcinogen required to initiate the growth of a cancer tumor, it must be assumed that any amount of any carcinogen is unsafe. Thus, what is the school administrator to do when he is required by law to post signs in his schools stating that asbestos is present? He has no option -- he has to have it removed for our cancer dogma translates the words on his building sign to "a little child breathing just one asbestos fiber may get mesothelioma 30 years later." The school administrator, the teachers, the parents, are in a box. The only way out of the box is to call for full ripout of the asbestos even though well informed experts know that many asbestos ripouts, perhaps most, will put more fiber into the air than if it were left in place. And what are we to do about asbestos substitutes that also may be carcinogenic in man -- for example the man-made mineral fiber products such as rock wool and fiber glass? Since these products apparently cause excess lung cancer in long-term production workers a logical and honest cancer policy would require that they also be removed from schools and homes.

The presence of asbestos in schools, homes, and other buildings is generating a national crisis of such proportions that the U.S. economy could be very adversely affected; this because full ripout of asbestos is the only viable option in view of the "one fiber can kill you" concept. Building owners, school systems, and local and state governments are suing many businesses for costs and damages related to asbestos removal, insurance companies are dropping liability coverage on workers removing asbestos, and occupants of buildings are suing over health effects or potential effects alledged to be caused by exposure to asbestos during renovation. The asbestos removal workers themselves, I predict, will soon be suing their contractors because they too will believe that their health is threatened by exposure to asbestos. Finally, this whole scenario may soon be replayed, the main actor being fiber glass products rather

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than asbestos. Where will this all end? When a dozen major corporations go bankrupt? When the court system becomes so clogged with lawsuits that it can no longer keep up? When people become to afraid to work or live in any buildings that contain asbestos? When enough buildings are closed and vacated for ripout, putting an inordinate number of people out of work? And all this because a small number of people hypothesize a small health effect due to non-occupational exposure to asbestos in buildings, a postulated effect too small to measure in any feasible epidemiological study.

In a letter to Mr. Don R. Clay, then Director of the Office of Toxic Substances, EPA, I predicted that such a crisis might occur. This letter, dated 17 December, 1981, was a response to a request from Mr. Clay that I critically review an EPA Guidance Document on asbestos-containing materials in schools. I wrote then that "fear of asbestos" might require removal of asbestos from all buildings, homes, appliances, as well as replacement of asbestos cement water pipe. I also wrote that the requirement to notify the building occupants that asbestos is present may cause them to refuse to work in the building until all asbestos is removed. This correspondence is enclosed.

Concluding, I think it is imperative that our national health policy be re-evaluated to recognize that many products, though carcinogenic under certain conditions, can be manufactured and used safely under economically feasible controls. This policy should also recognize that it does not make sense to spend huge sums of money to reduce or eliminate very small risks, for example, attempting to reduce cancer risk in schools through a potential multi-billion dollar asbestos removal program, when money (and initiative) is lacking to reduce the huge health risks associated with poor nutrition, tobacco use, and abuse of drugs and alcohol.

I hope that this letter will be of use to you and other decision makers in gaining a somewhat different perspective on this asbestos and health issue.

I thank you for this opportunity to reply to your letter.

Very sincerely,

Malcolm Ross

Malcolm Ross, Ph.D.
Research Mineralogist

12 Enclosures:
Scientific papers by:
Churg et al., 1984
Gylseth et al., 1983
Churg and Warnock, 1980
Churg, 1982
Churg and Wiggs, 1984
McDonald and Fry, 1982
Newhouse and Berry, 1979
McDonald, 1984

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Scientific papers by (continued):
Brown et al., 1984
Ross, 1984
Saracci et al., 1984
Correspondence with Mr. Don R. Clay, 1981-1982

cc: Mr. Martin Smith
Mr. Donald Ryan
Dr. John A. Moore, D.V.M.
Dr. Andrew Churg, M.D.
Dr. J. C. McDonald, M.D.

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