

LETTER TO THE EDITOR

Response to Dr. C.-G. Ohlson

Key words: asbestos, railroad workers, mesothelioma, lung disease

With reference to Ohlson's first comment [1989] concerning the description of asbestos exposure, he says "it is stated that the machinists were almost 'exclusively, if not solely' exposed to chrysotile." A more careful reading of my paper [Mancuso, 1988, p 641] would show the text as follows: "From the evaluation of the data relative to the type of asbestos, year and amount and the time period use of various types of asbestos, it is reasonable to conclude that the type of asbestos used for the *lagging insulation* for the steam locomotives for railroad company A was almost exclusively, if not solely chrysotile, during the 1920-29 period."

That statement in the text was preceded by two paragraphs. The first emphasized the lagging operation: "In terms of historical perspective of exposure to asbestos in the railroad industry, *the principal and most extensive exposure in volume and dimensions* occurred in the *application and removal of asbestos lagging* on the boilers of steam locomotives." A description of these work practices was provided in Mancuso [1983]. Chrysotile was the type of asbestos used since the first application of asbestos in the steam locomotive era for the *lagging*, and this continued through the succeeding decades. In the next paragraph, it is acknowledged that there was a potential of other forms of asbestos being used in other parts of the locomotive: "In addition to chrysotile, if some other potential exposure to amosite or crocidolite as packing or other material occurred at some point in time, the actual exposure would have been *comparatively* limited in nature and scope."

The article clearly refers to *lagging* and *removal of lagging* asbestos as the principal and by far the most extensive exposure in terms of volume and dimensions. The boilers were usually 30-35 feet long, with a diameter of 7 or 8 feet. The asbestos lagging used was approximately 4 feet long, 4 inches thick, and 6 inches wide applied as blocks. For a 30 foot boiler, approximately 375 blocks of that asbestos insulation would be used for the outside of the boiler alone. With these dimensions, assuming 24 pounds to a cubic foot, this would represent approximately 6,000 pounds of asbestos insulation material. In the work practices described [Mancuso, 1983], asbestos exposure in the *removal* of the lagging was very great indeed, sometimes literally a "snow storm," as when huge chunks of asbestos lagging, tossed from a height of 10-25 feet into lagging wagons, created an enormous amount of dust. This

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States in 1920–1930 and 1931–1939, and this limited amount of fiber was never used for insulation (according to the largest manufacturer of asbestos products) but only for such special items as valve packings and braid.

This was recognized by Hueper [1965]. “Chrysotile furnishes the chief cause of asbestos pneumoconiosis and cancer, especially in the United States because contact with amosite and crocidolite have been comparatively minor, and these varieties represent a small fraction of the total amount of asbestos produced and used.”

It is inappropriate to compare the amounts and purposes of different types of asbestos that may have been used for insulation in Sweden with what has occurred in the United States, in particular for the study years designated in my railroad study.

The railroad facility for the Mancuso study of cohorts of machinists and other crafts was considered one of the largest railroad heavy duty overhaul shops in the world.

The Ohlson comment “even small amounts of amphiboles would have been sufficient to explain the occurrence of the mesotheliomas due to the high durability of the amphiboles and low durability of chrysotile in the lungs” is misleading. As cited in my text, Sébastien et al. [1980] have demonstrated that chrysotile has a predilection for the pleura; in their amphibole studies this tended to be found in the lungs, and chrysotile was found in the pleura. Within this context, more recently, Churg [1988, p 237] commented on this aspect relative to the lung burden and made several points: 1) chrysotile itself does not accumulate in the lung to any great extent (it is more readily dissolved in vivo compared with amphiboles); 2) this does not mean that only amphiboles are causing disease; 3) it would be most inappropriate for anyone to conclude that just because the lung burden was considerably less, for example, among cases of asbestosis than in a matched series of mesotheliomas, that chrysotile does not cause asbestosis; 4) these data on lung burden do indicate that chrysotile is a poor marker of the amount of chrysotile exposure; and 5) it is incorrect to conclude that chrysotile plays no role in mesothelioma. Indeed, Churg and his colleagues [1984] recently reported that in a consecutive series of 90 autopsies of Québec chrysotile miners and millers, six deaths were from pleural mesothelioma.

The premise of an agreed (?) latent period is questionable. Latent periods, observed in the various cohorts reported, are a reflection on the definition time of the cohorts and the period of follow-up. The Mancuso study, by the very nature of its design, had a much longer period of follow-up. The reality is that chrysotile causes mesothelioma and that the risk is much higher than had been previously appreciated, in addition to its widely acknowledged risk for other asbestos-associated diseases including the most common of all asbestos neoplasms, lung cancer [McDonald and Liddell, 1979; Dement et al., 1983].

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