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LETTER TO THE EDITOR

Amphiboles and Chrysotile Asbestos Exposure

Dr. Dunnigan eloquently and persuasively argues [1988] that in cases of patients with mesothelioma who have been exposed to both amphiboles and chrysotile asbestos, it is the amphibole exposure that is responsible for producing the tumor. Support for this argument derives from epidemiologic studies and analysis of pulmonary asbestos content. Few would disagree with the contention that the amphiboles are more potent than chrysotile asbestos in the production of mesotheliomas. Furthermore, environmental exposures to mineral fibers [Baris et al., 1979; Langer et al., 1987] as well as experimental studies [Stanton et al., 1981] indicate that it is the long, thin fibers that are most likely to produce mesotheliomas. Hence, factors other than fibertype are also relevant to the pathogenesis of mesothelioma. Durability and prolonged retention of fibers in tissues are probably additional important factors.

However, before accepting the conclusion advocated by Dr. Dunnigan, some additional points should be considered by the reader. Although only a few cases of mesothelioma are referred to in cohorts exposed exclusively (at least as far as anyone can ascertain) to chrysotile asbestos, there are additional case reports not mentioned by Dr. Dunnigan of mesothelioma occurring in patients allegedly exposed only to chrysotile-containing products [Castleman, 1984]. Even though the few cases reported in cohort studies may not seem impressive, they need to be considered in the context of the background rate of mesothelioma, which is one or two cases per *million* population per year [McDonald and McDonald, 1980]. Also, in consideration of the very long latency period of mesothelioma, cohorts need to be followed for 30 or 40 years in order to ascertain the full extent of asbestos-related disease in any given group of workers [Enterline et al., 1987].

One must similarly be cautious in drawing the conclusion advocated by Dr. Dunnigan based on tissue fiber analysis. The classic animal exposure studies by Wagner et al. [1974] showed that, with prolonged exposure to amphiboles, there was a progressive accumulation of asbestos fibers in the lungs that was proportional to the duration of exposure. However, during similar exposures to chrysotile, the tissue fiber burden reached a plateau after a couple of months and remained steady thereafter. If one assumes that this behavior might also occur with mixed fiber exposures, then it would follow that the ratio of amphiboles to chrysotile would increase with increasing cumulative exposure. There is some data based on analysis of lung tissue from chrysotile miners and millers to support this assumption (Churg,

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1984; Churg et al., 1984). Nonetheless, in Wagner's studies referred to above [Wagner et al., 1974], chrysotile was just as efficient in producing asbestosis as were the amphiboles. Furthermore, just as many mesotheliomas occurred in animals that breathed Canadian chrysotile dust as occurred in animals breathing crocidolite [Wagner et al., 1974]. Finally, few would argue with the statement that, in humans, asbestosis occurs in individuals exposed only to chrysotile dust or chrysotile containing products, in spite of the fact that chrysotile does not accumulate in lung tissue to the same extent that the amphiboles do.

When one considers these additional points, we do not believe one can be as complacent as Dr. Dunnigan suggests with the thought that, in patients with mesothelioma who have been exposed to both amphiboles and chrysotile asbestos, the latter plays no significant role in the production of the disease.

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