

Editorial

Role of manmade mineral fibres in the causation of cancer

The findings from two large epidemiological studies of manmade mineral fibre (MMMF) workers have recently been reported.^{1,2} These studies of over 40 000 production and maintenance workers were designed to determine whether MMMF have health effects similar to those of asbestos. Findings from these two studies, one conducted in the United States and one in Europe, were surprisingly similar. Workers who produced fibrous wool using rock or slag had a 30% increase in respiratory cancer 20 years or more after first exposure in the United States and a 40% increase in the European study; workers producing fibrous wool from glass had much smaller increments—roughly a 10% excess in both the United States and European studies. No excess was observed in a small group of workers who produced glass filament. In reviewing the findings from these two studies Doll concluded that there has been an occupational hazard of lung cancer among workers producing rock/slag wool and that there may have been a hazard among workers producing glass wool.³

One of the difficulties in attributing a causal relation between respiratory cancer and MMMF is that the estimated fibre concentrations to which workers were exposed were relatively low. At a meeting in Copenhagen in 1982 McDonald reviewed the epidemiological findings available at that time and thought that, given the low exposure levels, a greater excess of lung cancer would not be observed "even if we were dealing with asbestos."⁴ In reviewing updated data in 1986 Doll made a similar comment.⁵ He thought that the low fibre counts observed were difficult to associate with any risk in the light of our knowledge of the effects of chrysotile asbestos and that the observed risk would suggest that MMMF must be more carcinogenic than chrysotile asbestos. That is, even had the studies dealt with workers exposed to chrysotile asbestos, excesses of the magnitude observed would not have been expected. As an alternative explanation for the apparent high potency of MMMF Doll suggested that perhaps the exposure estimates were too low.

It seems unlikely that MMMF is more carcinogenic than asbestos. In fact the animal studies indicated that fibre for fibre MMMF is much less

carcinogenic than asbestos.³ The explanation for this appears to be that on average MMMF are less durable than asbestos and, rather than breaking longitudinally as does asbestos so as to form thinner and thinner fibres, MMMF tends to break laterally. As for the exposure estimates in the two large studies, these were the result of a considerable effort and there is remarkable agreement between those made for the United States study and for those made for the European study. For rock/slag wool workers where exposures were believed to be highest an important element was the estimation of exposures that occurred in the distant past, since these are the exposures that are likely to be responsible for any excesses of cancer observed to date. For the United States study a key to the historic fibre estimates for rock/slag wool workers was a 1934 study made of a mineral wool production plant.⁶ From this it was estimated that plantwide exposure to respirable fibres seen by light microscope during the early years was about 1.5 fibres per cc.⁷ This level was closely approximated in a European study in which there was an experimental simulation of a rock-wool production process with environmental conditions similar to those in the 1940s.⁸ Actual estimates of fibre exposure were made only in the United States study and suggest that for rock/slag wool workers exposures averaged 0.3–0.4 respirable fibres per cc whereas for glass wool workers exposures were about a tenth as high. The United States data suggest that the rock/slag wool workers in the United States study had on average a cumulative exposure 20 or more years more after first exposure of around 10 fibre per cc years. A widely accepted estimate of the effects of such an exposure for asbestos is that there is a 1% increase in lung cancer for each fibre per cc year of exposure.⁹ Thus it would be expected that in the rock/slag worker cohorts, where exposure was greatest, no more than a 10% excess in lung cancer should have been observed. In the light of this the 30–40% excesses seen in the United States and the European studies of rock/slag wool workers are curious indeed.

One explanation for the excess in the rock/slag wool industry is that asbestos may have been used extensively in the past, despite the fact that it was not

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the intention of the investigators to study the health effects of asbestos. Nevertheless, data have now been made available by the rock/slag wool industry which makes it fairly certain that asbestos was used in four of the six plants in the United States study. The proportion of workers in the areas where exposure to asbestos could have occurred was not large, however, and in the four plants where asbestos was used the excess of respiratory cancer is no greater than that in the two plants where it was not. In the European study there was a similar experience. Four rock/slag wool plants were identified where asbestos was used; however, there was no excess of deaths from lung cancer in these plants when compared with plants that did not use asbestos.

There is a problem in comparing epidemiological studies of workers who produce MMMF with studies of workers who use asbestos. Workers who produce MMMF are exposed to a wide variety of disease causing substances in addition to the fibres themselves. These include furnace fumes that include polycyclic aromatic hydrocarbons and contaminants in the feed stock. The asbestos workers who were the basis for the estimate by Doll and Peto that each asbestos fibre per cc year results in a 1% increase in lung cancer did not have such exposures.⁹ Thus some of the excess of cancer in MMMF workers might be due to exposures other than exposure to fibres.

There is some evidence that the excess of cancer in rock/slag wool workers is due mainly to the use of slag rather than rock. This was investigated in the European study.² During the use of slag as the feed for mineral wool plants, lung cancer mortality 20 years from first exposure was about double that expected whereas when slag was not used deaths were about as expected. When the slag was received from a copper smelter and was believed to be contaminated with arsenic deaths were two and a half times expected. In that study, however, there is a question as to whether exposure to fibres might have been higher during the use of slag. In the United States study four of the six mineral wool plants used slag almost exclusively. The other two plants used rock in the early years and then changed to slag. Estimated fibre exposure levels in the plants that used slag were not higher than exposures in plants that used rock. As of the end of 1982, the four plants that always used slag had a respiratory cancer death rate 20 years from first exposure double that expected, whereas the two plants that used rock had no excess of respiratory cancer. In one United States plant that received slag from a copper smelter and which was known to be contaminated with arsenic deaths from respiratory cancer 20 years from first exposure were three and a half times expected.¹ Thus the United States data support the European data in that it appears that the use of slag in producing mineral wool may play a part in the excess of respiratory cancer seen in rock/slag wool workers.

When all the epidemiological data on MMMF

workers are examined it is the data on slag wool plants that show the most impressive excess of respiratory cancer. It is doubtful whether much meaning can be attached to the smaller excesses among glass wool workers and there are no excesses in rock wool plants. Cancer causing contaminants in the slag may be one of the reasons for the excesses. What needs to be known is whether these contaminants are incorporated in the fibres themselves. Probably, however, fibres do not play the major part in the excess of cancer among slag wool workers so that fibre potency should not be estimated from epidemiological data on workers who produce these fibres.

Recently, the United States Environmental Protection Agency proposed to classify rock/slag wool as a probable human carcinogen and glass wool as a possible human carcinogen.¹⁰ Using a weight of evidence approach and attributing all of the observed excesses in respiratory cancer in the two large epidemiological studies to fibres, this appears to be a reasonable classification. Unfortunately to most people this will appear to be a classification of the potency of rock/slag wool fibres and glass wool fibres and as such is probably in error. It will lead fibre users to conclude that rock/slag wool is more hazardous than glass wool. This could be a disaster for the rock/slag wool industry and is probably misleading to the public. Neither animal experiments nor any known attributes of these two types of fibre would lead to such a conclusion. There is undoubtedly some small cancer hazard attached to ordinary MMMF whether made from rock, slag, or glass, a hazard much less than that of asbestos and perhaps some other kinds of MMMF.

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Department of Biostatistics
Graduate School of Public Health
University of Pittsburgh, 130 De Soto Street
Pittsburgh, PA 15261, USA

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