arguments based on the assumed equivalence of the fibre types to cause lung cancer. They also show an analysis of the mesothelioma mortality of a small subset of the US insulators study which shows that the pattern of deaths over time implies that members of this cohort were exposed to a pleural carcinogen before 1935. Since, reportedly, amosite was first used from around 1935, and prior to this date only chrysotile was used, some of these deaths must have been due to chrysotile. The authors do not mention the possible role of crocidolite, but if we accept that no amphibole fibre was used before 1935 by US insulation workers, these observations do show that some of the cases in this cohort must have been caused by exposure to chrysotile prior to 1935. These exposures will often have been heavy. In fact the contemporary US trade journal 'Asbestos' (published monthly from July 1919) makes it clear that both amosite and crocidolite were used in the US through the 1920s, though probably in limited quantities, since the US industry seems to have been resistant to their use on technical grounds, and chrysotile was the fibre of choice for most applications.

Stayner *et al.*'s review of the issues (1996), sets out similar arguments, and also points to the evidence that all three commercial fibre types have produced a similar level of lung tumours in animal inhalation experiments. This is the most problematic evidence to reconcile with the human evidence that amphibole fibres are substantially more potent lung carcinogens. However, the time periods needed to induce cancer in humans (yr) and in rats (months) are very different, and it is at least plausible that all fibres are equally potent in rats because none of them are materially cleared from the rat lung over the months needed to initiate a rat lung tumour. By contrast, in humans chrysotile (cleared in months) might have less effect than the amphibole fibres (cleared in years). A detailed elaboration of this argument has recently been published by Berry (1999). It may also be relevant that the animal experiments were made with exposure concentrations massively in excess of those represented in the human data. The differences in the human data summarised in this review seem reasonably clear (certainly in respect of mesothelioma), and are based on a range of independent data. In the end, if a choice has to be made between animal and human evidence as a basis for assessing human risk, adequate human data must be given priority.

Many of the arguments presented against the 'amphibole hypothesis' in connection with mesothelioma are variants on the basic theme that it is simply unbelievable that such a small component of the exposure could be responsible for the observed risk. If it is true that the mesothelioma risk is proportional to a less than unit power of exposure, then these arguments are correct in their basic perception that a disproportionate effect of the amphibole component was required to explain the data. Low levels of amphibole *do* have a disproportionate effect.

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