This review has presented evidence suggesting that this may not be the case. Uncertainty about the slopes of exposure–response lines has an increasing impact with increasing distance from the observed range. Also the strength of qualitative arguments such as those advanced in the HSE review (Meldrum, 1996), in favour of a threshold for the lung cancer effect increase as exposure falls.

All the above implies that simply to present a table of risk estimates—or even risk ranges—for different cumulative exposures cannot capture the changing balance of the different kinds of uncertainty. Table 11 gives a verbal assessment of risk at a range of representative cumulative exposures. No estimates have been given for lifetime risks lower than 1 in 100 000, and this level is referred to as 'insignificant'. A lifetime risk of 1 in 100 000 corresponds to an annual risk well below 1 in a million, which HSE has suggested (Health and Safety Executive, 1999) as a "guideline for the boundary between the broadly acceptable and tolerable regions [of fatal risk to an individual]." It is also well below the level at which it is suggested that mesothelioma would occur in the absence of asbestos exposure: a clear majority of the very few mesotheliomas that would occur at this level would not be caused by asbestos.

Mesothelioma risks in the observed cohorts have been expressed as a percentage (P_M) of total expected mortality in order to standardise observations from different follow up configurations. To make predictions of risk this measure must be converted back into absolute terms, and this is done using the average male life table discussed in Appendix A. For exposures starting at age 30 the excess mortality estimate P_M is applied to the total expected mortality from age 40 to age 79 (allowing a 10 yr minimum latency, and truncating risk at age 80). The life table predicts that about 70% of survivors to age 30 will die between the ages of 40 and 80. Absolute risk estimates can therefore be derived from the P_M value for a given exposure by multiplying by a factor of 0.7.

Lung cancer risks have been expressed as a percentage excess of expected lung cancer mortality. The major determinant of this underlying lung cancer risk is smoking—especially cigarette smoking—and the number of asbestos-related lung cancers will be affected by the prevalence of smoking in the exposed population. Currently (in 1997) about 9.5% of male deaths between the ages of 40 and 79 are due to lung cancer. For women the figure is 7%, reflecting differences in past smoking. Total survival to age 80 is lower in men than in women, and combining data for survival and proportionate mortality from lung cancer it can be predicted that for 1000 30-yr-old men 54 will die of lung cancer between the ages of 40 and 79. For women the number is 28. Thus for a population with the past smoking habits of British men aged 60+ (the ages at which most lung cancers occur), the lung cancer risk from asbestos exposure is given by $0.054P_L$. For women with typical past smoking habits the figure would be $0.028P_L$.

Table 11 makes statements about the lifetime risks of exposures accumulated over short (up to 5 yr) periods from age 30. The factors given in Table 10 can be used to apply the mesothelioma estimates to other ages at exposure. The lung cancer estimates are based on 1997 male lung cancer rates. They are not sensitive to age at exposure.

For the lung cancer risk due to chrysotile two principal figures are given: a best estimate and a cautious estimate. A risk estimate derived from the Carolina cohort is also given, with the qualification that this might be arguable in 'exceptional circumstances'. These exceptional circumstances cannot be defined with any certainty since the features of exposure at this plant responsible for the very high lung cancer risks there are not known. Exposure to textile grade (i.e. long fibre) chrysotile is presumably necessary, but does not seem to be sufficient, since other textile plants have recorded much lower exposure-specific risk (even with additional exposure to amphibole fibre). The spraying of the raw fibre with mineral oil (as a dust suppression measure) has been suggested as a possible explanation. This hypothesis seems to be supported by a case-control study of lung cancers at Carolina (though the relevant results have not been fully reported), and by observations from another asbestos textile plant (Rochdale), where men first employed after oil spraying was introduced had three times the exposure-specific risk of those first employed in earlier periods (though still lower than the Carolina risk).

The main uncertainties in this picture relate to the effects of chrysotile, particularly at low doses. The application of these estimates in the assessment of a particular risk situation will depend on the purposes of that particular assessment, and the extent to which a precautionary approach is appropriate.

DISCUSSION

There have been a number of papers (Cullen, 1998; Stayner *et al.*, 1996; Nicholson and Landrigan, 1996; Smith and Wright, 1996), in the literature recently which directly or indirectly consider whether there are differences in potency between the fibre types as causes of mesothelioma and lung cancer. The claim that there are important differences is often described as 'the amphibole hypothesis'. In its strongest form this has been said to claim that pure chrysotile (i.e. without any associated tremolite fibre) would present little or no carcinogenic risk. At the other extreme, it has been argued (Smith and Wright, 1996), that there is virtually no difference between the risks presented by the different fibre types. Most commentators (e.g. Doll and Peto, 1985; Hughes and Weill, 1986; Health Effects Institute, 1991) have considered that the amphibole fibre types are more dangerous, parti-