Table 8. Estimated coefficients[a] with 95% confidence intervals for constants in the risk prediction equation for P_M at three levels of the slope coefficient r

Slope/Fibre	A_M	95% CI	A_P	95% CI
Best estimate slope (r=0.75, t=2.1)				
Crocidolite	0.94[b]	(0.71,1.2)	0.0022	(0.0011,0.0039)
Amosite	0.13[b]	(0.060,0.25)	0.0006	(0.00025,0.0012)
Chrysotile	0.0047[b]	(0.0030,0.0069)		
High slope (r=1, t=2.5)				
Crocidolite	0.43	(0.33[b],0.54)	0.00053	(0.00029,0.00087)
Amosite	0.052	(0.022[b],0.099)	0.00012	(0.000049,0.00024)
Chrysotile	0.000970	(0.00064[b],0.0014)		
Low slope (r=0.6, t=1.7)				
Crocidolite	1.5	(1.1,1.9[c])	0.0083	(0.0043,0.014)
Amosite	0.24	(0.11,0.44[c])	0.003	(0.0013,0.0058)
Chrysotile	0.012	(0.0078,0.018[c])		

[a]Coefficients used for risk extrapolation at low doses shown in bold:
[b]best estimate, [b]lowest arguable, [c]highest arguable (see Table 11). Numbers of peritoneal mesotheliomas at low doses are negligible. For short exposure, chrysotile coefficients should be multiplied by 1.4.

Table 9. Adjustment factors to convert estimates of mesothelioma mortality due to asbestos exposure starting at age 30 to other exposure start ages

Age	20	25	35	40
Factor	2.1	1.5	0.6	0.4

Table 10. Estimated coefficients with 95% confidence intervals for constants in the risk prediction equation for P_L for chosen levels of the slope coefficient r

Fibre/model	A_L	95% CI
Amphibole		
Linear (r=1)	4.8	–[a]
Best (r=1.3)	1.6	(1.2, 1.9)
Steepest (r=1.6)	0.49	(0.37,0.62)
Chrysotile[b]		
Best (r=1.3)	0.028	–[b]
Cautious model-max of:		
Linear (r=1)	0.5	–[b]
Steepest (r=1.6)	0.039	–[b]

[a]A linear model is not strictly statistically consistent with the observed data. The line with A_L=4.8 is the single best fit.
[b]Non-statistical uncertainties dominate choice of chrysotile models, 95% confidence intervals cannot be properly calculated. *See text for discussion.*

is a fall beyond 50 yr. Qualitatively it seems clear that the risk does not increase indefinitely, but there is insufficient evidence on very long follow up to fix the risk profile in this period. A rough and ready way of limiting the predicted risk at very long follow up periods is to truncate the predictions at some age. The Doll and Peto and HEI reports both truncated their predictions at age 80, and we will follow this convention. It is likely that this would still overstate the risk from exposure at ages below 20, and truncation of the predicted effect at 60 yr follow up might then be appropriate.

Lung cancer

The data in Table 7 and Fig. 9 suggest that the relation between lung cancer and cumulative exposure may be concave—i.e. that the excess lung cancer risk is proportional to a power greater than 1 of cumulative exposure. Statistically the range of powers consistent with all the amphibole data is from 1.1 to 2.1. Without the two extreme cohorts the range becomes 0.89–2.0 with a central estimate of 1.4. No previous analysis of the epidemiological data has suggested a concave relationship, though experimental data for a wide range of carcinogens (Hoel and Portier, 1995) suggest they may be quite common. Across the range of exposures in a single study, and given the uncertainties in individual exposure estimation, a moderate degree of non-linearity will be difficult to detect.

The reasonably arguable values for r fall in the interval 1 to 2: a degree on conservatism and some doubts about the two extreme cohorts lead us to prefer the lower end of this interval. We will take r=1 (a linear relationship) and r=1.6 to represent the flattest and steepest slopes for risk assessment, and the mid point of this range (r=1.3) as our best estimate assumption.

The estimates and 95% confidence limits for the constant term A_L in a model for lung cancer $P_L = A_L X^r$ with r=1 (linear) 1.3, and 1.6 based on amphibole data are shown in Table 10. As already discussed, the inconsistencies in the pure chrysotile data rule out a direct estimate of the exposure-response slope based on this data. The dominant uncertainties for chrysotile are the reasons for the observed differences in exposure-specific lung cancer risk, rather than the statistical uncertainties in estimating this risk level. This uncertainty is already reflected in the five-fold difference between our 'best' and 'cautious' estimates of R_L (0.1 and 0.5 respectively). In the absence of a better approach we will assume