and 1.3 respectively) but both confidence intervals are very wide. Even if the mines cohorts are excluded there is still very clear statistical inconsistency between the Carolina results and those from Connecticut and New Orleans (P=0.0013). The Carolina results are also out of line with the two other (mixed fibre) textile cohorts—Rochdale and Pennsylvania—whose 95% confidence intervals for R_L have no overlap with those for Carolina.

RISK ASSESSMENT AT MODERATE AND HIGHER CUMULATIVE EXPOSURES

Mesothelioma

The quantified risk for mesothelioma at the kinds of cumulative exposure levels recorded in the reviewed cohorts—say, from 10 f/ml.yr upwards—presents a reasonably coherent picture, with values of R_M, in round figures, of 0.5, 0.1 and 0.001 (at most 0.003) for crocidolite, amosite and chrysotile respectively (see Fig. 2).

Lung cancer

It is more difficult to come to a clear view of the quantified risks of lung cancer, because of the inconsistency of the results especially for the chrysotile cohorts (see Fig. 3). The amphibole estimates are reasonably consistent. In round figures the estimates fall in the range 2–10% per f/ml.yr. The mean for the crocidolite group is rather lower (4.2) than that for the amosite group (5.2), though their confidence limits overlap substantially. The mean risk for all amphibole cohorts is 4.8% per f/ml.yr (95%CI 3.9–5.8), but with some evidence of heterogeneity (P=0.027). If the SA amosite cohort data are set aside, the remaining data are reasonably consistent (P=0.072), and the mean estimate becomes 5.1 (95%CI 4.1–6.2). In round figures, a value of 5% per f/ml.yr would represent a reasonable risk estimate for both amphibole fibre types.

The pure chrysotile cohorts produce estimates of R_L spanning two orders of magnitude, from a value of 6.7 for the Carolina women to 0.03 for Balengero mine. How should this very wide range of R_L estimates be interpreted? As far as evidence from 'pure' exposure goes there are only two strongly informative cohorts: Quebec and Carolina. The differences between these two has been studied and discussed extensively but, finally, inconclusively. The hypothesis that mineral oil used to suppress dust in the Carolina plant may have contributed to the lung cancer excess has been addressed by an internal case-control analysis of this factor reported by Dement *et al.* (1994) and Dement (1991)). The most recent report (Dement *et al.*, 1994), shows that the odds ratios for different cumulative asbestos exposure categories are essentially unchanged by the addition of a variable representing subjects' typical level of exposure to mineral oil (slight, moderate, high). The coefficients for these categories in the joint model were not reported, but were as follows, expressed as odds ratios relative to 'slight' exposure:

Mineral oil exposure	Odds ratio	95% Confidence interval
Moderate	1.12	0.57–2.21
High	1.47	0.8–2.75

(Dement, personal communication)

Although these ORs are not statistically significant (and do not form a statistically significant trend), there is some suggestion that mineral oil may have a role in enhancing the asbestos effect, particularly since all the effect of exposure *duration* is absorbed in the asbestos measure (workers were assigned to oil exposure categories according to the assessed oil exposure level at which they had spent the longest *proportion* of their employment in the plant). Early results from this case control study showed a cross tabulation of cases and controls by asbestos exposure and mineral oil category (Dement, 1991), without formal modelling. Crude odds ratios on this data suggest that the asbestos response is progressively steeper with increasing mineral oil category. If mineral oil does have an enhancing effect, the anomalous increase in estimated exposure specific lung cancer risk for men in the Rochdale cohort first exposed after 1950 could be explained, since dust suppression using mineral oil was introduced from that date (Peto *et al.*, 1985). The regression slope estimate of R_L for the men first exposed after 1950 is 1.3 (95%CI 0.37–2.6), three times the value for men first exposed between 1930 and 1950.

The plausible suggestion that the longer fibre used in textile processes are responsible seems to be contradicted by the comparative analyses of lung fibre burdens in Quebec and Carolina cohorts reported by Sébastien *et al.* (1989). They found that the proportionate distribution of fibres by length was very similar in Quebec and Carolina lungs. Nevertheless, the notion that the longer fibres used in textile processes do represent a higher risk, is consistent with experimental evidence that longer fibres are more carcinogenic (Meldrum, 1996; Stanton *et al.*, 1981; Miller *et al.*, 1999). Green *et al.* (1997) have shown that the mean length and aspect ratio of chrysotile fibres in the lungs of Carolina workers are greater than in a local population control series; and than in the lungs of workers from the Albin cohort (Albin *et al.*, 1990a,b).

Both studies on the lung content of Carolina workers have found amphibole (crocidolite or amosite) fibres in an appreciable proportion of them, though at much lower levels than for chrysotile and its associated tremolite. Sébastien *et al.* (1989 report that amphibole fibres at concentrations >0.1 f/μg (fibres >5 microns long) were only found in the lungs of work-