Table 3. Additional data on pleural and peritoneal mesothelioma from cohorts with predominant exposure to crocidolite or amosite (but not both), and without reported quantified cumulative exposures

Cohort No.	Reference	Process	Fibre	Sex	Expected all cause morality	Pleural No.	Pleural % Excess mortality	Peritoneal No.	Peritoneal % Excess mortality
18	Jones *et al.* (1996)		o	f	400[a]	53	13	14	3.5
19	Acheson *et al.* (1982) (Leyland group)	Gas masks	o	f	185	3	1.6	2	1.1
20	McDonald and McDonald (1978)		oy	mf	41[a]	3	7.3	6	14.6
21	Hilt *et al.* (1981)	O	o	m	5[b]	1	20	1	20
22	Levin *et al.* (1998)	I	a	m	133.6	4	3	2	1.5
23	Parolari *et al.* (1987)	I	a	mf	115.1	2	1.7	1	0.87
24	Finkelstein (1989)	I	a	m	1.89			2	106
25	Acheson *et al.* (1984)	I	ay	m	298.8	4	1.3	1	0.33

[a]Estimated as observed deaths less asbestos related deaths.
[b]Estimated assuming 25% mortality from age 31 to 68.

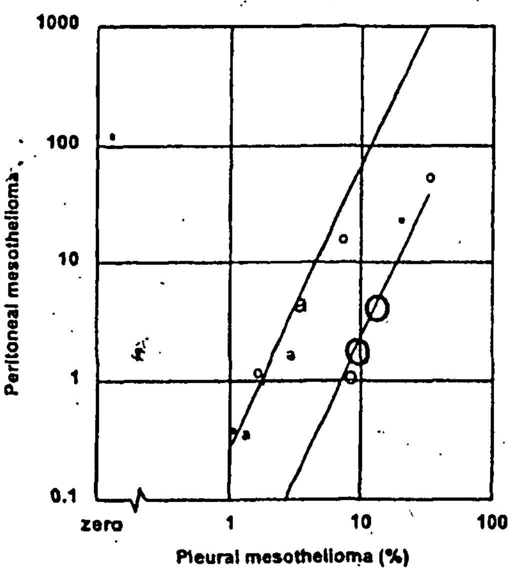

Fig. 5. Joint distribution of excess mortality from pleural and peritoneal mesothelioma, showing fibre type. (Note: Label size (area) roughly proportion to total mesothelioma numbers in each cohort).

residuals for these two cohorts, the overall residual deviance for the inclusive data (model 2) indicates a satisfactory fit (P=0.22). If the two outliers are removed, the separate fibre model fits the data almost exactly, and the slopes for the two fibres are very similar (model 3) and higher (around 3.2) than the [illegible] for the fit including them. In either case the single [illegible]e model is rejected in favour of separate fits to the two fibre types, with similar slopes. The peritoneal rate is proportional to at least the square—perhaps as much as the cube—of the pleural rate.

The form of the relationship is unusual and somewhat surprising, since both outcomes reflect the effect of the same carcinogenic insult to the same type of tissue. If true, it is presumably related to the dynamics controlling the distribution of asbestos fibres around the body. Note that this relationship does not depend on the cumulative exposure, and is therefore not subject to the uncertainties attached to exposure estimation. Whatever its physical/biological explanation, these observations imply that at least one of these outcomes has a non-linear relationship with exposure.

Pleural mesothelioma and cumulative exposure

To examine this question more closely, Fig. 6 shows a plot of excess mortality from pleural mesothelioma against cumulative exposure with cohorts represented by their fibre type code. Figure 7 shows a similar plot for peritoneal mesothelioma. The points for the pure amphibole cohorts show a clear pattern of alignment, with the slopes for pleural mesothelioma less than 1 and those for peritoneal mesothelioma greater than 1.

Table 5 summarises the results of Poisson regression fits to the relationship between percentage excess mortality from pleural cancer and cumulative exposure, and the observed data points and selected regression lines are shown in Fig. 6. The relationship is modelled as linear on a log scale for each variable, and therefore has the form $P_{pl} = A_{pl}X^r$ where P_{pl} is the percent excess mortality from pleural cancer, X is cumulative exposure and A_{pl} and r are regression parameters. The corresponding predicted number of pleural cancers for a given cohort is $A_{pl}X^rE_{Adj}/100$ (where E_{Adj} is expected all cause deaths adjusted to an age at exposure of 30). The parameters were estimated by minimising the residual deviance between the observed and predicted numbers of pleural cancer for each (pure fibre) cohort.

It is clear that a wide range of slopes (r) are statistically consistent with the data. With independent fits to each fibre type the slopes are 0.62, 1.2 and 0.72 for crocidolite, amosite and chrysotile respectively.