Table 8 *Estimated prevalence of possible asbestosis after uniform exposure to 2 f/cm³ and estimated concentration giving a prevalence of 1%*

Half-life of elimination of dust from lungs (yr)	*Estimated prevalence (%)-2f/cm³*			*Estimated concentration (f/cm³)-1% prevalence*		
	Length of exposure (yr)			*Length of exposure (yr)*		
	30	*40*	*50*	*30*	*40*	*50*
Logit model (no lag)						
0 = cumulative dose	2	4	7	1·5	1·1	0·9
5	4	7	11	1·0	0·7	0·5
10	5	9	15	0·8	0·5	0·4
25	6	12	19	0·6	0·4	0·3
∞ = no elimination	7	14	24	0·5	0·3	0·2
Logit model (5-year lag)						
0 = cumulative dose	4	6	9	1·0	0·7	0·5
5	6	10	14	0·4	0·3	0·2
10	8	12	17	0·3	0·2	0·1
25	8	14	21	0·2	0·1	0·1
∞ = no elimination	9	16	24	0·2	0·1	0·1
Alternative model (5-year lag)						
0	—	—	—	—	—	
5	7	10	13	0·3	0·2	0·1
10	8	12	17	0·3	0·2	0·1
25	8	14	21	0·2	0·1	0·1
∞ = no elimination	9	17	26	0·2	0·1	0·1

relationship with no lag period, the prevalence varies from 4% with cumulative dose to 14% with cumulative dose weighted by time since exposure (Table 8). Incorporating a five-year lag into the model changes this range to 6–16%, and using the alternative model makes very little difference. Hence, the method of accumulating exposures to dust over a period of time to produce a single measure of exposure is critical, mainly because of the unknown rate of dust elimination.

It is impossible with the data in the present study to discriminate in a statistical sense between any of the possibilities listed in Table 8, except that those based on cumulative dose may be relatively unsatisfactory.

One reason for the wide range of values in Table 8 is that only six men had average exposures of less than 2 f/cm³, but all of the figures in the Table are below this value. Another reason is that the maximum follow-up in the data is only 23 years, but longer exposures are considered. Therefore the figures in Table 8 are all predictions derived from extrapolations, and illustrate the difficulties of drawing any firm conclusions on the safety of present standards from data relating to the dustier conditions which existed until recently.

Discussion

By restricting the main analysis to men first employed after 1950, two important sources of bias have been reduced. First, estimated dust concentrations for earlier years were not used, although it was necessary to use the thermal precipitator counts between 1951 and 1960. Second, the selective effect of men leaving the factory for health reasons was largely eliminated.

There are errors in both the response and dose in the data analysed. Evidence of uncertainty in response is provided by the differences in the recording of crepitations by the factory medical officer and the Pneumoconiosis Medical Panel. Even when the medical findings are not in dispute it does not follow that exposure to asbestos is necessarily the cause. Crepitations may be caused by bronchitis, and pulmonary fibrosis may be detected on the chest radiograph in the absence of exposure to asbestos; for example, Weiss (1969) reported its presence in 0·6% of non-smokers and 2·2% of smokers. The prevalence of crepitations and radiological changes in the absence of asbestos exposure in the area where the factory is situated could be established only by examining a control group; this was not done in the present study. The dust concentrations used were obtained from static sampling sites and, therefore, took no account of the work-style of individual men. This may be the reason why the dose-response relationship fitted the data just as well with time since first exposure as with the various measures of cumulative dose. The effect of errors in response and dose, even if it were valid to regard them as purely random, would be to give lower dust levels associated with low prevalences of signs than would have been the case if it had been possible to collect the data without error. Thus, for example, the dust concentrations given in Table 8 would have to be increased before being applied to a situation in which the dust was measured by personal samplers.