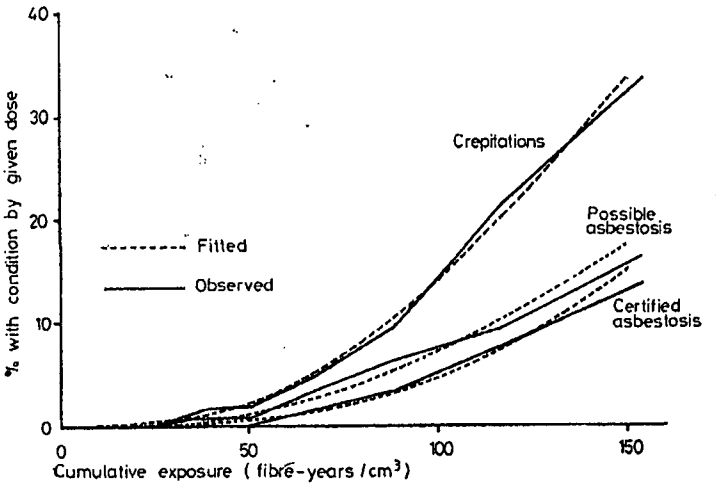

Fig. 4 *The relationships between the percentage developing the conditions, crepitations, possible asbestosis and certified asbestosis, and cumulative exposure to asbestos, for men first employed after 1950. The observed relationships were obtained by life table methods.*

and certified asbestosis the 1% prevalences correspond to 50 years' exposure to 0·19 and 0·37 f/cm³ respectively.

Thus, the choice of a suitable measure of exposure is highly critical, and it is not known what measure is most appropriate. If, over a long period, dust is eliminated from the lungs at a rate proportional to the amount present, and disease is caused by cumulative dose weighted by residence time of each contribution, then a family of exposure measures is produced. This family is indexed by the rate of exponential elimination, or half-life time, and, as extreme cases, contains the cumulative dose (half-time zero) and the cumulative dose weighted by time since exposure (half-time infinity). Further details are given in the Appendix.

This family of curves has been fitted to crepitations and possible asbestosis for a number of half-life times for men first employed after 1950. It was impossible to estimate precisely the half-life time giving the best fit to the data, and any value in excess of three years was considered to be adequate. Failure to estimate the half-life time reliably is not surprising, because the critical data would be observations on men who had left the factory many years previously.

Another way in which the exposure measure may be inappropriate is that a given dose is assumed to be effective immediately. The measure can be amended to accommodate a lag period for the development of an observed effect by assuming that, however severe the exposure, disease is not observed until some minimum time has elapsed since the start of exposure. There are insufficient data to estimate this lag period and the model was fitted incorporating a lag of five years.

Cumulative dose is correlated with years since first exposure; using this time as a measure of dose gives just as satisfactory a fit to the data as using cumulative dose and its generalisations; however, taking account of the dust level is more appropriate biologically.

ALTERNATIVE DOSE-RESPONSE MODEL

The dose-response model used has a reasonable biological basis, but other models may be appropriate. An alternative is that the incidence rate of diagnosis of an adverse effect is proportional to the amount of dust in the lungs (see Appendix). The analyses have been repeated with this model, which was satisfactory for the case with a five-year lag provided that the half-life was at least five years.

IMPLICATIONS FOR MODERN CONDITIONS

The disease pattern observed in this study has occurred as a result of higher dust levels than those which occur now (Table 1). It is interesting to predict the likely disease pattern with current standards and the concentrations estimated to result in a given percentage of men with disease. This has been done for continuous lengths of exposure of 30, 40 or 50 years. The percentage of men estimated to have reached the possible asbestosis category by the end of a period of exposure to 2f/cm³ has been calculated and, in addition, the concentration necessary to limit this percentage to 1% has been estimated. These calculations have been based on the parameter estimates from the post-1950 group for both models for a range of values of the half-life time of dust elimination (Table 8).

Varying the half-life time of dust elimination has a marked effect. For example, after 40 years' exposure to 2f/cm³, and using the logit dose-response