and of McDonald to suggest a dose-response relationship for gastrointestinal tumours after intestinal exposure. The authors came to the following tentative conclusions: "the Enterline data are possibly consistent with a threshold somewhere above 10 mppcf-yr, the McDonald data show no evidence of a threshold" and: "the dose-response observed in the occupationally exposed populations suggest that at the lower levels of exposure there was a slight or perhaps no risk of excess gastrointestinal cancer. Thus, there appears to be even less probability that the ingestion of asbestos from the higher (0.119 µg asbestos/l tap water) of the two potable asbestos-cement pipe systems would reach the risk level for gastrointestinal cancer of the occupationally exposed groups". However, this report did not take into account possible differences in fibre length and/or diameter.

Dohner et al (1975) observed 5 patients with known occupational asbestos exposure, each of whom had 2 definite or probable primary malignant tumours; however, with high exposure levels one may expect statistically that such combinations occur.

In the U.K. the BOHS published occupational hygiene standards for airborne chrysotile (1968) and amosite (1973) asbestos dust. They concluded that the risk of being affected with the earliest demonstrable effects on the lung due to asbestos will be less than 1 percent for an accumulated exposure of 100 fibres/ml yr, e.g. 2 fibres/ml for 50 years exposure. However, this limit protects 99% of selected (!) workers only against early asbestosis. According to the BOHS (1973) "the quantitative relationship between intensity of exposure and risk of cancer is less well defined. Evidence at present available indicates that if a standard is maintained such that the risk of asbestosis is small, the risk of cancer of the lung attributable to asbestos will be smaller still. The position in relation to mesotheliomata of pleura and peritoneum is still uncertain". It should be remembered that Enterline et al (1972) provided evidence that intermittent exposure to high concentrations might induce excess cancer mortality and not excess asbestosis mortality.

This review of data makes it clear that at this moment inadequate data exist on which to base a quantitative dose-response relationship for asbestos-related malignancies. On the other hand, it is also clear that there is often a clear dependency on dose:duration of exposure, type of jobs (intensity of exposure) but quantitation is not adequately possible.

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