environmental asbestos pollution in an asbestos textile factory. *Annals of Occupational Hygiene* 29, 305–335.
Piolatto, G., Negri, E., La Vecchia, C., Pira, E., Decarli, A. and Peto, J. (1990) An update of cancer mortality among chrysotile asbestos miners in Balangero, northern Italy. *British Journal of Industrial Medicine* 47, 810–814.
Rödelsperger, K., Woitowitz, H.-J., Brückel, B., Arhelger, R., Pohlabein, H. and Jöckel, K.-H. (1999) Dose response relationship between amphibole fiber lung burden and mesothelioma. *Cancer Detection and Prevention* 23, 183–193.
Rogers, A. (1990) Cancer mortality and exposure to crocidolite. *British Journal of Industrial Medicine* 47, 286.
Rogers, A., Leigh, J., Berry, G., Ferguson, D. A., Mulder, H. B. and Ackad, M. (1991) Relationship between lung asbestos fibre type and concentration and relative risk of mesothelioma. *Cancer* 67, 1912–1920.
Rossiter, C. E. and Coles, R. M. (1980) HM Dockyard, Devonport: 1947 mortality study. In *Biological Effects of Mineral Fibres. Scientific Publication no. 30*, ed. J. C. Wager, pp. 713–721. IARC, Lyon.
Sébastien, P., McDonald, J. C., McDonald, A. D., Case, B. and Harley, R. (1989) Respiratory cancer in chrysotile textile and mining industries: exposure inferences from lung analysis. *British Journal of Industrial Medicine* 46, 180–187.
Seidman, H. and Selikoff, I. J. (1990) Decline in death rates among asbestos insulation wokers 1967–1986 associated with diminuation of work exposure to asbestos. *Annals of New York Academy of Science* 609, 300–318.
Seidman, H., Selikoff, I. J. and Gelb, S. K. (1986) Mortality experience of amosite asbestos factory workers: dose–response relationships 5 to 40 years after onset of short-term work exposure. *American Journal of Industrial Medicine* 10, 479–514.
Sluis-Cremer, G. K. (1991) Asbestos disease at low exposures after long residence times. *Annals of New York Academy of Science* 643, 182–193.
Sluis-Cremer, G. K., Liddell, F. D. K., Logan, W. P. D. and Bezuidenhout, B. N. (1992) The mortality of amphibole miners in South Africa, 1946–80. *British Journal of Industrial Medicine* 49, 566–575.
Smith, A. H. and Wright, C. C. (1996) Chrysotile asbestos is the main cause of pleural mesothelioma. *American Journal of Industrial Medicine* 30, 252–266.
Stanton, M. F., Layard, M., Tegeris, A., Miller, E., May, M., Morgan, E. and Smith, A. (1981) Relation of particle dimension to carcinogenicity in amphibole asbestos and other fibrous materials. *Journal of the National Cancer Institute* 67, 965–975.
Stayner, L. T., Dankovic, D. A. and Lemen, R. A. (1996) Occupational exposure to chrysotile asbestos and cancer risk: a review of the amphibole hypothesis. *American Journal of Public Health* 86, 179–186.
Stayner, L., Smith, R., Bailer, J., Gilbert, S., Steenland, K., Dement, J., Brown, D. and Lemen, R. (1997) Exposure–response analysis of risk of respiratory disease associated with occupational exposure to chrysotile asbestos. *Occupational and Environmental Medicine* 54, 646–665.
Talcott, J. A., Thurber, W. A., Kantor, A. F., Gaensler, E. A., Danahy, J. F., Antman, K. A. and Li, F. P. (1989) Asbestos-associated diseases in a cohort of cigarette-filter workers. *New England Journal of Medicine* 321, 1220–1223.
Thomas, H. F., Benjamin, I. T., Elwood, P. C. and Sweetnam, P. M. (1982) Further follow-up study of workers from an asbestos cement factory. *British Journal of Industrial Medicine* 39, 273–276.
Vainio, H. and Boffetta, P. (1994) Mechanisms of the combined effect of asbestos and smoking in the aetiology of lung cancer. *Scandinavian Journal of Work Environment and Health* 20, 235–242.
Weill, H. (1994) Biological effects: asbestos cement manufacturing. *Annals of Occupational Hygiene* 38, 533–538.

Appendix A

EXTRACTED COHORT DATA

The details of extracted data is shown in Tables 12 and 13, with explanatory notes in Tables 14 and 15.

SUMMARISING MORTALITY AND EXPOSURE MEASURES - THE CHOICE OF FOLLOW UP PERIOD IN RELATION TO EXPOSURE PERIOD

LUNG CANCER

For most of the studies considered in this review, mortality was reported for the period 20 or more years from first exposure. The reason for this is that it is assumed that this represents a period in which the effect of exposure will be fully expressed. Deaths observed in the period immediately following first exposure will be unaffected by that exposure, and the effect of exposure will become progressively more apparent as follow up time increases. Comparisons of observed and expected deaths that include periods immediately after first exposure will introduce some downward bias to the assessed risk level.

Evidence on the levels of excess in different periods after exposure suggests that between 10 or 20 and about 40 yr from exposure the lung cancer r is reasonably stable. Beyond 40 or 45 yr follow there may be some decline in risk, but the extent to which this may have diluted recorded lung cancer risk in these cohorts seems limited (for example, there is very little difference between the US/Canada insulators lung cancer SMR calculated over all follow up from 20 yr and one restricted to the period between 20 and 40 yr). No adjustment for differences in maximal follow up between cohorts has therefore been applied.

The impact of follow up less than 10 yr being included in the reported results, and the related problem of choosing an average exposure appropriate to the observed mortality needs to be considered. If observed and expected mortality from observations less than 10 yr from first exposure are uninformative of the possible effects of the exposure the inclusion of such observations in the reported results for a cohort, will dilute any actually occurring effect. Provided there is a reasonable amount of informative follow up on every cohort member this dilution effect can probably be ignored, since the observed and expected deaths generated from the early (uninformative) follow up will be outweighed for all individuals by their later observations. But if a high proportion of a cohort generates mainly uninformative follow up, reported overall results may be seriously distorted. This can happen if recruitment to cohort continues to the end of follow up. Subjects starting within 10 yr of the end of follow up will contribute no informative mortality data.