We also conclude that particles retained in the upper lobe are slightly larger than those in the lower lobe, and that this is true for every type of mineral particle. This phenomenon may also be a smoking effect.

Last we conclude that there is a remarkable homogeneity of mean particle size both from mineral type to mineral type and from patient to patient, and there is a reasonable homogeneity of particle concentration from patient to patient. These observations may indicate that, provided dusts loads are not overwhelming, the lung is able to control both the size of retained particles and the concentration of retained particles within relatively narrow limits.

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REFERENCES

CHURG, A. and WIGGS, B. (1985) Mineral particles, mineral fibers, and lung cancer. *Environ Res* **37**, 364–372.

SCHLESINGER, R.B. and LIPPMANN, M. (1978) Selective particle deposition and bronchogenic carcinoma. *Environ Res* **15**, 424–431.

STENBACK, F., ROWLAND, J. and SELLAKUMAR, A. (1976) Carcinogenicity of benzo(a)pyrene and mineral dusts in the hamster lung. *Oncology* **33**, 29–34.

ASBESTOS FIBRES IN LUNG TISSUE FROM CHRYSOTILE PRODUCTION AND TEXTILE WORKERS. PRELIMINARY REPORT

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TWO COHORT studies of mortality made previously by our group (MCDONALD *et al.*, 1983; MCDONALD *et al.*, 1980) using similar methods showed that the risk of respiratory cancer appeared much higher in chrysotile textile workers (plant in Charleston) than in Québec chrysotile production workers (mine and mills at Thetford Mines and Asbestos). Most of the chrysotile processed in Charleston came from Thetford Mines. In both cohorts, cumulative exposure was estimated from each employee individually from work histories and all available measurements of past dust concentrations. Although the accumulated exposures in Charleston were reported to be one tenth of those in Québec, the Charleston workers experienced a 5 times higher risk of respiratory cancer.

Two explanatory hypotheses were considered: 1) exposure had not been correctly measured; 2) cocarcinogens were present in the Charleston textile plant. In support of

hypothesis 1 was the fact that cumulative exposure estimates were mainly based upon air measurements made in the past with the midget impinger and gave the concentration in millions of dust particles per cubic foot (mpcf). The use of oil sprays to reduce dust at the Charleston plant could support hypothesis 2.

In order to test hypothesis 1, other information on past exposure was obtained by measuring fibres retained in lung tissue from autopsied cases from both cohorts. Autopsied cases were traced through death certificates and pathologists were contacted in the USA and Québec. Specimens of lung tissue in formalin or paraffin block were obtained for 75 subjects from Charleston and 228 from Québec. Fibre content of the lung was measured using a combination of optical microscopy (OM) and analytical transmission electron microscopy (ATEM). We report here preliminary data for 70 subjects from Charleston and 37 from Thetford Mines.

Chrysotile and tremolite were the principal mineralogical types encountered by ATEM. Tremolite, which accounts for about only 1% of airborne asbestos in Thetford Mines, was found in the lung in higher concentrations than those of chrysotile. Length distributions of fibres in the lung were similar in the two locations, although fibres were slightly longer in the Charleston textile plant. In each location, a clear relationship was found between cumulative exposure and lung concentrations of tremolite and asbestos bodies. The pattern was less clear with chrysotile. Overall, in relation to cumulative exposure expressed in mpcfy, the numerical concentrations of chrysotile fibres in lung were also somewhat higher for Québec subjects.

These preliminary findings indicate that the employees in Québec and Charleston were exposed to the same type of asbestos materials but in higher concentrations, certainly of tremolite, in Québec. This suggests that some factor other than fibre exposure *per se* was responsible, at least in part, for the higher lung cancer SMR in the textile plant.

REFERENCES

McDONALD, A.D., FRY, J.S., WOLLEY, A.J. and McDONALD, J.C. (1983) Dust exposure and mortality in an American chrysotile textile plant. *Brit. J. Ind. Med.*, **40**, 361–367.

McDONALD, J.C., LIDDELL, F.D.K., GIBBS, G.W., EYSSEN, G.E. and McDONALD, A.D. (1980) Dust exposure and mortality in chrysotile mining, 1910–1975, *Brit. J. Ind. Med.*, **37**, 11–24.

FACTORS AFFECTING PREVALENCE OF MUCUS HYPERSECRETION AND AIRFLOW OBSTRUCTION IN THE COAL INDUSTRY OF NEW SOUTH WALES, AUSTRALIA

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THE RELATIVE importance of occupational factors in the aetiology of chronic mucus hypersecretion (CMH) and airflow obstruction (AO) in the coal industry is still