TABLE I.—*Number of Rats Exposed*

Length of exposure	Amosite	Anthophyllite	Crocidolite	Chrysotile (Canadian)	Chrysotile (Rhodesian)	Control
1 day	49	49	49	49	49	48
3 months	52	52	52	52	52	58
6 months	24	24	24	24	25	
12 months	25	28	26	23	27	48
24 months	21	19	20	24	20	

TABLE II.—*Mean Respirable Dust Concentration (mg/m³) and Cumulative Dose (mg/m³ hours)*

Length of exposure	Amosite		Anthophyllite		Crocidolite		Chrysotile (Canadian)		Chrysotile (Rhodesian)	
1 day	14·1	99	12·8	90	12·5	88	9·7	68	14·7	103
3 months	12·4	5000	13·5	5240	12·6	5030	12·1	4930	12·3	5070
6 months	11·2	8550	10·9	8540	10·7	8430	10·2	8240	10·7	8590
12 months	10·8	17000	11·4	17100	10·6	17000	10·7	17100	10·9	17100
24 months	10·6	33500	10·6	33700	10·3	33200	10·1	33200	10·1	33600

included in the 12-month groups and hence the number of rats in these groups is slightly higher, and in the 24-month groups slightly lower, than planned.

In Table II the mean respirable dust concentrations and the cumulative doses, the products of concentration and time, are shown. The one-day exposure was 7 hours for all dusts. For the other 4 time intervals of exposure, there were slight variations in the number of hours required to achieve approximately equal doses but the mean times were 402, 788, 1574 and 3237 hours respectively. The mean concentrations were usually higher in Experiment 1 than Experiment 2, and the 3-month group had an average dose of 60% of that of the 6-month group. Reasonable equality of dose between the dusts was achieved for all the lengths of exposure, except for the one-day which was too short to allow any adjustments.

Interpretation of histological findings

Classification of asbestosis.—The lesions seen in the lungs of rats exposed to all types of asbestos were similar to those described in guinea-pigs (Wagner, 1963, 1965). There were 2 main differences; firstly, asbestos bodies were never seen in the lung tissue of the rat although they are frequently seen in the pleural granulomata which follow the intra-pleural inoculation of amphibole fibres; secondly, there was a far greater production of granular pneumocytes (type II) alveolar epithelial cells in the rat.

The lesions consist initially of a deposition of asbestos fibres, alveolar macrophages and cell debris in the alveoli arising directly from the respiratory bronchioles. These deposits become organized firstly by being enmeshed in a thin reticulin network which coarsens with time and becomes replaced by collagen fibres. The alveolar epithelium reacts with replacement of the type I cells, and becomes completely lined by granular pneumocytes. In some of the alveoli the epithelium is shed into the lumina, in others there is a walling of the alveoli by these cells. This usually occurs at the bifurcations where groups of alveoli are closed off from the lumina of the respiratory bronchioles, giving the so-called pseudo-acinar appearance. In the guinea-pigs these small cystic spaces contained asbestos fibre and degenerating macrophages, but in the rats numerous granular pneumocytes were also present. The initial lesions were confined to occasional discrete individual respiratory bronchioles scattered throughout the lung substance. After further exposure, more and more respiratory bronchioles become involved and all the respiratory bronchioles arising from terminal bronchioles become thickened as the fibrous tissue network extends into the wall of the respiratory bronchiole, and this interstitial reaction spreads down into the peripheral elements of the primary unit, involving the alveolar ducts, atria and finally the air sacs and alveoli. With progression, the individual lesions tend to coalesce, leading to the development of a

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