Biologic monitoring of workers exposed to a hazardous dust must be directed to the detection of early unfavorable changes in the hope that removal from exposure will prevent the progression of disease. Since chest radiography and pulmonary function are most widely used to monitor the respiratory health of asbestos-exposed workers, it is appropriate to review the relative sensitivity of these two methods in detecting an adverse biologic response to dust exposure in this industry. At the top of Fig 6, selected simple spirometric measurements in average percent of the standardized value are plotted by exposure group. It can be seen that after a cumulative exposure of 100 mppcf-yr there is a gradual reduction in the mean values as dust exposure increases. At the lower half of the figure, the proportion of workers with irregular or rounded small opacities at the one or two category levels, is plotted for each exposure group. It is apparent that the major increase in workers at each level of radiographic change also begins to appear after a cumulative exposure of 100 mppcf-yr, which again reveals a dose-response relationship. One must conclude from these data that in considering groups of workers with varying levels of dust exposure, radiographic evidence of small opacities within the lung fields and pulmonary functional change are equally sensitive in detecting diffuse parenchymal abnormalities associated with exposure to these fibrogenic dusts. However, it must be remembered that even in the absence of x-ray film changes, individuals may show decline in pulmonary function unexplained by other factors and presumably related to dust exposure. The reverse is also true, ie, radiographic abnormalities may appear before changes in pulmonary function. It is suggested that both methods of biologic monitoring be used in order to detect the earliest abnormalities in the largest number of exposed workers.

In contrast to studies of mortality from malignant diseases in asbestos-exposed workers, there seems to be little interaction between cigarette smoking and the diffuse fibrogenic effect of dust exposure in this industry. As indicated previously, this is demonstrated by the equally good exposure-pulmonary function relationship in nonsmokers after allowance for personal smoking habits. This is not to say that cigarette smoking has not reduced pulmonary function in workers who smoke; that it has is shown by a lower position of pulmonary function measurements, while the additional rate of decline resulting from dust exposure is similar for the smoking groups.

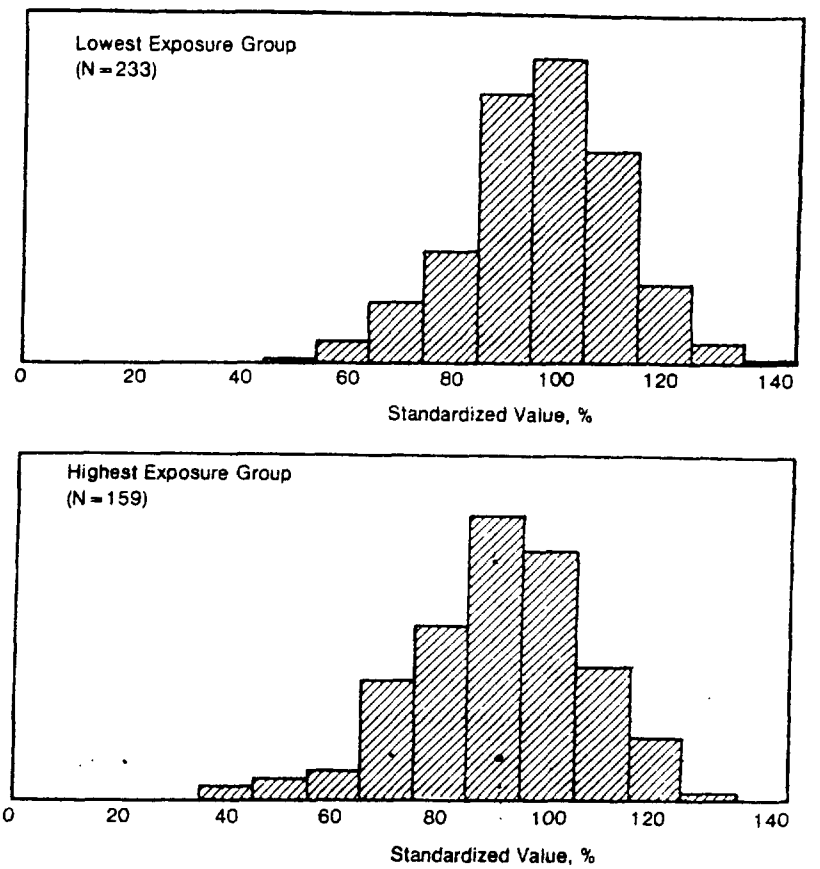

Fig 5.—FEV_1: Distribution of standardized values.

The diffuse parenchymal and possible airways damage associated with asbestos dust exposure seem to be relatively unaffected by host factors, as evidenced by the distribution of pulmonary function in the lowest and highest exposure groups. In the FEV_1 for example, we demonstrated primarily a shift in the group mean observed values (downward in the high-exposure group), rather than a significant "skewing" or "tailing" effect.

Available evidence suggests that crocidolite asbestos has a greater carcinogenic effect than chrysotile.[3] This suspicion has led previously to a different standard for these two fiber types in the United Kingdom. It has not been demonstrated previously that crocidolite has a more marked effect on the diffuse pulmonary parenchymal process. Although crocidolite exposures in the pipe-making area of one of the two plants studied were considered reasonably low, there appears to have been a significantly greater adverse effect on pulmonary function in workers exposed to crocidolite than on a matched group in

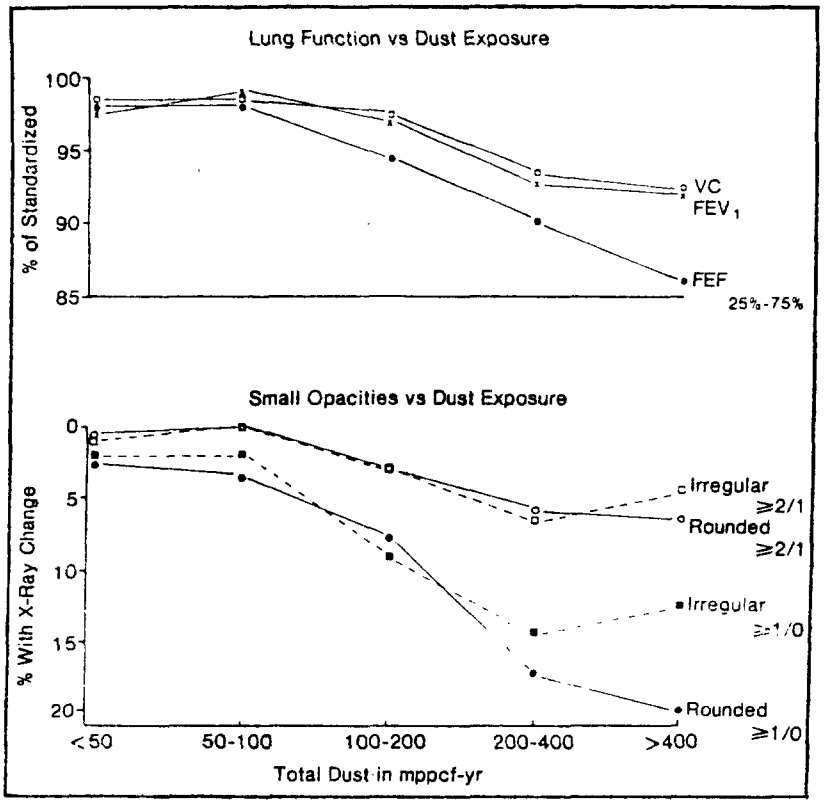

Fig 6 —Selected spirometric indexes of lung function and prevalence of small opacities on the chest x-ray films in relation to dust exposure.

the same plant for whom no crocidolite exposure was documented. The crocidolite group has smaller lung volumes, lower FEV_1, and reduced D_L. This subject will be separately considered in the future report.

Finally, threshold limit values must obviously take into consideration all demonstrated adverse health effects. This report considers only the diffuse fibrogenic effect of asbestos exposure in the asbestos cement products manufacturing industry. However, the data from this study demonstrate not only a dose-response relationship but suggest that a threshold dust level exists in this industry below which diffuse pulmonary fibrosis does not occur. While past dust levels were expressed in total particulate units, conversion to fiber levels is desirable for evaluation of asbestos exposure. A conservative calculation using data obtained previously from simultaneous sampling with the impinger and fiber counting method, leads to a conversion ratio of 2 fibers/ml for 1 mppcf.[17] The cumulative total dust exposure level of 100 mppcf-yr would therefore be equivalent to 200 fiber/yr or an exposure to 5 fibers/ml for a working lifetime of 40 years. While it is hoped that this level of exposure is "safe" in regard to the development of diffuse lung changes, any such analysis must be cautiously interpreted, since follow-up of exposed workers continuing well beyond the time of their active employment may increase the incidence of radiographic and functional abnormalities. These important additional data should result from the longitudinal investigation that involves a cohort of this population. A mortality study of all workers who have ever been employed in these two plants will determine if there is an excess risk of the development of neoplasms in workers employed in this industry.

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