fibrosis without the presence of "asbestos bodies" cannot be called asbestosis. Conversely, however, "asbestos bodies" in sputum or lung tissue without fibrosis is not asbestosis but simply an evidence of exposure. Phagocytosis by macrophages is a major cleansing mechanism of the lung; they engulf the offending mineral fiber producing the typical "asbestos body." No relationship has been established between the number, type, or size of "asbestos bodies" and the time of exposure, fiber size, concentration, or form of asbestos.

Individuals who show "asbestos bodies" on lung smears at autopsy do not seem to have an increased incidence of any specific pulmonary disease. "Asbestos bodies" may be found in lung smears and not in the correlating tissue. Cauna et al.[13] found that only 4% of lungs showed "asbestos bodies" on histological section, whereas smears of the same lungs showed "asbestos bodies" in 41%. Our findings are similar—lung smears in consecutive autopsies without known exposure to asbestos revealed an incidence of "asbestos bodies" in 45% while search of the matched lung tissue yielded only 3%. This may be a function of the amount of material available for study since the lung *smear* represents a much greater anatomic area than the lung tissue studied in histological section. Meurman found a considerably higher correlation of lung smears to lung sections but he used special techniques to demonstrate "asbestos bodies."

Our own interpretation is that while the differences in amount of material which is available for study may account for some of the disparity, there is evidence that mineral fibers are cleansed through the macrophage system and hence found in the alveoli and bronchial lumina but do not enter into the interstitial tissue. Only overwhelming exposure or perhaps some other factors which permit fibers to penetrate with formation of interstitial "asbestos bodies" lead to lung malignancy, pleuritis and mesothelioma. "Asbestos bodies" are not seen frequently in routine histologic study of lung pathology. This is not an oversight by the pathologist since sections of lung especially stained and studied for "asbestos bodies" do not reveal an increased incidence (only about 3%).

Our present view is that asbestos fibers are phagocytized in the airways and excreted in the sputum. In some instances the fibers penetrate the bronchial tree, giving rise to fibrosis, or into the pleura producing pleuritis. Co-factors, which are not completely understood at present, operating with the asbestos cause an increase in lung cancer susceptibility and in the appearance of mesothelioma. As the role of the "asbestos body" becomes clearer and more data becomes available on the distribution, type, fiber size, and concentration of airborne asbestos and the role of co-factors, the better we will be able to assess the potential environmental hazard from this ubiquitous mineral.

In summary, if one accepts the assumptions (1) that asbestos minerals increase the risk of lung cancer in occupational groups, (2) that they lead to an unusual risk of mesothelioma of the pleura and peritoneum in occupational groups and those living near asbestos plants, (3) that such malignancies usually result from exposures 30 to 50 years earlier, (4) that the "asbestos bodies" found in from 25 to 50% of lung smears from routine autopsies are probably due to asbestos in most cases, (5) that these "asbestos bodies" may result from recent exposures as well as those many years earlier, (6) that world production and use of asbestos has increased from 500,000 tons to 3,500,000 tons in 30 years, it is important to consider whether or not asbestos is a major threat to public health. One is not yet justified in such a conclusion, in view of the fact that much of the world tonnage goes into uses that do not lead to air contamination; asbestos is not actually indestructible, that the effects are dose-dependent, and that low doses probably lead to lower rates and longer latency.

Nevertheless, there is need for epidemiologic studies directed to groups with intermediate exposures and for evaluation of the beneficial effects of cessation of smoking. Another need is for experimental and industrial hygiene studies to determine the nature and degree of exposure. Biologic studies centered on the meaning of the "asbestos body" are crucial to understanding the clinicopathologic response. Strict control of industrial and neighborhood environments is essential, but it is premature to extrapolate from the effects of heavy exposures to minor and low level exposures.

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