measuring both coated and uncoated fibers, is needed to define a gradient of accumulated fibers for correlation with incidence of disease, on the one hand, and history of environmental exposure, on the other.

Although there appears no doubt that asbestos fibers are present in many human lungs, there are sources of airborne fibers other than asbestos. Some are probably derived from the burning of leaves and plant products, such as paper, wood and coal. Man-made (mostly vitreous) fibers have also been identified in the sediment isolated from human lungs. Talc, often used generously as a dusting powder, may contain a significant amount of tremolite asbestos fibers.

Information is sparse concerning possible increase of fibers in lungs with increasing use of asbestos and concerning the existence of significant differences between urban and rural populations. Selikoff and Hammond compared lung tissues obtained in 1934 and 1967 and found no significant increase in the proportion containing ferruginous bodies. This suggested that, despite increasing use of asbestos in New York City between 1934 and 1967, fibers of a type producing ferruginous bodies had not been increasing at a corresponding rate. Other commentators, however, have noted an increase over each decade in asbestos bodies in samples of lungs from persons who died in London in 1936, 1946, 1956 and 1966.

A review of the literature related to pleural calcification and asbestos exposure strongly suggests an association between pleural calcification and nonoccupational exposures to asbestos. See Nat'l. Acad., Ex. 19, p. 14.

Industrial experience has shown that prolonged inhalation of asbestos can increase the risk of neoplastic (tumor producing) disease. Examination of lung tissue has made it apparent that a much larger proportion of the general public has inhaled and retained asbestos fibers than had formerly been realized; in fact, most urban dwellers have some such fibers in their lungs. The basic issue is whether the general public, as well as persons working near occupational sources, living in the households of asbestos workers, living in the neighborhoods of asbestos plants, or having occasional random exposures, have a detectably increased risk of malignancy or other disease because of airborne asbestos. What information there is to answer these questions comes either from direct epidemiologic studies of groups with various levels of nonoccupational exposure or by extrapolation from the experience of industrial populations with direct or indirect asbestos exposures.