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The Subject of Continuous Vigilance

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Asbestos disease in the occupational setting has been documented since the beginning of this century. Modern usage of asbestos fiber has brought with it concomitant and widespread contamination of the environment. The need to control asbestos is required in the forms of legislation and surveillance, to prevent the spread of asbestos disease into the general population.

The Asbestos Problem

Because so many important areas of research require microscopical techniques, it is noteworthy that the asbestos problem has gained such widespread prominence. For example, the Environmental Protection Agency is now promulgating position documents regarding National Emission Standards for toxic substances. One of the first important materials selected for such regulation was asbestos *(1)*. This decision by the EPA was based on an intensive investigation by a panel of scientists, drawn from academic and industrial areas, who reviewed the many existing problems and concluded that asbestos was one of the more important substances which must now be controlled in our environment. In most scientific communities the importance of the asbestos problem is recognized, with most inquiry centering around the relative biological activities of the various fiber types and the magnitude of their effects in terms of carcinogenic potential.

The Nature of Asbestos

Asbestos is a generic term applied to a group of specific fibrous silicate minerals which have a number of useful properties in common. These fibers tend to be both electrical and thermal insulators, so flexible that they may be woven as fabrics. Different fiber varieties resist chemical degradation in alkalies or acids, have high tensile strengths, and possess a host of other properties which render them essential in literally thousands of modern applications *(2)*.

The mineral fibers which have been designated as asbestos are amosite, anthophyllite, chrysotile, crocidolite, and tremolite *(3)*. Although each asbestos mineral is commonly regarded as a single chemical and physical entity, the fibers themselves belong to more complex crystal chemical systems, so that they may range in both chemical and physical properties. Bulk chemistry, minor elements, trace elements, adsorbed hydrocarbons, surface properties, size distribution produced on comminution may vary considerably for each of the asbestos mineral types. However, their properties are such that they tend to fall within some well defined limits which are unique for that particular mineral species. They may, therefore, be uniquely identified by microscopical methods.

Asbestos Disease

Disease among workmen occupationally exposed to asbestos mineral fiber was reported in the literature in the third decade of this century *(4)*. The first report suggested that asbestos dust was a potent fibrogenic agent capable of producing fatal lung scarring similar in intensity and severity to that from silica exposure. The term asbestosis was applied to this particular pneumoconiosis. In 1935, the first report of a

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malignant neoplasm occurring in the scarred lung tissue of a person, exposed to dust in an asbestos textile factory, was made *(5)*. At this time, this occurrence of asbestosis and lung cancer was regarded as a chance association. It was not until almost 20 years later, in Great Britain, that the first epidemiological study clearly demonstrated the strong association between occupational exposure to asbestos dust and the development of lung cancer *(6)*. The lung cancer rate was almost ten times that observed in a matched nonexposed cohort. In 1960, Wagner and colleagues *(7)* observed a remarkably high prevalence of an extraordinarily rare tumor among workmen exposed to asbestos dust in the Northwest Cape Province of South Africa. Here, pleural mesotheliomas were reported in a number of workmen exposed to crocidolite fiber *(7)*. Several years later, gastrointestinal cancer, as well as lung cancer and mesothelioma, was epidemiologically established as an additional risk among insulation workers in the United States *(8)*. Also, another form of mesothelioma, of the peritoneum, was observed with great frequency among workmen exposed to crocidolite fibers in a factory in Great Britain *(9)*. It appeared by the middle 1960's that occupational exposure to asbestos dust produced fatal lung scarring (asbestosis) and significantly elevated risk of lung cancer, pleural and peritoneal mesothelioma, and cancers of the gastrointestinal tract.

Because of the high attributible cancer risk among workmen exposed occupationally to such fibers and the great number of uses of these materials, scientists around the world began to question whether or not the disease stigma might not extend beyond the boundaries of the occupational setting. In 1965, it was observed that members of the families of asbestos workmen and residents who lived within a half mile of an asbestos factory, none of whom were occupationally exposed to fiber, also incurred the risk of developing mesothelioma *(10)*. Although these latter observations were made in Great Britain, a similar study, involving a different fiber type, suggested the same findings here in the United States *(11)*. Asbestos disease was then traced into working populations who were only indirectly exposed to asbestos aerosols. These men were employed in shipyards *(12)*. Here, mesothelioma occurred in workmen whose activities were merely in the areas in which asbestos applications were being carried out.

To add to these worrisome observations that individuals need not be occupationally exposed to high concentrations of dust to contract asbestos disease, an epidemiological study demonstrated that asbestos fiber may interact with other substances to produce an elevated disease risk. In 1968, it was established that the synergism between asbestos fiber exposure and cigarette smoke inhalation produced a multiplicative effect in relation to lung cancer risk *(13)*. Work was begun, following these observations, to determine whether or not large portions of the general population might be exposed to asbestos fiber. It was established in 1971, on a series of random cases of people who died in New York City, that everyone had some chrysotile asbestos fiber in their lungs *(14)*. A recent study showed that asbestos is found in the air of 50 cities sampled in the United States *(15)*. Although the amount of fiber observed in human tissues from the general population was small, and the fiber in ambient air in large urban areas in the United States were many magnitudes less than that observed in a working environment, even these low fiber levels may have biological importance. Could asbestos fiber in the environment be another link in the continuing increased occurrence of lung cancer among people in the general population? Could ingestion of small amounts of asbestos fiber be associated with increased gastrointestinal cancer in the United States?

Asbestos in the Environment

Chrysotile asbestos has been identified as a common air pollutant in the ambient air in large urban areas across the United States *(15)*; work in our laboratory has even demonstrated the presence of chrysotile in the polar ice cap of Greenland (unpublished data); amosite fiber has been observed as a contaminant of potable water supplies derived from Lake Superior (brought about by mine waste discharge) *(16)*; chrysotile fiber has been added to clothing fabrics which have been used in the manufacture of women's coats *(17)*; chrysotile has been identified as a contaminant in pharmaceuticals,

apparently arising from the physical breakdown of asbestos filters used during processing *(18)*. We have observed the presence of asbestos fiber in disintegrated brake-drum dust, in children's papier mache mixes, and in consumer repair and construction materials and talcum products (unpublished data). Asbestos is now used in thousands of materials in modern society. Its presence may be regarded as ubiquitous.

Reason for Concern

It has been estimated that upwards of 75 to 85% of all human cancers occur through interaction with an environmental agent *(19)*. Environmental cancers have been recognized for almost 200 years *(20)*, with most observations made in working populations exposed to generally higher levels of some carcinogenic agent compared to the general population. These relationships have often been difficult to establish even in well defined working groups because of the long lapsed period between exposure to the responsible agent and the resultant clinical manifestation of the disease. Some of these lapsed periods have extended up to 40 years *(21)*. But other factors may influence the development of disease. For example, cigarette smoking increases lung cancer risk in asbestos workmen. Therefore, several factors can come into play with the resultant effects being multiplicative rather than additive. We are left with the caveat which states that "If an occupational group of individuals are exposed to high levels of some substance which increases cancer risk, it need not mean that the general population at large, exposed to much lower levels of the implicated substance, also need have risk. *However, it should give us some caution, and suggest that additional studies be made to prevent the occupational hazard from becoming a general environmental one" (21)*. Caution may also be translated to include surveillance.

There remain a number of important observations and disquieting possibilities: (a) the occupational standard which has been established for asbestos fiber exposure in workmen is a level designed to prevent lung scarring, not cancer; (b) occupational deaths due to neoplasms (lung cancer, pleural and peritoneal mesothelioma, and gastrointestinal cancer) are greater than from lung scarring (asbestosis); (c) cancers may occur in workmen with minimal evidence of lung scarring; (d) there may be a zero tolerance level for exposure to asbestos fiber in terms of carcinogenesis; (e) once asbestos fiber is inhaled, a large proportion tends to remain in the human body, and therefore, "exposure" continues throughout the life of the individual; (f) asbestos fiber may interact with other substances in the environment producing a multiplicative carcinogenic effect, and small amounts of fibers may interact with other substances, producing a greater cancer risk than for any singular agent involved by itself; (g) the number of sources of asbestos fiber in the environment is increasing and each source may add its fraction to the total body burden.

One of the many concerns which face us is the identification of such fiber sources and the magnitude of material that each source adds to the environment. The importance of defining these sources of exposure was eloquently summarized in the Lord Robens' report in 1970 *(22)*: "The proliferation of more subtle hazards, and particularly potential carcinogens, must also be the subject of continuous vigilance. Cancer producing chemicals share with asbestos and other fibrogenic dusts a latent period before the disease is manifest. Any failure at this time to bring these risks under control can only, therefore, be reaped as a bitter harvest, not by us, but the next generation."

Microscopists will play the key roles in this effort of continuous vigilance. This vigilance will prevent the unnecessary exposure of the people of our country to asbestos fiber. In a larger sense, microscopists will be preventing disease.

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