COMMITTEE REPORT

The Pulmonary Response to Fiberglass Dust

Report of the Committee on Environmental Health*
American College of Chest Physicians

Pneumoconiosis has recently been redefined by the International Labour Organization to be the accumulation of dust in the lungs and the tissue reaction to its presence. In terms of this definition, even the inhalation of soot by city dwellers leads to pneumoconiosis. The pulmonary response to the presence of fiberglass particles is similar to that following the inhalation of soot, namely, a macrophagic reaction.[1]

Animal Studies

Although Schepers[2] described bronchial epithelial hyperplasia and endobronchiolar as well as peribronchiolar lesions in rats exposed to fibrous glass dust, inasmuch as these lesions occur spontaneously in rats, their relation to the dust is highly dubious. In another article, Schepers and Delahant[3] state that glass fibers are not fibrogenic and provoke no microscopically detectable reaction in the lungs. A conglomerate dust composed of undetermined concentrations of fibrous glass plastic and calcium carbonate was tested on small laboratory animals, and as a result the dust was classified as biologically "inert."[4] It also has been noted elsewhere that the "pneumoconiotic lesions" undergo resolution with termination of the exposure and that no residual pulmonary fibrosis occurred.[4,5]

Inasmuch as the diameter of a fiber determines to a large extent its aerodynamic behavior and, therefore, the site of its deposition in the respiratory tract, major attention is paid to the fibers in the ambient air that are greater than 3.5μ in diameter. This is the size of fiber most likely to be deposited in the lung when inhaled. In an extensive exposure by inhalation of rats and hamsters to fiberglass dust with a mean diameter of 0.5μ and an average length of about 10μ at a high concentration (100 mg/cu m, approximately ten times the recommended threshold limit value) for two years, the pulmonary response was found to consist only of relatively small accumulations of macrophages without significant stromal changes. Allowed to live out their lives, the animals failed to develop pulmonary or pleural tumors.[6] In contrast to the atelectasis that tends to sequester many other nuisance-type (nonfibrogenic) dusts within alveoli, the air spaces containing fiberglass dust remained open, thereby facilitating its clearance. Another aspect of this study involved the intratracheal injection of glass fibers with a mean diameter of 1μ and a length that was 50μ or less. These injections resulted in endobronchial polypoid inflammatory reactions. The latter were attributed to the mechanical trauma of injection and could also have been caused by the entanglement and entrapment of the fibers at points of bronchial division. Since such lesions were not seen in animals that had inhaled fibrous dusts, the lesions were considered to be artifactual.

Kushner[7] reported the presence of fibrotic pulmonary changes in guinea pigs following the intratracheal injection of glass fibers predominantly longer than 10μ, but not after the intratracheal injection of glass fibers shorter than 10μ. He pointed to the artificial character of the method and emphasized the need for an inhalation-type of experiment for the acquisition of valid and relevant conclusions.

Other more recent animal studies involved the introduction of glass fibers as well as fibers composed of other materials into mesothelium-lined cavities of rats. These procedures caused the production of fibrosarcomatous tumors classified as mesotheliomas,[8-12] which, in turn, led to the conclusion that thin ($\leq$ 0.5μ diameter) fibers longer than 10μ[10] or 20μ[9] were cancerogenic to rats. The impli-

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cation was voiced that such fibers were potentially cancerogenic to man when inhaled;[12] however, this implication is based on experiments in which the fibers are administered by a highly artificial route and in doses that are far in excess of what man is ever likely to meet. Furthermore, there are reasons for believing that the cancerogenesis by fibers in rats is merely a modification of the well-documented solid-state carcinogenesis of rats. The latter has been extensively reviewed by Bischoff and Bryson[13] and probably has little relevance to man.

It should also be noted that subcutaneous fibrosarcomas are common spontaneous tumors in aging rats and that control rats in the previously mentioned experiments developed "mesotheliomas" following the intracavitary introduction of nonfibrous materials such as ultrafine noncrystalline silicon dioxide,[8] barium sulfate,[10] aluminum oxide,[10] and pulverized glass.[10] Fibrosarcomas have also been known to develop in rats at the injection sites of materials that are noncancerogenic to man. For instance, acetaldehyde, carboxymethyl cellulose, and even repeated injections of sodium chloride have produced fibrosarcomas in rats.[14] Nonetheless, the studies of Timbrell and colleagues[15] suggest that the physical characteristics and, in particular, the cross-sectional diameter of the inhaled fibers determine whether a particle is likely to be retained in the lungs. The investigations of Timbrell et al[15] provide a satisfactory explanation for the differences in carcinogenicity between asbestos amphiboles, as compared to chrysotile, and tend to suggest that the chemical composition of the inhaled fibers is of much less importance than was formerly believed. For these reasons, and although the weight of the evidence is against fiber glass being carcinogenic, the issue must remain *subjudice* until data are available from long-term exposures in man.

Studies on Humans

Under the title of "Fiber Glass Pneumoconiosis," Murphy[16] described bronchiectatic abscesses localized to the right lower lobe in a man who had developed cough, weight loss, and hemoptysis after heavy exposure to fiberglass dust. The pus of the bronchiectatic abscesses contained fibers resembling fiber glass. The diseased portion of the lung was resected, and the patient was well and working 3½ years later. A cause-and-effect relationship between the pulmonary disease and the inhalation of fiber glass was not demonstrated.

A paper published in 1969 was concerned with 691 reports by physicians of adverse symptoms caused by fiber glass.[17] Only 38 of these reports suggested the presence of respiratory-tract irritation. There were, in addition, 28 other similar cases of upper-respiratory-tract symptoms. Because of the nature of the reports, neither the character of the dust clouds nor the duration of the dust exposures was defined by the writers of this paper. It is, therefore, impossible to evaluate the pathogenic potential of fiber glass on the basis of this paper, except to note that under some undefined conditions of fiberglass exposure, some workers have experienced transitory upper-respiratory-tract irritation.

In a paper published in 1964, an interesting difference was noted between the effects of exposure to two widely different types of fibrous dusts, man-made vitreous fiber dust and asbestos dust.[18] Six workers exposed to glass-wool and rock-wool dust and eight workers exposed to asbestos dust were examined, and no detectable impairment in cardiopulmonary function was found in the former, whereas the latter had marked restriction in dynamic pulmonary function and a reduced diffusion capacity.

A series of four publications reported the results of roentgenologic or pulmonary-function studies on the same large population of fiber-glass workers by different investigators over different time periods.[19-22] No adverse roentgenologic or pulmonary functional results were demonstrable secondary to long-term fiberglass exposure at any time. Two investigations dealing with the health of British fiberglass workers also indicated that long-term occupational exposure to fiberglass dust results in no demonstrable health effect.[23,24] Epidemiologic studies in this country, one on 1,448 fiber-glass workers, resulted in the finding that there was no excess in mortality or increased risk of cancer from occupational exposure to fiberglass dust.[25,26]

Autopsy Studies of Human Lungs

Postmortem examinations were made of the lungs of 28 fiberglass workers and the lungs of 26 urban dwellers of both sexes.[27] The fiberglass workers had been exposed to fiber-glass dust for periods of 16 to 32 years, whereas the urban dwellers had presumably not been occupationally exposed to fiberglass dust. Both groups of people had died from causes usually encountered in hospitals. The total dust content and the mineral-fiber content of the lungs, as well as the average dimensions of the mineral fibers, were not significantly different in the two groups. The most important conclusion drawn from this study was that "long-term exposure to the dust of fiber glass used for insulation causes no demonstrable or microscopic pulmonary damage."[27]

In a continuing study of mineral fibers deposited in human lungs, the fiber content of the pulmonary tissue and of their satellite lymph nodes was deter-

mined on 16 long-term fiberglass workers, six people of Charleston, SC, and eight people from Pittsburgh.[28] The lungs of fiberglass workers had a higher concentration of optically counted fibers than the satellite nodes; whereas in the people from other regions, the concentration of such fibers in the lymph nodes was greater than in the lungs. No correlation was found between the fiber concentration in the lymph nodes and in the lung, nor was there any correlation between the fiber concentration of mineral dust in either the lung or the lymph nodes. Whereas about 6 percent of the fibers were identified as chrysotile, the other 94 percent remained unidentified. Because these unidentified fibers are transparent and resistant to sodium hypochlorite, as well as to perchloric acid, it is a safe assumption that they are silicates. It seems also a reasonable assumption that many of the fibers are derived from plants, since the ash of burning leaves or other vegetation-derived products, such as wood, paper, and coal, is known to contain such fibers.[29]

Because extrapolation of the experimental results in rats with fiber cancerogenesis has focused attention on airborne fibers of certain dimensions, the data from Dement's paper[30] are of interest. In four facilities manufacturing glass "wool" insulation, the number of fibers less than or equal to 10μ in diameter in the breathing zone of the workers ranged from 0.04/ml to 0.69/ml (40,000/cu m to 690,000/cu m). Of these fibers, 35 to 98 percent were less than or equal to 3.5μ in diameter, and 39 to 74 percent were between 5μ and 50μ in length. The fraction of fibers with diameters less than or equal to 1.0μ ranged from 2 to 46 percent.

Summary

Fiberglass inhalation seems to produce a minimal tissue response in the lungs, and the reaction is one of macrophagic mobilization and is characteristic of the pulmonary response to those nonfibrogenic dusts classified as nuisance dusts. In order to merit the designation of a nuisance dust, the pulmonary response must fulfill the following three requisites:[31(p5)] (1) The alveolar architecture must remain intact. (2) The stromal proliferation is minimal and consists mainly of reticulin. (3) The tissue reaction is potentially reversible. Inasmuch as the pulmonary reaction to the dusts of fiber glass fulfills all of these requirements, it should be classified as a nuisance dust.[31(p5)] There is no evidence to indicate that inhaling fiber glass is associated with either permanent respiratory impairment or carcinogenesis; however, the final verdict as far as the latter is concerned must await the findings of long-term mortality studies.

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Apropos of Age

The oldest known rocks are in Central Canada, Western Australia, the Ukraine, and on the Kola Peninsula in the Soviet Union. Their ages range between two and a half and three and a half billion years. None represents the original crust of the solidifying earth. Rather they are the roots of long-vanished volcanoes, lava flows that must have poured out on previously existing solid floors, or else ancient sediments collected in depressions on a solid surface. The earth must be appreciably older than the oldest of them. To determine the age of our planet we must appeal to astronomers and especially to cosmologists who are concerned with the origin of the universe. They report that many lines of evidence suggest an age for the earth of four to six billion years. The most widely accepted figure is about four and a half billion years, which is in accord with the geologic data. Our galaxy may be ten billion years old and the universe ten or twenty billion years or even more. Some stars visible to us today, on the other hand, are probably only a few million years old.

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