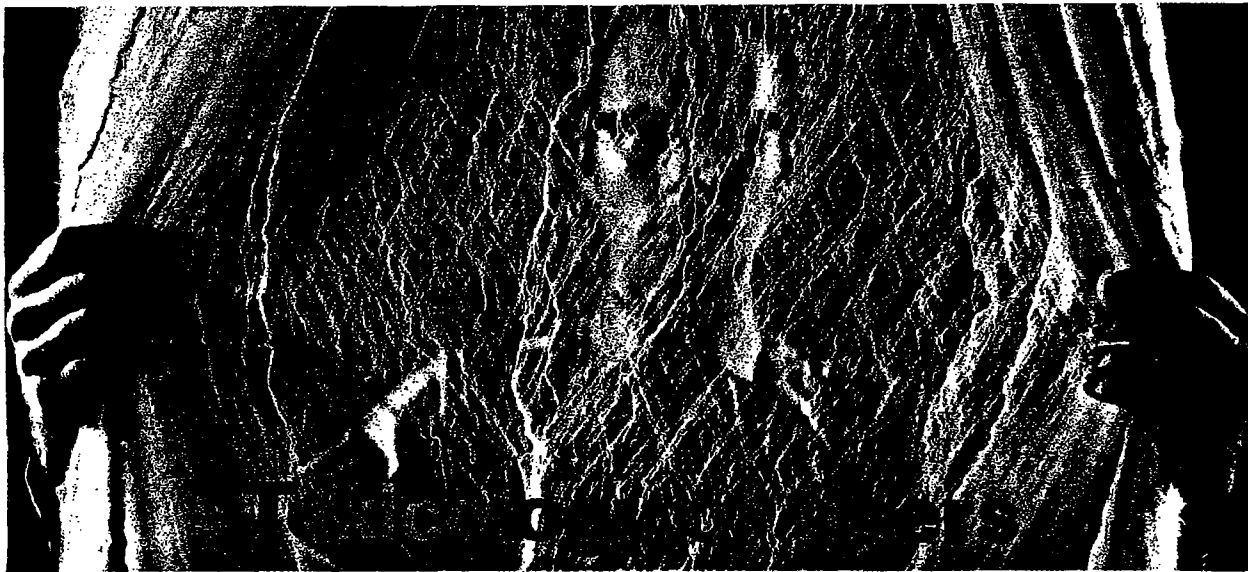


2
nyle



Can you imagine today's industry without the use of fibers? Without any fibers—be they organic or mineral, natural or man-made? Because industrial fibers are so ubiquitous and because industrial workers are exposed to these fibers during manufacture and routine use, we must examine whether fibers constitute any health hazard.

Let's first consider the two types of mineral fibers: natural materials such as asbestos and man-made ones such as fibrous glass, mineral wool, and ceramic aluminum silicate.

Mineral fibers have received great attention in recent years because of the serious diseases that have been associated with excessive exposures to asbestos dust. Four kinds of asbestos are in commercial use: chrysotile from Canada, which constitutes more than 95% of the asbestos used in this country; crocidolite and amosite from South Africa, which make up the remainder of the asbestos used; and anthophyllite, which is mined in Finland but not used in the United States.

All asbestos, when inhaled in excessive amounts, causes destruction of lung tissue and the development of scars that result in inadequate ventilation of the lungs, decreased oxygenation of the blood, pulmonary hypertension, and right heart failure. These lung changes comprise the disease asbestosis. If severe, it is a debilitating and often fatal disease.

All four varieties of asbestos are also associated with the development of lung cancer. However, epidemiologic studies have revealed that nearly all patients with asbestotic lung cancer have also been severely addicted to cigarette smoking for most of their lives (1).

A third major asbestos-related disease is mesothelioma. This rapidly fatal, but relatively rare cancer develops in the chest cavity or abdomen and occurs mostly among people exposed to South African asbestos. Chrysotile has a very low potential for causing mesotheliomas (2). In a study of workers exposed only to chrysotile, only three out of more than 2400 deaths that occurred over a span of 30 years were caused by mesothelioma.

Laboratory investigations have shown that short asbestos fibers (less than 5 μm in length) do not cause lung disease in animals (3). Moreover, the lungs of city dwellers, men and women alike, have been found to contain asbestos fibers without demonstrable disease attributable to them. This raises the as yet unanswered

question: At what level of pulmonary asbestos content does disease begin?

The Occupational Safety and Health Administration (OSHA) has ruled that in the workplace there shall be not more than two fibers longer than 5 $\mu\text{m}/\text{cm}^3$ of air. The ruling is restricted to fibers longer than 5 μm since one cannot count shorter fibers accurately with a light microscope.

Because of the health hazard associated with inhalation of asbestos dust, there has been an urgency to find mineral fibers that do not endanger health. These substitutes, all man-made, include not only fibrous glass, mineral wool, and ceramic aluminum silicate, but also fibers composed of aluminum oxide, carbon, silicon carbide, titanium phosphate, and potassium titanate.

Of all these man-made mineral fibers, only fibrous glass and mineral or rock wool have been produced over a long enough period (more than 40 years) to allow studies on chronic effects. Information on the health of workers exposed to its dust, bolstered by experimental data, allow us to conclude that fibrous glass dust does not produce disease. It is a nuisance-type dust, so classified by the American Conference of Governmental Industrial Hygienists (ACGIH) (4):

"Nuisance dusts have a long history of little adverse effect on lungs and do not produce significant organic disease or toxic effect when exposures are kept under reasonable control. The lung-tissue reaction caused by the inhalation of nuisance dusts has the following characteristics: 1) The architecture of the air spaces remains intact. 2) Collagen (scar tissue) is not formed to a significant extent. 3) The tissue reaction is potentially reversible."

Although it appears that at the present time, no known pulmonary disease can be attributed to the inhalation of fiberglass (5), the classification of fiberglass dust as a nuisance-type dust has recently been challenged. This challenge threatens to impose crippling constraints upon the utilization of all types of man-made mineral fibers that might serve as safe replacements for asbestos. The challenge is based on animal experiments in which fiberglass as well as other mineral fibers were injected into the abdomen of rats or implanted surgically into the chest cavity of these animals (6-8). Many of the rats so treated developed cancer in the region of the embedded mineral fibers. It was concluded from these experiments that

mineral fibers, regardless of their chemical composition, are capable of causing the development of cancer if their dimensions are within certain limits, namely, less than 0.5 μm in diameter and more than 10–20 μm in length.

Is it proper to extrapolate from these results in rats to man? Those who answer "No" to this question support their conclusion by the somewhat facetious, but nevertheless pertinent statement that no one has advocated the injection of fiberglass or other mineral fibers into the chest cavity or the abdomen of man. But there are other reasons for opposing this extrapolation from rat to man: Rats develop similar cancers when injected with many nonfibrous materials which are known to be incapable of causing cancer in man. According to the National Cancer Institute's Ad Hoc Committee on the Evaluation of Low Levels of Environmental Chemical Carcinogens, a substance, although carcinogenic to animals, should *not* be considered a potential cancer hazard to man if the carcinogenic effect clearly results from physical, rather than chemical induction or where the route of administration is grossly inappropriate in terms of conceivable human exposure (9).

It is therefore apparent that the challenge to the present classification of man-made mineral fibers as nuisance-type dusts rests upon inappropriate extrapolation of the results of rat carcinogenesis experiments to man. It is also apparent that until OSHA makes an official ruling with respect to this challenge, there will remain much indecision, not only in the production, fabrication, and use of man-made mineral fibers, but also in the research and development of promising new substitutes for asbestos.

Let's turn now to organic fibers, both natural and synthetic. Natural organic fibers include those of vegetable origin, such as cotton, jute, and hemp, and those derived from animals, such as wool, cashmere, and hair. Synthetic fibers may be cellulosic, e.g., rayon, cellulose acetate and triacetate, or noncellulosic, e.g., acrylic, polyester, and nylon.

The literature indicates that few respiratory difficulties are associated with synthetic fibers, although they may present special problems as discussed later. Natural fibers, on the other hand, and their vegetable or animal dusts can pose a threat to the respiratory system. The diseases attributable to exposure to these fibers and dusts often result from an immunological reaction to antigens consisting of bacteria, fungi, molds, and plant enzymes or to protein toxins in the fibers themselves. Examples include byssinosis from raw cotton, hemp, and flax and bagassosis from sugar cane trash. The symptom-complex varies from one substance to another. Byssinosis is characterized by shortness of breath on the first workday of the week and a decrease in ventilatory function.

Byssinosis, also called "brown lung disease," unlike "black lung disease," is *not* distinguished by abnormal pigmentation of the lung tissue. It is a disease characterized by disturbed lung function without recognizable microscopic changes in the lung tissue. Byssinosis is caused by the bract associated with the raw cotton and not by the cotton fibers themselves.

Other organic fibers may cause asthma or an acute pneumonitis.

Problems associated with synthetic cellulosic fibers and resins stem from chemicals used in the production process and from unreacted precursors or contaminants coating the fibers. For example, in the viscose process of artificial silk or rayon production, two toxic substances are present,



Paul Gross is director of pathology research at the Industrial Health Foundation. He served as distinguished research professor of pathology at the Medical University of South Carolina and as research professor of pathology of industrial diseases at the University of Pittsburgh. Dr. Gross is a graduate of Adelbert College School of Medicine, and the Graduate School of Western Reserve University. He has published more than 200 articles and served on numerous advisory boards.



Daniel C. Braun is president of the Industrial Health Foundation, Inc. and was a lecturer on industrial hygiene at the University of Pittsburgh School of Medicine. He is also a lecturer on occupational health at the University of Pittsburgh Graduate School of Public Health. Dr. Braun graduated as a chemical engineer from the Carnegie Institute of Technology night school and from the University of Pittsburgh School of Medicine. He has served as medical director of the Pittsburgh Coal Company and U.S.

Steel and he has published extensively on industrial hygiene and occupational medicine.

carbon disulfide and sulfuretted hydrogen. The first is a chemical essential to the process; the second is a by-product, troublesome and of no use. Carbon disulfide is a dangerous toxin, capable of causing changes in the gastrointestinal, cardiovascular, endocrine, and central nervous systems. Sulfuretted hydrogen causes severe conjunctivitis and disturbances of vision including color blindness. These ill effects are almost completely confined to the manufacture or processing of the organic fibers and do not occur in their use or application.

The great majority of the noncellulosic, synthetic fibers are inert and innocuous. When fibers in this class cause problems, it is likely to be due to some unreacted ingredient used in the synthesis of the resin or fiber.

We thus note that some dangers exist due to the presence of fibers and their precursors. It is essential that workers in industry be protected from known harmful exposures. Yet it is equally important that we don't prevent the use of harmless material through the inaccurate extrapolation of animal data.

References

- (1) Selikoff, I. J.; Hammond, E. C.; Churg, J. "Asbestos Exposure, Smoking and Neoplasia," *J. Am. Med. Assoc.* 1968, 204, 106.
- (2) McDonald, J. C., et al. "Mortality in the Chrysotile Asbestos Mines and Mills of Quebec," *Arch. Environ. Health* 1971, 22, 677.
- (3) Gross, P. "Is Short-Fibered Asbestos Dust a Biological Hazard?," *Arch. Environ. Health* 1974, 29, 115.
- (4) "TLVs® Threshold Limit Values for Chemical Substances in Workroom Air," Adopted by ACGIH for 1977, American Conference of Governmental Industrial Hygienists.
- (5) Morgan, W. K. C.; Seaton, A. "Occupational Lung Diseases," W. B. Saunders Co.: Philadelphia, 1975, p. 116.
- (6) Stanton, M. F.; Wrench, C. "Mechanism of Mesothelioma Induction with Asbestos and Fibrous Glass," *J. Natl. Cancer Inst.* 1972, 48, 797.
- (7) Pott, F.; Friedrichs, K. H. "Tumors in Rats after Intraperitoneal Injection of Fibrous Dust (German)," *Naturwissenschaften* 1972, 59, 318.
- (8) Wagner, J. C., et al. "Mesothelioma in Rats after Inoculation with Asbestos and Other Materials," *Brit. J. Cancer* 1973, 28, 173.
- (9) National Cancer Institute, Ad Hoc Committee on the Evaluation of Low Levels of Environmental Chemical Carcinogens: Report to the Surgeon General, USPHS, April 22, 1970.

Authors' address: Industrial Health Foundation, Inc., 5231, Centre Ave., Pittsburgh, Pa. 15232.