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EDITORIAL

Asbestos -- Still a Carcinogen

Asbestos is an important cause of human illness. Clinical and epidemiologic studies have established incontrovertibly that asbestos causes cancer of the lung, malignant mesothelioma of the pleura and peritoneum, cancer of the larynx, and certain gastrointestinal cancers. (1) Asbestos also causes asbestosis, a progressive fibrotic disease of the lungs. The risk of these diseases increases with cumulative exposure and also with the length of time since the first exposure. Asbestos has been declared a proven human carcinogen by the Environmental Protection Agency (EPA) and by the International Agency for Research on Cancer of the World Health Organization. (2,3) The total number of deaths in the United States that will eventually be caused by exposure to asbestos is estimated to exceed 200,000. (4)

Asbestos is a generic term applied to a group of minerals, all of them fibrous. There are four commercially important forms: chrysotile, crocidolite, amosite, and anthophyllite. Chrysotile is the most important. It accounts for more than 95 percent of current world production. Nearly all asbestos used in North America has been chrysotile from the province of Quebec, Canada. (2)

All forms of asbestos are carcinogenic. All have been shown in clinical, epidemiologic, and laboratory studies to be fully capable of causing lung cancer, mesothelioma, and the full range of asbestos-related diseases. (3) Although crocidolite appears to be two to four times more potent than chrysotile or amosite in its capacity to induce mesothelioma, all forms appear to be equally potent in their capacity to cause cancer of the lung. (5)

New use of asbestos has almost completely ended in the United States and in most other developed nations as the result of government bans and market pressures. Those forces were stimulated by the landmark epidemiologic studies of Selikoff and colleagues (6) and by the release of information on the carcinogenicity of asbestos that had previously been suppressed by the industry. (7) By contrast, extensive and aggressive marketing of asbestos by Canada and other exporting nations continues in the developing world, where sales remain strong. (8)

With the virtual cessation of high-dose occupational exposure to asbestos, medical and public health attention has turned to the risks of exposure at lower doses in the general environment. Particular concern focuses on the asbestos that remains as a legacy of past construction practices in many thousands of schools, homes, and commercial buildings. (2)

Mistakes were made initially in dealing with asbestos in buildings. Parents in a few communities caused great harm by tearing out asbestos from schools with little regard for proper safety procedures. With the passage in 1986 of the federal Asbestos Hazard Emergency Response Act (AHERA), a rational set of legally enforceable controls was put in

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place. The guiding principle of AHERA is that asbestos in a building is deemed to pose no hazard to health unless fibers become airborne and can be inhaled. (9) So long as asbestos remains in place and is protected from disturbance, it can safely be left alone.

AHERA requires all school authorities to conduct visual inspections to identify asbestos-containing materials; air sampling is inaccurate and is not required. The results of inspection and plans for dealing with any asbestos detected must be made public. Overall, AHERA has been a success. In the great majority of schools, it has been feasible to manage asbestos in place. Removal (also referred to as abatement) is required only when asbestos-containing materials are visibly deteriorating or when renovation is imminent. Yet debate continues about the risks of low-dose exposure to asbestos.

In a study reported in this issue of the Journal, (10) Camus et al. used two epidemiologic approaches to assess the risks of nonoccupational exposure to asbestos. Both analyses were undertaken in a population of women in townships in the province of Quebec that have long been the major sites of asbestos mining in North America. Camus et al. first compared mortality among women in these communities with that among women in 60 other areas of Quebec (after excluding cities and shipbuilding areas). They then compared the number of deaths due to lung cancer among women in the mining areas with the number predicted by a risk-assessment model developed by the EPA. (3)

Camus et al. found no excess mortality due to lung cancer among women in the mining communities. In addition, they found the number of excess deaths due to lung cancer among women in these areas to be smaller by at least a factor of 10 than the number predicted by the EPA model. Beyond lung cancer, Camus et al. observed that the rate of death due to "pleural cancer," presumably mesothelioma, was more than seven times the rate in the nonmining areas. They also found substantial excess mortality due to asbestosis.

How can these findings be explained? Why does there appear to be no excess mortality from lung cancer among these women? One possibility, noted by Camus et al., is that the dose-response relation between asbestos and lung cancer may be less steep at low doses than is assumed in the EPA model. Indeed, it has been suggested that there may exist a threshold level of exposure to asbestos below which no carcinogenicity is evident. (10) While interesting, this explanation is entirely speculative and not based on data.

Another possibility is that the past exposure of the Quebec women may have been overestimated; such overestimation would inflate their calculated risk. It seems most unlikely, though, that this factor could account for more than a small fraction of the difference observed in mortality due to lung cancer. A third possible explanation is based on the fact that the populations of workers on which the EPA model was based are known to have been exposed to the amphibole forms of asbestos; the women studied by Camus et al. were exposed only to chrysotile asbestos, however, and the model may therefore not apply to the study group. Although it is true that exposures differed, there is no evidence that chrysotile is less potent in causing lung cancer than other types of asbestos. (11,12)

The most plausible explanation of the low mortality from lung cancer observed in this study is that women in the mining areas were exposed to an asbestos aerosol in which many particles were too large to reach their lungs. Previous studies have established that the risk of cancer in the mining and milling industry is much lower than that in the industries that process and use asbestos, such as textile manufacture and insulation. (13) In mining and

milling, many fiber agglomerates and long curly fibers are suspended in the air. These large particles are easily seen and counted under the light microscope, but they tend not to reach the pulmonary alveoli as efficiently as small particles. Thus, air sampling based on light microscopy in the mining environment produces a spuriously high estimate of true alveolar exposure.

By contrast, in the asbestos-processing industries, large mineral bundles are broken up into shorter, thinner fibers. Many of those smaller fibers are invisible under the light microscope and can be seen only with an electron microscope. They are, however, readily inhaled and retained in the alveoli. It is this fundamental difference in actual pulmonary exposure -- at the same level of exposure as measured with a light microscope -- that accounts for the profound difference in the risk of lung cancer observed between asbestos-mining workers and those in the industries that process and use asbestos; these risks differ by a factor of 10 to 50. (13) The EPA was fully aware of this difference when its model was constructed, and accordingly, the data from studies of workers in asbestos mining and milling were excluded. (2)

Camus et al. (10) go beyond their data when they assert, without qualification, that the EPA's model overestimates the risk of lung cancer among persons with nonoccupational exposure to asbestos by at least a factor of 10. The EPA's current regulatory controls embody a level of caution commensurate with the hazard. The data of Camus et al. from their study of a population in rural Quebec with highly atypical exposure to asbestos would be an inappropriate basis for revamping regulatory standards for asbestos in buildings throughout the United States.

A final, take-home lesson from this report is that chrysotile asbestos is still indisputably a human carcinogen. The observation by Camus et al. of a more than sevenfold mortality rate (relative risk, 7.63) from pleural cancer in mining areas, as compared with nonmining areas, corroborates an enormous body of literature showing that Canadian chrysotile, like all forms of asbestos, is a potent carcinogen. (1,6) This finding is sufficient by itself to argue against any relaxation of public health controls on chrysotile asbestos. Moreover, it underscores the inaccuracy of recent efforts to portray chrysotile asbestos as safe. (14) Assertions that chrysotile can be used without risk in developing nations are contrary to fact and extremely dangerous. (15)

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