somesty of the authors needs to be explored. I believe he would consider the late Irving Selikoff to be a good friend. D'Agostino and Wilson (2) wrote:

Each of these questions [about asbestos] has elicited heated disagreements, often including personal attacks on motives and integrity. But as the best known authority [Selikoff] on asbestos has stated: "Arguments should be evaluated on their merits and not by reference to the interests of those who make them."

I agree with this, and to my knowledge none of the authors of the Science paper have, until this date, departed from this policy. If now any one of us should, after repeated similar aspersions, explode with hurt or outrage, I for one would fully understand. As for my own activities, something of them is in the public domain (3); they are neither hidden from editors nor the subject for any apology.

J. Bernard L. Gee
Yale University School of Medicine

REFERENCES

1. Mossman BT, Bignon J, Corn M, Seaton A, Gee JBL. Asbestos: scientific developments and implications for public policy. Science 247:294–301(1990).
2. D'Agostino R Jr, Wilson R. Asbestos: the hazard, the risk, and public policy. In: Phantom risk: scientific inference and the law (Foster K, Bernstein DE, Huber PW, eds). Cambridge, MA: MIT Press, 1993:183–210.
3. Gee JBLP. Occupational lung disease. In: Current pulmonology, vol 13. (Simmons D, ed). Chicago: Mosby Year Book, Inc, 1992: 221–255.

Response

In response to Mossman's comments, it is clear that one form of asbestos or other is somewhat more or less potent than other forms. But given the complexities of the many epidemiological studies and the variability of human response, it is impossible to state much more than that. These differences are not likely to have much clinical importance. The Health Effects Institute-Asbestos Research (HEI-AR) report (1,2) seems to agree. HEI-AR was an industry government consortium that organized a review of asbestos and the potential hazards from asbestos in buildings. I quote from section 6.2.2.5 (p. 6–34):

1. In cohorts of persons exposed occupationally to elevated concentrations of airborne asbestos fibers, the risks of lung cancer and mesothelioma have been observed to increase with extent (level and duration) of exposure.
2. The data do not suffice to define the exposure-risk relationships precisely but are consistent with conventional lung cancer increases in proportion to the extent of exposure to asbestos, and the increase in absolute risk of mesothelioma caused by each brief increment of exposure is proportional to the extent of the additional exposure to the 2nd or 3rd power of time thereafter . . .
3. Comparisons among the different cohorts provide evidence that the risk of pleural mesothelioma is appreciably higher with exposure to crocidolite than with exposure to chrysotile or amosite. Peritoneal mesothelioma has almost always been attributed to amosite or crocidolite exposure.
4. The absence of adequate exposure measurements for the cohorts studied to data severely limits the reliability of any quantitative risk assessments that can be made at this time, especially insofar as the risks of low level exposure to fibers of different sizes and types may be concerned.
5. Many of the groups of asbestos workers that have been studied epidemiologically were exposed to more than one type of asbestos, and the data on risks caused by each separate variety are inadequate and inconsistent. The panel therefore calculated average risks for mixed exposures. These are appropriate for the purpose of this report, as some buildings contain more than one type of asbestos.

(Item numbers 3 and 4 were excluded because they deal with smoking and lung cancer and mesothelioma.)

Thus the HEI-AR panel did not find large differences between the types of asbestos and found no evidence of a threshold.

The HEI-AR panel found that the data from in vivo and in vitro experimental studies "are insufficient to indicate whether there is a significant departure from linearity" (p. 6–75).

Ilgren and Brown (3) are cited as showing a threshold for asbestos: "Epidemiological evidence of the type discussed in this paper cannot establish a negative result, so final proof must rest with a greater understanding of the underlying biological mechanisms."

Kohyama and Suzuki (4) have found many chrysotile fibers in mesotheliomas and pleural plaques. Dement et al. (5) has recently updated his South Carolina cohort. About half are alive, the standardized mortality ratio for lung cancer is 2.24; for all cancer it is 1.46, and two cases of mesothelioma have been seen in this cohort exposed to pure chrysotile. The slope of the exposure-response relationship was one of the highest seen for asbestos exposed cohorts irrespective of fiber type or industry (5). These reports make it clear that chrysotile asbestos is a dangerous carcinogen and that no threshold has been demonstrated.

The question of the abatement of asbestos that is securely in place is not technical. At some future time it, like most man-made structures, will be disturbed. It can be removed under precautions now or it can be left so that some time in the future, with or without proper precautions, it will be disturbed.

In response to Sternberg's comments, he should have additionally pointed out that the decrease in breast cancer was not statistically significant and the decrease in uterine cancer was only one subgroup.

David P. Rall
Former Director, NIEHS

REFERENCES

1. HEI. Asbestos in public and commercial buildings: report of the asbestos literature review panel. Cambridge, MA: Health Effects Institute, 1991.
2. HEI. Asbestos in public and commercial buildings: supplementary analyses of selected data previously considered by the literature review panel. Cambridge, MA: Health Effects Institute, 1992.
3. Ilgren EB, Browne K. Asbestos-related mesothelioma: evidence for a threshold in animals and humans. Regul Toxicol Pharmacol 13: 116–132 (1991).
4. Kohyama N, Suzuki Y. Analysis of asbestos fibers in lung parenchyma, pleural plaques, and mesothelioma tissues of North American insulation workers. Ann NY Acad Sci 643:27–52(1991).
5. Dement JM, Brown DP. Cohort mortality in case control studies of white male chrysotile asbestos textile workers. J Occup Med and Toxicol 2:355–363(1993).