School of Medicine, 135 College St, 3rd Floor, New Haven, CT 06510-2483.

6. *Stayner and Colleagues Respond*

Langer and Nolan, and Mossman and Gee, express several criticisms of our recent review of the amphibole hypothesis.[1] Langer and Nolan suggested that we failed to present the amphibole hypothesis in a developmental perspective. Our objective was to put this hypothesis in a public health perspective.

The scope of the amphibole hypothesis has been confusing to many, scientists and laypeople alike. We thank Langer and Nolan for reminding us that the hypothesis was first proposed in regard to asbestosis and later extended to mesothelioma. Mossman and Gee[2] may have contributed to this confusion by suggesting that chrysotile may also be less pathogenic than crocidolite in the causation of lung cancer and fibrosis. Therefore, we welcome their statement that the thrust of the amphibole hypothesis is only for mesothelioma. This restriction sharply limits the public health relevance of the hypothesis, since most studies have found that asbestos produces more lung cancers than mesotheliomas.

Langer and Nolan cite several early South African studies as evidence for the hypothesis that crocidolite is more potent than chrysotile in the induction of mesothelioma. We recognized in our paper that "chrysotile *may be* less potent than ... some amphiboles with regards to ... mesothelioma [italics added]"[1(p18)] and cited the most recent report on South African miners.[3] However, the interpretation of these epidemiologic findings is severely hampered by the lack of information on fiber exposure concentrations and dimensions, so no firmer conclusion can be drawn.

Langer and Nolan cite lung burden studies as evidence that tremolite, rather than chrysotile, could be the agent in the induction of asbestosis and mesothelioma. We do not share their enthusiasm for the lung burden studies. Given that chrysotile has a lung half-life of a few months and that mesothelioma has a latency period on the order of 20 to 30 years, it is unlikely that the chrysotile fibers found at autopsy are a meaningful indicator of historical exposure to chrysotile. As an analogy, if we failed to find cigarette smoke in the lungs of a deceased ex-smoker, should we then conclude that cigarettes could not have caused the death?

Mossman and Gee complain that our review failed to cite conference reports "endorsing the amphibole hypothesis." However, the publications they cited generally involved issues of asbestos exposure in buildings and were not pertinent to occupational exposures to chrysotile, which was the subject of our paper. We did cite papers from one of the proceedings[4] that they referred to; in fact, the first reference in our paper, to an article by Pigg,[5] was from this workshop.

Mossman and Gee misquote us as stating that the experimental evidence for the increased pathogenicity of crocidolite is primarily derived from in vitro studies; in fact, we stated that it comes primarily from lung burden studies. They also state that we failed to recognize dozens of references that support the role of superoxide radicals and the increased pathogenicity of amphiboles relative to chrysotile. We note that the BéruBé et al. study[6] that they mentioned was published a month after our own paper. Although we are aware of the additional mechanistic studies referred to, we would argue that theories based on mechanistic arguments, however attractive, must give way to substantive empirical evidence. In this case, the epidemiologic and toxicologic evidence for the pathogenicity of chrysotile is overwhelming.

Finally, Mossman and Gee suggest that critical reviews and annotations should be written by scientists in the "mainstream of relevant panels and scientific meetings." We find this suggestion bizarre. Our own experience in this area is substantial. One of us (RA Lemen) has been active in this area for more than 25 years, has authored numerous scientific papers on asbestos (including a book[7]), was the principal drafter of the International Agency for Research on Cancer's monograph on asbestos, and has testified on asbestos issues to the US Congress and the US Department of Labor on numerous occasions. Another one of us (LT Stayner) has participated in several recent asbestos-related meetings, including a World Health Organization task force on this issue. Frankly, we had hoped that the fact that some of us do not have a long track record in this area would bring a fresh perspective to the debate. We suggest that critical reviews should be written by scientists who are willing to examine all of the relevant data critically, whether or not the data support their own beliefs. We have endeavored to do just that. □

Leslie T. Stayner, PhD
David A. Dankovic, PhD
Richard A. Lemen, PhD

Leslie T. Stayner and David A. Dankovic are with the National Institute for Occupational Safety and Health, Cincinnati, Ohio. Richard A. Lemen is the assistant surgeon general [retired], Atlanta, Ga.

Requests for reprints should be sent to Leslie T. Stayner, PhD, National Institute for Occupational Safety and Health, Robert A. Taft Laboratories, 4676 Columbia Pkwy—Mailstop C15, Cincinnati, OH 45226-1998.

References

1. Stayner LT, Dankovic DA, Lemen RA. Occupational exposure to chrysotile asbestos and cancer risk: a review of the amphibole hypothesis. *Am J Public Health.* 1996;86:179–186.
2. Mossman BT, Bignon J, Corn M, Seaton A, Gee JBL. Asbestos: scientific developments and implications for public policy. *Science.* 1990;247:294–301.
3. Sluis-Cremer GK, Liddell FDK, Logan WPD, Bezuidenhout BN. The mortality of amphibole miners in South Africa, 1946–80. *Br J Ind Med.* 1992;49:566–575.
4. Workshop on the Health Risks Associated with Chrysotile Asbestos, International Commission on Occupational Health. *Ann Occup Hyg.* 1994;38:397–646.
5. Pigg BJ. The uses of chrysotile. *Ann Occup Hyg.* 1994;38:453–458.
6. BéruBé KA, Quinlan TR, Moulton G, et al. Comparative proliferative and histopathologic changes in rat lungs after inhalation of chrysotile asbestos. *Toxicol Appl Pharmacol.* 1996;137:67–74.
7. Lemen RA, Dement JM. *Dust and Disease.* Park Forest South, Ill: Pathotox Publishers; 1979.

Integrating HIV Prevention, STD, and Family Planning Services

1. The Availability of HIV Services at Different Types of Clinics: A Survey

We concur with Zena Stein's observations and concerns regarding the separation of services for family planning, sexually transmitted disease, and acquired immunodeficiency syndrome (AIDS), as voiced in her editorial.[1] Recent preliminary animal data suggesting that Depo-Provera—the injectable hormonal contraceptive used widely in the United States and in the developing world—may increase vaginal permeability to HIV under-