butions in the work environments; there are important differences in the physical and chemical properties of fibers as well.[3,4] There are also considerable data on chrysotile's degradation in a biologic host and subsequent detoxification.[4] The existence of a threshold cannot be proved, but a change in the exposure–response slope at low doses must certainly take place.[5]

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To the Editor: Landrigan is wrong in concluding that "a more than sevenfold mortality rate . . . from pleural cancer in mining areas, as compared with nonmining areas, corroborates an enormous body of literature showing that Canadian chrysotile . . . is a potent carcinogen." This mortality rate (seven cases) is entirely explained by the few cases among women in the area who had occupational exposure to amphiboles in the manufacture of gas masks,[1] the repair of burlap bags that contained imported fibers,[2] and possibly, in one case, the tremolite brought home on miners' clothes.[2] Seven such women received workers' compensation in Quebec during the period of the study by Camus et al. Indeed, there is now a scientific consensus that chrysotile asbestos is not a cause of malignant mesothelioma, even among chrysotile-asbestos miners and millers.[1] Reasonable caution should continue to be used in exposing workers or bystanders to chrysotile, but if the levels of exposure currently recommended by the Occupational Safety and Health Administration and the National Institute for Occupational Safety and Health can be maintained, public health workers can concentrate their work on lung cancer where it belongs: on smoking.

With respect to women in the mining area, it has been established that the content of chrysotile and tremolite in the lungs is directly proportional to the number of years lived in the mining region and inversely proportional to the distance between the place of residence and the mining region.[2] In fact, these women were exposed to levels of chrysotile as high as 1 fiber per milliliter of air as recently as one month in 1984.[2]

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To the Editor: Landrigan misleads readers with the statement, "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." To prove his point, he cites only one report. The cohort cited by Landrigan is only one of many asbestos-exposed cohorts described in the medical literature. That cohort has an atypical pattern of lung cancer, as compared with other cohorts. Our review of mortality from gastrointestinal cancer in occupational-cohort studies[1] has shown that there is little evidence that asbestos causes gastrointestinal cancer. Similarly, there is a body of literature that indicates that there is no increased risk of laryngeal cancer.[2,3] Landrigan presents a biased view of the literature in an attempt to discredit the interesting and valuable contribution of Camus et al.

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The authors reply:

To the Editor: Some of the correspondents attribute to us inferences we did not make. We did not conclude that chrysotile asbestos is not carcinogenic, nor did we discuss the regulatory implications of our findings. We observed that the EPA's model greatly overestimated the risk of deaths from asbestos-induced lung cancer in our study population and that the model "may also overestimate the risk . . . in other populations with nonoccupational exposure." In the absence of any other published validation of the EPA's model in a population with nonoccupational exposure to asbestos, and given the fact that the model was based on a number of unverified and conservative assumptions, this statement seemed fairly restrained to us.

With regard to Demiroğlu's letter, the much higher rates of lung cancer and mesothelioma in the populations of some Turkish villages than in our study population are generally attributed to exposure to erionite (fibrous zeolite) rather than chrysotile asbestos.[1] Sokas misunderstood

the following aspects of our study: 2142 was the number of deaths, not the size of the population; there were deaths from asbestosis and mesothelioma in the control population; and both the control and the exposed populations consisted predominantly of small-town French-Canadian housewives with similar socioeconomic status and lifestyle. Furthermore, we presented some data on smoking habits and argued that confounding could not have accounted for the large discrepancy between the EPA model's prediction and the observed relative risk of lung cancer.

We agree with Costas et al. that the study had low statistical power to detect small risks; this was conveyed by the wide confidence intervals for our risk estimates. Our study would, however, have detected a relative risk as large as that predicted by the EPA's model. Churg's suggestion that the cases of pleural cancer and asbestosis were attributable to occupational rather than nonoccupational exposure is a possibility that will be explored in our ongoing research.

There are many possible explanations for the EPA model's overestimation of the risk of lung cancer in Quebec's chrysotile-mining towns. Our report offered six for consideration, and the correspondents' letters and Landrigan's editorial added others. At this time, all the explanations are speculative. We tend to disagree with the hypothesis that asbestos fibers in the air of mining towns were too large to be respirable, because, among other things, the main source of asbestos pollution in the mining towns was not the mines but the mills, which involved air-intensive processes designed to retain larger fibers and expel smaller ones.

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To the Editor: Langer ignores the pivotal point that the size distribution of airborne asbestos fibers in the mining areas of Quebec is very different from that encountered in any setting in the United States. This disparity provides the most plausible explanation for the difference in the risk of lung cancer in these two environments. In Quebec, exposure to asbestos involves crude, unprocessed asbestos, and many of the fibers are too large to be taken into the alveoli. In contrast, the asbestos in American industries, schools, and office buildings has been carded, spun, and woven. In these high-energy manufacturing processes, fiber bundles are broken, and millions of shorter, thinner, respirable fibers are created. It is probably this difference in the size distribution of fibers that accounts for the 10-fold to 50-fold difference in the risk of lung cancer reported between miners in Quebec and asbestos-mill workers in South Carolina.[1]

To be sure, there are respirable asbestos fibers in the Quebec mining townships. Although relatively few in number, they are responsible for the excess mortality from "pleural cancer" (probably mesothelioma) and asbestosis observed among women there. The apparent paradox of an excess risk of pleural cancer in the absence of lung cancer in these women is explained by a well-characterized difference in the dose–response relation between asbestos exposure and these two types of tumor.[2]

Morgan and Goodman are highly selective in their reading of the literature on the risk of gastrointestinal and laryngeal cancer in persons exposed to asbestos. The International Agency for Research on Cancer considers chrysotile asbestos a cause of gastrointestinal as well as laryngeal cancer.[3] Also, contrary to Morgan and Goodman's interpretation, the study they cite by Newhouse et al.[4] found dose-related excess mortality for both these cancers.

Case is inaccurate in his claim that chrysotile asbestos from Canada is not a cause of mesothelioma, and his assertion that there is a scientific consensus on this point is not true.[5] Also, he is disingenuous in his one-sided quest for factors other than chrysotile that would explain away the observed sevenfold excess of mesotheliomas among women in the chrysotile-asbestos–mining areas of Quebec. Experimental as well as epidemiologic studies have shown conclusively that Canadian chrysotile is fully capable of causing malignant mesothelioma,[5] and the International Agency for Research on Cancer acknowledges that chrysotile is a cause of mesothelioma.[3]

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Endometriosis in the Thoracic Aorta

To the Editor: Manifestations of endometriosis in thoracic organs are very rare.[1,2] We describe a case of endometriosis in the thoracic aorta.

After an uneventful pregnancy, a 28-year-old woman delivered a healthy girl by elective cesarean section. The procedure was complicated by severe, intractable hypertension. No cause was found for the severe hypertension, which was still present several weeks after delivery. A chest x-ray film and computed tomographic scan obtained postoperatively showed a pseudoaneurysm of the descending aorta with a maximal diameter of 9 cm in the area of a patch repair of a coarctation, which had been performed 16 years earlier. During her first pregnancy at the age of 24, which had also resulted in cesarean section, the patient had hypertension but no signs of gestosis. Arterial hypertension resolved without treatment after delivery. A chest x-ray film was normal at that time.

We operated on the patient three weeks after the second cesarean section. Intraoperatively, we found a rupture of the suture line of the distal part of the patch, with a false