Most importantly, Kazan-Allen also misrepresents the substance of the panel's valid and valuable report. As shown by the important extracts that Kazan-Allen quoted, the panel did not recommend a worldwide ban on asbestos. Indeed, the panel recommended research concerning the economic and practical feasibility of substitution for chrysotile asbestos as well as further research on the risks of cancer following exposure to relatively low levels of chrysotile.

Finally, whether chrysotile is suitable for "Korean, Indian and Japanese lungs" is surely not for Canadians to decide; but neither is it for the English or Americans to decide. Although scientific postulates have a universal character, public health policy must be rooted in social realities specific to each country. Even if they share a common understanding of the risks associated with a given factor, it is entirely legitimate for different countries to devise different policies in light of their different local circumstances.

Regarding David Muir's letter, surely the principle he espouses would apply not only to asbestos and pesticides but to all export products whose use might involve differing standards of health and safety for workers or consumers. Canada would have to set up monitoring systems in each country to which each such product was exported. For example, before exporting cars to a foreign country, we would need to monitor that country's tobacco and alcohol regulations and practices as well as all aspects of its national road safety policies (such as seat belt laws, speed limits, highway design and policing of driving safety). National and local policies and practices regarding fossil fuel combustion and its control would have to be monitored before oil was exported anywhere. There are many more examples of products (pharmaceuticals, nickel, plastics, various foods) that might not be used as safely abroad as we would hope. The sheer magnitude of the effort required to establish and maintain bilateral multi-product monitoring programs with each country to which Canada exports goods renders the proposal a non-starter, not to mention the potential for diplomatic conflict.

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3. Siemiatycki J. Should Canadian health care professionals support the call for a worldwide ban on asbestos? [editorial]. *CMAJ* 2001;164(4):495-7.

[Michel Camus responds:]

I proposed that the toxicity of chrysotile asbestos is much lower than that of other types of asbestos and that it may be close to that of substitutes.[1] Additionally, before a decision is made on whether or not to ban asbestos the technical efficiency of substitutes compared with chrysotile must be weighed for products that have intrinsic safety characteristics. Overall, like Richard Wilson and colleagues, I favour a comparative risk assessment approach. Although substitutes may prove to be better products with respect to human health, this has not yet been shown. Substitutes are associated with some risks, however small, and must therefore be considered critically. In fact, even a substitute 10 times less toxic than chrysotile should be regulated and controlled as tightly as chrysotile if we want to reduce risks. If we tolerate higher exposures to a substitute than to chrysotile, we could well offset the benefits of the lower toxicity of that substitute. Any ban or substitution policy should stipulate standards for substitutes likely to reduce risks.

The letters to *CMAJ* on banning chrysotile exhibit various viewpoints. I cannot address all of the important issues here, but I caution against putting moral judgements before fact-finding. No doubt all of the letter writers would agree that chrysotile is a carcinogen, but some of them seem to dismiss exposure–response relationships and the lower, possibly "acceptable" risks associated with lower exposures today. Any chrysotile-related risk may seem immoral to them, yet they are not critical about risks associated with chrysotile substitutes. How is it more moral to apply the precautionary principle only to chrysotile rather than to both chrysotile and its substitutes? Oversimplification and avoidance of evidence make it easier to make decisions but they result in hazardous policies.

David Muir and Laurie Kazan-Allen raise the issue of exporting hazardous materials and products. It seems desirable to caution the countries to which we export such materials and products against incorrect uses and careless exposures. Such cautions would apply to both asbestos and substitute products. However, it is not obvious how to do this without being paternalistic. This problem may be addressed by better labelling, cooperative education, training programs and improvements in the "traceability" of products. International laws might be enacted to hold producers and exporters responsible for the detrimental health effects of their products. I am not sure. Generally, more care should be taken to protect the most vulnerable sectors of any society against overexposure to toxic substances such as chrysotile and its substitutes.

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1. Camus M. A ban on asbestos must be based on a comparative risk assessment [editorial]. *CMAJ* 2001;164(4):491-4.

Methylmercury poisoning

Erica Weir's otherwise excellent public health article on the risks of methylmercury was flawed by misinformation on the clinical management of patients with methylmercury poisoning.[1] The information provided appears

to be based on the characteristics of elemental and inorganic mercury, not methylmercury.

The half-life of methylmercury in blood is relatively long (approximately 44 days) and the concentrations in newly formed hair are about 250 times higher than in blood.[2] Once concentrated in hair, the level of methylmercury remains unchanged; measurements in consecutive hair segments are thus useful indicators of past exposure (depending on the length of the hair). Measurements in hair correlate with the total body burden. Indeed, measurements in the mother's hair corresponding with the last month of pregnancy are proportional to the methylmercury levels in autopsy brain samples from infants who have died within a few weeks of birth. It is not useful to measure urine levels because methylmercury is not excreted by the kidneys. Likewise, chelation has no place in the treatment of acute or chronic methylmercury poisoning; there is no specific treatment.

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References
1. Weir E. Methylmercury exposure: fishing for answers. *CMAJ* 2001;165(2):205-6.
2. Clarkson TW. The toxicology of mercury. *Crit Rev Clin Lab Sci* 1997;34(3):369-403.

[The author responds:]

I thank John Ruedy for his careful reading of this public health column[1] and for bringing forth Clarkson's excellent article[2] on the physiology and toxicology of mercury. In writing the column, I relied on a clinical review article[3] that distinguished diagnostic and management practices for mercury poisoning primarily on the basis of acute versus chronic exposure, rather than by type of mercury compound. It suggested that, in principle, blood samples provide the best modality for assessing acute poisoning, whereas urine and hair samples reliably measure chronic exposure. It also suggested that chelation therapy should be considered in cases of acute poisoning, with the caveat that chelation therapy is most effective for elemental mercury and least efficacious for methylmercury, although it cites a reference[4] to substantiate the effectiveness of 3 chelating agents in ameliorating methylmercury-induced developmental toxicity.

It is important that physicians be familiar with these principles because it may not be clear in most cases of suspected mercury exposure which mercury compound (elemental, inorganic or organic) is responsible for the poisoning. Having said that, it is evident both by Ruedy's letter and by Clarkson's article that these principles fail to translate into practice in the case of methylmercury poisoning, which, as Ruedy rightly points out, was the focus of the column. Methylmercury avidly accumulates in growing scalp hair and is mostly eliminated as inorganic, not organic, mercury through the fecal route.[2]

Trust Mercury, the messenger of the gods, to shun principles, to assume a disguise and to slip surreptitiously through the back door.

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Associate Editor
CMAJ

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1. Weir E. Methylmercury exposure: fishing for answers. *CMAJ* 2000;165(2):205-6.
2. Clarkson TW. The toxicology of mercury. *Crit Rev Clin Lab Sci* 1997;34:369-403.
3. Ozuah P. Mercury poisoning. *Curr Probl Pediatr* 2000;30:91-9.
4. Domingo JL. Prevention by chelating agents of metal-induced developmental toxicity. *Reprod Toxicol* 1999;9:105-13.

Corrections

In a recent letter to the editor by Kevin Kain,[1] the first sentence of the second paragraph should read as follows: "Unfortunately, artemisinin-based drugs have not been shown to be better than parenteral quinine (the current drug of choice in Canada) in decreasing the mortality associated with severe malaria.[2,3]"

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1. Kain C. Ammunition against malaria [letter]. *CMAJ* 2001;165(5):529.
2. Tran TH, Day NP, Nguyen HP, Nguyen TH, Tran TH, Pham PL, et al. A controlled trial of artemether or quinine in Vietnamese adults with severe falciparum malaria. *N Engl J Med* 1996;335(2):76-83.
3. Van Hensbroek MB, Onyiorah E, Jaffar S, Schneider G, Palmer A, Frenkel J, et al. A trial of artemether or quinine in children with cerebral malaria. *N Engl J Med* 1996;335(2):69-75.

In a recent *CMAJ* public health article, advice on the clinical mangement of methylmercury poisoning in fact pertained to poisoning with elemental or inorganic mercury.[1] The error is addressed in letters in this issue.[2,3]

References
1. Weir E. Methylmercury exposure: fishing for answers. *CMAJ* 2000;165(2):205-6.
2. Ruedy J. Methylmercury poisoning [letter]. *CMAJ* 2001;165(9):1193-4.
3. Weir E. Methylmercury poisoning [letter]. *CMAJ* 2001;165(9):1194.