testing of soil, water, house dust, food or house paint.

I hope that future studies in Vancouver involve a more representative sample of children at risk, including those under 24 months of age, and are conducted at a more appropriate time of year. Such studies could produce very different results and better address the objectives set in the study by Jin and colleagues.

Timothy Johnstone, MB, BS, DPH, MFCM
Acting Regional Medical Health Officer
Capital Regional District
Victoria, BC

Before coming to Canada, I worked in London, England, where I treated children with clinical lead poisoning, a condition now rarely seen in Canada. However, my colleagues and I in Victoria do occasionally see an infant with an elevated blood lead level (higher than 0.72 μmol/L [15 μg/dL]). These infants usually live in old homes with lead-soldered water pipes or lead-based paint. Hence, I read the article by Jin and colleagues, the subsequent letter by Mr. Jack Rowe and associates and the response by Jin and colleagues (*Can Med Assoc J* 1995; 153: 395–397) with considerable interest.

I consider the critical remarks by Rowe and associates valid and justified. I share their difficulty in accepting with confidence the unusually low geometric mean blood lead level (0.26 μmol/L [5.4 μg/dL]) found in Vancouver children. This result is much lower than that reported in other Canadian cities and surprisingly similar to that of the children living in nonindustrialized countries such as Nepal and Papua New Guinea (see Table 3 of the article). In a similar report on a 1989 study conducted in Trail, BC, a relatively small city with a population of 10 000, 368 children were tested for lead.[1] By comparison, in this study, conducted in a city with a population greater than 600 000, only 172 children were tested.

The conclusion that "Vancouver did not have a lead-contamination problem that warranted a screening program or environmental investigation," although possibly correct, would be more credible had it not been based on such a small sample and on a population sampling method that appears biased toward children from higher-income families (there were only 24 children from the lowest income group in a frame of 5520 children). Because of the limited screening of participants, I doubt that many of these children were exposed to excessive contamination of soil, water or paint. However, pockets of high lead exposure probably existed in inner-city areas in Canadian cities in 1989 and likely exist today to a lesser degree. Therefore, the Vancouver blood lead measurements probably represent the general population of children but do not reflect groups at high risk of lead exposure. Because Jin and associates did not conduct concurrent environmental investigations, it is very likely that they missed a significant number of children at high risk.

As well, Jin and associates underrated the importance of the fact that 8.1% of the blood samples had lead concentrations exceeding 0.48 μmol/L (10 μg/dL), the present threshold for environmental intervention.[2] Such blood lead levels could have affected about 2000 children, by a conservative estimate.

When testing children in large cities for blood lead levels, it is socially responsible to direct efforts toward selective screening of people at high risk, whose health is most threatened, rather than conducting widespread random testing.[2]

Paul Gelpke, MD, FRCPC
Department of Pediatrics
Greater Victoria Hospital Society
Victoria, BC

References

1. Hertzman C, Ward H, Ames N et al: Childhood lead exposure in Trail revisited. *Can J Public Health* 1991; 82: 385–391
2. *Final Report by the Working Group on Blood Lead Intervention Levels and Strategies for the Federal/Provincial Committee on Environmental and Occupational Health*, Working Group on Blood Lead Intervention Levels and Strategies, Ottawa, 1994

[The authors respond:]

Concerning our survey's sample size, the sampling fraction, the season during which the survey was conducted, the age range studied and low income as a risk factor for elevated blood lead levels, we refer readers to our reply to a previous letter to the editor (*Can Med Assoc J* 1995; 53: 396–397).

Dr. Johnstone's assertion that we studied only 0.8% or 177 of 22 430 children up to 4 years of age is irrelevant and potentially misleading. As the title of our article clearly states, the survey's target age range was 24 to 36 months, which is approximately one fifth of the population from birth to 4 years of age. In any case, it is the sample size (177), not the sampling fraction (i.e., the percentage of the population sampled), that determines sampling precision and the power to detect differences between subgroups.

Dr. Gelpke's suggestion that our sample was too small because a study in Trail, BC,[1] involved a sample of 368 is incorrect. In the Trail study, the sample size was determined by the need for adequate numbers to conduct a case–control analysis in which children were grouped by blood lead levels and those in the approximate highest quartile (86) compared with those in the approximate lowest quartile (75).

Johnstone and Gelpke correctly state that we did no concurrent testing of soil, water, house dust, food or paint. We discussed this in our article, and we see no reason why this would invalidate our conclusions.

As for the representativeness of