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[One of the authors responds:]

Lead exposure in children is equivalent to alcohol exposure during pregnancy: there is no safe dose. No threshold value (below which lead has no apparent effect) has been identified.[1] Warren Bell and Kelly O'Grady are certainly right in saying that we have no recent data on lead levels in Canadian children. When we mentioned in our article that lead poisoning was rare in Canada,[2] we should have specified that this statement refers to cases requiring chelation therapy. Through discussions with medical toxicology colleagues working in Toronto and Montréal, I was able to identify only 7 cases in the past 10 years in which children required chelation for lead poisoning in those 2 cities: 3 children who were poisoned by unknown sources,[3] 2 children who had recently immigrated to Canada and were most likely poisoned in their countries of origin, 1 autistic child with pica and the case presented recently in *CMAJ*.[2] Others may have existed but not come to the attention of medical toxicologists, but in those cases the levels were probably above the intervention level of 0.48 mmol/L but below the recognized chelation threshold of 2.17 mmol/L.[4]

We chose to present the case of the 4-year-old boy in *CMAJ* to illustrate that lead poisoning can occur if a child with pica eats paint with lead levels below those set by Canadian law. Many physicians think that such paint is lead free, but, as Kathleen Cooper points out, this is not the case. Blood lead level should be determined for any child with pica, regardless of the age of the child's home, because eating a large quantity of chips of so-called "lead-free" paint can result in lead levels that require intervention with or without chelation.

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Possible causes of cognitive decline

Two things came to mind from reading the report by Jessica Simon and associates,[1] which thoroughly documents the cause of cognitive decline, seizure and stroke in a 52-year-old man as a rare genetic variation. My questions are inspired in part by the negative family history and the necessary supposition of de novo mutation. Was the patient tested for elevation of homocysteine? This marker is associated with geneti-

cally caused deficiencies of vitamins B6 and B12 and folic acid, the condition we associate with McCully, from his extensive studies on children with homocystinuria.[2,3] Multiple strokes because of premature, extensive vascular disease resembling arteriosclerosis were noted throughout the body tissues of those children. In addition, the early, progressive cognitive decline seen in the patient described by Simon and associates[1] closely resembles the cognitive decline we are seeing as a previously unrecognized side effect of statin therapy.[4,5] Was this patient receiving any statin drugs?

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[Two of the authors respond:]

Cerebral autosomal-dominant arteriopathy with subacute infarcts and leukoencephalopathy is a phenotypically heterogenous disease. Although the parents of the patient we described[1] were asymptomatic by history, one of them could well have harboured the same R182C mutation in the Notch3 gene as did the proband. We suggested a new mutation as a possibility only.

We did not test the patient for fasting serum homocysteine levels because the result would not have changed management. To date, it has not been shown that a reduction in homocysteine levels reduces clinical events in terms of stroke or any other vascular disease.[2] Nevertheless, we routinely counsel all stroke patients to eat sufficient fruits and green leafy vegetables to ensure that they receive enough B vitamins including pyridoxine, folic acid and cobalamin.

The patient was not initially treated with a statin agent. Since diagnosis, we have observed elevation of his total cholesterol and have begun treatment with a statin. In our opinion, it is more likely that this therapy will result in slowing, rather than acceleration, of the dementing process.[3]

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