Clinical Review
Environment and Health Series

Lead and children

Clinical management for family physicians

Alan R. Abelsohn MB ChB CCFP FCFP **Margaret Sanborn** MD CCFP FCFP

During a 1-year well-baby checkup, John's mother asks whether lead might be affecting her son. She has heard about lead in drinking water and children's toys. Questioning reveals that they live in a 1920s house and have been renovating for a year. The front porch, where the paint is in poor condition, has yet to be renovated. The house has original plumbing and the family drinks unfiltered municipal tap water. John is healthy and is growing and developing normally.

How should John's mother be counseled? Are any tests or follow-up visits required? We discuss possible sources of lead exposure for Canadian children, lead toxicity to children, and a prevention and management approach for family physicians.

Health effects of lead

Lead is a developmental neurotoxin, interfering with neurotransmission, cellular migration, and synaptic plasticity during central nervous system development.[1] Children are more vulnerable than adults to the effects of lead for several reasons: they are exposed more often by hand-mouth behaviour and spend time on dusty floors; they absorb lead more efficiently from the gastrointestinal tract (40% vs 5% to 15% for adults),[2] a capacity increased by concurrent iron or calcium deficiency[1]; and their blood-brain barrier and liver detoxification systems are biologically immature.[1]

Exposure to lead has been associated with many cognitive and motor deficits (eg, deficiencies in reading and math skills, fine and gross motor skills, and memory) and distractibility and other characteristics of attention deficit hyperactivity disorder.[3-6] Epidemiologic studies show associations between even low blood lead levels (BLLs) and lowered intelligence quotient (IQ).[5]

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Abstract

OBJECTIVE To provide family physicians with a practical, evidence-based approach to screening for and preventing children's exposure to lead.

SOURCES OF INFORMATION MEDLINE was searched using terms relevant to lead exposure and poisoning. We reviewed English-language articles published in 2003 to 2008. Most cited studies provide level 2 or 3 evidence.

MAIN MESSAGE Lead is a developmental neurotoxin. Children are most commonly exposed and they are most vulnerable. Lead exposure has been associated with many cognitive and motor deficits, as well as distractibility and other characteristics of attention deficit hyperactivity disorder. Although children's blood lead levels have declined considerably over the past 3 decades with removal of lead from gasoline and paint, children can still be exposed to lead from lead paint in older homes, toys, and other sources. Because post-exposure treatment cannot reverse the cognitive effects of lead exposure, preventing lead exposure is essential.

CONCLUSION Family physicians have an important role in screening for children at high risk of lead exposure, and in educating families to prevent the exposure of children to lead.

Résumé

OBJECTIF Suggérer au médecin de famille une approche pratique et fondée sur des preuves permettant de dépister et de prévenir l'exposition au plomb chez les enfants.

SOURCES DE L'INFORMATION On a consulté MEDLINE à l'aide de termes en rapport avec l'exposition et l'empoisonnement au plomb. On a révisé les articles de langue anglaise publiés entre 2003 et 2008. La plupart des études retenues offraient des preuves de niveaux 2 ou 3.

PRINCIPAL MESSAGE Le plomb est une neurotoxine qui agit sur le développement. Les enfants sont les plus souvent exposés et les plus vulnérables. On a attribué à l'exposition au plomb plusieurs déficits cognitifs et moteurs, de même qu'une tendance à la distraction et à d'autres caractéristiques du trouble déficitaire de l'attention/hyperactivité. Même si les niveaux sanguins de plomb ont considérablement diminué chez les enfants au cours des 3 dernières décennies en raison de la suppression du plomb dans l'essence et les peintures, les enfants risquent encore d'être exposés au plomb contenu dans la peinture des maisons et des jouets anciens et d'autres sources. Comme les effets cognitifs de l'exposition au plomb ne peuvent être renversés par un traitement ultérieur, il est essentiel de la prévenir.

CONCLUSION Le médecin de famille a un rôle important pour identifier les enfants à haut risque d'exposition au plomb et pour montrer aux parents comment prévenir l'exposition des enfants au plomb.

Higher BLLs (and higher lead body burdens measured by levels in bones and teeth) are associated with adolescent delinquent behaviour, poor educational outcomes, reduced adult brain volume, and increased criminal arrests.[5,7-11] Clinical symptoms such as anemia, abdominal pain, or neurologic deficits are rare and occur only at very high BLLs. Because lead crosses the placenta and accumulates in fetal tissue, fetuses are especially vulnerable to lead-related neurologic injury.[12]

Measuring BLLs is the best available way to determine lead exposure. Measuring BLLs accurately reflects recent exposures but poorly captures the cumulative body burden. There is no practical, clinically available measure of lead body burden.[13,14]

Blood lead levels are measured and reported differently in Canada and the United States (**Table 1**). The US Centers for Disease Control and Prevention (CDC) action level for blood lead is 10 µg/dL (0.48 µmol/L). This level has been recently questioned, because many recent studies demonstrate neurodevelopmental effects below 10 µg/dL, with no lower threshold for injury identified, suggesting that the decline rate (dose-response curve) in IQ scores might be greater at lower doses.[1,5,8,15-18]

Table 1. Measurement of blood lead levels in Canada and the United States

MEASUREMENT	CANADA	UNITED STATES
Units of measurement	µmol/L	µg/dL
Current action level	0.48 µmol/L	10 µg/dL
Proposed action level	0.24 µmol/L	5 µg/dL

Sources of information

In preparing this article, we searched MEDLINE using terms relevant to lead exposure and poisoning. We reviewed English-language articles published in 2003 to 2008. Most cited studies provide level 2 or 3 evidence.

Home sources of lead exposure

In the United States, 70% of children's lead exposure is from lead-based paint in the home.[19] In Canada, rental housing and paint surfaces are better maintained than in the United States, resulting in less lead exposure. Lead was eliminated from all paint in Canada in 1991, and Canadian regulations have limited concentrations in interior paint since 1978. Half of Canadian dwellings predate 1970 and would still have lead paint,[20] likely covered by lead-free paint. Lead is liberated in dust or chips from paint in poor repair, or during renovation activities including sanding or heat-stripping; children are exposed by ingesting lead-containing paint dust or chips. Children with developmental delays are at higher risk of lead ingestion and are more likely to present with lead poisoning after age 3.[1]

The remaining 30% of elevated BLLs in US children are from nonpaint sources,[19] including soil around homes (from exterior paint, nearby highways, or industries), toys, food, and herbal products. Recently, many children's toys, including brands such as Thomas and Friends Wooden Railway, Batman, and Dora the Explorer, have been withdrawn from the market because of high lead content, usually in the paint. Any recalled toys should be discarded. Food is a less common source, but lead-soldered pots or lead-containing glazed pottery and crystal glassware occasionally contaminate food.[21] Lead sources are listed in **Table 2**.[1,13]

Table 2. Sources of childhood lead poisoning

SOURCES	CAUSES AND EXAMPLES
Lead-based paint	• Houses built before 1978 • Paint in poor condition • Renovations
Lead in soil	• Exterior paint • Nearby industry • Nearby highways
Lead-containing toys	• See Health Canada's advisories, warnings, and recalls: **www.hc-sc.gc.ca/ahc-asc/media/advisories-avis/alpha-eng.php#lead** • See US Consumer Product Safety Commission recalled product search: **www.cpsc.gov/cgi-bin/haz.aspx** (Choose "Lead" and click on "Find")
Lead in drinking water	• Exterior pipes • Old plumbing
Lead in food	• Lead-soldered pots • Lead-containing glazed pottery • Crystal glassware • Imported spice mixtures[1]
Imported products	• Folk remedies • Cosmetics[1,13]
Other consumer products	• Crayons, jewelry, soft vinyl lunch boxes, candle wicks, PVC mini-blinds, and synthetic turf dust
Hobbies	• Stained glass • Pottery • Furniture refinishing

PVC—polyvinyl chloride.

Childhood lead exposure from drinking water has been in the news recently.[22,23] Drinking water in many older Canadian homes and schools with original plumbing contains lead, which leaches from lead service connection pipes outside the home and lead-soldered plumbing inside the home. Lead pipes were used in "well-built" homes until the 1920s, and 50%-lead solder was used until the late 1980s.[24] Although lead in drinking water can occasionally cause serious exposure,[23] its levels are generally low.[22,24] Tests for lead in water can be done through public health units or private water testing companies.[25]

Public health interventions, such as removing lead from gasoline, paint, plumbing supplies, and food and

drink cans, have successfully reduced children's BLLs. The percentage of US children with BLLs above 0.48 µmol/L (10 µg/dL) dropped from 77.8% to 1.6% between 1980 and 2002.[1] Less than 1% of adult Canadians have BLLs above 0.48 µmol/L (10 µg/dL).[26] No population-based data are available for Canadian children younger than age 6; children in only 3 communities having been studied. Children in Vancouver, BC, were found to have a mean BLL of 0.29 µmol/L in 1989; 8% of this sample had levels greater than 0.48 µmol/L.[27] Emissions from a large lead-zinc smelter in Trail, BC, contaminated local air and soil, raising childhood BLLs; a childhood lead-exposure prevention program, in which improved technology reduced smelter emissions, reduced children's mean BLLs to 0.28 µmol/L (5.9 µg/dL) by 1999.[28] A small study of 5-year-old Inuit children from Nunavik showed mean BLLs of 0.26 µmol/L (5.3 µg/dL).[3]

Immigrant or adopted children from countries with higher endemic lead exposures might have elevated BLLs and should be considered to be high risk. Information on high-risk countries can be found in the CDC tool kit, *CDC Lead Poisoning Prevention in Newly Arrived Refugee Children*.[29]

Screening

Because post-exposure treatment cannot reverse the cognitive effects of lead exposure, prevention is essential. The Rourke Baby Record recommends screening questions for lead exposure at 12 months and 2 years of age (**Box 1**).[30]

The CDC criteria for high-risk groups meriting screening include those identified by the first 3 Rourke Baby Record questions (although the cutoff date for housing is 1978) but add the following:

- children living near lead smelting or processing plants, battery recycling facilities, or other point sources of lead contamination;
- children having parents or other household members with lead-related occupations or lead-related hobbies (eg, pottery and furniture refinishing); and
- all refugee children aged 6 months to 6 years, within 3 months of arrival and again in 3 to 6 months.[29]

There are no Canadian screening guidelines, although in 1995 the Canadian Task Force on Preventive Health Care found fair evidence supporting targeted screening in high-risk children and infants (B recommendation), but insufficient evidence to recommend for or against universal screening (C recommendation).[31]

Screening questions have some limitations. All 4 questions in the Rourke version target the 70% of children whose lead exposure comes from household paint. The US version was validated in a suburban population, and the questions might not be generalizable to other groups or regions.[32]

Box 1. Screening for lead exposure with the Rourke Baby Record

Lead screening is recommended for children who ...

- in the past 6 months lived in a house or apartment built before 1950,
- live in a house with recent or ongoing renovations or peeling or chipped paint,
- have a sibling, housemate, or playmate with previous history of lead poisoning, or
- have been seen eating paint chips.

Data from Rourke et al.[30]

Office approach to prevention

During preconception counseling, antenatal appointments, or well-baby checkups, family physicians can educate parents about avoiding lead-exposure risks. A patient handout on protecting children from lead is available from **CFPlus.***

Preconception detection. Preconception detection of elevated maternal BLLs is vital to primary prevention. Women in occupations or who have hobbies with high lead exposure are at risk.[1] Studies correlating elevated cord BLLs with impaired child neurodevelopment provide direct evidence that preventing lead exposure during pregnancy is important.[5,12,33] A modest 11% reduction in maternal BLLs was achieved in a randomized controlled trial of maternal supplementation with 1200 mg of calcium throughout pregnancy; if more than 75% of prescribed calcium doses were taken, there was a 24% reduction in BLLs.[34] The same dose of calcium during lactation reduced maternal BLLs by 15% to 20%[35] and increased the decline in breast milk lead by 5% to 10%.[36]

Preventing lead paint exposure. Screening infants and toddlers 9 to 18 months of age is encouraged, using screening questions and measuring BLLs when indicated. However, more benefit is obtained from primary prevention questioning and education of prospective parents about their housing, state of repair of paint, and renovation activities *before* their children's birth.

Drinking water. In homes, schools, and day-care centres with older plumbing, letting water run for 5 minutes or more until it is cold before drinking eliminates standing water that has been in contact with the lead in pipes, reducing ingested lead.[22]

High-risk children. Children with developmental delays such as autism spectrum disorder and pervasive

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*A patient handout on protecting children from lead exposure is available at **www.cfp.ca**. Go to the full text of the article online, then click on **CFPlus** in the menu at the top right of the page.

developmental delay constitute a high-risk group whose persistent hand-mouth behaviour carries risk of lead poisoning beyond the usual 18- to 30-month peak.[1,37] Accordingly, family physician inquiry and screening of lead levels in older children with these diagnoses might be useful.

Case discussion and management

In our case, the child met 2 risk factors for higher lead-exposure risk: he lived in an old house with peeling paint on the porch, and renovations were in progress.

> John's BLL was measured; the result was 0.576 µmol/L (12 µg/dL). This is above the previously recognized "action" level of 0.48 µmol/L (10 µg/dL), and more recent evidence suggests there is no safe BLL.

What should be done?

John's BLL should be retested immediately to confirm the results, and complete blood count and ferritin levels should be tested to rule out iron deficiency.[14]

A careful exposure history[38] should be taken to rule out nonpaint lead exposures. In this case, the only source of lead exposure was high-lead paint, applied before 1978, liberated by interior renovations and in deteriorating condition on the porch. Hand-mouth activity, with ingestion of lead in dust, was the exposure route for this child.

To prevent continuing exposure, John must immediately be removed from the home until the home has been cleaned up or the situation has abated.[1] The Canada Mortgage and Housing Corporation provides careful advice on work-site cleanup and managing deteriorating paint.[24] Public health inspectors can perform home inspections and dust-wipe testing to confirm dust as a source of lead exposure.

If the child is iron deficient, iron supplementation (4 to 6 mg/kg daily) can be neuroprotective by reducing gastrointestinal lead absorption. Calcium and zinc supplementation might also be indicated.[1]

Chelation to decrease BLLs by promoting urinary lead excretion is not indicated for BLLs below 2.16 µmol/L (45 µg/dL)[1,39] and should only be done by pediatricians experienced in such treatment. Blood lead levels naturally decline over time, but the half-life is long and variable.[14]

Neuropsychological evaluation and follow-up is suggested for children with levels exceeding 0.96 µmol/L (20 µg/dL).[1]

Conclusion

There is a long history of progressive reductions of the BLLs in children requiring action, as research has extended the levels associated with poor developmental outcomes ever lower. New research showing considerable neurodevelopmental effects at BLLs well below 0.48 µmol/L (10 µg/dL), the current "level of concern,"[14] and the importance of preventing exposure have led to debate about further reducing the level of concern to 0.24 µmol/L (5 µg/dL) or even 0.10 µmol/L (2 µg/dL).[5,40-42] A new action level of 0.24 µmol/L (5 µg/dL) has been proposed in the United States. It was estimated this change would reclassify 7.3% of children aged 1 to 5 and 2.8% aged 6 to 11 with BLLs of 0.24 to 0.48 µmol/L (5.0 to 9.9 µg/dL) to a group requiring evaluation and treatment,[42] creating new health care and housing remediation expenses. However, there is a powerful economic argument for lowering lead action levels: lower IQs associated with lead exposure are strong indicators of future lost earning power and antisocial behaviour.[11,43]

Because the interventions that reduce high BLLs have not been proven effective in preventing

EDITOR'S KEY POINTS

- Lead is a developmental neurotoxin, interfering with neurotransmission, cellular migration, and synaptic plasticity during central nervous system development. Epidemiologic studies show associations between even low blood lead levels and lowered intelligence quotient.
- Interventions that reduce high blood lead levels have not been proven effective for preventing neurocognitive deficits; as a result, screening is considered too little too late. Preventing exposure through office-based and public education about the hazards of lead exposure is optimal.
- During preconception counseling, antenatal appointments, or well-baby checkups, family physicians can educate parents about avoiding lead-exposure risks.

POINTS DE REPÈRE DU RÉDACTEUR

- Le plomb est une neurotoxine qui agit sur le développement en interférant avec la neurotransmission, la migration cellulaire et la plasticité synaptique au cours du développement du système nerveux central. Les études épidémiologiques indiquent une association entre un faible quotient intellectuel et la présence de plomb dans le sang, même à des niveaux faibles.
- On n'a pas démontré que les interventions pour réduire les niveaux élevés de plomb dans le sang sont efficaces pour prévenir les déficits cognitifs; en conséquence, on juge que le dépistage est « trop peu trop tard ». La meilleure façon de prévenir l'exposition au plomb consiste à renseigner la population sur ses dangers, tant au bureau du médecin que dans les services publics.
- Lors des consultations antérieures à la conception, des visites prénatales ou du suivi du nouveau-né, le médecin de famille peut montrer aux parents comment éviter les risques d'exposition au plomb.

neurocognitive deficits, screening is considered too little too late. Preventing exposure through office-based and public education about the hazards of lead exposure is optimal.

Dr Abelsohn is a family physician in Toronto, Ont, and an Assistant Professor in the Department of Family and Community Medicine and the Dalla Lana School of Public Health at the University of Toronto. **Dr Sanborn** is a family physician at the Chesley Medical Clinic in Chesley, Ont, and an Assistant Clinical Professor in the Department of Family Medicine at McMaster University.

Contributors
Drs Abelsohn and **Sanborn** contributed to the literature search and preparation of the article.

Competing interests
None declared

Correspondence
Dr Alan Abelsohn, 1735 Bathurst St, Toronto, ON M5P 3K4; telephone 416 483-8111; e-mail **alan.abelsohn@utoronto.ca**

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