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LEAD INTOXICATION IN A CHILD RELATED TO THE INGESTION OF PLAYGROUND PAINT CHIPS — QUEBEC

Case Description

A 5-year-old boy from the Montreal area undergoing assessment for autistic behaviour was tested for blood lead level following his mother's comment that he often mouthed and swallowed unusual items, including chips of dried paint. While his blood lead level was 0.89 µmol/L (18 µg/dL) on 1 June, 1994, a repeat sample on 7 July showed a much higher level of 2.00 µmol/L (41.5 µg/dL). Public health authorities in both Canada[1] and the United States[2] have recommended 0.48 µmol/L (10 µg/dL) as an intervention level for pediatric blood lead. The child's mother and 7-year-old sister, also tested on 7 July, showed normal blood lead levels (0.13 µmol/L, and 0.14 µmol/L, respectively).

The child was hospitalized 12 July. On admission he had no abdominal pain, vomiting, constipation, or signs of acute lead intoxication. Hemoglobin was 130 g/L and free erythrocyte porphyrin (FEP)[3] was 0.67 µmol/L, both within normal limits. Abdominal x-rays showed paint chips in the intestines. He was chelated with calcium disodium edetate ($CaNa_2EDTA$) over 5 days; following this treatment, his blood lead level had fallen to 0.75 µmol/L.

Attending physicians asked staff at the Montreal Regional Public Health Program to assess potential sources of the child's exposure to lead. Interviews with his mother revealed that the boy had attended day care until April 1994. After that date he was being cared for full time at home, where the daily program included a 30 to 60-minute play period at nearby parks. His mother related that he would occasionally eat chips of paint, which he stripped or bit from the metal playground structures.

Inspection of the family's apartment revealed no obvious sources of lead: walls were of recent construction and paint was in good repair; there were no old painted toys and no folk remedies or lead-based ceramic pottery were present. Paint was peeling on outdoor metal railings but there was no evidence of any teeth marks.

At the two parks, metal playground structures were covered in a variety of colours of paint, much of it flaking.

Paint chips sampled in early August were analyzed by the *(Québec) Institut de Recherche en Santé et Sécurité au Travail* and by the *Centre de Toxicologie du Québec* with the following results:

Lead concentration (dry weight) in paint chip samples from the home and nearby parks of a lead-intoxicated child

Location	Colour	% Lead
Apartment, Metal balcony	Gray	0.06
Apartment, Living room wall	White	0.0068
Park 1, Swing set	Red	6.5
Park 1, Swing set	Blue	4.3
Park 1, Swing set post	White	2.1
Fence between the parks	Green	10.0
Park 2, Horse swings	White	5.4
Park 2, Horse swings	Red	0.011
Park 2, Horse swings	Blue	0.030

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Following this investigation, municipal authorities are in the process of removing existing deteriorated paint in playground areas; the new paint being applied is lead free.

Discussion

All of the evidence strongly suggests that metal play structures were the source of this child's intoxication. The potential of lead-based paint as a source of intoxication in small children is well known; most such cases are, however, related to indoor exposures[4,5], an issue which has received considerable attention in Canada[6].

Since 1975, the *Hazardous Products Act* has prohibited the use of paint containing greater than 0.5% lead (dry weight) for indoor residential surfaces, furniture, and toys. While regulations prohibit the use of lead-based paint in consumer products designed for children's use[7], playground equipment is not considered a consumer product in Canada. In the absence of a specific warning, municipalities and other proprietors of playgrounds have and may continue to apply lead-based paint. This presents a particular hazard to young children who bite and mouth foreign objects; risk of intoxication increases where painted surfaces are chipped or flaked.

As for remedial procedures, applying low-lead paint to existing painted surfaces would be inadequate to control exposure, as chipping tends to include all layers. Removal of old paint by heating or sanding risks contaminating nearby playground soil. Careful solvent-based stripping is recommended; rags used should be treated as hazardous waste. Any new paint applied should contain less than 0.5% lead. Replacement of metal structures with plastic ones would also eliminate this source of childhood lead exposure.

Recommendations

1. Avoid using lead-containing paint on all structures to which children have easy and frequent access.
2. Remove flaking lead-containing paint in such a way as to eliminate danger of exposure, or replace aging metal structures with plastic ones.
3. Make parents aware of the potential risk to their children of eating metal paint chips.
4. Encourage physicians to determine blood lead levels for children with a history of unusual mouthing behaviour and to report elevated results to their colleagues in public health.

Acknowledgments

The assistance of the following persons is greatly appreciated: K David, Canadian Paint and Coatings Association; V Tramonti, Inspector, Product Safety, Health Canada; JP Weber, Laboratory Director, *Centre de toxicologie du Québec*.

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Source: *SI Moore, MS, MD, Resident in Community Health, McGill University; T Kosatsky, MD, MPH, M Beausoleil, BSc, Regional Program in Public Health, Montreal-Centre; N Eade, DPhil, MD, Montreal Children's Hospital, Montreal, Quebec.*

Editorial Comment: The final report by the Working Group on Blood Lead Intervention Levels and Strategies for the Federal/Provincial Committee on Environmental and Occupational Health is in the process of being published.

The purpose of the Working Group was to review the medical/scientific evidence for the lowest observed adverse effect level of exposure to lead in children and adults, **to make recommendations** regarding the maximum blood lead level where no health effects are evident and **to make recommendations** on intervention levels and strategies for specific population groups. ***The second of these aims was precluded by the findings of the toxicologic review, which provided little evidence on clear threshold no-effect levels***. A previous report to the Federal/ Provincial Advisory Committee on Environmental and Occupational Health in 1987 recommended a maximum blood lead concentration of 1.0 to 1.25 µmol/L (20 to 25 µg/dL). The higher figure was chosen as one that would protect the health of **all** Canadians. It was initially the Working Group's understanding that any new intervention level would be all encompassing. However, as the evidence accrued it became apparent that a uniform intervention level was inappropriate for the different population groups.

Over the past 4 or 5 years evidence has accumulated that health effects occur at the 0.5 to 0.7 µmol/L (10 to 15 µg/dL) level and there is probably no threshold for these effects. The health effects of concern are cognitive and developmental in infants and children, and blood pressure and reproductive in adults. The infant *in utero* is also vulnerable to nervous system effects and may be the most susceptible population group.

It is assumed that 5% to 10% of urban children without point-source exposure have blood lead levels exceeding 0.50 µmol/L (10 µg/dL). Probably less than 1% or 2% of children in rural areas have blood lead levels greater than 0.5 µmol/L (10 µg/dL). Consequently, assuming there are about 1,325,705 urban children in Canada (an estimate), if 5% exceed 0.5 µmol/L (10 µg/dL), there may be as many as 66,285 urban children with blood lead levels greater than 0.5 µmol/L (10 µg/dL).

Blood lead levels of children living in communities with previous point-source exposures are substantially higher than those found in children living in non-contaminated areas, but the former have been lowered where abatement programs (i.e., efforts to remove the sources of lead) have been undertaken.

The major recommendation of the Working Group is that intervention strategies be implemented for the Canadian population beginning at blood lead levels of 0.5 µmol/L (10 µg/dL). The

intervention strategies adopted need to be tailored to the particular population group affected (i.e., pregnant women, children, adults, etc.), because each of the groups **has** their particular vulnerability to lead as described in the report. Further recommendations largely concern the institution of monitoring programs for specific population groups and the reporting of blood lead levels above 0.5 µmol/L (10 µg/dL) to appropriate public health authorities.

Anyone wishing to obtain a copy of this report, when available, or educational materials on lead in the home, please address your requests to the **Environmental Health Centre, Room 104, Tunney's Pasture, Ottawa K1A 0L2; fax (613) 941-8632.**

Preliminary Report

CONTACT TRACING AND FOLLOW-UP OF A CASE OF LARYNGEAL TUBERCULOSIS — ALBERTA

A 26-year-old Canadian-born health care worker was diagnosed with pulmonary and laryngeal tuberculosis on 20 July, 1994, after symptoms of cough and fatigue of about 3 months' duration. His sputum was smear and culture positive for *Mycobacterium tuberculosis*, which was sensitive to all drugs tested. More than 1,600 persons were identified as contacts of the health care worker during the symptomatic period. They were classified as family, friends, classmates, attendees at various social and recreational events, co-workers, patients and airline passengers. Alberta Tuberculosis Services is following all contacts residing in the province. Regional health units and family physicians are acting as the field component for patient evaluation. The Laboratory Centre for Disease Control (LCDC) in Ottawa is coordinating the airline passenger contact follow-up in other provinces, territories and countries. The Centers for Disease Control in Atlanta, Georgia, is coordinating the contact follow-up of American passengers. An LCDC field epidemiologist participated with Alberta Tuberculosis Services in the contact follow-up, focusing on the occupational contacts and the ongoing data analysis.

This report describes the outcome of contact follow-up to 30 November, 1994, exclusive of airline passenger contacts residing outside of Alberta. Of the identified contacts, 1,175 (70%) remain purified protein derivative (PPD), skin-test negative; 208 (13%) have a record of previous positive skin tests (i.e., prior to 1 January, 1994); 99 (6%) are new positives (the first positive skin test was documented on or after 1 January, 1994, with no previous negative skin test); 66 (4%) are converters (documented positive skin test on or after 1 January, 1994, with a documented previous negative skin test); and 127 (8%) have not yet had PPD skin testing. Five cases of suspect active tuberculosis were identified, 4 based on chest x-ray abnormalities with one being culture confirmed and fingerprint identical to the index case. Fifty-two (79%) converters and 55 (56%) first positives have started isoniazid (INH) prophylactic therapy. Those refusing INH prophylaxis will be followed at 6, 18 and 30 months with symptom inquiry and chest x-rays.

This outbreak illustrates the importance of primary prevention of tuberculosis, as well as the intense effort needed to prevent secondary infection once an active case has been identified.

Source: *S Demeter, MD, Field Epidemiologist, LCDC, Ottawa; A Fanning, MD, Director, TB Services, Alberta Health, F MacDonald, MD, A Singh, BMBS, Local Board of Health, Edmonton, Alberta.*

Clarification

1993 CANADIAN RECOMMENDATIONS FOR THE PREVENTION AND TREATMENT OF MALARIA AMONG INTERNATIONAL TRAVELLERS, CCDR 1993; 19S1

Table 4. Chemotherapy of Severe Falciparum Malaria, p. 12.

The following clarifications in quinidine dosing should be made for numbers 1, 2, and 4 treatment recommendations in Table 4:

1. **Quinidine (<u>base</u>) 6.2 mg/kg loading dose** (quinidine gluconate (salt) 10 mg/kg) by intravenous infusion over 1 to 2 hours, followed by quinidine (**<u>base</u>**) **0.0125 mg/kg/min** (quinidine gluconate (salt) 0.02 mg/kg/min) by infusion pump for 72 hours or until the patient can swallow, then quinine tablets to complete 7 days of treatment.

In the following reference, this is the dose of quinidine recommended for the treatment of 16 patients with severe falciparum malaria. Eleven of the patients also received exchange blood transfusions. The patients who received transfusions, on the average, had higher parasitemias (24.5% vs 9%) prior to therapy than the patients who were not transfused. The three patients that died were in the transfusion group but had much higher parasitemias on average (40.1% vs 18.4%) than other transfused patients.

References

1. Miller KD, Greenberg AE, Campbell CC. *Treatment of severe malaria in the United States with a continuous infusion of quinidine gluconate and exchange transfusion*. N Engl J Med 1989;321:65-70.
2. CDC. *Public Health Service inter-agency guidelines for screening donors of blood, plasma, organs, tissues, and semen for evidence of hepatitis B and hepatitis C and treatment with quinidine gluconate of persons with severe **Plasmodium falciparum** infection: discontinuation of parenteral quinine from CDC drug service*. MMWR 1991;40(RR-4):21-3.

OR

2. **Quinidine** (**<u>base</u>**) **15 mg/kg loading dose** (quinidine gluconate (salt) 24 mg/kg) in a volume of 250 mL of normal saline infused over 4 hours followed by a maintenance dose, beginning 8 hours after the beginning of the loading dose, **of quinidine** (**<u>base</u>**) **7.5 mg/kg** (quinidine gluconate (salt) 12

mg/kg) infused over 4 hours, every 8 hours for 7 days or until oral therapy can be instituted.

In the following report of the treatment of 14 patients with severe falciparum malaria, two of five patients with cerebral malaria died, but the parasitemia was eliminated in the 12 survivors. Two patients had recurrent parasitemia within 28 days of treatment. In these patients the median parasite count (*P. falciparum* rings per microlitre of blood) was 189,000, with a range of 66,971 to 1,736,106.

Reference

1. Phillips RE, Warell DA, White NJ et al. *Intravenous quinidine for the treatment of severe falciparum malaria*. N Engl J Med 1985;312:1273-78.

OR

4. **Quinine 15 mg base/kg (loading dose)**, (quinine dihydrochloride 20 mg salt/kg), by intravenous infusion over 4 hours, then 8 hourly until the patient can swallow, followed by quinine tablets to complete 7 days of treatment.

Table 5. Base Equivalents of Selected Antimalarial Drugs, p. 12.

The correct base salt conversion for quinidine gluconate is as follows:

Base	Salt
7.5 mg	12 mg
10 mg	16 mg
15 mg	24 mg

The molecular weight of quinidine gluconate, which can be calculated from its chemical formula, is 520.6. The quinidine component of this salt has a molecular weight of 324.41. The base/salt ratio is 62.3%. Of the following references only POISINDEX (Micromedex) gives a higher percentage of base due to incorrect calculation of the molecular weight of the salt.

References

1. Budavari S, ed. *The Merck index*. 11th ed. New Jersey: Merck & Co., Inc., 1989.
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Announcements

DRUG ABUSE-RELATED DEATHS

Document Available

A WHO consultation held in November 1993 reviewed the fatal consequences of drug abuse and identified inadequacies in data collection mechanisms, as well as a lack of common terminology and classification systems, as being the main reasons for the absence of internationally comparable data.

To ensure uniformity, the meeting proposed the use of the term *drug abuse-related deaths* to mean the fatal consequences resulting from the abuse of internationally controlled substances, and/or of a non-medical use of other substances for psychic effects. The term will include, when dependence is present, deaths due to acute intoxication, long-term harmful use of drugs and poisoning when accidents/suicides/homicides are directly connected with drug abuse.

A number of recommendations such as the adoption of the relevant ICD-10 codes are presented with the aim of standardizing national data collection methods, promoting research and preventing drug abuse-related deaths.

Free copies of the report on this consultation (document WHO/PSA/93.14) can be obtained in English only from the **Programme on Substance Abuse, WHO, 1211 Geneva 27, Switzerland.**

FOOD VIROLOGY

Documents Available

The WHO Collaborating Centre for Food Virology, Food Research Institute, University of Wisconsin, Madison, WI, United States, has compiled an updated version of the 1992 *Literature Review* and *List of Food Virologists*.

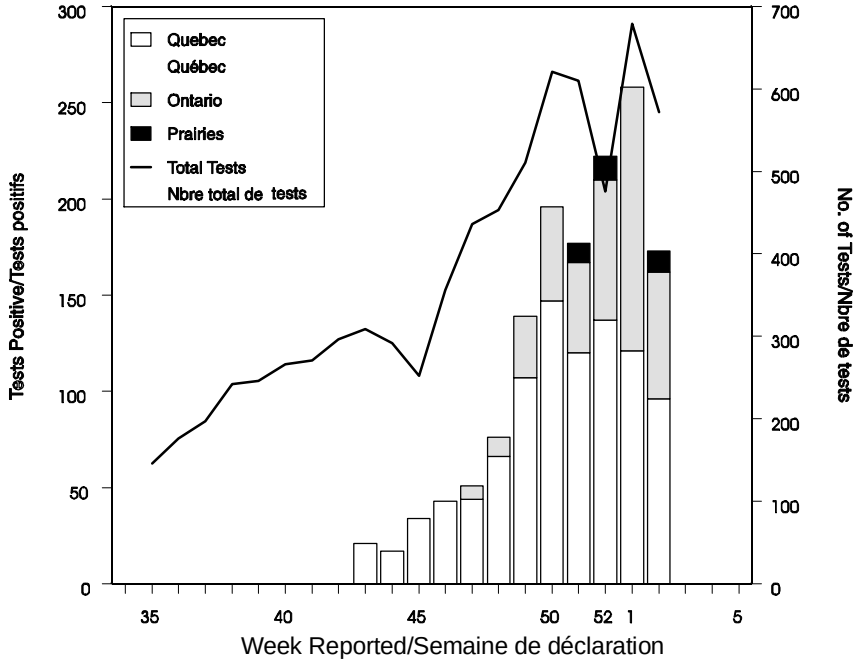
Requests for copies of *Literature Review* - 1993 and 1994 *List of Food Virologists* should be forwarded to **Food Safety Unit, World Health Organization, 1211 Geneva 27, Switzerland**.

RESPIRATORY VIRUS SURVEILLANCE
(as of 13 January, 1995)

Respiratory Syncytial Virus (RSV)

The number of reported detections of RSV increased markedly in December 1994 (Figure 1). Over half of those reported were from Quebec, reflecting an epidemic, which began at the end of 1994, affecting particularly the Montreal area. The number of RSV reports from the Toronto area has also increased in recent weeks. The World Health Organization (WHO) reports that RSV is currently very active in several European countries.

Figure 1
Positive RSV tests in Canada by region* and by week of report



* The Atlantic Provinces and British Columbia have each reported < 7 positive tests for RSV.

Influenza

WHO reports that influenza remains low in North American and most European countries.

Canada: Influenza activity remains low in Canada. Nineteen detections of influenza viruses are recorded from laboratories contributing to the Respiratory Virus Surveillance Program (influenza A, 11; influenza B, 8) from Montreal (3), Toronto (3), Manitoba (3), Regina (2), Calgary (1) and British Columbia (7).

United States: As of the end of December 1994, influenza activity was reported as regional in Connecticut, Kentucky, New York, Maryland and Virginia, and sporadic in 22 states and the District of Columbia. Although most initial identifications were of influenza A, influenza B was increasingly reported in December.

Europe: Influenza activity is low in most European countries with only sporadic reports of both influenza A and B virus identifications. Influenza outbreaks were reported in several parts of China at the end of 1994; identifications of influenza A (H3N2) have been recorded.

Source: *Laboratories contributing to the Respiratory Virus Surveillance Program, Disease Surveillance Division, Bureau of Communicable Disease Epidemiology, LCDC, Ottawa, and WHO.*

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