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Childhood Lead Poisoning in 2 Families Associated With Spices Used in Food Preparation

Alan D. Woolf, MD, MPH*†§, and Nicholas T. Woolf||

ABSTRACT. Although most cases of childhood lead poisoning are caused by contaminated paint and dust in older homes, a variety of unusual sources of lead exposure are occasionally found. We report here 2 families whose children were poisoned by lead-contaminated spices that were purchased in foreign countries, brought to the United States, and then used in the preparation of the family's food. Six children (2–17 years old) in a family from the Republic of Georgia were poisoned by *swanuri marili* (lead content: 100 and 2040 mg/kg in separately sampled products) and *kharchos suneli* (*zafron*) lead content: 23 100 mg/kg) purchased from a street vendor in Tbilisi, Georgia. The second family had purchased a mixture of spices called *kozhambu* (lead content: 310 mg/kg) while traveling in India. Both the parents and their 2-year-old child subsequently suffered lead poisoning. The young children in both families required short-term chelation to bring their blood lead levels down to a safer range. Clinicians should be vigilant for all sources of lead contamination, including spices, when whole families are found to have elevated blood lead levels despite a confirmed lead-safe home environment. Families traveling abroad should be aware of the potential health risks associated with the purchase and use of spices that have not been tested for purity. *Pediatrics* 2005;116:e314–e318. URL: www.pediatrics.org/cgi/doi/10.1542/peds.2004-2884; *plumbism, lead poisoning, children, lead toxicity, heavy-metals poisoning, spices.*

ABBREVIATIONS. PEHC, Pediatric Environmental Health Center; BLL, blood lead level; ZPP, zinc-chelated protoporphyrin.

Most cases of childhood lead poisoning in the United States are related to the ingestion of lead-contaminated house dust in the course of hand-mouth activities and oral behaviors. Peeling paint and deteriorating plaster in older homes are

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also common sources of lead hazard in young children with pica. However, other unexpected sources of lead in the home account for occasional cases of childhood lead poisoning.

We describe here 2 separate circumstances sharing a common point source of lead hazard: a family with 5 children whose childhood lead poisoning was related to a family's use of spices brought with them from an Eastern European country (the Republic of Georgia) and a case of lead poisoning in an Indian boy and both his parents related to a mixture of spices bought in India. In both families, the younger children who had been poisoned required chelation therapy to lower their bodies' burden of lead.

FAMILY 1

A family from the Republic of Georgia was referred to the Pediatric Environmental Health Center (PEHC) at Children's Hospital Boston (Boston, MA) after elevated venous blood lead levels (BLLs) were discovered during routine screening procedures at a well-child examination by their local pediatrician. The children were otherwise in good health. There was no history of pica behaviors or developmental delays.

The family had immigrated to the United States in 2003; they were living in a rented single-family house. The house was built in the 1960s and had no chipping paint or plaster according to parental report. An inspection was performed after the first elevations in BLLs were discovered among the children in February 2004 at a routine child care visit. One small area of lead contamination in front of a fireplace in the living room was discovered during the inspection; however, the parents were skeptical that this was a source of exposure, because the children spent little time in the room. There were no known occupational sources or hobbies, and the family did not live near a smelter or other industrial source of lead contamination. They did not use imported cookware or pottery for food preparation.

The 2 children who had well-child visits in February 2004 had elevated BLLs initially and at follow-up (22–37 $\mu\text{g}/\text{dL}$ [level of concern: 10 $\mu\text{g}/\text{dL}$]). The other 4 children in the family were tested in May 2004 and found also to have elevated BLLs ranging from 21 to 29 $\mu\text{g}/\text{dL}$. Blood zinc-chelated protoporphyrin (ZPP) levels were also elevated (see Table 1 for details). During the initial intake examination in the PEHC, the 2 younger children had both abdominal and long-bone radiographs, both of which were negative for evidence of recent lead ingestion or changes in the long bones associated with lead-related injury. Other laboratory measurements of the blood of the 2 children appeared normal, including hemoglobin (range: 12.7–13.4 g/dL), hematocrit (range: 35.7–39.5%), and red blood cell indices (mean cell volume range: 79–83.1 fL). Iron studies were normal, and there was no basophilic stippling on a peripheral blood smear.

Spices, including *swanuri marili* and *kharchos suneli*, were bought by the mother from a street vendor in Tbilisi, the capital of the Republic of Georgia before the family's emigration to America in 2003. These spices were liberally used by the family in many dishes at almost every meal. The spices were tested along with other household items and were found to be contaminated with

TABLE 1. BLLs and ZPPs in Family 1: 6 Children From the Republic of Georgia

Case	Age, y	Feb 2004, BLL	Mar 2004, BLL	Apr 2004, BLL	May 2004, BLL (ZPP)	Jun 2004, BLL (ZPP)	Jul 2004, BLL (ZPP)	Nov 2004, BLL
1	2.5	30	27	22	37 (120)	20 (124)	19 (106)	15
2	4	28	25	29	31 (120)	12 (146)	17 (109)	15
3	8	—	—	—	29	23 (112)	19 (85)	—
4	10	—	—	—	26	—	—	—
5	15	—	—	—	23	—	—	—
6	17	—	—	—	21	—	—	—

All BLLs are expressed in $\mu\text{g}/\text{dL}$ (BLL of concern: $\geq 10 \mu\text{g}/\text{dL}$). ZPP is expressed as $\mu\text{mol}/\text{mol}$ of heme (normal ZPP [based on a hematocrit of 35%]: 25–65 $\mu\text{mol}/\text{mol}$).

lead at a concentration up to 23 100 mg/kg. No other metal contaminants were detected. Table 2 lists the testing results along with items purchased in the United States in which lead was nondetectable.

No other credible environmental sources were found to be contaminated despite a diligent search of the household. The younger children (2 and 4 years old) were chelated with dimercaptosuccinic acid for a single 4-week course of therapy. The dramatic decline in the children's BLLs over the weeks after chelation (Table 1) and the discontinuation of use of the spices suggested that the spices were the sole toxic agents responsible for the elevation in the children's BLLs.

FAMILY 2

A 2-year-old Indian boy was referred to the PEHC because a blood lead test performed at the local pediatrician's office showed an elevated level of 31 $\mu\text{g}/\text{dL}$. A repeat BLL 10 days later showed 34 $\mu\text{g}/\text{dL}$, with a near-normal ZPP (73 $\mu\text{mol}/\text{mol}$ [laboratory range of normal values: up to 25–65 $\mu\text{mol}/\text{mol}$]). His hemoglobin was 13 g/dL, hematocrit was 36.5%, and the mean red blood cell corpuscular volume was 76 fL. He had no apparent health problems and had met all of his developmental milestones. The apartment complex in which the family resides had been built in the 1970s. Inspection of the family's apartment by officials from the state Comprehensive Lead Poisoning Prevention Program revealed no areas of contamination and no lead violations in either the residence or the common area. The patient was chelated with both d-penicillamine and dimercaptosuccinic acid over the ensuing 3-month period, which lowered his steady-state BLL to 12 $\mu\text{g}/\text{dL}$, with a ZPP of 49 $\mu\text{mol}/\text{mol}$. Subsequent follow-up over 12 months did not reveal any evidence of developmental or speech delays or behavioral problems.

TABLE 2. Results of Environmental Testing of Suspected Contaminated Products

Sample	Origin	Lead Concentration, mg/kg
Bees wax	United States	ND
Swanuri marili (sample #1)	Georgia Republic	2040
Trigonella coerulea ("utskho suneli")	Georgia Republic	12
Kinza	United States	ND
Swanuri marili (sample 2)	Georgia Republic	100
Onbalo	United States	ND
Zafron ("kharchos suneli")	Georgia Republic	23 100
Incense	Somalia	ND*
Incense	Ethiopia	13
"Kozhambu"	India	310
Complain	India	ND
Rasam	India	ND
Kai powder	India	ND
Dried tamerin	India	ND

All testing of samples was performed at the regional laboratory of the Environmental Protection Agency using radiograph fluorescence. The detection limit of the instrument is 1.0 mg/kg. ND indicates not detectable.

* Result was estimated due to small sample volume.

The family indicated that they had been using a mixture of spices, termed *kozhambu*, which they had obtained during a trip to India earlier the previous year. *Kozhambu* is a combination of turmeric, coriander seeds, chilis, and lentils ground together and sold as a mix; it is then added to casseroles and other Indian foods. Typically large quantities of *kozhambu* were used in the family meal preparation several times per week. They had just begun using this batch within the month before the initial well-child care clinic visit. Testing of the *kozhambu* by radiograph fluorescence revealed an elevated lead concentration of 310 mg/kg (see Table 2). No other metals were detected. Other household spices tested were negative for heavy metals. The mother and father subsequently were both tested and had elevated BLLs (22 and 17 $\mu\text{g}/\text{dL}$, respectively). They were not treated by their physician but were subsequently monitored to ensure declining lead levels.

METHODS

Blood Lead Level

Lead in whole blood is measured by atomic absorption (atomic absorption spectrophotometer model A analyst 600 and model A analyst 800 equipped with graphite furnace, Zeeman background correction system, and lead hollow cathode lamp; Perkin Elmer, Norwalk, CT). Ten microliters from a blood-specimen mixture (30 μL of blood + 60 μL of matrix modifier) is heated in an electronically heated graphite furnace to 2700°C. When emission spectra from a lead hollow cathode lamp pass through the tube, the atomized lead absorbs the energy of the emission spectra. A magnetic field applied horizontally eliminates nonspecific absorption. Standards of 0.5, 25, and 55 $\mu\text{g}/\text{dL}$ are prepared from human blood spiked with aqueous lead standard solution of 1000 $\mu\text{g}/\text{mL}$, purchased from Perkin Elmer. Prepared standards are verified by standard reference material (National Institute of Standards and Technology, Gaithersburg, MD). The lead-method precision for the quality-control levels at 7.8, 30.5, and 54.3 $\mu\text{g}/\text{dL}$ was 7.2%, 3.8%, and 4.8% as coefficients of variation ($N = 200$ each), respectively. The laboratory at Children's Hospital Boston participates in the Centers for Disease Control and Prevention Blood Lead Laboratory Reference System and the College of American Pathologists Proficiency Survey for Blood Lead.

Zinc Protoporphyrins

ZPPs in whole blood are measured by hematofluorometry (ProtoFluor-Z Hematofluorometer; Helena Laboratories, Beaumont, TX). One drop of a mixture (1 drop of blood + 2 drops of reagent) is placed on a glass coverslip and inserted into the instrument, in which a quartz lamp beam of light at a wavelength of 415 nm excites 1 to 2 layers of cells. Heme absorbs the light, but ZPP fluoresces and emits light at 595 nm. A second lens-filter system collects, filters, and focuses the 595-nm light beam to a photomultiplier tube, which produces a level of current. The intensity of current is analyzed by microcomputer, and the results are presented as a ratio of fluorescence/absorption or μmol of ZPP per mol of heme. The precision of measurement (range: 0–600 μmol of ZPP per mol of heme) on quality-of-control levels of 25.2, 55.8, and 128.4 $\mu\text{mol}/\text{mol}$ is 8.1%, 8.5%, and 6.2% in coefficient of variation ($N = 24$ each), respectively. The laboratory at Children's Hospital Boston participates in the National EP Proficiency Testing Program (Wisconsin State Laboratory of Hygiene, Madison, WI).

Radiograph Fluorescence

Lead measurements were performed on samples of spices at the regional Environmental Protection Agency laboratory in Chelmsford, Massachusetts, by using a Spectro X-Lab 2000 XRF instrument and the turboquant method.¹ Standard reference materials 2709 and 2711 used for quality control indicated 87% and 95% recovery, respectively, with a reporting limit of 5 mg/kg (5 ppm).

DISCUSSION

These cases demonstrate that the ingestion of spices contaminated with lead can result in clinically significant childhood lead poisoning. The cases were discovered through the policy of universal screening of BLLs in young children in Massachusetts; such case findings are a benefit of this program.

Spices comprise the leaves, seeds, flowers, and/or other plant parts of herbs containing pungent oils and other chemicals, which give the spice its characteristic taste and aroma. Many spices used in foods are purchased after grinding has produced a powdered product, and mixtures of individual spices are commonly used to enhance the flavoring of casseroles, sauces, and other prepared foods. All of the implicated spices reported here were purchased by the families during vacations in the Republic of Georgia or India and brought home with them on their return to the United States. Table 2 suggests that both *swanuli marili* (2040 mg/kg in sample 1; 100 mg/kg in sample 2) and *kharchos suneli* (zafron) (23 100 mg/kg) were the offending spices in case 1 and *kozhambu* (310 mg/kg) was the offending spice in case 2. By comparison, the Food and Drug Administration has set threshold limits for the amount of lead in foodstuffs that include, for example, no more than 2.0 mg/kg lead in salt,² no more than 1.0 mg/kg in aspartame,³ and no more than 5.0 mg/kg lead in spice oleoresins.⁴

Contamination of foodstuffs with lead in the course of their processing in preparation for sale in the marketplace has been well documented in previously published literature. Corn flour was inadvertently contaminated with lead at a concentration of 38 700 mg/kg during the grinding of corn after the repair of slits in the grindstones using lead filler, which resulted in lead poisoning of all 6 members of a single Greek family, whose BLLs ranged from 31 to 64 $\mu\text{g}/\text{dL}$.⁵ Other clusters of lead poisoning involving both children and adults have occurred in the Middle East and Turkey by contamination of wheat flour during the use of molten lead to fill fissures in worn drive shafts of the millstones.⁶⁻⁸

Contamination of herbs used in traditional ethnic and Ayurvedic remedies by heavy metals has also been well documented previously, although the circumstances under which the herbs become contaminated with lead may not always be apparent. Herbs and spices can presumably acquire the metal during growth in lead-contaminated soils or in the course of milling or other processing procedures. Use of pesticides contaminated with heavy metals during the growing of herbs and spices may also be a source of lead contamination in the final product.⁹ How-

ever, some Indian herbal (Ayurveda) remedies, folk medicines, and homeopathic remedies are purposefully adulterated with metals in the mistaken belief that they confer a health benefit to the user. There are several previous reports of clinically significant lead poisoning during the use of such remedies by adults.¹⁰⁻¹² Other metals besides lead, such as mercury, arsenic, and gold, have also been found to contaminate East Indian herbal remedies.^{13,14} Contaminated folk medicines from Asia and Mexico have also been associated with significant lead poisoning and death involving children as well as adults.¹⁵⁻¹⁹ A recent market-basket sampling of South Asian Ayurvedic herbs sold in the Boston area revealed 20% to be contaminated with such metals as lead, mercury, and arsenic.²⁰

Children are particularly vulnerable to the development of lead poisoning after chronic dosing with small amounts of such ethnic remedies, because they receive a higher dose of contaminant per unit of body weight than do adults. In 1 report, Ayurvedic capsules contained as much as 72 990 μg of lead per g of product as an adulterant and were implicated as the sources of lead poisoning among 5 adults and 2 children.²¹ A 2-month-old boy developed lead encephalopathy resulting from a lead-containing herbal powder that was routinely applied to his buccal mucosa.²² There is also a report of severe neonatal lead poisoning acquired by the maternal use of ethnic remedies during pregnancy.²³

Our report of lead poisoning in these 2 families differs from previous reports of herbal contamination, because these products were not sold as medicinal or health-enhancing products but for use as flavorings in foods. Other spices have occasionally been reported in the past to be contaminated with lead, resulting in the poisoning of adults. A 33-year-old German man suffered clinically significant lead poisoning that required chelation with intravenous Na-CaEDTA after using paprika that had been purchased in Yugoslavia and was adulterated with minium ("red lead") containing lead tetroxide; it had a measured lead concentration of 142.2 mg/kg.²⁴ In 1994, Hungarian health officials reported that the intentional adulteration of paprika with minium had resulted in widespread poisoning of >141 adults, many of whom were symptomatic and required chelation therapy.²⁵

We do not know the point at which the spices cited here were contaminated. It seems unlikely that contamination during the plant's growth by lead-containing soil or pesticides could result in such high final concentrations. Because the spices are mixtures that are typically obtained by grinding individual ingredients together, it is possible that lead filler in the millstones or other types of grinding machinery used locally could have been the source. An alternative explanation could be the intentional addition of small amounts of lead to increase the value of a commercial product that is sold by weight.

Children in both of these families required chelation therapy. Short-term follow-up revealed no evi-

dence of neurologic or developmental injury. The children did not suffer comorbidities such as iron deficiency. However, the long-term effects of even relatively short-duration exposures to lead in young children are unclear. Recent studies have demonstrated adverse effects on development and cognition in young children even at BLLs of $<10 \mu\text{g}/\text{dL}$.²⁶ Close periodic monitoring of children who have had documented BLLs of $>10 \mu\text{g}/\text{dL}$ is advisable.²⁷ Previous studies have documented the potential for damage of frontal lobes and other brain structures associated with childhood lead poisoning, with attendant decrements in cognitive and behavioral functioning that may not become apparent until later in childhood or adolescence.^{28–30} Such adverse neurodevelopmental, cognitive, and behavioral effects may persist well into adulthood.³¹

CONCLUSIONS

There are some important implications from these case reports regarding the prevention of lead poisoning. Although it does not seem probable that contaminated spices are commercially imported to the United States, this report supports the need for vigilance and continued routine testing by governmental agencies to ensure the interdiction of contaminated spices. Travelers to other countries should be warned that local spices purchased from merchants at open-air markets or from street vendors may not have been inspected by governmental officials or tested for purity.

It is incumbent on pediatric health care providers to perform a diligent search for unconventional sources of environmental lead when confronted with cases of childhood lead poisoning without an apparent etiology. This warning for vigilance concerning unsuspected lead sources is also expressed by other authors.³² The lead poisoning in the first family described in this report included older children and adolescents as well as preschoolers, suggesting a common source exposure that was not associated with the hand-mouth behaviors and pica usually implicated in the lead poisoning of young children. Clinicians confronted with such a cluster of lead poisoning should inquire about the family's history of recent travel and whether during their visit they had purchased any spices that were used subsequently in cooking. It is hoped that the reporting of these cases will serve to alert others and avert such sources of preventable childhood lead poisoning in the future.

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REFERENCES

- Office of Environmental Measurement and Evaluation, Environmental Protection Agency, Region I. Standard operating procedure for screening of soils by XRF using the Spectro X-Lab 2000. Document EIASOP-INGXRF2. September 16, 2002
- Committee on Food Chemicals Codex. *Food Chemicals Codex*. 4th ed. Washington, DC: Institute of Medicine, National Academy Press; 1996
- Committee on Food Chemicals Codex. *Food Chemicals Codex*. Third Supplement to the 4th ed. Washington, DC: Institute of Medicine, National Academy Press; 2001
- Committee on Food Chemicals Codex. *Food Chemicals Codex*. Second Supplement to the 4th ed. Washington, DC: Institute of Medicine, National Academy Press; 1997
- Dona A, Dourakis S, Papadimitropoulos B, Maravelias C, Koutselinis A. Flour contamination as a source of lead intoxication. *J Toxicol Clin Toxicol*. 1999;37:109–112
- Eisenberg A, Avni A, Grauer F, et al. Identification of community flour mills as the source of lead poisoning in West Bank Arabs. *Arch Intern Med*. 1985;145:1848–1851
- Hershko C, Eisenberg A, Avni A, et al. Lead poisoning by contaminated flour. *Rev Environ Health*. 1989;8:17–23
- Kocak R, Anarat A, Altintas G, Evliyaoglu N. Lead poisoning from contaminated flour in a family of 11 members. *Hum Toxicol*. 1989;8:385–386
- Galal-Gorchev H. Dietary intake of pesticide residues: cadmium, mercury and lead. *Food Addit Contam*. 1991;8:793–806
- Keen RW, Deacon AD, Delves HT, Moreton JA, Frost PG. Indian herbal remedies for diabetes as a cause of lead poisoning. *Postgrad Med J*. 1994;70:113–114
- Smitherman J, Harber P. A case of mistaken identity: herbal medicine as a cause of lead toxicity. *Am J Ind Med*. 1991;20:795–798
- Olujohungbe A, Fields PA, Sandford AF, Hoffbrand AV. Heavy metal intoxication from homeopathic and herbal remedies. *Postgrad Med J*. 1994;70:764
- McElvaine MD, Harder EM, Johnson L, Baer RD, Satzger RD. Lead poisoning from the use of Indian folk medicines. *JAMA*. 1990;264:2212–2213
- Sheerin NS, Monk PN, Aslam M, Thurston H. Simultaneous exposure to lead, arsenic and mercury from Indian ethnic remedies. *Br J Clin Pract*. 1994;48:332–333
- Bayly GR, Braithwaite RA, Sheehan TM, Dyer NH, Grimley C, Ferner RE. Lead poisoning from Asian traditional remedies in the West Midlands—report of a series of five cases. *Hum Exp Toxicol*. 1995;14:24–28
- Centers for Disease Control and Prevention. Folk remedy-associated lead poisoning in Hmong children—Minnesota. *MMWR Morb Mortal Wkly Rep*. 1983;32:555–556
- Centers for Disease Control and Prevention. Lead poisoning from Mexican folk remedies—California. *MMWR Morb Mortal Wkly Rep*. 1983;32:554–555
- Centers for Disease Control and Prevention. Lead poisoning associated with use of traditional ethnic remedies—California, 1991–1992. *MMWR Morb Mortal Wkly Rep*. 1993;42:521–523
- Centers for Disease Control and Prevention. Lead poisoning-associated death from Asian Indian folk remedies—Florida. *MMWR Morb Mortal Wkly Rep*. 1984;33:638, 643–645
- Saper RB, Kales SN, Paquin J, et al. Heavy metal content of Ayurvedic herbal medicine products. *JAMA*. 2004;292:2868–2873
- Prpic-Majic D, Pizent A, Jurasovic J, Pongracic J, Restek-Samarzija N. Lead poisoning associated with the use of Ayurvedic metal-mineral tonics. *J Toxicol Clin Toxicol*. 1996;34:417–423
- Yu ECL, Yeung CY. Lead encephalopathy due to herbal medicine. *Chin Med J Engl (Engl)*. 1987;100:915–917
- Tait PA, Vora A, James S, Fitzgerald DJ, Pester BA. Severe congenital lead poisoning in a preterm infant due to a herbal remedy. *Med J Aust*. 2002;177:193–195
- Lohmoller G. Lead poisoning caused by red lead in paprika powder [in German]. *Dtsch Med Wochenschr*. 1994;119:1756
- Kakosy T, Hudak A, Baray M. Lead intoxication epidemic caused by ingestion of contaminated ground paprika. *J Toxicol Clin Toxicol*. 1996;34:507–511
- Canfield RL, Henderson CR, Cory-Slechta DA, Cox C, Jusko TA, Lan-

- phear BP. Intellectual impairment in children with blood lead concentrations below 10 microg per deciliter. *N Engl J Med*. 2003;348:1517–1526
27. Centers for Disease Control and Prevention. *Managing Elevated Blood Lead Levels Among Children: Recommendations From the Advisory Committee on Childhood Lead Poisoning*. Atlanta, GA: Centers for Disease Control and Prevention; 2002
28. Needleman HL, Schell A, Bellinger D, Leviton A, Allred EN. The long-term effects of exposure to low doses of lead in childhood: an 11-year follow-up report. *N Engl J Med*. 1990;322:83–88
29. Bellinger DC, Stiles KM, Needleman HL. Low-level lead exposure, intelligence, and academic achievement: a long-term follow-up study. *Pediatrics*. 1992;90:855–861
30. Trope I, Lopez-Villegas D, Cecil KM, Lenkinski RE. Exposure to lead appears to selectively alter metabolism of cortical gray matter. *Pediatrics*. 2001;107:1437–1442
31. White RF, Diamond R, Proctor S, Morey C, Hu H. Residual cognitive effects 50 years after lead poisoning during childhood. *Br J Ind Med*. 1993;50:613–22
32. Mangas S, Fitzgerald DJ. Exposures to lead require ongoing vigilance. *Bull World Health Organ*. 2003;81(11):847

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