Dietary Calcium Intakes of Urban Children at Risk of Lead Poisoning

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Dietary calcium is well known to decrease gastrointestinal lead absorption and thereby reduce the risk for lead poisoning. Because children in economically deprived urban centers are especially likely to have excessive lead exposure, we surveyed dietary calcium intakes of 314 children from the greater Newark, New Jersey, area. The areas of Newark and adjacent communities studied had been previously identified as containing significant sources of environmental lead by geographic information systems technology. An abbreviated National Cancer Institute Health Habits and History Questionnaire, modified to focus on foods high in calcium, was used to determine dietary calcium. Calcium intakes were then compared to the new Dietary Reference Intake (DRI) guidelines. The respondents were primarily the parents of African-American and Hispanic children ranging in age from 1 to 8 years, with a mean age of 3.5 years. The most recent blood lead concentration was 11.4 ± 0.8 μg/dl (mean ± standard error), and 48.6% had concentrations at or above the current guideline of 10 μg/dl. Quintiles of calcium intake were: 221 ± 13; 488 ± 9; 720 ± 6; 885 ± 6; and 1,389 ± 49 mg/day. Fifty-five of 175 (31.4%) children aged 1–3 years had calcium intakes below the DRI, as did 82 of 139 (59.0%) children aged 4–8 years. The percentage of mothers reporting lactose intolerance in their children was 2.5%. The observation that the children in the highest quintile easily exceeded the DRIs for calcium suggests that urban parents who include dairy foods in their children’s meals can provide a diet that meets the DRI guidelines. Children in the lowest quintiles are at risk of increased absorption of the environmental lead to which they are inevitably exposed, as well as other problems associated with a low intake of dietary calcium. The data suggest that both lead exposure and low dietary calcium continue to pose significant health risks to urban minority children. Key words: African American, calcium, children, diet, Hispanic, lead, urban. Environ Health Perspect 107:431–435(1999). [Online 21 April 1999] http://ehpnet.niehs.nih.gov/docs/1999/107p431-435bruening/abstract.html

There is an abundance of evidence which demonstrates that dietary calcium decreases gastrointestinal lead absorption and thereby reduces lead toxicity. This includes evidence from studies with experimental animals and humans (1–5). Millions of American children continue to be exposed to excessive amounts of environmental lead from various sources, primarily as the legacy of the past widespread use of lead in gasoline and in exterior and interior paints. As a result of leaded paint use, an estimated 83% of all U.S. houses built before 1978 still contain potentially dangerous quantities of lead (6). The risk is particularly high in the 27% of U.S. housing units built before 1950 (6). Such housing is not uniformly distributed throughout the United States because older housing with relatively high indoor paint lead concentrations is particularly found in older northeastern cities such as Baltimore, Maryland; Boston, Massachusetts; New York, New York; Philadelphia, Pennsylvania; and Newark, New Jersey (6).

Our surveys of Newark children in the 1970s (7) demonstrated that the prevalence of elevated blood lead concentrations and lead poisoning was arguably the highest in the country as a result of lead exposure from interior paint. Much of the housing from the 1970s and earlier is still in use and therefore remains a source of excessive lead exposure. Young children 1–8 years old, particularly 1–3 years old, are at greatest risk of developing lead poisoning (8). There are a number of reasons for this, including their lower body weights, enhanced gastrointestinal lead absorption, greater hand-to-mouth activity, and their greater susceptibility to the toxic effects of lead than adults, particularly effects on the central nervous system (9,10).

Lead-containing paint dust within older homes is the main source of lead exposure in children (11). This dust can be inhaled or ingested as a result of normal mouthing behaviors.

Recent studies suggest that such mouthing behaviors are the key mechanism for lead exposure in urban children (12). However, absorption of lead via the gastrointestinal tract can be substantially reduced if the diet is relatively high in calcium (5,13), as demonstrated in children up to 2 years old by Ziegler et al. (4).

In August 1997, the Food and Nutrition Board of the National Research Council released new guidelines for the dietary intake of five essential nutrients important for bone health, including calcium (14). The dietary reference intakes (DRIs) for calcium replaced the version of the recommended dietary allowances (RDAs) that had been used as a guideline since 1989. Calcium intakes for several age groups, including very young children, were changed.

Although there are a number of studies that report dietary calcium intakes in various age and ethnic subgroups, few have focused on young urban inner-city minority group children. The objective of this study was to compare the calcium intakes of urban children at high risk of excessive lead exposure to the new DRI recommendations for calcium.

Methods

The protocol for this study was approved by the Institutional Review Board of the New Jersey Medical School, and informed consent was obtained from each participant. Subjects were mothers and their children who were consecutive attendees receiving routine medical care at well-baby clinics or other community-based pediatric clinics. All participants were residents of the Newark, New Jersey, area, including the contiguous communities of Irvington and East Orange. Most were residents of sections of Newark and adjacent communities previously identified as having significant sources of environmental lead using geographic information systems (GIS) technology (15). In addition, the target area has had a high incidence of pediatric lead poisoning for at least 30 years (7).

Children’s dietary calcium intakes were assessed using a modified food frequency instrument targeted at the major food contributors of calcium. The instrument was adapted from a pediatric version of the National Cancer Institute/Block food frequency questionnaire (16,17). It contained 14 items in which the parent was asked to describe the portion size and frequency of consumption of dairy foods and mixed foods containing dairy products, e.g., macaroni and cheese and pizza. One question asked about the presence of lactose intolerance. Mothers typically required less than

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10 min to complete the questionnaire.

The validity of the instrument was tested by administering it to a group of parents of 18 preschoolers who participated in a dietary intake study. The children attended day care centers in metropolitan New Jersey, where their food consumption was observed and recorded by trained data collectors for 2 weeks. Each evening, data collectors phoned the children’s parents to obtain a 24-hr recall of the child’s food and beverage consumption before and after day care and on the weekends. The resulting 14 days of dietary intake data were analyzed for calcium intake using The Food Processor (version 3.0, ESHA Research, Salem, OR). Calcium intake, as measured by the modified food frequency instrument, was compared with the mean calcium intake from the 14-day intake data using t-tests. No significant differences between the two methods were found, and calcium intakes calculated by the two methods were in good agreement ($r = 0.61; p = 0.007$).

Other information obtained included family income and the most recent blood lead concentration, which was obtained from medical records available at the community clinics. Assessment of relationships between blood lead concentrations and dietary calcium was not an objective of the study because the most recent blood lead concentration had usually been determined months prior to assessment of dietary calcium, and the range for the half-life of lead in blood is only about 36 ± 5 days (18). In addition, blood lead concentrations are especially labile in children and respond rapidly to increases or decreases in environmental lead exposure and other factors (19).

**Results**

Of the 314 children studied, 277 (88.2%) were members of African–American families, 28 (8.9%) were Hispanic, and 6 (1.9%) were members of white non-Hispanic families. The racial group was not identified for three children (0.9%). The mean age ± standard error (SE) was 3.5 ± 0.1 years. There were 175 children aged 1–3 years and 139 children aged 4–8 years. The percentages of males in the 1–3- and 4–8-year-old age groups were 53.7% and 50.7%, respectively.

Quintiles of calcium intake for all children studied (mean ± SE) were 221 ± 13; 400 ± 9; 720 ± 6; 885 ± 6; and 1,389 ± 49 mg/day.

Figure 1 depicts quintiles of daily calcium ingestion for the 1–3-year-old children, and Figure 2 contains the same information for the 4–8-year-old children. Quintile means of calcium intake (± SE) were 222 ± 18; 491 ± 13; 710 ± 8; 890 ± 9; and 1,416 ± 65 mg/day for the 1–3-year-old children and 234 ± 21; 493 ± 14; 739 ± 9; 887 ± 10; and 1,367 ± 78 mg/day for the 4–8-year-old children. These data demonstrate that there are very substantial differences in calcium intakes among urban children and considerable percentages with very low and very high intakes.

The mean ± SE calcium intake for 1–3-year-old children was 746 ± 34 mg/day. The corresponding value for 4–8-year-old children was 739 ± 36 mg/day. Figure 3 provides the percentages of 1–3- and 4–8-year-old children with calcium intakes below their respective DRIs of 500 and 800 mg/day. The percent of 4–8-year-old children not meeting the DRI for calcium (59.0%) was significantly ($\chi^2, p = 0.001$) greater than the proportion of 1–3-year-old children below the DRI (31.4%). The percentages of children with very low calcium intakes of less than 200 mg/day were 7.4% for the 1–3-year-old and 7.2% for the 4–8-year-old children, respectively (Figures 1 and 2).

Hispanic and African–American children in each age group had a similar incidence of calcium intakes below the DRI (32.0 and 29.4% for 1–3-year-old, and 60.5 and 63.6% for 4–8-year-old African–American and Hispanic children, respectively). Attendance at well-baby clinics did not appear to influence calcium ingestion because percentages below the DRI for 1–3-year-old children attending well-baby clinics (37%) were comparable to those attending other clinics (29%). They were also comparable for 4–8-year-old children attending well-baby clinics (52%) versus other clinics (63%). The DRIs set a tolerable upper level intake (UL) of 2,500 mg calcium/day for all age groups; only one child in the current study exceeded this value (Figure 2).

The medical records reviewed contained blood lead concentrations for 144 of the 314 (45.9%) children studied. The mean blood lead concentration was 11.4 ± 0.8 µg/dL with a range of 1.0–56.0 µg/dL. Mean values ± SE were 11.3 ± 1.1 µg/dL for 87 1–3-year-old children and 11.5 ± 1.3 µg/dL for 57 4–8-year-old children. The percentage of children with concentrations greater than or equal to the current guideline of 10 µg/dL was 48.6%.

Data on family income showed that 80% had incomes less than $20,000/year. 19% had incomes between $20,000 and $40,000 annually, and only 1% had incomes greater than $40,000/year. Dietary calcium intakes of children in the lower and middle income...
groups were 725 ± 27, and 763 ± 65 mg/day, respectively, and blood lead concentrations were 10.6 ± 0.9 and 12.5 ± 1.3 μg/dL, respectively; these differences are not statistically significant (t-test, p > 0.05). The small number of children in the high income category precludes calculation of meaningful descriptive statistical data for this group.

Eight (2.5%) mothers reported lactose intolerance in their children, 139 (44.3%) indicated that their children were not lactose intolerant, and 167 (53.2%) did not know or did not respond to this question. Mean calcium intakes of the eight lactose intolerant children (569 ± 152 mg/day) were substantially lower than those of the 139 not reporting lactose intolerance (770 ± 39 mg/day).

Discussion

Dietary calcium. The new DRI guidelines for dietary calcium are provided in the form of an adequate intake (AI), to be used as a goal for individual intakes (14). For children, AIs are 500 mg/day for 1–3-year-old children and 800 mg/day for 4–8-year-old children (14). High percentages of 1–3-year-old (31%) and especially 4–8-year-old (59%) children in this study had calcium intakes below these targets. About 7% had intakes of less than 200 mg calcium/day. These are clearly inadequate levels that may increase the risk of current and future disease, including lead poisoning.

In contrast, other children in the study, especially those 1–3 years of age, easily met the DRI for calcium. These results demonstrate that a considerable percentage of urban mothers include dairy and other foods high in calcium in their children’s diets. The data suggest that efforts to increase this percentage may well be successful, based on the high intakes of some urban children in the current study.

The higher percentage of 1–3-year-old versus 4–8-year-old children meeting or exceeding their respective AI is probably due to the higher adequate intake recommended for 4–8-year-old children. However, a recent report, citing evidence from both animal and human studies, suggested that as calcium intakes increase up to or well above 1,500 mg/day, lead absorption decreases (5). The DRI for 1–3-year-old children of 500 mg/day is lower than the RDA of 800 mg/day that it replaced (20). The basis for setting the DRI for 4–8-year-old children at 800 mg/day was evidence that this intake is sufficient to support maximal calcium retention (4). The lower DRI of 500 mg/day for 1–3-year-old children is an extrapolation from data for 4–8-year-old children. However, 500 mg/day will not protect against lead absorption and toxicity as well as 800 mg/day (4, 5). Thus, consideration should be given to increasing the DRI for 1–3-year-old children to the latter value, especially since 1–3 years is the age group with the highest incidence of lead poisoning and the group for whom its adverse effects are especially pronounced. For this and perhaps other age groups, both lead exposure and optimal skeletal development merit evaluation in setting a DRI.

Mean calcium intakes of the 1–3-year-old (746 mg/day) and 4–8-year-old (739 mg/day) children in the current study are comparable to intakes of 750 mg/day for 1–2-year-old and 772 mg/day for 3–5-year-old children reported in a large nationwide survey (21). However, the range of dietary calcium intakes found in young children investigated in the current study is broader than those typically found in other recent studies (22–24). This may be due to differences in the composition of the target population in the various studies, or it could also reflect as yet unidentified factors specific to the Newark area that would require additional investigation. The broad range of intakes suggests that there are no methodological biases producing systematic errors that either overestimate or underestimate dietary calcium in the sample studied.
Although only 2.5% of the mothers reported lactose intolerance in their children, there were probably other children in whom this condition was unrecognized. Thus, lactose intolerance probably contributed to low calcium intakes of some study subjects; it is known to be more prevalent in African Americans than in other ethnic groups (25).

The mechanisms by which dietary calcium reduces gastrointestinal lead absorption have been studied extensively in experimental animals and, to a lesser extent, in people, and involve complex interactions among lead, dietary calcium, intestinal calcium binding proteins, and vitamin D, specifically 1,25 dihydroxyvitamin D (13,26,27). These interactions explain why relatively large intakes of calcium are needed to reduce the gastrointestinal absorption of much smaller quantities of lead. Calcium may also provide protection against lead by other mechanisms because the toxic effects of lead are caused in part by its interference with calcium-mediated cellular functions (5,13,28–30).

It could be argued that providing calcium supplements to children would be a better approach than trying to increase calcium intake from food. However, diets that are high in calcium-containing foods are also rich in other important nutrients (31). In addition, the data from the current study demonstrate that a considerable percentage of urban children will have high calcium intakes from food, even in the absence of a concerted community effort to promote an increase in dietary calcium in this age group. Thus, a public health program to increase consumption of high-calcium foods by urban children with currently low calcium intakes may be rewarding. Nevertheless, the use of calcium supplements can be an additional approach for increasing the dietary intake of calcium by urban children. Some calcium supplements have been found to contain small amounts of lead, but identification of this problem should result in its resolution (32). Because dietary calcium as food or supplements may interfere with iron and zinc absorption (33,34), it will be important to recommend adequate intakes of these key micronutrients.

**Lead exposure.** The major sources of the lead to which young children are exposed are soil, street dust, indoor air, and interior lead-based paints (28). Regardless of the original source, the proximate route of exposure of young children is via the ingestion of lead-containing dust through hand-to-mouth behaviors (12,28) to a greater extent than inhalation of this dust. Thus, the ability of high dietary calcium to decrease gastrointestinal lead absorption can be a key factor in protecting young children.

A particular concern with lead exposure early in life is that it initiates the accumulation of skeletal lead which results in increasing bone lead stores with age (35,36). A recent study in rats of three different ages has shown that lead exposure early in life causes a greater retention of lead in the skeleton and other organs than exposure beginning only 5–10 weeks later (37). This study also suggests that greater bone turnover in young animals or children does not deplete their bone lead stores. In humans, increased skeletal lead stores have been associated with a lower IQ in children, aggressive behavior in young boys, and anemia, hypertension, and kidney disease in adults (38–42). Patterson et al. (43) demonstrated that skeletal lead stores of present day humans are about 3 orders of magnitude greater than those of our preindustrial ancestors. These data suggest the importance of reducing gastrointestinal lead absorption early in life in order to minimize the inevitable accumulation of skeletal lead with age. The best approach for doing this is a combination of minimizing lead exposure and providing a diet with an adequate to high calcium content.

Lanphear et al. (44) recently identified community characteristics that are predictive of elevated blood lead concentrations in children: residence within a city, black race, lower housing value, housing built before 1950, higher population density, a higher incidence of poverty, a lower percent of high school graduates, and lower rates of owner-occupied housing. Each of these characteristics is a feature of the communities in which the participants in this study live. In the current study, family income of less than $20,000/year versus more than this amount had little influence on blood lead or dietary calcium. Relatively high rents and the advanced age of the housing stock in Newark may contribute to comparable lead exposures in the income groups studied. Nevertheless, children living in communities with the above characteristics may benefit most from a successful attempt to increase their dietary calcium ingestion.

The children studied live in an area that has been a consistent source of cases of pediatric lead poisoning for at least 30 years (7,15). In addition, about half had recent blood lead concentrations >10 μg/dL. Thus, it is very likely that there is current excessive lead exposure in the population studied. Young children typically have higher blood lead concentrations than older children, but mean blood lead concentrations were similar for the 1–3- and 4–8-year-old children studied. This may be due to the conducting of blood lead analyses on 4–8-year-old children only when there is a relatively high degree of suspicion of excessive lead exposure.

**Conclusion**

In writing about lead poisoning, Bellinger and Matthews (45) stated that if one were given the task of designing a strategy to maximize exposure of an entire population to a neurotoxicant, it would be difficult to do better than to put it in the material used to line most interior surfaces of dwellings (i.e., paint) and to disperse it into the air and soil by emitting it from the tailpipe of a mobile-source whose reach is virtually unlimited (i.e., the automobile).

They further argue that the use of lead-enriched gasoline for six decades has contributed very substantially to the enormous reservoir of lead that is currently present in urban soils. These facts make it inevitable that young urban children will be exposed to lead, often in considerable amounts, which they will ingest during their normal daily activities. An emphasis on increasing their dietary calcium intake will help reduce the morbidity due to the exposure of urban and other children to environmental lead.

**References and Notes**

1. Mahaffey KR, Gartside PS, Slueck CJ. Blood lead levels and dietary calcium intake in 1–11-year-old children: the Second National Health and Nutrition Examination Survey, 1976–1980. Pediatrics 78:257–262 (1986).
2. Bogden JD, Gartner SB, Christakos S, Kemp FW, Yang Z, Katz SR, Chu C. Dietary calcium modifies concentrations of lead and other metals and renal calcium-binding in rats. J Nutr 122:1351–1360 (1992).
3. Quarterman J, Morrison JN, Humphries WR. The influence of high dietary calcium and phosphate on lead uptake and release. Environ Res 17:60–67 (1979).
4. Ziegler EE, Edwards BB, Jensen RL, Mahaffey KR, Fomon SJ. Absorption and retention of lead by infants. Pediatr Res 12:29–34 (1978).
5. Bogden JD, Oleske JM, Louie DB. Lead poisoning—one approach to a problem that won’t go away. Environ Health Perspect 105:1284–1287 (1997).
6. CDC. Screening Young Children for Lead Poisoning: Guidance for State and Local Public Health Officials. Atlanta, GA:Centers for Disease Control and Prevention, 1997.
7. Bogden JD, Singh NP, Joselow MM. Cadmium, lead, and zinc concentrations in whole blood samples of children. Environ Sci Technol 8:740–742 (1974).
8. Brody DJ, Pirkle JL, Kramer RA, Hegel KM, Mattes TD, Guttman EW, Paschal DC. Blood lead levels in the US population. JAMA 272:277–282 (1994).
9. CDC. Preventing Lead Poisoning in Young Children. Atlanta, GA:Centers for Disease Control, 1991.
10. Mitchel MD, Davis RM, Crockett AF, Grant LD. Prenatal and postnatal effects of low-level lead exposure: integrated summary of a report to the U.S. Congress on childhood lead poisoning. Environ Res 50:11–36 (1990).
11. CDC. Strategic Plan for the Elimination of Childhood Lead Poisoning. Atlanta, GA:Centers for Disease Control, 1991.
12. Lanphear BP, Roghamm KJ. Pathways of lead exposure in urban children. Environ Res 74:67–73 (1997).
13. Miller GD, Massaro TF, Massaro EJ. Interactions between lead and essential elements: a review. Neurotoxicology 11:98–120 (1990).
14. Standing Committee on the Scientific Evaluation of Dietary Reference Intakes. Food and Nutrition Board, Institute of Medicine. Dietary Reference Intakes for...
Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride. Washington, DC: National Academy Press, 1997.

15. Guthe WG, Tucker RK, Murphy EA, England R, Stevenson E, Luckhardt JC. Reassessment of lead exposure in New Jersey using GIS technology. Environ Res 59:318–325 (1992).

16. Cummins SR, Block G, McHenry K, Baron RB. Evaluation of two food frequency methods of measuring dietary calcium intake. Am J Epidemiol 126:796–802 (1987).

17. Block G, Norris JC, Mandel RM, DiSogra C. Sources of energy and six nutrients in the diets of low-income Hispanic–American women and their children: quantitative data from HHANES, 1982–1984. J Am Diet Assoc 95:195–208 (1995).

18. Landrigan PJ, Todd AC. Lead poisoning. West J Med 101:151–159 (1994).

19. O’Flaherty EJ. Physiologically based models for bone seeking elements. V: Lead absorption and disposition in childhood. Toxicol Appl Pharmacol 131:297–308 (1995).

20. Subcommittee on the Tenth Edition of the RDAs—National Research Council: Recommended Dietary Allowances. 10th ed. Washington, DC: National Academy Press, 1989.

21. Fleming KH, Heimbach JT. Consumption of calcium in the U.S.: food sources and intake levels. J Nutr 124:1426S–1430S (1994).

22. Wang MC, Crawford PB, Bachrach LK. Intakes of nutrients and foods relevant to bone health in ethnically diverse youths. J Am Dietetic Assoc 97:1010–1013 (1997).

23. Pennington JA, Schoen SA. Total diet study: estimated dietary intake of nutritional elements 1982–1985. Int J Vit Nutr Res 66:350–362 (1996).

24. Eck LH, Hackett-Renner C. Calcium intake in youth: sex, age, and racial differences in NHANES II. Prev Med 21:473–482 (1992).

25. Huang SS, Bayless TM. Lactose intolerance in healthy children. N Engl J Med 276:1283–1287 (1967).

26. Fulmer CI. Lead–calcium interactions: involvement of 1,25-dihydroxyvitamin D. Environ Res 72:45–55 (1997).

27. Bronner F. Calcium absorption—a paradigm for mineral absorption. J Nutr 128:917–920 (1998).

28. Adgate JL, Willis RD, Buckley TJ, Chow JC, Watson JG, Rhoads GG, Lioy PJ. Chemical mass balance source apportionment of lead in house dust. Environ Sci Technol 32:108–114 (1998).

29. Mahaffey KH. Nutrition and lead: strategies for public health. Environ Health Perspect 103(suppl 6):191–196 (1995).

30. Kerper LE, Hinkle PM. Cellular uptake of lead is activated by depletion of intracellular calcium stores. J Biol Chem 272:8346–8352 (1997).

31. Heaney RP. Food: what a surprise! Am J Clin Nutr 64:791–792 (1996).

32. Whiting SJ. Safety of some calcium supplements questioned. Nutr Rev 52:95–97 (1994).

33. Cook JD, Dassenko SA, Whitaker P. Calcium supplementation: effect on iron absorption. Am J Clin Nutr 53:106–111 (1991).

34. Argiris V, Samman S. The effect of calcium-carbonate and calcium citrate on the absorption of zinc in healthy female subjects. Eur J Clin Nutr 48:198–204 (1994).

35. Wittmers LE, Aufderheide AC, Wallgren J, Rapp G, Alich A. Lead in bone. IV. Distribution of lead in the human skeleton. Arch Environ Health 43:381–391 (1988).

36. Kosnett MJ, Becker CE, Osterloh JD, Kelly TJ, Pasta DJ. Factors influencing bone lead concentration in a suburban community assessed by noninvasive K X-ray fluorescence. JAMA 271:197–203 (1994).

37. Han S, Qiao X, Kemp FW, Bogden JD. Lead exposure at an early age substantially increases lead retention in the rat. Environ Health Perspect 105:412–417 (1997).

38. Needleman HL, Gwone CC, Leviton A, Reed R, Pernis H, Mather C, Barrett P. Deficits in psychologic and classroom performance of children with elevated dentin lead levels. N Engl J Med 300:689–695 (1979).

39. Needleman HL, Riess JA, Tobin MJ, Biessieker GE, Greenhouse JB. Bone lead levels and delinquent behavior. JAMA 273:363–368 (1995).

40. Hu H, Watanabe H, Payton M, Korrick S, Rotnizky A. The relationship between bone lead and hemoglobin. JAMA 272:1512–1517 (1994).

41. Hu H, Ara A, Payton M, Korrick S, Sparrow D, Weiss ST, Rotnizky A. The relationship of bone and blood lead to hypertension. JAMA 275:1171–1176 (1996).

42. Kim R, Rotnizky A, Sparrow D, Weiss ST, Wager C, Hu H. A longitudinal study of low-level lead exposure and impairment of renal function. JAMA 275:1177–1181 (1996).

43. Patterson C, Ericson J, Manea-Krichlen M, Shirahata H. Natural skeletal levels of lead in Homo sapiens sapiens uncontaminated by technological lead. Sci Total Environ 107:205–216 (1991).

44. Langhry R, Byrd RS, Auinger P, Schaffer SJ. Community characteristics associated with elevated blood lead levels in children. Pediatrics 102:264–271 (1998).

45. Bellinger DC, Matthews JA. Social and economic dimensions of environmental policy: lead poisoning as a case study. Perspect Biol Med 41:307–328 (1998).

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