Blood Lead Levels and Dietary Calcium Intake in 1- to 11-Year-Old Children: The Second National Health and Nutrition Examination Survey, 1976 to 1980

Kathyrn R. Mahaffey, PhD, Peter S. Gartside, PhD, and Charles J. Glueck, MD

From the National Institute for Occupational Safety and Health and the General Clinical Research Center, Lipid Research Center, the Division of Epidemiology and Biostatistics, and the General Clinical Research-CLINFO Centers, University of Cincinnati, College of Medicine, Cincinnati

ABSTRACT. Whether, and to what degree, dietary calcium is inversely associated with blood lead levels was examined in 2,926 black and white children, 1 to 11 years of age, from the Second National Health and Nutrition Examination Survey (NHANES II). Blood lead levels were significantly higher in black than in white children, whereas white children had significantly higher dietary calcium intake ($P < .0001$). Using covariance analysis and multiple regression analysis, determinants of blood lead levels were assessed as follows. The dependent variable was $\log_{10}$ lead, with independent variables age, sex, race, height, Quetelet index, dietary intake of calcium, phosphorous, fat, carbohydrate, and calories, community size index, poverty index ratio, geographic region, urbanization index, and all two-way interactions of the preceding. In the multiple regression analysis, the following independent variables were significant, $P < .05$. Race (black) and sex (male) were positively associated with blood lead level ($P < .0001$ for both). The lower the family income and the more urban the family residence, the higher were the blood lead levels ($P < .0001$, $< .008$, respectively). A significant independent inverse association of blood lead levels with year of examination was noted, reflecting a downward secular trend in blood lead levels. Height was inversely associated with blood lead level ($P < .0001$). Dietary calcium intake was also inversely associated with blood lead level ($P = .028$). Dietary intake of phosphorous, fat, carbohydrate, and total calories were not significantly associated with blood lead levels. The most direct strategy for prevention of childhood lead poisoning involves primary prevention to reduce exposure. However, increasing calcium intake might have value in secondary prevention of relative and absolute lead intoxication. We speculate that the NHANES II findings may be useful in identifying an avenue for secondary prevention of the effects of lead exposure by highlighting improved public health measures aimed at increasing intake of calcium and dairy products (when well tolerated), particularly in low-income black and white urban dwellers. *Pediatrics* 1986;78:257–262; *blood lead level, dietary calcium intake.*

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Reprint requests to (K.R.M.) National Institute for Occupational Safety and Health, C15, 4676 Columbia Pkwy, Cincinnati, OH 45226.

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The prevalence of elevated blood lead levels among children varies with demographic and socioeconomic factors.[1] Following a variety of environmental and public health efforts, mean blood lead levels for children and adults have declined, as demonstrated by the downward trend identified in the second National Health and Nutrition Examination Survey (NHANES II) conducted in the United States between 1976 and 1980.[2] Decreasing blood lead levels among the general population has resulted from an overall reduction in background lead exposure, eg, from air-borne lead, water, and food. However, the problem is far from solved for persons at greatest risk of lead intoxication: urban, poor, nonwhite children. Children from low-income families living in urban slums have a high prevalence of lead toxicity. Ingestion of paint containing high levels of lead has frequently been the cause of pediatric lead toxicity. Use of lead additives in residential paints was not finally banned until the US Consumer Product Safety Commission did so in 1977, setting a limit of 0.06% lead in the final dried solids, for regulatory purposes. It is estimated that 37,000,000 housing units containing high-lead paint remain in the United States.[3] The most direct strategy for prevention of childhood lead poisoning

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clearly is primary intervention to decrease lead exposure. However, because of the recognized difficulty in primary intervention with lead sources such as lead-based paints in houses,[3] secondary prevention strategies may have a role in controlling some of the effects of lead ingestion.

Nutrients, including calcium, are known to modify lead absorption by infants.[4] Metabolic balance studies with infants demonstrate that, as dietary calcium intake decreases, absorption of dietary lead increases.[4] Similar findings have been reported numerous times in experimental animal studies.[5-7] Dietary surveys conducted among high-risk groups of children from low-income families living in New York,[8] Washington DC,[9] and Milwaukee[10] have shown that children with elevated blood lead levels had lower dietary calcium intake. Whether, and to what degree, dietary calcium is inversely associated with blood lead levels, not only among the groups at highest risk for lead toxicity, but in a broadly based biracial probability sample, has not yet been examined. To address this issue we have evaluated independent associations between dietary calcium intake and blood lead concentrations using data from NHANES II.[11]

MATERIALS AND METHODS

Survey Sample Design

The NHANES II was a cross-sectional study to obtain health and nutrition data conducted from February 1976 through February 1980, which used a sample of 27,801 persons selected from 64 areas in the United States.[11] National estimates obtained by using data from these 64 areas are representative of the United States civilian noninstitutionalized population, 6 months to 74 years of age.[1,11] Further details of the complex survey design and collection of the blood samples for lead analysis are given elsewhere.[12-14] All subjects targeted for NHANES II sampling 6 months to 6 years of age and half of those 7 to 74 years of age were to have had blood lead analyses. However, 39.3% of these sample persons had missing values due to nonresponse at various stages of the survey.[14] Among medically examined persons in the lead subsample, those with missing blood lead values were randomly distributed by demographic categories other than age and socioeconomic categories.[13]

Blood lead data from blood specimens drawn by fingerstick sampling and from extreme cases of lead exposure (blood lead values of 70 μg/dL or greater) were excluded from computation in the data presented in this paper. We excluded fingerstick samples (n = 221) because they provided consistently higher lead levels than venous samples.[14] Three subjects with blood lead levels ≥70 μg/dL (>5 SD) were also excluded. After these exclusions, data on blood lead was available for 3,008 children, 1 to 11 years of age, and complete data for blood lead and dietary and environmental variables were available for 2,926 children.

Lead Analysis

Lead concentrations from venous blood specimens were measured using a modification of the microcup atomic absorption method with deuterium background correction.[15] Details of the methods and quality control systems are reported elsewhere.[12-14] Two quality control systems were used for blood lead analysis throughout the survey; "bench" quality control samples were inserted by the analyst and measured in duplicate with each analytic run to allow the analyst to make judgements on the day of analysis, and "blind" quality control samples were placed in vials, labeled with false patient identification numbers, and processed so that they were indistinguishable from regular NHANES II samples and analyzed in duplicate. The bench quality control coefficients of variation for blood lead levels of 30 μg/dL or less ranged from 7% to 15%.[12,14]

Collection of Dietary Data and Calculation of Nutrient Intake

As in NHANES I, dietary data were collected using 24-hour recall, 3-month food frequency, and dietary supplement forms.[11,16] The data presented in this report are from a 24-hour dietary recall nutrient composition tape (tape 2).[11,16,17] Nutrient values used to update foods on this tape reflect the nutrient data available between 1976 and 1980. These nutrient values were obtained either from the manufacturer or from the US Department of Agriculture's revised handbood No. 8, sections 1 to 6, on data tape 456-3 (based on US Department of Agriculture handbook No. 456). For commercial foods that were reported ingested at least 20 times, the nutrient values reflect the most appropriate information (1980) available from the manufacturer.[11,16,17] Those foods reported fewer than 20 times were not updated and may not reflect the most current nutrient analysis from the manufacturer. If the food company did not provide the needed information, nutrient values from other sources were used, where possible.[11,16,17]

For each examined person, a 24-hour recall was administered by trained dietary interviewers.[11,16,17] Specific and quantitative detail of every food or drink consumed during the previous day was recorded and calculated, thus providing estimates of

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calories, protein, carbohydrates, fat, unsaturated fats, cholesterol, and specific vitamins and minerals consumed.[17]

Statistical Methods

Of the 20,322 records contained within the NHANES II survey data tapes, 10,049 had entries for blood lead value. After exclusion for persons with capillary blood samples (n = 221) and three subjects with highly atypical lead exposure (>70 μg/dL), 9,825 subjects remained. In this analysis, the records were restricted to black and white respondents of at least 1 year of age and less than or equal to 11 years, yielding 4,165 children 1 to 11 years old with data for dietary calcium, 72% of whom (3,008) had records for blood lead levels; data on "other" (nonwhite/nonblack) races were excluded from our analyses. In the multiple regression model (Table), of the total of 3,008 children with blood lead levels, there were 2,926 (97.3%) for whom all other variables were available.

The lead values were transformed by taking their square roots and their logarithmic (base 10) values. The distributions of these data were then examined by histogram plots, and it was found that logarithms (base 10) possessed the classical appearance of symmetry and unimodality. To verify this choice of transformation, a further examination of the data was carried out. A multiple regression model was fitted to the lead values, with age, race, sex, poverty index, urbanization index, height, Quetelet ratio (weight/height^2), dietary phosphorous, dietary fat, dietary carbohydrate, calories, dietary calcium, geographic region, date of examination, and interactions as independent variables. This analysis was repeated with square root and logarithmic (base 10) transformed lead values. The residual deviations of the data from the model were then examined by histogram plots as above, and again the logarithm (base 10) values were the only ones that possessed a unimodal, symmetric distribution.

The set of 2,926 were then (first) analyzed by unweighted covariance analysis using the SAS package of statistical computer programs.[18] This unweighted, unstratified analysis provided an indication of which relationships between lead and the explanatory variables were likely to be significant in the subsequent weighted and stratified SURREGR regression model.[19] The dependent variable was $\log_{10}$ lead, with independent variables age class (1 to 5 years; 6 to 11 years), sex (male, female), race (white, black), height, Quetelet ratio, dietary calorie intake, dietary calcium intake, dietary fat intake, dietary carbohydrate intake, dietary phosphorus intake, community size index (1,000,000 or more; 2,500 to 999,999; less than 2,500), poverty index ratio (PIR) ($1 \leq \text{PIR} \leq 155$; $156 \leq \text{PIR} \leq 280$; $280 < \text{PIR}$),[20] geographic region (northeast, midwest, south, west), date of examination, and all two-way interactions of the preceding.

The final set of analyses were performed using the SURREGR statistical computer program[19] which necessarily takes into account the variable sampling weights and the various strata that were used in gathering the data in the NHANES II survey.[11,13] The dependent variable was $\log_{10}$ lead, with exactly the same set of independent variables as used above. All of the independent variables were initially entered into the regression model together, with no special entry order, so that possible sources of bias would be adjusted for. The net contribution of each independent variable to reduction of variance in this model[19] is only obtained by its simultaneous entry into the model along with other independent variables, irrespective of its correlation with the other independent variables. After each weighted and stratified multiple regression analysis was completed, the least significant independent variable was deleted from the model. This was continued until the remaining independent variables were each significant at the .05 level (Table).

TABLE 1. Relationship of Blood Lead Level to Race, Sex, Poverty Class, Region, Degree Urbanity, Examination Date, Calcium Intake, and Height*

Variable	Partial Regression Coefficients	Standardized Partial Regression Coefficients	*df*	*F* Value	*P* Value
Race	0.112	8.83	1	78.92	.0001
Sex	−.033	−4.93	1	24.18	.0001
Poverty class			2	17.97	.0001
Region			3	5.56	.0035
Urbanity			1	5.71	.0075
Examination date	−.049	−7.22	1	52.06	.0001
Calcium intake	−.000023	−2.35	1	5.28	.0282
Height	−.002	−129.79	1	103.46	.0001

* Poverty class, urbanity, and region were included in the model; their partial regression coefficients are not shown because they were class variables. Multiple $R^2 = .357$, $P = .0001$; n = 2,926.

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RESULTS

The mean (±SD) and 25th, 50th, and 75th percentiles for blood lead levels in the 2,926 children (Table) were, respectively, 15.7 ± 6.6 and 11, 14, and 19 μg/dL; corresponding dietary calcium intakes were, respectively, 851 ± 461 and 522, 789, and 1,110 mg/d. Those independent determinants of blood lead levels that were significant at the $P \leq .05$ level in the multiple regression model are shown in the Table. The explanatory variables race (black) and sex (male) were highly significantly and positively associated with blood lead level ($P < .0001$). The lower the family income and the more urban the family residence, the higher the blood lead levels were (Table). There was a significant inverse association of blood lead with examination date; lower blood levels were observed as the study progressed.[2] This was not reflection of systematic laboratory drift but, as previously reported, a reflection of a secular trend in blood lead levels.[2]

Height was a significant explanatory variable, inversely associated with blood lead level ($P < .0001$) (Table).

Dietary calcium intake was also a significant explanatory variable for blood lead level ($P = .028$); this relationship was inverse (Table). White children had higher calcium intake than black children ($P < .0001$). None of the other dietary variables examined (calories, fat, carbohydrate, phosphrous) were significant explanatory variables for blood lead level.

The variance in blood lead levels was explained best by height and in diminishing order by race, examination date, sex, and dietary calcium intake, using standardized partial regression coefficients (Table).

DISCUSSION

In the current study of dietary calcium-blood lead interactions in 1- to 11-year-old children in the NHANES II national probability sample, we evaluated a large biracial sample, with a diverse regional and demographic sampling frame, not selected because of high risk for lead intoxication. A significant, independent, inverse association was observed between dietary calcium intake and blood lead levels. There was relative imprecision of the 24-hour dietary recall as a tool to quantitate calcium intake and more precision of the measurement of the other well-known explanatory variables for blood lead levels including race, sex, poverty class, urban class, and height.[1,13] Such imprecision in quantitation of dietary calcium intake would make a significant dietary calcium to blood lead level relationship harder to detect.

Our observation of an inverse association between dietary calcium intake and blood lead level in the national probability sample of the NHANES II study parallels findings of three previous smaller studies which were much more narrowly focused on high-risk, low-income, preponderantly black populations.[8-10] These antecedent reports did not allow black/white comparisons, did not include suburban and rural populations, did not provide large numbers of children with low lead exposures, and did not represent a national probability sample. These three antecedent studies were of children from inner city areas of New York,[8] Washington, DC,[9] and Milwaukee.[10] The children were preponderantly black,[8-10] but the groups were matched for age, race, sex, geographic residence, economic level, and freedom from other diseases. Similar to our findings in the NHANES II sample, children in New York,[8] Washington, DC,[9] and Milwaukee[10] with lower blood lead concentrations had higher dietary calcium intakes. Sorrell et al[8] determined dietary calcium intakes from groups of 1- to 6-year-old children living in New York City. Dietary calcium intake in these children was assessed by a trained interviewer who administered a dietary questionnaire to the child's parent and calculated dietary calcium intakes using tables of food composition. Children who had blood lead levels >60 μg/dL had significantly lower intakes of calcium and vitamin D. Johnson and Tenuta[10] determined dietary intake using three-day records for 43 children 1 to 6 years of age living in Milwaukee. The racial distribution of the group of 43 children included 35 black, four Mexican American, three white, and one Puerto Rican. There were significant inverse correlations between blood lead levels and both calcium intake and number of servings of a milk group of foods.[10] Similar results were found when the individual values were used in regression analysis or by categorical analysis of average daily dietary calcium intake when the subjects were grouped by low, moderate, and high blood lead levels. In Washington, DC, Mahaffey et al[9] investigated dietary calcium intake among children 12 to 43 months of age, using four-day duplicate diet samples that were analyzed for calcium. Dietary calcium intakes were consistently lower in black children with elevated blood lead levels.[9]

In these small, narrowly focused studies,[8-10] low dietary calcium intakes were associated with a greater prevalence of elevated blood lead levels, a finding replicated in the much larger, national probability NHANES II sample which included both black and white races. In the three studies reviewed above,[8-10] nearly all of the subjects were nonwhite and of low socioeconomic status, limiting the gen-

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eralization of the data. Interpreting the calcium-lead interaction was further complicated in these three studies[8-10] because the same socioeconomic and racial groups that consumed the diets lower in calcium also had a much higher prevalence of elevated blood lead levels and potentially higher environmental lead exposures. In contrast, the NHANES II data set allows examination of the calcium-lead interactions in urban, suburban, and rural areas in both blacks and whites and in individuals with variegated socioeconomic status.

The lower intake of dietary calcium in blacks observed in the NHANES II survey parallels previous observations from a variety of smaller cohort studies, which also showed lower dietary calcium intake and dairy product intake in blacks. Thus, for example, in the Lipid Research Clinics collaborative studies,[21] there were significantly lower intakes of milk, cheese, and dairy products in black children than in white children from variegated socioeconomic status backgrounds. Similar results were obtained from the National Center of Health Statistics analysis, which indicated that consumption of calcium was lower among families whose income levels were below the poverty level in the United States[16] compared to families with incomes above the poverty level. In addition, the National Research Council reported that use of dairy products was generally lower among black, Asian and Hispanic persons.[22]

The basis for lower use of dairy products in blacks (as compared with whites) has been discussed extensively, with a number of reasons cited, including lactose intolerance, cultures less accustomed to drinking milk, low income, restricted access to market, and limited food storage facilities. For example, Koh and Caples[23] conducted a nutrition survey of 1,000 black families living in rural poverty in southwestern Mississippi. Of all nutrients studied, calcium was the greatest nutritional deficiency. Many of the households rarely or never used milk or milk products. In the study by Koh and Caples,[23] several factors combined to account for this low use of dairy products, including limited access to food markets, limited food storage facilities, not recognizing calcium as a nutrient, and poverty.

What is the potential importance of these cross-sectional epidemiologic observations of inverse dietary calcium to blood lead level interrelationships? We speculate that our findings may be useful in providing an intelligent base for some health policy decisions. There is broad general agreement that primary prevention is the optimal approach to restriction of exposure to environmental lead.[24-26] Whereas this broad consensus is widely embraced, the continuing problems of substandard housing with lead-based paint[24,25] and the still substantial environmental contamination resulting from lead-containing gasolines[26] will not be resolved soon. Dedicated efforts to deal with environmental lead contamination must continue to receive prioritized governmental and scientific support and must accompany any type of secondary prevention, such as dietary calcium management aimed at reducing lead absorption.[27]

A variety of metabolic studies have suggested that interventions that increase dietary calcium intake may influence lead absorption and retention among infants and young children,[4] as well as adults.[28,29] For example, Ziegler et al[4] in 1978 conducted metabolic balance studies among infants and very young children. Food intake during and between the balance periods was known. Ziegler et al[4] reported an inverse association between dietary calcium intake and retention and absorption of lead by young infants. Blake and Mann[28] in 1983 found that ingestion of milk reduced the 96-hour retention of labeled lead by two fasting adult subjects from greater than 60% of the ingested dose to less than 20%. However, the magnitude of the change in lead absorption produced by milk was smaller than that produced by similar quantities of calcium carbonate and phosphorous, given as an organic salt. Similar findings were reported by Heard and Chamberlain[29] among adult subjects.

In addition to human metabolic studies, as above, there are extensive animal data that suggest that, when higher levels of calcium are fed during long periods of time, substantially reduced levels of tissue lead are realized.[5-7] Thus, there may well be a role for secondary prevention and/or direct dietary intervention in those subjects identified as having unacceptably high exposures to lead. Inasmuch as there is a significant independent and inverse association of family income to blood lead levels, reducing poverty should, to some degree, improve diet and housing, with concomitant reductions in blood lead levels. Public health measures might beneficially be aimed at increasing intakes of calcium and dairy products (when well tolerated), particularly in poor black and white urban dwellers. Urban children younger than 6 years of age, particularly black children from low income families, should have blood lead measured, and those with levels ≥ 25 $\mu g/dL$ should have the analysis repeated. If the repeat measurement is ≥ 25 $\mu g/dL$, comprehensive environmental modification is mandated[30] and dietary calcium intake should be substantially increased.

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