Influence of Nutrient Intake on Blood Lead Levels of Young Children at Risk for Lead Poisoning

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Although removal of lead paint hazards from at-risk houses remains the primary means of preventing elevated blood lead among young children, reduction of risk through nutritional factors has also been of interest. In this study we evaluated the effect of nutrient intake on blood lead levels by analyzing whether the intakes of certain dietary components *a*) were associated with blood lead levels independent of lead exposure or *b*) modified the effect of lead exposure on blood lead. Subjects were 205 children from low-income families who were approximately 1 year of age and living in old, urban houses. The data collected for each child included blood lead level, nutritional status, and amount of lead exposure, which was assessed from samples of household dust. Multiple linear regression analyses showed a statistically significant positive association between lead exposure and blood lead. Statistically significant positive associations were found between blood lead and total fat as well as blood lead and saturated fat, independent of lead exposure and age of the child. Regression modeling and stratified analysis showed that mean blood lead increased with increasing lead exposure as well as with increasing caloric intake, suggesting that caloric intake modifies the association between lead exposure and blood lead. The findings from this study, if replicated in other studies, support a dietary intervention to reduce the amount of total calories, total fat, and saturated fat among children 1 year of age at risk for lead exposure, while maintaining adequate intake of these dietary components. Our results also reinforce recommendations that removal of lead paint hazards from at-risk houses should be the primary means of preventing lead exposure. *Key words:* caloric intake, children, lead, modification, nutrition. *Environ Health Perspect* 110:A767–A772 (2002). [Online 12 November 2002]
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Despite a drop in the prevalence of lead poisoning among children in the United States, the Centers for Disease Control and Prevention estimated that in the early 1990s, approximately 890,000 preschool children had blood lead levels greater than 10 μg/dL (Pirkle et al. 1998). Most children with elevated lead levels are exposed to lead through lead-contaminated house dust, which originates primarily from lead-based paint in old, urban homes (Lanphear et al. 1995; Lanphear and Roghmann 1997). Despite environmental interventions to reduce the number of houses with lead paint exposures, it has been estimated that more than 25 million homes still contain significant amounts of lead paint (Jacobs et al. 2002).

Although removal of lead paint hazards from at-risk houses remains the primary means of preventing elevated blood lead, reduction of risk through nutritional factors has also been of interest. Previous epidemiologic studies have found significant inverse associations between blood lead levels and the dietary intake of a number of nutrients, including iron, calcium, vitamin D, and vitamin C ([Anonymous] 1978; Ballew et al. 1999; Hammad et al. 1996; Johnson and Tenuta 1979; Mahaffey et al. 1976, 1986; Sorrell and Rosen 1977). In addition, findings from human studies suggest that total fat and caloric intake are positively associated with blood lead levels (Lucas et al. 1996). Few of these studies, however, have adjusted adequately for differences in lead exposure. Furthermore, to our knowledge, the question of whether nutrient intake modifies the association between blood lead and lead exposure in children has not been examined, although animal studies have shown a stronger association between blood lead and lead exposure for animals on high fat or high calorie diets compared with animals on low fat or low calorie diets (Barltrop and Khoo 1975; Bell and Spickett 1983; DeLuca et al. 1982; Kello and Kostial 1973; Nzelibe et al. 1986).

To address the effect of nutrient intake in young children at risk for elevated blood lead levels, we focused on whether the intakes of certain dietary components *a*) are associated with blood lead levels independent of lead exposure or *b*) modify the effect of lead exposure on blood lead. Data were collected from a sample of children of approximately 1 year of age living in old, urban houses. Lead exposure was assessed by collecting samples of dust from the household.

Methods

Sample. Subjects were children born to women enrolled in a randomized trial designed to evaluate the effectiveness of an intervention in reducing lead exposure in young children. Women were eligible for the study if they were less than 6 months pregnant, 17 years of age or older, Medicaid registrants, residents of selected urban neighborhoods with a high proportion of houses built before 1950, and willing to participate in the study. Each woman who was enrolled in the study signed a consent form approved by the Investigational Review Board of the University of Maryland, Baltimore.

When the child was 1 year of age, data were collected regardless of randomization assignment. Of the 357 women initially enrolled in the study, 205 had children on whom complete 1-year follow-up data were obtained. These children were included in the analysis.

Measures. **Nutrition.** Nutritional status of the children was assessed at 12 months using the Children's Nutrition Questionnaire, which was designed and validated by Harvard University School of Public Health experts (Blum et al. 1999) and analyzed by them using a nutrient composition database. The questionnaire was administered to the child's mother by a trained interviewer and assessed the child's frequency of intake of 85 food items over the previous 4 weeks. Because previous studies have reported or suggested an association between blood lead and the following dietary component intakes per day, these components were selected for analysis: total caloric intake, total fat intake, protein, carbohydrates, saturated fat, monounsaturated fat, polyunsaturated fat, cholesterol, animal fat, vegetable fat, calcium, iron, magnesium, phosphorus, zinc, vitamin D, and vitamin C.

Blood lead. When the child was 1 year of age, the mother was asked to bring her child to the study clinic where a sample of the child's blood was taken. Approximately 67% of the children had their blood taken at the

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study clinic. At the clinic visit, venous blood was obtained by a trained phlebotomist or nurse using a Stat Sampler Blood Collection System (Fisher Health Care, Houston, TX). Blood taken at the study clinic was analyzed for lead at the Maryland Department of Health and Mental Hygiene by atomic absorption spectrophotometry using a stabilized temperature platform furnace technique. The level of detection for this method is 2 µg/dL. Blood lead results were measured in duplicate and the reported result was the mean of the two analyses. Quality control and assurance (QC/QA) measures included the use of three levels of commercially prepared controls that were run with each batch of samples.

If the mother was not able to bring her child to the study clinic, she was asked to sign a release form allowing us to obtain the child's most recent blood lead measurement from his or her pediatrician. Approximately 33% of the blood lead measurements were obtained in this manner. The method of blood collection and place of blood lead analysis for these subjects were not known.

Exposure. Exposure to lead was assessed from samples of household dust. Dust wipe samples were taken at the 12-month housing inspection by experienced visual inspectors certified by the Maryland Department of the Environment. The rooms from which lead dust samples were obtained were the kitchen, the child's playroom/living room, and the child's bedroom. Dust samples taken from the windowsills in all of the rooms were combined into a composite sample. Similarly, composite samples were obtained for window wells, noncarpeted floors, and carpeted floors (if carpeting was present).

Samples were collected with baby wipes and placed in prelabeled centrifuge tubes. All samples of each type from a house (carpeted floor, noncarpeted floor, windowsill, and window well) were placed in a single tube. To prevent contamination, the inspector performing the dust wipe collection used new, clean, disposable gloves for each wipe sample. In addition, all templates were wiped down prior to sampling. METS Laboratories (Waldorf, MD), accredited under the U.S. Environmental Protection Agency (U.S. EPA) National Lead Laboratory Accreditation Program, analyzed the lead content using U.S. EPA protocol SW846-7420, which implements a microwave-digestion process and flame atomic absorption (FLAA) methods (U.S. EPA 1994). The method detection limit using FLAA is 5.0 µg total lead per sample; the reporting limit is 10.0 µg total lead per sample. QC/QA was performed in accordance with the American Industrial Hygiene Association's internal quality control procedure manual (AIHA 1997). For internal quality control purposes, METS included method spike samples and blanks in each dust wipe analysis run. In addition, the study staff added field spikes and blanks to each batch of samples sent to METS. The error rates of the field spike samples and blanks were monitored by the study staff over the length of the study. All reported spike values were within 20% of the known value.

Statistical analysis. Because of non-normal distributions, the blood lead, dust lead levels, and caloric intake variables were analyzed using logarithmic values. The remaining dietary component values were transformed by adjusting for total calories.

Mothers whose children had complete data were compared with mothers whose children had missing data using Wilcoxon rank-sum tests for continuous variables and chi-square tests for categoric variables. The unadjusted associations between blood lead and dust lead, as well as blood lead and the dietary component intake values, were assessed using both multiple linear regression and Pearson correlations.

Multiple linear regression models were constructed for each dietary component to determine whether dietary component intake modified the association between dust lead levels and blood lead. For each model, dust lead level (windowsill, window well, or floor), the dietary component intake variable, and the interaction term were included. In addition, the age of the child at the time of the blood draw was included as a confounder. If the interaction term was significant or marginally significant ($p < 0.1$), stratified analyses were carried out. If the interaction was not significant, the term was dropped from the model and the association between dietary component intake and blood lead controlling for dust lead level and the age of the child was assessed. Because results were essentially the same for models using windowsill, window well, and floor dust lead levels, only the results of the regression models and stratified analyses using windowsill dust lead levels are reported. In addition, due to the high degree of collinearity among the dust lead types, we did not include all types in one model.

For the stratified analyses, "low" dust lead exposure was defined as dust lead levels in the lowest tertile (2–19 µg/ft^2), "average" dust lead exposure was defined as dust lead levels greater than 19 µg/ft^2 and less than or equal to the U.S. EPA dust lead hazard standard for sills (250 µg/ft^2), and "high" dust lead exposure was defined as greater than the U.S. EPA dust lead hazard standard for sills. Three categories

Table 1. Comparison of mothers of children included and not included in analyses.

Characteristic	Included[a]	Not included
Sample size	205	127
Age (median, range)	22 (17–39)	23 (18–38)
No. others in household (median, range)	4 (1–17)	4 (1–10)
Race (%)*		
White non-Hispanic	2.9	7.1
Black non-Hispanic	96.6	89.9
Other	0.5	3.1
Marital status (%)		
Married	4.9	9.5
Single	90.7	84.2
Other	4.4	6.3
Education (%)		
Some high/junior high school completed	40.0	34.7
High school graduate or GED	44.9	43.3
Some college/college graduate	15.1	22.0
Household Income (%)		
< $10,000	40.0	37.8
$10,000–19,000	17.6	16.5
$20,000–29,000	2.4	6.3
Not given	40.0	39.4
Employed (%)		
Yes	35.1	44.9

[a]Included if child had blood lead level, household dust samples, and nutrition information at 1 year. *$p < 0.05$.

Table 2. Characteristics of infants at 1-year study visit.

Characteristic		
Females, *n* (%)	98	(47.8)
Age at blood lead measurement, mean (SD), months	12.3	(2.3)
Height, mean (SD), in (*n* = 194)	29.6	(1.7)
Weight, mean (SD), lbs (*n* = 187)	22.0	(3.3)
BMI, mean (SD), kg/in^2 (*n* = 186)	17.8	(2.3)
Head circumference, mean (SD), cm (*n* = 190)	46.0	(2.1)
Blood lead level, median (range), µg/dL	4.0	(1–19)
Blood lead level ≥ 10 µg/dL, *n* (%)	10	(4.9)

BMI, body mass index.

of caloric intake were created based on tertiles, with the "average" category defined as above 1,219 kcal and below 2,091 kcal. The mean blood lead for children in each level of caloric intake and of dust lead was computed.

Stratified analyses were also completed to assess whether the modification of the association between lead exposure and blood lead by caloric intake differed by sex. Because of smaller numbers for these analyses, only two lead exposure categories were created based on the U.S. EPA lead dust standard for sills ("meets U.S. EPA standard" and "above U.S. EPA standard").

All statistical analyses were performed using SAS (SAS Institute Inc. 1987). Results were considered statistically significant for *p*-values less than 0.05.

Results

As shown in Table 1, the 205 mothers of children included in the study were significantly more likely to be black non-Hispanic than those who were not included in the study ($p < 0.05$); however, over 85% of women in both groups were black non-Hispanic. The two groups did not differ significantly with regard to any other demographic characteristics.

The overwhelming majority of mothers whose children were included in the study were single and black non-Hispanic. About half had received a high school diploma or GED and had a household income < $10,000. Their median age was 22.0 years and, on average, there were four other people living in the household.

The infants who were included in the study had their blood lead measured between the ages of 9 and 23 months (mean ± SD, 12 ± 2.3 months; Table 2). Approximately 4% of the children had their blood drawn before they were 11 months of age and 15.6% had their blood drawn after they were 13 months of age. The median blood lead level was 4 µg/dL. Ten children (4.9%) had blood lead levels measuring 10 µg/dL or greater; the highest was 19 µg/dL. Older age at blood lead measurement was marginally associated with a higher mean blood lead ($p = 0.09$).

The measurements of household lead obtained from the dust wipes are presented in Table 3. Median dust lead concentrations of 40 µg/ft^2 and 448 µg/ft^2 were found for windowsills and window wells, respectively. Dust lead levels from noncarpeted floors and carpeted floors had median concentrations of 10 µg/ft^2 and 6 µg/ft^2, respectively. A large variation in levels can be seen from the ranges computed for each dust wipe type.

In Table 3, the crude correlations are presented for blood lead and dust lead level for each surface. Crude correlations between blood lead and dust lead levels for each of the four surfaces were significant; uncarpeted floors had the highest correlation at 0.43. A significant correlation of 0.22 was found between blood lead and the average dust lead level across all surfaces (data not shown).

The estimated daily level of those dietary components examined and the association between each of them and blood lead are shown in Table 4. Both the unadjusted correlations and the unadjusted regression coefficients are included. Statistically significant positive associations were found between blood lead and calories ($r = 0.15$; $\beta = 0.15$), total fat ($r = 0.17$; $\beta = 11.08$), saturated fat ($r = 0.18$; $\beta = 21.35$), monounsaturated fat ($r = 0.16$; $\beta = 27.45$), and animal fat ($r = 0.14$; $\beta = 9.41$). Significant negative associations were found between blood lead and carbohydrates ($r = -0.13$; $\beta = -2.96$) and vitamin C ($r = -0.14$; $\beta = -1.14$).

In multiple linear regression analyses, the interactions between windowsill dust lead levels and each dietary component intake were examined. Total caloric intake was found to be a marginally significant effect modifier of the association between lead exposure and blood lead ($p = 0.06$). In addition, statistically significant positive associations were found between blood lead and total fat ($p = 0.03$) and between blood lead and saturated fat ($p = 0.02$), independent of the age of the child and the windowsill dust lead level. As expected, in each multiple linear regression model, we found a statistically significant positive association between blood lead and windowsill dust lead level ($p < 0.0001$). In addition, in these models, either a statistically significant ($p < 0.05$) or marginally significant ($p < 0.1$) positive association was found between blood lead and the age of the child.

The analysis examining the association between blood lead and lead exposure stratified by level of caloric intake is shown in Table 5. At the low level of dust lead exposure, a consistent pattern of increasing or decreasing mean blood leads was not observed across caloric intake groups. However, at the higher levels of dust lead exposure, the mean blood lead increased with increasing dust lead exposure (average to high) as well as with increasing caloric intake (low to average to high). In addition, the largest difference in mean blood lead from low to high dust lead exposure was seen among children in the high caloric intake group. The smallest difference was seen among children in the low caloric intake group. As expected, in each caloric intake group, children categorized as having high dust lead exposure had the highest mean blood lead.

Table 3. Dust lead measurements by type and correlation of log-normalized dust lead levels and log-normalized blood lead levels.

Sample collection site[a]	Nontransformed dust lead levels			Correlation of log dust lead levels with log blood lead levels[b]
	No.	Median (µg/ft^2)	Range	
Windowsills	204	40	(2–19,347)	0.38*
Window wells	159	448	(2–875,833)	0.21*
Uncarpeted floors	200	10	(3–1,538)	0.43*
Carpeted floors	114	6	(3–108)	0.36*

[a]Composite dust sample. [b]Log of dust lead levels used to normalize variables for analysis. *$p < 0.05$.

Table 4. Nutrient intake, correlation of normalized intake with normalized blood lead levels, and unadjusted regression coefficient for the association of normalized nutrient intake and normalized blood lead levels.

Nutrient	Nontransformed nutrient intake	Transformed nutrient intake[a]	
	Median daily intake (range)	Unadjusted correlation with log blood lead levels	Unadjusted regression coefficient
Calories (kcal)	1,697 (221–7,079)	0.15*	0.15*
Protein (g)	53 (9–199)	0.01	0.46
Carbohydrate (g)	238 (26–1,067)	–0.13*	–2.96*
Total fat (g)	53 (6–267)	0.17*	11.08*
Saturated fat (g)	19 (1–89)	0.18*	21.35*
Monounsaturated fat (g)	18 (2–112)	0.16*	27.45*
Polyunsaturated fat (g)	10 (2–47)	0.09	24.94
Cholesterol (mg)	178 (8–860)	0.10	0.97
Animal fat (g)	22 (1–136)	0.14*	9.41
Vegetable fat (g)	23 (4–182)	0.06	5.73
Calcium (mg)	562 (66–3,263)	0.09	0.22
Iron (mg)	10 (1.5–39)	–0.05	–20.05
Magnesium (mg)	237 (29–872)	–0.06	–1.15
Phosphorus (mg)	1,032 (142–3,794)	0.08	0.29
Zinc (mg)	7 (1–27)	0.05	24.20
Vitamin D (IU)	125 (2–1,032)	0.09	0.58
Vitamin C (mg)	132 (14–649)	–0.14*	–1.14*

[a]For normalization of variables, calories analyzed as log of calories; other nutrients adjusted for total calories. *$p < 0.05$.

Subgroup analyses were performed to determine whether modification of the association between blood lead and lead exposure by caloric intake held for both males and females. Table 6 shows separately the stratified analysis for males and females. Males had an increasing mean blood lead with increasing dust lead exposure ("meets standard" to "above standard") as well as with increasing caloric intake (low to average to high). The largest difference in mean blood lead from low to high dust lead exposure was seen among males in the high caloric intake group. The smallest difference was seen among males in the low caloric intake group. A similar pattern was seen for females; however, females who had an average caloric intake level and were exposed to dust lead levels above the U.S. EPA standard had a higher mean blood lead than females who had a high caloric intake level and were exposed to dust lead levels above the U.S. EPA standard.

Discussion

Our results are consistent with the position that environmental lead exposure is the primary cause of blood lead elevation in young, urban children (Lanphear et al. 1996, 1998; Rhoads et al. 1999). A substantial percentage of houses had windowsill and well dust lead levels above the current U.S. EPA dust lead hazard standards (28% and 43%, respectively). Our study found a strong statistically significant positive association between blood lead and lead exposure, as measured by windowsill, window well, and floor dust lead levels, even in very young children with somewhat limited mobility. The high-risk children in our study were younger than children assessed in most other studies of lead and nutrient intake. At about 1 year of age they had slightly elevated blood lead levels, and therefore are typical of the vast majority of high-risk children today. Elucidating the effect of nutrient intake on blood lead in children as young as 1 year of age would aid in identifying specific dietary interventions that could protect them from the potential damaging effects of lead.

Results from this study suggest that total caloric intake modifies the association between lead exposure and blood lead. In a stratified analysis of the entire sample, children in the highest tertile of daily caloric intake had the largest difference in mean blood lead across dust lead exposure groups, whereas children in the lowest tertile of daily caloric intake had the smallest difference. These patterns persisted for both males and females as well as for younger children (< 12 months of age) and for older children (12 months of age or older; data not shown for age). To our knowledge, there have been no other human studies investigating whether caloric intake modifies the association between dust lead exposure and blood lead; however, this modification is supported by data from animal studies (Barltrop and Khoo 1975; Bell and Spickett 1983; DeLuca et al. 1982; Kello and Kostial 1973; Nzelibe et al. 1986). In addition, findings from human studies have been reported for the association between caloric intake and blood lead. Lucas et al. (1996) showed a positive association between blood lead and caloric intake, independent of lead exposure. These investigators assessed exposure using a lead exposure index constructed from self reports. The consistency of our results using an objective lead exposure assessment with those of Lucas et al. (1996), who used a nonobjective lead exposure assessment, strengthens the confidence of the findings with regard to blood lead and caloric intake.

One explanation for the finding of a modification of the association between lead exposure and blood lead by caloric intake is that those who eat more calories may be ingesting more lead through food that has been contaminated, either by lead in the air or in packaging (Lucas et al. 1996). In addition, those children in the highest caloric group may have eaten more finger foods, which are more likely to be contaminated, resulting in increased lead ingestion. Although the types of foods eaten in each caloric group in our study were not assessed, Freeman et al. (1997) showed that consumption of finger foods such as hamburgers, donuts, peanut butter and jelly sandwiches, and cold cuts was associated with elevated blood lead levels among children with low to moderate blood lead levels. Additionally, consideration must be given to biologic explanations.

A statistically significant positive association was found between blood lead and total fat intake, independent of child's age and dust lead level. Previous human epidemiologic studies and experimental animal studies support this finding (Barltrop and Khoo 1975; Bell and Spickett 1983; DeLuca et al. 1982; Lucas et al. 1996; Mahaffey 1995). A possible mechanism by which dietary fat increases lead absorption involves the stimulation of bile secretion into the gastrointestinal tract by fat (Bell and Spickett 1983; Hilburn et al. 1980; Lucas et al. 1996). Bile, which aids in the digestion and absorption of fat, also increases lead absorption in the gastrointestinal tract (Bell and Spickett 1983; Hilburn et al. 1980). In addition to total fat intake, we found saturated fat intake was significantly associated with blood lead levels; few previous human and animal studies have reported on the different types of fat and lead absorption.

We did not find significant associations between blood lead and iron, calcium, vitamin C, or vitamin D, independent of lead exposure and child's age. These nutrients have been studied extensively with regard to blood lead, and other investigators have consistently found significant inverse associations between blood lead and the intake of these nutrients ([Anonymous] 1978; Ballew et al. 1999; Hammad et al. 1996; Johnson and Tenuta 1979; Mahaffey et al. 1976, 1986; Sorrell and Rosen 1977). Metabolic studies have shown that low intake of iron, calcium, vitamin D, and vitamin C may enhance the intestinal absorption and tissue retention of lead, resulting in increased lead toxicity (Barton et al. 1978a, 1978b; Edelstein et al. 1984; Hamilton 1978; Hammad et al. 1996; Hashmi

Table 5. Mean blood lead at different levels of dust lead and caloric intake.[a]

	Caloric intake level[b]		
Windowsill dust lead level	Low	Average	High
Low	3.74 (1.94) *n* = 17	4.23 (2.05) *n* = 17	3.33 (2.04) *n* = 20
Average	3.27 (2.09) *n* = 33	4.12 (2.07) *n* = 33	5.67 (4.30) *n* = 26
High	4.41 (1.97) *n* = 17	6.29 (4.22) *n* = 17	6.63 (4.32) *n* = 24

[a]Mean blood lead given as µg/dL (SD). [b]Categories defined by tertiles.

Table 6. Mean blood lead at different levels of dust lead and caloric intake by sex.[a]

	Caloric intake level[b]		
Windowsill dust lead level	Low	Average	High
Males			
Meets U.S. EPA standard	3.34 (1.91) *n* = 29	4.57 (2.24) *n* = 22	4.85 (3.85) *n* = 23
Above U.S. EPA standard	3.90 (1.20) *n* = 10	5.89 (4.73) *n* = 9	7.08 (4.44) *n* = 12
Females			
Meets U.S. EPA standard	3.55 (2.24) *n* = 21	3.84 (1.85) *n* = 28	4.46 (3.54) *n* = 23
Above U.S. EPA standard	5.14 (2.67) *n* = 7	6.75 (3.85) *n* = 8	6.17 (4.34) *n* = 12

[a]Mean blood lead given as µg/dL (SD). [b]Categories defined by tertiles.

et al. 1989; Hsu et al. 1975; Klauder and Petering 1975; Mahaffey and Goyer 1972; Ragan 1977; Singh et al. 1991; Six and Goyer 1970; Sobel and Burger 1955; Suzuki and Yoshida 1979). The lack of consistency between the results of this study with previous reports may be due to a number of reasons. First, the adjustment for lead exposure using dust wipe levels may have eliminated the previously observed associations in human studies between nutrient intakes and blood lead. Few studies have controlled for lead exposure when examining the role of nutrients. This adjustment is important because children with insufficient intakes of nutrients such as iron, calcium, vitamin D, and vitamin C may also be children who are exposed to high levels of lead. Second, in most of the previous human and animal studies, the levels of lead toxicity measured by blood or tissue lead concentration were higher than the levels we found. The level of lead exposure in this study may not have been high enough to see significant biologic effects of differential lead absorption (measured by blood lead) due to nutrient intake. Third, in previous human studies, the mean ages of the children were older. Significant associations that may be relevant in supporting nutritional interventions in older children may not apply to children as young as the ones in this study; additional studies of younger children are needed. Thus, a comparison of previous studies with this one may not be informative. Finally, the food frequency questionnaire that we used may not be useful in discriminating different levels of intake in the diets of children 1 year of age.

Several limitations of this study must be considered when interpreting the results. First, not all children of the 357 women initially enrolled in the study were included in the analyses. Those who were not included may have differed in some important characteristic. The data available to assess this possibility showed that women whose children were included in the analyses had similar characteristics to those who were not included. Thus, the results should be generalizable to all study participants and perhaps to a larger population of children with similar characteristics.

Second, although a validated food frequency questionnaire (Blum et al. 1999) was used to obtain estimates of nutrient intake, it is possible that women did not accurately report the type and frequency of food intake for their children. Some mothers were not with their children at all times during the day because of work or school commitments and may have given inaccurate information. Third, the assessment of lead exposure may have been incomplete, as lead levels in food and in the child's outside environment (such as soil) were not assessed. In addition, blood lead levels were low and had limited variation; only 10 subjects had levels greater than 10 μg/dL. Thus, the study may not have had a sufficiently large number of subjects to detect whether nutrient intake modified the association between lead exposure and blood lead within this restricted range. Furthermore, the study may not have had adequate statistical power to detect significant associations between the intakes of specific nutrients and blood lead, independent of lead exposure and age.

This study also has several important strengths. First, we used a validated food frequency questionnaire to assess nutrient intakes, which measures dietary intake reasonably well among preschool children of low-income families (Blum et al. 1999). In addition, we used lead dust wipes, an objective measure, to assess and control for lead exposure in the study. Dust wipes are currently accepted as the best method of evaluating lead hazards in the household (Lanphear et al. 1995, U.S. EPA 2001, U.S. HUD 1995). The few previous studies that controlled for lead hazards in assessing the association between nutrient intake and blood lead used exposure indexes from self reports, which may be a less accurate measure of lead exposure (Hammad et al. 1996; Lucas et al. 1996).

The strong positive association between dust lead levels and blood lead that was found in all regression models reinforces recommendations that removal of lead paint hazards from at-risk houses should be the primary means of preventing lead exposure. However, our findings warrant more research into secondary prevention strategies, such as nutritional interventions, to control blood lead levels. The findings from this study, if replicated in other studies, support a dietary intervention to reduce the amount of total calories, total fat, and saturated fat among children 1 year of age at risk for lead exposure, while maintaining adequate intake of these dietary components. In addition, future studies should address specific types of foods eaten by young children, especially finger foods (hamburgers, peanut butter and jelly sandwiches), and their relationships to blood lead. The incorporation of dietary interventions with regard to other nutrients, such as iron and calcium, should be further explored in children 1 year of age who have relatively low blood lead levels to determine their role in the prevention or development of elevated lead.

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