Zinc and iron deficiency and their interrelations in low-income African American and Hispanic children in Atlanta[1–4]

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ABSTRACT

Background: Information about the zinc status of low-income minority children in the United States is lacking.

Objective: The objective was to determine the prevalence of zinc deficiency and anemia and their interrelation among low-income African American and Hispanic preschool children.

Design: This was a cross-sectional study in which a prospective 3-d food diary was completed, and hemoglobin, serum ferritin, zinc, copper, and C-reactive protein concentrations were measured. Children with elevated C-reactive protein concentrations were excluded from analysis.

Results: Of 292 children recruited, 280 (mean ± SD age: 2.5 ± 1.2 y) qualified for analysis. One hundred forty-six (52%) children were African American and 134 (48%) were Hispanic; 202 (72%) were enrolled in the Women, Infants, and Children nutrition program. A low serum zinc concentration (<10.7 μmol/L) was present in 34 (12%) children, and 37 (13%) were anemic (hemoglobin < 110 g/L). African American (odds ratio: 3.47; 95% CI: 1.51, 7.96) and anemic (odds ratio: 2.92; 95% CI: 1.24, 6.90) children had an increased risk of zinc deficiency. Serum zinc correlated with hemoglobin ($r = 0.24$, $P < 0.001$). Children with a height/length less than the fifth percentile had significantly lower mean serum zinc concentrations than those with a height/length greater than the fifth percentile (12.4 ± 1.8 compared with 13.0 ± 2.2 μmol/L; $P < 0.001$). In a multiple logistic regression model, African American race-ethnicity was associated with zinc deficiency (odds ratio: 0.26; $P = 0.02$). The main sources of iron and zinc in the diets were meat products and cereals.

Conclusions: The prevalence of zinc deficiency and anemia was high in this population of low-income minority children, especially among African Americans. Further investigation of the incidence of zinc deficiency and the ability of anemia to screen for it is warranted. *Am J Clin Nutr* 2010;91:1027–34.

INTRODUCTION

Micronutrient deficiencies are caused by inadequate dietary intake, increased losses from the body, and/or increased requirements (1). Zinc and iron usually occur together in food sources. Foods with a high content of dietary zinc and iron include oysters, beef, turkey, chicken, fortified cereals, and processed beans (2). The zinc content in a recommended serving size varies in these foods, ranging from 76 mg in oysters to 15 mg in fortified cereals, 11 mg in turkey, 9 mg in beef, and 6 mg in chicken (2). However, the bioavailability of micronutrients is less than anticipated despite the substantial quantities found in some of these foods (3, 4). Certain components of cereals and legumes, such as phytic acid, fiber, and calcium, affect zinc and iron absorption (5, 6). Poverty, poor food choices, lack of availability or decreased accessibility to certain foods, coupled with the lack of knowledge about the importance of food group diversity for the health and growth of young children may limit the inclusion of micronutrient-rich foods in the diets of children. Deficiencies of iron, iodine, zinc, and vitamins A and B-12 are a major concern among children globally (7, 8). Considerable evidence exists to show that such deficiencies affect physical growth, cognitive development, reproduction, physical work capacity, and exposure to risks of infectious and several adult-onset chronic diseases (8–11). Despite this evidence, the most recent "Special Supplemental Nutrition Program for Women, Infants, and Children" (WIC) food package for children aged 1–4 y does not include the best dietary sources of iron and zinc (2, 12).

Iron deficiency anemia is the most common micronutrient deficiency–associated anemia in the world, affecting up to 60% of children globally (13). In the United States, the prevalence of anemia (defined as low hemoglobin) continues to be high among children from low-income families, with a prevalence of 15% for preschoolers (14.6% reported for the state of Georgia), despite the efforts of the WIC program and other agencies, including the

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Centers for Disease Control and Prevention (CDC) (14–17). Iron deficiency without anemia has an estimated prevalence of 7% in US toddlers (18, 19). Although zinc deficiency is not usually recognized as a major problem in developed countries, it rivals iron deficiency as a major contributor to overall micronutrient deficiency in developing countries (20, 21). Significant linear relations have been identified between the iron and zinc statuses of individuals (22). There have also been reports of an increased prevalence of zinc deficiency among 12–36-mo-old and 6–9-y-old Hispanic children from low-income families in the United States (23, 24).

The primary objective of this project was to assess the prevalence of zinc deficiency in at-risk African American and Hispanic children in urban Atlanta, Georgia, and to identify associations with population demographics, race, anemia, growth, and dietary intake. The primary outcome was the presence of zinc deficiency, as defined by a serum zinc concentration of <10.7 μmol/L. We hypothesized that such a deficiency is prevalent among low-income minority children and that it is associated with anemia and poor growth in this population.

SUBJECTS AND METHODS

Study population

This was a cross-sectional study performed in 2 metropolitan Atlanta clinics (the Pediatric Clinic at Children's Health Care of Atlanta Hughes Spalding/Grady Memorial Hospital and the North Dekalb Grady satellite clinic) and was approved by the Research Oversight Committee of Grady Memorial Hospital and the Institutional Review Boards (IRBs) of both Emory University and Children's Health Care of Atlanta. Procedures were in accordance with the ethical standards of the IRBs on human experimentation of the respective institutions and the 1983 revision of the Helsinki Declaration of 1975. The primary caregivers were approached, and the study was explained in both English and Spanish through oral presentation and printed documents. Primary caregivers consented by signature, and the children (subjects) were not compelled to participate.

The clinics were sampled because they primarily serve low-income families and WIC-eligible children (12). WIC offices are located in the North Dekalb Grady satellite clinic and in a building adjacent to Children's Health Care of Atlanta at Hughes Spalding. The convenience method of sampling was used to recruit preschool children (1–5 y of age) between February 2006 and July 2007. Recruitment occurred during morning clinic hours between 0800 and 1300. Children were enrolled if they were between the ages of 12 and 60 mo and attending the clinic for a check-up visit (eg, immunizations or school physical examinations). Children with sickle cell anemia, acute diarrhea, or respiratory illness and those with screening C-reactive protein (CRP) concentrations >10 mg/L were excluded from the study.

Baseline data were collected on date of birth, sex, race, the number of children/adults in the household, daycare attendance, birth information of parents, and other potential risk factors. Growth was assessed by direct measurement of weight and height by a single study coordinator at each study site. Children <2 y of age had supine lengths measured to the nearest 0.1 cm with the use of a rigid length board with a movable foot piece (Aryton Infantometer, model M-200; Seca, Hamburg, Germany). Children 2 y of age or older had standing heights measured to the nearest 0.1 cm with a stadiometer (235 heightronic digital; Seca, Hamburg, Germany). These instruments were standardized every 6 mo. Heights/lengths were measured in duplicate, and the mean height/length of the measurements was recorded. Weight was measured to the nearest 0.1 kg with a Precision scale (model 500; SR instruments, Tonawanda, NY). The scale was standardized annually. The weights were also taken in duplicate, with the mean weight recorded. A single study coordinator measured heights and weights at each site. The coordinator was trained to reduce intrameasurer variability in length/height and weights before data collection at each site. Height and weight percentiles were calculated by using Epi-Info 3 version 3.3.2 software (CDC, Atlanta, GA). Height and weight percentiles were used to assess nutritional status and to make comparisons with age-matched children in the US population.

Laboratory analysis

The children were not required to fast. Blood was obtained from the supine subject through venipuncture by a trained phlebotomist study coordinator at the 2 clinic sites between 0800 and 1300. Tourniquet use for occlusion of the child's arm was limited to <1 min. Vacutainer (21 g and 23 g) butterfly needles (Becton Dickinson, Franklin Lakes, NJ) and 7-mL royal blue–topped Vacutainer tubes (Becton Dickinson) certified as trace element–free were used for blood collection (25). The phlebotomist used prescreened disposable polyethylene gloves, free of talc or other coatings. Samples of the collected whole blood were pipetted into HemoCue microcuvettes, and hemoglobin concentrations were measured within 5 min of blood collection with the use of the HemoCue B-Hemoglobin machine (Ängelholm, Sweden). The blood samples were transported on ice to our laboratory, where it was allowed to clot for 2 to 4 h at room temperature, and serum was removed after centrifugation (at 2400 rpm for 15 min at 25°C) and stored at -80°C until analyzed. CRP was assessed by using a NycoCard reader II (Polymedco Inc, Cortlandt Manor, NY). Ferritin, zinc, and copper analyses were conducted at the laboratories of the Division of Laboratory Sciences, National Center for Environmental Health, CDC. Zinc and copper analyses were performed by using standard inductively coupled plasma mass spectrometry (ICP-MS; Perkin-Elmer, Paris, France), whereas ferritin was measured by immunoturbidimetry with a Roche/Hitachi 912 clinical analyzer (Roche Diagnostics, Indianapolis, IN) (26). Zinc deficiency was defined pre hoc as a serum zinc concentration <10.7 μmol/L (1, 27). Low serum copper was defined as a concentration <14.1 μmol/L (27). Anemia was defined as a hemoglobin concentration <110g/L (27). Low iron stores were defined as a ferritin concentration ≤ 35.95 pmol/L (16.0 ng/mL) (28).

Food records

The caregiver was instructed on how to complete the 3-d food record diary in English or Spanish for 3 consecutive days. This included detailing the time at which the food was consumed and in which setting (home, school, or restaurant). Families were provided with measuring cups and pictures to be used in assessing the amount and portion sizes of food consumed. They were also instructed to provide details of food preparation at home (29). The

food record diary was modified from a validated food record that is used for clinical purposes. The document and a stamped addressed envelope were given to the caregiver. After completing the 3-d record prospectively, the caregiver mailed it to the research team. Once received, this food record was assessed and entered into the Nutrition Data System for Research (NDSR; University of Minnesota, Minneapolis, MN) dietary analysis program by the research nutritionist at the bionutrition core of the Emory University Hospital site of the Atlanta Clinical and Translational Science Institute for analysis of the average daily dietary intake of calories, carbohydrate, fat, protein, phytate, iron, copper, and zinc.

Statistical methods

This was a cross-sectional study in which prospective food collection records were used to assess nutritional intake. Descriptive statistics (proportions, means, and SDs) were used to calculate baseline information. Calculation of the sample size was based on the estimation of mean serum zinc concentration difference of 0.8 μmol/L between the 2 groups ($1 - \beta = 0.80$, $\alpha = 0.05$). This calculation required that 119 children be recruited in each of the 2 groups (total $n = 238$); however, 292 children were recruited to account for a dropout rate of 15% as well as elevated CRP concentrations, unsuccessful blood draws, and hemolyzed blood samples. Nonlinearity was observed through inspection of sample size description plots and the use of a Kolmogorov-Smirnov test for ferritin, serum copper, and serum zinc. These nonlinear data were log transformed for statistical analyses and converted back to the original units as geometric means and SDs. Chi-square and Fisher exact tests were used to compare baseline characteristics of the study participants between the 2 groups (zinc deficient and zinc sufficient). Every subject was categorized as zinc deficient or zinc sufficient, with the zinc deficiency boundary set at $<10.7\mu$mol/L.

Relative odds ratios (ORs) and 95% CIs were calculated to identify potential risk factors associated with zinc deficiency in the study population. Prevalence ORs were calculated from logistic regression models. As this was a risk prevalence study, a multiple logistic regression model, with the outcome variable dichotomized as zinc deficient or zinc sufficient, was developed. Relevant independent variables were included in the model, including race. Covariates included measures of weight, height, anemia, and socioeconomic indicators (birth country of parents, WIC participation, and maximum education attained by parents). The model was developed by manual backward stepwise elimination, removing the covariate with the largest P value at each step until the remaining variables were significant at the 0.05 level in the final model. Bivariate analysis using Pearson's correlation was applied to serum zinc, nutrient groups and other biochemical measures (hemoglobin and copper) to assess for potential confounders and to test the strength of this association. All statistical analyses were assessed by using SAS 9.1 (SAS Institute Inc, Cary, NC). A P value <0.05 was deemed as statistically significant for all analyses. Data are shown as means ± SDs.

RESULTS

During the 18-mo study period, 292 preschool children were enrolled and had blood samples drawn. Twelve children were excluded from the analysis because of elevated CRP concentrations, which were defined as >10 mg/L. Thus, 280 children (95.9%) were included in the analysis. The demographics of the children are shown in **Table 1** and **Table 2**. The mean (±SD) age of the children was 2.5 ± 1.2 y, and most (66%) of the children were aged <3 y. African American and Hispanic children were enrolled in roughly equal proportions at 52.1% and 47.9%, respectively. The mean (± SD) household size was 4.7 ± 1.7, and 2.4 children in each household were aged <18 y. Mean (± SD) serum zinc and copper concentrations among these children were 13.0 ± 2.0 and 21.4 ± 3.9 μmol/L, respectively. A total of 12.1% of the children had low serum zinc concentrations (<10.7 μmol/L). All of the studied children had serum copper concentrations within the normal range (Table 1).

Mean serum zinc concentrations were significantly lower among African American preschool children than among Hispanic preschool children, and they were significantly lower among those covered by government-sponsored health insurance programs than among those with private health insurance (Table 2). Standardizing weights and heights/lengths indicated that 20 (8.4%) of the children had weight-for-age values less than the fifth percentile, and 24 (10.1%) had height/length-for-age values less than the fifth percentile. Those with a height/length less than the fifth percentile for age had significantly lower mean serum zinc concentrations than did those with a height/length greater than the fifth percentile (12.4 ± 1.8 μmol/L compared with 13.0 ± 2.2 μmol/L; $P = 0.0001$). However, those with weights less than the fifth percentile had significantly higher mean serum zinc concentrations than did those with weights greater than the fifth percentile (13.3 ± 2.5 μmol/L compared with 12.9 ± 2.1 μmol/L).

The prevalence of anemia was 13.2%; anemic children had a significantly lower mean (± SD) serum zinc concentration of 12.0 ± 1.8 μmol/L than did nonanemic children ($P = 0.004$) (**Table 3**). However, mean serum zinc concentrations were not significantly different between children with low iron stores (defined as a ferritin concentration ≤35.95 pmol/L, or 16 ng/mL) and those with adequate iron stores. Serum copper concentrations were significantly higher in children with normal serum ferritin than in those with low ferritin concentrations (Table 3). African American children had a mean (± SD) hemoglobin concentration of 119 ± 11 g/L, and the Hispanic children had a mean (± SD) hemoglobin concentration of 130.8 ± 77.7 g/L ($P = 0.09$). A significantly higher proportion of African

TABLE 1
Characteristics of the subjects and their households

Variable	n	Mean ± SD	Range
Age (y)	280	2.5 ± 1.2	1–5
No. of children aged <18 y in household	280	2.4 ± 1.2	1–7
Household size (n)	280	4.7 ± 1.7	2–12
Zinc (μmol/L)[1]	280	13.0 ± 2.0	8.2–20.5
Copper (μmol/L)[1]	279	21.4 ± 3.9	11.7–36.3
Hemoglobin (g/L)	280	121.3 ± 11.0	86–160
Ferritin (pmol/L)[1,2]	278	68.7 ± 43.5	6.7–301.1

[1] Data were log transformed for statistical analysis.
[2] To convert pmol/L to ng/mL or μg/L, divide by 2.247.

TABLE 2
Demographic characteristics and mean serum zinc concentrations in low-income minority preschool children[1]

Variable	Subjects	Serum zinc[2]	P^3
	n (%)	*μmol/L*	
Age			0.13
1–2 y	185 (66)	12.8 ± 2.2	
3–5 y	95 (34)	13.3 ± 2.2	
Sex			0.45
Female	139 (49.6)	12.9 ± 2.2	
Male	141 (50.4)	13.1 ± 2.2	
Race-ethnicity			0.0001
African American	146 (52.1)	12.5 ± 2.0	
Hispanic	134 (47.9)	13.6 ± 2.3	
Weight-for-age percentile			0.0001
<Fifth	20 (8.4)	13.3 ± 2.5	
≥Fifth	218 (91.6)	12.9 ± 2.1	
Height/length-for-age percentile			0.0001
<Fifth	24 (10.1)	12.4 ± 1.8	
≥Fifth	214 (89.9)	13.0 ± 2.2	
WIC nutrition program participation			0.86
No	78 (27.9)	12.9 ± 2.0	
Yes	202 (72.1)	13.0 ± 2.3	
Insurance			0.02
Government sponsored	218 (77.9)	12.8 ± 2.2	
Other	62 (22.1)	13.5 ± 2.0	
Daycare attendance			0.35
No	235 (83.9)	13.0 ± 2.2	
Yes	45 (16.1)	12.7 ± 2.2	
Mother's educational level[4]			0.15
<12 y	119 (43.6)	12.7 ± 2.0	
≥12 y	154 (56.4)	13.1 ± 2.3	
Father's educational level[5]			0.93
<12 y	107 (44.2)	13.0 ± 1.9	
≥12 y	135 (55.8)	13.0 ± 2.4	
Single-parent household			0.02
Yes	58 (20.7)	13.1 ± 2.2	
No	222 (79.3)	12.4 ± 1.9	
Birth weight			0.69
<1500 g	213 (76.1)	13.0 ± 2.2	
≥1500 g	67 (23.9)	12.9 ± 2.1	
Ever breastfed[6]			0.88
Yes	50 (19.9)	13.0 ± 1.9	
No	201 (80.1)	12.9 ± 2.1	

[1] WIC, Women, Infants, and Children.
[2] Values are means ± SDs.
[3] Determined by using Student's *t* test.
[4] Information on mother's educational level was missing for 7 enrollees.
[5] Information on father's educational level was missing for 38 enrollees.
[6] Information on history of breastfeeding was missing for 29 enrollees.

American children (18.1%) than of Hispanic children (7.6%) had anemia ($P = 0.03$). Serum zinc and hemoglobin concentrations were positively correlated ($r = 0.26$, $P < 0.001$).

Risk factors among zinc-deficient children were evaluated (**Table 4**). African American children had a greater risk of zinc deficiency than did Hispanic children (OR: 3.47; 95% CI: 1.51, 7.96). There was also an association between zinc deficiency and anemia, because anemic children had a greater risk of being zinc deficient (OR: 2.92; 95% CI: 1.24, 6.90). Logistic regression was used with all the variables to determine the adjusted ORs associated with zinc deficiency (**Table 5**). This model confirmed the significant relation between race-ethnicity and the risk of zinc deficiency. If the definition of zinc deficiency was changed to serum zinc <12.3 μmol/L, African American race-ethnicity (OR: 0.46; 95% CI: 0.24, 0.90) and government-sponsored insurance status (OR: 0.34; 95% CI: 0.16, 0.74) were identified by logistic regression model to have a significant relation with the risk of zinc deficiency.

Food records were received from 179 of 280 (64%) participants. There was no difference in the demographic characteristics or serum concentrations between the children who had food records returned and those who did not have their records mailed back to the research team. These 179 food records (51.4% from Hispanic families and 48.6% from African American families) indicated that African American children consumed more calories and fat than did Hispanic children (**Table 6**). The major sources of iron and zinc in the children's diets were meat products and cereals, with dietary zinc increasing with age ($r = 0.02$, $P = 0.007$). There was no difference in the zinc-rich foods consumed by African American and Hispanic children. There was no correlation between dietary zinc and serum zinc ($r = 0.01$, $P = 0.84$). However, dietary zinc rose with increasing intakes of energy ($r = 0.58$, $P = 0.0001$), iron ($r = 0.69$, $P < 0.001$), animal protein ($r = 0.68$, $P < 0.001$), plant protein ($r = 0.64$, $P < 0.0001$), and phytate ($r = 0.57$, $P < 0.001$). Phytate intake also correlated with intake of iron ($r = 0.64$, $P < 0.001$), but had no correlation with serum zinc ($r = 0.08$, $P = 0.23$) or ferritin ($r = 0.04$, $P = 0.61$) concentrations. There was also no correlation between serum zinc concentrations and the molar ratio of phytate to zinc ($r = 0.08$, $P = 0.29$) in this population.

DISCUSSION

Zinc is an important micronutrient that is essential for normal growth and development among humans. Zinc supplements reduce childhood morbidity and mortality in developing countries (30–32). Zinc deficiency is thought to be uncommon in the United States and other developed countries, because the consumption of zinc-rich food is common in these countries, even in low-income households (12). As a result, few studies in the United States have focused on examining the status of this micronutrient and on identifying sociodemographic groups at risk of deficiency.

Our study was unique in that it focused on evaluating zinc and concomitant anemia/iron status among low-income, urban, minority preschool children in the United States, most of whom (66%) were younger than 3 y. Our results support the hypothesis that zinc deficiency occurs in this population, and the risk among African American (black) children is 4-fold that of Hispanic (nonblack) children. The study showed a prevalence of serum zinc deficiency (<70 μg/dL, or <10.7 μmol/L) of 12% overall, but with 19.4% among low-income, African American, preschool children compared with 4.8% in Hispanic children, which indicated that zinc deficiency is more widespread in low-income, urban, African American children than expected and potentially should be monitored. Because serum zinc may be an insensitive indicator of zinc status relative to other kinetic zinc parameters, such as the various zinc pool compartments, the serum zinc cutoff was changed to 81 μg/dL (12.3 μmol/L) to evaluate the prevalence of zinc deficiency, which increased to 44% at this cutoff (22). Given the significant relation between

TABLE 3
Serum zinc and copper concentrations and their relation with anemia and iron status (ferritin)[1]

	Anemia (hemoglobin < 110 g/L)			Low iron status (ferritin < 35.95 pmol/L)[2]		
	Yes	No	*P*	Yes	No	*P*[3]
n	37	243	—	49	224	—
Serum zinc (μmol/L)	12.0 ± 1.8[4]	13.1 ± 2.2	0.004	12.7 ± 1.6	13.0 ± 2.3	0.29
Serum copper (μmol/L)	22.0 ± 3.9	21.4 ± 3.9	0.36	19.9 ± 3.3	21.8 ± 4.0	0.002

[1] Data were log transformed for statistical analysis.
[2] A ferritin concentration of 35.95 pmol/L = 16 ng/mL. To convert from pmol/L to ng/mL or μg/L, divide by 2.247. Data were missing for 2 subjects.
[3] Estimated by using Student's *t* test.
[4] Mean ± SD (all such values).

serum zinc concentrations and hemoglobin ($r = 0.26$, $P < 0.001$), children who are diagnosed with anemia should possibly be evaluated for zinc deficiency, a concept that requires more comprehensive population-based studies (23, 33). This relation between zinc concentration and hemoglobin was previously reported and is likely due to the common dietary sources of zinc and iron or the role of zinc in erythropoiesis (34–36). Preschoolers from low-income families who are previously known to have a relatively high prevalence of anemia were recruited (16, 17). The concentration of serum ferritin (≤35.95 pmol/L, or 16 ng/mL) used as the cutoff for low iron stores has been shown to be associated with absent bone marrow iron and identified 18% of the children as iron depleted (28). Generally, absent bone-marrow iron stores is considered to indicate depleted iron stores (22). The National Health and Nutrition Examination Surveys (NHANES) previously defined iron deficiency as an abnormal value for 2 of 3 of the iron indicators: serum ferritin, erythrocyte protoporphyrin, and transferrin saturation (37). However, in an effort to be consistent with the World Health Organization, the body iron model (which uses serum ferritin and soluble transferrin receptor) is currently used to define iron deficiency (38, 39). Because of restrictions by the IRB on the amount of blood that can be obtained from preschool children for research purposes, we limited the assessment of iron status to serum ferritin.

Anemia was also more common in the African American children (18.1%) than in the Hispanic children (7.6%). The prevalence of anemia in this population has been documented in national surveys (15). Zinc has been shown to modulate erythropoiesis, immunity to infectious diseases, and iron metabolism. In a state of zinc deficiency, these processes may be impaired (40, 41). Thus, it has been previously suggested that a discussion regarding the role of zinc in managing anemia is needed (40).

Race-ethnicity (African American) was associated with low serum zinc in this analysis. This finding is contrary to the findings of Schneider et al (23), who recruited predominately Hispanic children in California and found that race-ethnicity was not significantly associated with zinc status. However, in the current study, the population consisted of essentially equal numbers of Hispanic and African American children so that race-ethnicity between these groups could be evaluated as a risk factor. This selection process strengthened the findings. The prevalence of low zinc status was lower in this study than in that reported by Schneider et al because of the association between serum zinc and the time of blood collection (23, 25). This association was nonlinear, with peak serum zinc concentrations in the morning in both fasted and nonfasted individuals (42, 43). In the California study, the zinc status of the children was not adjusted to account for the time of the day and the physiologic variation of serum zinc (23, 42). This was done in the current study by obtaining blood samples only in the morning. It is essential that health care practitioners and investigators who intend to use serum zinc as a marker for zinc status account for the effect of diurnal variation on serum zinc when interpreting the results.

A cohort of low-income children of white descent was not available based on the demographics of patients attending the clinics from which patients were recruited. However, data on zinc, iron, and hemoglobin in such children, especially in comparison with African American and Hispanic children living in a similar urban metropolitan area, would be of interest.

Zinc deficiency has been attributed to the low intake of zinc-rich foods and to the low bioavailability of zinc, placing children from low-income groups at risk from such diets (41). Investigators previously reported slow physical growth, poor appetite, and diminished taste acuity among healthy infants and children with zinc deficiency (44, 45). We assessed the physical growth of the children and found that mean serum zinc concentrations were significantly lower among those with lengths less than the fifth percentile for age than among those with lengths greater than the fifth percentile for age, similar to previously reported data from Canada (45). Food intake was not a problem in either group, as documented in the food diaries, especially those of the African American children who consumed very high mean daily energy. However, the bioavailability of zinc in the foods consumed and the interactions between zinc and other components of the diet might be responsible for the zinc status among these children. Overall, the molar ratio of phytate to zinc calculated from the food diaries suggests that the zinc in the diets have average bioavailability, which could be improved by either the phytate reduction or zinc fortification of diets that are high in phytate (5, 6). Also, estimated nutrient intakes obtained from food diaries have well-known inaccuracies, which include day-to-day variation in the appetite of the child and the inaccuracies of documenting food intake appropriately. The major limitation with this method is that intake from consecutive days may not be independent of one another (29).

This study, conducted in a defined single metropolitan area in the southeastern United States, suggests that African American ethnicity is associated with low serum zinc concentrations among low-income children aged <5 y living in Atlanta. Our data also

TABLE 4
Frequency of exposure and relative odds ratios (and 95% CIs) among children with zinc deficiency (n = 34)[1]

Exposure variable and group	Children in group with zinc deficiency (<10.7 μmol/L)[2]	Relative multivariate-adjusted odds ratio (95% CI)[3]	P[4]
	%		
Age		1.60 (0.77, 3.31)	0.205
1–2 y	14.7		
3–5 y[5]	9.7		
Sex		1.16 (0.57, 2.38)	0.682
Female	13.0		
Male[5]	11.4		
Race-ethnicity		3.47 (1.51, 7.96)	0.003
African American	18.1		
Hispanic[5]	6.0		
WIC nutrition program participation		2.45 (0.91, 6.57)	0.076
Yes	14.4		
No[5]	6.4		
Insurance		2.31 (0.78, 6.85)	0.131
Government sponsored	13.7		
Other[5]	6.5		
Daycare attendance		1.42 (0.58, 3.49)	0.446
No	15.6		
Yes[5]	11.5		
Mother's educational level		1.34 (0.65, 2.76)	0.422
<12 y	14.3		
≥12 y[5]	11.0		
Father's educational level		0.85 (0.38, 1.92)	0.700
<12 y	10.3		
≥12 y[5]	11.9		
Single-parent households		1.79 (0.79, 4.03)	0.160
Yes	17.2		
No[5]	10.4		
Country of birth of parents		0.48 (0.15, 1.51)	0.208
At least one parent is foreign born	6.7		
Both parents born in USA[5]	13.2		
Hemoglobin		2.92 (1.24, 6.90)	0.015
<110 g/L	25.0		
≥110 g/L[5]	10.3		
Ferritin		1.02 (0.40, 2.62)	0.970
<35.95 pmol/L (16.0 ng/mL)	12.2		
≥35.95 pmol/L[5]	12.1		
Weight-for-age percentile		1.04 (0.29, 3.68)	0.955
<Fifth	12.5		
≥Fifth[5]	12.1		
Weight-for-height percentile		1.12 (0.24, 5.19)	0.885
<Fifth	13.2		
≥Fifth[5]	12.0		
Height-for-age percentile		1.13 (0.37, 3.46)	0.833
<Fifth	13.3		
≥Fifth[5]	12.1		
History of breastfeeding		1.01 (0.39, 2.61)	0.991
No	12.0		
Yes[5]	11.9		
Stool frequency per day		1.81 (0.87, 3.80)	0.115
≥3	17.5		
<3[5]	10.5		

[1] WIC, Women, Infants, and Children.
[2] Defined as serum zinc < 10.7 μmol/L. To convert to μg/dL, divide by 0.153.
[3] Estimated with logistic regression.
[4] Wald chi-square test.
[5] Reference group in relative odds risk assessment.

suggest the potential need for further study of the utility of evaluating anemic children for concomitant zinc deficiency. However, the cross-sectional design of the study prevented the establishment of a temporal relation between exposures and the outcome variables of interest; thus, we could not determine causality from our findings. The strengths of this study are the

TABLE 5
Multivariable predictors of zinc deficiency by multiple logistic regression[1]

Covariates	Point estimate	SE	Odds ratio (95% CI)	*P* (Wald test)
Age (*n* = 280)	0.053	0.229	1.112 (0.454, 2.725)	0.816
Sex (*n* = 280)	−0.153	0.219	0.736 (0.312, 1.734)	0.483
Race-ethnicity (*n* = 280)	−0.682	0.298	0.256 (0.080, 0.822)	0.022
Insurance (*n* = 280)	−0.192	0.348	0.682 (0.174, 2.670)	0.582
WIC nutrition program participation (*n* = 280)	−0.411	0.264	0.440 (0.156, 1.237)	0.119
Daycare (*n* = 280)	0.147	0.273	1.341 (0.460, 3.905)	0.591
Ferritin (*n* = 278)	0.142	0.553	1.329 (0.152, 11.618)	0.797
Stool output (*n* = 271)	−0.382	0.232	0.466 (0.187, 1.158)	0.100
Height-for-age percentile (*n* = 280)	−0.099	0.323	0.819 (0.231, 2.908)	0.758
Mother's educational level (*n* = 273)	−0.1923	0.217	0.680 (0.290, 1.594)	0.375
Single-parent household (*n* = 280)	−0.198	0.248	0.673 (0.454, 2.725)	0.424

[1] WIC, Women, Infants, and Children. Zinc deficiency defined as a serum zinc concentration <10.7 μmol/L. To convert to μg/dL, divide by 0.153.

large number of Hispanic and African American children who were recruited from a defined American urban area. Selection bias was not introduced, other than the order in which the children attended the clinic. The food diaries provided significant information about the diet of these children. Further research is needed to identify the potential causes of pediatric zinc deficiency, especially in the African American population, and to implement possible interventions that may include increased monitoring and/or supplementation strategies.

TABLE 6
Mean of 3-d intakes by race-ethnicity

Nutrient and race-ethnicity[1]	Mean ± SD	95% CI	*P*[2]
Calories (kcal/kg body wt)			<0.0001
Hispanic	92.1 ± 36.0	84.6, 99.6	
African American	119.7 ± 45.7	109.9, 129.4	
Carbohydrate (g)			0.336
Hispanic	187.8 ± 78.5.	171.5, 204.2	
African American	198.4 ± 66.9	184.1, 212.6	
Fat (g)			<0.001
Hispanic	42.9 ± 20.3	38.6, 47.1	
African American	56.9 ± 21.5	52.4, 61.5	
Total protein (g)			0.948
Hispanic	53.2 ± 21.3	48.8, 57.7	
African American	53.0 ± 17.5	49.3, 56.8	
Animal protein (g)			0.981
Hispanic	37.3 ± 15.7	34.1, 40.6	
African American	37.3 ± 14.7	34.2, 40.4	
Plant protein (g)			0.894
Hispanic	15.9 ± 8.3	14.2, 17.6	
African American	15.8 ± 6.3	14.4, 17.1	
Iron (mg)			0.06
Hispanic	11.2 ± 6.1	9.9, 12.5	
African American	12.9 ± 5.5	11.7, 14.1	
Copper (mg)			0.958
Hispanic	0.96 ± 0.87	0.77, 1.14	
African American	0.95 ± 0.67	0.81, 1.09	
Zinc (mg)			0.554
Hispanic	7.7 ± 3.1	7.1, 8.3	
African American	8.0 ± 2.9	7.4, 8.6	
Phytic acid (mg)			0.345
Hispanic	339.4 ± 200.8	297.6, 381.2	
African American	365.7 ± 167.9	329.9, 401.5	
Phytate:zinc molar ratio			0.106
Hispanic	4.55 ± 1.93	4.15, 4.94	
African American	5.06 ± 2.28	4.57, 5.54	

[1] *n* = 179 for total dietary data; *n* = 92 Hispanics and *n* = 87 African Americans.
[2] Calculated by using Student's *t* test.

This study provides important public health information that should be considered by health providers, federal agencies, and organizations responsible for the health of disadvantaged children in the United States. We found a previously unknown relatively high prevalence of zinc deficiency among low-income African American and Hispanic children in Atlanta, with a 4-fold greater prevalence in the African American cohort. These data can serve as a platform for larger population-based studies in the future to ascertain the true prevalence of zinc depletion and interrelations with iron deficiency anemia in similar populations of children and that should include a white cohort. As the new WIC food packages are implemented, there is also a potential need to identify the amount of zinc (and iron) supplied in the specific food brands offered and its bioavailability so that the zinc and iron nutriture of low-income American children receiving such support can be optimized.

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