The use of multiple logistic regression to identify risk factors associated with anemia and iron deficiency in a convenience sample of 12–36-mo-old children from low-income families[1–3]

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ABSTRACT

Background: The prevalence of iron deficiency (ID) anemia among preschool-age children remains relatively high in some areas across the United States. Determination of risk factors associated with ID is needed to allow children with identifiable risk factors to receive appropriate education, testing, and follow-up.

Objective: We aimed to evaluate risk factors associated with anemia and ID in a sample of children participating in or applying for the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC).

Design: The study was a cross-sectional study of a convenience sample of 12–36-mo-old children recruited from WIC clinics in 2 California counties ($n = 498$).

Results: Current WIC participation by the child and a greater rate of weight gain were negatively associated, and current maternal pregnancy was positively associated with anemia (hemoglobin $<$ 110 g/L at 12–$<$24 mo or $<$ 111 g/L at 24–36 mo) after control for age, sex, and ethnicity. Maternal WIC participation during pregnancy, child age, and the intake of $\geq$125 mL orange or tomato juice/d were negatively associated, and being male and living in an urban location were positively associated with ID ($\geq$2 of the following abnormal values: ferritin $\leq$ 8.7 μg/L, transferrin receptors $\geq$ 8.4 μg/mL, and transferrin saturation $\leq$ 13.2%).

Conclusions: Current WIC participation by the child and maternal WIC participation during pregnancy were negatively associated with anemia and ID, respectively. It is anticipated that the risk factors identified in this study will be included in the development of an educational intervention focused on reducing the risk factors for ID and ID anemia in young children. *Am J Clin Nutr* 2008;87:614–20.

KEY WORDS Anemia, iron deficiency, low-income status, child nutrition, Special Supplemental Nutrition Program for Women, Infants, and Children

INTRODUCTION

The prevalence of iron deficiency anemia (IDA) among preschool-age children remains relatively high in some areas across the United States; it is highest in certain racial-ethnic populations and in low-income populations (1). Healthy People 2010 reported iron deficiency (ID) prevalences of 17% in 1–2-y-old Mexican American toddlers and of 12% in low-income children (1). Because of the continued high prevalence of anemia, federal and state authorities have targeted low-income children to reduce the prevalence of anemia and ID in at-risk children through programs such as the Special Supplemental Nutrition Program for Women, Infants, and Children (WIC). WIC provides assistance for at-risk pregnant and lactating women, their infants, and their children from 1 to 5 y old by providing supplemental nutritious foods, nutrition education and counseling, and screening and referrals to other health, welfare, and social services. Eligibility for WIC enrollment requires participants to be at nutritional risk and at or below the 185% poverty threshold of the US Poverty Income Guidelines (2).

Iron status can progress from normal to low iron stores as a result of inadequate iron absorption or increased losses (ie, negative iron balance). Continued consumption of foods with low iron availability can lead to ID. If ID is not corrected, it may lead to IDA. ID without anemia has been associated with abnormal neurodevelopment (3). IDA may cause poor growth (4–7), low mental and motor test scores (5, 8), behavioral disturbances (9), altered central nervous system development and brainstem responses (10), and greater susceptibility to lead poisoning (11). Thus, the identification of children who are at risk is vital.

In a clinical setting, anemia in children is often determined by hemoglobin values alone. Screening for anemia will not identify all children with ID, because IDA is the last manifestation of poor iron status and because it has been shown to be a poor predictor of iron status (11, 12). ID can be identified by clinical assays of markers of iron status, such as serum ferritin, serum transferrin receptors, and transferrin saturation. Because of the expense, these tests are not typically used in clinical settings, which makes

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routine screening of all children unfeasible. However, selective routine screening for ID is recommended for high-risk populations (13–15). Determination of the risk factors that are associated with ID is needed to allow children with identifiable risk factors to receive education and further testing and to undergo follow-up. In addition, knowledge of these risk factors can aid in the development of successful education intervention programs to reduce the prevalence of ID and IDA. In the present study, we evaluated the risk factors for anemia and ID in12–36-mo-old children from low-income families in 2 California counties.

SUBJECTS AND METHODS

Study population

All study participants were recruited from California WIC waiting rooms between August 2000 and June 2002. The WIC clinics are located in Richmond (Contra Costa County) and Earlimart and Dinuba (Tulare County). Richmond is an urban community with a population of ≈99 216, and Earlimart and Dinuba have populations of 5881 and 16 849, respectively (16). Hispanics and Latinos compose ≈27%, 75%, and 88% of the populations of Richmond, Dinuba, and Earlimart, respectively (17). Median household income is approximately $44 000, $33 000, and $21 000, and 13%, 21%, 38% of families fall below the poverty level in Richmond, Dinuba, and Earlimart, respectively (17). The Richmond, Earlimart, and Dinuba clinics have ≈6800, 1600, and 3800 WIC participants, respectively.

Trained bilingual (English/Spanish), bicultural interviewers (1/county) approached all women in the WIC waiting rooms to recruit subjects for the study. Interviewers were instructed to introduce themselves, to briefly describe the study, and to ask women whether they had a child between 12 and 36 mo old. Men were excluded because the interview included questions pertaining to pregnancy. Subsequently, the interviewers asked whether the mothers would like to participate in the study. Approximately 673 women with children 12–36 mo old were approached, and 498 gave consent to participate in the study (≈74% of those who were eligible). To be eligible for the study, a mother could not have received information about IDA from a doctor or nurse, because such information could have influenced feeding behaviors (only one mother with a child in the target age range was excluded for this reason). Although recruitment occurred in WIC clinics, subjects were not required to be WIC participants at the time of recruitment.

Written informed consent was obtained from the women before their participation in the study. The University of California at Davis Institutional Review Board approved the study protocol.

Questionnaire

A risk factor questionnaire was developed in English and Spanish to collect demographic information; data on acute illness (eg, ear infection, fever, respiratory tract infection, vomiting, and diarrhea) at the time of study or in the previous month, maternal iron status, and pregnancy history; and dietary information, including infant feeding history and timing of introduction of foods that could affect iron nutriture. Select dietary variables were included in the risk factor questionnaire, based on a detailed dietary analysis in a group of children similar to the current sample (18). Previously, this questionnaire was validated in a low-income sample of 1–4-y-old children; energy and nutrient intakes were calculated by comparing a food-frequency questionnaire with multiple 24-h recalls. Pearson's correlations between estimates of nutrient intake obtained from 24-h recalls and the food-frequency questionnaire ranged from 0.40 to 0.47 for dietary iron, copper, and zinc in low-income children, nearly 80% of whom were Hispanic (18). Intakes of ready-to-eat cereals, beverages, commercially prepared infant foods, meat, poultry, fish, and legumes were measured by using typical portion sizes and frequency of intake in the past 30 d. Measuring cups, sippy cups, bottles, commercially prepared infant foods in jars, and food models were used to assist in estimating portion sizes. In addition to current beverage intake, duration of breastfeeding and the timing of the use of commercial infant formulas were calculated. The questionnaire also addressed the frequency and amount of intake, by the mother during her pregnancy or by the child, of nonfood substances such as clay, dirt, ice, paper, wax, and laundry starch.

Four additional questions addressed maternal and child participation in WIC, including current maternal WIC participation, maternal WIC participation during pregnancy, child's participation at any time in WIC, and child's current participation in WIC. These questions assessed WIC participation status from never or none (WIC nonrecipient) to current or active (WIC recipient).

Nutrition scientists, health educators, and University of California Cooperative Extension advisors reviewed the risk factor instrument for content validity. The instrument was then pilot-tested for clarity in a sample from the intended population before use in the study, and revisions were made accordingly. Recruitment for testing the instrument took place at the WIC Clinic of the Sacramento County Department of Health and Human Services.

Laboratory analysis

Between 0900 and 1900, venous blood samples were collected from the toddlers by phlebotomists in laboratories adjoining the respective WIC sites. Subjects were not required to fast. Serum samples were analyzed for ferritin (Coat-A-Count Ferritin immunoradiometric assay; Diagnostic Products, Inc, Los Angeles, CA), transferrin receptor (human transferrin receptor immunoassay kit; Ramco, Houston, TX), transferrin (nephelometric assay; Beckman Coulter, Brea, CA), iron (19) (Atomic Absorption Spectrophotometry 300; PerkinElmer, Boston, MA), and C-reactive protein (Nanorid radial immunodiffusion; The Binding Site, Birmingham, United Kingdom). Transferrin saturation was computed by using serum transferrin (sTf) and serum iron. Total iron–binding capacity (TIBC) was computed as TIBC = sTf/0.68 (19). The percentage of transferrin saturation (Tfsat) was subsequently computed as Tfsat (%) = (serum iron/TIBC) × 100 (20). Hemoglobin was measured with automated analyzers: the Max M (Coulter, Fullerton, CA) in Richmond, the Cell-Dyn 4000 (Abbott Laboratories, Abbott Park, IL) in Earlimart, and the Cell-Dyn 3200 (Abbott Laboratories) in Dinuba. Further details can be obtained from a previously published report (12).

Anemia was defined as hemoglobin < 110 g/L (12–<24-mo-old children) or < 111 g/L (24–36-mo-old children) (13, 21). ID was defined by using a multiple indicator model—ie, ≥2 of the following: ferritin ≤ 8.7 μg/L, transferrin receptors ≥ 8.4 μg/mL, and Tfsat $\leq 13.2\%$ (12).

Statistical analysis

Frequencies were used for prevalence data. We used chi-square tests to compare proportions and *t* tests to compare means

so as to examine risk factors associated with anemia or ID. Exploratory analyses were used to reduce the number of potential confounders. Any potential confounder that had at least a modest ($P < 0.20$) relation with both the outcome variable and the predictor of interest was included in later multivariate models. Multiple logistic regression (stepwise) models were developed, and odds ratios (ORs) were used to evaluate risk factors associated with anemia (anemia model) or ID (ID model). All children with hemoglobin values were selected for the anemia model. Children whose serum showed no evidence of hemolysis or elevated C-reactive protein (>10 mg/L) and who could be classified as iron deficient or iron sufficient were selected for the ID model. Only subjects with values for all variables were included in the analyses. The demographic variables age, sex, and ethnicity were forced into these stepwise models. Nonmonotonic continuous variables were dichotomized for multiple logistic regression analysis. Statistical significance was defined as $P \leq 0.05$. All statistical analyses were performed with SPSS software (version 10.0; SPSS Inc, Chicago, IL).

RESULTS

In total, 498 subjects were recruited for the study; of this group, 432 had successful blood draws. Thirty-three children had elevated serum C-reactive protein, 32 children had serum samples with evidence of hemolysis, and 3 children had both. In total, 359 subjects provided data for the anemia model and 344 subjects provided data for the ID model. Subject characteristics in the sample are compared in **Table 1** and **Table 2**.

Risk factors associated with anemia

Dietary factors were not found to be significantly associated with anemia in bivariate analysis. Current WIC participation by the child, rate of weight gain [(current weight − birth weight)/age (in mo)], and current maternal pregnancy were significantly associated with anemia in multiple logistic regression after control for age, sex, and ethnicity (**Table 3**). Anemic children were significantly less likely than nonanemic children to have participated in WIC (OR: 0.343; 95% CI: 0.128, 0.918). The mean rate of weight gain for anemic and nonanemic children was 376 ± 112 and 418 ± 102 g/mo, respectively ($P = 0.01$, Mann-Whitney U test). The rate of weight gain and the prevalence of anemia were inversely associated. Thus, children with a greater rate of weight gain were less likely to be anemic (Table 2). For example, a child in the 25th percentile (mean weight gain of 345 g/mo) would have an OR of 2 (95% CI: 1.1, 3.6) (OR: $1/e^{(128\ g \times -0.0055)} = 2.0$) for anemia when compared with a child in the 75th percentile (mean weight gain of 474 g/mo). Children with anemia were significantly more likely than nonanemic children to have a currently pregnant mother (OR: 3.492; 95% CI: 1.375, 8.869). Age, sex, and ethnicity (Hispanic or nonHispanic) were not significantly associated with anemia.

TABLE 1
Characteristics of 12–36-mo-old subjects from low-income families at risk of anemia[1]

	Child currently a WIC participant	
	Yes	No
Age (mo)	23.1 ± 6.6[2] (385)[3]	24.3 ± 7.1 (40)
Male (%)	53.5 (206 of 385)	42.5 (17 of 40)
Hispanic (%)[4]	93.5 (358 of 383)	97.5 (39 of 40)
Anemic (%)	10.1 (39 of 385)	20.0 (8 of 40)
Current weight (kg)[5]	12.5 ± 2.3 (373)	12.7 ± 2.4 (39)
Mean weight gain (g/mo)[6]	414 ± 104 (371)	405 ± 98[7] (38)
Mother is currently pregnant (%)[8]	8.9 (34 of 383)	10.3 (4 of 39)

[1] WIC, Supplemental Nutrition Program for Women, Infants, and Children. Hemoglobin <110 g/L (12–<24 mo) or hemoglobin <111 g/L (24–36 mo) (13, 21).
[2] $\bar{x}$ ± SD (all such values).
[3] n in parentheses (all such values).
[4] Hispanic or multiethnic including Hispanic.
[5] Weights were self-reported.
[6] Mean weight gain per month was defined as (current weight − birth weight)/age (in mo).
[7] Significantly different from children who are current WIC recipients, $P = 0.011$ (t test).
[8] Pregnancies were self-reported.

TABLE 2
Characteristics of 12–36-mo-old subjects from low-income families at risk of iron deficiency[1]

	Maternal participation in WIC during pregnancy	
	Yes	No
Age (mo)	23.1 ± 6.6[2] (280)[3]	25.2 ± 6.8 (70)
Male (%)	51.8 (145 of 280)	58.6 (41 of 70)
Hispanic (%)[4]	94.3 (263 of 279)	92.9 (65 of 70)
Iron deficient (%)	13.6 (38 of 280)	27.1[5] (19 of 70)
Urban (%)	39.3 (110 of 280)	57.1[5] (40 of 70)
Drinks ≥125 mL vitamin C juice/d (%)[6]	22.9 (64 of 279)	24.3 (17 of 70)
Mother is currently pregnant (%)[7]	8.6 (24 of 279)	10.3 (7 of 68)

[1] WIC, Supplemental Nutrition Program for Women, Infants, and Children. Iron deficiency defined as ≥2 of the following: ferritin ≤8.7 μg/L, transferrin receptors ≥8.4 μg/mL, and transferrin saturation ≤13.2% (12).
[2] $\bar{x}$ ± SD (all such values).
[3] n in parentheses (all such values).
[4] Hispanic or multiethnic including Hispanic.
[5] Significantly different from children whose mothers participated in WIC during pregnancy, $P < 0.01$ (chi-square test).
[6] Vitamin C juices: orange juice and tomato juice.
[7] Pregnancies were self-reported.

Risk factors associated with iron deficiency

All the responses regarding WIC participation were highly related. Thus, analyses were performed to select the best predictor of ID, and maternal WIC participation during pregnancy was selected. Of the factors examined in multivariate analysis, age, sex, maternal WIC participation during pregnancy, current maternal pregnancy, urban location, and juice (orange and tomato) intake were associated with ID in multiple logistic regression (**Table 4**). Younger children were more likely to be iron deficient: there was a 5% reduction in the odds of ID for each 1-mo increase in age. Boys were significantly more likely than females to be iron deficient (OR: 1.960; 95% CI: 1.052, 3.650). Children whose mothers did not participate in WIC during their pregnancy had an OR for ID of 2.6 (OR: 1/0.387 = 2.6; 95% CI: 1.3, 5.1).

TABLE 3
Regression coefficients and odds ratios (and 95% CIs) for risk factors associated with anemia after adjustment for potential confounding factors in children 12–36 mo old from low-income families[1]

Factor	β value	P value	Odds ratio (95% CI)
Age (mo)	−0.011	0.717	0.989 (0.929, 1.053)
Male[2]	−0.092	0.793	0.913 (0.455, 1.832)
Hispanic[3]	0.222	0.776	1.249 (0.263, 5.931)
Current WIC participation by the child[4]	−1.070	0.030	0.343 (0.128, 0.918)
Weight gain (1 g/mo)[5]	−0.005	0.017	0.995 (0.990, 0.999)
Mother is currently pregnant[6]	1.251	0.007	3.492 (1.375, 8.869)

[1] WIC, Supplemental Nutrition Program for Women, Infants, and Children. Hemoglobin <110 g/L (12–<24 mo) or hemoglobin <111 g/L (24–36 mo) (13, 21). $R^2 = 0.048$; $n = 359$; significance level of model, $P = 0.008$.
[2] Male = 1; female = 0.
[3] Hispanic or multiethnic including Hispanic = 1; non-Hispanic = 0.
[4] Child is currently participating in WIC = 1; child is not currently participating in WIC = 0.
[5] Weight gain is defined as (current weight − birth weight)/age (in mo). Weights were self-reported.
[6] Mother is currently pregnant = 1; mother is not currently pregnant = 0. Pregnancies were self-reported.

As was found in the anemia model, children with pregnant mothers were significantly more likely to be iron deficient than were children whose mothers did not report being pregnant. Children living in urban locations were less likely than were children living in rural locations to be iron deficient.

Several dietary factors—including duration of iron supplementation during pregnancy (negative), duration of formula intake (negative), current use of a bottle (positive), intake of meat (negative), and intake of juice (tomato, orange, or both) (negative)—were found to be associated (positively or negatively) with ID in bivariate analysis. However, only juice intake remained significant in multivariate analysis. The mean juice intake in iron-sufficient children was 95 ± 176 mL/d, and that in iron-deficient children was 47 ± 97 mL/d ($P = 0.022$, Mann-Whitney U test). The juice intake variable was dichotomized to <125 and ≥125 mL/d, on the basis of the American Academy of Pediatrics recommendation to limit juice intake to 4–6 oz/d (118–177 mL/d) for 1–6-y-olds (23). Children consuming ≥125 mL juice/d were less likely to be iron deficient than were children consuming <125 mL juice/d (OR: 0.360; 95% CI: 0.154, 0.841). Ethnicity (Hispanic or non-Hispanic) was not associated with ID.

DISCUSSION

Several risk factors were found to be associated with anemia and ID. Notably, child or maternal WIC participation was negatively associated with anemia and ID. Specifically, children currently receiving WIC benefits were less likely to be anemic than were children whose mothers were applying for WIC benefits. This is consistent with the finding that the prevalence of anemia was greater in children before enrollment in the WIC program than in those already enrolled (22) and is also consistent with the decline in anemia in several states in the United States (23). Studies also suggest an improvement in nutrient intake in young children, specifically those served by WIC (24–26). The nonrecipients were not referred to the WIC program for ID or anemia, because the selection criteria omitted any child whose mother was told about ID or IDA by a doctor or nurse. Control for previous diagnosis of anemia or ID did not change the significance, in multivariate analysis, of a child's current WIC participation. It must be appreciated that the association of current WIC participation likely was diluted because sampling occurred only at WIC clinics. A mother whose child is participating in WIC

TABLE 4
Regression coefficients and odds ratios (and 95% CIs) for risk factors associated with iron deficiency after adjustment for potential confounding factors in children 12–36 mo old from low-income families[1]

Factor	β value	P value	Odds ratio (95% CI)
Age (mo)	−0.053	0.028	0.949 (0.905, 0.994)
Male[2]	0.673	0.034	1.960 (1.052, 3.650)
Hispanic[3]	1.085	0.173	2.958 (0.621, 14.096)
WIC participation during pregnancy[4]	−0.950	0.006	0.387 (0.196, 0.763)
Urban[5]	0.731	0.024	2.077 (1.100, 3.921)
Consumes >125 mL vitamin C juice/d[6]	−1.021	0.018	0.360 (0.154, 0.841)
Mother is currently pregnant[7]	0.920	0.040	2.509 (1.044, 6.027)

[1] WIC, Supplemental Nutrition Program for Women, Infants, and Children. Iron deficiency defined as ≥2 of the following: ferritin ≤8.7 μg/L, transferrin receptors ≥8.4 μg/mL, and transferrin saturation ≤13.2% (12). $R^2 = 0.079$; $n = 344$; significance level of model, $P < 0.001$.
[2] Male = 1; female = 0.
[3] Hispanic or multiethnic including Hispanic = 1; non-Hispanic = 0.
[4] Mother participated in WIC during pregnancy = 1; mother did not participate in WIC during pregnancy = 0.
[5] Urban = 1; rural = 0.
[6] Vitamin C juices: orange juice and tomato juice. Consumes <125 mL vitamin C juice/d = 1; consumes ≥125 mL vitamin C juice/d = 0.
[7] Mother is currently pregnant = 1; mother is not currently pregnant = 0. Pregnancies were self-reported.

may be more proactive with respect to the child's health and well-being than would a mother whose child does not participate in WIC. Thus, the apparently protective effect of WIC participation is likely to be even lower in this study sample than would be expected had children outside of WIC clinics been recruited.

Maternal participation in WIC during pregnancy also was negatively associated with pediatric ID. WIC participation during pregnancy could positively influence a child's iron status through increased intake of iron during gestation and infancy. However, prenatal supplement use, birth weight, and weeks of gestation were not significantly associated with pediatric iron status in the 12–36-mo-old children. Although we did not measure gestational dietary intake, iron intake during pregnancy is unlikely to be the factor contributing to positive iron status in toddlers. It is reasonable to suggest that women who participate in WIC during pregnancy may have more knowledge of nutrition and iron-rich foods than may women in the same socioeconomic class who do not participate. Thus, because certain risk factors associated with poor iron status may have been reduced by WIC participation, the prevalence of anemia and ID may be lower in the study sample and higher in a comparable population not participating in WIC.

With the exception of the intake of orange and tomato juices, no association was observed between beverage or food intake and anemia or ID. Children consuming less juice were more likely to be iron deficient. It should be noted that the bioavailability of iron is enhanced by vitamin C (27, 28). Current recommendations to limit juice intake are based on limiting dietary simple sugars, which can contribute to dental carries, and on reducing the displacement of more nutrient-dense foods for infants and children (29). Thus, despite the relation between juice and ID observed in the present study, it may not be prudent for children to consume more than the current recommendation for 1–6-y-olds—ie, 4–6 oz juice/d (118–177 mL/d) (30).

In these analyses, cow milk intake was not associated with ID, hemoglobin, ferritin, transferrin receptor, or Tfsat (data not shown). Reports in the literature regarding the association of cow milk intake with iron status are conflicting. It has been suggested that consuming large quantities of cow milk may predispose children to ID because milk has a low iron content and may displace iron-rich foods (31), or because it contains substances that inhibit iron absorption, such as calcium (32). Several studies have shown negative associations between milk intake by children and serum ferritin (33–36) and the ratio of transferrin receptor log to ferritin (37). The intake of calcium, milk, or cheese has been shown to reduce iron absorption in single-meal studies (32). However, consistent with results from our study, Ames et al (38) reported that high- and low-calcium diets resulted in similar iron incorporation into red blood cells in 3–5-y-old children.

Rapid growth during infancy, combined with a marginal supply of dietary iron, is associated with depletion of iron stores (39). We did not find an association between the rate of weight gain and the iron status in our sample; in contrast, children with a lower rate of weight gain had a greater risk of anemia. Others have found positive weight gain with WIC participation. Black et al (40) found that WIC participants were less likely to be underweight than were those who were not participating in WIC because they had access problems. Consistent with the findings of the present study, Emond et al (41) reported that weight gain was positively associated with hemoglobin and negatively associated with ferritin in 8-mo-old infants. A study using Massachusetts WIC program data found that weight was positively associated with hemoglobin concentration at age 1 y (baseline analysis) but negatively associated with hemoglobin concentration at age 2 y (follow-up analysis) (42). There is some evidence that ID is related to inadequate growth rates and that these rates can be improved with iron supplementation (4, 6, 7). Thus, in the sample in the present study, it is possible that low hemoglobin concentrations limited the rate of weight gain, which would explain the association observed. Additional research is needed to clarify the 2-way relation between iron status and child growth.

Children of mothers who were currently pregnant had a higher risk of anemia and ID than did children of nonpregnant mothers. In contrast, the number of living children, the number of children living in the household, and the number of siblings diagnosed with anemia were not associated with a higher risk of anemia or ID. It is conceivable that maternal behavioral factors not measured in this study contribute to a toddler's likelihood of being anemic or iron deficient. For example, many pregnant women experience pregnancy-related symptoms such as fatigue, nausea, heartburn, and odor intolerance (43, 44), and, because of these physiologic changes, pregnant women may alter their usual dietary habits and those of their families.

We found that boys were more likely to be iron deficient than girls. Forcing the variables birth weight and current weight (or relative weight change) into the logistic regression model did not alter this relation. Sex differences in iron status in infants and toddlers have been reported by others (31, 36, 45, 46). In a 2-site study conducted in Honduras and Sweden in children 4–9 mo old, there were significant sex differences in plasma ferritin and transferrin receptor after control for birth weight, postnatal weight gain, and other factors (46). The sex difference in transferrin receptor could be explained by a true difference in iron status, but the difference in plasma ferritin could not, and, for that reason, the authors suggested that sex-specific cutoffs for low plasma ferritin may be needed. In a previous report from the same study from which the sample for the present study was drawn, we stated that the prevalence of low serum ferritin was significantly greater in boys than girls (12). There may be a need for sex-specific ferritin cutoffs in 12–36-mo-old children; however, that need could not be established in the present study.

We controlled potentially confounding variables in regression analysis, and only significant variables remained in the parsimonious models. It is possible that we did not include a confounding variable in the analysis, which may bias the findings. However, the survey instrument was extensive. Examples of confounding variables not found to be significant in the final models include income, prenatal care, breastfeeding, and use of supplements. We did not assess the number of child health care visits. There are several possible reasons that a discrepancy exists between risk factors found to be associated with anemia and ID. It is possible that the anemia was due to factors other than poor iron status. We previously reported the prevalences of anemia, ID, and IDA to be 11.1%, 16.2%, and 3.4%, respectively (12). With the use of receiver operating characteristic curves, the sensitivity of anemia in predicting ID was low in that sample (12). Another limitation of that study was the low R^2 values (≈5% and 9%) that explained a small portion of the variation in anemia and ID, respectively. Random measurement errors also may have contributed to the low R^2 values. In addition, the results are not generalizable to a

larger pediatric population. The present study is based on a convenience sample of children in which WIC participants and nonparticipants recruited at WIC clinics were compared. In a multisite study, researchers reported differences in health outcomes between nonparticipants who perceive they do not need WIC services and nonparticipants who do not have access to WIC services (40). Despite these limitations, this study shows the importance of identifying factors associated with anemia and ID that may be addressed by the WIC program for educational purposes.

In summary, we found that current child WIC participation and maternal WIC participation during pregnancy were negatively associated with anemia and ID, respectively. We used multiple logistic regression to determine associations between risk factors and anemia or ID, after control for confounding variables. Our results suggest that WIC participation was protective against ID in the study sample. However, the current study was cross-sectional, and temporal relations between "exposures" and the outcome variables of interest cannot be established; thus, causality cannot be determined. Studies designed to measure the incidence of new cases may determine causality. Our findings cannot be generalized to all low-income children residing in California, because the sample was largely composed of Latino or Hispanic children. However, ≈47% of California children (<5 y old) around the year 2000 were Latino or Hispanic, regardless of race (47). It is anticipated that the risk factors identified in the current study will be considered in the development of an educational intervention focused on reducing the risk factors for ID and IDA among young children.

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