Iron Deficiency in Massachusetts Communities: Socioeconomic and Demographic Risk Factors among Children

James D. Sargent, MD, Therese A. Stukel, PhD, Madeline A. Dalton, PhD, Jean L. Freeman, PhD, and Mary Jean Brown, RN

ABSTRACT

Objectives. This study examined the association between community rates of iron deficiency in children and sociodemographic characteristics of Massachusetts communities.

Methods. Between April 1990 and March 1991, 238 273 Mssachusetts children 6 through 59 months of age were screened; iron deficiency was defined as an erythrocyte protoporphyrin concentration of 0.62 μmol/L or higher and a blood lead level of less than 1.2 μmol/L. Sociodemographic data were obtained from the 1990 US Census.

Results. Five percent of communities had iron deficiency rates greater than 13.9 per 100 children screened. Iron deficiency rate was positively associated with proportion of Southeast Asians (odds ratio [OR] = 1.10, 95% confidence interval [CI] = 1.08, 1.12), proportion of Hispanics (OR = 1.008, 95% CI = 1.002, 1.013), and high school incompletion (OR = 1.028, 95% CI = 1.020, 1.035). Similarly, an examination of three Massachusetts cities indicated that the iron deficiency rate was higher for children with Southeast Asian (relative risk [RR] = 3.6, 95% CI = 3.3, 3.8) and Hispanic (RR = 1.6, 95% CI = 1.5, 1.8) surnames than for all other children.

Conclusions. Wide variation exists in iron deficiency rates for children in Massachusetts communities. Community iron deficiency was associated with low socioeconomic status and high proportions of Southeast Asians and Hispanics. (*Am J Public Health.* 1996;86:544–550)

Introduction

Widespread supplementation of the American diet with iron has reduced the prevalence of iron deficiency dramatically in middle-class populations of children.[1,2] However, recent data indicate a causal relationship between iron deficiency anemia in early childhood and developmental impairment,[3] making the diagnosis of iron deficiency important. A thorough understanding of the prevalence of and risk factors for iron deficiency in the pediatric population is necessary in order to target high-risk groups of children for screening.

To learn more about community risk factors for iron deficiency in young children, we examined state screening data from Massachusetts. We performed a population-based analysis at the community level and examined the relationship between the prevalence of iron deficiency in children 6 through 59 months of age and socioeconomic and ethnic characteristics of the community. We also examined individual data from three Massachusetts cities to determine whether the associations between iron deficiency and ethnicity observed at the community level hold at the individual level.

Methods

We examined screening data from a statewide lead poisoning prevention program between April 1990 and March 1991, a period in which erythrocyte protoporphyrin was used to screen for elevated blood lead. High penetration into the eligible population was achieved because of mandatory annual screening for lead poisoning in Massachusetts children. The study population comprised all Massachusetts children 6 through 59 months of age who had an assay for erythrocyte protoporphyrin that was either analyzed by or reported to the Massachusetts Childhood Lead Poisoning Prevention Program during the study period. This included results of screening samples obtained from private health care providers; health centers; hospitals; Women, Infants, and Children (WIC) programs; and state-sponsored childhood lead poisoning prevention programs.

Approximately 85% of all screening samples were analyzed at the state or Boston lead laboratories, where capillary blood was analyzed for erythrocyte protoporphyrin by hematofluorometry.[4] Other laboratories submitting erythrocyte protoporphyrin results to the state included those of Worcester, Boston, and the Boston Children's Hospital. Atomic absorption spectrophotometry was used in performing blood lead analysis for all capillary screening samples with erythrocyte protoporphyrin concentrations of 0.62 μmol/L (35 μg/dL) or higher.[5] Although the purpose of this screening program was the identification of children with lead poisoning, the primary cause of erythrocyte protoporphyrin elevation in the absence of blood lead elevation is iron deficiency.[4,6] We defined any child with both an erythrocyte protoporphyrin con-

James D. Sargent and Madeline A. Dalton are with the Department of Pediatrics, and Therese A. Stukel is with Community and Family Medicine (Biostatistics), Dartmouth Medical School, Hanover, NH. Jean L. Freeman is with the Department of Medicine, University of Texas Medical Branch, Galveston, Tex. Mary Jean Brown is with the Massachusetts Lead Poisoning Prevention Program, Boston.

Requests for reprints should be sent to James D. Sargent, MD, Pediatrics and Adolescent Medicine, Dartmouth-Hitchcock Medical Center, Lebanon, NH 03756.

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centration of 0.62 μmol/L or higher and a blood lead level of less than 1.2 μmol/L (25 μg/dL) as iron deficient. Children with erythrocyte protoporphyrin concentrations of 0.62 μmol/L or higher and blood lead levels 1.2 μmol/L or higher were assumed to have erythrocyte protoporphyrin elevation as a result of their lead exposure and were included in the iron-sufficient group. This definition is consistent with results from a study conducted by Yip et al.[7] in which an erythrocyte protoporphyrin concentration of 0.62 μmol/L or higher identified iron deficiency (serum ferritin ≤ 15 μg/L) with sufficient sensitivity (88%) and specificity (90%) to allow its use as a screening test. The threshold value for erythrocyte protoporphyrin (0.62 μmol/L) is the upper limit of 95% confidence (mean plus two standard deviations) for erythrocyte protoporphyrin in iron-replete children (serum ferritin ≥40 μg/L).[7] Therefore, since approximately 3% of iron-replete children can be expected to have an erythrocyte protoporphyrin concentration of 0.62 μmol/L or more, we also analyzed these data using the stricter definition for iron deficiency: an erythrocyte protoporphyrin concentration of 0.9 μmol/L (50 μg/dL) or higher and a blood lead level of less than 1.2 μmol/L.

The cutoff for blood lead elevation in this definition was chosen to balance factors that would result in underestimation of iron deficiency prevalence with those that would result in its overestimation. There is no relationship between blood lead and erythrocyte protoporphyrin below a blood lead level of 17 μg/dL, the threshold at which the most sensitive children begin to experience elevated erythrocyte protoporphyrin as a result of lead.[8] Thus, by including children with erythrocyte protoporphyrin elevation from blood lead in the 17 to 24 μg/dL range as iron deficient, we were falsely increasing our estimates of the prevalence of iron deficiency. However, iron deficiency and lead exposure often occur in the same children.[9,10] For instance, in a study of 109 children screened for lead poisoning,[11] 52% had blood lead levels of 1.5 μmol/L or higher, 40% had evidence of iron deficiency, and 22% had both. Exclusion from the numerator of some iron-deficient children with both elevated erythrocyte protoporphyrin concentrations and blood lead levels of 1.2 μmol/L or higher would tend to balance our estimates.

For this study, Massachusetts communities corresponded to 1990 US Bureau of the Census definitions for minor civil divisions, which include small towns and cities. There are 351 minor civil divisions in Massachusetts. Population counts and sociodemographic characteristics of Massachusetts communities were obtained from the 1990 US Bureau of the Census STF 3A tape. Two communities were excluded from the study, one with a zero population count in 1990 for children 6 through 59 months of age and one because only two children were screened during the study period, one of whom was iron deficient.

The screening rate was equal to the number of children screened divided by census counts of the number of children living in a geographic area. The crude rate of iron deficiency was computed by dividing the number of children with iron deficiency by the number screened in a geographic area. We first determined the crude rate of iron deficiency for the state overall and by age (newborn through 12, 13 through 36, 37 through 59 months). We then computed the crude rate of iron deficiency for each of the 349 Massachusetts communities and reported quartiles and the 95th percentile for these rates. We also examined the crude rate of iron deficiency according to community-specific covariates that described the screening rate, demographics (percentage of population Black, Hispanic, Southeast Asian, or Chinese), and socioeconomic indicators (percentage of population 18 years of age or older that did not complete high school). All census-based measures of socioeconomic status (percentage of population 18 years of age or older that did not complete high school, percentage of female-headed households, percentage of households on public assistance, percentage of children under 5 years of age in poverty, median family income) were highly correlated; we chose educational status as a single measure of socioeconomic status because it had the highest bivariate association with iron deficiency rates. Education is also a social class measure reported to be closely associated with other health outcomes in children.[12,13] Inclusion of other variables did not increase the predictive validity of the model and resulted in instability due to multicollinearity. The community screening rate was computed by dividing the number of children screened by the total number of children living in the geographic area, as determined by the 1990 US census.

We used multiple logistic regression to assess the association between the community rate of iron deficiency and community-specific covariates. These analyses used community as the unit of analysis, weighted by the number of children screened in each community.[14,15] The response was the proportion of children iron deficient in each community; all covariates were analyzed as continuous variables. Point estimates and confidence intervals (CIs) for the odds ratios were obtained by transforming the corresponding regression parameters. For example, this procedure produced an estimate of the odds ratio of iron deficiency for each 1% increase in the proportion of individuals in a community who were Southeast Asian, adjusted for the effect of the other covariates in the model. We incorporated variance overdispersion in the estimates of standard error to account for clustering of iron deficiency within communities.[15,16] The effect of clustering was thus to increase the width of the usual binomial-based confidence intervals. All analyses were performed with the "glm" procedure in the S statistical software package.[17]

To investigate the association between ethnicity and iron deficiency that was observed in the community model at the individual level, we examined individual screening data from three Massachusetts cities (Lowell, Lynn, Revere) where census data indicate high proportions of Hispanics (10.7%) and Southeast Asians (5.4%). Because individual data on race, ethnicity, and socioeconomic status were not collected during screening, children in these communities were classified as Hispanic, Southeast Asian, or other on the basis of first and last name. Persons familiar with the names of these ethnic groups classified each record retrospectively as Southeast Asian surname, Hispanic surname, or other. The names and ethnic codes were then reviewed by another group of individuals. If there was any uncertainty as to the ethnicity of the name, the child was coded as other. Unadjusted relative risks for iron deficiency with 95% confidence intervals were determined in these two ethnic groups in comparison with all other children (non-Hispanic, non–Southeast Asian) by means of relative risk regression methods.[18] Finally, the distribution of erythrocyte protoporphyrin was compared for the three ethnic categories in these cities.

Results

During the study period, 238 273 children in 349 Massachusetts communi-

TABLE 1—Rate of Iron Deficiency among Massachusetts Children 6 through 59 Months of Age, April 1990 to March 1991

Age Range, mo	No. Screened	Iron Deficiency Rate[a] (per 100)	EP ≥ 0.9 μmol/L[b] (per 100)
6–12	36 715	15.0	3.3
13–36	107 348	10.0	2.8
37–59	94 210	4.8	1.0
Total	238 273	9.0	2.2

Note. EP = erythrocyte protoporphyrin.
[a]Number of children with both EP concentrations of 0.62 μmol/L (35 μg/dL) or higher and blood lead levels of less than 1.2 μmol/L (25 μg/dL) per 100 children screened.
[b]Number of children with both EP concentrations of 0.9 μmol/L (50 μg/dL) or higher and blood lead levels of less than 1.2 μmol/L (25 μg/dL) per 100 children screened.

TABLE 2—Distribution for the Community Rate of Iron Deficiency among Children 6 through 59 Months of Age in 349 Massachusetts Communities

Percentile	Iron Deficiency Rate[a] per 100 Screened (95% CI[c])	EP ≥ 0.9 μmol/L[b] per 100 Screened (95% CI[c])
25th	5.3 (4.9, 5.5)	0.6 (0.4, 0.8)
50th	6.9 (6.5, 7.3)	1.3 (1.2, 1.4)
75th	9.0 (8.3, 9.5)	2.0 (1.8, 2.2)
95th	13.9 (12.5, 14.7)	5.0 (3.9, 6.1)

Note. EP = erythrocyte protoporphyrin; CI = confidence interval.
[a]Number of children 6 through 59 months of age with both EP concentrations of 0.62 μmol/L (35 μg/dL) or higher and blood lead levels of less than 1.2 μmol/L (25 μg/dL) per 100 children screened.
[b]Number of children with both EP concentrations of 0.9 μmol/L (50 μg/dL) or higher and blood lead levels of less than 1.2 μmol/L (25 μg/dL) per 100 children screened.
[c]Calculated by the binomial-based method.

ties were screened for lead poisoning, an overall screening rate of 58.0%. Screening rates were highest in the 1-year to 2-year age group (61.8%) and lowest in the less than 1 year age group (50.2%). Community screening rates varied from 3.7% to 97.2%; nevertheless, 284 of 350 Massachusetts communities fell within the same tercile (33% to 67%). The community screening rate was directly associated with the census counts of the number of children 6 through 59 months of age living in a community and the percentage of screened children with blood lead levels of 1.2 μmol/L or higher. When these variables were controlled, none of the independent variables included in the logistic model were significantly related to the community screening rate. The association between these variables and the community screening rate is, in part, the result of state-operated childhood lead poisoning screening programs that supplement provider-driven screening in urban areas with high rates of lead exposure.

Two thirds of all communities had no children with blood lead levels of 1.2 μmol/L or higher; the 99th percentile for the case identification rate for such levels was 1.6%, and there were four outliers with case identification rates between 2% and 4%. The characteristics of the screened population and the incidence of lead poisoning have been fully discussed in another paper.[19]

The overall rate of iron deficiency was 9 per 100 children screened, with the highest rate in the 0- to 12-month age range (15 per 100) and the lowest (4.8 per 100) in the 37- to 59-month age range (Table 1). When the more restrictive definition of iron deficiency was used, rates were about one fifth as high in all age groups. Iron deficiency rates for Massachusetts communities (Table 2) ranged from 0 to 29 per hundred. Twenty-five percent of communities had rates of iron deficiency at or below 5.3 per hundred, while 5% had rates greater than or equal to 13.9 per hundred.

Table 3 reports iron deficiency rates according to screening rate, ethnicity, and socioeconomic characteristics of Massachusetts communities. Communities where more than 4% of the population was Southeast Asian had rates of iron deficiency double those in communities where less than 1% of the population was Southeast Asian. Increases in the iron deficiency rate were also seen in communities with large Hispanic and Black populations and in communities with higher percentages of adults who did not complete high school. After adjustment for all community-specific characteristics, the odds of iron deficiency were still strongly related to Southeast Asian ethnicity, Hispanic ethnicity, and educational attainment. The iron deficiency rate increased by a factor of 1.10 (95% CI = 1.08, 1.12) for each 1% increase in the Southeast Asian population and by 1.008 (95% CI = 1.002, 1.013) for each 1% increase in the Hispanic population. The rate of iron deficiency increased by a factor of 1.021 (95% CI = 1.015, 1.026) for each 1% increase in the number of adults who did not complete high school. The findings were similar when the more restrictive definition of iron deficiency was used, except that the odds of iron deficiency were then also related to the percentage of the population who were Black (P = .01) (Table 3). Finally, the findings were similar when the community prevalence of blood lead elevations of 1.0 μmol/L (20 μg/dL) or more was included as an independent variable (the coefficient for percentage Southeast Asian dropped from 1.10 to 1.04 [95% CI = 1.08, 1.12] with inclusion of this variable) and when the analysis was restricted to children 13 to 48 months of age (data not shown). The effect of clustering on the analysis was to increase the width of the usual binomial confidence intervals for the odds of iron deficiency by a factor of about 2, indicating that iron deficiency rates are related to other unmeasured characteristics of communities.

Of 11 988 individual records examined in Lowell, Lynn, and Revere, 1183 (9.9%) had Southeast Asian surnames and 1884 (15.7%) had Hispanic surnames (Table 4). Thirty-nine percent of children with Southeast Asian surnames and 17.9% of children with Hispanic surnames were iron deficient, as compared with 11.6% of the rest of the population. In addition, a higher proportion of Southeast Asian children had blood lead elevations of 1.2 μmol/L or more (3.7%, as compared with 1.2% in the other groups; P = .05). The

TABLE 3—Crude Rate of Iron Deficiency and Adjusted Odds Ratio for Iron Deficiency in Massachusetts Children 6 through 59 Months of Age, by Demographic, Socioeconomic, and Racial/Ethnic Characteristics of the Communities

Characteristic	No. Children Screened	Iron Deficiency[a] Rate (per 100 Screened)	Iron Deficiency[a] Adjusted OR[c] (95% CI)	Iron Deficiency[a] P	Iron Deficiency: Restrictive Definition[b] Rate (per 100 Screened)	Iron Deficiency: Restrictive Definition[b] Adjusted OR[c] (95% CI)	Iron Deficiency: Restrictive Definition[b] P
Screened, %							
≤50	33 606	8.5	0.997 (0.994, 1.000)	.075	1.8	1.0 (0.995, 1.004)	.94
51–60	85 191	8.7			1.9		
61–70	55 620	8.4			1.9		
>70	63 794	10.2			2.8		
Population ≥ 18 years of age not completing high school, %							
≤10	52 806	7.1	1.021 (1.015, 1.026)	<.0001	1.4	1.028 (1.020, 1.035)	<.0001
10.1–15	53 933	6.8			1.3		
15.1–20	80 585	9.0			2.3		
>20	50 887	13.2			3.6		
Population Southeast Asian, %							
0	52 722	7.1	1.102 (1.081, 1.123)	<.0001	1.4	1.151 (1.125, 1.177)	<.0001
0.1–1	127 282	8.7			1.9		
1.1–2	43 716	9.3			2.7		
2.1–4	9 046	15.9			4.6		
>4	5 445	19.0			6.7		
Population Hispanic, %							
0–2	116 451	7.3	1.008 (1.002, 1.013)	<.01	1.5	1.008 (1.001, 1.015)	<.01
2.1–3	14 380	7.8			1.7		
3.1–5	18 209	10.2			2.2		
>5	89 171	11.2			3.1		
Population Black, %							
0	6 229	6.5	1.003 (0.997, 1.009)	.31	1.4	1.011 (1.003, 1.019)	<.01
0.1–5	174 194	8.4			1.9		
5.1–10	15 350	14.0			3.6		
>10	42 438	10.0			2.7		
Population Chinese, %							
0	27 298	7.4	1.027 (0.989, 1.065)	.16	1.5	1.013 (0.959, 1.071)	.63
0.1–2	170 399	9.1			2.2		
>2	40 514	9.5			2.4		

Note. OR = odds ratio; CI = confidence interval.
[a]Erythrocyte protoporphyrin ≥ 1.62 μmol/L and blood lead < 1.2 μmol/L.
[b]Erythrocyte protoporphyrin ≥ 0.9 μmol/L and blood lead < 1.2 μmol/L.
[c]For each percentage increase in the community variable.

relative risks of iron deficiency in both ethnic groups were significantly higher than those in the rest of the population: 3.4 (95% CI = 3.2, 3.7) for children with Southeast Asian surnames and 1.5 (95% CI = 1.4, 1.7) for children with Hispanic surnames (Table 4). Similar findings applied when the more stringent definition of iron deficiency (erythrocyte protoporphyrin ≥ 0.9 μmol/L and blood lead < 1.2 μmol/L) was used: the relative risks were 6.8 (95% CI = 6.0, 7.7) for children with Southeast Asian surnames and 1.6 (95% CI = 1.3, 1.9) for children with Hispanic surnames in comparison with the rest of the population. Excluding children whose erythrocyte protoporphyrin elevation could be attributed to lead poisoning (erythrocyte protoporphyrin

TABLE 4—Blood Lead Elevation, Iron Deficiency, and Relative Risk of Iron Deficiency, by Ethnicity, in 11 988 Children 6 through 59 Months of Age in Cities (Lowell, Lynn, and Revere) with Large Hispanic and Southeast Asian Populations

Ethnicity	No.	Normal, No. (%)	Elevated Blood Lead,[a] No. (%)	Iron Deficient,[b] No. (%)	Relative Risk of Iron Deficiency[b] (95% CI)
Other	8921	7780 (87.2)	107 (1.2)	1034 (11.6)	1.0 (Reference)
Hispanic surname	1884	1522 (80.8)	24 (1.3)	338 (17.9)	1.5 (1.4, 1.7)*
SE Asian surname	1183	682 (57.7)	44 (3.7)	457 (38.6)	3.4 (3.2, 3.7)*

[a]Children with either capillary or venous blood lead levels of 1.2 μmol/L (25 μg/dL) or more.
[b]Children with both erythrocyte protoporphyrin concentrations of 0.62 μmol/L (35 μg/dL) or higher and blood lead levels of less than 1.2 μmol/L (25 μg/dL).
*$P < .0001$.

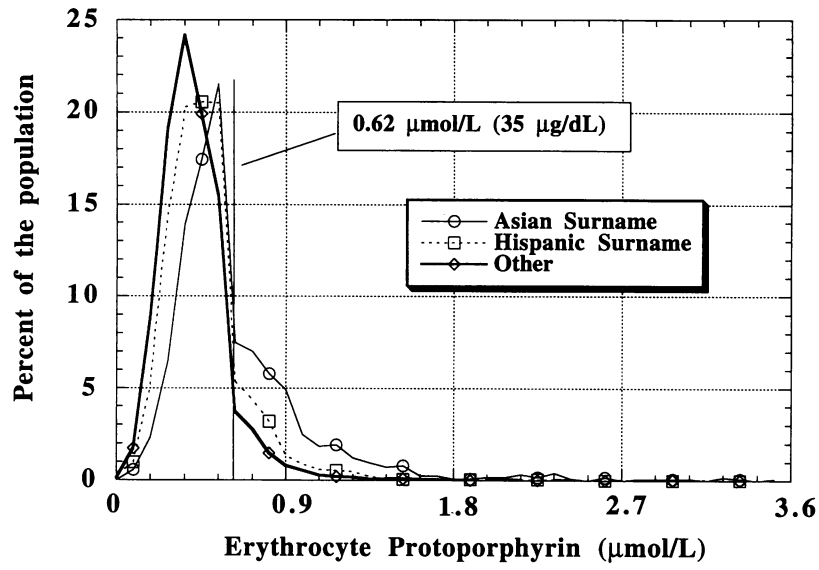

FIGURE 1—Distribution of erythrocyte protoporphyrin in three ethnic groups from three Massachusetts cities.

≥0.62 μmol/L *and* blood lead ≥1.2 μmol/L) from the analysis had little effect on the relative risk of iron deficiency.

As shown in Figure 1, the distribution of erythrocyte protoporphyrin for Hispanic and Southeast Asian children was more highly skewed to the right than the distribution for the "other" group; thus, a higher proportion of Southeast Asian and Hispanic children were found throughout the "iron deficient" range (erythrocyte protoporphyrin ≥0.62 μmol/L).

Discussion

Twenty years ago, iron deficiency and iron deficiency anemia were common conditions of early childhood.[20,21] However, reports have documented a decline in the prevalence of iron deficiency in US children,[1,22] probably as a result of the widespread fortification of infant foods with iron.[23] In addition, evidence suggests that participation in nutritional supplementation programs such as the WIC program reduces the incidence of iron deficiency in poor populations.[24-28]

Rates of iron deficiency vary widely by community in Massachusetts. Despite widespread nutritional supplementation, iron deficiency still affects more than 10% of young children in some communities. On the other hand, 20% of Massachusetts communities have little or no iron deficiency in young children. Our findings should be interpreted in the context of the limitations of a study using only one measure to define iron deficiency. There is no single laboratory measure that can be considered a "gold standard" in defining iron deficiency. As a result, studies designed to assess the iron status of a population, such as the National Health and Nutrition Examination Survey (NHANES), frequently involve three measures of iron status and define as iron deficient any individual in whom two of the three measures are abnormal.[29] In comparison with this more restrictive definition, use of only one measure of iron status would bias prevalence rates upward. There is no evidence that any other single laboratory measurement is superior to erythrocyte protoporphyrin in identifying children with iron deficiency. In a previous study, the erythrocyte protoporphyrin measure alone performed with a sensitivity and specificity equal to hemoglobin measurement in identifying children with low transferrin saturation.[30]

Some of the observed variation in community rates could be due to interlaboratory discrepancies, which can be substantial with the erythrocyte protoporphyrin assay,[31] but the use of one laboratory for the analysis of 85% of the samples in this study kept interlaboratory variability to a minimum. In addition, the magnitude and significance of odds ratios for socioeconomic status, Southeast Asian ethnicity, and Hispanic ethnicity were not affected by exclusion of data from Boston and Worcester, the two cities with data from other laboratories. Intralaboratory errors in erythrocyte protoporphyrin assay are small.[31] Another limitation is the confounding of our measure of iron status by blood lead elevation. Although we tried to minimize confounding by excluding children with erythrocyte protoporphyrin and blood lead elevations and by including prevalence of blood lead levels of 20 μg/dL or more as an independent variable, it is possible that there was some residual confounding of iron deficiency prevalence by lead exposure. We expect that this was minimal because of the low prevalence of blood lead elevations in comparison with the rates of iron deficiency in these communities.

Variation of iron deficiency prevalence by community has implications for iron deficiency screening. The American Academy of Pediatrics currently recommends universal screening for iron deficiency in the first 2 years of life. This study demonstrates that universal screening continues to be indicated in low-income communities or in communities with high proportions of certain racial/ethnic groups. However, the positive predictive value of any screening test decreases with decreasing prevalence of the disease. In communities where 5% or fewer children have erythrocyte protoporphyrin elevation, it is likely that a substantial majority of the screen-positive population is false positive,[32] in which case the positive predictive value of a positive screening test (child with erythrocyte protoporphyrin ≥0.62 μmol/L or hemoglobin <11.5) is very low. Children in such communities would be better served by screening programs that target children with individual risk factors, such as the early introduction of cow's milk,[33,34] for screening.

Low socioeconomic status is a predictor of community rates of iron deficiency, contrary to the findings in studies of smaller samples of children.[28] This finding suggests a continued need for population-based strategies for supplementing iron in the diets of children, such as the provision of iron-fortified infant formula to eligible families in low-income communities. We were not able to determine individual economic, medical, or nutritional characteristics of children and their families that continue to make poverty a risk factor for iron deficiency despite dietary supplementation with iron. These factors should be further investigated at the individual level to determine whether resources for pri-

mary and secondary prevention of iron deficiency in low-income communities can be targeted more effectively.

In addition, we found that communities with larger Hispanic and Southeast Asian populations exhibited higher rates of iron deficiency, even after socioeconomic status had been controlled for. Results for Black children were inconclusive; communities with larger proportions of Blacks do not seem to have higher adjusted odds for iron deficiency. However, the adjusted odds ratio using the more restrictive definition (erythrocyte protoporphyrin ≥ 0.9 μmol/L and blood lead < 1.2 μmol/L) was significantly higher than one and was similar in magnitude to that seen in Hispanic children. Because of mixed findings in the community analysis and our inability to identify Black children in the individual study, we are unable to reach a conclusion about risk of iron deficiency in the Black population.

Data on iron deficiency in young Hispanic children are sparse. A recent study using the Hispanic Health and Nutrition Examination Survey (HHANES)[35] found no differences in the iron status of these children as compared with White children in the NHANES II study. However, this examination was confined to children older than 5 years. Our findings indicate that results of the HHANES survey should be supplemented by a study of young Hispanic children in order to accurately determine the prevalence of iron deficiency in children under 5 years of age. If confirmed, further studies of the etiology of iron deficiency in young Hispanic children are needed.

The adjusted odds of iron deficiency in communities rose strikingly with increases in the Southeast Asian population (by about 10% for each 1% increase in the population). While we found a high rate of iron deficiency in children with Southeast Asian surnames at the individual level (39%), this rate was not high enough to account for the adjusted odds ratio seen at the community level. We can think of at least two possible explanations for this cross-level bias.[36] First, there could be other unmeasured predictors of iron deficiency, differentially distributed by community, that confounded the relationship between percentage Southeast Asian and iron deficiency rate. Second, and more likely, there was aggregation bias (i.e., bias in the way these groups were determined). For instance, census undercounts of Southeast Asian children in communities[37] would result in overestimation of the risk attributed to that segment of the population. Positive bias could be further increased by the oversampling of Southeast Asian children in comparison with other children in communities (such as Lowell) that have screening programs directed at the Southeast Asian population. Considerations of cross-level bias aside, the finding of higher rates of iron deficiency in Southeast Asian children at both the individual and ecological levels increases our confidence in this association.

Moreover, our finding is consistent with published evaluations of dietary practices and small studies of iron deficiency in this population. It has been shown that Southeast Asian children living in their native countries have high rates of iron deficiency. Dietary studies link iron deficiency with traditional Southeast Asian meals composed of rice, vegetables, and spices, which are low in bioavailable iron.[38,39] During the period of heavy immigration of Southeast Asians to the United States, studies reported a high prevalence of iron deficiency in these populations.[40,41] In another study, Brown et al.[42] examined the iron status of Southeast Asian children, some of whom were born in the United States, and found 15.9% of Southeast Asian children to have an erythrocyte protoporphyrin concentration greater than or equal to 1.06 μmol/L (60 μg/dL). This is similar to the rates we have reported. Seventy-six percent of Southeast Asian children with erythrocyte protoporphyrin elevation in the Brown et al. study had confirmation of iron deficiency with serum ferritin at follow-up, suggesting that elevation in erythrocyte protoporphyrin was associated with iron deficiency in these children.

However, it is possible that an upward shift in the distribution of erythrocyte protoporphyrin in Southeast Asians in comparison with other racial groups explains some of the increased prevalence of iron deficiency that we have reported. Since there has been no large study of erythrocyte protoporphyrin and iron status in Southeast Asian children, we do not know the 95th percentile of erythrocyte protoporphyrin in the iron-replete Southeast Asian population. The erythrocyte protoporphyrin cutoff value of 0.62 μmol/L that we used was determined from a study of iron status in a predominantly White population.[7] Southeast Asians are at risk for a number of inherited hematologic disorders, including hemoglobin E,[43,44] a- and b-thalassemia,[41] and glucose-6-dehydroginase deficiency. None of these disorders are associated with a rise in erythrocyte protoporphyrin; in fact, erythrocyte protoporphyrin has been used to discriminate between the microcytosis of iron deficiency and that of the thalassemias.[45] Nonetheless, it is possible that mild elevation in hematofluorimetrically determined erythrocyte protoporphyrin is more likely in Asian than in Caucasian populations. The fact that our findings for Southeast Asian persisted with the more restrictive definition of iron deficiency suggests that children with mild elevation of erythrocyte protoporphyrin from other factors were not driving these results. But our interpretation of the findings in individual Southeast Asian children was limited by lack of data on confirmatory measures of iron status as well as the possible misclassification of ethnicity, and we underline the need for further studies in this population of children, including the oversampling of Southeast Asians in nationally representative nutritional surveys such as NHANES.

Iron deficiency is a preventable disease. There have been major advances in its elimination in the middle-class population; indeed, our analysis indicates that routine screening for iron deficiency in some Massachusetts communities may not be necessary. However, there are still communities in which the prevalence of this condition is high enough to warrant the screening of all children. Young Hispanic and Southeast Asian children appear to be at high risk for iron deficiency, and further studies are required to define the prevalence of iron deficiency in these populations. If our findings of increased risk are confirmed, individual studies should be conducted to determine the more proximate risk factors, such as cultural beliefs about infant feeding, that mediate higher rates of iron deficiency in these children. This information will allow health providers to implement primary prevention and more efficiently target their secondary prevention strategies within these groups of children. Finally, for those who screen for iron deficiency using the erythrocyte protoporphyrin analysis, a determination of normative data for erythrocyte protoporphyrin and its usefulness in screening for iron deficiency in the Southeast Asian population is needed. □

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