Calcium and phosphorus supplementation of iron-fortified infant formula: no effect on iron status of healthy full-term infants

Madeline A Dalton, James D Sargent, Gerald T O'Connor, Elaine M Olmstead, and Robert Z Klein

ABSTRACT One objective of this clinical trial was to determine whether calcium and phosphorus supplementation of infant formula affects the iron status of healthy full-term infants. One hundred three infants were randomly assigned to receive iron-fortified, cow milk–based infant formula (465 mg Ca and 317 mg P/L) or the same formula with added calcium glycerophosphate (1800 mg Ca and 1390 mg P/L) for 9 mo. Reported calcium intake for supplemented infants was about four times that of control infants, ranging from a mean of 1741 mg/d at baseline to 1563 mg/d at 9 mo. There was no difference by treatment group in mean or median change from baseline of serum ferritin, total-iron-binding capacity, erythrocyte protoporphyrin, or hematocrit at 4 and 9 mo after enrollment. Incidence of iron deficiency was similar for both groups and no infant developed iron deficiency anemia during the trial. This study indicates that the well-documented inhibitory effect of calcium and phosphorus on iron absorption is not clinically important in infants fed iron-fortified infant formula. Am J Clin Nutr 1997;65:921–6.

KEY WORDS Calcium, phosphate, iron, infant feeding, calcium supplementation, iron absorption, iron-fortified formula

INTRODUCTION Studies show that infants consuming whole cow milk have higher rates of iron deficiency than do infants consuming human milk, despite the equivalent iron contents of these milks (1–6). Because cow milk contains four to five times as much calcium as human milk, calcium inhibition of iron absorption is a likely explanation for the difference in iron bioavailability. Several studies have documented the inhibitory effects of dietary calcium on the absorption of iron from food (7–13). One study showed that the addition of calcium to human milk reduced the bioavailability of iron (14).

Despite substantial evidence that calcium inhibits the absorption of iron from foods, the clinical significance of this for infants consuming iron-fortified infant formula is not known. The primary objective of this clinical trial was to examine the effect of calcium and phosphorus supplementation of infant formula on lead retention in growing infants. A secondary objective, reported herein, was to evaluate the effect of calcium and phosphorus supplementation on iron status.

SUBJECTS AND METHODS

Study population

The study was conducted in Lawrence, MA, a manufacturing city with a population of 60,000. Almost one-half of the residents in Lawrence are Latino. The majority of the residents have low incomes and many receive public assistance. Mothers and children were recruited for the study primarily from the Lawrence Women, Infants and Children Program (WIC), the Merrimack Lead Prevention Program, and community health centers. Subjects were enrolled on a continuous basis between October 1991 and May 1993. The study was approved by the Committee for Protection of Human Subjects at Dartmouth College, Hanover, NH.

Eligibility of the subjects was determined by the project manager and a pediatrician who were both unaware of treatment assignment. Primary entry criteria included the following: 1) the parent and infant resided in a geographic area identified as high risk for lead exposure; 2) the infant was drinking ≥ 600 mL cow milk–based formula per day; 3) the infant was between 2.5 and 5 mo of age; 4) the infant’s birth weight was > 2.0 kg; 5) the infant’s height and weight were above the 5th percentile for age; 6) there was no history of kidney stones in first-degree relatives; 7) the infant had no history of rickets, parathyroid dysfunction, or a recent fracture; 8) the infant was not receiving supplemental multivitamins or, if he or she was, the parent was willing to discontinue them; and 9) the infant was not chronically taking medications other than antihista-
mine decongestants, fluoride supplements, or prophylactic antibiotics to prevent recurrent otitis media.

If the primary entry criteria were satisfied, the infant was enrolled in a 1-mo run-in period to evaluate secondary entry criteria. These included the following: 1) the parent and infant returned for the 2-wk appointment; 2) the infant drank high-calcium formula for ≥ 2 wk; 3) the parent, family members, and pediatrician agreed to the infant’s participation and the parent gave written informed consent; 4) blood lead concentration was < 0.12 μmol/L (25 μg/dL); 5) the infant had no hematuria (≥ 5 red blood cells per high-powered field); and 6) no hypercalciuria after drinking the supplemented formula for 2 wk.

Infants who satisfied all of the entry criteria were randomly assigned in a double-blind fashion to one of two groups. Infants in the control group received standard iron-fortified infant formula (Enfamil with Iron; Mead Johnson Co, Evansville, IN) containing 465 mg Ca and 317 mg P/L. The treatment group received the same formula supplemented with the maximum soluble amount of calcium glycerophosphate, which resulted in a concentration of 1800 mg Ca and 1390 mg P/L. Both the control and treatment formulas contained 12.8 mg Fe/L.

For infants < 6 mo of age, 1 L of the standard infant formula provided slightly more than the recommended dietary allowances (RDAs; 15) for calcium (400 mg) and phosphorus (300 mg). For infants between 6 and 12 mo of age, the standard formula provided slightly less than the RDA for calcium (600 mg) and phosphorus (500 mg). One liter of the supplemented formula provided infants with 3–4.5 times the RDA for these minerals. One liter of either formula provided more than the RDA for iron for infants in their first year of life (6 mg for infants < 6 mo; 10 mg for infants 6–12 mo).

The standard infant formula and the calcium glycerophosphate–supplemented formula were supplied in 1-lb (453 g) cans of powder with identical labeling. Each can made ~3600 mL infant formula, prepared with one scoop (provided in the can) of powder for every 60 mL water. At the time of enrollment, parents were instructed to prepare the formula by adding one level scoop of formula for every 60 mL (2 oz) warm water. They were asked to add the water to the bottle first and then add the powder. After explaining and demonstrating how to prepare the formula, the project manager or research assistant observed the parent prepare a bottle. The amount of formula prepared was determined by the infant’s usual intake of formula. To ensure that parents continued to prepare the formula correctly, they were asked to demonstrate their preparation for the project manager or research assistant at least three times during the 9-mo period. In addition, at every monthly visit, parents were asked to describe in detail how they prepared the formula. This included the number of ounces of water used, number of scoops of powder used, whether the scoop was level or heaping, whether the water or the powder was added to the bottle first, and if any other food or liquid was added to the formula. If a parent reported any departure from the standard protocol for preparation, study personnel instructed them again on how to correctly prepare the formula and asked the parent to demonstrate the preparation. With two exceptions, parents consistently prepared the formula correctly.

**Collection and analysis of samples**

After random assignment, parents were asked to return with their infants every month for 9 mo. The primary outcome measure in this clinical trial was blood lead concentration (JS Sargent, MA Dalton, GT O’Connor, EM Olmstead, RZ Klein; unpublished report to the Maternal and Child Health Research Program, HRSA, PHS, DHHS, 1994) but, because of the possibility that added calcium would decrease iron absorption, we also measured iron status and hematocrit. Blood samples were obtained at baseline, month 4, and month 9 to measure serum ferritin, total-iron-binding capacity (TIBC), erythrocyte protoporphyrin, and hematocrit.

Serum ferritin was measured by using the Delfia instrument (Wallac Inc, Gaithersburg, MD). Two standards supplied by the manufacturer, at low and high concentrations, were used to monitor performance of this assay. The acceptable ranges for the quality control samples were 20–31 and 410–580 μg/L. The mean (± SD) for repeated analyses (n = 51) of the low-concentration sample was 23.6 ± 3.4 μg/L; for the high-concentration sample it was 486 ± 26.8 μg/L. TIBC was measured with the Ferrochem II instrument (Environmental Science Associates, Boston). The average CV for repeated analyses (once per month) of three blood samples over time was 8.0%. Erythrocyte protoporphyrin was determined by using the extraction method (16) with subsequent analysis on a compact digital filter-type fluorometer (model 450; Sequoia-Turner Corp, Mountain View, CA). Reference samples from the Centers for Disease Control and Prevention Proficiency Program were analyzed three times per month to monitor performance of the erythrocyte protoporphyrin assay. The mean (± SD) of the error for analysis of reference samples was −0.08 ± 0.08 μmol/L (4.3 μg/dL).

Hematocrit was determined on site by using an Autocrit II centrifuge (Clay-Adams, Parsippany, NJ). Duplicate microhematocrit tubes obtained from venous collection were placed into the centrifuge and spun for 3 min and the results read by the phlebotomist or the project manager. The hematocrit value we report represents the mean of these two results.

Three-day, concurrent formula records, completed by the parents, were used to estimate formula intake at baseline, 4 mo, and 9 mo. Although this method of data collection places a greater burden on the participant (17), there are several reasons why it was chosen: 1) we believe there would have been substantial recall bias with retrospective methods because of the frequency with which infants drink formula, 2) having the volume marked on the side of the bottle made it relatively easy for parents to record the quantities of formula consumed, and 3) the problem of altering intake during the 3-d period is less likely with infants than adults because they are unaware of the data collection.

Parents (or other caregivers) were asked to fill out a diary that indicated the time of each formula feeding, the number of scoops of powdered formula used to prepare the bottle, the total number of ounces in the bottle before feeding and the amount left in the bottle after feeding. Parents were also asked to record the same information for any food or drinks that were made with formula. At the end of the 3-d period, the research assistant or project manager reviewed the formula records with the parents to determine whether the diary was complete, collect additional information on formula preparation and intake, and
determine whether the infant drank any cow milk or ate any foods prepared with cow milk over the 3-d period.

At each monthly visit, parents were asked to report their infant’s average daily intake of formula and the number of cans of powdered formula used that month. Daily formula intake reported on the concurrent records at 4 and 9 mo were compared with parents’ monthly estimates of their infant’s usual daily intake. The Pearson correlation coefficients for the comparison of these measures were 0.63 (n = 69, P = 0.0001) and 0.65 (n = 54, P = 0.0001), respectively.

Laboratory norms

During the trial, infants in whom any biochemical measure indicated iron deficiency [serum ferritin ≤ 12 μg/L, TIBC > 86 μmol/L (480 μg/dL), or erythrocyte protoporphyrin ≥ 0.62 μmol/L (35 μg/dL)] (18), were prescribed iron sulfate syrup (5 mg elemental Fe · kg body wt⁻¹ · d⁻¹, given twice daily) for a period of 3 mo. In this report, an infant was considered to be iron deficient if two or more of the three biochemical test results were abnormal. An infant was defined as having iron deficiency anemia if two or more of the three biochemical tests were abnormal and the hematocrit was ≤ 0.32 (19, 20).

Statistical analysis

Unpaired t tests (21) were used to test for differences between group means of normally distributed data (formula, iron and calcium intakes, change in TIBC, and change in hematocrit). Wilcoxon rank-sum tests (21) were used to test for differences in group medians for all variables that were not normally distributed (change in serum ferritin, change in erythrocyte protoporphyrin). Fisher’s exact test was used to compare the proportion of infants with iron deficiency and iron deficiency anemia by treatment group.

RESULTS

Three hundred fourteen infants were enrolled in a 1-mo run-in period to evaluate eligibility. One-third (n = 103) of all infants who participated in the run-in completed it successfully and were randomly assigned into study groups. The primary reason for not completing the run-in was failure to return for the 2-wk visit (n = 162). We made two attempts to contact parents who did not return for their follow-up visit, but in most instances we were unable to find out why they did not want to or were unable to continue. Nineteen infants were excluded during run-in for reasons including the mother’s perception that her infant did not like the formula, that it caused constipation, that it would be better not to change formulas, or because a family member or pediatrician objected to the study. The project manager decided not to randomly assign an additional 27 infants because she thought they were unlikely to participate in the study for a full 9 mo. Only three children (1%) were excluded during run-in because of hypercalcemia after drinking the supplemented formula for 2 wk.

Of the 103 infants randomly assigned in the study (52 control, 51 treatment), 81 (41 control, 40 treatment) completed the 9-mo trial. Final blood samples were obtained from 10 of the 22 infants who dropped out before completion. Dropout rates were equivalent for both groups (11 control and 11 treatment subjects) and the the clinical measures of the dropouts did not differ significantly from those who completed the study. Reasons for leaving the study included moving from the study area (four control, three treatment), switching to cow milk or another formula (four control, one treatment), prolonged diarrhea that the mother attributed to the formula (one treatment), and precipitation of the milk protein when the formula was prepared in the microwave (one treatment). We were unable to contact parents of five infants in the treatment group and three infants in the control group to determine why they dropped out.

Demographic characteristics and blood lead concentrations were similar for both groups (Table 1). There were no differences between the control and treatment groups in ethnicity (92.3% and 92.2% Latino, respectively), marital status of the mother (50.0% and 44.0% single), and median household per capita income ($2545 and $2772/y).

As shown in Table 2, iron intake from formula was similar for the two groups at baseline, 4 mo, and 9 mo. In contrast, mean calcium intake for the treatment group was between three and five times higher than for the control group throughout the study.

There were no significant differences between groups at baseline, or 4 or 9 mo for mean TIBC or median erythrocyte protoporphyrin (Table 3). Median serum ferritin was significantly lower in the treatment group at baseline, representing a failure of randomization to yield similar groups with respect to this variable. Median serum ferritin remained significantly lower for supplemented infants for the duration of the trial.

Thirteen children were given nightly iron supplements during the study because of low serum ferritin concentrations at 4 mo: five in the control group and eight in the treatment group (P = 0.3). One-half of these children (n = 7) still had low serum ferritin at the end of the study. Children with persistently low serum ferritin were equally distributed between control and treatment groups. Additionally, three children in the treatment group had low serum ferritin at the end of the study.

Mean hematocrit was slightly, but not significantly, lower for treatment infants at baseline and 4 mo. At 9 mo, the difference between mean hematocrit for each group was marginally significant (0.36 in treatment compared with 0.37 in control infants; P = 0.05), but there was no significant difference in the proportion of infants with hematocrit < 0.33 at that time (2.9% of control infants, 2.8% of treatment infants).

Median change in serum ferritin from baseline to 4 and 9 mo ranged from –24.5 to –46.6 μg/L for both control and supplemented infants (Table 4). There was no difference in the mean or median change from baseline in serum ferritin, TIBC, erythrocyte protoporphyrin, or hematocrit by treatment group.

### TABLE 1

Baseline characteristics

<table>
<thead>
<tr>
<th>Baseline characteristics</th>
<th>Control (n = 52)</th>
<th>Supplemented (n = 51)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal age (y)</td>
<td>25.2 ± 5.7</td>
<td>25.5 ± 5.8</td>
</tr>
<tr>
<td>Maternal education (y)</td>
<td>11.0 ± 2.4</td>
<td>11.2 ± 2.9</td>
</tr>
<tr>
<td>Child’s age (mo)</td>
<td>3.6 ± 1.4</td>
<td>3.5 ± 1.3</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>6.7 ± 1.2</td>
<td>6.9 ± 1.2</td>
</tr>
<tr>
<td>Median blood lead (μmol/L)</td>
<td>0.12 ± 0.06</td>
<td>0.13 ± 0.07</td>
</tr>
</tbody>
</table>

*P < 0.05, unless otherwise noted.*
TABLE 2
Calcium and iron intakes from infant formula

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>Supplemented</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iron intake from formula (mg/d)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>12.8 ± 2.3 [52]</td>
<td>12.1 ± 3.1 [51]</td>
</tr>
<tr>
<td>4 mo</td>
<td>10.8 ± 3.5 [46]</td>
<td>11.9 ± 3.2 [43]</td>
</tr>
<tr>
<td>9 mo</td>
<td>9.2 ± 4.7 [35]</td>
<td>10.9 ± 4.9 [27]</td>
</tr>
<tr>
<td>Calcium intake from formula (mg/d)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>477.8 ± 86.6 [52]</td>
<td>1740.7 ± 451.4 [51]</td>
</tr>
<tr>
<td>4 mo</td>
<td>402.1 ± 130.1 [46]</td>
<td>1703.7 ± 456.6 [43]</td>
</tr>
<tr>
<td>9 mo</td>
<td>343.6 ± 173.1 [35]</td>
<td>1563.0 ± 703.6 [27]</td>
</tr>
</tbody>
</table>

*1 t = SD; n in brackets.

at 4 or 9 mo. There was no significant difference in the proportion of infants who were iron deficient at baseline (2% treatment, 2% control), 4 mo (10% treatment, 7% control), or 9 mo (5% treatment, 5% control). Moreover, no infant developed iron deficiency anemia during the course of the trial. All analyses were repeated excluding the infants who were placed on nightly iron supplements and the findings did not change.

DISCUSSION

There is a large body of literature supporting an inhibitory effect of dietary calcium and phosphorus on iron absorption. For some time it has been known that iron in breast milk has greater bioavailability than iron in cow milk (2, 4, 14, 22-25) or cow milk–based infant formula (24, 26, 27). Differences in iron bioavailability for these milks have been attributed to differences in protein, fat, lactose, lactoferrin, calcium, and phosphorus content (24, 26, 27). However, Hallberg et al (14) showed that iron bioavailability in adults fed human milk decreases as the concentration of calcium is increased. These data confirm studies in adults and animals that show a dose-related reduction of iron bioavailability with increases in calcium.

Our results suggest that the inhibitory effect of calcium on iron absorption is not nutritionally significant for full-term infants who are consuming iron-fortified cow milk–based infant formula. Infants who received the calcium glycerophosphate supplement did not have a higher incidence of iron deficiency or iron deficiency anemia.

However, there are several limitations to our study that should be taken into account when interpreting these results. First, we chose calcium glycerophosphate because of its high solubility in the formula. It is possible that the bioavailability of this supplement is different from that for other calcium preparations and this may affect its interaction with iron. Second, because > 90% of the study population was Latino we cannot be certain that our results are generalizable to other racial or ethnic groups. Third, we only measured iron and calcium intake from formula. During the latter half of the study infants were between 7 and 15 mo of age. Because this is a time when an increasing number of solid foods are typically introduced into the diet, we presumably underestimated both iron and calcium intakes. In addition, there is the possibility that parents over- or underestimated their infant’s formula intake. However, because this was a randomized trial, there is no reason to believe that either of these measurement errors would bias the results.

Our study complements one by Pizarro et al (20) in which Chilean infants consuming iron-fortified (15 mg Fe and 1200 mg Ca/L) whole cow milk prepared from powder, had a lower incidence of iron deficiency compared with infants consuming unfortified whole cow milk. In addition, the Chilean infants were exposed to whole cow milk protein, which has been shown to induce microscopic gastrointestinal bleeding in young infants (4, 12, 33). This suggests that neither high calcium content nor blood loss are relevant to iron status, given a formula iron content of 12–15 mg/L. Because neither this study nor that of Pizarro et al included a direct measure of iron absorption, the possibility exists that the study calcium does not inhibit iron absorption at all. We consider this unlikely given the strength of experimental evidence supporting an inhibitory effect of calcium. Therefore, we offer several reasons why calcium-supplemented infants in our study and that of Pizarro et al (20) remained iron-replete despite a probable calcium effect.

First, the majority of studies showing calcium inhibition of iron absorption were conducted in adults (8–10, 14) and there may be important differences between adults and infants in the absorption of dietary iron (22). Infants have a much greater dependence on dietary iron to sustain rapid increases in blood volume. In infants, 30% of iron comes from the diet compared with 5% for adults (34). Moreover, infants have marginal iron reserves compared with adults; average serum ferritin in a growing infant is 30 μg/L compared with 100 μg/L in adult males (35). Because body iron is regulated primarily through modulation of iron absorption over a 20-fold range (36), with iron absorption increasing as storage iron decreases, infants would be expected to have a much higher rate of iron absorp-
Mean erythrocyte protoporphyrin, and hematocrit in control and calcium-phosphorus-supplemented infants

<table>
<thead>
<tr>
<th>Iron status variable</th>
<th>Control</th>
<th>Supplemented</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median change in serum ferritin (μg/L)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 mo — baseline</td>
<td>28.6 ± 46.9 [40]</td>
<td>24.5 ± 45.9 [41]</td>
</tr>
<tr>
<td>9 mo — baseline</td>
<td>46.6 ± 66.8 [39]</td>
<td>31.4 ± 43.0 [38]</td>
</tr>
<tr>
<td>Mean change in total-iron-binding capacity (μmol/L)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 mo — baseline</td>
<td>0.7 ± 9.8 [40]</td>
<td>3.0 ± 7.4 [40]</td>
</tr>
<tr>
<td>9 mo — baseline</td>
<td>2.4 ± 10.1 [39]</td>
<td>4.5 ± 10.2 [37]</td>
</tr>
<tr>
<td>Median change in erythrocyte protoporphyrin (μmol/L)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 mo — baseline</td>
<td>0.03 ± 0.2 [42]</td>
<td>0.02 ± 0.1 [41]</td>
</tr>
<tr>
<td>9 mo — baseline</td>
<td>0.03 ± 0.2 [38]</td>
<td>0.02 ± 0.2 [38]</td>
</tr>
<tr>
<td>Mean change in hematocrit (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 mo — baseline</td>
<td>0.01 ± 0.03 [36]</td>
<td>0.01 ± 0.03 [32]</td>
</tr>
<tr>
<td>9 mo — baseline</td>
<td>0.02 ± 0.03 [31]</td>
<td>0.02 ± 0.03 [33]</td>
</tr>
</tbody>
</table>

1, 2: x over median ± SD; n in brackets. There were no significant differences between groups.

TABLE 4

Prevalence of iron deficiency and iron deficiency anemia in control and calcium-phosphorus-supplemented infants

<table>
<thead>
<tr>
<th>Iron deficiency</th>
<th>Control</th>
<th>Supplemented</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>2.1 [1]</td>
<td>2.1 [1]</td>
</tr>
<tr>
<td>9 mo</td>
<td>5.1 [2]</td>
<td>5.3 [2]</td>
</tr>
<tr>
<td>Iron deficiency anemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>2.0 [1]</td>
<td>0.0 [0]</td>
</tr>
<tr>
<td>4 mo</td>
<td>0.0 [0]</td>
<td>0.0 [0]</td>
</tr>
<tr>
<td>9 mo</td>
<td>0.0 [0]</td>
<td>0.0 [0]</td>
</tr>
</tbody>
</table>

1, 2: n in brackets.

1 Two or more of the following present: serum ferritin < 12 μg/L, total-iron-binding capacity > 86 μmol/L (480 μg/dL), erythrocyte protoporphyrin > 0.62 μmol/L (35 μg/dL).

Iron deficiency (as defined above) with hematocrit < 0.32.

calcium from 300 to 600 mg did not significantly decrease the amount of iron absorbed. If iron absorption was maximally inhibited by the 465 mg Ca in the unfortified formula, it is possible that we would have detected a calcium effect on iron status had we used a control formula with a lower concentration of calcium. In such circumstances, one would expect greater iron stores in infants receiving low-calcium formula.

Finally, studies of iron bioavailability in infants have focused on the percentage of iron absorbed, rather than net iron absorption. Breast milk contains < 5% of the amount of iron contained in iron-fortified infant formula (0.5 compared with 12.8 mg/L). Thus, although a higher percentage of iron is absorbed from breast milk compared with infant formula (50% compared with 5%), the total amount of iron absorbed in milligrams per day from breast milk is only 25% of the amount absorbed from iron-fortified infant formula (24, 26). We suggest that iron in the diets of the calcium- and phosphorus-supplemented infants was present in sufficient excess so that inhibition of 40–50% of its absorption did not result in iron deficiency. Supporting this hypothesis are studies in juvenile rats showing dose-related calcium inhibition of iron absorption from intestinal loops, but no difference in biochemical markers of iron deficiency in rats fed iron-replete diets with added dietary calcium compared with controls (29).

In conclusion, the effect of supplementation of infant formula with calcium and phosphorus to three to four times normal dietary amounts has no clinical effect on the iron status or the incidence of iron deficiency in full-term infants fed iron-fortified formula between the ages of 6 and 15 mo.

We acknowledge Miriam Nelson for her help in the analysis of the calcium and phosphorus intake data and David Bellinger and Robert Bornschein for their help in the design of the study. The clinical trial was implemented with the help of Alison Kiley, Belquis Zayas, Sandi Roda, Carmen Torres, Evelyn Kocher-Ahern, and the nutritionists at the Lawrence WIC program.

REFERENCES


