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Effects of a large-scale micronutrient powder and young child feeding education program on the micronutrient status of children 6–24 months of age in the Kyrgyz Republic

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Abstract

BACKGROUND/OBJECTIVES—To combat iron and other micronutrient deficiencies, the Ministry of Health of the Kyrgyz Republic launched a regional Infant and Young Child Nutrition (IYCN) program in 2009, which included promotion of home fortification with micronutrient powder (MNP) containing iron (12.5 mg elemental iron), vitamin A (300 μg) and other micronutrients. Every 2 months children aged 6–24 months were provided 30 sachets to be taken on a flexible schedule. The objective was to assess biochemical indicators of iron and vitamin A status among children aged 6–24 months at the baseline and follow-up surveys.

SUBJECTS/METHODS—Cross-sectional representative cluster surveys were conducted in 2008 (*n* =571 children) and 2010 (*n* =541). Data collected included measurement of hemoglobin,

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CONFLICT OF INTEREST

The authors declares no conflict of interest

DISCLAIMER

The findings and conclusions of this report do not necessarily represent the official position of the US Centers for Disease Control and Prevention.

Use of trade names is for identification only and does not imply endorsement by the US Department of Health and Human Services.

serum ferritin, soluble transferrin receptor (sTfR), retinol-binding protein, C-reactive protein (CRP) and α_1-glycoprotein acid (AGP).

RESULTS—Among all children, declines were observed in the prevalence of: anemia, 50.6% versus 43.8% (P =0.05); total iron deficiency (either low ferritin or high sTfR), 77.3% versus 63.7% (P<0.01); and iron deficiency anemia, 45.5% versus 33.4% (P<0.01). Among children without inflammation as measured by CRP and AGP, similar declines were observed, but only declines in total iron deficiency and iron deficiency anemia reached statistical significance. Among all children and those without inflammation, the prevalence of vitamin A deficiency remained the same.

CONCLUSIONS—One year after the introduction of home fortification with MNP, within a larger IYCN program, the prevalence of anemia, iron deficiency and iron deficiency anemia declined, but vitamin A deficiency remained unchanged.

Keywords

anemia; iron deficiency; micronutrients; dietary supplements; child preschool; powders

INTRODUCTION

Anemia is an important public health problem in the Kyrgyz Republic. In the 1997 Demographic and Health Survey, the prevalence of anemia was 50% in children 6–36 months of age and 38% in women of reproductive age.[1] Non-nationally representative studies undertaken since 1997 suggest that anemia remains a public health problem, despite various campaigns.[2] To address iron and other micronutrient deficiencies, the Ministry of Health (MOH) launched a pilot Infant and Young Child Nutrition (IYCN) program, which included a nutrition education program to encourage breastfeeding and appropriate complementary feeding, and distribution of micronutrient powder (MNP) for consumption by children 6–24 months of age.

MNP has been shown to prevent and reduce anemia. A systematic review of efficacy and effectiveness studies, in which home fortification (including MNP, crushable tablets and fat-based products) of complementary foods was the primary intervention for anemia prevention, concluded that the average effect was an increase in hemoglobin by 8 g/l and a 21 percentage point (PP) decrease in the prevalence of anemia in children 6–24 months of age.[3] A Cochrane review evaluated effects of home fortification of foods with MNP containing at least iron, zinc and vitamin A;[4] compared with no intervention or placebo, home fortification with MNP showed a 31% relative reduction in anemia and a 51% relative reduction in iron deficiency.

Based on the success of these studies, MNP programs are being implemented on a population level in many countries around the world.[5] Although the efficacy of MNP programs has been well established in numerous trials, effectiveness studies of MNP programs in the peer-reviewed literature are relatively few, and have been conducted among special populations such as refugee settings[6,7] or food aid recipients.[8] Two previous studies conducted in a general population were of 2–6 months duration.[2,9] With one exception,[9]

none have measured iron deficiency or accounted for effects of inflammation, which can influence concentration of biomarkers.[10,11]

We used a pre-post survey design to examine the prevalence of anemia, iron deficiency, iron deficiency anemia and vitamin A deficiency before and after the initiation of a large-scale integrated IYCN/MNP program, taking inflammation status into account.

MATERIALS AND METHODS

IYCN/MNP program

The program included both a community-based nutrition education initiative and distribution of MNP through government primary health-care clinics. The community-based nutrition education program was implemented in the rural areas of the Talas Oblast (province) of the Kyrgyz Republic. The education program was designed to improve diet during pregnancy, encourage breastfeeding until 24 months of age (exclusive breastfeeding until 6 months of age) and improve complementary feeding. The nutrition education program of the MOH was implemented by village health committees, which consist of volunteers who are trained on public health and nutrition issues and who share their knowledge through peer-to-peer interactions.

The education program on infant and young child feeding began in May 2008. One year later in June 2009, the MOH launched a program to distribute MNP through government primary health-care centers at no cost to all children 6–24 months of age. Primary health-care providers distributed MNP, instructed caretakers on proper use and distributed informational materials. The MNP was packaged in individual dose sachets that contained 12.5 mg of elemental iron (as microencapsulated ferrous fumarate); 160 μg of folic acid; 300 μg of vitamin A (retinol acetate); 30 mg of vitamin C (ascorbic acid); and 5 mg of zinc (zinc gluconate). Procured through United Nations Children's Fund, the product was required to meet strict quality control requirements.[12] As children reached 6 months of age, they were eligible to receive MNP at their primary health-care clinic and were invited to enroll during their routine well-child checkup. In some cases, primary health-care providers visited the family at home to recruit children. Trained health-care providers distributed MNP either at the health clinics or at the household. Using a 'flexible administration' approach, caretakers were instructed to give the MNP according to any schedule they wished, as long as they used all 30 sachets in 2 months. Caretakers were given an instructional flier and a reminder card with the date they should receive their next package.

Before the launch of the program, formative research was conducted. Focus group discussions were held with a sample of caretakers and primary health-care providers. Instructional and promotional materials were prepared for distribution. A logo and accompanying message ('For the health and mind of your child') were developed. Village health committees were encouraged to inform caretakers about MNP and to ask them to bring their children to the health-care provider to enter into the program. Village health committees delivered key messages with the aid of flip charts and brochures and also distributed a children's book. Primary health-care providers were solely responsible for distributing MNP and, with the aid of instructional brochures, informing caretakers about

appropriate preparation and use. The program also enlisted the strong support of both the local governmental authorities and the mass media through provision of press-kits at the kickoff celebration and the airing of advertisements, jingles and informational interviews on the widely popular regional public radio station.

Survey design and sampling

A baseline survey was conducted in June/July 2008, and a follow-up survey was conducted in July/August 2010, approximately one year after MNP distribution began in June 2009.

Based on a pre- and post-survey design with independent samples (estimated design effect = 2), we initially calculated that a target sample size of 540 participants would be needed for each survey (power = 0.80 and alpha = 0.05). This sample size was based on the number of participants necessary to detect a *relative* decline in the prevalence of anemia and iron deficiency of 20%. To adjust for nonresponse, 20 children were initially included in each cluster (30 clusters). Because initial response was lower than expected in the 2008 survey, the coordinator increased the number of children selected for interview to 22 in each cluster. This number was further increased to 24 in the 2010 survey to account for both the higher than expected nonresponse and the absence due to migration observed in the 2008 survey. The sample frame for the 2008 survey was children 6–24 months living in rural Talas; the sample frame for the 2010 survey was children 6–24 months living in Talas (26 rural clusters and 4 urban clusters).

For both surveys a two-stage cluster sampling design was used. All children living in the Kyrgyz Republic are assigned to a primary health-care center based on location of residence. Each primary health-care center was designated as a primary sampling unit. In the first stage of sampling, based on the number of preschool children assigned to each health center, 30 primary sampling units were selected through probability proportionate to size cluster sampling. In the second stage, a random number list was employed to select children from each cluster.

Surveys were exempted from review by the Institutional Review Board of CDC as the surveys were considered public health practice. The Ethics Committee under the Department of Drug Provision and Medical Equipment of the Kyrgyz Republic approved the survey protocols. In both the surveys, the field team informed children’s caretakers about the survey and asked them to provide written informed consent for participation. A timeline for the IYCN/MNP intervention and surveys is shown in Figure 1.

Data collection

Three field teams collected data for both surveys. In the 2008 survey, data were collected during June and July, and in the 2010 survey, data were collected during July and August. Selected children were invited to come to their health-care center on a predetermined day. If children did not arrive, survey team personnel visited the home.

Both the 2008 and 2010 questionnaires were written in English and then translated into the Kyrgyz and Russian languages.

Biochemical indicators

Hemoglobin was assessed using the HemoCue photometric instrument (Model 301, HemoCue AB, Angelholm, Sweden). Laboratory personnel collected capillary blood samples through a finger stick. After the first drop, the finger was wiped clean, and the second drop was drawn into a HemoCue cuvette. Afterwards, 500 μl of blood was collected in a Microtainer. Biochemical analysis was conducted using the 'sandwich assay'.[13] The biochemical indicators measured iron status (serum ferritin, soluble transferrin receptor (sTfR)), vitamin A (retinol-binding protein (RBP)), inflammation (C-reactive protein (CRP) and α_1-glycoprotein acid (AGP)).

Anemia was defined as an altitude-adjusted hemoglobin concentration of <11.0 g/dl.[14] Total iron deficiency was defined as either decreased serum ferritin concentration (<12 μg/l) or increased sTfR levels (>8.3 mg/l). Iron deficiency anemia was defined as having both a low hemoglobin value and either low serum ferritin or high sTfR.

RBP was used as an indicator of vitamin A status.[15] Based on a comparison of RBP and plasma retinol on a subsample of participants by the CDC Nutrition Laboratory, an RBP concentration less than 0.71 μmol/l was determined as the cutoff for vitamin A deficiency (personal communication, Rosemary Schleicher).

As serum ferritin and RBP are acute-phase reactants, two indicators of inflammation, CRP and AGP, were also measured.[10,11] Inflammation was considered present if either was elevated (CRP>5.0 mg/l or AGP>1.0 g/l). All biochemical results are presented for the total population and the population without inflammation.

Data analysis

As the 2008 survey included only children living in rural areas, in the 2010 survey, only children living in the 26 rural clusters were included in this analysis. Because children were first enrolled in the program either at the first well-child checkup after they reached 6 months of age or when the health-care worker visited their home, we expected that children 6–11 months of age would have had a shorter degree of exposure to the program than older children. Therefore, analyses were stratified by age (6–11 months and 12–24 months of age). Data analysis was conducted using Stata (version 10.0, StataCorp, College Station, TX, USA). Pearson χ^2 tests were run using methods that account for the design of the two surveys (standard errors were adjusted for the cluster survey design).

RESULTS

For the 2008 survey, 642 children were invited to participate; of those, 571 participated (88.9%). For the 71 children who did not participate, reasons for non-participation included: permanently moved (n =18), family refused (n =3), temporary migration or absence from the home (n =34) and other (n =16). Of those who participated, 571 had hemoglobin measurements and 569 had information on other biochemical indicators.

For the 2010 survey, 624 children from rural clusters were invited to participate; of those, 543 participated (87.0%). For the 81 children who did not participate, reasons for non-

participation included: permanently moved (*n* =6), family refused (*n* =5), temporary migration or absence from the home (*n* =58) and other (*n* =12). Of those who participated, 541 had acceptable hemoglobin measurements and 485 had information on other biochemical indicators.

In both surveys, participation was slightly higher in males, but fairly even across age groups. Inflammation was present in 42.0% of children in 2008 and 39.0% in 2010 (Table 1).

Among all children, the prevalence of anemia decreased from 50.6 to 43.8% (*P* =0.05); total iron deficiency (as measured by either low ferritin or high sTfR) from 77.3 to 63.7% (*P*<0.01); and iron deficiency anemia (defined as low hemoglobin and either low serum ferritin or high sTfR) from 45.5 to 33.4% (*P*-value<0.01; Table 2). The pattern of decline varied by age of the child. Among children 6–11 months of age, significant declines were observed only for iron deficiency (as measured by high sTfR). Among children 12–24 months of age, significant declines were observed for anemia (55.6–44.0%; *P* =0.01), total iron deficiency (84.5–67.2%; *P*<0.01) and iron deficiency anemia (53.0–34.3%; *P* <0.01).

Among children without inflammation, the prevalence of anemia decreased from 47.0 to 40.2% (*P* =0.13), but the decrease was not statistically significant. Total iron deficiency decreased from 79.7 to 67.2% (*P*<0.01) and iron deficiency anemia decreased from 43.3 to 34.1% (*P* =0.02). Among children 6–11 months of age, significant declines were observed only for iron deficiency as measured by sTfR. Among children 12–24 months of age, significant declines were observed for anemia (54.1–40.8%; *P* =0.01), total iron deficiency (88.5–70.9%; *P*<0.01) and iron deficiency anemia (52.6–35.0%; *P* <0.01).

Among all children, the prevalence of vitamin A deficiency was similar in the two surveys (19.5% vs 22.1%, *P* =0.44). Among those without inflammation, the prevalence of vitamin A deficiency was also similar (12.4% vs 9.8%, *P* =0.42).

DISCUSSION

Among all children, the decline in the prevalence of anemia was of borderline statistical significance; however, the decline in the prevalence estimates for total iron deficiency and iron deficiency anemia were statistically significant. Among children without evidence of inflammation, a similar magnitude of decline was observed for anemia and iron deficiency; however, only the declines in total iron deficiency and iron deficiency anemia reached statistical significance.

The 6.8 PP reduction in anemia observed in our population is similar to the findings of other effectiveness studies of home fortification. An earlier study by Lundeen *et al*[2] conducted in children 6–36 months living in the Kyrgyz Republic showed a 20-PP reduction in anemia (*P*<0.001) in the treatment group (72% vs 52%) compared with a 3-PP increase in the non-intervention comparison group (72% vs 75%). In that study, the treatment group received 60 MNP sachets (12.5 mg iron) to be taken daily over a 2-month period. In a study conducted in children 9–24 months of age living in rural Haiti, Menon *et al.* randomly allocated participants in a fortified wheat-soy-blend (WSB) food distribution program to either a treatment group that received both WSB and MNP sachets (12.5 mg iron) to be consumed

daily over a 2-month period or a control group that received WSB only.[8] From baseline to follow-up, the treatment group showed a 20-PP reduction (age and sex adjusted) in anemia (hemoglobin <100 g/l; 54% vs 24%, $P<0.001$) and the control group increased 4 PP (39% vs 43%, $P=0.07$). In a cluster randomized trial of children 6–11 months in Cambodia conducted by Jack *et al.*, children were assigned to either a treatment group that received both IYCN and MNP sachets (12.5 mg iron) or a control group that received IYCN alone.[9] At 12 months, compared with the control group, children in the treatment group showed a 20.6-PP reduction in anemia (84.7% vs 66.7%, $P<0.001$). In a series of cross-sectional surveys conducted in Bhutanese refugee children living in camps in Nepal, Bilukha assessed the anemia status of children 13 months before and at 26 months after distribution of MNP sachets (10 mg iron), which were to be consumed every other day.[7] Among children 6–23 month of age, a 10-PP lower prevalence of anemia was observed (71.6% vs 61.5%, $P<0.05$).

We found a statistically significant reduction between the 2008 and 2010 surveys in the prevalence of anemia, total iron deficiency and iron deficiency anemia among children 12–24 months of age, however, this was not the case among children 6–11 months of age. In contrast to our findings, the Lundeen study showed no difference in the impact of MNP by age group.[2] The Menon study showed a larger impact on younger children (6–17.9 months) compared with older children (18–21.9 months).[8] Although the reason for the differences between studies with regard to age impact is unclear, in our population the lesser impact in the youngest age group may have been due to delay in enrollment of the youngest children in the program leading to shorter treatment time and consequently lower impact.

Several possible factors could contribute to the other (non-age-related) differences in findings, including population size, duration of the intervention and adherence. Our intervention and the Bilukha refugee study were conducted over a 1- to 2-year period as part of a large-scale public health program.[7] The study by Jack *et al.* was conducted over a 6-month period, whereas the Lundeen and Menon studies were both conducted over a 2-month period and were small-scale interventions.[2,8,9] With regard to adherence, at our 1-year follow-up, 71.2% of children were reported to currently consume the MNP (data not shown). In the refugee population, Bilukha found that 90% of children were reported to currently consume the MNP at last follow-up. Rather than relying on self-report, other studies assessed adherence by counting the number of sachets remaining. On average, children consumed between 96%[8] and 75% of the sachets.[2,9,16]

We found that the prevalence of vitamin A deficiency was similar at baseline and follow-up. These findings are similar to those of three other studies of home fortification products in which there was no significant impact of the intervention on vitamin A status.[3,9,17,18] In our population, the lack of impact might be partially attributable to concurrent vitamin A capsule distribution, the insensitivity of current indicators to assess vitamin A status,[19] and the relatively low prevalence of vitamin A deficiency in the population without inflammation.

This pre-post cross-sectional survey design does not allow determination of causality. Although the demographic characteristics were similar between the baseline and follow-up surveys, the baseline survey was conducted 1 year before the MNP distribution. Without a

comparison population it is not possible to account for the economic and governmental changes, which occurred in the 2-year interval between the surveys.[20] Furthermore, we cannot distinguish the effects of the IYCN nutrition education program from the MNP program.

Based on the success of this pilot program, the Kyrgyz Republic will be among the first countries in the world to scale up its home fortification program nationwide. Our evaluation provides evidence that a large-scale MNP program delivered through the health-care system, combined with health education and extensive community mobilization, may have resulted in the reduction of anemia and iron deficiency in the Kyrgyz Republic.

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APPENDIX. The Kyrgyz Republic Working Group

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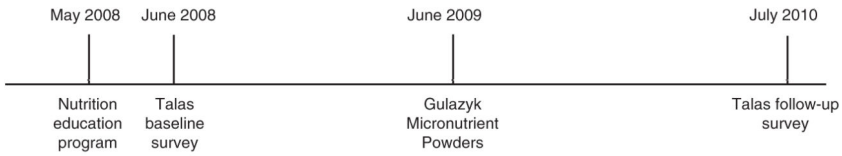

Figure 1.
Timeline for intervention and surveys.

Table 1

Characteristics of children[a] 6–24 months in rural Talas Oblast, Kyrgyz Republic, 2008 and 2010

Characteristics	2008		2010	
	n	% (95% CI)	n	% (95% CI)
Gender	571		543	
Male		52.4 (49.1, 55.6)		54.3 (50.0, 58.7)
Female		47.6 (44.4, 50.9)		45.7 (41.3, 50.0)
Age (months)	571		543	
6–11		35.4 (31.4, 39.3)		32.2 (29.5, 34.9)
12–17		33.5 (29.1, 37.8)		34.8 (32.0, 37.6)
18–24		31.2 (27.6, 34.7)		33.0 (29.7, 36.2)
Mother's education[b] (highest level completed)	488		528	
Less than secondary		0.8 (0.0, 2.1)		4.0 (0.3, 7.6)
Secondary		65.4 (60.5, 70.2)		64.2 (60.1, 68.3)
Technical		17.4 (13.7, 21.1)		13.4 (10.3, 16.6)
Higher education		16.4 (12.9, 19.9)		18.4 (14.5, 22.2)
Inflammation status				
Elevated CRP or AGP[c]	569	42.0 (37.0, 47.0)	485	39.0 (34.4, 43.6)
Elevated CRP		17.2 (14.4, 20.1)		17.3 (14.1, 20.5)
Elevated AGP		39.7 (35.0, 44.4)		35.1 (30.3, 39.8)
No inflammation[d]		58.0 (53.0, 63.0)		61.0 (56.4, 65.6)

Abbreviations: AGP, α1-glycoprotein acid; CI, confidence interval; CRP, C-reactive protein. 95% CI adjusted for cluster survey design.

[a] Calculated for children who had valid measurements for anthropometry, hemoglobin and/or other biochemical indicators (If a child had a valid measurement for at least one of these indicators they were included in this description of the sample.).

[b] Information on the mother's education level was not available for all participants with anthropometry and biochemical data.

[c] CRP >5.0 mg/l; AGP >1.0 g/l.

[d] CRP ≤5.0 mg/l and AGP ≤1.0 g/l.

Table 2

Prevalence of anemia, iron deficiency and vitamin A deficiency by age among children 6–24 months in rural Talas Oblast, Kyrgyz Republic, 2008 and 2010

Characteristics	All participants					Participants without inflammation[a]				
	2008 (Rural)		2010 (Rural)		P-value	2008 (Rural)		2010 (Rural)		P-value[b]
	N	% (95% CI)	N	% (95% CI)		N	% (95% CI)	N	% (95% CI)	
Anemia[c]										
6–11 (months)	202	41.6 (33.8, 49.4)	173	43.4 (36.0, 50.7)	0.74	121	34.7 (25.6, 43.8)	90	38.9 (28.9, 48.9)	0.54
12–24	369	55.6 (50.3, 60.8)	368	44.0 (38.1, 49.9)	0.01	209	54.1 (47.5, 60.6)	206	40.8 (33.2, 48.4)	0.01
Total 6–24	571	50.6 (45.7, 55.6)	541	43.8 (39.2, 48.4)	0.05	330	47.0 (41.0, 52.9)	296	40.2 (34.0, 46.4)	0.13
Low serum ferritin (<12.0 μg/l)										
6–11 (months)	201	43.8 (36.3, 51.3)	150	44.0 (34.0, 54.0)	0.97	121	49.6 (41.3, 57.9)	90	52.2 (41.4, 63.1)	0.70
12–24	368	72.0 (67.2, 76.8)	335	57.3 (51.8, 62.8)	<0.01	209	78.5 (72.4, 84.6)	206	66.0 (59.7, 72.4)	0.01
Total 6–24	569	62.0 (57.4, 66.7)	485	53.2 (47.6, 58.8)	0.02	330	67.9 (62.6, 73.2)	296	61.8 (55.2, 68.4)	0.16
High sTfR (>8.3 mg/l)										
6–11 (months)	201	59.2 (51.2, 67.2)	150	43.3 (36.3, 50.4)	0.01	121	58.7 (49.5, 67.9)	90	38.9 (30.4, 47.4)	<0.01
12–24	368	77.4 (72.9, 82.0)	335	48.4 (42.4, 54.4)	<0.01	209	81.8 (77.3, 86.3)	206	46.6 (40.4, 52.8)	<0.01
Total 6–24	569	71.0 (66.5, 75.5)	485	46.8 (41.9, 51.7)	<0.01	330	73.3 (68.2, 78.5)	296	44.3 (39.3, 49.2)	<0.01
Iron deficiency (low SF/high sTfR)										
6–11 (months)	201	64.2 (56.7, 71.7)	150	56.0 (47.6, 64.4)	0.15	121	64.5 (56.6, 72.3)	90	58.9 (48.8, 69.0)	0.39
12–24	368	84.5 (81.1, 87.9)	335	67.2 (61.4, 72.9)	<0.01	209	88.5 (84.4, 92.6)	206	70.9 (64.9, 76.8)	<0.01
Total 6–24	569	77.3 (73.1, 81.6)	485	63.7 (58.5, 68.9)	<0.01	330	79.7 (74.7, 84.7)	296	67.2 (61.3, 73.2)	<0.01
Iron deficiency anemia[d]										
6–11 (months)	201	31.8 (25.3, 38.4)	150	31.3 (22.0, 40.6)	0.93	121	27.3 (20.1, 34.5)	90	32.2 (20.8, 43.7)	0.46
12–24	368	53.0 (47.8, 58.1)	335	34.3 (28.7, 40.0)	<0.01	209	52.6 (46.4, 58.9)	206	35.0 (28.4, 41.5)	<0.01
Total 6–24	569	45.5 (41.0, 50.0)	485	33.4 (28.9, 37.9)	<0.01	330	43.3 (38.4, 48.3)	296	34.1 (28.8, 39.5)	0.02
Vitamin A deficiency[e]										
6–11 (months)	201	24.9 (18.8, 30.9)	150	20.7 (13.2, 28.1)	0.40	121	16.5 (9.2, 23.9)	90	18.9 (11.4, 26.4)	0.66
12–24	368	16.6 (12.2, 21.0)	335	22.7 (17.5, 27.9)	0.08	209	10.0 (6.0, 14.1)	206	5.8 (1.8, 9.9)	0.18
Total 6–24	569	19.5 (15.4, 23.6)	485	22.1 (17.0, 27.1)	0.44	330	12.4 (8.2, 16.7)	296	9.8 (5.2, 14.4)	0.42

Abbreviations: CI, confidence interval; SF, serum ferritin; sTfR, soluble transferrin receptor. 95% CIs adjusted for cluster survey design.

[a] C-reactive protein ≤5.0 mg/l; α1-glycoprotein acid ≤1.0 g/l.

[b] Bivariate tests of statistical significance were conducted using the Pearson χ^2 test, accounting for the cluster survey design.

[c] Anemia: hemoglobin <11.0 g/dl adjusted for altitude.

[d] Iron deficiency anemia: hemoglobin <11.0 g/dl and either low serum ferritin (<12.0 μg/l) or high sTfR (>8.3 mg/l).

[e] Vitamin A deficiency, defined as retinol-binding protein <0.71 μmol/l.